© Aeolus Press
Neuro-ophthalmology 0165-8107/89/US$ 3.50 (Accepted 18 July 1989)

Gaze movements: patterns linking latency and VOR gain

W. H. ZANGEMEISTER [1*] and L. STARK[2]

[1]*Department of Neurology, University of Hamburg, FRG;* [2]*Departments of Physiological Optics, Neurology and Bioengineering, University of California, Berkeley, CA 94720, USA*

ABSTRACT. Eye-movements alone can shift gaze. For head movement to contribute to gaze directly, it is necessary to suppress the vestibular ocular reflex (VOR) while rotating the head. The authors describe the connection between VOR gain and the latency defined gaze types especially with respect to amplitude of gaze shift. Eye dominant gaze shift strategies can then be distinguished from head dominant strategies.

Key words: vestibular ocular reflex; eye head coordination (eye head latency); gaze types

INTRODUCTION

Gaze movements are a coordinated sum of eye and head movements. Gaze means eye in space, eye means eye in orbit. Classical gaze studied by Bartz[1] and Bizzi[2] is a synchronous control via synchronous electromyographic signals (EMG) to the muscles of eye and head, with head lagging due to dynamical factors allowing the eyes to saccade first. A wide set of variable latencies between eye and head movement found experimentally led us to a logic of defined gaze types[3–6].

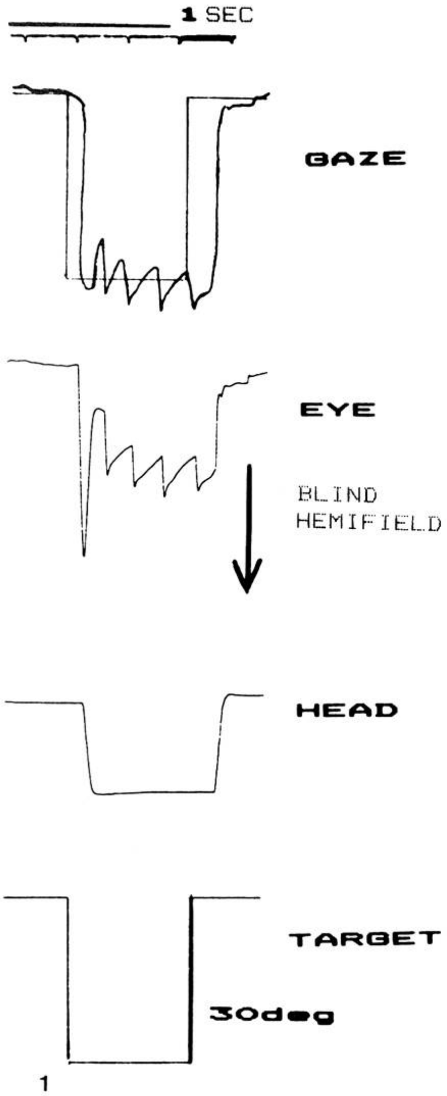
Eye movements alone can shift gaze. Head movements accompanied by the VOR (vestibular ocular reflex) with a high gain of ratio of eye-to-head velocity cannot shift gaze since the eyes in the orbits are carried in an opposite and equal direction to the head movement. Therefore, for head movement to contribute directly to gaze, it is necessary to suppress the VOR gain while rotating the head. Several recent studies have tried to elucidate the influence of adaptation[7] and conditional set[8–10] on coordinated gaze strategies[11] in both animals and humans.

Protocol conditions, such as target brightness, predictability and amplitude of eccentricity of rotation angle strongly influence the latency between eye and head movement and thus the resultant gaze type. Similarly, these protocol conditions shift the amount of VOR suppression accompanying the head movement. A linked pattern of latencies and VOR gains controls the choice between an eye dominant (types I and II) and a head dominant (types III and IV) strategy of gaze movement. These strategies can be interspersed; for example, a shift from an eye dominant to a head dominant gaze can take place in a few seconds (Fig. 1)[12, 13].

While these basic findings have been reported in earlier papers[6, 12, 13], the aim of this paper is to

---

* *Correspondence to:* Prof. Dr. med. W. H. Zangemeister, Neurologische Universitätsklinik und Poliklinik, Martinistraße 52, D-2000 Hamburg 20, BRD



1

Fig. 1. Instantaneous change of CEM gain in a patient with congenital hemianopia. Note high gain CEM (1.5) with gaze to right (down; to blind hemifield) and suppression of CEM, *i.e.* gain of zero with gaze to left (up) only two seconds later.

clarify the connection between VOR gain and latency/gaze type. As expected, in head dominant strategies the head latency is shorter than the eye latency and VOR gain is often suppressed (low) to allow the head movement to shift gaze. In eye dominant strategies, eye latency is shorter than head latency: the eye quickly acquires the target and the later head movement, with a high gain VOR, merely exchanges head position change for earlier eye-in-orbit position change.

METHODS

Head and eye movement recording techniques have been reported in our earlier papers[3, 4, 6, 14]. In order to repeat and specify the inherent connection of head/eye latency type with VOR gain, we compared our earlier data[4, 5] with more recent data[13] using similar, but improved recording techniques and experimental procedures. In this way, we were able to confirm our earlier results with special reference to the scope of this paper.

Eye movements were recorded by monocular DC electro-oculography and head movements by using a horizontal angular accelerometer (Schaevitz) and a high resolution ceramic potentiometer linked to the head of the subject through universal joints that were tightly connected to a head helmet. Control measurements showed that there was no delay between the beginning of the head movement and that of the accelerometer measurement.

The target appeared as a bright continuously lit target of 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 deg, 30 deg, 40 deg, 60 deg) were produced with frequencies between 0.1 and 1.9 Hz. Unpredictable (both in time and amplitude) target shifts were generated with amplitudes between 6 deg and 60 deg; both timing and amplitude selection were done by a microcomputer using a random number generator.

Subjects sat comfortably in front of the screen and forced themselves to perform intended time optimal[15, 16] head and eye movements as fast and as accurately as possible. Seven normal subjects participated in our original studies[4, 5] and 12 normal subjects (mean age 31) participated in our recent study[13]. The 12 underwent a second run where they were asked to perform eye dominant or head dominant gaze types through voluntary control, while viewing predictable targets. These results have been reported elsewhere[13].

For one type of stimulus condition, at least 35 samples were used for standard statistical analyses such as *t* test and linear regression fits[17]. Data were recorded on a rectilinear chart recorder, on FM magnetic tape and fed into the laboratory microcomputer system[18, 19].

RESULTS

1. Gaze types

The considerable difference between the dynamics of head and eye rotation are encompassed by the different parameter values used in the head and eye movement model[6, 15, 16, 20] (Table 1). The inertia of the head is about 10^4 that of the eye while the elasticity of supporting structures, kp, is of the same order of magnitude. These two systems are intimately coupled. That the eyes are mounted in the head means head movements are directly coupled to rotations of eye-in-space or gaze. When it is desired to shift gaze from one target to another, the proposed gaze movement must be allocated in time and space between the head and eye movement systems. Experimentally measured shifts of gaze in which both head and eye position are recorded, can be classified according to the relative timing or latency of eye movements and head movements. This can be further compared with the timing of EMG excitation to the relevant muscles of the head and the eye (Table 2). Based on these timing features, gaze shifts can be classified into types which are useful for describing normal and pathological subjects[5, 18].

TABLE 1. Parameters of the head movement model[15, 17, 28] showing dynamic differences concerning high inertia of the head and relatively low elasticity of the head.
(5)HM complete before EM saccade starts.

J	$g\text{-}s^2/deg$	Inertia	1.8×10^{-1}
B	$g\text{-}s/deg$	Viscosity	2.0
K_p	g/deg	Parallel elasticity	2.0
K_{SL}	g/deg	Series elasticity	40.0
F_{max}	g	Maximum muscle force	600.0 (10) 2000.0 (40)
F_{min}	g	Minimum muscle force	2.0
T_a	s	Activation time constant	5.0×10^{-2}
T_d	s	Deactivation time constant	5.0×10^{-2}

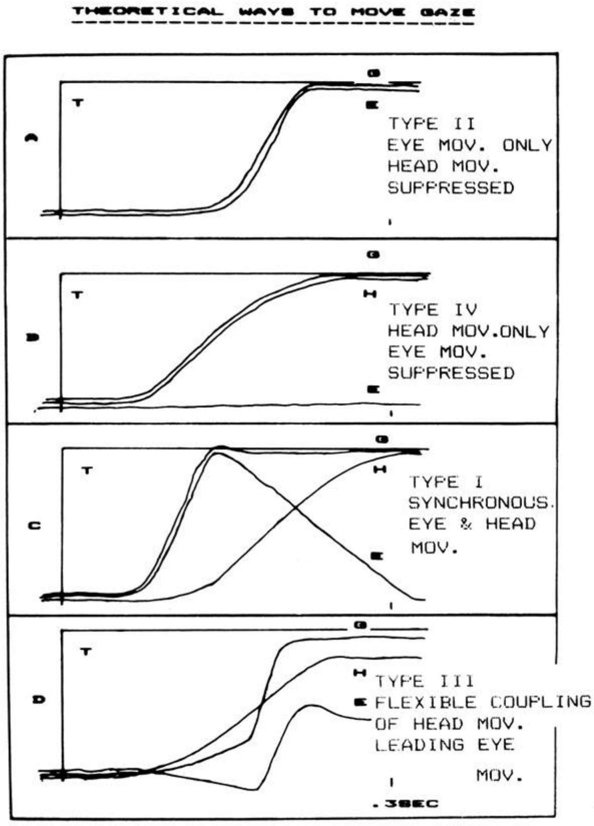
Type I (Fig. 2c; Table 2) resembles a rigid, nonflexible and more reflexive type of gaze shift. As this type shows synchronous and reflex coupling of head and eye, the differing dynamics (Table 1) lead to the characteristic delay of head position to eye position of 50 msec in about 30%.

Type II resembles the head movement suppression type. In about 5%, voluntary suppression or delay of head movement occurs with eye move-

TABLE 2. Gaze types defined by latency(1) differences of eye and head in terms of agonistic EMG and position.

Type	*EMG*	*Position*
I	0(2)	+ 50(3)
II	neg.	< 50(4)
IIIA	0 – 150	50 – 200
IIIB	150 – 500	200 – 550
IV	> 500(5)	> 550(5)

(1)Eye minus head latency (msec).
(2)Synchronous EMG.
(3)due to dynamic lag of head movement.
(4)'Z – zip' = anticipated CEM before reflex VOR.
(5)HM Complete before EM saccade starts.



2

Fig. 2. Theoretical ways to shift gaze. T, G, E, H : target, gaze, eye, head.

ments early and only, due to preset eye movements with high vigilance and forced intent (Fig. 2a), mostly seen with small amplitudes below 15 deg. Pathologically, it is found as a global suppression of head shifts in Parkinson's disease[21] and as a local suppression of head shifts in torticollis spasmodicus[13, 20].

Type II resembles the second mode to move gaze (Fig. 2d; Table 2). With eye movements of regard, schematic eye movements or in general predictive gaze movements, the head leads eye movements more (IIIb) or less (IIIa, *cf.*, Table 2), *i.e.*, this type demonstrates flexible coupling of head and eye. The VOR is suppressed to permit this second mode of gaze shift which is strongly favored by large amplitudes of gaze shifts occurring in about 45%. Pathologically, it is seen in saccadic apraxia[22] and as an early sign in Huntington's disease[19, 23].

In type IV (about 20%), head movement completely governs eye movement. Either the VOR is suppressed almost to zero (Fig. 2b) or, if the initial VOR was not suppressed, a corrective saccade follows. This is the suppressed eye movement type.

2. Protocol and latencies

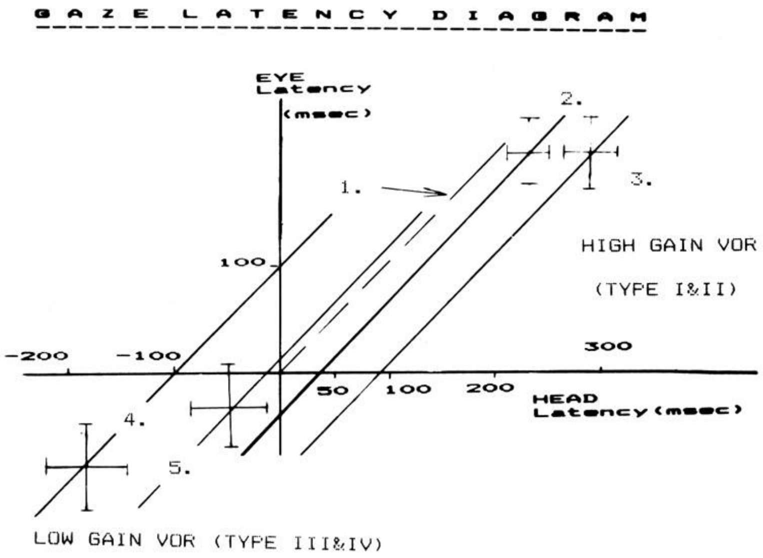
The dominant effect of amplitude on gaze performance is demonstrated in Table 3. It shows the four gaze types found experimentally (columns) and a variety of experimental protocol conditions (rows). For each condition, a ratio is formed to indicate the influence of that condition on the probability of different gaze types. Obviously, the synchronous and more reflexive type I is not very much influenced by any of the listed conditions. On the contrary, large gaze amplitudes favor the occurrence of head dominant types III and IV by a factor of 3, whereas purely eye dominant type II with large amplitudes becomes decreased ten times.

Of course, predictability together with intent play their role: for large target amplitudes, the flexibility of head latency is especially useful (Fig. 3). Predictive gaze shifts permit high VOR suppression and therefore relatively stable large shifts of gaze (Fig. 3, line 4); *i.e.*, without perturbation, VOR gains range between 0.3 and 0.6 with a considerable suppression[13], but with a comparably lower influence of prediction and intent on gaze than in type IV. Type III is therefore most often obtained (43%, *cf.*, Table 3), unless a very particular situation – natural or experimen-

TABLE 3. Protocol condition (rows) influences on frequency of gaze types (columns). For each condition, a ratio was formed (in brackets) to indicate the influence of each condition. * Mean percentage of frequency of occurrence[4].

Type / *Condition*	*I Synchronous eye-neck EMG (34%)**	*II Late head movement (4%)**	*III Early head movement (43%)**	*IV Late eye saccade (19%)**
Amplitude (60/15 deg)	0.90	0.09	3.05	2.10
Intent (forced/natural)	0.73	3.80	2.20	3.50
Predictability (high/low)	0.71	0.85	1.60	2.90

tal – favors the occurrence of other types, *e.g.,* small amplitude (II), reflexive gaze shift (I) or high intent together with high prediction (IV). With random targets, this efficiency of gaze to acquire targets quickly and accurately is highly decreased. Low VOR gain in eye dominant, more reflex-coupled gaze types I and II (Fig. 3, lines 2 and 3) resembles less efficient target acquisition in terms of delay and accuracy. The idealized model of equal eye and head latencies may be obtained mostly with small, predictive amplitudes (Fig. 3, line 5).



3

Fig. 3. Gaze latency diagram. Eye versus head latency in msec. (1) Model line of equal eye and head latency. (2) Responses to 15 deg random targets showing a shift of about 40 msec on head latency axis due to the visco-inertial lag of head movements. (3) Responses to 60 deg random targets, note: high gain VOR in 2 and 3; type I and II occur more often. (4) Responses to 60 deg predictable targets. (5) Responses to 15 deg predictable targets, note: low gain VOR in 4 and 5; type III and IV occur more often, especially with large amplitude of 60 deg: head carries the stabilized eye over a wide amplitude of gaze shift which is adaptive in terms of target acquisition, whereas little VOR suppression would be maladaptive in this case.

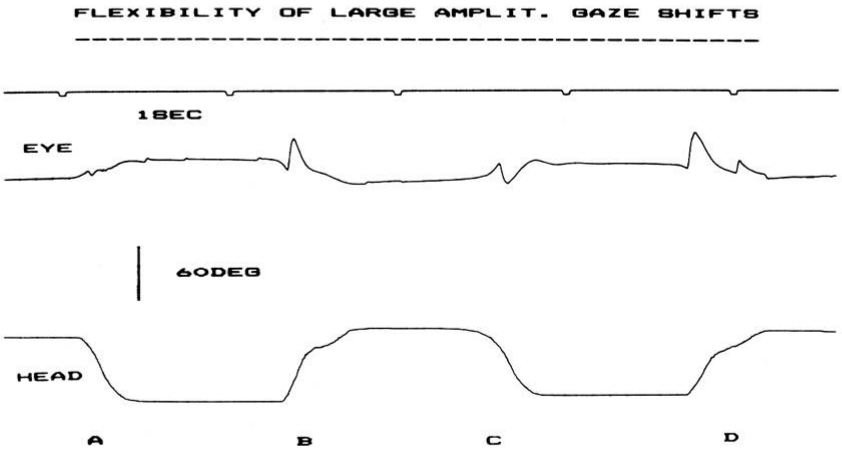
3. Flexible coupling of gaze type III

Original recordings[14] (Fig. 4, Table 4) as well as more recent results[13] confirm the flexibility, especially of gaze type III. Within four successive gaze shifts in response to predictive large 60 deg amplitudes, the latency difference of head minus eye latency changes largely from −200 to about 0 msec (Fig. 4, *cf.*, B and D). The VOR occurring before the saccade demonstrates these latency shifts, which occur only with gaze type III.

Gaze shift ‘A’ resembles type IV with no eye saccade, very low gain VOR (Table 4) and large head movement. Gaze shift ‘D’ resembles type III with larger eye saccade, high normal gain VOR of 1, smaller head movement and gaze falling about 40% short of the target. This is an example of lack of prediction in a predictive task. Examples ‘B’ and ‘C’ demonstrate the flexibility of gaze type III with immediate relative head-eye latency shifts and variable VOR gains. The type IV response (A in Fig. 4) suggests that it is an extreme example of head dominant type III. The quantitative analysis of the different amplitudes and VOR gains suggests that with the head dominant gaze types III and IV, the flexible suppression of the VOR gain changing between 0.14 (A) and 1.0 (D) resembles the interdependence of predictive large amplitudes and voluntary presetting of the VOR.

TABLE 4. Numerical values concerning amplitudes (deg), *cf.*, Fig. 4.

Amplitude of:	*A*	*B*	*C*	*D*
Target	60	60	60	60
Gaze	60	50	60	35
Head	70	75	70	45
Eye saccade	0	20	15	35
Eye VOR	−10	−35	−25	−45
Eye on target	−10	−15	−10	−10
Gain VOR	0.14	0.47	0.36	1.0



4

Fig. 4. Original recordings[5] as well as recent experimental reconfirmations[13] demonstrate for large 60 deg target amplitudes the variant gaze types within four successive gaze shifts. As seen from Table 4, gaze shift A resembles type IV with large head movement, no eye saccade and very low VOR gain. Gaze shift D resembles type I with smaller head movement, large eye saccade and high normal VOR gain of 1 : gaze falls about 40% short of target; this is an example of prediction lack in a predictive task. Examples B and C demonstrate the flexibility of gaze type III with variable VOR gains and shifts of eye head latencies.

4. Linkage between latency and VOR gain: the two strategies – head-dominant and eye-dominant

Frequency ratios indicate (Table 5) the effect of the amplitude protocol condition in shifting the relative occurrence of gaze types when gaze amplitude required changes from 15 to 60 degrees. These overall frequency ratios themselves change when the movement types are sorted into subsets with high (>0.7) or low (<0.3) VOR gains. The table lists these new ratios which change the VOR suppression types in such a way that the subsets with high VOR gains reinforce types I and II, while low VOR gains reinforce types III and IV. This demonstrates that for large amplitudes a useful adaptation takes place so that the head carries the stabilized eye to the target. Without that flexibility of VOR, suppression or a maladaptation would occur in terms of appropriate target acquisition: This is best seen in Fig. 1 which shows an instantaneous change of VOR gain with reversal of gaze movement direction in a patient with congenital homonymous hemianopia[12, 14].

TABLE 5. Gaze amplitude (60/15 deg) changes frequency ratios (FR) of gaze types: Active VOR with type I and II, suppressed VOR with type III and IV. Data from refs. 4, 5, 13.

	Active VOR types		*Suppressed VOR types*	
	I	*II*	*III*	*IV*
Basic (60/15) FR	0.9	0.2	1.4	1.6
FR for subset with VOR gain of >0.7	1.3	0.3	1.1	0.7
FR for subset with VOR gain of <0.3	0.1	0.01	1.7	2.9
Relative change of FR as function of VOR gain	13	30	0.6	0.2

DISCUSSION

Strategies

There are two ideally extreme modes to move gaze. Mode I uses an eye movement without a head movement. Mode 2 uses a head movement with VOR gain suppressed, *i.e.*, without an eye movement. These extremes define the two basic strategies of gaze movements: the eye dominant (type I and II) and the head dominant (type III and IV) strategy.

Linked selection of a pattern of gaze latencies and of VOR gains enables a subject to deploy one of the two general strategies. This infers a necessary connection between the latency and the VOR gain that occurs. The reasons for this are the requirement for low VOR gain to obtain gaze from head movement, and the complementary requirement for delayed head movement allowing an eye saccade unrestricted by head movement and its VOR when obtaining gaze from eye saccades.

Eye-dominant strategy

The description of the resultant behavior of gaze movement depends in the first instance upon eye movement in the classical type I. Here, the dynamic lag of head in spite of synchronous EMG control signals to eye and head, enables the eye saccade to be completed before the head moves. In type II, the head movement is delayed and the eye is further permitted to carry out the gaze movement. The much later head movement has a role, but not a basic contribution to gaze. It merely exchanges final head position for eye position, leaving head pointed at target with target in straight-ahead direction ego-centrically and eye back in primary position in orbit. The VOR gain may be quite close to values of 1 in gaze types I and II.

Head-dominant strategy

The description of the resultant behavior of this gaze movement depends largely upon head movement. This head movement is predicted and has decreased relative latency with respect to the eye movement. The VOR is suppressed; otherwise, the head will have no effect on gaze. The later occurring eye movement (if it occurs) merely adds to head movement to achieve the final, often large gaze amplitude.

Causal factors of eye dominant strategies

Experimental protocol conditions prescribe that in particular small amplitudes favor eye dominant strategies. Due to preset eye movements with high vigilance and forced intent (*cf.*, Table 3) eye movements are generated much earlier than the comparatively delayed head movements which may be completely suppressed. Kennard *et al.*[21] have shown that in Parkinson's disease, the eye-dominant gaze shift strategy is very much favored because eye saccades are involved in the disease process to a much lesser extent. This has also been shown by Bloxham *et al.*[24] with regard to latency of eye and arm/finger movements and by Deecke *et al.*[25] concerning the correlation of the readiness potential and the movement itself. Besides this globally elicited suppression of head movements, a more locally caused suppression may occur in patients suffering from torticollis spasmodicus as reported by Zangemeister and Stark[20, 26]. In this case, asymmetrical head positioning due to asymmetrical tonic neck muscle innervation produces an eye-dominant strategy that mirrors this asymmetry for head movements to the direction contralateral to the side of the torticollis. While these pathological examples resemble abnormal motor conditions, a purely sensory cause of asymmetrical suppression of head movements with gaze shifts is demonstrated in homonymous hemianopia. This is exemplified in particular in congenital homonymous hemianopia (Fig. 1), as was earlier reported by the present authors[12, 14, 18].

Causal factors of head dominant strategies

Large gaze amplitude is the most important protocol condition that prescribes head dominant strategies. It also favors flexibility of gaze control as demonstrated in Fig. 3B and C. Suppression of eye movement, *i.e.*, of the VOR gain, is the obvious consequence in most instances of large gaze shifts. Causally, prediction and intent are highly correlated with the generation of large gaze angles through a head dominant strategy. It is seen in the predictive situation of watching a tennis game with the players staying near the ground line most of the time. This strategy has been shown to be correlated with medium latency vestibularly evoked potentials by Zangemeister *et al.*[26], using suppression of VOR eye movements through fixation. As this mechanism relies on intact vestibulocerebellar functions[27], head dominant strategies are often suppressed in cerebellar patients[18].

The advantage of the head dominant strategy becomes especially obvious in patients with Huntington's disease who have well-functioning VOR's. They tend to acquire most targets using a head dominant strategy with variable suppression of the VOR. This has been noted by Starr[23] and Zangemeister and Mueller-Jensen[19], whereas others did not report this important early sign of Huntington's disease[28].

In another pathological example, the head dominant strategies are caused by the inability to perform voluntary saccades as seen in ocular motor apraxia[22]. In this case, the VOR gain appears to be variably suppressed, depending upon the fact that the deficit has been acquired (low suppression) or is congenital (high suppression)[18, 22, 29]. Here, the head dominant strategy is best described by the negative value of head latency minus eye latency, *i.e.*, start of head rotation is needed for the initiation of an eye saccade[22-28]. Performance of very large gaze amplitudes in conjunction with high prediction and intent, causes maximal suppression of the VOR (Fig. 3A), which is true for repetitive gaze shifts together with high concentration: only sometimes true while watching tennis matches, but mostly true in patients with congenital ocular motor apraxia.

CONCLUSION

In conclusion, it was demonstrated that several causal factors (protocol conditions such as desired gaze amplitude and pathological situations such as reduced inclination to move the head in hemianopia) influence the selection of a gaze mode even though the selection is a statistical one and the strategy employed may not be an extreme example. It was shown that the connection between gaze type defined by relative latency of eye and head movement and the VOR gain and gaze amplitude is a strong one. Further, this demonstrated connection makes sense in the functional control of gaze. Evidently, some motor brain mechanism (possibly via the cortico-cerebellar loop) organizes a relative latency between head and eye movement and in the same manner sets the VOR gain appropriately.

REFERENCES

1. Bartz J: Eye and head movements in peripheral vision: nature of compensatory eye movements. Science 114:1644-1645, 1965
2. Bizzi E, Kalil R, Tagliasco V: Eye-head coordination in monkeys. Science 173:452-454, 1971
3. Zangemeister WH, Jones A, Stark L: Dynamics of head movement trajectories: main sequence relationship. Exp Neurol 71:76-91, 1981
4. Zangemeister WH, Stark L: Gaze latency: variable interactions of eye and head movements in gaze. Exp Neurol 75:389-406, 1982a
5. Zangemeister WH, Stark L: Gaze types: interaction of eye and head movements in gaze. Exp Neurol 77:563-577, 1982b
6. Zangemeister WH, Stark L, Meienberg O, Waite T: Motor control of head movements: electromyographic evidence. J Neurol Sci 55:1-14, 1982
7. Berthoz A, Melvill Jones G: *Adaptive Mechanisms in Gaze Control*, pp. 177, Amsterdam-New York, 1985
8. Fuller J, Moldonado H, Schlag J: Vestibular-oculomotor interaction in cat eye-head movements. Exp Brain Res 271:241-250, 1983
9. Guitton D, Douglas R, Volle M: Eye-head coordination in cats. J Neurophysiol 52:1030-1050, 1984
10. Morasso P, Bizzi E, Dichgans J: Adjustment of saccade characteristics during head movements. Exp Brain Res 16:492-500, 1973
11. Roucoux A, Crommelinck M, Guerit JM, Meulders M: Two modes of eye head coordination and the role of the VOR in these strategies. In: Fuchs A, Becker W (eds): *Functional Basis of Ocular Motility Disorders*, pp 309-315. Amsterdam: Elsevier 1981
12. Zangemeister WH, Stark L: Active head rotation and eye-head movement coordination. Ann NY Acad Sci 374:540-559, 1981
13. Zangemeister WH, Schlueter D, Kunze K: Compensatory eye movement gain and head-eye latencies change with verbal feedback: voluntary adjustment of gaze types. Oto-Rhino-Laryngol 41:227-234, 1988
14. Meienberg O, Zangemeister WH, Rosenberg M, Hoyt WF, Stark L: Saccadic eye movement strategies in patients with homonymous hemianopia. Ann Neurol 9:537-544, 1981
15. Zangemeister WH, Lehman S, Stark L: Simulation of head movement trajectories: model and fit to main sequence. Biol Cybern 41:19-32, 1981

16. Stark L, Zangemeister WH, Hannaford B: Head movement models, optimal control theory and clinical application. In: Peterson B, Richmond F (eds): *Control of Head Movement*, p 361. Oxford Press 1988
17. Stark L: *Neurological Control Systems*. New York: Plenum Press 1968
18. Zangemeister WH, Stark L: Pathological types of eye and head gaze-coordination in neurological disorders. Neuro-Ophthalmology (Amsterdam) 3:259-276, 1983
19. Zangemeister WH, Mueller-Jensen A: The coordination of gaze movements in Huntington's disease. Neuro-Ophthalmology (Amsterdam) 5:193-206, 1985
20. Zangemeister WH, Stark L: Understanding dynamics and control of normal and abnormal head movements by use of a parameterized head movement model. IEEE Biomed Soc Vol 82CH12729-3:468-473, 1982c
21. Kennard C, Zangemeister WH, Mellers S, Hoyt WF, Stark L: Eye-head coordination in patients with Parkinson's disease. In: Lennerstrand G, Zee D, Keller E (eds): *Functional Basis of Ocular Motility Disorders*, pp 517-521, Oxford-New York: Pergamon Press 1982
22. Zee D, Yee R, Singer H: Congenital ocular motor apraxia. Brain 100:581-598, 1977
23. Starr A: A disorder of rapid eye movements in Huntington's chorea. Brain 90:545-556, 1967
24. Bloxham C, Mindel T, Frith C: Initiation and execution of predictable and unpredictable movements in Parkinson's disease. Brain 107:371-384, 1984
25. Deecke L, Englitz H, Kornhuber HH, Schmitt G: Cerebral potentials preceding voluntary movement in patients with bilateral or unilateral Parkinson akinesia. In: Desmedt JE (ed): Attention, voluntary contraction and event-related cerebral potentials. *Progress in Clinical Neurophysiology*, Vol. 1, pp. 151-163. Basel: Karger 1977
26. Zangemeister WH, Phlebs U, Huefner G, Kunze K: Active head rotations and correlated EEG potentials: experimental and clinical aspects. Acta Otolaryngol (Stockh) 101:403-415, 1986
27. Baloh RW, Yee RD, Kimm J, Honrubia V: Vestibular-ocular reflex in patients with lesions involving the vestibulocerebellum. Exp Neurol 72: 141-152, 1981
28. Leigh J, Zee D: *The Neurology of Eye Movements*, pp 109. Philadelphia: Davis 1983
29. Stark L, Zangemeister WH, Hannaford B, Kunze K: Use of models of brainstem reflexes for clinical research. In: Kunze K, Zangemeister WH, Arlt A (eds): *Clinical Problems of Brainstem-Disorders*, pp 172-184. Stuttgart-New York: Thieme 1986