Arch. Psychiat. Nervenkr. 226, 1—10 (1978)

Archiv für Psychiatrie und Nervenkrankheiten
Archives of Psychiatry and Neurological Sciences

Dynamic Changes of Optokinetic After-Nystagmus (OKAN) Caused by Brief Visual Fixation Periods in Monkey and in Man*

W. Waespe, Th. Huber, and V. Henn

Neurologische Klinik, Universität Zürich, CH-8091 Zürich, Switzerland

Summary. Optokinetic nystagmus (OKN) continues after the cessation of visual stimulation in complete darkness as primary optokinetic after-nystagmus (OKAN I). After variable periods of time, it is followed by secondary OKAN (OKAN II). Short periods of visual fixation during OKAN I in monkey and in man inhibit OKAN I, but enhance OKAN II. The enhanced OKAN II starts earlier, lasts longer, and often reaches higher slow-phase velocities than in control experiments. Therefore, OKAN II depends not on the occurrence or strength of OKAN I, but mainly on parameters of the preceding optokinetic stimulus. Results suggest that OKAN I duration is partially determined by the development of OKAN II.

Key words: Optokinetic after-nystagmus – Visual fixation – Monkey – Models – Nystagmic periods.

Zusammenfassung. Der optokinetische Nystagmus (OKN) dauert in Dunkelheit als optokinetischer Nachnystagmus (OKAN) fort. Der primäre OKAN (OKAN I) schlägt in dieselbe Richtung wie der vorausgehende OKN. Nach einer variablen Periode erfolgt eine Nystagmusumkehr (OKAN II). Beim Rhesusaffen und beim Menschen wurden die Effekte kurzer Fixationsperioden während des primären OKAN auf OKAN I und OKAN II untersucht. Durch solche Fixationsperioden wird OKAN I gehemmt, OKAN II jedoch nach Intensität und Dauer verstärkt. Deshalb ist OKAN II vorwiegend von den Parametern der vorausgegangenen optokinetischen Reizung abhängig. Die Resultate zeigen, daß OKAN II auch die Dynamik und Dauer des OKAN I mitbestimmen kann.

Schlüsselwörter: Optokinetischer Nachnystagmus – Visuelle Fixation – Rhesusaffe – Modelle – Nystagmusperiodik.

* Supported in part by Swiss National Foundation for Scientific Research 3.672—0.77 and the European Training Program for Brain and Behavior Research
Send offprint requests to Dr. V. Henn

0003-9373/78/0226/0001/$ 02.00

Introduction

The oculomotor response to a moving full-field visual pattern is *optokinetic nystagmus (OKN)*, and it is followed by *optokinetic after-nystagmus (OKAN)* in complete darkness (Ohm, 1926). Primary OKAN (OKAN I) has the same direction as the preceding OKN. The duration of OKAN I is variable and it may change direction to continue as secondary OKAN (OKAN II).

In order to explain the dynamic changes of OKAN, it has been suggested that during optokinetic stimulation two opposing tonic activities build up, which lead first to OKAN I and then to OKAN II (Brandt et al., 1974; Zee et al., 1976; Büttner et al., 1976; Waespe and Henn, 1978). To test this hypothesis further, brief periods of visual fixation were introduced during OKAN I to determine whether OKAN I and OKAN II are inhibited in the same way, or whether OKAN II is affected in a different way, which would then support the hypothesis that OKAN II does indeed have a separate generator. Experiments were performed both in monkey and in man.

Methods

1. Monkeys

Monkeys were not selected for response symmetry. Initially, spontaneous nystagmus was absent or only minimal (less than 2°/s). Details of methods have been reported elsewhere (Waespe and Henn, 1977a). DC electrodes were implanted in eight rhesus monkeys *(Macaca mulatta)* to monitor eye position (Bond and Ho, 1970) and bolts were attached to the skull to fix the head during experiments. Animals were seated in a primate chair and received amphetamine (0.3—0.5 mg/kg i.m.) to maintain alertness. They were totally enclosed by an optokinetic cylinder (diam 124 cm, height 86 cm) covered with vertical black and white stripes, each 7.5° wide. Monkeys were tested on consecutive mornings with optokinetic drum rotation at 60°/s (120°/s for monkeys 20 and 26) for 3 min. For control experiments, the lights were then turned off and the monkeys maintained in complete darkness for the next 4 min, during which OKAN occurred. The lights were then turned on again for 2 min and no stimulation was given. Before the next trial was started, lights were turned off again for 1 min to check for occurrence of any spontaneous nystagmus. A complete cycle lasted 10 min (Waespe and Henn, 1978). Test experiments were different from control experiments only by a 5-s light period allowing monkeys to fixate on the optokinetic drum that was then stationary. The light period was introduced:

1. immediately after termination of optokinetic stimulation, i.e., the drum was stopped in the light and 5 s later lights were turned off (monkeys 20, 21, 23, 24, and 25);
2. 2.5 s after termination of optokinetic stimulation, i.e., lights were turned off during optokinetic stimulation and the drum was stopped, 2.5 s later lights were turned on for 5 s (monkeys 26, 29, and 30);
3. or 10 s after termination of optokinetic stimulation 5-s light period (monkeys 21, 23, 24, 25, and 26).

As repetition of optokinetic stimulation by itself leads to changes of OKAN I and OKAN II (Waespe and Henn, 1978) control and test experiments as well as direction of OKN stimulation were systematically altered. For analysis, the following parameters were considered:

1. OKAN I: duration, measured from termination of optokinetic stimulation to onset of OKAN II;
2. OKAN II: peak slow-phase velocity and time of its occurrence.

The two phases of after-nystagmus cannot easily be measured using the same parameters. Duration (or total amplitude) is a reliable measure for OKAN I, but is not suitable for OKAN II, as it can outlast the recording period of 4 min or continue as spontaneous nystagmus. Therefore, peak slow-phase velocity was used to measure strength of OKAN II. Peak slow-phase velocity of OKAN I is the same as during the preceding OKN.

If spontaneous nystagmus was present in the dark period immediately preceding the trial, it was subtracted or added to that value. Nystagmus slow-phase velocity was measured by differentiating the eye-position signal. Eye movements were calibrated from optokinetic nystagmus responses; for velocities up to 60°/s, the slow-phase nystagmus velocity can be considered to equal stimulus velocity (Cohen et al., 1977).

2. Human Subjects

Forty-one healthy subjects were initially tested (average age 28 years); 27 of them participated in the experiments. The remainder either had spontaneous nystagmus in the dark or optokinetic after-nystagmus could not reliably be elicited. Subjects were seated in the center of a white circular screen that subtended an angle of 60° vertically and 180° horizontally at a distance of 100 cm. A pattern of moving black and white vertical stripes (stripe width 6° 40′ for black and 8° for white stripes) was projected onto the screen. Horizontal eye position was recorded with standard EOG electrodes and displayed on a strip chart recorder (velocity 5 mm/s). Eye position was calibrated by alternately fixating on small spots of light.

Velocity of optokinetic stimulation was 60°/s. In previous experiments, OKN stimulation for 2 min was found to be optimal for eliciting OKAN. In the control experiment, lights were turned off after 2 min of optokinetic stimulation and subjects were in total darkness continuously performing arithmetic tasks to maintain alertness. Test experiments differed by introducing a 2-s light period 5 s after termination of optokinetic stimulation, and allowing subjects to fixate on the now stationary pattern. Test and control experiments were systematically alternated. Duration and total amplitude of OKAN I and OKAN II were used for analysis. Since nystagmus was generally weak and subsided within 3 min, both phases of after-nystagmus could be measured using the same parameters.

Results

1. Monkeys

Optokinetic stimulation elicited OKAN I as well as OKAN II in all monkeys. The introduction of a 5-s visual fixation period immediately or 2.5 s after termination of optokinetic stimulation shortened the duration of OKAN I, compared with control experiments (Figs. 1 and 2). Usually, OKAN I was not totally suppressed during the light period. If it was, it reappeared during the subsequent dark period. Extreme values were found in monkeys 21 and 29. In monkey 21, OKAN I lasted on the average 6.8 s, i.e., outlasted the light period only by 1.8 s. In monkey 29, OKAN I was minimally inhibited and outlasted the light period by 21.4 s.

The 5-s fixation period had a different effect on OKAN II: its slow-phase velocity never decreased; it remained constant in some and even increased in others (Figs. 1 and 2). In monkeys 25, 26, and 29, OKAN II slow-phase velocity did not change, whereas in monkeys 20, 21, 23, 24, and 30, its value increased between 4.6°/s and 26.2°/s (between 32% and 113%). Furthermore, OKAN II appeared and reached its maximal slow-phase velocity much earlier after the fixation period than in the control experiments (Fig. 1). Figure 3 shows the

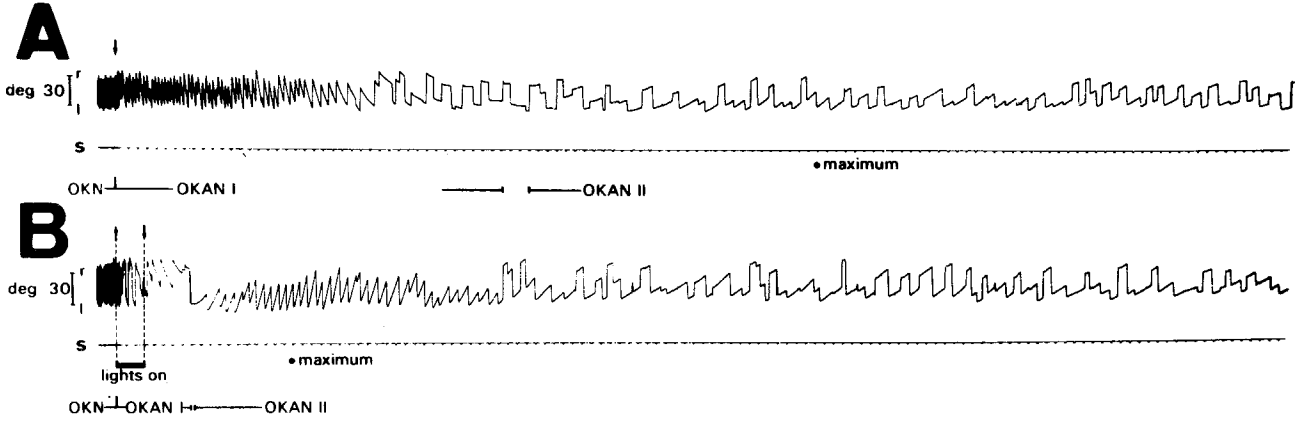

Fig. 1A and B. Original record of OKN and OKAN in monkey 20. Only horizontal eye movements are displayed. Note change in time base (dots below nystagmus trace). Optokinetic stimulus velocity 120°/s. Upward arrow marks stopping of the drum in the light, downward arrows mark switching lights off. In **A** (control experiment), lights were switched off during optokinetic stimulation. OKAN I lasts for 43.5 s and is followed by OKAN II. In **B**, the test experiment immediately preceding A, the drum was stopped in the light and 5 s later lights were turned off. OKAN I is inhibited and lasts only for 9.5 s, but it is followed by a much stronger OKAN II, which reaches peak slow-phase velocity earlier (max.)

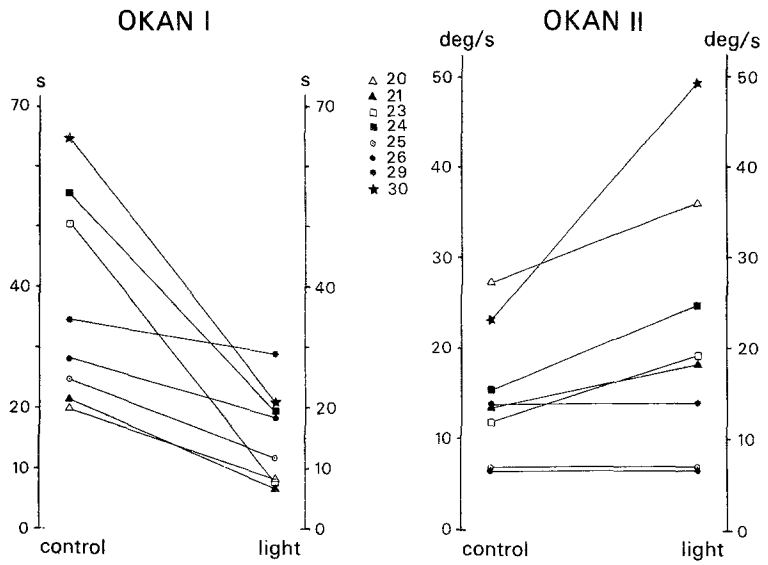

Fig. 2. Absolute values of OKAN I duration and OKAN II peak velocity in control and test experiments (light) averaged over all trials for every monkey ($n = 6$ for monkeys 20, 23, and 24; $n = 12$ for monkeys 21, 25, 26, 29, and 30). OKAN I duration is reduced, whereas OKAN II peak velocity remains constant or is increased

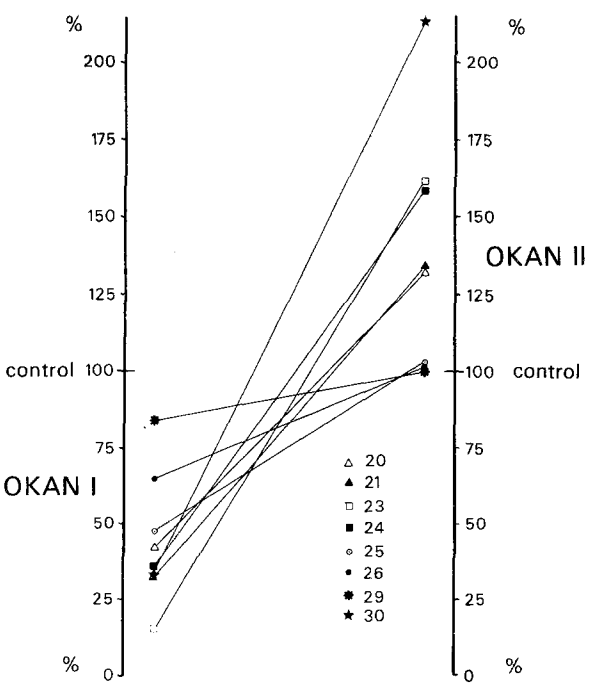

Fig. 3. Relative decrease of OKAN I duration and increase of OKAN II velocity compared with control experiments (control values 100%). OKAN I duration (*left*) is always reduced (mean decrease 65.5%, $n = 8$), and OKAN II peak slow-phase velocity (*right*) remained the same in three monkeys and increased in five monkeys (mean increase 38.8%, $n = 8$)

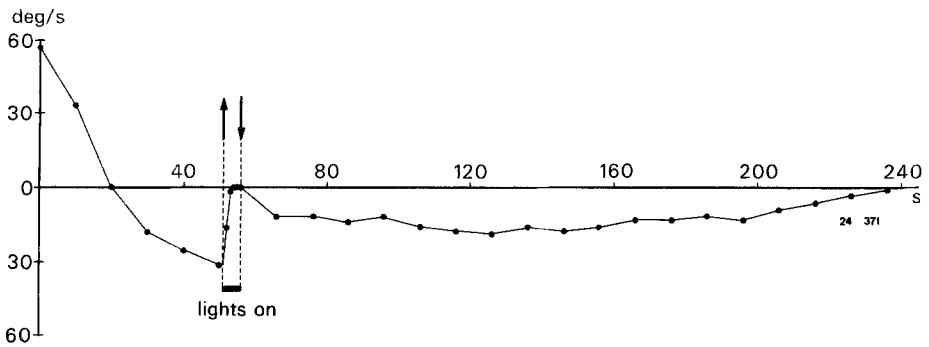

Fig. 4. Effect of a 5-s light period (with stationary surroundings) introduced immediately after OKAN II has reached peak slow-phase velocity in monkey 24. Ordinate is slow-phase velocity of nystagmus. OKAN II is totally inhibited, but recovers during the subsequent dark period. It reaches a second peak of 12.5°/s around 70 s after lights have been switched off again

relative decrease in OKAN I and the concomitant increase in peak slow-phase velocity of OKAN II.

In monkeys 21, 23, 24, 25, and 26, the 5-s light period was also introduced 10 s after termination of optokinetic stimulation, i.e., the lights were switched off during optokinetic stimulation and 10 s later they were turned on again for 5 s. Results were qualitatively similar as in the experiments reported above. OKAN I was totally suppressed and did not recover, while OKAN II remained unchanged or increased slightly.

In monkeys 23 and 24, introduction of the 5-s fixation period was delayed until maximal peak slow-phase velocity of OKAN II was reached (Fig. 4). In each trial, OKAN II was completely inhibited during the light period, but always recovered during the subsequent dark period. A second peak in slow-phase velocity of OKAN II was reached 52 s ($n = 8$) after lights were turned off again. The second peak slow-phase velocity was reduced to 48% (absolute value 11.8°/s). Under these stimulus conditions, there was no further nystagmus reversal after OKAN II. The results show that OKAN II recovers very slowly, but can reach high values.

2. Human Subjects

All 27 subjects were exposed to stimulation in both directions resulting in 54 test and control experiments. OKAN I could reliably be elicited in all control experiments and OKAN II could also be seen in 15 controls. By introducing a 2-s light period 5 s after termination of optokinetic stimulation, OKAN I was usually reduced (Fig. 5A) in duration and total amplitude. Its mean relative reduction in duration was 41.0% (SD 53.0%) and in total amplitude 37.5% (SD 41.0%). On the other hand, OKAN II showed a clear tendency to increase. Of the 15 experiments in which OKAN II was already present in the controls, the fixation period increased OKAN II in 13 experiments and decreased it in only two. Twelve subjects with no OKAN II during control experiments developed OKAN II for

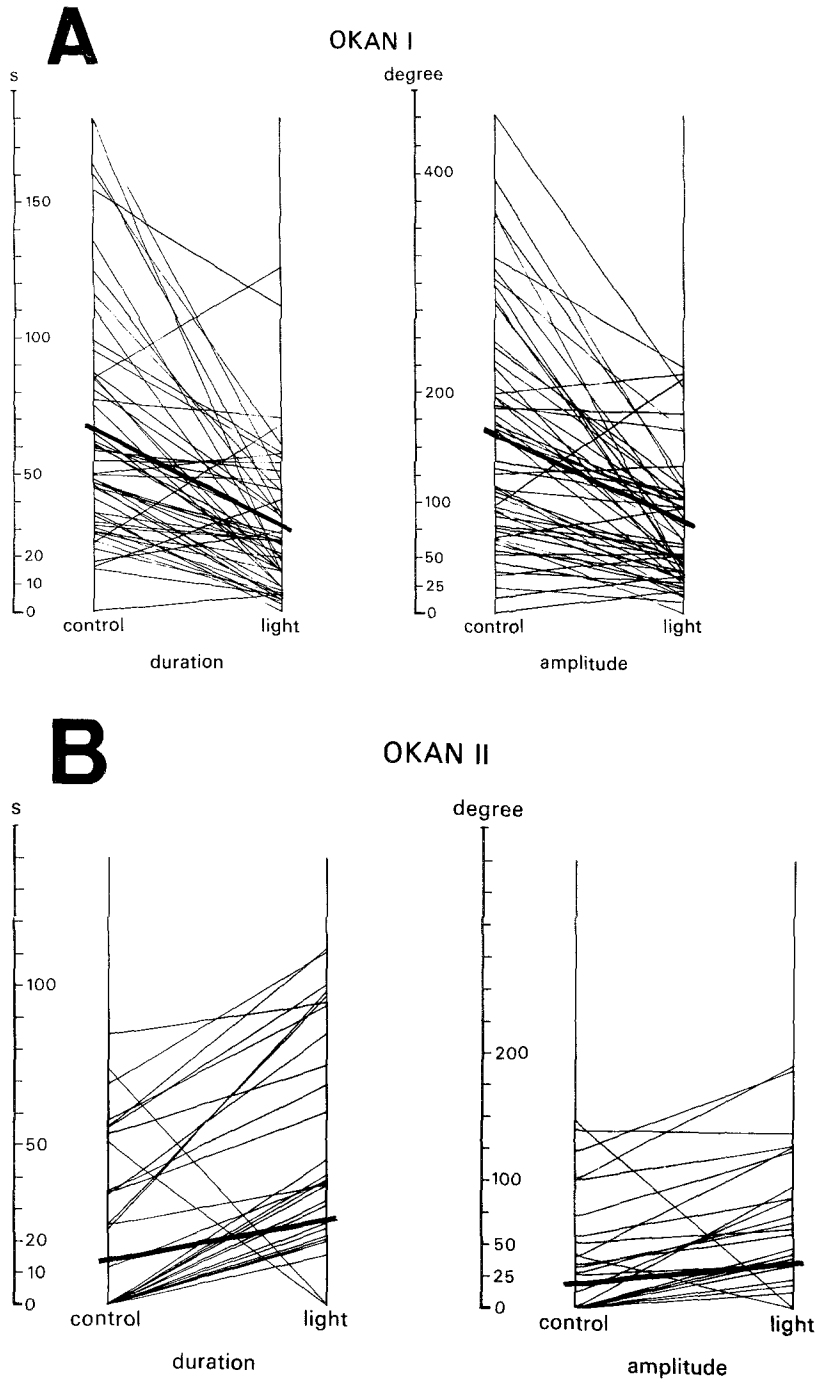

Fig. 5 A and B. Duration and total amplitude of OKAN I (**A**) and OKAN II (**B**) in men for control experiments and test experiments (light). Trials for left and right side are drawn separately ($n = 54$). The heavy lines represent average values. In the majority of cases, OKAN I is reduced in duration (averaged values from 65.9 to 30.7 s) and amplitude (from 159.7° to 76.7°) by the 2-s light period (**A**). Except for two cases, OKAN II is increased in duration and total amplitude. In 27 experiments, OKAN II could not be elicited, neither during control or test experiments, although OKAN I also decreased in these subjects. Averaged increase in duration of OKAN II was from 12.8 to 26.4 s and in total amplitude from 19.0° to 32.3°

the first time during test experiments. In 27 experiments, OKAN II did not develop even after fixation periods, although OKAN I had decreased. The mean increase of OKAN II for all subjects was 41.4% (SD 66.0%) in duration and 35.7% (SD 84.3%) in total amplitude.

Discussion

It is well known that visual fixation suppress not only spontaneous and post-rotatory nystagmus (Ohm, 1926), but also optokinetic after-nystagmus (Krieger and Bender, 1956; Cohen, 1974; Cohen et al., 1977). Usually only the primary phase of nystagmus has been examined. By introducing short periods of visual fixation during primary OKAN, we demonstrated that OKAN I and OKAN II are influenced in a reciprocal way: OKAN I is reduced (duration and total amplitude) whereas OKAN II remains unchanged or is even increased (peak slow-phase velocity, duration, and total amplitude).

Although different parameters have been analyzed in monkey and in man, the results are qualitatively similar. Despite pronounced inter- as well as intra-individual differences, it could be demonstrated that the stronger the reduction of OKAN I by visual fixation, the more likely an increase in OKAN II (Fig. 3). Human subjects with no OKAN II in the control experiments frequently showed OKAN II when OKAN I was inhibited. From the present and earlier studies (Büttner et al., 1976; Waespe and Henn, 1978), we conclude that the development of OKAN II primarily depends on the parameters of the preceding optokinetic stimulation. OKAN II cannot be considered as a central response to the occurrence of OKAN I. If it were so, the two phases of OKAN should always decrease or increase together. Our findings lend further support to the hypothesis that during optokinetic stimulation two opposing tonic activities build up in different structures, which discharge with different time constants (Brandt et al., 1974). The introduction of a short visual fixation period during OKAN I selectively discharges the first structure responsible for the ongoing phase of nystagmus (OKAN I), without influencing the second structure responsible for OKAN II. This unmasks OKAN II, which begins earlier and shows a higher slow-phase velocity. In subjects with no OKAN II during control experiments, OKAN II can occur when primary OKAN is inhibited.

OKAN II can also be inhibited by a 5-s fixation period introduced during its peak slow-phase velocity. However, OKAN II always returned and developed a second maximum. In contrast, OKAN I showed little recovery after light inhibition. A possible explanation is that OKAN I is always opposed by OKAN II and that there is no such force neutralizing OKAN II.

Brief periods of visual fixation have been found in man to have similar effects on the primary and secondary phases of vestibular nystagmus (VN) and for the sensation of turning (Guedry et al., 1961). Collins (1968a and b) reported that visual fixation during primary VN shortens it, but produces an accentuated secondary response. This agrees well with our results for OKAN. Therefore, it seems likely that the dynamics of VN and OKAN are similar. Guedry (1974) put forward the hypothesis that the stationary fixation pattern, viewed during OKAN I

and VN I, introduces retinal slip, which is considered to be the causal factor in producing an increased OKAN II and VN II, respectively. One argument against this is that OKAN I usually showed some recovery after lights off before OKAN II appeared. Secondly, the same fixation period during OKAN II did not lead to a further reversal of nystagmus.

In another study, we demonstrated a close correlation between neuronal activity in the vestibular nuclei and primary and secondary phases of VN and OKAN (Waespe and Henn, 1977b). During OKAN, the activity of the vestibular nuclei neurons reflect actual slow-phase velocity of nystagmus. Furthermore, when nystagmus (OKAN) is inhibited by a fixation period, neuronal activity is also inhibited. Therefore, the structures responsible for storing the activity that leads to the different phases of after-nystagmus must be located prior to the vestibular nuclei.

Several models have been proposed to simulate or to explain the dynamics of nystagmus (Robinson, 1977a and b; Cohen et al., 1977; Allum and Graf, 1977). However, only primary phases of nystagmus were considered. As we have shown, the duration and dynamics of primary after-nystagmus, which can globally be characterized by a dominant time constant (Raphan et al., 1977), is determined to a large extent by central activity, which may lead to secondary after-nystagmus. Therefore, in agreement with Brandt et al. (1974), we consider secondary after-nystagmus not as a simple aftereffect in response to primary nystagmus, but as a sign of central activity or counter regulation, which plays a decisive role during all phases of after-nystagmus. This phenomenon can also be observed during per- or postrotatory nystagmus, and introduces a periodicity in the nystagmus response.

Acknowledgements. We thank Ms. V. Isoviita for assistance during experiments and Mr. V. Corti for building and maintaining part of the electronic equipment.

References

Allum, J. H. J., Graf, W.: Time constants of vestibular nuclei neurons in the goldfish: A model with ocular proprioception. Biol. Cybern. **28**, 95—99 (1977)

Bond, H. W., Ho, P.: Solid miniature silver-silver chloride electrodes for chronic implantations. Electroencephalogr. Clin. Neurophysiol. **28**, 206—208 (1970)

Brandt, Th., Dichgans, J., Büchele, W.: Motion habituation: Inverted self-motion perception and optokinetic after-nystagmus. Exp. Brain Res. **21**, 337—352 (1974)

Büttner, U., Waespe, W., Henn, V.: Duration and direction of optokinetic after-nystagmus as a function of stimulus exposure time in the monkey. Arch. Psychiat. Nervenkr. **222**, 281—291 (1976)

Cohen, B.: The vestibulo-ocular reflex arc. In: Handbook of sensory physiology (Kornhuber, H. H., ed.). Vol. VI/1, pp. 447—540. Berlin, Heidelberg, New York: Springer 1974

Cohen, B., Matsuo, V., Raphan, Th.: Quantitative analysis of the velocity characteristics of optokinetic nystagmus and optokinetic after-nystagmus. J. Physiol. **270**, 321—344 (1977)

Collins, W. E.: Modification of vestibular nystagmus and "vertigo" by means of visual stimulation. Trans. Am. Acad. Ophthalmol. Otolaryngol. **72**, 962—979 (1968a)

Collins, W. E.: Special effects of brief periods of visual fixation on nystagmus and sensations of turning. Aerospace Med. **39**, 257—266 (1968b)

Guedry, F. E., Collins, W. E., Sheffey, P. L.: Perceptual and oculomotor reactions to interacting visual and vestibular stimulation. Percept. Mot. Skills **12**, 307—324 (1961)

Guedry, F. E.: Psychophysics of vestibular sensations. In: Handbook of sensory physiology (Kornhuber, H. H., ed.). Vol. VI/2, pp. 3—154. Berlin, Heidelberg, New York: Springer 1974

Krieger, H. P., Bender, M. B.: Optokinetic after-nystagmus in the monkey. Electroencephalogr. Clin. Neurophysiol. **8**, 97—106 (1956)

Ohm, J.: Über den Einfluß des Sehens auf den vestibulären Drehnystagmus und den Nachnystagmus. Z. Hals Nasen Ohrenheilkd. **16**, 521—540 (1926)

Raphan, Th., Cohen, B., Matsuo, V.: A velocity-storage mechanism responsible for optokinetic nystagmus (OKN), optokinetic after-nystagmus (OKAN) and vestibular nystagmus. In: Control of gaze by brain stem neurons. Developments in neuroscience (Baker, R., Berthoz, A., eds.). Vol. 1, pp. 37—47. Amsterdam: Elsevier 1977

Robinson, D. A.: Linear addition of optokinetic and vestibular signals in the vestibular nucleus. Exp. Brain Res. **30**, 447—450 (1977a)

Robinson, D. A.: Vestibular and optokinetic symbiosis: An example of explaining by modelling. In: Control of gaze by brain stem neurons. Developments in neuroscience (Baker, R., Berthoz, A., eds.). Vol. 1, pp. 49—58. Amsterdam: Elsevier 1977b

Waespe, W., Henn, V.: Neuronal activity in the vestibular nuclei of the alert monkey during vestibular and optokinetic stimulation. Exp. Brain Res. **27**, 523—538 (1977a)

Waespe, W., Henn, V.: Vestibular nuclei activity during optokinetic after-nystagmus (OKAN) in the alert monkey. Exp. Brain Res. **30**, 323—330 (1977b)

Waespe, W., Henn, V.: Reciprocal changes in primary and secondary optokinetic after-nystagmus (OKAN) produced by repetitive optokinetic stimulation in the monkey. Arch. Psychiat. Nervenkr. **225**, 23—30 (1978)

Zee, L. R., Yee, R. D., Robinson, D. A.: Optokinetic responses in labyrinthine defective human beings. Brain Res. **113**, 423—428 (1976)

Received May 20, 1978