# Neck muscle responses to abrupt free fall of the head: comparison of normal with labyrinthine-defective human subjects

Yatsuji Ito*†, Stefano Corna†‡, Michael von Brevern‡, Adolfo Bronstein‡, John Rothwell‡ and Michael Gresty‡§

*Department of Oto-Rhino-Laryngology, Gifu University School of Medicine, Tsukasamachi 40, Gifu 500, Japan, †Medical Centre of Rehabilitation, Instituto di Ricovero E Cura A Carattere Scientifico, Veruno, I-28010 Novara, Italy and ‡MRC Human Movement and Balance Unit, National Hospital for Neurology and Neurosurgery, Queen Square, London WC1N 3BG, UK

1. EMG responses from sternocleidomastoid (SCM) and orbicularis oculi were recorded in subjects who lay supine with their heads cradled in a sling. When the sling released abruptly, their heads fell freely. Normal and bilateral labyrinthine-defective subjects (LDs) were studied.

2. The normal response in SCM was a small burst of excitation at 22–25 ms latency, of 18 ms duration. This merged into a larger, later burst. The drop also produced eye blinks at 22–38 ms.

3. The onset of the SCM response in LDs was delayed (56–73 ms) even though the latency of their eye blinks was normal.

4. We conclude that the early response at ~22 ms in normal subjects is mediated by a vestibulocollic reflex. The delayed activity in LDs may be a stretch reflex. This is the first demonstration of the latency of the vestibulocollic pathway to natural stimulation in man.

Vestibular, cutaneous and stretch reflex inputs may all contribute to the control of neck muscle. However, the considerable inertia and complex geometry of the human head–neck system make it difficult to effect precise control of stimuli which move the head. As a consequence, the few studies which have attempted to identify sensorimotor pathways, for example, vestibulocollic or stretch reflex, have reported high variability in latencies and been unable to resolve uncertainties about the origin of stimuli responsible for neck muscle responses (Mazzini & Scieppatti, 1994; Kanaya, Gresty, Bronstein, Buckwell & Day, 1995). We report the findings from a technique which provides a way of exposing the head to a sharp onset, high acceleration which evokes reliable responses at short and consistent latencies. Comparison of normal subjects' responses to head drop with those of labyrinthine-defective subjects allows the vestibular component of the response to be identified.

## METHODS

The principle of the method is to drop the head, separately from the body, with a rapid release exposing the head to an approximately 1 *g* acceleration and thereby hyperextending the neck.

### Apparatus

The subjects lay prone and well-supported on a fixed horizontal platform which extended to the level of T1. The head, extended beyond the edge of the platform, in longitudinal alignment with the body, was supported by a cradle around the occiput (Fig. 1). The carry handles of the cradle were restrained from above by a Martin Baker® electromechanical aerial bomb release with $< 1$ ms release time.

Activation of the bomb release dropped the cradle which fell freely with the head until it was braked with a cushioned surface placed 10 cm below the initial level of the occiput.

Head drop was monitored with a precision piezo resistive DC-coupled accelerometer mounted with surgical tape on the forehead with sensitive axis aligned earth vertically.

§ To whom correspondence should be addressed.

This manuscript was accepted as a Short Paper for rapid publication.

Muscle activity in sternocleidomastoids (SCMs) was recorded with solid-state chloride surface electrodes with impedance typically 1 kΩ placed in differential pairs on the belly of the muscle at 5 cm separation. Eye blinks were recorded with a differential pair mounted supra- and infra-orbitally to record orbicularis oculi. Initial analog filtering of biological signals was performed with a 6 dB down passband of 32 Hz to 3·2 kHz.

EMG, accelerometric and trigger signals for the bomb release were acquired for processing at 1 kHz digitization rate.

### Protocol

For each drop 'trial' the subject was told to close his eyes and was dropped a variable length of time (up to half a minute) afterwards, to lessen predictability. Following the drop, the head was lifted back into the cradle which was reattached to the bomb release. Between each drop several minutes elapsed during which time the subject was encouraged to relax. Relaxation was apparent in the level of background EMG activity from the neck.

Ten drops were performed on each subject.

### Subjects

Ten normal subjects (eight males, two females; age range, 21–49 years) and three patients with defective labyrinthine function (LDs) gave their informed consent to the study according to the guidelines of the local ethics committee.

The three LDs tested were males aged 29, 48 and 34 years. The first subject lost vestibular function in childhood as a complication of meningitis and was deaf. The second lost labyrinthine function 4 years prior to testing in association with meningitis (*Streptococcus suis*) treated by potentially ototoxic medication and had bilateral moderate to severe hearing losses. The third subject had neurosarcoidosis with a severe right-sided hearing loss.

On clinical examination, all patients had saccadic ('broken up') compensatory eye movements and admitted to oscillopsia (visual instability due to absent vestibulo-ocular reflex compensatory eye movement) in response to rapid head shaking in pitch and yaw.

Nystagmus responses to 20 °C caloric irrigation to the external meati were absent. Steps of 80 deg $s^{-1}$ in rotational velocity and 80 deg $s^{-1}$ peak velocity sinusoidal rotation in yaw at frequencies up to 1 Hz failed to provoke nystagmus in the dark.

All values are means ± S.D.

## RESULTS

### Head movements induced by drop

Examples of the normal head movement response to drop are shown in Figs 1, 2 and 3. The head made a rapid entry into fall attaining a peak acceleration of 0·76 ± 0·1 *g* in 17 ± 4·0 ms. The period of negative acceleration lasted for 104 ± 22 ms until reversing to positive deceleration when the head is stopped by a combination of the cushion and, presumably, muscle contraction. Within the period of negative acceleration the pattern of acceleration fluctuated with wide intersubject variability. The head was not

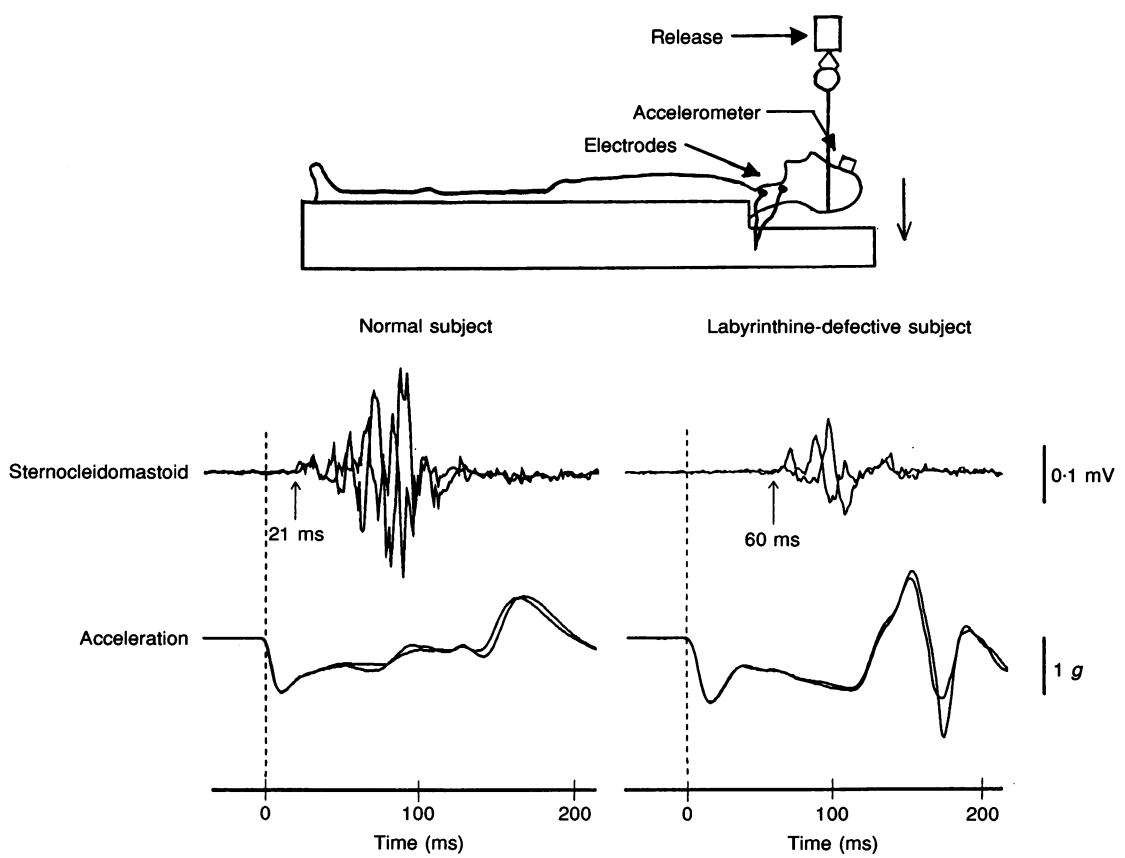


**Figure 1**

Apparatus and raw data records for head drop from a normal and LD subject.

allowed to fall until braked solely by muscle contraction because of safety considerations on neck hyperextension.

### Normal EMG response

On raw data records the EMG response to drop consisted of a small early burst of activity at ~20 ms which merged into a larger later burst of activity beginning at about 40–50 ms after the onset of the drop. The total response lasted upwards of 100 ms (Fig. 1). The same pattern could also be seen when the responses were rectified and averaged (Fig. 2). The initial part of the response remained consistent on repeated trials whilst the later part showed some adaptation (Fig. 3).

### Normal blink responses

In the two normal subjects that were tested, head drop also produced eye blink responses at latencies of 22 and 38 ms (Fig. 2). Unlike the initial part of the SCM response the blink rapidly adapted over several trials (Fig. 3).

### LDs head movement response

Head trajectories in normal and LD subjects were similar in onset (Figs 1, 2 and 3). As with normal subjects, the subsequent pattern of acceleration was variable. In the patients, peak head accelerations between 0·65 and 1 *g* were attained at mean latencies of 17–21 ms.

### EMG responses in LDs

An example of a raw EMG response to drop is given in Fig. 1. Latencies were measured on the averaged rectified EMG as shown in Fig. 2. On average, the earliest activity in SCM muscle in the three LD subjects was at 57, 59 and 73 ms (corresponding in time to the middle of the large burst of activity evident in normal subjects). The burst terminated at 160–180 ms. In contrast to the delayed SCM response the latency of the blink was normal (22 and 27 ms) in the two LD subjects in whom it was recorded (Fig. 3). As in normal subjects the blink rapidly adapted over several trials whereas the response in the neck muscle did not.

### Response consistency

The early part of the SCM response is physiologically significant in that it is the shortest latency response evoked by natural head movement that has been identified and probably involves a direct vestibular pathway to neck motor neurones. Accordingly we attempted to establish the automaticity and reliability of this response in four normal subjects exposed to four sessions of repeat testing spaced by several days. As shown in Fig. 4, the response latency was highly consistent with standard deviations of 2–3 ms, within and between subjects and from test session to test session.

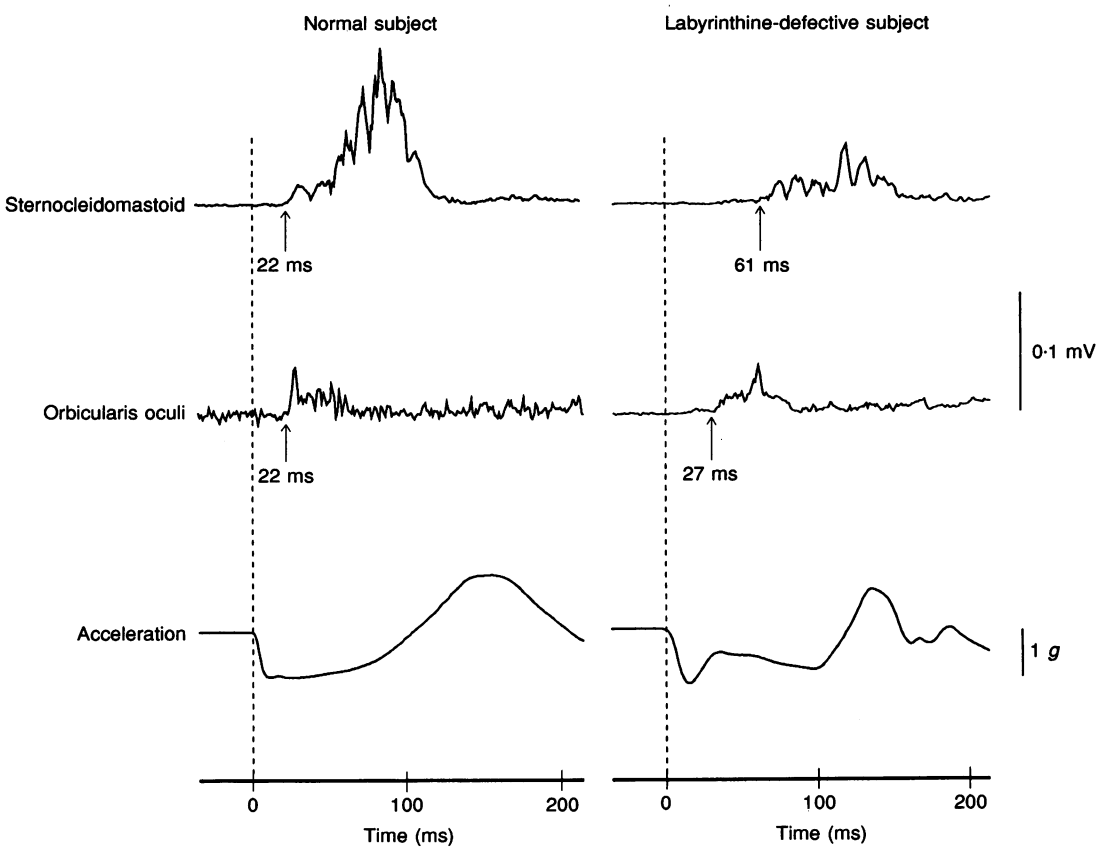


**Figure 2**

Averaged records of rectified neck EMG and blink responses to drop from a normal and LD subject.

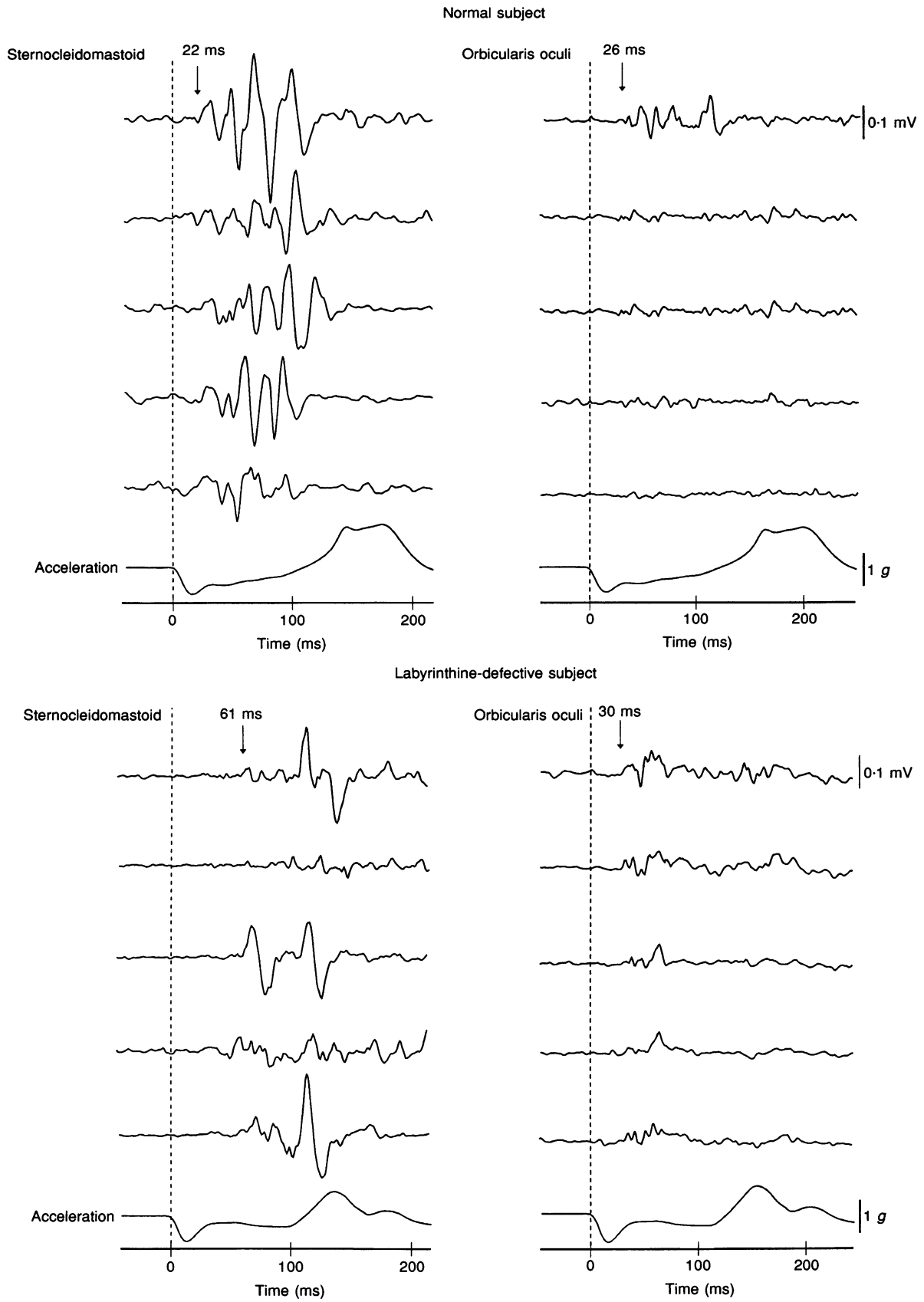


**Figure 3**

Responses of a normal and a LD subject to serially repeated drops showing adaptation of the response in SCM muscle in the normal subject and of the blink responses in both subjects.

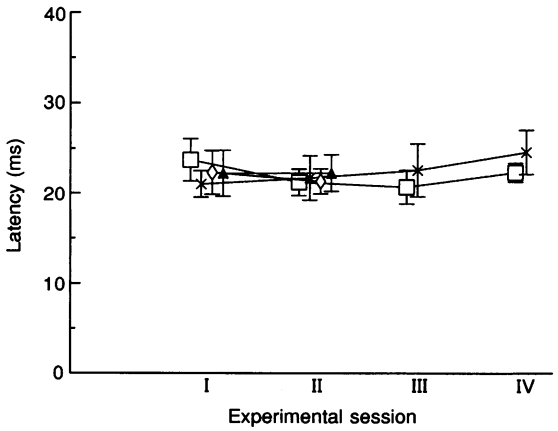


**Figure 4**
Latencies of the first vestibular response to head drop recorded in 4 normal subjects retested at 4 separate intervals. All values are the mean ± S.D. □, subject 1; ×, subject 2; ◇, subject 3; ▲, subject 4.

## DISCUSSION

The main physiological stimuli one expects the head drop to provoke are: (i) a combined linear and angular acceleration stimulus to the labyrinths, (ii) stretch of anterior neck muscle and shortening of muscle on the back of the neck, (iii) relative movement of neck articulations, (iv) a signal on C2 and C3 of the cessation of pressure on occiput as the cradle releases, and (v) transmitted mechanical disturbance via neck extension to sensory receptors in the thorax and thereon.

To understand the interaction of responses in this scenario it is important to note that the vestibulocollic and stretch reflexes would need to act synergistically to save the head from falling.

The long latency for neck EMG responses in LD subjects indicates that the shorter latency components of the responses of normal subjects originate in stimulation of the vestibular labyrinth. Activation of neck muscle at comparable short latencies has been demonstrated following direct mechanical stimulation of the vestibular apparatus by high intensity, short duration sound 'clicks' which evoke an inhibition of motor units followed by an excitation giving surface potentials P13 N23 (Colebatch & Halmagyi, 1992; Colebatch & Rothwell, 1993; Colebatch, Halmagyi & Skuse, 1994). This response is probably due to activation of a direct vestibulocollic pathway. The initial part of our response to head drop (in normals) may therefore be comparable to the N23 response to click. The natural stimulus used in the present experiments may account for the slightly longer latency.

As noted above, many types of sensory input (muscle, joint, cutaneous) may contribute to the late SCM response in LD subjects. Presumably, the same input occurs in normal subjects. We speculate that the complete response consists of an early vestibular reflex which merges into later (larger) activity evoked by input from many other receptors.

Colebatch *et al.* (1994) argue that the click-evoked responses in neck muscle are most likely to arise through stimulation of the sacculus since this has the lowest threshold to mechanical shock transmitted through the ear. The components of the labyrinth stimulated strongly by the head drop are the vertical semicircular canals, the utriculus and the sacculus and one must assume that all may partake in response generation. The labyrinthine signals at the onset of drop would be from the 'irregular' otolithic units in the VIIIth nerve: sensitive to rate of change of acceleration (Fernandez & Goldberg, 1976) and from the 'irregular' canal units which code angular head acceleration for high frequency stimuli (Fernandez & Goldberg, 1971).

The presence of normal latency blink reflex in LD subjects suggests that this part of the response is not dependent upon vestibular input. In view of its rapid adaptation to repeated stimuli it seems likely that the blink is part of the non-specific startle response provoked by a variety of inputs to a reticular startle generator (e.g. Bisdorff, Bronstein & Gresty, 1994). The blink response to whole-body fall can be preserved in LD subjects whence the input to the startle is thought to arise from sharp changes in tactile input as the body commences falling (Bisdorff *et al.* 1994). In our experiment the stimulus input to startle could be mediated via the sensory stimulus to dermatome C2 and C3 caused by the sudden change in forces on the skin over the occiput which occurs when the cradle is released.

The functional importance of the early small vestibular response is probably minimal. The main response commences at ~40 ms. Unfortunately the present experiments could not quantify the effectiveness of neck muscle activity in terms of head trajectory which would have been useful for quantitating the comparison between normal and LD responses. This was partly because we took the precaution to brake the head drop soon after fall, probably before full muscle braking was developed, and also because the wide intersubject variations in the

dynamics of head drop, as reflected in acceleration records, may have masked the effects of small muscle contractions.

## Conclusions

We have demonstrated for the first time activation of neck muscle in response to actual head motion in human subjects of the order of 20 ms which is of comparable latency to responses evoked by impulsive mechanical stimulation of the labyrinth and is most likely to be conducted by a direct vestibulocollic pathway. Later parts of the response (> 60 ms) may be produced by activation of many other types of receptor. These data provide a framework for understanding the neurophysiological organization of automatic sensorimotor modes of head control in human subjects.

BISDORFF, A. R., BRONSTEIN, A. M. & GRESTY, M. A. (1994). Responses in neck and facial muscles to sudden free fall and a startling auditory stimulus. *Electroencephalography and Clinical Neurophysiology* **93**, 409–416.

COLEBATCH, J. G. & HALMAGYI, G. M. (1992). Vestibular evoked potentials in human neck muscles before and after unilateral vestibular deafferentation. *Neurology* **42**, 1635–1636.

COLEBATCH, J. G., HALMAGYI, G. M. & SKUSE, N. F. (1994). Myographic potentials generated by a click-evoked vestibulocollic reflex. *Journal of Neurology Neurosurgery and Psychiatry* **57**, 190–197.

COLEBATCH, J. G. & ROTHWELL, J. C. (1993). Vestibular evoked EMG responses in human neck muscles. *Journal of Physiology* **473**, 18*P*.

FERNANDEZ, C. & GOLDBERG, J. M. (1971). Physiology of peripheral neurones innervating semi-circular canals of squirrel monkey. II. Response to sinusoidal stimulation on dynamics of peripheral vestibular system. *Journal of Neurophysiology* **34**, 661–675.

FERNANDEZ, C. & GOLDBERG, J. M. (1976). Physiology of peripheral neurones innervating otolith organs of the squirrel monkey. III. Response dynamic. *Journal of Neurophysiology* **39**, 996–1008.

KANAYA, T., GRESTY, M. A., BRONSTEIN, A. M., BUCKWELL, D. & DAY, B. (1995). Control of the head in response to tilt of the body in normal and labyrinthine-defective human subjects. *Journal of Physiology* **489**, 895–910.

MAZZINI, L. & SCHIEPPATTI, M. (1994). Short latency neck muscle responses to vertical body tilt in normal subjects and in patients with spasmodic torticollis. *Electroencephalography and Clinical Neurophysiology* **108**, 1–11.

## Acknowledgements

Dr Y. Ito was funded by a grant from Hayao Nakayama Foundation for Science and Technology and Culture. Dr S. Corna was funded by CEC Human Capital and Mobility Large Scale Facility Grant ERB CHGE CT93 0020. Dr M. von Brevern was a Margaret Ruth Dix Fellow

*Received 6 September 1995; accepted 12 October 1995.*