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1 **Management of Balance and Gait in Older Individuals with Parkinson Disease**

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16

17 **Summary**

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

27

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

30 **What is Parkinson Disease?**

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

38 PD is characterized by four cardinal features: tremor, bradykinesia, rigidity, and postural
39 instability. The symptoms of PD arise only after substantial loss of dopaminergic neurons in the
40 substantia nigra pars compacta. In fact, PD was originally attributed only to neuronal loss within
41 the substantia nigra pars compacta, and a concomitant loss of dopamine. Pharmacologic
42 replacement of dopamine is the most common, and arguably most effective, treatment for PD.
43 However, PD is now thought to involve not only the dopaminergic system, but also other
44 neurotransmitter systems whose role may become more prominent as the disease progresses
45 [6]. These non-dopaminergic systems may be particularly implicated in balance and gait
46 disorders, the management of which is the focus of this paper. Balance and gait impairments
47 are particularly problematic for individuals with PD, as they can lead to falls, injury, and fear of
48 future falls. As a result of injury and/or fear of falling, individuals may limit their physical activity
49 which may result in social isolation as well as subsequent muscle weakness and osteoporosis.
50 These in turn may contribute to increased risk of future falls and fall-related injuries [7]. The
51 purpose of this paper is to provide an overview of the gait and balance disorders associated
52 with PD and discusses the methods currently utilized to manage gait and balance problems
53 among people with PD. The ideal combination and prioritization of these approaches needs to
54 be determined on an case by case basis.

55

56 **Gait, Balance, and Falls in Parkinson Disease**

57 Gait Impairments

58 Many individuals with PD have gait difficulties that greatly limit functional mobility. The
59 majority of studies focusing on Parkinsonian gait examined walking in a straight line. These
60 studies showed that straight ahead walking in PD is characterized by: 1) a flexed posture, 2)
61 short, shuffling steps, 3) deficits in stride length regulation, 4) reduced foot clearance during
62 swing, 5) increased cadence, 6) limited extension at the hip and knee and reduced ankle
63 plantarflexion, 7) reduced transverse rotation of the trunk and pelvis, 8) inappropriate
64 coactivation of muscle agonist-antagonist pairs, and 9) reduced muscle activation amplitudes [8-
65 14]. The cause of these gait impairments is thought to be a disturbance in the motor set function
66 of the basal ganglia, which is involved in regulation of movement amplitude [15]. It has also
67 been hypothesized that the basal ganglia contribute to the regulation of postural alignment and
68 motor control, which may play a role in gait dysfunction in PD [16,17].

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

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

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

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

95

96 Balance Impairments

97 Postural instability is one of the cardinal symptoms of PD. Though it is often associated
98 with mid-to-late disease progression, recent studies have shown subclinical manifestations of
99 postural instability in the early stages of PD as well [37-40]. Many different factors are believed
100 to contribute to postural instability including impaired peripheral sensation and somatosensory
101 integration, decreased strength, axial rigidity, and impaired postural responses [41-46].

102 Disturbed motor programming within the basal ganglia as well as abnormally processed
103 proprioceptive signals are thought to be underlying causes of postural instability in PD [47].

104 Recent studies have also consistently shown that postural instability is not relieved with
105 dopaminergic medication, therefore, non-dopaminergic pathways have been hypothesized to
106 contribute to postural instability [48, 41].

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

114 Both anticipatory postural adjustments and reactive postural adjustments are impaired in
115 individuals with PD, with decreased amplitude and coordination of these movements [40, 54,
116 55]. An association exists between fear of falling and postural instability, though whether it is a
117 causative relationship or a correlation is unclear [56]. However, postural instability is also related
118 to falls in individuals with PD [57-59].

119

120 Falls

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

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

135 The majority of falls in this population occur in the home and have intrinsic causes (i.e.
136 disease processes such as postural instability, freezing, loss of concentration, etc.) as opposed
137 to extrinsic causes (i.e. a wet floor, obstacle, etc.) [60, 64, 65]. Falls often occur during transfers,
138 when bending or reaching, while walking especially in dual task situations, and during turning as
139 well as due to freezing and tripping/accidental causes [7, 60, 64, 65]. It is interesting to note that
140 2/3 of individuals who fell said their signs and symptoms were under control when they fell [60].

141 Many studies have compared individuals with PD who fall and those who do not fall.
142 Those who fall tend to have longer disease duration, increased disease severity, a history of
143 previous falls, increased levodopa replacement medication use, and increased freezing and
144 dyskinesia [64, 71, 43, 58, 59]. They perform more poorly in balance and functional mobility
145 measures, have increased fear of falling, have increased sway in standing, and have decreased
146 lower extremity strength [60, 71, 43, 72, 59]. Cognitive factors such as decreased memory,
147 power of attention, and reaction time have also been shown to be associated with falling [64,
148 58]. Though cardiovascular issues such as orthostatic hypotension and postprandial
149 hypotension have been shown to be present in individuals with PD, their contribution to falls has
150 been debated [64, 43, 58, 73].

151

152 **Therapeutic Approaches to Management of Gait and Balance in Parkinson Disease**

153 ***Pharmacological Treatment***

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

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

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

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

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

198

199 ***Deep Brain Stimulation***

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

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

216 Using quantitative measures, STN stimulation has been shown to improve gait
217 parameters such as stride length and gait velocity [109]. In a meta-analysis conducted by
218 Bakker et al., bilateral STN stimulation was found to improve gait disturbances and postural
219 instability, as assessed by the UPDRS, in the *off* medication state [108]. When examining anti-
220 Parkinson medication and STN DBS and their effects on gait and gait stability, Hausdorff et al.
221 [110] noted that combined effects of medication and DBS on gait speed, stride length, and gait
222 stability were larger than effects of DBS alone. Krack et al. [107] reported that STN stimulation
223 provided an initial improvement in gait and postural instability, but performance declined with
224 time, and by five years, patients' gait disturbances and postural instability were more impaired
225 than prior to surgery. For those patients experiencing freezing of gait (FOG) that is responsive
226 to levodopa (L-DOPA), STN stimulation has been shown to fully replicate the L-DOPA induced
227 improvement seen prior to surgery [107, 116-119]. However, regarding L-DOPA resistant FOG,
228 STN stimulation has proven to be ineffective [120, 121, 116, 118]. As such, new surgical targets
229 are necessary to determine if DBS surgery is effective at improving FOG in individuals with PD.

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

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

252

253 ***Physical Rehabilitation***

254 Treadmill Training

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

263 gait training, structured TT led to significant improvements in over-ground walking speed (1.37
264 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
265 as double stance duration [127]. Miyai et al. studied body weight-supported treadmill training
266 (BWSTT) versus conventional physical therapy in individuals with PD and found the BWSTT
267 group maintained improvements in gait speed and stride length at one and four months post
268 intervention [128]. Multi-directional TT (forward, backward, sideways) and step-perturbation
269 training have also been shown to improve cadence and gait speed in men with PD [129].

270 While gait parameters are commonly measured when investigating the effects of TT,
271 other studies have examined its effects on a variety of other outcome measures. Studies by
272 Cakit et al. and Protas et al. have shown that different protocols for TT can improve balance
273 [130], decrease fear of falling [130], and decrease number of falls [129]. A six week TT program
274 in which participants with PD walked at 70%-80% of maximal heart rate led to improvements in
275 lower extremity functional tasks (20-m walking time, timed U-turn, turning around a chair,
276 ascending/descending stairs, standing from a chair, and standing on one foot) [131]. In addition
277 to improvements in lower extremity capabilities and balance, Hermann et al. found that TT
278 significantly improved QOL [132].

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

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

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

297

298 Use of External Cues and Assistive Devices

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

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

310 Some studies have attempted to combine different cueing types to see if they have an
311 additive effect. When visual and auditory are combined for gait, it is less effective than either
312 separately [140]. However, when an audiovisual system was used to cue sit-to-stand transfers,
313 speed and torque of the transfer were better than without cueing [144]. An attentional strategy
314 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

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

345

346 Resistance Training

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

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

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

371

372 Balance Training

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

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

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

395

396 Flexibility Exercises

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

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

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

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

432

433 Alternative Exercise Programs

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

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

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

452

453 Multidisciplinary Care Approaches

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

463 Ellis et al.[200] further suggests that the comprehensive, multidisciplinary environment
464 might be effective for individuals with PD who are experiencing a decline in function, falling
465 more frequently, or are having more complex symptoms. After an average of 21 days of rehab,
466 71% of the patients in this cohort, including patients who did and did not require medication
467 alterations, showed a clinically important improvement according to the Functional
468 Independence Measure [200]. The inpatient rehabilitation team has been suggested to include
469 some combination of neurosurgeons, neurologists, physical therapists, occupational therapists,

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

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

481

482 **Remaining Challenges & Future Directions**

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

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

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

503

504 **EXECUTIVE SUMMARY**

505• **Gait, Balance and Falls in Parkinson Disease**

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

511○

512• **Therapeutic Approaches to Management of Gait and Balance Problems**

513○ Medical management of PD has, until relatively recently, focused primarily on pharmacological
514 approaches but many aspects of gait and balance impairments are not adequately addressed
515 by medication.
516○ More recently, deep brain stimulation has been added to the arsenal of treatment tools but
517 again does not fully address gait and balance issues.
518○ As such, physical rehabilitation approaches are a key aspect of managing gait and balance in
519 PD and can include specific gait and balance training, resistance training to improve muscle
520 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.

532

533 **References**

534

535 1. Fahn S: Description of Parkinson's disease as a clinical syndrome. *Annals of the New York*
536 *Academy of Science*. 991, 1-14 (2003). **** Very informative and easy to understand**
537 **overview of the major signs and symptoms of PD.**

538

539 2. Chade AR, Kasten M, Tanner CM: Nongenetic causes of Parkinson's disease. *J. Neural*
540 *Transm.* 70: 147-151 (2006).

541

542 3. Biskup S, Gerlach M, Kupsch A, et al.: Genes associated with Parkinson syndrome. *J.*
543 *Neurol.* 255, 8-17 (2008).

544

545 4. Taylor KS, Cook JA, Counsell CE: Heterogeneity in male to female risk for Parkinson's
546 disease. *J Neurol. Neurosurg. Psychiatry* 78, 905-906 (2007).

547

548 5. Spillman BC, Lubitz J: New estimates of lifetime nursing home use: have patterns of use
549 changed? *Med. Care.* 40, 965-975 (2002).

550

551 6. Perry, EK, et al.: Topography, extent, and clinical relevance of neurochemical deficits in
552 dementia of Lewy body type, Parkinson's disease, and Alzheimer's disease. *Annals of the New*
553 *York Academy of Science*. 640, 197-202 (1991).

554

555 7. Bloem BR, Hausdorff JM, Visser JE, et al.: Falls and freezing of gait in Parkinson's disease:
556 a review of two interconnected, episodic phenomena. *Mov. Disord.* 19(8), 871-884 (2004). ******
557 **Includes excellent model of how gait and balance impairments are part of a vicious cycle**
558 **that includes falling and reduced quality of life.**

559

560 8. Morris ME, Iansek R, Matyas TA et al.: Ability to modulate walking cadence remains intact in
561 Parkinson's disease. *Journal of Neurology, Neurosurgery and Psychiatry.* 57(12), 1532-4
562 (1994).

563

564 9. Morris ME, Iansek R, Matyas TA et al.: The pathogenesis of gait hypokinesia in Parkinson's
565 disease. *Brain.* 117(Pt.5), 1169-1181 (1994).

566

567 10. Morris ME, Iansek R, Matyas TA, et al: Stride length regulation in Parkinson's disease:
568 normalization strategies and underlying mechanisms. *Brain.* 119, 551-568 (1996).

569

570 11. Morris ME, McGinley J, Huxham F, et al: Constraints on the kinetic, kinematic, and
571 spatiotemporal parameters of gait in Parkinson's disease. *Hum. Mov. Sci.*, 18, 461-483 (1999).

572

573 12. Morris ME, Huxham FE, McGinley J, et al.: Gait disorders and gait rehabilitation in
574 Parkinson's disease. *Adv. Neurol.*, 87, 347-361 (2001).

575

576 13. Pedersen SW, Öberg B, Larsson LE, et al.: Gait analysis, isokinetic muscle strength
577 measurement in patients with Parkinson's disease. *Scand. J. Rehab. Med.* 29, 67-74 (1997).

578

579 14. Rogers MW. Disorders of posture, balance, and gait in Parkinson's disease. *Clin. Geriatr.*
580 *Med.* 12, 825-845 (1996).

581

582 15. Lewis GN, Byblow WD, Walt SE. Stride length regulation in Parkinson's disease: the use of
583 extrinsic, visual cues. *Brain.* 123(Pt. 10), 2077-2090, (2000).

- 584 16. Grasso R, Peppe A, Stratta F, et al.: Basal ganglia and gait control: apomorphine
585 administration and internal pallidum stimulation in Parkinson's disease. *Exp. Brain Res.* 126(2),
586 139-148, (1999).
587
- 588 17. Steiger MJ, Thompson PD, Marsden CD. Disordered axial movement in Parkinson's
589 disease. *J. Neurol. Neurosurg. Psychiatry.* 61(6), 645-648, (1996).
590
- 591 18. Camicioli R, Oken BS, Sexton G, et al.: Verbal fluency task affects gait in Parkinson's
592 disease with motor freezing. *J. Geriatr. Psychiatry Neurol.* 11(4), 181-185, (1998).
593
- 594 19. O'Shea S, Morris ME, Iansek R. Dual task interference during gait in people with Parkinson
595 disease: effects of motor versus cognitive secondary tasks. *Phys. Ther.* 82(9), 888-897 (2002).
596
- 597 20. Rochester L, Hetherington V, Jones D, et al.: Attending to the task: interference effects of
598 functional tasks on walking in Parkinson's disease and the roles of cognition, depression,
599 fatigue, and balance. *Arch. Phys. Med. Rehabil.* 85(10), 1578-1585 (2004).
600
- 601 21. Rochester L, Hetherington V, Jones D, et al.: The effect of external rhythmic cues (auditory
602 and visual) on walking during a functional task in homes of people with Parkinson's disease.
603 *Arch. Phys. Med. Rehabil.* 86(5), 999-1006 (2005).
604
- 605 22. Rochester L, Nieuwboer A, Baker K, et al.: The attentional cost of external rhythmical cues
606 and their impact on gait in Parkinson's disease: effect of cue modality and task complexity. *J.*
607 *Neural Transm.* 114(10), 1243-1248 (2007).
608
- 609 23. Plotnik M, Giladi N, Hausdorff JM. Bilateral coordination of gait and Parkinson's disease:
610 the effects of dual tasking. *J. Neurol. Neurosurg. Psychiatry.* 80(3), 347-50 (2009).
611
- 612 24. Hackney M, Earhart GM. Backward walking in Parkinson disease. *Mov. Disord.* 24(2), 218-
613 223 (2009).
614
- 615 25. Hackney ME, Earhart GM. The effects of a secondary task on forward and backward
616 walking in Parkinson disease. *Neurorehabil. Neural Repair*, in press.
617
- 618 26. Morris ME, Huxham F, McGinley J et al.: The biomechanics and motor control of gait in
619 Parkinson disease. *Clinical Biomechanics.* 16(6) 459-470 (2001).
620 * **Good description of gait in PD and effects of medication.**
621
- 622 27. Stack EL, Ashburn Am, Jupp KE: Strategies used by people with Parkinson's disease who
623 report difficulty turning. *Parkinsonism Relat. Disord.* 12, 87-92 (2006).
624
- 625 28. Crenna P, Carpinella I, Rabuffetti M, et al.: The association between impaired turning and
626 normal straight walking in Parkinson's disease. *Gait & Posture.* 26, 172-178 (2006).
627
- 628 29. Visser JE, Voermans NC, Oude Nijhuis LB, et al.: Quantification of trunk rotations during
629 turning and walking in Parkinson's disease. *Clin. Neurophysiol.* 118, 1602-1606 (2007).
630
- 631 30. Hong M, Perlmutter JS, Earhart GM: A kinematic and electromyographic analysis of turning
632 in people with Parkinson disease. *Neurorehabil. Neural Repair.* 23(2), 166-176 (2009).
633

- 634 31. Nieuwboer A, Dom R, De Weerd W, et al.: Abnormalities of the spatiotemporal
635 characteristics of gait at the onset of freezing in Parkinson's disease. *Mov. Disord.* 16, 1066-
636 1075 (2001).
637
- 638 32. Giladi N, Nieuwboer A: Understanding and treating freezing of gait in parkinsonism,
639 proposed working definition, and setting the stage. *Mov. Disord.* 23, S423-S425 (2008).
640
- 641 33. Michałowska M, Fischer U, Krygowska-Wajs A, et al.: Falls in Parkinson's disease. Causes
642 and impact on patients' quality of life. *Funct. Neurol.* 20(4), 163-8 (2005).
643
- 644 34. Robinson K, Dennison A, Roalf D, et al.: Falling risk factors in Parkinson's disease.
645 *NeuroRehabilitation.* 20(3), 169-82 (2005).
646
- 647 35. Moore ST, MacDougall HG, Ondo WG: Ambulatory monitoring of freezing of gait in
648 Parkinson's disease. *J Neurosci Methods*, 167, 340-348 (2008).
649
- 650 36. Cumming R, Klineberg R: Fall frequency and characteristics and the risk of hip fractures. *J.*
651 *Am. Ger. Soc.* 42, 774-778 (1994).
652
- 653 37. Benatru I, Vaugoyeau M, Azulay JP: Postural disorders in Parkinson's disease.
654 *Neurophysiologie Clinique.* 38(6), 459-465 (2008).
655
- 656 38. Chastan N, Debono B, Maltete D, Weber J: Discordance between measured postural
657 instability and absence of clinical symptoms in Parkinson's disease patients in the early stages
658 of the disease. *Movement Disorders.* 23(3), 366-372 (2008).
659
- 660 39. Frenklach A, Louie S, Koop MM, Bronte-Stewart H: Excessive postural sway and the risk of
661 falls at different stages of Parkinson's disease. *Movement Disorders.* 24(3), 377-385 (2009).
662
- 663 40. Mancini M, Zampieri C, Carlson-Kuhta P, Chiari L, Horak FB: Anticipatory postural
664 adjustments prior to step initiation are hypometric in untreated Parkinson's disease: an
665 accelerometer-based approach. *European Journal of Neurology.* 16(9), 1028-1034 (2009).
666
- 667 41. Grimbergen YA, Langston JW, Roos RA, Bloem BR: Postural instability in Parkinson's
668 disease: the adrenergic hypothesis and the locus coeruleus. *Expert Review of*
669 *Neurotherapeutics.* 9(2), 279-290 (2009).
670
- 671 42. Horak FB, Dimitrova D, Nutt JG: Direction-specific postural instability in subjects with
672 Parkinson's disease. *Exp. Neurol.* 193(2), 504-521 (2005).
673
- 674 43. Kerr GK, Worringham CJ, Cole MH, Lacherez PF, Wood JM, Silburn PA: Predictors of future
675 falls in Parkinson disease. *Neurology.* 75(2), 116-124 (2010).
676
- 677 44. Kim S, Horak FB, Carlson-Kuhta P, Park S: Postural feedback scaling deficits in Parkinson's
678 disease. *J. Neurophysiol.* 102(5), 2910-2920 (2009).
679
- 680 45. King LA, St George RJ, Carlson-Kuhta P, Nutt JG, Horak FB: Preparation for Compensatory
681 Forward Stepping in Parkinson's Disease. *Arch. Phys. Med. Rehabil.* 91(9), 1332-1338 (2010).
682
- 683 46. McVey MA, Stylianou AP, Luchies CW, et al.: Early biomechanical markers of postural
684 instability in Parkinson's disease. *Gait Posture.* 30(4), 538-542 (2009).

- 685 47. Grimbergen YA, Munneke M, Bloem BR. Falls in Parkinson's disease. *Curr. Opin. Neurol.*
686 17(4), 405-415, (2004).
687
- 688 48. Bohnen NI, Cham R:Postural control, gait, and dopamine functions in parkinsonian
689 movement disorders. *Clin. Geriatr. Med.* 22(4), 797-812, vi (2006).
690
- 691 49. Visser M, Marinus J, Bloem BR, Kisjes H, van den Berg BM, van Hilten JJ:Clinical tests for
692 the evaluation of postural instability in patients with parkinson's disease. *Arch. Phys. Med.*
693 *Rehabil.* 84(11), 1669-1674 (2003).
694
- 695 50. Valkovic P, Brozova H, Botzel K, Ruzicka E, Benetin J:Push-and-release test predicts
696 Parkinson fallers and nonfallers better than the pull test: comparison in OFF and ON medication
697 states. *Mov. Disord.* 23(10), 1453-1457 (2008).
698
- 699 51. Blaszczyk JW, Orawiec R, Duda-Klodowska D, Opala G:Assessment of postural instability
700 in patients with Parkinson's disease. *Exp. Brain Res.* 183(1), 107-114 (2007).
701
- 702 52. Buckley TA, Pitsikoulis C, Hass CJ:Dynamic postural stability during sit-to-walk transitions in
703 Parkinson disease patients. *Mov. Disord.* 23(9), 1274-1280 (2008).
704
- 705 53. Merello M, Fantacone N, Balej J:Kinematic study of whole body center of mass position
706 during gait in Parkinson's disease patients with and without festination. *Mov. Disord.* 25(6), 739-
707 746 (2010).
708
- 709 54. Bleuse S, Cassim F, Blatt JL, *et al.*:Anticipatory postural adjustments associated with arm
710 movement in Parkinson's disease: a biomechanical analysis. *J. Neurol. Neurosurg. Psychiatry.*
711 79(8), 881-887 (2008).
712
- 713 55. Dimitrova D, Horak FB, Nutt JG:Postural muscle responses to multidirectional translations in
714 patients with Parkinson's disease. *J. Neurophysiol.* 91(1), 489-501 (2004).
715
- 716 56. Adkin AL, Frank JS, Jog MS:Fear of falling and postural control in Parkinson's disease. *Mov.*
717 *Disord.* 18(5), 496-502 (2003).
718
- 719 57. Ashburn A, Fazakarley L, Ballinger C, Pickering R, McLellan LD, Fitton C:A randomised
720 controlled trial of a home based exercise programme to reduce the risk of falling among people
721 with Parkinson's disease. *Journal of Neurology, Neurosurgery & Psychiatry.* 78(7), 678-684
722 (2007).
723
- 724 58. Latt MD, Lord SR, Morris JG, Fung VS:Clinical and physiological assessments for
725 elucidating falls risk in Parkinson's disease. *Mov. Disord.* 24(9), 1280-1289 (2009).
726
- 727 59. Matinulli M, Korpelainen JT, Korpelainen R, Sotaniemi KA, Virranniemi M, Myllyla
728 VV:Postural sway and falls in Parkinson's disease: a regression approach. *Mov. Disord.* 22(13),
729 1927-1935 (2007).
730
- 731 60. Bloem BR, Grimbergen YA, Cramer M, Willemsen M, Zwinderman AH:Prospective
732 assessment of falls in Parkinson's disease. *J. Neurol.* 248(11), 950-958 (2001).
733
- 734 61. Fink HA, Kuskowski MA, Orwoll ES, Cauley JA, Ensrud KE, Osteoporotic Fractures in Men
735 (MrOS) Study Group:Association between Parkinson's disease and low bone density and falls in

736 older men: the osteoporotic fractures in men study. *J. Am. Geriatr. Soc.* 53(9), 1559-1564
737 (2005).
738
739 62. Temlett JA, Thompson PD:Reasons for admission to hospital for Parkinson's disease.
740 *Intern. Med. J.* 36(8), 524-526 (2006).
741
742 63. Woodford H, Walker R:Emergency hospital admissions in idiopathic Parkinson's disease.
743 *Movement Disorders.* 20(9), 1104-1108 (2005).
744
745 64. Allcock LM, Rowan EN, Steen IN, Wesnes K, Kenny RA, Burn DJ:Impaired attention
746 predicts falling in Parkinson's disease. *Parkinsonism Relat. Disord.* 15(2), 110-115 (2009).
747
748 65. Ashburn A, Stack E, Ballinger C, Fazakarley L, Fitton C:The circumstances of falls among
749 people with Parkinson's disease and the use of Falls Diaries to facilitate reporting. *Disabil.*
750 *Rehabil.* 30(16), 1205-1212 (2008).
751
752 66. Wood BH, Bilclough JA, Bowron A, Walker RW:Incidence and prediction of falls in
753 Parkinson's disease: a prospective multidisciplinary study. *J. Neurol. Neurosurg. Psychiatry.*
754 72(6), 721-725 (2002).
755
756 67. Melton LJ III, Leibson CL, Achenbach SJ, Bower JH, Maraganore DM, Ober AL, Rocca WA.
757 Fracture risk after the diagnosis of Parkinson's disease: influence of concomitant dementia. *Mov*
758 *Disord*, 21(9): 1361-1367, 2006.
759
760 68. Rahman S, Griffin HJ, Quinn NP, Jahanshahi M:Quality of life in Parkinson's disease: The
761 relative importance of the symptoms. *Movement Disorders.* 23(10), 1428-1434 (2008).
762
763 69. Bacon WE: Secular trends in hip fracture occurrence and survival: age and sex differences.
764 *J. Aging Health.* 8(4), 538-553 (1996).
765
766 70. Kempster PA, O'Sullivan SS, Holton JL, Revesz T, Lees AJ:Relationships between age and
767 late progression of Parkinson's disease: a clinico-pathological study. *Brain.* 133(Pt 6), 1755-
768 1762 (2010).
769
770 71. Dennison AC, Noorigian JV, Robinson KM, *et al.*:Falling in Parkinson disease: identifying
771 and prioritizing risk factors in recurrent fallers. *Am. J. Phys. Med. Rehabil.* 86(8), 621-632
772 (2007).
773
774 72. Mak MK, Pang MY:Fear of falling is independently associated with recurrent falls in patients
775 with Parkinson's disease: a 1-year prospective study. *J. Neurol.* 256(10), 1689-1695 (2009).
776
777 73. Martignoni E, Tassorelli C, Nappi G:Cardiovascular dysautonomia as a cause of falls in
778 Parkinson's disease. *Parkinsonism Relat. Disord.* 12(4), 195-204 (2006).
779
780 74. Moore ST, MacDougall HG, Gracies JM *et al.*: Locomotor response to levodopa in
781 fluctuating Parkinson's Disease. *Experimental Brain Research.* 184(4), 469-478 (2008).
782
783 75. Sethi K.: Levodopa unresponsive symptoms in Parkinson disease. *Movement Disorders.*
784 23(Suppl 3), S521-S533 (2008).
785

- 786 76. Blin O, Fernandez AM, Pailhous J et al.: Dopa-sensitive and dopa-resistance gait
787 parameters in Parkinson's disease. *Journal of the Neurological Sciences*. 103(1), 51-4 (1991).
788
- 789 77. Blin O, Fernandez AM, Serratrice G.: Quantitative analysis of gait in Parkinson patients:
790 increased variability of stride length. *Journal of the Neurological Sciences*. 98(1), 91-7 (1990).
791
- 792 78. Bowes SG, Clark PK, Leeman AL et al.: Determinants of gait in the elderly parkinsonian on
793 maintenance levodopa/carbidopa therapy. *British Journal of Clinical Pharmacology*. 30(1), 13-24
794 (1990).
795
- 796 79. Pedersen SW, Eriksson T, Oberg B. Effects of withdrawal of antiparkinson medication on
797 gait and clinical score in the Parkinson patient. *Acta Neurologica Scandinavica*. 84(1), 7-13
798 (1991).
799
- 800 80. McIntosh GC, Brown SH, Rice RR et al.: Rhythmic auditory-motor facilitation of gait patterns
801 in patients with Parkinson's disease. *Journal of Neurology, Neurosurgery and Psychiatry*. 62(1),
802 22-6 (1997).
803
- 804 81. Azulay JP, Mesure S, Amblard B et al.: Visual control of locomotion in Parkinson's disease.
805 *Brain*. 122(Pt.1), 111-120 (1999).
806
- 807 82. Hanakawa T, Katsumi Y, Fukuyama H et al.: Enhanced lateral premotor activity during
808 paradoxical gait in Parkinson's disease. *Annals of Neurology*. 45(3), 329-336 (1999).
809
- 810 83. Ebersbach G, Sojer M, Valldeoriola F et al.: Comparative analysis of gait in Parkinson's
811 disease, cerebellar ataxia and subcortical arteriosclerotic encephalopathy. *Brain*. 122(Pt.7)
812 1349-1355 (1999).
813
- 814 84. Morris ME, Matyas TA, Iansek R et al.: Temporal stability of gait in Parkinson's disease.
815 *Physical Therapy*. 76(7), 763-777 (1996).
816
- 817 85. Koozekanani SH, Balmaseda MT Jr, Fatehi MT et al.: Ground reaction forces during
818 ambulation in parkinsonism: pilot study. *Archives of Physical Medicine & Rehabilitation*. 68(1),
819 28-30 (1987).
820
- 821 86. O'Sullivan JD, Said CM, Dillon LC et al.: Gait analysis in patients with Parkinson's disease
822 and motor fluctuations: influence of levodopa and comparison with other measures of motor
823 function. *Movement Disorders*. 13(6), 900-6 (1998).
824
- 825 87. Morris M, Iansek R, Matyas T et al.: Abnormalities in the stride length-cadence relation in
826 parkinsonian gait. *Movement Disorders*. 13(1), 61-9 (1998).
827
- 828 88. Okuma Y.: Freezing of gait in Parkinson's disease. *Journal of Neurology*. 253(Suppl 7),
829 VII27-32 (2006).
830
- 831 89. Fahn S.: The freezing phenomenon in parkinsonism. *Advances in Neurology*. 67, 53-63
832 (1995).
833
- 834 90. Schaafsma JD, Balash Y, Gurevich T et al.: Characterization of freezing of gait subtypes
835 and the response of each to levodopa in Parkinson's disease. *European Journal of Neurology*.
10(4), 391-8 (2003).

- 837
838 91. Giladi N.: Freezing of gait. Clinical overview. *Advances in Neurology*. 87, 191-7 (2001).
839
840 92. Giladi N.: Medical treatment of freezing of gait. *Movement Disorders*. 23 (Suppl 2), S482-8
841 (2008).
842
843 93. Giladi N, McDermott MP, Fahn S et al.: Freezing of gait in PD: prospective assessment in
844 the DATATOP cohort. *Neurology*. 56(12), 1712-1721 (2001).
845
846 94. Shoulson I.: DATATOP: a decade of neuroprotective inquiry. Parkinson Study Group.
847 Deprenyl and tocopherol antioxidative therapy of parkinsonism. *Annals of Neurology*. 44(3
848 Suppl 1), S160-6 (1998).
849
850 95. Shoulson I, Oakes D, Fahn S et al.: Impact of sustained deprenyl (selegiline) in levodopa-
851 treated Parkinson's disease: a randomized placebo-controlled extension of the deprenyl and
852 tocopherol antioxidative therapy of parkinsonism trial. *Annals of Neurology*. 51(5), 604-612
853 (2002).
854
855 96. Giladi N, Treves TA, Simon ES et al.: Freezing of gait in patients with advances Parkinson's
856 disease. *Journal of Neural Transmission*. 108(1), 53-61 (2001).
857
858 97. Macht M, Kaussner Y, Moller JC et al.: Predictors of freezing in Parkinson's disease: a
859 survey of 6,620 patients. *Movement Disorders*. 22(7), 953-6 (2007).
860
861 98. Giladi N and Honigman S.: Botulinum toxin injections to one leg alleviate freezing of gait in a
862 patient with Parkinson's disease. *Movement Disorders*. 12(6), 1085-6 (1997).
863
864 99. Giladi N, Gurevich T, Shabtai H et al.: The effect of botulinum toxin injections to the calf
865 muscles on freezing of gait in parkinsonism: a pilot study. *Journal of Neurology*. 248(7), 572-6
866 (2001).
867
868 100. Gurevich T, Peretz C, Moore O et al.: The effect of injecting botulinum toxin type a into the
869 calf muscles on freezing of gait in Parkinson's disease: a double blind placebo-controlled pilot
870 study. *Movement Disorders*. 22(6), 880-3 (2007).
871
872 101. Pollak L, Dobronevsky Y, Prohorov T et al.: Low dose methylphenidate improves freezing
873 in advanced Parkinson's disease during off-state. *Journal of Neural Transmission*
874 *Supplementum*. 72, 145-8 (2007).
875
876 102. Auriel E, Hausdorff JM, Herman T et al.: Effects of methylphenidate on cognitive function
877 and gait in patients with Parkinson's disease: a pilot study. *Clinical Neuropharmacology*. 29(1),
878 15-7 (2006).
879
880 103. Vaamonde Gamo J, Cabello JP, Gallardo Alcañiz MJ, et al.: Freezing of gait unresponsive
881 to dopaminergic stimulation in patients with severe Parkinsonism. *Neurologia*. 25(1), 27-31
882 (2010).
883
884 104. Clarke CE, Worth P, Grosset D et al.: Systematic review of apomorphine infusion,
885 levodopa infusion and deep brain stimulation in advanced Parkinson's disease. *Parkinsonism &*
886 *Related Disorders*. 15(10), 728-741 (2009).
887

- 888 105. Tagliati M. Fine-tuning gait in Parkinson disease. *Neurology*. 71(2), 76-7 (2008).
889
- 890 106. Van Nuenen BF, Esselink RA, Munneke M et al.: Postoperative gait deterioration after
891 bilateral subthalamic nucleus stimulation in Parkinson's disease. *Movement Disorders*. 23(16),
892 2404-6 (2008).
893
- 894 107. Krack P, Batir A, Blercom N et al.: Five-year follow-up of bilateral stimulation of the
895 subthalamic nucleus in advanced Parkinson's disease. *The New England Journal of Medicine*.
896 349(20), 1925-1934 (2003).
897
- 898 108. Bakker M, Esselink RA, Munneke M et al.: Effects of stereotactic neurosurgery on postural
899 instability and gait in Parkinson's disease. *Movement Disorders*. 19(9), 1092-9 (2004).
900
- 901 109. Johnsen EL, Mogensen PH, Sunde NA et al.: Improved asymmetry of gait in Parkinson's
902 disease with DBS: gait and postural instability in Parkinson's disease treated with bilateral
903 stimulation in the subthalamic nucleus. *Movement Disorders*. 24(4), 590-7 (2009).
904
- 905 110. Hausdorff JM, Gruendlinger L, Scollins L et al.: Deep brain stimulation effects on gait
906 variability in Parkinson's disease. *Movement Disorders*. 24(11), 1688-1692 (2009).
907
- 908 111. Volkmann J.: Deep brain stimulation for the treatment of Parkinson's disease. *Journal of*
909 *Clinical Neurophysiology*. 21(1), 6-17 (2004).
910
- 911 112. Mazzone P, Lozano A, Stanzione P et al.: Implantation of human pedunculopontine
912 nucleus: a safe and clinically relevant target in Parkinson's disease. *Neuroreport*. 16(17), 1877-
913 1881 (2005).
914
- 915 113. Plaha P and Gill SS.: Bilateral deep brain stimulation of the pedunculopontine nucleus for
916 Parkinson's disease. *Neuroreport*. 16(17), 1883-7 (2005).
917
- 918 114. Moro E, Hamani C, Poon YY et al.: Unilateral pedunculopontine stimulation improves fall in
919 Parkinson's disease. *Brain*. 133(Pt.1), 215-224 (2010).
920
- 921 115. Stefani A, Lozano AM, Peppe A et al.: Bilateral deep brain stimulation of the
922 pedunculopontine and subthalamic nuclei in severe Parkinson's disease. *Brain*. 130(Pt.6), 1596-
923 1607 (2007).
924
- 925 116. Davis JT, Lyons KE, Pahwa R.: Freezing of gait after bilateral subthalamic nucleus
926 stimulation for Parkinson's disease. *Clinical Neurology and Neurosurgery*. 108(5), 461-4 (2006).
927
- 928 117. Limousin P, Krack P, Pollak P et al.: Electrical stimulation of the subthalamic nucleus in
929 advanced Parkinson's disease. *The New England Journal of Medicine*. 339(16), 1105-1111
930 (1998).
931
- 932 118. Stolze H, Klebe S, Poepping M et al.: Effects of bilateral subthalamic nucleus stimulation
933 on parkinsonian gait. *Neurology*. 57(1), 144-6 (2001).
934
- 935 119. Yokoyama T, Sugiyama K, Nishizawa S et al.: Subthalamic nucleus stimulation for gait
936 disturbance in Parkinson's disease. *Neurosurgery*. 45(1), 41-7 (1999).
937

938 120. Rodriguez-Oroz MC, Obeso JA, Lang AE et al.: Bilateral deep brain stimulation in
939 Parkinson's disease: a multicentre study with 4 years follow-up. *Brain*. 128(Pt.10), 2240-9
940 (2005).
941
942 121. Bejjani BP, Gervais D, Arnulf I et al.: Axial parkinsonian symptoms can be improved: the
943 role of levodopa and bilateral subthalamic stimulation. *Journal of Neurology, Neurosurgery and*
944 *Psychiatry*. 68(5), 595-600 (2000)
945
946 122. Nandi D, Aziz TZ, Giladi N et al.: Reversal of akinesia in experimental parkinsonism by
947 GABA antagonist microinjections in the pedunculopontine nucleus. *Brain*. 125(Pt.11), 2418-
948 2430 (2002).
949
950 123. Munro-Davies LE, Winter J, Aziz TZ et al.: The role of the pedunculopontine region in
951 basal-ganglia mechanisms of akinesia. *Experimental Brain Research*. 129(4), 511-7 (1999).
952
953 124. Benedetti F, Pollo A, Lopiano L et al.: Conscious expectation and unconscious
954 conditioning in analgesic, motor, and hormonal placebo/nocebo responses. *The Journal of*
955 *Neuroscience*. 23(10), 4315-4323 (2003).
956
957 125. Mehrholz J, Friis R, Kugler J et al.: Treadmill training for patients with Parkinson's disease
958 (Review). *Cochrane Database Systematic Review*. (1), 1-31 (2010).
959
960 126. Fisher BE, Wu AD, Salem GJ et al.: The effect of exercise training in improving motor
961 performance and corticomotor excitability in people with early Parkinson's disease. *Archives of*
962 *Physical Medicine & Rehabilitation*. 89(7), 1221-9 (2008).
963
964 127. Pohl M, Rockstroh G, Ruckriem S et al.: Immediate effects of speed-dependent treadmill
965 training on gait parameters in early Parkinson's disease. *Archives of Physical Medicine and*
966 *Rehabilitation*. 84(12), 1760-6 (2003).
967
968 128. Miyai I, Fujimoto Y, Ueda Y et al.: Treadmill training with body weight support: its effect on
969 Parkinson's disease. *Archives of Physical Medicine and Rehabilitation*. 81(7), 849-852 (2000).
970
971 129. Protas EJ, Mitchell K, Williams A et al. Gait and step training to reduce falls in Parkinson's
972 disease. *NeuroRehabilitation*. 20(3), 183-190 (2005).
973
974 130. Cakit BD, Saracoglu M, Genc H et al.: The effects of incremental speed-dependent
975 treadmill training on postural instability and fear of falling in Parkinson's disease. *Clinical*
976 *Rehabilitation*. 21(8), 698-705 (2007).
977
978 131. Kurtais Y, Kutlay S, Tur BS et al.: Does treadmill training improve lower-extremity tasks in
979 Parkinson disease? A randomized controlled trial. *Clinical Journal of Sports Medicine*. 18(3),
980 289-291 (2008).
981
982 132. Herman T, Giladi N, Gruendlinger L et al.: Six weeks of intensive treadmill training
983 improves gait and quality of life in patients with Parkinson's disease: A pilot study. *Archives of*
984 *Physical Medicine and Rehabilitation*. 88(9), 1154-8 (2007).
985
986 133. Bello O, Marquez G, Camblor M et al.: Mechanisms involved in treadmill walking
987 improvements in Parkinson's disease. *Gait and Posture*. 32, 118-123 (2010).
988

989 134. Rubinstein TC, Giladi N, Hausdorff JM.: The power of cueing to circumvent dopamine
990 deficits: a review of physical therapy treatment of gait disturbances in Parkinson's disease.
991 *Movement Disorders*. 17(6), 1148-1160 (2002).
992

993 135. Murray MP, Sepic SB, Gardner GM et al.: Walking patterns of men with parkinsonism.
994 *American Journal of Physical Medicine*. 57(6), 278-294 (1978).
995

996 136. Morris ME.: Movement disorders in people with Parkinson disease: a model for physical
997 therapy. *Physical Therapy*. 80(6), 578-597 (2000).
998

999 137. Rochester L, Baker K, Hetherington V, et al.:Evidence for motor learning in Parkinson's
1000 disease: acquisition, automaticity and retention of cued gait performance after training with
1001 external rhythmical cues. *Brain Res*. 1319 103-111 (2010).
1002

1003 138. Jiang Y, Norman KE:Effects of visual and auditory cues on gait initiation in people with
1004 Parkinson's disease. *Clin. Rehabil*. 20(1), 36-45 (2006).
1005

1006 139. Rochester L, Burn DJ, Woods G, Godwin J, Nieuwboer A:Does auditory rhythmical cueing
1007 improve gait in people with Parkinson's disease and cognitive impairment? A Feasibility study.
1008 *Movement Disorders*. 24(6), 839-845 (2009).
1009

1010 140. Suteerawattananon M, Morris GS, Etnyre BR, Jankovic J, Protas EJ:Effects of visual and
1011 auditory cues on gait in individuals with Parkinson's disease. *J. Neurol. Sci*. 219(1-2), 63-69
1012 (2004).
1013

1014 141. Willems AM, Nieuwboer A, Chavret F, et al.:Turning in Parkinson's disease patients and
1015 controls: the effect of auditory cues. *Mov. Disord*. 22(13), 1871-1878 (2007).
1016

1017 142. Nieuwboer A, Baker K, Willems AM, et al.:The short-term effects of different cueing
1018 modalities on turn speed in people with Parkinson's disease. *Neurorehabil. Neural Repair*.
1019 23(8), 831-836 (2009).
1020

1021 143. Rochester L, Rafferty D, Dotchin C, Msuya O, Minde V, Walker RW:The effect of cueing
1022 therapy on single and dual-task gait in a drug naive population of people with Parkinson's
1023 disease in northern Tanzania. *Mov. Disord*. 25(7), 906-911 (2010).
1024

1025 144. Mak MK, Hui-Chan CW:Cued task-specific training is better than exercise in improving sit-
1026 to-stand in patients with Parkinson's disease: A randomized controlled trial. *Mov. Disord*. 23(4),
1027 501-509 (2008).
1028

1029 145. Baker K, Rochester L, Nieuwboer A:The effect of cues on gait variability--reducing the
1030 attentional cost of walking in people with Parkinson's disease. *Parkinsonism Relat. Disord*.
1031 14(4), 314-320 (2008).
1032

1033 146. Baker K, Rochester L, Nieuwboer A:The Immediate Effect of Attentional, Auditory, and a
1034 Combined Cue Strategy on Gait During Single and Dual Tasks in Parkinson's Disease. *Arch*.
1035 *Phys. Med. Rehabil*. 88(12), 1593-1600 (2007).
1036

1037 147. Nieuwboer A: Cueing for freezing of gait in patients with Parkinson's disease: a
1038 rehabilitation perspective. *Mov. Disord*. 23 Suppl 2 S475-81 (2008).
1039

- 1040 148. Bryant MS, Rintala DH, Lai EC, Protas EJ:An evaluation of self-administration of auditory
1041 cueing to improve gait in people with Parkinson's disease. *Clin. Rehabil.* 23(12), 1078-1085
1042 (2009).
1043
- 1044 149. Morris ME, Martin CL, Schenkman ML:Striding out with Parkinson disease: evidence-
1045 based physical therapy for gait disorders. *Phys. Ther.* 90(2), 280-288 (2010).
1046 * **Review of current evidence to support specific physical therapy approaches to**
1047 **management of gait in PD.**
1048
- 1049 150. Satoh M, Kuzuhara S:Training in mental singing while walking improves gait disturbance in
1050 Parkinson's disease patients. *Eur. Neurol.* 60(5), 237-243 (2008).
1051
- 1052 151. Ford MP, Malone LA, Nyikos I, Yelisetty R, Bickel CS:Gait training with progressive
1053 external auditory cueing in persons with Parkinson's disease. *Arch. Phys. Med. Rehabil.* 91(8),
1054 1255-1261 (2010).
1055
- 1056 152. Nieuwboer A, Kwakkel G, Rochester L, et al.: Cueing training in the home improves gait-
1057 related mobility in Parkinson's disease: the RESCUE trial. *J. Neurol. Neurosurg. Psychiatry.*
1058 78(2), 134-140, (2007).
1059
- 1060 153. Bryant MS, Rintala DH, Lai EC, et al.: Evaluation of a new device to prevent falls in
1061 persons with Parkinson's disease.*Disabil Rehabil Assist Technol.* 4(5), 357-63 (2010).
1062
- 1063 154. Cubo E, Moore CG, Leugran S, et al.: Wheeled and standard walkers in Parkinson's
1064 disease patients with gait freezing.*Parkinsonism Relat. Disord.* 10(1), 9-14 (2003).
1065
- 1066 155. Constantinescu R, Leonard C, Deeley C, et al.: Assistive devices for gait in Parkinson's
1067 disease. *Parkinsonism Relat. Disord.*13(3), 133-8 (2007).
1068
- 1069 156. Bächlin M, Plotnik M, Roggen D, et al.: A wearable system to assist walking of Parkinson s
1070 disease patients.*Methods Inf. Med.* 49(1), 88-95 (2010).
1071
- 1072 157. Cano-de-la-Cuerda R, Perez-de-Heredia M, Miangolarra-Page JC et al.: Is there muscular
1073 weakness in Parkinson's disease? *American Journal of Physical Medicine & Rehabilitation.*
1074 89(1), 70-6 (2010).
1075
- 1076 158. Koller W and Kase S. Muscle strength testing in Parkinson's disease. *European Neurology.*
1077 25(2), 130-3 (1986).
1078
- 1079 159. Macaluso A and De Vito G.: Muscle strength, power and adaptations to resistance training
1080 in older people. *European Journal of Applied Physiology.* 91(4), 450-472 (2004).
1081
- 1082 160. Falvo MJ, Schilling BK, Earhart GM.: Parkinson's disease and resistive exercise: rationale,
1083 review and recommendations. *Movement Disorders.* 23(1), 1-11 (2008).
1084
- 1085 161. Dibble LE, Hale TF, Marcus RL et al.: High intensity eccentric resistance training
1086 decreases bradykinesia and improves quality of life in persons with Parkinson's disease: a
1087 preliminary study. *Parkinsonism & Related Disorders.* 15(10), 752-7 (2009).
1088

- 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