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RESEARCH NOTE

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Training BIG to move faster: the application of the speed–amplitude relation as a rehabilitation strategy for people with Parkinson's disease

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Abstract We have used the phenomenon that speed increases with movement amplitude as a rehabilitation strategy. We tested the hypothesis that the generalized training of amplitude in the limb motor system may reduce bradykinesia and hypokinesia in the upper and lower limbs in subjects with Parkinson's disease (PD) across disease severity (Stage I, $n=6$; Stage II, $n=7$; Stage III, $n=5$). While studies have separately examined the relationship of amplitude to speed in reaching and gait, the same study has not reported the relationship for both limb systems. Moreover, the rehabilitation intervention, Training BIG, is unique in that it applies well-established treatment concepts from a proven treatment for the speech motor system in PD [Lee Silverman Voice Treatment (LSVT®)] to the limb motor system. Subjects ($n=18$) participated in intense practice (1-h sessions/4× week/4 weeks) of large amplitude movements involving the whole body (i.e., head, arm, trunk, and leg) while focusing on the sensory awareness of "movement bigness." Testing procedures were designed to demonstrate the transfer of generalized amplitude practice to speed improvements during functional "untrained" tasks in "uncued" conditions with blinded testers. After therapy, the subjects significantly increased their speed of reaching and gait for the preferred speed condition. This effect was greater when the severity of the disease was less. The results support further application and efficacy studies of Training BIG. Amplitude-based behavioral intervention in people with PD appears to be a simple target that may be applied in different contexts for multiple tasks and results in improved speed–amplitude scaling relations across the upper and lower limbs.

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Introduction

The ability to generate movements varying in amplitude, speed, accuracy, and load is critical for adapting to natural environments. Studies have systematically manipulated these task variables in order to identify rules that govern the control of movement in different contexts. One of these rules, first proposed to describe the relation between speed and amplitude for fast single joint movements (Freund and Budingen 1978), states that larger amplitude movements are generated with increased movement speed. This linear speed–amplitude relation has been shown to generalize across distal and proximal arm joints, upper and lower extremities, and single and multijoint movements (Buneo et al. 1994; Hoffman and Strick 1986; Pfann et al. 1998). While most of these studies investigated point-to-point reaching, the speed–amplitude relation has also been described during repetitive tasks including handwriting, walking, and speech movements, suggesting that this phenomenon is generalized across different motor systems (Ostry et al. 1987; Van Gemmert et al. 2003; Zijlstra et al. 1995).

Studies investigating speed–amplitude relations in people with Parkinson's disease (PD) suggest that for any given movement amplitude, the velocity is reduced (Morris et al. 1994; Pfann et al. 2001; Van Gemmert et al. 2003). The reduction in speed has been shown to be greater for long movements (large amplitude) than short movements (small amplitude), suggesting that the velocity may saturate or disproportionately scale at large amplitudes (Flowers 1975; Horak et al. 1996). Task-specific external cueing or attentional strategies have been shown to help people with PD to overcome deficits in speed or amplitude scaling. During tasks where both amplitude and speed are free to covary, amplitude cues result in both bigger and faster movements that often approach or surpass control values (walking, Behrman et al. 1998; Morris et al. 1994; handwriting, Teulings and Stelmach 1991). On the other hand, velocity cues for the same type of tasks result in mostly faster movements (but not bigger) (Behrman et al. 1998; Morris et al. 1994; Teulings and Stelmach 1991; Suteerawattananon et al. 2004; Zijlstra et al. 1998). During upper limb reaching tasks, target distance (amplitude) is typically specified, leaving only speed free to vary. In this case, velocity cues often result in subjects with PD moving as fast as control subjects, but at the loss of interlimb coordination and/or accuracy (Ma et al. 2004; Phillips et al. 1994; Sheridan and Flowers 1990; Teasdale et al. 1990). Similar studies of the speech motor system show that cueing loudness (amplitude) increases amplitude of lip movements during speech, while speed (rate) manipulations result in greater variability (Kleinow et al. 2001). These studies suggest that cueing amplitude may be the best behavioral strategy to optimize the speed–amplitude relation across multiple tasks (reaching/walking/speaking) in people with PD, thereby, reducing the symptoms of hypokinesia and bradykinesia.

Training of amplitude, as a rehabilitation approach in people with PD, was first applied to treat the speech deficit of reduced loudness (LSVT®). After over 15 years of efficacy research, LSVT has demonstrated short and long-term (2-year) retention in loudness (Ramig et al. 2001), as well as generalized improvements in articulation, facial expression, swallowing, and communicative gesturing (Fox et al. 2002). Given the success of LSVT and the general nature of the speed–amplitude phenomenon, a logical extension is the training of amplitude for the limb motor system (BIG). Thus, using the same treatment principles established in speech for training of amplitude in the speech motor system (LSVT®-LOUD), we developed a protocol for training of amplitude in the limb motor system (Training BIG). The treatment principles (multiple repetitions, intensity, and complexity) are consistent with the literature citing key elements of exercise that contribute to neuroplasticity and brain reorganization in animal models of PD (Fisher et al. 2004) and human stroke-related hemiparesis (Taub 2004). We tested the hypothesis that the generalized training of large amplitude movements involving the whole body (i.e., head, trunk, arms, and legs) would concurrently impact both the amplitude and speed of functional and more isolated upper and lower limb tasks (reaching, walking) for both the preferred and as fast as possible speed conditions.

Materials and methods

Subjects

Eighteen subjects with PD (67 ± 9 years; 11 men:7 women) volunteered to participate in the study. All the participants signed an informed consent and the University of Arizona institutional review board approved the study. Subjects were eligible if they had no medical complications that would interfere with limb movements or exercise. In addition, subjects diagnosed with PD by a neurologist were required to be stable on medications, have no other neurological diagnosis, and had never participated in LSVT®. While participating in the study, medications for subjects with PD were unchanged and the subjects were required to abstain from physical or occupational therapy and weight training. Disease severity (Hoehn and Yahr 1967) was equally distributed among subjects with PD, Stage I ($n = 6$), Stage II ($n = 7$), and Stage III ($n = 5$).

Treatment intervention

The amplitude-based intervention (Training BIG) was provided by BGF, a physical therapist who did not participate in any aspects of data collection. All the subjects received 16 individual 1-h therapy sessions (4×/week for 4 weeks). Fifty percent of every session was spent performing multiple repetitions (minimum 10) of standardized whole-body maximal amplitude drills including sustained (10 s) BIG stretches (i.e., reach and twist to side in sitting and standing) and repetitive BIG multidirectional movements (i.e., step and reach sideward, step and reach forward). The subjects were required to perform maximal bigness and attend to the perceptual feedback (i.e., how big did that feel?). The remainder of every session involved using their "bigness effort" established in the amplitude drills, to practice emotionally salient functional tasks chosen by the subject (i.e., getting out of bed, putting on socks, getting out of a chair).

Experimental conditions and procedures

Blinded examiners tested subjects with PD the week before (PRE) and the week after training (POST) at their optimal period of medication. During testing, the subjects were "uncued," that is, no reference to "bigness" was suggested during walking or reaching procedures. For gait analysis, the subjects walked along a 14-foot electronic mat (GAITRite®, CIR Systems Inc., Clifton, NJ, USA) that generated the dependent variables, velocity, cadence, and stride length. The subjects performed multijoint point-to-point reaching in the horizontal plane while sitting at a table in a standardized initial position (refer to Fig. 1a, Farley et al. 2004). They reached for a weighted cup located at three distances from the index finger, arm's length (AL) and 10 cm shorter/longer than AL (AL ± 10 cm). A reflective marker was placed on the wrist of the most impaired arm. The wrist marker data was videotaped (60 Hz) and digitized offline (Peak Performance Technologies Inc., Centennial, CO, USA). Wrist position (x, y) was smoothed with a fourth-order Butterworth filter and used to calculate peak wrist linear velocity.

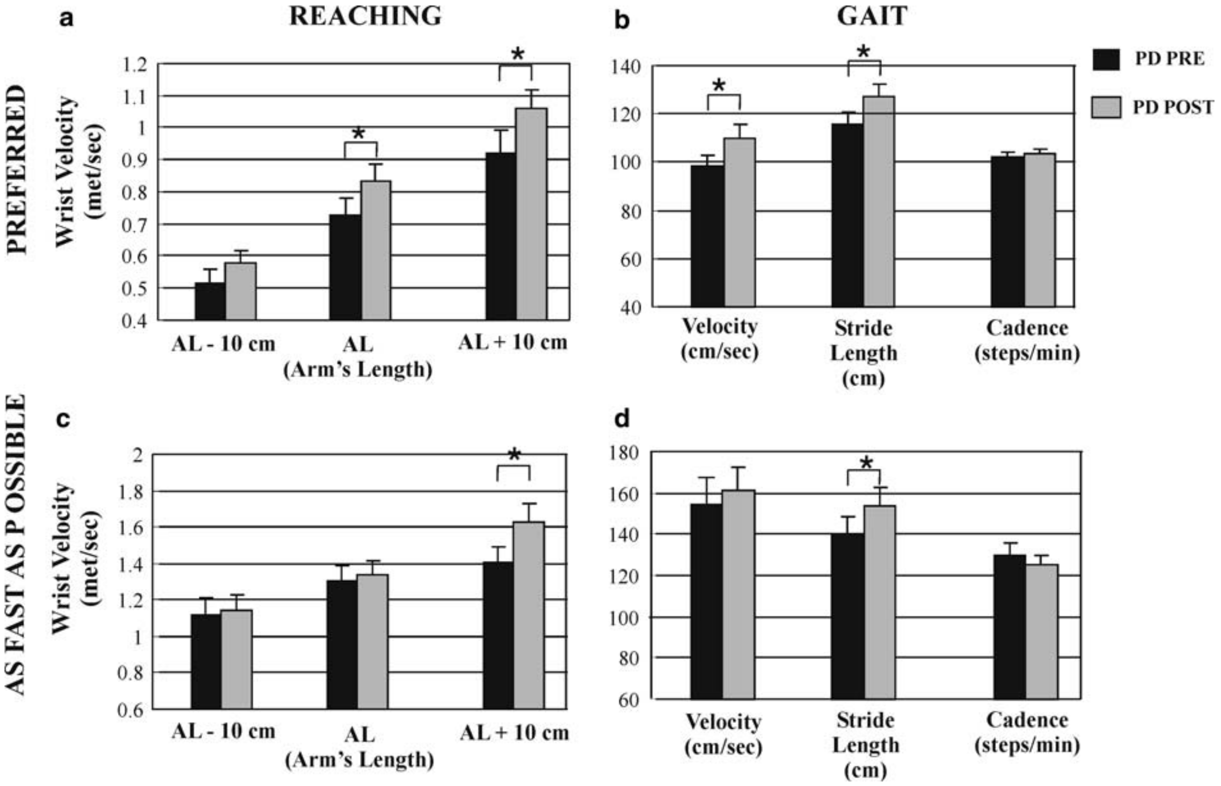

Fig. 1 **a**–**d** Averaged data (mean/SE) for reaching (*left column*, **a**, **b**) and gait (*right column*, **c**, **d**). Data from the preferred speed condition are on the *top row* and from the as fast as possible speed condition on the *bottom row*. * indicates a significance level of $P \leq 0.05$. For reaching, distance reached is on the *X*-axis and is relative to arm's length (AL). Reaching velocity on the *Y*-axis is in meters (m/s). For gait, units for velocity, cadence, and stride length are noted below each variable on the *X*-axis

Speed instructions were varied for both the gait and reaching such that subjects selected the speed (i.e., use your everyday way—called preferred condition) or the speed was explicitly controlled (i.e., as fast as possible). Prior to all testing, the subjects practiced three to seven trials. The data were averaged (two trials/each reaching distance, two to three trials/each gait condition) and were analyzed with nonparametric tests due to asymetrical distributions. The Wilcoxon matched-pairs signed-rank test was used for all within group comparisons (pre vs. post-intervention). A random effects regression model was used for the analysis of the slope and intercept values to describe the speed–distance relation. The results are reported for a significance level of $P < 0.05$.

Results

Reaching

Prior to intervention, the subjects with PD scaled speed with amplitude as evident by the increasing wrist velocity values for reaching at the three distances, AL and AL ± 10 cm (Fig. 1a, c). This linear relationship was significant for both the preferred ($P < 0.0001$) and the as fast possible ($P < 0.0005$) speed conditions. For each variable, the data are always shown in the same order, PD PRE on the left (black) and PD POST on the right (gray). In the preferred speed condition (Fig. 1a), subjects with PD (PD POST) increased wrist velocity for all the three target distances after intervention. The increase was significant for the longest two distances (AL, $P = 0.03$, AL + 10 cm, $P = 0.04$). Linear regression revealed no differences in the rate of rise in velocity (slopes) before versus after intervention. The average increase in preferred wrist velocity at the two longest distances was 14%, and appears to be treatment-related, as it was almost 3× more than the slight 5% increase observed in an untreated PD control group, tested before and after a 1-month period of no therapeutic intervention ($n = 11$, data not shown). For the as fast as possible speed condition, wrist velocity in subjects with PD significantly increased only for the longest distance by 16% (AL + 10 cm, $P = 0.009$), compared to a 5% increase in the untreated PD control group. As a result, the rate of rise (slope) increased significantly after intervention ($P = 0.01$). To summarize, for both the speed conditions, reaching velocity increased for the longest distances after Training BIG.

Gait

In the preferred speed condition (Fig. 1b), the subjects with PD after intervention (PD POST, gray) increased their gait velocity ($P = 0.01$) by increasing the stride

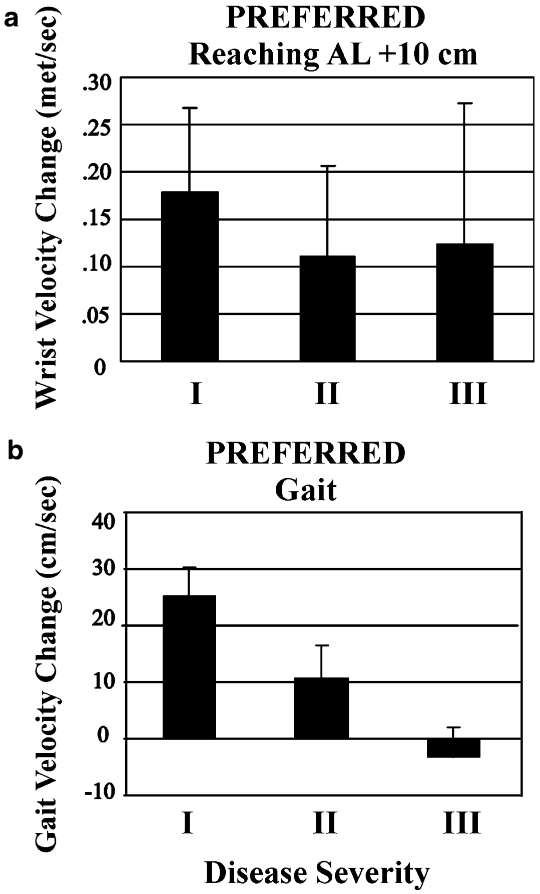

Fig. 2 a–b Absolute velocity change score (post-pre) versus disease severity (Hoehn and Yahr Scale). **a** Reaching velocity change score for preferred speed condition for the largest distance (AL + 10 cm). **b** Gait velocity change score for preferred speed condition

length ($P < 0.004$), but did not change cadence ($P = 0.91$). The 12% increase in the preferred gait velocity was 3× more than the 4% change observed in the untreated PD control group. In the as fast as possible speed condition (Fig. 1d), the subjects with PD increased their amplitude (stride length) (PRE/POST, $P = 0.02$), resulting in a slight increase in gait velocity and a decrease in cadence. To summarize, in the preferred speed condition, the subjects with PD increased both gait velocity and stride length after Training BIG. In the as fast as possible speed condition, the subjects with PD changed their strategy to take larger steps without a concomitant increase in velocity or cadence. This strategy of bigger but not faster may represent a ceiling effect for velocity, as studies have previously shown that subjects with PD are capable of near normal performance in externally cued or maximal performance type conditions (Behrman 1998; Morris et al. 1994).

Disease severity

To determine if Training BIG had differential or equivalent effects across levels of disease severity, the subjects were grouped according to their Hoehn and Yahr category (Fig. 2). Absolute change in velocity was calculated and is represented on the *y*-axis. Only data for the preferred speed conditions are shown in Fig. 2, because significant changes from Fig. 1 were most consistent in this condition for both reaching and gait, and the preferred speed represents the most uncued (spontaneous) condition. During reaching to the longest distance (AL + 10 cm) as shown in Fig. 2a, the subjects at all the three impairment levels (I, II, III) showed substantial changes in velocity. However, the subjects with milder impairment (Stage I) tended to make more improvement than the subjects with moderate impairment (Stage II and III). This trend was stronger for the change in gait velocity (Fig. 2b). In this case, there was an inverse relationship of the degree of improvement with the level of disease severity, and the data demonstrated a significant linear correlation ($P = 0.004$). Overall, it appears that the subjects with PD in Stage III, and possibly Stage II, were limited in their capacity to spontaneously generate increased velocity.

A more equal distribution of change during reaching as compared to gait may be partially explained by greater baseline impairment in the upper limb function ($P < 0.0001$) as revealed in a post hoc analysis of items specific for the upper (20, 21, 22, 23, 24, 25) or lower (20, 22, 26, 27, 29, 30) limb function on the Motor Section of the Unified Parkinson's Disease Rating Scale. However, this does not explain why the change in gait velocity was greater in mildly impaired subjects. One possible explanation may be that for the "uncued" transfer testing in this study, PD subjects with mild impairment are more spontaneously able to use their new control strategy (Think BIG), while PD subjects with moderate impairment may require ongoing or sporadic cueing to transfer their "bigness" to novel environments.

Discussion

The most significant finding of this study is that a single focus on generalized training of amplitude in people with PD resulted in faster upper and lower limb movements (decreased bradykinesia). Velocity improvements were most significant for the preferred speed condition for both the discrete (point-to-point reaching) and rhythmical (walking) tasks. During reaching, velocity improvements occurred for the largest distances, where velocity requirements are the greatest in both the preferred and as fast as possible speed conditions. During walking in the preferred speed condition, velocity improvements occurred by an increase in stride length and not cadence. In contrast, during walking in the as fast as possible speed condition, only the stride length increased without a concomitant increase in velocity.

These velocity improvements are even more remarkable given that PD subjects were tested at the peak of their medication cycle and were tested without cues. That is, testers gave no hints throughout the testing session to focus on bigness (the training cue), and the tasks were different from what the subjects practiced during their training sessions. In contrast, other studies have used a cue to improve one specific task and then retested subjects on the same task, with and without the cue (i.e., walk with long steps to improve gait, Lehman et al. 2005; take large steps to maintain balance, Jobges et al. 2004). Thus, even though they reported improvements in the cued and uncued conditions, the subjects were not required to extrapolate their ''long steps'' to perform in a novel uncued condition.

The concurrent increase in the amplitude of reaching and gait of this study could most likely be explained by the increased muscle activation (height and duration) to meet the force requirements for increased speed and distance (Brown and Cooke 1981). Several changes in muscle activation are associated with impaired speed–distance scaling in PD, including inadequate burst duration, reduced scaling of burst amplitude, magnitude saturation, and temporal overlap of agonist and antagonist activity (Berardelli et al. 1986; Farley et al. 2004; Hallett and Khoshbin 1980; Pfann et al. 2001). These deficits are progressive, but it is not known if the type of cue (amplitude vs. velocity), the amount of training, or the timing of training after diagnosis have a differential effect on the progressive features of muscle activation. In the speech motor system, a case study of two individuals with PD showed increased muscle activation in the laryngeal muscles after amplitude-based training (think loud) (Ramig et al. 2000). Similarly, for the limb motor system, the subjects with PD increase ankle muscle activation when walking with visual cues (Lewis et al. 2000). Future studies will examine the changes in EMG burst characteristics that may arise after Training Big.

The other major finding of this study was that improvements in amplitude and speed differed across disease severity, and the greatest improvement occurred in PD subjects with mild impairment (Stage I). While most physical therapy efficacy studies have not included Stage I subjects or subdivided subjects by disease severity, one study did report a similar finding (Formisano et al. 1992), suggesting that training capacity may be a function of disease severity. In the case of Training BIG, it is possible that mildly impaired subjects may be able to make use of spontaneous, less effortful mechanisms, as has been shown for Training LOUD/LSVT (PET imaging, Liotti et al. 2003) and hence, they could perform well on the uncued tasks of this study. On the other hand, moderately impaired subjects (Stage II and III) may need to consciously attend to bigness and require explicit cues, and hence their improvement in the uncued tasks of this study was limited. Future studies will focus on the differences in the allocation of attentional resources for maintaining bigness during dual task paradigms as well as test subjects performance on both ''over-trained'' and novel transfer tasks.

This study showed that mildly impaired subjects with PD have the potential for bigger and faster movements; yet, they do not use their full capacity to make normal amplitude/velocity movements in everyday situations. This pattern of early nonuse is especially relevant as recent research in animal models of PD (6-OHDA-lesioned rats, MPTP-lesioned mice) has shown that inactivity may actually contribute to degeneration (Tillerson et al. 2002) and that the continuous practice and forced use of impaired limbs prevent and/or reverse motor impairments (Tillerson et al. 2001; Fisher et al. 2004). Future studies will be needed for people with PD to determine if early training to maximize and sustain a person's speed–amplitude scaling potential may slow disease progression and reveal neurochemical correlates of improved motor functioning.

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References

Behrman AL, Teitelbaum P, Cauraugh JH (1998) Verbal instructional sets to normalize the temporal and spatial gait variables in Parkinson's disease. J Neurol Neurosurg Psychiatry 65:580–582

Berardelli A, Dick JPR, Rothwell JC, Day BL, Marsden CD (1986) Scaling of the amplitude of the first agonist EMG burst during rapid wrist movements in patients with Parkinson's disease. J Neurol Neurosurg Psychiatry 49:1273–1279

Brown SHC, Cooke JD (1981) Amplitude- and instruction-dependent modulation of movement-related electromyogram activity in humans. J Physiol 316:97–107

Buneo CA, Soechting JF, Flanders M (1994) Muscle activation patterns for reaching: the representation of distance and time. J Neurophys 71(4):1546–1558

Farley BG, Sherman S, Koshland GF (2004) Shoulder muscle activity in Parkinson's disease during multijoint arm movements across a range of speeds. Exp Brain Res 154:160–175

Fisher B, Petzinger G, Nixon K, Hogg E, Bremmer S, Meshul C, Jakowec M (2004) Exercise-induced behavioral recovery and neuroplasticity in the 1-methyl-4-phenyl 1,2,3,6-tetrahydropyridine-lesioned mouse basal ganglia. J Neurosci Res 77:378–390

Flowers K (1975) Ballistic and corrective movements on an aiming task. Neurology 25:413–421

Formisano R, Pratesi L, Modarelli F, Bonifati V, Meco G (1992) Rehabilitation and Parkinson's disease. Scand J Rehabil Med 24:157–160

Fox C, Morrison C, Ramig L (2002) Current perspectives on the Lee Silverman Voice Treatment (LSVT) for individuals with idiopathic Parkinson disease. Am J Speech Lang Pathol 11:111–123

Freund HJ, Budingen HJ (1978) The relationship between speed and amplitude of the fastest voluntary contractions of human arm muscles. Exp Brain Res 31:1–12

Hallett M, Khoshbin S (1980) A physiological mechanism of bradykinesia. Brain 103:301–314

Hoehn MM, Yahr MD (1967) Parkinsonism: onset, progression, and mortality. Neurology 17:427–442

Hoffman DS, Strick PL (1986) Step-tracking movements of the wrist in humans. I. Kinematic analysis. J Neurosci 6(11):3309–3318

Horak FB, Frank J, Nutt J (1996) Effects of dopamine on postural control in Parkinsonian subjects: scaling, set, and tone. J Neurophysiol 75(6):2380–2396

Jobges M, Heuschkel G, Pretzel C, Illhardt C, Renner C, Hummelsheim (2004) Repetitive training of compensatory steps: a therapeutic approach for postural instability in Parkinson's disease. J Neurol Neurosurg Psychiatry 75:1682–1687

Kleinow J, Smith A, Ramig L (2001) Speech motor stability in IDP: effects of rate and loudness manipulations. J Speech Lang Hear Res 44:1041–1051

Lehman DA, Toole T, Lofald D, Hirsch MA (2005) Training with verbal instructional cues results in near-term improvement of gait in people with Parkinson disease. J Neurol Phys Ther 29(1):2–8

Lewis GN, Byblow WD, Walt SE (2000) Stride length regulation in Parkinson's disease: the use of extrinsic, visual cues. Brain 123:2077–2090

Liotti M, Vogel D, Ramig L, New P, Cook C, Fox P (2003) Functional reorganization of speech-motor function in Parkinson disease following LSVT®: a PET study. Neurology 60:432–440

Ma HI, Trombly CA, Wagenaar RC, Tickle-Degnen L (2004) Effect of one single auditory cue on movement kinematics in patients with Parkinson's disease. Am J Phys Med Rehabil 83:530–536

Morris ME, Iansek R, Matyas TA, Summers JJ (1994) The pathogenesis of gait hypokinesia in Parkinson's disease. Brain 117:1161–1182

Ostry DJ, Cooke JD, Munhall KG (1987) Velocity curves of human arm and speech movements. Exp Brain Res 68(1):37–46

Pfann KD, Hoffman DS, Gottlieb GL, Strick PL, Corcos DM (1998) Common principles underlying the control of rapid, single degree-of-freedom movements at different joints. Exp Brain Res 118:35–51

Pfann KD, Buchman AS, Comella CL, Corcos DM (2001) Control of movement distance in Parkinson's disease. Move Disord 16(6):1048–1065

Phillips JG, Martin KE, Bradshaw JL, Iansek R (1994) Could bradykinesia in Parkinson's disease simply be compensation? J Neurol 241:439–447

Ramig L, Sapir S, Baker K, Hinds S, Spielman J, Brisbie A, Stathopoulos E, El-Sharkawi A, Logemann J, Fox C, Johnson A, Borod J, Luschei E, Smith M (2000) The "big picture" on the role of phonation in the treatment of individuals with motor speech disorders: or "what's up with loud?" Paper presented at the Motor Speech Conference, San Antonio

Ramig L, Sapir S, Countryman S, Pawlas A, O'Brien C, Hoehn M, Thompson L (2001) Intensive voice treatment (LSVT) for individuals with Parkinson disease": a two-year follow-up. J Neurol Neurosurg Psychiatry 71:493–498

Sheridan MR, Flowers KA (1990) Movement variability and bradykinesia in Parkinson's disease. Brain 113:1149–1161

Suteerawattananon M, Morris GS, Etnyre BR, Jankovic J, Protas EJ (2004) Effects of visual and auditory cues on gait in individuals with Parkinson's disease. J Neurol Sci 219:63–69

Taub E (2004) Harnessing brain plasticity through behavioral techniques to produce new treatments in neurorehabilitation. Am Psychol 8:692–704

Teasdale N, Phillips J, Stelmach GE (1990) Temporal movement control in patients with Parkinson's disease. J Neurol Neurosurg Psychiatry 53:862–868

Teulings HLO, Stelmach GE (1991) Control of stroke amplitude, peak acceleration, and Stroke duration in Parkinsonian handwriting. Human Mov Sci 10:315–334

Tillerson J, Cohen A, Philhower J, Miller G, Zigmond M, Schallert T (2001) Forced limb-use effects on the behavioral and neurochemical effects of 6-hydroxydopamine. J Neurosci 21(12):4427–4435

Tillerson J, Cohen A, Caudle M, Zigmond M, Schallert T, Miller G (2002) Forced nonuse in unilateral Parkinsonian rats exacerbates injury. J Neurosci 22(15):6790–6799

Van Gemmert AWA, Adler CH, Stelmach GE (2003) Parkinson's disease patients undershoot target amplitude in handwriting and similar tasks. J Neurol Neurosurg Psychiatry 74:1502–1508

Zijlstra W, Rutgers AWF, Hof AL, Van Weerden TW (1995) Voluntary and involuntary adaptation of walking to temporal and spatial constraints. Gait Posture 3:13–18

Zijlstra W, Rutgers AWF, Van Weerden TW (1998) Voluntary and involuntary adaptation of gait in Parkinson's disease. Gait Posture 7:53–63