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Gait initiation characteristics in elderly patients with unilateral vestibular impairment

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ABSTRACT

The study tested the hypothesis that vestibular patients ($n = 14$) with chronic unsteadiness caused by a documented peripheral unilateral vestibular dysfunction would display differences in muscular activation and movement pattern during gait initiation compared to age-, gender- and body-size-matched healthy Controls ($n = 14$). The displacements of the whole body Center of Pressure (CoP) during the preparatory phase before the swing leg is lifted, were markedly different in vestibular patients. The backward shift during this phase was significantly smaller than in Controls, coupled with a larger secondary corrective forward shift of the CoP. Conversely, the CoP-shift in the M-L direction towards the stance leg was larger in the vestibular patients. Most vestibular patients lacked the anticipatory tibialis anterior (TA) burst, which normally is a prerequisite for the backward displacement of the CoP that precedes the forward movement. The vestibular patients displayed more pronounced TA-Gastrocnemius coactivation in the stance leg when the swing leg was lifted. The duration of the preparatory phase was significantly longer in vestibular patients than in Controls, with no time differences in the later gait initiation events. The vestibular patients started from a more symmetrical stance and with less M-L variation than the Controls. It is concluded that chronically impaired vestibular function leads to a different strategy to create forward momentum to the body. In addition, there is evidence that vestibular patients have diminished postural stability, or alternatively a more cautious behaviour, when initiating the second step.

1. Introduction

Unsteadiness ranks among the most common complaints in medicine, affecting approximately 20–30% of the general population [1] and has a large impact on daily life [2]. The most common cause of persistent unsteadiness (65%) is a unilateral vestibular impairment [3]. There is limited information about the impact of vestibular impairments on postural control during intentional movements [4], although postural hypermetria (larger than normal postural responses) has previously been reported in vestibular patients [5].

Gait initiation (GI) involves change from a stable support with two legs on the ground to an unstable posture in the form of weight displacements and transition to single leg support. Therefore, GI may be challenging enough to disclose postural hypermetria. This could be suggested by a previous study of bilateral labyrinthectomy in the cat [6], where the compensatory movement to regain balance following a backward perturbation, occurred earlier, at a smaller displacement of the support surface and with a marked overshoot compared to controls.

That vestibular information is important during GI is supported by experiments where galvanic vestibular stimulation (GVS) was delivered to healthy subjects during different phases of GI [7]. GI-alterations hitherto reported with GVS are deviations in the upper body roll response and in first step foot placement [7], but it has been shown that whole body CoP is displaced when GVS is delivered during upright stance [8]. The direction of the CoP displacements in the sagittal and frontal planes are dependent on the position of the anode and on other factors [9,10]. Although the studies using GVS are not directly comparable to the present research setting, the GVS data make it likely that the CoP displacements occurring during GI would be altered by vestibular dysfunction.

Therefore, CoP displacements during GI was compared between vestibular patients with chronic unsteadiness caused by a documented peripheral unilateral vestibular dysfunction and

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Table 1
Subject characteristics, basal gait initiation variables, times of gait initiation events, EMG onset and offset times and the side of the swing leg (leading limb) in relation to the side of the vestibular impairment.

Variable name	Controls ($n = 14$)	Vestibular patients ($n = 14$)	Statistical significance
	Mean (SEM)	Mean (SEM)	*F*-test
Age, yrs	73.6 (0.8)	73.6 (1.6)	n.s.
Weight, kg	70.6 (3.0)	71.4 (2.8)	n.s.
Height, cm	164.4 (2.4)	169.6 (2.8)	n.s.
Initial standing, variability (SD) VGRF, %BW[a]	0.32 (0.02)	0.22 (0.02)	$F_{(1,26)} = 7.93$, $P = 0.009$
Initial standing, variability (SD) CoP, A-P, mm	1.5 (0.1)	1.7 (0.2)	n.s.
Initial standing, variability (SD) CoP, M-L, mm	0.63 (0.04)	0.44 (0.05)	$F_{(1,26)} = 8.13$, $P = 0.008$
Initial standing, weight on stance leg, %BW	53.9 (1.1)	50.0 (1.4)	$F_{(1,26)} = 4.82$, $P = 0.037$
1st step length, % body height	33.9 (0.9)	29.1 (0.9)	$F_{(1,26)} = 8.03$, $P = 0.009$
Reaction time, ms	178.6 (9.1)	251.5 (19.4)	$F_{(1,25)} = 12.18$, $P = 0.002$
Stance width, mm	162.8 (3.8)	176.2 (9.3)	n.s.
Duration of G.I., ms	1017.8 (15.1)	1177.4 (44.0)	$F_{(1,26)} = 11.76$, $P = 0.002$
Time, %GI			
Foot off, swing leg	46.1% (0.9)	51.3% (0.8)	$F_{(1,26)} = 18.10$, $P = 0.0002$
Foot contact, swing-to- stance leg	78.7% (0.8)	80.9% (0.8)	n.s.
Foot off, stance leg	100.0% (1018 ± 15 ms)	100.0% (1177 ± 44 ms)	
EMG, % GI			
Swing leg, TA on	−3.6 (1.1)	−0.30 (0.9)	$F_{(1,26)} = 5.85$, $P = 0.023$
Swing leg, TA off	35.3 (8.4)	38.1 (6.2)	n.s
Swing Leg, LG on	47.8 (8.4)	43.1 (5.3)	n.s
Stance leg, TA on	−3.0 (1.2)	−0.74 (0.8)	n.s
Stance leg, TA off	47.1 (2.9)	57.8 (3.8)	$F_{(1,26)} = 5.05$, $P = 0.033$
Stance leg, LG on	43.3 (2.7)	46.5 (3.0)	n.s
Of total 5 GI trials			
All with right leg	8	5	
All but one with right leg	2	4	
All with left leg	2	2	
All but one with left leg	0	1	
Mixed right and left legs	2	2	
All with leg on affected side		3	
All but one with leg on affected side		2	
All with leg on healthy side		4	
All but one with leg on healthy side		3	
Mixed affected/healthy side		2	

[a] The value was identical in the swing and stance leg.

healthy Controls. EMG registrations gave information about the calf muscle activation pattern that underlies the CoP displacements. CoP displacements and leg loading during initial standing provided insight into possible differences in posture before GI. Data on reaction time, durations of the different GI-phases, step length, stance width and the timing of ground reaction forces provided insight into possible alternative movement strategies used by the vestibular patients to perform GI, the latter variable also giving information into postural stability during GI.

2. Materials and methods

2.1. Subjects

The vestibular patient group (Patients) consisted of 10 females and 4 males recruited from the Department for Hearing- and Balance disorders at the Karolinska University Hospital (Table 1). Inclusion criteria were a history of sudden onset of vertigo caused by unilateral vestibular impairment followed by persistent unsteadiness, increased by head movements and present for at least one year. No spontaneous attacks of vertigo occurred during the last year. The diagnoses/symptoms were vestibular neuritis (nine), sudden onset of vertigo in combination with sudden ipsilateral loss of hearing (three) and Menieres disease (one bilateral, only active on one side; one previously treated with intratympanal gentamicin to eliminate vestibular function on the affected side). The assessment of vestibular function was based on the outcome in three tests, either alone or in their combination. The caloric test (reduced caloric response from the semicircular canal on one side/ear, quantified by the speed of the nystagmus' slow component), the head impulse test (lack of gaze stabilization indicating reduced vestibular function for the ear ipsilateral to the head thrust) and the utriculus test (incorrect subjective visual horizontal in darkness indicating impairment of utricular function on the side towards which the horizontal deviates). Eight Patients had right- and 6 left-sided vestibular impairment. Three Patients had spontaneous nystagmus. In 10 Patients, nystagmus could be elicited after head shake. None had medication that might interfere with normal balance or gait or any history of CNS disease or peripheral neuropathy affecting the lower extremities. Patients with auditory disorders were examined with auditory brain stem response or MRI to exclude retrocochlear pathology. All walked freely without aid/support.

The healthy elderly Controls (Controls) comprise 10 females and 4 males, recruited from pensioners' organizations (Table 1) and without any history of neurological illness, degenerative conditions or any general disease, or medication that might interfere with normal balance or gait. All walked freely and without aid/support. Foot length was identical in Patients and Controls, 15.2 ± 0.2% and 14.6 ± 0.1% of body height in men and women, respectively.

The study was approved by the Ethics Committee of the Karolinska University Hospital, Stockholm.

2.2. Procedure

The starting instruction was to stand as normally as possible, arms hanging by the sides and each foot resting on a separate force platform and, as a traffic light turned from red to green, initiate normal cross-the road-walking in 5 consecutive trials with free choice of the leading limb. Foot position was traced to maintain the same initial stance for all subsequent trials.

2.3. Equipment

A walkway (10 m × 2 m), built in the laboratory constituted a fictive street crossing (without a curb). A traffic light system, connected to the A/D box, was placed at the end.

2.4. Force and Center of Pressure (CoP)

Ground reaction forces (GRFs) were registred with 4 AMTI force platforms [11]. Whole body antero-posterior and medio-lateral CoP displacements were calculated from the two initial standing force plates. Variability (forces and CoP) during initial standing was calculated as the SD of the 50 values (at 100 Hz) registered immediately before the traffic light turned from red to green.

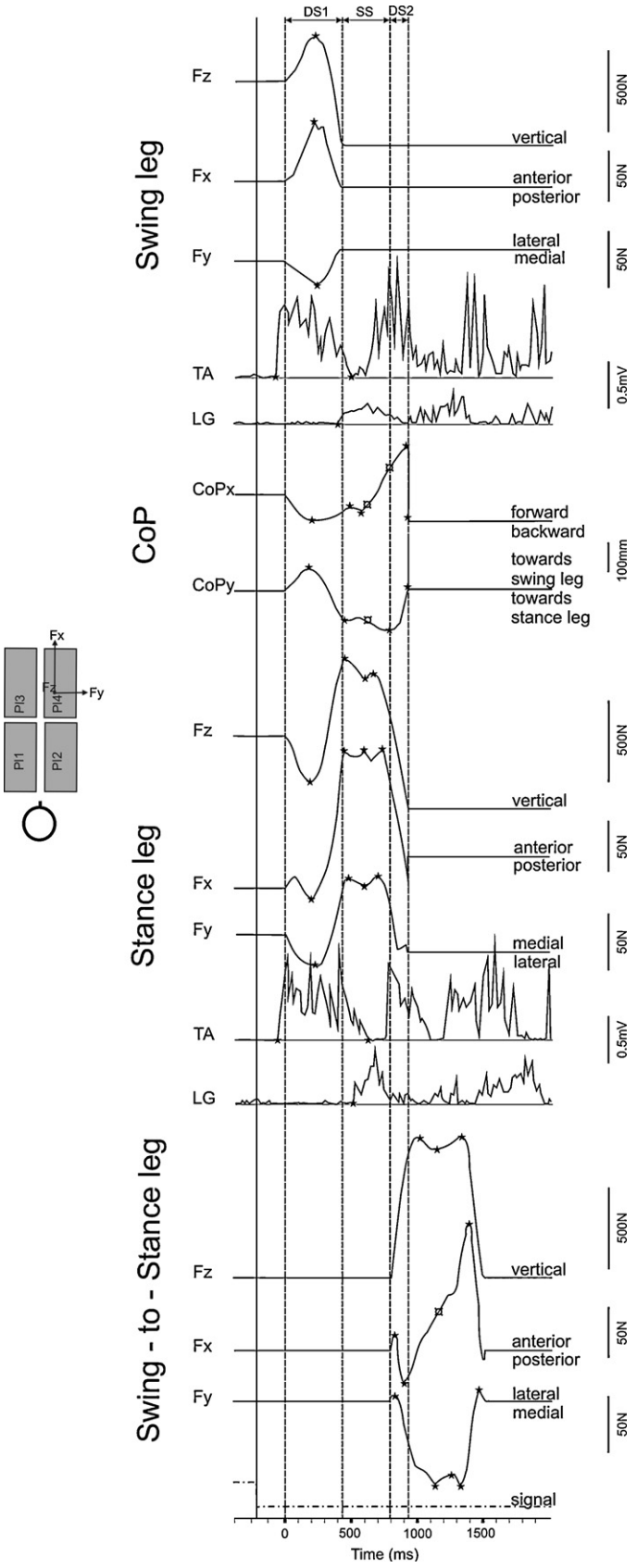

Fig. 1. A schematic figure of the recording of the vertical (Fz), antero-posterior (Fx) and medio-lateral (Fy) ground reaction forces (GRF), tibialis anterior (TA) and lateral gastrocnemius (LG) EMG activity and the displacements of the whole body Center of Pressure (CoPx, CoPy) in connection with gait initiation in an older adult with unilateral vestibular impairment. Signal (bottom tracing) denotes the time point at which the visual start cue (green traffic light) was provided. Time 0 is the onset of change in Fz_{SwL} (uppermost curve). The points of measurements are indicated on the curves and are summarized in Table 2: ★ denotes position on the curves (peaks or lowest points, start, stop); ¤ denotes measurements at defined time-points (times C, D and F as defined in Table 2). The small figure at the left indicates the relative positions of the force plates.

2.5. Electromyography

Surface electromyography with 800 Hz sampling frequency was collected from tibialis anterior (TA) and lateral gastrocnemius (LG) muscles bilaterally as described previously [9]. Onset and offset of muscular activity were defined in relation to the baseline EMG-level, determined during 200 ms, plus two standard deviations. Onset (offset) was when the EMG burst first exceeded (returned to) this level to be maintained for at least 30 ms [12]. TA-LG co-activation was the time interval (from LG onset to TA offset, normalized as % of GI) during which both muscles were determined to be active. EMG amplitude (mean average voltage) was the integrated EMG divided by the integration time.

2.6. Kinematics

An eight-camera optoelectronic system (Elite BTS, Milan, Italy) with spherical reflective markers, placed on heels and lateral malleoli were used to measure step length (lateral malleolus marker, antero-posterior displacement, initial standing to heel-strike) and stance width (medio-lateral distance between the heel markers).

2.7. Data analysis

Data were recorded for 7 s, starting 1 s before each trial. The ASCII files of GRF data, EMG and kinematics were analyzed manually in AxoGraph 4.9 (Axon Instruments, Inc., Foster City, CA, USA). Reference time for all variables was the first detectable increase in vertical force exerted by the swing leg (Fig. 1, uppermost curve). The sequence of events between this time (time 0) and foot off, stance leg was defined as the gait initiation (GI). The total GI duration (100% GI) was divided into three phases; Double-Stance 1 (DS1), Single-Stance (SS) and Double-Stance 2 (DS2), see Fig. 1. Reaction time (ms) is the interval between green light and time 0 (Fig. 1). CoP-displacements are given in mm; GRFs are normalized as %body weight (%BW); EMG on- and offset times and times of measurement of force and CoP-values are normalized as % of the total duration of gait initiation (%GI).

2.8. Statistics

One-way and Factorial Analyses of Variance were used (Statistica 9, StatSoft Inc., Tulsa, OK), following tests for normality by Shapiro–Wilk's W test. Significance level was $P < 0.05$. In factorial ANOVA, group differences were identified by significant interactions between the dependent variable and the categorical predictor (Patients or Controls). Data are means ± SEM, where the value for each subject was the average of the 5 GI trials.

3. Results

3.1. Initial standing

Controls supported themselves more on the forthcoming stance than the swing leg (Table 1). Although identically instructed, the Patients instead displayed weight equilibrium between the legs and exhibited less variability both in vertical loading of the legs and in medio-lateral displacements of the CoP (Table 1).

3.2. Gait initiation

The side of the swing leg in relation to the side of vestibular impairment is presented in Table 1. Patients had longer reaction time and GI duration, but shorter first step length. Four Patients had a stance width above 20 cm (Controls, range 14.2–18.7 cm), although the mean value did not differ between groups (Table 1). DS1 was longer in Patients than in Controls, but the following phases were of equal duration, Table 1.

3.3. Displacement of the whole body Center of Pressure (CoP)

In preparation for the first step, CoP moves backwards and sidewards (towards the forthcoming swing leg). The backward displacement was smaller in Patients (Fig. 2a, A–B). Thereafter, but before the swing leg leaves the ground, there is a slight forward CoP

Table 2
Time points used as references for movements and Points of measurement.

Time/event	Symbol/interval
First detectable increase in vertical force exerted by the swing leg	0 (zero)
Reaction time	Green light to 0
100% gait initiation	0-E
Maximal vertical force, swing leg	A
Foot off, swing leg	B
Lowest vertical force during SS, stance leg	C
Foot contact, swing leg	D
Foot off, stance leg	E
Lowest vertical force, swing-to-stance leg	F
Foot contact, stance-to-swing leg	G
Double-Stance Phase 1 (DS1)	0-B
Single-Stance Phase (SS)	B–D
Double-Stance Phase 2 (DS2)	D–E
Single-Stance Phase 2 (SS2)	E–G

Variable	Points of measurement
Whole body CoP, antero-posterior displacement	Maximal backward during DS1
	Next maximal forward displacement
	Next maximal backward displacement
	Time point C
	Time point D
	Maximal forward displacement
Whole body CoP, medio-lateral displacement	Maximal towards swing leg during DS1
	Final displacement towards stance leg
	Time point C
	Maximal displacement towards stance leg
Vertical GRF, swing leg	Time point A
Vertical GRF, stance leg	Time point A
	1st force peak
	Time point C
	2nd force peak
Vertical GRF, swing-to-stance leg	1st force peak
	Time point F
	2nd force peak
Antero-posterior GRF, swing leg	Anterior force peak during DS1
Antero-posterior GRF, stance leg	Posterior force peak during DS1
	1st anterior force peak during SS
	2nd anterior force peak during SS
	3rd anterior force peak during SS
Antero-posterior GRF, swing-to-stance leg	Anterior force peak during DS2
	Posterior force peak during DS2
	Time point F
	Anterior force peak during 2nd DS2
Medio-lateral GRF, swing leg	Medial force peak during DS1
Medio-lateral GRF, stance leg	Lateral force peak during DS1
	1st medial force peak during SS
	Lowest medial force during SS
	2nd medial force peak during SS
Medio-lateral GRF, swing-to-stance leg	Lateral force peak during DS2
	1st medial force peak during 2nd SS
	Lowest medial force during 2nd SS
	2nd medial force peak during 2nd SS
	Lateral force peak during 2nd DS2

displacement (as the CoP in the medio-lateral direction crosses the mid-line to move towards the stance leg). This forward CoP-displacement was exaggerated in the Patients (statistical interaction, forward movement*group, $P < 0.001$). Thereafter the CoP moved backwards again in the Patients until the swing leg left the ground, whereas a smaller (not statistically significant) backward CoP displacement was observed in the Controls (statistical interaction, backward movement*group, $P < 0.05$). During SS and DS2, the CoP moves forwardly, a movement which is larger in the Patients (Fig. 2a). At the same time, in the sideways direction, the CoP has been displaced to reach its position within the boundaries of the stance foot. This position was significantly more lateral in Patients than in Controls (Fig. 2b). CoP displacements displayed no trend towards an influence of whether the swing leg was ipsi- ($n = 5$) or contralateral ($n = 7$) to the vestibular defect. There was also no side difference in Patients having both right and left leg GI-trials ($n = 7$).

3.4. Ground reaction forces (GRFs)

The forwardly directed (propulsing) GRF's of the initial stance leg were lower in Patients (Fig. 3b). In addition, there were significant differences in the timing of the GRF's as explained in the legend to Fig. 3 (and marked by asterisks in Fig. 3a–c). These indicate that Patients obtained maximal loading of the stance leg during SS2 at a more unstable (single-stance) position compared to Controls (Fig. 3a: ^{*2}; Fig. 3b: ^{*4}; Fig. 3c: ^{*3}). The latter may be in accordance with the maximal forward CoP-displacement occurring later in the Patients (Fig. 2a).

3.5. EMG

No initial inhibition of LG was evident in either group. The burst of activity in TA of the swing leg occurred earlier in Controls than in Patients ($P < 0.05$, Table 1). The stance leg displayed a similar pattern (N.S.). When the electro-mechanical delay (time from EMG-burst to muscle contraction; approximately 36 ms or 3% GI [13,14]) is added to the TA-onset times, anticipatory TA contraction was found to occur in both the swing and stance legs in the majority of Controls, but only in 2/14 Patients. Stance leg TA-offset occurred later in Patients ($P < 0.05$) and, contrary to Controls, generally into SS. TA-LG co-activation was enhanced in Patients compared to Controls, especially in the stance leg (Table 1). EMG amplitudes (mean average voltage) were similar during the periods with (TA: 0.22 ± 0.03 mV; LG: 0.43 ± 0.25 mV) and without (TA: 0.27 ± 0.03 mV; LG: 0.38 ± 0.16 mV) co-activation, thus indicating that the co-activation period involved significant force contributions from both muscles.

4. Discussion

The initial backward shift of the whole body CoP during GI creates a large enough gap between the position of the CoP and the projection onto the floor (the center of gravity) of the center of mass in order to create forward momentum to the body [15]. This shift is decreased in healthy elderly compared to young individuals [16,17] and in frail elderly compared to healthy elderly [16], accompanied by a slower gait speed [16,18]. Decreased CoP-shifts also in the M-L direction, both towards the swing and stance legs, have been reported in healthy elderly compared to young individuals [19].

The similarity between these reports and the results of the present study may suggest that the decreased backward shift of the CoP and possibly other previously reported differences in GI parameters between healthy elderly and young individuals [11] to some extent is explained by an aging-induced impairment within the peripheral vestibular system, e.g. secondary to loss of vestibular hair and nerve cells [20,21].

A notable finding in the Patients was that the lower initial backward shift of the CoP was followed by a forward shift of the CoP combined with a secondary backward CoP-shift, both being more pronounced than in Controls. This pattern of a compensatory movement that occurs earlier and at a smaller displacement of the CoP than in Controls, and in addition with an overshoot, resembles the postural hypermetria that was previously observed after experimental labyrinthectomy [6]. Although the neural mechanism by which vestibular impairment may induce hypermetria is unclear [6], this large forward CoP-shift may be related to the postural hypermetria previously found in vestibular patients [5]. This may possibly constitute a corrective response because the initial backward CoP-shift was perceived as too destabilizing [22]

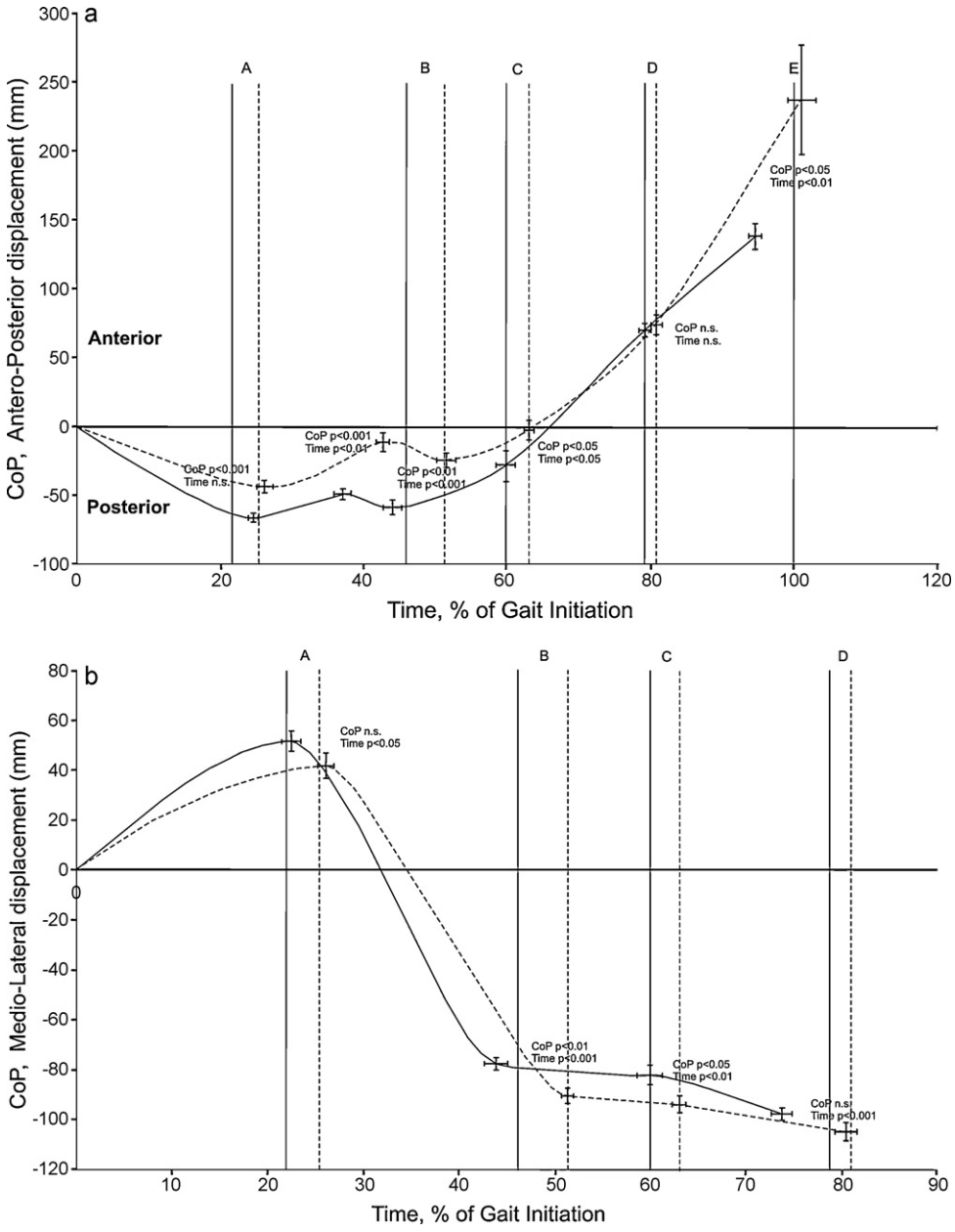

Fig. 2. Mean values ± SEM of the displacements of the whole body Center of Pressure (CoP) during Gait initiation (GI) for vestibular patients (broken lines, $n = 14$) and Controls (solid lines, $n = 14$). (a) CoPx (antero-posterior direction, see schematic tracing in Fig. 1 with the 6 points of measurement indicated as summarized in Table 2). Positive values are CoP displacements forwardly. (b) CoPy (medio-lateral direction, see schematic tracing in Fig. 1 with the 4 points of measurement indicated). Positive values are CoP displacements towards the initial swing leg. The times of the points of measurement for vestibular patients and Controls, respectively, are expressed in per cent of the total GI-time (mean ± SEM in the horizontal direction). Lines A–E, solid lines for Controls and broken lines for vestibular patients, are inserted for reference only to indicate the different phases of the GI-cycle as explained in Table 2.

and tantamount to a "decreased smoothness" of the A-P CoP trajectory noted in older adults with Parkinson's disease [18]. Other ways to obtain higher forward momentum in spite of a lower backward CoP-shift would be to prolong the time of postural adjustment [23] or to initiate walking by swaying forward [13]. That both of these strategies were used in the Patients, is supported by the longer time to foot off, swing leg and the shorter step length than in the Controls. It could be speculated whether, with unilateral vestibular impairment, there would be some imbalance in the CoP displacements, e.g. towards the less affected side. However, we found no trend towards a difference when the leg on the affected side was the swing or the stance leg, respectively. Nevertheless, it cannot be excluded that additional more directed explorations of a possible side difference in this respect could provide valuable insight into the vestibular influence on GI.

The backward shift of the CoP is normally dependent on one of two distinct EMG-events, the anticipatory motor sequence [15], occurring in both legs before there is detectable movement of the body. These include the inhibition of ankle plantarflexors (mm. soleus and gastrocnemius) and, approximately 100 ms later, a burst of activity in the tibialis anterior (TA) muscle [15]. No inhibition of gastrocnemius was found in either group. In contrast, anticipatory TA contraction was found to occur in both the swing and stance legs in the majority of the Controls, but only in 2/14 Patients. Still the backward CoP-shift started immediately from time zero in 11 of the 14 Patients (and in all

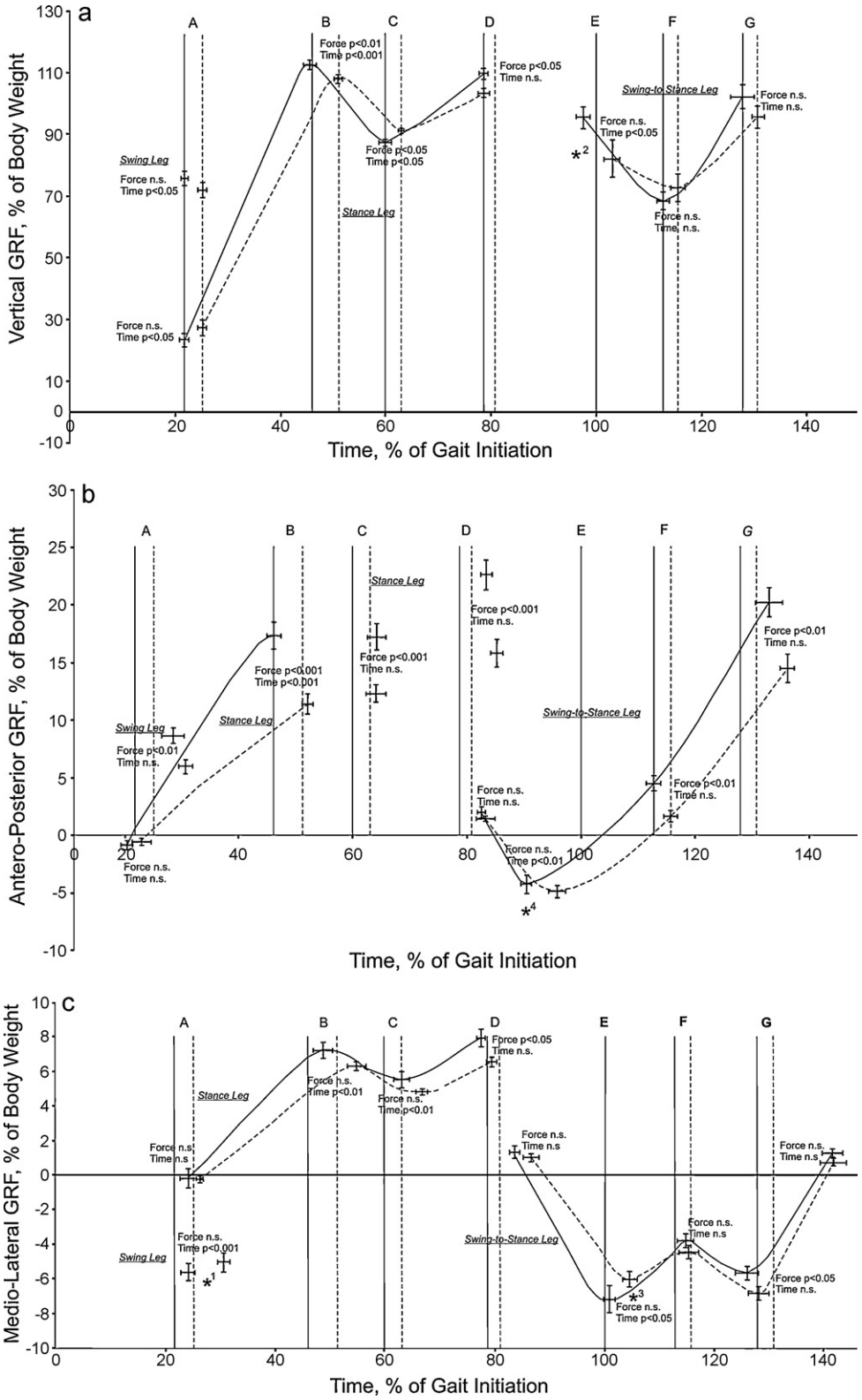

Fig. 3. Mean values ± SEM of vertical (a), antero-posterior (b) and medio-lateral (c) ground reaction forces (GRF's) during Gait initiation (GI) for vestibular patients (broken lines, $n = 14$) and Controls (solid lines, $n = 14$). For a, b and c, see schematic tracings (Fz, Fx and Fy) in Fig. 1 with the points of measurement indicated. The last three force values of the stance leg (b) are not connected by a line, since all are peak forces with lower values in between. * denotes differences between vestibular patients and Controls in the timing of the GRF's (statistical interaction time * Group, $P < 0.05$): *1, c: Peak medio-lateral GRF of the swing leg (during DS1) occurred more delayed relative to the maximal vertical load of this leg (the A-lines) in vestibular patients than in Controls; *2, a and *3, c: Peak vertical GRF and peak medio-lateral GRF at the end of DS2 occurred into the next single-stance phase (i.e. after line E) in the vestibular patients but not in the Controls *4, b: Peak antero-posterior GRF of the initial swing leg occurred significantly closer to the end of DS2 in the vestibular patients than in the Controls.

Controls). The absence of both muscle-activation events which are generally considered to be a prerequisite for the backward CoP-shift, indicates a different strategy to secure the backward CoP-shift that is necessary to initiate gait. One possibility is a rapid unilateral rotating arm movement (forward-outward followed by backward-inward), which we noted in several of the Patients. At foot-off of the swing leg, TA activity in the stance leg of Patients, unlike in Controls, was never silenced before the gastrocnemius had been fully activated. This therefore indicates a stabilizing function of TA in Patients, being different from controls.

The timing of the GRFs indicate that Patients had diminished postural stability at the start of SS2, the maximal loading of the stance leg occurring at a more unstable (single-stance) position compared to Controls. Alternatively, the Patients use a more cautious strategy than Controls in this situation, where the smaller propulsive forces may lead to both the heel-strike of the initial swing leg and the maximal forward CoP-displacement occurring later. It is possible that the higher degree of TA-LG coactivation in Patients, with prolonged TA-activation in the stance leg, may have offered increased stability of the ankle joint at the price of counteracting the propulsive force generated by the LG muscle [24]. A more cautious behaviour is supported by the Patients starting from a more symmetrical stance, swaying less in the M-L direction. Similar data of shrinkage of the area of perceived stability (i.e. the maximal displacement of the CoP around the center of gravity) in the M-L direction have been reported in chronic phobic vertigo in combination with a disturbed vestibular function [25].

It is concluded that chronically impaired vestibular function leads to a different strategy to create forward momentum to the body. In addition, there is evidence that this condition leads to a diminished postural stability, or alternatively a more cautious behaviour, at the start of SS2. It remains to be investigated whether the observed alterations may be reversed by vestibular rehabilitation, or alternatively, that the changes represent a necessary compensation for the vestibular deficit.

Acknowledgments

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Conflict of interest statement

There is no conflict of interest for any of the authors regarding this article.

References

[1] Nazareth I, Yardley L, Owen N, Luxon L. Outcome of symptoms of dizziness in a general practice community sample. Fam Pract 1999;16:616–8.
[2] Mendel B, Lutzen K, Bergenius J, Bjorvell H. Living with dizziness: an explorative study. J Adv Nurs 1997;26:1134–41.
[3] Bath AP, Walsh RM, Ranalli P, Tyndel F, Bance ML, Mai R, et al. Experience from a multidisciplinary "dizzy" clinic. Am J Otol 2000;21:92–7.
[4] Sasaki O, Asawa S, Katsuno S, Usami S, Taguchi K. Gait initiation in bilateral vestibular loss. Auris Nasus Larynx 2001;28:295–9.
[5] Horak FB. Postural compensation for vestibular loss. Ann N Y Acad Sci 2009;1164:76–81.
[6] Inglis JT, Macpherson JM. Bilateral labyrinthectomy in the cat: effects on the postural response to translation. J Neurophysiol 1995;73:1181–91.
[7] Bent LR, McFadyen BJ, Inglis JT. Is the use of vestibular information weighted differently across the initiation of walking? Exp Brain Res 2004;157:407–16.
[8] Hlavacka F, Horak FB. Somatosensory influence on postural response to galvanic vestibular stimulation. Physiol Res 2006;55(Suppl. 1):S121–7.
[9] Grasso R, Ivanenko Y, Lacquaniti F. Time course of gaze influences on postural responses to neck proprioceptive and galvanic vestibular stimulation in humans. Neurosci Lett 1999;273:121–4.
[10] Severac Cauquil A, Martinez P, Ouaknine M, Tardy-Gervet MF. Orientation of the body response to galvanic stimulation as a function of the inter-vestibular imbalance. Exp Brain Res 2000;133:501–5.
[11] Henriksson M, Hirschfeld H. Physically active older adults display alterations in gait initiation. Gait Posture 2005;21:289–96.
[12] Hirschfeld H. Do infants have motor responses to sudden surface rotations in prone position? J Vestib Res 1997;7:265–76.
[13] Corcos DM, Gottlieb GL, Latash ML, Almeida GL, Agarwal GC. Electromechanical delay: an experimental artifact. J Electromyogr Kinesiol 1992;2:59–68.
[14] Norman RW, Komi PV. Electromechanical delay in skeletal muscle under normal movement conditions. Acta Physiol Scand 1979;106:241–8.
[15] Crenna P, Frigo C. A motor programme for the initiation of forward-oriented movements in humans. J Physiol 1991;437:635–53.
[16] Halliday SE, Winter DA, Frank JS, Patla AE, Prince F. The initiation of gait in young, elderly, and Parkinson's disease subjects. Gait Posture 1998;8:8–14.
[17] Polcyn AF, Lipsitz LA, Kerrigan DC, Collins JJ. Age-related changes in the initiation of gait: degradation of central mechanisms for momentum generation. Arch Phys Med Rehabil 1998;79:1582–9.
[18] Hass CJ, Waddell DE, Wolf SL, Juncos JL, Gregor RJ. Gait initiation in older adults with postural instability. Clin Biomech (Bristol Avon) 2008;23:743–53.
[19] Martin M, Shinberg M, Kuchibhatla M, Ray L, Carollo JJ, Schenkman ML. Gait initiation in community-dwelling adults with Parkinson disease: comparison with older and younger adults without the disease. Phys Ther 2002;82:566–77.
[20] Rauch SD, Velazquez-Villasenor L, Dimitri PS, Merchant SN. Decreasing hair cell counts in aging humans. Ann N Y Acad Sci 2001;942:220–7.
[21] Rosenhall U. Degenerative patterns in the aging human vestibular neuroepithelia. Acta Otolaryngol 1973;76:208–20.
[22] Krishnamoorthy V, Latash ML, Scholz JP, Zatsiorsky VM. Muscle synergies during shifts of the center of pressure by standing persons. Exp Brain Res 2003;152:281–92.
[23] Couillandre A, Breniere Y, Maton B. Is human gait initiation program affected by a reduction of the postural basis? Neurosci Lett 2000;285:150–4.
[24] Shumway-Cook A, Woollacott M. Motor Control. Theory and Practical Applications, 2nd Ed. Lippincott, Philadelphia: Williams & Wilkins; 2001.
[25] Ödman M, Maire R. Chronic subjective dizziness. Acta Otolaryngol 2008;128:1085–8.