

Acute Peripheral vs. Central Vestibulopathy



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Disclosure

None

Learning objectives

- ❖ Key clinical features that aid in differentiation of acute central from peripheral vestibulopathy
- ❖ Application of appropriate evaluation for the differentiation
- ❖ Red flags for central vertigo

Key message

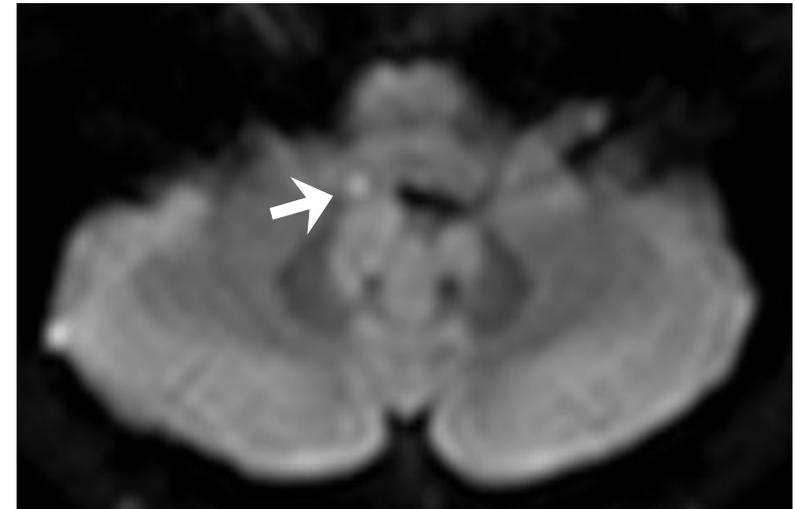
- ❖ Clinical features and findings of bedside neuro-ontological examinations are more important than neuroimaging in differentiating acute central from peripheral vestibulopathy.

DDx of Isolated Vertigo

	Peripheral	Central
SN	H-T	Pure D/U/T
Direction	Unidirectional	Direction-changing
Fixation	Decrease	No change

Isolated vestibular nuclear infarction: report of two cases and review of the literature

Hyo-Jung Kim · Seung-Han Lee · Jae Han Park ·
Jung-Yoon Choi · Ji-Soo Kim



(J Neurol, 2013)

Patterns of spontaneous and head-shaking nystagmus in cerebellar infarction: imaging correlations

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Horizontal head-shaking may induce nystagmus in peripheral as well as central vestibular lesions. While the patterns and mechanism of head-shaking nystagmus are well established in peripheral vestibulopathy, they require further exploration in central vestibular disorders. To define the characteristics and mechanism of head-shaking nystagmus in central vestibulopathies, we investigated spontaneous nystagmus and head-shaking nystagmus in 72 patients with isolated cerebellar infarction. Spontaneous nystagmus was observed in 28 (39%) patients, and was mostly ipsilesional when observed in unilateral infarction (15/18, 83%). Head-shaking nystagmus developed in 37 (51%) patients, and the horizontal component of head-shaking nystagmus was uniformly ipsilesional when induced in patients with unilateral infarction. Perverted head-shaking nystagmus occurred in 23 (23/37, 62%) patients and was mostly downbeat (22/23, 96%). Lesion subtraction analyses revealed that damage to the uvula, nodulus and inferior tonsil was mostly responsible for generation of head-shaking nystagmus in patients with unilateral posterior inferior cerebellar artery infarction. Ipsilesional head-shaking nystagmus in patients with unilateral cerebellar infarction may be explained by unilateral disruption of uvulonodular inhibition over the velocity storage. Perverted (downbeat) head-shaking nystagmus may be ascribed to impaired control over the spatial orientation of the angular vestibulo-ocular reflex due to uvulonodular lesions or a build-up of vertical vestibular asymmetry favouring upward bias due to lesions involving the inferior tonsil.

(Brain, 2011)

HSN in the Opposite Direction of SN in Dentate Hemorrhage

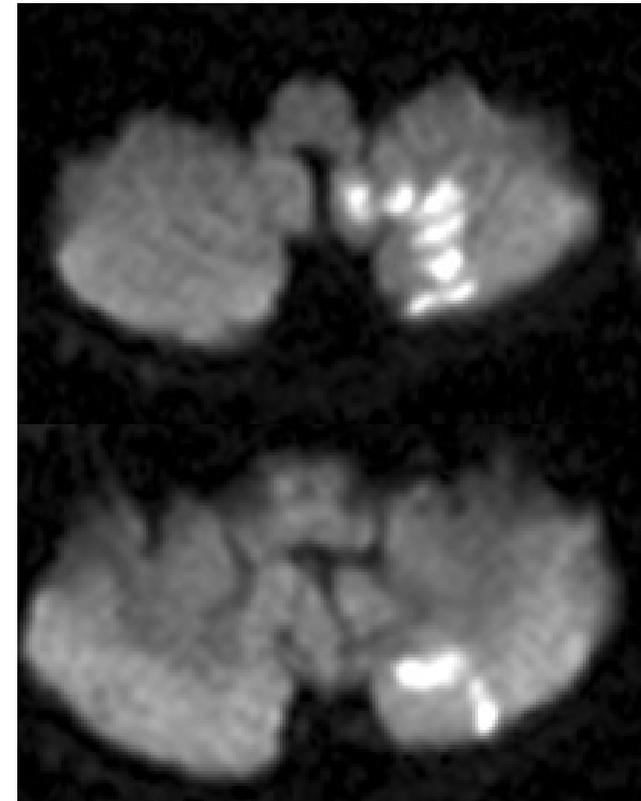
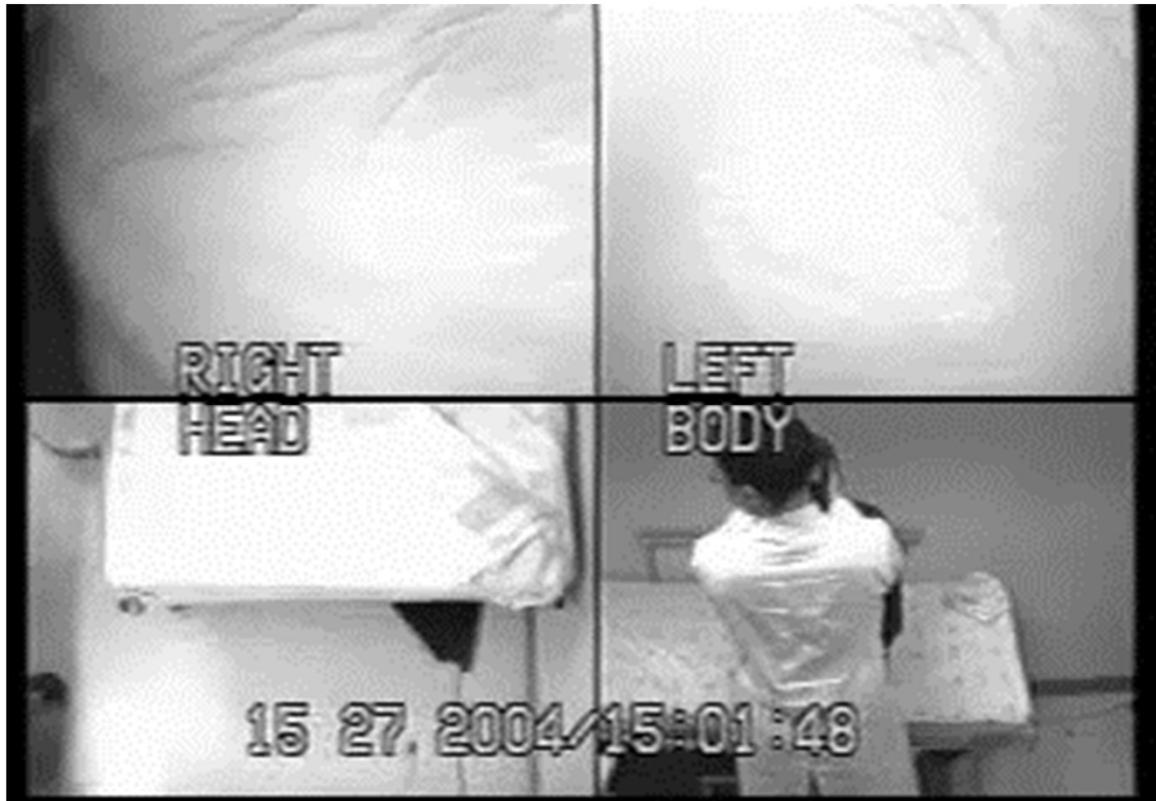
Head Shaking Nystagmus



VIDEO

Isolated perverted head-shaking nystagmus in focal cerebellar infarction

*J.S. Kim, MD; K.-W. Ahn, MD; S.Y. Moon, MD; K.-D. Choi, MD;
S.-H. Park, MD; and J.-W. Koo, MD*



Perverted responses: the responses occurring in the plane other than being stimulated.

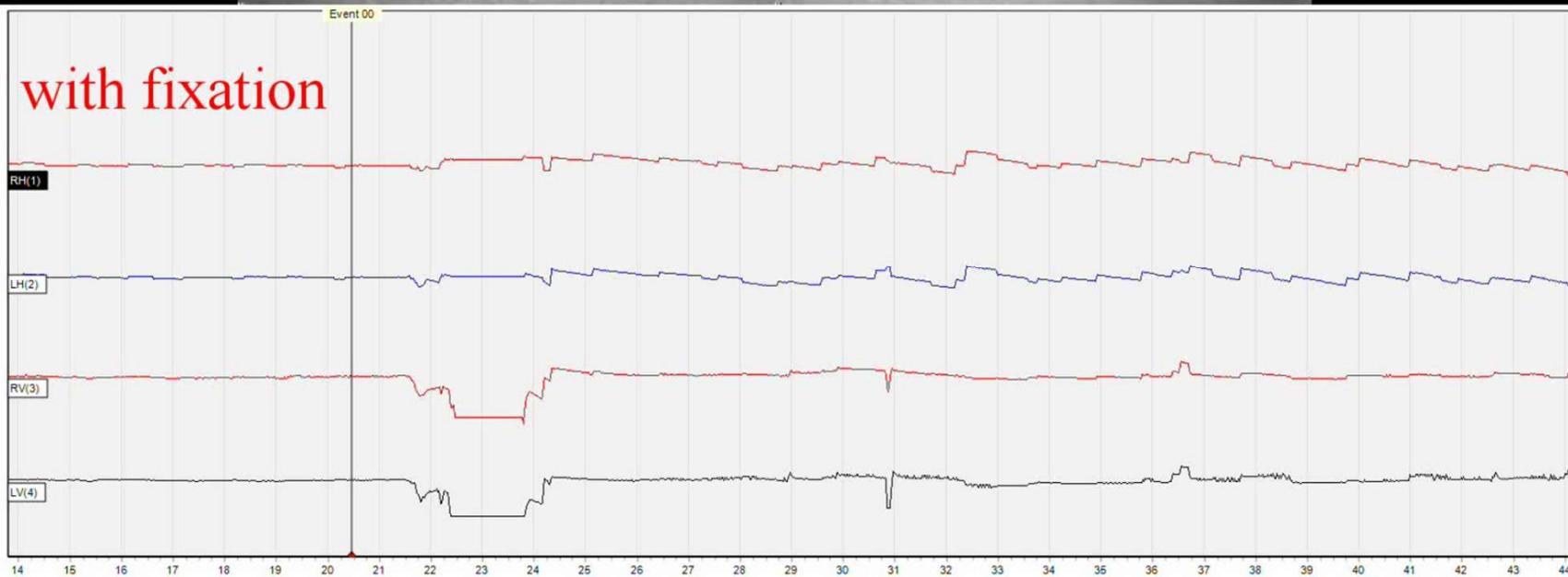
(Neurology, 2005)

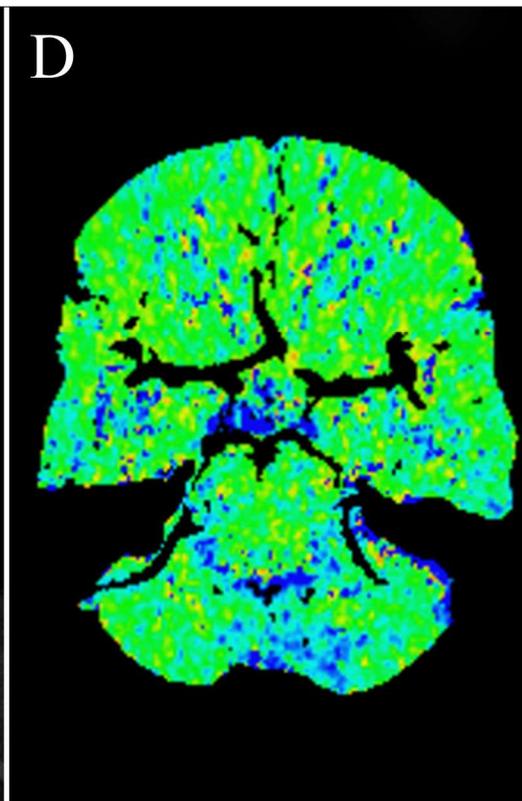
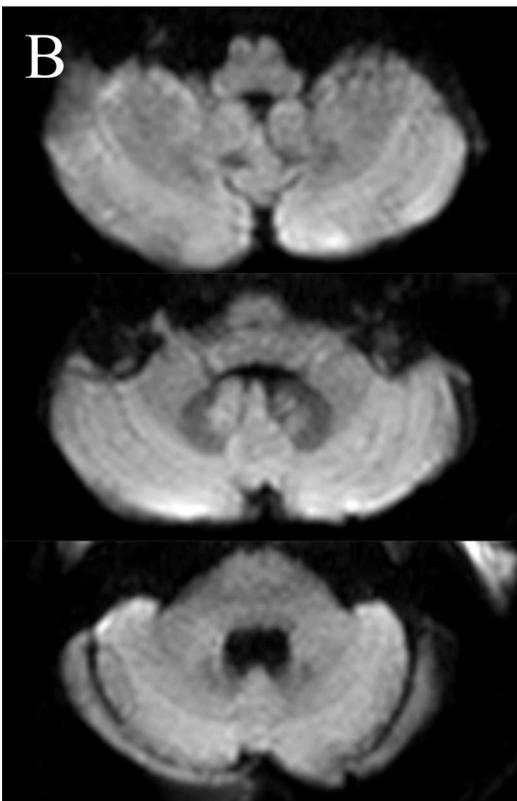
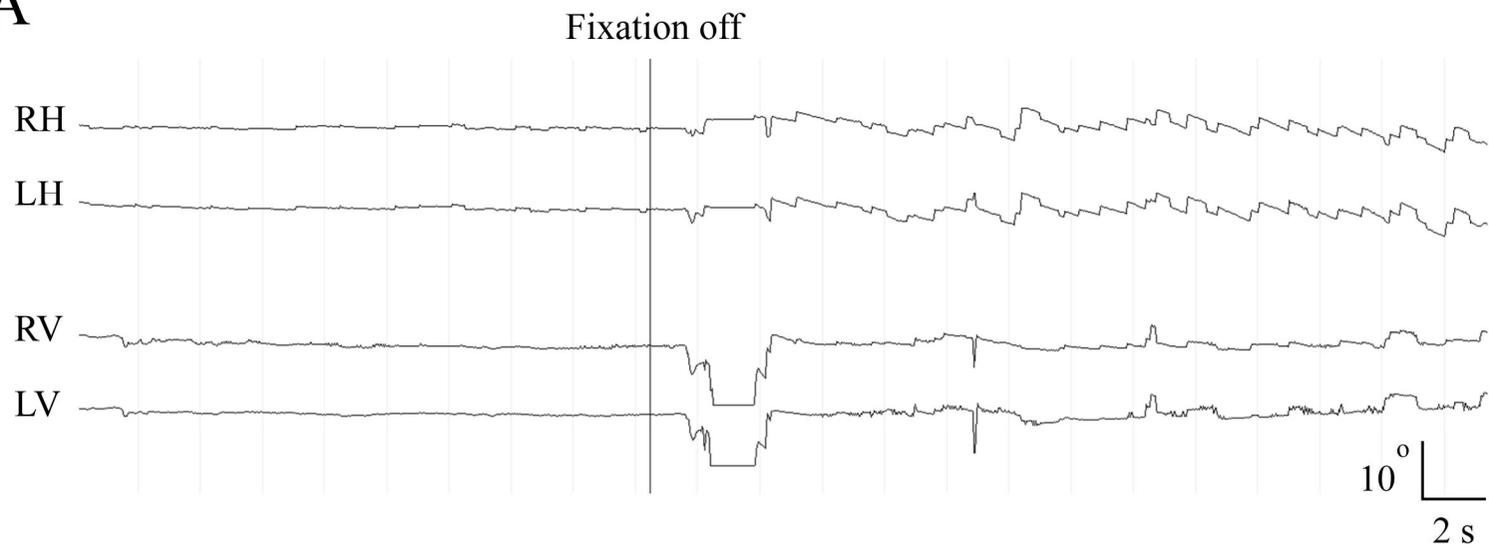
DDx of Isolated Vertigo

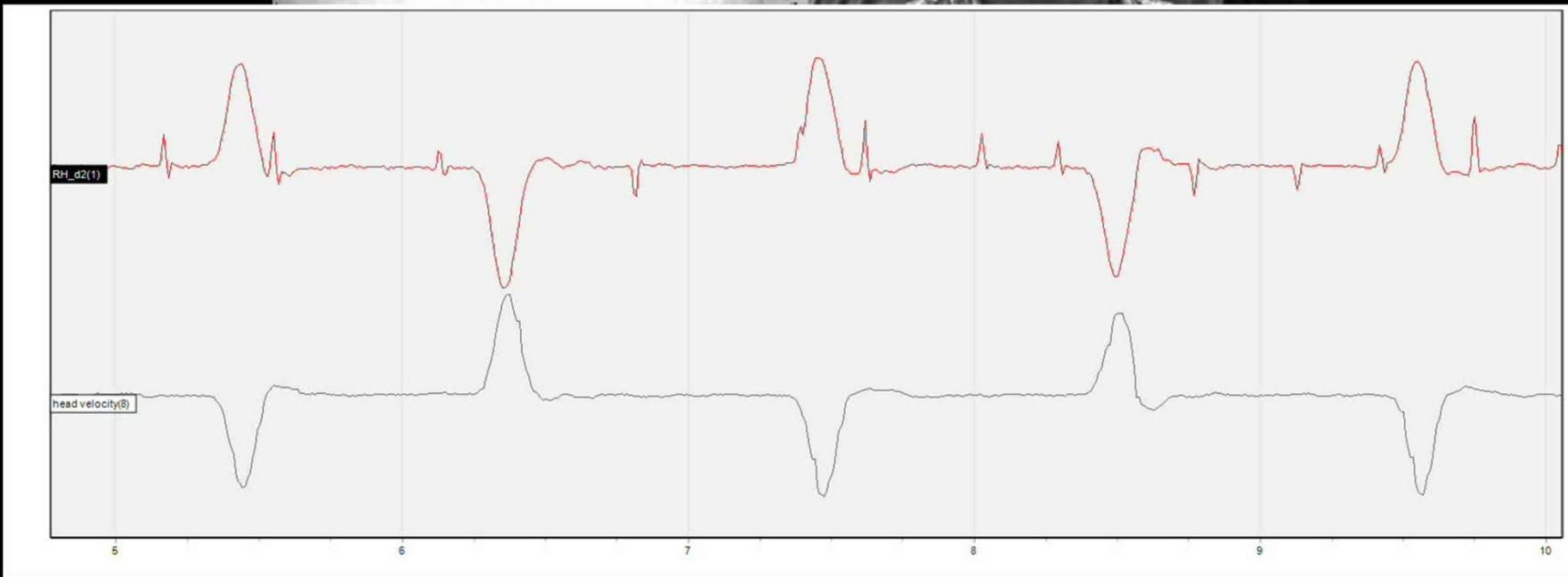
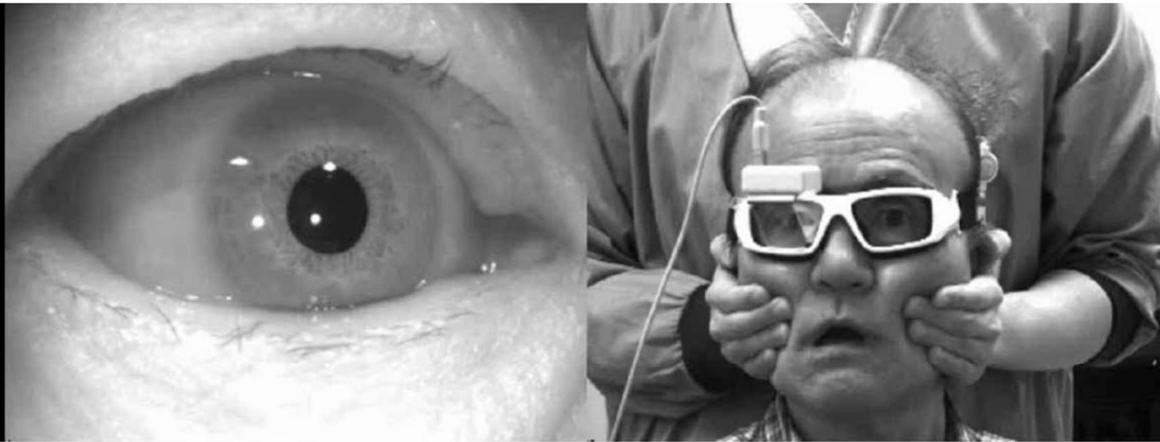
	Peripheral	Central
SN	H-T	Pure D/U/T
Direction	Unidirectional	Direction-changing
Fixation	Decrease	No change
HSN	Augmentation (Contra)	Reversed Perverted



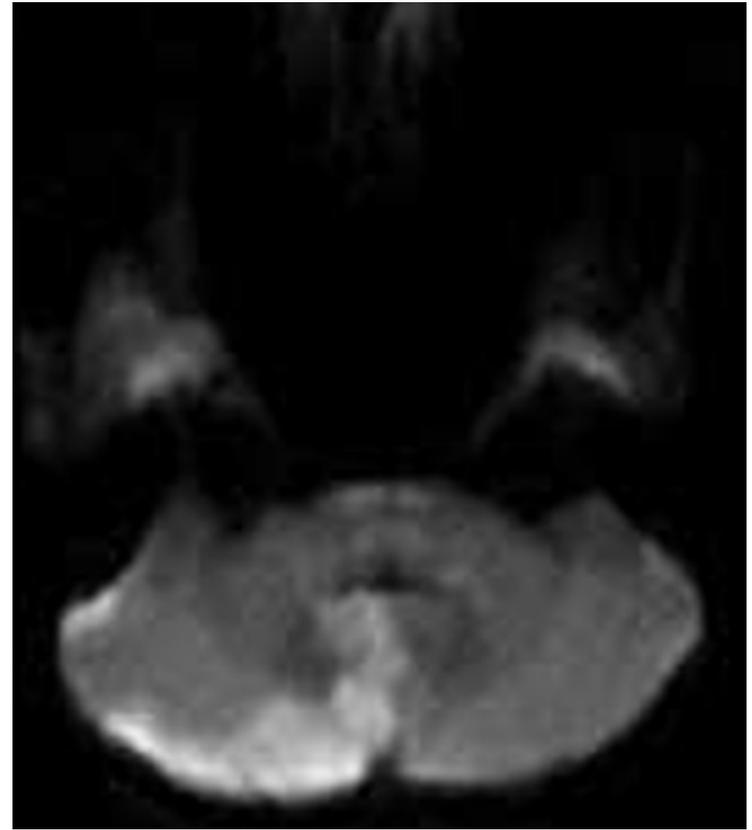
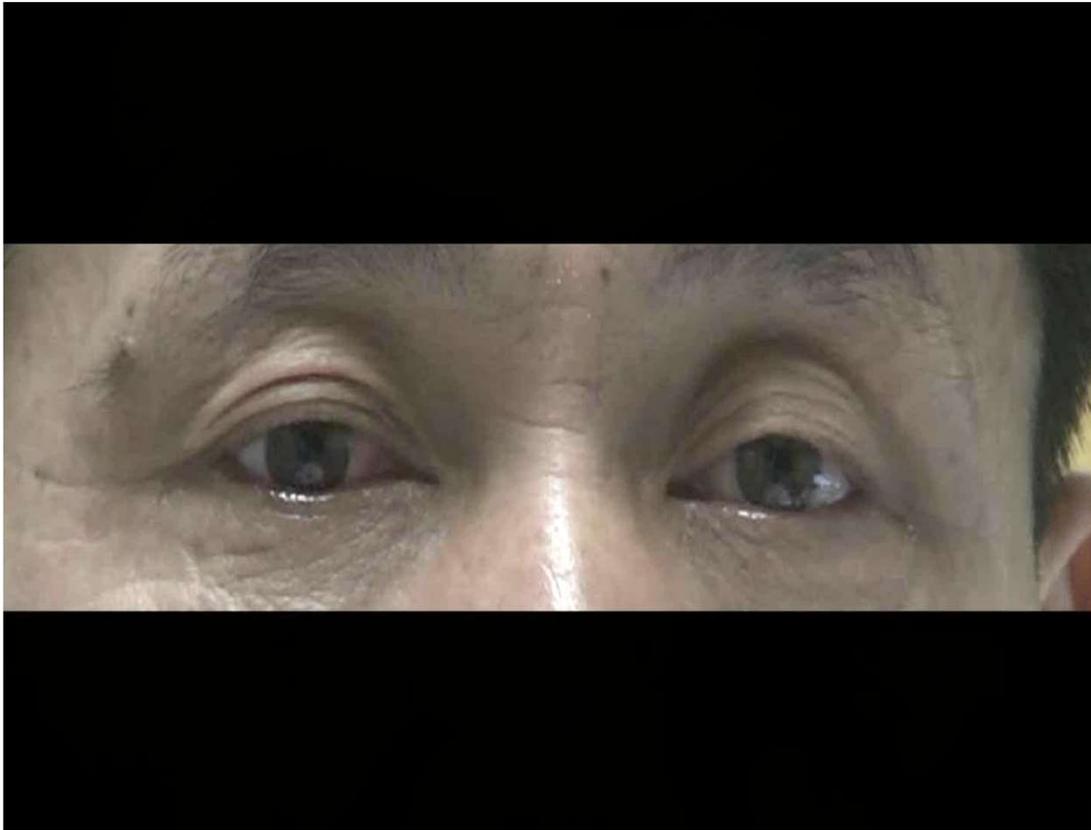
with fixation



A



Skew Deviation in mPICA Infarction



DDx of Isolated Vertigo

	Peripheral	Central
SN	H-T	Pure D/U/T
Direction	Unidirectional	Direction-changing
Fixation	Decrease	No change
HSN	Augmentation (Contra)	Reversed Perverted
HINTS	Negative	Positive

HINTS to Diagnose Stroke in the Acute Vestibular Syndrome

Three-Step Bedside Oculomotor Examination More Sensitive Than Early MRI Diffusion-Weighted Imaging

Jorge C. Kattah, MD; Arun V. Talkad, MD; David Z. Wang, DO;
Yu-Hsiang Hsieh, PhD, MS; David E. Newman-Toker, MD, PhD

Background and Purpose—Acute vestibular syndrome (AVS) is often due to vestibular neuritis but can result from vertebrobasilar strokes. Misdiagnosis of posterior fossa infarcts in emergency care settings is frequent. Bedside oculomotor findings may reliably identify stroke in AVS, but prospective studies have been lacking.

Methods—The authors conducted a prospective, cross-sectional study at an academic hospital. Consecutive patients with AVS (vertigo, nystagmus, nausea/vomiting, head-motion intolerance, unsteady gait) with ≥ 1 stroke risk factor underwent structured examination, including horizontal head impulse test of vestibulo-ocular reflex function, observation of nystagmus in different gaze positions, and prism cross-cover test of ocular alignment. All underwent neuroimaging and admission (generally <72 hours after symptom onset). Strokes were diagnosed by MRI or CT. Peripheral lesions were diagnosed by normal MRI and clinical follow-up.

Results—One hundred one high-risk patients with AVS included 25 peripheral and 76 central lesions (69 ischemic strokes, 4 hemorrhages, 3 other). The presence of normal horizontal head impulse test, direction-changing nystagmus in eccentric gaze, or skew deviation (vertical ocular misalignment) was 100% sensitive and 96% specific for stroke. Skew was present in 17% and associated with brainstem lesions (4% peripheral, 4% pure cerebellar, 30% brainstem involvement; χ^2 , $P=0.003$). Skew correctly predicted lateral pontine stroke in 2 of 3 cases in which an abnormal horizontal head impulse test erroneously suggested peripheral localization. Initial MRI diffusion-weighted imaging was falsely negative in 12% (all <48 hours after symptom onset).

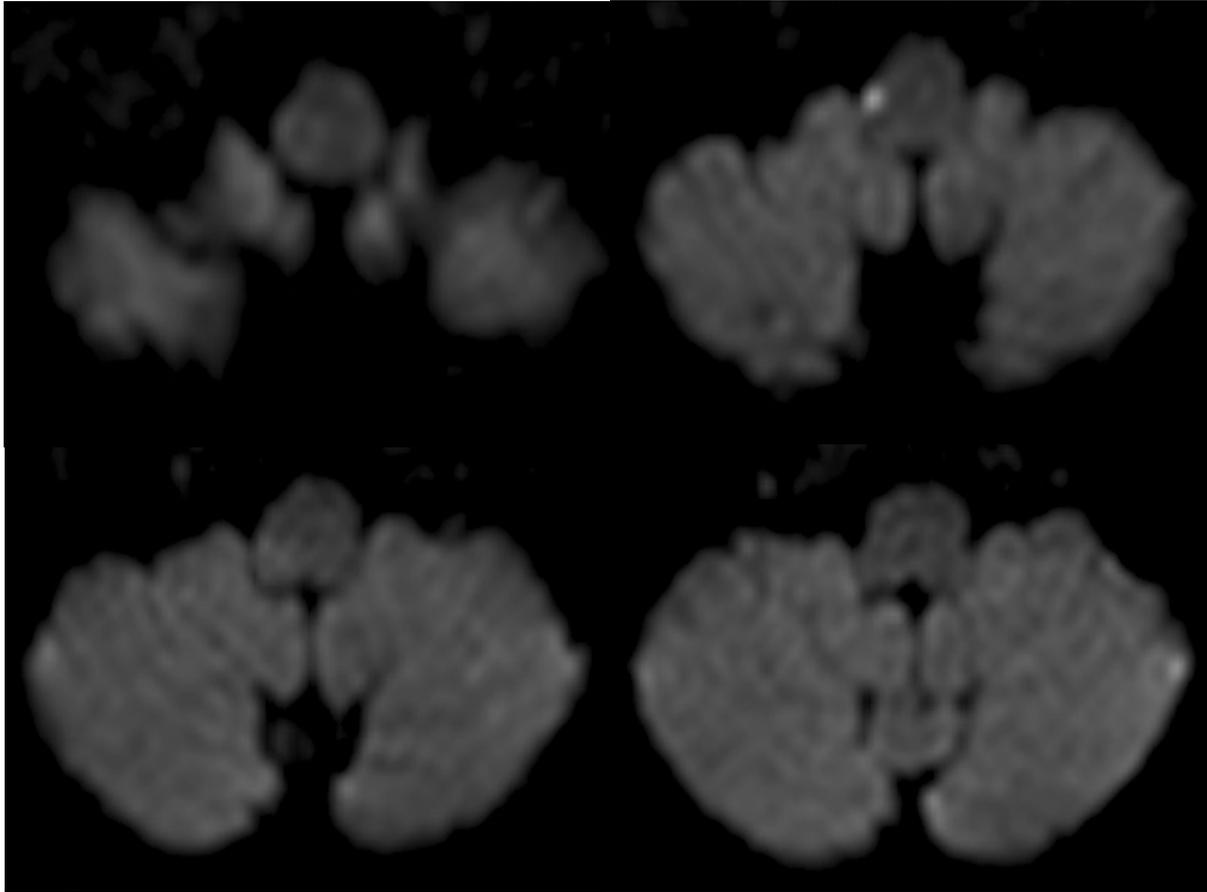
Conclusions—Skew predicts brainstem involvement in AVS and can identify stroke when an abnormal horizontal head impulse test falsely suggests a peripheral lesion. A 3-step bedside oculomotor examination (HINTS: Head-Impulse—Nystagmus—Test-of-Skew) appears more sensitive for stroke than early MRI in AVS. (*Stroke*. 2009;40:3504-3510.)

Key Words: cerebrovascular accident ■ diagnosis ■ neurologic examination ■ sensitivity and specificity ■ vertigo

HINTS to Diagnose Stroke in the Acute Vestibular Syndrome

Three-Step Bedside Oculomotor Examination More Sensitive Than Early MRI Diffusion-Weighted Imaging

- ❖ Spontaneous vertigo \geq 24 hours
- ❖ Spontaneous nystagmus
- ❖ \geq one vascular risk factor
- ❖ History of recurrent vertigo with or without hearing loss



[Video](#)

DDx of Isolated Vertigo

	Peripheral	Central
SN	H-T	Pure D/U/T
Direction	Unidirectional	Direction-changing
Fixation	Decrease	No change
HSN	Augmentation (Contra)	Reversed Perverted
HINTS	Negative	Positive
Imbalance	Mild	Severe

Nystagmus in UPV: Characteristics

- 1) Mixed H-T > V (upbeat >> downbeat)
- 2) Contralesional/Unidirectional
- 3) Follows Alexander's law
- 4) Linear slow phases
- 5) Suppressed by visual fixation
- 6) Augmentation by head-shaking
- 7) Positive HITs to the lesion side
- 8) Marked improvement within days



Cerebellar infarction presenting isolated vertigo

Frequency and vascular topographical patterns

H. Lee, MD; S.-I. Sohn, MD; Y.-W. Cho, MD; S.-R. Lee, MD; B.-H. Ahn, MD; B.-R. Park, MD;
and R.W. Baloh, MD

Abstract—Objective: To determine the frequency of cerebellar infarction mimicking vestibular neuritis (VN), the pattern of clinical presentation, and the territory of the cerebellar infarction when it simulates VN. **Methods:** We studied 240 consecutive cases of isolated cerebellar infarction in the territories of the cerebellar arteries diagnosed by brain MRI from the acute stroke registry at the Keimyung University Dongsan Medical Center. **Results:** We identified 25 patients (10.4%) with isolated cerebellar infarction who had clinical features suggesting VN. Two types of cerebellar infarction simulating VN were found: isolated spontaneous prolonged vertigo with imbalance as a sole manifestation of cerebellar infarction (n = 24) and isolated spontaneous prolonged vertigo with imbalance as an initial manifestation of cerebellar infarction (n = 1) followed by delayed neurologic deficits 2 days after the onset. The cerebellar infarction territory most commonly involved was the medial branch of the posterior inferior cerebellar artery territory (24/25: 96%), followed by the anterior inferior cerebellar artery territory (1/25: 4%). None of patients with infarcts in the territory of the superior cerebellar artery or multiple cerebellar arteries showed isolated spontaneous prolonged vertigo. **Conclusions:** Cerebellar infarction simulating vestibular neuritis is more common than previously thought. Early recognition of the pseudo-vestibular neuritis of vascular cause may allow specific management.

NEUROLOGY 2006;67:1178–1183

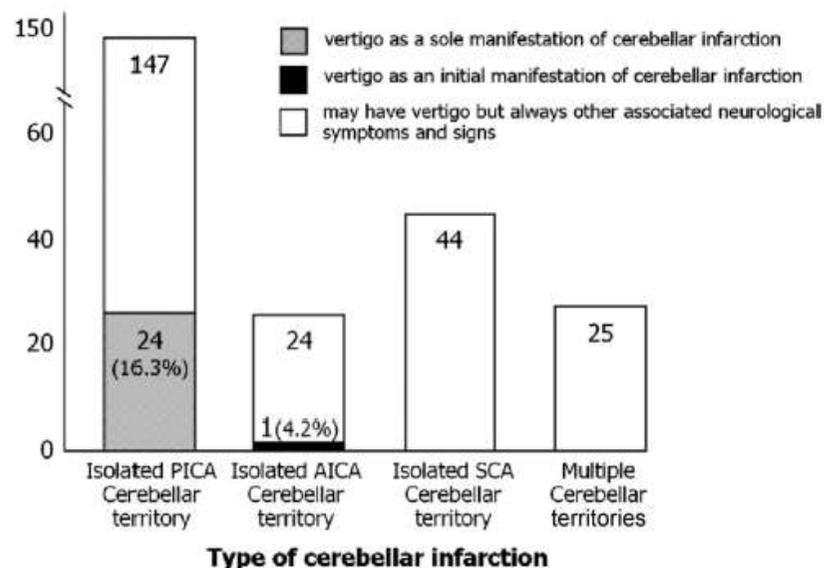
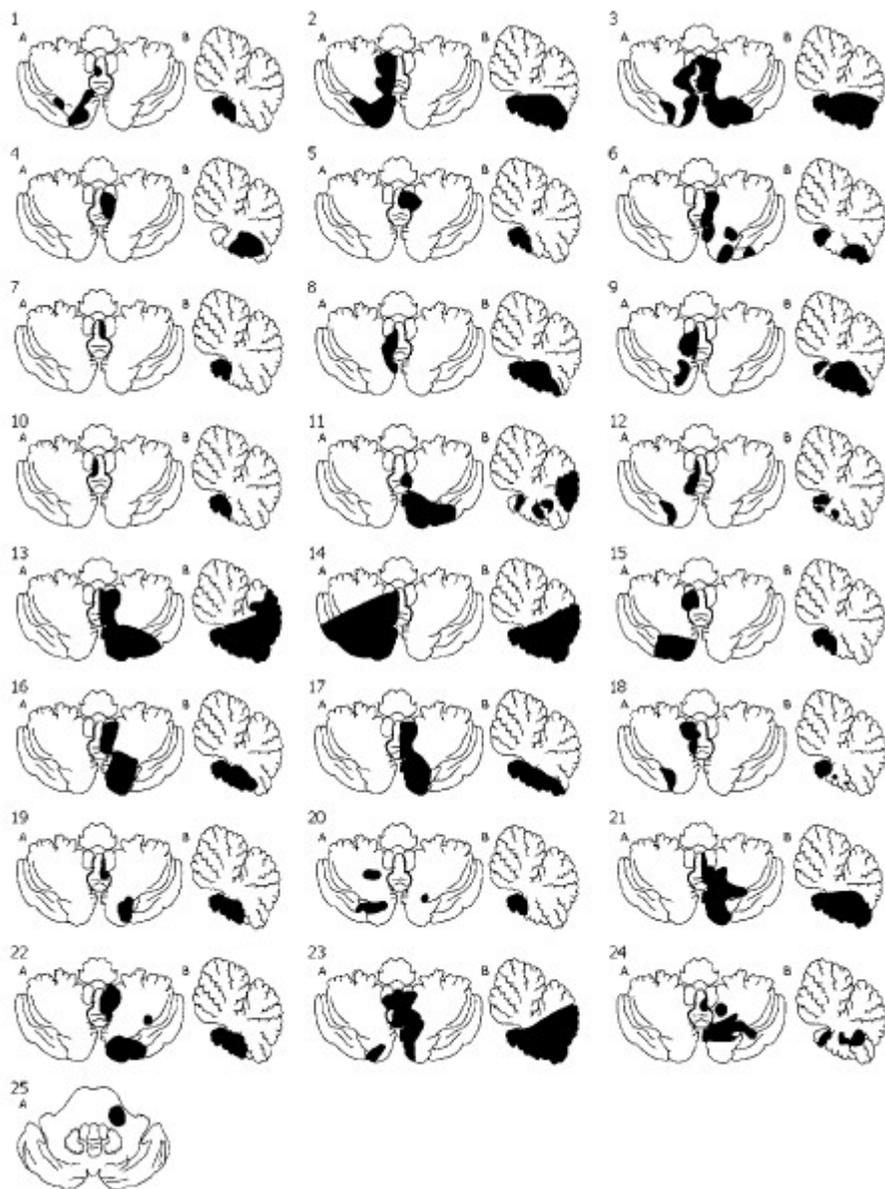
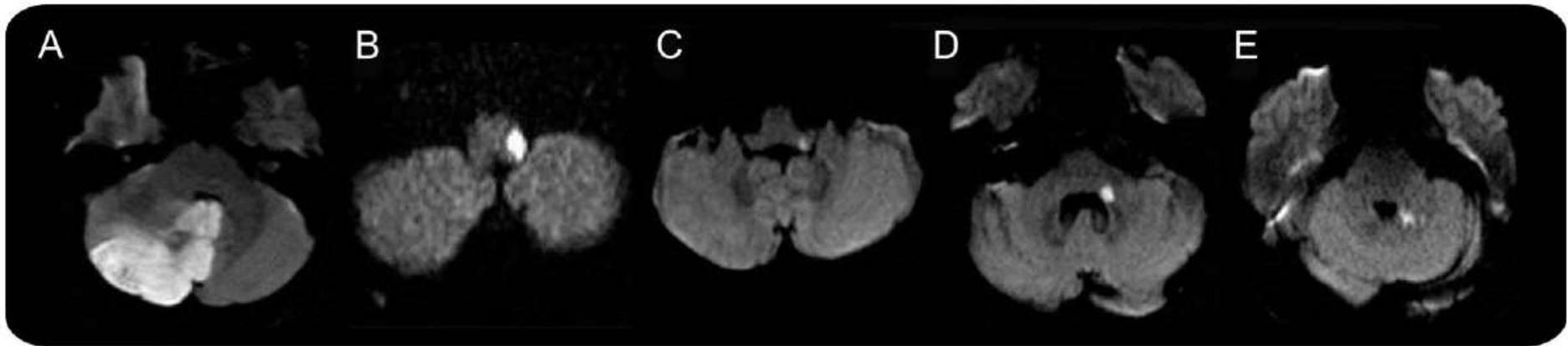


Figure 1. Frequency of cerebellar infarction subtypes simulating vestibular neuritis. PICA = posterior inferior cerebellar artery; AICA = anterior inferior cerebellar artery; SCA = superior cerebellar artery.

(Lee et al., Neurology 2006)

- ❖ Isolated vestibular syndrome in 34 (25.8%) of 132 patients with posterior circulation infarctions.
- ❖ Most common in the cerebellum (23/34, 67.6%).

Figure 1 MRIs of the representative patients with isolated vestibular syndrome



The lesions mostly involve the cerebellum in the territory of medial posterior inferior cerebellar artery (A), caudal lateral (B) or rostral-dorsolateral medulla (C), and inferior (D) or superior cerebellar peduncles (E).

(Choi et al., Neurol Clin Pract 2014)

Negative Neuroimaging in Isolated Vascular Vertigo

- ❖ 12% in AVS > 24 hrs with one risk factor (Kattah et al., 2009)
- ❖ 18% with isolated vestibular syndrome (Choi et al., 2014)
- ❖ 53% in small strokes causing AVS (Saber-Tehrani et al., 2014)

Indicators for acute vascular vertigo

Vertigo with focal neurologic findings

Vertigo with HINTS (+)

Vertigo with other central ocular motor findings

Vertigo with profound imbalance

Vertigo with new prominent headache

Vertigo lasting several minutes

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