CEREBROVASCULAR DISEASE

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CEREBROVASCULAR DISEASE(CVD):

Any abnormality of the brain resulting from a pathologic process in the cerebral circulation or its vessels.

CEREBROVASCULAR DISEASE

- Dilatation/Enlargement
- Narrowing/Obstruction
- Disruption of Vessel

CEREBROVASCULAR DISEASE

- Mass Effect
- Ischemia/Infarction
- Hemorrhage

STROKE:
A sudden loss or diminution in neurologic function caused by the rupture or obstruction of an artery of the brain.


CALL
911!!!
A BRAIN
ATTACK OR
STROKE IS AN
EMERGENCY
!

SUDDENLY!!!
you experience:

- Weakness, numbness or paralysis of face, arm or leg — especially on one side of the body.
- Blurred or decreased vision in one or both eyes.
- Slurred speech, difficulty speaking or understanding.
- Loss of balance or coordination.
- Severe headache.

THE GEORGE WASHINGTON UNIVERSITY MEDICAL CENTER

Progress

APRIL 1996

Brain Attack Team Aids Stroke Victims

The Brain Attack Team, a new collaboration of the Departments of Emergency Medicine, Neurology, Neurological Surgery, and Radiology and the D.C. Emergency Services, is now providing rapid and specialized treatment for stroke victims.

Expediting the care of stroke patients, notes **Ashfaq Shuaib, M.D.**, professor of neurology, who is co-director of the Brain Attack Team with **Robert F. Sheeser, M.D., M.P.H.**, acting chair of the Department of Emergency Medicine, enables the team to take advantage of the critical six hours following the onset of stroke—the "window of opportunity"—when the use of neuroprotective and thrombolytic drugs and surgical interventions can often prevent extensive neurological damage.

Ashfaq Shuaib, M.D., co-director of the Brain Attack Team (right), consults with interventional radiologist William D. Keirk, M.D.

care of stroke victims. The Hospital has also recently introduced a Stroke CareMap to standardize and streamline treatment of stroke patients.◆

Everyone gets a star!

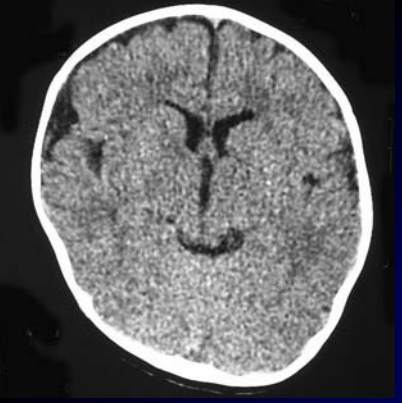
Supra sellar cistern

Courtesy Frank Brennan, CDR, USN (ret.)

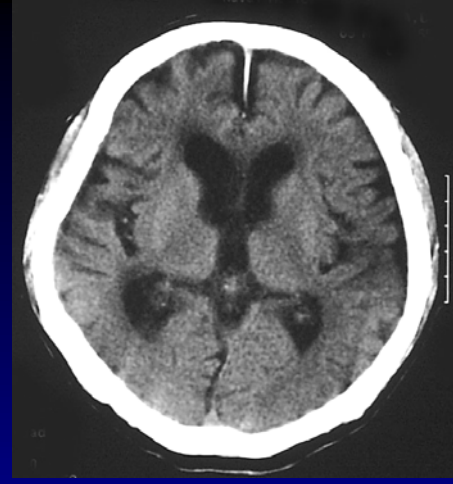
Everyone deserves a smile!

- Perimesencephalic cistern
- Quadrigeminal plate cistern

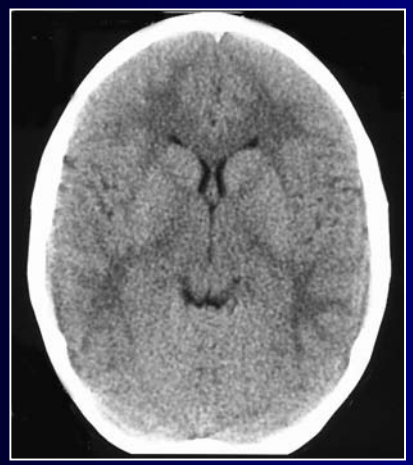
"Normal" age related



newborn



89 yrs



6 mos

20 yrs

38 yrs

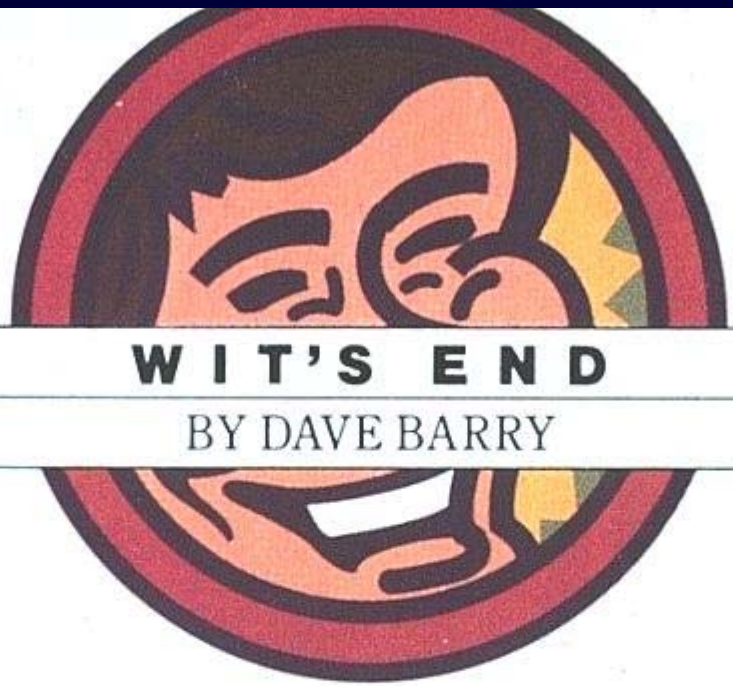
NEUROIMAGING OF STROKE: ADVANCES AND CONTROVERSIES

"STROKE"

- 500,000 per year in USA
- 200,000 deaths/year
- 28,000 are Aneurysm rupture

"STROKE"

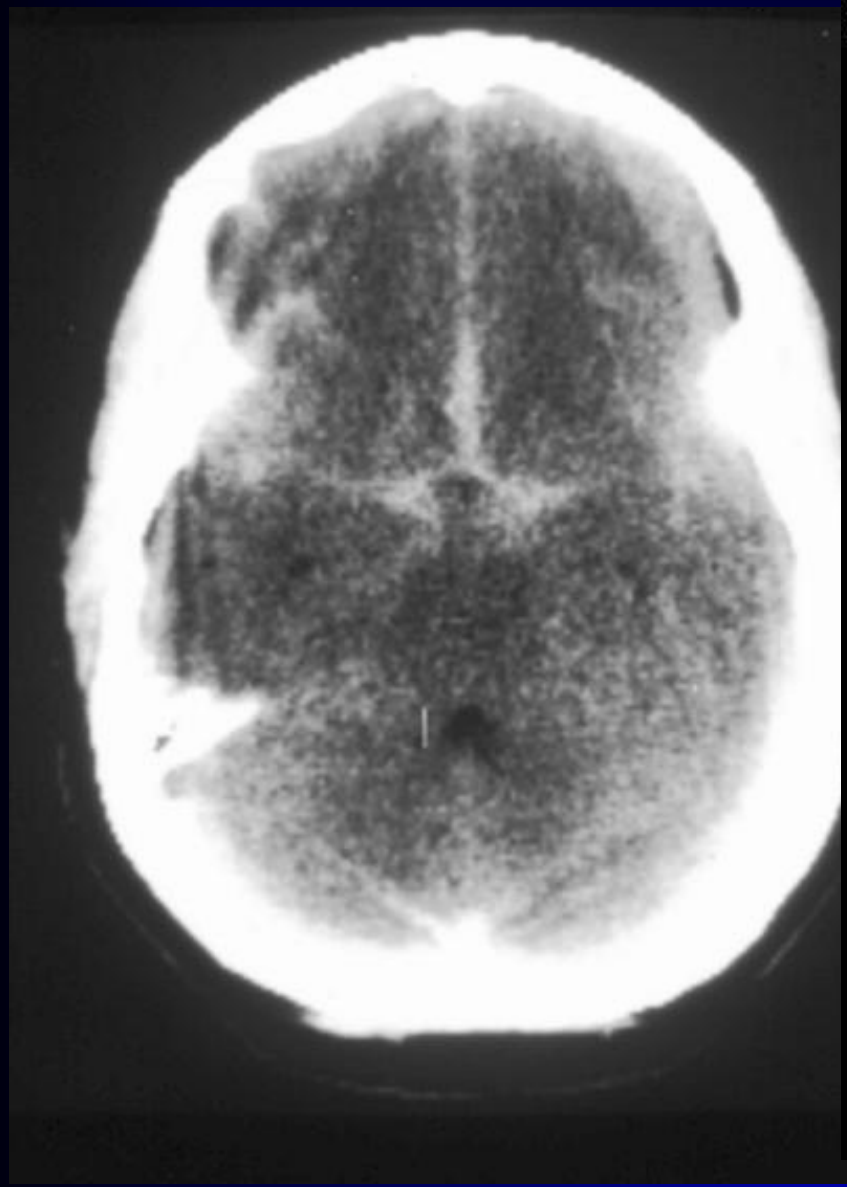
- 2.5 Million "stroke" survivors in USA
- 15% Totally disabled
- 55% Require ongoing special care

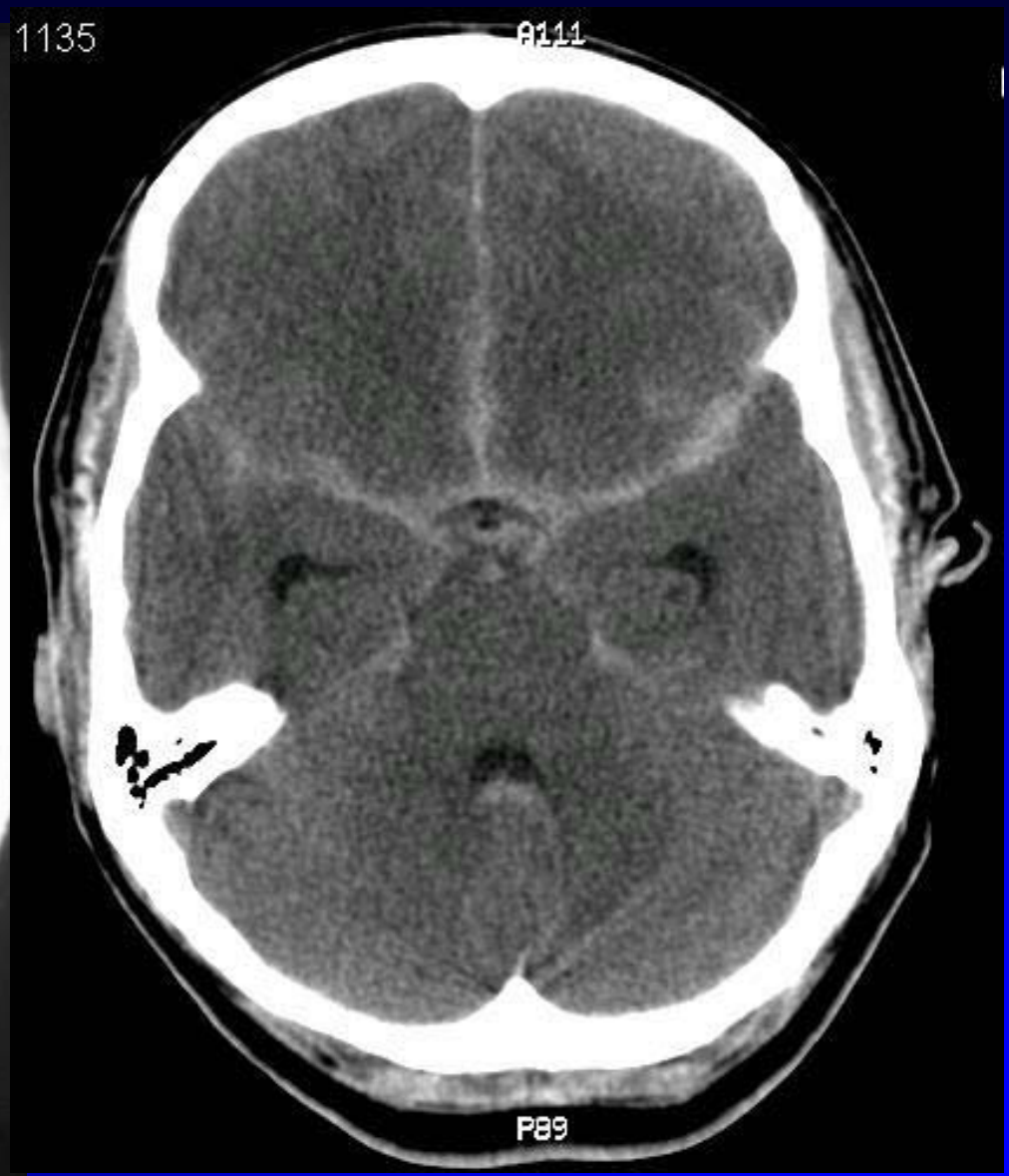


WIT'S END

BY DAVE BARRY

Exploding Heads and Other Medical Marvels

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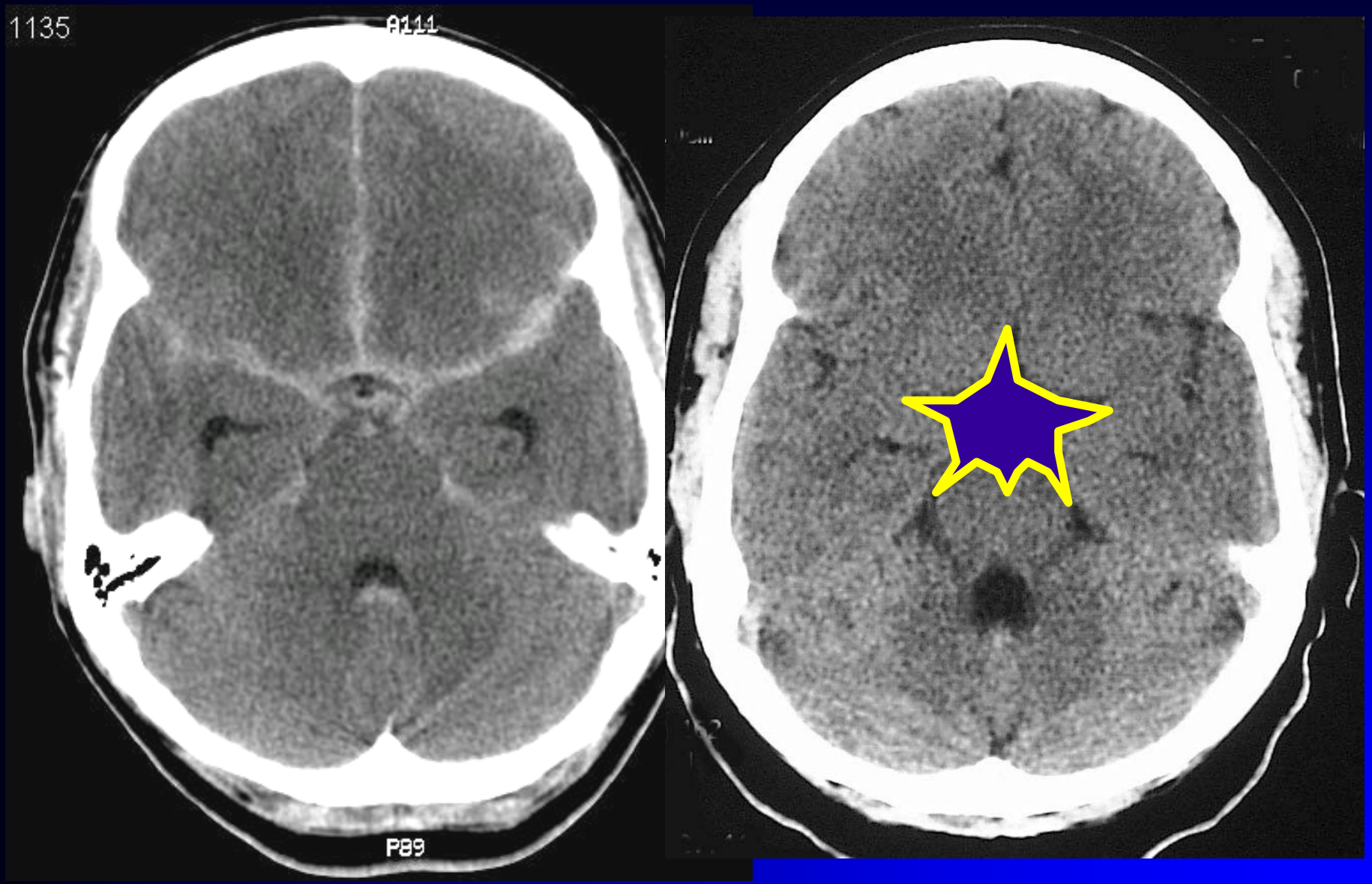
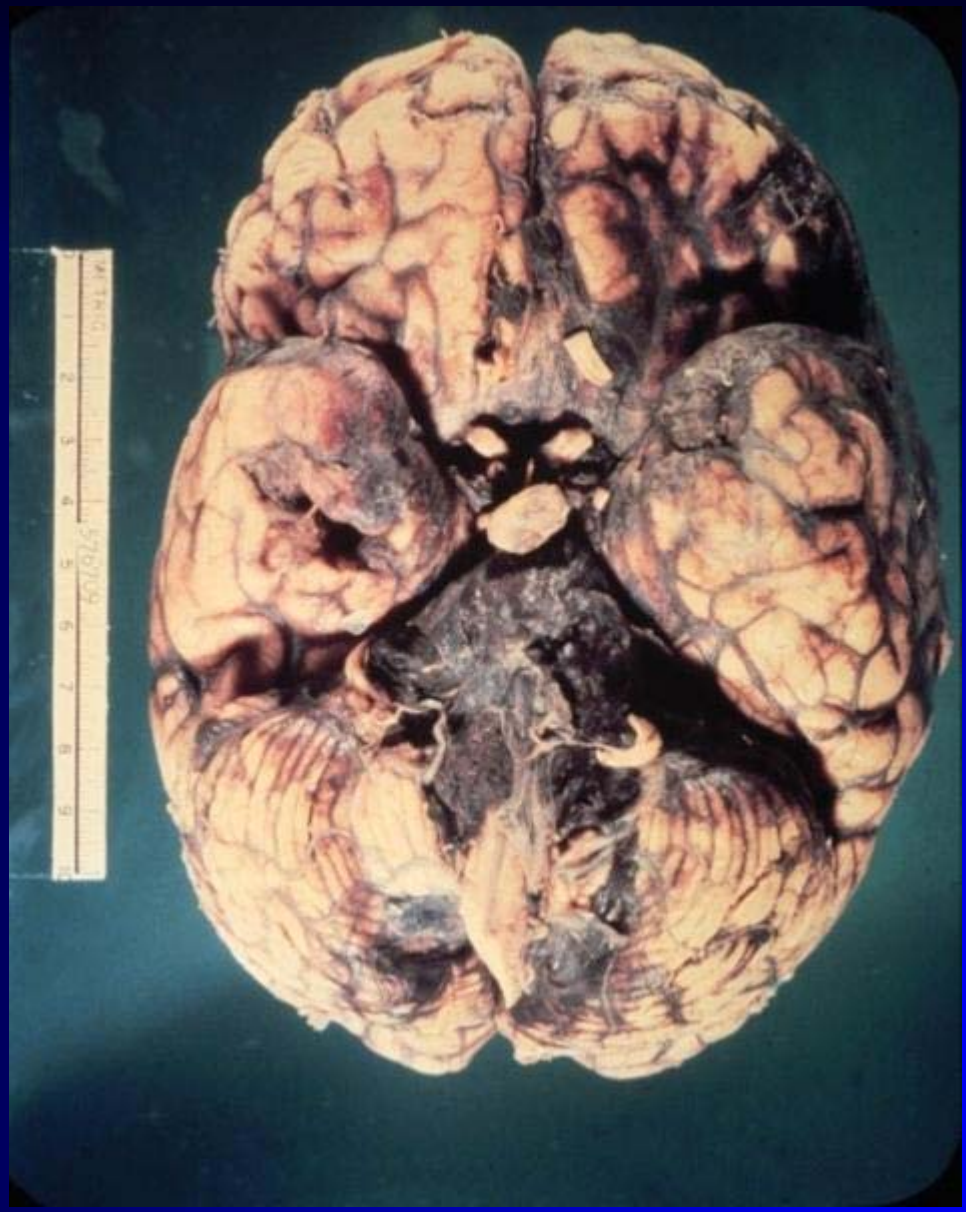
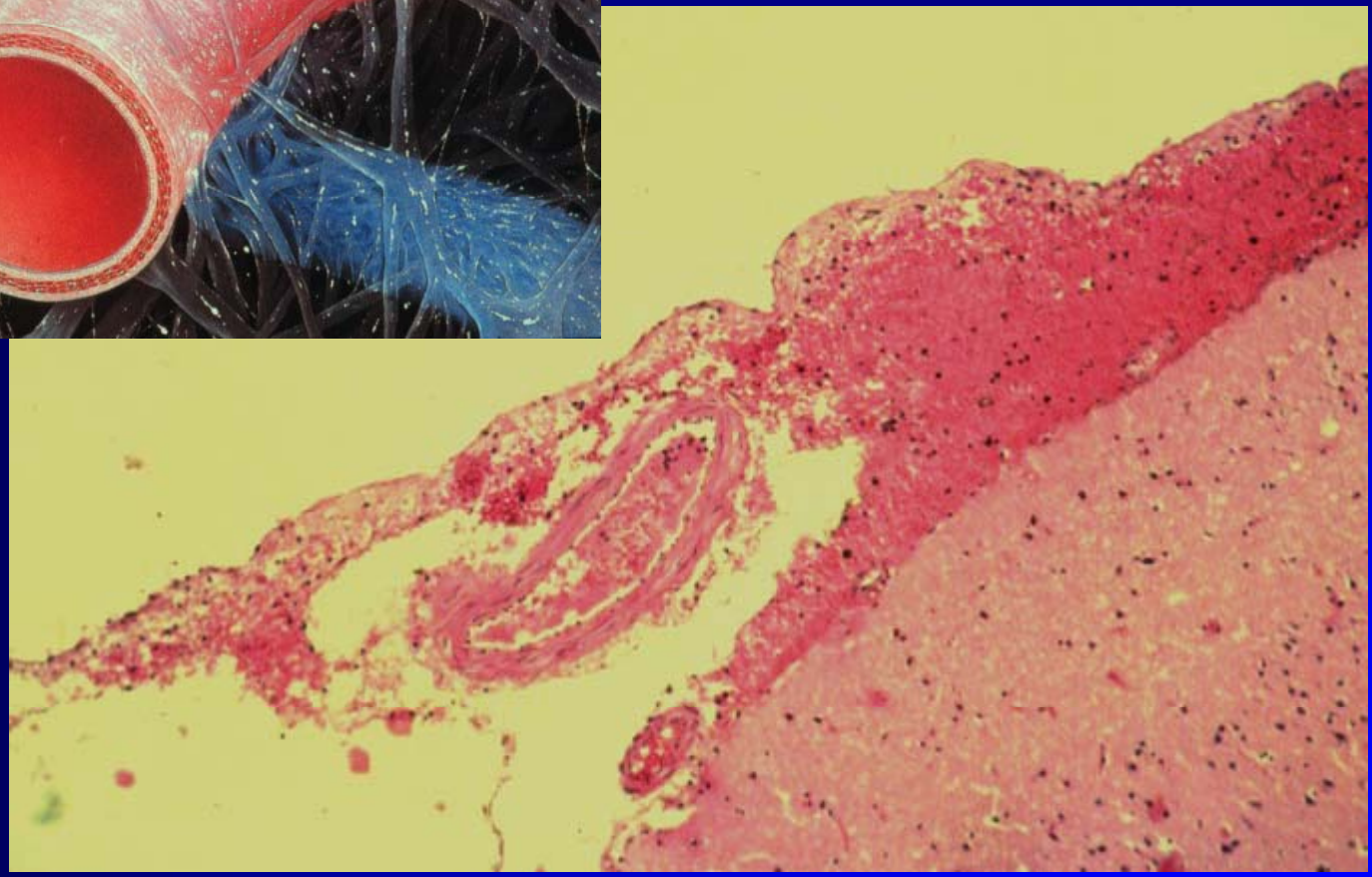
Worst HA: Non-Contrast CT



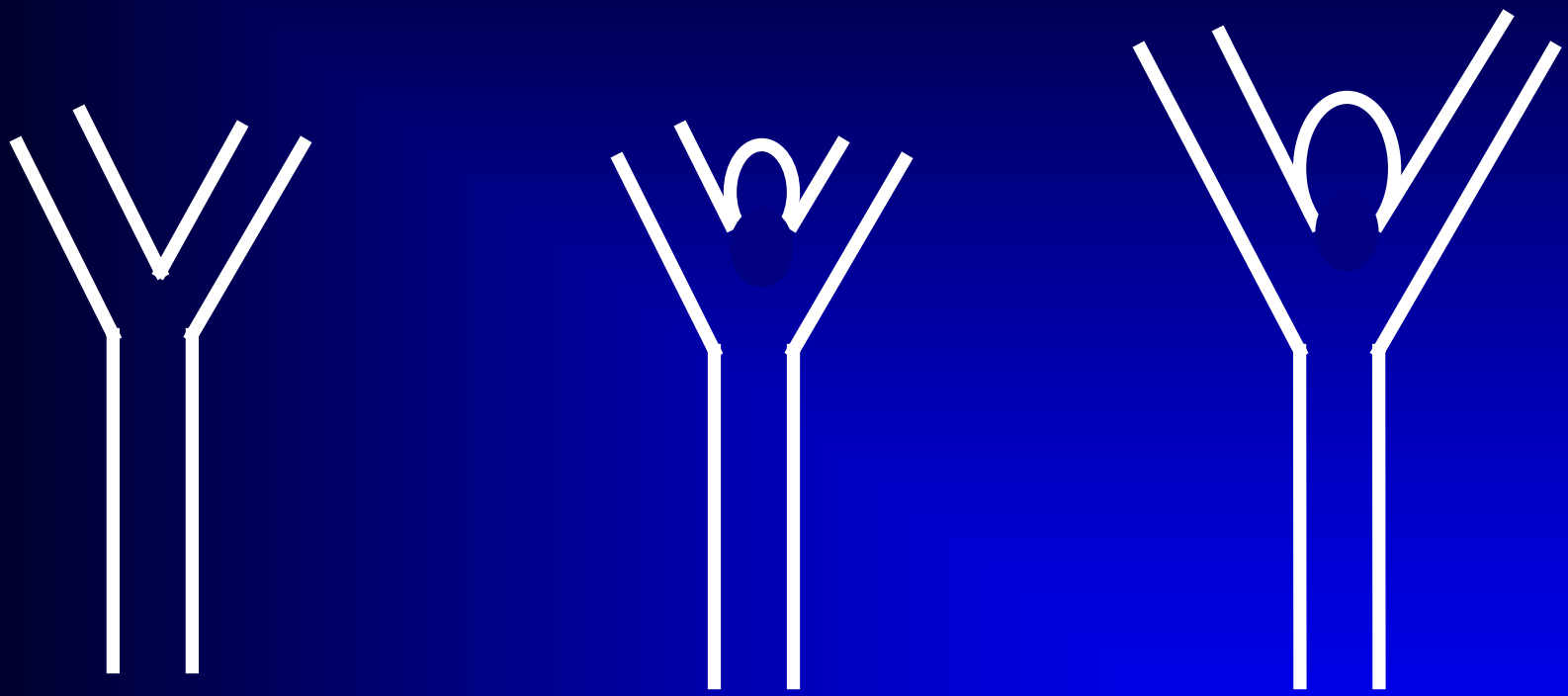


Subarachnoid Space



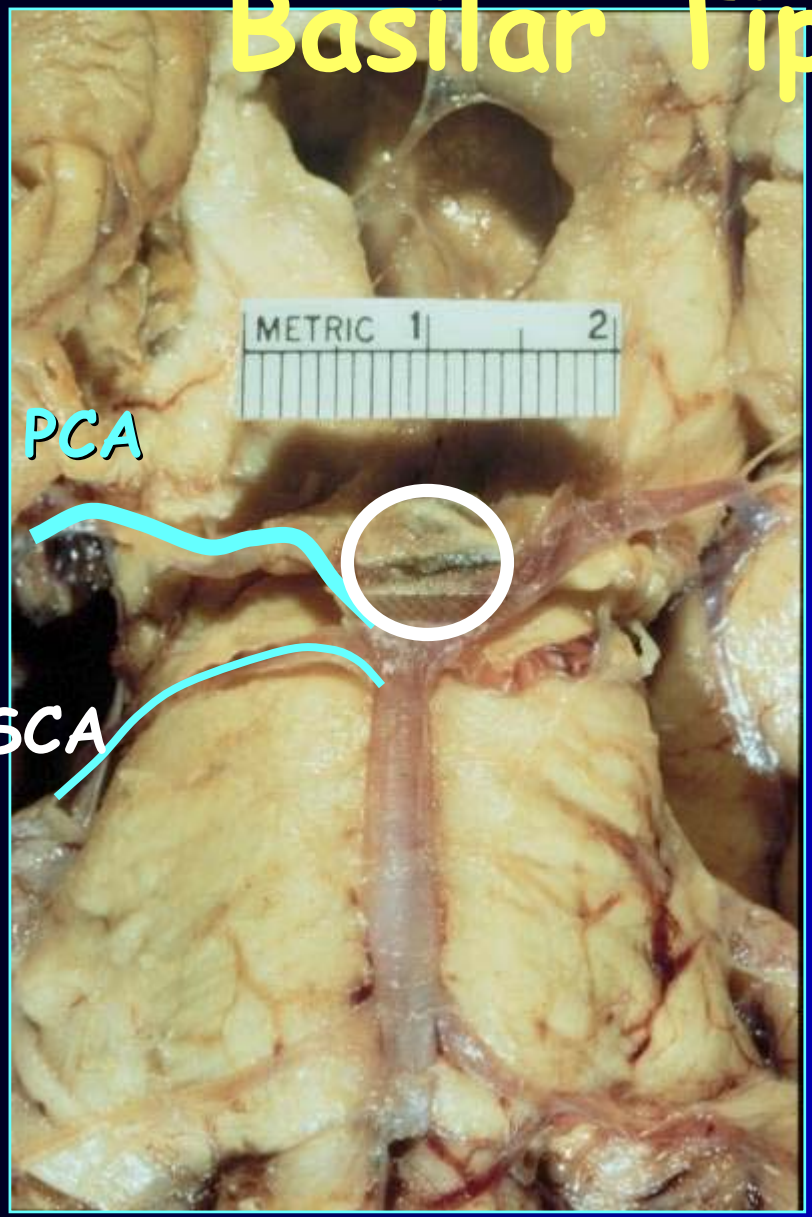

METRIC 1 2

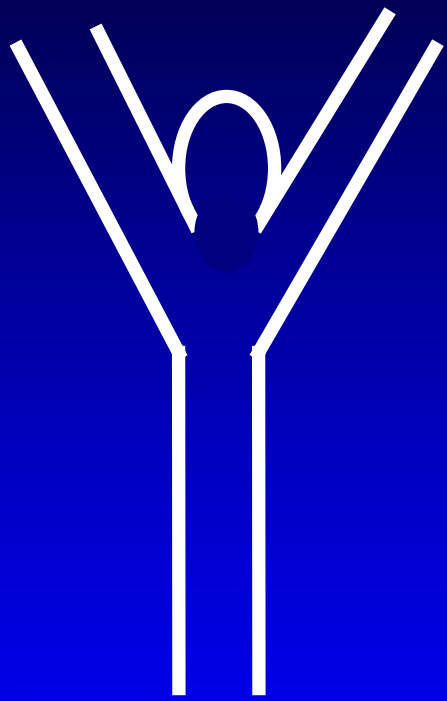
Aneurysm forms at Bifurcation



Goal!

Basilar Tip Aneurysm





Aneurysm and Rupture

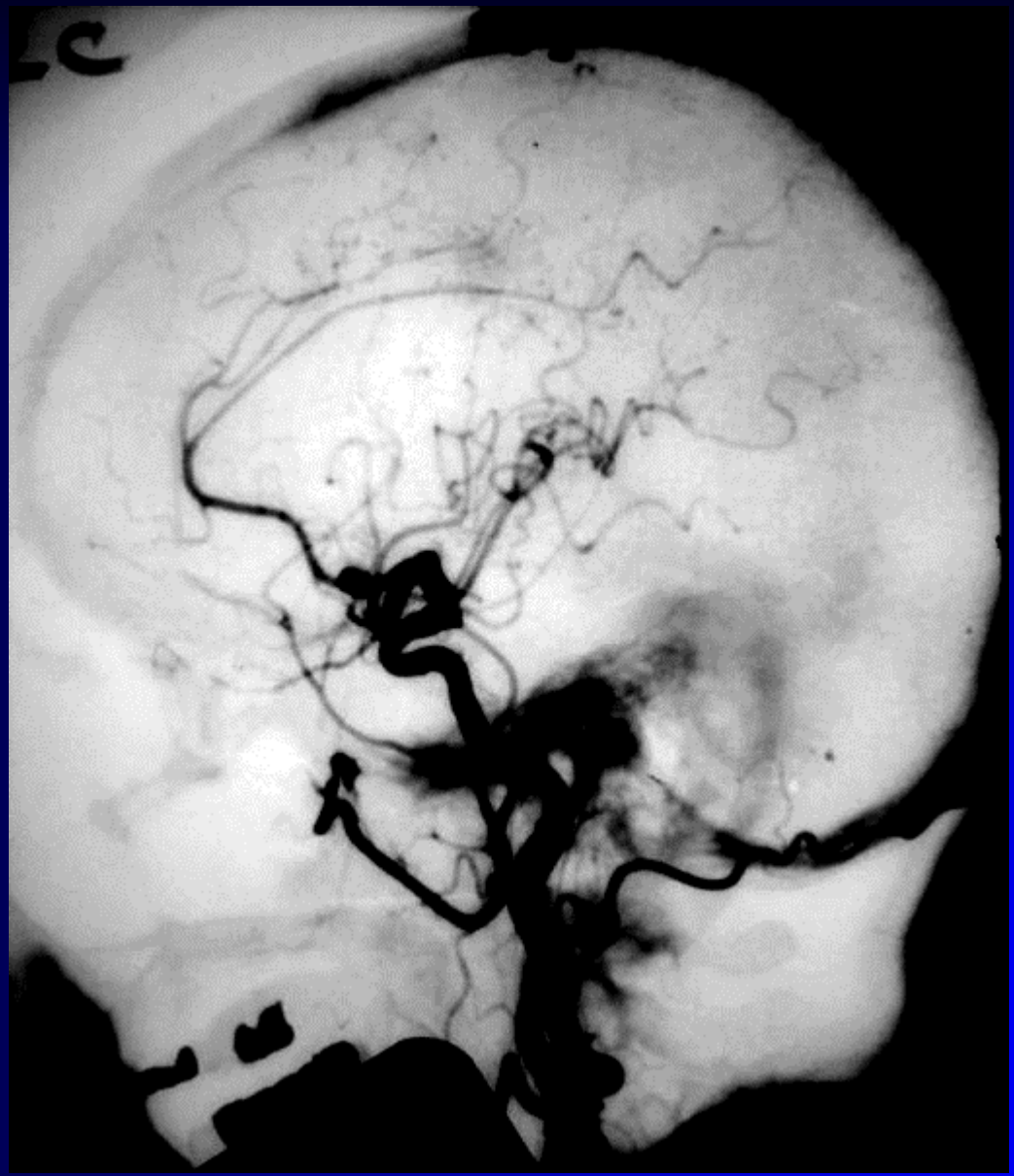
- SPASM - Infarction
- HEMATOMA - Herniation, etc.
- Re-BLEEDING
- HYDROCEPHALUS

Aneurysm and Rupture

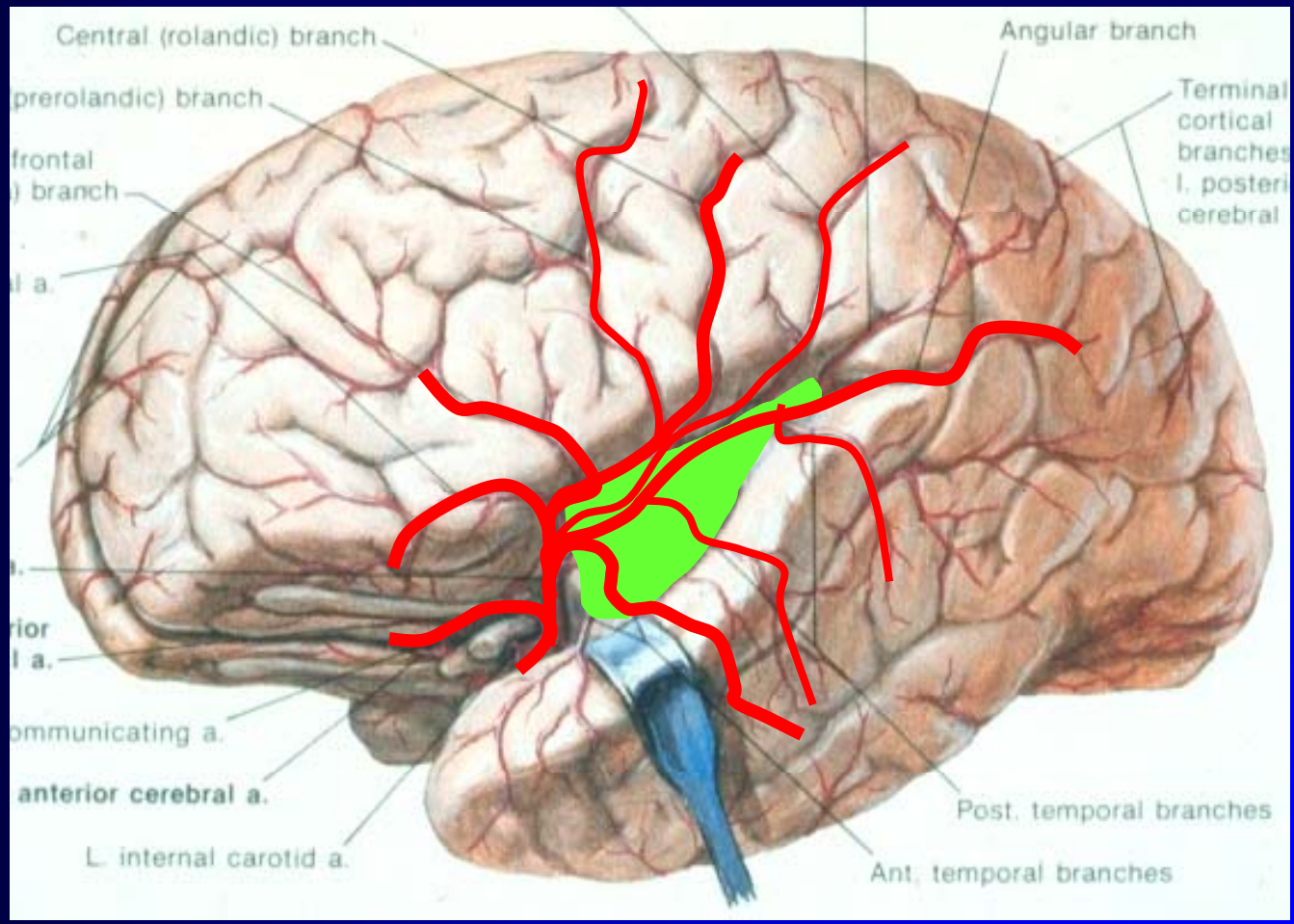
- ANGIOGRAPHY:
  - Location of Aneurysm
  - Multiple (25% are)
  - Visualize "Neck"
  - Spasm ?
  - If (-) REPEAT in 2-3 wks.

Aneurysm and Rupture

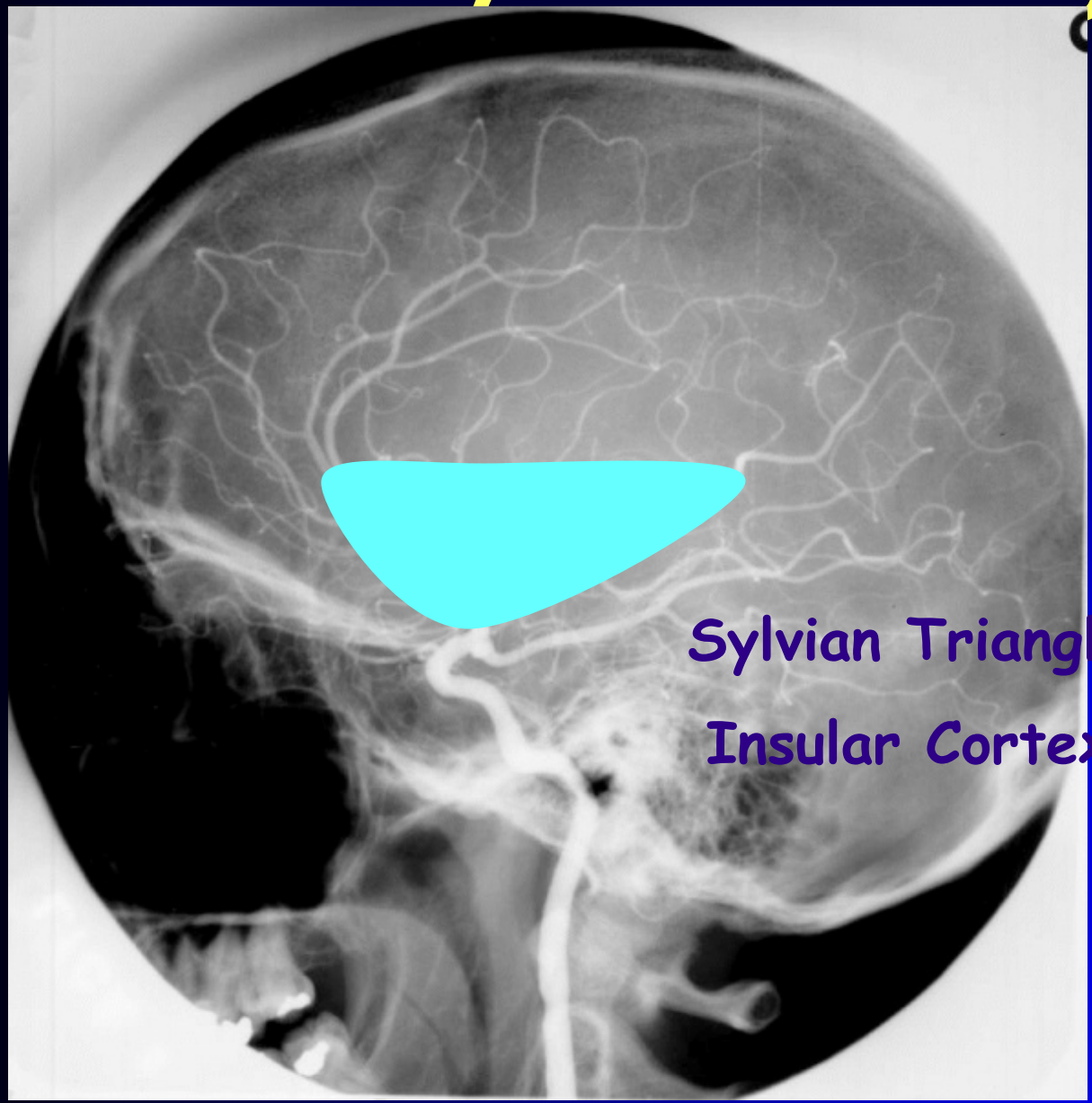
- LOCATION of ANEURYSM
  - 80-85% Anterior Circulation
  - 15-20% Posterior/Vertebral

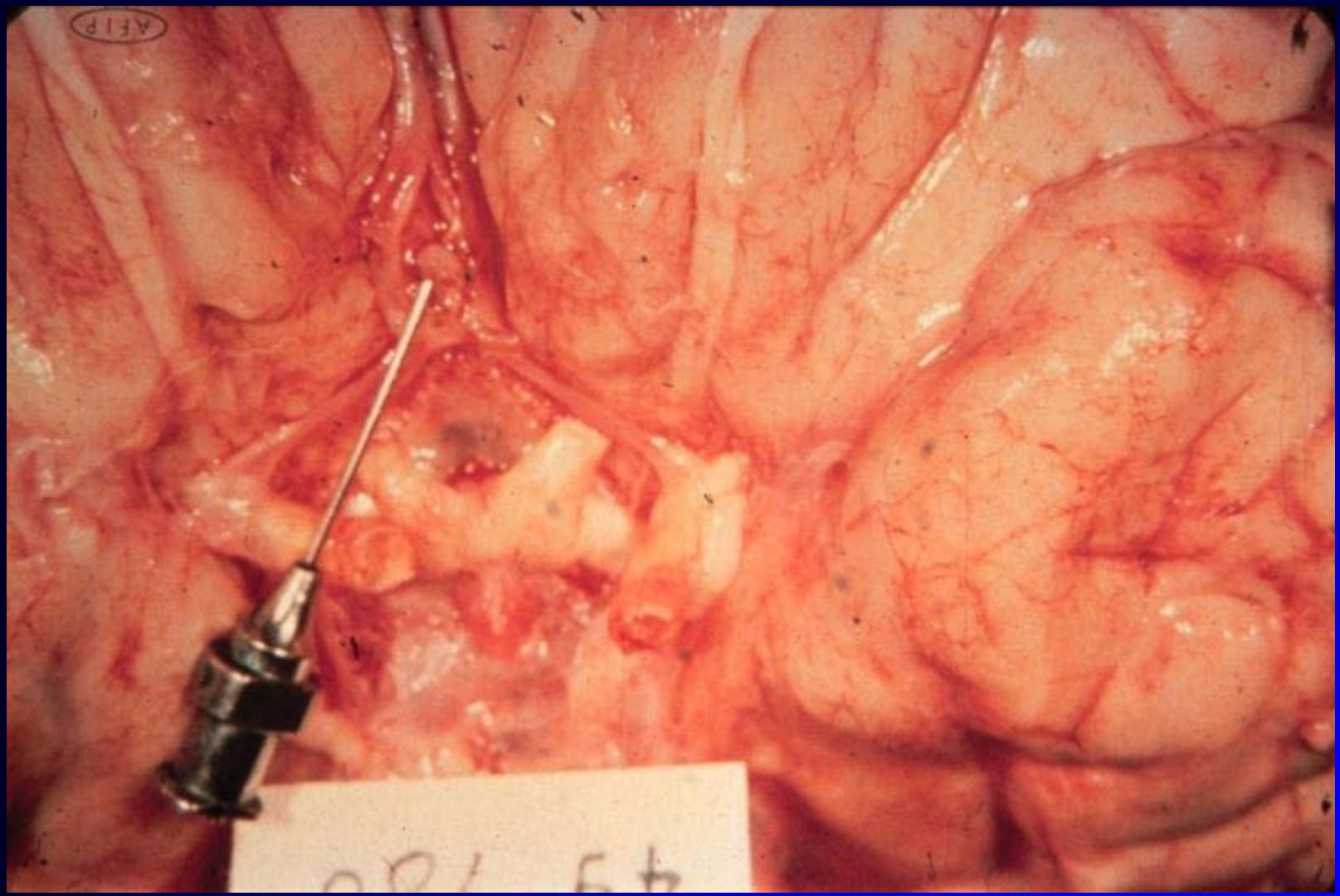


Sylvian Triangle - Insular Cortex



Sylvian Triangle

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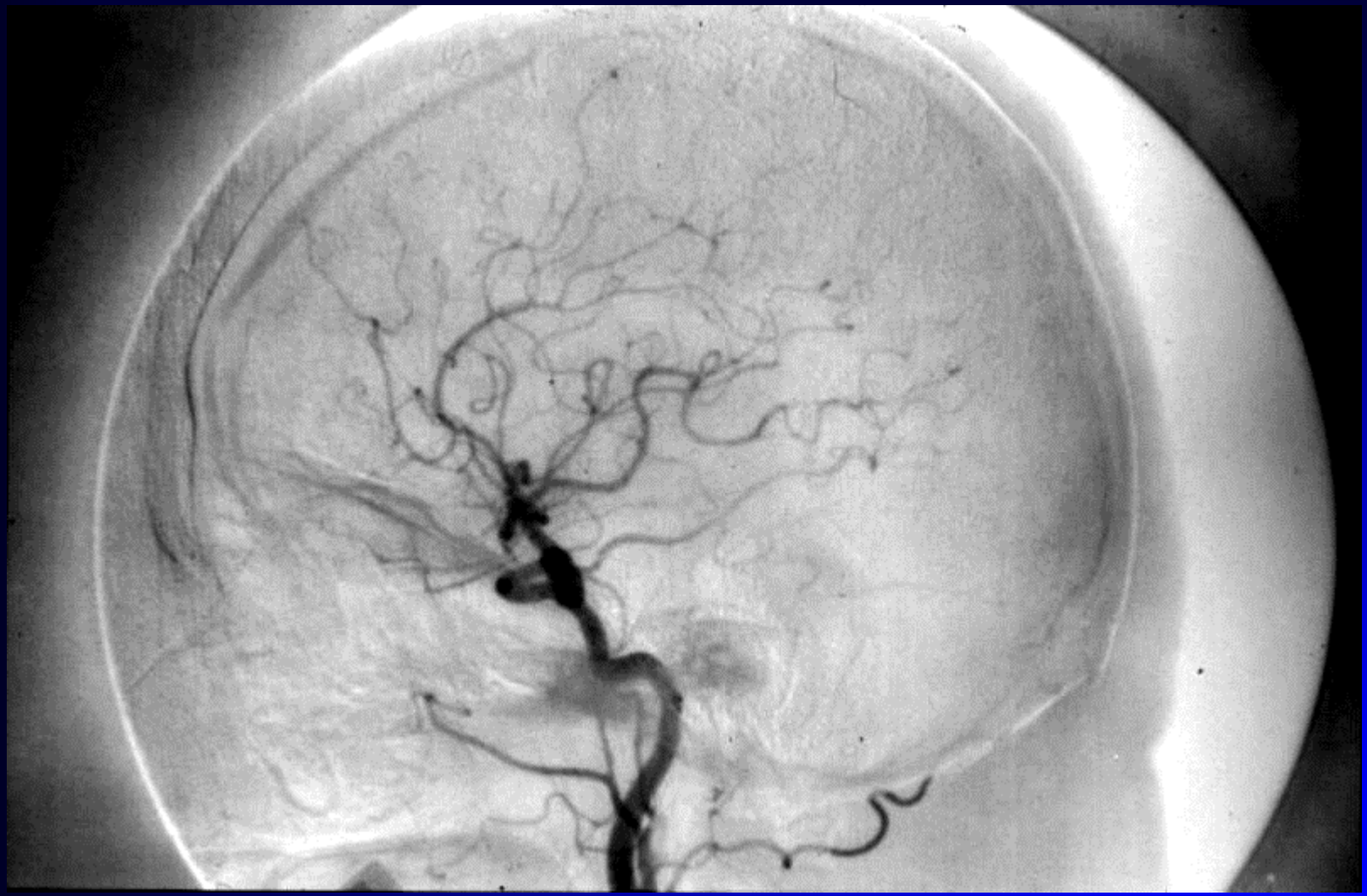

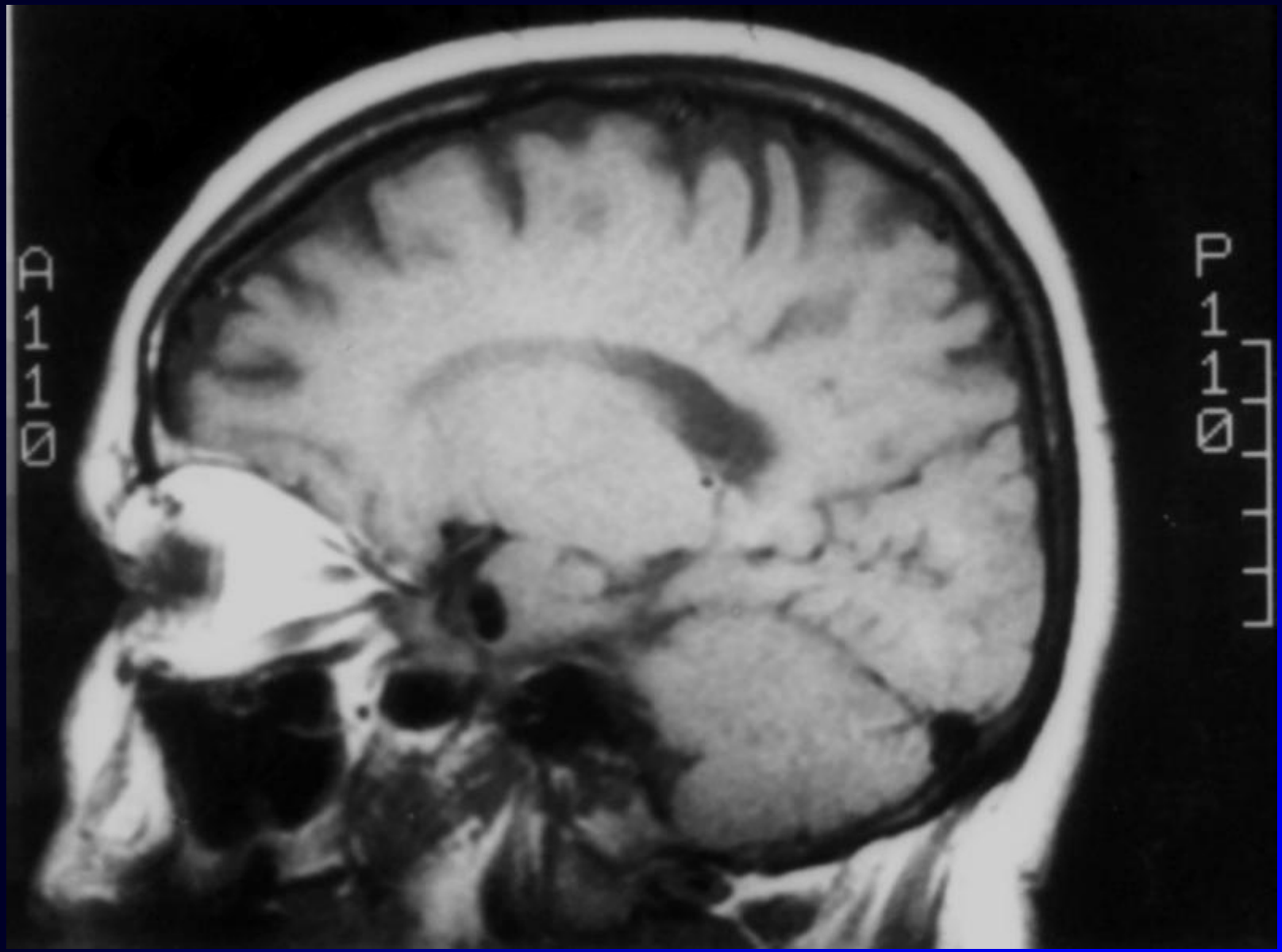
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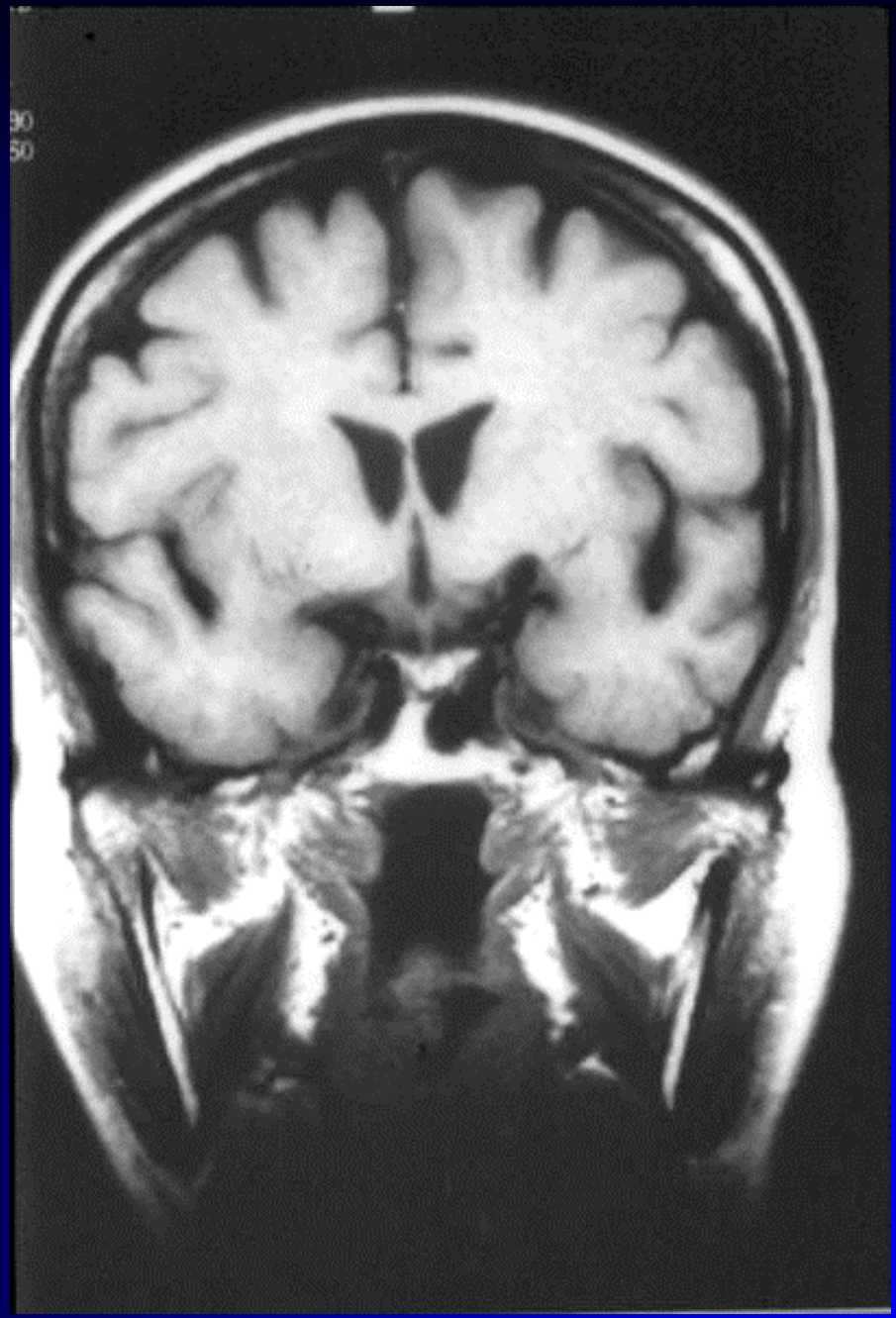


CEREBROVASCULAR DISEASE:

- Cerebral Infarction
  A necrotic process secondary to insufficient local blood flow or tissue anoxia; "ischemic necrosis" or, more accurately, "oligemic".

Infarction

- INFARCTS with BLOOD- HEMORRHAGIC
- INFARCTS w/o BLOOD- ISCHEMIC

Classification of Infarction

- HISTOLOGY
- MORPHOLOGY
- ETIOLOGY

Classification of Infarction

- HISTOLOGY
  - -Hemorrhagic
  - -Anemic

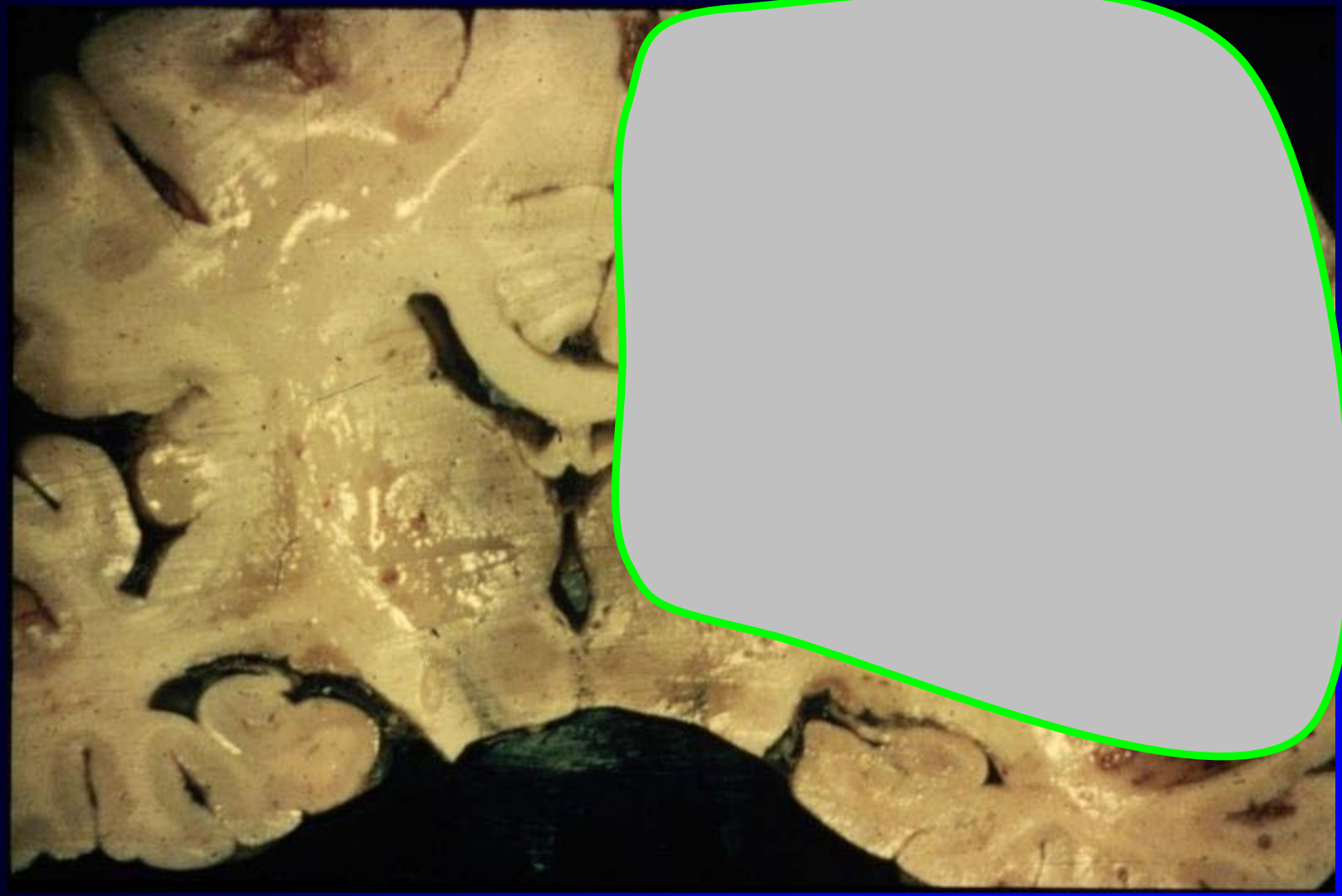
- MORPHOLOGY
  - -Wedge (arterial)
  - -Border Zone (flow)
  - -Laminar, etc.

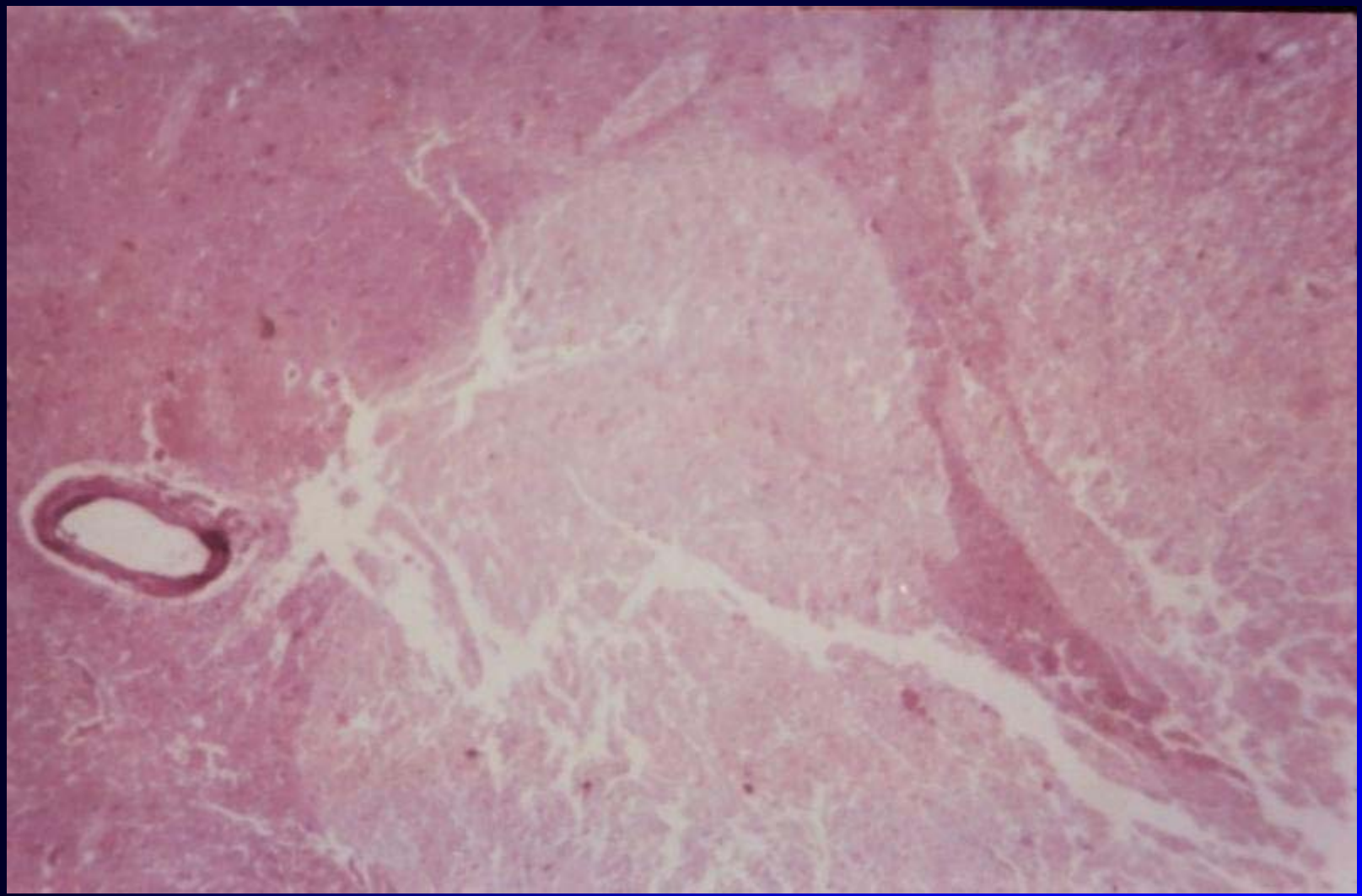
- ETIOLOGY
  - -Arterial Thrombosis
  - -Arterial Embolus, Herniation
  - -Venous Thrombosis
  - -Hypotension, Anoxia

NON-HEMORRHAGIC INFARCTION

Ischemic Infarction

- PALE, WHITE
- BLAND
- ANEMIC, ISCHEMIC



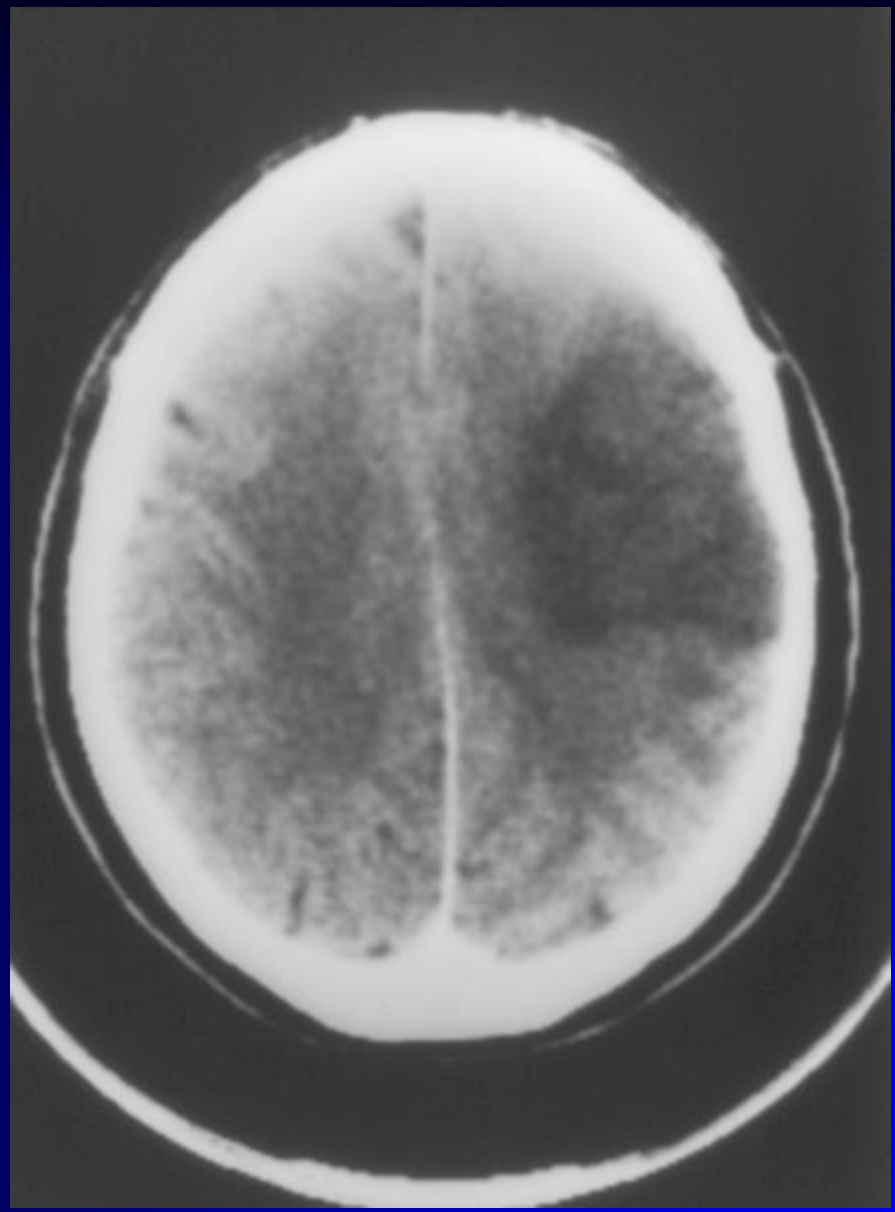


Ischemic Infarction

- Location of Infarction
  - -MCA 62%
  - -PCA 14%
  - -ACA 5%
  - -Posterior fossa 5%
  - -Multiple, Watershed 14%

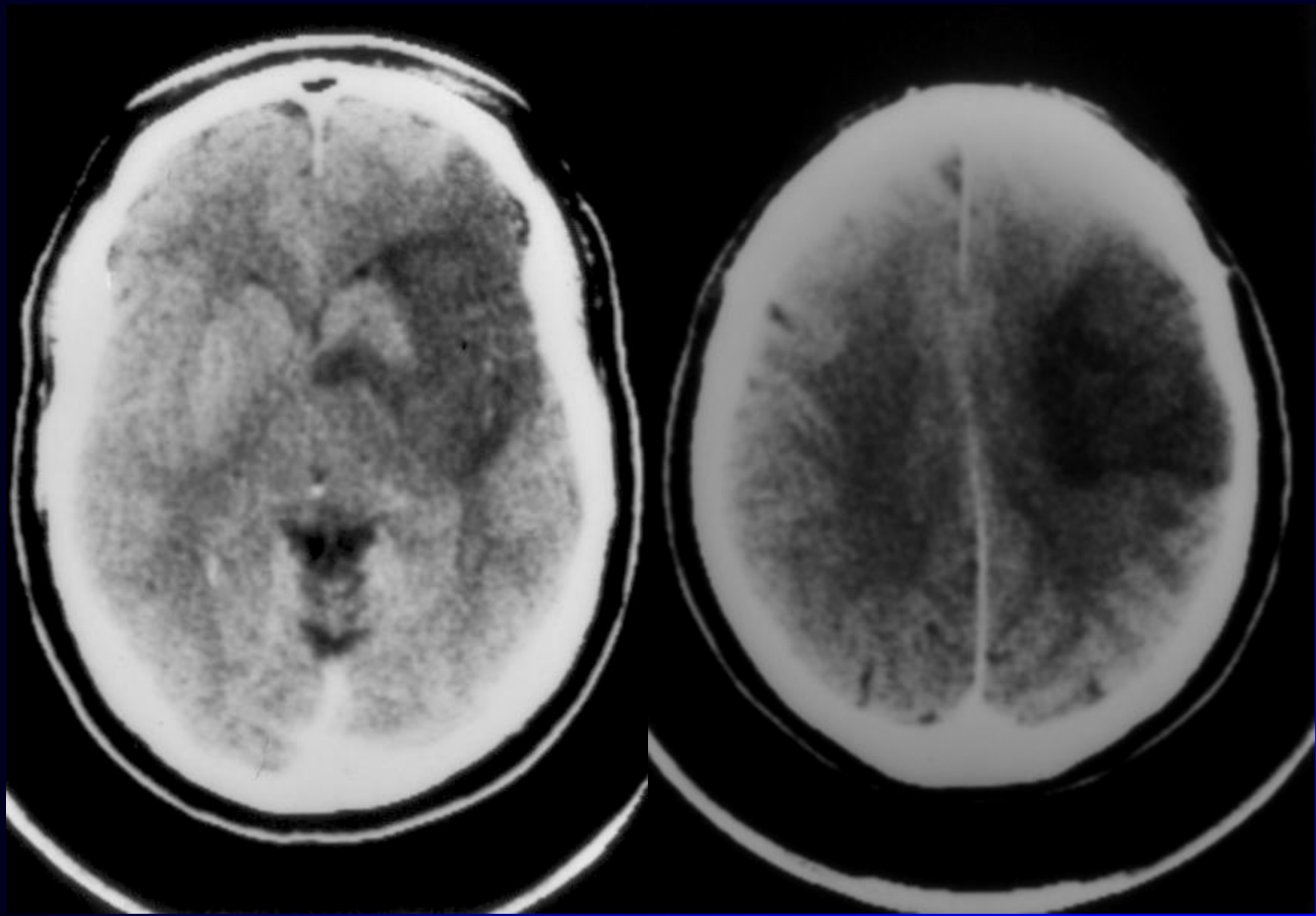
Ischemic Infarction

- Increased Water (Edema)
  -Low density on CT
  -Bright on T2W MR
  -Gray and/or White Matter
- SHAPE - vascular wedge
- MASS EFFECT
  -may be minimal
  -cortical (effacement of sulci)
  -Peaks at 3-5 days



Imaging of Infarction

- EDEMA
  - Cytotoxic (Gray and White)
  - Vasogenic (White only)
- Enhancement
  - Abnormal BBB
  - "Luxury Perfusion"



Ischemic Infarction

- Increased Water
  - Cyotoxic edema
- Prolonged T1/T2, Decreased specific gravity of tissue
- Decreased Attenuation on NCT
- +/- Sl. darker on T1W MRI
- Sl. brighter on PD
- Bright on T2W MRI
- Bright on DWI, Dark on ADC Map

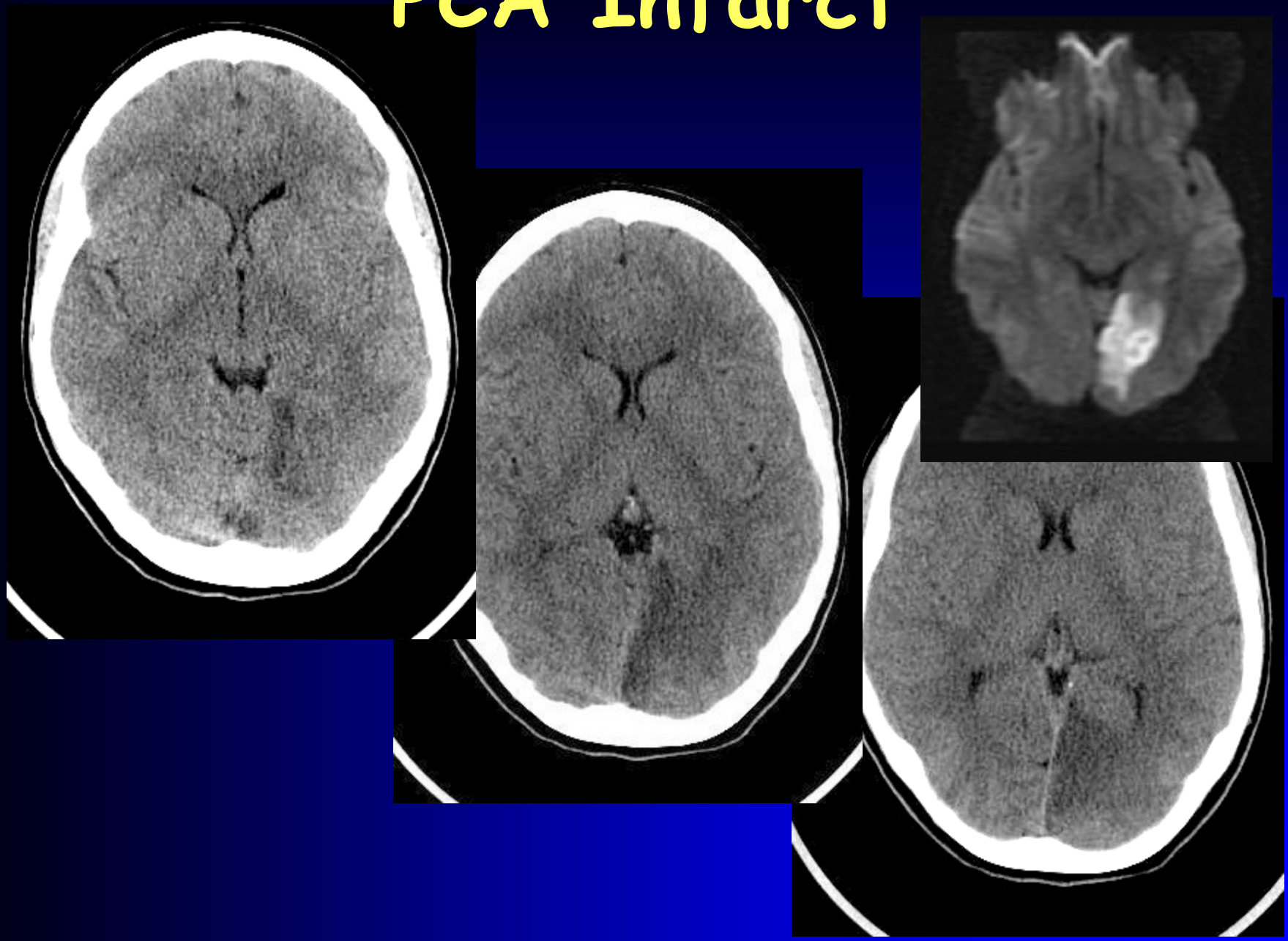
PATTERNS OF EDEMA

AJNR 3:251-255, 1982

- VASOGENIC SPREAD
  (neovascular edema)
  4/339 (1.2%) INFARCTS

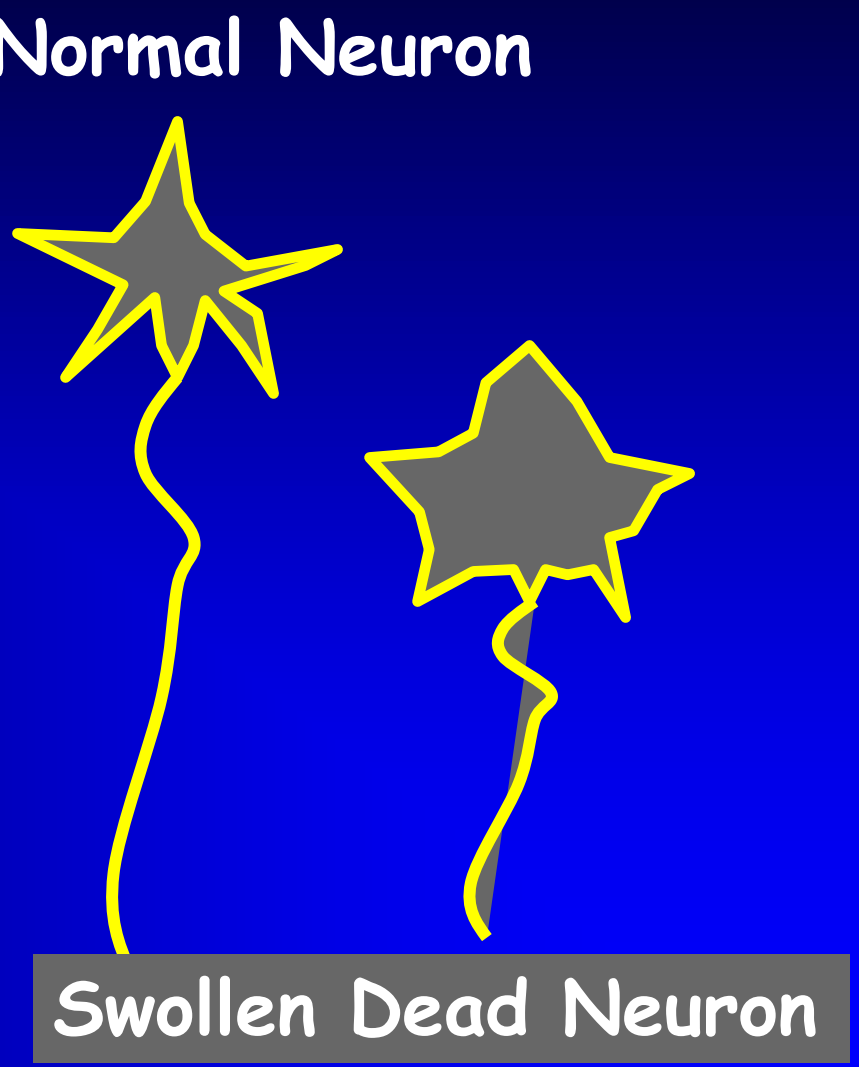
- GRAY MATTER EDEMA
  (Cytotoxic edema)
  2/155 (1.3%) TUMORS

PCA Infarct



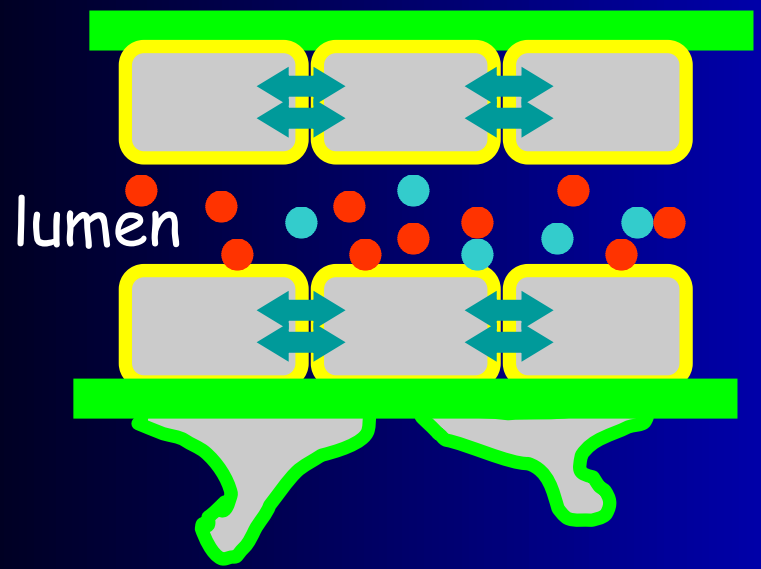
Cytotoxic Edema

- Normal Na+ ⇔ K+ pump
  - K goes In
  - Na goes Out
- Energy Dependent
  - Glucose
  - O2
  - ATP

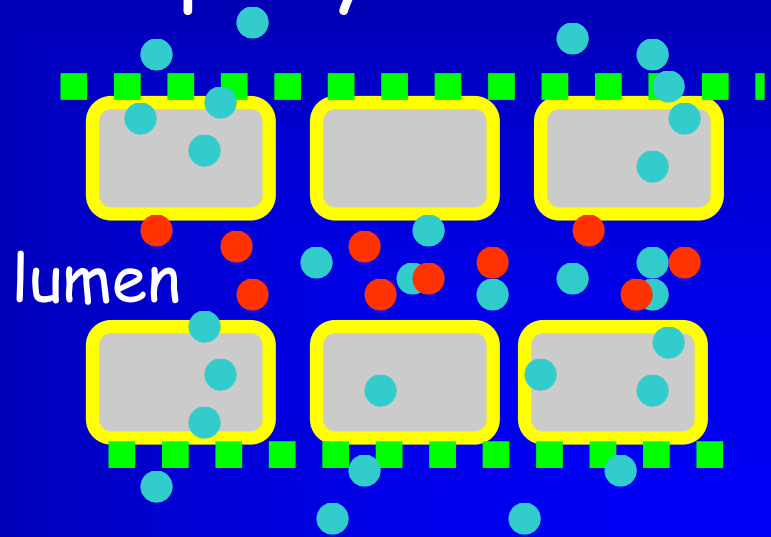


Ultrastructure of BBB

- Neural capillary
  - continuous BM
  - tight junctions
  - astrocytic feet
  - no pinocytosis



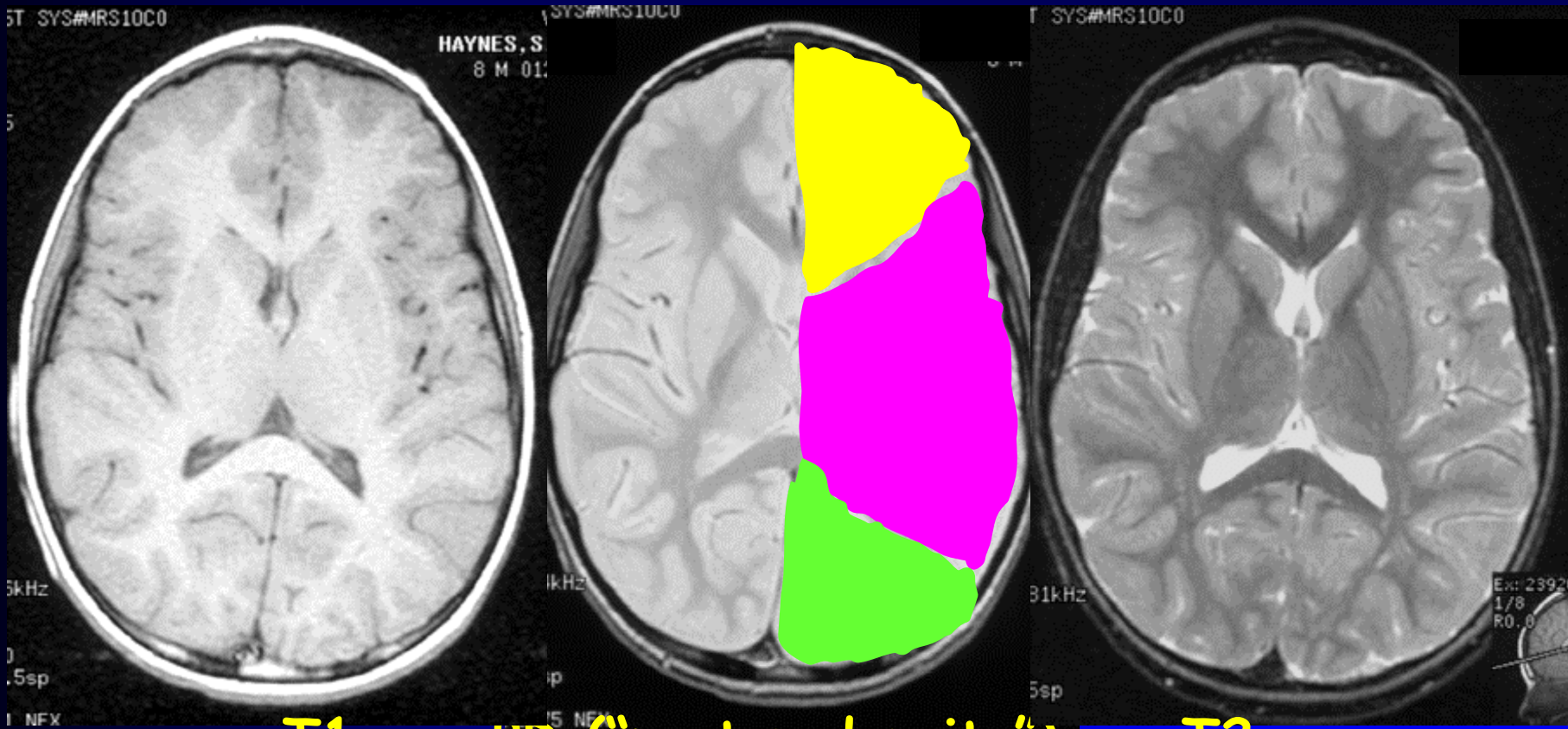
- Non-neural or ABBB
  - fenestrated BM
  - intercellular gaps
  - no astrocytic feet
  - pinocytosis



Edema in Infarction

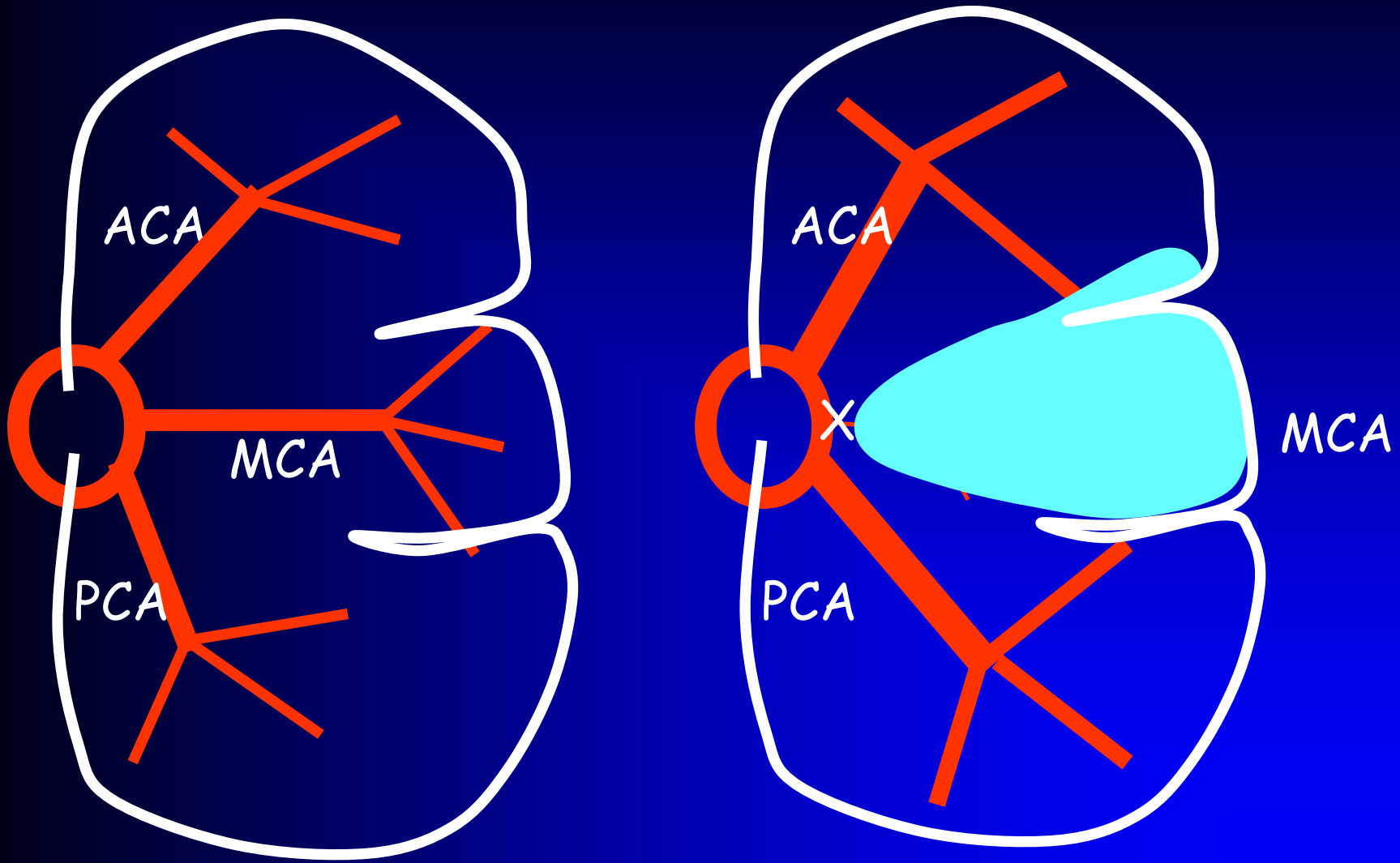
- Cytotoxic Edema
  - Gray Matter
  - White Matter
- Vasogenic Edema
  - Limited by reduced/absent perfusion
  - White Matter Only
  - Spreads away from damaged vessels

Vascular Territory

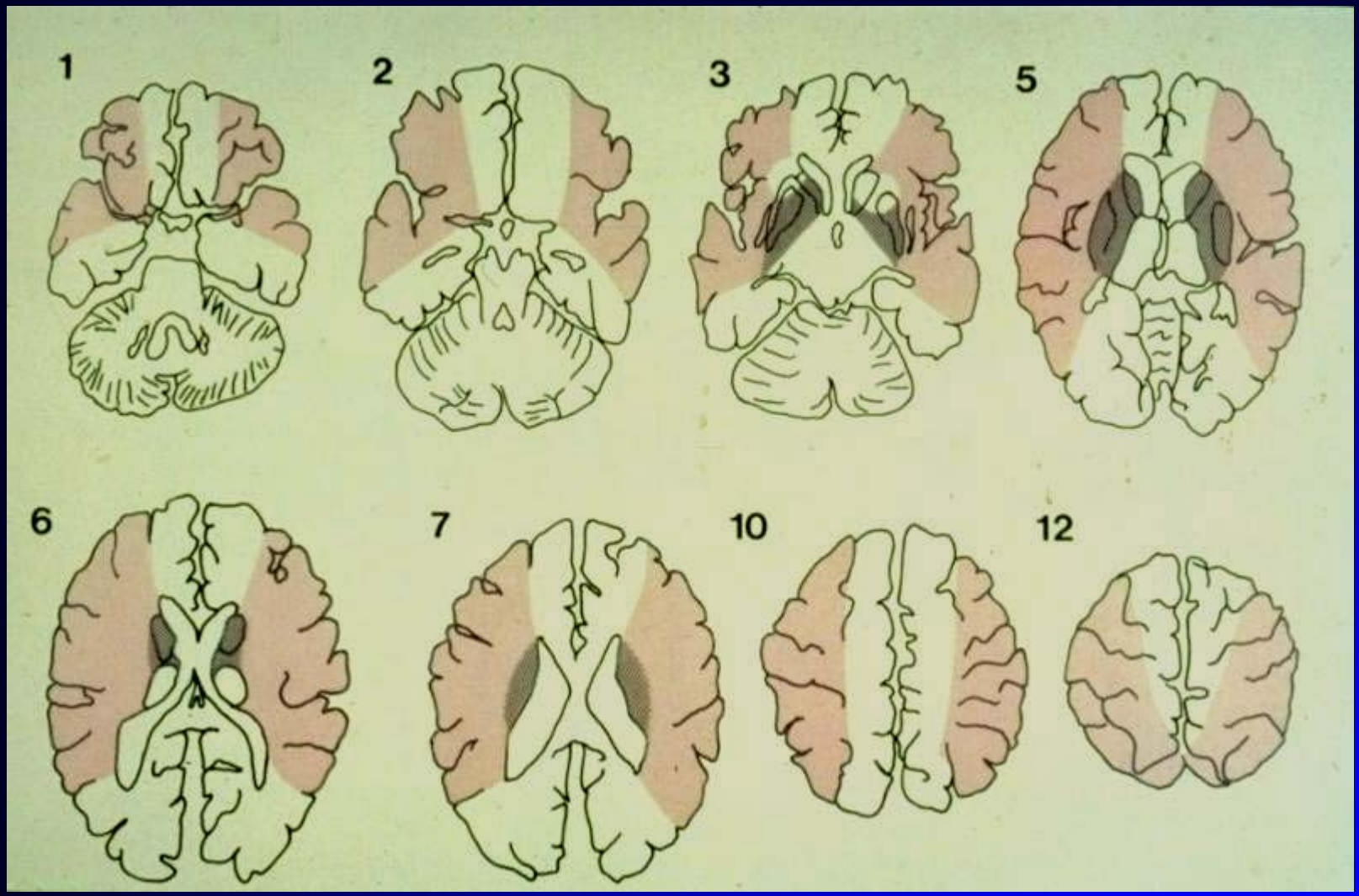


T1 PD ("proton density") T2

Single Vessel Occlusion - MCA



Flow is redistributed into patent vessels via Circle of Willis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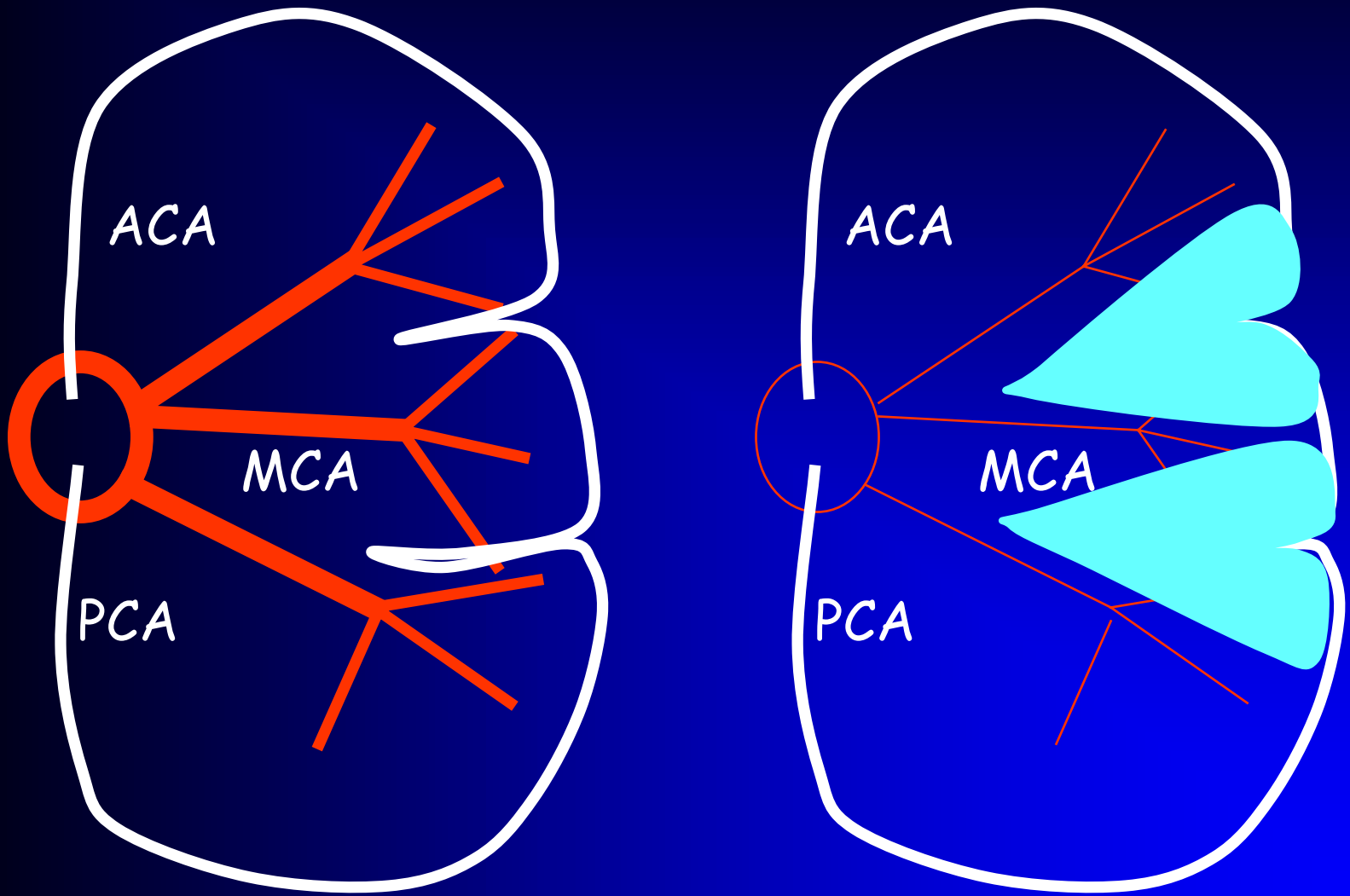
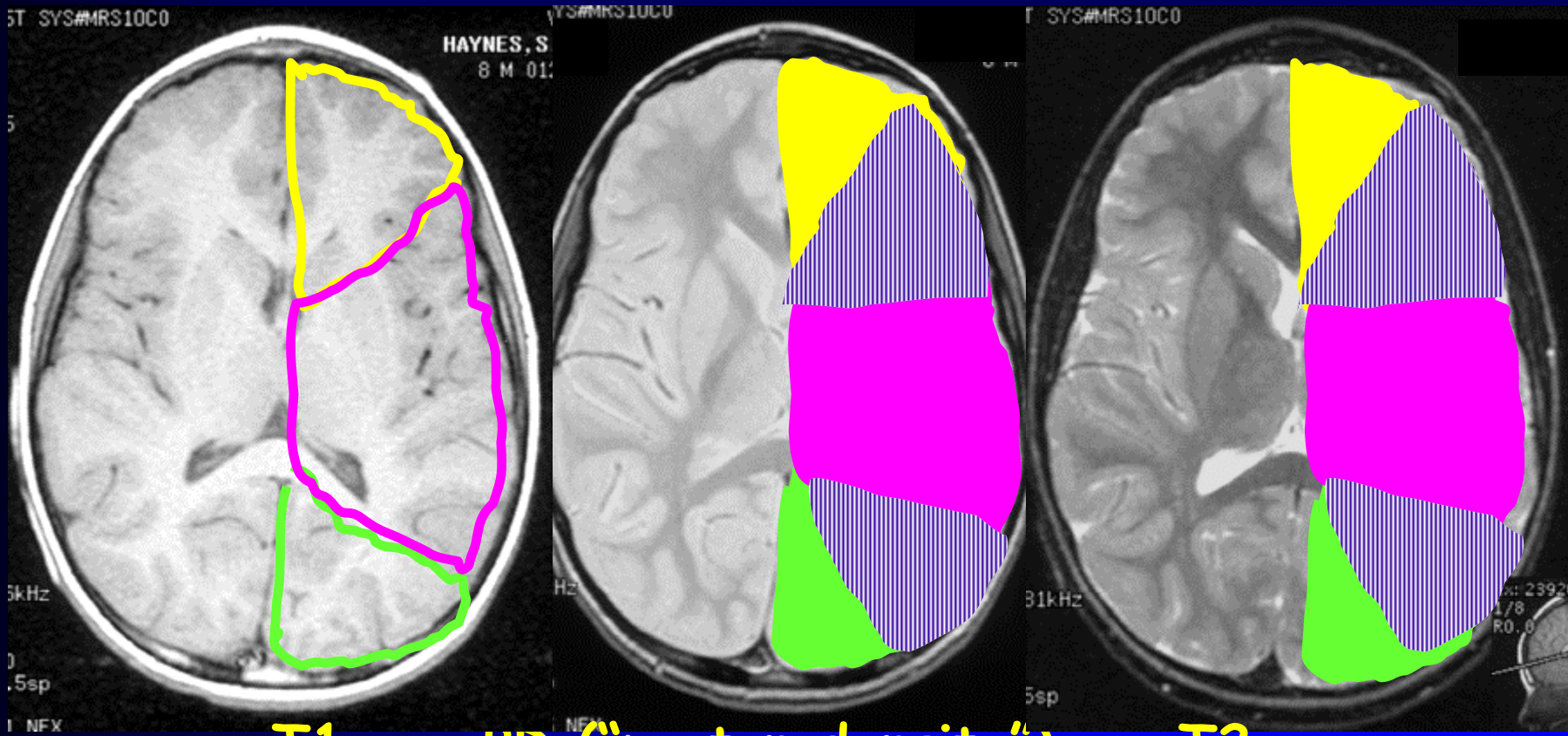
Hypotension and "Borderzone"

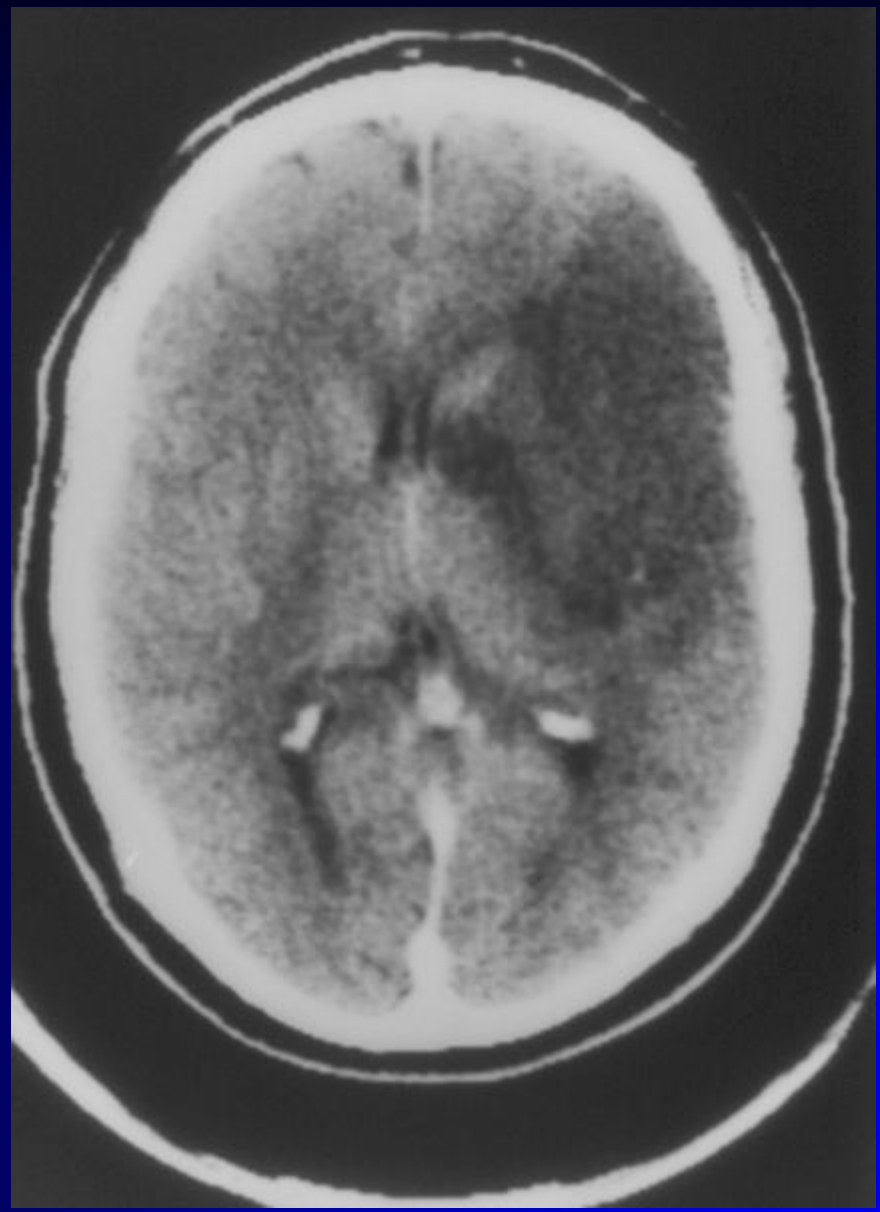


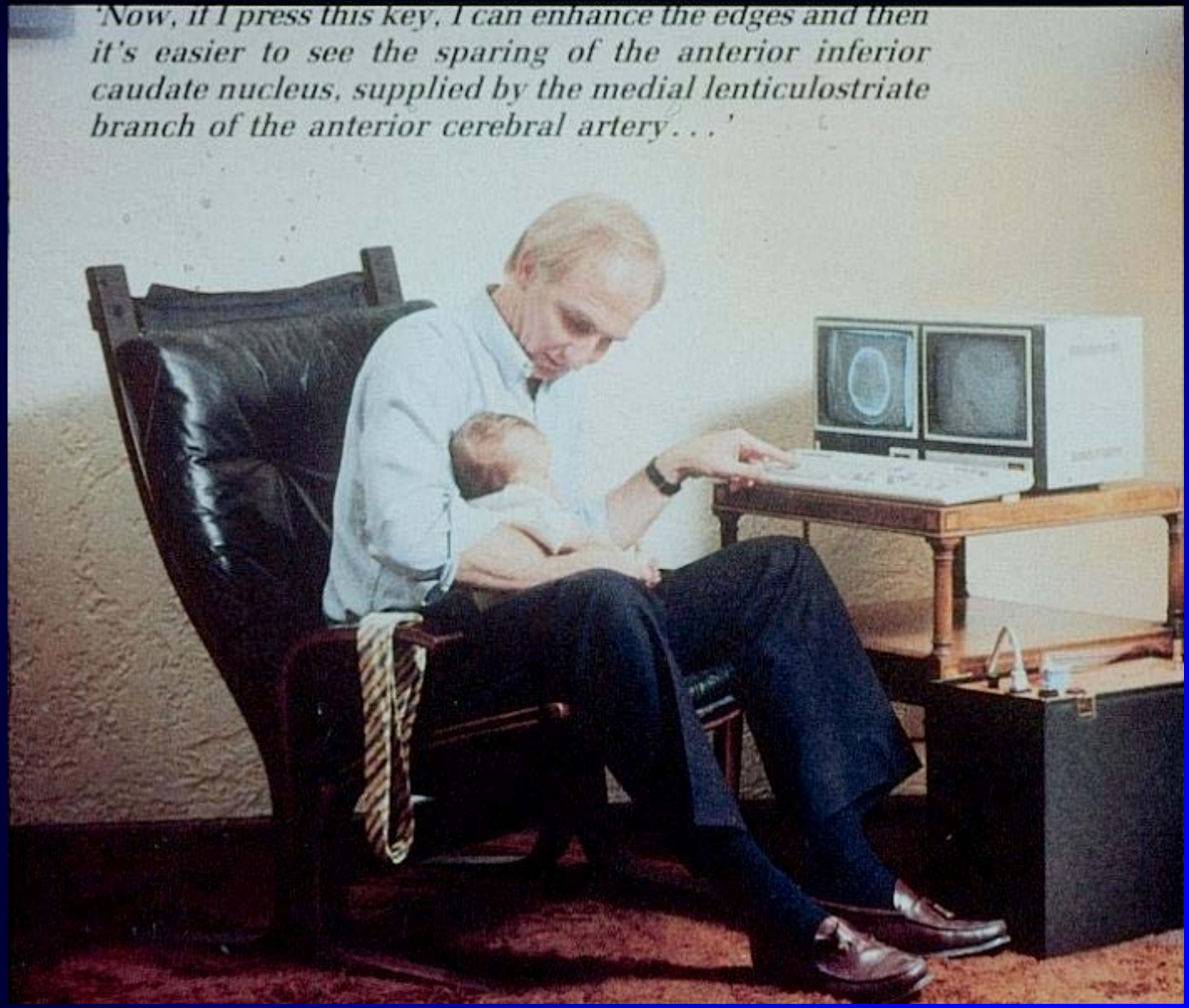
Hypotension and "Borderzone"

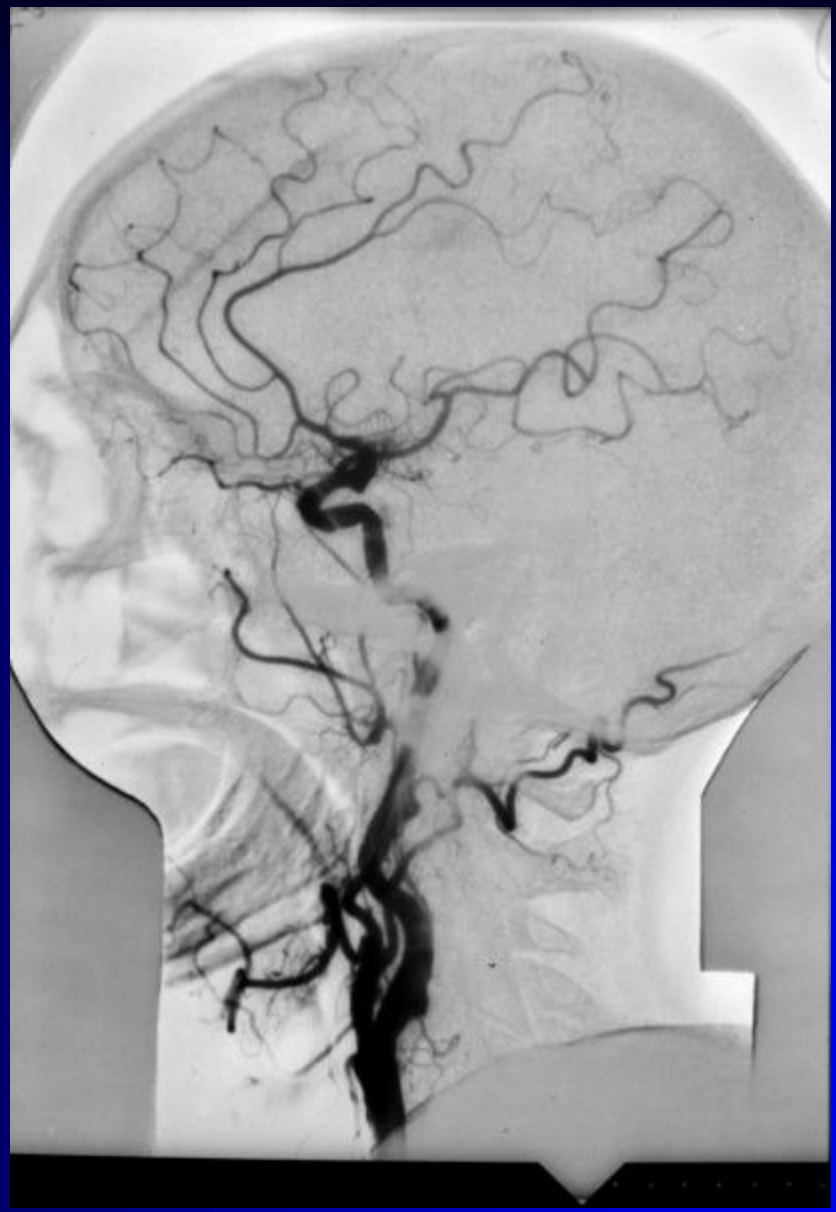


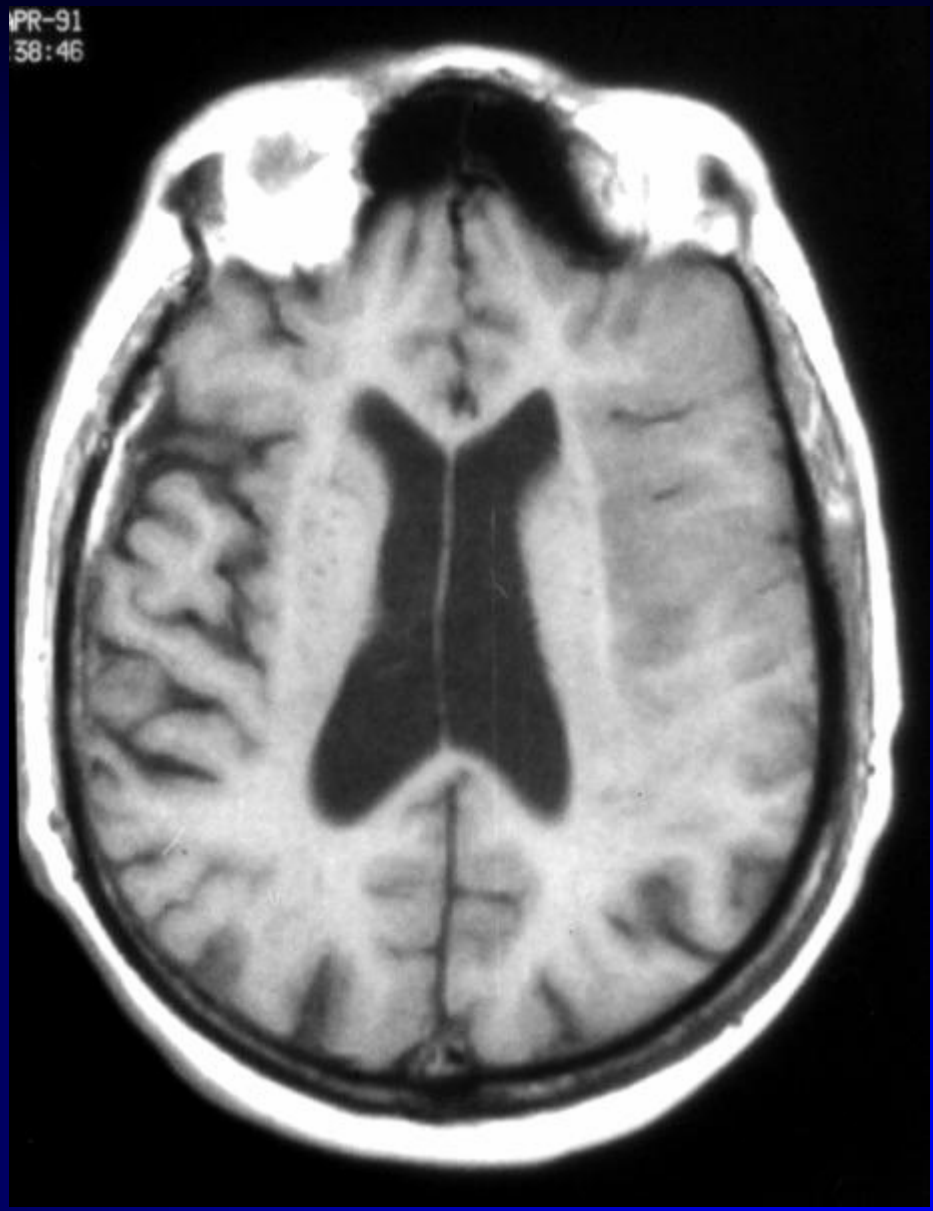
T1 PD ("proton density") T2

Border Zone Infarcts




'Now, if I press this key, I can enhance the edges and then it's easier to see the sparing of the anterior inferior caudate nucleus, supplied by the medial lenticulostriate branch of the anterior cerebral artery. . . '

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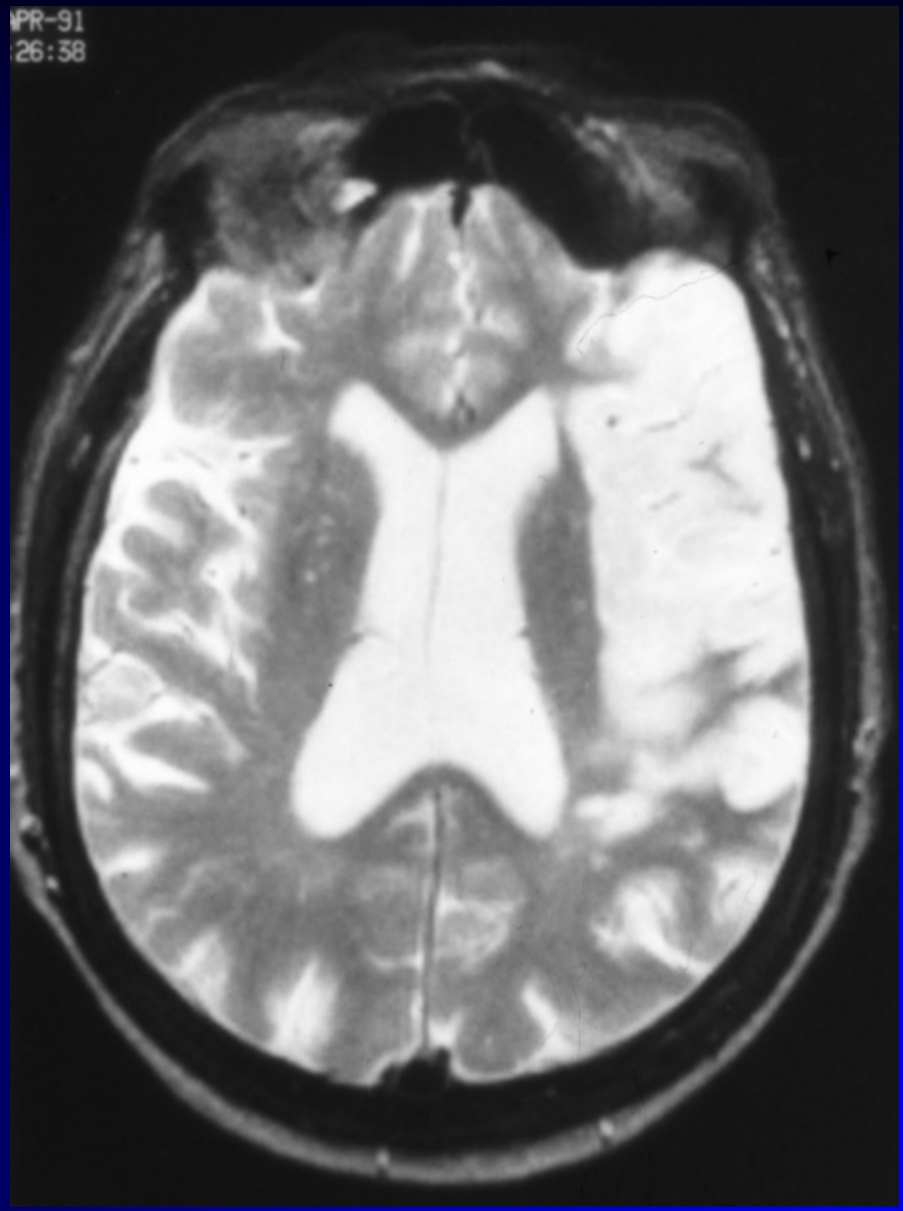
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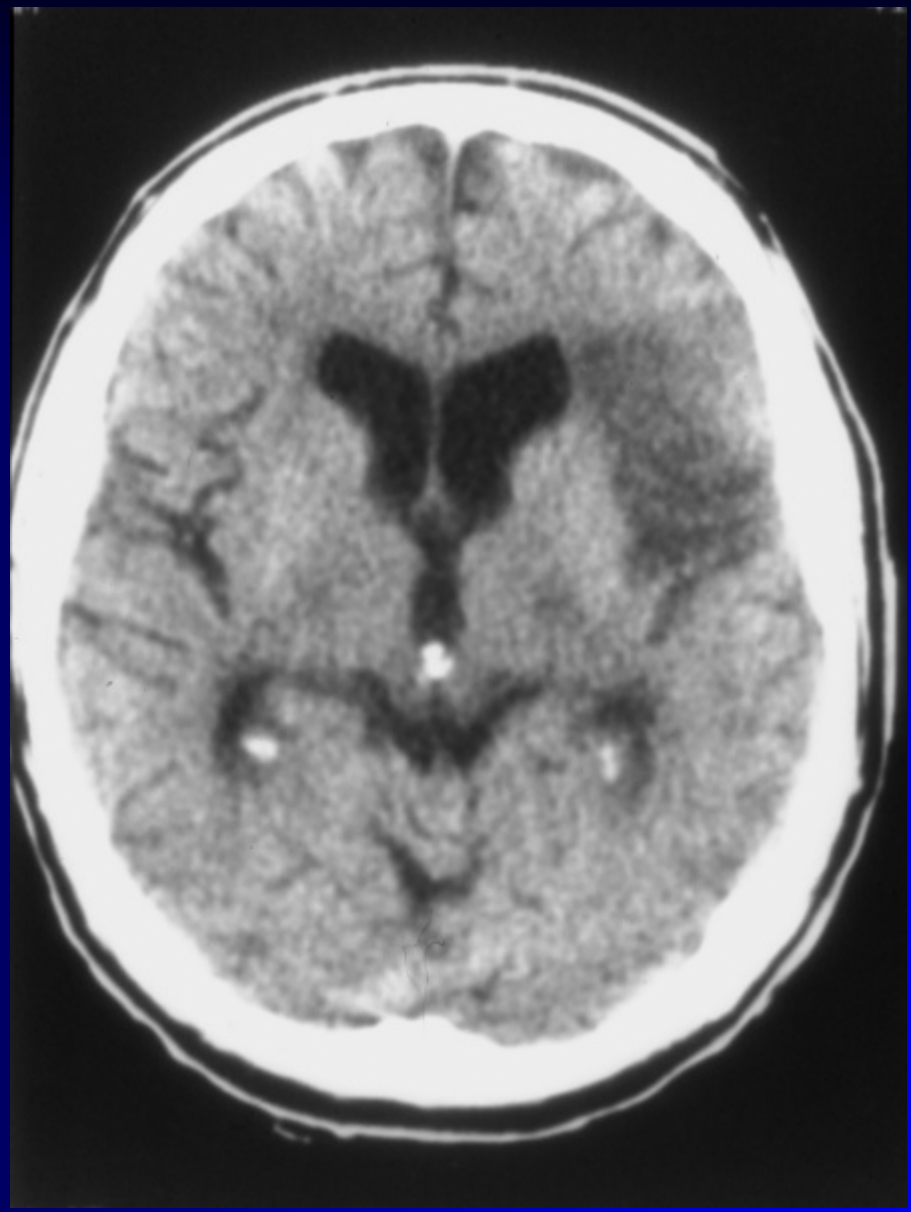
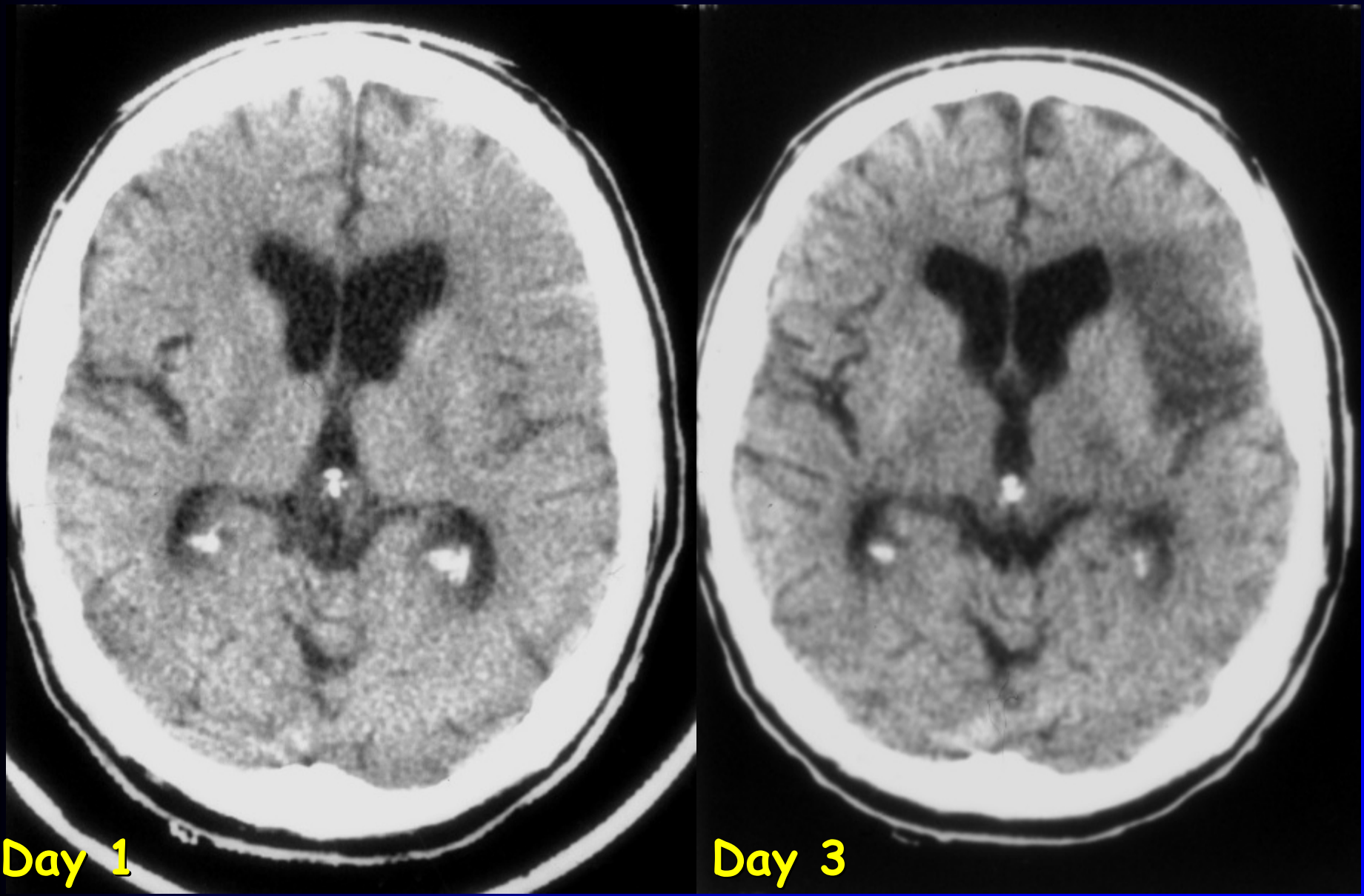
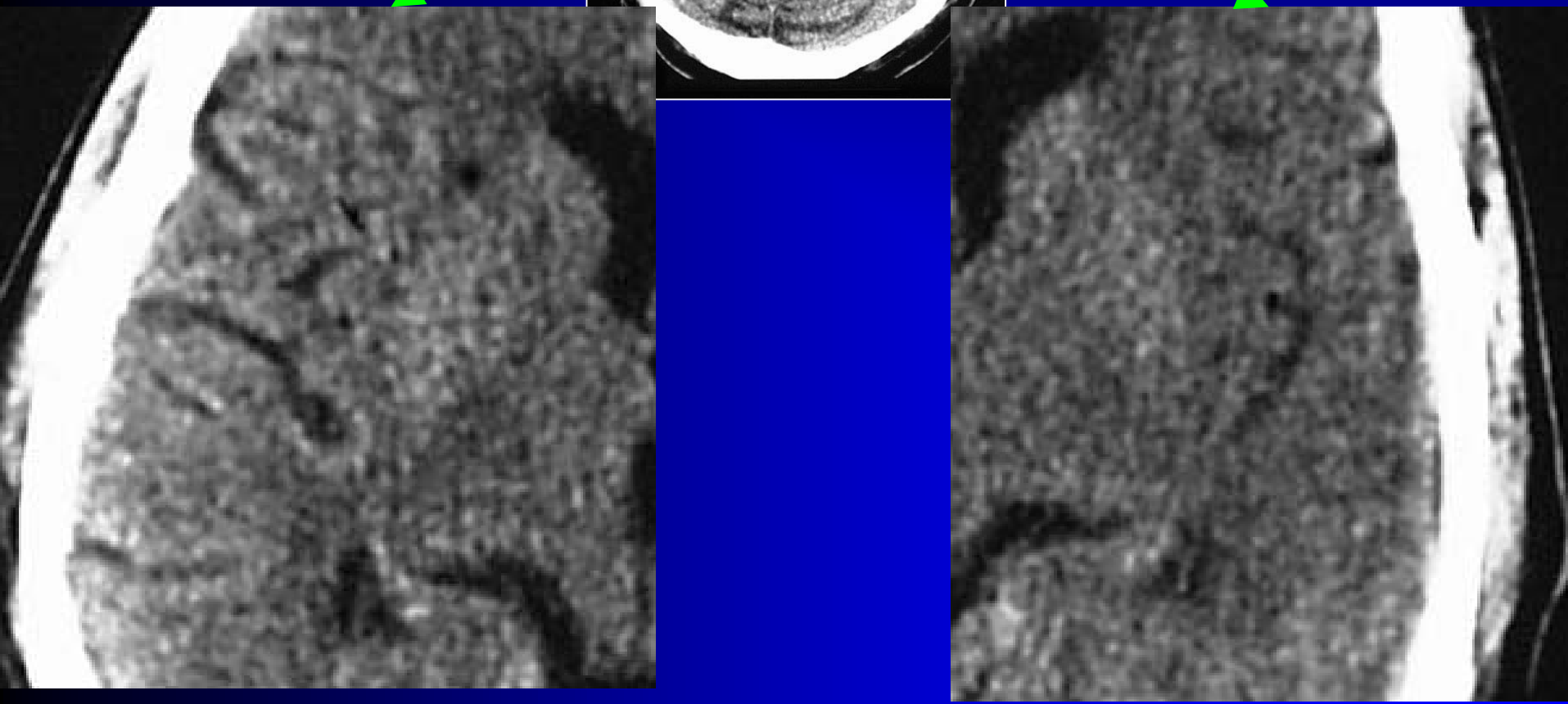



Day 1
Day 3

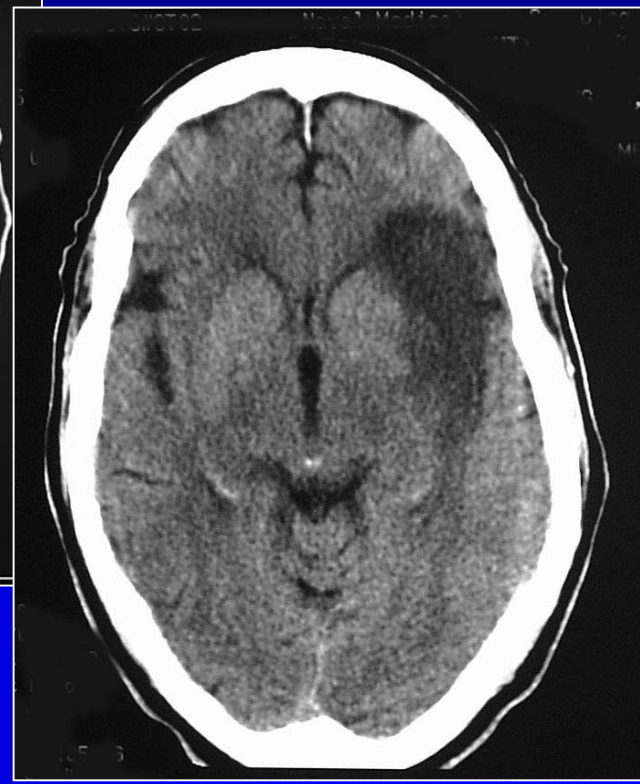
Early MCA stroke



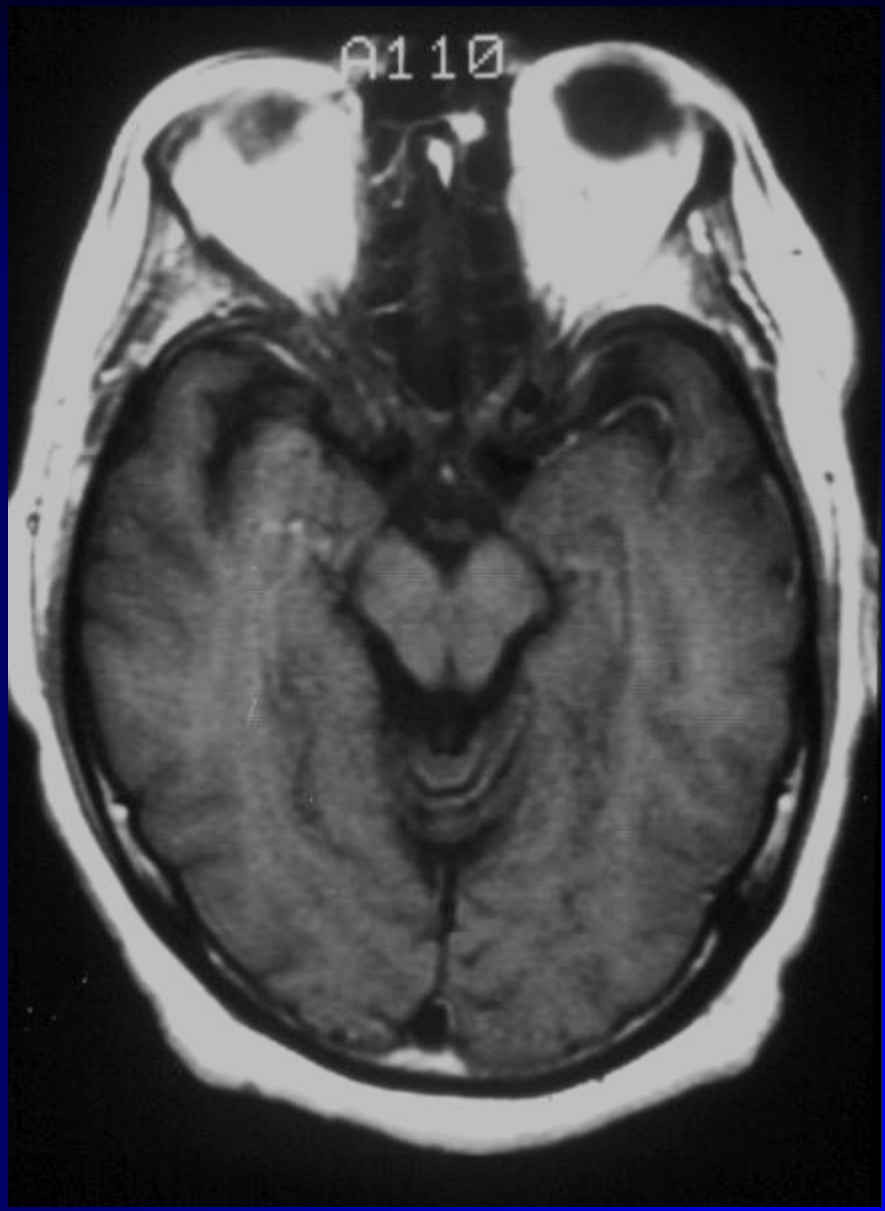
CVA : Progression of CT findings

Day 1

Day 2



Day 5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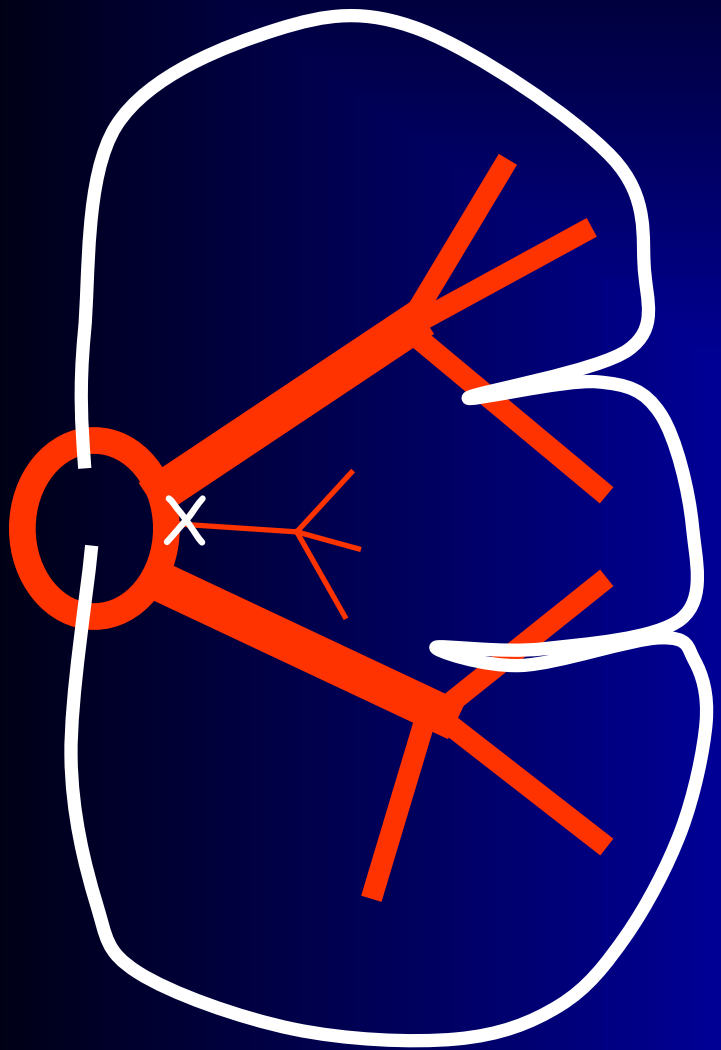
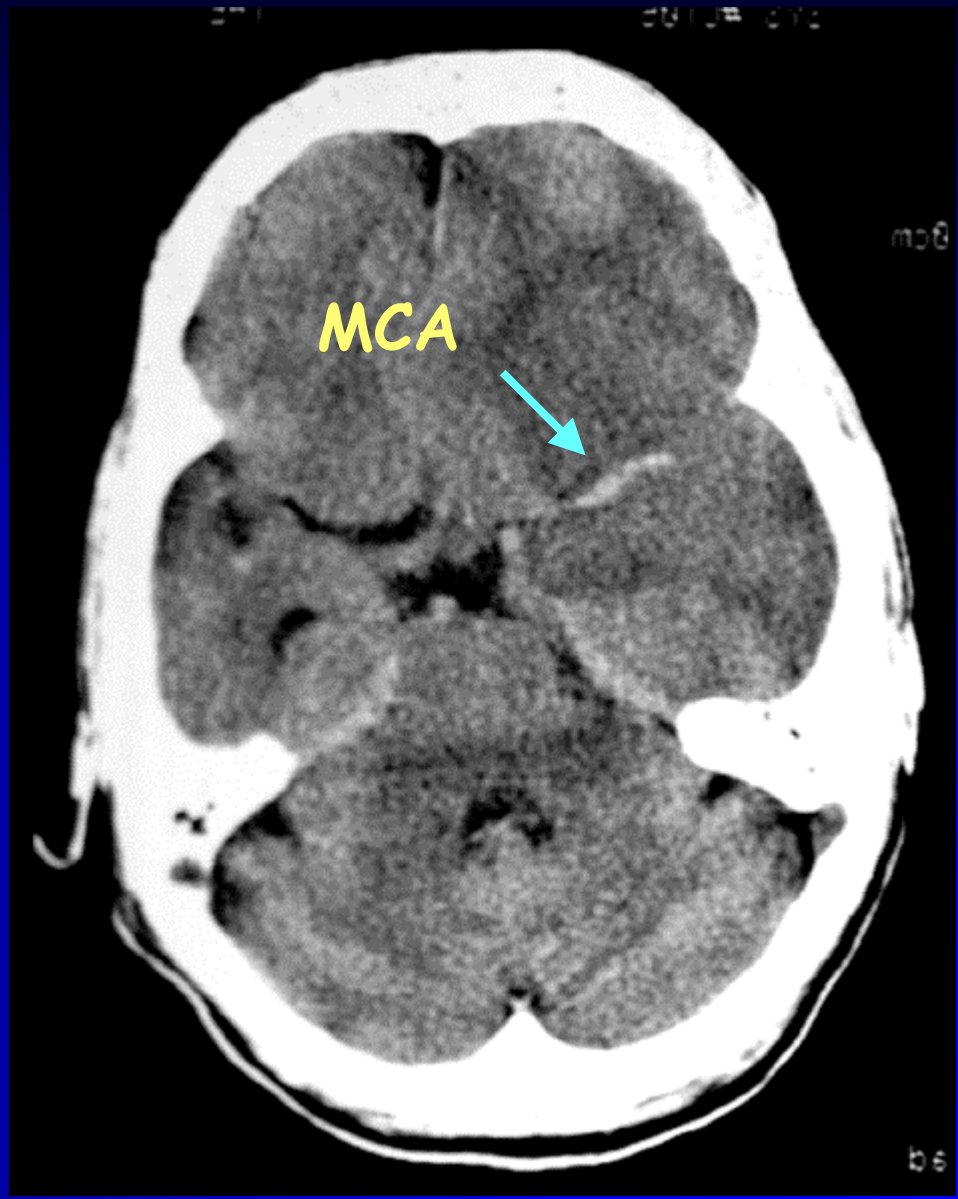
Carotid Thrombosis => MCA Clot



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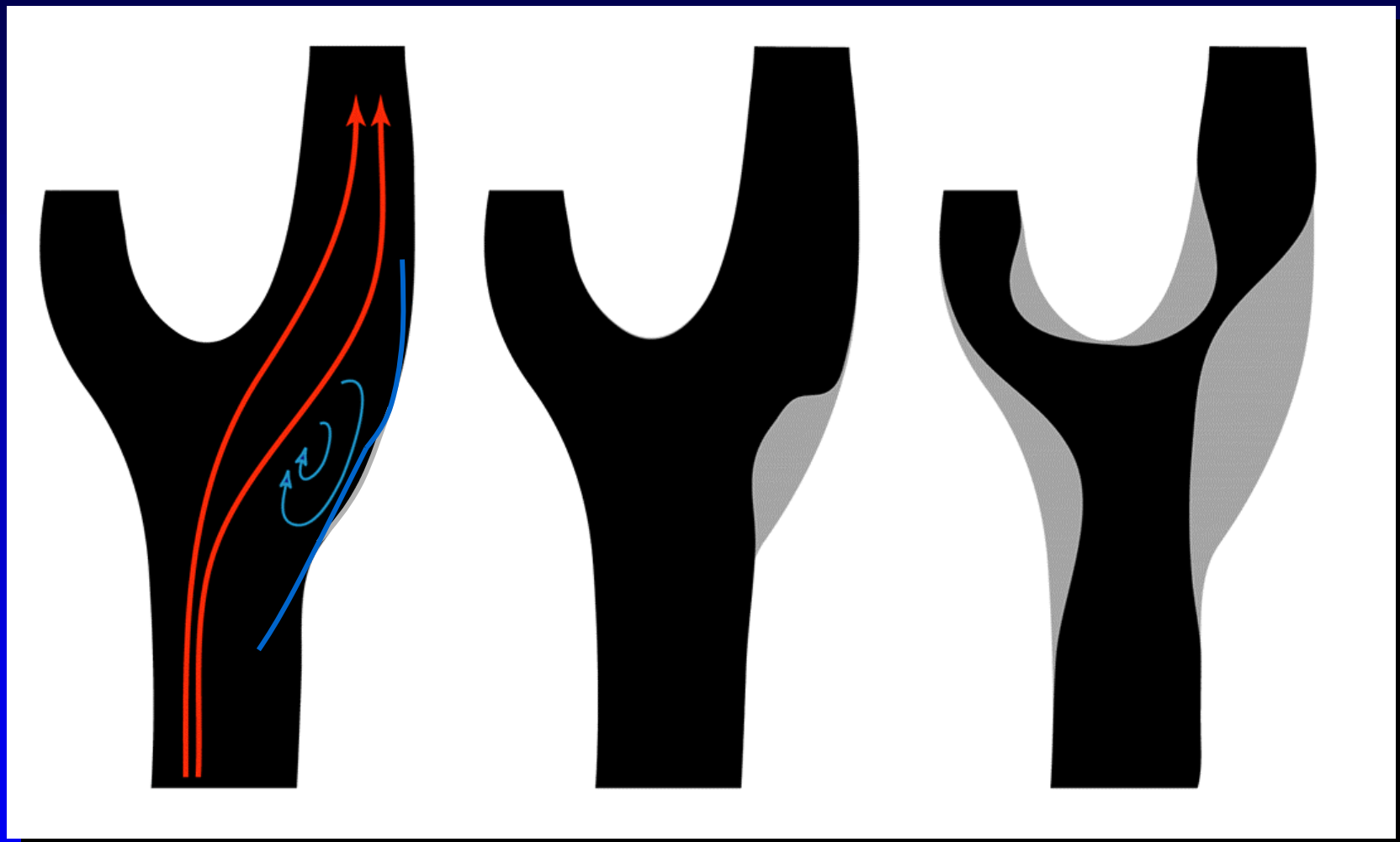
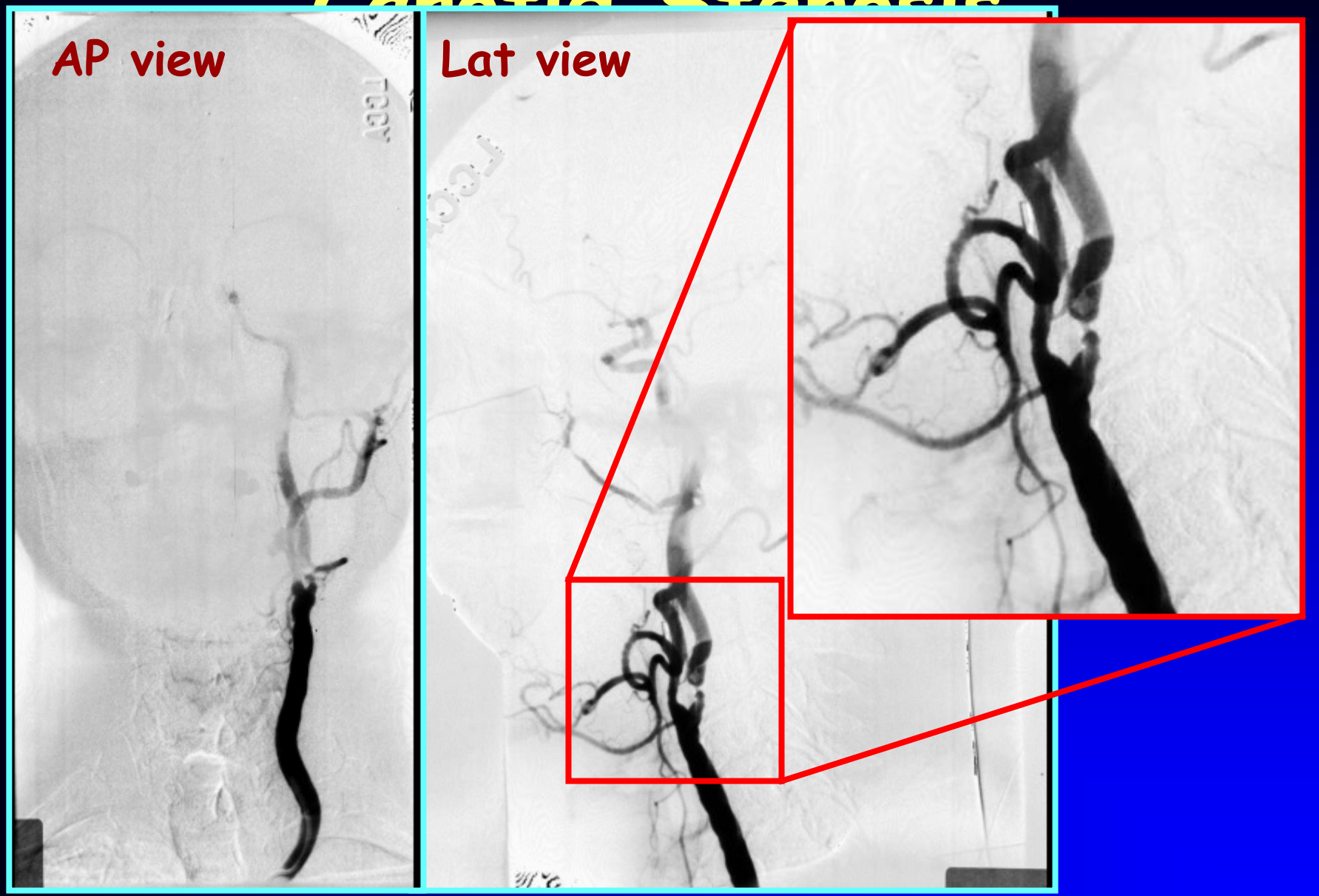
Carotid Bifurcation



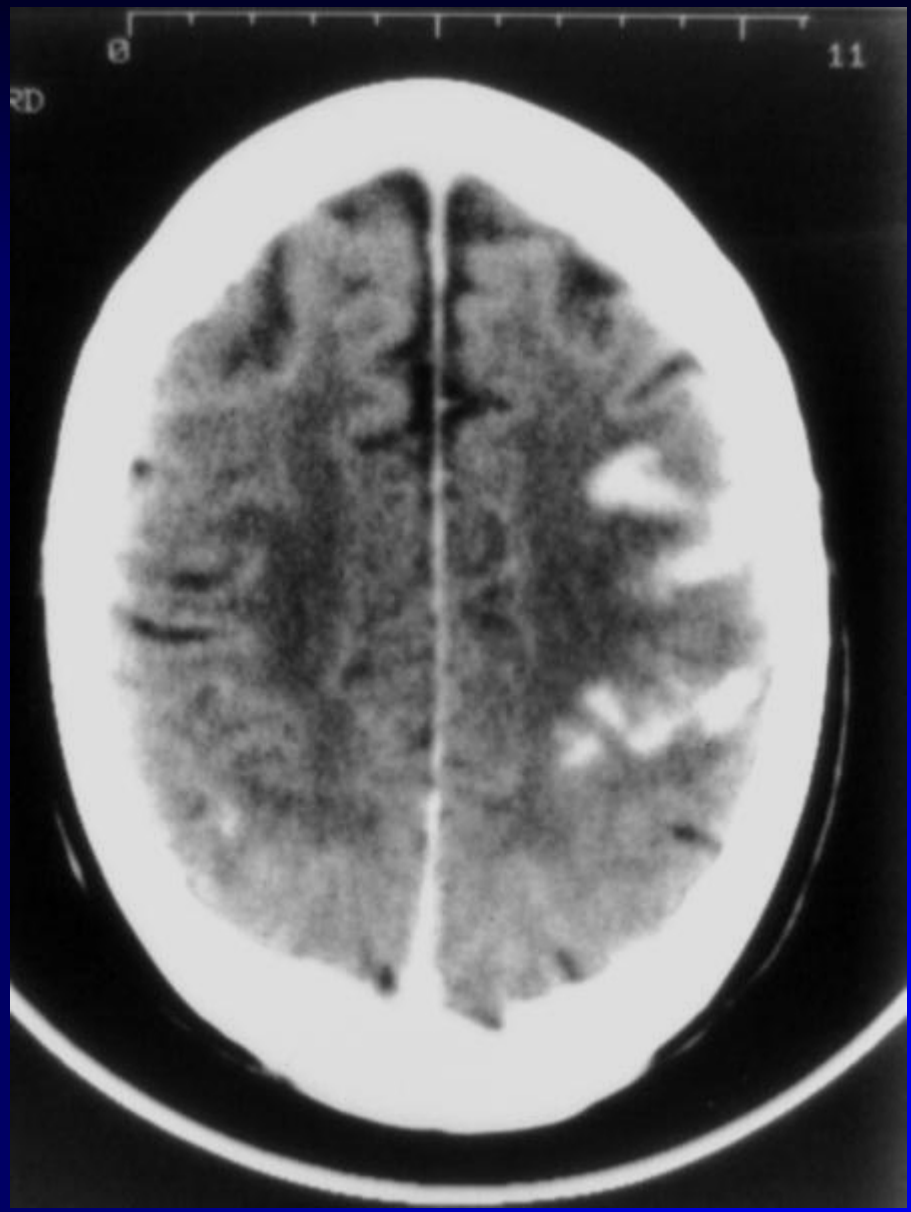
Early Turbulence => scarring => stenosis Late

Carotid Stenosis



MRA - Magnetic Resonance Angiography

- Cirrhosis
- Hyperbilirubinemia
- Bilirubin bound to Albumin
- Albumin can't cross the Blood-brain-barrier
- BBB is abnormal in infarct
- Mostly gray-matter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RD

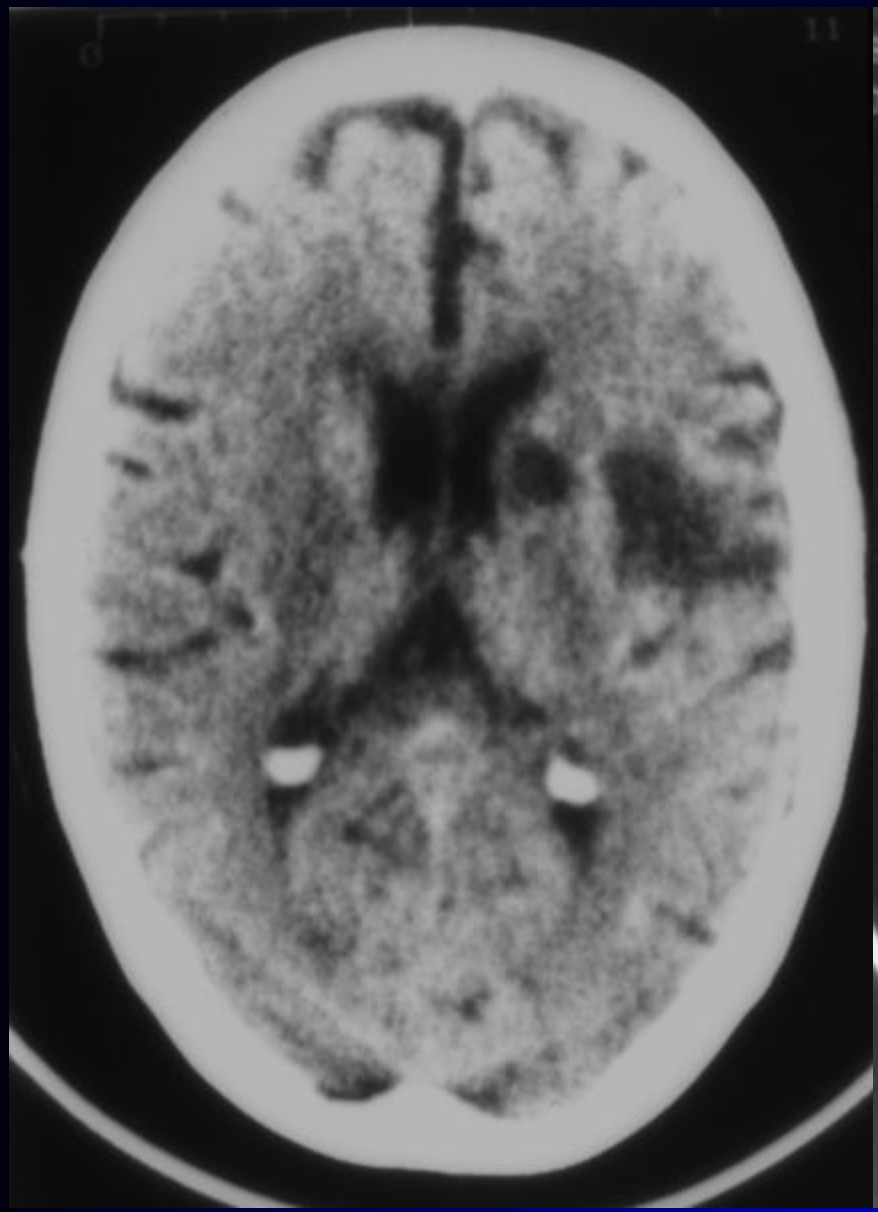
WHAT ABOUT CONTRAST?

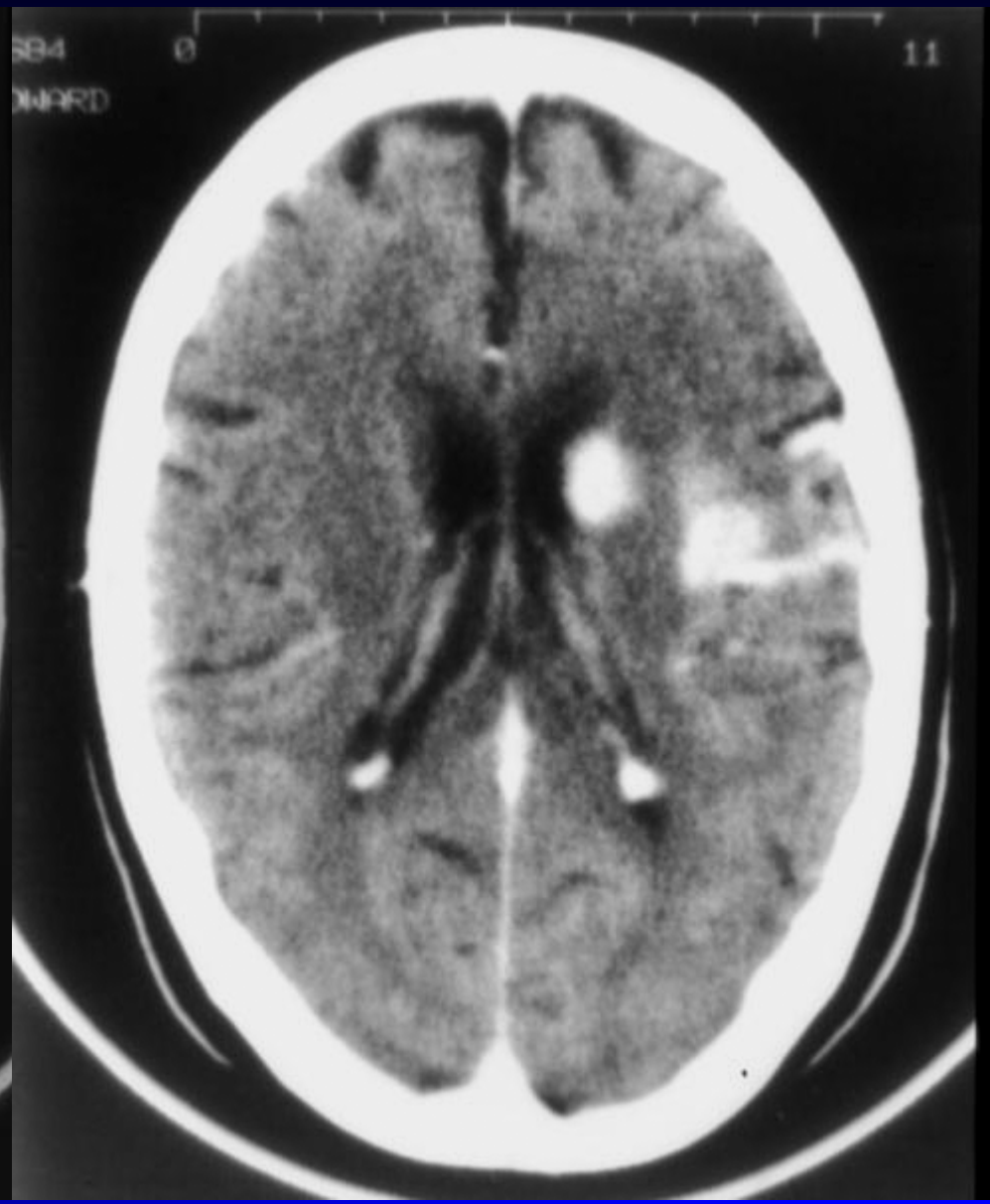
- If an infarct is caused by a loss of blood flow, then how do water and contrast get there?


SE
TR:550
TE:9/Fr
EC:1/1 16kHz
HEAD
FOV:24x18
6.0thk/1.0sp
256x192/1 NEX

Ischemic Infarction Contrast Enhancement

- "Ischemic Enhancement"
  -Early ("immediate") uncommon
  -Early (4-6 hrs.)
- SUBACUTE (7-12 days)
  PEAK in intensity and incidence
- LATE (6-9 months)
  - Fades Away as Atrophy Develops
- PERSISTENT (1-2 years) uncommon

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Ischemic Enhancement Misnomer?

- Contrast delivered by blood flow
- Gray Matter - "Gyriform" pattern
- Etiology:
  - increased flow (loss of Autoregulation)
  - Abnl. BBB outside infarct
  - Abnl. BBB during healing of infarct
- "Luxury Perfusion"
  - Angiographic term
  - increased perfusion/vascularity
  - implies re-perfusion

- *Re-perfusion*
  -collaterals (early)
  -recanalization (late)
  -healing (late)

Ischemic Infarction Contrast Enhancement

- "Ischemic Enhancement"
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- SUBACUTE (7-12 days)
  PEAK in intensity and incidence
- LATE (6-9 months)
  - Fades Away as Atrophy Develops
- PERSISTENT (1-2 years) uncommon

"Ischemic Enhancement"

- (Reperfusion in min. to hrs.) "LUXURY PERFUSION"
- BBBB (> 4 - 6 hrs. of ischemia)
  - original capillaries affected
  - New Capillaries (7-10 days) grow into infarct for healing w/o BBB

"LUXURY PERFUSION"

- *Angiographic observation*
- *FOCAL HYPEREMIA (relative or absolute)*
- *CORTICAL BLUSH and EARLY VEINS shunting*
- *LOSS OF AUTOREGULATION (caused by inc. pCO2, decr. pH/pO2)*
- *TRANSIENT minutes to hours, ends in 3-5 days*

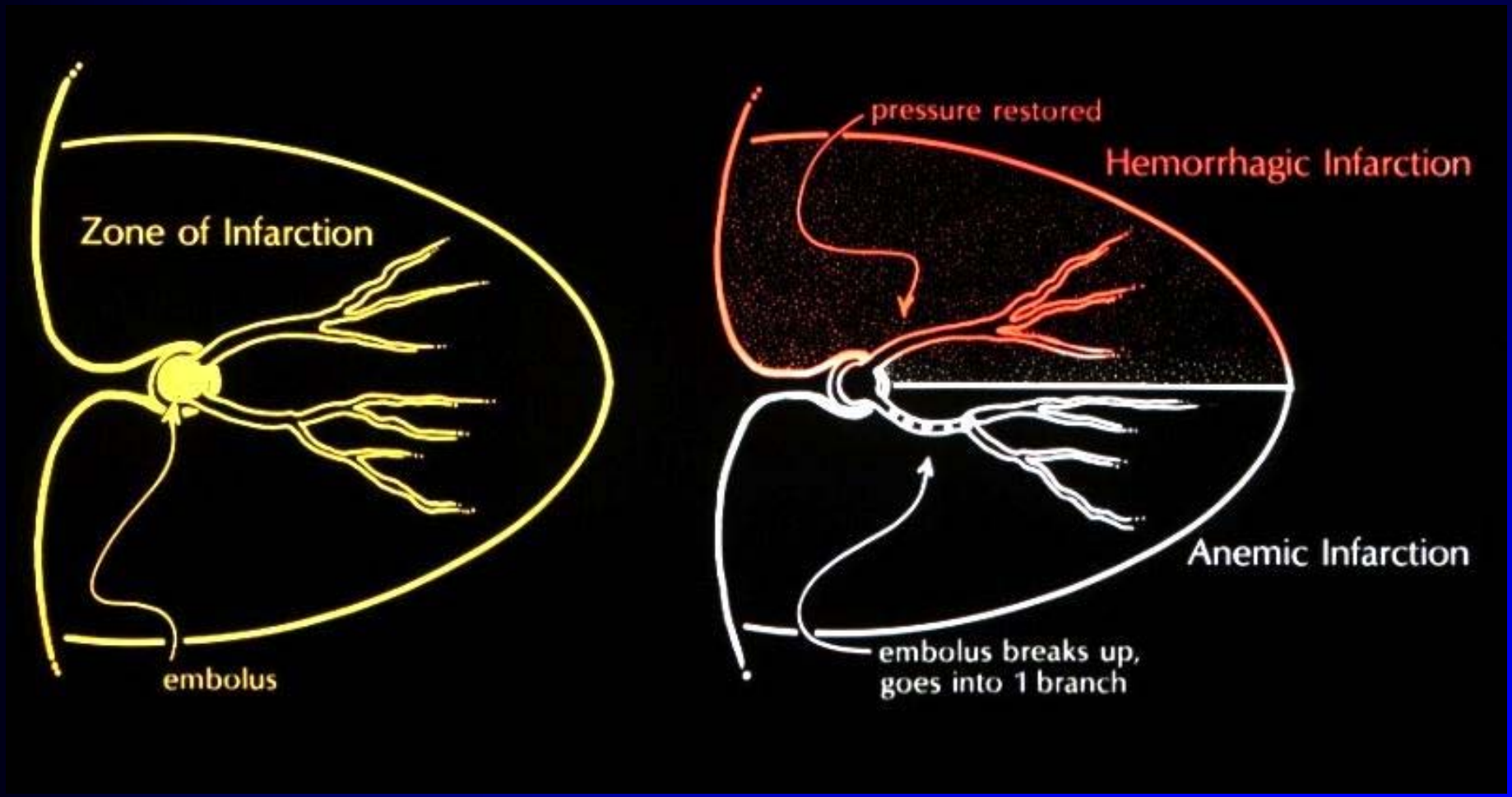
CONTRAST TOXICITY

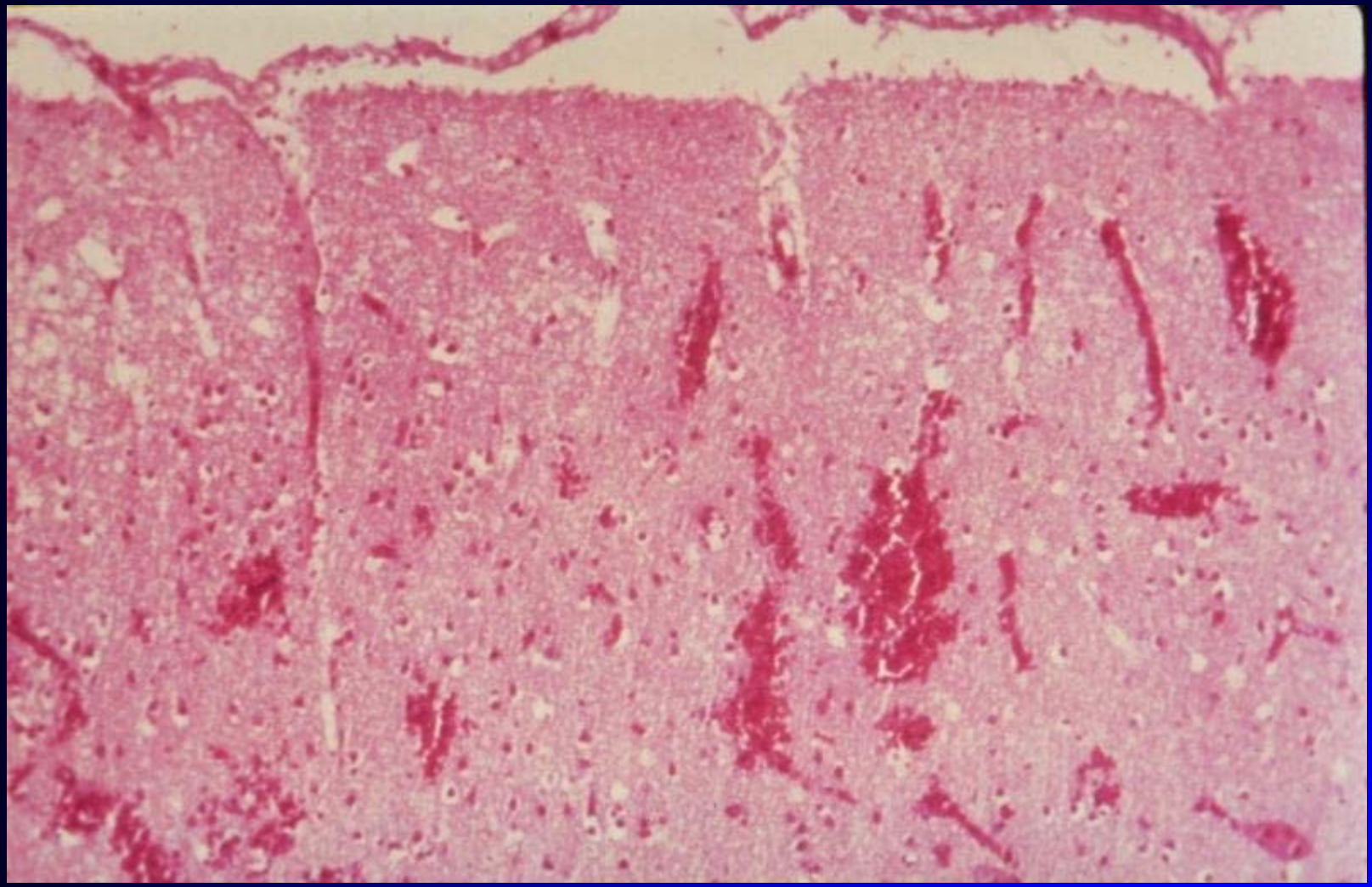
- Osmolarity
- BBB damage
- Circulatory effects
- Hypotension
- Vaso-vagal attack

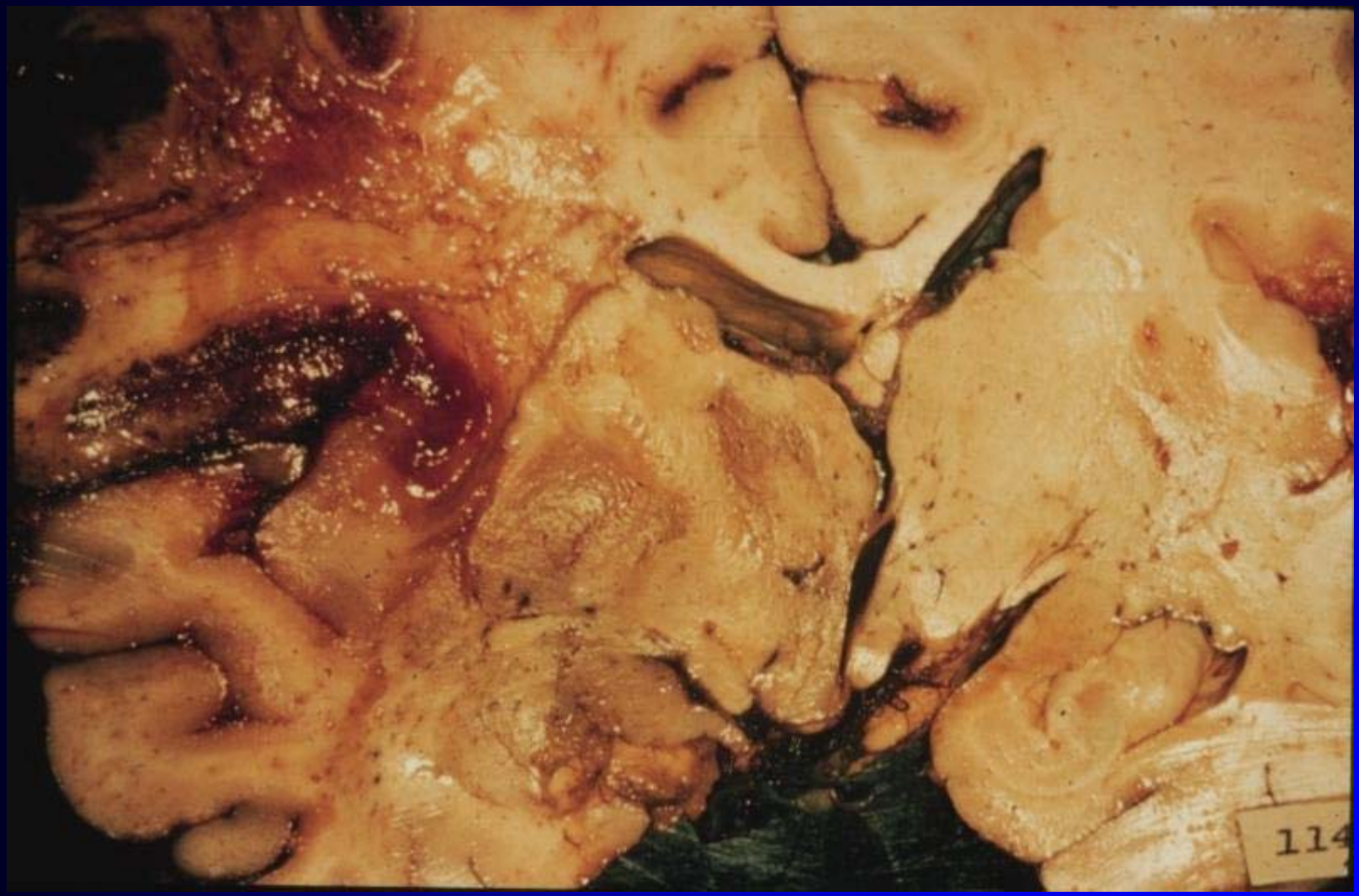
HEMORRHAGIC INFARCTION

CEREBROVASCULAR DISEASE:
Hemorrhagic Infarction

- ARTERIAL ETIOLOGY
  - -Embolic Occlusion (MCA)
  - -Herniation (PCA, AChA)
- VENOUS ETIOLOGY
  - -Sinus Thrombosis (SSST)
    - infarcts in strips, parallel falx
- CONVERSION in ISCHEMIC INFARCT
  - -Large Infarcts
  - -Anticoagulation (incl. ASA)


Zone of Infarction
embolus
pressure restored
Hemorrhagic Infarction
Anemic Infarction
embolus breaks up,
goes into 1 branch

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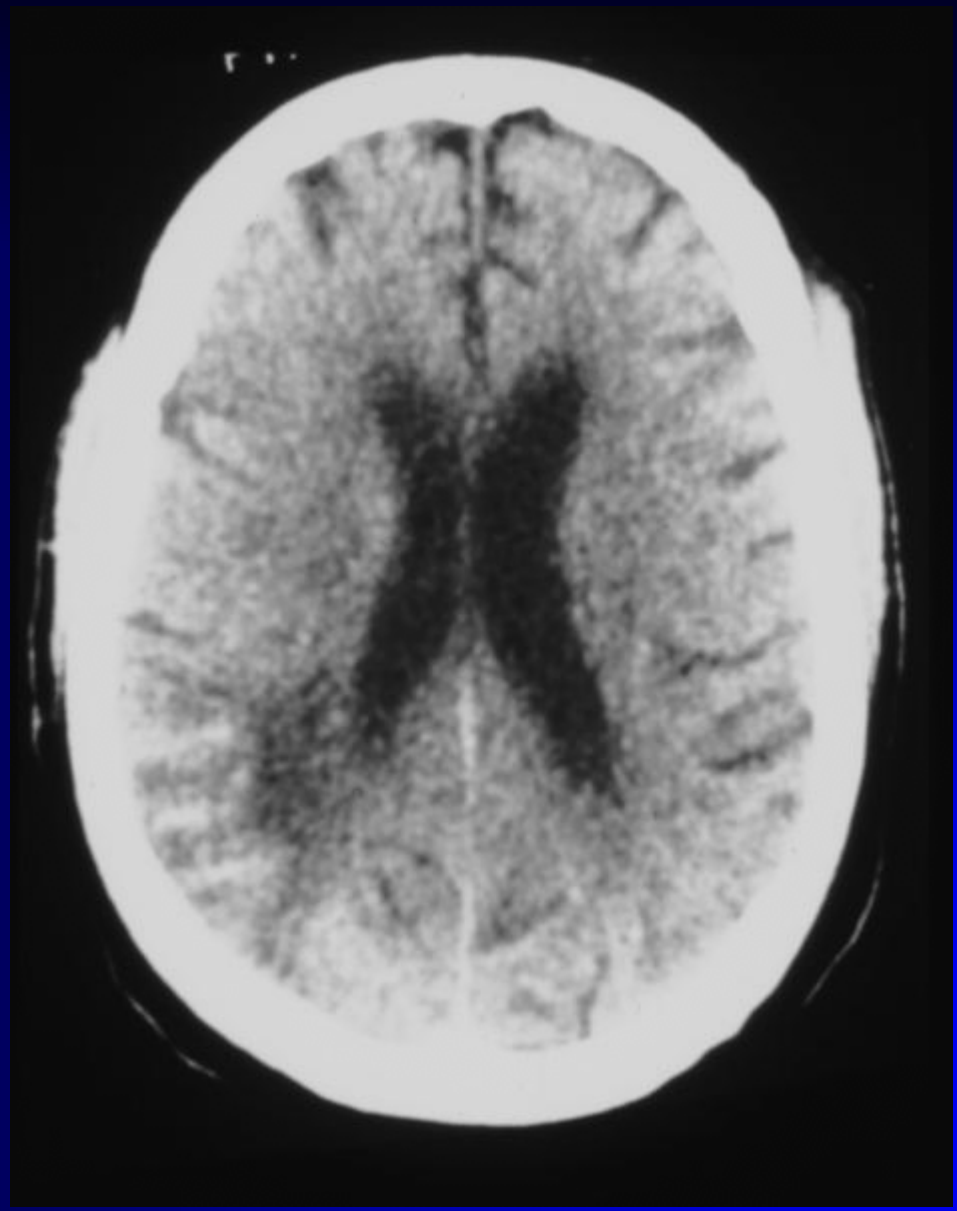
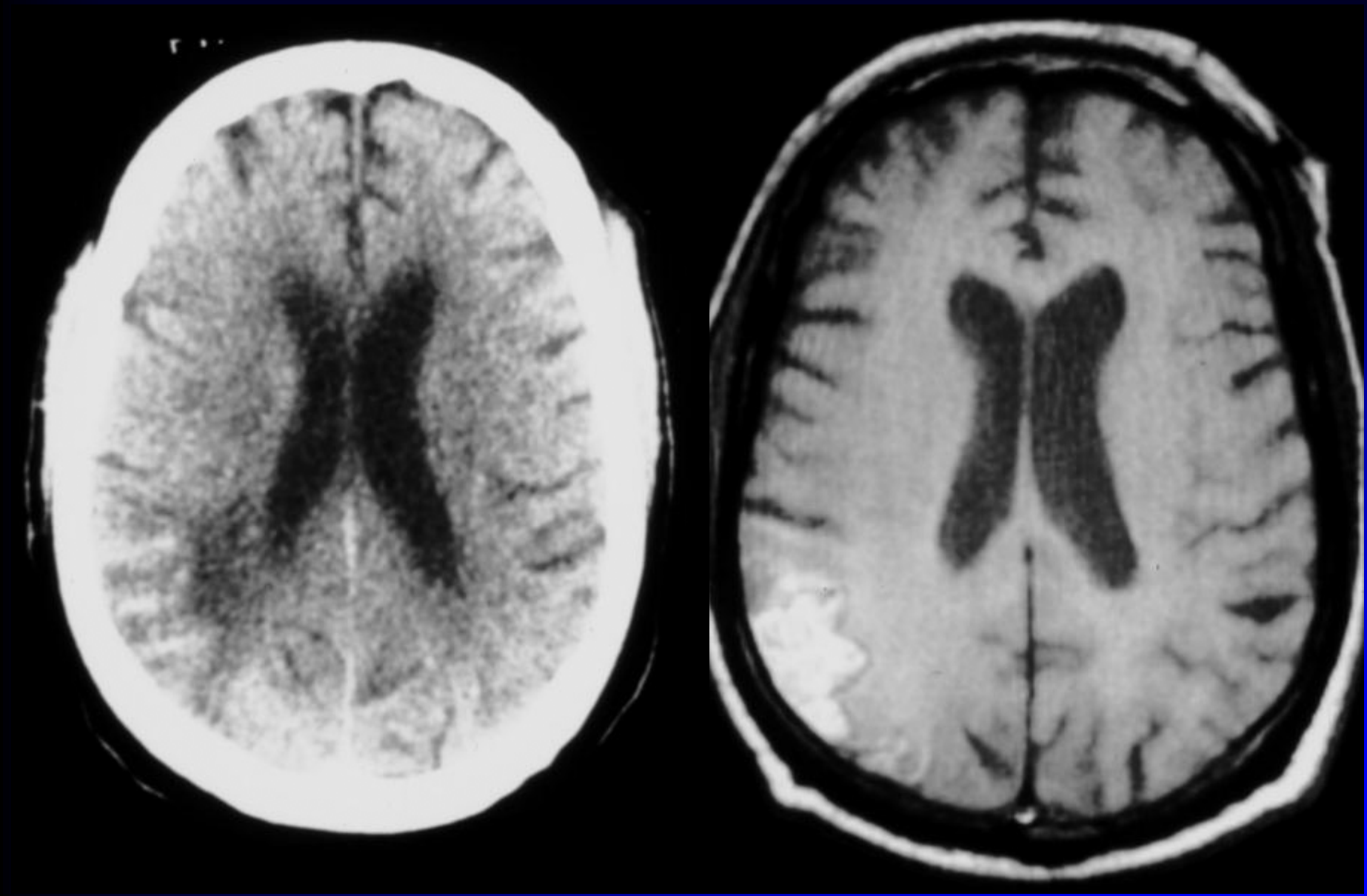
A-41-80

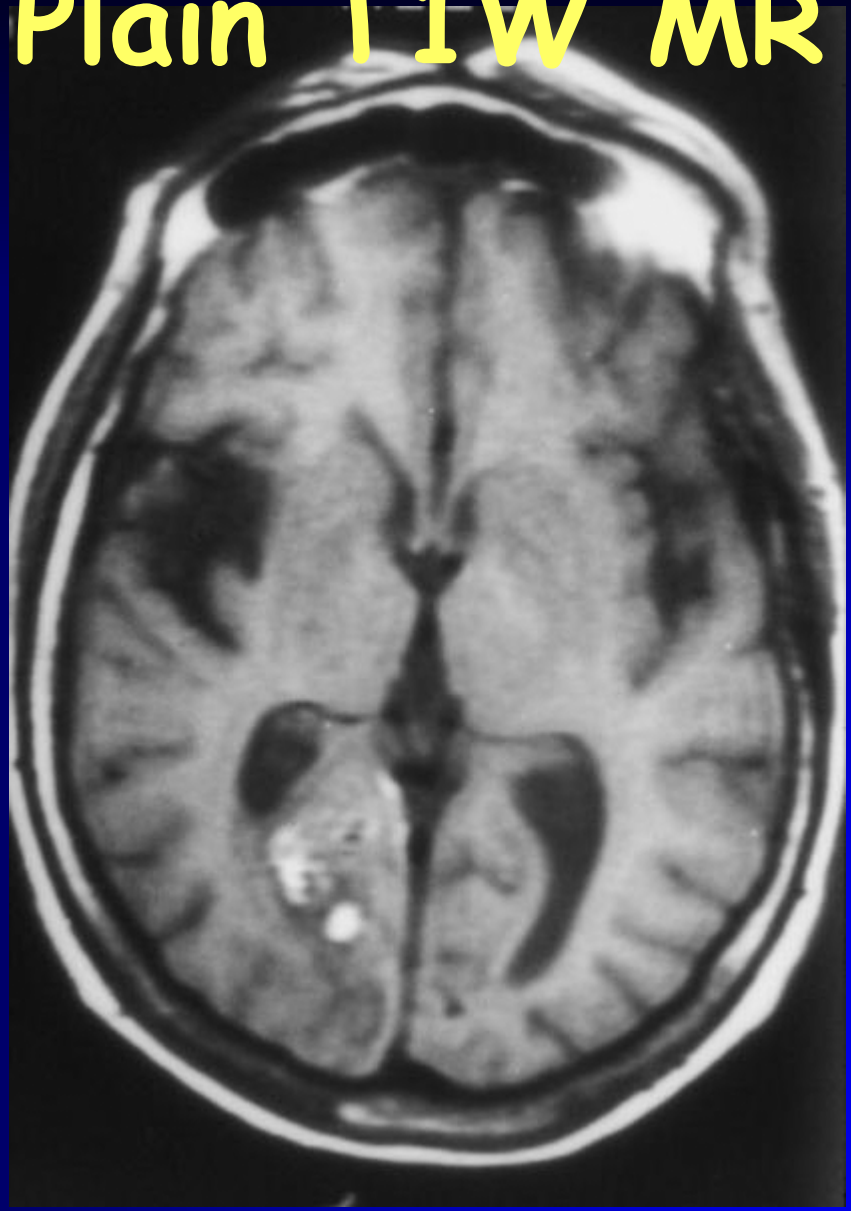
Hemorrhagic Infarction

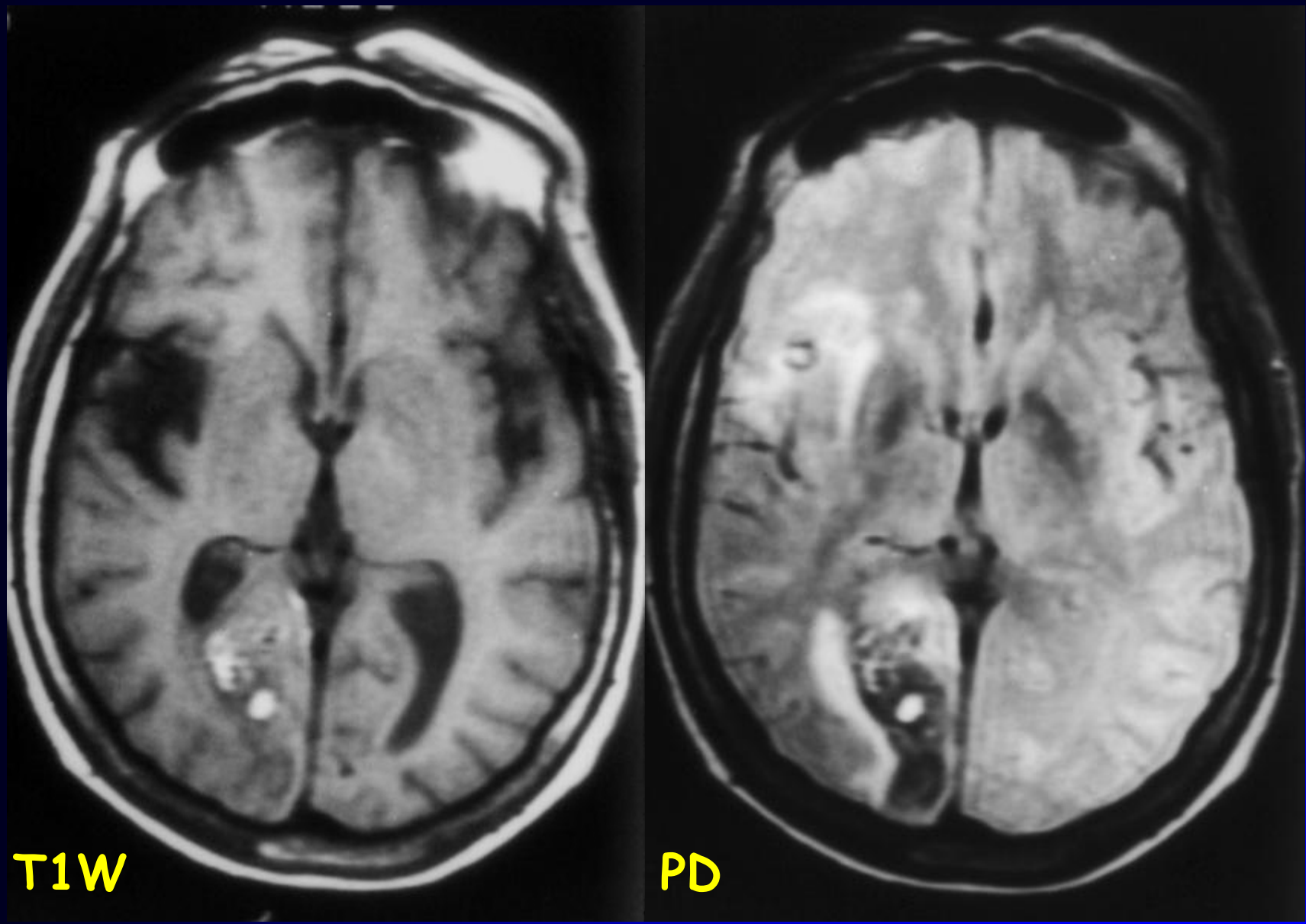
- IMAGING HEMORRHAGIC INFARCTION
  -can be smaller in embolic (MCA)
  -can be larger in herniation (PCA, AChA)
- Petechial Hemorrhage into Cortex
  -ISO- to HYPER- dense on NCT
  -BRIGHT on T1W MRI
- Enhance Early and Intensely
  -NM scans positive EARLY

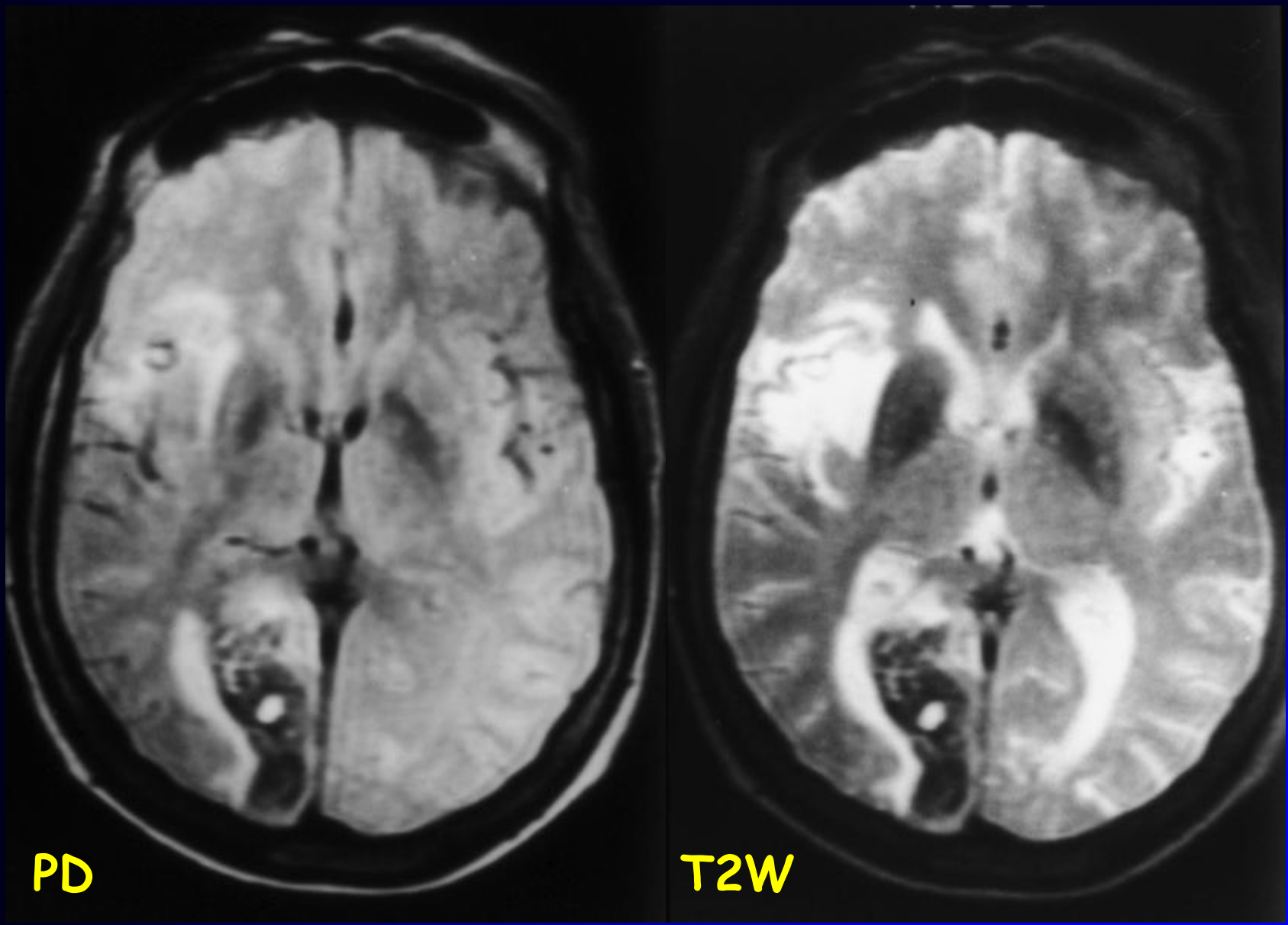




Plain T1W MR




T1W
PD

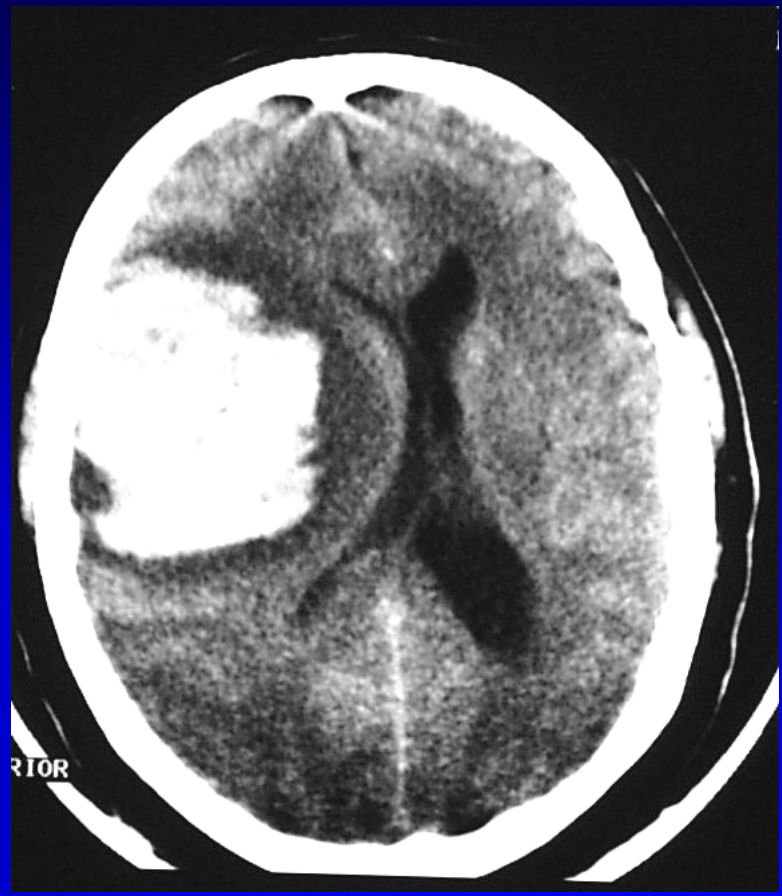

PD
T2W

MAGNETIC RESONANCE
Imaging of Hemorrhage

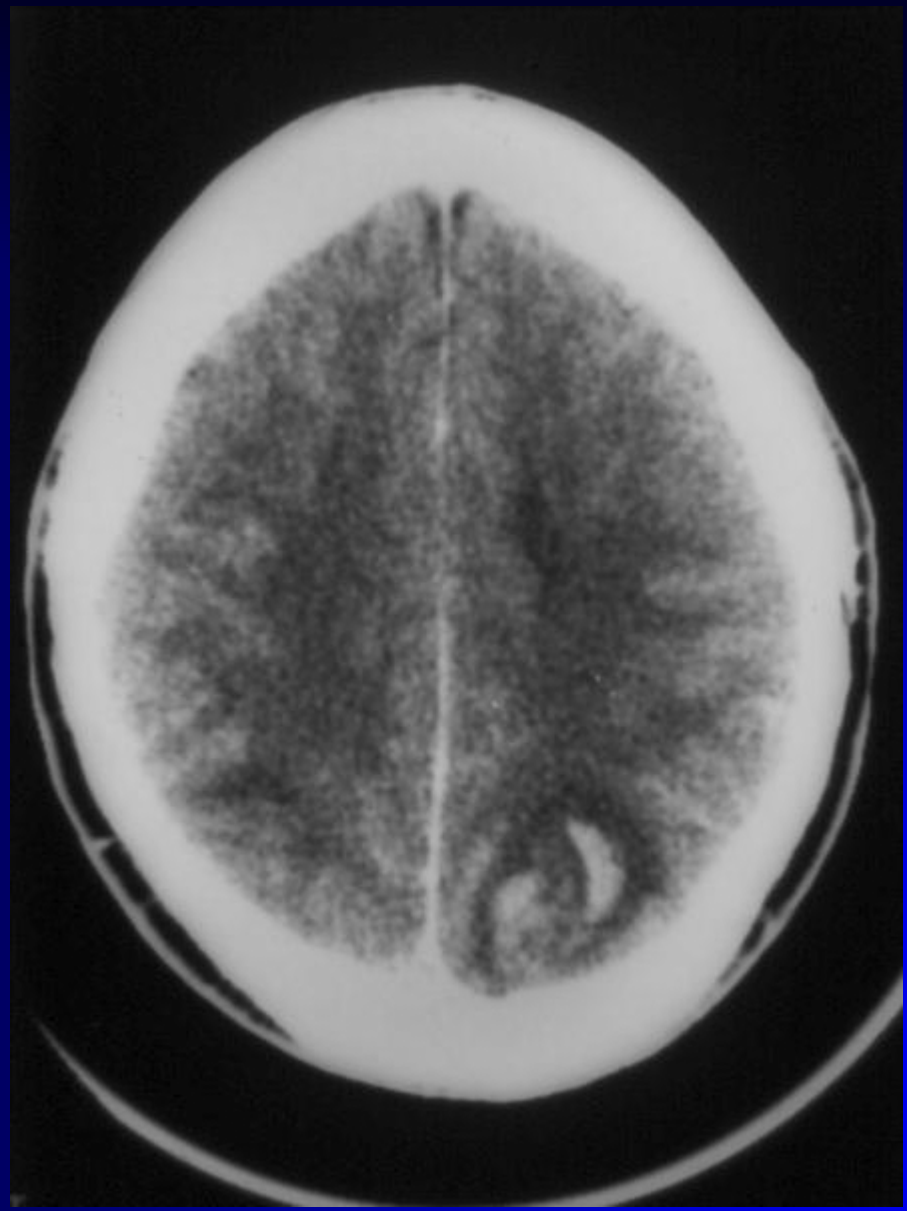
Blood Product	T1W	T2W
Oxyhemoglobin	Iso	Iso
De-oxy Hgb	Iso (1-3)	Low (2)
Met-Hgb (in cells)	Hi (3-14)	Low
		ISO
Met-Hgb (in soln.)	Hi	Hi (3-14)
Hemichromes		
Hemosiderin	Low	Lower

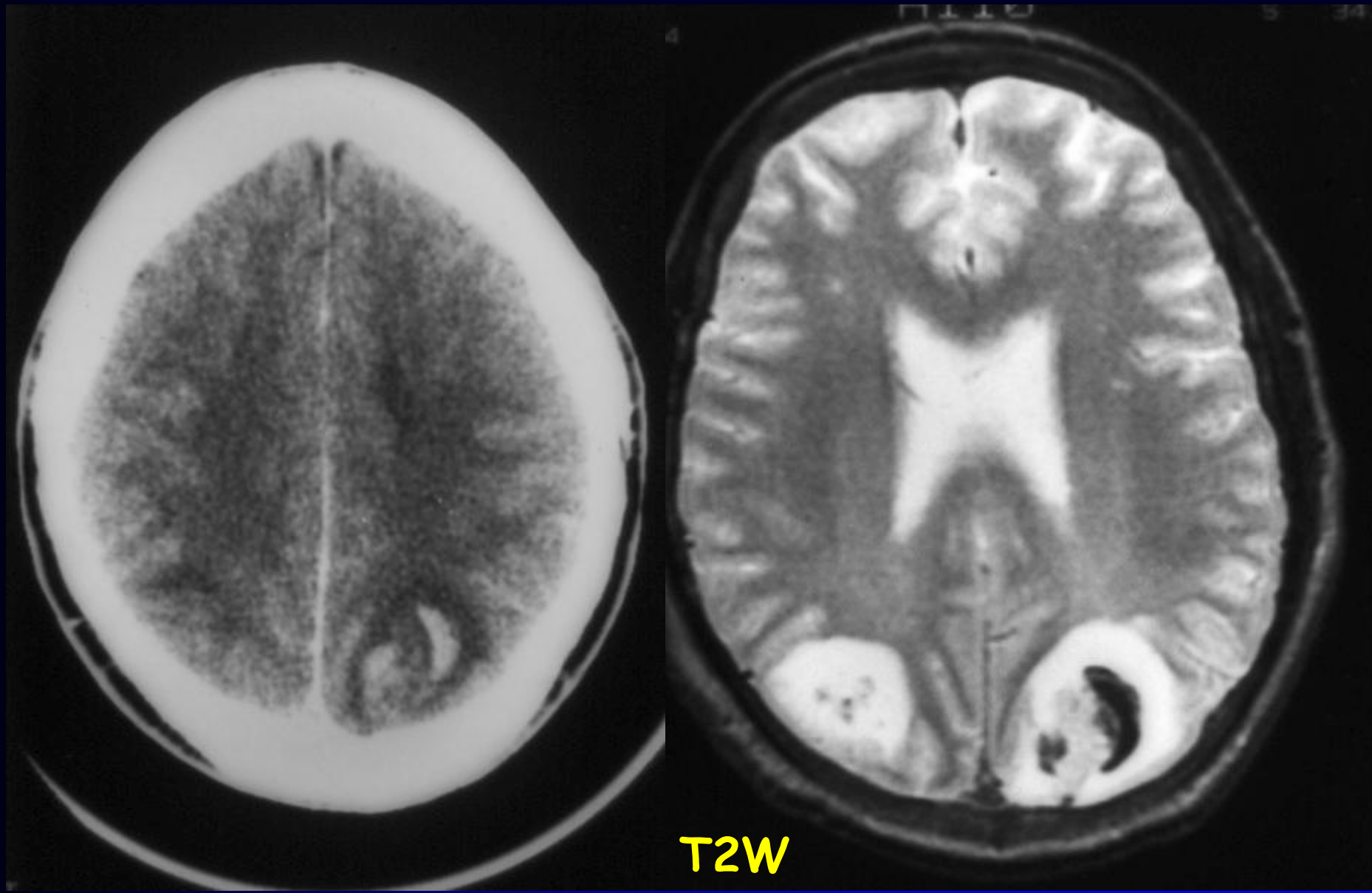
Hemorrhagic transformation

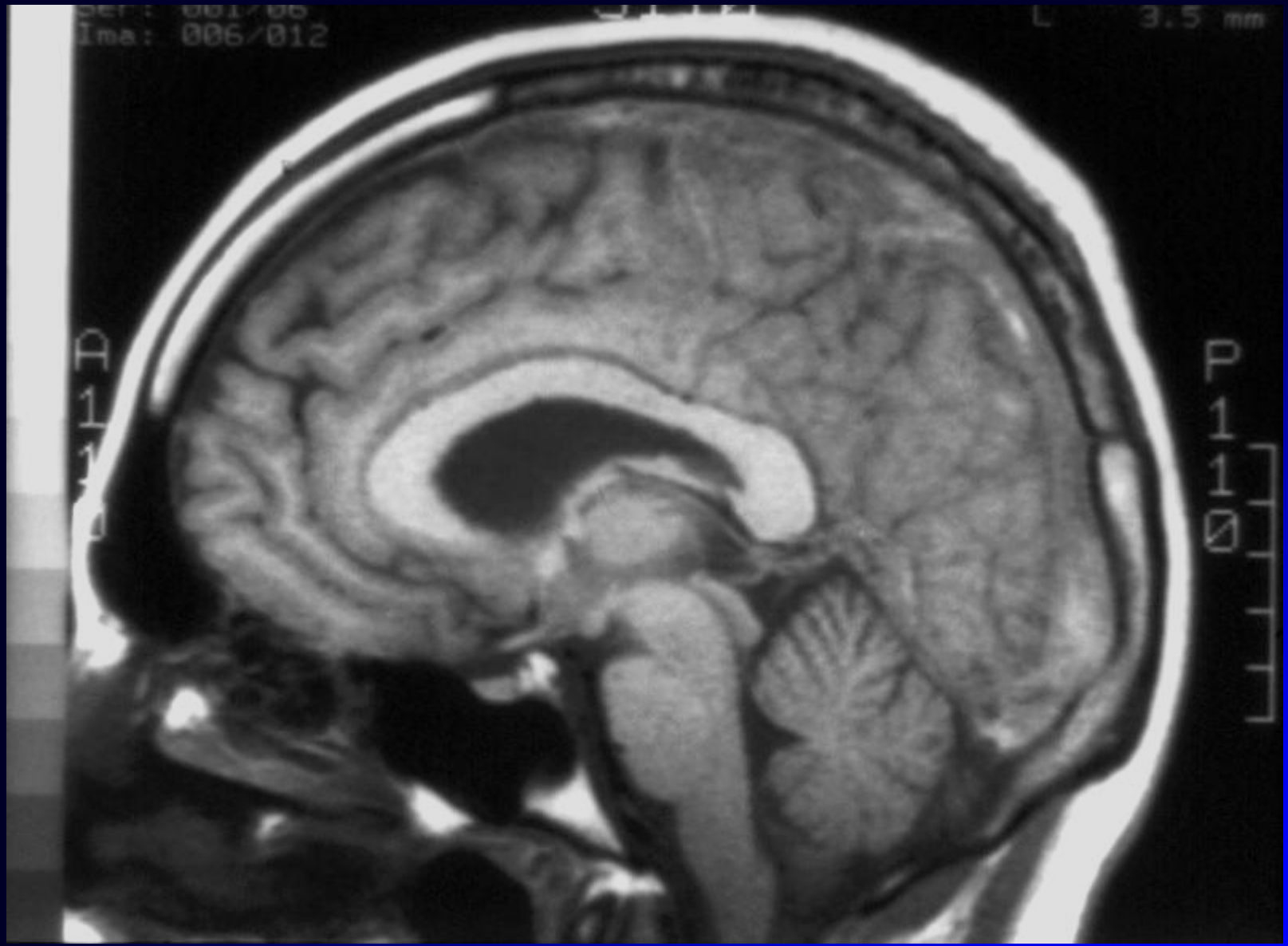
Initial



24 hours later




T2W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Superior Sagittal Sinus Thrombosis

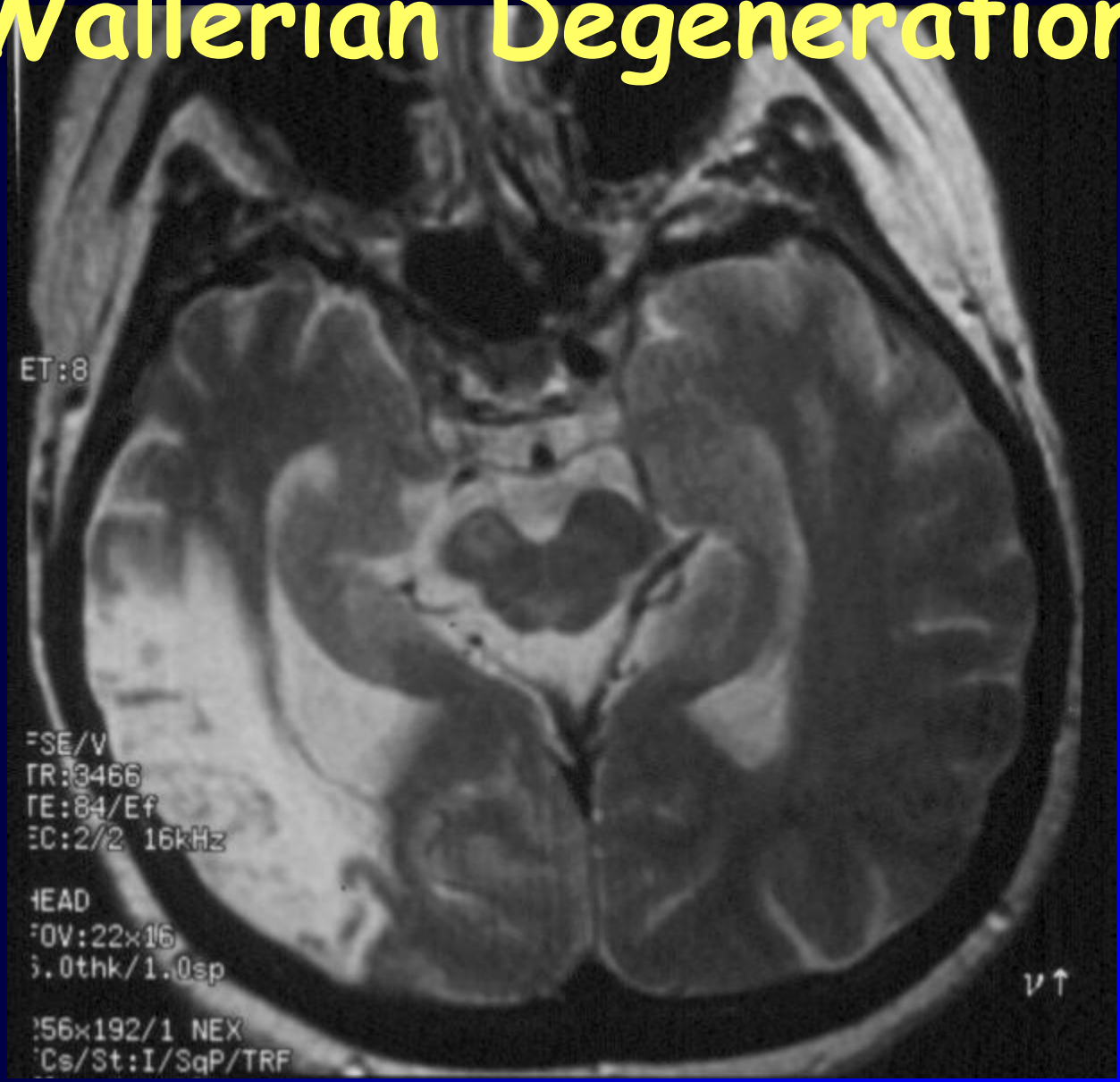
Sup. Sag. Sinus Thrombosis

- Dehydration
- Paraneoplastic Syndromes w/hypercoag
- Spinal Anesthesia
- Post-partum

Old Infarct


T:8
SE/V
R:3466
E:84/Ef
C:2/2 16kHz
EAD
OV:22x16
.0thk/1.0sp
56x192/1 NEX

Wallerian Degeneration



OTHER STROKES,
IN OTHER FOLKS

CEREBROVASCULAR DISEASE: HYPERTENSION

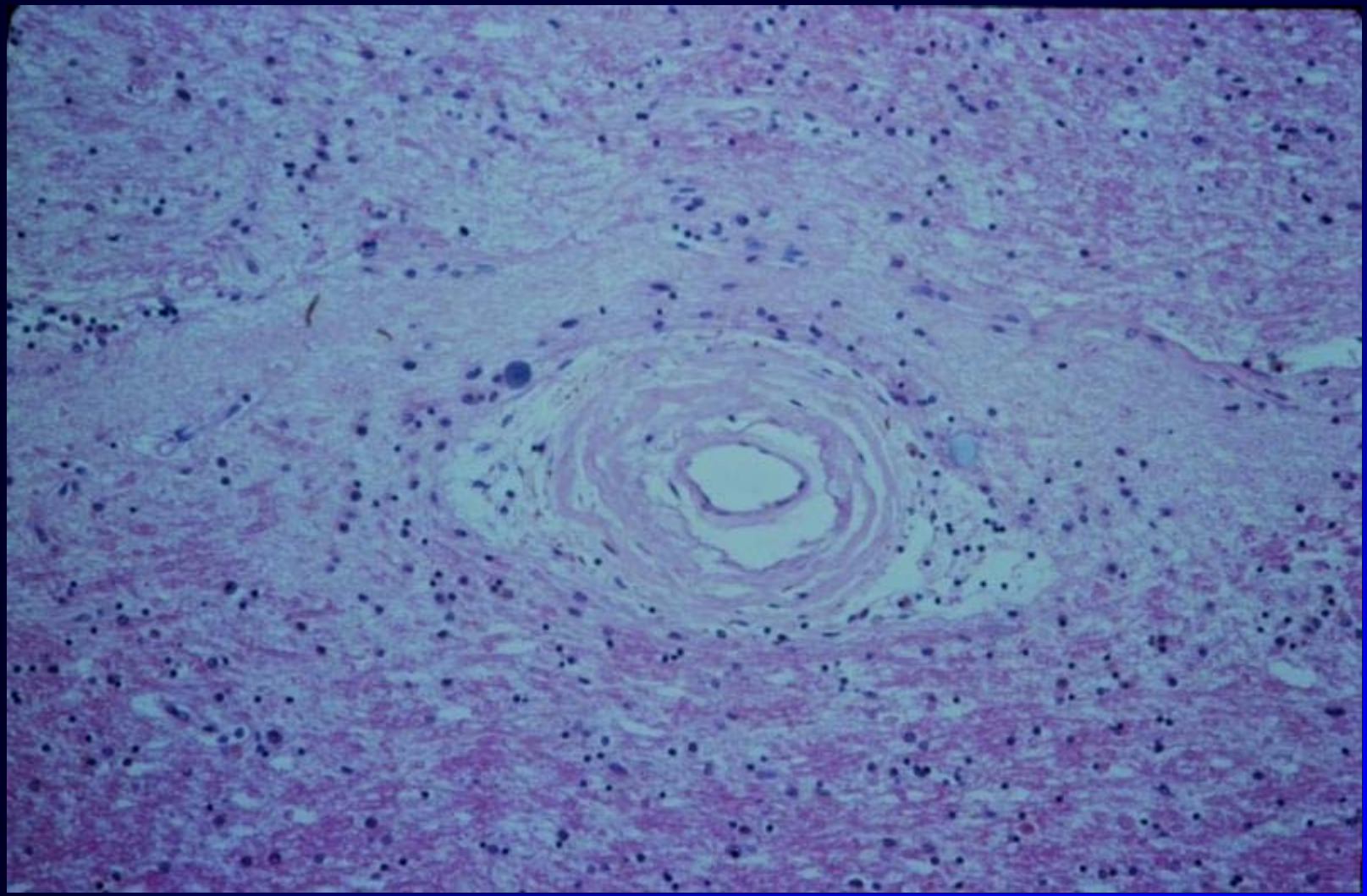
- *Hypertensive Hematoma (Hemorrhage)*
- *Lacunar Infarction*

CEREBROVASCULAR DISEASE
Arteriolar Sclerosis

- Spontaneous Arteriolar Occlusion
  - -lacunar infarcts
- Spontaneous Rupture
  - -"hypertensive hemorrhage"
- Ectatic Elongation
  - -Etat Crible

ARTERIOLOSCLEROSIS

- Hyaline arteriolosclerosis
- Hyperplastic arteriolosclerosis
- "Lipohyalinosis"



ARTERIOLOSCLEROSIS

- Hyaline arteriolosclerosis
- Hyperplastic arteriolosclerosis
- "Lipohyalinosis"

HYPERTENSIVE CVD

- Arteriolar occlusion - ischemia (Lacunae, Binswanger)
- Arteriolar rupture - hemorrhage (Hematoma-BG, Thal., etc.)
- Etat Crible (Dilated V-R) (status cribrosus/cribalis)

CEREBROVASCULAR DISEASE
Arteriolar Sclerosis

- LACUNAE (Lacunar Infarcts)
  - Multiple
  - Small (1-15 mm.)
  - Deep (Putamen, Thalamus, Pons)
  - Pure Motor Hemiplegia
  - Arteriolosclerosis (HT)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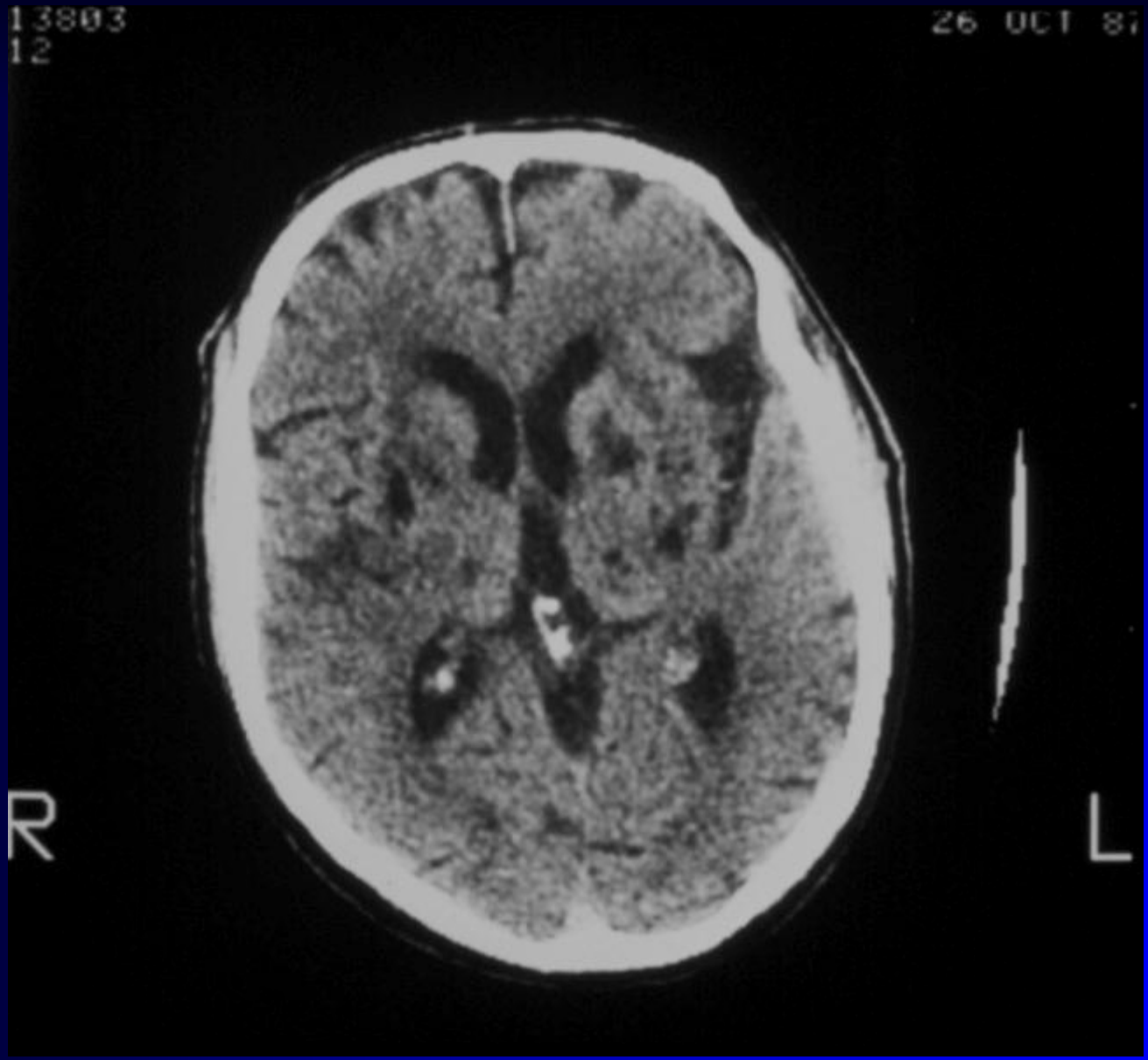
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INTRA-CEREBRAL HEMORRHAGE

- Dense and Homogeneous
- Round/oval shape
- Basal ganglia/deep white
- Proportional mass effect
- Extension into ventricle

HT HEMORRHAGE

- Putamen 55%
- Cerebrum 15%
- Thalamus 10%
- Pons 10%
- Cerebellum 10%

HT BLEED

- Ateriolar Sclerosis
- Penetrating Vessels
- Deep (Nuclear) Areas

CHARCOT-BOUCHARD MICRO-ANEURYSMS

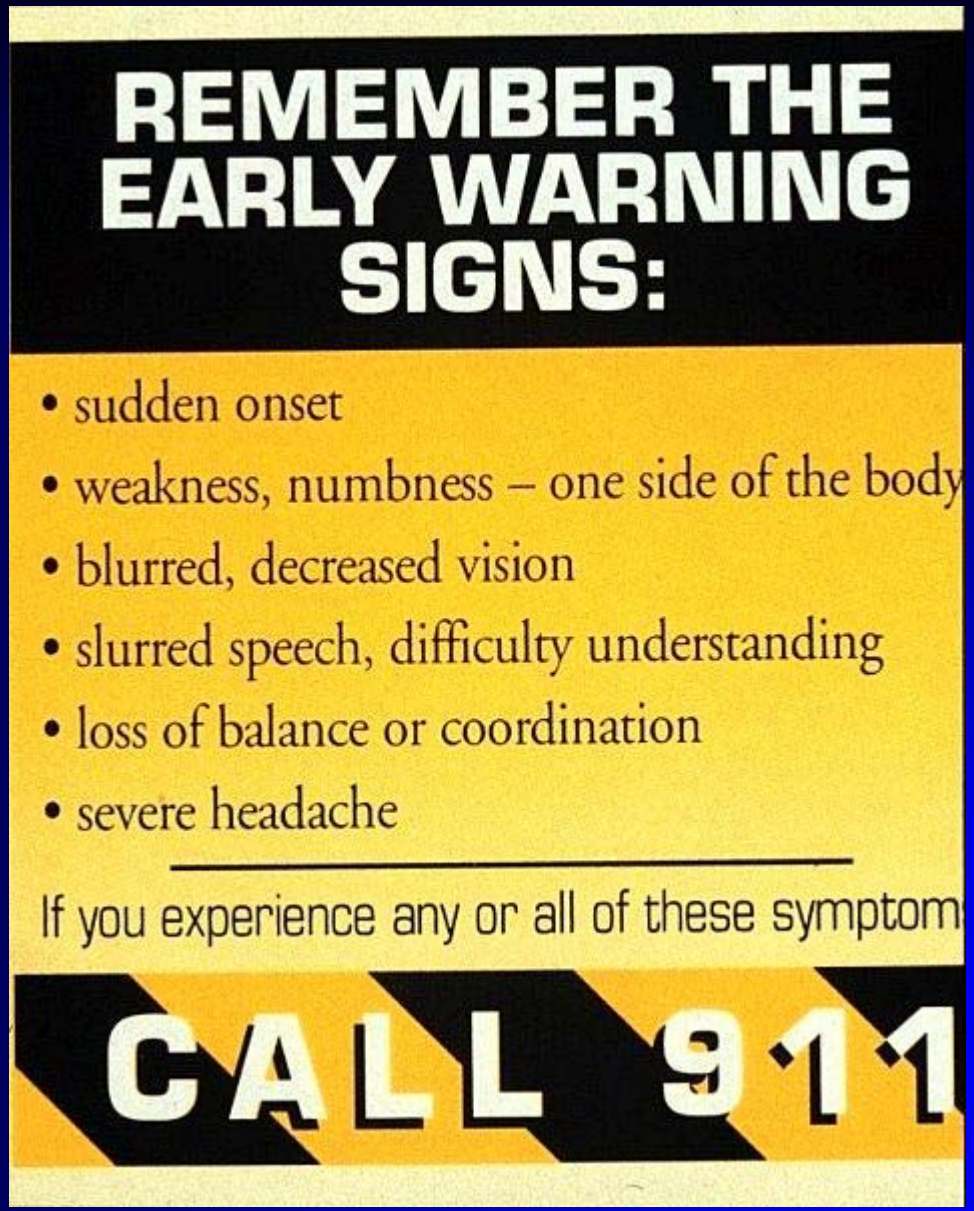

ALRIGHT, LET'S GET TO THE BOTTOM LINE

CEREBROVASCULAR DISEASE

Summary

- CT may be 'normal' (early)
- Mass Effect minimal/mild
  - Peak in 3-5 days, Edema
- Contrast may not be needed
- Difficult to R/O
  - Hemorraghic w/o MR
  - Easier to Rule in
- Dx Embolic vs. Thrombotic
- R/O other pathology


Brain Attack!


REMEMBER THE EARLY WARNING SIGNS:
• sudden onset
• weakness, numbness – one side of the body
• blurred, decreased vision
• slurred speech, difficulty understanding
• loss of balance or coordination
• severe headache
If you experience any or all of these symptom
CALL 911

CVA Quiz

CVA Quiz - 1

- The most common cause of SAH is:
  - A) Child abuse
  - B) Head Trauma
  - C) Ruptured AVM
  - D) Ruptured Aneurysm
  - E) A and D are the Most Common

CVA Quiz - 2

- Hypertensive Vascular Disease:
  - A) Causes Lacunar Infarcts
  - B) Causes Deep Hematomas
  - C) Affects Small Vessels
  - D) Causes AV Shunting
  - E) A, B, and C are ALL True

CVA Quiz - 3

- Which is the most common type of "CVA"?
  - A) Ruptured Aneurysm
  - B) Embolic Infarction
  - C) Thrombotic Infarction
  - D) Hypertensive Hemorrhage
  - E) Carotid Dissection

CVA Quiz - 4

- Cerebral Infarction causes:
  - A) Cytotoxic edema
  - B) Vasogenic edema
  - C) Hydrostatic edema
  - D) A and C are Both True
  - E) A and B are Both True

CVA Quiz - 5

- The incidence of CVA in the USA is recorded accurately by Autopsy?
  - A) TRUE
  - B) FALSE

End of MS-4 Lecture

OTHER STROKES,
IN OTHER FOLKS

Hemorrhagic Infarction

- IMAGING HEMORRHAGIC INFARCTION
  -can be smaller in embolic (MCA)
  -can be larger in herniation (PCA, AChA)
- Petechial Hemorrhage into Cortex
  -ISO- to HYPER- dense on NCT
  -BRIGHT on T1W MRI
- Enhance Early and Intensely
  -NM scans positive EARLY

"WATERSHED INFARCTS"

- WATERSHED INFARCTS
  Hypotension, Shock, CPR
  < 50mm Hg for > 10 min
  Depths of Sulci
  Boundary zones

SELECTIVE VULNERABILITY

- Watershed/Boundary Zones
  Hippocampus - Sommer's Sector
  Globus Pallidus
  Cerebellum
  Cerebral III, IV, V

AMYLOID/CONGOPHILIC ANGIOPATHY

- AMYLOID ANGIOPATHY
  - Age > 65
  - Lobar Hemorrhage (Not BG)
  - Cortex, Subcortical WM
  - MULTIPLE Hemorrhages
  - Simultaneous
  - Metachronous

Aneurysm and Rupture

- Clinical Hx:
  - Worst Headache of My Life
  - Nuchal Rigidity (meningismus)
  - Photophobia
  - Localizing Signs

Aneurysm and Rupture

- Demographics:
  - Common Cause of Stroke in Adults
  - Peak at 40-60yr
  - Risk Factors: Hypertension

Aneurysm and Rupture

- Aneurysm Incidence:
  - 1-8% of unselected Adult Autopsies
  - Implies most are ASYMPTOMATIC
  - Familial, ADPCKD
- Subarachnoid Hemorrhage
  - LP more sensitive than CT
  - Trauma is most common cause
  - SAH on CT usually Aneurysm/AVM

BRAIN

- 1/50 of body weight
- 1/6 of cardiac output
- 1/5 of Oxygen

NORMAL CEREBRAL BLOOD FLOW:

- Gray matter 4x white matter
  Cortex = 1.69 ml/gm/min
  Callosum - 0.40 ml/gm/min

Ischemic Infarction

- Non-Hemorrhagic
  Arterial Thrombosis,
  Hypotension
  Larger Vessel, Large Infarct
  Atherosclerosis

- *Hyperdense/Hyperintense MCA*
  Vessel enhancement w/Gd
  Infarct - Low Density
  NO BLOOD - Anemic Cortex
  EDEMA
  Cytotoxic (Gray and White)
  Vasogenic (White only)
  ENCEPHALOMALACIA
  necrosis
  phagocytosis (encephaloclysis)

Ischemic Infarction

- Increased Water
  - Cyotoxic edema
- Prolonged T1/T2, Decreased specific gravity of tissue
- Decreased Attenuation on NCT
- +/- Sl. darker on T1W MRI
- Sl. brighter on PD
- Bright on T2W MRI
- Bright on DWI, Dark on ADC Map

WHAT ABOUT CONTRAST?

- If an infarct is caused by a loss of blood flow, then how do water and contrast get there?

- *Re-perfusion*
  -collaterals (early)
  -recanalization (late)
  -healing (late)

CONTRAST TOXICITY

- Osmolarity
- BBB damage
- Circulatory effects
- Hypotension
- Vaso-vagal attack

- *"Ischemic Enhancement"*
  Misnomer?
  Contrast delivered by flow
  Cortical Gray - "Gyriform" pattern
  Etiology:
  increased flow
  Abnl. BBB outside infarct
  Abnl. BBB during healing of infarct
  "Luxury Perfusion"
  Angiographic term
  increased perfusion/vascularity
  implies re-perfusion

Ischemic Infarction

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- *"Ischemic Enhancement"*
  (Reperfusion in min. to hrs.)
  "LUXURY PERFUSION"
  BBBB (> 4 - 6 hrs. of ischemia)
  original capillaries affected
  New Capillaries (7-10 days) grow into infarct for healing w/o BBB

- *"LUXURY PERFUSION"*
  Angiographic observation
  FOCAL HYPEREMIA (relative or absolute)
  CORTICAL BLUSH and EARLY VEINS
  shunting
  LOSS OF AUTOREGULATION
  (caused by inc. pCO2, decr. pH/pO2)
  TRANSIENT
  minutes to hours, ends in 3-5 days

PATTERNS OF EDEMA

AJNR 3:251-255, 1982

- VASOGENIC SPREAD
  (neovascular edema)
  4/339 (1.2%) INFARCTS

- GRAY MATTER EDEMA
  (Cytotoxic edema)
  2/155 (1.3%) TUMORS

HEPARIN CONTRAINDICATIONS

- neoplasm
- AVM, aneurysm, SAH
- grossly hemorrhagic mass
- LARGE infarcts, any cause
- Hemorrhagic Infarction?
- Embolic Etiology?

EXCEDRIN HEADACHE #27:
(4:00 AM)
"CAN I ANTICOAGULATE?"

HEPARIN Indications:

- Stroke-In-Evolution
- progression of vascular territory
- Embolic Stroke

HEPARIN CONTRAINDICATIONS:

- Neoplasm
- AVM, aneurysm
- Grossly hemorrhagic
- Large infarcts

Hemorrhagic infarct? (embolic infarction)

EMBOLIZATION AND HEPARIN

- Recurrent emboli (<2 wks)
- 12% of untreated (2-22%)
- 5% of anticoagulated (0-5%)

Hemorrhagic complications (with Anticoagulation)

- 5% (with deterioration)
- 14-20% (without deterioration)
- Occur in 20% of large infarcts

Quiz Question

Quiz Question

Quiz Question

Quiz Question

Quiz Question