J Neurol (1982) 226:243–254

Journal of
Neurology

Eye-Head Coordination in Homonymous Hemianopia

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Summary. Quantitative studies of latencies and trajectories of eye-head movements during visual search and fixation in patients with occipital hemianopia show that (1) the latency of head movement is bilaterally increased, with significantly greater delays in movements toward the blind side; (2) the compensatory eye movements (CEMs) during head movements toward the blind side have increased velocity; (3) these CEMs entail a non-vestibular anticipatory component; and (4) head movements toward the blind side are comprised of multiple steps similar to staircase eye movements documented in previous studies. Hemianopic patients seemingly simplify search and fixation strategies by minimizing or entirely eliminating head movements and relying on eye movements instead.

Key words: Eye-head coordination – Homonymous hemianopia – Target search strategies

Zusammenfassung. Quantitative Untersuchungen der Augen- und Kopfbewegungen, die von Patienten mit occipitaler Hemianopsie zum Erfassen und Fixieren visueller Ziele verwendet werden, zeigen: (1) beidseits verlängerte Latenzen für Kopfbewegungen, bei einer signifikant zusätzlich verlängerten Latenz für Kopfbewegungen zum blinden Halbfeld; (2) erhöhte Geschwindigkeit der kompensatorischen Augenbewegungen für Kopfbewegungen zum blinden Halbfeld; (3) eine nicht-vestibuläre, antizipatorische Komponente dieser kompensatorischen Augenbewegungen bei Kopfwendungen zum blinden Halbfeld, und (4) multiple treppenförmige Kopfbewegungen, ähnlich den treppenförmigen sakkadischen Augenbewegungen dieser Patienten bei Wendungen zum blinden Halbfeld. Patienten mit homonymer Hemianopsie

* On leave from the Department of Neurology, University of Hamburg, FRG, with support from Deutsche Forschungsgemeinschaft Bonn, FRG

** On leave from the Department of Neurology, University of Bern, Switzerland, with support from Geigy Jubilaeumsstiftung Basel and Schweizerische Stiftung für Medizinisch-Biologische Stipendien

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0340-5354/82/0226/0243/$ 02.40

vereinfachen und sichern offenbar ihre Strategien zum Aufsuchen und Fixieren visueller Ziele dadurch, daß sie ihre Kopfbewegungen zugunsten von Augenbewegungen eliminieren.

Introduction

In looking to either side, normal persons coordinate eye and head movements in a single act consisting of a rapid saccade to the target followed by a head movement. To keep the gaze fixed on target during the head movement, the eyes are driven in the opposite direction by the vestibular ocular reflex. The quantitative aspects of these movements have been determined in monkeys [7, 11, 21, 26] and normal human beings [4, 5, 13, 17, 35, 42]; and some abnormalities in those movements have been reported in studies of patients [1, 19, 43].

Various clinical studies of eye movements of patients with occipital hemianopia have defined the adaptive ocular motor strategies employed by these patients [14, 23, 29, 30, 32, 36], and the possible role of extrastriate vision in these strategies has been considered [12, 25, 28, 39]. When searching for targets in their blind hemifield, such patients at first employ a safe but slow strategy consisting of a stepwise series of saccades (Fig. 1). Subsequently, they adopt more efficient strategies for particular situations. When the position of the target is predictable, they use a strategy consisting of a single, large saccade that slightly overshoots the target. When fixating targets in their seeing hemifield, they use a saccade that slightly undershoots the target.

This report documents and analyzes the adaptive changes in combined eye-head movements of patients with homonymous hemianopia.

Methods

Eye Movement Recordings

Horizontal eye movements were recorded by means of the infrared reflection method [2, 34]. The recordings were linear within the range of the eye movements examined. The system bandwith was 0 to 150 Hz, with the upper frequency limit determined by the range of the rectilinear chart recorder. A chart speed of 100 mm/s was used in order to allow measurement of latencies and detailed analysis of trajectories. In addition, all data were stored on an Ampex FR 1300 tape recorder and, as necessary, could be played back at different speeds or further analyzed on a PDP 8 Minicomputer.

To prevent interference from vergence movements, eye movements were recorded monocularly from the eye with the temporal field defect. The other eye was covered with a patch. In patient 1, comparison of recordings from each eye alone and both together showed no differences in movement patterns.

Head Movement Recordings

Rotational head movement in the horizontal plane was measured with an apparatus previously described [41]. Briefly, it consisted of a bicycle helmet, on top of which was mounted an acryl rod flexible enough to act as a universal joint; to the top of the rod was clamped a low-torque, low-backlash potentiometer fixed in a parallelogram linkage frame. The electrical signal from the potentiometer varied directly with torsional movement of the helmet. The apparatus was strapped on the subject's head and positioned at the center of a horizontal arc perimeter.

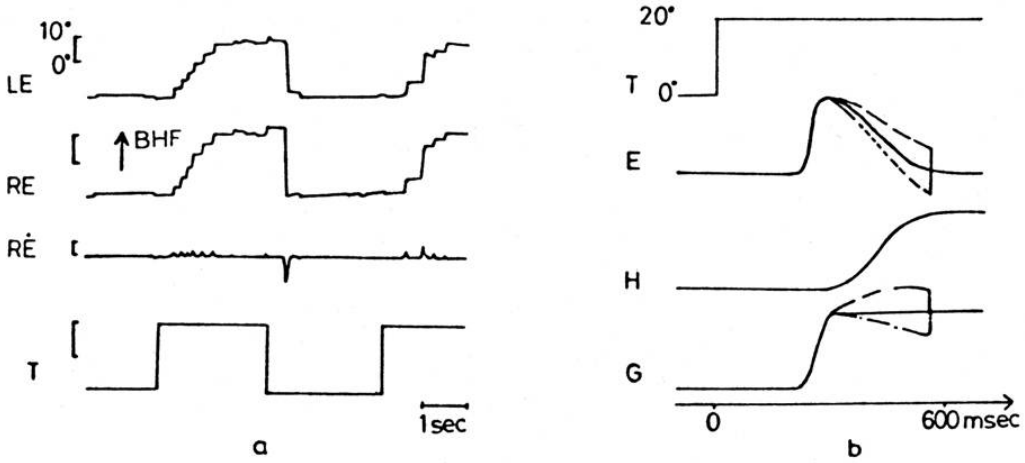

Fig. 1. a Eye movement traces in a patient with acquired hemianopia: *LE* = left eye position, *RE* = right eye position, *RE* dot = right eye velocity, *T* = target position. Note that in attempting to find and fixate an unpredicted target yields stairsteps to the BHF. b *T* = target, *E* = eye, *H* = head, *G* = gaze (i.e., eye position plus head position, which represents eye position in space) response to a jump of the target to 20° left. The thick line indicates unity in compensatory eye movement *(CEM)* gain; the short-dashed line, increased *CEM* gain; the long-dashed line, decreased *CEM* gain

A rigid metal rod 37 cm long, with a small, red fixation target at its end was attached to the front of the helmet so that it pointed straight ahead. This apparatus showed linearity over + 90° to each side of center with an accuracy of ± 0.3°. Bandwidth of the entire system was 150 Hz.

Standard statistical techniques were used in analyzing our data [8]. In all cases, at least 35 samples from the recording were used for *t*-test and linear regression analysis.

Stimulus Presentation

The target was a small, green spot presented on a Tektronix 611 cathode ray tube; it subtended a visual angle of 10 min and had a luminance of 6 ft-lamb (20.6 cd/m^2). The luminance of the screen was 0.4 ft-lamb (1.4 cd/m^2). The overall room illumination was about 6 cd/m^2, equivalent to a low photopic level.

The target was always presented in the horizontal plane within 10° of either side of center. Target presentation programs were controlled by a PDP 8 Minicomputer that produced target steps between 1° and 20° at an eye distance of 37 cm. Three programs of target presentation were used, each with different spatial and temporal parameters. In addition, predictable ramp targets moving at different velocities were used for testing head and eye pursuit movements.

Program 1: Predictable Amplitude and Time (Predictable Program). For the Predictable Program (Fig. 1), the eccentricity and duration of presentation were constant, the side alternated, and the target light was on continuously. Recordings were made with target shifts of 20°, 10°, 5°, and 2.5°. The interval between target shifts was 2.5 s.

Program 2: Random Amplitude and Time (Random Program). For the Random Program, the eccentricity, side, and duration of presentation were all randomized, and the target light was on continuously. Target eccentricity varied randomly between 0° and 20°. The target remained in one position for anywhere from 100 ms to 2.5 s.

Program 3: Predictable Amplitude with Random Time (Random On/Off Program). For the Random On/Off Program, the duration and side of presentation were randomized, the eccentricity was constant, and the target light was randomly off. Recordings were made for eccentricities of 20°, 10°, 5°, and 2.5°.

Protocol

The Predictable and Random Programs presented "forced-choice" situations [29, 32] in that as soon as the target moved, it had to be sought in either the seeing or the blind hemifield since it was continuously illuminated. The patients were instructed always to search for the target and to fixate on it as long as it did not move. For the Random On/Off Program, the patients were told that they should look at the target as soon as it appeared.

To avoid fatiguing the patients, each program was run in several short segments with frequent pauses. For the first run of each program, patients were instructed to move their eyes and head as accurately as possible in the way that felt natural. For subsequent runs, they were instructed to turn their head toward the target as accurately and quickly as possible (attempted time-optimal head movements [41]).

Patients

The subjects for this study were six patients with homonymous hemianopia. Patients 1, 2, and 3 illustrate acquired visual defects producing respectively decreasing degrees of disability in everyday life. Patients 4, 5, and 6 illustrate striking adaptation in everyday life to lifelong homonymous hemianopia.

Patient 1

Eight years before the study, this 55-year-old man with metastatic renal cell carcinoma experienced the sudden onset of left hemiparesis and complete, dense left homonymous hemianopia with macular splitting. Computerized tomographic (CT) scans and angiograms revealed a right occipital hemorrhage originating from a metastatic tumor extending into the parietal lobe and another located in the left temporal region. The hemiparesis completely resolved in the following weeks, but total hemianopia remained. The patient's vision was 20/40 in the right eye and 20/25 in the left. Optic fundi were normal. At the time of the study, the patient was still moderately disabled.

Patient 2

Three years ago, this 42-year-old man presented with "migraine" and signs of right homonymous hemianopic paracentral scotomas. Angiograms and CT scans revealed a left occipital arteriovenous malformation. After resection of the lesion, he had a total right homonymous hemianopia. Visual acuity was 20/20 in each eye. The fundi were normal, and neurological examination revealed no other abnormalities. When studied, the patient was not taking medications. He felt moderately disabled by his visual field defect, but it had been several weeks since he had walked into an object on his blind side.

Patient 3

Upon awakening after a cardiac operation, this 55-year-old man had a right hemiparesis and complete, dense right homonymous hemianopia that spared fixation. Angiograms and CT scans showed a left occipital infarction. The paresis resolved completely in the following weeks, but the visual defect remained. Visual acuity was 20/20 in each eye. Optic fundi were normal. The patient had ceased taking medication months before he was studied. He had not walked into an object on his right side for three months and no longer regarded himself as disabled by his homonymous field defect.

Patient 4

This 30-year-old man had had total left homonymous hemianopia all his life. Ocular fundus signs suggested transsynaptic degeneration from an occipital lesion. Visual acuity was 20/20 in each

eye. CT scans showed right occipital porencephaly. No additional neurological abnormalities were found. The patient had compensated so well for his congenital visual field defect that he had a record of 10 years of accident-free driving.

Patient 5

This 33-year-old man had a perinatally acquired total right homonymous hemianopia with typical ophthalmoscopic signs of transsynaptic retrograde degeneration. His visual acuity was 20/20 in each eye. CT scans revealed no occipital lobe abnormality. His lifelong compensation for his hemianopia was excellent.

Patient 6

This 23-year-old man presented with a total right homonymous hemianopia acquired at 2 months of age from an intracerebral hemorrhage. Since early childhood he had had generalized motor seizures involving the right side of the body. CT scans showed moderate ventricular enlargement and left posterior porencephaly. Ophthalmoscopic examination indicated transsynaptic retrograde degeneration in both optic fundi. Visual acuity was 20/40 in each eye. Reflexes in his right extremities were hyperactive, the right plantar reflex was extensor, and he had variable nystagmic eye movements during fixation. His lifelong compensation for his visual field defect was excellent.

Results

Patients with hemianopia differed in important ways from normal subjects described previously [41, 42]. The patients often reduced or altogether omitted the

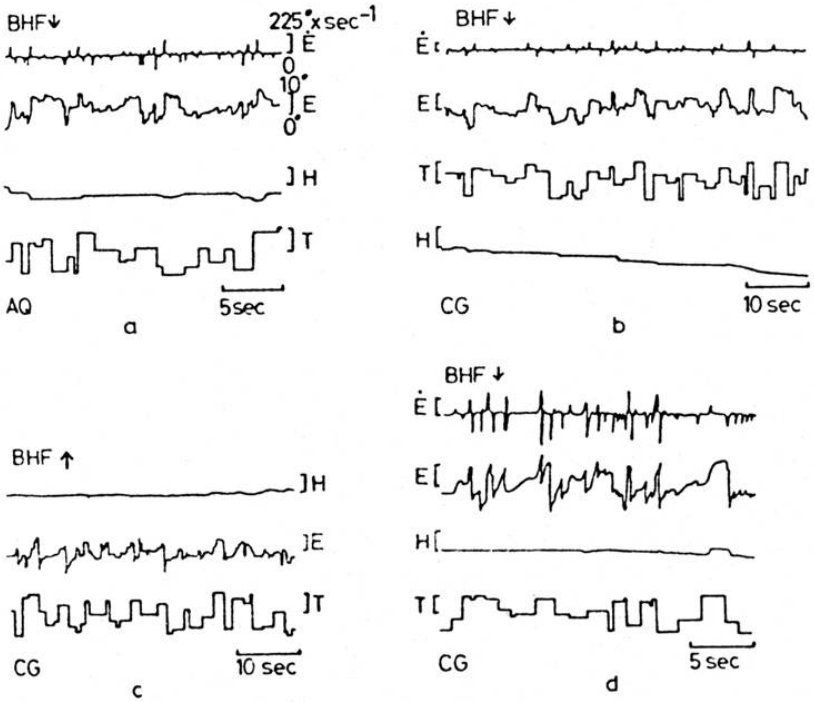

Fig. 2a–d. Eye- and head-movement patterns in target-searches by four patients with homonymous hemianopia, showing tendency to omit head movements, which is most pronounced in congenital (*CG*) cases **b**, **c**, and **d**, but present also in **a**, a patient with acquired (*AQ*) hemianopia. Note the more frequent use of head movements in the patient with acquired defect. Compare slow head drift to BHF in **b** to fast eye drift away from BHF in **d**

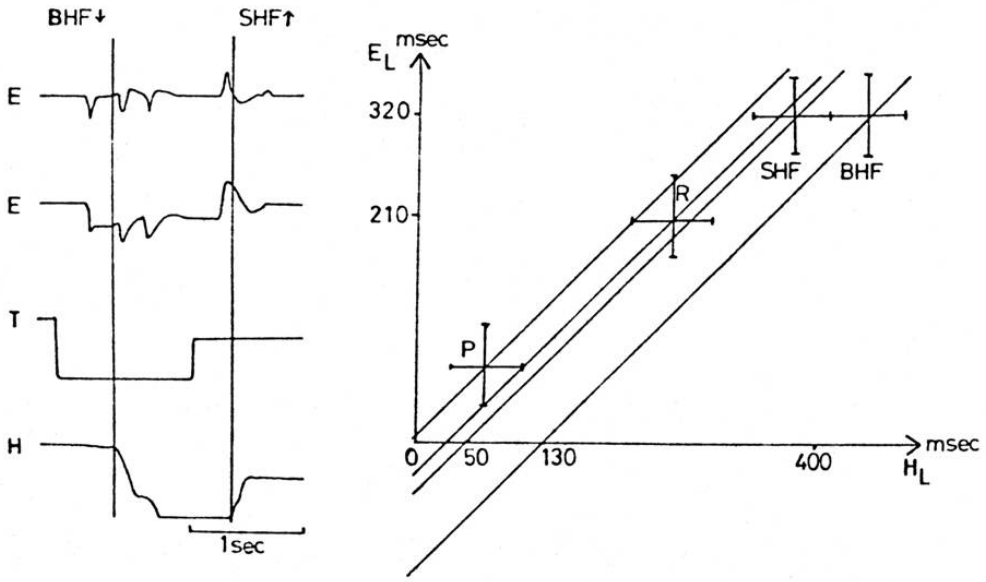

Fig. 3a and b. Latencies of eye (*EL*) and head (*HL*) movements of hemianopic patients when seeking unpredictable targets. **a** Original recording sample in a patient with acquired right hemianopia shows *EL* is similar in the time required for gaze shift to the *BHF* (290 ms) and to the *SHF* (240 ms), but *HL* takes significantly longer for gaze shift to the *BHF* (520 ms) than to the *SHF* (330 ms). **b** Model lines for *EL* and *HL*, including standard deviation [42] in normal subjects with random (*R*) and predictable (*P*) target jumps of 15°: head movement shows an additional delay of 40 ms with *R* (*EL*, 210 ms; *HL*, 40 ms). With *P*, *EL* and *HL* decrease to averages of 90 and 60 ms, respectively—the head predicting better than the eye. Correlation coefficients for *R* and *P* lines are: in normal subjects (*R*) $r = 0.86$, (*P*) $r = 0.90$; in hemianopic patients (*SHF*) $r = 0.95$, (*BHF*) $r = 0.85$. Model lines show, besides the increased *EL* (320 ms) and *HL* (370 ms), an additional head movement delay of 80 ms with gaze to the *BHF*

head-movement component of gaze shift (Fig. 2); consequently, the eye movement was more pronounced. Head movements tended to be more diminished in patients with congenital hemianopia (Fig. 2a, b) than in those with an acquired defect (Fig. 2c, d). Head movements to the seeing hemifield followed eye movements with the same 50 ms delay observed in normal subjects [42], but there was an additional delay of 80 ms on the average in head movements to the blind hemifield (Fig. 3a). Eye movements, by contrast, were symmetrically delayed by about 110 ms more than in normal subjects [23, 27, 37] (Fig. 3b).

In patients with acquired hemianopia, head movement velocities—and consequently the compensatory eye movement (CEM) velocities—varied considerably. The ratio of CEM velocity to head velocity (CEM gain, Fig. 4) was small with head movements toward the seeing hemifield, great with movements toward the blind hemifield. Moreover, the CEM on the blind side often preceded the head movement, resulting in an increase in the CEM gain. This anticipatory CEM could not have been driven by the vestibular ocular reflex. The fast CEM, whether resulting from an increased vestibulo-ocular reflex gain or from the addition of the anticipatory component, was often combined with a superimposed saccade as the head moved toward the blind hemifield.

Patients with congenital hemianopia had more complex patterns of eye-head movement (Fig. 4c, d). In eye-head movements toward the blind hemifield, there

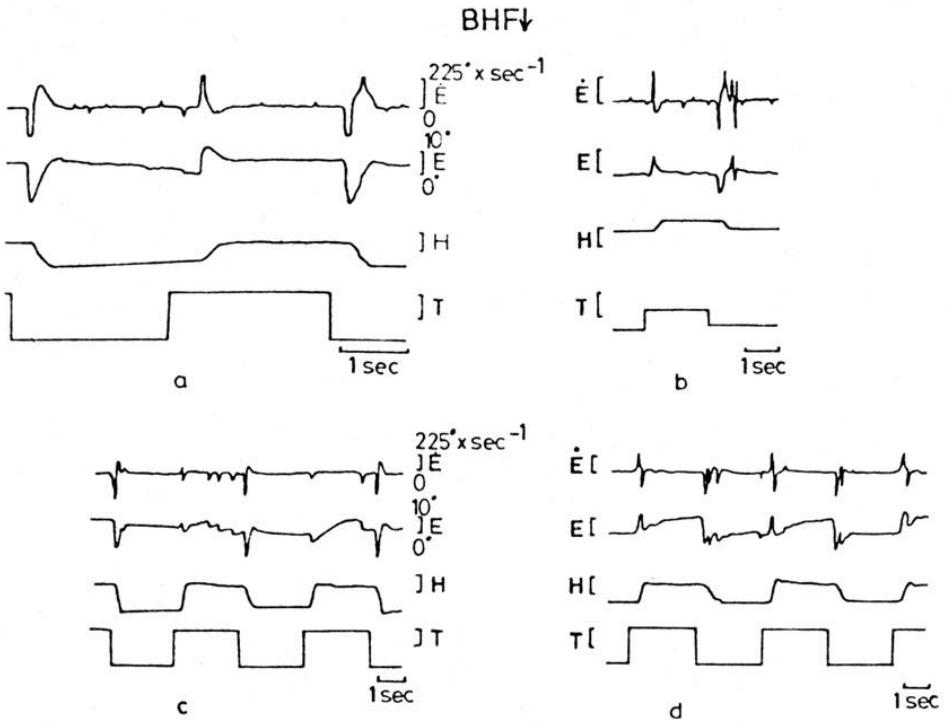

Fig. 4a–d. Eye- and head-movement traces in two hemianoptic patients: **a** and **b** patient with acquired hemianopia, **c** and **d** patient with congenital hemianopia. Note the especially high *CEM* gain in gaze shift to *BHF* in both **a**, **b** and **c**, **d**; the patient with the congenital defect shows, in addition, a fast, continuous drift away from the *BHF* that increases its velocity in relation to eccentricity on the blind side; this drift also occurs in eye movements to the *SHF*, where there is hardly any *CEM* and eye drift, and saccades appear to be used in a compensatory manner

was not only a high CEM gain, but also a continuous drift of the eye toward the seeing hemifield, regardless whether there was a head movement. The drift was faster when the eyes turned to the blind hemifield (Fig. 2d; 4c, d). The continuous drift was present also during eye movement toward the seeing hemifield, but the CEM was suppressed (Fig. 4d).

Head movements toward the blind hemifield often showed a staircase pattern (Fig. 5) that was similar to that of visual saccades but irregular and variable. During these staircase movements there was synkinesis of head and eye movements. Often, there were two to four saccades that interacted with the CEM (Fig. 5). By contrast, head movements into the seeing hemifield often consisted of a single, large movement.

For acquiring targets in the blind hemifield, patients with acquired hemianopia employed staircase patterns in their head movements more frequently than those with congenital hemianopia, who tended to eliminate head movements and substitute single, large, overshooting saccades with contraversive drifts.

To find and fixate Random On/Off targets in their blind hemifield (Fig. 6a–d), patients used various patterns of eye-head movements. In this task, more than the others, performance was strongly influenced by the patient's attentiveness. A frequently employed strategy was to move the head back and forth. Sometimes, these head movements were accompanied by simultaneous stairstep head move-

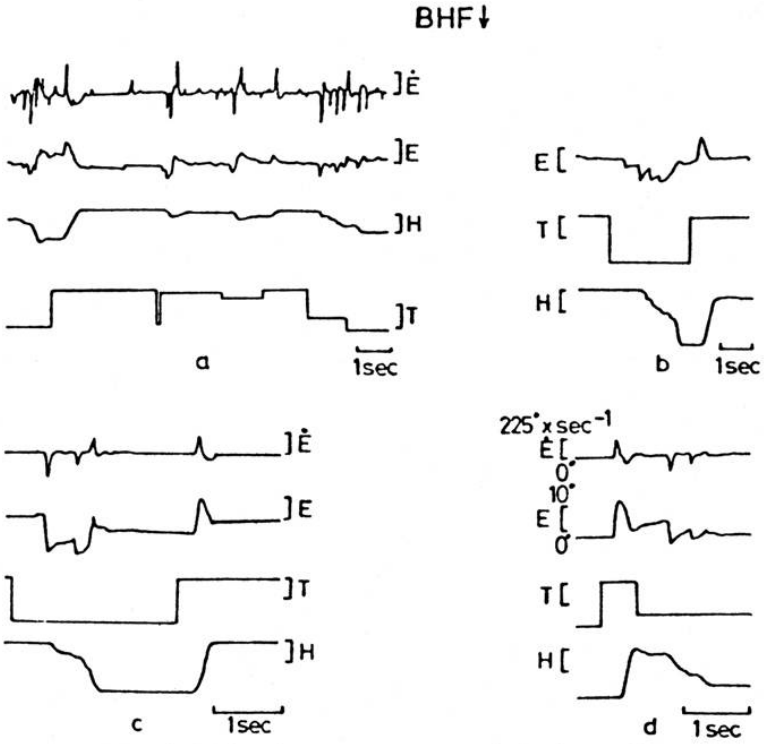

Fig. 5a–d. Eye- and head-movement traces in both a patient with congenital hemianopia (**a, b**) and a patient with acquired hemianopia (**c, d**) show staircase patterns of head movement, either **b** without *CEMs* or **a**, **c**, and **d** with *CEMs* of high gain (especially **a**); compensatory eye saccades (as in **a** and **c**) are frequently superimposed upon ongoing *CEMs*

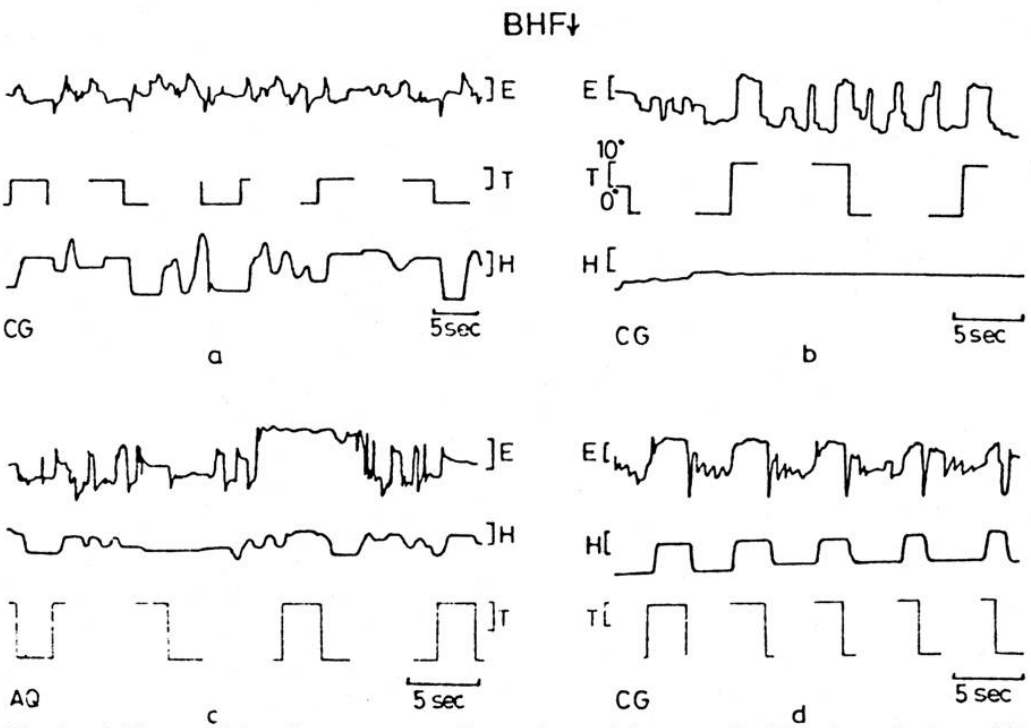

Fig. 6a–d. Eye- and head-movements of a patient with congenital hemianopia (**a** and **b**) and a patient with acquired hemianopia (**c** and **d**) attempting to find targets presented with the Random On/Off Program. **a** Large, searching head movements and **b** omission of head movements, including a slow drift of the head to and from the *BHF*. **c** The patient with acquired defect shows smaller searching head movements. **d** Amplitude-predicting head movements with idiosyncratic use of fast, continual eye drift to the *SHF*. Eye movements often show staircase and searching patterns

ments similar to the patient's eye movement patterns. Another strategy for dealing with Random On/Off targets was to eliminate head movements in favor of eye movements (Fig. 6b). A strategy sometimes employed by patients with congenital hemianopia was to shift their gaze into the blind hemifield as soon as the target disappeared and then to wait there for it to reappear. These four strategies were very similar to the strategies patients employed when they were allowed to use only eye movements to find targets.

Discussion

Our previous studies of patients with homonymous hemianopia [23] have shown eye movements to be highly able to predict target locations; the head movements measured and analyzed in the present study are much less effective in this capacity.

Not only is the head-movement portion of coordinated eye-movements more variable and definitely less able to predict target locations than the eye-movement portion, it is frequently omitted altogether from movements toward either hemifield. Perhaps the problems of finding and fixating targets are so difficult for hemianopic patients that they simply choose the safer strategy of keeping their head still and relying on eye movement.

When hemianopic patients move their eyes alone, the eye movement latencies to either side are increased equally; but when they move their head to shift gaze laterally, the latency of the head movement shows a definite increase; this is greater when shifting gaze toward the blind hemifield. We do not know why the latency is greater in the latter instance. We think it may be that, with unpredictable signals, head movement programming, which entails more complex movements than eye-movement programming alone [22, 42], takes more time for patients (such as our subjects) who have posterior hemispheric lesions.

The CEM plays a critical role in coordinated eye-head movement. CEMs are controlled not only by vestibular reflexes [16, 20, 40], but also by inputs from the visual [6, 9, 26, 38] and cerebellar [3, 10, 15, 18, 22, 24, 31, 33] systems. The modifications in CEM gain that we noted in our patients, whether with congenital or acquired hemianopia, were highly asymmetrical, the gain being greater with gaze shift toward the blind hemifield, and less with gaze shift toward the seeing hemifield. During gaze shift toward the blind hemifield, high-gain CEMs are usually preceded by an overshooting saccade. Here, high-gain CEM serves to keep the seeing side of the macula on target. However, high-gain CEM is a handicap when the patient employs a sequence of undershooting, staircase saccades to find and fixate the target on the blind side. Perhaps this maladaptation is one of the reasons that patients prefer to keep their heads still—and thus eliminate the need for any CEM—while searching for targets on their blind side. During the extended interval between saccade and head movement, hemianopic patients often employ a post-saccadic backward drift beginning before the initiation of the vestibularly driven CEM. While such an anticipatory component can occur as a normal component of synkinesis during eye-head movement [22, 42], it becomes pronounced in hemianopic patients because of the prolonged latency of head movement during gaze shift toward the blind hemifield.

The similarity of the staircase head movents employed by hemianopic patients in looking toward their blind hemifield to the staircase saccades they employ when seeking targets with the eye alone implies that there is a common, higher-level control of eye and head movements. We believe that the much greater variability of the staircase head-movement reflects greater volitional control over their generation.

Our previous studies of eye movements in patients with homonymous hemianopia clearly suggest the presence of useful compensatory strategies for finding and fixating objects. Our present study of the head-movement component in the gaze shift of hemianopic patients shows that there are compensatory modifications in head movements and their synkinesis with eye movements, but it does not provide any evidence of useful adaptations for finding and fixating targets. Rather, our study reveals erratic patterns that appear to demonstrate definite limitations in the capacity of the brain to adapt usefully to a complex sensory-motor task. The modifications in head movement latency, CEM gain, and anticipatory drift represent low-level effects in hemianopic patients. To make the adaptations that are necessary to achieve normal performance of complex sensory-motor tasks, a higher level of brain function is required. Finding and fixating targets in the presence of homonymous hemianopia presents a sensory-motor adaptive challenge so complex that the brain seemingly responds by simplifying the task: it eliminates the head-movement component of the synkinesis, stabilizing the head in the service of vision.

Acknowledgement. For clinical assistance, the authors are grateful to Dr. M. Rosenberg, Fellow in Neuroophthalmology, University of California, San Francisco.

References

1. Atkin A, Bender MB (1968) Ocular stabilization during oscillatory head movements. Arch Neurol 19:559–566
2. Bahill T, Clark M, Stark L (1975) Dynamic overshoot in saccadic eye movements is caused by neurological control signal reversals. Exp Neurol 48:107–122
3. Baloh RW, Jenkins HA, Honrubia V, Yee RD, Lau CGY (1979) Visual-vestibular interaction and cerebellar atrophy. Neurology 29:116–119
4. Barnes GR (1979) Vestibulo-ocular function during coordinated head and eye movements to acquire visual targets. J Physiol 287:127–147
5. Bartz J (1966) Eye and head movements in peripheral vision: Nature of compensatory eye movements. Science 152:1644–1645
6. Bisti S, Maffei L, Piccolino M (1974) Visuovestibular interactions in the cat superior colliculus. J Neurophysiol 37:146–155
7. Bizzi E, Kalil RE, Tagliasco V (1971) Eye-head coordination in monkeys: Evidence for centrally patterned organization. Science 173:452–454
8. Cochran WG, Cox GM (1957) Experimental designs, 2nd edn. Wiley, New York
9. Cohen B, Matsuo V, Raphan T (1977) Quantitative analysis of the velocity characteristics of optokinetic nystagmus and optokinetic after-nystagmus. J Physiol 270:321–344
10. Collewijn H, Kleinschmidt JH (1975) Vestibular-ocular and optokinetic reactions in the rabbit. Changes during 24 hours of normal and abnormal interaction. In: Lennerstrand G, Bach-y-Rita P (eds) The control of eye movements. Pergamon Press, Oxford, pp 477–483
11. Dichgans J, Bizzi E, Morasso P, Tagliasco V (1974) The role of vestibular and neck afferents during eye-head coordination in the monkey. Brain Res 71:225–232

12. Feinberg TE, Pasik T, Pasik P (1978) Extrageniculostriate vision in the monkey. VI. Visually guided accurate reaching behavior. Brain Res 152:422–428
13. Fleming DG, Vossius GW, Bowman G, Johnson EL (1969) Adaptive properties of the eye-tracking system as revealed by moving-head and open-loop studies. Ann NY Acad Sci 156: 825–850
14. Gassel MM, Williams D (1963) Visual functions in patients with homonymous hemianopia. II. Oculomotor mechanisms. Brain 86:1–36
15. Gauthier GM, Hofferer J-M, Hoyt WF, Stark L (1979) Visual-motor adaptation: Quantitative demonstration in patients with posterior fossa involvement. Arch Neurol 36:155–160
16. Goldberg JM, Fernández C (1980) Efferent vestibular system in the squirrel monkey: Anatomical location and influence of afferent activity. J Neurophysiol 43:980–1025
17. Gresty MA (1974) Coordination of head and eye movements to fixate continuous and intermittent targets. Vision Res 14:395–403
18. Ito M, Shiida T, Yagi N, Yamamoto M (1974) Visual influence on rabbit horizontal vestibulo-ocular reflex presumably effected via the cerebellar flocculus. Brain Res 65:170–174
19. Kasai T, Zee DS (1978) Eye-head coordination in labyrinthine-defective human beings. Brain Res 144:123–141
20. King WM, Precht W, Dieringer N (1980) Synaptic organization of frontal eye field and vestibular afferents to interstitial nucleus of Cajal in the cat. J Neurophysiol 43:912–928
21. Kowler E, Steinman R (1979) Anticipatory smooth eye movements are produced by expectations of highly predictable target motions. Abstracts of the Annual Spring Meeting of the Association for Research in Vision and Ophthalmology (Miami, Florida, May 4–9, 1979), p 101
22. Latto R (1978) The effects of bilateral frontal eye-field, posterior parietal or superior colliculus lesions on visual search in the rhesus monkey. Brain Res 146:35–50
23. Meienberg O, Zangemeister WH, Rosenberg M, Hoyt WF, Stark L (1981) Saccadic eye movement strategies in patients with homonymous hemianopia. Ann Neurol 9:537–544
24. Melvill Jones G, Davies P, Gonshor A (1977) Long-term effects of maintained vision reversal: Is vestibulo-ocular adaptation either necessary or sufficient? In: Baker R, Berthoz A (eds) Control of gaze by brainstem neurons: Proceedings of the symposium held in the Abbaye de Royaumont, Paris, July 12–15, 1977. Elsevier/North Holland Biomedical Press, Amsterdam, pp 59–68
25. Mohler CW, Wurtz RH (1977) Role of striate cortex and superior colliculus in visual guidance of saccadic eye movements in monkeys. J Neurophysiol 40:74–94
26. Morasso P, Bizzi E, Dichgans J (1973) Adjustment of saccade characteristics during head movements. Exp Brain Res 16:492–500
27. Ochs A, Hoyt WF, Stark L, Patchman M (1978) Saccadic initiation time in multiple sclerosis. Ann Neurol 4:578–579
28. Pasik P, Pasik T, Schilder P (1969) Extrageniculostriate vision in the monkey: Discrimination of luminous flux-equated figures. Exp Neurol 24:421–437
29. Perenin MT, Jeannerod M (1978) Visual function within the hemianopic field following early cerebral hemidecortication in man. I. Spatial localization. Neuropsychologia 16:1–13
30. Pöppel E, Held R, Frost D (1973) Residual visual function after brain wounds involving the central visual pathways in man. Nature 243:295–296
31. Robinson DA (1976) Adaptive gain control of the vestibuloocular reflex by the cerebellum. J Neurophysiol 39:954–969
32. Sharpe JA, Lo AW, Rabinowitch HE (1979) Control of saccadic and smooth pursuit system after hemidecortication. Brain 102:387–403
33. Simpson JI, Alley KE (1974) Visual climbing fiber input to rabbit vestibulo-cerebellum: A source of direction-specific information. Brain Res 82:302–308
34. Stark L, Vossius G, Young L (1962) Predictive control of eye tracking movements. IRE Trans, Human Factors in Electronics 3:52–57
35. Sugie N, Wakakuwa M (1970) Visual target tracking with active head rotation. IEEE Trans SSC 6:103–109
36. Troost BT, Weber RB, Daroff RB (1972) Hemispheric control of eye movements. I. Quantitative analysis of refixation saccades in a hemispherectomy patient. Arch Neurol 27:441–448

37. von Noorden GK (1961) Reaction time in normal and amblyopic eyes. Arch Ophthalmol 66: 695–701
38. Waespe W, Henn V (1977) Neuronal activity in the vestibular nuclei of the alert monkey during vestibular and optokinetic stimulation. Exp Brain Res 27:523–538
39. Weiskrantz L, Warrington EK, Sanders MD, Marshall J (1974) Visual capacity in the hemianopic field following a restricted occipital ablation. Brain 97:709–728
40. Wilson VJ, Maeda M, Franck JI (1975) Inhibitory interaction between labyrinthine, visual and neck inputs to the cat flocculus. Brain Res 96:357–360
41. Zangemeister WH, Jones A, Stark L (1981) Dynamics of head movement trajectories: Main sequence relationship. Exp Neurol 71:76–91
42. Zangemeister WH, Stark L (1980) Active head rotation and eye head coordination. Conference on Vestibular and Oculomotor Physiology and International Meeting of the Barany Society (New York City, September 1980). Ann NY Acad Sci (in press)
43. Zee DS (1977) Disorders of eye-head coordination. In: Brooks BA, Bajandas FJ (eds) Eye movements: ARVO symposium 1976. Plenum Press, New York, pp 9–39

Received June 19, 1981