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Tullio's phenomenon

N. Deggouj, S. Castelein and M. Gersdorff

Cliniques universitaires Saint-Luc, Brussels, Belgium

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Abstract. *Tullio's phenomenon.* Tullio's phenomenon corresponds to a pattern of sound-induced subjective and objective responses. The subject may feel sensations of unsteadiness, imbalance or vertigo, associated with disturbances of oculomotor and postural control. Tullio's phenomenon is provoked by very loud sound and should be considered physiological. It is pathological if it is provoked by normal sounds. Changes to the functioning and/or the morphology of the labyrinth should be looked for in patients with the pathological form: decreased thresholds for the acoustically evoked vestibular potentials, semicircular canal dehiscence, traumatic lesions of the labyrinth, ligament hyperlaxity.

Introduction

Tullio's phenomenon (TP) is a pattern of sound-induced imbalance symptoms, motor responses of the eyes (nystagmus), head (myogenic responses) and other spinal neuron synkinesis (postural sway).[1,2] It may be physiological or pathological.

Physiological TP

Very loud sounds (250-500-1000 Hz or clicks at 110 dB) applied mono-aurally (but not binaurally) may elicit postural responses in normal subjects.[1,3,4] This physiological Tullio's phonomenon results in postural sway, increasing with closed eyes, when recorded with posturographic techniques.

It also induces myogenic responses in the sterno-cleido-mastoidian muscles, when recorded with vestibular evoked myogenic potential (VEMP) techniques.[1,4-7] Those vestibulocollic responses (provoked by a physiological TP) are thought to be useful in exploring saccular function.

Pathological TP

This form corresponds to a vestibular hypersensitivity to sound, resulting in perceived vertigo or unsteadiness. The otolothic-like symptoms are elicited with less loud sounds, <70 dB nHL for clicks,[4,5] or by loud pure tones presented binaurally.

Normal sounds provoke acute modifications in oculomotor control, leading to nystagmus. They disturb the postural responses, inducing feelings of unsteadiness and increased postural sway. The posturographic recordings may show a fall in the vestibular and composite postural scores when subjects are exposed to loud sounds. This fall is not observed in neuro-otological patients without a history of TP, or in normal subjects.[3]

Acoustically-evoked vestibular potentials have lower thresholds and increased amplitudes. On the other hand, galvanic-evoked vestibulocollic responses present normal thresholds. It is therefore supposed that sound hypersensitivity in subjects with TP is likely to occur distally to the vestibular nerve.[4]

The mechanisms by which acoustic stimuli act on the vestibular end organs remain unclear. Studies of animals have shown that afferents from all the vestibular end organs could respond to acoustic stimuli.[8] However, some pathological changes lower thresholds, resulting in sono-vestibular symptoms.[8]

Aetiology

The superior semicircular canal dehiscence syndrome is a newly recognised syndrome characterised by vertigo and nystagmus (torsional and down beating) induced by sound (= TP), or pressure changes in the middle ear (= Hennebert sign), or intracranially.[4,5,8,9] The dehiscence renders the canal particularly sensitive to sound and pressure changes, probably because it works like a third window, allowing larger volume, pressure, deflections and displacements at the level of the canal.

Other pathological modifications may be involved in TP. A pathological contiguity of the tympano-ossicular chain and membranous labyrinth may be associated with it. It is observed in cases of dislocated ossicular chain, stapes hyperlaxity, fracture of the footplate or of the labyrinth, fibrotic damping of the ossicular chain, fibrosis of the inner ear, traumatic labyrinth, perilymphatic fistula and endolymphatic hydrops.[1,3-14]

The distance between the footplate and the utriculus is only 0.5 mm in the posterior part of the oval window.[11] Membranous connections exist between the utriculus and the footplate in 25% of subjects.[11] An increase in those connections may favour sonovestibular hypersensitivity.

Diagnosis

When a subject presents symptoms of sonovestibular hypersensitivity, the TP diagnosis must be considered. It is confirmed by different tests like a positive Hennebert sign, sound-induced nystagmus, sound-induced postural responses recorded with posturographic tests and falls in VEMP thresholds.

A morphological modification of the middle and inner ear must be considered. Hyperlaxity may be suspected in cases where there is a fall in the resonance frequency of the ear. A CT scan of the temporal bone should show any semi-circular canal dehiscence.

Conclusion

Pathological Tullio's phenomenon is characterised by subjective and objective sonovestibular symptoms resulting from abnormal hypersensitivity to normal sounds of the vestibular end organs secondary to morphological changes in vibration and pressure transmission between the external and the inner ear.

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Pr. N. Deggouj
Cliniques UCL - Saint-Luc
ENT Department
avenue Hippocrate 10
B-1200 Brussels, Belgium
E-mail: Naima.Deggouj@uclouvain.be