

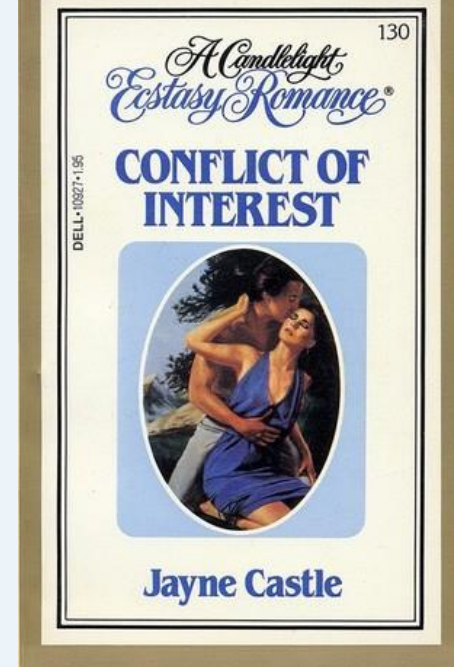
Fall 2021 Education Series- Forward Thinking About Posterior Stroke

Posterior Circulation Strokes: Identification, Recognition and Hyperacute Workup

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Disclosures-Acknowledgements

- Drs. Katsanos serves as the PI for the blooD prEssure management in sTroke following EndovasCular Treatment (DETECT) trial, funded by the New Investigator Fund from the Hamilton Health Sciences, and co-PI for the TRanscranial doppler Ultrasound after Endovascular Stroke Treatment (TRUEST) study, funded by the Division of Neurology Innovation Fund 2020, McMaster University.
- Serves as co-Chair for the OPTIMISE Research Committee.
- **Has been tricked by symptoms suggestive posterior circulation stroke several times during his training.**

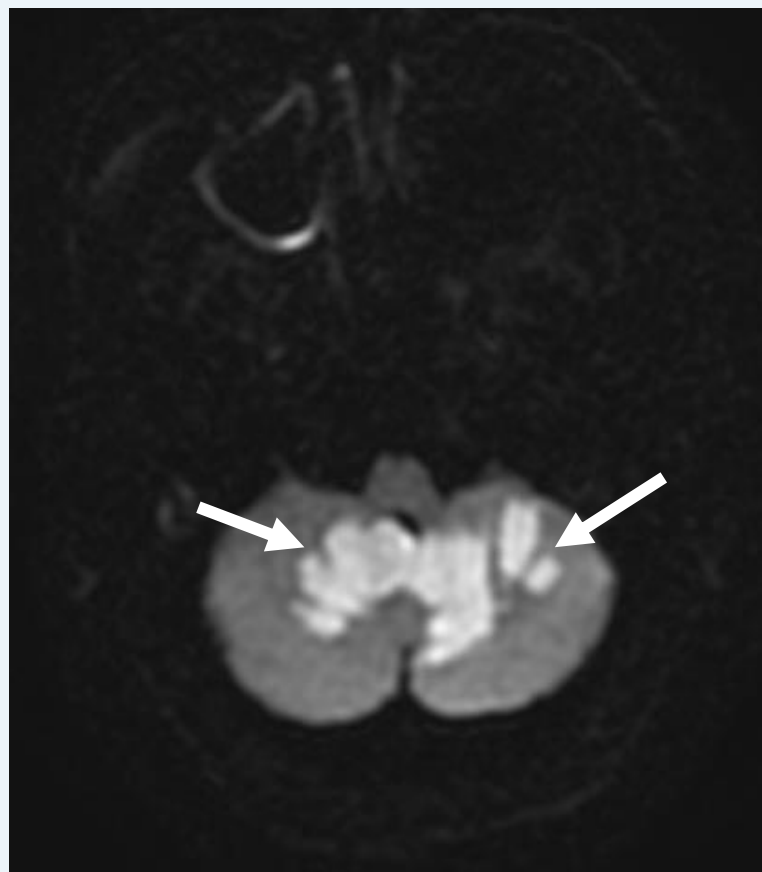


A case from last week

- 60 yo woman with history of hypertension, asthma, anxiety, GERD, CKD and orolingual cancer treated with chemotherapy and radiation therapy 7 years ago was transferred to the ED due to ongoing vertigo, nausea and vomiting.
- Patients reports the she experienced an acute onset posterior neck and occipital head pain 72 hours before her presentation to the hospital. The occipital headache started suddenly while she was sleeping at night and woke her up. Patient reports history of tension type headaches, but this one had a different intensity and characters.
- She took painkillers and the next morning when she woke up she had feelings of severe imbalance and unsteadiness, accompanied by severe vertigo with nausea and emesis.
- She experienced no visual symptoms, diplopia, numbness or weakness.
- Due to no improvement of the symptoms over the day and continuing emesis she called the EMS.
- Examination: right-sided ataxia, left beating horizontal nystagmus and hypometric saccades.



Non contrast CT



MRI – DWI sequence



CT angiography - carotids

Presentation outline

- Challenges in the diagnosis of posterior circulation stroke
- Anatomy of posterior circulation & mechanisms of posterior circulation ischemia
- Common posterior circulation stroke syndromes
- Diagnosis of posterior circulation stroke/TIA in the emergency setting
- Conclusions

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Posterior circulation stroke: a challenging diagnosis

- About 20-25% (range 17-40%) of the 150,000 ischaemic strokes in the United Kingdom each year affect posterior circulation brain structures.
- Early recognition of posterior circulation stroke or transient ischaemic attack (TIA) may prevent disability and save lives, but it remains more difficult to recognize and treat effectively than other stroke types.
- Delayed or incorrect diagnosis may have devastating consequences, including potentially preventable death or severe disability, if acute treatment or secondary prevention is delayed.
- Although in the past posterior circulation ischaemia was considered to have a lower recurrence risk than anterior circulation ischaemia, current data suggest that the risk is at least as high, if not higher.

1. Flossmann E & Rothwell PM. *Brain* 2003;126:1940-54.
2. Kuruvilla A, et al. *J Stroke Cerebrovasc Dis* 2011;20:523-7.

Non-consensus TIA

Vertigo only

Sudden onset of new non-recurrent isolated vertigo (with or without nausea or vomiting) not precipitated by head movement or trauma, and without associated ear pain, tinnitus, or hearing loss; cases with non-specific dizziness or light headedness are excluded

Ataxia only

Sudden onset of transient unsteadiness of gait without any other cause

Diplopia only

Sudden onset of transient isolated binocular double vision without an obvious ocular (eg, retinal detachment) or neuromuscular cause

Dysarthria only

Sudden onset of transient isolated slurred speech

Bilateral decreased vision only

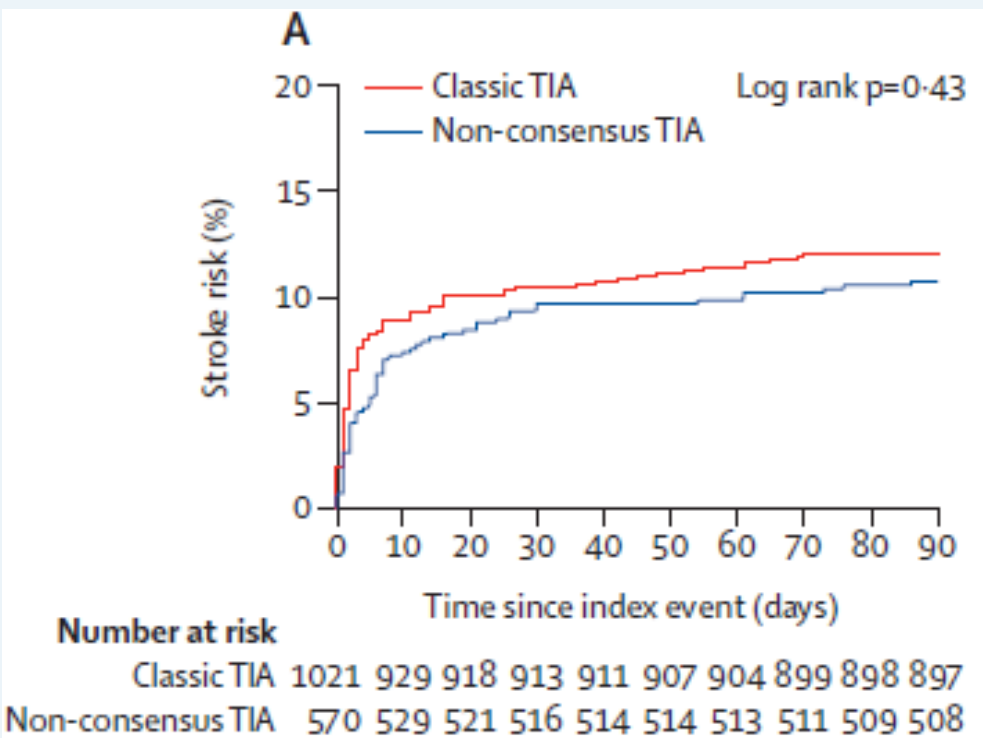
Sudden onset of transient isolated bilateral visual impairment (excluding hemianopia or quadrantanopia) without associated positive symptoms

Single segment sensory loss only

Sudden onset of transient isolated unilateral numbness in only one body segment (face, arm or hand, or leg) without march

	Intracranial or extracranial arterial stenosis $\geq 50\%$		p value
	Any anterior circulation	Any posterior circulation	
Classic TIA	193/896 (22%)	80/896 (9%)	<0.0001
Non-consensus TIAs	55/467 (12%)	84/467 (18%)	0.0001

- Patients with non-consensus TIA were **less likely than were those with classic TIA to be treated.**
- **Prescription of drugs for secondary prevention remained higher at 1-month follow-up for patients with classic TIA than for those with non-consensus TIA.**



Tuna MA & Rothwell PM. *Lancet*. 2021;397:902-912.

Posterior circulation stroke: a challenging diagnosis

- As many as 165,000 strokes/year may be misdiagnosed in US emergency departments
- Posterior strokes are **3 times more likely to be misdiagnosed!**
- According to the New England Medical Center Posterior Circulation Registry

Symptoms

- Dizziness(47%)
- **Unilateral limb weakness (41%)**
- **Dysarthria (31%)**
- Headache (28%)
- Nausea and Vomiting (27%)

Signs

- **Gait ataxia (31%)**
- **Unilateral limb ataxia (30%)**
- Nystagmus (24%)



Pazdera L et al. Arch Neurol 2021;69:346-351.



Four types of dizziness



Vertigo
strong sense
of motion or
spinning



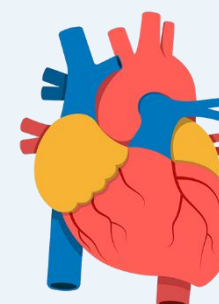
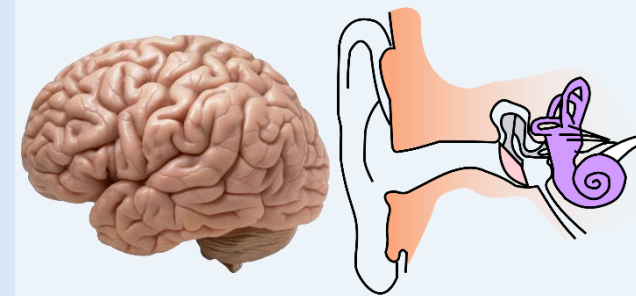
Disequilibrium
feeling off-balance,
unsteady or
wobbly



Lightheadedness
woozy
or disconnected
from environment



Presyncope
a feeling of losing
consciousness or
about to faint



Is the vertigo due to stroke ?

Consider stroke or TIA if:

- **Acute spontaneous onset** vertigo/imbalance
- **Patient cannot walk anymore**, even with help
- Associated with **acute hearing loss** (-> AICA)
- **New or unusual headache**
- Patients with vascular risk factors, elderly, cardiac sources
- Other **central symptoms** (patient) or **signs** (witness)
 - Hiccup
 - Dysarthria
 - New Horner
 - Mild long tract sign, etc.



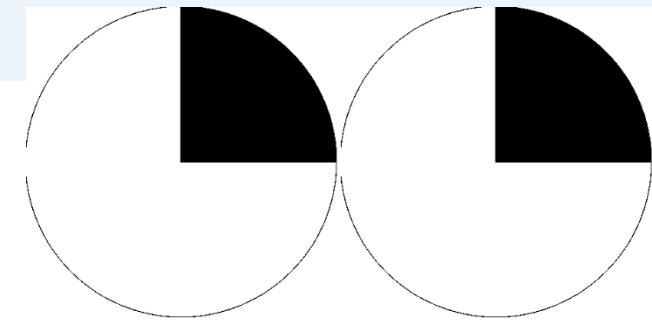
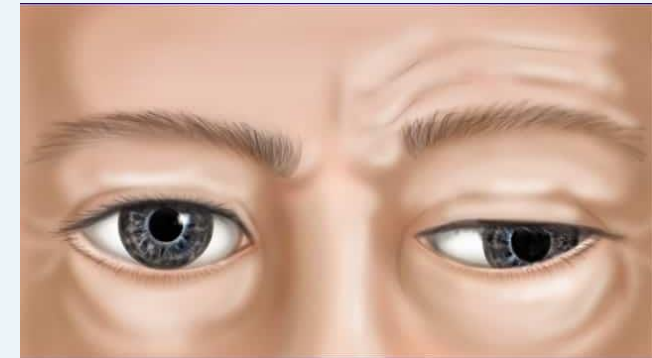
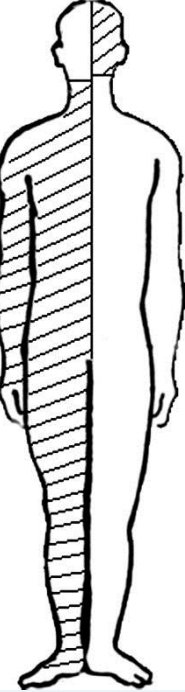
Posterior circulation stroke: a challenging diagnosis

In a large Chinese registry observational study of patients with posterior or anterior circulation stroke on confirmed magnetic resonance imaging, **the signs with the highest positive predictive values (PPV)** favouring a diagnosis of posterior circulation stroke were:

- **Crossed sensory deficits** (3.0% v 0%; $P < 0.001$; PPV 100%),
- **Crossed motor deficits** (4.0% v 0.1%; $P < 0.001$; PPV 92.3%)
- **Oculomotor (third) nerve palsy** (4.0% v 0%; $P < 0.001$; PPV 100%),
and
- **Quadrantanopia** (1.3% v 0%; $P < 0.001$; PPV 100%).

However, all of these signs had a **low sensitivity**, ranging from 1.3% to 4.0%

Tao WD, et al. Stroke 2012;43:2060-5.

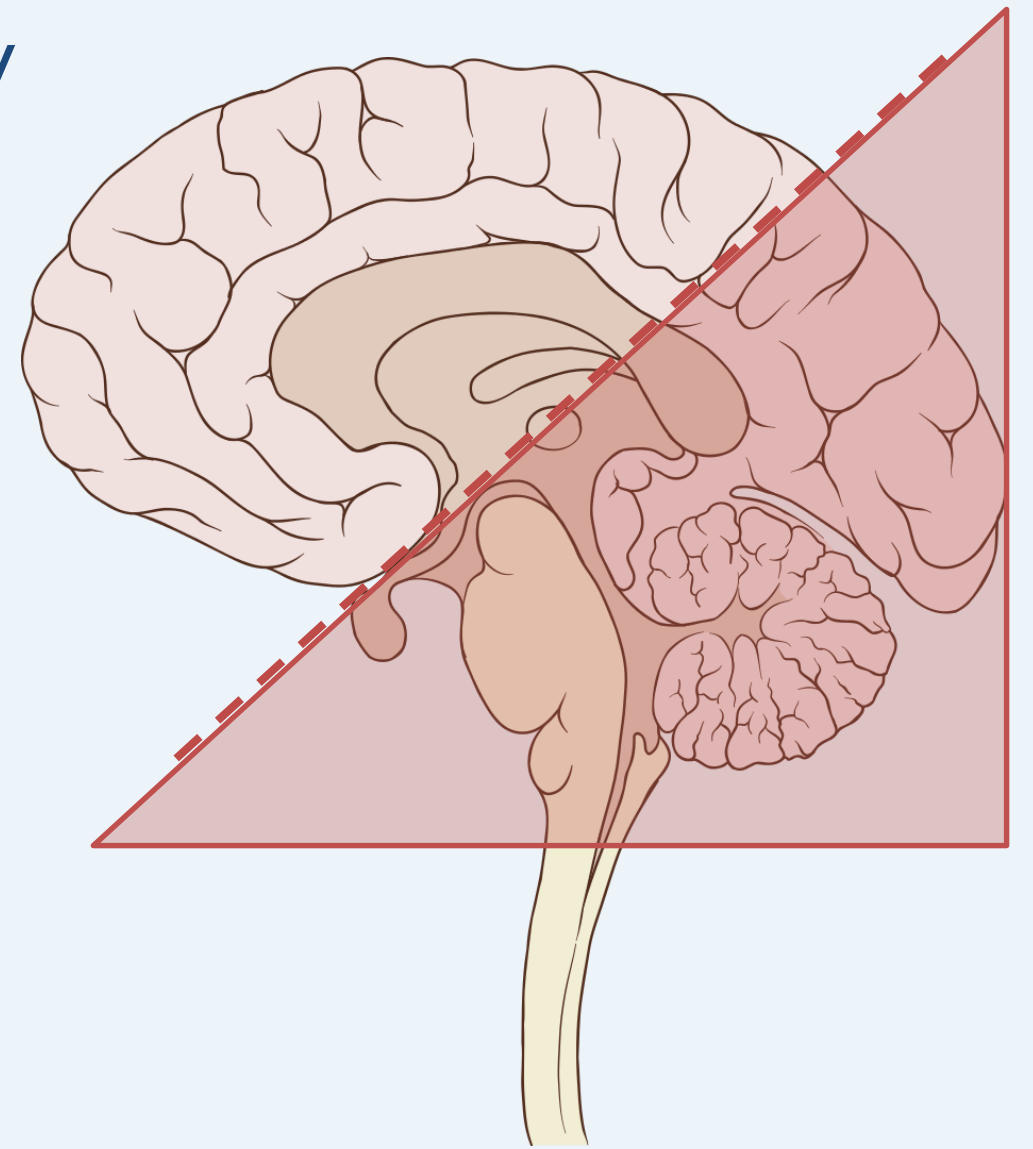


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Posterior circulation – Blood Supply

- ☐ Thalamus
- ☐ Hypothalamus
- ☐ Part of Temporal lobe
- ☐ Occipital lobe
- ☐ Brain stem
- ☐ Cerebellum
- ☐ Upper spinal cord

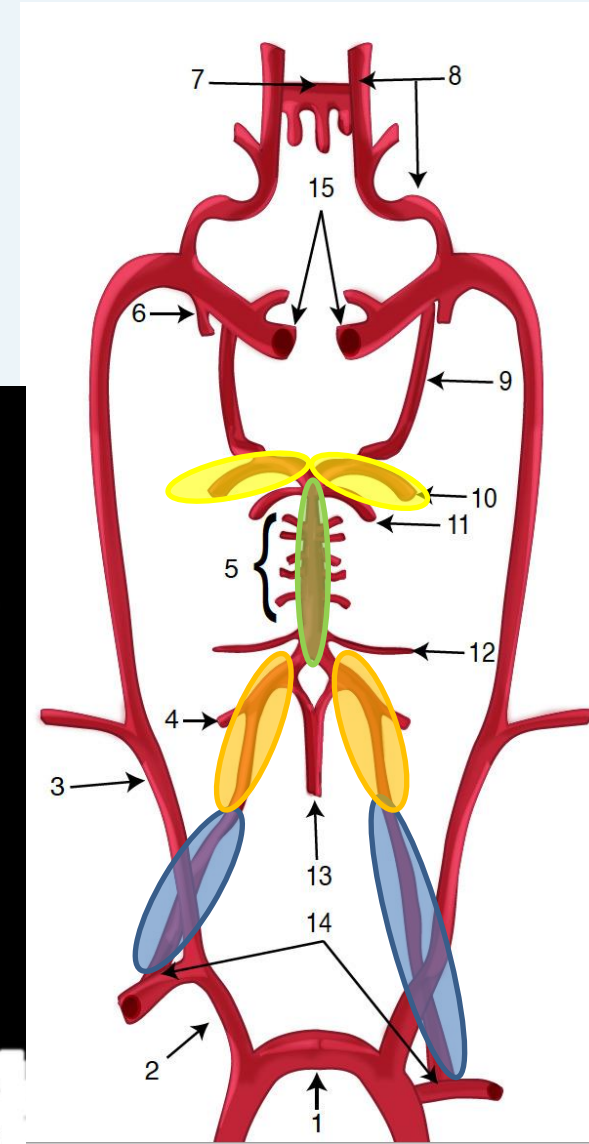
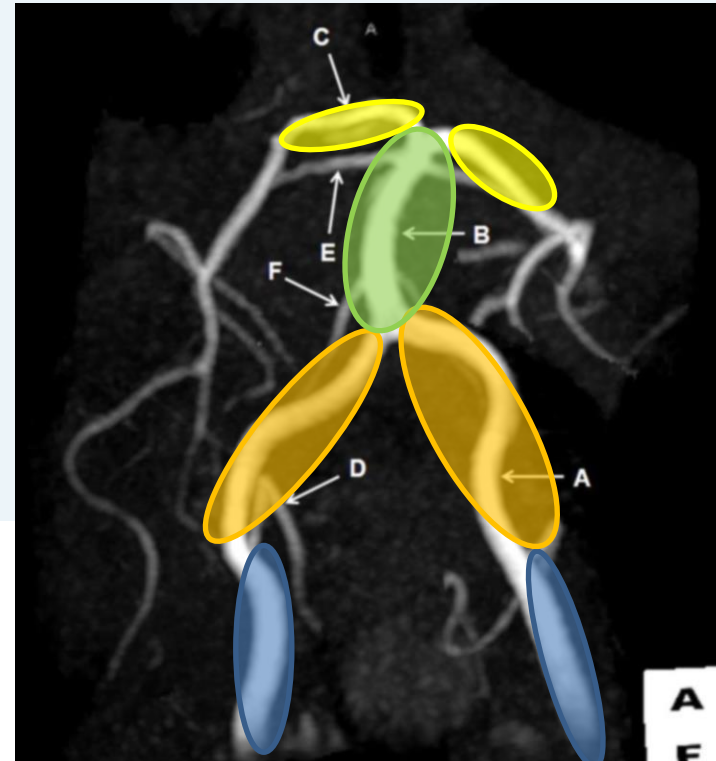


Posterior circulation vasculature

Posterior circulation ischaemic stroke is a clinical syndrome associated with ischaemia related to *stenosis*, *in situ thrombosis*, or *embolic occlusion* of the posterior circulation arteries:

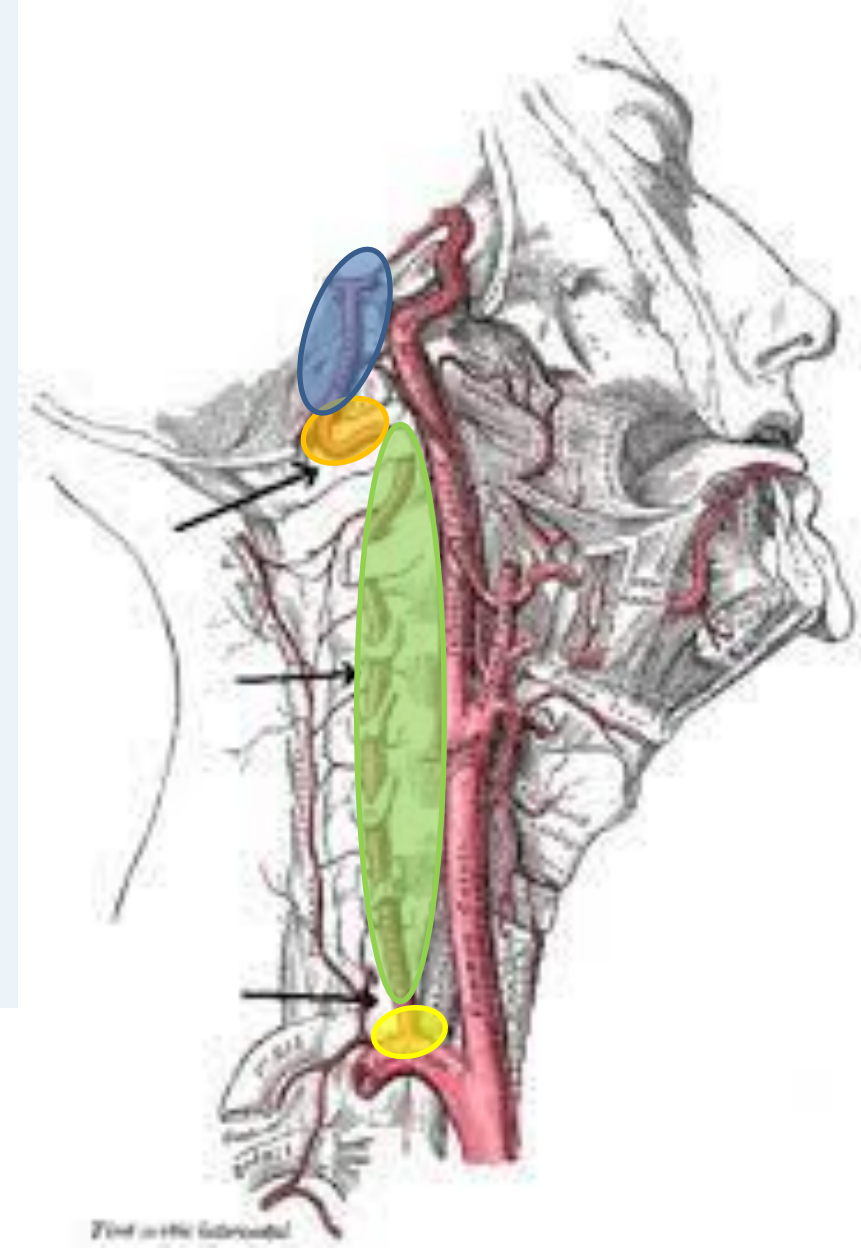
- The vertebral arteries in the neck,
- the intracranial vertebral arteries,
- The basilar artery,
- posterior cerebral arteries,
- and/or their branches

Merwick Á, Werring D. *BMJ*. 2014;348:g3175.



Vertebral artery anatomy

- ❖ **Segment 1 (V1):** takes off at the first branch of the subclavian artery until it reaches the foramina of C5-C6.
- ❖ **Segment 2 (V2):** runs within the transverse foramina from C5/C6 until C2.
- ❖ **Segment 3 (V3):** is the tortuous segment that starts at the transverse foramina of C2 and runs in the posterolateral part, looping around C1 and then passing between the atlas and the occiput. This segment of the artery is encased with nerves, muscles, and the atlanto-occipital membrane.
- ❖ **Segment 4 (V4):** is the intracranial segment; it pierces the dura at the foramen magnum runs until the junction of the medulla and pons; here it merges with the basilar artery.



Causes of posterior circulation strokes (I)

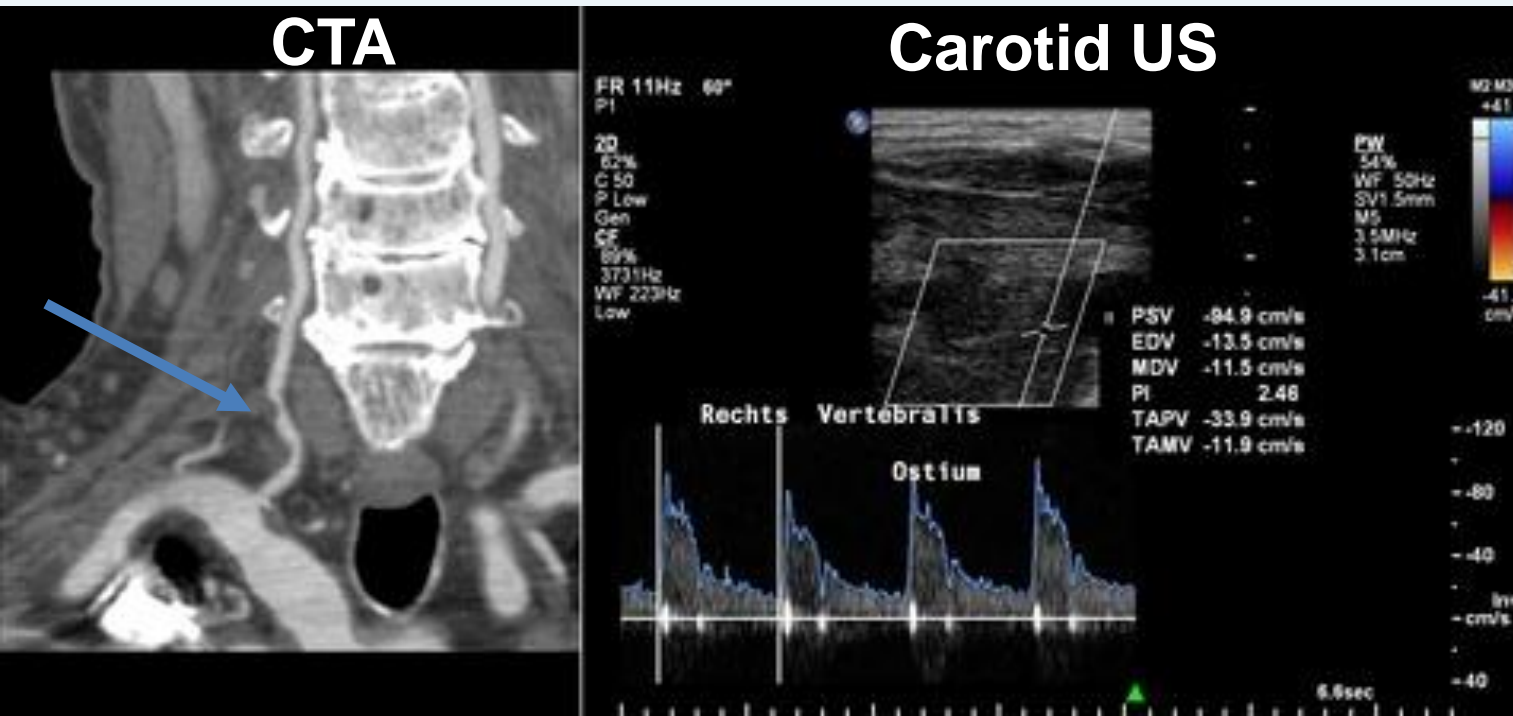
The most common causes of posterior circulation stroke are occlusion or embolism from large artery (vertebrobasilar atherosclerosis or dissection), and embolism from the heart. In a large US hospital registry study of 407 patients with posterior circulation stroke:

- **embolism was the most common mechanism (40% of patients)**
 - 24% had cardiac source,
 - 14% were caused by to artery-to-artery embolism, and
 - 2% had multiple sources of potential embolism
- **large artery occlusive lesions** caused haemodynamic brain ischaemia in **32%.**
- **28%** were due to **in-situ small vessel occlusion, other identified mechanisms** (e.g. dissection, vasculitis or dolichoectasia), or unknown causes.

1. Savitz SI, Caplan LR. *N Engl J Med* 2005;352:2618.
2. Caplan LR, et al. *Ann Neurol* 2004;56:389.

Causes of posterior circulation strokes (I)

- ❑ **Vessel stenosis:** Recent population based and hospital observational studies have shown a **3-fold increased risk of stroke after posterior circulation TIA or minor stroke in patients with symptomatic vertebrobasilar stenosis** than in those without stenosis.



Rozeman AD, et al. Brain Behav. 2017;7:e00750.

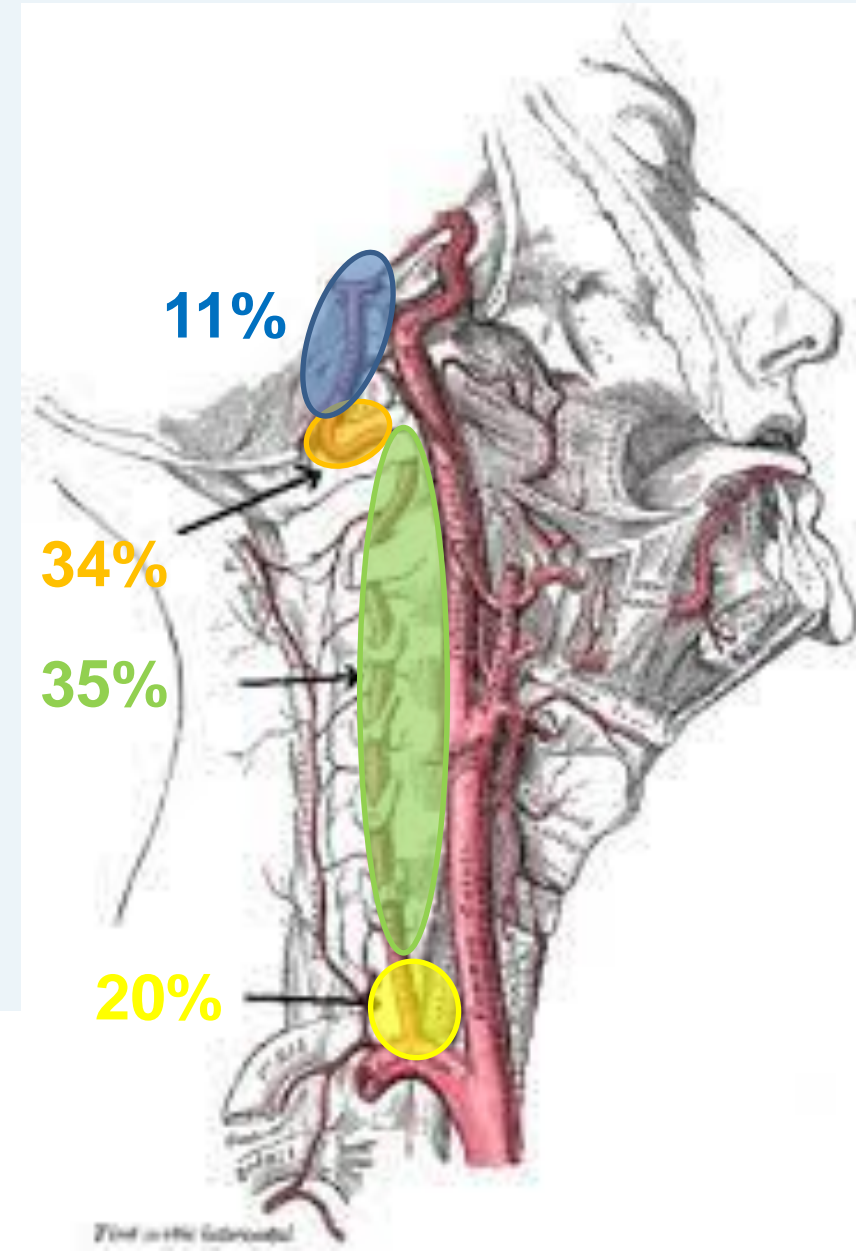
Causes of posterior circulation strokes (II)

Dissection: Extracranial vertebral artery dissection is an important cause, especially in young patients; it may be painless and **usually occurs without a clear history of trauma**. The most common symptoms were dizziness or **vertigo** (58%), **headache** (51%), and **neck pain** (46%).

The annual incidence of spontaneous vertebral artery dissection is estimated at 1-1.5 per 100,000 per year.

Complications:

- ✓ Cerebellar and brain stem infarction
- ✓ Subarachnoid hemorrhage
- ✓ Vertebral artery pseudoaneurysm, leading to compression neuropathy of the cranial nerves.



Causes of posterior circulation strokes – dissection (III)

- It is estimated that vertebral artery dissection is the cause of approximately 2% of all ischemic strokes. However, in middle-aged and younger patients (30 to 45 years of age), it is believed to be as high as 10% to 25%.
- From a study of 169 patients with spontaneous vertebral artery dissection (sVAD) in Switzerland:
 - ❑ **mean age, 43±9**; median, 43; range, 21–69 years
 - ❑ **15%** were found to have with **bilateral sVAD**
 - ❑ Median time interval from symptom onset to diagnosis was 4 days (range, 2 hours to 88 days)
 - ❑ **8%** had asymptomatic **sVAD**
 - ❑ **Presenting clinical symptoms** were:
 - **ischemic stroke** in 114 (**67%**),
 - **TIA** in 17 (**10%**),
 - **occipital head and/or neck pain** alone in 21 (**12%**) patients,
 - **SAH** without ischemia in three (**2%**) patients, and
 - sensorimotor cervical radiculopathy C5/C6 in one patient (1%)

Arnold M, et al. Stroke. 2006;37:2499-503.

Causes of posterior circulation strokes – dissection (IV)

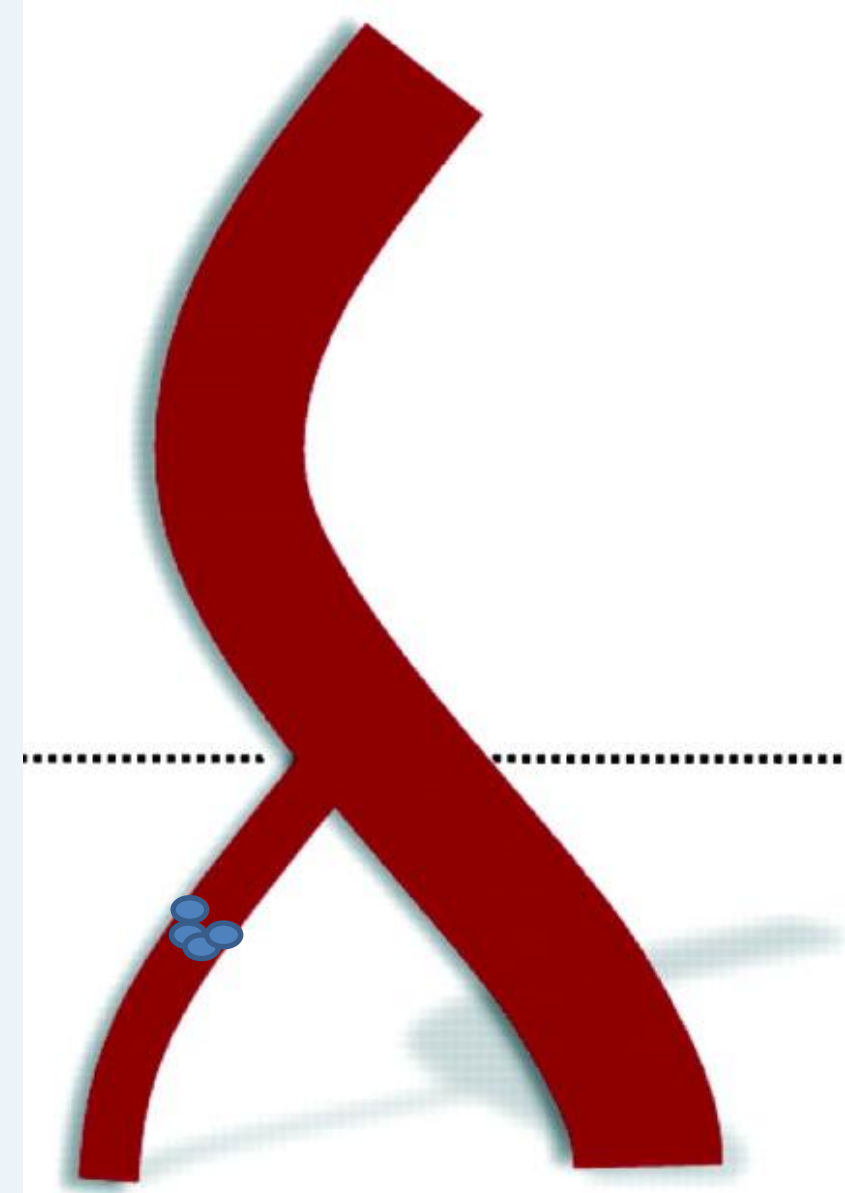
- Of the patients 114 patients with ischemic stroke due to sVAD three (2.6%) showed also **signs of SAH on brain imaging**.
- A total of 118 (88%) of 134 patients with ischemic or hemorrhagic symptoms had also occipital head and/or neck pain and seven patients (5%) a **pulsatile tinnitus**.
- **Median NIHSS score on admission** in patients with ischemic stroke was **3 (range, 1–35)**. Of the 114 patients with ischemic stroke.
- 15 (13%) of the patients with acute ischemic stroke **had a TIA before stroke**. **Median time interval from TIA to stroke onset was 1 day** (range, 1 hour to 17 days)



Arnold M, et al. Stroke. 2006;37:2499-503.

Vertebral artery hypoplasia

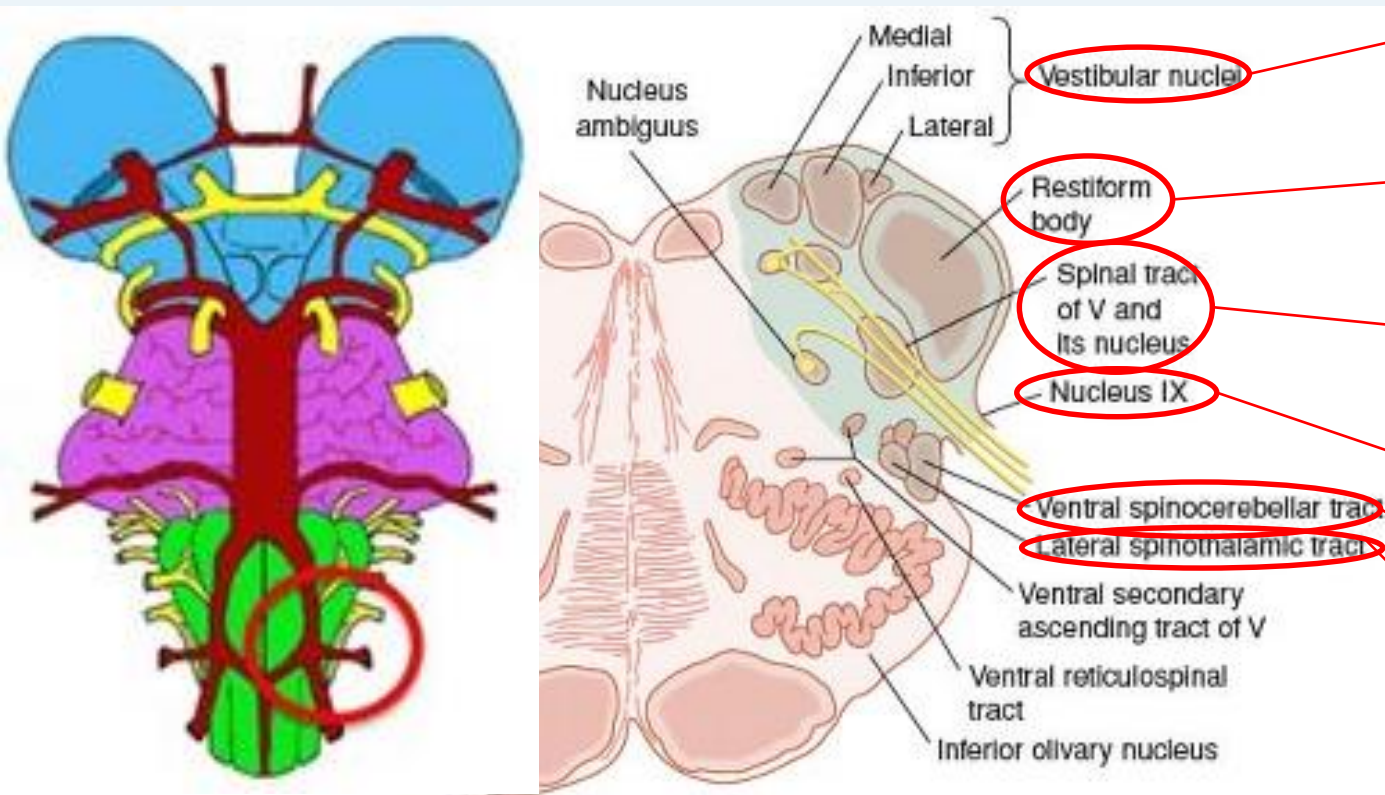
- Congenital anatomical variations of both vertebral arteries are relatively frequent; **left vertebral artery dominance presents in 50% of the population**, while similar size vertebral arteries present with an only 25% prevalence.
- **Vertebral artery hypoplasia is 2-times more prevalent in patients with posterior circulation ischaemia** compared to anterior circulation ischaemia (risk ratio = 1.81, 95%CI: 1.58–2.06).
- Small diameter arteries have also been reported to be more vulnerable to stenosis or occlusion, as its low flow velocity predisposes to **prothrombotic or atherosclerotic processes** in the presence of conventional vascular risk factors, while the **increased vessel diameter of the contralateral to the hypoplastic VA could provide a route prone to the transfer of cardiac emboli** due to its low resistance and increased blood flow.



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Lateral medullary (Wallenberg's) syndrome



Lateral medullary infarction (Wallenberg syndrome) is the most common syndrome related to intracranial vertebral artery occlusion. **It is often missed or misdiagnosed.**

Vestibulocerebellar symptoms	Nystagmus Vertigo D iplopia Tinnitus
Proprioception	Contralateral loss of vibration & proprioception
Sensory symptoms (face)	Ipsilateral loss of facial pain & temperature
Bulbar muscle weakness	D ysphagia D ysarthria
Cerebellar symptoms	Ipsilateral D ysmetria
Sensory symptoms (body)	Contralateral loss of body pain & temperature
Sympathetic fibers	Ipsilateral Horner's syndrome

Cuoco JA, et al. Edorium J Neurol 2016;3:4–16.

Medial medullary (Déjerine) syndrome – Anterior Spinal Artery occlusion

A. IPSILATERAL

1. Hypoglossal nerve palsy

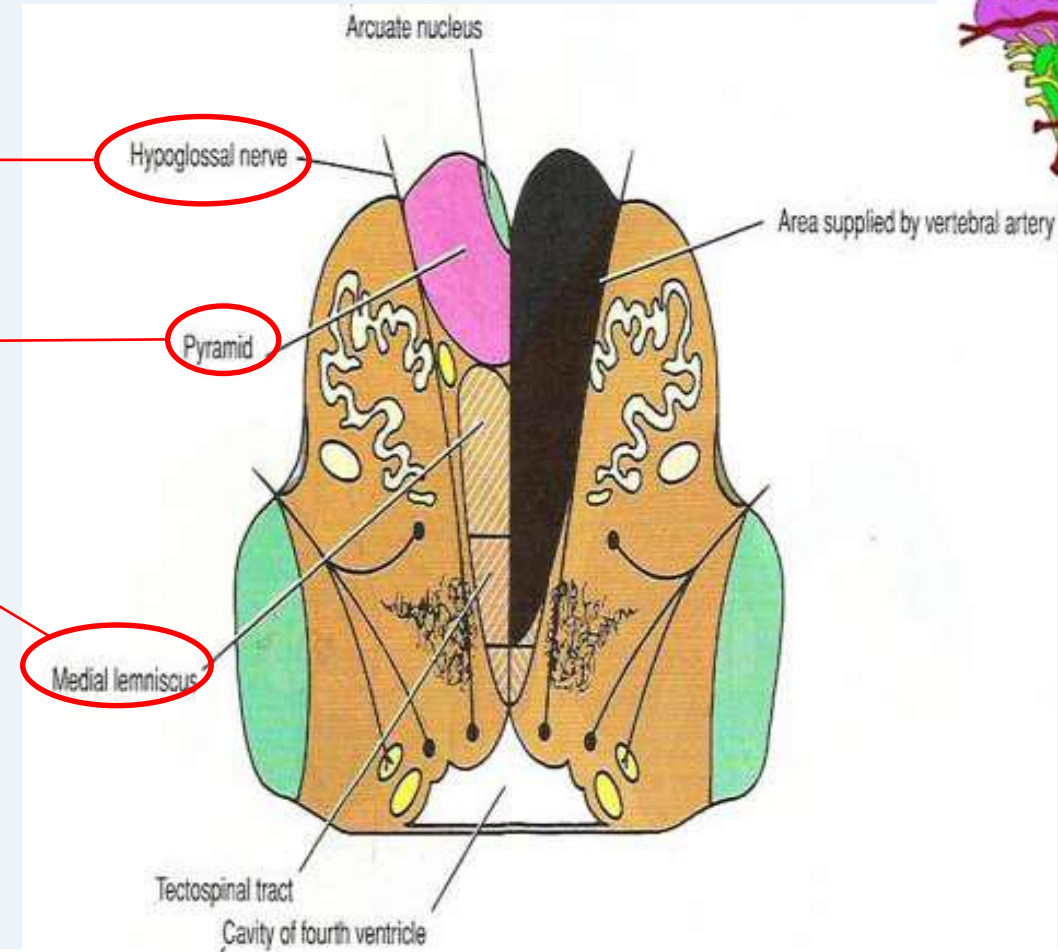
B. CONTRALATERAL

1. Hemiplegia – sparing the face

2. Hemianaesthesia sparing the face.



deviation of the tongue towards the side of the lesion



Cuoco JA, et al. Edorium J Neurol 2016;3:4–16.

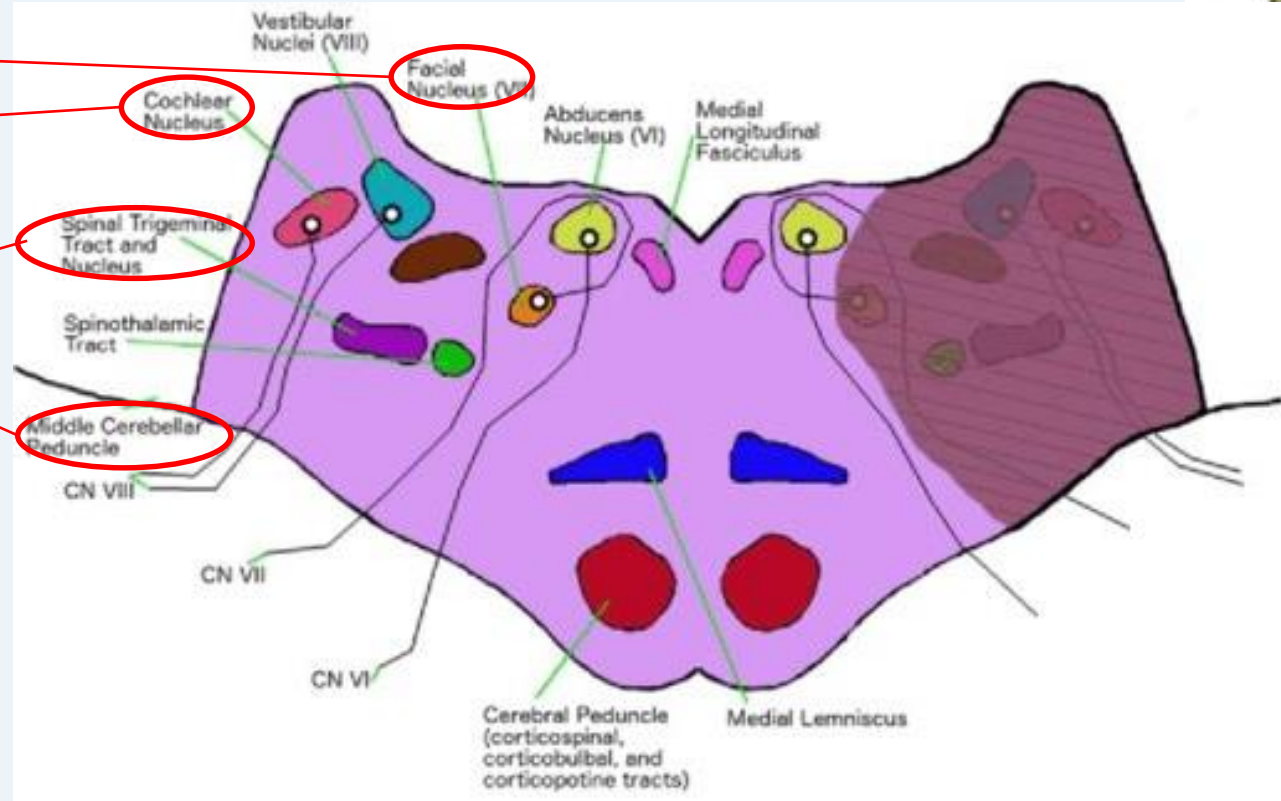
Lateral pontine (Marie-Foix) syndrome-occlusion of anterior inferior cerebellar artery

A. IPSILATERAL

1. LMN facial nerve palsy
2. Horner's syndrome
3. Deafness, tinnitus
4. Cerebellar signs

B. CONTRALATERAL

1. Impairment of pain and temperature on the body, arm and leg



Cuoco JA, et al. *Edorium J Neurol* 2016;3:4–16.



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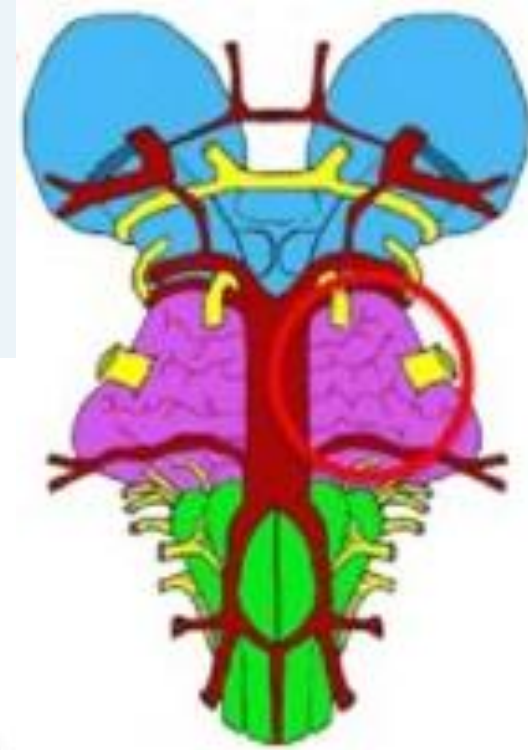
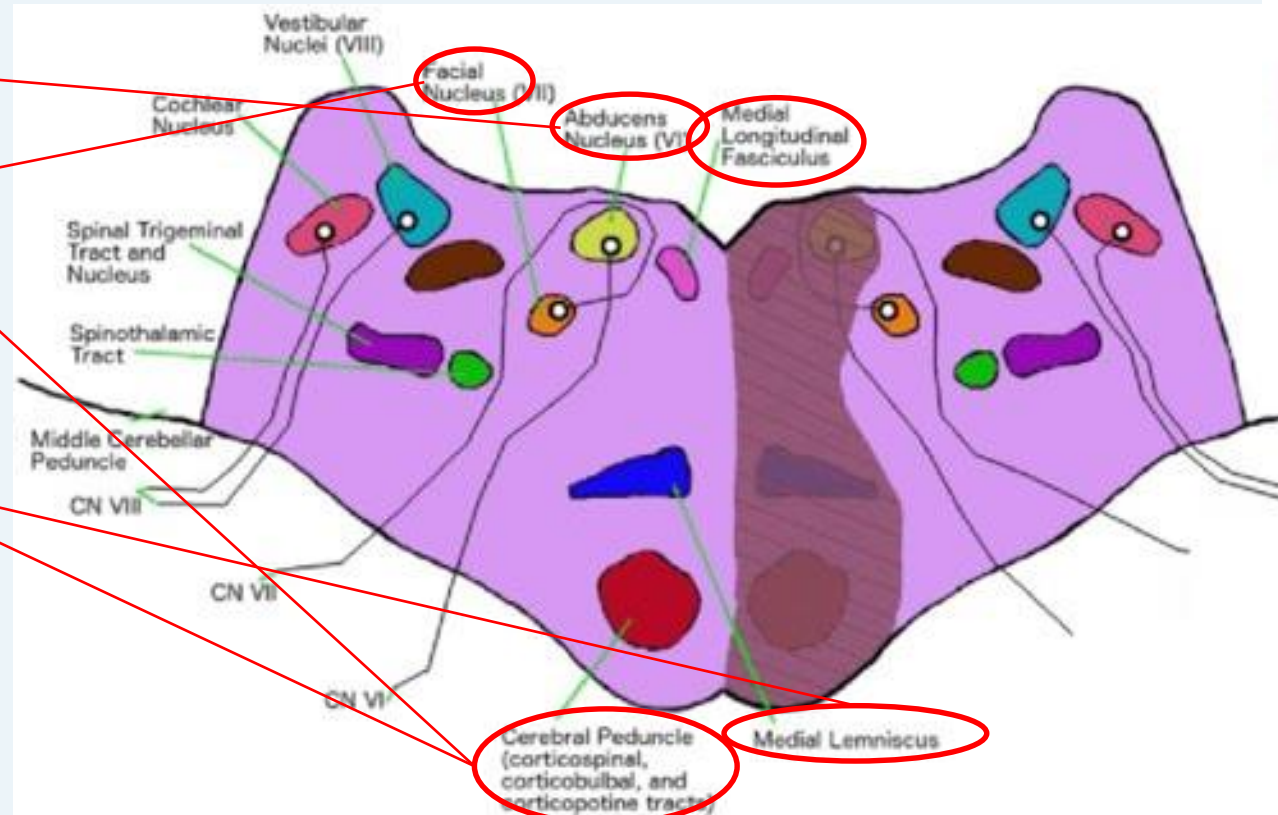
Medial pontine (Foville) syndrome – occlusion of paramedian branch of basilar artery

A. IPSILATERAL

1. Gaze paresis (INO)
2. Cerebellar signs
3. Facial nerve palsy

B. CONTRALATERAL

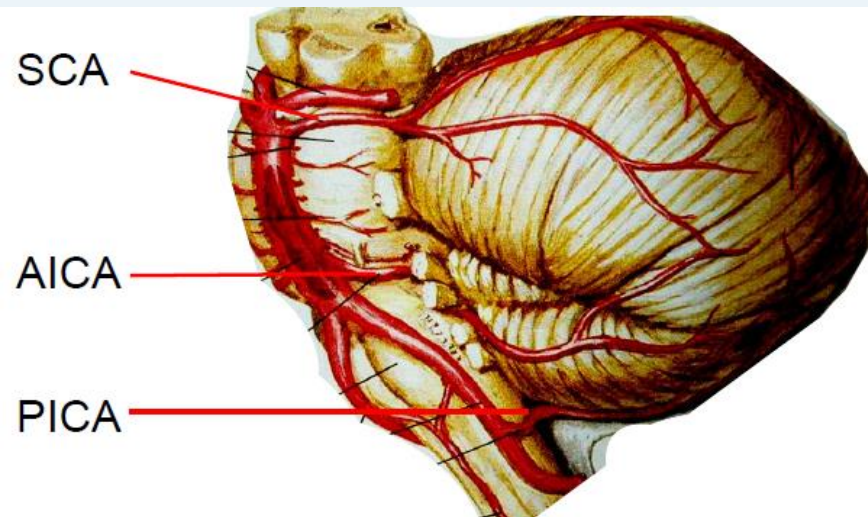
1. Hemiparesis
2. loss of vibration and proprioception arm & leg



Cuoco JA, et al. *Edorium J Neurol* 2016;3:4–16.



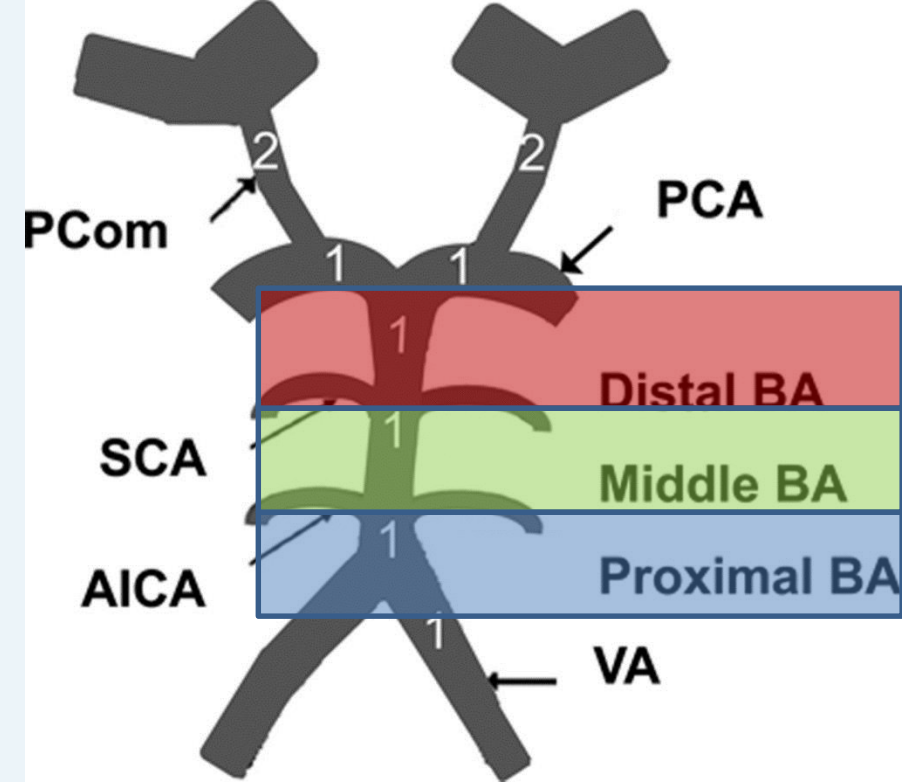
Cerebellar stroke can cause only vertigo!



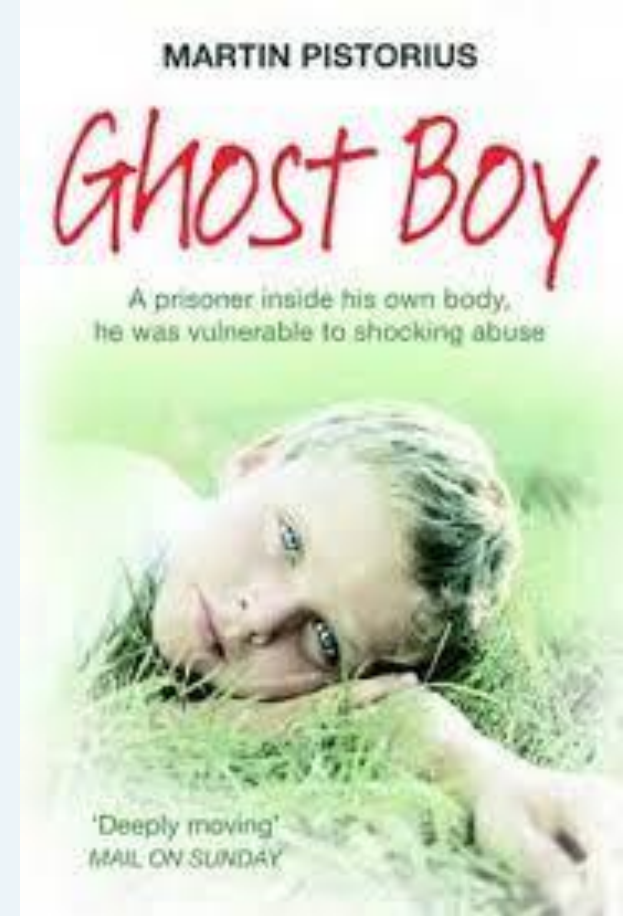
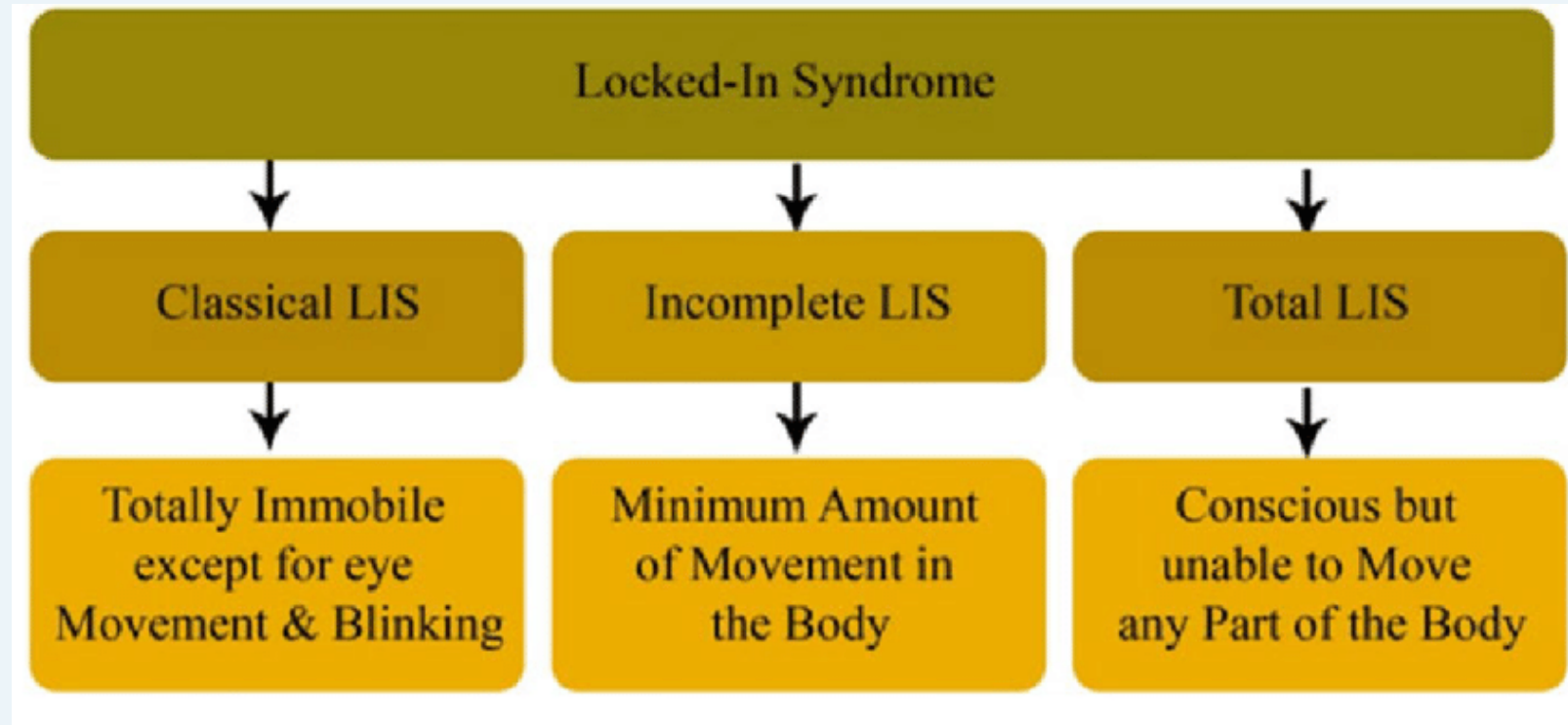
Cerebellar artery	Purely cerebellar territory	Plus adjacent brainstem territory
Superior (SCA)	Dysarthria	IV, contralateral spinothalamic signs Chorea, ipsilateral Horner
Antero-inferior (AICA)	Rotatory vertigo	Hypoacusis/tinnitus VII, V, ipsilateral Horner Contralateral spinothalamic signs
Postero-inferior (PICA)	Rotatory vertigo	Wallenberg's syndrome

Basilar artery occlusion

- **Proximal:** Wallenberg +/- cerebellar +/- medial medullary sdr.
vertebral artery disease
 - **Mid-basilar:** locked-in syndrome
local atherosclerosis or embolic
 - **Distal :** top of basilar syndrome
embolic from proximal arterial source or cardiac
-
- ❖ About **50% of patients have precursory symptoms** or a progressive onset
 - ❖ Overall **prognosis is poor, if left untreated.**



Locked In Syndrome



“For so many years, I was like a ghost. I could hear and see everything, but it was like I wasn’t there. I was invisible,”

“Top of the Basilar” syndrome

- **Symptoms:** hallucinations and behavioral changes, alternating abnormalities of alertness, disorientation, visual, oculomotor deficits and cortical blindness
- **Primary cause:** thromboembolic occlusions of the distal third or tip of the basilar artery
- **Often missed** due to patient confusion and unawareness of visual deficits.
- Coma is a rare initial feature in posterior circulation stroke (2% in one registry study), but **it is important to distinguish coma as a result of basilar thrombosis.**
- **HALLMARK is sudden onset of bilateral signs, including ptosis, pupillary asymmetry or lack of reaction to light, somnolence.**



Posterior cerebral artery stroke

- **Mesencephalo-thalamic territory**

See next slide

- **Occipital territory**

Homonymous visual field deficits

- **Medio-temporal territory**

Left: (transient)

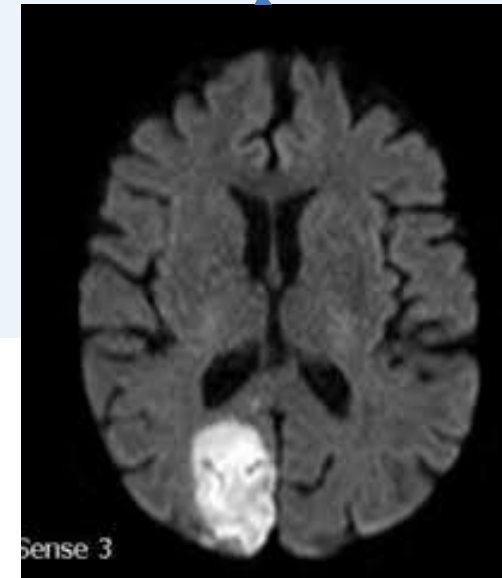
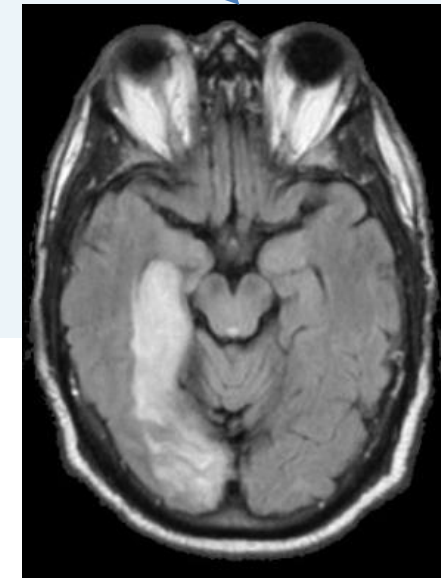
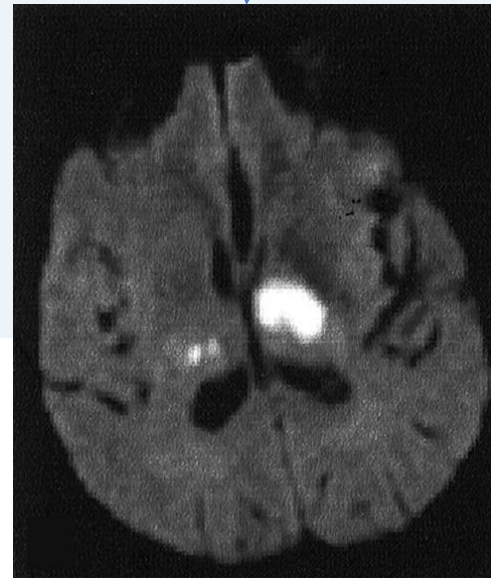
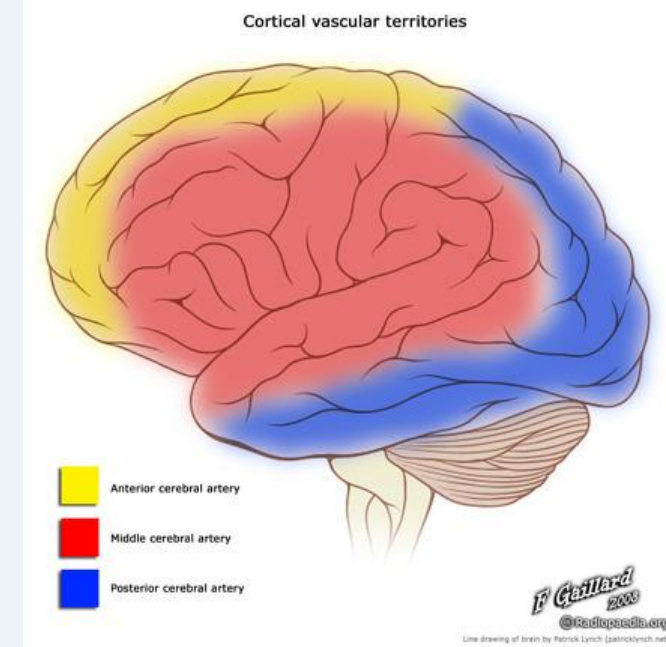
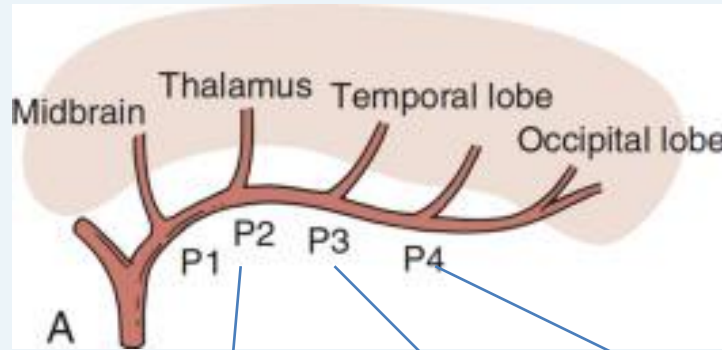
verbal amnesia, alexia, visual agnosia, color anomia

Right: (transient)

visual amnesia, color agnosia

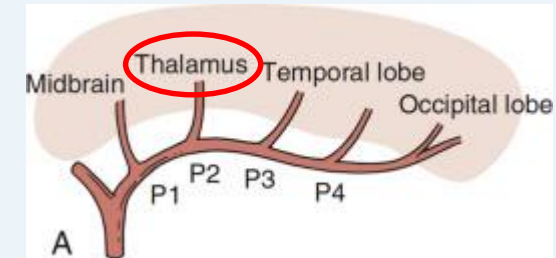
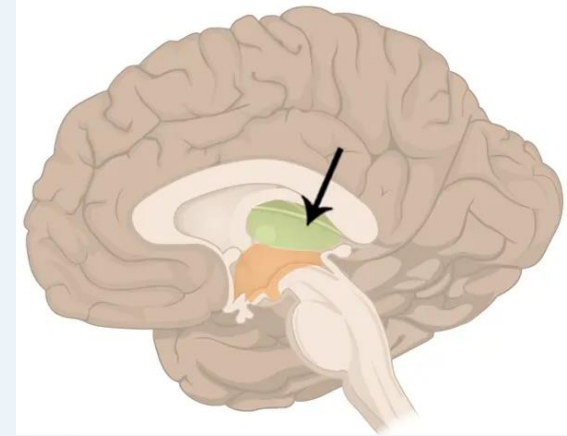
Bilateral (or right acute):

prosopagnosia



Thalamic stroke: a chameleon!

Artery	Thalamic territory	Signs
Thalamo-geniculate artery	→ Lateral	Sensory hemisindrome Hemiataxia, pain Hemidystonia
Polar artery	→ Anterior	Abulia, confusion L: aphasia, verbal amnesia R: visual amnesia
Thalamic-subthalamic art. → paramedian art.	→ Paramedian	↓Vigilance, amnesia, vertical gaze paresis Dystonia, asterixis
Posterior choroidal artery	→ Pulvinar, lateral corpus geniculatum	Visual field defects (Amnesia, aphasia, dystonia, hemisindrome)

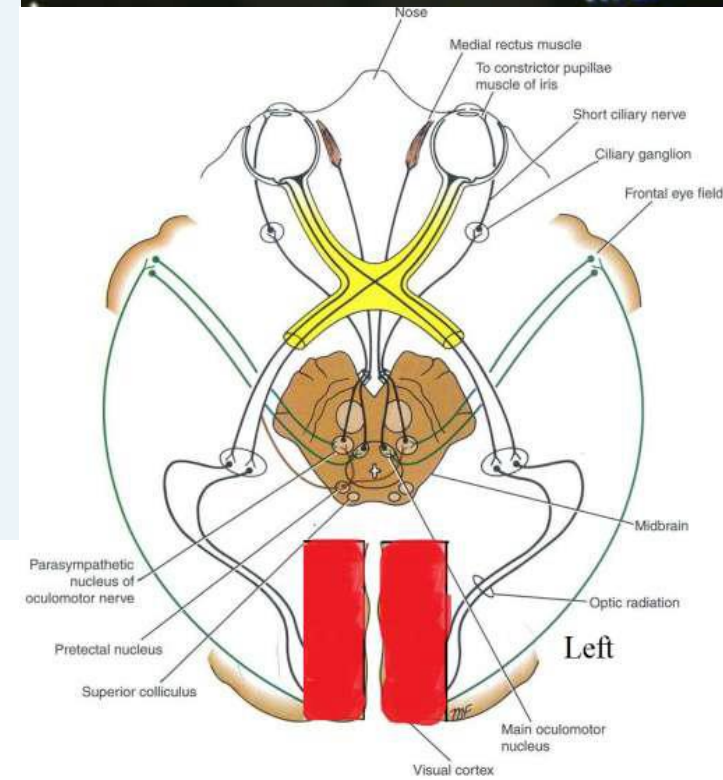
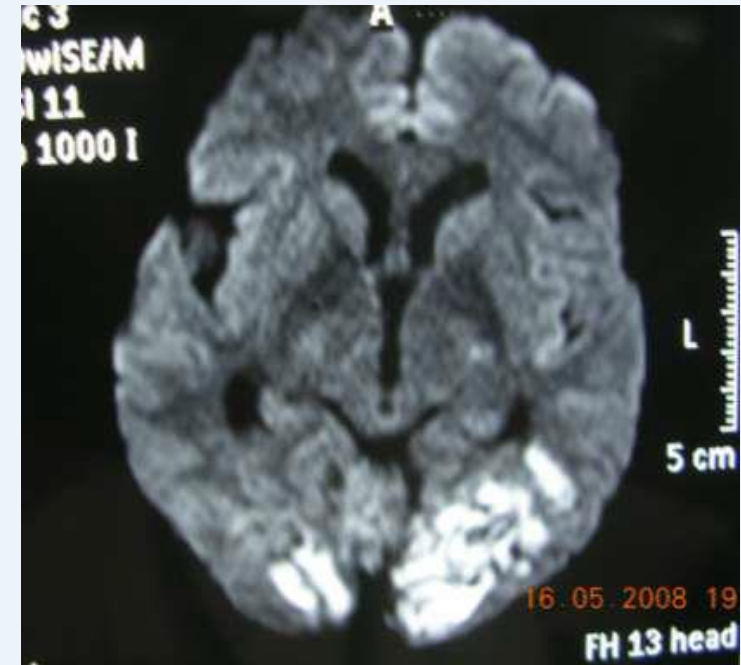


Occipital lobe-occlusion of both calcarine arteries

- ✓ Bilateral hemianopic cortical blindness (light reflex preserved)
- ✓ Symptoms: cortical blindness but unaware, confabulation
- ✓ Anton-Babinski Syndrome



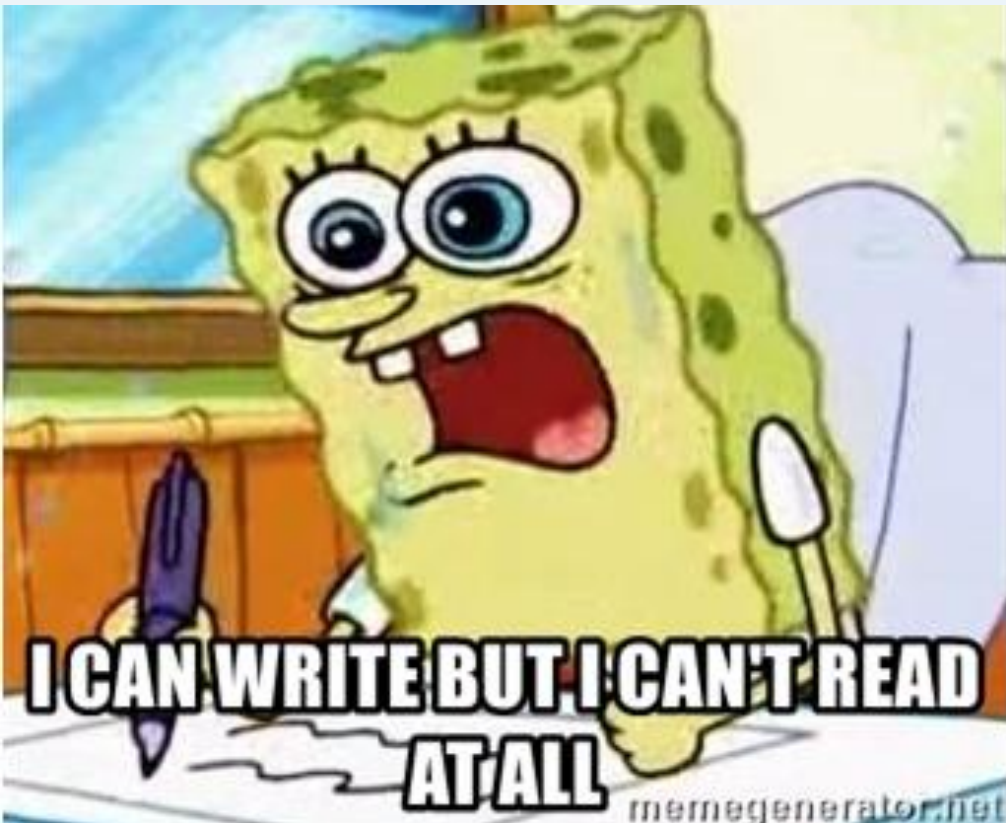
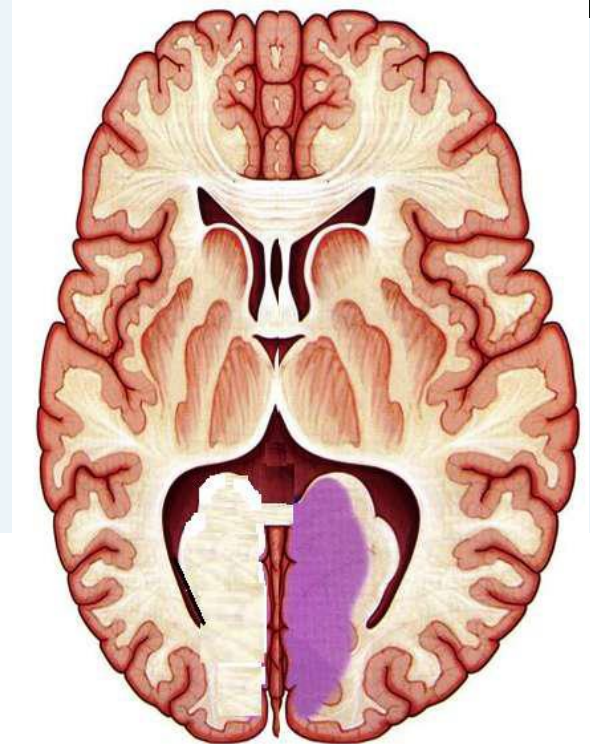
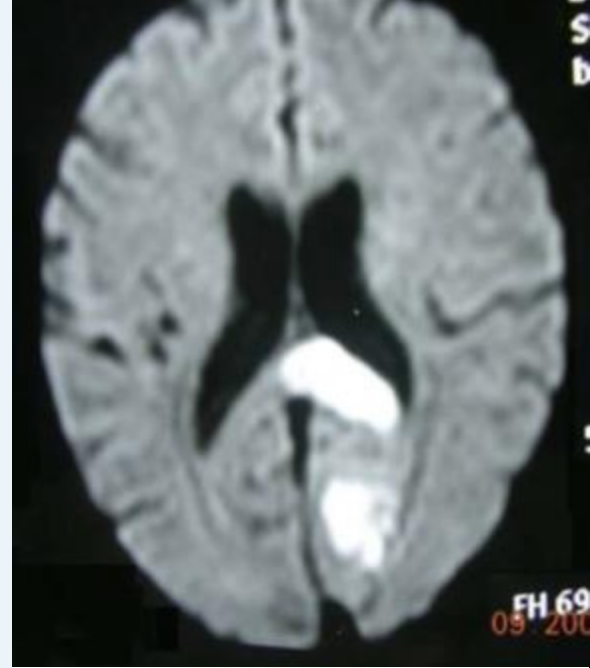
"Blind to Their Blindness"



Left occipital lobe with corpus callosum infarction

Clinical features

1. Right hemianopia
2. Alexia without agraphia



Differential diagnosis – Stroke chameleons

- Bilateral thalamic ischaemia -> **reduced consciousness level or a global amnesic syndrome**
- Bilateral occipital stroke -> present as **confusion or delirium**
- Infarcts limited to the medial vermis in medial PICA territory -> **vertiginous syndrome** that resembles peripheral vestibulopathy
- Rarely acute brainstem ischaemia can cause **bilateral rhythmic leg movements** that can resemble fasciculations or convulsive seizures



Differential diagnosis - Stroke mimics

Box 1: Differential diagnosis of acute vestibular syndrome based on expert opinion

Less urgent causes

Common

- Vestibular neuritis or labyrinthitis
- Multiple sclerosis

Uncommon (< 1%) or unknown frequency

- CNS adverse effects (e.g., antiepileptics)
- Medication ototoxicity (e.g., post-aminoglycoside)
- Other CNS inflammation (e.g., sarcoidosis)
- Prolonged attack of episodic ataxia syndrome
- Prolonged attack of Menière disease
- Prolonged attack of vestibular migraine
- Traumatic vestibulopathy (including surgical)

*Presumed possible**

- Atypical infection (otosyphilis, Lyme disease)
- Celiac disease
- Cerebello-pontine angle neoplasm
- Degenerative cerebellar ataxia
- Drug intoxication (e.g., alcohol, illicit drugs)

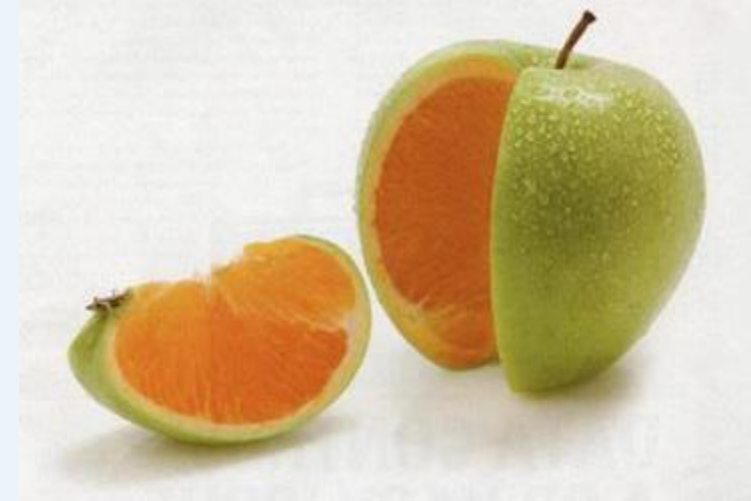
More urgent causes

Common

- Brainstem or cerebellar infarction
 - Brainstem or cerebellar hemorrhage
- #### *Uncommon (< 1%) or unknown frequency*
- Bacterial labyrinthitis/mastoiditis
 - Brainstem encephalitis (e.g., listeria, paraneoplastic)
 - Brainstem hypertensive encephalopathy
 - Herpes zoster oticus (Ramsay Hunt syndrome)†
 - Labyrinthine stroke‡
 - Wernicke syndrome (vitamin B₁ deficiency)
 - Miller Fisher syndrome

*Presumed possible**

- Altitude sickness or hypoxia
- Basilar meningitis (e.g., tuberculosis)
- Cerebral infarction or hemorrhage§
- CNS medication toxicity (e.g., lithium)
- Decompression sickness
- Electrolyte imbalance (e.g., hyponatremia)
- Endocrine disorders (e.g., acute adrenal insufficiency)
- Environmental toxins (e.g., carbon monoxide)
- Subarachnoid hemorrhage/aneurysm



Tarnutzer AA, et al. CMAJ. 2011;183:E571-92

Presentation outline

- Challenges in the diagnosis of posterior circulation stroke
- Anatomy of posterior circulation & mechanisms of posterior circulation ischemia
- Common posterior circulation stroke syndromes
- **Diagnosis of posterior circulation stroke/TIA in the emergency setting**
- Conclusions

➤ There are important differences between posterior and anterior circulation stroke.

Aspects	Anterior circulation (carotid territory)	Posterior circulation (vertebrobasilar territory)
Clinical recognition tools		
Prehospital triage tools and scores, such as FAST*	High sensitivity: >90%	Moderate sensitivity: ~60%
Imaging		
Computed tomography	Moderate sensitivity	Poor sensitivity
Magnetic resonance imaging	Very good to excellent sensitivity (>95%)	Very good sensitivity (>80%)
Clinical features†		
Isolated hemianopia	+	++
Quadrantanopia	–	+
Pupil abnormalities	+ (Horner's syndrome)	+++ (may be bilateral)
Diplopia	–	+++
Focal (unilateral) sensorimotor	+++	++
Bilateral sensorimotor	–	+++
Unsteadiness/ataxia	+	++
Vertigo	±	+++
Dysarthria	++	++
Dysphasia	+++	+ (thalamic infarcts)
Coma	+	+++

Coma unusual, unless there is mass effect and raised intracranial pressure (for example, as a result of large middle cerebral artery stroke); rare as an initial hyperacute presenting symptom; somnolence may occur

Coma well recognised in thalamic and brainstem ischaemia and may be an acute presenting symptom



The 4 pillars for the diagnosis of suspected posterior circulation strokes

Tips for non-specialists

Careful history taking is needed to identify patients with posterior circulation stroke, who may present with recurrent, stuttering, or progressive symptoms, which may include altered level of awareness (not a typical stroke symptom but seen in bilateral thalamic ischaemia)

Clinical signs that may help identify a posterior circulation stroke include the presence of homonymous visual field deficits, eye movement abnormalities, Horner's syndrome, or gait ataxia

Previously ambulant patients with acute focal neurological symptoms leading to acute loss of balance should never be discharged without ensuring they can walk if stroke is a possible explanation. Always consider a posterior circulation stroke if a patient is uncharacteristically disabled for the amount of alcohol reportedly consumed

Investigate posterior circulation transient ischaemic attack symptoms urgently to avoid preventable disability or death. Use rapid access transient ischaemic attack services or stroke specialist assessment if available, and use magnetic resonance imaging in the acute phase, especially if the diagnosis is unclear, because this modality has high sensitivity for identifying ischaemic lesions

Consider transferring patients at risk of deterioration in the acute phase of posterior circulation ischaemic stroke to a neuroscience centre because they may need urgent neurosurgery for mass effect or hydrocephalus

Merwick Á, Werring D. BMJ. 2014;348:g3175.



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- Face Arm Speech Time (FAST) did not detect 38% of posterior cerebral circulation strokes. Harbison et al recommended that **prehospital stroke assessment tools assess visual disturbance to improve posterior circulation stroke recognition.**
- Diagnostic tools such as the recognition of stroke in the emergency room (ROSIER) scale may help medical staff in the emergency department rapidly recognize acute stroke because this tool includes assessment of visual fields.

Has there been loss of consciousness or syncope?		
	Y (-1) <input type="checkbox"/>	N (0) <input type="checkbox"/>
Has there been seizure activity?		
	Y (-1) <input type="checkbox"/>	N (0) <input type="checkbox"/>
Is there a <u>NEW ACUTE</u> onset (or on awakening from sleep)?		
I.	Asymmetric facial weakness	Y (+1) <input type="checkbox"/> N (0) <input type="checkbox"/>
II.	Asymmetric arm weakness	Y (+1) <input type="checkbox"/> N (0) <input type="checkbox"/>
III.	Asymmetric leg weakness	Y (+1) <input type="checkbox"/> N (0) <input type="checkbox"/>
IV.	Speech disturbance	Y (+1) <input type="checkbox"/> N (0) <input type="checkbox"/>
V.	Visual field defect	Y (+1) <input type="checkbox"/> N (0) <input type="checkbox"/>

Harbison J, et al. Stroke. 2003;34:71–76.
Nor AM, et al. Stroke. 2004;35:1355–1359.

Visual manifestations of posterior circulation strokes

- ✓ **Vertical diplopia** and gaze paresis
- ✓ **Horizontal diplopia**
- ✓ Conjugate horizontal eye deviation / **gaze paresis** (« Wrong way »)
- ✓ **Ptosis +/- miosis**
- ✓ **Homonymous visual field deficits** (if present with sensory deficits = posterior cerebral artery stroke)



An augmented FAST for the diagnosis of posterior circulation stroke?

- Assessment in the emergency department for homonymous **visual field deficits**; **eye movement abnormalities** and looking for **Horner's syndrome** (ptosis, small pupil (miosis), and anhydrosis on the same side), bilateral small or fixed pupils, and **ataxia** may aid early diagnosis.
- Incorporation of ataxia and visual symptoms to FAST screening [**FAST ataxia and visual disturbance (AV)** or **FAST-ataxia and blindness (AB)**] may increase its sensitivity for detection of acute posterior circulation strokes.



An augmented ABCD2 score for the diagnosis of posterior circulation TIAs?

- The **ABCD3-I** score is an externally validated risk prediction tool, which incorporates abnormalities on diffusion weighted imaging MRI, multiple preceding TIAs, carotid stenosis, and the components of the ABCD2 score into a risk prediction score.
- **Substituting vertebrobasilar stenosis in posterior circulation cases for the 2 points scored for carotid stenosis in the ABCD3-I score, or incorporating the clinical features vertigo, visual symptoms, or ataxia into a risk prediction score,** may refine the prediction of stroke after posterior circulation TIA.

Merwick A, et al. Lancet Neurol 2010;9:1060-9.

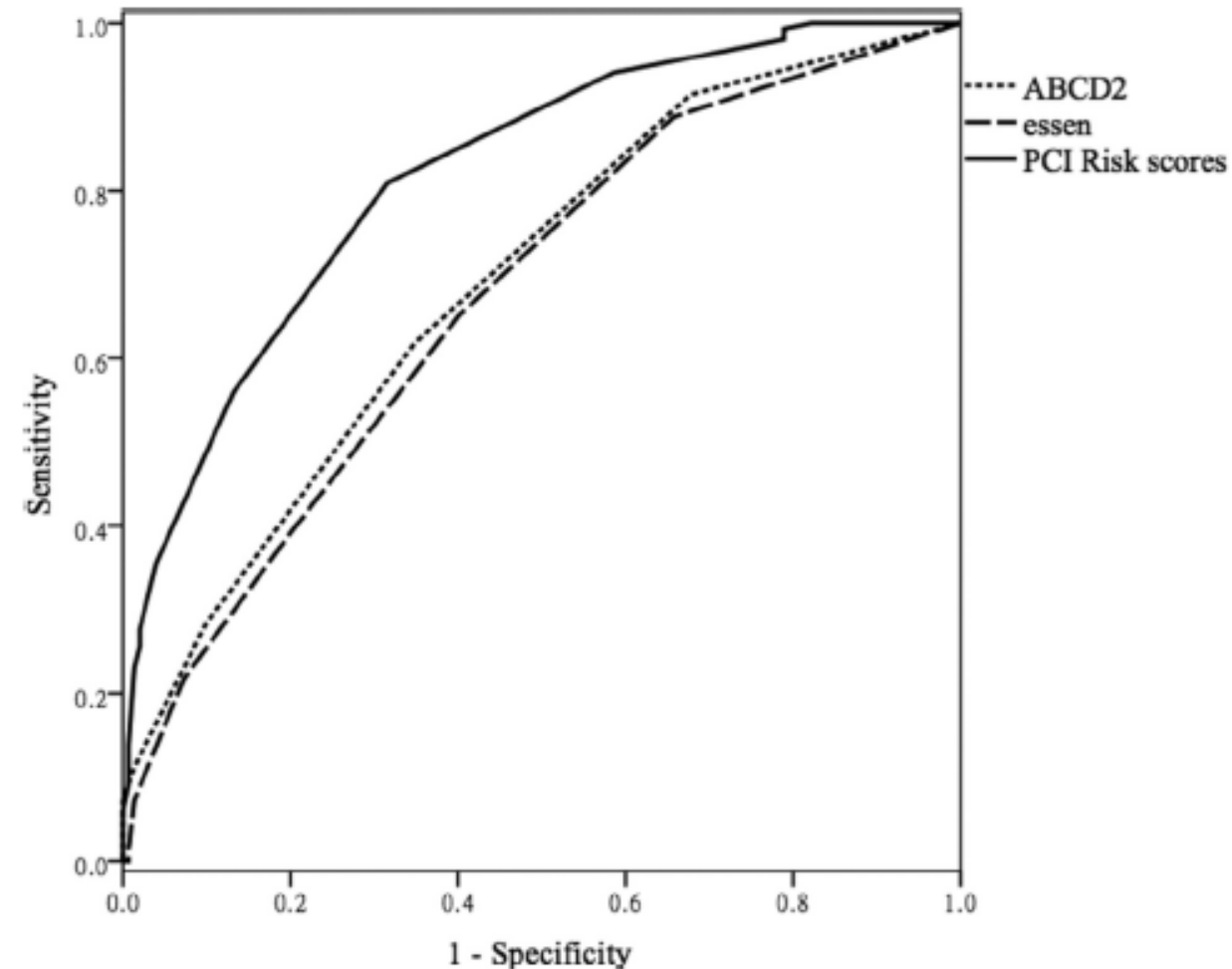
Factor	ABCD ₂	ABCD3-I
Age ≥60 years old	1	1
BP ≥140/90 mmHg ^a	1	1
Clinical features		
Unilateral weakness	2	2
Speech impairment without weakness	1	1
Duration of TIA		
≥60 min	2	2
10–59 min	1	1
Diabetes	1	1
Dual TIA (TIA prompting medical attention plus at least another TIA in the preceding 7 days)	NA	2
Imaging: ipsilateral ≥50 % stenosis of internal carotid artery	NA	2
Imaging: acute diffusion-weighted imaging hyperintensity	NA	2

The PCI risk score

Table 3. *PCI risk scores*

Risk factors	Scores
High blood pressure	1
Diabetes mellitus	1
Ischemic stroke	1
Rotating and rocking	-1
Difficulty in speech	5
Tinnitus	-5
Limb sensory deficit	5
Gait ataxia	1
Limb ataxia	5

PCI, posterior circulation ischemia.

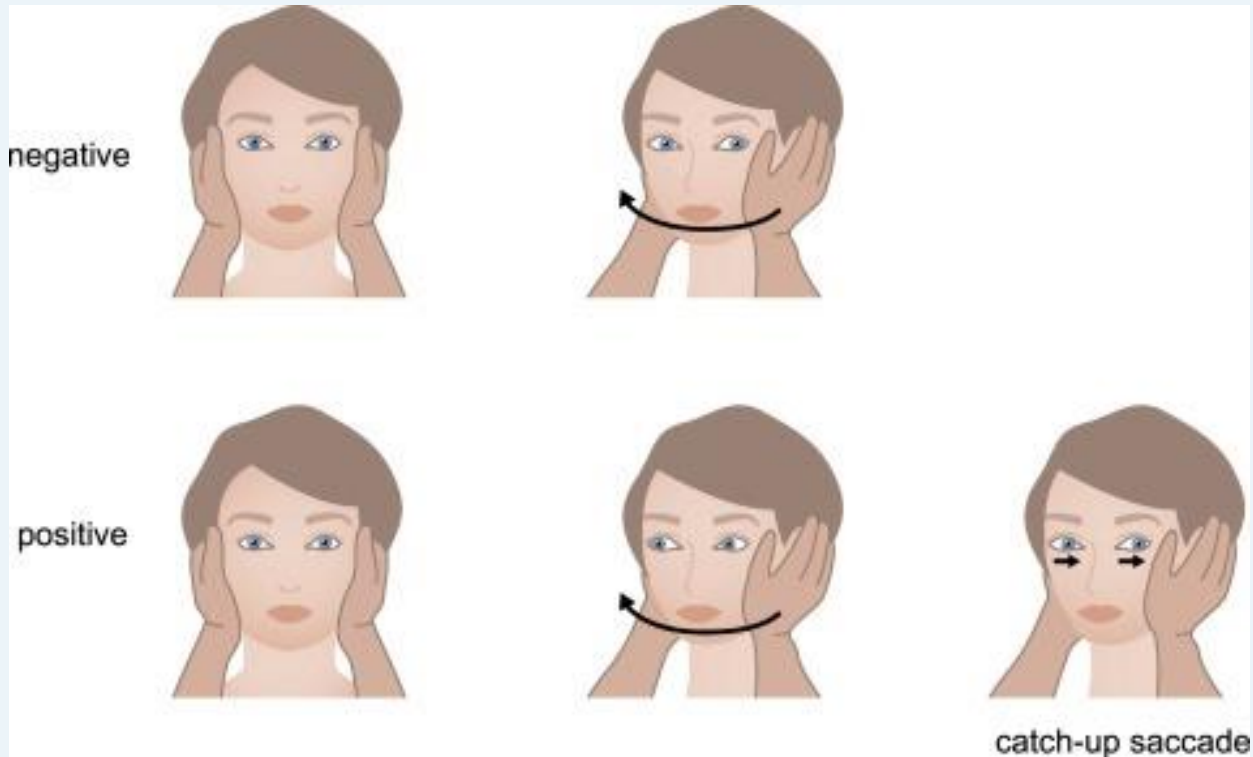


- When **0** was selected as a cutoff point for differentiating the patients with PCI from patients without PCI, the **sensitivity was 94.1%, with a specificity of 41.4%.**

Chen R, et al. J Stroke Cerebrovasc Dis. 2018;27:506-512.

HINTS

- The **h**ead **i**mpulse test is a simple clinical test comprising high acceleration head rotation.
- **Positive test -> peripheral cause:** In the presence of a severe unilateral vestibular weakness the normal vestibulo-ocular reflex is replaced by a misalignment of the eye followed by a series of corrective saccades



<https://www.youtube.com/watch?v=Wh2ojfgbC3I>



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HINTS

- Nystagmus is an involuntary rhythmic side-to-side, up and down or circular motion of the eyes

	Peripheral	Central
Onset	Sudden	Gradual
Intensity	Severe	Mild
Duration	Seconds	Continuous
Nystagmus	Fatigable	Non-fatigable
Direction of nystagmus	Unidirectional	Pure vertical, multidirectional (possibly unidirectional), may change with direction of gaze
Associated neurologic findings	None	Usually present
Hearing loss or tinnitus	May be present	None
Associated nausea or vomiting	Frequent, severe	Infrequent, mild



Bidirectional Nystagmus
(Worrisome)

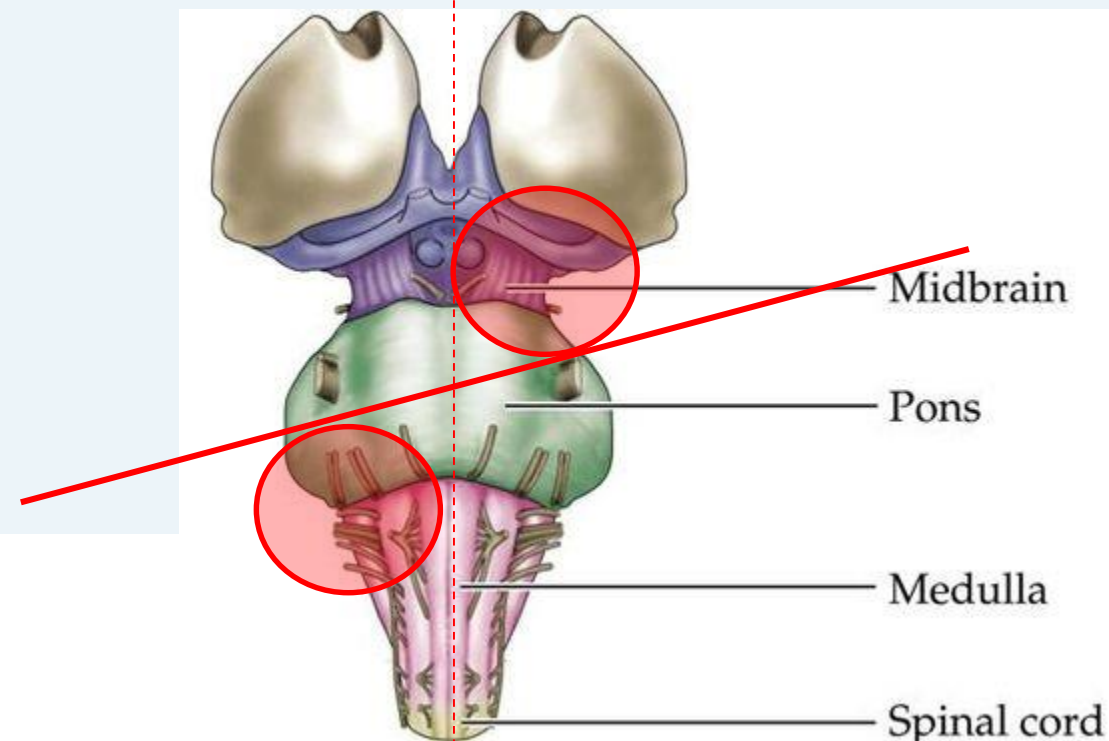
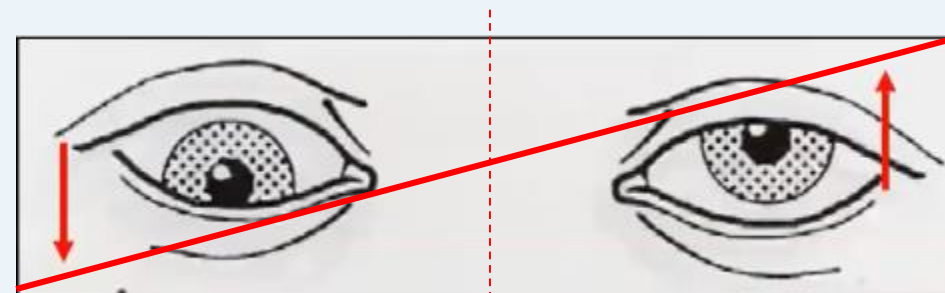
<https://gfycat.com/>

HINTS

- Test of **s**kew deviation is characterized as an acquired vertical misalignment of the eyes that is not due to any single muscle or ocular motor nerve



Abnormal Vertical Skew
(Worrisome)



	Peripheral Vertigo	Central Vertigo
Head Impulse Test	Abnormal; corrective saccade to midline with rotation of head	Normal; no corrective saccade
Nystagmus	Unidirectional; horizontal	Horizontal & direction-changing; vertical; torsional
Test of Skew	No skew deviation	Skew deviation present

HINTS to Diagnose Stroke in the Acute Vestibular Syndrome

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

Table 1. Key Clinical Features in Patients With Peripheral Versus Central AVS

Symptoms, Signs, and Imaging at Presentation	PAVS (n=25)	CAVS (n=76)	NLR Central (95% CI)
Associated symptoms	12%	41%	0.67 (0.53–0.85)*
Acute auditory symptoms	0%†	3%	0.97 (0.94–1.01)
Headache or neck pain	12%	38%	0.70 (0.56–0.88)*
General neurological signs (including truncal ataxia)	0%	51%	0.49 (0.39–0.61)*
Facial palsy	0%	1%	0.99 (0.96–1.01)
Hemisensory loss	0%	3%	0.97 (0.94–1.01)
Crossed sensory loss	0%	3%	0.97 (0.94–1.01)
Dysphagia/dysarthria	0%	3%	0.97 (0.94–1.01)
Limb ataxia	0%	5%	0.95 (0.90–1.00)
Mental status abnormality (lethargy)	0%	7%	0.93 (0.88–0.99)
Hemiparesis (including UMN facial weakness)	0%	11%	0.89 (0.83–0.97)
Severe truncal instability (cannot sit unassisted)	0%	34%	0.66 (0.56–0.77)*
Obvious oculomotor signs	0%	32%	0.68 (0.59–0.80)*
Dominantly vertical or torsional nystagmus	0%	12%	0.88 (0.81–0.96)
Oculomotor paralysis (3-4-6, INO, gaze palsy)	0%	21%	0.79 (0.70–0.89)*
Subtle oculomotor signs	4%	100%	0.00 (0.00–0.11)*
Direction-changing horizontal nystagmus	0%	20%	0.80 (0.72–0.90)*
Skew deviation present or untestable	4%‡	25%§	0.78 (0.67–0.91)*
h-HIT normal or untestable	0%	93%	0.07 (0.03–0.15)*

Kattah JC, et al. *Stroke*. 2009;40:3504-3510.

Table 4. Bedside Signs and Initial MRI With DWI Test Properties for Ischemic Stroke in AVS

	Sensitivity (n=69)	Specificity (n=25)	NLR Stroke (95% CI)
General neurological signs*	19%	100%	0.81 (0.72–0.91)
Obvious oculomotor signs	28%	100%	0.72 (0.63–0.84)
Severe truncal ataxia	33%	100%	0.67 (0.56–0.79)
Any obvious signs	64%†	100%	0.36 (0.27–0.50)
Initial MRI with DWI	88%‡	100%	0.12 (0.06–0.22)
Dangerous bedside HINTS	100%	96%	0.00 (0.00–0.12)

*Excluding severe truncal ataxia (this Table only).

†Of 25 ischemic strokes without obvious signs, 12 were pure cerebellar, 7 were lateral medullary, 5 were lateral pontine or middle peduncle, and one was a medial brainstem infarct.

‡False-negative initial MRI with DWI occurred in 5 patients with lateral medullary infarctions, one with lateral pontomedullary infarction, and 2 with middle cerebellar peduncle infarction.

- ✓ normal/untestable h-HIT **or**
- ✓ direction changing horizontal nystagmus present/untestable **or**
- ✓ skew deviation present/untestable

Caveat!

For patients consenting to screening, the study **neuroophthalmologist** (J.C.K.) conducted a neurological and vestibular examination (including h-HIT, prism cross-cover test for ocular alignment, and observation of nystagmus in different gaze positions)

Kattah JC, et al. Stroke. 2009;40:3504-3510.

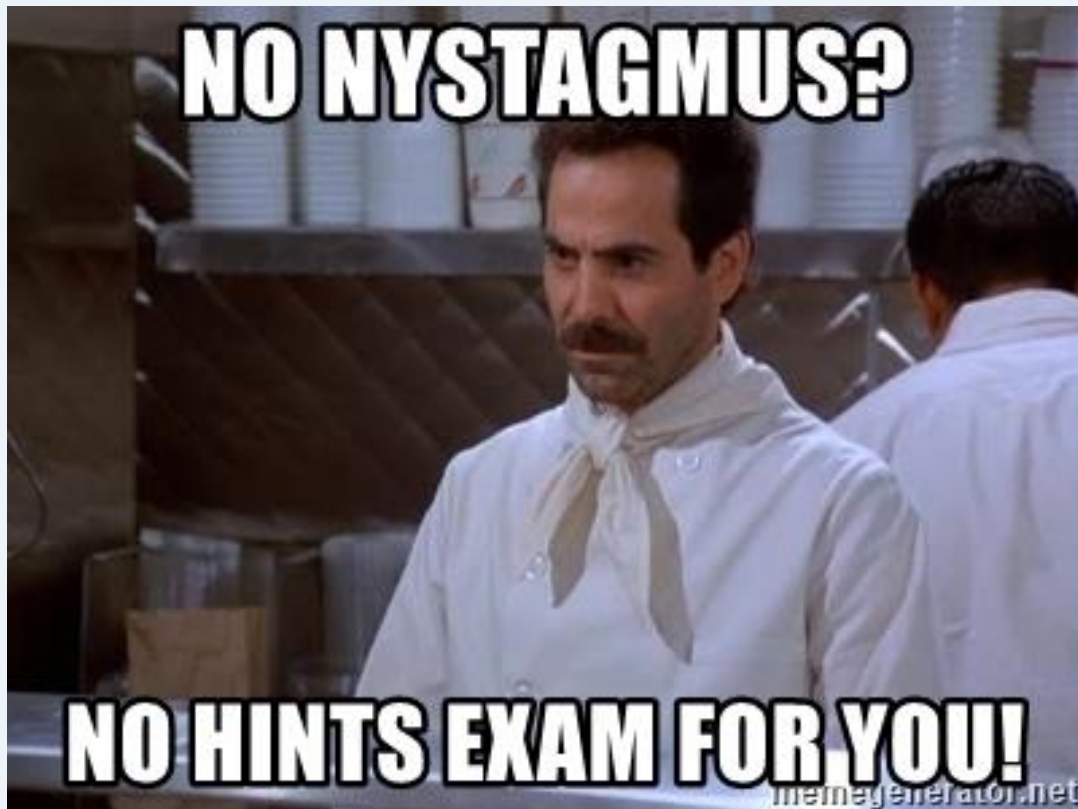
HINTS in the real world?

- In a systematic review and meta-analysis a **15-fold increased risk of posterior circulation stroke was found in patients with positive HINTS test** compared to those with no abnormality [1].
- For any stroke, the **pooled sensitivity was 95.5%** (95% CI: 92.6–98.4%) and **specificity was 71.2%** (95% CI: 67.0–75.4%) [1].
- In a single center cross-sectional study of high-risk patients (more than one stroke risk factor) with acute vestibular syndrome **HINTS was found to substantially outperform ABCD2 score for stroke diagnosis in ED patients with acute vestibular syndromes** (ABCD2 ≥ 4 for stroke had sensitivity of 61.1%, specificity of 62.3%) [2].
- **It also outperforms MRI obtained within the first 2 days after symptom onset** [2].

1. Krishnan K, et al. Eur Stroke J. 2019;4:233-239.

2. Newman-Toker DE, et al. Acad Emerg Med. 2013;20:986-96.

HINTS exam only if nystagmus is present



Dix-Hallpike Maneuver

Tests for **canalithiasis** of the **posterior semicircular canal**, which is the **most common cause of benign paroxysmal positional vertigo (BPPV)**



- 1 With the patient sitting up, turn the head 45 degrees to one side
- 2 Lie the patient down with head overhanging the edge of the bed and look for nystagmus
- 3 Repeat on the contralateral side

Positive if the maneuver provokes paroxysmal vertigo and nystagmus



❑ Sensitivity: 79% (95%CI: 65-94)

❑ Specificity: 75% (95%CI: 33-100)



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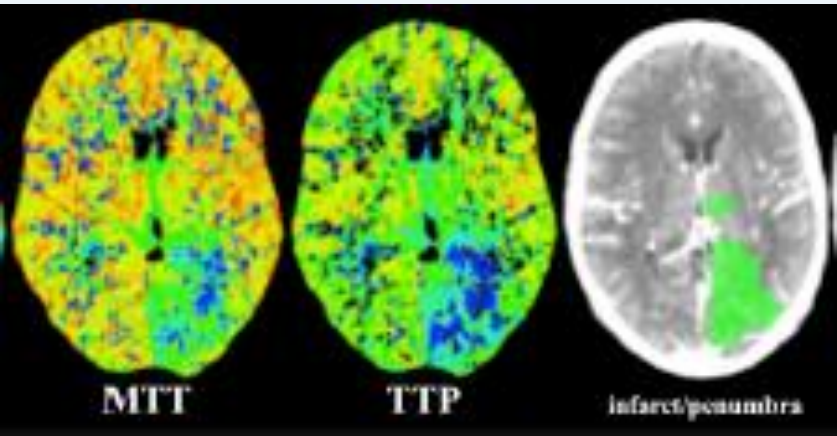
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Imaging for suspected acute stroke in posterior circulation

- **MRI with diffusion weighted imaging** is the brain imaging modality of choice for suspected posterior circulation stroke. It can help diagnose disorders that mimic stroke and TIA, can help verify vascular territory, and diffusion weighted imaging abnormalities independently predict early stroke risk after TIA.
- MRI is far more sensitive than CT in the diagnosis of acute ischaemic stroke for all vascular territories, with study results indicating 80-95% sensitivity in the first 24 hours when diffusion weighted imaging is used, versus 16% sensitivity with CT.
- Sensitivity may be lower in the posterior circulation and **false negatives can occur with early MRI**—a 19% false negative rate was reported in one single centre case series of 31 patients with
- vertebrobasilar stroke.
- MRI or magnetic resonance angiography with dedicated **fat saturated sequences** may help identify **vertebral dissection**.

CT perfusion for posterior circulation strokes?

- All cases of suspected stroke require urgent brain imaging with CT or magnetic resonance imaging (MRI) to exclude haemorrhage. If a patient is a candidate for thrombolysis therapy, **brain and vessel imaging with a technique such as CT angiography is essential to identify basilar artery occlusion.**
- **CT perfusion has been reported to have significant additional diagnostic values** to noncontrast CT and CT angiography source images for detecting ischemic changes in patients suspected of acute posterior circulation stroke.



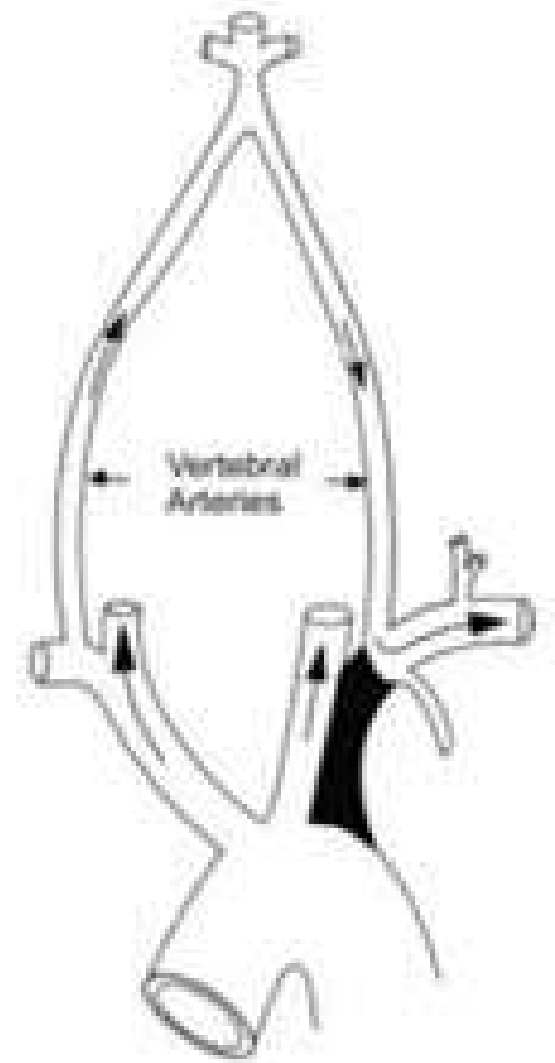
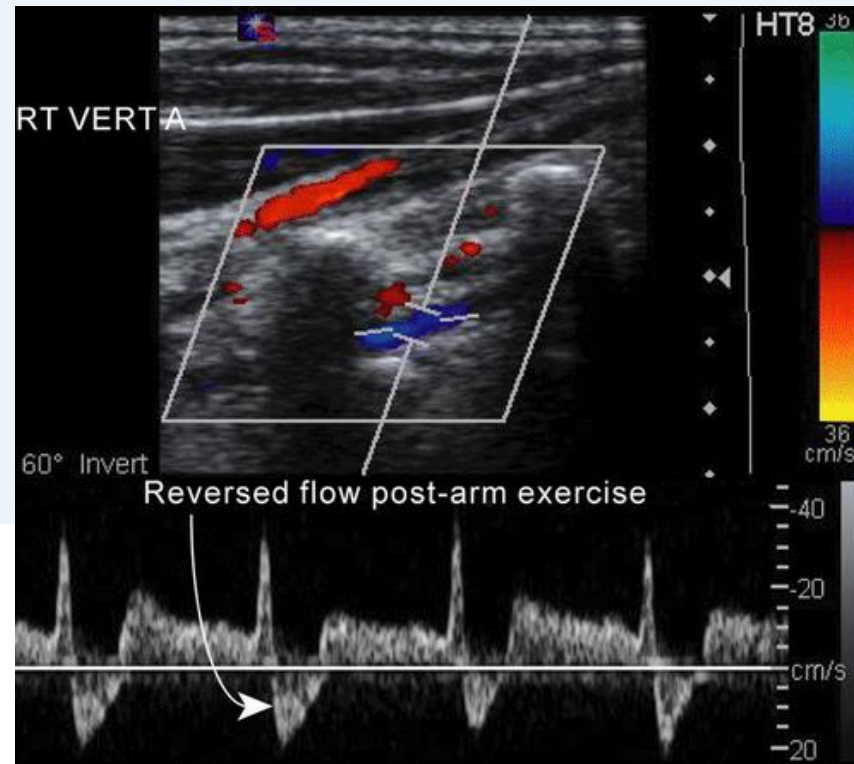
Erik JRJ, et al. Stroke. 2015;46:1113-1115.

	Infarct on Follow-Up					
	Yes	No	PPV	NPV	Sensitivity	Specificity
Any patient						
NCCT	13	1	93 (66–100)	61 (49–72)	31 (18–47)	98 (88–100)
CTA-SI	14	1	93 (68–100)	62 (50–73)	33 (20–50)	98 (88–100)
CTP	31	3	91 (76–98)	80 (66–89)	74 (58–86)	93 (82–99)
Total	42	46
Cerebellum						
NCCT	8	1	89 (52–100)	89 (83–93)	30 (14–50)	99 (96–100)
CTA-SI	11	1	92 (62–100)	90 (85–94)	41 (22–61)	99 (96–100)
CTP	23	6	79 (60–92)	97 (93–99)	85 (66–96)	96 (91–99)
Total	27	149
Thalamus						
NCCT	1	1	50 (1–99)	94 (89–97)	8 (0–38)	99 (97–100)
CTA-SI	0	0	NA	93 (88–96)	0 (0–26)	100 (98–100)
CTP	6	4	60 (26–88)	96 (92–99)	50 (21–79)	98 (94–99)
Total	12	164
PCA territory						
NCCT	5	1	83 (36–100)	95 (91–98)	38 (14–68)	99 (97–100)
CTA-SI	5	0	100 (48–100)	95 (91–98)	38 (14–68)	100 (98–100)
CTP	11	5	69 (41–89)	99 (96–100)	85 (55–98)	97 (93–99)
Total	13	163
Pons/midbrain						
NCCT	1	4	20 (0–72)	95 (90–98)	10 (0–45)	98 (94–99)
CTA-SI	1	4	20 (0–72)	95 (90–98)	10 (0–45)	98 (94–99)
CTP	1	4	20 (0–72)	95 (90–98)	10 (0–45)	98 (94–99)
Total	10	166

Ultrasound for patients with events suggestive of posterior cerebral ischemia?

Subclavian steal syndrome

- Subclavian artery occluded or severely stenosed proximal to origin of vertebral.
- Leads to reversal in the direction of blood flow in the ipsilateral vertebral artery.
- Exercise of ipsilateral arm may increase demand on vertebral flow, leading to posterior circulation TIAs.
- Carotid US is useful in cases to reveal flow reversal post exercise.



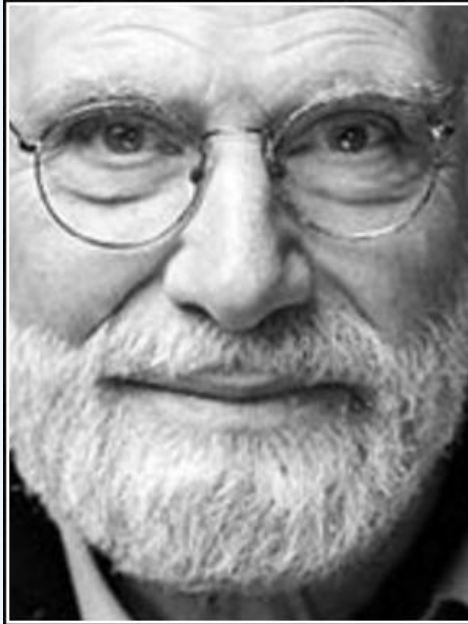
Presentation outline

- Challenges in the diagnosis of posterior circulation stroke
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- **Conclusions**

Conclusions

- The diagnosis of posterior circulation ischemia is based on **rapidly developing clinical signs of focal** (or occasionally global) **disturbance of cerebral function**, with no apparent cause other than that of vascular origin.
- Evaluation for the presence of **nystagmus**, **visual field defects** and **limb ataxia** should be performed in all patients with suspected posterior circulation ischemia.
- Any episode of new or **unusual headache + sudden onset of vertigo ± imbalance** should be thoroughly investigated to exclude a posterior circulation ischemic event.
- Evaluation with **MRI** should be performed in all patients with suspected cerebral ischemia. Negative findings should prompt to repeat the MRI in the absence of an alternative diagnosis.
- Patients presenting with **decreased LOC should receive CTA** in case basilar thrombolysis is suspected.
- Finally, a broader definition of TIA is likely to lead to more referrals of TIA mimics for assessment, with **implications for imaging capacity and other aspects of service provision**.

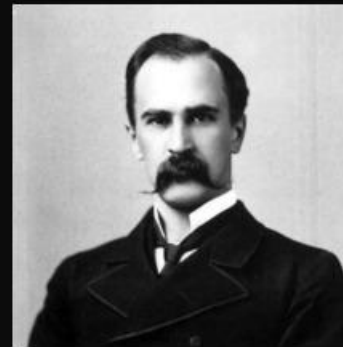
Thank you for your attention!



There is only one cardinal rule: One must always listen to the patient.

— *Oliver Sacks* —

AZ QUOTES



Listen to your patient, he is telling you the diagnosis.

~ William Osler

AZ QUOTES



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