Reprinted from Nature, Vol. 286, No. 5768, pp. 64-65, July 3 1980

Eye movement in strabismic cats

Max Cynader* & Laurence Harris

Department of Psychology, Dalhousie University, Halifax, Nova Scotia, Canada B3H 4J1

Strabismus is a common clinical condition in which the visual axes of the eyes do not intersect on the object being viewed[1]. As such, the ability to achieve single binocular vision by fusing the images of a single object in the two eyes is lost. In most cases of strabismus, one of the two eyes is clearly deviated and the other eye is used for fixation, although in some cases each eye is used alternately for fixation[2,3]. While much attention has been devoted to the motor capabilities of the deviating eye in strabismus[1-3], little attention has been given to the visuomotor competence of the other eye. We report here that, if one eye of a kitten is made to deviate by surgery, the visuomotor capacities of the other, 'normal', eye are affected. A reduction in the ability to follow the movement of a large striped drum is observed with binocular viewing, even when stimuli are viewed monocularly with the normal eye. This means that anomalous visual input from the deviated eye during stimulation is not the cause of the reduced oculomotor capacities.

Divergent strabismus was induced in three 10-day-old kittens by severing the medial rectus muscle of the right eye. The left eye was not touched. The kittens were then reared normally in a well-lit laboratory environment until they were 8–12 months old. At this time the normal undeviated eye of the cat was prepared for eye movement recording by implanting a magnetic search coil according to the technique of Robinson[4]. In addition, head restraining tubes were attached to the skull by means of dental acrylic[5]. Surgery was performed under Alfathesin anaesthesia and the animals were allowed at least 2 weeks to recover before recording began. The eye movements of the three strabismic cats were compared with those of three normal cats studied with the same techniques.

We primarily studied eye movements evoked in response to visual and vestibular stimuli in the horizontal plane. When a large striped drum is rotated about a cat, a characteristic pattern of eye movement called optokinetic nystagmus (OKN)[6] is evoked with a time course resembling a sawtooth. The slow phase of this response matches the velocity of the drum in normal cats and thus compensates for the motion of the drum and permits clear vision. Figure 1 compares the velocity of the slow corrective phase of optokinetic nystagmus (measured in the nondeviating eye) of strabismic cats with that of normal cats as a large striped drum is rotated about the animal at various velocities. The results show that the normal cat is able to match the velocity of his eyes to that of the drum rather well over most of the velocity range tested.

By contrast the velocity match of the apparently normal eye of the strabismic cat with that of the drum is much less accurate. For all drum velocities above 4 per s, the eye rotated more slowly than did the drum. This lessened response to visual stimulation is unlikely to be due to any generalized deficit in oculomotor capacity, due, for example, to mechanical impediments which might have resulted from implantation of the eye coil or from the early surgical procedures on the other eye. The eye movement responses to vestibular stimulation as assessed by rotating the cat sinusoidally in the dark were the same in normal and strabismic cats. In both normal and strabismic cats, the vestibulo-ocular reflex was able to compensate for the head and body movement induced by rotating the cat[5,7]. Thus, the deficit in oculomotor capabilities

*To whom correspondence should be addressed.

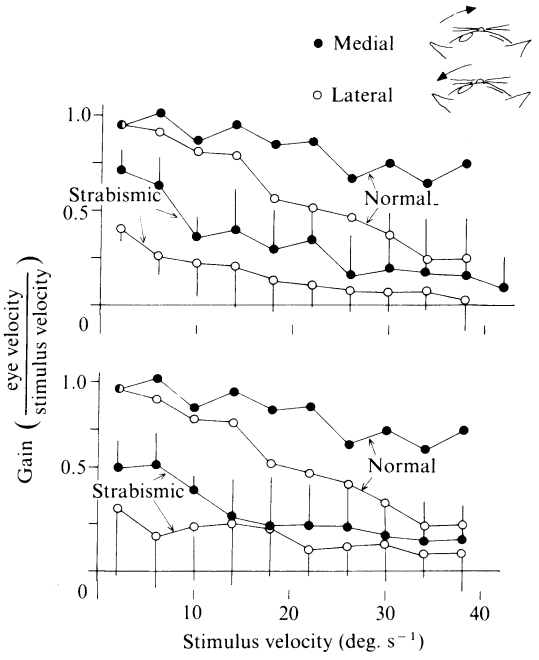

Fig. 2 *a*, The gain of the slow phase of optokinetic response in normal and strabismic cats when stimuli are viewed with the nondeviated eye only. The gain of the reflex is lower for laterally-directed high velocity drum movement than medially-directed movement in both the normal and strabismic cats. The gain in both directions is considerably lower for the strabismic cats than it is for normal cats even though the strabismic animals viewed the stimulus through the nondeviated eye. In normal cats, the gain of the reflex is the same regardless of whether the monitored eye or the other eye alone views the stimulus, as would be expected from Hering's Law[16]. *b*, Responses to drum motion when the strabismic animals view through the deviating eye alone are compared with those of normal cats viewing monocularly. The deficits observed in the visuomotor capacities of the strabismic cats are similar regardless of which eye views the moving stimulus. The standard deviations (s.d.) have been omitted from the curves representing responses of the normal cats for clarity. A typical value for the s.ds in this case is 0.15. Drum parameters are as in Fig. 1.

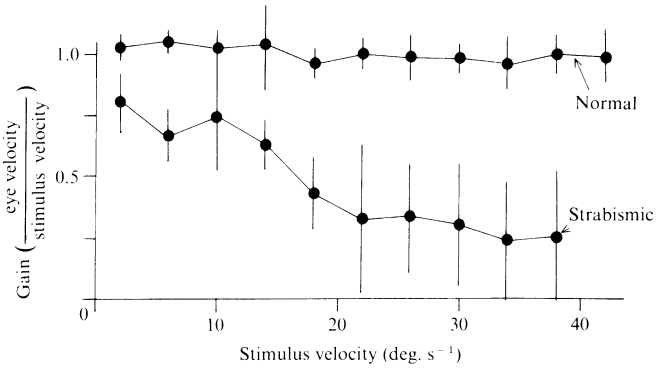

Fig. 1 The gain of optokinetic nystagmus as a function of drum speed for the normal and strabismic cats. The gain of the response is the velocity of the slow phase[6] of the optokinetic response divided by the velocity of the drum. A gain of 1 indicates perfect following of the drum by the eyes. The gain of the normal cats is close to 1 over most of the velocity range tested while that of the strabismic cats is markedly lower. The drum consisted of a large cardboard cylinder 90 cm in diameter on which high contrast black and white vertical stripes (period 20°) were painted. Background luminance was 0.1 cd m^{-2}.

seems to be specifically related to the response to visual stimuli.

The results illustrated in Fig. 1 were obtained with both eyes open and did not permit assessment of the contribution of vision with each eye to the reduced oculomotor response of the nondeviating eye. Accordingly we measured optokinetic responses of the 'normal eye' to visual stimuli presented through either eye. The interpretation of the monocular stimulation findings (Fig. 2) is rendered more difficult by the finding that monocularly-viewed stimuli moving medially (that is, rightward viewed through the left eye) were more effective at eliciting OKN than lateral-directed stimuli even in normal cats[8]. This difference in the effectiveness of medial and lateral-directed stimuli was also observed with monocular stimulation of the nondeviating eye in the strabismic cats. It is clear, however, that the efficiency of OKN was decreased markedly for both medial and lateral stimulus motion, relative to that of the normal animals. This defect in monocularly-evoked OKN rules out anomalous visual input through the deviating eye during the stimulation period as a possible cause of the reduced OKN observed with binocular stimulation. Visuomotor performance mediated by stimuli presented through the deviating eye was also severely impaired. Figure 2*b* shows optokinetic responses of the nondeviating eye to visual stimuli viewed monocularly through the deviating eye. These data show that reduced OKN followed stimulation of the deviating eye when stripes moved in either direction. The deficits of OKN were similar with stimulation of either the deviating eye or the nondeviating eye.

The present results show that the visually-evoked eye movements mediated through an apparently normal eye can be altered by surgical exodeviation of the other eye. The eye movement deficit seems to be specific to visual stimulation as vestibular stimulation evoked normal oculomotor responses. The mechanism underlying this reduced visuomotor competence remain unknown but several anomalies identified in various parts if the visual system in strabismic cats[9–11] help towards its understanding. We have recently recorded visual responses in the nucleus of the optic tract (NOT) of strabismic and normal cats[11]. Our findings in this structure, which has an essential role in optokinetic nystagmus[12,13], indicate marked changes in binocular connectivity among NOT neurones following surgically-induced strabismus. These neural changes may provide a basis for the visuomotor deficits found in the strabismic cats. It would be interesting to see if similar defects exist in the normal eye of human strabismics, particularly in view of the recent findings suggesting defects on OKN in the normal eye of amblyopic humans[14,15].

This research was supported by USPHS grant no. EY02248 and grants nos MT-5201 and A9939 from the MRC and NRC of Canada.

Received 12 March; accepted 3 April 1980.

1. Alpern, M. & Davson, H. eds *The Eye* Vol. 3, 1–127 (Academic, London, 1969).
2. Burian, H. M. & Von Noorden, G. K. *Binocular Vision and Ocular Motility* (C. V. Mosby, St Louis, 1974).
3. Duke, Elder, S. & Wybar, K. in *System of Ophthalmology* Vol. 6 (C. V. Mosby, St Louis, 1973).
4. Robinson, D. A. *IEEE*, Trans. Biomed. Electron. BME-10, 137–145 (1963).
5. Harris, L. R. & Cynader, M. *Invest. Ophthalm.* **18**, suppl. 263 (1979).
6. Ter Braak, J. W. G. *Arch neerl. Physiol.* **21**, 309–376 (1936).
7. Cohen, B. *Handbook of Sensory Physiology: Vestibular System* Vol. 6, Part 1 (ed. Kornhuber, H.) 477–540 (Springer Berlin, 1973).
8. Honrubia, V., Ward, P. H. & Scott, W. *Acta otolaryng* **64**, 388–402 (1967).
9. Hubel, D. H. & Wiesel, T. N. *J. Neurophysiol.* **28**, 1041–1059 (1965).
10. Cynader, M., Mustari, M. & Gardner, J. G. *Soc. Neurosci. Symp.* **4**, 99–120 (1979).
11. Cynader, M. & Hoffmann, K. P. (in preparation).
12. Collewijn, H. J. *J. Neurobiol.* **6**, 3–21 (1975).
13. Hoffmann, K. P. *Brain Res.* **99**, 359–366 (1975).
14. Schor, C. M. & Levi, D. *Invest. Ophthalm.* **18**, suppl. 201 (1979).
15. Crone, R. A. *Docum. Ophthalm.* **45**, 9–18 (1977).
16. Hering, E. *The Theory of Binocular Vision* (eds Bridgeman, B. & Stark, L.) 17–46 (Plenum, New York, 1977).

Printed in Great Britain by Macmillan Production Ltd., Basingstoke, Hampshire