Pergamon

PII: S0042-6989(96)00217-9

Vision Res., Vol. 37, No. 6, pp. 789–798, 1997

Printed in Great Britain
0042-6989/97 $17.00 + 0.00

Vertical Eye Position Control in Darkness: Orbital Position and Body Orientation Interact to Modulate Drift Velocity

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Received 16 March 1995; in revised form 4 April 1996

How stable is vertical eye-in-head position control in darkness when no visual targets are present? We evaluated this while varying both body-in-space orientation and eye-in-orbit position in six subjects who were free from oculomotor/vestibular disease. Vertical eye movements were monitored using a CCD-video tracking system, and results were confirmed on one subject with the magnetic search coil. Three body orientations were used: (1) seated upright; (2) supine; and (3) prone. In each of these body orientations starting eye-in-orbit position was varied in quasi-random order from −20 to +20 deg, while vertical eye drift was monitored for a 90 sec period at each position. Subjects were instructed to hold their eyes as steady as possible. The relationship between body orientation/eye position and vertical eye drift velocity was examined using a linear regression technique. In contrast to prior clinical reports, normals exhibit a vertical nystagmus/drift in darkness. Moreover, slow-phase eye velocity was found to be dependent on eye-in-orbit position in the upright and supine body orientations. This pattern of eye drift mirrors Alexander's Law, with significantly increased drift velocities when subjects looked in the direction of their re-centering saccades ($P < 0.05$ or better). Body-in-space orientation also modulated the eye drift velocity, with significant differences in rate of eye drift ($P < 0.05$ or better) between extremes of body orientation (supine and prone) for five out of six subjects. The stability of the vertical oculomotor control system in the absence of visual input is strongly affected by body-in-space orientation and eye-in-orbit position: manipulating either of these variables results in non-random patterns of drift. These results are discussed using a multiple-input model of vertical eye-in-head position control.

Drift Nystagmus Eye position Otolith Gravity

INTRODUCTION

How stable is vertical eye-in-head position control in darkness? The precision of eye-in-head position control when visual targets are present has been explored in detail. Many stimulus parameters have been manipulated, including the size and luminance (Steinman, 1965), shape (St. Cyr & Fender, 1969), color (Boyce, 1967), and retinal eccentricity (Rattle, 1969; Sansbury *et al.*, 1973) of the fixation targets. In brief, it appears that the saccadic system is used to acquire fixational targets, while drift eye movements serve as *slow control* for the stabilization of the oculomotor plant (Steinman *et al.*, 1973).

How eye-in-head position is maintained in the absence of visual feedback (in complete darkness) is not as well understood. Early investigators studied what happens during brief periods of attempted eye position holding in darkness, but they reported substantially different results (Cornsweet, 1956; Nachmias, 1959, 1961). Most agreed that the eye wanders rapidly from a visual target presented previously in the primary position, on both the horizontal and vertical meridia. There appears to be consensus that control of eye position in darkness is accomplished by saccades, unlike visual fixation control (Steinman *et al.*, 1967; Steinman & Cunitz, 1968; Skavenski & Steinman, 1970).

Early papers suggested that when a person attempted to hold the primary position in the dark, the mean value of the average deviation of the position of the eye-in-head from the starting point increased approximately monotonically with time for the first few seconds with no change in the rate of drift, and that horizontal eye position

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seemed to stabilize *ca* 2 deg from the former target position (Cornsweet, 1956) after about 100 sec (Skavenski & Steinman, 1970).

Cornsweet (1956) also suggested that the eye wandered in a random walk, both with and without a target to provide error feedback, and that the rate of this drift did not change in darkness. Matin *et al.* (1970) conducted a study in darkness whose results supported the random walk notion, but they did report a small but significant negative correlation between the direction of eye movements and the direction in which the eyes had wandered from their initial position. They suggested that the "nearly random walk" following the removal of the visual target may describe a transitory state between visual and extraretinal position control of the eye. On the other hand, Nachmias (1959, 1961) found that drift velocities were higher in darkness, and that along some meridia the drifts were corrective in nature, implying a role for drifts in the control of eye position. These findings were supported by later reports which also showed substantially higher drift rates in darkness in both horizontal and vertical meridia (Steinman *et al.*, 1967; Skavenski & Steinman, 1970) and a corrective tendency of the movements made in darkness (Skavenski, 1971).

Fiorentini and Ercoles (1966) reported that in darkness there is a tendency for saccades to the right to follow drifts to the left, implying that there is a velocity correction system. This was an important observation because it implies that an extraretinal signal, from inflow or monitored outflow, gives information to the oculomotor system about the direction of movement during slow drifts. This effect was not found during viewing of a stabilized image, which suggested that when an image is stabilized, the retina gives a false signal of a stationary visual axis. They proposed that this was due to visual inputs overruling the weaker extraretinal signal. Recent work by Epelboim and Kowler (1993) has also shown that drifts are velocity, rather than position sensitive.

It has been known for several years that the non-randomness in the drift of the eyes is much more apparent when a subject attempts to maintain an eccentric eye position. Skavenski and Steinman (1970) asked subjects to hold fixation 10 deg away from the primary position on the horizontal meridian after a visual target had been extinguished. They found that the eyes tended to drift back toward the central starting position in the absence of saccades. They also reported on some vertical data, noting that when fixation targets were extinguished, neither of their two subjects could stay within the narrow recording limits of the vertical photographic optical lever method for >20 sec.

Skavenski and Steinman's (1970) data implied that saccades correct for a mean drift toward the primary position. This suggestion was confirmed by an experiment conducted by Becker and Klein (1973). They had subjects maintain horizontal eccentric eye positions in the dark for angles as big as 70 deg. Their eye movement records showed that the slow drifts of the eyes tended to return them back toward primary position, but that saccades of up to 10 deg corrected for this drift. The velocity of the slow drift towards primary position increased with the eccentricity of eye position, to a maximum velocity of about 10 deg/sec. This demonstrated that extraretinal eye position control is inherently very noisy, but it detects when the eye has wandered too far off target and triggers saccadic corrective movements.

Eizenman *et al.* (1990) expanded on the work by Becker and Klein (1973) by quantifying factors affecting horizontal meridian eye drift in both light and darkness—namely target eccentricity, visual feedback and fatigue. They also proposed an integrated model of the saccadic, smooth pursuit and optokinetic sub-systems based on their results.

While there is a considerable body of literature documenting vertical meridian positional nystagmus (a nystagmus resulting from the head being held in a given position), these papers generally discuss patients with cerebellar lesions or vestibular disturbances of either peripheral or central origin (see Fisher *et al.*, 1983; Lin *et al.*, 1986; Rosenhall, 1988; Leigh & Zee, 1991). In fact, some clinicians report never having seen positional vertical nystagmus in darkness in normals (Barber, 1984).

In this paper we will demonstrate: (1) that normal subjects do exhibit vertical drift/nystagmus in the dark; and (2) that this drift is systematically modulated by the position of the eye-in-orbit and the orientation of the head-in-space. Previous brief reports from this lab on this topic have been published elsewhere (Goltz *et al.*, 1993, 1994).

METHODS

Subjects

Six subjects were tested for this series of experiments, two females and four males. Their ages ranged from 24–52 yr, with a mean age of 30.0 $\pm$ 9.57 yr. All subjects were free from neurological, vestibular, or oculomotor anomalies. Refractive error varied from emmetropic to -8 D of correction.

Apparatus

Vertical eye movements were recorded binocularly at 60 Hz using a headset-mounted CCD video-based cornea/pupil tracking system (El-Mar Series 2020 Eye Tracker, Toronto, Canada). This system is free from drift, and has a linear range of ± 25 deg on the vertical meridian and ± 30 deg in the horizontal, and a maximum resolution of 6 min arc when measured with an artificial eye (for a review see DiScenna *et al.*, 1995). To corroborate the results, vertical eye movements for one subject were also recorded monocularly at 100 Hz with a magnetic search coil system (CNC Engineering) using scleral annulus coils (Skalar).

Calibration

Prior to starting the experiment, each subject was calibrated by recording fixations at seven vertical and

seven horizontal points across a range of ±10 deg both vertically and horizontally at a distance of 2 m. Subjects were seated and their heads were steadied by a chinrest. The rest was adjusted for each subject with the head positioned so that the eyes were looking straight ahead when looking at 0 deg for both vertical and horizontal. The data in all three body orientations used in the experiment were adjusted based on this initial calibration. For the subject who was tested using the magnetic search coil system as well, calibrations were done separately for each body orientation, using five vertical fixation points 10 deg apart across a range of ±20 deg.

Head upright

To avoid visual cues during the experiment subjects were light adapted using room lights and incandescent table lamps prior to the onset of the experiment. Once this was accomplished the subjects' heads were draped in black felt cloth to remove all visual cues. As a further precaution, the room lights were dimmed to prevent light leakage. When the subjects reported complete darkness, the experiment was started. After sitting motionless in darkness for 1 min to allow possible semicircular canal inputs to subside, the eye movement recording began. The eye position of the subjects was monitored by the experimenter using a real-time display of eye position. Subjects were given verbal feedback until they acquired the appropriate starting eye-in-orbit position. Upon acquiring the desired starting position subjects were instructed to hold their eyes as steady as possible, and to try not to move them. Nine starting positions of eye-in-orbit were used, going from −20 to +20 deg in 5 deg steps. The order of the starting position trials was randomized for each subject. Each recording period lasted 90 sec, after which the subjects were given a short break.

Supine and prone body orientations

Subjects were also tested in supine (nose-up) and prone (nose-down) body orientations to explore the effect of gravity on attempted eye position holding in darkness. Body orientation was controlled in these trials by holding the subject rigidly using a manually operated Stryker Frame bed, which was designed to immobilize patients with spinal cord injuries. Once the subjects were calibrated, they were oriented in the frame and their entire upper body was covered in black felt to prevent light leakage. For these experiments, five eye-in-orbit starting positions were used: −20 to +20 deg in 10 deg steps. As in the upright condition, the order was randomized. The order of upright, supine and prone orientation trials was also quasi-randomized for all subjects.

Data processing and analysis

All eye movement records collected for each condition were analyzed off-line using a Macintosh PC. Eye movement data were low-pass filtered (10 Hz) and then velocity estimates were obtained by differentiating the data using a three point differentiator. Saccades, blinks and artifacts were then removed manually. No other data were excluded from the analysis. The mean of the remaining instantaneous drift velocities was calculated across each 90 sec trial, yielding one vertical drift velocity estimate for each starting eye-in-orbit position examined in this paper. Alternatively, mean velocity was calculated for each of the intersaccadic intervals, and the mean of those intervals was then calculated. These two techniques yielded the same results. In the text, tables and figures of this paper "−" always means downward eye movements, while "+" always refers to upward ones.

RESULTS

General observations on the nature of the slow drift

The instruction to the subjects not to move their eyes appears to have been an impossible task. In the absence of visual input the eyes drifted uncontrollably in all subjects tested. The waveforms produced by attempted holding of eye-in-head position in darkness varied from slow drift to a nystagmic pattern. Given the infeasibility of the requested task, the subjects appear to have interpreted the instructions not to move their eyes as "maintain the assigned starting eye-in-orbit position", which was accomplished largely by resetting saccades. The drift of the eyes was dependent on the position of the eye-in-orbit, with the nystagmus most evident when the subjects looked up in the orbit, no matter which body orientation. The slow drift/fast return pattern of eye movements began immediately after extinguishing the room lights, and did not abate for as long as 4 min (the maximum recording time implemented). The slow phase of this nystagmus exhibited a near-linear waveform (see Fig. 1 for representative segments of the raw data). In general, looking 20 deg downward produced the least drift [Fig. 1(A)], while looking upward by 20 deg produced the largest effect [Fig. 1(C)]. Horizontal vergence changes did not systematically affect the rate of vertical drift, as can be seen in Fig. 2. In any case, subjects were not able to maintain a consistent vergence state in complete darkness.

Re-centering saccades were also examined. In this non-visual condition the saccades corrected for deviation from the starting eye-in-orbit position caused by drift of the eyes. Since the rate of drift is dependent on orbital position and body orientation as described above, it follows that the rate of re-centering saccades would be expected to increase with increased drift. Saccade rates were systematically higher when the subjects' eyes were held higher in the orbit. As an example, when subject JS held her eyes 20 deg down in the orbit, she made 55 saccades in 90 sec, with an average beat frequency of 0.6 Hz. When her eyes were held at 0 deg she made 86 saccades in 90 sec, with an average beat frequency of 0.95 Hz. With her eyes starting at 20 deg up in the orbit, 105 saccades were made over a 90 sec recording period, averaging 1.16 Hz. All saccades could be considered compensatory since they opposed the direction of drift.

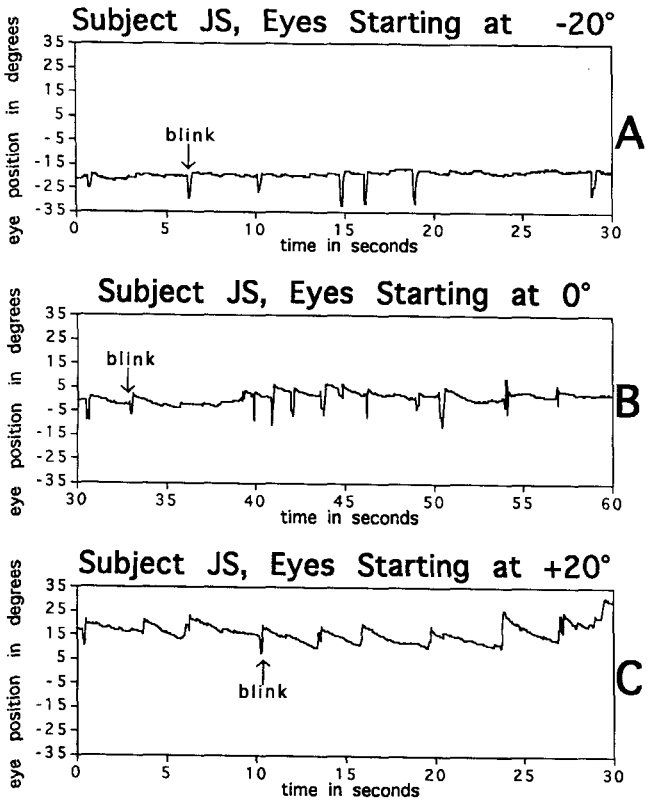

FIGURE 1. Thirty seconds of vertical eye movements collected on subject JS in the head upright orientation. Three different starting orbital positions are shown. (A) Subject looking 20 deg downward. Note the near-absence of drift. (B) Subject looking straight ahead. Drift is prominent, but diminished compared to 20 deg upward starting eye position below. (C) Subject looking 20 deg downward. Note the near-nystagmic waveforms.

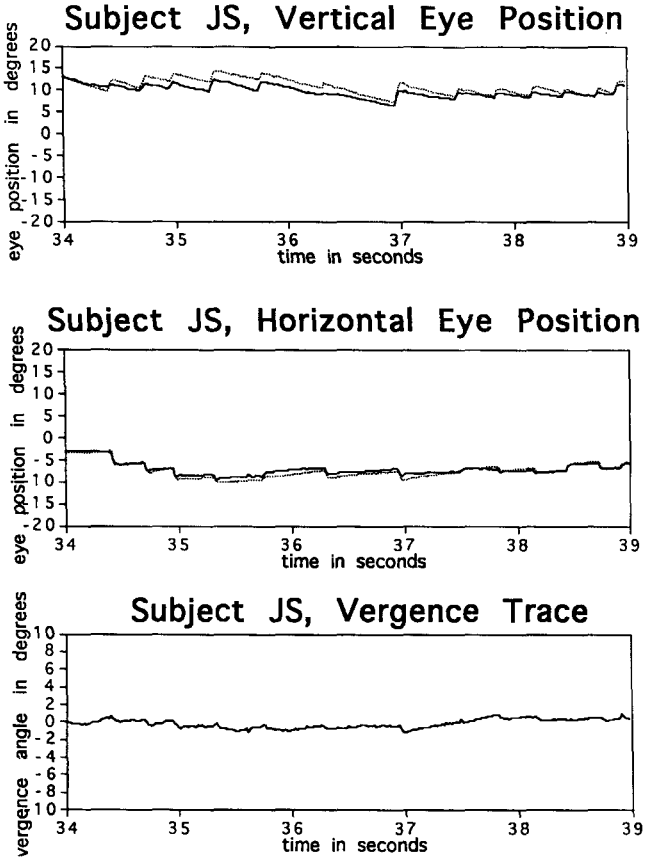

FIGURE 2. Five second binocular vertical, horizontal, and vergence records. Note that changes in vertical drift rate are not dependent on changes in vergence.

All subjects showed a similar pattern of relative saccade frequency, but amplitudes and absolute frequency differed. Saccade amplitude was also strongly correlated with drift rate, as can be seen in Fig. 1: the re-centering saccades are necessarily much larger in Fig. 1(C) (frequently >4 deg), where the eyes are oriented 20 deg up in the orbit, than in Fig. 1(A), where the eyes are oriented 20 deg down in the orbit.

Analysis of eye drift

Each 90 sec trial at a given eye-in-orbit position produced one mean slow-phase eye velocity value per eye and a corresponding mean eye-in-orbit position value. For each of the three body orientations, a simple linear regression of drift velocity on eye position was carried out initially. This involved a minimum of nine velocity/position data points for head upright, and five points each for supine and prone body orientations. Summary plots of the position/velocity relationships for all six subjects in all three body orientations are included below (see Fig. 3). From these plots it is evident that there is both an effect of vertical eye-in-orbit position, and an effect of body orientation on vertical drift velocity. Standard errors were calculated for each drift velocity estimate, but in most instances the error bars were completely obscured by the symbols used to plot the points. The horizontal component of the drift was also examined in these three body orientations in two of the six subjects, but there was no effect of body orientation on horizontal drift rate.

The drift measured in these subjects can be subdivided into two components. The first is a constant drift component (CD), defined as the drift velocity while attempting to hold the eyes straight ahead, as interpolated from the linear regressions fitted to the data. This value corresponds to the intercept of the regression. The second is an eye-in-orbit dependent drift component (EDD), described by the slope of the linear regression. It should be noted that these measures are not equivalent units (CD is in deg/sec, while EDD is in deg/sec/deg) and therefore cannot be summed. It is possible, however, to calculate a drift for a given eccentricity and then sum it with the CD to calculate overall drift. This type of analysis has been used to describe horizontal extraretinal eye position control in normals (Becker & Klein, 1973). The slope represents the change in velocity associated with a one degree change in eye position. Depending on the CD, the subject's total drift may be symmetrical with respect to the primary position (CD = 0) or biased upwards or downwards by the CD. In some instances the CD and the effect of the EDD can oppose each other. For the subjects studied it was possible, in most instances, to calculate a null position in the orbit, or equilibrium eye angle (EEA) where the CD and the influences of the EDD cancel each

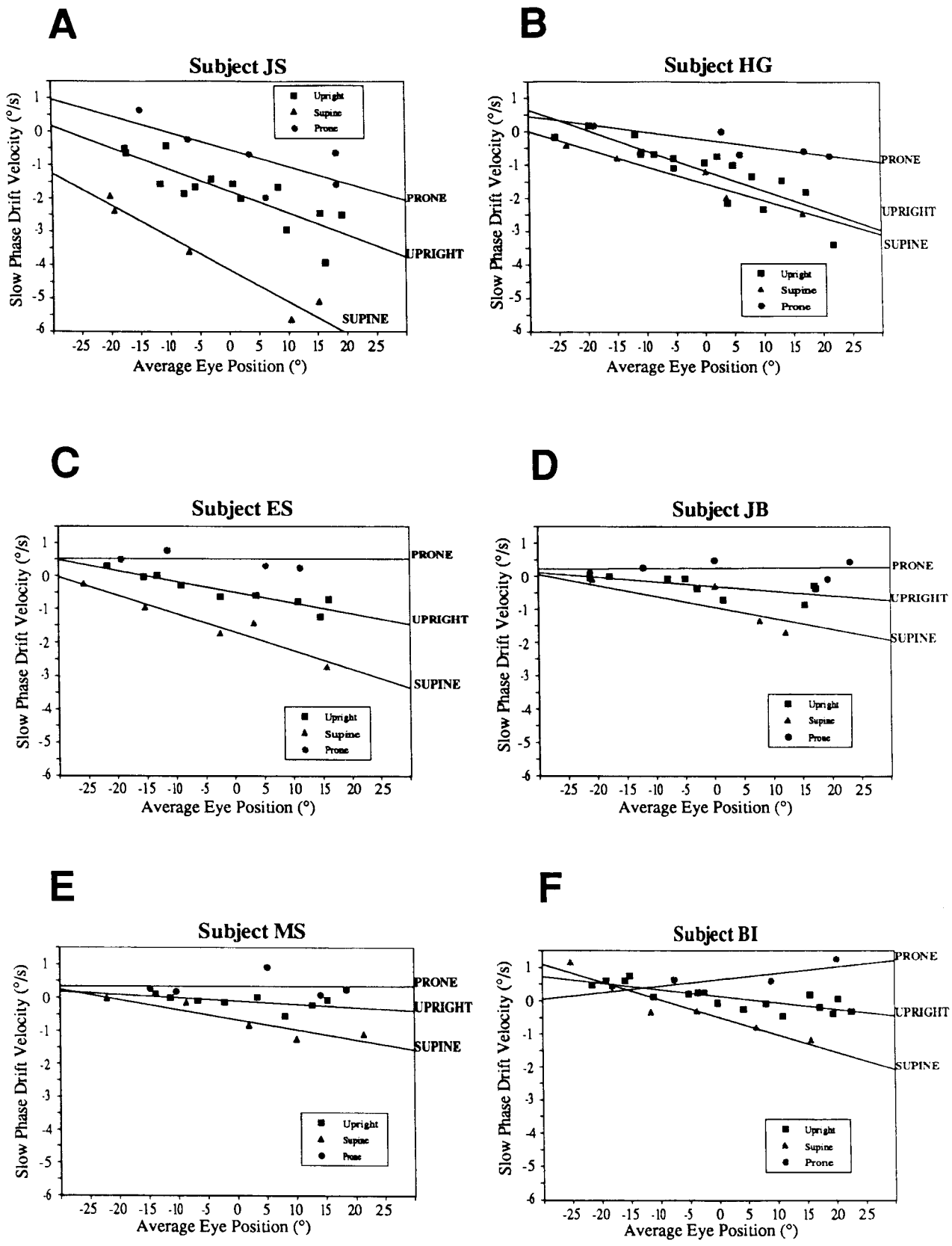

FIGURE 3. Summary plots of vertical drift for the six subjects studied. Average eye position across 90 sec trial is plotted on the x-axis, while slow-phase vertical drift velocity for the same time period is plotted on the y-axis. (A) Subject JS. Linear regression fits to data points as follows: prone, $y = -0.051x - 0.557$ $r^2 = 0.51$; upright, $y = -0.065x - 1.808$ $r^2 = 0.71$; supine, $y = -0.096x - 4.139$ $r^2 = 0.95$. (B) Subject HG. Linear regression fits to data points as follows: prone, $y = -0.023x - 0.234$ $r^2 = 0.72$; upright, $y = -0.060x - 1.165$ $r^2 = 0.75$; supine, $y = -0.051x - 1.549$ $r^2 = 0.93$. (C) Subject ES. Linear regression fits to data points as follows: prone, $y = -0.000x + 0.509$ $r^2 = 0.00$; upright, $y = -0.032x - 0.490$ $r^2 = 0.87$; supine, $y = -0.055x - 1.695$ $r^2 = 0.93$. (D) Subject JB. Linear regression fits to data points as follows: prone, $y = -0.001x + 0.230$ $r^2 = 0.01$; upright, $y = -0.014x - 0.313$ $r^2 = 0.42$; supine, $y = -0.033x - 0.954$ $r^2 = 0.68$. (E) Subject MS. Linear regression fits to data points as follows: prone, $y = -0.000x + 0.337$ $r^2 = 0.00$; upright, $y = -0.010x - 0.110$ $r^2 = 0.29$; supine, $y = -0.030x - 0.663$ $r^2 = 0.84$. (F) Subject BI. Linear regression fits to data points as follows: prone, $y = +0.019x + 0.629$ $r^2 = 0.57$; upright, $y = -0.020x + 0.134$ $r^2 = 0.65$; supine, $y = -0.053x - 0.487$ $r^2 = 0.84$.

other and no drift occurs. This point could be determined by dividing the slope into the intercept of the regression for any body orientation. Null points within the oculomotor range are also common in clinical patients with nystagmus (for a review see Dell'Osso, 1993).

Simple analysis of variance (ANOVA) was performed on each body orientation for each subject, testing the null hypotheses that the CD velocity = 0 deg/sec and that the eye-in-head dependent drift velocity = 0 deg/sec/deg. The results of the multiple regression and ANOVA for all three body orientations are summarized in Table 1.

Body upright

In the upright body orientation, all six subjects exhibited a baseline level of CD. These velocities were idiosyncratic, and they ranged from −1.81 deg/sec to +0.13 deg/sec across subjects (Table 1). In five out of six subjects, this CD was found to be statistically significant beyond the $P = 0.01$ level (ANOVA). The variable, eye-in-orbit dependent component of the drift/nystagmus ranged from −0.065 deg/sec/deg to −0.010 deg/sec/deg, and was significant beyond the $P = 0.05$ level in four out of six subjects (ANOVA). In the upright body orientation all the EDD was downward. Null points for eye drift varied from 27.85 to 6.50 deg down in the orbit (see Table 1).

Body supine

When the same subjects were in the supine orientation, a CD was evident in most cases: the drift rate while looking straight ahead, as interpolated from the linear regression varied from −4.14 to −0.49 deg/sec across subjects (Table 1). In five of six subjects the drift rate was significantly different ($P < 0.05$) from 0 deg/sec, as tested by ANOVA. The eye-in-orbit dependent component of the drift was significant in the same five subjects ($P < 0.05$), and varied from −0.096 to −0.030 deg/sec/deg across subjects. It should be noted that drift velocities for both CD and EDD were higher in most instances in the supine orientation than in the body upright orientation. This resulted in null points for eye drift that were even lower in the orbit, corresponding to eye positions that varied from 9.25 to 43.13 deg down in the orbit (see Table 1).

Body prone

When subjects were held in the prone orientation in the Stryker frame eye drift was also evident. One striking finding was that more than half of the subjects showed a direction reversal of CD when looking straight ahead—four subjects drifted upwards at between +0.23 and +0.63 deg/sec (see Table 1). The other two subjects continued to drift downwards, but at a much diminished rate of between −0.23 and −0.56 deg/sec. It should be noted that only the two largest up-drifting subjects had CD velocities that were significantly different ($P < 0.05$) from 0 deg/sec, as tested by ANOVA. The EDD in the prone body orientation was not found to be significant in any of the six subjects. Although the variability of the data in the prone orientation was not significantly higher than the points in the supine orientation in most instances, the slopes were smaller due to the lack of significant EDD, resulting in lower r^2 values and this yielded poor results in the ANOVA. Since the eye-in-orbit dependent component of the drift was not significant, some subjects did not exhibit a null point for drift within their oculomotor range (see Table 1).

Effect of eye-in-orbit position: drift rate depends on eye position

As well as the constant component (CD) of the drift when the subjects looked straight ahead, subjects also showed a variable EDD. In most subjects this component of the drift velocity was found to depend on the position of the eye-in-orbit, in a fashion that mimics Alexander's Law. In general, when subjects looked in the direction of their re-centering saccades the velocity of the drift increased. Alexander's Law states that a nystagmus due to a central or peripheral vestibular lesion is more intense when the patient looks in the direction of the re-centering saccades (Leigh & Zee, 1991). It should be noted that the six subjects tested in this experiment were vestibular and oculomotor normals, and we are not attributing the positional dependence of the drift to a pathological state.

TABLE 1. Average prone, upright and supine 0 deg eye position vertical drift velocities (CD), vertical EDD (increase of drift per degree of eye eccentricity), and vertical EEA between CD and EDD, or the null point for drift velocity

	CD (deg/sec)			EDD (deg/sec/deg)			EEA (deg)		
Subject	Prone	Upright	Supine	Prone	Upright	Supine	Prone	Upright	Supine
JS	−0.56 NS	−1.81**	−4.14**	−0.051 NS	−0.065**	−0.096**	−10.98	−27.85	−43.13
HG	−0.23 NS	−1.17**	−1.55**	−0.023 NS	−0.060**	−0.051**	−10.00	−19.50	−30.39
ES	+0.51*	−0.49**	−1.70**	−0.000 NS	−0.032**	−0.055**	†	−15.31	−30.91
JB	+0.23 NS	−0.31**	−0.95 NS	+0.001 NS	−0.014 NS	−0.033 NS	†	−22.14	−28.91
MS	+0.34 NS	−0.11 NS	−0.66*	0.000 NS	−0.010 NS	−0.030*	†	−11.00	−22.10
BI	+0.63*	+0.13**	−0.49*	+0.019 NS	−0.020*	−0.053**	+33.16	−6.50	−9.25

CD, constant drift; EDD, eye-in-orbit dependent drift; EEA, eye equilibrium angle. These data are based on the linear regression of drift velocity on eye position for each subject individually.

*$P < 0.05$; **$P < 0.01$.

†Equilibrium angle is indeterminate since slope is not significant or zero.

NS, not statistically significant (ANOVA).

TABLE 2. Interaction between vertical eye position and body orientation, as calculated by analysis of covariance (ANCOVA)

	CD (intercept) differences between upright and prone		CD (intercept) differences between supine and prone		EDD (slope) differences between upright and prone		EDD (slope) differences between supine and prone	
Subject	Parameter estimate	P	Parameter estimate	P	Parameter estimate	P	Parameter estimate	P
JS	−1.466	<0.01	−3.797	<0.01	−0.018	NS	−0.049	<0.05
HG	−0.931	<0.01	−1.316	<0.01	−0.037	<0.05	−0.029	NS
ES	−0.999	<0.01	−2.204	<0.01	−0.032	<0.01	−0.055	<0.01
JB	−0.544	<0.01	−1.184	<0.01	−0.015	NS	−0.034	<0.01
MS	−0.447	<0.01	−1.000	NS	−0.010	NS	−0.031	<0.05
BI	−0.494	<0.01	−1.115	<0.01	−0.039	<0.01	−0.072	<0.01

NS, not statistically significant (ANCOVA). These data are based on the linear regression of drift velocity on eye position for each subject individually. Other abbreviations as in Table 1.

In general, in the head-upright body orientation all six subjects drifted downward at their maximum rate when the starting position of the eye was 20 deg up in the orbit. Drift velocities tended to be smaller when the CD and EDD components opposed each other, such as when the starting position of the eyes was 20 deg down in the orbit. A similar relationship was reported for horizontal drift by Becker and Klein (1973).

Interaction of orbital position and body orientation

Besides the main effects of eye position on drift velocity reported above, it was also found that eye-in-orbit and body-in-space orientations interacted to modulate the drift velocity of the eyes. An analysis of covariance (ANCOVA) technique was used which regressed drift velocity on eye position for the three body orientations—upright, supine, and prone (see Table 2). This procedure tested two null hypotheses:

1. The difference between the intercepts for any two given body orientations = 0 deg/sec; and
2. The difference between the slopes for any two given body orientations = 0 deg/sec/deg.

The difference between intercepts (or primary position drift velocities) for upright and prone body orientations was found to be significant beyond the $P = 0.01$ level for all six subjects. The differences in primary position drift velocities (CD) between supine and prone body orientations were found to be statistically significant beyond the $P = 0.01$ level for five out of six subjects as well. The differences in slope (or EDD) between supine and prone were found to be significant beyond the $P = 0.05$ level for five out of six subjects, while the slope differences between upright and prone were only found to be significant in three of six subjects, but once again this is likely due to the lack of EDD in the prone body orientation.

A comparison of data collected with CCD-video tracker and magnetic search coil

The CCD-video based tracker was used to collect data on all the subjects reported on in this paper. These results were confirmed by repeating the experiment on subject HG using a magnetic search coil technique. The results obtained were qualitatively similar: the waveforms of the raw data agreed with the video-based data, and the linear regressions produced similar slopes and the relative order of the lines fitted to the different body orientations was the same. There was, however, some discrepancy between the values of the intercepts generated by the linear regression fitted to the data produced by the two methods. The video method tended to produce smaller intercepts (slower drift velocities at primary position) than the coil method. In this instance, we feel that the video tracker produced the superior data, since the same calibration (and hence the same relative 0 deg position) was used for all three body orientations, whereas the search coil had to be re-calibrated for each body orientation.

DISCUSSION

Our results demonstrate that significant vertical drift does occur in normals in darkness, in contrast to clinical reports (Barber, 1984). Moreover, in the upright and supine body orientations these drifts followed a pattern equivalent to Alexander's Law, with slow phase drift velocities that increased in magnitude when the eyes were turned away from the direction of drift. It should be noted, however, that all of our subjects were free from neurological or ocular disease, and that all of our testing was done in complete darkness without any visual feedback. The magnitude of the drift velocity varied widely across subjects, but the general pattern of drift modulation with changes in body orientation and eye-in-orbit position was the same for all subjects tested. Manipulating body orientation produced predictable drift patterns, with the least drift in the prone orientation, an intermediate amount in the upright orientation, and the most when subjects were oriented in the supine orientation. Our analysis separated the drift into constant and eye-in-orbit dependent components. In the supine orientation, both the CD and the EDD were most pronounced for all subjects.

We propose that the effects described in this paper are the result of a number of inputs to and forces acting on the oculomotor control system, and we will discuss each of these potential contributing factors separately. We feel that the drift patterns that we have reported can be modeled by including the following contributing factors,

TABLE 3. Summary of possible components which combine to create vertical drift patterns

Body orientation	Eye-in-orbit position (deg)	Passive effect of gravity	Neural compensation for passive effect	Orbital centering mechanism	Vestibular centering to horizon (otolith drives)
Supine	−20	Updrift	Down drive	Up	Down drive
	0	—	—	—	Down drive
	+20	Downdrift	Up drive	Down	Down drive
Upright	−20	Updrift	Down drive	Up	—
	0	Updrift	Down drive	—	—
	+20	Updrift	Down drive	Down	—
Prone	−20	Downdrift	Up drive	Up	Up drive
	0	—	—	—	Up drive
	+20	Updrift	Down drive	Down	Up drive

Assumptions: (1) passive effect of gravity: center of mass of eye behind center of rotation. (2) Neural compensation: response to eye muscle pull (Gauthier *et al.*, 1990; Knox and Donaldson, 1991). (3) Vestibular centering mechanism: independent of eye-in-orbit position. (4) Net result: additive nature of components—assume relative weighting of components helps explain individual differences.

and that individual differences in drift rate can be explained by differential weighting of the factors. For a summary of possible contributing effects and their directions in the three body orientations and the extremes of eye position, see Table 3.

The passive effect of gravity and possible neural compensation for this effect

The centers of mass and rotation are non-coincident in the human eye (Steinbach & Lerman, 1990; Steinbach, 1992). Steinbach and Lerman (1990) reported that the center of mass of the eye is behind the center of rotation, based on passive eye deviations in response to head tilt in roll in paralyzed and anesthetized patients undergoing surgery. It may be the case that the vertical drift reported here is caused in part by gravity acting on the natural dipole in the eye. There is some evidence, however, that the extraocular muscles in alert subjects are too powerful to be perturbed by the relatively small torque on the eyes: Robinson (1964) noted that increasing the moment of inertia of the eye by 96.5 times with weights results in an overshoot of only 18% on a 10 deg saccade, implying that the mass of the eye is easily overcome by the extraocular musculature. It should be noted, however, that Robinson's experiment involved visually guided saccades, and it is not clear how the eyes would respond in the absence of visual feedback. If the passive effect of gravity does play a role in the ocular drift patterns that we are reporting, it may be the case that individual differences in morphology may influence the relative contribution to the effect. Indeed, Steinbach and Lerman (1990) reported that only two-thirds of their subjects showed a passive effect of gravity on resting eye position. An exploration of individual differences in orbital structure would be sensible. We are currently examining this issue.

If there is an effect of gravity on the eyes of alert subjects, there may be a compensatory mechanism that corrects for perturbations of the eye in response to the force of gravity. While a traditional short latency stretch response in extraocular muscle has not been demonstrated (Keller & Robinson, 1971), there is a growing literature which suggests that the brain receives feedback about passive perturbations of the eye. Gauthier *et al.* (1990) demonstrated that deviating a covered human eye using a suction scleral contact lens resulted in visual localization errors of *ca* 17% of the extent of deviation in the same direction as the eye-pull. This result is also supported by the work of Knox and Donaldson (1991), who reported vestibulo-ocular reflex modulation in response to deviation of the non-seeing eye of the pigeon. The relatively small changes elicited by applying force to the extraocular muscles suggest that if neural compensation for passive effects of gravity is taking place, this response would certainly not compensate fully for the deviation of the eye-in-orbit.

Orbital centering mechanism and EDD

When the eye is moved to an extreme position within the orbit, there is a tendency for it to return to a more central position (Becker & Klein, 1973; Eizenman *et al.*, 1990; Eizenman & Sharpe, 1993). Maintaining eye position against the elastic forces of the orbit appears to depend on the tonic innervation of the extraocular muscles by motor neurons. This innervation is controlled by the tonic neurons, whose activity is the output of a cellular network known as the neural integrator (Skavenski & Robinson, 1973). It may be the case that our results for EDD can be explained partially in terms of a leaky neural integrator which transforms the eye velocity commands into eye position commands. In most instances, when our subjects were asked to hold their eyes in eccentric positions in the dark, there was a tendency for the eyes to drift back toward the center. Because the integrator leaks, the eyes return with an exponential time course. If the integrator becomes very leaky (the time constant becomes small) the slow-phase drift velocity drift will be large and the subject will exhibit EDD. It appears that the body orientation modulates the rate at which the integrator leaks. The changes in drift velocity with body orientation are summarized in Table 1.

While the drift we measured has similar properties to vertical and horizontal end-point nystagmus, it differs in several key ways. End point nystagmus is normally found

at eye-in-head angles ranging from 25 to 65 deg of eccentricity (Eizenman *et al.*, 1990), while the drift that we measured was present when the subjects looked to a maximum eccentricity of *ca* 25 deg. Substantial drift was also measured in all body orientations when the subjects looked to 0 deg of eccentricity (see Table 1), which also argues against end-point nystagmus as a contributing mechanism in this instance.

The CD bias

With the exception of the otolith–ocular reflex horizon-centering mechanism detailed below, all the mechanisms described above contribute to the eye-in-orbit dependent component of the eye drift that we measured. To complete this model, we need to include a mechanism that would drive the eyes when attempting to hold the eyes straight ahead in darkness as is documented in Table 1. The mechanisms behind the CD which recorded when subjects were looking straight ahead are not clear. It may be the case that these drifts are simply indicative of inherent imbalances in the drives to the extraocular muscles, without the benefit of visual feedback to stabilize eye-in-head position. Other workers have reported that these drifts could be increased by mental set (Robinson *et al.*, 1984), suggesting that the effect is indeed neural. Others have suggested other influences: similar patterns of drift have been demonstrated by researchers examining the effects of cigarette smoking. Sibony *et al.* (1987) postulated that nicotine induces the primary position upbeat nystagmus they recorded during tobacco smoking by means of excitation of the central vestibular pathways. It is unclear, however, what could be exciting the vestibular pathways during our experiment, where only eye position was varied. None of the subjects included in this study were smokers. We are presently evaluating the effects of mental load on drift velocity with changes in body orientation.

Vestibular centering to the horizon

As we reported above, slow-phase drift velocity is clearly modulated by changes in body orientation. This is most likely due to the effect of gravity, but it is unclear whether this is due to gravity altering the outputs of the vestibular end-organs, or whether this effect is related to gravity physically acting on the eye-in-orbit (see above). We feel that we can exclude semicircular canal stimulation as a contributor of the drift patterns we are reporting, as our subjects were immobilized for at least 1 min before the onset of recording, and were not moved during the trials. It may be the case that the changes in body orientation result in differing tonic drives of the otolith organs to the oculomotor plant (Ebenholtz & Shebilske, 1975). These tonic drives are part of a compensatory mechanism known as the otolith-ocular reflex (Leigh & Zee, 1991), and they serve to center the eyes with respect to the horizon in response to static tilts of the body. The prone orientation would result in an upward drive to the eyes in response to a forward tilt of the body. In the upright condition, there would be no need for centering to the horizon. When the body is tilted backward to the supine orientation, the eyes would be driven upward in their sockets. This otolith drive would be independent of the eye-in-orbit position, and therefore we would expect it to affect the CD only, which would be manifest as a change in the intercept in the linear regression, which is what we found (see Table 1). It should be possible to assess the role of static otolith drives by measuring drift in various body orientations in patients with known lack of otolith function (e.g. bilateral acoustic neuroma surgery patients). We are currently exploring this possibility.

Summary of passive effects and neural drives

We feel that the total effects of body orientation and eye-in-orbit position on slow-phase drift velocity reported in this paper can be modeled using the mechanisms detailed above (see also Table 3). It should be noted that this model is not meant to be a linear additive model with equal weights, rather it is meant to summarize the possible components of the effects we are reporting here.

The orbital centering mechanism is probably responsible for the Alexander's Law-like relationship of eye position to drift velocity. This pattern of drift and compensation would be modulated by the passive effect of gravity on the eye-in-orbit, and by neural compensation for this passive force on the eye. Both of these effects would also be dependent on eye-in-orbit position as well, thereby contributing to the EDD. Unlike the orbital centering mechanism, however, these two effects would be modulated by changes in body orientation, which would implicate them in the changes in slope associated with the different body orientations. The otolith–ocular reflex would explain the significant differences in the CD rate across body orientations, and would not contribute to the eye-in-orbit dependent portion of the drift. The rather large individual differences evident between subjects (see Fig. 3) may be explained by differential weighting of the individual components of the model. We are currently testing these possibilities.

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Acknowledgements—The authors would like to thank Roman Konarski from the Institute for Social Research for his assistance with the statistical analysis of the data. We also wish to thank Rob Allison and Jim Zacher for their expert technical assistance with the collection of the magnetic search coil control data and Ian Howard for lending us the coil system. Helpful comments were made by two anonymous reviewers on an earlier version of the manuscript. Some subjects were given movie passes that were generously donated by Cineplex Odeon Theatres of Canada. This research was supported by an NSERC operating grant to MJS (A7664), MRC of Canada grants to ELI and ME, Ontario Graduate Scholarship to HCG and a grant from the Jackman Foundation.