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RESEARCH ARTICLE

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Short-term changes in neck muscle and eye movement responses following unilateral vestibular neurectomy in the cat

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Abstract The purpose of this study was to investigate changes in neck muscle and eye movement responses during the early stages of vestibular compensation (first 3 weeks after unilateral vestibular neurectomy, UVN). Electromyographic (EMG) activity from antagonist neck extensor (splenius capitis) and flexor (longus capitis) muscles and eye movements were recorded during sinusoidal visual and/or otolith vertical linear stimulations in the 0.05–1 Hz frequency range (corresponding acceleration range 0.003–1.16 g) in the head-fixed alert cat. Preoperative EMG activity from the splenius and longus capitis muscles showed a pattern of alternate activation of the antagonist neck muscles in all the cats. After UVN, two motor strategies were observed. For three of the seven cats, the temporal activation of the individual neck muscles was the same as that recorded before UVN. For the other four cats, UVN resulted in a pattern of coactivation of the flexor and extensor neck muscles because of a phase change of the splenius capitis. In both subgroups, the response patterns of the antagonist neck muscles were consistent for each cat independently of the experimental conditions, throughout the 3 weeks of testing. Cats displaying alternate activation of antagonist neck muscles showed an enhanced gain of the visually induced neck responses, particularly in the high range of stimulus frequency, and a gain decrease in the otolith-induced neck responses at the lowest frequency (0.25 Hz) only. By contrast, for cats with neck muscle coactivation, the gain of the visually induced neck responses was basically unaffected relative to preoperative values, whereas otolith-induced neck responses were considerably decreased in the whole range of stimulation. As concerns oculomotor responses, results in the two subgroups of cats were similar. The optokinetic responses were not affected by the vestibular lesion. On the contrary, otolith-induced eye responses showed a gain reduction and a phase lead. Deficits and short-term changes after UVN of otolith- and semicircular canal-evoked collic and ocular responses are compared.

Key words Antagonist neck muscles · Eye movements · Unilateral vestibular neurectomy · Visual substitution · Motor strategies · Cat

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Introduction

Unilateral vestibular neurectomy (UVN) leads to strong motor deficits, particularly during the acute stage (first 3 weeks postlesion). This vestibular syndrome is characterized in most of the species by strong static and dynamic symptoms. The dynamic symptoms consist of the impairment of oculomotor and cephalic reflexes (see Curthoys and Halmagyi 1995; Dieringer 1995 for reviews). Previous investigations in the cat dealt mostly with the dynamic oculomotor symptoms evoked during rotations. Deterioration of the vestibulo-ocular reflex (VOR) consisted mainly of phase lead and gain decrease. VOR asymmetrical responses were observed for rotations in the horizontal plane; the VOR amplitude was bilaterally decreased and lower for rotations towards the lesioned side than those towards the intact side (Maioli et al. 1983). As far as neck muscle responses are concerned, strong dynamic changes were evidenced after hemilabyrinthectomy by Berthoz and Anderson (1971). They demonstrated that extensor neck muscles ipsilateral to the rotation showed a significant phase lead during sinusoidal rotation around the longitudinal axis. It is generally admitted that all these behavioral disorders correlate with the imbalance in the vestibular influences normally exerted bilaterally on neck and extraocular muscles by the vestibulospinal and vestibulo-ocular pathways, respectively (see Precht and Dieringer 1985).

Even though these deficits are not completely compensated before 4 or 5 weeks in most of the species, adaptive mechanisms begin very early after lesion (see Smith and

Curthoys 1989 for a review). The vestibular compensation processes involve either changes in the sensory modalities cooperating in a function or subsystems usually serving other behavioral purposes. Behavioral and electrophysiological studies on compensation after UVN have strongly suggested that potentiation of visual information efficiently compensated the impairment of the dynamic properties of the vestibular reflexes involved in eye and head (gaze) stabilization (Precht and Dieringer 1985; Lacour et al. 1989 for reviews). In addition, adaptive strategies substitute for defective oculomotor, canal-related responses. For example, the saccadic system supplemented the weak gain of the VOR in vestibular-defective patients (Berthoz 1985) and in hemilabyrinthectomized frogs (Dieringer 1987). To our knowledge, only Conway (1987) reported changes in the spatial properties of the forelimb muscle responses after unilateral vestibular lesion; that author showed abnormal postoperative patterns of labyrinth and neck reflexes.

All these data on vestibular compensation have been collected during vestibular stimulation activating the semicircular canal system. Little is known, however, about the adaptive mechanisms of the otolith system. Static vestibulocollic reflex responses were strongly impaired in acute UVN cats, and compensation remained incomplete in the compensated stage (Zennou-Azogui et al. 1993). We described the dynamic properties of the otolith-neck responses (Lacour et al. 1987) as well as the dynamics of both otolith and optokinetic reflexes and their functional coupling (Borel and Lacour 1992a) during linear motion in the normal cat. Consistent vestibulocollic (otolith-neck) reflexes were recorded during linear motion in the three space directions. The resultant pattern of neck muscle activation was consistent with the pattern of synaptic potentials produced by selective stimulation of the utricular and saccular nerves (Sasaki et al. 1991; Sato et al. 1994). Otolith- and visual-induced neck muscle activities were synergistic, with a marked predominance of the visual and otolith inputs for low- and high-frequency motion, respectively.

The present study was designed to analyze the modifications of the neck muscle and eye movement responses in the early postlesion stage after UVN in the head-fixed alert cat. We evaluated changes in the dynamic characteristics of these neck responses during visual and/or otolith vertical linear stimulations. We focused on the electromyographic (EMG) pattern of antagonist neck muscle responses, since these muscles provide further insight into the motor strategies compensating the lack of dynamic otolith inputs, and we examined the concomitant changes in the dynamic properties of the vertical eye movements. Some of these results have been published in a previous paper (Borel and Lacour 1992b).

Materials and methods

Experiments were performed on seven cats (3–4 kg) prepared for chronic investigations. Electromyographic (EMG) and electro-oculographic (EOG) recordings were done in the same animals before and after UVN. Postoperative testings were carried out during the early stages of vestibular compensation (day 4–18).

Surgical procedures

The cats were preanesthetized with ketamine dihydrochloride (20 mg/kg, i.m.). Surgical procedures were performed with the cats under general anesthesia (fluothane 2%) and under aseptic conditions. Principles of laboratory animal care and specific French recommendations from the Ministère de l'Agriculture et de la Pêche were fully followed.

Animal preparation

Polyunitary EMG activity of neck muscles was recorded by bipolar intramuscular electrodes chronically implanted in two pairs of antagonist muscles: the splenius capitis implicated in head extension and rotation, and the longus capitis, a ventral flexor muscle, acting at the occipital-atlantal joint level. Intramuscular bipolar electrodes made of Teflon-insulated silver wire (0.5 mm diameter) were stripped 2 or 3 mm at the tip, inserted, and sutured in each muscle. Inside each muscle the electrodes were separated by about 1 cm. Then, wires were tunneled subcutaneously to a connector on the head of the cat.

Vertical eye movements were recorded by Ag-AgCl EOG electrodes implanted above and below one eye in a vertical plane. Lastly, a metal implant was stereotaxically fixed onto the skull with dental acrylic anchored by stainless steel screws. Three bolts fixing the implant to the head-holder immobilized the head during the recording sessions.

Unilateral vestibular neurectomy

Vestibular lesion was monitored through a dissecting microscope. The surface tissues behind the pavilion ear were incised. An aperture was drilled at the middle ear level to access the oval and round windows. Further drilling between the windows exposed the VIIIth nerve in the internal auditory canal. Both the cochlear and vestibular nerve branches were transected posterior to Scarpa's ganglion. The missing bone was then replaced with sterile hemostatic sponge and the incision was closed. The eye deviation towards the lesioned side constituted an immediate clinical symptom attesting UVN.

Experimental procedures

The cat was placed on a platform with its head fixed to a holder locked onto a stereotaxic frame rigidly attached to the table (Fig. 1A). Its head was pitched 23° nose down to bring the saccular maculae in the vertical plane and the horizontal canals in the earth horizontal plane. The animal was kept in a hammock to minimize body and limb movements. EMG and EOG recordings were investigated during vertical linear motion of the cat and/or of its visual surround. The experiments were done in awake animals submitted to four types of vestibular (otolith) and/or visual (optokinetic) stimulations.

Otolith stimulation was elicited by sinusoidally moving the animal in total darkness, with a 290-mm peak-to-peak amplitude in the 0.05–1 Hz frequency range. The corresponding peak-to-peak acceleration amplitude ranged from 0.003 to 1.16 g (i.e. from 0.03 to 11.45 m/s^2). This experimental paradigm activates mainly the saccular organs.

Optokinetic stimulation was done by sinusoidally moving a pseudo-random visual pattern onto a translucent screen. The visual pattern was deflected by a mirror driven by a galvanometer system and back-projected onto the screen (1.5 m×1.5 m) located 0.5 m in front of the cat. Dynamic characteristics of the visual stimulation were similar to those used for otolith stimulation. Linear motion of the visual pattern on the screen was 290 mm peak-to-peak in the frequen-

cy range, which led to peak-to-peak velocity profiles from 10°/s (0.09 m/s) to 203°/s (1.80 m/s).

Congruent visual-otolith interaction (costimulation) was obtained by translating the animal in front of the earth-stationary visual surround. *Conflicting visual-otolith interaction* was investigated by moving the visual surround together with the cat, in phase and at the same velocity. This procedure was used to abolish the visual motion cues.

The cats were maintained aroused during the course of the experiments by making noises and giving them milk between trials. Each cat was tested before vestibular neurectomy (control data) and throughout the postoperative period (day 4–18).

Data analysis

EMG responses

The procedure for processing EMG signals has been fully described (Borel and Lacour 1992a). Polyunitary EMG activity was amplified (gain 5 K), band-pass-filtered (50–5000 Hz), full-wave rectified, and integrated (time constant 20 ms). The EMG signals from both pairs of neck muscles were fed into a Bull SPS 5 computer that provided sequential histograms calculated by superimposing data of 6–10 successive cycles divided into equal-time intervals (128 or 64 bins depending on stimulus frequency). The sequential histograms expressing the muscular amplitude (in millivolts), elaborated from the linear envelope of the global EMG full-wave rectified and averaged, were submitted to a fast Fourier transform (FFT). FFT gave amplitude and phase of the fundamental frequency and of the first 32 harmonics contained in the motor output. Harmonic distortion was determined as the ratio of the square root of the sum of the squares of the second to the fifth harmonics to amplitude of the fundamental frequency. When distortion exceeded 40%, EMG modulations were rejected except when such modulations were observed over more than two consecutive cycles of stimulation. This was found in some recordings from the longus capitis muscle, in the high range of vestibular stimulation, where EMGs were peak-shaped instead of nicely sinusoidally modulated. As a rule, mean EMG D.C. level increased with stimulus frequency. Taking into account this observation, we expressed EMG response gain in decibels relative to DC level as percentage modulation per meter per second, according to the following mathematical expression:

$$G = 20\log\left[\frac{A/(\text{D.C.})/100}{V}\right]$$

where A and V are half peak-to-peak amplitude of the motor response and peak stimulus velocity, respectively. Phase was defined as the difference (in degrees) between the fundamental component of peak stimulus velocity and the fundamental component of peak motor output. By convention, peak downward animal velocity and peak upward visual surround velocity were used as reference 0° during animal motion (otolith stimulation and visual-otolith interaction) and visual surround motion (optokinetic stimulation), respectively. Negative values indicated a phase lag of the EMG response with respect to peak stimulus velocity, whereas positive values reflected a phase lead.

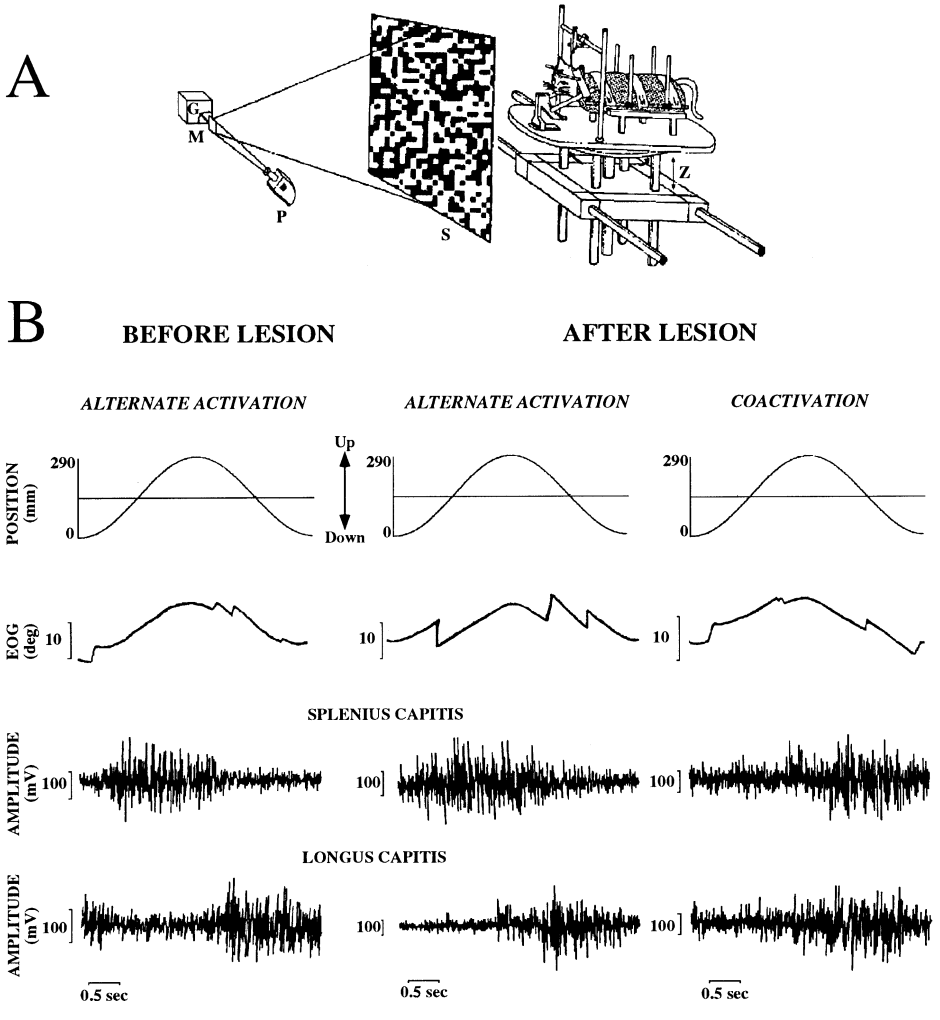

Fig. 1A, B Neck muscle and eye movement responses to sinusoidal visual stimulation in the vertical plane. **A** Experimental setup (see text). **B** EMG and EOG recordings during visual stimulation at 0.25 Hz in a normal cat (*left* before lesion) and in a unilateral vestibular neurectomy (UVN) cat from each subgroup (*right* after lesion). *From top to bottom*: visual pattern displacement; vertical eye movements (EOG); raw EMG activity from the neck extensor muscle (splenius capitis); raw EMG activity from the neck flexor muscle (longus capitis). The alternate activation of the antagonist neck muscles (out-of-phase EMG activities) was observed in all the cats (n=7) tested before lesion. By contrast, the UVN cats examined during the acute stage (day 4–18) exhibited either alternate activation (n=3) or coactivation (in-phase EMG activities: n=4) of the antagonist neck muscles

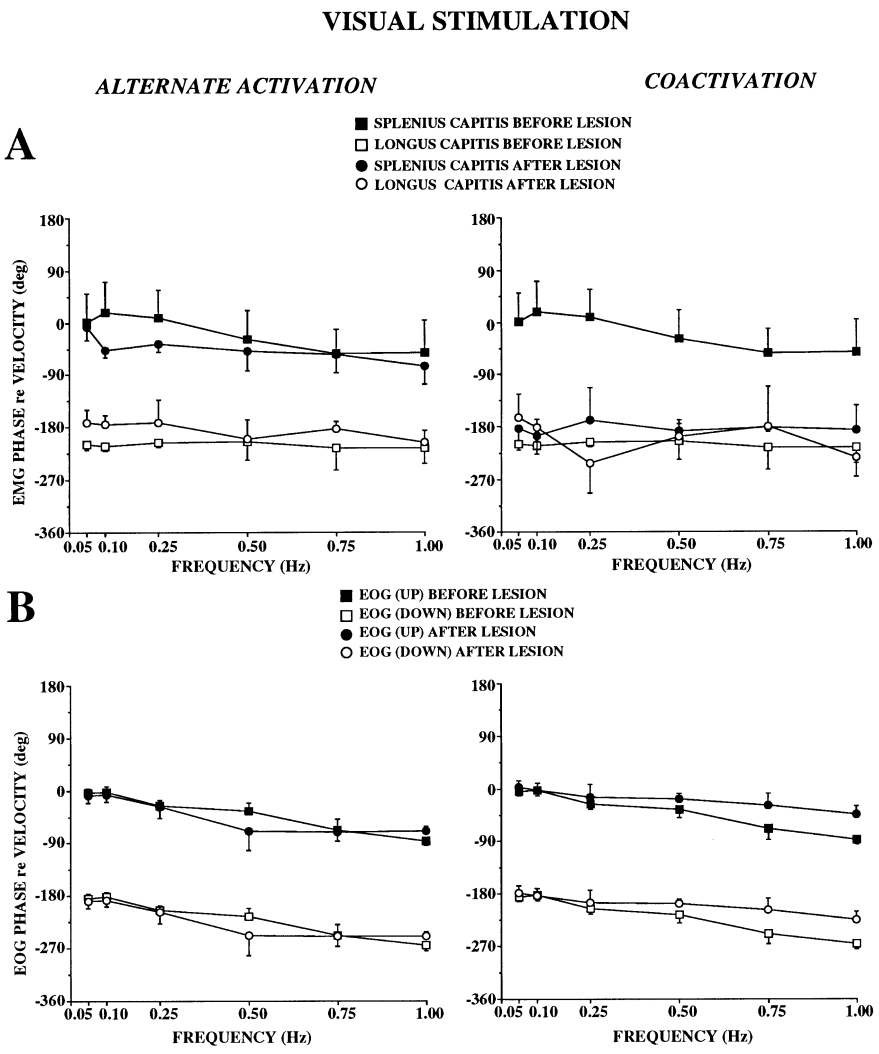

Fig. 2A, B Vestibular lesion-induced changes in the phase of the neck muscle and eye movement responses during visual stimulation. Bode plots illustrating the phase modifications of the EMG activity from the splenius and longus capitis neck muscles (**A**) and of the vertical eye movement responses (**B**). **B** EOG in the two subgroups of UVN cats showing alternate activation (*left*) or coactivation (*right*) of the antagonist neck muscles. Mean EMG and EOG phase values (degrees) are plotted with respect to stimulus velocity (*ordinates*) in the frequency range (*abscissae*). Mean data before UVN are reported for comparison. *Vertical bars* SD

Vertical EOG responses

A typical optokinetic reflex response was elicited by vertical translation of the visual surround in front of the cat, in its frontal plane. Animal motion in the vertical plane gave rise to otolith-ocular responses both in light and in darkness. Optokinetic responses were made up of slow eye movements in the direction of the visual pattern motion, interrupted by saccades bringing the eyes in the opposite direction. Otolith stimulus-induced responses were characterized by slow eye components directed away from the head motion and by saccades in the same direction as the head motion. EOG recordings were calibrated at the beginning and at the end of each experimental session by lighting visual targets (diodes) fixed on a vertical plate in front of the cat, at the same distance as that used in the course of the experiments (0.5 m). The cat had to perform ocular saccades on two alternately lighted visual targets at 290-mm intervals. The amplitude of the resulting ocular saccades served to calibrate the vertical EOG. Vertical eye position signals as well as position recordings of the platform (head displacement during otolith and visual-otolith interaction) or of the visual surround (optokinetic stimulation) were continuously recorded on a paper chart throughout the stimulation period. The on-line control of the vertical eye movements served to check the alertness of the cat, particularly during the optokinetic stimulation. Stimulus velocity traces were obtained by electronically differentiating the position recordings of the platform and of the visual surround. These traces and the eye position signal were fed into a computer. A semi-automatic program reconstructed the slow, cumulated eye position curve after quick phases were manually removed from the eye-position signals. The eye velocity curve was thereafter computed. Bode plots of the vertical eye movement responses were evaluated from eye velocity signal and stimulus velocity recordings. Gain was expressed as the ratio of the peak amplitude of the fundamental component of eye velocity to that of stimulus velocity. Phase was calculated as the difference in degrees between the fundamental component of peak upward stimulus velocity (optokinetic stimulation) or peak downward stimulus (otolith and visual-otolith interactions) velocity and the fundamental component of peak upward (or downward) eye velocity. Then, vertical EOG phase was expressed with respect to peak upward optokinetic stimulus velocity or peak downward otolithic stimulus velocity, since eye movements in the same direction are produced in both cases.

EMG and EOG responses recorded before UVN were computed first for each cat, giving individual phase and gain means and standard deviations (SDs). These values were obtained independently for the splenius and longus capitis neck muscle responses and for the upward and downward eye movement responses. As a rule, unlesioned cats showed low intra-individual variability. By contrast, after UVN two subgroups of cats were clearly distinguished on the basis of their EMG responses (alternate activation or coactivation of the antagonist neck muscles). Means were then calculated for each UVN cat of these two subgroups examined in each experimental condition. Values for each subgroup of cats were compared with the preoperative values for the entire population of control cats. Because of the small number of UVN cats in each subgroup (three and four lesioned cats), which preclude reasonable assumptions about the presence of Gaussian distributions, these unpaired data were statistically evaluated by the Mann-Whitney test.

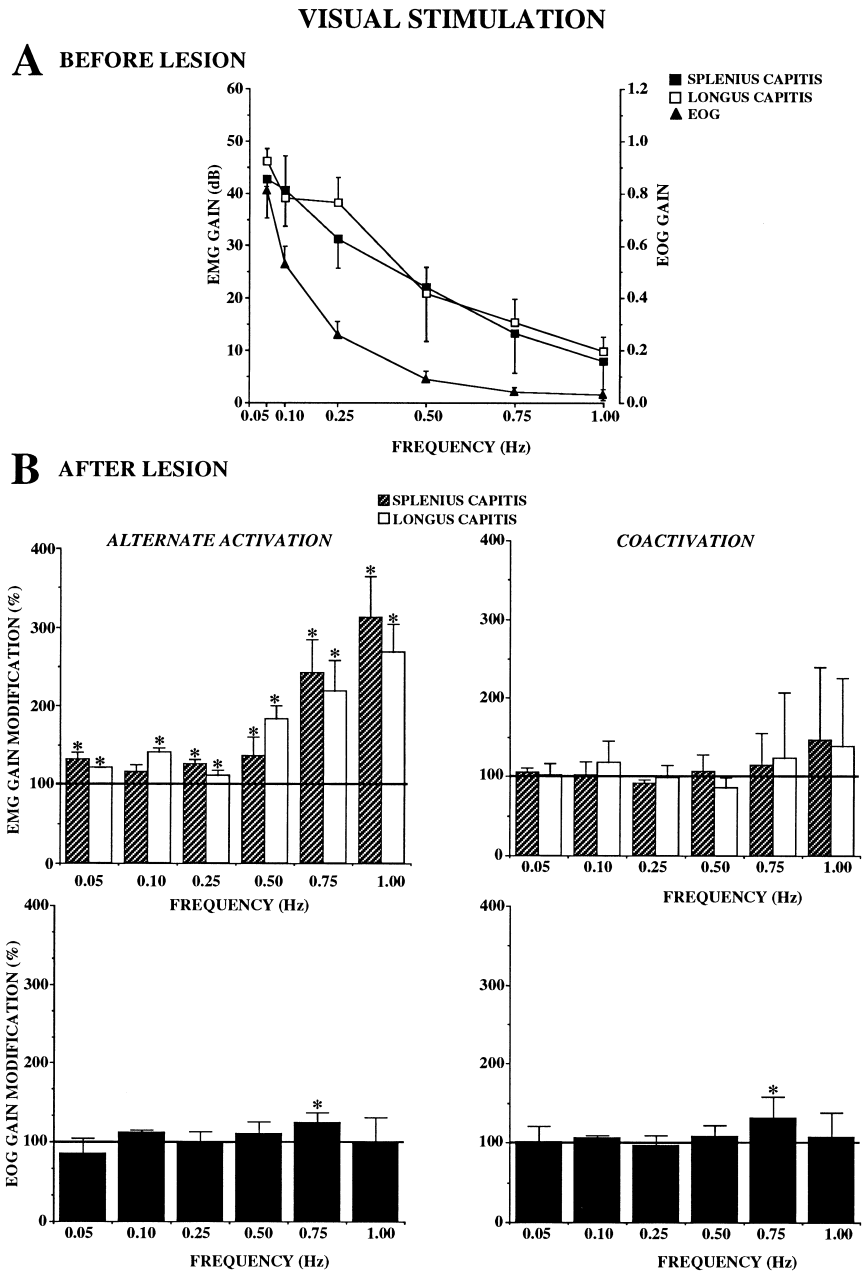

Fig. 3A, B Vestibular lesion-induced changes in the gain of the neck muscle and eye movement responses during visual stimulation. **A** Bode plots showing the mean gain values of the EMG and EOG responses in the frequency range before lesion. **B** Histograms illustrating the postoperative gain modifications of the EMG activity from the splenius and longus capitis neck muscles (*upper part*) and of the vertical eye movement responses (*lower part*) in UVN cats with alternate activation (*left*) or coactivation (*right*) of the antagonist neck muscles. Gain modifications are expressed in percentage of the preoperative values. *Vertical bars* SD of the means. *Asterisks* represent significant statistical difference between preoperative and postoperative data

Results

We previously described the neck muscle responses and the eye movement responses to visual (optokinetic), otolith, and visual-otolith interaction in a population of 14 alert normal cats (Borel and Lacour 1992a). The dynamic properties of the eye and neck responses recorded during sinusoidal linear motion of the animal and/or of its visual surround have been fully described in the 0.05- to 1.39-Hz frequency range. It was found that visual, otolith, and visual-otolith stimulations induced sinusoidal EMG modulations in the extensor (splenius capitis) and flexor (longus capitis) neck muscles and that these modulations were associated with upward and downward eye movements, respectively. With these three stimulations, the EMG pattern was always characterized by an alternate activation of the antagonist neck muscles. Figure 1B is an illustration of the raw alternate EMG activities from the antagonist splenius and longus capitis muscles in a normal cat during sinusoidal motion of the visual pattern at 0.25 Hz.

Postlesion recordings performed in the acute stage after UVN, from day 4 (when spontaneous ocular nystagmus had disappeared in the light) to day 18, revealed two EMG patterns. Among the seven UVN cats, three exhibited a pattern of neck muscle responses similar to that recorded preoperatively, that is, consisting of out-of-phase activation of the antagonist neck muscles. By contrast, the other four UVN cats showed a qualitative change in their motor response pattern, now exhibiting coactivation, that is, in-phase activity of their antagonist

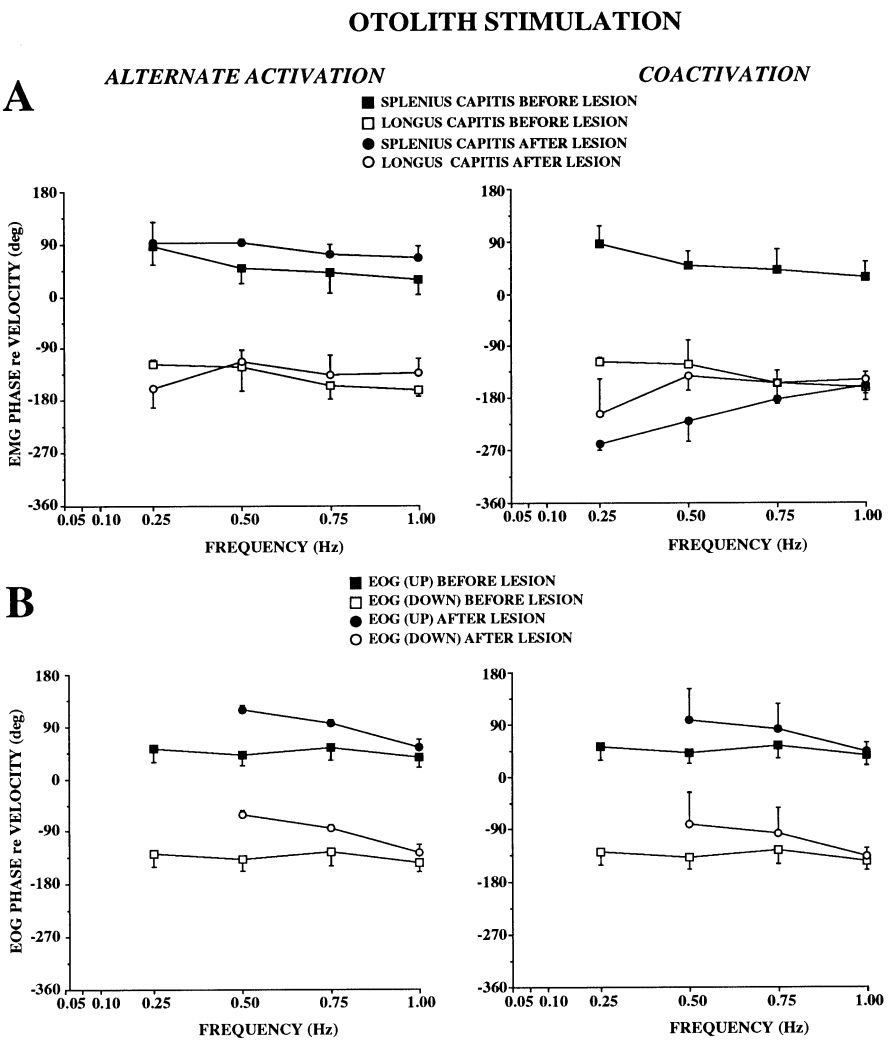

Fig. 4A, B Vestibular lesion-induced changes in the phase of the neck muscle and eye movement responses during otolith stimulation. Bode plots illustrating the phase modifications of the EMG activity from the splenius and longus capitis neck muscles (**A**) and of the vertical eye movement responses (**B**). **B** EOG in the two subgroups of UVN cats displaying alternate activation (*left*) or coactivation (*right*) of the antagonist neck muscles. Same conventions as in Fig. 2

neck muscles. Figure 1B illustrates the raw EMG activities of a UVN cat displaying alternate activation and one displaying coactivation of the extensor (splenius) and flexor (longus) neck muscles during optokinetic stimulation at 0.25 Hz. These two distinct EMG patterns found during visual stimulation were also consistently observed in the same subgroups of cats during otolith and visual-otolith interactions.

The vestibular lesion-induced changes of the dynamic properties of the neck muscle and eye movement responses were evaluated by comparing the preoperative data to the postoperative gain and phase values recorded in the seven UVN cats. The control data did not differ significantly from those recorded in our previous investigation on unlesioned cats (Borel and Lacour 1992a). Nevertheless, to statistically compare the same individuals before and after UVN, we used only the data from this study instead of pooling all the control data.

Vestibular lesion-induced changes during visual stimulation

In the subgroup of cats with neck muscle alternate activation

For the three cats with alternate activation of the antagonist neck muscles, mean phase values of the visually induced EMG responses were basically unaffected by vestibular lesion, compared with the controls. Figure 2A illustrates the mean Bode plots showing the phase of the splenius and longus capitis muscles before and after UVN. No significant changes were observed after UVN: the splenius muscle remained in phase relation with upward stimulus velocity peak (–6°±25° at 0.05 Hz and –75°±31° at 1 Hz, respectively), whereas responses from the longus muscle remained related to downward stimulus velocity peak (–175°±22° and –186°±20° at 0.05 Hz and 1 Hz, respectively). By contrast, the gain of these two antagonist neck muscles was strongly modified after vestibular lesion. Figure 3A plots the mean gain values of the EMG responses recorded in the frequency range, before UVN. EMG gain modifications

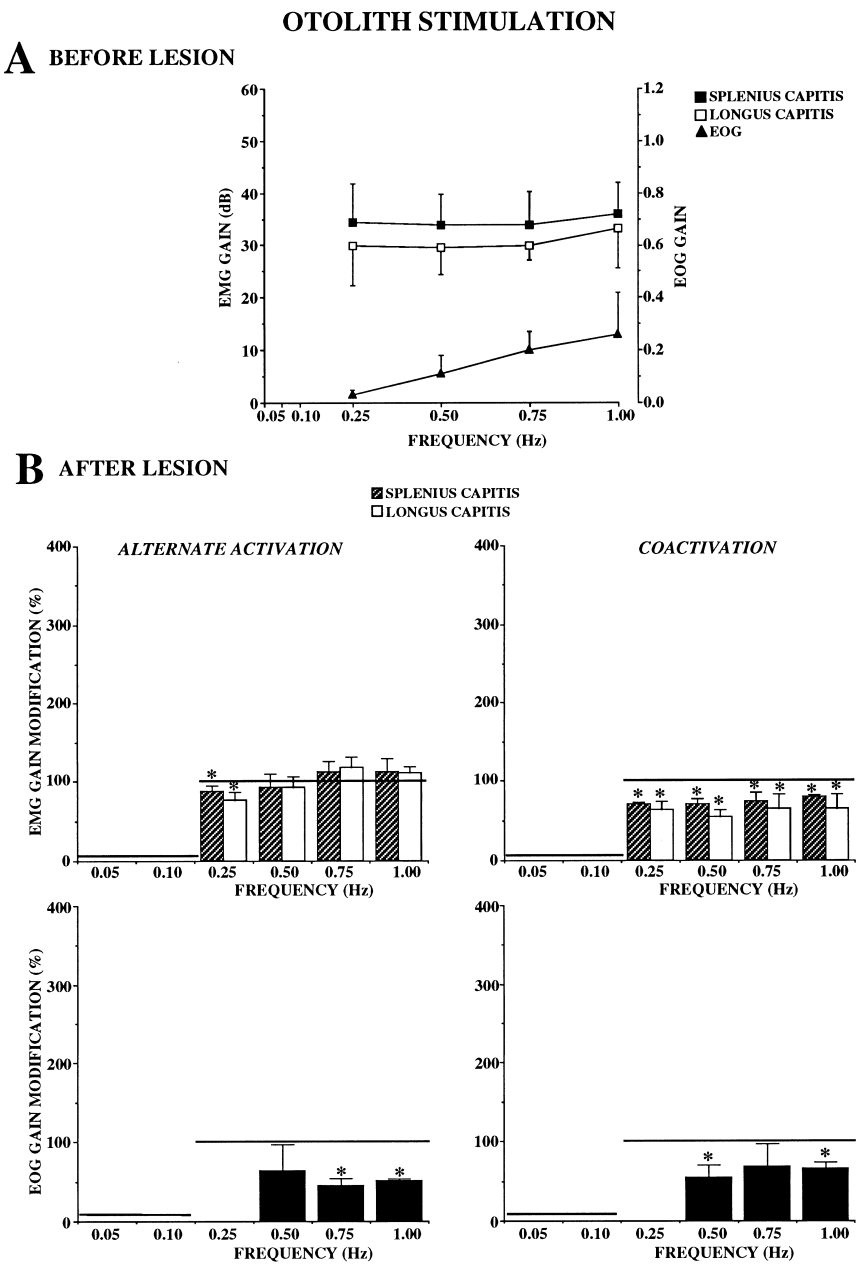

Fig. 5A, B Vestibular lesion-induced changes in the gain of the neck muscle and eye movement responses during otolith stimulation. **A** Bode plots showing the mean gain values of the EMG and EOG responses in the frequency range before lesion. **B** Histograms illustrating the postoperative gain modifications of the EMG activity from the splenius and longus capitis neck muscles (*upper part*) and of the vertical eye movement responses (*lower part*) in UVN cats with alternate activation (*left*) or coactivation (*right*) of the antagonist neck muscles. Same conventions as in Fig. 3

for the 3 cats with alternate activation, expressed in percent of the preoperative values, are reported in Fig. 3B. The visually induced EMG output was significantly increased in the whole frequency range. A gain increase averaging 132%±9% and 312%±52% ($P<0.03$) was seen for the splenius capitis at 0.05 Hz and 1 Hz, respectively. Data from the longus capitis showed similar significant gain modifications. Taken together, these results point both to an enhanced gain and to an extension of the functional working range of the visually induced neck muscle responses.

In spite of such changes in the gain values of the visually induced neck EMGs, no significant modifications were found in the associated oculomotor responses. Mean phase (Fig. 2B) and gain (Fig. 3B) of the optokinetic responses were not affected by the vestibular lesion in this subgroup of UVN cats.

In the subgroup of cats with neck muscle coactivation

Coactivation of the antagonist neck muscles during optokinetic stimulation was recorded in the other four cats. EMG activity from the extensor muscle (splenius capitis) showed a mean phase value 180° out of phase with that recorded before UVN, whereas response phase of the flexor muscle (longus capitis) remained unchanged. This unusual pattern of neck muscle responses did not depend on stimulus frequency, since it was seen in the whole frequency range (Fig. 2A). On average, splenius

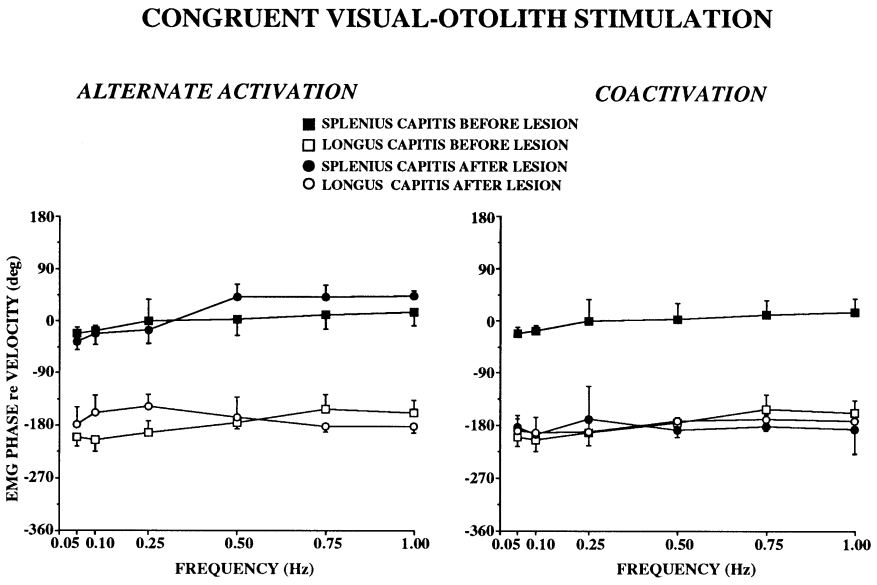

Fig. 6 Vestibular lesion-induced changes in the phase of the neck muscle responses during congruent visual-otolith interaction. Bode plots illustrating the phase modifications of the EMG activity from the splenius and longus capitis neck muscles in UVN cats displaying alternate activation (*left*) or coactivation (*right*) of the antagonist neck muscles. Same conventions as in Fig. 2

capitis response phase was –183°±21° and –185°±42° at 0.05 Hz and 1 Hz, respectively. By contrast with the previous subgroup of cats, mean gain values of the EMG responses did not significantly differ from the preoperative data (Fig. 3B). Finally, vestibular lesion had no significant consequences on the optokinetic response parameters. Phase (Fig. 2B) and gain (Fig. 3B) values of the eye movement responses also remained globally unchanged in the whole range of visual stimulation for this second subgroup of cats with respect to the controls.

Vestibular lesion-induced changes during otolith stimulation

The two patterns of neck muscle EMG activity seen during visual stimulation, that is, alternate activation and coactivation, were also observed in the same subgroups of cats when otolith stimulus was given alone.

In the subgroup of cats with neck muscle alternate activation

Neck muscle responses to otolith stimulation still slightly led the downward and upward stimulus velocity peaks for the splenius and longus capitis muscles, respectively (Fig. 4A). Gain modification was observed at the lowest frequency only (0.25 Hz), with significantly decreased EMG activities for both muscles (Fig. 5B). At higher frequencies, otolith stimulus-induced EMG responses did not differ from the controls. By contrast, eye movement responses to otolith stimulation were strongly depressed in the whole frequency range tested (Fig. 5B). The EOG gain significantly dropped to about 47%±8% of the control values on average in the high range of motion (0.50–1 Hz), in which otolith-induced eye movements normally developed in the control cats (see Fig. 5A). No asymmetry was found in the oculomotor responses. In addition, this EOG gain decrease was accompanied by a slight but significant phase lead of the eye movement responses below 1 Hz (Fig. 4B).

In the subgroup of cats with neck muscle coactivation

The oculomotor deficits in this subgroup of UVN cats were similar to those described for the other subgroup. Mean Bode plots show gain decrease (Fig. 5B) and phase lead (Fig. 4B) in the 0.5- to 1-Hz frequency range. Recordings from the antagonist neck muscles displayed in-phase EMG activities of both muscles. The splenius capitis showed a mean phase value 180° out of phase with that recorded before UVN. At the lowest 0.25-Hz stimulus frequency, the longus capitis muscle showed a significant phase lag relative to the controls. In the upper frequency range, EMG responses from the two antagonist neck muscles did not significantly differ from those of the longus capitis for the controls (Fig. 4A). By contrast with the previous subgroup of cats, EMG recordings from both muscles exhibited a considerable gain decrease in the whole range of otolith stimulation in the subgroup of UVN cats with EMG coactivation (Fig. 5B). On average, gain drop was 26%±4% and 39%±5% of the preoperative values for the splenius and the longus capitis muscles, respectively.

Vestibular lesion-induced changes during visual-otolith interaction

Congruent otolith-visual interaction was monitored by moving the cat in front of the earth-stationary visual pattern. As a rule, the two EMG patterns described above during separate visual or otolith stimulations (alternate

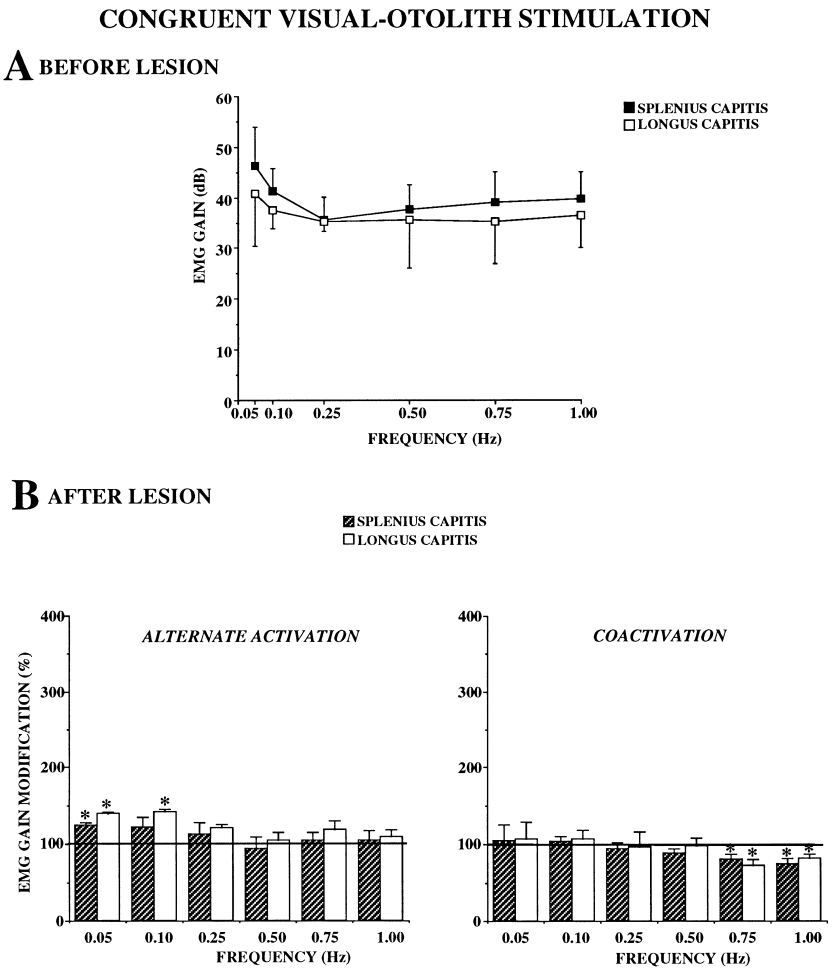

Fig. 7A, B Vestibular lesion-induced changes in the gain of the neck muscle responses during congruent visual-otolith stimulation. **A** Bode plots showing the mean gain values of the EMG responses in the frequency range before lesion. **B** Histograms illustrating the postoperative gain modifications of the EMG activity from the splenius and longus capitis neck muscles in UVN cats with alternate activation (*left*) or coactivation (*right*) of the antagonist neck muscles. Same conventions as in Fig. 3

activation and coactivation of the antagonist neck muscles) were also observed in the same subgroups of cats during combined otolith-visual interactions. Bode plots illustrating the mean phase values of the neck muscle responses in both subgroups (Fig. 6) again show that splenius EMG activity lags the controls by about 180° in the UVN cats exhibiting coactivation, whereas response phase of the longus is not modified.

In the subgroup of cats with neck muscle alternate activation

Gain values of the antagonist neck muscles were significantly increased in the low-stimulus frequency range (0.05–0.10 Hz; see Fig. 7B). Gain enhancement averaged 23%±0.8% and 40%±1% in this low range for the splenius and the longus capitis muscles, respectively. At higher frequencies, no gain modification was observed.

In the subgroup of cats with neck muscle coactivation

EMG activities from both muscles were significantly reduced in the high range of motion (more than 0.50 Hz; see Fig. 7B) with respect to the controls and to the subgroup of UVN cats with alternate activation of the antagonist neck muscles ($P<0.03$).

Concerning oculomotor responses, no drastic changes were found in either subgroup of UVN cats compared with their preoperative data. Bode plots recorded during congruent visual-otolith interaction (not illustrated herein) showed EOG gain reduction at the highest stimulus frequency only (1 Hz), which was significant (58%±8% at 1 Hz; $P<0.04$) in the only subgroup with EMG coactivations.

The modifications of eye movements and neck muscles responses occurring after UVN during conflicting visual-otolith interaction (not illustrated herein) were similar to those during pure otolith stimulation for both subgroups of UVN cats: (1) EOG responses showed gain reduction in the whole frequency range and phase lead below 1 Hz; (2) the dynamic properties of the neck muscles were not significantly modified in the subgroup with alternate activation, except at the lowest frequency, whereas gain decreased sharply in the whole frequency range for the UVN cats with coactivation. Gain decrease averaged 32%±3% and 30%±8% for the splenius and the longus capitis muscles, respectively.

Discussion

Lesion-induced changes in neck muscle responses

EMG activity from the splenius and longus capitis muscles was recorded preoperatively. The data showed a pattern of alternate activation of antagonist neck muscles in all the cats. In the acute stage of the vestibular compensation process after UVN, two motor strategies were observed. For three of the seven cats, the temporal activation of the individual neck muscles was the same as that recorded before UVN. For the other four cats, vestibular neurectomy resulted in a shift from the initial pattern of alternate activation to a pattern of coactivation of the flexor and extensor neck muscles. In both subgroups, the response patterns of the antagonist neck muscles were consistent for each cat independently of the experimental conditions, throughout the 3 weeks of testing.

The role of visual inputs in the early stages of vestibular compensation differed greatly in the two subgroups of UVN cats. Cats displaying alternate activation of the antagonist neck muscles showed an enhanced gain of the visually induced neck responses, particularly in the high range of stimulus frequencies. Such gain changes have been reported (Borel and Lacour 1992b). Gain increase very probably reflects the functional substitution role played postoperatively by the visual motion cues. The adaptive value of this visual substitution process is corroborated by data recorded during congruent visual-otolith interaction, since the postoperative motor response gain is both significantly increased in the low range of motion and similar to that recorded before lesion in the high-frequency range. Behavioral and electrophysiological data support the view that visual sensory substitution serves as a compensatory process for the defective dynamic properties of the vestibular reflexes (Precht and Dieringer 1985; Lacour and Borel 1993; Curthoys and Halmagyi 1995 for reviews). Similar adaptive changes have been demonstrated at the vestibular neuronal level by Zennou-Azogui et al. (1994) with the same experimental paradigm as ours. They found an increased sensitivity in the visually induced responses in the deafferented Deiters' nucleus neurons during the first 3 weeks postlesion. The changes in the dynamic properties of the vestibular neurons, which become able to code for high motion on the basis of visual motion cues, would probably constitute the neuronal substrate of the visual sensory substitution process underlying the neck response changes described here. Finally, since the strong gain enhancement for both neck muscle responses occurs without concomitant modification in the dynamic properties of the optokinetic responses, we suggest that increased efficacy of the visual motion cues substitutes for the lack of otolith inputs. In a previous paper (Borel and Lacour 1992a), we proposed that the tight correlation between neck EMG activity and eye movements in the normal cat could underlie the so-called eye-head synergy, that is a functional coupling of neck and eye responses, with neck muscle activities driven by eye velocity signals. However, the splenius muscles are no longer excited during upward eye movements in the coactivation strategy, suggesting that optokinetic responses and visually induced neck responses are activated in parallel. This suggestion is also supported by Vidal et al. (1982) and Wilson et al. (1983), who investigated the coupling between neck muscle activity and eye movements.

Results from the subgroup of UVN cats with coactivation provide the first description of temporal changes in the antagonist neck muscles after UVN. Such temporal changes of the antagonist muscle response pattern have been reported by Conway (1987) for forelimb extensors and flexors in decerebrate hemilabyrinthectomized cats. Conway noticed that labyrinth reflexes contralateral to the lesion were reversed in flexors, which showed coactivation along with the extensors during head rotation about an anterior-posterior axis. The coactivation strategy might subserve a behavioral substitution role operating during vertical linear translation in the UVN cat. However, the change from alternate activation to coactivation could be an instantaneous switch from one program to another. Indeed, although we found no preoperative coactivation in our experimental conditions, this motor program could be present before UVN and used in a context-dependent fashion by intact cats. Keshner et al. (1989) described such a shift between central programs in humans during isometric head stabilization. Moreover, simultaneous contraction of the antagonist muscles has been observed in the ontogenesis of motor tasks including head stabilization (Assaiante and Amblard 1993) and grasping behavior (Gachoud et al. 1983). It appears also in our study that vision does not fulfill a substitution function for this subgroup of cats. This lack of visual substitution is corroborated by the decreased gain of the EMG responses evoked during congruent visual-otolith interaction. These results indicate that EMG responses are not able to compensate for animal motion in the high frequency range, in which the vestibular system normally ensures adapted neck responses. Finally, the gain decrease in the otolith-induced neck responses evoked in darkness suggests that the coactivation strategy has a limited adaptive value.

Lesion-induced changes in eye movement responses

Eye movement recordings showed that the vertical optokinetic responses remained unchanged after UVN. On the contrary, the dynamic responses of the otolith-induced eye movements were similar to those observed in the semicircular canal system, with gain reduction and phase lead. However, we show that gain reduction was similar for both the upward and downward direction of translation. This symmetric response to linear acceleration may reflect the anatomy and the physiology of the primary saccular neurons (Fernandez and Goldberg 1976). Opposite conclusions concerning the utricular system have been drawn in labyrinthine-defective patients by Curthoys et al. (1991). Using a perceptual paradigm, they evidenced a response asymmetry of the horizontal otolith function, with marked deficits in the perception of hori-

zontal linear acceleration directed towards the lesioned side. Taken together, the effects of UVN on otolith-ocular function lead to the conclusion that a single otolith system is symmetrically sensitive for vertical acceleration and asymmetrically sensitive for horizontal acceleration, as for the semicircular canal-ocular reflex function (see Curthoys and Halmagyi 1995).

Surprisingly, while it is obvious that two response patterns of the antagonist neck muscles are used in the acute stage of vestibular compensation, there is no crucial difference between oculomotor responses in these two subgroups of cats. Therefore, different processes must be implicated in otolith-spinal and otolith-ocular compensation, although some vestibular neurons are known to be involved both in head and eye movement control. Indeed, independent recovery processes for vestibulospinal and vestibulo-ocular symptoms have been reported (see Schaefer and Meyer 1974; Precht 1986).

In conclusion, this study provides insight into the early adaptive mechanisms underlying the vestibular compensation process after UVN in the alert cat. A more complex picture of compensation for the otolith function seems to be emerging from these data, since the presence of visual substitution process depends on the animals themselves. Studying vestibular-defective patients, we recently evidenced such a differential weighting of visual cues for balance control: some patients relied heavily on vision and others did not (Lacour et al. 1997). The next step will be to determine whether the different adaptive mechanisms described herein are restricted to the early stage of vestibular compensation.

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