Dear Sirs

We read with great interest the article by Wenzel and colleagues (1) published in your journal. The authors describe four patients with bilateral vestibular hypofunction, sound and/or pressure-evoked nystagmus and normal hearing thresholds, in the absence of an ulterior vestibular disorder. The authors suggest that this syndrome may represent the clinical correlate of a pathological diagnosis termed vestibular atelectasis - a collapse of the semicircular canal walls protruding within the canal lumen that was described by Merchant & Schuknecht in eight patients (2).

We are grateful to Wenzel et al. for acknowledging in the letter attached to their article that the syndrome of the Tullio phenomenon with bilateral vestibular failure, a pure horizontal nystagmus in response to sound, normal hearing, and the absence of semicircular canal dehiscence was previously described in a single patient by Acierno et al. in 1997 (3), and in 2012 by Kaski et al. in three patients that underwent thorough audiovestibular testing (4).

Whilst the possibility that the Tullio phenomenon with bilateral vestibular hypofunction is related to vestibular atelectasis is an attractive hypothesis, we believe there is insufficient evidence to substantiate this claim.

Firstly, as Wenzel et al. indicate, only five of those 8 subjects with pathologically-confirmed VA had accompanying clinical histories of dizziness with reduced vestibular function. Indeed, the clinical hallmark of the cases reported by Wenzel et al., Kaski et al., and Acierno et al. (i.e 8 cases in total) is the Tullio phenomenon, not described in patients with VA.

Secondly, 3/5 patients with VA reported paroxysmal vertigo, most (2/3) with complete resolution, rather than progressive or continuous disequilibrium or oscillopsia that one would expect with bilateral vestibular hypofunction.

Thirdly, 4/5 patients with pathologically-confirmed primary vestibular atelectasis had unilateral disease, whereas all patients reported by Wetzel et al. and Kaski et al. had bilateral vestibular hypofunction.

In summary, whilst the clinical syndrome described by Wetzel et al. is now well-defined and consistent with prior case descriptions, we believe that further clinic-pathological correlation is required to substantiate the proposal that the Tullio phenomenon with horizontal sound/pressure-induced nystagmus, bilateral vestibular hypofunction and normal audiometry relates to vestibular atelectasis. One difficulty is that the original report of VA used limited retrospective clinical data, although it is perhaps unlikely the Tullio phenomenon would go unnoticed. Further research will thus be required to delineate the pathological correlate of this syndrome.

References

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