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Motion Habituation: Inverted Self-Motion Perception and Optokinetic After-Nystagmus*

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Summary. The *oculomotor and perceptual after-effects* (AE) of optokinetic motion stimulation as well as the adaptive changes during stimulation were studied. The intensity and duration of optokinetic after-nystagmus (OKAN) and self-motion after-sensation (CV) are a function of stimulus duration. The direction is a result of two competing processes: positive tonus continuing the actual response to the stimulus and negative tonus causing a reversal in direction. Positive AEs increase with stimulus durations up to 1 min; the negative AEs increase up to the longest stimulus duration tested (15 min) and by antagonizing the positive AEs shorten their duration once stimulus duration exceeds 3 min. Negative AEs are interpreted as the consequence of a central counter-regulation to the actual stimulus effects: *motion habituation.*

During prolonged stimulation, motion habituation causes an apparent decrease in perceived velocity and may result in the sensation of periodic reversals of the direction of perceived self-motion, concurrent with a shift in average eye position towards the direction of the apparent reversal of self-motion, i.e. the nystagmic "Schlagfeld" reverses from the normal rapid-phase-side to the slow-phase as does also the CV.

Oculomotor and perceptual AEs similarly depend on stimulus qualities (e.g. duration, area of the moving stimulus and its location in the visual field). It is argued that positive AEs are due to an imbalance induced in the vestibular nuclei by the stimulus, and it is hypothesized that motion habituation acts upon the vestibular nuclei.

Key words: Motion habituation — Optokinetic after-nystagmus — Circular vection — Man

Introduction

After termination of visual motion stimulation *perceptual and oculomotor after-effects* usually occur. Perceived movement after effect and optokinetic after-nystagmus (OKAN) may be demonstrated after object referred motion perception (egocentric localization) as well as after self-referred motion perception (exocentric localization) when self-motion sensation is induced through relative movement of the seen environment (Fischer and Kornmüller, 1930; Brandt *et al.*,

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1971). The visual stimulus can create two perceptual interpretations. Equally the motion after-effect may be referred to visual objects or the observer himself.

Motion after-effects contain two components: 1. *Positive after-effects* which correspond in direction to the stimulus and are assumed to be due to after-activity in the central structures subserving optokinetic nystagmus (OKN) and self-motion perception and 2. *Negative after-effects* which are defined by their reversed direction and usually follow the positive after-effects. It will be shown that both inverted optokinetic after-nystagmus and inverted self-motion sensation can be interpreted as the result of a central counterregulation to the primary effects during actual motion stimulation. The intensity of the latter would allow a quantification of *motion habituation*. Since motion habituation requires time to develop one should expect the intensity of negative after-effects to increase with the duration of stimulus exposure while the intensity of positive after-effects, eventually counter-acted by motion habituation, might decrease. Thus in extension of earlier experiments (Brandt and Dichgans, 1972; Brandt *et al.*, 1973) motion habituation was investigated as a function of stimulus exposure time in terms of the duration and direction of OKAN and self-movement after-sensation. To us it seemed, however, even more challenging to trace out the functional significance of habituation during stimulation. We therefore attempted to determine its consequences on the sensation of self-motion during stimulation.

It will be shown that indeed perceptual and optomotor after-effects are determined by two stimulation time-dependent processes that compete with each other: an early fascilitation of positive after-effects that is antagonized by stronger and longer-lasting negative after-effects prominent after prolcnged stimulation. The relativeness of self-motion perception with respect to a given physical stimulus will be strikingly demonstrated by the finding of a periodical inversion of perceived motion. An adaptive shift of the internal scale for motion-perception including the zero point (impression of no motion) is also indicated by the finding of a decrease in the apparent velocity during stimulation and by the long-lasting negative after-effects on apparent self-motion.

Methods

1. Subjects

In the main series investigating the intensity of after-effects in relation to the duration of motion stimulation, five subjects, previously familiar with psychophysical experiments on motion perception, were tested repeatedly. In addition, 9 students took part in the experimental series concerned with the effects of restricted stimulus areas, discontinuously or sinusoidally oscillating optokinetic stimulation and supplementary investigations on inverted motion perception. They were paid for their participation.

2. Apparatus

Subjects sat on a rotatable chair located in the center of a closed cylindrical drum, 1.5 m in diameter, whose inner walls were painted with alternating black and white stripes each subtending 7° of visual angle. Both the chair and the drum could be rotated independently in either direction following either a constant velocity or a sinusoidal program. Angular velocities were stabilized by servocontrol. Accelerations, speed and angular position of both drum and chair were continuously recorded. Possible cues from sounds generated by the moving equipment were masked by white noise. The homogeneous illumination within the drum-cabin was controlled by the experimentor from outside.

The subject's head was restrained by a headholder. To stabilize the direction of their visual axis, subjects were asked to fixate on a 1° luminous spot presented at a distance of 60 cm straight ahead and connected to the chair.

Optokinetic stimulation by the moving drum could be restricted to any desirable spatial extent and presented at different locations within the visual field. This was achieved by using black masks which were mounted immediately adjacent to the inner wall of the drum. An *ambivalent optokinetic stimulus* providing contradictory motion stimuli to the center and to the periphery of the visual field was given by means of a mirror, 30° in angular diameter, in which the moving stripes reflected from the rear wall of the drum were seen to move in a reverse direction.

3. Recordings

Horizontal and vertical components of eye movements were separately recorded by means of electrooculography (Jung, 1953) and after DC-amplification were displayed on a strip-chart recorder. OKAN was recorded with the eyes open in complete darkness. Subjects were required to continuously indicate their self-motion sensation (circularvection, CV) by giving magnitude estimations (Stevens, 1957) of the apparent angular velocity through turning a potentiometer accordingly. This difficult task required a very high level of attentiveness. The tracking performance thus allowed for a control of vigilance by the experimentor continuously surveying the record.

4. Experimental Procedure

a) Determination of *direction* and *duration* of optokinetic after-nystagmus and subjective self-motion as a function of the *duration of optokinetic stimulation* (Ganzfeld) in the range of 5 sec to 15 min.

Drum speed was kept constant at an angular velocity of 60°/sec and as in the following experiments (b—e) stimuli were presented under two conditions. The subjects either pursued the pattern by means of optokinetic nystagmus (OKN) or fixated the stationary target which resulted in pattern movement across the retina. Within a single day only one of the five stimulus durations was presented under one of the two observation conditions. The resulting ten different experimental conditions were presented in random order once to each subject in daily intervals. The same five subjects were tested in two additional trials in which the five stimulus durations were presented successively starting with the short stimulation and separated by 5 min rest periods. One trial was given during steady fixation and the other during OKN.

b) Determination of *OKAN-intensity* in relation to *optokinetic stimulation* (60°/sec for 1 min) of either the *entire visual field*, a *central area* (30° or 60° in diameter), the *peripheral field* (central mask, 120° in diameter) or after *ambivalent stimulation* of the center (30°) and the remaining periphery of the visual field.

c) *Motion habituation during prolonged stimulation* (constant velocity 60°/sec, 15 min) by drum or chair rotation, respectively. Continuous monitoring of self-motion sensation and DC-recording of eye movement.

d) *Effects and after-effects* of *discontinuous stimulation:* stimulus velocity 60°/sec, stimulation periods alternating with rest periods in darkness lasting 5 sec each, total duration 10 min.

e) Investigation of the *after-effects of oscillating motion* periodically changing its direction, pendular motion of the drum at 0.05 or 0.1 Hz with a peak to peak amplitude of 300° or 180° respectively and a total duration of 1, 3 or 15 min.

Results

a) Optokinetic After-Nystagmus and After-Effects on Self-Motion Sensation as a Function of Stimulus Exposure Time

Kommerell and Thiele (1970) have shown that stimuli presented for a fraction of a second cause optokinetic after-nystagmus; the shortest stimulus duration presented in this study (that is concerned with the long-term effects on optokinetic stimulation) was 5 sec. With this stimulus duration one usually

observes not only optokinetic after-nystagmus (OKAN) but also a sensation of self-motion outlasting the stimulus. The direction of both after-effects is the same as during the preceding inspection, i.e. there are *positive after-effects*. The positive OKAN increases significantly in maximal slow phase velocity, total amplitude and duration with increasing stimulus durations up to 60 sec ($p2a \leqq 0.01$) and becomes weaker with a further increase of stimulus exposure time. With a 1 min stimulus exposure, the mean duration of positive OKAN was 36 sec and the mean total amplitude 137°, while after a 15 min stimulus exposure the mean duration significantly decreased to 17.2 sec with a total amplitude of 68°. In contrast to the positive after-effect, the reversed OKAN — *negative after-effect* — continues to increase in duration and total amplitude up to the longest stimulus exposure tested (15 min). Reversed OKAN finally reached an average duration of 37.4 sec and a total amplitude of 78° in our experiments. As depicted in the upper graph of Fig. 1, the latency of reversed OKAN decreases because of the reduced duration of positive OKAN. A schematic drawing of the possible interaction of positive and negative after-effects is presented as an insert in Fig. 7.

Optokinetic after-nystagmus also occurs if during stimulation the eyes are kept stationary by fixation, thus preventing optokinetic nystagmus during inspection of the moving drum (confirming the results of Mackensen and Rudolf, 1962). Obviously this condition results in pure afferent motion detection through moving retinal images (Dichgans *et al.*, 1969). As indicated in the lower part of Fig. 1 and in Table 1 positive OKAN again increases in duration and total amplitude up to stimulus durations of 60 sec but its weakening with longer stimulus duration is much more marked under this condition ($p2a \leqq 0.05$). Positive OKAN could not be observed after a 15 min stimulation, whereas the reversed OKAN started immediately after lights off and lasted an average of 45.6 sec with a total amplitude of 66°.

The *after-effects on self-motion sensation* with respect to the direction of previous CV and their time course mostly correspond to those observed for optokinetic after-nystagmus (Fig. 2).

Table 1. Total amplitude (degrees) of positive and negative OKAN as a function of stimulus duration

a) OKAN during stimulation

stimulus duration	5 sec	30 sec	60 sec	3 min	15 min
pos. OKAN	29	108	137	91	68
neg. OKAN	9	21	34	48	78

b) Fixation of a stationary target during stimulation

stimulus duration	5 sec	30 sec	60 sec	3 min	15 min
pos. OKAN	8	44	42	9	0
neg. OKAN	6	22	18	29	66

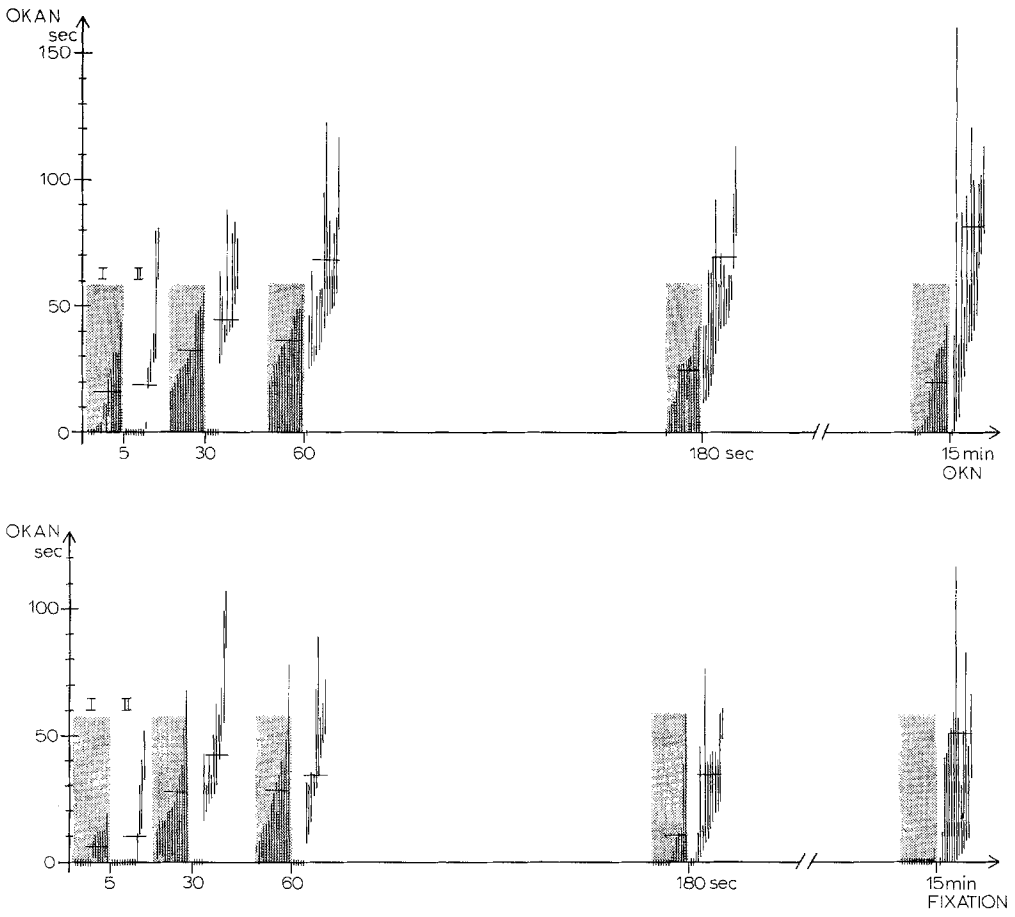

Fig. 1. Duration of optokinetic after-nystagmus (ordinate) in relation to stimulus duration (abscissa). The stimulus was observed either during optokinetic nystagmus (upper graph) or while fixating a small stationary target (lower graph). The individual durations of positive OKAN are depicted by the length of vertical lines within the shaded area (I); on the right of each shaded area the individual durations of negative OKAN usually following the positive OKAN are drawn (II). Latencies of negative OKAN are represented by the amount of upward displacement on the ordinate. Horizontal bars within I and II indicate respectively mean duration of positive OKAN and the mean of the latency plus the duration of negative OKAN

As can be seen from the lowest trace, both optokinetic after-nystagmus and circularvection reverse their direction almost immediately after termination of a 15 min stimulus exposure with the eyes kept stationary. Tracking the circularvection after-effect was particularly difficult at the very end of the fading self-motion sensation since it was hard to tell whether there was still a movement. Our data indicate that the direction of the *oculomotor and perceptual after-effects* of optokinetic stimulation are positively correlated. During stimulation the direction of OKN and CV may dissociate (Brandt *et al.*, 1973; Dichgans and Brandt, 1974).

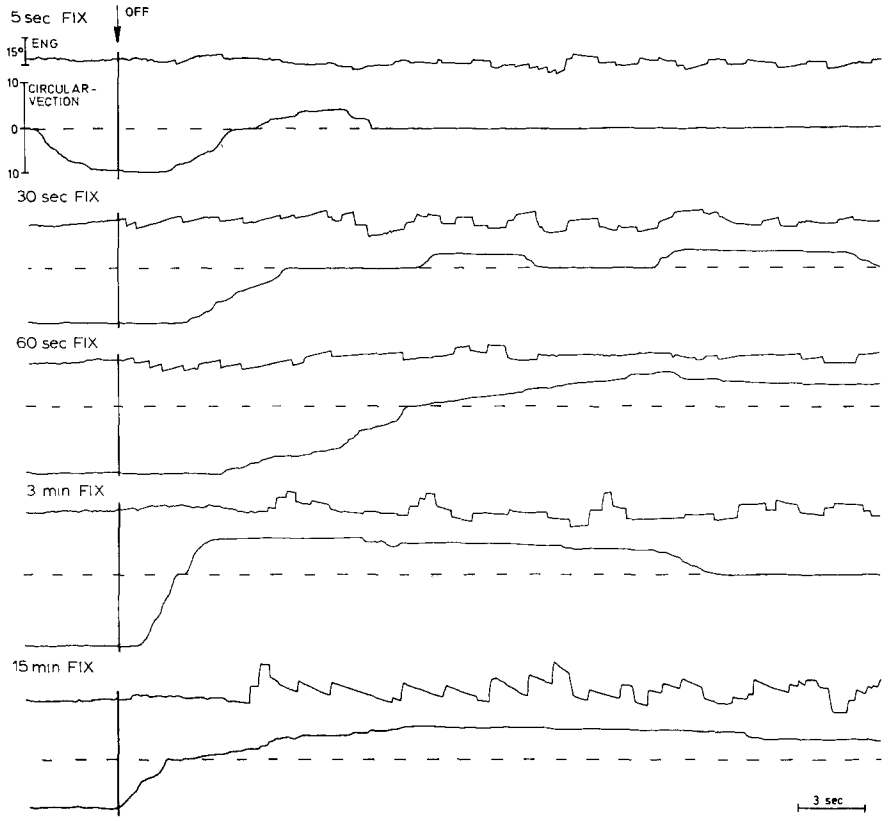

Fig. 2. Recordings of optokinetic after-nystagmus and visually induced after-sensation of self-movement, after termination of optokinetic stimulation (arrow) by moving retinal images exclusively (fixation) of different durations. Since the stimulus moved to the right, positive OKAN defined according to the direction of fast phases continues to the left and is accompanied by circularvection to the left. Positive after-effects increase in duration up to stimulus exposure times of 60 sec and then decrease after longer stimulations. Negative after-effects, however, are maximal after the longest stimulus duration

b) Optokinetic After-Nystagmus Following Central, Peripheral and Ambivalent Motion Stimulation

The effects of stimulus location within the visual field were examined while fixating a stationary target and holding stimulus duration constant (1 min). With this method OKAN was found to be maximal in intensity and duration when the moving stripes previously presented, filled the entire visual field. OKAN is, however, still present after exclusively stimulating the retinal periphery, which was achieved by masking off the centre of the visual field by a black cardboard subtending 120° of visual angle. Under this condition, OKAN intensity was less and duration shorter as compared to full field stimulation. Compared to the after-effect of peripheral stimulation, the OKAN following central and paracentral stimulation (area 30° or 60° in diameter), however, was even less regularly elicited and, when present, was weaker (Fig. 3).

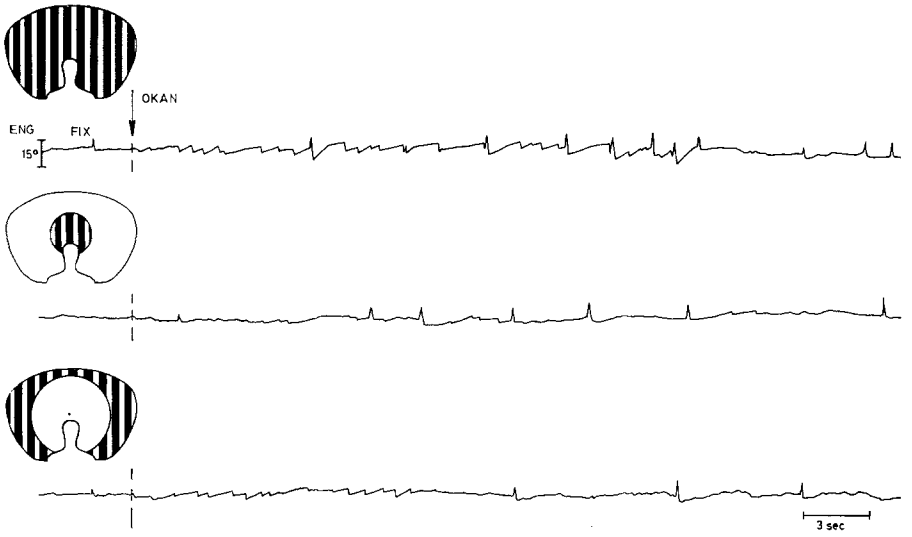

Fig. 3. OKAN in relation to stimulus area and location within the visual field. The records are taken from one subject. Nystagmus is clearly stronger when the retinal periphery was stimulated than after stimulation of the more central parts. The "spikes" mostly superimposed on the early part of rapid phases of nystagmus represent blink artefacts

Since the OKAN after fixation and with restricted stimulus area is weak and irregular, a comparison was not made of the intensity of OKAN for periphery and centre with matched areas. This task could probably be better studied in the monkey who has a much stronger OKAN response. Thus, it can so far only be concluded that in man as in the rabbit (Ter Braak, 1936) and monkey (Körner and Schiller, 1972) *exclusive stimulation of the periphery is able to elicit OKAN*. Stimulation of either the foveal or parafoveal regions can also elicit OKAN.

Ambivalent stimulation of centre and periphery with the two stimuli moving in opposite directions was chosen to demonstrate the two possible input components subserving OKAN: one being directly dependent on movement of the stimulus across the retina — possibly predominantly the periphery of the visual field — the other being contingent on the previous occurrence of OKN. If optokinetic nystagmus was allowed during stimulation, it was in the direction of the central stimulus and followed by a OKAN in the direction of the pursuit movement. If the optokinetic nystagmus was prevented by fixation, then the subsequent OKAN direction was determined by the peripheral stimulus and its intensity on the average exceeded that of OKAN in the former condition (Fig. 4).

Optokinetic after-nystagmus can indeed be driven by two processes: one contingent on the motor response and the other due only to the sensory motion information provided by moving retinal images. Under natural conditions the two different processes frequently work synergistically, but the ambivalent stimulation presented in the laboratory which causes antagonistic effects also occurs under real life conditions.

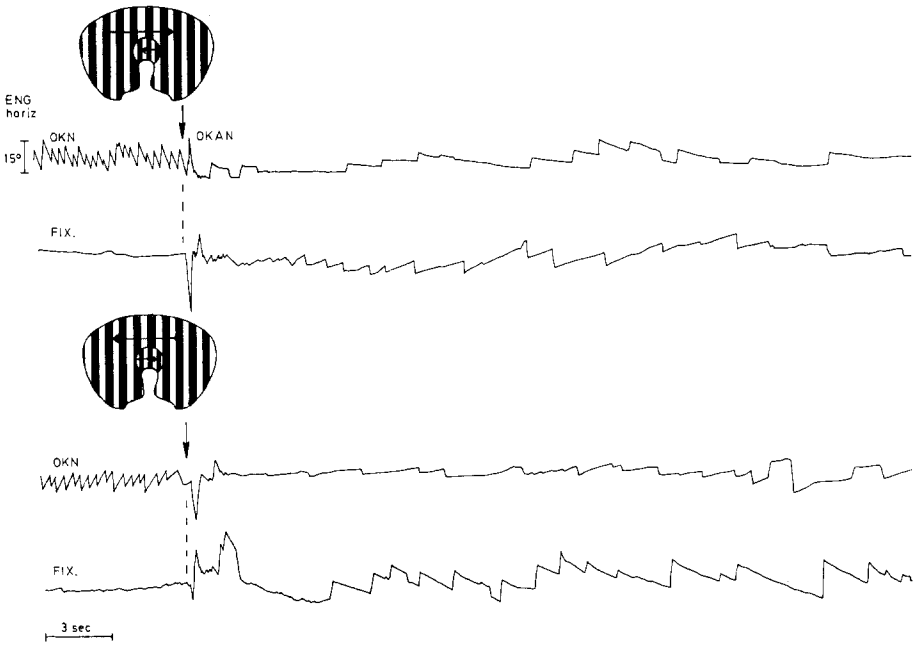

Fig. 4 Optokinetic after-nystagmus following ambivalent stimulation. If the pattern is viewed during optokinetic nystagmus (upper trace of each pair) which is driven by the central stimulus then the direction of after-nystagmus corresponds with its direction. If the pattern is viewed during fixation of a stationary point (lower trace of each pair) the much stronger after-nystagmus is determined by the peripheral stimulus moving in the opposite direction

c) Inverted Self-Motion Perception During Prolonged Optokinetic Stimulation

Habituation effects can be demonstrated after stimulus termination and also during optokinetic stimulation. During prolonged stimulation, the perceived velocity of visually induced circularvection on the average slows down. This can be well demonstrated by consecutive magnitude estimations, but is difficult to demonstrate in the records of continuous trackings of perceived CV speed because the changes are mostly rather slow. The intermittent faster and more distinct changes in CV speed which occur periodically, however, can be easily documented in tracking experiments using continuous magnitude estimations. After several minutes of a constant velocity chair- or drum rotation[1] perceived velocity of self-motion slows down and with still longer stimulation ceases for intervals lasting several seconds. Finally subjects periodically experience a reversal in direction of subjective self-motion (Fig. 5).

[1] With respect to the afferent sensory information, constant angular velocity chair rotation to the right is equivalent to surround rotation to the left since in both situations motion information is exclusively provided by the visual sense. The vestibular system detects only velocity changes and the cupula returns to resting position within a few seconds after termination of acceleration.

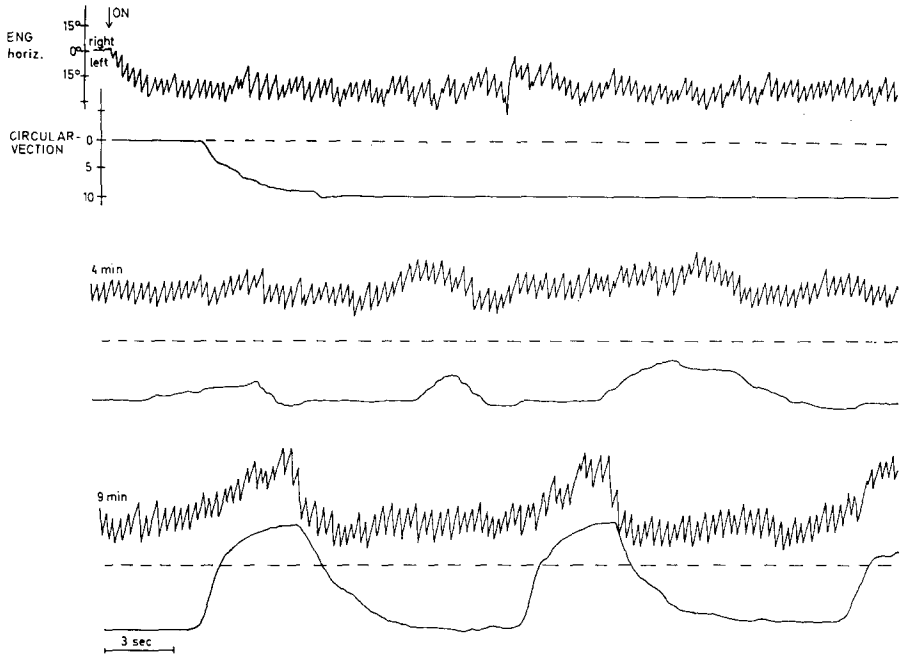

Fig. 5. Periodic breakdown and inverted motion perception concurrent with shift in average eye position, increasing in intensity with increasing stimulus duration. Inverted self-motion perception can be seen in the bottom trace

The latency of these reversals shows considerable inter-individual variability (ranging from 4—12 min). During the short paradoxical *reversals of self-rotation*, subjects feel themselves moving *in the direction of the moving surround* while the surround apparently rotates faster, as though it were dragging the subject's chair with it. Thus the entire apparatus including drum and chair seemed to move. The "*inverted self-motion perception*" as we would like to call it, is accompanied by a *shift in the average eye position* (Schlagfeld, Jung and Mittermaier, 1939) *of OKN from the side of the quick phase to that of the slow phase* (Fig. 5). A voluntary deviation of the OKN-Schlagfeld towards the direction of the slow phase, however, does not trigger an inversion of self-motion perception and active deviation back to the side of the quick phase does not destroy this illusion once it has occurred spontaneously.

Inverted motion perception (as it is generally the case with visually induced self motion perception) can be facilitated by fixation of a stationary light spot instead of pursuing the pattern by optokinetic nystagmus. Facilitation of inversion also occurs after a sudden decrease in actual drum velocity, after a stepwise decrease in illumination inside the drum, and when a small body acceleration in a direction opposite to that of the previously experienced CV is applied. Inverted self-motion perception may also be observed if CV is elicited by exclusive stimulation of the peripheral retina.

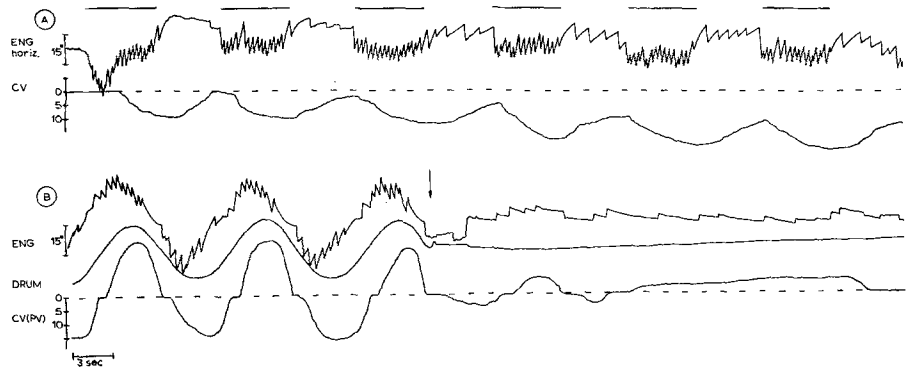

Fig. 6. Optokinetic after-nystagmus and circularvection during and between discontinuous motion stimulation (upper graph) and sinusoidal motion stimulation (lower graph). In A stimulus intervals are indicated by horizontal bars. In B the actual drum velocity is depicted by the middle trace. Note the marked deviations in average eye position during drum stimulation (analogous to the vestibulo-ocular reflex during sinusoidal body movement) and the occurrence of positive optokinetic after-nystagmus after termination (arrow) of 15 min of sinusoidal drum rotation

d) Self-Motion Sensation and After-Nystagmus in Discontinuous Optokinetic Stimulation

The after-effects of long-term stimulation can also be shown with discontinuous stimulus presentation. Subjects exposed to an optokinetic pattern whose motion subtends the entire visual field for a 5 sec period alternating with 5 sec dark intervals, experience the consequences of mutual interaction between the actual effect of stimulation and the after-effects originating from the previous stimulus. Over the first several cycles both after-nystagmus and CV are progressively facilitated during dark intervals. OKAN slow phases become steeper and frequency increases. The apparent velocity of on-going CV similarly increases during the first dark intervals. During stimulation intervals the after-effect of CV convincingly shortens the latencies until the entire motion is subjectively taken on by the observer[2] (Fig. 6a).

During stimulation periods, the perceived velocity of CV due to the biasing after-effect over the first few cycles may seem to progressively increase beyond stimulus velocity. When the discontinuous stimulation lasts more than 3—5 min, positive optokinetic after-nystagmus decreases progressively and CV rapidly stops after stimulus termination and eventually may reverse its direction.

Oscillating after-effects could not be produced after sinusoidal stimulations lasting 1, 3 or 15 min (Fig. 6b) neither with drum, (optokinetic stimulation) nor with chair-oscillations (vestibular stimulation) at the frequencies and amplitudes used (see methods). After-effects, when at all present where determined in their direction by the last stimulus phase.

[2] Similar facilitation of CV-latencies through simultaneous acceleration of the body represents an analogous phenomenon which is currently being investigated in cooperation with J. Allum. This vestibular facilitation is a precondition for allowing visual motion information to take part in dynamic spatial orientation, because of the resulting shorter latencies.

Discussion

Optokinetic After-Nystagmus (OKAN)

Optokinetic after-nystagmus, first described by Ohm (1921, 1922, 1927) is physiological in humans (Grüttner, 1939; McLay *et al.*, 1957; Mackensen and co-workers, 1959, 1961, 1962) but certainly more prominent in primates (Ter Braak, 1939; Krieger and Bender, 1956; Körner and Schiller, 1972) and rabbits (Ter Braak, 1936; Collewijn, 1970). The occurrence of optokinetic nystagmus is not a necessary precondition for after-nystagmus. OKAN may be observed after very brief stimulation, the duration of which is within the reaction-time of optokinetic nystagmus (Kommerell and Thiele, 1970) and also when a human subject fixates upon a stationary target while the stationary retina is exposed to a moving pattern (Mackensen and Rudolf, 1962).

In contrast to post-rotational vestibular nystagmus, OKAN beats towards the direction of the nystagmus during stimulation, whereas the deviation of average eye-position towards the direction of fast phase present during stimulation is rapidly restituted after stimulus termination (Fig. 6). As in vestibular nystagmus, OKAN may reverse its direction (Aschan and Bergstedt, 1955; Mackensen and Wiegmann, 1959; Collewijn, 1969; Körner and Schiller, 1972), a fact that, in analogy to the second post-rotatory nystagmus, has been interpreted as the result of central counter regulation by Kornhuber (1962). Our measurements of intensity of reversed OKAN in relation to stimulus duration confirm this interpretation and allow for quantitative statements about the time course of the habituative counter regulation.

So far the only information concerning this relationship stems from experiments in monkeys where Körner and Schiller (1972) found that both positive OKAN and reversed OKAN increased in intensity with increase in stimulus duration. Our data are consistent with this observation but indicate a more complex dependency of positive and negative after-effects upon the duration of stimulus exposure. Negative after-effects, within the range of exposure times tested, continue to increase in duration and total amplitude, whereas positive after-effects reach peak intensities after stimulus durations of about 1 min and may be absent if the stimulus was inspected with the eyes kept stationary through fixation for 15 min stimulus exposure. This finding, to our understanding, allows the conclusion that the negative after-effects prominent in this instance are independent of the occurrence of positive after-effects. Consequently, the central counter regulation does not — as it is the case with vestibular postrotatory nystagmus — just counteract OKAN but is the result of the perrotatory stimulation.

It also seems justified to conclude that at stimulus termination the optomotor response is determined by two opposite tendencies: the rapidly fading positive optokinetic tonus and the much longer lasting habituative counter-tonus. As long as the first outweighs the second, positive after-nystagmus is observed. At an intermediate period the two mechanisms may balance each other, at which time nystagmus disappears until with further decay of positive tonus the counter-regulation determines the reversal of OKAN. The time-course of slow

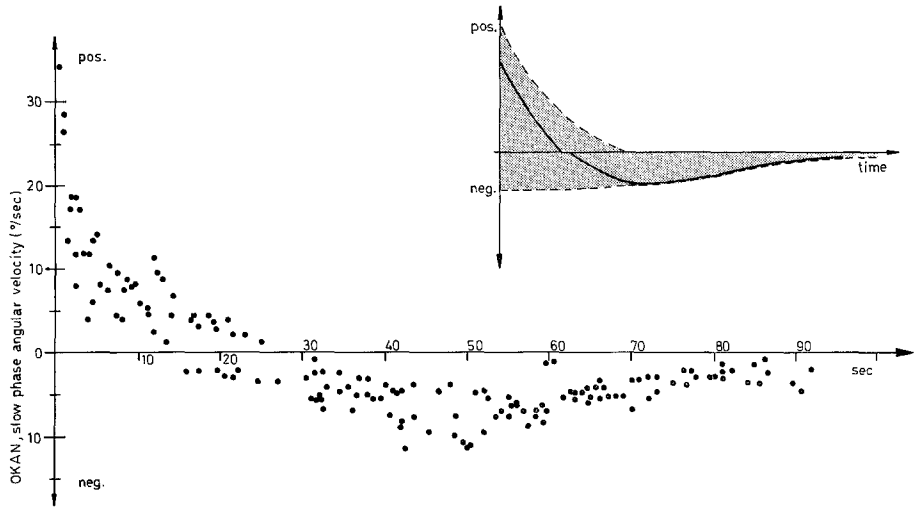

Fig. 7. Time course of angular velocity of individual slow phases of OKAN after a 15 min drum rotation. Data from 3 subjects are superimposed. The insert is a schematic drawing of the hypothetical interaction between positive and negative optokinetic after-tonus (dashed lines). The solid line represents the summation of the positive and negative components

phase velocity in positive and negative OKAN and a hypothetical scheme of their interaction are depicted in Fig. 7.

Moving retinal images by themselves may induce optokinetic after-nystagmus without the prior occurrence of optokinetic nystagmus as is demonstrated in the experiments where the pattern is inspected while the eyes are kept stationary during fixation. Nevertheless, optokinetic after-nystagmus is stronger if optokinetic nystagmus occurred, an instance where image motion on the retina is minimized. Thus one may conclude that the optokinetic tonus (positive as well as negative) has two sources: firstly retinal image motion and secondly optokinetic nystagmus itself. The existence of two separate mechanisms may be easily visualized by the results of ambivalent stimulation (Fig. 4). With this the after-nystagmus is determined by the central stimulus as long as optokinetic nystagmus is performed. But somewhat stronger nystagmus opposite in direction driven by the oppositely moving peripheral stimulus is obtained when the subject fixated during stimulation. In addition, it may be noted that stimulation of the peripheral visual field may evoke optokinetic after-nystagmus although, in the presence of an oppositely moving central stimulus, optokinetic nystagmus cannot be evoked through peripheral stimulation.

The structures by which optokinetic nystagmus and after-nystagmus are driven are not known and may in part be different. In man, velocity adequate optokinetic nystagmus certainly depends on cortical structures as it may be concluded from disturbances of optokinetic nystagmus observed in patients with frontal, parietal, temporal and occipital hemispheric lesions (Jung and Kornhuber, 1964). Impaired OKN, however, is also observed in patients with brain-

stem lesions affecting the supra-nuclear oculomotor structures in the pontomesencephalic reticular formation (Teng *et al.*, 1958; Jung and Kornhuber, 1964). Brainstem lesions also affect the OKAN in monkeys (Shanzer *et al.*, 1968).

Since Ohm (1929) and Spiegel and Teschler (1929) it has been repeatedly postulated that the vestibular nuclei participate in optokinetic nystagmus. This assumption has recently been substantiated by Cohen and co-workers who found that after labyrinthectomy maximal slow phase velocity of OKN was markedly reduced (Cohen *et al.*, 1973), and demonstrated through bilateral flocculectomy that an even stronger impairment results from interference with the vestibulo-archicerebello-vestibular loop (Takemori and Cohen, 1974). However, after labyrinthectomy no change in maximum OKN capability was observed by Westheimer (1974). The results of partial lesions within the vestibular nuclear complex were less conclusive (Uemura and Cohen, 1972). Within the context of our work it seems particularly interesting to know that after bilateral labyrinthectomy monkeys permanently loose their ability to perform optokinetic after-nystagmus (Uemura and Cohen, 1972; Cohen *et al.*, 1973).

Our results seem to be consistent with the assumption of an important *contribution of the vestibular nuclei to optokinetic after-effects*. It has been shown that the excitation of vestibular neurons through the visual sense alone is at least one of the possible neurophysiological mechanisms on which the visually induced self-rotation sensation is based. In the vestibular nuclei a direction-specific modulation of spontaneous activity of second order neurons has been demonstrated in the stationary animal (Dichgans and Brandt, 1972; Leopold *et al.*, 1973, in the rabbit; Dichgans *et al.*, 1973, in the goldfish; and Henn *et al.*, 1973, in the monkey).

The visually elicited sensation of self-motion as well as the visually induced excitation of units in the vestibular nuclei outlast the termination of the visual stimulus. The after-sensation of self-motion and optokinetic after-nystagmus correspond in direction and exhibit a similar time-course, a fact suggesting that both are driven by excitation of a common structure which we assume, resides in the vestibular nuclei. These have intimate connections to the oculo-motor nuclei. Further experimental evidence is presented by the fact that both the perceptual and oculo-motor after-effects similarly depend on the stimulus parameters tested (stimulus duration, dominance of retinal periphery). A close relationship between self-motion sensation and oculomotor activity is finally revealed by the shift in average eye position that occurs concurrent with the inversion of self-motion perception during longterm visual motion stimulation (Fig. 5). The occasional observation of very weak optokinetic after-nystagmus, without the subjects reporting the experience of self-motion, does not vote against this concept since the oculomotor consequences and the perceptual phenomena might have different thresholds (Brandt and Dichgans, 1972). This has been shown for vestibular nystagmus and the perception of actual body rotation (Groen, 1960).

We like to stress that the after-effects of object referred motion, e.g. the spiral after-effect (Holland, 1965) are different from those following self-referred motion perception. They invariably are of reversed direction and certainly originate from different structures residing in the visual rather than the vestibular system (Barlow and Hill, 1963; Richards and Smith, 1969; Walls, 1953).

Habituation of Self-Motion Sensation

The central counter-regulation to continuous motion-stimulation which can be termed motion-habituation becomes not only evident in motion after-effects but may also be experienced as an apparent reduction in velocity during stimulation. This phenomenon may be regularly observed after long rides in fast driving vehicles, is well decomented in our tracking experiments with discontinuous stimulation, and has even been observed with stimuli lasting only 30 sec (Goldstein, 1957). Since the habituative changes are so slow, they can only be detected by discontinuous magnitude estimation. A very peculiar habituative effect, also occasionally observed in real life, has first been documented and investigated in this study. This second effect consists in periodical reversals of the direction of apparent self-motion that are the more likely to occur the longer the stimulation lasts. The intermittent occurrence of inverted motion perception may be interpreted as the result of rivalry between the actual stimulus effect and the habituation. The concurrent shift in average eye position may hint to the site where this competition occurs. It may be interesting to note that the average position of OKN (Schlagfeld) now not any longer corresponds to the stimulus itself but to the perceived direction of body-movement.

The circuits by which habituation is generated are unknown. It is tempting to speculate about a possible role of the flocculus which is known to receive primary and secondary vestibular fiber projections through mossy fibers (Brodal and Hoivik, 1964; Precht and Llinás, 1969) and receives visual input (Maekawa and Simpson, 1972) via climbing fibres and via the inhibitory output of Purkinje cells projects back to the vestibular nuclei (Baker *et al.*, 1972; Fukuda, *et al.* 1972; Precht, 1972; Ito *et al.*, 1973).

We view our results as one example of the plasticity of the central nervous system which manages not only to adapt its motor outputs to rearrangement of the perceived world (Held and Hein, 1963; Gonshor and Melvill-Jones, 1971) but also the perception itself (Kohler, 1956). In the case of motion habituation, the counter-regulation acts as to recreate subjective stationarity and to finally accept physical motion as normal internally defined as stationarity.

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