Movement Disorders
Vol. 17, No. 6, 2002, pp. 1148–1160

Research Review

The Power of Cueing to Circumvent Dopamine Deficits: A Review of Physical Therapy Treatment of Gait Disturbances in Parkinson's Disease

Tamar C. Rubinstein, MSc,[1] Nir Giladi, MD,[1]* and Jeffrey M. Hausdorff, PhD[1,2]

[1] *Movement Disorders Unit, Neurology Department, Tel-Aviv Sourasky Medical Center, and Sackler School of Medicine, Tel-Aviv University, Tel-Aviv, Israel*
[2] *Gerontology Division, Beth Israel Deaconess Medical Center, Boston, Massachusetts, USA*

Abstract: Gait disturbances are among the primary symptoms of Parkinson's disease (PD) and contribute significantly to a patient's loss of function and independence. Standard treatment includes antiparkinsonian drugs, primarily levodopa. In addition to the standard drug regime, physical therapy is often prescribed to help manage the disease. In recent years, there have been promising reports of physical therapy programs combined with various types of sensory cueing for PD. In this brief review of the literature, we summarize the evidence regarding the clinical efficacy of different physical therapy programs for PD, specifically with respect to improving gait. We also discuss the potential therapeutic mechanisms of sensory cueing and review the studies that have used cueing in the treatment of gait in PD. This review of the literature shows two key findings: (1) despite its relatively long history, the evidence supporting the efficacy of conventional physical therapy for treatment of gait in PD is not strong; and (2) although further investigation is needed, sensory cueing appears to be a powerful means of improving gait in PD. © 2002 Movement Disorder Society

Key words: Parkinson's disease; review; gait; physical therapy; exercise; sensory cues; visual cueing; auditory cueing

Ever since James Parkinson first described Parkinson's disease (PD) in 1817, physicians and researchers have been searching for effective treatment options. Before the introduction of levodopa in the late 1960s, physical therapy was widely considered to be a critical component in the treatment of PD. By the early 1970s, levodopa had become widely available and many believed it to be a cure for PD. This led to a decreased interest in alternative treatments, such as physical therapy. Within a few years, however, it became apparent that levodopa's efficacy does not last indefinitely, and that with extended use it can cause side effects that may even surpass the original parkinsonian symptoms. As science continues to search for a lasting cure for PD, there has been a renewed interest in nonpharmacological treatment options as adjuncts to drug therapy.

Functional neurosurgery, including pallidotomy and deep brain stimulation, shows much promise for extended relief of certain parkinsonian symptoms. Presently, however, these invasive techniques are expensive, carry a certain degree of risk, and are generally recommended only for a small subset of patients as a last resort, when all other treatment options have failed. Furthermore, although initial results are promising, the effect on gait has not been well studied.

Today, in addition to the standard drug regime, physical therapy is often prescribed by itself or with other treatment options, such as occupational therapy and speech therapy. In recent years, there have been promising reports of physical therapy programs combined

*Correspondence to: Nir Giladi, MD, Movement Disorders Unit, Department of Neurology, Tel-Aviv Sourasky Medical Center, 6 Weizmann Street, Tel-Aviv 64239, Israel. E-mail: ngiladi@tasmc.health.gov.il

Received 23 September 2001; Revised 10 February 2002; Accepted 7 March 2002

Published online 24 June 2002 in Wiley InterScience (www.interscience.wiley.com). DOI 10.1002/mds.10259

with various sensory cues. In this brief review, we summarize the evidence (or lack thereof) regarding the clinical efficacy of different physical therapy programs for PD, specifically with respect to improving gait. We also discuss the potential therapeutic mechanisms of sensory cueing and review the studies that have used cueing in the treatment of gait in PD.

We focused on the effects of physical therapy and cueing on gait because gait disturbances and falls are among the most significant motor complaints in advanced PD, and are a leading cause of loss of independence and institutionalization.[1–3] We show two key findings: (1) despite its relatively long history, the evidence supporting the use of conventional physical therapy for the treatment of gait in PD is not strong; and (2) sensory cueing appears to be a powerful means of circumventing dopamine deficits in PD and improving gait. However, despite the promise of cueing, its use outside of a research setting appears to be virtually nonexistent.

MATERIALS AND METHODS

Search Methodology

Articles were compiled for this study from a number of sources. A search of Medline (1966 to July 2001) and CINAHL (1982–2001) was performed, using the following key word combinations: [physical therapy or physiotherapy or exercise or rehabilitation] *and* [Parkinson's or Parkinsons or Parkinson]; [cue or cueing] *and* [Parkinson's or Parkinsons or Parkinson]. The Cochrane Library Database was searched using the following key word combinations: physical therapy *and* Parkinson's disease; exercise *and* Parkinson's disease; physiotherapy *and* Parkinson's disease. References from the publications identified were also examined for relevant studies. Only studies that had gait or some type of motor test that included gait as an outcome variable were considered. Articles and abstracts written in English and French were included.

Conventional Physical Therapy

The most common nonpharmacological treatment prescribed for treatment of gait disturbances in PD is physical therapy. Twelve studies were found that discussed the effects of conventional physical therapy programs,[4–15] and three were found that evaluated the effect of conventional physical therapy combined with occupational therapy.[16–18] In addition to the conventional physiotherapy regimes, a number of experimental physical therapy programs have been evaluated, such as upper body karate training[17] and treadmill training with a body-weight support system.[18] Eight of the 17 physical therapy studies examined demonstrated a significant improvement in gait, but 6 either did not find any improvement or the improvement noted did not reach statistical significance (Table 1). The remaining studies documented improvements in areas other than gait, such as global motor function and activities of daily living scores.

It has been suggested that the improvements observed in these studies may have nothing to do with the pathology of PD per se. Relatively unfit adults improve their gait and motor function in response to appropriate physical therapy training programs.[19,20] In fact, in one study in which the same exercise program was given to both PD patients and aged-matched controls, improvements in stride length and gait speed were observed in both groups.[15] Although this does not detract from the clinical benefit of such programs in PD, it is important to clarify whether a specific program is directly targeting the pathological aspects of the PD gait, or whether it is a training program that can benefit all older people, including PD patients. We will further discuss the effects of conventional physical therapy on gait in PD (see Discussion).

Sensory Cueing for the Treatment of Gait

Putative Mechanisms.

While trying to unravel the complex pathology of PD, researchers became aware of the fascinating phenomenon of sensory cueing. One of the most interesting features of PD is that, despite severe motor symptoms, patients are sometimes still able to perform complex movements almost normally under certain conditions.[21] For example, some patients will inexplicably freeze while walking through a doorway, but will have no trouble at all climbing a flight of stairs.[22] It appears that the underlying problem has more to do with motor control than with actual motor function. The challenge lies in enabling patients to access this ability at will and to use it in normal, daily function.

A summary of what is known and unknown about PD pathology is beyond the scope of this review. Nonetheless, a brief description is needed to put the studies of cueing in proper context.

The basal ganglia, the focal point of impairment in PD, have been shown to be involved in the execution of automatic and repetitive movement.[23–25] Morris and colleagues hypothesized that the basal ganglia are involved in two separate elements of motor control.[26] First, they are involved in providing phasic cues to the supplementary motor area (SMA), which is responsible for activating and deactivating each submovement within the movement sequence. Second, they are involved in the transmission of motor set information, i.e., they are responsible for accurate execution of each submovement element.

With this theory, it is possible to explain two primary gait deficiencies seen in PD.[27] First, the movement execution is not smooth because internal rhythmic cues are not being properly supplied.[23,28,29] Two of the hallmark features of PD, freezing[22,30] and festination,[31] may both be manifestations of a problem with the maintenance of an internal gait rhythm.[22] Second, because of the deficient motor set, we find abnormalities in the actual movement elements.[22,26] Thus, for example, PD gait is characterized by short, shuffling steps and decreased or absent arm swing.

Different cueing techniques have been used (see Table 2). To enhance basal ganglia function, some studies provide external rhythmic auditory cues to supplement the absent or deficient internal rhythm. Other studies use visual cues to set the proper stride length, thus providing external information to help augment the defective motor set. To reroute the movement through a nonautomatic pathway, attentional cues have been used to focus attention on walking and thus remove it from the automatic basal ganglia pathway. Alternatively, visual cues may be employed to activate the visual motor pathway instead of the automatic motor pathway.

Auditory Cueing.

Auditory cueing, in the form of rhythmic auditory stimulation (RAS), has been gaining popularity over the past 10 years. In a number of single-session studies, PD patients were able to match their cadence to a beat that was set at 10% faster than their baseline values, significantly improving their velocity, cadence, and stride length[23,32,33] (Table 2). These improvements remained evident in the immediate short term even after the cues were removed. In terms of actual gait training, patients who exercised daily while listening to music with an overlaid RAS beat showed more significant and more lasting improvements in gait than patients who did the same exercise program without RAS.[32] Apparently, the effective element of the RAS/gait training was actually the RAS, rather than the gait training per se. This finding is supported by a recent study that showed that patients who listened to music with RAS every day for a month without any gait training also showed significant improvements in their gait velocity and step length.[34]

It is not yet clear exactly how RAS is able to improve PD gait. Thaut and colleagues[32] suggest that perhaps RAS provides an external rhythm that is able to compensate for the defective internal rhythm of the basal ganglia. The finding that the improvements remained even when the cues were removed suggests that RAS may also provide some sort of rhythmic training mechanism. Proponents of this theory base their support on research that has shown that there is a basic deficit in cadence and the consistency of the timing between steps, implicating a deficit in internal rhythm mechanisms.[29,35–37] Conversely, Morris and associates found that when cadence was "fixed" using a metronome for auditory cueing, the stride length and velocity remained significantly below normal, supporting the view that rhythm (cadence) is not the problem. When stride length was "fixed" with visual cues, cadence and velocity were normal.[37] They concluded that the underlying pathology in PD gait was stride length regulation, not cadence control, and suggested that perhaps the problem with generating correct stride length was a result of a defective motor set. A later case study evaluating the kinetics of PD gait with and without visual cues observed that even when spatiotemporal and kinematic variables were normalized, an underlying pathology of gait kinetics remained.[38] This suggests that the movement disturbances in PD have a more complex cause than we are presently able to understand. Although auditory cueing appears to be quite promising, with potential long-term carryover effects, additional larger-scale studies are needed to confirm and further assess the efficacy of auditory stimulation before it can be clinically applied.

Visual Cueing.

Placement of visual cue floor markers is one technique that can be very effective in regulating stride length. Floor markers were reported to be effective in improving the gait of PD patients as early as 1967.[39] In some cases, the patient is instructed to walk over each marker, and thus achieve the desirable stride length for each step. Alternatively, the patient may be given no specific instructions regarding the floor markers. Interestingly, only certain visual stimuli are apparently effective in improving gait. Transverse lines work, whereas zigzag or parallel lines do not. In addition, the lines must be separated by an appropriate width and a color that contrasts with the floor in order to achieve the best results. In a number of single-session studies, velocity and stride length increased when PD patients were provided with floor markers to externally cue their stepping patterns.[26,37,39–41] Some of these studies found that patients retained a positive carryover effect even after the cues were removed.[26,41] This suggests that a certain degree of training took place, even with a short exposure to the cues. Visual cues have also been found to be helpful in alleviating freezing episodes. For example, carrying an inverted walking stick, so that the handle acts as a

TABLE 1. *Studies of the effects of conventional physical therapy*

Reference	Group size, n (experimental group/control)	H&Y stage, age* (yr)	Study design	Type of therapy	Duration of study (wk)	Results	Signigicant positive effect on gait**
Gibberd et al., 1981[62]	23	— W:73.1 (mean) M:68.9 (mean)	Crossover; patients were blinded	Proprioceptive neuromuscular facilitation using a combination of Bobath and Peto methods vs. infrared radiation to thorax and diversional games	4	No significant improvement found in gait posture, balance, speed, or dexterity in either group; no long-term follow-up.	No
Szekely et al., 1982[6]	7	I–III —	Longitudinal study	Family therapy and physical program	13	Significant improvement of step length and walking speed; no long-term follow-up.	Yes
Crossley et al., 1986[63]	12	— 65 (mean)	Longitudinal study	Retreat with intensive rehabilitation therapy	2	Slight but insignificant improvement in ADL and gait analysis, summary results not available; no long-term follow-up.	No
Palmer et al., 1986[17]	(7/7)	II–III 63–65	Controlled; single-blind	Stretch exercises from the United Parkinson's Foundation vs. karate exercises	12	Significant improvement in PD motor battery walk index in the karate group (−13% change). There was also improvement in both groups in motor coordination, and a subjective, patient-evaluated improvement in walking confidence. In one task involving whole-body coordination, there was a decline in function; no long-term follow-up.	Yes (karate group only)
Schenkman et al., 1989[13]	2	— 67, 68	Case studies	Exercise techniques teaching self-relaxation of rigidity and restoring normal automatic movement patterns through repetition	6	Improvements in balance, gait, and functional movements observed in both patients (from 1.04 m/second to 1.41 m/second in one patient and from 0.69 m/second to 1.24 m/second in the other patient); no long-term follow-up.	Yes
Hurwitz 1989[11]	(14/15)	I–III —	Controlled	Home exercise program	33	No significant improvement in gait variables in either group; no long-term follow-up.	No
Pederson et al., 1990[12]	10	I–III 60–73	Longitudinal study	Dynamic movements with variation in speed and adjustment to space	12	Significant *decrease* in maximum speed (from 1.63 m/second before training to 1.27 m/second after training) and stride length (from 0.89 m to 0.74 m) after therapy; this significant decrease persisted at a 4-mo follow-up evaluation.	No
Barnes et al., 1991[8]	12	II–III —	Longitudinal study	Gait training, breathing techniques, therapeutic exercises emphasizing posture and flexibility	2–3	Improvement documented in all areas of assessment, including gait speed, heel strike, and mobility skills, as well as subjective improvement in functional ability, summary results not available; this improvement persisted at 6- and 12-mo follow-up evaluations.	—
Formisano et al., 1992[10]	(16/17)	II–III 59–76	Controlled; attempts made to blind the control group	Passive and active mobilization exercises, adopted for postural control and equilibrium vs. repeated evaluation sessions	16	Significant improvement in the exercise group in gait speed (from 0.51 to 0.65 m/second); control group did not improve; no long-term follow-up.	Yes
Cedarbaum et al., 1992[16]	45	— 39–84	Longitudinal study	Physical therapy to improve range of motion, mobility, strength, and endurance; coping strategies; occupational therapy	2–11	Significant improvement was noted in maximum ambulation distance as well as in some ADL activities. Nonsignificant improvement was found in the amount of ambulatory assistance required. No long-term follow-up.	Yes

TABLE 1. (*Continued*)

Reference	Group size, n (experimental group/control)	H&Y stage, age* (yr)	Study design	Type of therapy	Duration of study (wk)	Results	Signigicant positive effect on gait**
Comella et al., 1994[9]	(8/8)	II–III —	Crossover; single-blind	69 repetitive exercises to improve range of motion, endurance, balance, gait, and motor dexterity vs. no exercise program	4	Significant improvement in the exercise group in UPDRS motor scores (from 26 to 20), (particularly scores related to bradykinesia and rigidity). There was also significant improvement in UPDRS ADL scores. No improvement was seen in the control group; the improvement in the experimental group disappeared at 6-mo follow-up.	No
Kamsma et al., 1995[5]	(25/13)	II–IV 57–78	Controlled	Alternative movement strategies for important skill domains (chair, walking and bed-related) vs. a non-specific in-group exercise program	52	No significant improvement was found in either group in UPDRS scores for gait, walking speed, or step length. However, there was an observed improvement in certain gait characteristics, such as heel strike in the experimental group; no long-term follow-up.	No
Sunvisson et al., 1997[14]	12	I–III 60–78	Longitudinal study	Daily walks in the mountains	1	Significant improvement found in functional motor assessment test; improvement remained at 3-mo follow-up, but had disappeared by the 6-mo follow-up.	Yes
Homann et al., 1998[4]	(8/7)	— —	Controlled	Bobath PT program focusing on proprioceptive skills vs. no exercise program	5	No significant difference between the two groups in the UPDRS scores. No long-term follow-up.	No
Schenkman et al., 1998[7]	(23/23)	II–III 64–78	Controlled	Individualized exercise program designed to enhance participation of appropriate synerigistic muscles, using relaxation, slow movements and gentle diaphragmatic breathing. The program includes a series of exercises divided into seven graduated stages.	10–13	No significant differences in physical performance, although the usual care group consistently performed worse than did the intervention group; no long-term follow-up.	No
Miyai et al., 2000[18]	(5/5)	II½–III ~67.6 (mean)	Crossover	Treadmill training and BWS vs. conventional physical therapy	4	Significant improvement in the BWS group in gait speed (from 1.00 m/second before treatment to 1.20 m/second after treatment). In addition, there was a significant reduction in UPDRS motor and ADL scores. No significant improvement was seen in the group receiving conventional PT; no long-term follow-up.	Yes
Scandalis et al., 2001[15]	(14/6)	II–III 48–78	Controlled (PD patients vs. healthy controls)	Rigorous resistance training-exercises predominantly geared toward the lower body, including leg presses, toe raises, leg curls and abdominal crunches	8	Significant improvement in the PD patients in stride length (from 0.83 m before treatment to 9.95 m after treatment). The healthy control group also demonstrated improvement, but this did not reach statistical significance; no long-term follow-up.	Yes

*Some studies provide only stage or only age range (in yr).

**Significant positive effect on gait is defined here as a significant effect of $P < 0.05$ for any gait variable, such as stride length or velocity, as well as for any functional assessment test that primarily examines gait, but does not include significant improvement in global motor assessment scales such as the UPDRS motor test.

H&Y, Hoehn and Yahr; ADL, activities of daily living; PD, Parkinson's disease; UPDRS, Unified Parkinson's Disease Rating Scale; BWS, body weight support.

TABLE 2. *Studies of the effect of sensory cues**

Reference	Group size, n (PD/control)	H&Y stage, age (yr)	Type of therapy	Duration of study	Results	Signigicant positive effect on gait**
Bagley et al., 1991[41]	8	— 69–88	Visual cues (brightly colored triangular rods placed along a walkway at a customized distance for each subject)	Single session	Significant improvement with cues found in stride time (from 1.32 second in the uncued trial to 1.6 second in the cued trial) and step length (from 0.41 m to 0.58 m), as well as a significant decrease in double support time (from 33% of stride to 21% of stride). Follow-up trial with no cues showed a significant carryover effect remaining in step length (0.51 m), but not in the other gait parameters.	Yes
Weissenborn 1993[52]	1	— 85	Visual cue (visual target placed just above eye level)	3 times, fortnightly	Improvement with cues found in step length (from 0.18 m in the uncued trial to 0.32 m in the cued trial) and speed (from 0.36 m/second to 0.63 m/second). No carryover effect seen when subject was not concentrating on the visual cue target.	Yes
Morris et al., 1994[40]	(15/15)	— 60–85	Instructional sets and visual cues (white strips of cardboard placed on the floor at a customized distance for each subject)	Single session	Significant improvement in stride length and speed with visual cues (alone and with instructional set). When visual cues were combined with instructions to walk fast, PD patients showed stride length and speed comparable to that of healthy controls.	Yes
Morris et al., 1994[37]	(34/34)	— >60	Visual cues (white strips of cardboard placed on the floor at a customized distance for each subject), auditory cues (metronome), and verbal instructions	Single session	Visual cues alone produced significant improvements in stride length and speed but not in cadence. Visual cues with verbal instructions produced stride length, speed, and cadence rates that were equivalent to or greater than those for healthy controls. Auditory cues produced a cadence comparable to the control group's; however, speed and stride length remained significantly below normal values. Auditory cues combined with verbal instructions produced near-normal speed; however, cadence was significantly elevated and stride length significantly decreased.	Yes
McIntosh et al., 1994[33]	6 (mean)	— —	Auditory cues (RAS)	Single session	Fast RAS produced significant improvement in mean gait speed, cadence, and stride length. Numeric details were not provided.	Yes
Azulay et al., 1996[64]	(13/7)	— —	Visual cues (transverse white stripes placed on the floor 45 cm apart)	Single session	Improvement in stride length and speed were found in 7 of the 13 PD patients, but no significant changes occurred for the overall group of patients.	No
Morris et al., 1996[26]	54	— 59–82	Visual cues (white strips of cardboard placed on the floor at a customized distance for each subject) and attentional strategies (mental picture of appropriate stride length)	Single session	Significant improvement with visual cues was found in stride length (from 0.96 m at baseline to 1.30 m with cues), speed (from 0.83 m/second to 1.20 m/second), and double support time (from 35.9% of cycle to 30.2% of cycle). Improvement using attentional cues was found in stride length (from 0.97 m at baseline to 1.32 m with cueing strategy), speed (from 0.87 m/second to 1.19 m/second), and double support time (from 35.9% of cycle to 29.9% of cycle). Follow-up trials without cues found that the imnprovements could be maintained for the maximum monitoring time of 2 hours for both visual and attentional cue strategies. However, secondary tasks and covert monitoring reduced the gait parameters to baseline values.	Yes

TABLE 2. (*Continued*)

Reference	Group size, n (PD/control)	H&Y stage, age (yr)	Type of therapy	Duration of study	Results	Signigicant positive effect on gait*
Enzensberger and Fischer 1996[65]	22	III–IV —	External rhythmic cues, including metronome stimulation, march music stimulation, and tactile stimulation (rhythmic tapping on the patient's shoulder)	Single session	Significant reduction in the time and number of steps needed for the test course. March music stimulation was less effective and tactile stimulation produced negative results.	Yes
McIntosh et al., 1997[32]	(31/10)	II–IV 67–75	Auditory cues (RAS)	Single session	Significant improvement with RAS found in stride length (from 0.86 m at baseline to 1.02 m with RAS), speed (from 0.70 to 0.95 m/second), and cadence (from 98 to 108 steps/minute). The healthy control group also showed a significant improvement in speed and cadence, but not in stride length. In a follow-up trial without RAS, these improvements persisted with a small decay rate.	Yes
Behrman et al., 1998[53]	(8/8)	II–IV 72.9 (mean)	Verbal instructional sets: 1) walk while swinging arms 2) walk and count aloud 3) walk with large steps 4) walk fast	Single session	Improvements were seen in step length and speed with all four instructional sets, for both the control group and the PD group. With instructions to walk fast, the PD group achieved step length and speed comparable to those of health controls.	Yes
Azulay et al., 1999[39]	(16/16)	II–III 62–72	Visual cues (transverse white lines placed on the floor 45 cm apart) with and without stroboscopic lighting	Single session	Under normal lighting, visual cues induced a significant improvement in gait speed (from 0.76 m/second at baseline to 0.82 m/second with visual cue) and stride length (from 0.93 to 0.97 m), but no significant improvement in cadence in PD patients. With stroboscopic illumination and visual cues, this improvement was suppressed. The healthy controls showed no significant changes in any parameters with the addition of visual cues with or without stroboscopic lighting.	Yes
Ito et al., 2000[34]	25	II½–III½ 62.3–77.7	Auditory cues (music with metronome beat in the background—RAS)	1 h a day for 3–4 wk	Significant improvement of stride length (from 0.42 m before the RAS therapy to 0.47 m after) and gait speed (from 0.83 to 0.97 m/second), but no significant improvement in cadence (from 117 to 121 steps/minute).	Yes
Lewis et al., 2000[66]	(14/14)	II–III½ 58–84	Visual cues (taped SL markers and an individualized SMLD)	Single session	Significant improvement in stride length (from 1.10 m without visual cues to 1.34 m with SL markers and 1.29 m with SMLD) and in gait speed (from 1.04 to 1.17 and 1.22 m/second). The control group did not show any significant change in any gait parameters with either SL markers or SMLD.	Yes

A study by Muller et al.[67] was not included in this table. Although visual, acoustic, and tactile cues were used, they were part of a larger framework of behavioral therapy, and thus difficult to compare with other studies that examined sensory cues alone (Table 2) or sensory cues combined with physical therapy (Table 3).

*See footnotes to Table 1.

H&Y, Hoehn and Yahr; PD, Parkinson's disease; RAS, rhythmic auditory stimulation; SL, step length; SMLD, subject-mounted light device.

horizontal cue at foot level, was able to decrease the number of freezing episodes in certain patients.[42–45]

It is not clear exactly how visual cues improve PD gait. One possibility is that visual cues help to fill in for the motor set deficiency by providing visual data on appropriate stride length.[26] When patients are told to step over each marker, they are forced to take properly sized steps, normalizing their stride length. Another theory is that visual cues help because they focus attention on gait.[26,39] Once the patient is concentrating on walking, it is no longer an automatic task that is being processed through the defective basal ganglia.

This theory is supported by the findings of Morris and coworkers.[26] They evaluated the effects of visual cues and attentional cues, i.e., visualization of the appropriate step length. They found that the two strategies produced similar improvements in walking. Similarly, visual and attentional cues acted in the same way to improve micrographia in PD.[15] Then they evaluated the carryover effects that each strategy had, evaluating patients every 15 minutes for the first 2 hours after the initial session, while interspersing increasingly more complex secondary tasks in between each evaluation and covertly evaluating the patients. They found that, for both visual cues and attentional strategies, patients were able to maintain the improvements over a 2-hour time period, as long as they were concentrating on their gait. When complex secondary tasks were interspersed between evaluations, the improvements decreased as the complexity of the tasks increased. Similarly, when the patients did not know they were being evaluated and consequently were not focusing on their stride, their gait parameters returned to baseline levels. Because the visual cues and attentional strategies behaved so similarly in terms of carryover effect and its deterioration, they conclude that visual cues operate in the same way as attentional strategies. This conclusion is supported by studies that found that PD patients have difficulty performing secondary tasks while walking. Presumably, if the "automatic" task—walking—is being routed through the cortical pathways, then a more complex secondary task, which also needs to be processed by the frontal cortex, will interfere with the optimal execution of both tasks.[41,46–48]

There are, however, problems with this explanation. If visual cues work simply by focusing attention on stride, then why do horizontal lines work, but not zigzag lines?[39] Azulay and colleagues hypothesized that perhaps visual cues work because they act as moving targets, activating the cerebellar visual-motor pathway. To evaluate this theory, they tested patients with visual cueing under two conditions: normal lighting and stroboscopic illumination. They maintain that, normally, visual floor marker cues appear to be moving downward in the patient's field of vision, but with stroboscopic lighting, this dynamic element to the cues is eliminated. They found that the visual cues were only effective in improving gait parameters under normal lighting conditions, but not with stroboscopic illumination. This favors the hypothesis that visual floor cues generate optical flow that activates a cerebellar visual–motor pathway, as opposed to the cortical–motor pathway. This hypothesis is further supported by a number of studies that found an increased dependence on visual information for control of motor activity in PD.[49–51] Furthermore, in a case study, merely looking at a visual cue marker target above eye level was sufficient to cause an increase in velocity and a normalization of the walking pattern.[52]

To further evaluate attentional cueing strategies, the effects of different verbal instructional sets on PD gait have been studied.[53] Interestingly, while different instructions were able to improve gait velocity, the effects were not equivalent. The greatest increase in velocity was seen when patients were told to walk fast. However, the resulting gait was highly abnormal, with an extremely elevated cadence and small stride length. In contrast, walking while focusing on arm swinging or large steps improved the overall velocity to a lesser degree, but resulted in an almost normal gait pattern. This finding suggests that there are a number of different mechanisms at play in the abnormal generation of gait in PD, and that different aspects of gait can be influenced by different techniques.

Physical Therapy Combined with Sensory Cueing

A number of published studies have evaluated the effect of physiotherapy or gait training combined with sensory cues (Table 3). Gauthier and colleagues initiated a program that utilized visual and auditory cues as triggers to facilitate initiation and speed of movement,[54] and Patti and associates utilized rhythmic and auditory cues to assist in continuous movement.[55] Both Patti and Gauthier and their coworkers compared their experimental group with a control group that did not receive any exercise training, and found that the experimental group showed significant improvement in gait immediately after the program, whereas the control group did not. Some of these improvements were still significant at 5- and 6-month follow-up. The other studies compared the effects of a conventional physical therapy protocol to those of the same physical therapy protocol enhanced by sensory cueing. Thaut and colleagues compared a group that undertook 3 weeks of walking with rhythmic auditory stimulation (RAS) with a control group with internally self-paced walking and a control group with no exercise

TABLE 3. *Studies of the effect of physical therapy combined with sensory cueing*

Reference	N (group size) (PD/control)	H&Y stage, age (yr)	Type of study	Type of therapy	Duration of study (wk)	Results	Signigicant positive effect on gait*
Gauthier et al., 1987[54]	(30/29)	II–IV 54–73	Controlled study with matched pairs; single-blind	General mobility, dexterity, functional and educational activities, using visual and auditory cues vs. no exercise program	10	The experimental group showed a significant improvement in gait and posture as well as in bradykinesia and akathisia. Summary values were not available. At 1 yr there was still a significant decrease in bradykinesia compared with baseline, but not in gait values.	Yes
Thaut et al., 1996[56]	(15/11/11)	II½ (mean) 61–79	Two control groups	Gait training with RAS vs. gait training with self-pacing vs. no training	3	The experimental group showed significant improvements in gait speed (from 0.81 to 0.97 m/second), stride length (from 0.99 to 1.10 m), and step cadence (from 96.8 to 105.7 steps/minute). There was no significant improvement found in either of the control groups. No long-term follow-up.	Yes
Dam et al., 1996[57]	(20/20)	II–V 61–72	Controlled; single-blind	Sensory-enhanced PT utilizing both visual and auditory cues vs. conventional PT	4 (3 cycles, with 3 mo in between cycles)	Both the experimental and control groups showed significant improvement in the GAIT subscore of the Northwestern University Disability Scale (from 5.3 at baseline to 6.5 after treatment in the sensory-enhanced PT group, and from 5.3 to 6.0 in the conventional PT group). Three mo later, the GAIT score remained elevated (5.7) for the sensory-enhanced PT group, but had returned to baseline in the conventional PT group. At the end of the study, 3 mo after the third cycle of therapy, the sensory-enhanced group still showed a significant elevation in gait score (6.1), whereas the conventional PT group had returned to the baseline score. In addition, both groups showed improvement in ADL immediately after therapy, and this improvement persisted in the sensory enhanced PT group at the 3-mo follow-up.	Yes
Patti et al., 1996[55]	(8/20)	II 43–72	Controlled; single-blind	Rhythmic symmetrical movements of increasing amplitude,[a] neck and trunk rotation exercises, practice using walking aids, breathing exercises, and occupational therapy vs. no exercise program	4	The experimental group showed a significant increase in gait speed (from 0.66 to 1.01 m/second) and in mean stride length (from 0.85 to 1.02 m). The control group had a nonsignificant decrease in both speed and stride length. Five mo after the therapy had ended, the experimental group still showed a significant elevation in speed (0.83 m/second) and a nonsignificant increase in stride length (0.95 m). At this time, the control group showed a significant decrease in both of these parameters. In addition, the experimental group showed significant improvements in the Functional Independence Measure (reflecting an improvement in ADL) which persisted (although not significantly) at the 5-mo follow-up.	Yes
Marchese et al., 2000[58]	(10/10)	I½–III 59–77	Controlled; single-blind	Physical therapy protocol (including exercises for stimulation of postural control, active or assisted limb mobilization, and exercises for articulation and pendular movement in various positions) plus visual, auditory, and tactile cues vs. physical therapy alone	6	Both the experimental and control group showed significant reduction in scores on the motor subsection of the UPDRS. The experimental group had a mean decrease of 3.4, and the control group showed a decrease of 1.7. At the 6-week follow-up, the experimental group still showed a significant decrease in score (−2.9), whereas the control group showed an increase in score (+2). In addition, there was a significant improvement in the ADL subsection of the UPDRS in both groups after the initial therapy, which persisted in the experimental group at the 6-week follow-up.	—

*See footnotes to Table 1.

[a]The article mentions the importance of using rhythmic movements to capitalize on the positive effects of rhythmic and auditory cues. However, the exact nature of the rhythmic and/or auditory cues actually used is not specified.

RAS, rhythmic auditory stimulation; PT, physical therapy; ADL, activities of daily living; UPDRS, Unified Parkinson's Disease Rating Scale.

at all.[56] They found that the RAS group had significant improvements in gait parameters, whereas the other two groups did not. Dam and colleagues[57] and Marches and associates[58] compared the efficacy of a conventional physiotherapy program with that of a sensory-enhanced physiotherapy program. They found that both the conventional physical therapy and the cue-enhanced physical therapy groups showed improvement immediately after training, but that the improvement was greater in the cue-enhanced group. This improvement disappeared in the conventional physical therapy group at follow-up assessments performed 6 weeks and 3 months after the therapy had ended, but persisted in the cue-enhanced group.

DISCUSSION

Research on the utility of physical therapy and cueing has clearly advanced in the past decade, although many questions remain. Considering the fact that sensory cueing, with or without physical therapy, is a noninvasive, virtually risk-free treatment option, it is remarkable that it has received so little attention when compared with far more complicated pharmacological and surgical options. This neglect is reflected in the current paucity of well-designed studies, especially those evaluating the various physical therapy regimens commonly prescribed, and the virtual absence of cueing from clinical practice.

We reviewed studies that evaluated the effects of physical therapy, cueing, and physical therapy combined with cueing on PD symptoms, specifically with respect to gait. Eight of the 17 physical therapy studies, 12 of the 13 sensory cueing studies, and 4 of 5 sensory-enhanced physical therapy studies demonstrated a significant improvement in gait.

It is difficult to extract a practical take-home message with respect to the efficacy of physical therapy (without cueing) on PD gait. The results of the "uncued" physical therapy studies are equivocal (Table 1). The physical therapy programs examined were diverse (programs ranging from strength and resistance training to daily walks in the mountains), and some studies included occupational therapy as well as physical therapy, so it is not easy to pinpoint which element of the treatment was helpful. Another problem was that no study was designed to identify optimal "dosing" (e.g., how many times per week) or intensity and length of intervention, and there was tremendous variation between different studies (ranging from daily activities for a week to bimonthly activities for a full year). Also, the outcome measures used to evaluate improvement varied widely, with virtually every group reporting improvements in a different set of parameters. Finally, there were a number of methodological problems in many of the physical therapy studies, such as small experimental group sizes, lack of strict diagnostic criteria for idiopathic PD, and improper controlling for the time of evaluation with respect to medication intake. Because many of the studies did not have a control group and were not blinded, it is also likely that at least some of the improvements observed were due to the placebo effect. Many studies also had unhomogeneous sample groups with ranges in age as large as 45 years and in disease progression as large as three full stages on the Hoehn and Yahr scale. This is particularly problematic because training capacities may be a function of disease stage. In fact, one study found that when patients were subdivided according to the stage of their PD, those in the earlier stages showed improvement with physical therapy, while those in the later stages did not.[10]

Other articles examining the existing literature have reached similarly unsatisfying conclusions. Two Cochrane reviews, one evaluating physiotherapy versus placebo or no intervention in PD and one evaluating the relative efficacy of different physiotherapy techniques in PD (including studies that dealt with gait), noted many methodological problems in the existing studies and a lack of a robust positive effect in most of the trials. They concluded that there was insufficient evidence to support or refute the efficacy of physical therapy in PD.[59,60]

A recent meta-analysis examined six studies that evaluated the effects of physical therapy on PD gait.[61] They assessed the methodological quality of each study and concluded that only three of the studies could be classified as truly experimental and the other three as quasi-experimental. The results of this meta-analysis showed a small but significant improvement in both gait speed and stride length. However, the meta-analysis included the three quasi-experimental studies and pooled interventions that evaluated physical therapy alone with those that evaluated physical therapy combined with sensory cues. Mechanisms and efficacy of the different modalities, however, may be very different.

Results of the sensory cueing studies were more consistent (Table 2), with 12 of 13 showing significant improvements in gait. Methodologically speaking, the cueing studies were generally more rigorous. Although the cueing studies had small experimental group sizes, they were generally properly controlled, with control groups matched for age and disease severity; they had strict criteria for the diagnosis of PD; and nearly all were careful to test at the same time of day in relation to levodopa intake. On the other hand, it is problematic to view all the cueing studies as involving a single intervention, since they include different types of cueing techniques (e.g., visual and auditory cues, attentional strategies, and instructional sets). Although it remains unclear which types of cues are the most effective and how exactly they work, almost every cueing study showed significant positive effects on gait.

It is difficult to compare the efficacy of physical therapy with that of sensory cueing, for a number of reasons. Firstly, the physical therapy studies were all multiple-session interventions, whereas most of the sensory cueing studies were single-session interventions. Secondly, the target measures for improvement were different, with most sensory cueing studies focusing mainly on gait and many of the conventional physiotherapy studies focusing primarily on global outcome measures of motor performance. Finally, it is difficult to isolate the impact of physical therapy alone, because the physical therapy programs were often combined with occupational or group therapy. Some of the conventional physical therapy protocols may have even inadvertently included certain techniques that utilized some form of cueing (e.g., Crossley used counting and rhythm as an aid to some of their exercises).

As cueing has become more popular, researchers have begun to incorporate different types of cueing techniques into their physical therapy programs, in the hope that the addition of cues may augment the effects of the physical therapy. Indeed, in the five studies we found that combined physical therapy with cueing (Table 3), four found significant improvements in gait variables. The other study did not directly evaluate gait, but did find significant improvements in the UPDRS motor subsection. In addition, four of five of these studies evaluated for long-term effects, and all four found that the improvements persisted for 6 weeks to 6 months after the physical therapy had ended in the groups who had cueing techniques added to their program, but not in those who had "uncued" physical therapy. In contrast, only 3 of the 17 "uncued" physical therapy studies evaluated for long-term improvement. One found that the improvement had disappeared at a 6-month evaluation,[9] one found that some improvement persisted at a 3-month follow-up evaluation but had disappeared by 6 months,[14] and one found a decline in performance that persisted at a 4-month follow-up evaluation.[12] There was no real long-term follow-up for the sensory cueing (alone) studies, as they were primarily single sessions. However, some studies checked to see if the improvements noted with cues persisted when the cues were removed. Three studies found that the improvement did persist, although with a slight decay as time passed,[26,32,41] and one case study found that the improvement did not persist at all once the cue was removed.[52] This raises the possibility that perhaps cueing is a key factor behind the improvements seen in many of these studies, particularly with respect to those improvements that persist after the physical therapy has ended.

A patient with PD recently complained to us that his health insurance refused to reimburse him for physical therapy expenses because "it does not help" patients with the disease. Our review, however, suggests otherwise. Results from cueing studies showed that certain techniques were able to produce normal walking velocity and stride length in PD patients, and a number of studies that combined physical therapy with cues demonstrated significant improvements that lasted up to 6 months after the treatment was discontinued. Nonetheless, this incident highlights the need for well-designed, larger-scale studies of the effects of physical therapy programs on PD symptoms, especially those that utilize cueing techniques.

As evidence mounts demonstrating the usefulness of various sensory cues for PD patients, the focus in research seems to be shifting away from conventional physical therapy and toward newer innovative types of therapy, including auditory and visual cueing, attentional strategies, and instructional sets combined with innovative physical therapy exercises. This trend reflects rehabilitation programs that take into account the mechanisms that underlie the motor dysfunction of PD. In order to capitalize on the potential therapeutic benefits of cueing in PD, future research should include carefully controlled physical therapy programs that use a variety of different external cueing techniques, perhaps in combination with more conventional physical therapy, to further assess efficacy, appropriate intensity, and duration, and to determine which types of cues are the most effective. In addition, more studies are needed to evaluate the effect of sensory cues on their own and to determine how to transform cueing into a tool that can be incorporated into a patient's everyday life.

Acknowledgments: This work was supported in part by the National Institutes of Health (AG14100, HD39838, and P41-RR13622) and by the Israel Ministry of Science.

REFERENCES

1. Ashburn A, Stack E, Pickering RM, Ward CD. A community-dwelling sample of people with Parkinson's disease: characteristics of fallers and non-fallers. Age Ageing 2001;30:47–52.
2. Gray P, Hildebrand K. Fall risk factors in Parkinson's disease. J Neurosci Nurs 2000;32:222–228.
3. Playfer JR. Falls and Parkinson's disease. Age Ageing 2001;30: 3–4.
4. Homann C, Crevenna R, Kojnig H, et al. Can physiotherapy improve axial symptoms in parkinsonian patients? A pilot study with the computerized movement analysis test battery Zebris. Mov Disord 1998;13:234.

5. Kamsma Y, Brouwer W, Lakke J. Training of compensational strategies for impaired gross motor skills in Parkinson's disease. Physiother Theory Pract 1995;11:209–229.
6. Szekely BC, Kosanovich NN, Sheppard W. Adjunctive treatment in Parkinson's disease: physical therapy and comprehensive group therapy. Rehabil Lit 1982;43:72–76.
7. Schenkman M, Cutson TM, Kuchibhatla M, Chandler J, Pieper CF, Ray L, Laub KC. Exercise to improve spinal flexibility and function for people with Parkinson's disease: a randomized, controlled trial. J Am Geriatr Soc 1998;46:1207–1216.
8. Barnes W, Seiz J, Kiel D, Elbe R, Manyam B. Outpatient Parkinson's disease rehabilitation program. Arch Phys Med Rehabil 1991;72:796.
9. Comella C, Stebbins G, Brown-Toms N, Goetz C. Physical therapy and Parkinson's disease: a controlled clinical trial. Neurology 1994;44:376–378.
10. Formisano R, Pratesi L, Modarelli F, Bonifati V, Meco G. Rehabilitation and Parkinson's disease. Scand J Rehabil Med 1992;24:157–160.
11. Hurwitz A. The benefit of a home exercise regimen for ambulatory Parkinson's disease patients. J Neurosci Nurs 1989;21:180–184.
12. Pederson S, Opberg B, Insulander R, Vretman M. Group training in Parkinsonism: quantitative measurements of treatment. Scand J Rehabil Med 1990;22:207–211.
13. Schenkman M, Donovan J, Tsubota J, Kluss M, Stebbins P, Butler R. Management of individuals with Parkinson's disease: rationale and case studies. Phys Ther 1989;69:944–955.
14. Sunvisson H, Lokk J, Ericson K, Winblad B, Ekman S. Changes in motor performance in persons with Parkinson's disease after exercise in a mountain area. Neurosci Nurs 1997;29:255–260.
15. Scandalis TA, Bosak A, Berliner JC, Heiman LL, Wells MR. Resistance training and gait function in patients with Parkinson's disease. Am J Phys Med 2001;80:38–43.
16. Cedarbaum J, Toy L, Silvestri M, Green-Parsons A, Harts A, McDowell F. Rehabilitation programs in the mangaement of patients with Parkinson's disease. J Neurol Rehabil 1992;6:7–19.
17. Palmer S, Mortimer J, Webster D, Bistevins R, Dickinson G. Exercise therapy for Parkinson's disease. Arch Phys Med Rehabil 1986;67:741–745.
18. Miyai I, Fujimoto Y, Ueda Y, Yamamoto H, Nozaki S, Saito T, Kang J. Treadmill training with body weight support: its effect on Parkinson's disease. Arch Phys Med Rehabil 2000;81:849–852.
19. Hausdorff J, Nelson M, Kaliton D, Layne JE, Bernstein MJ, Nuernberger A, Singh MA. Etiology and modification of gait instability in older adults: a randomized controlled trial of exercise. J Appl Physiol 2001;90:2117–2129.
20. MAcKay-Lyons M, Turnbull G. Physical therapy in Parkinson's disease. Neurology 1995;45:205.
21. Glickstein M, Stein J. Paradoxical movement in Parkinson's disease. TINS 1991;14:480–482.
22. Giladi N, McDermont M, Fahn S, Przedborski S, Jankovic J, Stern M, Tanner C; The Parkinson Study Group. Freezing of gait in Parkinson's disease. Neurology 2001;56:1712–1721.
23. Cunnington R, Iansek R, Bradshaw J, Phillips J. Movement-related potentials in Parkinson's disease: presence and predictability of temporal and spatial cues. Brain 1995;118:935–950.
24. Georgiou N, Iansek R, Bradshaw J, Phiullips J, Mattingley J, Bradshaw J. An evaluation of the role of internal cues in the pathogenesis of parkinsonian hyperkinsias. Brain 1993;116:1575–1587.
25. Morris ME, Matyas TA, Iansek R, Summers JJ. Temporal stability of gait in Parkinson's disease. Phys Ther 1996;76:763–777.
26. Morris ME, Iansek R, Matyas TA, Summers JJ. Stride length regulation in Parkinson's disease. Normalization strategies and underlying mechanisms. Brain 1996;119:551–568.
27. Giladi N, Balash Y, Hausdorff J. Gait disturbances in Parkinson's disease. Mapping the progress of Alzheimer's and Parkinson's disease. New York: Kluwer Academic/Plenum Publishers; 2001.
28. Phillips J, Martin K, Bradshaw J, Iansek R. Could bradykinesia in Parkinson's disease simply be compensation? J Neurol 1994;241:439–447.
29. Hausdorff J, Cudkowicz M, Firtion R, Wei J, Goldberger A. Gait variability and basal ganglia disorders: stride-to-stride variations of gait cycle timing in Parkinson's disease and Huntington's disease. Mov Disord 1997;13:428–437.
30. Giladi N, McMahon D, Przedborski S, Flaster E, Guillory S, Kostic V, Fahn S. Motor blocks in Parkinson's disease. Neurology 1992;42:333–339.
31. Giladi N, Shabtai H, Rozenberg E, Shabtai E. Gait festination in Parkinson's disease. Parkinsonism Relat Disord 2001;7:135–138.
32. McIntosh GC, Brown SH, Rice RR, Thaut MH. Rhythmic auditory-motor facilitation of gait patterns in patients with Parkinson's disease. J Neurol Neurosurg Psychiatry 1997;62:22–26.
33. McIntosh G, Thaut M, Rice R, Miller R. Stride frequency modulation in parkinsonian gait using rhythmic auditory stimulation. Ann Neurol 1994;36:316.
34. Ito N, Hayshi A, Lin W, Ohkoshi N, Watanabe M, Shoji S. Music therapy in Parkinson's disease: improvement of parkinsonian gait and depression with rhythmic auditory stimulation. In: Nakada T, editor. Integrated human brain science: theory, method application (music). New York: Elsevier Science; 2000. p 435–443.
35. Freeman J, Cody F, Schady W. The influence of external timing cues upon the rhythm of voluntary movements in Parkinson's disease. J Neurol Neurosurg Psychiatry 1993;56:1078–1084.
36. Blin O, Ferrandez A, Serratrice G. Quantitative analysis of gait in Parkinson patients: increased variability of stride length. J Neurol Sci 1990;103:51–54.
37. Morris ME, Iansek R, Matyas TA, Summers JJ. The pathogenesis of gait hypokinesia in Parkinson's disease. Brain 1994;117:1169–1181.
38. Morris M, McGinley J, Huxham F, Collier J, Iansek R. Constraints on the kinetic, kinematic and spatiotemporal parameters of gait in Parkinson's disease. Hum Mov Sci 1999;18:461–483.
39. Azulay J, Mesure S, Amblanrd B, Blin O, Sangla I, Pouget J. Visual control of locomotion in Parkinson's disease. Brain 1999;122:111–120.
40. Morris ME, Iansek R, Matyas TA, Summers JJ. Ability to modulate walking cadence remains intact in Parkinson's disease. J Neurol Neurosurg Psychiatry 1994;57:1532–1534.
41. Bagley S, Kelly B, Tunnicliffe N, Turnbull G, Walker J. The effect of visual cues on the gait of independently mobile Parkinson's disease patients. Physiotherapy 1991;77:415–420.
42. Dietz M, Goetz C, Stebbins G. Evaluation of a modified inverted walking stick as a treatment for Parkinsonian freezing episodes. Mov Disord 1990;5:243–247.
43. Dunne J, Hankey G, Edis R. Parkinsonism: upturned walking stick as an aid to locomotion. Arch Phys Med Rehabil 1987;68:380–381.
44. Kompoliti K, Goetz C, Leurgans S, Morrissey M, Siegel I. "On" freezing in Parkinson's disease: resistance to visual cue walking devices. Mov Disord 2000;15:309–312.
45. Nieuwboer A, Feys P, De Weerdt W, Dom R. Is using a cue the clue to the treatment of freezing in Parkinson's disease? Physiother Res Int 1997;2:125–132.
46. Bond J, Morris M. Goal-directed secondary motor tasks: their effects on gait in subjects with Parkinson's disease. Arch Phys Med Rehabil 2000;81:110–115.
47. Benecke R, Rothwell J, Dick J, Day B. Performance of simultaneous movements in patients with Parkinson's disease. Brain 1986;109:739–757.
48. Brown R, Marsden C. Dual task performance and processing resources in normal subjects and patients with Parkinson's disease. Brain 1991;114:215–231.
49. Cooke J, Brown J, Brooks V. Increased dependence on visual information for movement control in patients with Parkinson's disease. Can J Neurol Sci 1978;413–415.
50. Praamstra P, Stegeman DF, Cools AR, Horstink MW. Reliance on external cues for movement initiation in Parkinson's disease. Evi-

dence from movement-related potentials. Brain 1998;121:167–177.
51. Montgomery EB Jr, Nuessen J, Gorman DS. Reaction time and movement velocity abnormalities in Parkinson's disease under different task conditions. Neurology 1991;41:1476–1481.
52. Weissenborn S. The effect of using a two-step verbal cue to a visual target above eye level on the Parkinsonian gait: a case study. Physiotherapy 1993;79:26–31.
53. Behrman A, Teitelbaum P, Cauraugh J. Verbal instructional sets to normalize the temporal and spatial gait variables in Parkinson's disease. J Neurol Neurosurg Psychiatry 1998;65:580–582.
54. Gauthier L, Dalziel S, Gauthier S. The benefits of group occupational therapy for patients with Parkinson's disease. Am J Occup Ther 1987;41:360–365.
55. Patti F, Reggio A, Nicoletti F, Sellaroli T, Deinite G, Nicoletti F. Effects of rehabilitation therapy on parkinsonians' disability and functional independence. J Neurol Rehabil 1996;10:223–231.
56. Thaut MH, McIntosh GC, Rice RR, Miller RA, Rathbun J, Brault JM. Rhythmic auditory stimulation in gait training for Parkinson's disease patients. Mov Disord 1996;11:193–200.
57. Dam M, Tonin P, Casson S, Bracco F, Piron L, Pizzolato G, Battistin L. Effects of conventional and sensory-enhanced physiotherapy on disability of Parkinson's disease patients. Adv Neurol 1996;69:551–555.
58. Marchese R, Diverio M, Zucchi F, Lentino C, Abbruzzese G. The role of sensory cues in the rehabilitation of parkinsonian patients: a comparison of two physical therapy protocols. Mov Disord 2000;15:879–883.
59. Deane KH, Jones D, Playford ED, Ben Shlomo Y, Clarke CE. Physiotherapy for patients with Parkinson's disease (Cochrane review). Cochrane Database Syst Rev 2001;3:CD002817.
60. Deane KH, Jones D, Ellis-Hill C, Clarke CE, Playford ED, Ben Shlomo Y. A comparison of physiotherapy techniques for patients with Parkinson's disease. Cochrane Database Syst Rev 2001;1:CD002815.
61. de Goede CJ, Keus SH, Kwakkel G, Wagenaar RC. The effects of physical therapy in Parkinson's disease: a research synthesis. Arch Phys Med Rehabil 2001;82:509–515.
62. Gibberd F, Page N, Spencer K, Kinnear E, Hawksworth J. Controlled trial of physiotherapy and occupational therapy for Parkinson's disease. Br Med J 1981;282:1196.
63. Crossley S. Intensive physiotherapy for Parkinson's disease in a holiday environment. Physiotherapy 1986;72:383–384.
64. Azulay JP, Van Den BC, Mestre D, Blin O, Sangla I, Pouget J, Serratrice G. [Automatic motion analysis of gait in patients with Parkinson disease: effects of levodopa and visual stimulations][article in French]. Rev Neurol (Paris) 1996;152:128–134.
65. Enzensberger W, Fischer PA. Metronome in Parkinson's disease. Lancet 1996;347:1337.
66. Lewis GN, Byblow WD, Walt SE. Stride length regulation in Parkinson's disease: the use of extrinsic, visual cues. Brain 2000;123:2077–2090.
67. Muller V, Mohr B, Rosin R, Pulvermuller F, Muller F, Birbaumer N. Short-term effects of behavioral treatment on movement initiation and postural control in Parkinson's disease: a controlled clinical study. Mov Disord 1997;12:306–314.