Vestibular Myogenic Evoked Potentials in Patients With Benign Paroxysmal Positional Vertigo

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To the Editor,

Benign paroxysmal positional vertigo (BPPV) is a recurrent balance disorder attributed to the appearance of lithiasic material from the otolithic maculae of the saccule and the utricle in the semicircular canals. The paper published by Boleas-Aguirre et al[1] presents the results obtained from the registration of vestibular evoked myogenic potentials (VEMP) in 19 patients with BPPV of the posterior semicircular canal (PSC). Although the series is limited, it is significant that the recording of both ears is absent in 5 individuals, attributed to their great age, as is the absence of any ipsilateral recording in another 4 cases.

The study describes a lack of response in 52% of the ears of patients with BPPV. Although caloric tests were performed on these individuals, the results are not described.

If we assume that these individuals had a normal caloric response (as indicated in the inclusion criteria) and did not present any transmission hearing loss (that might justify the absence of VEMP), these findings raise a number of questions of the greatest interest in the physiopathology of BPPV: *a*) some individuals with clinical symptoms of BPPV might have inferior vestibular neuritis diagnosed by means of VEMP, although we do not know if the neuritis might have developed prior to their BPPV[2]; *b)* the observation of a positional nystagmus attributed to lithiasis of the PSC necessarily implies that the paresia of the inferior vestibular nerve would be incomplete; and *c)* the positional nystagmus originates in other causes, such as lithiasis in other canals[3] or the loss of the inhibiting afferences of the saccule on the PSC secondary to the loss of inferior vestibular nodal neurons.[4] Thus, the experimental denervation of the saccule, maintaining the innervation of the posterior semicircular canal, caused an upward vertical nystagmus with a rotating component in cats.[4]

The study by Boleas-Aguirre et al[1] indicates that the dysfunction observed in the saccule might explain the postural instability seen in patients with BPPV after their vertigo is resolved and the nystagmus has disappeared, although it has not described the relationship between this post-therapeutic instability and the absence of response in the VEMP.

Furthermore, the otolithic dysfunction has been attributed to the utricle after assessment of the otolithic-ocular reflex by utricular stimulation using excentric rotation.[5]

The answers to the questions raised might be found through a study monitoring both the caloric response and the VEMP in patients with BPPV over time, as it is possible that the paresia of the superior and inferior vestibular nerves may develop gradually and independently of the semicircular canal affected.

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