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Head-Trunk Coordination During Linear Anterior-Posterior Translations

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Keshner, Emily A. Head-trunk coordination during linear anterior-posterior translations. *J Neurophysiol* 89: 1891–1901, 2003; 10.1152/jn.00836.2001. The purpose of this study was to evaluate the relative contributions of inputs from the vestibular system and the trunk to head-trunk coordination. Twelve healthy adults and 6 adults with diminished bilateral labyrinthine input (LD) were seated with their trunk either fixed to the seat or free to move. Subjects received 10-cm, 445-cm/s^2 anterior-posterior ramps and 0.35- to 4.05-Hz sum-of-sines translations while performing a mental distraction task in the dark. Kinematics of the head and trunk were derived from an Optotrak motion analysis system and a linear accelerometer placed on the head. EMG signals were collected from neck and paraspinal muscles. Data were tested for significance with multivariate ANOVA (MANOVA) and Bonferroni post hoc analyses. Initial linear and angular head acceleration directions differed in healthy subjects when the trunk was fixed or free, but did not differ in LD subjects. Peak head angular accelerations were significantly greater with the trunk fixed than when free, and were greater in LD than in control subjects. EMG response latencies did not differ when the trunk was fixed or free. Low-frequency phase responses in the healthy subjects were close to 90° and had a delayed descent as frequency increased, suggesting some neural compensation that was absent in the LD subjects. Results of this study revealed a strong initial reliance on system mechanics and on signals from segmental receptors. The vestibular system may act to damp later response components and to monitor the position of the head in space secondary to feedback from segmental proprioceptors rather than to generate the postural reactions.

INTRODUCTION

Despite a great deal of research attempting to characterize the contribution of the vestibular reflexes to stabilization of the head in space during active motion, it is still unclear how and when those reflexes participate (Gresty 1987; Guitton et al. 1986; Keshner and Peterson 1995; Keshner et al. 1995). One limitation to completely characterizing vestibular mechanisms is that the head must be locked to the trunk to isolate the actions of the vestibular reflexes. Once the head has been released from the trunk, other controllers, such as voluntary mechanisms, neck proprioception, and passive mechanical (i.e., inertial, viscous, and elastic) properties of the neck muscles could assist in returning the head to its upright position in space and may even become the primary controllers of this action (Bizzi et al. 1978; Forssberg and Hirschfeld 1994; Horak and Hlavacka 2001; Keshner et al. 1999; Massion et al. 1995; Mergner et al. 1997; Vernazza et al. 1996).

Using a simple model of the head and neck with parameters representing musculoskeletal geometry and soft tissue of the head and neck, Viviani and Berthoz (1975) suggested that active modulation of neck muscle properties could provide the torques necessary to counter force disturbances thereby minimizing motion of the head in space. Feedforward control of the trunk (McFadyen et al. 1994; Wu et al. 1998), possibly determined by individual morphology (Vibert et al. 2001), may reduce ascending disturbances to the head so that the head could maintain its position in space. It might also be that active and passive mechanics of the trunk completely damp any forces arising from the lower body so that the passive mechanical properties of the neck provide sufficient muscle/tissue stiffness and damping forces to minimize motion of the head with respect to the body (Allum et al. 1993; Cappozzo 1981; Nashner 1985). Or, acceleratory forces on the head could elicit the vestibular reflexes which act primarily to maintain position of the head in space while the trunk is moving. During walking, head pitch rotation was found to be highly coherent with trunk linear acceleration (Hirasaki et al. 1999), and it was proposed that the angular vestibulocollic reflex (VCR) was responsible for keeping the head stable in space during low-frequency translations of the trunk while the linear VCR acted at higher frequencies.

This study examined the kinematic properties of the head with respect to the trunk during linear translations to evaluate the relative contributions of inputs descending from the vestibular system or ascending from the trunk to head-trunk coordination. Responses from individuals with labyrinthine deficit (LD) were compared with those of healthy adults to further distinguish the role of the vestibular reflexes.

METHODS

Subjects

Subjects in this study were 12 healthy young adults (age, 22–33 yr; mean age, 25 yr) and 6 patients with diminished bilateral labyrinthine input (LD: age, 34–71 yr; mean age, 55 yr) who gave informed consent according to the guidelines of the Institutional Review Board of Northwestern University Medical School. The healthy subjects had no history of central or peripheral neurological disorders or problems related to movements of the spinal column (e.g., significant arthritis or musculoskeletal abnormalities).

Bilateral LD was determined in the patient population by an otoneurologist and was defined as a positive dynamic illegible "E" test (Longridge and Mallinson 1987) and a vestibuloocular reflex gain

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<0.4 to 0.32 Hz sinusoidal yaw rotations in the dark. All patients were several years past onset of symptoms, and all but one had participated in a physical therapy rehabilitation program. The remaining subject was a young (34 yr old) active male who participated in sports. Detailed descriptions of each of the patients are in Table 1.

Apparatus

A linear accelerator (sled) that could be translated in the anterior-posterior (a-p) direction was situated within a light-tight room. Motion of the sled was controlled by D/A outputs from an on-line Macintosh computer. Subjects wore a thin, plastic, molded helmet, firmly secured to the head (Fig. 1). A triaxial linear accelerometer (Entran Devices, Fairfield, NJ) was attached to the side of the helmet about the level of the auditory meatus, which corresponded to the theoretical axis of rotation between the head and the upper part of the cervical spine (C_1-skull). However, because the cervical spine has multiple axes of rotation, predominantly at both C_1 and T_1, and because the accelerometer was not oriented with respect to gravity for all potential axes of motion, there was a potential for the up-down accelerations to reflect cross-talk with the anterior-posterior accelerations and the angular motion of the head. Therefore only the anterior-posterior and left-right directions will be presented here. The weight of the entire helmet and attachments was 485 g, with the center of mass located on the vertical center line and lower one-third of the helmet. The helmet was light compared with the mass of the head (typically 4.0 kg), and the helmet center of mass was at the same position as the center of mass of the head; thus the helmet did not significantly affect head movement.

Stimuli

Position ramp stimuli consisted of a series of six position ramps that started from a neutral position, held for 3–4 s, and returned to the neutral position and was held for a period of 8–12 s. Every 82-s waveform consisted of two 2.5-cm translations with an average peak acceleration (across subjects) of 122 ± 4 (SD) cm/s^2, two 5-cm translations (243 ± 7 cm/s^2), and two 10-cm translations (445 ± 13 cm/s^2), each lasting approximately 400 ms. Translations were randomized for excursion and direction (fore-aft).

A sum-of-sines (SSN) position command that consisted of relatively prime (i.e., having no common divisors) harmonics of a common base frequency was composed of frequencies ranging from 0.35 to 4.05 Hz to obtain the dynamic frequency response characteristics of the head control mechanisms. SSN component frequencies were chosen so that the first, second, and third harmonic of each component did not match the frequency of any other component. Sled velocity was held around 5 cm/s across most frequencies except at 3.05 Hz, where the dynamics of the sled required an increase to approximately 7 cm/s. Amplitude decreased and linear accelerations increased with frequency (Table 2).

TABLE 1. *Clinical characteristics of labyrinthine deficient subjects*

Subject	Age-Sex	Diagnosis (no. years past onset)	0.32 Hz VOR Gain
1	34-Male	Vestibular neuritis—acoustic neuroma (5)	0.05
2	42-Male	Idiopathic (15)	0.28
3	50-Male	Idiopathic (2)	0.13
4	65-Female	Gentamicin toxicity (4)	0.20
5	68-Male	Gentamicin toxicity (3)	0.28
6	71-Male	Idiopathic (3)	0.05

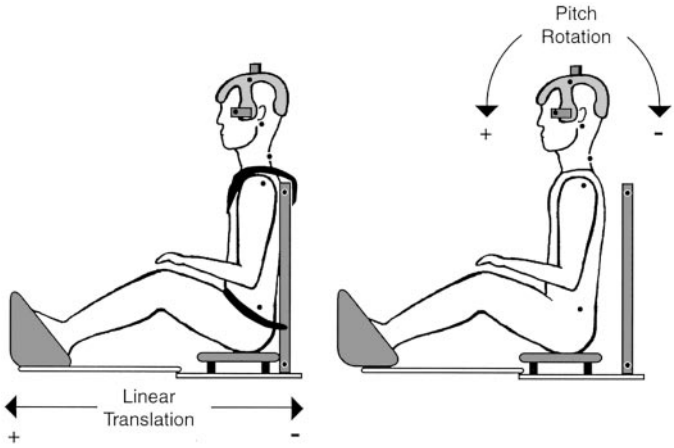

FIG. 1. Schematic of the experimental paradigm illustrating the position of the subject with the trunk fixed (*left*) and trunk free to move (*right*). Locations of the Optotrak markers (●) are shown for the head, C_7, acromion process, tubercle of the iliac crest, and sled. The linear accelerometer (rectangular box) is placed on the helmet over the ear. Sign conventions for sled linear translation (anterior is positive, posterior is negative) and head and trunk angular acceleration (pitch downward rotation is positive, pitch upward rotation is negative) directions are shown.

Data collection

Three-dimensional (3-D) kinematic data from the head and trunk were collected at 135 Hz using 3-D video motion analysis (Optotrak, Northern Digital, Ontario, Canada). Optotrak data were sent to a dedicated Macintosh computer for later analysis of relative head and trunk position. Infrared markers placed on the helmet near the lower border of the eye socket and the external auditory meatus of the ear were used to define the Frankfort plane and to calculate head angular position relative to the earth vertical. The auditory meatus corresponds to the relative axis of rotation between the head and the upper part of the cervical spine (C_1-skull). Other markers were placed on the back of the neck at the level of C_7, the acromion process, and the tubercle of the iliac crest. C_7 corresponds to the relative axis of rotation between the cervical spine and the trunk. Markers placed at C_7 and the iliac crest were used to calculate trunk position relative to earth vertical. Frankfort re trunk (head angular position relative to body) was calculated as the relative angle between the Frankfort plane and the trunk segment. Markers placed on the seat recorded chair position. *X-Y-Z* coordinates of each anatomical marker and the sled position signal were collected. Segmental angles were calculated with respect to an inertial coordinate system fixed on markers placed on the sled at neutral position (preperturbation). Triaxial linear accelerometers were also placed on the helmet and on the back of the moving platform to directly record the magnitudes of head and sled linear acceleration.

Electromyographic data were collected from the right semispinalis capitis (SEMI), sternocleidomastoid (SCM), thoracic (THOR), and lumbar (LUM) paraspinal muscles with pairs of active parallel bar electrodes (Delsys, Boston, MA). Neck and trunk muscle electrode

TABLE 2. *Sled motion parameters during the SSN stimulus*

Frequency (Hz)	Amplitude (cm)	Acceleration (cm/s^2)
0.35	2.2	11
0.55	1.4	17
0.85	0.9	27
1.20	0.6	36
1.45	0.5	44
2.15	0.4	65
3.05	0.3	92
4.05	0.14	58

placements have been anatomically and physiologically verified in earlier studies (Keshner et al. 1989; Vasavada et al. 1998). Raw EMG and acceleration data were filtered through identical analog low-pass filters (8-pole, Bessel) with a 500-Hz cutoff frequency, immediately before analog to digital conversion at 1,644 Hz. Rectification and low-pass filtering was done digitally.

Procedures

Subjects sat on a raised bench (24 cm above the moving platform) with arms crossed, knees slightly flexed, and feet strapped into plastic boots riding with the sled platform (Fig. 1). During the trunk free trials subjects held themselves erect. During the trunk fixed trials a cushioned plate was fixed behind the bench and subjects were securely strapped to the plate with shoulder and lap belts with only the head unrestrained.

In the first trial of each trunk fixed and trunk free series, the subject selected the position of the head that felt comfortably erect and centered over the trunk. The positions of the markers on the lower border of the eye socket and the meatus of the ear were recorded, and that position was defined as initial head position for that subject and replicated at the start of each trial.

Subjects performed a mental activity task in the dark to eliminate or reduce voluntary responses by keeping their attention focused on something other than the postural disturbance. Subjects were asked to mentally compile a list of objects such as "cities beginning with A" or "items you would find in a hardware store." Subjects received three ramp stimulus trials and one SSN trial with the trunk fixed and free for a total of eight trials. The final trial of each experiment was for recording resisted active contractions of the neck and trunk muscles. With the sled in a fixed position, an experimenter stood behind the seated subject, and the subject was asked three times to push their head into extension and flexion and their trunk into extension as the experimenter resisted their motion.

Data analysis

For position ramp data, all raw data were processed and analyzed using the mathematical analysis software Matlab (Math Works, Natick, MA). EMG data were examined in individual ramp trials and averaged within subjects. Area under the curve of each EMG signal was measured for a 30-ms period, using Simpson's Rule (Gerald and Wheatley 1999), following the onset of the initial EMG response defined as the first peak rising 2 SD above resting level activity. This value was then divided by the peak amplitude in the maximal contraction trial and scaled by 1,000 to determine the relative ratio of EMG response to resisted voluntary contraction. The latency of the first peak of the EMG response was determined from the onset of acceleration of each position ramp.

The onset latency of the first change in head and trunk angular position was calculated from the onset of acceleration of the position ramp that was when the sled acceleration, calculated from the unfiltered position signal, rose significantly above the baseline. Latencies to the first response and to the peak minimum or maximum response of the head and trunk were calculated from the onset of acceleration of the position ramp. The marker positions were low-pass filtered at 30 Hz with a Butterworth filter. The position data were double differentiated, and maximal angular acceleration of the head and trunk was calculated as the differential value between the minimum and maximum response peaks.

For frequency domain data, amplitude and phase (orientation) of linear sled position, head angular position, trunk angular position, and EMG responses were calculated with a Fast Fourier Transform (FFT). FFT components at frequencies corresponding to the SSN component frequencies described the input and output amplitudes and phases. Sled linear position was considered to be the input to the head and trunk sensory and mechanical systems. Frankfort plane angular position responses were analyzed as the output. Head response output with respect to the sled input was described by the output to input amplitude ratio (gain) and output to input phase difference. Phases of 0° indicate that the output is in phase with the input (i.e., direction of motion of output has the same sign as the direction of input). Phases of $\pm 180°$ indicate that the output is moving out of phase with the input (i.e., if the sled is moving in a positive direction, then the sign of the head and trunk would be negative). Magnitude squared coherence was calculated to provide a measure of system linearity and to determine the presence of noise in the response. Angles at each segment along with the stimulus were plotted relative to time, and root mean square (RMS) values were calculated for the head and trunk in space. Values of the head and trunk RMS were then used to form a ratio to indicate the amount of power at all frequencies with respect to the trunk. The ratio of the RMS values should illustrate the relative extent of motion of the head with respect to the trunk.

Data were tested for significance with a $2 \times 3 \times 2$ (population $\times$ excursion $\times$ trunk fixed/free) multivariate ANOVA (MANOVA) with repeated measures for each dependent variable (i.e., EMG latency, EMG amplitude, and head and trunk angular acceleration latency and magnitude) in each ramp direction. Additional MANOVAs were performed on the SSN data with gain and phase of the head and trunk angular acceleration and the EMG response as dependent variables. Bonferroni post hoc comparisons were performed at the $P < 0.05$ level. RMS responses of the head, the trunk, and head with respect to trunk when the trunk was free to move were compared within and across populations with *t*-tests with a significance level of $P < 0.01$ corrected for multiple tests.

RESULTS

Linear responses to ramp translations

Subjects were tested with three excursions and accelerations of the sled. Although onset latencies of muscle EMG responses were not significantly affected by the acceleration of the translations, the muscles responded sporadically at the smaller amplitudes and velocities of sled motion. In fact, robust and consistent responses were exhibited only at the highest translation acceleration, while angular accelerations of the head and trunk demonstrated an almost linear increase with increasing sled velocities (Fig. 2*B*). Thus to increase the reliability of data interpretation, this paper will focus exclusively on the responses to the 10-cm, 445-cm/s^2 translations.

Linear acceleration trajectories from the accelerometer placed over the external auditory meatus are shown for one healthy and one LD subject (Fig. 2*A*). These traces demonstrate the earliest accelerations of the head with respect to sled translation and provide some measure of the response to the initial input received by the vestibular labyrinths (Forssberg and Hirschfeld 1994). Acceleration trajectories primarily occurred in the a-p direction (the sensor was not working in the left-right direction for the LD subjects, but data from the Optotrak was consistent with the assumption of no significant motion occurring in that direction). Although linear accelerations also appeared in the up-down directions, these were probably contaminated by the a-p accelerations, because the accelerometer was not oriented with respect to gravity or located at all potential axes of cervical motion.

Responses of the LD and healthy subjects were not dramatically different except for larger response magnitudes in the LD subjects that appeared during the period of sled deceleration (between 200 and 400 ms following translation onset). In the

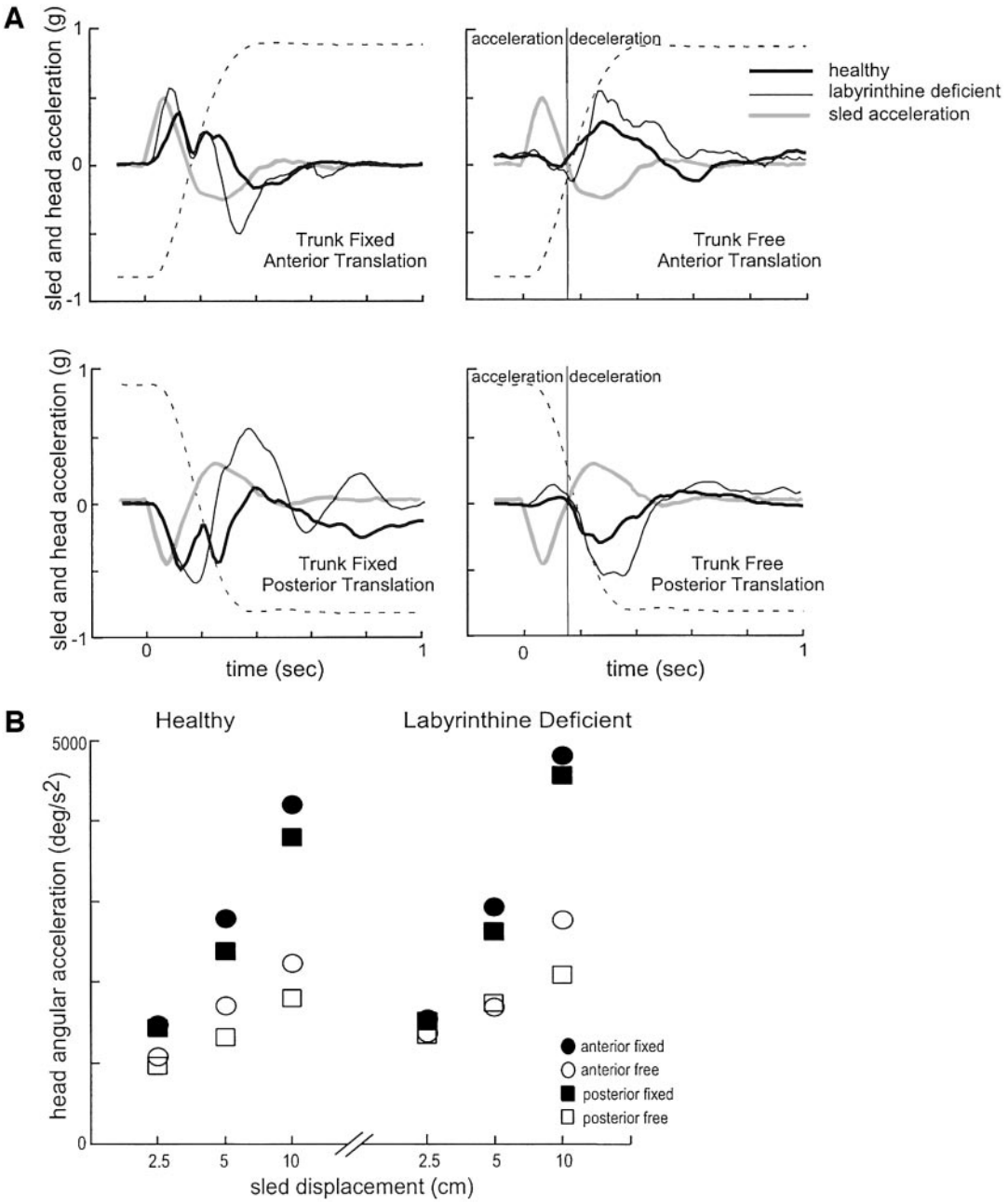

FIG. 2. *A*: linear acceleration traces of the head in the anterior-posterior direction in 1 healthy young adult (bold line) and 1 labyrinthine-deficient (LD) subject (thin line) during the 4 trials with ±10-cm ramp translations. Positive values represent the forward direction. Sled excursion (dashed line) and sled linear acceleration (gray line) are superimposed on each plot. The vertical line on the trunk free plots is a demarcation between sled acceleration and deceleration. *B*: scatter plot of mean peak head angular accelerations across subjects demonstrates a linear trend as sled acceleration increases. Each symbol represents 1 experimental condition and is plotted at the 3 sled excursions for the anterior and posterior directions of translation with healthy subjects (*left*) and LD subjects (*right*).

a-p direction, the primary difference in the response to trunk fixed and free was an early peak of head linear acceleration (about 25 ms following onset of translation) that mirrored the direction of sled acceleration when the trunk was fixed, but was absent when the trunk was free. With the trunk free, the first linear acceleration response of the head was in the direction opposite that of the sled. With sled deceleration, the head accelerated about 50–125 ms after onset of translation in the direction opposite the sled when the trunk was both fixed and free.

Initial angular responses to ramp translations

With the trunk fixed, the first observable change in angular motion appeared in all subjects as a small (about 5°), short-latency (Table 3), angular displacement of the trunk with the opposite sign to that of the sled translation (Fig. 3). Any motion of the trunk was probably due to an inability to completely restrain the mass of the trunk, permitting a lag due to inertia. The smaller mass of the head also exhibited angular motion opposite to the sign of sled translation, but with longer laten-

TABLE 3. *Mean latencies of the onset of head and trunk angular excursions during 10-cm anterior and posterior translations*

	Anterior Translations		Posterior Translations	
	Fixed trunk	Free trunk	Fixed trunk	Free trunk
Healthy subjects				
HEAD*	41 ± 9	66 ± 20	44 ± 11	65 ± 14
TRUNK†	15 ± 6	32 ± 13	21 ± 10	40 ± 10
Labyrinthine deficient				
HEAD*	49 ± 20	69 ± 17	38 ± 14	71 ± 45
TRUNK‡	35 ± 16	31 ± 13	16 ± 6	30 ± 13

Values are mean ± SD (ms). * Anterior translations significantly shorter with fixed trunk than with free trunk [$F(1,34) = 11.68$, $P < 0.001$]. Posterior translations significantly shorter with fixed trunk than with free trunk [$F(1,34) = 5.82$, $P < 0.02$]. † Anterior translations significantly shorter with fixed trunk than with free trunk [$F(1,34) = 5.82$, $P < 0.02$]. Posterior translations significantly shorter with fixed trunk than with free trunk [$F(1,34) = 20.23$, $P < 0.0001$]. ‡ LD significantly shorter than healthy subjects with posterior translations [$F(1,34) = 4.34$, $P < 0.05$].

cies than the trunk and with magnitudes two to three times that of the trunk. Thus the head rotated upward (negative direction) as the sled translated anteriorly and downward (positive direction) as the sled translated posteriorly when the trunk was fixed. Onset of head angular motion was significantly earlier when the trunk was fixed than when free (Table 3).

When the trunk was free to move (Fig. 4), the sign of the initial angular response of the trunk was again opposite to the sign of sled translation. As seen previously (Vibert et al. 2001), motion of the trunk always preceded that of the head. In the healthy subjects, the sign of angular motion of the head was the same as the sign of sled translation and opposite that of the trunk angular motion. Thus for anterior sled translation, the trunk rotated upward and the head rotated down. With posterior translations, the trunk rotated down and the head rotated up. In the LD subjects, the sign of angular motion of the head was in the same direction as trunk angular motion, i.e., both head and trunk rotated upward with anterior translations and rotated down with posterior translations. The timing (Table 3) and direction of the onset of head angular motion suggested a head that was lagging and moving counter to the trunk in the healthy subjects, and lagging but moving with the trunk in the LD subjects.

Peak angular responses to ramp translations

The latency-to-peak angular acceleration (Table 4) revealed significantly longer latencies of the trunk when the trunk was free to move than when the trunk was fixed. Trunk angular acceleration magnitudes did not change (Fig. 5) between trunk fixed and free, suggesting that the trunk was matched to the acceleration of the sled in both conditions. For anterior translations, peak head angular acceleration preceded that of the trunk. With posterior translations, peak head angular acceleration more often followed that of the trunk. Amplitudes of the minimum to maximum peak head angular acceleration response (Fig. 5) demonstrated that head angular acceleration was significantly greater with the trunk fixed. Peak-to-peak head angular acceleration amplitudes were also significantly greater in the LD subjects than in the healthy subjects.

EMG responses to ramp translations

A burst of EMG activity at the onset of sled movement often appeared in the neck muscle responses of the healthy subjects (see SCM and SEMI in Figs. 3 and 4). This latency was not included in the group mean; the next burst was selected as the primary response latency for these muscles. Possible mechanisms generating the early burst of activity will be discussed later.

EMG response latencies with respect to the onset of the stimulus were not significantly affected by whether the trunk was fixed or free in the healthy subjects (Figs. 3 and 4; Table 5), although the latency of head motion differed in the two conditions. During anterior translations, a backward lag of the head due to inertia should occur. If the VCR were acting to maintain the position of the head in space, SCM should exhibit an early response to the accelerations acting on the head. Indeed, when the trunk was free, latencies between head angular acceleration onset and the SCM EMG response (compare Tables 3 and 5) were small (<20 ms). However, when the trunk was fixed, these latency differences doubled and tripled because of the significantly shorter latencies of head angular acceleration with a fixed trunk. If the VCR were active during posterior translations, SEMI should exhibit an early response to the accelerations acting on the head. A similar pattern emerged where latencies between head acceleration and SEMI EMG onset were <20 ms with the trunk free, but much greater (30–40 ms) when the trunk was fixed. Differences between the onset of trunk angular acceleration and the latency of the trunk extensor (THOR and LUM) EMG responses were less variable, ranging from 30–50 ms across both conditions and populations in both directions of translation (compare Tables 3 and 5).

Only three of the LD subjects had consistent and identifiable EMG responses; thus this analysis cannot be extended to a LD population in general. These three subjects were the oldest of the group (ages 65, 68, and 71 yr), and increased activation of their neck muscles may reflect an aging process rather than result from the LD. When the LD subjects did respond, latencies of the EMG responses to onset of sled translation were fairly stable across all conditions (Figs. 3 and 4; Table 5). This meant that, like the healthy subjects, their EMG onset occurred much later than onset of head angular acceleration when the trunk was fixed, but much closer to the onset of head angular acceleration when the trunk was free (compare Tables 3 and 5). There were no significant differences in onset latencies between the two groups. Significant population differences were apparent in the amplitude of the muscle EMG responses, however (Table 6), where the LD subjects that did respond demonstrated significantly larger SCM responses than did the healthy subjects.

Frequency characteristics of the response

There were no dramatic differences between the two populations in response to a random SSN stimulus. When the trunk was fixed, LD subjects exhibited lower gains and phases closer to 0° for the head with respect to the sled at frequencies below 1 Hz (Fig. 6*A*). In the healthy adults, the head lagged the sled by 90° at frequencies below 1 Hz. Because the trunk was not perfectly "fixed" to the sled, it moved approximately 90° out of phase with the sled, which would mean that the head was 180°

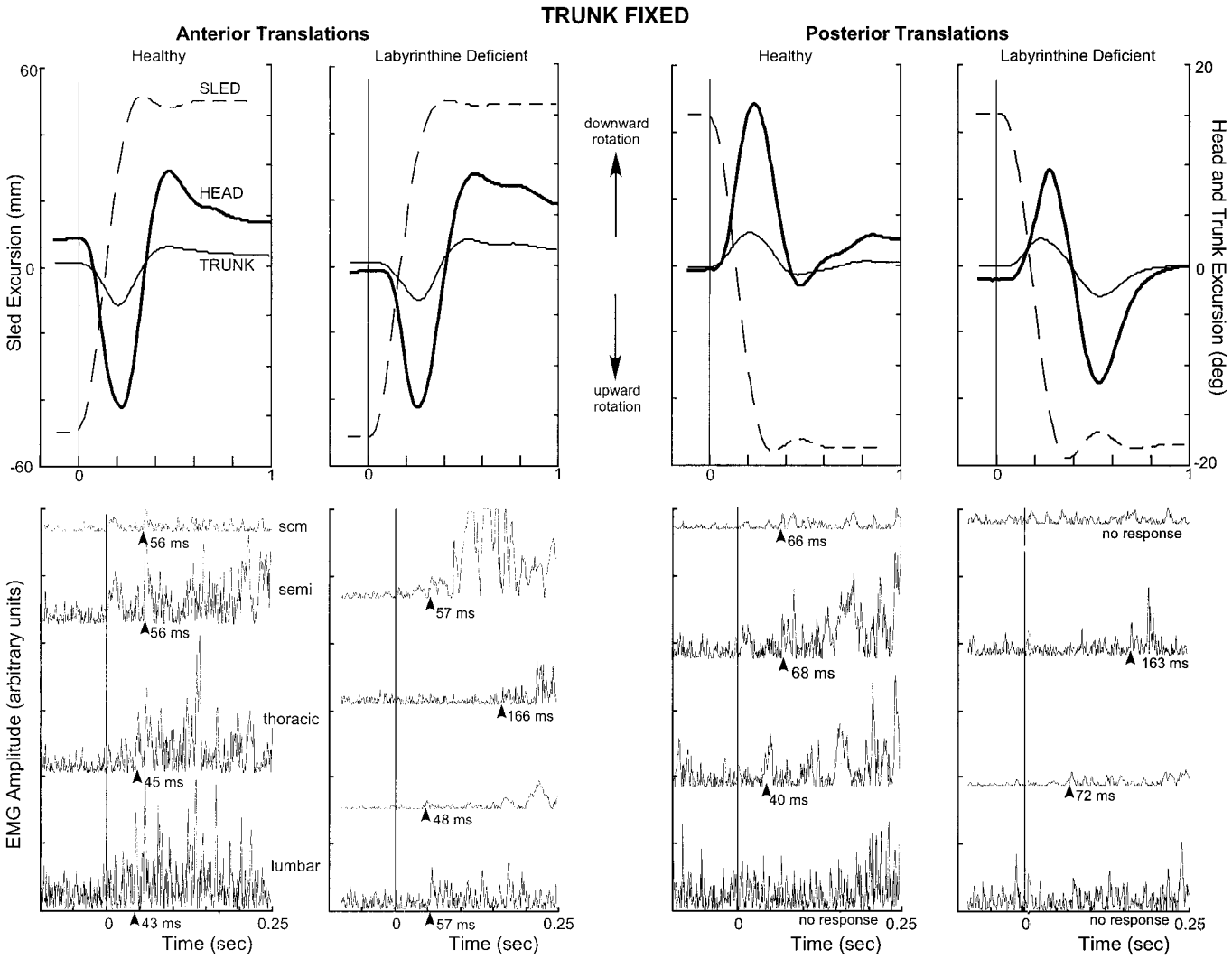

FIG. 3. *Top*: response to 10-cm ramp translations of 1 healthy young adult and 1 LD adult to anterior and posterior translations with the trunk fixed. *Top row*: sled excursion relative to time (broken line) is plotted on the *left y* axis. Head (bold line) and trunk (thin line) angular position are plotted relative to time (*x* axis) on the *right y* axis. The thin vertical line indicates onset of sled translation. *Bottom*: raw muscle EMG responses are plotted for 100 ms before and 250 ms following the onset of sled translation (thin vertical line). EMG responses were normalized to the largest response of each muscle across the experimental conditions. Sternocleidomastoid (scm) is the *top trace*, followed by semispinalis capitis (semi), thoracic, and lumbar paraspinals. Onset latencies for each response are indicated by the black arrowhead.

out of phase with, or moving counter to, the trunk in the healthy subjects. The low gains and phases near 0° in the LD subjects would suggest a head that was moving with the sled and potentially lagging the trunk. Phase responses of the head with respect to the sled descended to 180° above 1 Hz in both groups. When the trunk was free (Fig. 6*B*), response gains were very similar for the two groups, but a 90° phase difference between the two groups was still evident at low frequencies.

Both populations demonstrated an effect of trunk motion on the gains and phases of the head with respect to the sled. When the trunk was fixed, response gains exhibited a rise in gain across the frequency range and approximately a 180° phase shift. Some healthy subjects exhibited a flattening of the gain responses at frequencies below 1 Hz, and some LD subjects exhibited gain plateaus around 1 Hz. When the trunk was free, response gains were lower than with trunk fixed and all subjects exhibited a sudden, dramatic drop off of the response gains at frequencies above 2 Hz. Response phases were level in the healthy subjects below 1 Hz and then descended steeply in both groups across the frequency range, resulting in large phase shifts that crossed 0° around 2–3 Hz and then continued to descend toward −180°.

In the healthy adults, the mean head re trunk ratio across all frequencies was close to one (RMS ratio = 1.07 ± 0.3). Head angular motion in the healthy subjects was significantly greater when the trunk was fixed than when the trunk was free [$F(1,35) = 7.22$, $P < 0.01$]. In the LD subjects, head amplitudes were about 25% greater than the trunk (RMS ratio = 1.28 ± 0.3). In all cases, coherence values were close to unity, indicating that head motion was well described by this relationship.

DISCUSSION

This study examined the kinematic properties of the head with respect to the trunk during linear translations to begin to

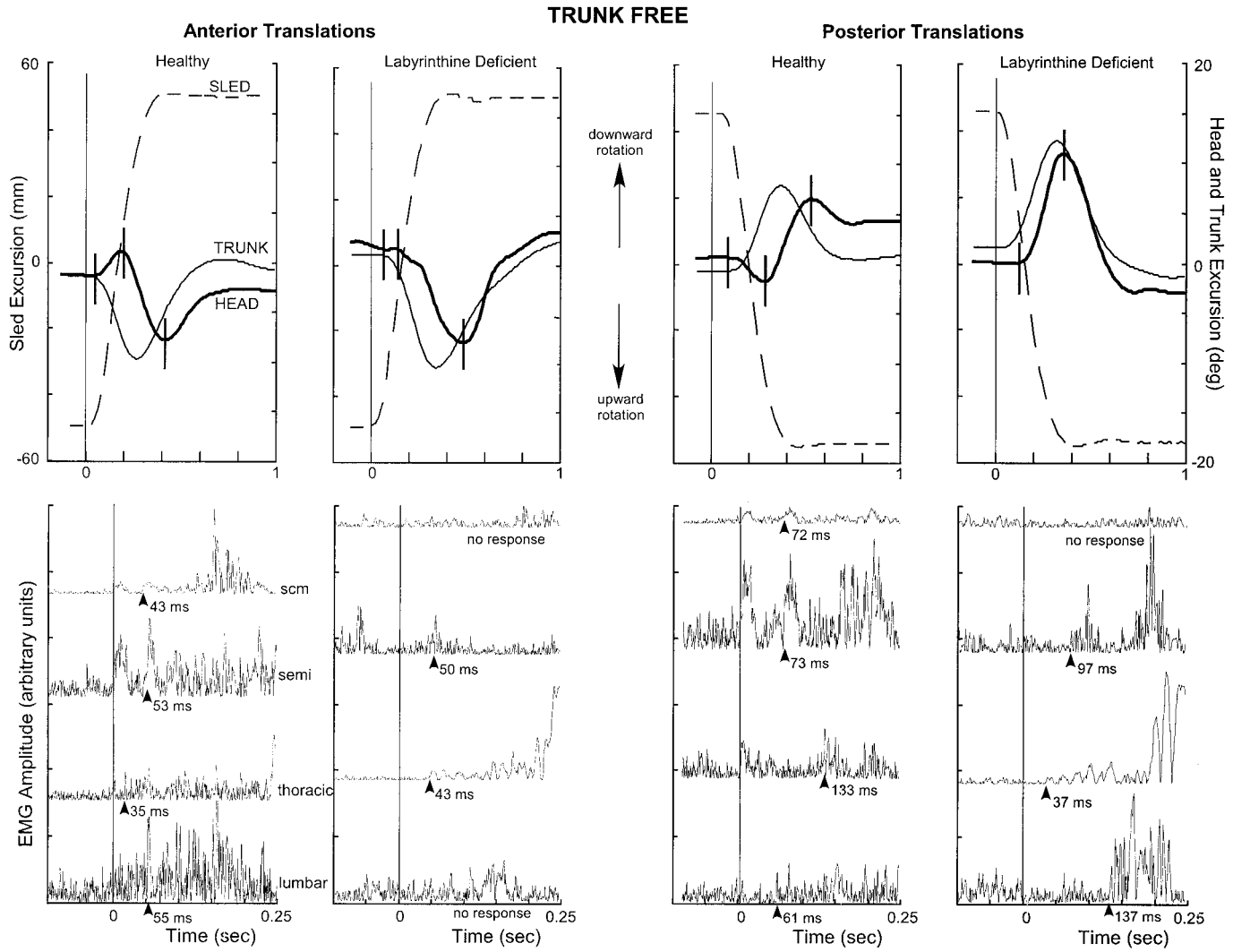

FIG. 4. *Top*: response to 10-cm ramp translations of 1 healthy young adult and 1 LD adult to anterior and posterior translations with the trunk free. Plot layout is the same as in Fig. 3*A*. Short lines on the head trace approximate where the latencies and magnitudes of head angular acceleration were selected. The 1st line, closest to onset of sled translation, indicates the onset of head and trunk angular acceleration. The next line indicates the onset of peak angular acceleration. Angular acceleration magnitudes between this point and the next line were used to calculate differential peak-to-peak acceleration. In some cases (note the LD subject), initial onset and onset of peak angular acceleration were the same. *Bottom*: raw muscle EMG responses with the trunk free. Plot layout is the same as in Fig. 3*B*.

TABLE 4. *Mean latencies of the first peak of head and trunk angular accelerations during 10-cm anterior and posterior translations*

	Anterior Translations		Posterior Translations*	
	Fixed trunk	Free trunk	Fixed trunk	Free trunk
Healthy subjects				
HEAD	360 ± 70	280 ± 130	220 ± 30	360 ± 50
TRUNK	380 ± 56	385 ± 165	214 ± 32	336 ± 54
Labyrinthine deficient				
HEAD	341 ± 80	339 ± 96	217 ± 22	353 ± 89
TRUNK	383 ± 68	460 ± 110	236 ± 40	338 ± 91

Values are mean ± SD (ms). * Fixed head [$F(5,66) = 45.30$, $P < 0.0001$] and trunk [$F(5,66) = 22.92$, $P < 0.0001$] responses significantly shorter than free responses with posterior translations.

evaluate the relative contributions of inputs descending from the vestibular system or ascending from the trunk.

Role of the VCR in initiating postural reactions

The angular VCR is certainly well suited through its dynamic and somatotopic characteristics to initiate head movements in reaction to positional disturbances of the head and neck with respect to the trunk (Dutia and Price 1987; Outerbridge and Melvill Jones 1971). However, the actual contribution of this reflex to head control in humans has only been inferred, either by comparing the neck muscle responses of LD patients with those of healthy subjects or by imposing random perturbations on healthy subjects when the position of the body was fixed (Gresty 1987; Guitton et al. 1986; Ito et al. 1997; Kanaya et al. 1995; Keshner and Peterson 1995).

Response properties of the linear VCR are less well defined (Raphan et al. 2001), but there is evidence that it plays a role

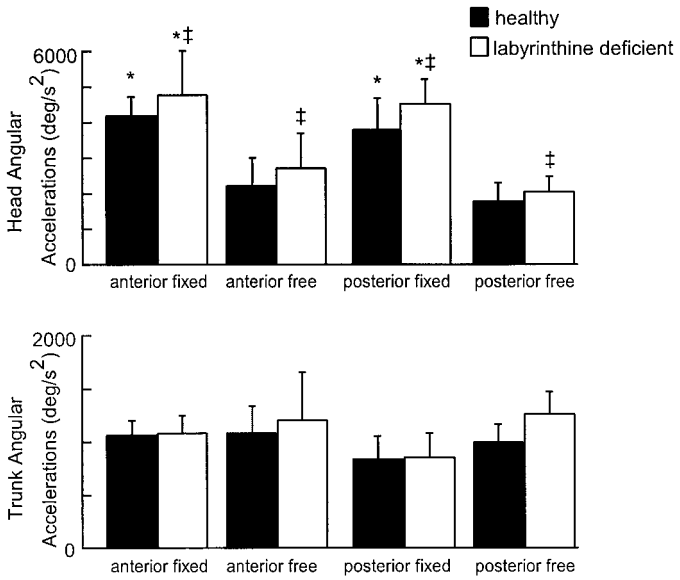

FIG. 5. Mean ± SD of the differential peak-to-peak head (*top*) and trunk (*bottom*) angular accelerations across subjects during the 10-cm ramp translations. Responses to the 4 experimental conditions are plotted for both populations on the same axis. Head angular accelerations were significantly greater (*) with the trunk fixed than with trunk free for both populations during anterior [$F(5,87) = 46.38$, $P < 0.0001$] and posterior [$F(5,87) = 70.24$, $P < 0.0001$] translations. LD subjects had significantly greater (‡) head angular accelerations than the healthy subjects during both anterior [$F(1,87) = 6.63$, $P < 0.01$] and posterior translations [$F(1,83) = 11.85$, $P < 0.001$].

in gaze stabilization and spatial orientation during locomotion (Hess 2001; Hirasaki et al. 1999; Imai et al. 2001) and is sensitive to changes in gravitoinertial acceleration (Raphan et al. 2001). In this study, neck muscle EMG responses that emerged almost at the same time as the initial acceleration of the sled in the healthy subjects, but which were absent in the LD subjects, could be due to stimulation of the otoliths. Vestibular evoked responses have been observed in the sternocleidomastoid muscle as early as 8 ms following a loud click or a tap on the forehead (Halmagyi and Curthoys 1999). These responses were present in healthy subjects and absent in subjects with LD. The short latency and ipsilateral distribution of these responses suggests that they are mediated by disynaptic

TABLE 5. *Mean onset latencies of neck and trunk muscle EMG responses during 10-cm anterior and posterior translations*

	Anterior Translations		Posterior Translations	
	Fixed trunk	Free trunk	Fixed trunk	Free trunk
Healthy subjects				
SEMI	70 ± 19	62 ± 15	75 ± 11	77 ± 25
SCM	80 ± 17*	67 ± 27	69 ± 19	60 ± 20
THOR	58 ± 18	55 ± 23	51 ± 14‡	83 ± 19
LUM	58 ± 18	65 ± 18	50 ± 38	84 ± 21
Labyrinthine deficient*				
SEMI	90 ± 30	65 ± 17	76 ± 23	87 ± 21
SCM	60 ± 12†	60 ± 29	60 ± 16	80 ± 11
THOR	66 ± 26	56 ± 2	82 ± 16‡	74 ± 11
LUM	51 ± 2	83 ± 1	70 ± 21	46 ± 20

Values are mean ± SD (ms). * Only 3 of the subjects had measurable responses. † LD fixed trunk responses are significantly shorter than healthy subjects [$t(46) = 1.74$, $P < 0.05$]. ‡ LD fixed trunk responses are significantly longer than healthy subjects [$t(55) = 2.28$, $P < 0.05$].

TABLE 6. *Muscle EMG responses represented as a proportion of the resisted voluntary contraction*

	Anterior Translations		Posterior Translations	
	Fixed trunk	Free trunk	Fixed trunk	Free trunk
Healthy subjects				
SEMI	0.18 ± 0.34	0.07 ± 0.08	0.07 ± 0.05	0.09 ± 0.10
SCM	0.12 ± 0.10	0.09 ± 0.5	0.09 ± 0.05	0.07 ± 0.04
THOR	0.07 ± 0.05	0.07 ± 0.04	0.07 ± 0.06	0.17 ± 0.33
LUM	0.08 ± 0.05	0.08 ± 0.07	0.05 ± 0.02	0.11 ± 0.12
Labyrinthine deficient*				
SEMI	0.04 ± 0.01	0.15 ± 0.17	0.06 ± 0.04	0.16 ± 0.17
SCM†	0.28 ± 0.28	0.15 ± 0.11	0.18 ± 0.14	0.10 ± 0.08
THOR	0.05 ± 0.03	0.06 ± 0.03	0.05 ± 0.01	0.05 ± 0.01
LUM	0.08 ± 0.03	0.07 ± 0.09	0.10 ± 0.05	0.04 ± 0.01

Values are mean ± SD. * Only 3 of the subjects had measurable responses. † Significantly larger responses than healthy subjects with anterior [$F(2,150) = 14.88$, $P < 0.0001$] and posterior [$F(2,137) = 5.88$, $P < 0.005$] translations.

pathways. Disynaptic connections between the otoliths and the neck flexor and extensor muscles have been identified in decerebrate cats (Kushiro et al. 1999; Uchino 1997). Thus the very short latency EMG responses could be related either to the initial acceleration of the sled or to the sound transmitted by the onset of sled acceleration.

Contributions from segmental inputs

Subsequent neck muscle latencies in this study were dramatically longer than those previously reported for VCR responses in healthy subjects. For example, during transient free-fall of the head or during whole body ascent and descent (Aoki et al. 2001; Bisdorff et al. 1994; Ito et al. 1995, 1997), sternocleidomastoid was activated at about 24–44 ms in healthy subjects compared with about 67–80 ms in LD subjects. The shorter latency response of the healthy subjects was assumed to be vestibular in origin, and the longer neck muscle responses were described as a stretch reflex rather than a response due to activation of vestibular receptors (Ito et al. 1995). In this study, neck muscle response latencies for both groups fell into the range of latencies observed in the labyrinthine deficient subjects of the prior studies.

Trunk angular accelerations always occurred prior to the head muscles, and the trunk muscles tended to respond earlier than the neck muscles, suggesting a contribution from ascending somatosensory inputs to head stabilization with respect to the body (Forssberg and Hirschfeld 1994; Gresty 1989; Vibert et al. 2001). However, the responses observed here might not be due solely to a cervicocollic reflex (CCR). Neck muscle latencies were not directionally specific, and muscle response amplitudes did not vary when the trunk was fixed or free, although both the angular and linear accelerations of the head did vary across these conditions. The latencies of the neck muscles with respect to the onset of the translation would fall in the range of long latency postural reactions (Allum and Honegger 1998; Keshner et al. 1987, 1988; Nashner 1977) and could result from multi-sensory processing rather than simple stretch responses. Thus spinal mechanoreceptors along several segmental levels could be responsible for triggering the muscular responses in this study (Allum and Honegger 1998;

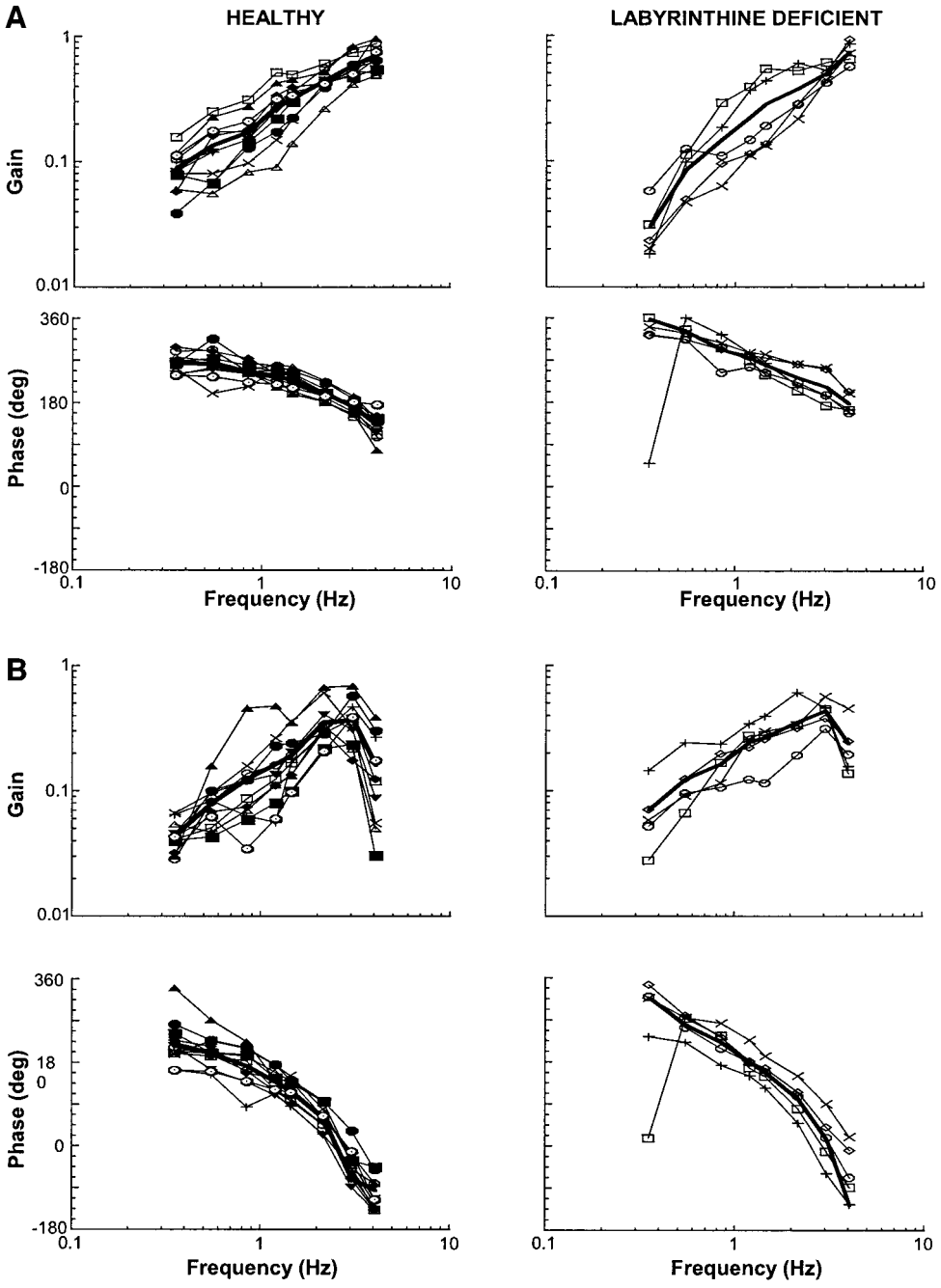

FIG. 6. Bode plots of the gains and phases of head angular position with respect to sled linear position during sum-of-sines (SSN) translations. Responses in each group are plotted for trunk fixed (*A*) and trunk free (*B*). Each symbol portrays the response of 1 subject. Bold black line in each plot is the mean of the group. According to the phase conventions, 0° indicates the head rotating in downward pitch as the sled moved forward; ±180° indicates the head rotating in upward pitch as the sled moved forward; and ±90° indicates a lag or lead of the head with respect to the sled.

Forssberg and Hirschfeld 1994; Keshner et al. 1988; Mittelstaedt 1996, 1997) either directly or through more complex pathways.

One goal of spinal segmental control of the neck muscles could be to damp the response to the ascending forces so as to minimize subsequent motions of head (Barnes and Rance 1975; Cappozzo 1981; Gresty 1989) and could reflect a role for the vestibular system other than direct stabilization of the head in space. Damping parameters could be altered through changes in reflex activation (Keshner et al. 1999; Peng et al. 1996, 1999) or through changes in the viscoelastic properties of the system via descending controls (Gresty 1989; Woollacott et al. 1988). A physiological correlate for the participation of the vestibular system in this process has been observed in secondary horizontal canal neurons that were specifically related to neck velocity (Gdowski and McCrea 2000). It was speculated that the neck proprioceptive inputs would provide a feedback signal about trunk velocity so that the vestibular neurons could remain sensitive to perturbations of the body even as the vestibular drive was reduced by reduction of head motion in space.

A loss of vestibular damping would explain the increased magnitudes of the later components of trunk and head angular and linear motion in the LD subjects. Unity ratios between the head and trunk were observed in the healthy subjects, but these ratios were increased in the subjects with LD, thereby implicating modification in the damping parameters. Frequency characteristics of the current data from healthy subjects were

similar to earlier studies that concluded the damping mechanism was reflex controlled (Keshner and Peterson 1995; Keshner et al. 1995, 1999; Peng 1996). With the trunk fixed, lower response gains and phases close to 0° at the lowest frequencies in the LD subjects suggest a head that is moving with the sled, possibly through CCR control. A 90° phase advance in the healthy subjects suggests some compensation of the head for sled translation, potentially as a result of otolith inputs. With the trunk free, the flattened phase responses at frequencies below 1 Hz in the healthy subjects imply a neural controller damping the system mechanics (Keshner et al. 1999). In the LD subjects, the steep phase descent was continuous across the frequency range as would be expected if the only controller were system mechanics and there were no neural delays present in the response (Keshner et al. 1995). However, steeply descending phases and gain drops observed above 2 Hz when the trunk was free might also be due to increasing time delays in the control system as would occur if the descending controllers required more processing time than simple reflex mechanisms.

Initial control by system mechanics

Perhaps the most compelling argument against labyrinthine inputs exclusively generating the reactive head movements in this study was finding that, although the sled translation was reasonably consistent, head motion was very dependent on actions of the trunk taking place before that of the head (this would include changes at the neck). Linear motions of the head clearly imitated the linear motion of the sled when the trunk was fixed. When the trunk was free to move, linear head movements occurred in the direction of trunk motion (i.e., opposing the direction of translation) and with a longer latency than when the trunk was fixed. Thus linear motion of the head matched the direction of linear motion of the trunk whether the trunk was fixed or free. The longer response times with a free trunk might have been due to a shift in the primary axis of rotation—from the neck when the trunk was fixed to the hip when the trunk was free.

It is possible that the angular response of the head was completely controlled by movement mechanics. Head angular accelerations were larger when the trunk was fixed to the sled than when the trunk was free. This could be explained by the smaller moment of inertia acting on the head when the trunk was fixed than when the trunk was free to move. The counter movement of the head on the trunk could be due to torques created by the inertial forces acting on the trunk center of mass. During a posterior translation of the sled, the shoulders moved in an anterior direction in space thus producing an inertial torque on the head that would rotate the head nose up or in the direction opposite the trunk rotation. This would explain why counter rotation of the head to the trunk was observed in the trunk free but not the trunk fixed condition where anterior motion of the upper trunk was constrained. The LD subjects might not have shown as much counter rotation of the head because of changes in body mass distribution in the trunk (i.e., a trunk center of mass closer to the midpoint of the trunk would lessen the anterior movement of the shoulders relative to the base of the trunk) or because of increased passive stiffness of the head/neck mechanical system. More likely, however, was that these subjects actively altered their system mechanics (Gresty 1987; Keshner et al. 1999; Peng et al. 1996; Viviani and Berthoz 1975), potentially through an increased contribution from the CCR for modulating stiffness and the viscoelastic properties of the head-neck system (Dutia and Price 1987; Goldberg and Peterson 1985; Peterson et al. 1985; Schor et al. 1988).

In conclusion, the kinematics of head-trunk coordination during linear translations revealed a strong reliance of the head on passive trunk mechanics and on signals from segmental receptors to react initially to the linear translations and to establish the position of the head with respect to the trunk. With LD, there might be greater reliance on actively adjusting the mechanical parameters of the system through ascending segmental or descending higher order neural controls. The vestibular system does not appear to be directly implicated in generating the initial postural reaction. The role of the vestibular system may actually be one of damping the response to the mechanics of the system and of monitoring the position of the head and trunk in space, secondary to feedback from segmental proprioceptors, to minimize the sustained effects of destabilization and maintain orientation in space.

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