EDITORIAL COMMENTARY

Oscillopsia: impaired vision during motion in the absence of the vestibulo-ocular reflex

The vestibuloocular reflex evolved to guarantee clear vision of the environment during perturbations of the head, especially those occurring during locomotion.¹ This point was elegantly made by the anonymous physician, JC, who had lost his “balancing mechanism” as a consequence of toxic doses of streptomycin²:

“During a walk I found too much motion in my visual picture of the surroundings to permit recognition of fine detail. I learned that I must stand still in order to read the lettering on a sign.”

However, with time, such symptoms partially abate. Thus, JC reported that he was able to return to tennis doubles after about 4 years. The nature and extent of the visual symptoms that persist after loss of the VOR are the subject of the paper by Morland *et al* (this issue, pp 828–835).

Clear vision of objects in the environment requires that their images be held fairly steadily, close to the foveal region of the retina, where photoreceptor density is highest.³ Thus head movements constitute a threat to clear vision unless eye movements can be generated promptly to compensate for them, and thereby keep the line of sight aimed at the object of regard. An important implication of JC's report is that visually mediated eye movements cannot compensate for the head movements that occur during locomotion, which contain high frequency perturbations due to transmitted vibrations from each foot fall. This is because the visual system acts at relatively long latency (>70 ms). On the other hand, the vestibuloocular reflex acts at a latency <15 ms, and so is uniquely able to generate promptly eye movements that can compensate for head perturbations.⁴

Using psychophysical techniques pioneered by the late Keith Ruddock and colleagues, Morland *et al* have asked the question: Are the responses to both “temporal vision” mediated by transient, magnocellular channels and “spatial vision” mediated by sustained, parvocellular channels

responsible for the visual symptoms reported by patients who have lost vestibular function? They found that temporal vision, and specifically the ability to detect a moving stimulus, was preserved during head oscillations, suggesting a cortical adaptation. On the other hand, spatial vision during head oscillations was impaired so that fine spatial information (such as high contrast Snellen optotypes) was obscured by increased sensitivity to lower spatial frequencies. This persistent impairment of spatial vision was similar whether the subject or the visual stimulus was oscillated, and implies a functional defect early in the visual pathway that can be correlated with the rate of image drift on the retina.

Thus although the ability to detect a moving target during self motion may be preserved after loss of vestibular function, those spatial properties of vision which allow us to read type or recognise faces do not. Fortunately, other mechanisms—such as activation of the normally vestigial cervico-ocular reflex and the ability to generate eye movements in anticipation of head movements—help improve overall function.⁴ Such mechanisms led JC to conclude: “Is there any man-made machine designed like the human apparatus—with so many alternate systems to accomplish its end?”

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1 Crane BT, Demer JL. Human gaze stabilization during natural activities: translation, rotation, magnification, and target distance effects. *J Neurophysiol* 1997;78:2129–44.

2 JC. Living without a balancing mechanism. *N Engl J Med* 1952;246:458–60.

3 Carpenter RHS. The visual origins of ocular motility. In: Cronly-Dillon JR, ed. *Vision and visual function*. Vol 8. *Eye movements*. London: MacMillan; 1991:1–10.

4 Leigh RJ, Zee DS. *The neurology of eye movements*. 3rd edition. Oxford: Oxford University Press, 1999.