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RESEARCH ARTICLE

# The effect of gait approach velocity on the broken escalator phenomenon

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**Abstract** Walking onto a stationary surface previously experienced as moving generates an after-effect commonly known as the "broken escalator" after-effect (AE). This AE represents an inappropriate expression of the locomotor adaptation necessary to step onto the moving platform (or escalator). It is characterised by two main biomechanical components, an increased gait approach velocity (GAV) and a forward trunk overshoot on gait termination. We investigated whether the trunk overshoot and other biomechanical measures are the direct inertial consequence of the increased GAV or whether these are the result of an independent adaptive mechanism. Forty-eight healthy young adults walked onto a movable sled. They performed 5 trials with the sled stationary at their preferred walking velocity (BEFORE trials), 5 with the sled moving (MOVING or adaptation trials), and 5 with the sled stationary again (AFTER trials). For the AFTER trials, subjects were divided into four groups. One group was instructed to walk slowly ("slower"), another with cueing at the BEFORE pace ("metronome"). The third group walked without cueing at the BEFORE pace ("normal"), and the fourth, fast ("faster"). We measured trunk pitch angle, trunk linear horizontal displacement, left shank pitch angular velocity and surface EMG from lower leg and trunk muscles. In the AFTER trials, an AE was observed in these biomechanical measures for all gait speeds, but these were not strongly dependent on GAV. An AE was present even when GAV was not different from that of BEFORE trials. Therefore, we conclude that, although contributary, the trunk overshoot is not the direct consequence of the increased GAV. Instead, it appears to be generated by anticipatory motor activity "just in case" the sled moves, herewith termed a "pre-emptive" postural adjustment.

**Keywords** Gait approach velocity · Locomotor after-effect · Pre-emptive postural adjustment

## Introduction

One is able to remain standing under hazardous circumstances using conscious (voluntary) and subconscious (reflex-like) neural processes. Regaining stability following a perturbation without excessive sway relies upon prior experience, and motor adaptation. Adaptative responses to balance perturbations have been examined in several studies (Horak and Nashner 1986; Prokop et al. 1995; Bloem et al. 1998; Reynolds and Bronstein 2003, 2004, 2007; Sparrow and Tirosh 2005; Choi and Bastian 2007; Hollman et al. 2010). Understanding these processes is important in determining how humans learn to maintain postural equilibrium when perturbed and may have implications for rehabilitation of patients with neurological gait and balance disturbances.

An important aspect of learning is applying what has been learned to the appropriate context. An inappropriate expression of adaptation can occur when walking onto a broken escalator or walkway which normally moves (Shadmehr and Brashers-Krug 1997; Reynolds and Bronstein 2007; Bronstein et al. 2009). Although the subject is aware that the escalator is stationary, the motor actions are prepared as if the escalator would move, resulting in a stumble (Reynolds and Bronstein 2003, 2004, 2007). When subjects

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are asked to walk onto the stationary escalator a second or third time, re-adaptation occurs and the stumble disappears (Reynolds and Bronstein 2003, 2004, 2007).

This stumble is a locomotor after-effect (AE) characterised by two main components, an increase in gait approach velocity (GAV) and a trunk forward overshoot (Reynolds and Bronstein 2003; Bunday et al. 2006). The AE occurs with just 1–2 exposures to a moving sled, cannot be suppressed consciously and is context dependent (Reynolds and Bronstein 2004; Bunday et al. 2006). Walking onto the broken, and hence stationary, walkway for the first time causes an internally generated motor action in preparation for a moving walkway (a "just in case" strategy (Bunday et al. 2006)). Therefore, the forward stumble is the result of the inappropriate expression of learned motor activity (Reynolds and Bronstein 2003, 2007; Bronstein et al. 2009).

In this manuscript, we ask whether the trunk overshoot component of the AE is the direct consequence of the increased GAV, that is, an inertial process just like the forward body sway that occurs when a bus stops, or whether the AE is an independent adaptive mechanism. For instance, the trunk overshoot could be an anticipatory postural adjustment, in a broad sense of the term, brought about by the preceding experience that, on foot contact with a moving platform, the body tends to fall backwards and therefore must be countered by forward trunk motion. However, it is equally plausible that the trunk overshoot is caused by the increased GAV, since higher gait speed leads to more forward trunk motion (Goutier et al. 2010).

Another way of considering the AE is as a first trial effect. Such effects are usually observed the first time a balance perturbation is presented (Nashner 1976). These contain an exaggerated response, similar to a startle response, plus the adequate response for the perturbation (Tang et al. 2012). The exaggerated response comprises a forward trunk flexion due to activation of trunk muscles (Oude Nijhuis et al. 2010; Tang et al. 2012), and as such could underlie the trunk overshoot of the AE. Successive presentation of the balance perturbation leads to an adaptation in the 1 $^{st}$ trial response (Nashner 1976; Horak and Nashner 1986; Tang et al. 2012) just as occurs with the AE (Reynolds and Bronstein 2003, 2004, 2007). However, in the case of the AE, the balance perturbation would be internally generated.

To assess the relationship between the trunk overshoot and other biomechanical measures and GAV, we investigated the characteristics of the AE at 4 different gait velocities in the same setting as previous studies (Bunday et al. 2006; Bunday and Bronstein 2008, 2009; Bronstein et al. 2009). Changes in walking speed were obtained by asking subjects to walk at different speeds onto a laboratory realisation of the "broken escalator" phenomenon. We hypothesised that if the trunk overshoot is the direct consequence of GAV, then increased GAV should result in increased trunk overshoot and related muscle activity, and a slower gait would produce less or possibly no trunk overshoot. Alternatively, the AE may be dependent on the subjectively assumed walkway velocity observed by subjects in MOVING trials rather than GAV. In this case, despite changes in GAV onto the broken walkway, a trunk overshoot would consistently occur.

## Experimental procedures

### General procedure

Subjects were asked to walk from a fixed platform on to a movable sled (Reynolds and Bronstein 2003; Fig. 1). Three blocks of 5 trials were presented. The first block of 5 trials, called BEFORE trials, consisted of normal walking at the subject's preferred speed onto the movable but stationary sled. In the second block, the sled moved with a velocity of 1.4 m/s, triggered on gait initiation by crossing an infrared light beam mounted just in front of the subject (MOVING trials). Following these trials, subjects were informed that the sled would no longer move and further reassured by ostensibly shutting down the motor and placing two wooden wedges under the sled wheels. In this block (AFTER trials), subjects were asked to walk at different speeds depending on the group to which they were assigned (see "Groups" below).

For all trials, subjects were asked to start walking after hearing three beeps. For all trials, the walking sequence consisted of three steps starting with the right leg. For the second step, the left foot should land on the movable sled. For the last step, the right foot should be placed next to the left foot, thereby stopping (gait termination). When stopped, subjects were asked to adopt a quiet stance until recording was completed. Each trial lasted 16 s. Subjects were instructed to avoid using the handrails at the sides of the sled when stepping on to it unless absolutely necessary.

### Groups

Forty-eight healthy young adults gave written informed consent to participate in the study, approved by the local ethics committee. Subjects were randomly assigned to 4 equally sized groups ($n = 12$, 50 % men, 50 % women). The mean height and weight of all subjects were 170 ± 9 and 63.7 ± 12.8 kg, respectively. There was no statistical significant difference in height and weight between subjects in the four groups ($\Psi^2 < 1.3$, $df$ 3, $p > 0.7$). Subjects in the first three groups were first shown a 5-point visual scale containing the words "Very Slow", "Slower than normal", "Normal", "Faster than normal" and "Very Fast" to aid them in their selection of walking speed. One group of subjects were asked to walk "slower than normal", another group

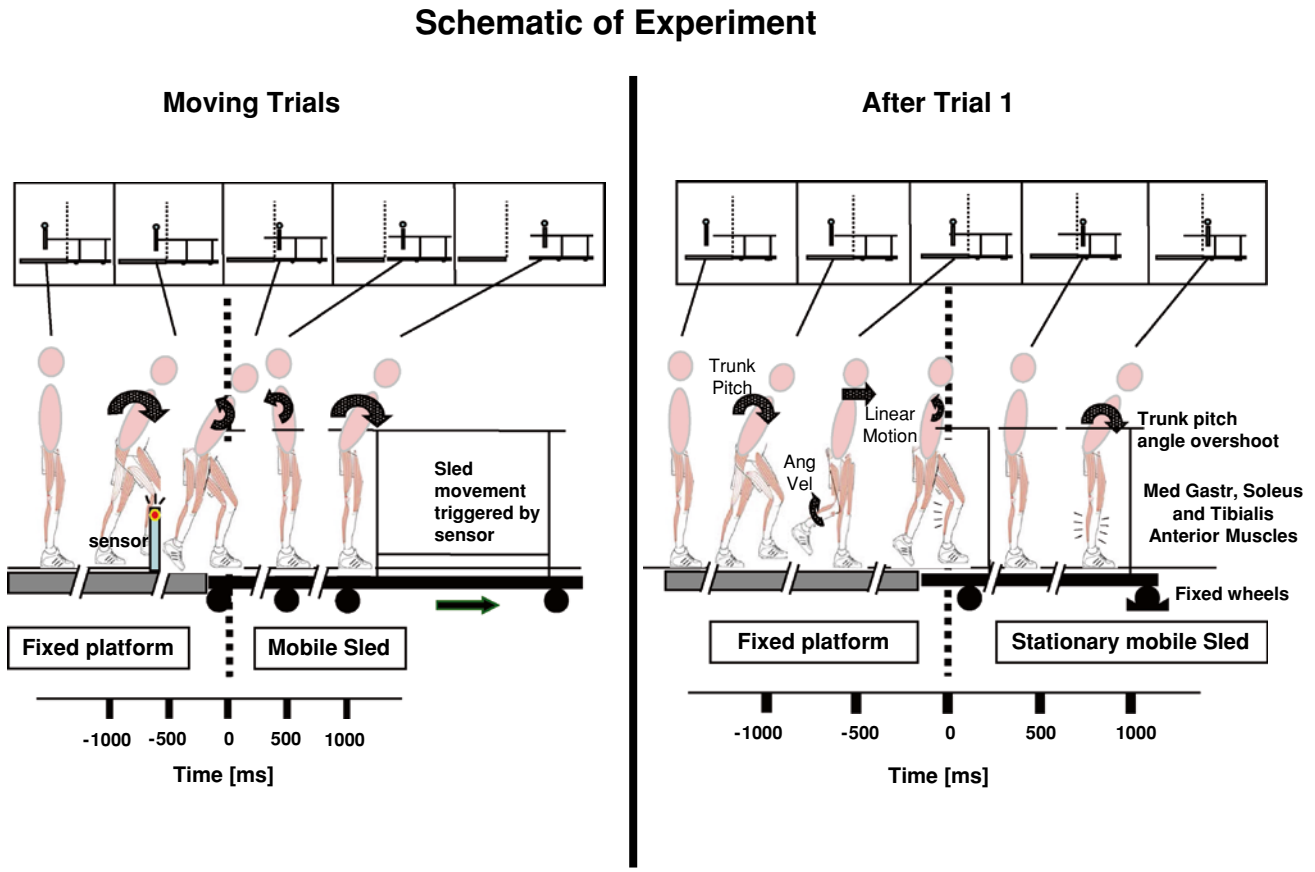


**Fig. 1** Schematic overview of MOVING (*left panel*) and AFTER trial 1 (*right panel*) of the experiment. The subject is asked to make 3 steps on to the mobile sled, starting with the right leg, then stepping onto the sled with the left foot and finally terminating by bringing the right foot to the left. The left heel strike is marked by the *doted vertical line* at 0 ms and is aligned with a peak in shank longitudinally directed acceleration. For the MOVING trials, the first step is on the fixed platform and triggers the platform movement. The second step is onto the moving sled as it is moving away from the subject. The cartoon illustrates the approximate movement of the subject based on the linear/angular position data as shown in Fig. 2. For the AFTER trials, the subject steps onto the previously moving platform which is now stationary, as the motor is switched off and wedges are placed under the moving platform wheels. The main after-effects (*AE*) noted in this study are displayed schematically using *arrows* (trunk) and *radiating lines* (muscles). Furthermore changes in trunk pitch angles for MOVING trials with respect to BEFORE trials are also displayed. Trunk pitch means trunk pitch angle; *Ang Vel*, shank pitch angular velocity; *Med Gastr*, medial gastrocnemius

at their preferred normal pace, and the third group "faster than normal", during the AFTER trials. This procedure was used to minimise the gait velocity deviations within a group (Goutier et al. 2010). The fourth group walked at a cued normal speed in the AFTER trials. Cueing was accomplished by asking the subjects to walk 5 times onto the stationary sled as if they were doing the BEFORE trials. During these trials, we adjusted a metronome to their walking cadence. Then, in the BEFORE and in the AFTER trials, we asked them to walk in rhythm with the metronome in an attempt to provide subjects cues to "over-ride" any increase in gait approach velocities during AFTER trials. Subjects were naïve to the detailed purpose of the experiment. The sled movement was shown twice to subjects before the start of the experiment.

## Equipment

The mobile sled (Reynolds and Bronstein 2003) was powered by two linear induction motors. After an acceleration period of 1.35 s, it moved with a peak velocity of 1.40 m/s, as measured with a tachometer. The sled was stationed partially under a fixed platform, from which it could move out smoothly. Sled motion was computer controlled and programmed to move 600 ms after triggering by the subject crossing a light beam. To measure the subject's linear motion forward and gait velocity (Fig. 2), a Fastrak electromagnetic tracking device (model 3SF0002, Polhemus, Vermont, USA) was placed over the C7 vertebrae of the subject. The transmitting device for this system was attached to the moveable sled. To provide a first approximate time

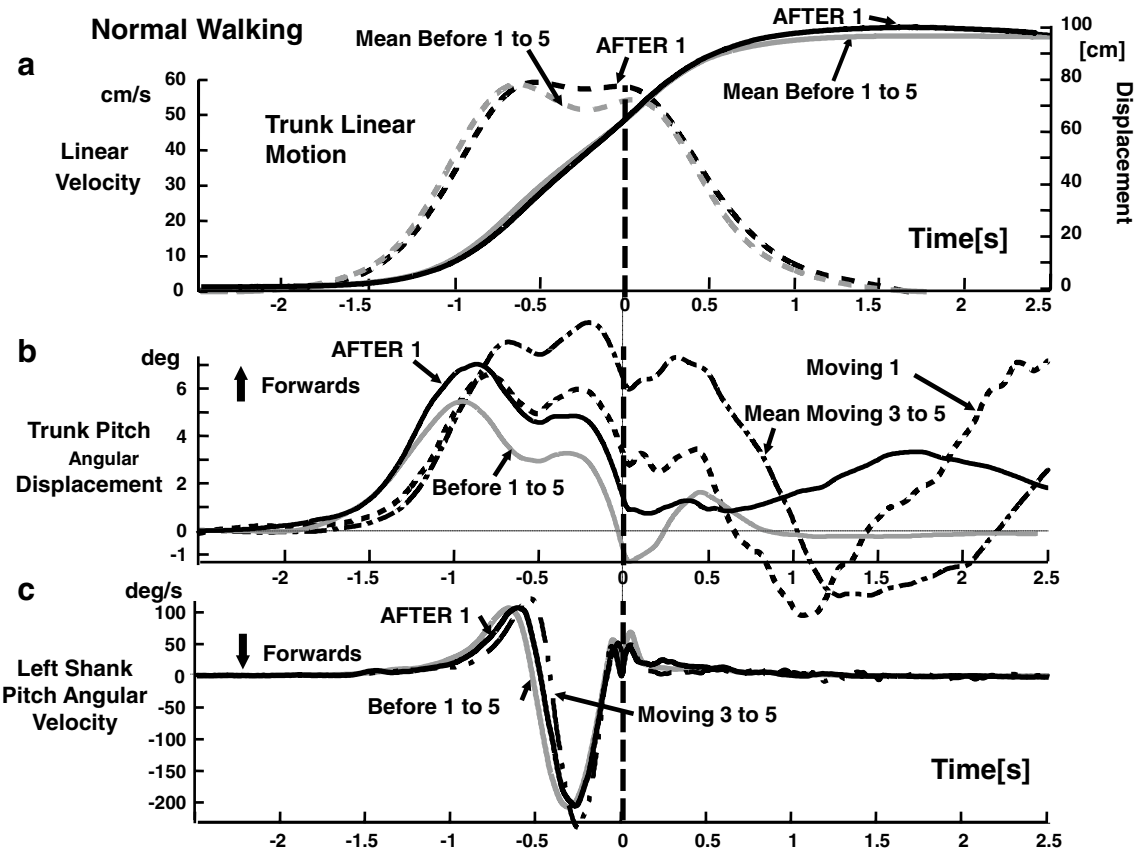


**Fig. 2** Average population traces of trunk linear displacement and velocity (**a**), upper trunk pitch angle (**b**), left shank pitch velocity (**c**) of the group asked to walk at normal speed during AFTER trials. *Traces* shown are for the mean of all BEFORE trials 1–5 (*grey lines*) all the AFTER trials (*black lines*), 1st MOVING and MOVING trails 3–5 (*dotted* and *dash-dot lines*, respectively). 0 ms is defined with respect to left heel strike on the sled, hence the apparent shift in left shank pitch velocity and trunk pitch angle traces in 1st AFTER and MOVING trials. This shift is due to the faster gait velocity for these trials with respect to BEFORE trials. Note that the apparent delayed rise of gait velocity (and shank pitch velocity) at −1.5 s in AFTER trial 1 s is an artefact of aligning traces to heel strike as the gait cycle is shorter in the AFTER trials

of foot contact, modified anti-static heel grounders served as footswitches and were strapped under the shoes at the first metatarsal-phalangeal joint and the heel. In addition, a linear accelerometer (Entran R6309 ±5 g, Hampton, USA) was attached to the sled and provided independent information on foot-sled contact. This data were recorded on a PC, sampled at 500 Hz.

Upper trunk pitch and roll angular velocity were measured with a SwayStar™ system (Balance Int. Innovations GmbH, Switzerland)—see Fig. 2—and these signals were integrated on-line to yield angular position (Allum and Carpenter 2005). This system was mounted on a converted shoulder harness and measured trunk sway at the level of T3-5. The data were collected by another PC wirelessly at a rate of 100 Hz via Bluetooth™. A triaxial motion sensor (XSens model MTi, XSens Technologies B.V. Enschede, The Netherlands) was strapped to the upper left shank to measure shank pitch angular velocity (see Fig. 2) and shank linear acceleration of the lower leg along its longitudinal axis. The latter signal was used as an indicator of the foot-sled contact event. XSens data were also sampled at 100 Hz and stored with SwayStar data on the same PC.

Using pitch angle (SwayStar) and linear motion (Fastrak) data in combination allowed us to explore whether linear trunk overshoot results from trunk flexion (about the hip or ankle) or whether it is simply due to subjects moving further along the walkway.

Surface EMG was recorded from the left soleus, left and right gastrocnemius, left and right tibialis anterior, left obliquus externus and left and right paraspinalis at L4-5, using pairs of electrodes spaced 2 cm apart. EMG signals were band-pass filtered between 20 Hz and 2 kHz, amplified 1,000 times and then sampled at 5 kHz. EMG samples were stored on both PCs simultaneously and later synchronised off-line using synchronisation signals issued between PCs. Offline analysis consisted of full-ware rectification and the calculation of areas under the resulting EMG curves over intervals with respect to left heel strike.

### Data analysis

Left foot–sled contact time (here termed heel strike) was established by linear shank acceleration along its longitudinal axis (Jasiewicz et al. 2006), as this proved to be the most reliable signal for this purpose and confirmed with the sled accelerometer and foot switch recordings. This time point was set as 0 ms for analysis as displayed in the figures. For each trial, forward trunk sway during BEFORE and AFTER trials was measured as the maximum forward deviation (or trunk overshoot) of the Fastrak sensor, relative to the resting baseline (average trunk position in the last 3 s of the trial). Gait approach velocity was calculated from the first derivative of the Fastrak position data. The GAV was calculated over a 0.5-s time window ending at the left foot–sled contact, as in previous studies (Bunday et al. 2006).

Due to differences in requested gait speeds across groups and during AFTER trials, gait cycles were of different durations. In order to analyse changes in the characteristics of the AE with GAV in the form of changes in the amplitude of peak trunk angle, we aligned recordings at the peak trunk forward flexion angle (see Figs. 2, 4). Similarly, we also aligned recordings of shank angular velocity at the peak backwards shank angular velocity (see Figs. 2, 5). EMG recordings and trunk pitch movements post-heel strike were aligned with foot-sled contact (0 s). EMG signals were analysed as areas over the period −0.1 to 0.2 and 0.2–0.5 s post-left foot–sled contact.

We characterised the AE in two ways. Firstly, measures of the 1st AFTER trials were compared with average measures from the BEFORE trials. That is, we considered the response in AFTER trial 1 as an AE. Secondly, we compared measures in AFTER trial 1 with the same measures in trials 3–5 of the AFTER trials. That is, we considered the 1st response as a 1st trial effect. All the groups walked at the same preferred natural speed in the BEFORE trials, but were instructed to walk at different speeds in the AFTER trials.

Statistics were performed using SPSS 19.0 software. Significance levels were set at $p \leq 0.05$ and adjusted to higher levels (Bonferroni correction) according to the number of multiple comparisons. Shapiro–Wilk tests revealed that 14 of 16 kinematic measures (4 measures for 4 groups) were not normally distributed; therefore, nonparametric tests were used. These consisted of Kruskal–Wallis tests, Mann–Whitney tests for comparing groups and Wilcoxon Signed Ranks tests for within-subject comparisons. Means and standard errors of means are presented in the figures.

## Results

Figure 2 illustrates traces for all BEFORE and MOVING trials, and AFTER trial 1 for subjects walking at their preferred self-paced speed in AFTER trials, hereafter referred to as "normal" speed. In addition to the previously described increase in gait approach velocity and forward trunk overshoot, other components of the AE were apparent in AFTER trial 1. Firstly, we observed an increased left shank pitch velocity at gait onset in AFTER trial 1 compared to BEFORE trials (Fig. 2), visible also in the inset of Fig. 5a. Secondly, we observed an increased trunk pitch angular displacement in AFTER trial 1. The increased trunk pitch before and after left heel strike was coincident with an increase in trunk linear motion (the trunk overshoot) post-heel strike. Note that in the time after heel strike (+0.5 to +1.5 s), trunk pitch motion was moving backwards in MOVING trials, forwards in AFTER trial 1 and stable in BEFORE trials. Foot sensor records confirmed that the increased linear trunk movements in AFTER trial 1 s were not the result of subjects moving their feet further forward or backwards, but, as indicated by trunk pitch measurements, the result of increased trunk sway.

### Gait approach velocity (GAV)

Figure 3a shows the average GAV trial by trial. The mean GAV was 0.47 (sd 0.1) m/s for all BEFORE trials. GAV in the BEFORE trials did not differ among the groups ($\Psi^2 = 3.1$, $df$ 3, $p = 0.4$). For the AFTER trials 3–5, mean GAV was 0.42 m/s (sd 0.12) for "slower" gait; 0.48 m/s (sd 0.08) for "metronome"; 0.53 m/s (sd 0.08) for "normal" gait; and 0.75 m/s (sd 0.07) for "faster" gait, that is, successively faster in accordance with the instructions given to the subjects. The GAV in normal and metronome AFTER trials 3–5 was not different from that for BEFORE trials ($p > 0.05$). In the slower group, the GAV for AFTER trials 3–5 was less than for BEFORE trials but not significantly smaller ($p = 0.084$). GAV for the faster AFTER trials 3–5 was, however, significantly greater ($p = 0.002$).

For the normal and metronome groups, increased GAV in AFTER trial 1 indicated the presence of an AE both with respect to BEFORE trials ($p = 0.034$ for normal; $p = 0.05$ for metronome) and AFTER trials 3–5 ($p = 0.041$ for normal; $p = 0.038$ for metronome). In the slower group, GAV in the AFTER trial 1 was greater than trials 3–5 ($p = 0.021$) but not significantly different to the BEFORE trials ($p = 0.433$). In the Faster group, the GAV for AFTER trial 1 was greater than in BEFORE trials ($p > 0.05$), as expected from instructions to the subjects in this group. GAV in AFTER trial 1 was greater than for AFTER trials 3–5 in the faster group, but this difference was not statistically different ($p = 0.06$).

### Trunk motion

Figure 3b–d provides details of trunk component of the AE for different GAVs. Following heel strike in AFTER trial 1,

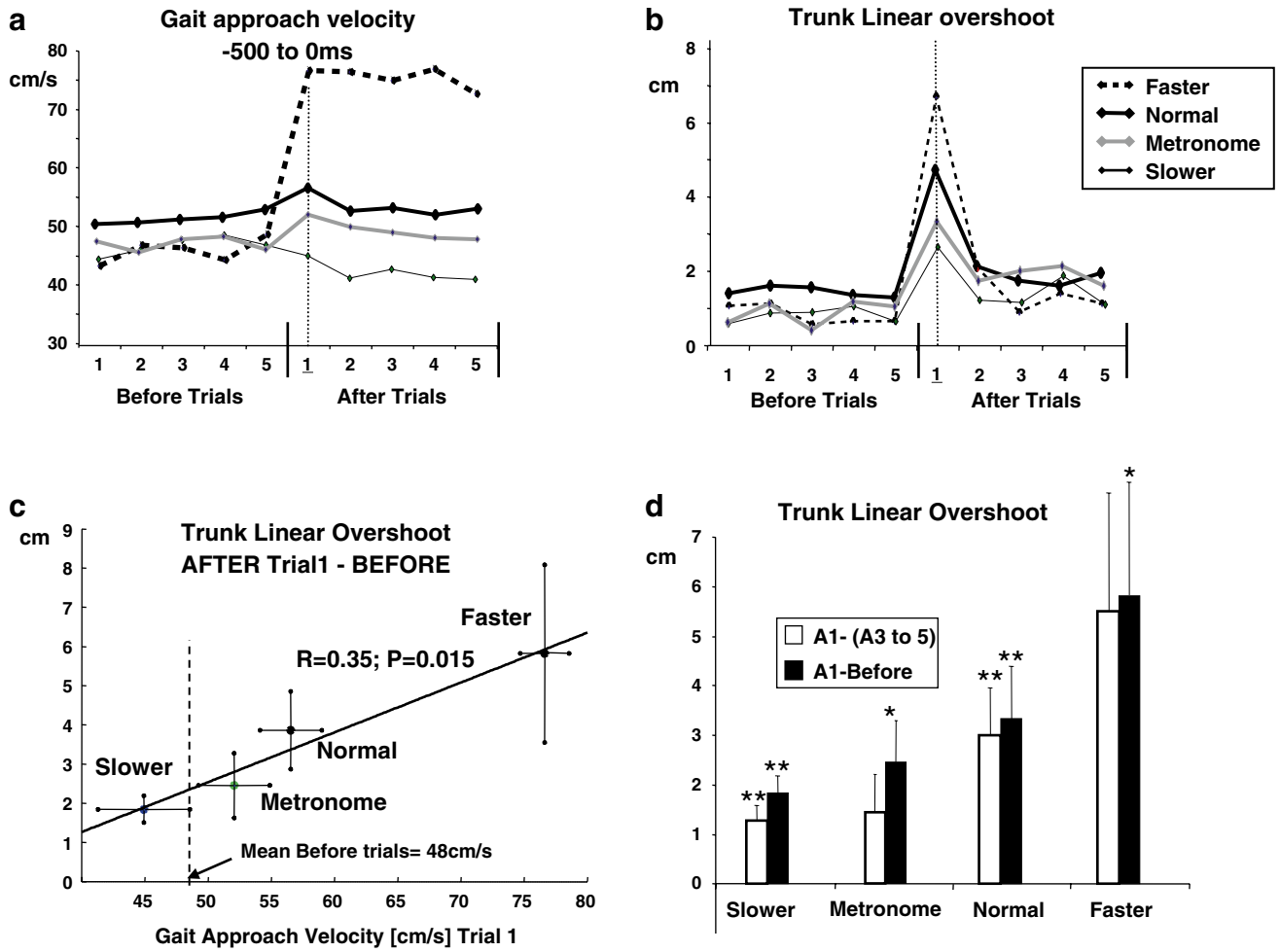


**Fig. 3** Mean values of gait approach velocity (*GAV*) and trunk linear overshoot for the 4 different requested gait velocities in AFTER trials. **a**, **b** The trial by trial means for these two measures. **d** The mean trunk linear overshoot with respect to BEFORE trials plotted against GAV. Means and standard errors of means for each group are shown. The *regression line* shows the regression across data from all subjects. **d** The mean trunk linear overshoot as the difference between AFTER trial 1 and the mean of all BEFORE trials or AFTER trials 3, 4 and 5. The column height represents the mean value and the *vertical bars* on columns the SEM. Significant differences (from zero) are marked as * representing $p < 0.05$, ** representing $p < 0.01$

a "trunk overshoot" was manifest, due to the cumulative effect of trunk flexion peaking prior to and at ca. 1.5 s after left heel strike (Fig. 2). This trunk overshoot component of the AE has been described using only linear motion recordings in previous experiments (e.g. Reynolds and Bronstein 2003). For MOVING trials, trunk pitch motion was backwards post-heel strike presumably as a consequence of the forwards motion of the sled (see Fig. 2). This suggests that the forwards trunk flexion and resulting linear trunk overshoot during AFTER 1 is a learned compensatory response to counteract for the tendency to fall backwards during the MOVING trials.

The trunk linear overshoot component, when computed as the difference between AFTER 1 and BEFORE trials, was significant for all gait velocities (slower $p = 0.008$; metronome $p = 0.012$; normal $p = 0.002$; faster $p = 0.023$)—see Fig. 3d. There was a weak ($R = 0.35$) but significant ($p = 0.015$) correlation between trunk overshoot and GAV in AFTER trial 1 (Fig. 3c). Linear trunk overshoot increased as a function of GAV (see Fig. 3c), but did not quite reach significance for faster walking ($p = 0.06$) when compared to AFTER trials 3–5—see Fig. 3d. Compared to AFTER trials 3–5, slower and normal walking provided a significant linear trunk overshoot ($p < 0.05$), showing that the trunk overshoot component of the AE can occur even when subjects walk at these speeds.

Trunk forward pitch changes prior to heel strike also contribute to trunk linear motion AE. For analysis purposes, peak

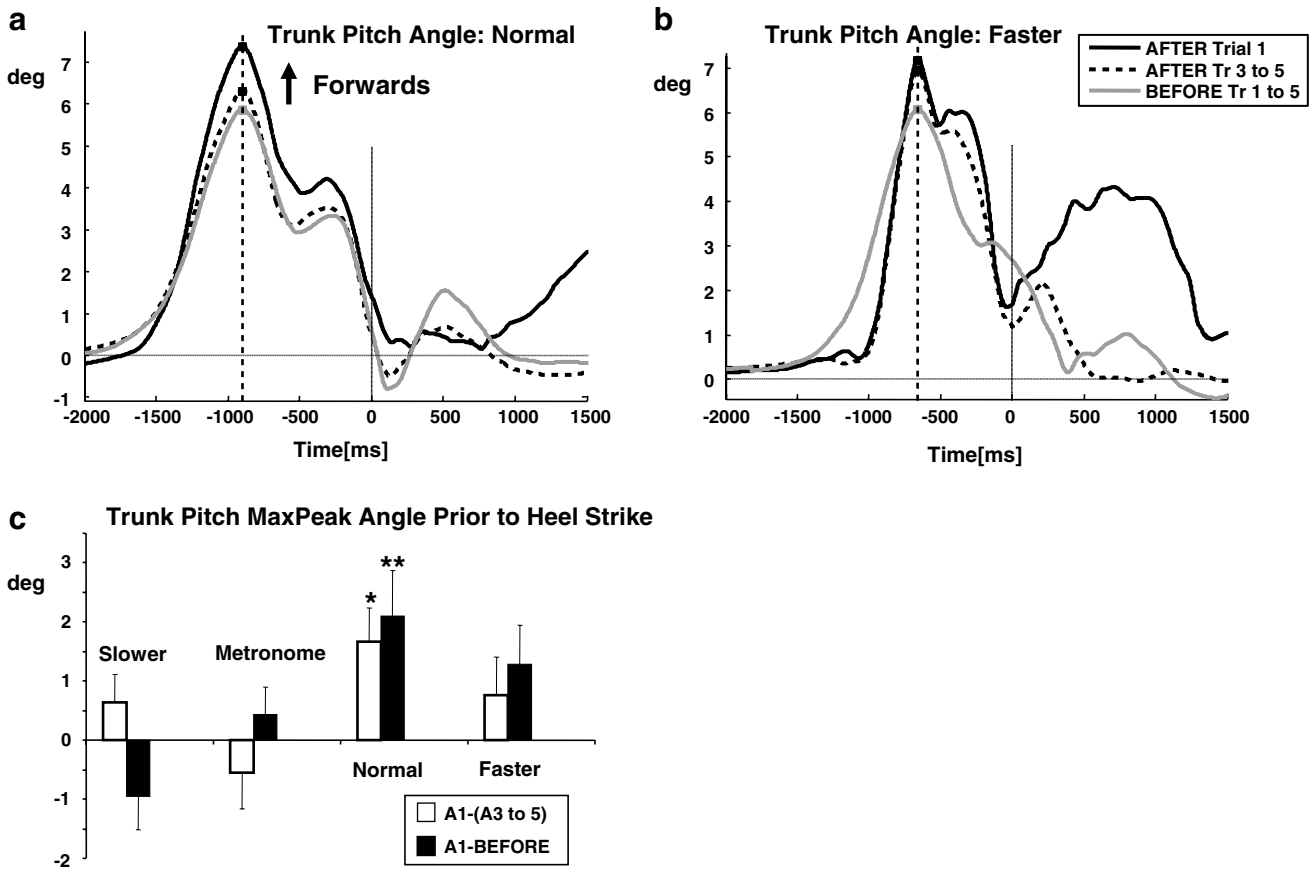


**Fig. 4** Trunk pitch prior to left heel strike. Mean traces for 10 subjects of trunk pitch aligned at a mean alignment time of 898 ms prior to heel strike for normal walking (**a**) and at 656 ms for faster walking (**b**). The mean AFTER trial 1 amplitude is greater than BEFORE and AFTER trials 3–5 for normal walking, but only greater than BEFORE trials for faster walking. Note that the trunk flexion after heel strike in 1st AFTER trials is shifted forward for faster walking. **c** The mean trunk pitch AE for 4 different walking speeds. Refer to the legend of Fig. 3 for details of the layout of **c**

trunk pitch angles between −2 s and −0.5 s (prior to heel strike) were aligned to account for differences in gait cycle times with GAV. The average time point of this peak was −1.064 s for slower, −0.842 s for metronome, −0.898 s for normal and −0.656 s for faster walking. That is, shorter shifts for faster GAV, as expected. For display purposes, in Fig. 4a, b, we aligned traces to the average shift of 0.898 and 0.656 s, respectively. As shown in Fig. 4c, trunk pitch angle in AFTER Trial 1 was greater than BEFORE trials for all groups except the slower group. However, the difference was only significant for normal walking speed ($p = 0.023$). The increase in peak trunk pitch angle prior to heel strike with respect to BEFORE trials showed a weak correlation ($R = 0.24$, $p < 0.05$) between the trunk pitch AE and GAV. Similar results were obtained with respect to AFTER trials 3–5 for the normal walking group.

## Left shank angular velocity

Shank angular velocity increased in the first backward swing of the left shank as the body moved forward (Fig. 5). We analysed peak backward shank velocity after aligning the peak velocity between 2 and 0.5 s prior to left heel strike (see Fig. 5a, b), to account for differences in gait cycles with GAV. As increased GAV was a component of the AE (see above) and peak shank angular velocity is highly correlated with GAV (see Fig. 5c; $R = 0.69$, $p < 0.0001$), we were not surprised to observe an AE component in shank velocity with respect to AFTER trials 3–5 for all velocity groups ($p \leq 0.03$; Fig. 5a). Shank velocity was also increased with respect to BEFORE trials ($\Psi^2 = 25$, $df\,3$, $p < 0.001$), except for slower trials ($p \geq 0.05$; Figs. 3a, 5d).

## EMG activity in lower leg muscles

Increased EMG activity was observed as AE components in two phases of lower leg muscle activity terminating 200 ms prior to heel strike and starting 200 ms post-heel strike. These AE components were not observed in trunk muscles. The first phase of activity was present prior to forward swing of the left foot as seen in previous studies,

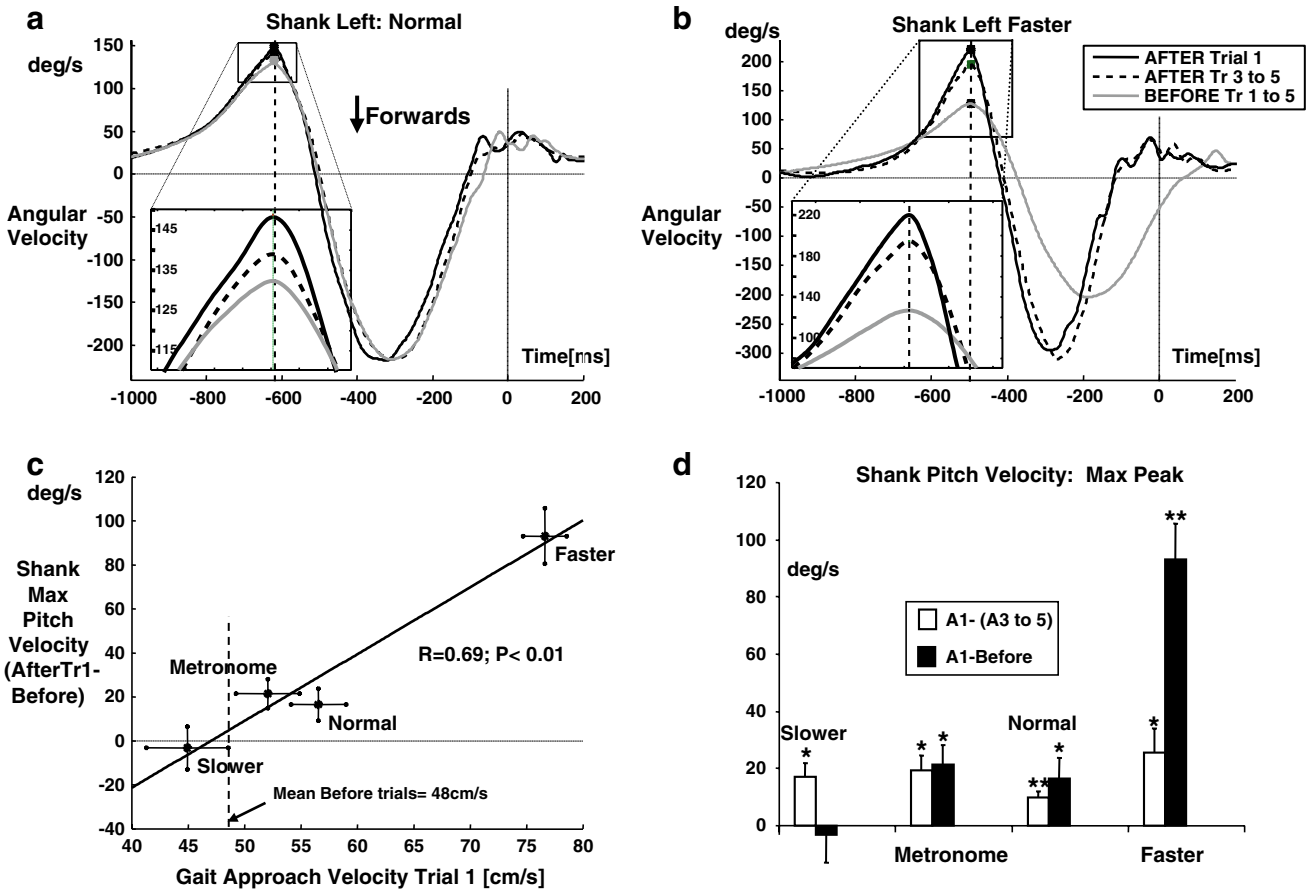


**Fig. 5** Maximum shank velocity at gait onset. **a**, **b** Mean traces of shank velocity at gait initiation aligned at population mean peak time: 620 ms prior to left heel strike for normal walking and at 496 ms for faster walking. The profiles of the peak backward shank pitch velocity (relative to pitch at gait onset) are shown expanded in inserts. **c** The regression between GAV and the shank pitch velocity in AFTER trial 1 with respect to BEFORE trials, and indicates no AE in shank velocity when GAV is the same as in BEFORE trials as for slow walking AFTER trial. **d** The increased shank velocity associated with increased gait velocity of AFTER trial 1 for each of the 4 requested velocities during AFTER trials. The means and SEM are depicted as shown in Figs. 3c and 4d

prior to time −0.2 s (Reynolds and Bronstein 2003; Bunday and Bronstein 2008, 2009). This activity was particularly prominent in tibialis anterior for faster gait (see traces in Fig. 6a). Simultaneous with left heel strike on the sled, there was a period of strong co-activity in the lower leg (Fig. 6a) and trunk muscles (not shown) which was highly correlated with GAV in the lower leg muscles (Fig. 6c), but weakly correlated in the trunk muscles (Fig. 6d). This activity presumably acted to terminate gait. After this large burst of activity associated with heel strike, EMG activity decreased. The second phase of increased activity only occurred during AFTER trial 1. It commenced in the leg muscles 200 ms post-heel strike and again was not seen in trunk muscles. This "extra" braking activity in AFTER trial 1, as measured between 200 and 700 ms post-heel strike, was greater than BEFORE trials and AFTER trials 3–5 for normal and faster walking in the tibialis anterior and soleus muscles ($p < 0.05$, see Fig. 6b).

## Discussion

We manipulated requested baseline gait approach velocity (GAV) during the AFTER trials to explore the relationship between the trunk overshoot and increased GAV components (above baseline) of the "broken escalator" after-effect. We considered two possibilities as cause of the trunk overshoot. The first is that an increase in the trunk flexion or overshoot is a direct inertial consequence of the increased GAV, much as people inside a bus that suddenly stops sway further when the bus is travelling at a faster rather than slower velocity. The second is that the increased trunk overshoot component of the after-effect (AE) is independent of the increased GAV. Our data suggest that both mechanisms contribute to the AE—the trunk overshoot increases with GAV, but is also present when gait had the same ("normal") speed as in BEFORE trials (as seen in the "slower" group for AFTER trial 1 with no increased GAV). Thus, the forwards trunk

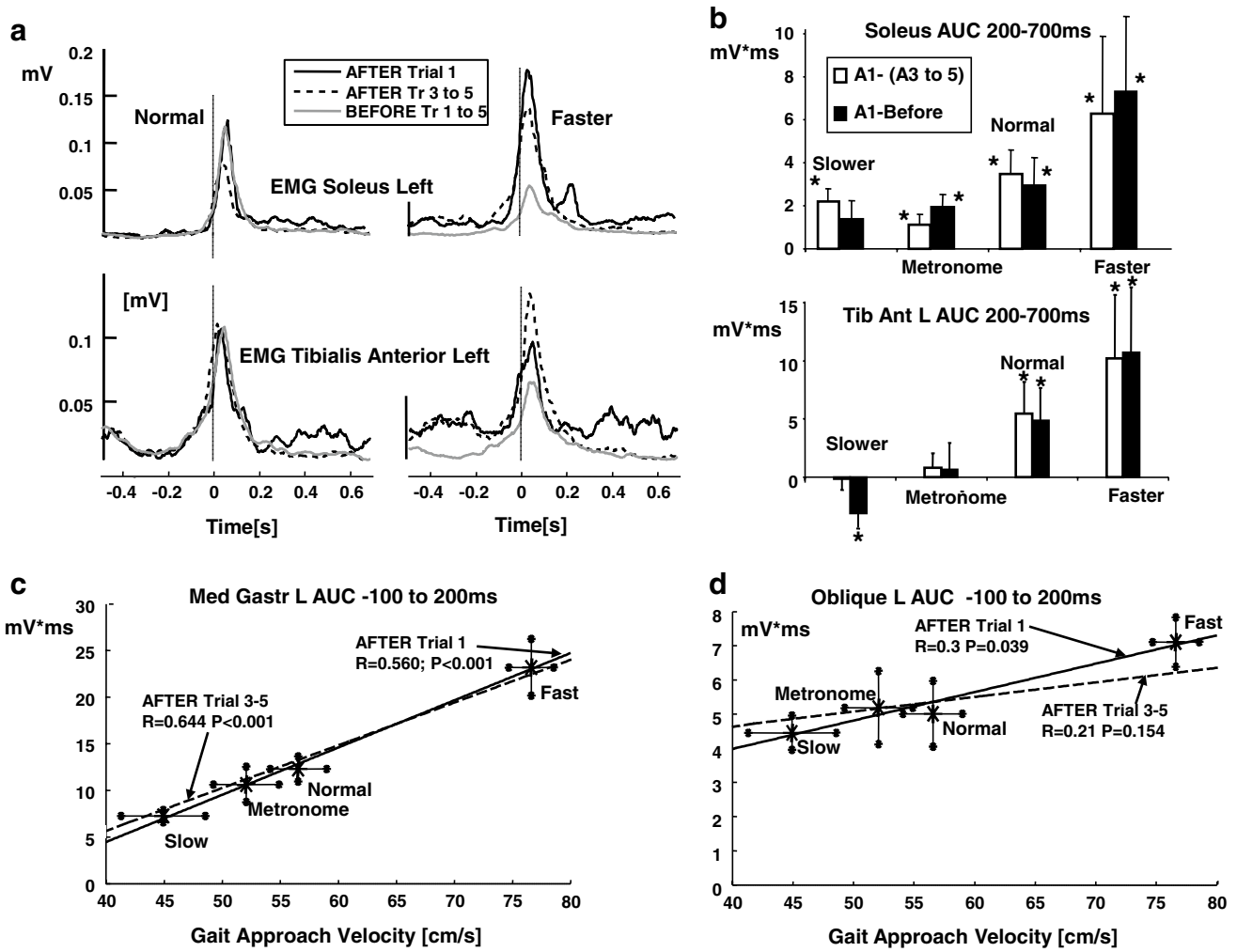


**Fig. 6** Lower leg EMG responses at left heel strike. In **a** mean traces are shown aligned with heel strike (0 s) for the left soleus, and tibialis anterior muscles. The mean areas under the curves (*AUC*) of these responses between 0.2 and 0.7 s post-heel strike with respect to BEFORE and AFTER trials 3–5 are shown in **b** for the soleus and tibialis anterior muscles. In **c**, **d** regressions of the AUC of left medial gastrocnemius and left external oblique muscles for the period −0.1 to 0.2 s post-heel strike against GAV are shown. The means and SEM for each walking velocity are depicted for AFTER trial 1 only

overshoot component of the AE is not just a consequence of higher gait speed, as shown by the regression in Fig. 3c, but part of an additional postural phenomenon that we call "pre-emptive". However, GAV and trunk overshoot cannot be completely dissociated. As GAV increased, an increase in the size of the AE was observed in trunk, shank and EMG measures. Therefore, there is a significant but not fully explanatory association of these AE measures with GAV.

## Gait approach velocity

Increased gait velocity before foot-sled contact in AFTER trial 1 is one component of the locomotor AE, even when subjects were explicitly asked to walk at normal speeds as in BEFORE trials (Reynolds and Bronstein 2003, 2004, 2007; Bunday et al. 2006) or a metronome is provided as a cue for normal speed. Here we show that despite no increase in GAV, when walking "slower" in AFTER trials, subjects had both a trunk overshoot (Fig. 3d) and increased left leg EMG activity (Fig. 6a, b). This provides direct evidence that the trunk overshoot AE is not the direct (inertial) consequence of the increase in gait velocity observed in the first AFTER trial. Previous evidence supporting this view was indirect, based on the low level correlation present between GAV and the magnitude of the trunk overshoot (Bronstein et al. 2009). More recently, differential effects on GAV and trunk displacement were observed in healthy subjects using non-invasive cortical brain stimulation over primary and pre-motor cortex, suggesting that the two are subserved by different neuronal mechanisms (Kaski et al. 2012).

Analysis of shank pitch velocity showed that the increase in the gait velocity component of the AE was present at gait

initiation, in line with EMG changes observed during the onset of gait (Bunday and Bronstein 2008). The fact that biomechanical changes were observed at gait initiation and prior to foot-sled contact supports the concept that the increase in gait velocity is mediated by open loop feed-forward mechanisms (Reynolds and Bronstein 2003).

A number of additional features in our data indicate that GAV is a contributing, but not fully explanatory factor to the generation of the trunk overshoot components of the after-effect. Thus, although shank velocity in AFTER trial 1 was equal to that of BEFORE trials for the "slower" group (Fig. 5d), forward trunk linear displacement increased compared to BEFORE trials (Fig. 3d). Additionally, trunk overshoot and increased leg muscle activity were observed in the "faster" group only in AFTER trial 1, not AFTER trials 3–5 (Fig. 6a, b), even though GAV for AFTER trial 1 was only marginally increased with respect to AFTER trials 3–5 (Fig. 3a).

### Trunk displacement

The forwards pitching of the trunk prior to stepping on the sled was greater during the MOVING trials 3–5, indicating that this is a main adaptation strategy deployed to protect balance stability (e.g. note the large and potentially destabilising backwards pitching at time +1 s during the first MOVING trial in Fig. 2). Also, in the first AFTER trial, the continuing forwards pitching of the subjects' trunk is more than that of the BEFORE trials, implying a subconscious expectancy that the sled may move. After foot-sled contact, however, the MOVING and AFTER trial 1 traces diverge. During MOVING trials, the forwards sled motion still induces inertial backwards trunk pitching, whereas in AFTER trial 1, the trunk pitches forward between times +0.5 and +1.7, synchronously with the forwards linear trunk overshoot detected in this and previous studies (Reynolds and Bronstein 2003, 2004, 2006, 2007; Bunday et al. 2006). Our results therefore show firstly that the linear trunk overshoot is largely the result of trunk pitching and secondly that such forwards inclination of the trunk adaptively develops during the MOVING trials in order to counteract the risky backwards pitching of the trunk (compare MOVING 1st and AFTER trials 3–5 at time ca. +1 s in Fig. 2). The trunk flexion is not solely an inertial consequence of increased GAV but rather a learnt or "adaptive" mechanism, as discussed below. The muscles responsible for the trunk pitching overshoot cannot be fully ascertained here, but our current recordings suggest a strong role of ankle muscles. Trunk muscle activity underlying trunk flexion observed during 1st trial effects to balance perturbations (Oude Nijhuis et al. 2010) was not observed to contribute to the AE of this study.

What is the source of the trunk overshoot? We consider that it is related to the compensatory forward pitching of the trunk during the MOVING trials and its inappropriate release in AFTER trial 1—presumably as the CNS (wrongly) predicts that the sled may move despite external reassurances that it will not. Such predictions are needed since relying on visual input at perturbation onset inevitably leads to delayed responses and greater instability (Jacobs and Horak 2007). Given our inference that it is not just the inertial consequence of increased GAV, the trunk AE appears to be an independently generated adaptive postural phenomenon. It has been suggested that postural responses may be "primed" prior to a semi-predictable perturbation (Burleigh and Horak 1996) and that further, later adjustments are triggered by the nature of the perturbation itself. Such primed responses occur "in anticipation of an impending loss of balance" (Jacobs and Horak 2007) in situations of upcoming external perturbations. In the case of the broken escalator phenomenon, a better term for the forward trunk flexion would be "pre-emptive postural response" (PEPA) as there is no external perturbation—the sled is stationary. The trunk motion observed in AFTER trial 1 is pre-empting the consequences of a possible destabilising stimulus. If the sled did move, subjects would fall backwards, so the pre-emptive forward trunk motion on heel-sled contact would counteract the effects of sled motion. The question that could be raised in the light of recent work (Campbell et al. 2012) is whether the pre-emptive response has been conditioned during the BEFORE trials. The conditioning event, in this case, could be stepping on to the sled or the non-startling acoustic beeps given to start the trial.

One could think of this pre-emptive movement as an anticipatory postural adjustment (APA) in a broad sense; however, this term is usually reserved for neuro-physiological events taking place prior to actual movements of other segments, such as EMG and biomechanical changes in the leg contralateral to the one initiating gait (Breniere et al. 1987; Martinez-Mendez et al. 2011) or calf muscle activation before raising the arms (Friedli et al. 1984, 1988). The underlying mechanism of this pre-emptive postural adjustment maybe an aversive or "fear-related" conditioned reflex (Davis 1992). In this case, the fear of a fall should the platform move would be the aversive stimulus and the trunk flexion the conditioned response. The observation that the broken escalator AE can be experimentally induced in just one adaptation trial, as in aversive conditioned responses (Maren 2001), has been invoked in support of this view (Bunday et al. 2006). Thus, we consider this pre-empive response occurs when there is a change from the existence of a perturbations (in MOVING trials) to no perturbation (as in AFTER trail 1). In order to provide further support for the pre-emptive concept, future investigations should consider qualifying the level of confidence subjects have that the walkway will

not move. Presumably, low levels of confidence would be correlated with a greater AE.

An alternative concept for the AE generation would be that it is simply a 1st trial effect. Similar to the AE, 1st trial effects following balance perturbations diminish with three repetitions and contain startle-like components (Oude Nijhuis et al. 2010). There are, however, two major differences between the AEs described here and 1st trial effects to balance perturbations. Firstly and perhaps crucially, the balance perturbation is internally generated for the AE and not externally applied. Secondly, AEs are present as additional activity in leg and not trunk muscles, whereas 1st trial effects are present predominantly in trunk, arm and neck muscles (Tang et al. 2012).

### Gait velocity modulation of the trunk after-effect

Although the trunk overshoot component of the after-effect is not the direct result of GAV, the two are necessarily related. Two points need to be considered. Firstly, Green et al. (2010) demonstrated that the trunk overshoot increases if gait velocity is increased in the moving trials by employing sled velocities greater than 1.4 m/s. However, in contrast to the current experiments where sled velocity was kept constant, the increase in trunk motion in the Green et al. experiments was explained by the need for a stronger postural compensation required for a faster stimulus and the associated "risk assessment" implemented by the subjects (faster sled, greater risk; Green et al. 2010). Secondly, in the current experiments, a state of greater neuronal excitability in locomotor circuits required for walking faster may modulate the size of the trunk overshoot despite the standard sled velocity (1.4 m/s) employed in the moving trials. This may be due to sharing of common neuronal networks by postural and locomotor mechanisms, as observed in other balance mechanisms (Allum et al. 2008). Alternatively, there is likely some inertial contribution to trunk flexion with a faster gait. A separation of these two hypotheses would require new experiments keeping gait velocity constant whilst varying the subject's inertial load with added weights.

## Conclusion

A trunk overshoot and increase in shank and gait approach velocity characterise the after-effect of stepping onto a stationary sled immediately following adaptation to a moving sled—the "broken escalator phenomenon". The trunk overshoot is not directly caused by increased gait approach velocity although there may be a synergistic interaction between locomotor and postural mechanisms. We propose a new category of pre-emptive postural adjustment (PEPA) to explain the trunk overshoot observed as part of the after-effect although we can not completely rule out involvement of a first trial effect.

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