

## Orientation of Vestibular Nystagmus is Modified by Head Tilt

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A visual image needs to be kept stable on the retina for visual processing to operate effectively (1,2): movements of the head present a threat to this stability. During head movements in the dark, the eyes make compensatory movements that are generated by the vestibuloocular reflex (VOR), and in the light, the visual system is able to make up for any inadequacies in the vestibular response. In general, the most important stabilizing influence upon the vestibular system is that which arises from stimulation of the semicircular canals. However, various experiments have also indicated that the otolith organs can generate approximately compensatory eye movements when stimulated by linear accelerations (3–5), although their role in head-eye coordination is not yet clear.

When making natural head movements, the otolith organs must be continually stimulated by changes in orientation of the head with respect to gravity and may thus modify or augment the response from the canals. Indeed, in the monkey (6) and cat (7), the otoliths appear able to take over some of the functions of the canals. During constant rotation about an earth vertical axis, the compensatory nystagmus induced by lateral-canal stimulation decays exponentially, whereas rotation about an axis inclined to the vertical induces a sustained compensatory nystagmus. The sustained response is apparently evoked by otolithic stimulation (8) and develops slowly to match the decaying response of the canals (6). In man, however, rotation about a tilted axis evokes a rather different response. A continuing undirectional nystagmus is usually evoked, but it is of low velocity and is heavily overlaid with a sustained sinusoidally modulated nystagmus (9–12). In an attempt to elucidate the functional significance of such a response, we have conducted further tilted-axis rotation experiments in which simultaneous recordings of both lateral and vertical eye movements have been made in human subjects. The preliminary results presented here appear to indicate that one role of the otolithic response is to modify pre-existing lateral eye movements when the head is tilted so as to direct the resultant eye movement more toward true earth-horizontal.

## METHODS

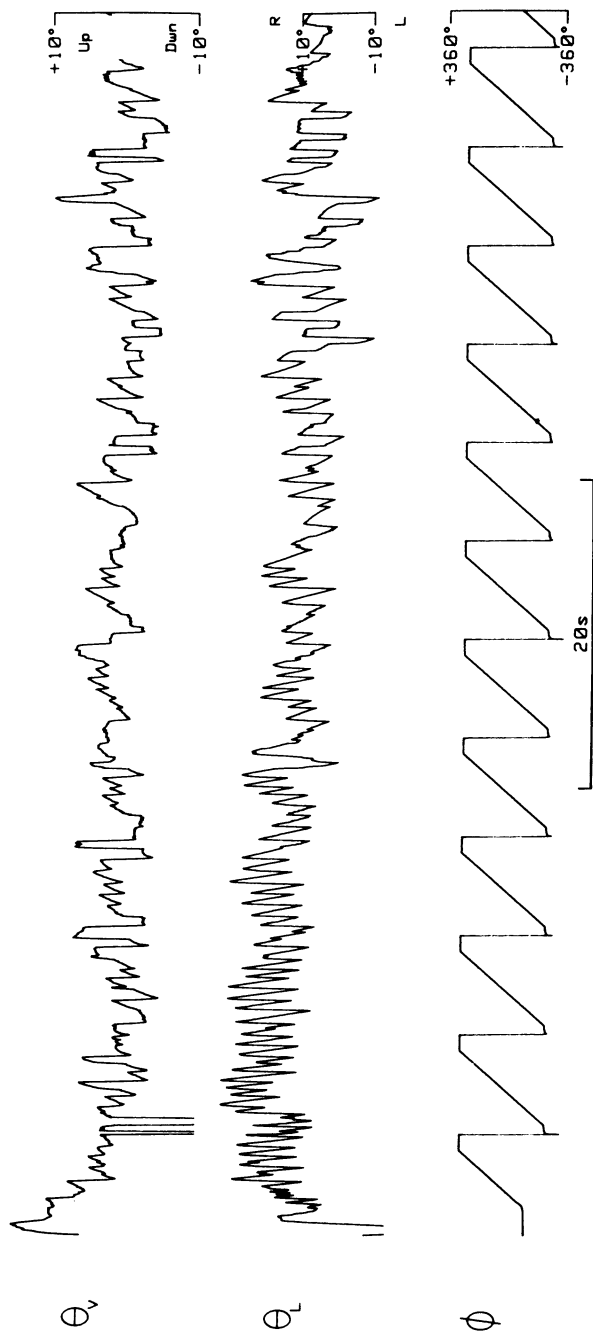
Seven subjects were exposed to continuous rotation at 60°/sec about the longitudinal body axis which was tilted 30° from earth vertical. During and after rotation, lateral and vertical eye movements were monitored simultaneously by the scleral search coil technique (13). The subjects' heads were firmly clamped within a rigid nonmetallic framework which also housed the field coils for the eye movement transducers. This framework was rigidly coupled to the turntable, so the whole assembly rotated with the turntable, the head thus remaining in a fixed position with respect to the field coils. Subjects were rotated in total darkness at constant velocity for 90 sec, following which they were brought to an abrupt halt with either their right or left ear downward. Rotation was either to the left or right, and controls were carried out with rotations about an earth vertical axis. Because of the combination of the discomfort of wearing the scleral coil and the nauseogenic nature of the stimulus, complete results were obtained from only four subjects.

## RESULTS

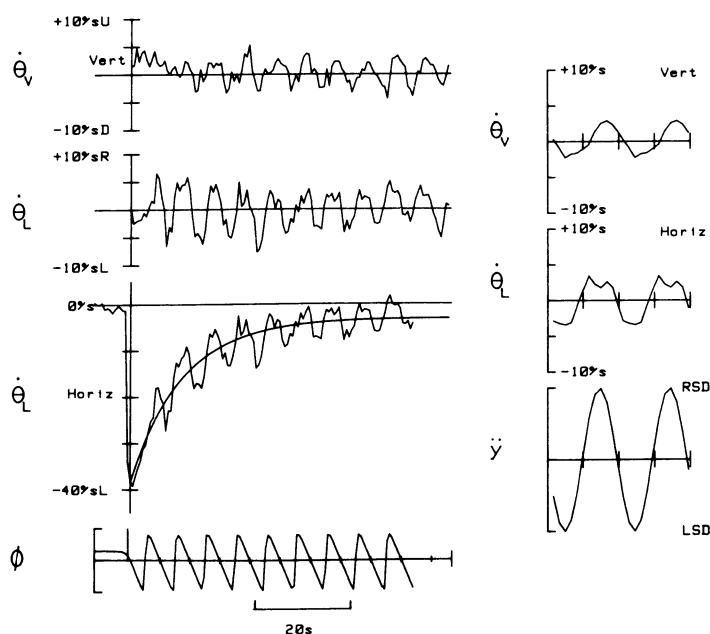
### Per-Rotational Responses

At the onset of rotation, lateral eye movements were evoked with a mean peak velocity of 25.4°/sec ( $SD \pm 10.8^\circ/\text{sec}$ ). There was no significant difference in peak velocity between rotations about the vertical axis and rotation about a tilted axis. When rotation was about the vertical axis, the slow-phase velocity of the induced nystagmus declined to zero with an approximately exponential time course (mean time constant 19.3 sec  $SD \pm 7.6$  sec). However, when rotation was about an axis tilted at 30° to the vertical, the response declined not to zero but to a steady state in which lateral eye movements (Fig. 1) exhibited a nystagmus with a sinusoidally modulated slow-phase velocity. This modulated component was superimposed on a constant-velocity "bias" component, the direction of which was opposite that of the head rotation.

An example of the slow-phase velocity of the lateral eye movements is shown in Fig. 2. Analysis was carried out by fitting an exponentially decaying function, representing the lateral canal response, to the slow-phase velocity trajectory, as indicated in Fig. 2. This enabled the time constant of decay and the magnitude of the bias component to be determined. The time constant had a mean value of 16.5 sec ( $SD \pm 5.6$  sec), which was somewhat less than for rotation about the vertical, but the difference was not significant. The bias component had a mean value of 3.2°/sec ( $SD \pm 2.3^\circ/\text{sec}$ ), and this represented a rather ineffectual compensatory response with a gain of only 0.05. After removal of the exponential component from the slow-phase velocity, it could clearly be seen that the sinusoidally modulated component was present from the onset of rotation and exhibited a periodicity (6 sec) equivalent to the rate of rotation. The mean amplitude of the sinusoidal



**FIG. 1.** An example of the vertical (upper trace— $\theta_v$ ) and lateral eye movements (center trace— $\theta_L$ ) evoked by constant rotation at  $60^\circ/\text{sec}$  to the right about an axis tilted  $30^\circ$  to earth vertical. Bottom trace indicates turntable position( $\phi$ ).



**FIG. 2.** The slow-phase eye velocity derived from the example of Fig. 1. From bottom to top the traces are as follows. On the left: turntable position ( $\phi$ ); lateral eye velocity ( $\dot{\theta}_L$ ) (with best-fit exponential); the sinusoidal component of lateral eye velocity (after removal of exponential component); and vertical eye velocity ( $\dot{\theta}_v$ ). On the right: equivalent linear acceleration stimulus in lateromedial direction ( $\ddot{y}$ ); cycle-by-cycle average of sinusoidal component of lateral eye velocity ( $\dot{\theta}_L$ ); and similar average for vertical velocity ( $\dot{\theta}_v$ ).

component was  $\pm 2.73^\circ/\text{sec}$  ( $\text{SD} \pm 2.3^\circ/\text{sec}$ ). The large standard deviation in the bias and sinusoidal components reflected the wide variability in response, one subject in particular exhibiting almost no sinusoidal or bias component.

The phase relationships were calculated with reference to the effective linear acceleration stimulus (bottom RH trace, Fig. 2). Thus the effective peak acceleration to the left was provided when the subject was right-side down (RSD). Lateral eye velocity (after removal of the bias component) reached a peak that was slightly phase advanced (mean value  $19.7^\circ$   $\text{SD} \pm 25.5^\circ$ ) on this acceleration stimulus.

In addition to the lateral eye movements, vertical eye movements were observed (Fig. 1) that also exhibited a sinusoidal modulation in eye velocity (Fig. 2) upper traces. The mean amplitude of vertical eye velocity was only  $1.4^\circ/\text{sec}$  ( $\text{SD} \pm 0.89^\circ/\text{sec}$ ) and was thus approximately half that of the peak lateral eye velocity. There was also a small vertical bias component in the upward direction (mean  $1.2^\circ/\text{sec}$   $\text{SD} \pm 0.9^\circ/\text{sec}$ ). The phase relationship between vertical eye velocity and the effective lateral acceleration stimulus was dependent on the direction of rotation. When referred to the accelera-

tion stimulus in the anteroposterior direction, this dependency on rotational direction was eliminated, so that peak velocity in the downward direction was phase-advanced on nose-up position (equivalent to peak forward acceleration) by a mean angle of  $14^\circ$  ( $SD \pm 43.8^\circ$ ).

### Post-Rotational Responses

When rotation of the turntable was brought to an abrupt halt, a vigorous lateral nystagmus was evoked (Fig. 3) with a slow-phase velocity that decayed exponentially (Fig. 4). The rate of this decay was found to be significantly ( $p < 0.01$ ) less following rotation about the vertical axis (mean time constant 24.2 sec;  $SD \pm 5.6$  sec) than when the subject was brought to a halt in a side-down position after tilted-axis rotation (mean 11.5 sec;  $SD$  5.0 sec). However, initial peak velocity was not significantly modified by stopping position, having an overall mean of  $23.4^\circ/\text{sec}$  ( $SD \pm 11.2^\circ/\text{sec}$ ).

Of particular interest was the observation that in all subjects, a vertical nystagmus was also evoked after cessation of rotation about the tilted axis (Fig. 3). The direction and magnitude of the vertical slow-phase velocity depended both on which ear was downward and on the direction and magnitude of the simultaneous lateral eye movements. Examples of the responses to the four combinations of side-down position and lateral eye movement directions are shown in Fig. 4. The direction of the lateral component is, as expected, in the same direction as the preceding rotation. On the other hand, the vertical nystagmus was always appropriately directed so as to bring the resultant eye movement more toward earth horizontal. The example in Fig. 4a serves to illustrate this effect. Because the head is tilted at  $30^\circ$  to the vertical with the right side down, the right-going lateral eye movement actually has a downward component with respect to earth horizontal. However, the vertical eye movement compensated for this by directing the eye upward.

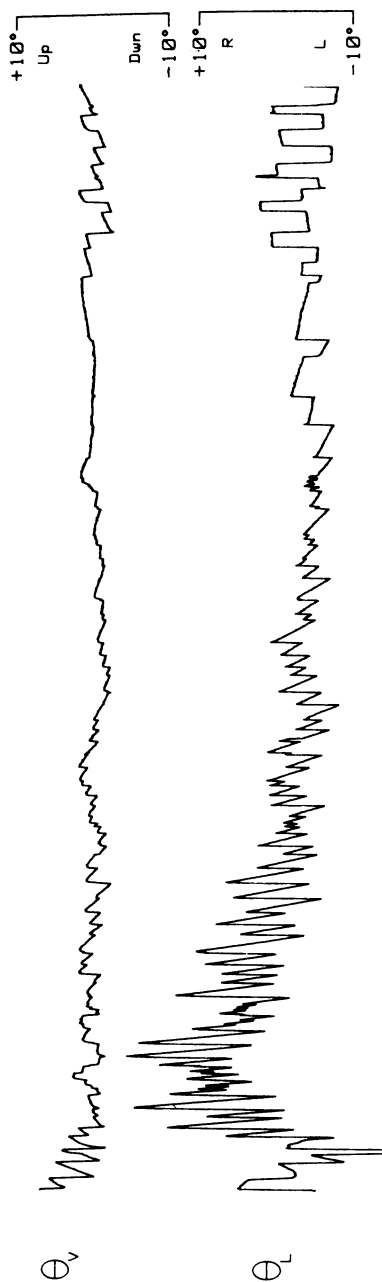
The vertical component built up rather slowly, reaching a peak after about 10 sec, but then gradually decayed in the same manner as the lateral eye velocity. As a consequence, the resultant direction of eye movement remained relatively constant. This effect is illustrated in Fig. 5, where the resultant magnitude and phase with respect to the lateromedial axis of the head have been calculated from the lateral and vertical eye movement trajectories. The phase of the resultant, after a slow buildup, approaches a constant level, which in some examples is very close to  $30^\circ$ , the angle required to compensate for the imposed head tilt.

Although the responses shown in Fig. 4 are from a single subject, qualitatively similar responses were obtained from all the subjects tested. However, there was considerable variation in the magnitude of the vertical eye movements evoked.

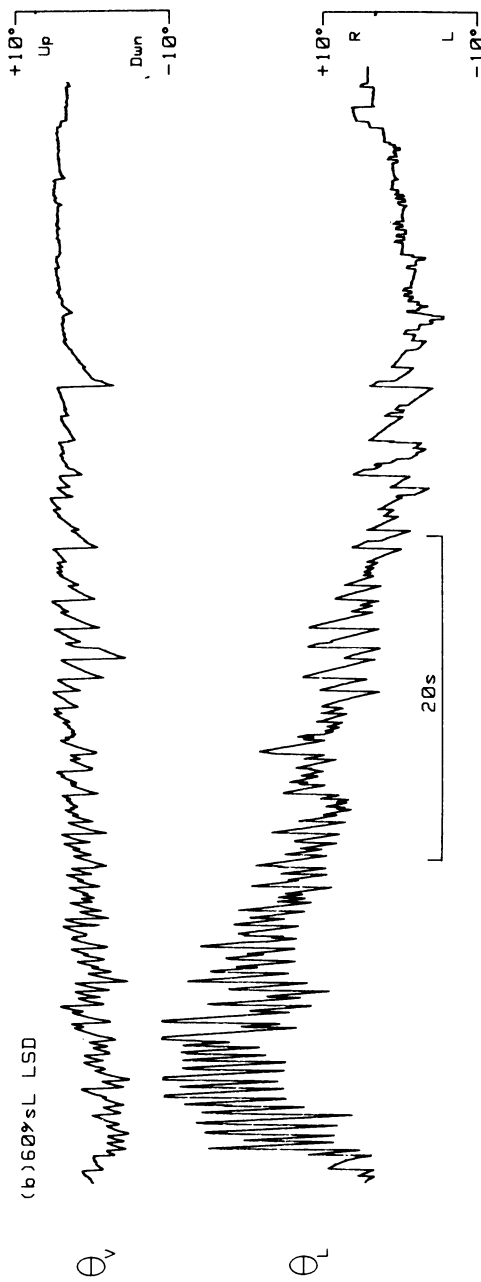
### DISCUSSION

During rotation about an off-vertical axis, the responses exhibit two clearly separable characteristics—a sinusoidal modulation and a constant-

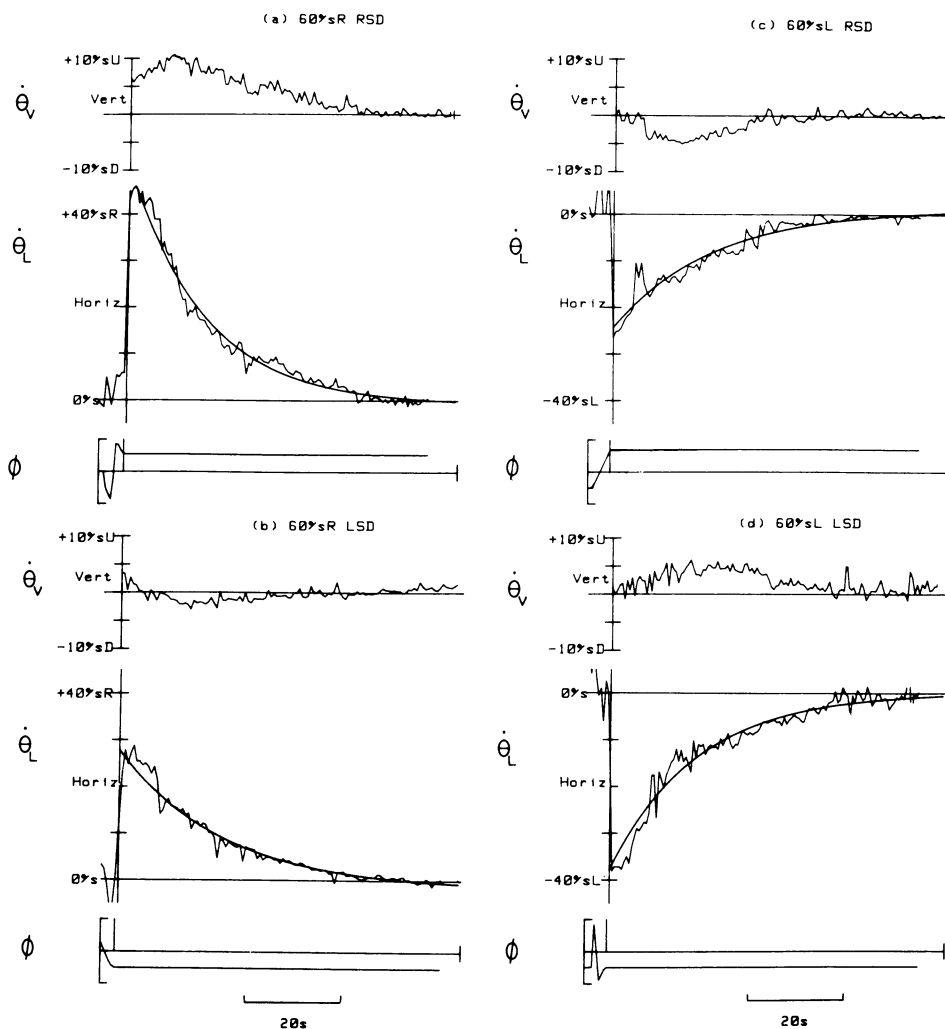
(a) 60°sL RSD



(b) 60°sL LSD

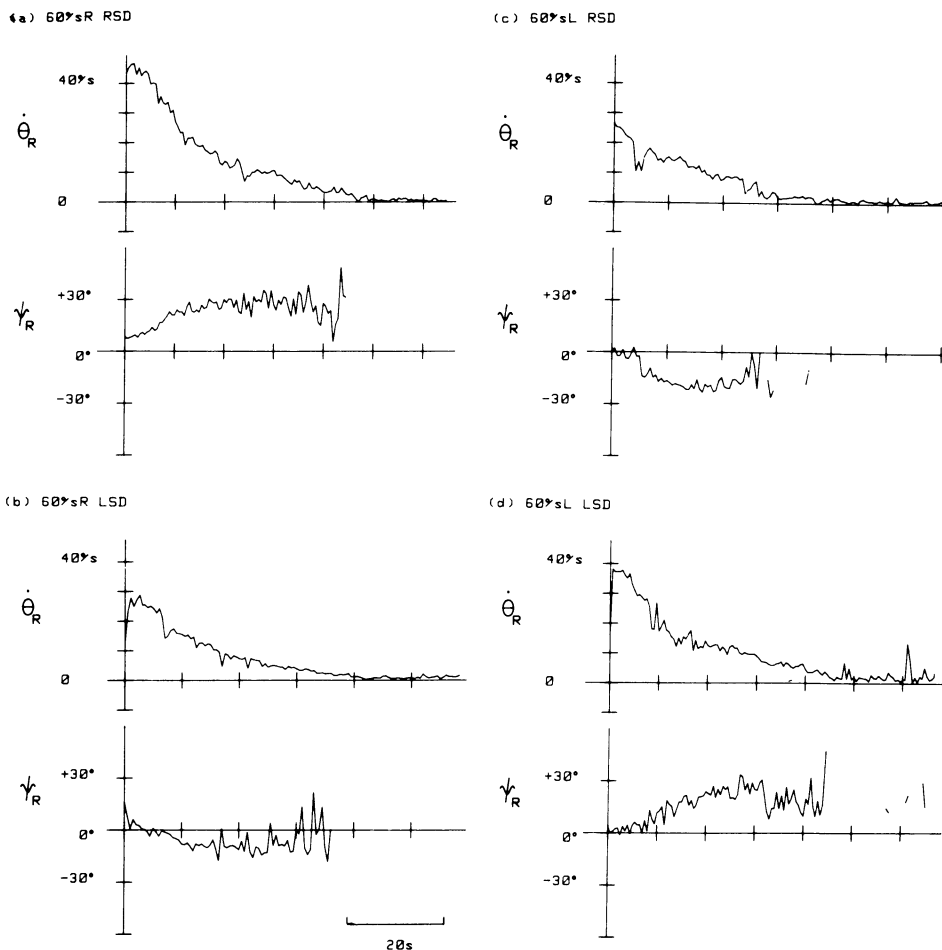


**FIG. 3.** Examples of vertical ( $\theta_v$ ) and lateral ( $\theta_L$ ) nystagmus evoked by the cessation of rotation to the left at 60°/sec about an axis tilted 30° to the vertical. In (a) the final resting position was right side down (RSD), whereas in (b) it was left side down (LSD).



**FIG. 4.** The vertical ( $\dot{\theta}_v$ ) and lateral ( $\dot{\theta}_l$ ) eye velocity evoked by cessation of rotation about a tilted axis. Rotation was to the right in (a) and (b) and to the left in (c) and (d). Final resting position was right (RSD) or left (LSD) side down.

velocity bias component which is generally in the opposite direction to that of the rotation. In the monkey (6) and in the cat (7), the bias component is the more significant, apparently indicating that in these species the stimulus is correctly interpreted as a continuous rotation. Indeed, the unidirectional response in these species is almost entirely effective in compensating for the rate of rotation at even modest angles of tilt. In man, however, it seems that the same stimulus is interpreted more in terms of the translational acceleration components, appropriate to counterrotation at the end of a rotating



**FIG. 5.** The magnitude ( $\dot{\theta}_R$ ) and phase ( $\psi_R$ ) of the resultant eye velocity derived from the vector summation of the lateral and vertical components shown in Fig. 4. Phase is defined with respect to the lateromedial axis of the head.

arm, so that the sinusoidal components of lateral eye movement form the most significant part of the response.

As the subject is rotated about the tilted axis, the gravitational vector rotates in the opposite direction. This results in maximum stimulation of the utricular otolith in the lateromedial direction when the subject is left or right side down with a sinusoidal modulation between these maxima as the vector rotates (14). It has already been established that a purely linear acceleration in the lateromedial (Y) body axis can induce reflex lateral eye movements (3,4), and several previous experiments have established that a similar sinusoidally modulated lateral eye movement may be evoked when the linear



acceleration vector rotates around the longitudinal (Z) body axis (9–11,14). The values of peak eye velocity and phase obtained in the present experiment are very similar to those obtained in other tilted-axis rotation experiments but somewhat different from those obtained when the subject is seated in an upright posture (15). The usefulness of such reflex lateral angular movements of the eyes in response to linear acceleration is open to question, since the effectiveness of compensation is dependent on the distance of a viewed object from the observer. Whether there is some interaction with accommodation and convergence mechanisms is a possibility yet to be investigated.

Whereas acceleration in the lateromedial (Y) axis can evoke lateral eye movements, linear acceleration in the anteroposterior (X) direction does not appear to induce vertical eye movements in the same manner (3). As might be expected, acceleration in the X axis can lead to changes in convergence of the eyes (5) but is unlikely to generate significant vertical movements, since this function can be quite adequately performed by the vertical canals. There is some evidence to suggest that the otoliths contribute to the pitch VOR in the cat at very low frequencies (16), but recent experiments on human subjects show no significant difference between responses when the subject is either vertical or lying in a side-down position (Barnes and Donnelly, unpublished observations). It is thus unlikely that the vertical eye movements observed in this experiment arise directly from the linear acceleration component in the X axis. Indeed, if they did, they would be ineffectual as a compensatory mechanism. The effective pitch stimulus is a sinusoidal oscillation with a period of 6 sec and an amplitude of  $\pm 30^\circ$ , giving a peak angular velocity of  $\pm 31.4^\circ/\text{sec}$ . Thus the mean amplitude of vertical eye velocity observed in this experiment ( $\pm 2.03^\circ/\text{sec}$ ) represents an extremely low gain (0.06) response.

It is conceivable that the vertical eye movement does not itself represent a primary utriculoocular response but is the result of an interactive process with the lateral eye movement component. This type of rather nonspecific interaction has been shown previously between canal and otolith stimuli (12), and an example of this effect is the shortening of the time constant of postrotational nystagmus decay observed in this and previous experiments. More significantly, the responses shown in Fig. 5 indicate that the purpose of the vertical eye movement may be to direct an already existing lateral nystagmus more toward true earth horizontal. The vertical component is thus dependent on the prior existence of lateral nystagmus, and it is probable that the vestibular stimulus responsible for this modification arises from the steady-state deflection of the utricle, which also gives the sensation of head tilt in this condition. Subsequent experiments using electrooculography have established that this vectoring of the lateral eye movement can be observed when a rapid voluntary roll displacement is made following an abrupt stop from rotation about a vertical axis. This finding implies that the vertical component is not dependent on prior otolithic stimulation, although, again, the considerable variability in response among the six subjects tested makes it difficult to draw a firm conclusion.

If the function of the vertical component is to vector the eye movement, is it possible that the vertical eye movements observed during tilted-axis rota-

tion are derived from lateral eye movement components in the same manner? Unfortunately, this hypothesis does not appear to hold true. As an example, consider the responses shown in Fig. 2 during rotation to the right. Peak lateral eye velocity to the left occurs when the head is approximately left side down, so that to make the movement more horizontal would require an upward movement of the eye. But at this point, the vertical eye velocity has peak velocity downward; in other words, the response is approximately  $180^\circ$  out of phase with that which would be predicted. It is conceivable that such an effect could arise from phase changes attributable to the dynamic characteristics of the central processing of canal-otolith interactions, but such an interpretation must await the results of further experiments.

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