

# Cerebrovascular diseases

Angelo Antonini

# Stroke Objectives

- Review etiology of strokes
- Identify likely location/type of stroke based of physical exam
- Acute management of ischemic stroke
- Acute management of hemorrhagic stroke

# Stroke Fast Facts

- Affects ~ 800, 000 people per year
- Leading cause of disability, cognitive impairment, and death in the United States
- Accounts for 1.7% of national health expenditures.
- Estimated U.S. cost for 2012 = \$71.5 billion
  - Mostly hospital (esp. LOS) & post stroke costs
  - Appropriate use of IV t-PA ↓s long-term cost
  - Appropriate billing for AIS w/ thrombolysis (↑ hospital reimbursement from \$5k to \$11.5k)

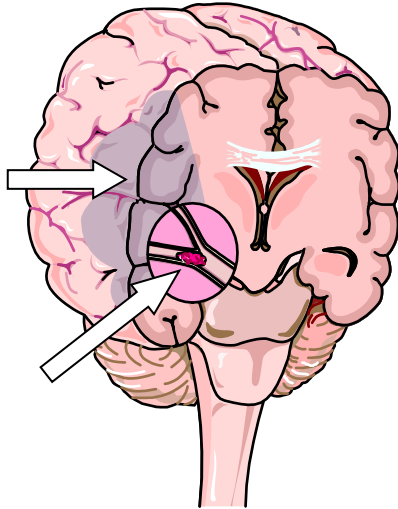
# Where We're Headed

- By 2030 ~ 4% of the US population over the age of 18 is projected to have had a stroke
- Between 2012 and 2030, total direct stroke-related medical costs are expected to increase from \$71.55 billion to \$183.13 billion
- Total annual costs of stroke are projected to increase to \$240.67 billion by 2030, an increase of 129%



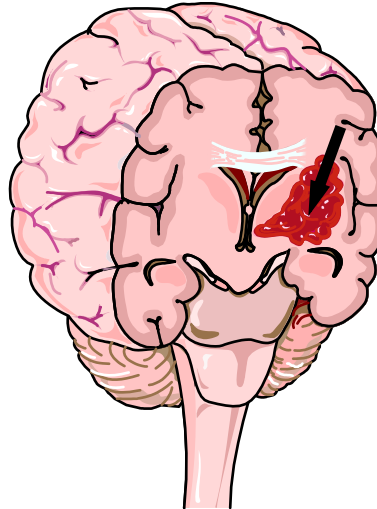
# Three Stroke Types

**Ischemic Stroke**



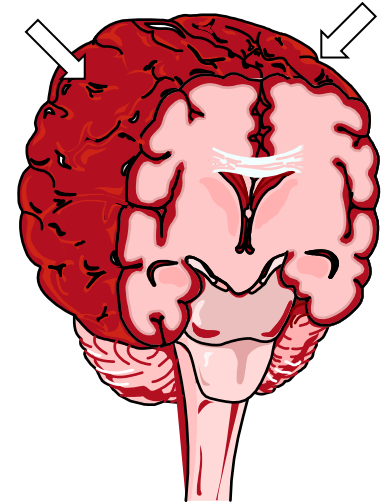
Clot occluding artery  
85%

**Intracerebral Hemorrhage**



Bleeding into brain  
10%

**Subarachnoid Hemorrhage**



Bleeding around brain  
5%

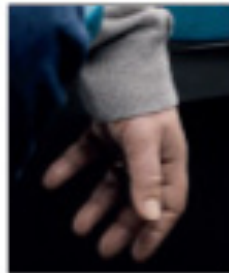
# When Stroke Strikes, Act F.A.S.T.



## FACE

Smile.

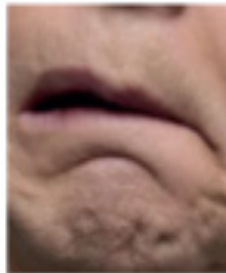
Does one side of the face droop?



## ARMS

Raise both arms.

Does one arm drift downward?



## SPEECH

Repeat a sentence.

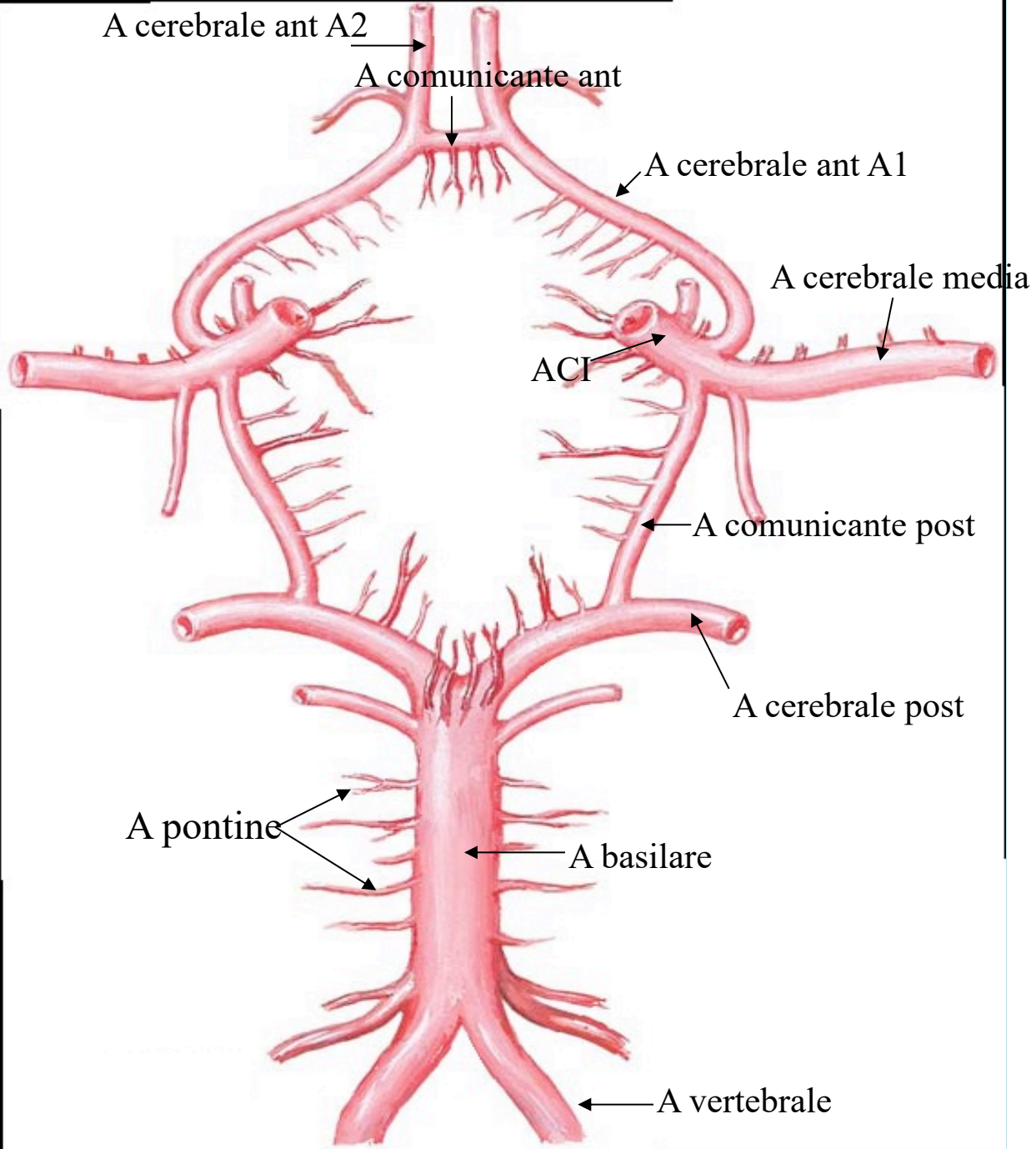
Are they able to speak clearly?  
Can they repeat the sentence?



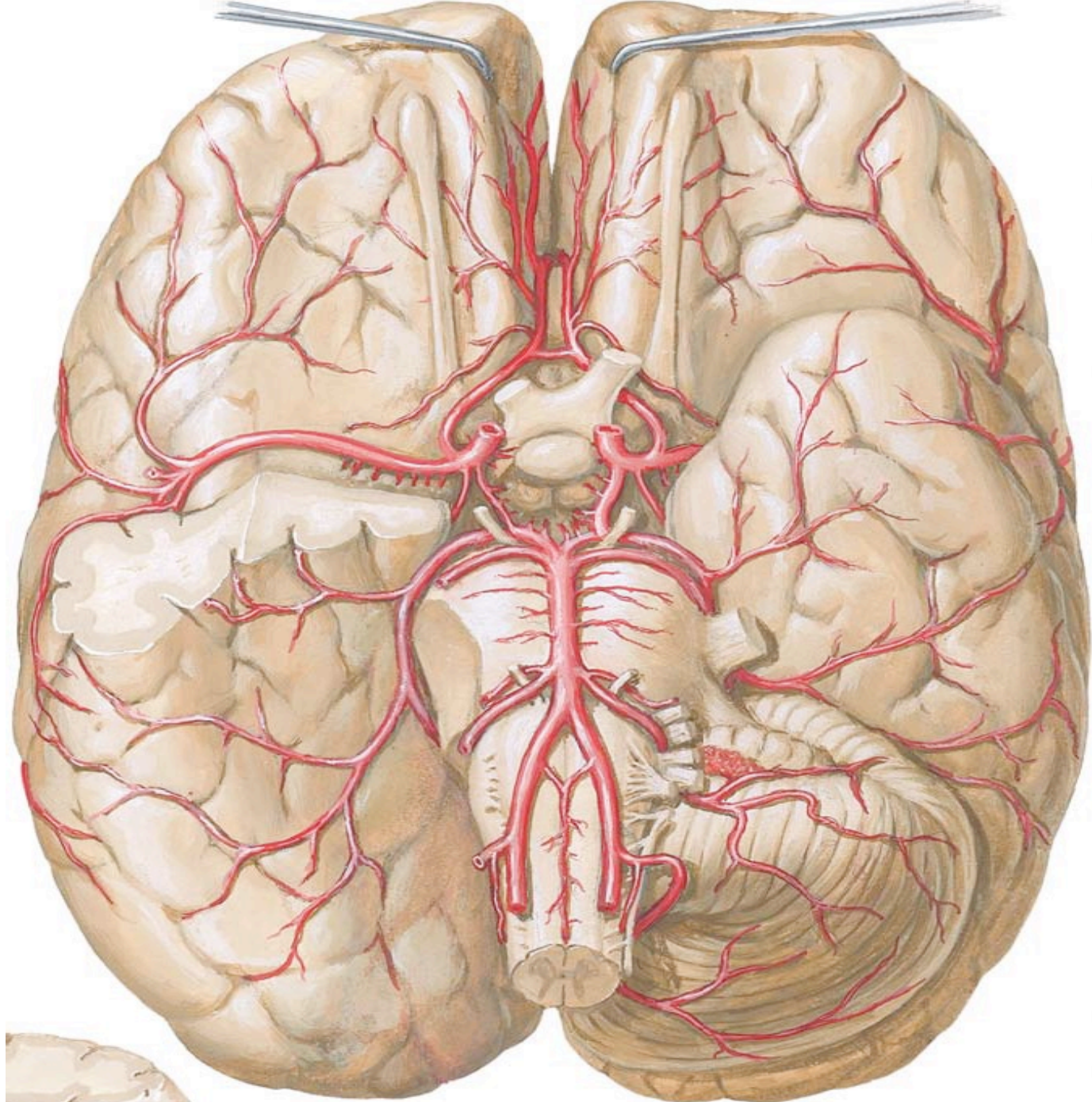
## TIME

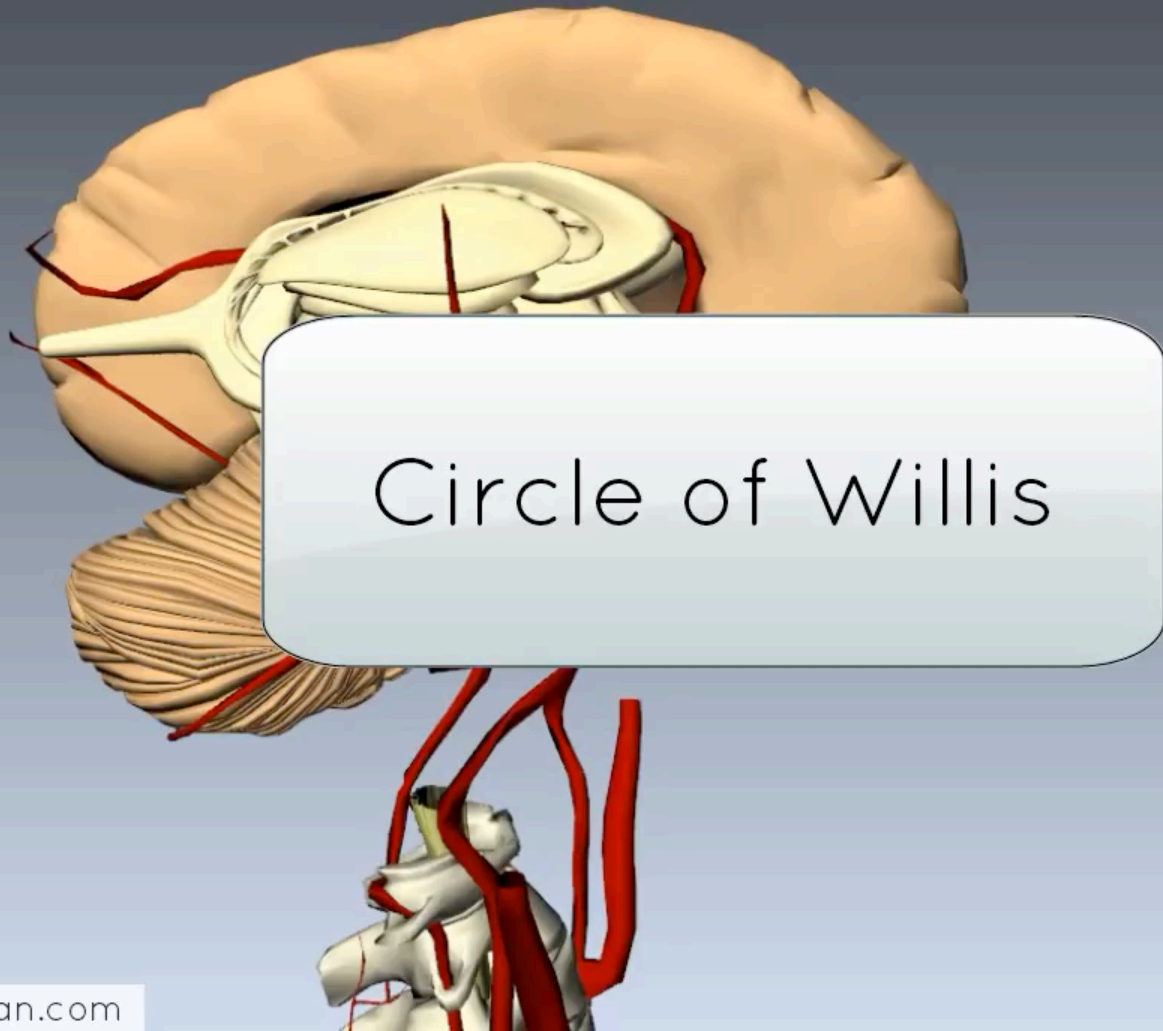
Time is critical.

**Call 911.** Get to the hospital immediately. Brain cells are dying.  
**Every Minute Counts!**





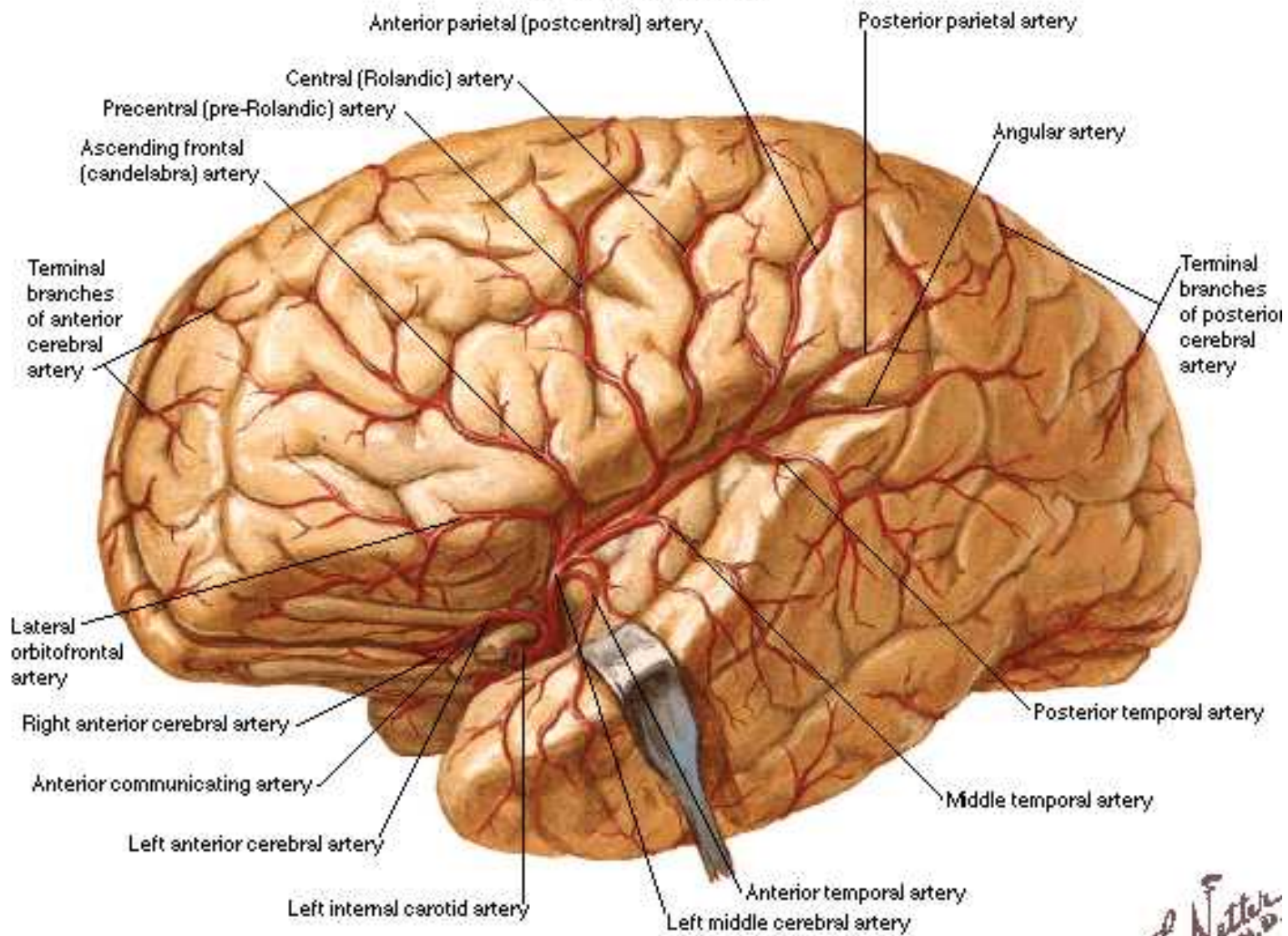




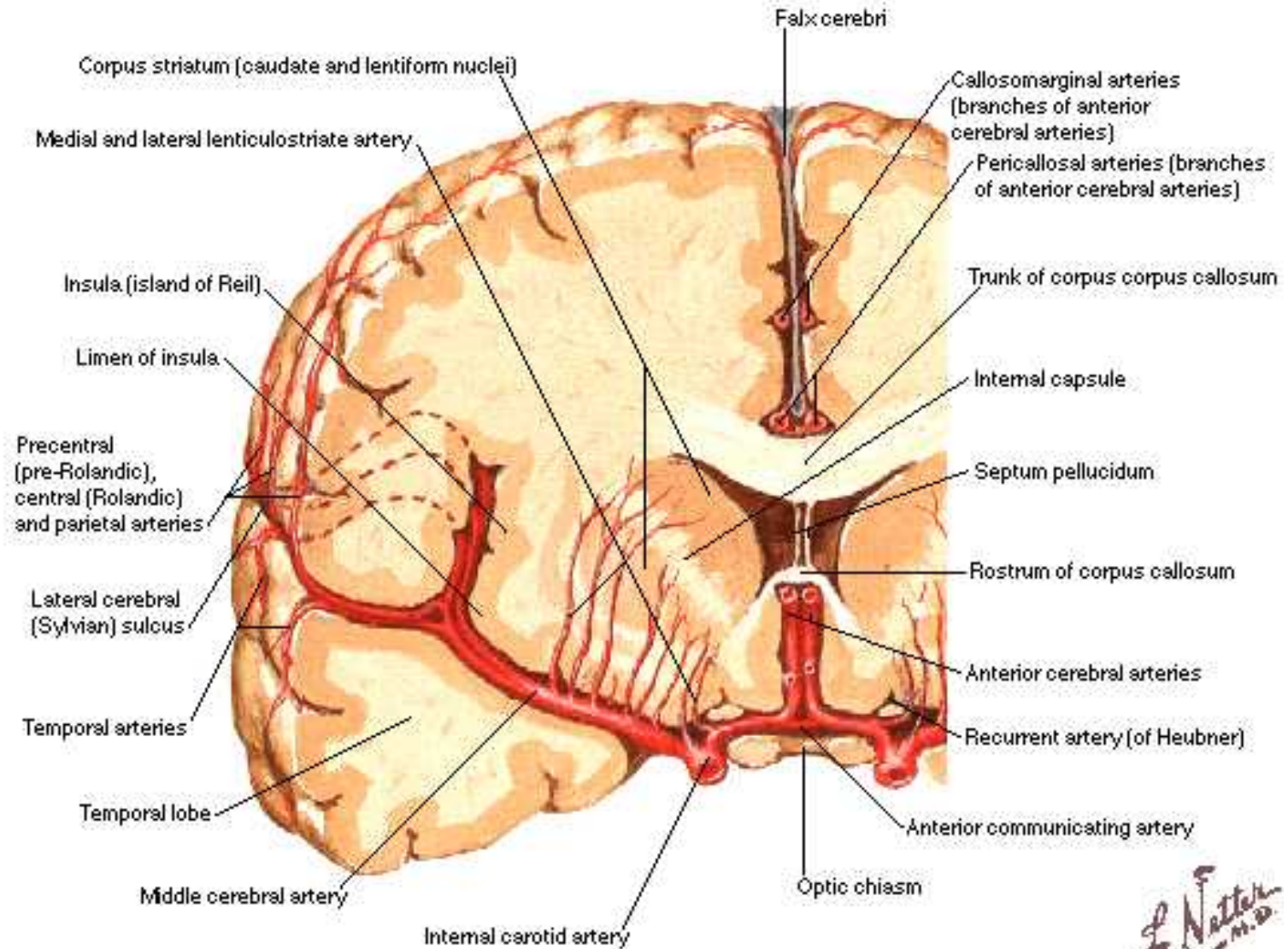
# Circle of Willis



# Lateral View

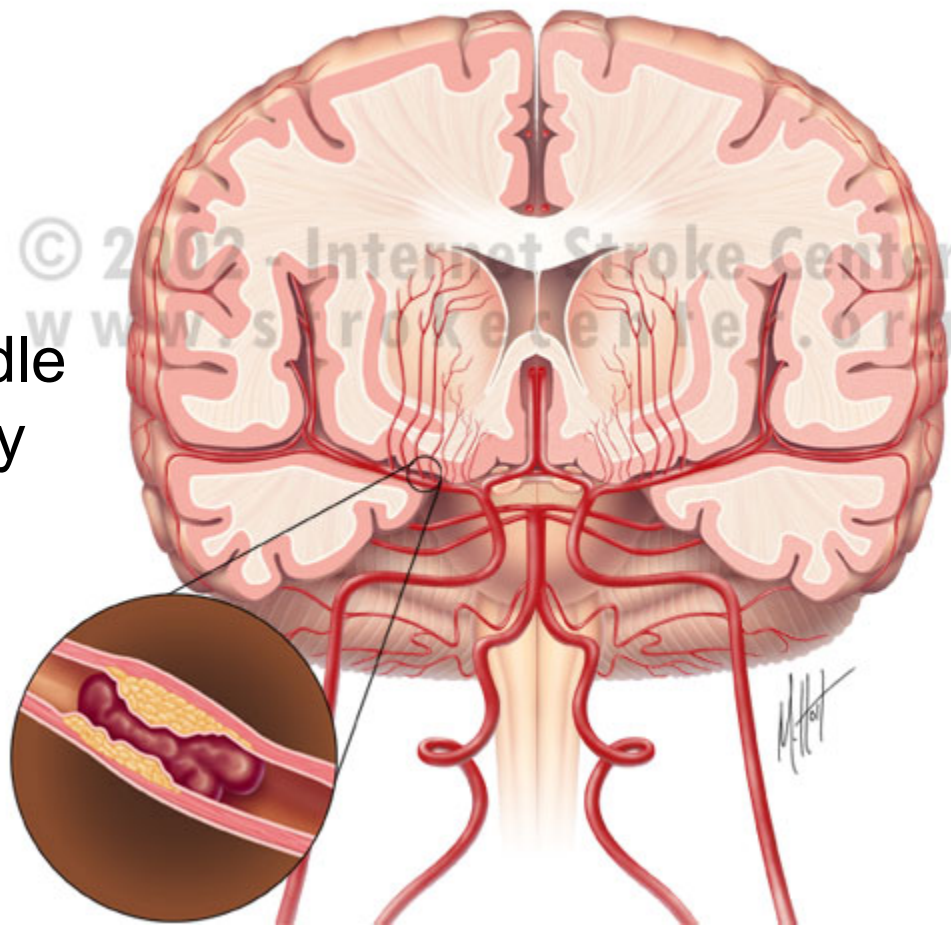


*F. Natter*  
M.D.



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Clot in the middle cerebral artery







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# NIHSS

- NIHSS (National Institute of Health Stroke Scale)
  - Standardized method used by health care professionals to measure the level of impairment caused by a stroke
  - Purpose
    - Main use is as a clinical assessment tool to determine whether the degree of disability is severe enough to warrant the use of tPA
    - Another important use of the NIHSS is in research, where it allows for the objective comparison of efficacy across different stroke treatments and rehabilitation interventions
  - Scores are totaled to determine level of severity
  - Can also serve as a tool to determine if a change in exam has occurred

# Breaking Down the Scale

- 13 item scoring system, 7 minute exam
- Integrates neurologic exam components
- CN (visual), motor, sensory, cerebellar, inattention, language, LOC
- Maximum score is 42, signifying severe stroke
- Minimum score is 0, a normal exam
- Scores greater than 15-20 are more severe

# NIHSS cont.

- NIHSS Interpretation

Stroke Scale	Stroke Severity
0	No Stroke
1-4	Minor Stroke
5-15	Moderate Stroke
15-20	Moderate/Severe Stroke
21-42	Severe Stroke

# NIHSS and Outcome Prediction

- NIHSS below 12-14 will have an 80% good or excellent outcome
- NIHSS above 20-26 will have less than a 20% good or excellent outcome
- Lacunar infarct patients had the best outcomes

*Adams HP Neurology 1999;53:126-131*

Baseline NIH Stroke Scale score strongly predicts outcome after stroke (TOAST)

# Etiology of Ischemic Strokes

## LARGE VESSEL THROMBOTIC:

Virchow's Triad....

- **Blood vessel injury**
  - HTN, Atherosclerosis, Vasculitis
- **Stasis/turbulent blood flow**
  - Atherosclerosis, A. fib., Valve disorders
- **Hypercoagulable state**
  - Increased number of platelets
  - Deficiency of anti-coagulation factors
  - Presence of pro-coagulation factors
  - Cancer

# Etiology Of Ischemic Stroke:

## LARGE VESSEL EMBOLIC:

- **The Heart**
  - Valve diseases, A. Fib, Dilated cardiomyopathy, Myxoma
- **Arterial Circulation (artery to artery emboli)**
  - Atherosclerosis of carotid, Arterial dissection, Vasculitis
- **The Venous Circulation**
  - PFO w/R to L shunt, Emboli

# What is a TIA?

Transient ischemic attack (TIA) is defined as a transient episode of neurologic dysfunction caused by focal brain, spinal cord, or retinal ischemia, without acute infarction



# Determining the Location

- Large Vessel:
  - Look for cortical signs
- Small Vessel:
  - No cortical signs on exam
- Posterior Circulation:
  - Crossed signs
  - Cranial nerve findings
- Watershed:
  - Look at watershed and borderzone areas
  - Hypo-perfusion

# Cortical Signs

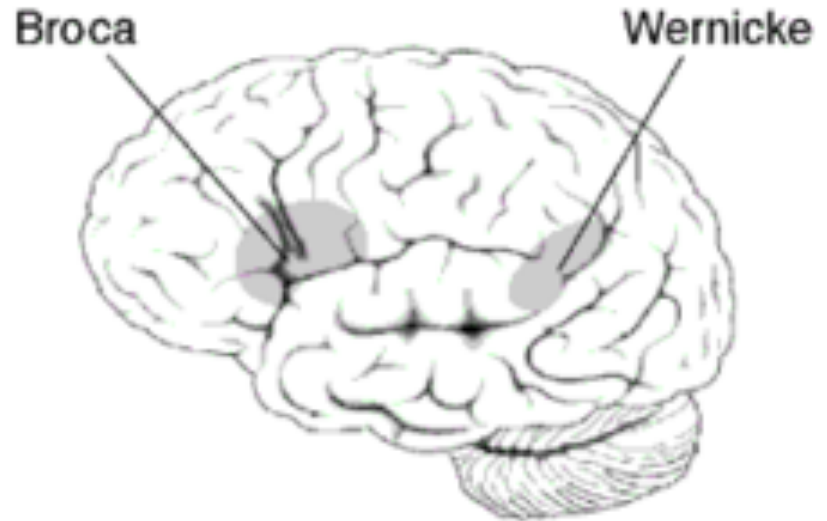
<b>RIGHT BRAIN:</b>	<b>LEFT BRAIN:</b>
- Right gaze preference	- Left gaze preference
- Neglect	- Aphasia

- If present, think **LARGE VESSEL** stroke

There are things  
we know. There  
are things we  
own unknowns. That  
is, there are things  
we know we don't  
know. But there are also  
things we don't know  
we don't know.

# Aphasia

- Broca's
  - **Expressive** aphasia
  - Left posterior inferior frontal gyrus
- Wernicke's
  - **Receptive** aphasia
  - Posterior part of the superior temporal gyrus
  - Located on the dominant side (left) of the brain





# Large Vessel Stroke Syndromes

- MCA:
  - Arm>leg weakness
  - LMCA cognitive: Aphasia
  - RMCA cognitive: Neglect, topographical difficulty, apraxia, constructional impairment
- ACA:
  - Leg>arm weakness, grasp
  - Cognitive: muteness, perseveration, abulia, disinhibition
- PCA:
  - Hemianopia
  - Cognitive: memory loss/confusion, alexia
- Cerebellum:
  - Ipsilateral ataxia

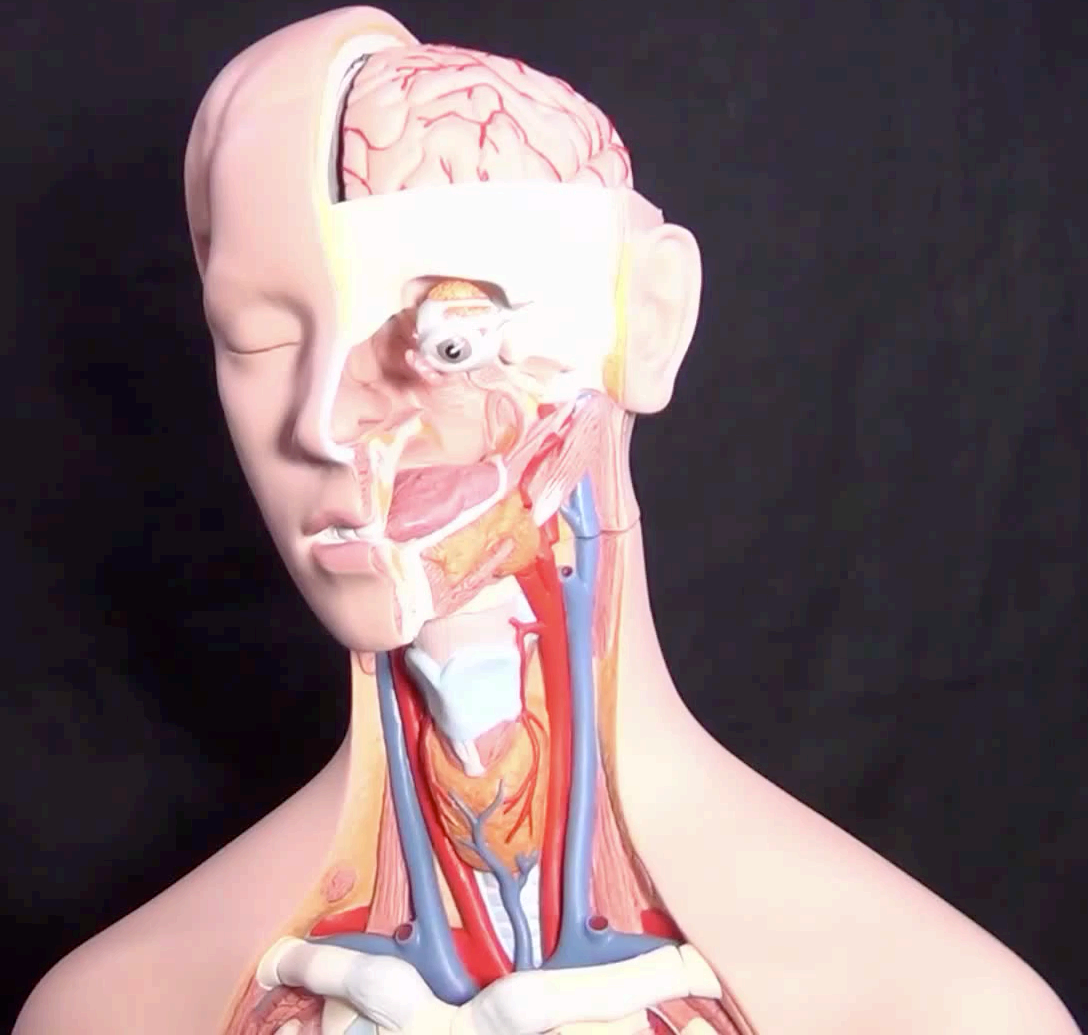
# Cerebral Blood Flow (CBF)

- $CBF = CBV/t$
- 750 mL/minute, which is 15% of the cardiac output
- The normal cerebral blood flow is 45-50ml/100g/min, ranging from 20ml 100g<sup>-1</sup> min<sup>-1</sup> in white matter to 70ml 100g<sup>-1</sup> min<sup>-1</sup> in grey matter. Highest in neurohypophysis

# CBF

- When CBF falls to less than 10-23ml/100g/min, physiological electrical function of the cell begins to fail- “ischemic penumbra”.
- Below 8 ml/100g/min irreversible cell death- ionic membrane transport failure





# Cerebral Perfusion Pressure (CPP)

- Cerebral Perfusion Pressure (CPP)

$$\text{MAP} - \text{ICP} = \text{CPP}$$

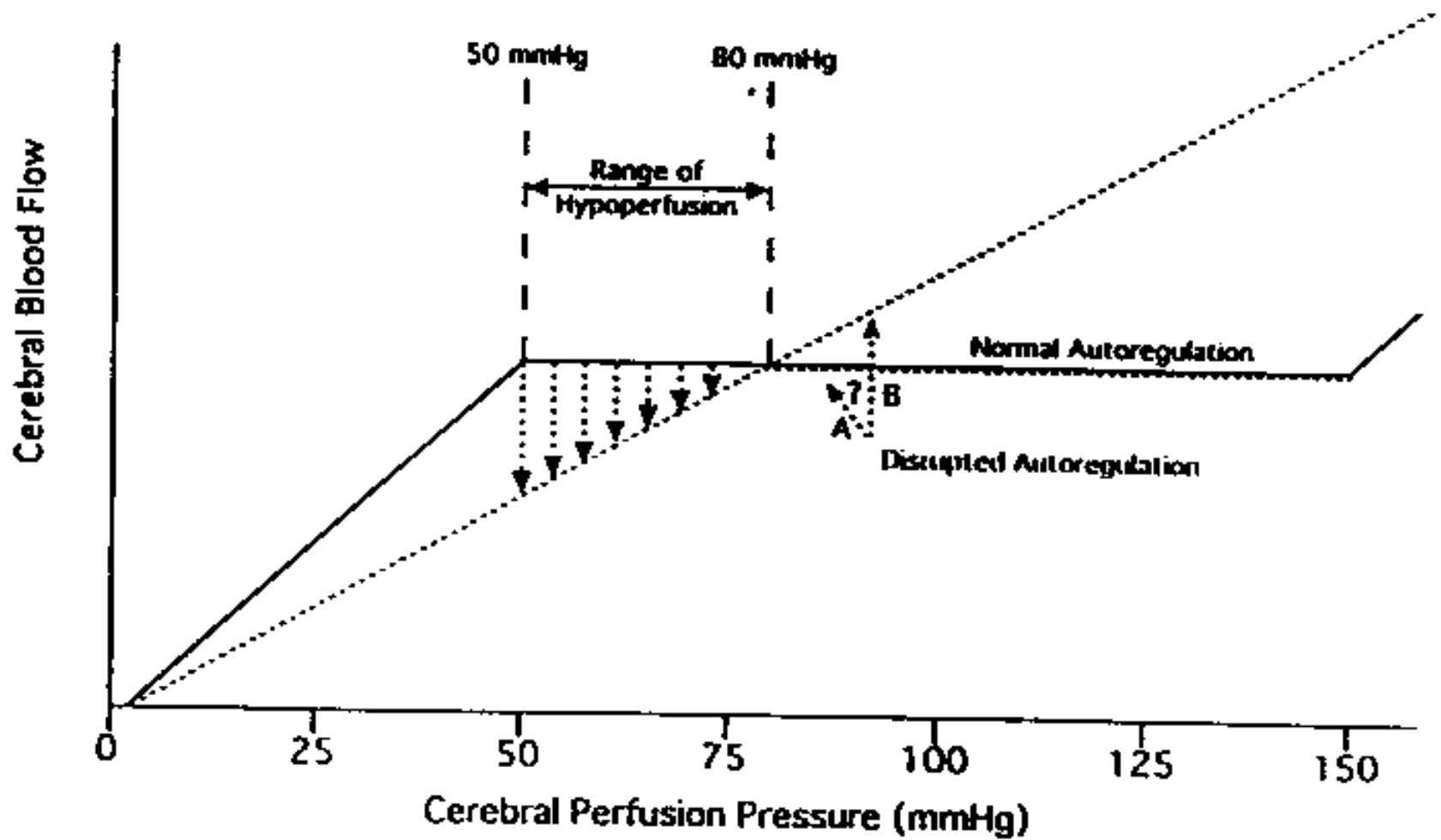
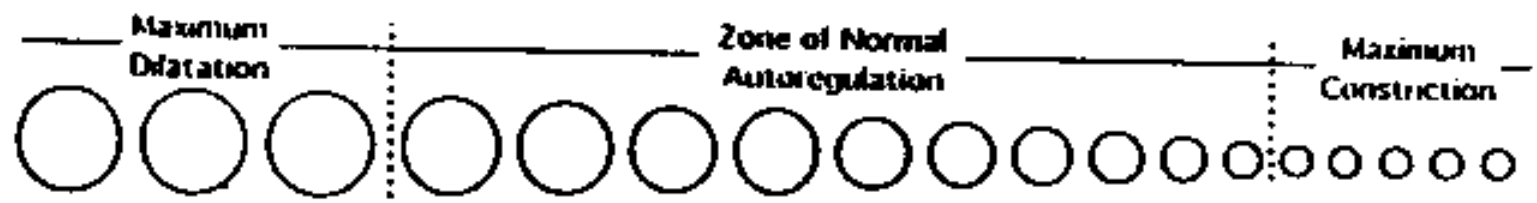
normal CPP is between 50-150 mmHg

- <50 mmHg --> ischemia
- >150 mmHg --> hyperemia

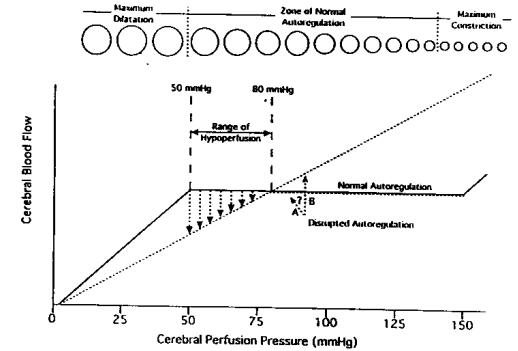
# Autoregulation

- CBF is maintained at a constant level in normal brain in the face of the usual fluctuations in blood pressure by the process of autoregulation. It is a poorly understood local vascular mechanism. Normally autoregulation maintains a constant blood flow between CPP 50 mmHg and 150 mmHg.
- Poiseuille's law- flow through a rigid vessel:

$$Q = \Delta P \pi r^4 / 8L\eta$$



# Autoregulation



- Dysregulation can occur in pathologic states
- In traumatised or ischaemic brain, or following vasodilator agents (volatile agents and sodium nitroprusside) CBF may become blood pressure dependent. Thus as arterial pressure rises so CBF will rise causing an increase in cerebral volume. Similarly as pressure falls so CBF will also fall, reducing ICP, but also inducing an uncontrolled reduction in CBF.

# Autoregulation

- **pressure/myogenic autoregulation**

arterioles dilate or constrict in response to changes in BP and ICP in order to maintain a constant CBF

- “myogenic theory”- vascular smooth muscle within cerebral arterioles intrinsically contract to stretch thereby regulating pressure
- NO- limited role overall, but if completely abolish NO production then loss of autoregulation; with CBF being completely BP-dependent

# Metabolic Autoregulation

- arterioles dilate in response to potent chemicals that are by-products of metabolism such as lactic acid, carbon dioxide and pyruvic acid

CO<sub>2</sub> is a potent vasodilator

increased CO<sub>2</sub>/decreased BP --> vasodilation

decreased CO<sub>2</sub>/increased BP -> vasoconstriction

# Neurogenic Autoregulation

- Autonomic- sympathetic adrenergic receptors seen in cortical layers IV and V.
- $\beta_1$ ,  $\beta_2$ , and  $\alpha_2$  (“dilators”), and  $\alpha_1$  (“constrictor”) receptors
- Overall sympathetic system plays minor role unlike in non-cerebral vascular beds.



# Neurogenic Autoregulation- cont

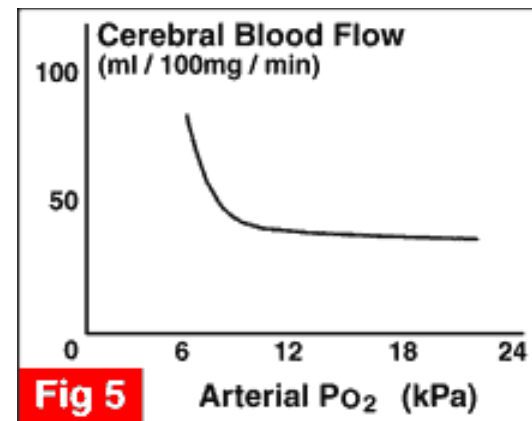
- 5-HT- potent “constrictor,” antagonized by NO
- Neuropeptide Y- “vasoconstriction”, in assoc with NO and sympathetic system
- Vasoactive intestinal polypeptide (VIP) and peptide histidine isoleucine (PHI)- “vasodilators”
- Substance P, neurokinin A, calcitonin gene-related peptide histamine H<sub>2</sub> -”vasodilatory” esp. substance P
- CCK, neurotensin, somatostatin, vasopressin, endorphin

# Neurogenic Autoregulation-cont

- Autonomic system and neurochemical control of CBF in general is a minor control
- Overall, pressure and metabolic autoregulation most important

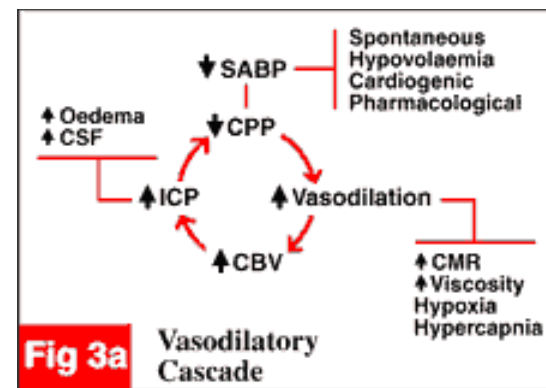
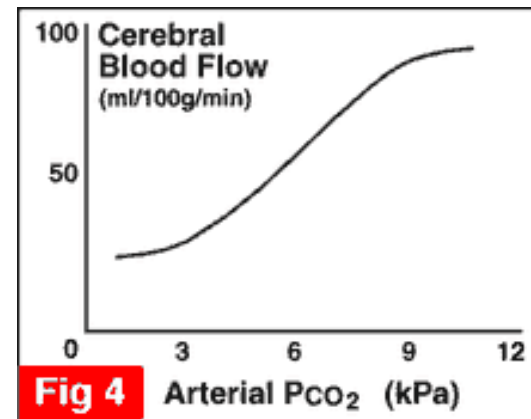
# Increasing CBF-Hyperemia

- Low arterial oxygen tension has profound effects on cerebral blood flow. When it falls below 50 mmHg (6.7 kPa), there is a rapid increase in CBF and arterial blood volume



# CBF and CO<sub>2</sub>

- Carbon dioxide causes cerebral vasodilation. As the arterial tension of CO<sub>2</sub> rises, CBV and CBF increases and when it is reduced vasoconstriction is induced.

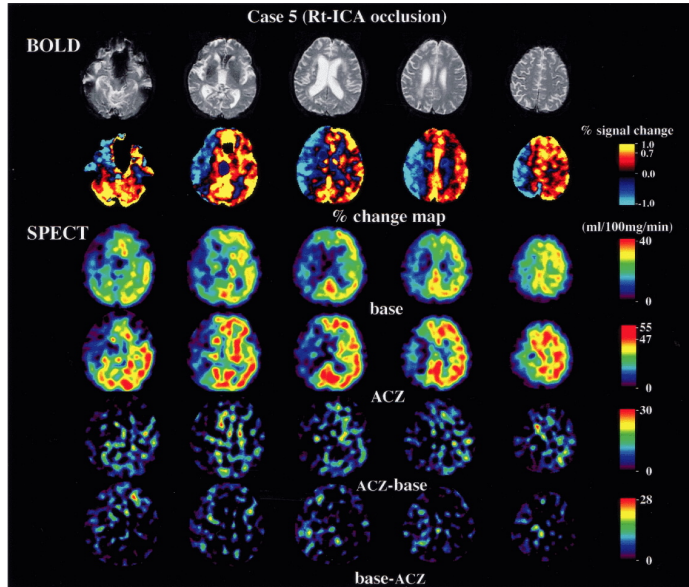


# “Cerebrovascular Reserve”

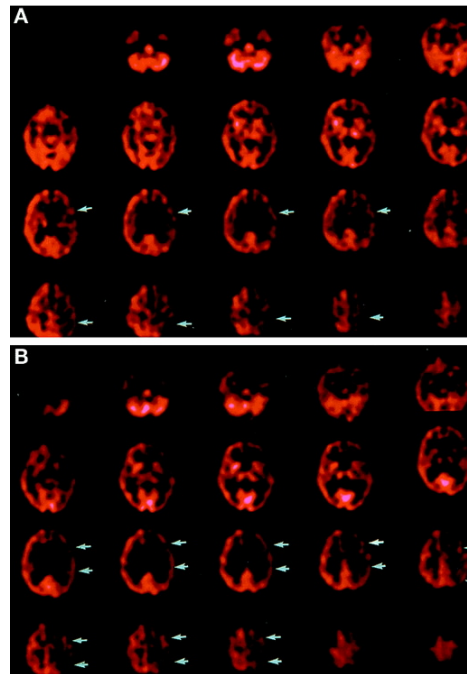
- In functionally activated areas, CBF augmentation exceeds the small increases in oxygen utilization and the concentration of deoxyhemoglobin is relatively low. Thus, this excess supply of oxygen in response to a demand stimulus reflects the *cerebral perfusion reserve capacity*
- Cerebrovascular reserve capacity is impaired by risk factors such as hypertension and diabetes, carotid/cerebral vasc. stenosis, and can be an etiologic factor in ischemic stroke

# Cerebrovascular Reserve

- PET, SPECT, Xe-CT, CT-perfusion to assess. Pre/post diamox challenge.
- acetazolamide challenge and the CO<sub>2</sub>-loading (breath-holding) test raise global CBF
- (MRI) of T<sub>2</sub>-weighted or Blood oxygenation level-dependent (BOLD)-weighted images correlate well with changes in the total amount of oxygenated hemoglobin

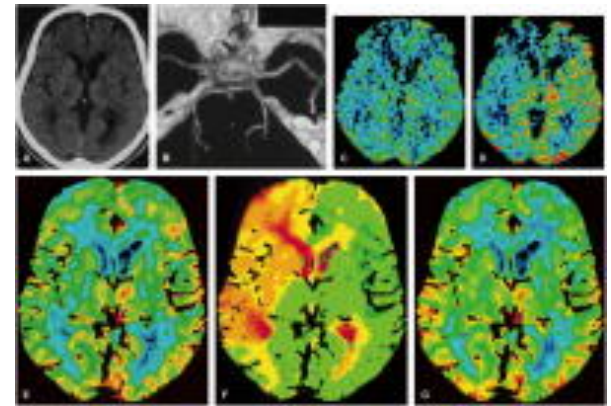


BOLD-MRI and single-photon emission computed tomography (SPECT)



(SPECT)

Xenon CT



perfusion CT





# Who's at Risk?

Every year, nearly 800,000 Americans have a stroke. Here's what increases risk:

- Family history of stroke
- A history of TIAs (Transient Ischemic Attacks)
- Medical conditions such as Afib or fibromuscular dysplasia
- Age – After age 55, stroke risk doubles every decade
- Race – African Americans, Hispanics, and Asian/Pacific Islands have a greater risk
- Gender

# Stroke and Women

Annually, nearly 55,000 more women than men have strokes.

Characteristics that are specific to women, such as taking birth control pills or hormone replacement therapy, can increase stroke risk.

Women who have high blood pressure before or during pregnancy have a greater risk of stroke.

# Ask About the Four H's!

- Heredity
- High Blood Pressure
- Heart Disease
- High Cholesterol

# Heredity

Alert your doctor if someone in your family has had a stroke, high blood pressure, or heart disease.

If you don't know your history, ask around. A sibling or other relative may have more information.

## ***What can you do?***

If you have a family history of stroke, take extra precaution to manage the risk factors you can control.

# High Blood Pressure

Ask your doctor whether you have high blood pressure – the No. 1 cause of stroke.

## *What can you do?*

Start a plan to eat healthier and exercise.

Ask about medications that can be effective in lowering your blood pressure and keeping it down.

# Heart Disease

Determine your overall heart health.

Conditions like atherosclerosis, coronary artery disease, Afib, valve defects, and enlarged heart chambers can block vessels in or leading to the brain.

## ***What can you do?***

Ask you doctor about treatment, such as surgical procedures or taking aspirin or blood-thinning therapy to prevent clots.

# High Cholesterol

Cholesterol is a fatty substance in blood. If there's too much cholesterol in your blood, it can clog arteries and cause a stroke.

**What you want:**

**Total Cholesterol: < 200 mg**

**LDL "Bad" Cholesterol: < 100 mg**

**HDL "Good" Cholesterol: > 40 for men, > 50 for women**

***What can you do?***

Take steps to eat healthier and start exercising.

Ask about medications. They can be effective in reducing your cholesterol and keeping it down.

# More Conditions to Ask About

- Sleep Apnea – Untreated, this can cause high blood pressure, heart failure, irregular heartbeat, heart attacks, and stroke
- Diabetes – People with diabetes are two to four times more likely to have a stroke
- Circulation Problems – Strokes can be caused by complications with any component of your circulation, including your heart, arteries, veins, and blood.



- Hormones – Hormone levels, especially estrogen in women, can play a role in stroke risk.
- Fibromuscular dysplasia – With this condition, some of the arteries that carry blood throughout the body are not fully developed.
- Patent foramen ovale – This is an opening between the two chambers of the heart where blood can clot.

# ***What can YOU do?***

Be aware of these conditions, and talk to your doctor if you're experiencing any of them.

Oftentimes, lifestyle changes, medication, or both can help offset your risk of stroke.

# Stop Smoking

Smoking accelerates the forming of clots, thickens blood, and increases plaque buildup in the arteries. If you smoke, stop!

- Ask your doctor about nicotine patches, counseling, or programs that have worked for others.
- Don't get discouraged – It could take several attempts.
- Keep trying – Quitting smoking can have almost immediate beneficial effects on your health.

# Get to Your Healthy Weight

Carrying extra weight can make you more apt to develop high blood pressure, heart problems, and diabetes – all increasing your risk of stroke.

- Talk to your doctor about your ideal weight, body mass index, and percentage of body fat to make sure you're in a healthy range.
- Losing just 10 pounds can improve your health and reduce your risk of stroke.

# Be Active

Physical activity can help you lose weight and reduce stress which can lower blood pressure and cholesterol, control diabetes, and improve your overall health.

- Talk to your doctor about starting an exercise program.
- Try to be active for 30 minutes every day.
- If you can't do it all at once, try to be active for 10 to 15 minutes at a time.

# Watch What You Eat

This is about nutrition – not dieting. The food choices you make can improve your health and reduce your risk of stroke.

- Eat plenty of fruits and vegetables, which are high in fiber.
- Limit salt to help lower your blood pressure.
- Eat less cholesterol and fat which can create plaque buildup in your arteries.

# Limit Alcoholic Drinks

Drinking too much alcohol can increase blood pressure and the risk of stroke.

- Drink in moderation – No more than two drinks a day for men and one drink a day for women.
- Know your portions – A standard portion is 5 ounces of wine, 12 ounces of beer, or 1.5 ounces of hard liquor.
- Opt for red wine – It contains resveratrol which can protect the heart and brain.

# Reduce Your Risk Recap

## At the doctor

Assess the 4 Hs:

- Heredity
- High blood pressure
- Heart disease
- High cholesterol

Take steps to treat:

- Sleep Apnea
- Diabetes
- Circulation problems
- Hormone issues
- Heart issues, such as , AFib, fibromuscular dysphasia, or patent foramen ovale.

## At home

- Stop smoking
- Maintain a healthy weight
- Be Active
- Watch what you eat
- Drink less alcohol





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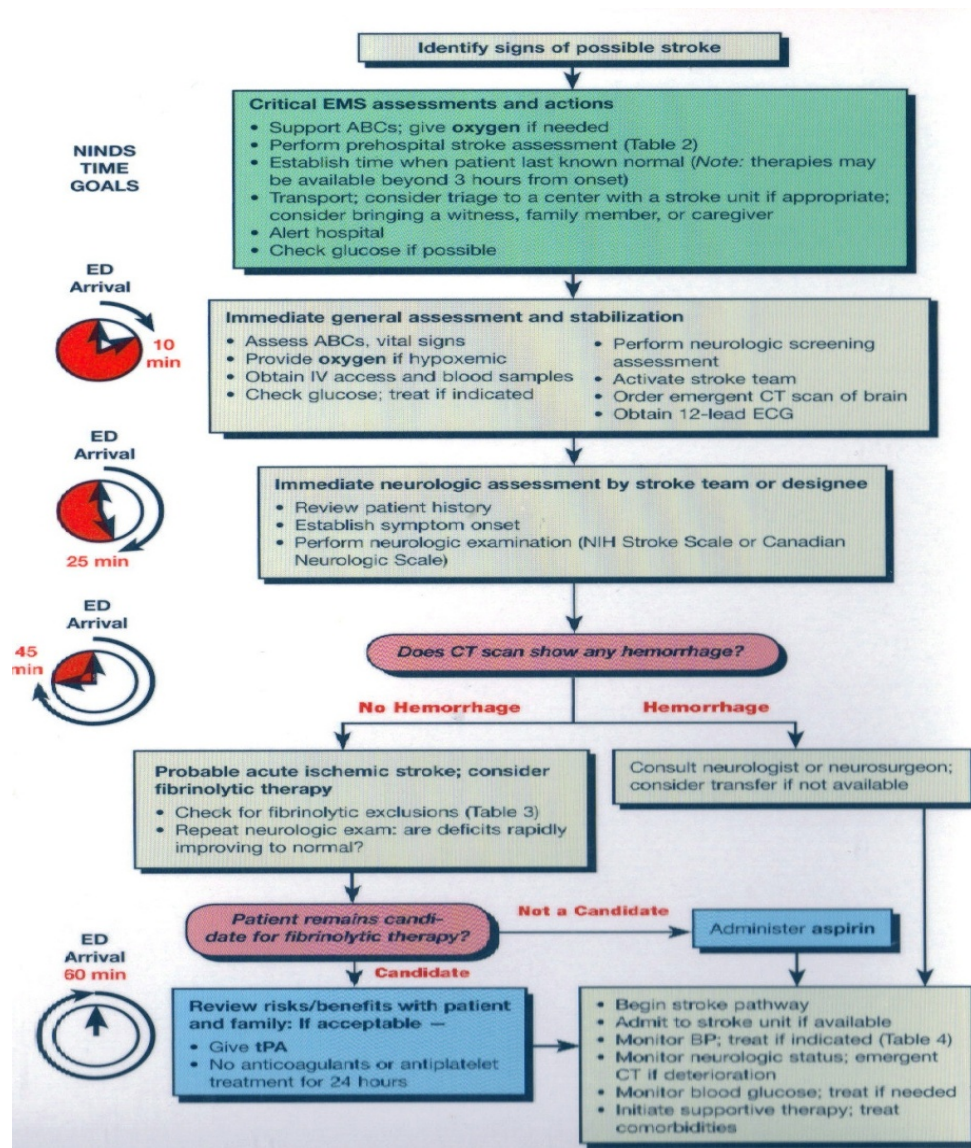
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# Ischemic Sindroms

- Transient ischemic attack (TIA), isolated or multiple
- Acute infarct
- Infarct with hemorrhagic complication

# Stroke Algorithm

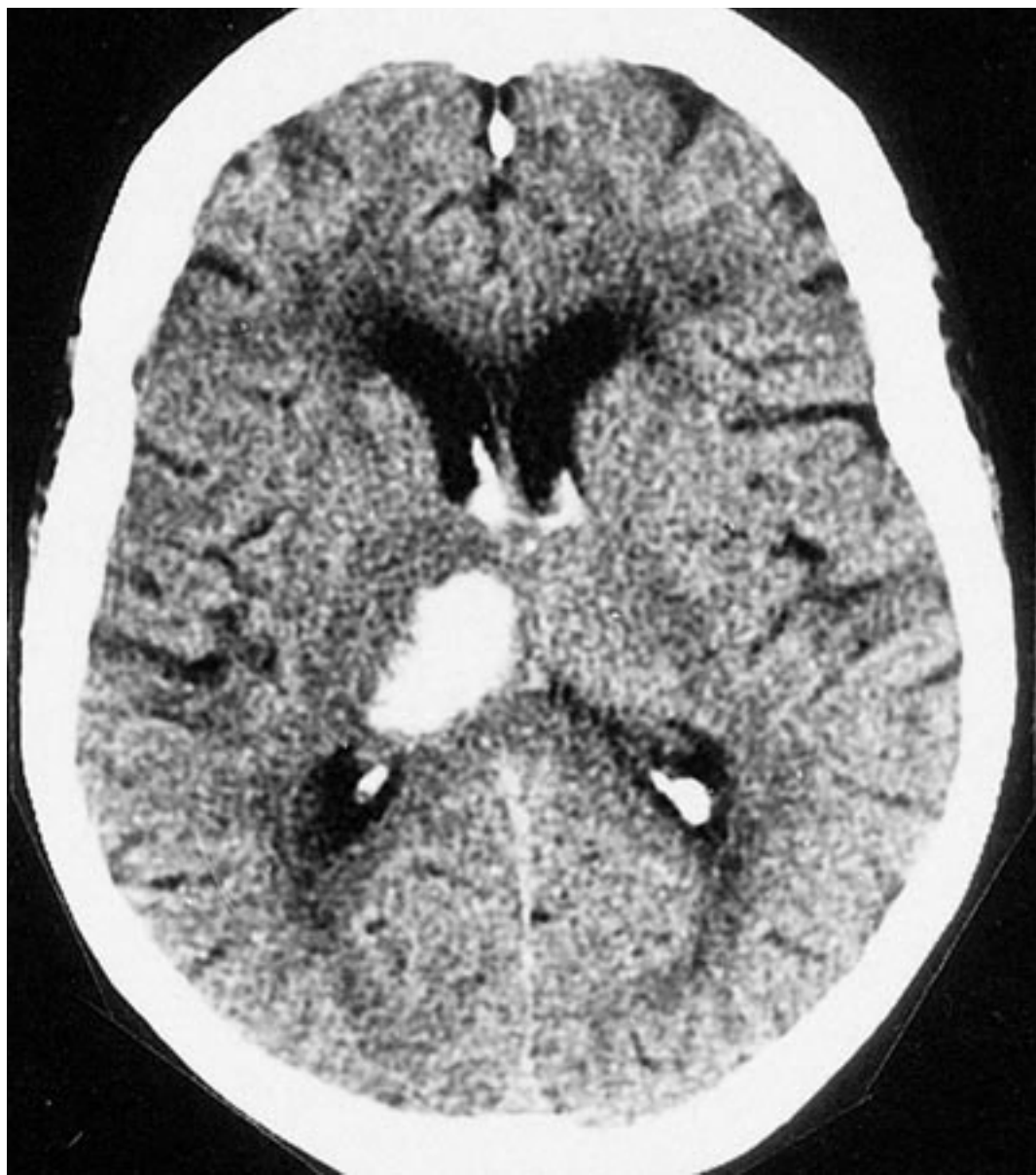


# Intracranial Hemorrhages

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# Hemorrhage at CT



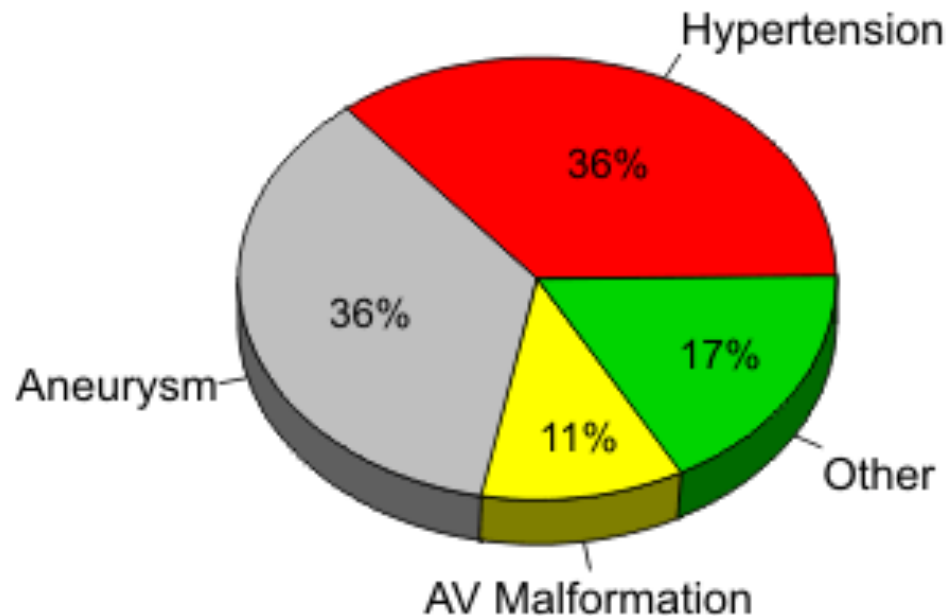
# Etiology of ICH

- Traumatic
- Spontaneous
  - Hypertensive
  - Amyloid angiopathy
  - Aneurysmal rupture
  - Arteriovenous malformation rupture
  - Bleeding into tumor
  - Cocaine and amphetamine use

# Causes of ICH

## Intracerebral Hemorrhage

Non-traumatic



Other causes: bleeding into tumor, hypocoagulable state, hemorrhagic infarction, iatrogenic, and trauma



# Hypertensive ICH

- Spontaneous rupture of a small artery deep in the brain
- Typical sites
  - Basal Ganglia
  - Cerebellum
  - Pons
- Typical clinical presentation
  - Patient typically awake and often stressed, then abrupt onset of symptoms with acute decompensation

# Ganglionic Bleed

- Contralateral hemiparesis
- Hemisensory loss
- Homonymous hemianopia
- Conjugate deviation of eyes toward the side of the bleed or downward
- AMS (stupor, coma)

# Cerebral Hemorrhage



# Cerebellar Hemorrhage

- **Vomiting** (more common in ICH than SAH or Ischemic CVA)
- Ataxia
- Eye deviation toward the opposite side of the bleed
- Small sluggish pupils
- AMS

# Cerebellar Hemorrhage



# Pontine Hemorrhage

- Pin-point but reactive pupils
- Abrupt onset of coma
- Decerebrate posturing or flaccidity
- Ataxic breathing pattern

# Pontine Hemorrhage



# Subarachnoid Hemorrhage

- “Worst headache of my life”
- AMS
- Photophobia
- Nuchal rigidity
- Seizures
- Nausea and vomiting



# Subarachnoid Hemorrhage





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**Management**

# Airway

- Most likely related to decreased level of consciousness (LOC), dysarthria, dysphagia
- GCS < 8 - INTUBATE
- Avoid Hyperventilation or Hypoventilation
- NPO until swallow assessment completed- high aspiration risk
- Begin mobilization as soon as clinically safe
- Keep HOB greater than 30 degrees

# Imaging

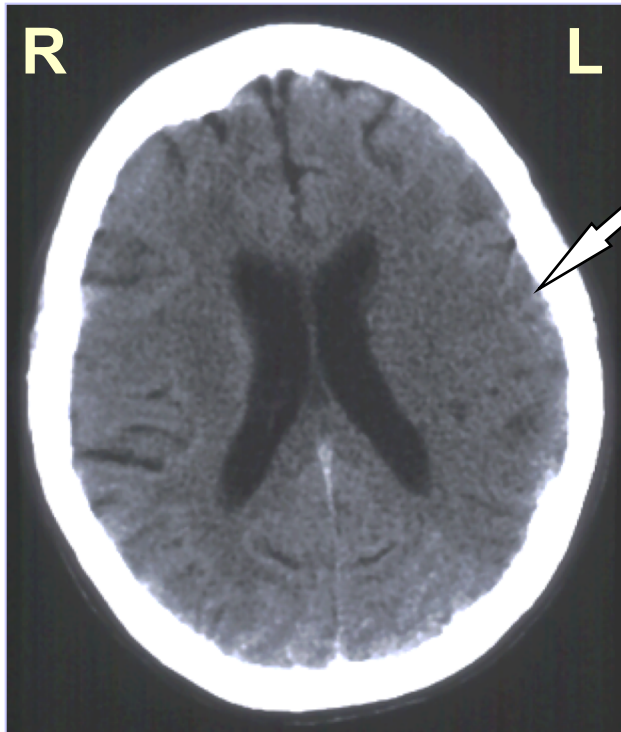
## CT scan

- Non-contrast CTH remains the gold standard as it is superior for showing IVH and ICH
- CT with contrast may help identify aneurysms, AVMs, or tumors but is not required to determine whether or not the patient is a tPa candidate

## MRI

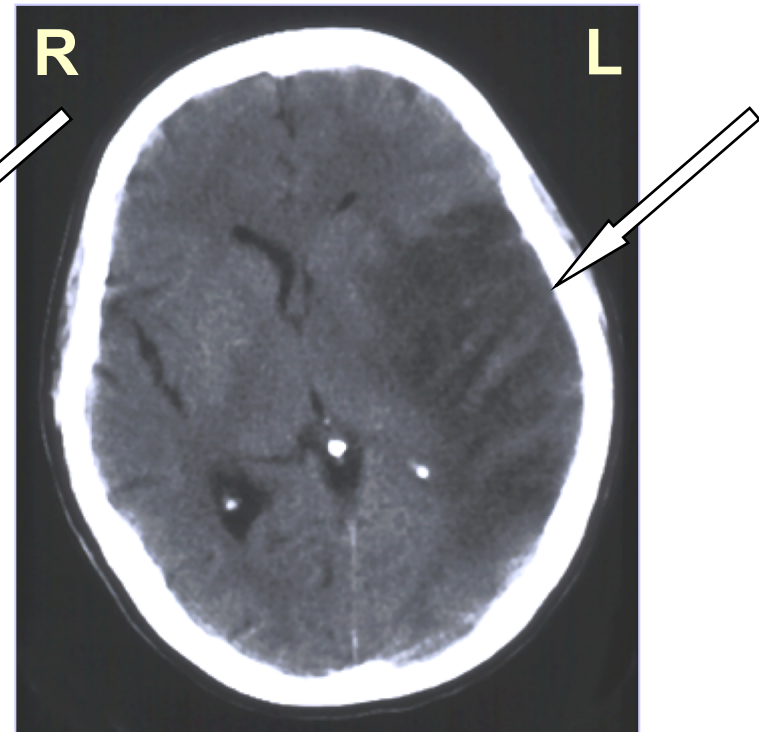
- Superior for showing underlying structural lesions
- Contraindications

**Acute (4 hours)  
Infarction**



Subtle blurring of gray-white junction & sulcal effacement

**Subacute (4 days)  
Infarction**



Obvious dark changes & "mass effect" (e.g., ventricle compression)

# Multimodal Imaging

## Multimodal CT

- Typically includes non-contrast CT, perfusion CT, and CTA
- Two types of perfusion CT
  - Whole brain perfusion CT
  - Dynamic perfusion CT

## Multimodal MRI

- Standard MRI sequences ( T1 weighted, T2 weighted, and proton density) are relatively insensitive to changes in cerebral ischemia
- Multimodal adds diffuse-weighted imaging (DWI) and PWI (perfusion-weighted imaging)

# tPa

## Fast Facts

- Tissue plasminogen activator
- “clot buster”
- IV tpa window 3 hours
- IA tpa window 4.5 hours
- Disability risk ↓ 30% despite ~5% symptomatic ICH risk

## Contraindications

- Hemorrhage
- SBP > 185 or DBP > 110
- Recent surgery, trauma or stroke
- Coagulopathy
- Seizure at onset of symptoms
- NIHSS >21
- Age?
- Glucose < 50



# Mechanical Thrombolysis

- Often used in adjunct with tPa
- MERCI (Mechanical Embolus Removal in Cerebral Ischemia) Retrieval System is a corkscrew-like apparatus designed to remove clots from vessels
- PENUMBRA system aspirates the clot

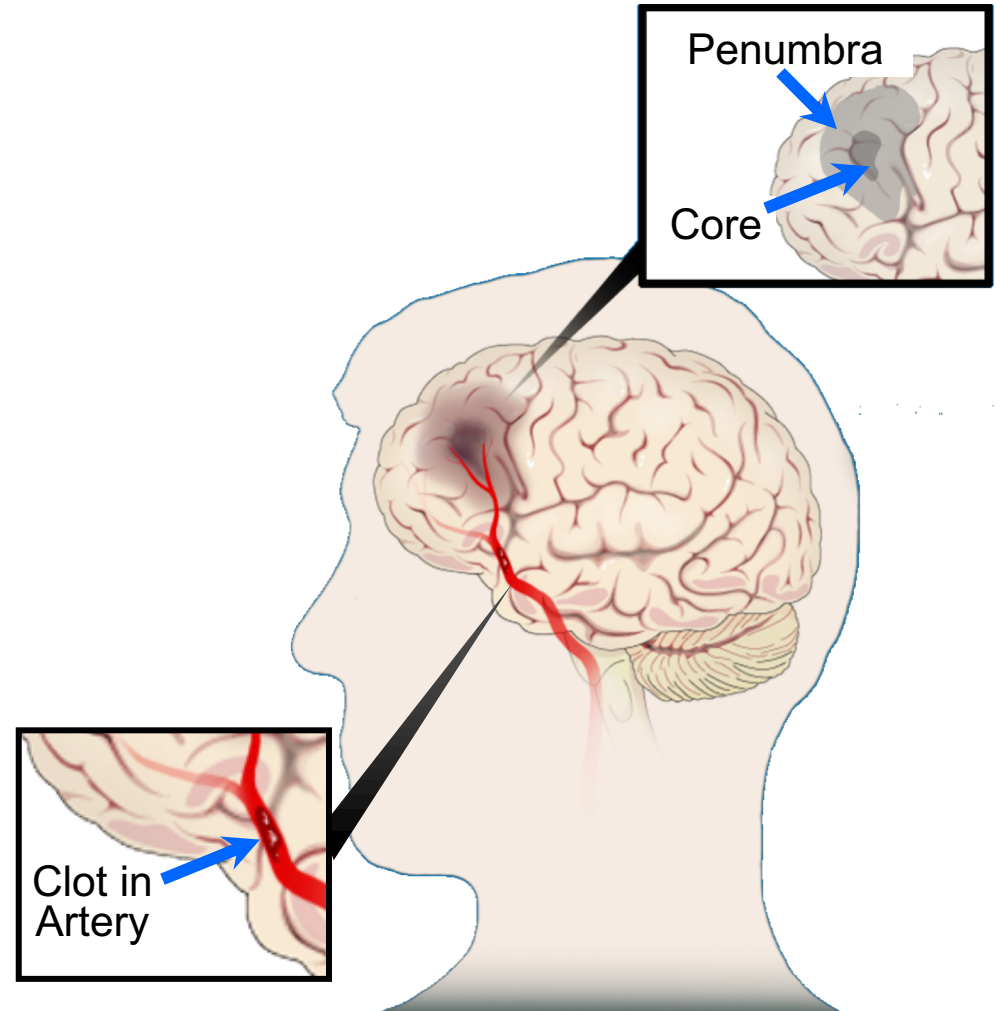
# Blood Pressure Management

- BP Management

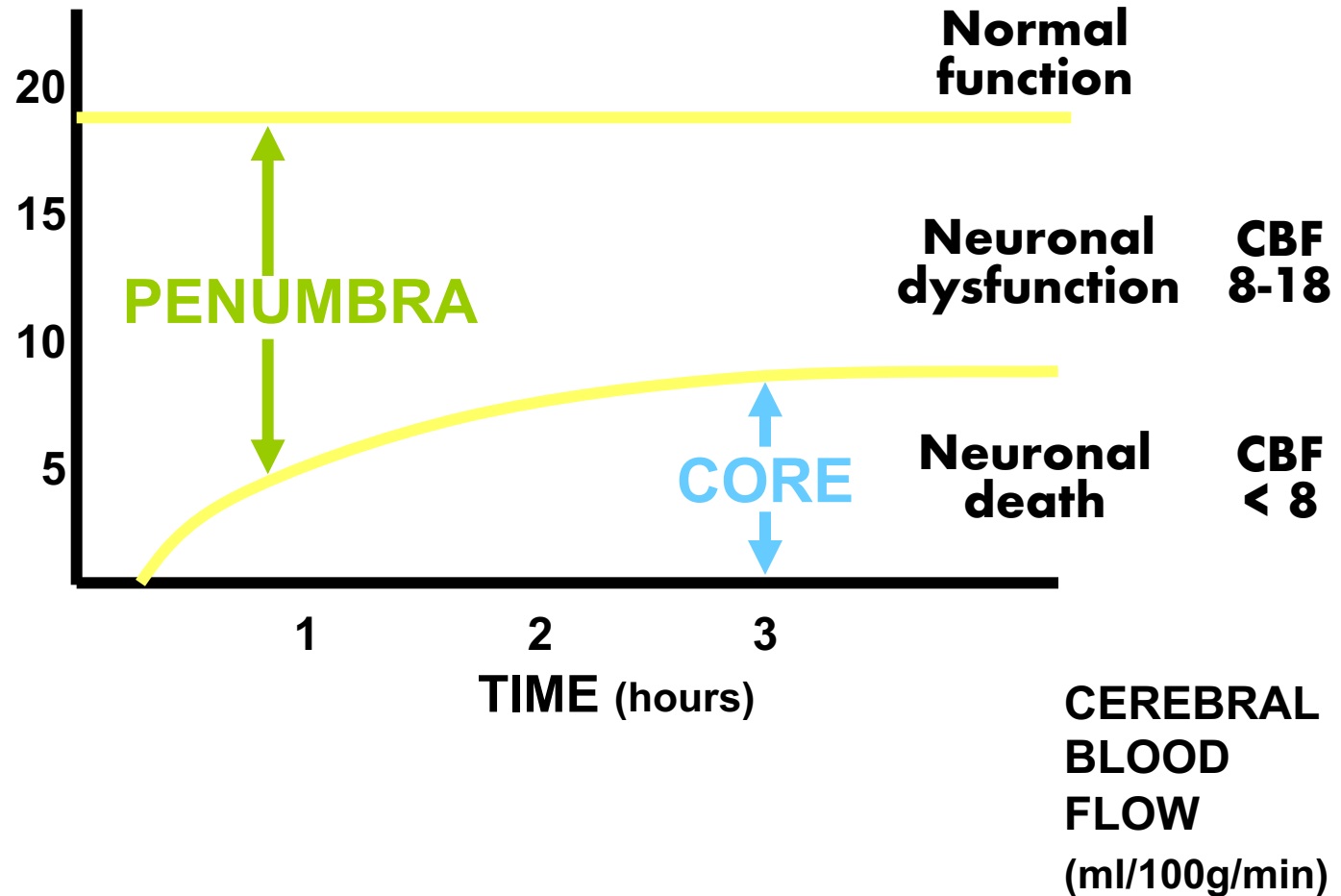
- **The goal is to maintain cerebral perfusion!!**
- $CPP = MAP - ICP$  (needs to be at least 70)
- Higher BP goals with Ischemic stroke
- Lower BP goals with Hemorrhagic stroke (avoid hemorrhagic expansion, especially in AVMs and aneurysms)

# BP-AIS Relationship

- BP increase is due to arterial occlusion (i.e., an effort to perfuse penumbra)
- Failure to recanalize (w/ or w/o thrombolytic therapy) results in high BP and poor neuro outcomes
- Lowering BP starves penumbra, worsens outcomes



# Save the Penumbra!!



# Supportive Therapy

- Glucose Management
  - Infarction size and edema increase with acute and chronic hyperglycemia
  - Hyperglycemia is an independent risk factor for hemorrhage when stroke is treated with t-PA
- Antiepileptic Drugs
  - Seizures are common after hemorrhagic CVAs
  - ICH related seizures are generally non-convulsive and are associated to with higher NIHSS scores, a midline shift, and tend to predict poorer outcomes