MEDICINE

432 Team

46 Cerebrovascular Disease

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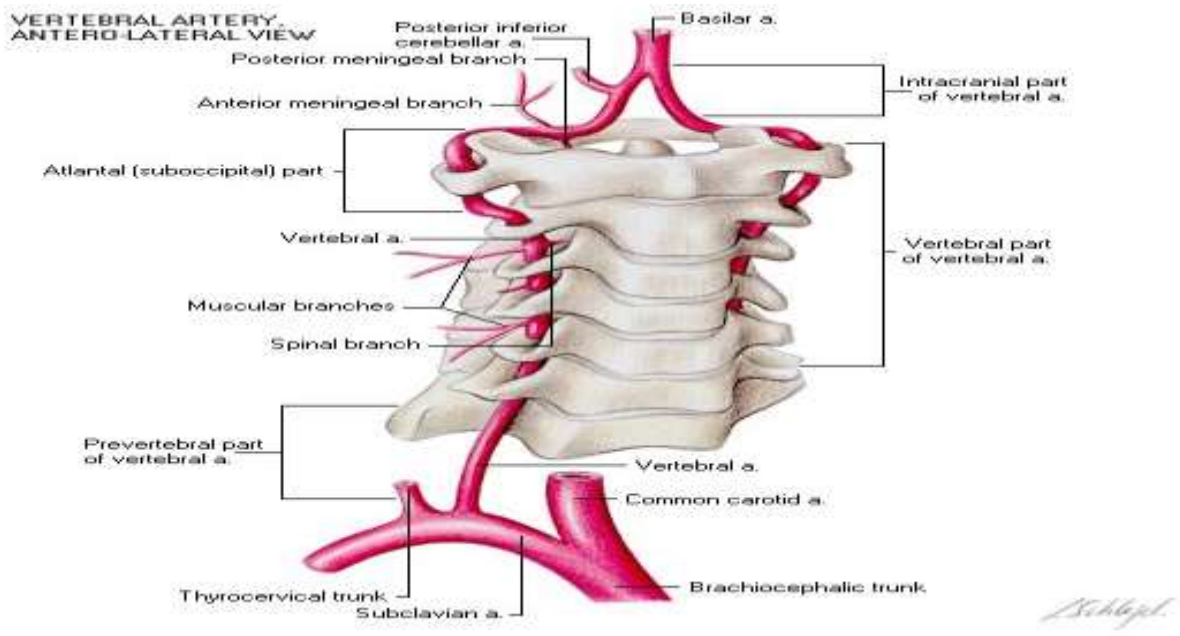
COLOR GUIDE: • Females' Notes • Males' Notes • Important • Additional

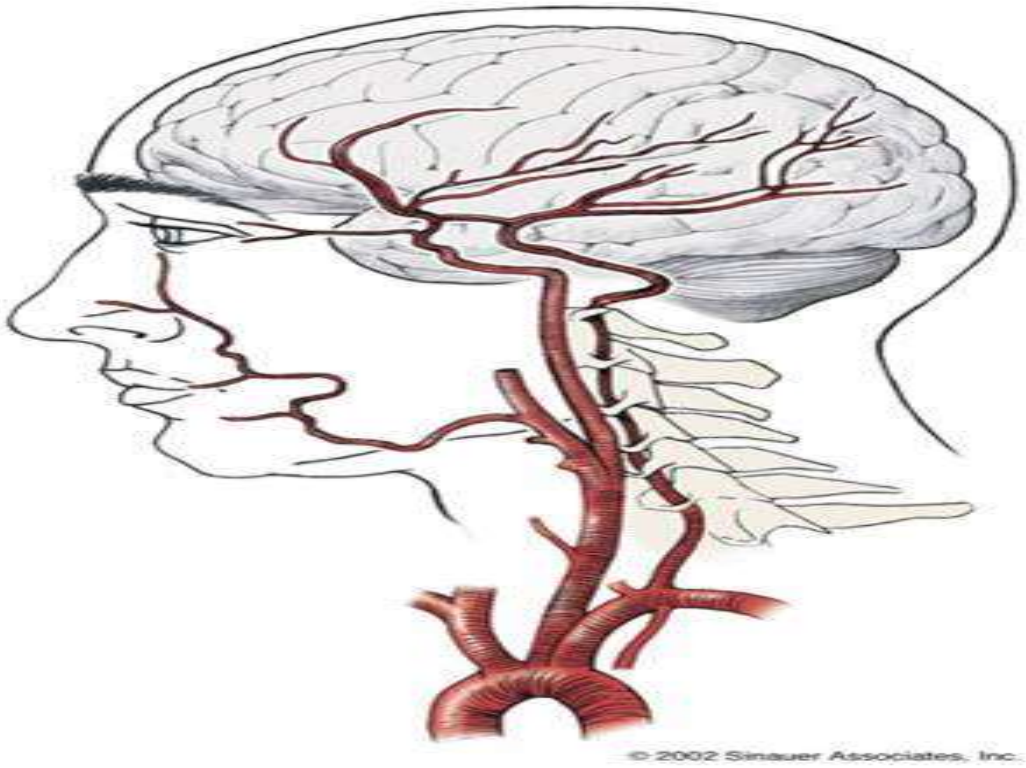
Objectives

Were not given ☹

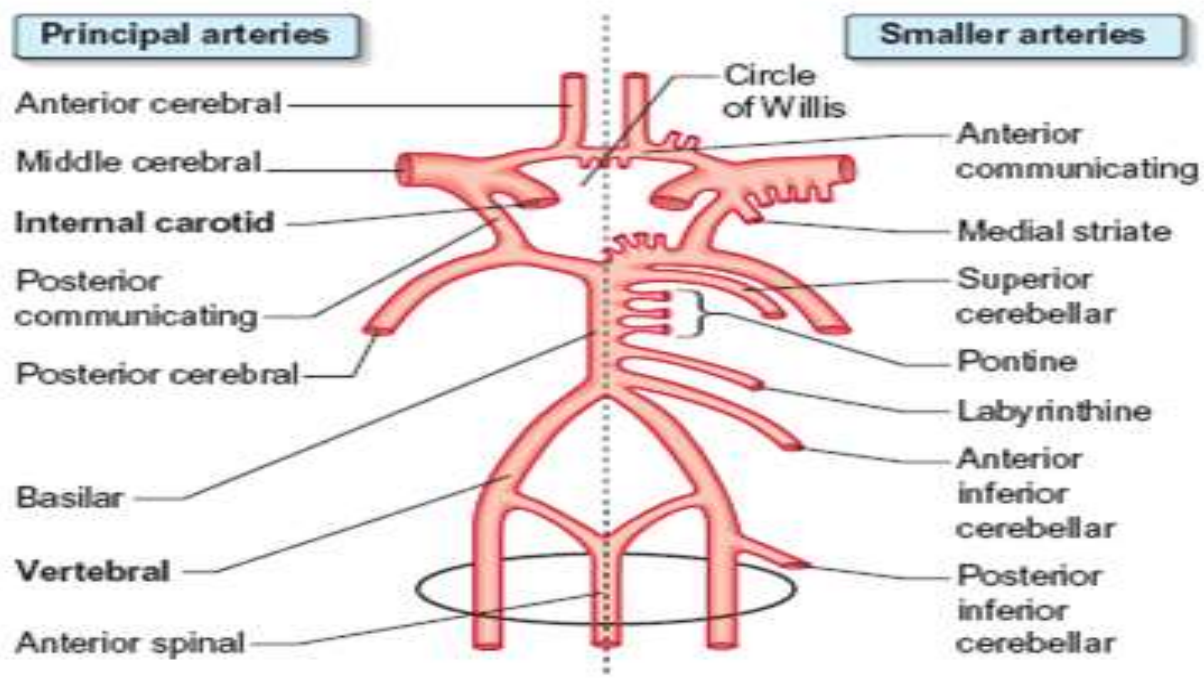
Anatomy:

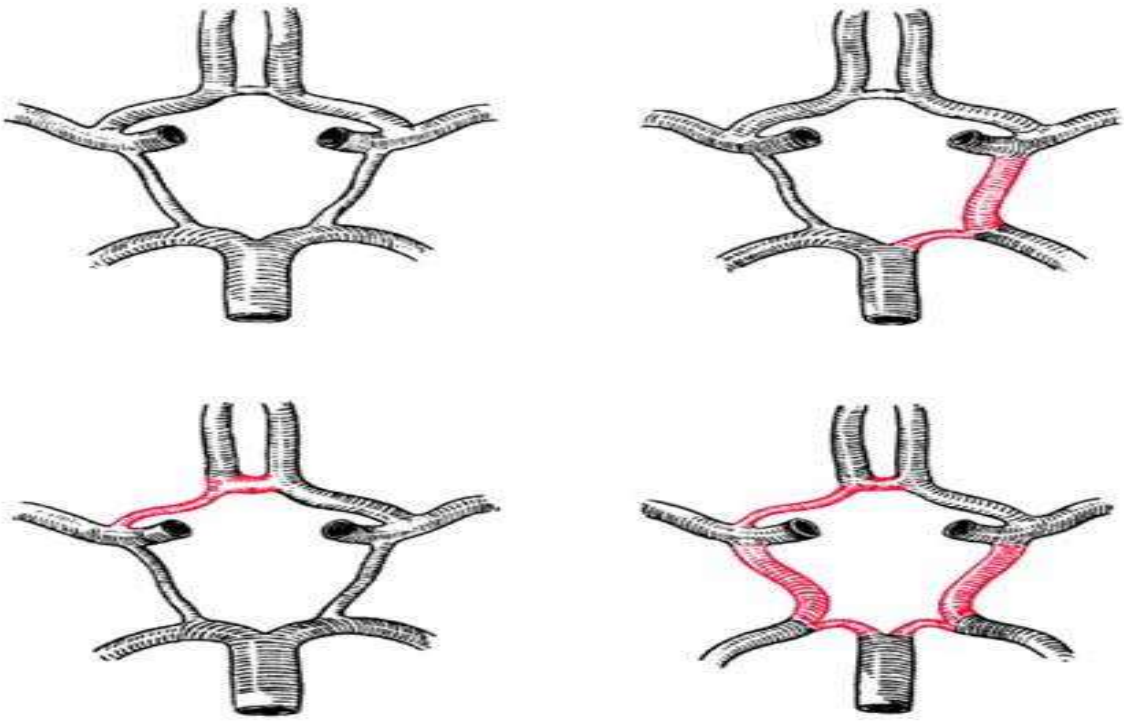
1- the carotids and the Vertebral arteries



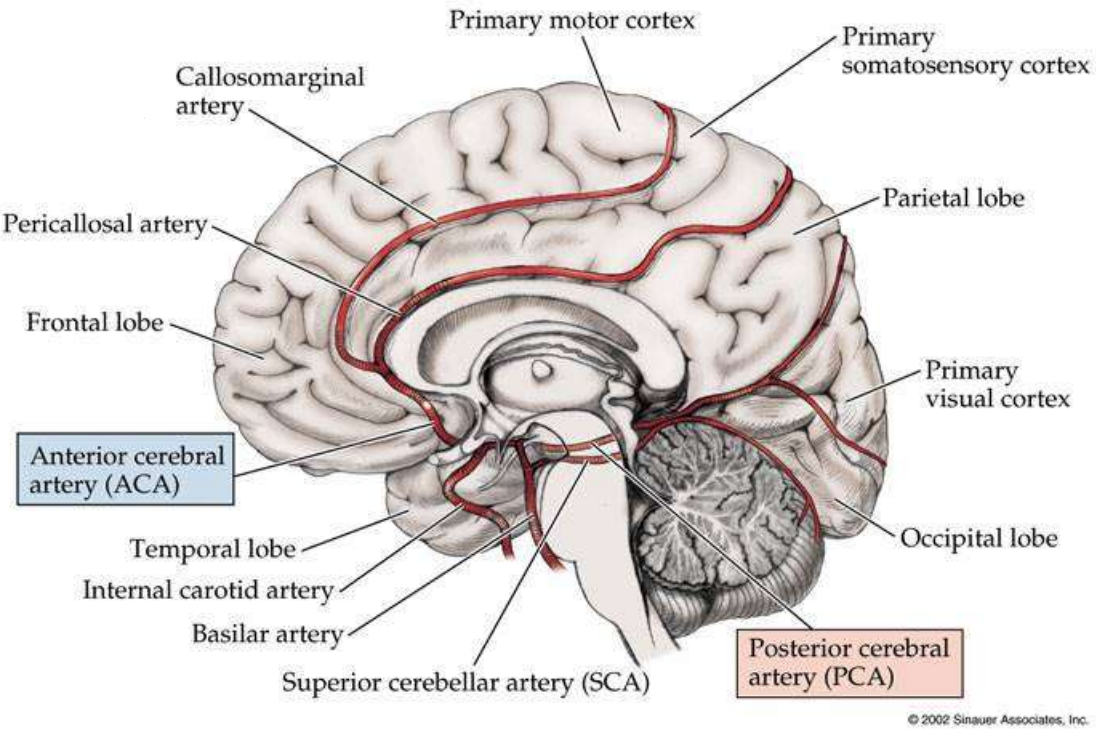


2- Intracranial cerebro-vascular system/COW variations:





3- ACA/PCA/MCA


Primary motor cortex
Primary somatosensory cortex
Callosomarginal artery
Parietal lobe
Pericallosal artery
Frontal lobe
Primary visual cortex
Anterior cerebral artery (ACA)
Occipital lobe
Temporal lobe
Internal carotid artery
Basilar artery
Posterior cerebral artery (PCA)
Superior cerebellar artery (SCA)
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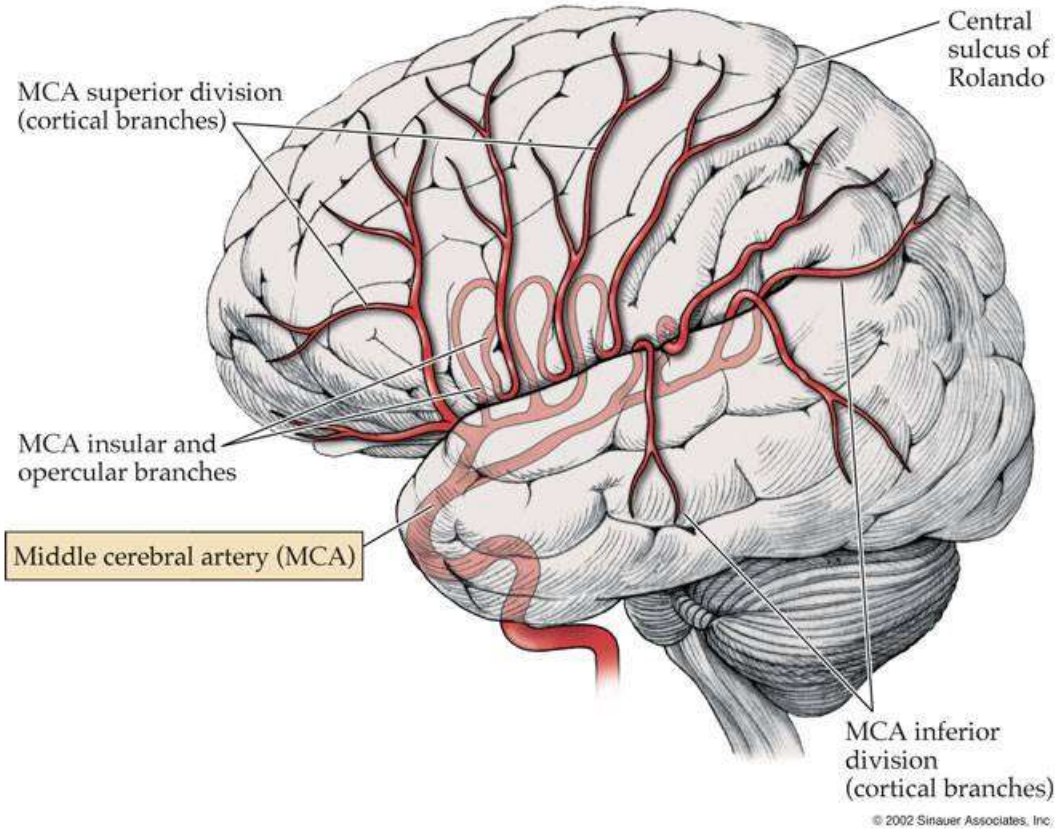
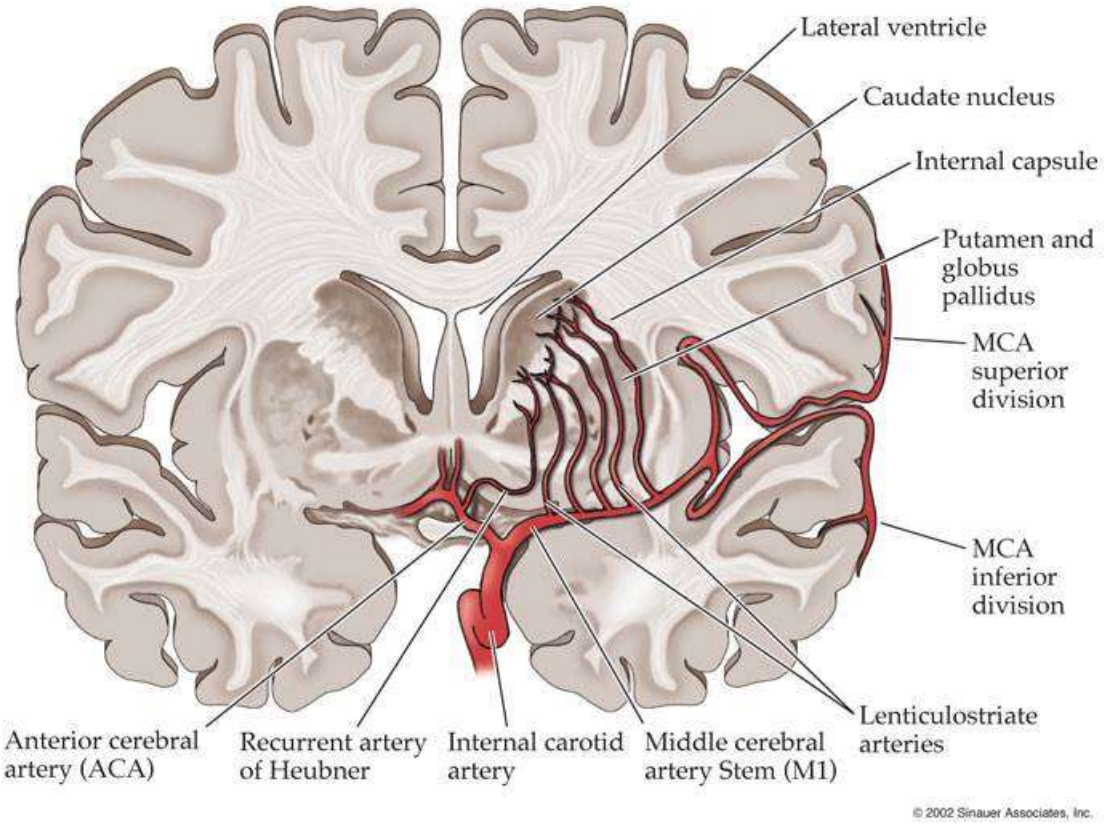
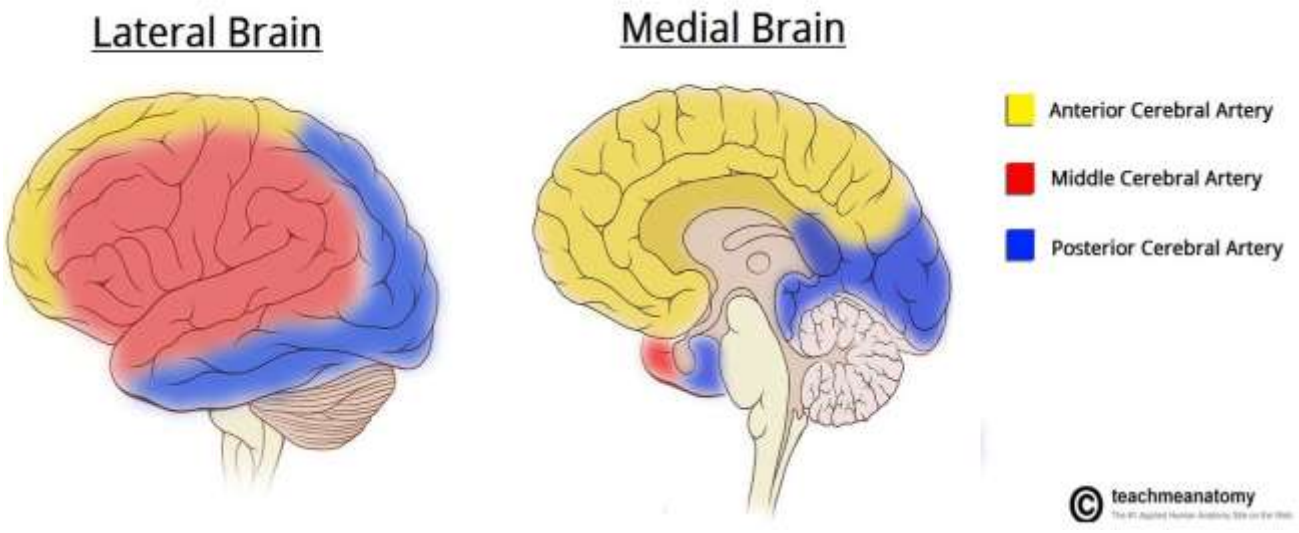

Central sulcus of Rolando
MCA superior division (cortical branches)
MCA insular and opercular branches
Middle cerebral artery (MCA)
MCA inferior division (cortical branches)
© 2002 Sinauer Associates, Inc.



4- Artery distribution:



Common acute stroke presentation based on arterial distribution:

1- ACA:

Sudden contralateral leg weakness and personality changes.

2- MCA:

1. Contralateral weakness and sensory loss of face and arm and leg
2. Cortical sensory loss
3. May have contralateral homonymous hemianopia or quadrantanopia
4. If left hemisphere: aphasia
5. If right hemisphere: hemispatial neglect (Rt MCA stroke is more difficult in rehabilitation than the left)
6. Eye deviation towards the side of the lesion and away from the weak side

3- PCA:

1. Contralateral hemianopia or quadrantanopia
2. Midbrain findings: CN III and IV palsy/pupillary changes, hemiparesis
3. Thalamic findings: sensory loss, amnesia, decreased level of consciousness
4. if bilateral: cortical blindness or prosopagnosia

4- basilar artery (locked-in syndrome):

1- Reduced level of consciousness/coma with weakness and numbness on both sides of the body, think of basilar stroke.
2- quadriparesis
3- dysarthria
4- impaired eye movements

Note: this is commonly misdiagnosed as seizure, always think about it!

5- (lateral medullary or Wallenburg syndrome):

Lateral medulla doesn't have motor fibers

Ipsilateral Horner's with contralateral limp numbness/sensory.

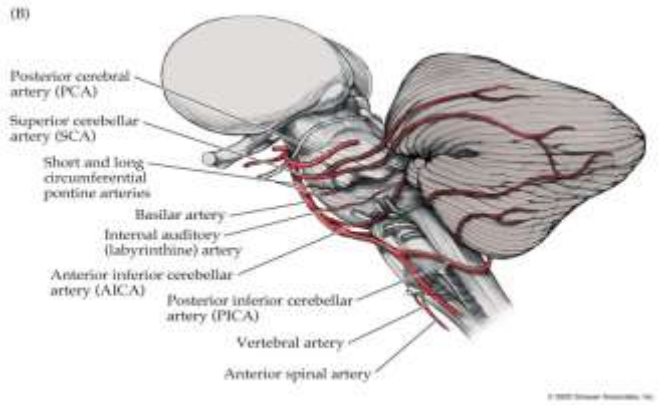
6- Lacunar:

1- pure motor hemiparesis: posterior limb of internal capsule: contralateral arm, leg, and face
2-pure sensory loss: thalamic: hemisensory loss
3-ataxic hemiparesis: ipsilateral ataxia and leg paresis
4-dysarthria-clumsy hand syndrome: dysarthria, facial weakness, dysphagia, mild hand weakness and clumsiness

7- Vertbero-Basilar System:

- Vertigo
- Diplopia/ dysconjugate gaze, ocular palsy homonymous hemianopsia
- Sensorimotor deficits - Ipsilateral face and contralateral limbs (crossing sign)
- Dysarthria
- Ataxia
- Sudden LOC

It is usually Multi-domain, for example:
<u>vertigo, ataxia and weakness</u>



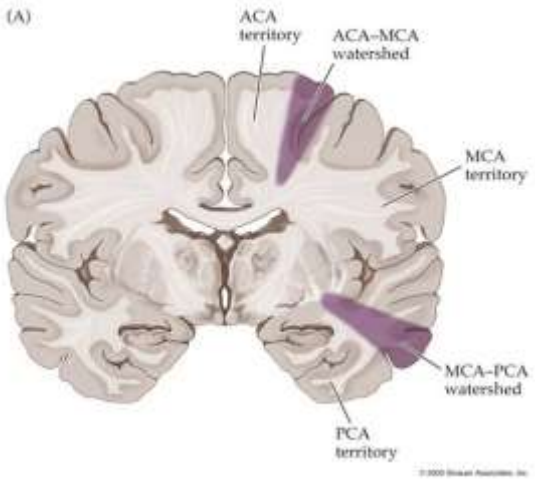
8- Watershed Areas: **<u>hypoperfusion.</u>**

When the BP is reduced, the first to be affected are the distal branches, blood flow is reduced in both regions and you get the stroke in the area between the two as shown in the pic below.

This is usually seen in patients undergoing open-heart surgery, where the BP suddenly drops, or in cases of Atrial or ventricular tachycardia.

An underlying carotid stenosis will increase the likelihood of WS.

Treatment is maintaining the BP



Stroke Risk Factors: very important!

Non-Modifiable:

- **Age, the most important overall**
- Male gender
- Family History
- Genetic causes
- Congenital abnormality like in heart or AVM in CNS
- Hyper-coagulopathies

Modifiable:

- **HTN, the most important modifiable**
- Diabetes
- Hyperlipidemia
- Atrial Fibrillation
- Carotid artery disease
- Physical inactivity
- Obstructive sleep apnea
- Smoking
- Substance abuse
- Medications: Oral Contraceptives.
- Dissection

Types of stroke:

Ischemic:

1- Thrombosis: the clot formed within the brain.

2- Embolism: the clot coming from other sites.

3- Lacunar: arterioles/small blood vessel occlusion.

4- Hypoperfusion: Watershed areas, related to hypotension.

Hemorrhagic: they usually co-exist

1- Epidural

2-Subdural

3-Subarachnoid

4-Intra-cerebral

5-Intra-ventricular

Venous:

1- Venous sinus thrombosis

2- Cortical vein thrombosis

Stroke Epidemiology:

- About 30-40K new cases annually in Saudi Arabia (estimation)
- Lacunar strokes makes near 50 % because of prevalent diabetes
- Increasing prevalence of stroke in young because of increasing HTN, diabetes, & substance abuse added to cardiogenic causes (MCC) and hypercoagulopathies
- Although stroke incidence is higher in men, women have equal life time risk because they live longer
- Stroke is a preventable & predictable disease ➔ never use the word: ACCIDENT

Note: Lacunar stroke number one cause is diabetes, while in large strokes cardiogenic/thrombophilia are more common.

HTN causes both (large and small vessel strokes).

Transient Ischemic Attack: (it is a Stroke with complete resolution)

A brief episode of neurological dysfunction caused by focal brain or retinal ischemia, with clinical symptoms typically lasting <1 h, and without evidence of acute infarction. (Normal imaging)

- 20% of TIA pts will have stroke within 3 months ➔ ALARM FOR COMING STROKE

Always act as if it is a stroke, and look for the cause.

• Case

A 67 yr old lady brought to ER because of sudden difficulty in talking, and weakness in Rt face, arm, and leg without sensory deficit.

What is difference between slurred speech and aphasia? Mechanical vs content

Is there any clinical scale can be used to determine the severity of the stroke? NIHSS

What is most likely affected artery? Lt MCA (incomplete because sensory is intact)

No headache; it must be an ischemic stroke? NO

When can I treat with thrombolysis using IV-tPA? Imaging first!

Guidelines for EMS Management of Patients with Suspected Stroke:

- Manage ABCs
- Cardiac monitoring
- Intravenous access
- Oxygen (keep O2 sat >92%)
- Assess for hypoglycemia
- NPO
- Alert receiving ED
- Rapid transport to closest appropriate facility capable of treating acute stroke

Not Recommended:

- Dextrose-containing fluids in non-hypoglycemic patients
- Excessive blood pressure reduction
- Excessive IV fluids

26.56 Management of acute stroke

Airway
- Check that the patient can protect his/her airway and swallow without evidence of aspiration
- Perform a swallow screen and keep patient nil by mouth if swallowing unsafe

Breathing
- Check that the patient is breathing adequately; check oxygen saturation and give oxygen if saturation < 95%

Circulation
- Check peripheral perfusion, pulse and blood pressure adequate and treat with fluid replacement, anti-arrhythmics and inotropic drugs as appropriate

Hydration
- Screen for signs of dehydration and give fluids parenterally or by nasogastric tube if necessary

Nutrition
- Assess nutritional status and provide nutritional supplements if necessary
- If dysphagia persists for a day or two, start feeding via a nasogastric tube

Medication
- If the patient is dysphagic, consider alternative routes for essential medications

Blood pressure
- Unless there is heart failure or renal failure, evidence of hypertensive encephalopathy or aortic dissection, do not lower the blood pressure in the first week since cerebral perfusion may decrease. Blood pressure often returns towards the patient's normal level within the first few days

Blood glucose
- Check blood glucose and treat with insulin when levels are ≥ 11.1 mmol/L (200 mg/dL) (via infusion or glucose/potassium/insulin (GKI)). Monitor closely to avoid hypoglycaemia

Temperature
- Check for pyrexia and investigate and treat underlying cause
- Give antipyretics since raised brain temperature may increase infarct volume

Pressure areas
- Check pressure areas and introduce measures to reduce the risk of bed sores
  - Treat infection
  - Maintain nutrition
  - Provide a pressure-relieving mattress
  - Turn immobile patients regularly

Incontinence
- Check for constipation and urinary retention and treat appropriately
- Avoid urinary catheterisation unless the patient is in acute urinary retention or incontinence is threatening pressure areas

Acute Ischemic Stroke Work-up

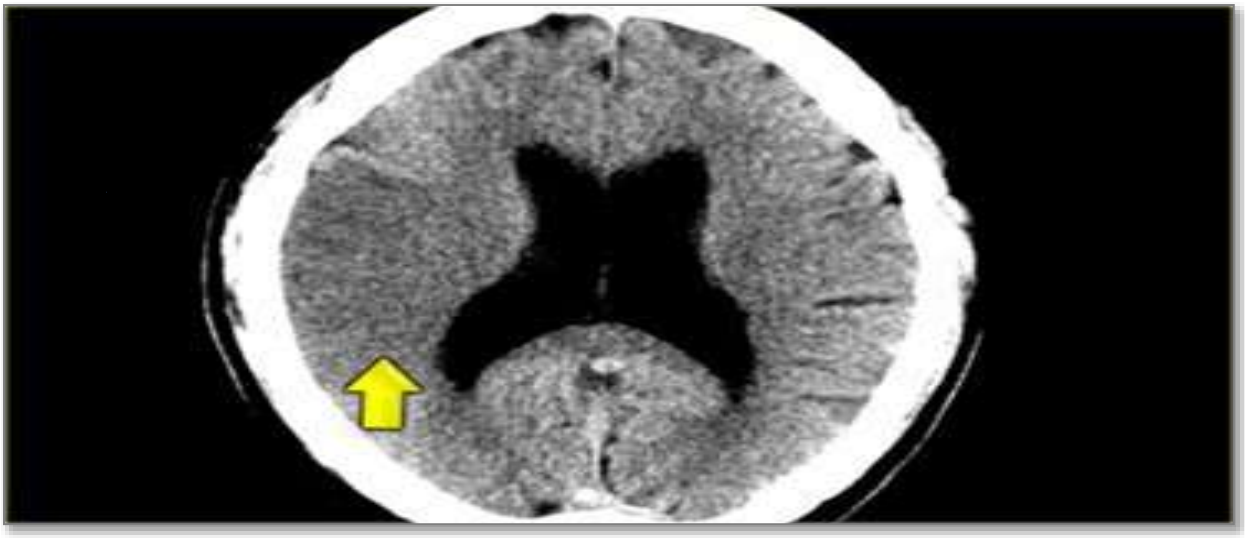
- Detailed and accurate history is ESSENTIAL

At ER:

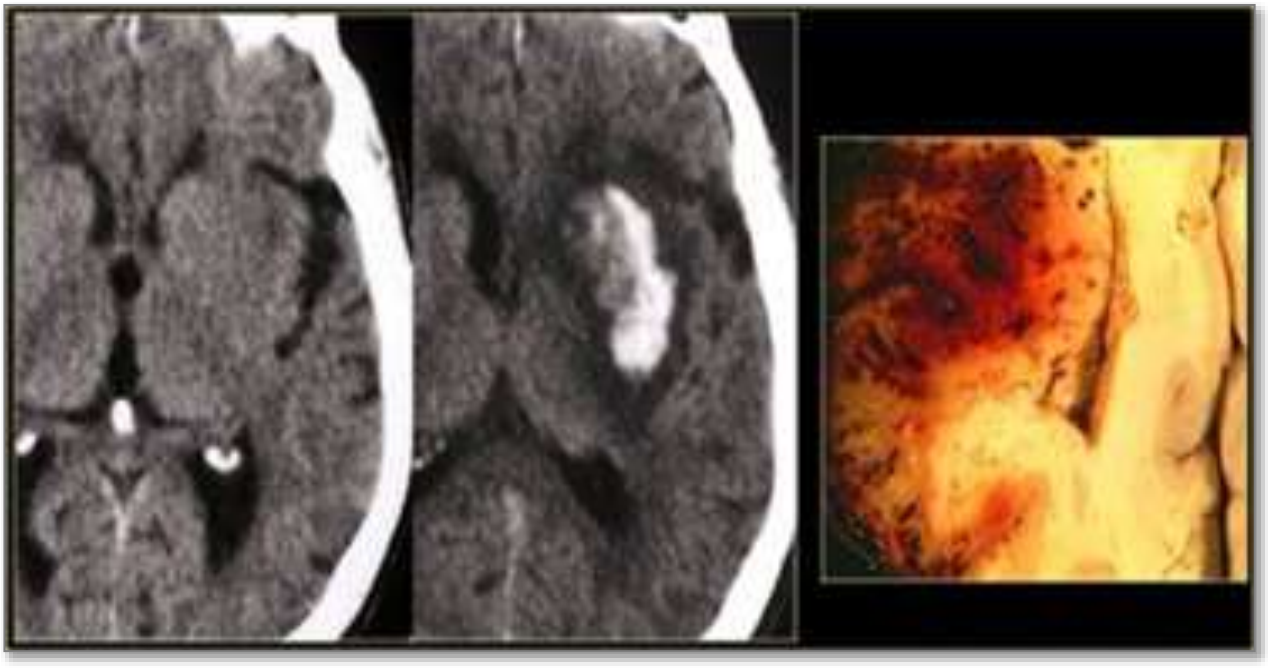
- CBC (check platelets, for thrombolytic intervention), lytes, Cr (kidney function if contrast is needed), and coagulation profile
- Blood sugar,look for hypoglycemia diabetes is the #1 stroke mimicker
- 12 leads ECG, and troponin to R/O MI
- CT (brain)… mainly to R/O hemorrhage (CT is the Best initial test and MRI is the most accurate test)
- Then acute stroke Rx if met indications and no contraindication but needs approval from pt or his family

• Acute stroke imaging:

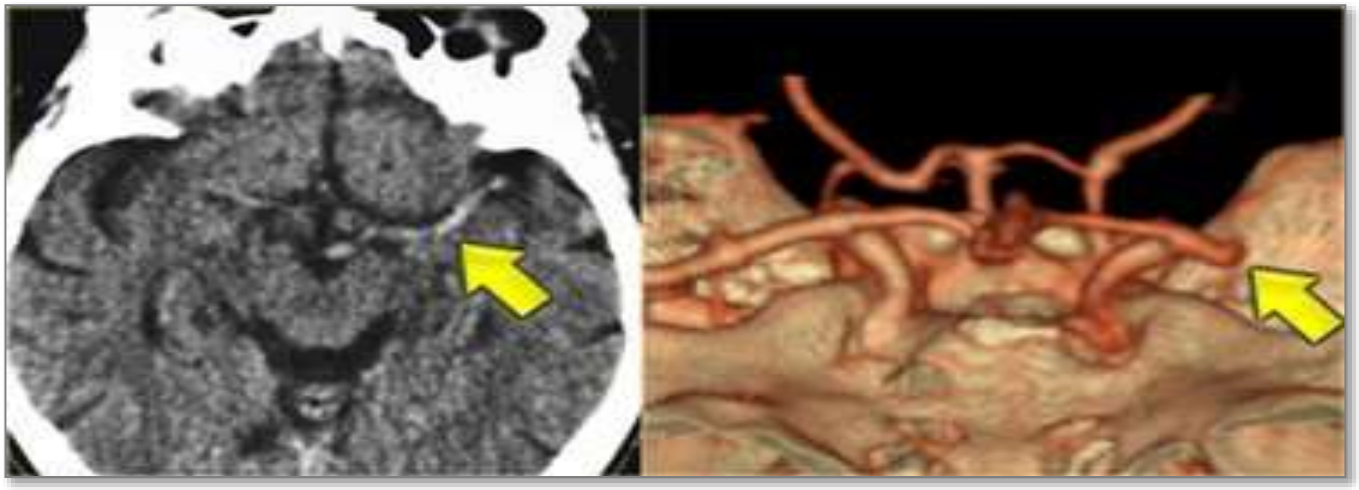
- Hypo-attenuation of brain tissues: loss of white/grey matter differentiation
- Sulcal effacement
- Insular ribbon sign
- Obscuration of lentiform nucleus
- Hyperdense sign (MCA>basilar>PCA)



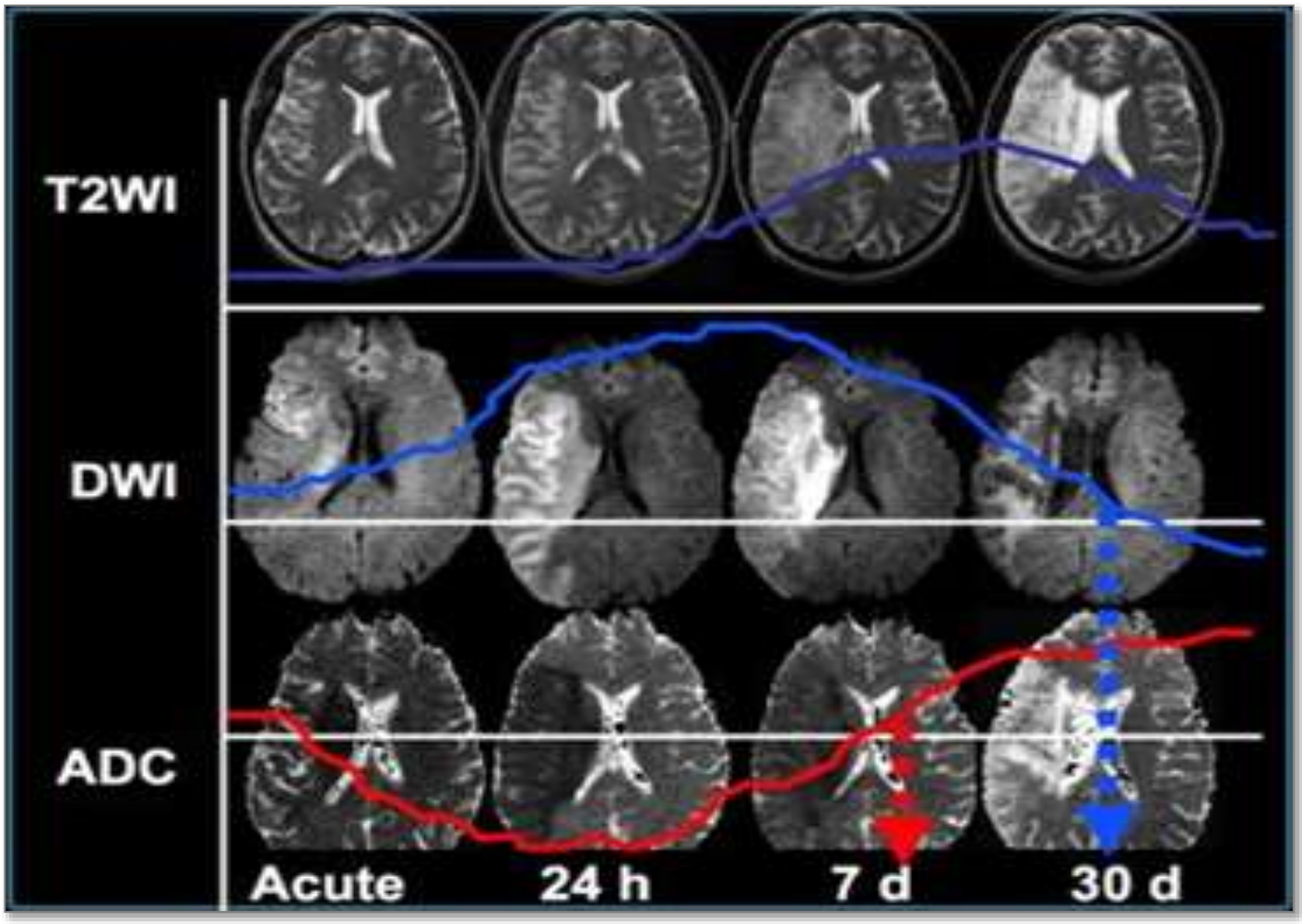
You can see the difference between the two, on one side the sulci and whit/grey matter margins are clear, while on the other side (arrow), effacement of sulci and cortical edema is present.



Hemorrhagic transformation, when recanalization happens to necrotic tissue, this is dealt with as ischemic! Stop aspirin until everything settles, then do an imaging after 4 days, and restart the treatment with antithrombotics.



Hyperdense sign



DWI is the most sensitive for hyperacute stroke.

Acute Stroke Treatment Options:

1. IV t-PA ➔ standard of care

2. Endovascular & mechanical disruption with/without IA t-PA ➔ for proximal MCA and basilar clots.

- may follow IV t-PA

Acute Ischemic stroke treatment using IV t-PA:

- Target: salvage the penumbra tissues (at risk, can be salvageable)

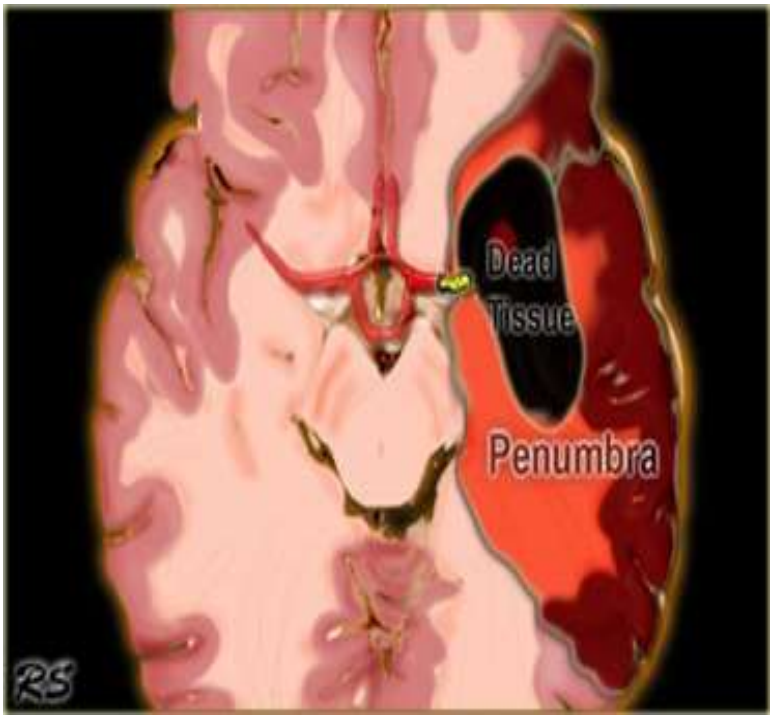
  (If you do DWI and all the tissue is already necrotic, no benefit of treatment and it's not worth the risk).

- 30 % more likely to have minimal or no disability at 3 months (NINDS trial)

- Chance of harm= 6.4% vs. 0.6% increase in the frequency of all symptomatic haemorrhage

  Chances of patient dying: With thrombolysis= 15%/ Without=10%

Penumbra is the area surrounding an ischemic event such as an ischemic, thrombotic or embolic stroke. Immediately following the event, blood flow and therefore oxygen transport is reduced locally, leading to hypoxia of the cells near the location of the original insult.



The doctor didn't go through them:

IV t-PA Contraindications:

B.P. > 185/110

Acute MI

Recent hemorrhage

LP within 7 days

Arterial puncture at incompressible site

Surgery within 14 days

Bleeding diathesis

Head trauma within 3 months

History of intracranial hemorrhage

Minor or rapidly improving stroke symptoms

V –tPA side effects:

6% develop **symptomatic intracerebral hemorrhage** within 36 hours following treatment (0.6% in placebo group).

• Half of the tPA associated symptomatic hemorrhages were fatal, however tPA treatment was not associated with an increase in mortality in the three-month outcome analysis.

Facial angioedema: another side effect which may cause airway obstruction.

Antiplatelet Rx:

Oral administration of ASA 325 mg within 24 to 48 hours after stroke onset is recommended for tx of most pts.

BP management:

- For IV-tPA: follow NINDS guidelines

185/110

- Not candidate for thrombolysis:

220/120 (we reduce it to 200)

- Use Labetalol IV 10 mg Q 30 min. PRN
- ➢ Avoid quick reduction in BP and look for bradycardia.
- Alternative: Hydralazine IV
- Avoid strong vasodilators

Stroke Work-up (after acute stroke Mx):

- Fasting blood glucose and lipid profile
- Carotid US ➔ in all pts
- Echocardiogram/ 24 hr holter monitor to R/O paroxysmal At.Fib ➔ for pts with embolic stroke

<u>In selected cases:</u>

- MRI/MRA brain
- CT angio (extrcranial and intracranial BVs)
- Screen for hyper-coagulopathies
- Many other tests to identify the cause and then improve the secondary prevention strategy

➔ Each patient is different, old is different than young, thrombophilia and blood or heart causes usually in young/ cardiogenic, atherosclerosis etc… old

Secondary stroke prevention:

- Antiplatelet therapy (aspirin, dipyridamole, or plavix)
- Combined antiplatelet Rx …. In special scenarios
- Statin….. Keep LDL cholesterol 1.3 – 2
- Antocoagulation for At.Fib or hypercoagulopathy
- Avoid unnecessary anticoagulation
- Carotid artery surgery (CEA or stent)
- *Many uncommon causes of stroke exist and each require special approach and wt benefits vs risks. Therefore;* ***secondary stroke prevention is better done at specialized stroke prevention clinics run by stroke experts***

Post stroke care:

- Maximize secondary stroke prevention

- Rehabilitation (motor, language, behavioral,…)
- Special care for swallowing and DVT prophylaxis
- Most limiting factors for rehab are:

  1) Vascular dementia

  2) Extensive large stroke

- Prognosis:

  Without thrombolysis: 10% die, 30% mild, 30% moderate, and 30% severe disability

  With thrombolysis: 9% die, and 30% more chance of complete recovery *(great Rx but not perfect)*

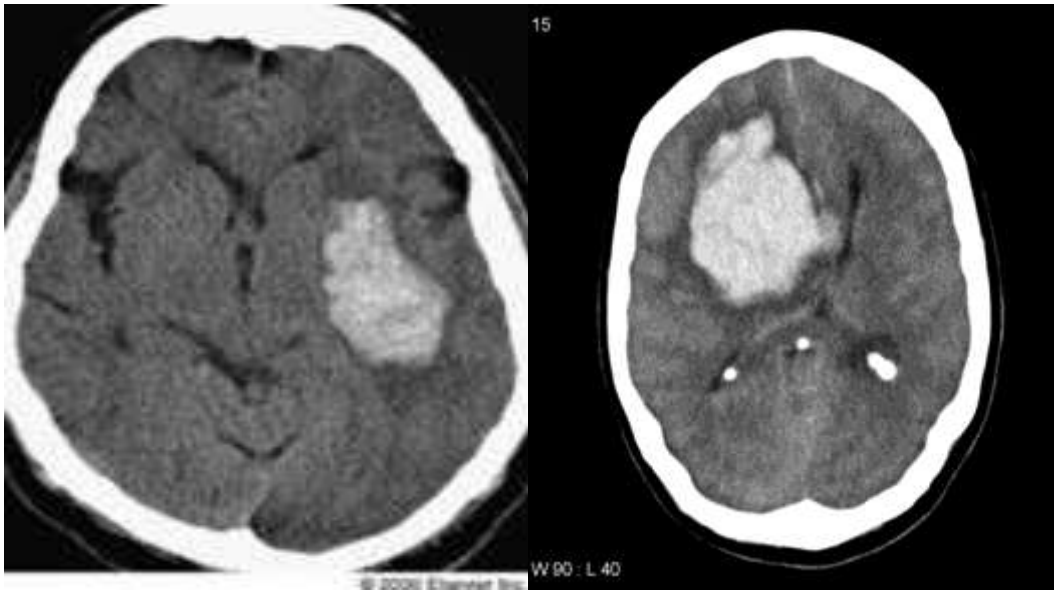
Intracranial Hemorrhage:

Common causes

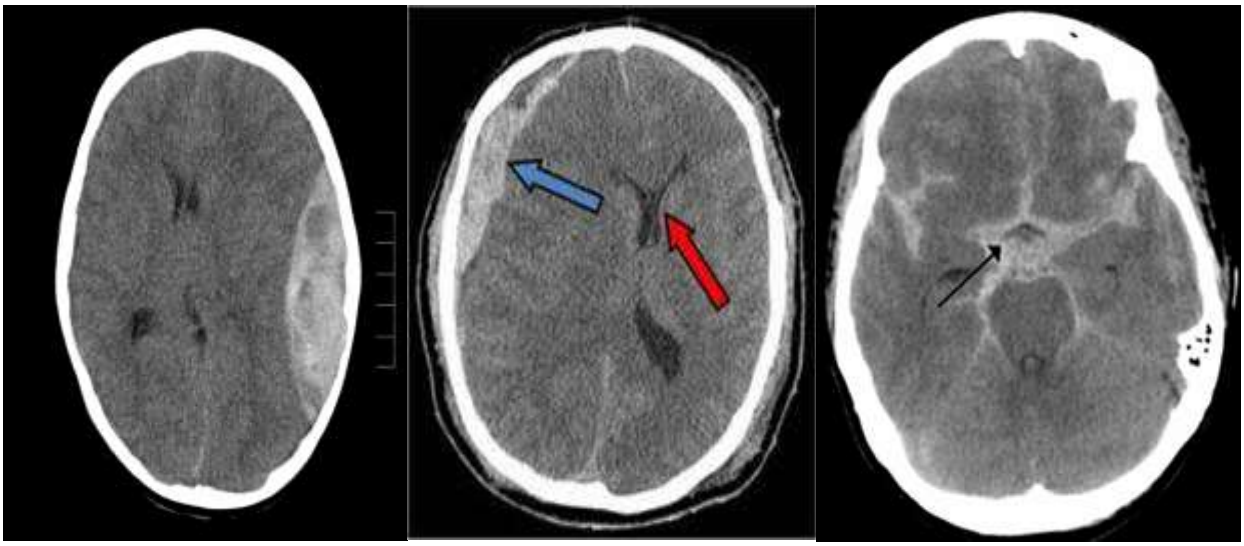
1. Hypertension
2. Trauma
3. Amyloid angiopathy.
4. Ruptured vascular malformation.
5. Coagulopathy (a disease or drug-induced)
6. Hemorrhage into a tumor.
7. Venous infarction.
8. Drug abuse.

HTN- Induced ICH:

- Can be putaminal, thalamic, cerebellar, or lobar.
- Can be seen in acute HTN or chronic one
- Can be fatal



Traumatic Intracranial Hemorrhage:



From right to left: Subarachanoid,subdural,epidural.

Subarachnoid Hemorrhage:

- Worst headache ever
- Use: H&H scale
- Spont. Vs. traumatic
- Risk of aneurysms increase with smoking (X40 times)(doc said times 25)
- Sacular anurysms are more in anterior circulation (90%) while fusiform more in basilar
- 1st & 4th tubes of CSF for cells
- Need conventional angiogram and neurosurgery consultation for clipping or coiling

Case:

- 30 year old lady in postpartum developed severe diffuse headache and blurred vision for about 1 day. Clinical exam showed papilledema bilaterally
- DDx?? Cerebral venous sinus thrombosis

  Pseudotumor cerebri (of exclusion if MRI/V is Normal)
- Approach?? Imaging (brain MRV or CTV are preferred)

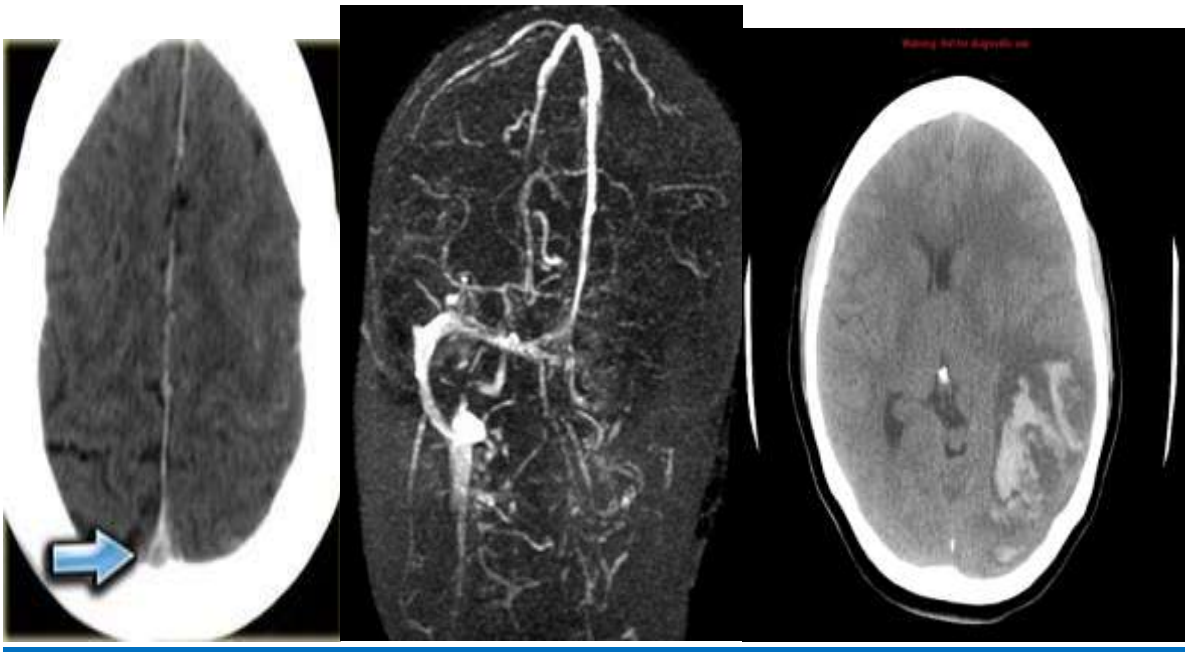
  Opening pressure in LP will be HIGH in BOTH!!
- Management?? Anticoagulation, and look for the CAUSE

Cerebral Venous Sinus Thrombosis

#1 cause here is dehydration., oral contraceptives can cause it.

#1 cause in children is otitis media to transverse sinus, in adults superior sagittal sinus is more common, causes headache and visual problems.

cortical vein thrombosis causes headaches and focal seziures.



Empty delta sign **MRV** **Venous Hge in CT(brain)**

CT (brain) with contrast

SUMMARY

1- Stroke can be ischemic, hemorrhagic or venous

2- Age is the most important risk factor, while HTN is the most important modifiable one.

3- the presentation in a patient depends on the location of the attack

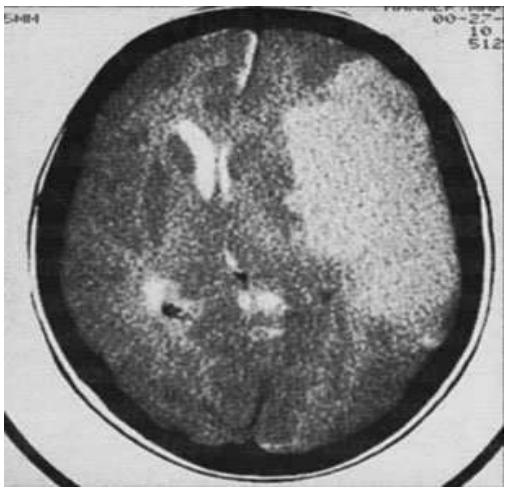
4- the aim of antithrombotic treatment is to salvage the penumbra

5- IV Tp-A is the standard of care

6- don’t treat unless you R/O hemorrhage by imaging

Questions

1) A 68-year-old man with a history of hypertension and coronary artery disease presents with right-sided weakness, sensory loss, and an expressive aphasia. Neuroimaging studies are shown. In the emergency department the patient's blood pressure is persistently 160/95. Which of the following is the best next step in management of this patient's blood pressure?



a. Administer IV nitroprusside.
b. Administer oral clonidine 0.1 mg po until the blood pressure drops below 140/90.
c. Observe the blood pressure.
d. Administer IV mannitol.
e. Administer IV labetolol.

2) A 72-year-old woman is found unconscious at home by her daughter. In the emergency room the patient does not respond to verbal or noxious stimuli. Which of the following is the most likely cause of her condition?

a. Hypoglycemia
b. Left posterior cerebral artery occlusion
c. Lacunar infarct in the right internal capsule
d. Middle cerebral artery occlusion
e. Anterior cerebral artery occlusion

1) The answer is C. Although hypertension is an important cause of stroke, it should not be aggressively treated in the setting of acute cerebral ischemia. Since cerebral autoregulation is disrupted in acute stroke, a drop in blood pressure can decrease perfusion and worsen the so-called ischemic penumbra. Generally, blood pressure elevation up to 185/110 is not treated. Some stroke specialists recommend more aggressive blood pressure control in acute intracranial hemorrhage, but this patient has an ischemic (not hemorrhagic) stroke. Mannitol is of minimal benefit in cere- bral edema associated with acute stroke.

2) The answer is a. Focal disorders of the cerebral hemisphere do not cause coma unless the brainstem is compressed by edema or mass effect. Coma implies either severe metabolic derangement of the brain (ie, hypoglycemia, hyponatremia, intoxication), brainstem dysfunction (affect- ing the reticular activating system of the pons), or else bilateral hemispheric insults. Posterior cerebral artery occlusion will cause an occipital lobe infarc- tion with homonymous hemianopsia but should not affect the level of con- sciousness. Similarly, a lacunar infarct will cause a pure motor or pure sensory stroke but not global brain dysfunction. Although the patient with a middle cerebral artery stroke may be unable to speak, she should be awake and alert. Anterior cerebral artery occlusion causes motor and sensory deficits of the con- tralateral leg and foot but does not impair global brain function.