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Relationships Among Oscillopsia, the Vestibulo-Ocular Reflex, and Nystagmus

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The purpose of eye movements is to guarantee a clear and stable view of our environment. This is possible only if images are held relatively steady on the retina (1,2). In addition, our best vision of objects occurs when their images are close to the fovea. During natural activities, the major threat to clear vision is the rotational perturbations of the head that occur during locomotion. When we view near objects, translations (linear displacements) of the head become important (3,4). If the vestibulo-ocular reflex (VOR) is not working appropriately, during head movements, the view of the world may become indistinct, and oscillopsia—illusory motion of the environment—may result. Another cause of indistinct vision and oscillopsia is excessive motion of images on the retina caused by spontaneous nystagmus. In this chapter, we set out to determine those visual conditions that seem to be both necessary and sufficient for clear and stable vision, with the ultimate goal of developing strategies to improve the vision of patients with vestibular disturbances and nystagmus.

CLEAR VISION DURING LOCOMOTION

Head Stability During Locomotion

Before examining the performance of the VOR during locomotion, it is important to define the characteristics of head perturbations that occur. The frequency and velocity ranges of rotational head perturbations that occur during walking and running in place (5,6) are summarized in Fig. 1. Because of transmitted vibrations during heel strike, the head is subjected to rotations, particularly in pitch, that have fundamental frequencies of up to 5 Hz, or even higher during running. Although head perturbations during locomotion are of relatively high frequency (higher frequencies than are usually tested in vestibular laboratories), the peak velocities do not usually exceed about 150 degrees/sec, which is well within the operating range of the VOR (7).

Patients with vestibular disturbances frequently complain that their symptoms are worse when they get up and walk. Bilateral

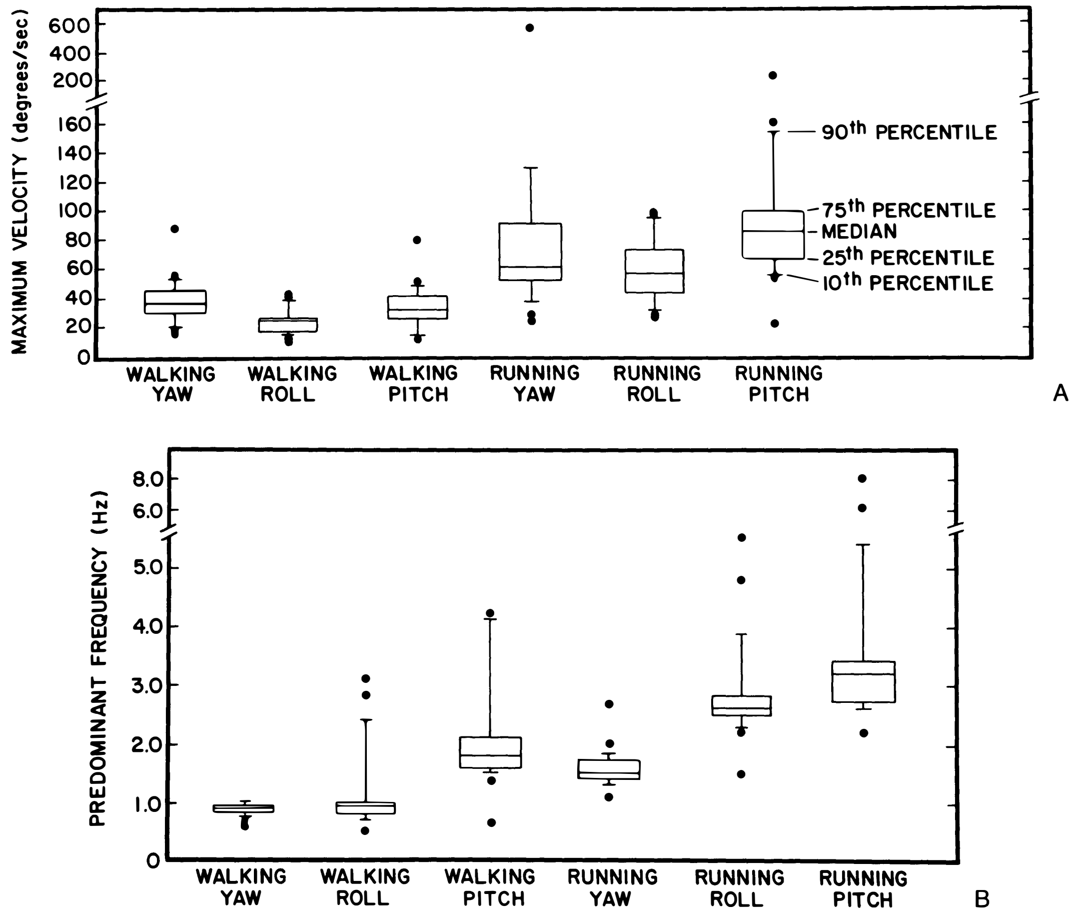


FIG. 1. Summary of the ranges of **(A)** maximum velocity and **(B)** predominant frequency of rotational head perturbations occurring during walking or running in place. Distribution of data from 20 normal subjects are displayed as Tukey box graphs, which show selected percentiles of the data. All values beyond the 10th and 90th percentiles are graphed individually as points (From ref. 6.)

loss of vestibular function causes oscillopsia during locomotion, as was described by the anonymous physician, J.C. (8). An important first question in understanding such phenomena is: Does vestibular loss lead to instability of the head? Although some investigators have suggested that this is the case, our own measurements of four patients with vestibular loss indicate that the frequency and velocity ranges of head movements occurring during locomotion are not increased. Two of these patients, who have been reported previously (9), were tested using the magnetic search coil technique. Although these measurements were precise, it is possible that the presence of the scleral search coil caused them to restrict head movements while walking in place. Because of this we used an angular rate sensor (Watson Industries, Eau Claire, Wisconsin) to measure yaw, pitch, and roll rotations while two additional subjects walked or ran in place. One was a 46-year-old woman who had idiopathic vestibular loss (10). She also had a mild essential tremor of the head, which was familial. The second patient was a 62-year-old woman who had lost vestibular function secondary to aminoglycoside antibiotics that were given for severe sepsis. Both patients lacked responses to ice-cold caloric stimuli. Neither patient showed any degree of head instability during walking or running in place. As an example, a record of pitch rotations during running in place from the second patient is shown in Fig. 2. Peak head velocity does not exceed 50 degrees/sec, and the predominant frequency is 2.9 Hz,

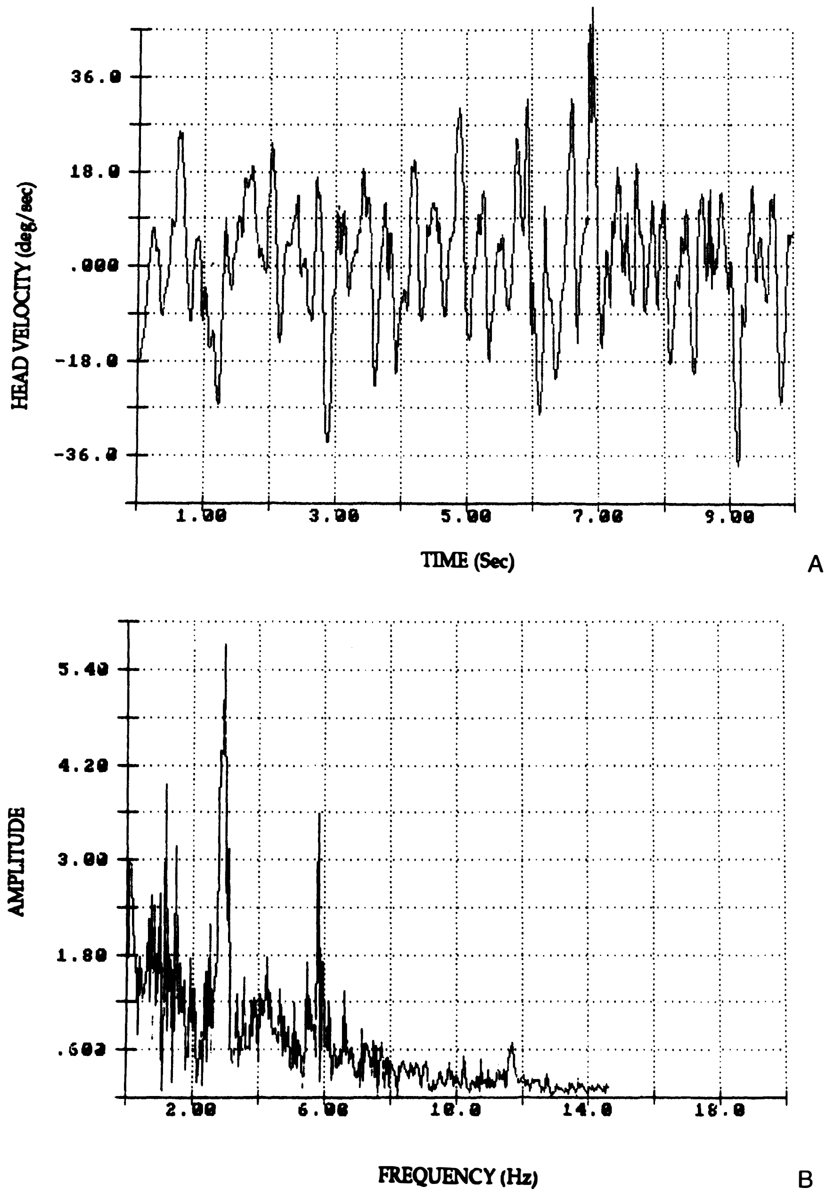


FIG. 2. Head stability in a patient with deficient vestibular function (see text). **A:** Record of pitch head velocity as she ran in place. Note that peak head velocity did not exceed 50 degrees/sec. **B:** Fourier transform based on head pitch data (2048 points). Amplitude (relative scale) indicates a predominant frequency of 2.9 Hz.

with a harmonic at 5.8 Hz. Thus, all head perturbation of the four patients with deficient labyrinthine we have studied were within the ranges of our normal subjects (Fig. 1). Such a finding is consistent with studies of the mechanical properties of the head and neck (11,12). These reports indicate that the main factor determining stability of the head for stimuli of similar frequencies to those occurring during locomotion is mechanical rather than neurogenic. If this is accepted, the next logical step is to determine the disturbance of gaze stability that occurs in individuals with vestibular disturbance during natural activities, such as locomotion.

Stability of Gaze During Locomotion

We measured gaze stability in two individuals during walking in place while they viewed a distant object and demonstrated that both gaze position and gaze velocity were increased compared with control subjects (9,13). These two patients also noted a decline in visual acuity during locomotion. A coherence analysis of these data showed that their VOR performed poorly during walking in place. On the other hand, during voluntary head rotations, eye movements that compensated for over 90% of head rotations were achieved, even at frequencies of above 1.0 Hz. An important point here is that walking produces head perturbations that have a *randomness* to them and that cannot be easily compensated for in individuals with vestibular loss. Thus, although the cervicoocular reflex and efferent mechanisms enable labyrinthine-deficient patients to generate compensatory eye movements during predictable or self-generated head rotations (14, 15), only the VOR is able to provide the prompt ocular compensation required to guarantee clear vision during locomotion. We have measured the latency of action of the VOR in response to high-acceleration head rotations. For four subjects, the median value was 10.0 msec, with a range of 6 to 15 msec (16).

CLEAR VISION IN INDIVIDUALS WITH CONGENITAL NYSTAGMUS

Further insights into the relationship between retinal image movement and visual acuity have been provided by studies of patients with nystagmus (17,18). Patients with acquired nystagmus often complain of oscillopsia, but individuals with congenital nystagmus seldom do so (19). The latter may be due, in some subjects, to use of an efference copy (internal neural signal) of the nystagmus to negate the visual effects of the waveform (19). In addition, the threshold for detecting motion may be elevated in subjects with congenital nystagmus (20). Perhaps the most important factor in providing clear and stable vision is the presence of consistent and well-developed foveation periods during which the image of an object of interest is placed on the fovea and moves at less than 4 degrees/sec for periods as long as 100 msec. Foveation periods have been identified as a component of all types of waveform encountered in congenital nystagmus (21). They occur after the quick phases (which tend to bring the image of a target to the fovea) and before the slow phase starts to accelerate (while the velocity of image drift is low). Thus, it seems that individuals with congenital nystagmus extract a clear and stable view of the world by concentrating purely on these snapshots during steady foveation.

We have studied two patients with congenital nystagmus who developed oscillopsia during adult life. One of these patients developed his symptoms after an episode of loss of consciousness (22), and the other did so in the setting of lithium therapy for affective disorder.

The first patient had intermittent oscillopsia, and we were able to correlate this with a change in the waveform during which he did not have well-developed foveation periods (Fig. 3, *left*). As the image of the object of regard passed over the retina, its speed was typically greater than 15 degrees/sec. At times when he did not experience oscillopsia (Fig. 3, *right*), he had well-developed foveation periods during which the image of the object of regard was consistently held on the foveal region (about 1 degree) at speeds of less than 4 degrees/sec for periods of over 100 msec.

The second patient complained of ellip-

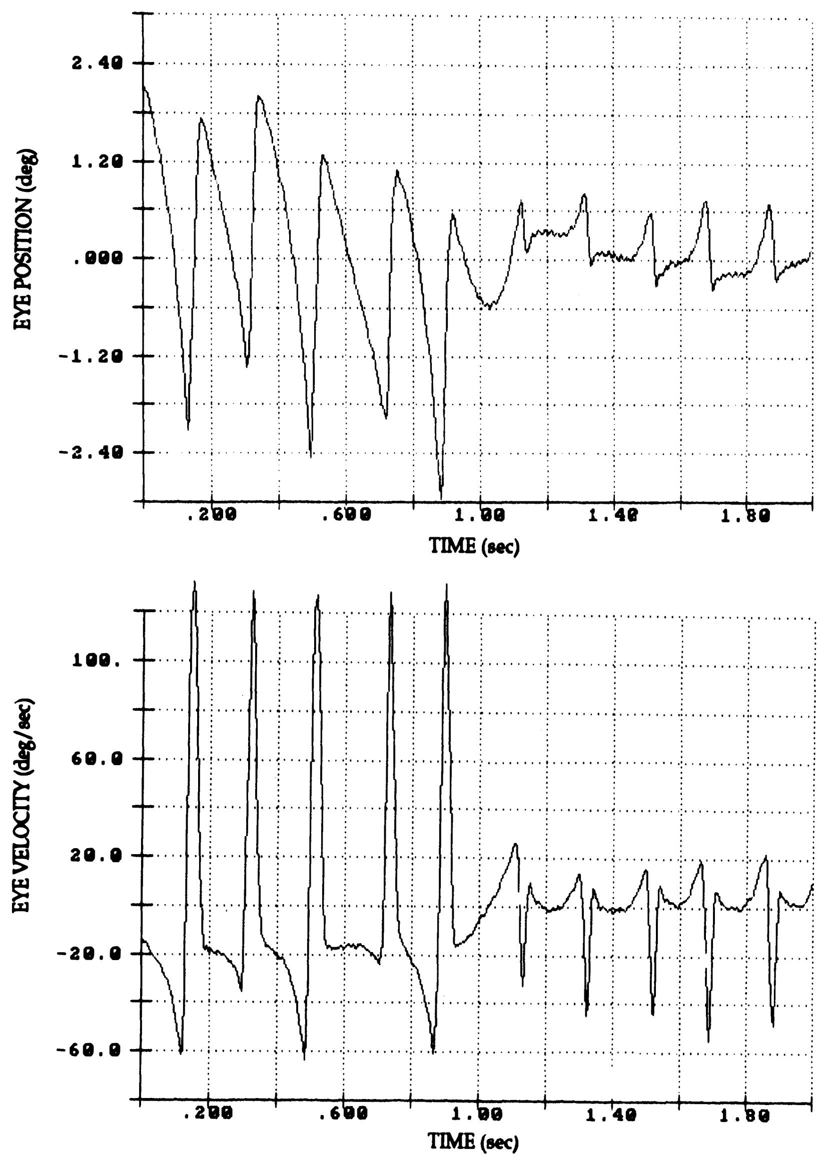
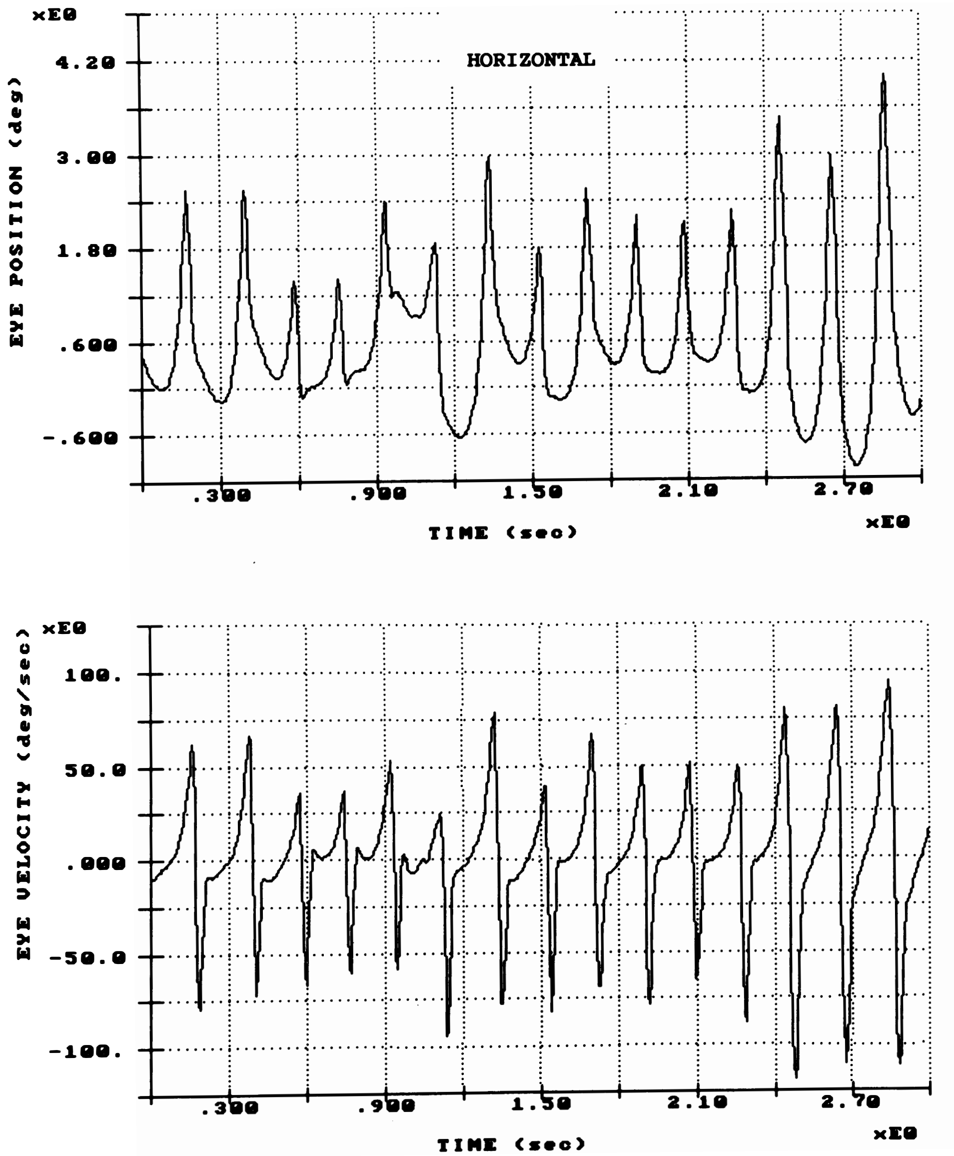


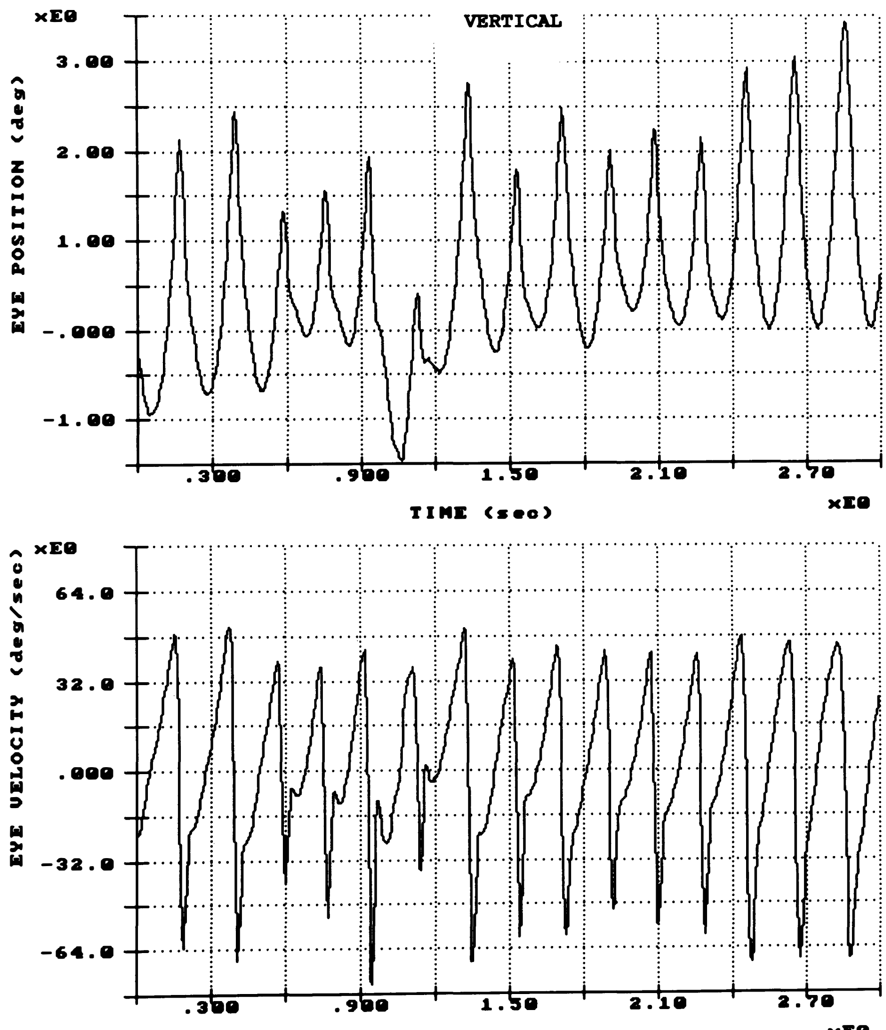
FIG. 3. Intermittent oscillopsia in congenital nystamgus (see text). In the first part of the record, the subject complained of oscillopsia at a time when there were no foveation periods. In the second part of the record, his nystagmus changed to a waveform with well-developed foveation periods, and his oscillopsia resolved. This patient has been reported elsewhere (22).



A

FIG. 4. Elliptical oscillopsia in congenital nystagmus (see text). Neither the horizontal **(A)** nor vertical **(B)** components of the subject's right eye oscillation show consistent, well-developed foveation periods. When both horizontal and vertical oscillations are considered together in a scan-path plot **(C)**, it is evident that during many cycles of nystagmus, foveation periods do not occur. The circle indicates the region of foveal isoacuity.

tical oscillopsia. She had horizontal and vertical components to her nystagmus, neither of which had consistent, well-developed foveation periods (Fig. 4A,B). Consequently, when both components of her waveform were taken into account (Fig. 4C), adequate foveation was not achieved during many cycles of nystagmus. The trajectory of the waveform could be related to the direction of the oscillopsia.


VERTICAL
xE0
3.00
2.00
1.00
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-1.00
EYE POSITION (deg)
.300
.900
1.50
2.10
2.70
TIME (sec)
xE0
64.0
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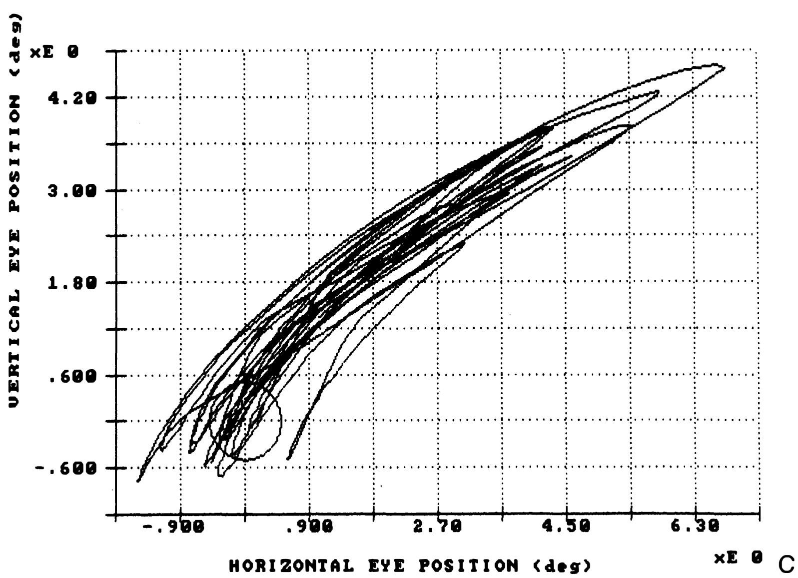
.000
-32.0
-64.0
EYE VELOCITY (deg/sec)
TIME (sec)
B


xE 0
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VERTICAL EYE POSITION (deg)
-.900
.900
2.70
4.50
6.30
HORIZONTAL EYE POSITION (deg)
xE 0
C

IMPLICATIONS OF FOVEATION PERIODS

Minimal Conditions for Clear Vision

Further study of individuals with congenital and acquired nystagmus may help us define criteria for clear vision. Provisional criteria are that the image must be within half a degree of the center of the fovea, moving with a velocity of less than 4 degrees/sec for a period exceeding about 30 msec. This last criterion—the length of the foveation period—is the factor that we know least about. If patients with vestibular loss could develop strategies for such brief periods of foveation during locomotion, it is possible that they also would gain improvement of vision. Similarly, for patients with acquired nystagmus to achieve clear vision, the nystagmus need not be abolished but only reduced to provide adequate periods of foveation.

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REFERENCES

1. Barnes GR, Smith R. The effect on visual discrimination of image movement across the stationary retina. *Aviat Space Environ Med* 1981; 52:466–472.
2. Westheimer G, McKee SP. Visual acuity in the presence of retinal-image motion. *J Opt Soc Am* 1975;65:847–850.
3. Paige GD. The influence of target distance on eye movement responses during vertical linear motion. *Exp Brain Res* 1989;77:585–593.
4. Viirre E, Tweed D, Milner D, Vilis T. A reexamination of the gain of the vestibuloocular reflex. *J Neurophysiol* 1986;56:439–450.
5. Grossman GE, Leigh RJ, Abel LA, Lanska DJ, Thurston SE. Frequency and velocity of rotational head perturbations during locomotion. *Exp Brain Res* 1988;70:470–476.
6. King OS, Seidman SH, Leigh RJ. Control of head stability and gaze during locomotion in normal subjects and patients with deficient vestibular function. In: Berthoz A, Graf W, Vidal PP, eds. *Second symposium on head-neck sensory-motor system*. New York: Oxford, 1992;568–570.
7. Pulaski PD, Zee DS, Robinson DA. The behavior of the vestibulo-ocular reflex at high velocities of head rotation. *Brain Res* 1981;222:159–165.
8. JC. Living without a balancing mechanism. *N Engl J Med* 1952;246:458–460.
9. Grossman GE, Leigh RJ. Instability of gaze during locomotion in patients with deficient vestibular function. *Ann Neurol* 1990;27:528–532.
10. Baloh RW, Jacobson K, Honrubia V. Idiopathic bilateral vestibulopathy. *Neurology* 1989; 39:272–275.
11. Gresty M. Stability of the head: studies in normal subjects and in patients with labyrinthine disease, head tremor, and dystonia. *Mov Disord* 1987;2:165–185.
12. Guitton D, Kearney RE, Wereley N, Peterson BW. Visual, vestibular and voluntary contributions to human head stabilization. *Exp Brain Res* 1986;64:59–69.
13. Grossman GE, Leigh RJ, Bruce EN, Huebner WP, Lanska DJ. Performance of the human vestibulo-ocular reflex during locomotion. *J Neurophysiol* 1989;62:264–272.
14. Kasai T, Zee DS. Eye–head coordination in labyrinthine-defective human beings. *Brain Res* 1978;144:123–141.
15. Bronstein AM, Hood JD. Oscillopsia of peripheral vestibular origin. *Acta Otolaryngol (Stockh)* 1987;104:307–314.
16. Maas EF, Huebner WP, Seidman SH, Leigh RJ. Behavior of human horizontal vestibulo-ocular reflex in response to high-acceleration stimuli. *Brain Res* 1989;499:153–156.
17. Leigh RJ, Rushton DN, Thurston SE, Hertle RW, Yaniglos SS. Effects of retinal image stabilization in acquired nystagmus due to neurologic disease. *Neurology* 1988;38:122–127.
18. Wist ER, Brandt Th, Krafczyk S. Oscillopsia and retinal slip. *Brain* 1983;106:153–168.
19. Leigh RJ, Dell'Osso LF, Yaniglos SS, Thurston SE. Oscillopsia, retinal image stabilization and congenital nystagmus. *Invest Ophthalmol Vis Sci* 1988;29:279–282.
20. Dieterich M, Brandt T. Impaired motion perception in congenital nystagmus and acquired ocular motor palsy. *Clin Vision Sci* 1987;1:337–345.
21. Dell'Osso LF, Daroff RB. Congenital nystagmus waveforms and foveation strategy. *Docu Ophthalmol* 1975;39:155–182.
22. Dell'Osso LF, Leigh RJ. Foveation period stability and oscillopsia suppression in congenital nystagmus. *Neuro-ophthalmology* 1992;12:169–183.

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