

Response to Letter by Dr. Cristina Brandolini

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Dear Sir:

We appreciate the interest of Dr. Cristina Brandolini and co-workers in our paper¹ on the audiovestibular loss of a vascular cause. We agree that vascular mechanisms and sensitivity to ischemic damage may differ between the vestibular and cochlear organs. In line with our previous report,² they reported that cochlear symptoms, such as hearing loss and tinnitus, may be warning signs of an impending ischemic disturbance.³ The hearing loss and tinnitus reported by Dr. Cristina Brandolini and co-workers may result from ischemia to the cochlea due to systemic cardiovascular hemodynamic impairment (i.e., not vascular occlusion), although they did not present convincing evidence supporting this assumption. However, in our previous report,² hearing loss and tinnitus as initial manifestations of posterior circulation ischemic stroke may have resulted from ischemia to the cochlea owing to anterior inferior cerebellar artery (AICA) territory ischemic disease (i.e., vascular occlusion). The inner ear is particularly vulnerable to ischemia as it completely lacks collateral circulation, unlike the other structures supplied by AICA.^{4,5}

Their second suggestion on the vulnerability of each organ (i.e., cochlea or vestibule) to ischemic injury deserves careful consideration. In the guinea pig, transient occlusion of the internal auditory artery completely inhibits auditory function within several minutes and causes irreversible degeneration of the cochlea, but the vestibular end organ is relatively more resistant.⁶ On the contrary, clinical studies have suggested that episodic vertigo, not hearing loss or tinnitus, is the most common manifestation of transient ischemia within the vertebrobasilar circulation.⁷⁻⁹ Moreover, isolated episodic vertigo may be the sole manifestation of transient ischemia within the vertebrobasilar circulation.^{7,8} In the rarely occurring rotational vertebral artery syndrome, tinnitus develops several seconds

after the onset of vertigo and nystagmus,¹⁰ suggesting that the vestibular system is more sensitive to ischemia than is the cochlear system.⁹ In summary, multiple vascular mechanisms have been proposed for producing the vestibular and/or cochlear symptoms stemming from ischemic damage to the inner ear, and the issue of which structures are more sensitive to ischemia in humans is still unclear and requires further elucidation.

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