Exp Brain Res (1985) 61: 94–108

Experimental Brain Research

Canal-neck interaction in vestibular neurons of the cat's cerebral cortex

T. Mergner, W. Becker, and L. Deecke

Abteilung Neurologie und Sektion Neurophysiologie, Universität Ulm, Steinhövelstr. 9, D–7900 Ulm, Federal Republic of Germany

Summary. Interaction of semicircular canal and neck proprioceptive inputs was studied in the cerebral cortex of awake, intact cats. Neuronal responses were recorded extracellularly in the anterior suprasylvian gyrus of the left hemisphere. Stimulations consisted of horizontal rotations in the dark applied as sinusoids or position ramps. There were three stimulus conditions: (1) Pure canal stimulation; rotation of whole body. (2) Pure neck stimulation; rotation of trunk about stationary head. (3) Canal-neck interaction; rotation of head about stationary trunk.

(1) We recorded 105 neurons with either Type I or Type II canal response. These showed often pronounced non-linearities such as a clear firing increase upon rotation in the "on-direction" and hardly any decrease in the opposite direction. The responses reflected mostly angular velocity, but angular position signals were also obtained. (2) In 79 neurons, either Type I or Type II neck responses were obtained. They coded either angular velocity, velocity plus position, or position. (3) Canal-neck convergence was found in 67 of 88 neurons tested. In the majority of neurons, interaction was "*antagonistic*" in the sense that the canal and neck responses tended to cancel each other during rotation of the head about the stationary trunk. These neurons could signal *trunk rotation in space* rather than head in space or head relative to trunk. Most of the remaining neurons showed a "*synergistic*" interaction such that the response upon head rotation was enhanced as compared to whole body or trunk rotation. These neurons might be involved in the dual task of monitoring *head rotation in space and relative to trunk*. Interaction was compatible with linear summation of canal and neck inputs in 70% of the neurons. In part of these, however, the assumption had to be made that the interaction had taken place already at some stage prior to the cortical neurons investigated. The response characteristics of cortical canal neurons are discussed in comparison to vestibular nuclear neurons. Furthermore, parallels are drawn between the observed canal-neck interactions in the cortical neurons and (i) interactions of canal and neck dependent postural reflexes in the decerebrate cat, and (ii) interactions of canal and neck induced turning sensations in man.

Key words: Cerebral cortex – Canal-neck interaction – Cat

Offprint requests to: Dr. T. Mergner (address see above)

Introduction

It is by now well established that there exists a cortical vestibular representation in the parietal lobe of cat. First described by Walzl and Mountcastle (1949), this representation has been confirmed by a number of authors who elicited field potentials or single neuron responses in the anterior suprasylvian gyrus (ASSG) or adjacent regions by electrically stimulating the vestibular nerve or its branches (Kempinsky 1951; Mickle and Ades 1952; Andersson and Gernandt 1954; Milojevic and St. Laurent 1966; Landgren et al. 1967; Sans et al. 1970; Copack et al. 1972; Roucoux-Hanus and Boisacq-Schepens 1974; Mills and Taylor 1974), or the vestibular nuclei (Massopust and Daigle 1960; Spiegel et al. 1965; Watanabe et al. 1975).

A functional characterization of the neuronal responses in this field was first attempted by Kornhuber and da Fonseca (1964) who used caloric and galvanic stimulations. Recently, such studies also have been performed using natural vestibular stimulation (Becker et al. 1979; Deecke et al. 1979; Sestokas 1980). A finding in several of the above

studies was that the field does not only receive vestibular input but also somatosensory, visual and acoustic inputs, suggesting that it constitutes a complex kinesthetic rather than a pure vestibular center.

Functionally, the convergence between vestibular and neck proprioceptive inputs deserves particular interest. Interaction of these two inputs is known to be involved in postural stabilisation (cf. Kornhuber 1966; Roberts 1973). Also, from psychophysical studies on human turning sensations (Mergner et al. 1983) it appears that the vestibular message about head rotation in space is complemented by the neck proprioceptive message about head-to-trunk excursion, allowing separate evaluations of head and of trunk rotations in space, respectively. In our preliminary study on ASSG neurons of the cat (Becker et al. 1979), we were indeed able to demonstrate convergence of horizontal canal and neck signals and to define two different modes of interaction during passive rotation of the head on the stationary trunk. Unfortunately, in the course of the experiments it became evident that the responses of ASSG neurons exhibited pronounced non-linearities. The conventional approach of characterizing vestibular functions by their frequency responses upon sinusoidal stimulation as used in our study had, therefore, in retrospect to be considered inadequate. In particular, the exclusive use of sinusoidal stimulation made it difficult to compare the responses evoked by pure vestibular and pure neck stimulation with those evoked by simultaneous stimulation of both inputs, since in many cases the superposition law was not applicable. Thus, in order to elaborate the previous findings and to improve functional understanding of the cortical vestibular field and its role in integrating canal and neck information, position ramps of short duration were employed in addition to sinusoidal oscillations. The present approach allowed us, indeed, to corroborate two basic mechanisms of canal and neck interaction during head rotation, which are (1) mutual enhancement and (2) mutual suppression. A preliminary report of these data has already been published (Mergner et al. 1981a).

Methods

Animal preparation

The experiments were performed in six adult cats, which initially were prepared for chronic microelectrode recordings under deep halothane anesthesia. The following interventions were performed: (a) The skull over the left ASS sulcus was trephined, and a stainless steel recording chamber was implanted using screws and dental acrylic. (b) By similar means, a bolt fitting to a chronic head holder system was fixed over the sagittal suture. (c) Two Ag/AgCl screws were twisted into the skull for later recordings of the EEG. (d) In order to allow electrical stimulation of the labyrinth on the right side, the tympanic bulla was opened, two AG/AgCl ball electrodes were placed on the round window and on the adjacent bone and fixed by dental acrylic; the leads of these electrodes were threaded under the skin to a connection plug fixed on the skull. (e) A flexible catheter for later intravenous injections was permanently inserted in the right jugular vein, its end also being fixed onto the skull. – Finally, all wounds were carefully sutured, and still exposed parts on the skull were covered with dental cement. The animals were allowed at least one week to recover before recordings started.

Stimulus procedures

The animals were placed on a foam rubber cushion inside a restraining box, the latter being attached to a turntable. Their heads projected out of the box and were coupled, in a 20 deg nose down position, to a gear mounted on top of the box. By means of this gear the animal's head could be horizontally rotated with respect to the trunk. Both head gear and turntable were driven by separate servomotors. The turning axes of the two devices were colinear and passed through the dorsoventral axis of the animal's first cervical vertebra (C1). This set up allowed the application of the following stimuli: (1) Rotation of the whole body with the head fixed relative to the trunk in straight position, providing stimulation of only the horizontal canals (L; turntable rotating, head gear fixed to turntable). (2) Rotation of only the trunk, providing pure neck stimulation (N; turntable rotating, head gear coupled to an earth fixed bar). (3) Rotation of only the head, thereby stimulating both the canal and the neck system (turntable stationary, head gear rotating). Henceforth, a rotation will be defined to be of positive direction if it leads to activation of the receptors ipsilateral to the recording site. For canal receptors, this is ipsilateral rotation of the head in space, for neck receptors it is stretch of the ipsilateral neck, i.e. contralateral rotation of head relative to the trunk (cf. Mergner et al. 1982). During rotation of only the head, activation of the ipsilateral canal receptors is associated with inactivation of ipsilateral neck receptors and vice versa. The corresponding stimulus combination will, therefore, be denoted L,-N (cf. Fig. 5C).

Two different stimulus wave forms were used: (a) Sinusoids with frequencies of 1.0, 0.2 and 0.05 Hz. The peak angular velocities used at these frequencies ranged from 5 to 20, 5 to 80, and 5 to 20 deg/s, respectively (L-stimuli) and from 5 to 10, 5 to 40, and 5 to 10 for the N and the L,-N stimuli (maximum possible excursion between head gear and turntable was limited to ±32 deg). (b) "Position ramps", i.e. back and forth ramps of angular position (e.g., Fig. 1B–E). They were symmetrical about the primary position and had a total amplitude of 40 deg, duration of 1.8 s, and peak angular velocity of 35 deg/s.

The ball electrode on the round window was used for electrical stimulation of the labyrinth. During the initial surgical interventions while the animal was still under anesthesia, single electric shocks (duration: 0.2 ms) were used in order to elicite vestibular field potentials in the ASSG. This was of considerable help in localizing the rather tiny and variable vestibular spot in the ASSG. Characteristically, a negative potential was recorded by the microelectrode in the ASS gyrus or the anterior wall of the ASS sulcus having a minimum latency of 3.5 ms. Neuronal spike activity often was superimposed on the field potentials. However, the neurons usually did not show spontaneous activity and did not give consistent responses upon natural canal stimulation (cf. below). In the later chronic experiments, the electrode was also used to apply labyrinthine polarisation as an arousal stimulus. This was necessitated by the strong tendency of the cats to fall asleep during the monotonous rotations. The stimulus consisted of a ramp-like negative current increase within 1.5 s up to the 1.5–2-fold of the

threshold (30–70 μA) at which oblique head movements were initiated in the awake and unrestrained cat. The animal also showed a transient widening of the pupils, but no signs of aversive (e.g. escape) reaction.

Visual (optokinetic) stimulation was excluded by placing a black mask over the eyes. Acoustic cues were only partially excluded by the dental acrylic in the right tympanic cavity. Also, care was taken to assure that the neurons responses did not arise from tactile stimulation.

Data acquisition and analysis

For neuron recordings, an x–y platform carrying a lightweight (100 g) microdrive was fixed on the recording chamber. A glass coated tungsten microelectrode (impedance: 0.5–1.5 MΩ) was lowered perpendicularly through the intact dura into the cortex. Extracellular neuron activity was conventionally recorded, displayed on an oscilloscope, and made audible by an audiomonitor. When sufficiently isolated, spike potentials of single neurons were converted into standard pulses, which also were displayed on the oscilloscope. The pulses, together with the turntable or head gear positions, were fed into a PDP-12 computer, which constructed event-related average pulse density histograms of the neuronal activity. With sinusoidal stimulation, a single sweep of the computer extended over two cycles of the stimulus. At 1 Hz, a total of 16 sweeps were averaged, the bin width being 10 ms; the corresponding figures for 0.2 and 0.05 Hz were 8 (50 ms) and 4 (200 ms), respectively. Trapezoidal responses were averaged from 8 repetitions of a single cycle with a bin width of 150 ms. The histograms were stored on digital tape for later analysis.

The analysis provided the following parameters: (1) The neurons' spontaneous rates (imp./s.) with the turntable at rest and the head in the primary (straight-to-trunk) position. (2) The "phase" of the sinusoidal response, which was calculated as the phase difference between the fundamental contained in the response as obtained by Fourier analysis and the angular velocity of the stimulus. (3) The sensitivity to the sinusoidal stimuli ("gain" of the response; imp./s per deg/s), calculated as the ratio between the neurons' peak firing increase above resting rate and peak angular velocity of rotation in the "on"-direction. Note, that this measure does not contain the neurons' sensitivity for rotations in the "off"-direction, which usually was low and tended to saturate rapidly (cf. Results).

Recording conditions

At the onset of this investigation, the animals were immobilized during the recording sessions by packing their trunks into a small cotton bag and fixing their heads to the head holder system. With this procedure it was, in fact, possible to record reliable canal responses. However, responses to neck or combined canal and neck stimulation usually were inconsistent, as the animals actively counteracted the imposed stimuli by contracting the neck musculature to a variable degree. Several attempts to overcome this problem by rewarding the animals for passiveness during head or trunk rotation failed. Only in one cat could a few reliable responses (from 9 neurons) be obtained with this procedure. In the remaining five cats, we therefore tried to suppress the animals' active interference by administering Dihydrobenzperidol (Janssen; 0.3–1 mg/kg) and Diazepam (Valium, Roche; 0.2 mg/kg). However, if the dosage was high enough to sufficiently reduce neck muscle activity, it also affected the canal responses of the cortical neurons, which became inconsistent or disappeared altogether. In an attempt to lower the dosage of these drugs we combined them with a muscle relaxant (Pancuronium Organon; animals artificially respirated). However, as in a study of Mills and Taylor (1974), who tried low dosage barbital, alpha-chloralose or urethane anesthesia, there were no reproducible responses upon natural canal stimulation, although electrical pulses delivered to the labyrinth still were effective in eliciting vestibular field potentials and neuronal spikes. Remarkably, in neighbouring cortical regions (acoustic field AI, somatosensory field SII, whisker representation in SIII), neuronal activity still could be evoked by adequate natural stimulation under this condition. This particular sensitivity to narcotics of the vestibular neurons in the ASS cortex is in line with the profound effect anesthetics have upon other vestibular sites in the central nervous system (e.g. Buettner et al. 1978; Morzorati and Barnes 1977).

In order to obtain consistent responses under identical conditions for both the canal and the neck systems, we therefore had to resort to an awake, paralyzed and artificially respirated preparation. It is known from self-observations in humans that visual and somatosensory stimuli may well be perceived under muscle relaxation (Unna et al. 1950; Stevens et al. 1976). In animal experiments, quantitative data on vestibular responses of cortical neurons may also be obtained under these conditions (e.g. Tomko et al. 1981). It is to note that we observed in our experiments no overt difference between the responses obtained in the non-paralyzed state and those in the paralyzed state.

In the attempt to avoid distress to the animals, we followed the generally-accepted standards for this kind of preparation (cf. Werner and Whitsel 1968). Under initial ketamine anesthesia (Ketanest, Parke-Davis; 35 mg/kg as single dose i.v.) the animals were intubated with a flexible tracheal tube for babies, coated with a local anesthetic (Meaverin Gel, Woelm). A non-polarizing muscle relaxant (Pancuronium Organon; 0.35 mg/kg i.v.) was then administered every 20 min, and the animals were artificially respirated. The following parameters were observed throughout the experiments: (1) endtidal CO_2, (2) ECG, (3) EEG, (4) rectal temperature, and (5) pupillary size. Awakening from anesthesia was assumed if the EEG showed desynchronisation, if the pupils reacted promptly to illumination or to objects approaching the eyes, and if tactile stimuli were followed by short lasting mydriasis and increase in heart rate. Constantly widened pupils and elevated heart rate, as may possibly result from distress or pain of the animal, were usually not observed, but if so, the experiment was terminated.

Neuron recording started about 30 min after the animal awoke from anesthesia. Consistent responses to canal and neck stimulation could be evoked as long as the animals stayed awake. However, after 1 h or so, the responses often declined in amplitude and became irregular. This was generally accompanied by a synchronisation of the EEG, a narrowing of the pupils, and a decrease in heart rate. These signs were indicative of drowsiness or even sleep, much the same as had been observed in the non-paralysed animal. Polarisation of the labyrinth was used to reawaken the animal and restore consistent responses; such stimulation produced no aversive effects in the non-paralysed animal (cf. above). Recording sessions lasted between 4 and 10 h. One cat was exclusively used in the nonparalysed state. Two of the remaining 5 cats were used only once in the paralysed state and sacrificed immediately after the experiment. In order to keep the total number of animals to be sacrificed to a minimum, the other 3 cats were used in 4 sessions of short duration (4–6 h) with breaks of at least one week between sessions. In these cats, the session was terminated by anesthetizing the animal again with ketamine, clearing their respiratory tract with a suction tube, discontinuing relaxation, and awaiting spontanous breathing. When the animal's spontaneous breathing was reestablished, the intubation was removed, and the animal was put under a warming lamp. Upon awaking from anesthesia, the animals were returned to the colony, where they were cared for by the staff. Respiratory troubles or

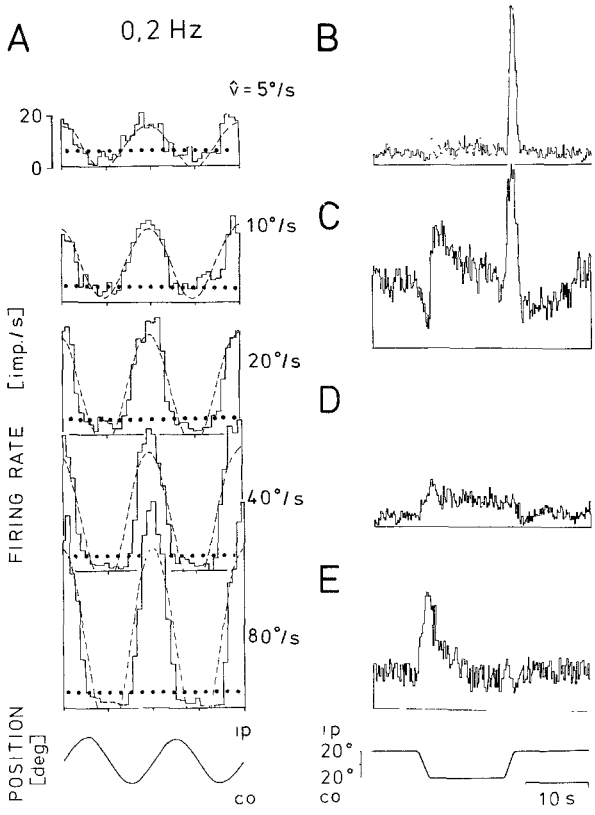

Fig. 1A–E. Neuronal responses in the ASSG upon horizontal canal stimulation **A** Type I neuron, sinusoidal stimulation. Stimulus frequency (0.2 Hz) kept constant, peak angular velocity ($\hat{v}$) varied from 5 to 80 deg/s. Averaged pulse density histograms of neuronal discharge rate, above. Position curve of turntable, below (ip, ipsilateral; co, contralateral). Dashed curves give fundamental component contained in response, horizontal dotted lines indicate spontaneous rate at rest. **B–E** Stimulation with position ramps. Different response patterns from four neurons (neuron in **B** same as in **A**)

pressure induced pareses as a consequence of the experiments were not observed, and the animals remained tame.

At the end of the final experiment, the animals were put under deep barbiturate anesthesia, and an especially successful electrode penetration was marked by electrolytic lesions (AC current of 7 kHz, 200 μA, and 1s duration). For histology, the animals were perfused and their brains fixated; serial sections were taken from the relevant piece of cortex and stained with cresyl-violet.

Results

Data base

In 48 penetrations, we recorded a total number of 117 neurons which we suspected to respond either to L- or to N-stimulation or to both. Twenty five of these neurons were tested with only the L-stimulus, and 4 with only the N-stimulus. Eighty eight neurons were tested with either stimulus; of these, 60 neurons also were investigated with the stimulus combination L,-N.

Table 1. Responses to the standard stimulus (sinusoid; f = 0.2 Hz, $\hat{v}$ = 10 deg/s). Population means ± S.D.

	Canal responses		Neck responses	
	Type I	Type II	Type I	Type II
No. (%)	33 (37)	56 (63)	19 (47)	21 (53)
Spont. Rate [a]	10.0(±6.3)	12.3(±9.0)	10.3(±5.9)	14.5(±5.9)
Sensitivity [b]	0.87(±0.71)	0.82(±0.48)	1.09(±0.87)	1.04(±0.74)
Phase re Vel. [c]	+4.4(±16.5)	−1.8(±19.8)	−6.4(±40.3)	−12.4(±29.8)

[a] [impulses/s] [b] [impulses/s per deg/s] [c] [deg]

Responses to canal stimulation

Of the 113 neurons tested with the canal stimulus, 105 (93%) showed a clear modulation. From 89 of these neurons the response to the standard *sinusoidal stimulus* (f=0.2 Hz, $\hat{v}$=10 deg/s) was obtained. Thirty three neurons (37%) increased their discharge rate upon rotation towards the recording side ("excitatory response" upon "ipsilateral rotation") and decreased it upon rotation in the opposite direction ("inhibitory response" upon "contralateral rotation"; example in Fig. 1A). They will be referred to as Type LI neurons by analogy to the terminology of Duensing and Schaefer (1958), "L" standing for labyrinthine response as opposed to neck response ("N"). The remaining 56 neurons had the opposite response pattern (Type LII). The response parameters of the two types of neurons were very similar (Table 1); the spontaneous rates of both populations were rather low, and peak discharge rate occurred almost in synchrony with peak excitatory velocity at 0.2 Hz, on the average. As will be noted from Fig. 2A, however, the phases of the individual neurons are scattered over a wide range. These data include the canal responses of nine neurons that were obtained in the non-paralysed cat, since no systematic difference of phase or sensitivity was noted as compared to those in the paralysed cats.

Sixteen Type LI and 18 Type LII neurons were also investigated at 0.05 Hz and at 1.0 Hz with a peak stimulus velocity of 10 deg/s. The individual frequency characteristics of phase and sensitivity (Bode diagrams) obtained are shown in Fig. 3A. On average, Type LI and Type LII neurons led stimulus velocity at 0.05 Hz by 21.4 deg and 20.1 deg, respectively. With increasing stimulus frequency, they decreased in phase, reaching, at 1 Hz, a lag of 6.4 deg and 22.4 deg. However, in view of the

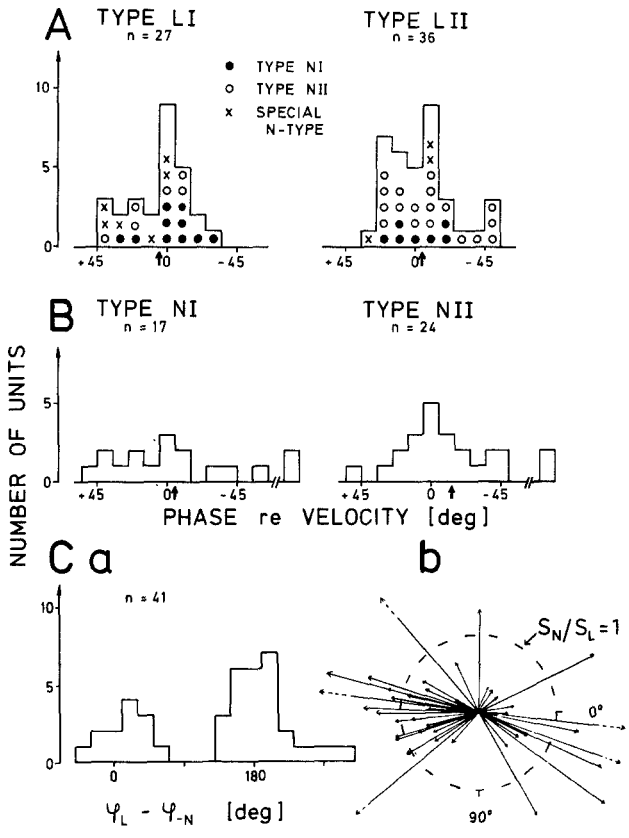

Fig. 2. **A** Phase histograms of ASSG neurons. Sinusoidal canal stimulation at 0.2 Hz. Mean values indicated by arrows. Neurons also responsive to neck stimulation specified by symbols. **B** Corresponding phase histograms of neck responses. **C** Phase differences between canal and neck responses ($\varphi L - \varphi N$); **a** frequency histogram, **b** polar plot. Note maxima in a at about 0 deg ("synergistic convergence") and at 180 deg ("antagonistic convergence"). In b, length of vectors gives sensitivity ratio between neck response and canal response (SN/SL)

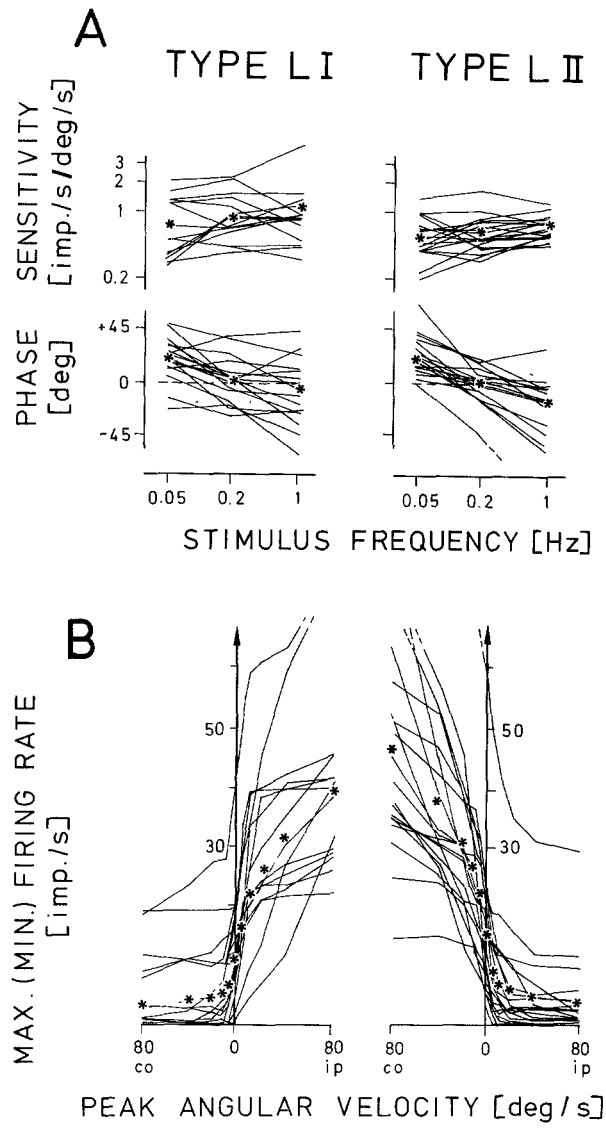

Fig. 3A and B. Characteristics of Type I and Type II canal responses to sinusoidal stimulation. **A** Frequency characteristics as Bode diagrams. Response parameters referred to stimulus velocity. **B** Amplitude characteristics. Stimulus frequency, 0.2 Hz. Maxima and minima of firing rate (ordinates) as function of peak angular velocity (abscissae) during rotation to ipsilateral and contralateral side (ip and co, respectively). Intersections of ordinate correspond to spontaneous rates ($\hat{v} = 0$ deg/s). Compare Fig. 1A. Mean values in **A** and **B** indicated by asterisks

considerable divergence between individual phase curves as well as between individual sensitivity curves, we thought it inappropriate to describe the neurons frequency characteristics by transfer functions fitted to the population averages.

In a number of neurons we were able to study the relationship between stimulus intensity and response magnitude in some detail. An example is given in Fig. 1A, where the stimulus frequency of 0.2 Hz was kept constant, and the peak angular velocity was varied from 5 to 80 deg/s. Figure 3B shows the family of amplitude characteristics that were obtained in a corresponding way for 14 Type LI and 20 Type LII neurons tested. Each curve consists of two branches, one showing the discharge maximum as a function of the peak stimulus velocity in the "on"-direction, the other the discharge minimum as a function of peak "off"-velocity. The point, where the two branches intersect the y-axis marks the resting rate. The discharge maxima of LII responses (right panel) tended to be somewhat larger than those of the LI responses (left panel), but otherwise the two populations behaved very similarly. The most striking feature of the amplitude characteristics is the marked asymmetry between maxima and minima. The minima of most neurons already reached, at 5 deg/s, a "saturation" level which frequently was different from zero. Increasing the velocity up to 80 deg/s did

not further decrease the minima of these neurons. Thus, in terms of the population average, there is no silencing even at high "off"-velocities. By contrast, the maxima of most neurons increased continuously along with the "on"-velocity exhibiting only a gradual saturation above 10 deg/s. Note, however, that in some neurons saturation was abrupt and also occured at low velocity for rotation in the on-direction. For the population as a whole, the increment of the discharge above resting level could be approximated by a power function of "on"-velocity with an exponent of 0.40 (Type LI) and of 0.56 (Type LII), respectively. Despite these striking saturation characteristics, the phases showed little change with increasing stimulus intensity; averaged phases at 0.2 Hz stimulus frequency showed a steady advance from about zero deg at $\hat{v} = 5$ deg/s to 12.3 deg (Type I) and to 6.9 deg (Type II) at $\hat{v} = 80$ deg/s.

Besides the amplitude asymmetry other factors such as skew caused the responses to deviate from perfect sinusoidal modulation. Particularly at 0.05 Hz stimulus frequency, skew was a frequent phenomenon (rising slope of modulation generally steeper than falling slope). When comparing the frequency response characteristics of these canal responses with those obtained in our previous study on cortical vestibular neurons (Becker et al. 1979), they resembled each other closely.

As can already be anticipated from the large scatter of the individual Bode diagrams, the *ramp stimulus* led to a variety of different response patterns. These patterns were grouped into three classes:

(1) *Phasic neurons* ("velocity neurons"; 28/70). Examples are shown in Figs. 1B, 5A, and 6A, B, D. The response upon the ramp stimulus consisted of a monophasic change in firing frequency, which can be considered a differentiation of the position profile of the stimulus. In all but three of these neurons the response was direction specific. Firing frequency increased upon rotation in one direction ("on-direction"), which was ipsilateral in 10 neurons and contralateral in 15 neurons. Upon rotation in the other direction, it either decreased slightly ("bidirectional response") in 50% of the neurons or remained essentially unchanged ("unidirectional response"). The responses to sinusoidal stimulation of these neurons showed a slight phase lead relative to stimulus velocity at 0.2 Hz. Nine of the neurons were also tested at 0.05 and 1.0 Hz; with decreasing frequency, there was a steady advance of phase and a decrease of sensitivity. The remaining three velocity neurons, which were not direction specific, were excited upon rotation in either direction (analogous to an asymmetric Type III canal response). Surprisingly, the responses to sinusoidal stimulation originally had suggested either a Type I or a Type II behavior. In two of these neurons, however, the amplitude of the response in one direction was only a fraction of that in the other direction. We could not exclude that the former stemmed from activation of vertical canals, since they also responded to rotation in "pitch" and in "roll" (note, that the animal's head was positioned slightly off the "null plane" of the vertical canals; cf. Methods). By contrast, the responses of the remaining neuron were of about equal amplitude for either direction of rotation, and the Type III behavior could not be attributed to activation of vertical canals.

(2) *Phasic neurons with secondary component* ("velocity neurons with protracted overshoot"; 25/70). These neurons showed a monophasic change in firing frequency during the ramp period as described above ("primary response"). Upon cessation of rotation, the "trailing edge" of the response overshot the resting level by as much as 50% of the primary response component and gave rise to an "after-effect" of opposite sign. The firing rate then slowly returned to resting level with a time constant of 5 s or longer. An example, where there was a clear phasic inhibitory response upon rotation in off-direction, is given in Fig. 1C. In general, however, such inhibitory responses were poor or even absent like in the pure velocity neurons. Yet, when the rotation in the off-direction stopped, there was a clear secondary response in the form of a sharp firing increase with subsequent slow decay (Fig. 5B). In case the decay time constant was long in comparison to the standstill period of the turntable, the secondary response constituted an almost tonic component. Responses to different frequencies were obtained in 10 of these neurons. In six cases, the phase and sensitivity curves resembled those of the pure velocity neurons. In the remaining four neurons, other forms of frequency curves were observed.

(3) *Tonic neurons* ("position neurons"; 3/70). These neurons maintained most of the firing increase (decrease) associated with rotations in the on-direction (off-direction) during the standstill period of the turntable in between the ramps (about 15 s; examples in Figs. 1D and 6C). However, slight fatigue of the animal caused these static responses to "degenerate" into an exponential decay. The static character of the responses returned when the animal was aroused. This fact was appreciated only in the course of the experiments, so that a number of tonic neurons are likely to have escaped out attention. In the responses

of these neurons to sinusoidal stimulation, the phase clearly lagged stimulus velocity.

The response patterns of a further 14 neurons did not fit into the above 3 classes. Occasionally the responses clearly outlasted the rotation periods (cf. Fig. 1E); conceivably, some of these cases may have been "degenerated" tonic responses. Other patterns contained only "delayed" responses, in which consistent changes of discharge frequency occured only after the end of the ramp. Five of these neurons showed a kind of Type III behaviour that consisted of excitatory responses upon rotation in either direction (in 3 of these cases, a contribution from the vertical canals was possible; cf. above). Three neurons showed no excitatory response at all, but only a transient decrease of firing rate upon rotation in one direction, a feature that had not been recognized with sinusoidal stimulation.

Responses to neck stimulation

Of the 92 neurons tested with the neck stimulus, 79 showed a clear response. In 42 of these neurons, the response to *sinusoidal stimulation* was evaluated. Nineteen neurons (47%) increased their firing rate upon ipsilateral rotation of the trunk about the stationary head, that led to a contralateral excursion of head relative to trunk and thus to a stretch of the neck on the ipsilateral side (example in Fig. 4A). These neurons will be referred to as Type NI (cf. Mergner et al. 1982). Stretch of the contralateral neck evoked excitatory responses in 21 neurons (53%; Type NII; Fig. 4B). Two neurons exhibited a Type NIII behaviour with excitatory responses to stretch on either side. The quantitative characteristics of the sinusoid responses are summarized in Table 1. It will be noted that, on the average, the N-responses lag peak stimulus velocity. The scatter of the individual phase values (Fig. 2B) is even larger than that of the L-responses.

In 17 neurons, the frequency responses were evaluated using 0.05 and 1 Hz in addition to the 0.2 Hz stimuli. The corresponding Bode diagrams formed a very heterogeneous population in which at least 3 different types could be discerned. Their details will be better understood if described with reference to the neurons' ramp response (cf. below). A complete examination of the amplitude characteristics (response magnitude vs. stimulus intensity) was obtained in only two neurons, and this only over a restricted range of stimulus velocities (up to $\hat{v} = 40$ deg/s), due to the mechanical limits of the trunk-to-head stimulation system. These amplitude characteristics were qualitatively similar to those shown in

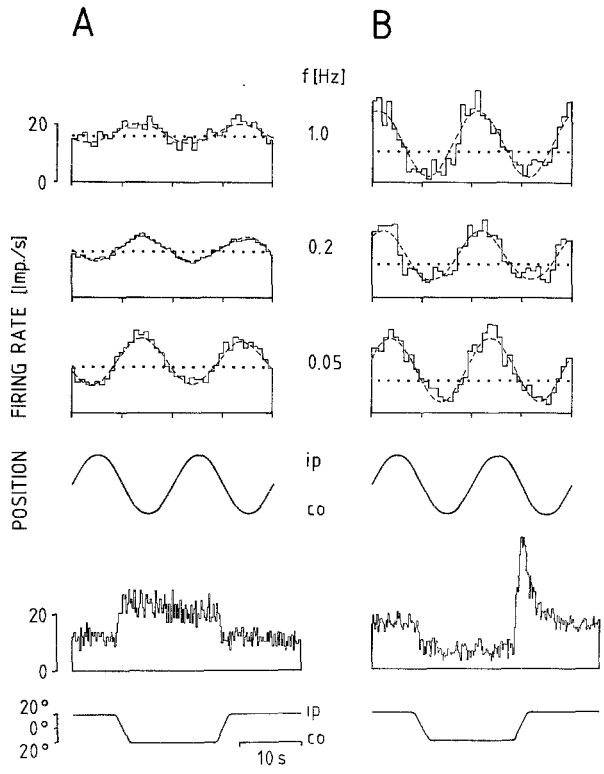

Fig. 4A and B. Responses to horizontal neck stimulation. Upper parts, sinusoidal stimulation, peak velocity 10 deg/s, frequencies as indicated. Lower parts, stimulation with position ramp. **A** Type NI neuron with tonic response. **B** Type NII neuron with phasic-tonic response. Position traces represent head-to-trunk deflection during rotation of trunk relative to stationary head. Otherwise as in Fig. 1

Fig. 3B for the L-responses. In particular, they suggested a clear-cut asymmetry with the decrease of firing rate during rotation in the off-direction being smaller and saturating earlier than the firing increase in the on-direction.

In a total of 61 neurons, we studied the neck responses by means of *ramp stimulation*. The result was a great variety of response patterns, which were again grouped into three different classes, using criteria similar to those for canal responses:

(1) *Phasic neurons* ("velocity neurons"; 24/61). These neurons exhibited a transient change of firing rate during the ramp period similar to the canal induced velocity responses (examples in Figs. 5A and 6C). Seven of these neurons had bidirectional responses (increase of firing rate upon rotation in one direction and some decrease in the other direction), while 12 neurons had unidirectional responses (11 cases with only a firing increase, one case only with a decrease). A Bode diagram was obtained for only one of these unidirectional neurons; the phase remained close to

zero (i.e. to stimulus velocity) at all frequencies tested, which is quite consistent with the pure velocity response evoked by the ramp. Finally, five neurons were excited upon rotation in either direction (example in Fig. 6D). It is noteworthy that in two of the latter neurons a Type NIII behavior had not been recognized from the responses to sinusoidal stimulation.

(2) *Phasic plus tonic neurons* ("velocity plus position neurons"; 30/61). The responses of these neurons can be considered a combination of one of the above velocity patterns with an additional position component. Again, only a few of them (6) had bidirectional velocity components, while the majority (20) showed a unidirectional velocity component for only one direction of head-to-trunk rotation (Figs. 4B, 5B, 6A, B). With respect to the directionality of the velocity and position components, two subgroups of neurons were distinguished:

(a) In 3 neurons, the velocity and position components had opposite on-directions. Consider the example in Fig. 6B: When rotating the trunk towards the contralateral side of the head, i.e. stretching the ipsilateral neck, the discharge of this neuron peaked. However, once the rotation was finished and the ipsilateral neck remained stretched, the discharge showed a tonic decrease below its prerotation level. Such response pattern would seem to result from a transfer function of the following structure:

$$r(s)/v(s) = -P/s + D \quad (I)$$

(r, discharge rate; v, stimulus velocity; P and D, gain of tonic and phasic components, respectively). The phase corresponding to this transfer function decrease along with increasing stimulus frequency from a 90 deg lead (re velocity) towards 0 deg. Bode plots were established in two of these neurons; in both cases the phase did indeed recede in a manner compatible with (I). The same frequency behaviour was also seen in two more neurons, although their trapezoid responses showed only faint position components. As a pattern, these responses resembled those canal responses, which contained in addition to the primary velocity response an almost tonic after-effect in the opposite direction.

(b) In the majority of the velocity plus position neurons, the two components had the same on-direction (example in Fig. 4B). For this response behaviour, the following transfer function can be formulated:

$$r(s)/v(s) = P/s + D \quad (II)$$

According to this function, the phase of the frequency response was expected to advance from a 90 deg phase lag to 0 deg as stimulus frequency increased. Seven of the Bode plots obtained from N-responses suggested a phase advance compatible with (II). Five of these neurons had also been tested with trapezoids, and all five belonged in fact to this group of velocity plus position neurons. There were three more neurons of this velocity plus position type, where the Bode plot did not show the expected phase advance along with frequency, but agreed with (II) at least in that the phase consistently lagged stimulus velocity.

(3) *Tonic neurons* ("position neurons"; 4/61). Their responses coded static trunk-to-head deflection (example in Fig. 4A). By contrast to the tonic L-responses, the tonic N-response did not degenerate much into an exponential decay pattern when the animal became drowsy, but maintained its tonic character during standstill of the platform (this held also for the tonic component of the velocity plus position neurons described under 2).

Three neurons did not match the above three categories and will be disregarded in the following considerations.

Convergences and interactions

Of the 88 neurons tested with both the canal and the neck stimuli, a total of 67 (76%) were responsive to both modalities. Responses to *sinusoidal stimulation* were obtained in 35 of these neurons. In order to characterize the different modes of convergence of the two inputs, we first considered their directionalities. It will be noted from Fig. 2A that the convergence patterns LI–NII and LII–NI were less frequent than the patterns LI–NI or LII–NII.

The patterns of convergence bear a functional significance. As an example, consider a Type LI–NI neuron during sinusoidal head rotation with the trunk stationary (stimulus L,-N). During ipsilateral head rotation, the canal input tends to increase the neuron's discharge rate, while the neck input (stretch of the contralateral neck; cf. above) tends to decrease it, and vice versa during contralateral rotation. A corresponding consideration holds also for Type LII–NII neurons. Patterns of this type of convergence, with reciprocal L- and N-inputs, therefore, are likely to be functionally "antagonistic". Conversely, the patterns of LI–NII and LII–NI convergence are regarded as "synergistic".

For a more precise characterisation of the convergences one has to compare the vector properties of the neurons' L- and N-responses. Figure 2Ca gives a frequency histogram of the phase differences between the two responses at 0.2 Hz. The fact that these differences are clustered around $\Delta\varphi=0$ and $\Delta\varphi=180$ deg supports the above notion of a subdivision into a synergistic ($\Delta\varphi=0$) and an antagonistic ($\Delta\varphi=180$ deg) mode of convergence. Figure 2Cb includes in addition to the phase difference also the sensitivity ratio between N- and L-response. As evident from this polar plot, the ratios vary considerably about unity (circle) without any obvious preference in either of the two populations. In this respect cortical vestibular neurons differ from vestibular nuclei neurons of the cat (Anastasopoulos and Mergner 1982), where synergistic convergences with a ratio of < 1 prevail.

The actual interactions of canal and neck inputs during sinusoidal head rotation were recorded in 35

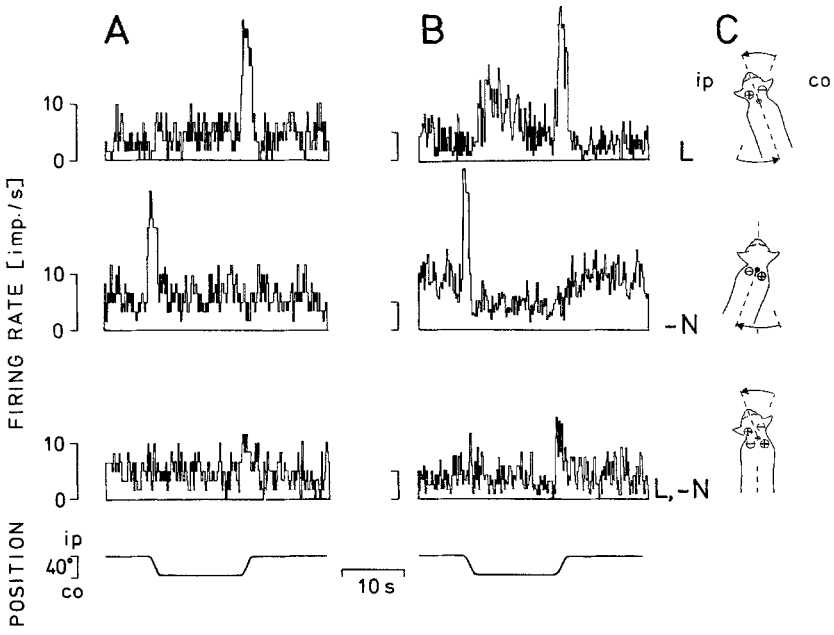

Fig. 5. A–C Responses upon canal and neck stimulations and upon canal-neck interaction obtained with position ramps. Examples of neurons with antagonistic convergence, where interaction leads to almost complete response cancellation. **C** Schematic presentation of stimulus conditions (cat from above; recording from left cerebral cortex). L, canal stimulus; upon rotation to left (counter-clockwise), ipsilateral canal receptors are activated (+) and contralateral receptors deactivated (–). -N, neck stimulation (rotation clockwise); receptor activation on contralateral and deactivation on ipsilateral side. L, -N, canal-neck interaction during head rotation; reciprocal activation/deactivation of canal and neck receptors

neurons. These "interaction responses" qualitatively confirmed the functional concept of synergism and antagonism of the two inputs. However, in view of the non-linear characteristics of most cortical vestibular neurons, we did not quantitatively compare experimental results to theoretical predictions (as can be constructed, e.g., by linearly superimposing L- and N-responses).

We felt it would be more instructive to compare the modes of convergence to actual interaction effects in terms of the responses to *ramp stimulation*. This also, because it allowed us to treat separately the interaction between phasic responses on one hand and between tonic N-component and secondary L-response, if present, on the other.

Convergence and interaction of phasic ("velocity") responses was compared in 46 neurons. In 39 of these, the interaction response was compatible with a summation of L- and N-responses. A schematic summary is given in Fig. 7a–e. The scheme distinguishes five different classes:

(a) *Antagonism.* This was the most common case (28/46). The phasic responses of these neurons showed either a Type LI–NI or a Type LII–NII convergence, which we considered antagonistic with respect to the interaction condition (head rotation; L,-N). In fact, the phasic response upon head rotation in on-direction was always smaller than either that upon the L- or the N-stimulus. In 5 neurons, the excitatory responses were cancelled almost completely. Quite surprisingly, such an extinction could be seen even in neurons with no overt inhibitory response. Consider the example in Fig. 5A. There is no inhibitory response to the L-stimulus, which could account for the disappearence of the excitatory response to the N-stimulus, and vice versa. As will be discussed below, the interaction response can, nevertheless, be interpreted as resulting from summation. In 19 neurons, extinction was not complete. In 11 of theses, the excitatory responses to both, the L- and the N-stimulus survived, though clearly reduced in amplitude, giving rise to a weak interaction response of Type III appearance. In the other 4 neurons, similarly, one of the two inputs survived (note clearly reduced phasic N-component in the interaction responses of Figs. 5B and 6A), while the other response was cancelled. Finally, in 4 neurons with antagonistic convergence the interaction was not compatible with summation.

(b) *Synergism.* Eight neurons had a Type LI–NII or a Type LII–NI convergence, which was considered synergistic. Upon interaction, the excitatory responses of 6 neurons were enhanced as by summation of the individual responses. Inhibitory responses, if present at all, remained poor. The other 2 neurons with such synergistic convergence had more complex interaction patterns.

(c) Synergism in a more general sense comprised also 6 more neurons, where at least one, the L- or the N-response, had a Type III appearance, and where none of them had a clear inhibitory component. In five of these, the interaction response again had a Type III character, one of the discharge peaks being enlarged by summation of two coincident excitatory responses (example in Fig. 6D).

(d) Synergism and antagonism, depending on the direction of rotation. In 2 neurons, one of the inputs evoked a Type III response, while the other yielded a

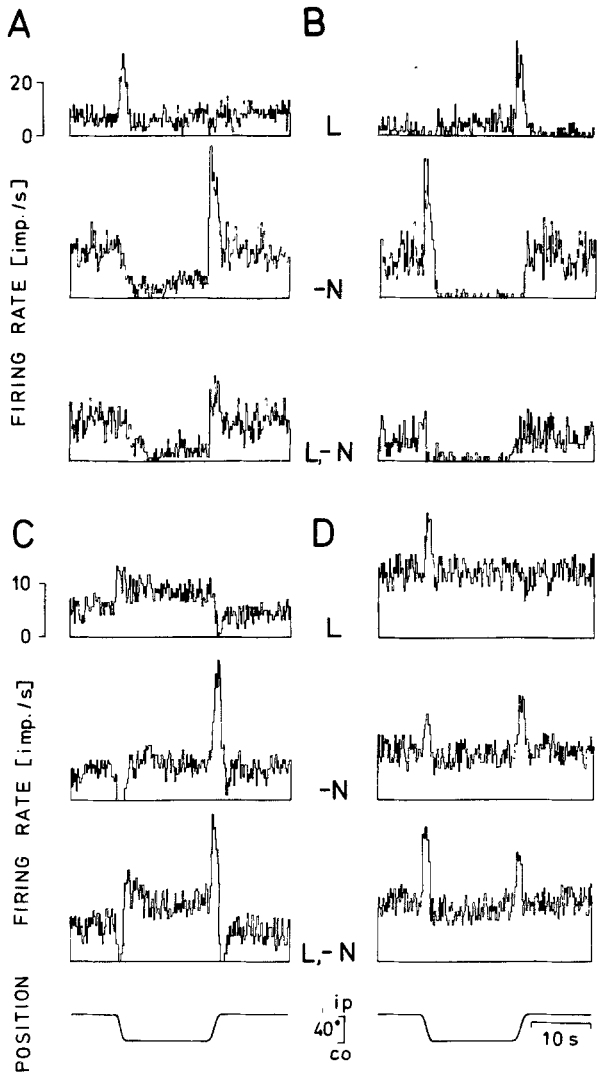

Fig. 6A–D. Examples of canal, neck, and interaction responses from four ASSG neurons. **A, B** Antagonistic interaction of phasic canal and neck responses. Tonic components of neck responses (in **A**, same on-direction as phasic response; in **B**, opposite on-direction) are maintained during head rotation. **C** Tonic canal response and phasic neck response appear essentially unaltered in interaction response. **D** Synergistic interaction of Type II canal response and Type III neck response. Phasic interaction response upon contralateral head rotation is enhanced, while response upon ipsilateral rotation appears to reflect neck input alone

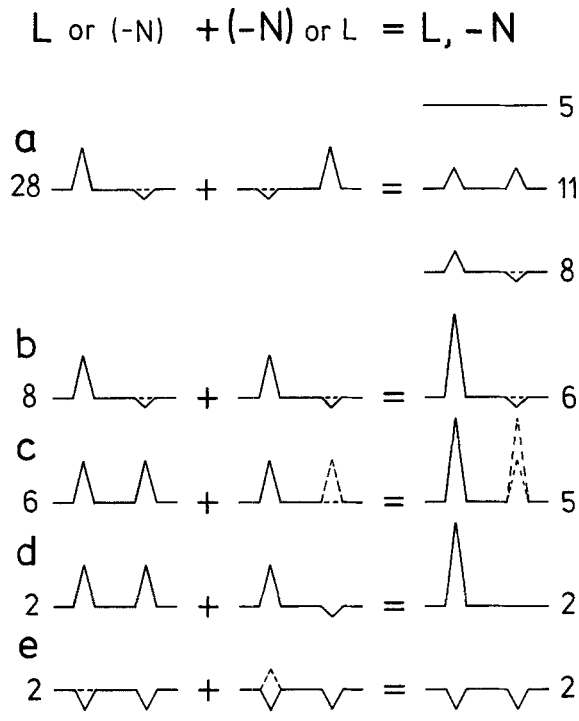

Fig. 7a–e. Schematic summary of most common canal-neck convergences and interactions. Considered are only phasic responses upon ramp stimulation. Figures on left give number of neurons per class of convergence, figures on right the number of those cases, where interaction was interpreted as resulting from linear summation. Response variations indicated by dashed lines. For details, cf. text

Type I or Type II response with clear excitatory and inhibitory components.

(e) Synergism of inhibitory responses. In 2 neurons, inhibitory responses prevailed. Interaction led to firing decreases upon head rotation in either direction (Type IV appearance).

In 25 of the 46 neurons, either the N- or the L-response contained a tonic or secondary component, respectively. This component was maintained essentially unaltered in the interaction response (cf. Figs. 6A, B and C). In 4 neurons, a secondary component in the L-response coincided with a tonic component in the N-response. The interaction responses had a tonic component approximately equal to the sum of the former two. An example is given in Fig. 5B, where these two components have about the same amplitude, but opposite on-directions; they cancel each other almost completely. Two of the neurons, including the one depicted in Fig. 5B, showed subtraction of their tonic components, the two others showed addition.

It should finally be mentioned that neurons with the same pattern of convergence were occasionally encountered in close succession within the same electrode tract. Except for such local patches no consistent topography of convergence patterns could be detected within the area investigated. Histology revealed that recordings were made from two rather than from one region. One of these comprised the crest of the ASSG just at the tip of the posterior branch of the ansate sulcus. The other was located more deeply in the anterior wall of the ASS. It was the latter region where all neurons with canal-neck convergence apart two were encountered.

Discussion

Canal responses

The response properties of ASSG neurons differ from those of vestibular nuclear (VN) neurons (as previously found in the cat using a similar experimental approach; Anastasopoulos and Mergner 1982) in that they accentuate a number of phenomena that are already present in the VN:

(1) *Susceptibility to narcotics.* Most VN neurons give reliable responses upon canal stimulation under light ketamine anaesthesia. The cortical neurons did not; they gave consistent responses only if the animal was fully awake and attentive (cf. Methods). This raises the question whether the single ketamine administration used to prepare the animals for intubation at the outset of the experiments had a residual effect on the responses obtained during the subsequent recording session. Apparently this was not the case; by the time recording began, the responses were most consistent, and they were qualitatively not different from those obtained in the non-paralysed animal. Furthermore, their frequency characteristics resembled closely what we have observed in a previous study on cortical vestibular neurons (Becker et al. 1979) using a similar approach, but administering halothane-nitrous oxide instead of ketamine for the initial anesthesia.

(2) *Spontaneous rate.* Mean rate of the cortical neurons was less than half as compared to VN neurons, where it averaged 25 imp./s (for an even higher rate in the awake cat, cf. Keller and Precht 1979). It is still higher in primary horizontal canal afferents ($\bar{x}$ = 59 imp./s; Ezure et al. 1978). Thus, the spontaneous rate appears to decline steadily from the periphery via the brain stem to the cortex. A similar reduction has been observed in the rhesus monkey, where the mean rate declines from 102 imp./s in the horizontal canal nerve (Büttner and Waespe 1981) to 48 in VN (Buettner et al. 1978), to 10 in thalamic neurons (Büttner et al. 1977), and to 8 in cortical neurons (Büttner and Buettner 1978).

(3) *Direction specificity.* VN neurons code a given plane of rotation; the direction in this plane is represented by the sign of the discharge modulation it evokes. By contrast, cortical neurons respond more or less only upon rotation in the on-direction. Thus, at cortical level, it is the neurons' "address" that signals rotation in a given direction. Due to the occurrence of Type I and Type II neurons, both horizontal directions are represented in the cortical field. The lack of clear inhibitory responses is concomitant to but not a direct consequence of the decrease in spontaneous rate. We suggest that it is due to a "replacement" of the vestibular tone by a background activity of different origin. A formal description of this is given in Fig. 8. Note that the vestibular tone is lost on the way to the cortex along with the inhibitory response, while background activity from other sources is added.

(4) *Secondary component.* Almost half of the VN neurons show a prolonged overshoot in their canal responses, the amplitude being less than 20% of the primary response. Such components are found in a similar proportion of the cortical neurons. There, however, their amplitudes showed a relative increase amounting up to 50% and more of the primary response. This overshoot might reflect an adaptation-like process, as present already at peripheral level (Goldberg and Fernandez 1971). An additional explanation is the pronounced amplitude saturation of some cortical neurons, that might affect the primary response more than the secondary response, thus changing the amplitude ratio between these two. Interestingly, the Bode plot of overshooting neurons often suggested a band pass behaviour; the phase had a steep negative slope and the gain peaked at 0.2 Hz decreasing at the lower and the higher frequency (cf. Fig. 3A).

It remains open whether this phenomenon represents a functional benefit or an unavoidable side-effect of information processing on the way to, or in, the cortex. Adaptation, for instance, might help to reduce spontaneous baseline fluctuations, which otherwise could be mistaken as true rotations. If this was the case, the secondary response per se would represent an unwanted and possibly disturbing feature. It remains to be investigated, whether it is compensated for under more physiological conditions, i.e. when vision is allowed during rotation.

(5) *Sensitivity.* The sensitivity of cortical neurons, and concomitantly their signal-to-noise ratio, was considerably reduced as compared to that of VN neurons.

(6) *Position coding.* As appears from the few observed (and more often suspected) tonic responses upon canal stimulation, there are cortical neurons that are related to horizontal angular position in space. This would necessitate a "neural integrator" transforming the velocity signal supplied by the cupulae into a position signal, as postulated for the vestibulo-ocular reflex (cf. Robinson 1975). Neurons containing an eye position signal have been observed

in the VN of the awake cat (Keller and Precht 1979). However, we deem it unlikely that the observed cortical neurons deal with eye position; we never observed tonic shifts of firing as one would expect to occur during longer periods of rest in relation to the animal's attempt of eye position changes. It is at one's own discretion whether one considers the position signal merely a quantitative change by comparison to the VN (prolongation of time constant) or as a new quality introduced by the cortex. In similar vein, the other differences listed above may be interpreted as either quantitative or qualitative.

Neck responses

Similar differences between ASSG and VN neurons were observed for neck responses. This applies to the reduction of spontaneous rate and tone as well as to unidirectionality. Interestingly, there were no responses compatible with an integration (in the mathematical sense) of tonic neck responses; this is of course what one would expect from a functional point of view, since it is difficult to conceive how the integration of an already existing position signal could yield a meaningful information. Furthermore, we observed convergence of a tonic canal response with a dynamic neck response (Fig. 6C). Thus, if integration is present in one input, it is not necessarily present in the other. This suggests that there are cases where information processing is different in the two channels.

As noted above (Methods), the neurons' responses to neck stimulation are strongly modulated in the non-paralysed animal if it actively interferes with the imposed head rotation. In the course of a previous investigation (Mergner et al. 1982), we have observed a similar interference in neurons of the cat's medulla oblongata that relay neck proprioceptive information to the cerebellum (unpublished data). These observations raise the question, what the responses obtained under the imposed muscle relaxation can tell us about the neurons' activity in the naturally behaving cat. The data certainly do not allow definite predictions for active head movements. They may reflect, however, the neural discharge in situations where the animal's head and/or trunk is passively moved by sudden external forces.

Canal-neck interaction

Basically, two modes of interaction were observed in ASSG neurons, i.e. synergism and antagonism of phasic response components. These two modes

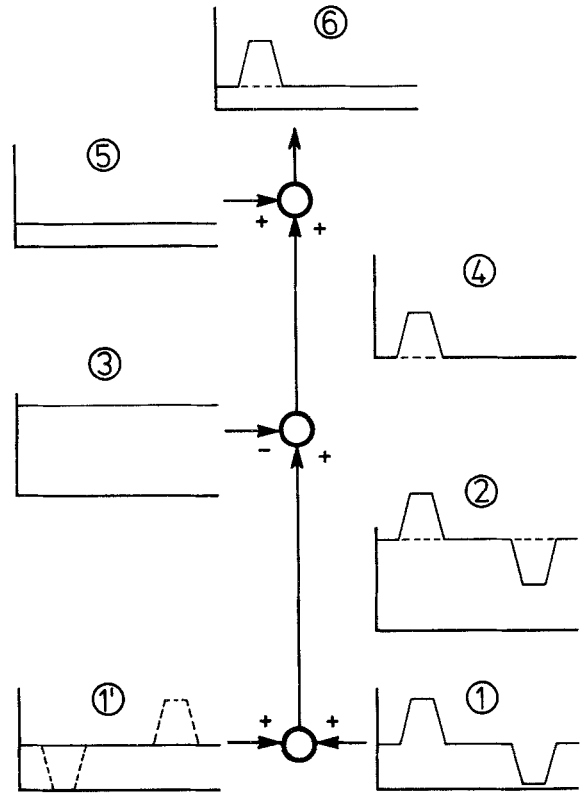

Fig. 8. Formal explanation of direction specificity and "hidden" summation. Input 1 (containing phasic on- and off-responses upon ramp stimulation) and input 1' (not activated) converge onto "subcortical neuron" 2. Subsequently, the background tone is lost (represented by subtraction of input 3), yielding truncation of off-response in "neuron" 4. Activity from other sources (input 5) is added, which gives the output at "cortical level" (6) some background activity. If input 1' is activated concommitantly, but in a reciprocal way to input 1, the cortical on-response is cancelled (dashed lines)

resemble closely what we have previously observed with horizontal rotation (Anastasopoulos and Mergner 1982) and what other have seen with vertical rotation (Boyle and Pompeiano 1981) in VN neurons. It could well be that the interaction observed in the cortical neurons actually is the result of a preprocessing in the VN. Consider for instance those neurons, where the excitatory response of one input was cancelled during interaction, although there was no corresponding inhibitory response in the other input (Figs. 5A, B and 6A, B). The above explanation for the direction specificity of the cortical neurons could also explain this phenomenon. In Fig. 8, the output (6) reflects only the excitatory response of input 1. If input 1' is activated concomitantly (dashed lines), its inhibitory response cancels the excitatory response of input 1 (and vice versa) and thus the output response. From the output during activation of either of the two inputs alone

one would not have suspected any inhibitory response. In the previous study, some VN neurons also showed a kind of "hidden" summation, although the inhibitory responses often could be brought to light if increasing the tonic activity of the neurons (e.g., by static neck deflection). There is anatomical evidence that neurons in caudal parts of the VN complex, where we previously studied canal-neck interaction, project to thalamic sites (posterior margins of ventro-basal complex), which in their turn project to the vestibular field in the ASSG (cf. Mergner et al. 1981b). Other subcortical structures might also take part in such a preprocessing.

In VN, the outcome of canal-neck interaction could reasonably well be attributed to linear summation of the two inputs for horizontal rotations (Anastasopoulos and Mergner 1982) as well as for vertical rotations (Boyle and Pompeiano 1981). This also was the case with almost all cortical neurons showing synergistic interaction of inhibitory responses and with several neurons showing antagonistic interaction. If we also take into account those neurons, where we assumed a "hidden" subtraction, linear summation could be assumed for 70% of all cortical neurons. Recently, Grüsser et al. (1982) reported preliminary results on neuronal responses upon canal, optokinetic, and neck stimulation in a retroinsular region of the parietal cortex of Java monkey. They observed neurons that showed an antagonistic convergence of canal and neck inputs, resulting in cancellation of the responses during head rotation. In their view, the result of interaction could best be described by the algebraic mean of the two inputs rather than by their sum. We feel, however, that their results, like ours, can be explained by linear summation of canal and neck inputs as long as the responses are equated to the stimulus-evoked modulation about resting rate.

What might be the functional role of the observed modes of interaction? To answer this question, one might compare the neuronal responses to the physical rather than the physiological stimuli. Canal stimulation was obtained by rotation of the body as a whole, i.e. of 'head in space' (HS) together with 'trunk in space' (TS). Neck stimulation was obtained by rotation of 'trunk relative to head' (TH); since the head remained stationary, it included a rotation of 'trunk in space' (TS). Interaction was obtained by rotation of 'head in space' (HS) and of 'head relative to trunk' (HT= –TH). Neurons with antagonistic convergence showing clear responses upon canal and upon neck stimulation but not upon interaction obviously reflect TS rather than HS or HT. Thus, these neurons would be apt to monitor horizontal trunk rotation in the dark, despite the fact that the receptor systems are located outside the trunk (canal receptors in the skull and proprioceptors in the neck). We cannot exclude, of course, that somatic and visceral receptors in the trunk are also activated by centrifugal forces and contribute, to some extent, to the cortical representation of trunk rotation. However, the main contributions to the neurons' responses certainly stemmed from canal and neck receptors, since (i) the neurons had their highest sensitivity at low stimulus intensities, and (ii) the neurons also responded to electrical stimuli applied to the labyrinth (cf. Methods) as well as to manually pressing the neck.

Synergistic neurons, on the other hand, apparently are related to HS *and* HT, and seem to "emphasize" particularly situations where the head rotates on a more or less stationary trunk.

An additive kind of interaction has been recently observed in the decerebrate cat for the vestibulocervical and the cervico-cervical reflexes evoked in the horizontal plane (Peterson et al. 1981a, 1981b). According to these findings, the effect of both reflexes add in the attempt to resist a passive head rotation versus the stationary trunk. Thus, there appears to exist a parallelism for synergistic canal-neck interaction between these postural reflexes and the observed responses in the cortical neurons.

We do not know of a similar study on the interaction of vestibulo-spinal and cervico-spinal reflexes on the limbs upon rotation in the horizontal plane. Yet, a subtractive interaction has been described for the effect of "roll" on cat's triceps muscle (Lindsay et al. 1976; Ezure and Wilson 1984). Subtractive interaction between vestibular and neck input has also been observed in neurons of the cat's cervical spinal cord, which are possibly involved in mediating these reflexes (Wilson et al. 1984). Such an antagonistic vestibular-neck interaction originally had been postulated for trunk stabilisation during head movements by von Holst and Mittelstaedt (1950), Kornhuber (1966), and Roberts (1973).

However, the postural reflexes observed in the decerebrate cat cannot be consistently evoked in the intact cat where we observed the neuronal effects. A widely accepted explanation for this difference between the intact and the decerebrate animal is that supramesencephalic structures modify or suppress the reflexes as a function of the animal's behavioral situation. It might well be that the observed neurons participate in this "intelligent control" of the postural reflexes. It is tempting to hypothesize that similar neurons exist in the human cerebral cortex, and that these neurons mediate perception of trunk and of head rotation. This hypotheses led us recently to study psychophysically the perception of horizontal

rotations using largely similar conditions of canal-neck interaction in man (Mergner et al. 1983). Interestingly, estimates of trunk turning in space could be described by linear subtraction of canal and of neck induced turning sensations, and those of head turning in space by addition.

References

Anastasopoulos D, Mergner T (1982) Canal-neck interaction in vestibular nuclear neurons of the cat. Exp Brain Res 46: 269–280

Andersson S, Gernandt BE (1954) Cortical projection of vestibular nerve in cat. Acta Oto-laryngol (Stockh) Suppl 116: 10–18

Becker W, Deecke L, Mergner T (1979) Neuronal responses to natural vestibular and neck stimulation in the anterior suprasylvian gyrus of the cat. Brain Res 165: 139–143

Boyle R, Pompeiano O (1981) Convergence and interaction of neck and macular vestibular inputs on vestibulospinal neurons. J Neurophysiol 45: 852–868

Büttner U, Buettner UW (1978) Parietal cortex (2v) neuronal activity in the alert monkey during natural vestibular and optokinetic stimulation. Brain Res 153: 392–397

Büttner U, Waespe W (1981) Vestibular nerve activity in the alert monkey during vestibular and optokinetic nystagmus. Exp Brain Res 41: 310–315

Büttner U, Henn V, Oswald HP (1977) Vestibular-related neuronal activity in the thalamus of the alert monkey during sinusoidal rotation in the dark. Exp Brain Res 30: 435–444

Buettner UW, Büttner U, Henn V (1978) Transfer characteristics of neurons in the vestibular nuclei of the alert monkey. J Neurophysiol 41: 1614–1628

Copack P, Dafny N, Gilman S (1972) Neurophysiological evidence of vestibular projections to thalamus, basal ganglia, and cerebral cortex. In: Frigyesi T, Rinvik E, Yahr MD (eds) Corticothalamic projections and sensorimotor acitivities. Raven, New York, pp 309–339

Deecke L, Becker W, Jürgens R, Mergner T (1979) Interaction of vestibular and somatosensory afferents for perception and postural control. Agressologie 20C: 179–184

Duensing F, Schaefer KP (1958) Die Aktivität einzelner Neurone im Bereich der Vestibulariskerne bei Horizontalbeschleunigungen unter besonderer Berücksichtigung des vestibulären Nystagmus. Arch Psychiat Nervenkr 198: 225–252

Ezure K, Wilson VJ (1984) Interaction of tonic neck and vestibular reflexes in the forelimb of the decerebrate cat. Exp Brain Res 54: 289–292

Ezure K, Schor RH, Yoshida K (1978) The response of horizontal semicircular canal afferents to sinusoidal rotation in the cat. Exp Brain Res 33: 27–39

Goldberg JM, Fernandez C (1971) Physiology of peripheral neurons innervating semicircular canals of the squirrel monkey. I. Resting discharge and response to constant angular accelerations. J Neurophysiol 34: 635–660

Grüsser O-J, Pause M, Schreiter U (1982) Neuronal responses in the parieto-insular vestibular cortex of alert Java monkeys (Maccaca fascicularis). In: Roucoux A, Crommelink M (eds) Physiological and pathological aspects of eye movements. Dr W Junk, The Hague Boston London, pp 251–270

von Holst E, Mittelstaedt H (1950) Das Reafferenzprinzip (Wechselwirkungen zwischen Zentralnervensystem und Peripherie). Naturwissenschaften 37: 464–476

Keller EL, Precht W (1979) Adaptive modification of central vestibular neurons in response to visual stimulation through reversing prisms. J Neurophysiol 42: 896–911

Kempinsky WH (1951) Cortical projection of vestibular and facial nerves in cat. J Neurophysiol 14: 203–210

Kornhuber HH (1966) Physiologie and Klinik des Zentralvestibulären Systems (Blick- und Stützmotorik) In: Berendes J, Link R, Zöllner F (eds) Hals-Nasen-Ohren-Heilkunde III/3. Thieme, Stuttgart, pp 2150–2351

Kornhuber HH, da Fonseca JS (1964) Optovestibular integration in the cats cortex: a study of sensory convergence on cortical neurons. In: Bender MB (ed) The oculomotor system. Hoeber, New York, pp 239–279

Landgren S, Silfvenius H, Wolsk D (1967) Vestibular, cochlear, and trigeminal projections to the cortex in the anterior suprasylvian sulcus of the cat. J Physiol (Lond) 191: 561–573

Lindsay KW, Roberts TDM, Rosenberg JR (1976) Asymmetric tonic labyrinthine reflexes and their interaction with neck reflexes in the decerebrate cat. J Physiol (Lond) 261: 583–601

Massopust LC, Daigle HJ (1960) Cortical projection of the medial and spinal vestibular nuclei in the cat. Exp Neurol 2: 179–185

Mergner T, Anastasopoulos D, Becker W, Deecke L (1981a) Comparison of the modes of interaction of labyrinthine and neck afferents in the suprasylvian cortex and vestibular nuclei of the cat. In: Fuchs A, Becker W (eds) Progress in oculomotor research. Elsevier/North-Holland, New York, pp 343–350

Mergner T, Deecke L, Wagner H-J (1981b) Vestibulo-thalamic projection to the anterior suprasylvian cortex of the cat. Exp Brain Res 44: 455–458

Mergner T, Anastasopoulos D, Becker W (1982) Neuronal responses to horizontal neck deflection in the group x region of the cat's medullary brainstem. Exp Brain Res 45: 196–206

Mergner T, Nardi GL, Becker W, Deecke L (1983) The role of canal-neck interaction for the perception of horizontal trunk and head rotation. Exp Brain Res 49: 198–208

Mickle WA, Ades HW (1952) A composite sensory projection area in the cerebral cortex of the cat. Am J Physiol 170: 682–689

Mills KR, Taylor A (1974) The projection of the vestibular nerve to the cerebral cortex in the cat. J Physiol (Lond) 239: 165–178

Milojevic B, St. Laurent J (1966) Cortical vestibular projection in the cat. Aerospace Med 37: 709–712

Morzorati L, Barnes CD (1977) Sensitivity of components of the central vestibular complex to pentobarbital. Neuropharmacology 16: 295–298

Peterson BW, Bilotto G, Fuller JH, Goldberg J, Leeman B (1981a) Interaction of vestibular and neck reflexes in the control of gaze. In: Fuchs AF, Becker W (eds) Progress in oculomotor research. Elsevier/North Holland, New York, pp 335–342

Peterson BW, Bilotto G, Fuller JH, Goldberg J, Wilson VJ (1981b) Dynamics of vestibulo-ocular, vestibulo-collic, and cervico-collic reflexes. Ann NY Acad Sci 374: 395–402

Roberts TDM (1973) Reflex balance. Nature 244: 156–158

Robinson DA (1975) Oculomotor control signals. In: Lennerstrand G, Bach-y-Rita P (eds) Basic mechanisms of ocular motility and their clinical implications. Pergamon, Oxford, pp: 337–374

Roucoux-Hanus M, Boisacq-Schepens N (1974) Projections vestibulaires au niveau des aires corticales suprasylvienne et postcruciée chez le Chat anesthesié au chloralose. Arch ital Biol 112: 60–76

Sans A, Raymond J, Marty R (1970) Résponses thalamiques et corticales à la stimulation èlectrique du nerf vestibulaire chez le Chat. Exp Brain Res 10: 265–275

Sestokas AK (1980) Response characteristics of vestibulo-cortical neurons to rotations of the cat in the dark and light. Soc Neurosci Abstr 6: 559

Spiegel EA, Szekely EG, Gildenberg PL (1965) Vestibular responses in midbrain, thalamus, and basal ganglia. Arch Neurol 12: 258–269

Stevens JK, Emerson RC, Gerstein GL, Kallos T, Neufeld GR, Nichols CW, Rosenquist AC (1976) Paralysis of the awake human: visual perception. Vision Res 16: 93–98

Tomko DL, Barbaro NM, Ali FN (1981) Effect of body tilt on receptive field orientation of simple visual cortical neurons in unanesthetized cats. Exp Brain Res 43: 309–314

Unna KR, Pelikan EW, Macfarlane DW, Cazort RJ, Sadove MS, Nelson JT (1950) Evaluation of curarizing agents in man. J Am Med Ass 144: 448–451

Walzl EM and Mountcastle VB (1949) Projection of vestibular nerve to cerebral cortex of the cat. Am J Physiol 159: 595

Watanabe S, Jijiwa H, Itoh K (1975) Responses of cortical vestibular center produced by stimulation of vestibular nuclei and visual association area in the cat. Acta Otolaryngol (Suppl) 330: 147–158

Werner G, Whitsel BL (1968) Topology of the body representation in somatosensory area I of primates. J Neurophysiol 31: 856–869

Wilson VJ, Ezure K, Timerick SJB (1984) Tonic neck reflex of the dererebrate cat: Response of spinal interneurons to natural stimulation of neck and vestibular receptors. J Neurophysiol 51: 567–577

Received December 17, 1984 / Accepted July 10, 1985