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# Frequency-Specific Modulation of Vestibular-Evoked Sway Responses in Humans

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**Dakin CJ, Luu BL, van den Doel K, Inglis JT, Blouin J-S.** Frequency-specific modulation of vestibular-evoked sway responses in humans. *J Neurophysiol* 103: 1048–1056, 2010. First published December 23, 2009; doi:10.1152/jn.00881.2009. Galvanic vestibular stimulation (GVS) results in characteristic muscle and whole-body responses in humans maintaining standing balance. However, the relationship between these two vestibular-evoked responses remains elusive. This study seeks to determine whether mechanical filtering from conversion of lower-limb muscle activity to body sway, during standing balance, can be used to attenuate sway while maintaining biphasic lower-limb muscle responses using frequency-limited stochastic vestibular stimulation (SVS). We hypothesized that SVS deprived of frequencies <2 Hz would evoke biphasic muscle responses with minimal whole-body sway due to mechanical filtering of the higher-frequency muscle responses. Subjects were exposed to five stimulus bandwidths: two meant to induce sway responses (0–1 and 0–2 Hz) and three to dissociate vestibular-evoked muscle responses from whole-body sway (0–25, 1–25, and 2–25 Hz). Two main results emerged: *1*) SVS-related sway was attenuated when frequencies <2 Hz were excluded, whereas multiphasic muscle and force responses were retained; and *2*) the gain of the estimated transfer functions exhibited successive low-pass filtering of vestibular stimuli during conversion to muscle activity, anteroposterior (AP) moment, and sway. This successive low-pass filtering limited the transfer of signal power to frequencies <20 Hz in muscle activity, <5 Hz in AP moment, and <2 Hz in AP trunk sway. Consequently, the present results show that SVS delivered at frequencies >2 Hz to standing humans do not cause a destabilizing whole-body sway response but are associated with the typical biphasic lower-limb muscle responses.

## INTRODUCTION

Galvanic vestibular stimulation (GVS) has long been used as a means to probe vestibular function (for review, see Fitzpatrick and Day 2004). In humans maintaining standing balance, GVS provides an isolated vestibular error signal, allowing the study of the resulting whole-body movements and myogenic responses in muscles involved in the control of balance (Britton et al. 1993; Fitzpatrick et al. 1994; Iles and Pisini 1992; Lee Son et al. 2008; Lund and Broberg 1983; Nashner and Wolfson 1974). Recently, we have shown that stochastic vestibular stimulation (SVS) over a 0 to 50 Hz bandwidth elicits vestibular-evoked balance and muscle responses similar to those observed using GVS (Dakin et al. 2007). SVS–electromyographic (EMG) coupling was observed over the 0 to 20 Hz bandwidth coinciding with previous estimates of the dynamic range of vestibular function (Armand and Minor 2001; Grossman et al. 1988; Huterer and Cullen 2002). Whole-body responses to vestibular stimulation, however, appear to follow the vestibular stimulus when frequencies <5 Hz are provided (Fitzpatrick et al. 1996; Latt et al. 2003; Lund and Broberg 1983; MacDougall et al. 2006; Moore et al. 2006; Pavlik et al. 1999), with the largest responses elicited when the frequency content of the stochastic vestibular stimulus is <2 Hz (Fitzpatrick et al. 1996; Pavlik et al. 1999).

Ongoing control of upright balance is thought to occur through low-frequency lower-limb muscle activity (~2.5 Hz), which is mechanically filtered to produce an even lower frequency body sway (<1 Hz) (Bawa and Stein 1976; Fitzpatrick et al. 1996; Latt et al. 2003; Loram et al. 2005). As the activation frequency of lower-limb muscle activity increases, its amplitude must also increase to maintain impulse magnitude (and therefore sway amplitude) due to the inertial load of the body. However, muscle-sway gain decreases with increasing frequency, such that muscle activity at 5 Hz evokes body sway, which is 100-fold smaller than that evoked by similar muscle activity amplitude at 0.5 Hz (Fitzpatrick et al. 1996). Thus higher-frequency muscle activation is mechanically filtered out in its transfer to lower-limb moment production and resulting sway. SVS, in contrast, evokes muscle activity at frequencies from 0 to 20 Hz. By removing the lower-frequency content of the SVS stimulus (<2 Hz), we predict that the remaining higher-frequency signal is mechanically filtered out, resulting in attenuation of the whole-body sway response while maintaining the biphasic lower-limb muscle response.

The primary aim of this study was to determine whether stochastic vestibular stimuli excluding low-frequency bandwidths could elicit vestibular-evoked lower-limb biphasic myogenic potentials with a reduction in associated sway responses. This could be particularly beneficial for exposing basic physiological phenomena that would otherwise be masked by the balance response or for studying vestibular responses in patients with balance disorders who are already unstable. To unveil frequencies specific to the SVS-evoked sway responses, participants were exposed to five SVS stimuli: two meant to maximally elicit SVS-related whole-body sway (0–1 and 0–2 Hz) and three meant to dissociate sway from SVS-evoked muscle responses (0–25, 1–25, and 2–25 Hz). Two control trials were also performed to compare sway associated with SVS to sway observed with random 1 mA GVS pulses and free standing (no vestibular stimulation). We hypothesized that vestibular stimuli with low frequencies removed (stimulus bandwidth between 1–25 and 2–25 Hz) would evoke biphasic muscle responses with minimal whole-body sway due to me-

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chanical filtering of the SVS-evoked higher-frequency muscle responses.

## METHODS

### *Subjects*

Twelve healthy subjects [9 male, 3 female; mass 70 ± 10 kg and height 1.72 ± 0.10 m ($\bar{X}$ ± SD)] between the ages of 21 and 33 yr, with no known history of neurological disease or injury participated in this study. The experimental protocol was explained to each subject and their written, informed consent was obtained. All procedures used in this study conformed to the standards of the Declaration of Helsinki and were approved by the University of British Columbia's clinical research ethics board.

### *Stimulus*

Stochastic vestibular stimulation (SVS) and galvanic vestibular stimulation (GVS) were delivered using a bipolar binaural electrode configuration with carbon rubber electrodes (9 $cm^2$), coated with Spectra 360 electrode gel (Parker Laboratories, Fairfield, NJ), secured over the mastoid processes with an elastic headband. The stimuli were created on a PC computer using LabVIEW software (National Instruments, Austin, TX) and delivered as an analog signal via a data acquisition board (PXI-6289; National Instruments) to an isolated constant-current unit (Model 2200 Analog Stimulus Isolator; AM Systems, Carlsborg, WA). The stochastic signals (Fig. 1*A*) lasted 133 s and were designed to provide similar power amplitude to each frequency component within and across all stimuli (Fig. 1*B*). This resulted in different root-mean-square (RMS) amplitudes for the different bandwidth stimuli: 0.20 mA (0–1 Hz), 0.29 mA (0–2 Hz), 0.98 mA (0–25 Hz), 0.96 mA (1–25 Hz), and 0.93 mA (2–25 Hz). On additional trials, 1 s 1 mA GVS pulses were provided, with each trial consisting of five anode right (cathode left) and five anode left (cathode right) pulses presented randomly, for a total of 20 pulses. Galvanic pulses were delivered with a variable interstimulus interval of 10–15 s.

### *Test procedures*

Participants were required to stand on a force plate (Bertec 4060–80; Bertec, Columbus, OH) with their feet 2–3 cm apart (as measured at the medial malleoli). The participants were instructed to stand relaxed with their eyes closed, arms by their sides, and their head turned to the left with Reid's plane tilted nose up 18° from parallel to the floor. This head position maximizes the postural response to vestibular stimulation in the anteroposterior direction (Cathers et al. 2005; Day and Fitzpatrick 2005), aligning the postural response to the line of action of the soleus, gastrocnemius, and tibialis anterior muscles. Head pitch and yaw and trunk sway, measured at the level of the sternal notch, were monitored on-line using a three-dimensional motion-tracking system (TrakStar: Ascension Technology, Burlington, VT) to control for changes in head position (Cathers et al. 2005). Monitoring of head pitch limited RMS variability around the desired position to 0.33°.

Participants were exposed to five stochastic stimuli (0–1, 0–2, 0–25, 1–25, and 2–25 Hz) to determine whether prolonged sway responses to vestibular stimulation are primarily associated with vestibular stimulus frequencies <2 Hz. Two trials were designed to induce sway (0–1 and 0–2 Hz) and three trials to examine the potential dissociation between muscle and sway responses (0–25, 1–25, and 2–25 Hz). Each subject also performed a single free-stance trial and two GVS trials (see stimulation parameters cited earlier) as controls to compare mean removed RMS trunk sway amplitude elicited by the SVS trials. Rest periods were provided at the request of the participant to avoid any sign of fatigue.

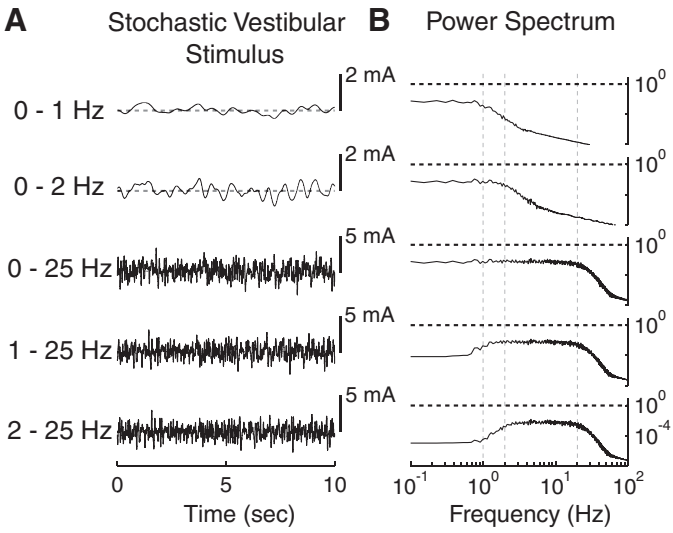


FIG. 1. Vestibular stimuli and corresponding power spectra. *A*: vestibular stimuli for each bandwidth of stimulation. *B*: log-based power spectra for each of the bandwidths of stimulation. Signal power for each bandwidth of stimulation is localized in the bandwidth of interest.

### *Electromyography and signal analysis*

EMG was collected for the soleus, medial gastrocnemius, and tibialis anterior of the right leg (Fig. 2*A*). EMG was amplified (×2,000; NeuroLog, Digitimer, Hertfordshire, UK) and band-pass filtered (10–1,000 Hz). EMG, vestibular stimuli, and force plate data were digitized and recorded at 5,000 Hz with a standard data acquisition board (PXI-6289; National Instruments) using a custom LabVIEW software program. Horizontal forces acting on the subject and anteroposterior/mediolateral trunk displacement were used to describe the balance and sway behavior of the subjects (Fig. 2*A*). Sway was recorded with the TrakStar at 240 Hz, low-pass filtered at 20 Hz, and interpolated to 5,000 Hz for correlation analysis with the vestibular stimuli.

Cumulant density estimates were used to represent the time–domain relationship between vestibular stimulation and muscle activity. The cumulant density estimate provides similar temporal and spatial characteristics to the muscle responses observed with trigger-averaged GVS (Dakin et al. 2007). A consequence of this technique is that the cumulant density estimate between two measured signals provides a correlation-like measure (equivalent to the cross-covariance) and therefore must be interpreted as an associative rather than causal relationship. In this study, cumulant density estimates were determined for a random, controlled input signal (vestibular stimuli) and measured physiological signals (EMG, force, and sway); thus responses correlated to the vestibular stimuli must have been evoked by the vestibular stimuli. This is supported by the phase relationship between SVS and EMG responses exhibiting linear slopes consistent with GVS-evoked responses (Dakin et al. 2007). SVS–EMG, SVS–force, and SVS–sway cumulant density estimates are accordingly referred to as related responses that hold no physical values (e.g., N or m). It should be noted, however, that the physiological signals likely contain SVS-evoked contributions from both an open-loop process and the associated recurrent feedback.

Gain functions were used to provide an estimate of the SVS transfer function at six subsections in the SVS–sway pathway (Fig. 2*B*) (Fitzpatrick et al. 1996; Halliday et al. 1995; Rosenberg et al. 1989). Neural control of sway is limited to muscle excitation and thus changes in SVS-evoked movement patterns resulting after muscle activation should be related to mechanical factors. Gains were therefore calculated along the SVS–sway pathway for the 0 to 25 Hz stimulus bandwidth (SVS–EMG, SVS–AP moment, SVS–AP sway,

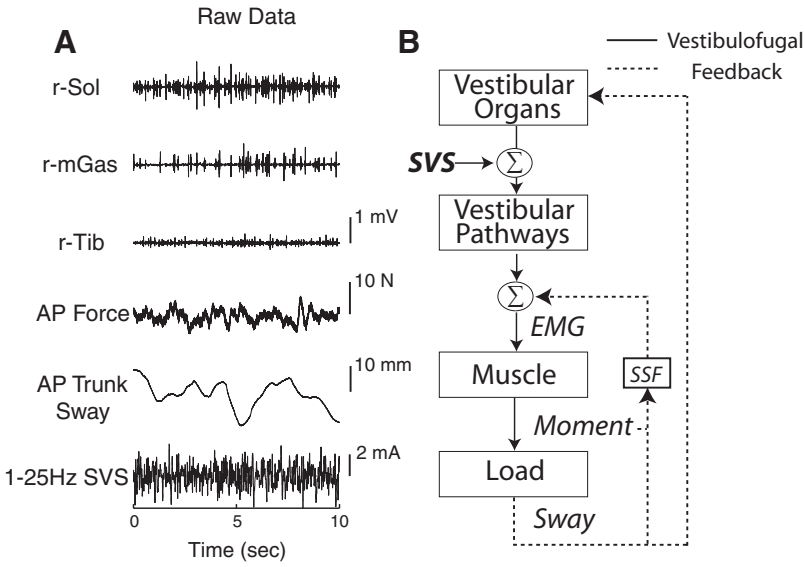


FIG. 2. Raw data and vestibular-sway pathway. *A*: 10 s of raw data displaying muscle EMG, anteroposterior directed force, anteroposterior directed trunk sway, and SVS stimulus. *B*: schematic describing the vestibular-sway pathway. AP, anteroposterior; *r*-Tib, right tibialis anterior; *r*-mGas, right medial gastrocnemius; *r*-Sol, right soleus; SSF, somato-sensory feedback; EMG, electromyography; SVS, stochastic vestibular stimulation.

EMG–AP moment, EMG–AP sway, and AP moment–AP sway) to *1*) determine whether reductions in SVS to sway signal bandwidth are due to mechanical factors and *2*) localize sources of mechanical filtering of the signal (Fig. 2*B*). Gains were calculated using two methods: the first method was to directly calculate the SVS–EMG, SVS–AP moment, and SVS–AP sway gains (Fig. 5*A*). The direct approach has one central caveat in that it assumes an open-loop pathway and neglects the effects of feedback. The resulting closed-loop transfer functions therefore must be assumed to include the frequency characteristics of associated feedback. To separate the effects of feedback we used a second method to calculate the gains: the joint input–output approach (described by van der Kooij et al. 2005) (Fig. 5*B*). By dividing two closed-loop transfer functions, a mathematical cancellation of feedback occurs, thus allowing estimation of the inferred open-loop transfer function (Fitzpatrick et al. 1996; Kiemal et al. 2008); for example, to identify the inferred open-loop EMG–sway transfer function the SVS–sway closed-loop transfer function is divided by the SVS–EMG closed-loop transfer function. This approach was used to estimate the inferred open-loop transfer functions between EMG–AP moment, EMG–AP sway, and AP moment–AP sway (Fig. 5*B*).

Coherence, cumulant density, and direct gain estimates were derived using a Matlab script based on the methods described by Rosenberg and colleagues (1989). Coherence estimates were calculated for each participant using segments of $2^{12}$ data points with 95% confidence limits. Gains were calculated only at frequencies exhibiting significant coherence since gain has meaning only when a relationship exists between two compared signals (Halliday et al. 1995). For the cumulant density and gain estimates, digitized EMG, force plate, and sway data recorded during the stochastic SVS trials were time-locked to SVS onset and cut to provide 10 disjoint segments of $2^{16}$ data points within each participant for each condition. Participant data were then averaged across all participants to provide grouped means. EMG data were full-wave rectified and cumulant density and gain functions between SVS–EMG, SVS–horizontal forces, and SVS–sway signals were estimated for each trial condition within and across all participants. SVS–EMG, SVS–horizontal forces, and SVS–sway cumulant densities and gain estimates were analyzed with resolutions of 0.076 Hz (13.1 s/segment) to identify the lower-frequency components of both functions. Amplitudes of the cumulant density functions were normalized by the product of the vector norms of the input (SVS) and the output (EMG, force, or sway) signals

$$\frac{(2\pi/T)\sum_{|j|\le T/2} f_{xy}(\lambda_j)e^{i\lambda_j u}}{\sqrt{\bar{x}^2}\sqrt{\bar{y}^2}}$$

where $f_{xy}$ is the cross spectrum, $\lambda_j$ are the Fourier frequencies, $T$ is the number of points in the Fourier transform, $u$ is the lag, $i$ is the square root of $-1$, and $x$ and $y$ are the input and output data series, respectively. This normalization procedure transforms the cumulant density values into standard coefficients of correlation ($r$ values bounded between $-1$ and $+1$), providing meaningful units of magnitude. A consequence of the normalization process is that the amplitudes of the cumulant density estimates are scaled by the RMS of the contributing signals, thereby changing the cumulant density estimates relative magnitudes. This normalization procedure primarily influenced the relative magnitudes of the 0 to 1 and 0 to 2 Hz cumulant density estimates, while having little influence on the 0 to 25, 1 to 25, and 2 to 25 Hz cumulant density estimates, and did not change whether a stimulus exhibited significant EMG, force, or sway responses on a subject-per-subject basis.

Confidence intervals (CIs) were calculated for individual subject cumulant density functions based on the methods described by Rosenberg and colleagues (1989). Cumulant density functions were evaluated on a subject-by-subject basis to determine the presence of significant responses (i.e., when the values exceeded the computed 0.95 CIs) and then averaged across subjects. The final averages are presented without CIs. By convention, anode right currents are represented as a positive vestibular signal. Thus a positive cumulant density function indicates that anode right currents induced muscle facilitation or anterior directed forces or sway.

Multiphasic muscle and force responses are observed following GVS (Fitzpatrick et al. 1994) and broad-bandwidth SVS (Dakin et al. 2007), although only the early and middle latency vestibular-evoked force or muscle correlations are described due to their potential physiological relevance (Britton et al. 1993; Cathers et al. 2005). In contrast, narrow-bandwidth (0–1 and 0–2 Hz) muscle and force correlations are described as having first and second peaks, rather than early and middle latency responses because they also exhibit multiphasic patterns but with spatial and temporal characteristics different from those of the broad-bandwidth stimuli. Local maxima and minima for the correlated trunk sway, force, and muscle responses were extracted from the individual subjects' cumulant density function only when they reached significance. Mean removed RMS values for AP trunk sway were also calculated to provide estimates of AP trunk

sway variability around the mean value during the vestibular stimulation and control trials.

### *Data reduction*

Mean removed RMS values for AP trunk sway measured for each SVS frequency bandwidth, square-wave GVS, and control trials were compared using a one-way repeated-measures ANOVA. The early and middle latency components of the EMG and force responses were determined using the peak correlation (or trough) observed in the time cumulant densities and compared between the 0 to 25, 1 to 25, and 2 to 25 Hz trials using a one-way repeated-measures ANOVA, whereas the first and second peaks of the EMG and force in the 0 to 1 and 0 to 2 Hz trials were compared using paired *t*-tests. Direct comparisons of the 0 to 1 and 0 to 2 Hz trials with the 0 to 25, 1 to 25, and 2 to 25 Hz trials were not performed because: *1*) the narrow bandwidth of the 0 to 1 Hz trial limited the random nature of the wave, leading to correlations prior to zero time lag, similarly to what is observed when the input signal is a sine wave; and *2*) the 0 to 1 and 0 to 2 Hz stimuli resulted in biphasic waveforms, which did not resemble the spatial and temporal characteristics of the 0 to 25, 1 to 25, and 2 to 25 Hz trials. Magnitude comparisons were not performed in the tibialis anterior because significant correlations in SVS–muscle activity were not observed for all participants (see Table 1). Decomposition of the main effects were performed using Fisher's least significant difference (LSD) tests due to our a priori hypothesis—i.e., that sway would be reduced when vestibular stimuli excluded frequencies <1 or 2 Hz. Statistical significance was set at $P < 0.05$. Data are presented as means ± SD.

## RESULTS

### *Electromyographic responses*

Lower-limb muscle activity was correlated with each of the 0 to 25, 1 to 25, and 2 to 25 Hz vestibular stimuli (Fig. 3). The plantar flexor responses exhibited early latency positive and middle latency negative going peaks, whereas the right tibialis anterior exhibited corresponding peaks of opposite polarity (see Table 1 for latencies). The timing and polarity of the early and middle latency muscle responses were comparable across each of the 0 to 25, 1 to 25, and 2 to 25 Hz bandwidths (Fig. 3), whereas the amplitude of the early and middle latency peaks varied depending on the bandwidth of the stimulus provided. In the soleus muscle, the peak correlations of the early latency response for the 2 to 25 Hz stimuli (0.073 ± 0.05) were 26 and 35% larger than those for the 1 to 25 and 0 to 25 Hz stimuli, respectively [$F_{(2,11)} = 37.5$ , $P < 0.05$; Fisher LSD, multiple $P < 0.05$], whereas in the medial gastrocnemius, the peak correlations of the early latency response for the 2 to 25 Hz stimulus (0.105 ± 0.06) were 38 and 64% larger than those for the 1 to 25 and 0 to 25 Hz stimuli, respectively [$F_{(2,11)} = 63.4$, $P < 0.05$; Fisher LSD, multiple $P < 0.05$]. There was no significant difference in the magnitude of early latency muscle responses for the 0 to 25 and the 1 to 25 Hz stimuli (multiple $P > 0.05$; 0.054 ± 0.03 vs. 0.058 ± 0.03, *r*-Sol; 0.064 ± 0.03 vs. 0.076 ± 0.03, *r*-mGas). The medium latency correlation, however, was not consistent across muscles, showing similar magnitude between stimuli in the soleus [$F_{(2,11)} = 36.9$, $P > 0.05$; −0.084 ± 0.06 (2–25 Hz), −0.090 ± 0.05 (1–25 Hz), −0.092 ± 0.05 (0–25 Hz)], but smaller magnitude for the 2 to 25 Hz trial than that for both the 0 to 25 and 1 to 25 Hz trials in the medial gastrocnemius [$F_{(2,11)} = 46.2$, $P < 0.05$; −0.100 ± 0.07 (2–25 Hz), −0.121 ± 0.06 (1–25 Hz), −0.121 ± 0.05 (0–25 Hz)].

The muscle responses correlated with SVS at bandwidths of 0 to 1 and 0 to 2 Hz did not resemble the typical motor responses triggered by GVS or 0 to 25 Hz SVS. The low-frequency SVS signals were associated with a biphasic correlation in lower-limb muscles, characterized by a first peak starting before or at the zero second time lag due to the narrow bandwidth of the 0 to 1 and 0 to 2 Hz SVS (Fig. 3). In the plantar flexors, the related muscle correlation exhibited a negative followed by a positive going waveform, whereas the

TABLE 1. *Early/first, middle/second, and late EMG latencies for each of the stochastic stimuli*

| | Stimulus, Hz | Early/First | Middle/Second | Late | Number of Subjects |
|---|---|---|---|---|---|
| *r*-Sol | 0–1 | −39 ± 109 | 693 ± 173 | | |
| | 0–2 | 89 ± 42 | 477 ± 180 | | |
| | 0–25 | 62 ± 6 | 104 ± 12 | 198 ± 47 | *n*=12 |
| | 1–25 | 62 ± 6 | 103 ± 11 | 185 ± 32 | |
| | 2–25 | 63 ± 8 | 116 ± 26 | 192 ± 43 | |
| *r*-mGas | 0–1 | −25 ± 88 | 720 ± 144 | | |
| | 0–2 | 95 ± 40 | 522 ± 174 | | |
| | 0–25 | 62 ± 7 | 109 ± 12 | 244 ± 44 | *n*=12 |
| | 1–25 | 63 ± 7 | 109 ± 11 | 231 ± 44 | |
| | 2–25 | 65 ± 8 | 116 ± 24 | 225 ± 40 | |
| *r*-Tib | 0–1 | 66 ± 240 | 1,166 ± 613 | | *n*= 7 |
| | 0–2 | 59 ± 68 | 563 ± 265 | | *n*= 7 |
| | 0–25 | 69 ± 20 | 123 ± 26 | 191 ± 41 | *n*=12 |
| | 1–25 | 75 ± 9 | 132 ± 23 | 198 ± 30 | *n*= 7 |
| | 2–25 | 78 ± 13 | 109 ± 25 | 161 ± 32 | *n*= 7 |
| AP forces | 0–1 | 269 ± 95 | 991 ± 149 | | |
| | 0–2 | 318 ± 63 | 773 ± 166 | | |
| | 0–25 | 120 ± 16 | 294 ± 61 | 825 ± 321 | *n*=12 |
| | 1–25 | 123 ± 18 | 289 ± 54 | 584 ± 117 | |
| | 2–25 | 152 ± 28 | 296 ± 42 | 504 ± 71 | |

Values are means ± SD, with number of subjects (*n*) contributing to the mean presented at right. The table displays EMG peak correlation latencies and peak anteroposterior (AP) force correlation latencies (in milliseconds) for each of the stochastic stimuli. The 0 to 1 Hz and 0 to 2 Hz trials exhibited biphasic patterns, which are respectively labeled the first and second responses, whereas the 0 to 25 Hz, 1 to 25 Hz, and 2 to 25 Hz trials exhibited triphasic patterns that are respectively labeled early, middle, and late latency responses. Responses in the right tibialis anterior were unreliable in some subjects. *r*-Sol, right soleus; *r*-mGas, right medial gastrocnemius; *r*-Tib, right tibialis anterior.

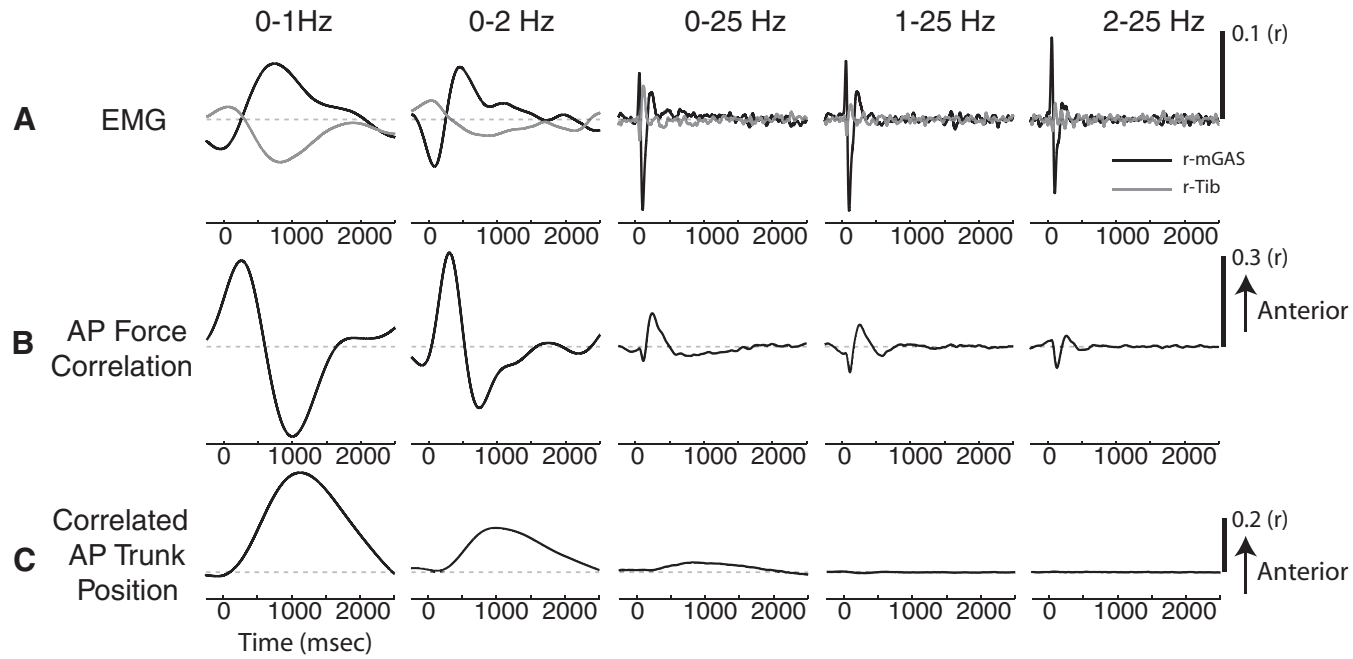


FIG. 3. EMG, force, and trunk position cumulant density estimates elicited by the following stimuli (in Hz): 0–1, 0–2, 0–25, 1–25, and 2–25. *A*: pooled ($n = 12$) muscle responses for the right medial gastrocnemius and ($n = 7$) tibialis anterior. Biphasic muscle correlations were observed for the 0 to 1 and 0 to 2 Hz stimuli, whereas triphasic muscle responses are observed for the 0 to 25, 1 to 25, and 2 to 25 Hz stimuli. *B*: anteroposterior force correlations to the 5 stimulus bandwidths. *C*: trunk position correlations showed SVS-related sway correlation is attenuated as the low-frequency content of the stimulus is removed. The 2 to 25 Hz trial exhibited no observable correlated sway in the cumulant density estimate. Cumulant density magnitude was measured as an *r* value. AP, anteroposterior; *r*-Tib, right tibialis anterior; *r*-mGas, right medial gastrocnemius.

opposite was observed in the right tibialis anterior (Fig. 3). The correlation for the first muscle peak to the 0 to 2 Hz bandwidth was greater than that of the 0 to 1 Hz bandwidth in both the soleus [$t_{(11)} = 4.47$, $P < 0.05$; $-0.020 \pm 0.01$ vs. $-0.009 \pm 0.08$] and medial gastrocnemius [$t_{(11)} = 2.87$, $P < 0.05$; $-0.056 \pm 0.026$ vs. $-0.033 \pm 0.018$]. Magnitudes for the second peak of the biphasic correlation were similar for the 0 to 1 and 0 to 2 Hz bandwidths in the plantar flexors (multiple $P > 0.05$; $0.031 \pm 0.014$ vs. $0.023 \pm 0.015$, *r*-Sol; $0.068 \pm 0.039$ vs. $0.066 \pm 0.033$, *r*-mGas).

### *Force responses*

All of the SVS stimuli evoked significant force responses, as illustrated by the correlations between SVS and Force (Fig. 3). The force correlations to the 0 to 25, 1 to 25, and 2 to 25 Hz stimuli bandwidths were characterized by an initial posterior (negative) peak followed by an anterior (positive) peak in the direction of the anode (see Table 1 for latencies). The magnitude of the early latency correlation for the horizontal anteroposterior (AP) force was 77 and 54% larger for the 1 to 25 and 2 to 25 Hz stimuli, respectively, compared with that of the 0 to 25 Hz stimulus ($-0.057 \pm 0.03$) [$F_{(2,11)} = 57.6$, $P < 0.05$; Fisher LSD, multiple $P < 0.05$], but was similar in magnitude between the 1 to 25 and 2 to 25 Hz stimuli ($-0.101 \pm 0.04$ and $-0.088 \pm 0.05$). In contrast, the magnitude of the middle latency correlation for AP force grew larger as the low-frequency content of the stimuli increased. The 2 to 25 Hz trial exhibited the smallest middle latency peak correlation response ($0.055 \pm 0.05$), followed by a larger response for the 1 to 25 Hz trial ($0.114 \pm 0.05$), with the largest response recorded in the 0 to 25 Hz trial ($0.154 \pm 0.06$) [$F_{(2,11)} = 58.8$ , $P < 0.05$; Fisher LSD, multiple $P < 0.05$].

Stochastic stimuli with bandwidths of 0 to 1 and 0 to 2 Hz were associated with biphasic force correlations (see Table 1 for latencies). The first anterior directed peak correlation was similar [$t_{(11)} = 1.89$ $P > 0.05$] in magnitude for 0 to 1 Hz ($0.310 \pm 0.058$) and 0 to 2 Hz stimuli ($0.347 \pm 0.079$). The second posterior directed peak correlation was 35% larger [$t_{(11)} = 3.23$ $P < 0.05$] for the 0 to 1 Hz ($0.335 \pm 0.074$) stimulus than that for the 0 to 2 Hz ($0.249 \pm 0.078$) stimulus.

### *Trunk sway*

The correlation between trunk sway and SVS decreased or was absent as frequencies <2 Hz were removed from the stimuli (Fig. 3). The SVS stimuli evoked a single trunk sway response in the direction of the anode, peaking at 1,198 ± 178 ms for 0 to 1 Hz, 1,155 ± 266 ms for 0 to 2 Hz, and 1,008 ± 354 ms for 0 to 25 Hz. In contrast, the 1 to 25 Hz stimulus evoked very small biphasic sway responses that were present in only some subjects and were characterized by an early negative correlation at 284 ± 90 ms ($n = 6$) and a later positive correlation in the direction of the anode at 833 ± 381 ms ($n = 4$). The 2 to 25 Hz stimulus was not associated with any apparent sway (Fig. 3). Trunk sway correlations were not formally compared across stimulus conditions due to the inconsistent sway patterns: the responses associated with the 0 to 1, 0 to 2, and 0 to 25 Hz stimuli were monophasic and observed in all subjects; a measurable sway response to the 1 to 25 Hz stimulus was observed in only one third of the subjects; whereas the 2 to 25 Hz stimulus produced no measurable trunk sway in any of the subjects tested. Although both the 1 to 25 and 2 to 25 Hz stimuli appeared to minimize or abolish the prolonged AP sway response observed in the cumulant density function, only the 2 to 25 Hz stimulus did not increase RMS

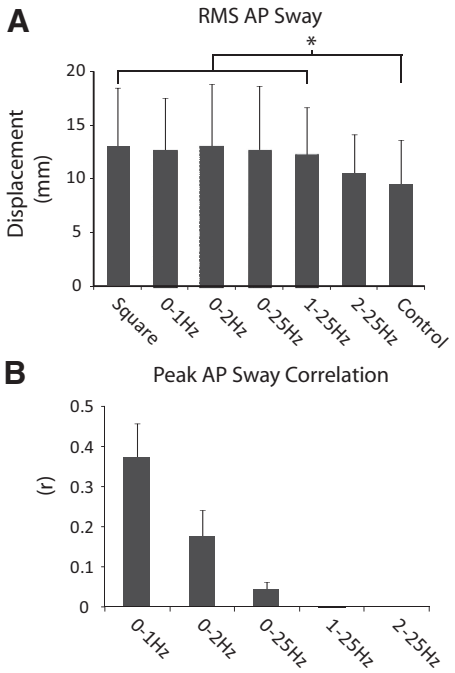


FIG. 4. Root-mean-square (RMS) of AP sway and peak AP sway correlation. *A*: average RMS AP trunk sway for each trial condition. Square trials are 1 mA, 1 s square-wave pulses, whereas participants received no stimulus during control trials. Stimulus bandwidths are labeled on the abscissa. Error bars are SDs. *B*: average peak AP trunk sway correlation as measured on the cumulant density function. Stimulus bandwidths are labeled on the abscissa.

AP trunk sway compared with the no stimulation trial [$F_{(6,11)} = 89.6$ , $P < 0.05$; 12.3 ± 4.4 (1–25 Hz) vs. 9.6 ± 4.0 (Control), Fisher LSD, $P < 0.05$; 10.5 ± 3.6 (2–25 Hz) vs. 9.6 ± 4.0 (Control), Fisher LSD, $P > 0.05$] (Fig. 4). All trials except the 2 to 25 Hz stimulus exhibited increased mean removed RMS trunk sway compared with the control trial (Fig. 4).

### *SVS–sway gain response*

From the initial 0 to 25 Hz SVS stimulus, signal power was successively low-pass filtered at the muscle, then at ankle moment production, and finally at trunk sway (Fig. 5*A*). The gain of the closed-loop transfer function from SVS to EMG exhibits two peaks localized within the 0 to 20 Hz bandwidth: one low-frequency peak (<1 Hz) and a second peak around 4.5 Hz. When the closed-loop transfer function is extended a step further to the SVS to AP ankle moment, the signal gain exhibited a large low-pass filtering effect, decreasing in signal power by 50-fold at 5.5 Hz. Signal power for SVS to AP sway exhibited a 50-fold decrease in power at 1.5 Hz.

Signal transfer from EMG to sway was determined through inferred open-loop transfer functions for EMG to AP moment, EMG to AP sway, and AP moment to AP sway (Fig. 5*B*). The inferred open-loop transfer functions were similar to the closed-loop estimates. Signal power decreased by 50-fold, at 6.5 Hz, from EMG to AP moment and by 40-fold, at 1.9 Hz, from EMG to AP sway. These results suggest that SVS signal power is low-pass filtered by the mechanics of the body as muscle activation is converted into body sway.

## DISCUSSION

The primary aim of the current study was to develop a vestibular stimulus using a specific bandwidth to minimize SVS-related sway responses. The main findings in this study supported our hypotheses: *1*) stimuli with low frequencies removed (1–25 and 2–25 Hz) attenuated or abolished the sway response, whereas multiphasic muscle responses were retained; and *2*) decreases in sway to the 1 to 25 and 2 to 25 Hz stimuli appear to derive from mechanical filtering of the higher-frequency muscle responses as muscle activation is transferred to sway.

Vestibular stimuli at low frequencies (<2 Hz) were mainly associated with prolonged vestibular-related trunk sway, whereas typical muscle responses were evoked by vestibular stimulation excluding these low frequencies. Indeed, removal of the 0 to 1 or the 0 to 2 Hz content from the stochastic stimuli abolished prolonged trunk sway responses to the SVS stimulus, as represented in the time cumulant densities, resulting in a very small or absent residual sinusoidal sway pattern. This reduction of sway in response to bandwidth-limited SVS appears to be the result of a change in the relative magnitude of the early and middle latency components of the correlated AP force peaks (Fig. 3). Although correlated responses between SVS and EMG, or force, have no physical values, they do possess similar spatial and temporal characteristics to the trigger-averaged EMG (Dakin et al. 2007) and force responses (Mian and Day 2009) observed with GVS. Thus the relative area within the correlation is discussed using a framework similar to that of the responses evoked by GVS.

Removing the 0 to 1 Hz bandwidth increased the peak correlation of early latency force response, whereas attenuating that of the middle latency response resulted in each component having nearly equivalent areas. The short time course of the SVS-related early force response and rapid onset of the opposing middle latency force response of similar magnitude provide little time for sway to accumulate, thereby reducing the magnitude of the resulting correlated sway. Similarly, the SVS-related force response to the 2 to 25 Hz bandwidth stimuli exhibits three small peaks occurring over a very short time period, preventing the production of any observable sway response. In contrast, the correlation between force and the 0 to 25 Hz stimulus exhibits a very small short latency component but a larger opposing medium latency component, leading to a prominent sway response in the direction of the medium latency force response. This allows significant sway to be produced by the middle latency force response prior to correction. Regarding the standing balance behavior, GVS pulses (1 mA, 1 s duration) delivered randomly every 15 to 25 s, as well as most stochastic vestibular stimuli bandwidths (0–1, 0–2, 0–25, and 1–25 Hz), increased the variability of trunk sway observed compared with the control of normal standing balance. Only the 2 to 25 Hz vestibular stimuli were not associated with more variable trunk sway in the AP direction (Fig. 4). This is a likely consequence of the minimal amount of horizontal force and the absent trunk sway associated with this stimulation bandwidth.

Frequency-related changes in sway may be explained as a purely mechanical process. By increasing the frequency of the vestibular stimulus, the frequency of the corresponding envelope of muscle activity also increases, requiring higher gain to

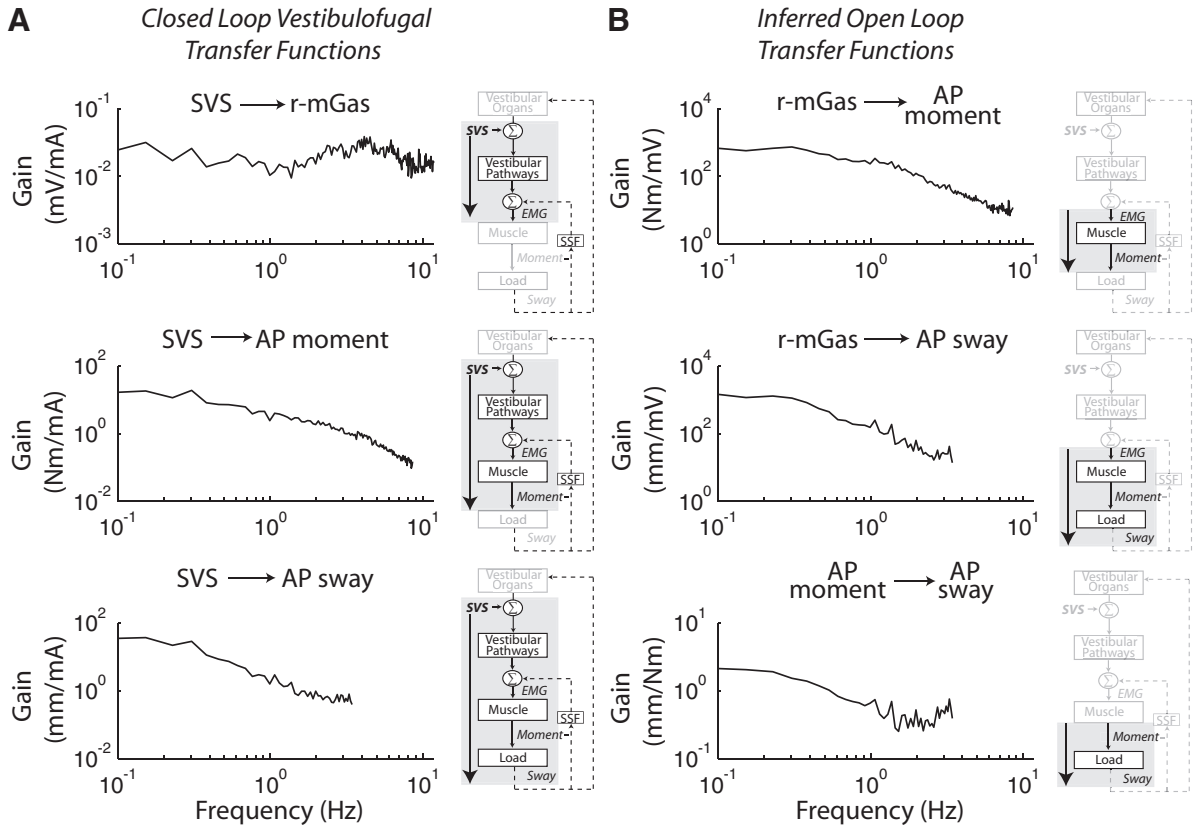


FIG. 5. Gain plots along 6 stages of the SVS-sway relationship. *A*: gain of the closed-loop transfer functions from SVS input to three output measures: EMG muscle response (*r*-mGas), AP moment, and AP sway. SVS–AP moment gain shows a strong filtering effect reducing prominent signal power transfer to frequencies <5.5 Hz. SVS–AP sway gain shows a further reduction in signal power transfer to frequencies <1.5 Hz. *B*: gain of the inferred open-loop transfer functions between EMG (*r*-mGas) and AP moment, EMG (*r*-mGas) and AP sway, and AP moment and AP sway. EMG (*r*-mGas)–AP moment and EMG (*r*-mGas)–AP sway gain functions mirror the SVS–AP moment and SVS–AP sway functions with filtering of signal transfer frequency to <6.5 and <1.9 Hz. AP moment–AP sway gain function shows signal transfer below 1.5 Hz. AP: antero-posterior; anterior; *r*-mGas: right medial gastrocnemius, SSF: somato-sensory feedback.

maintain the impulse necessary to overcome the inertia of the body and induce sway. In contrast, signal transfer gain from stimulus to sway experiences a strong low-pass filtering effect when transferring from net muscle activity to AP trunk sway (Fig. 5). The majority of the filtering effect arises from the conversion of net muscle activity to AP moment reducing signal power to frequencies <6.5 Hz, whereas transfer from AP moment to AP trunk sway further limits signal power to frequencies <2 Hz. Low-pass filtering of muscle activation to force output is a mechanical consequence of intrinsic muscle properties and net motor activity across one or several muscles acting on a single common effector, in this case the ankle joint, resulting in a smoothed force output with a bandwidth usually <3 to 5 Hz (Bawa and Stein 1976; Fitzpatrick et al. 1996; Oleny and Winter 1985). Signal filtering is also observable between AP moment and AP sway (Fig. 5, *A* and *B*), likely attributable to the inertial load of the body resisting applied forces (Fitzpatrick et al. 1996; Latt et al. 2003).

It is conceivable that the CNS specifically encodes for the mechanical filtering of the vestibular–sway pathway. Identifying the locus of potential neural structures representing the mechanical filtering described here cannot be performed in awake humans but experiments in animals may provide potential mechanisms. For example, animal models revealed that sinusoidal electrical vestibular stimulation affects vestibular afferents (albeit predominantly the irregular ones) and the second-order vestibular neurons at stimulation frequencies ≤100 Hz (Goldberg et al. 2004; Kleine and Grusser 1996). Once in the fastigial nucleus, however, two distinct populations of vestibular-related neurons have been described: one group that responds to low-frequency (<1 Hz) sinusoidal vestibular stimulation and another group that is tuned toward frequencies ≤10 Hz (Schlosser et al. 2001). The fastigial nucleus contains vestibular-related neurons that respond to vestibular signals with the appropriate coordinate transformation necessary to elaborate motor commands appropriate for whole-body responses (Brooks and Cullen 2009; Kleine et al. 2004; Manzoni et al. 1999). These behaviors are similar to the vestibular-evoked muscle and sway responses that show well-defined spatial transformations related to the position of the head relative to the feet (Britton et al. 1993; Fitzpatrick et al. 1994; Lund et al. 1983; Mian and Day 2009) and distinct low- and higher-frequency response characteristics to vestibular stimuli (as described here). Although it is possible that the nervous system and potentially cerebellar nuclei represent or independently code the mechanical dissociation between muscular and whole-body responses, additional experiments are required to test this hypothesis for balancing actions.

Removal of the low-frequency content of the stochastic stimuli enlarges the early latency component of the correlated AP force response that is applied to the body, suggesting that SVS void of frequencies <1 or 2 Hz may be an effective way

to investigate the early latency response. Traditionally, the early and middle latency vestibular-evoked force or muscle responses have been described as independent entities (Britton et al. 1993; Cathers et al. 2005)—the early latency response has been suggested to originate from the otoliths and travel via the reticulospinal pathways, whereas the middle latency response originates from the semicircular canals and travels via the vestibulospinal pathways. The early and middle latency peaks also appear to be contributed to by stimuli of different frequency content. The early latency peak is strongly contributed to by stimuli with higher-frequency content (Dakin et al. 2007; Rosengren and Colebatch 2002), whereas the middle latency response is largely influenced by stimuli with increased low-frequency content (Britton et al. 1993; Dakin et al. 2007; Rosengren and Colebatch 2002). Physiologically, there is some evidence to suggest that low-frequency natural stimuli are transduced somewhat differently, mainly through regular firing vestibular afferents, than higher-frequency natural inputs, which are transmitted through both regular and irregular firing vestibular afferents (Sadeghi et al. 2007). Thus catering stimulus bandwidth to evoke a response component of interest (early or middle latency response) may be an important attribute of SVS, enabling further examination of the often difficult to isolate early latency response with less confounding interference from the middle latency response.

### *Functional relevance*

SVS could provide an ideal tool for examination of lower-limb vestibular-evoked responses in patient populations. First, SVS requires relatively short stimulus durations to evoke prominent EMG and forces responses: 180 s total stimulus duration in Dakin et al. (2007) and 133 s trials in this study compared with 300 s for square-wave GVS in Dakin et al. (2007) and between 260 and 300 s, for 20 pulses, in this study. Second, if induced sway resulting from vestibular stimulation confounds interpretation of results or is unwanted when assessing vestibular function in patients with a balance disorder (Liechi et al. 2008; Marsden et al. 2005; Pastor et al. 1993), the stimulus may be tuned to reduce unwanted sway responses by excluding frequencies $<2$ Hz. Third, stimulus bandwidths may also be tuned to study a particular physiological response such as the early (1–25 or 2–25 Hz) or middle (0–25 Hz) components of the vestibular-evoked responses. This could prove particularly important to investigate the possible mechanisms proposed to contribute to the vestibular-evoked balance phenomenon: otoliths and semicircular canals (Cathers et al. 2005) or independent spinal pathways (Britton et al. 1993).

### *Limitations*

A limitation regarding interpretation of this study is that the use of narrow-bandwidth, low-frequency stimuli (0–1 and 0–2 Hz) results in correlations in the cumulant density occurring prior to the zero lag point. Prior to zero, correlations occur with narrow bandwidths because the random nature of the stimulus is reduced, creating a quasi-sinusoidal stimulus that, much like a sine wave, will experience some correlation with the waveform's previous period along with the current one (Matthews 1993). This prior to zero correlation confounds interpretation of the timings of the associated vestibular-related response peaks but can be avoided by using vestibular stimuli with large bandwidths (e.g., 0–25, 1–25, or 2–25 Hz).

### *Conclusion*

Removal of the low-frequency content of the SVS stimulus resulted in attenuation of vestibular-evoked sway responses and an increased amplitude of the early latency vestibular-evoked force response. These results demonstrate that SVS with a bandwidth containing frequencies $>2$ Hz may be used to provide a vestibular stimulus that does not cause a destabilizing sway response. Overall, SVS provides multiple benefits over GVS: it is more comfortable, can elicit similar responses in a shorter stimulation period, and its parameters may be tuned to limit prolonged sway responses or assess specific components of the muscle or balance behavior.

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