CONSTANT ERROR OF VISUAL EGOCENTRIC ORIENTATION IN PATIENTS WITH ACUTE VESTIBULAR DISORDER

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INTRODUCTION

Visual projection in space is peripherally based upon the possession by each retinal receptor of 'local sign', a term originally introduced by Helmholtz (1910). Local sign is pre-eminently a function of the fovea which forms the primary point of directional reference. Images falling on the fovea are projected upon a line passing through the nodal point of the eye, constituting the line of 'principal visual direction' (see Duke-Elder, 1973). In binocular vision, corresponding retinal points have identical local sign values and the two eyes appear to act as a single, centrally-situated 'cyclopean eye' (Hering, 1861). According to Hering's law of cyclopean projection, superimposed images formed on the fovea have identical 'oculocentric direction'. To localize an object in space, however, information is also needed about position of the eyes and the fovea in relation to the head and body, which means that spatial judgements must be complemented by some kind of extraretinal information. The perceptual counterpart of oculocentric direction is the 'egocentric direction' (Roelofs, 1959), which is judged with reference to co-ordinates of the head and body. Bi-directional judgements of visual objects in space, such as 'straight ahead, to the left or to the right', etc. are made in relation to the egocentre, which is fixed in relation to the body and situated in the median plane of the head (see Howard and Templeton, 1966). The line of principal visual direction normally corresponds to the perceptual concept of 'straight ahead'.

The ultimate, visual, directional orientation depends upon the central processing and synthesizing at a perceptual level of retinal inflow and extraretinal factors related to postural activity of the body and the head and notably to the activity of the extraocular muscles. Disturbances of any of these systems may result in an illusory perception of false visual projection.

The aim of the present paper is to present the effects upon directional, visual judgements of an acute, unilateral, vestibular disorder. The problem more specifically concerns the effects of an asymmetric, vestibular inflow upon ocular position and the correlation between eye position and subjective visual space perception.
This is really part of a question which has attracted much interest in the past amongst psychologists, physiologists and clinicians: how do we perceive a spatially stable visual world?

Two theories have been proposed to explain extraretinal, oculomuscular influences upon visual space perception. One principal line of thought, originally presented by Helmholtz (1910), states that perception of space is based upon a central calculation of the motor 'outflow' to the extraocular muscles. In other words, correct localization in space depends upon the relation between intended and effected eye movements on the one hand and retinal projection on the other. This view has been supported by many authors, for instance, Bielschowsky (1935), Brindley and Merton (1960) and Festinger and Canon (1965). The second theory is based upon the supposition that space perception depends on proprioceptive 'inflow' from the extraocular muscles. The foremost advocate of this opinion was Sherrington (1918), later on followed by others (Tschermak-Seysenegg, 1947; Oppel, 1967). For a review of these and related problems see Gregory (1958) and Merton (1964).

MATERIAL

Normal Subjects
Twenty-five subjects aged between 20 and 56 years were examined for resting eye position in darkness and for direction of the visual 'straight ahead' with and without a visual frame of reference. The subjects were chosen at random amongst the staff at the Söder Hospital, Stockholm. The following criteria for normality were used; absence of any otological disease or episodes of vertigo, no history of nervous system disorders, no gross defects of visual acuity, no defects of the visual fields and no primary disturbance of ocular motility.

Patients
Fifteen patients with clinical signs pointing to an acute disorder of the peripheral vestibular system ('vestibular neuronitis') were examined for resting eye position in darkness and thereafter asked for visual directional judgements during simultaneous control of eye position in darkness. The patients were examined from one to four days after onset of the vertiginous symptoms. Six patients were female, 9 patients were male aged between 22 and 64 years. There were no limitations of range of pursuit or command eye movements tested in light. Neurological examination was normal except for the signs associated with the acute vestibular disorder.

METHODS
The subjects were all examined in the supine position. A special stand was mounted on a bed for support and fixation of the head. Above the head was mounted a metallic projection arc, on which the fixation target was presented. The arc had a radius of curvature of 30 cm. The subject's head and eyes were in the centre of the arc, which was graded in 5 degree intervals with zero corresponding to the median plane of the head. The stand also supported an infra-red television camera and four infra-red, solid-state lamps. Eye movements and eye positions were monitored on an ordinary television screen. (For details of the television equipment, see Hörnsten, Högman and Örnberg, 1973). The television method, of course, allowed inspection in ordinary visible light as well as in darkness. For
observations in darkness the subject, the projection arc and the camera system were enclosed in a black box to exclude visual cues caused by light from the monitor screen.

For evaluation of the extent of ocular deviation in darkness, individual calibrations were made by having the subject direct his gaze towards the graduations on the arc subtending different visual angles. Deviations could then be marked by a pen on the monitor screen. Stability of head position in relation to the recording camera system was checked by marking the position of the median plane of the head on the monitor screen.

The fixation object in darkness was a small, self-luminous spot with a diameter of 4 mm. The spot was painted in a fluorescent colour on a small magnetic cylinder, which was moved on the projection arc by the experimenter and could be attached to the arc at any point. In a second set of experiments, a foveal after-image was used, together with the image of the real object. The after-image was a small cross produced by an electronic flashlight whilst the subject fixed his gaze on the centre of the figure.

PROCEDURE

1. With Visual Frame

The subjects had to indicate when a fixation point of the same magnitude as the spot used in darkness was perceived in the straight-ahead position on the projection arc (scale markings on the back of the arc). The fixation object was moved on the arc from the periphery of the visual field towards the median plane. The object was moved alternately from the left and right periphery and ten trials in total were made for each direction.

2. Darkness

(a) Moving object; voluntary gaze deviations. In this trial, which was performed only on normal subjects, the straight-ahead position of the spot had to be judged by the same procedure as described with visual frame, whilst the subjects voluntarily gazed to either side approximately 10 and 20 degrees, respectively.

(b) Moving object; with and without after-image. After determination of the resting position of the eyes in complete darkness the subjects were requested to judge when the moving luminous spot appeared in the straight-ahead position. The object was moved on the arc in the same way as described above, whilst the subjects were instructed to relax and look straight ahead. Thereafter the same procedure was repeated with the simultaneous presence of a real image (luminous spot) and an after-image.

(c) Fixed object; with and without after-image. In this trial, the luminous spot was placed in the objective median plane at the zero position of the arc whilst the subjects kept their eyes closed. Immediately after eye-opening (eyes deviated or non-deviated), they had to judge the direction of the spot in relation to the median plane. Then the subjects were instructed to gaze voluntarily from any deviated position until the eyes reached the primary position and then again report the apparent visual direction of the luminous point. As in the foregoing trial, the procedure was thereafter repeated with a simultaneous after-image.

Comment

Some comments concerning the exactness of the experimental situation in darkness are required. In situation 2, there were sporadic tendencies for the subjects to direct the eye towards the moving object. Such trials were excluded. The experimental situation allowed quantitative evaluation of steady ocular position within 2 to 3 degrees. In situation 2(c), only two kinds of qualitative reports were possible; judgements of the relative positions of the real image and the after-image and the position of both the images in relation to the visual egocentre.

The head fixation device did not completely exclude the possibility of very small head movements. It was, however, for practical reasons not convenient to use a bite-board for the patients with an
acute vestibular disorder. The presence of significant head movements could be excluded by the checking procedure described above.

In all patients there was spontaneous nystagmus to a varying degree, which was the foremost obstacle to an exact evaluation of ocular position. Nystagmus, however, was beating within a rather limited range, not exceeding 2 to 4 degrees, and the average eye position could be reliably estimated by the markings on the monitor screen. The error introduced by spontaneous nystagmus is inherent in the experimental/pathological condition. The nystagmus in most cases was accompanied by some oscillopsia, which could render precise judgements of target localization difficult for the patients. Significant disturbances introduced by autokinetic sensations did not appear in the trials.

All methodological limitations considered, the most serious of which were inherent to the pathological condition, it can be stated that the recording inaccuracies were small compared to the high degree of ocular deviations.

RESULTS

Normal subjects

With access to a visual frame of reference, the straight-ahead judgements always coincided with the objective median plane. In darkness, a slight tendency to a horizontal eye deviation, of less than 5 degrees, was observed in 6 subjects. For the remainder, no appreciable deviation of the resting position of the eyes in darkness was noted.

When a fixation point was introduced in darkness, no lateral displacement of the eyes occurred and the straight-ahead judgements always corresponded to the median plane within a range not exceeding 2 to 3 degrees. No consistent difference could be noted between those subjects who exhibited small lateral deviations of the resting eye position and the rest of the normal subjects. Absence of such errors could be explained by the limitations of exactness of the measuring procedure. Voluntary lateral eye deviations did not alter the relation between judged straight ahead and the objective median plane within the same limits of error as described above.

Patients

1. Visual frame of reference. With a visual frame of reference, all patients accurately judged the visual straight ahead as aligned with the objective median plane. That is, oculocentric and egocentric visual directions coincided.

2. Darkness. All patients disclosed quite marked horizontal, continuous eye deviations of constant amplitude in the absence of a visual frame of reference. The deviations ranged from approximately 10 to 25 degrees. Spontaneous nystagmus was in all cases beating in the opposite direction to ocular deviation. (Where oscillopsia was present, a more or less distinct sensation of a ‘streaming’ motion of the target towards the fast phase of nystagmus was reported.)

With the eyes in the resting, deviated position 2(b), the perception of the subjective straight ahead was in all cases displaced towards the side of ocular deviation to an extent which closely corresponded to ocular position (see fig. 1). When an after-image was introduced, the real image and the after-image were superimposed.
With the eyes in the resting, deviated position and the fixed spot presented in the objective median plane 2(c), all patients, after eye opening, judged the spot to be dislocated far to the side opposite to the direction of ocular deviation (for example, with ocular deviation to the right, the spot was displaced to the left). With simultaneous presentation of a real object and an after-image, there was a separation between the two images. The image of the real object (spot) was still judged as displaced, in the same direction and to approximately the same extent as before. The after-image, however, was judged to be straight ahead.

In the second part of trial 2(c) when the patients voluntarily moved their eyes to the median plane, the spot fixed at zero position still seemed to be displaced in a direction opposite to the basic resting deviation. Most patients judged the displacement to be of the same magnitude as in the first part of this trial. Some patients, however, seemed to experience an increased sensation of displacement. When the trial was repeated with a simultaneous after-image, the real image and the after-image were judged to be superimposed or situated very close to each other.

**DISCUSSION**

Disturbances of visual egocentric localization have been clinically recognized in patients with recent paresis of extraocular muscles for more than a century (von Graefe, 1854) and are a commonplace experience in clinical practice (see Cogan, 1966; Walsh and Hoyt, 1969).
A simple explanation of false visual egocentric localization (as indicated by past-pointing) with extraocular paresis could be given by referring to the abnormal retinal image projection caused by the failure of the eye to be accurately directed against the object of intended gaze. The extent of false localization then would be determined by the retinal image projection in relation to the fovea. Such a theory exclusively utilizes retinal local sign and has been supported by, amongst others, Adler (1945), Goswami (1967), Bedrossian (1969) and Walsh and Hoyt (1969). It has, however, been demonstrated that false orientation cannot be satisfactorily explained by retinal local sign alone, but is caused by the added effects of retinal image projection and extraretinal factors related to the extraocular muscles. In fact, this was realized by von Graefe (1854) who stated that past-pointing could not be due exclusively to retinal local sign, but was related to oculomuscular abnormality; 'the patient is given the impression that he has turned his eye much more than is in fact the case: which results in a dislocation of the visual field in relation to his body', a statement which illustrates the distinction of egocentric visual localization.

Experimental evidence of extraretinal influences upon visual egocentric localization has been offered after pharmacological paralysis of extraocular muscles (Kornmüller, 1931) or when ocular movements are mechanically restrained (Mach, 1886). Brindley and Merton (1960) performed related experiments and noted an apparent displacement of the visual world in the direction of intended gaze when the eyes were fixed by forceps and after partial paresis of extraocular muscles by administration of curare. They concluded that the perceptual errors could only be explained by the effort of will employed in attempts to move the eyes. The same kind of explanation for past-pointing in paralytic strabismus was proposed by von Noorden, Awaya and Romano (1970), who convincingly demonstrated that false orientation is caused by a disproportion between motor innervation and its effects.

Time-honoured observations of the perceptual consequences of passive and active eye movements may illustrate the role of the extraocular muscles. When the eyes are voluntarily moved, the visual world is stable. With passive eye movements, for instance, by pushing the eye with the finger, a movement of visual space is perceived in a direction opposite to the ocular displacement. Evidently, retinal image displacement may or may not cause a sensation of apparent movement and displacement of visual space. The perceptual divergence caused by active and passive eye movements was ascribed by Helmholtz (1910) to a central monitoring of oculomotor outflow; 'our judgements as to the direction of the visual axis are simply the result of the effort of will involved in trying to alter the adjustment of the eyes'. It was emphasized by MacKay (1958) that an attempt to move the eyes is accompanied by an expected displacement of the retinal image, which means that oculomotor signals and information of retinal image displacement cancel each other out in the calculation of visual space. With passive eye movements, there is no cancellation of the retinal image, hence an apparent displacement of visual space occurs.
On the other hand, an induced after-image will be immobile with passive eye movements because there is allowance for neither a central oculomotor command, nor for any displacement of the retinal image. With active eye movements, the behaviour of an after-image is quite different. The after-image seems to move whether the intended eye movement can be effected or not (Helmholtz, 1910; Jampolsky, 1970); there is no cancellation of the oculomotor signal by any retinal image displacement, which means that the retinal after-image changes its space value, corresponding to the impulse for the eye movement.

Although most psychophysical and clinical evidence seems to confirm the 'outflow theory', it has not been completely unchallenged.

Some experimental observations on a few subjects directly contradict the outflow theory. According to Siebeck and Frey (1953) and Siebeck (1953), no illusory movement appears with attempted gaze in a completely curarized subject. Similar experiments have recently been performed on one subject by Stevens, Emerson, Gerstein, Kallos, Neufeld and Rosenquist (1976) who, after orbital administration of curare, reported that with subparalytic doses, an apparent movement was perceived, whereas no movement appeared with paralytic doses. However, apparent spatial displacement, as indicated by past-pointing, was reported with partial as well as complete paresis of extraocular muscles. The authors commented upon the results of Siebeck and Frey and pointed out that the latter had not made the distinction between apparent movement and displacement. They concluded that perception of spatial localization depends upon a spatial system (separate from an independent pattern visual system responsible for perceptions associated with movement) operating by monitoring the oculomotor outflow. An additional recent report (Brindley, Goodwin, Kulikowski and Leighton, 1977) confirms in all essentials the findings of Stevens et al. (1976). According to these authors, the results 'strongly suggests that proprioceptive information can be used in correcting eye movements; but the argument is not compelling'. Indications have also been presented in favour of an afferent inflow mechanism utilized for eye position information. Skavenski (1972) and Skavenski, Haddad and Steinman (1972) investigated the relative contribution of inflow and outflow to the control of eye position and the perception of direction. They concluded that some non-visual, afferent information can be used for position control but perception of direction depends primarily upon the outflow to the extraocular muscles.

Thus recent research indicates that outflow and inflow may contribute differentially to ocular position control and to perception of movement and direction; for perception of direction the outflow mechanism seems undisputed.

The principle of a central monitoring of oculomotor outflow has been called 'efference copy' (von Holst and Mittelstaedt, 1950) or 'corollary discharge' (Sperry, 1950). Modern cybernetic models of oculomotor control systems include the operation of such a principle; in the functional control of the pursuit system (see Young, 1971), for retinal error correction in the saccadic system (Robinson,
1975) and in visuovestibular interaction to account for stability of the visual world during head movements (Raphan, Cohen and Matsuo, 1977, Robinson, 1977).

From a clinical point of view, the discussion of the relative influences of outflow and inflow in extraocular paresis is summarized by von Noorden (1975): ‘There is currently no clinical evidence to indicate the significance of inflow in either complementing or counteracting the information provided by outflow in the control of eye position or the perception of direction.’

Beside investigations concerned with the effects of clinical and experimental paresis of extraocular muscles upon visual egocentric localization there are several studies of the influence of experimental, vestibular stimulation upon visual perception and orientation. Only a few papers, however, have more specifically been concerned with egocentric localization.

A fixation target presented in the objective median plane and rotating with the observer in darkness would appear to move in the direction of acceleration (fast phase of nystagmus). The apparent movement is called the oculogyral illusion and was originally explained by retinal image displacement during the slow phase of nystagmus (Graybiel and Hupp, 1946). Conclusive arguments against an explanation in terms of retinal image tracking are based upon the fact that the illusion may appear even in the absence of any ocular nystagmus or retinal tracking. The illusion is thus present in patients with oculomuscular paresis or complete ophthalmoplegia (which renders nystagmic reactions impossible), with a stabilized retinal image (van Dishoeck, Spoor and Nijhoff, 1954, Byford, 1963) and with an induced visual after-image (Whiteside, Greybiel and Niven, 1965).

During angular acceleration of healthy subjects an apparent static target-displacement has been reported in the direction of rotation (Fischer, 1928, Fischer and Kornmüller, 1931, Morant, 1959). The target must be adjusted in the direction opposite to acceleration (slow phase of nystagmus) to be perceived in the apparent median plane. Brecher, Brecher, Kommerell, Sauter and Sellerbeck (1972) were able to demonstrate a progressive deviation of the subjective straight ahead with increasing angular acceleration. According to other authors, however, there is only a transitory displacement of the fixation target during vestibular stimulation (Byford, 1963, Whiteside et al., 1965).

According to Fischer and Kornmüller (1931) the deviation of the subjective straight ahead may be caused by a deviation of the eyes in direction of the slow phase of nystagmus. In those experiments, however, there were no recordings of eye movement or eye position. In this context the observations of Byford (1963) are of special interest. By means of a sensitive, photoelectric contact-lens device, a deflection of the eyes in direction of the slow phase of nystagmus was recorded during strong angular acceleration. Byford pointed out that the displacement of the apparent median plane could be correlated to eye position. The deflection was found to ‘be in broad quantitative agreement with the subject’s estimate of the initial angular difference between the primary visual axis and his fixation lamp’.
However, in one subject with an abducent palsy (incapable of eye movement in one direction) and in subjects with stabilized retinal images, the illusion was not accompanied by any static displacement of the target.

The apparent visual target movement and displacement of the oculogyral illusion has been explained by Whiteside et al. (1965) as an effect of the efferent oculomotor program of those muscles which are antagonistic to the slow phase of nystagmus. This kind of explanation presupposes the operation of an efference copy. The explanation holds true for real images as well as for after-images, whether the movement is effected or not: for example, whether or not the fast-phase efferent activity results in an eye movement or not, there is no retinal image displacement to be cancelled out with a real object (saccadic suppression) or with an after-image. The resulting illusory movement thus will be in the direction of fast-phase efferent activity.

False localization occurring with extraocular paresis may be said to represent the end point of a kinetic illusory perception, which is characterized by a strong feature of illusory movement during an attempted saccade. The efference copy suggests that the eye has been moved and the movement is expected to be cancelled by a corresponding displacement of the retinal image of a stationary object. Thus, allowance is made for an eye movement which has not been effected and which is consequently not accompanied by any displacement of the retinal image. This is subjectively interpreted as if the stationary object is moving with the eye, and is displaced in the direction of attempted gaze.

An analogous hypothesis may be proposed to explain the apparent displacement of the median plane which occurs with steady eye deviations (continuous deviation of constant amplitude) in a vestibular disorder. Subjectively, the patients are unaware of the strong conjugate eye deviation. The efference copy tells them that the eyes are in a position of rest or straight ahead (which means zero value for ocular position). A visual object, isolated from other visual references, must also be projected with a zero value, that is, in the fovea, to be perceived as straight ahead. When the target is presented in the objective median plane and the eyes are still in a deviated position, the retinal image of the target is projected outside the fovea towards the ocular deviation. According to the efference copy, the target will be subjectively displaced away from the direction of ocular deviation. A foveally induced after-image, however, is perceived as separated from the real target and subjectively as straight ahead, because eye position and retinal projection both have zero values.

If the eyes are voluntarily deviated to the objective median plane and the visual target is presented in the same plane, the real target will still be perceived as displaced in the same direction and to the same extent. The displacement occurs in spite of foveal projection of the real target, because the efference copy suggests that the eyes are deviated (for example, in a patient with a left-deviated resting position, the efference copy suggests a right ocular deviation when the target is objectively in the median plane). This is interpreted as if the real target is displaced
in the direction of efferent activity—the resemblance with false localization in oculomuscular paresis is apparent. A foveally induced after-image is now subjectively displaced in the same direction and to the same extent as the real target and is approximately superimposed upon the real target. The efference copy suggests that the eyes are deviated when the after-image is on the fovea; thus it moves with the direction of gaze in exactly the same manner as an after-image normally behaves with change of voluntary gaze.

Conjugate, horizontal eye deviation (of which the patient is totally unaware) towards the side of the lesion is a prominent feature of the lateral medullary syndrome (Hagström, Hörnsten and Silfverskiöld, 1969; Hörnsten, 1974). Kommerell and Hoyt (1973) tested the visuospatial orientation in a patient with a left lateral medullary syndrome. The patient was asked to look at a dim flashlight presented in complete darkness and to indicate the position of the fixation light immediately after it had been turned off. Past-pointing occurred to the right and the authors ascribed the spatial mislocation to the patient’s false interpretation of gaze direction. The spatial disorientation in this case is the same as was found with subjects in the second test situation of trial 2(c). The constant error of visuospatial localization in the trials presented in this paper must basically be related to the conjugate eye deviation and the patients’ false interpretation of direction of gaze.

The perceptual consequence of the ocular deviation can be represented by a simple diagram (modified from Robinson, 1975), which includes the operation of an efference copy or extraretinal position signal in the control of saccadic eye movements (see fig. 2). According to Robinson information of initial eye position relative to the skull is needed for the calculation of goal-directed saccadic eye movements. This requires that retinal error (e) (difference between eye position in space (E) and target position in space (T)) is added to an efference copy to create an internal representation of target position into a head-co-ordinate reference frame. ‘How this signal is extracted from the visual system and where and how the efference copy is used is entirely unknown at present, and is, of course, of intense interest to those concerned with the problem of how we perceive the spatial location of seen objects’ (Robinson, 1975).

In the presented cases the efference copy told that the eyes in darkness were directed straight ahead in spite of a strong lateral deviation. It thus seems reasonable to assume that the efference copy signal is taken off before the vestibular bias is added as shown in the diagram. Consequently, the efference copy does not reflect the entire, final oculomotor outflow. It must come from some place more centrally situated (Robinson, personal communication, 1979). The final outcome will be that a continuous error is introduced to the outer feed-back loop E-T with a resulting constant error of eye position relative to the visual target. The magnitude of the error will be proportional to the pathological vestibular inflow defined as degrees of lateral deviation.

Besides the psychophysical and clinical approach to problems of visuospatial stabilization, some important neurophysiological results have been obtained during recent years by single-cell recordings. The classic investigations of Hubel and Wiesel (1968) have shown that striate neurons in the monkey have a very high specificity for different visual parameters, such as orientation and direction. In the context of the matter of stabilization of the visual world, Wurtz (1969a) investigated the responses of striate neurons in the monkey during rapid eye movements and compared the effects of eye movement and stimulus movement.
He found that of a total sample of 188 neurons giving an excitatory response to a stationary or slowly moving stimulus when the eyes were still, about half did not respond at all during a rapid eye movement. During rapid eye movements some neurons reversed from an excitatory response to a stationary stimulus to a suppression response when the eye rapidly moved over the same stimulus. According to Wurtz, the suppression neurons may be well suited to signal rapid translation of the visual background and provide some information that a rapid eye movement has occurred. Those findings may indicate that cues for stability of the visual world are available in terms of striate response patterns. Wurtz (1969b) also considered the question whether absence of response in half of the 188 striate neurons during rapid eye movement could be explained by an active inhibition of a corollary discharge, by comparing the response during a rapid eye movement across a stationary stimulus with the response during rapid stimulus movement in front of a stationary eye. With a corollary discharge, a difference of the neural response would be expected depending on whether a retinal stimulus movement is caused by a real movement of the object or a movement of the eye. Wurtz did not find any evidence of a corollary discharge operating at the level of the striate cortex. The findings, however, did not exclude the possibility of a corollary discharge at other levels of the visual system. Such evidence was found by Robinson and Wurtz (1975) in neurons in the superficial layers of the monkey’s superior colliculi which can differentiate between retinal stimulus movement induced by actual movement of an object and by eye movement. There is a striking suppression of activity in cells which responds to visual stimuli in association with eye movements. The suppression is present in light as well as in darkness and the inference is made that the suppression must result from an extraretinal signal with characteristics which would be expected by a corollary discharge although ‘... proprioception as a source of the extraretinal signal can not be excluded’.
According to Robinson and Wurtz (1975), information coded in the collicular cells may have a perceptual function via colliculo-thalamocortical projections. Execution of a goal-directed saccade presupposes coding of target position in space, that is, in a head-body frame of reference. This, as stated above, besides a retinal error signal, demands an extraretinal error signal, which may be conveyed by 'quasivisual' cells of the superior colliculi in the monkey (Sparks, Mays and Pollack, 1977). Those findings are compatible with Robinson's model (1975) of the saccadic system, in which the extraretinal signal (effference copy) is added to retinal error to provide an internal representation of target position with respect to the head.

Finally, there is some physiological evidence for interaction between oculomotor proprioception and the primary visual cortex in the cat. Fiorentini and Maffei (1977) have shown that interruption of extraocular proprioceptive inflow in the cat results in instability of ocular position and pendular oscillations in darkness, which may be an indication of the role of ocular proprioception for oculomotor function and visual stability.

To conclude with a quotation from a recent workshop summary of supranuclear influences upon brain-stem gaze neurons (Roucoux and Crommelinck, 1977) '... an effference copy would elegantly solve the problem of perceptual space stability. In this context, the role of proprioception remains mysterious'.

For the time being, we must accept the operation of an effference copy to explain the phenomenon of false visual localization. In the case of an extraocular muscle paresis, the copy comprises an intact prenuclear and nuclear gaze mechanism which does not result in an adequate motor response. In the case of an acute peripheral vestibular disorder, there are no primary oculomotor defects at prenuclear, nuclear or peripheral levels. The pontine centres for horizontal gaze, however, are unevenly loaded by the asymmetric vestibular inflow at a prenuclear oculomotor level, which results in a directionally constant distortion of the effference copy, and, at the perceptual level, results in a constant error of visuo-spatial localization.

**SUMMARY**

Errors of visual egocentric localization are well-known in patients with paresis of ocular muscles or paresis of conjugate gaze. In the present paper a series of patients with unilateral vestibular disorder disclosed a constant lateralization of the visual egocentre in the absence of any ocular paresis. The perceptual illusion is associated with an altered resting position of the eyes caused by the vestibular imbalance. The disturbance of visual, egocentric localization was revealed only after elimination of the visual frame of reference and the extent of lateralization of the visual egocentre was proportional to the degree of resting deviation of the eyes.

Although the findings are of limited clinical importance they have a considerable theoretical interest. From the clinical point of view they may provide a basis for
further understanding of the complex perceptual illusions which may accompany disorders affecting central vestibulo-ocular connections.

The results indicate that the perceptual effects are related to an altered central evaluation of the oculomotor programme and thus depend upon the operation of an 'efference copy'. This hypothesis is discussed with reference to earlier and current theories of visual localization.

REFERENCES


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