

# Neck muscle vibration alters visually-perceived roll after unilateral vestibular loss

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Unilateral sternocleidomastoid muscle vibration was applied to 21 normal and six unilateral vestibular deafferented (uVD) human subjects at head erect and during 30° left and right whole body roll-tilt. In normal subjects, neck vibration had no effect upon the settings of a visual bar to subjective visual horizontal (SVH) in any roll-tilt condition. In uVD subjects settings to SVH were significantly altered by neck vibration,

with ipsilesional neck vibration increasing the SVH bias at head erect. Further, during contralesional roll-tilt, ipsilesional neck vibration in uVD subjects significantly increased the *E*-effect. These results suggest that compensation after vestibular loss allows cervical signals to influence visual perception of roll-tilt. *NeuroReport* 11:2659–2662 © 2000 Lippincott Williams & Wilkins.

**Key words:** Cervical; Gravitational horizontal; Labyrinth; Perception; Proprioception; Roll-tilt; Somatosensory; Subjective visual horizontal; Vertigo; Vestibular

## INTRODUCTION

Stimulation of the dorsal neck by vibration in normal human subjects produces illusions of head or body rotation [1–3], illusory movements of a stationary dot of light in darkness [4–7], and actual eye displacements [5,7]. These effects are not artefacts of head movements [6]. These results support the hypothesis that information from cervical somatosensory receptors plays an important role for head and body position sense and visual orientation. Physiological evidence [8,9] demonstrates that passive, cervical muscle and joint stimulation modulates neural activity in various parts of the vestibular nucleus. Spontaneous and positional nystagmus can be induced in rabbits, cats and monkeys by injection of local anaesthetics into the soft tissues on one side of the neck [10]. These and other data [11] suggest that stimulation of neck receptors may affect processing of vestibular information.

Karnath *et al.* [3] found that when normal subjects were exposed to both vestibular (caloric) stimulation and neck vibration simultaneously, the effect on horizontal deviation of perceived straight ahead was additive (or subtractive) with respect to the effect induced by each type of stimulation alone. Yagi and Ohyama [12] found in patients with uVD that nystagmus with horizontal, torsional and vertical components can be induced by dorsal neck vibration. Strupp *et al.* [7] found that in patients with vestibular neuritis, unilateral dorsal neck muscle vibration caused horizontal eye displacement to the side of stimulation which was significantly greater in magnitude when the vibration was ipsilateral to the lesion. Further, they found

that eye displacement and deviations in the visually perceived straight ahead were highly correlated.

Torsional eye deviations can occur with neck vibration of uVD patients [12,13], and given that the visual perception of roll-tilt of visual stimuli is determined in part by torsional eye position [14,15], we considered that neck vibration would affect the perceived roll-tilt of visual stimuli. To test this hypothesis we measured subjective visual horizontal (SVH) during unilateral vibration of the sternocleidomastoid (SCM) muscles alone at head erect and in combination with 30° roll-tilt of the whole body (to study possible interactions with a stimulus that is known to change visual roll-tilt perception) in patients after unilateral vestibular loss.

## MATERIALS AND METHODS

Twenty-seven adults were tested. Of these, five had left-sided uVD and one right-sided uVD (two vestibular neurectomy, one cochleo-vestibular neuro-labyrinthitis, three vestibular neuro-labyrinthitis; mean age 32 years; two female, four male). The remaining 21 subjects were healthy controls (mean age 40 years; 4 female, 17 male) who were screened for abnormal vestibular function via a vestibular impulse test. A motorised tilt-chair conducted roll-tilts of thirty degrees at 3°/s. The chair was converted to support two Aura subsonic actuators assembled into vibrators with a 25 mm tip; a computer mouse for remote control of bar; and a Compaq 420 CX laptop loaded with a Labview 3.1 virtual instrument (vi) program. The vibrators were connected to an Aura motor assembly amplifier and a Trio

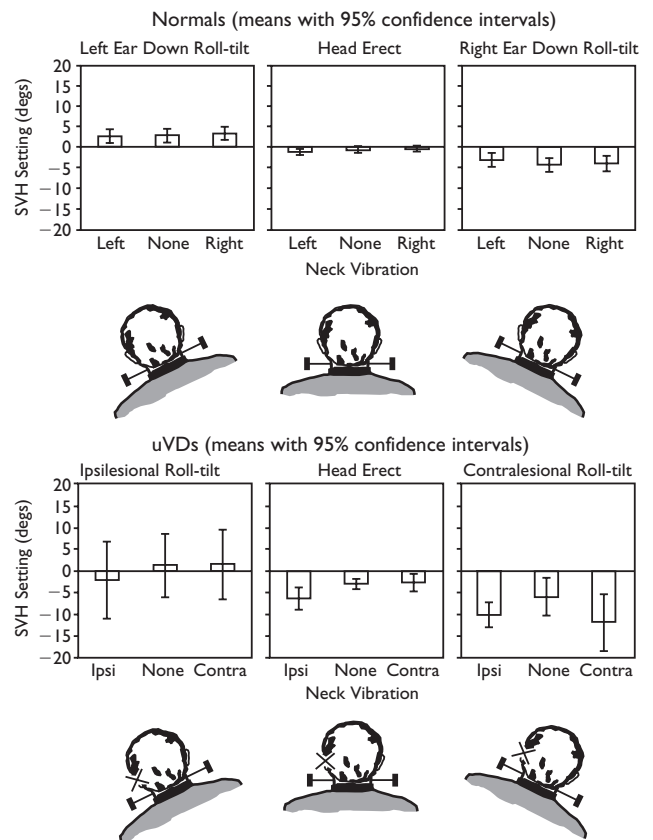
signalling generator set at 100Hz and vibrated at a constant peak-to-peak amplitude of 0.4 mm [4]. The locations of the SCM muscles were outlined with a felt-tipped pen during neck stretch and the vibrator tip target was marked at 25 mm below the mastoid process. Once seated in the tilt chair, supports were fitted against the subject's shoulders and straps were positioned and tightened around the lower thigh of each leg and across the chest. The subject's head was restrained in a natural erect position by two curved shaped clamps just above the ears. The laptop was adjusted to gravitational horizontal in direct line of sight at a distance of 65 cm from the right eye and each vibrator was placed firmly in position on the sternocleidomastoid mark on both sides. An eye patch was placed over the subject's left eye. All testing was performed in a completely dark room.

The program presented a bar composed of white dots randomly offset from gravitational horizontal ( $\pm 10^\circ$ ). Using the computer mouse to rotate the bar, subjects were required to adjust the bar to subjective visual horizontal within a 10 s interval for each of 10 successive trials in each condition. The data point for each subject for each condition was the average of these 10 trials. The first three conditions obtained horizontal baseline (no neck vibration) settings at head erect,  $30^\circ$  left ear down (LED) and  $30^\circ$  right ear down (RED) respectively. Horizontal settings were obtained for each subject during six neck vibration conditions: head erect, left side;  $30^\circ$  LED right side;  $30^\circ$  RED left side; head erect right side,  $30^\circ$  LED left side and  $30^\circ$  RED right side conditions respectively for all subjects. The sign of roll direction and setting error is consistent with the right hand rule: clockwise is positive from the subject's viewpoint.

## RESULTS

ANOVA of normal subject data showed a main effect of roll-tilt angle on SVH ( $F = 21.52$ ;  $df = 2,40$ ;  $p < 0.05$ ). However, there was no effect of neck vibration on SVH ( $F = 0.496$ ;  $df = 2,40$ ;  $p > 0.05$ ) and no interaction of roll-tilt angle and neck vibration on SVH ( $F = 1.44$ ;  $df = 4,80$ ;  $p > 0.05$ ). Fig. 1 shows our subjects to be a representative normal sample under no neck vibration conditions, with accurate SVH judgement at head erect (mean =  $-0.59^\circ$ ; 95% CI = 0.66) and normal E-effects at  $30^\circ$  roll-tilts (an E-effect being a small setting error in the direction opposite roll-tilt).

ANOVA of uVD patient data showed no effect of roll-tilt angle on SVH ( $F = 3.45$ ;  $df = 2,10$ ;  $p > 0.05$ ) but unlike the normal data there was a main effect of neck vibration on SVH ( $F = 4.38$ ;  $df = 2,10$ ;  $p < 0.05$ ). There was no significant interaction effect of roll-tilt angle and neck vibration on SVH ( $F = 2.12$ ;  $df = 4,20$ ;  $p > 0.05$ ). In Fig. 1, the uVD results are transformed so that all patients were treated as if they had left-sided uVD. The graph shows that uVD subjects at head erect have an SVH setting bias [14] so that they set the bar down on the side of their affected ear (mean =  $-2.89^\circ$ ; 95% CI = 1.15). This is significantly different from the SVH setting of normal individuals ( $t = -3.26$ ;  $df = 25$ ;  $p < 0.05$ ). Both ipsilesional and contralesional neck vibration at head erect in uVDs produced SVH settings in the bias direction that were significantly different from normals under the same conditions ( $t = -5.36$ ;  $df = 25$ ;



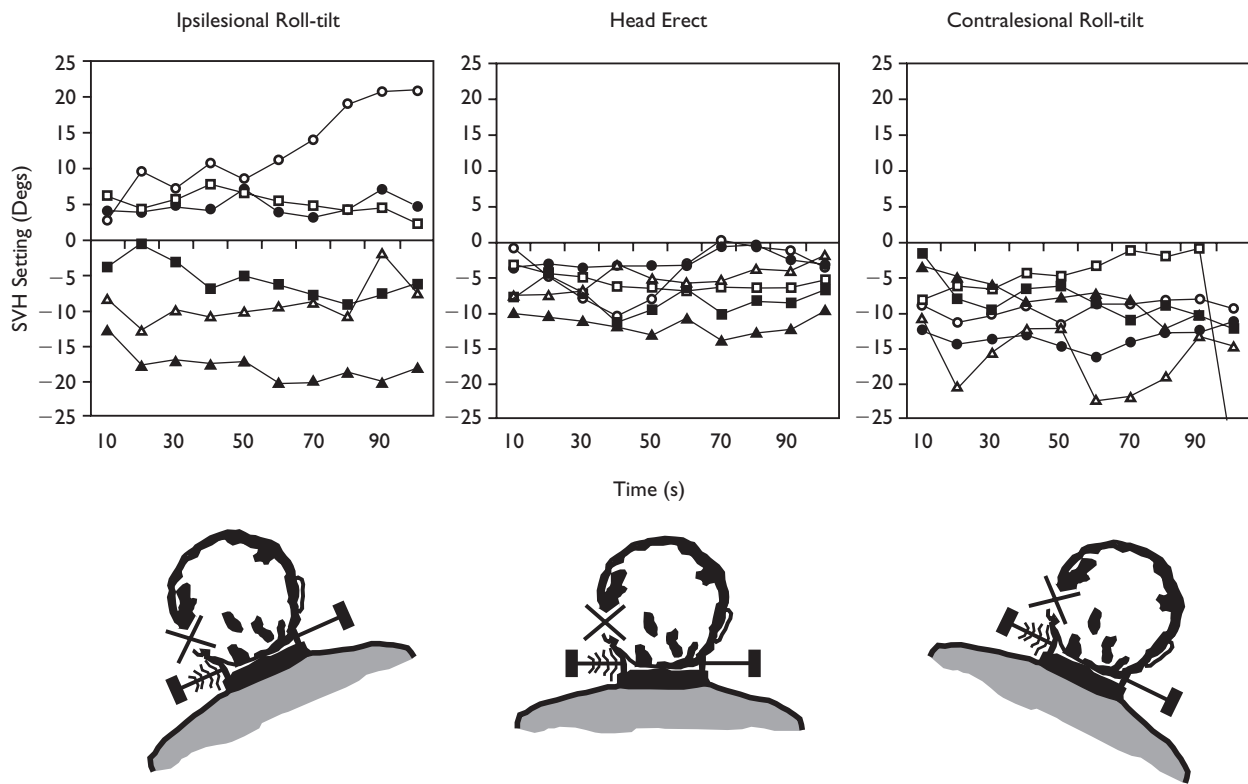
**Fig. 1.** Comparison of normal and uVD subjects' SVH settings during left sided, baseline and right sided neck vibration at  $30^\circ$  LED roll-tilt, head erect, and  $30^\circ$  RED roll-tilt (all uVD patients treated as if left uVD).

$p < 0.05$ ). Ipsilesional neck vibration significantly increased the settings compared to the bias usually seen in the uVD patients.

When uVD patients were roll-tilted  $30^\circ$  to the contralesional side, SVH settings showed an E-effect no different from normal individuals ( $t = -0.78$ ;  $df = 25$ ;  $p > 0.05$ ). However, a  $30^\circ$  ipsilesional roll-tilt produced such variable and apparently idiosyncratic responses from patients (see Fig. 1) that no significant E-effect was demonstrated.

When uVD patients were roll-tilted  $30^\circ$  contralesional, neck vibration (NV) produced E-effects significantly greater than those seen in normal individuals under identical neck vibration conditions (ipsilesional NV,  $t = -3.82$  and contralesional NV,  $t = -3.09$ ;  $df = 25$ ;  $p < 0.05$ ). On the other hand, neck vibration of uVD patients when roll-tilted  $30^\circ$  ipsilesional failed to change SVH settings compared with controls.

In Fig. 2, the 10 SVH settings made by each uVD subject over 100 s during ipsilesional neck vibration are plotted to show the consistent idiosyncratic ipsilesional response apparent in Fig. 1. Fig. 2 shows that while ipsilesional roll-tilt produces high intersubject SVH setting variance, the intrasubject variance in SVH settings is relatively low. In contrast, at head erect and during contralesional roll-tilt SVH settings have both low inter- and intrasubject variance (Fig. 2).



**Fig. 2.** Six individual uVD SVH settings over 100 s for ipsilesional neck vibration at 30° ipsilesional roll-tilt, head erect, and 30° contralateral roll-tilt.

A subject with left uVD was re-tested after 7 months in all conditions. The low intrasubject SVH setting variance seen in Fig. 2 for all subjects was maintained in this subject on both testing occasions. Further, the pattern of discrepancy from normals of the subject's SVH response in each condition was similar in the two test sessions ( $r=0.74$ ;  $df=8$ ;  $p<0.05$ ).

## DISCUSSION

The results of this study suggest that stimulation of the sternocleidomastoid muscles (through vibration) in normal humans does not affect visual perception of orientation in roll (i.e. SVH). This is at odds with the demonstrated effect [4] of dorsal neck muscle vibration on visual perception of straight ahead. An alternative conclusion is that the stimulus used here may not have been sufficient to increase firing rates of primary nerve endings; however, applying the stimulator to other regions of the neck produced the perceptual effects reported by previous investigators [7].

This study demonstrated for the first time that stimulation of the SCM muscles of uVD patients changes visual perception of roll-tilt. This is particularly clear even at head erect where ipsilesional neck vibration increased the bias in SVH settings usually seen in uVDs. This is consistent with the results of Strupp *et al.* [7] for visual perception of straight ahead, in that ipsilesional neck vibration changes perceived straight ahead more than contralateral neck vibration. During contralateral roll-tilt ipsilesional neck vibration increased the E-effect.

It has been suggested that following unilateral vestibular

loss, the brain compensates by using signals from other graviceptors in the body (e.g. the neck [16,17]). In uVD subjects, our results suggest that the weight of sternocleidomastoid muscle input is increased and specifically ipsilesional signals from the sternocleidomastoid muscles are given more weight in determining roll-tilt perception following vestibular loss than are contralateral signals from the sternocleidomastoid muscles (Fig. 1, uVD head erect).

During actual roll-tilts of the head, the interaction of the vestibular and neck signals becomes more complicated. Our results suggest that the weighting of the neck to vestibular signal is very predictable in the case where the head is tilted contralaterally. In this case it appears as if compensation has occurred so that any neck vibration adds to the otolith signal, indicating an increased roll-tilt. On the other hand, the weighting seems quite idiosyncratic when head roll-tilt is ipsilesional and neck vibration is applied. The mean and individual effects in uVD subjects were shown to be highly stable (both within testing sessions as well as at retest after 7 months).

Studies of the perceptual effects of neck muscle vibration have primarily stimulated the dorsal neck muscles which have a major role in yaw head rotations. However, in natural lateral head tilts, which change the perception of gravitational horizontal, the sternocleidomastoid muscles are contracted or stretched. If it is true that neck muscles contribute somatosensory information of head and body position via muscle spindle activity, and that muscle spindles are sensitive to vibration (increasing the firing rate

of primary endings [18]), then vibration of the sternocleidomastoid should induce changes in visual perception of gravitational horizontal in normal subjects, with uVDs showing an abnormal pattern of response. This is not consistent with our results, suggesting that sternocleidomastoid muscle stimulation does not contribute to visual orientation perception in the roll-plane in normal people.

It is possible that neck vibration has a direct effect on vestibular, rather than cervical pathways. To determine the amount of vibration that might directly stimulate the vestibular system during sternocleidomastoid muscle vibration, the magnitude of skull vibration was measured. The head acceleration values were well above threshold for sensitivity found in squirrel monkey vestibular afferents ( $-25$  dB re  $1$  g at  $80$  Hz) as reported by Young *et al.* [19]. These measures suggest that the vibration might reach the labyrinth and stimulate the vestibular receptors directly. However, our results are more consistent with sternocleidomastoid muscle activation because ipsilesional neck vibration in uVD subjects at head erect changes SVH settings, while contralesional neck vibration does not.

## CONCLUSION

Our results extend the understanding of the way in which the vestibular and cervical position receptors are integrated to provide veridical perception of orientation with respect to gravity. In normal subjects, it appears that vestibular (otolith) system signals have the primary role, with the cervical system providing head orientation signals only in subjects who have compensated after unilateral vestibular loss. Previous studies showing changes in horizontal visual orientation [4–7] through neck vibration in normals suggest that only certain (e.g. especially dorsal) muscles influence visual perception. Overall, neck vibration studies suggest that there is a hard wiring of reflexes from neck muscles to the vestibular nucleus and that only specific patterns of neck muscle activation (e.g. those that yaw rotate the head but not roll-tilt the head) activate these reflexes in normal subjects. Compensation appears to allow cervical signals to influence visual perception with signals from ipsilesional neck muscles being favoured particularly.

Given that previous studies [12,13] have shown that torsional eye position is affected by neck vibration, and that such rotations lead to changes in roll-tilt perception [14,15], it seems likely that our results are caused by torsional eye position changes just as the visual perceptual effects of dorsal neck vibration were shown to be caused by horizontal eye position changes. Further studies in this area are required to determine if the effects in uVDs of neck vibration on visual perception in roll are artefacts of vestibular stimulation and secondary to changes in torsional eye position. The high intrasubject stability of SVH settings made by normal and uVD subjects in this study suggests that our paradigm might be a useful additional method of diagnosing unilateral vestibular damage.

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