High jugular bulb in a cohort of patients with definite Ménière’s disease

Dear Sirs,

We read the article by Redfern et al.,1 entitled ‘High jugular bulb in a cohort of patients with definite Ménière’s disease’, with great interest. This study supports the findings of the recent study by Park et al.,2 which focused on the same topic using a different methodology. Both studies show an increase of jugular bulb abnormalities in Ménière’s disease compared with the asymptomatic side1 or a control group.2 This approach provides a new means to understand Ménière’s disease physiopathology. In addition, the findings have clinical consequences that are worth knowing by the surgeon. Indeed, as explained by Redfern et al., some Ménière’s symptoms have already been cured by surgical3,4 or endovascular5 treatments of jugular bulb abnormalities. Nevertheless, jugular bulb abnormalities – even with inner-ear or aqueduct dehiscences – are asymptomatic in nearly half of the cases.6

It is thus important to differentiate between those jugular bulb abnormalities that are responsible for the Ménière’s-like syndromes and those which are fortuitous associations. To do so, the surgeon must look for four clues which indicate that a jugular bulb abnormality is symptomatic: (1) vertigo may be induced by high venous pressure (coughing, Valsalva manoeuvre) or sound (i.e. Tullio phenomenon);7 (2) tinnitus may be pulsatile (in 33 per cent of cases);5,7 (3) a nystagmus may be identified when performing vestibular nystagmography during the Valsalva manoeuvre;8 and (4) vestibular evoked myogenic potential may show a threshold below 70 dB (in 63 per cent of cases).5,6 These low thresholds (as in superior canal dehiscence) result from a third window phenomenon,8 they differ from typical Ménière’s disease, which exhibits no vestibular evoked myogenic potential responses despite a stimulus of 100 dB (in 54 per cent of cases).9

In conclusion, this study by Redfern et al. reminds us that Ménière’s disease is, by definition, idiopathic, which means its physiopathology is not completely understood. It is likely that different mechanisms are hidden within the ‘idiopathic’ element, including symptomatic dehiscence induced by jugular bulb abnormalities. It is therefore important that screening for symptomatic jugular bulb abnormalities is conducted and a specific treatment proposed.

References
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