Motion Perception without Nystagmus—A Novel Manifestation of Cerebellar Stroke

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Objective: The motion perception and the vestibulo-ocular reflex (VOR) each serve distinct functions. The VOR keeps the gaze steady on the target of interest, whereas vestibular perception serves a number of tasks, including awareness of self-motion and orientation in space. VOR and motion perception might abide the same neurophysiological principles, but their distinct anatomical correlates were proposed. In patients with cerebellar stroke in distribution of medial division of posterior inferior cerebellar artery, we asked whether specific location of the focal lesion in vestibulocerebellum could cause impaired perception of motion but normal eye movements. *Methods/Results:* Thirteen patients were studied, 5 consistently perceived spinning of surrounding environment (vertigo), but the eye movements were normal. This group was called "disease model." Remaining 8 patients were also symptomatic for vertigo, but they had spontaneous nystagmus. The latter group was called "disease control." Magnetic resonance imaging in both groups consistently revealed focal cerebellar infarct affecting posterior cerebellar vermis (lobule IX). In the "disease model" group, only part of lobule IX was affected. In the disease control group, however, complete lobule IX was involved. *Conclusions:* This study discovered a novel presentation of cerebellar stroke where only motion perception was affected, but there was an absence of objective neurologic signs. **Key Words:** Perception—Purkinje neuron—posterior inferior cerebellar artery—velocity storage.

Introduction

In natural behavior, the brain computes precise estimate of self-motion to ensure appropriate reflexive behavior and perception of motion. Basic principles of reflexive eye movements, the vestibulo-ocular reflex (VOR), are well known. Direct and indirect pathways mediate VOR. The direct pathway featuring "three-neuron arc"—vestibular afferents, premotor vestibular neurons, and ocular motor neurons—guarantees prompt compensatory eye movement in response to head movement.[1] The indirect pathway comprising the cerebellar cortex, deep cerebellar nuclei, and vestibular nuclei increases the bandwidth over which VOR is compensatory.[2]

Vestibular processing related to motion perception has enticed several recent investigations, some of these studies have emphasized the role of cerebellum.[3-7] Two hypotheses were proposed. First, VOR and motion perception share same groups of brainstem and cerebellar neurons. Second, the neurons responsible for VOR and motion perception are anatomically distinct but abide the same physiological principles. Recent studies were in support of second hypothesis.[5,7] These proposals, however, relied on indirect observations such as the differences in the dynamics of VOR and perceived angular velocity,[5,6] disparity in the effects of drugs to reduce the decay time constant of the VOR and perceived angular velocity,[7] and trends of altered motion perception in patients with ophthalmoplegia because of peripheral etiology.[3,4,8] The second hypothesis was also supported by studies in macaques that revealed the

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presence of neurons in cerebellar nodulus, rostral fastigial nucleus, and the vestibular nuclei that have pure vestibular sensitivity but lack responsiveness to eye movements, that is, the "vestibular-only" neurons.[9-17] Perception is one of the putative functions of such non–eye movement-sensitive central vestibular neurons. Perception as the possible function of these neurons became more evident with the discovery of the thalamic projections of the cerebellar and brain stem regions featuring the "vestibular-only" neurons in macaques.[11]

Signs and symptoms related to vestibular and ocular motor deficits in patients with focal cerebellar lesions might support experimental and theoretical framework depicting the differences between the neural mechanisms responsible for VOR and motion perception. For example, vertigo (perception of rotation), spontaneous nystagmus, gaze-evoked nystagmus, and body lateropulsion are known manifestations of focal stroke affecting the posterior cerebellar vermis.[18,19] We asked whether focal cerebellar lesion exists that only manifests as vertigo but the absence of ocular motor deficits including nystagmus.

Methods

Thirteen cases with acute cerebellar stroke were examined. The University Hospitals Case Medical Center institutional review board approved this retrospective study. Clinical assessment was performed as a part of hospital admission. Patients with acute onset of vertigo, ability to accurately provide history, qualitative description, duration, and severity of vertigo and an evidence of acute stroke on magnetic resonance imaging (MRI) were included in the study. Patients were excluded if there was a chronic history of vertigo without change in its quality and severity, if there was an evidence for the pathology affecting brainstem vestibular or eye movement–sensitive nuclei, if MRI did not reveal acute or subacute stroke affecting the vestibulocerebellum, and if there was a clinical or neuroimaging evidence of a degenerative cerebellar disease.

The patients were scanned on 1.5 T scanner. Axial T1- and T2-weighted images, diffusion-weighted images (DWIs), apparent diffusion coefficient (ADC), and FLAIR sequences were acquired. Slice thickness for each image was 5 mm. Picture Archiving and Communication System was used to determine the anatomical location of the affected cerebellar lobule in the MRI. The areas of diffusion restriction on DWI sequences were correlated with ADC to confirm the ischemic lesion. Cross-referencing tool in the PACS software was used to colocalize the area of diffusion restriction in the fluid-attenuated inversion recovery and T1-weighted axial and saggital sequences. The latter facilitated accurate anatomical localization of acute lesions that were identified on DWI and ADC sequences.

Three-dimensional MRI atlas of cerebellum was used to accurately identify the lobules of interest in MRI.[20] The areas of interest in vestibulo-cerebellum, identified in this article as lobules IX and X, were diversely labeled in the past. For lobule IX, some authors used the term paraflocculus[21-25]; the same area was also called tonsil[26-32] or uvula.[33] Schematic in Figure 1 identifies various lobules in the map of cerebellum in sagittal and axial sections in nomenclature proposed by Schmahmann et al.[20]

Results

Clinical Presentation

Thirteen patients (9 men and 4 women) with abrupt onset of vertigo, intractable nausea, and vomiting were assessed. Assessment was performed within 24 hours of symptom onset. Patients described vertigo as spinning sensation. Four patients could clearly describe the direction of spinning—one perceived spinning of surrounding environment from the left to right, whereas three perceived rightward to leftward motion. Eight patients were unclear about the direction of perceived spinning. In eight patients, the severity of the vertigo was dependent on the head orientation with respect to gravity. The details of symptoms are outlined in Table 1.

Neurologic examination classified this cohort into two groups. One group had no nystagmus in primary or eccentric gaze or with removal of visual fixation using Frenzel goggles during upright, supine, left ear down, and right ear down orientations. The VOR was normal during head impulses and head shaking maneuver. Saccades and pursuit eye movements and optokinetic nystagmus were normal. The vertigo was severe in three patients to keep them confined to bed. Two patients could stand up, and they had body pulsion toward the side of cerebellar lesion. Remaining neurologic examination was normal. This was "disease model" group.

The second group, the "disease control," featured ocular motor deficits on examination. Nystagmus was present in at least one of the four head orientations with respect to gravity (ie, supine, upright, right ear down, or left ear down positions). In five of eight patients, the pattern of nystagmus was head position dependent. Saccadic hypometria was seen in one patient, and pursuit was interrupted by quick phase of nystagmus. Two patients had acute onset of hypoactive VOR in addition to gravity dependence of spontaneous nystagmus. It was concluded that the patients with hypoactive VOR and gravity-dependent nystagmus had acute stroke affecting the vascular distributions of anterior and posterior inferior cerebellar arteries.[34] The VOR testing was deferred in one patient who presented with cerebellar stroke symptoms and neck pain after injury, concerning for vertebral artery dissection. All patients had profound imbalance and body lateropulsion. Table 1 illustrates the