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Cervico-Ocular Responses (COR) During Slow Sinusoidal Head Movements in Subjects with Bilateral Labyrinthine Lesions

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Summary. Eye movements in five patients with chronic bilateral labyrinthine loss were tested with sinusoidal movements during cervico-ocular stimulation and active head movements (0.05, 0.1 and 0.2 s^{-1}; 20, 40, and 60°) and were compared with healthy subjects. Consideration was given to saccadic activity and slow phase velocity of nystagmus and overall gaze shift. The cervico-ocular response was not altered in the patients. During active pendular head movements the saccadic activity and slow phase velocity of nystagmus were more reduced than the eye shifts. The phase relation of eye shifts was not changed. In our patients neck to eye responses did not compensate for the abolished vestibulo-ocular reflex.

Key words: Bilateral labyrinthine loss – Cervico-ocular reflex (COR) – Vestibulo-ocular reflex (VOR)

Introduction

The functional role of neck afferents from cervical joint and muscle receptors in the control of eye and head movements is still not completely understood. Cervico-ocular responses (COR), recorded in the absence of vision, with the head fixed in space during sinusoidal trunk movements of a seated subject, are highly influenced by the perception of movements. These responses increase with the illusion that the head is moving, when the trunk is swinging sinusoidally (Doerr et al. 1982; Thoden et al. 1983). This indicates not only that this reflex system is modulated by a short reflex pathway via the vestibular nuclei to the oculomotor system (Anastasopoulos and Mergner 1982; Hikosaka and Maeda 1973; Kasper and Thoden 1981), but also by long loop connections and cortical influences.

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Moreover it was hypothetised that the average eye shifts and large saccadic eye movements at the beginning of an active head movement may depend more on cervical input than the nystagmus which is superimposed on these slow deviations (Doerr et al. 1981).

Patients with chronic bilateral labyrinthine loss offer the possibility to study COR without the contamination by vestibular influences. It must be recognized, however, that the COR in this situation may be enhanced for the compensation of the absent vestibulo-ocular reflex (VOR), as described in acute animal experiments (Dichgans et al. 1973).

Methods

Five patients with complete bilateral loss of labyrinthine function for more than one year were studied (Table 1) and compared with five healthy subjects of comparable age, who were described in our previous study (Doerr et al. 1981).

Horizontal eye movements were recorded using dc electro-oculography with the electrodes placed on the outer canthus of each eye. Loss of labyrinthine function was confirmed by the absence of oculomotor reaction to bithermal caloric stimulation, rotation tests and absent optokinetic-after-nystagmus (Zee et al. 1976). In a second session COR during passive trunk movements (with the head stationary) and during active head movements were investigated.

The head was fixed in a head holder, allowing movement in the horizontal plane, and angular positions were recorded by a potentiometer. With covered eyes, the patients sat on a chair (Tönnies) which could be rotated sinusoidally at frequencies of 0.05, 0.1 and 0.2 Hz and total amplitudes of 20, 40 and 60°. Resulting maximum velocities ranged between 6.28 and 75.39°/s. To achieve pendular active head movements within the same range of parameters, a rhythmic tactile stimulus, indicating frequency and amplitude was applied to the patients. A short practice was necessary before beginning the test.

Thoden et al. (1983) have recently shown that the gain of COR can be modified by instructing the subjects to imagine head or trunk movement. Accordingly during these experiments, the subjects were simply instructed to stare blankly ahead in the dark while performing mental arithmetic. They did not know the purpose of the experiment.

During the COR recording the eyes were covered. We found no difference between COR with covered and closed eyes (Takemori and Suzuki 1971), with the exception that with closed eyes muscle artefacts were more prominent.

For optokinetic stimulation a stripe pattern was projected on a large cylindrical screen in front of the patient. Stimulus velocities were 60 and 90°/s to both sides for 10 s. Optokinetic-after-nystagmus was tested in the dark following optokinetic stimulation with 60°/s for 1 min.

The recorded eye movements were analysed by hand and related to trunk and head movements. Only fast eye movements with a velocity greater than 100°/s were assumed to be saccades (Barnes and Forbat 1979).

The following parameters were calculated:

a) Total Saccadic Amplitudes. The saccadic amplitudes per period were summed and averaged over five cycles for right and left sided saccades. Since no significant directional preponderance was found, mean values for left and right saccades were used for further evaluation. These mean values therefore represent the net saccadic activity for half a period of the sinusoidal stimulation.

b) Gain of Nystagmus Slow Phase Velocity. It is defined as the maximum slow phase velocity versus maximum stimulus velocity. For its determination the fastest slow phase velocity found during the stimulus period was chosen and averaged over five periods of stimulation. The form of a single nystagmus in COR resembled a staircase pattern more than a usual saw-

Table 1

Patient	G.E.	Sch.T.	E.H.	D.E.	J.A.
Age	16 years	16 years	58 years	63 years	70 years
Sex	♂	♂	♂	♀	♀
Diagnosis	Deaf mute	Deaf mute	Meningitis at 2 years	Metastases of the basal skull	Bilateral labyrinthine lesion, unknown aetiology
Duration of illness	16 years	16 years	56 years	1 year	4 years
Hearing	Deaf	Deaf	Deaf	Deaf	Hypacusis
Central neurological disturbances	Bilateral horizontal gaze nystagmus	Slight gaze nystagmus to the left	Disturbed slow pursuit eye movements	(Bilateral VII paresis)	Disturbed slow pursuit eye movements
Gait disturbance	–	+	+	+	+

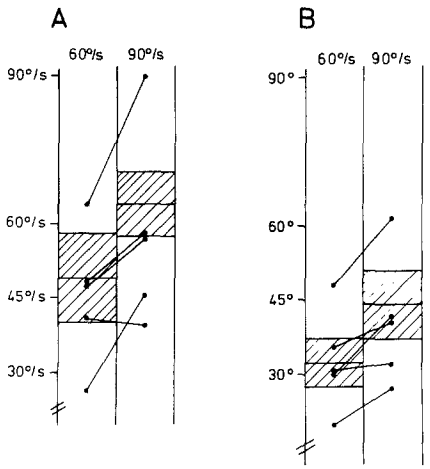

Fig. 1A, B. Optokinetic responses of five patients compared with those of five normal subjects. Shown are the mean slow phase velocities (**A**) and mean total saccadic amplitude/s (**B**) at stimulus velocities of 60° and 90°/s (abscissa) for each patient. Hatched area indicates mean values with standard deviations of five healthy subjects of comparable age

tooth nystagmus. This is because both slow eye deviations and fast components of nystagmus were directed parallel to the relative head movement i.e. opposite to the trunk movement (Barnes and Forbat 1979).

c) Amplitude and Phase of Maximum Eye Deviation. The peak to peak amplitudes of the extreme eye positions reached during one cycle of stimulation were averaged over five periods and its relation to the stimulus position determined (phase).

Results

The clinical data of the five patients are given in Table 1. All of them had bilateral complete lesions for more than one year. Their gait disturbances where characteristically pronounced in the dark. A spontaneous nystagmus was never seen.

Slow phase velocities of optokinetic nystagmus at stimulus velocities of 60 and 90°/s were compared with mean values of five normal subjects. Only at a stimulus velocity of 90°/s were the slow phase velocities below normal values in four of the patients (Fig. 1). The saccadic amplitudes, averaged/s were slightly diminished in these patients. One patient, however, a deaf mute (G.E.), exhibited higher slow phase velocities and saccadic amplitudes than the control group. This patient did not show constant differences in all the other tested parameters (Fig. 1). Optokinetic-after-nystagmus was absent in all patients. A typical COR and eye movements during an active head turning are shown in Fig. 2. The characteristics of these eye movements are described below and compared to normal subjects.

Nystagmus Activity

The total *saccadic amplitudes* of passive COR were not significantly different from normal subjects. Only at 0.05 Hz (20° and 60° amplitude) were values slightly higher in the patients (Fig. 3).

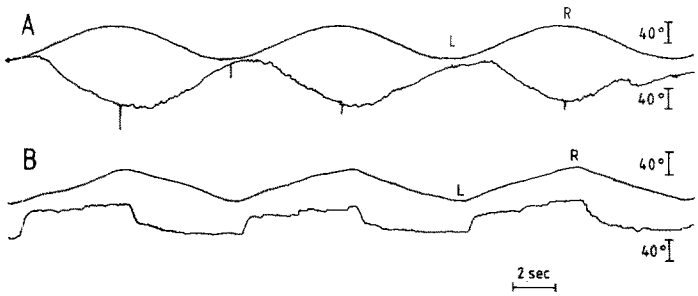

Fig. 2A, B. Eye movements during trunk turning with the head fixed in space (**A**) and with active head movements (**B**). Lower traces represent the horizontal EOG upper traces the trunk movement (**A**) and head movement (**B**)

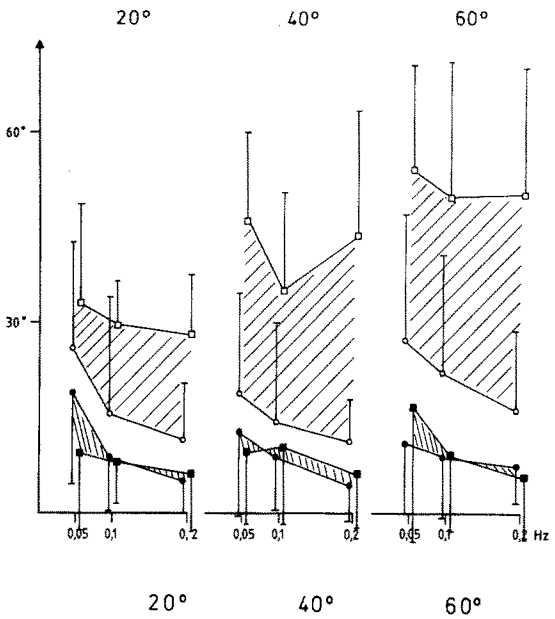

Fig. 3. Total saccadic amplitudes for half a period averaged over five periods with standard deviations during COR *(black symbols)* and active head movement *(open symbols)* in patients with bilateral labyrinthine loss (○) or in normal subjects (□). Hatched area indicates the difference between healthy subjects and patients

During *active head movements* the total saccadic activity increased most for the amplitude of 60°, where the values were almost twice as high as during passive neck stimulation. But patients never reached the very high values of normals. In addition, total saccadic amplitudes during active head movements increased with increasing stimulus amplitude more clearly in normals than in patients (Fig. 3).

The *different gains of nystagmus* (maximum slow phase velocity/maximum stimulus velocity) for different modes of stimulation in comparison to normals resemble the differences found in total saccadic amplitudes, with the exception that in patients higher values for active head movements were only found at 0.05 Hz and 20° to 40° of stimulation (Fig. 4). The gain of the cervically induced nystagmus is above 0.2 at 0.05 Hz.

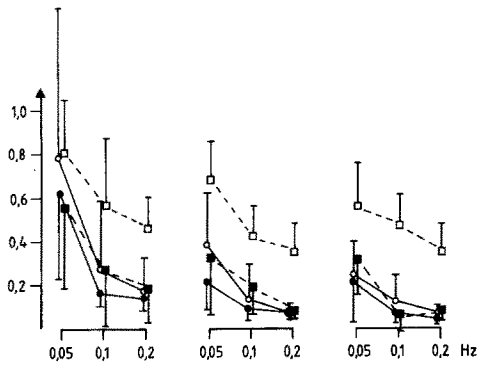

Fig. 4. Gain of nystagmus (maximum slow phase velocity/maximum stimulus velocity). Symbols as in Fig. 3

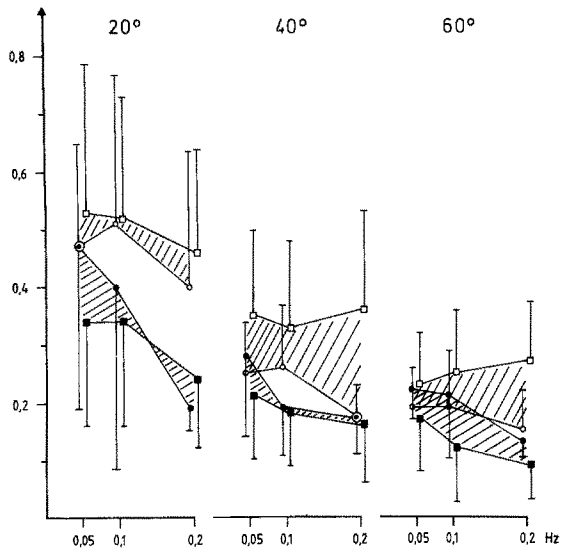

Fig. 5. Gain of overall eye deviations with respect to stimulus amplitude (mean values with standard deviation) in healthy and labyrinthless subjects. Symbols as in Fig. 3

Amplitudes and Phases of Maximum Eye Deviations

The deviations of eye position in passive COR of patients with destroyed labyrinths are not significantly above normal values (Fig. 5). During active head movements they are generally not higher than during COR with the exception of 0.1 and 0.2 Hz at 20° amplitude. Normals show higher values at 40° and 60° but the differences are not so clear as in total saccadic amplitudes (Fig. 5).

The eye deviations are roughly in phase with the relative head position against the turning trunk. But as mentioned earlier (Leopold et al. 1982; Thoden et al. 1983) in some cases the eye shifts compensate for the direction of head movement (Fig. 6) as they do in the VOR.

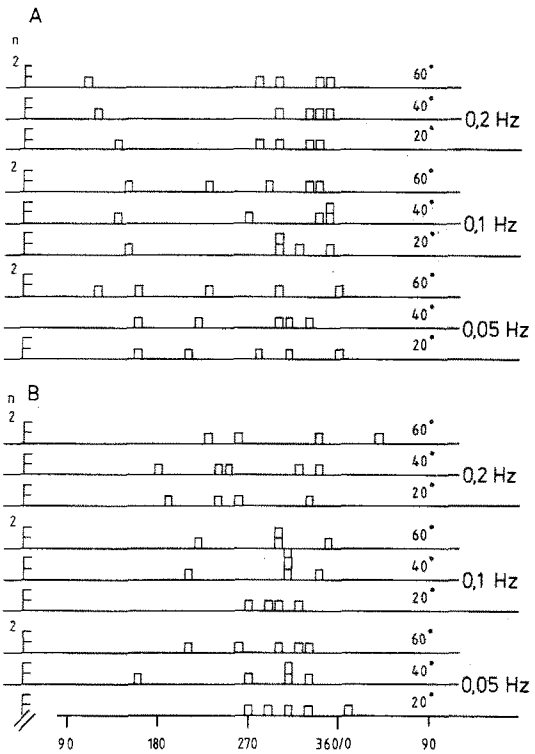

Fig. 6A, B. Distribution of maximum amplitudes of gaze shifts with relation to stimulus position. Averaged values for each patient during active head movements (**A**) and COR (**B**)

Discussion

Under our test conditions, chronic loss of labyrinthine function results in a defect of eye movements during active head turning. Passive cervical reflexes, tested by body torsion with the head held stationary in space, do not compensate for the defect. Accordingly the phases of eye shifts are not altered as compared with normals and show mainly the cervical pattern with the eye shift towards the direction of the relative head movement (against trunk torsion = anticompensatory movement).

In *passive COR* there are no significant differences between normal subjects and patients with complete bilateral loss of labyrinthine function. This unchanged passive COR is in accordance with the data from the literature. One of the two patients with total canal paresis tested by Barnes (1979) showed no measurable COR gain and the other showed a normal response. Also, Kasai and Zee (1978) demonstrated COR gains of a comparable low range in three patients and Atkin and Bender (1968) reported that the observed ocular stabilization responses were "only weakly dependent—if at all—upon proprioceptive feedback from the neck". Only one patient of the three described by Gresty et al. (1977) showed good compensatory vertical eye movements after a passive downward movement of the head with high frequencies up to 5 Hz, but under the same test conditions, also anticompensatory eye movements as mostly seen in our

patients. These findings in humans are in contrast to the results in labyrinthectomized monkeys (Dichgans et al. 1973) which showed an increase in the neck ocular loop during passive head rotation. Besides possible differences due to the species and the duration of labyrinthine loss, the experimental situation may determine the degree of reflex activation and thus explain the controversial findings.

Compared to the passive COR during *active head movements,* total saccadic amplitudes and shifts of eye positions are also enhanced in the patients, but never reach normal values. Smaller differences between the gain of eye shifts in healthy persons and patients with bilateral labyrinthine lesions have been found by Takahashi et al. (1981), who used higher stimulus frequencies (0.33–1 Hz). The high number of saccades and eye shifts in healthy subjects during active head movements are due to an interaction between cervical and vestibular inputs and additional central activating mechanisms for those systems (Thoden et al. 1983). According to our data, there is a considerable amount of central vestibular activation during active head movements.

As discussed first by Frenzel (1928) and demonstrated recently (Barlow and Freedman 1980; Leopold et al. 1982; Takemori and Suzuki 1971; Thoden et al. 1983) the eye deviations of COR are mostly anticompensatory and lead the intended head movement. In contrast, some authors (Meiry 1971; Kasai and Zee 1978) found only eye shifts compensatory for the relative head movement during passive COR. This compensatory oculomotor behaviour has been observed only exceptionally in healthy volunteers (Thoden et al. 1983), patients with Huntington's Chorea (Leopold et al. 1982) and with bilateral labyrinthine loss.

During active head movements the eyes deviate always in the direction of the head motion. In this situation the VOR influences the saccadic activity more than the eye shifts, the latter being more controlled by the cervical input.

The *gain of nystagmus* slow phase velocity usually used as a parameter for the efficacy of compensatory eye movements, is critically influenced by the direction and magnitude of the overall eye deviations. A nystagmus gain of unity would allow fixation of an object fixed in space during head movement, whereas a gain around zero would support following of a target moving with a velocity, which is matched by the head turning.

Our results do not show a compensation for bilateral labyrinthine defect by neck to eye reflexes. A visual compensation may be possible. One of the three patients described by Gresty et al. (1977) and one of ours showed better optokinetic responses than normals; both patients suffered from loss of labyrinthine function since childhood. Our second deaf mute patient, on the contrary, showed a slight impairment of optokinetic nystagmus at 90°/s. Since there is a considerable disturbance of visual-vestibular interaction (Barnes 1979; Collewijn 1981; Uemura et al. 1981) and optokinetic nystagmus and after-nystagmus (Collewijn 1981; Zee et al. 1976) a compensation by visual mechanisms should be an exception. A compensation by changed central programmes for eye-head movements as shown by Dichgans et al. (1973) and Kasai and Zee (1978) cannot be ruled out on the basis of our data. Moreover a possible compensation for labyrinthine loss in higher frequencies of head motion could not be tested, because of the limits of our equipment.

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