Orthostatic Intolerance in Acute Vestibular Neuritis

To the Editor: Orthostatic intolerance is often related to excessive pooling of blood in veins below the heart. The vestibular system mediates peripheral vasoconstriction, which can modulate venous pooling during posture changes.[1-3] No previous study has focused on postural cardiovascular responses in acute vestibular neuritis (AVN). We present evidence of vestibular autonomic hypofunction in AVN.

Case 1

A 35-year-old woman had acute onset of continuous, prolonged vertigo, nausea, vomiting, and gait unsteadiness, consistent with AVN. At the onset of symptoms, her only medication was fexofenadine. Nine days after onset of symptoms, her vertigo had resolved, but she reported postural light-headedness. Her supine blood pressure was 117/73 mm Hg, and her heart rate was 85 beats/min (Table). On standing, her blood pressure was 122/78 mm Hg, her heart rate was 109 beats/min, and she had orthostatic light-headedness that intensified over 1 minute. Passive rightward head thrusts elicited corrective saccades. Hearing was normal. Caloric tests revealed 41% right-sided paresis. Twenty-three days after symptom onset, her supine blood pressure was 99/64 mm Hg, and her heart rate was 80 beats/min. After standing for 4 minutes, her blood pressure was 100/68 mm Hg, her heart rate was 89 beats/min, and she had minimal light-headedness.

Case 2

A 52-year-old woman had acute onset of continuous, prolonged vertigo, nausea, vomiting, and gait unsteadiness, consistent with AVN. At the onset of symptoms, she was not taking any medications. Five days after onset of symptoms, her vertigo had resolved, but she reported postural light-headedness. Her supine blood pressure was 114/79 mm Hg, and her heart rate was 48 beats/min. On standing, her blood pressure was 116/79 mm Hg and her heart rate was 79 beats/min. Two minutes after standing, she reported feeling hot and sweaty. Her blood pressure was 108/84 mm Hg, and her heart rate was 81 beats/min. Three minutes after standing, her blood pressure was 109/82 mm Hg, her heart rate was 89 beats/min, and she asked to lie down. She subsequently felt better, and her blood pressure and heart rate were 115/74 mm Hg and 47 beats/min, respectively. With fixation eliminated, spontaneous right-beating nystagmus was observed. Hearing was normal. Caloric testing revealed 36% left-sided paresis. Eighteen days after symptom onset, her orthostatic light-headedness had resolved.

TABLE. Evolution of Vital Signs in 2 Patients With Acute Vestibular Neuritis

Time after symptom onset (days)	Supine BP (mm Hg)	Lowest standing BP (mm Hg)	Supine HR (beats/min)	Highest standing HR (beats/min)	Change in HR from supine to standing (beats/min)
Case 1					
9	117/73	122/78	85	109	24
23	99/64	100/68	80	89	9
Case 2					
5	114/79	108/84	48	89	41
18	116/81	108/82	50	73	23

BP = blood pressure; HR = heart rate.

Discussion

Both patients met criteria for AVN and manifested symptomatic postural tachycardia that abated as their condition improved. Acute vestibular neuritis affects normal sympathetic autonomic function, as documented by impaired cold pressor test responses.[4] In one study, no association was found between caloric responses and tilt test results, but all patients were tested more than 1 week after onset of symptoms, allowing a period of vestibular and autonomic compensation before laboratory testing.[5] In contrast, our patients were seen while acutely symptomatic, indicating limited compensation.

The otolith organs, which sense head position relative to gravity, regulate muscle sympathetic nerve activity.[1,3] We speculate that AVN, which often involves otolith organ function, may result in loss of normal graviception, leading to reduced muscle sympathetic nerve activity while upright and pooling of blood in veins below the heart. If further studies confirm vestibular autonomic hypofunction in AVN, potential clinical interventions might include intravascular volume repletion, medication adjustments during compensation, and avoidance of sudden postural changes.

Acknowledgments

Dr Whitman appreciates the support of the Massachusetts Eye and Ear Infirmary and Harvard Medical School through the Eleanor and Miles Shore 50th Anniversary Fellowship Program for Scholars in Medicine.

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1. Yates BJ, Bolton PS, Macefield VG. Vestibulo-sympathetic responses. *Compr Physiol*. 2014;4(2):851-887.

2. Yates BJ, Bronstein AM. The effects of vestibular system lesions on autonomic regulation: observations, mechanisms, and clinical implications. *J Vestib Res*. 2005;15(3):119-129.
3. Carter JR, Ray CA. Sympathetic responses to vestibular activation in humans. *Am J Physiol Regul Integr Comp Physiol*. 2008;294(3):R681-R688.
4. Jáuregui-Renaud K, Hermosillo AG, Gómez A, Márquez MF, Cárdenas M, Bronstein AM. Vestibular function interferes in cardiovascular reflexes [published correction appears in *Arch Med Res*. 2003;34(5):444]. *Arch Med Res*. 2003;34(3):200-204.
5. Heidenreich KD, Weisend S, Fouad-Tarazi FM, White JA. The incidence of coexistent autonomic and vestibular dysfunction in patients with postural dizziness. *Am J Otolaryngol*. 2009;30(4):225-229.

http://dx.doi.org/10.1016/j.mayocp.2014.11.015