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VESTIBULAR NEURONES IN THE PARIETO-INSULAR CORTEX OF MONKEYS (*MACACA FASCICULARIS*): VISUAL AND NECK RECEPTOR RESPONSES

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SUMMARY

1. One hundred and fifty-two vestibularly activated neurones were recorded in the parieto-insular vestibular cortex (PIVC) of four awake Java monkeys (*Macaca fascicularis*): sixty-two were tested systematically with visual stimulation and seventy-nine were tested with various somatosensory stimuli. With very few exceptions all vestibular neurones tested responded to visual and somatosensory stimulation, therefore being classified as polymodal vestibular units.

2. A most effective stimulus for all fifty-eight visually activated PIVC units was movement of a large structured visual pattern in an optimal direction. From forty-four units responsive to a horizontally moving optokinetic striped drum, twenty-nine were activated with optokinetic movement in the opposite direction to the activating vestibular stimulus ('synergistic' response), thirteen were activated optokinetically and vestibularly in the same direction ('antagonistic' responses) and two were biphasic. The gain of the optokinetic response to sinusoidal stimulation (average 0·28 (impulses s^{-1}) (deg s^{-1})$^{-1}$ at 0·2 Hz, 56 deg amplitude) was in a range similar to that of the vestibular gain at low frequencies. At 1 Hz some units only showed weak optokinetic responses or none at all, but the vestibular response was still strong.

3. With different 'conflicting' or 'enhancing' combinations of optokinetic and vestibular stimulation no generalized type of interaction was observed, but the responses varied from nearly 'algebraic' summation to no discernible changes in the vestibular responses by additional optokinetic stimuli. With all visual–vestibular stimulus combinations the responses to the vestibular stimulus remained dominant.

4. The optokinetic preferred direction was not related to gravitational co-ordinates since the optokinetic responses were related to the head co-ordinates and remained constant with respect to the head co-ordinates at different angles of steady tilt.

5. Almost all PIVC units were activated by somatosensory stimulation, whereby mainly pressure and/or movement of neck and shoulders (bilateral) and movement of the arm joints elicited vigorous responses. Fewer neurones were activated by

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lightly touching shoulders/arms or neck, by vibration and/or pressure to the vertebrae, pelvis and legs.

6. A most effective somatosensory stimulus was sinewave rotation of the body with head stationary. The gain of this directionally selective neck receptor response was in the range of vestibular stimulation. Interaction of vestibular and neck receptor stimulation was either of a cancellation or facilitation type. Active head rotation, occasionally observed, did not produce different discharges from those with passive head rotation.

7. The PIVC neurone discharge pattern was not correlated to saccades. With different optokinetic velocities, however, different activation levels were observed. In a few examples reduction in discharge rate occurred parallel to a decrease in optokinetic gain, presumably due to fluctuating attention.

INTRODUCTION

As everyday experience indicates, we perceive the position and movement of our head and body with respect to the co-ordinates of extrapersonal space (field of gravity) fairly correctly over a wide range of conditions. This achievement is also maintained during locomotor head and body movements and visually guided pursuit of a moving object by means of eye, head and body movements, provided the movement acceleration or angular rotation remains within certain limits. The afferent neuronal signals from the labyrinths, the retinae, the neck receptors and other proprioceptive receptors of the body, together with internal feedback signals of the motor status ('efference copy' signals), are evidently integrated by a complex neuronal machinery to achieve correct information about self-movement relative to the stationary world. Concerning the role of the afferent vestibular system within this multimodal signal processing, efferent control signals modify the sensitivity of the most peripheral vestibular neurones (Klinke & Galley, 1974), while in the brain stem vestibular nuclei, visual and proprioceptive information is incorporated into the afferent signal flow (Ohm, 1943; Fredrickson, Schwarz & Kornhuber, 1966; Henn, Young & Finley, 1974).

The ascending axons, originating in the vestibular brain stem nuclei, transfer signals in part to neurones located in the somatosensory *thalamic* relay nuclei (ventro-postero-lateral complex, VPL, and ventro-postero-inferior complex, VPI). Whether additional integration of visual and/or proprioceptive afferent signals with vestibular signals exists at the thalamic level has not yet been proven. Apart from Mergner's (1979) description of vestibular–neck receptor signal interaction in neurones of the cat ASSS region (anterior supra-Sylvian sulcus), no detailed studies have been published on the responses of cortical vestibular neurones to natural stimulation of the visual and somatosensory modalities. In man some recent psychophysical studies of the effect of sinusoidal body rotation on circular movement (apparent self-rotation) while the head was fixed in space indicated that neck receptor signals induce movement and even modify the optokinetic responses of neurones coding 'vestibular' sensation (de Jong, Bles & Bovenkerk, 1981; Mergner, Nardi, Becker & Deecke, 1983). A strong optokinetic input has already been described by Büttner & Buettner (1978) in vestibular units of the cortical area 2v.

Linear or circular movement has been studied for a long time and is the most

prominent phenomenon of visually induced vestibular sensations pointing to a strong impact of optokinetic signals on vestibular neurones (Mach, 1875; Dichgans & Brandt, 1978). Comparing vection induced by an optokinetic drum (moving stripe pattern) and by simulated 'three-dimensional' visual pattern flow as applied in flight simulators, the latter type of visual stimuli appear to be much more effective and vection latencies are reduced to about 250 ms. This observation indicates that visual parallax movement is an important factor in visual–vestibular integration. For technical reasons most of the studies on movement were restricted so far to experiments with rotating cylinders or similar stimuli (cf. Bötzel, Dalbasti & Grüsser, 1981). In the present report we also applied these 'traditional' optokinetic stimuli only.

In the following the convergence of multimodal signals in single neurones of the monkey's parieto-insular vestibular cortex (PIVC) will be described. This area, among other brain regions, probably serves the function of recognizing and controlling head and body position in space.

METHODS

The methods applied in the present study have been extensively described in a preceding report (Grüsser, Pause & Schreiter, 1990). The data in the present report were obtained from the same four Java monkeys (*Macaca fascicularis*).

RESULTS

Visual responses to PIVC neurones

Activation by small-field visual stimulation

Out of one hundred and fifty-two vestibular PIVC neurones recorded, sixty-two were systematically tested with visual stimuli; fifty-eight neurones exhibited a change in their impulse rate when adequate visual stimuli were applied. No responses were obtained by changing general room illumination, 'on' or 'off' of small stationary spots of light or illumination of stationary desirable objects. The most effective visual stimulus in activating PIVC neurones was a well-structured large pattern (30 deg or more of the visual field) moving in an optimal direction. Some of the PIVC neurones, however, were also activated when small light stimuli were moved in certain 'preferred' directions. We could not measure the spatial extent of the respective visual receptive fields precisely, since the animals were not trained to maintain a steady fixation. We therefore had to rely on activity changes noted when black discs 5–10 cm in diameter were moved at a velocity of about 10–20 cm s^{-1} over a white homogenous background about 40–50 cm away (monocular or binocular stimulation). With this rather crude method we found large (at least 50 deg) visual receptive fields in all neurones, covering parts of both visual hemifields in each eye. In less than 20% of the neurones visual receptive fields were restricted to the contralateral visual half-field of both eyes. Binocular facilitation, as a rulë, was absent.

In a region adjoining PIVC at its posterior border, action potentials of 'pure' visual neurones were frequently recorded. These neurones did not respond to vestibular or somatosensory stimuli. They also exhibited a directional selectivity to

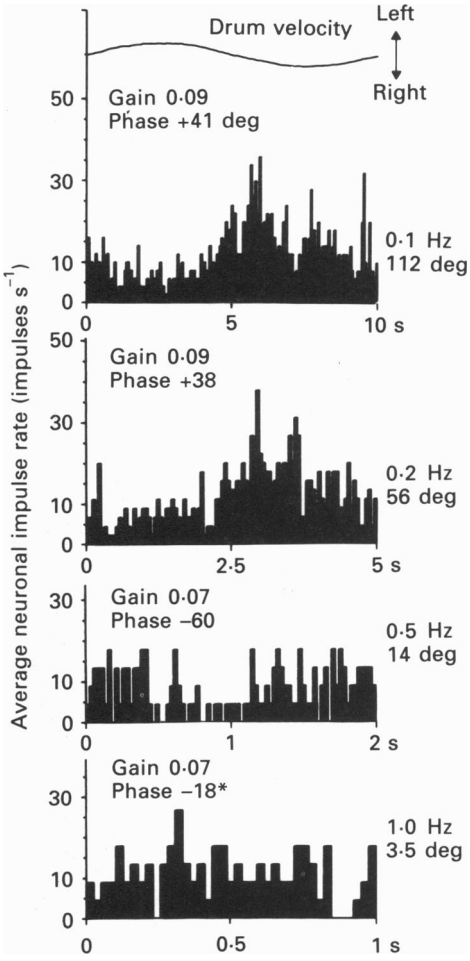

Fig. 1. PIVC neurone responding to sinewave horizontal rotation of vertical stripe cylinder (1·15 deg period of black–white stripes). The vestibular responses of this neurone were classified as type I yaw, type II roll and type II pitch. The neurone could also be activated by pressure on both sides of the neck and by sinusoidal rotation of the trunk (head, fixed, whereby trunk movement towards the left was the activating stimulus). The neurone responded to optokinetic stimulation at 0·1 and 0·2 Hz with a gain clearly above our threshold criterion, but with insufficient gain to stimulation with 0·5 and 1·0 Hz. (The calculated phase lag with 0·5 and 1·0 Hz of course is not relevant in a condition where a sufficient correlation of the neuronal activation with the stimulus is not present.)

movement of visual targets. Only weak responses, if at all, were obtained when general room illumination was turned on or off. Most of these visual neurones also responded vigorously and were directionally selective when an optokinetic drum was rotated around the animal, but neither tilting nor rotation of the animal in the dark nor neck receptor stimulation led to a change in neuronal activity.

Responses to optokinetic stimulation

From the fifty-eight visually responsive PIVC neurones investigated, fourteen required a broad-stripe pattern moving perpendicularly to the stripe orientation

across the visual field in vertical or oblique directions to evoke a significant modulation of spontaneous impulse activity. Since this stimulus (disc of 90 cm diameter, covered by black and white stripes of 3·5 cm period) had to be moved by hand (at a distance of about 40–50 cm from the monkey), no systematic quantitative

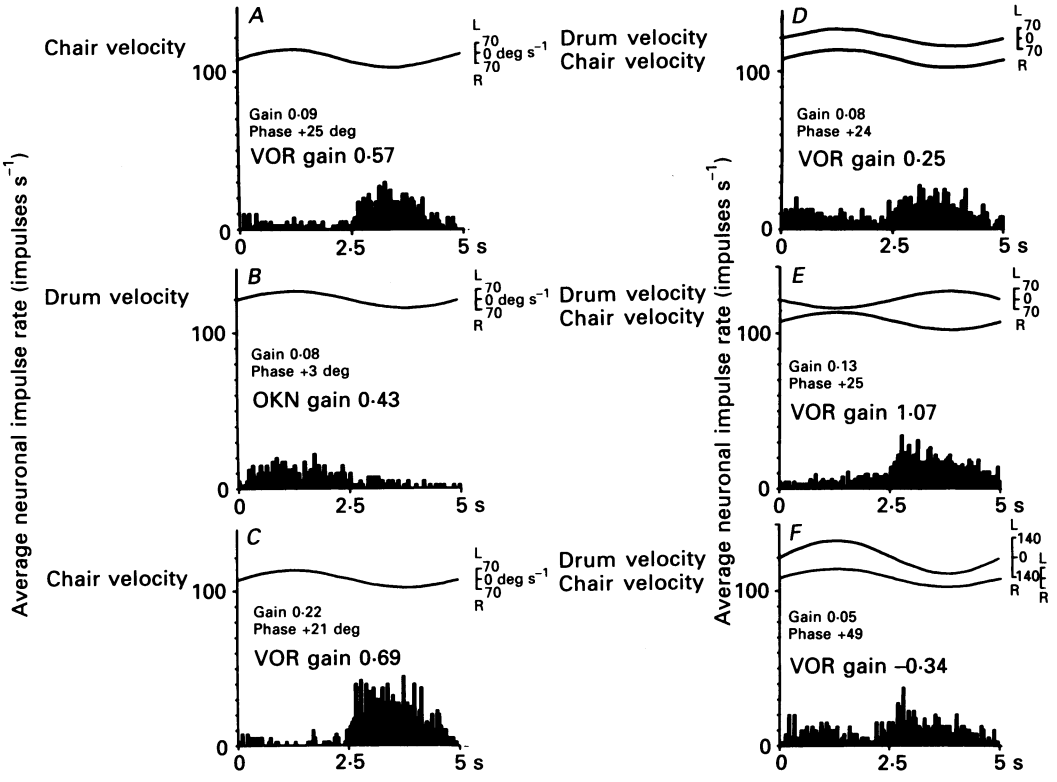

Fig. 2. PIVC neurone from monkey OL responding to visual and vestibular stimulation according to the synergistic mode. In addition to the response to horizontal rotation, this unit was activated by roll movement according to the response type II as well as by pressure on both sides of the neck. This neurone was activated by horizontal chair rotation in darkness towards the right (*A*) and optokinetic stripe pattern movement towards the left (*B*). Correspondingly, the neuronal response was facilitated when the chair was turned inside the stationary illuminated striped drum (*C*) and a biphasic activation was visible when drum and chair rotated in phase at the same amplitude (*D*) or different amplitudes (*F*). The neuronal response was not enhanced, however, when stripe cylinder and chair rotated 180 deg out of phase (*E*). Vestibulo-ocular reflex (VOR) gain is given as the average value of the ten periods.

measurements could be performed in these fourteen neurones. It became evident, however, that all of them were directionally selective. Machine-controlled optokinetic stimulation was applied in the remaining forty-four neurones and was restricted to horizontal rotation of a vertically striped cylinder (black and white stripes, period 1·15 deg; Fig. 1 in Grüsser, Pause & Schreiter, 1990) around the animal. Constant angular velocity stimulation of sinusoidal horizontal movement at different frequencies was applied (Fig. 1). Optokinetic directional selectivity was found in all

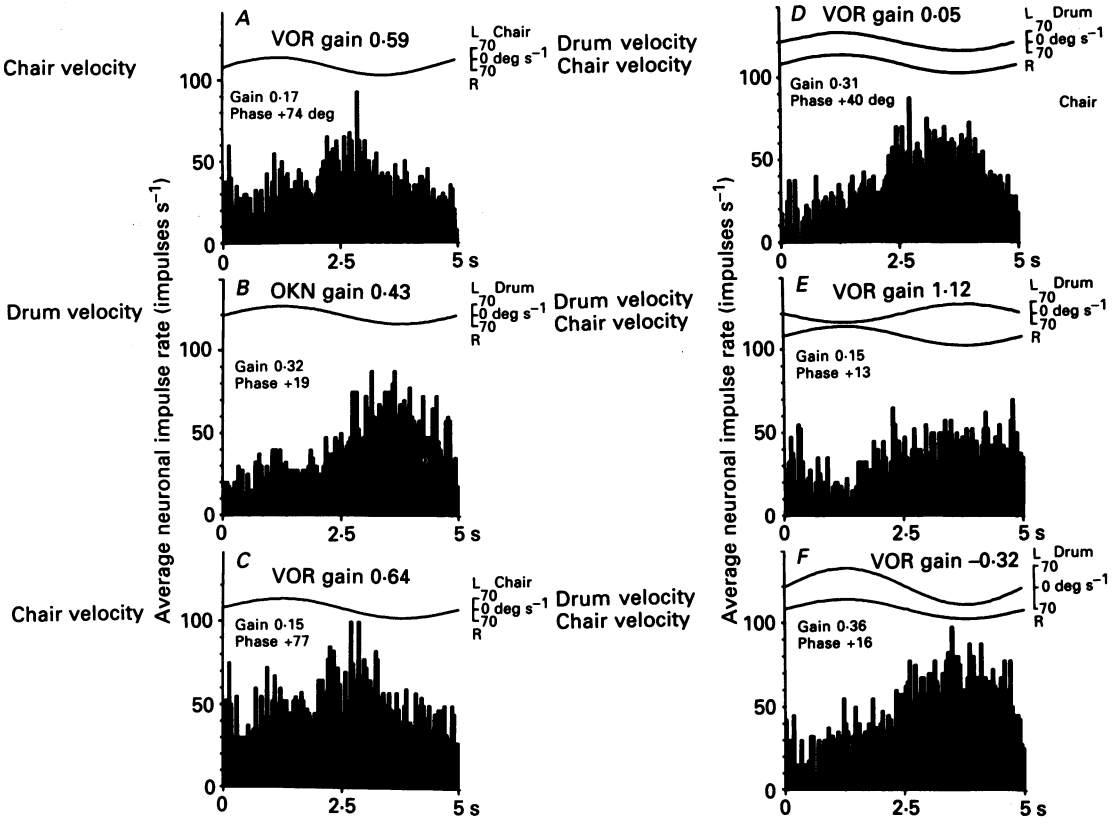

Fig. 3. Example of PIVC neurone from monkey RV exhibiting an antagonistic response to visual and vestibular stimulation. The neurone also responded to vestibular stimulation in roll direction (type I) and to trunk rotation, whereby the interaction between neck receptor input and the vestibular input was also antagonistic. Horizontal eye movements during the conflict experiments shown in this figure depended predominantly on the visual stimulus, but the vestibulo-ocular reflex was not completely suppressed. The neurone was activated by horizontal sinusoidal rotation in the dark when the chair moved towards the right (*A*); the same was true with rotation of the optokinetic drum around the stationary animal in the light (*B*). When the chair was rotated in the light, the visual response was less than the vestibular (*C*). When chair and drum were rotated in phase (*D*), the response resembled that aroused by drum rotation alone, while when drum and chair were 180 deg out of phase (*E*), the modulation of the neurone was considerably less than with optokinetic and vestibular stimulation alone. When both stimuli were in phase but the amplitude of the optokinetic stimulation was twice that of the vestibular sinusoidal rotation (*F*), the response was again enhanced. VOR gain was averaged from responses to ten stimulation periods.

neurones and was classified conversely to vestibular directional selectivity: activation by rotation of the striped drum towards the side contralateral to the recording site was called type Ih, towards the ipsilateral side type IIh. Two combinations of vestibular and optokinetic directional sensitivity were found: the larger group of PIVC neurones (twenty-nine neurones, 66%) was activated when the drum rotated in the opposite direction to the preferred direction of chair rotation in

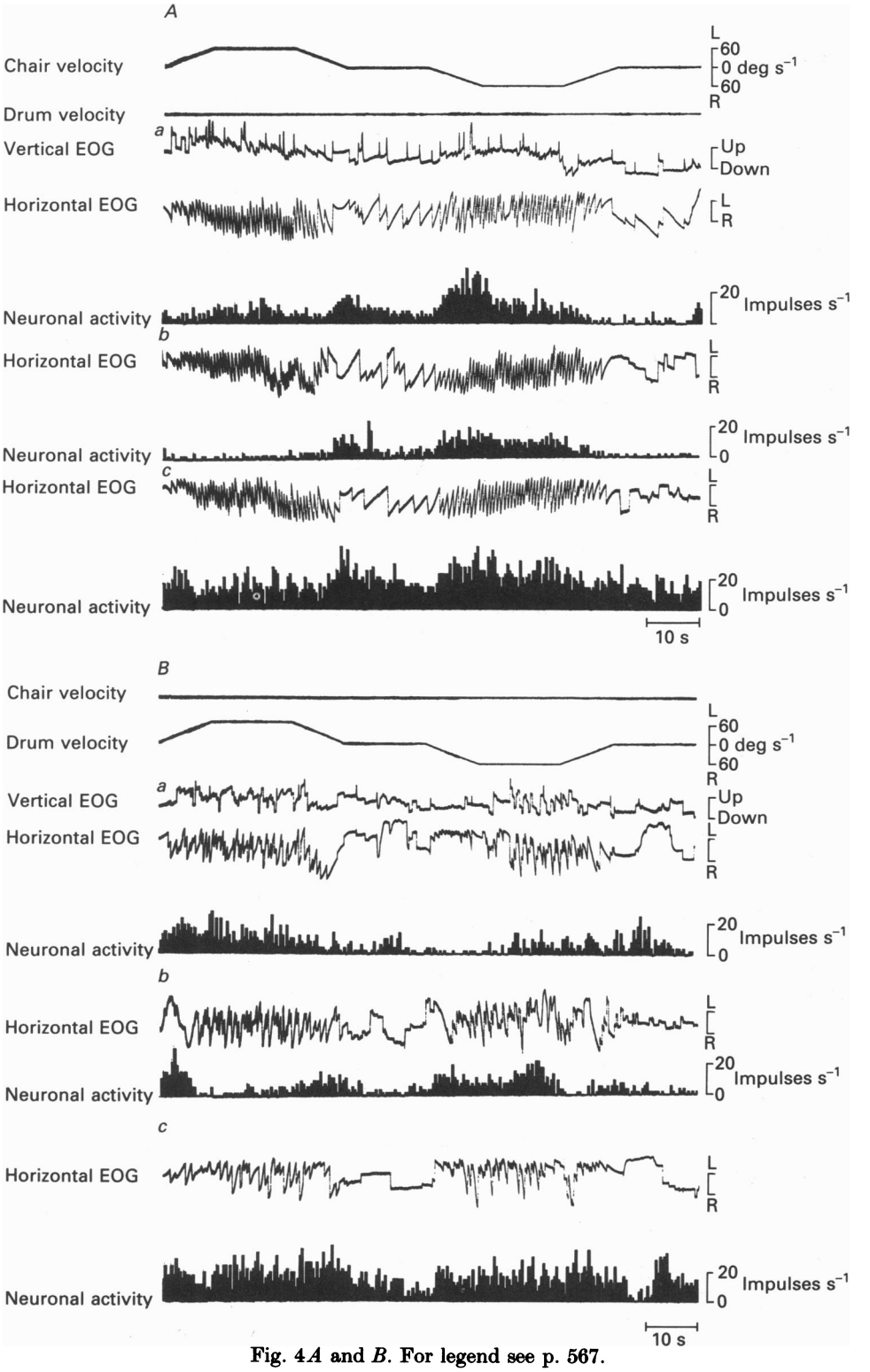

Fig. 4*A* and *B*. For legend see p. 567.

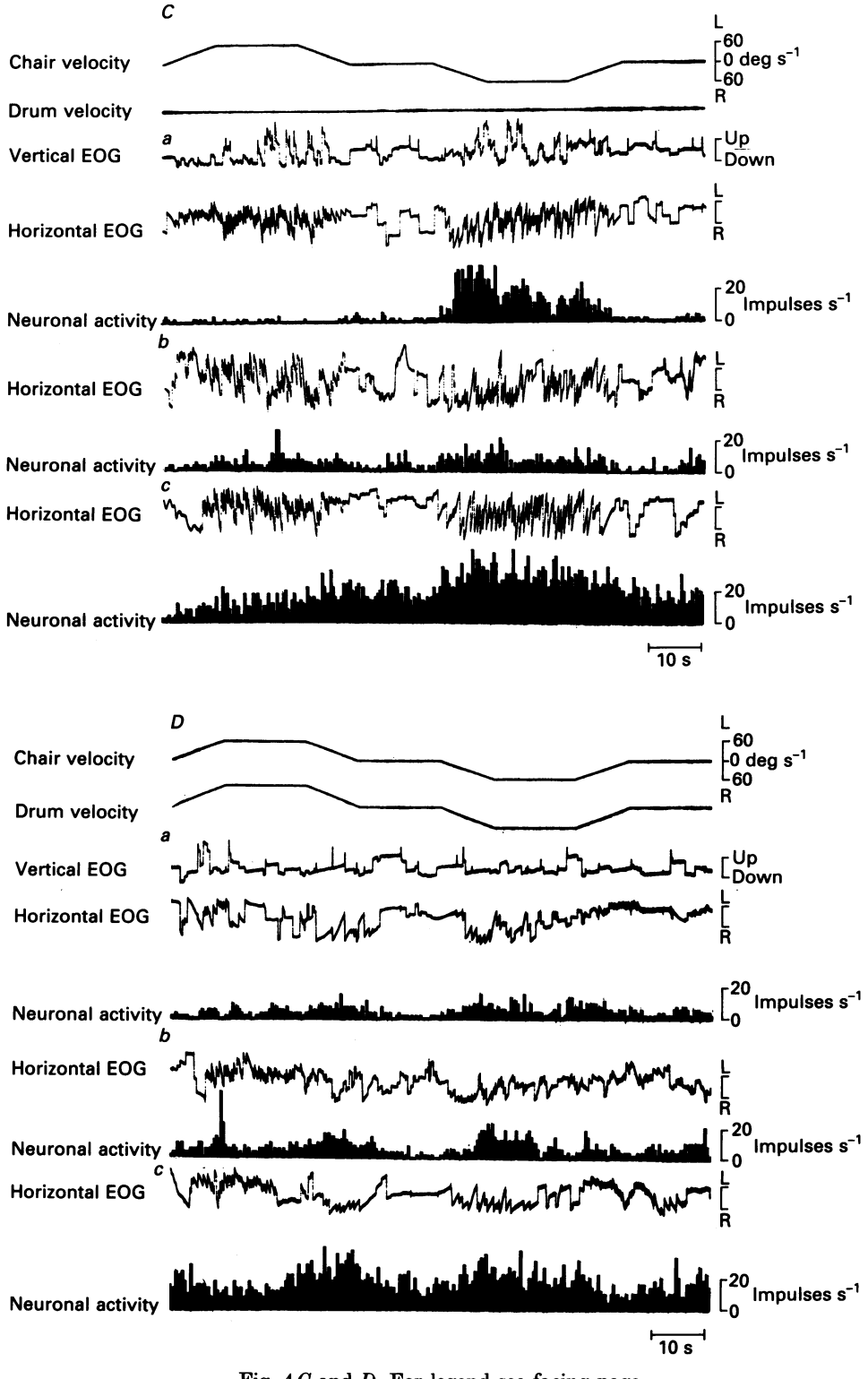

Fig. 4*C* and *D*. For legend see facing page.

the dark. From these twenty-nine units, eighteen were type I y as classified by chair rotation and type I h as classified by drum rotation. The remaining eleven neurones showed a type II y and type II h response. We called this type of visual–vestibular neurone 'synergistic', because during horizontal rotation of the head to one side in the light, the visual surroundings shift relative to the head rotation in the opposite direction. Examples of the responses of a 'synergistic' neurone to visual and vestibular stimulation in different combinations are shown in Fig. 2.

The smaller group of neurones tested (thirteen neurones, 30 %) was activated when the drum was moved in the direction also preferred for vestibular responses. For head movements in the light this response pattern obviously induced a 'conflict' of visual and vestibular signals. Therefore this type of visual–vestibular interaction was called 'antagonistic'; examples of neuronal responses are shown in Fig. 3. Of the thirteen neurones, four units reacted as type I y and type II h, nine units at type II y and type I h. Finally, two neurones (4 %) were found to be activated during drum rotation in both directions as well as during horizontal chair rotation in both directions (type III y and type III h).

To compute the *gain* and *phase* of responses during optokinetic sinewave stimulation we determined the sinewave optimally fitting the peristimulus time histograms (PSTHs). Then the amplitude of this sinewave was related to the maximum stimulus velocity of the sinusoidal drum rotation. Hence gain dimensions are given in (impulses s^{-1}) (deg s^{-1})$^{-1}$. With optokinetic stimulation in the dark at 0·2 Hz and 56 or 15 deg amplitude the mean gain found in sixteen units was $0{\cdot}28 \pm 0{\cdot}33$ (impulses s^{-1}) (deg s^{-1})$^{-1}$. The average phase angle in relation to the angular velocity at this frequency was +28 deg. Increase in optokinetic stimulus frequencies from 0·2 to 1 Hz led in five PIVC neurones to an increase in the response gain similar to that observed with vestibular stimulation. Three PIVC neurones, however, which were activated by 0·2 Hz drum rotation, did not respond to the 1 Hz optokinetic stimulus, as did the neurone shown in Fig. 1, while with horizontal chair rotation in the dark at 1 Hz the units responded vigorously. In these three units there was a remarkable phase shift with different frequencies, while in the other units the phase in relation to velocity remained as stable as with vestibular stimulation.

Fig. 4. Responses of three different PIVC neurones to visual and vestibular stimulation with 'trapezoidal' velocity profiles and corresponding combined visual and vestibular stimuli. This figure demonstrates the different response types of PIVC neurones to horizontal vestibular and visual stimulation. The neurone of panel *a* was a synergistic unit, that of panel *b* an antagonistic unit, while that of panel *c* exhibited a biphasic response. This neurone (*c*) was classified for horizontal rotation type I yaw and type III optokinetic. In addition it responded to movement in a roll direction (type I) and pitch (type II). Interaction between neck receptor and vestibular input was synergistic. The neurone of panel *c* was also activated by pressure exerted on neck and shoulders and by deep mechanoreceptors in the hip region and along the vertebrae. Responses of the three neurones to horizontal chair rotation in the dark (*A*), horizontal drum rotation with stationary chair (*B*), horizontal chair rotation in the light with stationary drum (*C*) and coupled horizontal chair and drum rotation (*D*) are shown with horizontal electro-oculogram (EOG) and vertical electro-oculogram recordings for the neurone of panel *a*. The PSTHs are obtained from the continuous responses and are not averaged. Note that with synchronous rotation of chair and drum VOR was only partially suppressed. Further remarks are given in the text.

As expected, visual directional selectivity was also present when constant speed horizontal rotation instead of sinewave rotation of the striped drum was applied successively in both directions. Examples of responses to this type of optokinetic stimulation, which we called 'trapezoidal' with respect to velocity changes, are depicted in Fig. 4*B*. For the neurone of panel *a*, responses to optokinetic sinewave and to optokinetic 'trapezoidal' stimulation can be compared (Figs 3 and 4). This neurone was activated by constant-speed horizontal cylinder rotation to the left and exhibited a 'synergistic' vestibular response (Fig. 4*A* and *C*). In contrast, the neurone of panel *b* was activated during cylinder rotation to the right (Fig. 4*B*) and, in an 'antagonistic' manner, also during rotation of the animal in darkness towards the right (Fig. 4*A*).

The average neuronal impulse rate increased, as a rule, with the angular velocity of the optokinetic stimuli moving in the preferred direction up to about 60 deg s^{-1}. Correspondingly, an inhibitory effect during maintained unidirectional optokinetic stimulation in the non-preferred direction became more pronounced when optokinetic angular velocity increased (neurone of Fig. 4*Ba*). All fifteen neurones tested with the 'trapezoidal' velocity profile of optokinetic or horizontal rotational stimuli exhibited responses which were qualitatively concordant with the vestibular and optokinetic classification obtained during sinewave stimulation. Activation evoked by constant-speed horizontal rotation in the dark decreased as anticipated with the duration of the stimulus, while optokinetic stimulation at a constant velocity led to little adaptation.

Optokinetic–vestibular interaction

As in man, sinusoidal horizontal rotation of a monkey in the dark evoked a vestibulo-ocular reflex (VOR): smooth eye movements in the opposite direction to head rotation interrupted by fast backward saccades. VOR was suppressed in awake animals when the visual surround rotated with the animal. At high stimulus frequencies or when the animals were fatigued, the VOR suppression was not complete.

We combined optokinetic and vestibular stimuli in different ways: (a) chair rotation in light ('natural' combination), (b) chair and drum in phase rotation (VOR suppression), (c) synchronous chair and drum rotation 180 deg out of phase (VOR doubling) and (d) synchronous chair and drum rotation in phase but with doubled drum amplitude (VOR inversion).

These stimuli were also used by Waespe & Henn (1978) in vestibular nuclei recordings. Of the twenty-nine synergistic neurones, eleven were tested quantitatively with 0·2 Hz sinewave stimuli and these four stimulus combinations. For six neurones stimulus amplitudes of 56 deg were chosen, for five neurones amplitudes of 15 deg (the average gain of the neurones tested with the larger amplitudes was about a third of that with the 15 deg amplitude). The mean gain of these 'synergistic' neurones during rotation in the light ($0{\cdot}36 \pm 0{\cdot}39$ (impulses s^{-1}) $(\text{deg } s^{-1})^{-1}$) did not essentially change as compared with the gain for rotation in darkness, which was on average $0{\cdot}35 \pm 0{\cdot}4$ (impulses s^{-1}) $(\text{deg } s^{-1})^{-1}$. The analysis of the data obtained in individual neurones showed that a few increased their activation significantly during rotation in the light as opposed to rotation in the dark. Other neurones, however,

decreased neuronal activation under the same conditions although the opposite was to be expected due to the 'synergistic' response mode found when tested with vestibular or visual stimulation alone. Thus a considerable variability in visual–vestibular interaction in synergistic PIVC neurones was noted, extending from a reduction of gain to a significant summation of vestibular and visual responses. Figure 4*A–D* demonstrates the response patterns of three different neurones obtained with trapezoidal velocity profiles using the four experimental protocols (a)–(d) mentioned above. In the 'synergistic' neurone of panel *a* angular acceleration to the right in total darkness led to an activation (Fig. 4*A*), while horizontal drum rotation was an activating stimulus when the rotation direction was to the left. Chair rotation in the light led to an increased directional selectivity (Fig. 4*C*). The neurone only responded when the animal was rotated towards the right. Deceleration during rotation to the left, which led to a slight activation in the dark (Fig. 4*A*), suppressed neuronal activity, due to the inhibiting visual input aroused by rotation in the light (Fig. 4*C*).

Seven PIVC neurones having a visual–vestibular antagonistic response were quantitatively tested for visual–vestibular interaction with sinewave stimuli of 0·2 Hz. The comparison of the mean gains obtained with rotation in darkness ($0{\cdot}16 \pm 0{\cdot}1$ (impulses s^{-1}) $(\text{deg } s^{-1})^{-1}$) and in light ($0{\cdot}20 \pm 0{\cdot}18$ (impulses s^{-1}) $(\text{deg } s^{-1})^{-1}$) revealed no significant change in the activity of the entire neurone population. In all of these units the vestibular stimulus always remained dominant (determined by phase angle *vs.* velocity) when the animal was rotated in the light, as shown in Fig. 3.

Eleven synergistic PIVC neurones were tested with in-phase rotation of drum and chair (0·2 Hz, VOR suppression). The mean gain was $0{\cdot}41 \pm 0{\cdot}5$ (impulses s^{-1}) $(\text{deg } s^{-1})^{-1}$ for samples with 56 and 15 deg stimulus amplitude and was not significantly different from the mean gain of rotation in darkness or when general room illumination was turned on and the cylinder was stationary. An example of the neuronal behaviour evoked by these stimulus conditions is shown in the neurone of Fig. 2. The response pattern of this neurone became biphasic when drum and chair were rotated in phase (Fig. 2*D*) and the modulation was significantly below that observed when the chair was rotated within a stationary illuminated surround (Fig. 2*C*). The same general response changes were observed when trapezoidal velocity stimuli were used (compare Fig. 4*C* and *D*).

As in the synergistic neurones, no significant difference in the average gain of antagonistic PIVC neurones ($0{\cdot}17 \pm 0{\cdot}1$ (impulses s^{-1}) $(\text{deg } s^{-1})^{-1}$) was present when responses to rotation in the dark were compared to those obtained with rotation within a stationary illuminated surround, or with in-phase rotation of the cylinder and the chair. Some neurones, however, showed a slight enhancement of the gain when drum and chair were rotated together as compared to rotation in the dark. An example of this response type is illustrated in Fig 3*A* and *D*.

Only seven neurones (four synergistic, three antagonistic) were systematically tested with out-of-phase rotation of drum and chair. All four synergistic units increased the gain of the responses (mean from $0{\cdot}13 \pm 0{\cdot}05$ to $0{\cdot}33 \pm 0{\cdot}28$ (impulses s^{-1}) $(\text{deg } s^{-1})^{-1}$) at 0·2 Hz, all neurones tested with 56 deg amplitude from in-phase rotation to out-of-phase rotation (Fig. 2*D* and *E*). In three antagonistic neurones

tested the average gain did not change under these stimulus conditions, showing again that the vestibular input was dominant (Fig. 3*D* and *E*).

The responses of the same seven neurones were explored with the in-phase stimulus combination, in which the drum amplitude was twice that of chair rotation. All synergistic neurones then displayed a biphasic response (frequency doubling), indicating that under these conditions visual and vestibular activation were dissociated and the vestibular dominance was weaker than during rotation within an illuminated stationary surround (compare panels *C* and *F* of Fig. 2). In the three antagonistic neurones tested with this stimulus combination, the neuronal responses were not significantly changed from those evoked by the in-phase stimulus combination (compare panels *D* and *F* of Fig. 3), and the vestibular input remained dominant. In summary, studying visual–vestibular interaction in synergistic and antagonistic PIVC neurones we found a fairly regular dominance of vestibular input. Despite the fact that the optokinetic stimulus alone led to a fairly strong modulation of the neuronal impulse rate, all intermediate types of visual–vestibular summation from 'no summation' to approximately algebraic summation co-existed in different neurones.

Concerning the VOR gain, it can be seen from Figs 2 and 3 that apart from a difference with the 'VOR suppression' condition similar values were measured for both units. Monkey OL only suppressed the VOR incompletely, whereas monkey RV could do this almost completely. With the 'VOR inversion' stimulation the VOR gain is given in negative values, indicating that the visual stimulation had a stronger impact on the resulting eye movements. This 'VOR inversion' was observed while testing all seven units with this stimulation and was present in all animals when this stimulation was used for measurement of VOR and optokinetic nystagmus interaction. Because of the rather similar VOR gain the possibility can be excluded that the division in antagonistic and synergistic units is an artifact due to different (reflexive) eye movements.

Visual directional selectivity of the neuronal response

As mentioned in the section on responses to optokinetic stimulation, we explored the efficacy of optokinetic stimulus patterns moving in different directions through the visual receptive fields of PIVC neurones. During this study it became clear that the respective preferred stimulus directions evoking a maximum neuronal activation were related to the visual field co-ordinates and not to extrapersonal space co-ordinates. This question was systematically explored in three PIVC neurones. A large visual pattern, a disc of 90 cm diameter covered by parallel black and white stripes of 3·5 cm period, was moved approximately sinusoidally to and fro 40 cm in front of the animal. Movement directions were always perpendicular to the stripe orientation and were varied systematically (Fig. 5): the head and body position of the animal was either upright or tilted 30 deg towards the left or the right. Corresponding to the optimum vestibular *vector* (cf. Grüsser, Pause & Schreiter, 1990) this neurone was activated maximally to optokinetic pattern movement from the lower left to the upper right quadrant of the field of gaze. As Fig. 5 demonstrates, a weak activation was also evoked when the optokinetic stimulus moved to the right or vertically upwards. After tilting the monkey 30 deg to the right, horizontal

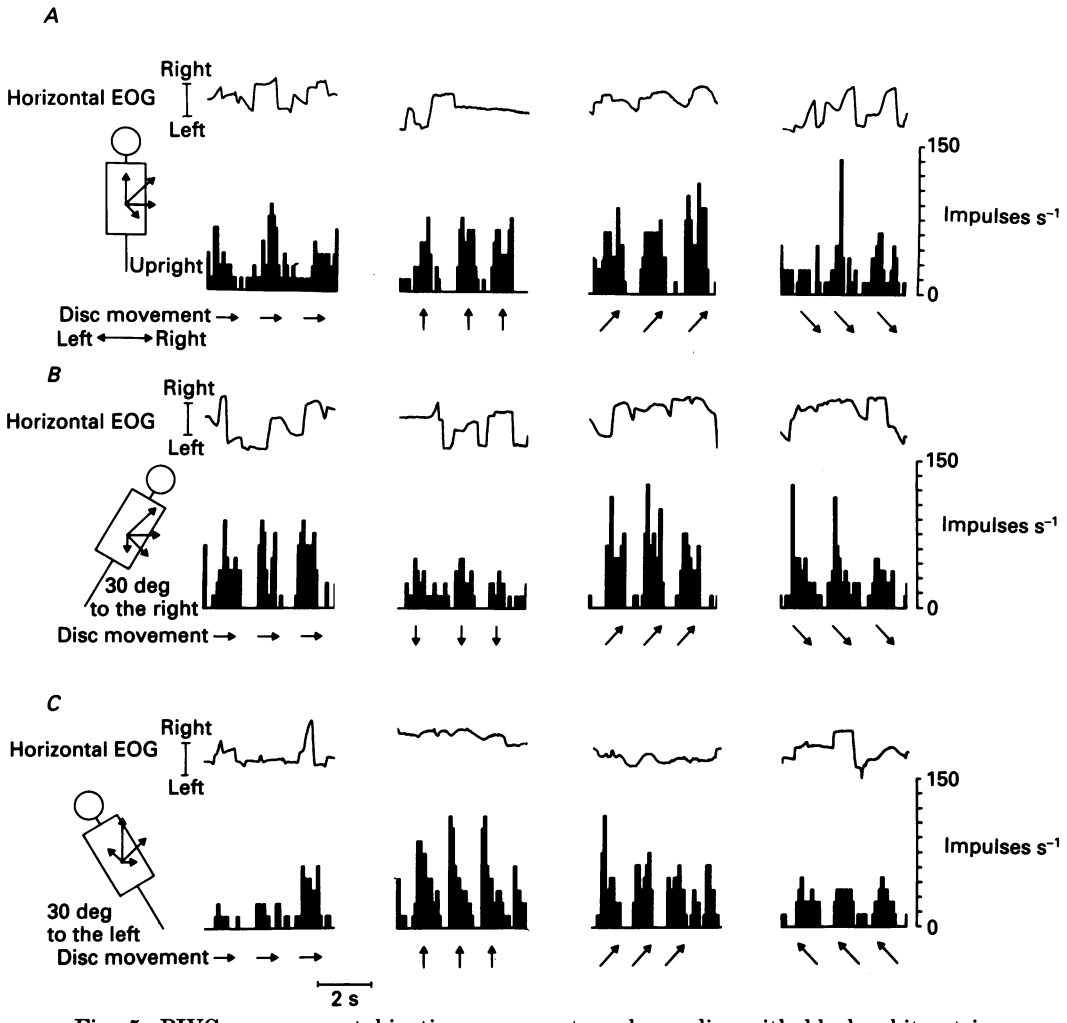

Fig. 5. PIVC neurone optokinetic responses to a large disc with black–white stripe patterns (3·5 deg period) moved approximately sinusoidally in front of the animal in different directions. The neurone exhibits a clear directional selectivity, which is relative to the co-ordinates of the visual field and not to the earth-related co-ordinates. This head-fixed directional selectivity is demonstrated when the animal is brought into different tilt positions (roll direction). *A*, animal in upright position. The arrows below the PSTHs mark movement direction of the striped disc and are always adjusted to the activating direction. *B*, animal tilted 30 deg to the right; arrows indicate, as in *A*, movement direction of striped disc in earth-related co-ordinates. *C*, animal tilted 30 deg to the left. Note from the horizontal EOG recordings that pursuit movements aroused by the movement of the stripes and voluntary saccades are intermingled. The length of the arrows within the schematic drawing of the animal body indicates the relation of average discharge rates from consecutive disc movement periods in the direction shown by the arrows. Vestibular response types of this synergistic neurone were type I yaw, type II roll, type III pitch.

pattern movement towards the right (direction relative to earth-fixed co-ordinates) aroused the strongest response, while activation evoked by pattern movement in the vertical direction was weak. Tilting the animal 30 deg towards the left again shifted the preferred movement direction. Maximum responses were not obtained when the stripe pattern moved upwards, while only a weak activation was seen when the pattern was moved in the horizontal direction (Fig. 5; lower line). These findings are readily explainable by the assumption that the preferred direction was related to the co-ordinates of the visual field or the field of gaze (head co-ordinates) and not to the gravitational co-ordinates of the extrapersonal space. The same characteristics were observed in the other two PIVC neurones tested systematically with this set of stimuli.

Responses of PIVC neurones to somatosensory stimulation

Somatosensory receptive field properties

Nearly all vestibular PIVC neurones responded to adequately selected somatosensory stimuli applied in total darkness. The majority of PIVC neurones had somatosensory receptive fields located in the neck and/or shoulder region. Lightly touching the skin or slight movement of a stimulus across the skin was rarely an adequate stimulus (only one unit with receptive field contralateral, two units with receptive fields bilateral), while an increase in neuronal activity was recorded in six units with ipsilateral, in six units with contralateral and forty-one units with bilateral deep pressure exerted on the muscles. Five neurones were activated by contralateral, five neurones by bilateral passive movement of arm joints. We tried to identify neurones which were activated only by simultaneous movement of more than one joint (called 'disjoint neurones' by Mountcastle, Lynch, Georgopoulos, Sakata & Acuna, 1975; Sakata, 1975; Hyvärinen, 1982). No such neurones could be found in the area PIVC. A small percentage (five units = 12%) of PIVC neurones responded to vibration of the vertebral column and/or the pelvis bilaterally. These neurones were also activated as a rule when the whole monkey chair was joggled. Other PIVC neurones (one contralateral, eight bilateral) responded to pulling the legs or arms or to proximal joint movements of the extremities. When the monkey performed active movements mimicking the effective passive stimulus pattern, about the same activation was evoked as with passive stimulation. Thus the main activating input to PIVC neurones related to movements of legs or arms seems to originate in peripheral mechanoreceptors and not within the central nervous system.

Neck receptor stimulation

Rotation of the body while the head was fixed in space was the most effective somatosensory stimulus activating PIVC neurones. This trunk rotation stimulates the mechanoreceptors of joints, tendons, muscles and the skin of the neck region. Since superficial skin stimuli did not activate PIVC neurones, we assume that horizontal trunk rotation, to be referred to hereafter as 'neck receptor stimulation', changes PIVC neurone activity by deep mechanoreceptor input. A pronounced directional selectivity was observed for this type of stimulation as well: about half of the thirty-two PIVC neurones tested were activated when the trunk was rotated

towards the side contralateral to the hemisphere in which the neurones were recorded (fifteen type IIn, n = neck); the other half responded to trunk rotation towards the ipsilateral side (fourteen type In units). Three neurones exhibited a type IIIn response, i.e. trunk rotation to the ipsilateral and contralateral side led to an increase

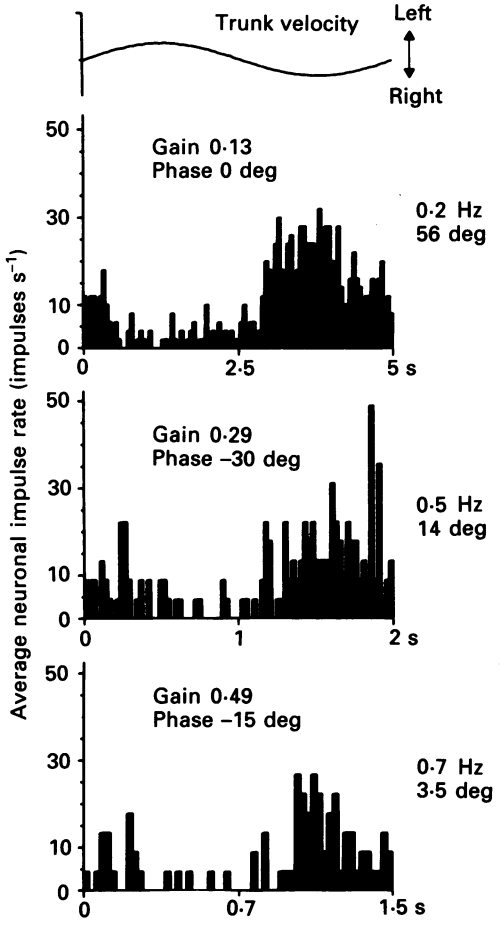

Fig. 6. Sinusoidal trunk rotation in the dark with different frequencies. Note the increase in (computed) gain with higher stimulation frequency.

in neuronal activity. The neck receptor response evoked by trunk rotation was not dependent on the visual input from a stationary visual surround and was the same whether recorded in the dark or in light. In our experiments trunk rotation in the dark or light (head fixed in space) rarely evoked a cervico-ocular reflex (Fig. 8*C*, Fuller, 1980). Thus responses to stimulation of neck receptors in our study were not caused indirectly by eye movements.

The neuronal activity was dependent upon trunk rotation frequency and amplitude. The 'gain' of trunk rotation (Figs 6 and 7) was found to be in the same range as that of the responses to vestibular stimuli (cf. Fig. 8 in Grüsser, Pause &

Schreiter 1990). The phase angle between neuronal responses as revealed by the PSTHs and stimulus velocity was rather variable between different PIVC neurones. With trunk rotation above 0·5 Hz in the dark or light an average phase lead of about 30 deg (relative to the trunk rotation velocity in the effective direction) was found.

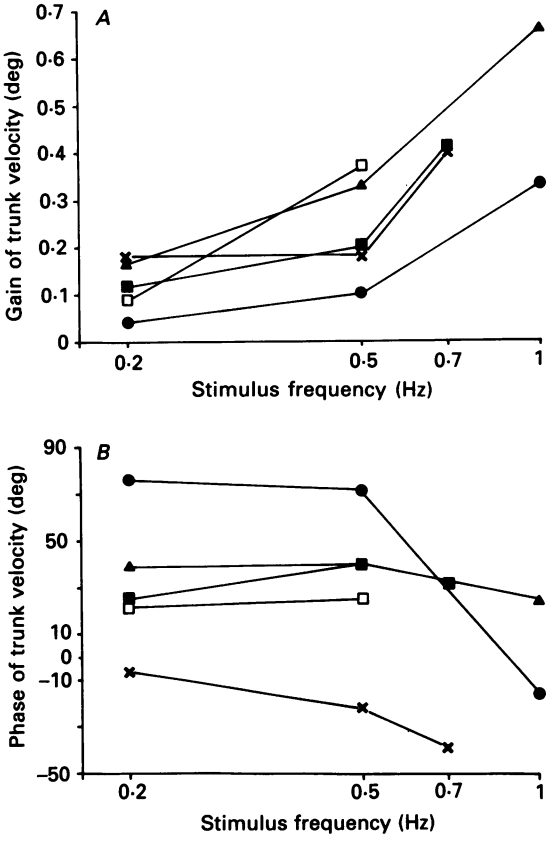

Fig. 7. Phase (*A*) and gain (*B*) computed from PSTHs obtained in five different PIVC neurones with horizontal trunk rotation (head fixed in space) of different stimulus frequencies (abscissa). Room illumination turned on; the monkey saw a stationary vertical stripe drum.

Thus not only stimulus velocity but also stimulus acceleration had an impact on the neuronal impulse rate (Fig. 7).

Interaction of neck receptor and vestibular inputs

In twenty-two PIVC neurones the interaction of horizontal trunk rotation (neck receptor stimulation) and horizontal vestibular stimulation (chair rotation) was studied. The responses to these two stimulus classes were compared to those evoked by passive sinusoidal head rotation in the dark (trunk stationary in space). Examples of data obtained in these studies are shown in Figs 8 and 9. We could distinguish two

types of vestibular–neck receptor interactions: the 'cancellation type' is represented by the responses of the neurone shown in Fig. 8 (the vestibular and optokinetic responses of the same neurone are shown in Fig. 4, panels *a*). An activation was evoked when the trunk was rotated to the right and the same was true for vestibular

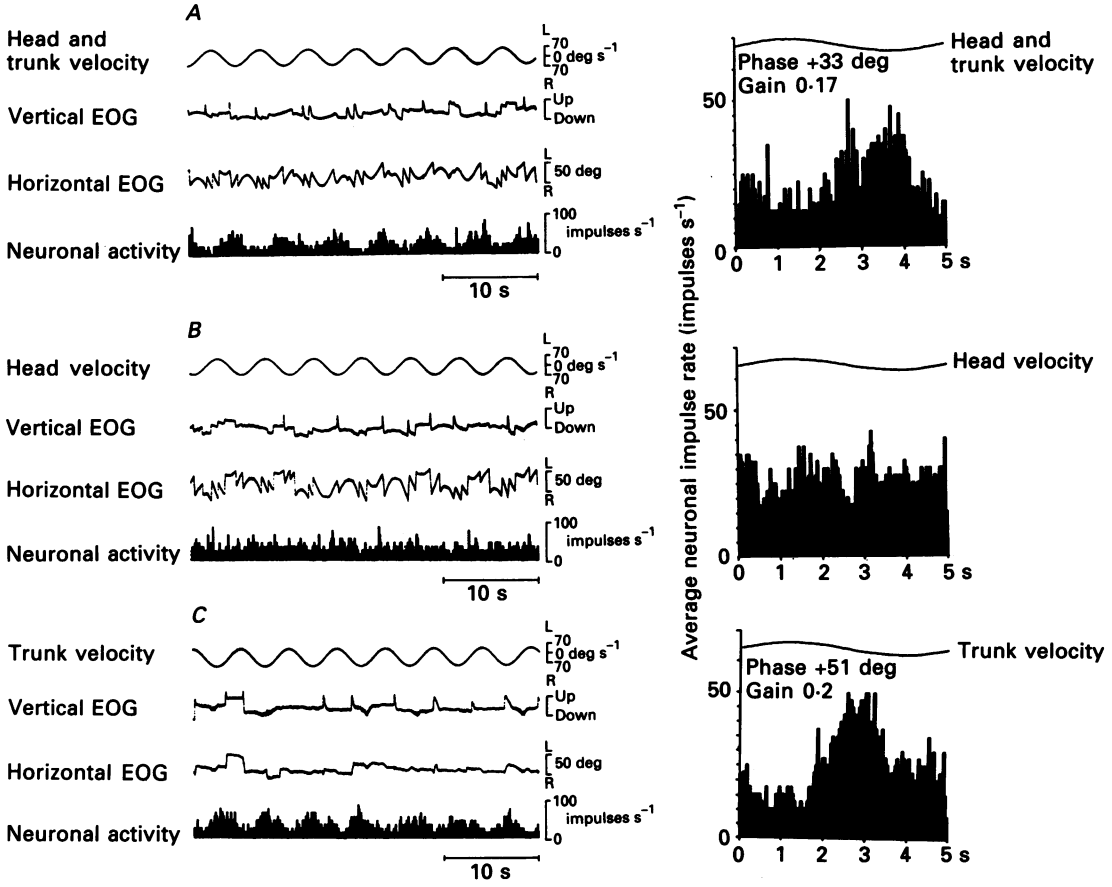

Fig. 8. PIVC neurone responding to vestibular input and neck receptor input with cancellation mode. Horizontal sinusoidal rotation (0·2 Hz) in the dark. Activation when the animal was rotated towards the right. Note the pronounced vestibulo-ocular reflex visible in the horizontal EOG (*A*). When the head is fixed in space and the trunk is rotated sinusoidally, the unit is also activated during trunk movement to the right (i.e. head position changes relative to the trunk to the left (*C*)). Consequently passive rotation of the head in the dark with stationary trunk elicits a rather irregular neuronal activation with the tendency to small biphasic modulation (*B*). Note that trunk rotation did not evoke a cervico-ocular reflex (*C*).

stimulation (chair rotation). By sinusoidal head rotation with the trunk stationary, movement of the head towards the right is a vestibular stimulus to the right, but corresponds to a neck receptor stimulus present when the trunk is rotated to the left (head remains stationary). As the example in Fig. 8*B* indicates, the modulation of

the PSTH was minimal when the head was rotated sinusoidally on the stationary trunk, but the average neuronal impulse rate was about twice the spontaneous activity in the dark. Nineteen units were classified as 'cancellation type'; of these ten showed the combination type In/type Iy and nine the combination type IIn/type IIy.

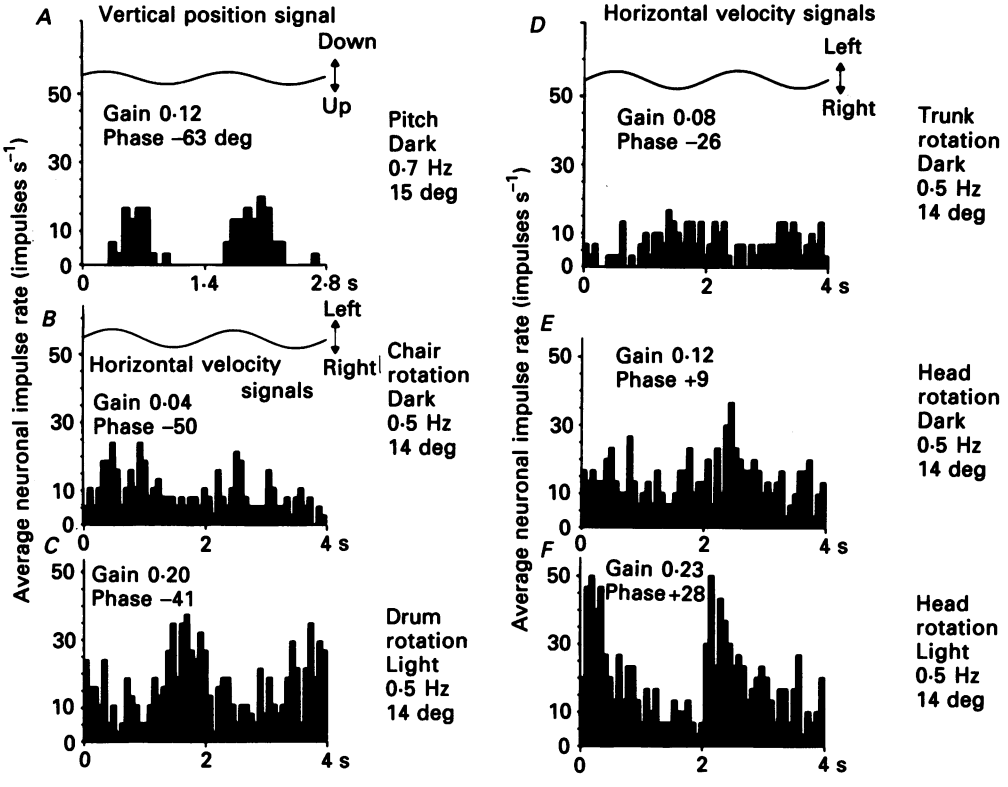

Fig. 9. Interaction of vestibular, visual and proprioceptive inputs in a PIVC neurone. The vestibular response type was pitch I (*A*) and yaw I (*B*). The neurone responded to horizontal sinusoidal rotation of the stripe cylinder (*C*) in a synergistic way with the vestibular input. The vestibular–neck receptor interaction was also of the synergistic type, but the activation by selective stimulation of neck receptors (trunk rotation in the dark, *D*) was rather weak. Response to head rotation in the dark (*E*) was significantly stronger than to chair rotation in the dark (*B*). The most prominent modulation of impulse rate was obtained when the head was rotated and a stationary vertical stripe cylinder was visible (*F*).

In addition to the nineteen neurones with responses corresponding to this 'cancellation type', three PIVC neurones (one type In/type IIy and two type IIn/type Iy) were recorded which showed the contrary directional selectivity in their responses evoked by trunk rotation and chair rotation. Consequently the vestibular and neck receptor activation evoked during head rotation facilitated each other. An example of such facilitatory responses is shown in Fig. 9. This neurone responded to vertical sinewave rotation in the pitch direction when the nose was moved upwards (Fig. 9*A*) and somewhat less to horizontal chair rotation towards the

left (Fig. 9*B*). Trunk rotation towards the right led to an increase in neuronal activity. Therefore a considerably stronger modulation of the neuronal impulse rate was found when the head was rotated in the dark, since neck receptor and vestibular activation were then in phase. Maximum activity was obtained when the head was

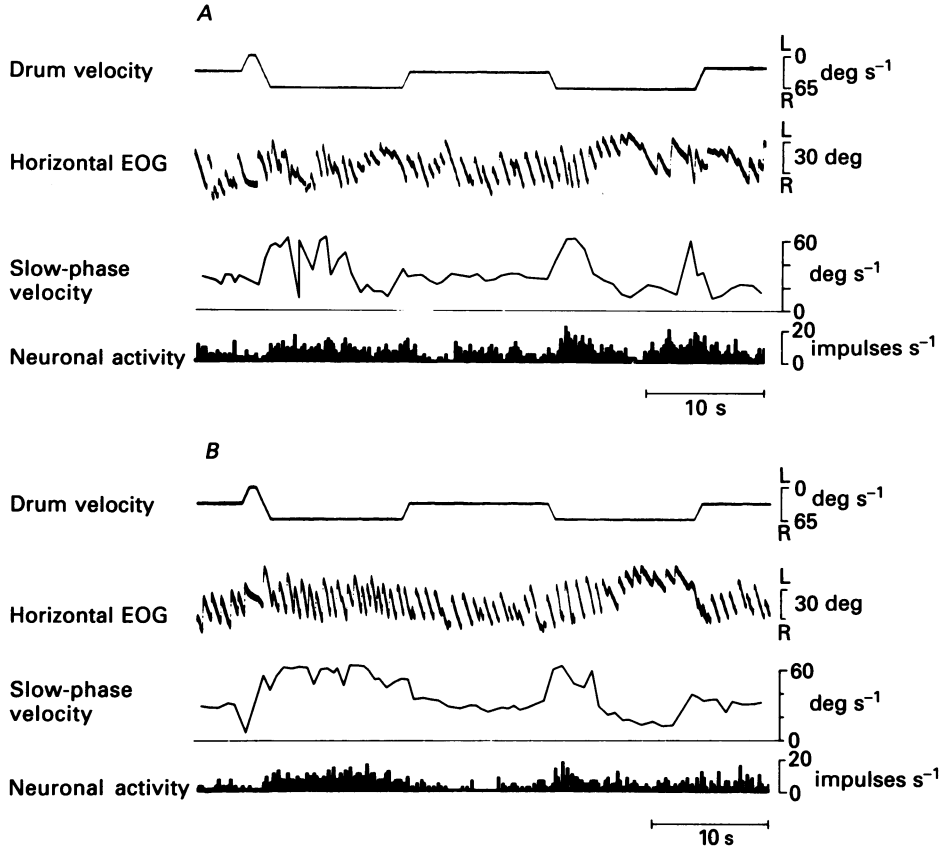

Fig. 10. PIVC neurone responding to optokinetic horizontal drum rotation when the stripe cylinder moved towards the right. Responses to different velocities (32 and 65 deg s^{-1}) and positive or negative velocity steps. In addition to the neuronal activity the horizontal EOG and the measured slow-phase velocity of horizontal optokinetic nystagmus are displayed. Note that activity of this neurone was loosely correlated with the horizontal optokinetic slow-phase angular velocity. *A* and *B* show responses to the same sequence of optokinetic stimulation. The vestibular response type of this neurone was type II yaw, type I roll and type I pitch. It was activated by touch and pressure exerted on the neck and both shoulders and by trunk rotation to the right. During the recording period shown in this figure the monkey was in a fully alert state.

rotated to the right on the stationary trunk. Comparing panels *E* and *F* of Fig. 9 it can be seen in addition that the retinal stimulation caused by head rotation in the light enhanced the neuronal activation further. This finding corresponded to the observation that this neurone did belong to the 'synergistic' vestibular/optokinetic response type.

In a few PIVC neurones the changes in neuronal activity obtained during passive head rotation were compared with those observed during active head rotation in the dark. No obvious differences were found. This problem, however, should be studied quantitatively when animals have been trained to perform specific head movements

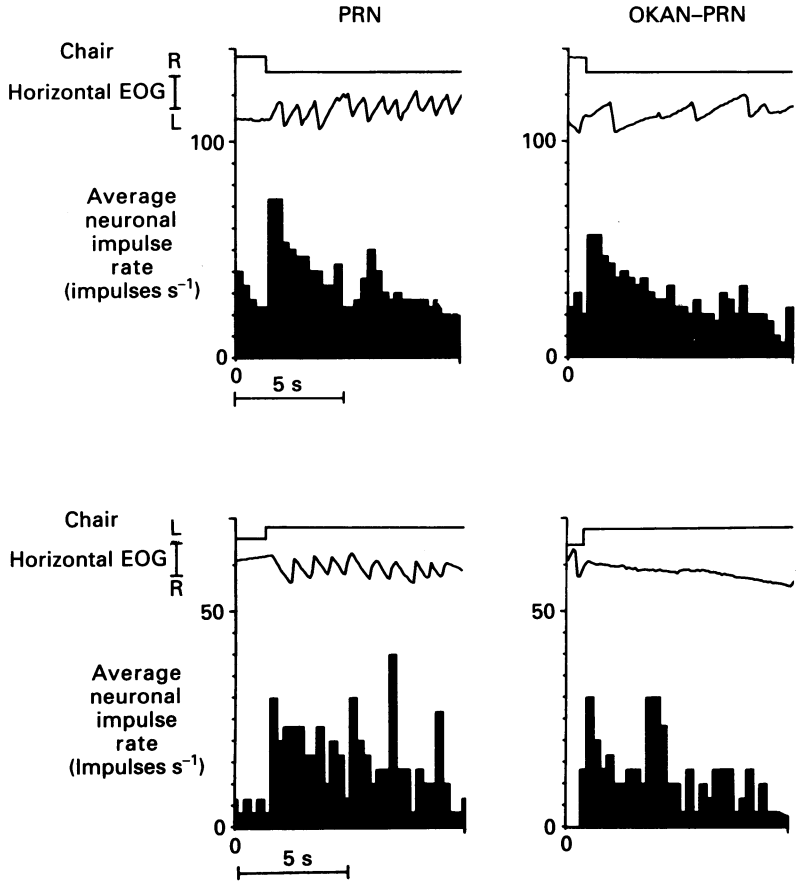

Fig. 11. Response of two PIVC neurones to a sudden stop after constant chair rotation in the dark, eliciting a postrotatory nystagmus (PRN, left side). The postrotatory nystagmus is cancelled by OKAN directed to the opposite side since light within the striped drum was on during constant rotation and switched off 0·5 s before the sudden stop (OKAN–PRN, right side).

repeatedly. In our experiments we had to wait until the animal moved its head spontaneously to the left or the right.

Saccadic eye movements and PIVC neurone activity

Recording the activity of single neurones of the brain stem vestibular nuclei one finds that many of these neurones change their activity shortly before or during a saccade (Miles, 1974; Tomlinson & Robinson, 1984; authors' observations). We never observed such saccade-related changes (burst or pauses) in the neuronal discharge rate of PIVC neurones when the monkey was sitting in the dark gazing about at will.

The same was true when saccades were performed in the light across the wall of the stripe cylinder or the well-structured laboratory surroundings.

Nystagmus and PIVC neurone activation

The angular velocity of the optokinetic or vestibular nystagmus slow phase was determined and correlated with the neuronal activity. Figure 10 demonstrates that with identical optokinetic velocity stimulus profiles the responses of this neurone varied approximately in parallel to the angular velocity of the optokinetic slow phase. This finding, however, does not suggest a direct interdependence of the two values. In other neurones no close relationship between optokinetic nystagmus slow-phase angular velocity and PIVC neuronal impulse rate was found. The difference between the eye velocity and stimulus angular velocity, the retinal 'slip velocity', usually increased with increasing stimulus speed. Retinal slip velocity might influence neuronal activation during optokinetic nystagmus. On the other hand neuronal activity during optokinetic after-nystagmus (OKAN I or OKAN II) was only loosely correlated, if at all, to the angular velocity of the slow nystagmus phases. When the drum illumination was switched off during optokinetic stimulation, the neuronal impulse rate decreased within a few seconds, while the optokinetic after-nystagmus OKAN I lasted up to 20 s (cf. also Büttner & Henn, 1976). The independence of nystagmus mechanisms and PIVC neurone activity was corroborated by another observation: long constant-velocity rotation of the animal inside a stationary illuminated stripe drum led to a strong optokinetic nystagmus. When the animal was suddenly stopped after 25 s of constant rotation and simultaneously the illumination was turned off, postrotatory nystagmus and OKAN were in opposite directions and cancelled each other more or less completely. The change in the PIVC neurone impulse rate, however, corresponded closely to that evoked by suddenly stopping the rotating chair in the dark as shown for two different units in Fig. 11.

DISCUSSION

Visual and optokinetic responses of PIVC neurones

In his 'Die Lehre von den Bewegungsempfindungen' (1875) Ernst Mach described vestibular sensations (circular and linear vection) induced by visual stimuli. He postulated that visual signals are integrated into the vestibular signal flow somewhere within the central nervous system. Ohm (1943) deduced from clinical observations that visual and vestibular signals interact at the brain stem vestibular nuclei. He believed this mechanism to be essential for controlling eye movement during optokinetic nystagmus. The study of Henn *et al.* (1974) confirmed Ohm's hypothesis in demonstrating that visual (optokinetic) stimulation changes the activity of brain stem vestibular nuclei neurones in monkeys. Visual–vestibular interaction was also observed in neurones of the ventro-posterior nucleus of the monkey thalamus (Büttner & Henn, 1976) and the perigeniculate region (Magnin & Fuchs, 1977). In the cortical area 2v of Rhesus monkeys Schwarz & Frederickson (1971) did not find 'a single' visually activated neurone, while Büttner & Buettner (1978) reported that 80% of vestibular units of area 2v were activated by

optokinetic stimulation. In single neurones of the cat ASSS field, the presumed homologue of the PIVC area, Grüsser, Grüsser-Cornehls & Saur (1959) found short latency activation to electrical polarization of the labyrinth but no responses to diffuse stationary 'on–off' light stimuli. Mergner (1979) mentioned briefly that moving visual patterns might change the activity of cat ASSS neurones.

None of the authors mentioned could find well-defined visual receptive fields, but all agreed that large optokinetic patterns were effective stimuli to activate cortical vestibular neurones. These observations are congruent with the present report. Evidently the majority of monkey PIVC neurones have very large binocular visual receptive fields. The question whether this is also true for those PIVC neurones which responded to small moving visual targets must remain open till monkeys trained to fixate the centre of gaze during exploration of the visual fields can be studied. It should be mentioned, however, that visual tracking neurones of area 7 were found to have a well-defined, relatively small, visual receptive field (Robinson, Goldberg & Stanton, 1978). Some of these visual tracking neurones are also activated by vestibular stimulation, according to the results of Kawano, Sasaki & Yamashita (1980, 1984).

Comparing the optokinetic responses of brain stem and thalamus vestibular neurones with the present data, one is led to the conclusion that a change in visual–vestibular interaction occurs along the afferent vestibular pathway. Neurones with 'antagonistic' visual–vestibular responses were found in less than 5% in the vestibular nuclei (Waespe & Henn, 1977*a*), in about 13% in thalamus (Büttner & Henn, 1976) and in area 2v (Büttner & Buettner, 1978). Since one-third of the PIVC neurones in our study belonged to the antagonistic type of visual–vestibular interaction, one can speculate that the vestibular thalamic and cortical neurones receive additional optokinetic signals from the central visual system.

Synergistic PIVC neurones seem to monitor predominantly head-in-space movement, whereby the visual input supports the vestibular signals originating in the semicircular canal receptors by extending the sensitivity into the lower velocity range (cf. also Waespe & Henn, 1978). The functional role of the antagonistic neurones is more difficult to explain. From the differences in the responses of antagonistic and synergistic neurones, however, the system could discriminate within limits between body movements and surround movements.

In the vestibular nucleus of the monkey brain stem the visual–vestibular interaction was analysed only for 'synergistic' neurones by Waespe & Henn (1977*a*, *b*) and by Buettner & Büttner (1979). Comparing their data with the PIVC neurone responses, some similarities became evident. Responses such as those found in the neurone of Fig. 2 and panel *a* of Fig. 4 were very similar to those obtained in brain stem vestibular neurones: reduction in impulse rate with synchronous rotation of drum and chair, summation of activation evoked by optokinetic and vestibular stimuli with the 'VOR doubling' condition. As in cortical neurones, brain stem vestibular neurones showed only a slight increase in neuronal activation when the responses obtained during the VOR doubling condition were compared to those measured during rotation in light. Thus, a non-linear visual–vestibular interaction seems to be characteristic of vestibular neurones in the brain stem and in the vestibular cortex. An essential difference between visual–vestibular interaction in brain stem vestibular nuclei and PIVC neurones was a definitely higher dependence

of cortical unit activity on the animal's state of alertness. In addition, cortical vestibular neurones have a lower general discharge level and discharge less regularly than brain stem vestibular neurones.

We believe that PIVC neurones are involved in the perception of head rotation or self-rotation as induced by either body rotation or circular vection. The results of psychophysical examinations by Büttner & Henn (1981) and Zacharias & Young (1981) demonstrated that the intensity of circular vection declines parallel to an increase in phase lag with stimulus frequencies above 0·5 to 1·0 Hz, a finding which correlates with the decrease in optokinetic sensitivity and the phase shifts of some PIVC neurones at 0·5 and 1·0 Hz. From our sample consisting of synergistic as well as antagonistic units, however, we could not confirm the conclusion of Henn *et al.* (1974) that circular vection can be explained by the 'reciprocal' visual movement sensitivity of neurones in the vestibular nuclei.

PIVC unit responses to neck receptor stimulation and vestibular neck movement interaction

In creating an internal representation of body and head position and their movement in space, reliable information about head and trunk movement is necessary. Oblique neck muscles contain numerous muscle spindles and Golgi tendon organs and the density of mechanoreceptors in the joints of the neck is also high (Bakker & Richmond, 1982; Richmond & Bakker, 1982). Several authors are of the opinion that the proprioceptive signals from the neck muscles form the main non-vestibular input, activating the neurones of the vestibular nuclei in cats (Rubin, Young, Milne, Schwarz & Fredrickson, 1975; Boyle & Pompeiano, 1979; Kasper & Thoden, 1981; Anastasopoulos & Mergner, 1982). In Rhesus monkeys the thalamic vestibular nuclei (ventro-posterior complex) also contain a high percentage of neurones activated by vestibular and by neck proprioceptive inputs (Deecke, Schwarz & Fredrickson, 1977). These thalamic neurones also receive some proprioceptive signals from the arms. In cat cortical ASSS neurones, Becker, Deecke & Mergner (1979) and Mergner, Anastasopoulos, Becker & Deecke (1981) found that the sensitivity to vestibular and to neck receptor stimulation was about the same. As in the present study, the 'complexity' of the ASSS neurone responses was high and a fairly high variability of multimodal signal convergence was present in different neurones (Mergner *et al.* 1981).

Since 'vestibular' cortical neurones of PIVC were intensively activated by dynamic neck mechanoreceptor signals, one expects neck receptor stimulation by trunk rotation to induce movement in psychophysical experiments. This was indeed observed by de Jong *et al.* (1981), Bles & de Jong (1982) and Mergner *et al.* (1983).

Pathways for transmission of visual signals to PIVC

While vestibular as well as somatosensory signals reach PIVC through the vestibular nuclei and the thalamic relay nuclei, the visual input to PIVC probably uses two main connections. One extends from the retina to the nucleus of the optic tract (NOT), and from there to the vestibular nuclei, which project via thalamic relay nuclei to PIVC. The other input uses the projections from the retina to the superior colliculi, from there to the visual inferior pulvinar and then to the anterior pulvinar, which projects directly into PIVC, as recent horseradish peroxidase studies

have revealed (Akbarian, Berndl, Grüsser, Guldin, Pause & Schreiter, 1988). Surprisingly, no direct visual input seems to exist from the occipital or occipito-temporal visual regions, which are sensitive to large movement visual stimuli (e.g. the areas MT or MST). A strong input to PIVC was found from area 3 (neck region) and from the prefrontal eye fields (area 8 and surrounding regions) in the tracer studies mentioned. One could speculate that these input signals might be used to modify the spatial co-ordinates of retinal input to PIVC.

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REFERENCES

AKBARIAN, S., BERNDL, K., GRÜSSER, O.-J., GULDIN, W., PAUSE, M. & SCHREITER, U. (1988). Responses of single neurons in the parieto-insular vestibular cortex of primates. *Annals of the New York Academy of Sciences* **545**, 187–202.

ANASTASOPOULOS, D. & MERGNER, T. (1982). Canal–neck interaction in vestibular nuclear neurons of the cat. *Experimental Brain Research* **46**, 269–280.

BAKKER, D. A. & RICHMOND, F. J. R. (1982). Muscle spindle complexes in muscles around upper cervical vertebra in the cat. *Journal of Neurophysiology* **48**, 62–74.

BECKER, W., DEECKE, L. & MERGNER, T. (1979). Neuronal responses to natural vestibular and neck stimulation in the anterior suprasylvian gyrus of the cat. *Brain Research* **165**, 139–143.

BLES, E. & DE JONG, J. M. B. V. (1982). Cervico-vestibular and visuo-vestibular interaction. *Acta otolaryngologica* **94**, 61–72.

BÖTZEL, K., DALBASTI, T. & GRÜSSER, O.-J. (1981). Horizontal circular vection and eye movements. *Freiburger Universitätsblätter* **74**, 113—117.

BOYLE, R. & POMPEIANO, O. (1979). Frequency response characteristics of vestibulospinal neurons during sinusoidal neck rotation. *Brain Research* **173**, 344–349.

BUETTNER, U. W. & BÜTTNER, U. (1979). Vestibular nuclei activity in the alert monkey during suppression of vestibular and optokinetic nystagmus. *Experimental Brain Research* **37**, 581–593.

BÜTTNER, U. & BUETTNER, U. W. (1978). Parietal cortex (2v) neuronal activity in the alert monkey during natural vestibular and optokinetic stimulation. *Brain Research* **153**, 392–397.

BÜTTNER, U. & HENN, V. (1976). Thalamic unit activity in the alert monkey during natural vestibular stimulation. *Brain Research* **103**, 127–132.

BÜTTNER, U. & HENN, V. (1981). Circular vection: psychophysics and single-unit recordings in the monkey. *Annals of the New York Academy of Sciences* **374**, 274–283.

DEECKE, L., SCHWARZ, D. W. F. & FREDRICKSON, J. M. (1977). Vestibular responses in the rhesus monkey ventroposterior thalamus. II. Vestibulo-proprioceptive convergence at thalamic neurons. *Experimental Brain Research* **30**, 219–232.

DE JONG, J. M. B. V., BLES, W. & BOVENKERK, G. (1981). Nystagmus, gaze shift, and self-motion perception during sinusoidal head and neck rotation. *Annals of the New York Academy of Sciences* **374**, 590–599.

DICHGANS, J. & BRANDT, T. (1978). Visual–vestibular interaction: effects on self-motion perception and postural control. In *Handbook of Sensory Physiology*, vol. VII, *Perception*. ed. HELD, R., LEIBOWITZ, H. & TEUBER, H.-L., pp. 755–804. Springer, Heidelberg, New York.

FREDRICKSON, J. M., SCHWARZ, D. & KORNHUBER, H. H. (1966). Convergence and interaction of vestibular and deep somatic afferents upon neurons in the vestibular nuclei of the cat. *Acta otolaryngologica* **61**, 168–188.

FULLER, J. H. (1980). The dynamic neck–eye reflex in mammals. *Experimental Brain Research* **41**, 29–35.

GRÜSSER, O.-J., GRÜSSER-CORNEHLS, U. & SAUR, G. (1959). Reaktionen einzelner Neurone im optischen Cortex der Katze nach elektrischer Polarisation des Labyrinths. *Pflügers Archiv* **269**, 593–612.

GRÜSSER, O.-J., PAUSE, M. & SCHREITER, U. (1990). Localization and responses of neurones in the parieto-insular vestibular cortex of awake monkeys (*Macaca fascicularis*). *Journal of Physiology* **430**, 537–557.

HENN, V., YOUNG, L. R. & FINLEY, C. (1974). Vestibular nucleus units in alert monkeys are also influenced by moving visual fields. *Brain Research* **71**, 144–149.

HYVÄRINEN, J. (1982). *The Parietal Cortex of Monkey and Man*. Springer Verlag. Berlin, New York.

KASPER, J. & THODEN, U. (1981). Effects of natural neck afferent stimulation on vestibulo-spinal neurons in the decerebrate cat. *Experimental Brain Research* **44**, 401–408.

KAWANO, K., SASAKI, M. & YAMASHITA, M. (1980). Vestibular input to visual tracking neurons in the posterior parietal association cortex of the monkey. *Neuroscience Letters* **17**, 55–60.

KAWANO, K., SASAKI, M. & YAMASHITA, M. (1984). Response properties of neurons in posterior parietal cortex of monkey during visual–vestibular stimulation. I. Visual tracking neurons. *Journal of Neurophysiology* **51**, 340–351.

KLINKE, R. & GALLEY, N. (1974). Efferent innervation of vestibular and auditory receptors. *Physiological Reviews* **54**, 316–356.

MACH, E. (1875). *Grundlinien der Lehre von den Bewegungsempfindungen*. Leipzig. Reprinted by E. J. Bonset, Amsterdam 1967, 128 pp.

MAGNIN, M. & FUCHS, A. F. (1977), Discharge properties of neurons in the monkey thalamus tested with angular acceleration, eye movement and visual stimuli. *Experimental Brain Research* **28**, 293–299.

MERGNER, T. (1979). Vestibular influence on the cat's cerebral cortex. In *Progress in Brain Research*, vol. 50, *Reflex Control of Posture and Movement*, ed. GRANIT, R., & POMPEIANO, O. pp. 567–579. Elsevier, Amsterdam.

MERGNER, T., ANASTASOPOULOS, D., BECKER, W. & DEECKE, L. (1981). Discrimination between trunk and head rotation: a study comparing neuronal data from the cat with human psychophysics. *Acta psychologica* **48**, 291–302.

MERGNER, T., NARDI, G. L., BECKER, W. & DEECKE, L. (1983). The role of canal–neck interaction for the perception of horizontal trunk and head rotation. *Experimental Brain Research* **49**, 198–208.

MILES, F. A. (1974). Single unit firing patterns in the vestibular nuclei related to voluntary eye movements and passive body rotation in conscious monkeys. *Brain Research* **71**, 215–224.

MOUNTCASTLE, V. B., LYNCH, J. C., GEORGOPULOS, A., SAKATA, H. & ACUNA, C. (1975). Posterior parietal association cortex of the monkey: command functions for operations within extrapersonal space. *Journal of Neurophysiology* **38**, 871–908.

OHM, J. (1943). *Die Mikroneurologie des Auges und seiner Bewegung*. Ferdinand Enke, Stuttgart.

RICHMOND, F. J. R. & BAKKER, D. A. (1982). Anatomical organization and sensory receptor content of soft tissues surrounding upper cervical vertebrae in the cat. *Journal of Neurophysiology* **48**, 49–61.

ROBINSON, D. H., GOLDBERG, M. E. & STANTON, G. B. (1978). Parietal association cortex in the primate: sensory mechanisms and behavioural modulations. *Journal of Neurophysiology* **41**, 910–932.

RUBIN, A. M., YOUNG, J. H., MILNE, A. C., SCHWARZ, D. W. F. & FREDRICKSON, J. M. (1975). Vestibular–neck integration in the vestibular nuclei. *Brain Research* **96**, 99–102.

SAKATA, H. (1975). Somatic sensory responses of neurons in the parietal association area (area 5) of monkeys. In *The Somatosensory System*, ed. KORNHUBER, H. H., pp. 250–261. Thieme, Stuttgart.

SCHWARZ, D. W. F. & FREDRICKSON, J. M. (1971). Rhesus monkey vestibular cortex: a bimodal primary projection field. *Science* **172**, 280–281.

TOMLINSON, R. D. & ROBINSON, D. A. (1984). Signals in vestibular nucleus mediating vertical eye movements in the monkey. *Journal of Neurophysiology* **51**, 1121–1136.

WAESPE, W. & HENN, V. (1977*a*). Neuronal activity in the vestibular nuclei of the alert monkey during vestibular and optokinetic stimulation. *Experimental Brain Research* **27**, 523–538.

WAESPE, W. & HENN, V. (1977*b*). Vestibular nuclei activity during optokinetic after-nystagmus (OKAN) in the alert monkey. *Experimental Brain Research* **30**, 323–330.

WAESPE, W. & HENN, V. (1978). Conflicting visual–vestibular stimulation and vestibular nucleus activity in alert monkeys. *Experimental Brain Research* **33**, 203–211.

ZACHARIAS, G. & YOUNG, L. R. (1981). Influence of combined visual and vestibular cues on human perception and control of horizontal rotation. *Experimental Brain Research* **41**, 159–171.