EXPERIMENTAL NEUROLOGY 75, 389-406 (1982)

Gaze Latency: Variable Interactions of Head and Eye Latency

WOLFGANG H. ZANGEMEISTER AND LAWRENCE STARK[1]

Departments of Physiological Optics, Neurology, and Bioengineering, University of California, Berkeley, California 94720

Received August 28, 1980; revision received July 30, 1981

Head movement latencies are greater than eye movement latencies because of dynamic biomechanical lags. Our EMG recordings show equal latencies in the controller signals to eye movement and to head movement. The increased dynamic lag of head movement leads to the classical gaze pattern. First a rapid saccadic eye movement directs gaze onto target; a slower head movement follows. Its accompanying vestibular ocular reflex exchanges head position for eye position; the eye stays on target throughout. At the end of the movement, the eye is returned to the primary position. Head movement latencies are readily modified by experimental conditions such as instructions to the subject, frequency and predictability of the target, amplitude of the movement, and development of fatigue. They are affected by neurological disease processes. Effects on head latency are mirrored by idiosyncratic or covarying changes in eye movement latency. Covariance of latency in head and eye movements is attributed to concomitant higher level neurological processing because it is sensitive to stimulus predictability and to neural fatique. These experimental results may be readily demonstrated using a gaze latency diagram. They are also illustrated in a table derived from a branching model assignment of latencies according to a hypothetical neurological schema. The potential of these coordinated gaze latency studies for neurological diagnosis is illustrated in patients with homonymous hemianopsia.

Abbreviations: CEM—compensatory eye movement, VOR—vestibular ocular reflex, EMG—electromyogram.

[1] We are pleased to acknowledge partial support from the NCC 2-86 Cooperative Agreement, NASA-Ames Research Center, and from the National Eye Institute Training Grant, EY07043. We appreciate the helpful criticism from our colleagues, Gabriel Gauthier, Christopher Kennard, John Findlay, Stephen Feldon, and especially Tom Waite who also interacted with our experimental program. We want to thank the students of ME210L (Fall, 1979) for sharing their enthusiasm and data with us—Susan Hanum, John Winters, Patrick Donohoe, N. Farahbakhsh, John Shang, Victor Leowen, Allan Burbaum, John Fields, V. Lakshminarayanan, and Ted Merrill. Dr. Zangemeister was on leave from the Department of Neurology, University of Hamburg, F.R.G., supported by Deutsche Forschungsgemeinschaft, Bonn, F. R. G.

0014-4886/82/020389-18$02.00/0

INTRODUCTION

Coordinated gaze movements consist of an eye movement saccade, a head movement saccade, and a compensatory eye movement driven mainly by the vestibular ocular reflex. First a rapid eye movement directs gaze onto the target. A slower head movement saccade follows. The head movement is controlled by an electromyogram synchronous with the eye movement saccade. However, the head, being a larger mechanical object, requires an increased lag before its initial position change becomes noticeable, especially compared with the faster eyeball dynamics. The head movement is accompanied by the vestibular ocular reflex; acceleration of the head produces an eye movement opposite in direction and velocity to the head movement. This compensatory eye movement returns the eye to its primary position in the orbit, exchanging head position shift for the initial eye position jump. Coordinated gaze thus has the advantages of initial capture of the target by the fast eye saccade, of the eye staying on target throughout, and of the eye ending up in primary position in the orbit so that the direction sense points straight ahead at the target.

There are a large number of variants of this classical movement which show the importance of latency as a possible clue to the construction and generation of the neurologic control signals driving this important orienting and information processing system. Many researchers have studied sensory latency, e.g., of the visual system (27, 35, 36, 38), and also motor latency in eye as well as in head and arm movements (10, 19, 26, 31, 32, 34, 36). Saccadic eye movement latencies (10, 31, 34, 36, 38) show the influence of predictability and frequency of the target signal. Latency studies on coordinated gaze movements (1, 3–5, 7, 8, 14, 16–18, 22, 28, 37, 42) have begun to demonstrate the increased complexity and variability of head–eye movement interaction.

Clinicians have used latencies to define and elucidate various kinds of neurologic deficits. For example, studying the suppressed eye of strabismic and amblyopic patients, Mackensen (26) showed that the central fovea produced longer latency in hand movements and Ciuffreda *et al.* (10) showed that stimuli falling onto the "near periphery" of the retina, within about 10°, also produced longer-latency eye movements. An important finding is that in common neurologic diseases such as multiple sclerosis, latencies provide a precise and reliable indication of disease processes occurring in conductive pathways (31).

In the present studies of latency of coordinated gaze movements, we refine the experimental methods and define stimulus conditions to demonstrate important dependencies of the various components of gaze latency,

to show an application to a neurological disease (27, 42).

METHODS

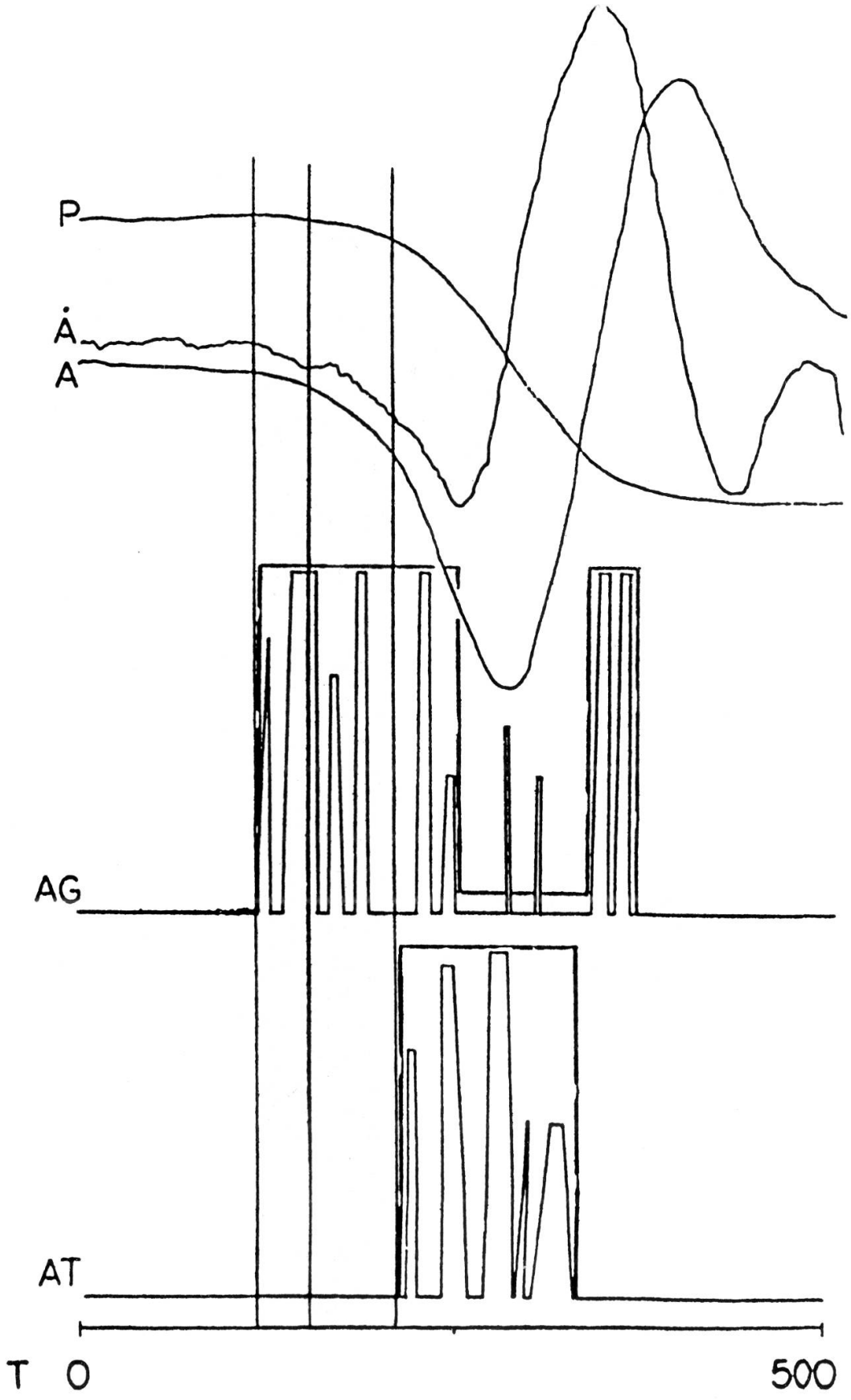
Head and eye movement recording techniques used in our laboratory were recently reported (39). Briefly, mechanical linkages and potentiometers are attached to a helmet that the subjects or patients wore while viewing targets. Residual play between helmet and head resulted in 30-ms artifactual latency determined by head-mounted (via bite bar) photocell calibration and accounted for by subtraction in the results below. The eye movement recordings used either the infrared limbus reflection technique (2, 36) or, for large amplitudes such as combined eye–head movements of 30° or 60°, monocular electroculogram miniature surface electrodes (Beckmann) and differential DC amplifiers (Tektronix 5002).

Electromyograms (EMG) were measured using surface electrodes on pairs of neck muscles, such as splenius and sternocleidomastoideus, that contribute to horizontal rotation of the head (40) and fed into the computer at 2000 samples per second.

The target appeared as a bright continuously lighted spot, 30 arc min in extent, on a dark screen. Stimulus programs included both predictable and unpredictable sequences of target steps. Predictable target jumps of four amplitudes, 15°, 30°, 40°, and 60°, were produced with frequencies between 0.1 and 1.9 Hz. Unpredictable (both in amplitude and time) target shifts were generated with amplitudes between 6° and 60°; both timing and amplitude selection were by a digital computer (PDP-8) using a random number generator.

Subjects sat comfortably in front of the screen and for the first run were advised to engage in "natural" (NAT) head and eye movements. In a second run they forced themselves to perform "intended" time optimal (39, 41) head and eye movements as fast and as accurately as possible ["forced (FOR) condition]. Three colleagues performed fast, accurate responses and served as test subjects together with four naive subjects. Clinical descriptions of the six patients with homonymous hemianopsia have been reported (27, 42). For any one type of stimulus condition at least 35 samples were used for standard statistical analyses such as t test and linear regression fits (11). Data were recorded on a rectilinear chart recorder, on FM magnetic tape, or fed into the laboratory minicomputer system (PDP-8) as previously described (39).

Compensatory eye movement (CEM) and vestibular ocular reflex (VOR) are terms generally used in the literature almost indistinguishably, often


P
Å
A
AG
AT
T 0
500

acceleration and the first derivative of head acceleration (Fig. 2). Agonist and antagonist EMG traces demonstrated reciprocal innervation—the agonist EMG reduced rapidly as the antagonist EMG appeared. The controller signal envelopes (heavy semidashed lines, Fig. 2) were the neurological expression of the EMG signal, summing up the firing of a large number of neurons that drove the many motor units of a particular muscle. The result of muscle force generation was a biphasic acceleration and deceleration (Fig. 2, lowermost trajectory, A) that produced a change of head position (Fig. 2, uppermost trajectory, P). Acceleration was a much more sensitive indicator of dynamic behavior than position change and showed closer dependence on underlying neurologic control signals. The dynamics could be further understood by considering rate of change of acceleration (Fig. 2, intermediate trajectory, Ȧ); early indications of results of EMG firing could be first noted in this time function that was intermediary between muscle force and head acceleration. The most important consequence of the dynamical lag between EMG and head movement was the approximate 55-ms delay to the beginning of noticeable position change. These recordings (Figs. 1, 2) documented the apparent delay of head movement with respect to eye movement as due to lags secondary to head movement dynamics.

Gaze Latency as Function of (i) Instructions, (ii) Amplitude, (iii) Predictability, and (iv) a Neurological Deficit

Gaze Latency Diagram. Forced or intended time optimal coordinated gaze movements had eye movement latencies shorter than head movement latencies. The gaze latency diagram (Fig. 3) demonstrated this as an offset or difference between two 45° lines; one 45° line, passing through the origin, was the line of synchronicity; that is, if all eye movements and head movements were synchronous with exactly the same latencies, then all such data points would lie on this line. The other 45° line with offset was a model line.

For 15° rapid head and eye movements, with instructions to subjects to "force" their head movements as rapidly as possible, the elliptical spread of the experimental data points could be fitted by the regression line of

---

FIG. 2. EMG and head trajectory. The 40° horizontal movement; vertical lines indicate initialization of agonistic EMG (AG), position (P), and antagonist EMG (AT) with position trace 40 ms later than AG; changes in acceleration (A) and rate of change of acceleration (Ȧ) from starting line can be seen earlier than with position trace. Note correlation between acceleration (even better Ȧ curve) and EMG envelopes (heavy semidashed lines). Time (T) in milliseconds.

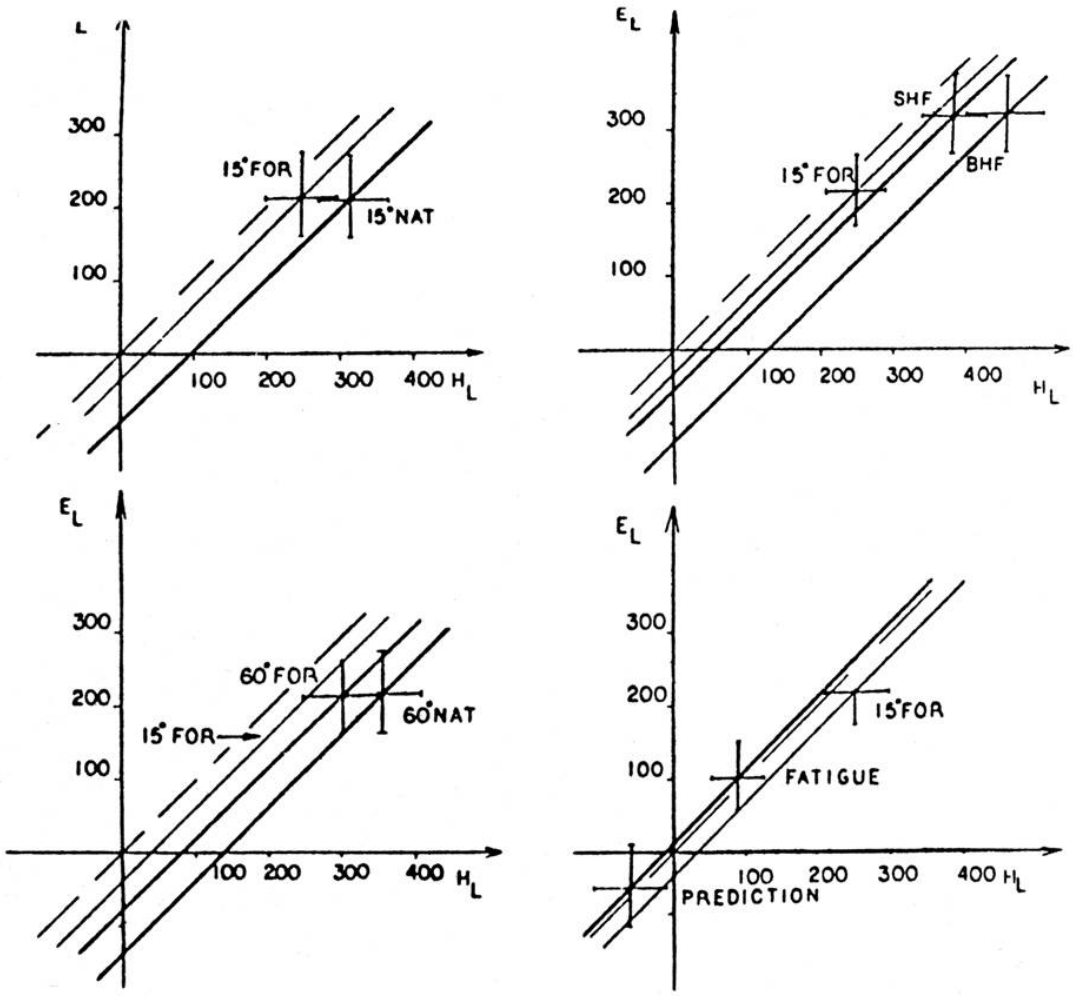


FIG. 4. Gaze latency diagrams for different experimental conditions. Forced (FOR) or natural (NAT) head movement task; predictable targets for (PREDICTION) and also (FATIGUE) experiments; patients gazing toward targets in seeing (SHF) and blind (BHF) hemifields; differing amplitudes (15° and 60°).

only the covarying, approximately 110-ms increase in latency, for both eye and head movement. The center of this second latency distribution (gaze toward SHF) was almost superimposed on the normal, 15° forced line; it has mostly moved in covarying fashion along this 45° line (Fig. 4, upper right).

Predictability and Frequency of Target Jump. As gaze followed a sequence of predictable target shifts from left to right to left visual field, actual eye and head latencies plotted as the sequential number of the response (cycle number, Fig. 5) showed covarying as well as noncovarying phenomena. With a 0.2-Hz stimulus (Fig. 5A), the latency for head movements was generally greater than for eye movements.

As the number of sequential responses increased, both eye and head

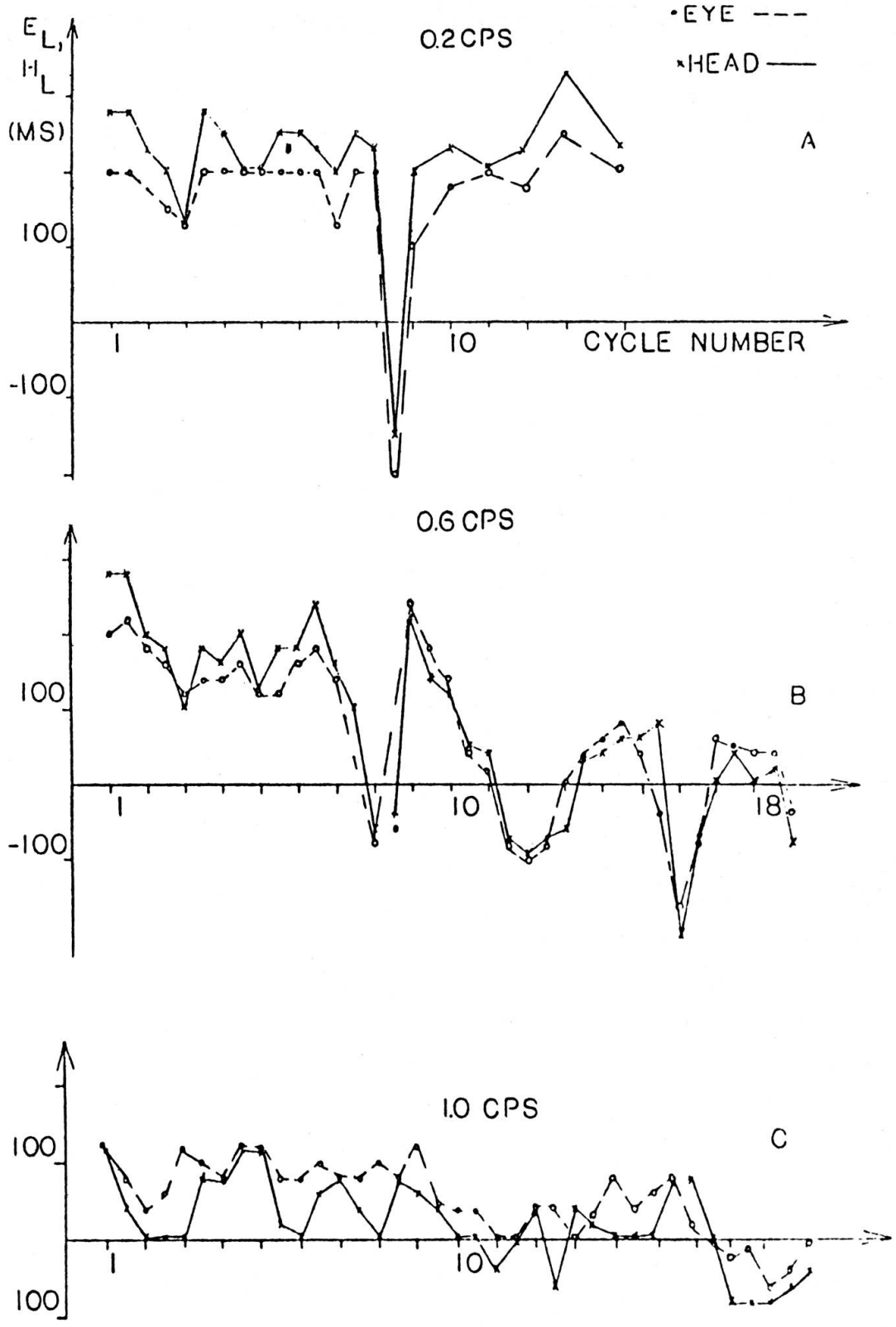


FIG. 5. Sequential eye and head latencies (E_L, H_L) for different target frequencies (CPS—cycles per second).

an additional 90 ms of only head movement latency increase on looking to their blind hemifield, although both head and eye latencies are increased by about 120 ms on looking to the seeing hemifield.

These quite different experimental conditions all demonstrate the more flexible latency of head movements compared with the rather stereotyped eye latency. Thus the neurologic processes that set in motion the head movement component of coordinated gaze are inherently more flexible or are tied to higher-level control mechanisms than those for eye movement. That this is also true for patients with higher level sensory lesions again implicates a neurological process operating at a higher, more flexible, level for head movement than for eye movement.

Flexibility and sensitivity in head movement latency to altered conditions of target predictability can be compared with previously studied changes in latency for arm movement (35) (Fig. 7). With high-frequency predictable target shifts, head and arm movements similarly reduce their latencies significantly more than eye movements in spite of the additional dynamic lags of head and likely also arm movements. Thus predictive processing

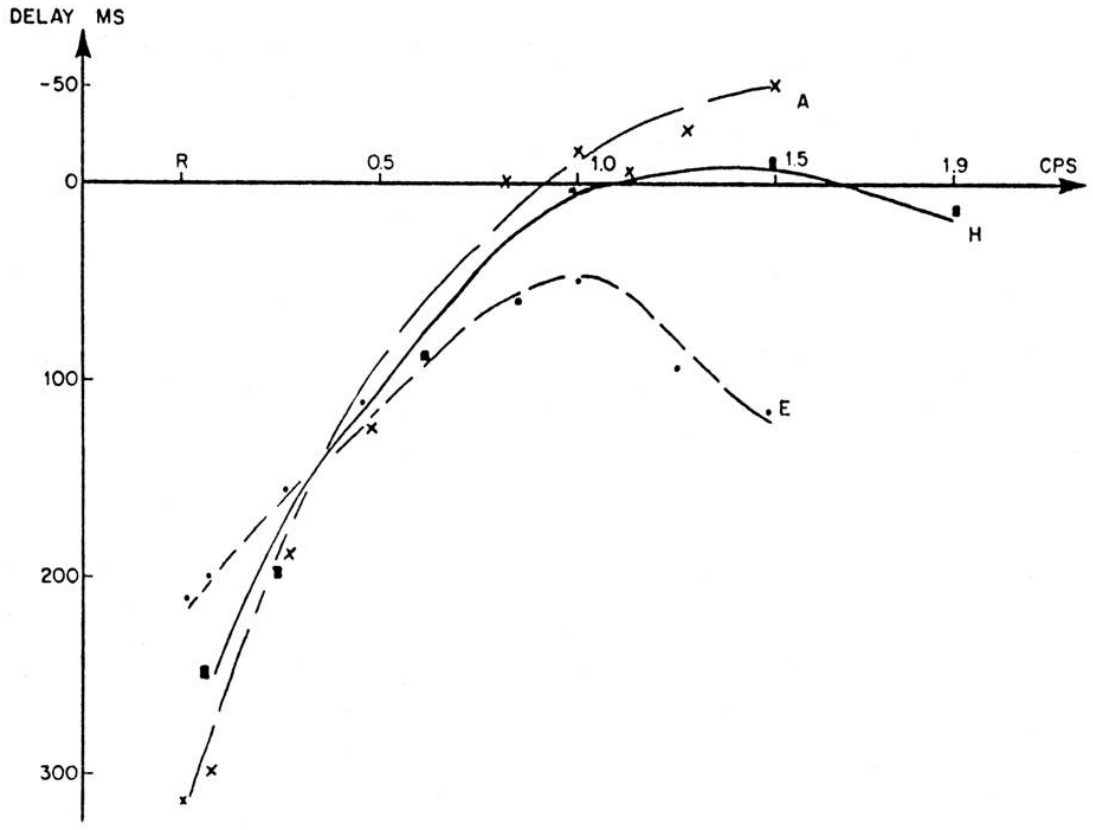


FIG. 7. Eye (E), head (H), and arm (A) movement comparison. Response delays as functions of predictable target frequencies (in cycles per second, CPS) or random target shifts (R); means in milliseconds.

in the central nervous system is similarly more extensive for arm movements.

Similarities in Eye and Head Latencies

Covariability of latencies is well demonstrated in our experimental results: similar mechanisms of conduction time and muscle physiology (Figs. 1, 2); elliptical distribution with high correlation coefficients (Fig. 3); similar changes to predictable targets, with fatigue and to occipital hemianopia (Fig. 4); moment-to-moment changes in sequential tracking (Fig. 5); joint distribution function shifts (Fig. 6); and similar dependencies on low-frequency predictable target shifts (Fig. 7). This suggests shared or similar mechanisms of neurological control at higher and lower levels. In Table 1 hypothetical assignments of subcomponents of these latencies is made and related to what is known in the literature concerning localization of gaze-coordination neurologic function in the central nervous system. The 120 ms of covarying delay, the 115 ms of covarying change with anticipation, and also the 116 ms of noncovarying change with anticipation for head alone are all assigned to a higher-level, in the Jacksonian sense, "cortical" component including peripheral sensory elements, visual cortex,

TABLE 1

Hypothetical Neurological Latency Schema

Site	Head (ms)	Eye (ms)
Delay[a]		
Higher level	120	120
Lower level	80	80
Conduction time and peripheral dynamics	50	15
Total mean latency	250	215
Anticipation[b]		
Higher level	−231	−115
Lower level	−30	0
Total anticipation	−261	−115
Mean latency with anticipation	−11	100

[a] Latencies to random targets as assigned to different neurological levels.

[b] Latency changes with anticipation of predictable targets; note both covarying and independent changes in eye and head latencies.

29. MORASSO, P., E. BIZZI, AND J. DICHGANS. 1973. Adjustment of saccade characteristics during head movements. *Exp. Brain Res.* **16:** 492–500.
30. MOUNTCASTLE, V. 1978. Brain mechanisms for directed attention. *J. Roy. Soc. Med.* **71:** 14–28.
31. OCHS, A., W. F. HOYT, L. STARK, AND M. PATCHMAN. 1978. Saccadic initiation time in multiple sclerosis. *Ann. neurol.* **4:** 578–579.
32. PRABLANC, C., J. ECHALLIER, E. KOMILIS, AND M. JEANNEROD. 1979. Optimal response of eye and hand motor systems pointing at a visual target. *Biol. Cybern.* **35:** 113–124.
33. ROBINSON, D. L., AND M. GOLDBERG. 1977. Visual mechanisms underlying gaze: function of the superior colliculus. In R. BAKER, AND A. BERTHOZ, Eds., *Control of Gaze by Brain Stem Neurons.* Elsevier, Amsterdam.
34. STARK, L., A. SCOTT, AND R. KENYON. 1977. Dynamic overshoot in saccadic eye movements. *Soc. Neurosci. Abstr.* **3:** 157.
35. STARK, L. 1968. *Neurological Control Systems.* Plenum, New York.
36. STARK, L., G. VOSSIUS, AND L. YOUNG. 1962. Predictive control of eye tracking movements. *IRE Trans. Hum. Factors Electron.* **3:** 52–57.
37. SUGIE, N., AND M. WAKAKUWA. 1970. Visual target tracking with active head rotation. *IEEE Trans. SCC* **6:** 103–109.
38. VAN NOORDEN, G. 1961. Reaction time in normal and amblyopic eyes. *Arch. Ophthalmol.* **66:** 694–703.
39. ZANGEMEISTER, W. H., A. JONES, AND L. STARK. 1981. Dynamics of head movement trajectories: main sequence relationship. *Exp. Neurol.* **71,** 76–91.
40. ZANGEMEISTER, W. H., L. STARK, O. MEIENBERG, AND T. WAITE. 1981. Motor control of head movements: electromyographic evidence. *J. Neurol. Sci.*, in press.
41. ZANGEMEISTER, W. H., AND L. STARK. 1980. Types of eye head coordination in gaze movements. In *OMS80 Conference on Eye Movement Control, Proceedings.* California Institute of Technology, Pasadena.
42. ZANGEMEISTER, W. H., O. MEIENBERG, L. STARK, AND W. F. HOYT. 1981. Eye head coordination in homonymous hemianopia. *J. Neurol.* (in press).