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Rhythmic Auditory Stimulation in Gait Training for Parkinson's Disease Patients

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Summary: Rhythmic auditory stimulation (RAS) was used as a pacemaker during a 3-week home-based gait-training program for Parkinson's disease (PD) patients ($n = 15$). Electromyogram (EMG) patterns and stride parameters were assessed before and after the test without RAS to evaluate changes in gait patterns. Data were compared with those of two control groups ($n = 11$), who either did not participate in any gait training or who participated in an internally self-paced training program. RAS consisted of audiotapes with metronome-pulse patterns embedded into the on/off beat structure of rhythmically accentuated instrumental music. Patients who trained with RAS significantly ($p < 0.05$) improved their gait velocity by 25%, stride length by 12%, and step cadence by 10% more than self-paced subjects who improved their velocity by 7% and no-training subjects whose velocity decreased by 7%. In the RAS-group, timing of EMG patterns changed significantly ($p < 0.05$) in the anterior tibialis and vastus lateralis muscles. Evidence for rhythmic entrainment of gait patterns was shown by the ability of the RAS group to reproduce the speed of the last training tape within a 2% margin of error without RAS. **Key Words:** Auditory rhythm—Gait training—Parkinsonism.

Gait deficits are among the most characteristic and most functionally debilitating signs of the motor neuropathology of Parkinson's disease (PD). Two of the most characteristic features of the gait profile of PD are bradykinesia and a shuffling stride pattern with shortened stride length and a reduced step cadence. Other changes include insufficient heelstrike and toe clearance, inadequate flexion about the hip, ankle, and knee, postural instability, and asymmetry between the stride times of the lower limbs (1).

Gait deficits in PD patients are often resistent to pharmacologic treatment despite the general effectiveness of dopaminergic drug therapy. In addition, prolonged drug intake may also be associated with decreased responsiveness to medication (2). Thus it is generally agreed that effective nonpharmacologic treatments need to be developed as an adjunct therapy to relieve symptoms and improve mobility (3).

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In the clinical literature, the use of sensory systems through visual or auditory cues to facilitate locomotor activity has been repeatedly mentioned as one form of nonpharmacologic treatment (4). In fact, >25 years ago, Purdon Martin (5), in his classic treatise, described the use of visual cues to facilitate gait in PD. Despite some encouraging data, however, quantitative research into the controlled application of sensory cuing to motor facilitation with PD patients has been limited.

In previous work in sensorimotor facilitation, we were able to increase cadence, stride length, and symmetry in gait patterns of PD patients using rhythmic auditory stimulation (RAS) as peripheral timekeeper in a frequency entrainment design (6). These data were in agreement with findings by Richards et al. (7) for PD patients and by Thaut et al. for healthy individuals (8) and stroke patients (9). Decreased variability in rhythmic timing of arm and finger movements of PD patients using auditory rhythm was also reported by Freeman et al. (10) and Pastor et al. (11).

Considering (a) the evidence for beneficial effects

of rhythmic cuing on motor performance, and (b) the need for effective alternative motor therapy strategies, we sought to determine in this study if possible entrainment effects of RAS on gait of PD patients could be used as an adjunct therapy strategy further to improve ambulatory function.

METHODOLOGY

Subjects

Study participants were volunteers who were randomly selected from referral lists from local PD support groups and primary care physicians. All subjects had a primary diagnosis of idiopathic PD. They all had significant gait deficits regarding velocity, stride length, and cadence (see description in Results) but were able to walk without physical assistance. The subject pool was randomly divided into an experimental group (EX) of 15 subjects (10 men and five women) and a control group, divided into a self (internally)-paced group (SPT; $n = 11$; eight men/three women) and a no-training group (NT; $n = 11$; eight men and three women).

Mean ages for the groups were EX, 69 ± 8 years; NT, 71 ± 8 years; SPT, 74 ± 3 years. Mean level of PD severity on the Hoehn and Yahr scale was 2.4 for EX, 2.6 for NT, and 2.5 for SPT. The EX group had a mean illness duration of 7.2 ± 4 years; the NT group, 8.5 ± 4 years; and the SPT group, 5.4 ± 3 years. The average number of reported falls per subject during the 12 months before the study was 4.5 for the EX group, 2.6 for the NT group, and 0.4 for the SPT group.

All patients were on a stable medication regimen (carbidopa/levodopa or selegiline/carbidopa and levodopa) during the study. No subjects were medicated with direct dopamine-receptor agonists such as bromocryptine or pergolide. Medication was monitored by a physician. All testing was done 90–120 min after first medication intake in the morning. No pronounced motor fluctuations or other cognitive, sensory, or mental health deficits were present in the study subjects.

Testing and Training

Subjects in each group participated in a pretest and posttest, 3 weeks apart, which involved measurement of their stride and electromyogram (EMG) patterns. Pretest and posttest performances were measured without the rhythmic timekeeper present. Subjects were instructed to walk at their normal speed.

During the training period, subjects in the NT group were instructed to just carry out their normal daily activities during the 3 weeks between tests. In the EX group, subjects exercised each day according to a prescribed program using RAS. The SPT group participated in the same exercise program but without the aid of RAS.

The EX subjects walked daily for 30 min with RAS. The RAS program consisted of walking on a flat surface, stair stepping, and stop-and-go exercises to rhythmically accentuated music at three different tempos. Subjects walked at each tempo for one third of the exercise time. Subjects could select from four short instrumental music pieces in four different styles familiar to the elderly age group in this study (folk, classical, jazz, country). Each selection was composed in 2/4 or 4/4 meter, and 32 measures in length. Rhythmic on-beats were enhanced by overlaying the click-function of the sequencer over the musical beat structure. We chose rhythmic stimuli embedded in a musical structure based on findings that rhythmic patterns within a musical context reduced response variability and synchronization offset more effectively than did single-pulse pattern in the frequency range of 1 to 2 Hz (60 to 120 steps/min) (12). Each selection was programmed on an eight-channel sequencer/synthesizer module in digital audio signal form (Alesys MMT8/Roland D5). Digital audiorecording allowed us to use the sequencer as a variable tempo driver to change the tempo of the music without loosing pitch control. The tempos were labeled "normal," "quick," and "fast."

For the first week of training, the normal tempo was the pretest cadence, the quick tempo was 5 to 10% faster, and the fast tempo an additional 5 to 10% faster. After each week, each tempo increased by 5 to 10%, so the quick tempo became normal, and so on. The rate of increase was based on the subjects' ability to match the tempos and the requirement to keep the fastest tempo from exceeding 130 steps/min. The subjects used portable tape players with light-weight headsets. Each musical selection was recorded for 30 min on tapes in the laboratory. The subjects exercised on their own or with spousal assistance at home or in the community.

The SPT group performed their walking sessions without RAS, following the same training protocol and training exercises for the same length of time. They were instructed to divide their exercise time into three equal periods, during which they would walk first at normal speed, then increase their speed for the next period, and increase it again for the last

period. Both the EX group and the SPT group were visited once a week by a research assistant and also were asked to keep daily logs of their walking activities to control for training compliance. Compliance was 100% for both groups. One fall was reported by one EX-group subject during the study.

Data Recording

Gait was studied through measurement of footfall patterns and EMG recordings of the medial gastrocnemius (GA), tibialis anterior (TA), and vastus lateralis (VL) muscles on both sides, averaged across five strides. Data were recorded from subjects walking at their normal speed along a 6-m walkway with 2 m on each side for acceleration and deceleration. Two trials were run; the first was a flat surface walk followed by a walk over an incline-step obstacle. The incline-step obstacle was 3 m long with a 1-m incline rising 20 cm, a 1-m platform, a 10-cm step down, another 1-m platform, and a final 10-cm step down.

Data were recorded with an IBM-compatible PC and analog-to-digital converter, surface EMG electrodes, and a computerized foot-switch recording system. Foot-switch data recorded foot contact at the heel, first and fifth metatarsal, and big toe.

Raw EMG data were digitized at 500 samples per second. The digitized EMG was high-pass filtered at 70 Hz with a zero-phase finite-impulse-response filter and then full-wave rectified with a RMS (root-mean-square) processor. Smoothing of the RMS signal values was achieved by low-pass filtering with a 100-ms Hamming window impulse response, which had a −3-dB gain at 6.6 Hz. The average RMS value was then computed at 1% increments of the total gait cycle for every stride in the trial.

EMG Analysis

EMG data were analyzed through measurement of variability, symmetry, and timing of the muscle-activation period. Variability was measured as the weighted average of the coefficient of variability (COV) at each percentage of the gait cycle (GC; 13). Bilateral symmetry was measured as the correlation coefficient between amplitude-normalized average profiles (14). Timing of muscle activation was initially assessed by computing a timing power index (duty cycle index), which reflected the duration of muscle activation by using the power of the average waveform, normalized in amplitude and shifted to a zero-mean value. After this initial analysis, changes in the period of EMG activity were analyzed further through detection of onset and termination time by a computer algorithm.

Statistics

Analysis of variance (ANOVA) procedures with post hoc multiple comparisons were used to analyze statistical differences in change scores between pretest and postest in each group. Change score analysis was used to offset possible differences of study participants at pretest and to minimize group data variability. However, to assure that change score differences were not solely due to group differences at the outset of the experiment, separate pretest and posttest ANOVAs were also computed. To account for the small sample size, nonparametric ANOVA (Kruskal–Wallis) using rank-order transformed data was performed on all parameters in addition to parametric ANOVA.

RESULTS

Because parametric and nonparametric ANOVA procedures revealed the same results, only nonparametric data are reported here as the more stringent approach to identify statistical significance. Furthermore, ANOVA comparisons of all measures at pretest found no significant ($p < 0.05$) differences between groups.

Velocity, Cadence, and Stride Length

Velocity data were computed for the flat and the inclined walks. Cadence and stride length were computed only for the flat walk. The pretest and posttest results are listed in Table 1. During pretest, all subjects showed abnormal gait patterns characteristic of PD. These included decreased velocity (mean, 45.2 m/min), shortened stride length (mean, 0.98 m), and slow cadence (mean, 92.6 steps/min). The accepted normal age-matched values reported in the literature are 73 m/min for velocity, 1.27 m for stride length, and 113 steps/min for cadence (15). All experimental subjects increased their individual gait velocity during posttest (Fig. 1). The mean increase for the EX group was 24.1% ($t = 3.84$; $p = 0.007$) in the flat walk (48.7 m/min to 58.3 m/min) and 26.1% ($t = 3.27$; $p = 0.009$) over the incline-step obstacle (40.8 to 49.4 m/min). The velocity of the NT group actually decreased slightly from pretest to posttest (42.1 to 38.7 m/min), which suggests that familiarity with the laboratory environment was not a factor in the experimental group's improvement. The SPT group did show an improvement in velocity of 7.4% (47.9 to 51.9 m/min), which

TABLE 1. *Means, standard deviations, and percentage change score means for velocity, cadence, stride length*

	Velocity (m/min)			Cadence (steps/min)			Stride length (m)		
	Pre	Post	Change (%)	Pre	Post	Change (%)	Pre	Post	Change (%)
Flat walk									
EXP									
$\bar{x}$	48.7	58.3	24.1	96.8	105.7	10.4	0.99	1.10	12.0
SD	13.6	12.6	12.3	13.5	11.5	13.5	0.19	0.17	12.8
NT									
$\bar{x}$	42.1	38.7	−7.3	88.5	96.6	5.7	0.93	10.84	−10.3
SD	15.5	16	15.8	13.4	9.6	11.9	0.24	0.32	17.9
SPT									
$\bar{x}$	47.9	51.9	7.4	92.6	92.2	−0.4	1.04	1.12	7.9
SD	7.3	12.5	10.7	7.6	8.4	1.7	0.16	0.21	9.2
Incline-step walk									
EXP									
$\bar{x}$	40.8	49.4	26.1						
SD	14	13	30.9						
NT									
$\bar{x}$	35.8	30.2	−10.5						
SD	15	13	20.9						
SPT									
$\bar{x}$	40.7	43.9	8.4						
SD	9.3	11.6	16.2						

EXP group, $n = 15$; NT and SPT groups, $n = 11$.

is less than one third of the experimental group gain. The EX group's increase in velocity was nearly equally achieved through an increase in cadence and stride length, 10.4% ($t = 2.9$; $p = 0.01$) and 12.0% ($t = 3.63$; $p = 0.009$) respectively, whereas the SPT group's increase was achieved solely through increases in stride length of 7.9%. The SPT group's cadence remained unchanged from pretest to posttest. The pretest to posttest changes in both control groups were all statistically nonsignificant.

ANOVA comparisons between groups revealed significant differences in improvement of flat and incline velocity (FLAT: $F = 13.77$; $p = 0.0001$; INCLINE: $F = 4.77$; $p = 0.0185$). Post hoc comparisons showed that the EX group improved significantly over the SPT group (FLAT: $p = 0.0307$; INCLINE: $p = 0.0347$) and the NT group (FLAT: $p = 0.0001$; INCLINE: $p = 0.0052$). Changes in stride length and cadence were also significantly different between groups (STRIDE LENGTH: $F = 4.86$; $p = 0.0144$; CADENCE: $F = 3.81$; $p =$

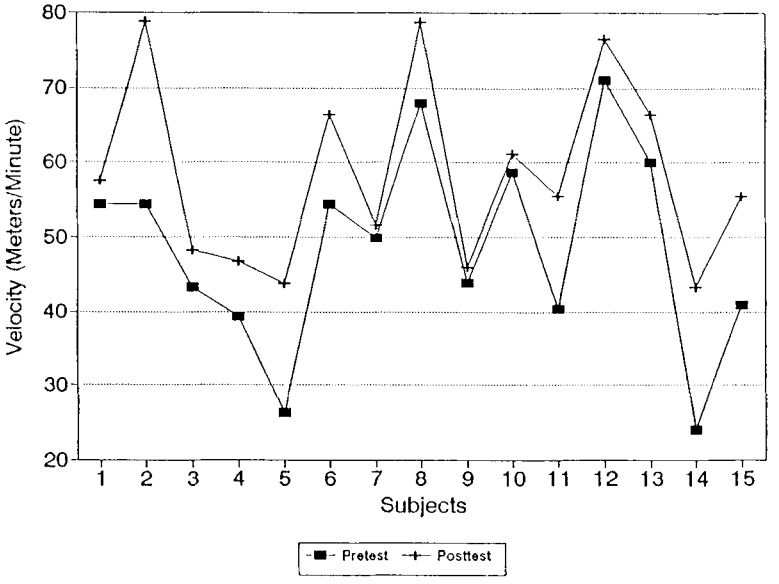

FIG. 1. Graph of gate velocity pretest (squares) and posttest (plus signs) for the 15 experimental subjects.

0.0328). However, in post hoc comparisons, the EX group's improvements in stride length were only significantly better than the NT group ($p = 0.0045$), and changes in cadence were only significantly higher than the SPT group ($p = 0.0340$).

To test for possible entrainment mechanisms between RAS-beat frequency and step frequency, subjects in the EX group were asked—after completion of the posttest—to reproduce the cadence of their fastest training tape from memory without RAS present. On average, the fastest training cadence was 18.2% faster than their normal posttest walk. The average absolute error in matching the training cadence was 5.0% (±2.9%), with the group average cadence only 1.8% (±5.6%) slower than the training tempo (Fig. 2).

The objective gains in gait performance were quite noticeable to the study subjects, who reported a 100% strong agreement on an exit questionnaire that RAS training had made their walking patterns more stable, had improved their speed, and had helped their walking in activities of daily life.

TABLE 2. *Means, standard deviations, and change score means for EMG variability (% amplitude ratio)*

	Gastrocnemius	Tibialis anterior	Vastus lateralis
Experimental group			
Pre			
$\bar{x}$	36.4	36.0	24.6
SD	12.0	10.0	5.1
Post			
$\bar{x}$	31.5	30.3	22.6
SD	9.8	10.4	7.3
Change			
$\bar{x}$	−4.9	−5.7	−2.1
SD	11.2	10.6	6.5
Control group (combined)			
Pre			
$\bar{x}$	35.0	43.1	28.7
SD	15.1	23.7	9.6
Post			
$\bar{x}$	31.6	36.6	27.2
SD	13.2	19.2	6.5
Change			
$\bar{x}$	−3.4	−6.5	−1.5
SD	8.4	10.5	5.9

EXP group, $n = 15$; CONTROL group, $n = 16$.

EMG Analysis

In each control group, three EMG records had to be rejected because of recording artifact. Thus data from the two groups were pooled to provide a sample size suitable for statistical analysis.

In Table 2, the percentage variability is listed for the GA, TA, and VL muscles. Pretest to posttest changes in the TA muscle approached statistical significance ($F = 2.29$; $p = 0.0559$), however, regardless of treatment condition. No changes in other muscles were found to be statistically significant.

EMG symmetry was examined by comparison of left and right side normalized average gait-cycle profiles in each muscle group, as listed in Table 3. In the EX group, the symmetry increased in each muscle pair. The posttest symmetry in each muscle was ~85%, which may be a practical limit due to variability in electrode placement (16). Symmetry in

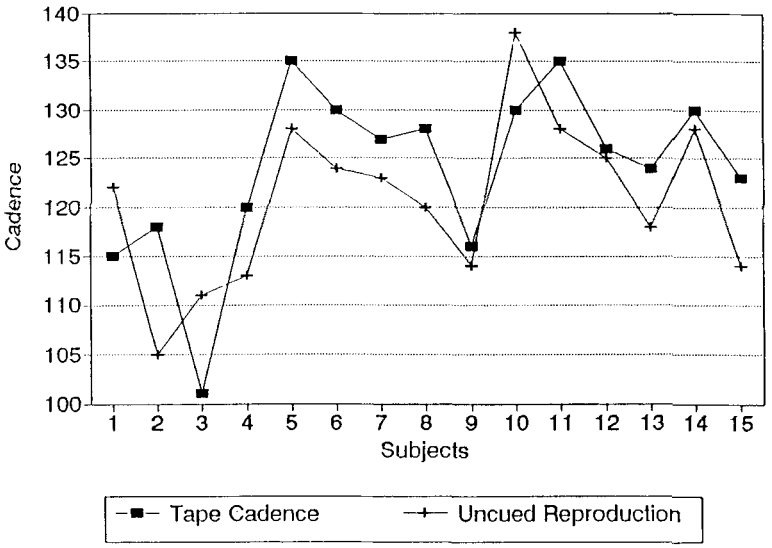

FIG. 2. Graph of maximal training tape cadence (squares) and uncued cadence reproduction (plus signs) for the 15 experimental subjects.

TABLE 3. *Means, standard deviations, and change score means for EMG symmetry (% symmetry ratio)*

	Gastrocnemius	Tibialis anterior	Vastus lateralis
Experimental group			
Pre			
$\bar{x}$	73.0	71.5	85.1
SD	24.3	16.5	9.7
Post			
$\bar{x}$	86.3	84.9	86.9
SD	8.2	7.7	9.0
Change			
$\bar{x}$	13.3	13.4	1.8
SD	21.4	15.5	13.5
Control group (combined)			
Pre			
$\bar{x}$	85.2	80.9	79.4
SD	3.4	10.4	20.9
Post			
$\bar{x}$	84.3	77.7	81.9
SD	7.4	12.3	12.3
Change			
$\bar{x}$	−0.9	−3.2	2.5
SD	6.2	15.5	14.4

EXP group, n = 15; CONTROL group, n = 16.

TABLE 4. *Means, standard deviations, and change score means for timing focus index (%)*

	Gastrocnemius	Tibialis anterior	Vastus lateralis
Experimental group			
Pre			
$\bar{x}$	107.0	82.6	77.3
SD	23.5	13.1	9.7
Post			
$\bar{x}$	100.1	88.5	86.4
SD	24.7	14.5	14.2
Change			
$\bar{x}$	−6.9	6.0	9.1
SD	14.0	12.3	13.9
Control groups (combined)			
Pre			
$\bar{x}$	97.1	92.4	97.0
SD	23.7	14.8	20.9
Post			
$\bar{x}$	101.5	89.7	90.5
SD	30.8	20.5	18.1
Change			
$\bar{x}$	4.3	−2.7	−6.4
SD	17.8	12.8	14.2

EXP group, n = 15; CONTROL group, n = 16.

the GA and TA muscle increased by 13%, the latter approaching a statistically significant difference in comparison with control group changes (F = 4.32; p = 0.0565). No other significant changes were seen in the symmetry analysis.

A timing index was computed to reflect the temporal focus of EMG activity without identifying onset and termination times. A large value would indicate EMG activity focused in a small percentage of the gait cycle, whereas a small value would reflect EMG activity spread throughout the gait cycle. The data for each muscle group are listed in Table 4. Statistically significant differences between EX and control group, indicating a more focused activation period, were seen only in the VL muscle (F = 6.43; p = 0.0220).

Pretest to posttest changes in the activation periods of the GA, TA, and VL muscles in the EX group were further investigated by determination of onset and termination times of EMG. Significant changes were observed in the durations of the VL (t = 2.59; p = 0.0303) and TA muscle (t = 2.30; p = 0.0471; Table 5). The duration of both muscle groups was shortened almost entirely by a quicker termination, ~4% GC for VL and 6% for TA. The onsets were as expected, during the preswing phase, at ~88% GC for VL and 58% for TA. The termination times were later than normal during pretest (17). TA termination is normally before the end of the loading phase, at 9% GC. Pretest TA termination was at 14% GC with posttest termination at 8% GC. The VL termination is normally in early mid-stance ~30% GC. Pretest VL termination was at 38.5%, and posttest termination, at 34.5% GC.

TABLE 5. *Means, standard deviations, and change score means for EMG onset and termination (% GC)*

Experimental group	Onset	Termination	Duration
Tibialis anterior			
Pre			
$\bar{x}$	58.7	14.4	
SD	3.4	10.5	
Post			
$\bar{x}$	57.6	8.0	
SD	3.2	7.9	
Change			
$\bar{x}$	−1.1	−6.5	−5.4
SD	2.2	6.9	7.4
Vastus lateralis			
Pre			
$\bar{x}$	87.7	38.5	
SD	4.0	6.3	
Post			
$\bar{x}$	87.7	34.5	
SD	3.5	7.7	
Change			
$\bar{x}$	0.1	−4.0	−4.0
SD	2.5	5.9	5.0

EXP group, n = 15; CONTROL group; n = 16.

DISCUSSION

Stride Parameters

The major goal of RAS training was to increase the normal gait velocity of PD patients for flat surface and incline walking. Subjects who participated in the RAS training improved on average 25% in both of these tasks. The improvement was facilitated by nearly equal percentage increases in cadence and stride length. The control group that participated in self-paced training also improved their gait velocity, but by less than one third of the improvement seen in the RAS group. This control group increased velocity solely with an increase in stride length but no apparent change in cadence. Subjects in the no-training control group showed no improvement, as would be expected from such a control condition. The difference in increase in velocity in the EX group over the SPT group appeared to be facilitated by the addition of RAS to the exercise program. In only 3 weeks, the RAS program, which was based on increasing walking tempo gradually through rhythmic auditory cuing, was effective in shifting the subjects' intrinsic gait tempo. Part of the mechanism involved with this tempo shift may have been a rhythmic entrainment effect evidenced by the subject's ability to reproduce the tempo of the musical rhythm without cuing. Although motivational factors through the music cannot be excluded as a reason for enhanced gait performance, their effects were minimized by the fact that each subject had to train with the same musical selection for 3 weeks. Repeated use of music of relatively low complexity is assumed to induce a great amount of redundancy into the perceptual process and thus strongly reduce affective arousal effects related to motivation (18). This assumption was supported by comments from the study subjects that the music seemed to get repetitive over time.

EMG Patterns

Some differences in variability, symmetry, and timing of EMG measures were observed between EX and control group data. However, the observed changes were fairly small and not consistent across muscles. Data pooling of the two control groups may have also masked EMG changes between NT and SPT subjects. However, similar trends in timing and variability of EMG patterns under the influence of auditory rhythm have been reported in previous studies (7,8,9,19,20).

The slight improvements in EMG symmetry tended to be actually more dramatic in subjects who had poor pretest symmetry, whereas subjects with good pretest symmetry generally improved little. This was illustrated by the decrease in standard deviation across the subject pool of the pretest and posttest symmetries. Pretest standard deviation values were 24.3% for GA and 16.5% for TA; posttest values were 8.2% and 7.7%, respectively.

Significant changes were also seen in the VL muscle activation period, with activity focused in a smaller time percentage of the gait cycle. A further analysis of EMG onset and termination times revealed that the VL and TA muscles had EMG onset times that corresponded to the proper phase of the gait cycle in both pretest and posttest. However, termination time showed a slight delay in the pretest trials. Biomechanical analysis would be needed to show if the mid and terminal stance periods had changed, or if the late EMG activity represented nonfunctional rigidity. Either way, at posttest, both termination times had shifted to a more normal profile.

Mechanisms

The subjects' ability to reproduce without cuing the fastest training cadence 24 h after the last training session indicates the possible effect of rhythmic entrainment mechanisms. The small error in reproduction was especially remarkable in light of previous findings that show that time estimation, recall, and reproduction in PD patients are impaired (11). Auditory rhythm may have acted as an external timekeeper clock to which the step cadence became synchronized during the training phase, thus helping to stabilize destabilized internal time keeping and rhythm formation processes in PD patients (10,11). Several patients reported pacing themselves by singing the music silently.

In summary, RAS training improved gait velocity, cadence, and stride length significantly after only 3 weeks. In addition, some features of EMG gait-cycle profiles changed toward more normal muscle-activation patterns. The data suggest a viable role for RAS as a sensorimotor-based technique for gait facilitation in PD patients.

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