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## **Vertical Canal Stimulation Abolishes Horizontal Velocity Storage: Effects on Optokinetic Nystagmus and Eye Movements Evoked by a Rotating Linear Acceleration**

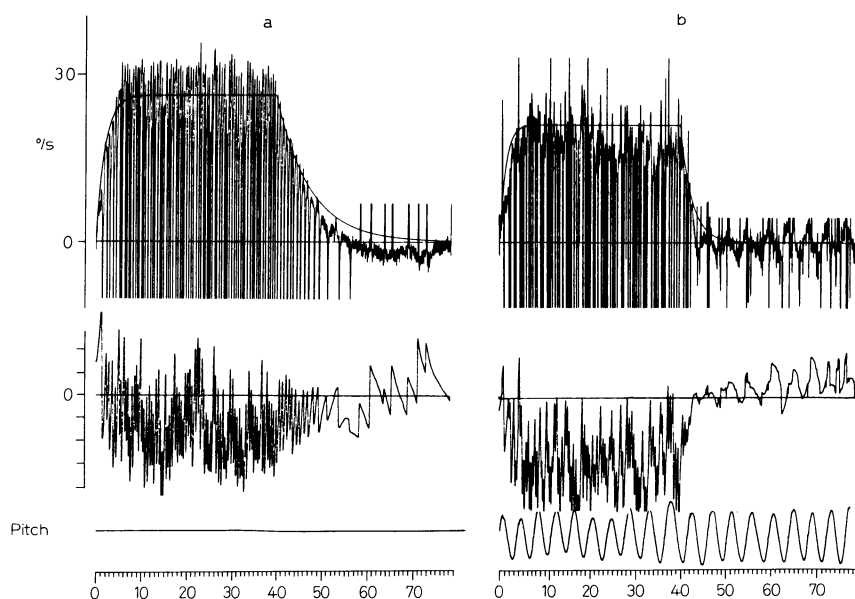
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For clear vision a stable retinal image is required. During head movements the eyes make compensatory rotations which tend to cancel movements of the retinal image. Head movements are detected by several mechanisms that contribute to this response. The mechanisms of the canal- and visual-evoked (optokinetic nystagmus, OKN) responses share a stored neural representation of the velocity of the head movement which acts like a flywheel and usefully prolongs the response beyond the duration of the stimulus [1–3].

Whenever the axis of a rotatory head movement is not perfectly vertical, the pattern of the stimulation of the otoliths (which detect linear accelerations: here gravity) conveys information about the continually changing direction of tilt during the rotation [4, 5]. A constant-velocity off-vertical-axis rotation (OVAR) is accompanied by a continuous compensatory nystagmus [4, 5] (fig. 2a) in the cat. If the animal is rotated until the canal-evoked response has decayed and then the axis tilted, compensatory eye movements build up with a time constant of about 5 s [5, 6]. This suggests the charging of a velocity store which may be held in common.

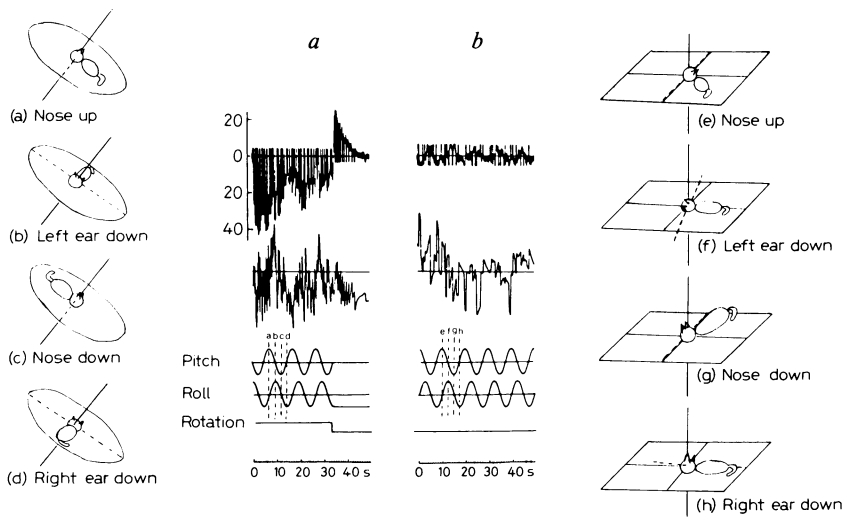
Stimulating the vertical canals by sudden tilting during optokinetic after-nystagmus (OKAN) suddenly discharges the visually charged velocity store [4]. OKN and OKAN (which depend on an intact velocity store [1, 2, 7]) were therefore measured during continuous sinusoidal activation of the vertical canals. Figure 1 contrasts OKN evoked in a stationary animal with that



*Fig. 1.* OKN in response to horizontal, full-field, visual movement produced by a planetarium projector projecting spots onto a 57-cm radius spherical screen centred on the cat's head. Eye movements were recorded from 3 cats with implanted scleral search coils. The animals were held at the centre of the screen by head restraining tubes (Kopf). The stimulus was moving at 30 °/s. The traces represent, from the top, horizontal eye velocity, horizontal eye position and pitch. The lights went out after 40 s. *a* Stimulation around a stationary vertical axis. Note the gradual decline of OKAN (time constant 7.4 s). The line through the data was derived from Robinson's model of velocity storage ( $K$ , the efficiency of the store = 0.65 [7], open-loop gain = 2.3 [8]). *b* The response to exactly the same optokinetic stimulus as in *a*, but in this case with simultaneous pitch of  $\pm 20^\circ$  at 0.23 Hz. The line plotted through the data has again been derived from Robinson's [7] model ( $K = 0$ ).

obtained with exactly the same visual stimulus during simultaneous sinusoidal pitch. The data suggest that pitching deactivates the horizontal velocity store. OKN and OKAN were measured in 3 cats during pitching movements ( $\pm 10$  and  $\pm 45^\circ$ , 0.05–0.3 Hz). The gain (eye velocity/stimulus velocity) of OKN was attenuated to  $0.7 \pm 0.1$  from its control values ( $0.94 \pm 0.06$ ) and the time constant of OKAN was reduced from  $7.4 \pm 2.8$  to  $1.5 \pm 0.5$  s.

In order to produce a rotating gravity vector with simultaneous stimulation of the vertical canals (and thus a deactivated velocity store), I subjected



**Fig. 2.** Eye movements evoked by rotating linear accelerations in complete darkness. The traces represent, from the top: horizontal eye velocity and horizontal eye position, pitch (with respect to gravity: up represents up), roll (with respect to gravity: up represents left ear down), horizontal angular velocity (up represents leftward movement). *a* OVAR at  $36^\circ/\text{s}$  to the left around an axis tilted at  $20^\circ$ . The initial canal response declines to a continuous, sinusoidally modulated nystagmus with an average velocity of  $13^\circ/\text{s}$ . *b* The response to sequential tilting. The animal was rocked sinusoidally in the pitch and roll planes with a  $90^\circ$  phase difference as shown in the stimulus traces. This thus produced exactly the same pattern of stimulation of the otoliths as in *a*. It is equivalent to leftward rotation.

3 cats to 'sequential tilting' (fig. 2b). Although the changing tilt is the same in both OVAR and sequential tilting, the vertical canals are only stimulated during sequential tilt. In response to sequential tilting there is no horizontal nystagmus at all (fig. 2b). Sequential tilting was carried out in 3 cats ( $\pm 10$ – $20^\circ$ ,  $0.05$ – $0.2$  Hz: equivalent to OVAR of  $18$ – $72^\circ/\text{s}$ ). In no case was any horizontal nystagmus evoked.

These data argue strongly for the involvement of a central velocity storage mechanism in the generation of the response to OVAR. Since OKN and OKAN are also affected (fig. 1) it further suggests that the velocity store that is involved in OVAR is also used by OKN (known to be shared by VOR) [1, 2]. Since the response to a rotating gravity vector is abolished completely, this suggests that there is no direct pathway in the system that processes OVAR.

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