Sensory-motor factors triggering the suppression of post-rotary vestibular responses in different gravitoinertial force backgrounds

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Summary. We studied suppression of oculomotor and perceptual after-responses by post-rotary head movements in high (1.8 G), low (0 G), and normal (1 G) gravitoinertial force backgrounds in parabolic flight. Our aim was to identify what sensory and motor signals are critical for triggering suppression. In a prior experiment (DiZio and Lackner 1988), we found suppression using 40° post-rotary head tilts in 1 G and 1.8 G but not 0 G force backgrounds. However, in free fall even without head tilts there was a significant suppression of nystagmus relative to 1 G and 1.8 G force backgrounds, thus potentially masking an effect of head tilt on suppression in 0 G. We have retested four of the original subjects with 90° head tilts to maximize the likelihood of detecting suppression in 0 G. Although nystagmus and illusory after-rotation were suppressed by post-rotary head tilts in normal and high gravitoinertial force environments, the evidence was still no evidence of suppression in free fall. We present evidence that the lack of suppression in 0 G is not attributable to post-rotary responses already being at a "baseline" level, but rather that suppression depends on the registration of a change in head position relative to a significant level of gravitoinertial force.

Key words: Vestibular – Head movements – Gravitoinertial force – Post-rotary nystagmus – Illusory self-motion – Velocity storage – Motion sickness – Human

Introduction

On Earth, the durations of illusory after-rotation (Purkinje 1820) and after-nystagmus elicited by sudden termination of constant velocity rotation are shortened by head tilts (Benson and Bodin 1966a). Such head tilts also evoke more symptoms of motion sickness than when the head is kept still during the post-rotary period (Benson and Bodin 1966a, 1966b; DiZio and Lackner 1988; Guedry 1965; Purkinje 1820). The suppression of post-rotary nystagmus by head tilts has been called "dumping", and can be modeled as a rapid discharge of a velocity storage mechanism (Cohen et al. 1977; Raphan et al. 1977; Robinson 1977) owing to sudden closure of a negative feedback pathway across a neural integrator (Raphan et al. 1979). An intact cerebellar nodulus and uvula are necessary for dumping (Waespe et al. 1985), but it is unclear which sensory-motor signals associated with post-rotary head tilt activate dumping and whether the same signals are implicated in the elicitation of motion sickness when it accompanies dumping.

It appears that a reorientaion of the head relative to gravity is critical for activating dumping (Benson and Bodin 1966a, 1966b; Guedry 1965). The otolith organs can signal such reorientation (Fernandez and Goldberg 1976), and signals from the neck reflecting reorientation relative to gravity may also be sufficient to elicit dumping during active head movements (Schrader et al. 1985a, 1985b). The former is possible even though the presence of dumping during post-rotary tilt of the head and torso as a unit means that cervical signals are not necessary (e.g. Benson and Bodin 1966a). When the background gravitoinertial force (0 G) level is zero, otolithic receptors cannot signal a static reorientation of the head, but two other factors are present. Independently of gravitoinertial force level during active post-rotary head tilts, the semicircular canal afferent signals will indicate the presence of inertial head velocity not in the same plane as velocity storage activity, and, in addition, the movement of the head relative to the torso will elicit afferent and efferent information about the changing head position.

To determine which of these factors may activate the dumping mechanism and be important for elicitation of motion sickness symptoms, we have been studying how oculomotor and perceptual responses to post-rotary head tilts are influenced as a function of gravitoinertial force level in parabolic flight experiments. We found that without post-rotary head tilt, the dominant time constant of decay of horizontal vestibular after-nystagmus was significantly shorter in the high force (1.8 G peak) and low force (0 G) phases of parabolic flight than in straight-and-level flight (1 G) (DiZio et al. 1986, 1987). The time constant was further shortened by post-rotary head tilts of

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40° amplitude in 1 G and 1.8 G but not in 0 G (DiZio and Lackner 1988; Lackner and DiZio 1986, 1988). The duration of perceived self-rotation followed the same pattern.

It is important to note that head tilts following rotation in 0 G did not generate graviceptor information concerning the reorientation but did generate information about inertial head velocity and head movement relative to the torso; moreover, such head movements did not suppress either post-rotary nystagmus or illusory after-rotation, and were only mildly nauseogenic. By contrast, similar 40° head tilts in 1 G altered the tonic graviceptor activity level even more than in 1 G; and they also suppressed post-rotary nystagmus and illusory after-rotation more than in 1 G and were much more nauseogenic. From these considerations, it would appear that information about inertial head velocity and head movement relative to the torso are not sufficient to trigger dumping, and that afferent and efferent activity signalling a change in head position relative to a significant gravito-inertial force vector are required.

In the studies just described, voluntary 40° pitch-back head movements were used in the conditions in which subjects made post-rotary head tilts. In the present study, we measured eye movements and illusory self-motion elicited by sudden stops followed by voluntary 90° pitch-back head movements. The major reason for testing with larger amplitude head movement is that the lack of nystagmus suppression by smaller post-rotary head tilts in 0 G may reflect a basement effect. That is, because the time constant of horizontal vestibular nystagmus decay is already low in 0 G without head tilts (10.4 s) a 40° head tilt may not elicit any further decrease whereas a 90° head tilt might (Schrader et al. 1985a, 1985b). If a 90° head tilt were to elicit dumping in 0 G, then a stimulus other than change in head position relative to the direction of the G vector would have to be causing it. Stimuli that are not contingent on head position relative to gravity have been identified as important in several aspects of the response to sudden stops. For example, DiZio and Lackner (1989) have proposed that one’s perceived orientation after post-rotary head tilts is partially determined by registration of a change in head position relative to the torso, not simply by the plane of canal activity relative to gravity.

Methods

Subjects

The subjects were four individuals who had participated, at least 14 months earlier, in the experiments involving 40° post-rotary head tilts. All had normal vestibulo-ocular reflex gains and symmetries despite past exposure to vestibular stimulation in the laboratory and parabolic flight.

Parabolic flight profile

Experimental observations were made during the steady state 0 G, 1G and 1.8G force level periods of parabolic flight. The parabolic flight maneuvers are illustrated and described in prior publications (cf. DiZio and Lackner 1988).

Procedure

We used the same apparatus and a procedure similar to that of our prior experiment (DiZio and Lackner 1988), except that the movement endpoints for post-rotary head movements were different. A lightweight device mounted to a rotating chair confined head movements to the pitch plane. Mechanical stops were set to limit head movement amplitude to 90°. The movement endpoints varied across subjects for the sake of comfort; the starting head position (position during rotation and the sudden stop) ranged from 55° to 45° pitched forward from the perceived upright, and the final position ranged from 35° to 45° pitched backwards. Head movements were not required were conducted with the head locked in the 20° forward or 45° forward positions. A few seconds prior to the sudden stop subjects were reminded (a) either to keep their head still or, if instructed, to move it to the opposite endpoint, (b) to look straight ahead of the head into the distance and avoid voluntary fixation or saccades, (c) to press a hand-held button when the sense of after-rotation ceased. In flight, each subject completed at least two trials in each direction, for each G-level, and for each head movement condition. Ground-based 1 G trials were conducted prior to in-flight sessions or at least eleven days post-flight.

Eye position was recorded using conventional electro-oculography with appropriate calibration procedures. Data recording and analysis were carried out in the fashion previously described (DiZio and Lackner 1988). Four out of 157 trials were rejected from statistical analysis because the time constants deviated by more than 2.5 standard deviations from the group mean.

Results

Every sudden stop elicited illusory self-rotation about the body’s z-axis and horizontal nystagmus beating opposite to the direction of prior rotation. Figure 1 displays typical eye movement data records after sudden stops with and without head tilts. In trials without post-rotary head tilts, the nystagmus slow phase velocity rose to a peak value after a mean delay of 1.2 s following the sudden stops and then decayed toward zero at a G dependent rate. Horizontal slow phase velocity usually declined transiently during a post-rotary head tilt (mean duration of head tilt, 0.8 s) and then returned toward or above its previous magnitude after the head movement ceased (mean latency from the sudden stop to the end of head tilts, 1.7 s); velocity then decayed toward zero at a rate that was G dependent and quicker than when the head was not moved.

Peak slow phase velocity occurred in trials without post-rotary head tilts at a time when eye velocity was temporarily suppressed in the trials with head movements. Therefore, we decided to measure peak velocity and to initiate the computation of the time constant of slow phase velocity decay 0.5 s after a head movement had ended or after an equivalent latency from the sudden stop if no head movement had been made. This results in a slight underestimation of peak velocity for trials without head movements but provides the best comparison across conditions. Repeated measures analyses of variance were used to assess the overall effects of G level (0G, 1G on the ground, 1 G in flight, 1.8 G) and head movement condition (head fixed 20° ventralflexed, head fixed 45° ventralflexed, head tilted 90° back starting at about 45° ventralflexed) upon peak slow phase eye velocity, time
constant of slow phase eye velocity decay, and duration of illusory after-rotation. Sheffe contrasts ($p<0.05$, at least) were used to compare specific subsets of the data.

Table 1 presents the results for peak slow phase velocity. Head tilt condition affected peak velocity ($F(2,6)=7.18$, $p<0.026$) but G level did not. Peak velocity was higher when the head remained fixed at $20^\circ$ ventriforned after sudden stops than when the head was fixed at $45^\circ$ ventriforned or was tilted backward $90^\circ$ from the $45^\circ$ position. There was no difference in peak velocity between the latter two conditions.

Table 2 presents the time constants of slow phase velocity decay. Significant effects were present for G level ($F(3,9)=5.8$, $p<0.041$), head movement condition ($F(2,6)=15.8$, $p<0.004$), and an interaction of the two ($F(6,18)=5.33$, $p<0.003$). After sudden stops with the head fixed at $20^\circ$, the time constant was shorter in $0 \, G$ ($10.1 \, s$) than in both $1 \, G$ in flight ($16.8 \, s$) and $1.8 \, G$ ($13.5 \, s$); it was also shorter in $1.8 \, G$ than in $1 \, G$. Similarly, with the head remaining fixed at $45^\circ$, the time constant was shorter in $0 \, G$ ($11.4 \, s$) than in $1 \, G$ ($14.7 \, s$) and $1.8 \, G$ ($15.1 \, s$); there was no difference between the latter two. In $1 \, G$ in flight

Fig. 1. Typical plots displaying 20 s of vestibular nystagmus in 1 G following sudden stops from $60^\circ$ without post-rotary head tilt (top) and with (bottom). The dark bar indicates a head tilt. The top plot of each trial shows eye position, the middle plot instantaneous eye velocity with the quick phases clipped at zero, and the bottom plot shows the average velocity of each nystagmic slow phase on a logarithmic scale. The slope of the regression line on the bottom plots is proportional to the time constant of slow phase velocity decay (T); the line’s value 0.5 s after a head movement ends or after an equivalent latency in the trial without a head movement, is the peak slow phase velocity (MAX).
Table 1. Peak slow phase velocity (°/s) of post-rotary nystagmus in different G levels and with and without 90° head tilts. N=4, $\bar{X}$=mean, $S_s$=standard error

<table>
<thead>
<tr>
<th>Head tilt condition</th>
<th>0 G</th>
<th>1 G (ground)</th>
<th>1 G (flight)</th>
<th>1.8 G</th>
</tr>
</thead>
<tbody>
<tr>
<td>No tilt, 20° ventr</td>
<td>$\bar{X}$</td>
<td>34.4</td>
<td>29.9</td>
<td>33.1</td>
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<tr>
<td></td>
<td>$S_s$</td>
<td>(4.32)</td>
<td>(5.16)</td>
<td>(4.68)</td>
</tr>
<tr>
<td>No tilt, 45° ventr</td>
<td>$\bar{X}$</td>
<td>23.4</td>
<td>24.3</td>
<td>25.3</td>
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<tr>
<td></td>
<td>$S_s$</td>
<td>(4.03)</td>
<td>(4.36)</td>
<td>(4.98)</td>
</tr>
<tr>
<td>90° tilt back from 45° ventr</td>
<td>$\bar{X}$</td>
<td>20.5</td>
<td>23.6</td>
<td>20.7</td>
</tr>
<tr>
<td></td>
<td>$S_s$</td>
<td>(4.00)</td>
<td>(4.76)</td>
<td>(3.81)</td>
</tr>
</tbody>
</table>

Table 2. Time constant (s) of slow phase velocity decay of post-rotary nystagmus in different G levels with and without 90° head tilts. N=4, $\bar{X}$=mean, $S_s$=standard error

<table>
<thead>
<tr>
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<th>0 G</th>
<th>1 G (ground)</th>
<th>1 G (flight)</th>
<th>1.8 G</th>
</tr>
</thead>
<tbody>
<tr>
<td>No tilt, 20° ventr</td>
<td>$\bar{X}$</td>
<td>10.1</td>
<td>15.0</td>
<td>16.8</td>
</tr>
<tr>
<td></td>
<td>$S_s$</td>
<td>(1.99)</td>
<td>(2.90)</td>
<td>(1.98)</td>
</tr>
<tr>
<td>No tilt, 45° ventr</td>
<td>$\bar{X}$</td>
<td>11.4</td>
<td>16.7</td>
<td>14.7</td>
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<td></td>
<td>$S_s$</td>
<td>(2.61)</td>
<td>(2.92)</td>
<td>(2.24)</td>
</tr>
<tr>
<td>90° tilt back from 45° ventr</td>
<td>$\bar{X}$</td>
<td>10.0</td>
<td>8.7</td>
<td>8.6</td>
</tr>
<tr>
<td></td>
<td>$S_s$</td>
<td>(1.79)</td>
<td>(1.99)</td>
<td>(1.66)</td>
</tr>
</tbody>
</table>

Table 3. Duration (s) of illusory after-rotation in different G levels with and without 90° post-rotary head tilts. N=4, $\bar{X}$=mean, $S_s$=standard error

<table>
<thead>
<tr>
<th>Head tilt condition</th>
<th>0 G</th>
<th>1 G (ground)</th>
<th>1 G (flight)</th>
<th>1.8 G</th>
</tr>
</thead>
<tbody>
<tr>
<td>No tilt, 20° ventr</td>
<td>$\bar{X}$</td>
<td>13.5</td>
<td>17.3</td>
<td>15.6</td>
</tr>
<tr>
<td></td>
<td>$S_s$</td>
<td>(3.10)</td>
<td>(5.88)</td>
<td>(2.54)</td>
</tr>
<tr>
<td>No tilt, 45° ventr</td>
<td>$\bar{X}$</td>
<td>8.7</td>
<td>14.8</td>
<td>12.1</td>
</tr>
<tr>
<td></td>
<td>$S_s$</td>
<td>(2.85)</td>
<td>(3.14)</td>
<td>(3.85)</td>
</tr>
<tr>
<td>90° tilt back from 45° ventr</td>
<td>$\bar{X}$</td>
<td>8.2</td>
<td>13.3</td>
<td>11.1</td>
</tr>
<tr>
<td></td>
<td>$S_s$</td>
<td>(4.32)</td>
<td>(2.06)</td>
<td>(3.47)</td>
</tr>
</tbody>
</table>

and 1.8 G, making 90° pitch back post-rotary head tilts led to significant comparable reductions in the time constant relative to when the head remained fixed at 45°. Such head movements did not shorten the 0 G time constant. The time constants obtained after head tilt were longer in 0 G (10.0 s) than in 1 G in flight (8.6 s) and 1.8 G (8.7 s).

Table 3 presents the durations of illusory after-rotation for the test conditions. There were main effects of G level (F(3, 9) = 4.74, p < 0.029) and head movement condition (F(2, 6) = 111.66, p < 0.0001). The duration of illusory after-rotation was longer overall in 1 G on the ground than in 1 G in flight. In flight with the head fixed at 20° ventrified, the duration of illusory after-rotation was shorter in 0 G (13.5 s) than in 1 G (15.6 s) or 1.8 G (15.2 s). This was also true when the head was fixed at 45° ventrified, the durations being 8.7 s in 0 G, 12.1 s in 1 G, and 12.6 s in 1.8 G. Making 90° pitch back head tilts shortened the duration of illusory after-rotation significantly in 1 G and 1.8 G.

Discussion

We have found in the present study that 90° head tilts suppress post-rotary perceptual and oculomotor responses in 1 G and 1.8 G but not in 0 G. The lack of reduction in the time constant in trials with head tilts in 0 G is not just a basement effect because the lowest values observed in 1 G and in 1.8 G are lower than every value observed in 0 G. This implies that the elicitation of suppression relies on registration of head tilt relative to a "gravity" vector. Factors which are not alone sufficient to activate dumping include moving the head out of the plane of post-rotary nystagmus and changing the head's position relative to the torso because these events occurred in our 0 G trials with head movements but there was no suppression.

These conclusions are supported by data summarized in Fig. 2, which presents the time constants of slow phase decay of eye velocity obtained in the current study, along with those from a previous study (DiZio and Lackner 1988). There are three independent test conditions not involving post-rotary head tilts, but involving different static head positions relative to the torso and to the resultant force, being 20° in two of them and 45° in the other. Within each G level, there were no systematic differences in the time constants. This weighs against the magnitude of post-rotary nystagmus suppression being primarily a function of static initial or final head position relative to gravity. This is a key point because, it is known that head orientation relative to gravity modulates the vestibulo-ocular reflex (Raphan et al. 1981; Fetter et al. 1986) but this modulation is much too small to account for the attenuating effect of post-rotary head tilts on the time constant. This lack of significant differences also means that the potential differences in nystagmus decay attributable to different combinations of canals being stimulated with the head in different orientations is not significant here. Figure 2 also shows that in 1 G and in 1.8 G larger amplitude head tilts produce bigger reductions in the decay constant of post-rotary nystagmus.

The registration of head reorientation relative to a non-zero gravitoinertial force could be accomplished by the otolith organs and possibly cervical afferent and efferent elements. The otoliths transduce the shear forces created by moving and holding the head in a gravitoinertial force field and the cervical proprioceptors transduce the torques and loads engendered. Neural pathways for carrying information necessary for suppression from the
Fig. 2. Bar graph of the time constant of slow phase velocity decay of nystagmus following sudden stops with and without post-rotary head tilts in high, low and normal gravitational force levels in the present experiment and in our prior experiment. The error bars indicate two standard errors in an. - 20°, O - 20° Tilt, 20° (DiZio and Lackner 1988), □ - No Tilt, 45°, □ - 40° Tilt. (DiZio and Lackner 1988), ■ - 90° Tilt

macular and cervical receptors to the brainstem and cerebellum have been reported (see Bakker and Abrahams 1988; Fuller 1988; Wilson and Melvill Jones 1979 for reviews). The changed muscle activation levels necessary to support the head at different positions may also be implicated (Lackner and DiZio 1989a, 1989b). Terrestrial experiments in which a subject's entire body is passively tilted (Benson and Bodin 1966b) suggest that registration of the tilt by the otothol organs is sufficient for suppression, but it is still possible that information about head tilt relative to gravity from cervical proprioceptors would also be sufficient by itself. It is important to identify what sensory-motor events are sufficient to trigger dumping because they may also trigger the accompanying motion sickness symptoms.

As a final point, our data contain an important replication of results from one of our previous experiments. Figure 2 shows that after sudden stops with no head movements, both in this and in the previous experiment (DiZio and Lackner 1988), there is an effect of G level on the decay constant of the vestibulo-ocular reflex. The time constant is shorter in 0 G and in 1.8 G than in 1 G. The duration of illusory after-rotation also follows this pattern. Nevertheless, there is no effect of G level on peak eye velocity. This replication is important because the result was not originally expected on theoretical grounds and because many previous attempts to measure the effects of gravitational force level on the vestibulo-ocular reflex have produced conflicting results or results that are not comparable because of methodological differences or too little data (D'elong and Oosterveld 1986; de Jong et al. 1983; Benson and Vieville 1986; Jackson and Sears 1963; Vesterhauge et al. 1984; Vesterhauge et al. 1982). Our observations mean that when gravitoinertial force equals zero, the normal tendency for velocity storage to cross-couple to the plane orthogonal to the G-vector (Raphan and Cohen 1987, 1988) and, thereby, to augment the vestibulo-ocular reflex in that plane is lacking. This may explain why making yaw (the plane usually orthogonal to gravity) head movements in the free fall phase of parabolic flight evokes more symptoms of motion sickness than keeping the head still (Lackner and Graybiel 1984; Lackner and Graybiel 1986). Other factors differentially affecting pitch and roll head movements may explain why they are more nauseogenic than yaw.

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