CEREBROVASCULAR DISEASE

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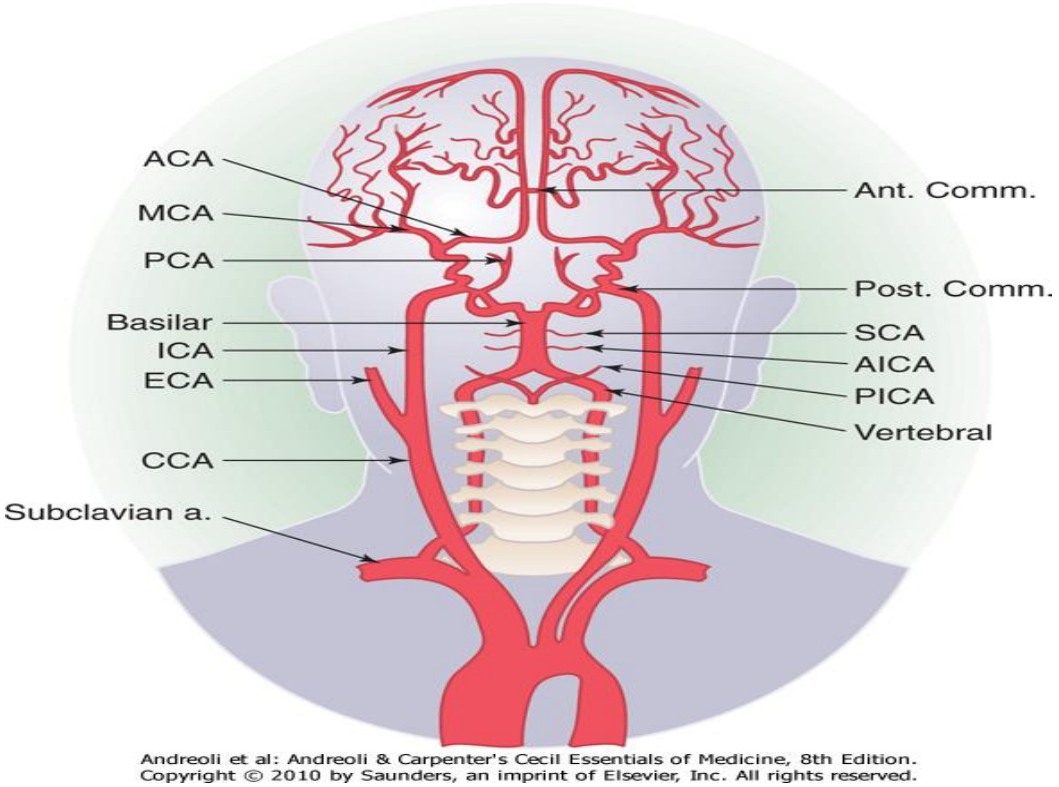
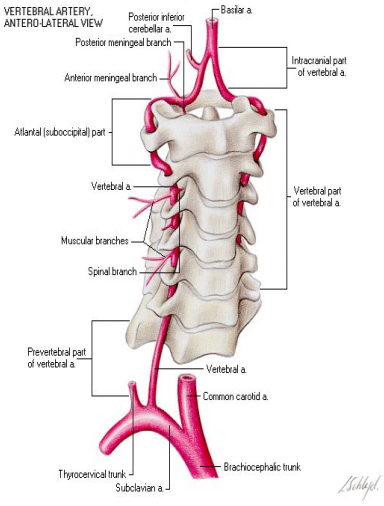
429 MEDICINE TEAM



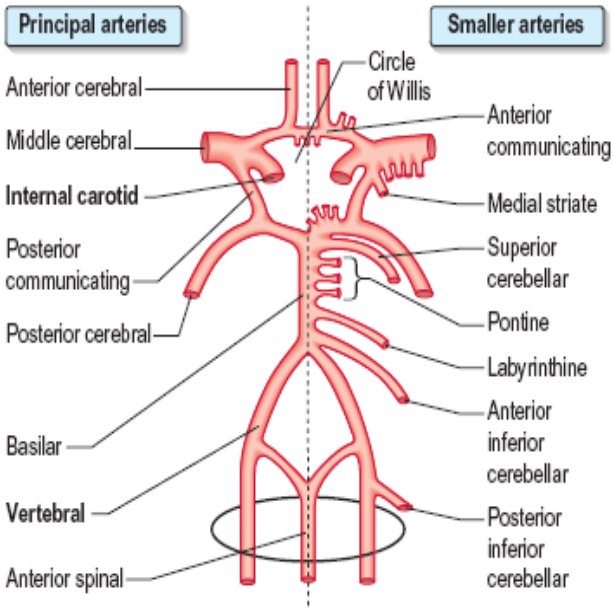
Sources: Slide show, lecture recording, Step up, Andreoli's Cecil and 427 booklets.

ANATOMY:

- Two pairs of major arteries, the carotid (anterior circulation) and vertebral (posterior circulation) arteries, supply the brain
- Anterior circulation:
  - The common carotid artery bifurcates into an internal and an external branch at the level of the thyroid cartilage in the neck(C4)
  - The ICA (internal carotid artery) enters the skull through the carotid canal and passes through the cavernous sinus
  - once the ICA pierces the Dura it gives off 5 branches:
    - ophthalmic
    - posterior communicating
    - anterior choroidal
    - anterior cerebral artery (ACA)
    - middle cerebral artery (MCA) which also gives lacunstraite branches to internal capsule

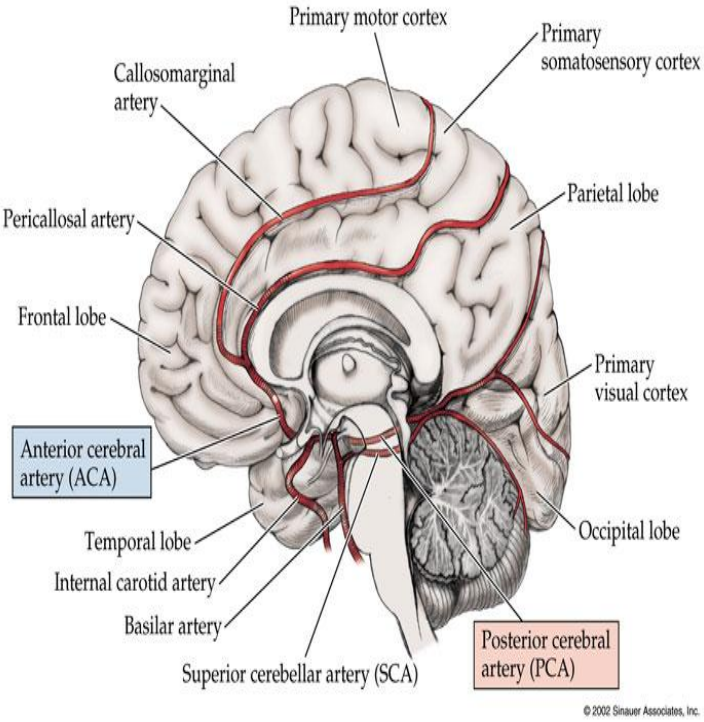


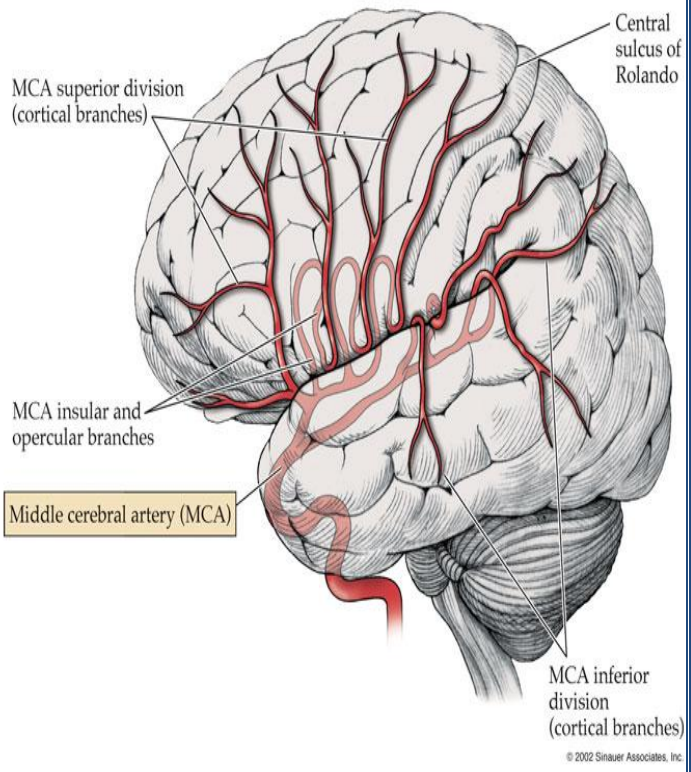
- posterior circulation:
  - The vertebral arteries (VAs) arise from the subclavian arteries and course upward within the transverse processes of the cervical vertebrae
  - They pierce the Dura at the level of C1, C2
  - The first intracranial branch of the VA is the posterior inferior cerebellar artery (PICA), (which supplies the posterolateral medulla, inferior vermis, choroid plexus of the fourth ventricle, and inferior cerebellum)
  - The two VAs unite to form the basilar artery (BA) at the pontomedullary junction
  - bifurcates into the posterior cerebral arteries (PCAs) at the level of the pontomesencephalic junction
- circle of Willis:
  - complete only in 20% of people
  - formed at the base of the brain by the union of both ACAs via the anterior communicating artery and the union of the ICA with the PCA via the posterior communicating artery
  - Hence communication exists between both anterior circulations, as well as between the posterior and anterior circulations on each side

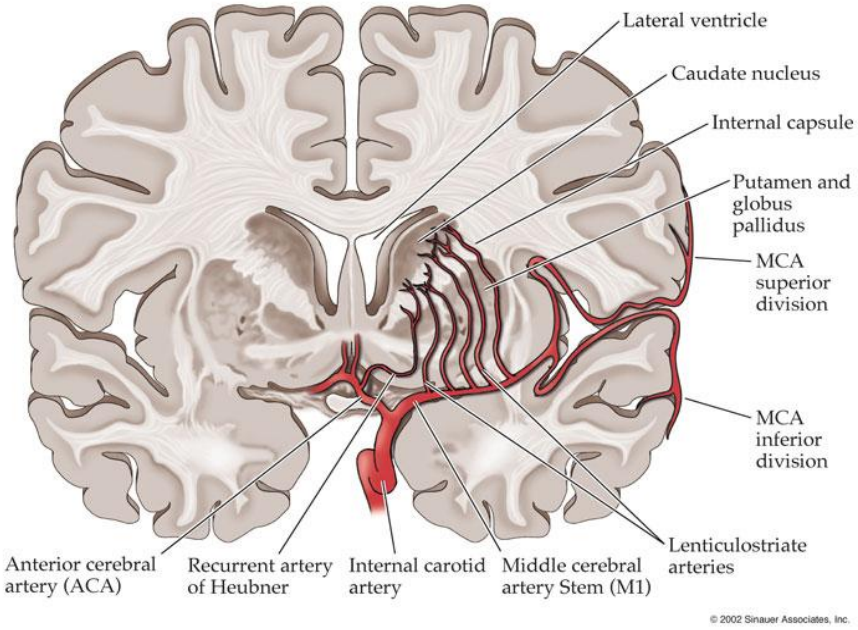


- Blood supply
  - ACA (frontal and parietal)
    - Motor homunculus and sensory homunculus are supplied medially by the ACA meaning it supplies the leg area (so remember that weakness in the leg, think of ACA stroke) also frontal lobe symptoms like personality changes, lack of initiation, depression, emotional changes, etc..
  - MCA: lateral surface (convexity), in addition to much of the basal ganglia and subcortical white matter
    - Patient would have aphasia, weakness in contralateral face, arm and leg.
    - MCA also supplies the internal capsule so it might affect the leg.

- PCA: medial part of the cerebral hemispheres posteriorly (occipital) :Contralateral homonymous hemianopsia







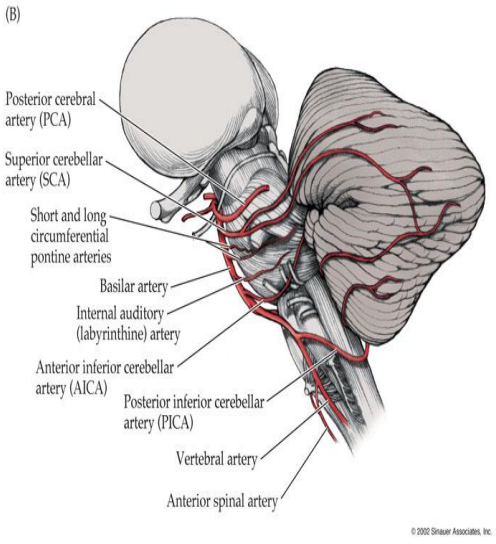
COMMON ACUTE STROKE PRESENTATION ACCORDING TO ARTERIAL SUPPLY (CHECK FIGURES AT THE END):

ACA

- weakness and sensory loss in the contralateral leg (hemiparesis and hemisensory loss)
- abulia: frontal lobe symptoms such as behavioural abnormalities
- large infarct can cause contralateral hemiplegia
- transcortical aphasia can be seen
- Grasp reflex

MCA

- Main MCA trunk:
  - Contralateral hemiplegia, hemianesthesia, and homonymous hemianopsia with a gaze preference away from the side of the hemiplegia (to the site of infarct).
  - The hemiplegia usually affects the face and arm more than the leg.
    - But it can affect the leg because it supplies the internal capsule
  - A global aphasia occurs with dominant hemisphere lesions
  - anosognosia occurs with nondominant hemisphere involvement
- Superior division of the MCA
  - Left MCA:
    - Broca's aphasia
    - Contralateral weakness in the face arm and leg
  - Right MCA:
    - Contralateral weakness in the face arm and leg
    - Left hemi-neglect
- Inferior division of the MCA
  - Wernicke's aphasia
  - Contralateral weakness in the face arm and leg
  - Visual disorientation (because of visual memory)
  - Short term memory loss



PCA:

- Proximal PCA stroke will mimic MCA stroke if it infarct at the site of crus cerebri at the brain stem
- Contralateral homonymous hemianopsia

BRAIN STEM STROKE:

- Inability to stand & Ataxia
- Sensorimotor deficits - Ipsilateral face and contralateral limbs (crossing sign)
- Diplopia/ dysconjugate gaze, ocular palsy homonymous hemianopsia
- Bilateral limb weakness and/or numbness
- Sudden LOC
- Vertigo
- Dysarthria

BASILAR ARTERY:

- Misdiagnosed sometimes as epilepsy because it can cause seizures,
- also can cause (Quadriparesis, Dysarthria, dysphagia, Diplopia, somnolence, amnesia)
- *locked-in syndrome,* in which patients are fully awake yet quadriplegic and can communicate only by means of vertical eye movements and blinking

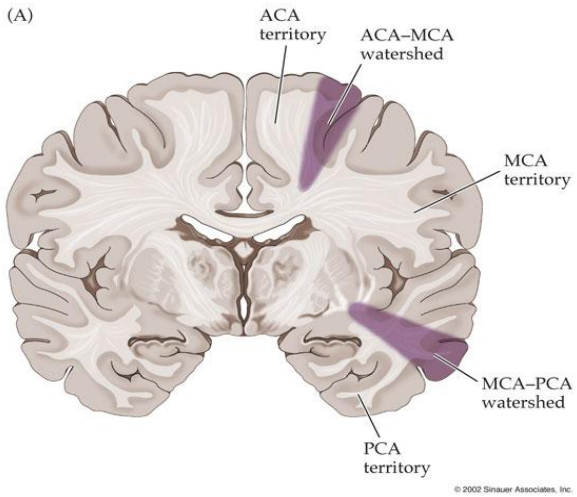
SUPERIOR CEREBELLAR ARTERY: **IPSILATERAL ATAXIA, NAUSEA, VERTIGO, DYSARTHRIA**

WALLENBERG (LATERAL MEDULLARY SYNDROME)

- Infarct to Vertebral artery or PICA (posterior inferior cerebellar artery)
- Spinothalamic tract infarct will cause the symptoms
- Symptoms: nausea, vertigo, Dysarthria, **dysphagia,** Ataxia.
- Signs: Ipsilateral facial sensory loss, hemi ataxia, nystagmus, Horner syndrome, Contralateral loss of temperature or pain sensation

LACUNAR STROKE

- Motor
  - Weakness face, arm, and leg contralateral (hemiparesis)
  - Lesion: Posterior limb of internal capsule or basis pontis (anterior Pons)
- Sensory: Lesion in Thalamus
- Motor and sensory: lesion in the thalamus and posterior limb of internal capsule
- Ataxic hemiparesis:
  - weakness and ataxia contralateral
  - lesion in Posterior limb of internal capsule
- Dysarthria-clumsy hand syndrome
  - ataxia in the hand and Dysarthria
  - lesion in the Pons



WATERSHED STROKE:

- areas that have less blood supply than others and usually at areas of distal anastomses are vulnerable in hypotension (cardiac arrest, surgery) or carotid stenosis

STROKE

EPIDEMIOLOGY

- The 3rd most common cause of death
- The 1st cause of permanent disability in western societies
- In the USA:
  - Incidence: 500000/year
  - Prevalence: 3 million survivors
  - Mortality: 150,000/ year
  - Cost: 16 billion dollars
- KSA:
  - Incidence 118/100,000
  - Prevalence: 178/100,000
  - 30-40 thousand new cases per year
  - 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
- Lacunar strokes makes near 50 % because of prevalent diabetes
- Stroke is a **preventable & predictable** disease ➔ never use the word: ACCIDENT

RISK FACTORS:

NON-MODIFIABLE:

- Age – most important
- Family history
- Genetic
- Congenital abnormality (heart, AVM in CNS)
- Hypercoagulable states

MODIFIABLE:

- HTN – most important modifiable
- Diabetes
- Cardiac disease
- Atrial fibrillation
- Carotid artery disease
- Physical inactivity
- Obstructive sleep apnea
- Smoking
- Substance abuse
- Medications (OCP)
- Dissection

TERMS:

1. TIA:

- A neurological deficit that lasts from a few minutes to no more than 24 hours
  - The 2002 definition is "A transient ischemic attack (TIA) is a brief episode of neurologic dysfunction caused by focal brain or retinal ischemia, with clinical symptoms typically lasting less than 1 hour" *doctor says this is the right one*!!
  - The duration of symptoms distinguishes it from stroke (>24 hours: stroke; <24 hours: TIA)
  - Symptoms are transient because reperfusion occurs
  - The blockage in the blood flow does not last long enough to cause permanent infarction > no evidence of infarction
  - Aetiology:
    - usually embolic
    - Hypotension with carotid stenosis
    - Thrombotic
  - Clinical significance IMP:
    - Once a patient has a TIA there is a high risk for strokes
    - 20% of TIA pts will have stroke within 3 months > alarm for stroke
    - The risk per year for a patient with TIA is 10%/year
    - And it carries a 30% 5 year risk of stroke

2. EVOLVING STROKE: A STROKE THAT IS WORSENING

3. COMPLETED STROKE: THE MAXIMAL DEFICIT HAS OCCURRED

CLINICAL TYPES OF STROKE

ISCHEMIC (85% OF CASES) :

- Thrombotic (40%)
  - Clinical features:
    - sudden, gradual or stepwise
    - classically the patient wakes up from sleep with the neurological deficit
    - Prior TIA in the same vascular distribution.
    - Concurrent coronary or peripheral artery disease
  - Causes:
    - atherosclerotic lesions (atheroma)
      - Internal carotid and MCA are most common
      - Concomitant with HTN, DM and smoking
    - Arteritis
    - Haematological disorders
- Embolic (25%)
  - Clinical features:
    - Symptoms are very rapid and deficits reach a maximum level initially
    - Recent TIA/stroke in other vascular territory
    - Sometimes Evidence of systematic embolization
  - Causes (origin of embolus) :

        - Heart
            - Most common
            - Mural thrombus in patients with Atrial fibrillation
            - Septic embolus from Infective endocarditis
            - Paradoxical embolism with congenital heart disease (clots in veins then pass through septal defects)
        - Atheroma of Aorta and ICA
        - Pulmonary vein thrombus
        - Complication of neck surgery
- Lacunar (30%)
    - Clinical features:
        - Causes 20%-30% of all strokes
        - Abrupt or gradual in onset
        - Infarcts <1.5 cm due to occlusion of small arteries 50- 200 µm
        - Risk factors: Hypertension (65 – 80%), DM IMP!!
        - Prior TIA in 20%
        - Relatively slow (sudden onset in 40 %)
        - No cortical findings, mainly subcortical! IMP
        - Consciousness is preserved and symptoms are highly focal
        - depends on the part affected but the most common is pure motor
    - Causes:
        - A history of HTN is found in 90% of patients
        - DM is an important risk factor
        - Narrowing of the blood vessel is due to thickening and not thrombosis
- Hypoperfusion : watershed ischemia

HEMORRHAGIC (15%):

- Causes of hemorrhagic strokes:
    - Vascular anomaly : aneurysm, angioma
    - Hypertension
    - Trauma
    - AV malformation
- Types:
    - Epidural:
        - from the middle meningeal artery
        - Lens like appearance on CT
    - Subdural : Crescent shape on CT
    - Subarachnoid
        - Mortality 40-50% in 30 days
        - Most common cause is ruptures berry aneurysm
    - Intra-cerebral
    - Intra-ventricular

VENOUS

- Venous sinus thrombosis : sudden headache, blurred vision , seizures
- Cortical vein thrombosis : severe headache and seizure

DIAGNOSIS

- On the way to the hospital (ambulance):
  - Manage ABCs
  - Cardiac monitoring
  - Intravenous access
  - Oxygen (keep O2 sat >92%)
  - Assess for hypoglycemia (gives stroke like presentation)
  - NPO
  - Alert receiving ED
  - Rapid transport to closest appropriate facility capable of treating acute stroke
- Detailed and accurate History and physical examination for localization
- Lab:
  - CBS, electrolytes, coagulation profile
  - 12 lead ECG and cardiac enzymes
- Imaging:
  - CT scan:
    - Differentiates between ischemic and hemorrhagic stroke IMP! (most important benefit from CT
    - First imaging study that should be ordered
    - No contrast
    - If you see an infarct that means it is irreversible because it takes 24-48 hours to see, so CT is useful at ruling haemorrhage
    - Identifies 95% of subarachnoid haemorrhage, tumours, abscesses, and hematomas
    - Findings in stroke:
      - Hypo-attenuation of brain tissues
      - Loss of sulcal effacement
      - Insular ribbon sign
      - Obscuration of lentiform nucleus
      - Hyperdense sign (MCA>basilar>PCA)
  - MRI:
    - More sensitive than CT
    - Identifies all infarcts before CT
    - Not preferred in ER because it takes longer time
  - Carotid duplex for carotid stenosis
    - Patients that have the following should be screened with duplex: carotid bruit, peripheral vascular disease, Coronary artery disease
  - MRA (magnetic resonance angiogram): identifies stenosis in the vessels of head and neck
- ECG – acute MI or A-fib may be the cause of stroke

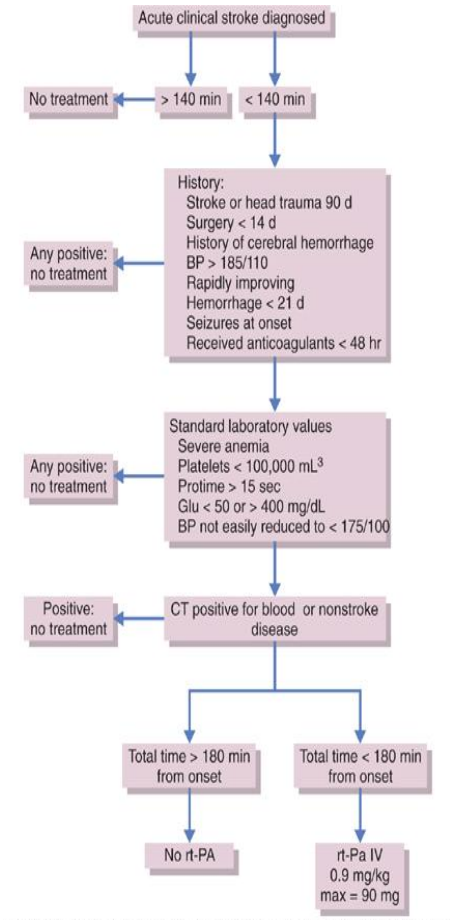
COMPLICATIONS

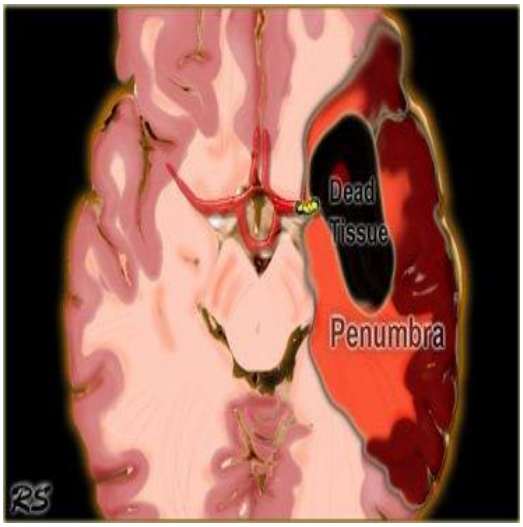
- Progression of neurological insult
- Cerebral edema within 1-2 days and can cause mass effect for up to 10 days
- Hemorrhage into the infarction – rare
- Seizures in fewer than 5% of patients

TREATMENT

ACUTE:

- ABC
- Thrombolytic therapy (t-PA):
  - If IV t-PA administered within 3 hours of the onset of an acute ischemic stroke the outcome improves at 3 months (NINDS trial)
  - Hyperdense sign in a patient makes him the best candidate for t-PA
  - Contraindications:
    - Time of stroke unknown or more than 4.5 hours
    - Uncontrolled HTN
    - Bleeding disorder or recent haemorrhage
    - 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
  - Target of t-PA: salvage the penumbra tissues (at risk)
  - Only TPA approved for ischemic stroke if given within 4.5 hours of stroke onset
  - Options : IV t-PA, IA t-PA, IV followed by IA
  - Mortality:
    - without thrombolysis 10-15%
    - With thrombolysis 3%
  - Intracerebral haemorrhage within 3 months:
    - Thrombolysis group 9%
    - Non thrombolysis group 15%
  - Side effects:
    - 6% develop **symptomatic intracerebral hemorrhage** within 36 hours following treatment
    - Half of the t-PA associated symptomatic hemorrhages were fatal
    - **Facial angioedema :** another side effect which may cause airway obstruction
  - **IA t-**PA (intra-arterial thrombolysis)
    - Option for treatment of selected patients who have major stroke of <6 hours' duration due to occlusion of MCA and basilar strokes!
    - reasonable in patients who have contraindication to use of IV-TPA, such as recent surgery (**new recommendation)**
    - requires pt to be at experienced stroke center with immediate access to cerebral angiography and qualified interventionists (**new recommendation)**
- Anticoagulants
  - Not been proven to have efficacy in acute stroke





    - not recommended for pts with moderate to severe strokes because of increased risk of serious ICH complications
- Antiplatelets:
    - administer within 24 -48 hours
    - do not give to patients who took t-PA due to increased risk of ICH (intracerebral hemorrhage)
- Endovascular and mechanical disruptions (catheter) : 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)**

BLOOD PRESSURE MANAGEMENTS

- In general only given in these three conditions:
    - Very high BP : 220/120
    - Medical indication: MI, Aortic dissection, heart failure, hypertensive encephalopathy
    - The patient is receiving thrombolytic therapy – blood pressure control is required to reduce risk of bleeding
- Use labetalol IV 10 mg or hydralazine and avoid vasodilators

FOLLOW UP WITH

- 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
- In selected cases: MRI/MRA, CT, screen for blood disorders

SECONDARY PREVENTION

- Prevention due to atherosclerosis:
    - Control risk factors (DM, HTN, smoking: risk disappears in 5 years, Obesity, hypercholesterol with statin (LDL: 1.3 – 2))
    - Aspirin
    - Surgery: carotid endartectomy
        - Symptomatic patients: benefit is >70%
        - Asymptomatic patients: reduce risk factors and use aspirin
- Prevention due to embolic disease:
    - For example A-fib
    - Anticoagulation and aspirin
    - Reduce atherosclerotic risk factors
- Control hypertension to prevent Lacunar strokes

POST STROKE CARE:

- Maximize secondary stroke prevention
- Rehabilitation (motor, language, behavioral,)
- Special care for swallowing and DVT prophylaxis
- Most limiting factors for rehab are:
    - Vascular dementia
    - 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:

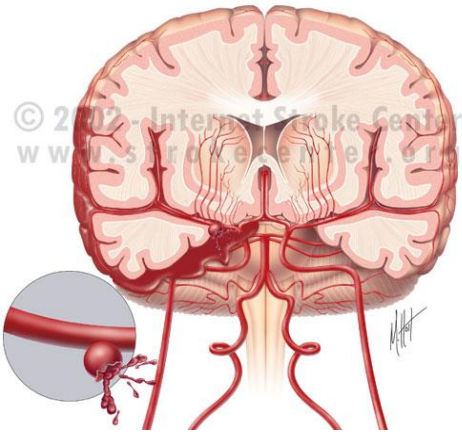
- Hypertension – most important
- Trauma
- Amyloid angiopathy.
- Ruptured vascular malformation.
- Coagulopathy (a disease or drug-induced)
- Hemorrhage into a tumor.
- Venous infarction.
- Drug abuse.

INTRACEREBRAL HAEMORRHAGE:

- High mortality (50% in 30 days)
- HTN is the main cause (ruptures small vessels/ seen in older ages)
- Location: basal ganglia (66%), Pons (10%), cerebellum (10%), other
- Features:
    - Abrupt onset of focal deficit that worsens steadily
    - Altered LOC, stupor or coma
    - N & V and raised ICP
- CT diagnosis in 95% of cases
- Complications: raised ICP, seizures, rebleed, vasospasm, SIADH
- Rx: ICU, ABC, Lower BP gradually with nitroprusside, mannitol to reduce ICP

SUBARACHNOID HAEMORRHAGE:

- Mortality is 40-50% in 30 days
- Causes:
    - Ruptured berry aneurysm is the most common cause
        - Sacular aneurysms are more in anterior circulation (90%) while fusiform more in basilar
        - Risk of aneurysms increase with smoking (X40 times)
    - Trauma
    - AV malformation
- Clinically:
    - Presents as the worst headache ever
    - LOC, Vomiting
    - Meningeal irritation, Nuchal rigidity, photophobia
    - Death
    - Retinal haemorrhage
- Diagnosis
    - Non contrast CT

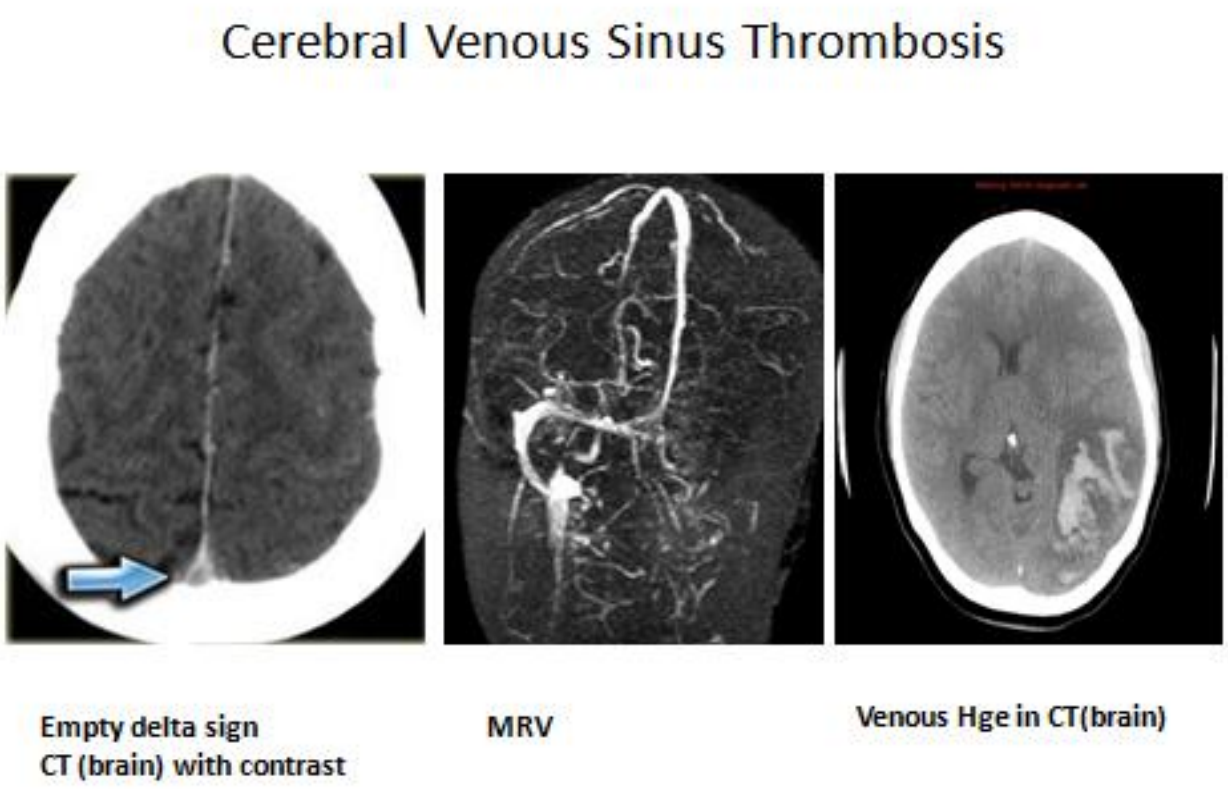


- LP if CT is negative with high clinical suspicion
  - Blood in CSF
  - Xanthochormia (yellow CSF due to RBC lysis) – gold standard
- Cerebral angio for location of bleed for surgery

- Treatment: neurosurgery consultation for clipping or coiling of berry aneurysm

CEREBRAL VENOUS SINUS THROMBOSIS

- Case: 30 year old lady in postpartum developed severe diffuse headache and blurred vision for about 1 day. Clinical exam showed papilledema bilaterally
  - Did? Cerebral venous sinus thrombosis
  - Approach? Imaging (brain MRV or CTV) / high opening pressure in LP
  - Management? Manage with anticoagulation (this is the only bleeding in medicine that is treated with heparin)



THE FOLLOWING PAGES HAVE THE FIGURES.

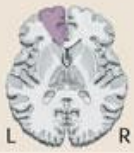
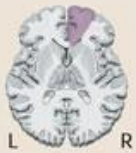
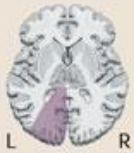
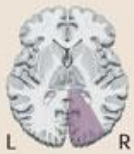
TABLE 10.1 Major Clinical Syndromes of the MCA, ACA, and PCA Territories *(Part 1)*

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.