KEGAWATDARURATAN NEUROLOGI

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BACKGROUND

Pemeriksaan neurologis akan memberikan informasi yang tidak dapat diberikan oleh pemeriksaan penunjang. Meskipun kedudukannya penting tetapi **sering** dikesampingkan terutama pada keadaan emergensi

Sebelum melakukan pemeriksaan neurologis ada 3 hal penting yang perlu diingat dan dilakukan yaitu: anamnesis, anamnesis dan anamnesis.

ASESMEN NEUROLOGI

01 Pemeriksaan Neuroemergensi yang paling penting:
1. Tingkat kesadaran
2. Pupil dan gerakan bola mata
3. Tanda rangsang meningeal
4. Fungsi saraf-saraf kranial
5. Fungsi motorik dan refleks

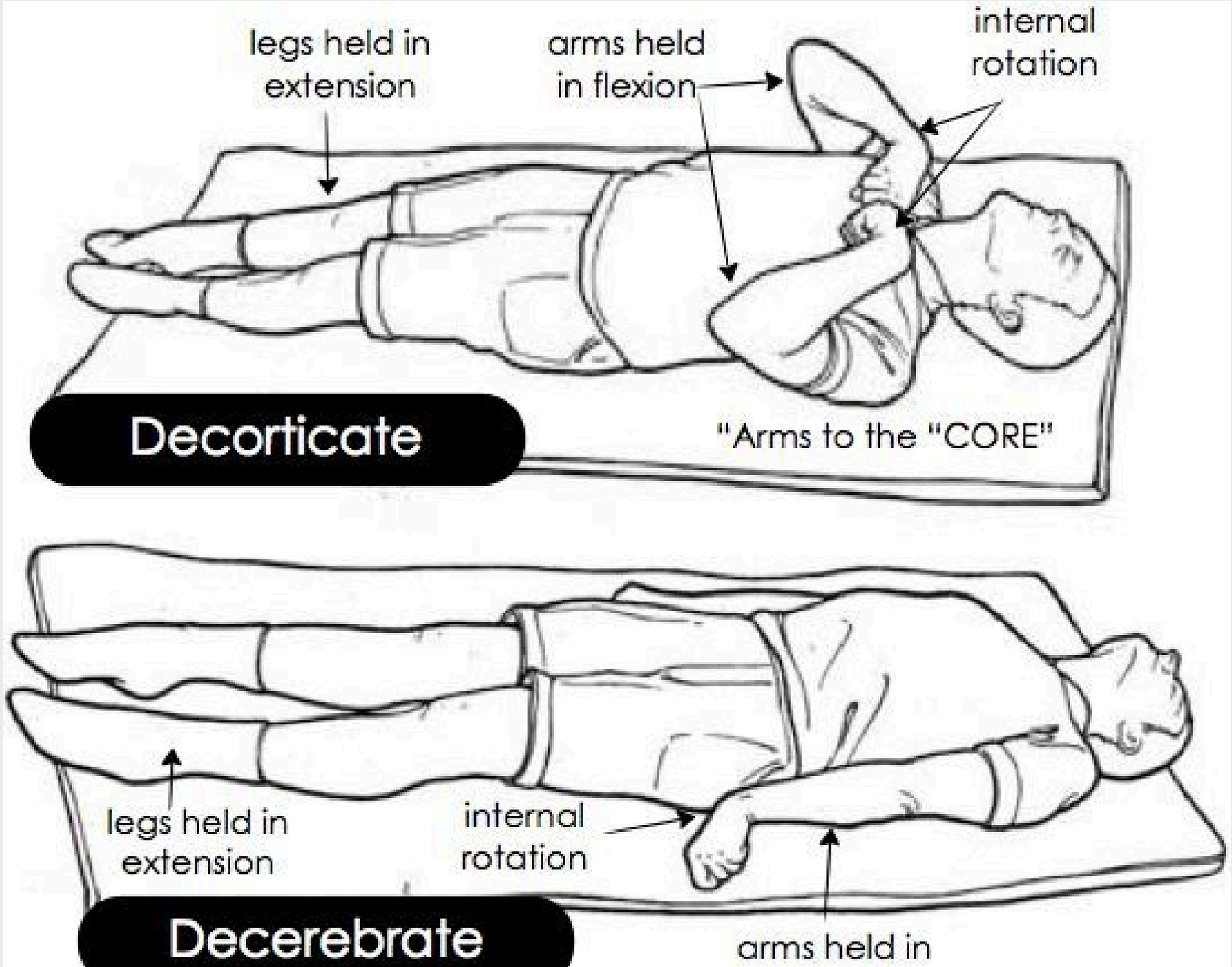
02 Pemeriksaan kegawatdaruratan neurologi dilakukan **bersamaan**, **segera** atau **setelah** dilakukan tindakan ABC

GCS

EYE (E)
Membuka mata spontan (4)
Membuka mata dengan (3)
Stimulus verbal
Membuka mata dengan (2)
Rangsang nyeri
Tidak membuka mata (1)

RESPON MOTORIK (M)
Dapat mengikuti perintah (6)
Dapat melokalisasi rangsang nyeri (5)
Tidak dapat melokalisasi rangsang nyeri, Fleksi menjauhi rangsang nyeri (4)
Dekortikasi (3)
Deserebrasi (2)
Tidak ada respon motorik (1)

RESPON VERBAL (V)
Orientasi tempat, waktu dan orang baik.
Konversasi seperti biasa. (5)
Disorientasi, confuse, tetapi masih dapat berbicara dalam bentuk kalimat. (4)
Kata-kata yang tidak berarti (3)
Hanya merintih atau mengerang (2)
Tidak ada respon verbal (1)


legs held in
extension
arms held
in flexion
internal
rotation
Decorticate
"Arms to the "CORE"
legs held in
extension
internal
rotation
Decerebrate
arms held in

M 3

M 2

DECORTICATE POSTURING

Arms flexed/adducted, wrists/fingers flexed; legs extended + plantar flexion

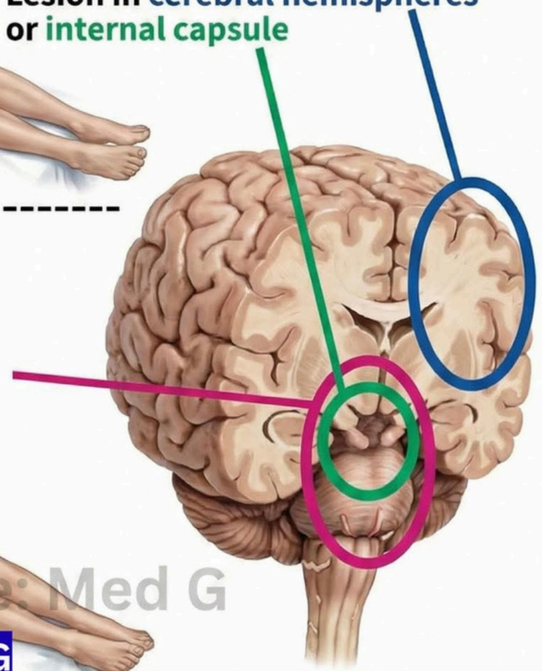
Common causes

- traumatic brain injury
- stroke (especially large hemispheric stroke)
- cerebral edema
- herniation syndrome (early)
- metabolic encephalopathy can rarely mimic

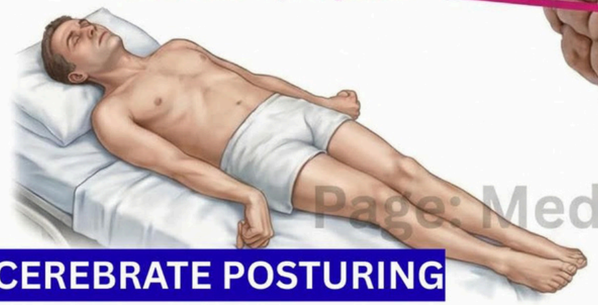
Prognosis

- usually better than decerebrate
- (because brainstem may still be intact)

Decorticate:
(abnormal flexion)
Lesion in cerebral hemispheres or internal capsule



Decerebrate:
(abnormal extension)
Lesion midbrain, brain stem, or pons



DECEREBRATE POSTURING

Arms extend/adduct + pronate; wrists flex; legs extended + plantar flexion

Common causes

- brainstem stroke
- brainstem compression
- uncal or tonsillar herniation
- severe TBI
- hypoxic-ischemic injury
- metabolic disorders (severe)

Prognosis

- worse (indicates brainstem involvement

🧠 Pathophysiology

- Decorticate: lesion above red nucleus → flexor pathways dominate
- Decerebrate: lesion below red nucleus → extensor pathways dominate

- So, decerebrate = more severe injury pattern.

PITFALLS

01 Komponen GCS **tdk dpt dinilai** misal, terintubasi, afasia, dll, nilai komponen tidak dinyatakan dlm angka TAPI ditulis T/ X

02 **Implikasi**: skor tidak dapat ditotal

Misal, pasien afasia global, E4M5Vx (afasia)

Bila penyebabnya karena sedasi: GCS tidak valid dinilai

GCS sebaiknya dijabarkan, perkomponen

KUALITATIF

01

- COMPOS MENTIS
- APATIS
- DELIRIUM
- SOMNOLEN
- SOPOR
- COMA

02

GCS 15 DENGAN:

- Gangguan atensi
- Gangguan konsentrasi

PUPIL

Pupillary reaction	Site of lesion/cause
Bilateral, small reactive 	Diencephalic (bilateral) injury or compression. All types of metabolic encephalopathy (so limited value in identifying structural causes of coma). Sedative drugs, except opiates.
Unilateral small pupil, normal reaction to light 	Left Horner's syndrome (sympathetic paralysis). Lesions involving descending sympathetic pathways in hypothalamus, midbrain, pontine tegmentum, medulla (e.g. lateral medullary infarction), ventrolateral cervical spine or carotid sheath (e.g. carotid artery damage). A unilateral dilated, reactive pupil may be secondary to parasympathetic paralysis.
Large, 'fixed', hippus (spontaneous and rhythmic fluctuation in pupil size). 	Dorsal tectal, pretectal or tegmental lesions. May occur in deep coma of any cause, particularly in barbiturate intoxication or hypothermia. Note: If there is hippus, the pupils may dilate in response to ipsilateral tactile stimulation of the neck/face/trunk (ciliospinal reflex). This is a normal phenomenon and distinguishes midbrain pupils from cases of brain death in which pupils are fixed, dilated and unresponsive.
Pinpoint, reactive (small range of contraction) 	1. Pontine lesions (haemorrhage) 2. Opiate intoxication
Mid-position, fixed (4–6 mm, often irregular) 	Midbrain injuries cause a wide range of pupillary abnormalities. Bilateral midbrain tegmental infarction, involving oculomotor nerves or nuclei bilaterally, results in large fixed pupils (if descending sympathetic tracts are preserved) or mid position (if they are not). Mid-position pupils may dilate with the ciliospinal reflex, distinguishing midbrain pupils from cases of brain death.
Unilateral dilated, and fixed 	IIIrd (oculomotor) nerve palsy ('down and out' appearance, not shown). Distal injury after oculomotor nerve leaves brainstem. The IIIrd nerve is vulnerable to damage by either uncul herniation ('coning') of the temporal lobe through the tentorial opening (a neurosurgical emergency) or posterior communication artery aneurysm.
Irregular oval, unequal pupils 	Brainstem transtentorial herniation which may then result in midbrain infarction.

ACUTE SYMPTOMATIC SEIZURE, EPILEPSI, STATUS EPILEPTIKUS?

Acute Symptomatic Seizure

Seizures are considered acute symptomatic if they occur **within 24 hours** in the presence of a severe metabolic disorder, **within seven days** after an **acute structural insult** to the brain such as a cerebrovascular event or a traumatic brain injury, or longer if there is evidence for an ongoing process that disturbs CNS integrity.

Epilepsy

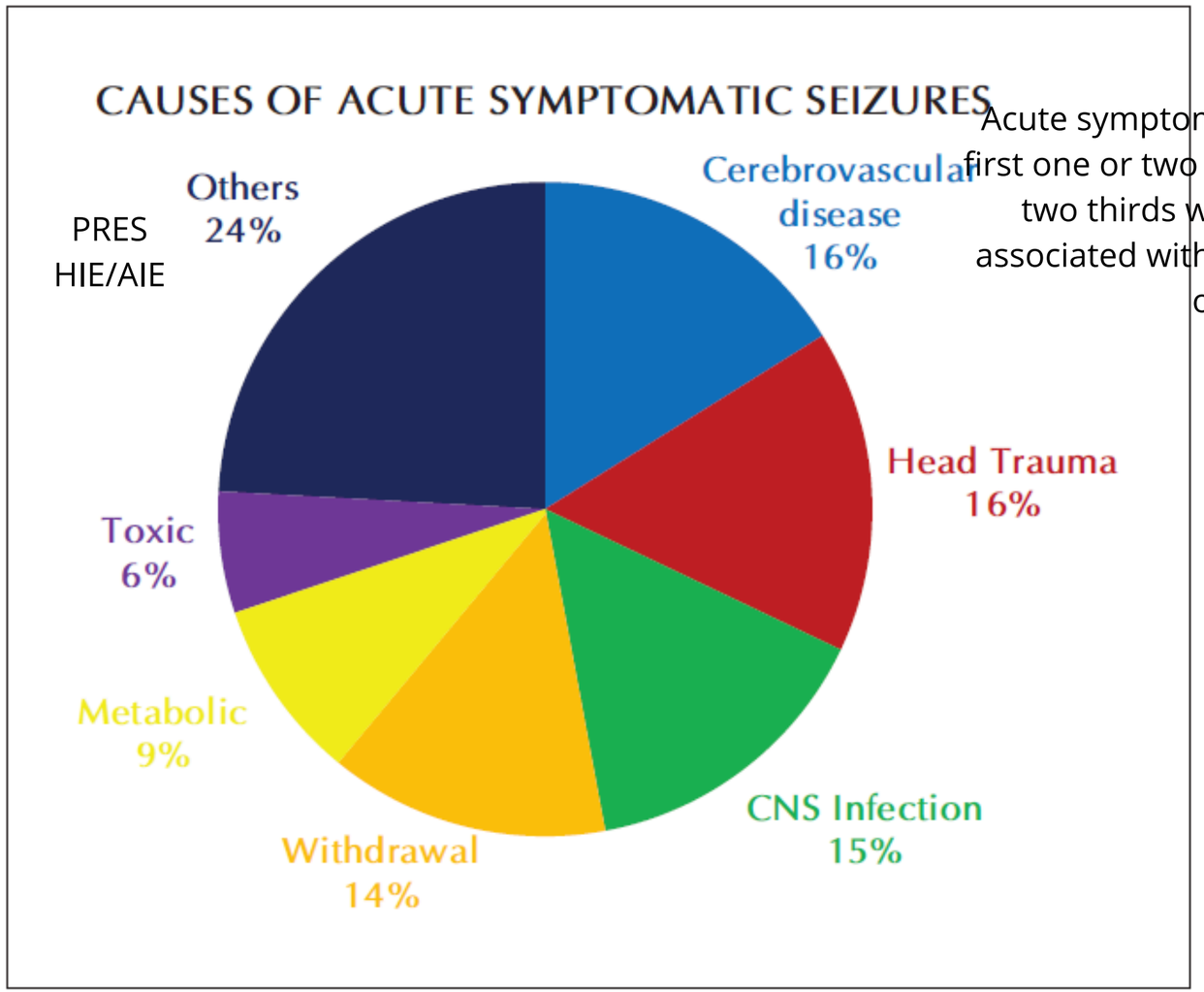
At least two unprovoked seizures **more than 24 hours** apart

One unprovoked seizure and has a **probability for the recurrence** of further seizures that is similar to the recurrence risk after two unprovoked seizures **(that is at least 60%) over the next 10 years**

Status Epilepticus

Status epilepticus occurs when a seizure **lasts more than 5 minutes or when seizures occur very close together and the person doesn't recover consciousness between them.**

ACUTE SYMPTOMATIC SEIZURE



Acute symptomatic seizures generally occur during the first one or two days after cerebral ischaemia, with about two thirds within the first 24 hours. Most seizures associated with haemorrhagic stroke occur at the onset or within the first 24 hours.

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ALGORITMA SE

Stabilisasi pasien (ABCD-PF neuro)
Catat durasi bangkitan, pantau TTV
EKG monitor
IV line
Cek GDS
Cek elektrolit, hematologi, skrining toksikologi

0-5 menit

Dzp iv (0.15-0.2mg/kgBB/dosis) iter 1x
Mdz im (10mg utk BB >40kg, 5mg utk BB <40kg) s.d

5-20 menit

2nd line ASM:
Fenitoin iv (15-18mg/kgBB kec. 50mg/mnt max 1500mg/dosis) s.d
As valproate po (40mg/kgBB max 3000mg) s.d
Levetiracetam po (60mg/kgBB, max 4500mg) s.d

Fenobarbital iv (15mg/kgBB) s.d

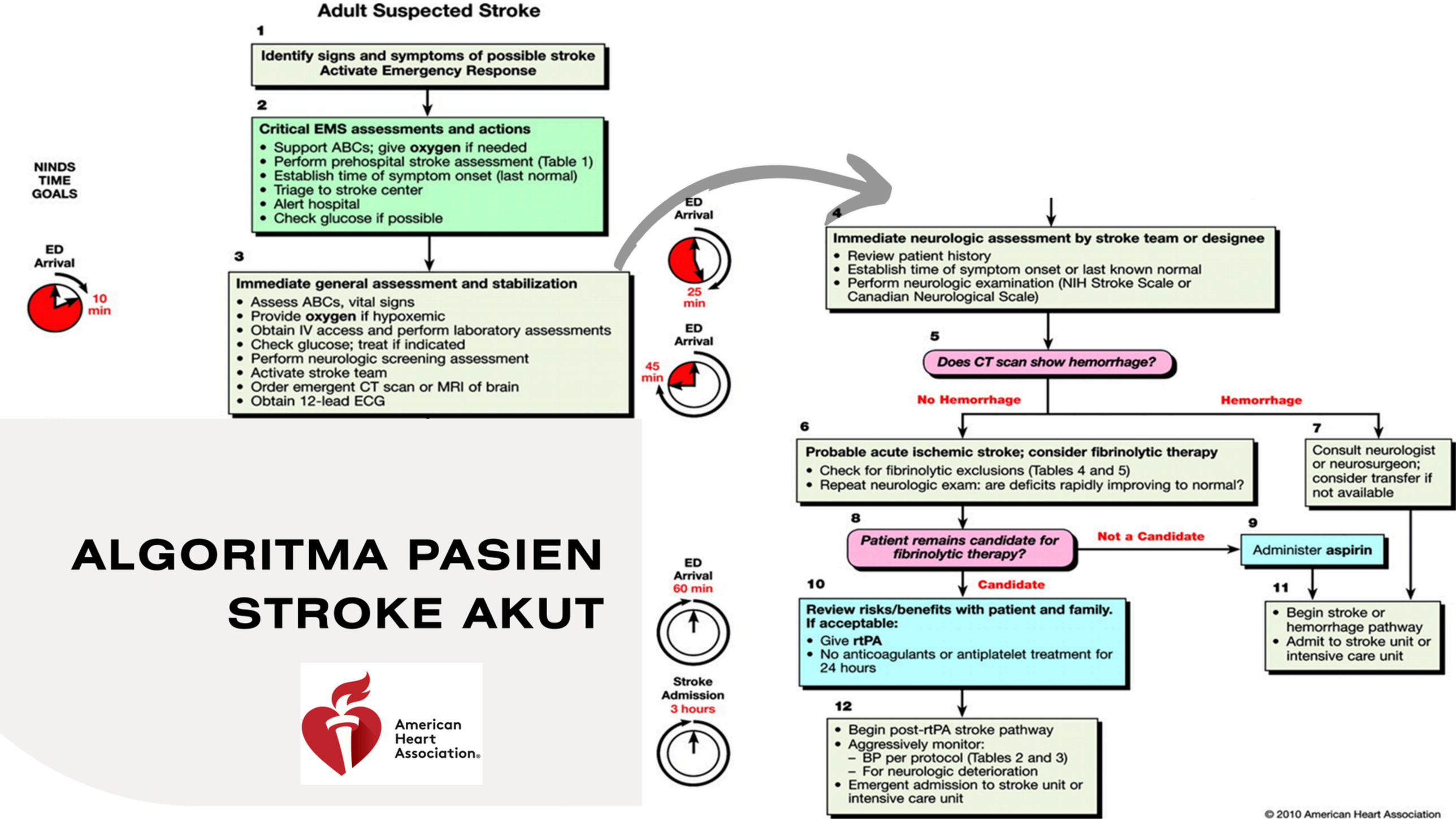
30-40 menit

2nd line ASM lainnya
3rd line ASM:
Topiramat po (200-400mg, lanjut 300-1600mg/hari dlm 2-4 dosis)

Fenobarbital iv (15mg/kgBB) s.d

40-60 menit

ALGORITMA PASIEN STROKE AKUT



MANAJEMEN HIPERTENSI PADA STROKE ISKEMIK AKUT

Table 2. Potential Approaches to Arterial Hypertension in Acute Ischemic Stroke Patients Who Are Potential Candidates for Acute Reperfusion Therapy

Patient otherwise eligible for acute reperfusion therapy except that blood pressure is >185/110 mm Hg

- Labetalol 10–20 mg IV over 1–2 minutes, may repeat ×1, or
- Nicardipine IV 5 mg/hr, titrate up by 2.5 mg/hr every 5–15 minutes, maximum 15 mg/hr; when desired blood pressure reached, lower to 3 mg/hr, or
- Other agents (hydralazine, enalaprilat, etc) may be considered when appropriate

If blood pressure is not maintained at or below 185/110 mm Hg, do not administer rtPA

Management of blood pressure during and after rtPA or other acute reperfusion therapy:

- Monitor blood pressure every 15 minutes for 2 hours from the start of rtPA therapy; then every 30 minutes for 6 hours; and then every hour for 16 hours

If systolic BP 180–230 mm Hg or diastolic BP 105–120 mm Hg

- Labetalol 10 mg IV followed by continuous IV infusion 2–8 mg/min, or
- Nicardipine IV 5 mg/h, titrate up to desired effect by 2.5 mg/hr every 5–15 minutes, maximum 15 mg/h

If blood pressure not controlled or diastolic BP >140 mm Hg, consider sodium nitroprusside

Table 3. Approach to Arterial Hypertension in Acute Ischemic Stroke Patients Who Are *Not* Potential Candidates for Acute Reperfusion Therapy

Consider lowering blood pressure in patients with acute ischemic stroke if systolic blood pressure >220 mm Hg or diastolic blood pressure >120 mm Hg

Consider blood pressure reduction as indicated for other concomitant organ system injury

- Acute myocardial infarction
- Congestive heart failure
- Acute aortic dissection

A reasonable target is to lower blood pressure by 15% to 25% within the first day

MANAJEMEN HIPERTENSI PADA STROKE HEMORAGIK AKUT

Expert consensus statements

- We suggest lowering systolic blood pressure **below 140 mmHg within 6 hours** of symptom onset in **minor or moderate ICH (haematoma volume < 30 mL**) to reduce haematoma expansion.
- For adults with spontaneous ICH, we suggest **avoiding** a reduction in systolic blood pressure of **more than 70 mmHg** from baseline and to avoid active reduction of systolic blood pressure below **110 mmHg**.
- **Caution** is advised when lowering very high systolic blood pressure (**>220 mmHg**), for patients with large haematoma volumes (**>30 mL**) or when there is planned haematoma evacuation.
- For adults with spontaneous **minor or moderate ICH** (haematoma volume < 30 mL), we suggest applying the following aspects:
  - Initiating antihypertensive treatment as early as possible, ideally within the first two hours following the onset of symptoms (**acute phase**):
    - Lower systolic blood pressure to **<140 mmHg** and **minimise variability** in blood pressure fluctuations.
  - After lowering systolic blood pressure below the target threshold (**up to 7 days**, sub-acute phase):
    - Maintain systolic blood pressure below 140 mmHg.
  - For secondary prevention (after sub-acute phase)
    - Follow the recommendations on secondary prevention, specifically section 6.1.1 on blood pressure management.

Vote: 15/15

DIZZINESS & VERTIGO

01

Dizziness is the sensation of disturbed or impaired spatial orientation without a false or distorted sense of motion. This includes sensations sometimes referred to as giddiness, lightheadedness, or non-specific dizziness, but does not include vertigo.

02

Vertigo is the sensation of self-motion (of head/body) when no self-motion is occurring or the sensation of distorted self-motion during an otherwise normal head movement. Incl. rotation (e.g., spinning, rocking), linear motion (e.g., feeling of falling downward, as in an elevator), or static tilt relative to gravity.

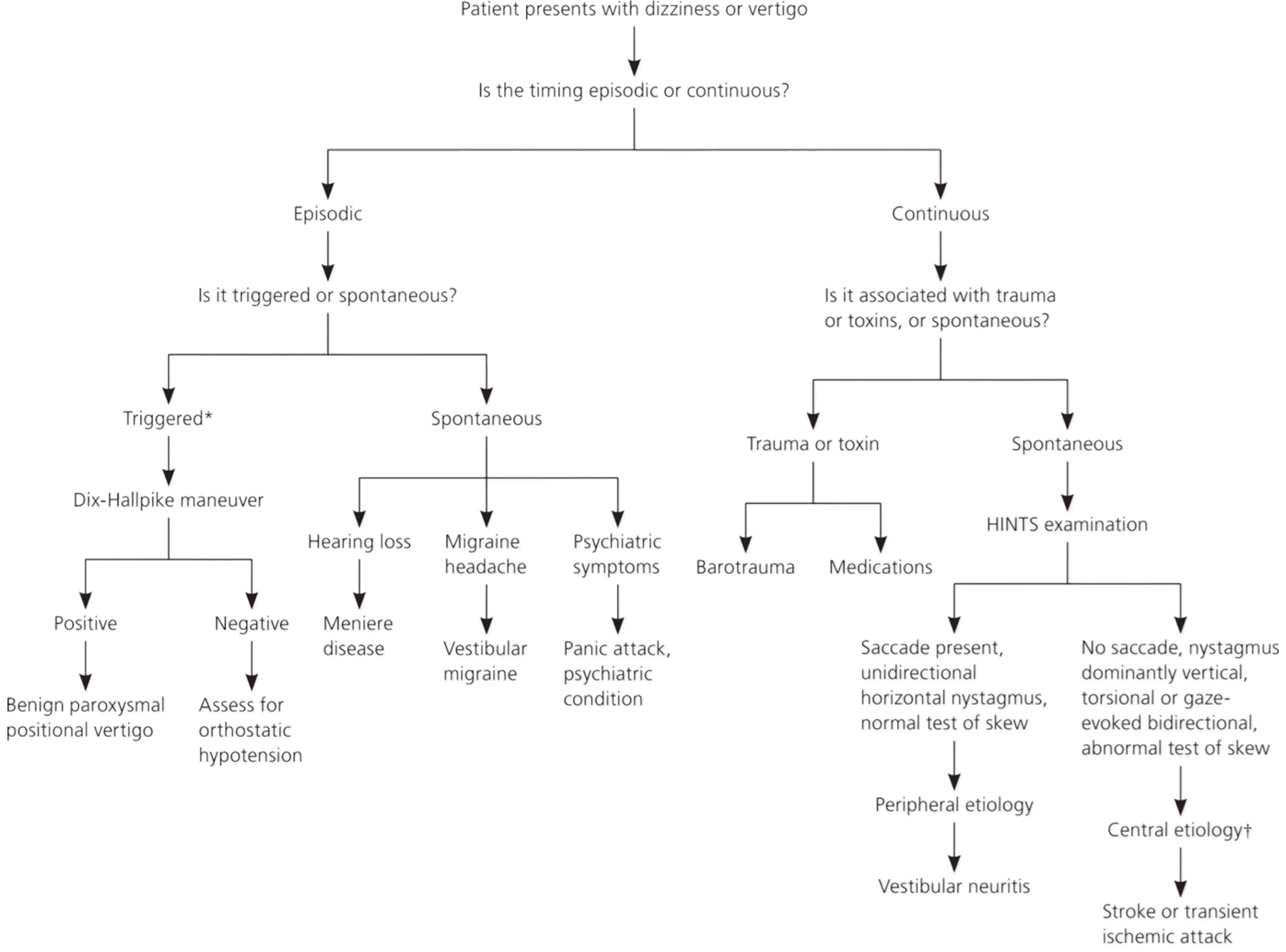
PRESYNCOPE & SYNCOPE

03

Presyncope (also near syncope or faintness) is the sensation of impending loss of consciousness. This sensation may or may not be followed by syncope.

04

Syncope (also faint) is transient loss of consciousness due to transient global cerebral hypoperfusion characterized by rapid onset, short duration, and spontaneous complete recovery. Syncope usually leads to loss of postural control and falling.



—Exacerbation of symptoms with movement does not aid in determining whether the etiology is peripheral vs. central.

†—Central causes can also occur with patterns triggered by movement.

	t-EVS	s-EVS	t-AVS	s-AVS	t-CVS	s-CVS
Tipe	Transient		Akut, persisten; continuous dizziness		Persisten	
Timing	Detik-menit	Detik-bbp hari (umumnya menit-jam)	Hari-minggu; sekuele (+)		Bulan-tahun; terkadang harian	
Triggers	Gerakan kepala Jarang – suara keras, manuver Valsava	Spontan Relied on anamnesis Fc persipitan (+/-)	Post exposure/Direct trauma/zat toksik	Spontan		
Target Exam	Dix-Hallpike	History taking: Migren, Meniere, TIA	History taking": mekanisme cedera; kontak zat toksik; obat-obatan	**HINTS** Tes pendengaran; nystagmus (-)	Nistagmus; Tes pendengaran	
Test	Canalith repos(BPPV) MRI (CPPV)	Workup for hipotensi ortostatik; NOT	Sesuai etiologi yg mendasari	Neuro workup	Neuroimaging	
Differential diagnosis	•BPPV, hipotensi ortostatik •CPPV, VBI	•Sindrom Meniere •TIA posterior	•Intox obat, CO •Posttraumatic vertigo •Wernicke ensefalopati	•Neuritis vestibuler •Labirintitis •Zoster/Ramsay-Hunt	•PPPD •Vertigo posttrauma kronik •Vestibulopati	Cerebellar/ brainstem syndrome

VERTIGO: PERIFER SENTRAL?

	PERIFER	SENTRAL
VERTIGO		
Onset	Akut-Gradual	Akut
Durasi	Menit-jam	Hari-minggu; persisten
Perubahan posisi	Memberat	(+/-)
Gejala auditorik	Sering	Bisa (+), seringnya (-)
Defisit neurologi lain	**(-)**	**(+); gejala sirkulasi posterior!**
NISTAGMUS		
Arah fase cepat	Unidireksional	Alternating; bidireksional
Orientasi	Horizontal; rotatoar	**Vertical upbeat is a RED FLAG!**
Fatigability	Fatigue (dalam 30-60 detik)	**Non fatigue; persisten!**
Gejala vertigo	Selalu ada	(+/-)
ATAKSIA		
Gait ataxia	(+), ringan-sedang	**(+) berat**
Truncal ataxia	Jarang	**Umum**
Cerebellar testing	**Normal**	Seringnya **abnormal**
Onset	Gradual	Akut
+ nyeri kepala	Jarang	Sering

THANK YOU