

Rhythmic auditory-motor facilitation of gait patterns in patients with Parkinson's disease

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Abstract

Objectives—The effect of rhythmic auditory stimulation (RAS) on gait velocity, cadence, stride length, and symmetry was studied in 31 patients with idiopathic Parkinson's disease, 21 of them on (ON) and 10 off medication (OFF), and 10 healthy elderly subjects.

Method—Patients walked under four conditions: (1) their own maximal speed without external rhythm; (2) with the RAS beat frequency matching the baseline cadence; (3) with RAS 10% faster than the baseline cadence; (4) without rhythm to check for carry over from RAS. Gait data were recorded via a computerised foot switch system. The RAS was delivered via a 50 ms square wave tone embedded in instrumental music (Renaissance style) in 2/4 metre pre-recorded digitally on a sequencer for variable tempo reproduction. Patients on medication were tested in the morning 60–90 minutes after medication. Patients off medication were tested at the same time of day 24 hours after the last dose. Healthy elderly subjects were tested during the same time of day.

Results—Faster RAS produced significant improvement ($P<0.05$) in mean gait velocity, cadence, and stride length in all groups. Close synchronisation between rhythm and step frequency in the controls and both Parkinson's disease groups suggest evidence for rhythmic entrainment mechanisms even in the presence of basal ganglia dysfunction.

Conclusions—The results are consistent with and extend prior reports of rhythmic auditory facilitation in Parkinson's disease gait when there is mild to moderate impairment, and suggest a technique for gait rehabilitation in Parkinson's disease.

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Keywords: Parkinson's disease; gait; auditory rhythm; sensorimotor facilitation

Gait abnormalities in Parkinson's disease are characterised by bradykinesia and a shuffling gait pattern, "marche a petit pas". More specific changes in Parkinson's disease gait include decreased walking speed, shortened stride length, insufficient heelstrike and toe clearance, inadequate flexion about the hip, ankle, and knee, and asymmetric stride times for both lower limbs.^{1,2} Impaired control of

purposive limb movement resulting, in part, from postural instability and disequilibrium contribute to the overall parkinsonian profile.

Gait deficits in patients with Parkinson's disease may persist despite the general effectiveness of dopaminergic drug treatment.³ Decreased responsiveness to medication related to motor fluctuations or the onset of side effects related to prolonged or high dose drug intake necessitates the search for effective alternative or adjunct motor rehabilitation strategies in Parkinson's disease. The effectiveness of utilising sensory systems—for example, vision—to facilitate locomotor activity was first described by Martin⁴ over 25 years ago. In a later study, Forsberg *et al*⁵ reported beneficial effects of visual guidance on gait movements in patients with Parkinson's disease. Recently, Richards *et al*⁶ compared the effects of visual and auditory cues on gait patterns in patients with Parkinson's disease on and off levodopa. In that study patients walked faster with both cues. Auditory cues, however, led to increases in both stride length and cadence, by contrast with visual cues (floor markers) which resulted in longer stride length but slower cadence. In a recent study we used RAS as a three week home based training technique to increase gait velocity in patients with Parkinson's disease compared with two Parkinson's disease control groups, one without any gait training and one using a self paced gait exercise programme.⁷

In the clinical literature, recommendations for the use of rhythm and music to facilitate gait training for patients with Parkinson's disease have been mentioned on occasion.⁸ Despite some encouraging data, however, quantitative research into the controlled application of sensory cueing in motor facilitation in patients with Parkinson's disease is limited.

We report data from a study which used a frequency entrainment design to investigate the effect of rhythmic auditory stimulation (RAS) on velocity, cadence, stride length, and symmetry in gait patterns of patients with Parkinson's disease. To assess possible contributions of the basal ganglia to rhythmic entrainment mechanisms we studied patients on dopaminergic medication and patients who had been off medication for at least 24 hours. Ten healthy elderly subjects served as a normal control group.

Subjects and methods

SUBJECTS

Twenty one patients (six women, 15 men) with a primary diagnosis of idiopathic

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Parkinson's disease were studied while on dopaminergic medication (mean age 71 (SD 4) years; eight patients were modified Hoehn and Yahr stage II, 10 were stage III, and three were stage IV; mean duration of disease 7.5 years). The three patients at Hoehn and Yahr stage IV needed hand held assistance by a physiotherapist during their walking. Ten patients (four women, six men) were studied 24 hours after their last medication (mean age 73 (SD 3) years; four patients were modified Hoehn and Yahr stage II, six patients were stage III; mean duration of disease 7.8 years). One patient (a man aged 73 years, Hoehn and Yahr stage III, duration of Parkinson's disease 8.5 years) had voluntarily stopped medication for 48 hours. No pronounced motor fluctuations were present in the patients with Parkinson's disease. Ten healthy elderly subjects (six women, four men) without any known diagnosed cognitive, sensory, or physical impairments served as a normal control group (mean age 72 (SD 5) years). Informed consent was obtained before the study.

DESIGN

Patients were allowed a brief warm up consisting of a two minute walk covering about 20 m. All subjects then walked along a 15 m walkway, turned 180° at the walkway end, and returned to the starting position. Four gait trials, each for a total walking distance of 30 m, were recorded under the following conditions and in the following order: (1) their own maximal speed with no external rhythm (baseline); (2) in time to RAS matched in tempo to each patient's baseline cadence; (3) in time to RAS set at a tempo 10% faster than baseline; (4) with no external rhythm to check for immediate carry over effects.

Gait data were recorded and analysed with a computerised foot switch system consisting of four separate sensors measuring surface contact for heel, toe, and first and fifth metatarsal. The RAS was provided by a click tone stimulator generating a 50 ms square wave pulse embedded in instrumental music in Renaissance style in 2/4 metre. The tempo of RAS was expressed in beats per minute to allow matching the stimulator generated auditory beats with the baseline cadence of each

patient. The music was digitally prerecorded on a synthesiser/sequencer to allow for variable tempo reproduction without losing pitch control. Auditory beats and stride data were recorded simultaneously to allow for an analysis of the synchronisation pattern between rhythmic stimulus and step patterns (fig 1). Patients on medication (ON) were tested in the morning 60 to 90 minutes after medication (sinemet or sinemet/eldepryl). Patients off medication (OFF) were tested during the same time of day 24 hours after the last dose. The healthy elderly controls were also tested during the same morning hour.

Results

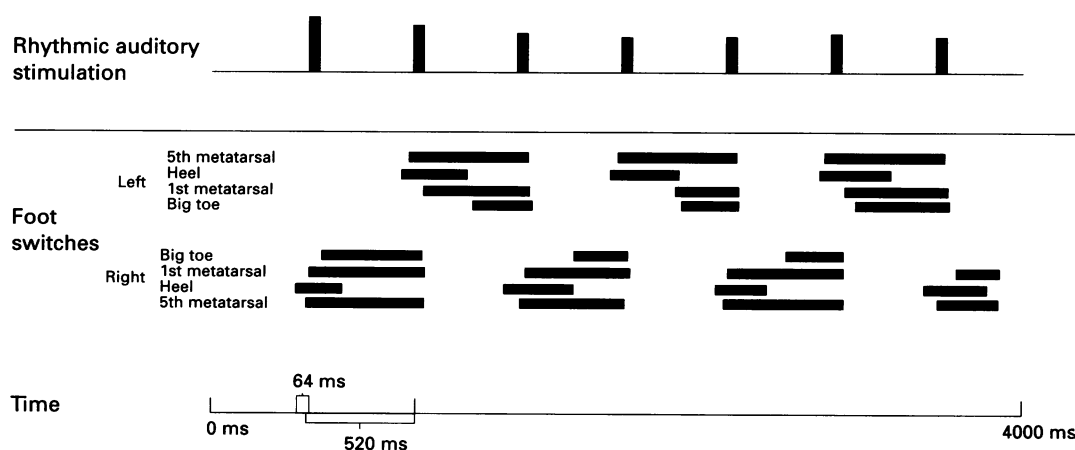
BASELINE

In the absence of RAS all patients with Parkinson's disease showed abnormal gait patterns characteristic of Parkinson's disease. For the ON group this included decreased velocity (mean 42.0 (SD 12.2) m/min) and shortened stride length (mean 0.86 (SD 0.19) m). The OFF group showed even further decreased velocity (mean 33.7 (SD 15.1) m/min), reduced stride length (mean 0.74 (SD 0.21) m), and moderate stride asymmetry (mean 0.82 (SD 0.15)%) when symmetry was calculated as the time ratio between two successive steps using the longer step time as denominator. The accepted normal age matched values reported are 73 m/min for velocity and 1.27 m for stride length.⁹ Average cadence (steps/min) for the ON group was 98 (SD 10) and 91 (SD 12) steps/min for the OFF group compared with the 113 steps/min typical of normal age matched subjects. The stride data of the normal elderly group fell well within normal age ranges (velocity 74.4 (SD 6.4) m/min, cadence 111 (SD 7) steps/min, stride length 1.34 (SD 0.17) m, symmetry 0.97 (SD 2)%).

CADENCE/RAS MATCHING

All ON patients showed close phase coupling between the initial surface contact of the foot and the onset of the beat when RAS was matched to each patient's baseline cadence (fig 2). Surface contact preceded the rhythmic cue in 75% of all measures with a small but consistent mean synchronisation error of 75

Figure 1 Sample recording of auditory signal (RAS) and foot switch traces of a representative patient with Parkinson's disease. RAS interstimulus interval in this recording is 520 ms. The subject commits an anticipatory synchronisation error on the first recorded stride and maintains this pattern throughout the displayed strides (right heel strike occurs 64 ms before onset of RAS).



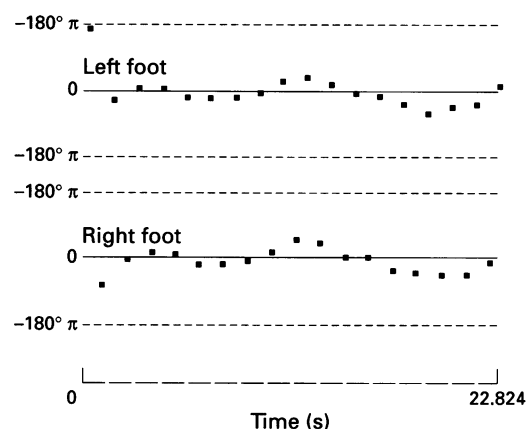


Figure 2 Step/RAS synchronisation pattern of a patient with Parkinson's disease who shows close phase coupling between onset of auditory signal (zero axis) and foot surface contact (black squares). Vertical deviations of squares from the zero axis display the synchronisation error between foot contact and auditory signal (below zero = anticipatory synchronisation error; above zero = delayed synchronisation error). For normalisation, the amplitude of synchronisation error is converted into phase angle (π) deviations. A synchronisation error of 180° out of phase would indicate a footstrike on the auditory signal cueing the opposite lower limb. Data in this recording are RAS frequency (interstimulus interval) = 658 ms; step cadence = 92/min; mean synchronisation error (left) = -0.045 ms ($\pi = -24.4$); mean synchronisation error (right) = -0.024 ms ($\pi = -13.4$).

Figure 3 Sequence of synchronisation errors between foot surface contact and auditory signal of a patient with Parkinson's disease who walks at a faster step cadence than the RAS frequency (see fig 2 for legend details). By drifting ahead of the auditory signal the synchronisation error pattern is characterised by continuously alternating phase angle shifts without phase locking.

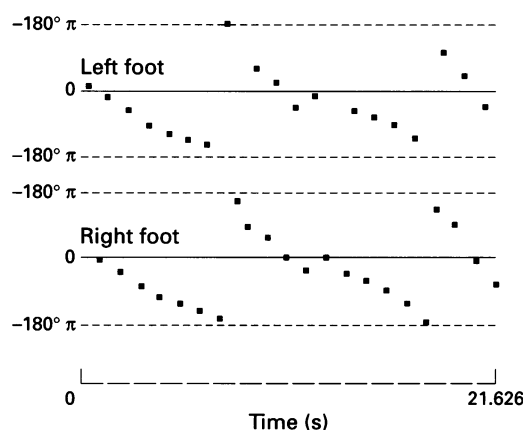


Table 1 Means (SD) of synchronisation data

	Synchronisation error (ms)		VAR/IRI (ms)	
	Normal	Fast	Normal	Fast
Controls	72 (23)	89 (32)	47 (32)	36 (26)
Parkinson's disease ON	75 (30)	81 (51)	50 (38)	51 (30)
Parkinson's disease OFF	110 (54)	105 (57)	54 (43)	57 (45)

VAR/IRI = standard deviation of IRI.

Table 2 Mean values of stride variables for elderly controls and patients with Parkinson's disease (PD)

		Baseline	RAS	Carry over
Velocity (m/min)	Controls	74.4	85.5*	85.0
	PD ON	42.0	57.0*	56.2
	PD OFF	33.7	42.3*	42.7
Cadence (steps/min)	Controls	111.0	123.0*	120.0
	PD ON	98.0	108.0*	107.0
	PD OFF	91.0	100.0*	98.5
Stride length (cm)	Controls	134	139	137
	PD ON	86	102*	98
	PD OFF	74	88*	83
Symmetry	Controls	0.97	0.97	0.96
	PD ON	0.96	0.96	0.96
	PD OFF	0.82	0.88	0.88

n = 10 for controls; n = 21 for PD ON; n = 10 for PD OFF.

* $P < 0.05$.

(SD 30) ms. In the OFF group exact phase coupling was seen in seven out of 10 patients. Two patients drifted ahead of the beat (100 steps/min to RAS at 85; 103 steps/min to RAS at 100), and one patient fell slightly behind the beat (95 steps/min to RAS at 98). The mean synchronisation error was 110 (SD 54) ms. All normal elderly subjects were able to synchronise their steps to the rhythm with a mean synchronisation error of 72 (23) ms.

FREQUENCY MODULATION

After a 10% increase in RAS, 19 out of 21 ON patients were able to increase their step frequency and retain their step/beat phase coupling with a mean synchronisation error of 81 (SD) 51 ms. One patient walked faster with RAS but did not match his step cadence to the external cue (cadence increase 81 to 97 steps/min; RAS increase 81 to 89 beats/min; fig 3). The patient with the most severe gait deficits was not able to modulate his step frequency to the increased RAS frequency.

In the OFF group seven patients retained exact step/beat phase coupling whereas two patients' cadences continued to drift slightly ahead of the beat, by 2.5% and 4% respectively. Only one patient showed a considerable phase drift with his cadence 25% faster than the RAS frequency. The mean synchronisation error for the OFF group was 105 (SD 57) ms. The normal group was able to maintain rhythmic step synchronisation with a mean synchronisation error of 89 (SD 32) ms. Table 1 summarises the synchronisation data.

In addition to step synchronisation, the difference between step durations (interresponse interval = IRI), and the constant rhythmic interval (interstimulus interval = ISI) was computed as an indicator of how closely subjects were able to match external (step) to internal (rhythm) frequency intervals. Whereas synchronisation error measures are indicators of phase locking mechanisms—that is, positional errors between response and cueing signal, frequency matching probably involves an absolute time matching process in the brain independent of fluctuations in phase locking.¹⁰ ON and OFF patients showed remarkably similar variability measures in interval matching: 50 v 54 ms for matched cueing and 51 v 57 ms for faster cueing (table 1).

GAIT VARIABLES

Statistical analysis (repeated measures ANOVA) disclosed significant improvement in velocity ($F = 13.5$; $P < 0.01$), cadence ($F = 7.3$; $P < 0.02$), and stride length ($F = 6.8$; $P < 0.03$) for the faster RAS condition across all three groups, as shown in table 2. For the ON group mean velocity increased by 36% ($t = 3.11$; $P < 0.01$), cadence increased by 10.8% ($t = 2.04$; $P < 0.05$), and stride length increased by 18.6% ($t = 2.68$; $P < 0.02$). In the OFF group, velocity improved by 25% ($t = 3.05$; $P < 0.01$), cadence by 9.9% ($t = 2.02$; $P < 0.05$), and stride length by 18.9% ($t = 2.70$; $P < 0.02$). The normal elderly group improved velocity by 14.9% ($t = 2.34$; $P < 0.04$), cadence by 10.8% ($t = 2.07$; $P < 0.05$).

0.05), and stride length non-significantly by 3.7%. Improvement in symmetry ratios was not significant in any group.

SHORT TERM CARRY OVER

Increases in gait velocity, cadence, and stride length persisted during the last, uncued walking condition in all three groups with a small decay rate of 1%–5 %, indicating a short term carry over effect of RAS on gait patterns.

Discussion

The results of the present study confirm and extend previous reports of rhythmic auditory facilitation on parkinsonian gait. In an extension of previous studies, we were able to determine the effect of changes in RAS frequency on gait patterns and associated stride synchronisation errors in an entrainment design. We were also able to show that most patients with Parkinson's disease in the absence of dopaminergic medication were still able to access rhythmic entrainment mechanisms to improve their gait patterns. Although phase locking measures indicated larger and more variable synchronisation errors in the OFF patients than in medicated and healthy subjects, deviations in frequency matching were remarkably similar to the other two groups.

Gait velocity, cadence, and stride length improved in 19 out of 21 ON patients. The same stride variables improved for all OFF patients with the exception of the stride lengths of two. The close rhythmic synchronisation patterns across different RAS frequencies found in the vast majority of ON and OFF patients suggests that RAS can serve as an external timekeeper to which the step cadence becomes entrained. The patient in whom RAS was able to increase walking speed but without a synchronised cadence, may have responded to a more general arousal effect of the music. A research design presenting RAS as a single pulse train without musical context would control for this effect in future research. The apparent inability of the most severely impaired patient to respond to RAS raises the question of a differential effect of rhythmic auditory facilitation dependent on severity of the disease.

An important finding regarding sensory facilitation of disordered basal ganglia function was made in the patient with Parkinson's disease who was off medication for 48 hours. His gait movements froze at the turn in the walkway during his baseline walk and again during his last walk after RAS had been removed. However, he was able to turn smoothly and retain rhythmic synchronisation during the two RAS conditions. Although this finding is only anecdotal in the context of this study, it provides further evidence for the effectiveness of an auditory rhythm to facilitate movement sequencing and overcome akinesia often associated with basal ganglia dysfunction.

Sound, music, and rhythm have been used throughout history and across all cultures to stimulate and organise motor function.

However, the neurophysiological basis for auditory-motor interactions is not well understood. There is some evidence that rhythmic sound patterns can increase the excitability of spinal motor neurons via the reticulospinal pathway, thereby reducing the amount of time required for the muscles to respond to a given motor command. For example, Paltsev and Elnor¹¹ found that auditory "go" signals reduced reaction time in a voluntary motor task. Rossignol and Melvill Jones¹² later showed auditory facilitation of the H-reflex response elicited before hopping movements made in time to a musical rhythm. They also showed that movement related gastrocnemius activity occurred during the period when the H-reflex was maximal, suggesting that descending motor commands became entrained to the auditory pacing signal so as to make best use of a potential audiospinal facilitation effect.

Apart from a putative role in enhancing spinal motor neuronal excitability, the mechanism by which auditory signals assist in coordinating rhythmic or sequential movements remains to be determined. Indeed, it is still unclear what part various movement related brain structures may play in the coordination of complex movements. However, there is increasing evidence that the basal ganglia may play an important part in the proper sequencing of repetitive motor tasks.^{13–15} Locomotor deficits associated with Parkinson's disease, particularly gait festination and freezing, may reflect a disturbance of internal rhythm formation as has been recently suggested by Freeman *et al.*¹⁶ However, the evidence for entrainment effects in the OFF group suggests that rhythmic synchronisation with an auditory timekeeper can be achieved in the absence of intact basal ganglia function. Studies in non-human primates have shown that movement related phasic discharge of pallidal neurons may serve as an internal cue to the supplementary motor area signalling the end of one movement and allowing the onset of the next.¹⁷ Several authors have suggested that predictive external sensory cues, such as auditory rhythm, can provide the necessary trigger in Parkinson's disease to switch from one movement component in a movement sequence to the next and thus bypass defective internal pallidocortical projections,^{18–19} possibly via the lateral premotor cortex which receives sensory information in the context of externally guided movements.^{20–21} This would explain why patients with Parkinson's disease in the absence of external cues show dramatically slowed movement initiation and execution times which can improve significantly when external sensory cues are present. Recently, Georgiou *et al.*²² found that auditory cueing led to a significant reduction in initiation and execution times in a sequential button pressing task performed by patients with Parkinson's disease. Further, predictable rhythmic metronomic cues were found to be more effective in improving motor timing compared with non-rhythmic auditory cues which were presented contingent on depress-

ing the buttons, or visual cues sequentially illuminating the button path.

Rhythmic entrainment of step cadence in patients with Parkinson's disease has been previously shown by Richards *et al*⁶ and Morris *et al*,¹⁸ however, without providing specific synchronisation data. The specific problem in gait of patients with Parkinson's disease to generate steps of appropriate length has been demonstrated by Morris *et al*.¹⁸ Our data, similar to those of Richards *et al*,⁶ show increases in cadence as well as stride length when rhythmic cues are accelerated by 10% over the baseline step rate. In previous work we have found a reduction in EMG shape variability in the gastrocnemius muscle in patients with Parkinson's disease during RAS cued walking, indicating more consistent motor unit recruitment patterns.²³ Physiological entrainment effects on lower limb EMG have been previously reported by Rossignol and Melvill Jones.¹² Because steps are generated by phasic activation from the triceps surae muscles in the late stance phase, increased stride length during rhythmic gait facilitation may be due in part to the modulatory effect of RAS on muscle activity.

The results of this study provide strong evidence that rhythmic auditory stimulation can facilitate locomotor function in patients with Parkinson's disease suggesting rhythmic entrainment as a possible facilitating mechanism. The exact nature of such auditory-motor facilitation, however, remains to be determined and further studies of sensory facilitation in various movement disorders is clearly warranted.

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