Effects of vestibular and cerebellar deficits on gaze and torso stability during ambulation

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We measured gaze, head, and torso stability during ambulation to determine how vestibulo-ocular reflex dysfunction caused by unilateral vestibulopathy, bilateral vestibulopathy, and cerebellar dysfunction might affect image stabilization on the retina. Subjects were tested during standing, walking, and running on a treadmill. Gaze velocity, vestibulo-ocular reflex gain, and head velocities were calculated from angular positions of the eye and head, as well as linear positions of the head and trunk. Mean gaze velocity with a visible, distant target was below 4°/second for all measurement conditions in control and vestibulopathic subjects. The performance of unilaterally vestibulopathic subjects was indistinguishable from that of control subjects except that the former had less vertical translation during walking. Bilaterally vestibulopathic subjects demonstrated less head translation than control subjects but had higher gaze velocity. In subjects with cerebellar dysfunction, gaze velocity was elevated by pathologic nystagmus, but head movements were similar to those of control subjects. (Otolaryngol Head Neck Surg 2000;123:22-9.)

The vestibulo-ocular reflex (VOR) plays a key role in maintaining visual acuity by minimizing head motion–induced image instability on the retina. In vestibulopathic subjects, oscillopsia and degradation of visual acuity can occur during ambulation caused by retinal image instability.[1-3] Several forms of cerebellar dysfunction are associated with abnormal VOR gain, the ratio of compensatory eye velocity to head velocity,[4] and abnormal modification of VOR gain with target distance.[5,6] It is not known how cerebellar deficits might influence VOR function or retinal image stability during ambulation. This study was thus conceived to investigate gaze stability during ambulation in subjects with vestibular and cerebellar dysfunction.

During ambulation, the velocity on the retina of the images of distant, stationary targets normally remains less than 2° to 4°/second,[7,8] the critical range above which acuity is detectably degraded by image motion.[9,10] Gaze stability is maintained partly because an actively generated pattern of head translation partially offsets the destabilizing effects of head rotation so as to reduce the ocular counterrotation required to eliminate retinal image motion.[8,11] This pattern of head movement changes in an appropriate way based on target distance[8,11] and the wearing of telescopic[8] or reversing spectacles.[11] Although vestibular or cerebellar dysfunction might be expected to impair compensatory head movements, such a phenomenon and any related effects on gaze stability have not previously been described.

During sudden, high-acceleration head rotation, a persistent deficit caused by unilateral vestibular deafferentation is evident because of saturation of the VOR during rotation toward the side of the lesion.[12,13] There may be differences in VOR function during lower velocity (peak velocity less than 200°/second) steady-state head rotation with unilateral deafferentation, but such asymmetries are subtle.[14] Although subjects with bilateral vestibular deafferentation have previously been shown during ambulation to have significantly higher gaze velocities than control subjects,[2] the more subtle effects of unilateral vestibular loss during ambulation have not been assessed.

Dynamic posturography is sensitive to vestibular loss. For testing while standing on a tilting platform, the center of pressure motion and body sway in vestibulopathic subjects exceed those of control subjects.[15,16] However, performance during dynamic posturography and VOR gain during whole-body rotation were not predictors of oscillopsia during ambulation.[3] This enigmatic finding implies that additional mechanisms of gaze stabilization must operate during ambulation in subjects with vestibulopathy.

Head movement strategy can change to compensate for loss of vestibular or oculomotor function. One

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Table 1. Subjects with vestibulopathy and cerebellar dysfunction

Subject No.	Age (y)	Sex	Diagnosis	Laterality	Surgery	Postoperative (mo)	Horizontal VOR gain 0.05 Hz	0.8 Hz	1.25 Hz
Vestibulopathy									
1	61	M	AN	Right	LX	15	0.23	0.48	0.62
2	69	M	MD	Left	LX	79	0.19	0.81	1.09
3	67	M	AN	Right	LX	4	—	—	—
4	59	M	AN	Right	NX	60	0.00	0.39	0.56
5	45	F	AN	Right	NX	61	0.01	0.17	0.30
6	49	F	AN	Right	NX	87	0.29	0.33	0.92
7	72	M	VL	Bilateral	None	NA	0.00	0.79	0.55
8	69	M	VL	Bilateral	None	NA	0.00	0.18	0.58
Cerebellar dysfunction									
9	22	F	CVA	—	None	NA	0.11	0.30	0.53
10	48	F	CVA	—	None	NA	0.11	0.72	0.59

Sinusoidal VOR gain was averaged bilaterally but was not available for subject 3.
AN, Acoustic neuroma; *MD*, Meniere's disease; *VL*, idiopathic vestibular loss; *CVA*, cerebellar vermian atrophy; *LX*, labyrinthectomy; *NX*, vestibular neurectomy; *NA*, not applicable.

example is the compensatory abnormal head posture associated with loss of oculomotor range.[17] A purpose of this study is to investigate the possibility that changes in head movement are used as a strategy for increasing gaze stability in those with vestibulopathy or cerebellar dysfunction. The possible importance to gaze stabilization of such a postural adaptations has not been previously investigated.

METHODS

Subjects

The University of California, Los Angeles, human subjects protection committee gave permission for this study to be conducted, and all subjects understood and signed an informed consent agreement before participation. The control group consisted of 9 healthy, paid volunteers (7 women and 2 men), aged an average of 37 ± 13 years (mean ± SD, range 20-59 years). Ten subjects had pathologic conditions (Table 1): 6 had unilateral vestibulopathy caused by vestibular neurectomy or labyrinthectomy (mean age 59 ± 10 years), 2 had idiopathic bilateral vestibular loss (aged 70 and 72 years), and 2 (a mother and daughter, aged 48 and 22 years) had nonprogressive, idiopathic familial congenital cerebellar dysgenesis. Both subjects with cerebellar dysfunction had mild ataxia, impaired visual pursuit, and upbeating and rebound nystagmus. All subjects underwent ophthalmologic examination to verify that they were free of disease and would be able to focus the targets clearly. Only the older of the cerebellar dysfunction subjects was unable to converge to near targets.

Subjects with vestibulopathy underwent conventional caloric and rotational testing to verify loss of function as previously described.[18] Subjects with unilateral vestibulopathy lacked ipsilesional caloric responsiveness, except for subject 4, whose ipsilesional response was less than 50% that of the average control subject. Those with bilateral vestibulopathy lacked caloric responses in both ears. Subjects with cerebellar dysfunction had normal caloric responses. During rotational testing subjects were rotated in yaw by a motorized chair in complete darkness at 0.05 Hz, 0.8 Hz, and 1.25 Hz.

Measurements

Angular eye and head positions were measured with scleral magnetic search coils (Skalar Medical, Delft, The Netherlands), as previously described.[8] Three-dimensional linear and angular position were obtained with flux gate magnetometer sensors (Flock of Birds; Ascension Technology, Burlington, VT), as previously described.[8] One receiver was mounted atop the subject's head with a headband. A second flux gate magnetometer receiver was fixed to the back with straps that held the sensor between the scapula like a small, tight-fitting backpack. Subjects stood on a motorized treadmill (1.1 kW) during 10-second periods of the following activities: standing with the feet together, slow walking (0.4 m/second), walking (0.9 m/second), and running (1.4 m/second), as previously described.[8] Each trial was performed in the light and repeated immediately afterwards in darkness, with instruction to the patient to remember the earth-fixed target in the location where it had previously been visible. Because the subject was free to move over a small area, nominal target distances (100, 150, and 500 cm) were measured relative to a fixed point behind the subject's head. Subjects were tested during standing with 500-, 150-, and 100-cm distant targets, in that order. Next, running and walking trials were performed starting with the target at 100, 150, and 500 cm.

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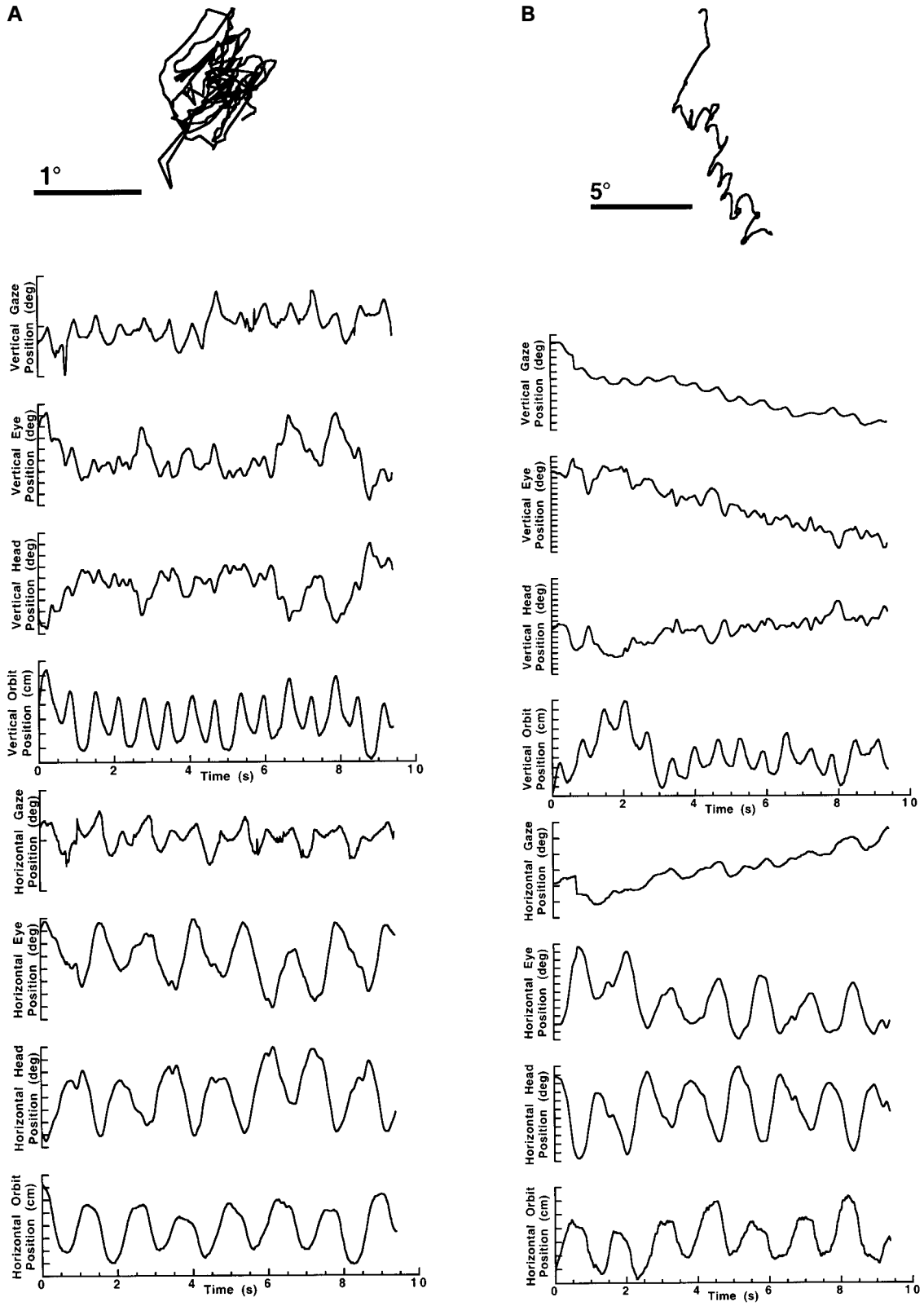

Fig 1. Gaze position on the retina, head, and eye movement during walking at 0.9 m/second with the target 500 cm in subject 5, who had right-sided vestibulopathy. Head translation is shown for the right orbit. **A,** Lights on. **B,** Immediately after, with lights off.

Analysis

Data were automatically analyzed with removal of the occasional saccades, as described previously.[16] Gain of the angular VOR was calculated for each trial with a linear regression technique on angular eye and angular head velocity, as described previously.[8,16] Angular gain measurements inevitably fail to capture the effects of translation, necessitating analysis of gaze, as detailed previously.[8] In brief, gaze position was calculated by projecting the line of sight from the measured translational center of the eye into the plane of the target. The 2-dimensional linear distance in the target plane from the intersection of the line of sight to the target itself was then back-projected into retinal coordinates (degrees) so that the gaze measurement reflected image motion on the retina as a result of effects of translation, rotation, and target distance.[8] Fourier analysis of head and gaze positions was performed with a rectangular window, taking the peak frequency as the maximum component above 0.1 Hz. Results were considered to be significant for $P < 0.01$, unless otherwise noted.

For each trial during which the treadmill was moving, Fourier analysis was used to determine the predominant frequency of motion (the frequency of at least 0.5 Hz having the highest spectral density). The phase differences between the predominant frequency of translation of the orbit and rotation in the same plane were determined and compared. If different predominant frequencies were found for translation and rotation within a plane, the phase differences at both frequencies were determined. If either phase difference was within the range of 135° to 225°, that trial was considered antiphase.

RESULTS

Gaze Stability

No subject ever perfectly maintained gaze on a target during standing or ambulation. Although average target position corresponded to the retinal fovea, calculated image position always varied somewhat, as illustrated by the example data in Fig 1*A* during room illumination and Fig 1*B* in darkness. The SD of gaze is a measure of the precision with which the target image is maintained on the fovea of the eye in each of the two retinal dimensions, horizontal and vertical, with higher values indicating greater dispersion of image position from the average position of the fovea. With the lights on, the dispersion of gaze position of control subjects was 0.40° ± 0.07° horizontally and 0.6° ± 0.1° vertically. In darkness, the calculated retinal position of the distant target slowly drifted from the fovea during the 10-second trial so that the SD increased to 1.1° ± 0.1° horizontally and vertically. During standing and walking, mean root-mean-square (RMS) gaze velocity of control subjects was ≤ 2°/second with the target at 500 cm. During running, mean gaze velocity was the highest at 3.4° ± 0.4°/second (mean ± SE). Surprisingly, the performance of unilaterally vestibulopathic subjects was similar to that of control subjects. Typical data collected in darkness and light for a subject with unilateral vestibulopathy are shown in Fig 1. Bilaterally vestibulopathic subjects had less stable gaze during standing and walking; their advanced age and lessened vigor prevented them from completing the running portion of the protocol. The two subjects with cerebellar pathology had no difficulty with the required ambulation, although they had nystagmus that confounded analysis of their VOR eye movement.

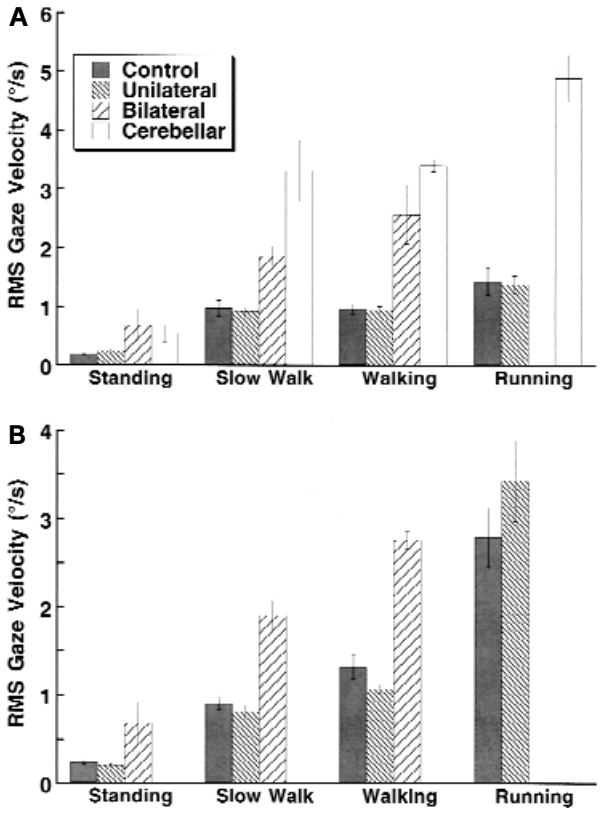

Fig 2. Mean RMS (0-8 Hz) gaze velocity ± 1 SE in a lit room with a 500-cm distant target. **A,** Horizontal. **B,** Vertical.

Gaze velocity in the light (Fig 2) for unilaterally vestibulopathic subjects did not significantly differ from that of control subjects and did not exceed 4°/second RMS for the visible far target during ambulation. In the two bilaterally vestibulopathic subjects, gaze velocity significantly exceeded that of control subjects for every condition tested. Subjects with cerebellar dysfunction had the highest gaze velocity, frequently exceeding 4°/second, largely because of superimposed upbeating nystagmus.

In darkness, gaze position errors accumulated over

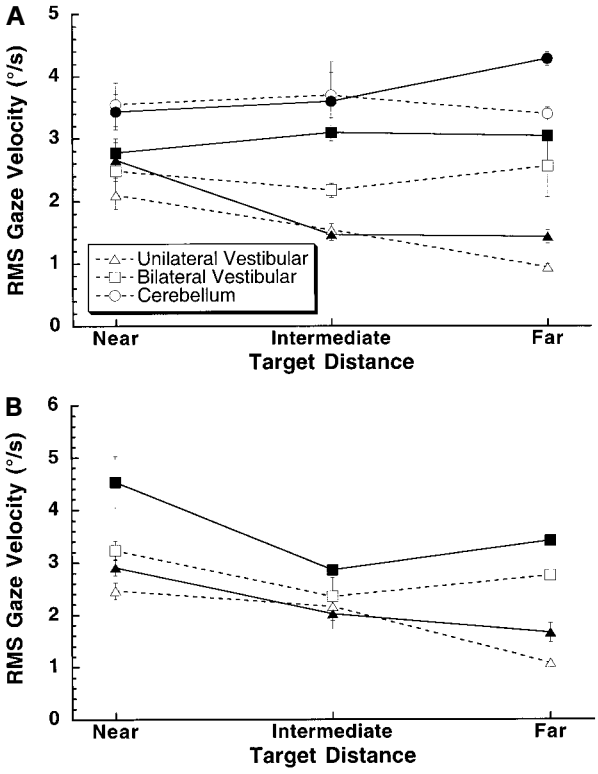

Fig 3. Mean RMS (0-8 Hz) gaze velocity varied with target distance during walking at 0.9 m/second. Data of unilaterally vestibulopathic subjects did not significantly differ from those of control subjects. For clarity, control data are not shown. *Filled symbols with solid lines,* in darkness; *open symbols with dashed lines,* in light. **A,** Horizontal. **B,** Vertical.

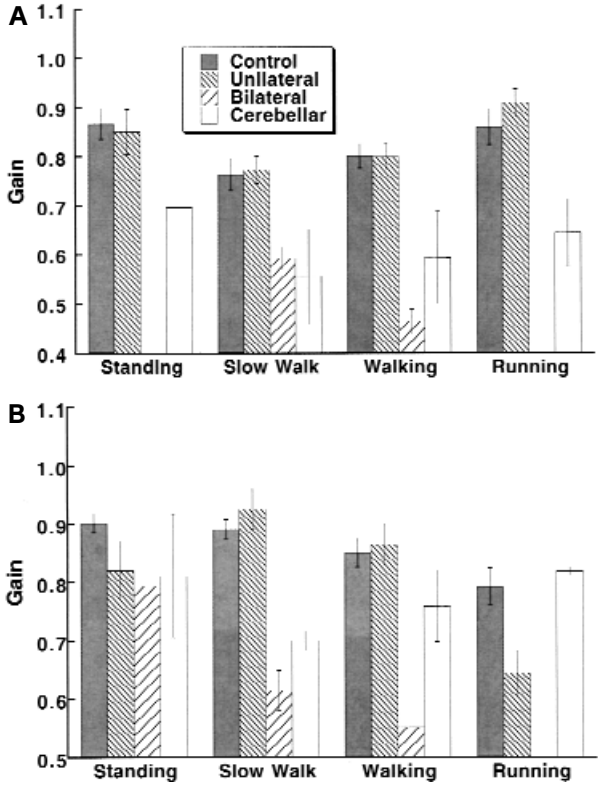

Fig 4. Mean angular VOR gain for a 500-cm distant visible target. Error bars represent ± 1 SE, except for datum based on single trial. **A,** Yaw (rotation about a vertical axis). **B,** Pitch (rotation about a horizontal axis that passes through both ears).

time, and gaze velocity increased because of slow drift in eye position as seen in the position tracings of Fig 1*B*. The slow drift was most prominent in bilaterally vestibulopathic subjects. Mean gaze velocity data are summarized in Fig 3 for subjects with pathology during walking; Fig 3 also demonstrates that target distance had little effect on gaze velocity in these subjects. In unilaterally vestibulopathic subjects, RMS gaze velocity increased in darkness compared with light by an average of 0.3° ± 0.1°/second (mean ± SE, not significantly different in horizontal or vertical directions). In bilaterally vestibulopathic subjects during walking, RMS gaze velocity averaged 0.6° ± 0.2°/second higher in darkness than in light (Fig 3). The increase in gaze velocity caused by drift in darkness was not dependent ($P > 0.05$) on the speed of ambulation (including standing) or the distance to the target for any group.

Angular VOR Gain

Angular VOR gain (Angular eye velocity/Angular head velocity) was calculated for each trial. Only values from trials when the gains were determined to be reliable (based on the criteria cited in the Methods section) are summarized here. During standing and ambulation, average VOR gain was always significantly less than unity in each subject group. Although gains for unilaterally vestibulopathic subjects did not differ significantly from those of control subjects, subjects with bilateral vestibulopathy or cerebellar deficit had significantly subnormal VOR gains for nearly every condition (Fig 4). The subject groups with low mean VOR gains also tended to have high average gaze velocities. To examine the correlation between gaze velocity and VOR gain among individual subjects, we correlated VOR gain and gaze velocity within testing conditions of target distance, lighting, group, and activity. Within individual conditions there was no significant correlation between VOR gain and gaze velocity.

Head and Trunk Movement

An antiphase relationship was consistently observed between head translation and head rotation such that, as

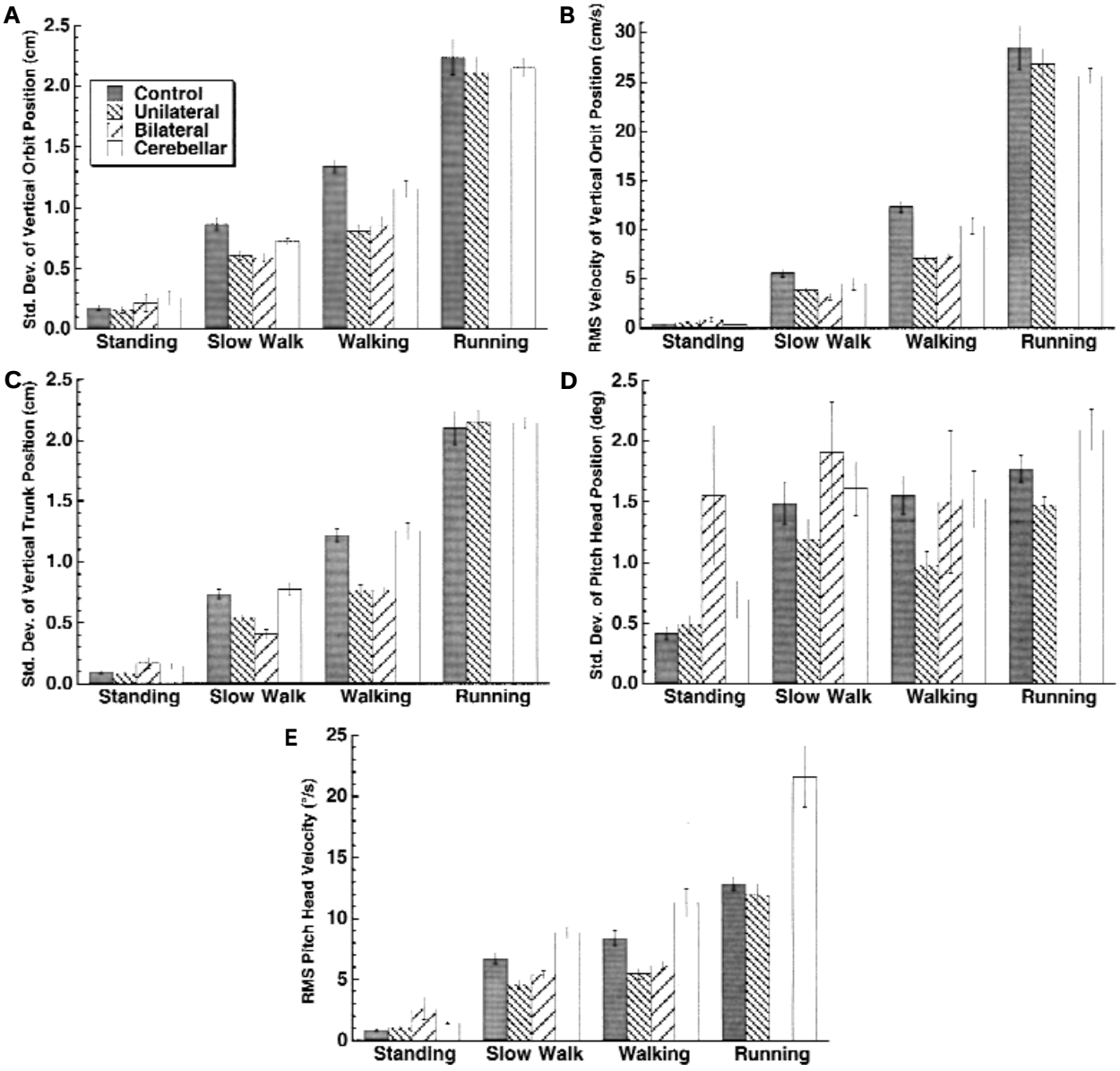

Fig 5. Mean vertical head and trunk movement ± 1 SE during standing and ambulation while viewing a far target. **A,** SD of linear vertical orbit position. **B,** RMS velocity (0-8 Hz) of linear vertical orbit position. **C,** SD of linear vertical trunk position. **D,** SD of angular head position in pitch. **E,** RMS angular head velocity in pitch.

the head rotated up, the orbit translated down, and as the orbit translated to the left, it rotated to the right. This effect is clearly seen for the horizontal direction in the data records of Fig 1. Although this effect occurred in all subjects, the antiphase behavior was not always at the predominant frequency of head motion. For the vertical data in Fig 1, antiphase behavior occurred at the second harmonic of head translation.

The prevalence of an antiphase relationship between orbit translation and head rotation was studied by Fourier analysis (Table 2). Because of the short sampling interval and variable frequency content of head movement during gait, we explored both a liberal (180° ± 80°) and a more strict (180° ± 45°) definition of antiphase behavior. According to either definition, the presence of antiphase behavior was similar in control subjects and vestibulopathic subjects. In the vertical plane, antiphase behavior was increasingly prevalent with increasing speed of ambulation. In contrast, motion in the horizontal plane was more com-

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Table 2. Translation-rotation antiphase behavior according to treadmill speed

	Vertical (%)			Horizontal (%)		
	0.4 m/s	0.9 m/s	1.3 m/s	0.4 m/s	0.9 m/s	1.3 m/s
Antiphase ± 80°						
Control	54	57	82	99	91	57
Unilateral	46	44	76	96	80	35
Bilateral	56	91	ND	100	100	ND
Cerebellar	67	39	11	83	50	28
Antiphase ± 45°						
Control	22	15	23	75	63	21
Unilateral	19	19	50	81	57	2
Bilateral	22	27	ND	67	82	ND
Cerebellar	6	0	0	50	28	0

Percentage of trials exhibiting antiphase relationship between orbit translation and head rotation, within broad limits of 180° ± 80° and more rigorous limits of 180° ± 45°.
ND, Not done.

monly antiphase during lower velocity ambulation than during running. Unlike in control and vestibulopathic subjects, in subjects with cerebellar dysfunction the antiphase relationship decreased with increasing velocity.

Subjects with vestibulopathy generally moved their heads and trunks less during ambulation than did control subjects. During walking, vertical RMS head velocity averaged 7.1 ± 0.4 and 7.4 ± 0.3 cm/second in unilaterally and bilaterally vestibulopathic subjects, respectively, significantly less ($P < 0.01$) than the 12.4 ± 0.6 cm/second average of control subjects (Fig 5). A similar trend was found for angular and linear head displacement. Like the head, vertical trunk motion during walking was smaller in those with vestibulopathy (Fig 5*C*). No significant differences between control subjects and vestibulopathic subjects could be found either in the horizontal direction or while standing. No significant differences in head or trunk motion were manifest during running, although only a subset of vestibulopathic subjects was able to complete this task. When compared with control subjects, subjects with cerebellar dysfunction had no significant difference in the amplitude or velocity of head movements.

To test whether changes in head movement were modulated by changing the position of the center of the head mass relative to the trunk, we determined the average position of the head sensor relative to the trunk sensor for each trial. No significant variation with target distance of head location relative to the trunk was observed in any subject group.

DISCUSSION

Gaze Stability

Gaze measurements fully consider both angular and linear motion of the head and eyes. Gaze stability in unilaterally vestibulopathic subjects was indistinguishable from that of the control subjects (Fig 2), demonstrating that during ambulation any effect of unilateral vestibular loss on gaze, and thus by inference vision, is completely compensated by residual VOR function and limitation of head movement. Although their gaze was significantly less stable than control subjects', subjects with bilateral vestibulopathy nevertheless achieved a gaze retinal slip velocity of 4°/second or less during standing and walking while viewing a visible, distant target (Fig 2). The threshold for loss of visual acuity being image motion of 2° to 4°/second,[19] bilaterally vestibulopathic subjects would not experience much if any loss of acuity caused by poor oculomotor function. Although gaze velocity increased in darkness in all subject groups, the increase was most striking in those with bilateral vestibulopathy (Fig 3), suggesting greater reliance on vision in these subjects.

Head and Trunk Stability

This study extends the finding of poor correlation in control subjects between VOR gain and gaze (retinal slip) velocity during ambulation[8,16] to vestibulopathic subjects, indicating that eye movement is not the only important factor in determining gaze stability. Gaze velocity is poorly correlated with angular VOR gain because gain calculation ignores translational motion, which has a significant effect on gaze stability. Gain of the VOR must be interpreted in the context of angular and translational head movement. One potential strategy to avoid excessive image motion despite vestibulopathy might have been to vary the ratio of head translation to rotation so that gaze stability could be achieved with a lower VOR gain.[8] This strategy was apparently not used because the ratio of head translation and rotation during ambulation was similar in unilaterally vestibulopathic and control subjects. However, vestibulopathic subjects exhibited less overall head translation and rotation, as well as less trunk motion (Fig 5). Limited head motion probably enabled VOR gain of the unilaterally vestibulopathic subjects to be similar to that of control subjects (Fig 4) by avoidance of head movements too fast for the VOR to compensate. Only during high-velocity rotation is VOR gain for ipsilesional head rotation deficient.[13,20]

Unilaterally vestibulopathic subjects exhibited subnormal velocity and amplitude of head motion only during walking (during standing there was a slight increase and during running the decrease was insignificant), and this difference was only significant for motion in the ver-

tical plane. This is probably because the velocity of head motion and the challenge of maintaining gaze were greatest in the vertical plane. Because decreased VOR gain in subjects with unilateral vestibulopathy occurs predominantly at high velocity, it is possible that these subjects only need to minimize vertical head motion. During running, unilaterally vestibulopathic subjects had both vertical and pitch head velocities similar to those of control subjects. Vertical gaze velocity in unilaterally vestibulopathic subjects exceeded that of control subjects for this condition (Fig 2*B*), suggesting that head movement is more difficult to control in a running gait.

In bilaterally vestibulopathic subjects head translation during walking was less than that of control subjects (Fig 5), possibly for the same reason as for the subjects with unilateral vestibulopathy. Despite decreasing head motion, gaze velocity was higher in bilaterally vestibulopathic subjects, probably because of reduced VOR gain.

Effect of Variation in Target Distance

To test the theory that the relationship of head translation to rotation is modulated by controlling the static position of the head relative to the trunk, we measured both head and trunk position. There was no trend in the variation of the head position relative to the body with changes in target distance. This indicates that the control of head movement and its dependence on target distance are mediated through a more complex neural mechanism that does not involve changing the position of the head relative to the trunk.

Performance During Ambulation as a Test of Vestibular Function

Measurement of gaze during treadmill ambulation, as implemented in this study, is a unique test that can determine gaze velocity under environmentally relevant conditions. A previous study demonstrated that both dynamic posturography and VOR gain during whole-body rotation are poor predictors of oscillopsia.[3]

This finding is not surprising because we confirmed here that head movement is as important as VOR gain in determination of gaze velocity.[8] Nevertheless, gaze stability is not a sensitive test of chronic, unilateral vestibulopathy, probably because of adaptive use of vestibular function on the intact side as a form of compensation. Other measures such as sway during dynamic posturography,[16] rapid head thrusts,[12] or caloric testing provide more useful measures to test for unilateral vestibulopathy.

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