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Reciprocal Changes in Primary and Secondary Optokinetic After-Nystagmus (OKAN) Produced by Repetitive Optokinetic Stimulation in the Monkey*

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Summary. In six rhesus monkeys *(Macaca mulatta)* the effect of repetitive periods of whole-field optokinetic stimulation upon the different phases of optokinetic after-nystagmus (OKAN) was studied. The preceding optokinetic stimulus consisted of rotating a striped cylinder around the stationary monkey. Experiments were performed on up to 8 successive days. The results demonstrate that OKAN I and OKAN II are affected in a reverse manner by repeated optokinetic stimulation: The duration of OKAN I strongly decreases, whereas OKAN II increases in duration and intensity. The mechanisms underlying the generation of OKAN I and OKAN II and the role of OKAN II in the habituation process are discussed.

Key words: Optokinetic after-nystagmus – Habituation – Monkey.

Zusammenfassung. Bei sechs Rhesusaffen *(Macaca mulatta)* wurden die Effekte repetitiver optokinetischer Reizung auf die verschiedenen Phasen des optokinetischen Nachnystagmus (OKAN) untersucht. Ein vertikales, schwarzweißes Streifenmuster wurde an 6—8 aufeinanderfolgenden Tagen mehrere Male um den stationären Affen gedreht. Die Befunde zeigen entgegengesetzte Effekte optokinetischer Reize auf den primären (OKAN I) und den sekundären OKAN (OKAN II): Während der OKAN I stark verkürzt wird, nimmt der OKAN II sowohl an Intensität wie auch an Dauer zu. Die für die Bildung von OKAN I und OKAN II verantwortlichen Mechanismen sowie die Rolle des OKAN II bei der Habituation werden diskutiert.

Schlüsselwörter: Optokinetischer Nach-Nystagmus – Habituation – Rhesusaffe.

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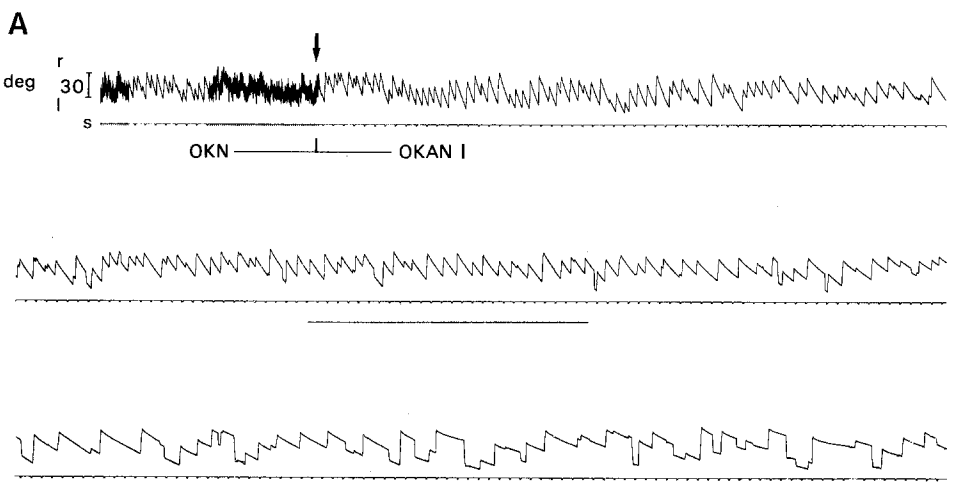
Introduction

Optokinetic nystagmus (OKN) is the oculomotor response to a moving full-field visual pattern. It is followed by optokinetic after-nystagmus (OKAN) in complete darkness (Ohm, 1927; Ter Braak, 1936). Primary OKAN (OKAN I) beats into the same direction as the preceding OKON. Secondary OKAN (OKAN II), i.e., nystagmus beating into the opposite direction, and tertiary OKAN (OKAN III) have also been observed in monkeys (Koerner and Schiller, 1972; Büttner et al., 1976).

In a preceding paper the effects of stimulus exposure time on the duration and intensity of the different phases of optokinetic after-nystagmus were studied (Büttner et al., 1976). Results in monkeys were very similar to those previously reported in humans (Brandt et al., 1974). The aim of the present study was to characterize the different phases of after-nystagmus in response to repetitive optokinetic stimulation. It was expected that changes that have variously been described as habituation would occur after the same stimulus was applied repetitively. The findings support the hypothesis that during optokinetic stimulation two opposing tonic activities build up, leading first to OKAN I and then to OKAN II (Brandt et al., 1974; Zee et al., 1976; Waespe and Henn, 1977b).

Methods

Nystagmus was recorded in rhesus monkeys (*Macaca mulatta*) chronically prepared with bolts in the skull to fix the head in a position tilted 25° forward (further details: Waespe and Henn, 1977a). Eye position was monitored with chronically implanted DC electrodes (Bond and Ho, 1970). Alertness was increased by injection of amphetamine (0.3—0.5 mg/kg i.m.) 20 min before stimulation. Animals were seated in a primate chair that was enclosed by a cylinder (diam 124 cm, height 86 cm) and covered with vertical black and white stripes, each 7.5° wide. Monkeys were tested on consecutive mornings with optokinetic drum rotation at 60°/s for 3 min. Then the lights were turned off and the monkeys maintained in complete darkness for the next 4 min, during which OKAN occurred. Then lights were turned on again for 2 min and no stimulation



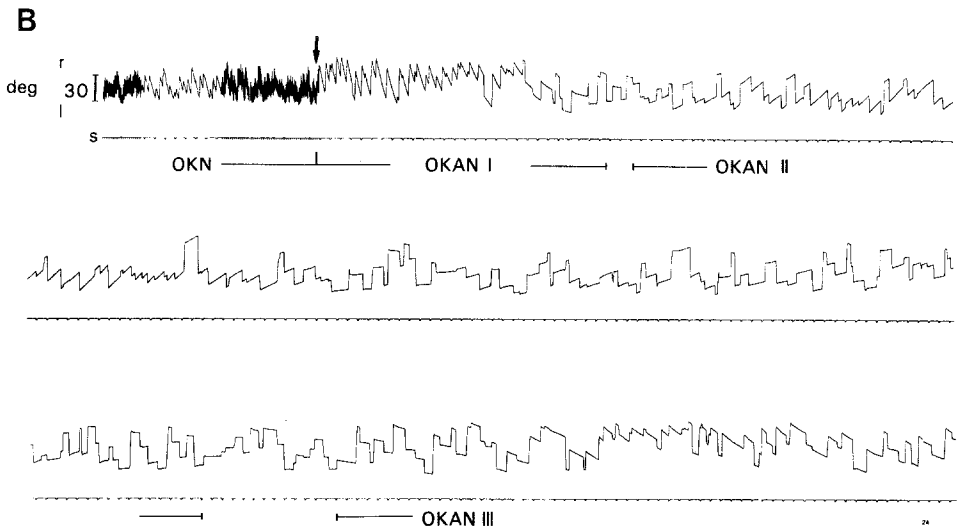


Fig. 1A and B. OKAN in monkey 24. **A:** First stimulation on the first day. **B:** Last (fifth) stimulation on the fifth day. In between were 23 stimulations in both directions. Only horizontal eye movements are displayed. *Arrow* marks switching lights off, i.e., beginning of OKAN I. Note the change of time basis. In A only OKAN I occurs lasting more than 240 s. In B, OKAN I is followed by OKAN II after 29 s. Observe the temporary slowing of OKAN II in the middle of the second row. After 140 s of OKAN II the direction of the nystagmus changes again to OKAN III

given. Before the next trial was started, the lights were turned off again for 1 min to check for the occurrence of any spontaneous nystagmus. A complete cycle lasted 10 min and was repeated 5—8 times each day on 6—8 successive days. The direction of optokinetic stimulation was changed every day. Measurements were made from data written out on a rectilinear oscillograph.

For analysis the following parameters were considered:

a) OKAN I. Duration

b) OKAN II. Peak slow-phase velocity and the time of its occurrence. Nystagmus slow-phase velocity was measured by differentiating the eye position signal. Eye movements were calibrated from optokinetic nystagmus response. For velocities up to 60°/s, the slow-phase nystagmus velocity can be considered to equal stimulus velocity (Cohen et al., 1977). The gain of the EOG depends on the state of light or dark adaptation (Kris, 1958). No effort was made to compensate for the error introduced by changing illumination levels. The duration of OKAN II was measured if it did not outlast the recording period of 4 min.

c) OKAN III. If OKAN III occurred, its maximal slow-phase velocity and duration were measured.

Results

Spontaneous Nystagmus

Monkeys were not selected for response symmetry and initially only two monkeys had no spontaneous nystagmus in the dark. In the other four monkeys, spontaneous nystagmus was less than 2°/s. By the last trial of each day there was

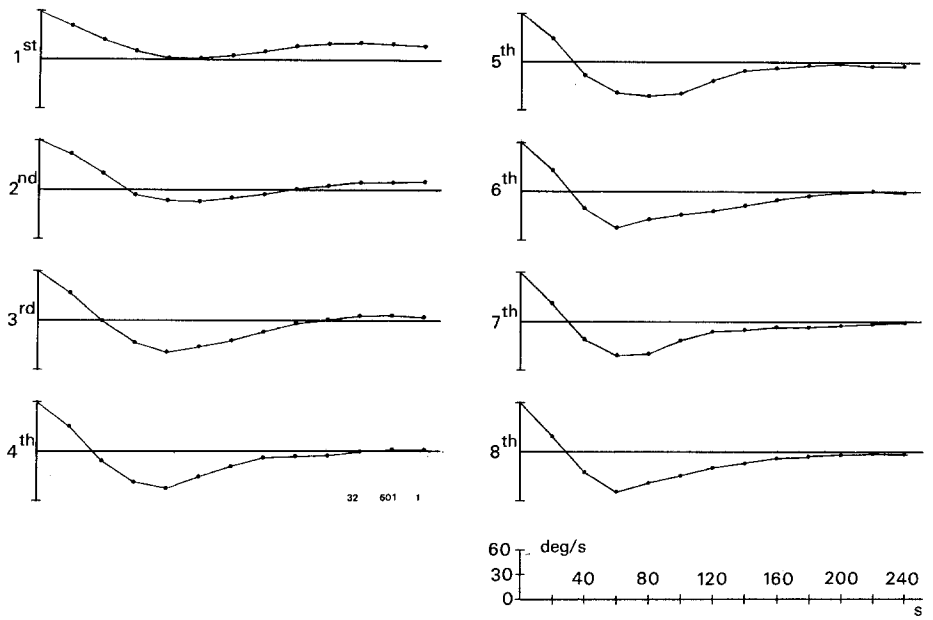


Fig. 2. Effect of eight optokinetic stimulations upon OKAN on the first day in monkey 32. Slow-phase nystagmus velocity is measured every 20 s (ordinate). Points above horizontal base line refer to OKAN I or OKAN III, below base line to OKAN II. Note that in the first trial OKAN I shows a minimum after 90 s without appearance of OKAN II. In the second trial this minimum corresponds to the maximum of OKAN II, which is later followed by OKAN III

usually spontaneous nystagmus in the direction opposite to that of optokinetic stimulation. This spontaneous nystagmus was on the average 5°/s (range 0—11°/s). For measurement, the velocity of spontaneous nystagmus immediately preceding individual experiments was subtracted from the peak of OKAN II velocity.

Primary OKAN (OKAN I)

In all monkeys, repeated exposure to optokinetic stimulation led to a reduction of OKAN I, not only over several days (Fig. 1 A and B), but also within the same day (Fig. 2). In several monkeys OKAN I initially outlasted the recording period of 240 s, whereas finally it was always reduced to 20—40 s. Initially the long-lasting OKAN I either showed a continuous decrease of slow-phase velocity or exhibited a minimun after 80—160 s and then increased again without appearance of OKAN II (Fig. 2). The duration of OKAN I in the first trial of a given day compared to the last trial 2 days earlier (rotation into the same direction) was usually longer, suggesting a partial recovery. This is shown in Figure 3 for one monkey. Monkey 21 had received repetitive optokinetic stimulations 3 weeks earlier. In the present series of experiments this monkey initially had an OKAN I lasting only 25 s (stimulation to the right) or 19 s (stimulation to the left). Figure

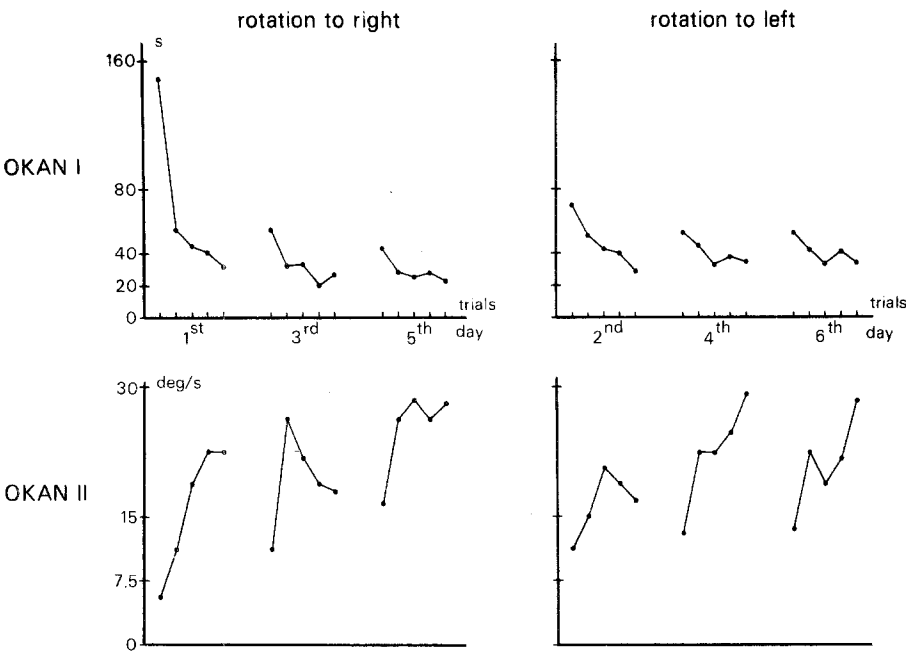


Fig. 3. Duration of OKAN I and peak velocity of OKAN II for all trials in monkey 22. On the first day in the first trial OKAN I lasted for 147 s followed by only weak OKAN II. Note the strong reduction of OKAN I between the first and third trial on each day. OKAN in the last trial of a given day is usually shorter and secondary OKAN stronger when compared with the first trial 2 days later

4 A shows the reduction of the duration of OKAN I between the first and the last trial, averaged over the whole experimental program for each monkey. The strongest changes occurred between the first and third trial on each day.

Secondary OKAN (OKAN II)

Initially OKAN II was absent in some monkeys, but with repeated exposure to optokinetic stimulation a strong OKAN II developed in all monkeys. Figure 4B shows the values of peak slow-phase velocity averaged over all days. Two monkeys had a rather strong OKAN II with velocities between 40—50°/s. As in OKAN I, the most marked changes occurred between the first and third trials. With further trials on the same day the changes were only moderate. The repetition of stimulation increased not only the intensity of OKAN II, but also its duration, and maximum slow-phase velocity occurred earlier.

Tertiary OKAN (OKAN III)

Initially OKAN III was always absent. It was found sometimes to appear and disappear again with repetition of optokinetic stimulation, depending on the

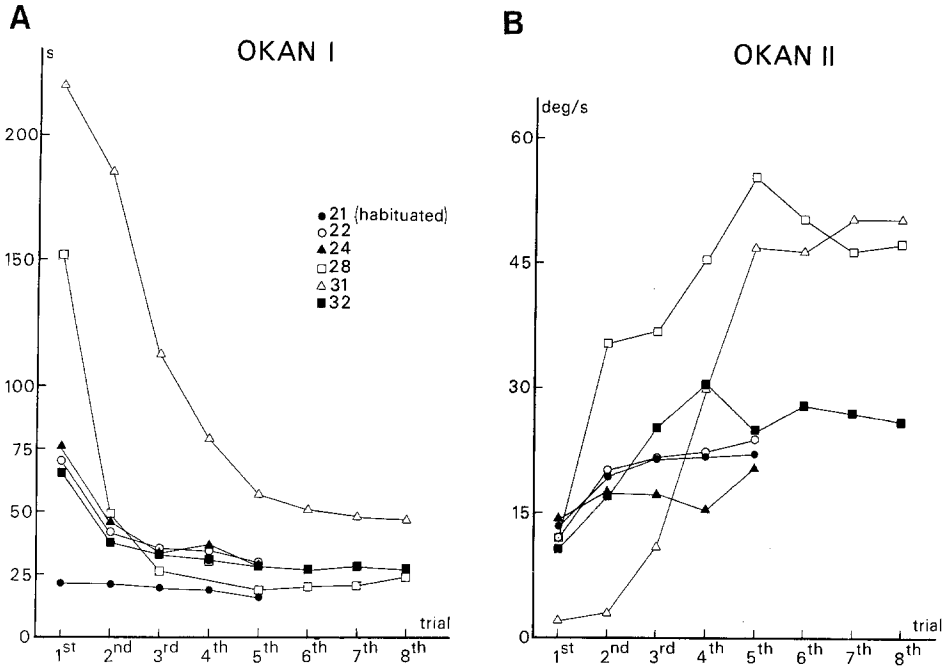


Fig. 4A and B. Effects of repetitive optokinetic stimulations upon duration of OKAN I (**A**) and upon peak slow-phase velocity of OKAN II (**B**) in all monkeys averaged over all days. Monkey 21 had experienced optokinetic stimulations 3 weeks earlier. Number of trial on each day is given on the abscissa. In A, average values ($n=6$ for monkeys 21, 22, 24, and 28; $n=8$ for monkeys 31 and 32) of the duration of OKAN I; in B, peak slow-phase velocity of OKAN II. The strongest changes in the duration of OKAN I and in the peak slow-phase velocity of OKAN II occurred between the first and the third trial

intensity and duration of OKAN II. OKAN III could be seen only after a low velocity OKAN II (Figs. 1 and 2). It is uncertain whether strong OKAN II masked the development of OKAN III or whether longer recording periods would finally always have led to the appearance of OKAN III. OKAN III was seldom followed by a further reversal of nystagmus.

Discussion

The Appearance of OKAN

All monkeys showed a primary and a secondary OKAN and sometimes also a tertiary OKAN to repeated optokinetic stimulation. The direction-reversal of nystagmus from OKAN I to OKAN II is known from experiments on humans (Mackensen and Wiegmann, 1959), monkeys (Krieger and Bender, 1956; Komatsuzaki et al., 1969), and rabbits (Collewijn, 1969). Tertiary OKAN can be found in normal monkeys, depending on stimulus conditions (Büttner et al., 1976). Brandt et al. (1974) have hypothesized that two opposing tonic activities

are generated during optokinetic stimulation. After stimulation terminates, these two opposing activities first lead to OKAN I and then to OKAN II. The tonic activity responsible for OKAN II was interpreted as a central response to optokinetic stimulation and not to OKAN I. Our results support this hypothesis by showing that repeated exposure to a moving visual pattern affects primary and secondary OKAN in a reverse manner: OKAN I shows a response decrease whereas OKAN II shows an increase. If OKAN II depends on the occurrence and strength of OKAN I, both OKAN I and OKAN II should show a similar response decline. The decrease of long-lasting OKAN I was frequently not monotonic, but showed a minimum of slow-phase velocity after 80—160 s and increased again afterward. With repetitive stimulation this minimum corresponded to the maximum of OKAN II. The occurrence of OKAN III can then be viewed as a rebound in the direction of OKAN I.

Preliminary experiments in our laboratory, during which the stimulus direction was kept constant, showed that monkeys develop a marked spontaneous nystagmus opposite to the stimulus direction. Thus, to keep interference with spontaneous nystagmus at a minimum, it was decided to change the stimulus direction daily. However, it would be desirable to have another series of repetitive and unidirectional optokinetic stimulations to study whether OKAN changes are basically the same and whether there is any influence on the response in the other nonhabituated direction.

Habituation of Nystagmus

The term 'habituation' is generally used to indicate a 'response-decline' (Hallpike and Hood, 1953) to repetitive stimulation. There is vast literature on the effects of repeated rotation on vestibular nystagmus. Despite differences in experimental procedures, it is generally accepted that the primary as well as the secondary phase of vestibular nystagmus decreases with repetitive stimulation (Hood and Pfaltz, 1954; Crampton, 1964; Collins, 1973 and 1974; Jeannerod et al., 1976). Besides 'fatigue' and 'learning,' other mechanisms have been proposed to explain these results (literature summarized in Howard and Templeton, 1966). If 'habituation' of vestibular nystagmus (VN) and OKAN are compared, the primary phase of both VN and OKAN decreases with repeated stimulation. However, for the secondary phase there seems to be a marked difference: Secondary vestibular nystagmus also decreases, whereas OKAN II increases. The results suggest that one of the mechanisms decreasing OKAN I during 'habituation' is the development of a stronger OKAN II (Brandt et al., 1974). Therefore, the secondary phase of nystagmus should not be neglected in studies of 'habituation,' since it may profoundly change primary nystagmus. It also means that to term 'habituation' as a simple response-reduction fails to describe the dynamic changes in the overall response (Collins, 1974).

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