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Optokinetic Nystagmus in Homonymous Hemianopia due to a Strictly Occipital Lesion

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Summary. Optokinetic nystagmus (OKN) was tested in 15 patients with unilateral strictly occipital brain lesions and complete homonymous hemianopia and compared with findings in a group of normal controls. Quantitative analysis of the results in the patients revealed a bilateral disorder of OKN. When the stimulus was moved in the direction opposite to the hemianopic field, there was a significant decrease in optokinetic gain, and the amplitude of nystagmus decreased. There was no correlation between OKN and size of the residual macular field.

Key words: Optokinetic nystagmus – Homonymous hemianopia – Occipital lesion

Introduction

In 1921 Barany published a first description of two patients with homonymous hemianopia who failed to demonstrate horizontal optokinetic nystagmus (OKN) when a visual stimulus was moved in the direction opposite to the hemianopic field. He believed that the visual field defect itself was responsible for this finding and considered it to be an objective criterion of hemianopia. However, there were simultaneous reports of patients with homonymous hemianopia and normal OKN to both sides (Ohm 1922). In addition, differences in OKN were also found in brain lesions without associated visual field defects (Stenvers 1924), and Cords (1926) suspected a lesion in a centrifugal pathway leading to the mesencephalic centre of conjugate eye movements to be the cause of this disorder. Subsequent publications cast doubt on the influence of the visual field defect on OKN, since pathological differences in OKN were not found in strictly occipital brain lesions (Levenson and Smith 1966; Smith and Cogan 1959), with the exceptions of the antero-medial region (Kjällman and Frisen 1986). Table 1 summarizes the various findings in this disorder.

A number of different studies of OKN in test subjects with artificial hemianopia revealed that there was a tendency of the slow phase to favour foveopetal movement of the stimulus (van Die and Collewijn 1982, 1986). The simulated visual field defects in these studies were not equivalent to actual clinical conditions in regard to foveal and macular function, but the study did document the fact that the visual field defect provoked asymmetry in OKN in the absence of a brain lesion. The purpose of our study was to clarify the extent to which homonymous hemianopia caused by an occipital lobe lesion might affect

Table 1. Optokinetic nystagmus (OKN) in unilateral occipital lobe lesions with homonymous hemianopia. Only reports with exact data on the number of patients studied are included

Reference	Number of patients	OKN		
		Normal	Changed	Missing
Ohm (1932)	3	1	2	–
Lenz (1941)	1	–	1	–
Kestenbaum (1946)	10	8	2	–
Carmichael et al. (1954)	2	–	2	–
Smith and Cogan (1959)	5	5	–	–
Smith (1963)	35	29	6	–
Gassel and Williams (1963)	2	2	–	–
Levenson and Smith (1966)	4	4	–	–
Jung and Kornhuber (1969)	1	1	–	–
Kömpf (1986)	3	3	–	–
Total	66	53	13	–

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OKN. Special attention was paid to the size of the residual macular visual field.

Material and Methods

Studies were performed in 15 patients with unilateral occipital lobe lesion (Table 2). CT studies documented the location of the lesion and excluded additional space-occupying effects on adjacent structures due to oedema. In Fig. 1a all lesions – eight on the right and seven on the left occipital lobe – are projected onto the left hemisphere. CT scans were evaluated with a grid in order to localize the predominant area of the lesions (Fig. 1b). Twelve patients had suffered brain infarction, 2 had undergone resection of the occipital lobe (for oligodendroglioma in one case and haemangioma in the other), while the last patient had an inoperable haemangioma. Complete homonymous hemianopia was demonstrated by kinetic perimetry in all patients. Hemianopia to the right side was found in 7 cases and to the left in the remaining 8 patients. The size of the macular defect was determined on the tangent screen and ranged from 2° to 10°. In all cases OKN studies were performed over 1 month after the onset of hemianopia. Ocular disorders were excluded in all patients, none of whom took medication affecting the central nervous system.

Table 2. Clinical data on the 15 patients enrolled in the present study

Patient	Sex	Age (years)	Homonymous hemianopia	Macular sparing	Aetiology
S.M.	F	23	Right	2.0°	Tumour/ surgery
T.C.	M	23	Left	2.0°	Stroke
J.H.	M	65	Left	2.0°	Stroke
L.A.	M	78	Left	2.5°	Stroke
K.F.	F	68	Right	2.5°	Stroke
R.K.	M	81	Left	2.5°	Stroke
S.J.	M	54	Left	3.5°	Stroke
F.W.	M	68	Left	5.0°	Stroke
H.H.	M	70	Right	5.0°	Stroke
B.I.	F	60	Left	5.0°	Stroke
N.A.	M	35	Right	6.0°	Angioma/ surgery
W.M.	M	62	Right	6.0°	Stroke
B.N.	M	50	Right	8.0°	Stroke
D.G.	F	39	Left	10.0°	Stroke
F.I.	F	29	Right	10.0°	Angioma

OKN was tested by full-field stimulation. The subject sat at the centre of a semi-circular screen with a homogeneous white surface. Fifteen minutes were allowed for adaptation before testing was begun. There were no changes in light intensity during the test procedure. Optokinetic responses were recorded with an AC electronystagmograph (time constant 2 s). The width of the stripe was 7.5°, and the light density of the white stripe was 29 cd/m^2, while that of the black stripe was 6.9 cd/m^2, which yields a contrast of 0.615. The patients were requested to look straight ahead and to observe the stimulus without following a given stripe. Constant stimulus pattern velocities of 20°, 30° and 50°/s were used, corresponding to stimulus frequencies of 1.33, 2.0 and 3.3 Hz, respectively. Each examination lasted 30 s. Calibration was performed every 2 min. The results were compared with those from a similar healthy control group. Neither the patients nor the subjects in the control group had taken substances affecting the central nervous system.

The frequency and amplitude of nystagmus as well as the velocity of the slow phases (SPV) were evaluated. The 15 most rapid nystagmus beats were analysed quantitatively. Opto-

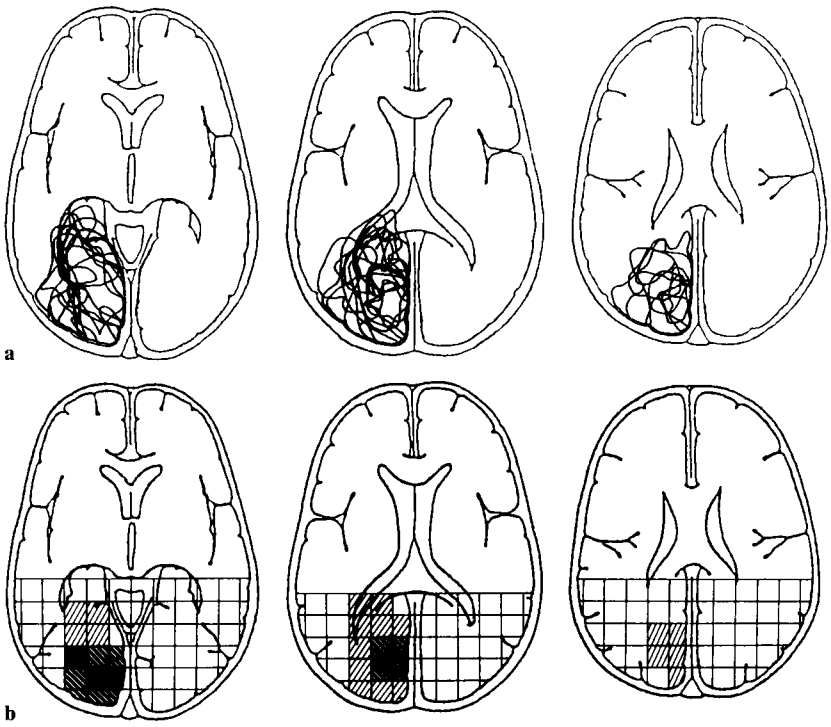

Fig. 1a. Findings of CT scans from all patients projected onto the left hemisphere. (**b**) Brain fields which are involved in more than 50% (▨), 75% (▧), or in 100% (■) of patients

kinetic gain was calculated as the quotient of the SPV and the velocity of the stimulus patterns.

Results

Three different pathological patterns of OKN were found among the 15 patients when the stimulus pattern was moved in the direction opposite to the hemianopic field.

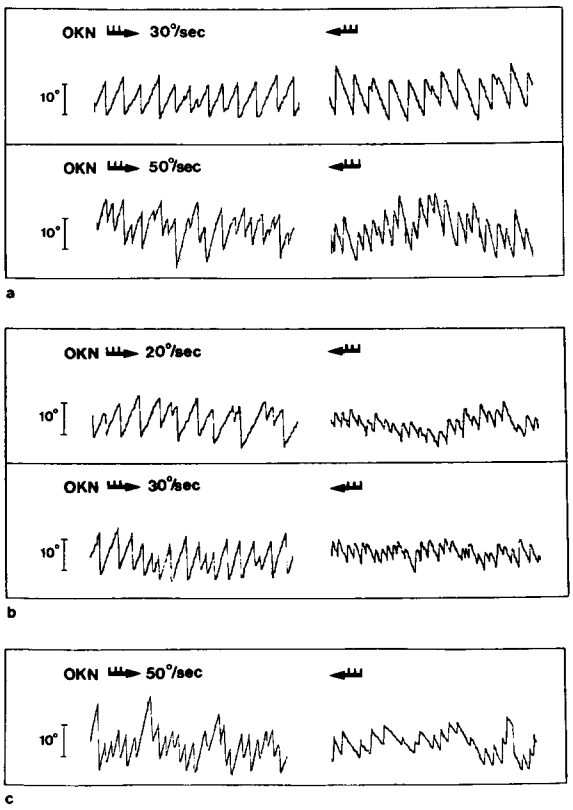

Fig. 2a. Patient P8. Homonymous left hemianopia with 5° of residual macular vision. Slight disorder of OKN when the stimulus was moved to the right. (**b**) Patient P11. Homonymous right hemianopia with 6° of residual macular vision. Evident disorder of OKN when the stimulus was moved to the left. (**c**) Patient P1. Homonymous right hemianopia with 2° of residual macular vision. Severe disorder of OKN when the stimulus was moved to the left

1. The first characteristic pattern was found in 7 patients and involved a moderate reduction in amplitude, variation in amplitude, a reduction in the SPV and a slight disruption of eye movement, with some stripes being skipped over. Slight disorders were demonstrated best when the pattern was moved at 30° and 50°/s (Fig. 2a).

2. There were 6 patients who showed evident reductions in amplitude, and the nystagmus frequency was higher than the stimulation frequency, which resulted in loss of coordination between the nystagmus and the stimulus. These changes were particularly apparent at rates of 20°–30°/s (Fig. 2b). There was no apparent relationship between these findings and the age of the patient or the size of the residual macular field.

3. Two patients demonstrated a severe disorder of optokinetic response. There were significant reductions in amplitude and in the velocity of the slow phase of nystagmus (Fig. 2c).

Interindividual comparisons were made with the help of the unpaired *t*-test. Figure 3a presents mean sample values of patient nystagmus when the stimulus pattern was moved out of the hemianopic field as compared with findings in the control group. The differences in mean sample values between the two groups were highly significant ($P < 0.001$). Comparison of the SPV in the control group with mean values from the patient group when the stimulus pattern was moved out of the intact visual field (Fig. 3b) revealed significant differences between the two groups ($P < 0.05$) at stimulus velocities of 30° and 50°/s.

Discussion

A disorder of OKN associated with a lesion in the contra-lateral white matter of the parietal lobe has

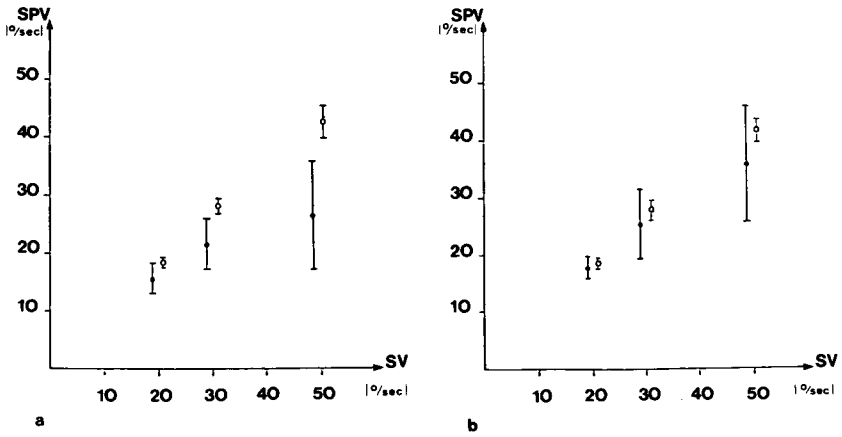

Fig. 3a. Slow phase velocity (SPV) ± 1 SD *(vertical bars)* at different stimulus velocities *(SV)*. Patient group (•): stimulus moved in the direction opposite to the hemianopic field. Control group (○): corresponding nystagmus. (**3b**) Slow phase velocity (SPV) ± 1 SD *(vertical bars)* at different stimulus velocities *(SV)*. Patient group (•): stimulus direction into the hemianopic field. Control group (○): corresponding nystagmus

been described in many patients (Baloh et al. 1980; Cogan and Loeb 1947; Kjällman and Frisen 1986). The role of the occipital lobe has been a subject of controversy. Kömpf (1986) found no changes in this region, while Kjällman and Frisen (1986) found disorders of OKN in lesions affecting the antero-medial occipital lobe.

Our study was designed to allow quantitative analysis of the phases of nystagmus and demonstrated a reduction in the SPV when the stimulus was moved in the direction opposite to the hemianopic field in all patients with strictly occipital lesions and complete homonymous hemianopia (contralateral OKN). In addition, we found qualitative changes in OKN, such as skipping of stripes in the stimulus pattern, and variation in amplitude. There was no statistically significant correlation between changes in OKN and the size of the residual macular field. Comparison with a control group of similar age demonstrated also a reduction in the SPV of nystagmus when the stimulus moved into the hemianopic field (ipsilateral OKN).

Our findings do not conform to the classic theory, which postulates deep parietal lobe lesions in asymmetry of OKN (Smith 1963). Baloh et al. (1980) and Yee et al. (1982) suggested a possible course of the optokinetic tract which conforms to our findings. Their suggestion is derived from the "slow component" hypothesis proposed by Cords (1926). The authors maintained that OKN may be caused by lesions in the centrifugal tracts in the parietal lobe and by lesions at the parieto-occipital junction, where afferent impulses enter and efferent impulses originate. With regard to smooth eye movements in monkeys Tuna and Ungerleider (1988) demonstrated – in agreement with our findings – the localization of a cortico-cortical pathway within one hemisphere, consisting of arcuate fibre bundles and connecting striate cortex with areas in occipito-parietal and parietal cortex.

In addition Tuna and Ungerleider (1988) demonstrated an interhemispheric pathway with fibers passing through the tapetum, major forceps and the splenium of the corpus callosum. Within the smooth pursuit system each hemisphere obtains information from contralateral areas. In fact, both occipital lobes are involved in right and left nystagmus through their commissural fibres. Our findings, similar to those described by Kaga and Suzuki (1982), confirm this interaction, since a unilateral occipital lesion caused a disorder of ipsilateral OKN, though the findings were not as pronounced as in contralateral OKN.

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