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Impaired Suppression of Vestibular Nystagmus by Fixation in Cerebellar and Noncerebellar Patients*

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Summary. The suppression of vestibular nystagmus (VN) by fixation of a small visual target moving with the observer was tested while subjects seated on a rotatable chair were oscillated at 0.1 Hz and peak accelerations of ± 10 to 130°/s^2. Total amplitudes of nystagmus during movement towards one direction occurring despite intended fixation were compared to slow phases of optokinetic nystagmus (OKN) and smooth pursuit (SP) towards the opposite direction.

Results

1. Normals completely suppress VN up to peak accelerations of 50°/s^2.

2. Deficits in fixation suppression are only found if OKN and SP are impaired. They are always counter in direction to impaired SP, but quantitative correlations between the two are not very strict.

3. Deficiencies of OKN, SP, and fixation suppression, partially due to the occasional vestibular hyperexcitability, are most prominent in patients with cerebellar lesions but also occur with hemisphere and brainstem lesions.

4. Fixation suppression of VN is diminished towards the ipsilateral direction in patients with hemisphere and cerebellar lesions and towards the contralateral in patients with brainstem damage.

5. A simple bedside test is proposed to demonstrate this symptom which is considered to be valuable for clinical diagnosis in neurology.

Key words: Fixation suppression of VOR – Optokinetic nystagmus – Cerebellum.

Zusammenfassung. Es wurde die Suppression des vestibulären Nystagmus durch Fixation während sinusförmiger Körperdrehung um die vertikale

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Achse bei einer Frequenz von 0,1 Hz und einer Maximalbeschleunigung von ± 10 bis $130°/s^2$ untersucht. Die Gesamtamplitude des vestibulären Nystagmus in einer Richtung während des Versuches der Unterdrückung durch Fixation wurde mit der Güte der langsamen Phasen des optokinetischen Nystagmus, gemessen an deren Gesamtamplitude, und der Güte von Folgebewegungen, gemessen an der Gesamtamplitude von eingestreuten Saccaden, verglichen.

Ergebnisse

1. Gesunde können den vestibulären Nystagmus bis zu Maximalbeschleunigungen von $50°/s^2$ komplett unterdrücken.

2. Störungen der Fixationssuppression finden sich nur, wenn auch der optokinetische Nystagmus und die Folgebewegung gestört sind. Sie bestehen dann immer in Gegenrichtung der gestörten Folgebewegung und der Störung der langsamen Phasen des optokinetischen Nystagmus. Aber die quantitativen Entsprechungen sind ungenau.

3. Störungen des optokinetischen Nystagmus und der Folgebewegung sowie der Fixationssuppression sind am deutlichsten bei Patienten mit Kleinhirnerkrankungen, kommen aber auch bei Patienten mit Hemisphären- und Hirnstammläsionen vor.

4. Die Fixationssuppression des vestibulären Nystagmus ist bei Patienten mit Läsionen der Großhirnhemisphäre oder des Kleinhirns nach ipsilateral gestört, bei Patienten mit Hirnstammläsionen nach kontralateral.

5. Es wird ein einfacher und diagnostisch hilfreicher Test zur Untersuchung am Krankenbett beschrieben.

Schlüsselwörter: Fixationssuppression des vestibulären Nystagmus – Optokinetischer Nystagmus – Cerebellum.

Introduction

Vestibulo-ocular reflexes (VOR) in moving animals and man subserve the spatial stabilization of gaze directed towards stationary targets within the visual surround. This phyllogenetically very old, rather direct, and simply designed brainstem reflex must be suppressed whenever a visual target moves with the observer. This happens in vehicles or when the vestibular system is stimulated by the sudden breaking of a constant velocity movement and the subject is looking at the stationary visual surround.

From anatomic connections and physiologic experiments, Ito (1972), Ito et al. (1974), Fukuda et al. (1972), Melvill Jones and Gonshor (1975), and Robinson (1975) developed the concept of a cerebellar control of the vestibular-ocular reflex which they located in the flocculus. Takemori and Cohen (1974) showed more specifically that fixation suppression of VOR in monkeys is severely impaired after flocculectomy. In addition, their animals also exhibited a marked reduction of optokinetic nystagmus (OKN) slow-phase velocity at higher pattern speeds.

The clinician seeking a possible application of these basic physiologic findings will be interested in two questions: first, is the ability to suppress VOR by fixation specific to the cerebellum and can its impairment therefore be used for topographic diagnosis? Second, is the deficient fixation suppression an independent symptom or is there a causal relationship to the impairment of smooth pursuit (SP) and OKN? The causal relationship may conceivably exist if VOR are normally suppressed or cancelled by optokinetic output signals.

To assess the usefulness of a fixation suppression test we collected data from three groups of subjects:

1. patients with cerebellar lesions, mainly atrophies and tumors;
2. patients without known cerebellar lesions, but with disturbances of OKN and/or SP, and
3. normal subjects serving as controls.

To answer the above questions VOR, OKN, SP, and fixation suppression of VOR were quantitatively determined.

Methods and Patients

Stimulation and Recordings. Eye movements were electronystagmographically recorded according to Jung (1953). Electrodes were placed bilaterally over the outer canthus of each eye to record the horizontal component of eye movements. This component only was processed for the purpose of this paper. The vertical component was also monitored to detect lid artifacts. Most of the recordings were done with AC coupling (time constant = 3 s).

Fixation targets and moving optokinetic patterns (consisting of black and white stripes, 7.5° in width) were projected onto a semicircular screen 80 cm in front of the subject. Optokinetic stimuli covered a visual angle of 140° horizontally and 100° vertically and moved at a constant velocity of 60°/s. Smooth pursuit was elicited by pendular oscillation (0.3 Hz, 60° peak to peak amplitude) of a small target. Subjects sat on a rotatable chair (Tönnies, Freiburg, FRG), the axis of which was aligned with the center of the projection screen. The head was supported by a head rest. For vestibular stimulation the chair was motor-driven according to a servocontrolled velocity profile which was either trapezoidal or sinusoidal.

Fixation suppression was tested while the rotatable chair was sinusoidally oscillated about the vertical body axis at a frequency of 0.1 Hz. Peak acceleration was varied between $\pm 10°$ and $\pm 130°/s^2$. The corresponding peak to peak amplitude of body oscillation ranged between 51° and 659°. A luminous fixation target, rigidly attached to the chair, was placed 55 cm in front of the subject's head at eye level. In normal subjects, the test was first applied within an illuminated environment and then in an otherwise dark room, so as to assess any possible influences of optokinetic stimulation of the retinal periphery as elicited by body motion relative to the stationary surround. After this had been found to cause no difference in fixation suppression in normal subjects, patients were tested in the illuminated environment only.

Another method to determine fixation suppression of VOR is to compare the total amplitude and duration of postrotatory nystagmus with eyes closed and with fixation of the stationary surroundings in the light. This was tested in normal subjects, after a 90°/s constant velocity rotation was suddenly stopped. The second test was not systematically used in patients after variability in normal subjects had been proven to be much higher than with the pendular body oscillation test.

Measurements. Efficiency of fixation suppression was assessed by measuring the total amplitude of fast phases of nystagmus over each half period of sinusoidal body oscillation, i.e., the total duration of acceleration to right and left, respectively. Two measurements were taken for each

direction and then averaged. With the postrotatory fixation suppression test, total amplitudes and durations were compared for the two conditions—eyes open and eyes closed. OKN was measured by summing fast phases over 10 s for each direction of horizontal pattern motion. To assess the inadequacy of SP, the total amplitudes of saccades occurring during each half period of sinusoidal target motion were determined three times for each direction and then averaged.

Cerebellar Patients

This group consisted of 22 patients with exclusive or at least predominant cerebellar symptomatology: seven with slowly progressing cortical cerebellar atrophies either due to alcoholism (patients 4, 7, 9, 13 and 15)[1] or of unknown origin (patients 6 and 18); three with large pontine angle tumors with considerable cerebellar symptomatology (patients 1, 10 and 12); six with cerebellar tumors (patients 2, 3, 5, 16, 17 and 22); two with recurrent cerebellar strokes (patients 8 and 11); one with Wilson's disease (patient 20); one with severe multiple sclerosis mainly involving the cerebellum (patient 19); one with severe cerebellar dysfunction after septic embolization (Klebsiella) complicating a M. Crohn (patient 14); and one with lympholytic meningoencephalitis mainly involving the cerebellum (patient 21).

Non-Cerebellar Patients

This group consists of 11 patients in whom signs of a cerebellar lesion could not be found, although in some cases (patients 4, 5, 6, 10) such a lesion could not be absolutely excluded. Patients within this group were selected on the basis of their diminished OKN. The group comprises two patients with a unilateral stroke of one medial cerebral artery (patients 1 and 2), two with strokes involving the posterior cerebral artery (patients 3 and 7), one with a precentroparietal glioblastoma (patient 8), two with an acute pontomesencephalic demyelinization due to multiple sclerosis (patients 5 and 6), and one with recurrent and partially lasting symptoms of spinal and brainstem demyelinization due to multiple sclerosis (patient 4). One patient was admitted with a lateral pontine syndrome (homolateral palsy, contralateral hemihypaesthesis) due to a metastasis of a malignant melanoma (patient 10), and one patient with a severe unilateral OKN and SP deficit of unknown origin (patient 11). All of these patients were carefully studied by clinical testing and neuroradiologic methods including CAT, if possible, to exclude cerebellar lesions in as far a possible.

Results

Fixation Suppression in Normal Subjects. Ten normal subjects ranging between 23 and 34 years of age were tested for comparison with our patients. As may be seen from Table 1 and Figure 1, fixation suppression was very effective in normal subjects. With the body oscillation test, suppression of vestibular nystagmus was invariably complete up to peak accelerations of $50°/s^2$ at our test frequency of 0.1 Hz. Only six of the ten subjects showed a very slight fixation suppression deficit at $70°/s^2$ peak acceleration and even at $110°/s^2$, which was the highest value systematically tested, the total amplitude of nystagmus per half oscillation averaged only 15°, which amounts to 2.7% of the total body oscillation amplitude (560°). As may also be seen from Table 1, the relative movement of the actually stationary surround in an illuminated environment does not significantly in-

[1] The numbers identifying patients are also given in the figures

Table 1. Fixation suppression in ten normal subjects

Peak acceleration of sinusoidal rotation		Total amplitude of nystagmus per one-half oscillation (degrees)	
		in light	in dark
		$\bar{x}$ ±SD	$\bar{x}$ ±SD
50°/s² (peak to peak amplitude 253°)	Right	0.64 —	— —
	Left	0.53 —	— —
70°/s² (peak to peak amplitude 355°)	Right	2.25±2.08	2.40± 3.70
	Left	2.18±2.11	3.02± 4.67
90°/s² (peak to peak amplitude 456°)	Right	5.99±3.15	5.98± 5.04
	Left	6.52±4.39	4.57± 3.53
110°/s² (peak to peak amplitude 560°)	Right	15.63±7.85	15.22±12.81
	Left	15.09±6.50	10.56± 6.69

fluence fixation suppression (Wilcoxon test). In normal subjects, foveal fixation apparently totally overrides peripheral retinal motion information. The standard deviations are sufficiently low to warrant the use of this test. Table 2 indicates that postrotatory nystagmus is less useful as a test of fixation suppression because of its large variability. Both normal subjects and patients show a considerable variability in total amplitude and total duration.

Fixation Suppression in Cerebellar Patients. Figure 1 summarizes the results obtained with the pendular body oscillation test in cerebellar patients and normal subjects. A comparison of the data from both groups shows a considerable deficit in fixation suppression in the vast majority of cerebellar patients. Only three of them fall within the normal range: one had cerebellar cortical atrophy (patient 7); one had been recently operated for a large medulloblastoma (patient 16); and the third had been operated for an angioblastoma in the right posterior cerebellar hemisphere (patient 17). Whereas in normal subjects fixation suppression is practically complete up to peak accelerations of 70°/s², cerebellar patients reach an average total amplitude of vestibular nystagmus of 75° (12.5% of body oscillation amplitude) at this stimulus intensity. The total amplitude of nystagmus at each stimulus intensity varied greatly among patients. The four subgroups—cortical cerebellar atrophies, cerebellar tumors, pontine angle tumors, and other cerebellar lesions—did not differ significantly in nystagmus intensity.

Most of the cerebellar patients showed a deficit in OKN as well. This was frequently in both horizontal and vertical directions. Whenever a lesion was unilateral, the OKN with fast phases to the contralateral side was predominantly impaired. Correspondingly, SP was mainly cogwheeled towards the ipsilateral side. The deficit in OKN and SP is illustrated in Figure 2: the recordings are from a patient from whom a large and partially cystic astrocytoma was removed. This tumor involved the corpus restiforme and the inferior semilunar and biventer

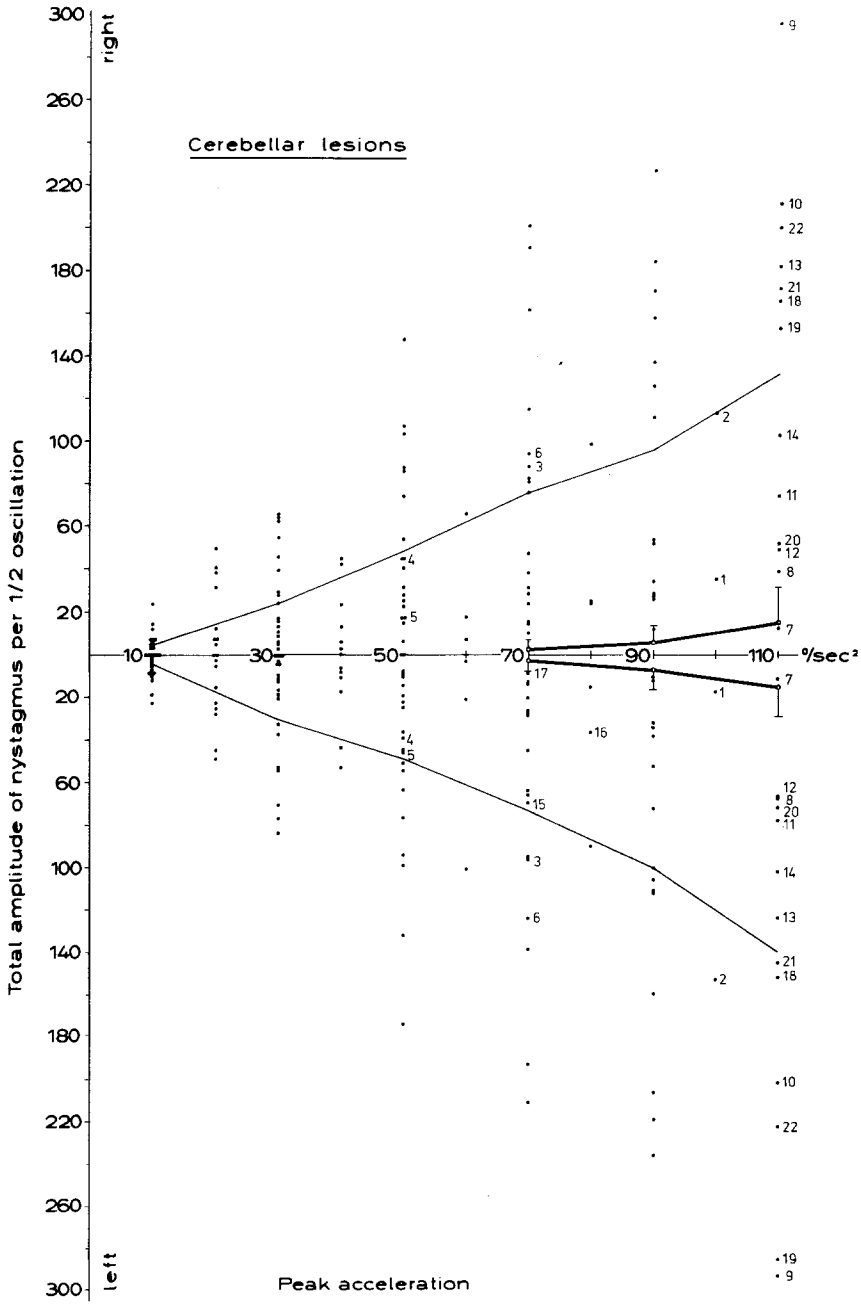


Fig. 1. Fixation suppression in cerebellar patients. Peak accelerations of sinusoidal body oscillations are plotted on the abscissa and total amplitudes of nystagmus—despite the attempt to fixate—on the ordinate. Individual data from patients are represented by single dots; the average data by thin lines. Average data from normals with their doubled standard deviations are represented by heavy lines. The small numbers on the right represent the 22 patients

Table 2. First postrotatory nystagmus (PI)

	In light	With eyes closed
	$\bar{x}$ ±SD	$\bar{x}$ ±SD
Total amplitude (degrees)	27.37 ± 27.81	667.58 ± 430.45
Total duration (s)	6.95 ± 7.17	44.81 ± 12.3

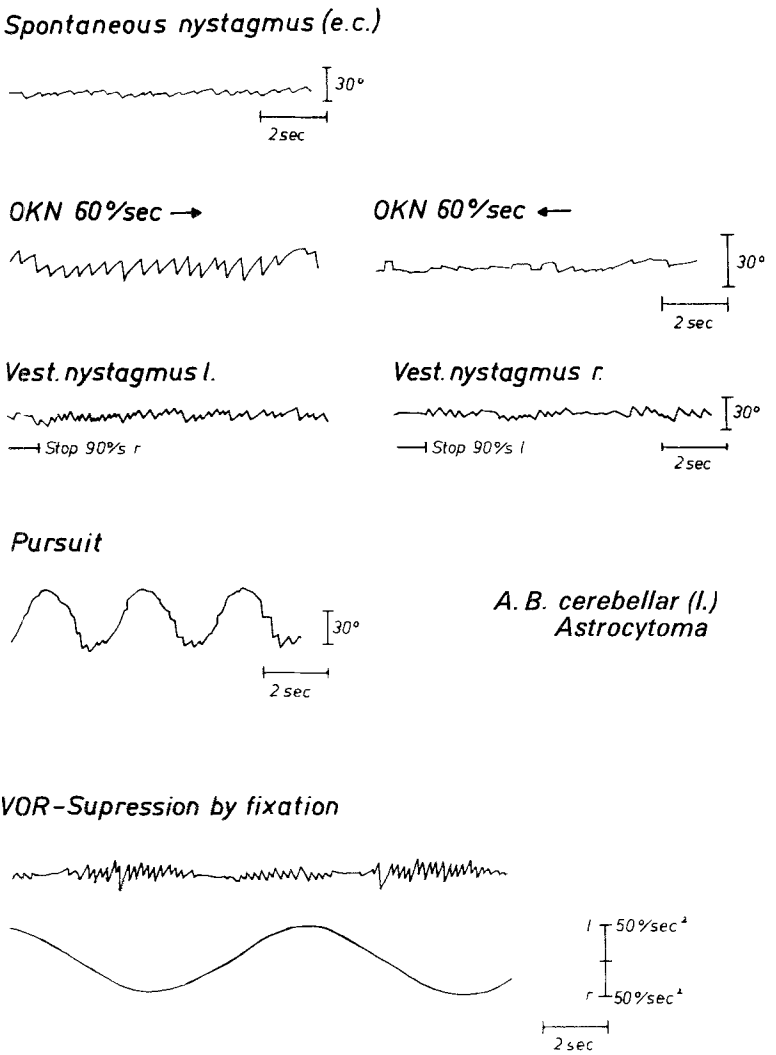
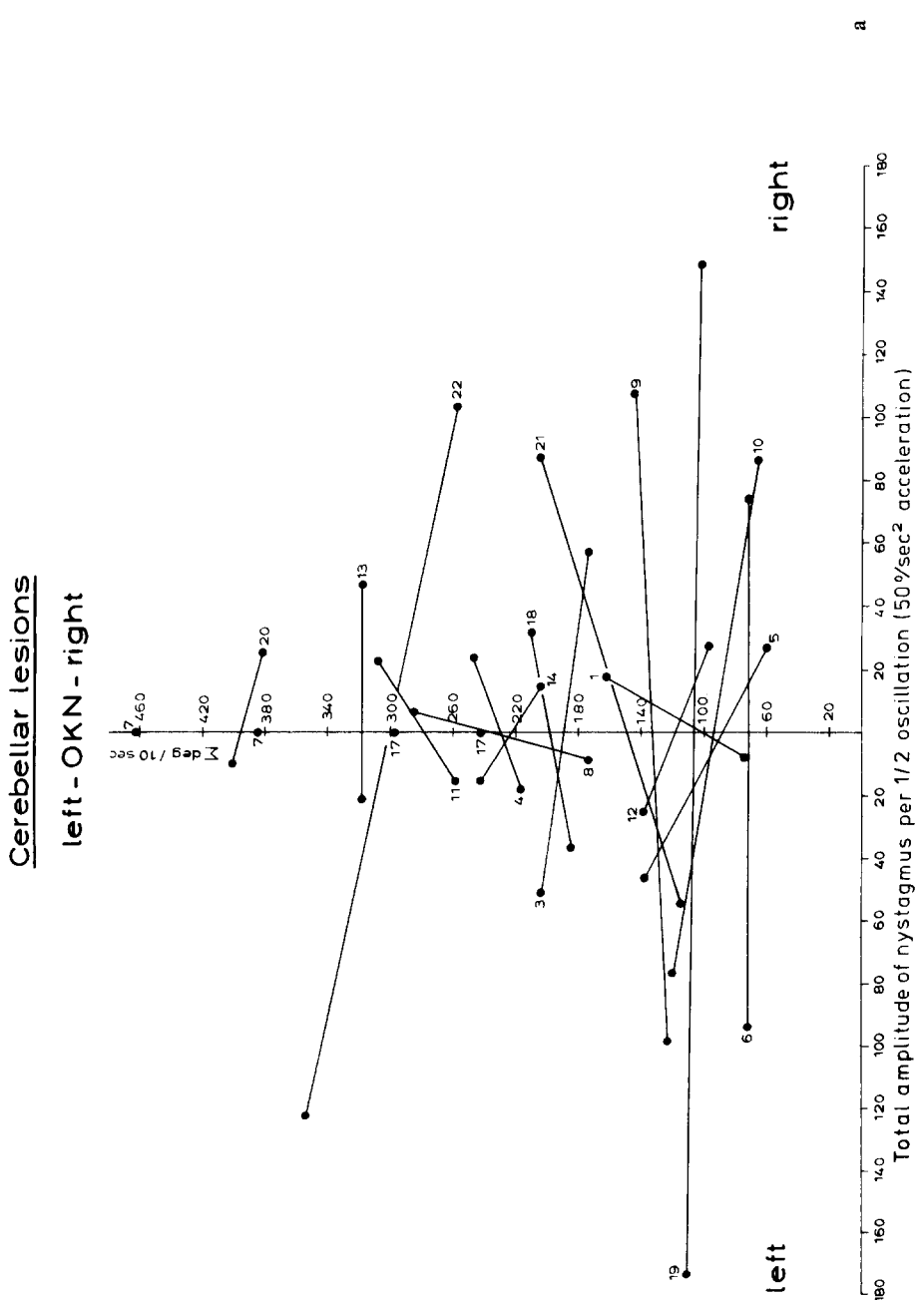


Fig. 2. Nystagmogram of a patient with a cerebellar astrocytoma on the left. Spontaneous nystagmus to the left (down), diminished OKN and vestibular nystagmus to the right, cogwheeled SP mainly to the left, and impaired fixation suppression mainly to the left


Cerebellar lesions
left - OKN - right
M deg / 10 sec
460
420
380
340
300
260
220
180
140
100
60
20
left
right
Total amplitude of nystagmus per 1/2 oscillation (50°/sec² acceleration)
20
40
60
80
100
120
140
160
180
a

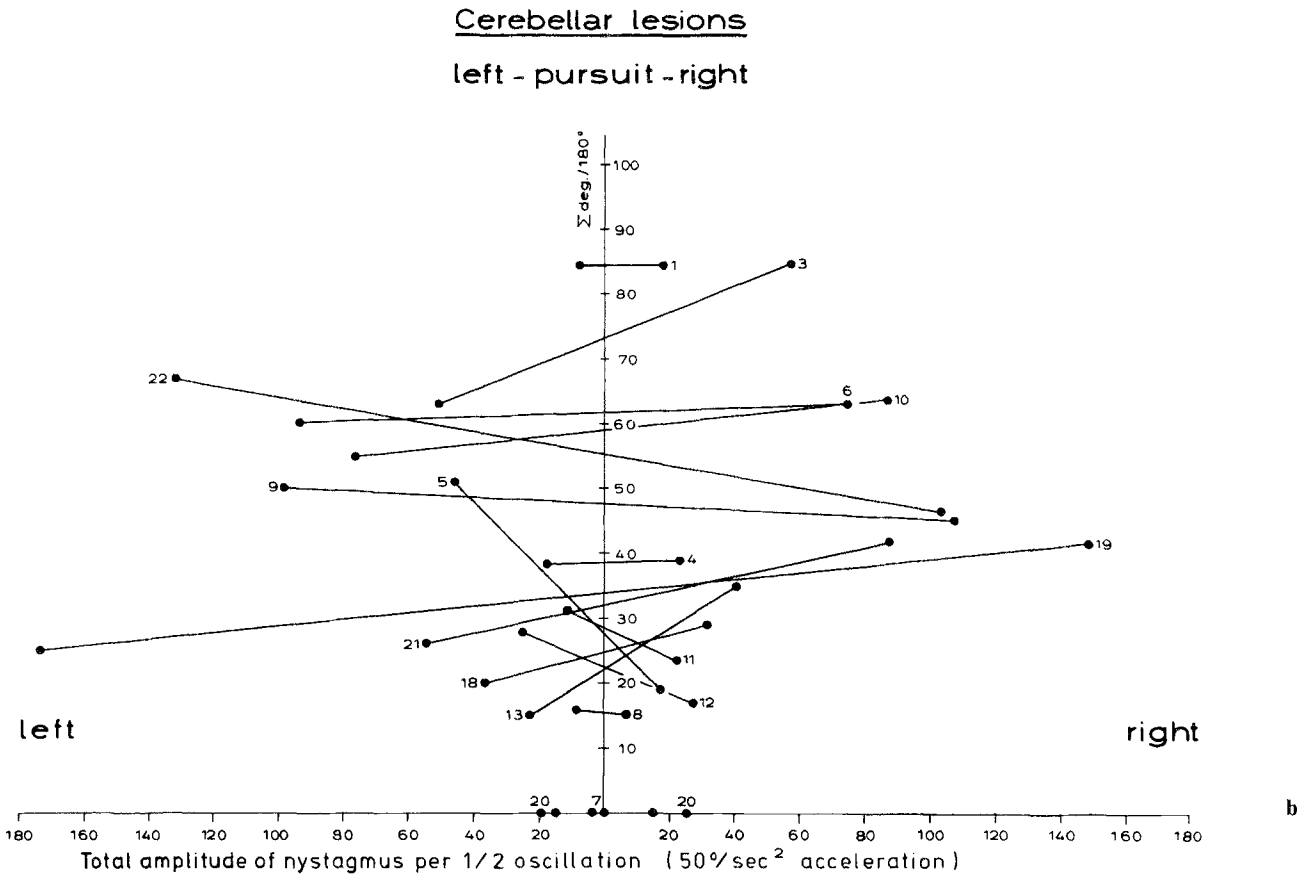
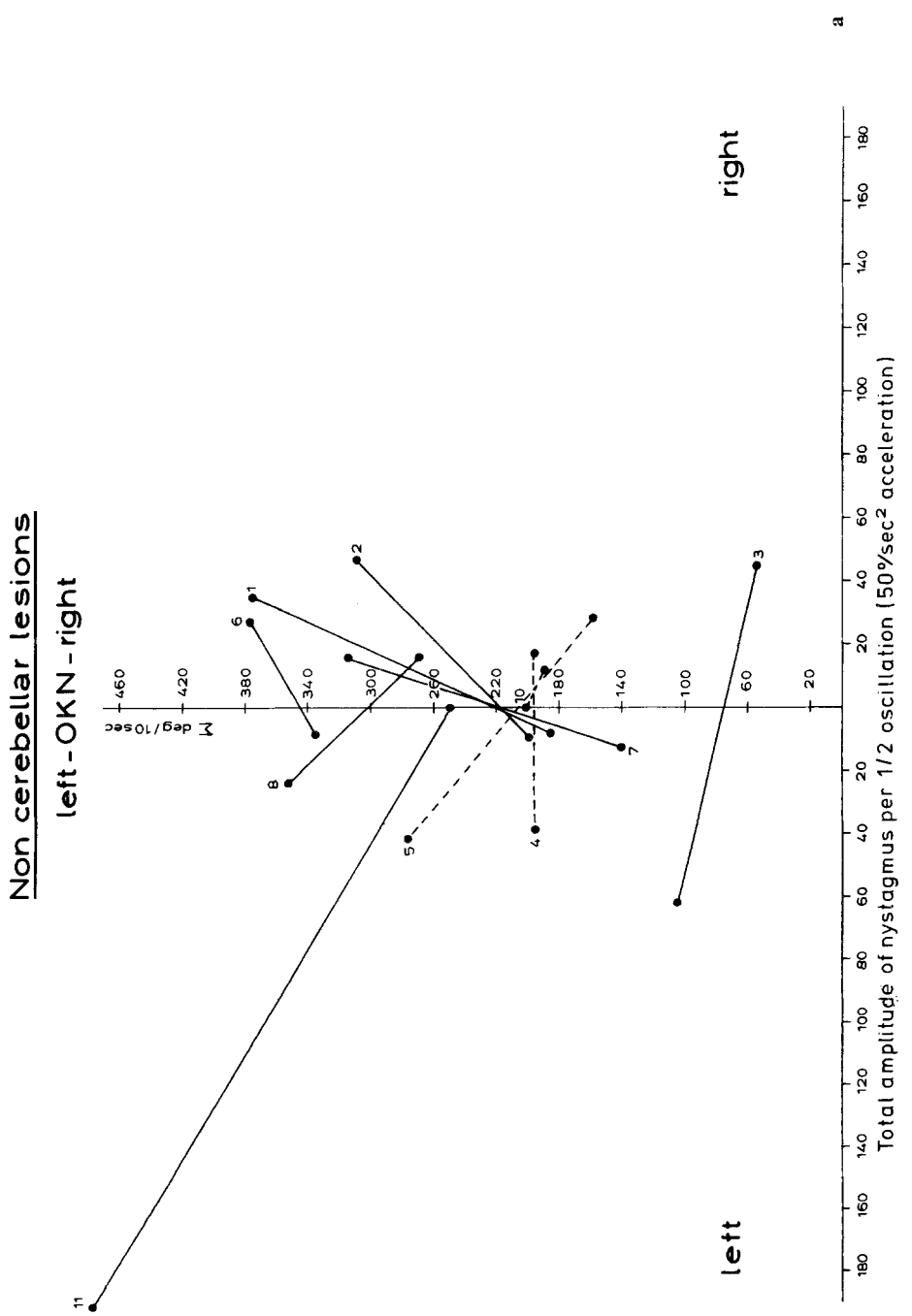


Fig. 3a and b. Impaired fixation suppression in correlation to OKN deficits (**a**) and SP deficits (**b**) in patients with cerebellar lesions. Abscissa plots total amplitude of nystagmus despite intended fixation per one-half body oscillation (0.1 Hz, maximal acceleration 50°/s^2, amplitude of body oscillation 253°). Ordinate plots total amplitude of OKN/10 s (**a**). Total OKN amplitude/10 s averages 380° in normals. In (**b**) ordinate is the total amplitude of saccades interspersed in one-half oscillation of a target sinusoidally moving, at 0.3 Hz. It is shown that, on the average, fixation suppression is less efficient the less the total amplitude of OKN (**a**), and the larger the total amplitude of saccades compensating for the SP deficit (**b**). This relationship is not perfect because of the high variability in vestibular excitability. The stronger this excitability, the larger the expected values on the abscissa and vice versa (patient 5 in Fig. 2)


Non cerebellar lesions
left-OKN-right
Σ deg/10sec
460
420
380
340
300
260
220
180
140
100
60
20
1
2
3
4
5
6
7
8
11
left
right
Total amplitude of nystagmus per 1/2 oscillation (50°/sec² acceleration)
a

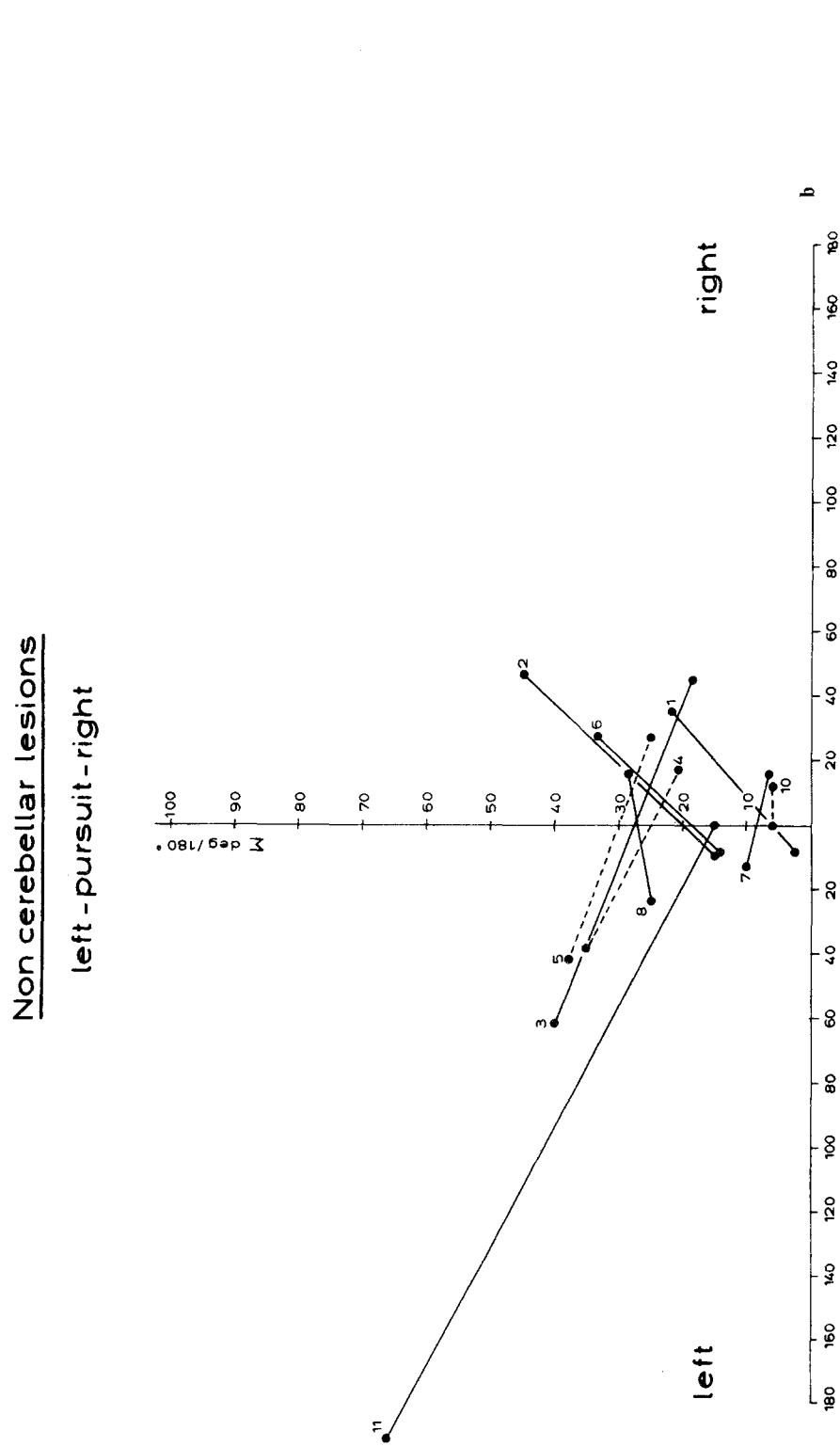


Fig. 4a and b. Impaired fixation suppression in correlation to OKN deficits (**a**) and SP deficits (**b**) in patients with non-cerebellar lesions. Plots are analogous to the ones in Figure 3. The original records of patient 1 are depicted in Figure 5 and those of patient 11 in Figure 6

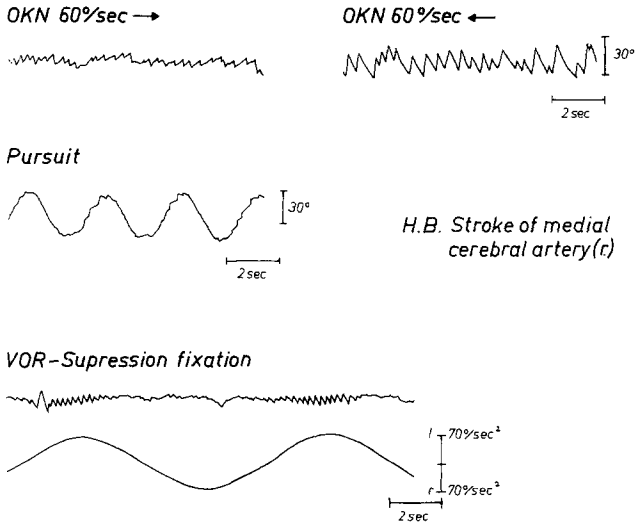


Fig. 5. Records of a patient (Number 1) after a stroke within the supply area of the right medial cerebral artery; OKN deficit to the left, pursuit deficit to the right, and fixation suppression deficit to the right

lobules on the left, but not the vermis. The vestibulocerebellum was specifically reported to have been saved during the operation. In addition to lateralized OKN and pursuit deficit, this patient (5) also showed a considerably less efficient fixation suppression towards the side ipsilateral to the tumor. A predominantly ipsilateral loss of fixation suppression was the rule in patients with unilateral cerebellar lesions.

The small number of patients examined so far does not allow for a closer localization of the minimal cerebellar defect necessary to produce this deficit. Cortical cerebellar atrophies exhibiting this symptom primarily involve the anterior lobe (Victor et al., 1959). But, most of our patients with cerebellar tumors and severe deficits of fixation suppression had lesions in the caudal part of the posterior lobe.

The symptoms best correlated with a fixation suppression deficit are OKN and SP deficits (Fig. 3). On the average, the disturbance of fixation suppression as measured by the total amplitude of nystagmus per half body oscillation is the stronger, the less the total amplitude of OKN and the larger the total amplitude of saccades interspersed in SP. The finding of a directional preponderance of OKN allows the prediction of a preponderance of VOR in the opposite direction when fixation suppression is tested.

Patients with poor vestibular excitability (as measured by the total amplitude of postrotatory nystagmus after stop from a 90°/s constant velocity body rotation in the dark) also tend to produce less nystagmus during the fixation suppression task, as expected. But in some cases, vestibular nystagmus intensity seems to be increased while fixating the target. Patient 10, for example, who suffered from a pontine angle tumor, exhibited practically no postrotatory

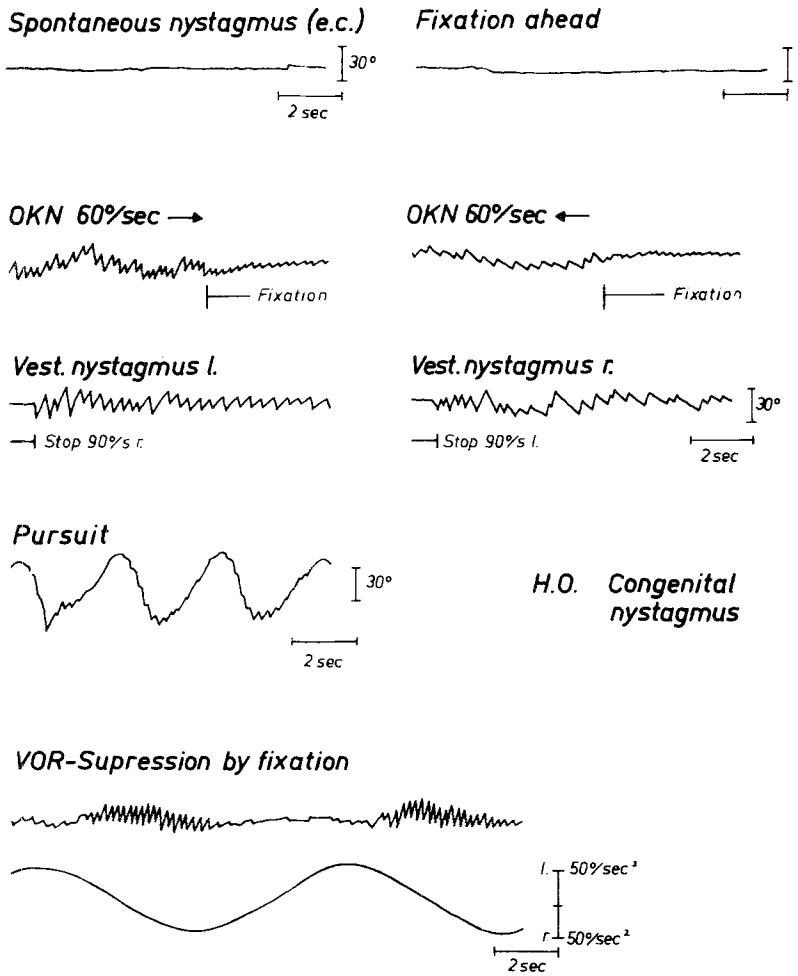


Fig. 6. Records of a patient (Number 11) with suspected congenital nystagmus. One of the reasons for this assumption is the lack of any other neurologic symptom and the characteristic lack of the ability to suppress large field OKN by fixation. Again, fixation suppression of vestibular nystagmus is severely impaired counter in direction to the impairment of OKN.

nystagmus with closed eyes in the dark, but showed a considerable nystagmus activity during the fixation suppression task. Similar results were obtained from patient 19 (multiple sclerosis) and patient 22 (angioblastoma of the cerebellar hemisphere). On the average, however, the total amplitude of nystagmus during fixation suppression increases with increasing vestibular excitability and decreasing OKN and SP performance.

Fixation Suppression in Non-Cerebellar Patients. Figure 4 depicts the fixation suppression data of patients exhibiting a clear OKN and SP deficit originating from an extracerebellar lesion. The main findings observed in cerebellar diseased patients are also obtained in noncerebellar patients, providing the disease causes a deficit of optokinesis. Granted a considerable variation in intensity of the

symptom, we have so far not found a patient with a severe OKN deficit and intact vestibular excitability who exhibited a normal capability to suppress VOR by fixation. As in cerebellar patients, impaired fixation suppression is always evident on the side ipsilateral to a SP deficit and the side contralateral to an OKN deficit. Lateralization is very clear in patients 1 and 2 who suffered from a stroke within a cerebral hemisphere. Figure 5 shows that this type of lesion leads to an impairment of OKN towards the contralateral side and correspondingly an impairment of SP and fixation suppression towards the ipsilateral side. Case 11 (Fig. 6) is of special interest. She most probably suffered from congenital nystagmus with fixation nystagmus and gaze nystagmus to the left, SP deficit to the left, OKN deficit to the right, and poor foveal suppression of large field optokinetic nystagmus, as well as a slight directional preponderance of vestibular nystagmus to the left. Repeated neurologic examinations including computer tomography showed no other neurologic symptoms and did not allow localization of the lesion. This patient again showed a very prominent unidirectional fixation suppression deficit corresponding in direction to the unidirectional disturbance of SP and OKN. With the exception of patient 11, fixation suppression tended to be somewhat better in noncerebellar patients. This may be attributable to the comparatively lower average vestibular excitability in this group (roughly minus 25%).

Discussion

Impaired fixation suppression of vestibular nystagmus in patients with cerebellar diseases had been observed earlier (Alpert, 1974; Hart, 1967; Zee et al., 1976). But, until now, it has not been quantitatively studied and compared to the performance of normal subjects. Decreased fixation suppression was also observed in patients with toxic encephalopathy (Maccario et al., 1972) and with lesions of the pontine reticular formation (Preber and Silfverskiöld, 1960). These reports already suggested extracerebellar lesions as an alternative cause of disturbed fixation suppression. The direction-specific disturbance of fixation suppression in patients with unilateral cerebral or pontine lesions substantiates this observation (Fig. 3).

Troost et al. (1976) studied fixation suppression mainly in patients with supranuclear palsy and assumed that the SP deficit caused this defect. Our study substantiates this concept: a lesion located anywhere in the cerebral hemispheres, the brainstem, or the cerebellum causing a SP deficit also impairs fixation suppression. Vice versa, among our cerebellar patients, fixation suppression was disturbed only if SP was cogwheeled and OKN slow phases were reduced, corresponding to a decrease in pursuit and OKN gain.

Patient 1 of the cerebellar group represents an instructive exception to the rule that any patient with SP and OKN deficit also exhibits a fixation suppression deficit. She shows saccadic pursuit and reduced OKN, but can well suppress vestibular nystagmus in the fixation task. Her results may be explained largely by her low vestibular excitability. The large variation in vestibular excitability partly explains the loose quantitative relationship between strength of vestibular nystag-

mus during the fixation suppression task and strength of OKN (Figs. 3 and 4). Another observation should be noted. In the nystagmus suppression task, some patients show a peculiar activation instead of an inhibition of vestibular nystagmus (patients 10, 19, and 22 of the cerebellar group). This finding is similar to the observations of Holmes (1917) and Hart (1967). They observed activation of vestibular nystagmus through the intention to fixate only in cerebellar patients. One possible explanation is that the total lack of fixation suppression present in these patients is combined with the nonspecific activation of vestibular nystagmus through the intention to look. Such an activation is also observed in normal subjects accelerated in the dark while simultaneously performing mental arithmetic or other mental tasks (Kornhuber, 1966), though their fixation suppression is more efficient.

Conceptually, fixation suppression can be considered as a superposition of vestibular nystagmus and a visually induced SP movement. If SP signals were added to vestibulo-ocular signals in order to achieve fixation suppression, one would assume the deficit to be always opposite in direction to the SP deficit and graded accordingly. As nystagmus is defined according to the direction of its fast phase the deficit of fixation suppression and OKN should always be opposite in direction. For our patients, this is indeed the case.

Earlier observations in patients with unilateral cerebral lesions showed a decreased ability to suppress caloric nystagmus towards the side of the lesion (Anderson et al., 1954; Carmichael et al., 1954; Bader and Kornhuber, 1965). This can be explained by the often observed OKN deficit towards the contralateral side. Patients with brainstem lesions involving the pontine reticular formation, however, show impaired OKN responses towards the ipsilateral side, and consequently a decreased fixation suppression of VN towards the contralateral side. Patients with a unilateral cerebellar damage have their OKN deficit towards the contralateral side (Dichgans and Jung, 1975; Dichgans, 1978; v. Reutern and Dichgans, 1977) and a fixation suppression deficit towards the ipsilateral side. Unilateral hemicerebellectomy in monkeys also yielded contralateral OKN deficits (Westheimer and Blair, 1974).

The frequency range of optimal fixation suppression is very similar to that of SP (Fender and Nye, 1961; Sünderhauf, 1960), whereas the optimum domain of the vestibulo-ocular reflex lies in the high frequency range (Benson, 1970). Correspondingly, fixation suppression is reduced in the high frequency range (Barr et al., 1976). Whether the ability to suppress the vestibulo-ocular reflex entirely depends on the OKN and SP gains or whether the cerebellum adds a specific component can not be decided at present. Fixation suppression deficits are particularly prominent in patients with cerebellar diseases, but so are OKN deficits (Baloh et al., 1975; Corvera et al., 1973; Dichgans and Jung, 1975; van Noorden and Prezioso, 1966; Zee et al., 1976). Vestibular hyperexcitability also is frequently found within this group (Baloh et al., 1975; Dichgans and Jung, 1975; Zee et al., 1974; Zee et al., 1976). The cerebellum, by way of its direct inhibitory connections from Purkinje cells to the vestibular nuclei (Ito, 1972; Ito et al., 1974; Fukuda et al., 1972), and on the basis of its visual inputs (Maekawa and Simpson, 1973; Simpson and Alley, 1974), could theoretically perform visually controlled

gain adjustments. The lack of gain control due to constantly diminished cerebellar inhibition of the vestibular nuclei would then combine with the SP and OKN deficit.

A Bedside Test for Fixation Suppression

Clinical testing of fixation suppression may be very useful in examining patients with cerebellar and noncerebellar diseases. The symptom is most prominent in cerebellar patients, but within the context of other neurologic symptoms it may also be used to detect and lateralize defects in other brain regions. A simple bedside method may be performed as follows: The patient either stands or is seated on a rotatable chair. He is asked to fixate his extended thumbs while holding his hands clasped together about 30 cm in front of his chest. The elbows are held tightly to the body. The examining physician takes the patient by the shoulders and rotates him horizontally to and fro, while observing the eyes. Normal subjects keep their eyes perfectly steady. If, however, suppression is inadequate, the patient's eyes continuously deviate off the fixation target by the vestibulo-ocular reflex and refixate by repetitive saccades, thus producing a nystagmus pattern (Dichgans, 1976; Zee, 1977).

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