CASE SHARING

台北榮總神經內科

演講者: 王晨羽 醫師

指導者: 許立奇 醫師

CASE 1

73 y/o Female

- ADL independent

Underlying diseases

- HTN
- Type 2 DM, with ESRD under HD
- RA
- CAD with TVD s/p DES*3 and CABG, taking Plavix
- PAOD s/p PTA of Lt popliteal a.

Chief complaint

- Acute onset dizziness, vomiting and nausea for 2 weeks
- Intermittent headache for one week

CASE 1

2019/10/13

- acute onset vertigo, persistant
- nausea +, vomiting +

2019/10/17

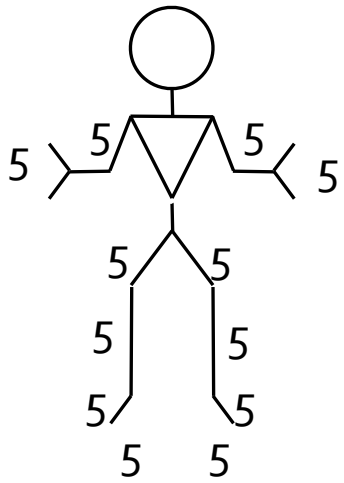
- 1st NEURO OPD visit

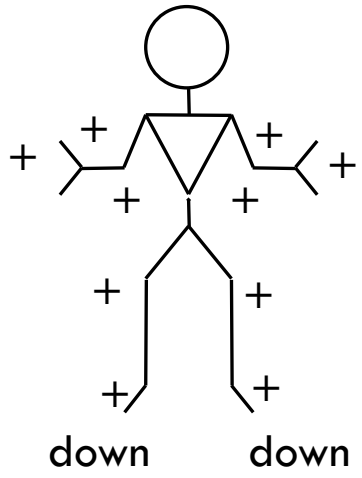
NE

Consciousness: E4V5M6

Cranial nerves: normal

Motor function:





Coordination: normal

Gait: normal gait

Sensory function:

- Light touch: normal
- Pinprick: normal
- Vibration: normal
- Joint position: normal

EPS: Rigitidy(-), Tremor(-), bradykinesia(-)

Involuntary movement: nil

CASE 1

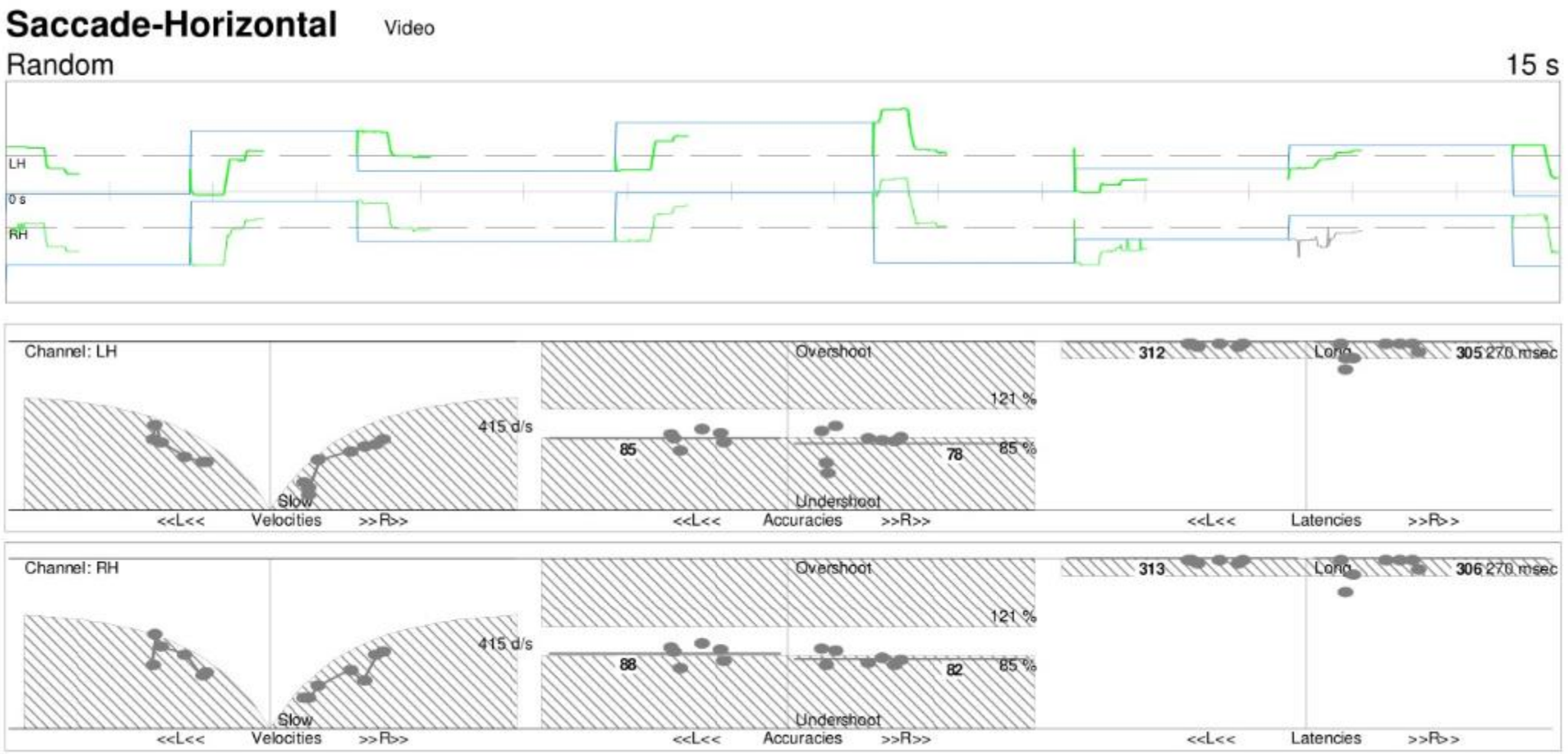
2019/10/13

- acute onset vertigo, persistant
- nausea +, vomiting +

2019/10/17

- 1st NEURO OPD visit
- NE: no nystagmus, no diplopia, no atatxia, no wkn
- Arrange ENG, caloric test

2019/10/30 OPD



Slow saccade **Hypometric saccade**

CASE 1

2019/10/13

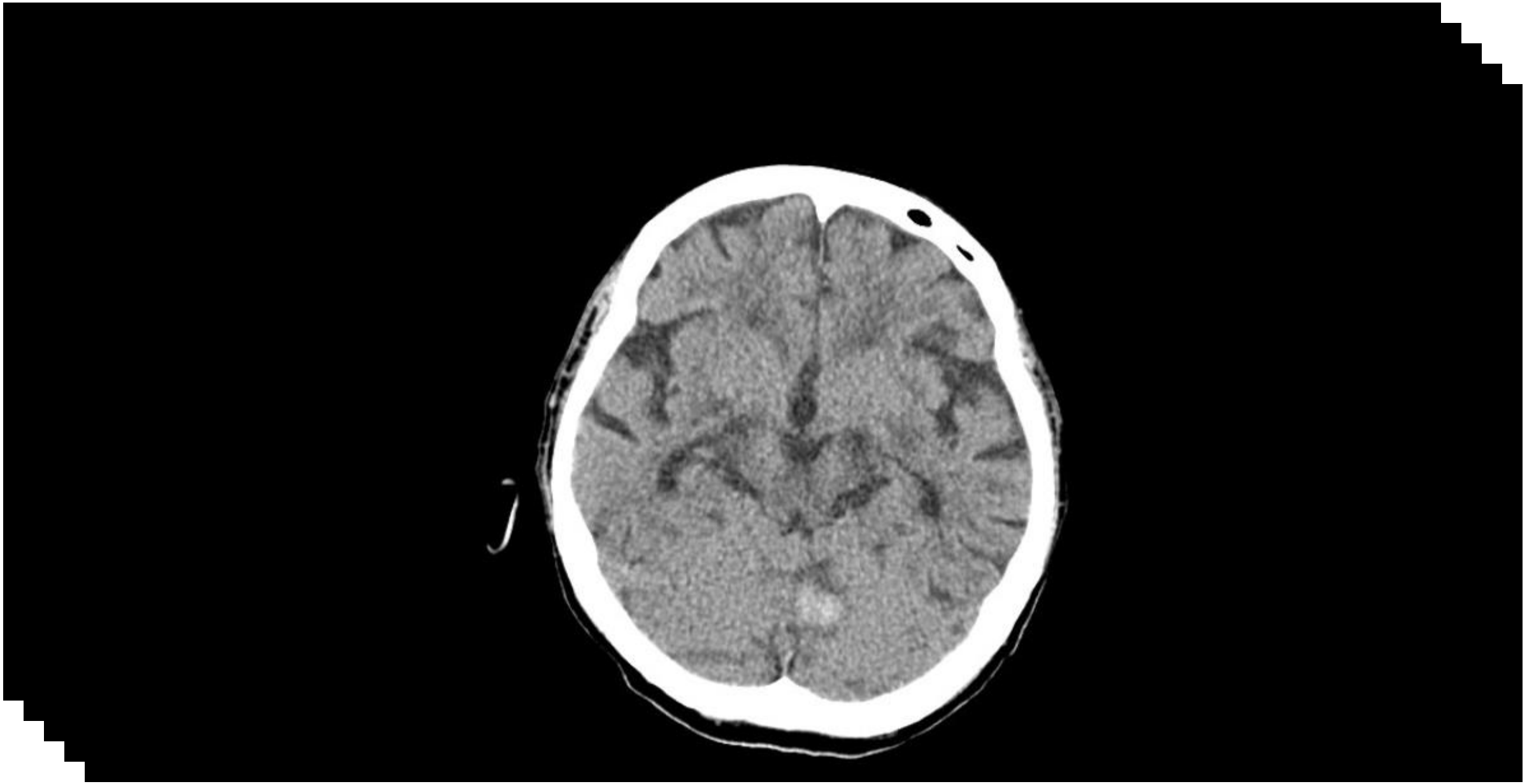
- acute onset vertigo, persistant
- nausea +, vomiting +

2019/10/17

- 1st NEURO OPD visit
- NE: no nystagmus, no diplopia, no atatxia, no wkn
- Arrange ENG, caloric test

2019/10/30 OPD

- Suspect cerebellar lesion
- Refer to ER for brain CT



Case 1

- Diagnosis: Left cerebellum ICH 2.88 mL, ICH score 1
- NIHSS = 0
- Management: hold Plavix, BP control (SBP<140 mmHg), glycerol, rehabilitation

CASE 2

44 male

- ADL independent

Underlying diseases

- HBV carrier

Chief complaint

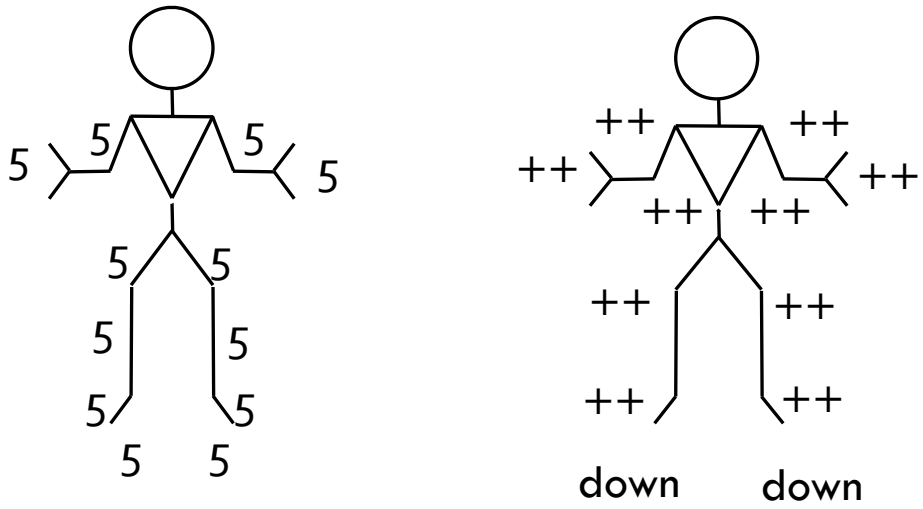
- Acute onset of dizziness for one day

NE

Consciousness: E4V5M6

Cranial nerves: normal

Motor function:



Coordination:

- F-N-F: left dysmetria
- H-N-H: left dysmetria

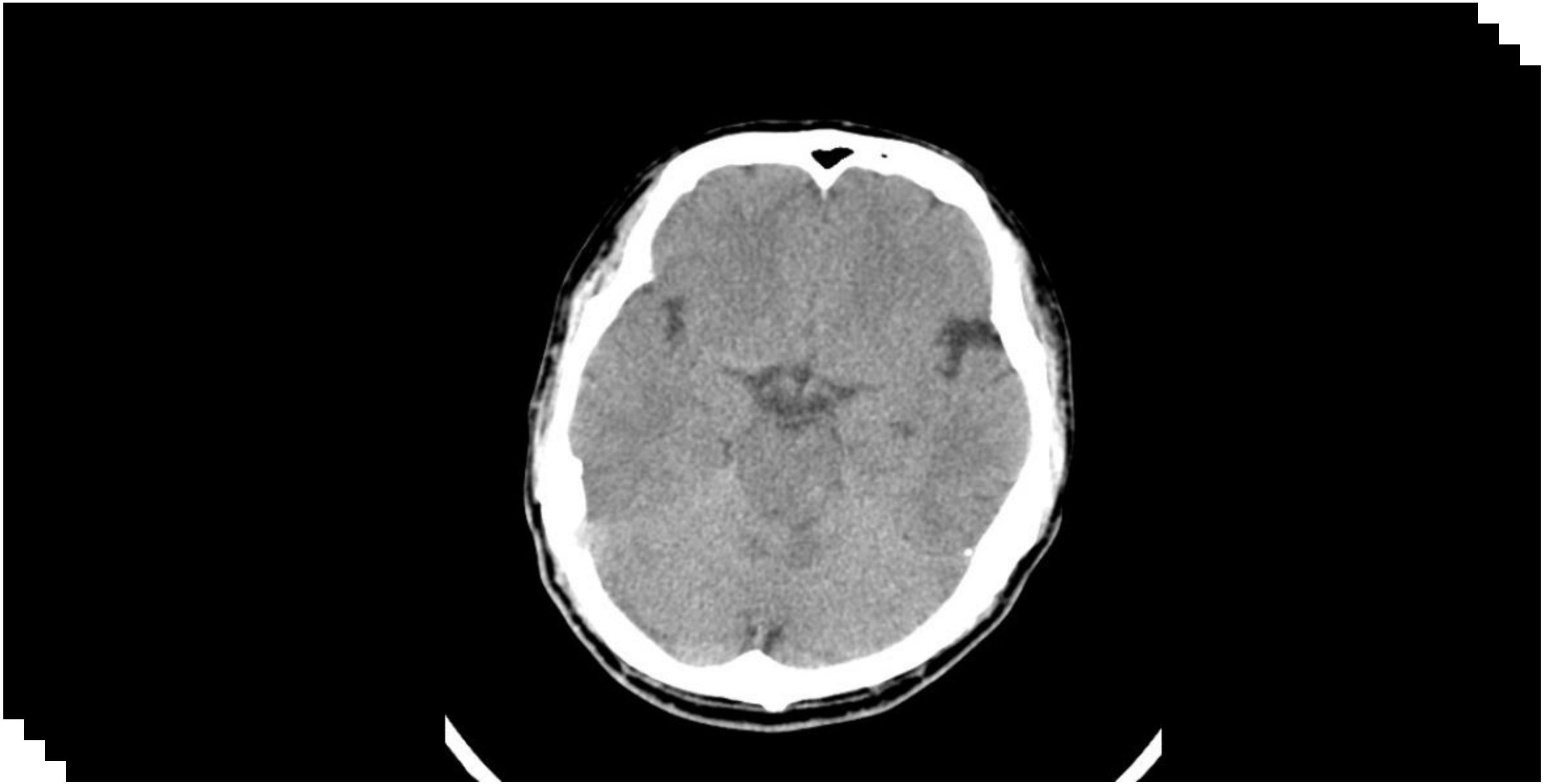
Gait: wide-based gait

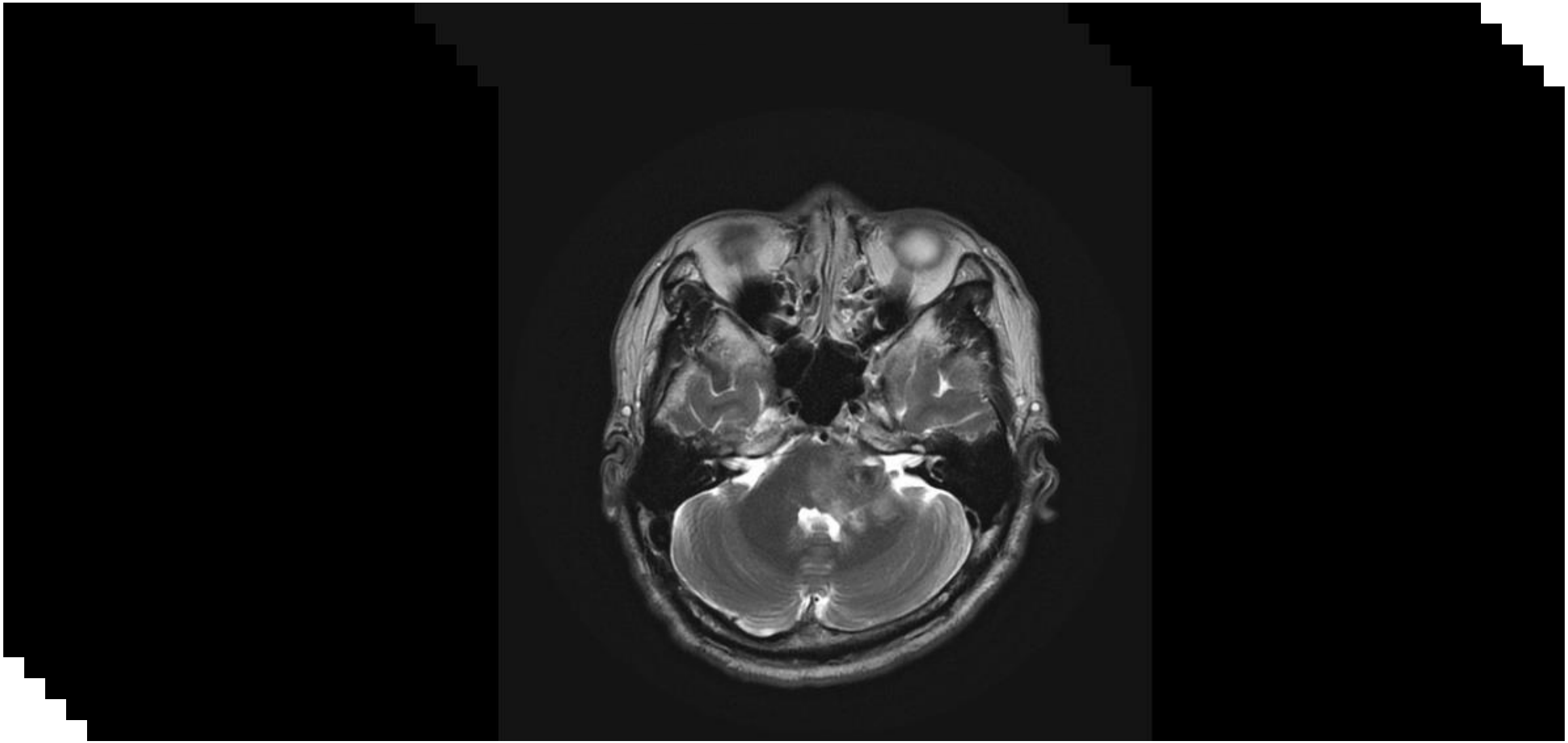
Sensory function:

- Light touch: normal
- Pinprick: normal
- Vibration: normal
- Joint position: normal

EPS: Rigidity(-), Tremor(-), bradykinesia(-)

Involuntary movement: nil





cavernous angioma about 2.2cm at left pons

Case 2

- Diagnosis: ICH at left pontocerebellar junction about 2.2cm, cavernous angioma with intralesional bleeding favored, ICH=1, NIHSS=2
- NIHSS = 2
- Management: ICH treatment

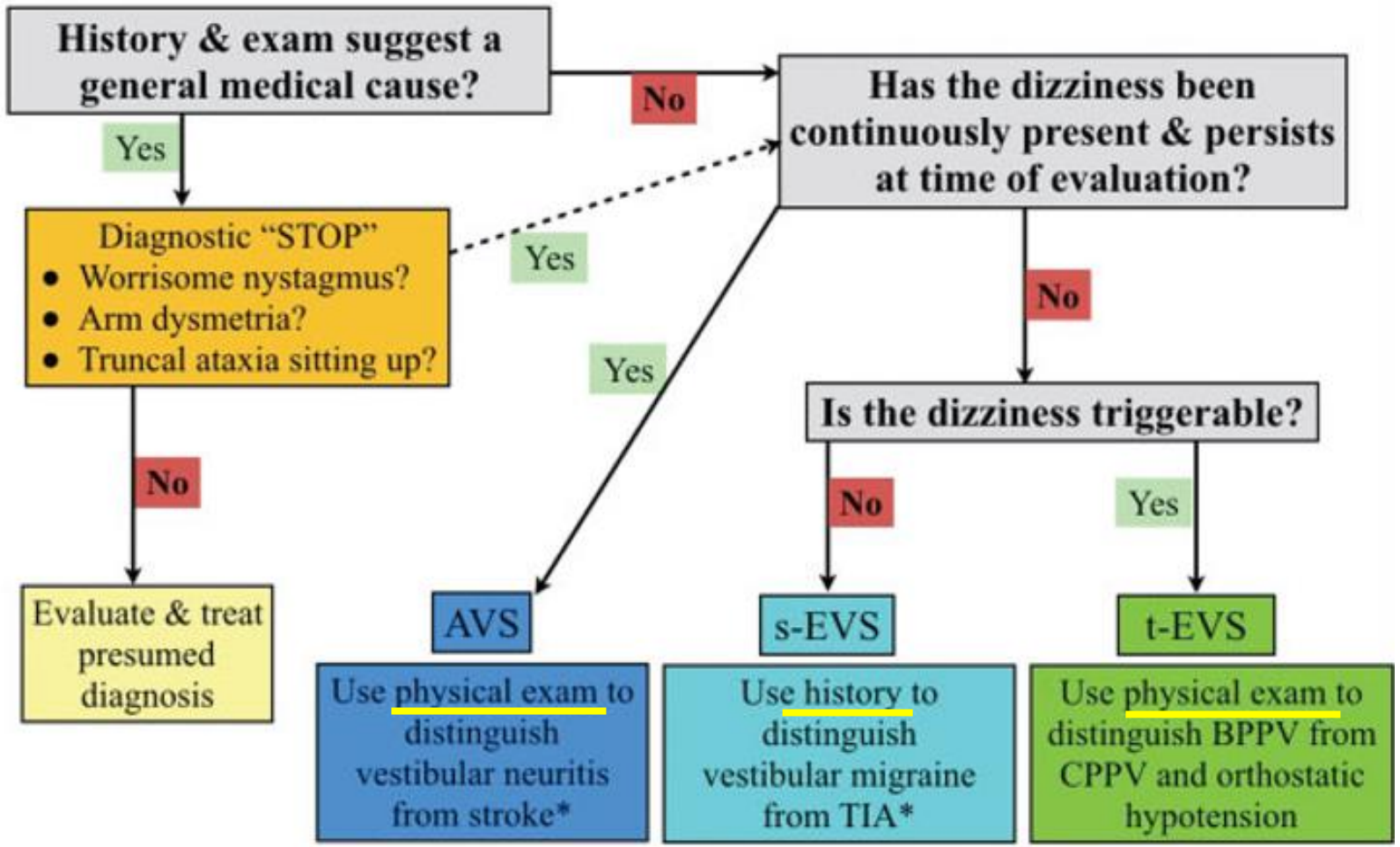
Pitfalls

- Isolated vertigo vs. stroke?
- How to D/Dx peripheral/ central vertigo?
- How to D/Dx benign/dangerous diseases?

DISCUSSION

Acute dizziness

ATTEST: Diagnostic Approach to the Acutely Dizzy Patient



- AVS, acute vestibular syndrome
- s-EVS, spontaneous episodic vestibular syndrome
- t-EVS, triggered episodic vestibular syndrome

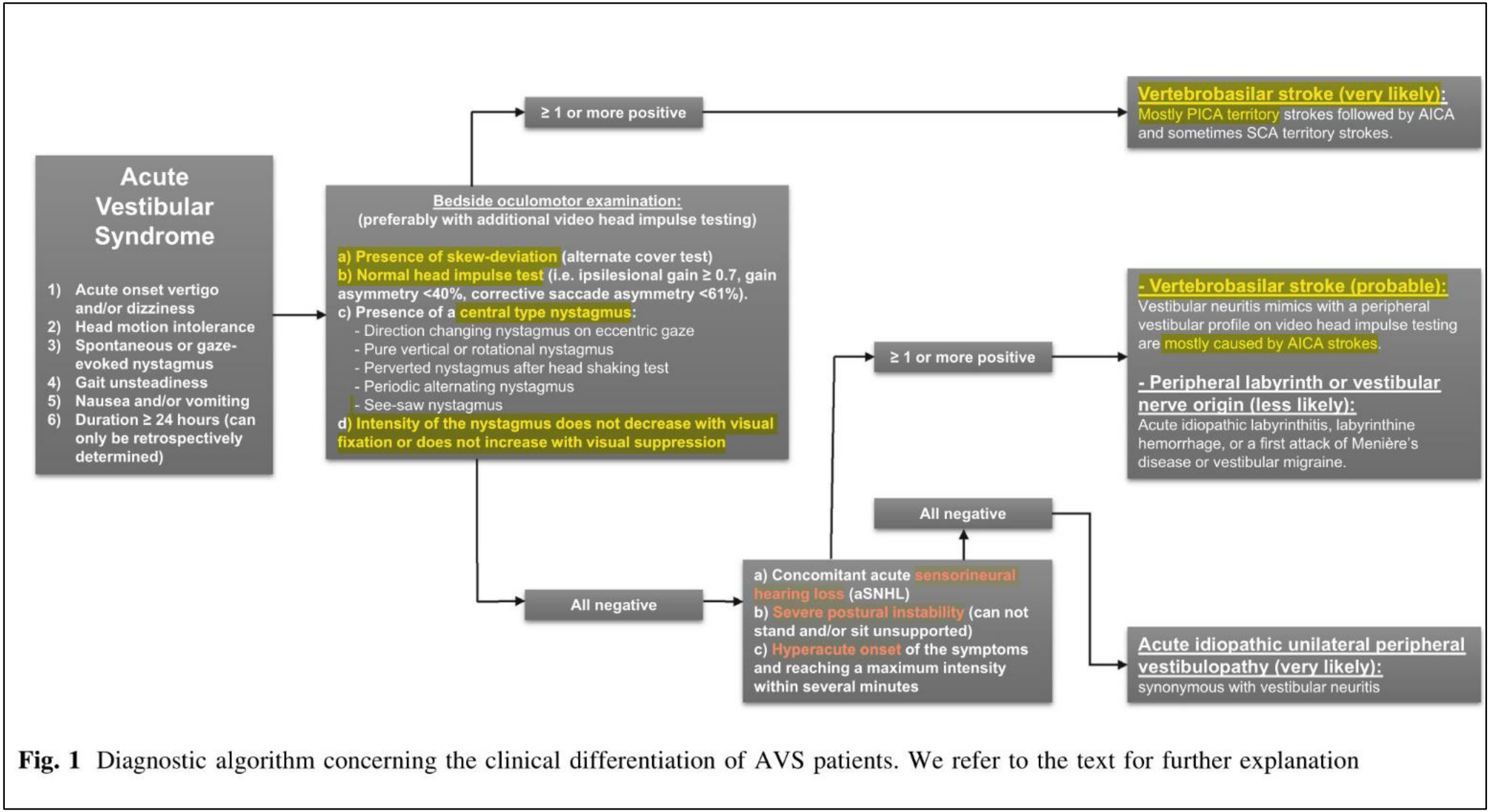


Fig. 1 Diagnostic algorithm concerning the clinical differentiation of AVS patients. We refer to the text for further explanation

Table 2 Timing-and-trigger-based "vestibular syndromes"[a] in acute dizziness

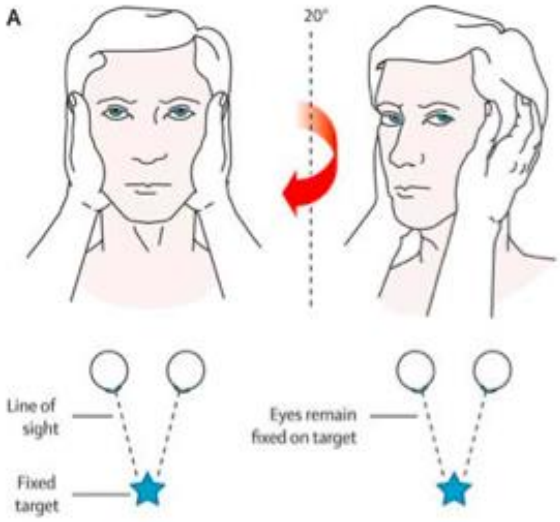
Syndrome	Description	Common benign causes	Common serious causes
AVS	Acute, continuous dizziness lasting days, accompanied by nausea, vomiting, nystagmus, head motion intolerance, and gait unsteadiness	• Vestibular neuritis • Labyrinthitis	• Posterior circulation ischemic stroke
s-EVS	Episodic dizziness that occurs spontaneously, is not triggered,[b] and usually last minutes to hours	• Vestibular migraine • Meniere's disease	• TIA
t-EVS	Episodic dizziness brought on by a specific, obligate trigger (typically a change in head position or standing up), and usually lasting less than 1 min	• BPPV	• CPPV • Orthostatic hypotension due to serious medical illness

Acute Vestibular Syndrome

- **Most common benign cause**
  - vestibular neuritis (dizziness only)
  - labyrinthitis (dizziness + hearing loss/ tinnitus)
- **Most frequent dangerous cause**
  - posterior circulation ischemic stroke
- **Other causes**
  - multiple sclerosis
  - cerebellar hemorrhage
  - thiamine deficiency
  - autoimmune, infectious, metabolic conditions

Posterior fossa strokes

- **Dizziness Pt at ED:** 3 - 5% cerebrovascular disease
- **AVS: 25%** cerebrovascular disease (96% ischemic)
- **Sensitivity of CT:** only 7-16% in the first 24 hours
- **Physical examination → HINTS plus**
  - Head Impulse
  - Nystagmus
  - Test of Skew
  - Hearing



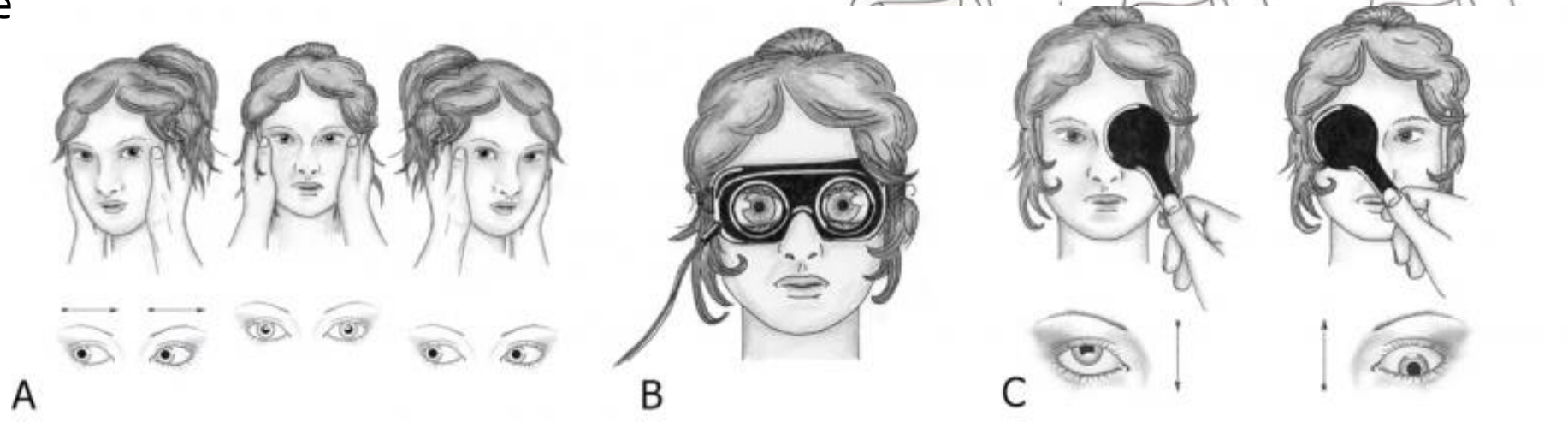


Table 3 Use of the physical examination to diagnose patients with acute vestibular syndrome (AVS)

Exam component	Peripheral (*all* must be present to diagnose vestibular neuritis)	Central (any *one* of these findings suggests posterior fossa stroke)
Nystagmus (straight-ahead gaze and rightward and leftward gaze)	Dominantly horizontal, direction-fixed, beating away from the affected side[a]	Dominantly vertical and/or torsional or dominantly horizontal, direction-changing on left/right gaze[b]
Test of skew (alternate cover test)	Normal vertical eye alignment and no corrective vertical movement (i.e., no skew deviation)	Skew deviation (small vertical correction on uncovering the eye)[c]
Head impulse test (HIT)	Unilaterally abnormal with head moving toward the affected side (presence of a corrective refixation saccade toward the normal side)[d]	Usually bilaterally normal (no corrective saccade)
Targeted neurological exam (see text)	No cranial nerve, brainstem, or cerebellar signs	Presence of limb ataxia, dysarthria, diplopia, ptosis, anisocoria, facial sensory loss (pain/temperature), unilateral decreased hearing
Gait and truncal ataxia	Able to walk unassisted and to sit up in stretcher without holding on or leaning against bed or rails	Unable to walk unassisted or sit up in stretcher without holding on or leaning against bed or rails

Spontaneous Episodic Vestibular Syndrome

- recurrent, spontaneous (non triggerable)
- **Most common benign cause:** vestibular migraine
- **Most common dangerous cause:** posterior circulation TIA
- **Other cause**
  - Meniere's disease
  - vasovagal syncope
  - panic attacks
  - cardiovascular (cardiac arrhythmia, unstable angina pectoris, pulmonary embolus)
  - endocrine (hypoglycemia, neurohumoral neoplasms)
  - toxic (intermittent CO exposure)

Table 4 Diagnostic criteria for vestibular migraine and differences between it and posterior circulation TIA

• At least 5 episodes with vestibular symptoms[a] of moderate[b] or severe intensity lasting between 5 min and 72 h
• Present migraine or previous history of migraine with or without aura (according to the International Classification of Headache Disorders)
• One or more migraine features with at least 50% of the vestibular episodes
o Headache with at least two of the following characteristics: unilateral location, pulsatile quality, moderate or severe pain, aggravation by routine physical activity
o Photophobia or phonophobia Nausea/ vomiting
o Visual aura
• No other vestibular explanation

Posterior Circulation TIA

- **5%** of TIA patients developed stroke **within 48h**

- Traditionally: isolated vertigo ≠ TIA symptom
  Evidently: **isolated dizziness = most common S/S** of **vertebrobasilar TIAs**

- **TIA causing dizziness/ vertigo: easily missed**
  - **BA occlusion:** 20% without other neurological symptoms
  - **VA dissection:** younger patients, mimics migraine, easily misdiagnosed

Posterior Circulation TIA *(Lancet Neurol 2013;12 (01):65–71)*

	Territory of stroke		OR (95%CI)	p value
	Vertebrobasilar	Carotid		
TNA in vertebrobasilar territory*	45/275 (16%)	10/759 (1%)	14·7 (7·3–29·5)†	<0·0001
0–2 days	22/252 (9%)	2/751 (<1%)	35·8 (8·4–153·5)	<0·0001
3–7 days	4/256 (2%)	2/753 (<1%)	6·0 (1·1–32·7)	0·04
8–90 days	19/275 (7%)	6/759 (1%)	9·3 (3·7–23·6)	<0·0001
TNA in uncertain territory‡	9/225 (4%)	17/717 (2%)	1·7 (0·8–3·9)	0·20
Definite TIA	5/221 (3%)	32/732 (4%)	0·5 (0·2–1·3)	0·16

TNA=transient neurological attack. TIA=transient ischaemic attack. Data are n/N (%) unless otherwise stated. *Isolated vertigo, vertigo plus other symptoms of any type, isolated double vision, transient generalised weakness, and binocular visual disturbance. Days indicate the time of the transient event before the stroke event. †This association was independent of age: OR 12·8 (95% CI 3·6–45·9) for ages <65 years; OR 14·7 (7·3–29·5) for ages 65–74 years; and OR 11·0 (4·6–26·4) for ages ≥75 years. ‡Isolated slurred speech, migraine variant, transient confusion, and isolated hemisensory tingling. Denominators are based on first TNA or TIA during the 90 days preceding stroke; therefore patients with TNAs or TIAs during the initial period were censored.

Table 2: **TNA or TIA in the 90 days preceding stroke by territory**

	Isolated vertigo (n=23)	All other vertebrobasilar TNA (n=22)
Age ≥60 years	17 (74%)	15 (68%)
Any vascular risk factors (diabetes mellitus, hypertension, smoking, or atrial fibrillation)	19 (83%)	11 (50%)
Previous vascular events (myocardial infarction, peripheral vascular disease, TIA, or stroke)	4 (17%)	9 (41%)
Any vascular medication*	12 (52%)	17 (77%)
Duration of symptoms		
<1 h	11 (48%)	15 (68%)
≥1 h	12 (52%)	7 (32%)

Data are number of patients (%) unless otherwise stated. TNA=transient neurological attack. TIA=transient ischaemic attack. *Any antiplatelet, statin, or antihypertensive.

Table 3: **Vascular risk factors and duration of isolated vertigo versus other vertebrobasilar TNAs preceding vertebrobasilar stroke**

Triggered Episodic Vestibular Syndrome

- **most common:** BPPV and orthostatic hypotension
- **dangerous causes:** central mimics of BPPV, serious causes of orthostatic hypotension

- **Central mimics of BPPV**
  - posterior fossa neoplasm, infarction, hemorrhage, demyelination
- **Orthostatic hypotension**
  - **SBP ↓ 20 mmHg; DBP ↓ 10 mmHg** within 3 mins of standing
  - MI, occult sepsis, adrenal insufficiency, DKA, …

Table 5 Physical examination maneuvers and findings that are used to diagnose and treat the typical forms of BPPV

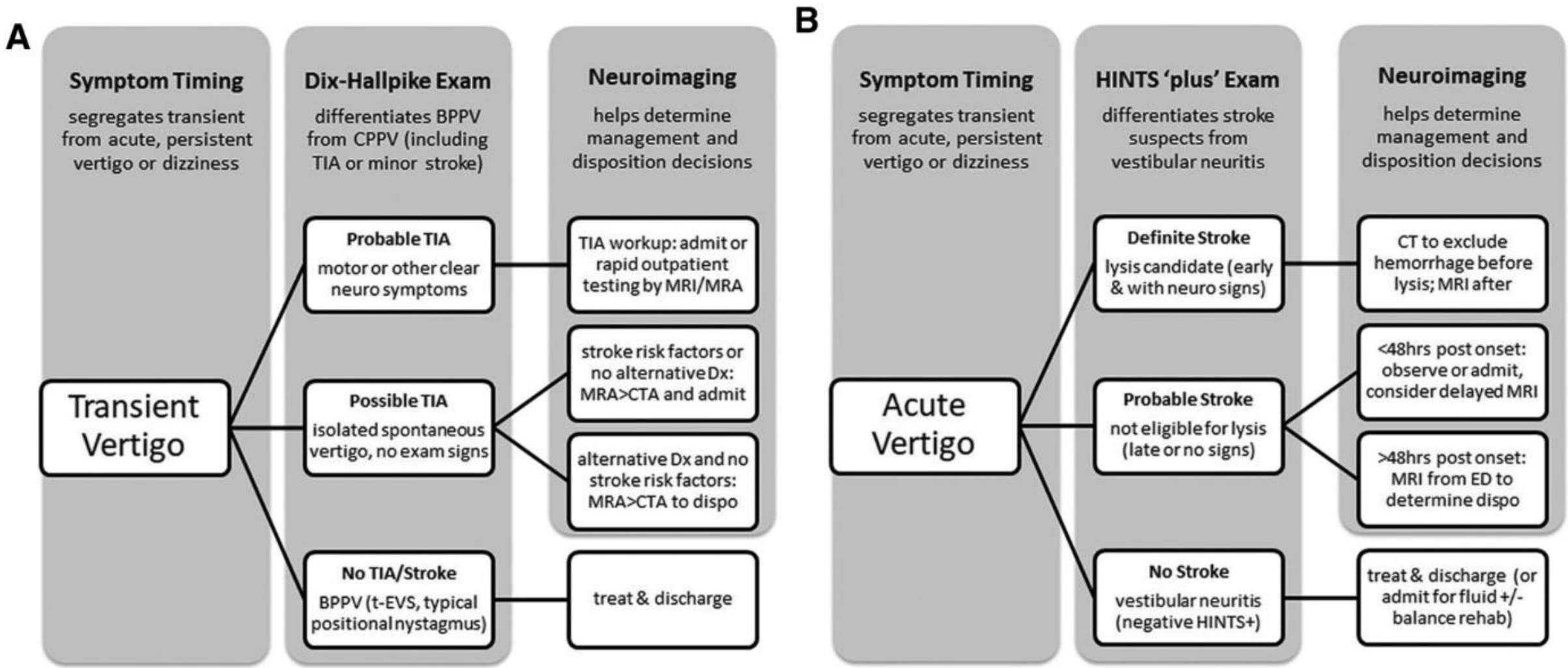
Canal involved, mechanism (proportion of BPPV cases)	Provocative diagnostic maneuver/test	Expected type of nystagmus[a]	Therapeutic maneuver
pc-BPPV (80–85%)	Dix-Hallpike	Up beating (from patient's perspective) and torsional[b]	Epley's maneuver Alternative: Semont's maneuver
hc-BPPV (15–20%; sometimes called lateral canal)			
Canalolithiasis (majority of horizontal canal cases)	Supine head roll	Geotropic (beats toward the floor) horizontal that is transient[c] Occurs on both sides, but is more intense on the *affected* side	Lempert's log roll maneuver Alternative: Gufoni's maneuver
Cupulolithiasis (minority of horizontal canal cases)	Supine head roll	Apogeotropic (beats toward the ceiling) horizontal, that is persistent Occurs on both sides, but is more intense on the *healthy* unaffected side	Gufoni's maneuver
sc-BPPV (~1–2%) (sometimes called anterior canal)	Dix-Hallpike	Downbeating vertical nystagmus[d]	Can use Epley but this form of BPPV usually resolves spontaneously

Table 7 Characteristics of patients with t-EVS that suggest a central mimic (CPPV) rather than typical BPPV

1. Presence of symptoms or signs that are *not* seen in BPPV
a. Headache
b. Diplopia
c. Abnormal cranial nerve or cerebellar function
2. Atypical nystagmus characteristics or symptoms during positional tests
a. Downbeating nystagmus[a]
b. Nystagmus that starts instantaneously, persists for longer than 90 s, or lacks a crescendo–decrescendo pattern of intensity
c. Prominent nystagmus with mild or no associated dizziness or vertigo
3. Poor response to therapeutic maneuvers
a. Repetitive vomiting during positional maneuvers
b. Unable to cure patient with canal-specific canalith repositioning maneuver[b]
c. Frequent recurrent symptoms

[a]Downbeating nystagmus can be seen with anterior canal BPPV. However, because BPPV of this canal is relatively rare and because downbeating nystagmus is most often the result of central structural lesions, it is safer for emergency physicians to consider this finding **always** worrisome prompting imaging and/or specialty consultation or referral.

[b]Modified Epley's maneuver or equivalent for posterior canal BPPV. Barbecue maneuver or equivalent for horizontal canal BPPV.


A
Symptom Timing
segregates transient from acute, persistent vertigo or dizziness
Dix-Hallpike Exam
differentiates BPPV from CPPV (including TIA or minor stroke)
Neuroimaging
helps determine management and disposition decisions
Transient Vertigo
Probable TIA
motor or other clear neuro symptoms
TIA workup: admit or rapid outpatient testing by MRI/MRA
Possible TIA
isolated spontaneous vertigo, no exam signs
stroke risk factors or no alternative Dx: MRA>CTA and admit
alternative Dx and no stroke risk factors: MRA>CTA to dispo
No TIA/Stroke
BPPV (t-EVS, typical positional nystagmus)
treat & discharge
B
Symptom Timing
segregates transient from acute, persistent vertigo or dizziness
HINTS 'plus' Exam
differentiates stroke suspects from vestibular neuritis
Neuroimaging
helps determine management and disposition decisions
Acute Vertigo
Definite Stroke
lysis candidate (early & with neuro signs)
CT to exclude hemorrhage before lysis; MRI after
Probable Stroke
not eligible for lysis (late or no signs)
<48hrs post onset: observe or admit, consider delayed MRI
>48hrs post onset: MRI from ED to determine dispo
No Stroke
vestibular neuritis (negative HINTS+)
treat & discharge (or admit for fluid +/- balance rehab)

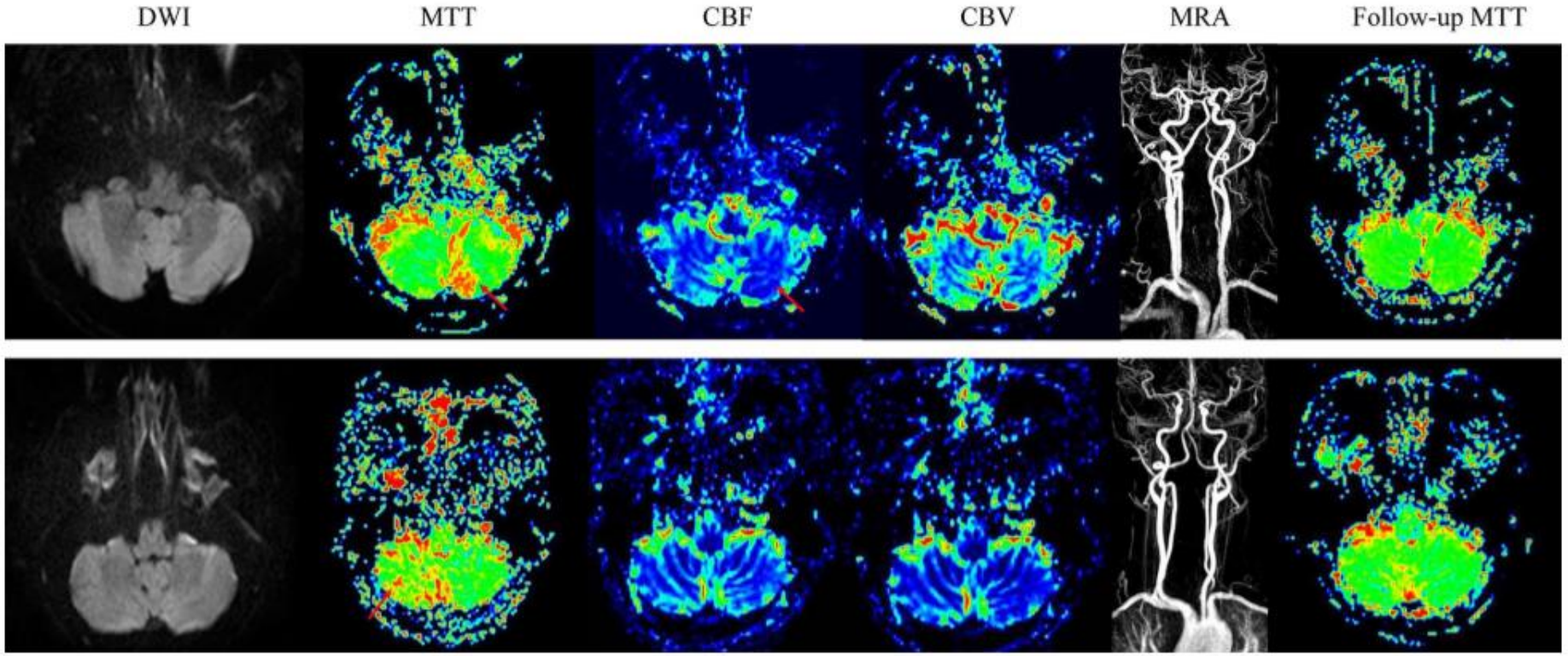


Fig. 1 Diffusion-weighted image (DWI, first panel), MR angiography (MRA, fifth panel), and initial (second, third, and fourth panels) and follow-up perfusion-weighted images (PWI, sixth panel) in 2 patients with acute transient vestibular syndrome due to cerebellar hypoperfusion. In the first patient, the initial PWIs reveal a prolonged mean transit time (MTT) and a reduced cerebral blood flow (CBF) in the left medial cerebellum without a diffusion restriction and cerebral blood volume (CBV) change. MRA shows a left vertebral artery (VA) hypoplasia. Follow-up PWI 3 days later discloses a normalized MTT in the left medial cerebellum. In the second patient, the initial PWI demonstrates a prolonged MTT in the right whole cerebellum without a diffusion restriction, CBF/CBV changes, and VA anomaly. Follow-up PWI 15 days later is normalized (adapted and modified from Ref. [9])

1. **perfusion-weighted imaging (PWI)** may help identifying strokes in ATVS of **unknown causes**
2. ATVS = Acute transient vestibular syndrome = resolution of acute vestibular symptoms and signs within 24 h

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THANKS
FOR
LISTENING