First trial and StartReact effects induced by balance perturbations to upright stance

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Submitted 30 August 2012; accepted in final form 13 August 2013

Campbell AD, Squair JW, Chua R, Inglis JT, Carpenter MG. First trial and StartReact effects induced by balance perturbations to upright stance. J Neurophysiol 110: 2236–2245, 2013. First published August 14, 2013; doi:10.1152/jn.00766.2012.—Postural responses (PR) to a balance perturbation differ between the first and subsequent perturbations. One explanation for this first trial effect is that perturbations act as startling stimuli that initiate a generalized startle response (GSR) as well as the PR. Startling stimuli, such as startling acoustic stimuli (SAS), are known to elicit GSRs, as well as a StartReact effect, in which prepared movements are initiated earlier by a startling stimulus. In this study, a StartReact effect paradigm was used to determine if balance perturbations can also act as startling stimuli. Subjects completed two blocks of simple reaction time trials involving wrist extension to a visual imperative stimulus (IS). Each block included 15 CONTROL trials that involved a warning cue and subsequent IS, followed by 10 repeated TEST trials, where either a SAS (TESTSAS) or a toes-up support-surface rotation (TESTPERT) was presented coincident with the IS. StartReact effects were observed during the first trial in both TESTSAS and TESTPERT conditions as evidenced by significantly earlier wrist movement and muscle onsets compared with CONTROL. Likewise, StartReact effects were observed in all repeated TESTSAS and TESTPERT trials. In contrast, GSRs in sternocleidomastoid and PRs were large in the first trial, but significantly attenuated over repeated presentation of the TESTPERT trials. Results suggest that balance perturbations can act as startling stimuli. Thus first trial effects are likely PRs which are superimposed with a GSR that is initially large, but habituates over time with repeated exposure to the startling influence of the balance perturbation.

first trial effect; postural responses; startle; StartReact effect; reaction time

IN A REPEATED SEQUENCE of trials involving discrete balance perturbations, amplitudes of postural responses (PRs) evoked by the first trial are significantly larger compared with subsequent trials involving the same postural stimulus (Bloom et al. 1998a; Chong et al. 1999; Hansen et al. 1988; Keshner et al. 1987; Oude Nijhuis et al. 2009, 2010). This observation has come to be known as the first trial effect (FTE) (Allum et al. 2011). Although it is common to observe FTEs following many types of balance perturbations to both sitting and standing postures (Allum et al. 2011; Bloom et al. 2006, 2007; Oude Nijhuis et al. 2009, 2010; Siegmund et al. 2008; Tang et al. 2012), the underlying mechanisms that contribute to the FTEs remain largely unknown.

One possible explanation for FTEs is that the first exposure to an unpredictable balance perturbation elicits a generalized startle response (GSR) that is superimposed on the basic PR (Bloom et al. 2006; Nanhoe-Mahabier et al. 2012; Siegmund et al. 2008). GSRs are stereotyped patterns of bilateral flexion reactions that can be elicited by intense stimulation to visual, auditory, or somatosensory systems (Bradley et al. 1990; Carlsen et al. 2011; Scott et al. 1999; Yeomans and Frankland 1995). Arguments in support of a GSR contribution to FTEs draw on similarities observed between GSRs and FTEs. For example, both GSRs and PRs are known to habituate rapidly after the first exposure to a series of repeated stimuli (Oude Nijhuis et al. 2010). Secondly, excitation of muscles normally associated with GSRs [i.e., masseter and sternocleidomastoid (SCM)] is frequently observed during the first response to a balance perturbation, but to a much lesser degree in subsequent perturbations (Oude Nijhuis et al. 2010). Finally, coherence between muscle responses in frequency bandwidths typically associated with GSRs has been observed in the first response to a seated perturbation, but not during subsequent balance responses (Bloom et al. 2006).

It is notable that most of the evidence in support of a GSR contribution to FTEs is indirect and involves a number of significant limitations and assumptions. For example, SCM muscle activity onsets evoked by known startling stimuli [i.e., startling acoustic stimuli (SAS)] differ significantly from those induced by balance perturbations (Oude Nijhuis et al. 2010). Thus it is plausible that perturbation-induced SCM activity may represent a response that is characteristically distinct from GSRs. Secondly, determining whether GSRs are evoked by balance perturbations is complicated by the fact that they develop in similar muscles and within similar time frames to startling stimuli as PRs induced by balance perturbations. For example, GSRs evoked by known startling stimuli are oftentimes observed in muscles, such as tibialis anterior (TA) and soleus (SOL), within 100 ms of startle stimulus onset (Brown et al. 1991), which is similar to PRs evoked in these muscles to support-surface balance perturbations (Nashner and Cordo 1981). Consequently, GSRs induced by balance perturbations may superimpose onto PRs in certain muscles and potentially limit the ability to analyze, or to assess the presence of, one response independent of the other. Thirdly, direct support of GSR contributions to FTEs cannot be garnered by previous reports of muscle activity coherence, as this statistical technique relies upon correlational measures and thus may not be used to imply causation (Siegmund et al. 2008). Finally, although it has been assumed that balance perturbations can stimulate sensory systems with sufficient intensity to elicit a GSR (Bisdomff et al. 1994; Commissaris et al. 2002), it has yet...
to be confirmed empirically whether balance perturbations can, in fact, act as a startling stimulus.

One approach to investigate whether a stimulus is capable of inducing GSRs is to determine if it can also induce the StartReact effect. The StartReact effect is characterized by the involuntary release of a prepared motor response that occurs when a startle stimulus is paired with a “go” stimulus [i.e., an imperative stimulus (IS)] to initiate movement (see Carlsen et al. 2011 for review). Theoretically, any stimulus that can induce a GSR can also induce the StartReact effect; however, for its ease of implementation, short-duration SAS of ~120 dB have been considered the standard for inducing both (Carlsen et al. 2011). SAS have been used to induce the StartReact effect in various motor behaviors that range from simple wrist extension (Valls-Solé et al. 1999) to anticipatory postural adjustments that precede step-initiation (MacKinnon et al. 2007) and conditioned PRs (Campbell et al. 2012). StartReact effects are focal and task-specific and thus are highly distinguishable from bilaterally symmetric, nonspecific flexor muscle activity characterizing the GSR (Landis et al. 1939). StartReact effects also persist even when GSRs are attenuated by prepulse inhibition (Valls-Solé et al. 2005), thus suggesting that they could be a relatively more robust indicator of the presence of startle stimuli than GSRs themselves.

Therefore, the main purpose of this experiment was to utilize a StartReact effect paradigm as a probe to determine if balance perturbations can act as startle stimuli. Assuming that balance perturbations can act as startling stimuli, we hypothesized that balance perturbations would induce StartReact effects in a similar manner as SAS, when they are paired with a voluntary wrist extension (Valls-Solé et al. 1999) to anticipatory postural adjustments that precede step-initiation (MacKinnon et al. 2007) and conditioned PRs (Campbell et al. 2012). StartReact effects are focal and task-specific and thus are highly distinguishable from bilaterally symmetric, nonspecific flexor muscle activity characterizing the GSR (Landis et al. 1939). StartReact effects also persist even when GSRs are attenuated by prepulse inhibition (Valls-Solé et al. 2005), thus suggesting that they could be a relatively more robust indicator of the presence of startle stimuli than GSRs themselves.

Experimental Procedures

Quiet stance. With a stance width equal to 100% of their measured foot length, subjects first stood quietly on a stage mounted to a rotating platform for 60 s with their arms relaxed at their sides while focusing on an eye-level target located ~2 m away. During this time, a mean ± 2 SD of resting wrist position was calculated and used as a threshold for initiating subsequent reaction time trials. After 60 s, two stimuli were presented spaced ~15 s apart (with order counterbalanced across participants). One stimulus was a red LED (200-ms duration) located at the center of the visual target that would later function as an IS to initiate the reaction time task (see below). The other stimulus was an auditory cue (~80 dB, two 50-mS pulses separated by 50 ms) that would later be used to warn subjects of an upcoming trial (WARN). The presentation of these stimuli during quiet stance served to verify that IS and WARN cues were nonstartling.

Reaction time protocol. Subjects had five practice trials with the reaction time task. During each reaction time trial, subjects were first presented with the auditory warning cue (WARN) followed by the visual IS after a random 1.5- to 3.5-s interval. After detecting the IS, subjects were instructed to fully extend the wrist as quickly as possible and then hold the extended position for ~0.5 s before returning back to resting position. Wrist position was monitored in real-time, and subjects were coached back to resting positions, if necessary, to within resting thresholds calculated during the quiet standing trial. At the beginning of practice trials, subjects were told to “react as quickly as possible,” which was reiterated at predefined five-trial intervals throughout the experimental session.

After completing the practice trials, subjects performed two experimental blocks [SAS and perturbations (PERT)] that were each counterbalanced across participants. In each block, subjects first performed 15 reaction time trials (CONTROL) (Fig. 1). After CONTROL trials, subjects then performed a series of 10 TEST trials. In the SAS block, TEST trials involved the WARN cue, followed 1.5–3.5 s by the IS.
presented simultaneously with a SAS (i.e., TESTSAS) (Fig. 1). For the PERT block, TEST trials (i.e., TESTPERT) involved the WARN cue followed 1.5–3.5 s by the IS presented simultaneously with a toes-up support-surface rotation (12°, 120°/s, 100-ms duration) (Fig. 1). Subjects were entirely unaware of when TEST trials were to begin and how many would be in each block. Subjects were also informed that there would be instances interspersed throughout each block where the WARN cue would not be followed by the IS (CATCH trials) (Fig. 1), and thus they should not react. During each block, a CATCH trial was pseudorandomly presented once for every five CONTROL and TEST trials. Thus 3 CATCH trials were presented during the sequence of 15 CONTROL trials, and a further 2 CATCH trials were presented during the sequence of 10 TEST trials. Each CONTROL and TEST trial was separated by a random intertrial interval lasting 10–20 s. Independent of performance, subjects were reminded to react as quickly as possible to the IS at regular five-trial intervals.

At the end of each experimental block, subjects were guided off the platform and were given a 5-min rest period while seated. After which time, they stepped onto the platform to receive a sequence of five Perturbation-Only trials, spaced 10–15 s apart, that involved only the support-surface rotation, which was not accompanied by the IS. During Perturbation-Only trials, subjects were told that the IS would never be illuminated, and thus they were no longer to complete the reaction time task.

Dependent measures. Wrist kinematics. In all CONTROL and TEST trials, reaction times for wrist extension were calculated as the latencies between onsets of the IS and wrist extension. Mean and 2 SD measures of resting wrist positions were determined from the goniometer for 500 ms prior to the onset of the IS within each trial. Wrist extension onsets were determined in each reaction time trial as the time when goniometer displacements exceeded mean +2 SD of resting amplitudes and remained suprathreshold for a minimum of 200 ms. From onset, peak wrist displacements were determined as the displacement value achieved at full extension when movement had ceased (i.e., achieved zero velocity).

Wrist EMG. Onsets of EMG responses for ECR were calculated during each CONTROL and TEST trial. Thresholds were calculated as the mean +2 SD of 500 ms of background EMG levels prior to the start of each trial. Onsets were determined as the first time after IS that processed EMG signals surpassed and remained suprathreshold for at least 30 ms while at no time dropping below threshold for >3 ms (Carpenter et al. 2008).

SCM EMG. Onsets of SCM muscle activity were determined using the same algorithm applied to ECR (see above) and were similarly referenced to IS onset in each trial. Amplitudes were determined by subtracting 100-ms integrals of pre-onset EMG signals from 100 ms of post-onset EMG signals. This duration of analyses of response amplitudes was used because it is a period where sensory feedback has limited influence over triggered reactions (Wadman et al. 1979).

PRs. Both EMG and kinematic data quantified PRs evoked by support-surface rotations. For muscle activity related to PRs, absolute onsets and amplitudes were determined using the same algorithms applied to other records of EMG activity (see above). For the kinematic dataset, onsets of segment displacements were determined as the latency between perturbation onset and the time they surpassed a mean ±2 SD threshold of resting positions calculated 500 ms immediately before the onset of the IS and remained beyond threshold for >200 ms. Peak displacements were also calculated as the greatest change achieved within 800 ms of perturbation onset.

Statistical Analyses

Reaction time. Wrist kinematic data for one subject had to be removed due to a technical issue. All dependent measures for wrist kinematics as well as for EMG of ECR for each block (SAS/PERT) were averaged across CONTROL trials and compared with the 1st and 10th TEST trials of each block using a 2 × 3 [Block (SAS, PERT) × Trial (CONTROL, 1st TEST, 10th TEST)] repeated-measures ANOVA. Because SCM activity was neither expected nor observed during CONTROL trials, measures of its EMG activity were compared using a 2 × 2 [Block (SAS, PERT) × Trial (1st TEST, 10th TEST)] repeated-measures ANOVA.

To examine FTEs on PRs, preplanned t-tests were conducted to compare EMG and kinematic responses observed between the 1st and 10th TESTPERT trials. P values were set at 0.05 for all statistical tests of main effects and interactions. For post hoc analyses, actual P values were corrected for multiple comparisons using the Bonferroni method.
interactions were significant for absolute onset latency measures of wrist extension or ECR (\(P > 0.05\)) (see Table 1).

Neither main effects of Block and Trial, nor Block x Trial interactions were significant for peak wrist displacements (\(P > 0.05\)) (Table 1), suggesting that the task of producing maximal wrist extension was consistently reproduced throughout the experiment.

FTEs

The within-subject pattern of SCM responses over repeated TEST trials varied across subjects. However, there was a predominant trend across subjects of an increased prevalence of SCM responses in the 1st TEST trials compared with the 10th TEST trial (Fig. 3A). In the 1st TEST trials of both SAS and PERT blocks, SCM responses were detected in 11 of 12 and 12 of 12 subjects, respectively (Fig. 3A). However, by the 10th TEST trials, SCM responses were detected in only 5 of 12 subjects in SAS blocks and 8 of 12 subjects in PERT blocks (Fig. 3A).

The decrease in the prevalence of SCM responses was accompanied by a significant decrease in the amplitude of SCM responses after the 1st TEST trial (Fig. 3, B and C). For subjects whose SCM activity exceeded the detection threshold in both the 1st and 10th trials of SAS and PERT blocks, the amplitudes were significantly reduced in the 10th compared with 1st trial [main effect of Trial: \(F_{(1,4)} = 53.44, P = 0.002\)], whereas onset latencies were unaffected by Trial (\(P > 0.05\)). Significant main effects of Block were observed for onset

![Graphs showing wrist angular displacements and extensor carpi radialis (ECR) electromyographic (EMG) responses during a single CONTROL trial as well as in the 1st and 10th TEST trials in SAS and PERT blocks for a representative subject. Time “zero” represents the onset of the IS in each trial.](http://jn.physiology.org/)

Table 1. Dependent measures and results from statistical tests

<table>
<thead>
<tr>
<th>Wrist kinematics</th>
<th>SAS Block</th>
<th>PERT Block</th>
<th>Statistics</th>
</tr>
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<tbody>
<tr>
<td></td>
<td>CONTROL</td>
<td>First TEST</td>
<td>Last TEST</td>
</tr>
<tr>
<td></td>
<td>CONTROL</td>
<td>First TEST</td>
<td>Last TEST</td>
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<tr>
<td>Onset, ms</td>
<td>238 ± 7</td>
<td>165 ± 13</td>
<td>146 ± 11</td>
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<tr>
<td>Peak displacement, °</td>
<td>60 ± 4</td>
<td>56 ± 5</td>
<td>60 ± 3</td>
</tr>
<tr>
<td>ECR EMG</td>
<td></td>
<td></td>
<td></td>
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<tr>
<td>Onset, ms</td>
<td>193 ± 10</td>
<td>95 ± 14</td>
<td>102 ± 10</td>
</tr>
<tr>
<td>SCM EMG</td>
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<tr>
<td>Onset, ms</td>
<td>N/A</td>
<td>58 ± 11</td>
<td>60 ± 7</td>
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<td>Area, (\mu V\cdot s)</td>
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<td>3.4 ± 0.7</td>
<td>0.5 ± 0.1</td>
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</table>

Each numerical cell value represents mean ±1 SE. SAS, startling acoustic stimuli; PERT, perturbations; ECR, extensor carpi radialis; SCM, sternocleidomastoid; ns, nonsignificant result for corresponding measure and level of ANOVA.

\(J\ Neurophysiol\) • doi:10.1152/jn.00766.2012 • www.jn.org
latencies of SCM [F(1,4) = 24.26, P = 0.008] where earlier responses were observed in SAS compared with PERT blocks (Fig. 3, B and C, and Table 1). There was no significant main effect of Block for SCM amplitude, nor Trial × Block interactions for either the amplitudes or onsets of SCM muscle activity (P > 0.05).

Support-surface rotations presented during TESTPERT trials evoked characteristic EMG activity in various postural muscles (Fig. 4A). Following the onset of balance perturbation, stretch reflexes observed in SOL occurred earlier than PRs in TA, RF, and EO (Fig. 4A). Repeated experience with balance perturbations during TESTPERT trials did not significantly influence the onset latencies of any of the postural muscle activity examined (P > 0.05) (Fig. 4B). However, EMG amplitudes significantly decreased from the 1st to the 10th TESTPERT trial in all muscles [SOL: t(11) = 3.41, P = 0.006; RF: t(11) = 2.51, P = 0.029; EO: t(11) = 2.38, P = 0.037] except TA (P > 0.05) (Fig. 4B).

Toes-up support-surface tilts caused substantial angular displacements of the upper arm, trunk, and shank segments (Fig. 5). Onsets of shank, trunk, or upper-arm angular displacements were not significantly influenced by Trial (P > 0.05) (Fig. 5). However, peak angular displacements of the trunk [t(11) = 3.65, P = 0.004] and upper-arm [t(11) = 4.24, P = 0.002] segments were significantly attenuated in the 10th TESTPERT trial compared with the 1st TESTPERT trial. Peak shank angular displacements were not significantly affected by Trial (P > 0.05) (Fig. 5).

**DISCUSSION**

The main purpose of this experiment was to utilize a StartReact paradigm as a probe to determine if balance perturbations, like SAS, constitute startling stimuli. We hypothesized 1) that balance perturbations would induce StartReact effects in all reaction time trials in a similar manner as SAS; and 2) that evoked responses in SCM (a marker for GSR) and postural muscles would be greatest in amplitude in the first trial and would decrease in subsequent trials.

**Balance Perturbations as Startling Stimuli**

Balance perturbations produce whole body displacements that induce signals in the vestibular, visual, and somatosensory systems (Horak 2006; Peterka 2002). Each of these sensory systems is capable of triggering a GSR independently. For example, GSRs have been elicited by stimulation of the vestibular system via unexpected vertical drops of the body (Bisdorf et al. 1995; Gruner 1989), high-intensity visual stimuli (Bradley et al. 1990), as well as tactile (Gokin and Karpukhina 1985) and sural nerve stimulation (Delwaide and...
Crenna 1984). The convergence of multiple sources of sensory feedback, as would be expected in a balance perturbation, is known to further facilitate the triggering of GSRs (Yeomans et al. 2002), likely via multimodal afferent connections with the caudal pontine reticular formation. More importantly, the multisensory signals generated by a balance perturbation could potentially reach the reticular formation within short enough latencies to trigger a GSR, which can appear as early as 30–50 ms poststartle in muscles of the face and neck (Brown et al. 1991). For example, vertical head accelerations are recorded as early as 20 ms following a support-surface perturbation (Allum et al. 2008), while cortical evoked potentials in response to stretch of the triceps surae muscle (Davis et al. 2011) or visual stimuli (Di Russo et al. 2001) are observed with average latencies of 35 ms and 55 ms, respectively.

Through the novel application of a StartReact paradigm, the current study has provided the first empirical evidence in support of the hypothesis that balance perturbations can act as startling stimuli. First trial exposures to balance perturbations (i.e., TESTPERT) induced a StartReact effect, whereby earlier onsets of wrist extension and ECR activity were observed compared with CONTROL trials. The ability of a balance perturbation to elicit a StartReact effect was not limited to the first trial, as earlier onsets of wrist extension and ECR activity were still observed at the end of repeated TESTPERT trials compared with CONTROL trials. The StartReact effects elicited by the TESTPERT conditions during the first, and subsequent trials, were similar to the StartReact effects elicited by the SAS, suggesting that balance perturbations, like SAS, can act as a startling stimulus, as previously proposed (Bisdorff et al. 1994; Commissaris et al. 2002).

What Are FTEs?

As hypothesized, there was an observed change in the PRs evoked by repeated balance perturbations. Specifically, the amplitudes of EO, RF, and SOL were significantly decreased in the 10th compared with the 1st TESTPERT trial, while no changes to response onsets were observed for any of the muscles tested. These changes with repeated perturbations are consistent with those observed in prior studies of PR habituation where amplitudes of both EMG in postural muscles and kinematic displacements of body segments significantly decreased after the first balance perturbation (Oude Nijhuis et al. 2008).

Fig. 4. A: external oblique (EO), rectus femoris (RF), soleus (SOL), and tibialis anterior (TA) EMG responses evoked during the 1st (black) and 10th (gray) TESTPERT trials for a representative subject. Vertical black dashed line denotes the simultaneous onset of the balance perturbation and the IS within each trial. B: plots depicting the average (±1 SE) onset and amplitude of EO, RF, SOL, and TA during all TESTPERT trials. Note, only the 1st and 10th trials (i.e., data colored in black) were compared statistically. Black connector lines indicate significant differences between conditions.
Kinematics

Onset Latency

Amplitude

Upper Arm

Trunk

Shank

PERT

IS

Time (s)

Displacement (°)

Displacement (°)

Displacement (°)

Time (s)

Displacement (°)

Displacement (°)

Fig. 5. Left: upper arm, trunk, and shank absolute angular displacements evoked by the 1st (solid black) and 10th (dashed black) TESTPERT trials for a representative subject. Vertical dashed line denotes the simultaneous onset of the balance perturbation and IS. Right: plots depicting the average (±1 SE) onset and amplitude of upper arm, trunk, and shank absolute angular displacements during all TESTPERT trials. Note, only the 1st and 10th trials (i.e., data colored in black) were compared statistically. Black connector lines indicate significant differences between trials.

2009, 2010), without a reported change in their onset latency (Keshner et al. 1987). The changes in PRs were paralleled by changes to activity in SCM, which is commonly recognized as a primary indicator of GSRs (Carlsen et al. 2011). SCM responses were large and frequent during the 1st TESTPERT trial, with only minimal SCM activity observed by the 10th TESTPERT trial. The decrease in GSR response in SCM activity over repeated perturbations cannot be explained by a decrease in the intensity of the startle stimulus itself, as all balance perturbations, including the 1st and 10th, were shown to be capable of eliciting the same StartReact effect and thus represent the same intensity of startling stimuli. Therefore, the observations of decreased SCM responses over repeated balance perturbations more likely reflects a habituation of a GSR response over repeated exposure to the same startle stimulus. These observations support the hypothesis that FTEs reflect PRs which are superimposed with a GSR that is initially large, but habituates with repeated exposure to the startling influence of the balance perturbation over time (Blouin et al. 2006). Interestingly, the proposed relationship between the habituation of GSR and PRs to repeated balance perturbations is also supported by clinical observations, with Parkinsonian patients displaying delayed habituation of PRs to postural perturbations (Nanhoe-Mahabier et al. 2012) as well as slower habituation of GSRs to repeated SAS stimuli (Nieuwenhuijzen et al. 2006).

Limitations

Although SCM activity is assumed to be a marker of a GSR, there is also the possibility that early SCM activity observed during TESTPERT could reflect a PR to the early head accelerations induced by the postural perturbation (Blouin et al. 2006; Oude Nijhuis et al. 2009; Siegmund et al. 2008; Tang et al. 2012). While we recognize this as a potential limitation, there is ample evidence to suggest that SCM activity observed during toes-up rotations are more likely associated with the GSR as opposed to a PR. For example, in the current study, there were similar amplitudes of SCM activity evoked by balance perturbations and SAS trials during both the first and subsequent trials. Furthermore, during support-surface rotations, the timing and amplitude of SCM activity have been shown to be independent of direction of the perturbation during first trial (Tang et al. 2012) and habituated responses (Oude Nijhuis et al. 2009; Tang et al. 2012), despite differences in induced head accelerations (Carpenter et al. 1999), and direc-
tional dependent changes in other postural muscles (Carpenter et al. 1999, 2004, 2008). Similarly, Oude Nijhuis et al. (2010) observed SCM activity during support-surface rotations that was more similar in latency and pattern to other nonpostural muscles (i.e., masseter), compared with muscles engaged in postural control. Thus we would argue that early SCM activity observed during TESTPERT conditions is still a valid indicator of GSRs, in the current application.

Another limitation of the study is that the voluntary wrist extension task used to assess the StartReact effect could also have been confounded by a PR elicited in the wrist by the balance perturbation. As shown in Fig. 6, the wrist responses to Perturbation-Only trials were minimal compared with the amplitude of wrist movement elicited during TESTPERT trials. However, we recognize that this comparison may be limited by the potential order effect caused by having the Perturbation-Only trials always performed after the SAS and PERT blocks. Furthermore, the relatively late onset of wrist movements during Perturbation-Only trials, suggests that any potential PR of the wrist would not be able to explain the very early initiation of the wrist response (StartReact effect) observed during TESTPERT trials (Fig. 6). As an alternative task to assess StartReact effects, future studies could incorporate other voluntary tasks that are completely independent from the PR, including vocalizations (Chiu et al. 2011) or displacements of the tongue (Regnaux et al. 2005) and counterbalance the Perturbation-Only trials with others within the methodological design.

Finally, although StartReact effects were observed in TEST conditions, they were delayed compared with those evoked by SAS in other experiments. For example, ECR onsets were observed by Valls-Solé et al. (1999) and Carlsen et al. (2007) within 80 ms of SAS onsets, whereas, in our study, ECR onsets occurred on average ~100 ms after startle stimuli onset [mean range of 95 ms and 110 ms (Table 1)]. We believe these differing methodological procedures and analysis techniques may help explain differences between studies. Firstly, our experiment involved a simple reaction time task that was completed while subjects were standing upright, whereas other studies required subjects to remain seated during testing. The attentional resources required of bipedal stance may have been shifted away from the reaction time task and directed toward the task of maintaining balance (Woollacott and Shumway-Cook 2002), which could have a delaying effect on reaction time (Mangun and Buck 1998). Secondly, our experimental analyses included all reaction time data, regardless of the presence of SCM. Considering that Carlsen et al. (2007) observed significantly earlier reaction times during a StartReact paradigm in trials where SCM activity was observed compared with trials where startle stimuli were delivered but did not induce SCM activity, we recognize that the overall response latencies we observed in TEST trials may have been slightly increased, on average, by analyzing all StartReact trials, independent of SCM activity. However, the actual effect, if any, appears minimal in our data, as there were no significant effects of Trial observed on the StartReact latencies, despite a higher prevalence, and amplitude, of SCM responses in the 1st compared with the 10th TEST trials.

Implications

Our findings highlight the potential for GSRs to influence FTEs and the subsequent time course of PR habituation to repeated perturbation. The latter underscores a need to account for this potentially confounding factor when comparing PRs across groups that may respond, and habituate, differently to repeated startle stimuli, such as patients with Parkinson’s disease (Nieuwenhuijzen et al 2006), Steele-Richardson-Olszewski syndrome (Vidalheth et al. 1992), hyperekplexia (Brown et al. 1991), progressive supranuclear palsy (Rothwell et al. 1994), and dementia with lewy bodies (Kofler et al. 2001). Likewise, it is important to recognize that factors such as fear that can potentiate GSRs (Davis et al. 1993) and influence the rate of habituation to repeated startle stimuli (Grillon et al. 1991) may be an additional confounding variable when comparing PRs in individuals with fear of falling, or conditions/contexts in which fear and arousal may differ, or change over time.

Evidence supporting balance perturbations as potentially startling stimuli also provides new perspectives on previous work that has incorporated voluntary movements and postural perturbations to understand cognitive load and adaptation processes involved in balance control (Müller et al. 2004, 2007; Redfern et al. 2002; Woollacott and Shumway-Cook 2002). Out of these dual-task experiments has emerged the “posture first” or “postural prioritization” theories which collectively suggest not only that cognitive resources are involved in mediating PRs, but also that their influences vary with time after perturbation. These conclusions are the result of observed changes to onset latencies of voluntary motor behaviors such that, for a given time frame after perturbation, an increase in voluntary reaction time would reflect relatively greater cognitive demand placed on PRs and vice versa for decreases in reaction time. In light of our findings, decreases in reaction time of voluntary tasks performed in the presence of balance perturbations may instead be the result of StartReact effects. For reasons unrelated to startle, some dual-task experiments utilize choice-reaction time paradigms where more than one response alternative exists instead of the simple reaction time paradigm utilized in the current study. In motor control literature, movements produced during choice reaction time para-
digms are inconsistently influenced by StartReact effects compared with the highly robust results observed when producing the same movement in simple reaction time paradigms (Carlsen et al. 2004, 2011). Thus, although the full extent of StartReact effects in dual-task scenarios remains unclear, the possibility for StartReact effects to interact with voluntary movements during dual-task paradigms alone warrants further examination.

The results of this study also have important implications for understanding the potential neural circuitry that contributes to GSRs and StartReact effects. Recent evidence has revealed that GSRs and StartReact effects are perhaps mediated by partially independent circuits (Alibiglou and MacKinnon 2012; Valls-Solé et al. 2005). For example, techniques such as prepulse and transcranial magnetic stimulation have been used to respectively influence either the SCM response or the StartReact effect in isolation (Alibiglou and MacKinnon 2012; Maslovat et al. 2012; Valls-Solé et al. 2005), suggesting that aspects of the GSR and StartReact circuits are at least partially independent. Our results provide additional evidence that supports the existence of disassociated mechanisms. The significant differences between SCM onsets in TESTPEKT and TESTSAS trials suggested that the timing of the GSR differed depending on stimulus type. However, significant delays of SCM onsets were not met with similar delays in StartReact effects evoked by perturbations and SAS. One would expect that, if both GSRs and StartReact effects were mediated by the same mechanism, the ~40-ms delay in SCM onsets between TESTPEKT and TESTSAS trials would have carried over into a similar delay in StartReact effects. That not being the case aligns with recent work suggesting that GSRs and StartReact effects are the end results of two partially disassociated neural mechanisms.

Conclusions and Future Directions

Our results have supported the notion that FTEs are mediated by a GSR induced by balance perturbations and have added further details regarding the persistent effects of startle stimuli induced by repeated balance perturbations. It appears that GSRs may be a mediating factor in not only a single trial, but possibly in as many 10 repeated trials. Thus the role and importance of GSRs in trials beyond the first must be considered. Future experiments in dynamic postural control must not ignore the possibility for GSRs or the startling nature of balance perturbations to influence their measures, especially if they are temporally based. Furthermore, considering GSRs as a natural consequence of postural instability may open new avenues of research into the neural mechanisms governing PRs and perhaps the relationship between PRs and clinical disorders, such as hyperekplexia and Parkinson's disease, where abnormal responses to startle and postural instability are known to coexist (Brown et al. 1991; Nanhoe-Mahabier et al. 2012).

GRANTS

Funding for this project was provided by the Natural Sciences and Engineering Research Council of Canada (A. D. Campbell, M. G. Carpenter, R. Chua, J. T. Inglis).

DISCLOSURES

No conflicts of interest, financial or otherwise, are declared by the author(s).

AUTHOR CONTRIBUTIONS

Author contributions: A.D.C. and M.G.C. conception and design of research; A.D.C. and J.W.S. performed experiments; A.D.C. and J.W.S. analyzed data; A.D.C. and M.G.C. interpreted results of experiments; A.D.C. prepared figures; A.D.C. drafted manuscript; A.D.C., J.W.S., R.C., J.T.I., and M.G.C. edited and revised manuscript; R.C., J.T.I., and M.G.C. approved final version of manuscript.

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