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Visual Suppression of Torsional Vestibular Nystagmus in Rhesus Monkeys

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Juvenile rhesus monkeys, placed on a motorized turntable, were rotated at constant velocity and then decelerated about an Earth-vertical axis. The animals were implanted with dual search coils to measure eye movements in three dimensions. By changing the monkey's body position (upright, ear-down, supine), postrotatory nystagmus was elicited in the horizontal, vertical, or torsional direction. Peak slow phase eye velocity and time constant of velocity decay were compared between decelerations in the dark and in the light. In all nystagmus directions, illumination reduced the time constant (T_c) to values around 5 sec. Peak velocity (V_p) was markedly attenuated in the horizontal and vertical directions (around 50%), but the effect of light on V_p in the torsional direction was small (<20%). These findings were independent of the velocity step size. Our hypothesis is that the two dynamic components of optokinetic nystagmus, as they interact with postrotatory nystagmus during visual suppression, differ in their dimensionality: the early component (fast component, direct pathway, pursuit system) is mainly activated in the horizontal and vertical directions, while the late component (slow component, indirect pathway, optokinetic system) effectively operates in all three dimensions.

Vestibulo–ocular reflex Optokinetic nystagmus Three-dimensional eye movements

INTRODUCTION

Vestibulo–ocular reflex (VOR) and optokinetic nystagmus (OKN) serve to stabilize the retinal image (for reviews see: Berthoz & Melvill Jones, 1985). When the head rotates and the visual surround is stationary, VOR and OKN cooperate in producing the necessary compensatory eye movements. Conflicts between OKN and VOR arise in two situations: (1) At the beginning of optokinetic stimulation, the visual surround starts to move while the head is kept still. (2) During visual suppression of vestibular nystagmus, head and visual surround start to move in the same direction. In the latter case, VOR leads to retinal slippage and hence activates OKN towards the opposite side (Waespe & Henn, 1978), i.e. vestibularly induced slow eye movements are rapidly suppressed. The same conflicting situation is produced by sudden illumination during postrotatory nystagmus (PRN): OKN-activation strongly speeds up the decay of vestibular nystagmus and drives the slow phase velocity of PRN quickly to zero (Waespe, Cohen & Raphan, 1985).

VOR and OKN show different but overlapping working ranges: VOR is tuned to high angular accelerations, while OKN works at low accelerations and constant velocity. OKN consists of two parts which differ in their dynamics. In primates, Cohen, Matsuo and Raphan (1977) called them the *direct* and *indirect pathways*, while Robinson (1981) used the terms *pursuit* and *optokinetic system*. The direct pathway or pursuit system shows fast dynamics: it leads to a rapid increase of slow phase eye velocity during sudden exposure to optokinetic stimulation and is responsible for the immediate velocity drop after lights are turned off. The indirect pathway or optokinetic system is slower and outlasts the stimulus, giving rise to optokinetic after-nystagmus (OKAN). Over a wide range, activity in neurons of the vestibular nuclei mirror activity in the optokinetic system (Waespe & Henn, 1977a, b).

Both components of OKN contribute to the visual suppression of PRN: the *early component* leads to a marked reduction of the peak slow phase velocity; the *late component* decreases the time constant of velocity decay. So far these phenomena have been investigated for PRN in the horizontal and vertical directions (e.g. Ohm, 1926; Collins, 1966; Waespe *et al.*, 1985), but only few investigations are available for the torsional direction (Melvill Jones, 1960, 1965). As the VOR works in three dimensions but torsional pursuit has not been demonstrated yet, the question arises whether the early OKN-component can be shown to interact with VOR elicited about the line of sight. Specific hypotheses can be put forward: (1) if the early OKN-component is non-operative in the torsional direction, the peak slow

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phase velocity of torsional PRN should be minimally affected by illumination; (2) if the late OKN-component is equally effective in all three dimensions, the fast velocity decay of PRN in the light should be observed in all directions.

To test these hypotheses, we compared PRN of monkeys in the dark and in the light. As shown below, we found marked differences in how PRN is attenuated by illumination, depending on whether nystagmus was in the horizontal, vertical, or torsional direction. We will discuss how these differences can be explained by the presumption that the early OKN-component operates mainly in two dimensions.

METHODS

Experiments were performed on three juvenile rhesus monkeys, which were chronically prepared with scull bolts to fixate the head during experiments. A dual search coil was implanted in one eye for three-dimensional recording of eye movements (Hess, 1990). Surgery was done under inhalative anesthesia with an O_2/N_2O-mixture, initiated by i.p. pentobarbital and supplemented by halothane as required.

The monkeys were seated in a primate chair with their heads fixed, whereby the horizontal stereotaxic plane* was tilted 15 deg in nose-down direction to position the lateral semicircular canals approximately horizontal. The primate chair was placed inside the inner frame of a vestibular turntable with three motor driven axes (manufactured by Acutronic, Jona, Switzerland). This apparatus was surrounded by an optokinetic sphere with a radius of 80 cm. The inner surface of the sphere was white and randomly covered by black solid circles with diameters ranging between 3 and 15 cm to produce an optimal visual stimulus for drift velocities between 50 and 200 deg/sec (Miles, Kawano & Optican, 1986). The circles overlaid about one-quarter of the surface. A light attached to the inner axis just above the monkey's head could be turned on and off during the experiment; the actual onset of illumination was measured by a photcell.

Eye movements were recorded with a "Type 3000 Eye Position Meter" (Skalar Instruments, Delft, The Netherlands): The animal's head was centered inside the driver coil frame (side length, 30 cm), which generated a horizontal and a vertical magnetic field in phase-quadrature (frequency, 20 kHz). Signals were detected by amplitude discrimination (Robinson, 1963). The dual search coil yielded two sensitivity vectors: one pointing approximately in the direction of the visual axis, and the other roughly perpendicularly to the first vector. Thus, by the two magnetic fields, two voltages were generated in each coil. This 4-channel analog output together with the chair velocity signal and the light status was digitized (Cambridge Electronics Device 1401) with a sample rate of 833.3 Hz and directly stored on the hard disk of a computer (IBM PS/2 Model 80). For each sample point, the corresponding rotation vector, describing three-dimensional eye position (Haustein, 1989), was computed off-line.

*Defined by the outer acoustic meatus and the lower orbital rim.

Calibration

The computation of rotation vectors from the 4 dual search coil voltages took into account calibration values obtained before coil implantation (*in vitro calibration*) and calibration values acquired by a fixation task (*in vivo calibration*). Details of this combined *in vivo/in vitro* calibration procedure will be published elsewhere (Hess, Van Opstal, Straumann & Hepp, 1992). In short, the *in vitro* calibration yielded the lengths of the two coil vectors as well as the angle between the vectors. Due to the stable geometry of the dual search coil, this angle did not change with implantation. During *in vivo* calibrations, monkeys had to fixate light dots along the mid-sagittal plane. According to Listing's law (Helmholtz, 1866), the corresponding secondary eye positions were assumed to have no torsional component. From the voltages measured at these eye positions we could calculate the orientation of both coil vectors and possible vertical and horizontal voltage offsets, considering also the length of the vectors and their fixed angle relative to each other, as determined in the *in vitro* calibration. It was essential for the quantitative analysis of our experiments that the torsional component of eye movements was as reliably calibrated as the horizontal and vertical components. Therefore *in vivo* calibrations were repeated on each experimental day.

Experimental protocol

Monkeys were accelerated in the dark to a specified velocity (values are given below). This velocity was kept until the first phase of vestibular nystagmus had ended. Then the animals were decelerated with 100 deg/sec^2. Two situations were studied: DARK, PRN in total darkness; LIGHT, PRN in the light, which was turned on at the onset of deceleration.

In all experiments the chair was rotated about an axis collinear with the gravity vector: for horizontal, vertical, or torsional nystagmus, the animal was placed in upright, ear-down side, or supine position, respectively. We obtained most data with decelerations from 200 deg/sec. Experiments were repeated with chair velocities of 100, 50, 25 and 12.5 deg/sec. To simplify the analysis, only rightward, upward, downward, and counterclockwise PRN were included in the analysis since horizontal and torsional nystagmus showed symmetrical properties. One animal (monkey II) was also exposed to decelerations in oblique directions, e.g. 45 deg backward pitch for combined horizontal-torsional PRN or 45 deg ear-down from supine for combined vertical-torsional PRN.

Data analysis

The calibrated data were first differentiated with a polynomial filter (Savitzky & Golay, 1964). Differentiation of a three-dimensional eye position signal yields a *coordinate eye velocity vector*. Due to the non-commutativity of rotations, the coordinate eye velocity vector is different from the *angular eye velocity vector*

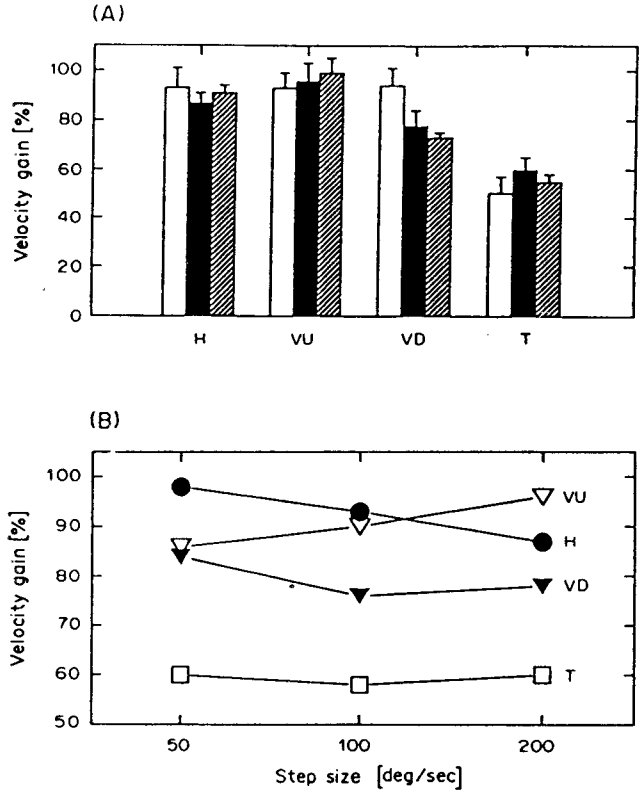

FIGURE 1. Peak slow phase eye velocity in the dark for different directions of PRN (H, horizontal; VU, vertical-up; VD, vertical-down; T, torsional). (A) Velocity gain (in %) after a chair velocity step of 200 deg/sec with 100 deg/sec^2 (monkey I, open bar; monkey II, solid bar; monkey III, hatched bar). Error bars indicate one standard deviation of 4–7 experimental runs. (B) Velocity gain as a function of the chair velocity step size in monkey II. Similar diagrams were obtained in the other two animals. Each symbol denotes the mean of 5–7 experimental runs. Standard deviations are omitted, they were all below 15% of the mean value.

unless the eye is in (or close to) the reference position. Since during nystagmus the beating field shifts mean eye position away from the reference position, it is important to use angular eye velocity (Tweed & Vilis, 1987).*

Angular velocity data were further processed to obtain the slow phase eye velocity envelope. Details of the procedure are described elsewhere (Straumann, 1991). In short: (1) each zero-crossing of the eye velocity from negative to positive values was marked. Marks divided the velocity trace into intervals of different length. Intervals above a minimal length were further processed. (2) In each interval, the velocity data points were sorted in an ascending order. The data point at the least slope (mode value) was taken as the representative slow velocity for the processed interval. (3) By a spline fit, the representative values were connected. The resulting envelope gave an accurate description of slow phase velocity change over time.

For the slow phase envelope of each experimental run, the peak velocity (V_p) and the time constant of velocity decay (T_c)† was computed and analyzed statistically. In calculating standard deviations we divided by N. In the following, PRN-direction denotes the direction of the slow phase velocity vector.

RESULTS

Before reporting the suppressive influence of light on vestibular nystagmus, we give the baseline data of PRN in the dark. During horizontal and vertical nystagmus after decelerations from 200 deg/sec, the three animals reached peak velocities around 80–90% of the chair velocity [Fig. 1(A)]. In two animals, values were considerably smaller in the vertical-down than in the vertical-up direction. For torsional PRN, peak velocity ranged around 50–60%. Similar gains for torsional VOR have been found in humans (Collewijn, Van der Steen, Ferman & Jansen, 1985; Ferman, Collewijn, Jansen & Van den Berg, 1987b; Leigh, Maas, Grossman & Robinson, 1989; Seidman & Leigh, 1989) and monkeys (Crawford & Vilis, 1991).

Monkeys were also subjected to decelerations from 100 and 50 deg/sec. Peak velocity gains did not change much for smaller velocity steps [Fig. 1(B)]. In contrast, the time constant showed considerable fluctuation between monkeys [Fig. 2(A)] and step sizes [Fig. 2(B)],

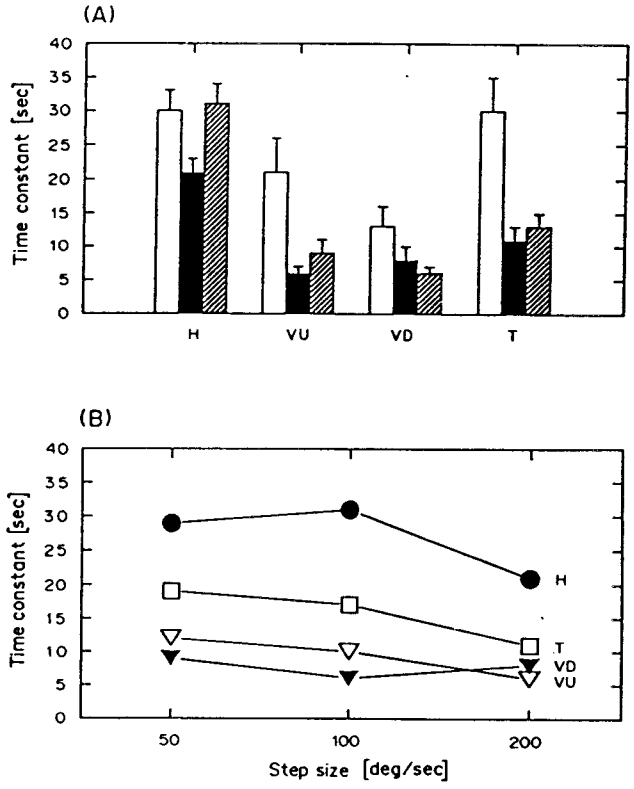

FIGURE 2. Time constant of slow phase eye velocity decay in the dark for different PRN directions (H, horizontal; VU, vertical-up; VD, vertical-down; T, torsional). Analysis covers the same experimental runs as in Fig. 1. (A) Time constant values (in sec) after a chair velocity step of 200 deg/sec with 100 deg/sec^2 (monkey I, open bar; monkey II, solid bar; monkey III, hatched bar). Error bars indicate one standard deviation of 4–7 experimental runs. (B) Time constant as a function of the chair velocity step size in monkey II. Similar diagrams were obtained in the other two animals. Each symbol denotes the mean of 5–7 experimental runs. Standard deviations are omitted, they were all below 20% of the mean value.

*For a rotation vector r, the angular velocity v (rad/sec) is: $v = 2(\dot{r} + r \wedge \dot{r})/(1 + |r|^2)$ (Hepp, 1990).

†"Time constant": the time elapsed between maximal slow phase velocity (100%) and attenuation to 37%.

even though individual standard deviations for a specific step size were small. The term "time constant" does not imply that the velocity decay was exponential; it rather denotes a practical way of indicating suppressive effects on nystagmus decay. Comparing the time constants in different directions, it was evident that the velocity storage mechanism in all three monkeys was more effective for torsional than for vertical, and most effective for horizontal vestibular nystagmus.

PRN-experiments in the light were first conducted with decelerations from 200 deg/sec with 100 deg/sec^2. Already qualitatively, it was obvious that the peak slow phase velocity of torsional PRN was little suppressed by light (Fig. 3). Contrary, nystagmus duration was reduced in all directions, including torsion.

Evaluating the visual suppression of PRN, each experimental run in the light was compared with its preceding run conducted in the dark with otherwise identical parameters (Fig. 4). Suppression of peak slow phase velocity was described by:

$$\text{Gain suppression } (\%) = \frac{V_{p(\text{dark})} - V_{p(\text{light})}}{V_{p(\text{dark})}} \times 100.$$

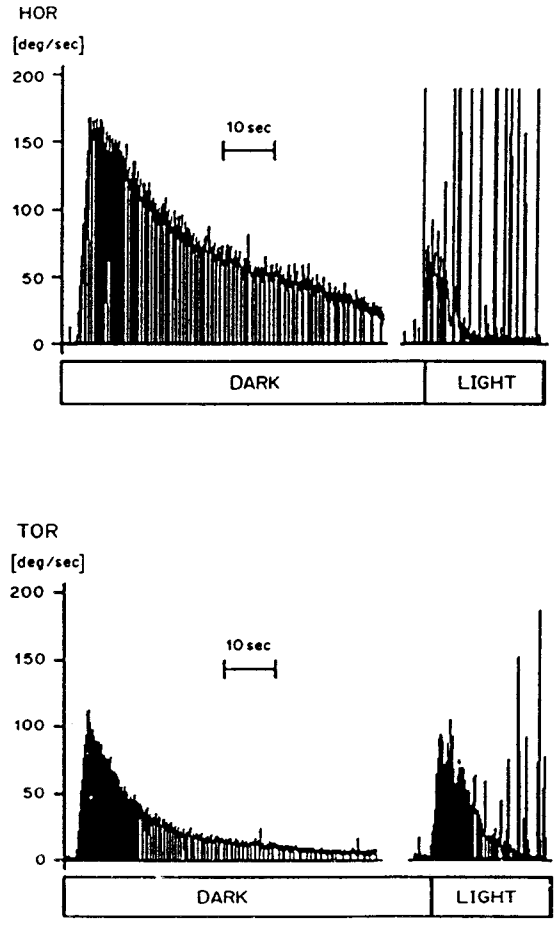

FIGURE 3. Decelerations from 200 to 0 deg/sec with 100 deg/sec^2 in the dark and in the light. Example of PRN in monkey III. First row: horizontal PRN, monkey in upright position. Second row: torsional PRN, monkey in supine position. Peak slow phase eye velocity of PRN is more suppressed by light in the horizontal than in the torsional direction. Nystagmus duration in the dark is longer for horizontal step responses, while durations of horizontal and torsional PRN in the light show similar values. For statistics see Figs 5 and 6.

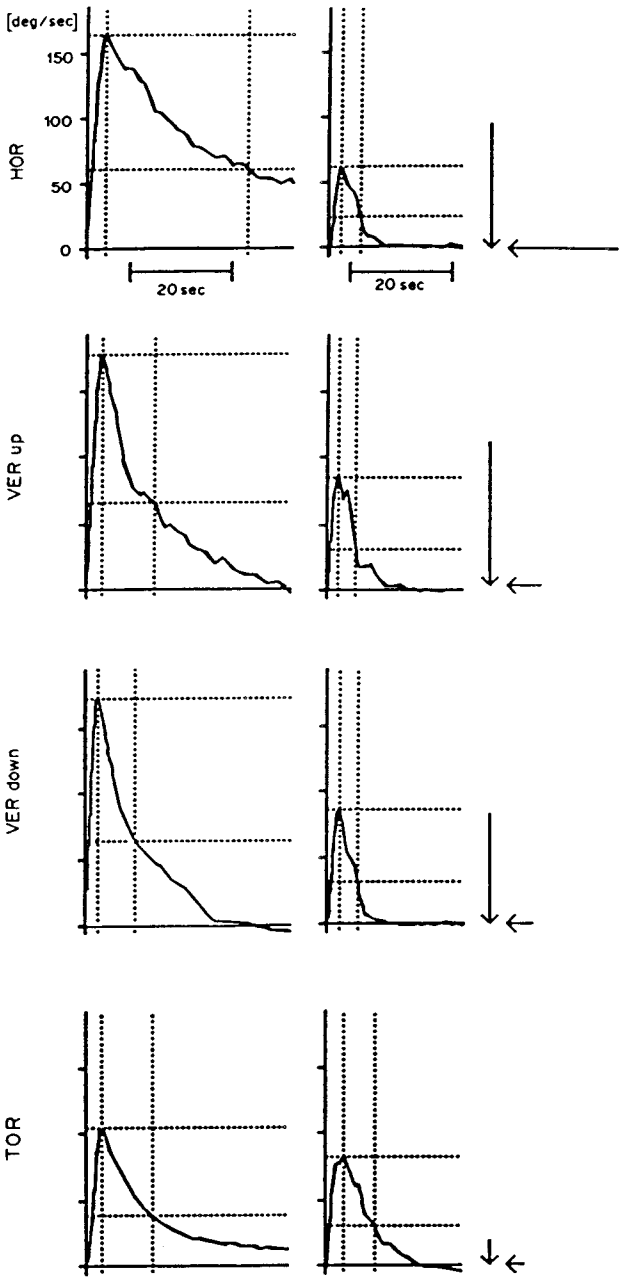

FIGURE 4. Quantitative analysis of PRN in the dark and in the light (example: monkey III). The procedure of the analysis is outlined in the text. For each experimental run, the computer automatically determined the peak slow phase eye velocity and the time constant of velocity decay. Arrows on the right indicate the suppressive effects of light on peak velocity (vertical arrows) and the time constant (horizontal arrows). HOR, horizontal; VER up, vertical-up; VER down, vertical-down; TOR, torsional. For statistics see Figs 5 and 6.

In all three monkeys the mean suppressive effect of light on torsional peak velocity was below 20% [Fig. 5(A)]. For horizontal and vertical PRN, the values were around 50%. There was a clear tendency that peak velocity was less suppressed in the vertical-up than in the vertical-down direction.

The impact of light on the velocity decay was substantial in all nystagmus directions [Fig. 6(A)]. Remarkably, time constants (T_c) in the light were all around 5 sec, independent of the corresponding values in the dark. As a result, the light-induced decrease of time constant was

largest for horizontal PRN since nystagmus duration in the dark was longest in this direction (see Fig. 2).

PRN-experiments were repeated for smaller velocity steps. For 100 and 50 deg/sec results were similar as for 200 deg/sec velocity steps. In the light, peak velocity for horizontal and vertical nystagmus was reduced by about 50%, while the values for torsional nystagmus ranged below 20% [Fig. 5(B)]. Time constants decreased to around 5 sec in each direction, including torsion [Fig. 6(B)]. Hence, in all the experiments, we found torsional PRN to be unique in the sense that light had little effect on its peak slow phase velocity. In terms of the light-induced decrease of time constant, torsional PRN showed no exceptional behavior. Qualitatively, the same phenomenon was observed for decelerations from 25 and 12.5 deg/sec; however, for smaller maximal slow phase eye velocities visual VOR-attenuation varied much more with the animal's alertness.

The above results led to the prediction that visual suppression of oblique PRN in torsional-horizontal or torsional-vertical directions would tilt the eye velocity vector towards the torsional eye rotation axis. In other words: if the torsional gain component of oblique VOR is less attenuated by light than the horizontal or vertical

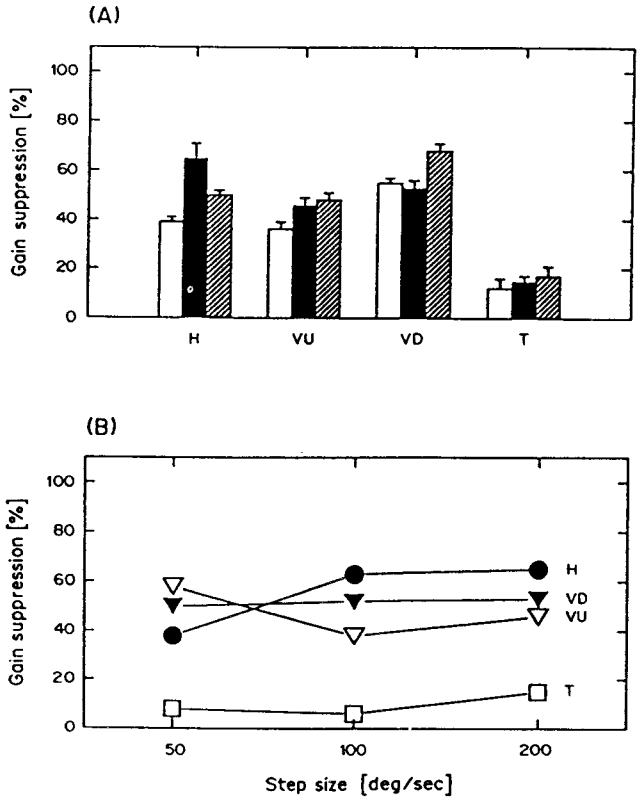

FIGURE 5. Gain suppression by light for different PRN directions (H, horizontal; VU, vertical-up; VD, vertical-down; T, torsional). The formula for calculating the suppression value (in %) is given in the text. (A) Gain suppression after a chair velocity step of 200 deg/sec with 100 deg/sec^2 (monkey I, open bar; monkey II, solid bar; monkey III, hatched bar). Error bars indicate one standard deviation of 6–7 experimental runs. (B) Gain suppression as a function of the chair velocity step size in monkey II. Similar diagrams were obtained in the other two animals. Each symbol denotes the mean of 4–7 experimental runs. Standard deviations are omitted, they were all below 20% of the mean value.

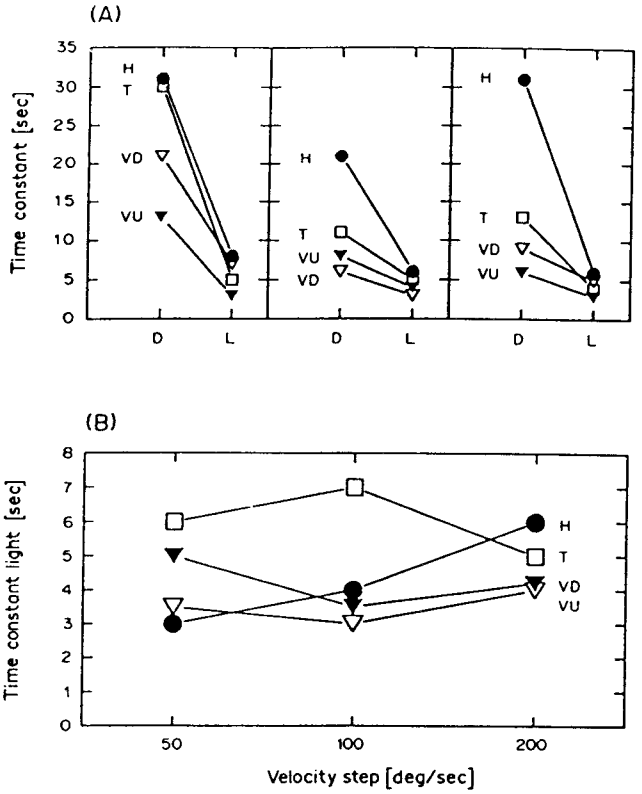

FIGURE 6. Reduction of velocity decay time constant in the light for different PRN directions (H, horizontal; VU, vertical-up; VD, vertical-down; T, torsional). Analysis covers the same experimental runs as in Fig. 5. (A) Time constant in the light (L) and in the dark (D) after a chair velocity step of 200 deg/sec with 100 deg/sec^2 (monkey I, 1st column; monkey II, 2nd column; monkey III, 3rd column). Each symbol denotes the mean of 6–7 experimental runs. Standard deviations are omitted; they were always below 20% of the mean value in the dark and below 2.5 sec in the light. (B) Time constant in the light as a function of the chair velocity step size in monkey II. Similar diagrams were obtained in the other two animals. Each symbol denotes the mean of 4–7 experimental runs. Standard deviations are omitted; they were all below 3.0 sec.

gain component, the velocity axis of eye rotation in the light should have a bias in the torsional direction. In the animal tested (monkey II) we found clear evidence for this tilt: in all oblique directions that included a torsional velocity component, eye velocity vectors in the light approached the torsional eye rotation axis (Fig. 7). A similar shift of rotation axis was not observed for oblique horizontal–vertical directions.

DISCUSSION

The objective of this study was to test whether the early component of OKN would interact with torsional vestibular nystagmus. Our prediction was based on a specific hypothesis concerning the directional degrees for freedom of OKN. The early OKN-component (direct pathway, pursuit system) was presumed to be two-dimensional, the late OKN-component (indirect pathway, optokinetic system) three-dimensional.

The description of visual suppression of PRN included two parameters: maximum slow phase velocity (V_p) and velocity decay time constant (T_c). It has been shown for

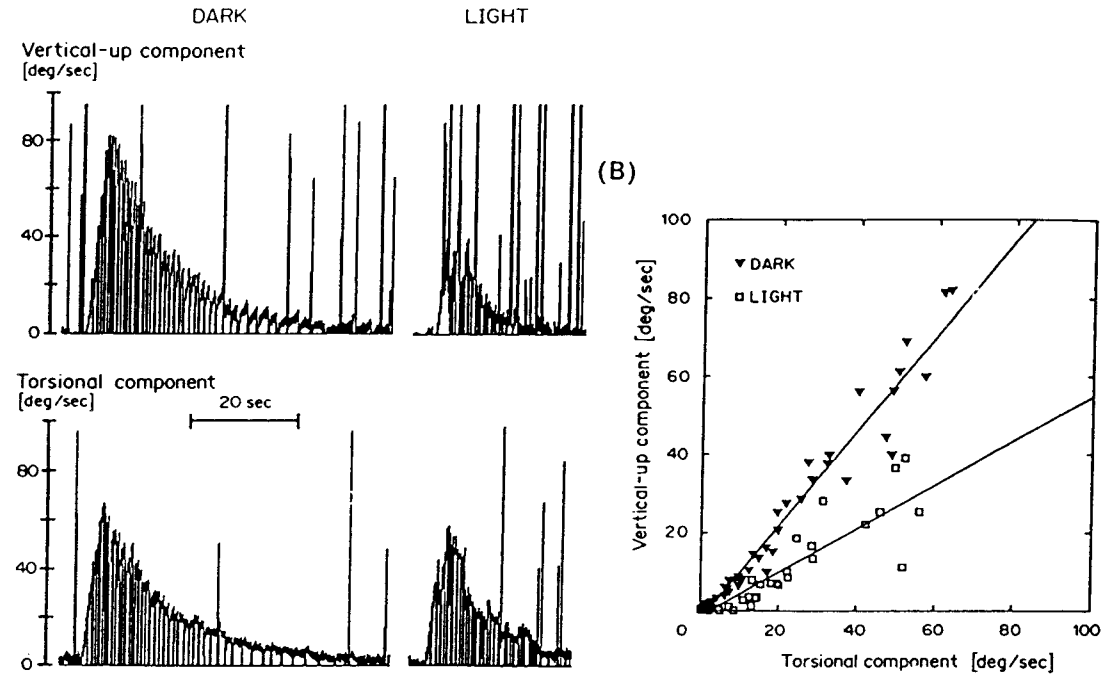

FIGURE 7. Deceleration of monkey II in the dark and in the light about an oblique torsional-vertical axis. The monkey was tilted from supine to 45 deg right-ear-down and then rotated about an earth-vertical axis with 100 deg/sec. As the chair stopped with 100 deg/sec^2, PRN slow phase was evoked in a combined vertical-up/torsional direction. Because of a smaller gain in the torsional direction, nystagmus direction in the dark was not collinear with the rotation axis but tilted towards the vertical axis. In the light, peak slow phase velocity was more suppressed in the vertical-up than in the torsional direction. (A) Vertical-up and torsional components of eye velocity in the dark and in the light. (B) Analysis of nystagmus direction. Each symbol denotes a slow phase angular velocity vector with a vertical-up and a torsional component. As expected from the velocity traces, a linear regression through all slow phase velocity data points in the dark (solid triangles) yields an eye rotation axis which is slightly tilted towards the vertical-up component. In the light (open squares), the rotation axis shifts towards the torsional component.

the visual suppression of horizontal PRN that V_p is predominantly reduced by the early, T_c by the late OKN-component (Waespe *et al.*, 1985). Expanding these measurements to PRN in three dimensions, we found similar suppressive light effects on PRN for all velocity step sizes. In the tested directions (horizontal, vertical, torsional) T_c was reduced to values around 5 sec, representing roughly the time constant of the peripheral vestibular organ. However, V_p was much more attenuated in the horizontal and vertical directions than in the torsional direction. The suppression of V_p in torsional PRN consistently was $<20\%$, which suggests that in the context of visual VOR-suppression, the early OKN-component operates mainly in the horizontal and vertical directions.

Our data agree with studies in human subjects, showing that torsional VOR can be attenuated in the light (Collewjin *et al.*, 1985; Leigh *et al.*, 1989). These investigators used oscillatory stimuli and did not separate early and late OKN-components. By applying vestibular velocity steps in the light, we found that OKN, as its interacts with PRN, behaves differently in the early and late phase of visual suppression. To further support this finding, we tried to quantify the initial drop of eye velocity during optokinetic after-nystagmus (OKAN). However, this approach had severe drawbacks. Contrary to horizontal OKN, slow phase eye velocity elicited by torsional optokinetic stimulation shows large fluctuation over time, which does not yield consistent eye velocities immediately before the light is turned off. Besides, the fluctuation leads to uncertainties concerning the loading of the velocity storage during OKN (Fig. 8). Another way to study a possible early torsional OKN-component is to quantify the initial rise of slow eye velocity at the beginning of optokinetic stimulation. In our entire data base (3 monkeys) with different velocities of the optokinetic sphere, torsional eye movements never exceeded 15 deg/sec during the first 500 msec of stimulation. If there is an early torsional OKN-component, it is certainly rudimentary as compared to horizontal and vertical OKN-directions.

Based on our findings on visual suppression of vestibular nystagmus in three dimensions, we speculate that brain structures involved in the late OKN-component show a three-dimensional representation of eye movements, while structures involved in the early OKN-component are two-dimensional. Further investigations should address this hypothesis on the level of single-cell recordings. A possible corollary of the absence of an early torsional OKN-component is that eye (Westheimer, 1957; Ferman, Collewijn & Van den Berg, 1987a; Tweed & Vilis, 1990), head (Tweed & Vilis, 1988), and gaze (Straumann, Haslwanter, Hepp-Reymond & Hepp, 1991) movements to visual targets obey Listing's law (Helmholtz, 1866; Hepp, 1990); i.e. in natural

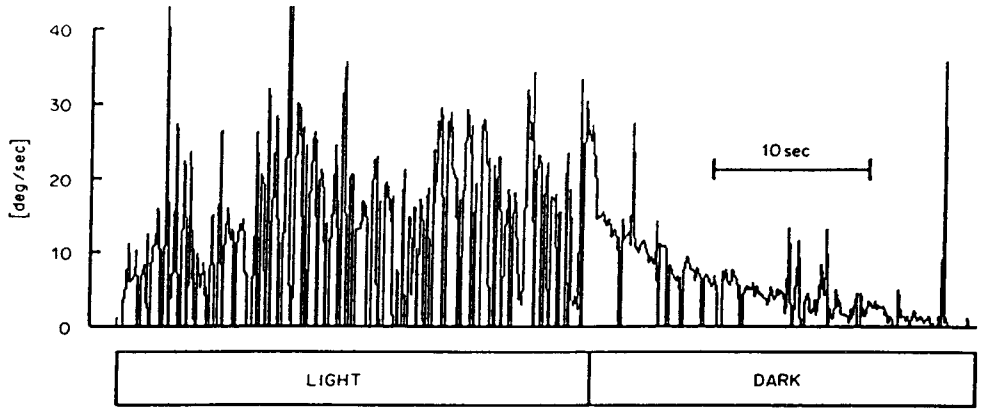

FIGURE 8. Torsional OKN and after-nystagmus (OKAN). The animal (monkey II), placed in a supine position, was rotated in the dark about an earth vertical axis with 120 deg/sec until vestibular nystagmus had stopped. Then the stationary optokinetic sphere was illuminated for 30 sec, after which the light was turned off again. In this figure, torsional eye velocity below zero is clipped since slow phase velocity was always positive in the experimental run shown. Eye movement recordings were stopped when secondary OKAN started. The given example of torsional OKN is typical in three ways: (1) There is considerable fluctuation of torsional slow phase velocity. Therefore it is not clear, whether the fast velocity drop after lights are turned off is light-induced or due to physiological fluctuation. (2) Torsional peak velocities hardly reach values above 30 deg/sec, even with a strong optokinetic stimulation of 120 deg/sec. This is a further indication that the direct OKN-pathway is non-effective in the torsional direction. (3) The initial rise of slow phase eye velocity can be interpreted as an early torsional OKN-component. However, in our data base with different velocities of the optokinetic drum, these movements never exceeded 15 deg/sec during the first 500 msec of stimulation.

conditions, these three motor systems follow trajectories with no torsional component. If the two-dimensional gaze performance matches with the two-dimensional retinal image, there is no need for a fast correcting visuo-motor mechanism in the torsional direction.

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