Clinical Neurophysiology 121 (2010) 240–247

Contents lists available at ScienceDirect

Clinical Neurophysiology

journal homepage: www.elsevier.com/locate/clinph

Alternate rhythmic vibratory stimulation of trunk muscles affects walking cadence and velocity in Parkinson's disease

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ARTICLE INFO

Article history:
Accepted 30 October 2009
Available online 1 December 2009

Keywords:
Parkinson's disease
Alternate muscle vibration
Gait
Cadence
Velocity

ABSTRACT

Objective: During the administration of timed bilateral alternate vibration to homonymous leg or trunk muscles during quiet upright stance, Parkinsonian (PD) patients undergo cyclic antero-posterior and medio-lateral transfers of the centre of foot pressure. This event might be potentially exploited for improving gait in these patients. Here, we tested this hypothesis by applying alternate muscle vibration during walking in PD.

Methods: Fifteen patients and 15 healthy subjects walked on an instrumented walkway under four conditions: no vibration (no-Vib), and vibration of tibialis anterior (TA-Vib), soleus (Sol-Vib) and erector spinae (ES-Vib) muscles of both sides. Trains of vibration (internal frequency 100 Hz) were delivered to right and left side at alternating frequency of 10% above preferred step cadence.

Results: During vibration, stride length, cadence and velocity increased in both patients and healthy subjects, significantly so for ES-Vib. Stance and swing time tended to decrease. Width of support base increased with Sol-Vib or TA-Vib, but was unaffected by ES-Vib.

Conclusions: Alternate ES vibration enhances gait velocity in PD. The stronger effect of ES over leg muscle vibration might depend on the relevance of the proprioceptive inflow from the trunk muscles and on the absence of adverse effects on the support base width.

Significance: Trunk control is defective in PD. The effect of timed vibratory stimulation on gait suggests the potential use of trunk proprioceptive stimulation for tuning the central pattern generators for locomotion in PD.

1. Introduction

Muscle vibration provides substantial proprioceptive inflow to the central nervous system from primary muscle spindle endings (Roll and Vedel, 1982). Powerful changes in tendon-tap excitability are produced by muscle- or tendon-vibration (Hagbarth, 1973; Burke et al., 1976; Desmedt and Godaux, 1978; Schieppati and Crenna, 1984). Vibration also affects the muscle responses elicited by postural perturbations, in both healthy subjects and patients (Beckley et al., 1993; Bove et al., 2003a,b; Nardone and Schieppati, 2005). Vibration of several muscles along the body axis produces major systematic changes in standing posture (Eklund, 1972; Quoniam et al., 1995; Bove et al., 2001, 2002, 2007; Smiley-Oyen et al., 2002; Valkovic et al., 2006; Courtine et al., 2007). Moreover, changes in the velocity or trajectory of locomotion in healthy subjects have been reported as a consequence of bilateral and unilateral (Ivanenko et al., 2000; Schmid et al., 2005) vibration of different body muscles.

We have recently shown that bilateral alternate vibration trains applied to postural muscle groups during quiet stance produce cyclic transfers of the centre of foot pressure, in both healthy subjects and PD patients (De Nunzio et al., 2008). This timed stimulation induced normal or near-normal changes in PD in a number of variables. The time to initiate and terminate the postural responses was also comparable between normal subjects and PD patients. These effects have been interpreted as evidence that the proprioceptive inflow was correctly integrated in PD, much as in healthy subjects. In particular, the centre of foot pressure (CoP) (therefore the body's centre of mass) increased its displacement both along the antero-posterior and medio-lateral direction. The cyclic

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1388-2457/$36.00
doi:10.1016/j.clinph.2009.10.018

oscillations of the CoP were induced not only by vibration of leg muscles, but also by paraspinal muscle vibration, an unanticipated finding in the light of previous reports of altered trunk control in PD (van Wegen et al., 2001; van der Burg et al., 2006; Vaugoyeau et al., 2007; Wright et al., 2007).

Those findings have suggested the possibility for patterned muscle vibration to ultimately improve gait by favouring antero-posterior or medio-lateral body movements (Rocchi et al., 2006), given that abnormal postural adjustment for weight transfer is a cardinal feature in PD and may contribute to gait problems (van Wegen et al., 2001; Boonstra et al., 2008; Mille et al., 2007). We therefore tested the hypothesis that alternate muscle vibration of postural muscles can enhance locomotor performance in patients. A simple protocol was used, whereby patients were administered bilateral alternate vibration to lumbar paraspinal muscles, or soleus or tibialis anterior while walking on an instrumented walkway.

2. Methods

2.1. Participants

Fifteen patients, with a diagnosis of idiopathic PD and in on-phase (8 women and 7 men, mean age 68.4 years ± 10.9 SD), participated in the study. Their characteristics are summarized in Table 1. None had dyskinesia or freezing episodes at the time of the evaluation. Fifteen healthy subjects (7 women and 8 men, mean age 60.2 years ± 11.6 SD) served as controls. None reported history of otological, neurological, or orthopedic abnormality. Subjects and patients had never participated in vibration experiments previously. Experiments have been conducted in accordance with the Declaration of Helsinki. All procedures were carried out with the adequate understanding and written informed consent of the subjects involved. The institutional review board gave ethical approval to the investigation.

2.2. Vibratory device

A battery-operated custom-made system delivered vibration trains to the muscles (Fig. 1A). The system is composed of two vibrating units connected to a wearable control unit. The user interface permits to choose the vibration frequency (here 100 Hz) and the duty cycle of the alternate vibration sequence (Fig. 1C). The vibrating units (Schmid et al., 2005; De Nunzio et al., 2008) were fixed to homonymous muscles of both sides. For tibialis anterior (TA) and soleus (Sol), the vibratory units were fixed on the distal tendons by elastic bands. For erectores spinae (ES), the units were contained in two compartments sewed to a belt and separated by 10 cm; the belt with the vibrators were then tightened around the trunk by an elastic band at the height of the 2nd lumbar vertebra. The vibratory units were kept in place during both control (vibrators off) and vibration trials.

2.3. Task and procedures

At the beginning of the session, 4 control trials were carried out in each participant in order to calculate their preferred walking cadence (the average of the mean cadences of the 4 trials). Then, the procedure consisted of a sequence of 4 series of 4 walking trials each: no vibration (no-Vib), and vibration of TA (TA-Vib), Sol (Sol-Vib) and ES (ES-Vib). The sequence of vibration sites was randomized across subjects, in order to cancel in the grand average any possible carry-over effect of one vibration type onto the next. There was a rest period of at least 2 min between series. Each trial consisted in a 10 m straight walk with eyes open; in the middle of the pathway, participants walked on a 4 m sensorized walkway (GAITrite©, CIR Systems, USA) (Chien et al., 2006). When ready, subjects started walking after a verbal 'go' command. Vibratory stimulation started about 10 s before walking onset and lasted for the entire trial. The vibratory stimuli were not synchronized with the gait phases, nor was the 'go' command synchronized with a vibration cycle. Subjects and patients were instructed to walk at comfortable speed during both control and vibration trials, and to ignore the vibration stimuli. This procedure minimized the possibility that patients interpreted the alternating vibratory stimulation as a cue for triggering their gait cycles or for deliberately locking their steps to the vibration cycles. The entire procedure lasted about 30 min.

The pattern of stimulation was a sequence of bilateral alternate (right–left–right–left and so on) trains of vibration (Fig. 1C) with a

Table 1
Clinical characteristics of the patients.

Patient	Sex	Age (years)	Duration (years)	Medication (mg/day)	Equivalent dose (mg/day)	UPDRS Section III (Motor Examination)	Hoehn–Yahr
1	M	73	10	LDI-CR 750 Ropinirole 6, Cabergoline 2	1070	47	3
2	M	75	1	LDI 325	325	26	1.5
3	F	66	11	LDI 1000, Entacapone 600 Pramipexole 2.5	1450	45	3
4	F	72	3	LDI 250 Pramipexole 1.1, Rasagiline 1	360	23	2
5	F	75	8	LDI 315 Pramipexole 2.1	577	18	3
6	F	77	4	LDI 250, Melevodopa 65 Pramipexole 2.1	512	35	3
7	F	75	2	LDI 250	250	11	2.5
8	M	42	2	Melevodopa 375 Pramipexole 0.54, Rasagiline 1	354	19	2
9	M	69	16	LDI 375, Entacapone 600 Ropinirole 4, Rasagiline 1	584	16	2.5
10	M	67	1.5	Pramipexole 2.1	210	26	3
11	F	69	4	Pramipexole 2.6	260	23	1.5
12	F	81	9	LDI 440, Rasagiline 0.5	440	37	3
13	M	72	4	LDI 1000	1000	26	2.5
14	M	68	4	LDI 665	665	26	3
15	F	45	1	Rasagiline 1, Ropinirole 4.5	90	23	1.5

Abbreviations: LDI, levodopa + dopa decarboxylase inhibitor; CR, controlled release.
Equivalent dose was calculated according to Tonolli et al. (2000).

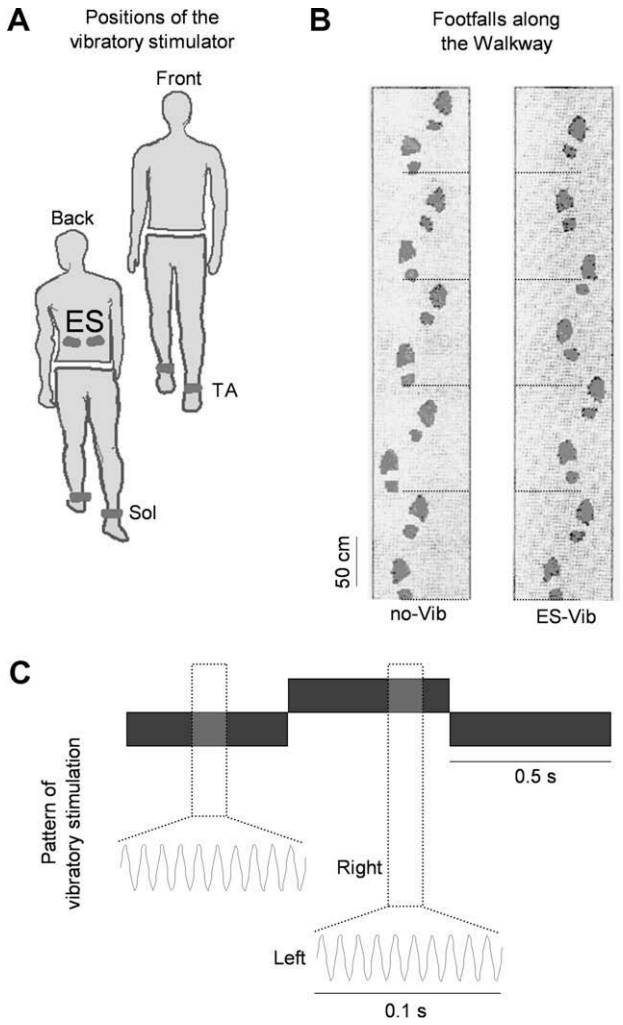

Fig. 1. (A) Positions of vibrators during walking trials. (B) Footsteps of right and left foot and their relative position during the stance phases of gait (no-Vib and ES-Vib) along the sensorized walkway. (C) The stimulus consisted in vibration trains (internal frequency 100 Hz), alternately applied to left and right homonymous muscles, with a duty cycle equal to 110% of the preferred cadence.

duty cycle of alternation arbitrarily set at about +10% of the subject's preferred cadence, anticipating that this would have increased cadence and walking velocity. We considered that a smaller difference (less than +10%) might have produced no significant effects during walking, owing to the expected variability in preferred gait cadence within patients. Conversely, a higher value might instead be too 'demanding' for patients. All patients completed the session without fatiguing.

2.4. Detection and analysis of gait variables

The walkway captures the geometry of each footfall as a function of time (Fig. 1B). The application software processes the raw data into footfall patterns and computes temporal and spatial parameters. For each trial, we computed the mean values of stride length (collected from right and left stride lengths), step cadence, walking velocity, width of base of support, stance and swing time.

2.5. Data reduction and statistical analysis

Shapiro–Wilk and Levene tests were used to assess normality and homogeneity, respectively, for all employed gait variables. Under no-Vib condition all variables were normally distributed (Shapiro–Wilk test) except stance phase duration ($p < 0.005$). Variances were homogeneous for all variables (Levene test), except stance phase duration ($p < 0.05$). The effects of vibration were tested first within each group by means of 1-way repeated-measures ANOVA. Then, the effects on each variable were compared by means of 2-way repeated-measures ANOVA between groups (healthy, PD) and within vibration conditions (no-Vib, TA-Vib, Sol-Vib, ES-Vib). When ANOVA gave a significant result, Newman–Keuls test was used for post-hoc comparisons. The post-hoc correction was applied both between and within factor levels. In the case of stance phase duration, the Friedman test was used to test the difference between vibration conditions within each group. When the Friedman test was significant, the post-hoc Wilcoxon test was run after Bonferroni correction. Correlation coefficients between clinical and spatio-temporal gait variables were calculated using Spearman's rank method for ordinal variables. The software package Statistica (StatSoft©, USA) was used.

3. Results

The position of the vibratory devices, the time course of the vibratory stimulus and the effect thereof are presented in Fig. 1 for the ES-Vib.

3.1. Stride length (Fig. 2A)

Mean stride length during no-Vib was 130.6 ± 3.9 cm for healthy subjects and 102.7 ± 5.4 cm for PD. One-way ANOVA, separately performed in healthy subjects and patients, showed no effect of conditions on stride length in healthy subjects ($F(3,42) = 0.27$, $p = 0.84$). Conversely, there was a significant effect on stride length in PD ($F(3,42) = 6.55$, $p < 0.005$). Two-way ANOVA showed a difference between groups ($F(1,28) = 17.49$, $p < 0.005$) and vibration conditions ($F(3,84) = 5.58$, $p < 0.005$). There was a significant interaction ($F(3,84) = 3.94$, $p < 0.05$). Post-hoc analysis showed that stride length significantly increased in PD for vibration of ES muscles ($p < 0.05$), whereas no significant increase was found for Sol-Vib; a significant reduction in stride length was induced by TA-Vib ($p = 0.019$).

3.2. Walking cadence (Fig. 2B)

One-way ANOVA showed that there was an effect of conditions on walking cadence in both healthy subjects ($F(3,42) = 7.58$, $p < 0.005$) and patients ($F(3,42) = 14.7$, $p < 0.005$). Post-hoc test showed that during ES-Vib there was a significant increase in cadence in both healthy subjects ($p < 0.005$) and patients ($p < 0.005$); in the latter group, also Sol-Vib significantly ($p < 0.005$) increased cadence. Two-way ANOVA showed no difference between groups, preferred cadence being only slightly lower in PD ($F(1,28) = 1.27$, $p = 0.26$). In particular, during ES-Vib, PD reached a cadence not different from the preferred cadence of healthy subjects (116.7 ± 3.6 step/min in PD during ES-Vib and 117.7 ± 2.0 step/min in healthy subjects under no-Vib condition).

3.3. Velocity (Fig. 2C)

Mean velocity during no-Vib was 128.2 ± 5.3 cm/s for healthy subjects and 96.2 ± 7.0 cm/s for PD. One-way ANOVA showed an effect of conditions on velocity only in patients ($F(3,42) = 9.4$, $p < 0.005$). Post-hoc test showed a significant increase in velocity in patients during ES-Vib ($p < 0.005$). Two-way ANOVA showed a difference between groups ($F(1,28) = 12.79$, $p = 0.005$) and between vibration conditions ($F(3,84) = 11.78$, $p < 0.005$). The interaction did not reach significance ($F(3,84) = 2.01$, $p = 0.11$).

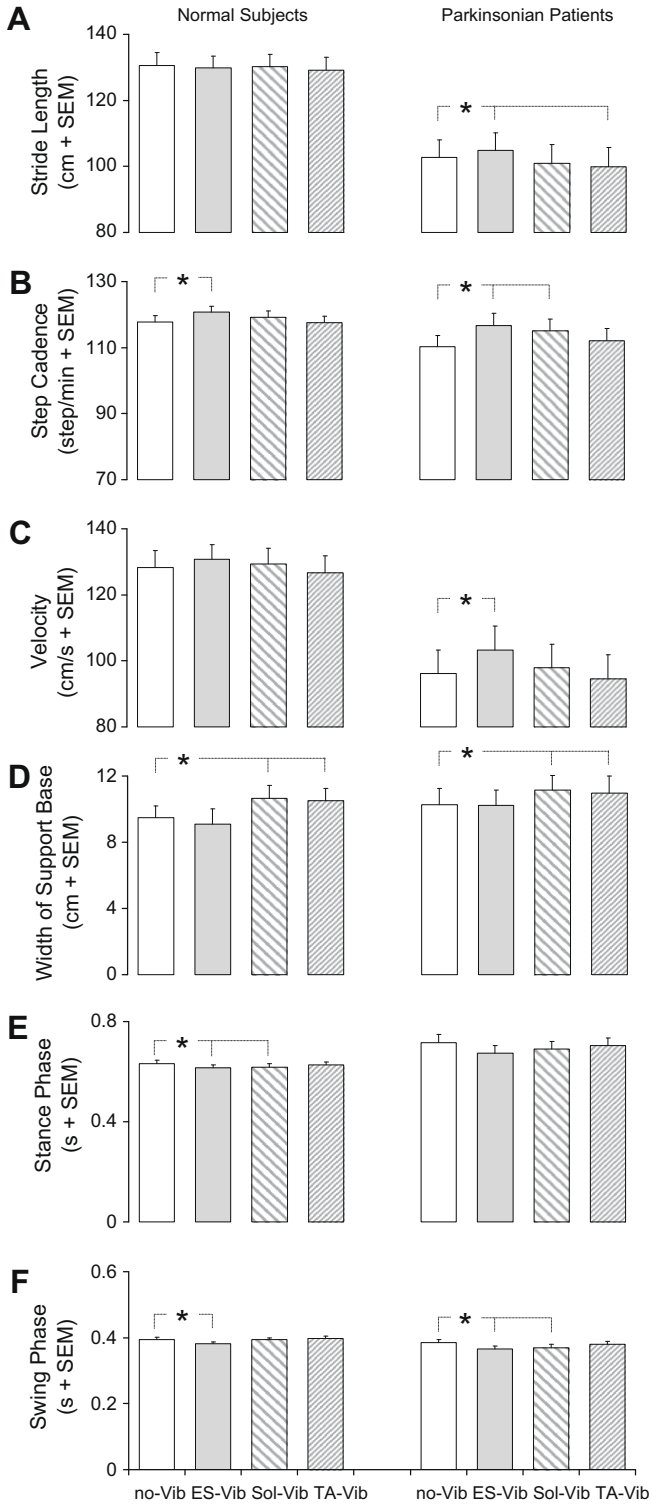

Fig. 2. The effects of vibration in normal subjects and PD patients. (A) Mean stride length as a function of the three vibration sites (white bars: no-Vib conditions). (B) Same as A, but for step cadence. (C) Same as A, but for walking velocity. (D) Same as A, but for the width of support base. (E and F) Same as A, but for duration of stance and swing phases. Asterisks point to significant differences between no-Vib and Vib data.

3.4. Width of support base (Fig. 2D)

Mean width of the support base during no-Vib was 9.5 ± 2.7 cm/s for healthy subjects and 10.4 ± 3.4 cm/s for PD. One-way ANOVA showed an effect of conditions on width of the support base in both healthy subjects ($F(3,42) = 4.89$, $p < 0.005$) and patients ($F(3,42) = 3.86$, $p < 0.05$). Post-hoc test showed a significant increase in support base during both Sol-Vib and TA-Vib, in both healthy subjects and patients ($p < 0.05$, for both muscles).

3.5. Phases of gait cycle (Fig. 2E, F)

The mean stance time was 0.63 ± 0.01 s for healthy subjects and 0.71 ± 0.03 s for patients under no-Vib condition. There was a difference between vibration conditions only in the healthy subject group (Friedman test, χ^2 ANOVA ($n = 15$, df = 3) = 20.84, $p < 0.005$). Post-hoc analysis showed a significant decrease in stance time for ES-Vib and Sol-Vib compared to no-Vib (Wilcoxon test, $p < 0.005$).

Fig. 2F shows the swing phase duration. One-way ANOVA showed an effect of conditions on swing duration in both healthy subjects ($F(3,42) = 14.1$, $p < 0.005$) and patients ($F(3,42) = 9.3$, $p < 0.005$). Two-way ANOVA showed no difference between healthy subjects and PD patients ($F(1,28) = 2.47$, $p = 0.13$). A difference was found between vibration conditions ($F(3,84) = 17.16$, $p < 0.005$). The interaction was significant ($F(3,84) = 3.59$, $p < 0.05$). Post-hoc analysis showed a decrease of swing time for both healthy subjects and PD during ES-Vib ($p < 0.005$, for both comparisons); in the patients, also Sol-Vib decreased the swing time ($p < 0.005$).

3.6. Consistency of the findings

There was a large variability in the no-Vib values for stride length and velocity across PD patients, likely connected to the different severity of their disease, even if p values for regression between velocity ($y = 123.14–1.01x$, $p = 0.16$, $r^2 = 0.15$) or stride length and UPDRS score ($y = 60.02–0.33x$, $p = 0.24$, $r^2 = 0.10$) did not reach significance. However, the consistency of the effects of vibration within patients was high. In particular, Fig. 3 shows that the effects of ES-Vib on stride length (Fig. 3A), cadence (Fig. 3B) and velocity (Fig. 3C) were common to most PD patients. An increase in cadence was observed in all patients except one; for stride length and velocity three patients escaped the effect.

3.7. Correlations between clinical variables and spatio-temporal gait data

Across the patients, no relationship was found between any spatio-temporal gait data and age, duration of disease, UPDRS score, Hoehn–Yahr staging, for either no-vibration or vibration walking conditions. As an example, the equation of the regression line between Hoehn–Yahr and walking velocity under no-Vib condition was: $y = 101.85–2.28x$ ($p = 0.85$, $r^2 = 0.003$). There was no significant correlation between equivalent levodopa dose and vibration effects across the patients, regardless of the vibrated couples of muscles. As an example, the equation of the regression line between increase in velocity induced by Vib-ES and equivalent levodopa dose was $y = 5.50 + 0.003x$ ($p = 0.64$; $r^2 = 0.02$).

4. Discussion

4.1. Selection of patients

The walking tests have been performed in on-phase patients only. This was deliberately decided at the beginning of the investigation, since: (1) patients were more willing to walk with and without vibration when in on-phase; (2) the session duration was already extended in time; for practical reasons, any off-phase session would have required a longer, hardly sustainable experiment for these patients; (3) the gait variability would have

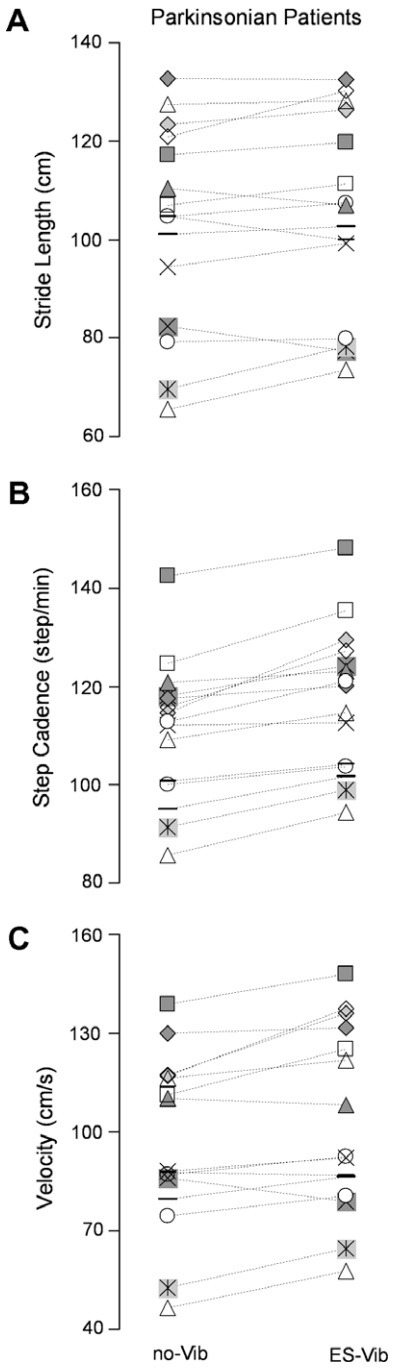

Fig. 3. Stride length (A), step cadence (B) and velocity (C), compared between no-Vib and ES-Vib condition within each PD patient. All patients consistently increased their step cadence during ES-Vib; most of them also increased stride length and velocity.

increased, diminishing the likelihood of finding potential differences; (4) it was deemed unethical to put these patients through lengthy sessions, in the absence of any evidence that vibration might really represent a walking aid.

4.2. Muscle vibration improves walking cadence and velocity

The walking velocity under control condition (no-Vib) was smaller in PD patients than in healthy subjects (Sofuwa et al., 2005; Carpinella et al., 2007). All patients increased walking velocity under vibration conditions. This was produced by increased cadence and to a lesser extent by increased stride length. In the healthy subjects, velocity was not affected by vibration to a significant extent, because the increase in step cadence was smaller, and stride length was unaffected. There was consistency across healthy subjects and across patients. In particular, walking was selectively improved by vibration of the erectores spinae (ES-Vib) muscles, weak or no effects being produced by vibration of soleus (Sol-Vib) or tibialis anterior (TA-Vib) in both groups. On average, the velocity during ES-Vib increased by about 2% in healthy subjects and 7% in patients. During ES-Vib, patients attained a cadence not different from the no-Vib cadence of healthy subjects.

4.3. Is the improvement in cadence connected to a vibration-related 'cue'-effect?

There is abundant literature on the effect of cueing in PD (see Lim et al., 2005; Nieuwboer et al., 2007; Arias and Cudeiro, 2008). This protocol was not designed for, and does not add new information to this controversial issue. However, since acoustic (Howe et al., 2003; Suteerawattananon et al., 2004; Jiang and Norman, 2006), somatosensory and proprioceptive cues have been administered to patients with some success (Frenkel-Toledo et al., 2005; van Wegen et al., 2006), we asked whether our findings could be interpreted as an effect of cueing. The vibrators, in addition to delivering a rhythmic proprioceptive input, did produce a faint though clearly perceptible noise (patients were not wearing earphones, as they had found them uncomfortable). However, vibration of different muscles clearly did not produce the same outcome, in spite of the same 'acoustic cueing'. For example, soleus vibration had no effect on the gait variables, but was clearly perceived by all the patients. The differences in the effects of the three vibrated sites were consistent across all patients and subjects. This would argue against a cueing effect linked to the rhythmic acoustic or tactile or even proprioceptive stimulation produced by the vibrators.

Moreover, stepping cadence increased, but the cadence was rarely equal to the frequency of the alternate trains of vibration, as shown in Fig. 4. When, at the end of the session, participants were asked whether they deliberately used the rhythmic cue (be it acoustic or tactile) for setting the gait cycle, their answer was always negative. However, the cueing possibility should be left open as a cause for enhancing gait velocity, since the participants might have simply paid more attention to the trunk than leg muscle vibration. Further, the mere intent (more or less deliberate) to follow the vibration cycles (without necessarily succeeding in the task, as it turned out to happen) might have increased the stepping rate. This also opens the possibility that sort of a placebo effect might have affected the results: this cannot be ruled out, unless again referring to the clear-cut differences between vibration sites. We can only add that, albeit within a different context (unilateral vibration during walking in normal subjects, Bove et al., 2001; Courtine et al., 2001, 2007), the vibration site made a difference: neck and trunk, but not upper or lower limb sites, were effective in producing consistent deviations in the walking trajectory. It is necessary to mention, however, that others have reported improvement in PD gait by vibration of the foot sole triggered by foot contact (Novak and Novak, 2006). It is difficult to compare their findings to ours because the site of vibration differs and because their stimulus was locked to the gait phase. It is however interesting to consider that their effects might have been related to a synergic interaction between vibration and cueing.

4.4. Why did patients not reach the imposed cadence?

All patients except one increased their cadence and most of them increased velocity during vibration (in particular with ES-

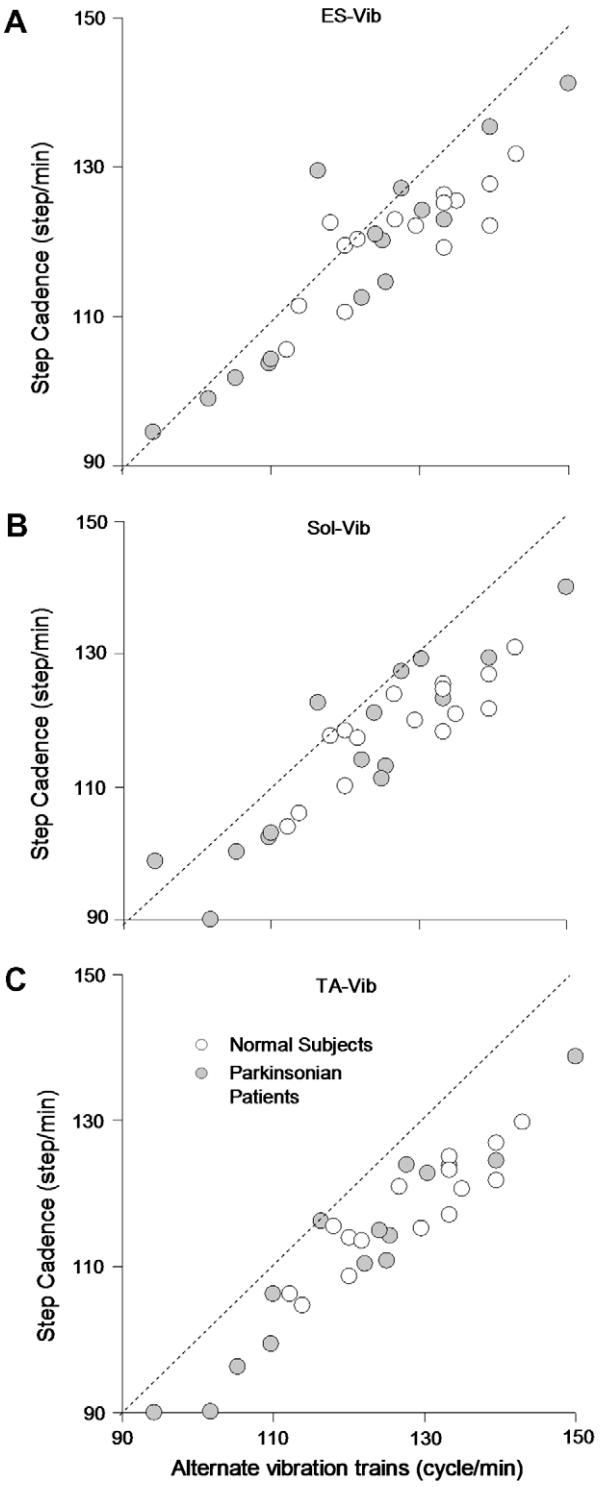

Fig. 4. Correlation between step cadence during vibration and frequency of alternate vibration cycles for each healthy subject (white circles) and PD patient (grey circles). The reported step cadence refers to ES-Vib (A), Sol-Vib (B) and TA-Vib condition (C). The scatter in the abscissa depends on imposed alternate vibration trains being set for each participant to about 10% above his or her preferred walking cadence no-Vib. All participants increased step cadence with respect to control conditions, though the cadence rarely reached the frequency of alternate vibration trains. The dotted identity line represents the relationship between the imposed alternate vibration pattern and step cadence, which would held in the case that vibration cycles entrained the step cadence.

Vib). One patient walked at a frequency greater than the vibration-train frequency, two at the same frequency, and all the others at frequencies greater than their own control cadence but lower than the imposed vibration-train frequency. Beyond the expected variability, however, we have no simple explanation for the fact that step cadence was generally lower than the imposed frequency even during ES-vibration (Fig. 4). It might be argued that patients did not have the capacity to increase walking frequency beyond a certain limit. In this light, setting the vibration frequency to 110% of their control cadence might have been excessive. But there was apparently no ceiling effect, because the patients for whom the increase in cadence was minimal were not those spontaneously walking at the highest frequencies under no-Vib condition.

Notably, cadence regulation can be nearly normal in PD (Ferrandez and Blin, 1991; Morris et al., 1994), but it is resistant to dopa (Blin et al., 1991) and deep brain stimulation (DBS) (Faist et al., 2001; Lubik et al., 2006) in spite of the sometimes dramatic effects of DBS on step length. In a sense, therefore, the vibration-induced increase in cadence observed here may be considered a positive and specific effect of the vibration.

The lower than expected entrainment may have diverse explanations: (a) the imposed vibration frequency was too high: as said above, the anticipation that cadence could increase up to 110% of normal cadence was perhaps simply unwarranted; (b) the total number of strides in a trial was probably too short to allow entrainment of the stepping cycle with the vibration frequency; (c) the vibration trains were not synchronized to the gait cycle: the variability in cadence increase across subjects and trials might be a consequence of the variable phase-shift between frequency of alternate vibration and actual cadence. However, since, in spite of these limitations, both healthy subjects and patients showed an increase in gait cadence, we would deduce that alternate paraspinal (ES) muscles' vibration has an effect on the central gait pattern generator (Dietz, 2002; Zehr and Duysens, 2004). Yet, vibration may exert more of a permitting influence than an action related to hard-wired connections between the spindle afferents and the pattern generator, as it would have occurred instead if vibration trains were triggered at fixed intervals of the gait cycle.

4.5. Why is vibration more effective when delivered to paraspinal than leg muscles?

The extent to which increases in walking cadence and velocity are associated with vibration-induced increments in the antero-posterior or medio-lateral oscillations of the centre of mass (De Nunzio et al., 2008) remains to be demonstrated by appropriate movement analysis in further investigations. In a sense, the finding that vibration and gait cadence were not synchronized not only rules out a cueing effect, but also speaks against the possibility that the increase in walking velocity was assisted by a vibration-induced body shift during locomotion. Yet, any increase in antero-posterior or medio-lateral body shift *per se* by the vibration-induced rhythmic muscle action may not be the principal mechanism. The above mentioned medio-lateral shift, induced by alternate vibration during stance (both feet always on the ground), seemingly turned into an increase in the width of the support base during walking, i.e. when one foot at a time was on the ground. Increased width of support base is generally detrimental during walking (Stolze et al., 2002). It is not unlikely that the small increase in width of the support base produced by leg muscle (but not ES) alternate vibration represents an obstacle to the appropriate alternate transfer between gravitational-potential energy and kinetic energy within each stride (Cavagna et al., 1977), thereby impeding a fluent progression by interfering with any positive effect of vibration. This may explain why, during walking, the velocity-enhancing effects are limited to trunk vibration. In the case of soleus or tibialis anterior muscle vibration, the medio-lateral body shifts would be the direct consequence of the alternate activation of the muscles. Conversely, alternate paraspinal muscle vibration would ultimately favour progression by helping the central

generator of gait pattern (Ivanenko et al., 2006) to produce the appropriate pre-programmed body shifts that are defective in PD (Tonolli et al., 2000). An enhanced transmission in the interneuronal pathway activated by group II spindle afferents in PD patients has been described (Simonetta-Moreau et al., 2002): vibration might also favour trunk mobility by reducing the excitability of group-II related reflexes, as previously shown in a different context (Bove et al., 2003a,b).

As discussed above, unilateral vibration of trunk (Courtine et al., 2007) but not of limb muscles (Courtine et al., 2001), is able to powerfully modify walking characteristics. Besides, continuous vibration of the dorsal neck postural muscles is effective in inducing a moderate increment in the velocity of forward walking on a treadmill (Ivanenko et al., 2000). The axial muscles are innervated by medially descending motor systems while the extremity muscles by the phylogenetically more recent, laterally descending systems (Gramsbergen, 2005). By their nature, the two systems may be differently susceptible to decreases in levodopa availability or to other interventions, like subthalamic nucleus stimulation (Bejjani et al., 2000; Yamada et al., 2008).

4.6. Conclusions

Alternate vibration of the paraspinal muscles increases walking velocity by increasing cadence and stride length in PD patients. These effects are similar to or even larger than in healthy subjects. Therefore, patients may take advantage of this simple technique for improving gait performance. Before proposing the vibration procedure described here for extended clinical trials, the effects of vibration at different frequencies or of continuous vibration, or the possibility of a placebo effect, remain to be investigated. Controlled clinical trials should also assess whether off-phase patients are susceptible to vibration as well.

Acknowledgements

This investigation was supported by the Grants PRIN 2007 from the Italian Ministry of Education and Ricerca Finalizzata 2003 and 2005 from the Italian Ministry of Health. None of the authors has financial interests or conflicts of interests. We wish to thank the anonymous Referees for their helpful comments.

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