Abstract — Control of head position during postural responses is important to facilitate both the interpretation of vestibular signals and the stabilization of gaze. In these experiments, we compared head stabilization for two different postural tasks: 1) in response to perturbations at the head, and 2) in response to perturbations induced at the support surface, which perturb both body and head position. To determine whether normal vestibular function is necessary for head stabilization in these two tasks, responses to forward and backward mechanical perturbations of the head and body were compared for 13 normal subjects and 4 patients with profound bilateral vestibular loss (two with vestibular loss in adulthood and two in infancy). Normal subjects showed little neck muscle activity for body perturbations, but large, early activations in both neck extensors and flexors for head perturbations. In contrast, vestibular patients showed excessive neck muscle activation for body perturbations and reduced or absent neck muscle activity for head perturbations. Patients with vestibular loss in adulthood also showed increased head acceleration in response to both head and body perturbations, but patients with vestibular loss in infancy showed more normal head accelerations. For body perturbations, the differences in head acceleration between patients and normals were greater for later head acceleration peaks, indicating poor head control during the execution of the postural response. Trunk angle changes were also higher in the patients for forward body perturbations, indicating that poorer control of trunk position could have contributed to their poorer head stabilization. These results indicate that the vestibular system plays an important role in head and trunk stabilization for both head and body perturbations. However, the more normal head accelerations of the patients with infant vestibular loss also indicate that other mechanisms, possibly involving neck reflexes, can at least partially substitute for the vestibular system to provide head and trunk stabilization. Copyright © 1996 Elsevier Science Inc.

Keywords — vestibular; posture; head stabilization; somatosensory; EMG; neck.

Introduction

Any movement of the body, whether voluntarily initiated or in response to an externally imposed perturbation, can potentially result in movement of the head. Uncontrolled head movements could both compromise gaze stability and complicate the use of vestibular information for sensory orientation and postural control (1), and previous experiments have shown that the head is stabilized with respect to the environment during a variety of movement tasks (2-6). However, these studies have also indicated that the central nervous system may achieve head stability in different ways depending on the nature of the movement task. For example, passive viscoelastic properties of the neck, vestibulocollic and cervicocollic reflexes, and voluntary or anticipatory mechanisms may all contribute to stabilization of the head depending on the type of perturbation and the goal of the subject per-
forming the task. In studies of head control in response to horizontal and vertical plane rotations, Keshner and colleagues have shown that reflexive head stabilization mechanisms predominate for stimuli in the 1–2 Hz range, but voluntary mechanisms predominate below this range (5,6). However, in other studies, including studies of small perturbations delivered directly to the head, viscoelastic forces have been shown to predominate, especially for higher frequency perturbations (7,8).

Another example of the variety of ways in which head stabilization is achieved is provided by previous studies of the control of head position during responses to stance perturbations induced by movements of the support surface (3,9). Subjects responding to small, slow backward translations of their support surface showed small head movements and no systematic activation of neck muscles. The postural response in this type of perturbation is small in amplitude, with body sway resembling that of an inverted pendulum (“ankle strategy”). The absence of neck muscle activations indicates that the viscoelastic properties of the trunk and neck are sufficient to provide head stabilization for these postural responses. When subjects are translated backward standing on a short beam, however, they respond with large hip flexions (“hip strategy”) initiated by activation of abdominal muscles. In response to beam perturbations, neck flexors are activated simultaneously with abdominal muscles to stabilize the head, which might otherwise be rotated backwards by the forward flexion of the trunk.

If the way in which head stabilization is achieved changes depending on the task, the importance of sensory information from different sensory systems might also be expected to change with the task. For stance perturbations induced by small or slow movements of the subject’s support surface in which viscoelastic forces are thought to predominate in head stabilization, loss of vestibular and/or neck somatosensory information might have little effect on the stabilization of the head. For larger or faster perturbations, or for perturbations induced directly at the head or trunk (such as a slap on the back), however, vestibulocollic and cervico-collic reflexes would be expected to play a predominant role in stabilizing the head. Loss of a sensory system could either lead to poor head stabilization for some tasks, or the central nervous system might compensate for the loss by substituting sensory information from another system or by expanding the range over which a particular head stabilization strategy is used. For example, patients with bilateral vestibular loss could attempt to stabilize the head by voluntarily stiffening the head and neck system and thereby rely more heavily on viscoelastic force to control the position of the head. However, there is also evidence that head stability is poorer in patients with vestibular loss. In previous studies, patients with vestibular loss have been found to have poorer head stabilization than normals for some types of movement tasks, such as running or walking in place (10) or responding to translations of a short support surface using hip strategy (11). Abnormal neck muscle activation patterns have also been observed in vestibular loss patients responding to rotations of their support surface (12). However, if the sensory loss occurs earlier, rather than later, in development, compensation might be more complete. Studies of subjects who lost vestibular function in infancy show normal or near normal postural coordination in some situations (13,14).

In the experiments reported here we compared head stabilization for two different postural tasks that presented two very different challenges to head stability and postural control. In the first task, perturbations in head alignment were induced directly at the head and upper trunk using a back-pack mounted device (14), and in the second, perturbations of the whole body were induced at the support surface. The way in which head stability is achieved in response to support surface perturbations, which produce large motions of the body, but relatively little early head acceleration, might be expected to be different than for perturbations of the head and neck, which induce larger early head acceleration, with little or no center of mass movement (14). To compare the role of the vestibular system in head stabilization for these two different types of postural tasks, we compared the responses of normal subjects with those of patients with profound bilateral vestibular loss. Finally, we compared the results of patients with vestibular loss in adult-
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hood to those of patients with vestibular loss in infancy to examine the degree of compensation for vestibular loss in these two groups and to determine the extent to which other head stabilization mechanisms, including neck reflexes, may substitute for the missing vestibular information.

Materials and Methods

Subjects

Normal subjects were 13 adult volunteers of both sexes aged 18 to 44 (mean: 31.2 years), with no history of otologic, neurologic, or orthopedic abnormality. Four patients with profound bilateral vestibular loss also participated. These patients were profoundly deaf and had bilaterally absent caloric responses. Horizontal vestibulo-ocular reflex gains were less than 0.01 for sinusoidal rotation frequencies between 0.01 and 0.05 Hz, and vertigo could not be induced using either caloric or rotational stimuli. Two patients, A1, aged 57 years and A2, 65 years, lost vestibular function as adults, 6 to 8 years prior to the study. A1 lost vestibular function due to bilateral temporal bone fractures, A2 to exposure to ototoxic antibiotics during treatment for meningitis. The other two patients, aged 17 (C1) and 19 (C2), lost vestibular function within the first year of life due to bacterial meningitis. All subjects were free of neurological, orthopedic, or psychological deficits that might affect postural control. The experiment protocol described here was approved by the Institutional Review Board of the Good Samaritan Hospital & Medical Center and was performed in accordance with the 1964 Helsinki Declaration. All subjects were volunteers who gave their informed consent prior to participation in these studies.

Apparatus

During testing, subjects stood on a servo-controlled, hydraulically driven platform that could be moved horizontally backward or forward under the control of a DEC LSI 11/23 computer (see 15, 16 for more complete descriptions). Translation of the platform produced body sway in the opposite direction. Throughout testing, the subject also wore a backpack-mounted torque motor and cable system (see Figure 1) that suspended one 1-kg weight on each side of the head at the level of the mastoid bone (17). The torque motor was driven by computer to produce forward and backward horizontal translations of the suspended weights. Translation of the weights produced displacement of the head in the opposite direction. All subjects stood with a slight forward lean to compensate for the weight of the backpack, which totaled 7.5 kg. This forward lean was less than 3° in all cases and was not sufficient to alter the onset latency or activation amplitude of the postural muscles, which were comparable to previous studies (3, 18, 19).

Protocol

Pilot testing was carried out in 4 subjects to determine which platform and weight transla-
tion magnitudes would produce equivalent maximum horizontal linear head acceleration for both head and body perturbations. The translation magnitudes chosen were 3.6 cm at 15 cm/s for the platform and 3 cm at 20 cm/s for the weights. The average maximum forward linear head acceleration for the 13 subjects was 0.44 ± 0.20 m/s^2 for platform and 0.58 ± 0.11 m/s^2 for weight displacements. The average maximum backward linear head acceleration was -0.50 ± 0.22 m/s^2 for platform displacements and -0.69 ± 0.16 m/s^2 for weight displacements.

The experiment consisted of a total of 32 one-second trials in 4 blocks of 8 similar trials. Each subject underwent 8 backward and 8 forward platform translations ("body perturbations") and 8 backward and 8 forward translations of the suspended weights ("head perturbations") while standing on the surface of the stationary platform (32 trials). To eliminate the effect of vision, all testing was carried out with eyes closed. Vestibular patient A2 could not maintain independent stance when displaced backward at the support surface while wearing the backpack and was therefore not tested in this condition.

Data Collection and Analysis

Electromyography. Muscle activity was recorded using pairs of 2.5-cm surface electrodes spaced 2 to 4 cm apart on the neck extensors and neck flexors. The placement for the neck extensor electrode was over the upper trapezius (TRAP) above the level of C4 and slightly lateral to the midline; it is possible that some activity in splenius is also reflected in this recording. The placement for the neck extensors was over the sternocleidomastoid (STER). Results for leg and trunk muscles have been reported elsewhere (14). EMG signals were band-pass filtered (100 to 5000 Hz) and full-wave rectified prior to sampling (500 Hz). They were then low-pass filtered (100 Hz) and stored for offline analysis.

EMG burst onset latencies were defined for each muscle for each trial as the time the burst activation first exceeded the baseline level (average activation level for the 100 ms prior to perturbation onset) by at least 2 standard deviations and was maintained above baseline for longer than 25 ms. Muscles were considered active in response to a given perturbation if bursts were identified on 6 of the 8 trials within 200 ms following the onset of the perturbation. Burst amplitudes were determined by integrating the area under the rectified EMG signal for the first 75 ms following burst onset (IEMG).

Kinetics and kinematics. Horizontal linear head acceleration was measured by a linear accelerometer (Entran Devices Model EGA 3-F-5D, 15 mV/g, ±5g) affixed to the skin at the center of the forehead with adhesive tape. Head linear acceleration was sampled at 500 Hz, and anterior movement was coded as positive. Body and head positions in the sagittal plane were determined with an optoelectronic movement analyzer (Watsmart, Northern Digital, Waterloo, Ontario). Markers were placed on the lateral condyle of the knee, the lateral trochanter of the femur, the acromion of the shoulder, at the earlobe, and on the chin. Marker positions (error: ±1.5 mm) were sampled at 400 Hz, filtered at 5 Hz, and used to calculate hip (angle defined by the lateral condyle, lateral trochanter, and acromion) and neck angles (angle defined by the acromion, earlobe, and chin). Results for the body's center of mass and other joint angles are reported elsewhere (14). The timing and amplitude of individual peaks and the total amplitude (peak-to-peak) of horizontal linear head acceleration and hip and neck angles were determined for individual trials, and the values were averaged within subjects.

Statistical analysis. Because of the small sample size for the vestibular patients, parametric statistics were not used to compare the performance of normals and vestibular patients. The medians and percentile scores were determined for normal subjects, and the responses of patients were compared to the 10th to 90th percentile range of the normal distribution for EMG onsets. Mann–Whitney U tests were used for comparisons of horizontal linear head accelerations and hip and neck angles.
Results

EMGs

Responses to body perturbations. Normal subjects did not consistently activate neck flexor or extensor muscles in response to either forward or backward body sway in response to surface displacements and, with some exceptions, the level of tonic activity in neck muscles was typically low (Figure 2A and B). This suggests that the visco-elastic properties of the neck, by themselves, are sufficient to stabilize head position acceptably for this type of postural perturbation. The level of tonic activity in neck extensors was somewhat higher than in neck flexors, but this was variable (compare responses to forward and backward body perturbations for N3 through N5) and may have been due to increased weight of the backpack. Unlike healthy subjects, the subjects with vestibular loss showed consistently high levels of tonic activity in neck muscles, especially in neck flexors, and some subjects showed muscle bursts in response to body perturbations. In response to forward body perturbations, vestibular subject A2 activated a neck flexor burst (STER) at 98 ms and neck extensors (TRAP) at 177 ms. In response to backward body perturbations, A1 activated a neck extensor burst at 139 ms, but C1 showed a burst in neck flexors at 141 ms. The latencies for the neck muscle activation in the vestibular subjects responding to body perturbations were 30 to 70 ms after the earliest muscle activations in the legs and trunks of these subjects (14) and it is therefore possible that the neck muscle bursts represent responses to the vestibular subjects' own equilibrium movements.

Responses to head perturbations. In response to forward head perturbations, healthy subjects co-activated the neck flexor (STER at 54 ms, median) and extensor (TRAP at 49 ms, median; Fig. 3A). These neck muscle responses in healthy subjects were very consistent, both across subjects and from trial to trial within healthy subjects; within-subject standard deviations ranged from 2 to 19 ms. In contrast, vestibular loss subjects showed highly variable muscle burst patterns across subjects, and for trials where burst activation was present, the onset latencies were variable within subjects, with standard deviations ranging from 26 to 58 ms across trials. Vestibular loss subject A1 failed to activate either neck extensors or neck flexors. Subject A2 activated neck extensors only at latencies above the 90th percentile for healthy subjects (77 ms). The pattern of neck muscle activation for the subjects with vestibular loss in infancy was different from either pattern seen in the adult loss subjects. C1 and C2 activated neck flexors only, at latencies below the 10th percentile for normal subjects (32 and 34 ms, respectively).

In response to backward head perturbations, healthy subjects reciprocally activated neck extensors (TRAP at 48 ms; Figure 3B) then flexors (STER at 74 ms). In contrast to normal subjects, who changed their pattern of neck activation based on the direction of the head perturbation, the pattern of neck activation for subjects with vestibular loss was similar for backward and forward perturbations; A1 showed no burst activation, and subject A2 activated neck extensors only at an abnormally long latency (74 ms), and subjects C1 and C2 with vestibular loss in infancy activated neck flexors only, at latencies within the normal range (56 and 60 ms, respectively).

Head Accelerations

Responses to forward body and head perturbations. In addition to the abnormal pattern of neck muscle activation in the vestibular loss subjects, head accelerations tended to be larger and to show more peaks in patients with vestibular loss, especially in patients with vestibular loss in adulthood (Figure 4). For forward body perturbations, the median peak-to-peak amplitude of horizontal linear head acceleration for normal subjects was 1.18 m/s². Both subjects with adult loss showed peak-to-peak head acceleration amplitudes above the 90th percentile for the normal distribution (2.3 m/s² and 2.4 m/s² for subjects A1 and A2, respectively). The subjects with infant vestibular loss, however, showed peak-to-peak amplitudes within the interquartile range of normals. For forward head perturbations, the median peak-to-peak head acceleration amplitude for normals was 1.45 m/s².
A. Forward Body Displacement

Normal Subjects

N1
N2
N3
N4
N5
N7

Time (ms)
-200 0 200 400 600 800

B. Backward Body Displacement

Normal Subjects

N1
N2
N3
N4
N5
N7

Time (ms)
-200 0 200 400 600 800

Figure 2. Neck EMG responses to forward (A) and backward (B) body perturbations in 6 representative healthy adults (left panel) and 2 patients with bilateral vestibular loss in adulthood and 2 patients with vestibular loss in infancy (right panel). Responses are averages of 8 trials. In each pair of responses, trapezius (TRAP, neck extensor) is displayed over sternocleidomastoid (STER, neck flexor). A1 and A2 are adult-onset vestibular loss subjects and C1 and C2 are infant-onset vestibular loss subjects. The vertical line (time zero) indicates the onset of platform movement. Normal subjects show quiet baseline EMGs (with some exceptions) and no burst activity in response to body perturbations, but patients with vestibular loss show high levels of tonic activity and some burst activity. Small numbers indicate the onset latencies for the patients’ muscle bursts.
A. Forward Head Displacement

Normal Subjects

N1

N2

N3

N4

N5

N7

-200 0 200 400 600 800

Time (ms)

B. Backward Head Displacement

Normal Subjects

N1

N2

N3

N4

N5

N7

-200 0 200 400 600 800

Time (ms)

Figure 3. Neck EMG responses to forward (A) and backward (B) head perturbations in 6 representative healthy adults (left panel) and 2 patients with bilateral vestibular loss in adulthood and 2 patients with vestibular loss in infancy (right panel). Results shown are averages of 8 trials. In each pair of responses, trapezius (TRAP, neck extensor) is displayed over sternocleidomastoid (STER, neck flexor). A1 and A2 are adult-onset vestibular loss subjects, and C1 and C2 are infant-onset vestibular loss subjects. The vertical line (time zero) indicates the onset of weight movement. Normal subjects show quiet baseline activity and distinct bursts in both neck flexors and neck extensors in response to both perturbation directions, but patients with vestibular loss show high levels of tonic activity and absent or irregular burst patterns. Small numbers indicate the onset latencies for patients' muscle bursts.
and all vestibular subjects showed increased peak-to-peak head acceleration amplitude. The results for A1 and A2 both exceeded the 90th percentile for normals, and the difference between normals and vestibular loss subjects was statistically significant (Mann–Whitney U, U = 5, P < 0.02).

The increased head acceleration in the patients with vestibular loss was also associated with an increased number of head acceleration peaks (characteristic of an underdamped response) for the patients as compared to normals. In Figure 4, for example, subject A1 shows four positive (anterior head acceleration) peaks and four negative (posterior head acceleration) peaks in the response to body perturbation, whereas the normal subject shows only one early posterior peak and a later anterior peak. For forward head perturbations, the median number of peaks for the normals was 1.6, compared to 2.8 for the vestibular patients; this difference was statistically significant (Mann–Whitney U, U = 5, P < 0.02). For forward body perturbations, the median number of peaks for the normals was 3.2 and 5.2 for the patients, and this difference was also statistically significant (Mann–Whitney U, U = 5, P < 0.02).

The increase in head acceleration amplitude observed in the patients with vestibular loss occurred during the execution of the postural movement for forward body perturbations, but directly in response to the movement of the suspended weights for forward head perturbations. Head acceleration in response to forward body perturbation was characterized by a small amplitude early peak in the opposite direction to the perturbation ("first peak," indicated by a thin arrow in Figure 4) followed by the maximum forward acceleration at about 200 ms following platform movement ("second peak," indicated by a heavy arrow in Figure 4). In response to forward head perturbations, maximum forward head acceleration occurred at about 50 ms in all subjects ("first peak," indicated by a light arrow in Figure 4; "second peak" is indicated by a heavy arrow). The amplitudes of the first two peaks of head acceleration in response to body perturbation were not different in patients and normals. The increased peak-to-peak amplitude of head acceleration found in patients was accounted for by increases in later peaks, occurring at more than 200 ms after the onset of the platform movement and at about 100 ms after the onset of the EMG activity in leg muscles; that is, during the execution of the postural movement (14). For forward head perturbations, the pattern of results was reversed; the amplitudes of both the first and second peaks of head acceleration were larger for the vestibular loss subjects. This difference approached significance for the first peak, which occurred at about 70 ms (Mann Whitney U, U = 9, P = 0.054), and was significant for the second peak (Mann Whitney U, U = 5, P < 0.02).

Responses to backward body and head perturbations. For backward body perturbations, the median of the normal subjects’ responses was 1.13 m/s². Subject A1, in contrast, showed a peak-to-peak amplitude of 3.26 m/s², outside the 90th percentile for the normal distribution. Subject A2 could not perform this task, and fell repeatedly when displaced backwards. The peak-to-peak amplitudes for backward body perturbations for subjects with infant loss, however, were within or below the interquartile range of normals (see Figure 5). For backward head perturbations, the median for the normal subjects was 1.58 m/s². Only the subjects with vestibular loss in adulthood showed peak-to-peak head acceleration exceeding the 90th percentile limit for normals, but the values for C1 and C2 were within the normals’ interquartile range. Head acceleration was less well damped in response to backward perturbations than in response to forward perturbations for both normals and patients, but patient A1 showed almost twice as many peaks in head acceleration as did normal subjects. Also, the differences in amplitude occurred at the same time for forward and backward head accelerations, that is, during the execution of the postural movement for backward body perturbations and directly in response to perturbation of the suspended weights for forward perturbations.

Joint Angles

To determine whether the patients' increased head acceleration might be related to increased
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Forward Displacements

Normal Subject

Body Displacement

Head Displacement

-200 0 200 400 600 800

Time (ms)

Adult Vestibular Loss

Body Displacement

Head Displacement

Infant Vestibular Loss

Body Displacement

Head Displacement

-200 0 200 400 600 800

Time (ms)

Figure 4. Horizontal linear head acceleration in response to forward body and head perturbations for a representative normal subject and patients A1 (vestibular loss in adulthood) and C2 (vestibular loss in infancy). In each panel, responses for forward body perturbations are displayed above responses for forward head perturbations. For each perturbation type, the heavy line indicates the averaged response of the 8 individual trials displayed below it. The vertical line indicates the onset of the stimulus, and positive values indicate anterior motion. Thin arrows indicate the first peak of head acceleration analyzed for each trace, and heavy arrows indicate the second peak. Patients with vestibular loss show underdamped head accelerations (multiple peaks) and increased head acceleration amplitudes compared to normals.

trunk movement for responses to body perturbations, hip and neck angle changes were compared in patients and normals. Figure 6 shows hip angle changes for a representative normal subject and for patients A1 and C2. For normals, the median peak-to-peak hip angle change for forward body perturbations was 4.4° (interquartile range: 3.0 to 4.5). All vestibular subjects showed peak-to-peak changes exceeding the 75th percentile for normals, and the group...
Figure 5. Horizontal linear head acceleration in response to backward body and head perturbation for 1 representative normal subject and patients A1 (vestibular loss in adulthood) and C2 (vestibular loss in infancy). In each panel, responses for forward body perturbations are displayed above responses for forward head perturbations. For each perturbation type, the heavy line indicates the averaged response of the 8 individual trials displayed below it. The vertical line indicates the onset of the stimulus, and positive values indicate anterior motion. Thin arrows indicate the first peak of head acceleration analyzed for each trace, and heavy arrows indicate the second peak. Patients with vestibular loss in adulthood show underdamped head accelerations (multiple peaks) and increased head acceleration amplitudes, but the results of patients with vestibular loss in infancy are within normal limits.
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difference was statistically significant (Mann-Whitney $U$, $U = 6$, $P < 0.05$).

Similar to the findings for head acceleration, the values for the initial hip angle peaks were not systematically different for patients and normals, indicating that their difficulty in controlling trunk position occurred later, during the execution of the postural response. The pattern of hip angle movement consisted of an early extension peak followed by a later flexion peak. The median value of the extension peak for normals was $1.9^\circ$ (1.6 to 2.0 interquartile range) and the median for the patients was $2.0^\circ$, suggesting similar compliance of the trunk and leg segments to external perturbation. The median for the flexion peak for normals was $1.8^\circ$ (0.9 to 3.5 interquartile range), and 3 of the 4 patients showed flexion peaks that exceeded the 75th percentile of normals, suggesting that trunk motion is higher in patients than in the normals during the execution of the postural response. The pattern of results for peak-to-peak neck angle changes was similar, with all but one vestibular subject (C1) showing values exceeding the 75th percentile for normals. The group difference was also statistically significant (Mann-Whitney $U$, $U = 7$, $P < 0.05$). For backward body perturbations, both patients and normal subjects showed higher peak-to-peak hip and neck angle excursions than they did for forward body perturbations, but the differences between the groups were no longer significant.

Discussion

Normal subjects showed no systematic activation of neck muscles during responses to the range of support surface movements used in these experiments. This finding, which is similar to previous results (11), is consistent with the hypothesis that the passive visco-elastic properties of the neck without a contribution from vestibular or neck reflexes are sufficient to stabilize head position during this type of postural perturbation in normal subjects. However, this interpretation is not consistent with the finding that patients with profound bilateral vestibular loss show both excessive neck muscle activity and abnormally high head accelerations in response to displacements of the support surface.

An alternative interpretation of the poorer head control in patients with vestibular loss is suggested by the timing of the abnormally high head acceleration peaks in the vestibular patients. The abnormal peaks occurred later in the response, about 200 ms after the onset of support surface movement and about 100 ms after the onset of the postural response (14). This implies that the vestibular patients had difficulty controlling head acceleration during the execution of the postural response itself, rather than immediately in response to the movement of the support surface. It may therefore be the case that normal subjects limit head acceleration during postural movements by controlling trunk and limb movement. If the patients with vestibular loss have difficulty controlling the coordination of postural responses, increased movement of the trunk could result in increased head acceleration during postural movements.

Evidence from both previous studies and the work presented here suggests that patients with profound bilateral vestibular loss have difficulty controlling the trunk. Clinical studies of balance performance show that patients with vestibular loss perform poorly in tasks such as tandem stance or walking, or balancing or walking on narrow beams, both of which require controlling trunk angular accelerations about the hips (20). Patients with profound bilateral vestibular loss are also unable to respond to translations of a short support surface using a hip strategy (11,21). Ailim and colleagues have reported that vestibular loss patients responding to platform perturbations show abnormally low activation amplitudes in paraspinal muscles (22). Some patients with vestibulopathy (as opposed to vestibular loss) also show abnormally increased hip sway and decreased paraspinal activity (23).

In these studies, patients with vestibular loss showed increased hip angle movement as well as increased neck angle movement compared to normals in response to forward perturbations of the body, but not in response to backward perturbations. The increase in hip angle movement for forward body perturbations in patients with vestibular loss paired with high head accelerations in response to displacements of the support surface.
Figure 6. Hip angle changes in response to forward body perturbation for 1 representative normal subject and patients A1 (vestibular loss in adulthood) and C2 (vestibular loss in infancy). The heavy line in each set indicates the averaged response of the 8 individual trials displayed below it. The vertical line indicates the onset of the platform movement, and positive values indicate flexion. Patients with vestibular loss show increased hip angle movement compared to normals.

Tions is consistent with the idea that normal subjects limit the movement of the head during postural responses by controlling hip as well as neck movements. The failure to find a similar difference in the control of the trunk for backward body perturbations may result from differences in control at the knee or ankle. The amount of hip extension that can be achieved by the body is much smaller than the amount of hip flexion, and as a result, large backward body movements tend to be achieved by flexing the knee, rather than extending the hip. Larger amplitudes of knee flexion for the patients could have resulted in their larger amplitudes of head acceleration equally as well as larger amplitudes of hip extension. Thus, the biomechanical limits of the body may play a role in determining how subjects use control of lower limb segments to enhance head stability, and the control strategies for backward perturbations might be different from those for forward perturbations.

In contrast to their responses to support surface displacements, normal subjects with eyes closed show short latency, large amplitude activity in neck flexors and extensors in response to forward and backward head perturbations. The finding that these neck muscle activations are absent or abnormal in patients with vestibular loss implies that the vestibular system, and specifically, the vestibulocollic reflex (VCR) responds strongly to stimuli in
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The response of normal subjects to head perturbations consists of nearly simultaneous bursts in both extensors and flexors that stiffen the neck to resist the perturbation, regardless of its direction. In contrast, vestibular loss patients show high tonic levels of activation without the paired flexor and extensor bursts seen in normals. The high tonic level of EMG activity in neck muscles observed in the patients with vestibular loss in all tests suggests that they are attempting to use stiffening of the head and neck system to resist disturbances in head position and thereby compensate for their loss of vestibular head stabilizing reflexes. This stiffening of the neck would have the effect of stabilizing the head with respect to the trunk using a "strap-down" strategy for controlling the head during body movements (1). This strategy, however, will necessarily result in increased movement of the head with respect to gravity for postural tasks involving increased hip movements. Further, the increased neck angle movement in the patients suggests that the attempt to lock the head to the trunk may not have been completely successful.

The stiffening of the head and neck system may also account for the increased number of head acceleration peaks observed in the patients in these studies. Changes in the frequency of head accelerations in vestibular loss patients have been previously reported by Keshner and colleagues (12). The model of the head and neck system proposed by Viviani and Berthoz (7) suggests that stiffening of the head and neck system at different points along the spinal column would produce different resonant frequencies in response to perturbations. The difference in the number of acceleration peaks between normals and patients could therefore result from the changes in the visco-elastic properties of the neck brought about by cocontraction of neck muscles. Changes in the visco-elastic forces in the neck could substantially change the pattern of head movement resulting from changes in trunk position, and could therefore account for the different and highly variable pattern of neck muscle activation seen in the patients.

In these studies, the control of the head and neck for patients with vestibular loss in infancy was consistently closer to the normal responses for all types of perturbations. This is consistent with previous observations of postural and motor control in patients with congenital or early childhood loss of vestibular function. Patients who lose vestibular function in infancy and are tested as teenagers perform similarly to age matched controls on clinical tests of motor function (26,27). Their sway responses to tendon vibration in the legs is indistinguishable from those of age-matched controls when tested on a firm, flat support surface (13). They also show normal leg and trunk muscle activations in response to head perturbations, whereas these activations were absent in patients with vestibular loss in adulthood (14).

Although the more normal results for the patients with vestibular loss in infancy presented here are consistent with previous findings, there are other factors that could have contributed to the variability in the performance of the patients in these studies. Studies of head stabilization in response to backward support surface translations in normal subjects aged 70 to 84 years indicate that the head stabilized less well in older subjects (28). Studies of head stabilization during trunk rotations suggest that reflexive (VCR) mechanisms are weaker in this age group and that the elderly rely more on viscoelastic forces or possibly stiffening the head and neck system to control head position (29). Although the older patients in this study were aged 57 (A1) and 65 (A2), it is possible that their age contributed to their poorer performance. Further studies of patients with vestibular loss should include age matched controls to eliminate this possibility. The variability in the performance of the patients in these studies may also be due in part to the differing etiologies of the vestibular loss. Patient A1 lost vestibular function due to temporal bone fractures, whereas A2 lost function due to exposure to ototoxic antibiotics during treatment for meningitis, and both C1 and C2 lost function due to meningitis. While
clinical tests of vestibular function showed profound losses of horizontal canal function in all patients, it is possible that vertical canal or otolith function may be retained to some degree in some of these patients, and that may account for some of the variability in their performance.

The more normal head accelerations of the patients with profound vestibular loss in infancy indicates that other mechanisms can at least partially substitute for vestibular mechanisms for head stabilization during postural movements. Sensory information from the neck could substitute for vestibular information in cases of vestibular loss. Many of the cells in the vestibular nuclei that give rise to descending vestibulospinal tracts have been shown to receive sensory input from the neck, and the cervicocollic reflex has been found to interact linearly with the vestibulocollic reflex in the control of head position (see 30 for a review). It is also possible that other mechanisms are involved, however. Transection of the vestibulospinal output pathways have been found to leave vestibulocollic reflexes intact in animal experiments, which suggests that other pathways, possibly including reticulospinal pathways that receive convergent vestibular and neck proprioceptive input, are involved (30).

The results of these experiments show that the vestibular system plays an important role in head stabilization in response to externally imposed perturbations and that this role changes with changing postural control tasks. These experiments further suggest that one of the tasks of the postural control system is controlling head position during postural movements themselves. Loss of the vestibular system resulted in poor control of head acceleration during responses to movements of the support surface in these studies. This poorer control of head acceleration may be secondary to poorer control of the trunk, since normal subjects show little neck muscle activation but nevertheless greater head stability than patients. These findings suggest that the stabilization of head position during postural movements relies not exclusively on vestibular and neck reflexes, but rather on the integrated action of the entire postural control system, which has at its disposal a variety of mechanisms to achieve the goal of head and gaze stability during body movements. This variety of mechanisms permits the relatively better head control observed in patients who lose vestibular function at an early age by allowing functional adaptation to the vestibular loss.

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REFERENCES


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