First Trial Postural Reactions to Unexpected Balance Disturbances: A Comparison With the Acoustic Startle Reaction

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1Department of Neurology, Donders Institute for Brain, Cognition and Behavior, Center for Neuroscience, Radboud University Nijmegen Medical Center, Nijmegen, The Netherlands; 2Department of Otorhinolaryngology, University Hospital, Basel, Switzerland; and 3Servei de Neurologia, Institut Clinic de Neurociències, Institut d’Investigacions Biomèdiques August Pi i Sunyer, Hospital Clínica de Barcelona, Barcelona, Catalonia, Spain

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Oude Nijhuis LB, Allum JHJ, Valls-Solé J, Overeem S, Bloem BR. First trial postural reactions to unexpected balance disturbances: A comparison with the acoustic startle reaction. J Neurophysiol 104: 2704–2712, 2010. First published September 1, 2010; doi:10.1152/jn.01080.2009. Unexpected support-surface movements delivered during stance elicit “first trial” postural reactions, which are larger and cause greater instability compared with habituated responses. The nature of this first trial reaction remains unknown. We hypothesized that first trial postural reactions consist of a generalized startle reaction, with a similar muscle synergy as the acoustic startle response, combined with an automatic postural reaction. Therefore we compared acoustic startle responses to first trial postural reactions. Eight healthy subjects stood on a support surface that unexpectedly rotated backwards 10 times, followed by 10 startling acoustic stimuli, or vice versa. Outcome measures included full body kinematics and surface EMG from muscles involved in startle reactions or postural control. Postural perturbations and startling acoustic stimuli both elicited a clear first trial reaction, as reflected by larger kinematic and EMG responses. The ensuing habituation rate to repeated identical stimuli was comparable for neck and trunk muscles in both conditions, Onset latencies in EMG responses between the two different perturbation trials to stance. Our results show that platform tilting initially induces reactions larger than needed to maintain equilibrium. For neck and trunk muscles, these first trial postural reactions resembled acoustic startle reflexes. First trial postural reactions may be triggered by interaction of afferent volleys formed by somatosensory and vestibular inputs. Acoustic startle reactions may also be partially triggered by vestibular inputs. Similar muscle activation driven by vestibular inputs may be the common element of first trial postural responses and acoustic startle reactions.

INTRODUCTION

Standardized perturbations of a support surface are widely used to study human balance reactions (Allum et al. 2002; Nashner 1976). Actual falls are not supposed to be evoked, and several measures, such as safety harnesses or sidebars, are used to prevent such falls. However, subjects are notably less stable during the untrained and often unpredictable very first response to a sudden support-surface rotation compared with subsequent, identical perturbations (Keshner et al. 1987; Oude Nijhuis et al. 2009). This so-called “first trial reaction” is associated with large EMG responses. During subsequent trials, response amplitudes gradually diminish in a well-known fashion (habituation), and balance control becomes more stable (Nashner 1976a; Macpherson 1994). Being inherently unstable, the first trial response might actually be more relevant for studying the mechanisms underlying falls in daily life. It is customary, however, to draw conclusions about a tendency to fall based on a series of habituated trials, excluding the first trial from analysis.

The qualitatively different automatic postural response (APR) to the unpracticed very first trial has previously been termed “startle-like,” suggesting an analogy between the first trial APR and a startle response (Bloem et al. 1998; Hansen et al. 1988). Several observations support this notion. The classic body movement following startling stimuli is a “crouching” response (Landis and Hunt 1939), and the APR response to the first balance perturbation also shows a marked flexion movement of the upper body (Oude Nijhuis et al. 2009). In addition, startle reflexes, like postural responses, are known to rapidly habituate (Brown et al. 1991; Groves et al. 1974). Siegmund et al. (year) suggested that a startle response may explain the exaggerated first responses in the neck muscles to whiplash-like perturbations of the head in sitting subjects (Siegmund et al. 2008a,b). This notion would fit with the strong involvement of the neck muscles in the startle response (Brown et al. 1991). However, it remains unknown whether startle responses may also explain exaggerated reactions of trunk and leg muscles when unexpected perturbations are applied during stance. It is possible that, besides stretch reflexes and APRs, a startle reflex is also elicited in trunk and leg muscles during the very first perturbation trial to stance. The aim of this study was to examine the hypothesis that first trial reactions to postural perturbations are comparable—wholly or in part—to components of the startle reflex to acoustic stimuli. For this purpose, we analyzed the characteristics of kinematic responses triggered by unexpected postural perturbations and by startling auditory stimuli. We also used EMG to study the first trial reactions to postural perturbations and auditory startling stimuli and compared the observed synergies to the previously described characteristics of the human auditory startle reflex (Brown et al. 1991a; Landis and Hunt 1939; Wilkins et al. 1986). We specifically searched for indications of a startle reflex embedded within the postural first trial reaction. For this purpose, we compared muscle synergies in terms of habituation rate and pattern of onset latencies in EMG responses between the two different stimuli. We recorded activity from several types of muscles: those that are unlikely to be involved in postural control but

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are known to react to startling stimuli; muscles likely to be involved only in postural control; and muscles likely to be involved in both.

**METHODS**

**Subjects**

Eight healthy subjects participated in the study (4 men; mean age, 23 yr; range, 20–29 yr). None had self-reported neurological, balance, or musculoskeletal disorders or had ever before participated in a posturography experiment or in physiology studies of the startle reaction. Participants gave written informed consent before the experiment. 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.

**Procedure**

The experiment consisted of two conditions: the postural perturbation condition (PERTURBATION) and the startling acoustic stimulus condition (STARTLE). In both conditions, subjects stood on a servo-controlled dual-axis rotating platform with their arms hanging by their sides. The ankle joint was aligned with the pitch axis of the platform, and the roll axis passed between the feet. The platform and the techniques used for recording the responses to perturbation have been described in more detail (Allum et al. 2002, 2008; Carpenter et al. 1999). Two assistants were present to lend support in case of an actual fall.

In the PERTURBATION condition, subjects were perturbed by sudden tilts of the support-surface platform (7.5° at 60°/s). A previous study, as well as subsequent pilot experiments, showed that first trial reactions were greatest in backward directions slightly offset from the pitch plane (Oude Nijhuis et al. 2009). Therefore backward-directed tilts of 158° and 203° were used. Directions were defined in a clockwise manner, where the 0° direction is pure forward rotation, 90° denotes right roll, and 180° denotes a pure backward rotation. The roll component in these 158° and 203° stimuli, being oppositely directed, was averaged out when the two population means were computed. In the STARTLE condition, subjects received startling acoustic stimuli (113-dB SPL impulse) through loudspeakers placed at 0.5-m distance on the left and right side of the subjects’ ears. Although startle reactions can be evoked using different modalities, we used acoustic stimuli because these have been well described in standing humans (Brown et al. 1991). In both conditions, subjects received 10 stimuli in a series. The order of both conditions was counterbalanced across subjects, so that four subjects received a series of 158° directed postural stimuli followed by acoustic stimuli, and four subjects received first the acoustic stimuli followed by 203° directed postural stimuli. The interval between trials within a condition was randomly varied between 5 and 15 s.

Before the start of the experiment, subjects were merely informed that they were about to be perturbed by the platform, but the specific nature of the experiment and the number of possible trials was not specified. Thus subjects had no prior knowledge about the startle condition and the characteristics and the direction of the perturbation condition. The experiment started without any preceding trials (which are often administered to familiarize subjects with the experimental conditions), so the very first trial was fully unpracticed.

**Outcome measures**

We recorded kinematic and EMG responses. Participants were instrumented with 18 infrared emitting diodes (IREDs) to collect full body kinematics (Oude Nijhuis et al. 2009) at a rate of 64 Hz using an OPTOTRAK motion analysis system (Northern Digital Canada). Head accelerations were measured directly by four biaxial linear accelerometers (Entran ±5g) mounted on a tight fitting head band. All four biaxial accelerometers measured head vertical linear accelerations, and outputs of pairs of accelerometers were coupled together to measure pitch and roll angular accelerations. Head accelerations were sampled at 1 KHz. Surface EMG signals were recorded from the following muscles on the left side: tibia anterior, soleus, gluteus medius, external oblique, paraspinal at the L1–L2 level, triceps brachii, medial deltoid, and upper trapezius. Furthermore, EMG signals were recorded from the sternocleidomastoid (SCM) and malar muscles, because startle responses are consistently seen in these muscles (Bisdorff et al. 1994; Brown et al. 1991b; Siegmund et al. 2001). EMG recordings were band-pass analog filtered between 60–600 Hz, full wave rectified, and low-pass filtered at 100 Hz with a 3rd-order Paynter filter prior to sampling at 1 KHz. The recordings were initiated 100 ms before stimulus onset and had a sampling duration of 1 s.

**Data analysis**

**KINEMATIC ANALYSES.** Kinematic analyses were performed on 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 and head pitch angular acceleration (Oude Nijhuis et al. 2009). CoM displacements were calculated using the Optotrak data values (Oude Nijhuis et al. 2009; Visser et al. 2008). The total area under the curve (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 between 100 and 800 ms, because between 0 and 100 ms, CoM changes are negligible (Oude Nijhuis et al. 2009). To characterize head pitch angular acceleration, a similar AUC calculation was performed after rectification between 0 and 800 ms.

EMG. EMG onset latencies were calculated across each trial and muscle. We used a semiautomatic computer algorithm that determined when the signal deviated for the first time >2.5 SD from the mean baseline EMG for >50 ms. All onset latencies were visually inspected and manually adjusted when necessary by the same researcher, who was blinded for muscle and condition.

EMG amplitudes were calculated as areas under the EMG traces of the individual trials. Trapezoid integration was performed over an interval of 100 ms, starting at the onset latency of the EMG response. The EMG areas were corrected for baseline EMG activity before stimulus onset.

**Statistical analysis**

For analysis of the kinematic measures (vector CoM and head pitch angular acceleration), we used a linear random effects model (mixed model analysis) with random factor subject and fixed factor trial number to determine the presence of the first trial reaction within both the PERTURBATION and STARTLE conditions. Before analysis, data values were log-transformed to correct for skewed distributions and heteroscedasticity. The results of the analyses were back-transformed into percentages. Paired samples t-tests were used to determine differences between the first trial and the average of trials 6–10, and the same analysis was performed for the difference between the second trial and trials 6–10. To determine the presence of first trial reactions in EMG amplitudes, again a within-condition linear random effects model was used. In addition, between-condition (PERTURBATION and STARTLE) analyses were performed to test for differences in (habituation of) EMG amplitudes for trials 1 and 2 compared with trials 6–10. Two-sided P values and 95% CIs were calculated. EMG onset latencies are presented as means ± SD. The level of significance was set at P < 0.05.
RESULTS

Kinematics of the response to the PERTURBATION and STARTLE condition

Kinematic measures showed a large first trial reaction in the PERTURBATION condition, involving mainly the arms, trunk, and head (Fig. 1, A and B), consistent with earlier work (Oude Nijhuis et al. 2009). In contrast, body movements were considerably smaller in the STARTLE condition, except at the head (Figs. 1, C and 2).

For the PERTURBATION condition, the mixed model analysis showed a significant effect of trial number on the amplitude of the vector CoM ($P < 0.01$). The amplitude was significantly larger during the first trial compared with trials 6–10 (35% larger; 95% CI = 28–42%; $P < 0.01$), whereas the difference between trials 2 and 6–10 was not significant. For the STARTLE condition, no effect of trial number on the amplitude of the vector CoM was found.

Head pitch acceleration amplitude showed a significant effect of trial number for both the PERTURBATION and the STARTLE conditions ($P < 0.01$). In the PERTURBATION condition, head pitch acceleration was 64% (95% CI = 58–69%) larger during the first trial compared with trials 6–10 ($P < 0.01$), whereas the difference between trials 2 and 6–10 was not significant. In the STARTLE condition, head pitch acceleration was 54% (95% CI = 46–63%) larger during the first trial compared with trials 6–10 ($P < 0.01$). The difference between trials 2 and 6–10 was, although smaller (32%; 95% CI = 27–37%), also significant ($P < 0.01$).

Early changes in ankle and knee joint motion, as well as head accelerations, were observed within the first 25 ms after stimulus and before muscle response onsets for the PERTURBATION condition (cf. Figs. 2 and 3). These early changes did not change with trial number. Both ankle angular velocity, head linear vertical acceleration, and head pitch angular acceleration exceeded known proprioceptive and vestibular thresholds within the first 25 ms (see horizontal dotted lines in Fig. 2). Suprathreshold head linear vertical and pitch angular accelerations were also recorded for STARTLE conditions. However, these occurred later (after 40 ms) and with greater amplitudes of pitch acceleration and lower amplitudes of linear vertical acceleration compared with the PERTURBATION responses. In contrast to PERTURBATION responses, the early head pitch accelerations (during the first 50–100 ms) decreased in amplitude over trials (Fig. 2). The pitch angular...
acceleration of the head started with the onset of the sternocleidomastoid muscle response induced by the STARTLE. Weak first trial reactions were observed in the STARTLE condition at the ankle joints after 150 ms (plantar flexion velocity max. 2°/s) and knee joints (extension velocity max. 5°/s). These changes were consistent with the weak lower trunk and leg muscle activity after 100 ms for the STARTLE condition (Fig. 3).

Comparison of first trial EMG responses in PERTURBATION and STARTLE conditions

In the PERTURBATION condition, EMG responses during the first trial were clearly present in all muscles (Table 1; Fig. 3). In the STARTLE condition, first trial EMG responses were clearly seen in sternocleidomastoid and masseter muscles and also in lower trunk muscles (paraspinals, gluteus, external oblique), but less clearly in arm and lower leg muscles (Table 1; Fig. 3).

For both conditions, EMG responses in the sternocleidomastoid, masseter, and paraspinal muscles habituated rapidly between trials 1 and 2, whereas habituation was more gradual over trials 2–10 (linear mixed model analyses; effect of trial number, \( P < 0.01 \) for both conditions). No significant differences were recorded for the habituation between both conditions in these muscles (no significant interaction terms).

We looked in detail at the masseter muscle, because this muscle is not involved in postural control and therefore perhaps more indicative of a startle component. In the PERTURBATION condition, the masseter muscle showed larger activity during the first trial compared with trials 6–10 (31%; 95%
CI = 17–46%; \( P = 0.059 \)), whereas the difference between trials 2 and 6–10 was not significant. In the STARTLE condition, masseter amplitude was 43% (95% CI = 27–59%) larger during the first trial compared with trials 6–10 (\( P < 0.05 \)), whereas the difference between trials 2 and 6–10 was only 27% (95% CI = 15–40%; \( P < 0.01 \)).

Very similar patterns were observed for the other muscles. For example, for the sternocleidomastoid muscle, the amplitude was 139% (95% CI = 109–169%) larger during the first trial in the PERTURBATION condition compared with trials 6–10 (\( P < 0.05 \)), whereas the difference between trials 2 and 6–10 was not significant. In the STARTLE condition, sternocleidomastoid amplitude was 120% (95% CI = 80–160%) larger during the first trial compared with trials 6–10 (\( P < 0.05 \)), whereas the difference between trials 2 and 6–10 was only 58% (95% CI = 40–75%; \( P < 0.05 \)). Comparable findings were obtained for the paraspinal muscles.

The observed differences in response amplitude between the first trial and subsequent trials could not be explained by an increased background muscle activity (recorded over a 100-ms period before stimulus onset). Linear mixed model analysis showed no significant effect of trial number on background activity for both the postural PERTURBATION (\( P = 0.626 \)) and STARTLE conditions (\( P = 0.719 \)).

Between the PERTURBATION and STARTLE conditions, there were differences in the onset latencies of first trial reactions. In the PERTURBATION condition, a short latency reflex response was recorded in the soleus muscle (mean, 42.9 ms; SD, 5.0 ms) and was followed by APRs in the tibialis anterior, gluteus, sternocleidomastoid muscles, and muscles of the trunk and upper arm, as well as a response in the masseter muscle (Table 1; Fig. 3). The latter responses all occurred around 100 ms (see vertical line in Fig. 3, left column; Table 1). In the STARTLE condition, the onset of EMG responses was...
We studied whether the first trial reaction after a sudden postural perturbation contains a startle response synergy similar to that elicited during an acoustic startle reaction. Our results confirmed earlier reports that platform tilting induces a prominent first trial reaction, with muscular and kinematic responses that are larger than the reactions in the subsequent trials (Hansen et al. 1988; Keshner et al. 1987; Oude Nijhuis et al. 2001). The large postural reaction to the first balance perturbation (60°/s) resulted in a more delayed and dispersed input, thus explaining the later onset latencies. We therefore focused on patterns of muscle activations (synergies) rather than absolute onset latencies to address our research questions.

The postural reactions to movements induced by the first trial response also differed between the two test conditions. An example of this is the movement of the head following activation of sternocleidomastoid muscles during the STARTLE condition. We assume that this head movement triggered the later occurring responses in paraspinal muscles (Figs. 2 and 3). Changing in paraspinal activity were also noted when isolated head movements with a comparable amplitude as seen here were imposed directly on standing subjects (Horak et al. 1994, 2001).

The large postural reaction to the first balance perturbation under the PERTURBATION condition may be triggered by a large afferent volley formed by somatosensory inputs, vestibular inputs, or both, as we have indicated in Fig. 2. The abrupt auditory startling stimulus during the STARTLE condition, which is known to rapidly excite vestibular saccular afferents (Young et al. 1977) instead of a more slowly acting vestibular stimulus during the PERTURBATION condition (shown in Fig. 2), may account for the observed differences in onset latencies of sternocleidomastoid and masseter muscles. If, more divergent compared with the PERTURBATION condition (Table 1) and consisted first of the classic early responses in the sternocleidomastoid and masseter muscles, followed by responses in the upper arm and trunk muscles, consistent with previously described patterns of auditory startle reactions (Brown et al. 1991a; Wilkins et al. 1986). Significant differences between the STARTLE and PERTURBATION conditions were observed in onset latencies for the sternocleidomastoid, masseter, and paraspinal muscles (Table 1; Fig. 3).

Responses to the first trial were recorded significantly later in the PERTURBATION condition compared with the STARTLE condition in the sternocleidomastoid (99.7 vs. 59.4 ms; \( P < 0.01 \)) and in the masseter muscle (102.5 vs. 64.2 ms; \( P < 0.01 \)). In contrast, responses of the paraspinal muscles occurred significantly earlier, by 37 ms, in the PERTURBATION condition compared with the STARTLE condition (Fig. 3; Table 1). Responses in tibialis anterior occurred at the same time for both conditions, whereas responses in soleus were rarely seen in the STARTLE condition (Table 1).

### DISCUSSION

Differences in trigger mechanisms. Irrespective of the specific nature and location of the trigger of the first trial reaction, the postural first trial reaction is more time-synchronized across the body compared with the startle response. The significant differences in EMG onset latencies seen for the first trial reactions between the PERTURBATION and the STARTLE condition may well be caused by different trigger mechanisms. Indeed, the intensity, onset, build-up, and time course of the afferent volleys used to stimulate the common startle pathway were very different between both conditions. The auditory startle was generated by a high-intensity, short, and abrupt input that resulted in synchronous input of the response pathway. In contrast, the relatively slow postural perturbation (60°/s) resulted in a more delayed and dispersed input, thus explaining the later onset latencies. We therefore focused on patterns of muscle activations (synergies) rather than absolute onset latencies to address our research questions.

### Table 1. Onset latencies and probability of occurrence of EMG bursts in the first trials of the PERTURBATION and the STARTLE conditions

<table>
<thead>
<tr>
<th>Onset Latencies</th>
<th>PERTURBATION Trial 1</th>
<th>STARTLE Trial 1</th>
<th>PERTURBATION Trial 1</th>
<th>STARTLE Trial 1</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sternocleidomastoid</td>
<td>93.6 (26.5)</td>
<td>59.4 (8.6)*</td>
<td>100%</td>
<td>75%</td>
</tr>
<tr>
<td>Masseter</td>
<td>94.8 (10.5)</td>
<td>64.2 (15.2)†</td>
<td>88%</td>
<td>100%</td>
</tr>
<tr>
<td>Trapezius</td>
<td>98.0 (19.7)</td>
<td>85.1 (38.2)</td>
<td>100%</td>
<td>36%</td>
</tr>
<tr>
<td>Medial deltoid</td>
<td>99.2 (26.1)</td>
<td>97.2</td>
<td>100%</td>
<td>13%</td>
</tr>
<tr>
<td>Triceps brachii</td>
<td>93.41 (14.6)</td>
<td>117.2 (40.7)</td>
<td>88%</td>
<td>50%</td>
</tr>
<tr>
<td>External oblique</td>
<td>95.1 (16.5)</td>
<td>106.9 (32.7)</td>
<td>100%</td>
<td>88%</td>
</tr>
<tr>
<td>Paraspinal</td>
<td>112.4 (21.0)</td>
<td>148.9 (15.0)*</td>
<td>100%</td>
<td>75%</td>
</tr>
<tr>
<td>Gluteus</td>
<td>94.5 (16.6)</td>
<td>129.7 (33.4)</td>
<td>100%</td>
<td>88%</td>
</tr>
<tr>
<td>Soleus</td>
<td>46.0 (3.6)‡</td>
<td>130 n.c.</td>
<td>88%</td>
<td>25%</td>
</tr>
<tr>
<td>Tibialis anterior</td>
<td>120.0 (19.0)</td>
<td>112.3 (22.6)</td>
<td>100%</td>
<td>63%</td>
</tr>
</tbody>
</table>

Mean (SD) onset-latencies are shown in ms. **P < 0.01; *P < 0.05. Values are mean (SD) onset latencies (ms). *P < 0.05. †P < 0.01. ‡Soleus stretch reflex and not APR onset latency; therefore the comparison with the response to the first startle was classified as not comparable (n.c.). Probability of occurrence of EMG bursts is shown in percentages. APR, automatic postural response.

### Triggering of the first trial response

The large postural reaction to the first balance perturbation under the PERTURBATION condition may be triggered by a large afferent volley formed by somatosensory inputs, vestibular inputs, or both, as we have indicated in Fig. 2. The abrupt auditory startling stimulus during the STARTLE condition, which is known to rapidly excite vestibular saccular afferents (Young et al. 1977) instead of a more slowly acting vestibular stimulus during the PERTURBATION condition (shown in Fig. 2), may account for the observed differences in onset latencies of sternocleidomastoid and masseter muscles. If,
however, responses for the vestibular component of the STARTLE and PERTURBATION are relayed through the same nervous structures and if processing times within these structures dominate, the use of an abrupt acoustic stimulus may by itself not have made a difference concerning latencies. This might account for the lack of differences in response latencies between the response to a free fall and startling acoustic stimuli (Bisdorff et al. 1994).

INTERACTIONS BETWEEN TRIGGER SIGNALS. The presence of two or more triggering signals with complex interactions may also account for the observed differences in onset latencies between the auditory startle reaction and the postural first trial reaction. Here the question arises whether the temporal separation of vestibular and ankle proprioceptive trigger signals may contribute to the first trial effect, particularly for pitch plane balance perturbations (Allum et al. 2008). Acoustic startle reactions per se were probably not contributing to the reaction in the PERTURBATION condition in our study, because sounds generated by the platform never exceeded the threshold level of 60 dB peak equivalent SPL required for such reactions (Blumenthal 1988; Carlsen et al. 2007).

Because of the known effect of sound on otolith stimulation, particularly on the sacculus (Colebatch et al. 1994; Young et al. 1977), it may be necessary, as described above, to consider an otolithic and acoustic stimulus as being part of the same acoustic startle stimulus. Acoustic stimuli generate vestibular-evoked myogenic potentials (VEMPS) at ~40 ms when air-conducted 120 peak equivalent SPL click auditory stimuli are used (Colebatch 2001; Colebatch and Halmagyi 1992; Colebatch et al. 1994; Welgampola and Colebatch 2005). This stimulus level is comparable in hearing level to the 113-dB impulse SPL longer sound pulse we used. Louder clicks (by 25 dB) evoke shorter latency (15 ms) VEMPS (Colebatch et al. 1994). Thus we assume that, in the STARTLE condition, the auditory and associated saccular responses are evoked near the brain stem at the same time. This could be another reason why onset latencies in the STARTLE condition were shorter in the acoustic startle reflex. A concurrent masseter and sternocleidomastoid response is observed under both conditions, with comparable habituation rates during subsequent trials. In contrast, latency differences in the PERTURBATION condition in our study, because sounds generated by the platform never exceeded the threshold level of 60 dB peak equivalent SPL required for such reactions (Blumenthal 1988; Carlsen et al. 2007).

Consequences of startling-like influences on the manifestation of the postural response

Irrespective of the specific nature and location of the trigger mechanisms of the first trial reaction, which may not be different from those of subsequent reactions, we found indications that the first trial reaction to a platform rotation is a less differentiated reaction in terms of amplitude compared with the fully habituated response to the same platform rotation (see also Keshner et al. 1987; Oude Nijhuis et al. 2009). Following early stretch reflexes in the lower leg muscles evoked by the platform movement, a second response was elicited around 100 ms in all muscles during the first trial. This muscle activity comprises larger co-contraction of agonist and antagonist muscles, which would lead to increased stiffness (Keshner et al. 1987). This “stiffening up” may be one of the causes of the decreased stability and greater fall frequency during the first trial compared with subsequent perturbations that we found in a previous study (Oude Nijhuis et al. 2009).

The influence of startling reflexes with increased co-contraction within the first trial reaction on postural stability seems to be mainly negative. A clinical example of the detrimental effect of startling reflexes is shown by clinical syndromes such as hyperekplexia. In this disorder, startling can cause stiffness in these subjects, resulting in falls without subjects being able to break their fall (Bakker et al. 2006; Tijssen et al. 2002). The question arises whether startling reflexes could be beneficial in quickly inducing a speedy balance correction when suddenly losing balance. Startle reflexes are known to accelerate movements in simple reaction time tasks (Carlsen et al. 2003; Siegmund et al. 2001; Valls-Sole et al. 1999), but also in choice reaction time tasks (Oude Nijhuis et al. 2007), e.g., in an obstacle avoidance task when walking (Queralt et al. 2008), when subjects are not fully prepared. However, in our postural first trials, no significant decrease in onset latencies was observed.

Conclusion

One component of sternocleidomastoid and masseter muscle responses within the first trial reaction to perturbations in standing subjects is similar to the synergy evoked during a pure acoustic startle reflex. A concurrent masseter and sternocleidomastoid response is observed under both conditions, with comparable habituation rates during subsequent trials. In con-
trast, onset latencies differed for PERTURBATION and STARTLE muscle responses. The triggering of automatic postural responses by different sensory modalities, leading to startle-like components in the form of increased co-contraction across muscles and the presence of a masseter response within the first trial, makes it difficult to clearly disentangle startle reactions per se from reactions to perturbations. A longer participating proprioceptive afferent pathway for triggering signals from the feet, legs, or trunk to the brain stem may partially explain the differences in onset latencies at the head between reactions to perturbations and startling acoustic stimuli rather than the presence of vestibular excitation for both stimuli. Our approach using an acoustic stimulus to evoke startle reflexes provided limited insights into the underlying trigger mechanism for the increased co-contraction of first trial reactions to stance perturbations. Another approach may be to use alternative triggering modalities, such as tactile startling stimuli. For example, future experiments may use (electro-)tactile stimuli at the ankle or hips of different intensities combined with recordings of vertical head accelerations to further disentangle the influence of the various trigger modalities on the startle component within balance reactions. Such experiments may also explain the differences in latencies between the STARTLE condition and postural perturbations for trunk muscles during first trial reactions and indicate whether the startle component of the PERTURBATION condition may be the output of a different CNS structure than responsible for the acoustic startle response.

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DISCLOSURES

No conflicts of interest, financial or otherwise, are declared by the authors.

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