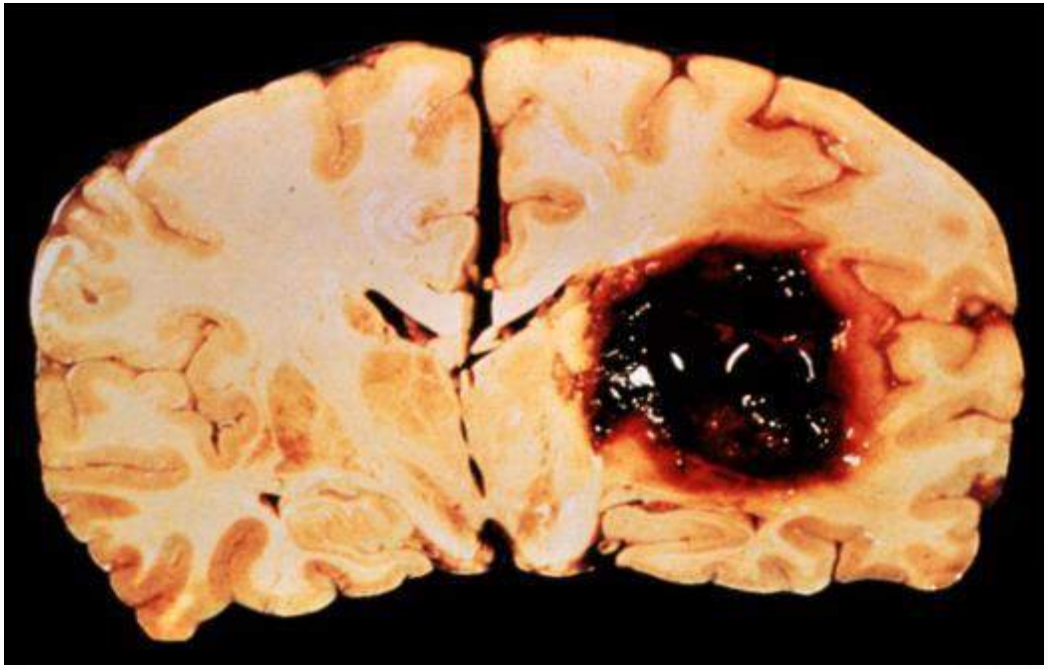
HEMORRHAGIC STROKE



Dr. Claire Karekezi

Worldwide statistics

- WHO: **15 million** people suffer a stroke each year;
- **5 million die**, about **5 million** permanently disabled.

- High blood pressure contributes to over **12.7 million strokes** worldwide

- Risk of stroke in blacks: almost **twice** that of whites

- In HIC incidence of stroke declining due to efforts to lower blood pressure and reduce smoking

- Overall rate of stroke high due to the **aging** of the population

ICH by numbers

- Result of a rupture of blood vessel in the brain
- Accounts for **10-15%** of all cerebrovascular accidents
- **2 million** strokes every year worldwide
- Rise of admissions in the past 10 years by **18%**
- Prognosis is poor: estimated mortality
  - **30% at 7 days**
  - **60% at 1 year**
  - **82% at 10 years**
  - **>90% at 16 years**

RISK FACTORS

NON-MODIFIABLE

- Age
- Sex
- Race
  - Asians > Afr. Amer. > White
- Genetics:
  - Cerebral amyloid angiopathy, coagulation disorders

MODIFIABLE

- HTN
- Cerebral amyloid angiopathy
- Cholesterol
- Anti-coagulation
- Anti-platelets
- High EtOH intake
- Smoking
- DM
- Microbleeds
- Dialysis
- Drug-Induced (e.g. cocaine, amphetamines)

Mechanisms

Traumatic	Non-traumatic
Head injury	Uncontrolled hypertension
	Anticoagulant therapy
	Platelet and coagulation disorders
	Vascular malformations
	Brain tumors
	Cerebral amyloid angiopathy
	Drug-induced: cocaine, amphetamines

Hypertension and ICH

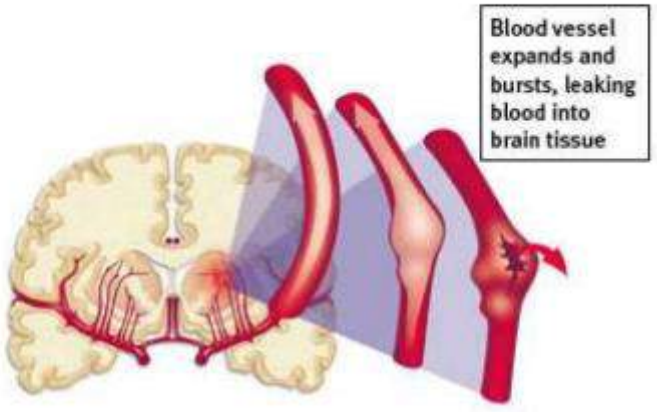
- **Most important risk factor (>70% of 1ry ICH)**
- Bifurcation of small penetrating arteries (50–700 μm diameter)
- Atherosclerosis
  - Lipid deposition, layering of platelet and fibrin aggregates, breakage of elastic lamina, atrophy and fragmentation of smooth muscle, dissections, and granular or vesicular cellular degeneration
- Charcot and Bouchard aneurysm
  - Fibrinoid necrosis of the subendothelium → focal dilatations → rupture of microaneurysm

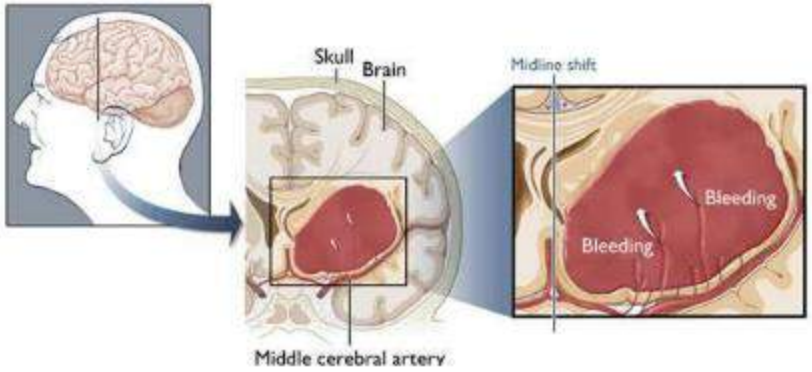
PHYSIOPATHOLOGY

Usually occurs spontaneously
Caused by vascular rupture with bleeding into brain

- Mass effect can further cause bleeding and hematoma expansion from neighboring vessels

Hematoma growth over several hours following presentation of symptoms is common (30-40%)
Hemorrhages commonly occurs at the basal ganglia, thalamus, pons, or cerebellum





Chronic hypertension → structural wall changes of small arteries and arterioles in the brain

- Fibrinoid necrosis
- Charcot-Bouchard aneurysms

Idiopathic hypertension (acute) → usually younger patients with history of drug abuse

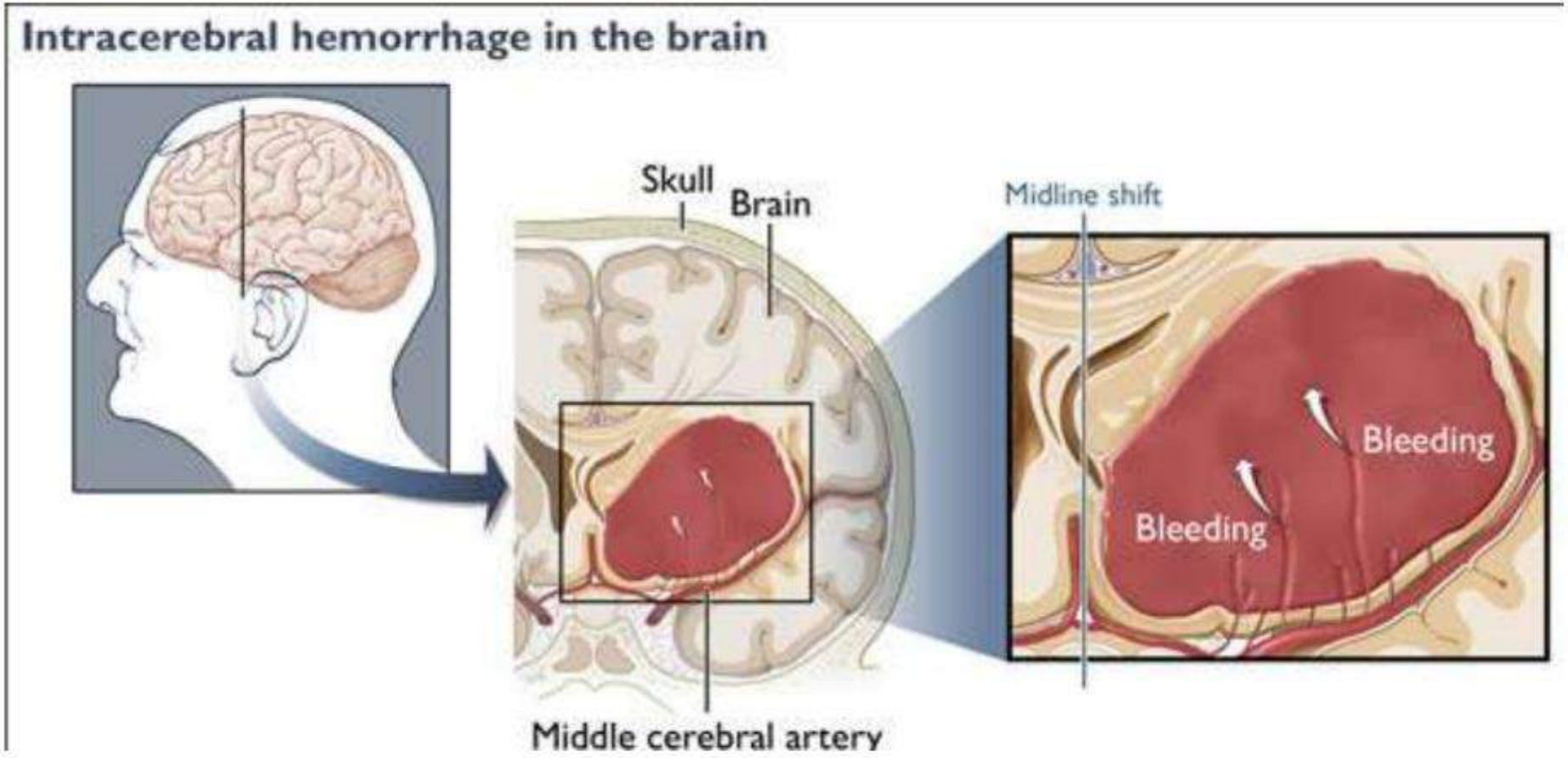
- Amphetamine, cocaine
- May occur minutes to hours after drug use

Vascular malformations

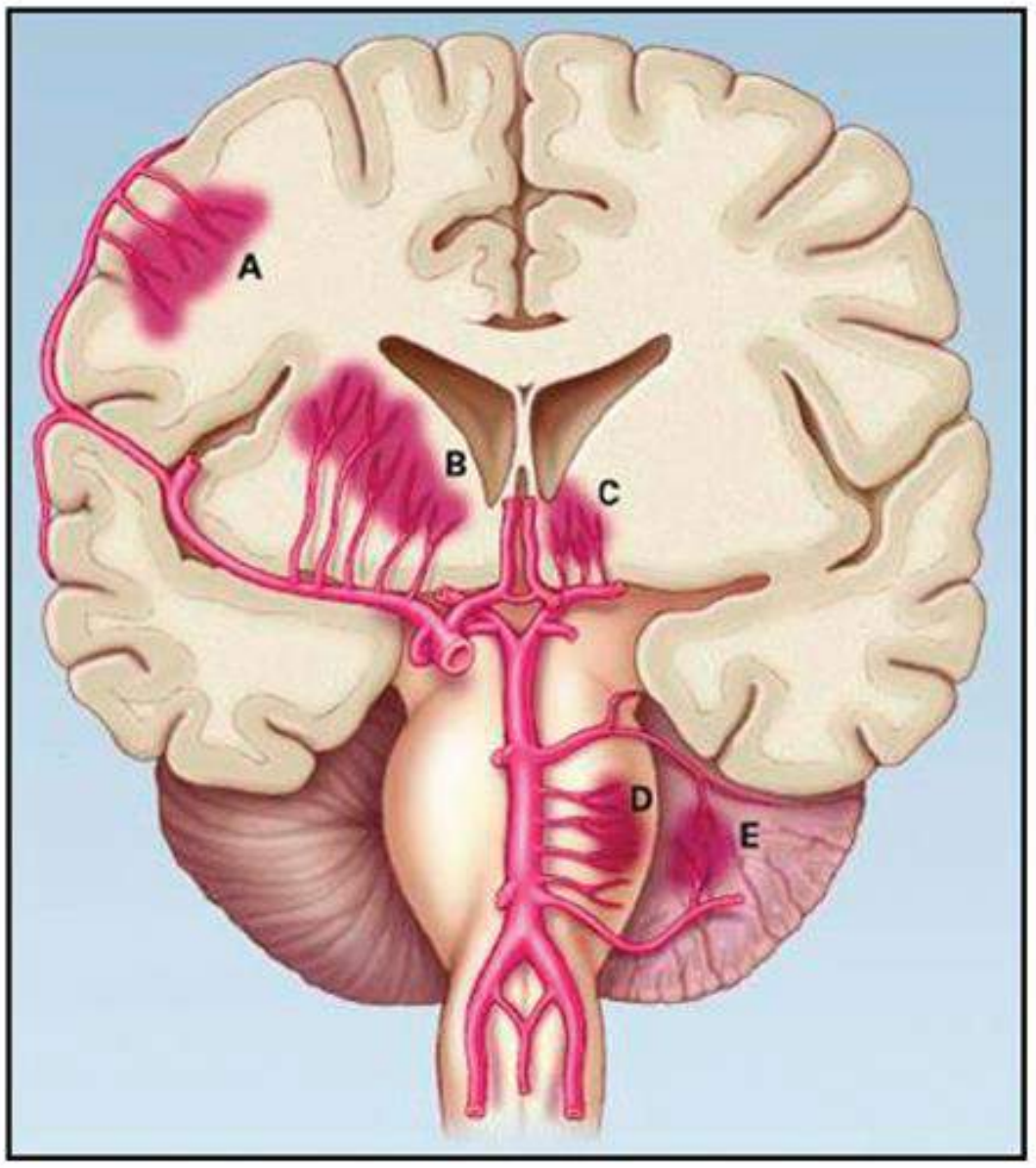
- Arteriovenous malformations (AVM): failure of formation of capillary beds
- Saccular (berry): results from developmental weakness of arteriole walls

Hemorrhages can cause compression to nearby brain tissues

- May result in brain tissue inflammation and edema


Intracerebral hemorrhage in the brain
Skull
Brain
Midline shift
Bleeding
Bleeding
Middle cerebral artery

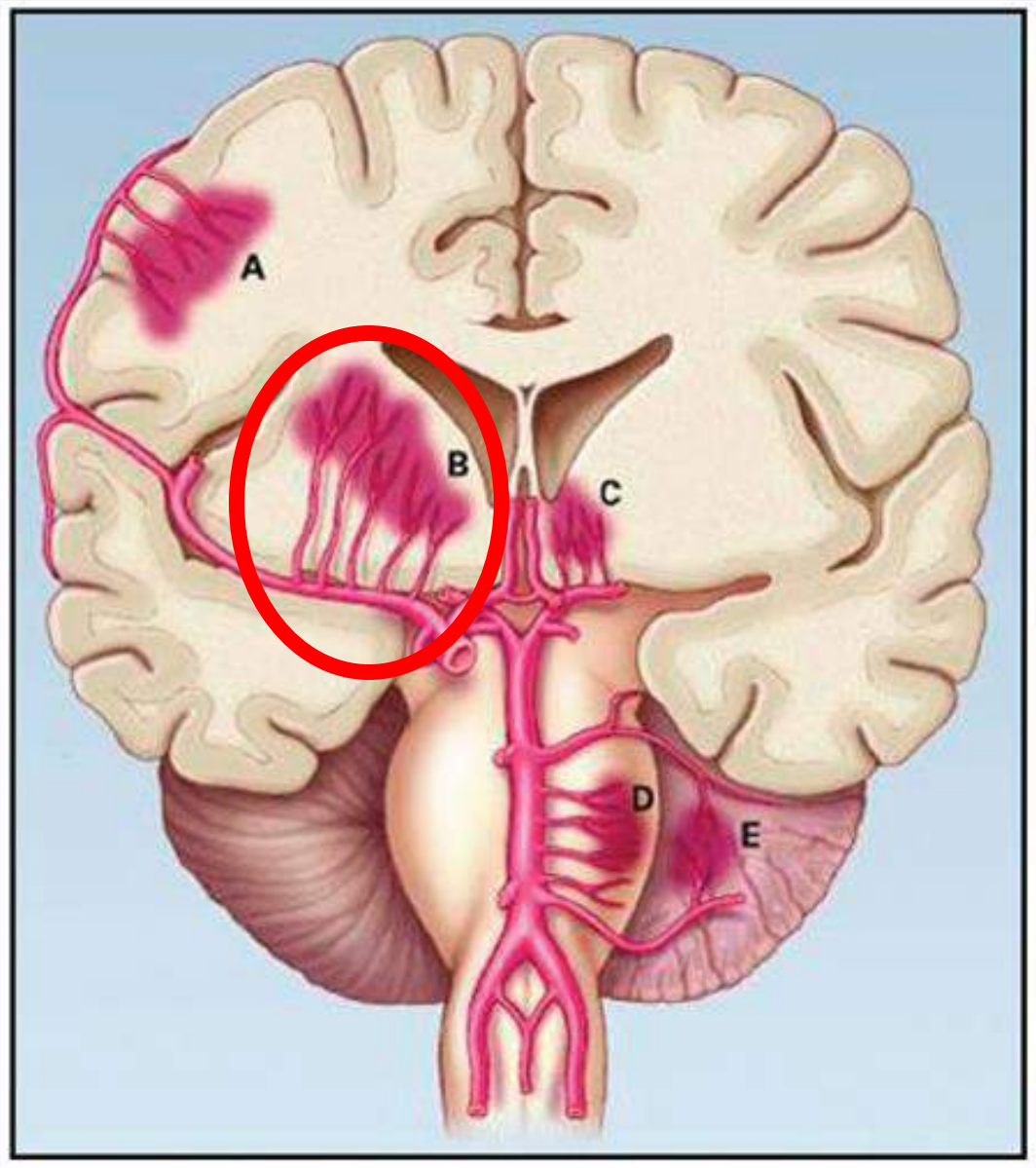
SITES



N Engl J Med 2001;344(19):1450–1460

Basal ganglia 35-40%

- Ascending lenticulostriate branches of MCA
- Wide spectrum of severity extending to coma and decerebrate rigidity
- Ventricular extension carries very poor prognosis



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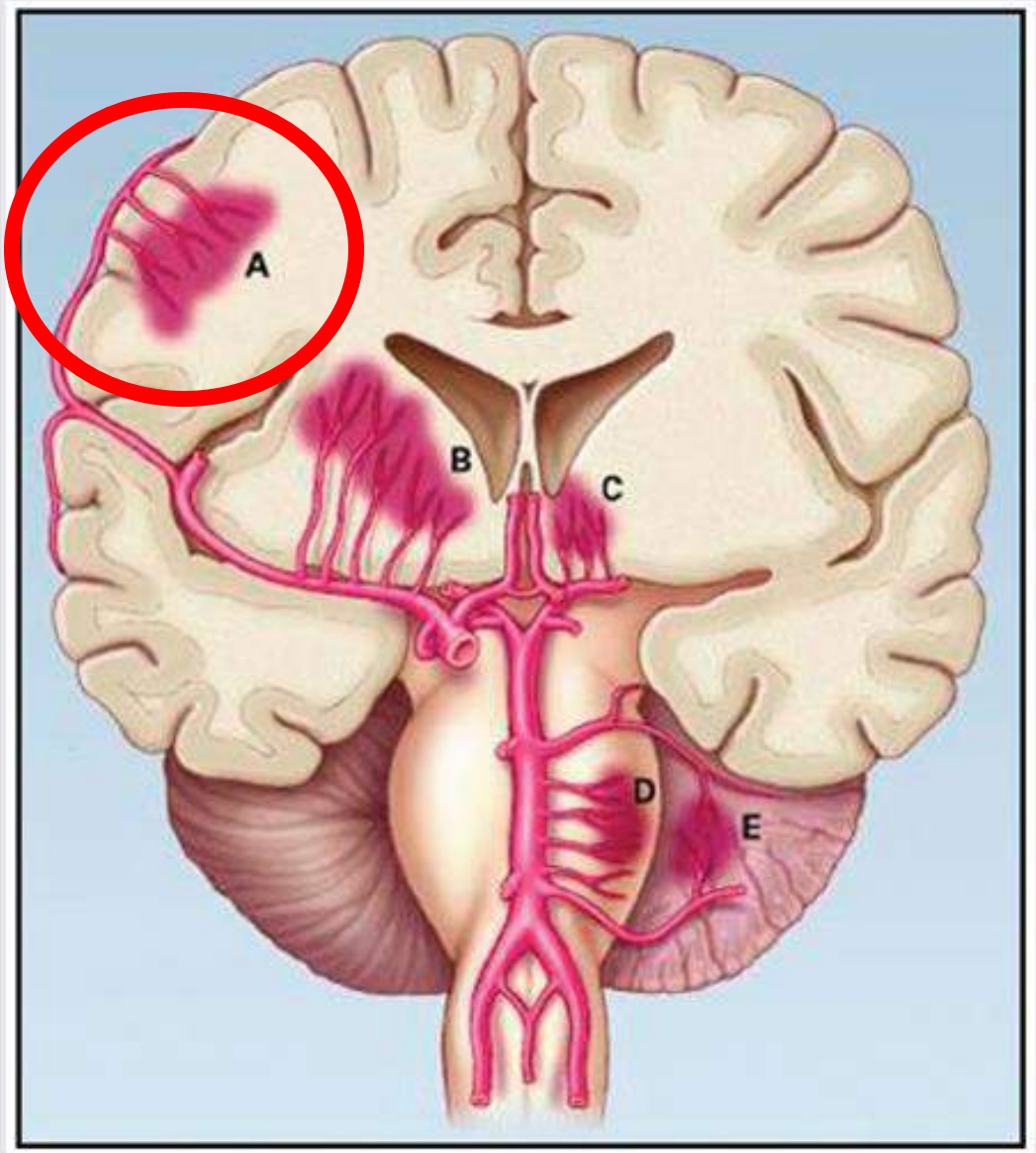
Lobar hemorrhage 25%

- Penetrating cortical branches of ACA, MCA, & PCA
- Peripheral location ∴ lower frequency of coma
- Lower mortality
- Better functional outcome



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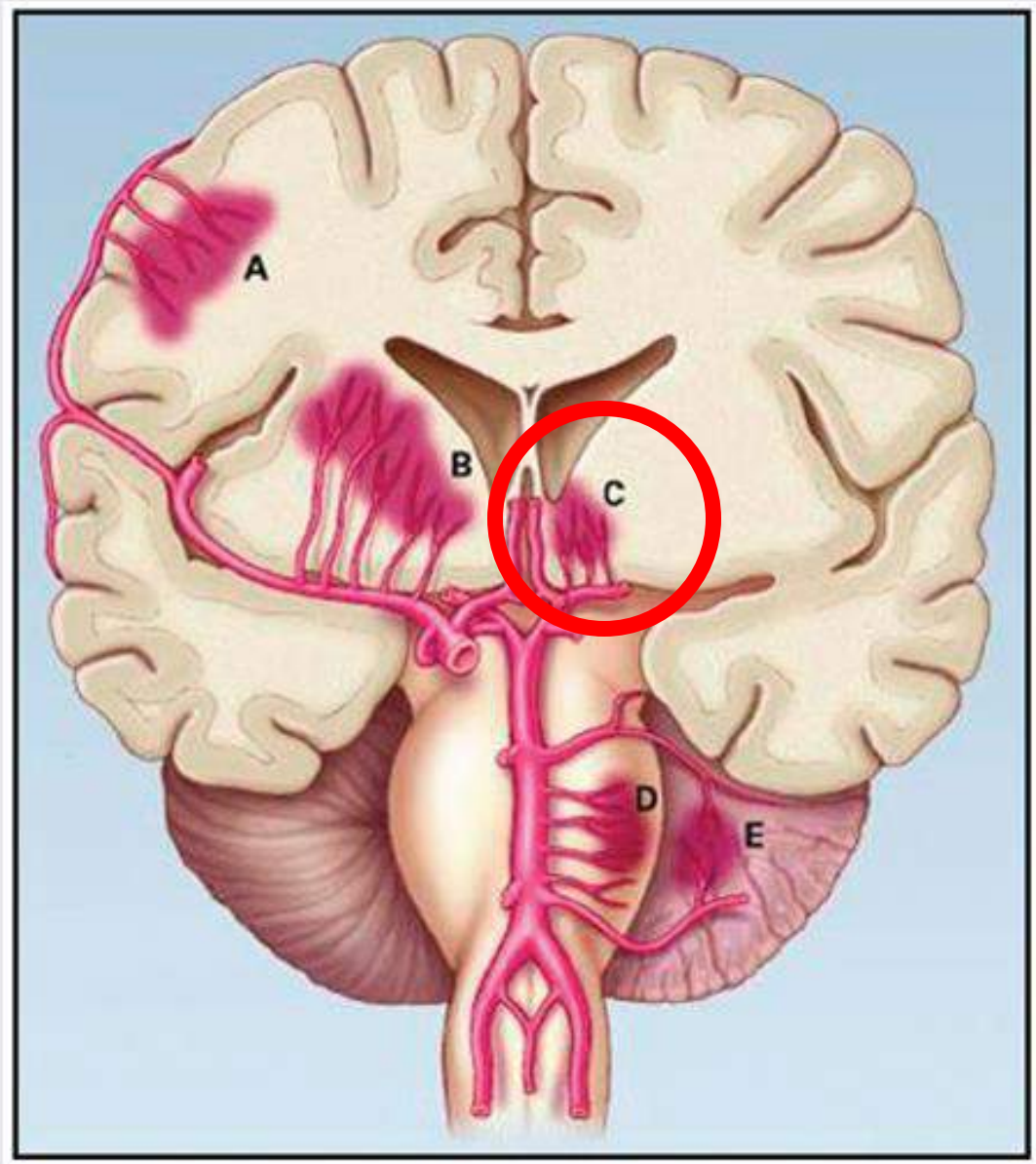

Thalamus 10-15%

- Ascending thalamogeniculate branches of PCA
- Abrupt hydrocephalus from aqueductal obstruction from intraventricular clot
- Responds to ventriculostomy

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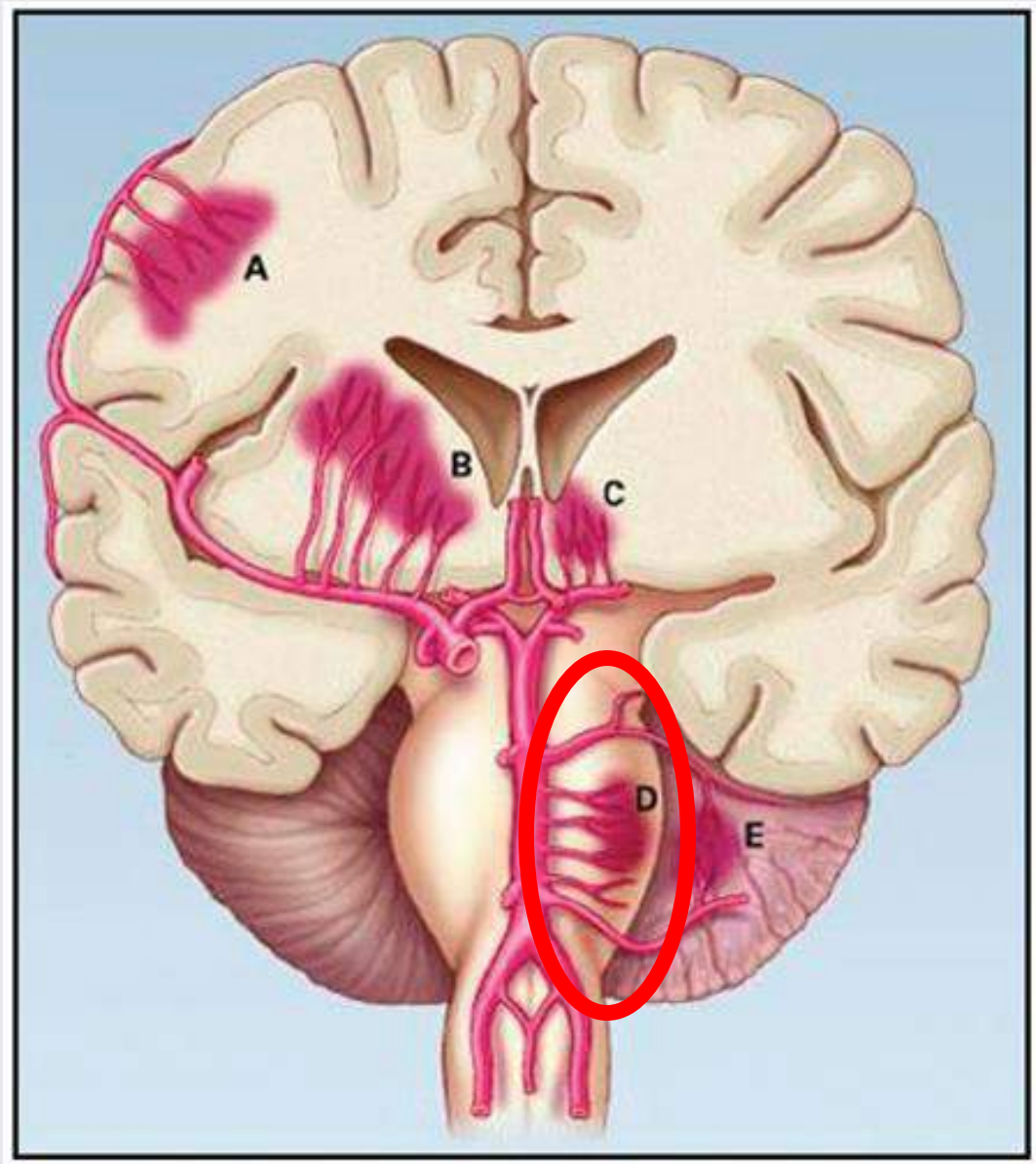

Pons 5%

- Paramedian branches of the basilar artery
- Bilateral carries very poor prognosis (coma, quadriplegia, decerebrate posturing, horizontal ophthalmoplegia, pinpoint reactive pupils)

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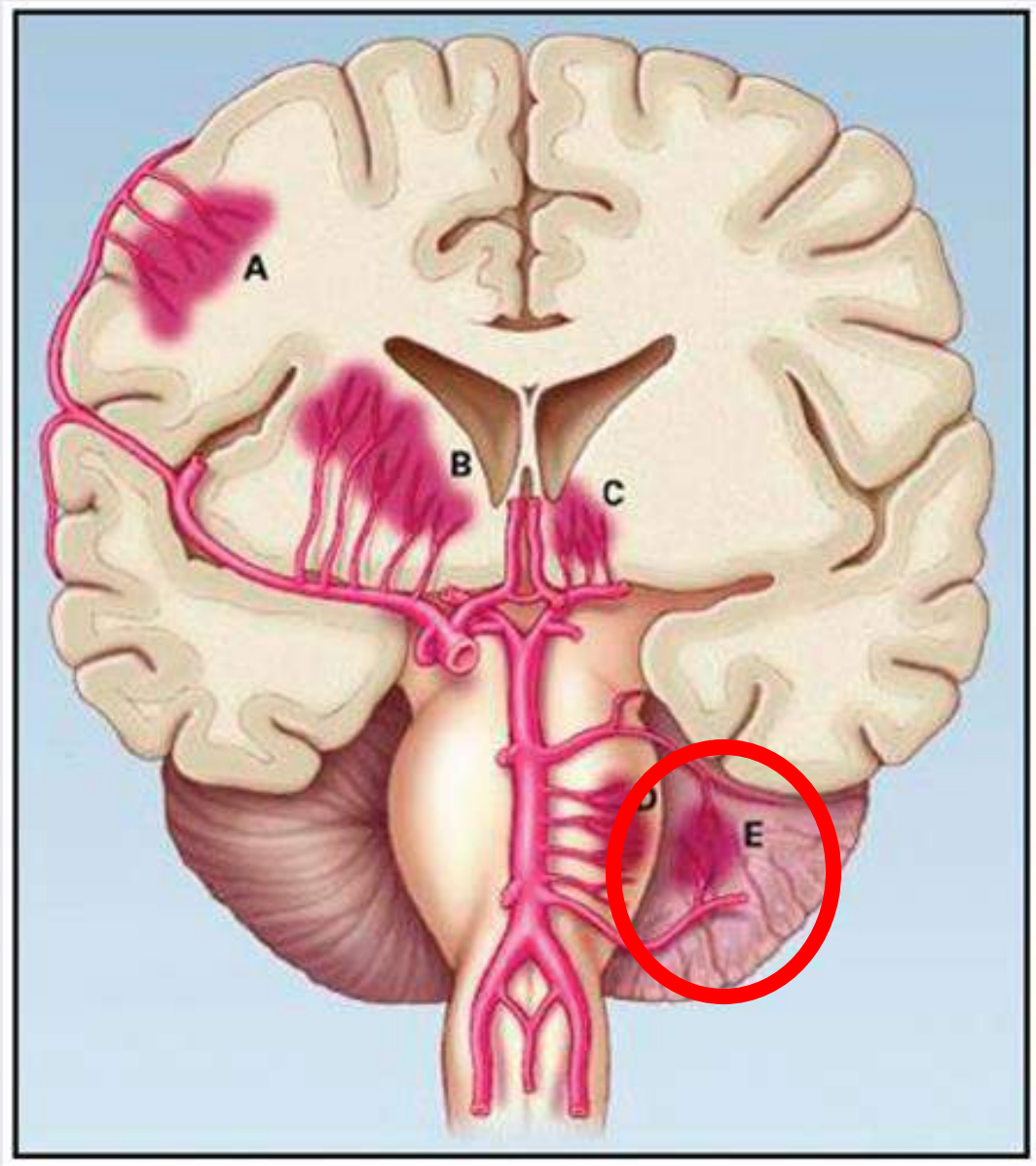

Cerebellum 5-10%

- Penetrating branches of the PICA, AICA, SCA
- Abrupt onset vertigo, h/a, n/v, inability to walk in absence of weakness
- Ipsilateral ataxia, horizontal gaze palsy, peripheral facial palsy
- Unpredictable deterioration to coma

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Anticoagulation associated ICH

- Warfarin is a Vit K antagonist
  - Inhibits biosynthesis of factors II, VII, IX, X
  - Maximum effect is 48 hrs after administration
- Incidence of ICH is 0.3-0.6% per year in patients on chronic warfarin anticoagulation
- Risk factors
  - Age, chronic hypertension, CAA, leukoaraiosis
  - Elevation of INR (doubled risk for 0.5 ↑ above 4.5!)
- INR correlated with hematoma expansion and prognosis

Clinical features

- Features of intracranial hypertension
  - Headache, vomiting, decreased LOC
  - Correlated with hematoma size and prognosis
  - Progressive over time
  - Seizures in lobar ICH
- Focal neurological deficits depending on the location of ICH

Think FAST


ACT FAST at the First Sign of STROKE
FACIAL WEAKNESS
ARM WEAKNESS
SPEECH DIFFICULTIES
CALL 911
TIME IS BRAIN
www.strokemn.org
Minnesota Stroke Association
Chapter of the National Stroke Association NSA


Face
Does the face look uneven?
Ask them to smile.
FAST
Arm
Does one arm drift down?
Ask them to raise both arms.
Speech
Bof fri flea.
Does their speech sound strange?
Ask them to repeat a phrase.
Time
Every second brain cells die. Call 9-1-1 at any sign of stroke!
Is it a stroke?
Check these signs FAST!
Call 9-1-1 at any sign of stroke.

Eye Opening (E)	Verbal Response (V)	Motor Response (M)
4 = spontaneous 3 = to voice 2 = to pain 1 = none	5 = normal conversation 4 = disoriented conversation 3 = words, but not coherent 2 = no words, only sounds 1 = none	6 = normal 5 = localized to pain 4 = withdraws to pain 3 = decorticate posture$^{\alpha}$ 2 = decerebrate$^{\beta}$ 1 = none

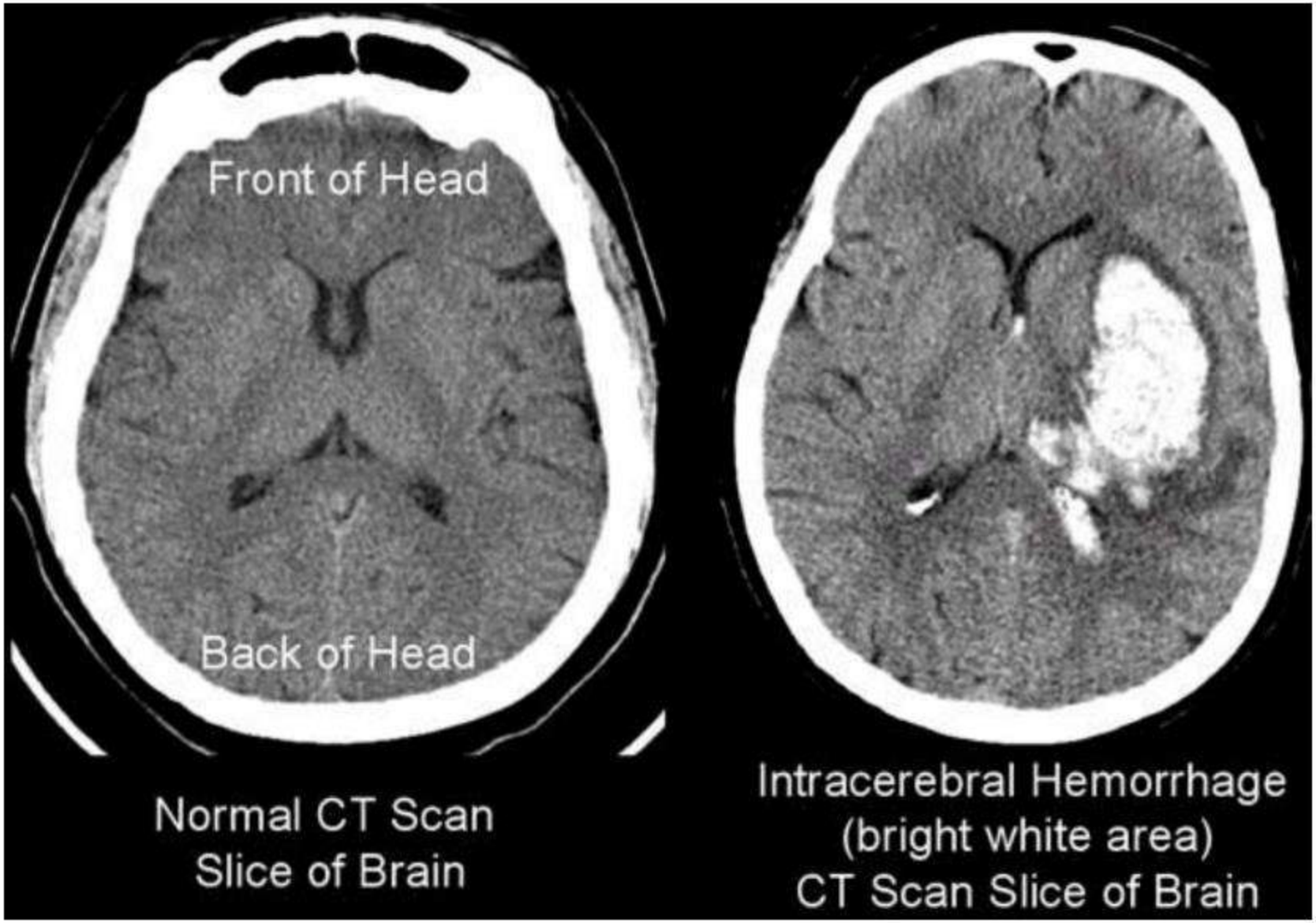
Assessment:

- Severe: GCS 3-8 (cannot score lower than a 3)
- Moderate: GCS 9-12
- Mild: GCS 13-15

INVESTIGUATION

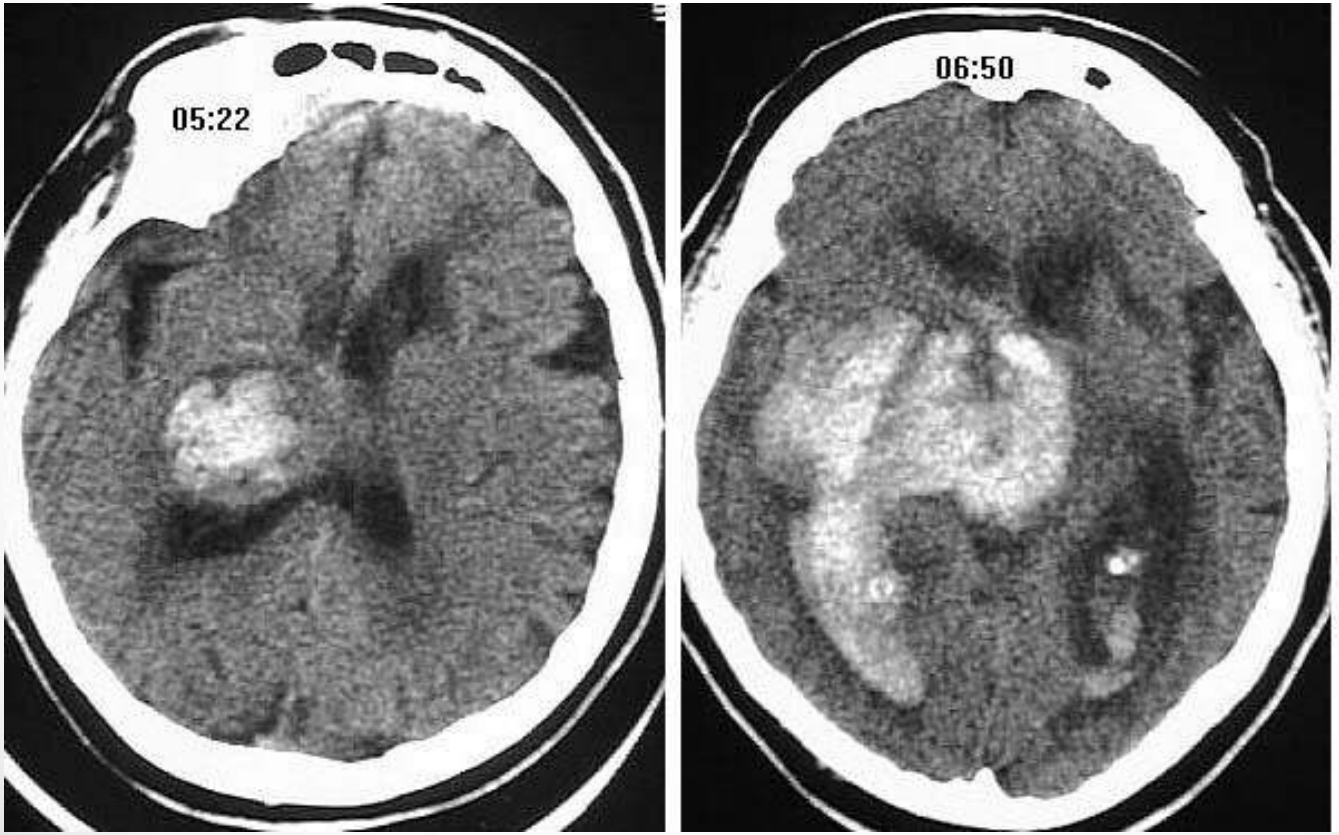
Test	Results
Head CT	• Differentiates hemorrhagic from ischemic stroke
ECG	• Signs of myocardial ischemia, large inverted T waves
Labs	• Chem panel – r/o conditions that have similar presentation • CBC – thrombocytopenia • PT/PTT – w/o coagulopathy as cause
MRI **CT angiography** **Invasive angiography**	• r/o aneurysm or arteriovenous malformation as a cause of bleeding • Recommended in all patients <45 years of age and in all patients with intracerebral hemorrhage in lobar brain regions

Head CT


Front of Head
Back of Head
Normal CT Scan
Slice of Brain
Intracerebral Hemorrhage
(bright white area)
CT Scan Slice of Brain

Hematoma expansion

- Hematoma enlargement
  - >70% have hematoma enlargement w/in 3 hrs of symptom onset; 1/3 clinically significant
  - Most occur within 3 hrs, can be up to 12 hrs
  - Independent predictor of worse outcome & ↑ mortality



Mainstay of ICH therapy is to treat the underlying cause when possible

General treatment approach is always patient specific depending on clinical condition

INITIAL MANAGEMENT

- Stabilization of Vital Signs
  - Neurological exam
- Supportive care
  - Management of seizures
- Blood pressure control
- Fever control
- Anticoagulation correction
- Blood sugar control
- Surgical/Invasive Interventions

- Antihypertensive agents for ICH have not been compared in controlled trials
- Suggested agents:
  - Labetolol
  - Enalapril
- For reftractory hypertension:
  - Nicardipine
  - Hydralazine
  - Nitroprusside
    - Can lead to elevated ICP

- Goal SBP to < 180 mm Hg within 1 hour is and maintain for next 24 hours
  - INTERACT study suggests more aggressive therapy with goal SBP < 140 mm Hg leads to better outcomes

Acute management of BP

PROs

- ↑ BP associated with poor outcome
- ↑ risk of hematoma enlargement
- ↑ edema formation
- Systemic damage (e.g. ongoing cardiac ischemia)

CONs

- Chronic HTN shifts cerebral auto-regulatory curve to the right
- ↑ ICP may require ↑ BP to maintain CPP
- Previously thought to induce ischemic damage to the at risk penumbra

Acute management of BP

Condition	Treatment Approach
SBP > 200 mmHg or MAP is 150 mmHg	Aggressive ↓BP with continuous IV infusion, with frequent BP monitoring Q5 min
SBP is 180 mmHg or MAP is 130 mmHg and there is the *possibility* of ↑ICP	Monitor ICP and ↓BP using intermittent or continuous IV meds while maintaining cerebral perfusion pressure 60 mmHg
SBP is 180 mmHg or MAP is 130 mmHg and there is *no evidence* of ↑ICP	Modest ↓ BP (eg, MAP of 110 mm Hg or target BP of 160/90 mm Hg) using intermittent or continuous IV meds to control BP and clinically reexamine the patient Q15 min

- Elevate head of bed to 30 degrees
  - Analgesia and sedation as needed
- Aggressive therapies:
  - Osmotic therapy
    - Mannitol
      - RCT failed to demonstrated difference in disability or death at 3 months
    - Hypertonic Saline
    - Barbituate anesthesia
  - Hyperventilation and glucocorticoids not recommended

- High blood glucose on admission predicts an increased risk of mortality and poor outcome in patients with and without diabetes and ICH
  - Use of insulin is controversial. Hypoglycemia should be avoided.

- CSF drainage may be appropriate in setting of obstructive hydrocephalus
  - High rates of complication : bacterial meningitis
- Endoscopic hematoma Evacuation
  - May improve long-term prognosis

- Use of platelet transfusions in ICH patients with a history of antiplatelet use is unclear
  - May be indicated for patients with severe thrombocytopenia
- Intermittent pneumatic compression recommended
- May consider low dose SQ LMWH or UFH for prevention of DVT
  - After 1 to 4 days from onset with lack of mobility

Approach to ICP management

CSF volume

- Mannitol or hypertonic solution
- External CSF drainage
- Ventricular catheter
- Ventriculo - peritoneal or atrial shunt
- Lumbar drain
- Serial lumbar punctures

Brain volume

- Mannitol or hypertonic saline
- Decompressive craniectomy
- Resection of tumor or other mass lesion

Seizure Control

Blood volume

- Mannitol or hypertonic saline
- Hyperventilation
- Hypothermia
- Head elevation, neutral neck position
- Deep propofol or barbiturate sedation ± paralysis

Decompressive Craniectomy

- Surgical removal of cranial bone flap to relieve intracranial pressure
- Useful in large ischemic CVA with profound edema
- Role in traumatic brain injury still needs to be established

Complications

- Aspiration pneumonia
  - Stroke-related dysphagia
  - Therapy: dysphagia/swallow evaluation before moving to PO status
- Hydrocephalus
  - Elevation of CSF pressure of the brain
  - Cognitive impairment, urinary/fecal incontinence
  - Therapy: external ventricular drain placement, ventriculoperitoneal shunt
- Seizures
  - May complicate treatment for ICH; higher risk in cortical bleeding
  - Therapy: benzodiazapines, phenytoin, fosphenytoin

- Delirium
  - May be fairly common following ICH
  - Therapy: supportive care, sedatives and neuromuscular blockade, careful hemodynamic management
- Deep venous thrombosis
  - Motor weakness, venous stasis
  - Therapy: anticoagulation, inferior vena cava filter
- Infection
  - Nosocomial pneumonia, urinary tract infection, cellulitis from pressure sores
  - Therapy: appropriate broad-spectrum coverage, then narrowing for cultured organisms

Preventive Measures

- Lifestyle modifications:
  - smoking cessation, refrain from alcohol, diet/exercise, weight control
- Control blood pressure
- Control LDL
- Clot prevention: Warfarin, Aggrenox, Plavix

Recurrence: Risk Factors

- Lobar location of initial ICH
- Uncontrolled hypertension
- Older age
- Ongoing anticoagulation
- Apolipoprotein E epsilon 2 or epsilon 4 alleles
- Greater number of microbleeds on MRI

- Monitoring by appropriate specialists for rehabilitation
  - Physical therapist, occupational therapist, speech and language pathologist
- Strict management of HTN
- Antiplatelet/anticoagulation therapy

Conclusions

- ICH has an increasing incidence, but continues to have a very poor prognosis
- Hypertension is a major risk factor
- Acute BP reduction of 15-20% is safe

References

- Goldstein, JN et al. Contrast extravasation on CT angiography predicts hematoma expansion in intracerebral hemorrhage, Neurology 2007;68:889–894
- Qureshi AI et al. Intracerebral hemorrhage, Lancet 2009; 373: 1632–44
- Wada, R et al. CT Angiography "Spot Sign" Predicts Hematoma Expansion in Acute Intracerebral Hemorrhage, Stroke 2007;38:1257-1262
- Diringer MN. Update on intracerebral hemorrhage, AAN Continuum, 2009
- Kincaid MS and Lam AM, Monitoring and managing ICP, AAN Continuum, 2006