Directional Sensitivity of “First Trial” Reactions in Human Balance Control

Lars B. Oude Nijhuis,1,2 John H. J. Allum,1 George F. Borm,3 Flurin Honegger,1 Sebastiaan Overeem,2 and Bastiaan R. Bloem2

1Department of Otorhinolaryngology, University Hospital, Basel, Switzerland; and 2Department of Neurology, Donders Institute for Brain, Cognition and Behavior, Center for Neuroscience and 3Epidemiology and Biostatistics, Radboud University Nijmegen Medical Centre, Nijmegen, The Netherlands

Submitted 11 September 2008; accepted in final form 4 March 2009

Oude Nijhuis LB, Allum JHJ, Borm GF, Honegger F, Overeem S, Bloem BR. Directional sensitivity of “first trial” reactions in human balance control. J Neurophysiol 101: 2802–2814, 2009. First published March 11, 2009; doi:10.1152/jn.90945.2008. Support-surface movements are commonly used to examine balance control. Subjects typically receive a series of identical or randomly interspersed multidirectional balance perturbations and the averaged response is used for further analyses. Other studies have used a random mix of multidirectional support-surface perturbations (to reduce stimulus predictability) and, again, the averaged response for a given perturbation direction is used post hoc for further analyses (Allum et al. 2002; Dimitrova et al. 2004; Henry et al. 2001).

One of several known drawbacks to the averaging approach is that the amplitude of postural reactions gradually diminishes with stimulus repetition—a phenomenon called adaptation or habituation (Keshner et al. 1987; MacPherson 1994; Nasher 1976). Studies using surface electromyography (EMG) have shown that the response amplitude is typically greatest when the postural perturbation is delivered for the very first time (Hansen et al. 1988; Keshner et al. 1987; Nasher 1976). This “first trial response” is a consistent phenomenon that has been observed under widely varying experimental conditions (Blouin et al. 2003; Brady et al. 2000; Chong et al. 1999; Marigold et al. 2003; Siegmund et al. 2003; Wu 1998). The greatest amplitude reduction across trials occurs between the first balance perturbation and the second, identical one. A further, more gradual habituation is seen across the next set of identical perturbations (Hansen et al. 1988; Keshner et al. 1987). The first trial response is usually excluded from further analyses because it looks different compared with subsequent reactions (Allum et al. 2002).

Excluding the first trial reaction might well lead to loss of relevant information. First, it is neurophysiologically interesting to examine the specific nature of the first trial reaction—for example, to identify the critical triggers that can provoke this first trial effect. Second, first trial effects may be significant in terms of impact on postural control depending on the direction of the balance perturbation. We suspect that large EMG amplitudes or early onsets associated with first trial responses may lead to excessive balance corrections and, perhaps, even adversely affect balance if cocontraction occurs with loss of intersegmental flexibility. However, it remains unknown whether first trial reactions in different directions are associated with worsening, no effect on, or perhaps even improvement of postural control.

In this study, we first investigated whether the difference in habituation rate of postural stability between the very first and following second trial was larger compared with the habituation rate in following trials regardless of perturbation direction. This was referred to as the first trial effect. Next, we investigated whether the magnitude of the first trial effect depended on the direction of the perturbation. Then, we investigated whether the first trial effect could be reinstated in fully habituated subjects by a sudden change of

INTRODUCTION

Investigating equilibrium and unraveling the pathophysiology underlying falls are complex because many factors contribute (Bloem et al. 2003a). One commonly used method to evaluate balance is dynamic posturography, using standardized balance perturbations, often through sudden movements of a support surface (Bloem et al. 2003b; Horak and Nasher 1986). In many of these experiments subjects are exposed to a series of identical support-surface perturbations and the averaged response is used for further analyses. Other studies have used a random mix of multidirectional support-surface perturbations (to reduce stimulus predictability) and, again, the averaged response for a given perturbation direction is used post hoc for further analyses (Allum et al. 2002; Dimitrova et al. 2004; Henry et al. 2001).

One of several known drawbacks to the averaging approach is that the amplitude of postural reactions gradually diminishes with stimulus repetition—a phenomenon called adaptation or habituation (Keshner et al. 1987; MacPherson 1994; Nasher 1976). Studies using surface electromyography (EMG) have shown that the response amplitude is typically greatest when the postural perturbation is delivered for the very first time (Hansen et al. 1988; Keshner et al. 1987; Nasher 1976). This “first trial response” is a consistent phenomenon that has been observed under widely varying experimental conditions (Blouin et al. 2003; Brady et al. 2000; Chong et al. 1999; Marigold et al. 2003; Siegmund et al. 2003; Wu 1998). The greatest amplitude reduction across trials occurs between the first balance perturbation and the second, identical one. A further, more gradual habituation is seen across the next set of identical perturbations (Hansen et al. 1988; Keshner et al. 1987). The first trial response is usually excluded from further analyses because it looks different compared with subsequent reactions (Allum et al. 2002).

Excluding the first trial reaction might well lead to loss of relevant information. First, it is neurophysiologically interesting to examine the specific nature of the first trial reaction—for example, to identify the critical triggers that can provoke this first trial effect. Second, first trial effects may be significant in terms of impact on postural control depending on the direction of the balance perturbation. We suspect that large EMG amplitudes or early onsets associated with first trial responses may lead to excessive balance corrections and, perhaps, even adversely affect balance if cocontraction occurs with loss of intersegmental flexibility. However, it remains unknown whether first trial reactions in different directions are associated with worsening, no effect on, or perhaps even improvement of postural control.

In this study, we first investigated whether the difference in habituation rate of postural stability between the very first and following second trial was larger compared with the habituation rate in following trials regardless of perturbation direction. This was referred to as the first trial effect. Next, we investigated whether the magnitude of the first trial effect depended on the direction of the perturbation. Then, we investigated whether the first trial effect could be reinstated in fully habituated subjects by a sudden change of
perturbation direction. For these studies we used a design that incorporated series of identical perturbations called blocks. These blocks of trials in different directions were randomized across subjects. Our primary hypothesis was that pitch-directed perturbations would induce the greatest first trial effects because this direction is associated with the largest and earliest head accelerations (Allum et al. 2008). In keeping with a generally absent influence of vestibular cues on the onsets but not on the amplitudes of balance corrections (Carpenter et al. 2001a), we assumed that first trial effects would be amplitude and not onset dependent.

METHODS

Participants

Thirty-six healthy subjects (18 men; mean age 23 yr, range 19–30 yr) participated. Exclusion criteria included self-reported neurological, balance, or musculoskeletal disorders. Participants gave written informed consent prior to the experiment. We purposely included a homogeneous group of young and healthy subjects, to reduce variability in the data and to obtain a clear view of first trial effects. Experiments were conducted according to the standards of the Declaration of Helsinki. The Institutional Review Board of the University Hospital Basel approved the study. Subjects were paid a nominal fee for their participation.

Experimental protocol

Balance control was assessed using previously used techniques (Allum et al. 2002; Carpenter et al. 1999). Subjects had never before participated in a posturography experiment. Participants stood on a servo-controlled dual-axis rotating platform with their arms hanging by their sides (Fig. 1A). The ankle joint was aligned with the pitch axis of the platform and the roll axis passed between the feet. Two assistants were present to lend support in case of an actual fall.

FIG. 1. Experimental setup and design. A: experimental setup. From the infrared emitting diode (IRED) positions the vector center of mass (CoM) was calculated as an “overall” measure of postural performance. B: view from above of the servo-controlled dual-axis rotating platform. The platform delivered standardized tilts in 6 different directions that were defined in degrees. C: schematic representation of the Latin-square design used. Subjects received 6 blocks of 10 identical stimuli, each block with a different perturbation direction. For a separate study, subjects received another 3 blocks with a different type of stimulus, which were not included in the present analysis. The order of all blocks was counterbalanced across subjects using a Latin-square design, so each possible stimulus was once delivered as first.
**Platform rotations**

Subjects were tilted by the support-surface platform at a constant amplitude of 7.5° and velocity of 60°/s. Platform tilts occurred in six different directions, defined in degrees, where 0° reflected facing forward (Fig. 1B). The directions were the same as previously published (Grin et al. 2007). Each platform tilt was preceded by a random 10- to 20-s delay, during which visual feedback of the participants’ own anterior–posterior and medial–lateral ankle torques was presented to the participant on a cross with light-emitting diodes positioned 4 m in front of the subject. The visual feedback was used to standardize the prestimulus position of participants across trials and a stimulus was not presented until ankle torque was within a range of 4 Nm.

**Design**

The experiment started without any preceding trials (which are normally done to familiarize subjects with the experimental conditions), so the very first perturbation was fully unpracticed. Subjects received six blocks of 10 identical stimuli (Fig. 1C), each block with a different perturbation direction. We used 10 stimuli per block in accordance with previous studies (Chong et al. 1999; Keshner et al. 1987; Nashner 1976). By using the blocked design we were able to quantify the first trial effect—the habituation between trials 1 and 2, compared with the habituation rate in trials 2 to 10—with perturbation direction. For a separate study, subjects received another three blocks with a different type of stimulus that were not included in the present analysis. The order of all blocks was counterbalanced across subjects using a Latin-square design, so each possible stimulus was delivered once as first. This randomization of blocks enabled us to investigate the possible reemergence of first trial effects with a sudden change of direction. Leaving out the three blocks with a different stimulus type, the randomization design resulted in 24 subjects receiving one “true” first trial that was fully unpracticed (including the new experience of standing on a tilting platform), as well as five more first trials whenever the perturbation direction was changed.

![FIG. 2. Amplitude of the vector CoM over time. A: traces of the vector CoM for trials 1, 2, and 10 in the first block of 10 identical tilts. B: total area under the curve (±95% confidence interval [CI]) of the vector CoM, averaged per trial number, irrespective of the direction of platform tilt.](http://jn.physiology.org/Download)
Before the start of the experiment, subjects were informed that they were about to be perturbed in multiple different directions, but the specific nature of the experiment and the number of possible perturbation directions was not specified. Subjects had no prior knowledge about the characteristics and the direction of the platform movements. The interval between trials and between blocks was randomly varied.

**Outcome measures**

We recorded kinematic, kinetic, and electromyographic responses. Participants were instrumented with 18 infrared emitting diodes (IREDS) to collect full body kinematics (Fig. 1A). The IREDs were placed on the following anatomical landmarks: frontally at the level of the malleoli, at the center of the patellae, frontally at the level of the greater trochanters, anterior superior iliac spine, elbow axis, acromion, processus styloïdeus, temple, one at the chin and one at the sternum angle. Three additional IREDs, placed at both front corners and one at a back corner of the rotational surface, were used to track all pitch and roll movements. The OptoTrak motion analysis system (Northern Digital Canada, Waterloo) tracked the IREDs with a frequency of 64 Hz.

Ankle torques were calculated from support-surface reaction forces measured with strain gauges embedded in the rotating support surface. Head linear and angular accelerations were computed using analog signal processing from the outputs of four dual-axis linear accelerometers (Entran, Lexington, KY), with ranges of ±5 g, each mounted at 90° separation (as viewed in the transverse plane) on a lightweight, adjustable, tight headband. Head-vertical linear acceleration was computed, for example, from the sum of all four accelerometer signals with a vertical component, whereas head roll acceleration was computed from the difference in vertical linear accelerations at the ears. Accelerometers were adjusted to be in the gravity plane at the start of the experiment, but not corrected thereafter. All analog-computed signals were sampled at 1,024 Hz.

Surface electromyography (EMG) signals were recorded on the left side from tibialis anterior, soleus, gluteus medius, external oblique, and paraspinous muscles at the L1–L2 level of the spine, and triceps brachii, medial deltoid, and sternocleidomastoid muscles. Pairs of silver/silver chloride electrodes were placed about 3 cm apart along the muscle bellies and the electrodes as well as lead lengths assigned to individual muscles were not changed between

---

**FIG. 3.** Amplitude of the vector CoM per platform direction. A: in the top panels, traces of the vector CoM displacement across trials 1, 2, and 10 are shown, for balance perturbations that were either directed backward (158 and 203°), forward (45 and 315°), or lateral (113 and 248°). B: mean total area under the curve (AUC) of the vector CoM for the forward (45 and 315°), backward (158 and 203°), and lateral (113 and 248°) directions. The error bars indicate the 95% CIs. For the statistical analyses, the 6 independent perturbation directions were divided into their forward, lateral, and backward components.
subjects. EMG amplifier gains were kept constant throughout the experiments. EMG recordings were band-pass analog filtered between 60 and 600 Hz, full-wave rectified, and low-pass filtered at 100 Hz with a third-order Paynter filter prior to sampling at 1 kHz. All biomechanical and electromyography recordings were initiated 100 ms prior to the onset of platform rotation and had a sampling duration of 1 s.

**Data analysis**

VECTOR COM. The primary outcome measure was an overall measure of balance called “vector CoM,” based on the displacement of the center of mass (CoM) in the anterior–posterior, medial–lateral, and vertical planes (Carpenter et al. 2001b). The CoM displacement was calculated using a 12-body segment adaptation of the 14-body segment model of Winter et al. (2003) (Visser et al. 2008). We calculated as the area under the curve (AUC) of the rectified total CoM displacement in each plane, using trapezoid integration within the interval of 100 to 800 ms from stimulus onset. Finally, the AUC of vector CoM was calculated as a vector “length” of the integrals in the anterior–posterior, medial–lateral, and vertical planes for each individual trial (Visser et al. 2008).

VECTOR COM: VERY FIRST TRIAL. To investigate the impact of the very first trial across all directions, the average vector CoM in trial 1 was compared with trial 2. The difference between the two trials was compared with trials 2 to 10, across all platform directions (Fig. 1C). This was referred to as the “first trial effect.” To investigate the first trial effect over time, the average vector CoM across trial 1 in the remaining blocks (trials 11, 21, etc.) was compared with trials 2 to 10 within those blocks. An example of the way the vector CoM was averaged in the aforementioned comparisons is given by the left gray column of Fig. 1C, showing the averaging of the first trial in the second block (trial 11).

Subsequent analyses showed that the first trial effect diminished throughout the experiment, being most prominent during the first two blocks. Therefore all following explorative analyses on the nature of the first trial effect used the first two blocks that displayed the greatest first trial effect.

VECTOR COM: INFLUENCE OF PLATFORM DIRECTION. To investigate the influence of rotation direction on the first trial effect, the vector CoM was averaged within each direction of platform rotation (Fig. 1C, right gray column). Thus the first trial was compared with trials 2 to 10 within each of the six rotation directions.

ANKLE TORQUES. To measure differences in ankle torques between the first and second trials, peak amplitudes of these biphasic signals were determined. An interval of 50 ms around these peaks was used for calculation of the AUC. Furthermore, the time of the peaks was calculated.

ELECTROMYOGRAPHY. EMG amplitudes were calculated from the EMG traces of the individual trials. Trapezoid integration was performed over a balance-correcting interval of 100 to 200 ms after onset of the platform rotation. Furthermore, the amplitude was calculated over an interval of 700 to 800 ms, when signals had reached plateau values as the body was set in a new stable position. The EMG areas were corrected for baseline EMG activity prior to stimulus onset. EMG onset latencies were calculated using a semiautomatic computer algorithm that searched for a response peak and subsequently moved backward in time and determined when the signal of the individual trace exceeded 2.5SDs above background muscle activity. All traces were visually inspected and checked for consistency.

BODY SEGMENT MOVEMENTS AND HEAD ACCELERATIONS. To investigate the changes previously observed in CoM more closely, the changes in individual segment angles that were incorporated in the CoM model and the head accelerations were calculated. Therefore the total area under the response curve was calculated over an interval of 100 to 800 ms after onset of platform movement.

NEAR-FALLS. The number of near-falls was registered as well as the trials and platform directions during which these occurred. A response...
was defined as a near-fall when the subject required external support to prevent a fall.

Statistical analyses

Prior to analysis all data values were log-transformed to correct for the skewed distributions. For analysis of the main outcome measure (vector CoM), we used a linear random effects model (mixed-model analysis): to determine the presence of the first trial effect within each block; to compare the first trial effect recorded in block 1 and block 2 with the first trial effects in blocks 3 to 9; and to compare the first trial effect between the different platform rotation directions. For these latter statistical analyses, the six independent platform directions were divided into their forward, lateral, and backward components. Results of the analyses were back-transformed into percentages. To explore the changes underlying our main findings, we used Student’s paired t-test to compare differences between the first and second trials for anterior–posterior ankle torque, body segments, head accelerations, and EMG values.

Vector CoM values are presented as means ± 95% confidence intervals (CIs). For all other values the data values were presented as means ± SDs. Differences with a P value of <0.05 were considered significant.

RESULTS

Biomechanical impact of the first trial reaction

All subjects had, by definition, only one very first trial that was fully unpracticed. When averaged across all directions, this very first balance perturbation resulted in greater postural instability (reflected by greater CoM displacements) compared with all subsequent responses to identical balance perturbations (Fig. 2A). For example, within the first block, the mixed-model analysis showed that the amplitude of vector CoM was 15% greater during the very first trial, compared with trials 2–10 (P < 0.0001; Fig. 2, A and B).

First trial effect over time

The first trial effect (with greater displacement of the CoM) reemerged in subsequent blocks (Fig. 2B). In other words, immediately following a change in rotation direction, the
amplitude of vector CoM was 5–15% greater in the first trial of a particular block compared with the subsequent and identically directed perturbations. However, the magnitude of the first trial effect diminished as subjects were exposed to increasingly more blocks of perturbations (Fig. 2). The first trial effect was 9% greater in the very first block compared with that in blocks 3 to 9 ($P < 0.01$). In addition, the effect was 6% greater in the second block compared with blocks 3 to 9 ($P = 0.10$).

**Influence of rotation direction on the first trial effect**

The first trial effect on vector CoM was greatest for backward-directed platform rotations (first trial 33% greater than subsequent trials; 95% CI = 22–45%; $P < 0.0001$), in particular for the backward right rotations (158°), and was smallest (3% difference; 95% CI = −4 to 10%; $P < 0.10$) for laterally directed rotations (Fig. 3, A and B). For forward rotations the first trial effect was 12% (95% CI = 5–19%; $P < 0.01$).

Overall instability was also directionally dependent: for backward rotations, the amplitude of vector CoM for the first trial was 19% (95% CI = 7–32%) greater compared with forward rotations and 28% (95% CI = 16–43%) greater compared with lateral rotations ($P < 0.0001$).

**Further characterization of first trial reactions**

The first trial reaction was greatest for responses obtained following backward-directed perturbations (Fig. 3B). To further characterize the response differences between the first and second identical rotations, we concentrated on the 158° direction because this had the greatest effect (Fig. 3B). For these studies, we analyzed ankle torques, EMG responses, body segment displacements, and head accelerations. The CoM displacement for the first trial in the 158° direction was mostly directed backward and, to a lesser extent, downward and rightward (Fig. 4).

**Ankle torques for 158° rotations**

Peak ankle torques to a first backward platform rotation were delayed and had increased amplitudes compared with the second, identical perturbation (Fig. 5A). Specifically, the early (maximum) peak in the plantar flexion torque was significantly delayed in the first trial compared with the second trial (by 58.4 ms; $P < 0.01$; Fig. 5B, left). Furthermore, the maximum amplitude of plantar flexion torque was about one third larger in the first trial ($P < 0.01$; Fig. 5B, right). The subsequent peak dorsiflexion torque (which is normally associated with stabilization of the upright position) was less well defined in the first trial (Fig. 5A). It was also delayed (by 278.3 ms; $P < 0.01$; Fig. 5C, left) and had a greater amplitude in the first trial compared with the second ($P < 0.01$; Fig. 5C, right).

**Muscle responses for 158° rotations**

The amplitudes of muscle activity were generally higher in response to the first trial compared with the second trial (Fig. 6). The earliest change was an increased synchronized activity between 100 and 200 ms in all muscles, except tibialis anterior, for which the increased activity was extended considerably past 200 ms. A second change in activity was noted after 400 ms in all muscles shown in Fig. 6 (and summarized in Table 1). The first trial effect was significant over the period 100–200 ms in all muscles except tibialis anterior and paraspinals. Increases in background muscle activity could not explain the observed differences in automatic postural responses amplitudes at 100 ms. In fact, background activity in soleus and gluteus medius was significantly lower in the first trial compared with the second trial ($P < 0.05$). Earlier stretch reflex activity, such as in soleus, was also not increased in the first compared with subsequent trials.

Onsets of automatic postural responses at about 100 ms were significantly earlier in the first compared with the second trial.
in three muscles—sternocleidomastoid, triceps brachii, and gluteus medius ($P < 0.05$)—and showed a tendency for a delayed response in tibialis anterior ($P < 0.05$) (Table 3). For comparison, no significant differences were recorded for forward right (45°) rotations (Table 3).

**Body segment responses**

The individual body segment responses are represented in Fig. 7A for the backward right (158°) direction. This figure illustrates the difference between the first and second trials, from which three observations can be distilled. First, the kinematic changes help to explain the observed differences in CoM displacement between the first and second trials. This is demonstrated in particular by the large backward displacement of the pelvis, which will cause a backward displacement of the CoM. Movements in all segments were significantly increased in amplitude during the first trial, except for the ankle angle (Table 2). Second, this figure shows that the overall kinematic

![A](image1)

![B](image2)

**FIG. 7.** Body segment responses to the 1st and 2nd rotations. A: average traces of body segment movements to the 1st and 2nd rotations, as well as the platform inclination for 158°. B: average traces for 315°.
pattern within the first trial was compatible with a flexion response. Interestingly, this flexion response appeared to be a relatively nonspecific reaction because a very similar flexion response was seen for reactions induced by oppositely directed platform rotations, as visualized by forward (315°) rotations (Fig. 7B). For example, knee flexion for backward rotations appeared to be similar to what would be expected for forward rotations, even though knee flexion is detrimental for balance control when being perturbed backward. Third, Fig. 7, A and B shows clear differences between first trial reactions and later, more habituated reactions of the second trial, and also between reactions to forward and backward rotations. However, none of the observed kinematic changes between the first and second trials seemed to occur early enough to account for the early triggering of the first trial effect, which was observed as early as 100 ms posttrigger in EMG responses.

Head accelerations and ankle dorsiflexion

The only kinematic recordings that were able to detect sufficiently fast reactions to serve as trigger for the first trial effect were the ankle dorsiflexion velocity and head-vertical linear acceleration. Initially, ankle dorsiflexion is equal to the imposed platform rotation. Both ankle dorsiflexion velocity and head-vertical linear accelerations occurred sufficiently early (peaking within 50 ms posttrigger) to act as potential triggers for first trial reactions, but no clear differences between first and second trials were observed prior to 100 ms (Fig. 8). Early head-vertical accelerations were oppositely directed for forward and backward rotations; therefore a change in the direction of head motion (rather than its absolute magnitude) might have triggered first trial reactions whenever a new perturbation direction was introduced.

Near-falls in the first trial for 158° rotations

During the experiments, four near-falls occurred, all of them following backward-directed platform rotations. We examined these near-falls in detail because this could clarify the relevance of the above-cited findings for maintaining stability. Two of the near-falls occurred during the very first trial (in the first block) and two others occurred during the first trial in the second block (i.e., trial 11). Three of these near-falls were seen after a backward right perturbation (158°) and one for backward left (203°). The averaged response of the three near-falls for 158° is shown in Fig. 9, relative to the remaining first trials for this same perturbation direction where no fall had occurred. All first trial effects were even more pronounced when subjects sustained near-falls. Thus although all responses were first trial reactions, the increased backward CoM displacement was even more pronounced for near-falls compared with the nonfalls (Fig. 9). The backward CoM movement in the first trials where no near-fall occurred was considerably larger compared with that of the following identical trials. This meant that not all increases in CoM displacement during the first trial could be ascribed to the near-falls. Additionally, the peak plantar flexion torque was increased and delayed even further for near-falls compared with the nonfalls (Figs. 8 and 9, indicated by an arrow). The backward movement of the CoM was reinforced by the larger backward motion of the pelvis.
DISCUSSION

We studied the postural responses of young subjects to blocks of 10 identical balance disturbances, where for each new block the perturbation was changed to a randomly selected new direction. Our main findings were as follows. First, regardless of perturbation direction, subjects’ CoM moved significantly more following the very first and fully unpracticed balance disturbance, compared with the consecutive (identically directed) disturbances within that block (first trial effect). This finding is similar to that of previous studies (Horak and Nashner 1986; Keshner et al. 1987; Nashner 1976). Second, the first trial effect was greatest when subjects were perturbed backward, but much smaller for forward-directed perturbations and smallest for laterally directed perturbations. This directional dependence is suggestive of a greater influence of first trial effects from sensory signals strongly present for the backward direction. Third, whenever the perturbation direction was suddenly changed, the first trial effect immediately reemerged. This was clear for the first two blocks of perturbations and, after these, the effects were smaller. The smaller effects may be similar in amplitude to responses obtained when perturbation directions are randomized. Fourth, a nonspecific (direction-independent) flexion response was observed within the first trial. In particular, ankle flexion responses following backward perturbation are known to be associated with increased instability (Oude Nijhuis et al. 2007).

Fifth, the mere observation that the first trial reaction is associated with appreciably increased sway and with occasional near-falls suggests that the first trial reaction is perhaps a maladaptive response.

Impact of the first trial on postural stability

It has long been recognized that postural responses to unexpected balance perturbations differ from those obtained under “habituated” circumstances (Nashner 1976). However, little is known about the exact nature of this “first trial” effect. Prior work reporting the first trial focused mostly on EMG recordings and found excessive response amplitudes during the first trial (Bloem et al. 1998; Hansen et al. 1988; Keshner et al. 1987). Similar changes to EMG amplitudes were observed in our study. Response onset latencies of these automatic postural responses were earlier in three muscles. A tendency for delayed responses was observed in the tibialis anterior muscles ($P = 0.055$). Prior studies seldom included detailed kinematic or kinetic analyses. Our results show, for the first time, that the first trial effect is associated with prominent changes in balance control due to a flexion response. This effect was most pronounced for the response to the very first and fully unpracticed balance perturbation (of which there can be, by definition, only one). In addition we observed an increased peak plantar flexion torque and a delay in onset of the balance-correcting ankle torque for the first trial when subjects were perturbed backward. We assume that this change to the ankle torque response is caused by the strong first trial effect in the soleus EMG amplitude, but weak effect in tibialis anterior, and the tendency of the tibialis anterior response to be delayed for the first trial. The relevance of these effects is underscored by the fact that we observed several near-falls, all of which occurred during a first trial and two of which followed the very first balance perturbation. These near-falls contributed in part to the greater CoM displacement within the first rotations in the backward right direction. However, the trials in which no near-falls were recorded also showed greater first trial effects for this direction.

When the direction of the platform rotations was suddenly changed, the first trial effect reemerged immediately, again resulting in increased movement of the CoM. This was significant for the first two blocks, but the first trial effect appeared to habilitate over the course of the experiment. This habitation across blocks could not be explained by ordering effects because the perturbation directions were randomly varied.
across subjects. Subjects conceivably grew more familiar with the testing circumstances as they were exposed to an increasing number of balance perturbations. We cannot exclude that some subjects consciously or subconsciously started to predict the perturbation direction during the experiment. At baseline, subjects were informed that they would receive perturbations in multiple directions, but the specific nature of the experiment and the number of possible perturbation directions were not specified. However, each new block had a new perturbation direction and the remaining number of possible perturbation directions decreased as the experiment progressed, so some anticipation remains possible. We hypothesize that the amplitude of the first trial effect over blocks 3 to 9 might well be similar to amplitudes when perturbations in different directions are randomly administered.

The recording period after onset of the platform perturbation did not always seem sufficient to record a peak in the CoM profile of the first trial response. Because this was not only observed in the first trials in which subjects had fallen (see Fig. 9, top traces), this suggests that subjects were overall more unstable during the first trial. However, in hindsight we would have preferred a lengthier recording period to fully capture the first trial effect on CoM. Habituation mostly occurred between the first and second trials, with little further adaptation over the next eight trials. This is in keeping with prior observations on habituation of EMG amplitudes (Bloem et al. 1998; Hansen et al. 1988; Keshner et al. 1987). We show that this rapid habituation also applies to overall balance control (as indexed by the vector CoM displacement).

**Influence of perturbation direction and implications for possible triggers**

In terms of impact on postural control, the first trial effect was clearly greatest for backward-directed perturbations. Smaller first trial effects were also present for forward perturbations and effects were smallest for lateral perturbations. Several mechanisms may independently or in combination underlie these directional differences. Differences in base of support and signals arising from visual proprioceptive and vestibular sensory systems with perturbation direction may be responsible.

First, the impact of a backward-directed perturbation may be stronger because the base of support is smaller for backward directions compared with forward and lateral directions. Furthermore, visual feedback is less effective when perturbed backward, possibly creating difficulty in organizing postural reactions to backward falls (Schmid et al. 2007). In fact, visual feedback (eye closure) appears to have no influence on first trial reactions whenever a new perturbation direction was introduced. This anxiety may be heightened by a difference in early kinematic responses, the earliest recorded event following sagittal support-surface rotation (Carpenter et al. 2001b), highly similar to what we observed in the present study.

Although we noted no changes in early kinematic responses that would lead directly to first trial effects, it could be that heightened anxiety enhances responses triggered or modulated by sensory inputs. The first trial effects were observed in automatic postural responses with onsets in the range 100 to 130 ms (see Table 3). These are preceded for backward platform rotation by stretch reflexes in soleus muscles with onsets around 40 ms (see Fig. 6 and Allum et al. 2008), suggesting an early triggering by lower leg proprioceptive reflexes at the level of the ankle joint. An explanation fitting the directional dependence of the first trial effect may be this dependence on early lower leg proprioceptive reflexes. One may argue that the observed knee angle changes occurred too late to serve as trigger for the much earlier EMG changes. In fact these are not as well correlated with pitch displacements as ankle angular velocities (Allum et al. 2008). Thus it is unlikely that proprioceptive inputs arising at the knee joints underlie first trial effects unless ankle propioceptive inputs are absent (Bloom et al. 2001, 2002).

In addition, vestibular feedback based on head-vertical accelerations may explain the directional dependence of first trial effects, at least in the sagittal plane, and shed light on possible triggering mechanisms. The earliest first trial effects (as seen in EMG balance correcting activity) occurred as early as 100 ms after onset of platform movement, so triggering must occur within this short time interval. As mentioned previously, examining the kinetics and kinematics of first trial responses, the earliest recorded event following sagittal support-surface rotations is vertical head acceleration. This vertical head acceleration would be registered by the sacculi, making it a possible candidate as trigger for first trial reactions. The acceleration could also explain the observed directional dependence because it was oppositely directed for backward- and forward-directed rotations and weaker in lateral directions. Because there were no clear differences in early head accelerations between first and second trials, we suspect that other factors, such as anxiety, enhance head motion triggering larger first trial reactions whenever a new perturbation direction was introduced. This anxiety may be heightened by a difference in timing between the arrival at the CNS of confirmatory ankle proprioceptive information on the perturbation strength compared with the earlier arrival of saccular inputs elicited by head accelerations (Allum et al. 2008). That is, that perhaps somatosensory information on the support-surface movement is not

**Table 3. Mean electromyography (EMG) onset-latencies (SD) of muscle activity to platform rotations in forward right (45°) and backward right (158°)**

<table>
<thead>
<tr>
<th>Trial</th>
<th>Tibialis</th>
<th>Soleus</th>
<th>Gluteus</th>
<th>Oblique</th>
<th>Paraspinal</th>
<th>Triceps</th>
<th>Deltoid</th>
<th>Sternocleidomastoid</th>
</tr>
</thead>
<tbody>
<tr>
<td>Onset latencies</td>
<td>1 113.39 (25.11) 194.83 (27.80) 94.33 (13.99) 100.13 (11.61) 119.30 (46.50) 103.10 (22.95) 91.86 (20.36) 96.24 (27.11)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Forward (45°)</td>
<td>2 97.30 (16.39) 173.59 (28.24) 112.80 (21.04) 99.74 (16.32) 119.67 (19.28) 113.78 (16.55) 109.28 (17.02) 107.59 (11.20)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Student’s t-test</td>
<td>n.s. n.s. n.s. n.s. n.s. n.s. n.s. n.s.</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Onset latencies</td>
<td>1 115.07 (25.92) 123.93 (28.57) 97.05 (13.00) 103.88 (11.44) 111.82 (17.43) 101.73 (15.73) 107.06 (22.75) 99.74 (21.80)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Backward (158°)</td>
<td>2 102.18 (17.25) 134.09 (26.67) 128.67 (38.40) 117.77 (33.70) 105.11 (11.77) 126.47 (26.95) 116.98 (17.57) 115.24 (21.07)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Student’s t-test</td>
<td>0.053 n.s. * n.s. n.s. n.s. * n.s.</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

*P < 0.05; n.s., not significant.
integrated as efficiently with vestibular information in the first compared with subsequent trials. Supporting evidence for the role of anxiety-driven vestibular responses comes from the observation that early head motion was followed around 100 ms by early activation of sternoiclomastoid muscles, which rapidly habituate (see Fig. 6). Thus muscles are sensitive to otolitic inputs, specifically those of the sacculus (Colebatch and Halmagyi 1992; Colebatch et al. 1994). Furthermore, we observed strong first trial effects in soleus muscles, which also depend on vestibular modulation (Carpenter et al. 2001). Some support for vestibular influences comes from EMG studies that showed that habituation of EMG responses is diminished in subjects with bilateral vestibular loss (Keshner et al. 1987). This reduced habituation is primarily explained by the first trial responses, which in vestibular loss patients are smaller than those of controls. If a vestibular influence is predominant in first trial effects then the habituation effects may be similar to those observed in vestibular ocular reflexes to whole body rotations (Cohen et al. 1992).

Comparisons with startle responses

We suggest that first trial effects may be triggered and modulated by vestibular or proprioceptive signals associated with changes in perturbation direction. Alternatively, a (presumably posturally ineffective or even detrimental) startle reaction—triggered by somatosensory signals associated with the fall—could be superimposed on the “pure” balance-correcting responses. Indeed, startle reactions can be elicited by somatosensory signals, as demonstrated in recumbent healthy and vestibular loss subjects who were suddenly dropped vertically (Bisdorf et al. 1999). In posturography experiments, the term “startle-like response” has been coined for muscle responses evoked by the first trial (Hansen et al. 1988), but this claim was not based on formal comparisons with the startle proper that is evoked by auditory or somatosensory startling stimuli. Our findings do not permit a more definitive statement. However, we anticipate that superposition of a startle reaction on the “normal” balance-correcting strategy might produce a flexion response and more instability, as seen in the present study. The kinematics of a pure startle response during stance are not well described, although early reports described a flexion response (Landis and Hunt 1939). Also, startling is part of the neuromuscular response to an unexpected rear-end impact (whiplash-like perturbation) (Blouin et al. 2006, 2007). A formal comparison between the first trial reactions seen here and acoustic startle reactions, both evoked in upright standing subjects, could clarify this. Acoustic startle reactions were probably not elicited during our experiments because sounds generated by the platform never exceeded the threshold level of 60-dB peak equivalent sound pressure level required for such reactions (Blumenthal 1988; Carlsen et al. 2007).

Future perspectives

Conventional posturography uses averaged responses to a series of perturbations to assess the ability to prevent a fall. This design might have obscured the characteristics of actual fall prevention because in daily life perturbations that might cause a fall typically occur under unexpected and unpracticed circumstances. Postural instability is larger in the first trial, especially if the perturbation induces a backward fall. However, using only these first trials poses specific new challenges to the experimental design in preserving a sufficient number of observations to overcome the observed variability. Therefore future studies should investigate whether the impact of the first trial effect is similar in studies using a protocol with different perturbation directions that are administered randomly. In addition, future research should investigate the mechanisms underlying the first trial, especially the presence of startle reactions within the first trial.

Furthermore, studies of patients with focal lesions might show specific alterations during the first trial and this could help to unravel the nature of first trial reactions and clarify the pathophysiology associated with real-life falls. In patients with progressive supranuclear palsy, backward falls are a hallmark of the disease (Bloem et al. 2004; Maher and Lees 1986) and patients with Parkinson’s disease are particularly unstable when perturbed backward (Carpenter et al. 2004; Dimitrova et al. 2004). Studying the first trial reactions in these patient populations may provide additional explanations for the directional sensitivity of the first trial effects. Such studies should also carefully record body accelerations (and not just of the head) to further determine whether there are early differences between first and second trials that could underlie first trial effects. Finally, future experiments could also investigate whether inclusion of first trial reactions adds extra information in discriminating between patients and healthy subjects, compared with the use of habituated series of identical or randomized postural responses.

**ACKNOWLEDGMENTS**

We thank J. Hegeman for help in performing the experiments.

**GRANTS**

This work was supported by Radboud University Nijmegen Medical Centre Research Grant RN0000099 to L. B. Oude Nijhuis, Swiss National Research Foundation Grant 3100A0-104212/1 to J. H. J. Allum and L. B. Oude Nijhuis, and Netherlands Organization for Scientific Research VIDI Research Grant 016.076.352 to B. R. Bloem and VENI Grant 916.56.103 to S. Overeem.

**REFERENCES**


