CEREBROVASCULAR DISEASES

for medical students

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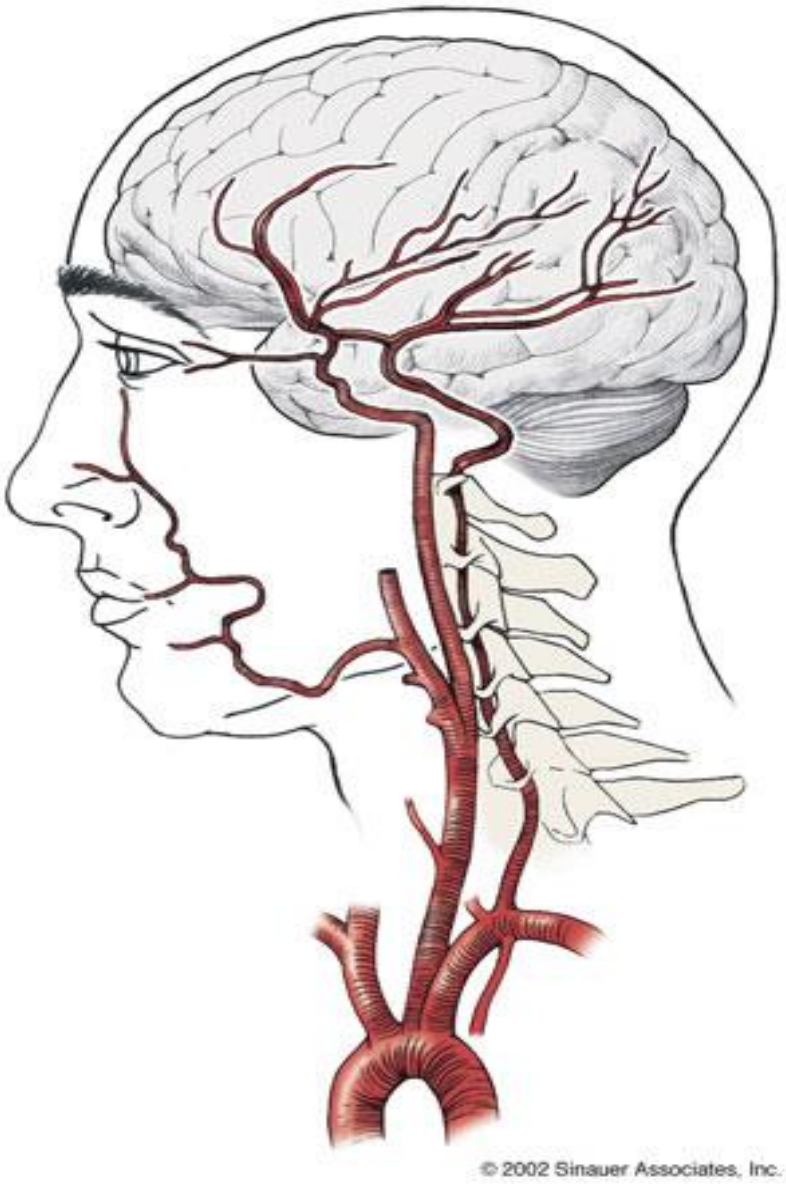
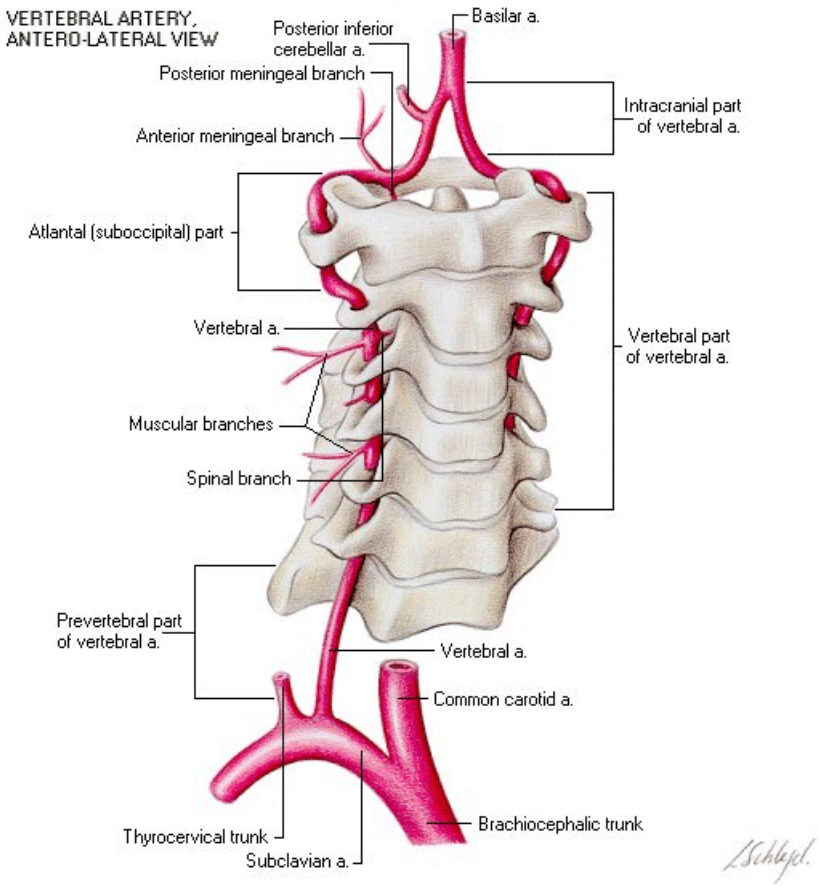
Assistant Professor

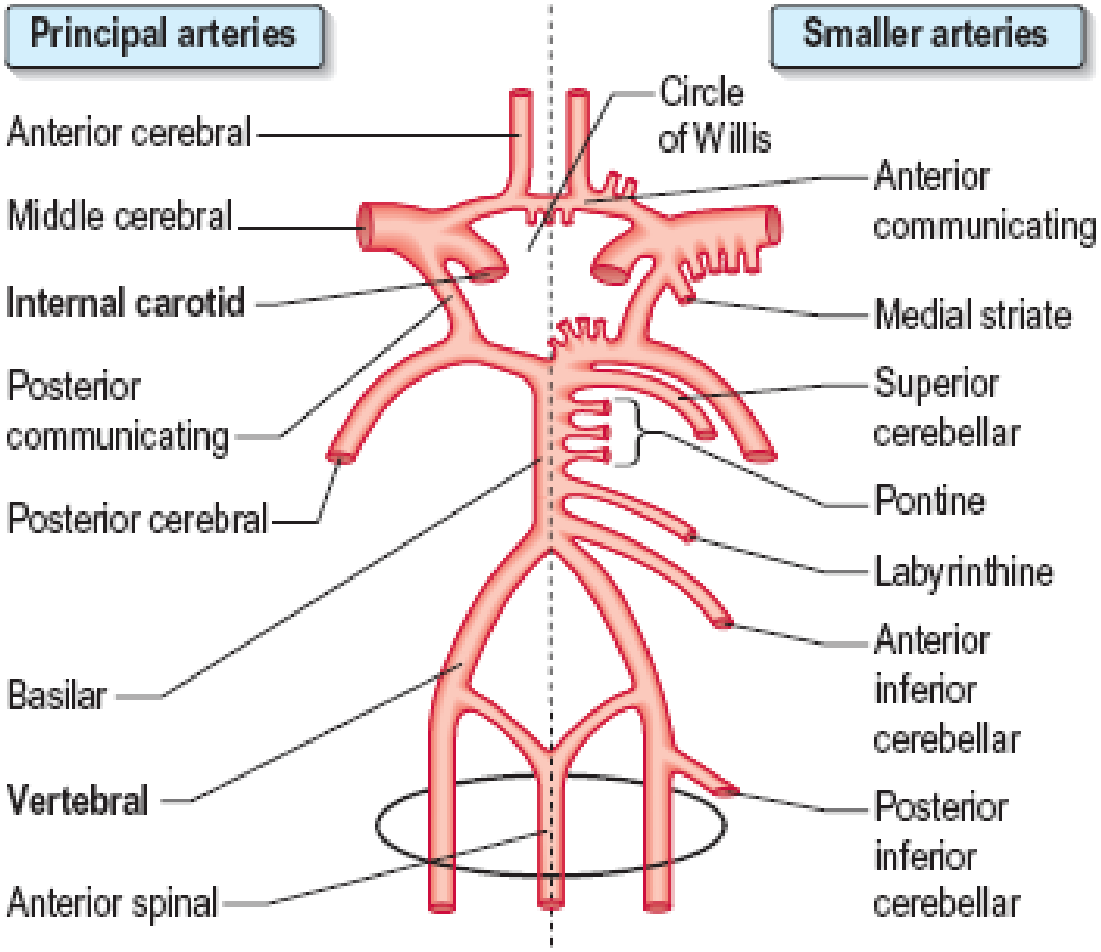
Stroke Neurologist

Anatomy:

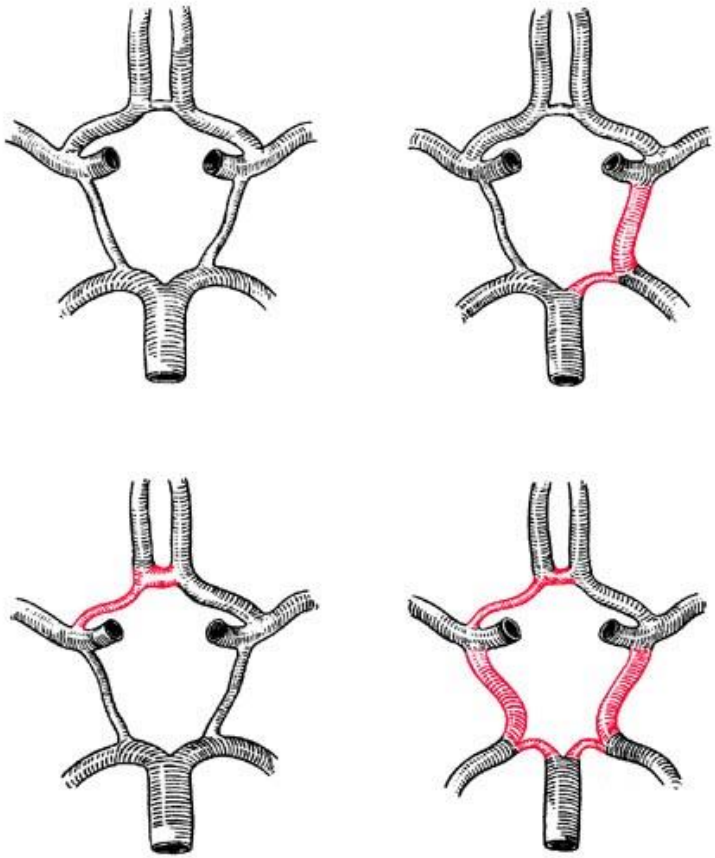



VERTEBRAL ARTERY, ANTERO-LATERAL VIEW
Basilar a.
Posterior inferior cerebellar a.
Posterior meningeal branch
Anterior meningeal branch
Intracranial part of vertebral a.
Atlantal (suboccipital) part
Vertebral a.
Vertebral part of vertebral a.
Muscular branches
Spinal branch
Prevertebral part of vertebral a.
Vertebral a.
Common carotid a.
Brachiocephalic trunk
Thyrocervical trunk
Subclavian a.

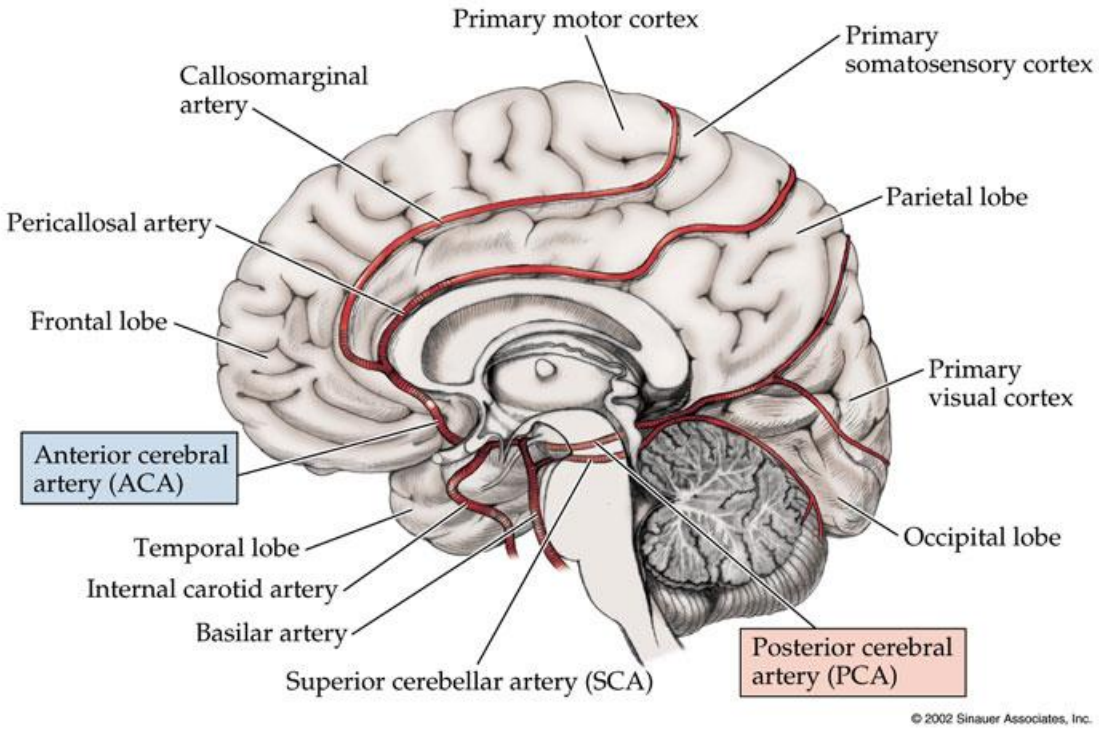
Intracranial cerebro-vascular system



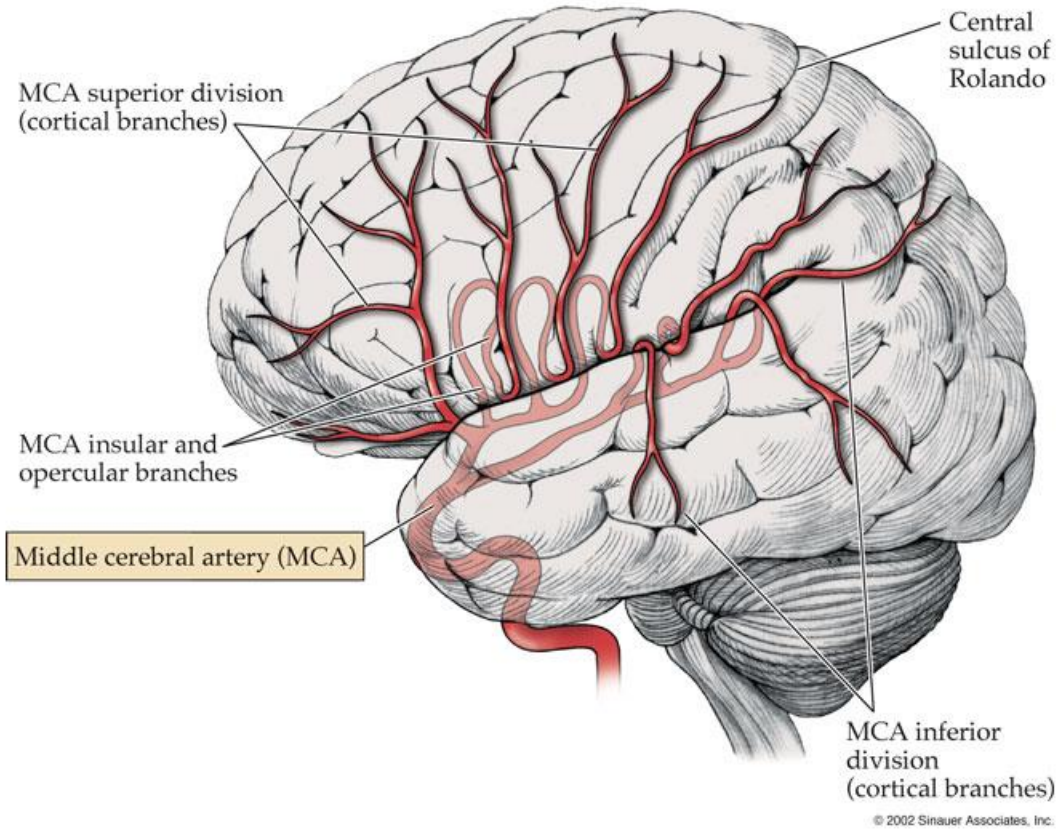
Circle of Willis: variants



ACA & PCA


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)
© 2002 Sinauer Associates, Inc.

MCA


Central sulcus of Rolando
MCA superior division (cortical branches)
MCA insular and opercular branches
Middle cerebral artery (MCA)
MCA inferior division (cortical branches)
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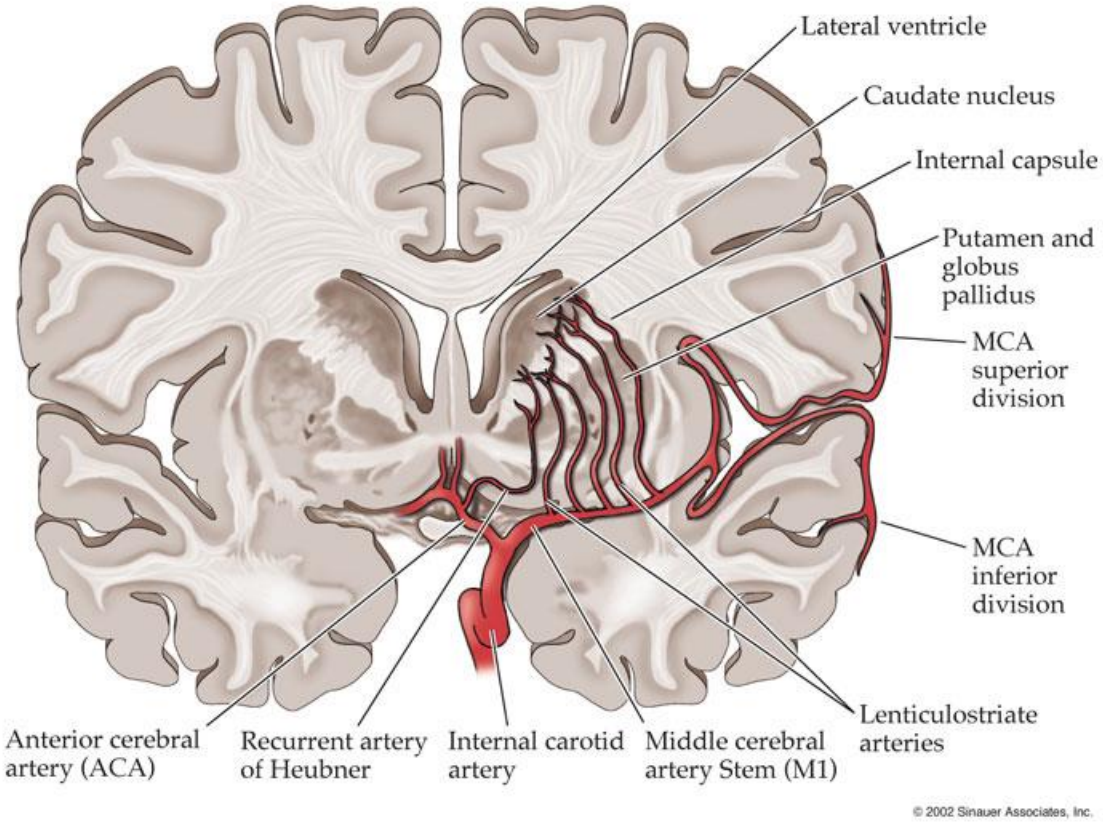
MCA


Lateral ventricle
Caudate nucleus
Internal capsule
Putamen and globus pallidus
MCA superior division
MCA inferior division
Lenticulostriate arteries
Anterior cerebral artery (ACA)
Recurrent artery of Heubner
Internal carotid artery
Middle cerebral artery Stem (M1)
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Common acute stroke presentation based on arterial distribution:

- ACA
- MCA → M1
  Supperior M2
  Inferior M2
- PCA
- Basilar
- Sup. Cerebellar artery
- Wallenberg (lateral medulary syndrome)

AND

- 5 Kinds of lacunar strokes (motor, motor & sensory, sensory, ataxic hemiparesis, and dysarthria-clumbsy hand syndrome)

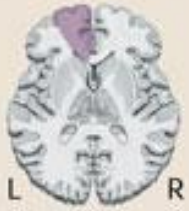
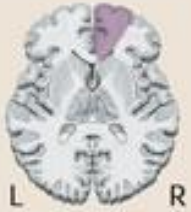
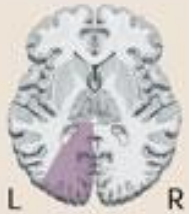
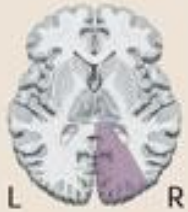
TABLE 10.1 Major Clinical Syndromes of the MCA, ACA, and PCA Territories *(Part 1)*

LOCATION OF INFARCT	AFFECTED TERRITORY	DEFICITS[a]
Left MCA superior division		Right face and arm weakness of the upper motor neuron type, and a nonfluent, or Broca's, aphasia. In some cases there may also be some right face and arm cortical-type sensory loss.
Left MCA inferior division		Fluent, or Wernicke's, aphasia and a right visual field deficit. There may also be some right face and arm cortical-type sensory loss. Motor findings are usually absent, and patients may initially seem confused or crazy, but otherwise intact, unless carefully examined. Some mild right-sided weakness may be present, especially at the onset of symptoms.
Left MCA deep territory		Right pure motor hemiparesis of the upper motor neuron type. Larger infarcts may produce "cortical" deficits as well, such as aphasia.
Left MCA stem		Combination of the above, with right hemiplegia, right hemianesthesia, right homonymous hemianopia, and global aphasia. There is often a left gaze preference, especially at the onset, caused by damage to left hemisphere cortical areas important for driving the eyes to the right.

TABLE 10.1 Major Clinical Syndromes of the MCA, ACA, and PCA Territories *(Part 2)*

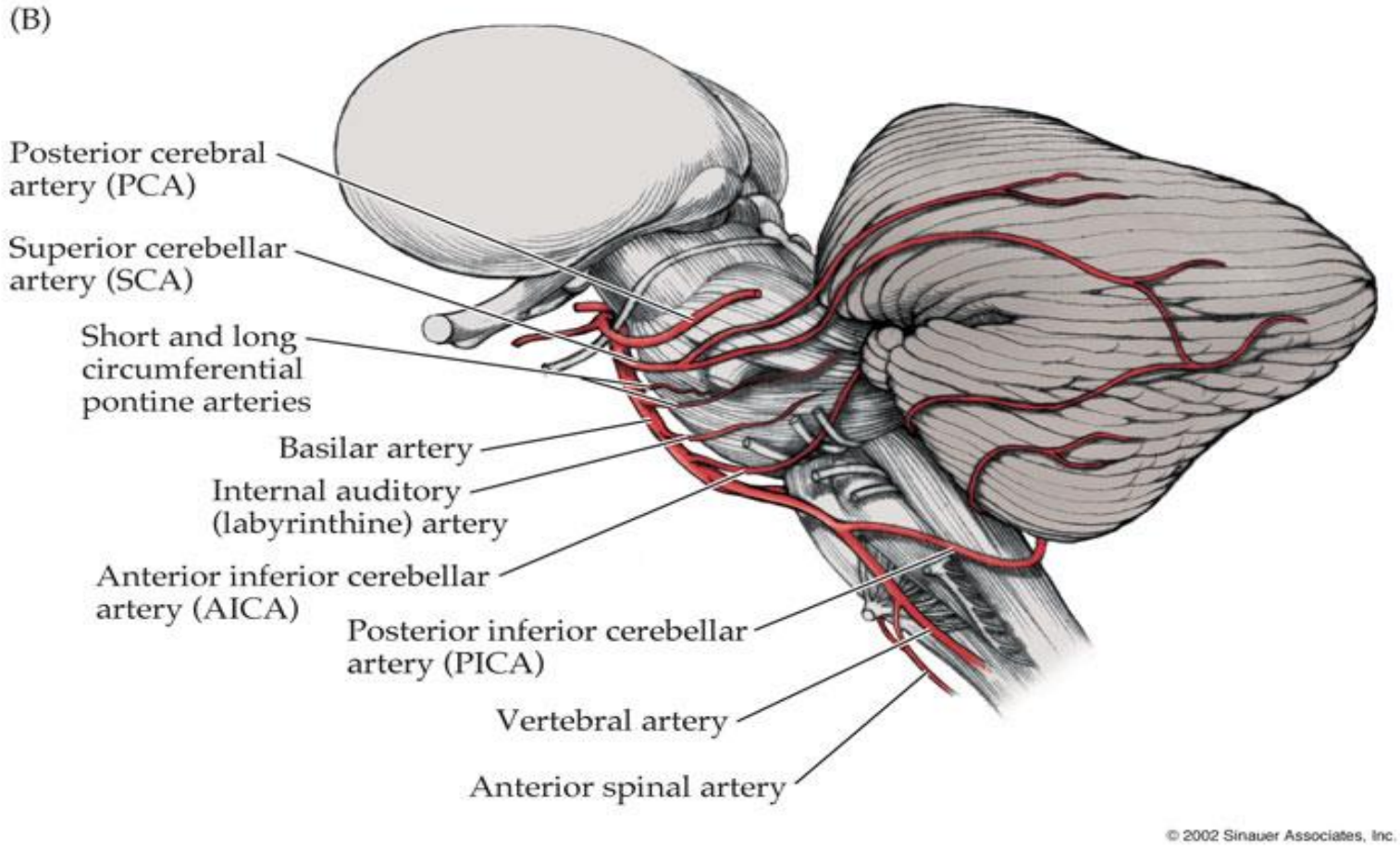
LOCATION OF INFARCT	AFFECTED TERRITORY	DEFICITS[a]
Right MCA superior division		Left face and arm weakness of the upper motor neuron type. Left hemineglect is present to a variable extent. In some cases there may also be some left face and arm cortical-type sensory loss.
Right MCA inferior division		Profound left hemineglect. Left visual field and somatosensory deficits are often present; however, these may be difficult to test convincingly because of the neglect. Motor neglect with decreased voluntary or spontaneous initiation of movements on the left side can also occur. However, even patients with left motor neglect usually have normal strength on the left side, as evidenced by occasional spontaneous movements or purposeful withdrawal from pain. Some mild right-sided weakness may be present. There is often a right gaze preference, especially at onset.
Right MCA deep territory		Left pure motor hemiparesis of the upper motor neuron type. Larger infarcts may produce "cortical" deficits as well, such as left hemineglect.
Right MCA stem		Combination of the above, with left hemiplegia, left hemianesthesia, left homonymous hemianopia, and profound left hemineglect. There is usually a right gaze preference, especially at the onset, caused by damage to right hemisphere cortical areas important for driving the eyes to the left.

TABLE 10.1 **Major Clinical Syndromes of the MCA, ACA, and PCA Territories** *(Part 3)*

LOCATION OF INFARCT	AFFECTED TERRITORY	DEFICITS[a]
Left ACA		Right leg weakness of the upper motor neuron type and right leg cortical-type sensory loss. Grasp reflex, frontal lobe behavioral abnormalities, and transcortical aphasia can also be seen. Larger infarcts may cause right hemiplegia.
Right ACA		Left leg weakness of the upper motor neuron type and left leg cortical-type sensory loss. Grasp reflex, frontal lobe behavioral abnormalities, and left hemineglect can also be seen. Larger infarcts may cause left hemiplegia.
Left PCA		Right homonymous hemianopia. Extension to the splenium of the corpus callosum can cause alexia without agraphia. Larger infarcts including the thalamus and internal capsule may cause aphasia, right hemisensory loss and right hemiparesis.
Right PCA		Left homonymous hemianopia. Larger infarcts including the thalamus and internal capsule may cause left hemisensory loss and left hemiparesis.

[a]Compare regions of infarcts to Figure 10.1.

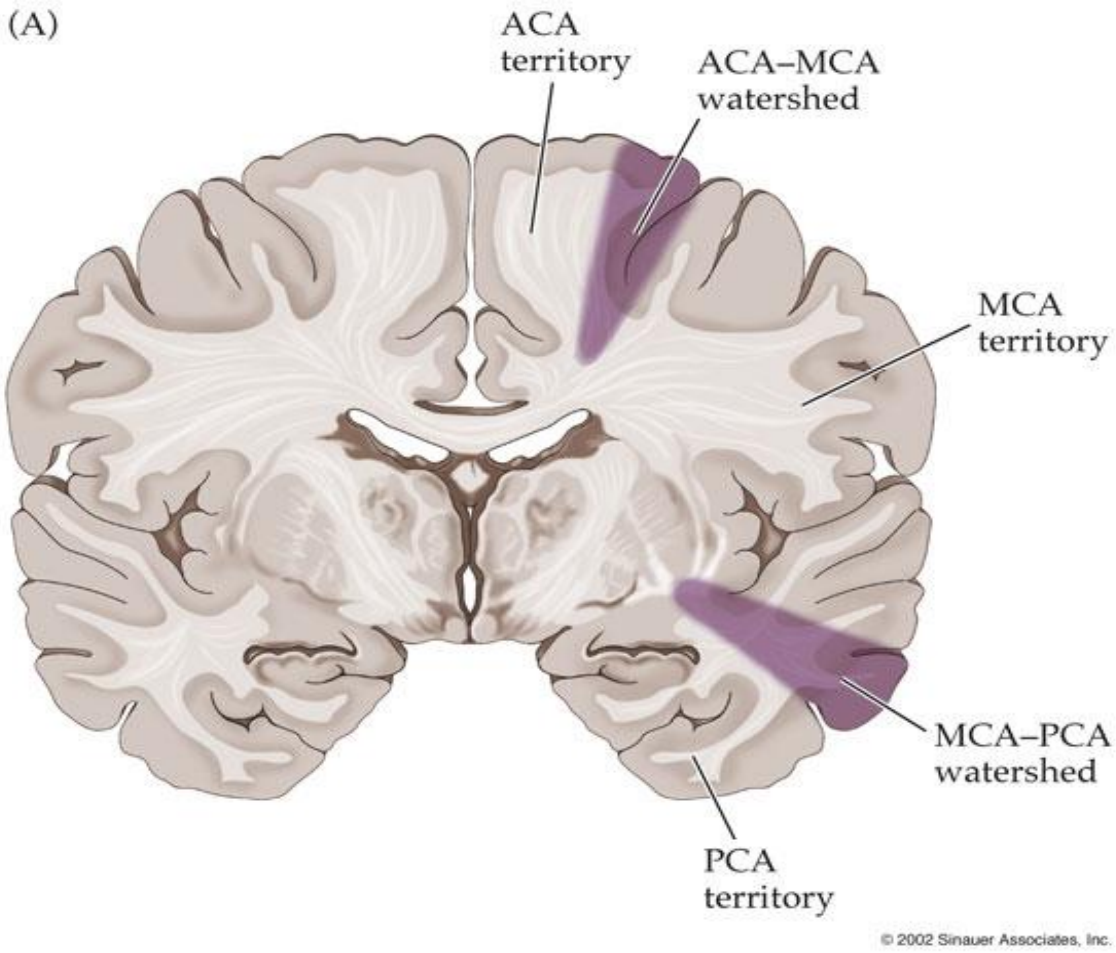
Vertbero-Basilar System



Features suggestive of brainstem stroke

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

Watershed Areas: vulnerable for hyopoperfusion



Stroke Risk Factors

- ***Age***
- Male gender
- Family History
- Genetic causes
- Congenital abnormality like in heart or AVM in CNS
- Hyper-coagulopathies

- ***HTN***
- Diabetes
- Hyperlipidemia
- Atrial Fibrillation
- Carotid artery disease
- Physical inactivity
- Obstructive sleep apnea
- Smoking
- Substance abuse
- Medications
- Dissection

AND MANY OTHERS,,,,,,,,,,,,,,,,

Stroke Types

Ischemic

- Thrombosis
- Embolism
- Lacunar
- Hypo-perfusion

Hemorrhagic

- Epidural
- Subdural
- Subarachnoid
- Intra-cerebral
- Intra-ventricular

Venous

- Venous sinus thrombosis
- Cortical vein thrombosis

Stroke Epidemiology

- About 30-40K new cases annually
- 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

Case-1

- 62 yr old man presented to ER because of slurred speech and mild weakness in Rt face and arm lasted about 30 minutes then resolved spontaneously. His neurological exam at ER was unremarkable.

Q-1: What is the most likely diagnosis? And would CT(brain) result matters??

Q-2: How would you manage such patient?

TIA

- 2002 definition:

  A brief episode of neurological dysfunction caused by focal brain or retinal ischemia, with clinical symptoms typically lasting <1 hr, and without evidence of acute infarction

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

Case-2

- 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.

Questions:

~ What is difference between slurred speech and aphasia?

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

~ What is most likely affected artery?

~ No headache; so it must be an ischemic stroke !

~ When can I treat with thrombolysis using IV-tPA?

Case-2

- 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.

Questions:

~ 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

~ No headache; it must be an ischemic stroke! NO

~ When can I treat with thrombolysis using IV-tPA?

Pre Hospital Mx:

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

Acute Ischemic Stroke Work-up

- Detailed and accurate history is ESSENTIAL

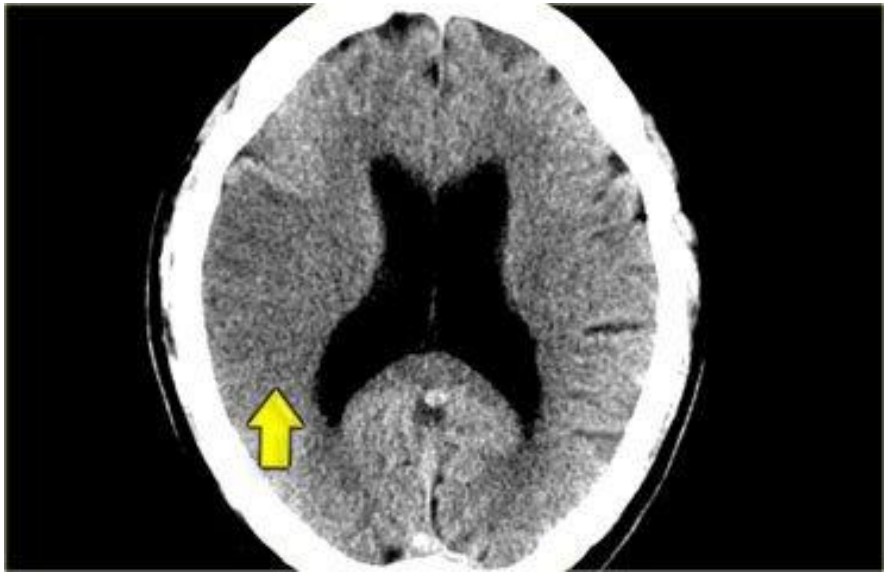
At ER

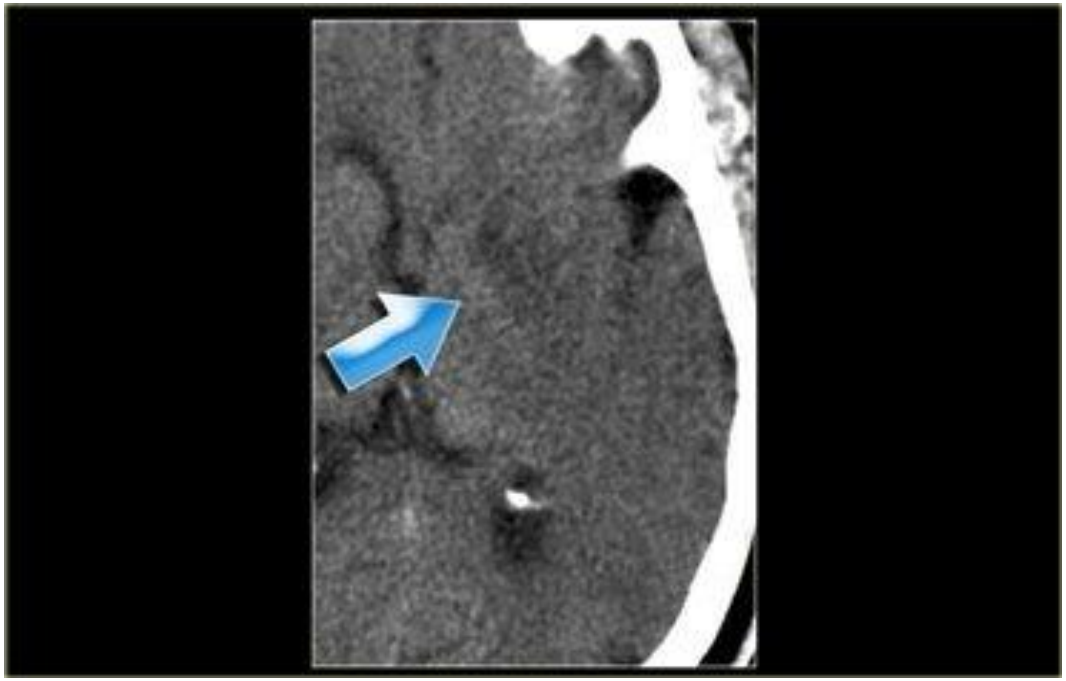
- CBC, lytes, Cr, and coagulation profile
- 12 leads ECG, and troponin
- CT (brain)… mainly to R/O hemorrhage

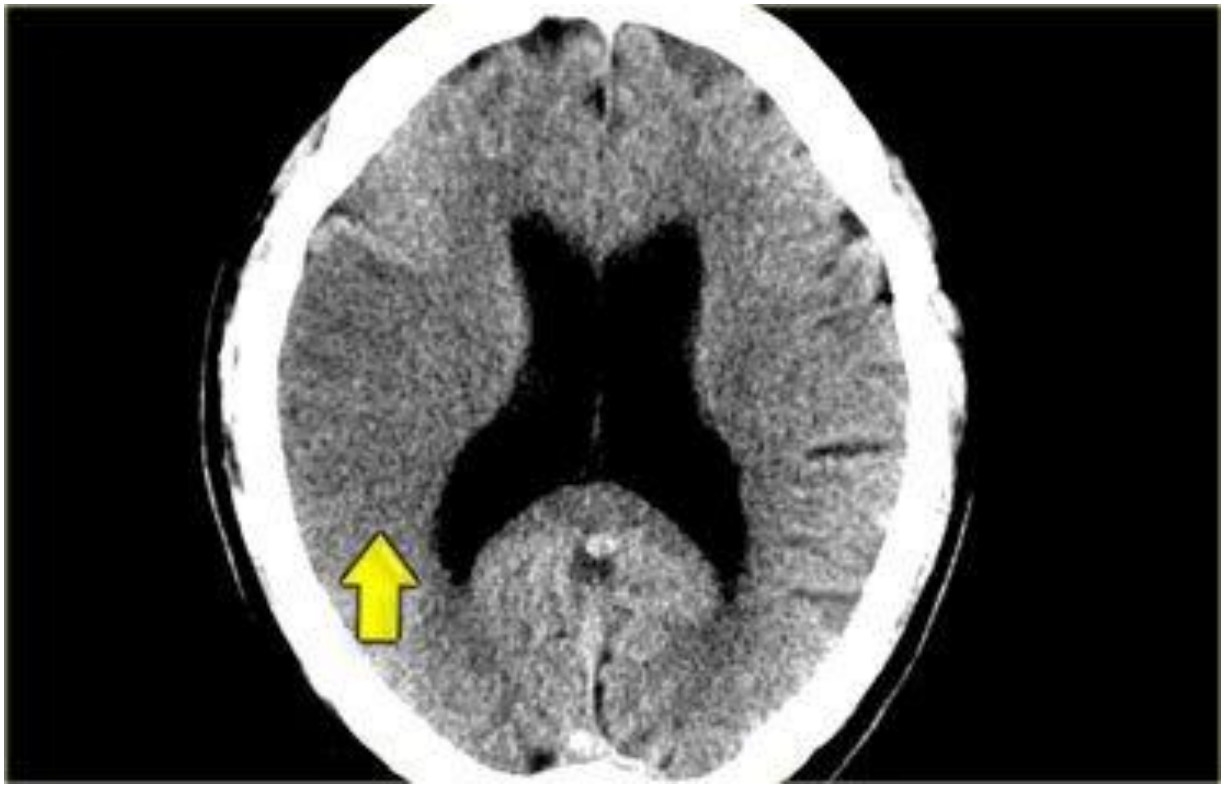
- Then acute stroke Rx if met indications and no contraindication but needs approval from pt or his family

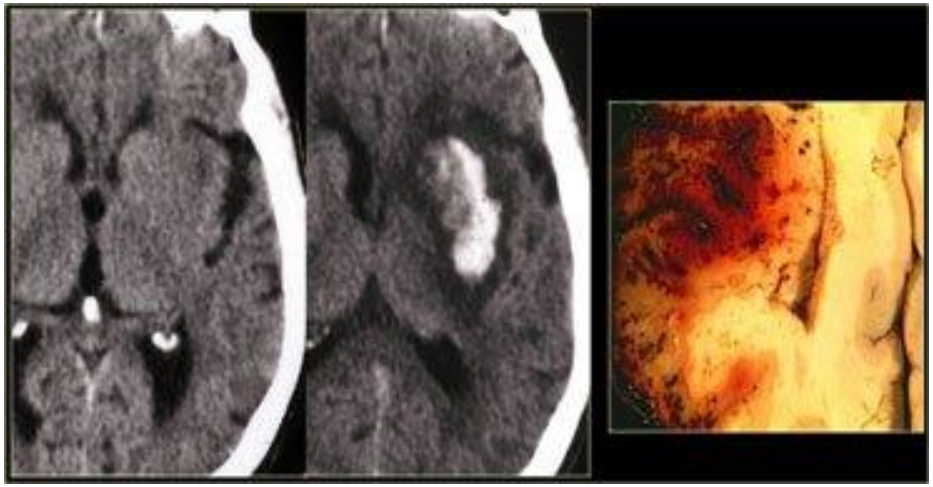
Acute stroke imaging:

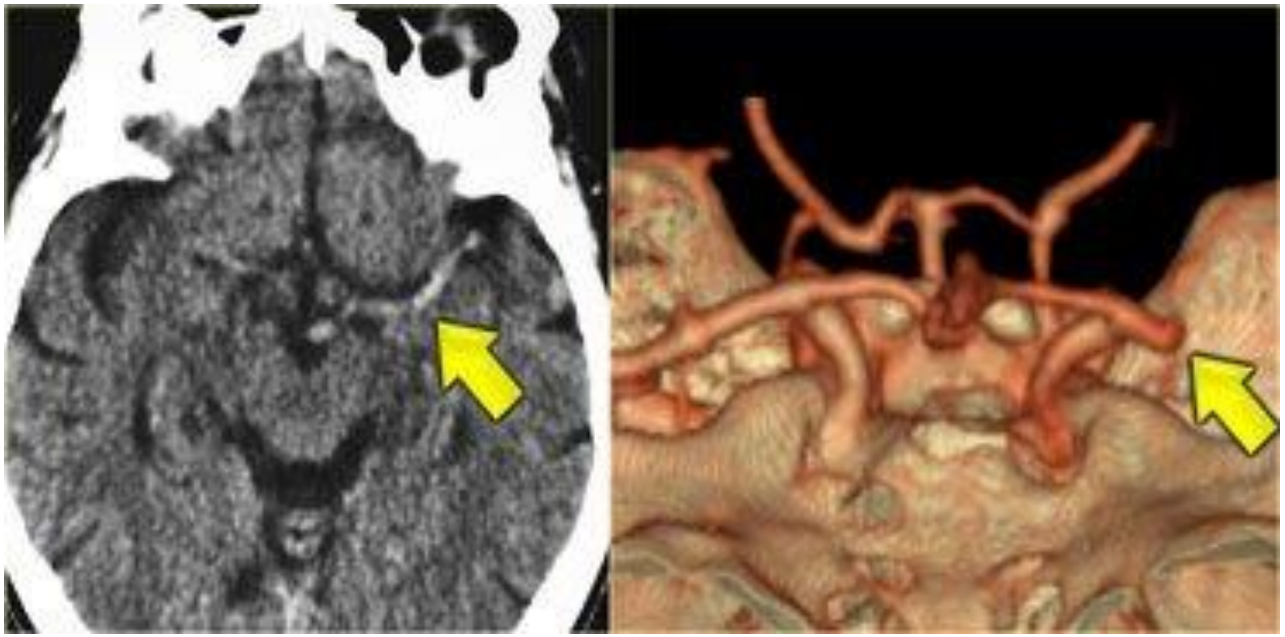
- Hypo-attenuation of brain tissues
- Loss of sulcal efffacement
- Insular ribbon sign
- Obscuration of lentiform nucleus
- Hyperdense sign (MCA>basilar>PCA)

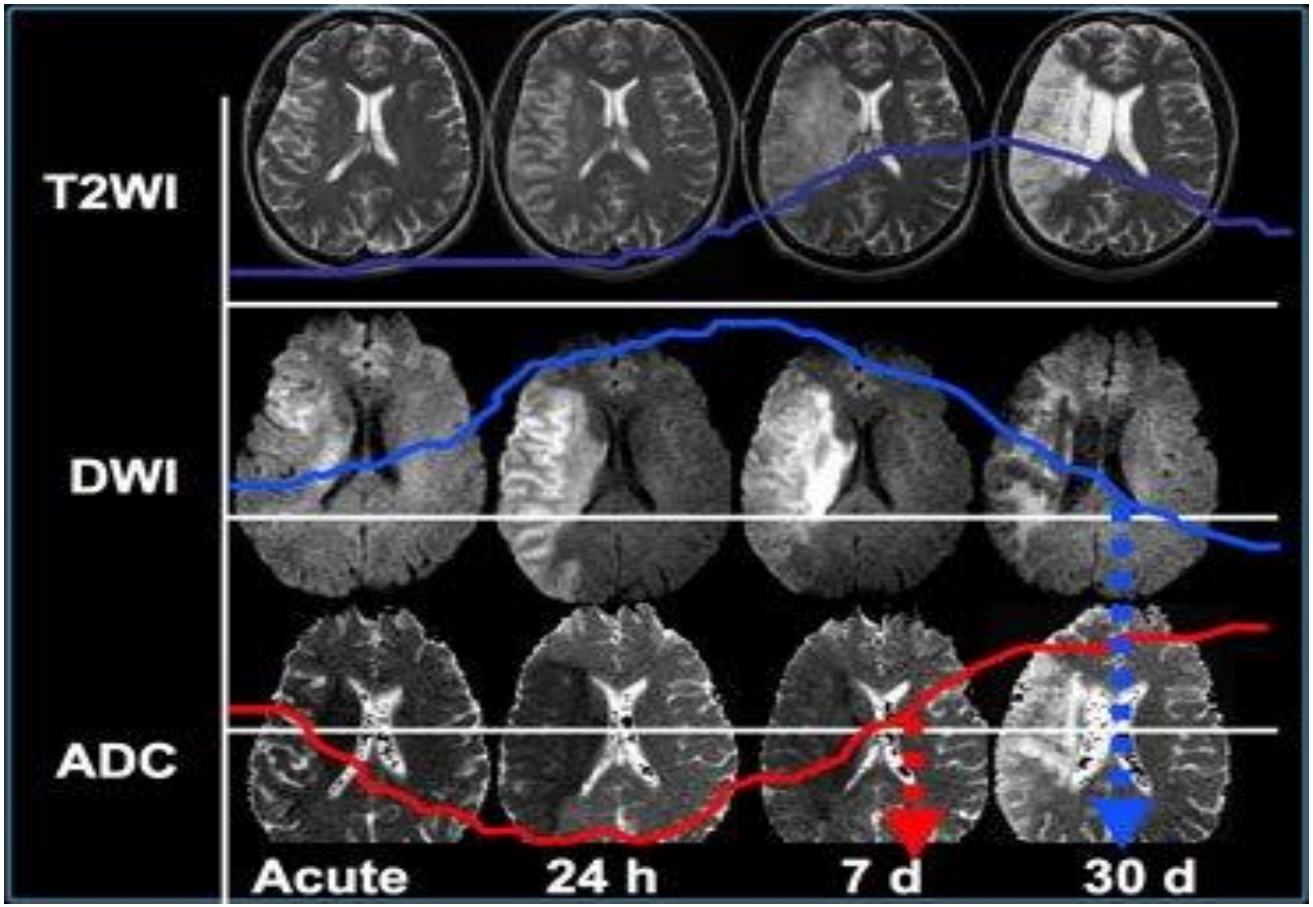










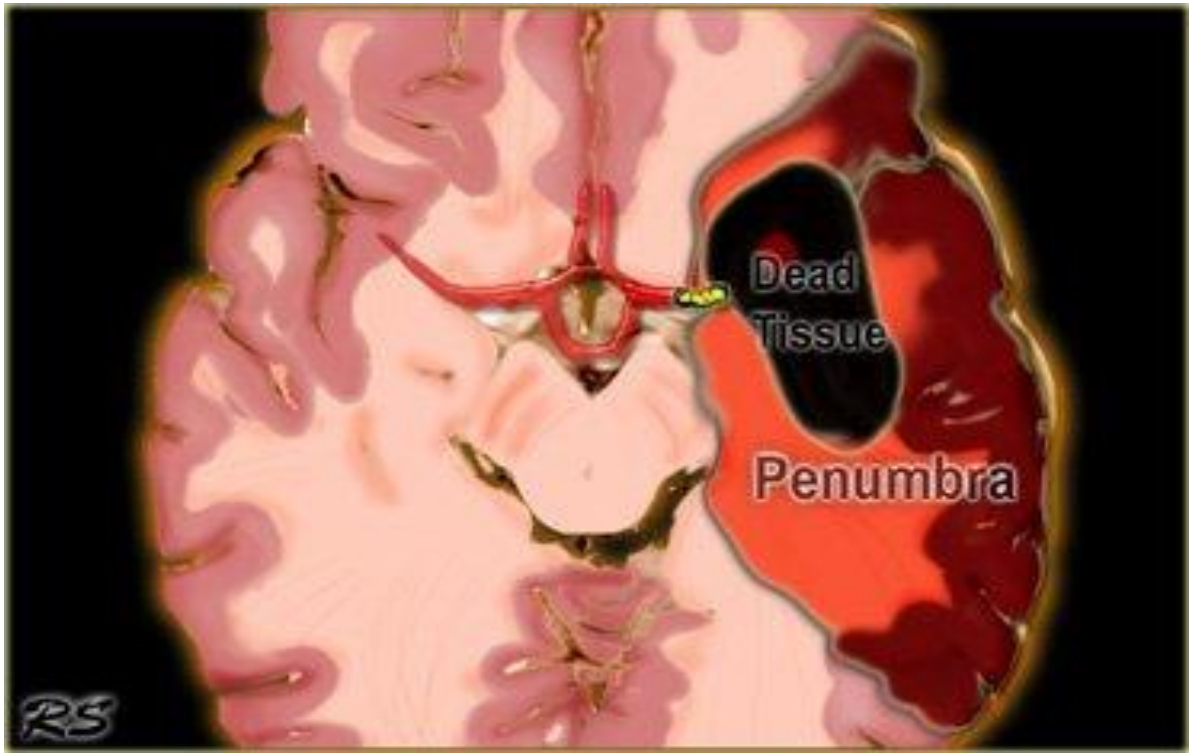

T2WI
DWI
ADC
Acute
24 h
7 d
30 d

Acute Stroke Treatment Options

- IV t-PA
- IA t-PA
- IV t-PA followed by IA t-PA
- Endovascular & mechanical disruption

Acute Ischemic stroke treatment using IV t-PA

- Only TPA approved for ischemic stroke if given within 4.5 hours of stroke onset
- Target: salvage the penumbra tissues (at risk)
- 30 % more likely to have minimal or no disability at 3 months (NINDS trial)
- 6.4% vs. 0.6% increase in the frequency of all symptomatic hemorrhage
- IA t-PA → in selected cases


Dead
Tissue
Penumbra
RS

Acute Ischemic stroke treatment using 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

THROMBOLYTICS: IV-TPA

Original NINDS trial:

- Absolute difference in favorable outcome of tPA versus placebo was 11-13% across the scales
- Depending upon the scale, the increase in relative frequency of favorable outcome in patients receiving tPA ranged from 33% to 55%.
- The effect of tPA was independent of stroke subtype, with beneficial effects seen in those with small vessel occlusive, large vessel occlusive and cardio-embolic induced ischemia.

IV –tPA side effects

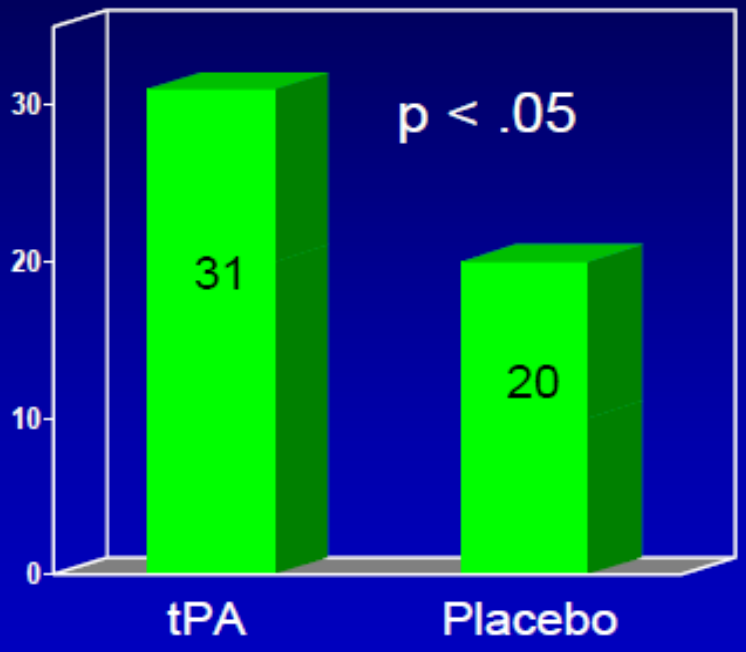
- 6% develop **symptomatic intracerebral hemorrhage** within 36 hours following treatment (0.6% in placebo group).

- 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.

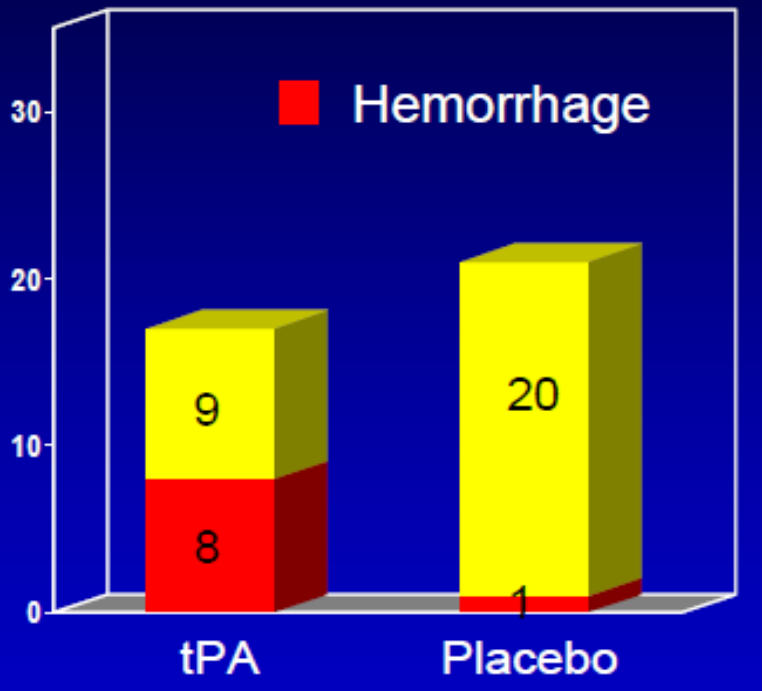
Facial angioedema : another side effect which may cause airway obstruction.

New England Journal, 1995

NINDS tPA Stroke Trial



NIHSS Excellent Recovery (%)



Total Death Rate (%)

THROMBOLYTICS: IA-TPA

IA thrombolysis is an option for treatment of selected patients who have major stroke of <6 hours' duration due to occlusion of MCA, and who are not otherwise candidates for IV-TPA.

IA thrombolysis is reasonable in patients who have contraindication to use of IV-TPA, such as recent surgery (**new recommendation).**

Tx requires pt to be at experienced stroke center with immediate access to cerebral angiography and qualified interventionalists (**new recommendation).**

Mechanical Disruption:

MERCI device is reasonable intervention for extraction of intra-arterial thrombi in carefully selected patients, but panel recognizes that utility of device in improving outcomes after stroke is unclear (**new recommendation).**

Anticoagulation in Acute Stroke !!

Urgent anticoagulation with goal of preventing early recurrent stroke, halting neurological worsening, or improving outcomes after acute ischemic stroke not recommended.

Urgent anticoagulation not recommended for pts with moderate to severe strokes because of increased risk of serious ICH complications.

Initiation of anticoagulant tx within 24 hours of IV-TPA not recommended.

Antiplatelet Rx:

Class I recommendation:

1. 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

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

Stroke Work-up (after acute stroke Mx)

- Fasting blood glucose and lipid profile
- Carotid U/S
  ➔ 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

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

- <u>Prognosis:</u>

  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)*

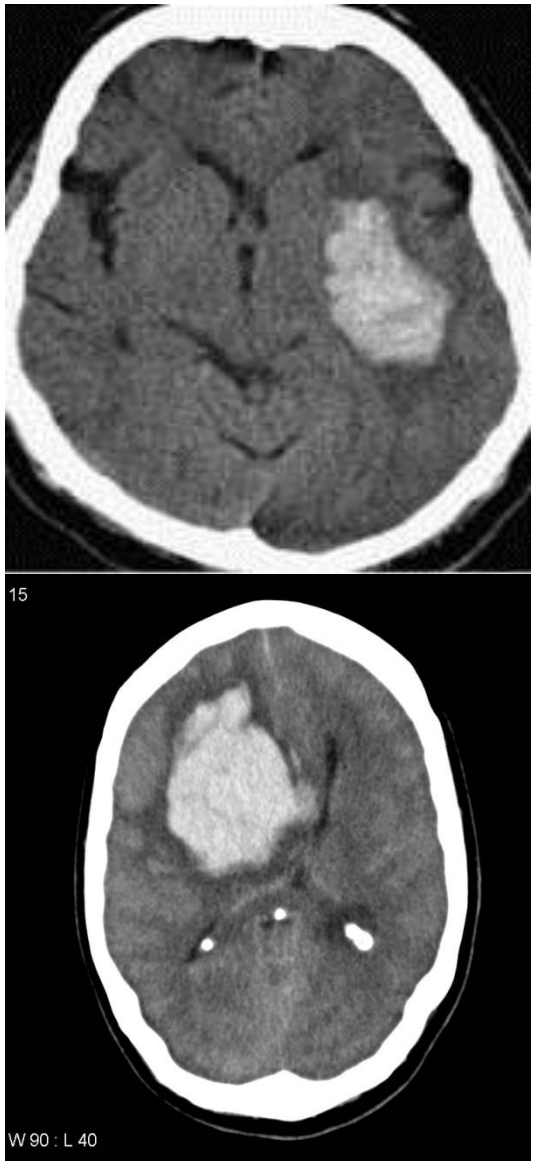
Intracranial Hemorrhage

Common causes

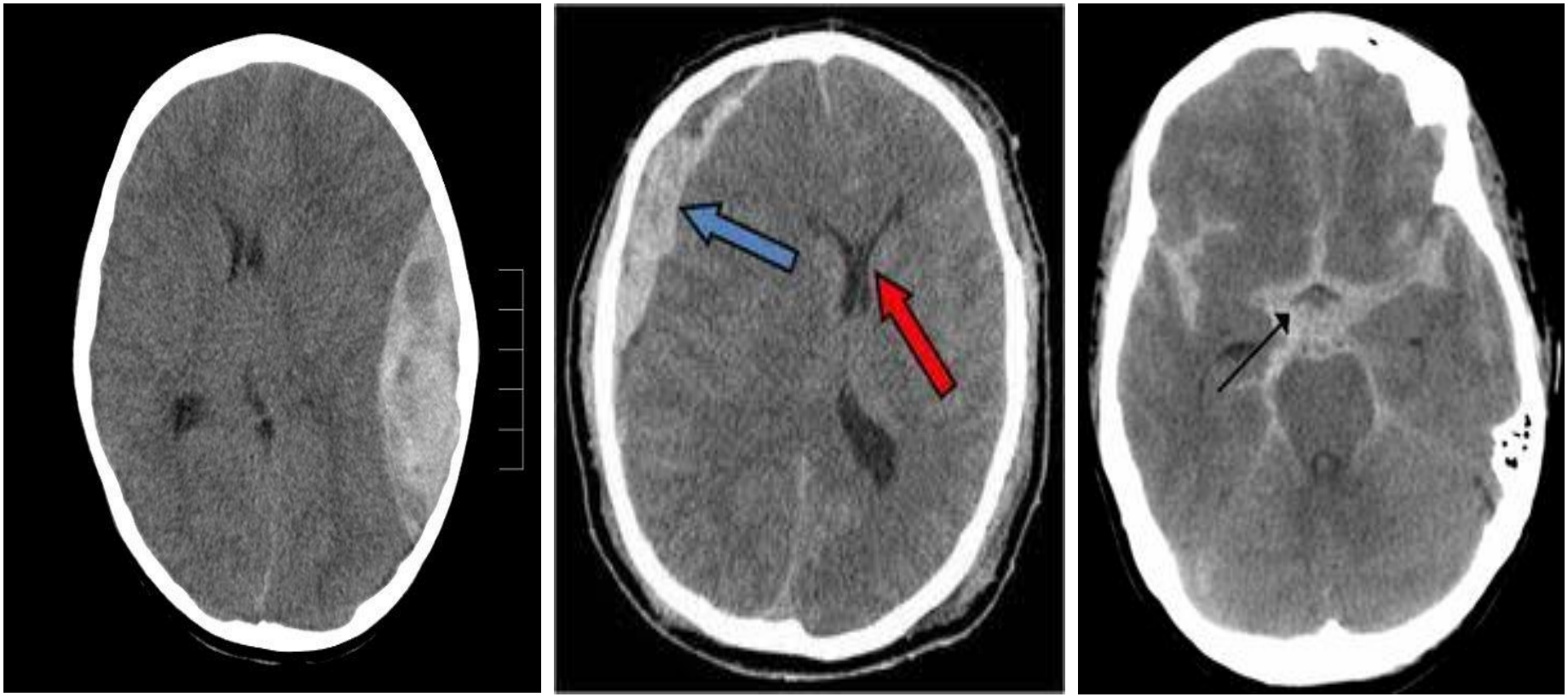
1. Hypertension
2. Trauma
3. Amyloid angiopathy.
4. Ruptured vascular malformation.
5. Coagulopathy (a disease or drug-induced)
6. Hemorrhage into a tumor .
7. Venous infarction.
8. Drug abuse.

HTN- Induced IC hge

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

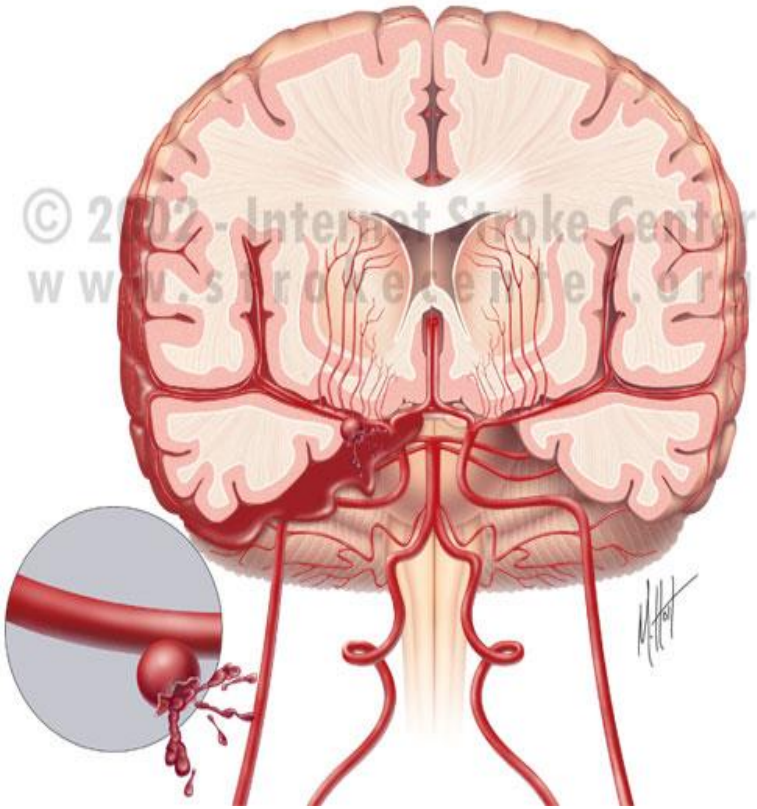


Traumatic Intracranial Hge



Subarachnoid Hge

- Worst headache ever
- Use: H&H scale
- Spont. Vs. traumatic
- Risk of aneurysms increase with smoking (X40 times)
- 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

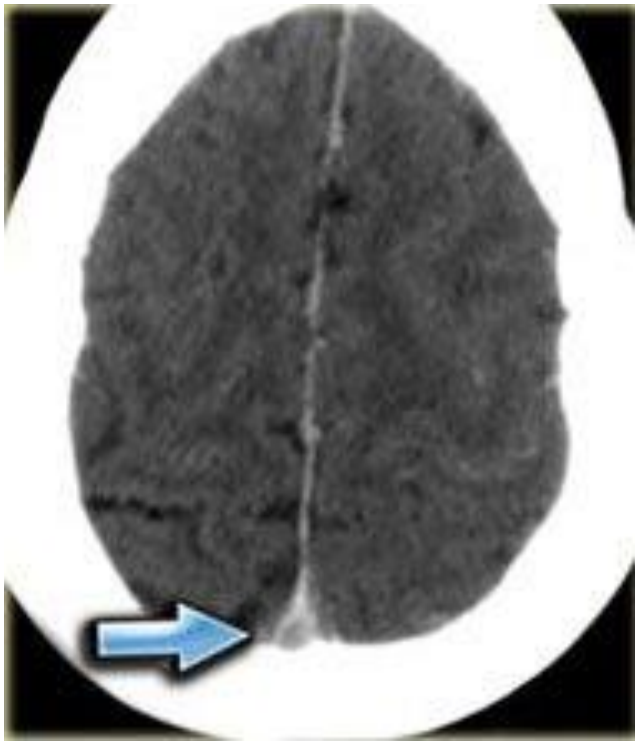


Case-3

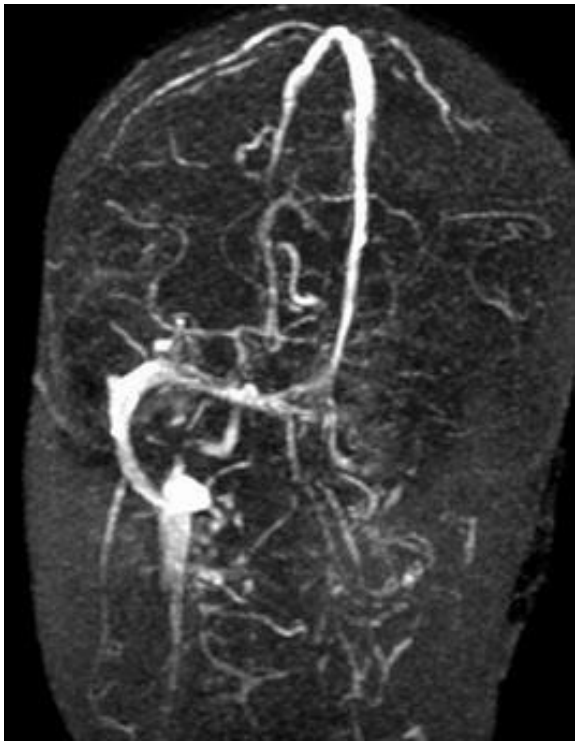
- 30 year old lady in postpartum developed severe diffuse headache and blurred vision for about 1 day. Clinical exam showed papilledema bilaterally

- DDx??
- Approach??
- Management??

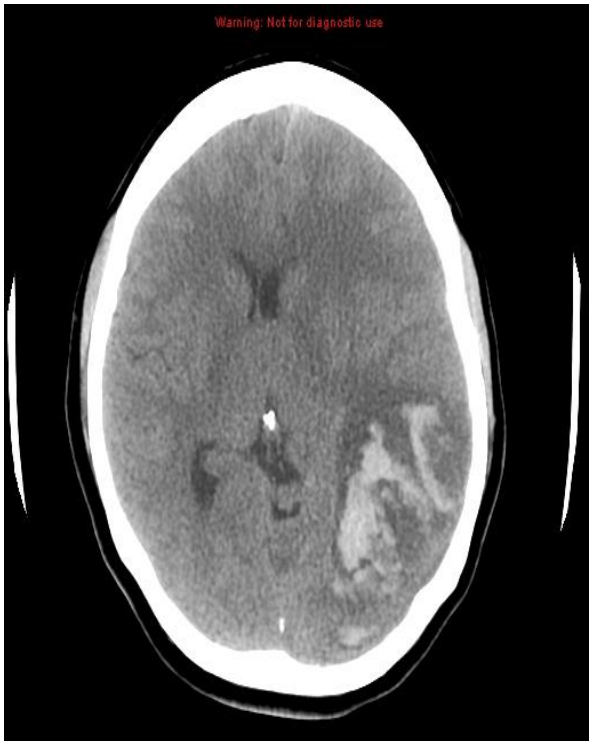
Cerebral Venous Sinus Thrombosis



Empty delta sign
CT (brain) with contrast



MRV



Venous Hge in CT(brain)

Case-3

- 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

STROKE WON THE WAR