25. Electronystagmographic Findings Following Traumatic Apallic Syndrome

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Introduction

For several reasons it appeared to be of interest to carry out electronystagmographic investigations in patients who had displayed a traumatic apallic syndrome. It is known from the clinical signs of this condition that there are functional disturbances in the optovestibular system during the traumatic apallic syndrome. So far, no reports have been published on systematic electronystagmographic examinations in such cases. It also seemed worthwhile to investigate the question of whether or not the electronystagmogram can provide an accessory parameter in evaluating the severity and prognosis of the clinical condition.

The present paper is only a preliminary report, since it is planned to continue the examinations on more patients. Despite the small number of cases studied up to now, some interesting results are already apparent, as will be shown.

Materials and Methods

Seventeen patients who developed an apallic syndrome following a craniocerebral injury were examined in this study. The accidents had occurred 2 months–6 years previously. Except for one girl, all of the patients examined were males. The age distribution ranged from 7 to 38 years. At the time of the electronystagmographic investigation three patients were in the intermediate remission phase, five patients were in the final remission phase, and nine patients had reached the definite defective state. Since electronystagmographic recordings with a Tönnies rotating chair (1932) can only be performed with the patient in a sitting position, it was not possible to carry out such examinations during the acute stage of the apallic syndrome.

With regard to the clinical signs of brain stem disturbances present at the time of these examinations, the cases were divided into three groups: cases with slight, moderate, or severe clinical signs. Similarly, the electronystagmographic abnormalities were assessed as slight, moderate, or severe. It is true that these classifications are somewhat subjective; however, errors in the results are not expected, since the clinical and electronystagmographic findings were always classified by the same investigator for each field. Moreover, the degree of severity of the electronystagmographic abnormalities was first assessed without knowledge of the clinical state.

The electronystagmographic examinations were carried out as described by R. Jung (1953). Here it proves useful to distinguish three different parts in the optovestibular investigation: (1) The search for spontaneous oculomotor phenomena, with the eyes open and closed, (2) the registration of optokinetic nystagmus by means of an optic pattern moving in the four different cardinal directions, and (3) the registration of vestibular nystagmus produced by the stimulus of rotation to the left and right.

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Both spontaneous and experimental (optokinetic and labyrinthine) eye movements were recorded continuously as changes of the periorbital electric field in its vertical and horizontal planes. The optokinetic stimulation was achieved by means of parallel black and white stripes projected onto an arched (concave) screen; the vestibular stimulation was produced by rotating the patient at a constant acceleration slightly above threshold until the final velocity of 90°/s was reached, and later by stopping the patient's rotation abruptly (deceleration = negative acceleration).

Results

1. Of the 17 patients examined who had undergone a traumatic apallic syndrome, seven showed spontaneous pathologic phenomena. Two cases showed gross alternating deviations of the eyes, especially when the lids were shut, combined with ocular dysmetria. In two other gross fixational eye movements and ocular dysmetria were found without the occurrence of alternating deviations. Spontaneous nystagmus with the eyes closed was seen in three patients, swinging horizontally in two cases, and swinging upward in one case. In one patient who also showed ocular dysmetria, the vehement vestibular stimulus caused by the abrupt stopping of the rotation chair brought forth a transitory provocational nystagmus swinging upward, a phenomenon that subsided after a short while.

Slow horizontal pendular deviations of the eyes, with the lids shut, like the eye movements seen in healthy persons during fatigue and drowsiness, were registered rather frequently, viz., in nearly half the cases (8 patients). One of these showed repeated periods of slow high-amplitude pendular eye movements alternating with periods of rapid low-amplitude to-and-fro eye movements (so-called *Gegenrucke*).

2. Abnormalities of the optokinetic nystagmus were present in nine out of the 17 injured persons examined. In three cases the disturbances were seen in only one direction and in two cases they were seen in both directions in the horizontal plane. One patient displayed optokinetic abnormalities in the vertical plane, especially in the downward, but also to a slighter degree in the upward direction. Three patients had omnidirectional optokinetic disturbances which were very strongly marked. As for the severity of optokinetic abnormalities, all possible gradations—from moderate dimunition of the expected physiologic response to complete decomposition of nystagmus—were encountered.

3. Out of the 17 patients examined, 12 showed disturbances of the vestibular nystagmus when examined on the rotating chair. The most frequent abnormality of this kind was vestibular directional preponderance (6 patients). In three injured persons, considerable hypoexcitability, in two inexcitability, and in one hyperexcitability of the vestibular system were found.

The electronystagmographic findings in the above-mentioned different parts of the investigation (1-3) were established separately in each patient. On the basis of these findings, an overall evaluation was made with regard to the absence or presence of functional disturbances of the optovestibular system. In the latter cases the quantitative aspect of such abnormalities was also taken into consideration. Thus the overall electronystagmographic evaluations were classified into four different groups: normal, slightly abnormal, moderately abnormal, and severely abnormal. All 17 cases examined are incorporated in Table 1 according to the severity of the clinical signs (disturbance of brain stem functions), recovery

Table 1. Survey of 17 patients who have undergone electronystagmographic examinations following traumatic apallic syndrome

Clinical recovery phase	Clinical brain stem signs slight	moderate	severe	Number of cases
Intermediate remission phase	+++		+++ +++	3
Final remission phase	+ + ++ +++		+++	5
Defective phase	N N N + + + +	++ ++		9
Number of cases	12	2	3	17

N = Normal electronystagmogram
\+ = Slightly abnormal electronystagmogram
++ = Moderately abnormal electronystagmogram
+++ = Severely abnormal electronystagmogram

In the intermediate remission phase the electronystagmogram was severely abnormal in all three patients. In the final remission phase the electronystagmographic findings were abnormal in all five patients of this group. However, they were highly abnormal in only two cases, whereas the abnormalities were considered to be of a slight degree in two patients and of a moderate degree in one patient. In the defective state there were two moderately abnormal, four slightly abnormal,and three normal electronystagmographic recordings.

Among the 12 patients with slight clinical signs of brain stem dysfunction, three had a normal, six had a slightly abnormal, one had a moderately abnormal, and two had a severely abnormal electronystagmogram. Both cases with clinical brain stem signs of a moderate degree were in the defective stage and showed moderate electronystagmographic disturbances. The three patients with severe clinical brain stem signs had highly pathologic electronystagmograms and were in the intermediate or final remission phase.

Discussion

Abnormalities of the electronystagmogram can be interpreted and assessed only in the light of the entire clinical picture, particularly taking into consideration the otiologic findings. For instance, vestibular directional preponderance, a rather frequent abnormality, can be caused by central or peripheral lesions. Vestibular hyperexcitability and optokinetic disturbances, however, are always of central origin. But even in these cases it is not always clear from the beginning whether the disorder arises from a brain stem lesion or not. Optokinetic abnormalities are related to hemispheric or brain stem damage. In hemispheric lesions,

though, there is a more rapid recovery of such disturbances. Long-standing optokinetic abnormalities are therefore always cause for suspecting lesions in brain stem structures. Only exceptionally is a given electronystagmographic disorder related to a lesion in a relatively small circumscribed area, e.g., vestibular hyperexcitability (location of lesion in the vicinity of the vestibular nuclei, i.e., part of the brain stem and cerebellum). In addition, physiologic phenomena may appear at an augmented rate. In the material studied this was particularly the case for slow horizontal pendular movements of the eyes, which must be considered as an expression of increased fatigability in posttraumatic conditions (Jung, 1953; Kornhuber, 1966; Scherzer, 1968). Successive periods of slow pendular deviations and rapid low-amplitude to-and-fro movements of the eyes are a special combination of different physiologic signs, which, in this constellation, are an important clue to considerable fluctuations in the patient's state of vigilance (Scherzer, 1968).

From what has been said, it is obviously essential that the electronystagmographic findings be integrated sensibly into the entire clinical picture. Not observing this exigency may easily give rise to incorrect interpretations. As can be gathered from Table 1, the electronystagmographic records of our patients in the intermediate remission phase were still severely pathologic. Normal findings were seen only in the defective state. Slight abnormal tracings were encountered first of all in the defective state, but sometimes even towards the end of the remission phase. Moderate pathologic findings could be seen both during the last period of the remission phase and during the definite defective state.

As for the quantitative relationship of clinical and electronystagmographic disorders, cases with pronounced clinical brain stem signs showed severely pathologic electronystagmograms. Patients with only slight clinical brain stem signs, on the other hand, had a diversity of electronystagmographic findings, whereby a correlation existed between the degree of electronystagmographic abnormality and the clinical phase in which the patient was at the time of the investigation.

Despite the small number of cases in our material, which does not allow for statistical evaluation, the trend of the electronystagmographic findings toward two dependences can be recognized: (1) dependence on the clinical recovery phase after the traumatic apallic syndrome; (2) dependence on the degree of severity of the brain stem signs as evidenced clinically without the aid of electronystagmographic examinations. Hence, it can be stated that the amount and extent of electronystagmographic disturbances are first dependent on the time factor (in the intermediate remission phase only severely pathologic tracings were found) and later, i.e., during the definite defective state, upon the clinical picture of the brain stem signs. However, toward the end of the remission phase, which is situated between the two above-mentioned stages, an interaction of (1) and (2) seems to exist.

Longitudinal surveys in individual cases (Scherzer, 1968) which do not belong to the material of this study, provide good support for these assumptions. Yet, for the actual scientific proof of the cited correlations, many more electronystagmographic examinations of patients who have passed through the clinical condition of traumatic apallic syndrome will be necessary. Only then can a definite answer be given on the value of electronystagmographic registrations in such cases.

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