

# Neurological Emergencies

## REVIEW

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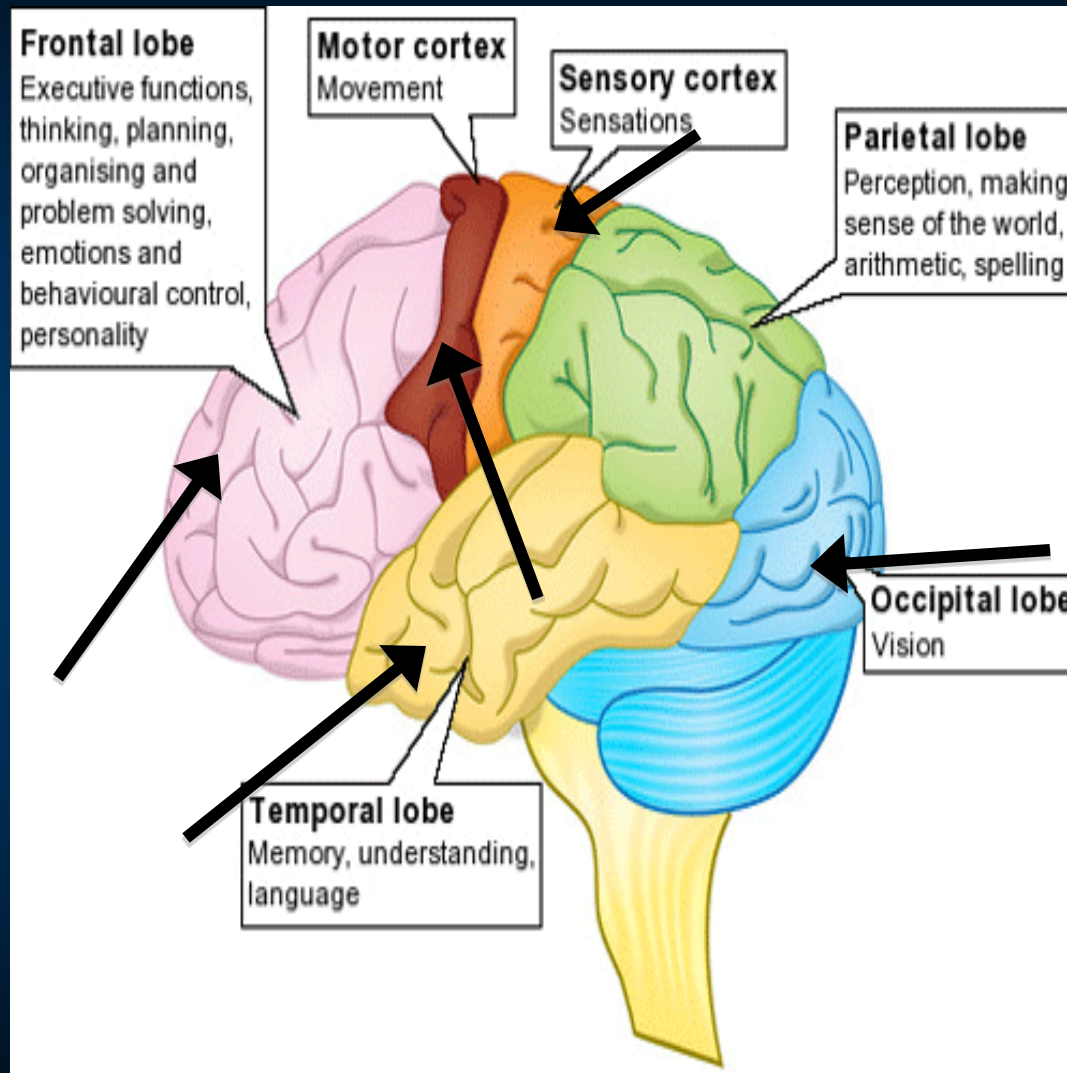
# Goal

- New information on Differential Dx of:
- Hemorrhagic vs. Ischemic Stroke

# The areas of the Brains

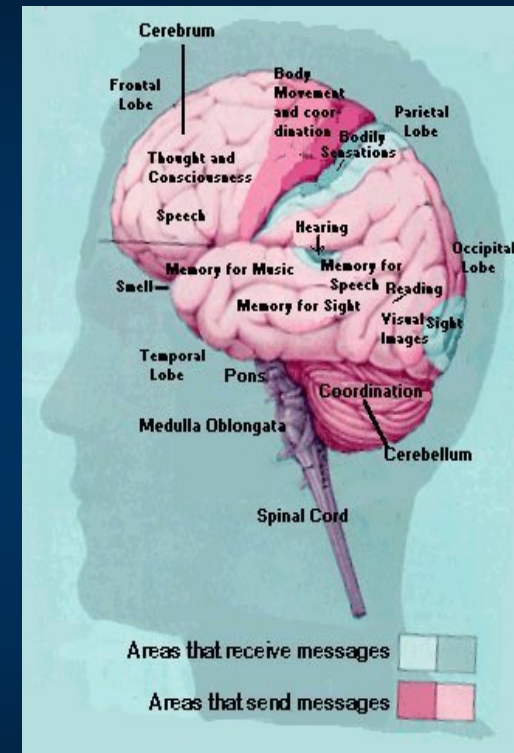
- Frontal Lobe- behavior, personality, sinus
- Parietal- speech, pupils vision
- Occipital- vision, heart rate, balance
- Temporal- acoustics, facial
- Brainstem- Pupil size, BP, HR, Balance

# Cerebral Circulation

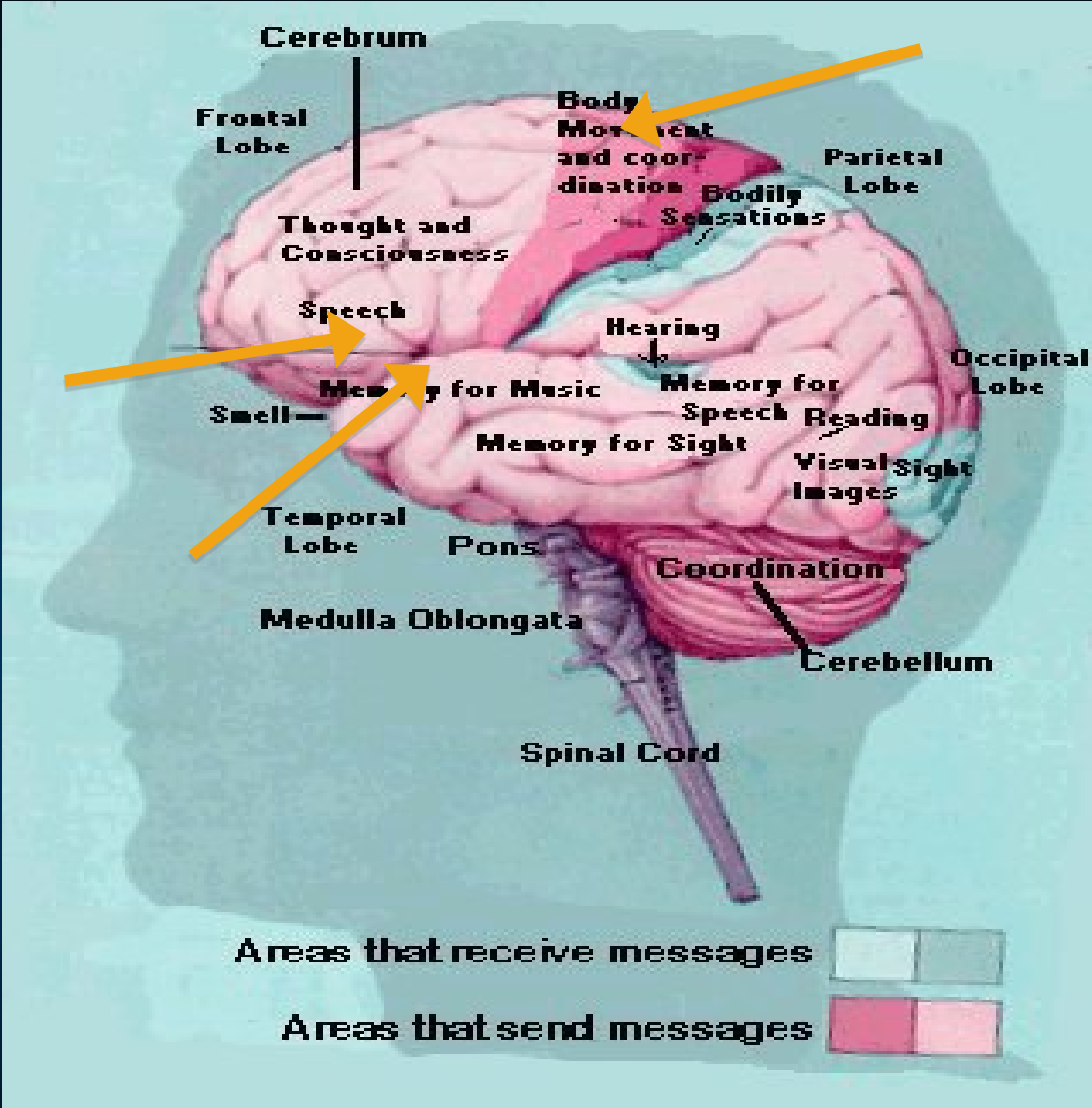


# Cincinnati stroke scale

- Facial droop: motor facial nerves
- Arm drift: motor cortex
- Speech: speech center



72% accurate to stroke if 1 or > criteria met



# Neurological Emergencies

- Acute Ischemic Stroke
- Intracranial Hemorrhage/ Hemorrhagic Stroke

# Acute Ischemic Stroke (AIS)

- AIS is caused by the sudden loss of blood circulation to an area of the brain resulting in ischemia and corresponding loss of neurological function.
- *Identical to Myocardial Ischemia*
- The goal of treatment for AIS is to prevent further damage hypoxic tissue.

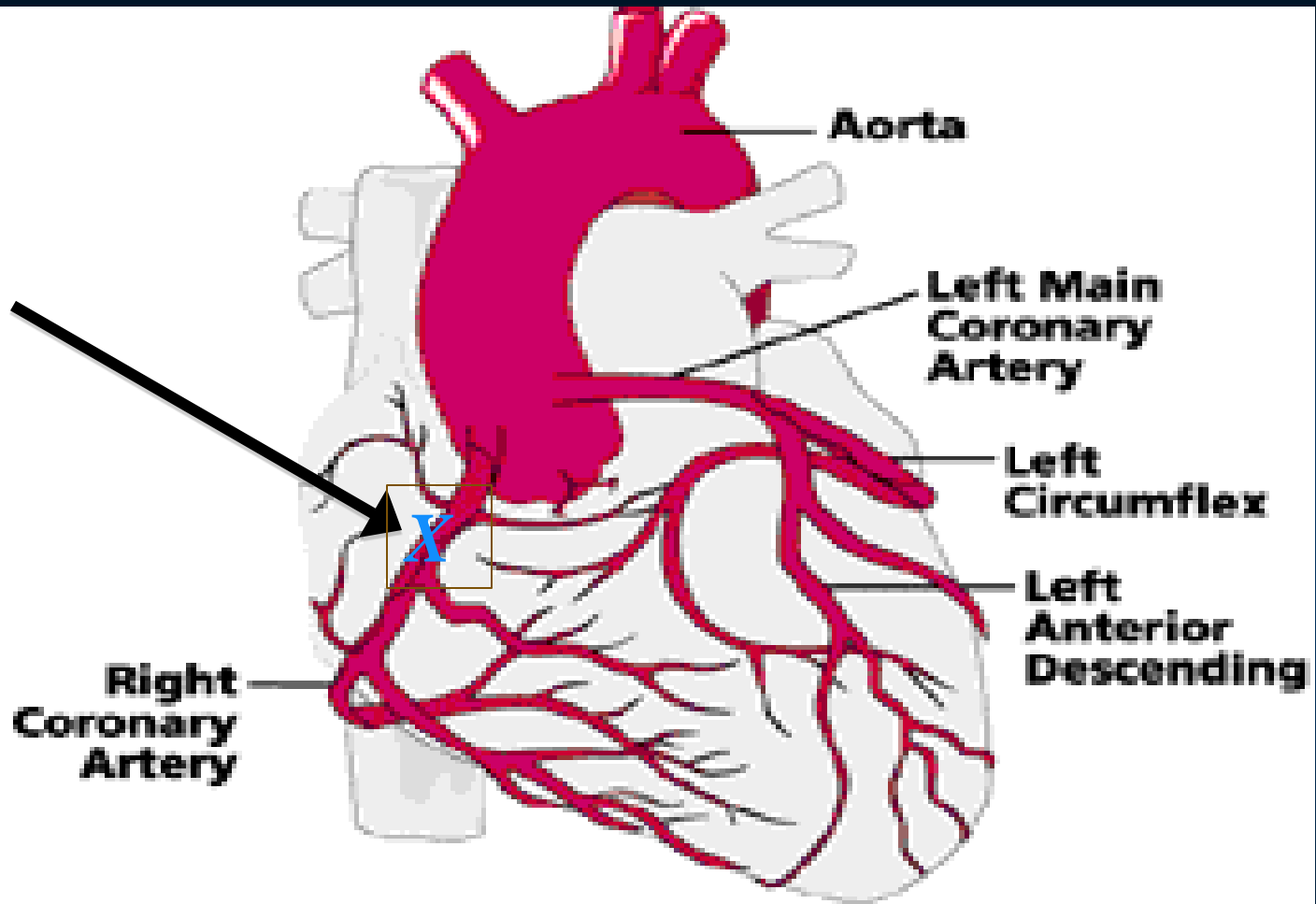


# Acute Ischemic Stroke (AIS)

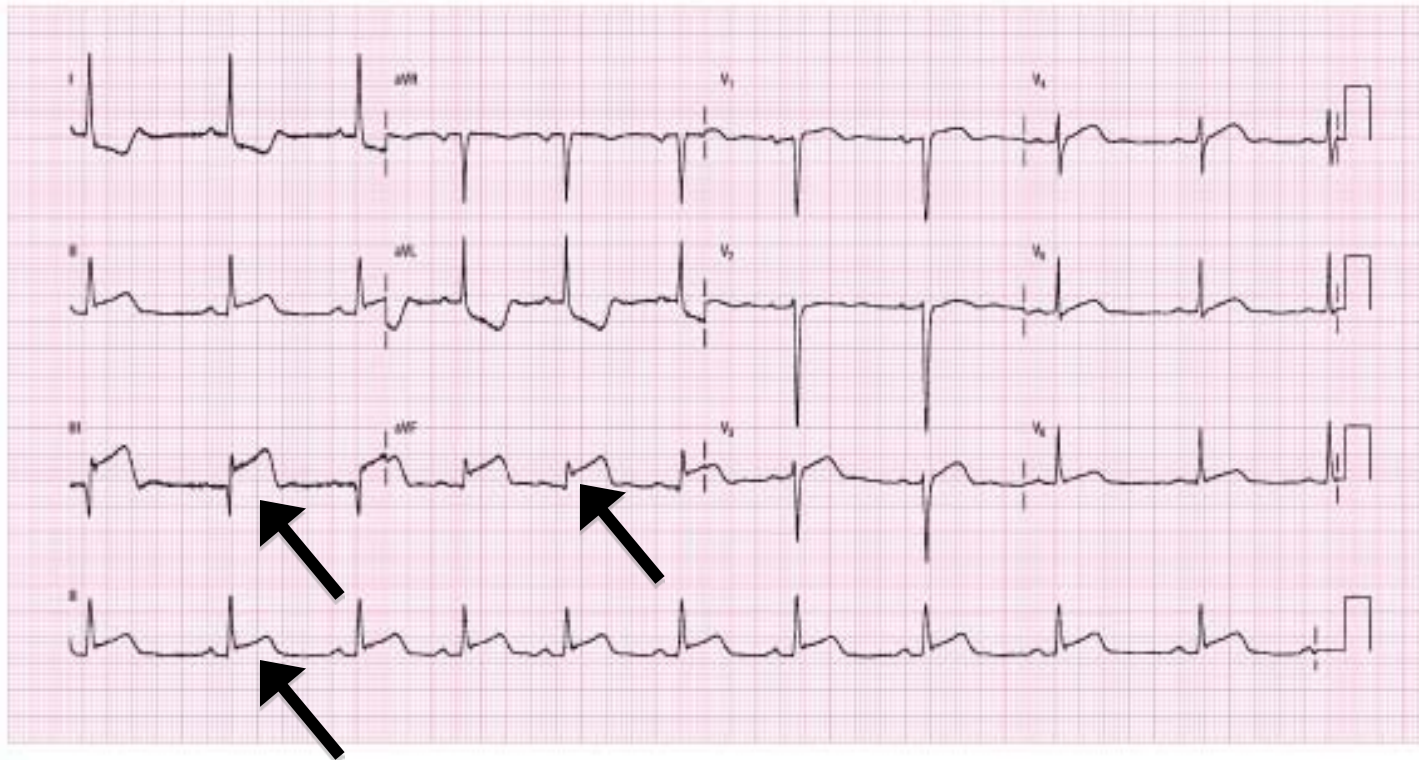
## Risk Factors

- Older Age
- High BP
- TIA History
- Diabetes
- High Cholesterol
- Tobacco Use
- Atrial Fibrillation

# MI and CVA Similarities



# Thoughts????

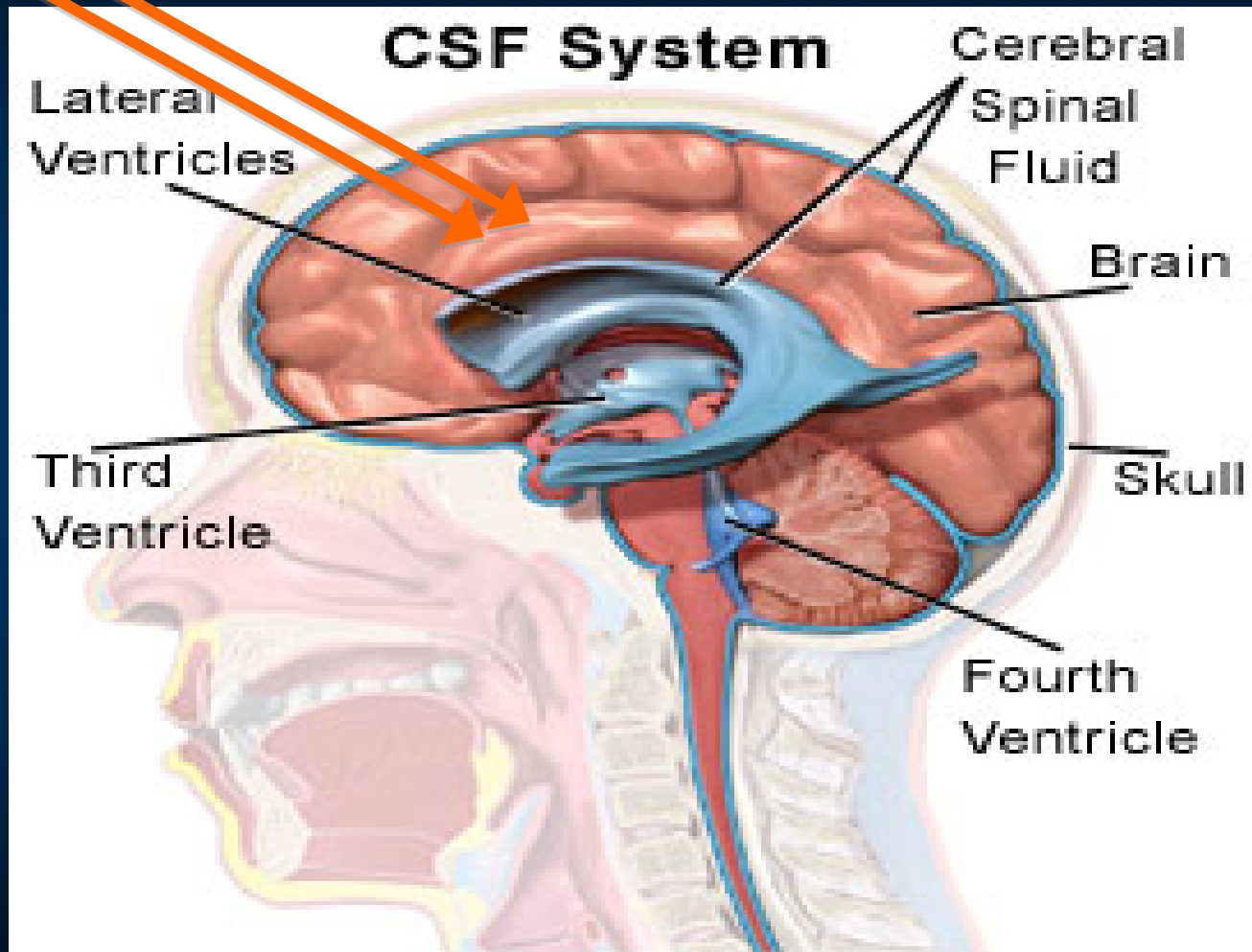


# What I do not have to definitively diagnose CVA



naughty

# Cerebral Ventricles



Within seconds to minutes of loss of perfusion, an ischemic cascade is unleashed **resulting in a central area of irreversible infarction (similar to a burn)** *surrounded by an area of potentially reversible ischemic tissue.*



# Acute Ischemic Stroke

## Ischemic Cascade

- Loss of O<sub>2</sub> and **Glucose** delivery to the brain cell results in cellular depolarization
- The resulting **Ca** - the un-oxygenated effected brain tissue is in "seizure", but the motor center is not yet impacted



# Acute Ischemic Stroke

## Ischemic Cascade

- Seizure or over stimulation of areas causing a brain/motor/sensory tetanic response, followed by flacid/non functioning motor responses.
- Edema ensues causing the release of  $H^+$ , Lactic Acid and  $K^+$ ...effecting non ischemic tissues and cells

# Acute Ischemic Stroke

## Clinical Presentation

- No 100% clinical feature reliably distinguishes AIS from hemorrhagic stroke
- Signs/Symptoms: headache, N/V, and altered mental status, time of day am:
  - Early daylight vs. mid day / evening
- PMH of HTN, change in GCS of 1 pt at one time make ischemic stroke more likely.

# Acute Ischemic Stroke

## Clinical Presentation

- Common symptoms of AIS include the **abrupt onset of hemiparesis**, ataxia, vertigo, aphasia, or **change in GCS by one point at a time**
- Establishing the onset of symptoms is essential when considering possible, **one sided visual loss** thrombolytic therapy.

# ACI Statistical PEARLS

- AM early morning Stroke
- 90% of strokes are ischemic
- AV PU rule = 94% CORRECT

# Acute Ischemic Stroke

## TIA

### Transient Ischemic Attack

- TIA's are defined as a transient ischemic neurological deficit that resolves within 24 hours
- 80% resolve within 60 minutes
- TIA's precede 30% of AIS
- Left untreated, 3/10 TIA's progress to stroke (20% within the first month and 50% within the first year)

# Acute Ischemic Stroke

## Physical Examination

- Goal of PE is to look for extra cranial causes of AIS and to distinguish AIS from stroke mimics ie other differential diagnoses (seizures, tumors, toxic-metabolic disturbances, positional vertigo, etc).
- HEENT: Look for trauma signs and nuchal rigidity, evaluate pulse strength.
- C/V: Signs of CHF / LHF (why??) secondary to : Atrial fibrillation and other arrhythmias.

# CVA Mimic's

## Presumptive Differential Diagnosis

### Adult

Hypoxia

Seizure

Infection (meningitis, encephalitis)

Hypertension

Drug or alcohol ingestion

Electrolyte disturbance

Hypoglycemia

Trauma (head or spine)

Shock

Anemia

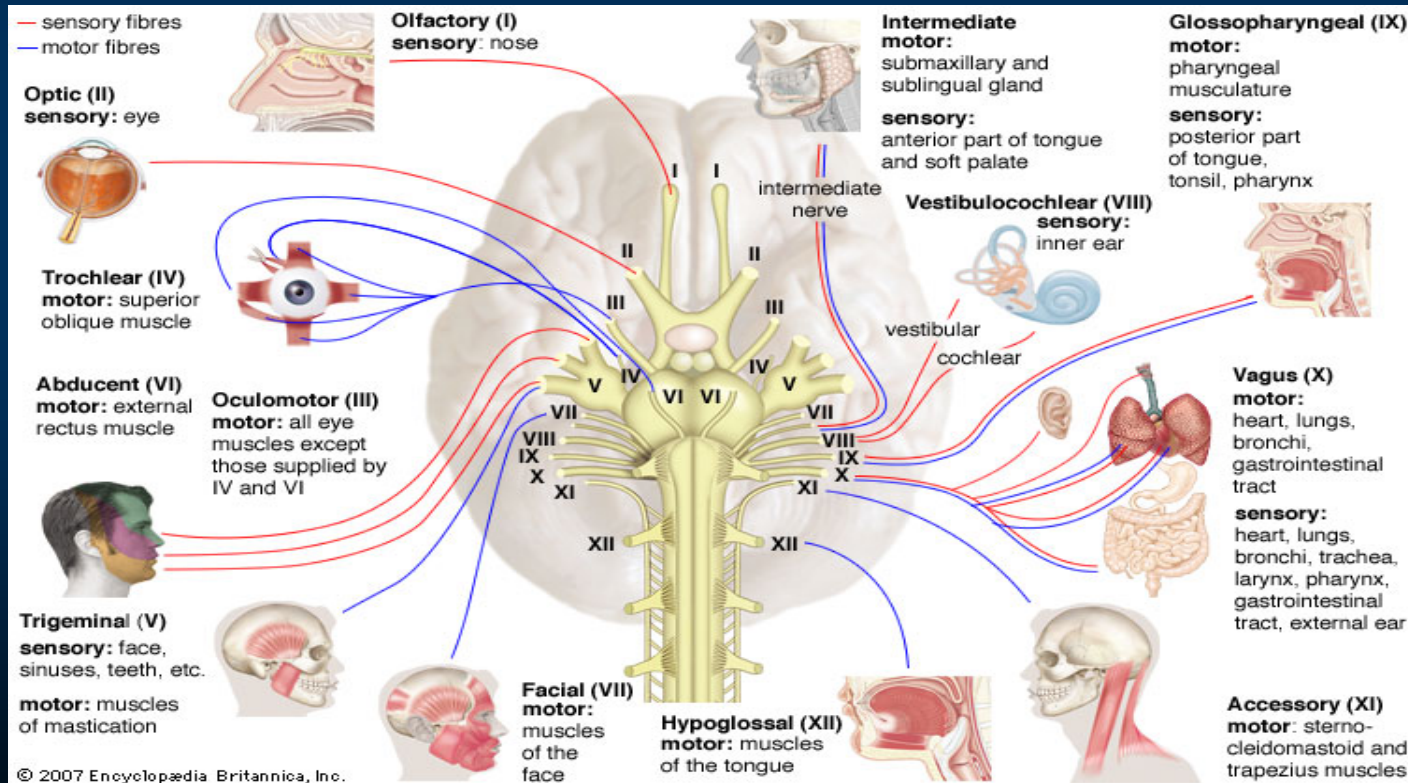
Psychiatric

Environmental (heat/cold exposure, CO poisoning)

# Acute Ischemic Stroke

## Neurological Exam

- Goal – establish baseline for monitoring response to therapy and to determine size and location of AIS, **watch for changes**





# Acute Ischemic Stroke

## Neurological Exam

- MS, CN, Motor, Coordination, Sensory and Gait need to be covered, however speed is of the essence!
- MCA: Contralateral : Hemiparesis, Hemianopia ( Vision change = opposite hemisphere, pupil = same hemisphere (motor)

Sensory loss

Ipsilateral (same side): Gaze preference/ looks at side of bleed

Aphasia (inability to speak)

Sensory deficits

# Conventional Angiogram

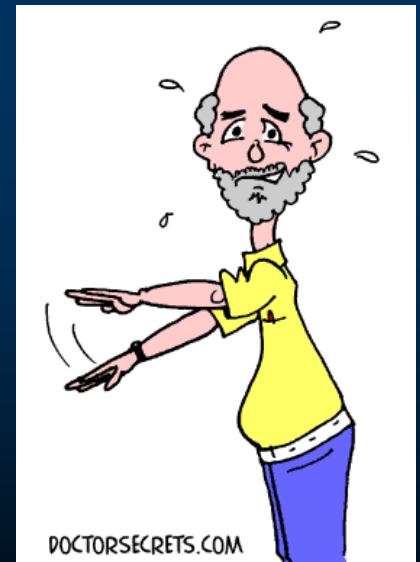


# Acute Ischemic Stroke

## Further Differentials

### Neurological Exam for Cerebral Accident

- ACA (acute): Disinhibition, primitive reflexes, contralateral **hemiparesis** (legs/arms), urinary incontinence.
- PCA (partial): **Contralateral hemianopsia** (hemy-an-op-see ya: hemi- half/ opsia- Optic view), cortical blindness, altered mental status, impaired memory.



# Hemianopsia



In this example the occlusion would be on the left and would be partial cerebral accident (PCA)

# “FAST” FINDINGS

- **F**ace/Feet- droop, sags, smiles
- **A**rms- motor unequal, sensory unequal
- **S**peech- altered, slurred, unrecognizable
- **T**ime- onset, GCS changes by one or multiple

# Right vs. Left brain hemisphere

- The right half (hemisphere) of the brain controls the movement of the left side of the body.
- The right half of the brain controls judging distance, size, speed, and position. This may cause a person with a right brain stroke to misjudge distances leading to falls. They have issues with short-term memory.
- The person may not be able to control the hand to pick up an object. Survivors of right-brain strokes often have problems making good decisions and become impulsive.
- Persons with right brain stroke are often unaware of the changes that have happened to them. They believe they can do the same tasks as they did before the stroke.

# Right vs. Left brain hemisphere

- **Left-Brain** Stroke effects the **right side** of the body
  - Trouble speaking or understanding words said or written Slow, careful movements.
  - Not able to see things on the right side of the body.
  - Facial weakness, unclear speech, or problems with swallowing
    - Cincinnati “smile” and “dog tricks”

VENTRICULAR INVOLVEMENT= motor and sensory





# Acute Ischemic Stroke

## Treatment

- ABCD' s
  - **Airway**: preparation for advanced airway management imminent for GCS <
  - **Breathing**: O<sub>2</sub> if hypoxic. Keep PCO<sub>2</sub> 32-36 mmHg
  - **Circulation**: Maintain adequate CPP (MAP-ICP). Treat HTN per protocols (with MC advise)
  - **D** = Dextrose/core temperature. Maintain normoglycemia (as hyperglycemia worsens neurological outcome)

# CO<sub>2</sub>

## and cerebral circulation

- **Increased CO<sub>2</sub>** from bradypnea or shallow hyperventilation-
  - CAUSES: Cerebral vasodilation
- **Decreased CO<sub>2</sub>** from over aggressive ventilation or hyperpnea:
  - CAUSES: Cerebral vasoconstriction

# Acute Ischemic Stroke

## Treatment

- Fever: Hyperthermia worsens ischemic injury
- Cerebral edema: Peaks 72-96 hours. Hyperventilation can decrease cerebral pressure by eliminating CO<sub>2</sub> and causing vasoconstriction.
- Seizure control: As needed, No prophylactic (vs. prophylaxis in the hemorrhagic stroke)

# Intracranial Hemorrhage

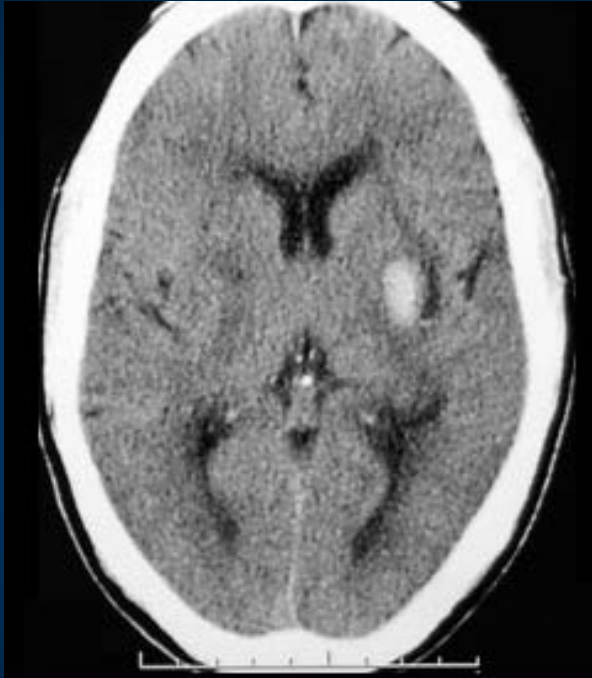
(non-traumatic)

Differential Diagnosis and Pathophysiology

# Intracranial Hemorrhage

## Intraventricular Hemorrhage

- Accounts for 3% of all non-traumatic ICH
- **Hypertension** is the most common etiology
- Often results from an intrahemisphere hemorrhage that extends into the ventricular system- effecting BOTH sides
- S/S: **Headache**, N/V, Progressive **deterioration of consciousness**, raised ICP, **Nuchal rigidity**



Intrahemisphere Hemorrhage

# Intracranial Hemorrhage

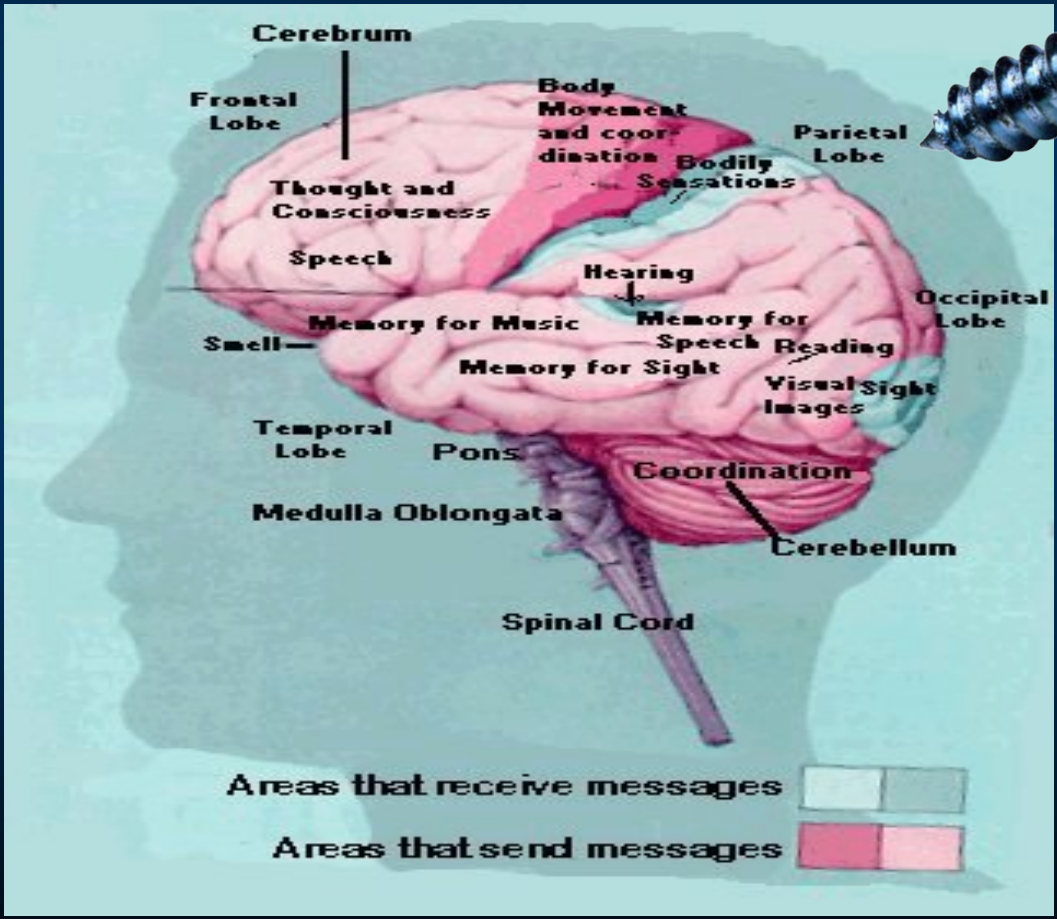
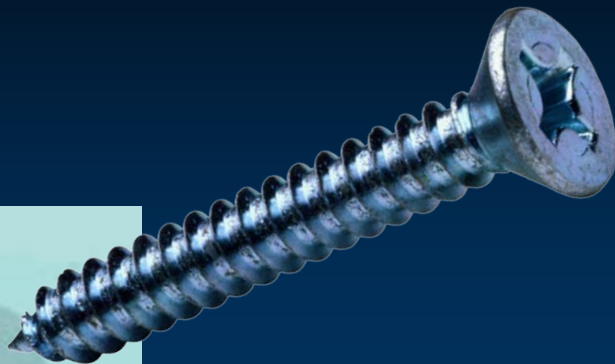
## Intraparenchymal Hemorrhage

- Pontine Hemorrhage
  - Abrupt onset of coma, pinpoint pupils, autonomic instability, horizontal gaze paralysis, and quadriparesis
  - The myopic pupils and depressed LOC may mimic opiate overdose

# Intracranial Hemorrhage

- Cerebellar Hemorrhage:
  - Sudden onset of vertigo, severe N/V, and ataxia leading to altered mental status and coma over a few hours
  - Edema contributes to brainstem herniation
  - Urgent posterior fossa decompression is essential for survival ??





# Hemorrhagic CVA

## Causes

- Hypertension is the #1 cause in adults
- Anticoagulation and Anti-Platelet Meds
- Sympathomimetic Drugs
- Aneurysms
- Brain Tumors
  - Metastatic (renal cell CA, malignant melanoma,

# Hemorrhagic CVA

## Treatment

- ABCD' s
  - Intubation, ETCO<sub>2</sub> on or about 30-33
  - Treat Hypertension per protocol (with MC advise)
- Fluid and Electrolyte Management
  - Use Normal Saline
- Prevent Hyperthermia
- **Seizure Prophylaxis +/-**
- Hyperventilate **only if brain stem herniation** is present (seizures, unequal pupils, sudden drop in GCS by 4 or >

# Hemorrhagic CVA

## (non-traumatic)

- Aneurysmal rupture accounts for 80% of cases
- Risk Factors
  - Advancing age, Smoking, HTN, Cocaine use, Hypertension, Heavy Alcohol use, Connective Tissue Disorders, Sickle Cell Disease, First Degree Relatives with Aneurysms
- Fatality rate is 50% within 2 weeks
- 15% of patients will have > 1 aneurysm
- 30% of survivors require lifelong care
- Outcome largely dependent on clinical presentation and CT findings
- Prehospital GCS is Key to Neurologist response in ED

# Hemorrhagic CVA

- Clinical presenting signs
  - Subarachnoid- Sudden-Onset  
“Thunderclap Headache”
  - “Worst Headache of my life” sudden onset
  - CN III palsy – pupil(s) non motor (aneurysm)
  - CN VI palsy- pons controlled, Facial Motor /Sensory (raised ICP)
  - Retinal Hemorrhages
  - Altered Mental Status
  - Nuchal Rigidity

# Hemorrhagic CVA

## Treatment

- Recognition
- Trending
- BP control as directed
- Airway management
- Seizure control...as this will eventually happen
- GCS “drops” accuracy

# Clinical Findings

- Sign and symptoms- AV / PU, %'s
- Allergies-
- Meds- HTN, Cholesterol, A-fib
- PMH- TIA's
- Last- events, med changes
- Events- day vs night/ early am

# Status Epilepticus

## Definitions

- A single seizure 10 minutes or longer  
and/or
- back-to-back seizures without return of consciousness



# Status Epilepticus

## Epidemiology

- 10% of all individuals with epilepsy will have at least one episode of SE in their lifetime
- 10% of patients experiencing a first unprovoked seizure will present in SE

# Status Epilepticus

## Acute Symptomatic Etiologies

- Vascular
  - Stroke (Hemorrhagic > Ischemic)
  - Subarachnoid Hemorrhage
  - Hypoxic Ischemic Swelling Cascade
- Toxic
  - Cocaine and other sympathomimetics
  - Alcohol withdrawal
  - Various Medications (Isoniazid, TCA's, various chemotherapy agents)

# Status Epilepticus

## Acute Symptomatic Etiologies

- Metabolic
  - Hyper or Hypo-Natremia
  - Hypoglycemia
  - Hypocalcemia
  - Liver or Renal failure
- Infectious
  - Meningoencephalitis
  - Brain Abscess
- Trauma

# Status Epilepticus

## Treatment

- ABCD's
  - Airway: Risk of aspiration, suction to bedside
  - Breathing: Give supplemental O<sub>2</sub>
  - C/V: Initial tachycardia giving way to hypotension (especially when Benzos or Barbiturates are given)
  - Dextrose: Symptomatic hypoglycemia is causing irreversible brain injury until corrected

# Status Epilepticus

- History
  - Fever, pre-existing epilepsy, trauma, baseline AED' s and their dosing
- Physical Exam
  - Signs of trauma, nuchal rigidity, end organ injury
  - Subtle signs of seizures (tachycardia, pupil nystagmus, irregular respirations)

Now you are better ready to go out there and confidently handle patients presenting with these various Neurological Emergencies!

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