VISUO-VESTIBULAR INFLUENCES ON THE MOVING PLATFORM

LOCOMOTOR AFTEREFFECT

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**ABSTRACT**

After walking onto a moving platform subjects experience a locomotor aftereffect (LAE), including a self-generated stumble, when walking again onto a stationary platform. Thus, this LAE affords examination of the role of vestibular input during an internally generated postural challenge. The experiments involved, walking onto the stationary sled (BEFORE trials), walking onto the moving sled (MOVING) and a second set of stationary trials (AFTER). We investigated 9 bilateral labyrinthine defective subjects (LDS) and 13 age-matched normal controls (NC) with eyes-open. We repeated the experiment in 5 NC and 5 LDS but this time the AFTER trials were performed twice, first eyes-closed and then on eye re-opening. During MOVING trials, LDS were considerably unstable thus confirming the established role of the vestibular system during externally imposed postural perturbations. During AFTER trials, both groups experienced an aftereffect with eyes-open and closed, shown as higher approach gait velocity, a forward trunk overshoot and increased leg EMG. However, there were no significant group differences due to the fact that stopping the forwards trunk overshoot was accomplished by anticipatory EMG bursts. On eye re-opening the aftereffect re-emerged, significantly larger in LDS than in NC. The lack of group differences in AFTER trials suggests that when facing internally-generated postural perturbations, as in this adaptation process, the CNS relies less on vestibular feedback and more on anticipatory mechanisms. Re-emergence of the aftereffect on eye re-opening indicates the existence of a feedforward visuo-contextual mechanism for locomotor learning, which is adaptively enhanced in the absence of vestibular function.
INTRODUCTION

It is well established that the vestibular system participates in postural reactions in response to head or support surface motion and, accordingly, humans devoid of vestibular function become unstable when facing unexpected postural perturbations (Ito et al 1995; Pozzo et al 1989; 1995; Carpenter et al 2001; Allum et al 2001; Allum and Pfaltz 1985; Allum and Honegger 1998; Beule and Allum 2006, Horak et al 1990; Buchanan and Horak, 2001). However, in regular everyday life activities, bilaterally labyrinthine defective subjects (LDS) function with only a moderate degree of impairment (Grunfeld et al 2000). This discrepancy may be explained by the use of anticipatory motor mechanisms in predictable conditions, whereby activation of postural muscles occurs in advance of a postural perturbation (Belenkiy et al 1967).

Anticipatory postural adjustments have been mostly examined in simple experimental situations, e.g. rapidly raising the arms whilst observing EMG activity in the lower limbs. These experiments show lower limb activation which is earlier than upper limb activation, i.e. the legs ‘anticipate’ the instability that will be induced by moving the arms (Belenkiy et al 1967; Bouisset and Zattara 1981; Horak et al 1984; Brown and Frank 1987; Friedli et al 1988). More recently, interest has shifted towards anticipatory mechanisms as they supposedly occur in everyday life (Cham and Redfern 2002; Marigold and Patla 2002; Pai et al 2003; Pavol and Pai 2002; Pijnappels et al 2001). We have recently studied a case in point, the ‘broken escalator’ phenomenon, characterised by an odd sensation of imbalance and stumble when walking onto an escalator which is stationary, despite full knowledge that the escalator will not move (Reynolds & Bronstein 2003). Experimentally, we
reproduced a similar scenario by asking subjects to walk onto a moving motorised sled and concluded that the ‘broken escalator’ phenomenon is an aftereffect of locomotor adaptation. Following locomotor adaptation on the moving sled (MOVING condition) subjects approach the stationary sled (AFTER condition) inappropriately fast and they experience a large forward overshoot of the trunk (see ‘Linear Fastrak’ Fig. 1).

An interesting aspect of this aftereffect is that it occurs despite unequivocal warnings that the sled will not move - an example of dissociation between declarative (cognitive) and executive (motor) systems in the CNS (Reynolds and Bronstein 2003; 2004; Flanagan & Beltzner 2000; Haffenden et al 2001). The motor behaviour expressed in this locomotor aftereffect (LAE), namely walking faster and leaning forwards, is exactly what would be needed if the sled were to move, a ‘just in case’ strategy (Bunday et al 2006). However, because the sled does not move, the LAE represents an inappropriate release of anticipatory motor behaviour (Reynolds and Bronstein 2004) which, in turn, induces postural instability.

Herewith, we report experiments with the LAE paradigm in LDS. On the basis that these patients have intact proprioceptive and CNS function we expected that locomotor adaptation would occur (Keshner et al 1987) and therefore LDS would display an aftereffect. However, a question addressed here is concerned with the ‘braking’ of the trunk overshoot, that is, what mechanisms (‘brakes’) stop subjects falling forwards once they have been self-destabilised by the aftereffect? In this paper we test bilaterally labyrinthine defective subjects (LDS) to see how, in the absence of vestibular function, they ‘brake’ this component of the aftereffect. More generally,
the question investigated is how complex anticipatory postural behaviour is modified by the absence of sensory (vestibular) feedback. A prediction was that LDS would have a larger than normal trunk movement aftereffect, that is, a bigger forward overshoot of the trunk in the AFTER trials due to the absence of vestibular function. However, if the braking during the aftereffect was also the result of anticipatory motor mechanisms, no difference between normal and LDS should be found.

In addition, since visual input is critical for balance in the absence of vestibular function we carried out additional experiments, in normal and LD subjects, to investigate the effects of vision on this LAE. In contrast to vestibular function, which may only play a part in ‘braking’ the aftereffect, vision could play a part both in the release of the aftereffect (e.g. visual confirmation that one is indeed on the sled) and, particularly in LDS, in ‘braking’ the aftereffect.
METHODS

Materials
The sled was powered by two linear induction motors, at a maximum velocity of 1.3m/s, and enveloped by a fixed platform under which it could freely pass (Fig. 1 top left). Movement of the sled was triggered by gait initiation via an infrared light switch (Reynolds and Bronstein 2003). On walking through the sensor, after a 600 ms delay, the sled would travel approximately 3.7m in 4.2s (maximum velocity reached by 896ms). A tachometer provided velocity output.

Subject sagittal trunk position was measured using a Fastrak electromagnetic tracking device (Polhemus, VT, USA), accurate within 0.08cm RMS for the receiver position. The sensor was placed over the C7 vertebrae and the transmitter was attached to the sled. A second sensor was earth-fixed so that the movement of the sled could be accounted for when calculating approach (trunk) velocity. Step timing information was given by pressure sensors (Flexiforce, Teckscan, MA, USA) placed under the first metatarsal phalangeal joint and heel. A linear sled-mounted accelerometer (Entran, Watford, UK) provided independent accurate foot-sled contact timing. Superficial bipolar EMG (bandpass filtered 10-500Hz) from the medial gastrocnemius (MG) and tibialis anterior (TA) muscles of each leg was also recorded in the LDS-NC experiments. All data were recorded at 500Hz.

Procedure

Experiment 1
This experiment was conducted to see the effect of vestibular loss on the LAE. Nine LDS, ranging from 42-69yrs [mean=57.7±3.0yrs (S.E.)], were tested. All patients were in chronic compensated phase and able to walk unassisted with eyes open. Rotational and caloric nystagmic responses in the dark were reduced to <10% of normal (Rinne et al 1999); aetiology was idiopathic (6), gentamicin toxicity (1), post meningitic (1), posttraumatic/idiopathic (1). Time since vestibular failure was 2-15 years. An age matched normal control (NC) group of 13 subjects, ranging from 43-66yrs (mean=57.5±1.7yrs), was tested. All normal controls were healthy and reported no history of neurological illness.

The experimental sequence consisted of conditions BEFORE, MOVING and AFTER, with eyes open throughout (Fig.1 bottom left). Subjects completed baseline trials walking 5 times onto the stationary sled (BEFORE condition). Gait was initiated after an auditory cue (3 ‘beeps’); they stepped with the right leg first onto the fixed platform, then with the left on the sled. Here they stopped and adopted a quiet stance with both feet on the sled, until recording was completed. Each trial lasted 16 seconds. Before continuing onto the MOVING trials each participant was shown how the platform would move. All subjects performed 15 MOVING trials; they were asked to use the available handrails only if absolutely necessary. When the MOVING trials were complete, a clear and unequivocal verbal warning was given that the sled would no longer move for the AFTER trials. A visual affirmation was given by wedging the sled in place with wheel-stops; the motor was ostensibly turned off to reassure subjects that the sled would no longer move. All subjects then completed another 5 stationary AFTER trials.
**Experiment 2**

This experiment was conducted to assess the effect of vision on the LAE in young normals; 16 healthy subjects, ranging from 19-30yrs (mean=22.6±0.4yrs), were tested. Eight subjects were allocated to each group, eyes open (EO) and eyes closed (EC), according to whether the AFTER trials were initially conducted with EO or EC respectively (Fig. 1 bottom left).

The experiment followed the same general sequence, BEFORE, MOVING and AFTER. The EO group followed the same protocol as experiment 1. The EC group completed a slightly different protocol, where they completed 5 stationary BEFORE trials with eyes open and then 5 BEFORE trials with eyes closed. Subjects closed their eyes and were blindfolded at the beginning of each trial, but used vision to navigate back to the start position. Thus, subjects did have the opportunity to view the sled prior to closing their eyes. MOVING trials were always conducted with eyes open for safety reasons. The EC group then completed two sets of 5 AFTER trials first with eyes closed and then with eyes open (Fig. 1 bottom left).

**Experiment 3**

Here we studied the aftereffect in LDS in the absence of visual input. Both patients and age matched controls followed the same sequence as the EC group (see experiment 2, above, and Fig. 1 bottom left). Due to the difficulty of walking without vision the LDS were selected on the basis of a higher approach gait velocity in experiment 1. The fastest 7 LDS and all 13 NC were asked to return, however only 5 LDS, age range 42-69yrs (mean=58.6±5.1), and 5 NC, age range 43-65 (mean=56.6±3.7) agreed to be re-tested. All subjects were tested at least 12 months
after experiment 1. Local ethical approval and informed consent was obtained in all cases.

**Data analysis**

Pre and post foot-sled contact epochs were derived from the sled-mounted accelerometer and corroborated with foot pressure data. Trunk position was provided by the Fastrak (Fig. 1 right). For stationary trials (BEFORE and AFTER), forward trunk sway was measured as the maximum forward deviation (‘overshoot’) of the trunk, relative to the mean final resting stance position in the last 3s of the trial [Fig. 1 right (a)]. Due to the nature of the MOVING data, trunk sway was measured as the maximum forward displacement of the trunk (peak backwards to peak forwards) [Fig. 1 right (b)]. The velocity at which subjects approached the platform (approach gait velocity) was calculated using the position sensor on the trunk, defined as the mean velocity in a 0.5s time window prior to foot-sled contact [Fig. 1 right (c)].

EMG signals of the left leg (sled contact leg) were rectified and integrated over a 500ms time window after foot-sled contact [Fig. 1 right (d)]; MOVING and AFTER trial values were normalised with respect to a mean baseline integrated EMG measured in the BEFORE trials 3-5.

EMG latencies during AFTER 1 trial were measured from non-filtered grand averages and from AFTER – BEFORE subtracted grand averages. Latencies of left TA (LTA), right MG (RMG) were measured from the second peak of forward trunk velocity, obtained from the differentiated trunk displacement signal (see Fig. 3). Left MG (LMG) was measured from left heel contact (Fig 3; Fig. 4). Latencies were calculated
automatically taking a mean baseline +/-3 SD over a 100ms baseline period at the beginning of each trial, and visually corroborated in all cases. For graphical display purposes, EMG was lowpass filtered at 35Hz. Latencies during the MOVING trials (due to linear motor and movement artefact) could not be measured reliably.

Statistical approach
For all experiments a two-way repeated ANOVA (mixed design) analysed each variable (Bunday et al 2006). Factors group (2 levels: LDS vs. NC, or EO vs. EC) and trial (2 levels: mean BEFORE vs. AFTER 1 - an aftereffect is defined as a significant trial effect) were investigated. Bonferroni multiple comparisons were performed for post-hoc trial effects. Any specific comparisons between groups were made through independent t-tests

A criterion was devised to assess how many LDS and NC subjects had an aftereffect. The mean+2SD for BEFORE trials 1-5 was calculated for each subject for variables forward trunk sway (trunk overshoot), approach velocity and EMG of the LMG (the leg leading on to the sled). If AFTER trial 1 was above the mean+2SD this was considered to be an aftereffect. If at least two out of the three variables yielded an aftereffect then this constituted an overall aftereffect. A chi-squared test was used to determine differences in the frequency of occurrence of the aftereffect between the groups.

RESULTS
**Experiment 1: Age matched NC vs. LDS (Eyes Open throughout)**

Qualitative observations: All subjects performed the BEFORE trials without difficulty. During MOVING trials NC were unsteady, using the handrails for 1-2 trials. The LDS were visibly more unsteady than the controls during MOVING trials and required hand-rail use, and occasional manual support by two assistants, for the first 4 trials on average. Gradually all subjects improved their stability. During the AFTER trials, most subjects subjectively reported an unusual or unexpected sensation of unsteadiness and could be seen to be unsteady on mounting the stationary sled for the first AFTER trial. Quantitative results now follow.

**BEFORE and MOVING trials**

*Approach gait velocity:* Figure 2 shows approach walking velocity data for both NC and LDS. All subjects increase their approach gait velocity during the MOVING trials. Note that patients and controls reach 85% and 89% (respectively) of the final approach velocity on the first adaptation trial (MOVING trial 1). This increase must be an estimate based on seeing the platform move once (see Methods), similar for both groups \[t= -0.955, p=0.351\]. Both controls and patients achieved steady-state velocity by the third trial (i.e. values plateau after MOVING trial 3; Fig. 2 ‘approach velocity’). LDS approached the MOVING sled slightly slower than NC although this difference did not quite reach statistical significance \[F(1,20) = 3.501, p=0.076\].

*Trunk sway:* Forward trunk sway was largest on the first MOVING trial for both groups (Fig. 2 top). Subsequently, sway gradually diminished as subjects adapted to the task; for NC trunk sway values plateau at trial 3 but in LDS this did not occur until
trial 6 (Fig 2). As expected, LDS were significantly more unstable throughout the MOVING trials [F(1,20) = 15.019, p=0.001] and displayed significantly more EMG activity in LMG (Fig. 2 bottom) during the MOVING trials than NC [F (1,20) = 6.331, p=0.021].

AFTER trials

Figure 3 shows grand averaged trunk motion signals and leg EMG recordings for the age-matched controls (dotted trace) and LDS (solid trace) during AFTER trial 1 (i.e. the aftereffect). Note the trunk overshoot in AFTER trial 1 i.e. an aftereffect. EMG bursts are also larger in AFTER trial 1. Some EMG amplitude/latency differences can be seen between the groups but this will be discussed below under ‘Grand averaged EMG’. Figure 2 summarises group data for normal controls and LDS. As in previous work (Reynolds and Bronstein 2003) an aftereffect was expressed as an increase in approach velocity, forward trunk sway and leg EMG when AFTER trial 1 values were compared with BEFORE values. Statistics are given below.

Trunk sway: Figure 2 (top; AFTER) shows the mean forward trunk sway (or overshoot) for the LDS and the NC during the AFTER trials. A significant aftereffect can be seen in both groups [F(1,20) = 35.851, p=0.000]. Although the LDS appear to have increased forward trunk sway compared to the NC, this was not significant [F(1,20) = 1.363, p=0.257].

Approach gait velocity: Figure 2 (2nd diagram from top; AFTER) shows that approach velocity in both subject groups was faster during the AFTER trials than BEFORE, in particular AFTER trial 1. This confirms the presence of significant
aftereffects \([F(1,20) = 109.787, p=0.000]\). LDS have slightly slower approach velocity compared to NC, thus a group effect was found \([F(1,20) = 5.894, p=0.025]\). However, the lack of a significant interaction between trial and group \((F(1,20) = 1.117, p=0.303)\) indicates that the significant group effect depends on a) LDS approaching the sled slightly slower than NC throughout, as expected, and b) both groups showing a similar approach velocity aftereffect.

**Integrated EMG - LTA and LMG:** Figure 2 (bottom two diagrams; AFTER) shows the mean normalised EMG activity of LTA and LMG during the AFTER trials, in the regions of interest (see Methods). The increased levels of EMG activity in both muscles during the AFTER trials, with respect to the BEFORE levels, indicates the presence of an aftereffect \([F(1,20) = 9.065, p=0.007; F(1,20) = 44.787, p=0.000,\) respectively]. Note a) no obvious differences between the subject groups \([LTA: F(1,20) = 1.895, p=0.184; LMG: F(1,20) = 1.410, p=0.249]\) and b) that high EMG values in AFTER trials 1-2 rapidly attenuate on successive AFTER trials.

A similar proportion of subjects in both groups showed an aftereffect. Based on the criteria described in Methods, 88.9% of the LDS and 84.6% of the NC displayed an aftereffect \([\text{chi-squared} (1, n =22) = 0.082, p=0.774]\).

**Grand averaged EMG**

The experiments required subjects to take a first step with the right foot onto the fixed platform, then a second step taking the left foot onto the sled, followed by the right foot onto the sled for gait termination. Figure 3 shows the EMG events during such gait initiation; LTA activation precedes TA activity lifting the foot in the swing leg
(RTA) (approximate time -2s, -1.5s in Fig 3). This is followed shortly by LMG activation (starting at approximately -1s), aiding forward motion of the centre of mass. Later activity in RTA (approximate time -0.8s) mediates right ankle dorsiflexion during single-limb stance for heel strike and controlled loading of the foot onto the fixed platform. The RMG activity and LTA dorsiflexion activity at ca. – 0.5s is another single-limb stance phase when subjects are preparing for left foot landing onto the platform. Within this general pattern of locomotor events we would like to highlight below three points of relevance.

LDS are known to display reduced EMG amplitudes, related to reductions in gait velocity (Sasaki et al 2001; Tucker et al 1998). In line with this, prior to foot contact the patients exhibit decreased activity in both right and left MG and TA, this is particularly noticeable at gait initiation in LTA, RTA and LMG, and later during single-limb stance activity in RTA (-1.75s to -0.5s). This reduced muscle activity can be associated to the slower approach gait velocity seen in the patients. By subtracting the mean BEFORE from the AFTER 1 data, the EMG activity specifically due to the aftereffect can be displayed for each group (Fig. 4). Pre-foot contact differences between the groups seen in Figure 3 now disappear due to the normalisation process. Post-foot contact, however, some EMG latency differences become apparent (Fig. 3; Fig. 4). Latencies were measured from peak trunk velocity (second hump in the differentiated trunk position signal) to the onset of EMG (n.b. time to peak trunk velocity from left heel strike did not differ between the groups by more than 20ms (LDS: 104±14ms; NC: 88±14ms). Due to the similarity of the grand averaged latencies (Fig. 3) and the grand averaged subtracted latencies (Fig. 4), only the
subtracted latencies are reported. The left TA burst seen after left foot contact (ca 0.5s) occurs later in the LDS (396ms) compared to the controls (242ms) (Fig. 4). Right MG is also delayed in the LDS (292ms) with respect to the NC (186ms) (Fig. 4; n.b. visual inspection of individual subjects’ recordings showed essentially similar latencies in all muscles). As LDS walked on average 15% slower than NC, we corrected latencies LDS by 15% but despite this they are still prolonged (TA: 337ms; MG: 248ms).

Despite these group differences, Figure 4 bears out one critical similarity, namely that at foot-sled contact (time 0s) LMG becomes suddenly active. This MG burst is an important active component of gait termination (Sparrow and Tirosh 2005), however, note that this activity, which was present in BEFORE trials, becomes significantly increased during the 1st AFTER trial (Reynolds and Bronstein, 2003; also Fig. 2). This burst is similar in timing and amplitude in the LDS and NC, and is in fact anticipatory (i.e. it occurs before foot-sled contact) at -20ms and -13ms, respectively. Visual inspection of all individual subjects’ recordings showed this anticipatory burst, e.g. latencies ≤ 0ms. This burst is completely absent in MOVING – BEFORE subtracted data (Figure 5), thus indicating that this LMG activity is not related to the adaptation induced by the MOVING trials but to the ‘braking’ of the aftereffect (i.e. stopping the body from falling forwards). We ascertained that grand averages were representative as individual EMG amplitudes did not differ by more than 14%.

**Experiment 2: Young normal subjects; Eyes Closed (EC) vs. Eyes Open (EO)**

**BEFORE Trials**
This experiment was conducted in young normal subjects to see if visual input played a part in aftereffect onset (triggering) or termination (braking). The EC group completed two sets of AFTER trials first with eyes closed then with eyes open and, accordingly, performed two sets of related baseline BEFORE trials. The EO group were the control group so all data were obtained eyes open. The groups in Figure 6 are labelled according to what subjects undertook first in the AFTER trials, eyes open or eyes closed. Data were similar to the normal controls in Experiment 1 but, naturally, in eyes closed BEFORE trials, subjects walked slightly slower (drop in approach velocity between trials 5 and 1’ (n.b.: the change from trial 5 to trial 1’, indicated by the vertical black dotted line, represents the transition from eyes open to closed and vice versa; Fig. 6 bottom; left).

**MOVING trials**

Overall, data are similar to those of the normal controls in experiment 1 as trunk sway plateaus by the 3rd trial and the 2nd trial for approach velocity (Fig. 6 MOVING). Recall that all subjects conducted the MOVING trials with eyes open, thus there are no significant differences during the MOVING trials [F(1,14) = 0.092, p=0.767; F(1,14) = 2.564, p=0.132, respectively].

**1st set of AFTER trials**

*Trunk sway and Approach gait velocity:* Figure 6 shows the mean forward trunk sway and approach velocity for groups EC and EO during the AFTER trials. Both groups show an aftereffect for trunk sway [F(1,14) = 6.999, p=0.019]) but no significant group difference [F(1,14) = 0.000, p=0.990]. Approach velocity also shows the presence of an aftereffect [F(1,14) = 47.051, p=0.000], although again without a
group difference \([F(1,14) = 0.272, \ p=0.610]\). The findings indicate that vision does not influence the release of the aftereffect.

2\textsuperscript{nd} set of AFTER trials (EC group)

Interestingly, when the EC group re-opened their eyes (AFTER trial 1'; Fig 6), a small aftereffect re-appears both in approach velocity \([t = -3.565, \ p=0.008]\) and trunk sway \([t = -2.550, \ p=0.038]\). This indicates that both components of the aftereffect re-emerge on eye re-opening.

Experiment 3: Normal Controls vs. LDS (Eyes Closed)

BEFORE & MOVING trials

NC and LDS data during BEFORE and MOVING trials were very similar to experiments 1 and 2 and so they are not shown. However, this time during the MOVING trials (always with eyes open) there was little group difference in approach velocity \([F(1,7) = 0.572, \ p=0.474]\) (recall that LDS were selected by superior approach gait velocity). As in Experiment 1, LDS swayed more than NC during the MOVING trials \((F(1,7) = 7.288, \ p=0.031)\).

1\textsuperscript{st} set of AFTER trials

Trunk Sway: Figure 7 (Left) shows the mean forward trunk sway for the LDS and NC retested with eyes closed. As in experiment 1 a significant aftereffect was found \([F(1,8) = 9.692, \ p=0.014]\). The aftereffect was similar in magnitude to the previous eyes open aftereffect (LDS Experiment 1 = 11.99±1.94 cm, Experiment 3 = 11.13±4.80 cm; NC Experiment 1 = 8.63±2.00 cm, Experiment 3 = 10.82±1.68 cm).
There were no differences between LDS and NC [F(1,8) = 0.357, p=0.567] although the LDS, unlike the controls, remain somewhat unstable in the subsequent AFTER trials (recall they are walking with eyes closed).

*Approach gait velocity:* An aftereffect was again seen in approach velocity [F(1,8) = 15.957, p=0.004] Like trunk sway, the eye closed condition saw no group effect [F(1,8) = 0.000, p=0.985] (Fig. 7 eyes closed).

**2\textsuperscript{nd} set of AFTER trials**

*Trunk Sway:* In agreement with what was observed during Experiment 2 the aftereffect re-emerged on eyes re-opening for both subject groups [F(1,8) = 10.425, p=0.012]. The main finding is that the re-emergence of the aftereffect on eye re-opening is significantly larger in LDS than in NC [F(1,8) = 11.982, p=0.009] as shown in Figure 7 Right, Top and Bottom.

*Approach velocity:* A re-emergence of the aftereffect on re-opening the eyes was also noticed for approach velocity (Fig. 7 eyes re-open) [F(1,8) = 10.498, p=0.012]. There was no significant difference between subject groups [F(1,8) = 0.832, p=0.388].

**Appendix:**

**C7 Angular Velocity:**

In 12 normal subjects angular velocity at C7 (as the nearest segment to the head) was measured (gyroscope; Silicone sensing systems, Japan) in order to ascertain that motion parameters during these experiments were within the optimal functional range of the vestibular system. Peak C7 angular velocity during MOVING trial 1 and
AFTER trial 1 was 61±15°/s and 43±8°/s (respectively) with frequency components of 1-1.7Hz. Therefore, peak C7 angular accelerations at these frequencies during MOVING trial 1 were 381±96-648±164°/s² and AFTER trial 1 were 274±49-465±83°/s² (respectively). Such angular accelerations are well above threshold values of the semicircular canals [0.25-1.75°/s² adapted from Guedry (1974)].
DISCUSSION

Walking onto the stationary sled, which was previously experienced as moving, induced a locomotor aftereffect (LAE). This included a faster approach gait velocity, a forward trunk overshoot and larger levels of leg EMG with respect to the baseline (BEFORE) trials (Reynolds and Bronstein 2003; 2004). In these experiments we investigated normal and LD subjects (eyes open and closed) to gain insight into the processes terminating (‘braking’) this aftereffect.

All subjects faced a postural challenge during the MOVING and the AFTER trials and the latter condition was used to study the role of the vestibular system in a predictable, self-generated postural perturbation. The challenge presented by the MOVING trials is an externally imposed postural perturbation. In contrast, the challenge posed during the AFTER trials is internally generated and so, the stumble and trunk overshoot observed in the aftereffect are only the result of the inappropriate release of learnt motor activity. Examples of ‘self-inflicted’ postural stimuli are scarce in the literature and, in the few available, they are the result of unexpected ‘catch’ trials (e.g. Mille et al 2003; 2005; Traub et al 1980), unlike the paradigm used in the current experiments where subjects had full warning.

The impact of vestibular loss during MOVING (adaptation) trials.

During the MOVING trials, forward trunk sway was almost twice as large in the patients as controls. This result is fully expected in that many studies have shown increased unsteadiness during support surface motion in LDS (Martin 1965; Allum & Pfaltz 1985; Buchanan and Horak 2001; Creath et al 2002; Beule & Allum 2006;
Horak et al, 1994; Allum and Honegger, 1998; Dichgans and Diener, 1989; Bacsi and Colebatch 2005). In addition, our results show that normal subjects plateau (i.e. learn the task as best as they can) by MOVING trial 3, whereas patients plateau by trial 6. In fact, LDS appear to continue to adapt throughout the MOVING trials, as shown by the progressive decline in the difference between trunk sway values in patients and normal controls (see grey area, Fig 2, top). This finding suggests that vestibular loss also interferes with postural adaptation to repetitive stimuli (i.e. the moving sled). However, vestibular loss slows down this adaptation process but does not prevent it altogether since, by the end of the MOVING trials LDS sway was approaching normal values. This is in agreement with some previous research (Keshner et al 1987) and in contrast to other (Black et al 1983; Nashner et al 1982; Buchanan and Horak 2001). However, stimuli used and measurements of adaptation from such research are not always comparable to the current study (e.g. Buchanan and Horak, 2001).

In contrast to forward trunk sway, approach gait velocity during the MOVING trials was only slightly and non-significantly decreased in LDS. Further, both groups’ approach gait velocity plateau similarly on MOVING trial 3. What is the reason for this apparent discrepancy, whereby LDS show normal approach velocity but abnormally increased trunk sway? The crucial difference is that approach velocity is measured for the 500ms prior to foot-sled contact whereas trunk sway is measured after subjects are on the sled, i.e. approach velocity as measured in this experiment is an anticipatory parameter. In this regard, note that the increase in approach velocity deployed by the subjects to facilitate landing onto the moving sled comprises of two components. Firstly, an increase in approach velocity already present in MOVING
trial 1 (see Fig. 2), which is entirely anticipatory and based solely on a visual estimate, namely watching the sled move once (c.f. Methods). From then on, the velocity with which subjects approach the sled is based on preceding visual estimates plus the multisensory feedback experience of stepping onto the moving sled. Perhaps not surprisingly then, this dual predictive behaviour is intact in LDS. Preservation of such anticipatory processes in LDS must be a crucial mechanism explaining their relatively well adapted performance in regular everyday life activities.

The impact of vestibular loss during the AFTER trials: ‘Braking’ the aftereffect.

The aftereffect is an expression of the adaptive behaviour seen during the MOVING trials. Since during the AFTER trials the sled is stationary, the increased approach velocity and forward trunk overshoot are inappropriate and the aftereffect itself is a threat to postural balance. Therefore, we hypothesised that, in the absence of vestibular function, the forward trunk sway overshoot would be larger than normal. However, this was not the case and forward trunk sway data did not even approach significance, either in experiment 1 (eyes open) or 3 (eyes closed). On the basis of this, the conclusion is that the vestibular system does not participate in the braking of the forward overshoot produced by our aftereffect. In contrast, however, the early phase of the podokinetic aftereffect (elicited after prolonged walking on a rotating platform) is significantly increased in LDS (Earhart et al 2004). As their task involves fairly constant velocity rotation, the difference between normal and LDS is fully expected as the patients will not receive opposing inputs from the semicircular canals during the early portion of the self-generated rotation (Earhart et al 2004). Our LAE, in contrast, does not expose subjects to prolonged rotations and, if phasic vestibular responses participated in the braking process, the trunk overshoot should
have been larger than normal. The lack of difference between LDS and NC could be explained if the head velocity induced by the LAE was outside the operational range of the semicircular canals. However, angular motion at C7 was within the optimal velocity/frequency/acceleration range of the vestibular system (Guedry 1974). It is therefore likely that the braking of the forward trunk overshoot observed in this aftereffect is not vestibular-based. In agreement, the only delayed EMG latencies found in our LDS occurred ca. 200ms after peak trunk velocity which would be too late for ‘braking’ the forward sway in our experiments.

The current experiments do provide insight into the mechanisms responsible for ‘braking’ the trunk overshoot aftereffect. The subtracted data identifying the specific EMG patterns associated with the 1st AFTER trial (Fig. 4) show that during the 1st AFTER trial the left MG becomes highly active just before foot-sled contact (Figs. 3 & 4). Such left MG burst is a strong candidate for stopping the body lurching forwards during the 1st AFTER trial. Two features are important in this left MG burst in AFTER trial 1: 1) that it just precedes foot-sled contact indicating that it is an anticipatory (internally generated) process. 2) That it is of a similar magnitude, shape and latency in NC and LDS. These findings, that the ‘braking’ process is essentially anticipatory and that it remains unchanged in LDS, explain why there are no significant differences in the forward sway aftereffect between the normal and LD subjects.

Such ‘braking’ MG bursts occur when subjects voluntarily stop locomotion, as in this study (Sparrow and Tirosh, 2005). The finding that this MG burst was larger in AFTER trial 1 than in BEFORE trials indicates that additional anticipatory EMG
activity (for braking the faster approach gait velocity and the forward trunk sway aftereffect) was also incorporated. The origin of the anticipatory braking, as represented by enhanced left MG burst before foot-sled contact, cannot be ascertained from the current experiments but anticipatory mechanisms, generating EMG activity in advance of sensory triggers, are known to play an important part in postural and motor control (Belenkiy et al 1967; Bouisset and Zattara 1981; Horak et al 1984; Brown and Frank 1987; Friedli et al 1988). In any case, the presence of this enhanced burst indicates, firstly, that the CNS is anticipating that a stumble (i.e. the aftereffect) is likely to occur on gait termination and, secondly, that relying on slow sensory feedback for avoiding a fall is likely to fail. Although proprioceptive mechanisms, which are intact or even enhanced in LDS (Lund and Broberg, 1983; Nashner and Wolfson, 1974; Day et al, 1997; Fitzpatrick et al 1994; Inglis et al, 1995), could also be good candidates for braking the trunk overshoot, data in patients with peripheral neuropathy shows that the early left MG braking activity seen in the LDS is also evident in neuropathy patients; in fact it occurs even earlier in these patients showing that this braking activity is even more anticipatory when proprioceptive inputs are defective (KL Bunday and AM Bronstein, unpublished observations).

**The role of visual input in the locomotor aftereffect.**

Previous experiments established that the aftereffect was observed only if subjects walked onto the same stationary sled but not if they walked on a different surface (Reynolds and Bronstein 2004). Thus, it could be that sighting of the sled is a necessary precondition for aftereffect expression. However, experiments 2 and 3 showed that the aftereffect did not change with eye closure.
Whilst it can be concluded that visual feedback during execution of the first AFTER trial is not necessary for LAE release, the reciprocal is not true. Visual input confirming that one will not walk onto the sled inhibits aftereffect emergence (e.g. walking on the fixed launch platform does not release an aftereffect; Reynolds and Bronstein, 2004). These apparently conflicting findings can be resolved if we accept that subjects know whether they are walking onto the sled or not on the basis of cognitive and/or contextual cues. Thus, an internal judgment that the learned motor behaviour might be required (i.e. the sled may move) releases the aftereffect, rather than just viewing the sled. In summary, the data shows that release of the aftereffect is not specifically dependent on visual feedback during the movement but on more general spatial or ‘place’ contextual cues. Vision still seems to play a part in building up this general spatial context as discussed below.

**Visual context and the re-emergence of the aftereffect.**

Vision is likely to be important in building up a spatial context for this learnt behaviour since the adaptation (MOVING) trials were always conducted with eyes open. Having completed one set of AFTER trials with eyes closed, subjects in experiments 2 and 3 completed a second set of AFTER trials re-opening the eyes. Noteworthy, eye re-opening re-released an aftereffect, both for approach gait velocity and trunk forward sway in the three subject groups (Figs. 6 and 7). This aftereffect was smaller than the previous aftereffect, yet consistent enough amongst subjects to be significantly larger than baseline BEFORE values. Re-instating the visual context in which the adaptation took place caused the adapted behaviour to re-emerge. This is in agreement with increasing evidence showing the importance of context in
sensorimotor learning (Varriane et al 2002; Salinas 2004; Krouchev and Kalaska 2003).

Of note, the re-emerged aftereffect was significantly larger in LDS than in NC (Fig. 7). This difference can be taken as evidence that LDS placed increased weight to visual information during the adaptation phase. In other words, LDS relied on vision more than normal controls when learning to negotiate the moving sled, as expected and previously shown in other support surface motion experiments (Buchanan and Horak, 1999; Patla et al, 2002; Hollands et al, 1996). Then, when the original visual context was re-visited, LDS showed an enhanced aftereffect. LDS are known to have enhanced sensitivity to visual input in tasks where vision is used for feedback control (e.g. increased postural sway with eye closure or in response to visual motion; Guerraz et al 2001; Peterka and Benolka 1995). The current experiments indicate that LDS also have increased up-regulation of visual mechanisms when vision is used for feed-forward control and context build up.

Several areas of the brain have been implicated in sensory context learning. These include the cerebellum (Lewis and Tamargo 2001), the basal ganglia (Turner and Anderson 2005), and the hippocampus (Etienne et al 1988; McNaughton et al 1991; Blair and Sharp, 1995 Poucet and Save 2005), the latter being important for visuo-vestibular spatial navigation (Semenov and Bures 1989; Brandt et 2005; Markus et al 1994; Ossenkopp and Hargreaves, 1993). One could speculate that in our experiments a distinct sensory-context map of the environment in which subjects learnt to walk onto the moving sled was made. On re-opening the eyes this distinct sensory context reappears, activating circuits which normally would include visuo-
vestibular input. In the patients, the lack of vestibular input could make context-driven circuits supersensitive to visual input, e.g. by denervation supersensitivity (Bannerman et al 2001), and on eye re-opening these up-regulated circuits would trigger a larger aftereffect in LDS.

In conclusion, we have shown that the LAE is not increased in the absence of vestibular function even though, as expected, LDS are abnormally unsteady during the MOVING (adaptation) trials. This result suggests that the vestibular system may be more involved in counteracting externally imposed, rather than internally generated, postural perturbations. In agreement, we observed that the braking of the aftereffect is executed by anticipatory EMG activity. This indicates that the CNS has advanced information that the aftereffect will occur and sets off feedforward mechanisms to deal with this potential source of unsteadiness. We have also found that the LAE does not necessitate vision to be triggered. However, visual input participates in building up a spatial context during the adaptation trials. The strength of this visuo-contextual mechanism appears to be increased in the absence of vestibular function.
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Reference List


LEGENDS

Figure 1. Experimental setup, protocol and analysis.

Top left. Experimental setup showing how the subject walks from the fixed platform with the right leg and landing on the moving sled with the left leg. Note the diagram shown represents a stationary trial; during moving trials sled motion will be triggered by the subject passing through the light switch. A Fastrak sensor placed on C7 recorded fore-aft trunk position. EMG electrodes placed on the lower legs recorded muscle activity of tibialis anterior (TA) and medial gastrocnemius (MG). Below the sled diagram, the stimulus velocity profile shows the speed (1.3 m/s) and duration of the sled (4.2 s). Bottom left. Experimental protocol for Experiments 1, 2 and 3 (5 & 15 represent number of trials). Eyes open (EO – grey diamond ended bars) and eyes closed (EC – black circled ended bars) trials are represented. Right. Measurements obtained: a) Forward trunk sway (BEFORE and AFTER trials) measured as the maximum forward deviation of the trunk, relative to the mean final resting stance position in the last 3 s of the trial. b) Forward trunk sway (MOVING trials) measured as maximum forward deviation of trunk peak backwards to peak forwards. c) Approach gait velocity, measured in a 0.5 s time window prior to foot contact. d) EMG measured as an integral value from an epoch of 500 ms.

Figure 2. Mean (± SE) group data for Experiment 1 during conditions BEFORE, MOVING and AFTER.

Numbers along the horizontal axis represent trial numbers. A change from stationary to moving, moving to stationary is represented by a vertical black line. Groups LDS (closed circles) and NC (open squares) are represented. The grey shaded area represents the NC subtracted from LDS. From Top to Bottom: trunk sway, approach
velocity, normalised left TA and normalised left MG. Note the aftereffect as increased trunk overshoot, approach gait velocity, left TA and MG particularly in AFTER trial 1.

Figure 3. Grand averaged LDS and NC data of movement and EMG responses in Experiment 1 during AFTER trial 1 (i.e. the aftereffect)

Walking activity, taking the subject onto the stationary sled is represented by trunk displacement and its differential (velocity), EMG activity and the corresponding cartoon man (derived from video footage). LDS (solid black line) and NC (dotted black line) are represented. Data were averaged for AFTER trial 1 with respect to foot-sled contact as indicated by the thick dotted vertical line at time zero (as derived from vibrations detected by a sled-mounted accelerometer). The thin vertical dotted line represents right heel contact. Note the trunk overshoot in both groups (i.e. aftereffect) as increased trunk displacement compared to the latter part of the trial (3.5s-4s). The velocity trace is the differentiated trunk displacement signal; the double peak represents the two steps to reach the sled. Arrows represent approximate EMG burst onset in NC (downward pointing arrows) and LDS (upward pointing).

Figure 4. Grand averaged subtracted (AFTER-BEFORE) data of movement and EMG responses in Experiment 1

Data were averaged for baseline mean BEFORE and AFTER 1 as in Fig. 3. Data represents AFTER 1 as subtracted by mean BEFORE (i.e. solely the aftereffect). LDS (solid black line) and NC (dotted black line) are represented. Arrows as in Fig. 3; for other details also see Fig. 3. Horizontal interrupted line represents the normalised ‘zero’ level.
Figure 5. Grand averaged subtracted (MOVING-BEFORE) data of movement and EMG responses in *Experiment 1*

Data was averaged for baseline mean BEFORE and MOVING (11-15) as in Fig. 3 & 4. Data represents MOVING (11-15) as subtracted by mean BEFORE. LDS (solid black line) and NC (dotted black line) are represented. For other details see Fig. 3 & 4.

Figure 6. Mean (± SE) group data for *Experiment 2* during conditions BEFORE, MOVING and AFTER.

Numbers along the horizontal axis represent trial numbers. Groups EO (black circle) and EC (grey circle) are represented, defined by whether the first set of AFTER trials (1-5) was conducted with eyes open or closed respectively. The EC group conducted a second set of after trials after re-opening the eyes (1’-5’). A change from stationary to moving, moving to stationary is represented by a vertical black line. **Top.** Mean trunk sway. **Bottom.** Mean approach velocity. Note the aftereffect as increased trunk sway and approach velocity particularly in AFTER trial 1. Also note the re-emergence of a (smaller) aftereffect when the EC group re-open their eyes (AFTER trial 1’).

Figure 7. Mean (± SE) group trunk sway and approach gait velocity data for *Experiment 3* during condition AFTER.

Numbers along the horizontal axis represent trial numbers. Groups LDS (closed circle) and NC (open square) are represented. Both groups conducted the first set of AFTER trials (1-5) with eyes closed (Left), and then re-open eyes in the second set of
AFTER trials (1’-5’) (Right). A change from eyes closed to eyes re-open is represented by a vertical solid black line. The mean BEFORE baseline values for the LDS (horizontal interrupted line) and NC (horizontal dotted line) are also represented.

**Top.** Mean trunk sway. **Bottom.** Mean approach velocity. **Insets.** AFTER trial 1 with eyes closed (Left) and AFTER trial 1’ (2nd set of AFTER trials on eye re-opening) (Right) for LDS (filled bars) and age-matched controls (non-filled bars).

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