



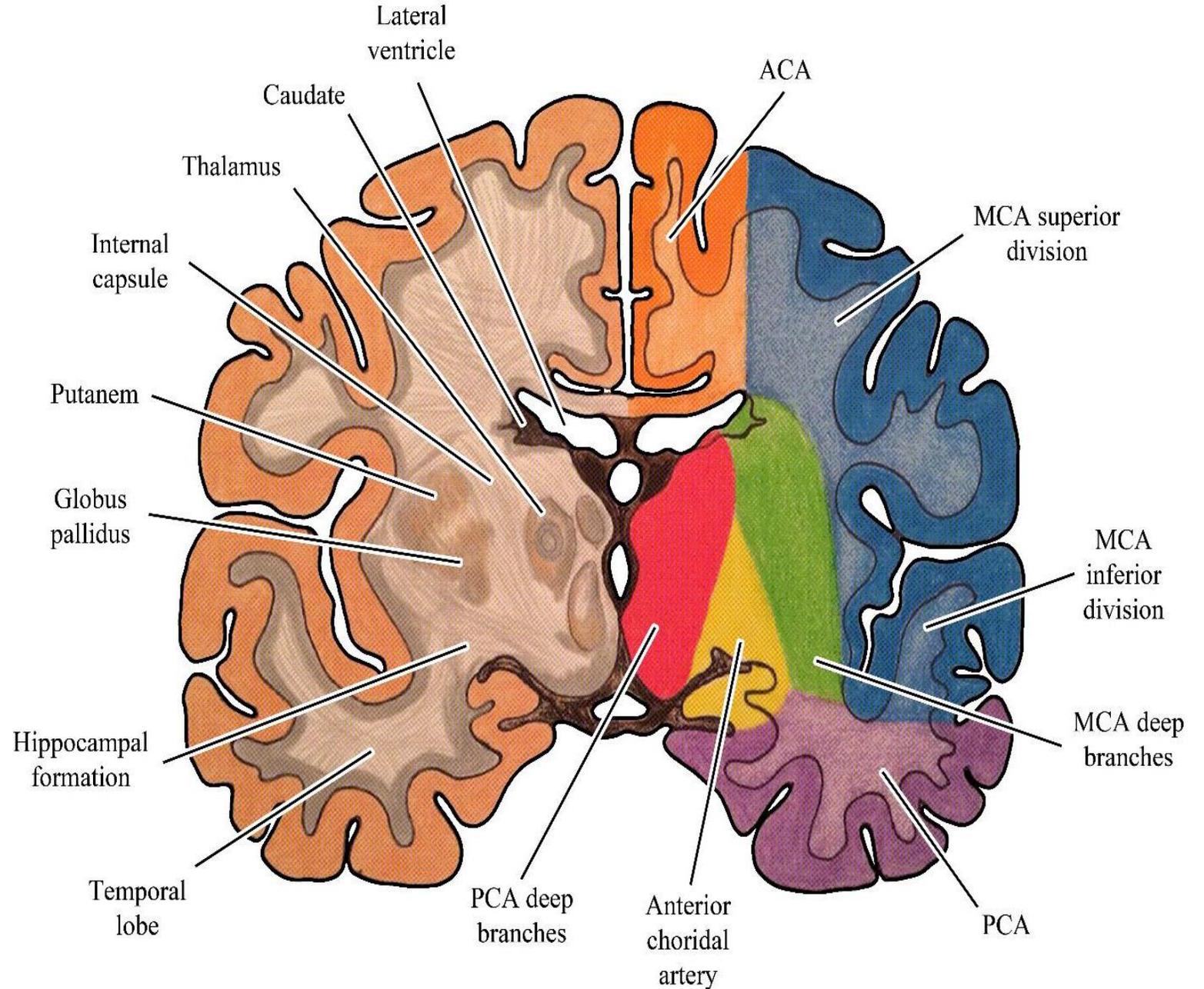
Cerebrovascular diseases

Neurology Rotation

Fourth Year Medical Students

Vascular Anatomy

The cerebral vasculature is divided into the **anterior** and **posterior** circulation, with the anterior (carotid) circulation supplying the **cerebral hemispheres** except the **medial temporal lobes** and a **portion of the occipital lobes**, and the posterior (vertebrobasilar) circulation supplying the **brainstem**, **thalami**, **cerebellum** and the **posterior portion of the cerebral hemispheres**



A) Anterior circulation

The right common carotid artery (CCA) branches from the innominate artery. The left CCA arises directly from the aorta.

The CCA divides in the neck into the internal carotid artery (ICA) and the external carotid artery.

The ICA travels behind the pharynx , entering the skull where it forms an S-shaped curve –the carotid siphon.

This portion of the ICA gives rise to the ophthalmic artery.

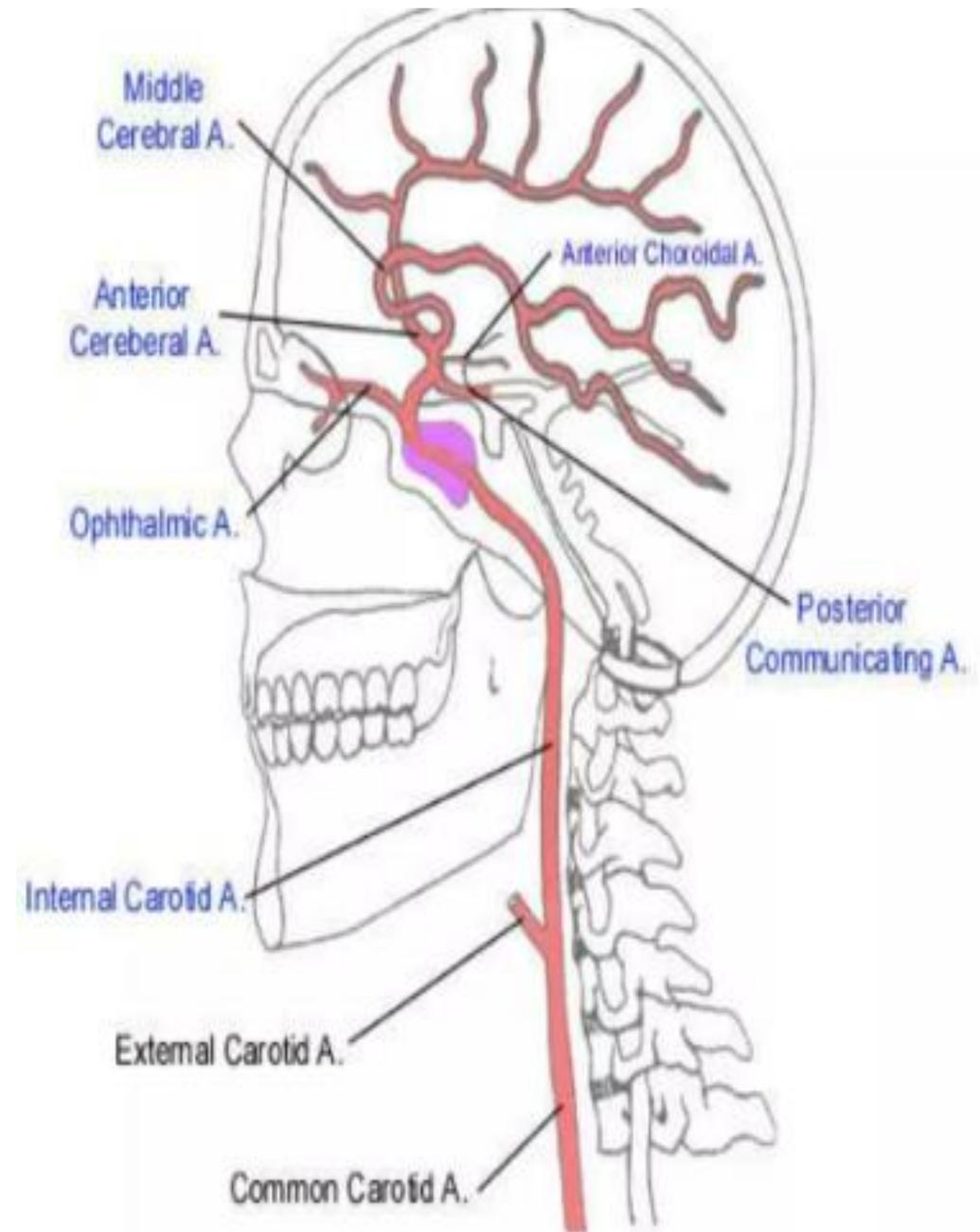
The ICA then penetrates the dura and gives off the anterior choroidal and posterior communicating arteries before bifurcating into the anterior cerebral (ACA) and middle cerebral arteries (MCA).

The ACA supplies the **anterior medial cerebral hemispheres** , the caudate nuclei and the basal frontal lobes.

The anterior communicating artery connects the two ACAs.

The MCA courses laterally , giving off lenticulostriate artery branches to the basal ganglia and internal capsule.

The MCA trifurcates into small anterior temporal branches and large superior and inferior divisions.

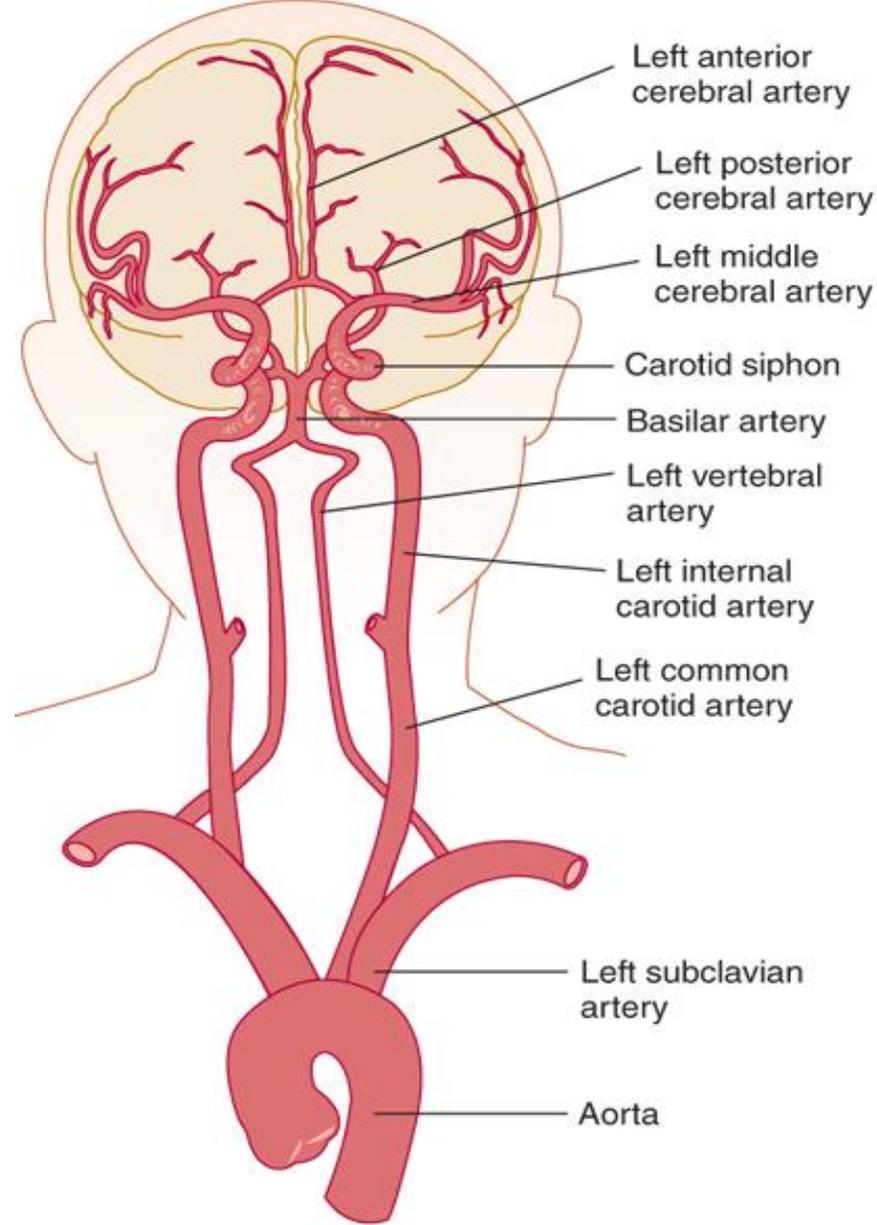


B) Posterior circulation

The first branch of each subclavian artery is the vertebral artery (VA).

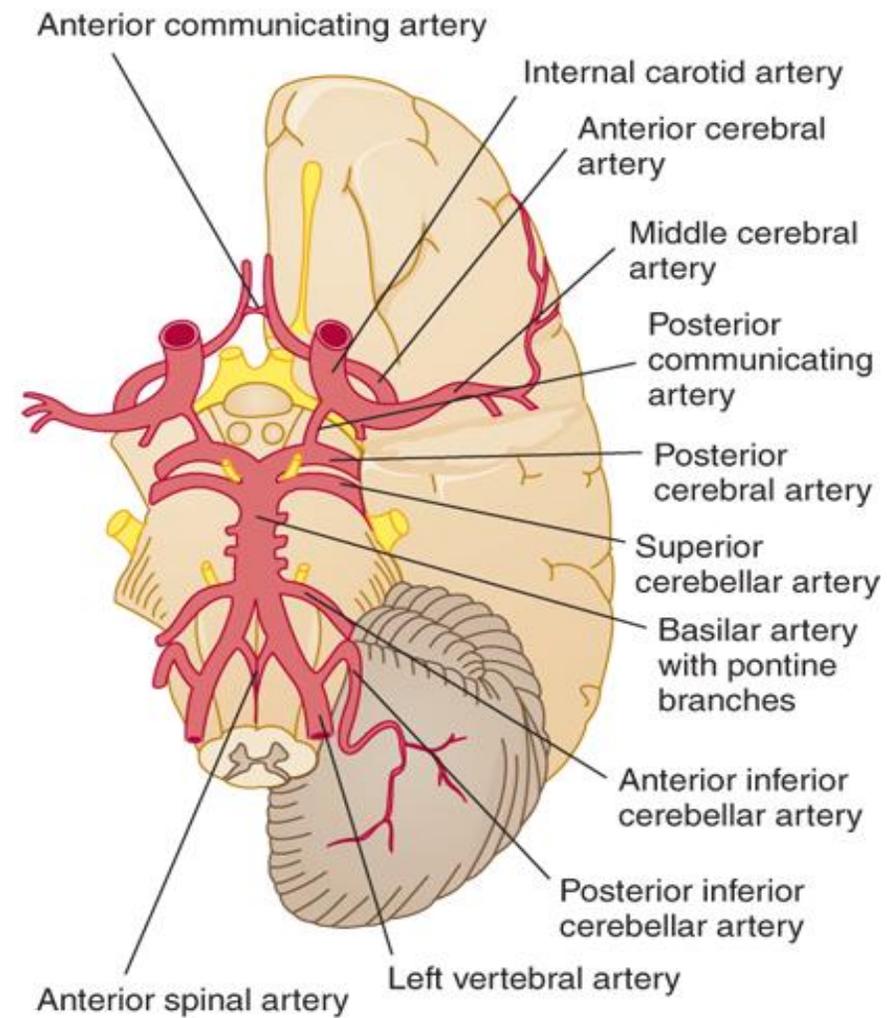
The VA enters the spinal column via the transverse foramina of C5 or C6 and runs within the intervertebral foramina, exiting to course behind the atlas before piercing the dura mater to enter the foramen magnum.

The intracranial VAs join to form the basilar artery at the ponto-medullary junction.



A

Source: Aaron L. Berkowitz: Clinical Neurology and Neuroanatomy: A Localization-Based Approach
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B

The intracranial VA gives off posterior and anterior spinal artery branches, penetrating arteries to the medulla and the posterior inferior cerebellar artery (PICA).

The basilar artery then runs in the midline along the clivus giving off bilateral anterior inferior cerebellar artery (AICA) and superior cerebellar artery (SCA) branches before dividing at the Ponto mesencephalic junction into the posterior cerebral arteries (PCA).

Small penetrating arteries arise at the basilar artery bifurcation to supply the medial portions of the midbrain and thalami.

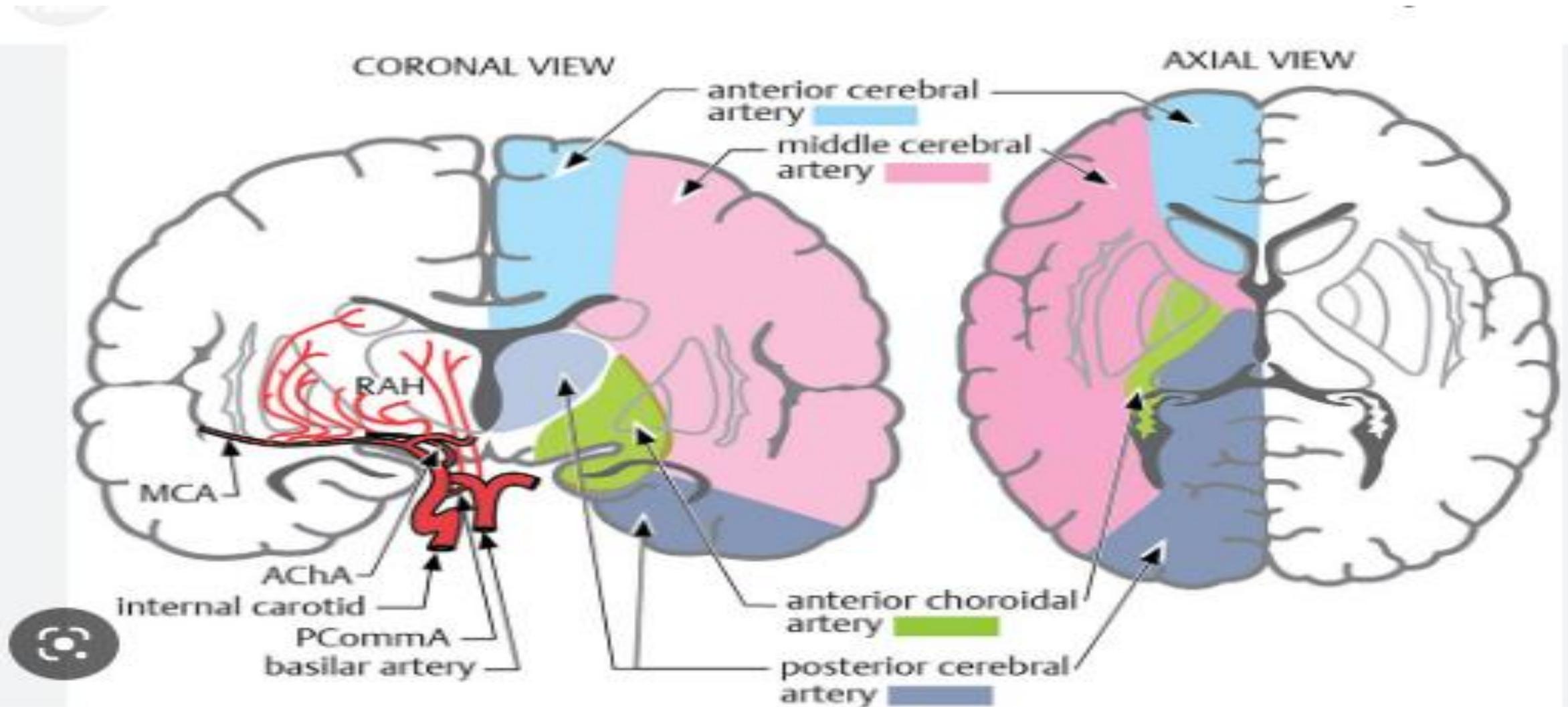
The vascular supply of the brainstem includes large paramedian arteries and smaller, short circumferential arteries that penetrate the basal portion of the brainstem into the tegmentum.

Long circumferential arteries course around the brainstem and give off branches to the lateral tegmentum.

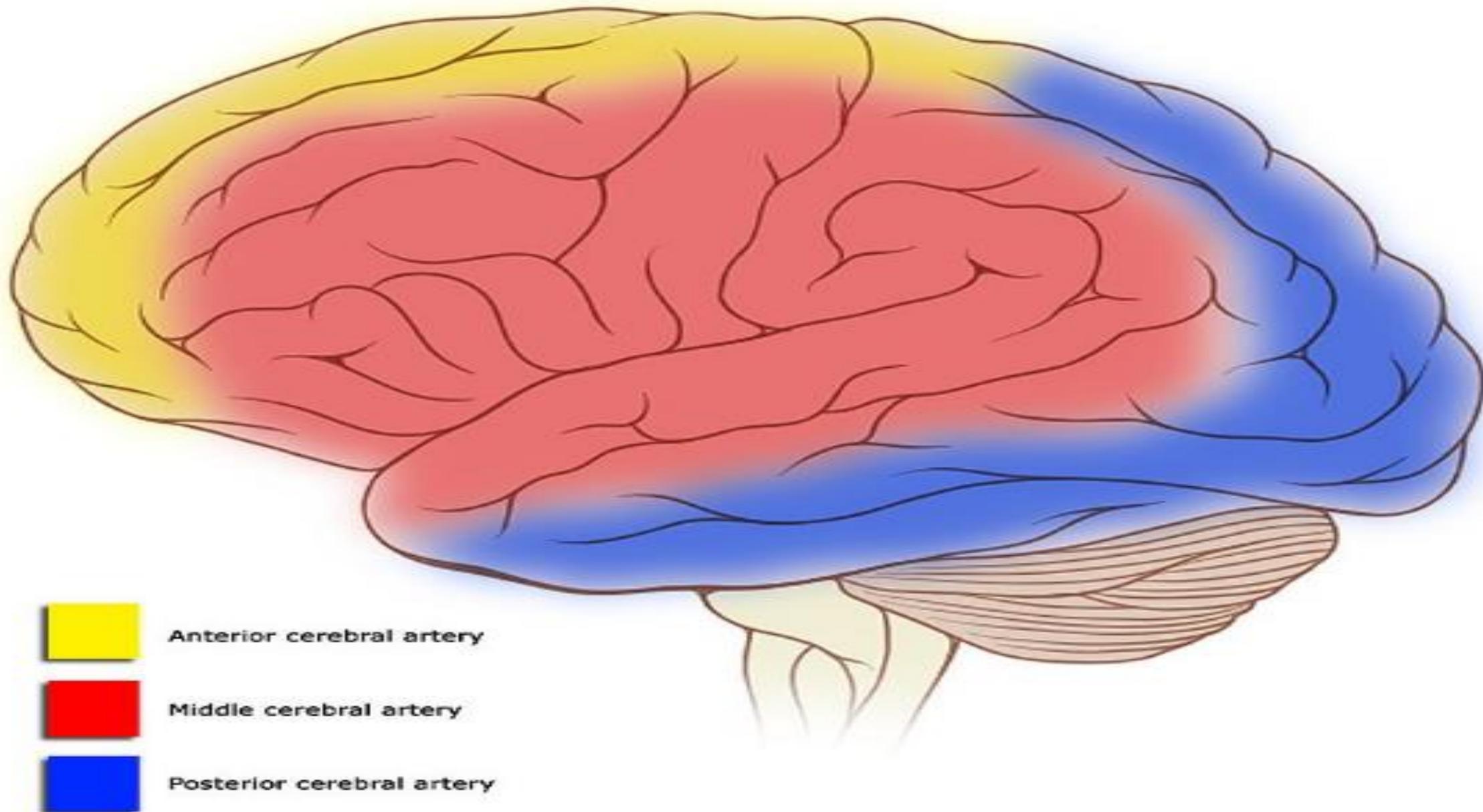
The PCA gives off penetrating arteries to the midbrain and thalamus, courses around the cerebral peduncles and then supplies the occipital lobe and the inferior surface of the temporal lobe.

The circle of Willis connects the anterior circulation of each side through the anterior communicating artery, and the posterior and anterior circulation of each side through the posterior communicating artery.

Each carotid artery supplies four-fifths of the brain, the vertebrobasilar circulation, one-fifth



Cortical vascular territories



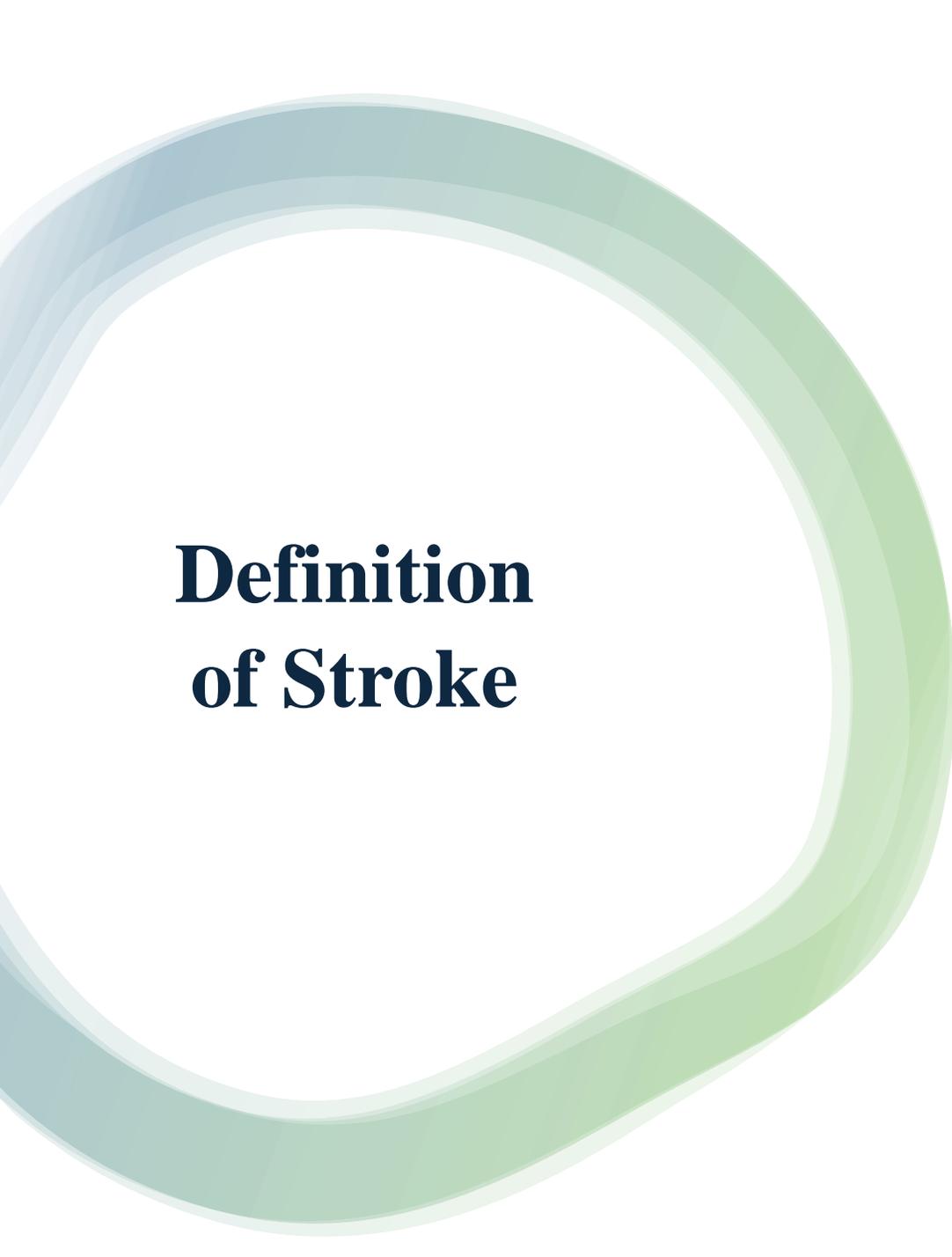
Anterior cerebral artery



Middle cerebral artery



Posterior cerebral artery



Definition of Stroke

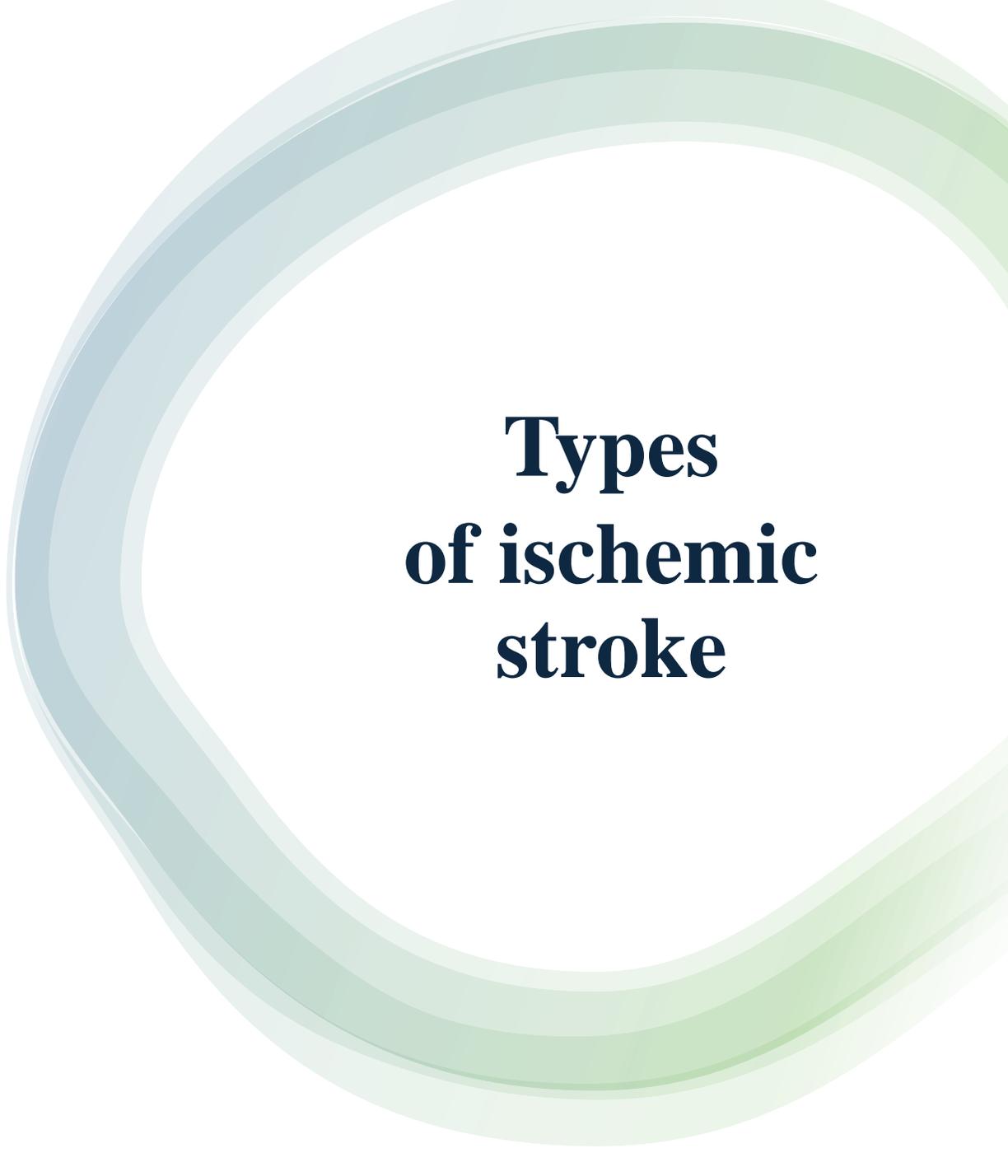
- **Strokes are a life-threatening emergency, and immediate medical attention is critical to prevent permanent damage or death.**
- **The World Health Organization (WHO) defines stroke as “rapidly developing clinical signs of focal (or global) disturbance of cerebral function, with symptoms lasting 24 hours or longer or leading to death, with no apparent cause other than vascular origin”**

- There are three main types of ischemic stroke:

1) Thrombotic strokes: caused by a blood clot that forms in an artery that supplies blood to the brain, also called an **atherosclerotic stroke**. **It affects mostly the large cervical and intracranial arteries.** Less commonly a clot forms within the lumen due to a **primary hematological disease (Polycythemia, Essential thrombocytosis or hypercoagulability).**

Vessel wall pathologies leading to thrombosis include vasoconstriction, **fibromuscular dysplasia and arterial dissection.**

Thrombosis of penetrating intracranial arteries is most often the consequence of hypertension, with hypertrophy of the media and deposition of fibrinoid material(lipohyalinosis) .

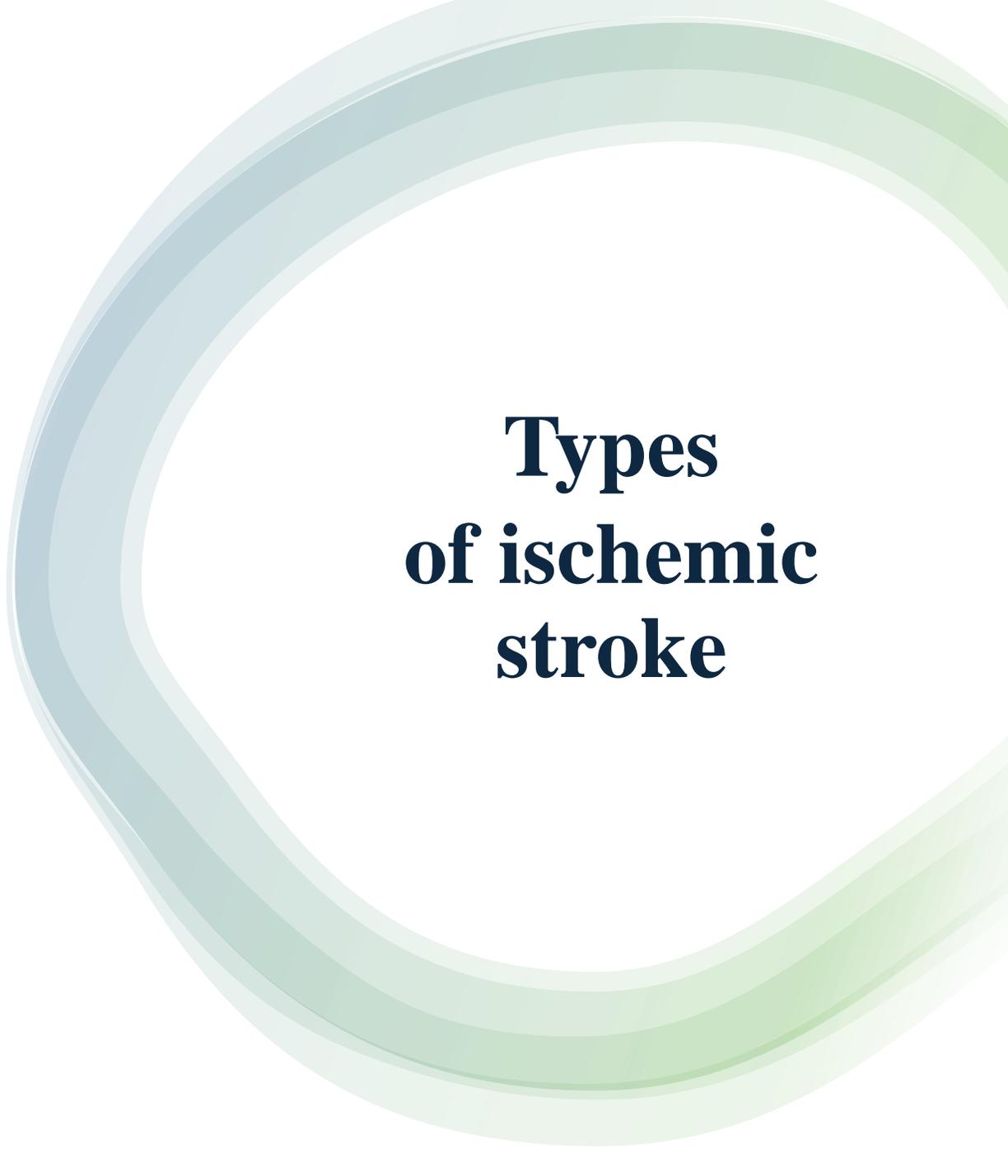


Types of ischemic stroke

2) Embolic strokes: When a clot forms somewhere else in the body and travels through the blood vessels to the brain. It gets stuck there and stops the flow of your blood. **The material arises proximally, mostly from the heart or from major arteries such as the aorta, ICAs and VAs and from systemic veins.** Atrial **fibrillation** increases the risk of clots forming in the heart that can then travel to the brain.

- **Cardiac sources of embolism include the heart valves, endocardium and clots or tumors within the atrial or ventricular cavities.**
- **Thrombi originating in systemic veins travel to the brain through cardiac defects such as an atrial septal defect or a patent foramen ovale, a process termed paradoxical embolism.**

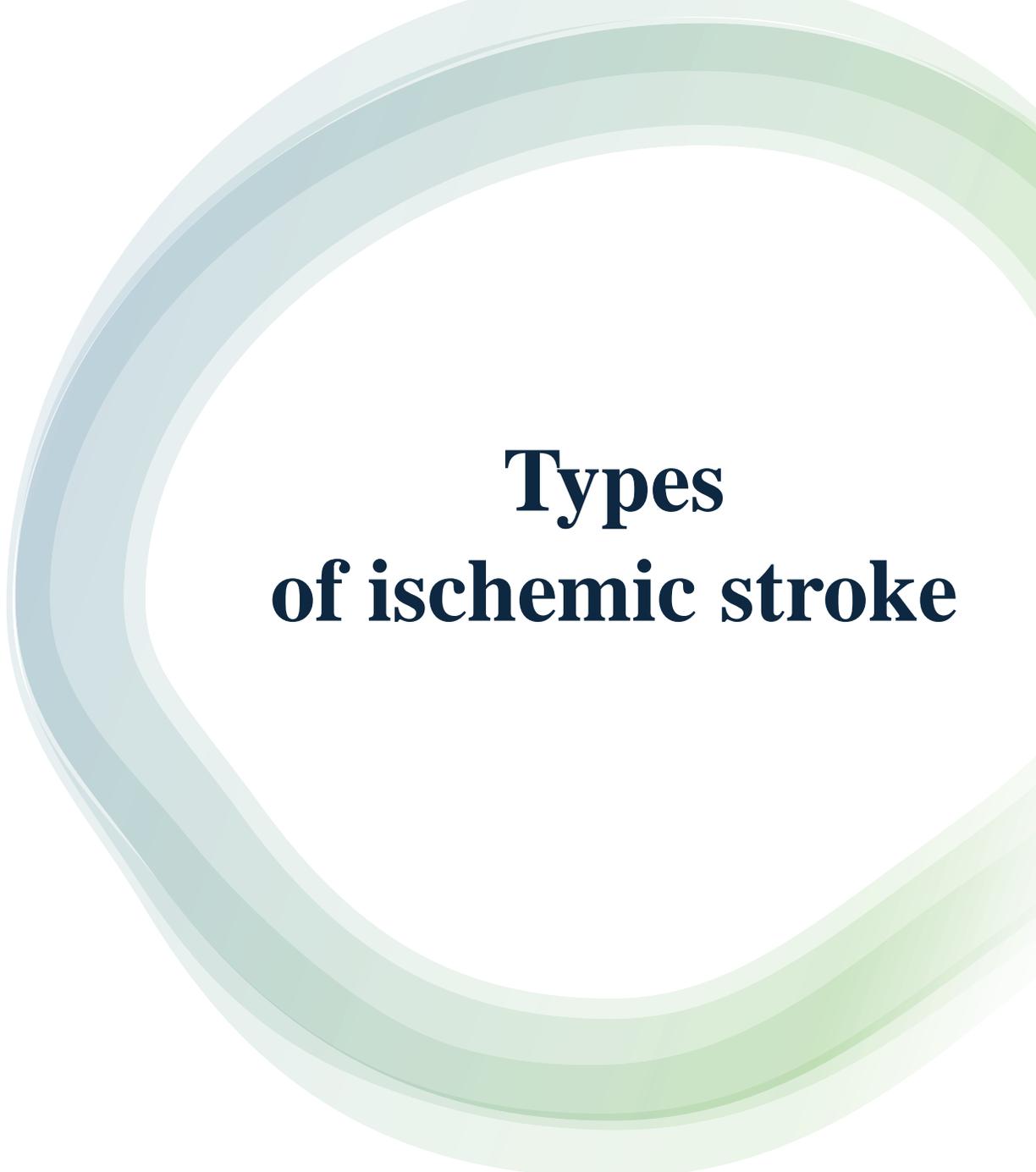
Occasionally, **air, fat, cholesterol crystals, bacteria and foreign bodies** enter the vascular system and embolize to brain vessels.



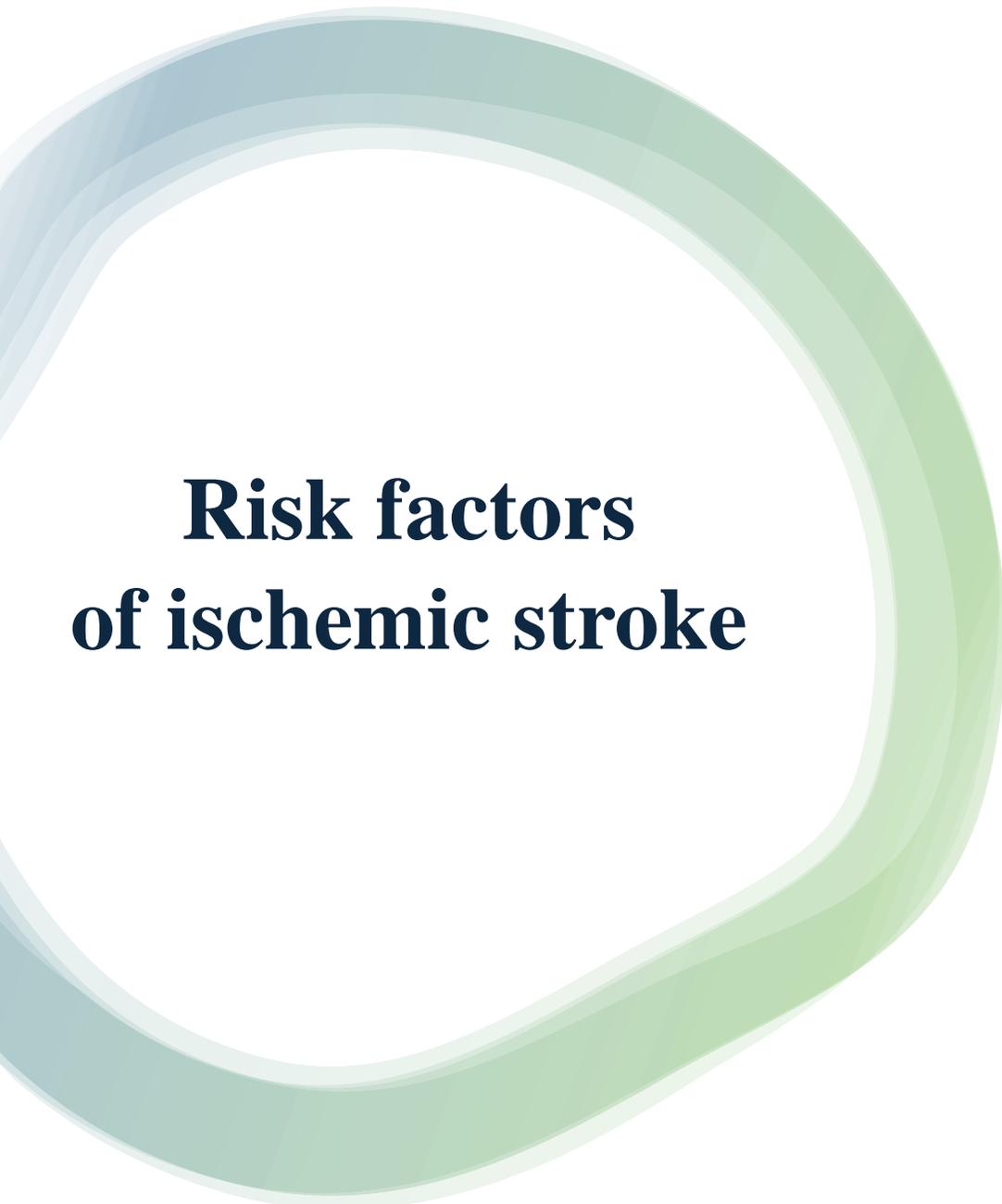
Types of ischemic stroke

3) Systemic hypoperfusion

- Decreased blood flow to brain tissue may be caused by low systemic perfusion pressure .
- The most common causes are **cardiac pump failure**(most often due to myocardial infarction or arrhythmia) and **systemic hypoperfusion** (due to blood loss or hypovolemia).
- The lack of perfusion is more generalized than in localized thrombosis or embolism and affects brain diffusely and bilaterally.
- **Poor perfusion is most critical in border zone or so-called watershed regions at the periphery of the major vascular supply territories**, for example, between the ACA and MCA or between the MCA and PCA.



Types of ischemic stroke



Risk factors of ischemic stroke

- Age over 60 years old
- History of hypertension, heart diseases, hyperlipidemia, or diabetes.
- Arrhythmia
- Smoking History
- Family history of strokes

Complications of ischemic strokes include Permanent weakness, Seizures and Vascular Dementia

Classification of Ischemic Stroke by Anatomy

Site of the lesion	Associated effects
Anterior cerebral artery	Contralateral hemiparesis and sensory loss, lower extremity > upper
Middle cerebral artery	Contralateral hemiparesis and sensory loss, upper extremity > lower Contralateral homonymous hemianopia Aphasia
Posterior cerebral artery	Contralateral homonymous hemianopia with macular sparing Visual agnosia

Weber's syndrome (branches of the posterior cerebral artery that supply the midbrain)

Ipsilateral CN III palsy
Contralateral weakness of upper and lower extremity

Posterior inferior cerebellar artery (lateral medullary syndrome, Wallenberg syndrome)

Ipsilateral: facial pain and temperature loss
Contralateral: limb/torso pain and temperature loss
Ataxia, nystagmus

Anterior inferior cerebellar artery (lateral pontine syndrome)

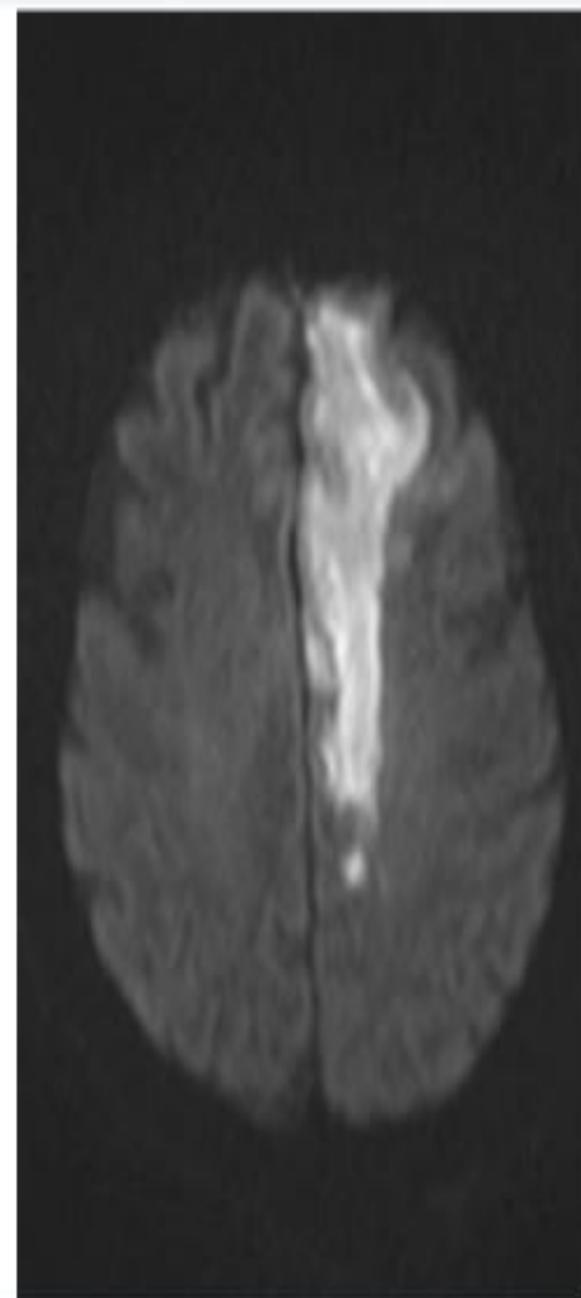
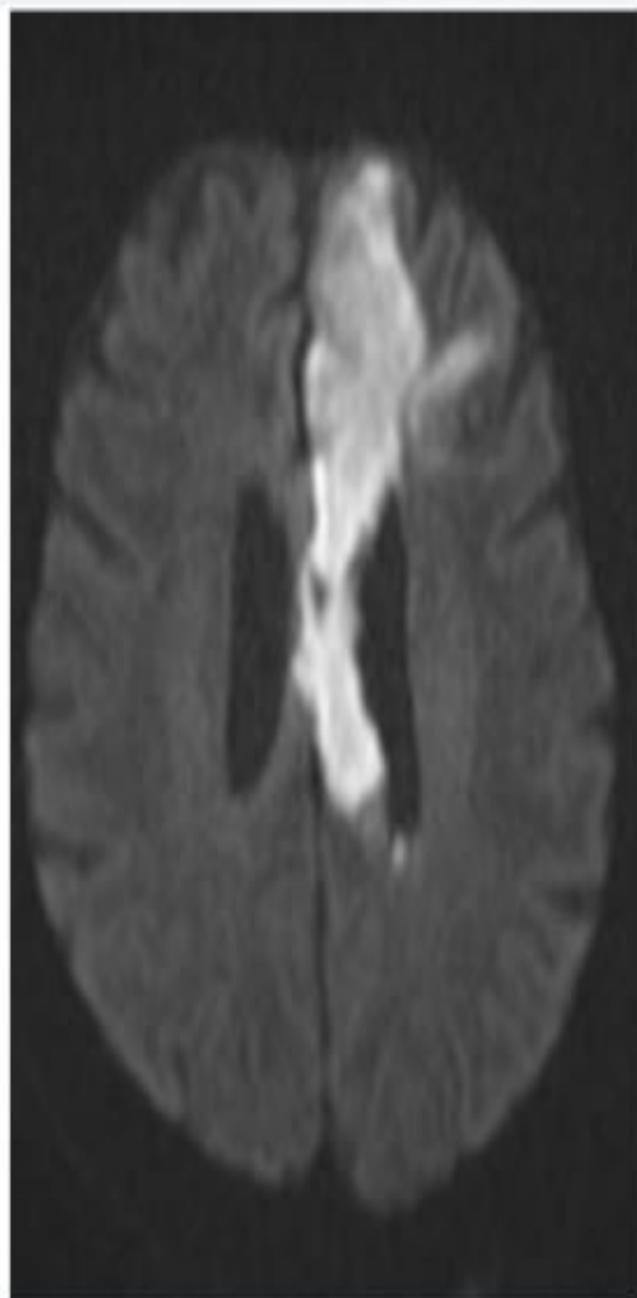
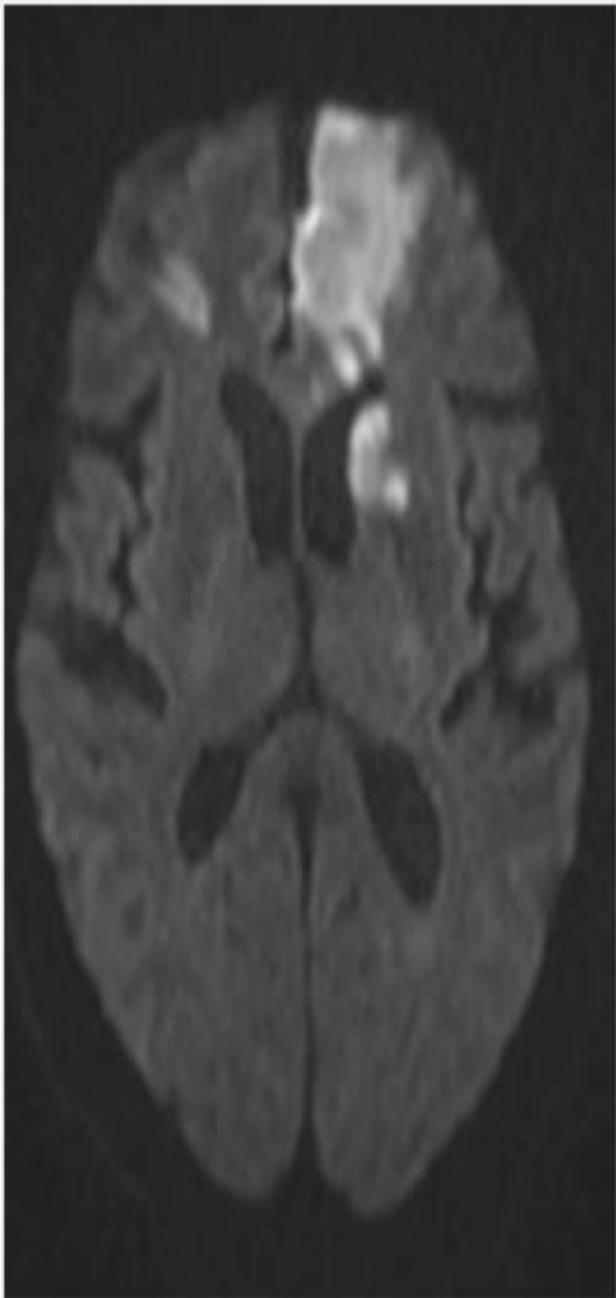
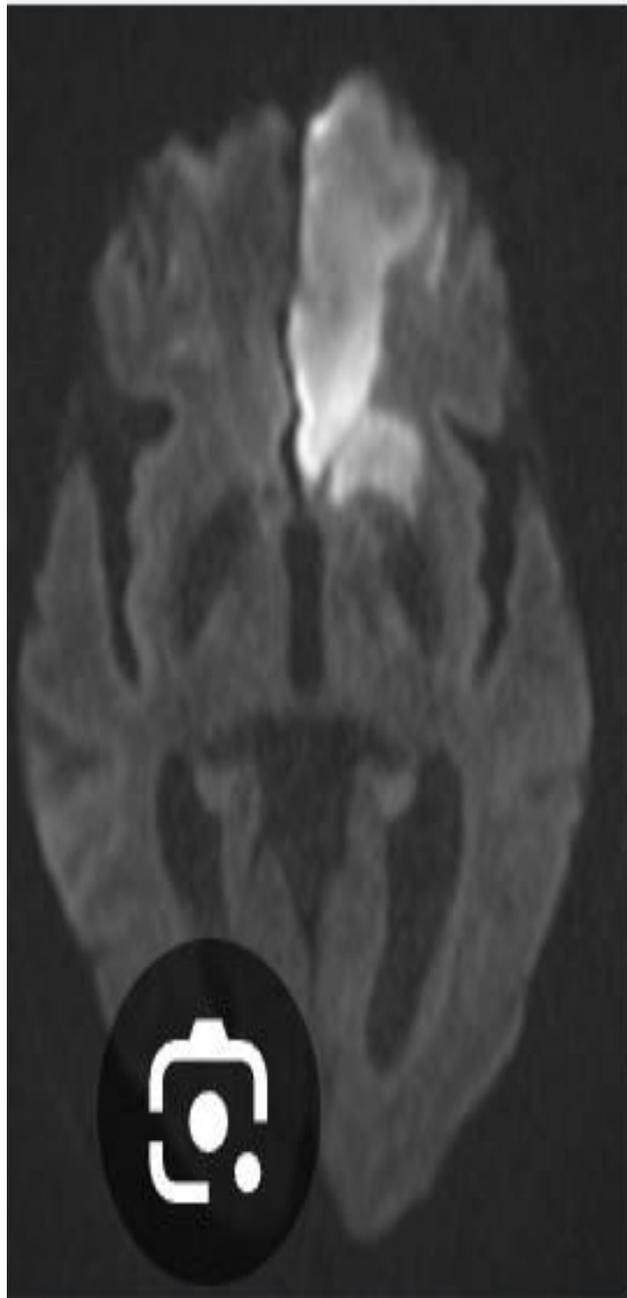
Symptoms are similar to Wallenberg's (see above), but:
Ipsilateral: facial paralysis and deafness

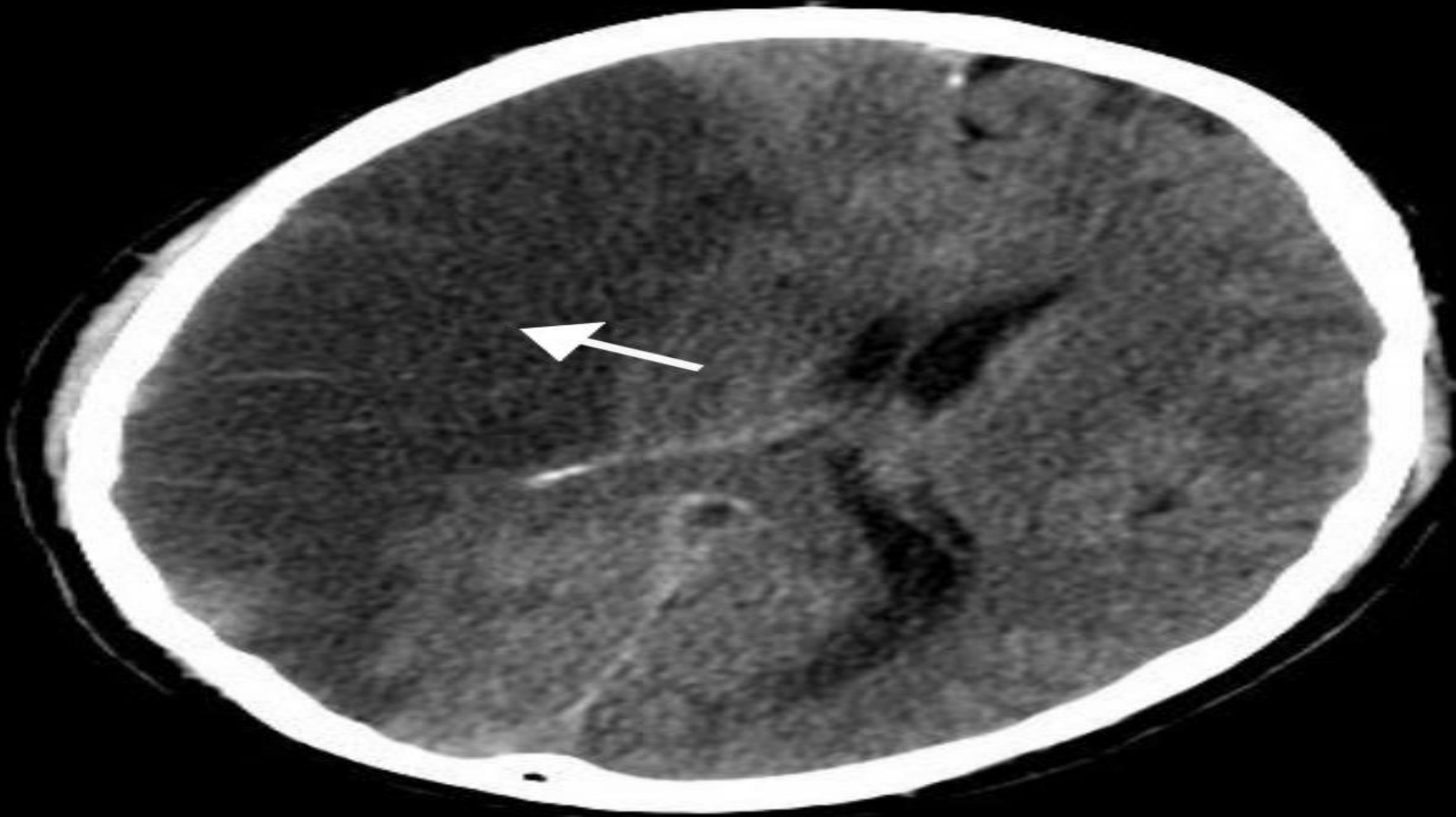
Retinal/ophthalmic artery

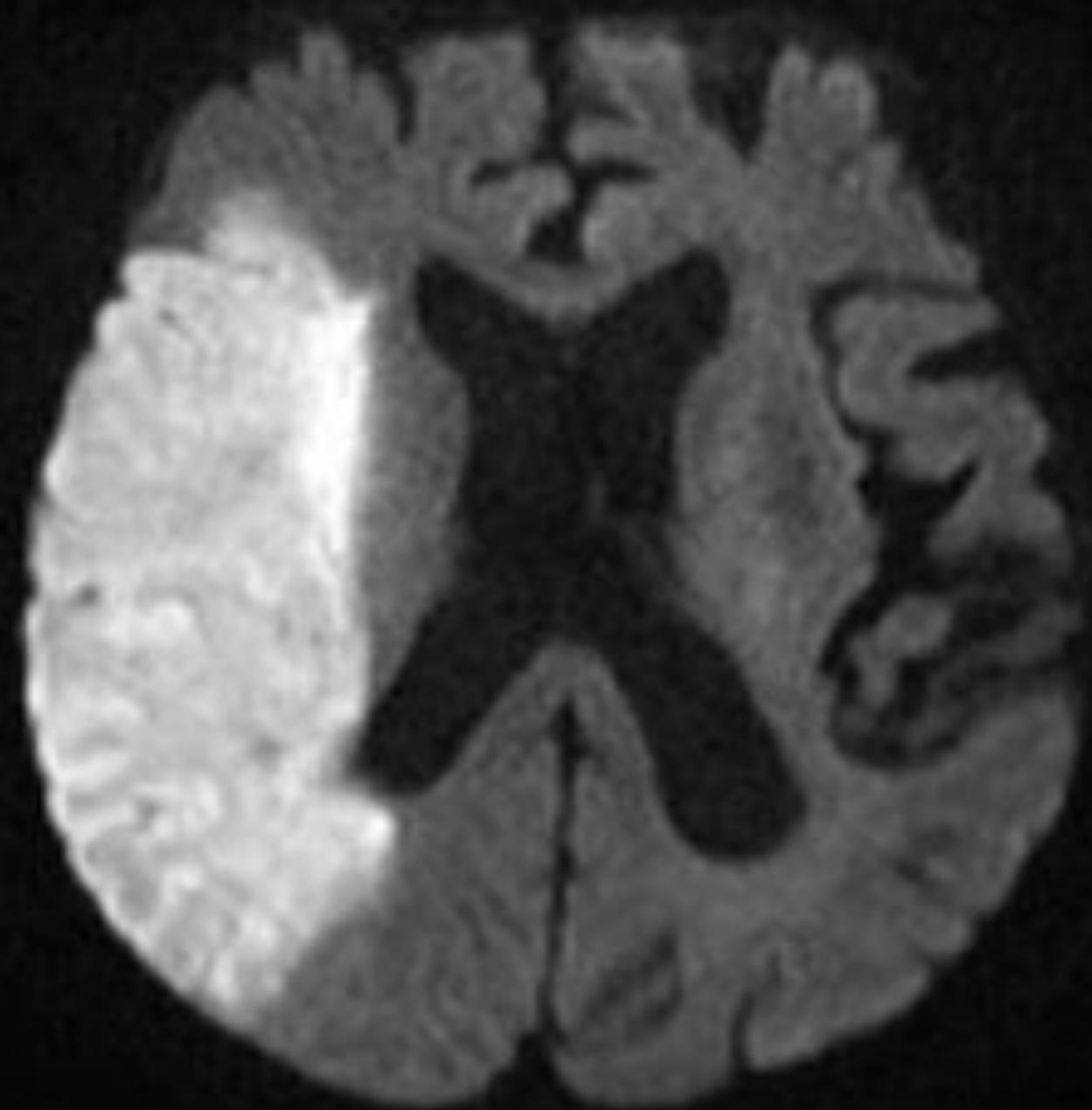
Amaurosis fugax

Basilar artery

'Locked-in' syndrome





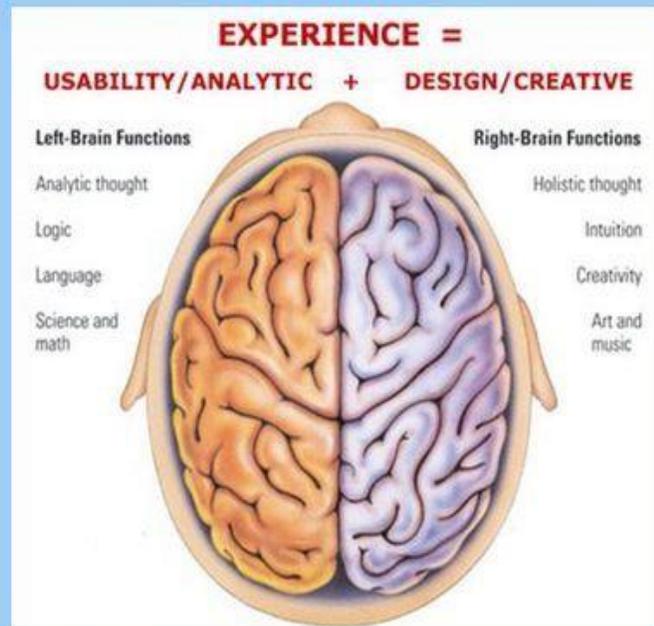


Common Ischemic Stroke Syndromes

- **Anterior Circulation:** Left/Right cerebral hemisphere lesions are most often caused by carotid artery occlusion, embolism to the MCA or its branches or basal ganglionic intracerebral hemorrhages.

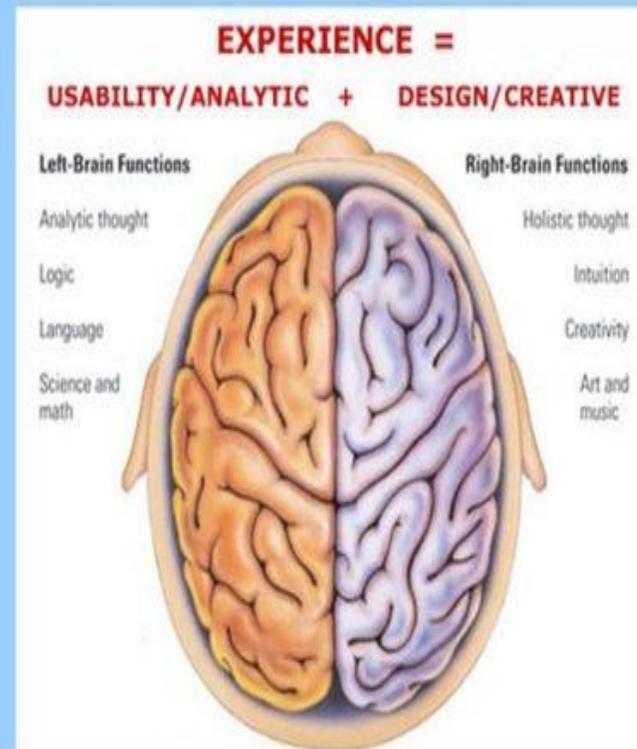
Left Hemisphere Stroke

- Effects right side of body
- Speech and language
- Aphasia
- Slow, cautious behavior
- Memory problems



Right Hemisphere Stroke

- Effects left side of body
- Spatial or perceptual abilities
- Impulsive, unaware of their impairments
- Left-sided neglect
- Short-term memory problems
- Often non-dominant



- **Posterior circulation:**

1. Lateral medullary stroke (Wallenberg syndrome, usually due o intracranial VA or posterior inferior cerebellar artery/ PICA occlusion)

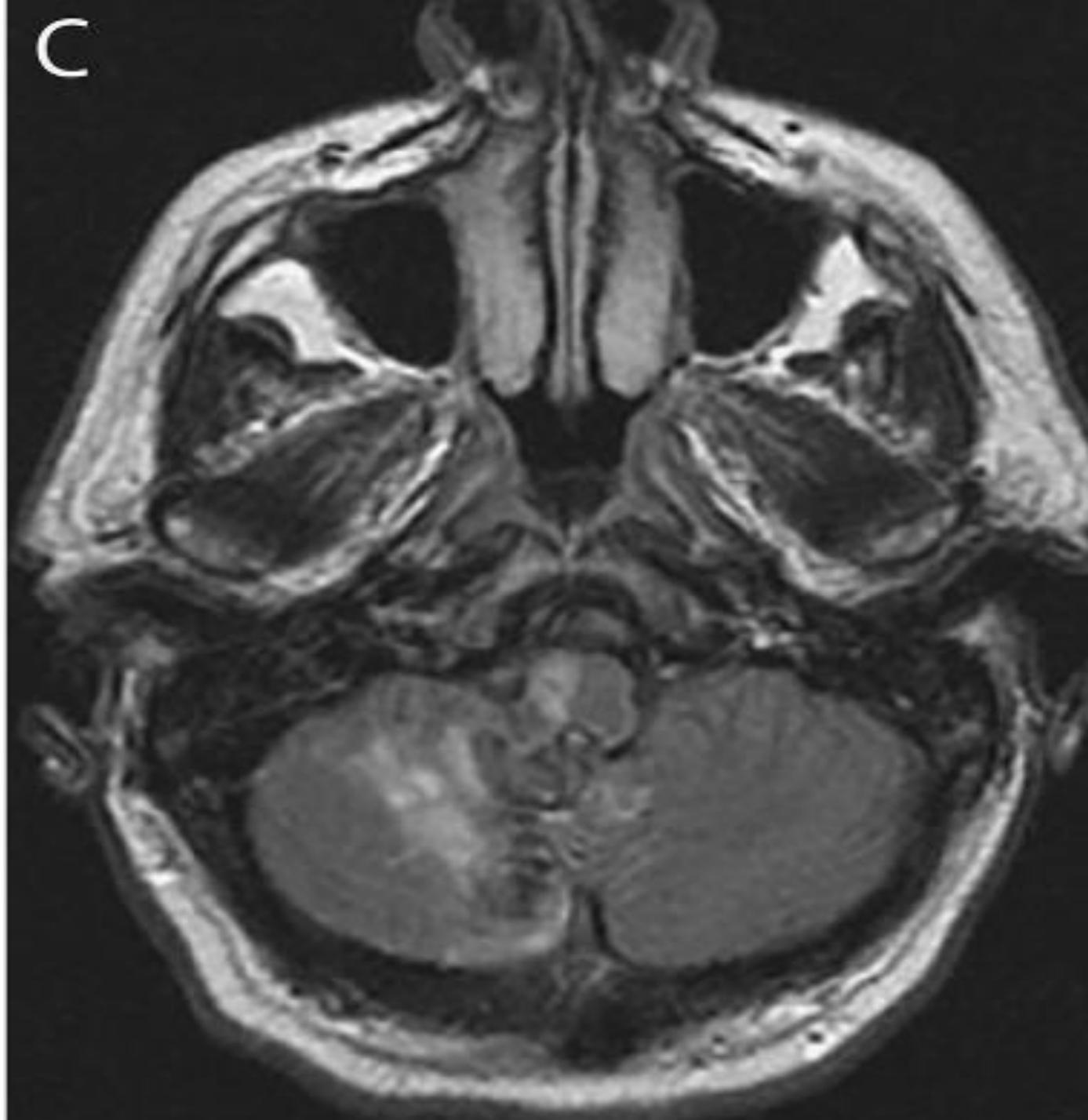
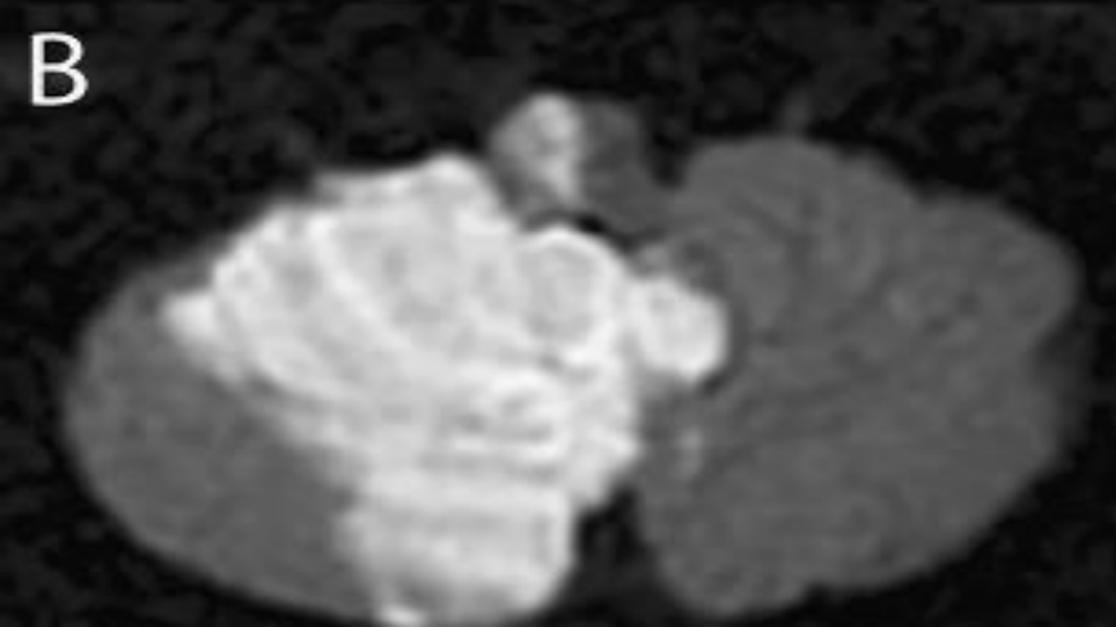
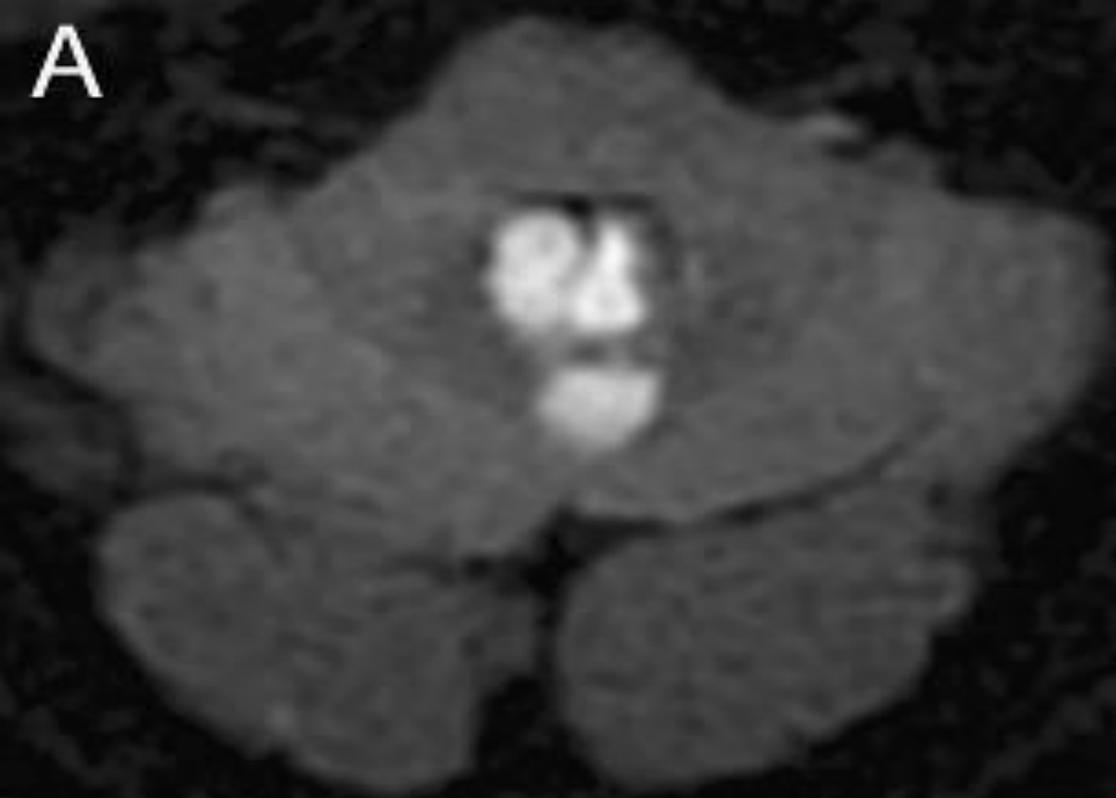
2. Bilateral pontine base and often medial tegmentum stroke(usually due to basilar artery occlusion or pontine hemorrhage) causes:

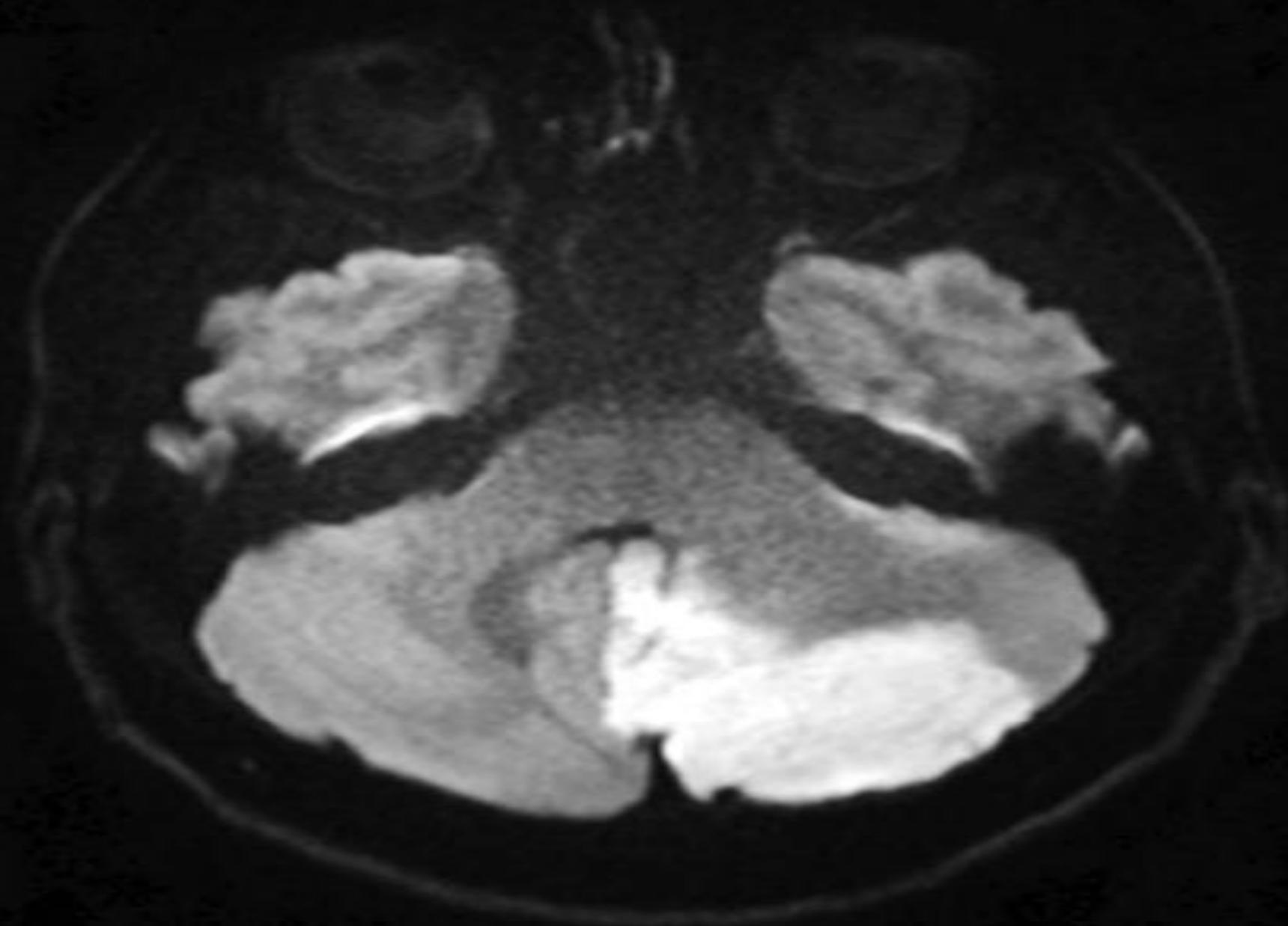
- Quadriparesis
- Unilateral or bilateral conjugate gaze paresis; sometimes internuclear ophthalmoplegia or 6th nerve palsy
- When the **medial tegmentum** is involved bilaterally, **coma**.

3. Cerebellar infarction (usually due to embolism to the PICA or SCA, or cerebellar hemorrhage) causes:

- Gait ataxia
- Dysarthria
- Ipsilateral arm dysmetria







4. Left PCA territory stroke

causes:

- a) Right homonymous hemianopia
- b) At times, amnesia
- c) Alexia (disorder of reading) without agraphia when the splenium of the corpus callosum is involved

5. Right PCA territory stroke

causes:

- a) Left homonymous hemianopia
- b) At times, left-sided visual neglect

PCA territory infarcts are most often caused by embolism arising from the heart, aorta or VAs



- Lacunar syndromes

Lacunar strokes are most often due to occlusion of a penetrating artery. Lacunar strokes may occur in either the anterior or the posterior circulations.

1. **Pure motor stroke**: Weakness of the contralateral arm, face and leg without sensory, visual or cognitive/behavioral signs. Common locations include **the corona radiata, posterior limb of the internal capsule and pons**.
2. **Pure sensory stroke**: Paresthesia of the contralateral body, limbs and face without motor, visual or cognitive abnormalities. The most common location is due to infarction in the **ventral posterior thalamus** .
3. **Sensorimotor stroke**: Combination of motor and sensory lacunes due to infarction in the **ventral posterior thalamus and adjacent posterior limb of the internal capsule**.
4. **Dysarthria-clumsy hand syndrome**: Slurred speech and clumsiness of the contralateral hand. The most common location is **in the base of the pons**.
5. **Ataxic hemiparesis**: Weakness and ataxia of the contralateral limbs, often greater in the leg and foot than in the arm and hand. The most common locations are the base of the **pons, posterior limb of the internal capsule and corona radiata**.

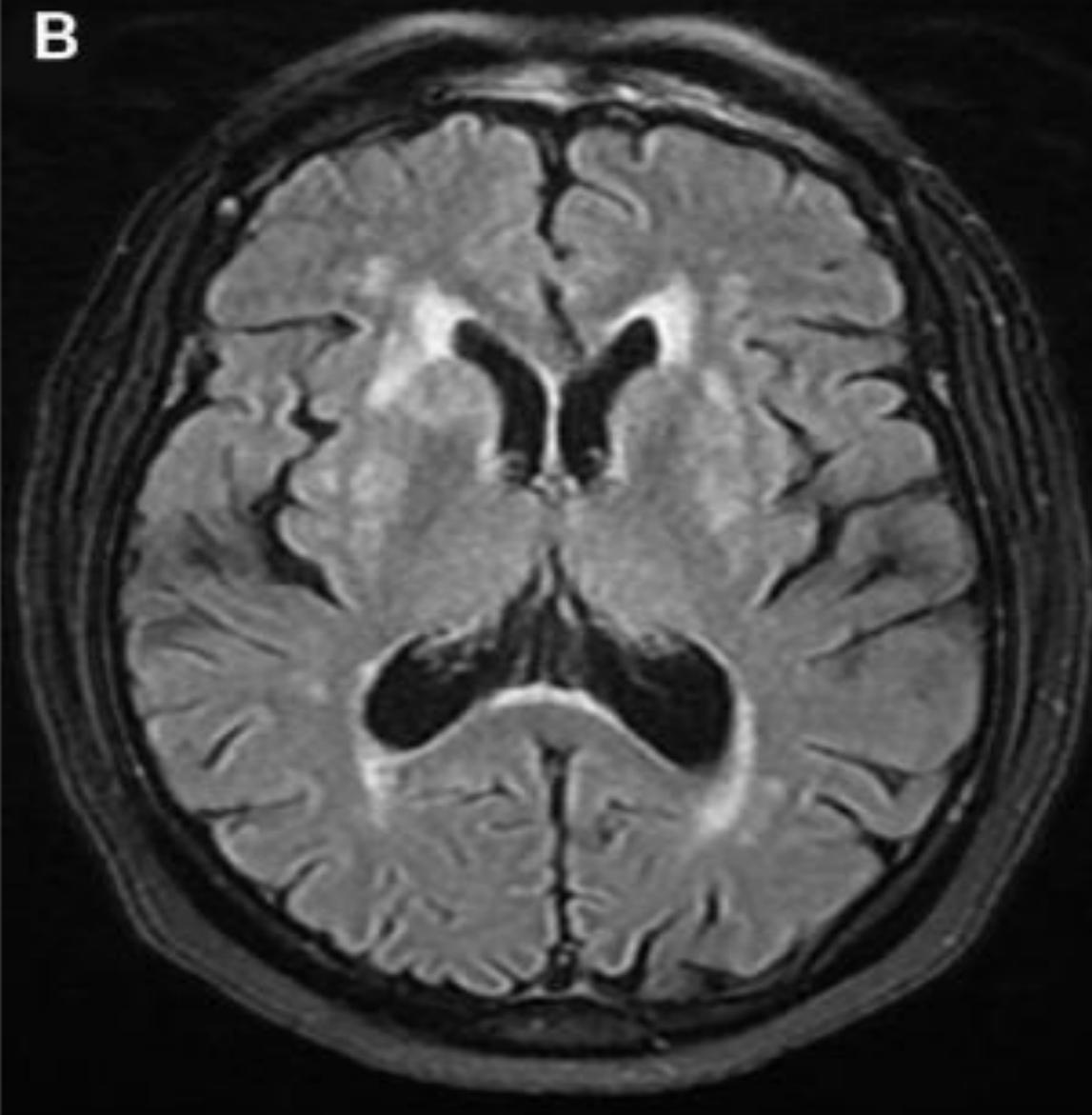
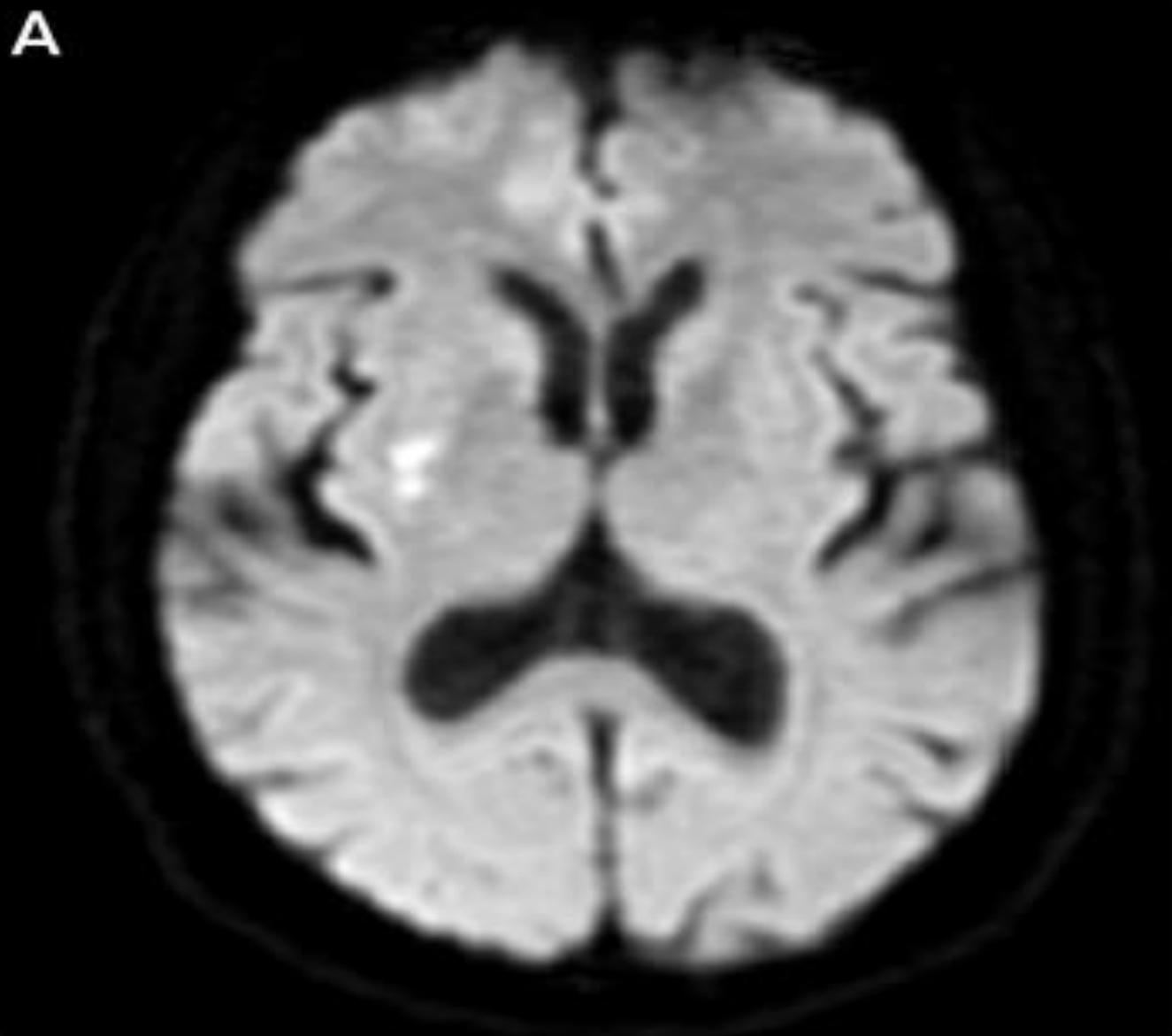


FIGURE 5-2

Imaging of the patient in **CASE 5-1**. Axial diffusion-weighted MRI (A) shows an acute infarct involving the right lentiform nucleus and internal capsule, and axial fluid-attenuated inversion recovery (FLAIR) MRI (B) shows moderate white matter hyperintensity burden.

- **Arterial dissection:**
- Dissection of the carotid or vertebral arteries may lead to ischemic stroke
- **a) Carotid dissection:**
- - Typically presents with severe retro-orbital headache ipsilateral to the lesion
- Strokes involve the anterior circulation and occur either by thrombosis of the ICA or more commonly by an embolus arising from the dissection.
- On examination: patients may have an **ipsilateral Horner's syndrome** due to the involvement of the ascending oculosympathetic tract. Perspiration is preserved because those fibers ascend with the external carotid artery.
- **b) VA dissection** may be produced by neck manipulation or trauma and is commonly associated with ipsilateral neck pain and stroke in the posterior circulation.

Transient Ischemic Attack

- Ischemic strokes also include something called a "mini-stroke" or a TIA (transient ischemic attack). This is a temporary blockage in blood flow to your brain. The symptoms usually last for just a few minutes or may go away in 24 hours.

The mechanisms of TIA are identical to those of ischemic stroke.

Patients who have had a TIA have a 10% risk of stroke in the 90 days following the event, and the greatest risk is within the first 24 hours following a TIA.

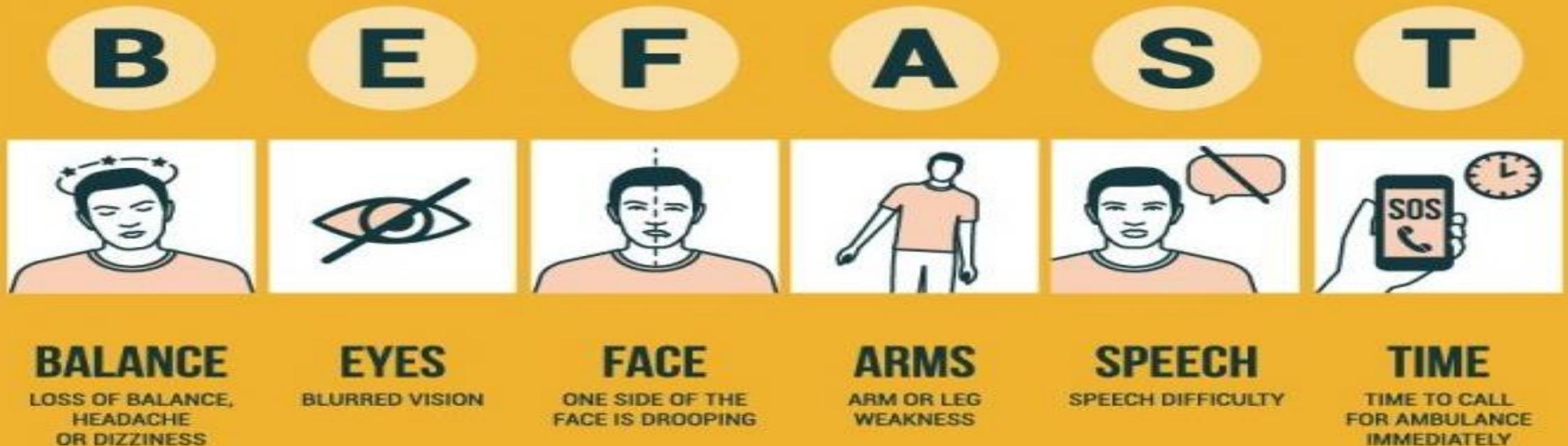
Therefore, the evaluation should be identical to that for a completed stroke and should be conducted just as quickly.



Assessment of Ischemic Strokes

Whilst the diagnosis of stroke may sometimes be obvious in many cases the presenting symptoms may be vague and accurate assessment difficult.

The BE FAST screening tool is widely known and has a positive predictive value of 78%. A variant of BE FAST called the ROSIER score is useful for medical professionals.



ROSIER score

Exclude hypoglycaemia first, then assess the following:

Assessment	Scoring
Loss of consciousness or syncope	- 1 point
Seizure activity	- 1 point
New, acute onset of:	
• asymmetric facial weakness	+ 1 point
• asymmetric arm weakness	+ 1 point
• asymmetric leg weakness	+ 1 point
• speech disturbance	+ 1 point
• visual field defect	+ 1 point

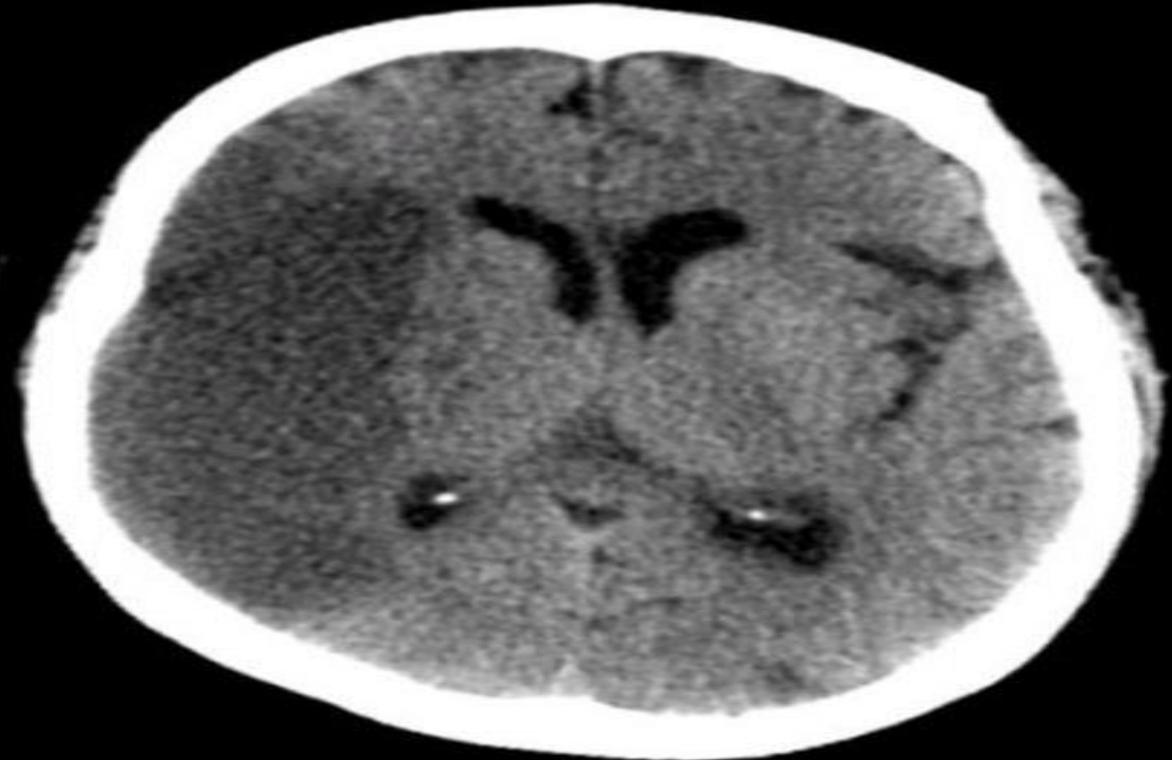
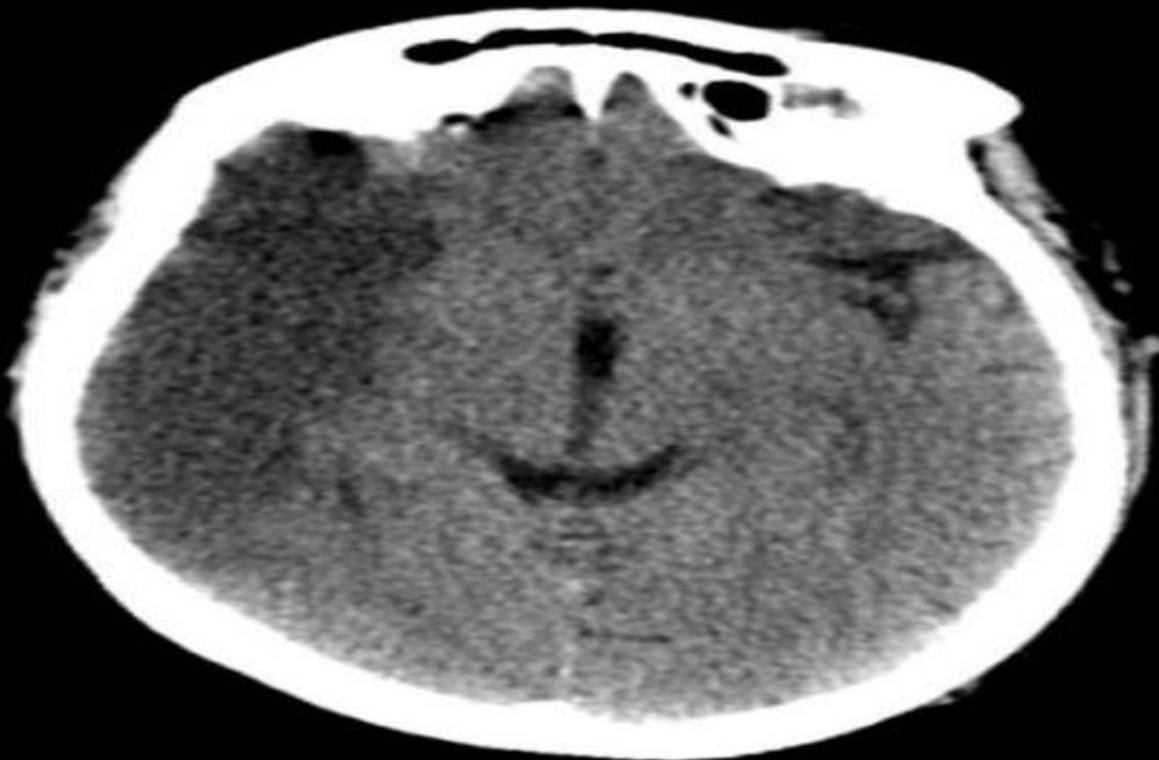
A stroke is likely if > 0 .

Assessment of Ischemic Strokes

A non-contrast CT brain scan is the first line radiological investigation for suspected stroke as it helps to exclude hemorrhagic strokes or another pathology such as brain tumors before considering the usage of any antiplatelets or thrombolytics and thrombectomy in acute Stroke Management.

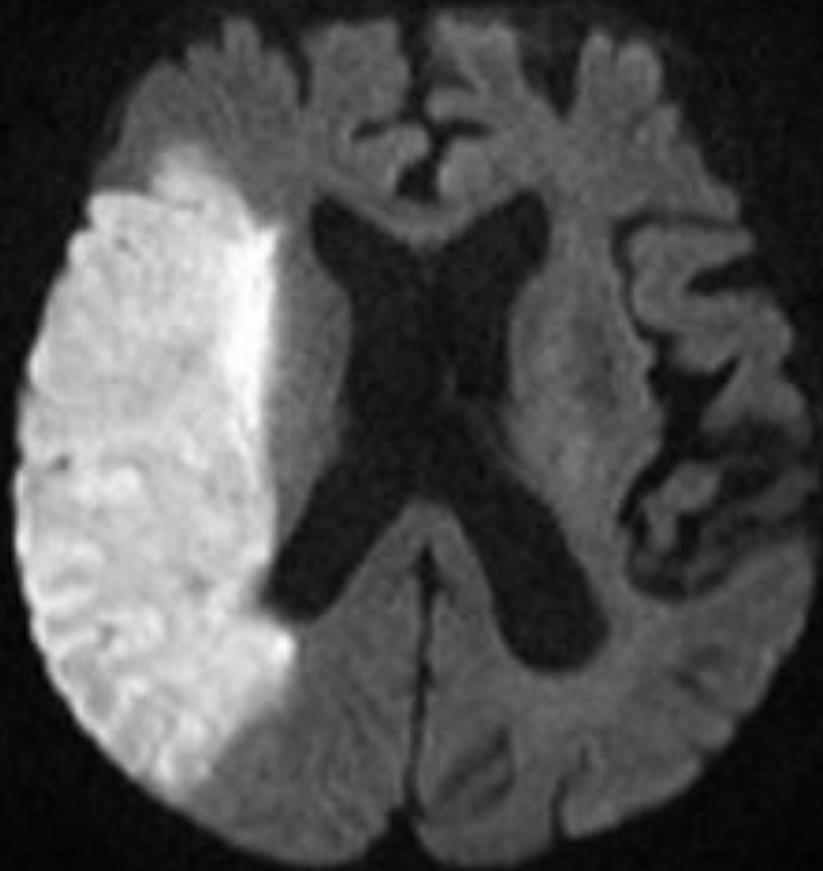
Acute ischemic strokes show areas of low density in the grey and white matter of the territory. Acute hemorrhagic strokes typically show areas of hyperdense material (blood) surrounded by low density (oedema).

MRI Brain with diffusion weighted imaging is more sensitive to acute brain infarction than is CT.





R

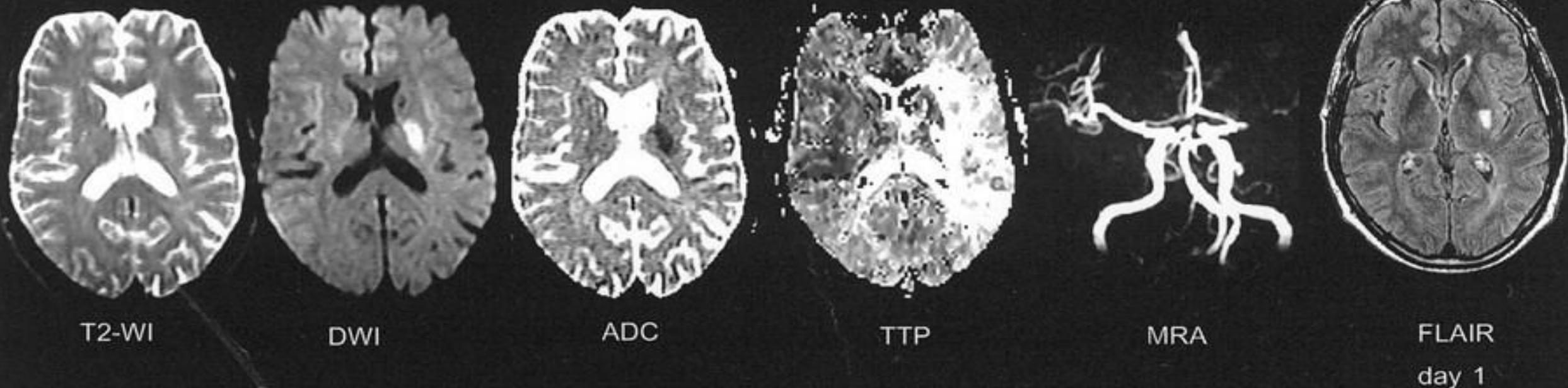


FrontalCortex.com

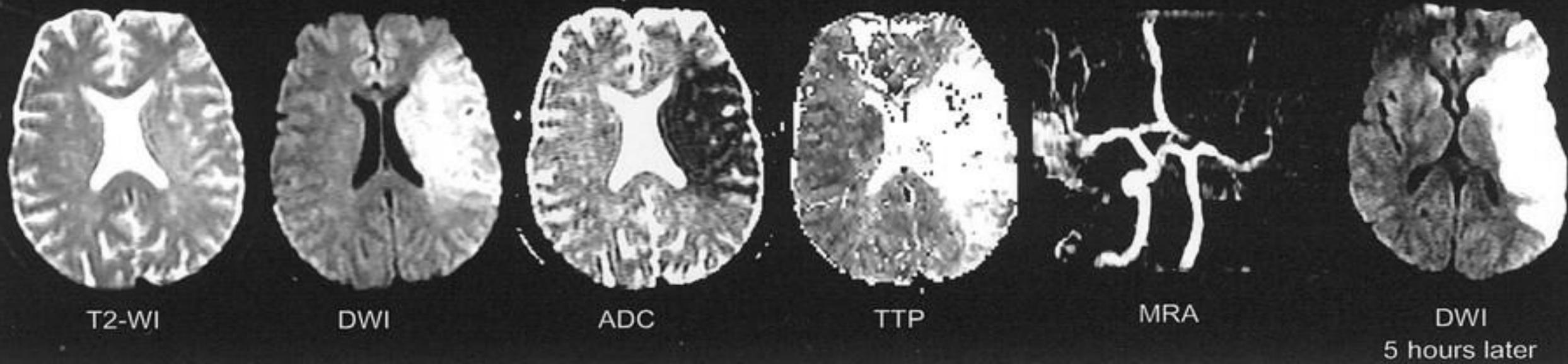


Acute Right MCA stroke on MRI (DWI on the left and ADC sequence on the right)

A: NIHSS 14, MRI 1.5 h after symptom onset, i.v. thrombolysis, non MMI



B: NIHSS 21, MRI 2h after symptom onset, MMI, hemicraniectomy

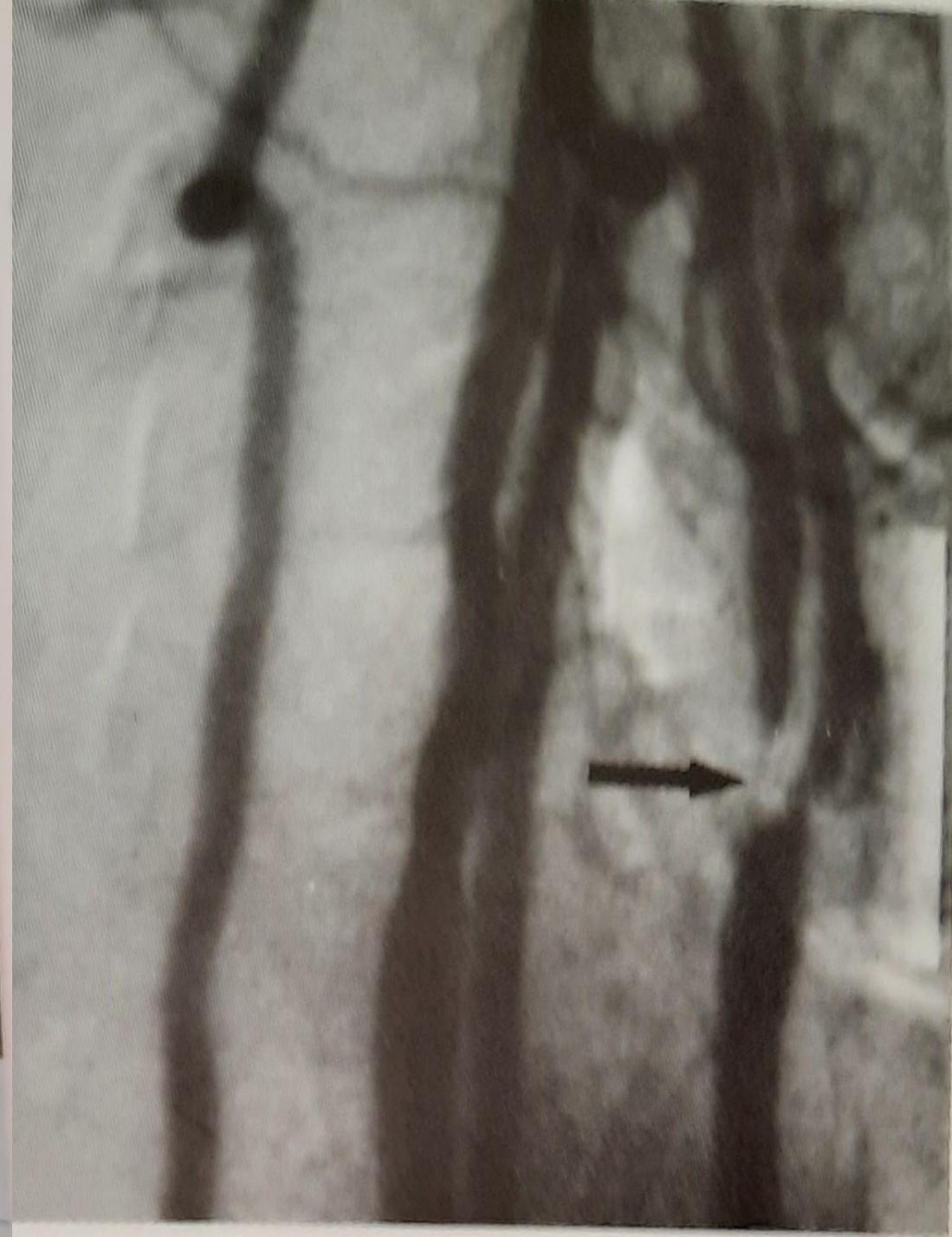


Diagnostic Work up for Ischemic Strokes

- **Laboratory Work Up:** Complete blood count, PT, PTT, Kidney function test, liver function test, Cardiac enzymes, Electrolytes, RBS level, HBA1C, Lipid Profile. Rheumatological work up is sometimes needed when history is significant for Antiphospholipid syndrome, Bechet disease or Vasculitis.
- **Computerized tomography (CT) scan.**
- **Magnetic resonance imaging MRI/MRA/DW Images.**
- **Carotid ultrasound:** shows buildup of fatty deposits called plaques and blood flow in the carotid arteries.
- **Echocardiogram:** can find a source of clots in the heart that may have traveled to the brain and caused a stroke.
- **In cases of suspected arterial dissection, CTA or MRA with fat-suppressed imaging (“fat sats”) to evaluate the cervical carotid and vertebral arteries should be obtained.**
- Intensive investigation for coagulopathy may be required for some patients.



FIGURE 2-4. MRA of the circle of Willis. MRA, magnetic resonance angiography.



Management of Ischemic Strokes

- Blood glucose, hydration, oxygen saturation and temperature should be maintained within normal limits.
- Blood pressure should not be lowered in the acute phase unless there are complications such as Hypertensive Encephalopathy.
- Aspirin 300mg orally or rectally should be given as soon as possible if a hemorrhagic stroke has been excluded.
- Thrombolysis with alteplase should only be given if:
 - it is administered within 4.5 hours of onset of stroke symptoms.
 - Brain hemorrhage has been definitively excluded (Imaging has been performed)
- Patients with acute ischemic stroke otherwise eligible for treatment with thrombolysis should have their blood pressure reduced to below 185/110 mmHg before treatment.

Table 3.5.1 Eligibility criteria for extending thrombolysis to 4.5-9 hours and wake-up stroke

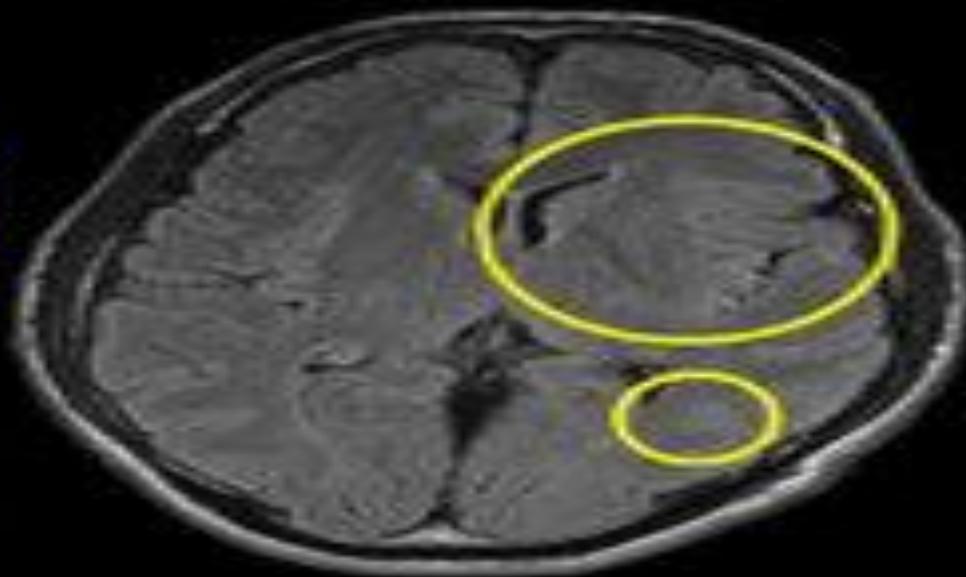
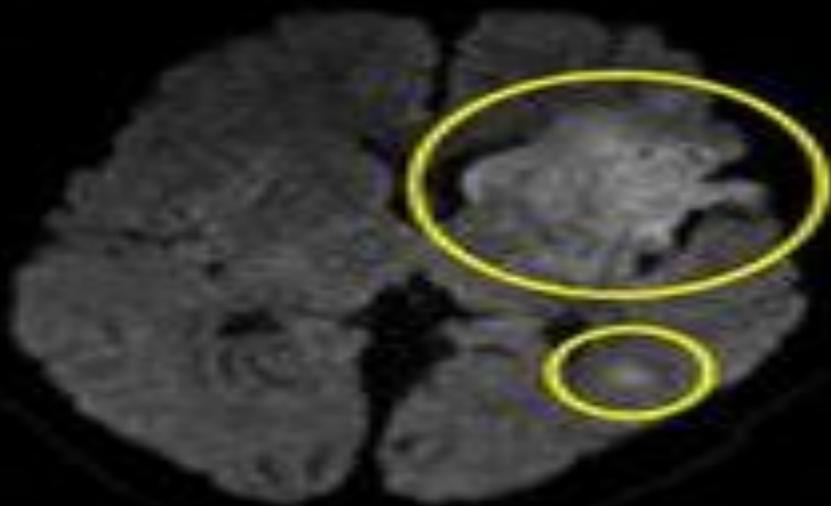
	Time window	Imaging	Imaging criteria
Wake-up stroke	>4.5 hours from last seen well, no upper limit	MRI DWI-FLAIR mismatch	DWI lesion and no FLAIR lesion
Wake-up stroke or unknown onset time	>4.5 hours from last seen well, and within 9 hours of the midpoint of sleep. The midpoint of sleep is the time halfway between going to bed and waking up	CT or MRI core-perfusion mismatch	Suggested: mismatch ratio greater than 1.2, a mismatch volume greater than 10 mL, and an ischaemic core volume <70 mL
Known onset time	4.5-9 hours	CT or MRI core-perfusion mismatch	Suggested: mismatch ratio greater than 1.2, a mismatch volume greater than 10 mL, and an ischaemic core volume <70 mL

[2023]

DWI

FLAIR

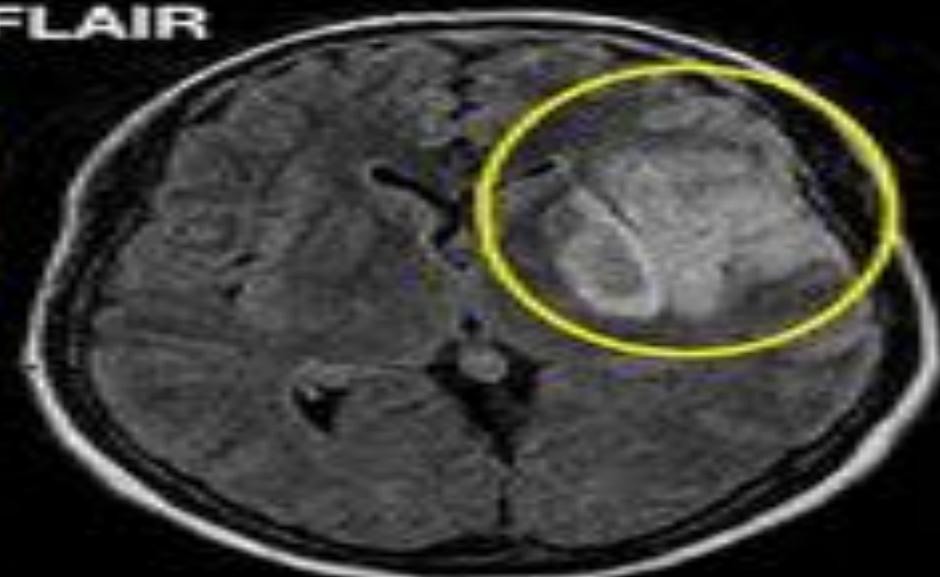
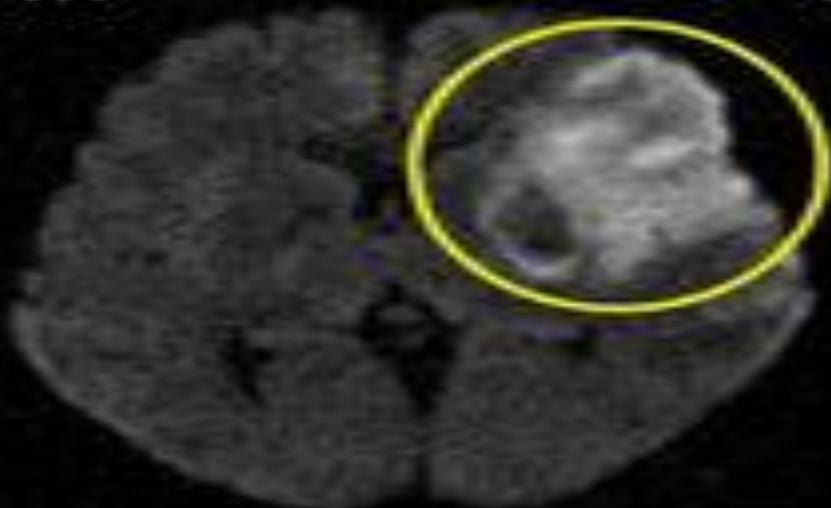
You can Give
Thrombolytics



DWI

FLAIR

You can not Give
Thrombolytics



Absolute Contraindications for thrombolytics:

- Symptoms suggestive of SAH even if CT scan of brain is normal
- Large or ruptured aneurysm > 10 mm
- Use of therapeutic dose of LMWH in the last 24 hours
- Associated or suspected aortic arch dissection
- CT hypodensity $> 1/3$ MCA territory
- Major surgery in the last 14 days
- Moyamoya disease

Absolute contraindications

- Prior intracranial hemorrhage
- Known structural cerebral vascular lesion
- Known malignant intracranial neoplasm
- Ischemic stroke within 3 months (excluding stroke within 3 hours*)
- Suspected aortic dissection
- Active bleeding or bleeding diathesis (excluding menses)
- Significant closed-head trauma or facial trauma within 3 months

Relative contraindications

- History of chronic, severe, poorly controlled hypertension
- Severe uncontrolled hypertension on presentation (SBP >180 mmHg or DBP >110 mmHg)
- History of ischemic stroke >3 months prior
- Traumatic or prolonged (>10 minutes) CPR or major surgery <3 weeks
- Recent (within 2 to 4 weeks) internal bleeding
- Noncompressible vascular punctures
- Recent invasive procedure
- For streptokinase/anistreplase – Prior exposure (>5 days ago) or prior allergic reaction to these agents
- Pregnancy
- Active peptic ulcer
- Pericarditis or pericardial fluid
- Current use of anticoagulant (eg, warfarin sodium) that has produced an elevated INR >1.7 or PT >15 seconds
- Age >75 years
- Diabetic retinopathy

Thrombectomy for acute ischemic strokes

- NICE recommend a pre-stroke functional status of less than 3 on the modified Rankin scale and a score of more than 5 on the National Institutes of Health Stroke Scale (NIHSS).
- Offer thrombectomy as soon as possible and **within 6 hours** of symptom onset, together with intravenous thrombolysis (if within 4.5 hours), to people who have: acute ischemic stroke and confirmed occlusion of the **proximal anterior circulation** demonstrated by computed tomographic angiography (CTA) or magnetic resonance angiography (MRA).
- Offer thrombectomy as soon as possible to people who were last known to be well **between 6 hours and 24 hours** previously (including wake-up strokes): confirmed occlusion of the **proximal anterior circulation** demonstrated by CTA or MRA and if there is the potential to **salvage brain tissue**, as shown by imaging such as CT perfusion or diffusion-weighted MRI sequences showing limited infarct core volume.
- Consider thrombectomy together with intravenous thrombolysis (if within 4.5 hours) as soon as possible for people last known to be well up to **24 hours** previously (including wake-up strokes): who have acute ischemic stroke and confirmed occlusion of the **proximal posterior circulation** (that is, basilar or posterior cerebral artery) demonstrated by CTA or MRA and if there is the potential to **salvage brain tissue**, as shown by imaging such as CT perfusion or diffusion-weighted MRI sequences showing limited infarct core volume.

Secondary Prevention of ischemic strokes

- With regards to carotid artery endarterectomy: It is recommended if patient has suffered stroke or TIA in the carotid territory and are not severely disabled. It should only be considered if **carotid stenosis > 70% according ECST** (European Carotid Surgery Trialists' Collaborative Group) criteria **or > 50% according to NASCET** (North American Symptomatic Carotid Endarterectomy Trial) criteria.
- Patients with high-risk TIA and low NIHSS stroke should be treated with clopidogrel load in the first 24 hours followed by **DAPT for 21 days**.
- Statin should be prescribed for patients who have high lipid profile.
- Patients with ischemic stroke or transient ischemic attack and **atrial fibrillation should receive oral anticoagulant therapy for secondary stroke prevention**.
- In patients with stroke due to **intracranial atherosclerosis**, **dual antiplatelet therapy with aspirin and clopidogrel** is favored.

Hemorrhagic Strokes

Bleeding inside the skull can be divided into **subarachnoid, intracerebral, epidural and subdural hemorrhages.**

The latter 2 types of hemorrhage are almost always traumatic.

Intracerebral hemorrhage (ICH) and subarachnoid hemorrhage (SAH) have different causes, clinical findings and management.

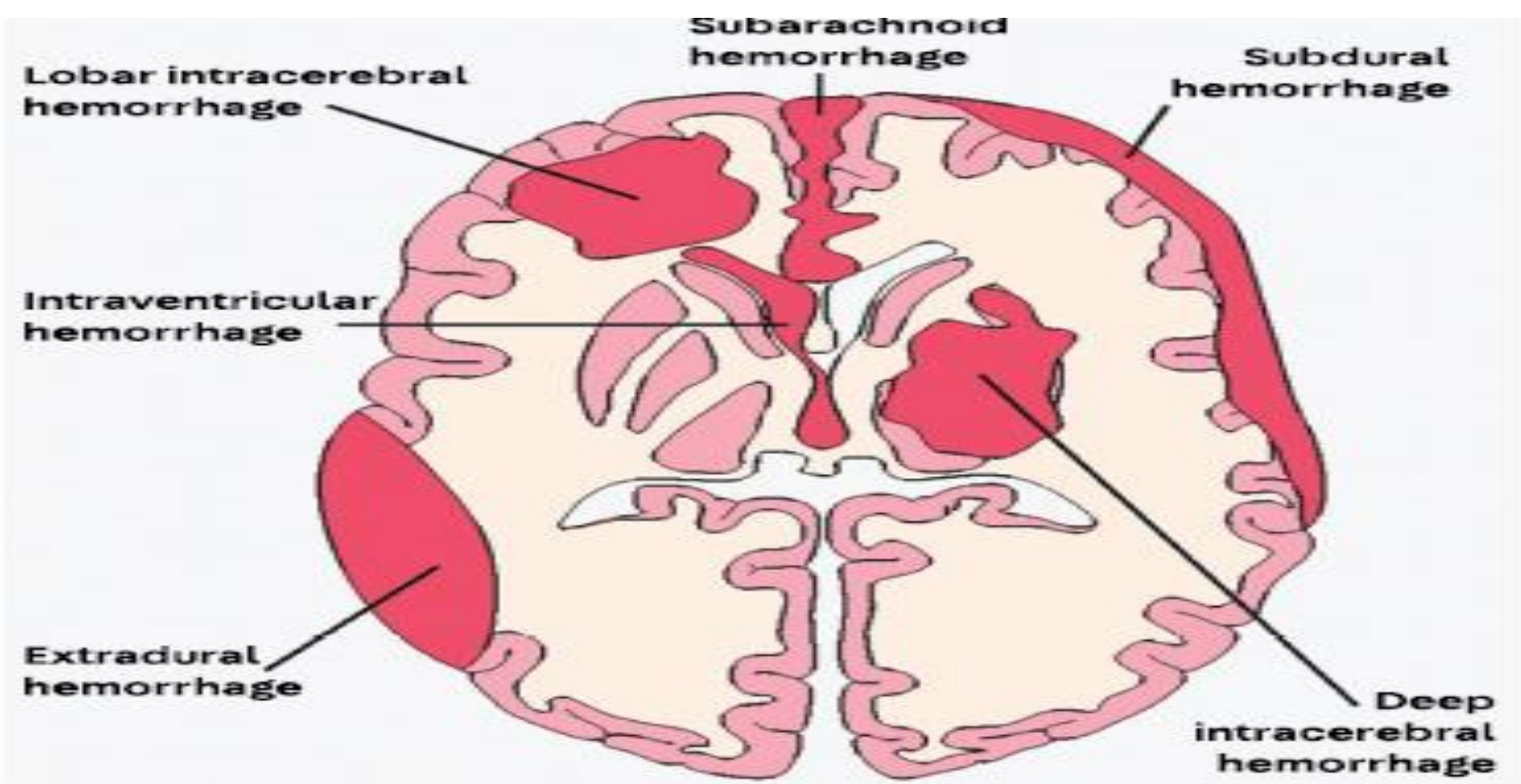


FIGURE 3-2

Locations and types of intracranial hemorrhage that may be seen on noncontrast CT.

- **Subarachnoid hemorrhage (SAH)**

- SAH is often due to traumatic injury. More serious, though, is SAH caused by bleeding from an aneurysm located along the circle of Willis.
- When blood under arterial pressure is suddenly released into the space around the brain, patients develop sudden-onset, severe headache.
- Often ,they vomit and cease what they are doing at the time of the hemorrhage.
- When the intracranial pressure increases rapidly or the insulae are affected, coma or death may ensue.

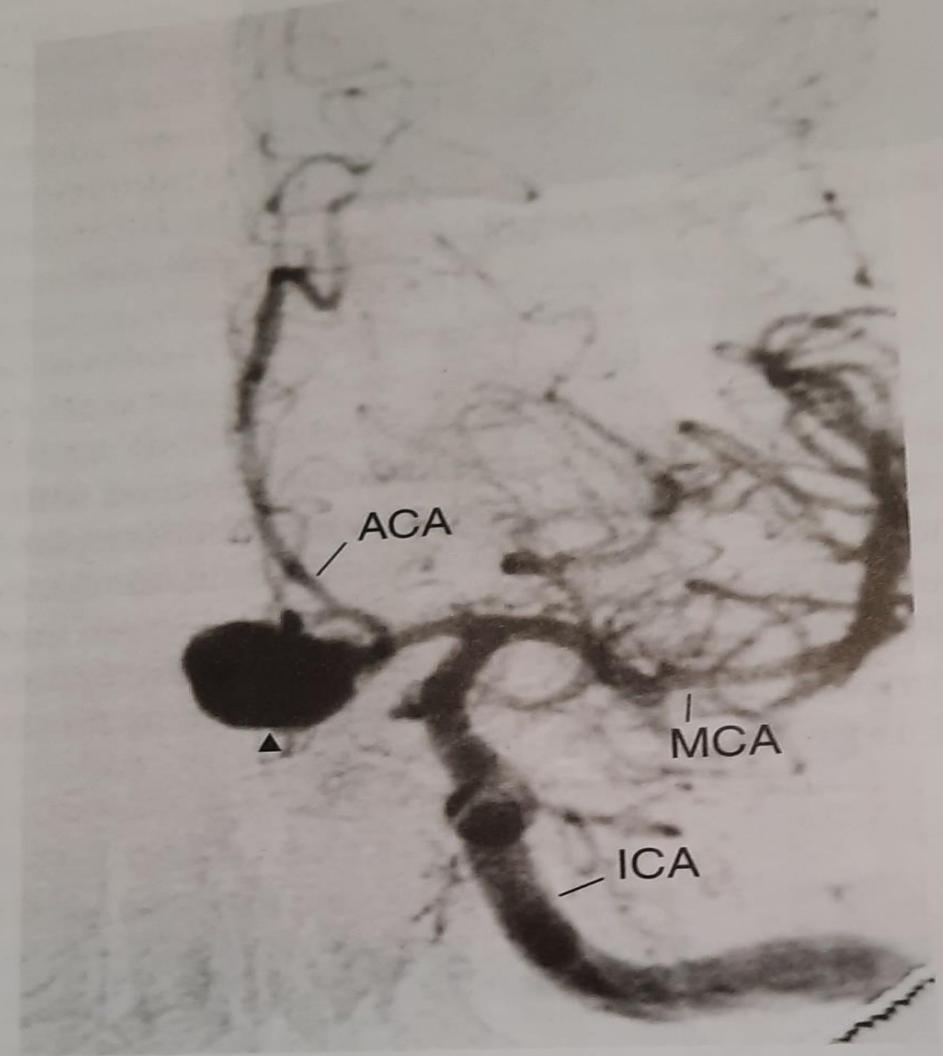
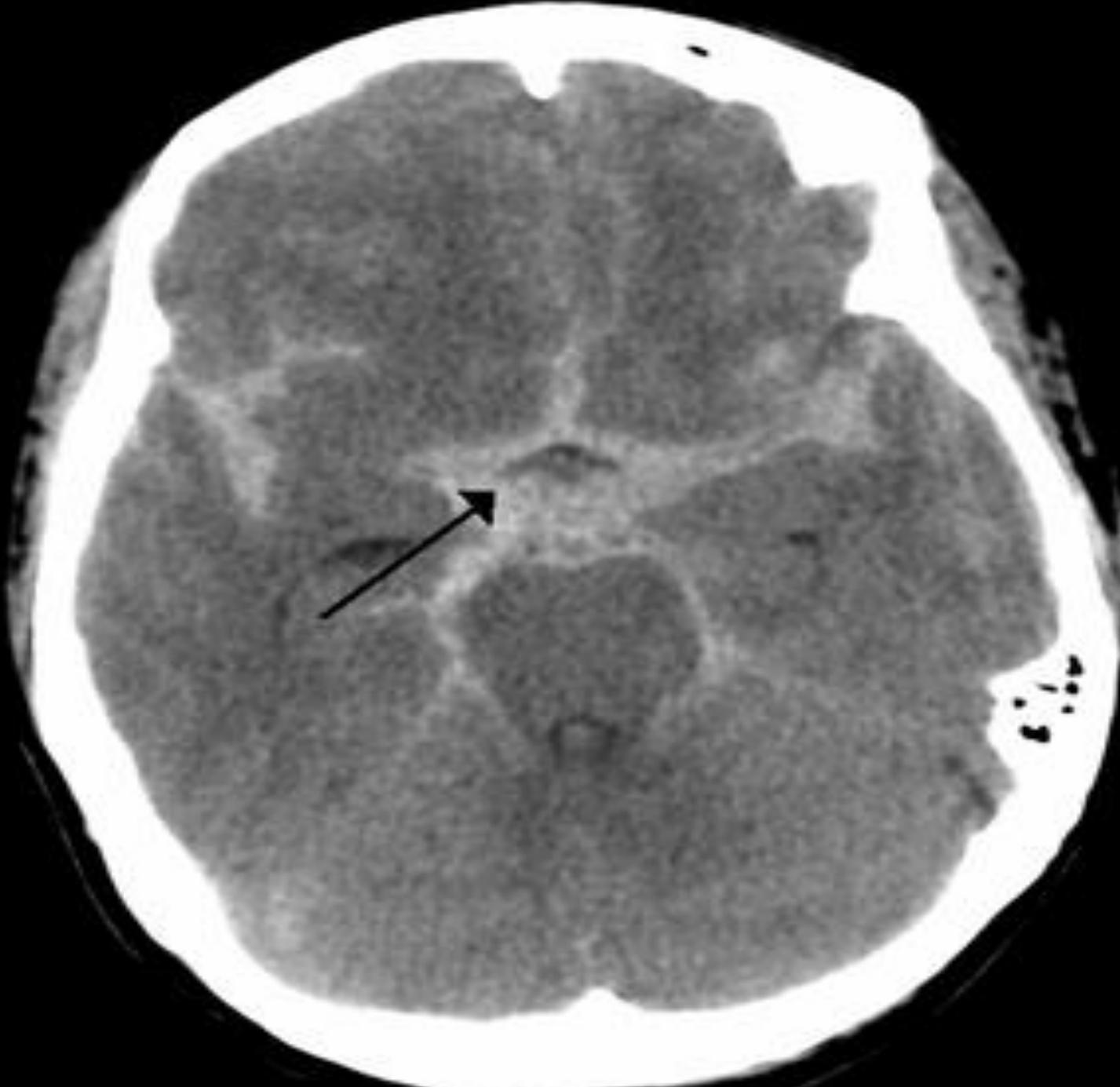


FIGURE 2-3. Conventional cerebral angiogram demonstrating aneurysm of the right middle cerebral artery (*arrow*). ACA, anterior cerebral artery; ICA, internal carotid artery; MCA, middle cerebral artery. (Reproduced with permission from Yochum TR, Rowe LJ. *Yochum and Rowe's Essentials of Skeletal Radiology*. 3rd ed. Philadelphia, PA: Lippincott Williams & Wilkins; 2004.)

Treatment is aimed at preventing the rebleeding and vasoconstriction that often follow SAH.

Aneurysms can be clipped surgically or “coiled” by interventional techniques.

The calcium-channel blocker nimodipine is used to minimize vasoconstriction and delayed brain ischemia.



- Intracerebral hemorrhage
- ICH is bleeding directly into brain parenchyma.

- The earliest symptoms are headache and neurologic signs referable to the region in which the bleeding occurs.

- **Hypertension (leading to Charcot-Bouchard microaneurysms) is the most common cause of ICH.**

- The most common locations for hypertensive ICH are the **basal ganglia-internal capsule, caudate nucleus, thalamus, pons and cerebellum.**

13-60-M

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135.50mm
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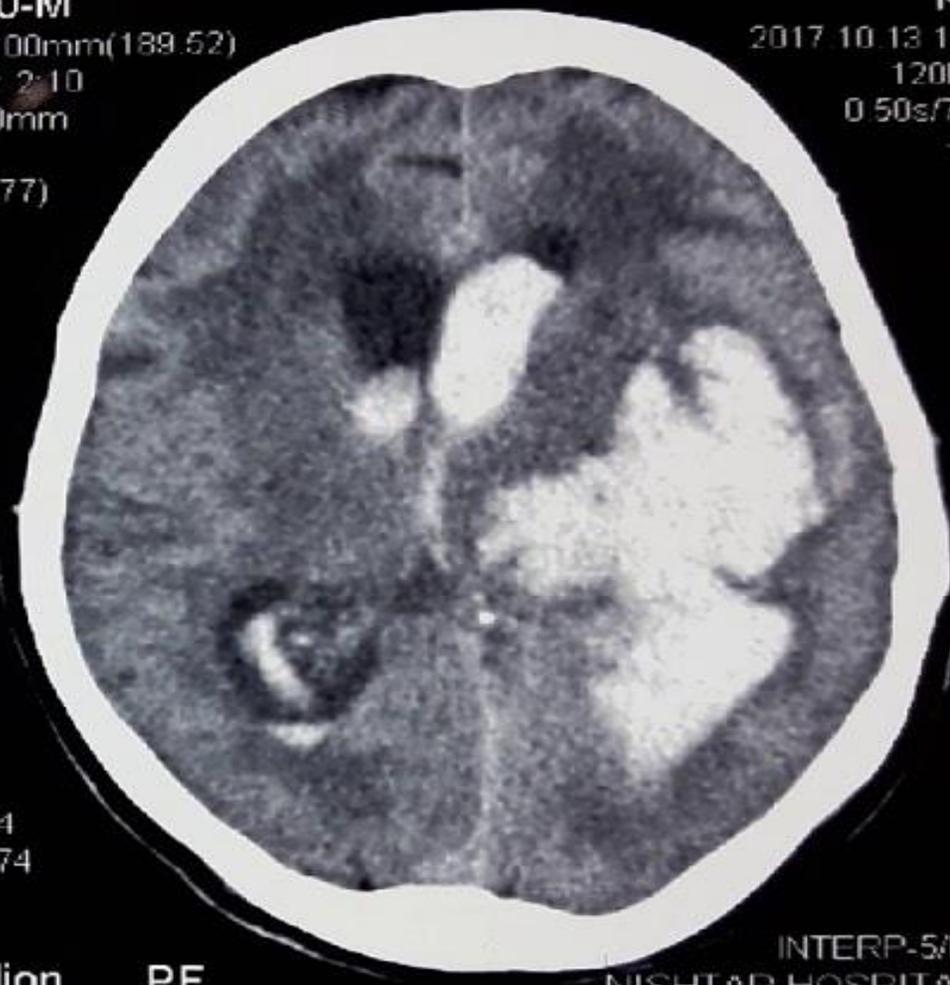


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WL=24
WW=74

Aquilion PF

KHAN BB 13-60-M

2017.10.13.16:44:36.532 M:250.00mm(189.52)
120kV/150mAs 48527.2:10
0.50s/7mm/1.0x16 142.50mm
+15.00mm/r 5.00
HP15.0 (256,277)



R
WL=24
WW=74

Aquilion PF

KHAN BB 13

2017.10.13.16:44:36.766 M:
120kV/150mAs 48
0.50s/7mm/1.0x16
+15.00mm/r 5.0
HP15.0 (2

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Aquilion PF

INTERP-5/FC27/ORG//

NISHTAR HOSPITAL MULTAN

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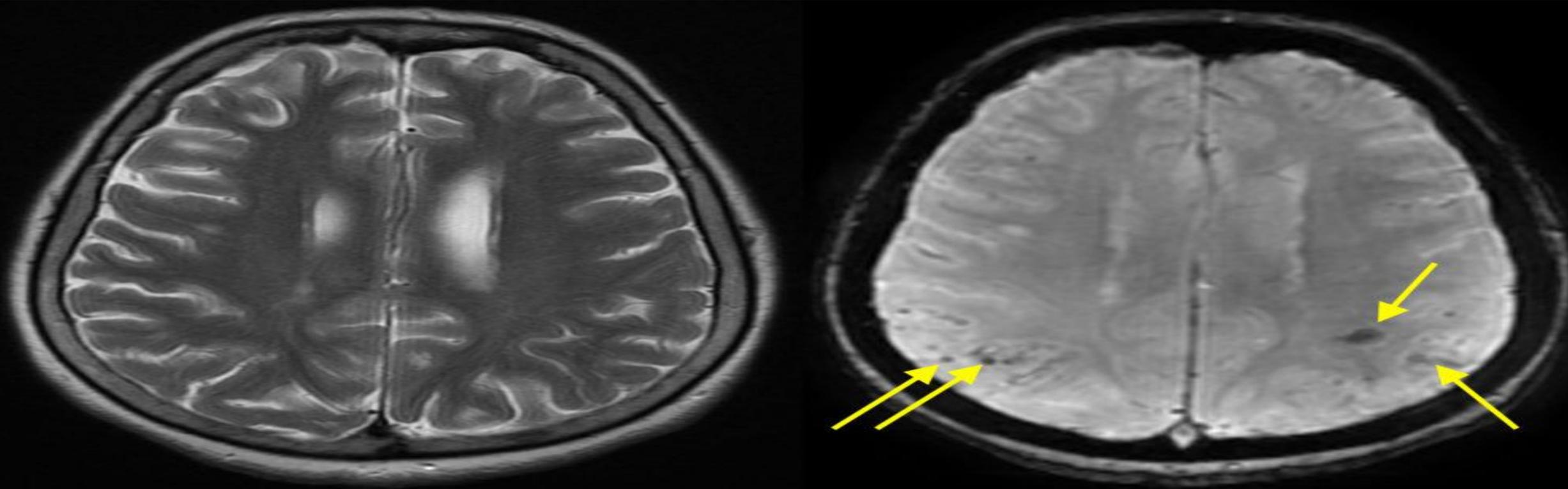
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NISHTAR HOSPITAL MULTAN A

Cerebral amyloid angiopathy is a cause of ICH that is more frequent in the elderly. and preferentially affects the parietal and occipital lobes.

Trauma, vascular malformations and bleeding diatheses (especially with patients who are taking anticoagulants) are other common causes.

ICH is often a devastating condition , and large hemorrhages are associated with high mortality rates.



Treatment involves correcting any coagulopathy.

In certain situations(particularly cerebellar hemorrhages) ,
surgical decompression is necessary.

Management of risk factors for hemorrhage, specifically hypertension, is necessary to prevent recurrence.

Vascular malformations:

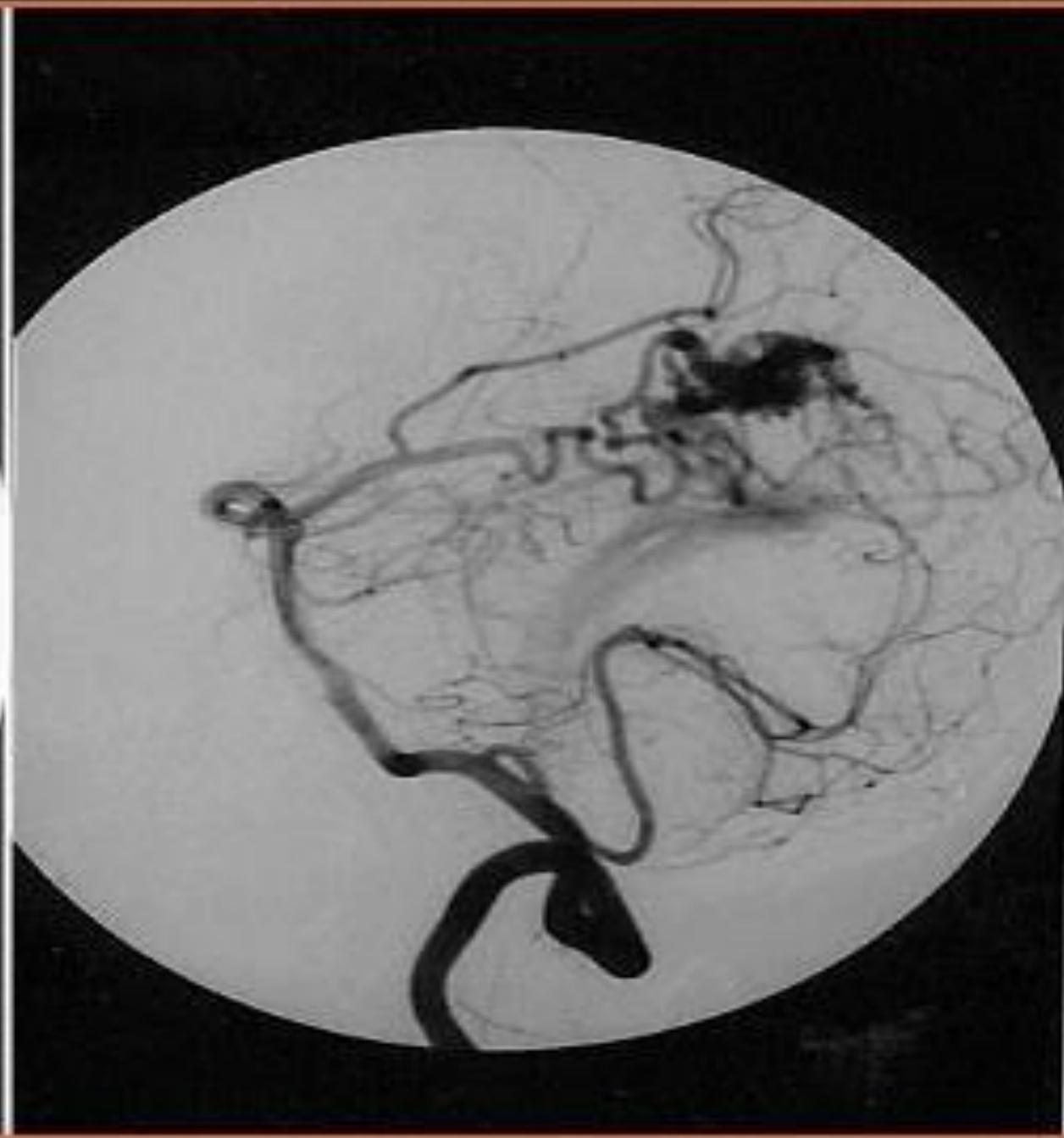
Variety of congenital and acquired vascular anomalies that have the potential to bleed, either within the brain (ICH) or around it.

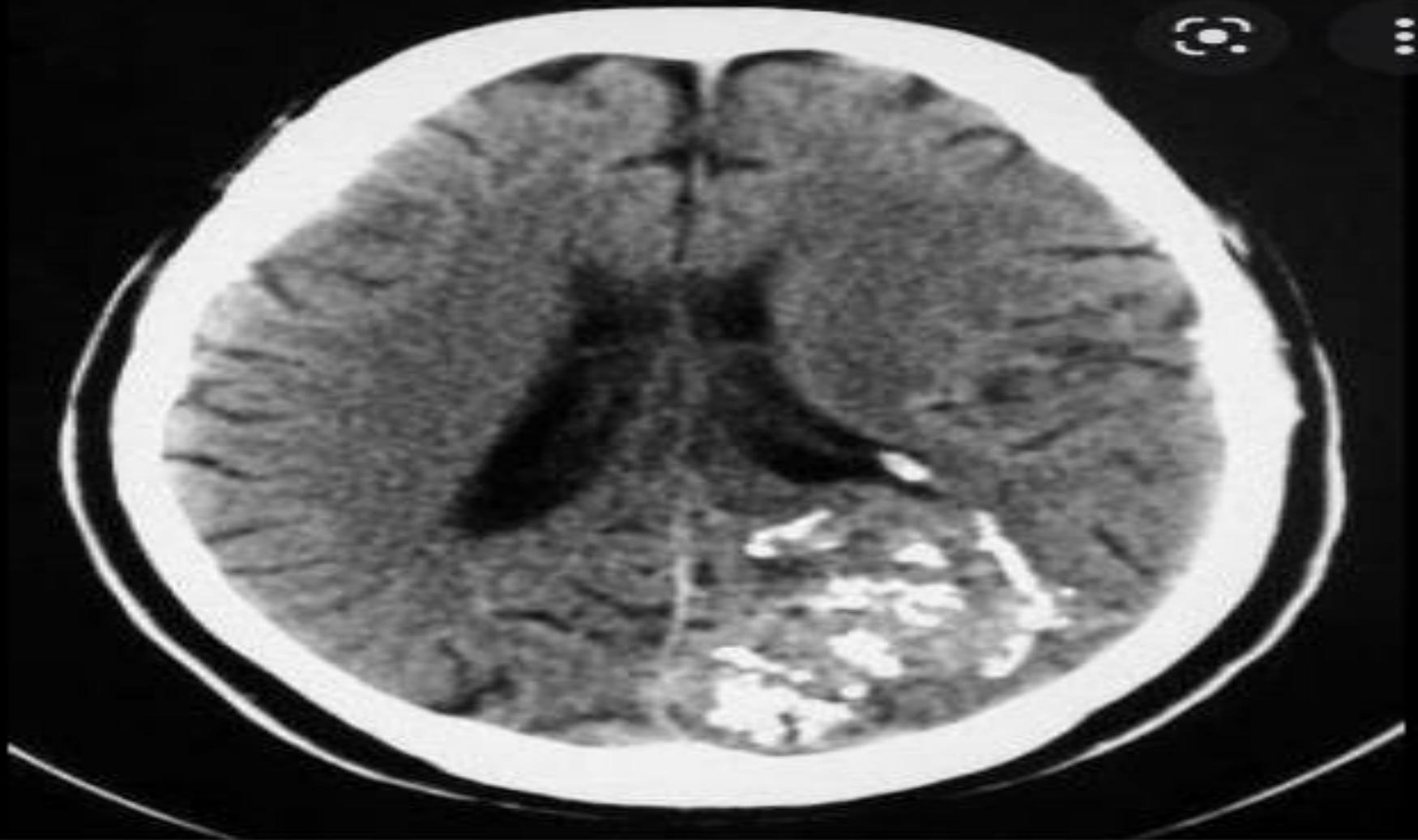
* Arteriovenous malformations (AVMs) contain arteries that empty into arterialized veins.

These lesions contain no recognizable normal capillary bed, but abnormal gliotic parenchyma can be found between the component vessels.

In addition to causing ICH , AVMs may result in seizures.

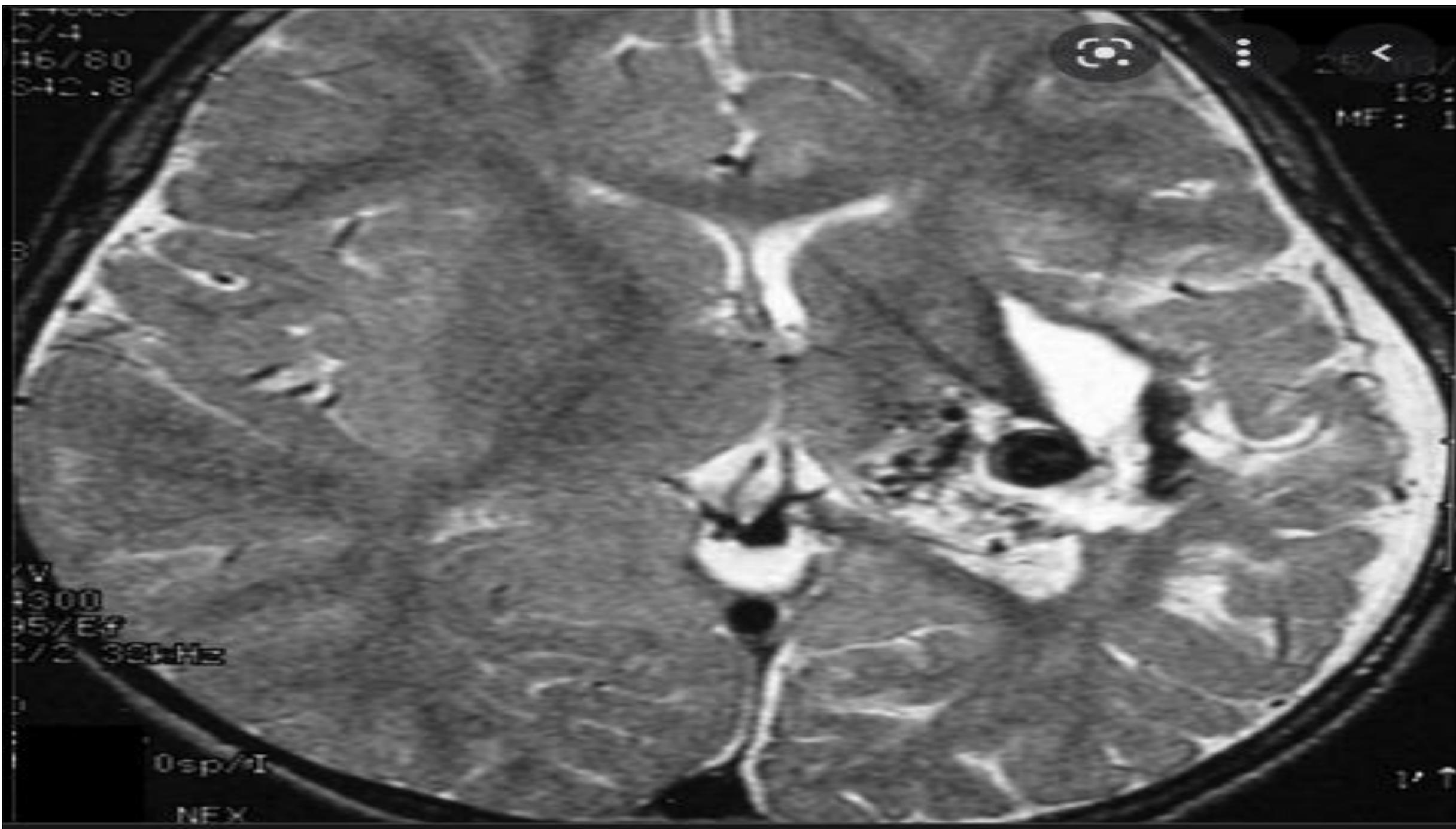
AVMs may be treated with embolization or surgical resection.





 Medscape Reference

Arteriovenous Malformation Brain Imaging: Practice Essentials, Ultrasonography, Computed Tomography



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Cavernous angiomas: consist of a relatively compact mass of **sinusoidal vessels close together**, without intervening brain parenchyma.

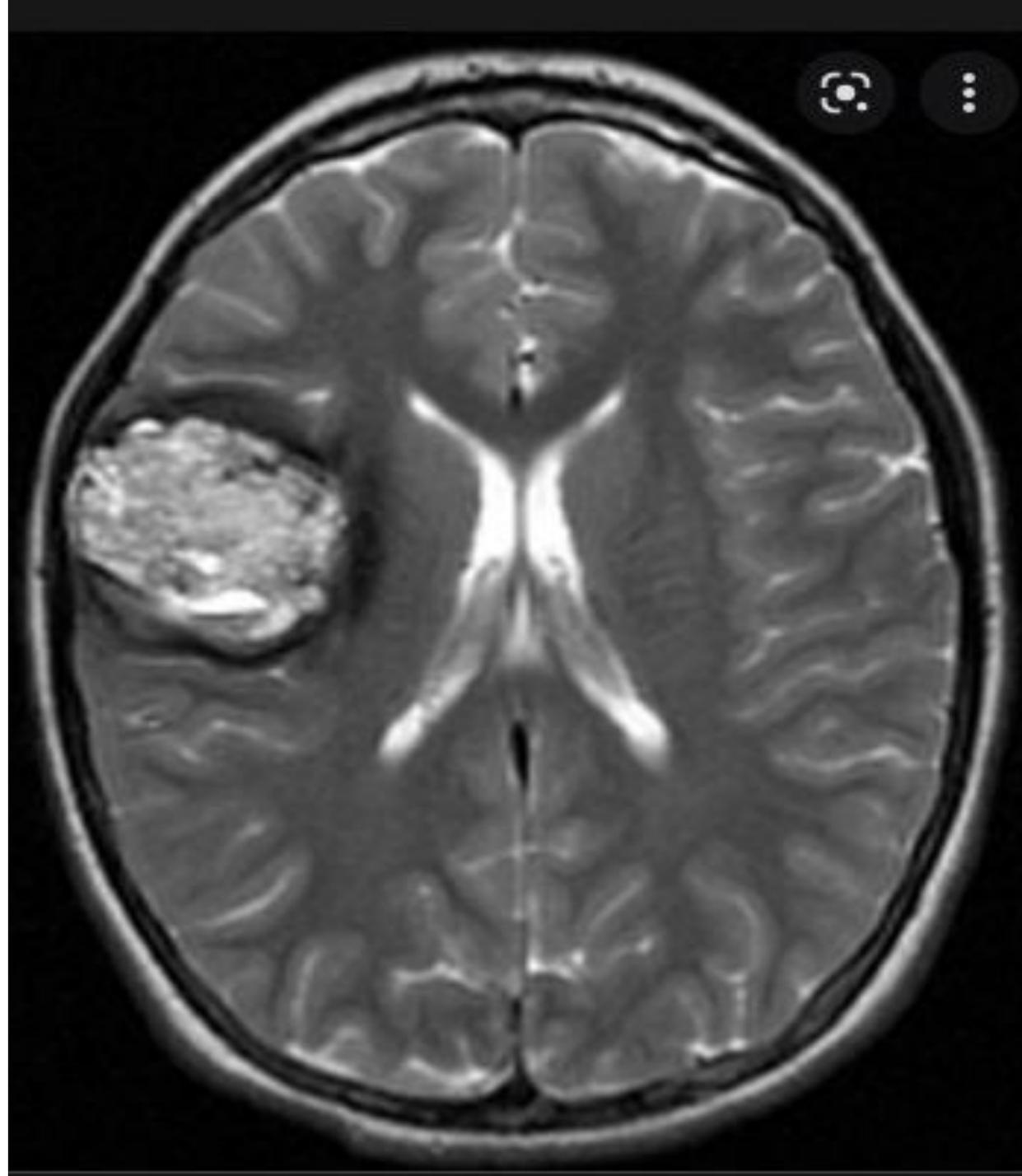
The lesions are well **encapsulated**.

Cavernous angiomas bleed or lead to seizures, occasionally, but are not threatening as AVMs are.

They may be followed with serial neuroimaging studies.

Surgery is required **rarely**.

They may require **antiseizure drug treatment** if recurrent seizures develop.

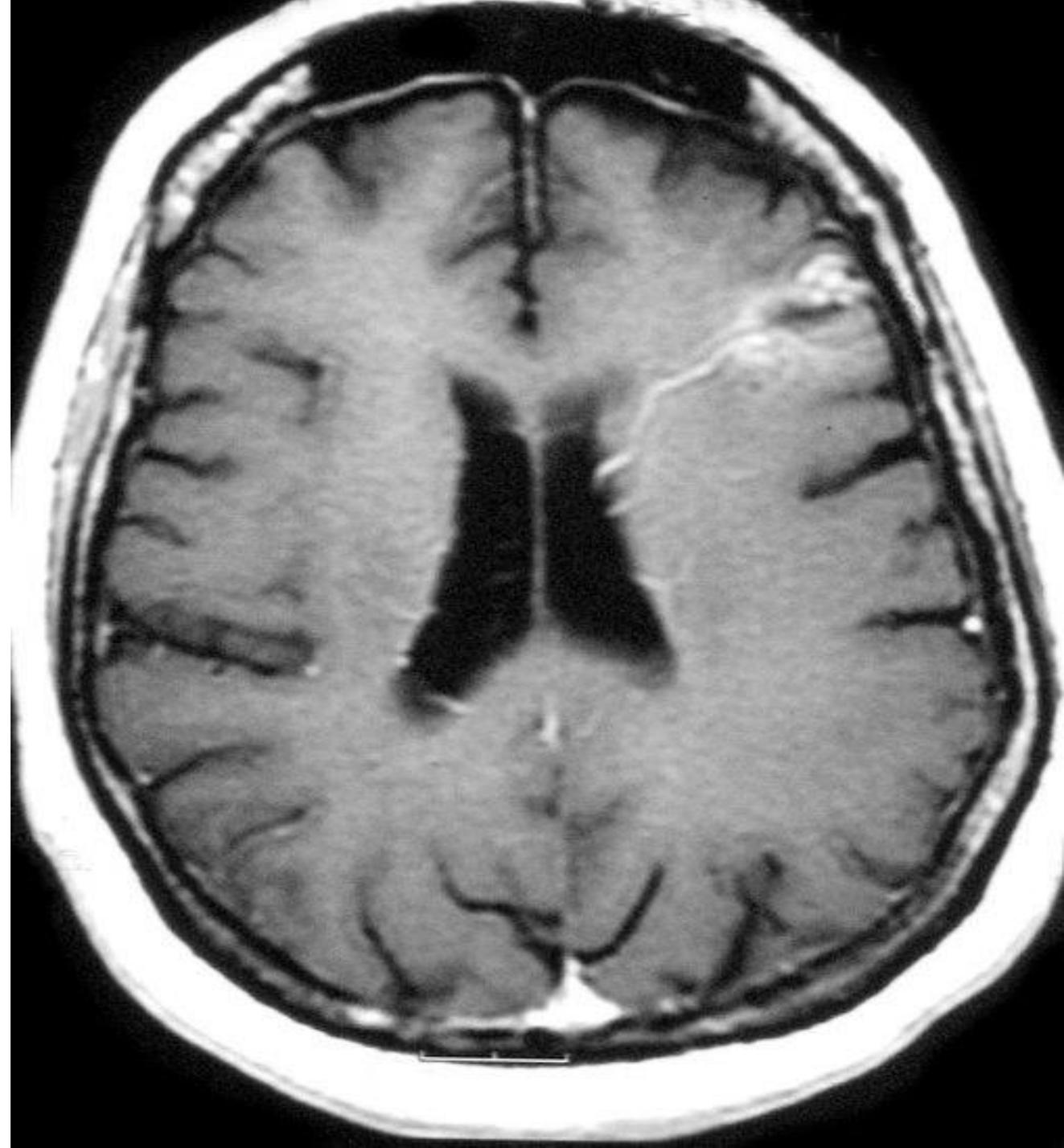


Developmental venous anomalies (DVAs): are composed of anomalous veins usually separated by morphologically normal brain parenchyma are the most common vascular malformations of the brain.

They seldom hemorrhage and are generally not treated surgically or followed with serial neuroimaging studies.

Telangiectasias are dilated capillaries with intervening brain parenchyma.

They are incidental findings and do not require treatment.



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