



Hyperventilation-Triggered Vertigo and Nystagmus in Vestibular Paroxysmia

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Dear Editor,

Vestibular paroxysmia (VP) is a rare vestibular disease characterized by brief attacks of spinning or nonspinning vertigo that last from around 1 second to a few minutes.¹⁻⁵ Most attacks occur spontaneously, but they can be induced by turning the head to the right or left in the upright position. Arteries (or veins in rare cases) in the cerebellopontine angle are the pathophysiological cause of a segmental, pressure-induced dysfunction of the eighth nerve (primary VP). Cranial MRI should be performed to exclude the presence of other causes such as a tumor in the area of the cerebellopontine angle, arachnoid cysts, megalodolichobasilaris, brainstem plaques in multiple sclerosis, brainstem infarctions, or other brainstem lesions (secondary VP). A previous study found that 3 minutes of hyperventilation induced transient nystagmus in 70% of the patients with VP.² However, the phenomenon was not considered the manifestation of a VP attack, instead being attributed to hyperirritability of the compressed vestibular nerve. We report a patient presenting with hyperventilation-triggered typical attacks of paroxysmal vertigo and nystagmus in VP.

An 81-year-old female presented with a 3-day history of episodic vertigo. Each attack lasted about 10 seconds, and she experienced more than 10 attacks daily. Video-oculography revealed no spontaneous or gaze-evoked nystagmus. Horizontal head oscillation, vibratory stimulation, and positional maneuvers did not induce nystagmus. Hyperventilation for 30 seconds provoked the same attack of paroxysmal vertigo with left-, down-, and counterclockwise-beating nystagmus for 14 seconds (Supplementary Video 1 in the online-only Data Supplement). The findings of video head-impulse tests, cervical and ocular vestibular-evoked myogenic potentials, and pure-tone audiometry were normal (Fig. 1). The findings for MRI of the internal auditory canal with contrast enhancement were similarly unremarkable, and constructive interference in steady-state images did not reveal arterial compression of the vestibulocochlear nerve. The attacks of vertigo and hyperventilation-induced nystagmus disappeared after administering 300 mg of oxcarbazepine twice daily. After discontinuing the medication 6 months later, the patient did not report episodic vertigo.

Our patient experienced recurrent stereotypical vertigo lasting for seconds that had no explainable lesions in MRI of the internal auditory canal, and this stopped after oxcarbazepine treatment. All of these findings fulfill the diagnostic criteria for primary VP.⁶ Remarkably, our patient demonstrates that typical attacks of VP (paroxysmal vertigo and nystagmus) can be triggered by hyperventilation, and this was the only objective finding of VP. This finding contrasts with previous reports of patients with VP mostly having abnormalities in neurophysiological investigations and/or neuroimaging studies.^{2,3,7,8} The present case highlights the importance of performing a hyperventilation test to confirm VP, even in the absence of evidence from electrophysiological and neuroimaging studies.

The hyperventilation-induced nystagmus in our patient could be attributed to improved axonal conduction in the partially demyelinated vestibular nerve due to vascular compres-

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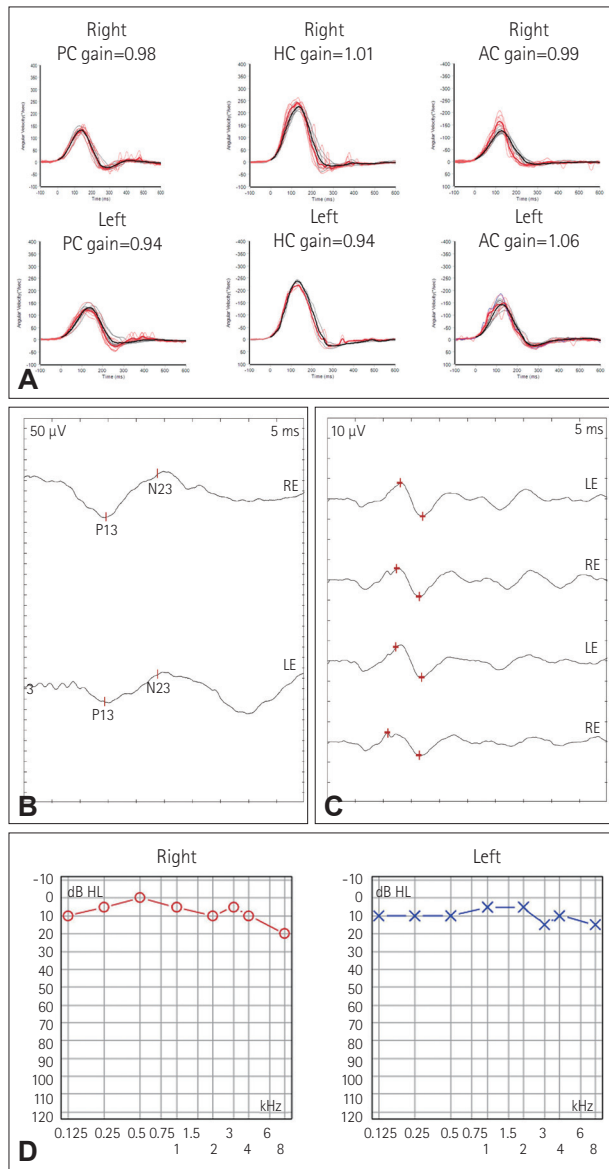


Fig. 1. Laboratory and radiological evaluations of the patient. The findings of video head-impulse tests (A), cervical and ocular vestibular-evoked myogenic potentials (B and C), and pure-tone audiometry (D) were normal. MRI of the internal auditory canal with contrast enhancement (E) and constructive interference in steady-state images (F) did not reveal other causes or arterial compression of the vestibulocochlear nerve. AC: anterior semicircular canal, HC: horizontal semicircular canal, LE: left-ear stimulation, PC: posterior semicircular canal, RE: right-ear stimulation.

sion. This effect has been also described in other diseases that damage the vestibular myelin, such as vestibular schwannoma.^{9,10} The left-beating, downbeating, and counterclockwise torsional nystagmus induced by hyperventilation would be ascribed to the transient excitation of the left superior division of the vestibular nerve innervating the anterior and horizontal semicircular canals.

Supplementary Video Legend

Video 1. Hyperventilation induced left-, down-, and counterclockwise-beating nystagmus.

Supplementary Materials

The online-only Data Supplement is available with this article at <https://doi.org/10.3988/jcn.2020.16.3.507>.

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Conflicts of Interest

The authors have no potential conflicts of interest to disclose.

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