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Postural responses to vibration of neck muscles in patients with idiopathic torticollis

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Summary

Vibration of the dorsal muscles of the neck, simulating lengthening, in standing man causes a visible forwards tilt of the body shown on posturography as a tonic sagittal sway deviation. According to the theory that posture is organized with respect to a 'body schema' this deviation is a result of an interpretation of the concurrent neck afferent and vestibular signals. Considering the hypothesis that neck afferent signals may be misinterpreted in patients with spasmodic torticollis (ST) causing abnormal postural responses, we recorded body sway induced by unilateral dorsal neck muscle vibration in 22 idiopathic ST patients (19 treated with botulinum toxin) during upright stance with eyes closed. Comparison groups were 19 normal subjects and 11 patients with bilateral loss of vestibular function (labyrinthine defective, LD) in whom neck afference should be intact. Both treated and untreated ST and LD patients had absent or diminished sway deviations. When sway deviation did occur, it was sagitally oriented as with normal subjects and unrelated to ST head turns. In most ST and LD patients, neck vibration induced neck extension, an effect which is observed in normal subjects only if the torso is restrained. The results suggest that neck proprioceptive input retains local postural functions in ST, however, it is relatively ignored in the context of the whole body postural control and spatial orientation. The mild disorders of vestibular function reported in torticollis patients may be due to an inability to calibrate vestibular signals by reference to corroborative signals from neck proprioception.

Keywords: spasmodic torticollis, postural control, muscle vibration, neck proprioception, labyrinthine defective

Abbreviations: LD = labyrinthine defective; ST = spasmodic torticollis

Introduction

Afferent signals from the neck have an important role in the co-ordination of whole body posture and the organization of movement; disturbance of these signals results in ataxia (Cohen, 1961). The distortion of head posture and muscle tone in idiopathic torsion dystonia raise the possibility that the altered pattern of neck afference in spasmodic torticollis (ST) may also cause a more general incoordination. To investigate this possibility we have attempted to stimulate neck muscle proprioceptors in patients with torticollis using localized vibration. Vibratory stimulation of the dorsal neck muscles induces characteristic postural responses in normal subjects comprising a predominantly forwards (sagittal) sway (Gregoric *et al*., 1978; Lund, 1980; Morizono, 1991). The sway is an involuntary response thought to be due to a central interpretation of neck afferent and vestibular signals of head motion (explained at length in the discussion). However, the subject is conscious of his sway, so voluntary intervention in the response cannot be precluded.

Rotation of the head to the shoulder redirects the sway so that it becomes more lateral; thus, stimulation of either the right or left side of the neck, with head turned to the left, induces left lateral sway and with head turned to the right induces right lateral sway (Smetanin *et al*., 1993).

Although vibration applied to the neck muscle may transmit to stimulate other structures, such as the labyrinth, the main effect appears to be on muscle. The stereotypical sway

induced by vibration over the lower aspect of trapezius is unlike the irregular idiosyncratic sway we have observed with stimulation over the lower lateral occiput cranium (close to trapezius insertion) which guarantees a much stronger vestibular stimulus. In support of this view a recent study has shown that the effects of neck muscle vibration and concurrent vestibular stimulation are additive (Karnath *et al*., 1994) which implies that the effects of vibrating neck muscle are distinct from vestibular responses.

Recent studies (Anastasopoulos *et al*., 1997*a*, *b*) suggest that the neck input is relatively neglected when forming estimates of postural uprightness and the visual vertical. For these several reasons the question arises as to how postural reactions to neck stimulation by vibration behave in ST and how they may relate to the pathological head turns.

Comparisons were made between the responses of ST patients and those of labyrinthine defective (LD) patients and normal subjects. The interaction of neck and vestibular signals has a theoretical importance in that the 'central' comparison of the neck signal with vestibular input determines the response to vibration (*see* Discussion). There is also the long held clinical suspicion that there is an 'involvement' of the vestibular system in the pathophysiological mechanism of torticollis (Barre, 1929) which prompted us to question whether ST patients behave in any way as if they were LD.

Methods

Apparatus

Subjects stood on a 50×50 cm square sway platform 20 cm above the ground. The centres of the heels were ~10 cm apart and the feet splayed out at ~35°.

An electromechanical vibrator, 6.0 cm long, 2.5 cm wide with a flat 6×3.5 cm^2 rectangular contact surface was positioned over the medial and superior aspect of the right (or left) trapezius muscle and held in position by an elasticated shoulder girdle. The vibrator had a fixed frequency of 90 Hz (with a –38 dB second harmonic). Typical application pressure over the vibrator was 1.5 kg/cm^2. The amplitude of vibration was ~0.5 mm.

Postural responses to vibration of the Achilles tendons were recorded for comparison. Two electromechanical vibrators, 8.0 cm long and 3.5 cm wide with a concave surface to fit over the tendons were held in position by elastic bands. The frequency of vibration was 70 Hz, the amplitude was ~0.5 mm.

Angular movement of the head was recorded in 12 patients by means of the FASTRAK system (Polhemus). The FASTRAK consists of a stationary transmitter generating near field, low-frequency, magnetic field vectors, a small (2.8×2.3×1.5 cm^3, 17 g) receiver, which was attached to a lightweight helmet, and an amplifier and processor which used a hard-wired algorithm to compute the receiver's linear and angular 3D orientation with a resolution of 0.025°, 0.005 cm/10 cm of range. The receiver operated at an update rate of 120 Hz.

Stimulus conditions

Vibration was applied under the following conditions: (i) a 'control condition' without vibration, namely 30 s with eyes open gazing at the room background at 2 m distance followed by 30 s with eyes closed; (ii) a '35 s epoch' of vibration; (iii) five bursts of vibration of 4 s duration with a duty cycle randomized between 10 and 20 s; (iv) the short duration bursts of vibration were reapplied with the subject's head turned voluntarily to the right or left; (v) five bursts of the Achilles tendons vibration of 4 s duration with a duty cycle randomized similarly. Eyes were closed for conditions with vibration. Stimulation was applied firstly to the right side. Rest intervals of 5 min were allowed between right and left sided stimulation and between each condition.

Sway analysis

For the 35-s epoch, whole body sway in antero-posterior (sagittal, '*x*') and lateral (frontal, '*y*') planes was recorded from 5 s before to 40 s after the application of the stimulus. For the short bursts of vibration sway was recorded from 4 s before each stimulus to 2 s after it. The sway signal was digitized at 100 Hz for processing and calibrated in V/(cm kg) by shifting a known weight through specific distances in *x* and *y* across the platform. Data were normalized by dividing by the subject's weight to calculate the sway parameters in units of linear displacement of the centre of pressure of the feet. Sway responses to short sequences of vibration were averaged.

Sway parameters calculated were as follows. (i) 'Sway path', which is the distance across the platform through which the projection of the centre of mass of the subject (centre of pressure for higher frequencies of sway) onto the platform moves during a given time and is the sum of the distances moved with each sample (Δx, Δy), calculated as $\Sigma|\Delta x|$) for sagittal sway; ($\Sigma|\Delta y|$) for frontal sway; $\Sigma\sqrt{(\Delta x^2 + \Delta y^2)}$ for the 2D 'Total' sway vector. (ii) 'Stability', which is the sway path estimated from 5 s after the onset to the offset of 35 s vibration, as well as during 30 s period after vibration but excluding the initial transient 5-s interval. (iii) 'Latency' of the onset of the sway response as detected by deviation from the upper and lower limits of the envelope of the preceding 4 s of sway for the average response to 4 s vibration. (iv) 'Sway deviation', which is postural tilt from the baseline level averaged from 5 s after the onset to offset of the stimulus for 35 s vibration (*see* Fig. 1), and from 0.5 s after the onset to 0.5 s after the offset of the stimulus for 4 s vibration.

Instructions

Postural responses to vibration can be suppressed therefore subjects were instructed to stand relaxed without tensing

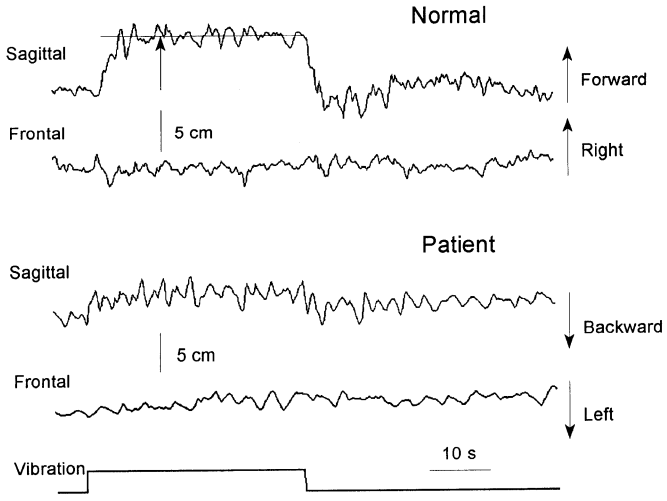

Fig. 1 Postural responses to 35 s vibration in a normal subject and in a torticollis patient, showing sagittal and frontal plane sway (platform signals in sagittal and frontal planes). The arrow indicates the normal sway deviation which is considerably smaller in torticollis.

themselves against sway. Normal subjects looked to the front. Patients were instructed to adopt their most relaxed head posture. For stimulus condition (iv), subjects were also asked to turn their heads to the right or left as far as comfortably possible.

Subjects

Nineteen normal subjects, 12 male and seven female with an age range of 24–49 years (mean ± SD, 33.8 ± 10.0 years), 19 patients with ST and 11 LD patients (with bilateral absence of labyrinthine function) gave their informed consent to the study according to the guidelines of the Ethics Committee of the Institute of Neurology and the National Hospital for Neurology and Neurosurgery, London, UK.

Patients

Of the 22 ST patients studied, 19 had been receiving botulinum toxin injections. These patients formed the main experimental group whose results were compared with the matched normal controls. Their characteristics are given in Table 1.

In addition we were able to test three patients with ST who had refused treatment with botulinum toxin because they preferred alternative medical treatment with relaxation exercises and herbal remedies whose efficacy was not apparent. All three patients were female (ages 50, 59 and 65 years) who had suffered dystonia for between 4 and 10 years. Their head deviations were 30° left tilt with 35° retrocollis, 20° right tilt with 30° left turn and 5° left tilt with 10° left turn, respectively, with severity Grades 4, 3 and 2, respectively.

The amplitude of head deviation was estimated by aligning a protractor with the axis of symmetry of the face for laterocollis and with the nasum–occiput axis for torticollis. Severity was graded on a five-point scale [modified from Stell *et al*. (1988) and Tsui *et al*. (1986)].

The 11 patients, five males and six females, with bilateral LD had a mean age of 52 ± 13.9 years. The time elapsed since loss of function ranged from 1 to 7 years. Six patients had idiopathic loss of function which we suspect was probably related to auto-immune factors. Two had lost function, probably because of a combination of meningitic (*Streptococcus suis*) infection and treatment with ototoxic medication. One subject had received ototoxic medication following coronary artery bypass, one subject had had bilateral neurectomies for neurofibromatosis Type 2 and one subject had chronic bilateral Ménière's disease.

All patients had an absence of slow phase compensatory eye movements as shown by 'broken up' doll's head eye movements in response to rapid head shaking and experienced oscillopsia. Nystagmus responses to 20°C caloric irrigation were absent. Steps of 80°/s in rotational velocity, and 80°/s peak velocity sinusoidal rotation in yaw at frequencies up to 1 Hz failed to provoke nystagmus. Vestibulo-myogenic responses in the neck to high intensity click stimulation were absent (Colebatch *et al*., 1994). The LD patients were only tested in control conditions and with head centred for 35 s during 35-s epochs of vibration.

Results

General characteristics of responses

The normal response to the vibratory stimulus consisted of a small initial backwards torque, seen better on averaged responses, followed by a visible forwards 'sway deviation' as the subjects posture tilted forwards (Fig. 1) by as much as 10° in extreme cases. The initial backwards sway may be caused by either a shift of weight to the heels in preparation for generating a moment to rotate the body forwards about the feet, or the moment itself. To correct for their sway deviation some subjects made corrective postural adjustments towards upright. At the offset of stimulation, sway deviation returned to baseline 'centre' and was sometimes accompanied by a few seconds of oscillatory sway.

The sway deviation induced by vibration was typically small in both ST and LD patients and often not visible to the onlooker (Fig. 1). However, unlike normal subjects, ST patients developed a visible backwards head tilt during vibration (*see* section on head movement below).

The following analyses are based on the 19 ST patients who were treated with botulinum toxin injections.

Quantitative analysis of responses

Sway paths

Sway paths for the control conditions, and during and after 35-s epochs of vibration are shown in Table 2. Two-way

Table 1 *Spasmodic torticollis patient characteristics*

Subjects/ Predominant head deviation	*n* (years)	Age (years)	Disease duration	Severity*
Torticollis				
Chin to right shoulder	9	46 ± 12	7.1 ± 5.2	3 ± 0.9
Chin to left shoulder	5	48 ± 7	11.3 ± 7.5	2.8 ± 0.8
Laterocollis				
Ear to right shoulder	3	32 ± 7	8.5 ± 6.4	3
Ear to left shoulder	1	37	12	3
Retrocollis	1	49	13	1

Means ± SD are shown. The patients comprised seven males and 12 females, all currently under treatment with botulinum toxin injections. *Severity grading (Stell *et al*., 1988; Tsui *et al*., 1986): absent (0), slight (Grade 1) if the deviation (tilt or turn whichever was greater) of the head was <5°, mild if between 5° and 15° (Grade 2) and moderate if between 15° and 30° (Grade 3). More severe states, with head deviation >30° (Grade 4).

Table 2 *Sway stability*

Subjects	Control data		Right side vibration		Left side vibration	
	Eyes open	Eyes closed	During vibration	After vibration	During vibration	After vibration
Normal subjects (n = 19)	897.69 ± 289.97	962.58 ± 287.26	1197.3 ± 338.92	938.79 ± 269.10	1198.99 ± 307.83	900.43 ± 356.44
Spasmodic torticollis patients (n = 19)	942.88 ± 204.21	1098.80 ± 339.56	1123.89 ± 380.43	1133.85 ± 447.84	1206.78 ± 775.38	1049.58 ± 293.33
Vestibular patients (n = 11)	1007.75 ± 410.95	1319.82 ± 495.27	1328.91 ± 472.76	1144.95 ± 438.08	1258.58 ± 524.99	1060.25 ± 375.31

Means ± SD are given.

ANOVA (analysis of variance) of sway paths for the three subject groups under the four conditions (eyes open or closed, and right or left side vibration) showed a difference in total sway between eyes open and closed, $P = 0.0059$, because sway was more stable with eyes open. The oscillatory component of sway was not different from normal subjects in ST patients, and sway during vibration was similar to sway with eyes closed for all subjects. The mean sway path for LD patients tended to be slightly greater across conditions but had higher variance so that, overall, LD patients were not significantly more unstable than ST patients or normal subjects.

Sway deviation during vibration: 35-s simulus epoch

Table 3 gives the postural deviations induced by 35-s epochs of vibration for normal subjects, and for ST and LD patients.

Three-way ANOVA of sagittal and frontal sway deviation as a function of subject groups and right versus left side vibration showed a marked difference between normal subjects and both groups of patients, $P < e^{-6}$. Sagittal and frontal sway deviations were markedly different for all subjects, $P < e^{-6}$. There was an interaction between subject groups and sagittal and frontal components of sway deviation; normal subjects had marked sagittal deviations in the forwards direction whereas both patient groups had only slight forwards sway deviations. Frontal sway was similar for all subjects.

The responses to right and left sided vibration were similar within all subjects and for sagittal and frontal planes. Subjects deviated a small amount to the left during right-side stimulation and to the right during left-side stimulation (two-way ANOVA, $P = 0.015$) (Table 3).

Sway deviation during vibration: 4-s stimulus epochs

The average postural responses to 4-s epoch vibration are given in Table 4. The responses to 4-s vibration resembled those evoked during 35-s stimulation but sway deviations were smaller. Normal subjects assumed head turns typically of 60°. Patients' pathological head turns ranged in amplitude from 40° left to 50° right (mean 22°) and they typically achieved a 70° voluntary turn in the direction of their pathological deviation and one of 50° in the opposite direction.

Three-way ANOVA comparing sagittal and frontal sway deviations in normal subjects and ST patients in different head positions showed a difference between subjects ($P = 0.0026$) and an effect of head position ($P = 0.035$). There were significant interactions between both subject groups ($P = 0.0002$) and head position ($P = 0.02$) with sagittal and frontal directions of sway deviation. ST patients had smaller sagittal sway deviations than normal subjects in all head positions and normal subjects developed additional sway in the frontal plane when their heads were turned sideways.

Table 3 *Mean sway deviation (35 s vibration)*

Subjects	Sagittal plane		Frontal plane	
	Right vibration	Left vibration	Right vibration	Left vibration
Normal subjects (n = 19)	4.424 ± 2.065	4.688 ± 2.633	−0.196 ± 1.287	0.685 ± 1.719
Spasmodic torticollis patients (n = 19)	1.578 ± 1.869	1.383 ± 2.362	−0.373 ± 0.783	0.034 ± 0.905
Vestibular patients (n = 11)	0.354 ± 1.129	0.348 ± 1.071	−0.057 ± 0.324	0.494 ± 1.191

Overall means ± SD are given.

Table 4 *Mean sway deviation (4 s vibration)*

Subjects	Head straight		Head to the right		Head to the left	
	Sagittal plane	Frontal plane	Sagittal plane	Frontal plane	Sagittal plane	Frontal plane
Normal subjects (n = 19)	2.314 ± 1.947	0.053 ± 0.434	1.65 ± 1.643	0.897 ± 1.464	1.606 ± 1.646	−0.812 ± 1.49
Torticollis patients (n = 19)	0.756 ± 0.820	0.178 ± 0.745	0.663 ± 0.786	0.205 ± 0.43	0.72 ± 0.855	−0.139 ± 0.277

Overall means ± SD.

This is shown graphically in Fig. 2, which includes the combination of frontal and sagittal sway as an oblique vector. ST patients' sway deviations were little affected by voluntary head turns.

Correlations of the amplitudes and directions of patients' pathological head turns and tilts with frontal and sagittal components of sway deviations showed that direction of head turn or tilt had no effect on sway direction.

Devation latency

Latency of postural deviation was estimated by averaging the responses to the 4-s epochs of vibration. The earliest component of the sway response to a 4-s vibration was a small backwards torque which could be detected above background in 18 out of 19 normal subjects and in 13 out of 19 torticollis patients. The latency of the backwards torque was 216 ± 17 ms in normal subjects and 219 ± 22 ms in torticollis patients (data for right-side and left-side neck vibration were combined). There was no significant difference in deviation latencies between patients and normal subjects.

Sway deviation in response to vibration of the Achilles tendons

Vibration applied to the Achilles tendons of both legs resulted in body sway backwards. Unilateral vibration induced an oblique sway deviation, which consisted of a backwards and frontal sway directed contralaterally to the stimulated side. For both bilateral and unilateral stimulation there was no significant difference in the sagittal sway deviation (mean deviation from the baseline in an average of five trials) between patients and controls. The frontal sway deviation in response to unilateral vibration tended to be smaller in patients than in normals ($P < 0.05$ for left-side vibration, non-significant for right-side stimulation).

Head movement induced by the neck vibration

Neck vibration in patients resulted in an involuntary movement of the head, detected visually in 10 ST and seven LD patients, which was not apparent in normals. Head movement during vibration was recorded in 12 ST patients by means of the FASTRAK during 35 s of vibration. In seven of these, the neck vibration induced a tilt of the head backwards which increased through the 35 s of stimulation eventually approaching a plateau which ranged from 4° to 16° (mean ± SD, 9.3 ± 3.7) (Fig. 3).

The amplitude of head tilt in response to right-side and left-side vibration was not related in a consistent way to the side of head turn in each particular ST patient or to the side of stimulation. In most patients, head tilt backwards was also accompanied by a turn and a lateral tilt movement, but these were inconsistent in magnitude and direction. The backwards head-movement response to vibration was not related to either the magnitude of sway path or the deviation.

Results of vibration in untreated ST patients

Two of the three patients with untreated ST had no sway deviation in response to either 35 s or 4 s of neck vibration. However, both had backwards head deviations (3°–10° and 10°) induced whilst standing. The third patient had no head deviation. She had had ST for 4 years and showed minimal

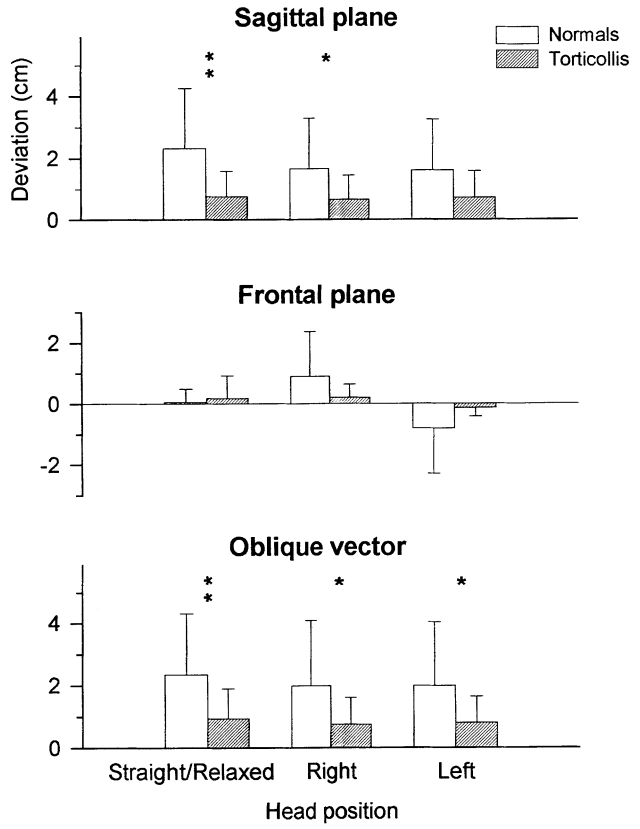

Fig. 2 Sway deviation in response to 4 s vibration. Normal data (head straight and turned to the right and left) are compared with those from botulinum toxin treated patients, with their pathological head turn ('relaxed') and with their head voluntarily turned right and left. 'Oblique' sway is the vector sum of sagittal and frontal plane sway. Responses to right and left sided neck vibration were similar for all conditions and have been combined in averages. Significant differences between normal subjects and patients are marked: $^{**}P < 0.01$; $^{*}P < 0.05$.

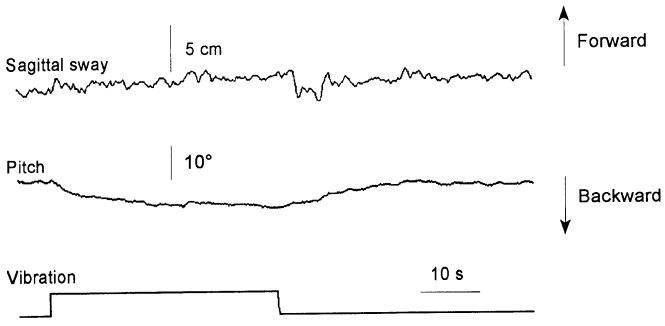

Fig. 3 Head deviation in a torticollis patient during a 35 s epoch of vibration of the posterior neck muscle. The traces show backwards head movement (pitch and sagittal sway).

sway deviations. The maximum deviation to 35 s stimulation was 2 cm of sagittal sway deviation for right sided vibration (<1 SD of the mean sway deviation in ST patients). The largest amplitude sway deviation evoked when averaging 4-s epochs of vibration was a 0.4 cm sagittal deviation when her head was turned to the right (within 0.5 SD of ST mean). Therefore, untreated patients also show little deviation of posture in response to neck vibration.

Discussion

The most significant finding of the study is that the standing posture of normal subjects deviates forwards when their neck is vibrated so that they are seen to be tilted, whereas ST and LD patients deviate little or not at all. Unlike normal subjects, ST patients respond to vibration with a neck extension. ST patients' sway responses were unrelated to their head deviations, showing a lack of directional specificity which has been noted before when testing torticollis patients on visuo-motor functions (Leplow and Stubinger, 1994). This is quite unlike normal subjects who, with the head turned sideways, acquired a sway deviation component in the frontal plane.

The paucity of sway deviation in ST patients was unrelated to overall 'stability' since quiet stance was similar in patients and normal controls (*see* also Straube and Dieterich, 1993). The sway deviation was smaller during both 35 s and 4 s of vibration. Since the latter response is probably less dependent on voluntary control the reduction of response is unlikely to be related to voluntary suppression.

The hypothetical explanation of why neck vibration induces sway deviation derives from the effect that vibration stimulates the primary endings of muscle spindles (Bianconi and van der Meulen, 1963; Burke *et al*., 1976) whose signals normally indicate muscle lengthening (Roll *et al*., 1989). Therefore, vibration of dorsal neck muscle simulates neck flexion. The central interpretation of the resulting postural changes is dependent on what body part is taken to be stationary. Cervical proprioceptive input is known to be processed together with vestibular afference to deduce an estimate of the head and trunk posture. In the case of a normal subject there is no concomitant signal from the labyrinth of head tilt when the neck is vibrated, so the brain interprets the neck signal to indicate that the body is moving forwards beneath a space-fixed head, with the neck as axis of rotation (Fig. 4). This 'illusory' tilt gives rise to a forwards sway response which is directed to returning the body to upright (so that the centre of gravity is restored to a position above the feet). Since these are not strong effects in ST patients, we must conclude that the rôle of neck input in the control of their standing posture is weakened.

To understand how such motor responses can occur consider that proprioceptive signals induced by a muscle vibration are always in conflict with other sensory inputs, and the brain has to resolve the conflict by estimating the likely body configuration based on the conflicting signals. For example, vibration applied to elbow flexors or extensors in a subject with forearm restraint results in an illusory extension or flexion of the elbow, although concomitant inputs from cutaneous and articular receptors indicate that the forearm remains stationary (Gilhodes *et al*., 1986). The subject would rather accept that the external restraint is

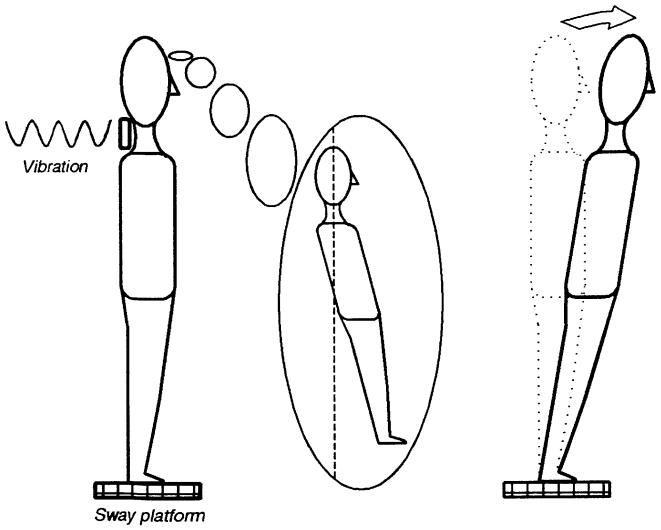

Fig. 4 Hypothetical explanation of the postural response to neck vibration based on the concept that sensory input is interpreted with respect to a postural schema. Vibration is applied to the dorsal neck surface of a blindfolded subject standing upright on a sway platform. The vibration activates selectively the muscle spindle receptors signalling to the brain that the neck muscles are lengthening. With no concomitant signal from the labyrinth, the brain interprets the signal as a movement of the body forwards beneath the space-fixed head. This illusion of backwards tilt gives rise to an involuntary forwards sway response which is directed to returning the body to upright.

moving together with the forearm than that the forearm remains stationary. Furthermore, vibration applied to elbow flexors gives perceptions of physically impossible arm positions, for example elbow extension beyond physiological limits (Craske, 1977). In another study (Brandt *et al*., 1977) an illusion of body rotation and nystagmus was induced when the arm of a stationary subject was passively rotated about a vertical axis in the shoulder joint, although there was no concomitant input from the labyrinth signalling head rotation. Similarly, with neck vibration, the absence of concomitant somatosensory signals from the ankle and feet signalling an alteration of the whole body posture does not suppress postural response to neck vibration, which arises from the central estimate of the body configuration.

The hypothetical explanation of minimal sway deviation in LD patients is along similar lines. In the LD subject there is no labyrinthine signal to corroborate or contradict the apparent neck flexion caused by neck muscle vibration. Accordingly, it is assumed that the head is tilting down on the chest and this is countered by extension of the neck.

That the neglect of an important source of proprioceptive input to spatial orientation in ST patients is specific to neck afference is shown by the fact that their response to Achilles-tendon vibration (‘vibration induced fall’; Eklund, 1972) was intact. Here it should be noted that the backwards-sway response to Achilles-tendon vibration is not a local reflex. The patterns of EMG in soleus, tibialis, biceps femoris and rectus femoris induced by vibration have long latencies and are typical of voluntary postural adjustments (Gurfinkel *et al.*, 1977). In addition, the vibration induces an illusion of forwards whole-body tilt in a standing subject as opposed to that of a local joint rotation in a sitting posture (Goodwin *et al.*, 1972; Gurfinkel *et al.*, 1977; Lackner and Levine, 1979). For these reasons it is thought that the response to Achilles vibration is a holistic postural adjustment due to an altered perception of orientation in space (Roll *et al*., 1993).

It is significant that, although vibration induces little overall body deviation in most ST patients, it did induce head movement. In normals, vibration of dorsal neck muscles or of the Achilles tendon induces different responses depending upon the postural context: neck vibration gives a whole body tilt if the subject is in a free standing posture, but a local response of head tilt if the torso is restrained; Achilles tendon vibration which gives a whole body deviation in a standing subject (and illusory whole-body tilt when the body is restrained) induces a local response, the tonic vibration reflex, in a sitting subject (and illusory dorsiflexion of the ankle when the actual movement is lacking). These findings show that central processing of proprioceptive afference depends on the postural context; specifically, what possible reference (e.g. ground, trunk, head) is taken to be stationary? Torticollis patients, relatively, seem to ignore cervical proprioceptive input in the whole body postural control although this input remains functional in the local control of the head on trunk.

Because our patients were almost all treated with botulinum toxin, it is possible that the explanation for some of the effects we have observed is that the intensity of afferent signals from the neck, as is seen in jaw muscle (Fillippi *et al*., 1993), is reduced after botulinum toxin injection. We attempted to control for this possibility by testing patients as soon as possible after injection; this, of course would not account for long-term changes induced by botulinum toxin. However, these fears may be unfounded for two reasons. First, the patients treated with botulinum toxin had robust local neck responses to vibration as shown by the backwards deviation of their head during stimulation which implies that neck afference was intact. Secondly, the small group of untreated patients in this study had typically small or absent sway-deviation responses to vibration, just like treated ST patients.

Conclusions and implications for vestibular dysfunction in torticollis

Our conclusion that neck afference in ST patients has a reduced role for the neck in spatial orientation is consistent with findings in recent psychophysical studies of orientation in ST patients (Anastasopoulos *et al*., 1996*b*). These have shown that ST patients with head tilts behave quite differently from normal subjects who assume head tilts when giving estimates of the ‘visual vertical’ or of the tilt orientation of their own face; the patients behave more like upright normal subjects who are ignoring the somatosensory signal of head deviation.

Mild disorders of the vestibular ocular reflex are known to occur in torticollis (Bronstein and Rudge, 1986; Stell *et al*., 1989). The reasons for this 'involvement' of the vestibular system in torticollis have never been clear since ST patients do not have serious vestibular disorder; they have robust vestibulo-ocular reflexes, perhaps with some dark asymmetry but certainly without oscillopsia, and can walk well in darkness showing no signs of vestibular ataxia. The similarity of responses to neck muscle vibration in ST and LD patients may give a clue as to why ST patients have mild vestibular dysfunction. The current physiological view is that neck and vestibular afference provide corroborative and complementary signals of motion of the body in space and, hence, of the relative motion of the head and trunk (Mergner *et al*., 1981). ST and LD patients are similar in that neither are able to relate neck input to labyrinthine signals. For the ST patient this may mean that his vestibular system lacks an important contextual input which provides verification and calibration. As a consequence, the vestibular system could accumulate mild inaccuracies which manifest themselves as the asymmetries of vestibular ocular reflexes reported in the literature.

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