

Pathological types of eye and head gaze-coordination in neurological disorders

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ABSTRACT. The patterns of coordinated eye-head movements in gaze shift are influenced by preconditions of the experimental protocol. Gaze shift movements fall into four distinct types with respect to eye-head latency, each one with a particular frequency of occurrence.

Patients with neurological diseases show characteristic deviations of their gaze type from normal. Their eye-head latencies are affected differently on variant levels of the CNS depending upon the type of disorders/disturbances of the motor or sensory systems only, or combined ones. Various modes of adaptation, or 'maladaptation', are demonstrated and analyzed in these patients.

Key words: eye-head coordination; eye-head latency; gaze types; gaze plane analysis; vestibular ocular reflex (VOR); active head movements; neurological disorders

INTRODUCTION

Under normal circumstances head movements play an important role in alterations of coordinated gaze or gaze shifts. A gaze shift is a movement of the fovea in space as a result of coordinated head and eye movements responding synkinetically to a target

jump. Although the neurological control signals – represented by the envelope of the electromyograms (EMG) – arrive simultaneously, the eye saccade is begun and completed before the head makes any significant movement (Bartz, 1965; Bizzi *et al.*, 1971; Zangemeister & Stark, 1980, 1981, 1982). Eye rotation is much faster because the eyeball requires only a simple visco-elastic effort of the very quick extraocular muscles; head rotation lags because the head provides a large visco-inertial load for the comparatively slow moving skeletal neck muscles (Clark & Stark, 1975; Zangemeister *et al.*, 1981a, b, 1982a, c).

While the head is continuing to rotate through its longer trajectory, after the eye saccade has found the target, the vestibular

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ocular reflex (VOR) and other components of compensatory eye movement (CEM) rotate the eye at an equal rate in the opposite direction. Thus, the eye is able to remain fixed on the target, while head rotation is substituted for orbital eye rotation, the eye reaching primary gaze position at the end of the coordinated movement. The basic patterns and the frequency of four gaze-shift types under variant conditions have been experimentally classified for normal human subjects (Zangemeister & Stark, 1980a, b, 1982a, b). The motor coordination features of the gaze-shift types have been abstracted for further analysis in the gaze plane.

Neurological disorders that may affect sensory or motor functions (e.g., homonymous hemianopia, Parkinson's disease), or both (e.g., multiple sclerosis), lead to changes in the coordination of eye and head gaze-shift types. This report describes the patterns and modifications of gaze-shift types in various neurological disorders. The description is aided by comparing and contrasting the pathological gaze-shift types to the conditions and frequency of normal gaze-shift types.

METHODS

Recording of eye and head movements. The techniques used for recording eye and head movement were reported (Zangemeister *et al.*, 1981). In brief, mechanical linkages and potentiometers were attached to a bicycle helmet worn by the subjects while viewing the targets. Eye movements were recorded either by the infrared limbus-reflection techniques (Stark *et al.*, 1962) or, for large amplitudes, with monocular electro-oculogram (EOG) using Beckman miniature

electrodes. EMG measurements were made with surface electrodes situated over pairs of neck muscles (such as the splenius and sternocleidomastoideus) that are involved in horizontal rotation of the head (Zangemeister & Stark, 1980 a, b; Zangemeister *et al.*, 1982c). The target appeared as a bright, continuously lighted spot of 30 arc min that was projected onto a dark screen.

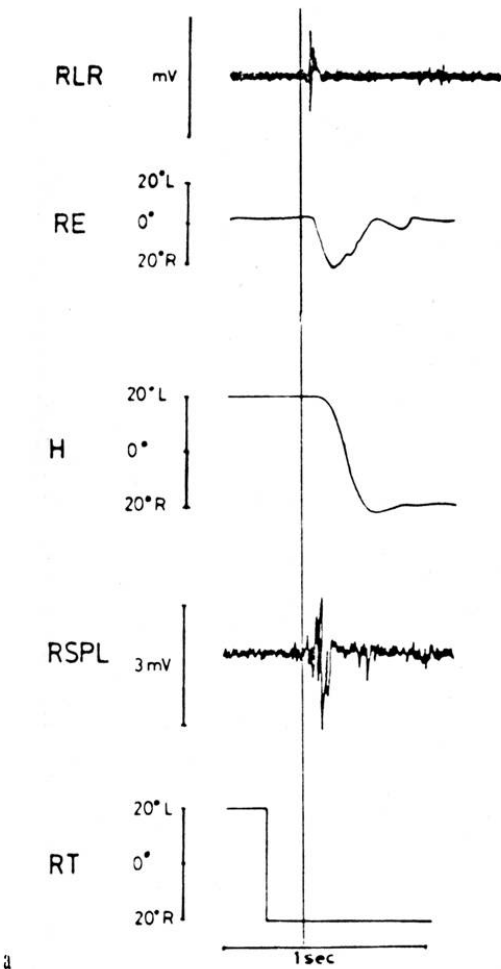
Programs. Stimulus programs included both predictable and unpredictable sequences of target steps. Predictable target jumps with amplitudes of 15°, 30°, 40° and 60° shifted with frequencies varying between 0.1 and 1.9 Hz. Unpredictable target shifts varied randomly in amplitude between 6° and 60° and in frequency between 0.1 and 1.9 Hz. Random amplitudes and frequencies were generated by a digital computer using a random number generator.

Subjects were seated comfortably in front of the screen and, for the first run, were advised to engage in 'natural' head and eye movements. For the second run they were instructed to force themselves to perform head and eye movements as rapidly and accurately as possible; these were intended time optimal movements (Clark & Stark, 1975; Zangemeister & Stark, 1980a, b, 1981a). Recordings were made of these movements performed by seven normal subjects, and neurological patients with: homonymous hemianopia (6), Parkinson's disease (7), cerebellar diseases (10), unilateral or bilateral labyrinthine defect (3), ocular motor-apraxia (1), progressive supranuclear palsy (2), spinocerebellar ataxia (5).

Analysis of data. At least 35 movements at each stimulus condition were obtained for analysis. Data were recorded on a rec-

tilinear chart recorder and on a tape recorder, from which they could be fed into the PDP-8 laboratory minicomputer system. Data were analyzed by standard statistical techniques, including Student's *t*-test and linear regression analysis (Cochran & Cox, 1957).

Fig. 1a. Response to 40° target shift from left (L) to right (R). Random (RT) target shift. Eye movement precedes head movement (45 msec), whereas right splenius EMG (R SPL), starts about 10 msec before initial eye position changes; (RE) right eye, (RLR) right lateral rectus. ↓

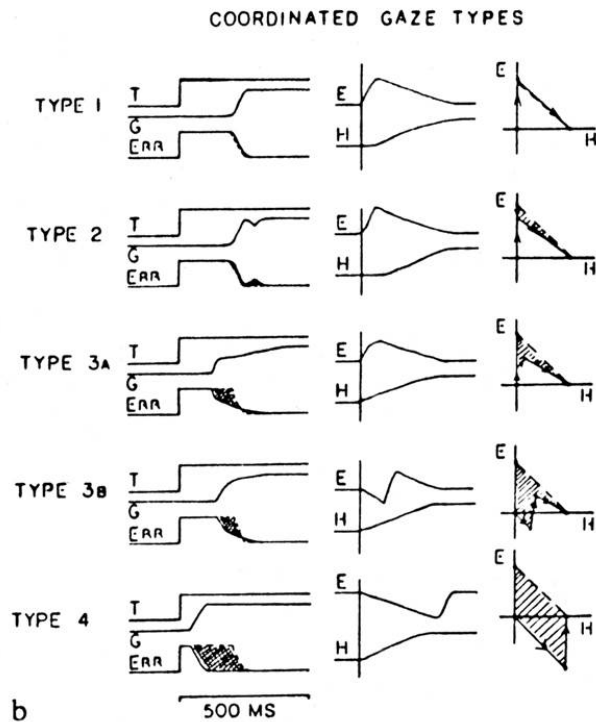


RESULTS

(I) Normal gaze shift types

The classical coordinated gaze movement may be described as a saccadic eye movement followed by a head movement, in turn that is synchronously accompanied by the compensatory eye movement (CEM) that returns the eye back to its primary position in orbit, thus exchanging head movement for eye movement. This description of a coordinated gaze movement has head movement occurring with a longer latency

Fig. 1b. Left: error displays, cross hatched lines excess error (Err) with respect to gaze (G) and target (T) position. Middle: Eye (E) and head (H) position, vertical line indicating the relative eye and head latency. Right: gaze plane display with time as implicit function showing excess error (dashed lines) and eye head interaction for the gaze types in normal subjects. ↓



than eye movement; however, the head being a larger mechanical object than the eyeball requires an increased dynamical lag period in order to move, in spite of a synchronous control signal. Neck EMG occurred synchronously with eye movement (Fig. 1a), since the eye movement is almost synchronous with its own EMG (Bizzi *et al.*, 1971; Henn & Cohen, 1973; Zangemeister *et al.*, 1980a, b, 1982a, c). A large amplitude head movement has agonistic neck muscle EMG turning on approximately 45 msec before the head movement occurs (Fig. 1a). Latency studies demonstrated the causal chain from target input to agonistic electromyogram (200 msec) to head acceleration (20 msec) and initial change in head position (45 msec), to final head position (240 msec) for a 20 degree head rotation. Antagonistic EMG, obeying reciprocal innervation, starts about 70 msec after agonistic EMG began.

Comparison of mean latencies of eye movements (with fixed head), with head and arm movements, demonstrated the better prediction in head movements, such that the increased dynamical lag of the head movement could be overcome by the better prediction operator of the head (Zangemeister & Stark, 1980a, 1982a). This prediction is of importance when understanding the different gaze types.

Gaze types and gaze plane

Errors, or differences between gaze angle and target angle are worthwhile studying, because both eye and head movements are visual feedback error actuated movement patterns. The error function monitored continually by the subject as the important variable to be minimized is the off-fovea eccentricity of the target. An additional aid

in understanding the gaze error that occurs with respect to certain gaze types is the 'gaze plane'. In the gaze plane plot, time is only an implicit function of eye and head positions (Fig. 1b) that are displayed as functions of one another. Arrows show movements of only the eye (vertical arrow) or the coordinated eye-head-movement (diagonal arrow, CEM).

Type I. When the EMG indicated that the neural control signals of eye and neck muscles were synchronous, the gaze shift pattern (Type I) consisted of a rapid eye saccade that attained the new gaze position before the head moved significantly (Fig. 1b), and then held the position while head movement was substituted for eye movement.

Type I gaze shifts showed considerable variability. This depended on varying CEM velocities and on varying acceleration of head movements in response to controller signal variables. Over all, type I occurred 35% of the time in our subjects. It has also been found to occur frequently in studies of monkeys (Bizzi & Schiller, 1970; Bizzi *et al.*, 1971). In our study, its frequency relative to other gaze shift types (Table 1) was affected by various conditions governed by the experimental protocol: type I occurred more frequently when target brightness was high or when the subjects' vigilance was low; its occurrence was comparatively unaffected by changes in amplitude or predictability of target presentation, or the subject's endeavour to be time-optimal.

Type II. Delay in the head movement component produced an interval following the completion of the eye saccade when either the gaze remained stationary or the initial component of the CEM occurred before the head movement began, producing

an anticipatory CEM (Fig. 1b, second row). This anticipatory CEM (ACEM) occurred more frequently and was increased in amplitude and velocity when the amplitude of the gaze shift was small and the endeavour to reach the target was high (forced intent). The velocity of the head movement generated as a result of the CEM (mostly vestibulo-ocular reflex) often equalled the initial anticipatory CEM. The overall frequency of type II gaze shifts was relatively low.

Type III. An early head movement resulted in superimposition of the saccadic eye movement upon the ongoing head trajectory with its concurrent VOR.

This interaction resulted in a slowed velocity of the eye saccade. Type III occurred 33% of the time in normal subjects. Its occurrence was more frequent in gaze shifts of large amplitude (Table 1), where the

head movement assumed increased importance because eye saccades were normally 15 degrees or less in amplitude. Other protocol conditions conducive to the occurrence of gaze type III were readily predictable target jumps and efforts by the subject to force rapid target acquisition. Type IIIa was distinguished from type IIIb: the former resulted in a truncation of the moderately late eye saccade, whereas the latter displayed a very late eye saccade that interacted with the, by then, well advanced CEM (Fig. 1b, third and fourth rows).

Type IV. Sometimes an eye saccade began so late that the head movement was already completed, or nearly so (Fig. 1b, fifth row). In this type of gaze shift, the VOR ongoing concurrently with the head trajectory was also completed before the eye saccade began, allowing scant opportunity for synchronous eye-head saccadic

TABLE 1. Influences on frequency of gaze types. The four coordinated gaze type (columns) and the three most influencing experimental protocol conditions (rows). For each condition a ratio is formed (length of arrows) to indicate the influence of that condition on the probability of different gaze types. Mean percentage of frequency of occurrence in parentheses.

Gaze type	I	II	III	IV
	<i>Synchronous eye neck EMG</i>	<i>Late head movement</i>	<i>Early head movement</i>	<i>Late eye saccade and early head movement</i>
Condition	(34%)	(4%)	(43%)	(19%)
Amplitude: 60°/15°	↓ -0.10	↓ -0.91	↑ +2.05	↑ +1.10
Intent: forced/natural	↓ -0.27	↑ +2.80	↑ +1.20	↑ +2.50
Predictability: high/low	↓ -0.29	↓ -0.15	↑ +0.60	↑ +1.90

coordination. In these type IV gaze shifts, eye saccades were variable and sometimes so delayed that the gaze remained off target for a comparatively long time. This resulted in head movements that preceded the gaze shifts. Thus, the early head movement did not afford early acquisition of the target in this gaze shift type as it did in types I and II. Conditions conducive to type IV gaze shifts were target movements at large amplitudes, endeavour of the subject to shift gaze as rapidly as possible, and low target brightness (Table 1).

Gaze plane. When the gaze shift types are represented in the gaze plane (Fig. 1b, third column) their motor coordination features can be abstracted for analysis. Because the error in type I gaze shift was due solely to eye latency, there was normally no excess error (excess error is defined as a greater error than occurs in type I, with CEM gain of 1). Variations in head acceleration may, however, result in excess error due to too much or too little CEM gain or to quick phase-like saccades. The delayed head movement at early target acquisition, characteristic of type II, typically resulted in a CEM that did not compensate for the VOR, but rather moved the gaze off target. Excess error showed up after acquisition of the target. With slow head velocities there were often low gain CEMs that made corrective saccades unnecessary. The early head movement in type III was not actually due to delay of the eye saccade, but to early movement of the head, which was more flexible in response to prediction of the target location. Thus, with predictable targets the excess error in gaze shifts of type III (Fig. 1b, left column) was often diminished. When the target location was correctly predicted with sufficient prompt-

ness, the excess error may actually be negative, as in types IIIa and b, and IV (Fig. 1b, left column). The minimization of error achieved by accurate prediction of the target location was not represented in the gaze plane, as time was not explicitly depicted. Instead, the cross hatched area in the gaze plane represented the potential extent of excess error, if the target location was not correctly predicted. Head movement often occurred so early, in relation to the initiation of the saccadic eye movements, that resultant dynamic interactions became significant. Type IIIa represented a saccade that occurred early in the head trajectory and was slowed down and truncated by the VOR. When the eye saccade occurred later in the head trajectory, as in type IIIb, it may be even further slowed and truncated as a result of interaction with the peak head acceleration and maximum VOR activity. Truncation of the saccade in such cases may be due to interactions of neuromuscular forces and peripheral biomechanical actions as the opposite direction of the VOR reduces the force of the muscles generating the saccade (Ono *et al.*, 1978; Kenyon *et al.*, 1980; Zangemeister *et al.*, 1980a; Nam *et al.*, 1981).

Alternatively, the truncation may be due to control signals of the central nervous system interacting at the level of the brain stem (Morasso *et al.*, 1973; Jürgens *et al.*, 1981). Truncation was often compensated for by diminished CEM gain which may, in turn, be due either to altered control systems from the central nervous system or else to the biomechanical effects of a saccadic movement on the ongoing VOR.

The extreme delay of the eye saccade in type IV could result in very extensive excess error, if the movement of the target was

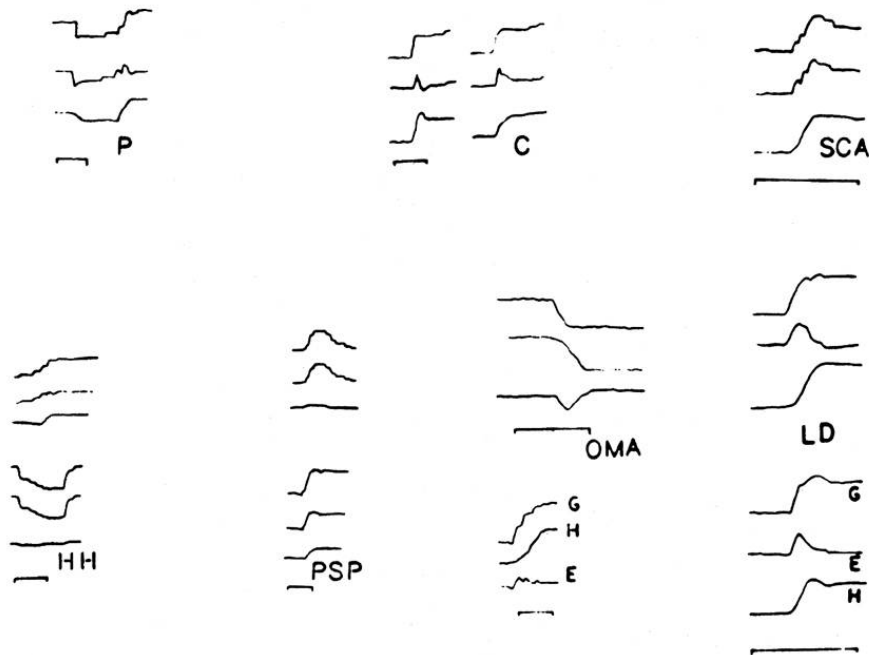


Fig. 2. Recordings of coordinated eye-head gaze shift responses in various groups of neurological patients (abbreviations correspond to Table 2). Note that for each disturbance the most characteristic gaze shift behaviour has been displayed. Calibration marks: 1 sec.

incorrectly predicted. This excess error showed up both in the gaze plane and in the time function depicted in the gaze trajectory. The gaze plane represented the extent of excess error that was possible if the target location was incorrectly predicted. Negative excess error occurs when the correct target location was predicted almost immediately and the head movement consequently occurs so early that the eye saccade, though very delayed, still occurred with less latency than the eye saccade in type I.

The differentiation of gaze shift types and their variants was mainly determined by the great flexibility of head movements that resulted from the influences of several levels of control of the central nervous system. Prediction and intention derive from the

higher levels because head movement latency is much more variable than eye movement latency. The lower levels are responsible for the variation in dynamic trajectories and the interaction of the VOR and eye saccades, that was governed in part by the highly variable head acceleration (Zangemeister & Stark, 1982b).

(II) Pathological gaze shift types in neurological disorders

In neurological patients variants of the normal gaze shift types most often occurred. *Parkinsonian* patients showing hypometria kept the target on fovea once they had slowly acquired it (Fig. 2). Additionally, parkinsonian patients tended to move their head much later than healthy subjects in response to visual targets. Gaze type II with its late head movement and anticipatory compensatory eye movement (ACEM) was therefore favoured. When the parkinsonian patients were asked to follow predictable and unpredictable target shifts using

natural head and eye movements, they either made no head movement at all, or a very small amplitude movement, that amounted to less than 25% of the full movement. In the forced condition, parkinsonian patients made adequate head movements to similar target shifts. An analysis of the frequency of the different gaze types is shown in Table 2. Forty-nine percent of the gaze movements showed the normal coordinated eye-head movement of type I with an eye saccade followed by a head rotation commencing immediately at the end of the saccade. Type II gaze movements were far commoner in parkinsonian patients than in normal controls. In this type the head movement was delayed, a pause occurring between the end of the saccade

and commencement of a head movement; often an anticipatory CEM occurred just before the head moved. There was also a marked reduction of both type III and IV gaze shift movements in the parkinsonian patients reflecting the unusual occurrence of a head movement before the commencement of the eye saccade. These results were further reinforced when the latency of both the eye and head movements were analyzed. The responses to unpredictable targets resulted in an eye and head latency of 228 ± 84 msec and 377 ± 102 msec ($n = 47$), respectively (Kennard *et al.*, 1982).

This was a significant (t -test, $P < 0.001$) delay of the mean head latency by some 150 msec, which was about three times the normal delay (45 msec). With forced eye-head movements the head movement was often slower than normal velocity and the velocity trace showed a 'fragmented', low maximum velocity curve.

Cerebellar patients with hypermetria

Fig. 3. Time functions (left) and gaze planes (right) of a normal gaze type I and of neurological patients. Up is left; G, E, H, T is gaze, eye, head, target position. \odot E, \odot H: eye and head position.

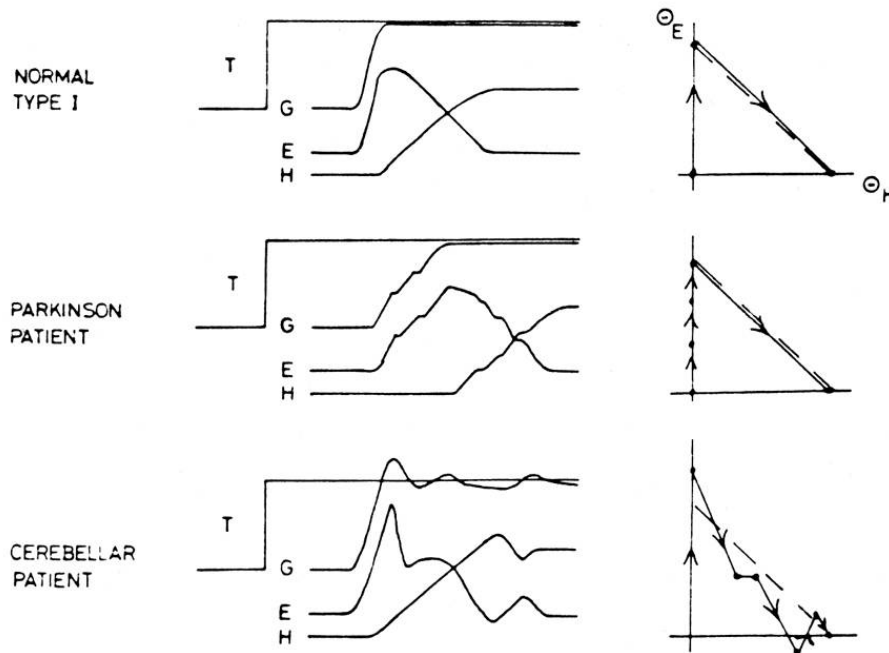


TABLE 2. Mean percentage of frequency of occurrence of the four gaze types in normal subjects as compared to seven groups of patients with different neurological diseases.

<i>Gaze type</i>	<i>I</i> <i>Synchronous</i> <i>eye neck EMG</i>	<i>II</i> <i>Late head</i> <i>movement</i>	<i>III</i> <i>Early head</i> <i>movement</i>	<i>IV</i> <i>Late eye</i> <i>saccade and</i> <i>early head</i> <i>movement</i>
<i>Normal</i>	34%	4%	43%	19%
Parkinson	49%	33%	18%	0%
Cerebellar	44%	2%	46%	8%
Homonymous hemianopia	52%	25%	21%	2%
Ocular-motor apraxia	0%	0%	77%	33%
Spinocerebellar atrophy	11%	0%	58%	23%
Progressive supranuclear palsy	5%	10%	70%	15%
Labyrinthine defective	52%	0%	46%	2%

demonstrated a long lasting difficulty in catching a random target and getting the gaze finally adjusted to it. When prediction was permitted, cerebellar patients showed significantly less dysmetria, that is a less overshooting gaze movement. Obviously some of the dysmetria could be overcome by the better head movement predictor. The main sequence of eye movements of cerebellar patients did not show a significant difference to normal values. However, the more intentionally governed head movements showed a high variability of their dynamical behaviour. Therefore as soon as the movement was unpredicted and intended to be fast, the increased dynamical variability led to more variable eye move-

ment patterns: to compensatory eye movements of relatively too high gain, because of a diminished cerebellar suppression of the VOR, and to dysmetric corrective saccades (Fig. 3). Cerebellar patients showed more frequently gaze type I and III (Table 2). The frequency of occurrence of gaze type II and IV was greatly diminished. The fact that movements showed a high amount of prediction as the eye in gaze type II (ACEM) or the head in type IV only seldom occurred could indicate either a decreased ability to predict a visual target in general or to adapt rapidly to a hidden predictive situation.

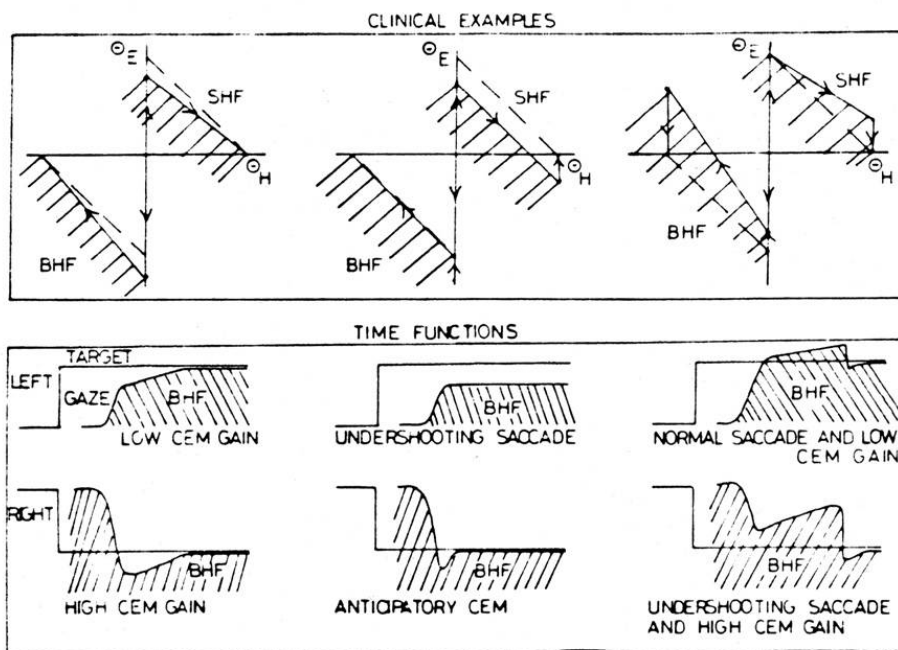
The behaviour of coordinated gaze shift movements in patients who suffered from an

isolated sensory defect with *homonymous hemianopia* was particularly interesting. When they tried to acquire a *predictable* target they tended to overshoot the target when looking to the side of the blind hemifield; they tended to undershoot the target when looking to the side of the seeing hemifield such that the target always stayed on the same side (Fig. 4). A high gain CEM when looking to the side of the blind hemifield and *vice versa* brought target exactly onto the fovea. Consequently, with gaze shift to the blind hemifield a high number of ACEMs occurred, that anticipated the CEM that occurred synchronously with the late head movement (Fig. 4, middle part). However, when confronted with *random targets* these patients tended to

eliminate this complicated strategy by keeping their head still; in case they moved, a great variety of gaze errors was likely to occur (Meienberg *et al.*, 1981; Zangemeister & Stark, 1980; Zangemeister *et al.*, 1982), one of which is depicted in Fig. 4 (right hand side). The above mentioned compensating strategies, that permitted safe looking, occurred in about 77% (Table 2: % sum of Type I and II), with a high amount of gaze type II shifts to the blind hemifield in about 25%. On the other hand gaze type IV occurred very rarely, and gaze type III, the interactional gaze type, occurred in only less than one half of the normal percentage (21%). Therefore, maladapted discrepancies were either avoided by a higher level and in most cases correct prediction, or by the safe strategy not to move the head, in case a particular target could not be predicted.

The patient with *oculo-motor apraxia indirectly* increased the size of his saccadic control signal by making larger and faster

Fig. 4. Gaze plane display (upper half) and time functions (lower half) for patients with homonymous hemianopia. Left and also middle show adaptive (s.s.) clinical examples; on right 'maladaptive' clinical responses.



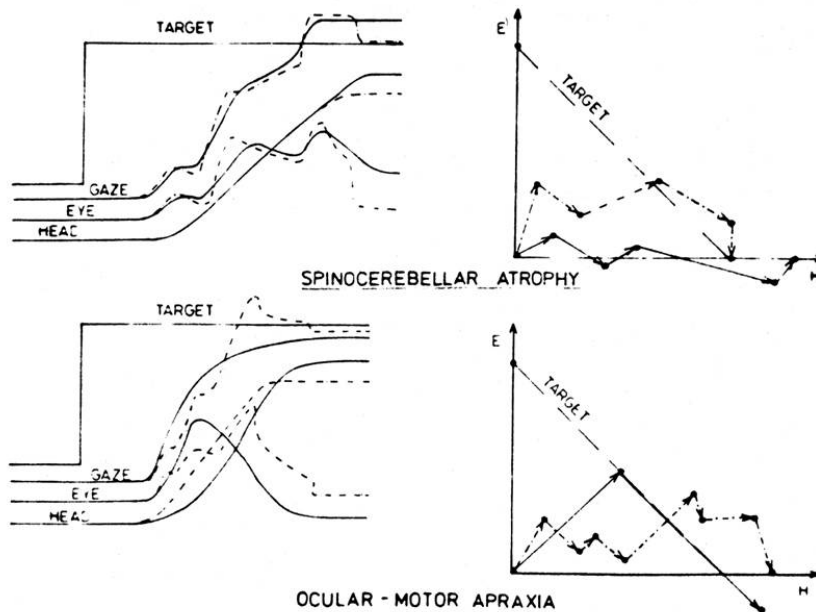


Fig. 5. Time functions (left) and gaze plane plots characteristic of two neurological disease (S.C.A.; O.M.A.), each with two typical response modes (continuous and interrupted line). E, H: eye, head.

head movements, that resulted together with the smaller eye movements in larger gaze shifts (Table 2: type IIIa: 48%). This was a strategy that appeared to be safer for early and accurately catching the target using just *one* head movement initiated voluntary saccade (Fig. 5, continuous line), and not relying on reflexive, quick-phase-like saccades. In the gaze plane it could be demonstrated that, following this comparatively large saccade, an overshooting head movement and its synchronous CEM with a gain equal to one kept the target exactly on the fovea throughout the second part of the movement. Another strategy this patient adopted was to stimulate the vestibular system firstly through a fast and relatively small early head movement, in order to induce *directly* quick-phase-like saccades during the early initiated CEM (Fig. 5, in-

terrupted line) (Table 2: type IIIb, 29%; type IV, 33%). In this case often additional small corrective saccades were necessary to bring the target exactly onto fovea, because the quick-phase-like saccades were inaccurate and/or too reflexive-like to catch exactly the target. This was depicted in the gaze plane with the interrupted line crossing the target line and ending finally with a head overshoot on the other side of the target line. This kind of strategy occurred more often with large and/or randomly occurring target amplitudes, whereas the former mentioned strategy was favoured by better prediction and smaller amplitudes.

In patients with *spinal cerebellar degeneration* both saccades *and* quick phases of nystagmus could be affected.

This leads to low velocities and small amplitudes together with prolonged latencies for initiation of these movements (Zee *et al.*, 1976). If the disease had not yet progressed too long, some slow but also some faster saccades could be generated by

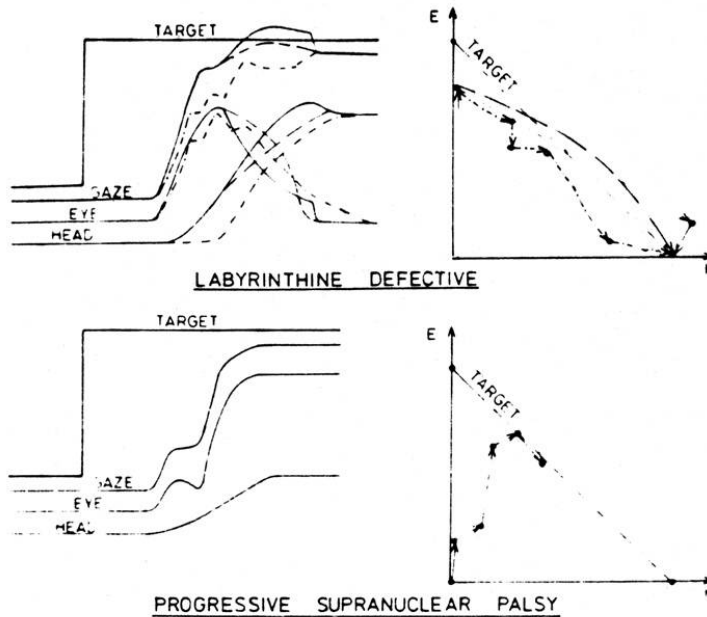


Fig. 6. Same as in Fig. 5, but for P.S.P. and Lab. Def. (three response modes in this case).

these patients: in this case an accurate head movement synchronous with a faster, first saccade and one or more quick-phase-like slow saccades, that were superimposed on the ongoing CEM (Fig. 5, interrupted line; note particularly the gaze plane plot), brought the fovea finally on to the target. In case the disease had further progressed, saccades and/or quick phases were so slow that a large overshooting head movement brought the fovea, mostly through the CEM (Fig. 5, continuous line, note gaze plane), across the target. Only with the second part of this gaze movement (not depicted in this figure) was the fovea brought back on to the target through the VOR or CEM. Gaze type IIIb and IV was consequently occurring more often in these patients (Table 2: IIIb 45%; IIIa 13%). Only for small gaze shifts and in comparatively

less affected patients did type I occur (11%).

Patients who suffered from *progressive supranuclear palsy (PSP)* synergistically enhanced their saccades by comparatively small head movements (Fig. 6, lower part). A sequence of saccades that were mostly small in amplitude was synchronously initiated with and superimposed on a relatively slow and small head movement and its concurrent CEM. Therefore, type IIIa (65%; IIIb: 5%) occurred most often in these patients; type IV may have occurred with high intent and high prediction, since head movement latency in this case appeared to be much shorter than eye movement latency for larger amplitudes. On the other hand, with small amplitudes type I (5%, unforced condition, low intent to quickly move the head) and, more prominent, type II (10%; high intent to move the head) may have occurred.

Patients with *absent labyrinthine function* (Fig. 6) frequently complain of oscillopsia (Zee, 1977; Brandt & Daroff, 1980; Atkin &

Bender, 1968). They are of special clinical interest. As Dichgans *et al.* (1973) have previously described in labyrinthectomized monkeys, there were three mechanisms that were used to compensate for this defect: the gain of the cervico-ocular reflex increased up to 0.8 with active head movements; an anticipatory CEM would be generated more often; the amplitude of saccades with active head movements was less than with immobilized head – supposedly to permit an additional gaze drift on to the target with a CEM of too low gain.

Labyrinthectomized patients generally showed hypometric saccades to target steps of various amplitudes. Because their CEM gain always was low, they used different strategies to acquire targets as quickly and accurately as possible. One strategy occurred mostly with unpredicted targets. After a hypometric saccade a CEM of too low gain was generated. This insufficient CEM was interrupted by saccades that went into the same direction as the CEM (Fig. 6, discontinuous line), or it was helped by an overshooting head movement and an additional corrective eye and head saccade on to the target (Fig. 6, continuous line).

Another strategy occurred more often with predictable targets. After a hypometric saccade a CEM would be generated, the gain of which was too low in the first part of the movement (Fig. 6, interrupted curved line). However, for the second part of this CEM its gain was increased over one; a sufficient target acquisition without use of an ipsiversive saccade was the result: with the target being predicted the CEM could be influenced in an adaptive way. This modification never took place before the head had actually moved like in the ACEM (Table 2, Type II: 0%). This is in agree-

ment with the results of Dichgans *et al.* (1973), because the initiation of the head movement was never unexpectedly blocked in our patients like in their experimental paradigm. The particular increase of the occurrence of gaze type I, and also type III, in our patients (Table 2) indicated that the head movement control signal was either earlier or synchronous with respect to the control signal for the eye saccade. It also indicated that there was never a late head movement control signal like in gaze type II (Table 2).

(III) Pathological gaze types illustrate (1) deviant ratios (DR) of gaze types, (2) reduced variance, (3) adaptation

The examination of saccades alone or the VOR alone may not reveal the real functional capacity of a patient to shift gaze promptly and accurately. The documentation of pathological gaze-shift types presented leads to the conclusion that only combined measurements of passive *and* active head rotations can be a sufficient test for gaze coordination. Adaptive as well as 'maladaptive' strategies in patients with disturbances of gaze coordination are then more accurately described and analyzed.

Normal subjects demonstrate the four gaze types with a widely varying frequency depending upon task and condition. With neurological disorders the frequencies are changed when compared to normal and the task to shift gaze is performed in many different modes that include: eye *and* head movement together; eye movement only; head movement only, with head movement triggered eye movements; either eye or head movement moderately earlier with respect to each other. The *pathological* gaze shift types show three characteristic response

TABLE 3. Deviation ratio (DR), the probability of a gaze type other than to expect normally under given experimental conditions for seven groups of neurological patients.

Diagnosis	Deviation ratio GTY pathol./GTY normal		Pathol./GTY normal III & IV
	I	II	
Cerebellar	1.3	0.5	0.9
Homonymous hemianopia	1.5	6.3	0.4
Labyrinthine defective	1.5	0.0	0.8
Parkinson	1.4	8.3	0.3
Ocular motor apraxia	0.0	0.0	1.6
Progressive supranuclear palsy	0.2	2.5	1.4
Spinocerebellar degeneration	0.2	0.0	1.2

modes under given laboratory or clinical testing conditions (*i.e.*, variant predictability and/or amplitude of target, variant intent of the subject), that are: reduction of gaze type variance (1), change of relative and absolute latencies (2) of eye and head movements, and the resultant adaptation (3) with positive (adaptation *sensu strictiori*) or negative adaptation ('maladaptation') for the given task. These modes occur with frequencies governed in part by the neurological disorders and in part by the deviant ratios.

(1) *Deviant ratios.* The deviation from normal of certain gaze types can be defined as the relative occurrence of a gaze type other than normally to expect in a given experimental condition, that is the deviation ratio: (DR = p (pathol. GTY)/ p (normal GTY), f (exp. condition) = 1 (normal), or \neq 1 (pathol.)). For example, this would be

for parkinsonian patients (see Table 3): 1.4 (type I), 8.3 (type II), 0.3 (type III & IV). This indicates that on the average late head movements (or, relative to head movements, early eye movements), are used eight times more often than normal, whereas early head movements (or late eye movements) occurred three times less than normal (Kennard *et al.*, 1982).

A very similar behaviour of DR occurs in hemianopic patients although the underlying neurological deficit is completely different (Meienberg *et al.*, 1981; Zangemeister *et al.*, 1982d). The DR of cerebellar, to a certain extent also of labyrinthine defective patients shows the relatively similar behaviour of gaze types in these patients compared to normal subjects. This is demonstrated in the upper half of Table 3 by the overall increase of gaze types II and I, and the decrease of III and IV. In terms of

stimulus response time it shows the prolonged latency or avoidance of head movements as compared to eye movements – even with large amplitudes, high prediction and high intent. This means that either avoidance (hemianopic patients) or increased latency of head movements (parkinsonian, to a lesser extent labyrinthine or cerebellar defective patients) lead to similar results.

(2) *Reduced variation.* It appears that different levels of the CNS, lead to similar gaze for head movements – that occur on different levels of the CNS, lead to similar gaze responses in quite different neurological disorders. On the other hand, the lower half of Table 3 demonstrates the opposite behaviour of gaze type occurrence and its DR. Here, early head movements (mostly late eye movements) prevail, whereas late head movements or synchronous eye and head movements occur rarely. This is a common pattern, although – compared to head movements – the prolonged latencies of eye movements are caused by disturbances on different levels of the CNS. Particularly, the patient with ocular motor apraxia was not able to generate eye movements without a concurrent head movement, that had to be generated always earlier than the eye movement because of the dependent dynamical behaviour of head and eye.

A hypothetical latency scheme (Table 4) gives an overview of the sites or levels where latency differences eventually might arise, when eye and head movement latencies are differently affected by a disease process (for further explanation of this branching latency model see also: Zangemeister & Stark, 1982a). Evidently, pathological processes on different levels/sites of the CNS yield different responses of the neural

gaze movement machinery.

(3) *Adaptation.* At the same time, with the beginning of a particular disturbance, the ability of the CNS to adapt to an unfavourably changed situation is tested. Each of the given examples demonstrates in this respect the variety of compensatory adaptation, like, e.g., the relatively accurate and fast acquisition of predictable targets in hemianopic patients, or the spino-vestibularly generated increase of loss of VOR gain in labyrinthine defective patients. This occurs in the latter patients in addition to a gaze shift strategy, that favours a synchronous eye-head movement gaze type (I). In the other noted neurological diseases in most cases possibilities exist to compensate at least partly for a disturbance: like in ocular motor apraxia with its head movement initiated eye movements, or in spino-cerebellar atrophy with small and slow saccades, and gaze shifts relying mostly on larger head movements. Of course, errors occur more often if gaze types III and IV are mostly used (Table 4, lower half), because interactions of all kinds between saccades and VOR/CEM can take place – the gaze movement by far not always being well prepared in advance or 'preprogrammed'. This is also demonstrated in hemianopic patients. The task to quickly and accurately acquire randomly given targets (in the seeing *and* blind hemifield) often leads to highly erratic gaze shifts and therefore confusion – even afterwards, when prediction is permitted.

The strategies the patients use to overcome or partly compensate for a given visual task appear to be a function of implicitly or explicitly given target predictability, or, less effective, a function of the situation wherein the target might

TABLE 4. Sites and levels of overall and relative latency differences of eye and head movements.

<i>Site of latency difference</i>	<i>Stim. to visual CX</i>	<i>Visual CX to prem.CX</i>	<i>Prem.CX to mot.CX</i>	<i>Mot.CX to BSGC</i>	<i>BSGC & SCINT.</i>	<i>Final per. pathway & DY</i>
<i>Disease</i>						
Homonymous hemianopia	one HH L↑↑	E _L & H _L ↑ asymm.: H _L >E _L	—	—	—	—
Ocular motor apraxia	—	E _L »H _L	—	—	—	—
Parkinson	—	—	E _L >H _L	—	—	secondary eff. H _L >E _L
Progressive supranuclear	—	—	—	E _L >H _L	—	—
Cerebellar	—	—	—	—	H _L >E _L	—
Labyrinthine defective	—	—	—	—	E _L >H _L	—
Spinocerebellar degeneration	—	—	—	—	E _L >H _L	—
Peripheral palsy	—	—	—	—	—	E _L ↑: H _L ↑

Abbreviations: Stim.: stimulus; CX: cortex; Prem.: premotor; Mot: motor; BSGC: brain stem gaze centres; SCINT: spino-ponto-cerebellar interaction; Per: peripheral; DY: dynamics; HH: homonymous hemifield; L: latency; E: eye; H: head; ↑ increased; eff.: effect.

appear. Failure to correctly acquire visual targets may be a kind of 'maladaptation', that is use of an otherwise sensible strategy in unpredictable situations: it results in more or less evident erratic behaviour with even more prolonged latencies to readjust to eventually given hints for predictability of the target.

This leads to the clinical impact of our studies. Certainly it might be difficult to use an abstracted scheme of eye and head latency differences and possible interactions because of independently shifting latencies. However, following simple principles of

latency differences depending upon a given task and situation as well as the site/level of the disturbance with its characteristic adaptation (or 'maladaptation') the neurologist has a valuable tool to estimate and follow-up the course of the disease and, furthermore, the practical outcome of any adaptation, that is its relevance for everyday's life. Careful observation of the gaze shift types under different conditions leads in so far to a critical evaluation of the patient's abilities to compensate for his defect, and to advise him about avoiding false or misleading strategies in specific situations.

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