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CASE REPORT

OPEN-LOOP SYNDROME: ONE PLEGIC AND ONE AMAUROTIC EYE

D. STRAUMANN and V. HENN
Neurology Department, University Hospital, CH-8091 Zürich, Switzerland

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Summary—1. Smooth pursuit eye movements, optokinetic and vestibular nystagmus were measured in a woman with subtotally resected bilateral spheno-petro-clival meningioma.

2. She had complete ophthalmoplegia of the left eye and amaurosis of the right eye. This combination constitutes the classic condition for opening the gaze pursuit feedback-loop, as the retinal-error velocity cannot be minimised by the plegic, but viewing eye.

3. Sinusoidal pursuit stimuli with an amplitude of 5 deg at 0.5 Hz caused the mobile eye to move with an amplitude of 35 deg, reflecting an open-loop gain of 7.

4. A similar value was observed during vestibular stimulation, suggesting that, in the absence of visual correction, the vestibulo–ocular reflex (VOR) changed its gain in tandem with the gaze pursuit system.

Key words—Smooth pursuit eye movements; optokinetic nystagmus; vestibulo–ocular reflex; neuro-ophthalmology; oculomotor disorder; electronystagmography.

INTRODUCTION

Retinal slip of an image usually leads to the perception of motion, which in turn provides the stimulus for smooth pursuit eye movements or optokinetic nystagmus (OKN) (Yasui and Young, 1975). Current concepts on the generation of these movements assume feedback-loops to correct eye velocity according to stimulus velocity (Robinson *et al.*, 1986; Deno *et al.*, 1989; Krauzlis and Lisberger, 1989). The negative feedback hypothesis represents the logic of the gaze pursuit system in the most simple way: retinal-error velocity (= target velocity – eye velocity, measured at the retina) serves, after amplification, as an updated eye velocity output signal (Fig. 1).

In normal subjects, the input–output gain* of gaze pursuit is close to unity, i.e. retinal slip is effectively minimised. One can open the feedback-loop by stimulating an artificially immobilised eye and recording movements of the mobile but covered eye. Such experiments have been conducted in rabbits (Ter Braak, 1936; Collewijn, 1969), monkeys (Körner and Schiller, 1972; Behrens *et al.*, 1989) and humans (Grüsser *et al.*, 1981). Input–output gains in the open-loop condition consistently were above unity and reached values in the range of 10 at stimulus velocities around 10 deg/s. Similar results have been obtained in patients with complete unilateral ophthalmoplegia by measuring the covered healthy eye (Ohm, 1926; Körner and Dichgans, 1967; Feldman *et al.*, 1969; Leigh *et al.*, 1982).

In the present study, we describe a patient with a chronic open-loop condition, as one eye was completely ophthalmoplegic the other blind. In addition to the predicted high gain during pursuit and optokinetic stimulation, we found the same increase in gain for vestibular–ocular responses. This finding suggests that the gain of the vestibulo–ocular reflex (VOR) changed in tandem with the gaze pursuit system and was not corrected separately.

*Gain is eye velocity divided by stimulus velocity.

CASE REPORT

A 57-yr-old woman was operated on because of a bilateral spheno-petro-clival meningioma (WHO-Grading I) that infiltrated the cavernous sinus on both sides. Prior to the operation, a progressive paresis of all left eye muscles developed. The larger portion of the tumor, situated on the right side, was extirpated, including the infiltrating mass in the right cavernous sinus. Tumorous tissue on the left side was not completely resected to prevent blindness of the left

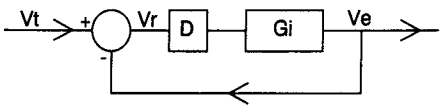

Fig. 1. Simple negative feedback control system for smooth pursuit eye movements and optokinetic nystagmus. Input is target velocity (Vt), that is compared with the actual eye velocity (Ve). The difference ($Vt - Ve$), as measured at the retina, yields the retinal-error velocity (Vr), which, after being amplified (Gi) with some delay (D), serves as the updated eye velocity signal (Ve). Depending on the amplification factor (Gi), the overall-gain (Gc) of the system can be predicted, and vice versa: $Gc = Ve/Vt = Gi/(1 - Gi)$. The system does not explain all aspects of normal gaze pursuit behaviour, but provides a scheme that illustrates some pathophysiological features in the patient presented.

eye. Postoperatively, the right eye was amaurotic, and the left eye showed a complete internal and external ophthalmoplegia including ptosis. Our study was undertaken 1 yr after surgery. The oculomotor deficits had remained stable with full visual field of the left eye and with a visual acuity* of 0.4, when the patient was wearing her glasses. Because of ptosis she used her left index finger to push up the left eye lid during the day. When released, the eye remained open for up to 30 s with a slow downward movement of the lid. Therefore the patient had some useful vision without permanently holding the lid.

MATERIALS AND METHODS

Informed consent was obtained from the patient after the procedures described below had been fully explained. Movements of the right eye were recorded using direct current electro-oculography (EOG). The patient was seated on a vestibular chair, which was surrounded by an optokinetic drum. Chair and drum were independently driven by servo-controlled torque motors. Pursuit stimuli were given in darkness by oscillating the drum on which a small light was mounted. Testing included pursuit, optokinetic, and vestibular stimulation in the horizontal plane. Due to amaurosis, the measured eye could not be calibrated by a fixation task. Therefore, eye movements were simultaneously recorded on videotape and with EOG. We estimated the eye excursion as seen in the video and compared it with the corresponding EOG-signal to calculate the approximate EOG-gain.

*Compares to 20/50 Snellen visual acuity.
†The eye could still be closed during blinks.

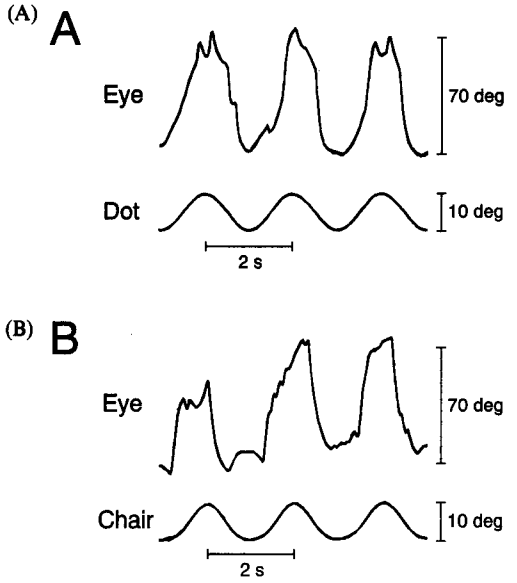

Fig. 2. Sinusoidal stimulations. Horizontal movements of the blind right eye. (A) Smooth pursuit eye movements elicited by a moving dot in an otherwise dark surround (±5 deg, 0.5 Hz). (B) Nystagmus elicited by rotation of the vestibular chair in the dark (±5 deg, 0.5 Hz). Note the large amplitude of anticipatory fast phases.

Despite her mental disorder, the patient readily followed instructions. During investigations, the left ptotic eye was kept open by a paper tape.†

RESULTS

In the light and in the dark, the mobile eye showed spontaneous centrifugal drift. Ocular re-centering by rapid eye movements was irregular in time and amplitude, often coupled with blinks.

Low-amplitude sinusoidal pursuit stimuli in the otherwise dark surround caused massive excursions of the mobile eye [Fig. 2(A)]. Taking into account some uncertainty of calibration, we estimated a gain of 6–9. Already with stimulus amplitudes of 10 deg, the eye reached the limits of the oculomotor range. Optokinetic stimulation of 10 deg/s led to slow-phase eye movements with gains between 7 and 11 [Fig. 3(A)]. There was no asymmetry between rightward and leftward optokinetic nystagmus. We did not specifically test for an increase of slow-phase velocity during long-duration optokinetic stimulation, which had been reported in experimental open-loop conditions.

Vestibular sinusoidal stimuli at low amplitudes and frequencies (e.g. ±5 deg, 0.5 Hz) produced nystagmus with large anticipatory fast phases in the stimulus direction resulting in

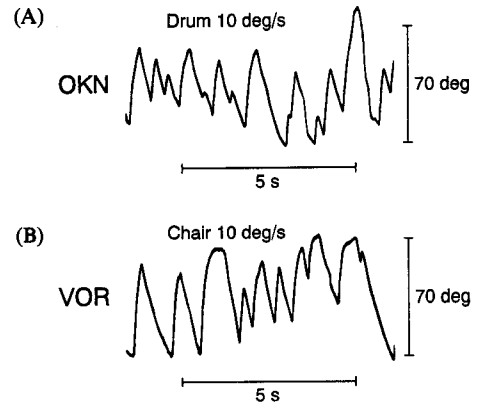

Fig. 3. Constant velocity stimulations. Horizontal movements of the blind right eye. (A) Nystagmus elicited by rotating the optokinetic drum, which totally surrounded the patient (10 deg/s). Mean slow-phase eye velocity of this section is 81 deg/s. (B) Nystagmus elicited by an angular velocity step of the vestibular chair in the dark (10 deg/s). The trace shows a section 5 s after starting chair rotation. The mean slow-phase eye velocity is 97 deg/s.

peak-to-peak eye displacements of about 70 deg [Fig. 2(B)]. This pattern is seen in normal subjects during high-amplitude (e.g. ± 40 deg, 0.5 Hz) vestibular oscillations. Gains of vestibular step responses at chair velocities of 10 deg/s were between 8 and 10 [Fig. 3(B)]. There was no difference between rightward and leftward vestibular nystagmus.

DISCUSSION

The patient showed the classic combination of disorders that would generate gaze pursuit eye movements in an open-loop mode: one eye was plegic, the other amaurotic. In an intact pursuit feedback system, the difference between target velocity and eye velocity provides, after amplification, the updated eye velocity signal. If the viewing eye becomes plegic and the moving eye is not able to see, eye velocity cannot be compared with target velocity. Consequently, eye velocity equals the inner amplification factor times the target velocity. In the reported case, we measured an open-loop gain between 6 and 11 at stimulus velocities of 10 deg/s. Similar results have been obtained in patients with unilateral complete ophthalmoplegia or in normal subjects with a pharmacologically immobilised eye when the mobile eye was covered. In these experiments the opening of the feedback-loop was acute and reversed after uncovering the moving eye. In our patient, the open-loop situation had persisted for 1 yr. Nevertheless, the open-loop gain was comparable to the reported acute experiments.

During vestibular stimulation, we measured similar high gains as during pursuit and optokinetic trials. Normal VOR-gain, which usually ranges somewhat below unity, is constantly adjusted according to the retinal slip occurring during head movements. Persistent image slip in the opposite direction of the head turn causes the VOR-gain to increase over hours and days (Miles and Fuller, 1974; Gauthier and Robinson, 1975). In the patient, this mechanism of visual VOR-adaptation had been driven to its upper limit as the retinal slip could not be minimised, even with a very high VOR-gain, i.e. the presumed negative feedback-loop for visual VOR-adaptation had also been opened. Since the estimated gains for smooth pursuit, OKN, and VOR were all similar we hypothesise that, in the absence of visual correcting factors, gain adjustments for optokinetic and vestibular nystagmus might not occur independently. This is supported by neurophysiological data which show that at least some of the pathways in the vestibular nuclei and the flocculus of the cerebellum are shared (review: Waespe and Henn, 1987).

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