Vestibular-dependent spinal reflexes evoked by brief lateral accelerations of the heads of standing subjects

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Stimuli. The stimuli consisted of a waveform of a third-order gamma distribution (20) with 4-, 8-, 14-, 20-, and 30-ms rise times,
a train of three consecutive “gamma” stimuli (30-ms duration), and a 500-Hz stimulus (20-ms duration). The stimuli were generated by customized software using a 1401plus D-to-A converter (Cambridge Electronic Design, Cambridge, UK), amplified, and delivered via a handheld mini-shaker device (model 4810, Brüel and Kjaer P/L) with an attached Perspex rod (diameter 2.5 cm, length 9.2 cm). The rod was held normal to the mastoid for the duration of the recording and delivered approximately 1–2 kg of force (22).

The initial movement of the rod was either toward or away from each subject’s head and is referred to as a “positive” or “negative” stimulus, respectively. The reference drive used for BC stimuli was 10 V peak for gamma stimuli and 10 V peak-to-peak for the 500-Hz stimulus (corresponding to peak forces of ~7 N). GVS (20-ms duration) was delivered using a trans-mastoid montage with self-adhesive electrosurgical plating (3M, St. Paul, MN) cut individually to fit behind each ear. The electrodes were connected to an isolated current stimulator (model DS2A), making one the cathode and the other the anode. Recordings were performed using the cathode on the left and anode on the right (CLAR), and with the cathode on the right and the anode on the left (CRAL). The current delivered was between 3 and 4 mA, as tolerated by the subjects.

Head acceleration was recorded during each experiment using two uniaxial accelerometers (Endevco 751–100) placed 5 cm above the external auditory meatus and held in place tightly using a bandage. The initial peak of head acceleration was used for each condition and the ipsilateral and contralateral sides were averaged for each stimulus polarity.

Recordings. Both rectified and unrectified EMG were recorded bilaterally using a standard differential electrode montage. Self-adhesive electrodes (Cleartrace 1700–030, Connem) were placed centrally on the posterior aspect of the lower leg, with reference electrodes being 5 cm above the inferior border of the medial malleolus of the tibia, and the active electrodes being 5 cm above that. An earth electrode was placed on the anterior aspect of their right forearm, 5 cm distal to the antebrachial fossa. For all stimuli, responses were averaged from a minimum of 128 individual trials.

Recordings were made from 50 ms prior to each stimulus to 200 or 250 ms following stimulus onset. EMG was amplified, bandpass filtered (32–1,600 Hz), and sampled, using a Power 1401 analog-to-digital converter (Cambridge Electronic Design, Cambridge, UK). The trials were rectified and averaged using Signal software (Cambridge Electronic Design) and digitally filtered using a 5-point moving average prior to analysis.

Experimental protocol. Except for the seated condition, all participants stood upright with their feet together, leaned forward to activate their soleus muscles, had their eyes closed, and rotated their heads as far as comfortably possible during the experiment. The direction of head rotation was pseudo-randomized such that half looked left (n = 5) and half looked right (n = 5) for the majority of the experiment. During all conditions both positive and negative acceleration stimuli were used. At the start of the experimental session the effect of rise time was assessed to determine which stimulus duration evoked the largest EMG response. This stimulus duration (referred to as the subject’s “optimal duration” stimulus) was then used for the rest of the experiment. The conditions tested using the optimal-duration stimulus included: the standard condition (subject standing, head rotated and mini-shaker applied to the ipsilateral mastoid), stimulation applied to the mastoid contralateral to the direction of head rotation, mastoid stimulation ipsilateral to the direction of head rotation but with head rotation in the opposite direction, the effect of high (+3 dB re 10 V peak = 14.1 V) and low-intensity (−3dB re 10 V peak = 7.08 V) stimuli, and during sitting with tonically activated soleus muscles. Other conditions tested when standing were a train of three consecutive gamma stimuli, a 500-Hz stimulus, and GVS. The experimental session concluded with recording of cVEMPs.

cVEMPs. Subjects were positioned supine (trunk at approximately 45 degrees above the horizontal) and were required to lift their heads up to activate their neck muscles for the duration of the recording. Electrodes were placed bilaterally with the reference electrode 3 cm above the sternum end of the clavicle, and the recording electrode over the sternocleidomastoid (SCM) muscle belly. The earth electrode was placed on the manubriosternal joint. Positive and negative acceleration stimuli were used with rise times of both 4 ms as well as each individual’s optimal stimulus duration. Rectified and unrectified recordings were made from 20 ms before to 80 ms after each stimulus and averaged from a minimum of 200 individual trials using customized software. Measurements were taken at the initial positive-negative peaks (i-p1, i-n1) and the initial positive peak (c-p1) from the SCM ipsilateral and contralateral to the stimulated mastoid, respectively (23).

Data analysis. Responses were analyzed from the leg contralateral to the direction of head rotation (4, 9). For responses which were unclear, amplitude values were calculated using intervals based upon the mean latency values across all conditions. Amplitude values were calculated by taking the average of the differences of the means of the excitatory and inhibitory components, and expressing this as a percentage of the background EMG activation prior to stimulus onset (31). Latency values were taken at the onset of the short-latency (SL), medium-latency (ML), and long-latency (LL) intervals. Corrected cVEMP amplitudes were measured by dividing the absolute amplitude by the mean level of rectified EMG prior to stimulus onset (32). Statistical analysis was performed using SPSS software (Version 18.0, IBM, Chicago, IL). Repeated-measures ANOVAs were used to assess the effects of rise time on SL, ML, and LL amplitudes. Similar methods were used for other experimental conditions, latencies, and accelerometer values. Post hoc paired t-tests were used to compare data between conditions. Calculated t-values are expressed as absolute values. Correlations were performed between cVEMP and EMG amplitude data, using the dominant vestibulocollic projection (ipsilateral n1-p1 corrected amplitudes for the 4 ms and optimum rise-time stimuli) and contralateral soleus EMG data (SL, ML, and LL amplitudes for 4 ms and optimum rise-time stimuli). Results are given in the text and tables as means ± SD and in figures as means ± SE.

Table 1. Mean amplitudes and latencies of the initial peak in head acceleration at various rise times (n = 10)

<table>
<thead>
<tr>
<th>Polarity</th>
<th>4 ms</th>
<th>8 ms</th>
<th>14 ms</th>
<th>20 ms</th>
<th>30 ms</th>
</tr>
</thead>
<tbody>
<tr>
<td>Amplitude, g</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Pos</td>
<td>0.28 ± 0.03</td>
<td>0.17 ± 0.02</td>
<td>0.11 ± 0.01</td>
<td>0.08 ± 0.01</td>
<td>0.06 ± 0.03</td>
</tr>
<tr>
<td>Neg</td>
<td>0.22 ± 0.03</td>
<td>0.15 ± 0.02</td>
<td>0.10 ± 0.01</td>
<td>0.08 ± 0.01</td>
<td>0.05 ± 0.01</td>
</tr>
<tr>
<td>Latency, ms</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Pos</td>
<td>3.3 ± 0.7</td>
<td>5.2 ± 0.8</td>
<td>7.8 ± 0.4</td>
<td>9.9 ± 0.5</td>
<td>12.2 ± 0.6</td>
</tr>
<tr>
<td>Neg</td>
<td>3.4 ± 0.8</td>
<td>5.4 ± 0.7</td>
<td>7.6 ± 0.3</td>
<td>9.5 ± 0.4</td>
<td>11.4 ± 0.4</td>
</tr>
</tbody>
</table>

Values are means ± SD. Pos, positive initial acceleration direction; Neg, negative.
RESULTS

Head acceleration. Peak head accelerations varied from averages of 0.05 to 0.28 g, with larger accelerations produced by the shorter rise time waveforms (F = 43.7, P < 0.001), and with positive accelerations (F = 46.7, P < 0.001; Table 1). The average latency of the peak acceleration varied from 3.3 to 12.2 ms and increased with increasing rise times (F = 49.4, P < 0.001). Positive stimuli evoked slightly later peaks than the negative stimuli, more so at longer rise times (F = 8.2, P = 0.019).

The magnitude of head acceleration for the standard condition was not significantly different from that using contralateral mastoid stimulation, ipsilateral mastoid stimulation with opposite head rotation, or seated conditions (t = 0.5–1.9, P > 0.05 for all comparisons; Table 2). The train of stimuli, high intensity and 500-Hz stimulus produced larger head accelerations than the standard condition (t = 3.5–19.1, P < 0.008 for all comparisons). There were significantly higher and lower acceleration values in parallel with changing the stimulus intensity by 3 dB (t = 7.9–8.8, P < 0.001 for all comparisons).

EMG responses. Responses recorded from the contralateral leg were typically triphasic: with SL, ML, and LL components. When applied to the mastoid ipsilateral to the direction of head rotation, a positive gamma stimulus evoked an excitatory SL peak, while inverting the stimulus polarity inverted the EMG response (Fig. 1A). EMG responses were less frequently observed in the soleus muscle ipsilateral to the direction of head rotation and were therefore not included in the analysis. Using the “optimal” rise time, the latencies for the three responses were 54.2 ± 4.8, 88.4 ± 4.7, and 121 ± 7.1 ms, respectively.

Effect of varying rise times. Response amplitudes were largest for the 14 ms rise time in four subjects and for 20 ms in six subjects. Amplitudes were significantly larger for the ML (14.2 ± 6.4%) interval compared with the SL (8.0 ± 2.9%) and LL (8.2 ± 3.8%) intervals (F = 22.4, P < 0.001). Rise times affected EMG amplitudes (F = 6.81, P < 0.001), and this was more marked for the ML interval (F = 5.11, P < 0.001; Fig. 1B). Amplitudes for the 20-ms rise time for the ML interval were significantly larger than at 4, 8, and 30 ms (t = 3.1–4.2, P < 0.012). There was no significant difference in ML amplitudes between the 14 and 20 ms rise times (t = 1.2, P > 0.05).

The SL, ML, and LL peak latencies increased with longer duration rise times (F = 19.8–64.3, P < 0.001). For the LL peak, positive stimuli produced longer onset latencies than negative stimuli (F = 27.4, P = 0.006). As the rise time increased, the peak latency of the response to the positive stimulus increased more than the response to the negative stimulus for all three peaks (F = 6.3–35.2, P < 0.004; Fig. 1C).

Effect of different conditions. Mean EMG responses under the different conditions tested are shown in Fig. 2. There was a clear series of responses evident for the “standard” condition—standing with head rotated with stimulation applied to the ipsilateral (posterior) mastoid—with average response amplitudes of: SL, 9.05 ± 3.44%; ML, 16.70 ± 4.41%; and LL, 9.75 ± 4.89%. Across conditions (ignoring the 500-Hz stimulus and seated conditions) the amplitude of the ML interval was significantly larger than for the SL and LL intervals (F = 35.6, P < 0.001). Repeated-measures analysis revealed that the ML interval was more affected by differing conditions than the SL and LL intervals were, and that the relative decrease in amplitude was proportionately larger for the ML interval than the SL and LL intervals when using the 500-Hz stimulus and seated conditions (F = 31.8, P < 0.001).

There was no significant difference in onset latencies for the SL, LL, and ML intervals with differing acceleration polarities (F < 0.2, P > 0.05 for all comparisons) or across conditions (F < 0.5, P > 0.05 for all comparisons, excluding the 500-Hz and seated conditions, where the EMG responses were not clear).

There was no significant difference in soleus muscle-rectified EMG activity between standard (48.6 ± 10.9 μV) and seated (39.9 ± 16.9 μV) conditions (t = 1.5, P = 0.127). There were no clear EMG responses when stimulated with the 500 Hz stimulus or when seated. Using average latencies, five subjects had small responses evoked by the 500-Hz stimulus, and three subjects while seated. The response amplitudes were significantly smaller for the seated condition (SL, 2.80 ± 1.67%; ML, 2.95 ± 3.10%; LL, 1.55 ± 1.26%), the 500-Hz stimulus (SL, 2.20 ± 1.16%; ML, 1.55 ± 1.07%; LL, 1.35 ± 1.25%), the lower intensity stimulus (SL, 4.45 ± 3.16%; ML, 11.20 ± 4.57%; LL, 6.50 ± 4.19%), and the train of gamma stimuli (SL, 4.30 ± 2.95%; ML, 12.95 ± 6.36%; LL, 6.90 ± 4.20%) than for the standard condition (t = 2.41–11.6, P < 0.039 for all comparisons).

Although there was a more variable response to negative accelerations when given anteriorly, overall there was no significant difference in amplitudes between the standard condition and when stimulation was applied to the anterior mastoid (SL, 8.80 ± 2.78%; ML, 14.75 ± 5.44%; LL, 9.60 ± 5.25%;

Table 2. Mean amplitudes and latencies of the initial peak in head acceleration at various conditions (n = 10)

<table>
<thead>
<tr>
<th>Polarity</th>
<th>Standard condition</th>
<th>Contra mastoid stim</th>
<th>Ipsi mastoid stim (opp side)</th>
<th>Low-intensity stim</th>
<th>High-intensity stim</th>
<th>Train of gamma stim</th>
<th>500-Hz stim</th>
<th>Seated (tonic contraction of leg muscles)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pos.</td>
<td>0.10 ± 0.02</td>
<td>0.10 ± 0.02</td>
<td>0.10 ± 0.02</td>
<td>0.07 ± 0.02</td>
<td>0.15 ± 0.03</td>
<td>0.28 ± 0.03</td>
<td>0.25 ± 0.13</td>
<td>0.10 ± 0.02</td>
</tr>
<tr>
<td>Neg.</td>
<td>0.09 ± 0.01</td>
<td>0.09 ± 0.02</td>
<td>0.09 ± 0.02</td>
<td>0.06 ± 0.01</td>
<td>0.13 ± 0.03</td>
<td>0.22 ± 0.02</td>
<td>0.26 ± 0.16</td>
<td>0.09 ± 0.02</td>
</tr>
<tr>
<td>Latency, ms</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Pos.</td>
<td>9.1 ± 1.2</td>
<td>9.0 ± 1.1</td>
<td>9.2 ± 1.3</td>
<td>9.1 ± 1.4</td>
<td>9.4 ± 1.1</td>
<td>3.5 ± 2.1</td>
<td>3.0 ± 2.1</td>
<td>9.2 ± 1.8</td>
</tr>
<tr>
<td>Neg.</td>
<td>8.8 ± 1.1</td>
<td>8.7 ± 0.9</td>
<td>8.6 ± 1.0</td>
<td>8.9 ± 1.2</td>
<td>8.9 ± 1.0</td>
<td>4.9 ± 2.9</td>
<td>5.1 ± 4.0</td>
<td>8.7 ± 1.2</td>
</tr>
</tbody>
</table>

Values are means ± SD. Standard condition consisted of standing with the head rotated to the right or left, with the impulse applied to the mastoid ipsilateral to the direction of head rotation (posterior). Pos, positive initial acceleration; Neg, negative; Ipsi, ipsilateral to direction of head rotation; Contra, contralateral; Stim, stimulation.

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t = 0.08–0.88; P > 0.40 for all comparisons), nor was there when the stimuli were given with the head rotated in the opposite direction (SL, 6.70 ± 2.94%; ML, 15.65 ± 6.28%; LL, 8.10 ± 4.81%) (t = 0.58–1.70; P > 0.12). Despite the greater acceleration induced, there was no significant difference in SL, ML, and LL amplitudes (8.05 ± 2.95%; 16.20 ± 3.54%; 9.65 ± 3.54%) between the higher stimulus intensity and the standard condition (t = 0.07–0.81, P > 0.43 for all comparisons).

Gamma stimulation vs. GVS. GVS elicited a biphasic EMG response while the potential produced using the gamma stimulus was triphasic. The SL amplitude was significantly larger when produced by GVS (GVS SL: 15.2 ± 4.6%; t = 2.55, P = 0.031), whereas the ML amplitude showed an opposite trend (GVS ML: 13.3 ± 4.17%; t = 1.96, P = 0.082).

The mean onset latency of the SL interval occurred significantly earlier with GVS (standard condition SL: 57.6 ± 3.9 ms; GVS SL: 50.3 ± 3.5 ms) (t = 3.9, P = 0.001). The onset of the ML response occurred significantly earlier with the gamma stimulus than GVS [standard condition ML: 87.7 ± 3.8 ms; GVS ML (onset): 94.6 ± 6.4 ms; t = 4.0, P = 0.001], while the end of the LL and the GVS ML was not significantly different from each other [standard condition LL: 155 ± 7.1 ms; GVS ML (end): 156 ± 12.5 ms; t = 0.7, P = 0.596].

cVEMP responses. All 10 subjects showed cVEMPs when stimulated using both the optimal stimulus duration for the soleus response (14 or 20 ms) and the 4-ms rise time stimulus (Fig. 3). Corrected amplitudes evoked by the 4-ms rise time stimulus were significantly larger than those evoked by the (postural) optimum duration for both the i-n1/p1 (4 ms: 1.45 ± 0.46; optimum-duration stimulus: 1.05 ± 0.41) and c-p1 peaks (4 ms: 0.96 ± 0.36; optimum-duration stimulus: 0.65 ± 0.29) (t = 2.7–3.0, P < 0.04 for both comparisons). Latencies were significantly longer when evoked by the optimum stimulus duration rather than the 4-ms rise time stimulus for the i-p1 (4 ms: 14.0 ± 3.0 ms; optimum-duration stimulus: 12.6 ± 2.7 ms; t = 2.3, P = 0.049).
ms: 13.7 ± 1.4 ms; optimum-duration stimulus: 14.8 ± 1.1 ms), i-n1 (4 ms: 20.9 ± 2.0 ms; optimum duration stimulus: 23.0 ± 2.1 ms), and c-p1 peaks (4 ms: 19.4 ± 2.1 ms; optimum-duration stimulus: 22.1 ± 2.1 ms) (t = 3.0 to 4.4, P < 0.007 for all comparisons).

There was a significant positive correlation only between the cVEMP corrected amplitude for the 4-ms rise time stimulus and the LL amplitude of the optimum rise-time stimulus (r = 0.75, P = 0.012).

Bilateral vestibular dysfunction. The three patients had no or minimal oVEMPs or cVEMPs evoked using either the gamma or conventional sound stimuli. No detectable EMG responses from the soleus muscles were observed from the patient group following stimulation applied to either the left or right mastoids (Fig. 4).
DISCUSSION

Human postural responses can be separated into those to sudden perturbations (phasic) and those to slow changes in posture (11). Consistent with such a distinction, Bacsi and Colebatch (2) provided evidence for a role of both short-latency vestibular reflexes and conscious perception of vestibular activity in determining levels of spontaneous sway. Sudden postural disturbances will activate both somatosensory and vestibular responses and both have been shown to be capable of evoking postural responses. Horak et al. (13), for example, were able to show postural responses to head-only displacements, which were lost in acquired vestibular disease and which were modified by changing somatosensory information. These observations support a role for vestibular afferents in responses to postural disturbances affecting the head. We have demonstrated postural responses triggered by head accelerations that were consistent with a stabilizing effect for any associated body displacement. Our stimulus was a physiological one with the size of our acceleration being similar to those occurring under natural conditions such as walking (20). The response appeared to saturate around the levels of intensity we were using. A positive acceleration stimulus applied to the mastoid ipsilateral to the direction of head rotation is similar to having the head pushed forward. In this condition, the response in soleus began with an initial SL excitation which would be expected to act to stabilize the disturbance of the body. Conversely, a negative stimulus applied to the mastoid ipsilateral to the direction of head rotation, or a positive stimulus on the mastoid contralateral to the direction of head rotation, similar to having the head pushed backward, evoked an initial relaxation of soleus.

The gamma stimulus is a smooth impulse of acceleration designed to avoid causing skull “ringing” and was first applied to evoking vestibular-dependent responses from extraocular muscles (oVEMPs; 26). There was a striking dependence of the initial polarity of the oVEMP on the direction of the impulse. A positive impulse, accelerating the head contralaterally, evoked an initial negativity while the oppositely directed acceleration evoked an initial positivity or delayed negativity. Given the sensitivity of the utricle to lateral accelerations (8), it was proposed as the probable vestibular organ primarily excited by the stimulus. As this pathway is known to be crossed (12, 16), the observations of Todd et al. (26) suggest the presence of an excitatory projection from the medial half of the utricle to the contralateral inferior oblique muscle, the likely origin of the surface response (22). Rosengren et al. (23) demonstrated that the gamma stimulus applied to the mastoid, as here, caused predominately linear head accelerations and that it also evoked short-latency cVEMP responses in the neck. Again, the responses were direction-dependent and of opposite initial polarity on the two sides of the neck for a given direction of head acceleration. Rosengren et al. (23) also showed that responses similar to the positive gamma impulse could be evoked by simply tapping the mastoids. The authors argued that a reflex arising primarily from afferents lying on the medial side of the utricle was likely to explain the effects seen. The present findings extend the range of effects evoked by short impulsive lateral head accelerations to short-latency EMG responses in leg muscles in standing subjects. The most effective rise time was longer than for vestibulocollic responses evoked using the same type of stimulus, consistent with a differing efferent pathway. Importantly, as previously demonstrated, the polarity of the
responses for the legs was determined by the direction of head acceleration.

GVS stimulation of the labyrinths has been widely applied as a method of investigating the postural consequences of vestibular activation (e.g., 4, 7, 9, 14, 15, 19; for review, see 10). Although the technique is not physiological, and causes activation of irregularly discharging afferents arising from all vestibular receptors (17), it appears that the nervous system can extract meaningful information from the ensemble of discharges (10). GVS has demonstrated fundamental properties of postural responses evoked by vestibular stimulation: the dependence upon head orientation, postural context (4, 9), and the presence of segmented EMG responses in leg muscles, properties that we have confirmed also hold for our physiological stimulus. A cathodal stimulus over the posterior mastoid of the rotated head evokes an initial excitatory SL response, as did our positive acceleration under the same circumstances. These findings are consistent with both stimuli being primarily excitatory for vestibular afferents (30). The latency of the initial short-latency response is similar for both types of stimuli. Overall these properties suggest that our acceleration impulses evoke vestibular-dependent spinal reflexes in leg muscles which may share the same central processing and pathways as the early responses to GVS. A role for the otoliths has previously been proposed for the initial effects of galvanic stimulation, specifically the utricle (10, 28). Modeled as a mixed effect of otolith and canal activation (28), more recently the role of otolith afferents in GVS-evoked responses has been questioned (6, 18), although the opposite view has also been put forward (5). Critical to the arguments against a role for the otoliths are the changes in the reflex responses that occur when head pitch is altered, thereby changing the predicted net effect of otolith excitation. The arguments are indirect and do not allow for associated changes in afferents from neck proprioceptors and implicitly assume that postural responses always accurately reflect the computed net vestibular acceleration vector. Our findings provide new evidence in support of an otolith, probably utricular, contribution to postural reflexes evoked by vestibular stimulation. Utricular activity is likely therefore also to contribute to the initial postural reflex responses to electrical stimulation (GVS).

In addition to the properties previously demonstrated using GVS, we have shown, using a physiological stimulus, that changing the rise time of the acceleration pulse mainly affects the ML component of the EMG response and that repeated stimuli, despite evoking larger accelerations, do not evoke larger postural responses than a single impulse. In addition, the 500-Hz stimulus, despite evoking larger accelerations, was relatively ineffective. Basci et al. (3), who used air-conducted sound, found their 500-Hz stimulus produced either smaller amplitude or absent vestibular-spinal reflexes compared with “matched” GVS effects, a difference they attributed to differences in the afferents stimulated by the two techniques. The poor EMG response we found may be because 500-Hz vibration stimulates different vestibular afferents, possibly the sacculus, with weaker projections to the legs or due to the lack of

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Fig. 4. Effects in vestibular failure. Grand means (n = 3) of rectified EMG recordings in patients with severe vestibular failure, recorded during standing. Average evoked accelerations recorded from each side of the head are shown. Dashed lines indicate stimulus onset. Despite adequate head accelerations and tonic levels of EMG activity, no definite responses were evoked by the acceleration stimulus.
clear directional information inherent in high-frequency skull vibration (33).

While GVS and our acceleration transient had several properties in common, their effects were not identical. Our acceleration pulse evoked a triphasic response while GVS evoked a biphasic one. This difference may indicate how the nervous system deals with a fundamental uncertainty associated with a linear acceleration. Due to Einstein’s equivalence principle, otolith afferents respond similarly to inertial (a translation or “shove”) and gravitational (“tilt”) stimuli and are inherently ambiguous (1). Thus similar changes in utricular discharge would be evoked by a shove to the left or a tilt to the right, for example. The difficulty is that the appropriate responses to these two types of stimuli are opposite. For the shove, the body should move to the right, while for the tilt the body should move to the left. Prolonged GVS has been demonstrated to simulate a tilt effect (7), analogous to righting reflexes shown in animal models (e.g., 25). The nature of the response evoked by our acceleration stimulus shows a simple way that this problem could be partly resolved. Changing the duration of the rise time mainly affected the size of the ML component while the SL and LL responses remained largely fixed. We have also previously shown using GVS that the ML component of the response to GVS is less sensitive to current rise time than is the SL component (21). These observations imply that for a short, phasic stimulus, the effect of the SL and late responses would dominate, giving one direction of response while, for a long-duration stimulus, the ML response would dominate, giving the opposite. The responses to GVS, usually given over a relatively long duration, would therefore be expected to simulate those of a tilt while those to a short acceleration, such as we used here, would probably be interpreted as a perturbation, to be resisted. A triphasic reflex, as shown here, with relatively fixed initial and final phases, can achieve these two distinct types of responses.

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DISCLOSURES

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

AUTHOR CONTRIBUTIONS

Author contributions: R.L. and S.G. performed experiments; R.L. and S.G. prepared figures; R.L. drafted manuscript; S.G. and J.G.C. analyzed data; S.G. and J.G.C. edited and revised manuscript; J.G.C. conception and design of research; J.G.C. interpreted results of experiments; J.G.C. approved final version of manuscript.

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