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Perceptual and oculomotor effects of neck muscle vibration in vestibular neuritis

Ipsilateral somatosensory substitution of vestibular function

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Summary

Afferent cervical somatosensory input may substitute for absent vestibular information as part of central vestibular compensation after unilateral peripheral vestibular deficit. In order to determine the particular contribution of neck muscle spindles to the perception of body orientation and to the oculomotor system, we measured (i) the subjective visual straight ahead (SVA) by psychophysical tests and (ii) the changes in eye position by video-nystagmography during unilateral stimulation of the posterior neck muscles by vibration (100 Hz). Twenty-five patients with subacute unilateral vestibular lesion (vestibular neuritis) and 25 controls participated in the study. Vibration elicited a horizontal displacement of SVA towards the side of stimulation in all subjects. Mean displacement (± SD) was 3.28 ± 2.96° for right-side and 3.45 ± 2.93° for left-side stimulation in controls. Muscle stimulation on the patients' lesion side induced a significantly higher displacement (11.51 ± 6.63°) than contralateral stimulation (3.04 ± 2.95°, P < 0.01, paired Student's t *test). The mean difference during stimulation between the two sides in the patients was 8.02 ± 5.52°; in the controls, however, it was only 0.74 ± 0.47° (P < 0.001, Student's* t *test). This asymmetry increased gradually in patients over a period of weeks, reaching a maximum at days 60–80 and declining thereafter. Video-nystagmography revealed that ipsilateral stimulation in patients induced large horizontal eye deviations of up to 25° towards the side of the lesion (9.1 ± 7.6°,* n *= 18). Contralateral stimulation induced only small shifts, which were within the range of controls. The correlation coefficient between displacement of the SVA and change in eye position was high (*r *= 0.94,* P *< 0.0001), indicating that the shift of SVA is the perceptual correlate of the directional change of gaze in space. This interpretation was supported by two control experiments in which the subject was required to (i) indicate the subjective straight ahead by finger-pointing with the eyes closed and (ii) adjust SVA when looking through horizontally reversing prisms. Vibration of neck muscles caused almost no displacement of the SVA when it was indicated by pointing with the eyes closed, but reversed the direction of the displacement if the subject wore reversing prisms. In summary, our data showed: (i) an increase in muscle spindle input following unilateral vestibular lesion; (ii) this increase is asymmetrical, restricted to the affected side, and gradually builds up over weeks; and (iii) the perceived effects during vibration are secondary to changes in eye position rather than changes in cortical representation of body orientation. This is the first study to demonstrate a unilateral increase in somatosensory weight, which substitutes for missing vestibular input.*

Keywords: vestibular neuritis; central compensation; cervico-ocular reflex; subjective straight ahead; neck muscle vibration

Abbreviations: PIVC = parieto-insular vestibular cortex; SVA = subjective visual straight ahead

Introduction

Unilateral peripheral vestibular failure causes a distressing vestibular tone imbalance with rotational vertigo, spontaneous horizontal rotatory nystagmus away from the affected side, and postural imbalance with falls towards the affected side. This tone imbalance is readjusted by central compensation, which involves multiple processes in distributed neuronal networks at different locations with different time courses (Fetter and Zee, 1988; Curthoys and Halmagyi, 1995; Dieringer, 1995; Brandt *et al*., 1997). Since central compensation of peripheral vestibular lesions is less perfect

than generally believed, other additional mechanisms must subserve the functionally insufficient compensation. One such mechanism is sensory substitution (Curthoys and Halmagyi, 1995). Proprioception and vision may substitute for parts of the missing vestibular input to allow better gaze stabilization during head movements (Dichgans *et al*., 1973; Gresty *et al*., 1977). For example, neck afferents provide information about head position, and the cervical proprioceptive system is especially important for body orientation (Magnus, 1924; Mergner *et al*., 1991, 1992; Hlavacka *et al*., 1996). The cervical proprioceptive system can be stimulated by neck muscle vibration (Biguer *et al*., 1988; Taylor and McCloskey, 1991; Karnath, 1994; Karnath *et al*., 1996; Popov *et al*., 1996; Lekhel *et al*., 1997), which elicits, for example, displacements of the subjective straight ahead.

It is difficult to separate the amount of somatosensory and visual substitution along the time course of central vestibular compensation in a patient with unilateral vestibular deficit. Vibration of unilateral neck muscle has different effects on spatial orientation and eye position, which may, however, indicate changes in the cervical proprioceptive input. In this study the following questions were addressed: (i) is there a (possibly side-specific) increase in the cervical proprioceptive influence on perception – measured by the subjective visual straight ahead (SVA) – and on the horizontal eye position in patients with unilateral vestibular deficit? (ii) if there is an increased influence on perception, what is the underlying mechanism? (iii) what is the time course of these changes?

Method

Patients and controls

We examined 25 patients with subacute vestibular neuritis and persisting vestibular deficit (13 males and 12 females aged 17–81 years, mean ± SD = 50.2 ± 12.3 years) and 25 age-matched, healthy control subjects (14 males and 11 females aged 49.1 ± 14.2 years). Thirty-five of the initial 60 patients with vestibular neuritis recovered within the first 3 weeks and were therefore excluded. All subjects gave their informed written consent to participate in the study according to the guidelines of the Ethics Committee of the Medical Faculty of the University of Munich. The experiments were done in accordance with the Helsinki II Declaration. The diagnosis of vestibular neuritis was based on (i) the patient's history (acute/subacute onset of severe prolonged rotational vertigo and nausea), (ii) clinical and neuro-ophthalmological examinations (horizontal–rotatory spontaneous nystagmus towards the unaffected ear without evidence of a central vestibular lesion, pathological bedside testing of high-frequency vestibulo-ocular reflex, and postural imbalance with ipsiversive Romberg fall), and (iii) electronystagmography with caloric irrigation (hypo- or unresponsiveness of the affected horizontal semicircular canal) as described elsewhere (Strupp *et al*., 1995). The maximum slow-phase velocity during caloric irrigation with warm (30°) and hot (44°) water had to be less than 2–3°/s or 0°/s on the affected side (hyporesponsiveness/unresponsiveness), otherwise the patients were not included in the study (exceptions are mentioned in the Method and Results sections).

All patients had normal MRI findings, especially of the temporal bone, vestibular nerve (Strupp *et al*., 1998) and brainstem.

Vibration of the neck muscles was induced in the subacute stage, i.e. later than 2–8 weeks after symptom onset (exceptions mentioned in Method and Results).

Neck muscle vibration

To activate the cervical proprioceptive system unilaterally, dorsal neck muscles were stimulated by vibration either on the right or on the left side. To find the best stimulation point, we adjusted the tip of the vibrator until subjects indicated that the visual target appeared to move (Biguer *et al*., 1988; Taylor and McCloskey, 1991). Changes in the position of the vibrator could cause alterations in the magnitude of the illusory movement. In most subjects the best stimulation point was ~5 cm below and ~2 cm lateral to the inion. Stimulation there should primarily affect the splenius capitis, semispinalis capitis and trapezius muscles. We used an experimental electromechanical physiotherapy vibrator with a fixed frequency of 100 Hz, an amplitude of vibration of 1 mm, and a round contact surface (diameter 1.5 cm). The application pressure was ~0.5 kg. Vibration was applied seven times for 20 s in each patient to measure the SVA and changes in eye position before and during stimulation (exceptions are mentioned in the Method and Results sections). The head of the subject was fixed by a head-holder and bite bar to prevent any head or body movement.

Vibration experiments were performed during the subacute stage of the disease, i.e. >2 weeks after symptom onset when (i) the spontaneous nystagmus was weak enough for the patients to be able to fixate a target (fixation suppression of the spontaneous nystagmus was important for measuring both SVA and the horizontal eye position) and (ii) the spontaneous tonic displacement of the SVA was <3–4°.

Measurement of the subjective straight ahead

The subjects were seated upright in the centre of a half-spherical screen in total darkness, instructed to sit relaxed and to look at a laser point in front of them. They were asked to direct the examiner how to move the laser spot on the screen until the point reached their straight ahead position. The SVA was measured horizontally (in degrees) in each subject seven times before and during neck muscle vibration; the resting time between each stimulation was 1 min. Subsequently the differences in the mean of the SVA before stimulation and during stimulation were calculated for each side.

The subjective straight ahead was also determined in two

patients and control subjects in a different way, which has been described by Biguer *et al*. (1988). The subject was also seated in the centre of a spherical screen in total darkness, but this time he had to indicate the subjective straight ahead by holding a laser pointer in the right hand below the index finger. In this way the subjective straight ahead was also measured horizontally (in degrees) before and during neck muscle vibration with the patient's eyes open and closed, i.e. when pointing to the subjective straight ahead with the eyes open and closed.

In addition, the subjective straight ahead was determined in two patients and six control subjects while wearing prism goggles, which reversed the visual field in the horizontal plane.

Measurement of eye position by video-nystagmography

The horizontal eye position was measured in 18 patients and 18 controls by video-nystagmography before and during neck muscle vibration; these recordings were made on the same day as the measurements of the SVA. The subjects were instructed to sit in a relaxed way and to look at a laser point in front of them (0° position). The head of the subject was fixed by a bite bar, to prevent any head or body movements.

Longitudinal study of changes in the SVA

The time course of changes in the displacement of the SVA during neck muscle vibration was followed in two patients for 1 year. The amount of peripheral deficit was measured by caloric irrigation.

Statistical analysis

Statistical analysis was performed with the F test and subsequently the unpaired or paired Student's t test, using the SAS program. To analyse the correlation between the changes in SVA and the changes in eye position induced by vibration of the dorsal neck muscles, a linear regression analysis ($y = a + bx$) was performed using Microcal Origin software (Microcal Software Inc., Northampton, Mass., USA). In all analyses statistically significant differences were defined as $P < 0.05$.

Results

Displacement of the subjective straight ahead

During neck muscle vibration all subjects reported motion of the laser spot in front of them. The direction of motion was always away from the side of the stimulation. This apparent movement was small and slow in control subjects and in patients, when vibrations were on the unaffected side. However, stimulation of the neck muscles ipsilateral to the vestibular loss caused large and fast apparent movements of the laser spot. Despite these apparent movements, the patients did not complain of vertigo.

Horizontal displacement of SVA adjustment was always towards the side of stimulation. In controls, the maximal displacement was ~10° (3.28 ± 2.96° during stimulation on the right side and 3.45 ± 2.93° on the left, mean ± SD, n = 25). The difference between stimulations on the right and left sides was small: ~0.1–4° (Fig. 1A).

Initially the patients exhibited a spontaneous tonic deviation of the SVA towards the affected side (up to 10°) during the first few days after symptom onset. The accompanying spontaneous nystagmus, however, prevented reliable measurement of the SVA at this stage. More than 2–3 weeks after symptom onset, when the vibration experiments were performed, the tonic deviation was smaller (<3–4°); this deviation under resting conditions was then used as the individual reference. Muscle vibration on the lesion side of the patients induced a significantly higher displacement (11.51 ± 6.63°) than contralateral stimulation (3.04 ± 2.95°, $P < 0.01$, paired Student's t test). The maximum displacement reached 30° during ipsilateral stimulation (Fig. 1B).

The difference in the displacement of the SVA between the ipsilateral and contralateral stimulations was significantly higher in patients than in controls (by up to 25°; compare A and B in Fig. 1; see also Fig. 2). The mean of the differences in controls was 0.74 ± 0.47°; in patients, however, it was 8.02 ± 5.52° ($P < 0.001$, Student's t test). This was solely due to the ipsilaterally increased effect, i.e. even those patients with the highest displacement on the affected side had normal values for the non-affected side (Fig. 1B). In patients, the displacement of the SVA during stimulation of the non-affected side showed the same distribution as the displacement in healthy controls, and there was no correlation of the displacement between the affected and non-affected sides in patients.

Determination of the subjective straight ahead in the finger-pointing experiments with the eyes closed revealed no or minimal displacements (0–6°) even during stimulation of the side with the vestibular lesion (Fig. 3A). With the eyes open, however, the displacement was as large as SVA. The wearing of horizontally reversing prisms reversed the directions of displacement (Fig. 3B).

Time course of changes in displacement of the SVA

An increase in ipsilateral spindle input was observed within the first 50–80 days in the two patients measured repeatedly over 1 year. Thereafter, this asymmetrical input gradually declined (Fig. 4). Both patients showed a partial recovery of peripheral vestibular function, which was first detected by caloric irrigation months after symptom onset. In both patients caloric irrigation with warm (30°) and hot (44°) water showed initially and after 4 weeks a maximum slow-phase velocity on the affected side of <2°/s. After 6 months maximum

slow-phase velocity was ~5°/s in patient KI and ~8°/s in patient CU (Fig. 4.); after 1 year maximum slow-phase velocity was ~10°/s in both patients.

Changes in horizontal eye position during neck muscle vibration

We observed small changes of up to 6° in horizontal eye position in controls during stimulation of the neck muscles. The differences between left and right were smaller than 2° (Fig. 5A). Like the displacements of the SVA, stimulation ipsilateral to the lesion induced large horizontal ipsiversive eye deviations of up to 25° in patients (10.1 ± 7.6°) (Fig. 5B, C); contralateral stimulation caused small eye deviations of up to 7° (3.3 ± 2.9°, $P < 0.001$, paired Student's t test). Ocular deviations occurred, although all subjects tried to fixate the small central laser spot.

Figure 6 illustrates the high correlation coefficient (r =

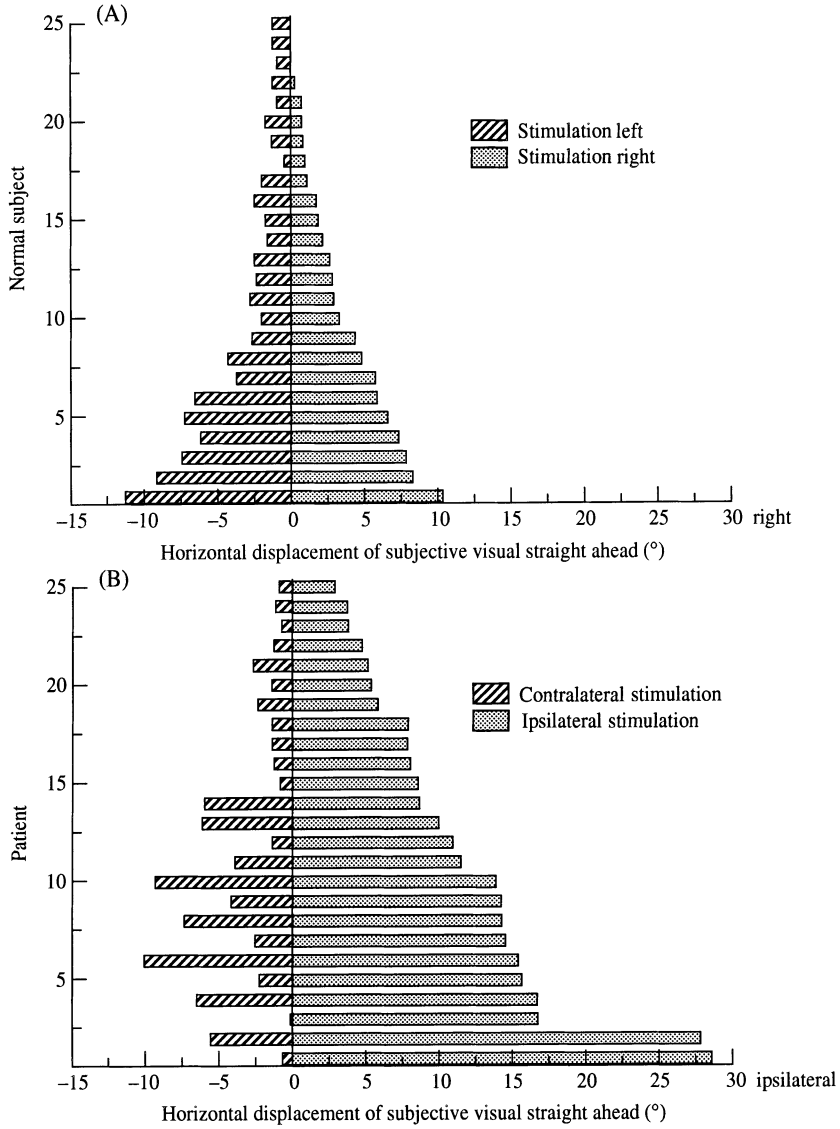

Fig. 1 Displacement of the SVA in control subjects (n = 25) and in patients with vestibular neuritis (n = 25). **A** illustrates the horizontal displacement of the SVA during stimulation of the right and left dorsal neck muscles for each of the 25 control subjects, arranged in descending order from ~10 to 0.1°. During vibration there was a horizontal displacement of SVA towards the side of stimulation in all control subjects. The maximal displacement was ~10° in controls. The difference in the displacement of the SVA between stimulation of the right and left sides was small (~0.1–4°). **B** shows the horizontal displacement of the SVA during stimulation ipsilateral and contralateral to the vestibular lesion for each of the 25 patients, arranged in the same way as in **A**. During vibration there was a horizontal displacement of SVA towards the side of stimulation in all patients. The maximum of displacement was ~10° in patients during stimulation contralateral to the lesion. However, during stimulation ipsilateral to the lesion the maximum displacement of the SVA was much higher (up to 30°).

0.94 ± 1.49, $P < 0.0001$) between the differences of the displacement in the SVA and the differences in the horizontal eye deviations in the 18 patients during neck muscle vibration ipsilateral to the peripheral vestibular lesion.

Discussion

Several studies (Biguer *et al*., 1988; Taylor and McCloskey, 1991; Karnath, 1994; Karnath *et al*., 1996) have demonstrated that stimulation of the cervical proprioceptive system by neck muscle vibration leads to displacement of the subjective straight ahead. Unilateral vibration of the posterior neck muscles elicits an apparent head motion (Taylor and McCloskey, 1991) and an apparent movement of a visual target (Biguer *et al*., 1988; Karnath, 1994) to the contralateral side. On the basis of these studies, we evaluated the effects of unilateral (side-specific) neck muscle vibration on horizontal SVA and on changes in horizontal eye position in patients with unilateral vestibular lesion. Three findings were new. (i) Displacement of SVA and of horizontal eye position was significantly higher during stimulation ipsilateral to the peripheral vestibular lesion, indicating an ipsilaterally increased cervical somatosensory input on the multisensory process of spatial orientation. We did not observe vibration-induced nystagmus, which has been described by Yagi and Ohyama (1996); in their experiments, however, they performed simultaneous bilateral stimulation. (ii) This asymmetry did not occur immediately after the vestibular loss. As regards the time course, the magnitude of neck muscle vibration effects built up over several weeks, reached a maximum between days 60 and 100 after symptom onset and then declined. (iii) There was a high correlation coefficient between the changes in SVA and those in the horizontal deviation of eye position. Further, the displacements of the subjective straight ahead by finger-pointing were opposite in direction when wearing reversing prisms. These findings raised further questions: are the deviations in eye position during neck muscle vibration induced by a modification of the cortical representation of visuospinal directions, or is the deviation of the SVA simply secondary to the gaze changes?

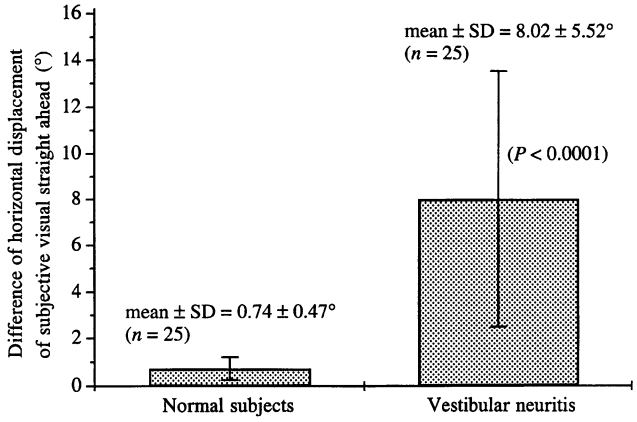

Fig. 2 The difference in the displacement of the SVA between stimulation of the right and left sides in controls and patients. The columns represent the difference, expressed as mean ± SD, in displacement of the SVA between stimulation of the right and the left neck muscles in controls, and ipsilateral and contralateral to the lesion in patients. For controls the mean difference was 0.74 ± 0.47° (SD). For the patients the mean value was more than 10 times higher (8.02 ± 5.52°; $P < 0.001$, Student's *t* test).

Vibration of dorsal neck muscles and the role of cervical proprioceptive input

Muscle vibration (by giving a false stretch signal) activates the primary endings of the muscle spindles, particularly Ia spindle afferents, and increases their firing rate, thereby eliciting a tonic contraction (tonic muscle reflex) (Goodwin *et al*., 1972). This leads to a kinaesthetic illusion of head movement when the actual movement is frustrated by a mechanical restraint. Due to the complex anatomy of the

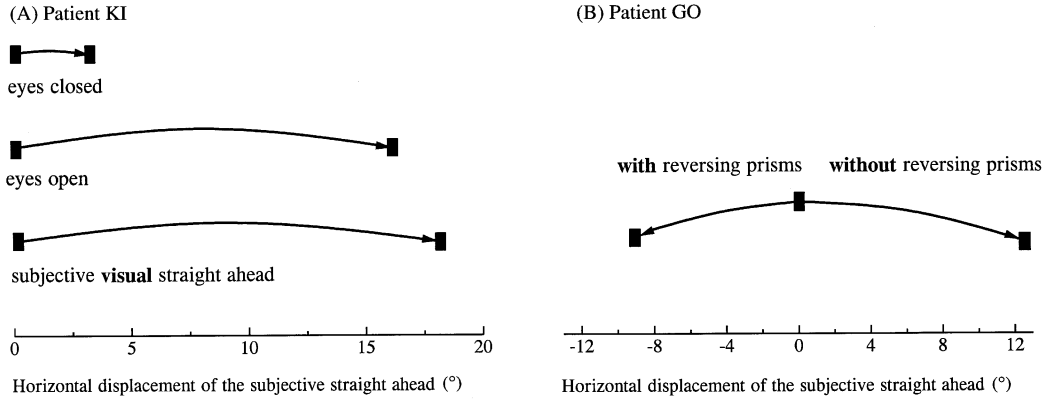

Fig. 3 (**A**) Displacement of the subjective straight ahead during neck muscle vibration on the affected side determined by finger-pointing with eyes closed (upper trace) and eyes open (middle trace) and as SVA (lower trace) in the same patient. (**B**) In another patient the subjective straight ahead was also determined during neck muscle vibration on the affected side while the patient was and was not wearing horizontally reversing prisms during finger-pointing: the prisms reversed the direction of displacement.

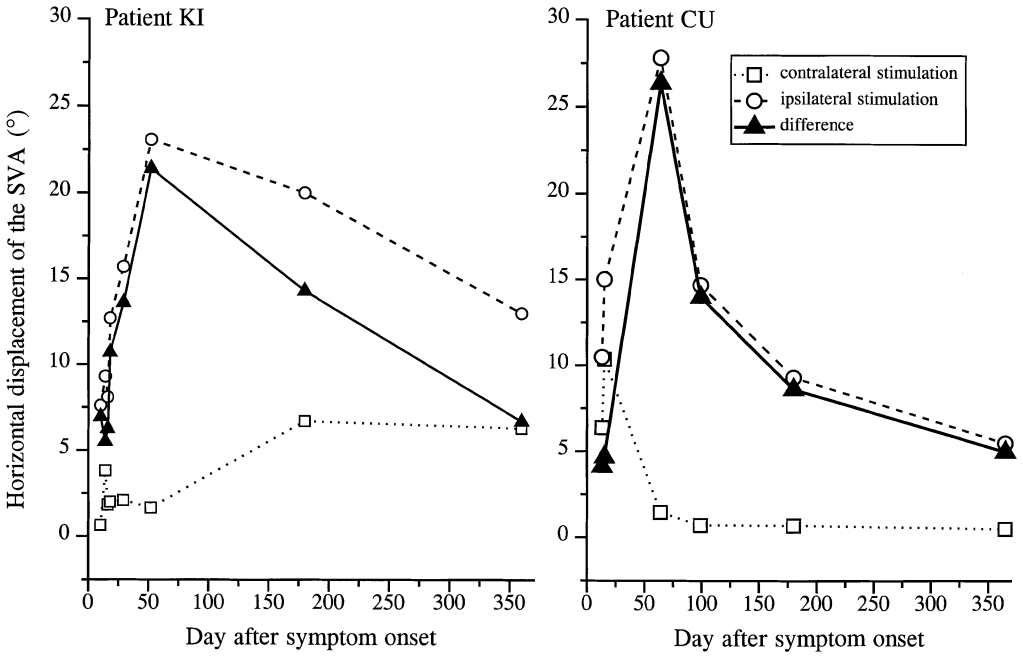

Fig. 4 Time course of changes in displacement of the SVA in two patients. The three curves represent the displacement of the SVA during ipsilateral and contralateral stimulation as well as the difference between the two curves. There was a pronounced increase in this difference over time, beginning at day 10 and continuing to day 60–80 after onset of the lesion. After this time the displacement of the SVA during ipsilateral stimulation and the difference declined. During the decline peripheral vestibular function partially recovered.

neck muscles, it is difficult to determine precisely which muscles were vibrated. The position of the vibrator in our experiments (~5 cm below and ~2 cm lateral to the inion) should have affected mainly the splenius capitis, semispinalis capitis and trapezius muscles. Using selective electromyography, Mayoux-Benhamou *et al.* (1997) recently showed that the splenius capitis and semispinalis cause ipsilateral head rotation in humans.

During stimulation of the left neck muscles, for instance, apparent movement of the laser spot towards the right and displacement of the SVA to the left are consistent with vibration of the left splenius capitis, which would rotate the head to the left if it were not fixated (an object with fixed retinal position would be interpreted as an object moving in the direction of the head rotation).

We found a significantly higher cervical influence on SVA and horizontal eye position during stimulation ipsilateral to the vestibular deficit. At first glance, a strictly unilateral modulation of the cervical input seems surprising if one considers that the head–neck system is organized bilaterally and symmetrically. Vibration, however, only simulates muscle activation. If one compares this system with the bilateral cooperation of the semicircular canals, activation of one side corresponds to simultaneous inactivation on the opposite side. Thus, the neck system seems to be organized in a similar activation/deactivation mode for the two sides.

Animal experiments have also reported a change in the weighting of spinal afferent input during compensation of unilateral and bilateral vestibular lesions. For instance, Dieringer *et al.* (1984) found anatomical evidence of increased spinal afferent projections in the frog following unilateral vestibular loss. Furthermore, Straka and Dieringer (1995) described a higher increase in the dorsal root input ipsilateral to the lesion after hemilabyrinthectomy in the frog than occurred on the contralateral side. It has been reported that the gain in the cervico-ocular reflex after bilateral vestibular lesions is potentiated (0.4–0.7) in the monkey (Dichgans *et al.*, 1973) and in humans (Kasai and Zee, 1978; Bles *et al.*, 1984; Bronstein and Hood, 1986; Huygen *et al.*, 1991). This helps to restore gaze stability during head movements [in normal subjects the cervico-ocular reflex makes a negligible contribution to the stability of gaze (gain < 0.07) (Sawyer *et al.*, 1994)]. Behavioural studies have also shown the importance of increased cervical afferent input: cutting the upper cervical dorsal roots causes decompensation (of earlier compensation) in rabbits (Manzoni *et al.*, 1979) and squirrel monkeys (Igarashi *et al.*, 1969).

What causes the displacement of the SVA during neck muscle vibration?

To create an internal representation of body and head position and their movement in space, reliable information about head and trunk movement is necessary (Mergner *et al.*, 1992, 1993). Neck muscles contain numerous muscle spindles, and the density of mechanoreceptors is also high in the joints of the neck (Bakker and Richmond, 1981; 1982). Proprioceptive

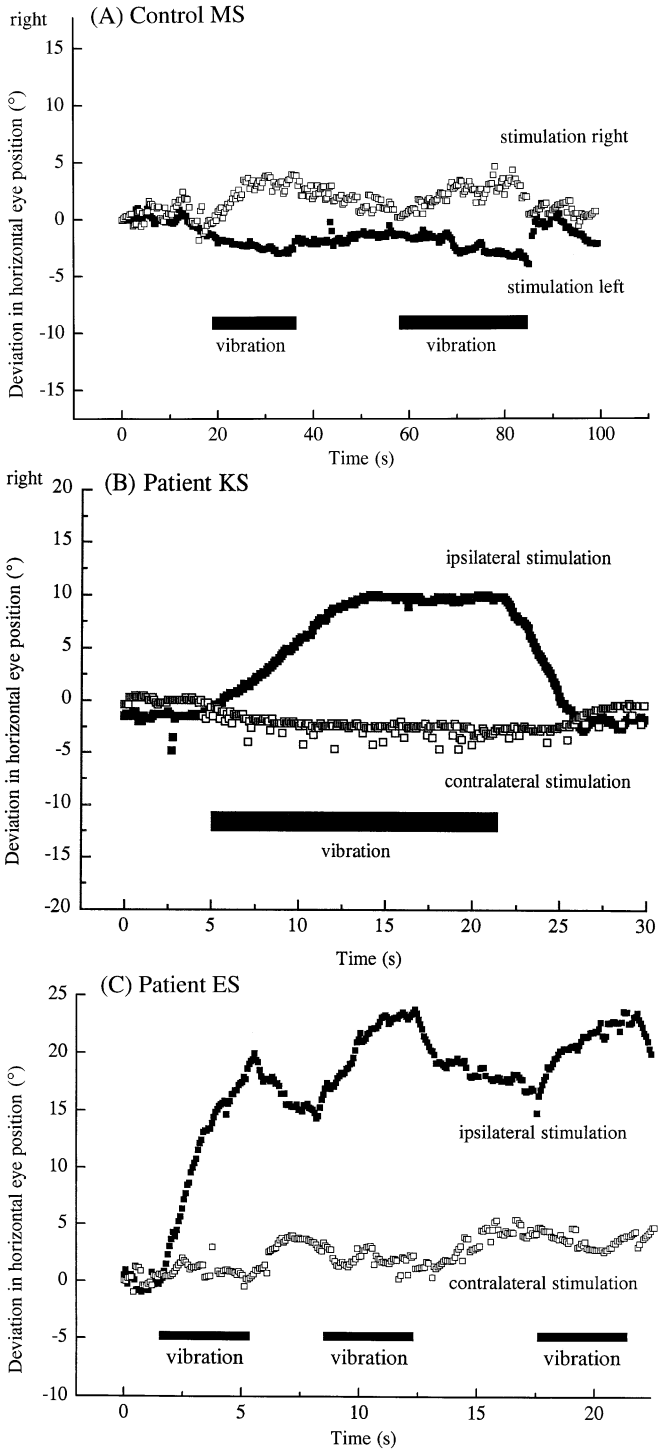

Fig. 5 Recordings of horizontal eye position. (**A**) Healthy control subject; (**B**) Patient with right-sided vestibular neuritis; (**C**) Patient with right-sided vestibular neuritis, repetitive stimulation. The patient's eye position was recorded using video-nystagmography before and during ipsilateral and contralateral neck muscle vibration. Stimulation contralateral to the lesion induced small changes in eye position. Ipsilateral stimulation in patients induced changes in eye position which reached 25° towards the side of the lesion.

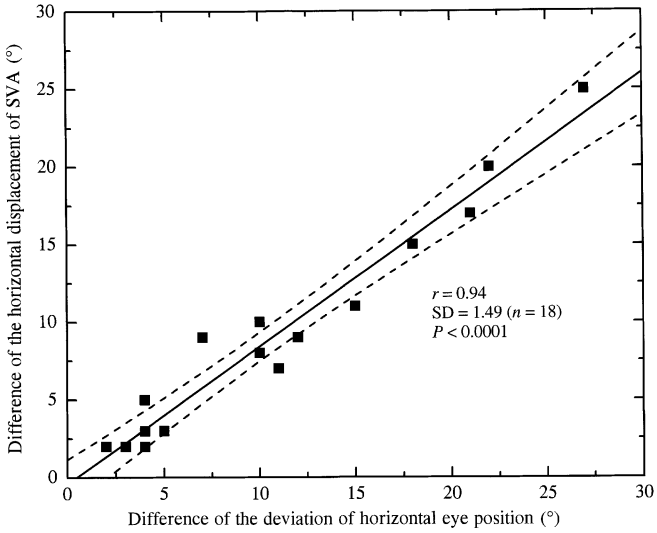

Fig. 6 The graph illustrates the correlation of the differences in the horizontal displacement of the SVA and the differences in changes in horizontal eye position during stimulation ipsilateral to the peripheral lesion in 18 patients. Linear regression analysis gives a high correlation coefficient ($r = 0.94$, $P < 0.0001$) between the two parameters. Confidence bands (confidence level 0.95) are also shown (dashed lines).

signals from the neck muscles form the main non-vestibular input, activating neurons in the vestibular nuclei (Rubin *et al.*, 1975, 1977) as well as 'vestibular' cortical neurons of the parieto-insular vestibular cortex (PIVC).

The correlation between the changes in SVA and those in eye position in our experiments and the well-known interactions between neck muscle afferents, vestibular nuclei and vestibular cortex suggest that either (i) the observed changes in eye position during neck muscle vibration are caused by proprioceptive changes of the cortical 'body-centred coordinate system', or (ii) the changes in eye position directly induce the observed displacement of the SVA via the cervico-ocular reflex. Both possibilities appear feasible in the light of neuroanatomical and physiological studies.

For example, Grüsser *et al.* (1990) showed that neurons in the PIVC of monkeys (*Macaca fascicularis*) were also activated by somatosensory stimulation, whereby mainly movements of the neck and shoulder joints elicited vigorous responses. They assumed that the changes in PIVC activity were caused by mechanoreceptor input ('neck receptor stimulation') because superficial skin stimuli did not activate PIVC neurons. Since 'vestibular' cortical neurons of the multisensory PIVC were intensively activated by dynamic neck receptors, one would expect neck receptor stimulation to induce movement in psychophysical experiments; this has indeed been observed (Bles and de Jong, 1982; Mergner *et al.*, 1983; Biguer *et al.*, 1988; Taylor and McCloskey, 1991; Karnath, 1994; Karnath *et al.*, 1996). To obtain a space-organized, body-centred and/or world-centred reference frame, the coordinates of the peripheral sensory organs, e.g. the neck receptors, must be transformed and integrated (Karnath *et al.*, 1996).

Other anatomical and physiological studies demonstrated projections of cervical primary afferents to vestibular and oculomotor nuclei (Hikosaka and Maeda, 1973; McKelvey-Briggs *et al*., 1989; Neuhuber and Zenker, 1989; Suarez *et al*., 1989). Using horseradish peroxidase, Neuhuber and Zenker (1989) demonstrated projections from proprioceptive C2–C5 roots to the medial vestibular nucleus in the rat. Injections into dorsal root ganglia C7–L5 failed to produce significant labelling within the vestibular nuclei. Taoka *et al*. (1990) also reported electrophysiological evidence of the direct projection of upper cervical afferents to the ipsilateral nuclei. Hikosaka and Maeda (1973) reported a direct effect of neck muscle afferents from the cervical roots C2–C5 on abducens motoneurons in cats.

In view of (i) the strong correlation between the changes in eye position and the displacement of the SVA, (ii) the missing displacement of the SVA in the experiments with patients who pointed to their subjective straight ahead by finger with eyes closed, and (iii) the change in direction of the subjective straight ahead while the patient was wearing reversing prisms, we propose the following hypothesis: the increased influence on spatial orientation, measured as the displacement of the SVA, is due to a stronger ipsilateral somatosensory cervical input on oculomotor neurons via a unilaterally increased cervico-ocular reflex, which causes larger changes in eye position. This is not due to horizontal deviations of the vertical midsagittal meridian, as found in patients with hemineglect. The patients' failure to report vertigo during neck muscle vibration, which would be expected if the signal went to vestibular and/or cortical vestibular neurons, supports our interpretation.

Time course of changes

The time course of changes in the SVA was measured in two patients for 1 year. In principle two time courses seem possible: (i) an immediate increase in the particular sensorial weight of neck muscle input to substitute for absent vestibular information, and (ii) a gradual increase as a dynamic readjustment by active experience and error control that occurs over a period of time. A rapid change can occur only due to functional changes of pre-existing neuronal connections, such as neural adaptation. A gradual change also allows structural changes, such as synaptogenesis of axonal sprouting. Our data support the second mode. Dieringer *et al*. (1984) have demonstrated structural spinal changes in animal experiments after hemilabyrinthectomy within weeks. The decrease in the spinal input in the two patients followed for a period of 1 year was accompanied by partial recovery of labyrinthine function. Bronstein *et al*. (1995) described changes in visual and cervico-ocular functions with similar time courses during recovery from a bilateral peripheral vestibular failure.

Sensory substitution is one component of the complex process of central compensation of peripheral vestibular failure. Although all single components contribute to a common improvement of sensorimotor function, they occur at different locations and have different time courses. If the differential effect of one component is analysed separately, as in our study using neck muscle vibration, it becomes evident that they can also function independently. The increased somatosensory effect was restricted to the ipsilateral side. It primarily involved neck muscle vibration-induced changes only in eye position, and had no effects on other vestibular functions such as perception of self-motion or postural control.

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