

Acute Dizziness may not Only be Cerebral or Vestibular, but Also Spinal or Neuropathic in Origin

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LETTER TO THE EDITOR

We were interested to read Edlow's article on the usefulness of HINTS, HINTS plus and the STANDING algorithm for differentiating between acute central (cerebral) and peripheral (vestibular) vertigo [Edlow, J. A, 2024]. The author regrets that these diagnostic tools have not yet found their way into routine emergency medicine, probably because most users are not familiar with the core instrument of these tests, the head impulse test [Edlow, J. A, 2024]. The author considers these three validated tests to be the most accurate for diagnosing patients with acute dizziness and identifying the underlying etiology [Edlow, J. A, 2024]. The review is impressive, but some points still need to be discussed.

The first point is that the author does not take into account that acute dizziness may be due not only to vestibular causes or ischemic stroke, but also to various other causes that may not be detected by the HINTs, HINTs plus or STANDING tests. These include, in particular, sensory or autonomic neuropathies of the peripheral nerves and spinal cord disorders that affect the centripetal pathways. Sensory and autonomic input is a cornerstone for keeping the body upright without deviating from the correct position. Therefore, any irritation of the mechanoreceptors and transmission of sensory signals from the mechanoreceptors of touch and proprioception can lead to unsteadiness when changing from supine to upright position or when walking. Mechanoreceptors of the sense of touch can be divided into "slowly adapting" receptors (Merkel cells, Ruffini bodies, Pinkus-Iggo tactile disks). "fast-adapting" receptors (Meissner corpuscles, hair follicle sensors, Krause end pistons) and "Pacinian corpuscles" (Vater-Paccini corpuscles, Golgi-Manzoni corpuscles). The proprioceptive mechanoreceptors include muscle spindles, Golgi tendon organs and Ruffini corpuscles [DoctorCheck Flexicon, 2024].

The second point is that stenosis or occlusion of the carotid/vertebral arteries has not been considered as a cause of acute dizziness [Edlow, J. A, 2024]. Since acute hypoxia due to carotid stenosis may become symptomatic due to low cardiac output, progression of stenosis or bradycardia, it is imperative to include carotid/vertebral artery disease in the considerations for diagnosis and therapeutic management of acute dizziness in the emergency department.

The third point is that we disagree with the description in Table 1 that nystagmus testing in acute stroke patients usually detects pure vertical torsional or direction-changing horizontal nystagmus [Edlow, J. A, 2024]. There are numerous reports of acute stroke patients who had spontaneous horizontal nystagmus in the acute phase that did not change direction [Saber Tehrani, A. S. *et al.*, 2018].

The fourth point is that medications or drugs have not been extensively discussed as causes of central and vestibular vertigo. Medications that commonly cause dizziness include antiepileptic drugs, analgesics, tranquilizers, muscle relaxants, antidepressants, anticholinergics, dopamine agonists or anti-inflammatory drugs.

In summary, this interesting article has some limitations that put the results and their interpretation into perspective. Taking these limitations into account could strengthen the conclusions and increase the validity of the report. Dizziness may be due not only to cerebral or vestibular causes, but also to a number of other causes that need to be ruled out before attributing acute dizziness solely to stroke or vestibular dysfunction.

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