Brain Research, 437 (1987) 393–396
Elsevier

BRE 22660

The eye movements evoked by a rotating linear acceleration vector in the cat depend on a central velocity storage mechanism

Laurence R. Harris

Department of Physiology, University College, Cardiff (U.K.)

(Accepted 15 September 1987)

Key words: Otolith; Semicircular canal; Off-vertical-axis rotation; Vestibulo-ocular reflex; Optokinetic nystagmus; Cat; Velocity store

The otoliths are stimulated in a particular pattern by any head movement that is not about an earth-vertical axis and evoke compensatory eye movements in the cat. Such eye movements are not produced if the otolith stimulation is accompanied by vertical canal stimulation. Vertical canal stimulation inactivates the velocity store (a central neural representation of head velocity) as seen by the attenuation of optokinetic after-nystagmus. These observations provide further evidence for the involvement of the central velocity store in the generation of otolith-evoked nystagmus.

Whenever the axis of a rotatory head movement is not perfectly vertical, the pattern of the stimulation of the otoliths (which detect linear acceleration: here gravity) conveys information about the continually changing direction of tilt during the rotation[1,6,12]. Off-vertical-axis rotation (OVAR) produces a rotating gravity vector since, as the animal rotates, it continuously progresses through a sequence of orientations: nose-up, left-ear-down, nose-down, right-ear-down and back to nose-up. This is illustrated graphically in the stimulus traces of Fig. 2a. Also, constant-velocity OVAR is accompanied by a continuous compensatory nystagmus (see refs. 6, 12 and Fig. 2a) which helps to reduce retinal image movement during head movements.

The mechanism by which the otolith signal evokes a compensatory nystagmus during OVAR is unknown but, as with the canal-evoked vestibulo-ocular reflex (VOR) and visual-evoked optokinetic nystagmus (OKN), a stored neural representation of the velocity of the velocity of head movement which I shall refer to as the 'velocity store' is involved (see ref. 11 for a review). Evidence for this comes from several observations[1,6,7,10,12,14,16]. If a cat or monkey is rotated about a vertical axis until the canal-evoked response has decayed and then the axis is tilted, compensatory eye movements are evoked with a time constant of about 5 s (both on tilting and on returning the axis to vertical[3,6]). This suggests the charging and discharging of a velocity store. VOR and OKN appear to have access to a common velocity store but it is not clear whether the store involved in the response to OVAR is the same one. Lateral canal nerve section abolishes the horizontal velocity store, as shown by changes in OKN and optokinetic after-nystagmus (OKAN) performance, and also abolishes the response to OVAR[2]. However, in order to realize the full response to OVAR, it is necessary that the signals from the canals and otoliths are compatible[5]. Clearly, after nerve sectioning this cannot be the case so the interpretation of the consequences of nerve section is not straightforward.

In order to test the involvement of a common store without lesions that might effect other contributors to the response, I developed a simple method of deactivating the velocity storage mechanism. Stimulating the vertical canals by sudden tilting during OKAN or post-rotatory nystagmus (PRN) discharges the

Correspondence: L.R. Harris, Department of Physiology, University College, Cardiff CF1 1XL, U.K.

store[12]. To test whether stimulation of the vertical canals alone deactivates the store, OKN and OKAN (which depend on an intact velocity store[4,13,15]) were evoked during continuous sinusoidal activation of the vertical canals. The experiments were performed on cats (which show all the eye movements described above) whose response to OVAR is qualitatively the same as of man and monkey[1,6,12]. The visual stimulus was produced by a planetarium projector projecting spots onto a 57-cm-radius spherical screen centered on the cat's head. Eye movements were recorded from 3 cats with implanted scleral search coils[8]. The animals were held at the centre of the screen by head restraining tubes (Kopf).

Fig. 1 contrasts OKN evoked by moving a full-field visual stimulus around a stationary animal (Fig. 1a) with that obtained with exactly the same visual stimulus during simultaneous sinusoidal stimulation of the vertical canals (Fig. 1b). This was achieved by pitching the entire apparatus; notice that the screen, projector and the cat itself all moved together so there were no visual cues as to the pitching. Also shown on Fig. 1 are theoretical curves plotted over the data for the theoretical performance of OKN and OKAN

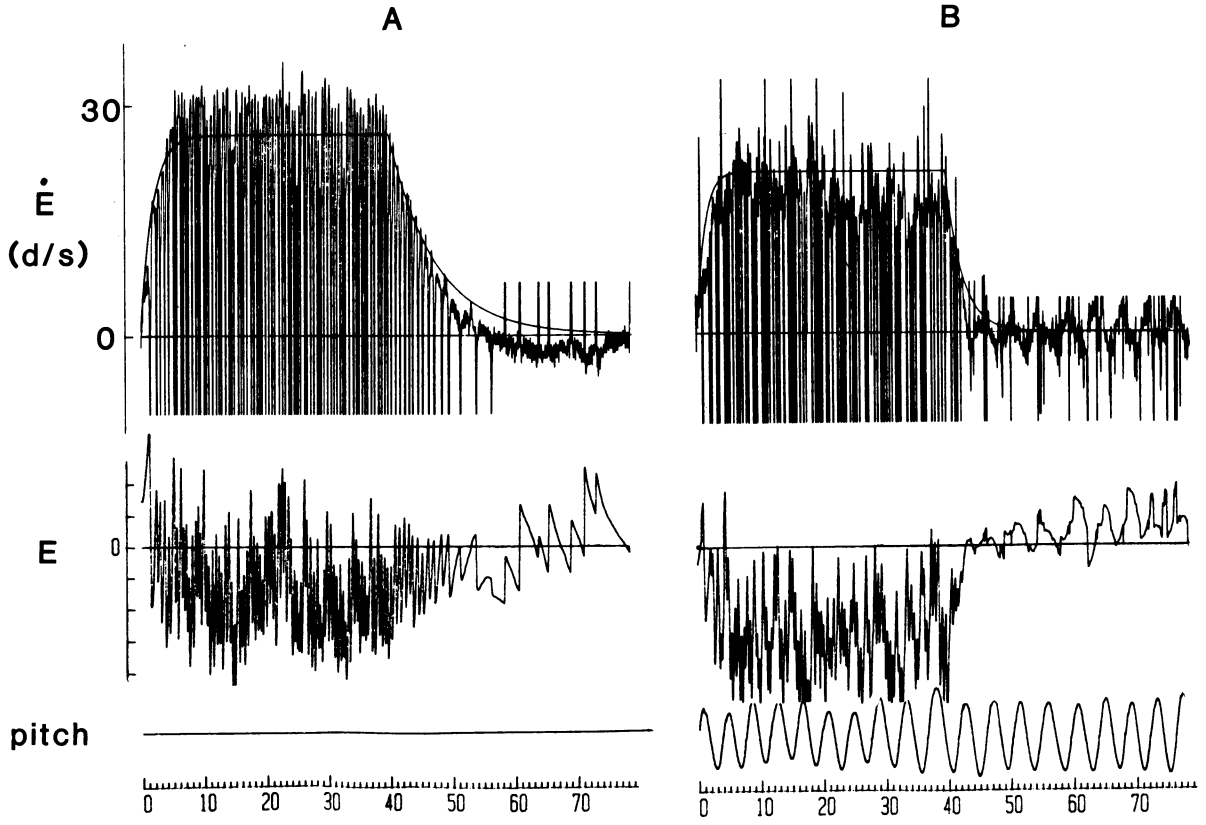

Fig. 1. Optokinetic nystagmus in response to horizontal full-field visual movement. The stimulus was moving at 30°/s. The traces represent, from the top, horizontal eye velocity (the fast phases have been artificially attentuated for clarity), horizontal eye position and pitch. The lights went out after 40 s. A: stimulation around a stationary vertical axis. The gain (eye velocity/stimulus velocity) is close to unity during the stimulation (see vertical axis). Notice the gradual decline of OKAN (time constant 7.4 s) when the animal is plunged into complete darkness. The line through the data was obtained by fitting a function to describe OKN and OKAN derived using Robinson's model of velocity storage[15]. K (the efficiency of the store) has been set equal to 0.65 (ref. 15), and the open-loop gain to 2.3 (ref. 9). B: the response to exactly the same optokinetic stimulus as in A but in this case with simultaneous pitch of $\pm 20°$ at 0.23 Hz. Notice that the pitch moved the entire apparatus so that the visual stimulus was the same in both A and B. The gain of the response during stimulation has decreased to 0.7 and the time constant of the decay of OKAN has been dramatically shortened to about 1.5 s. The line plotted through the data has again been derived from Robinson's model[15] of velocity storage, as for A, but with the value of K (the efficiency of the store) set to zero. The curve well describes the OKN and OKAN and is strong evidence for suggesting that stimulation of the vertical canals has blocked horizontal velocity storage.

with (Fig. 1a) and without (Fig. 1b) a velocity store[15]. The curves clearly fit the data well and suggest that stimulation of the vertical canals indeed deactivates the horizontal velocity store. OKN and OKAN were measured in 3 cats during pitching movements (around an axis through the cats' ears) of amplitudes between ±10° and ±45° and over a frequency range of 0.05–0.3 Hz. There were no differences over this range (values are means ± S.D.). OKN was attenuated to 0.7 ± 0.1 of its control values (0.94 ± 0.06) and the time constant was reduced from 7.4 ± 2.8 s to 1.5 (±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 the cats to sequential tilting. Sequential tilting was achieved by sinusoidally rocking in the pitch and roll (around an axis through the cats' nose) planes of a two-axis vestibular stimulation table simultaneously with the sine waves 90° out of phase. Thus: during the pitch-down position the animal is level in the roll plane; as the animal comes level in the pitch plane, it rolls towards right-ear-down. As it returns to level in the roll plane, it pitches towards nose-up and so forth (Fig. 2b). Although the changing tilt is the same in OVAR and sequential tilting, the vertical canals are only stimulated during sequential tilt.

A typical response to OVAR is shown in Fig. 2a. The direction of the continuous nystagmus seen during rotation is in the appropriate direction to com-

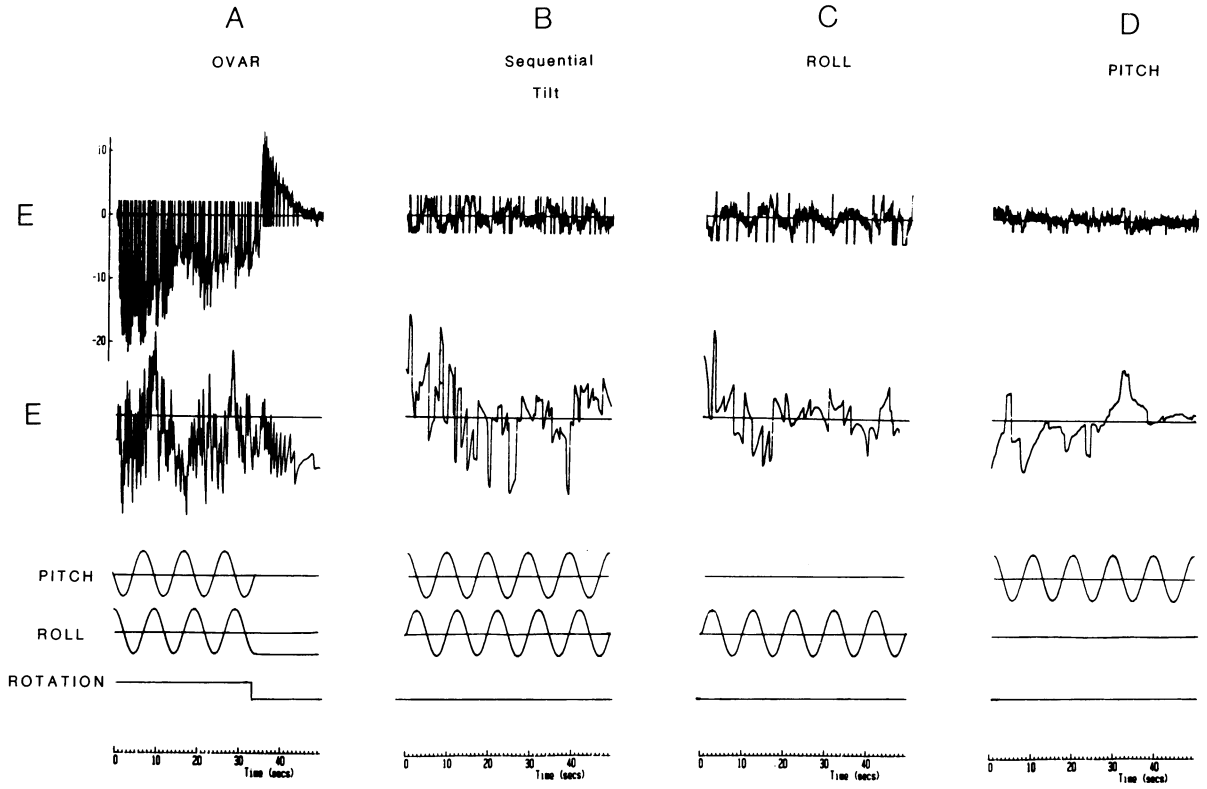

Fig. 2. Eye movements evoked by various patterns of vestibular stimulation in complete darkness. The traces represent, from the top: horizontal eye velocity (the fast phases have been artificially attenuated for clarity: up represents leftward eye movement), and horizontal eye position, pitch (with respect to gravity: up represents up), roll (with respect to gravity: up represents left ear down), horizontal angular velocity (up represents leftward movement). Below is the time scale. A: OVAR at 36°/s to the left around an axis tilted at 20°. The initial canal response declines to a continuous, sinusoidally-modulated nystagmus with an average velocity of 13°/s. This represents a compensatory gain of 0.36. After 34 s of constant velocity rotation, the table was suddenly stopped. Postrotatory nystagmus is attenuated. B: the response to sequential tilting. The animal was rocked sinusoidally in each plane with a 90° 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. The frequency is 0.1 Hz so that, as for the OVAR, a cycle is completed every 10 s (equivalent to an OVAR of 36°/s. The amplitude is ±20° in both planes so that the pitch and roll components are exactly the same as in the OVAR condition. C: the effect of roll alone. Frequency is again 0.1 Hz; amplitude ±20° to make it comparable to A and B above. D: the effect of pitch alone. Frequency is again 0.1 Hz; amplitude ±20° to make it comparable to A and B above.

pensate for the rotation and here, at a tilt of 20°, represents a compensation with an efficacy of about 0.36 (eye velocity/rotation velocity) (the gain depends on the size of the tilt reaching a maximum of 0.73 at a tilt of about 30°: ref. 6).

The response to OVAR contrasts startlingly with the response to sequential tilting (Fig. 2b) arranged to have the same rotating gravity vector as Fig. 2a. In response to sequential tilting there is no horizontal nystagmus at all. Sequential tilting was carried out in 3 cats with amplitudes of 10°–20° (corresponding to these values of tilts in the OVAR condition). The frequency was also varied over the range 0.05–0.2 Hz (corresponding to OVAR rotation velocities of 18–72°/s). In no case was any horizontal nystagmus evoked by sequential tilting.

These data argue strongly for the involvement of the 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 which is involved in OVAR is also used by OKN which is known to be shared by VOR. This observation extends the results obtained by deactivating the velocity store by lateral canal nerve section[2]. An inappropriate horizontal canal signal effects the response to OVAR directly[5] but sequential tilting now shows that it is indeed deactivating the velocity store that prevents the OVAR response after nerve section.

OKN performance, whilst reduced by being forced to rely on direct pathways by the lack of a velocity storage mechanism, is still quite effective: there is only a small reduction of gain (eye velocity/stimulus velocity) from 0.94 to 0.7 (Fig. 1). However, as a result of removing the horizontal velocity store, the response to a rotating gravity vector is abolished completely (Fig. 2b). This was also the effect of lateral canal nerve section[2]. This suggests that there is no direct pathway in the system that processes OVAR.

Signals from the semicircular canals are crucial in determining the pattern of eye movements in response to otolithic stimulation. This is sensible ecologically, since a rotating gravity vector can be produced in a number of ways each of which requires different compensatory eye movements. The cat uses the overall pattern of vestibular activity as a context in which to interpret the otolithic signal which *could* in some circumstances indicate movement in the horizontal plane. Man and cat are qualitatively similar in their response to OVAR. The mechanism of otolith processing is not yet understood but procedures such as those described here could form the basis of a clinical tool for assessing otolithic and canal status.

1 Benson, A.J. and Bodin, M.A., Interaction of linear and angular accelerations on vestibular receptors in man, *Aerospace Medicine,* 37 (1966) 144–154.

2 Cohen, B., Suzuki, J. and Raphan, T., Role of the otolith organs in generation of horizontal nystagmus; effect of selective labyrinthine lesions, *Brain Research,* 276 (1983) 159–164.

3 Cohen, B., Suzuki, J., Raphan, T., Matsuo, V. and De Jong, V., Selective labyrinthine lesions and nystagmus induced by rotation about an off-vertical axis. In G. Lennerstrand, E. Keller and D.S. Zee (Eds.), *Functional Basis of Ocular Motility Disorders,* Pergamon, Oxford, 1982, pp. 337–346.

4 Demer, J.L. and Robinson, D.A., Different time constants for optokinetic and vestibular nystagmus with a single velocity storage element, *Brain Research,* 276 (1983) 173–177.

5 Harris, L.R., The effect of opposing canal and otolith signals during off-vertical-axis rotation in the cat, *J. Physiol. (Lond.),* 371 (1986) 30P.

6 Harris, L.R., Vestibular and optokinetic eye movements evoked in the cat by rotation about a tilted axis, *Exp. Brain Res.,* 66 (1987) 522–532.

7 Harris, L.R. and Barnes, G., The orientation of vestibular nystagmus is modified by head tilt. In M.D. Graham and J.L. Keminck (Eds.), *The Vestibular System,* Raven, New York, 1987, pp. 571–581.

8 Judge, S.J., Richmond, B.J. and Chu, F.C., Implantation of magnetic search coils for measurement of eye position: an improved method, *Vision Res.,* 20 (1980) 535–538.

9 Maioli, C. and Precht, W., The horizontal optokinetic nystagmus in the cat, *Exp. Brain Res.,* 55 (1984) 494–506.

10 Raphan, T. and Cohen, B., Gravitational effects on visual vestibulo-oculomotor coordinate transforms generating compensatory eye movements, *Soc. Neurosci. Abstr.,* 13 (1984) 316.

11 Raphan, T. and Cohen, B., Velocity storage and the ocular response to multi-dimensional vestibular stimuli. In A. Berthoz and G. Melvill Jones (Eds.), *Adaptive Mechanisms in Gaze Control,* Elsevier, Amsterdam, 1985, pp. 123–144.

12 Raphan, T., Cohen, B. and Henn, V., Effects of gravity on rotatory nystagmus in monkeys, *Ann. N.Y. Acad. Sci.,* 374 (1981) 44–55.

13 Raphan, T., Cohen, B. and Matsuo, V., A velocity storage mechanism responsible for OKN, OKAN and vestibular nystagmus. In R. Baker and A. Berthoz (Eds.), *Control of Gaze by Brain Stem Neurones,* Elsevier, Amsterdam, 1977, pp. 37–47.

14 Raphan, T., Matsuo, V. and Cohen, B., Velocity storage in the vestibulo-ocular reflex arc (VOR), *Exp. Brain Res.,* 35 (1979) 229–248.

15 Robinson, D.A., Vestibular and optokinetic symbiosis: an example of explaining by modelling. In R. Baker and A. Berthoz (Eds.), *Control of Gaze by Brain Stem Neurones,* Elsevier, Amsterdam, 1977, pp. 49–58.

16 Ter Braak, J.W., Untersuchen über optokinetischen Nystagmus, *Arch. Neerl. Physiol.,* 21 (1936) 309–376.