



HEAD TRAUMA

TRAUMATIC BRAIN INJURY-EPIDEMIOLOGY

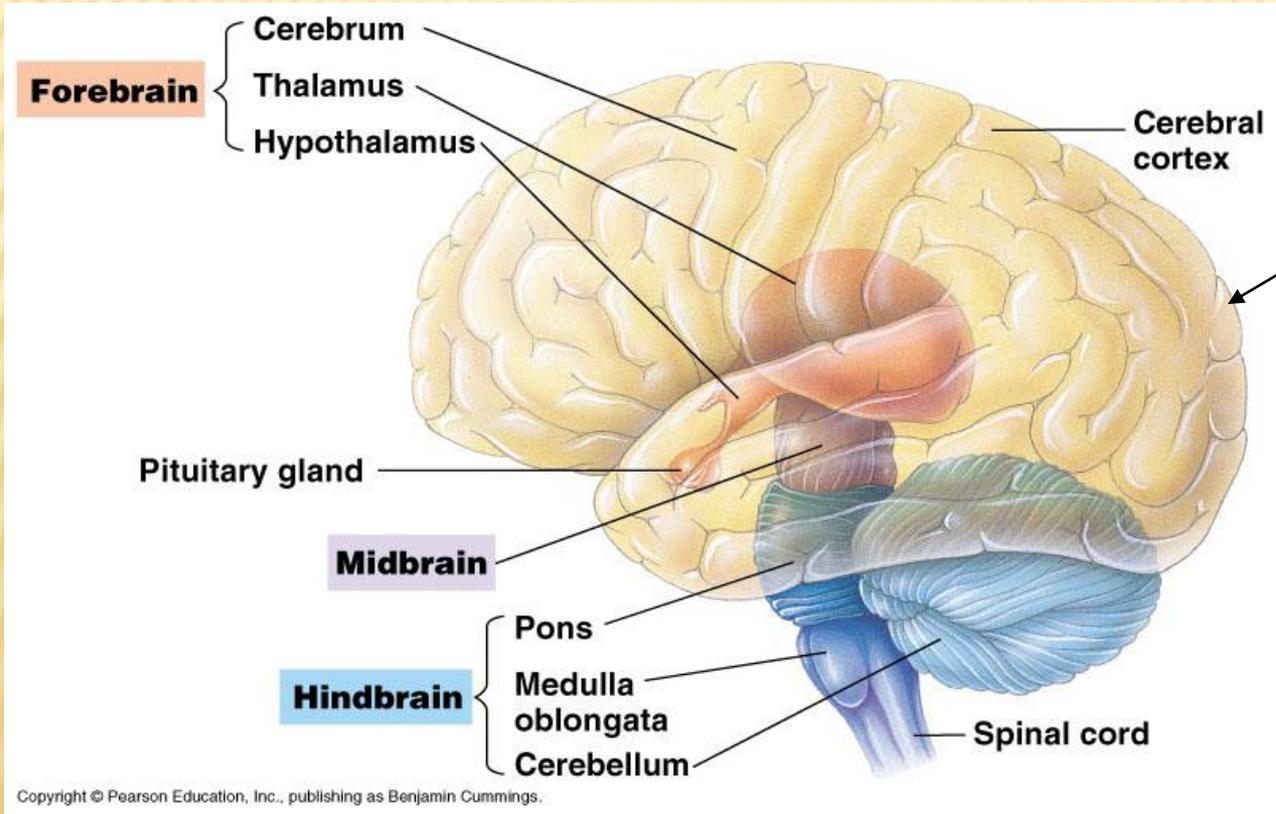
- ✘ US incidence 200/100,000 per year
- ✘ 1/7 dead on arrival
- ✘ 80% mild, 10% moderate, 10% severe
- ✘ Annual US mortality: 100,000 people
- ✘ Annual US cost: \$75-\$100 billion

HOW AND WHO?

- × MVA #1 cause
- × Falls
- × Violence
- × Sports
- × VICTIMS: young men (15-24 yrs)
 old men & women (>75 yrs)
 kids (<5 yrs)

THE HUMAN BRAIN

QuickTime™ and a TIFF (LZW) decompressor are needed to see this picture.

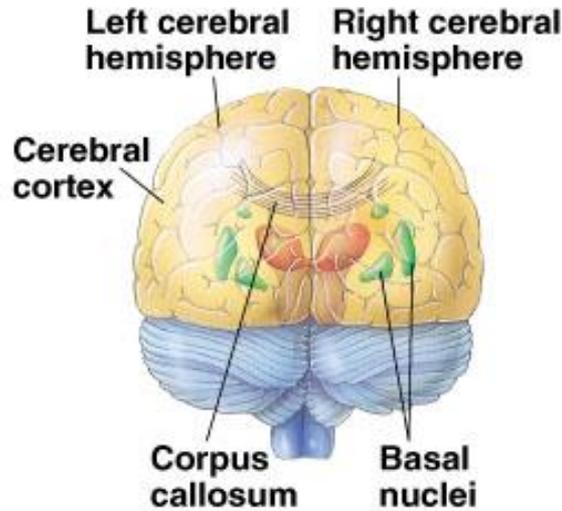


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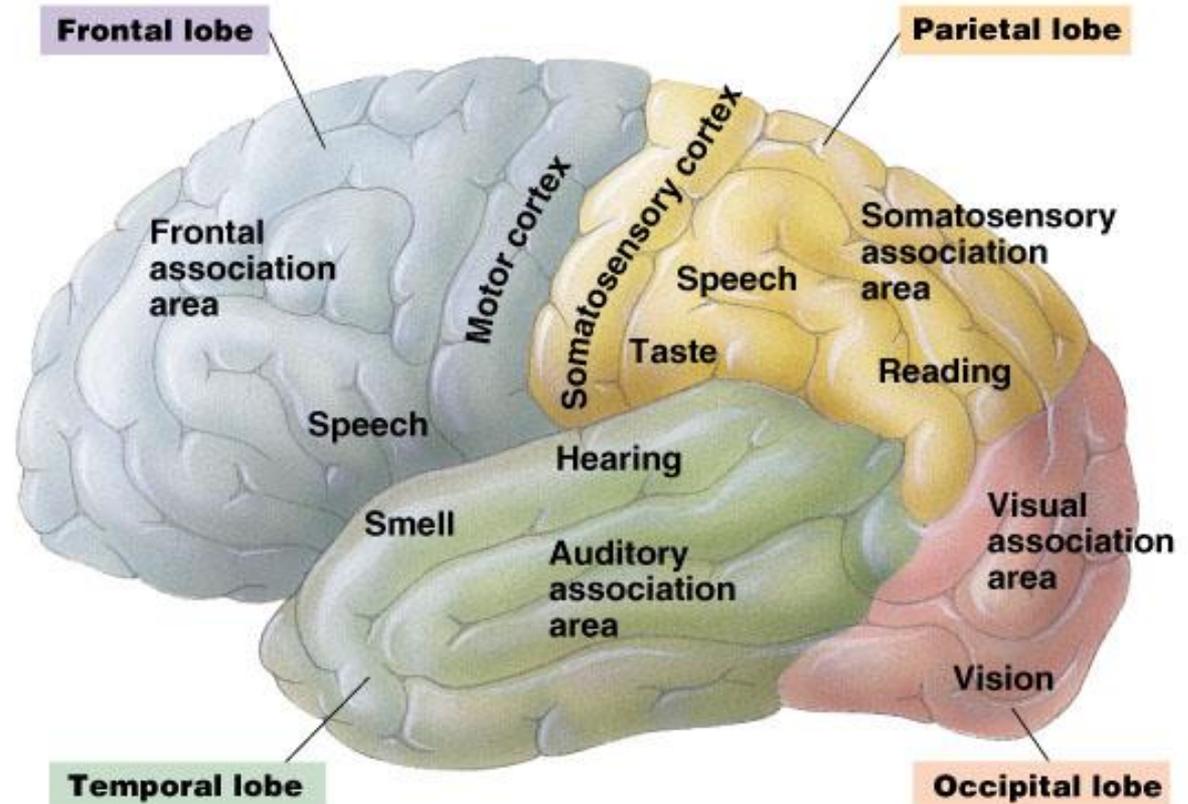
The Cerebrum has different areas for different functions

Four lobes: Frontal, Parietal, Temporal, Occipital

Left & right hemispheres: connected by the Corpus callosum



(a) Back of brain



(b) Left side of brain

Check out the Meningeal Layers in a REAL brain!

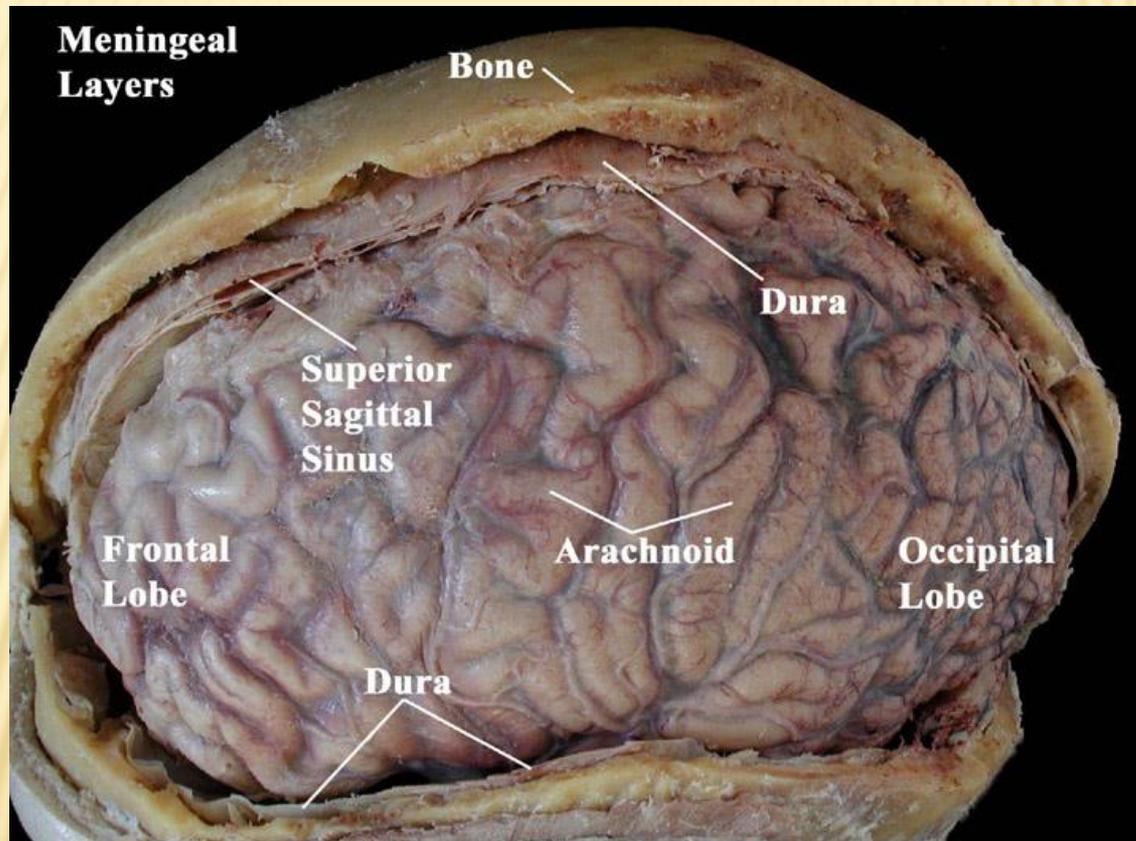
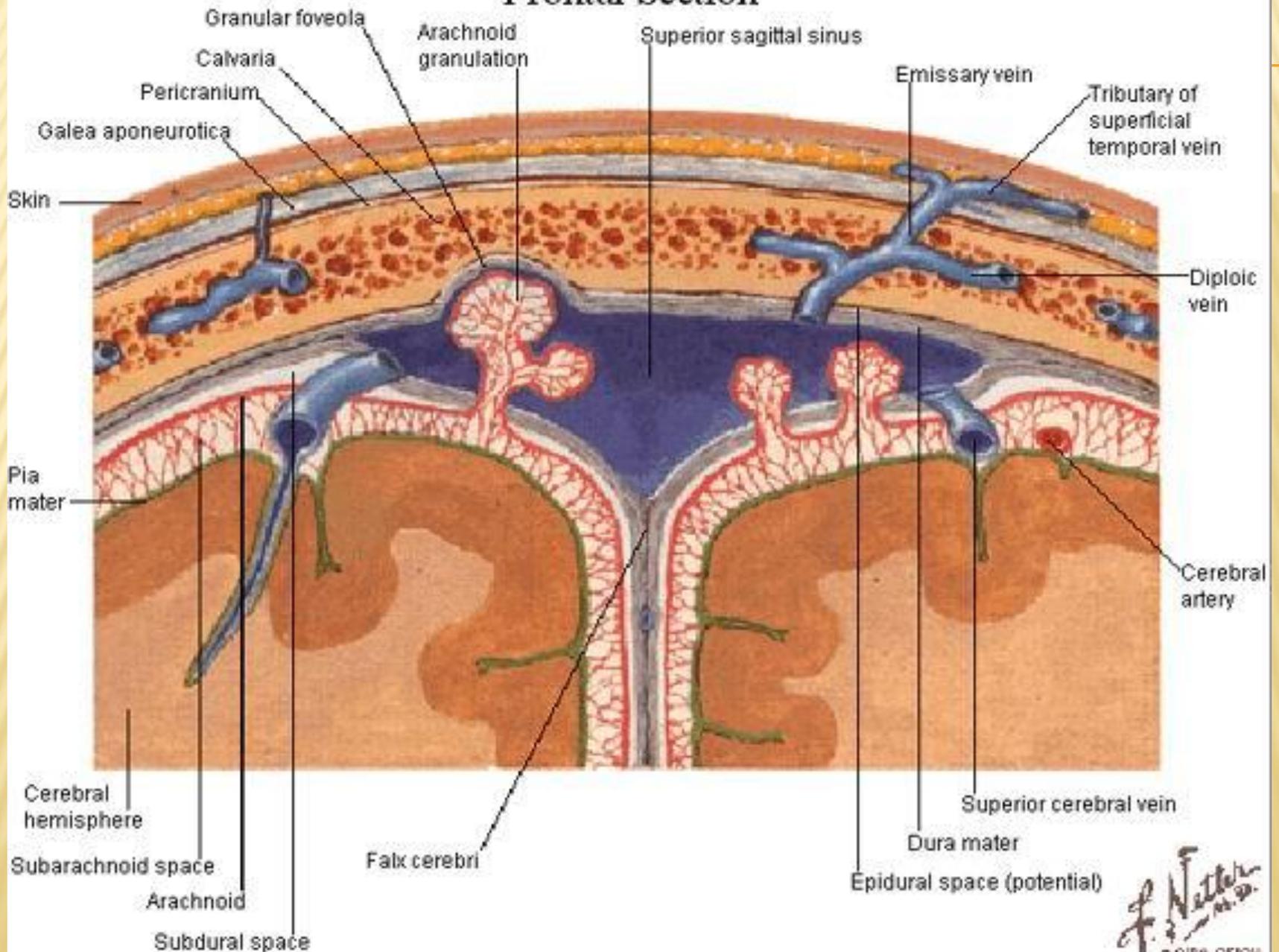


Image from

<http://faculty.une.edu/com/fwillard/Meninges/>

Meninges and Superficial Cerebral Veins

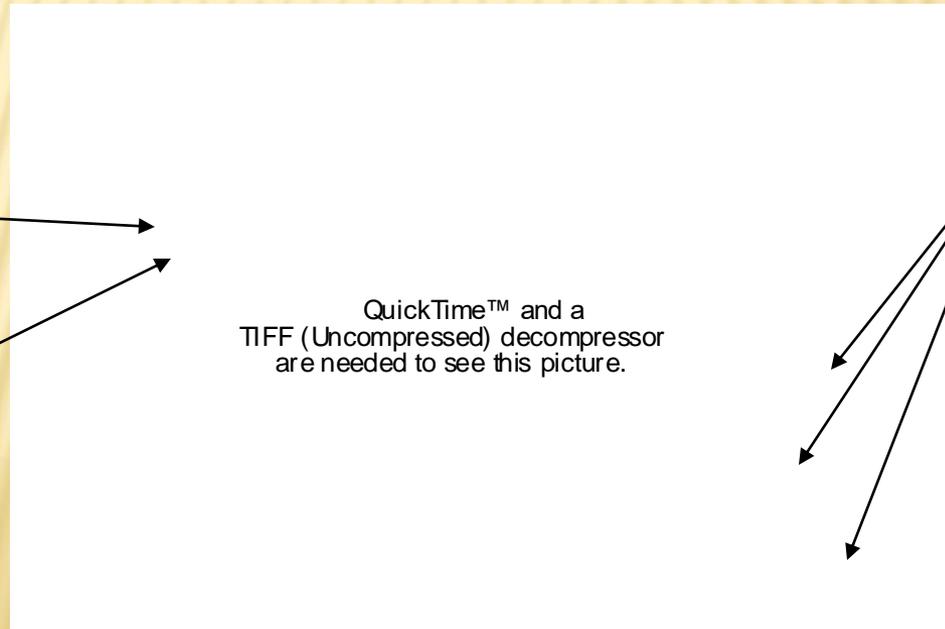
Frontal Section



THE BRAIN NEEDS LOTS OF TLC: PROTECTION AND NUTRITION

Brain Protection

1. Outer layer: scalp (skin)
2. Bony protection: Cranium (skull)



Ethmoid bone

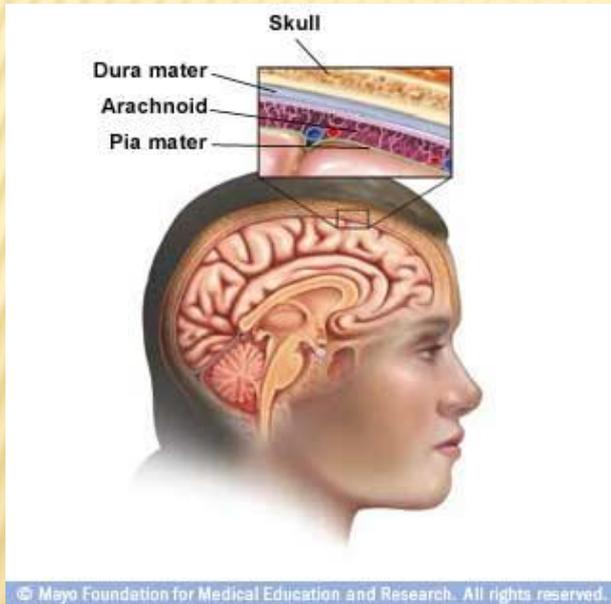
Sphenoid bone

Cranial sutures:

Areas where the bones have fused (early development) to form the cranium.

3. Specialized coverings called Meninges:

- × Dura Mater- outermost
- × Arachnoid Layer- middle
- × Pia Mater- innermost



**Here's a good way to remember
the order of the meninges (innermost
To outermost): "The meninges **PAD**
the brain." (Pia, Arachnoid, Dura)
www.washington.edu/chudler/meninges.html*

Foramen magnum:

Opening at base of
cranium (occipital bone)
where the medulla
oblongata extends, and
nerves & blood pass into
and out of the brain.

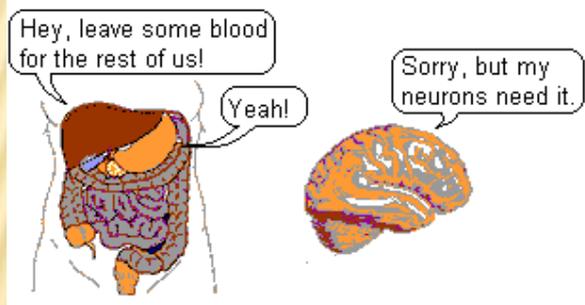
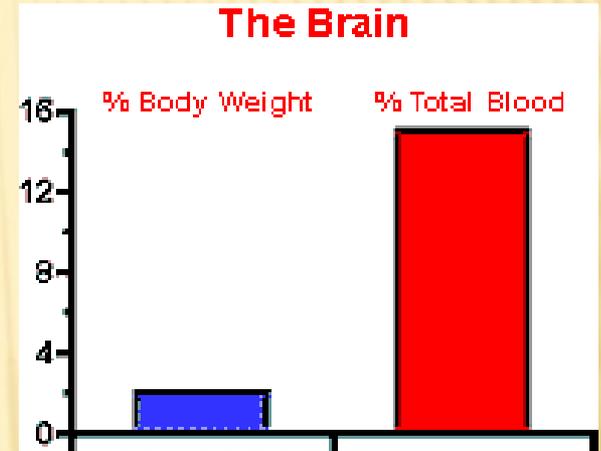
QuickTime™ and a
TIFF (Uncompressed) decompressor
are needed to see this picture.

Images from
<http://faculty.washington.edu/chudler/skul>



Brain Nutrition

- Deep and superficial blood vessels-> BRAIN
- Brain is 2% of the total body weight in humans
- Brain receives 15-20% of body's blood supply.

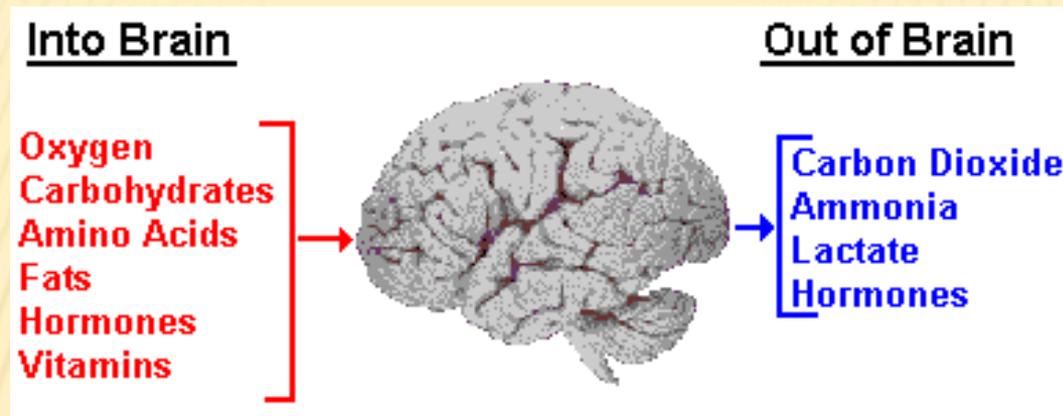


- Brain's blood supply stops = brain cell death
- Brain has top "blood priority" over all organs

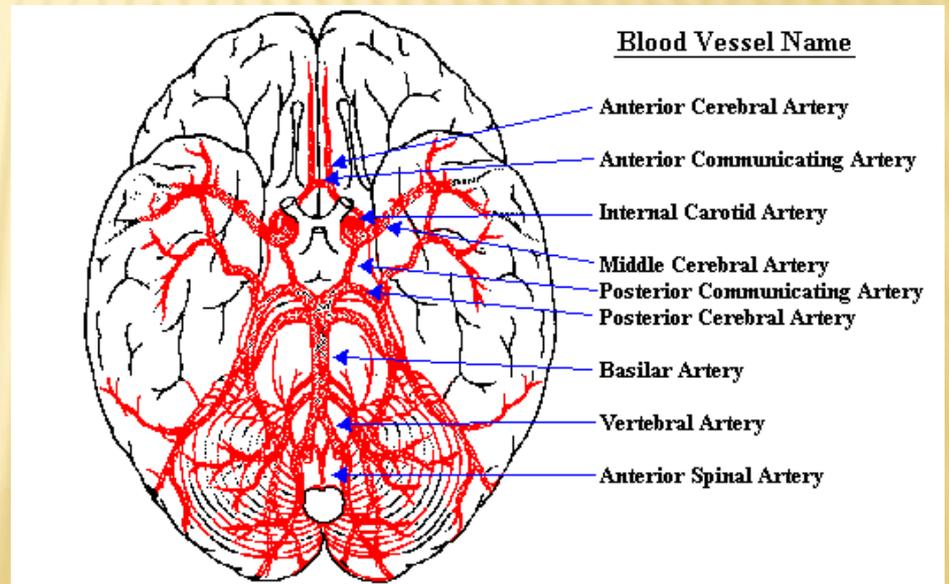
- Blood vessels reach the brain through the cranial foramina (labeled with **red arrows**).



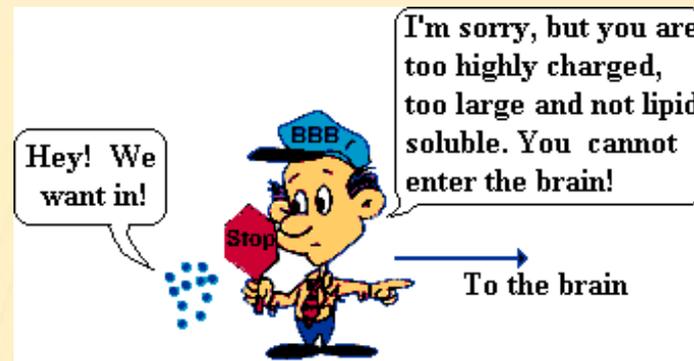
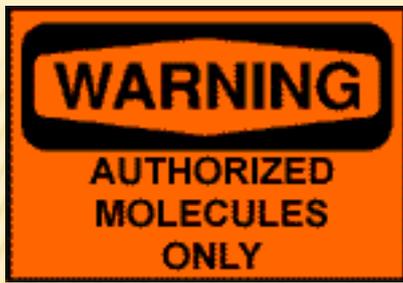
Brain Metabolism



An inferior view of the brain's blood vessels



What is a brain attack? For information, click [HERE](#).



Blood Brain Barrier (BBB)

- The brain is *selective* in what substances it allows in and out via blood
- Functions of the BBB:
 - Protects the brain from "foreign substances" in the blood
 - Protects the brain from hormones and neurotransmitters in the body
 - Maintains a constant environment for the brain.
- The BBB can break down under certain conditions:
 - hypertension, radiation, infection, and brain trauma, etc.

بِسْمِ اللَّهِ الرَّحْمَنِ الرَّحِيمِ

SKULL FRACTURE

- ✘ Linear, stellate , *comminuted* fracture
- ✘ Basillar fracture
- ✘ Depressed fracture
- ✘ Closed, open fracture

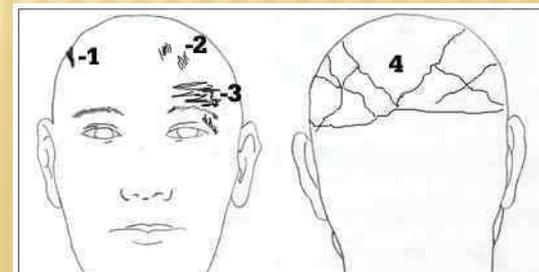
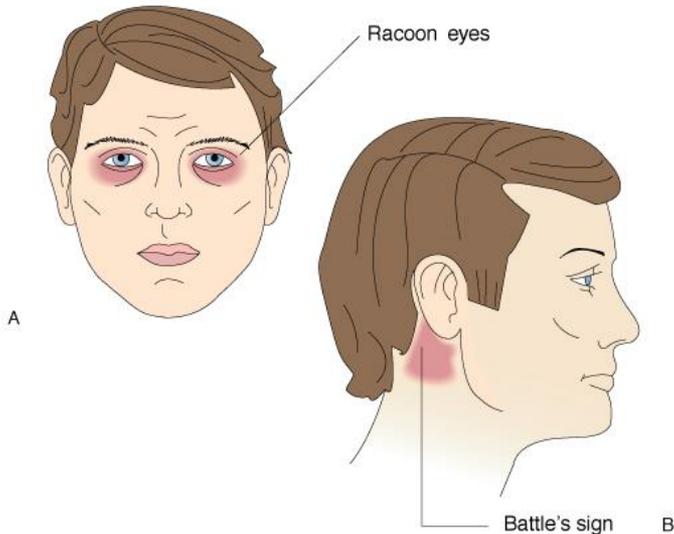
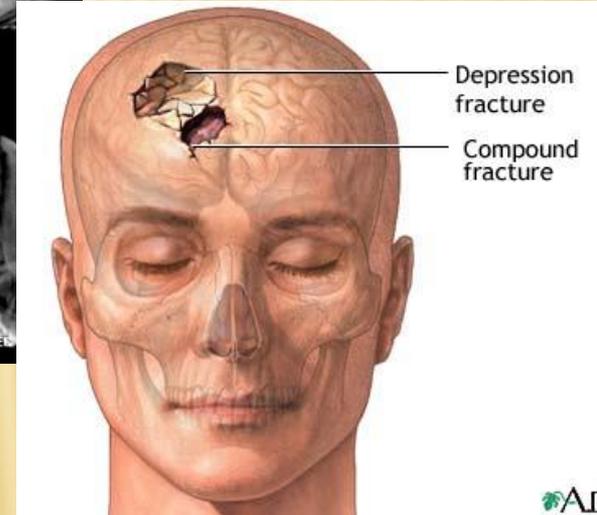
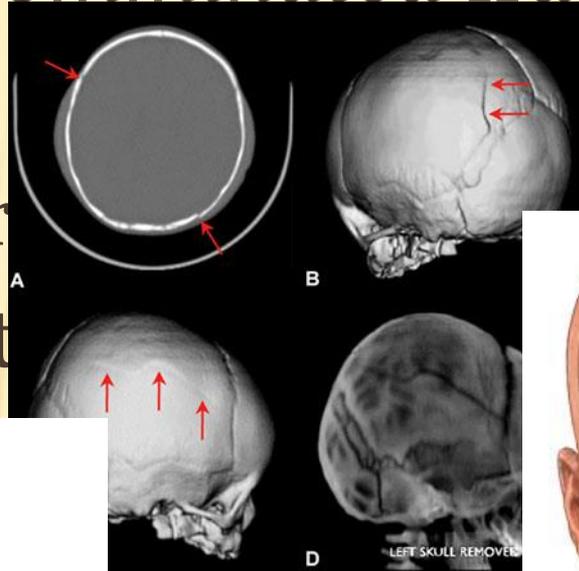


Figure 41-4 (A) Periorbital ecchymosis, called racoon eyes, and (B) periauricular ecchymosis, called

TYPES OF SKULL FRACTURE

× Depressed

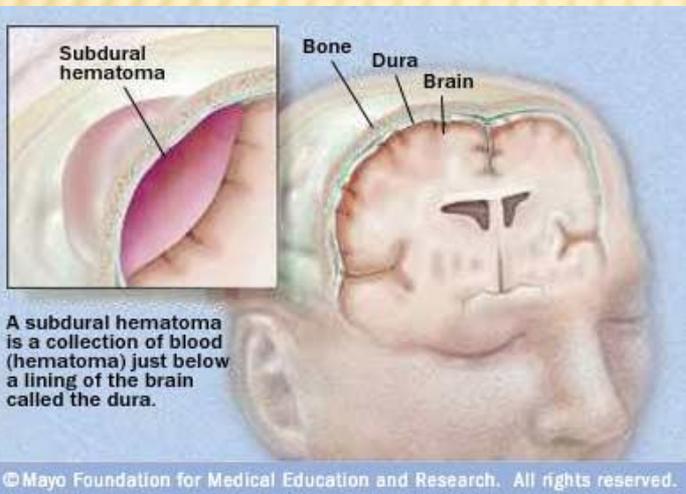
- + Bone fragments pressed inward
- + Places pressure on brain
- + Brain tissue may be exposed through injury

× Basilar

- + Fractures in floor of skull
- + Diagnosis made clinically
- + Signs and symptoms
 - × Periorbital ecchymosis (Raccoon eyes)
 - × Battle's sign
 - × CSF drainage from nose, ears

SOME RELATED CONDITIONS

- ✗ **Contusion and Edema**
- ✗ **Skull Fracture**
- ✗ **Intracranial Hematoma**
 - + Subdural or epidural
 - + A blood vessel ruptures
 - + collection of blood compresses brain tissue.

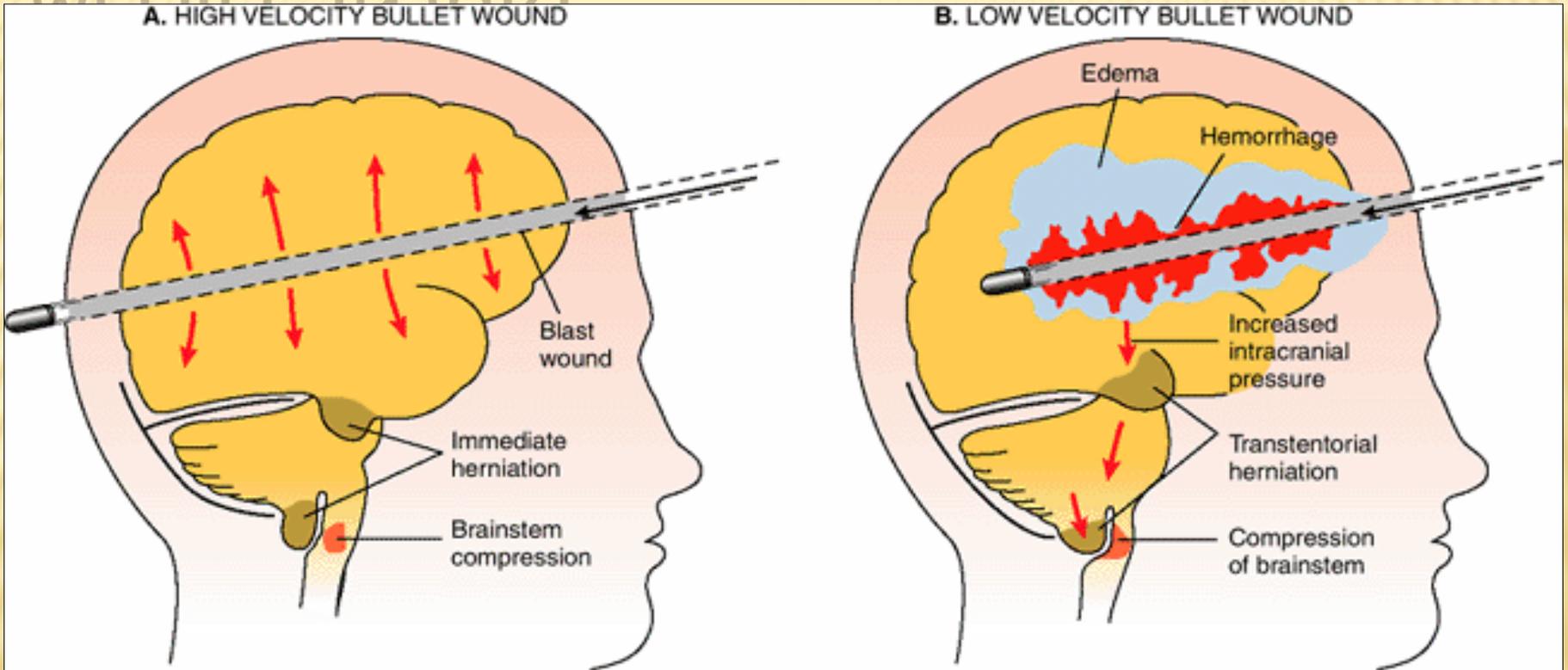


Left: Arrows indicate an epidural hematoma, a collection of blood between the skull and the outer covering of the brain, which is compressing the right frontal lobe.

Right: Arrows highlights tumors in both sides of the brain.

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MISSILE INJURY:



The “blast effect” of a high-velocity projectile causes an immediate increase in supratentorial pressure and results in death because of impaction of the cerebellum and medulla into the foramen magnum. A low-velocity projectile increases the pressure at a more gradual rate through hemorrhage and edema.



NON-MISSILE / BLUNT HEAD INJURY:

✘ Primary Injury:

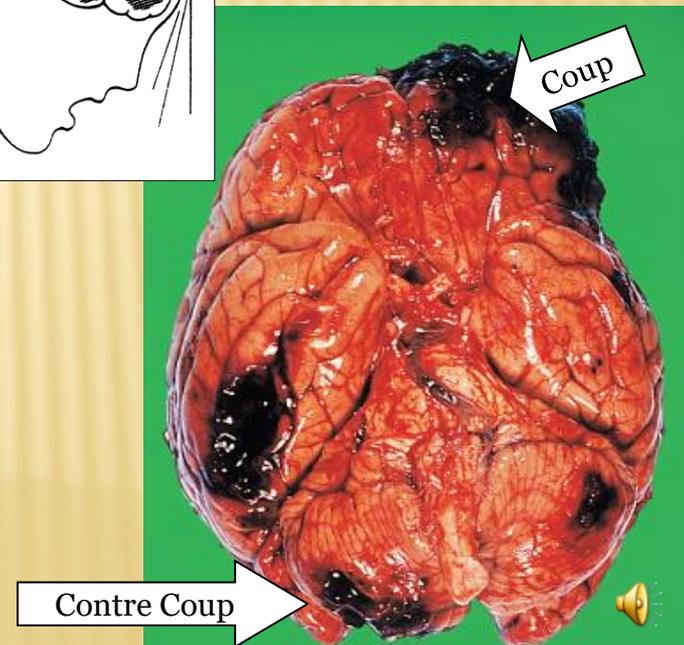
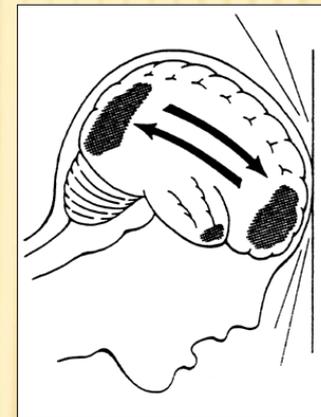
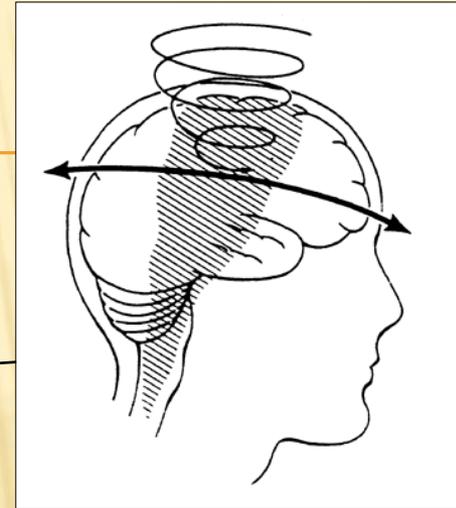
- + Coup & Contra-Coup
- + Focal damage-concussion, contusion,
- + Diffuse axonal injury:

✘ Secondary Injury:

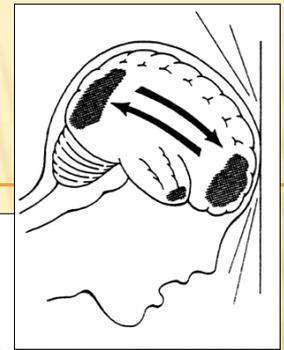
- + Concussion
- + Epidural/subdural Hematoma
- + Oedema
- + Infection

✘ Post Traumatic Complications:

- + Epilepsy
- + Dementia
- + Vegetative state – Coma.



WHIPLASH INJURY:



Closed Head Injury

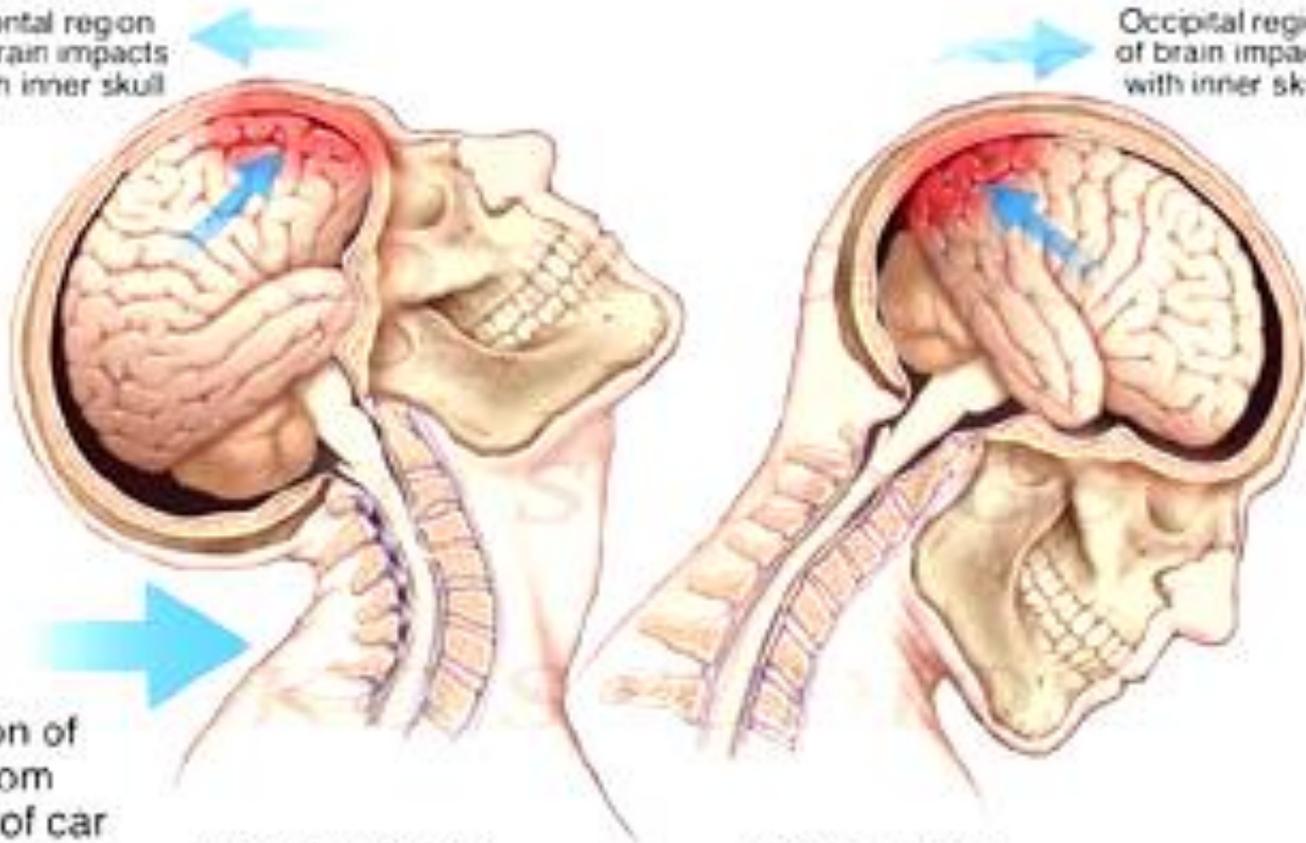
Frontal region of brain impacts with inner skull

Occipital region of brain impacts with inner skull

Direction of force from impact of car

Hyperextension of Head and Neck

Hypertexion of Head and Neck



+

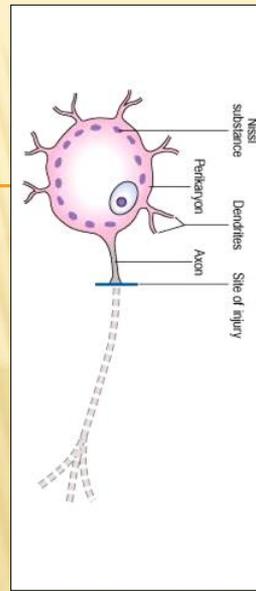
PATHOGENESIS OF INJURY:

Focal damage

Contusion – Local injury + haemorrhage.

Healing by gliosis – gross yellow-brown due to hemosiderin.

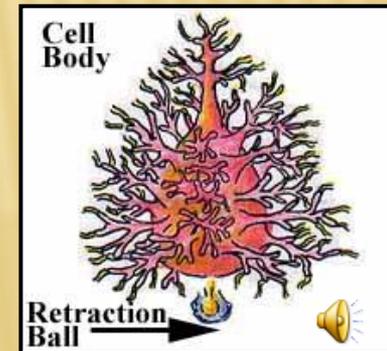
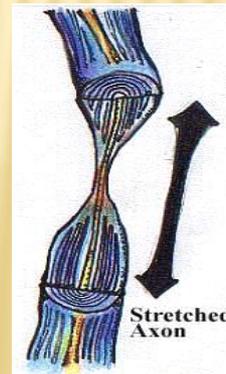
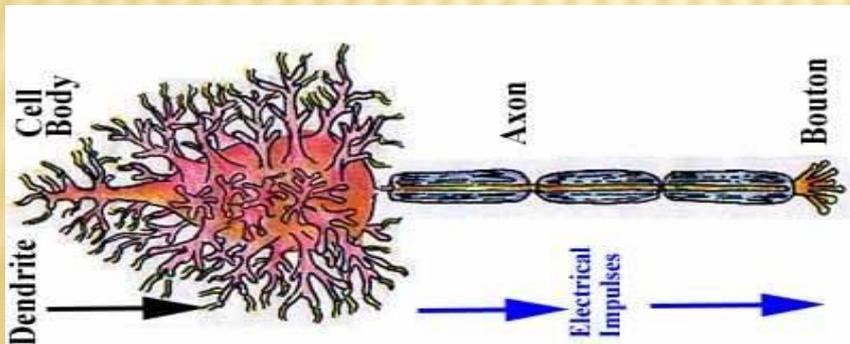
Diffuse axonal injury



×

Shearing of neurons. Two main components exist:

- + small haemorrhagic lesions - in the corpus callosum and dorsolateral quadrant of the brainstem;
- + diffuse damage to axons – Microscopic - axonal retraction balls & anterograde degeneration.



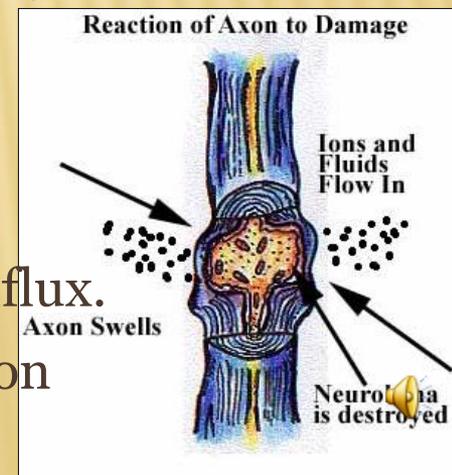
MECHANISMS OF DIFFUSE INJURY:

✘ Excitatory amino acids (EAA)

- + Elevation of glutamate and aspartate after Traumatic Brain Injury
- + EAAs can cause cell swelling, vacuolization, and neuronal death. Through influx of chloride and sodium, calcium. Decrease ATP or increase free radical production.

✘ Endogenous opioids & Other alterations:

- + behavioral suppression - Activation of muscarinic cholinergic systems in pons
- + Decreased glucose utilization → further brain injury - increased catecholamines
- + Extracellular potassium → leading to edema
- + Increased cytokines → Inflammation
- + Decreased intracellular magnesium → calcium influx.
- + Increased protein kinases → astrocyte proliferation



INTRACRANIAL HEMATOMA: PATHOLOGICAL MECHANISMS & CLINICAL MANIFESTATIONS

Site	Mechanism	Clinical manifestations
Epidural /Extradural	Skull fracture with arterial rupture, e.g. middle meningeal artery	Lucid interval followed by a rapid increase in intracranial pressure
Subdural space	Rupture of venous sinuses or small bridging veins due to torsion forces	Acute presentation with a rapid increase in intracranial pressure Chronic presentation with personality change, memory loss and confusion, particularly in the elderly
Subarachnoid space	Arterial rupture	Meningeal irritation with a rapid increase in intracranial pressure
Cerebral hemisphere	Cortical contusions Rupture of small intrinsic vessels with intracerebral haematoma 'Burst lobe' with intracerebral haematoma contusions and subdural haematoma	No special features Increased intracranial pressure with focal deficits; usually fatal Profound coma , usually rapidly fatal



EPIDURAL HEMATOMA

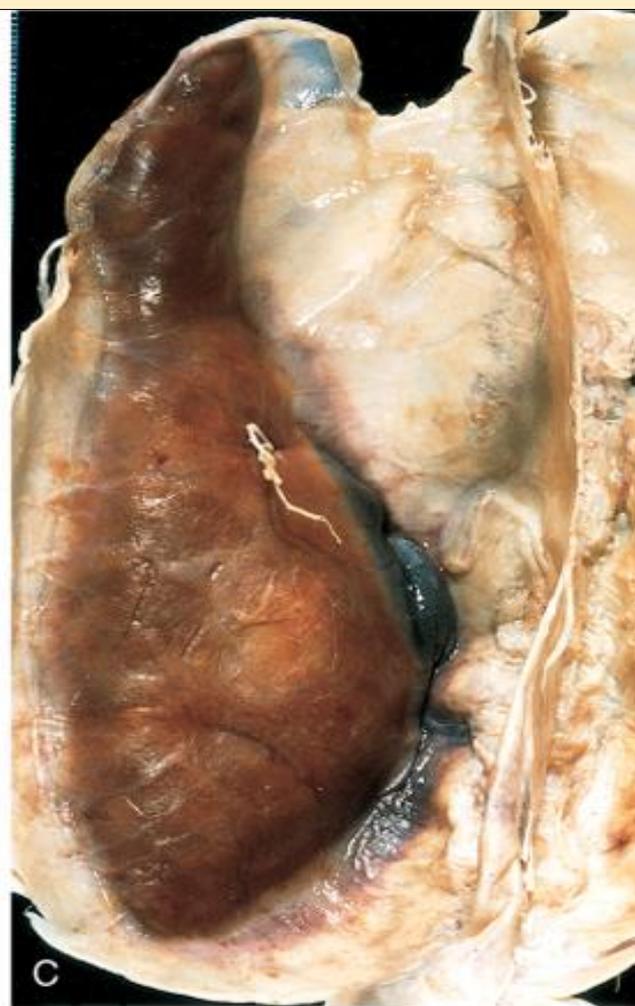
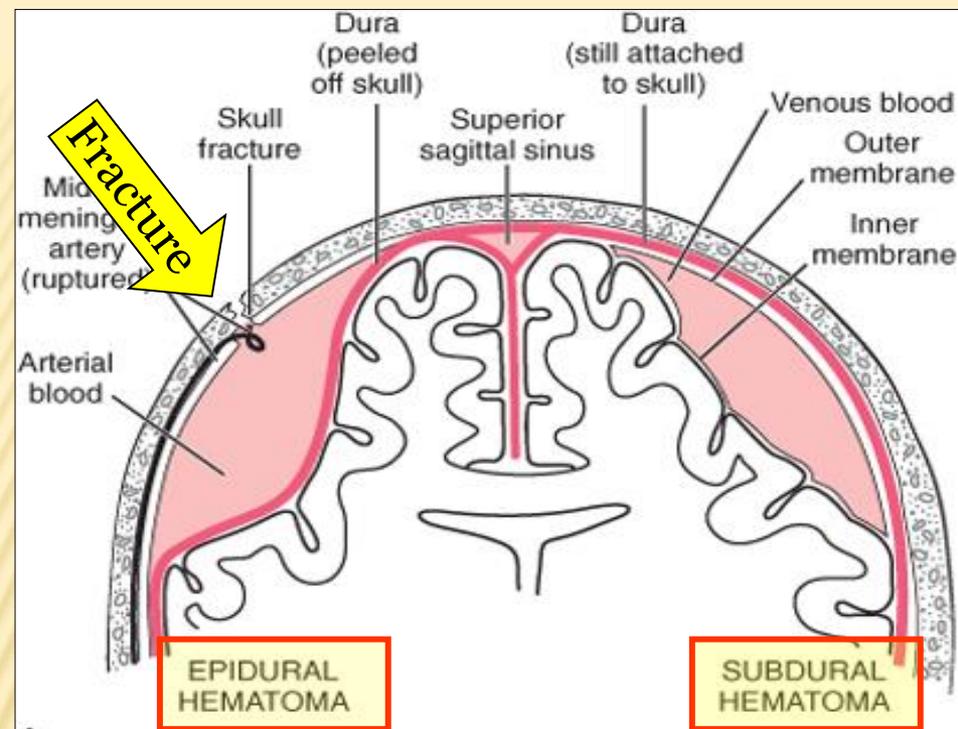
- ✘ Usually associated with skull fracture in temporal area
- ✘ Fracture damages artery on skull's inside
- ✘ Blood collects in epidural space between skull and dura mater
- ✘ Since skull is closed box, intracranial pressure rises

EPIDURAL HEMATOMA

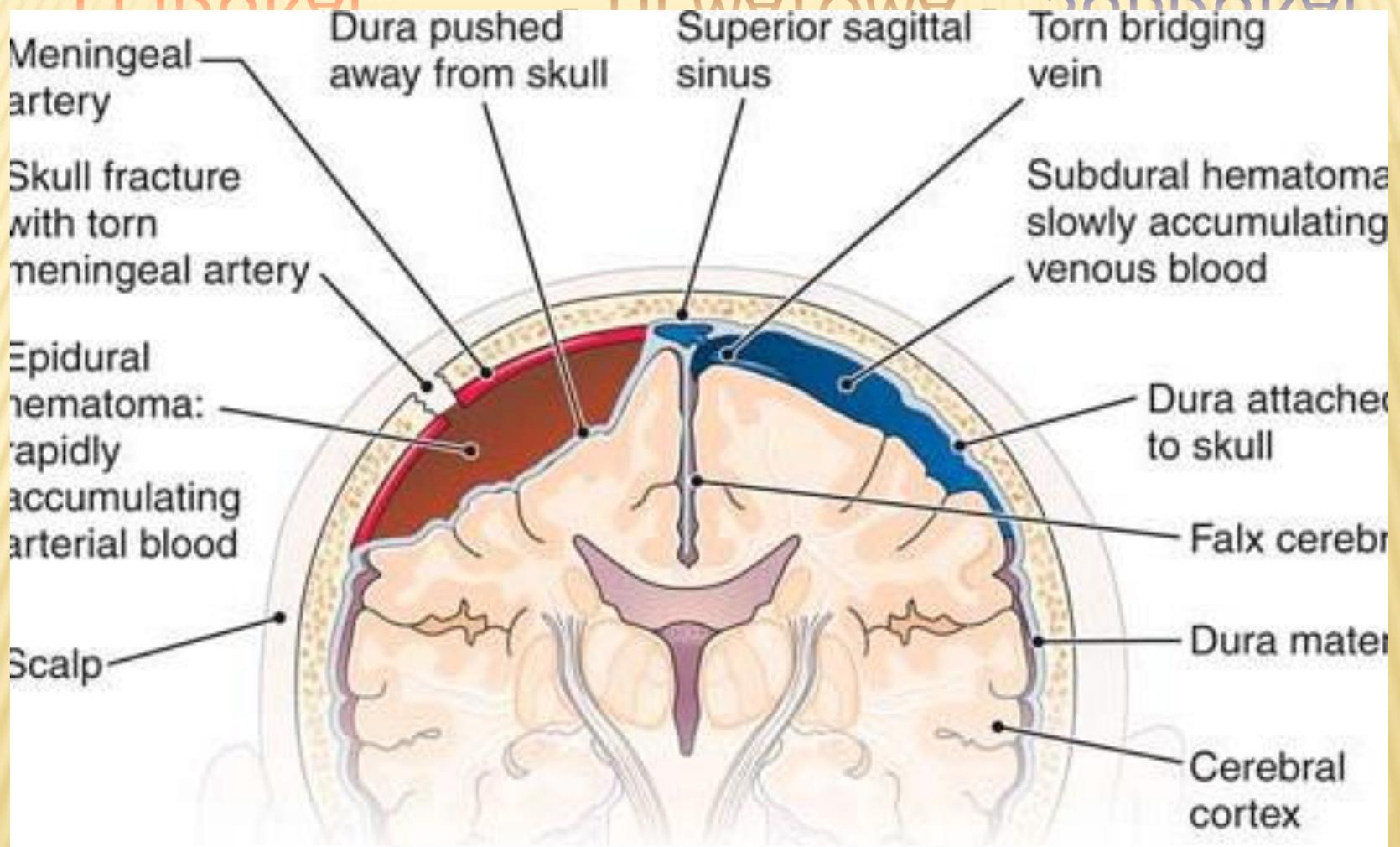
✘ Signs and Symptoms

- + Loss of consciousness followed by return of consciousness (lucid interval)
- + Headache
- + Deterioration of consciousness
- + Dilated pupil on side of injury
- + Weakness, paralysis on side of body opposite injury
- + Seizures

EPIDURAL HEMATOMA

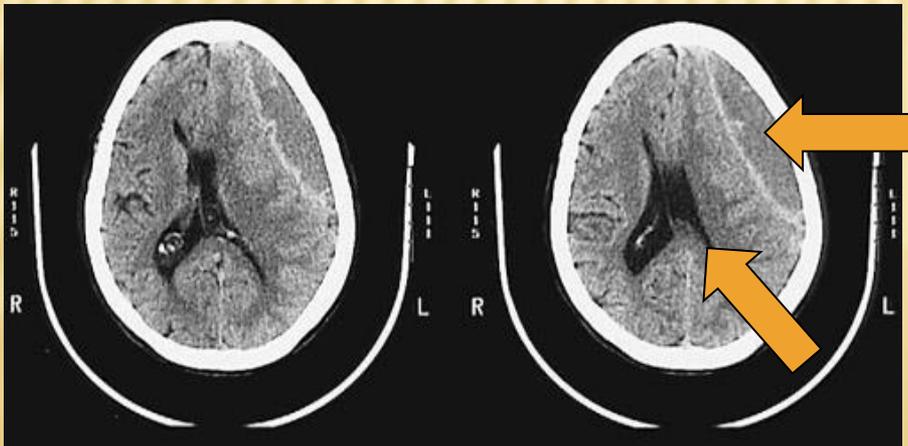
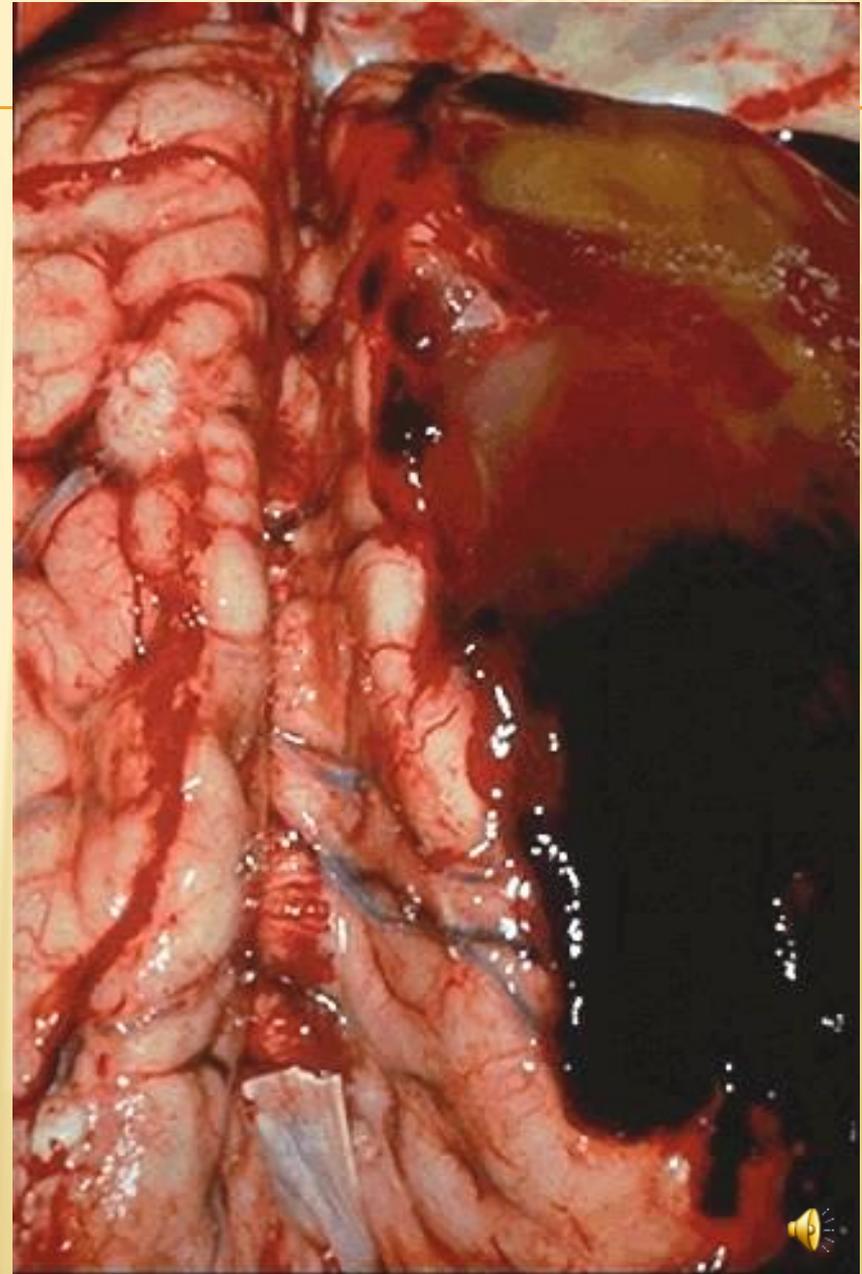


EPIDURAL - HEMATOMA - SUBDURAL

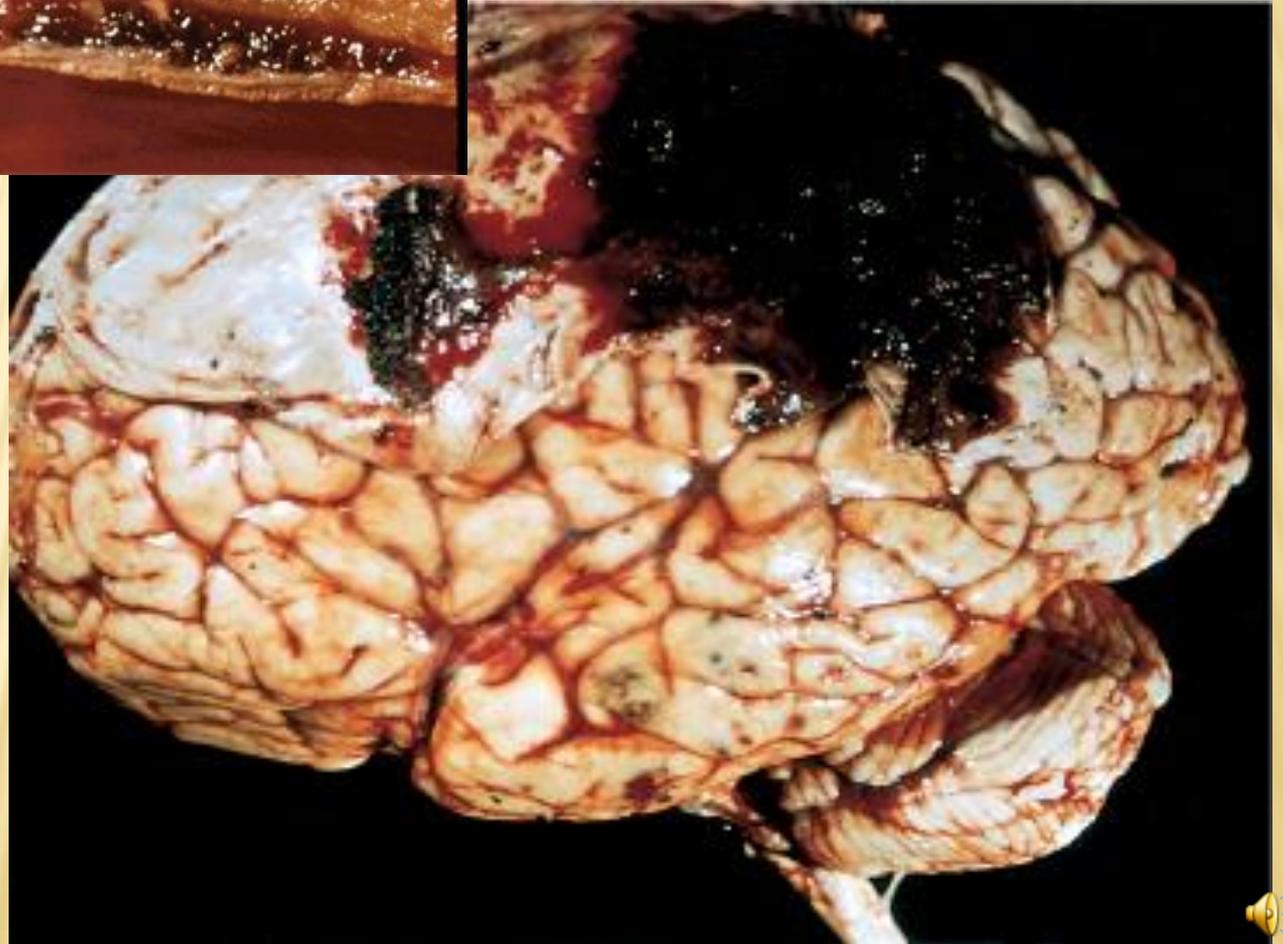


EPIDURAL HEMATOMA

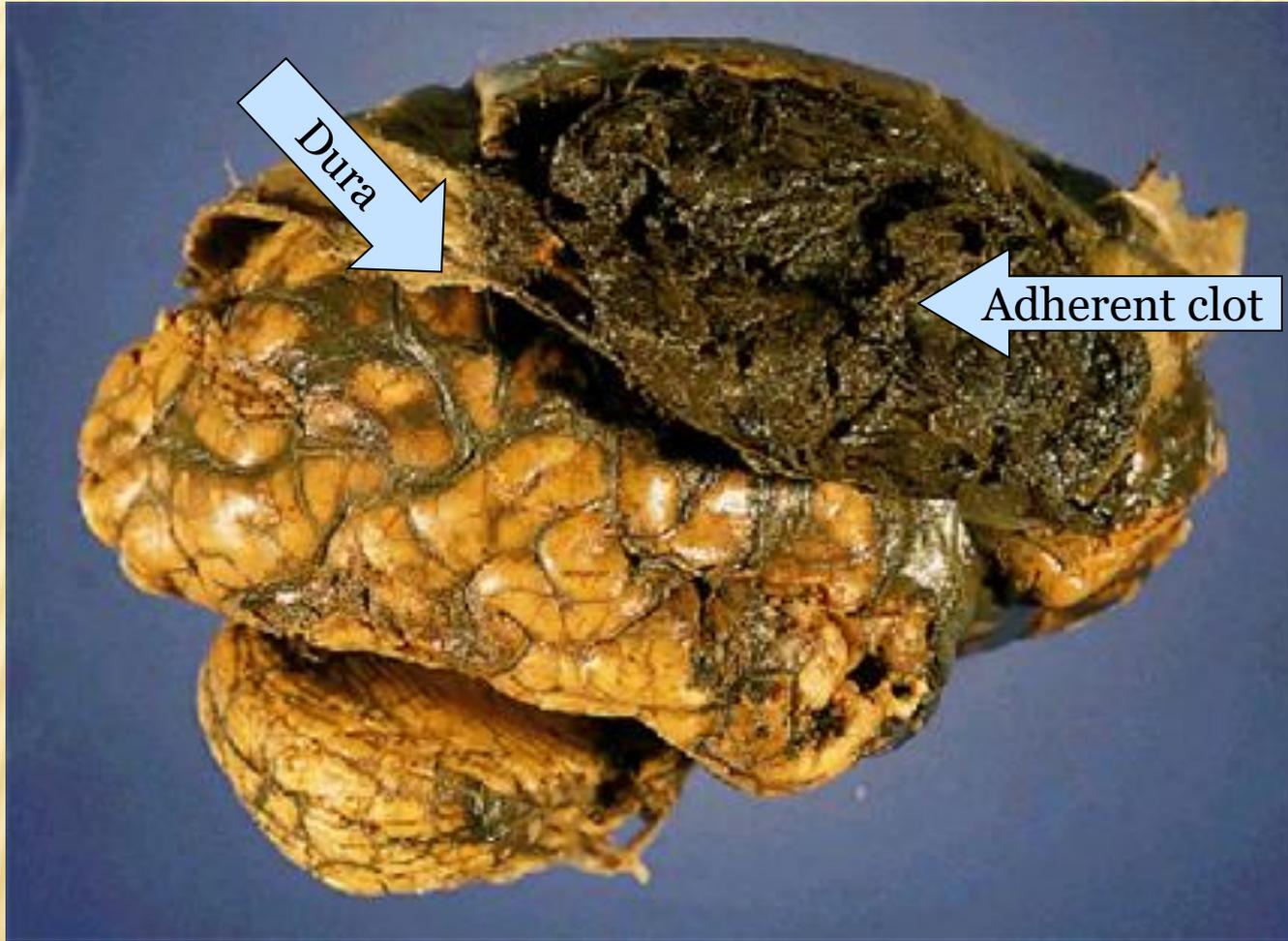
The dura has been reflected back (with a small portion visible at the lower right) to reveal a subdural hematoma. Such a blood clot is usually the result of trauma with tearing of the bridging veins.



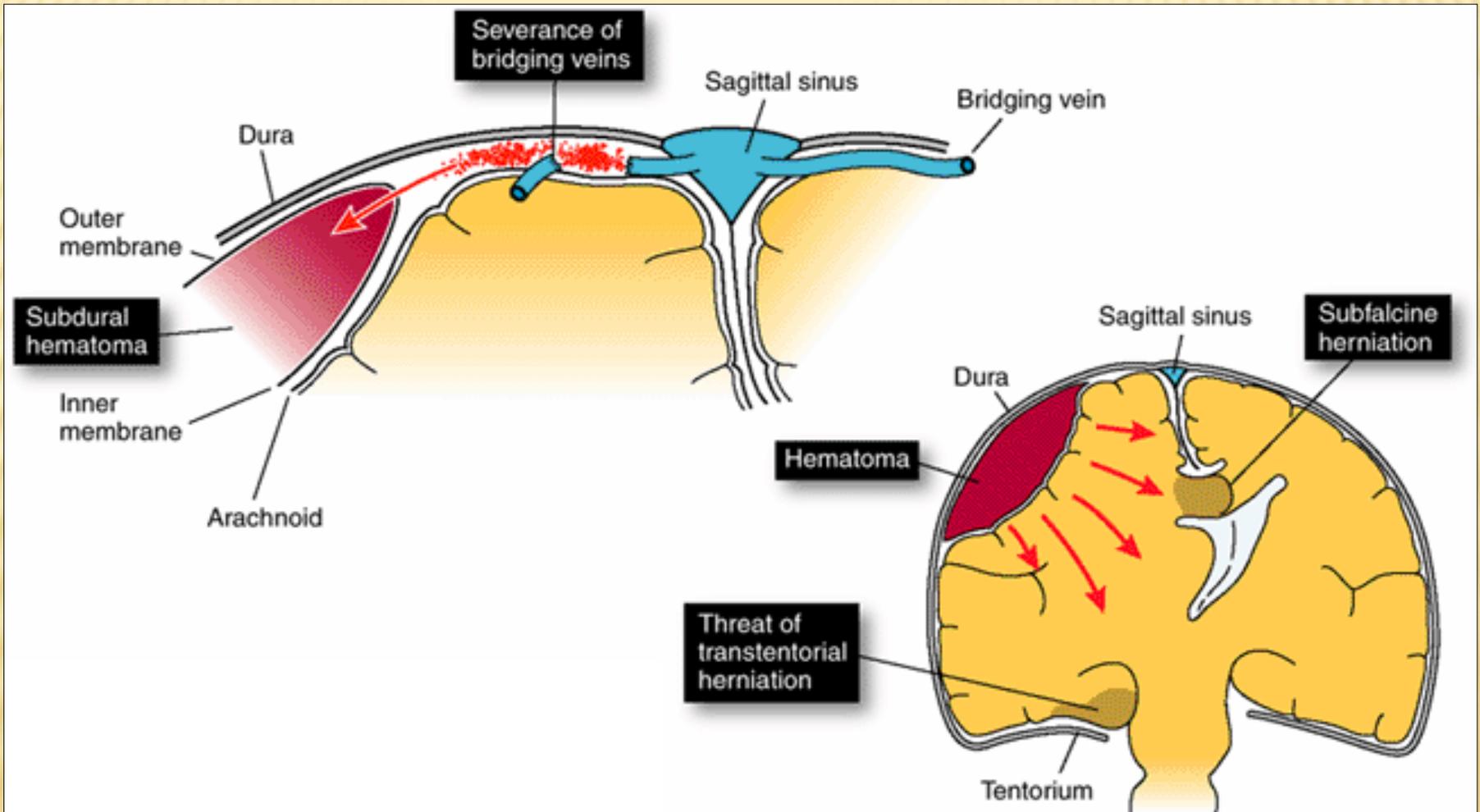
EPIDURAL HEMATOMA - SKULL FRACTURE.



EPIDURAL HEMORRHAGE



SUBDURAL HEMATOMA:



With head trauma, the dura moves with the skull, and the arachnoid moves with the cerebrum. As a result, the bridging veins are sheared causing hematoma in the expansile subdural space.

SUBDURAL HEMATOMA

- ✘ Usually results from tearing of large veins between dura mater and arachnoid
- ✘ Blood accumulates more slowly than in epidural hematoma
- ✘ Signs and symptoms may not develop for days to weeks

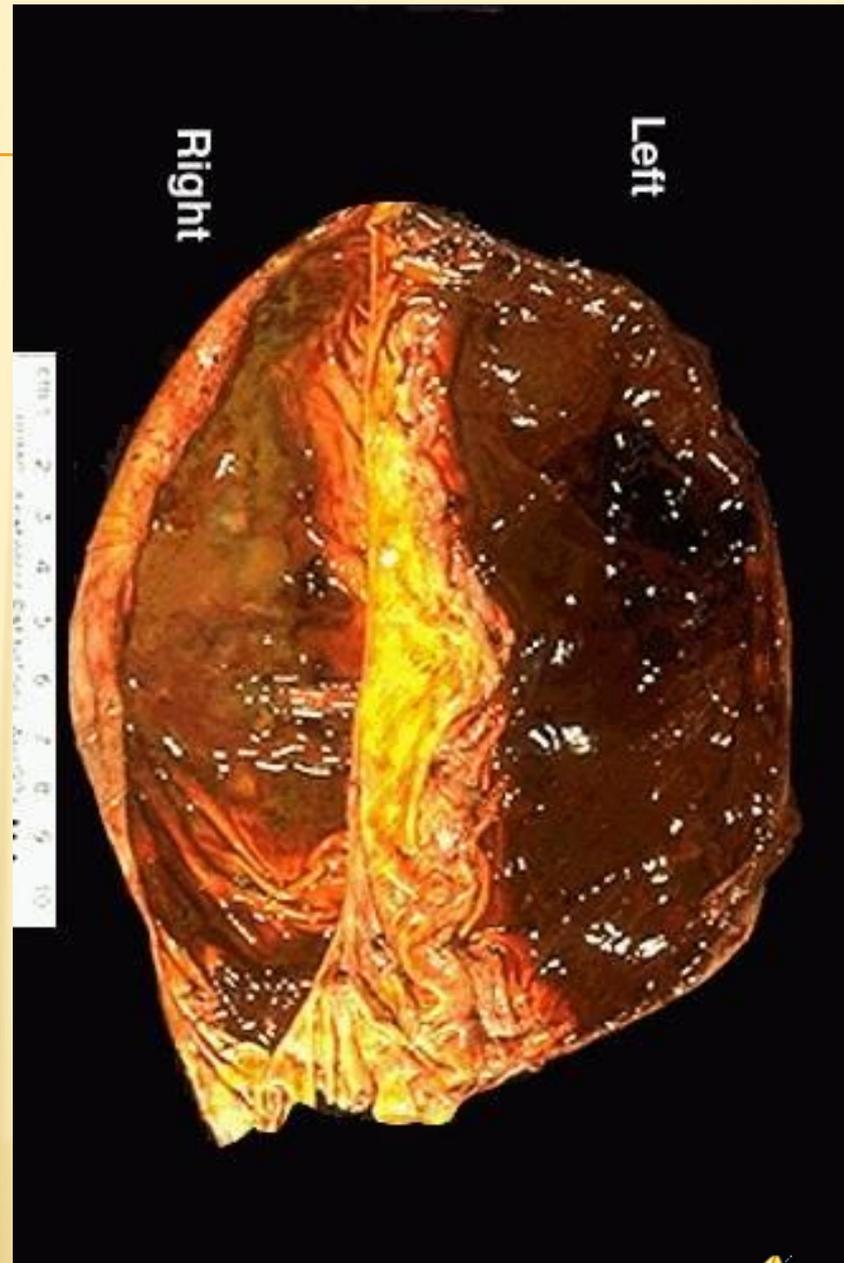
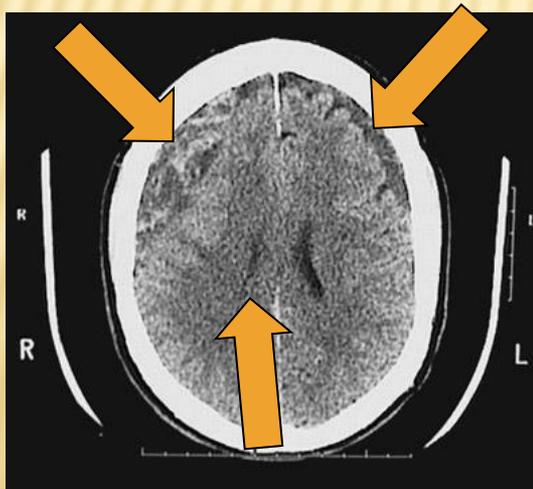
SUBDURAL HEMATOMA

- ✘ Signs and Symptoms
 - + Deterioration of consciousness
 - + Dilated pupil on side of injury
 - + Weakness, paralysis on side of body opposite injury
 - + Seizures

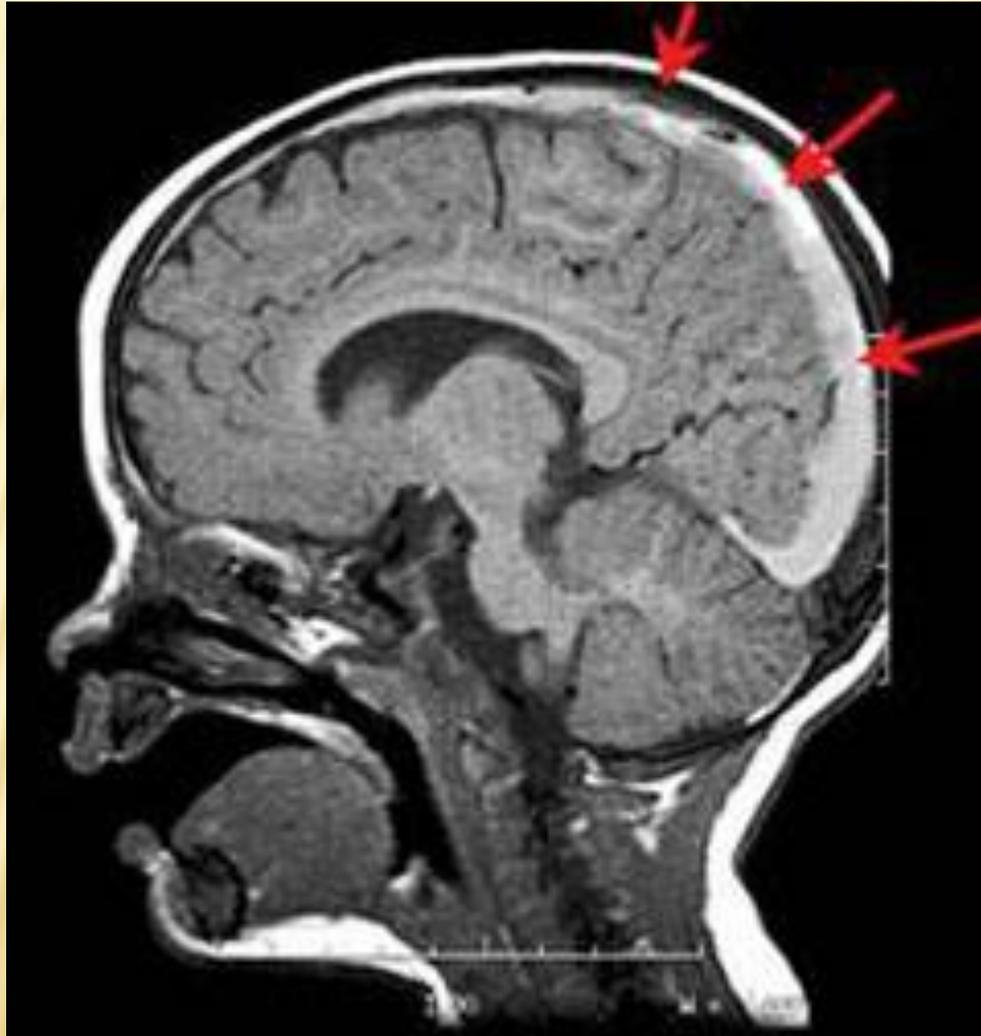
Because of slow or delayed onset, may be mistaken for stroke

SUBDURAL HEMATOMA

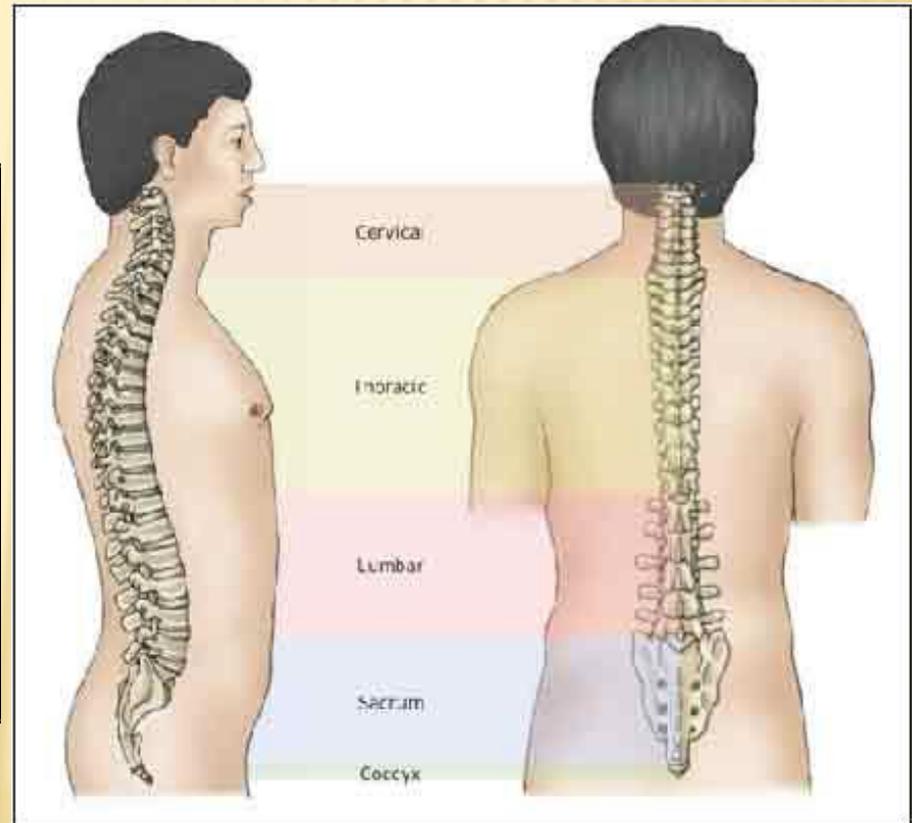
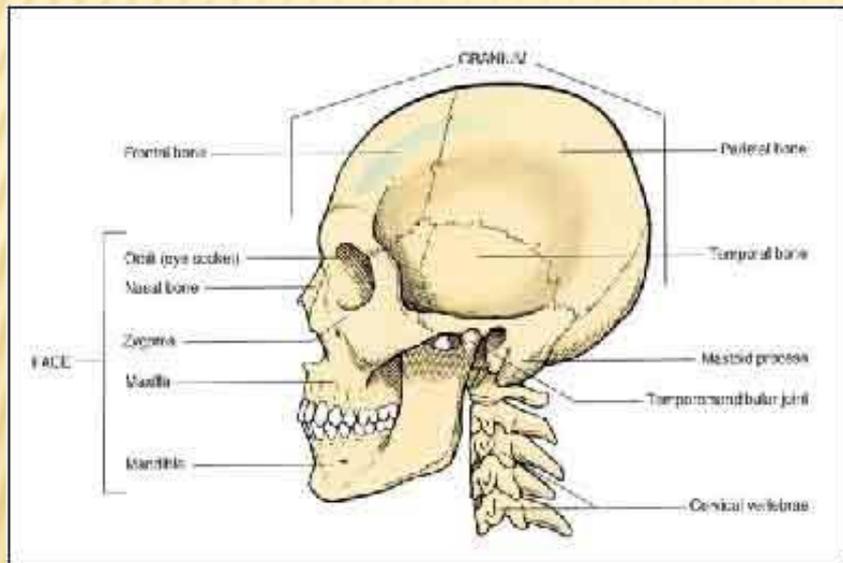
bilateral chronic subdural hematoma (brown). Since the bleeding is venous, subdurals can form more slowly and insidiously than arterial hemorrhages. Subdurals are most common in the very young and the elderly.



SUBDURAL HEMATOMA



ANATOMY AND PHYSIOLOGY OF THE SKELETAL SYSTEM



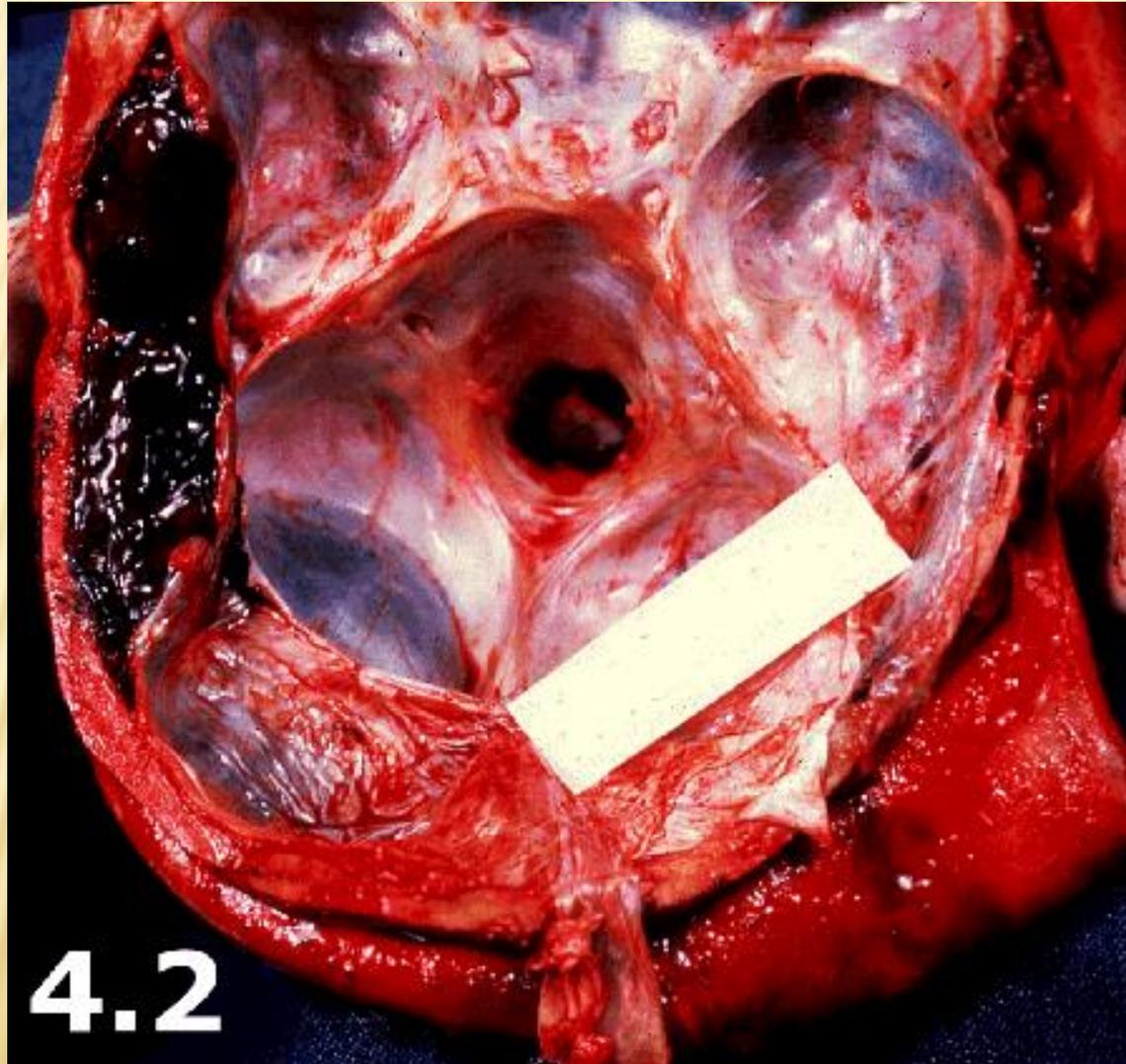
BATTLE'S SIGN



RACCOON EYES

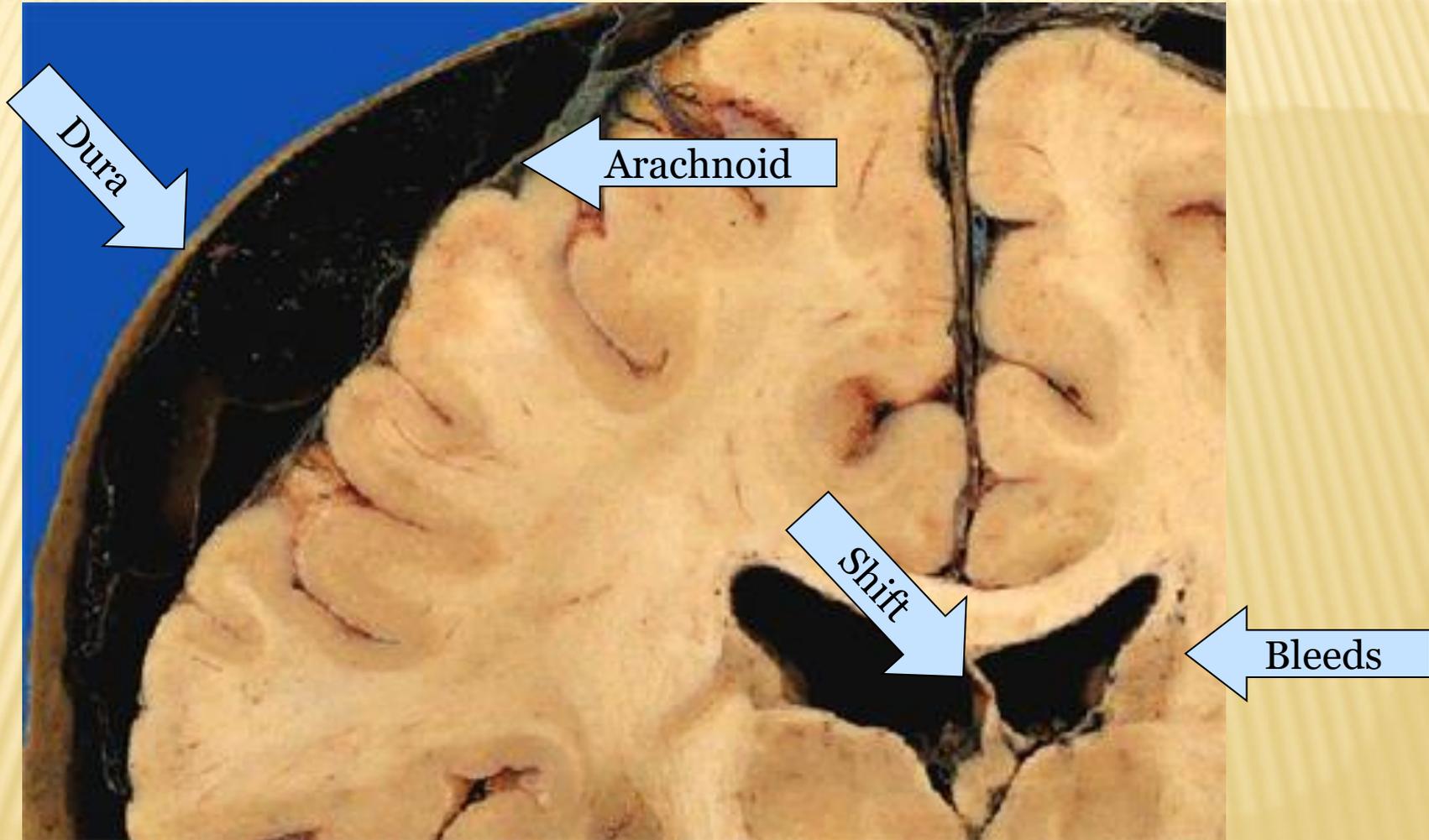


EPIDURAL HEMATOMA

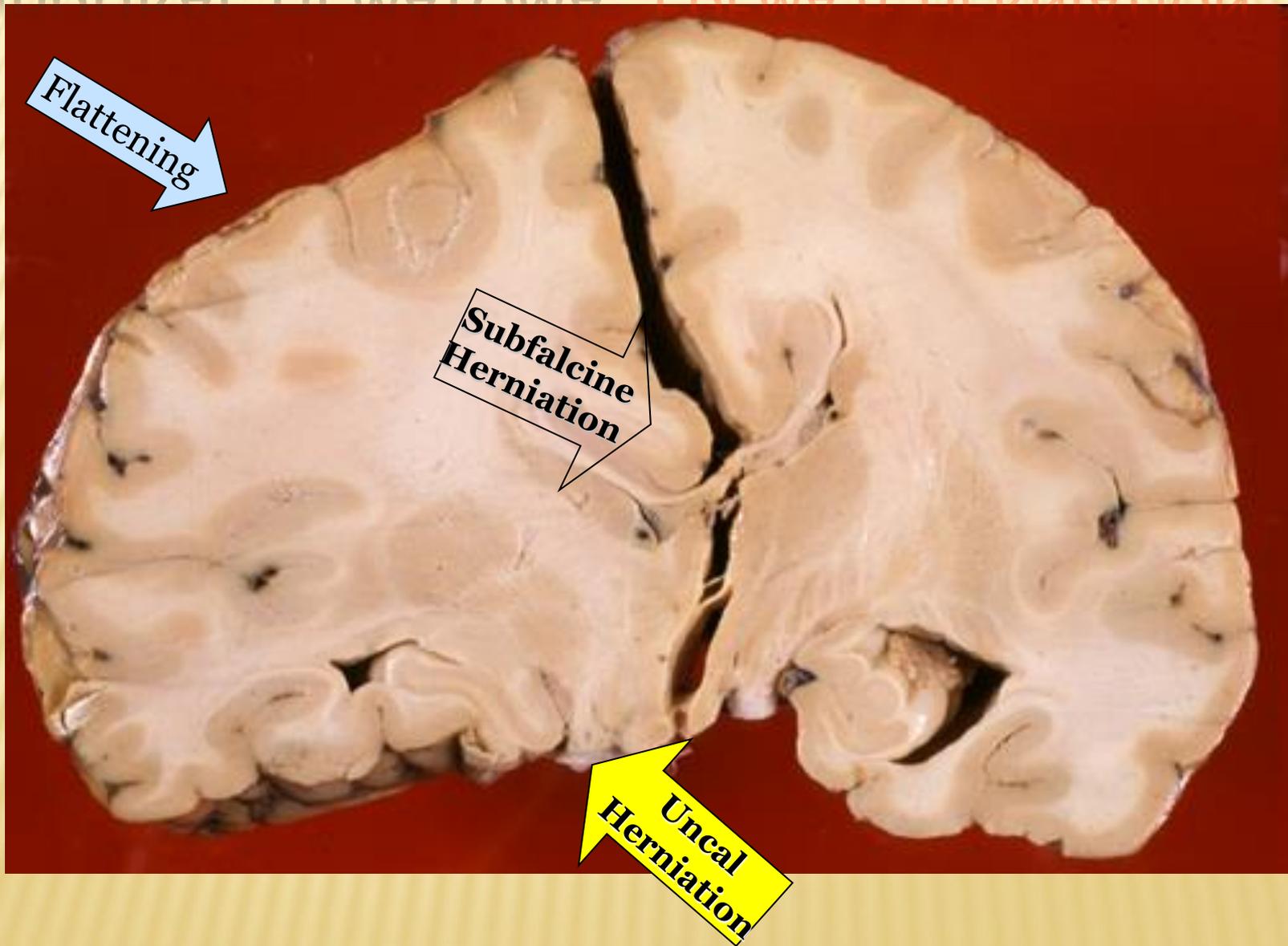


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SUBDURAL HEMORRHAGE



SUBDURAL HEMATOMA: EDEMA & HERNIATION



GLASGOW COMA SCALE

<u>Eye Opening</u>	<u>Best Verbal Response</u>	<u>Best Motor Response</u>
Spontaneous 4	Oriented, conversing 5	Obeys verbal commands 6
To verbal command 3	Disoriented, conversing 4	Localize to pain 5
To pain 2	Inappropriate words 3	Flexion/withdrawal 4
None 1	Incomprehensible sounds 2	Abnormal flexion (decorticate) 3
	No verbal response 1	Extension (decerebrate) 2
		No response (flacid) 1

GLASGOW COMA SCALE

- ✘ Score of 15 = normal
- ✘ 13 → 15 = mild head injury (awake, no significant focal deficits)
- ✘ 9 → 12 = moderate (altered sensorium and/or focal deficits, but can follow simple commands)
- ✘ 8 or below = severe (cannot follow even simple commands)



سنة ١٤٤٠ هـ

HERNIATION: COMPARTMENT SYNDROME OF THE HEAD

GOALS

- × Understand
- × Recognize
- × Treat

THEORY

- ✘ Compartment = skull contents
- ✘ Fixed volume:
 - + Brain (80%), blood (10%), csf (10%)
- ✘ Stable pressure: $CPP = MAP - ICP$

THEORY

Autoregulation of increased intracranial pressure (cough/valsalva):

Increased ICP



Increase MAP

Compartment's goal: Maintain CPP!!

ANOTHER LOOK

- ✘ Perfusion of a compartment:

$$CPP = MAP - ICP$$

If MAP cannot increase:

- ✘ Increased ICP = Decreased CPP
- ✘ Decreased CPP = Tissue ischemia
- ✘ Tissue ischemia = Edema

ANOTHER LOOK

- ✘ Edema = Further increased ICP
- ✘ Further decreased CPP = Tissue death

WHAT ELSE?

- ✘ **Monro-Kellie doctrine:**
 - Skull is a fixed volume
 - increase in one volume leads to decrease in others.

Brain > blood, csf

WHAT ELSE?

Increased ICP (via increased volume)



Displace blood/csf



****Displace brain** !!!!**

THEORY

Increased ICP via increased volume



Displaces blood, CSF, then brain
&

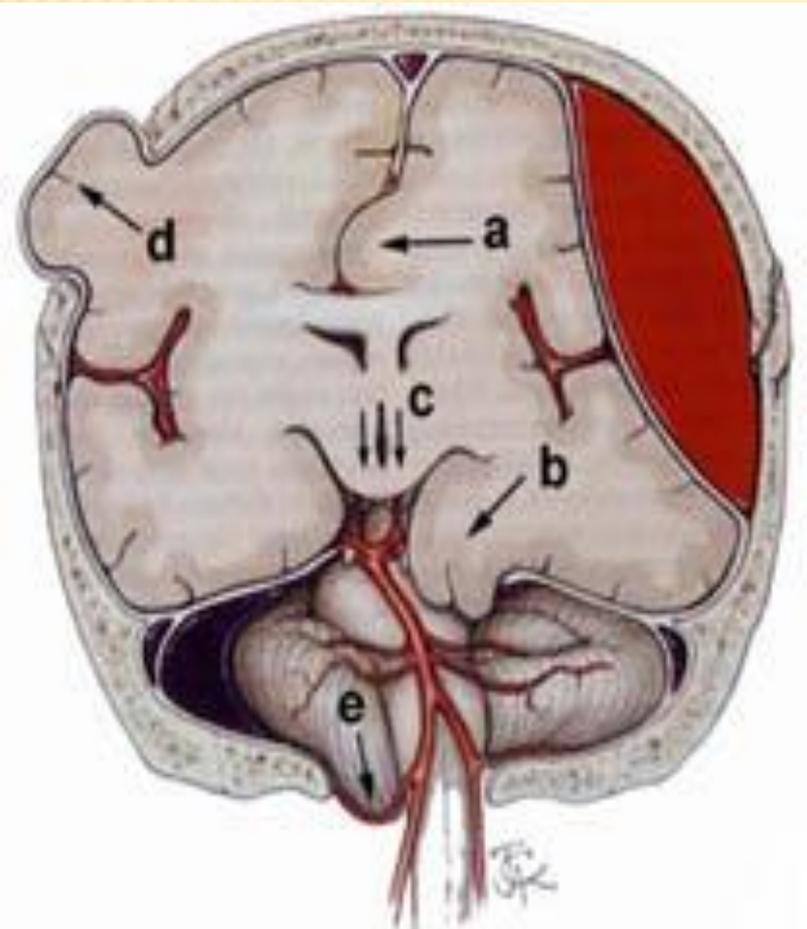
Reduces CPP causing brain ischemia

RECOGNIZE

Looking at a Head CT is not recognition

RECOGNIZE

Displaced brain will cause neurologic signs



A) SUBFALCIAL
(CINGULATE)
HERNIATION ;
B) UNCAL HERNIATION ;
C) DOWNWARD
(CENTRAL,
TRANSTENTORIAL)
HERNIATION ;
D) EXTERNAL
HERNIATION ;
E) TONSILLAR
HERNIATION.

SIGNS

- ✘ Where does displaced brain go?
 - + Side to side: subfalcine
 - + Side to bottom: uncal (transtentorial)
 - + Top to bottom: central tentorial
 - + Bottom to top: “upward”
 - + Bottom thru the “hole”: tonsillar



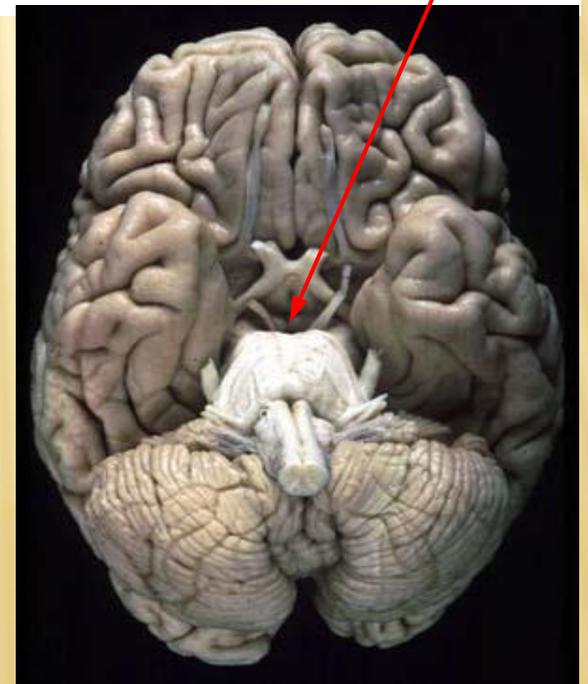
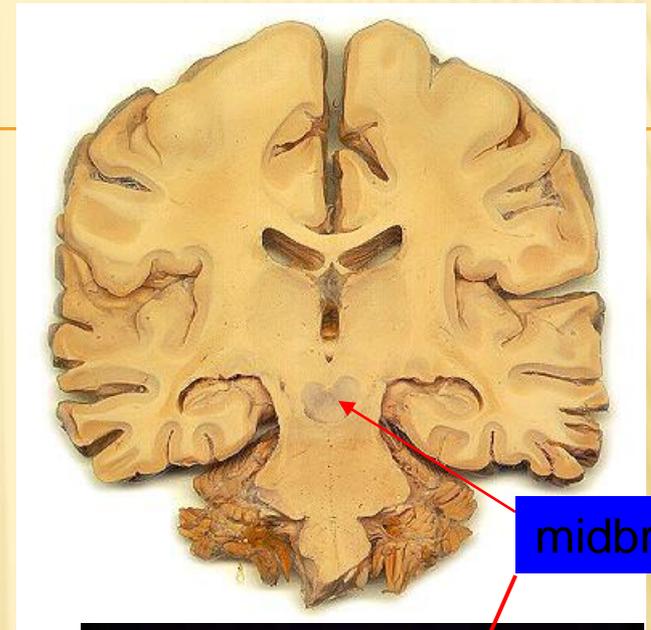
SIGNS

× Subfalcine

- + ACA compression:
contralateral leg paresis
- + **Somnolence**

× Uncal (transtentorial)

- + Anisocoria to “blown pupil”
- + Midbrain and PCA
compression:
Somnolence,
Contralateral hemiparesis,
occipital infarct
- + Decerebrate posturing
(extensor)

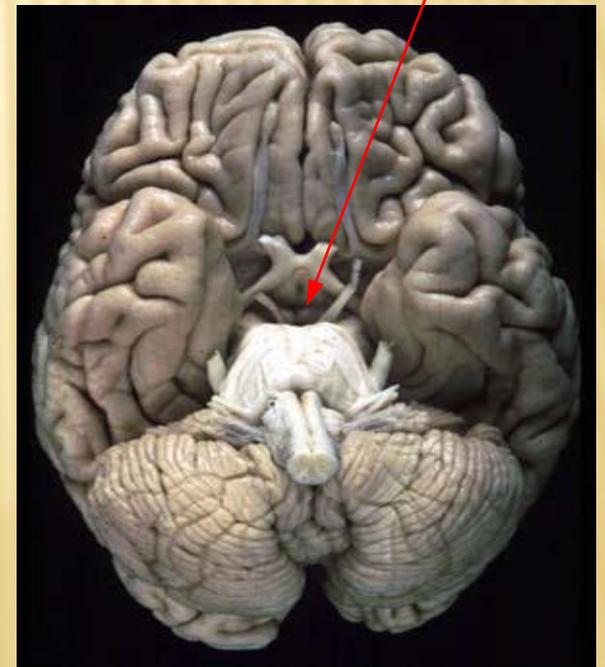
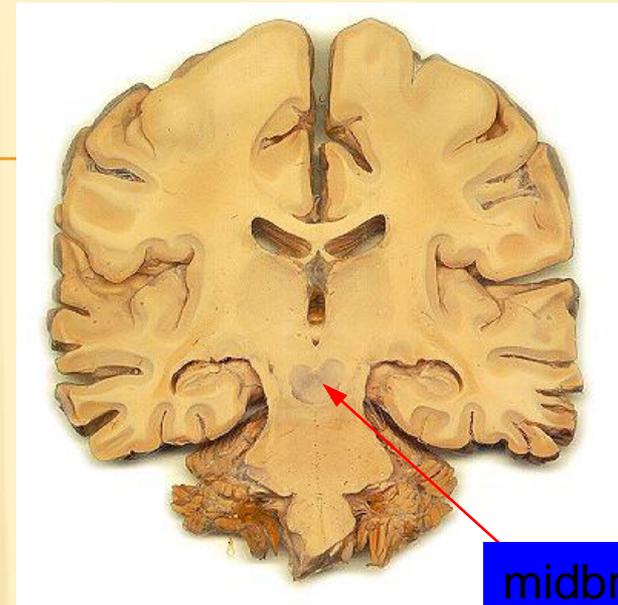


× SIGNS Central tentorial

- + Somnolence/coma
- + Bilaterally “blown” pupils
- + Decorticate/decerebrate posturing
- + Bilateral midbrain, PCA compression

× Upward (rare)

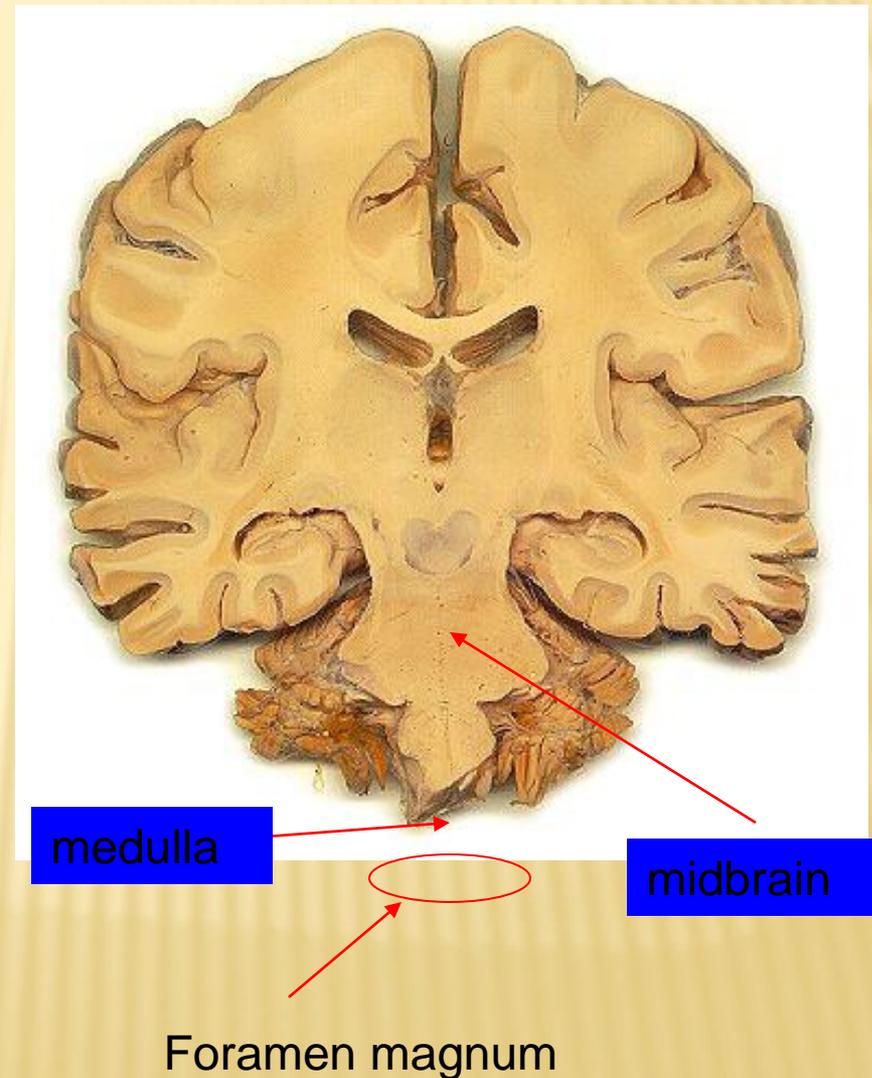
- + Midbrain compression
- + “Blown” pupils
- + Somnolence/coma



SIGNS

× Tonsillar

- + **Somnolence**
- + Quadriparesis
- + Cardiac arrhythmias
- + Respiratory failure



MORE SIGNS

- ✘ Vital sign changes (brainstem is being crushed):

“Cushing Reflex” :
Bradycardia/hypertension
respiratory change

- ✘ Somnolence/Coma
- ✘ EXAMINE PT: pupils, pupils, pupils

PUPILS?

- × Blown pupils: (large unreactive)
 - + Not medication unless ophthalmology came
 - + Or if under GENERAL anesthesia
 - + Compression of midbrain

- × Pinpoint pupils: (small unreactive)
 - + Often caused by medication (benzo's, opiates)
 - + Also from pontine damage

PUPILS?

× Anisocoria:

- + Sometimes normal or surgical
- + Sometimes meds: nebulizer
- + Compression of CN III

× Irregular:

- + Surgical
- + Ongoing ischemia: “cat eye”

EXAM SUMMARY

- ✘ Vital sign change is LATE
- ✘ Early exam change- somnolence
- ✘ Pupil change- anisocoria or less reactive
- ✘ Very late change: comatose, dilated pupils, posturing

TREATMENT

Head CT is not a treatment

TREATMENT

- ✘ Herniation = Brain CODE
- ✘ Stabilize your pt first: ABC's, then BCB
- ✘ Then conduct “secondary survey”

TREATMENT

- ✘ Control your compartment (BCB)
 - + Blood
 - + CSF
 - + Brain

BLOOD

- ✘ Allow outflow:
 - + Cut the tape off of the neck
 - + Head midline
 - + Head of bed at 45 degrees

- ✘ Constrict blood vessels:
 - + Hyperventilate: BAG!
 - + Goal is pH change- not pCO₂

CSF

× Drain CSF

- + Intraventricular catheter placement
- + Spinal CSF removal will let you herniate faster

BRAIN

Steroids only work on tumors

BRAIN

- × Shrink

- + Hyperosmolar agents: mannitol, hypertonic saline
- + Doses? Bolus vs infusion.

- × Cut

- + Surgical removal

- × Shut down

- + Neuroanesthetic agents: propofol, thiopental

HOW LONG?

Hyperosmolar agents and hyperventilation
lasts 6 hours at best.

THEN WHAT?

- ✘ Your pt is better
- ✘ Your pt is not better

THEN WHAT?

Head CT verifies your diagnosis
and identifies the problem

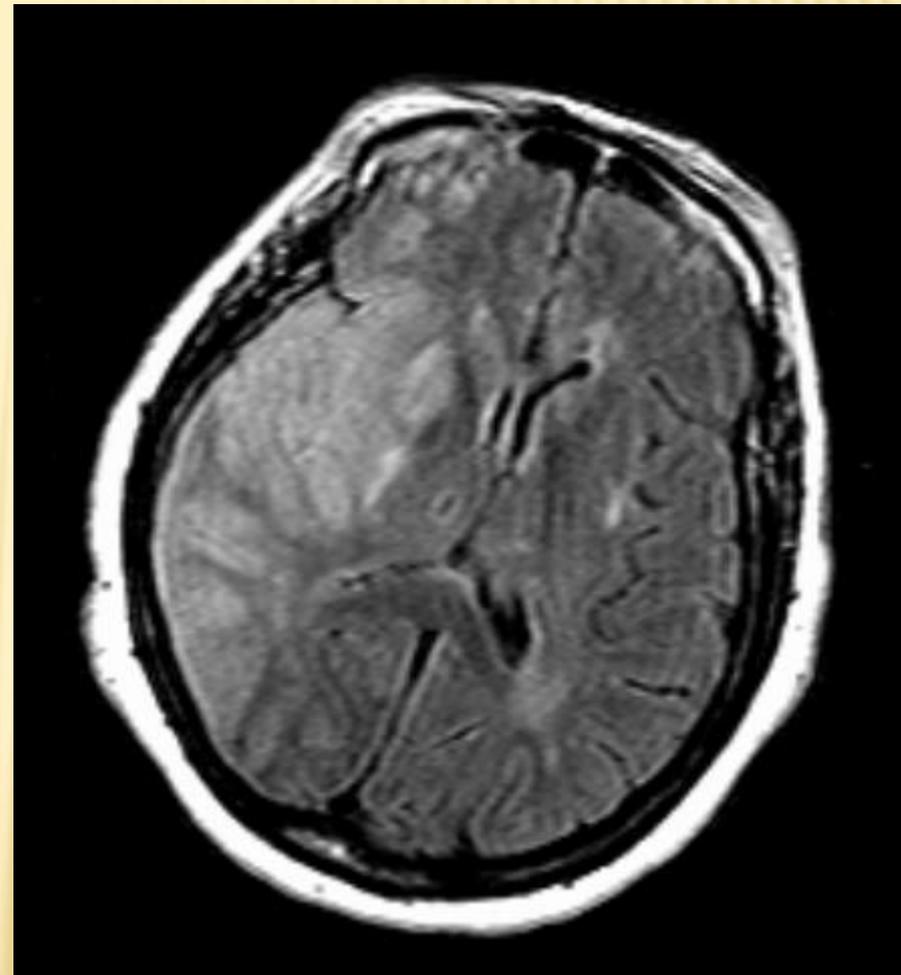
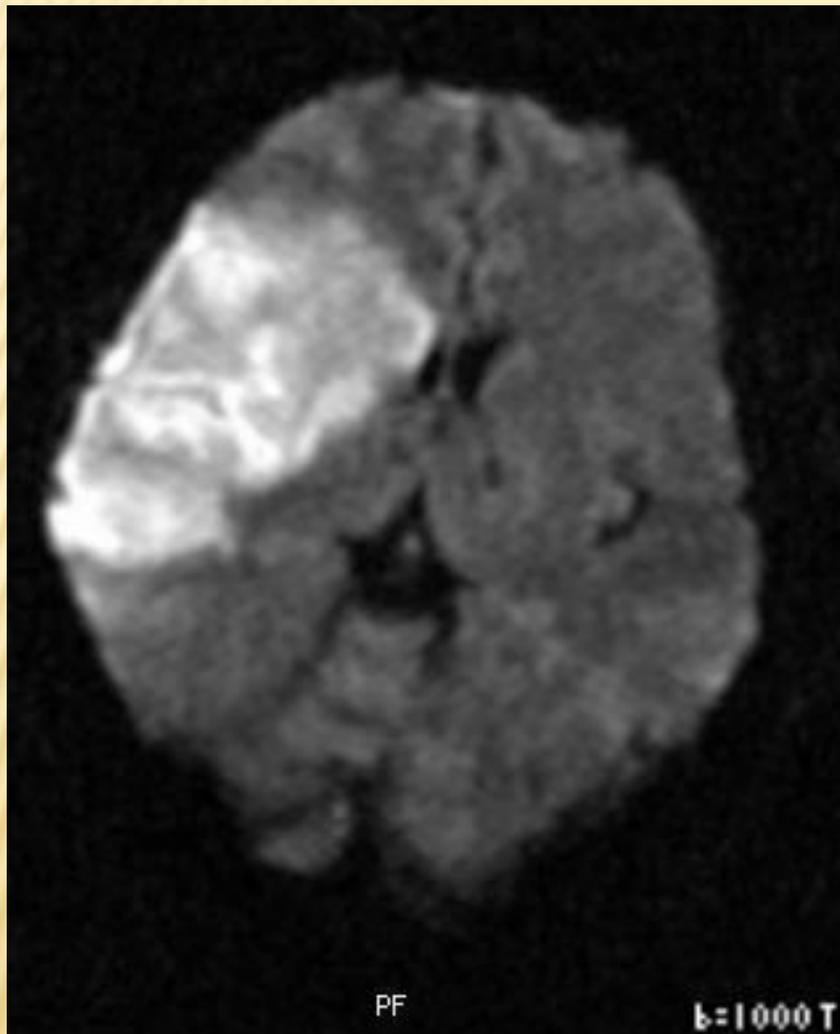
RELEASE

- ✘ Release the compartment : “pop the top”
- ✘ Early vs. late

CASE

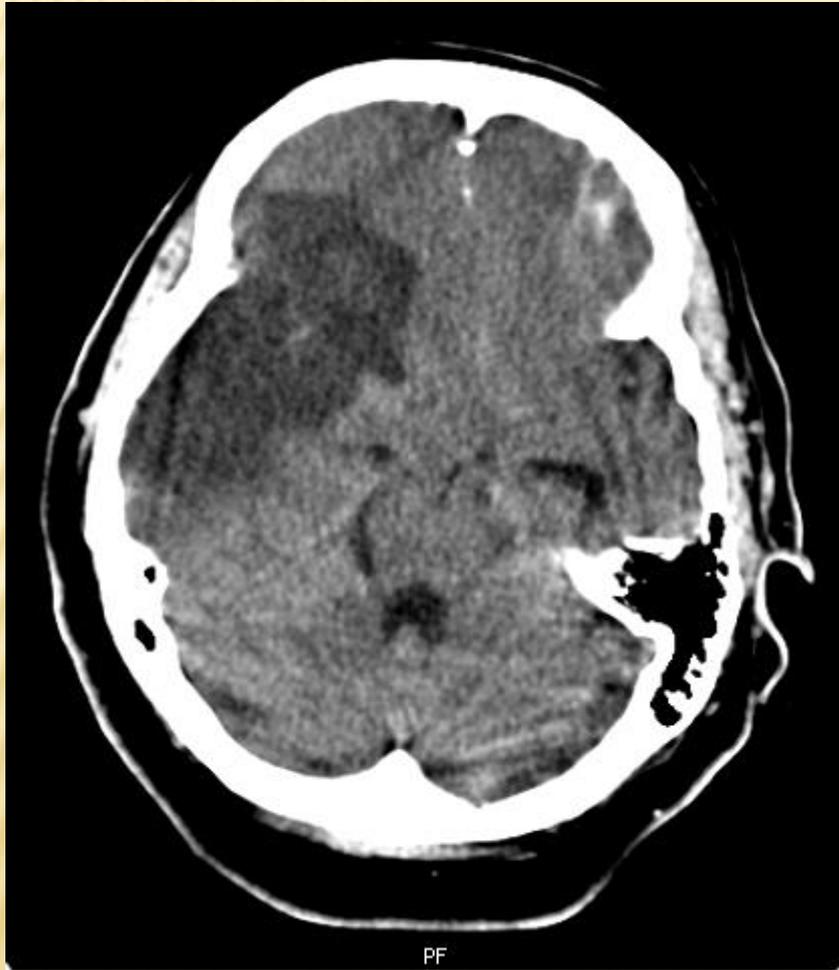
✘ 60 yo wm s/p acute right MCA stroke





CASE

- ✗ 4 days later
 - ✗ Pt becomes somnolent but arousable
 - ✗ There's new anisocoria
-
- ✗ Plan?



CASE

- × 12 hours later
- × Not arousable- comatose
- × Still anisocoria, right pupil stops reacting

- × Plan?



CASE

Other options?

2004-05-21
13:27:00



2004-05-21
13:27:05



SUMMARY

- ✘ Look outside your “box”
- ✘ Herniation is reversible
- ✘ Treat before scanning