

SHORT REPORT

Click-evoked vestibular activation in the Tullio phenomenon

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Abstract

Click-evoked vestibulocollic reflexes were studied in a patient with a unilateral Tullio phenomenon (sound induced vestibular symptoms) and the findings were compared with those of a group of normal subjects. Compared with normal subjects, the reflexes elicited from her symptomatic side were large and had an abnormally low threshold, but retained a normal waveform. The click-evoked responses in this patient show changes consistent with her symptomatology and are indicative of a pathological increase in the normal vestibular sensitivity to sound.

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Patients with the Tullio phenomenon experience vestibular symptoms when they hear specific, usually loud, sounds. Reported precipitants include rumbling noises of traffic and trucks, noises of a crowded bazaar,¹ campanology (bell ringing),² car horns,³ and saying certain vowels.^{4,5} Clinically it is possible to induce nystagmus in these patients with sound, which, although loud, has no such effect in control subjects.

Very loud sounds (over 130 dB SPL) can cause vestibular symptoms in normal subjects,⁶⁻⁸ but usually these intensities are only achieved near jet engines or explosives. It is thought that, under these conditions, the sound pressure wave set up in the inner ear can directly activate vestibular receptors. By making the sound duration very short (clicks), these high sound intensities can be presented safely.⁹ Colebatch *et al*¹⁰ showed that loud clicks evoked a short latency EMG response in the contracting sternocleidomastoid muscles and presented evidence that this represented a vestibulocollic reflex, possibly originating from saccular afferents. Given these findings, it is natural to speculate whether the Tullio phenomenon simply represents an exaggeration of the normal condition (a lower threshold than normal for acoustic activation of the vestibular apparatus) or whether there might also be a more fundamental alteration in the nature of the vestibular activity induced by sound in these patients. We have recently had the opportunity of applying this new technique in a patient with an established Tullio phenomenon to consider this issue.

Subjects and methods

CASE HISTORY

The patient, a 55 year old woman, reported that she had experienced left retroauricular pain and impaired balance after a series of forceful sneezes six years previously. Pressure over the mastoid area or loud sounds made her feel unsteady and veer to the left, and also made her retroauricular pain worse. Associated with these symptoms was the illusion of objects "swimming" in front of her. Clinical examination of her eye movements as well as the remainder of the neurological examination were normal. The Hallpike manoeuvre was negative bilaterally. Loud tones (1 kHz, 110 dB ISO) presented to the left ear through earphones caused visible nystagmus. This consisted mainly of conjugate torsional left beating and down beating components.¹¹ Similar loud sounds presented to the right ear did not cause nystagmus. Pure tone audiograms, stapedial reflex thresholds, bithermal caloric tests, CT of the temporal bone, and cranial MRI were normal. The middle ear was normal when surgically explored (HL).

Click stimuli were delivered through earphones with the patient sitting.¹⁰ Surface EMG recordings were made over the right and left sternocleidomastoid muscles with a reference electrode over the sternoclavicular joint and the active electrode over the muscle belly (interelectrode distance 6-8 cm). Clicks 0.1 ms long were applied at 3/s pseudorandomly to either the right or left ear. The click generator was purpose built and calibrated to produce clicks from 70 dB to 100 dB above normal hearing threshold (0 dB NHL = 45 dB SPL for 0.1 ms click). In this report, sound intensities are measured with respect to (normal) hearing threshold as this is the common method by which commercial click generators are calibrated. It is important to note that impulse noise intensity is formally defined in dB SPL⁹ and this physical rather than perceptual intensity is the more relevant to the effects on the vestibular apparatus.

The patient was instructed to activate her neck flexors tonically. Averages of both unrectified and rectified EMG were made (n = 256) with a laboratory interface and associated commercial software (SIGAVG Ver 6.0, Cambridge Electronic Devices, Cambridge, UK). The patient's results were compared with those of 25 normal volunteers (ages 22 to 65) tested under similar conditions but with a commercial click generator

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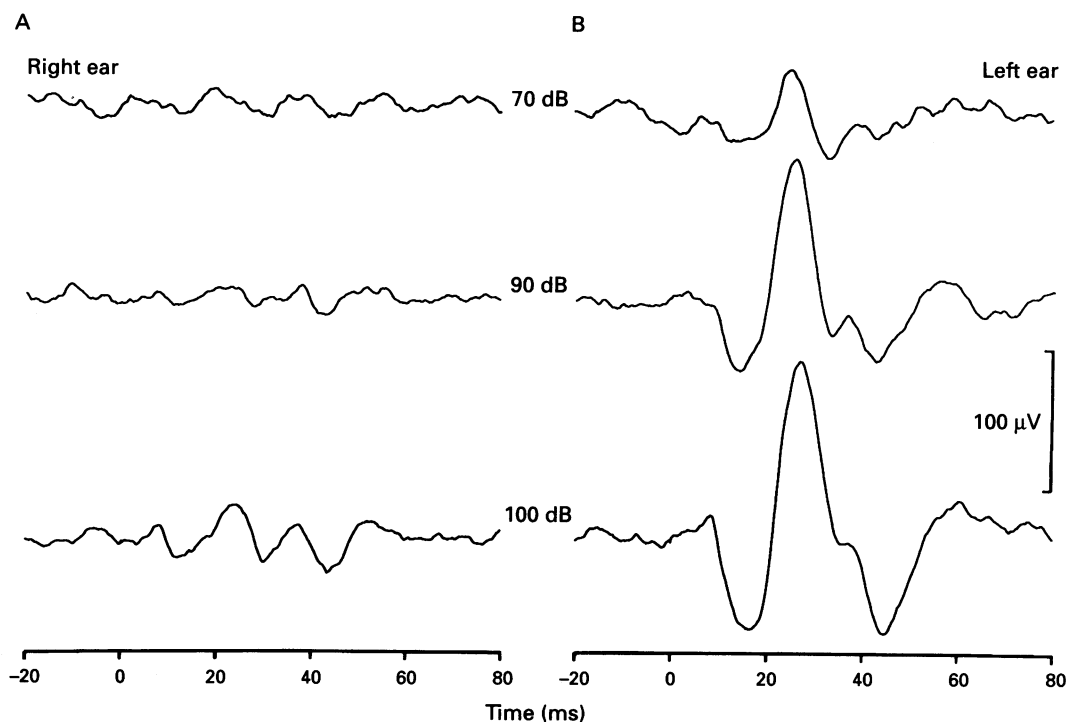
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Recordings made from over the patient's right sternocleidomastoid (A) and left sternocleidomastoid muscle (B). In each case, the averaged effects of stimulating the ipsilateral ear are shown. The effects of unilateral clicks of three different intensities are illustrated. Click intensities are given relative to the reference intensity (45 dB SPL: see text). All averages were made of the effects of 256 presentations of the click stimulus which was given 20 ms after the traces began, at time = 0. Positive potentials at the active electrode caused a downward deflection. When the symptomatic (left) ear was stimulated, a clear p13-n23 response was visible with lowest intensity used (70 dB), a lower level than for any of the normal subjects tested. By contrast, the p13-n23 response on the right side was only first seen with 90 dB clicks.



(ST10: Medelec, Surrey, UK). Peaks of the evoked waveforms have been described with the nomenclature of Colebatch *et al* in which the p13-n23 biphasic wave in the unrectified average is specifically generated by vestibular afferents.¹⁰ The size of the p13-n23 response is proportional to the level of tonic muscle activation¹⁰ and to allow for this reflex amplitudes have also been expressed as a ratio of the average level of the (prestimulus) rectified EMG.

Results

The responses evoked by stimulation of the patient's right and left ears were different (figure) and the table summarises these. The vestibular dependent p13-n23 response was present on the left side with the lowest inten-

sity stimulus (70 dB) and increased in amplitude and saturated with 90 dB clicks. Peaks consistent with n35 and p44¹⁰ were present after the more intense stimuli. The p13-n23 response on the right side began with 90 dB clicks and increased in size with louder clicks but was always much smaller than on the left. The latency of the initial positivity was slightly longer on the left than on the right (16.6 *v* 12.2 ms). There was no sensation of vertigo or visible eye or head movement during click testing.

All the normal volunteers had p13-n23 responses when given stimuli at 100 dB but none had responses at 70 dB (mean threshold: right 87 dB, left 86 dB). The two normal subjects with the lowest thresholds had small responses to 75 dB clicks, with corrected amplitudes smaller (0.35–0.51) than those seen in the patient with 70 dB clicks. The size of the patient's left sided response, corrected for background activity, was also larger than that in any control subject at an intensity of 80 dB. At intensities of 90 dB and higher, p13-n23 responses similar in amplitude to those of the patient were sometimes found in normal subjects: thus for 100 dB clicks, corrected amplitudes over 2.37 (the maximum size of the patient's response) were recorded from three normal volunteers (four ears). The threshold difference between the ears for eliciting the p13-n23 response was never greater than 10 dB in the normal volunteers.

Click induced vestibulocollic reflexes in left Tullio phenomenon

Intensity (dB NHL)	Right			Left		
	p13/n23	Rect EMG (µV)	Ratio	p13-n23	Rect EMG (µV)	Ratio
70	—	—	—	51.2	68.9	0.74
80	—	—	—	79.7	59.8	1.33
90	12.7	72.2	0.18	152.2	69.4	2.19
95	24.9	81.1	0.31	191.7	88.5	2.17
100	37.9	82.6	0.46	191.3	80.7	2.37

The intensity of the clicks (0.1 ms duration) is given in the left column where 0 dB NHL = 45 dB SPL. The three columns thereafter detail the vestibulocollic reflexes recorded from the right sternocleidomastoid muscle in response to clicks given to the right ear. The last three columns give the same results for clicks given to the left ear, using recordings made over the left sternocleidomastoid. The peak to peak amplitude of the p13-n23 response¹⁰ is given first, then the mean rectified EMG, and finally the size of the response as a ratio of the background EMG activity. The threshold and amplitudes on the right side were within the range of the normal controls. On the left, the response began at a lower threshold than normal and was correspondingly larger. Correction for background activity indicated that the size of the response on the left became saturated at 90 dB.

Discussion

Colebatch *et al*¹⁰ presented evidence, based on changes seen in patients with selective 8th nerve lesions, that the initial positive-negative

potential (p13-n23) seen in recordings from over the sternocleidomastoid muscle after loud clicks to the ipsilateral ear was the result of vestibular activation. Consistent with such a conclusion, we found that our patient with the Tullio phenomenon had a pathologically low threshold for this response when sound was applied to the symptomatic ear. Our estimate for the threshold for click activation of the vestibular apparatus in this ear, of the order of 110 dB SPL, is at least 5–10 dB lower than that in the most sensitive normal subjects and is consistent with the sound level required to induce nystagmus (110 dB ISO \approx 117 dB SPL at 1 kHz).¹² Her threshold for click activation of the vestibular apparatus on the right was normal, so it is likely that the process that resulted in her developing the Tullio phenomenon had the net effect of increasing her vestibular sensitivity to sound by over 20 dB (more than a 10-fold increase in sensitivity). The process by which this occurred is uncertain, but seemed not to be the result of dislocation of the stapes (compare Dieterich *et al*³).

At the higher intensities, the click induced vestibulocollic reflex on the left increased in amplitude and became saturated. Even with stimuli more than 30 dB above the patient's threshold—a relative intensity not achievable in normal subjects—the form of the evoked response remained the same as for normal subjects: indeed, the reflexes evoked by 100 dB clicks were indistinguishable from those occurring in some normal subjects after the same stimuli. Thus no evidence was found to

suggest a fundamental change in vestibular response to sound in addition to its increased sensitivity.

Click activation of the vestibular apparatus can indicate pathologically increased sensitivity to sound in cases of the Tullio phenomenon.

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