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Duration and Direction of Optokinetic After-Nystagmus as a Function of Stimulus Exposure Time in the Monkey *

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SUMMARY. 1. Parameters of the optokinetic after-nystagmus (OKAN) outlasting optokinetic stimulation were studied in monkeys. With constant pattern velocity (60°/s) exposure times were varied between 2 s and 15 min.
2. All monkeys showed a primary after-nystagmus moving in the same direction (OKAN I) as the preceding optokinetic nystagmus, under all conditions tested. A secondary after-nystagmus in the opposite direction (OKAN II), was only observed after exposure times of 30 s or longer. After a 15-min exposure, half of the monkeys showed an early onset of OKAN II in less than 1 min, whereas for the remaining half the transition to OKAN II occurred only after 4 min or not at all.
3. In monkeys showing an early onset of OKAN II, the duration of OKAN I decreased and the maximal slow phase velocity of OKAN II increased consistently with longer exposure times. In several instances OKAN III, moving in the same direction as OKAN I, was seen after OKAN II.
4. Monkeys in which OKAN II was late, or absent, often showed minima and additional maxima of slow phase velocity of OKAN.
5. The results are discussed in terms of two opposing mechanisms underlying the generation of OKAN and their connections to the vestibular system.

KEY WORDS: Optokinetic After-Nystagmus - Stimulus Duration - Monkey.

ZUSAMMENFASSUNG. 1. Optokinetischer Nachnystagmus (OKAN) wurde bei Makaken untersucht, wobei die Dauer der vorhergehenden optokinetischen Reizung zwischen 2 sec und 15 min variiert wurde. Die Geschwindigkeit der optokinetischen Reizung betrug 60°/sec.
2. Unter allen Reizbedingungen zeigten die Affen einen primären Nachnystagmus, der in dieselbe Richtung (OKAN I) wie der vorhergehende optokinetische Nystagmus schlug. Ein sekundärer Nachnystagmus, der in die entgegengesetzte Richtung schlug (OKAN II), kam nur nach einer Reizdauer von 30 oder mehr sec zur Darstellung. Nach einer Reizdauer von 15 min trat bei der Hälfte der Affen OKAN II in weniger als 1 min auf, während bei der anderen Hälfte der Übergang zu OKAN II frühestens nach 4 min oder gar nicht auftrat.
3. Bei Affen mit einem frühen Einsetzen des OKAN II nahm mit zunehmen-

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der Reizlänge die Dauer des OKAN I ab, während die maximale Geschwindigkeit der langsamen Phase des OKAN II zunahm. In einigen Fällen wurde ein OKAN III, welcher in dieselbe Richtung wie OKAN I schlug, im Anschluß an OKAN II beobachtet.
4. Bei Affen mit spätem oder fehlendem Auftreten des OKAN II zeigte die Nystagmus-Geschwindigkeit häufig Zwischen-Minima und -Maxima.
5. Die Ergebnisse werden unter der Annahme von 2 gegenläufigen Prozessen, welche bei der Entstehung des OKAN beteiligt sind, diskutiert und mit vestibulär induziertem Nachnystagmus verglichen.

SCHLÜSSELWÖRTER: Optokinetischer Nachnystagmus - Reizdauer - Rhesusaffe.

INTRODUCTION

Optokinetic nystagmus (OKN) is the oculomotor response to a moving pattern and can be elicited in all animals which can move their eyes. Optokinetic after-nystagmus (OKAN) is the oculomotor response outlasting the visual stimulation. Its first observation is usually credited to Ohm (1927). Systematic investigations were made by Ter Braak (1936) in several species, by Krieger & Bender (1956) and Komatsuzaki et al. (1969) in monkeys, and Mackensen et al. (1959, 1961) in humans, all with similar results. OKAN characteristics depend on the parameters of the preceding OKN. The primary OKAN (OKAN I) always has the same direction as the preceding OKN. In some instances a secondary OKAN (OKAN II) can also be observed. OKAN II is defined as optokinetic after-nystagmus which moves in the opposite direction to the preceding OKN.

Parameters of OKAN in monkeys, using exposure times of OKN up to 60 s, were recently published (Takemori, 1974). Brandt et al. (1974) investigated the time course of OKAN in relation to stimulus exposure time in humans. The present report will also be confined to the variation of OKAN as a function of stimulus exposure time. Using the same stimulus parameters as Brandt et al. (1974), a direct comparison with the data obtained from humans is possible. These data are also necessary to evaluate single unit studies in the vestibular nuclei during OKAN in monkeys (Waespe & Henn, 1976) and to test models of a supposed OKAN generator (Raphan et al., 1976).

METHODS

Eight juvenile monkeys (Macaca mulatta) were used for this study. None of the animals had spontaneous nystagmus in the dark. They all had bolts attached to the skull in order to fix their heads during experiments. DC electrodes (Bond & Ho, 1970) were chronically implanted around the bony orbit to measure horizontal and vertical eye position. The monkey sat upright on a turntable which was enclosed by a lightproof cylinder (radius 0.62 m) covered by alternating black and white vertical stripes. This cylinder could be rotated around the stationary monkey about a vertical axis by a servocontrolled motor.

Before the experiments the monkeys received amphetamine (max. dose 0.5 mg/kg) to guarantee a constant level of alertness. During experiments the monkey first sat in the dark. After the cylinder was

accelerated to a constant velocity of 60°/s, the lights were turned on for periods of 2 s, 5 s, 10 s, 30 s, 1 min, 3 min, or 15 min in a constant sequence of increasing exposures. These sequences were followed for cylinder rotation to the left and right. After each light period eye movements were recorded in the dark for 5-20 min with the cylinder stationary. Before the next longer light period the monkey was left with the lights on for an additional 5-10 min during which spontaneous eye movements occurred. Care was taken that time between experimental runs was long enough to avoid outlasting nystagmus at the onset of the next run. In some control experiments only one light period was presented per day, without effect on the results.

Horizontal and vertical eye positions were stored on FM tape along with the first derivative of horizontal eye position (velocity), cylinder velocity, a time marker for lights on and off, and a digital time code. Data were later written out on a rectilinear oscillograph. All measurements were made from these paper charts.

Eye movements were calibrated using optokinetic nystagmus. For pattern velocities up to 60°/s the slow phase nystagmus velocity can be considered to equal stimulus velocity (Aschoff & Cohen, 1971). The gain of the EOG depends on the state of light or dark adaptation (Arden & Kelsey, 1962; Gonshor & Malcolm, 1971). Amplitude measurements of eye movements in experiments with changing luminance levels are therefore distorted. Since dark adaptation leads to a decrease of gain in the first minutes, the error introduced tends to attenuate our velocity measurements. No effort was taken to compensate for this attenuation or to use alternate ways of measuring eye position.

All velocity measurements showed a high correlation to the number of nystagmus beats per s (see Figs. 1 and 3).

RESULTS

In all monkeys OKAN I could be reliably elicited even with a stimulus exposure time of only 2 s. With respect to OKAN II there were marked differences between the monkeys. This was particularly obvious after stimulation periods of 15 min. In 4 monkeys OKAN I changed consistently in less than 60 s to OKAN II (see Figs. 1 and 2A), whereas in the remaining 4 monkeys this transition occurred at the earliest after 4 min (see Figs. 2B and 3) or not at all. To simplify the description the monkeys will be divided into 2 groups according to their onset of OKAN II after a 15-min stimulus exposure time.

Monkeys Showing Early Onset of OKAN II

Figure 1A shows the original record of a typical response to a 15-min optokinetic stimulation period. About 30 s after the light is turned off the direction of the nystagmus changes from OKAN I to OKAN II. The change is easily seen in the velocity trace where the baseline, indicating the velocity of the slow phase of nystagmus, crosses zero and the fast phases of nystagmus reverse their direction. The slow phase velocity of OKAN II reaches a maximum after an additional 75 s. This is paralleled by an increase in the number of nystagmus movements per second.

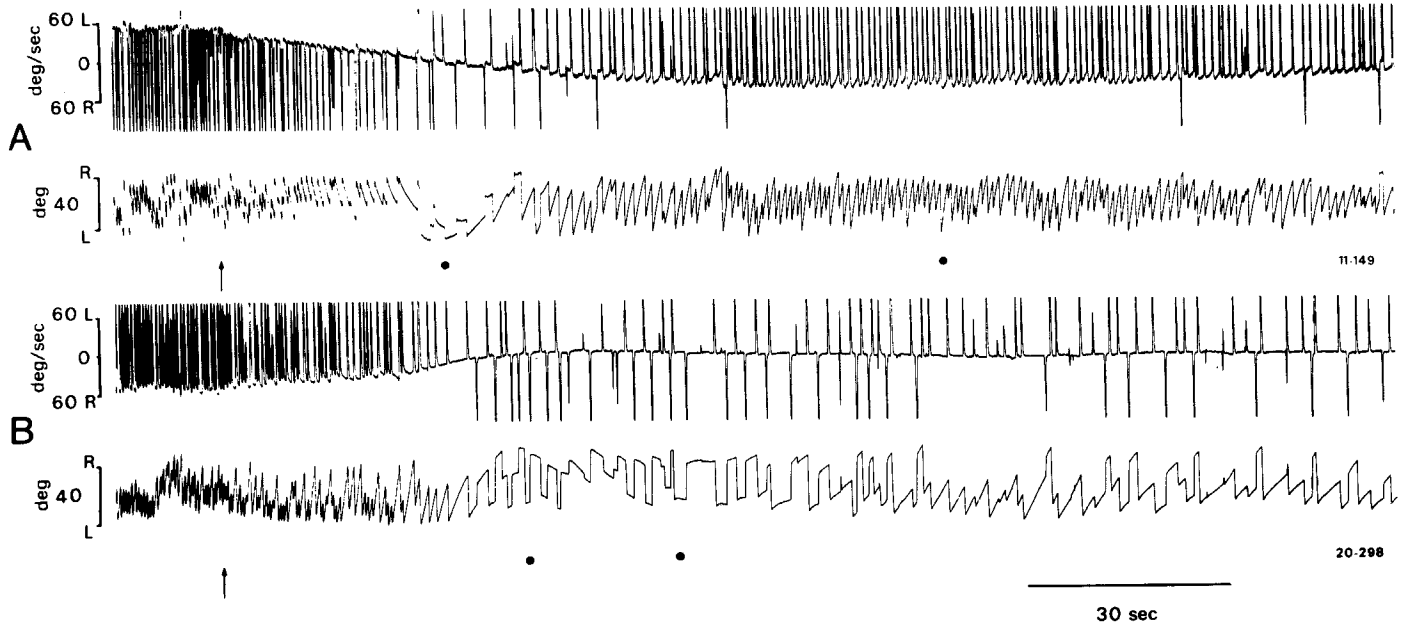

Fig. 1. Optokinetic after-nystagmus in 2 monkeys. A and B are original records taken from 2 different monkeys showing early onset of OKAN II after 15-min exposure to an optokinetic stimulus (pattern velocity $60^{o}/s$). Upper trace of two rows shows velocity of horizontal eye movements, displayed in lower trace. Velocity was obtained by differentiating eye position. Scales chosen only display show phase velocities, fast phase velocities are clipped off at an arbitrary level of about $80^{o}/s$. At upward arrow lights are turned off. Note that in A and B reversal of OKAN direction is accompanied by small change of field of movement (Schlagfeld). In A, onset of OKAN II is marked by first dot. It can be seen that with increasing slow phase velocity of OKAN II, number of left nystagmus movements increases. After reaching a maximum (marked by second dot) slow phase velocity decreases slowly to zero, i.e., spontaneous eye movements, after an additional 2 min (end not displayed here). In B, beginning and end of OKAN II are marked by dots. OKAN II is short and maximal slow phase velocity is small. In this case the nystagmus finally reverses again, i.e., from OKAN II to OKAN III, where nystagmus moves in same direction as during OKAN I

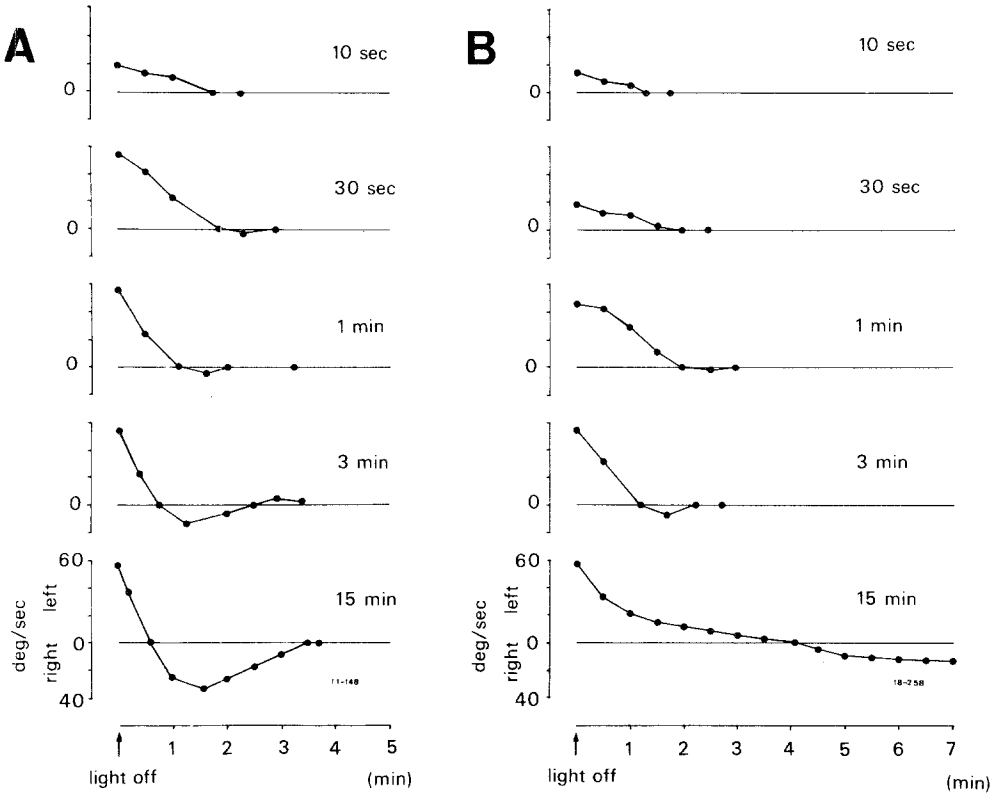

Fig. 2. Influence of exposure time on OKAN for monkey showing early (A) and late (B) onset of OKAN II after 15-min stimulus exposure time. Abscissa is time after "light-off" and ordinate, slow phase velocity of horizontal eye movements. Dots represent measured values, interconnected by straight lines. Times on right refer to preceding stimulus exposure time. In A, first indication of OKAN II seen after 30 s light period. With longer exposure times, onset of OKAN II is earlier and amplitude of OKAN II slow phase velocity increases. Note that after 3-min exposure OKAN II is followed by OKAN III. In B weak and short lasting OKAN II seen after 1- and 3-min exposure time, whose onset is much earlier than after 15-min exposure time. End of OKAN II for 15-min exposure time, which lasted for additional 12 min, not shown here

The time course of OKAN was greatly influenced by the preceding light exposure time. With short light periods (2-10 s) only OKAN I could be elicited. An increase in exposure time led first to an increase of the duration of OKAN I. Only after stimulus exposure times of between 30 s and 3 min (varying between monkeys and trials) was OKAN I followed by OKAN II. A further increase in exposure time most reliably altered two parameters of OKAN: the duration of OKAN I decreased, and the maximal slow phase velocity of OKAN II increased. Figure 2A shows the changes of OKAN

Table 1. Influence of exposure time on the duration of OKAN I and the maximal slow phase velocity of OKAN II. Average values from 15 experiments using 4 monkeys. S_D = Standard deviation

Exposure time	Duration OKAN I	Max. slow phase velocity OKAN II
1 min	76 s ($S_D = \pm 22.1$ s)	5^o/s ($S_D = \pm 4.1^o$/s)
3 min	45 s ($S_D = \pm 11.7$ s)	12^o/s ($S_D = \pm 7.0^o$/s)
15 min	33 s ($S_D = \pm 10.5$ s)	24^o/s ($S_D = \pm 10.7^o$/s)

with respect to exposure time. The longest light period (15 min) is followed by the shortest OKAN I and the highest slow phase velocity of OKAN II. This is further demonstrated by Table 1, which shows the average values for 4 monkeys. All cases of OKAN II were preceded by a period of OKAN I, which in no case was found to be shorter than 18 s. After 15-min stimulus exposure the maximum slow phase velocity of OKAN II was reached 83 s (SD 35.1 s) after lights off. Following this maximum in some cases OKAN II decreased continuously and after 1-3 min spontaneous eye movements occurred again. In other instances OKAN II outlasted the subsequent 10-min recording session. In the remaining cases another reversal of OKAN could be observed. Thus after OKAN II there was a transition back to a nystagmus with the same direction as OKAN I, which should therefore be classified as OKAN III (Koerner & Schiller, 1972). Figure 1B shows an original record of OKAN III. In such cases the maximal slow phase velocity of the preceding OKAN II was small. Sometimes OKAN III could also be observed after a 3-min exposure time as shown in Figure 2A. OKAN III occurred following a 3-min, but not a 15-min, exposure time. The difference of OKAN II slow phase velocity between the two exposure times should be noted. If OKAN III occurred after 15-min exposure time it lasted between 1 1/2 and 3 min. Its maximum slow phase velocity was always below 20^o/s, a value similar to the velocity of its preceding OKAN II. In a few experiments OKAN I decreased to zero slow phase velocity, i. e., spontaneous eye movements were seen within 1-2 min, before OKAN occurred in the same direction again. In spite of the large variations in OKAN responses, the same monkey tended to show a similar response pattern to both directions of stimulation.

Monkeys in Which OKAN II Was Late or Absent

Figure 3 displays an original record from one of 4 monkeys in which the transition to OKAN II occurred very late, in this case after more than 6 min following a 15-min exposure time. The figure also shows that the decrease of OKAN I is not continuous, but leads to a definite minimum of slow phase velocity with fewer right nystagmus movements per s after 1 min and 45 s. After this minimum the velocity increases again and reaches

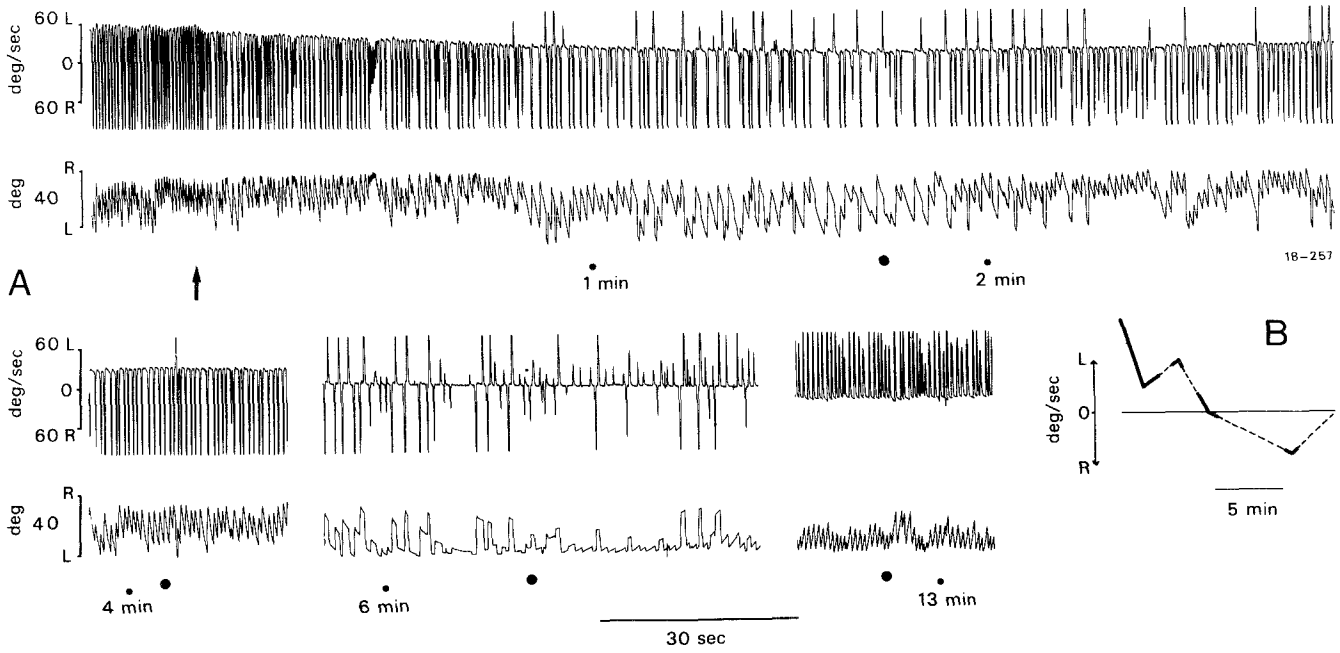

Fig. 3. Time course of OKAN in monkey with late transition from OKAN I into OKAN II. Monkey exposed for 15 min to an optokinetic stimulus (pattern velocity 60°/s) causing right nystagmus. In A, upper traces of two rows show velocity (first derivative) of horizontal eye movements, which are displayed in lower traces. The time after light is switched off (upward arrow) marked by small dots. Following 4 major points of OKAN changes are marked by large dots, respectively: a minimum of OKAN I velocity, another maximum of OKAN I velocity, transition from OKAN I to OKAN II, and maximum velocity of OKAN II. It also can be seen that during OKAN, nystagmus tends to move in field (Schlagfeld) of fast phase. Insert (B) shows schematically time course of OKAN slow phase velocity, whereby periods displayed in A are marked by heavy lines

a maximum before the transition to OKAN II occurs. The OKAN II maximum is only reached after 13 min with a slow phase velocity still 30°/s. In these monkeys a definite OKAN I minimum between 1 and 4 min was quite common and in a few cases even a second minimum could be observed before the slow phase velocity dropped to zero or changed into OKAN II. It should be noted that nystagmus was brisk and regular even during minima (Fig. 3), so there was no indication that these slow phase velocity changes were caused by changing levels of alertness. As mentioned earlier, in these monkeys OKAN I lasted at least 4 min and sometimes outlasted the recording period after a 15-min stimulus exposure. When OKAN II was observed it lasted up to 15 min, could reach slow phase velocities up to 40°/s, and sometimes also showed a clear second maximum.

Shorter exposure times usually only produced OKAN I, but in some instances exposures of 1 or 3 min led to OKAN II after 1-2 min (Fig. 2B). At this time if anything only a slow phase velocity minimum of OKAN I was seen, when these monkeys were exposed to longer (15 min) light periods.

The responses after left and right OKN were also similar for this group of monkeys, particularly with respect to OKAN I minima, and the transition to OKAN II. The closest similarities however, were again found after OKN in the same direction.

DISCUSSION

Comparison with Data Obtained from Humans

The existence of OKAN and its direction reversal is known from experiments on humans (Mackensen & Wiegmann, 1959), monkeys (Krieger & Bender, 1956; Komatzusaki et al., 1969; Koerner & Schiller, 1972), and rabbits (Collewijn, 1969). The effects of varying exposure times in monkeys are qualitatively similar to human data (Brandt et al., 1974). In monkeys with an early onset of OKAN II, OKAN I was on average twice as long as in humans. OKAN II in monkeys could only be elicited reliably after exposure times of 30 s or longer, whereas in humans OKAN II already occurred after 5 s. On the other hand, the maximal slow phase velocity of OKAN II was on average 3 times as large in monkeys as in humans. In several instances we found OKAN III (nystagmus movements in the same direction as OKAN I) occurring after OKAN II in monkeys. Such a reversal has not been described in humans, but was observed in the monkey under open loop conditions (Koerner & Schiller, 1972). In general these data confirm that OKAN is more prominent in monkeys than in humans (Ter Braak, 1936; Krieger & Bender, 1956). Brandt et al. (1974) do not mention response patterns in humans comparable to our second group of monkeys, in which OKAN II was late or absent.

Mechanisms Underlying OKAN

The results presented in this report show that the time course of slow phase velocity of OKAN I and OKAN II depends on the duration of the preceding OKN in a consistent manner. In 4 of the 8 monkeys, after an exposure time of 15 min, the direction of OKAN reversed in less than a minute. The others showed this transition either much later or not at all. It should be stressed

again that none of our monkeys had known neurologic deficits or spontaneous nystagmus in the dark. The OKAN pattern of each monkey had a similar response in both directions. This symmetry makes it unlikely that the different response patterns are due to an interaction with a supposed latent spontaneous nystagmus.

Several points of evidence suggest that OKAN is composed of two opposing mechanisms which normally add, leading first to OKAN I followed by OKAN II. This view is supported by experiments where OKAN I can selectively be inhibited without much effect on OKAN II. In humans Brandt et al. (1974) found that after fixation during long optokinetic stimulation OKAN II could occur without a preceding period of OKAN I. Zee et al. (1976) demonstrated that in patients without labyrinthine function OKAN I is diminished or absent, but OKAN II is still present.

Evidence that the vestibular system participates in the generation of OKAN was proposed on the grounds that its main function is to counteract vestibularly induced nystagmus during deceleration (Jung, 1948; Rademaker & Ter Braak, 1948). On an experimental basis, Cohen et al. (1973) report that after bilateral labyrinthectomy OKAN I could no longer be induced in monkeys. Also, OKAN shows an almost linear summation with postrotatory nystagmus (Cohen, 1974; Matsuo et al., 1976).

Our data could also be explained on the basis of two separate mechanisms: one driving the eyes in the same direction as the preceding OKN and the other in the opposite direction. They would both be enhanced by longer exposure times, often the opposing mechanism increasing more than the positive mechanism, which would then lead to OKAN II. It has to be further assumed that the decay of one or both mechanisms is not a monotone function and that an increase in exposure time does not always affect both mechanisms in the same relative amount. The OKAN resulting from such an interaction would account for several observations: OKAN III is seen after short stimulus exposure times, but disappears again after longer exposure times: and OKAN I and II show several maxima. In summary we feel that the differences between the monkeys are mainly due to quantitative differences of the two opposing mechanisms rather than to more basic differences.

It is not yet known which structures in the brain are responsible for these two different mechanisms. Brain stem lesions affect OKAN in monkeys (Shanzer, 1958; Uemura & Cohen, 1973), but only the effects on OKAN I were studied. Single unit studies in the vestibular system of several species show that first and second order neurons (Klinke & Schmidt, 1970; Dichgans & Brandt, 1972; Dichgans et al., 1973; Henn et al., 1974) and higher order neurons in the thalamus (Büttner & Henn, 1976) are influenced by optokinetic stimuli. Investigations using similar stimuli as reported here while recording single units in the vestibular nuclei, show that most units exhibit activity changes which parallel strength of the afternystagmus (Waespe & Henn, 1976). However, available data are still insufficient to explain all the phenomena of OKAN on a unitary level.

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