Imaging of Brain Trauma

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Goals and Objectives

- Epidemiology and wide ranging implications of CNS trauma
- Imaging options available for diagnosis of trauma
- Understand the role of the radiologist in the workup and diagnosis of traumatic brain injury
- Discuss the primary effects of CNS trauma with emphasis on extraaxial and parenchymal injury
- Be able to recognize those conditions associated with the greatest morbidity/mortality and the downstream complications of CNS trauma

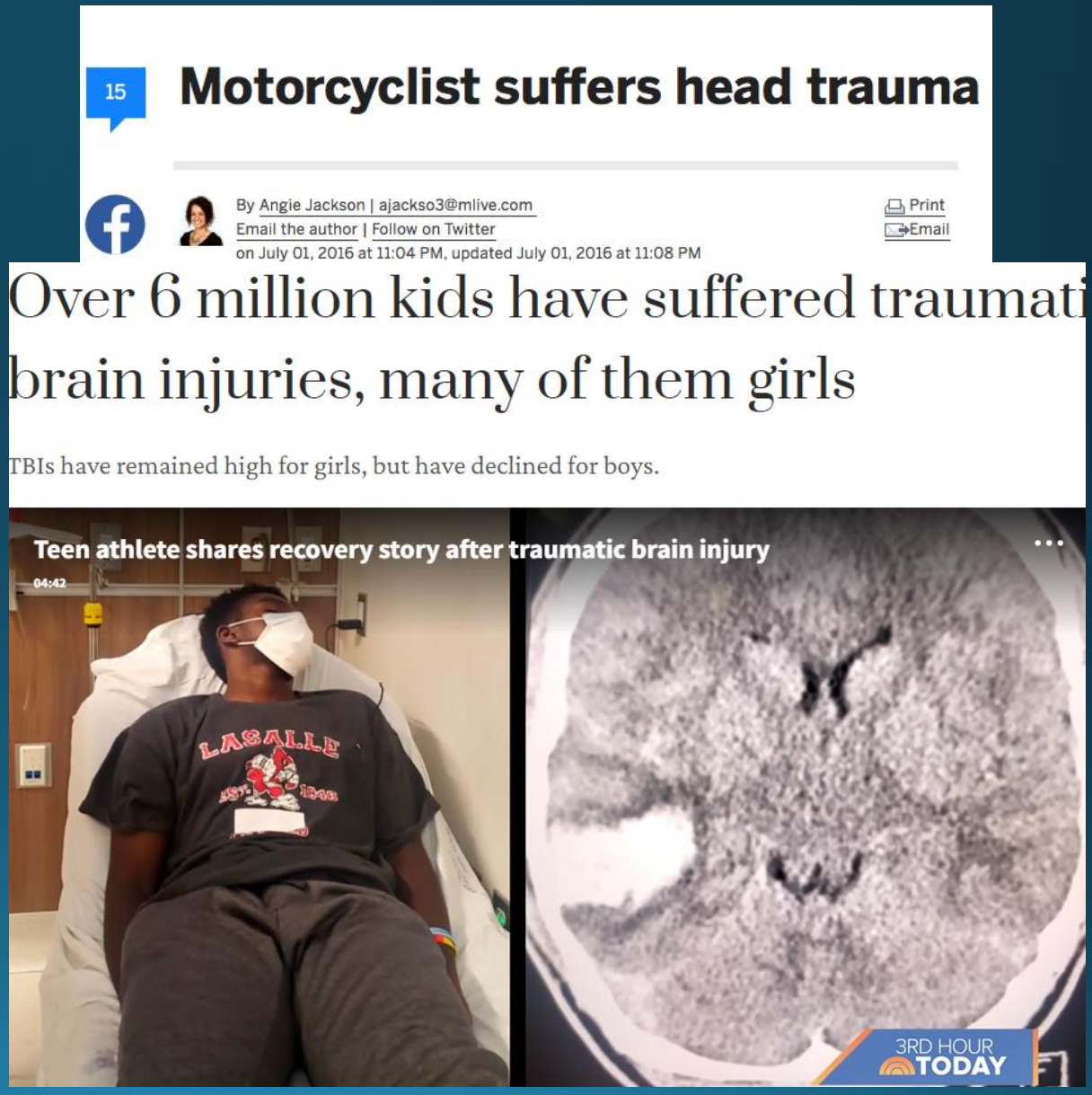
Acknowledgements

- Anne Osborn, MD, FACR
- Chris Therasse, MD
- Crystal Farrell, MD

Introduction

Epidemiology of head trauma:

- Most common worldwide cause of death and disability in children and young adults
- More than 2 million people suffer a traumatic brain injury annually (US alone), with nearly 25% requiring hospital care
- Of all head-injured patients, ~10% sustain fatal injury and 5-10% have serious permanent deficits



Introduction

Mechanisms of Injury:

- Missile injury
  - Penetration of the skull, meninges, and/or brain by a penetrating external object (bullet, ice pick, nail gun…)

- Non-missile injury
  - "Closed head injury"
  - Much more common cause of neurotrauma than missile injury
  - Acceleration/deceleration forces, rotation and sudden changes in angular momentum

Introduction

Etiology of Injury:

- Falls
  - Causative factor in 30% of traumatic brain injuries (TBIs)
  - Leading cause of TBI in children < 4 years and in elderly patients > 75 years

- Motor vehicle and auto-pedestrian collisions
  - Occur at all ages without gender predilection

- Gunshot wounds
  - Most commonly seen in adolescent and young adult males

Introduction

Classification:

- Glasgow Coma Scale (GCS)
  - Most common clinical classification of brain trauma
  - Assessment of three features: best eye, verbal, and motor responses
  - Based on a scale of 3-15
  - Injury designated as mild (13-15), moderate (9-12), or severe (3-8)

Glasgow Coma Scale		
Response	Scale	Score
Eye Opening Response	Eyes open spontaneously	4 Points
	Eyes open to verbal command, speech, or shout	3 Points
	Eyes open to pain (not applied to face)	2 Points
	No eye opening	1 Point
Verbal Response	Oriented	5 Points
	Confused conversation, but able to answer questions	4 Points
	Inappropriate responses, words discernible	3 Points
	Incomprehensible sounds or speech	2 Points
	No verbal response	1 Point
Motor Response	Obeys commands for movement	6 Points
	Purposeful movement to painful stimulus	5 Points
	Withdraws from pain	4 Points
	Abnormal (spastic) flexion, decorticate posture	3 Points
	Extensor (rigid) response, decerebrate posture	2 Points
	No motor response	1 Point
Minor Brain Injury = 13-15 points; **Moderate Brain Injury** = 9-12 points; **Severe Brain Injury** = 3-8 points		

Introduction

Classification:

- Primary effects
  - Scalp and skull injuries
  - Extraaxial hemorrhage
  - Parenchymal injuries
  - Vascular injuries
  - Miscellaneous injuries
- Secondary effects
  - Herniation syndromes
  - Cerebral edema
  - Cerebral ischemia

Imaging Acute Head Trauma

Goals of the radiologist:

- Identify treatable injuries, especially the emergent ones
- Detect the presence of secondary injuries or risk factors predisposing the patient to downstream complications
- Provide input on the choice of imaging study when consulted

Imaging Acute Head Trauma

How to image:

- Non-enhanced head CT:
  - Standard of care screening tool for imaging acute head trauma
  - Depicts both bone and soft tissue injuries
  - Fast(!), effective, widely accessible, and relatively inexpensive

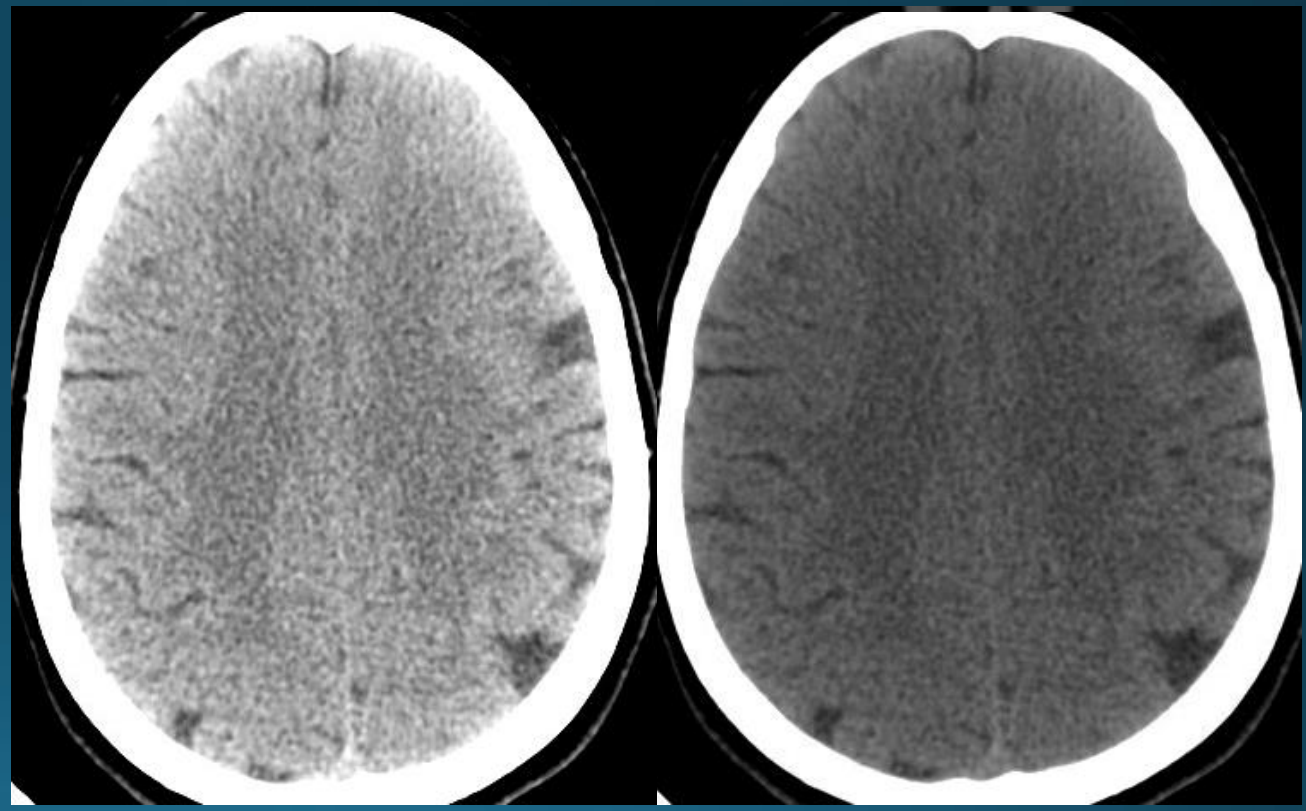
Technique:

- 2.5-3-mm sections from below the foramen magnum through the vertex
- "Brain" and "bone" algorithms should be obtained
- Use your reformats!
- Use a variety of windows:
  - "Blood windows": W 160; L 60
  - "Soft tissue windows": W 260; L 80
  - "Brain windows": W 80; L 40
  - "Stroke windows": W 40; L 40
  - "Bone windows": W 3000; L 500

Imaging Acute Head Trauma

Window / Level Selection:

- Brain windows optimize low contrast between brain structures
- Bone windows optimize visualization of bony detail
- Blood windows optimize window center around blood density to identify extraaxial collections near the skull interface



Brain windows

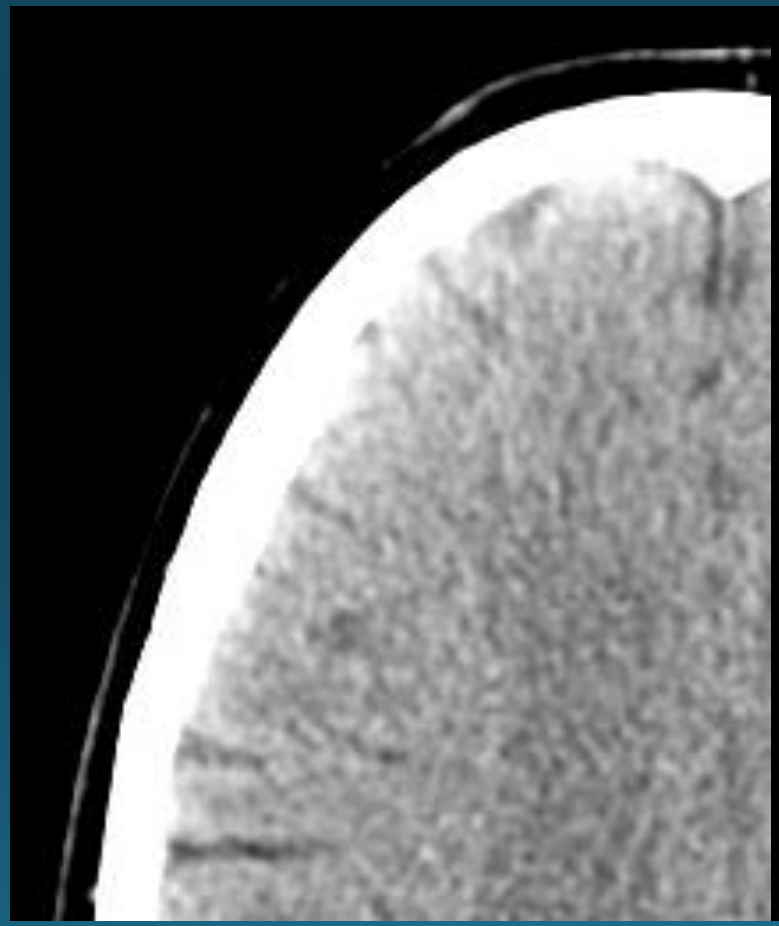
Blood windows

Imaging Acute Head Trauma

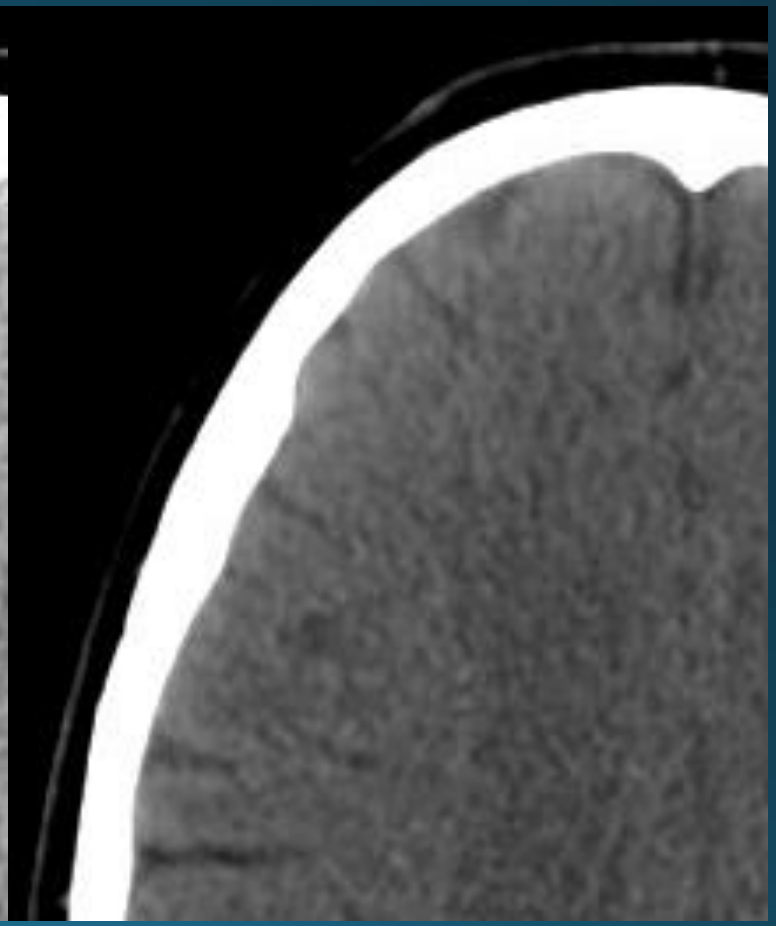
TRICKS OF THE TRADE:

"Cupping" reduced with blood windows.

Note the better definition of the brain and subarachnoid spaces adjacent to the skull



Brain windows

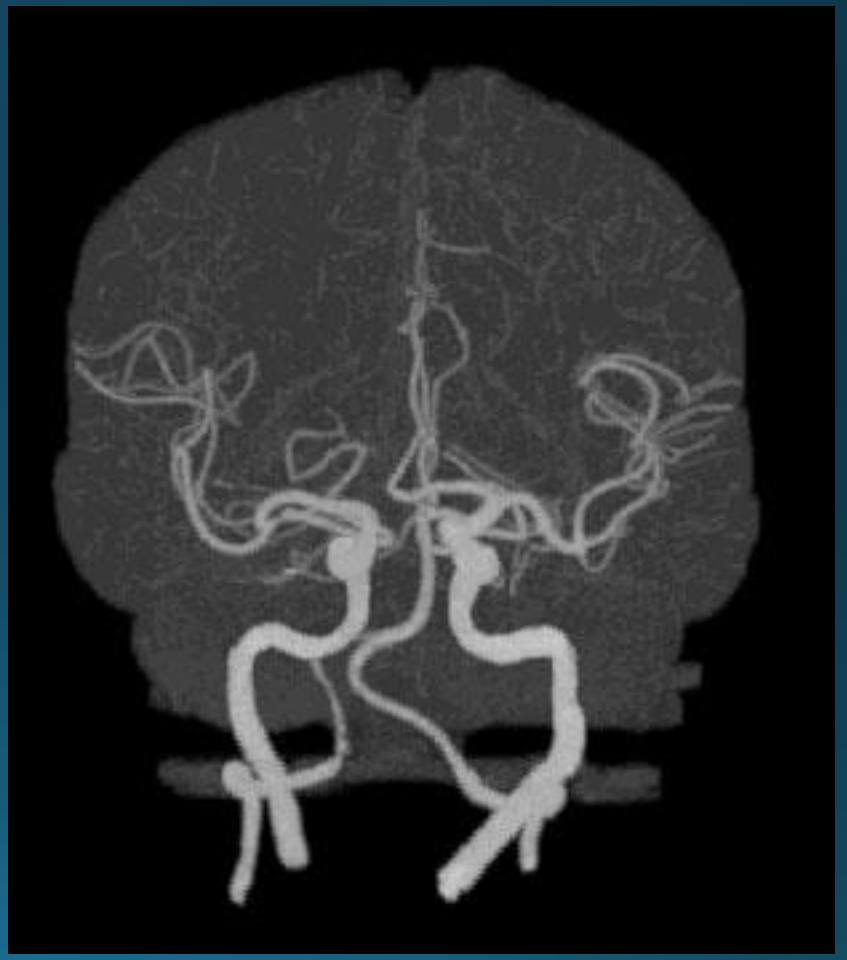


Blood windows

Imaging Acute Head Trauma

How to image:

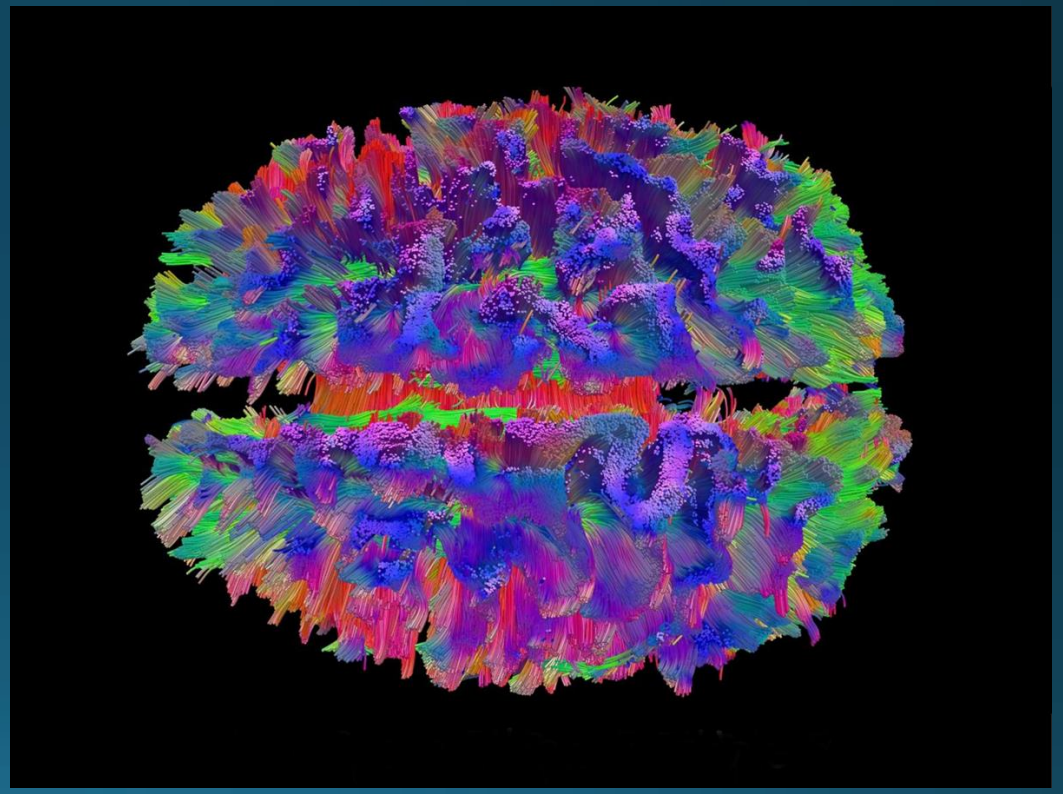
- CT angiography (CTA)
  - Considered in the setting of skull base fractures traversing the carotid canal or a dural venous sinus
  - Nicely display dissection, pseudoaneurysm, carotid-cavernous fistula, or dural sinus injury
  - Also useful in the setting of penetrating neck injury, fracture encroaching on the vertebral artery, or facet dislocations



Imaging Acute Head Trauma

How to image:

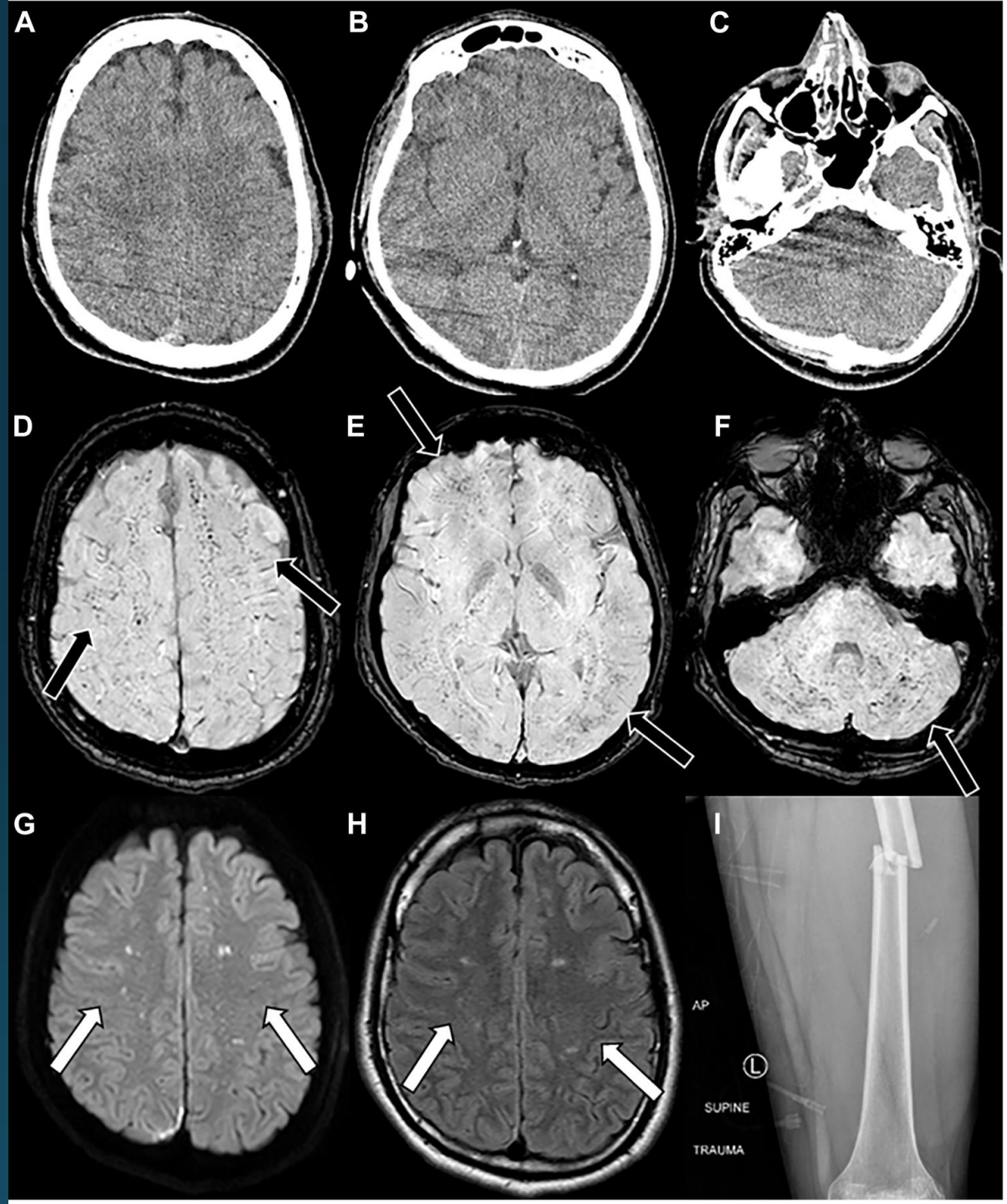
- MRI
  - Uncommonly used in the setting of acute brain trauma
  - Very useful for cases of suspected child abuse and in subacute/chronic stages of TBI
  - Advanced techniques such as diffusion tensor imaging (DTI) and functional MRI (fMRI) are gaining increased use in following patients with TBI



MR is an important adjunct to CT in TBI

- Early detection of focal intra-axial primary or secondary injuries
  - Diffuse axonal injury
  - Edema
  - Infarction
  - fat emboli

(Little to no correlation on CT in the acute phase)



Conventional MR Imaging in Trauma Management in Adults. Ange Diouf MD, FRCPCand Matylda Machnowska MD, FRCPC. Neuroimaging Clinics of North America, 2023-05-01, Volume 33, Issue 2, Pages 235-249, Copyright © 2023

Most extra-axial injuries are readily detectable on CT

- MR imaging shows slightly higher sensitivity for some extra-axial hemorrhages, especially in the subacute and chronic phases.
- Recent advances in image acquisition strategy have supported the rapidly increased use of MR imaging in trauma patients.

Analyzing Acute Head Trauma

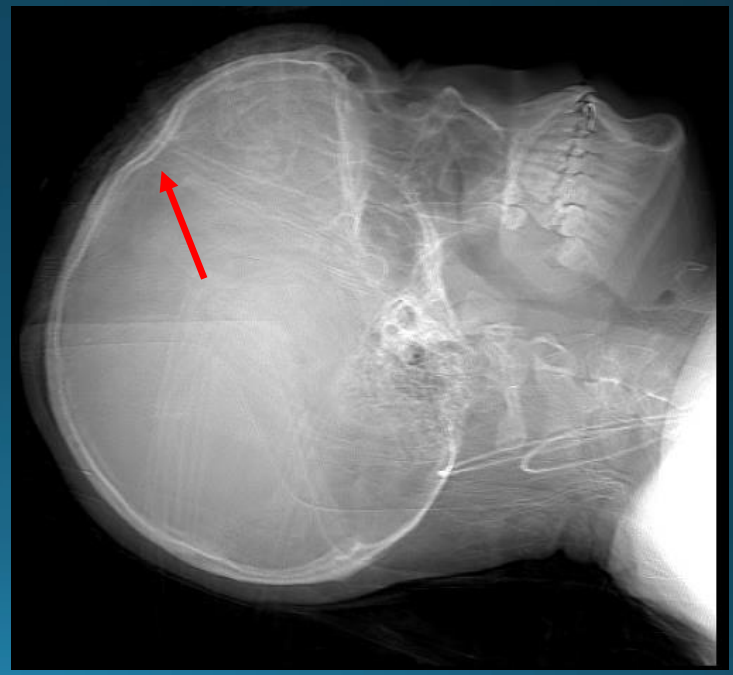
Approach to acute head trauma (CT):

- Scout image

- Evaluate multiple windows

Scout image

- ALWAYS review scout images on every case
- Cervical spine abnormalities (fracture, dislocation)
- Foreign bodies and jaw trauma that may be out of the field of view of the CT exam
- Some skull fractures may be best seen on the scout view (pediatrics), especially if reformats and not routinely performed at your institution



Blood Windows in Acute Head Trauma

- Width 160, level 60
- Small extraaxial collections can "blend" with the calvarium on standard narrow window widths and be overlooked
- These collections become more apparent on wider windows

TRICKS OF THE TRADE:

On bone algorithm images, use the "soft tissue" window to bring out subtle areas of hemorrhage

Brain Windows in Acute Head Trauma

- Work from outside to inside
  - Scalp → any swelling? If so, scrutinize for fracture and underlying blood
  - Extraaxial spaces → look for BLOOD!
    - Most common extraaxial hemorrhage is traumatic subarachnoid hemorrhage (SAH), followed by subdural and epidural hematomas
    - Most common place for SAH is in sulci adjacent to cortical contusions, within the sylvian fissures, and around the anteroinferior frontal and temporal lobes
    - Subtle SAH may collect within the interpeduncular cistern
  - Extraaxial spaces --> look for air
    - Always abnormal and indicates the presence of fracture (paranasal sinus or mastoid)

Brain Windows in Trauma

- Work from outside to inside
  - Brain → examine the cortex for contusion
    - "High yield" areas for contusion are the anteroinferior frontal and temporal lobes
    - If there is a scalp hematoma ("coup" injury), look directly opposite for a "contre-coup" injury
  - Brain → examine the subcortical white and deep gray matter
    - Subcortical hemorrhages often signify diffuse axonal injury (DAI) and are a "tip of the iceberg" finding
  - Brain → examine the ventricles
    - Look for blood-CSF levels and hemorrhage

Bone Windows in Trauma

- Evaluate carefully for fractures near site of trauma ("coup" injury)
  - If there is no overlying scalp injury, very unlikely to have a fracture
  - "Don't miss fractures"
    - Fractures involving the carotid canal
    - Fractures crossing other vascular structures (middle meningeal artery, dural sinus)
- Evaluate for mandibular dislocation

TRICKS OF THE TRADE:

Non-displaced fractures that don't cross vascular structures are basically meaningless in and of themselves!

Soft Tissue Windows in Trauma

- One last check for incidental findings:
  - Run the vessels → look for an obvious aneurysm
  - Check the neck → don't miss nasopharynx, parotid, or masticator space masses!
  - Evaluate the orbits → look at globes, muscles, and fat
  - Sagittal screen → exclude sellar mass or craniocervical junction abnormality (Chiari)

Primary Effects of CNS Trauma

Outline:

- Extraaxial hemorrhages:
  - Epidural hematoma
  - Subdural hematoma
  - Subarachnoid hemorrhage
- Parenchymal injuries
  - Cerebral contusions
  - Diffuse axonal injuries
- Miscellaneous injuries
  - Pneumocephalus
  - Missile and penetrating injuries

CT of Blood

	CT density	Time Course	Explanation
Hyperacute	High density	<6 hours	High Protein
Acute	High density	~8-72 hours	High Protein
Early Subacute	High density	~3 days to 1 week	High Protein
Late Subacute	Isodense	~1 wk to months	High Protein
Chronic	Low density	Months to years	Absorption of High Protein

Source: Neuroradiology Requisites. Grossman and Yousem, 2nd edition

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Timing	Blood product	T1	T2
Acute	Deoxy Hb	Isointense (Iso)	Hypointense (Dark)
Early Subacute	Intracellular Met Hb	Hyperintense (Bright)	Hypointense (Dark)
Late Subacute	Extracellular Met Hb	Hyperintense (Bright)	Hyperintense (Bright)
Chronic	Hemosiderin	Hypointense (Dark)	Hypointense (Dark)

Hyperacute (<6 hours) is D-B like CSF—mixture of oxy and deoxy Hb

MR of BLOOD

	Delay	Hemoglobin Degradation Status	T1WI	T2WI	T2*
Hyperacute	<6 h	Oxyhemoglobin	Mild hyperintense	Very hyperintense	Peripheral hypointensity
Late acute	6–72 h	Deoxyhemoglobin	Isointense to hypointense	hypointense	Hypointense (blooming)
Early subacute	3 days to 1 week	Intracellular methemoglobin	Very hyperintense	Hypointense	Hypointense (blooming)
Late subacute	1–2 weeks to 2 months	Extracellular methemoglobin	Very hyperintense	Very hyperintense (with peripheral hypointensity)	Variable
Chronic	2 weeks to years	Hemosiderin	Hypointense	Hypointense	Hypointense (blooming)

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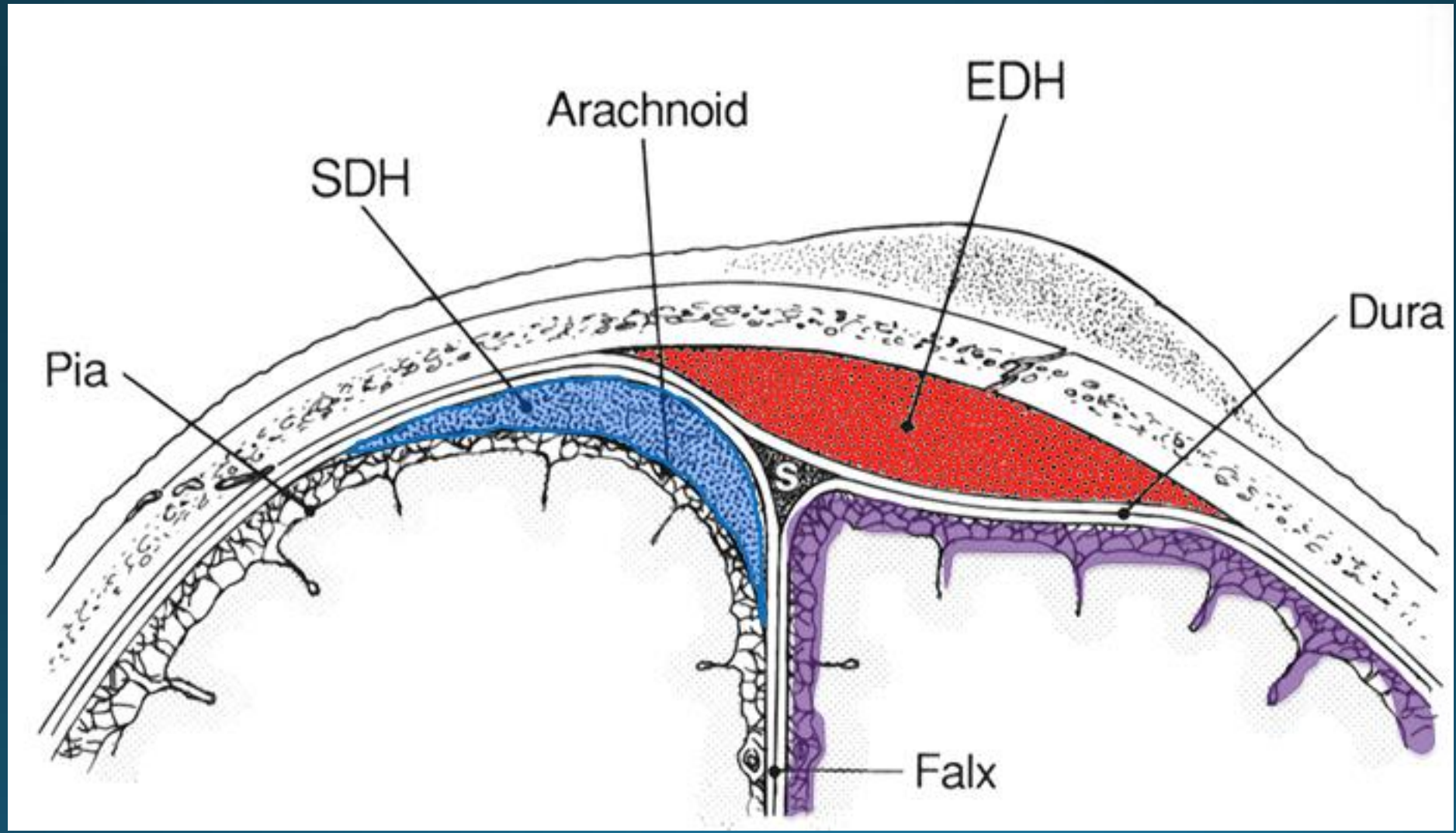
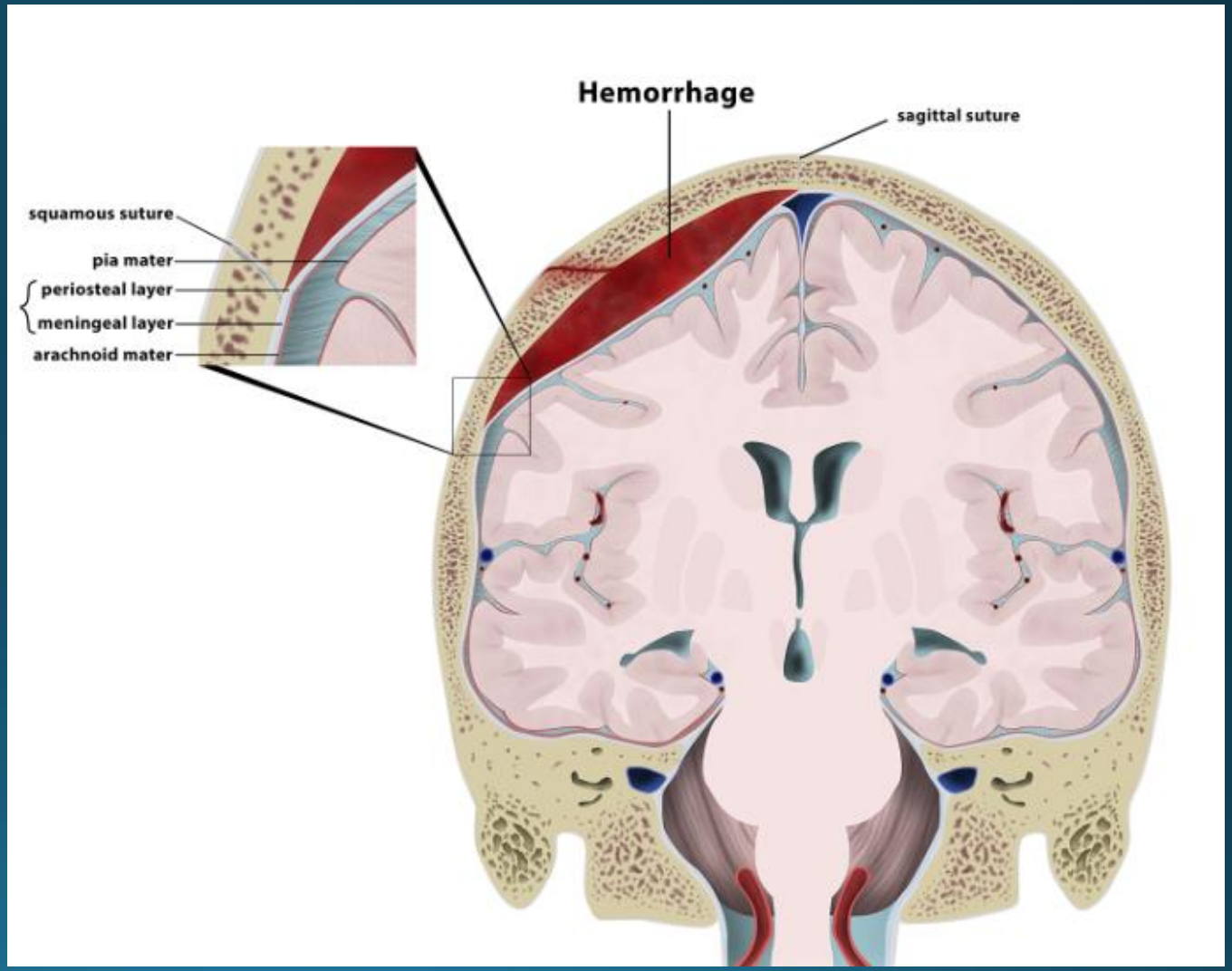
Extraaxial Hemorrhage



Source: https://sites.google.com/a/wisc.edu/neuroradiology/spine-anatomy-1/case-2/case-2-discussion-continued

Acute Epidural Hemorrhage

- Blood between the calvaria and outer (periosteal) layer of the dura
- Direct trauma to the skull with laceration of an adjacent artery (90%), most commonly the middle meningeal artery
- 10% are venous and usually secondary to a fracture crossing a dural venous sinus
- Vast majority are supratentorial in location

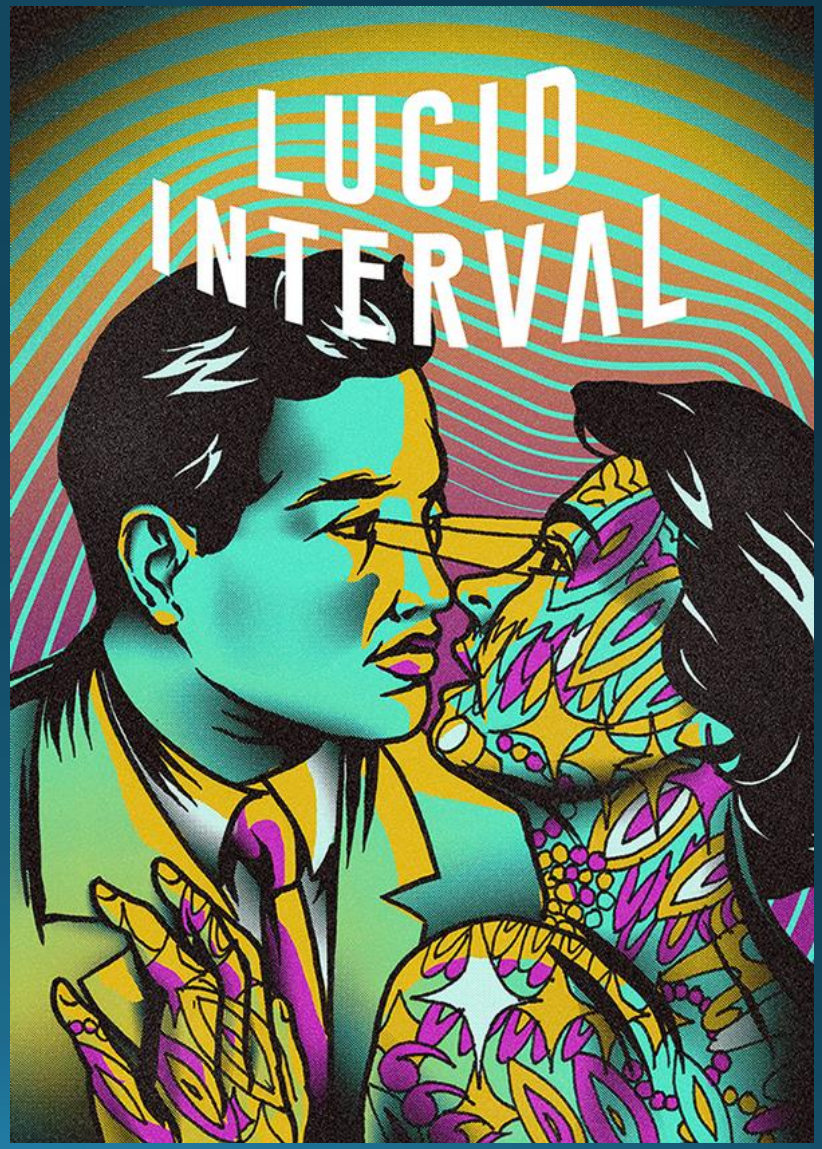


Source: http://radiopaedia.org/images/2917400

Acute Epidural Hemorrhage

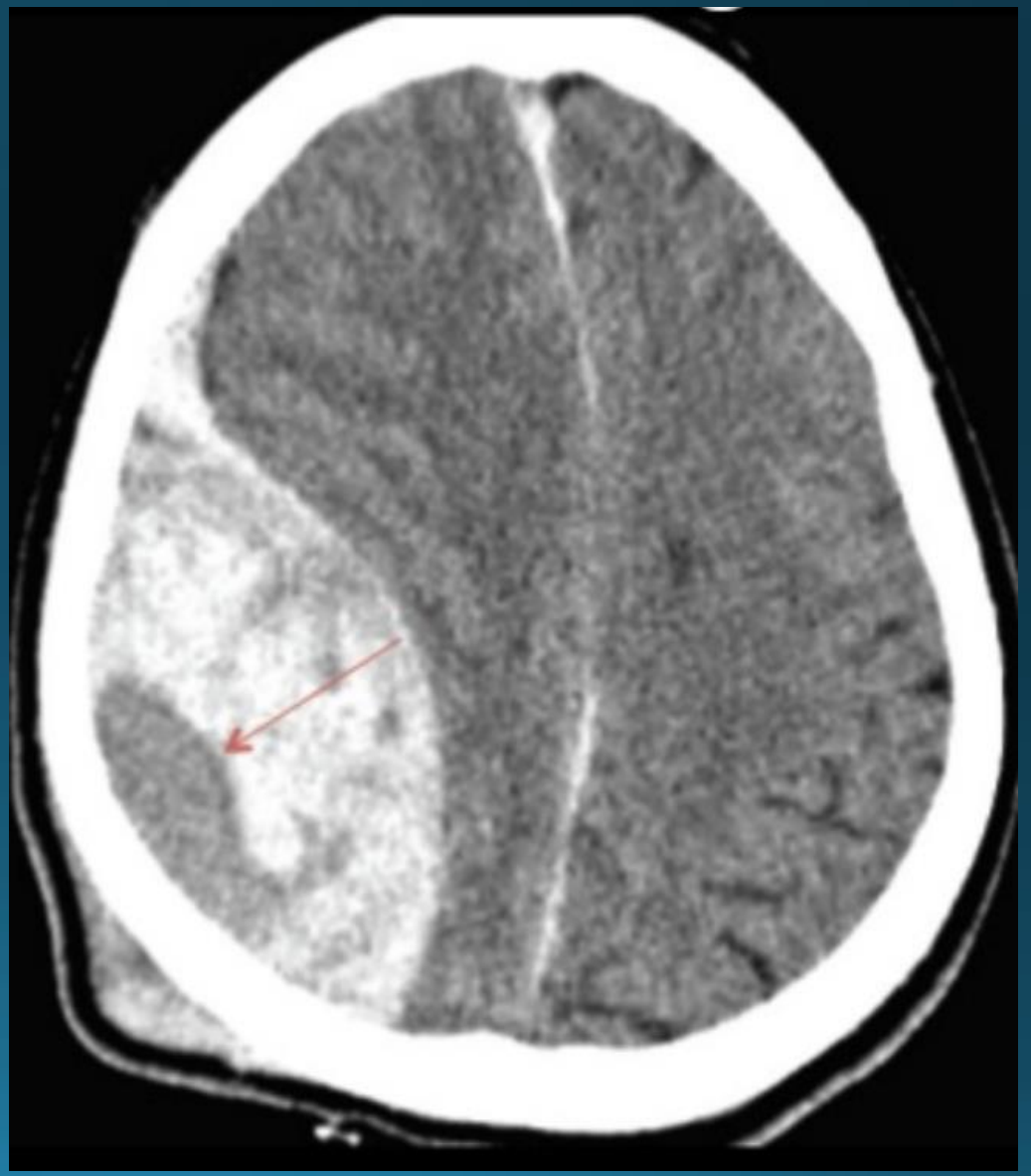
Clinical Issues:

- Much less common than SAH or subdural
- "Lucid interval" occurs in ~50%, refers to initial loss of consciousness followed by asymptomatic period prior to neurologic deficits
- Mortality rate with prompt recognition and treatment is <5%
- Most EDHs are surgically evacuated



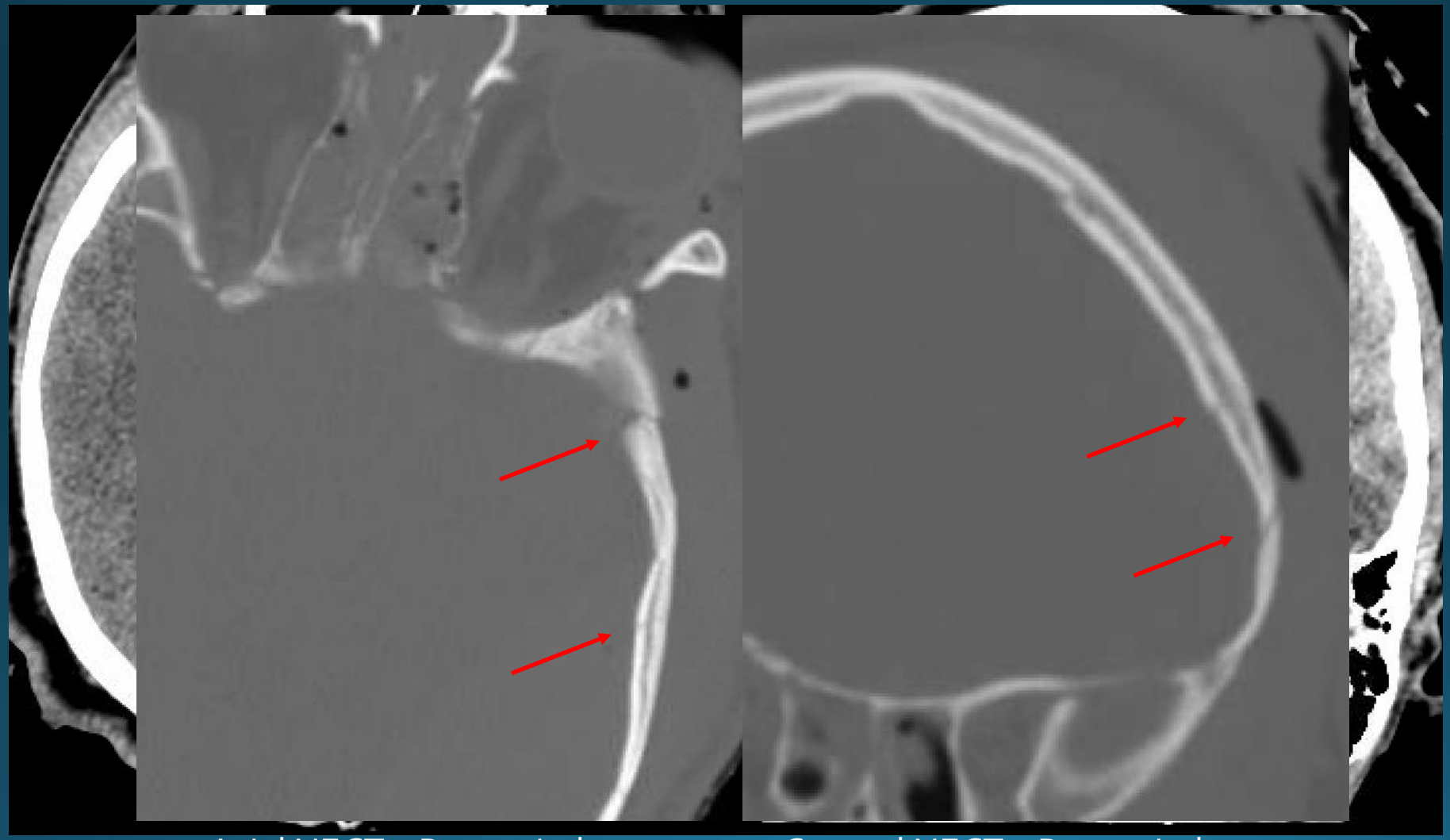
Biconvex extraaxial collection

- Confined by intracranial sutures but can cross falx or tentorium (more often with venous EDHs)
- Compresses/displaces underlying brain and subarachnoid spaces
- "Swirl" sign: active/rapid bleeding
- Skull fracture in 90-95%



Acute Epidural Hemorrhage

35-year-old with head trauma, unconscious at scene.



Axial NECT – Bone windows

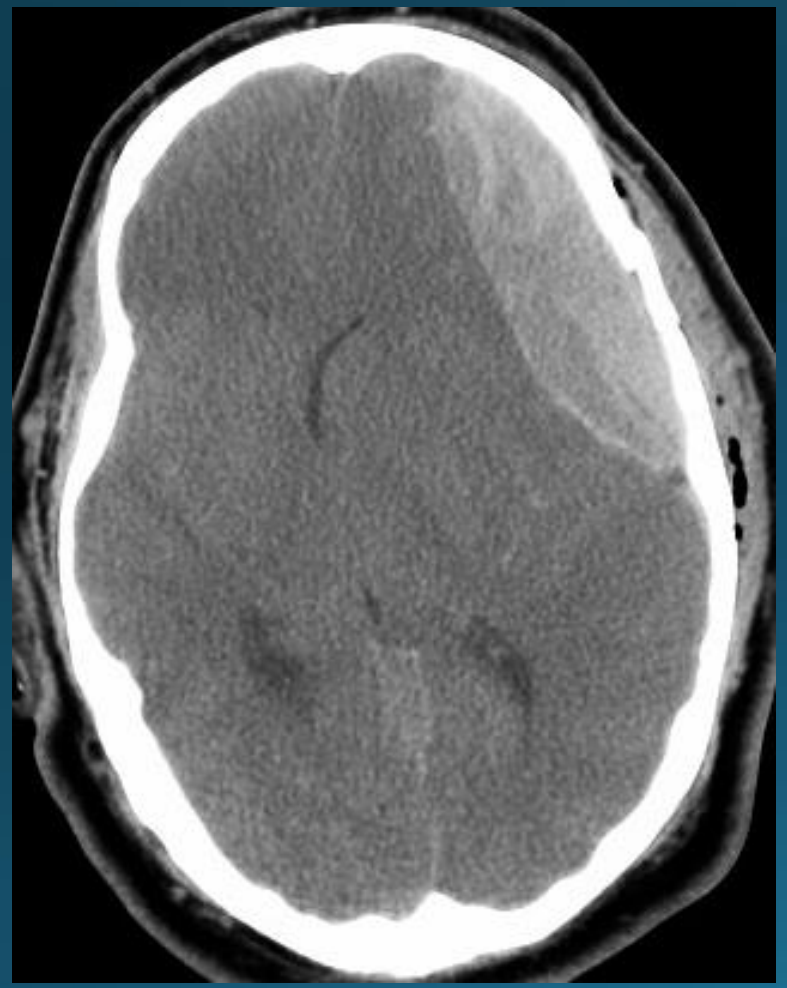
Axial NECT

Coronal NECT – Bone windows

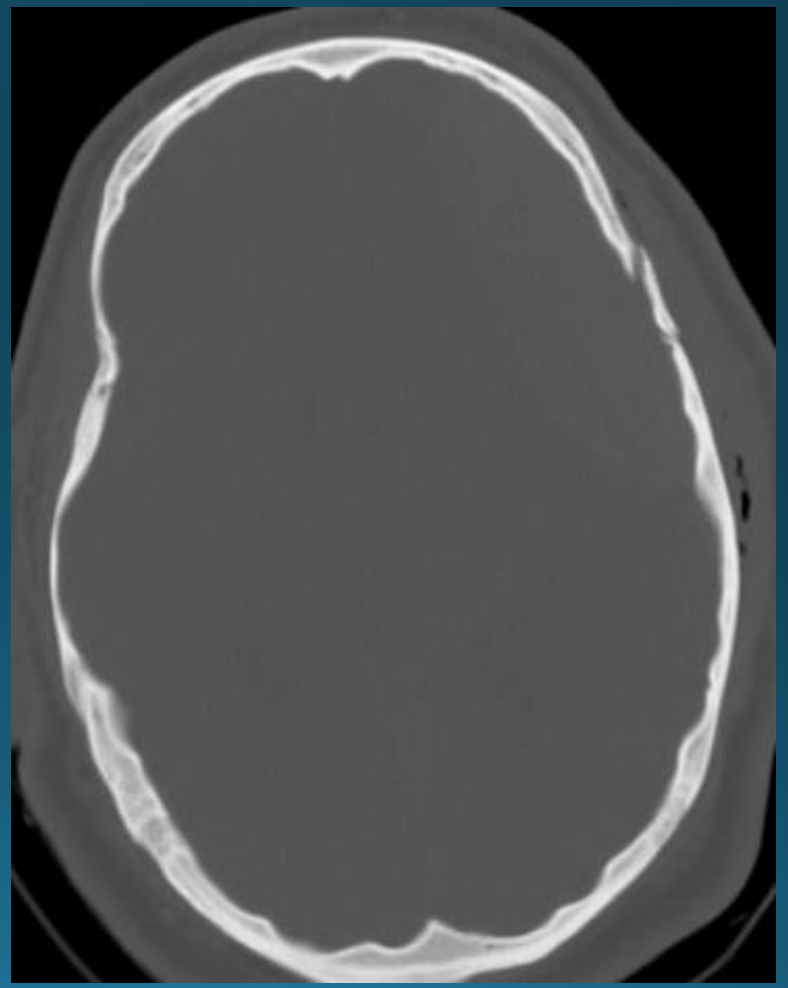
Coronal NECT

Acute Epidural Hemorrhage

27-year-old with head trauma, GCS of 3 at scene.



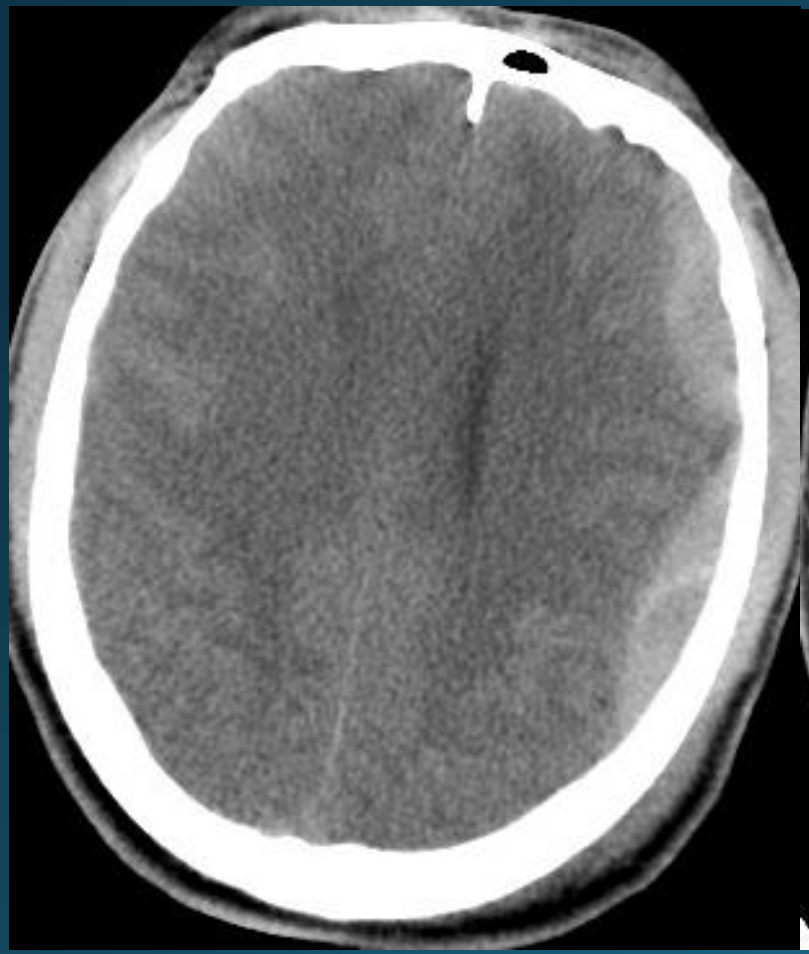
Axial NECT



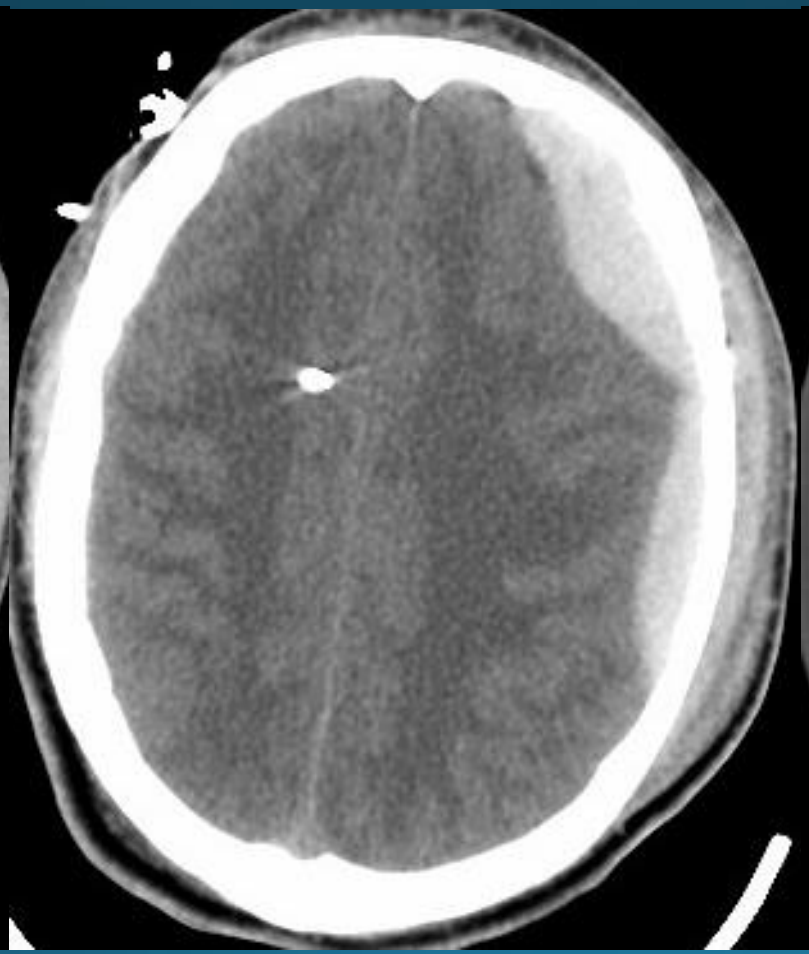
Axial NECT – Bone windows

Acute Epidural Hemorrhage

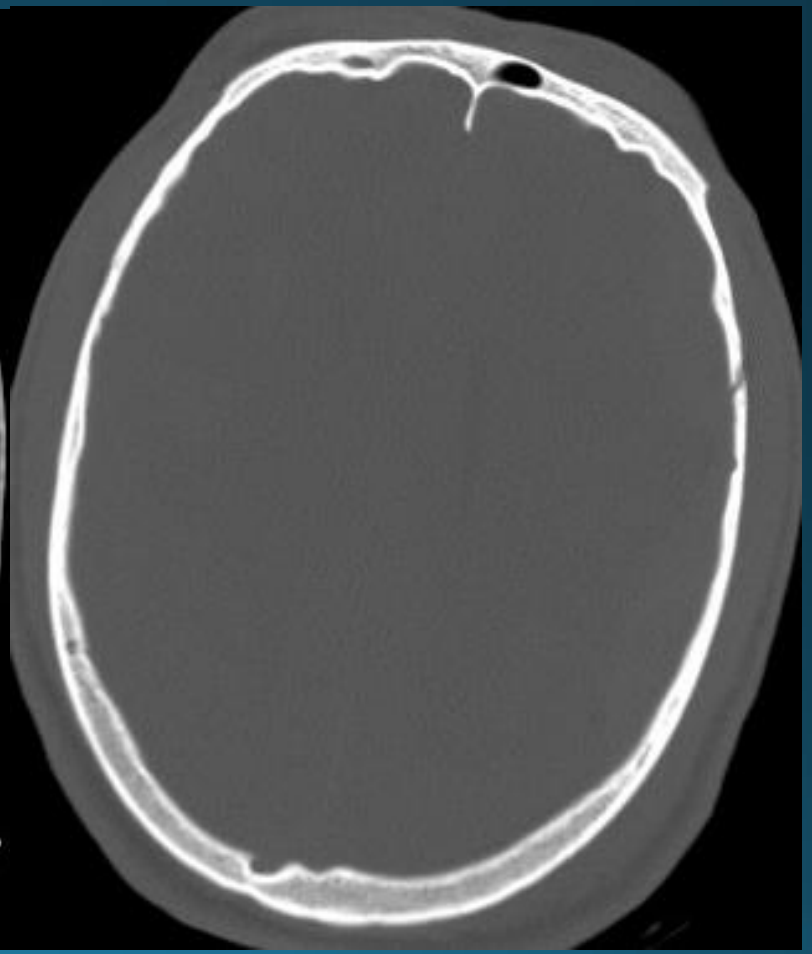
23-year-old ejected from vehicle during MVA, GCS of 3 at scene.



Axial NECT



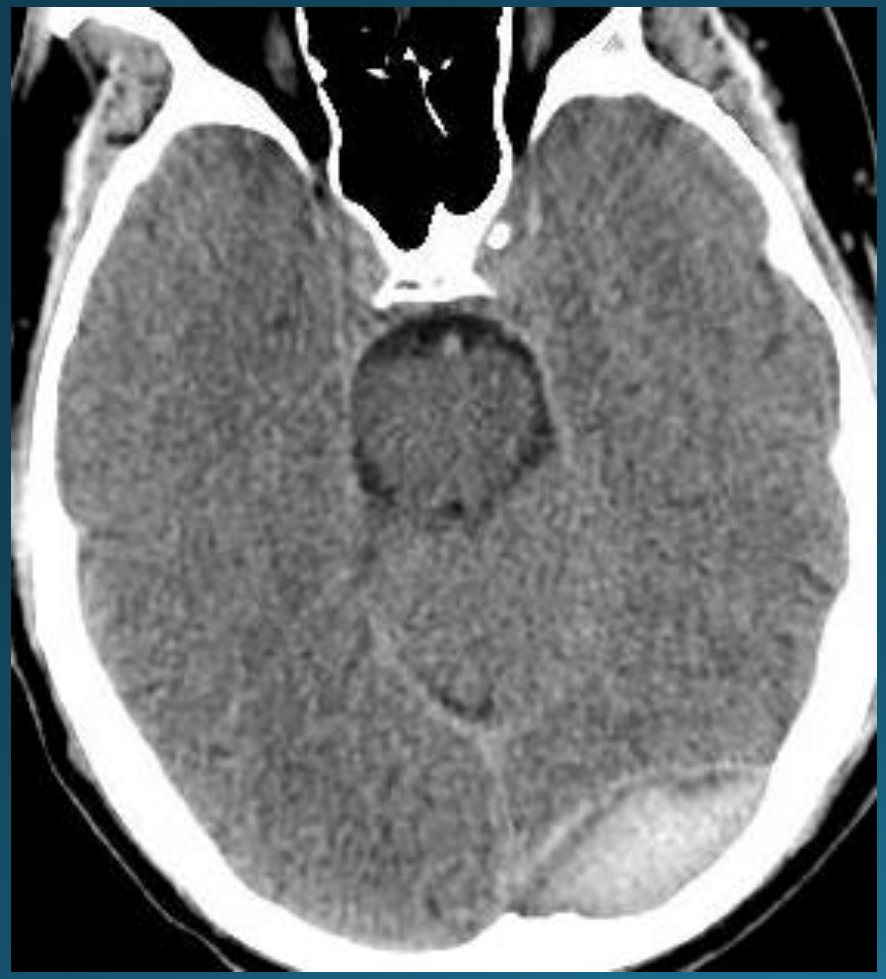
Axial NECT



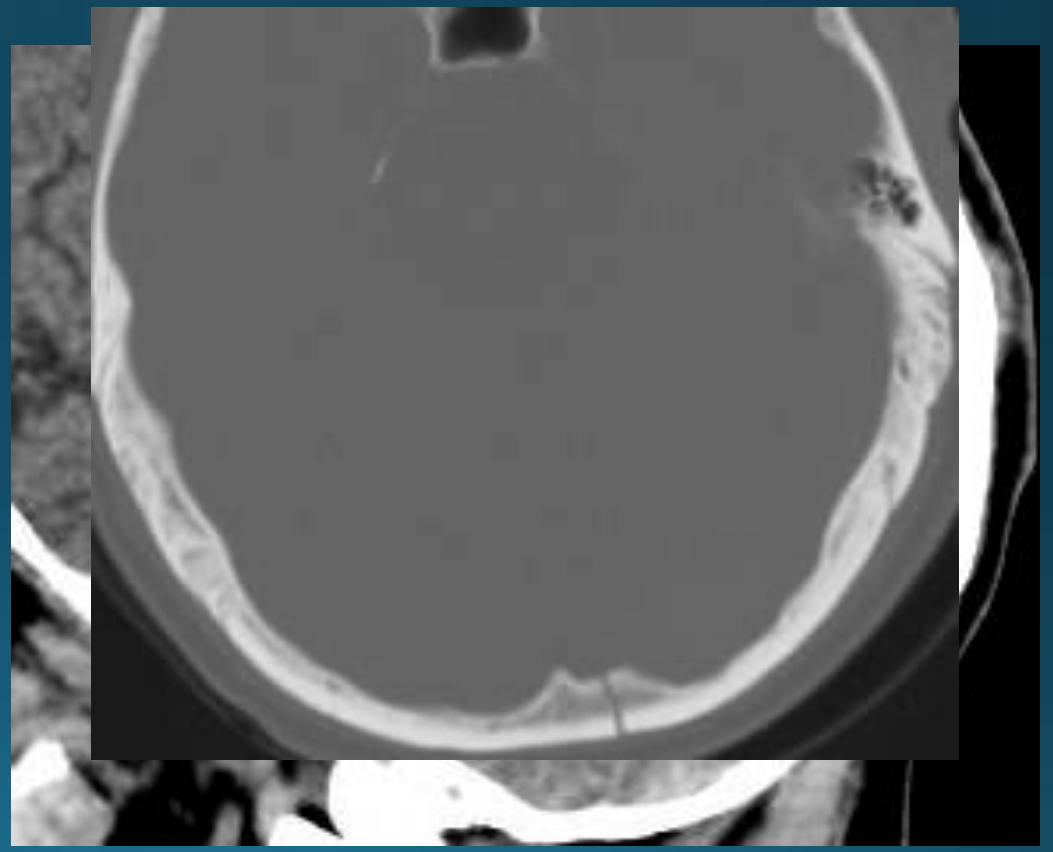
Axial NECT – Bone windows

Acute Epidural Hemorrhage

46-year-old with fall and head trauma.



Axial NECT



Axial NECT Soft Tissue Windows

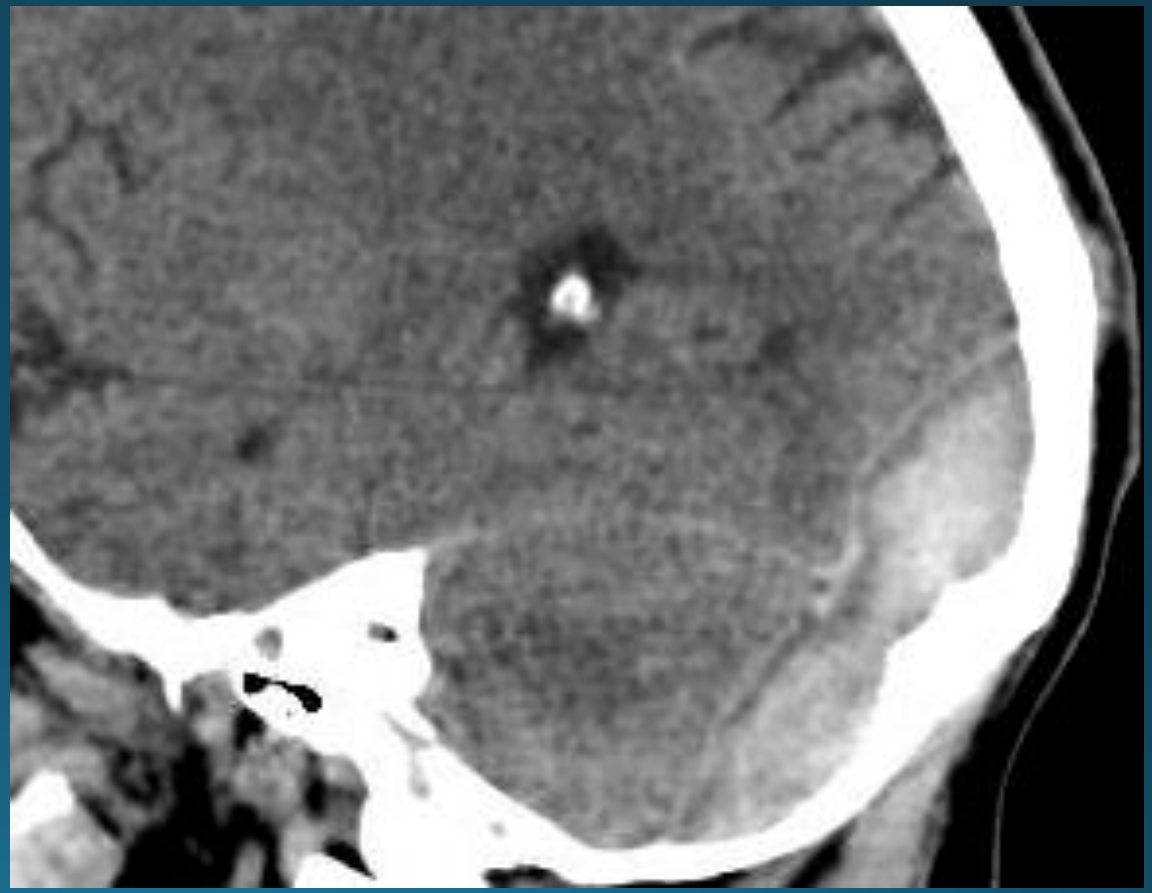
Sagittal NECT Bone Windows

Acute Epidural Hemorrhage

46-year-old with fall and head trauma.

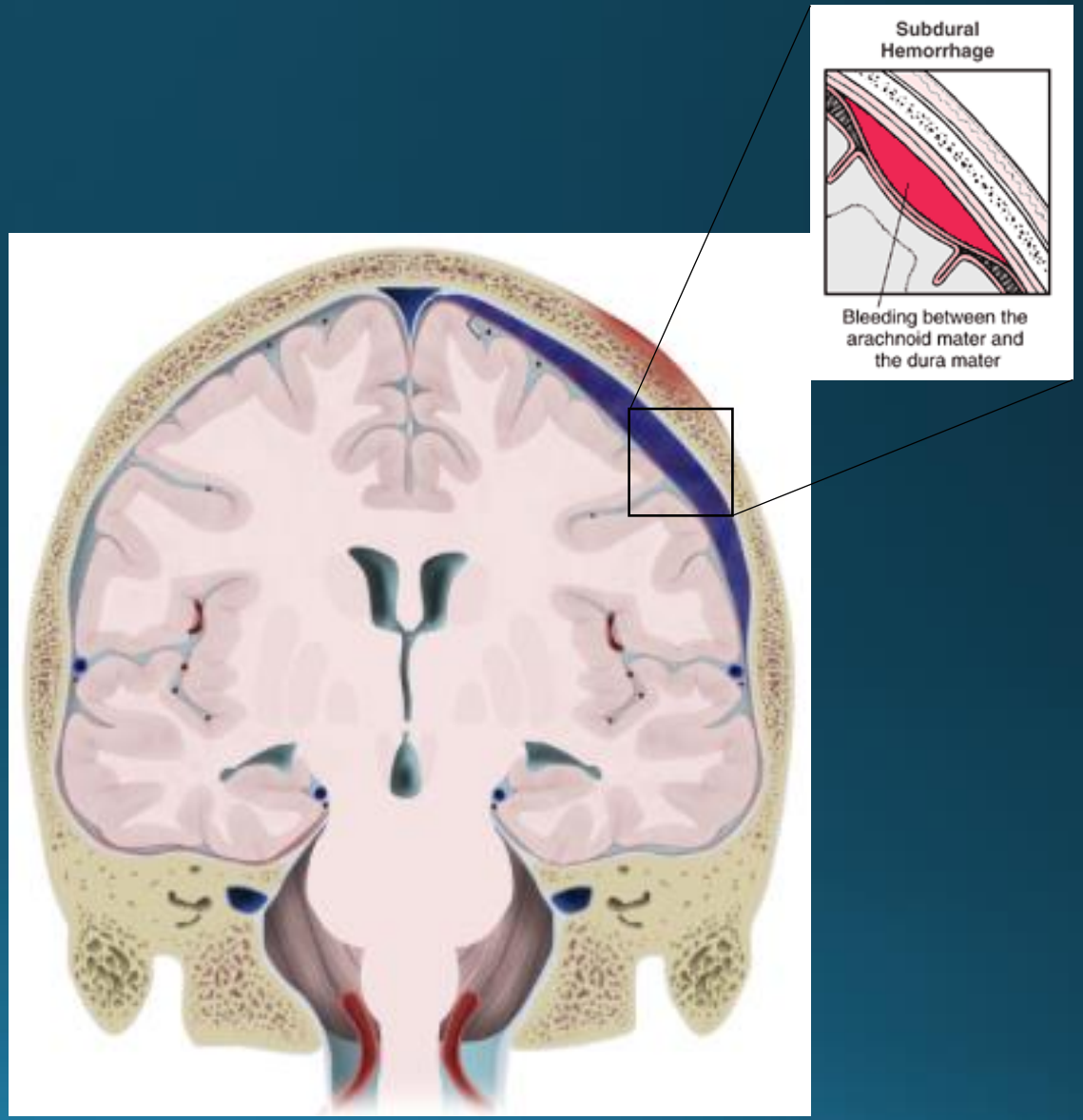
Case Discussion:

- Much less common venous epidural hematoma
- Skull fracture that crosses a dural venous sinus (as in this case)
- Most occur near the vertex (superior sagittal sinus) or skull base (transverse/sigmoid sinus)
- Can "straddle" intracranial compartments, crossing sutures and lines of dural attachment



Acute Subdural Hemorrhage

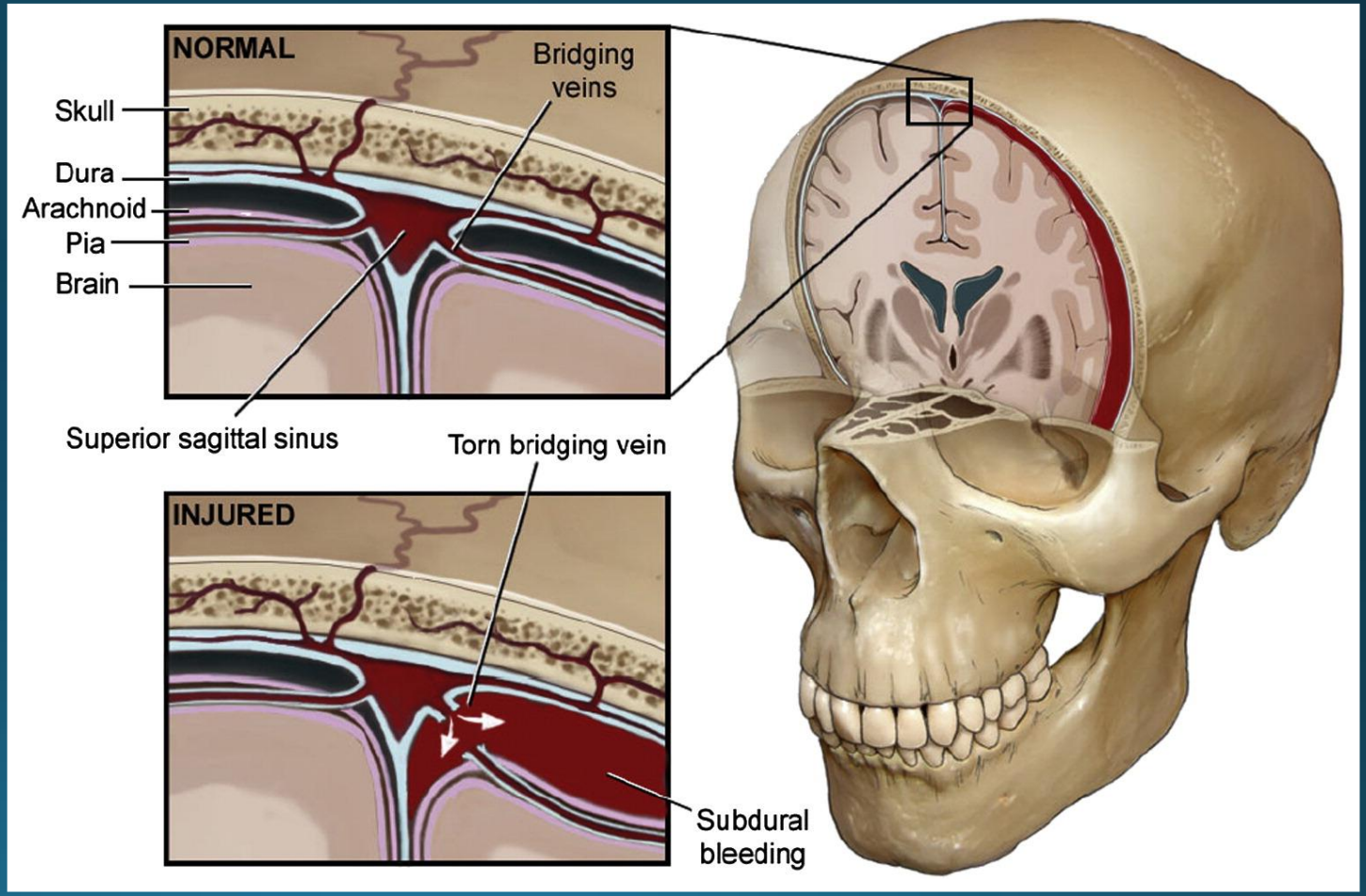
- Blood between the inner layer of the dura and the arachnoid
- Trauma is the most common cause, both direct trauma and non-impact injuries
- One of the leading causes of death and disability in cases of severe traumatic brain injury



Source: http://radiopaedia.org/images/2917400

Acute Subdural Hemorrhage

- Tearing of bridging cortical veins is the most common etiology
- Note the traumatic disruption of the bridging cortical vein traversing the subdural space

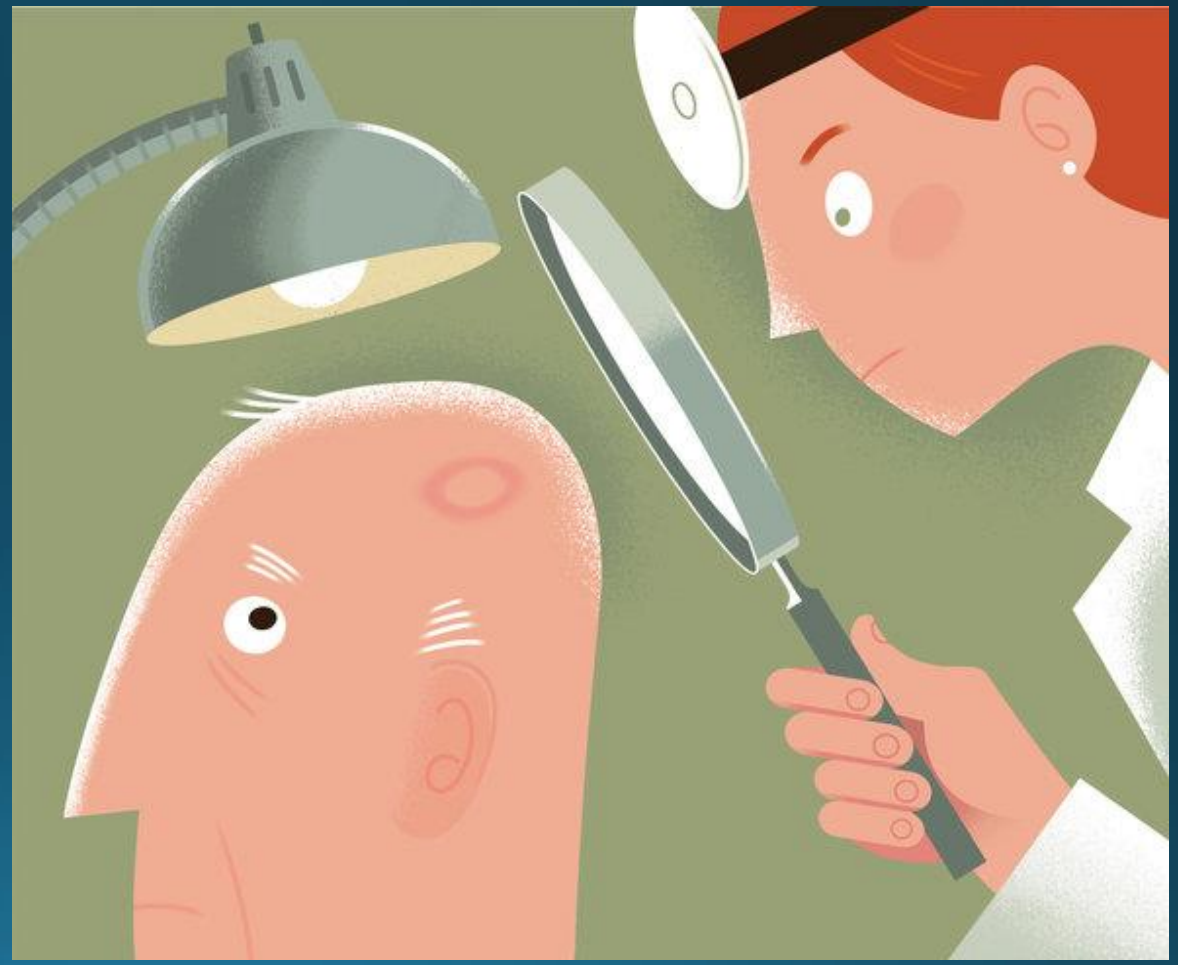


Source: Neuroimaging Clinics of North America 2010; 20: 527-556

Acute Subdural Hemorrhage

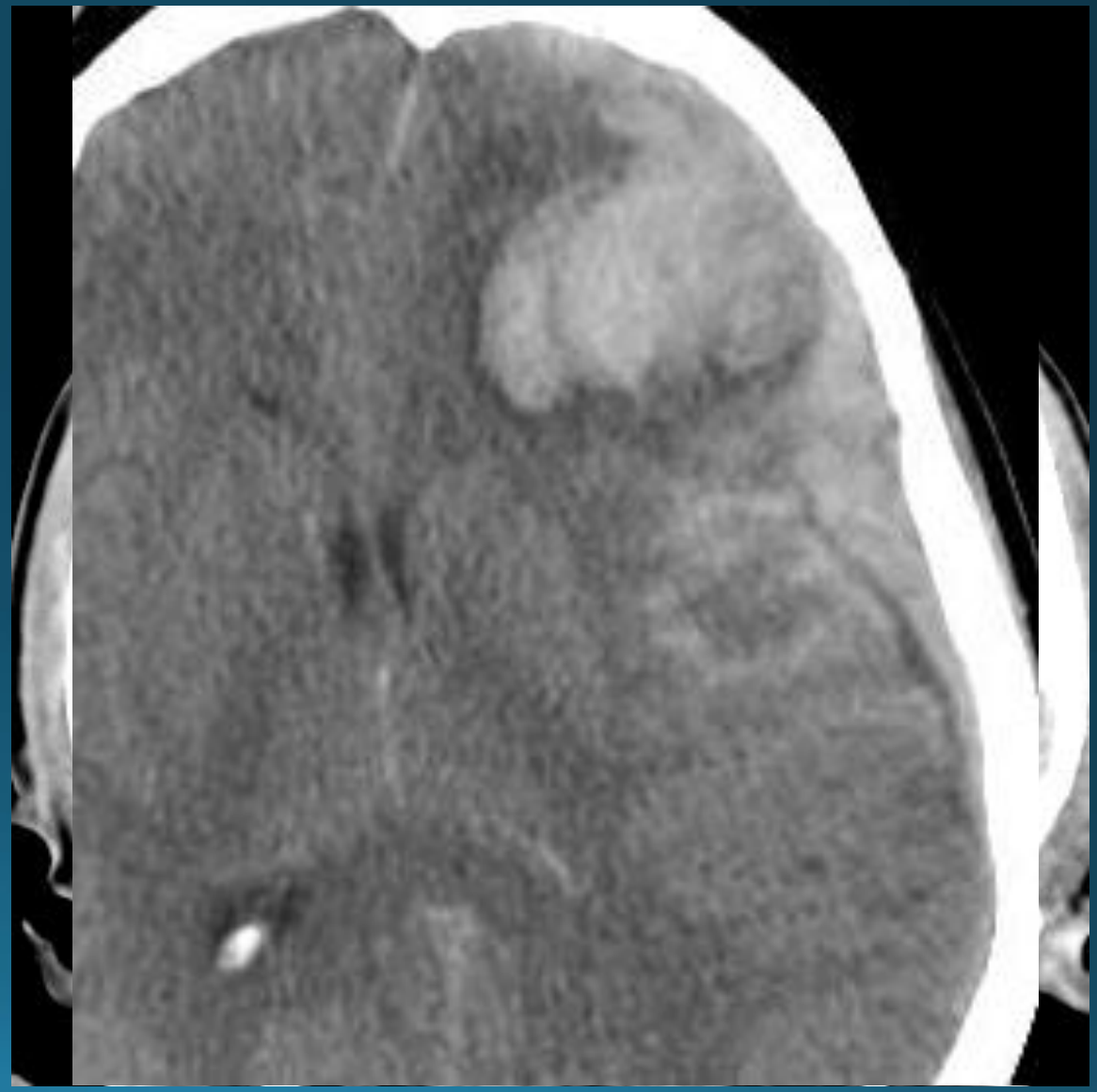
Clinical Issues:

- Second most common extraaxial hematoma, behind SAH
- Found in 10-20% of all patients with head injury and 30% of autopsied fatal injuries
- Commonly associated with other parenchymal injuries such as contusions or DAI
- Symptoms range from none to loss of consciousness and coma
- Natural history varies these may remain stable, grow slowly, or rapidly increase in size
- Treatment options include surgical evacuation or close observation and serial imaging



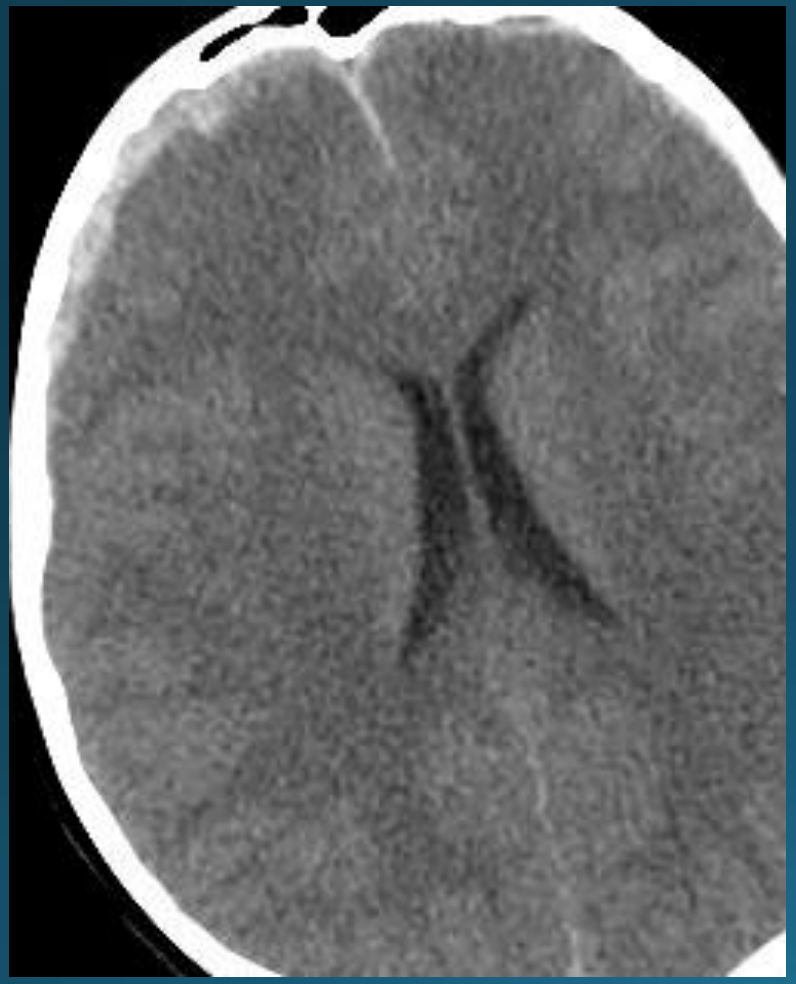
Acute Subdural Hemorrhage

- Crescent-shaped extraaxial collection displacing the grey-white matter interface medially
- Typically more extensive than EDHs, spreading along the falx, tentorium, and around the cranial fossa floors
- May cross suture lines but do not cross dural attachments
- Acute SDHs may be less dense in patients with severe anemia or coagulopathy
- Many SDHs are associated with other traumatic injuries such as subarachnoid hemorrhage, contusions, or DAI

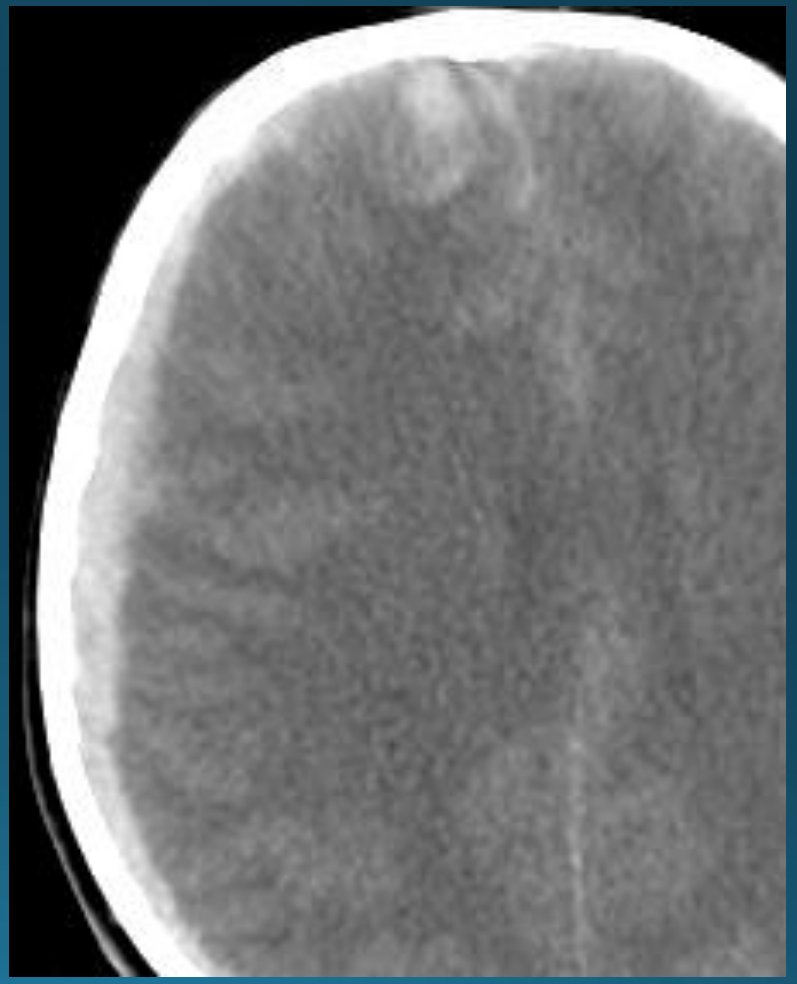


Acute Subdural Hemorrhage

11-year-old passenger in MVC, head trauma.



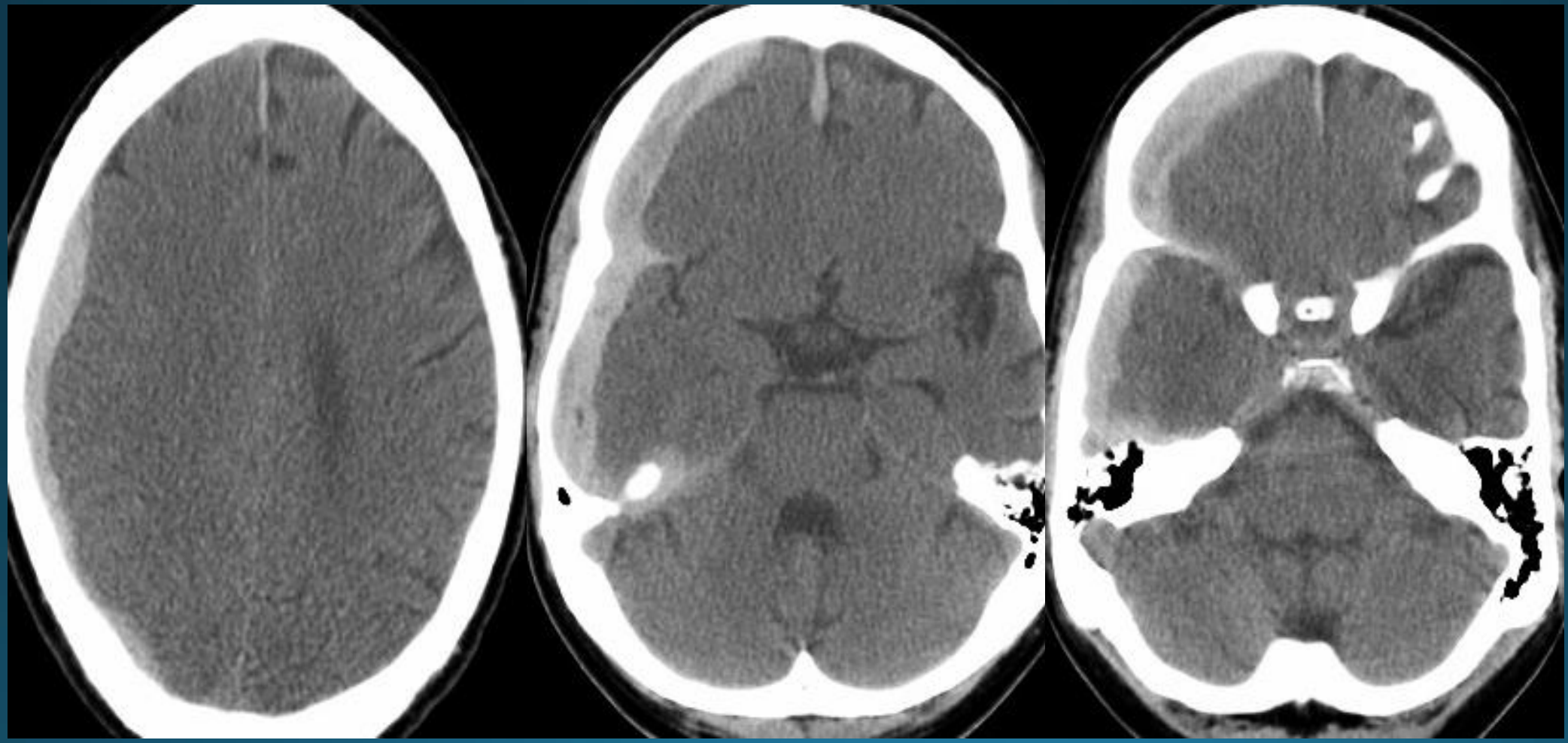
Axial NECT – Initial exam



Axial NECT – 2 hours later

Acute Subdural Hemorrhage

36-year-old in construction accident



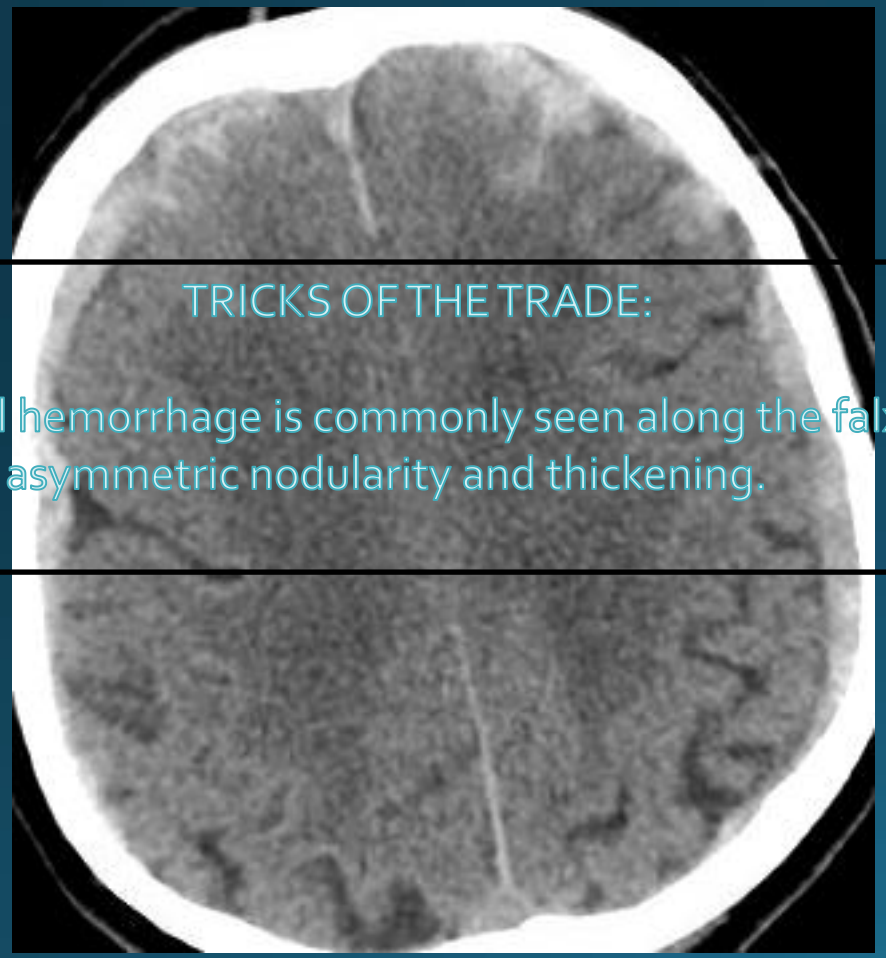
Axial NECT

Axial NECT

Axial NECT

Acute Subdural Hemorrhage

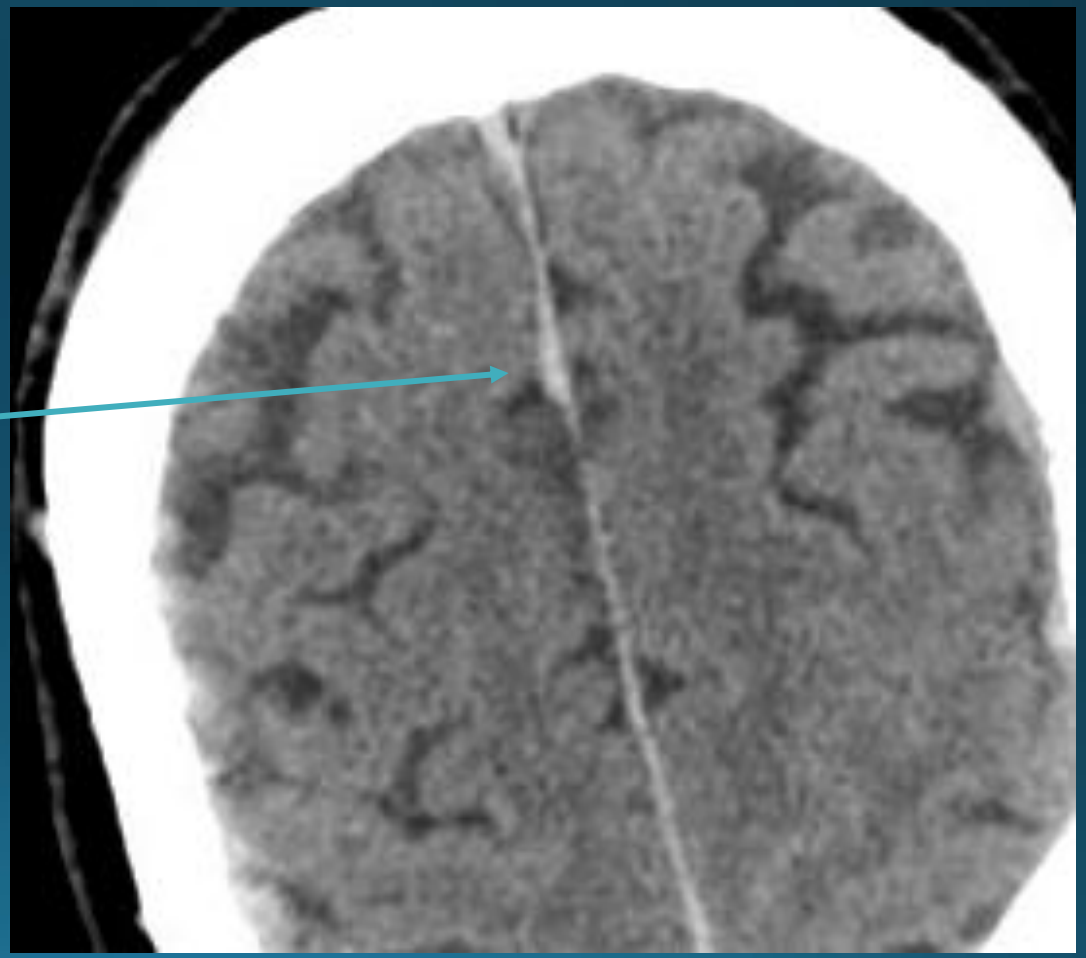
81-year-old with fall and head trauma, on anticoagulation.



TRICKS OF THE TRADE:

Subdural hemorrhage is commonly seen along the falx.
Look for asymmetric nodularity and thickening.

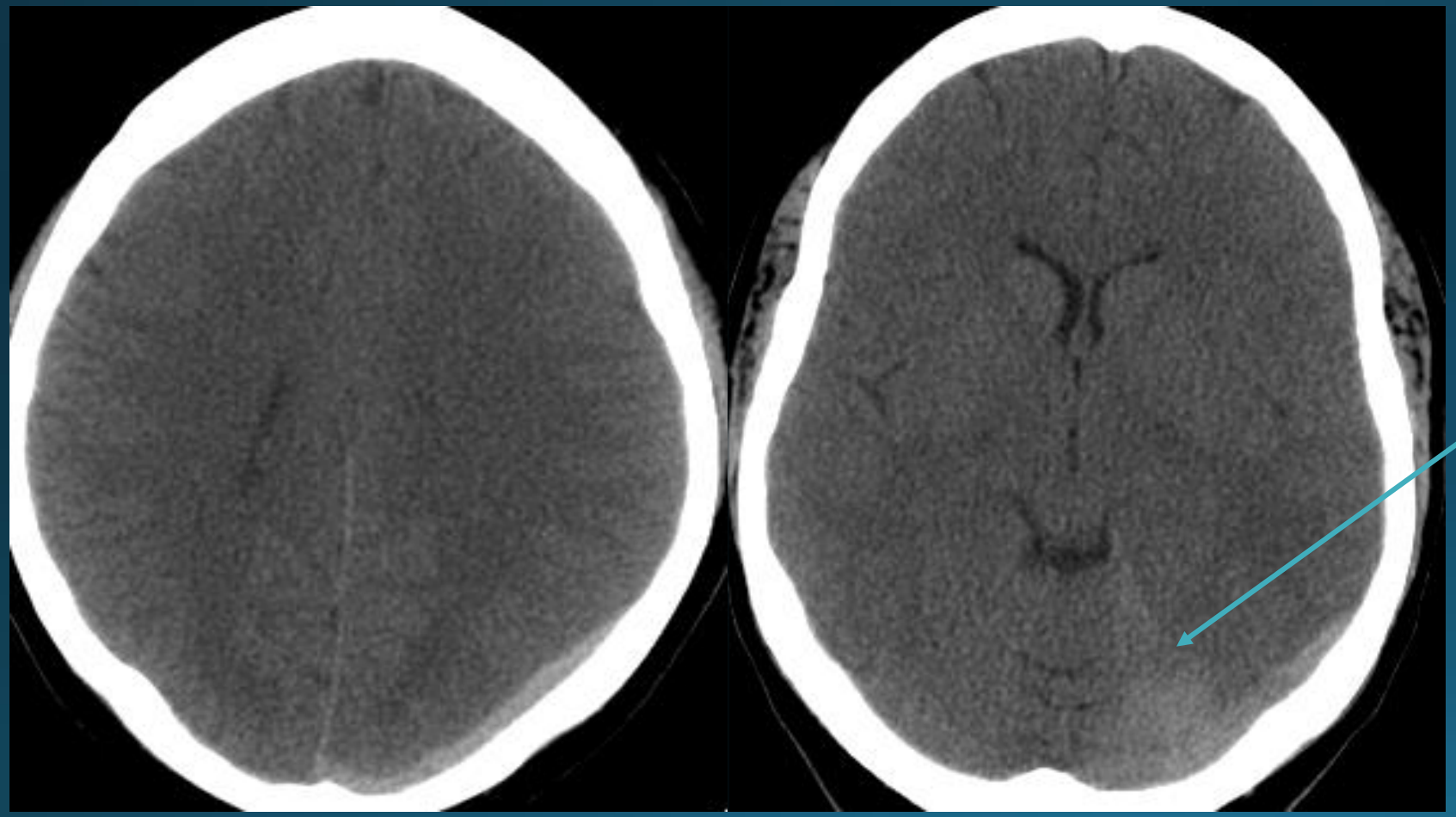
Axial NECT



Axial NECT

Acute Subdural Hemorrhage

52-year-old hit in head with bottle



Axial NECT

Axial NECT

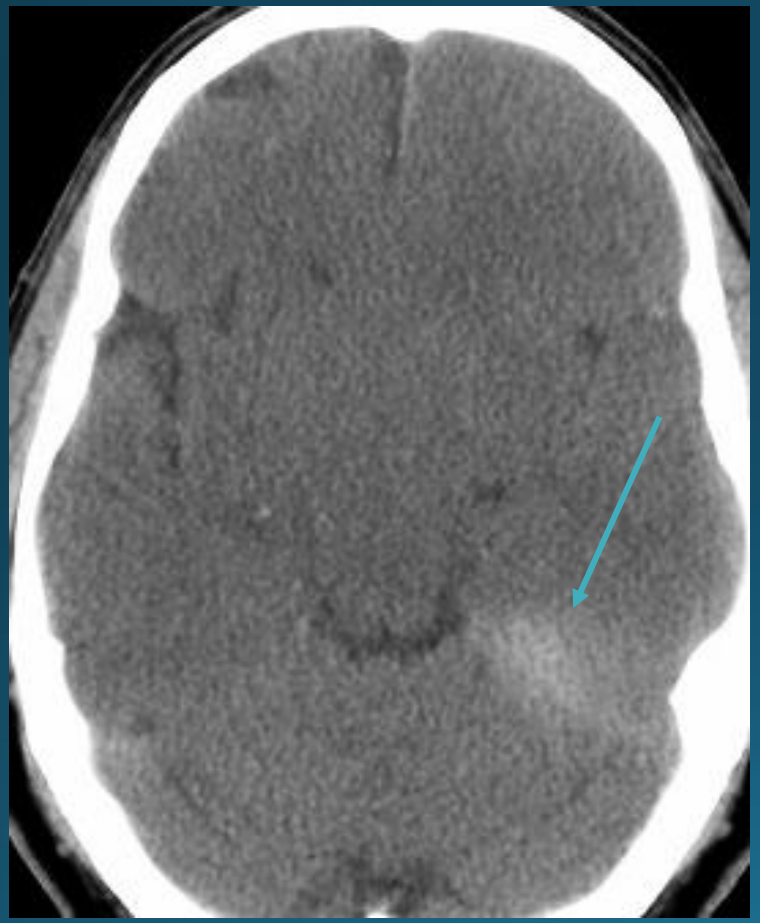
TRICKS OF THE TRADE:

A commonly missed location of subdural hemorrhage is along the tentorial leaflet.

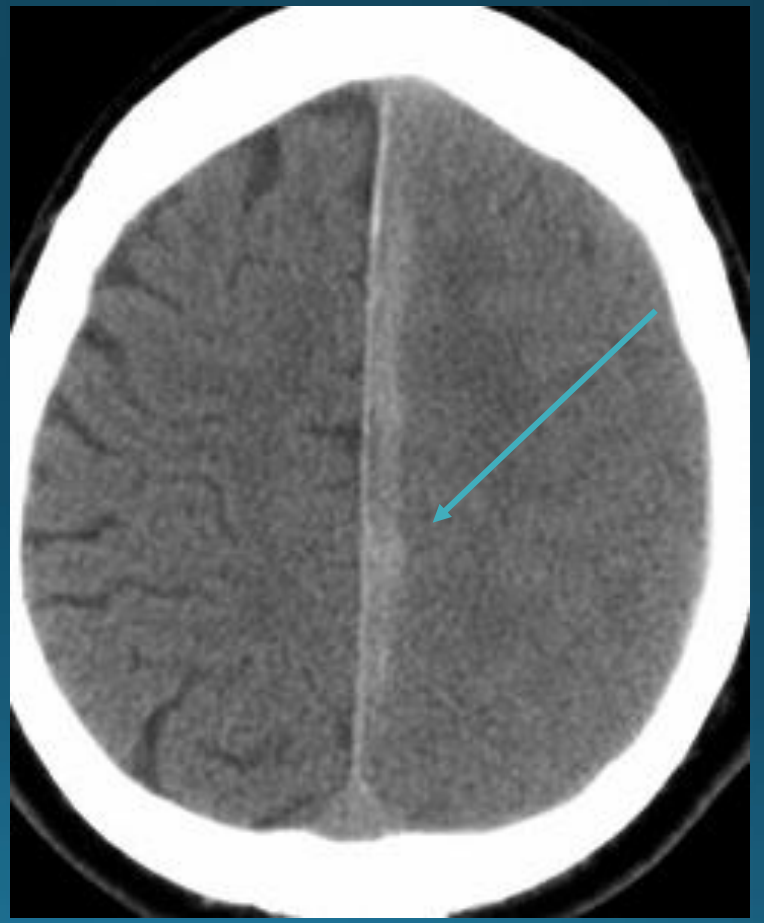
Note the asymmetric density along the left tentorial leaflet.

Acute Subdural Hemorrhage

63-year-old with fall and head trauma



Axial NECT

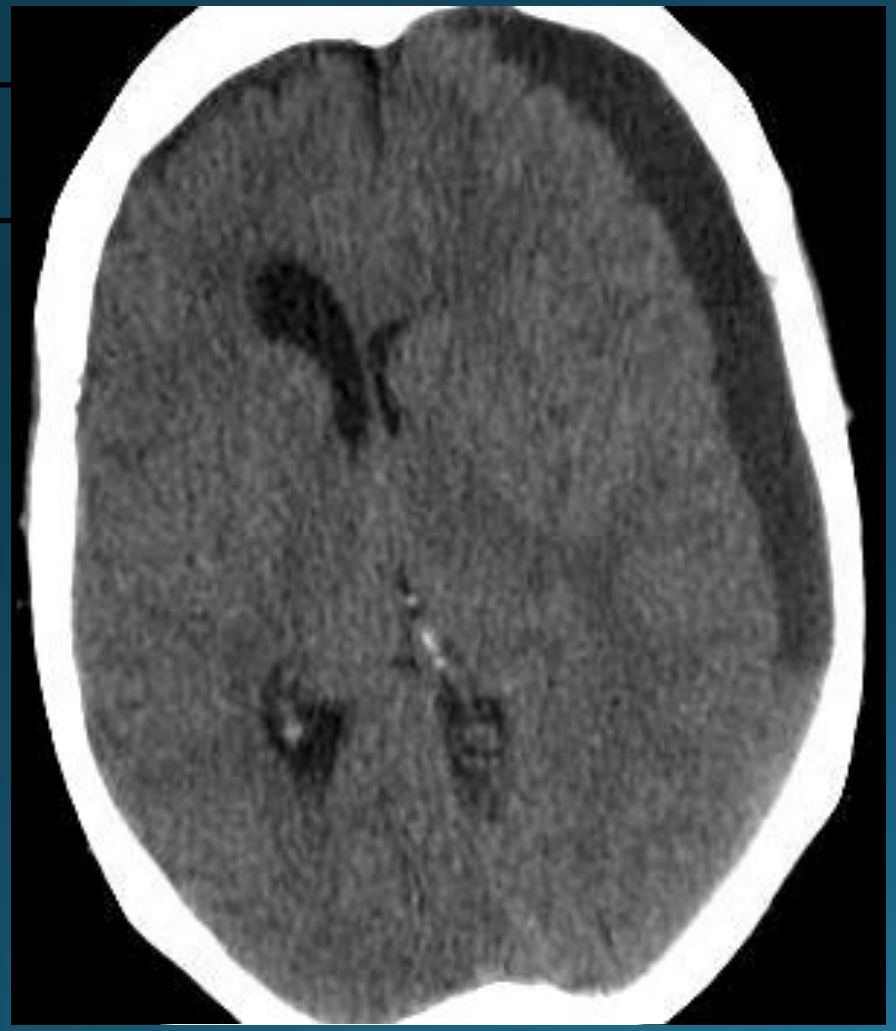


Axial NECT

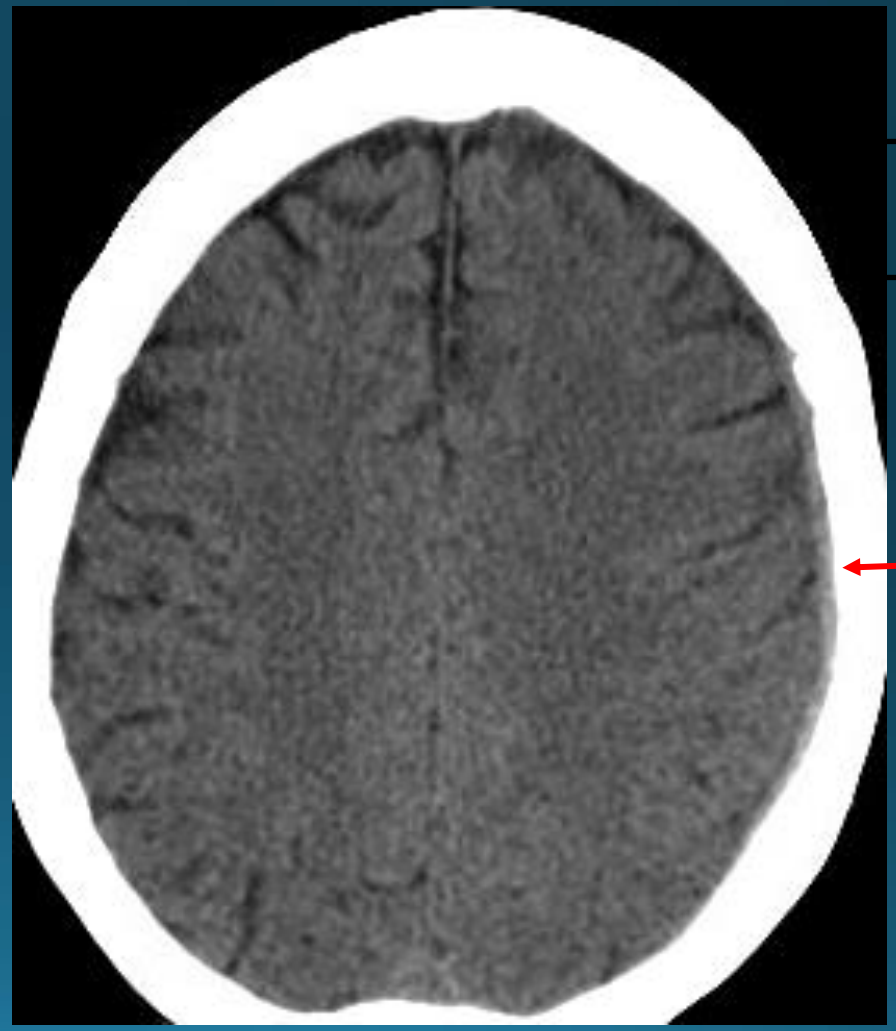
Acute Subdural Hemorrhage

86-year-old with minor head trauma.

2 months later



Axial NECT

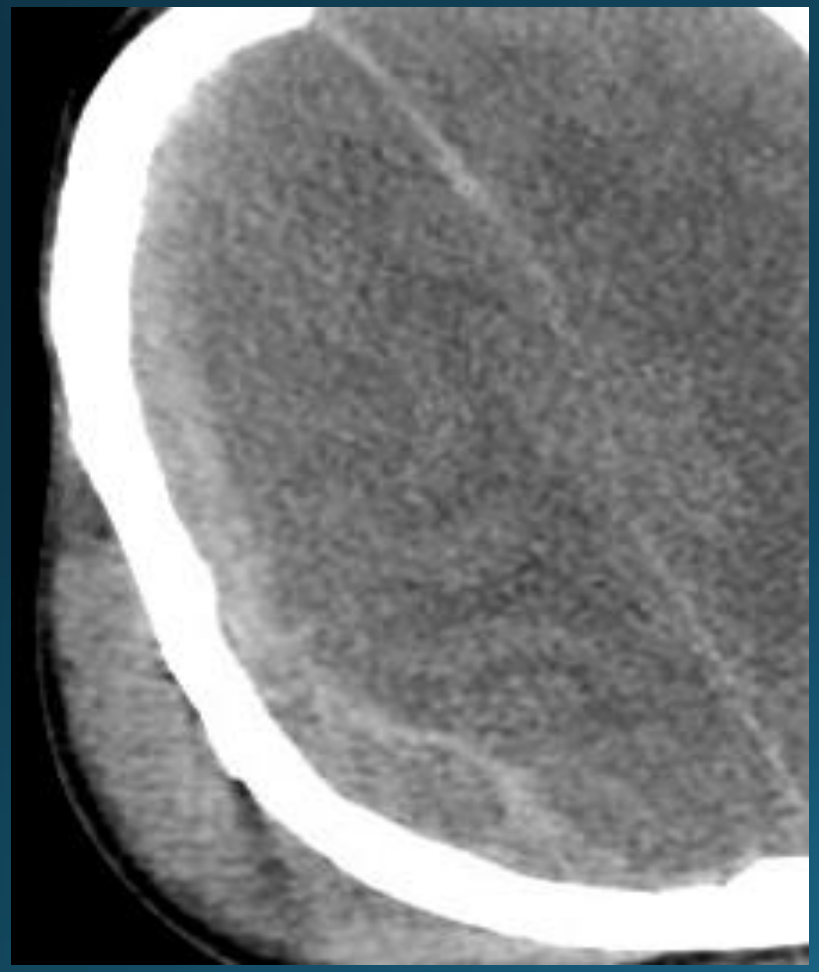


Axial NECT

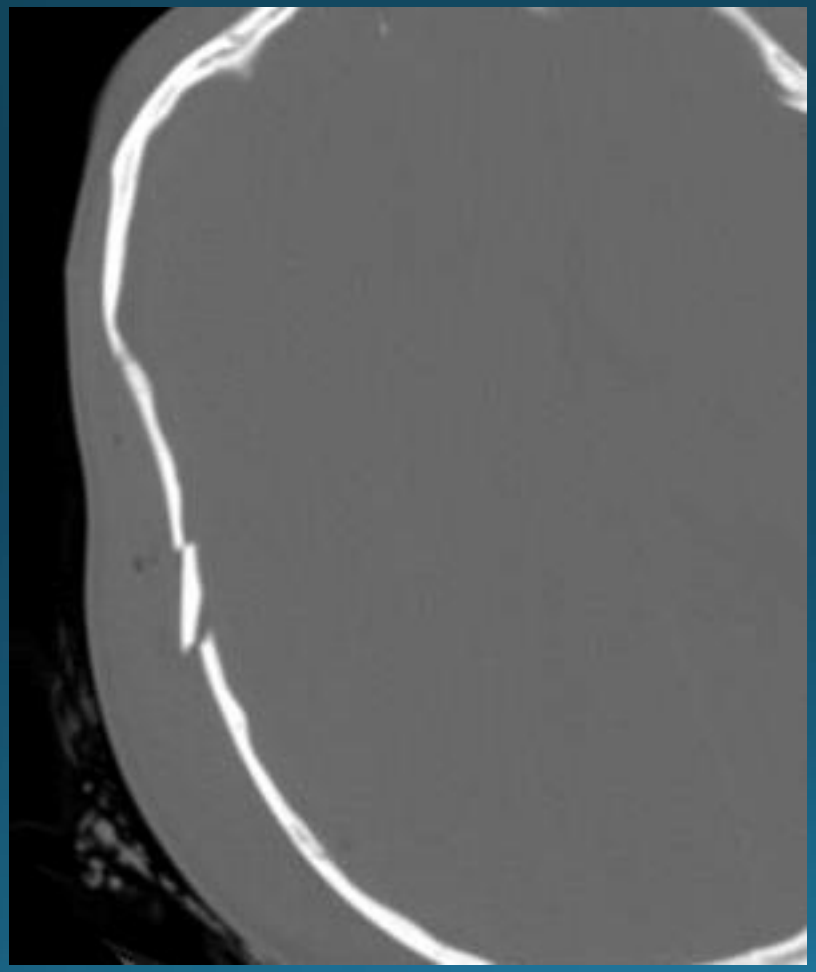
Called normal.

Acute Subdural Hemorrhage

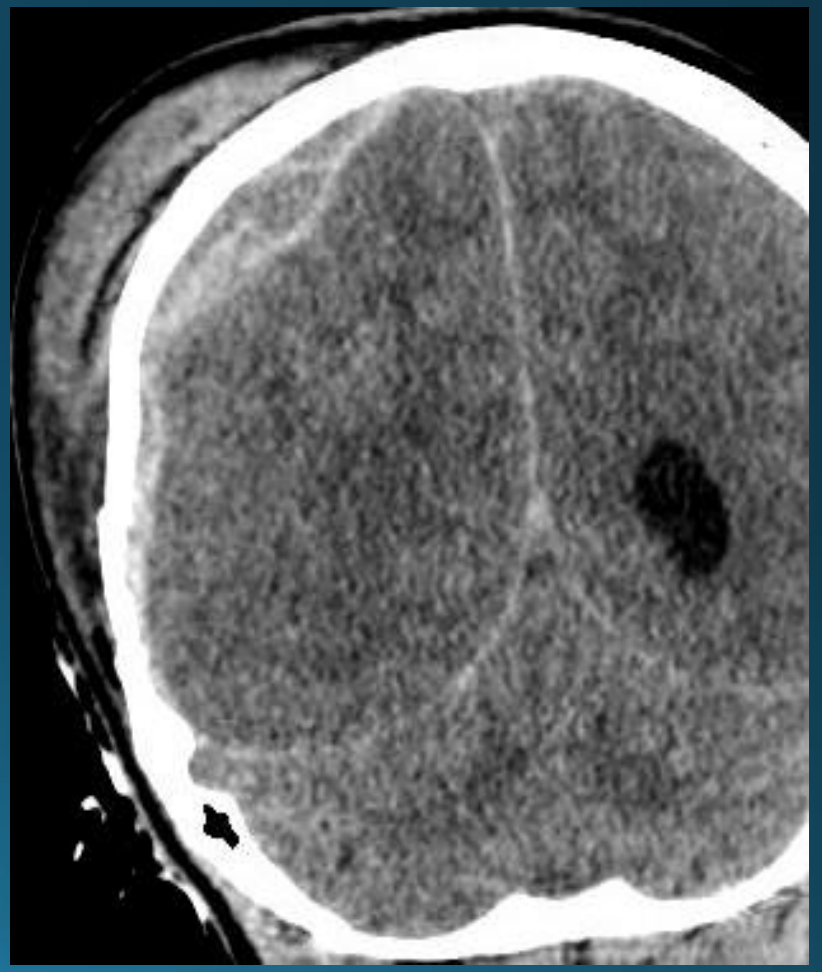
20-year-old unrestrained driver in MVC, severe head injury and GCS 4 at scene.



Axial NECT



Axial NECT - Bone windows



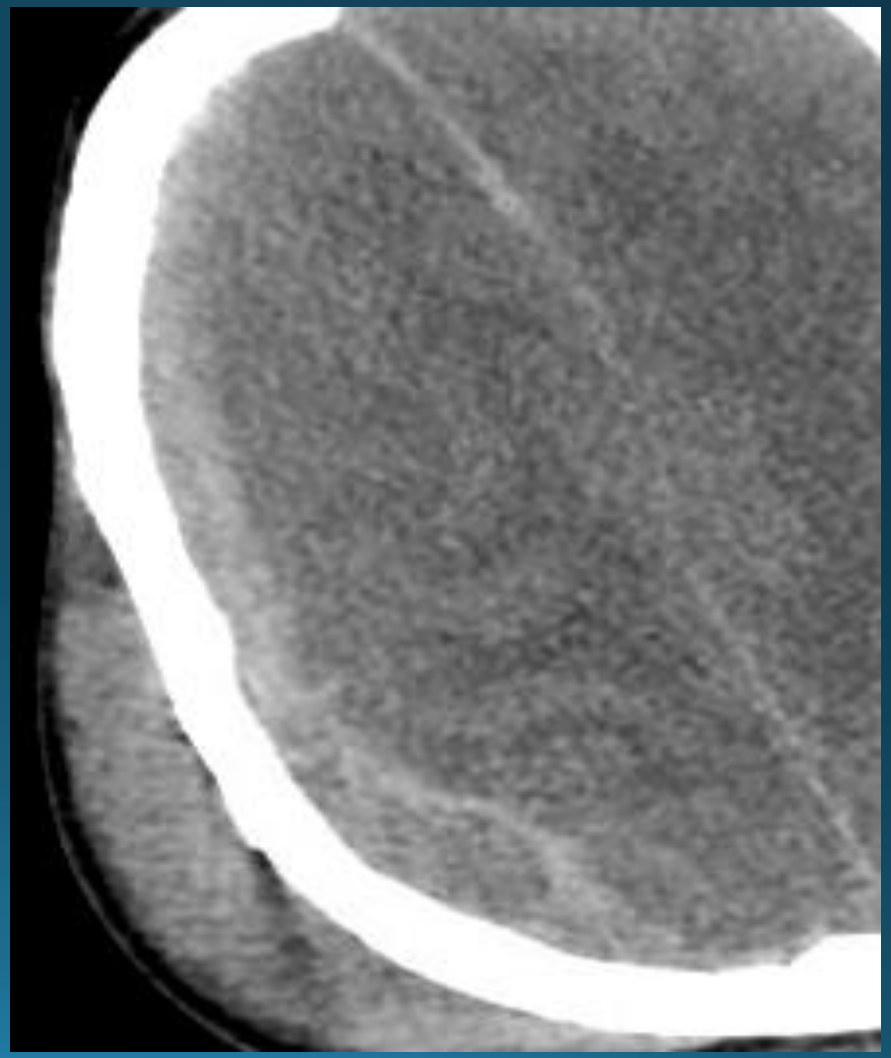
Coronal NECT

Acute Subdural Hemorrhage

20-year-old unrestrained driver in MVC, severe head injury and GCS 4 at scene.

Case Discussion:

- This case is likely an example of a mixed subdural and epidural hematoma
- The presence of a comminuted skull fracture and lentiform shaped collection (best seen on coronal) point toward the presence of an epidural hemorrhage
- The heterogeneous, low density within the extraaxial collections suggests active/rapid bleeding
- The patient ultimately died as a result of her injuries



Subacute / Chronic Subdural Hemorrhage

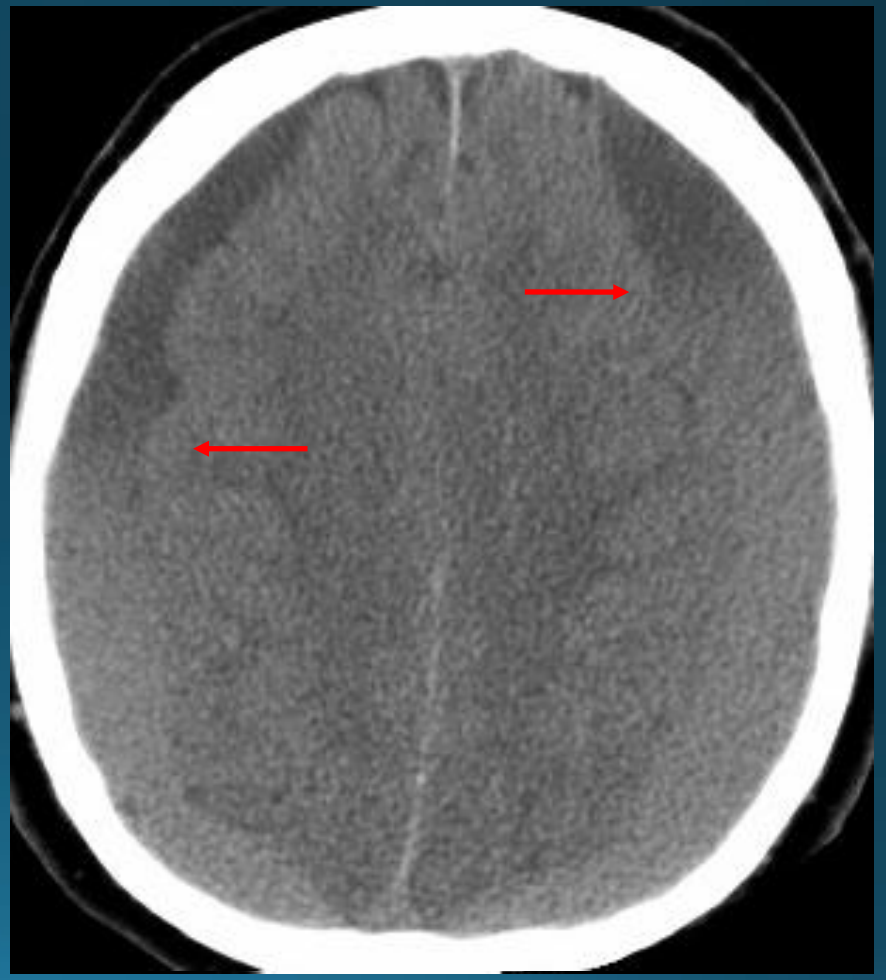
Introduction:

- Over time, SDHs undergo organization, lysis, and "neomembrane" formation (granulation tissue)
- In some cases, repetitive hemorrhages of different ages may be present
- Clinical symptoms vary from asymptomatic to acute neurologic change due to rehemorrhage

Subacute / Chronic Subdural Hemorrhage

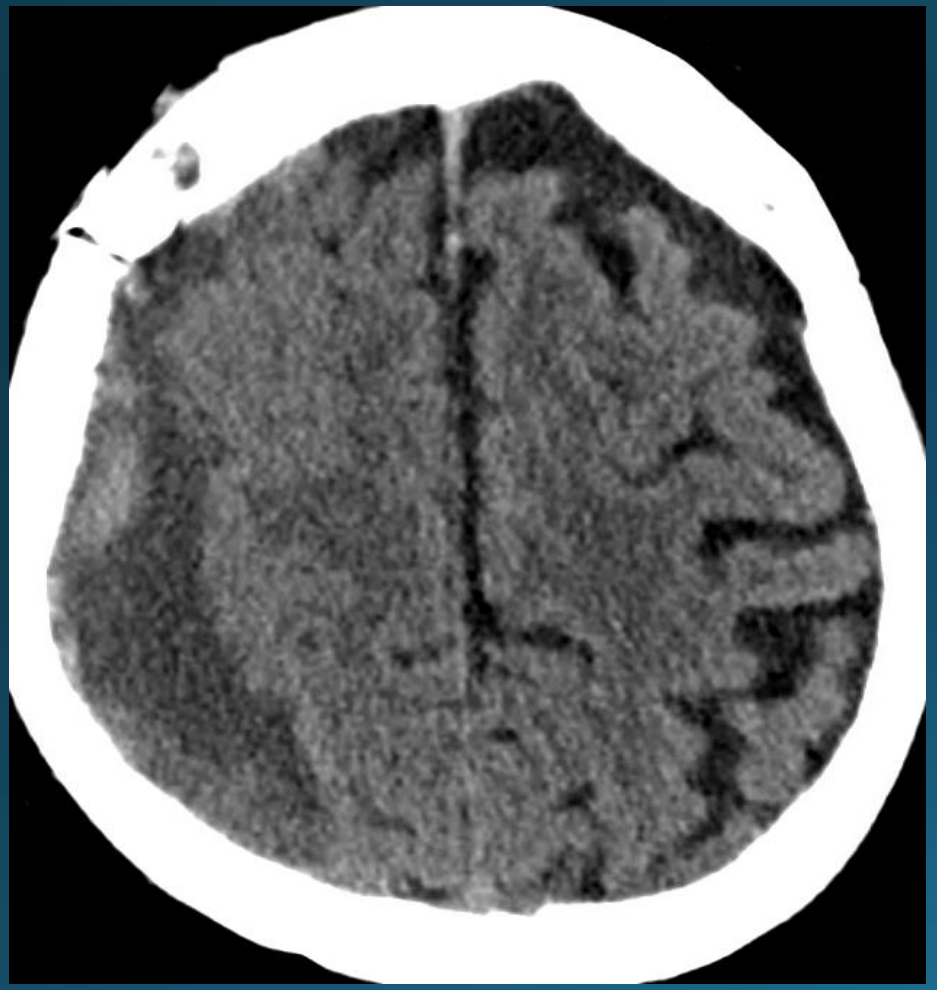
Imaging:

- SDHs will become nearly isodense with the underlying cortex within several days following trauma
- These collections may be difficult to detect due to similar attenuation to adjacent brain parenchyma but will result in "buckling" of the grey-white interface
- Rehemorrhage into chronic SDHs results in mixed attenuation collections, often multiloculated/septated with layering "hematocrit" levels (old blood on top, new blood on bottom)

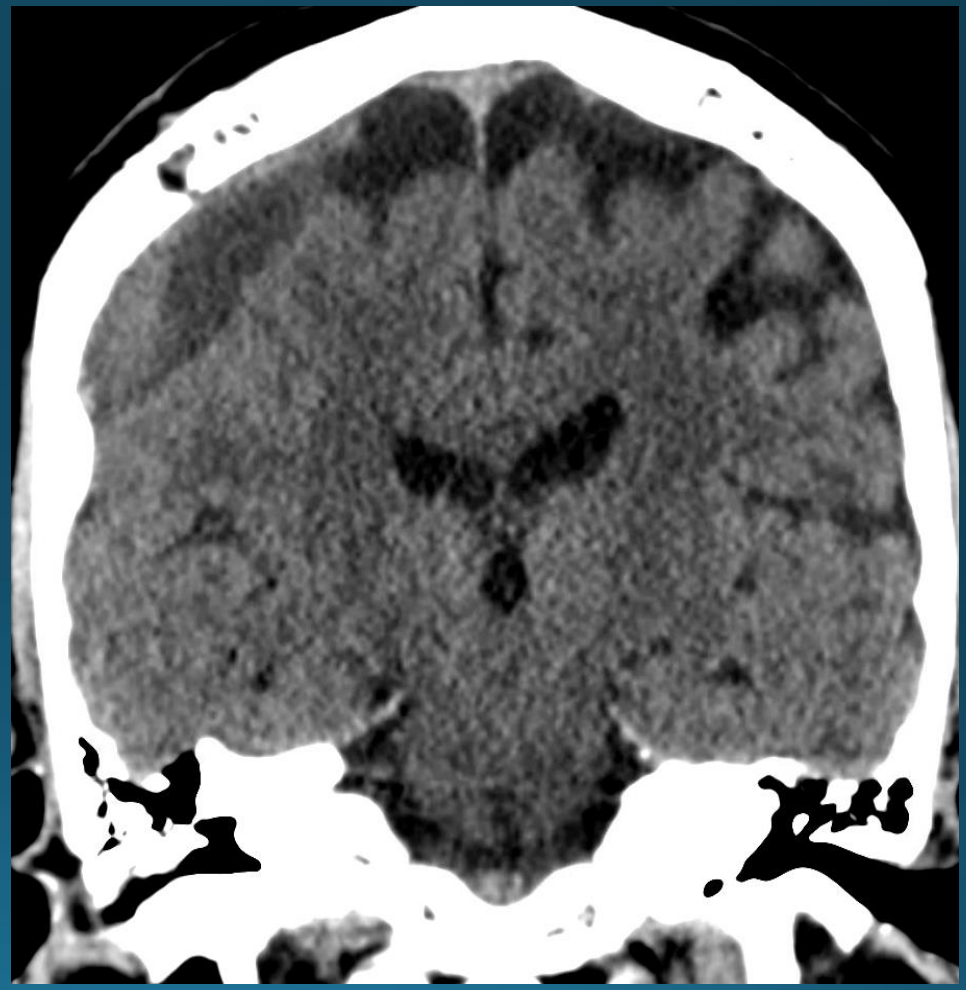


Subacute / Chronic Subdural Hemorrhage

83-year-old with headache, history of prior traumatic subdural hematoma



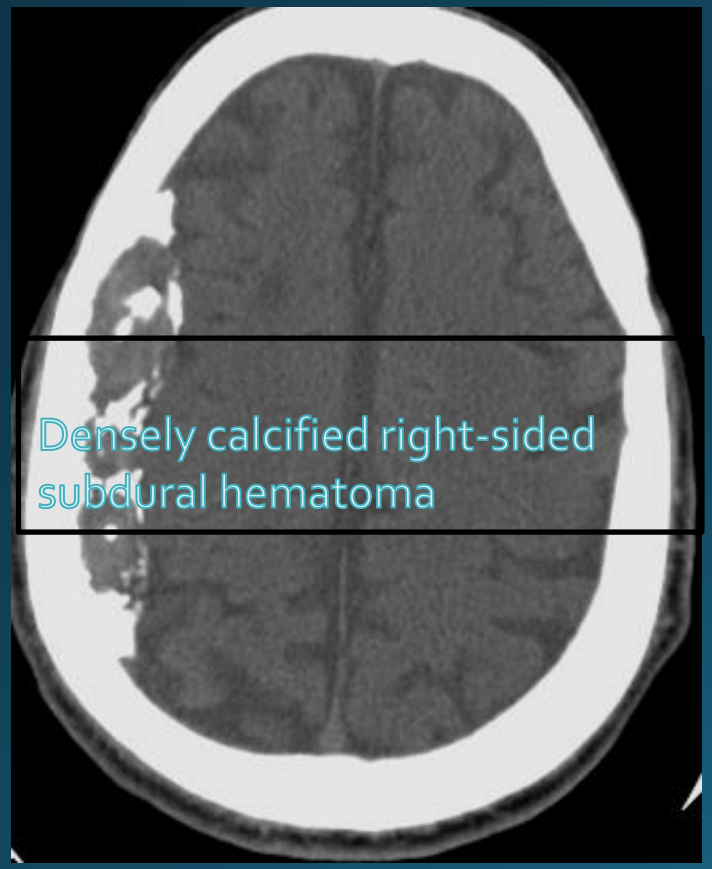
Axial NECT



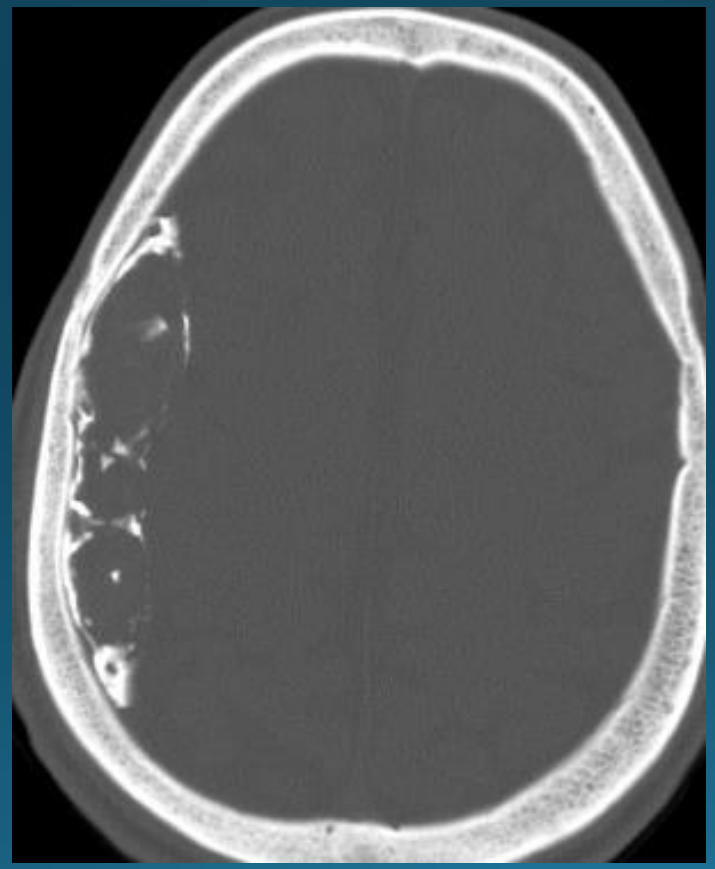
Coronal NECT

Subacute / Chronic Subdural Hemorrhage

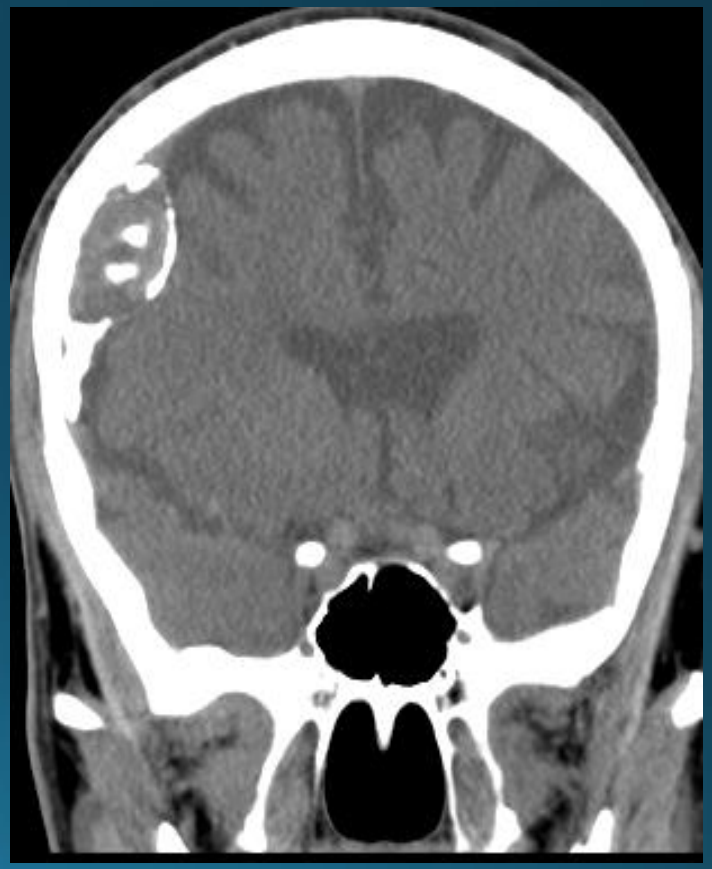
83-year-old with history of multiple intracranial shunts and drains



Axial NECT



Axial NECT – Bone windows

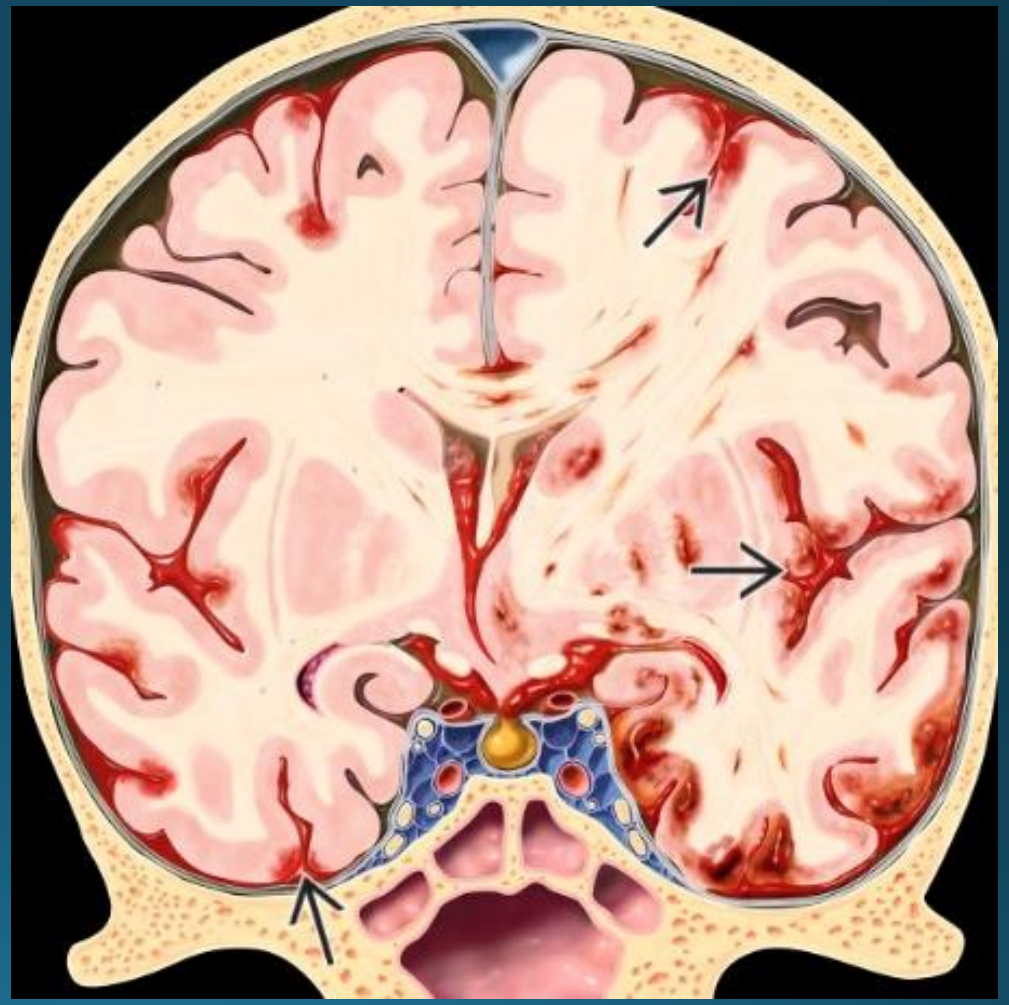


Coronal NECT

Traumatic Subarachnoid Hemorrhage

Introduction:

- Most common cause of intracranial subarachnoid hemorrhage
- Can occur with both direct trauma and non-impact closed head injury
- Occasionally occurs in isolation but often accompanied by other manifestations of brain injury
- Most common locations include perisylvian regions, anteroinferior frontal and temporal sulci, and over the hemispheric convexities

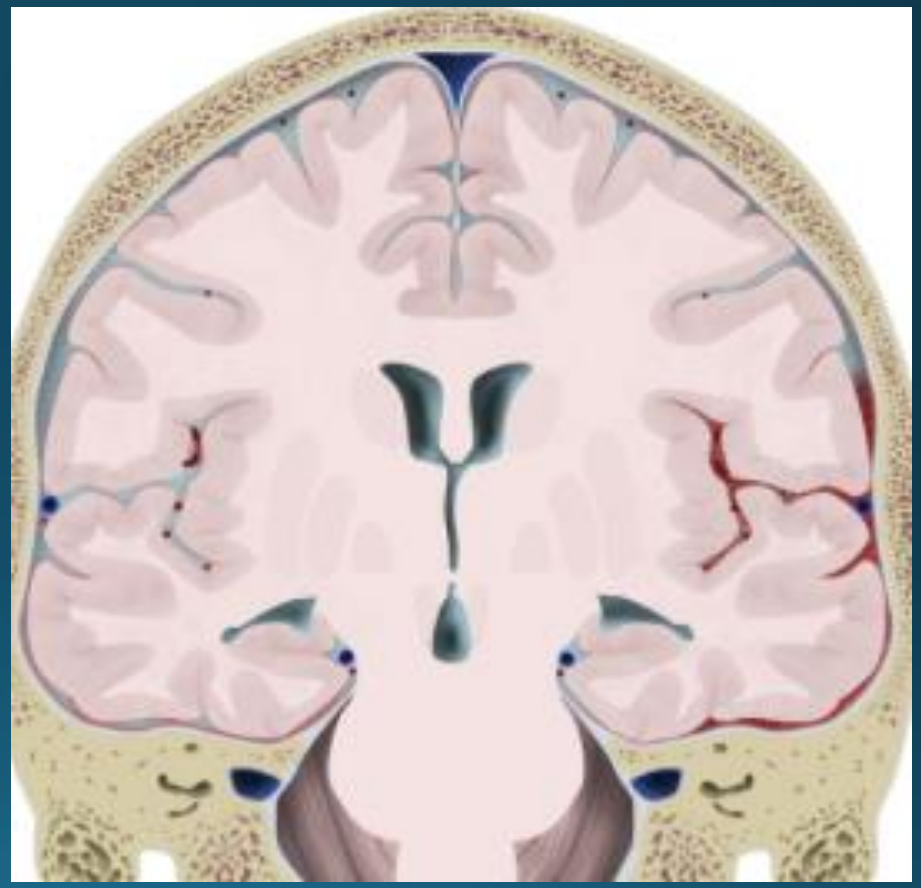


Source: http://my.statdx.com

Traumatic Subarachnoid Hemorrhage

Clinical Issues:

- Bimodal distribution, most commonly occurs in young adults (M>F) and the elderly
- Symptoms are primarily related to other traumatic injuries
- May result in delayed vasospasm and secondary ischemic symptoms
- Supportive therapy is the primary treatment
- Nimodipine or other calcium channel blockers (verapamil) may prevent vasospasm and its complications

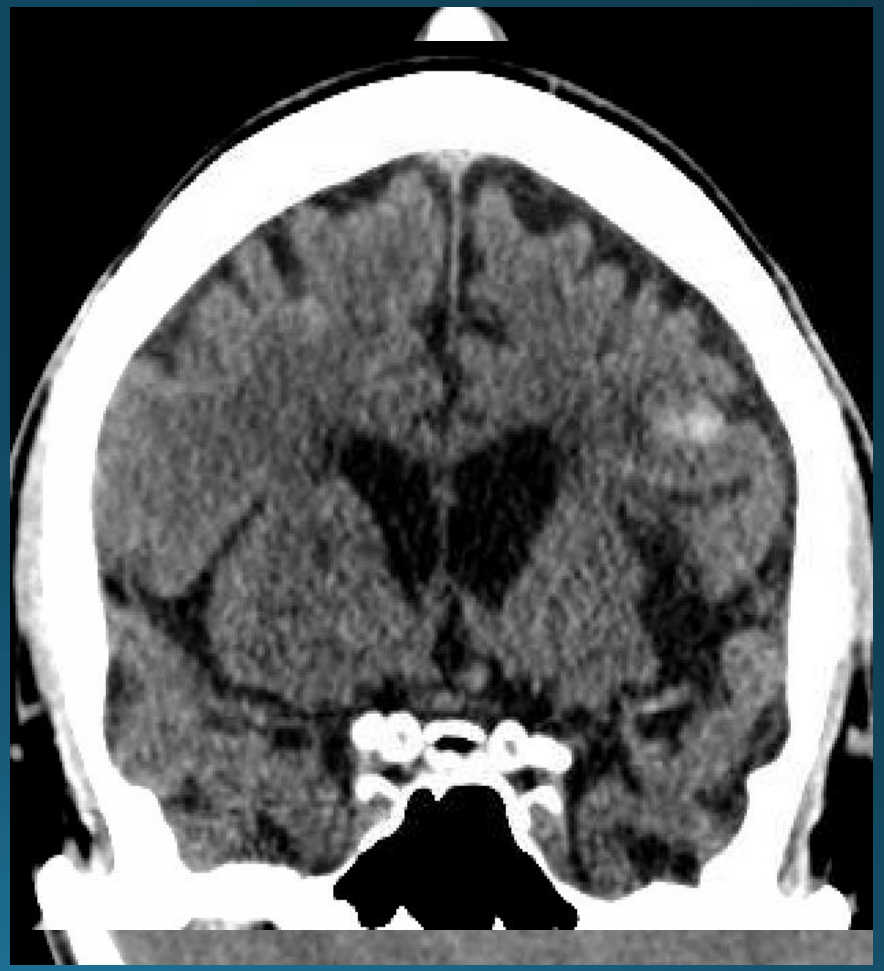


Source: http://www.radiopaedia.org

Traumatic Subarachnoid Hemorrhage

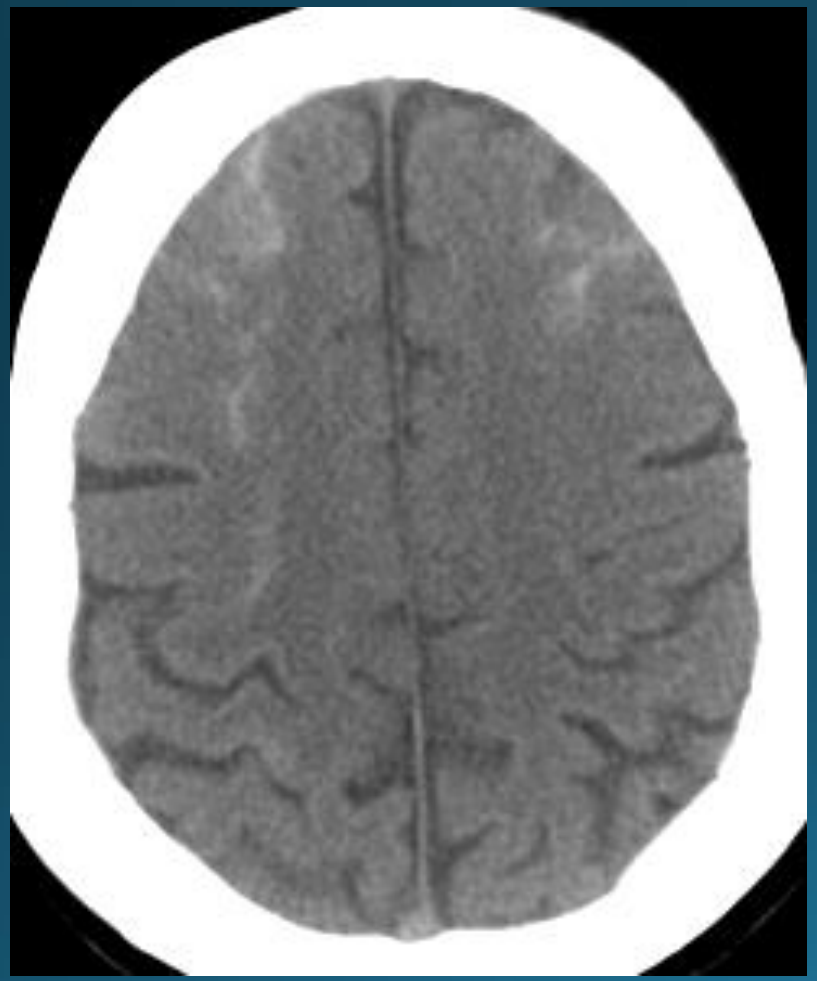
Imaging:

- Sulcal-cisternal hyperdensity
- Typically more focal or patchy than the diffuse SAH blood indicative of aneurysmal hemorrhage
- Often seen adjacent to cortical contusions or under epi- or subdural hematomas
- Isolated SAH may be seen within the interpeduncular fossa or a single convexity sulcus
- Chronic SAH may appear as hypodense fluid that expands the affected sulci
- DDx: aneurysm rupture, perimesencephalic non-aneurysmal hemorrhage, blood from AVM or other vascular malformation

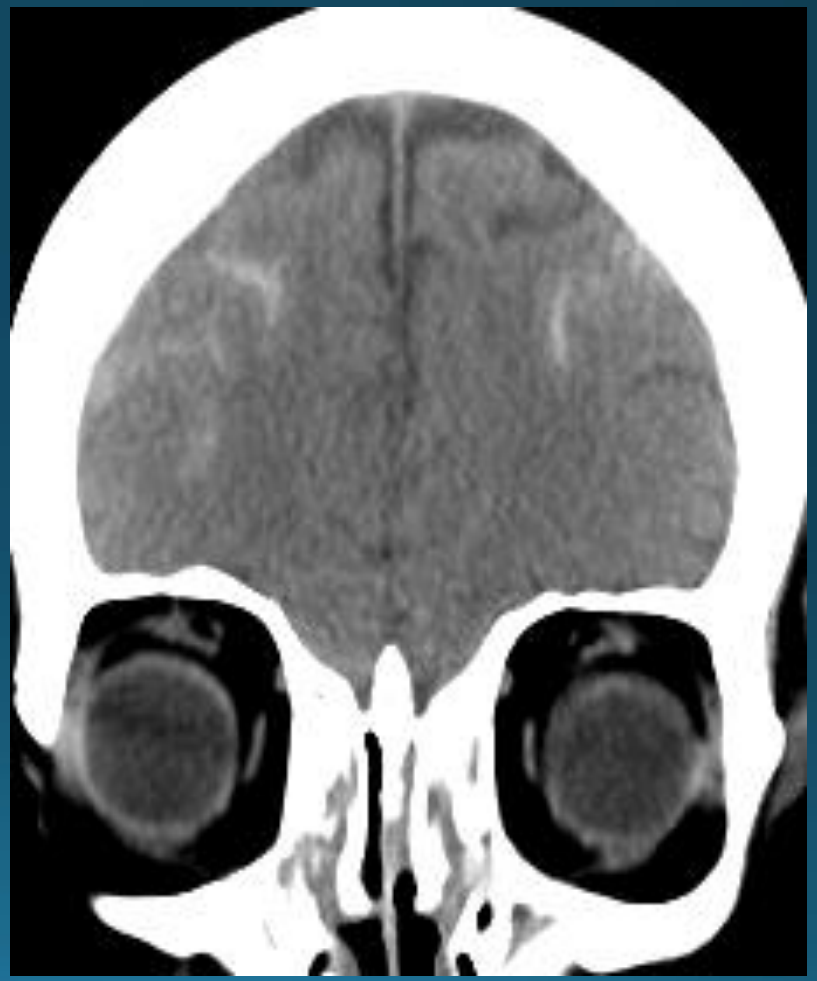


Traumatic Subarachnoid Hemorrhage

50-year-old with fall from ladder, head trauma, and headache



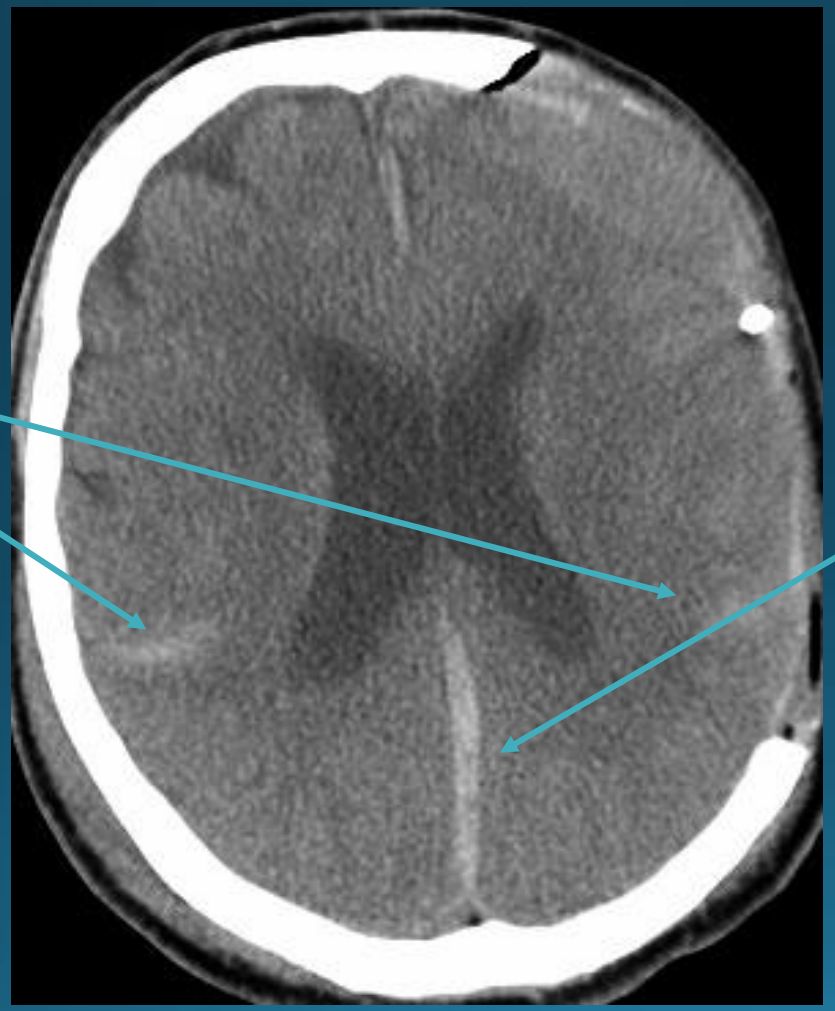
Axial NECT



Coronal NECT

Traumatic Subarachnoid Hemorrhage

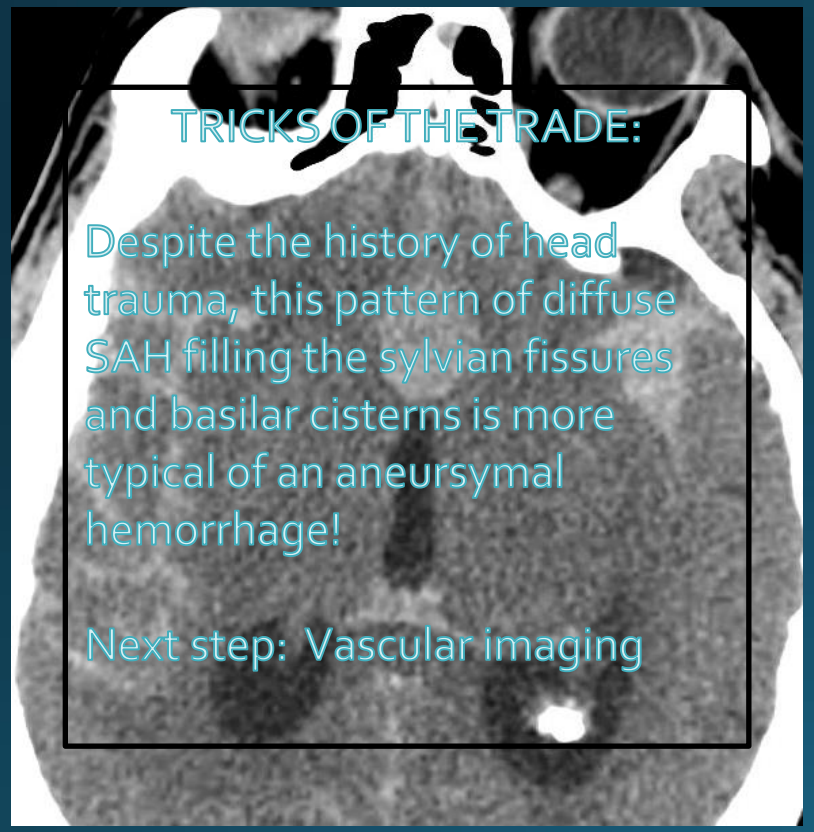
60-year-old with fall and head trauma



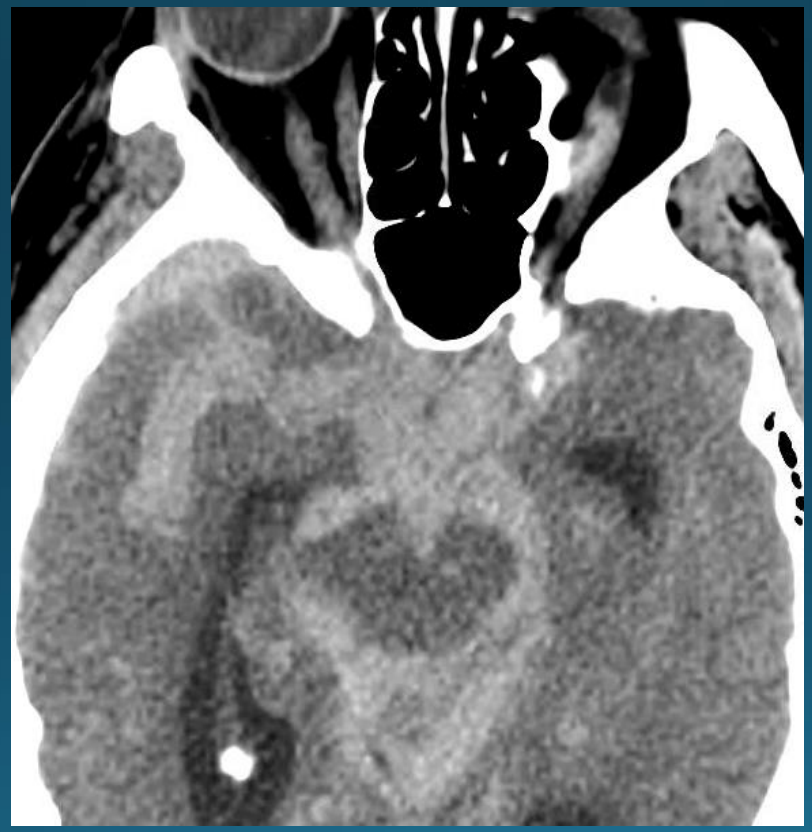
Axial NECT

Subarachnoid Hemorrhage

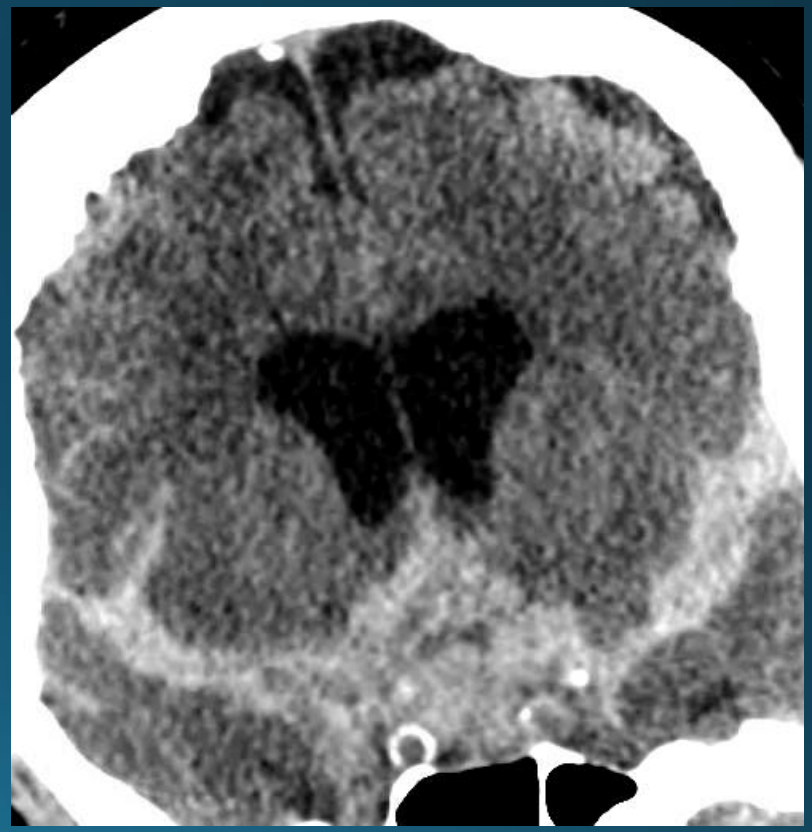
55-year-old with severe headache, fall, and head trauma



Axial NECT



Axial NECT



Coronal NECT

Primary Effects of CNS Trauma

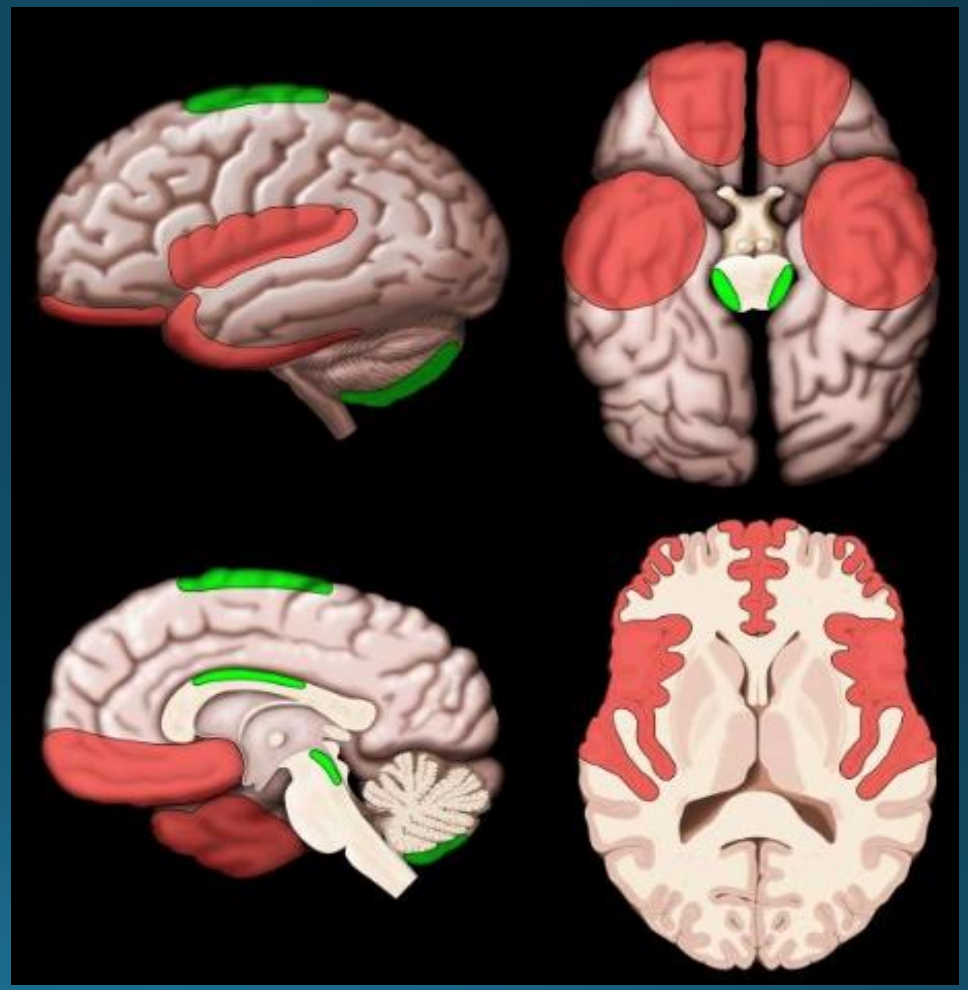
Outline:

- Extraaxial hemorrhages:
  - Epidural hematoma
  - Subdural hematoma
  - Subarachnoid hemorrhage

- Parenchymal injuries
  - Cerebral contusions
  - Diffuse axonal injuries

Cerebral Contusions

- "Brain bruises"
- Most common intraaxial TBI
- The majority result from blunt head injury, inducing abrupt change in angular momentum with impact of the brain against an osseous ridge or falx/tentorium
- Uncommonly, a depressed skull fracture may damage the underlying brain
- Nearly half involve the temporal lobes and inferior (orbital) surfaces of the frontal lobes



Source: http://my.statdx.com

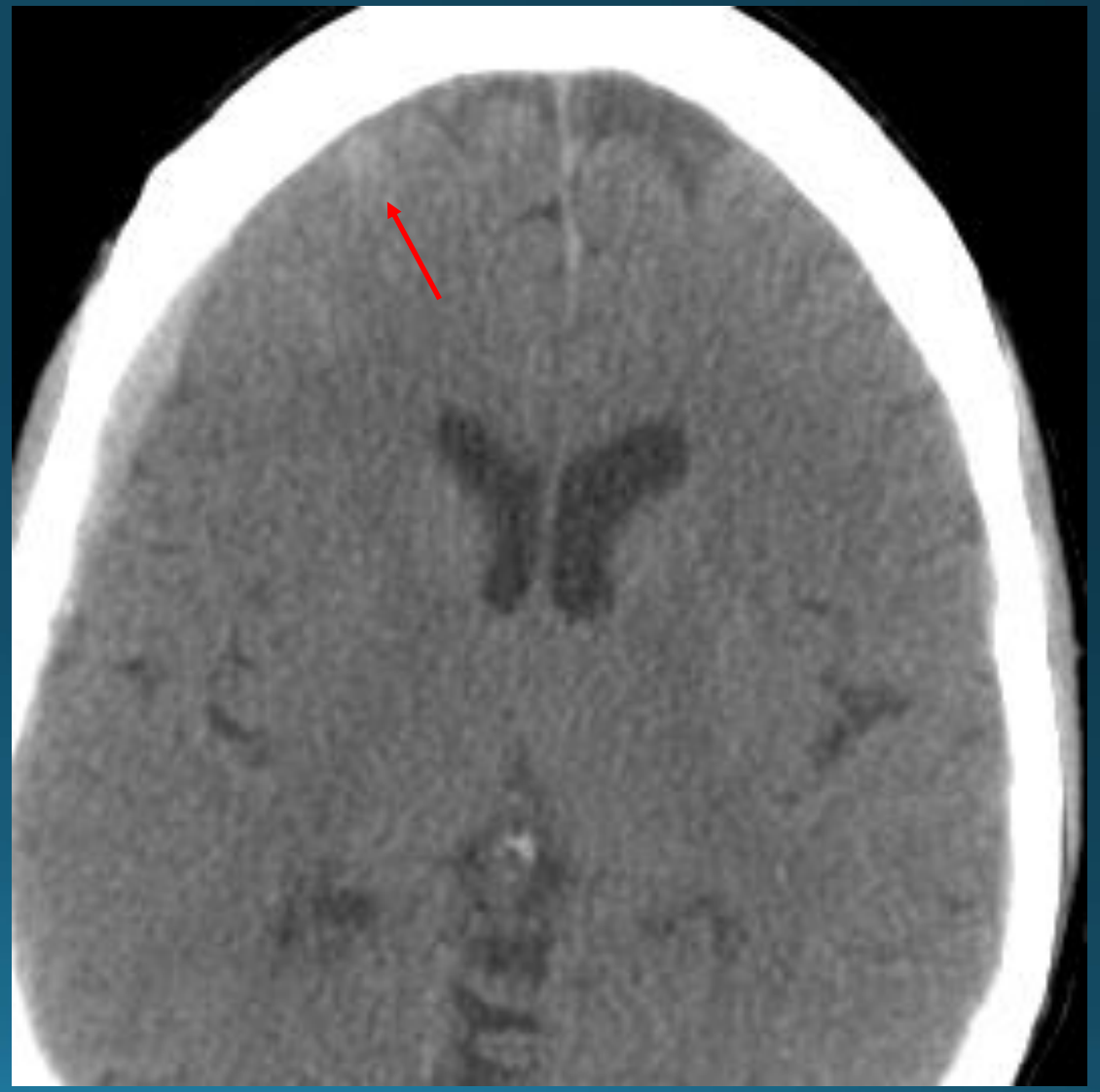
Cerebral Contusions

- Occur at all ages, peak age 15-24 (M>F)
- Initial symptoms vary from none to confusion, seizure, and obtundation
- Less frequently associated with immediate LOC unless extensive or occurring with other traumatic lesion (DAI)
- Tx options vary from conservative (serial imaging) to surgical evacuation of large lesions or severe brain swelling

Cerebral Contusions

Imaging:

- Initial scans obtained soon after closed head injury may be normal
- The most frequent abnormality is petechial hemorrhages along gyral crests adjacent to the calvaria
- Small lesions may coalesce, forming larger hematomas
- "Blooming" over time is the rule with increase in lesion size, number of lesions, and increase in mass effect and edema



Management of Concussion and Mild Traumatic Brain Injury: A Synthesis of Practice Guidelines

Noah D. Silverberg, PhD, Mary Alexis Iaccarino, MD, et al

Archives of Physical Medicine and Rehabilitation
Volume 101 Issue 2 Pages 382-393 (February 2020)
DOI: 10.1016/j.apmr.2019.10.179

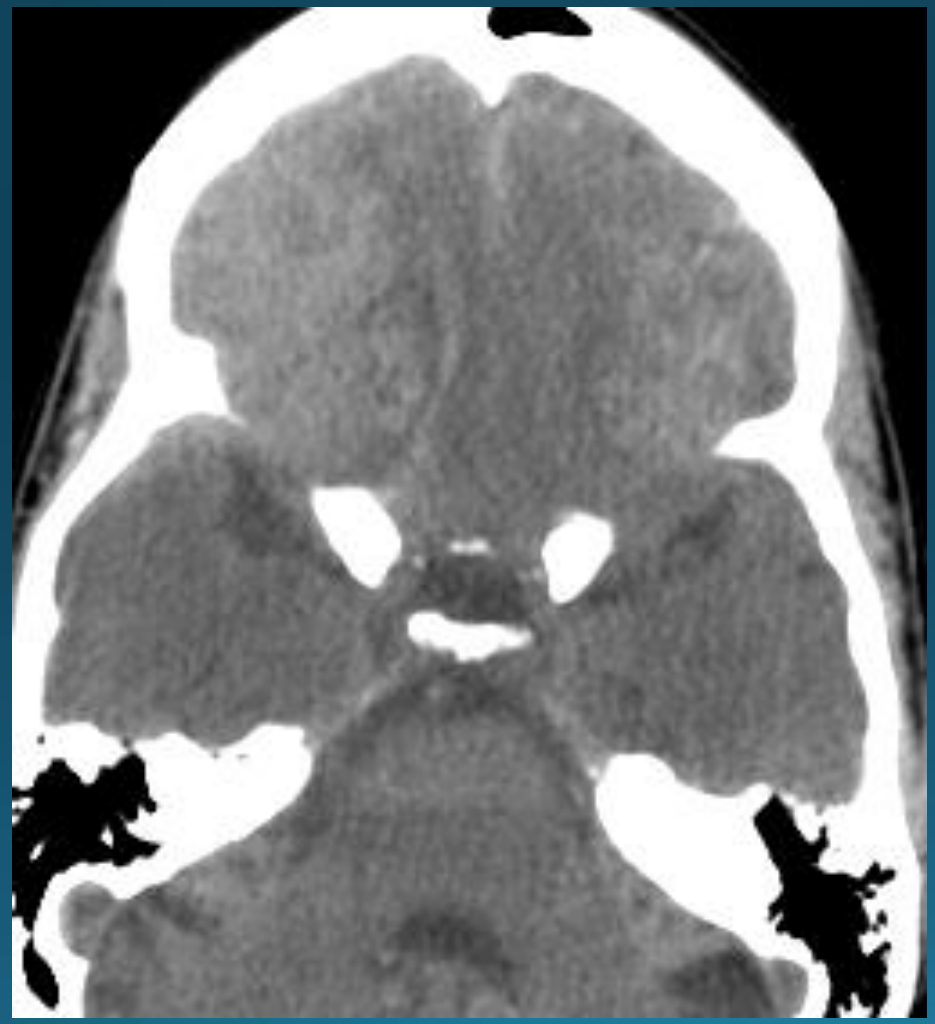
Cerebral Contusions

71-year-old with fall and head trauma, history of cancer on anticoagulation.

TRICKS OF THE TRADE:

The most common locations for parenchymal contusions are within the anteroinferior frontal and temporal lobes.

Always double check these locations in cases of head injury!

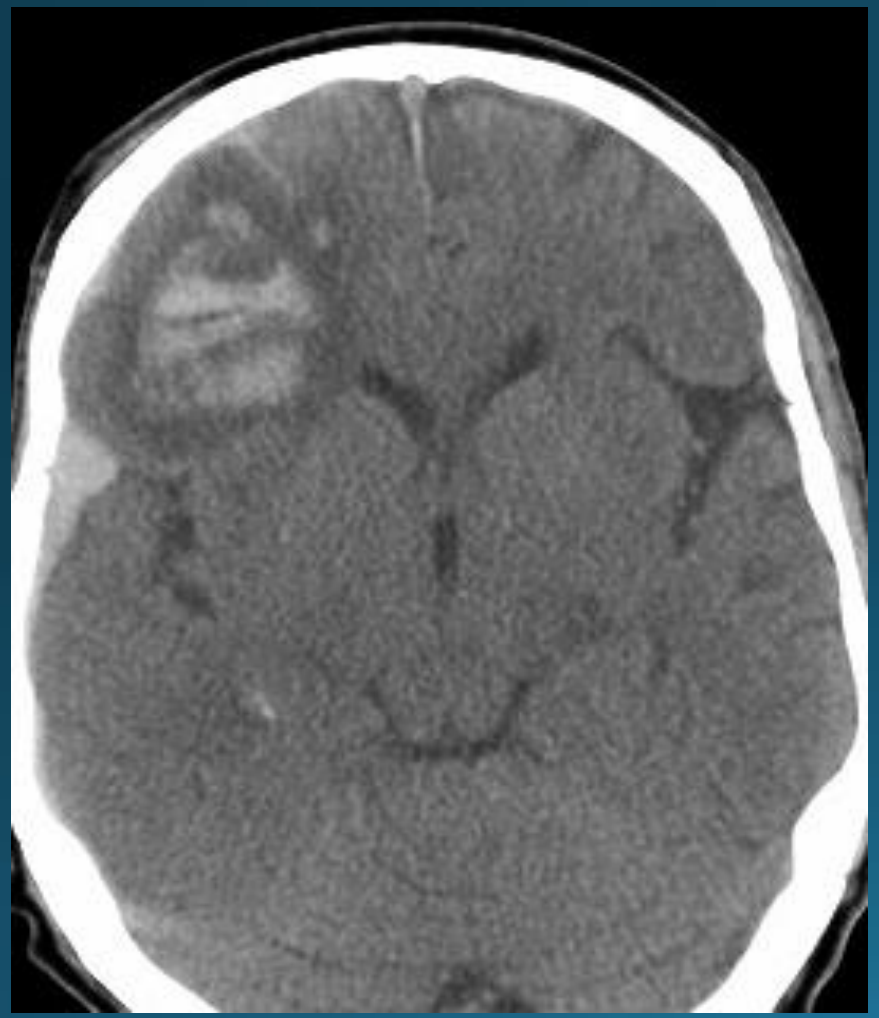


Axial NECT

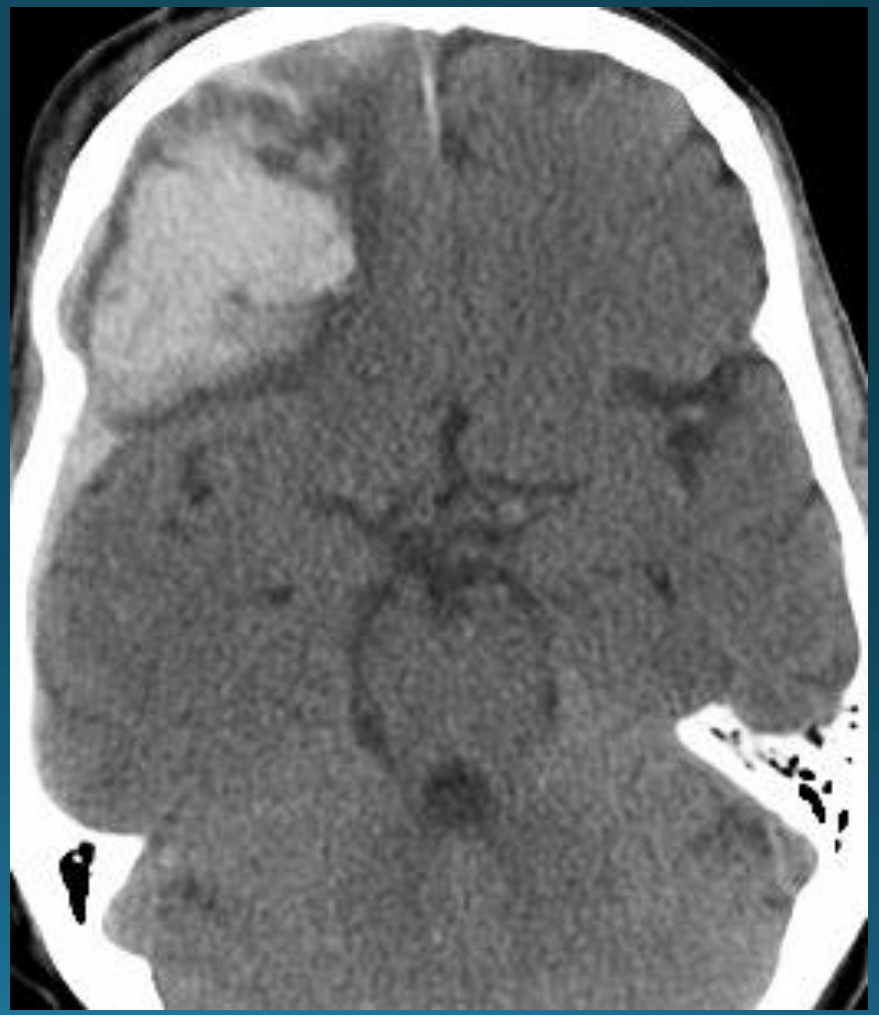
Cerebral Contusions

65-year-old with fall from ladder

The rim of surrounding edema is nicely demonstrated on these images



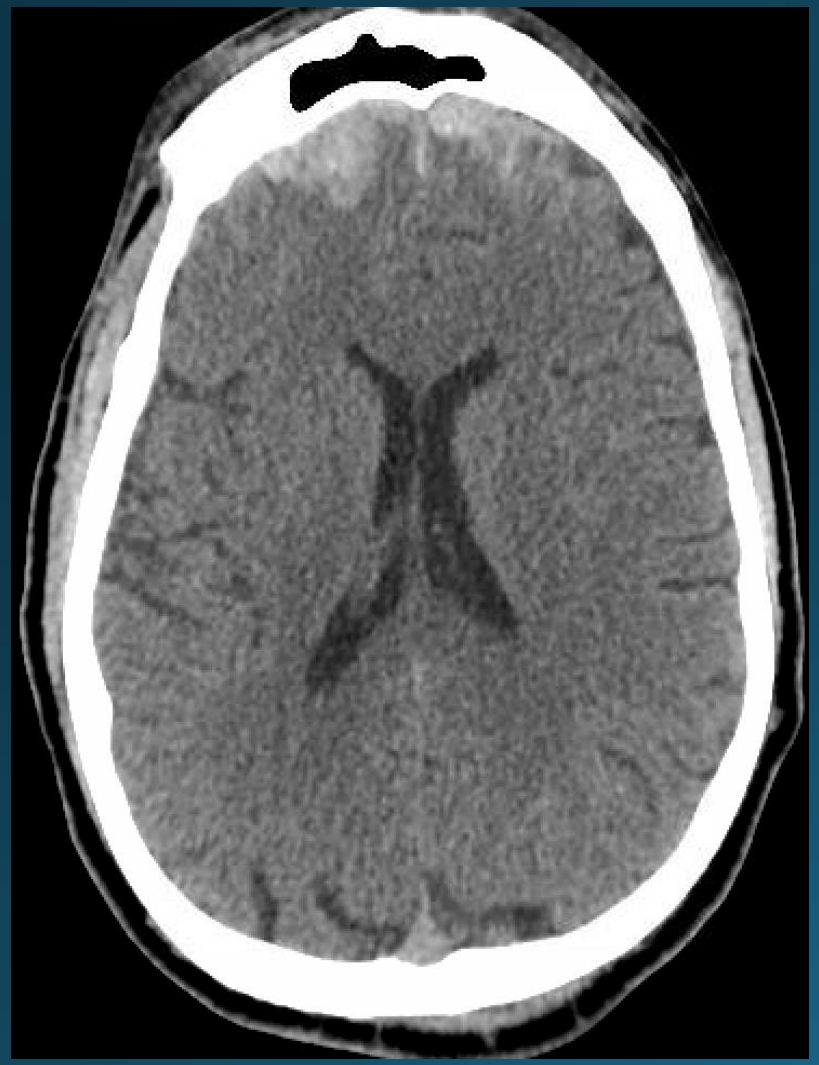
Axial NECT



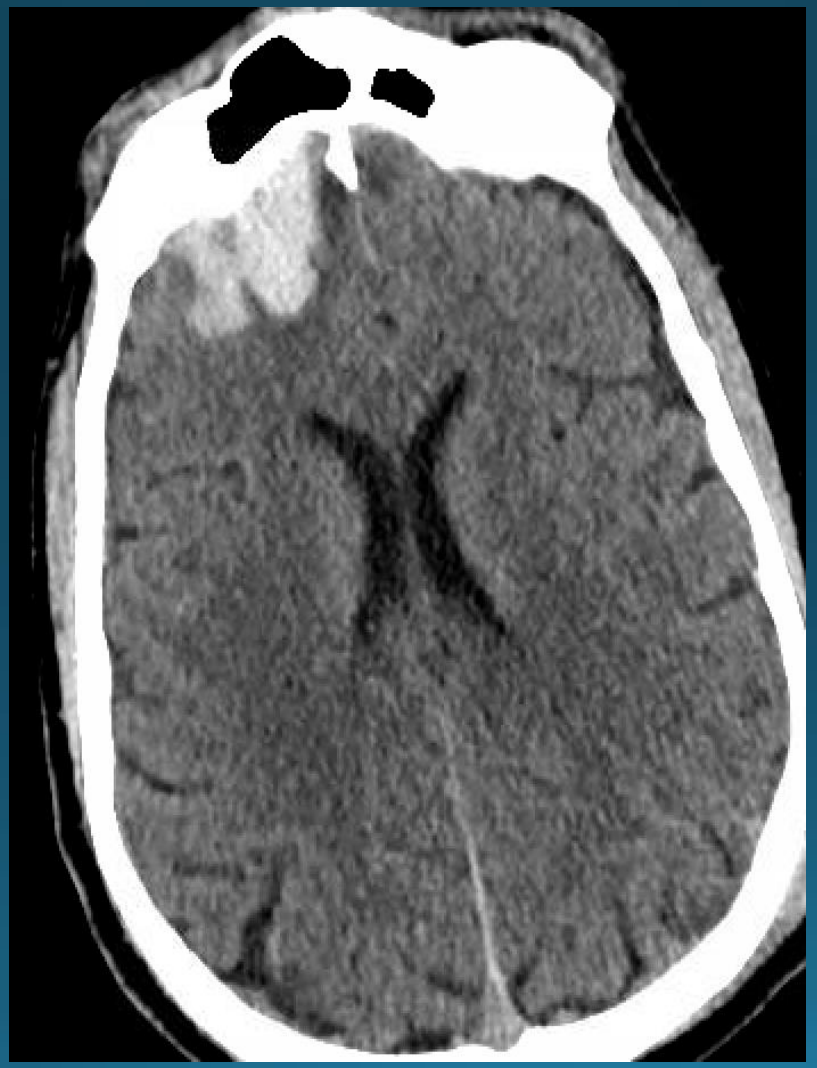
Axial NECT

Cerebral Contusions

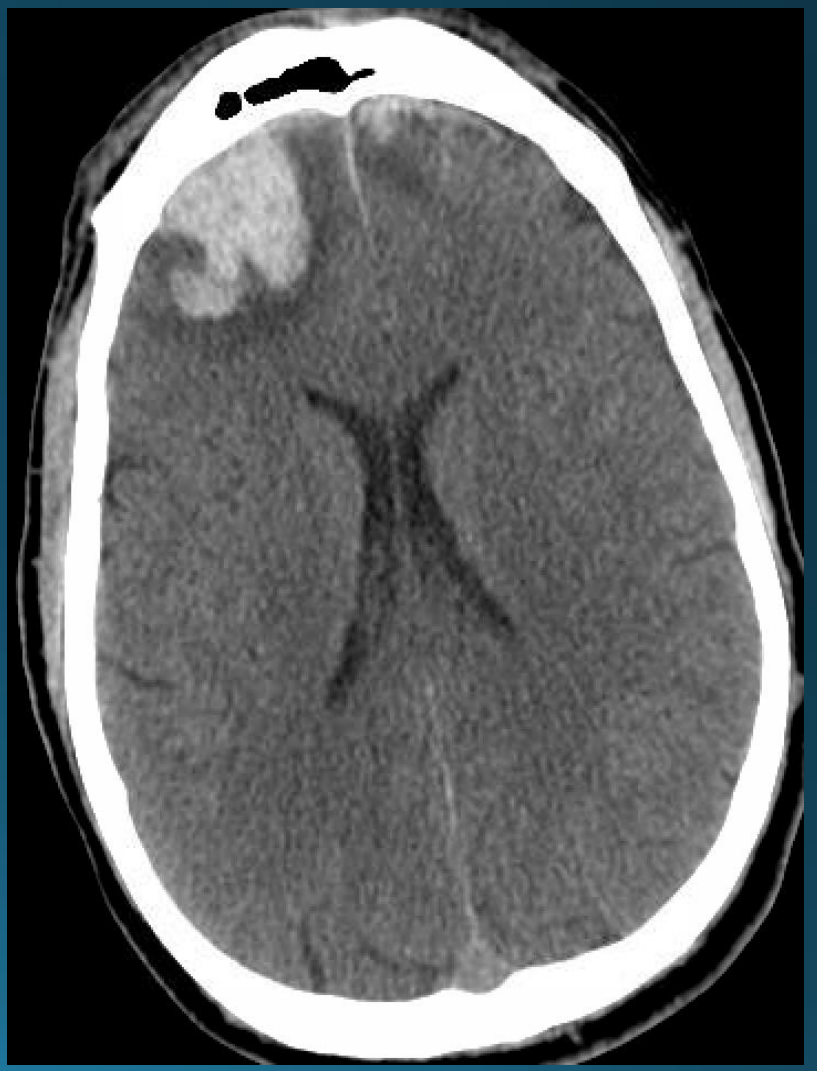
20-year-old with severe head trauma, ejected from vehicle



Axial NECT - Initial



Axial NECT – 1 day later

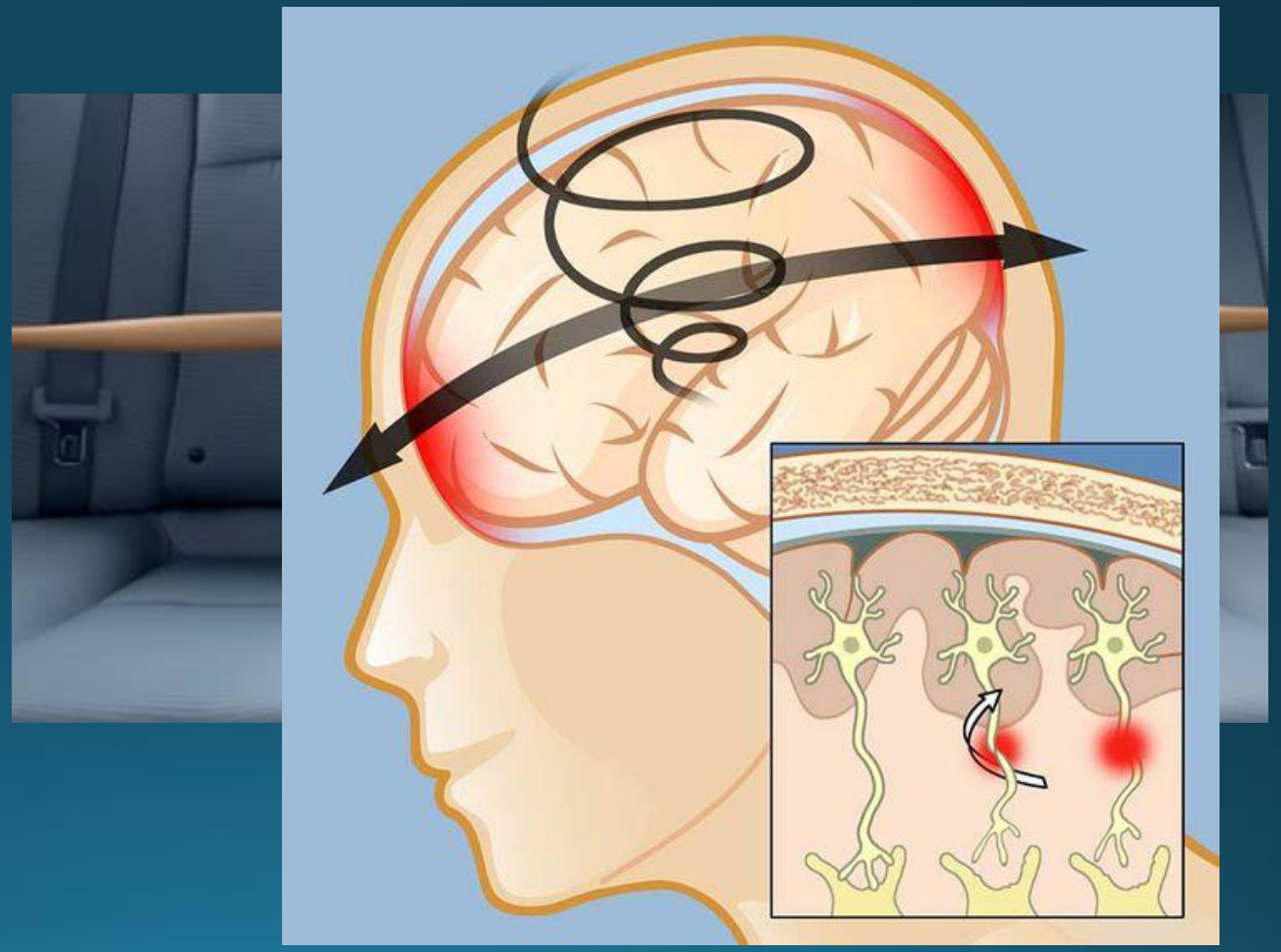


Axial NECT – 3 days later

Diffuse Axonal Injury

Introduction:

- AKA axonal stretch injury
- Second most common parenchymal lesion seen in TBI
- Usu. from high velocity MVAs and are non-impact injuries secondary to acceleration/deceleration forces
- The cortex moves at different speeds than the underlying deeper structure, resulting in axonal stretching (particularly at the interface of structures with varying densities)

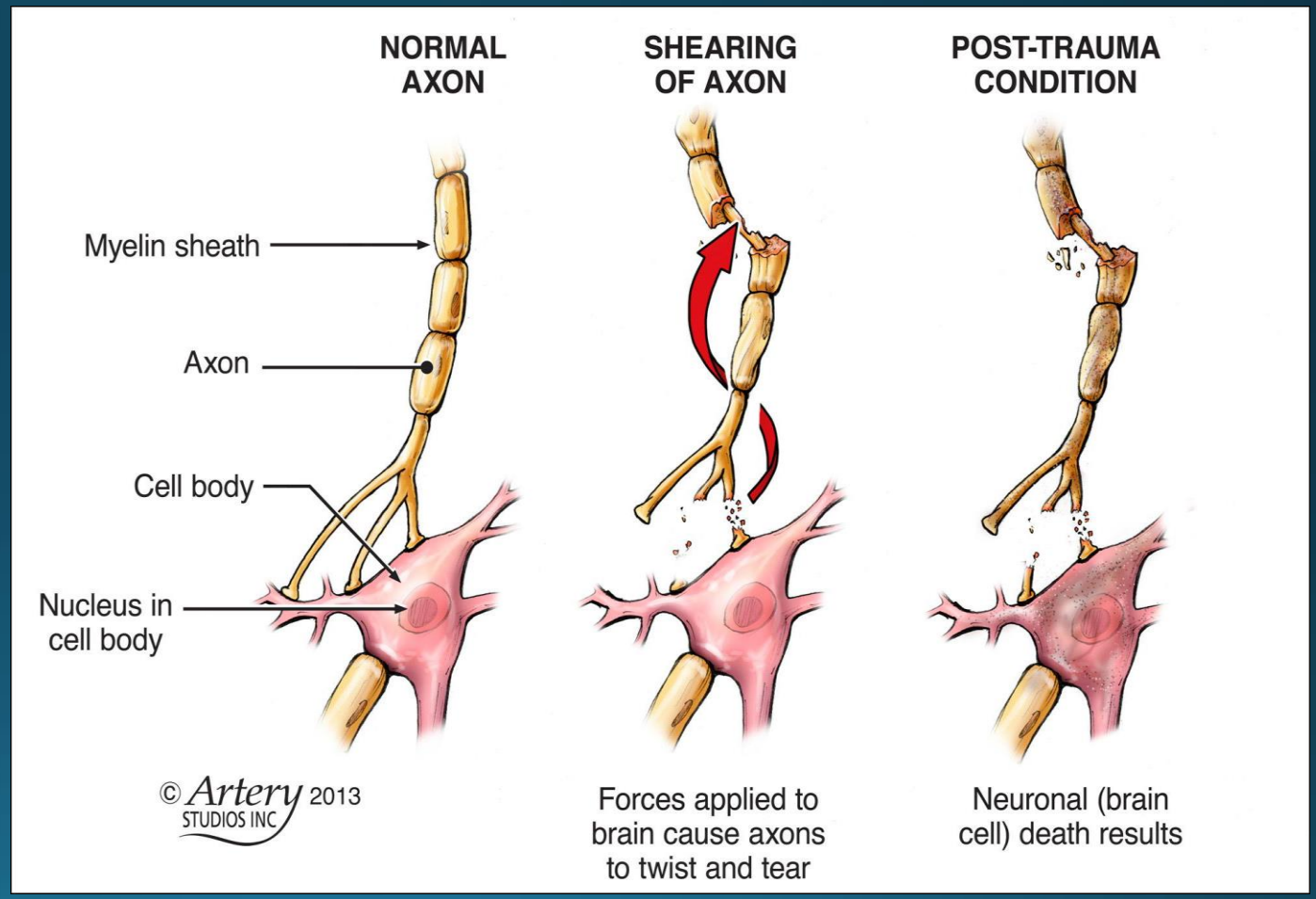


DAI CONSIDERATIONS

- Diffuse axonal injury (DAI):
  - Contusions predominantly involve the superficial areas of the brain, with significant cortical involvement
  - DAI usually shows cortical sparing.
- Brain laceration:
  - Actual laceration of the pial membrane characterized by extension of hemorrhage to the deep white matter/basal ganglia.
  - A full thickness laceration may extend from the cortex to the ventricle.

Diffuse Axonal Injury

- Downstream effects of axonal injury include disruption of transport, depolarization, ion fluxes, and release of neurotransmitters
- Associated cytotoxic edema contributes to altered anisotropy of the brain
- Results in widespread alteration of brain metabolism, lactate accumulation, cellular swelling, and apoptosis



Source: http://www.tbibraininjurysurvivor.com/

Diffuse Axonal Injury

Clinical Issues:

- DAI is suspected when clinical symptoms are disproportionate to imaging findings
- Loss of consciousness or immediate coma are typical
- Present in virtually all fatal TBIs and 75% of moderate or severe injury surviving the acute stage

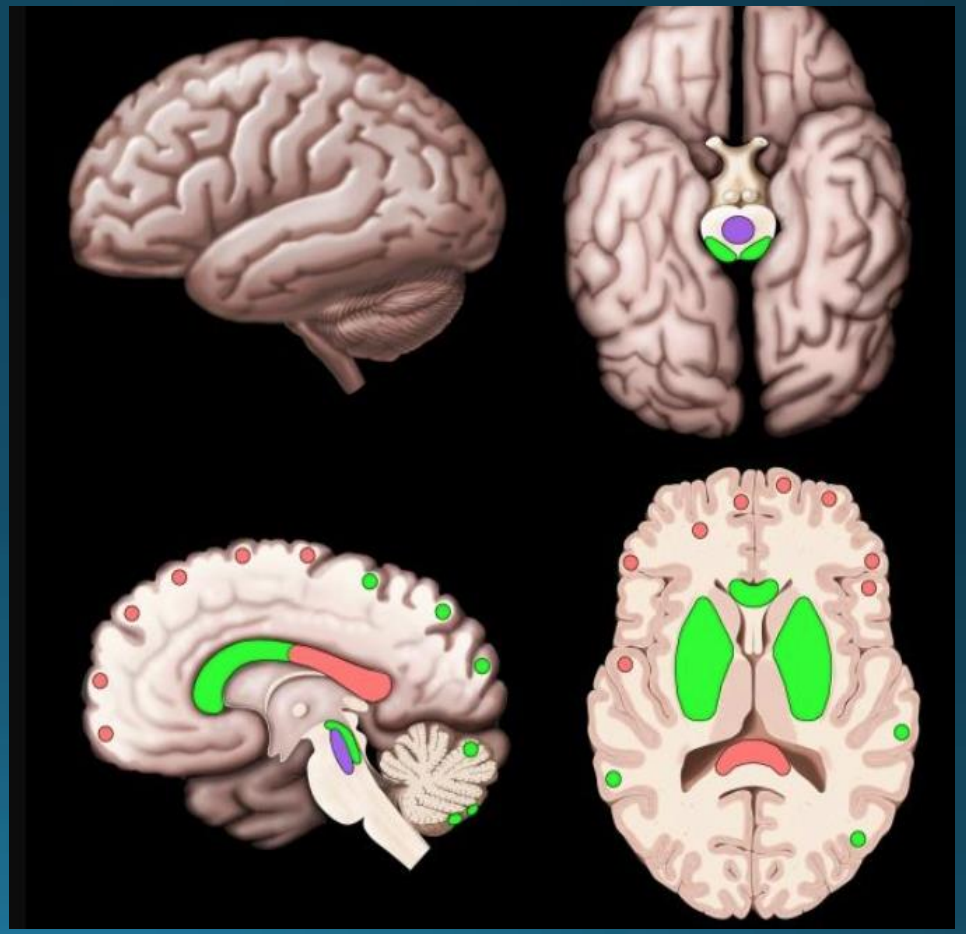
- Spectrum of severity:
  - Mild: clinical abnormalities may persist for months or longer and include headache, memory/cognitive impairment, or personality change
  - Severe: rarely itself causes death but may result in a persistent vegetative state

Diffuse Axonal Injury

Staging System:

Increasing severity of force collates with deeper brain involvement

- Stage I: Frontal/temporal lobe grey-white matter interface
- Stage II: Lobar deep white matter and corpus callosum (most commonly splenium and posterior body)
- Stage III: Brainstem involvement

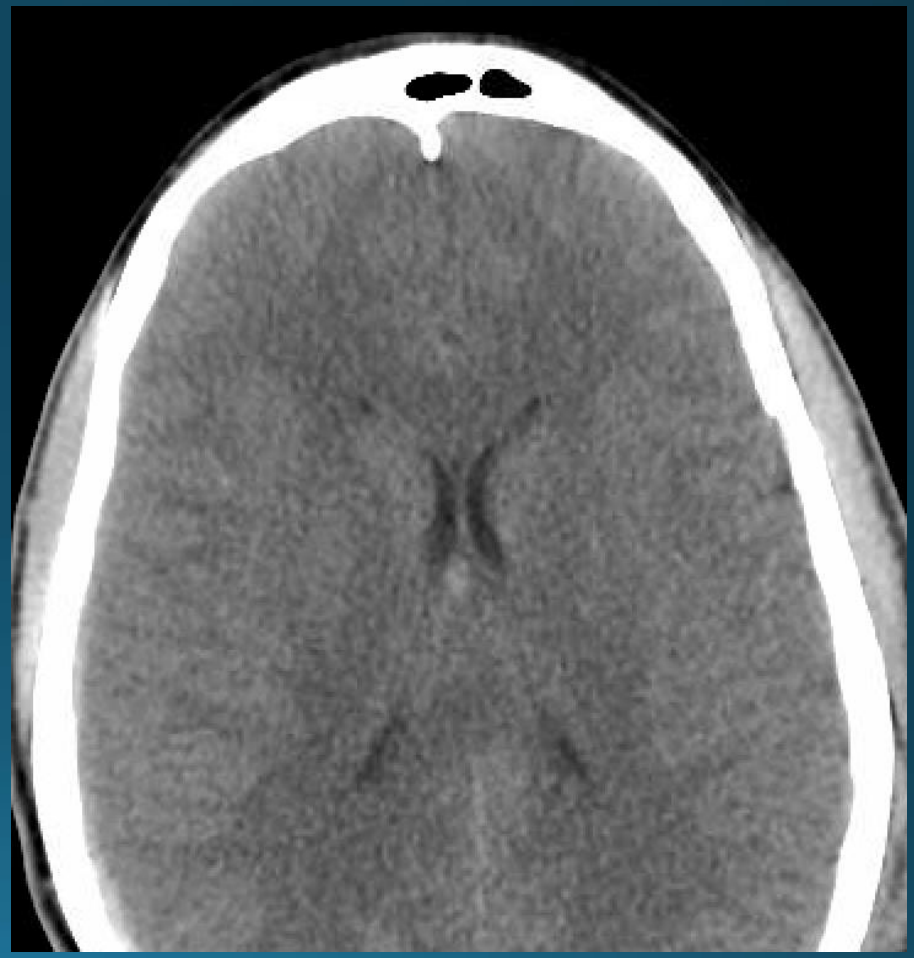


Source: http://my.statdx.com

Diffuse Axonal Injury

Imaging:

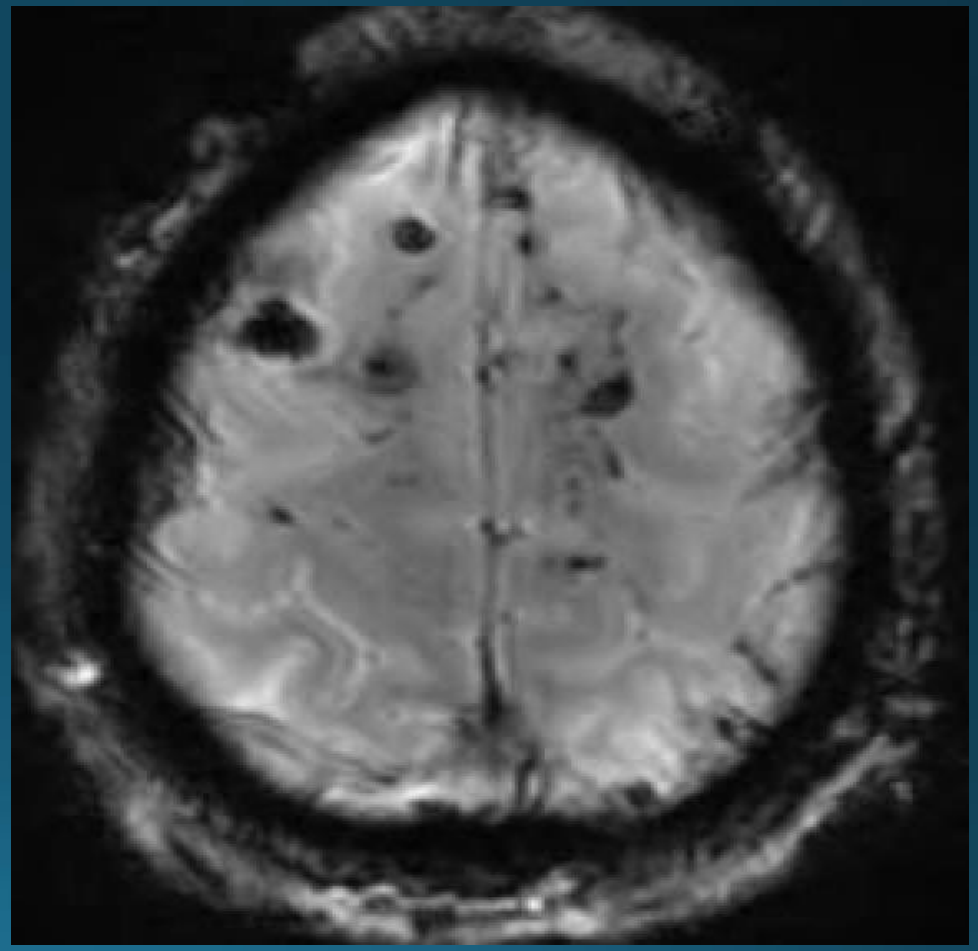
- CT:
  - Initial CT is often normal or may demonstrate mild brain swelling with sulcal effacement
  - A few small round/ovoid subcortical hemorrhages may be visible but underlying damage is much more diffuse
  - With increasing severity, small hemorrhagic lesions may be seen within deeper white matter, corpus callosum, or within the brainstem



Diffuse Axonal Injury

Imaging:

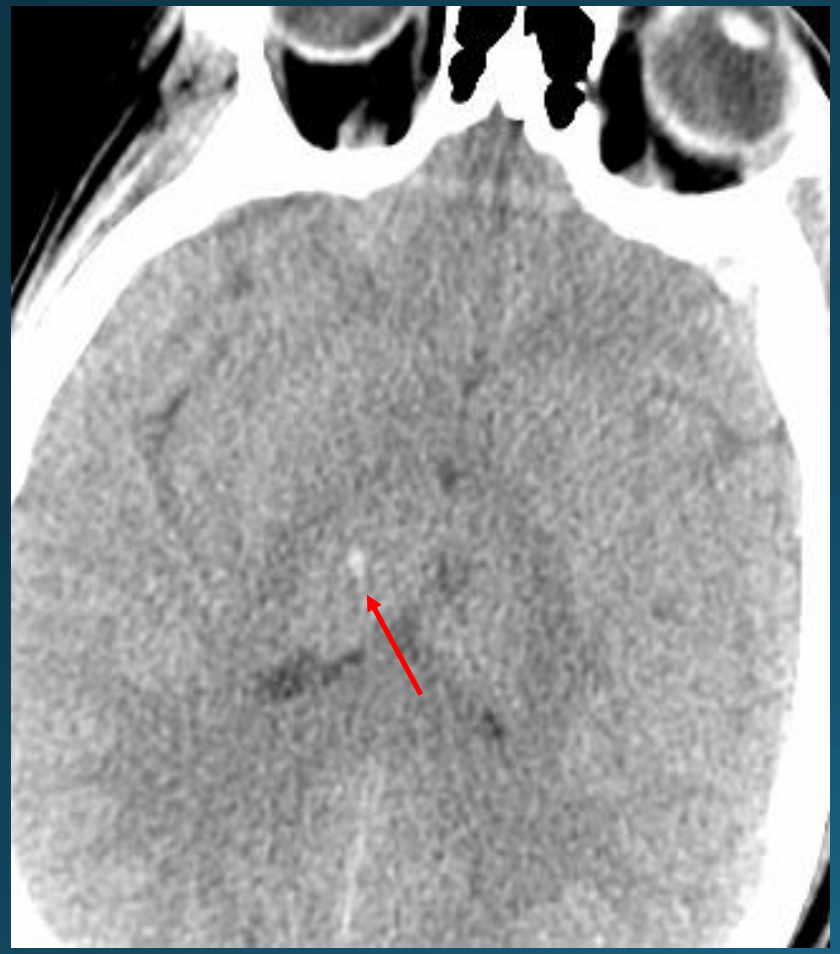
- MR:
  - T1: Most DAIs are nonhemorrhagic, thus T1 scans are often normal
  - T2/FLAIR: hyperintense foci in the subcortical white matter, corpus callosum, and brainstem (increasing severity)
  - GRE/SWI: very sensitive to the microbleeds of DAI, typically multifocal ovoid or linear hypointensities
  - DWI: may show restricted diffusion



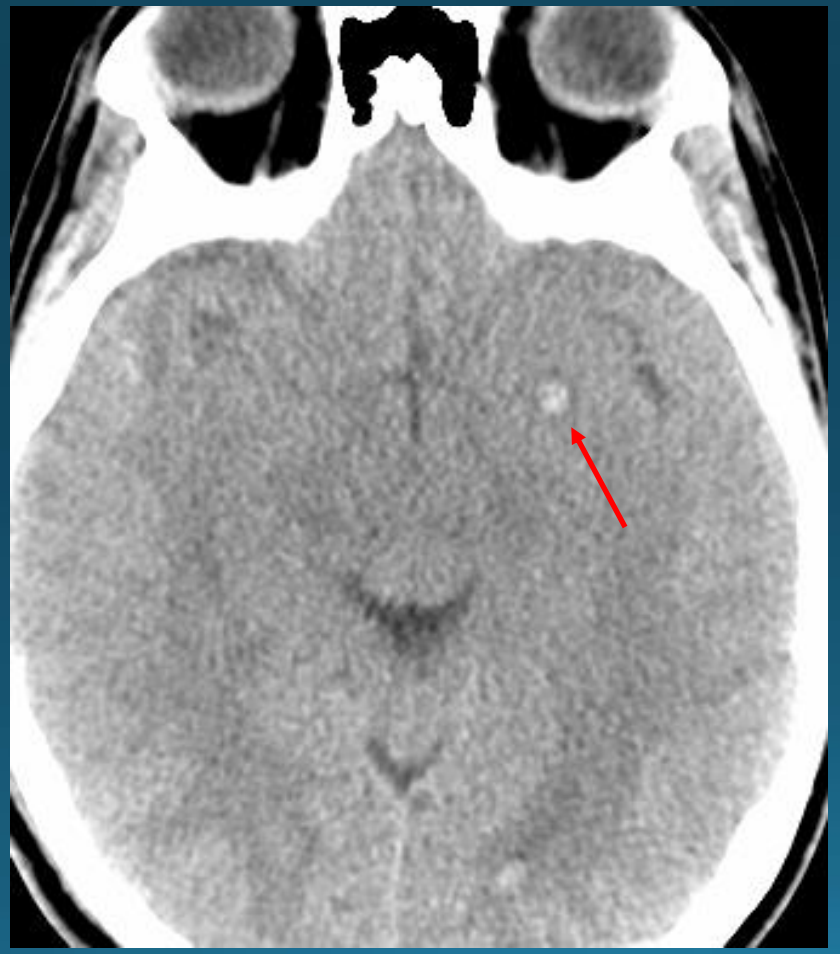
Source: http://my.statdx.com

Diffuse Axonal Injury

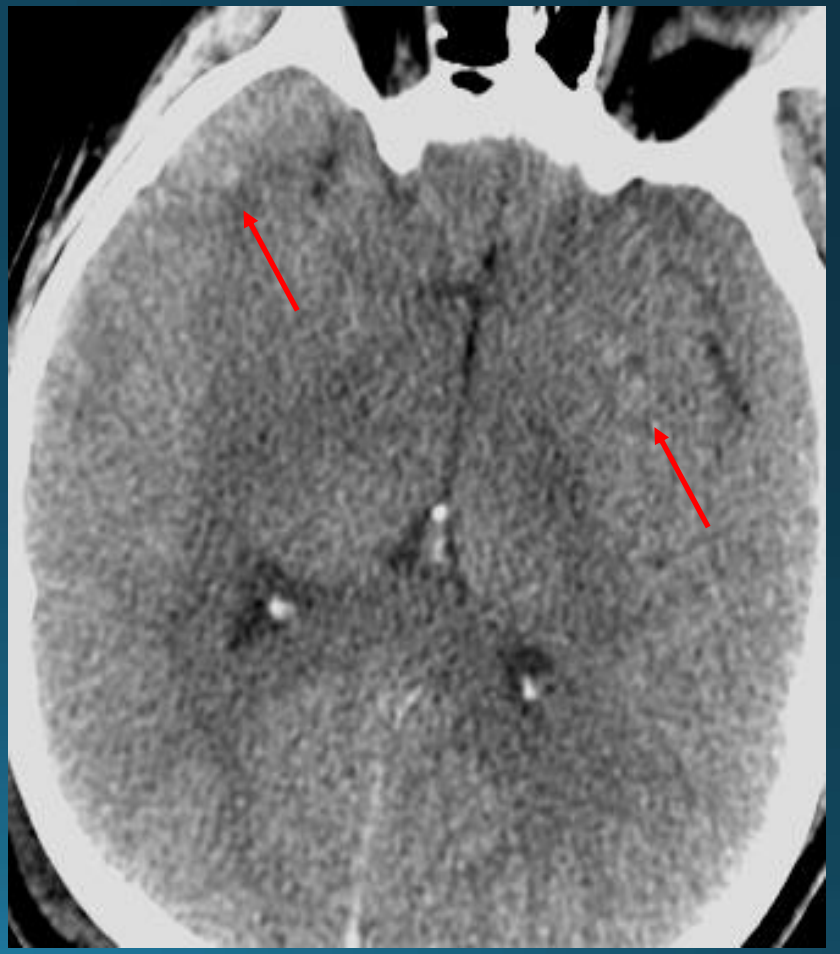
21-year-old involved in MVA, unresponsive at the scene



Axial NECT



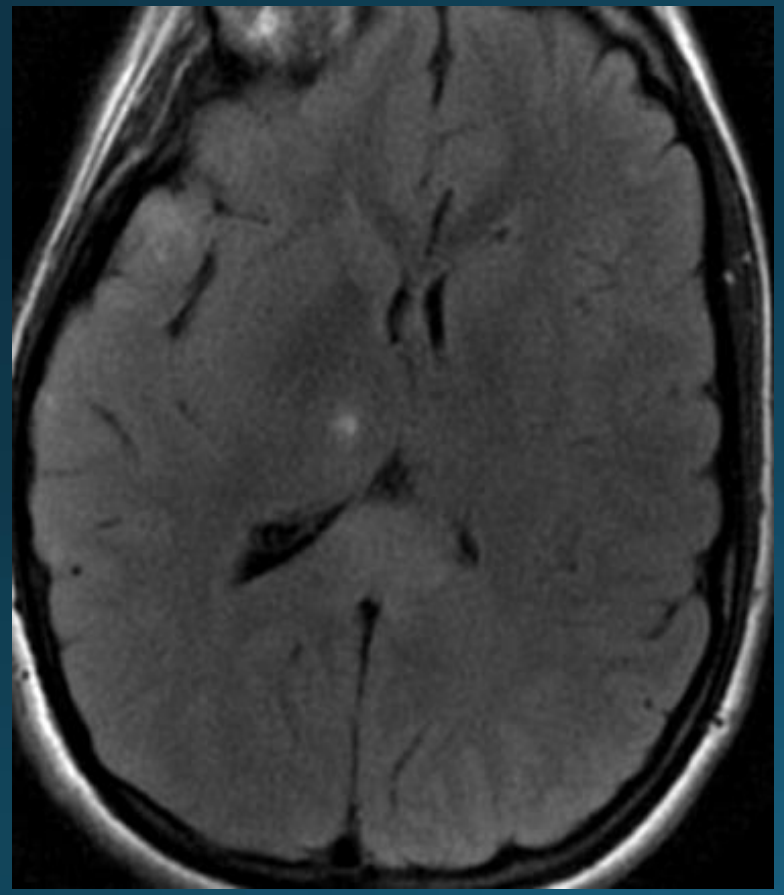
Axial NECT



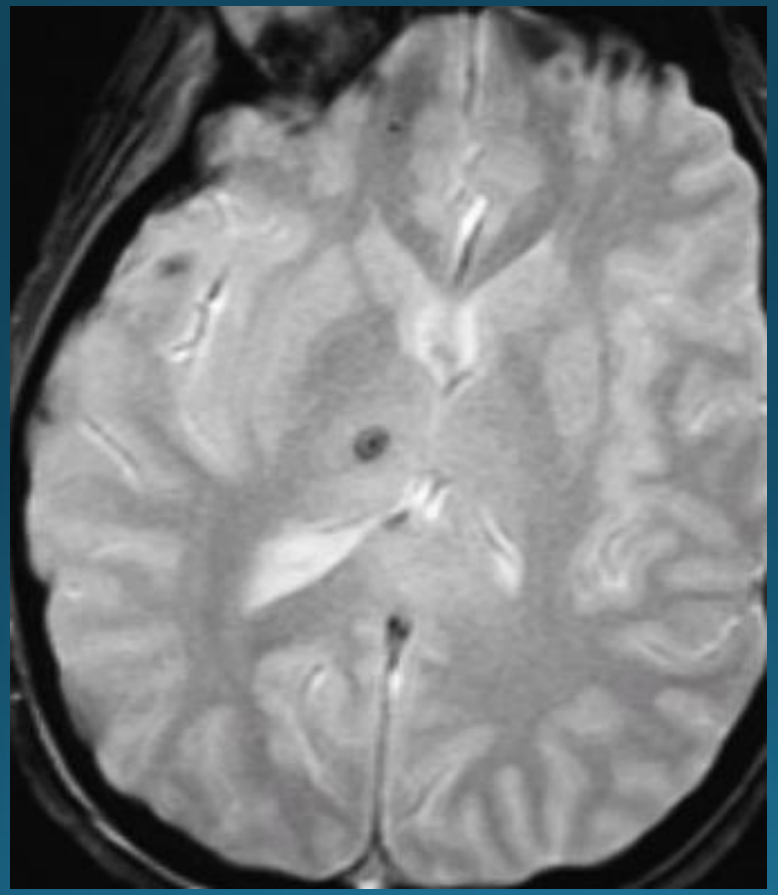
Axial NECT

Diffuse Axonal Injury

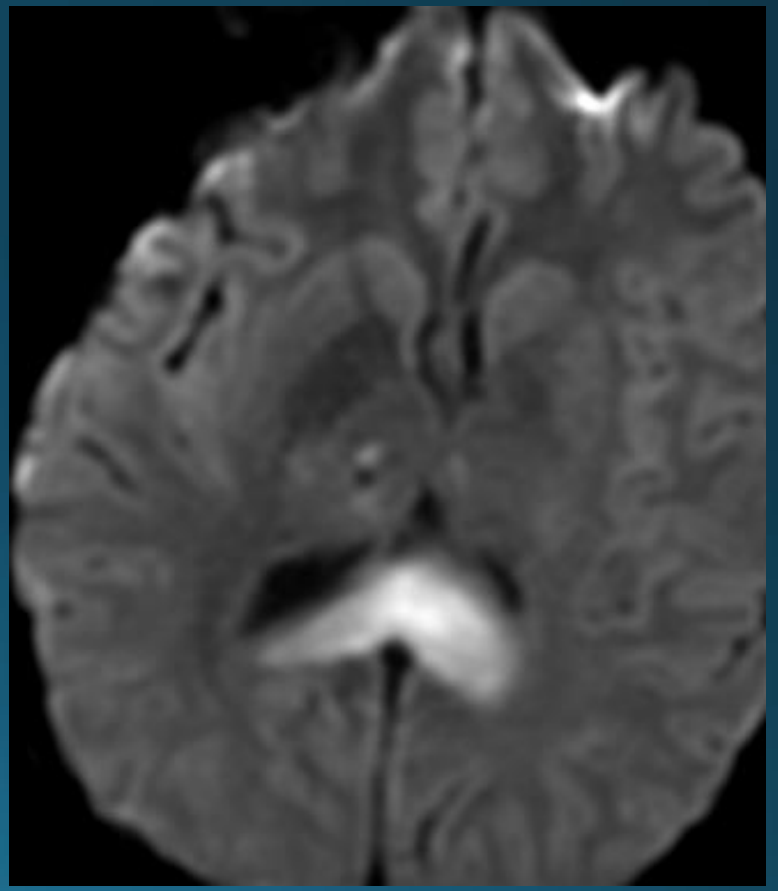
21-year-old involved in MVA, unresponsive at the scene



FLAIR



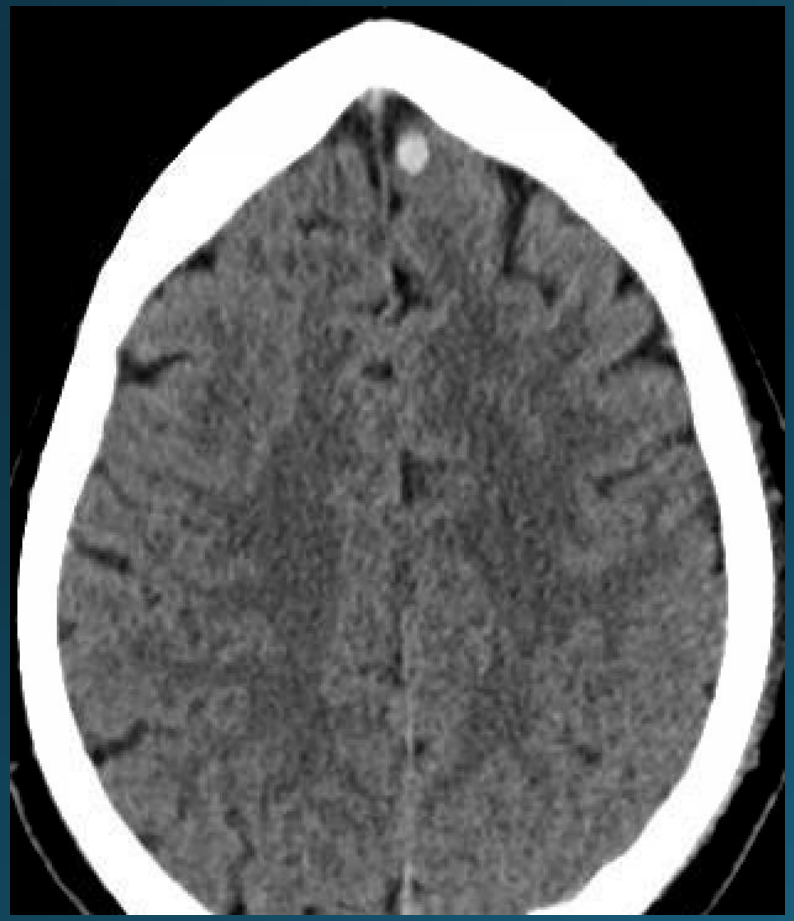
GRE



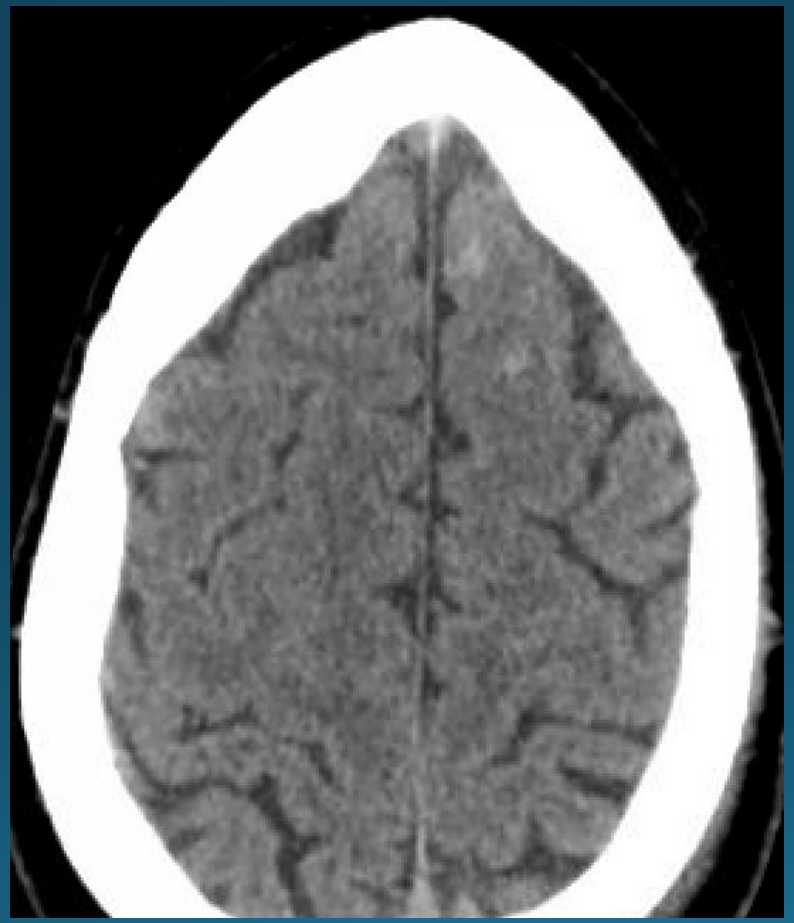
DWI

Diffuse Axonal Injury

34-year-old involved in rollover MVC



Axial NECT



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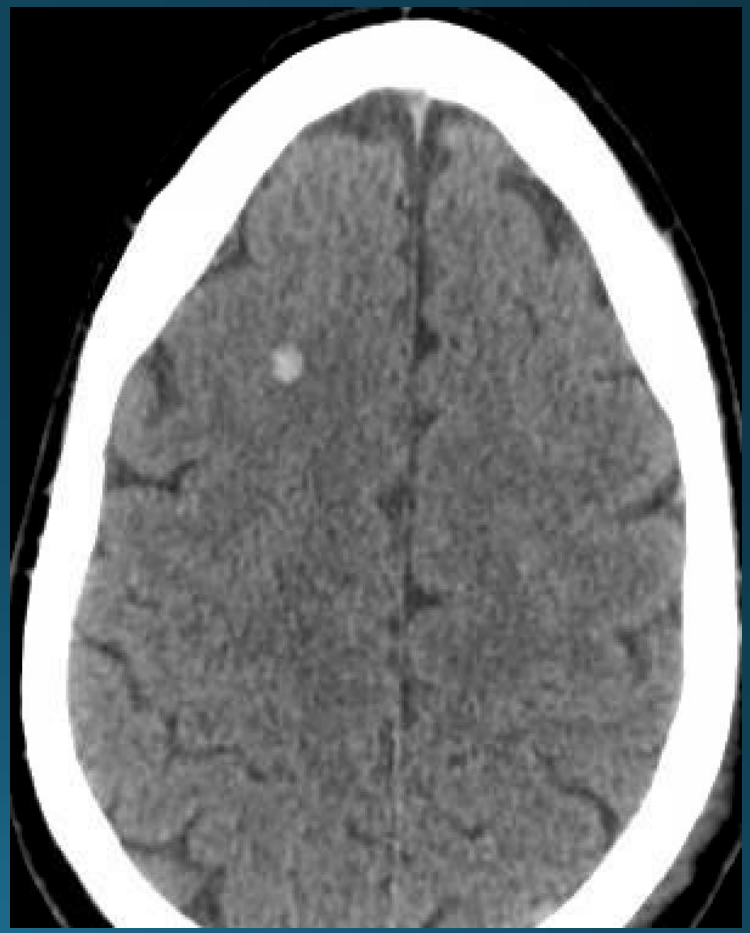
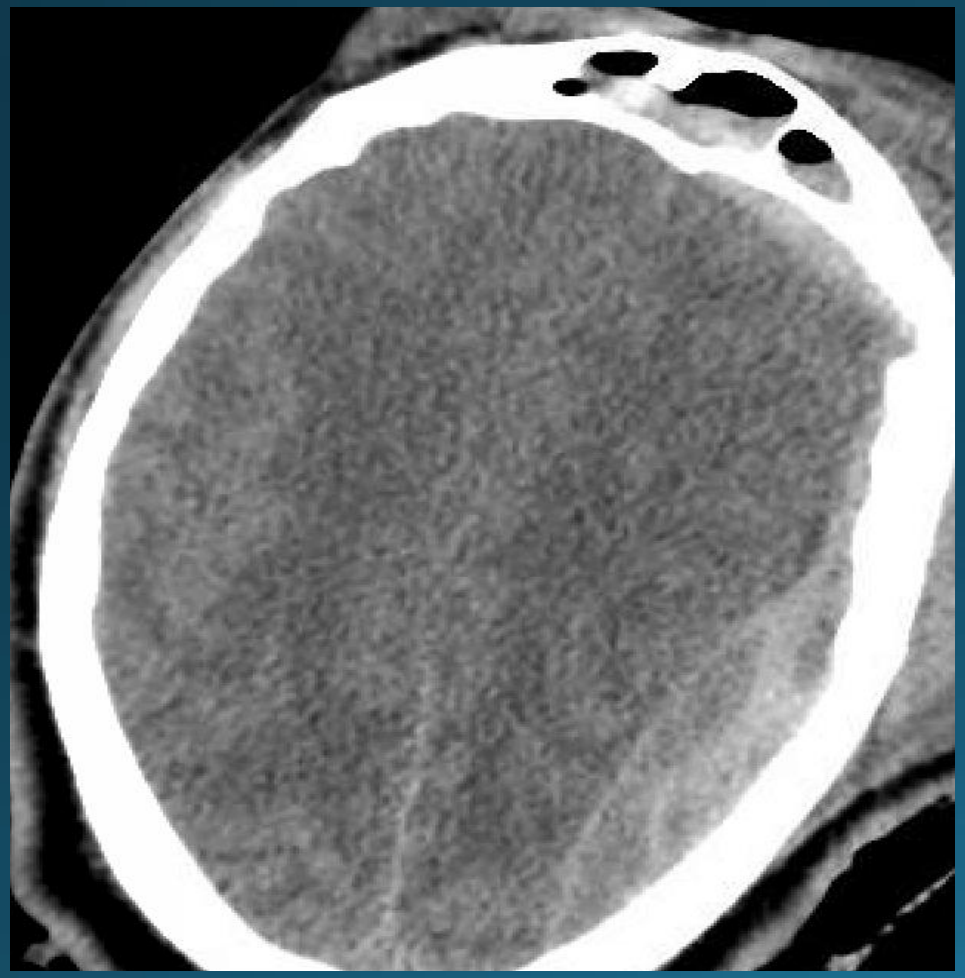


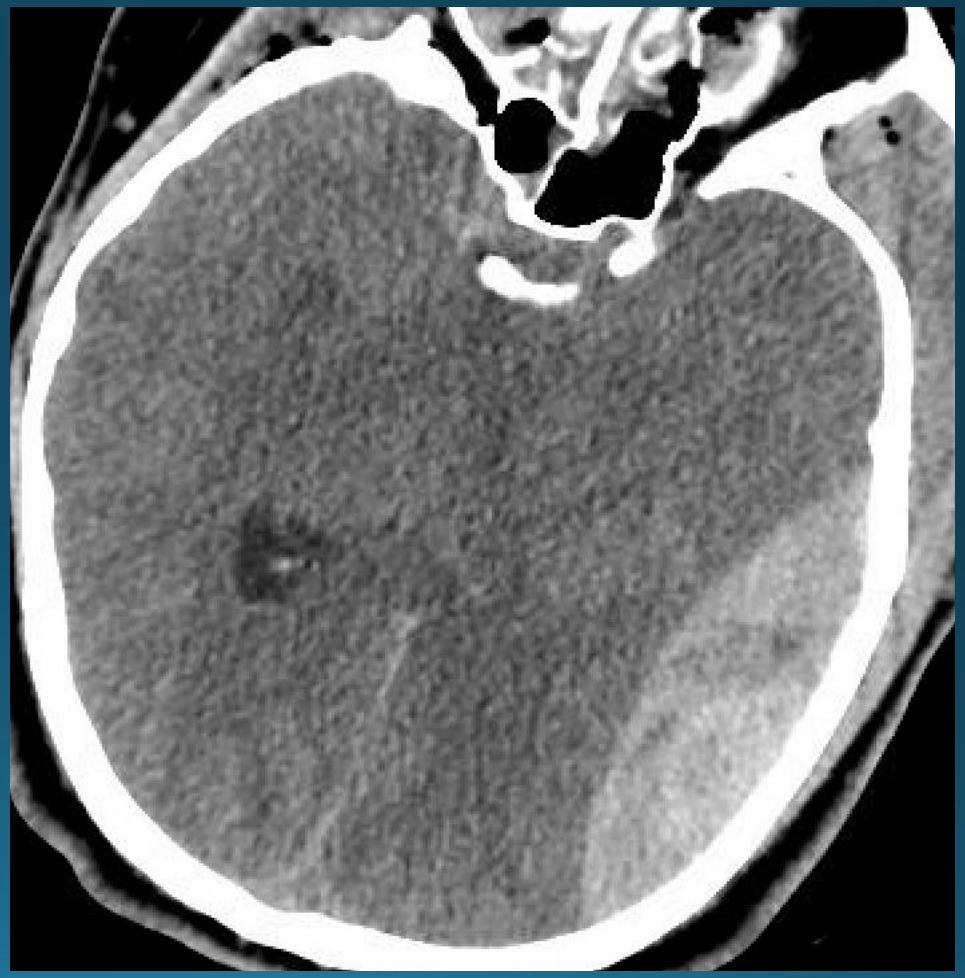
Axial NECT

Diffuse Axonal Injury

20-year-old with severe head injury



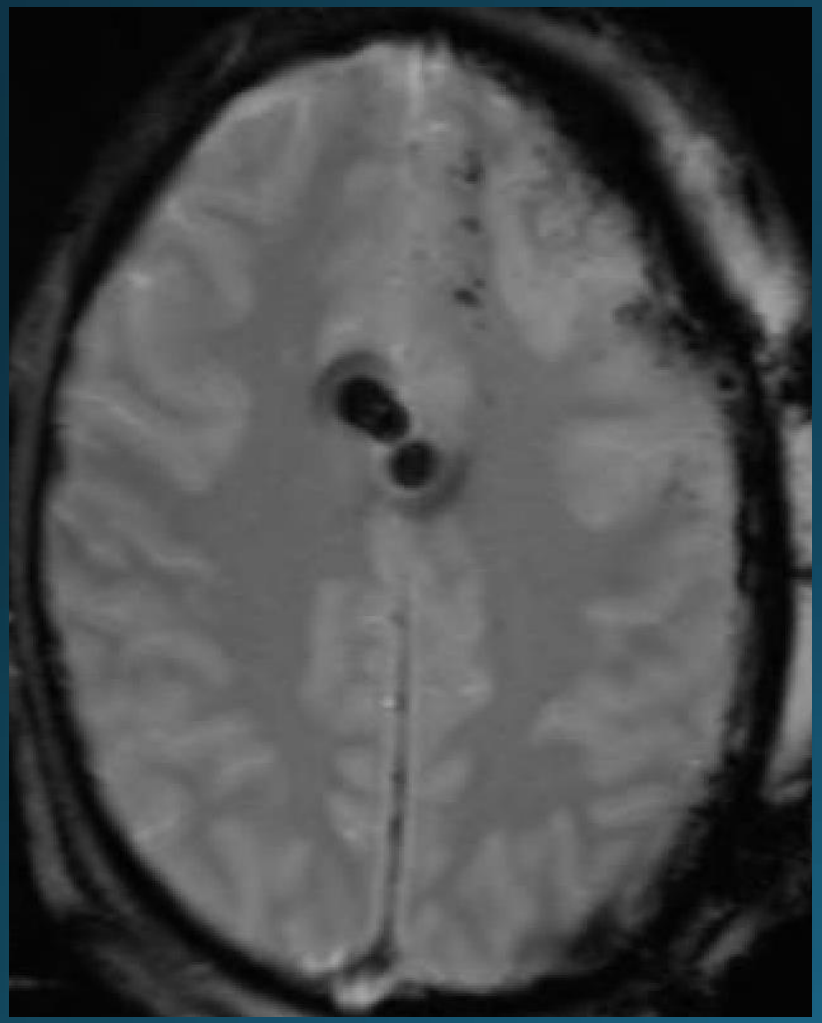
Axial NECT



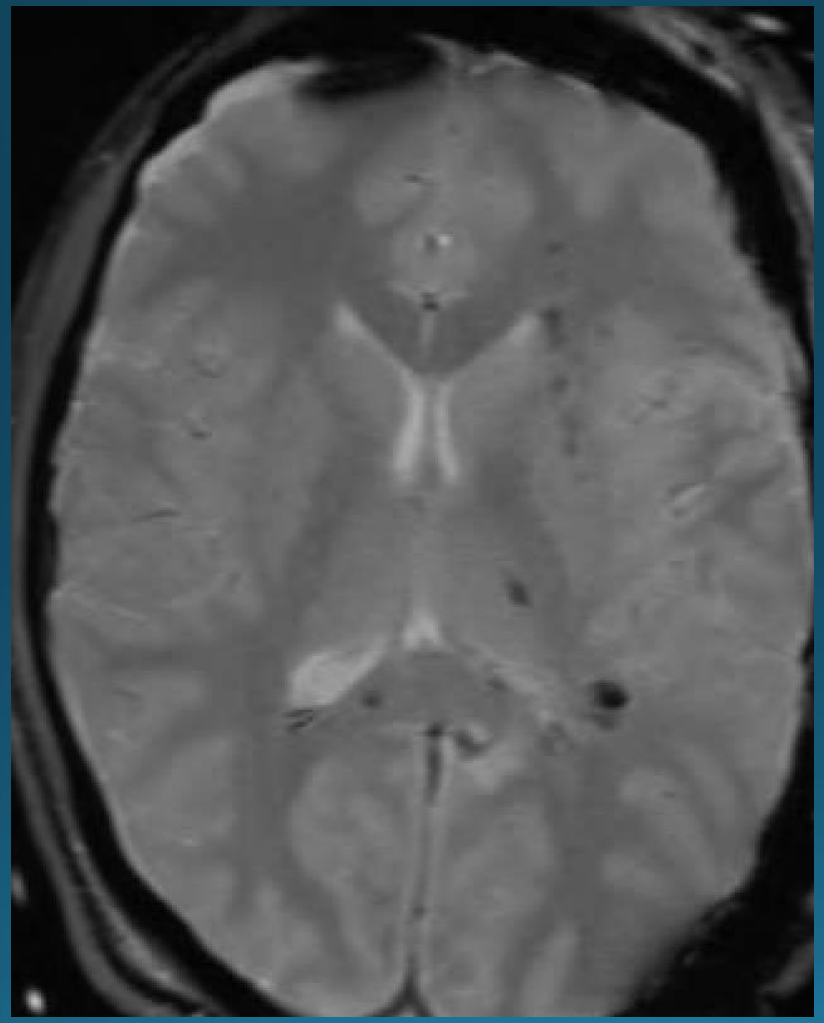
Axial NECT

Diffuse Axonal Injury

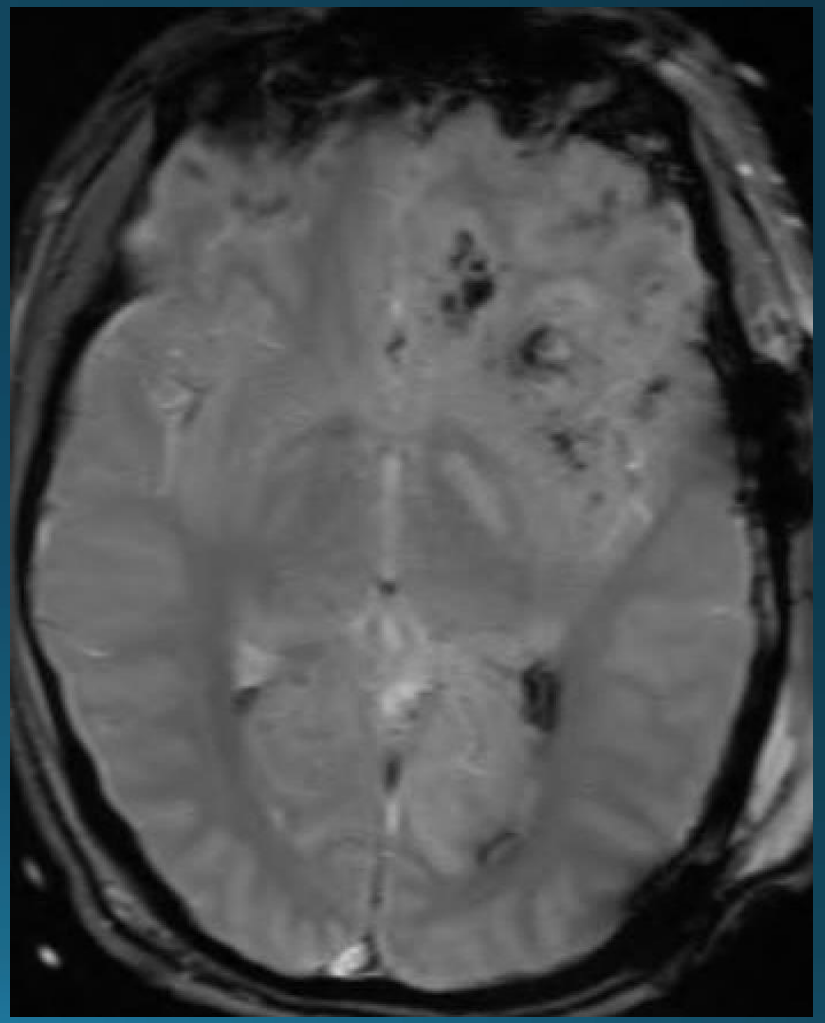
MRI obtained as the patient's mental status was not improving after craniotomy



GRE



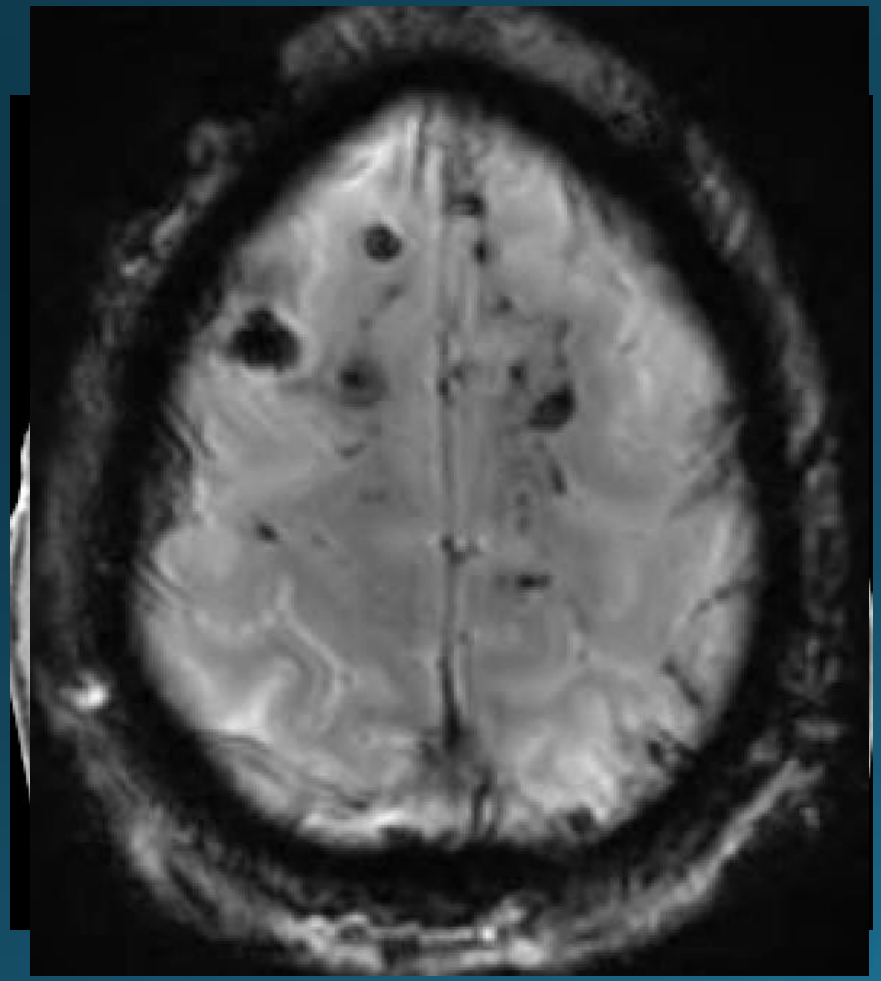
GRE



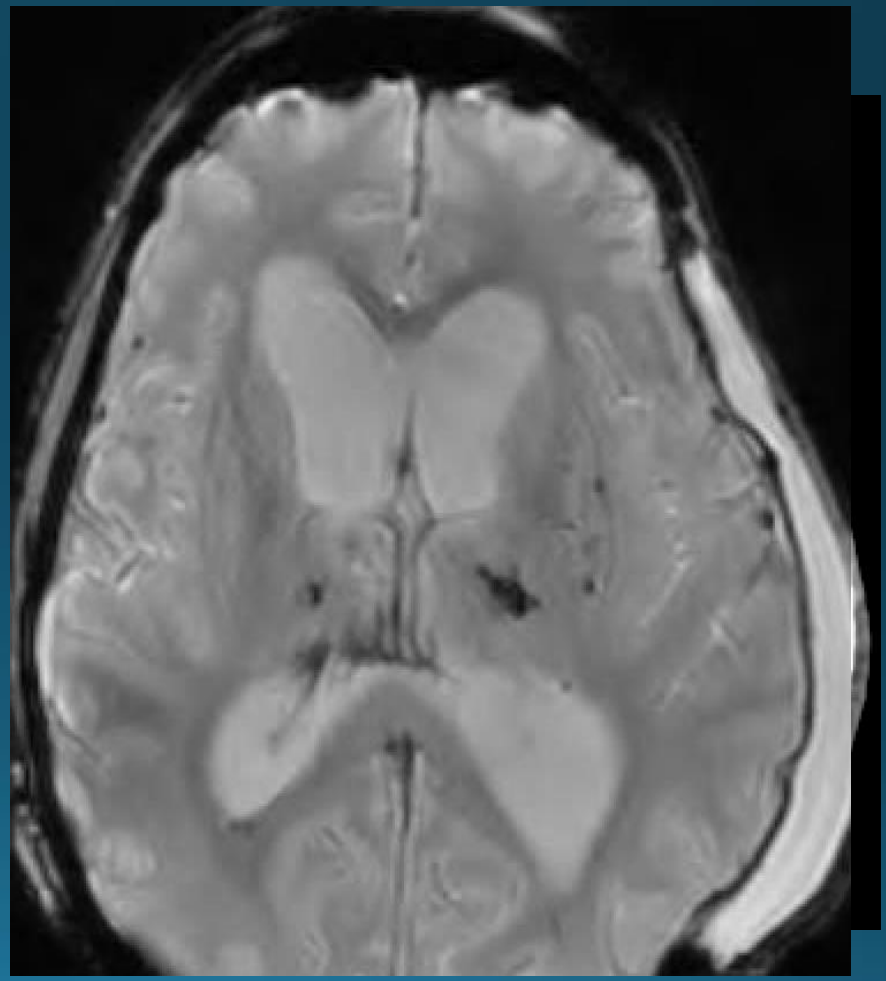
GRE

Diffuse Axonal Injury

43-year-old with severe head trauma, unconscious



Axial GRE

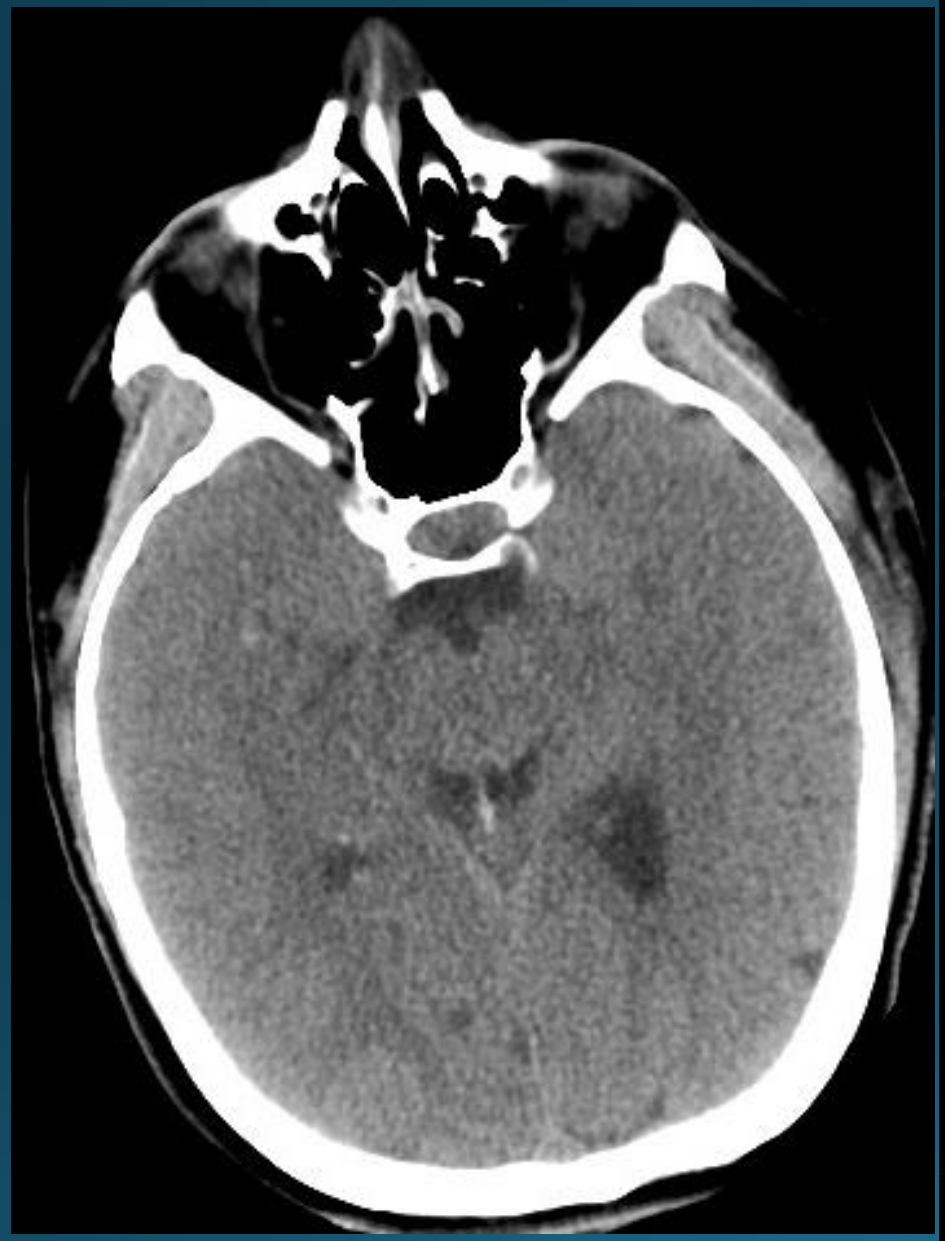


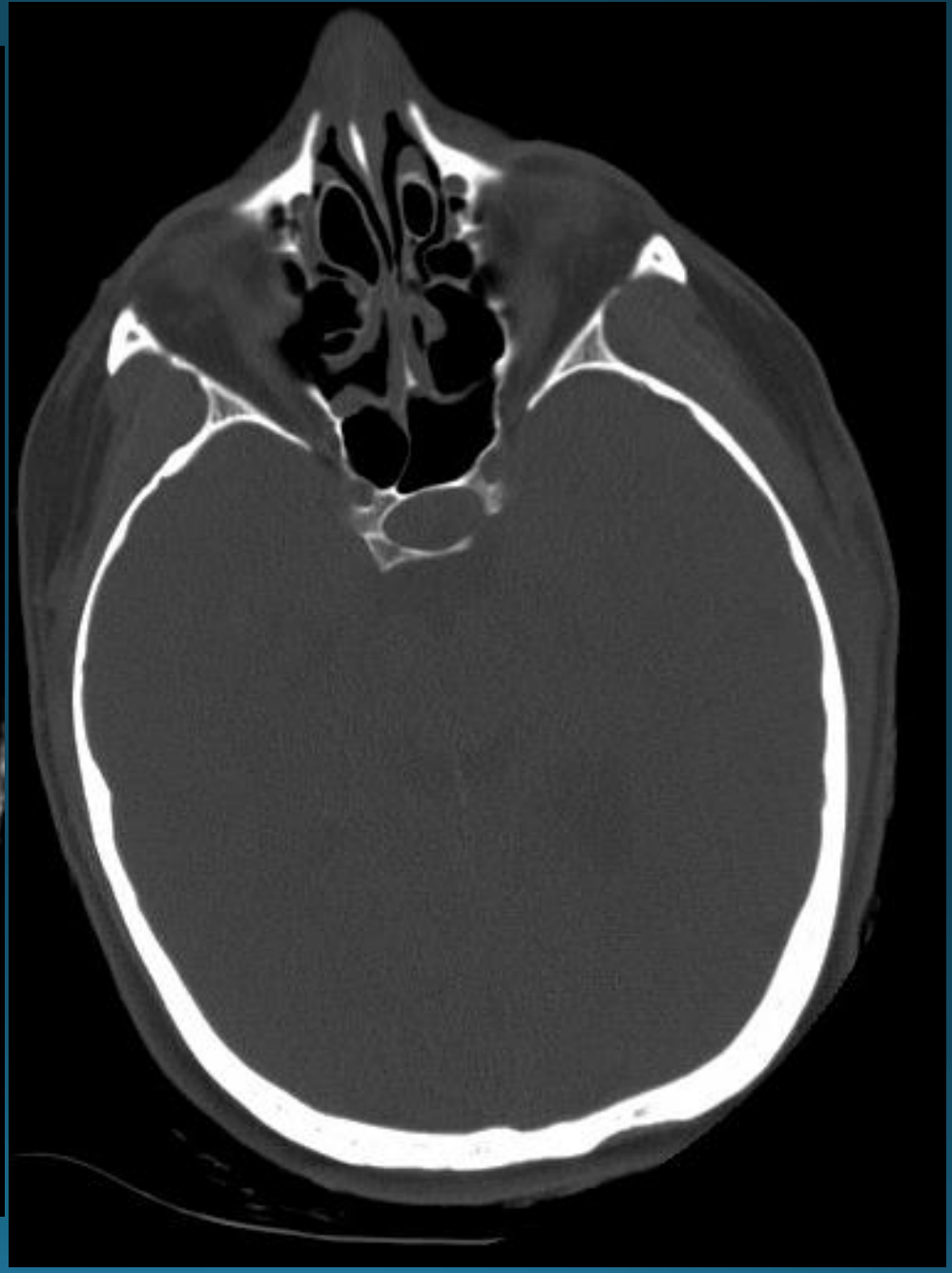
Axial GRE

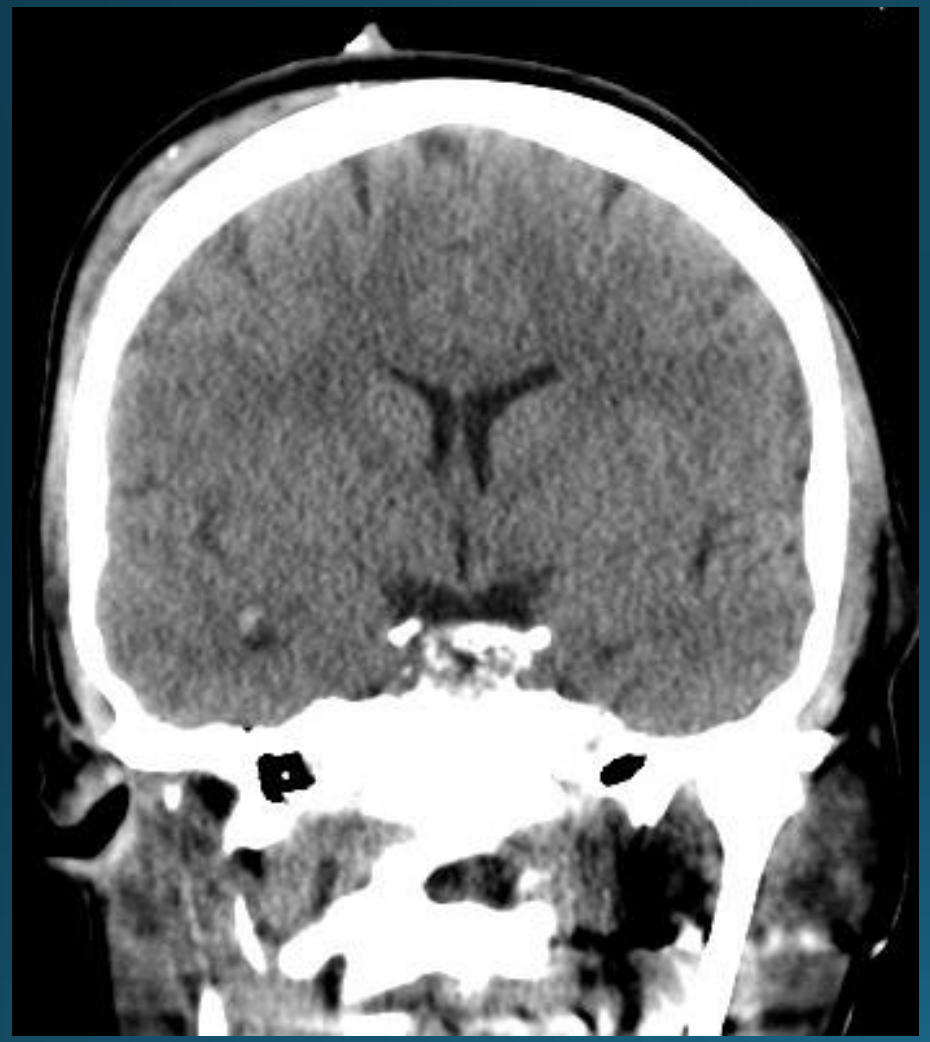
CT and MR SWAN GRE in TBI

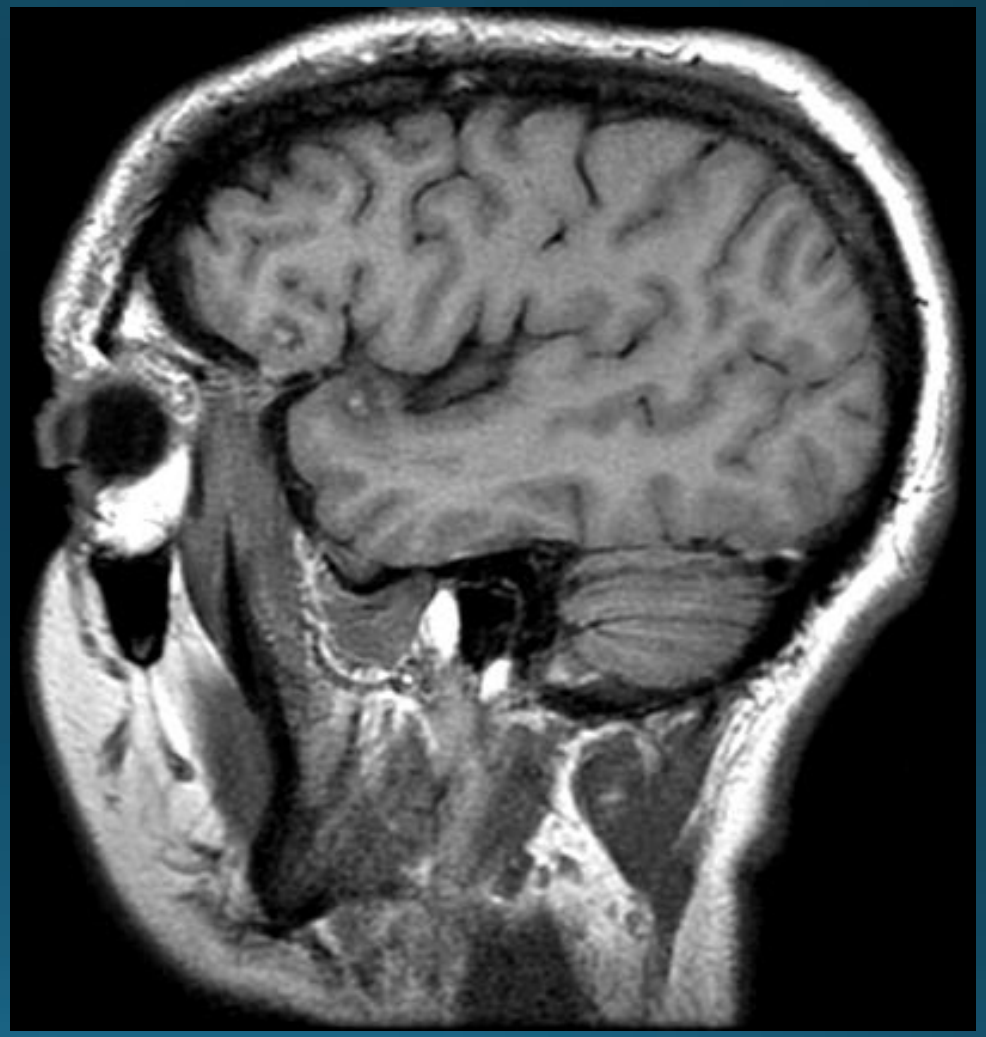
HPI:

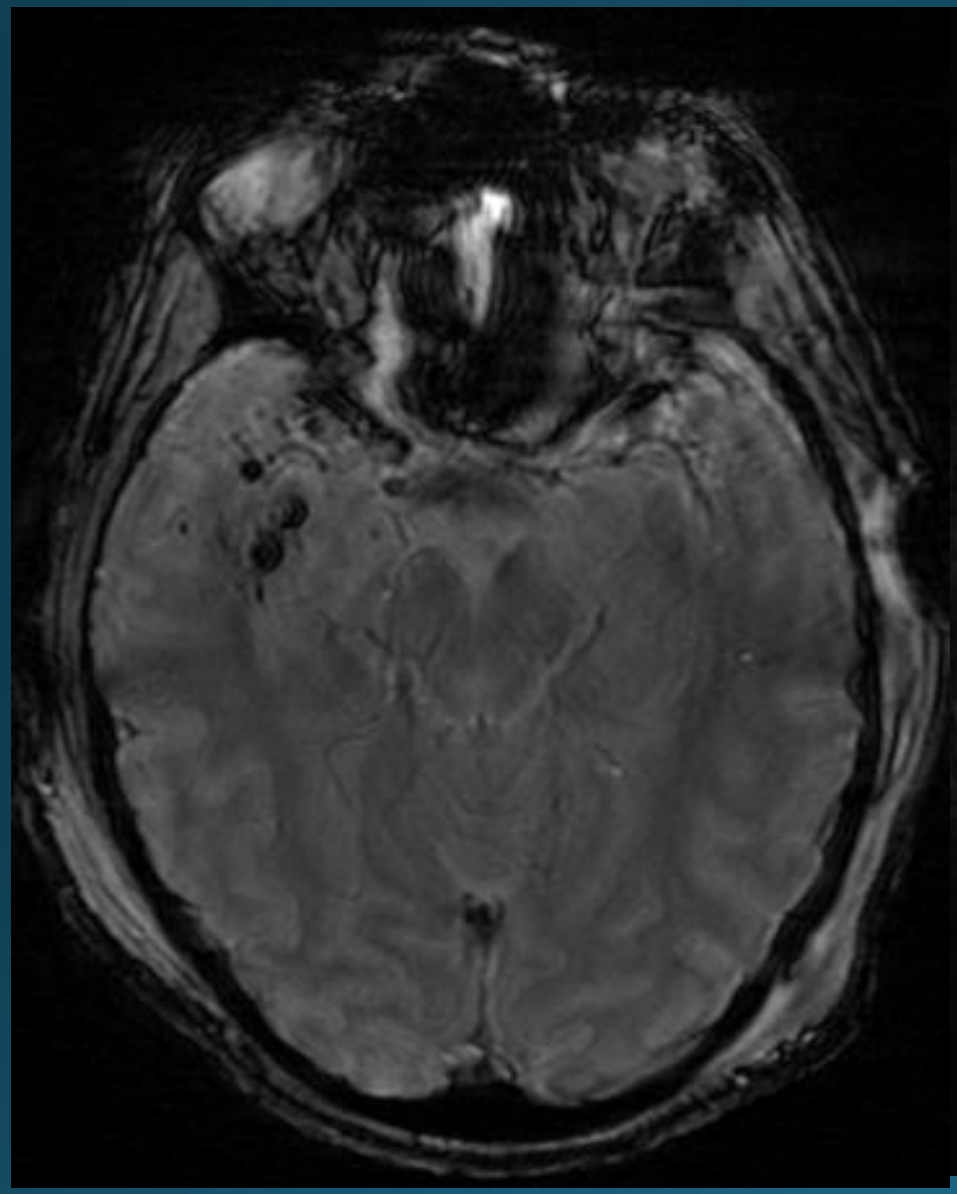
- Evaluate for mental status changes post trauma
- TBI; evaluate vascular injury seen on CT cervical spine
- Trauma; head injury.



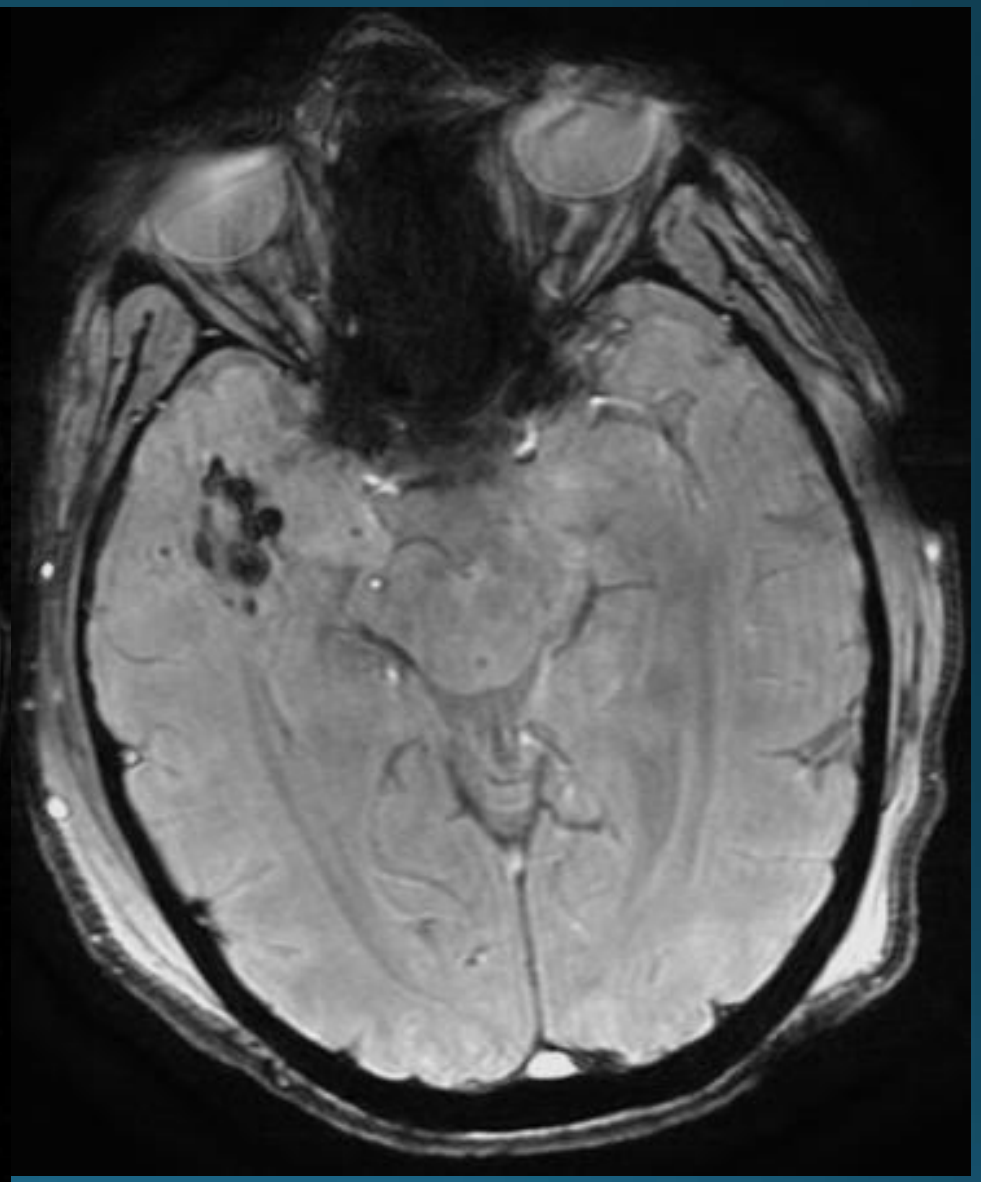








GRE



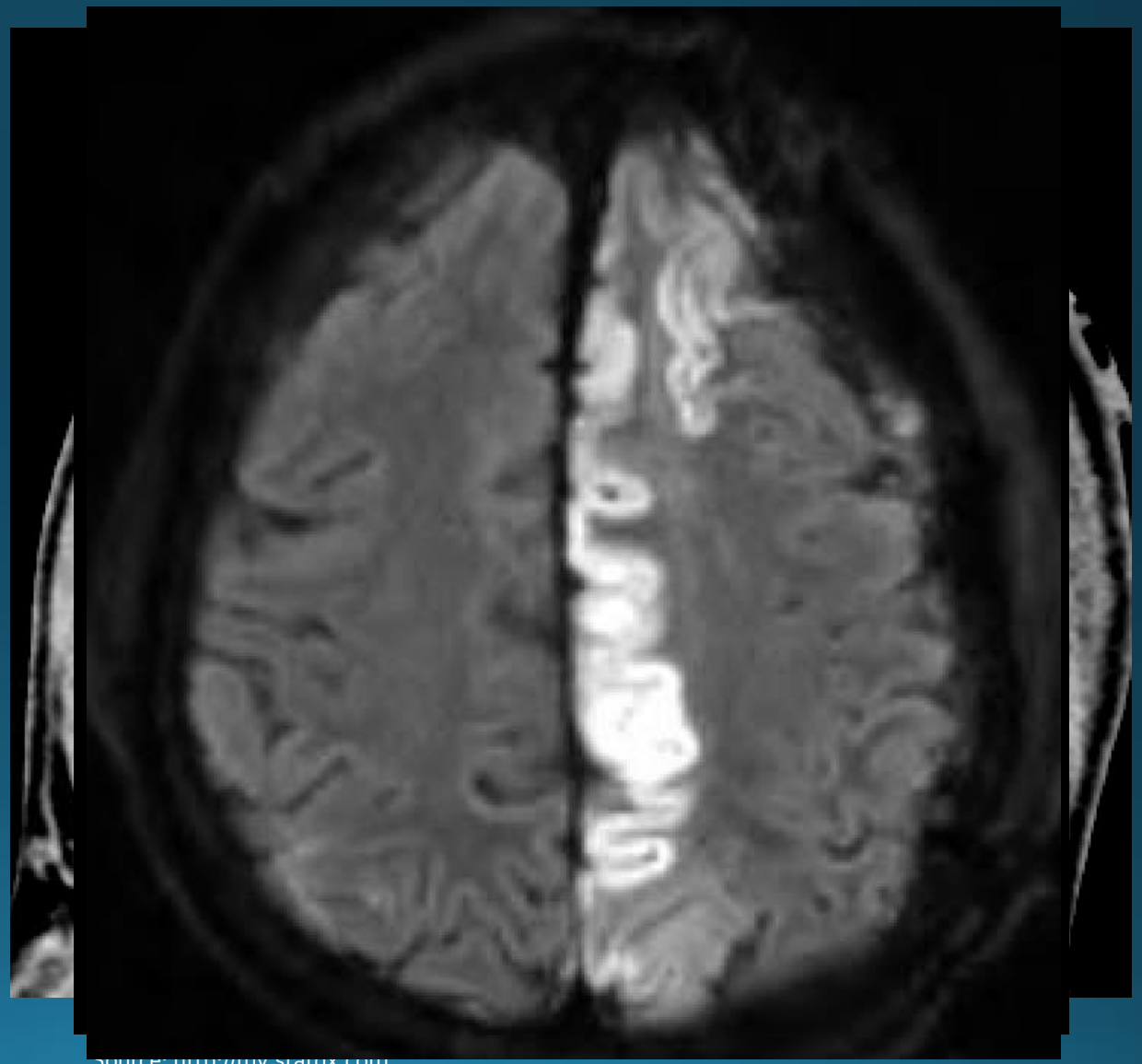
SWAN

Final Topic Herniations

- Subfalcine
- Uncal
- Descending transtentorial
- Ascending transtentorial
- Tonsilar
- Alar

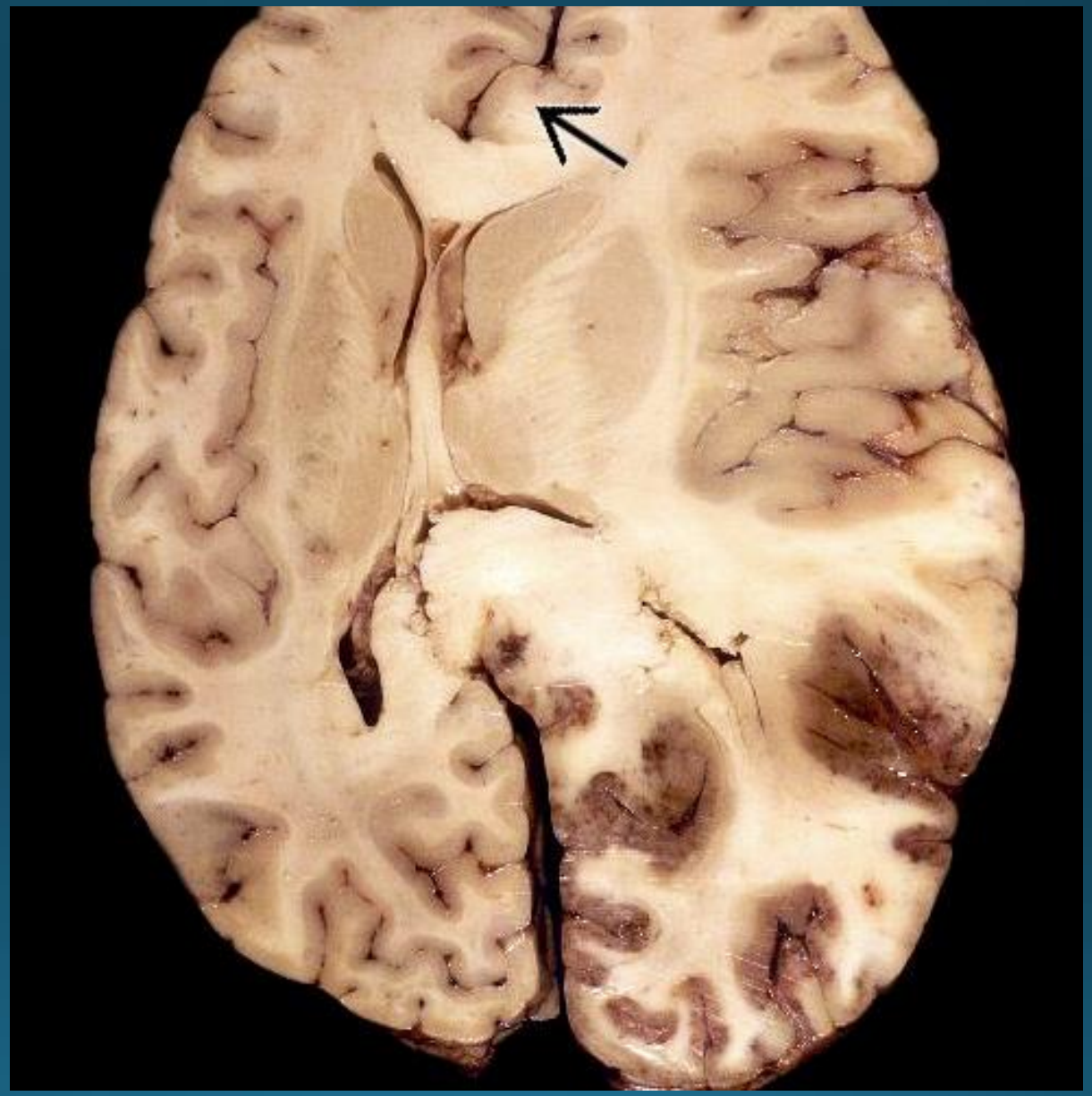
Subfalcine Herniation

- Features:
  - Most common cerebral herniation
  - The affected hemisphere extends across the midline under the "free margin" of the falx
- Complications:
  - Unilateral hydrocephalus (occlusion of foramen of Monro)
  - Secondary anterior cerebral artery infarction



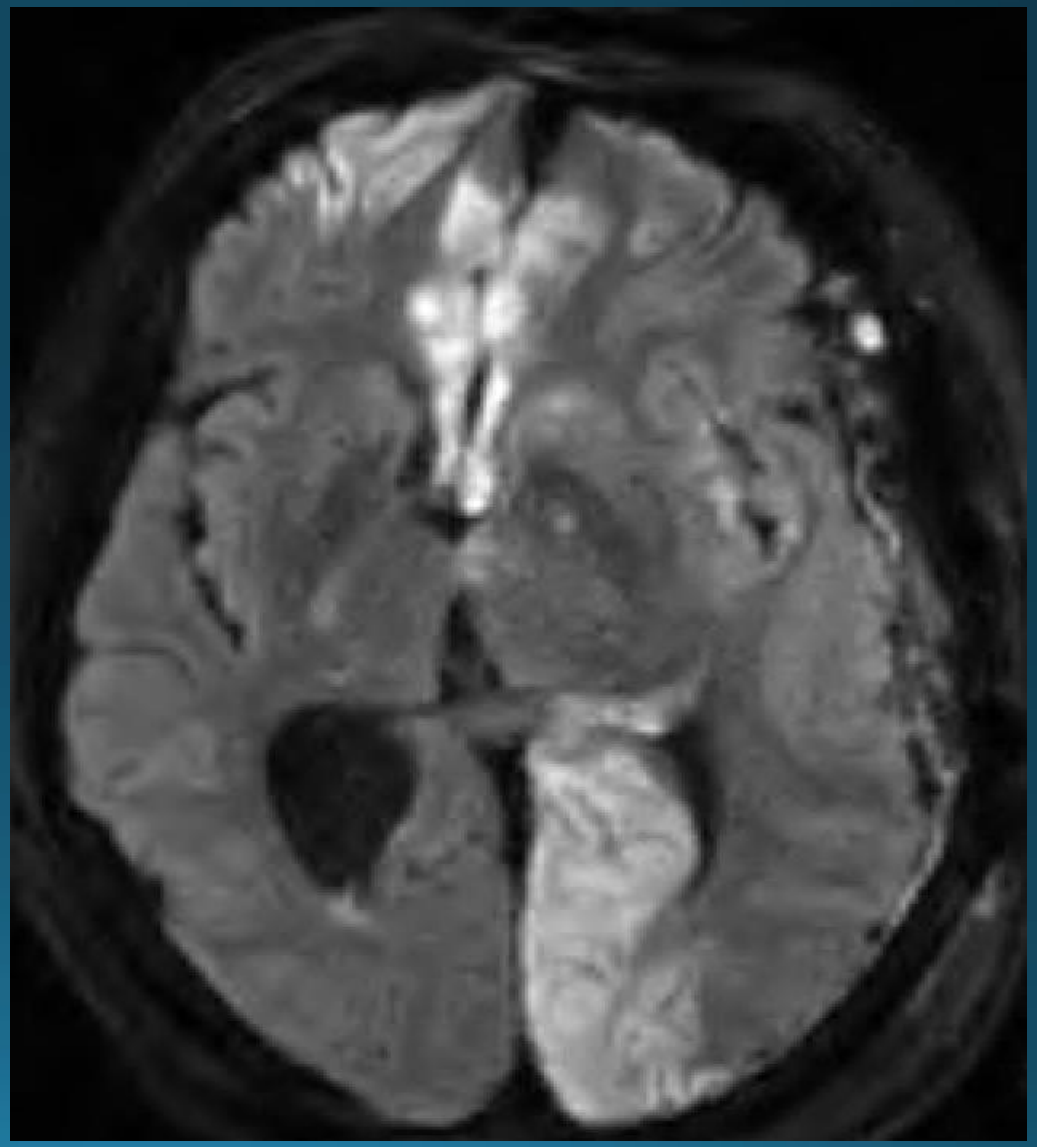
Source: http://my.statdx.com

Subfalcine



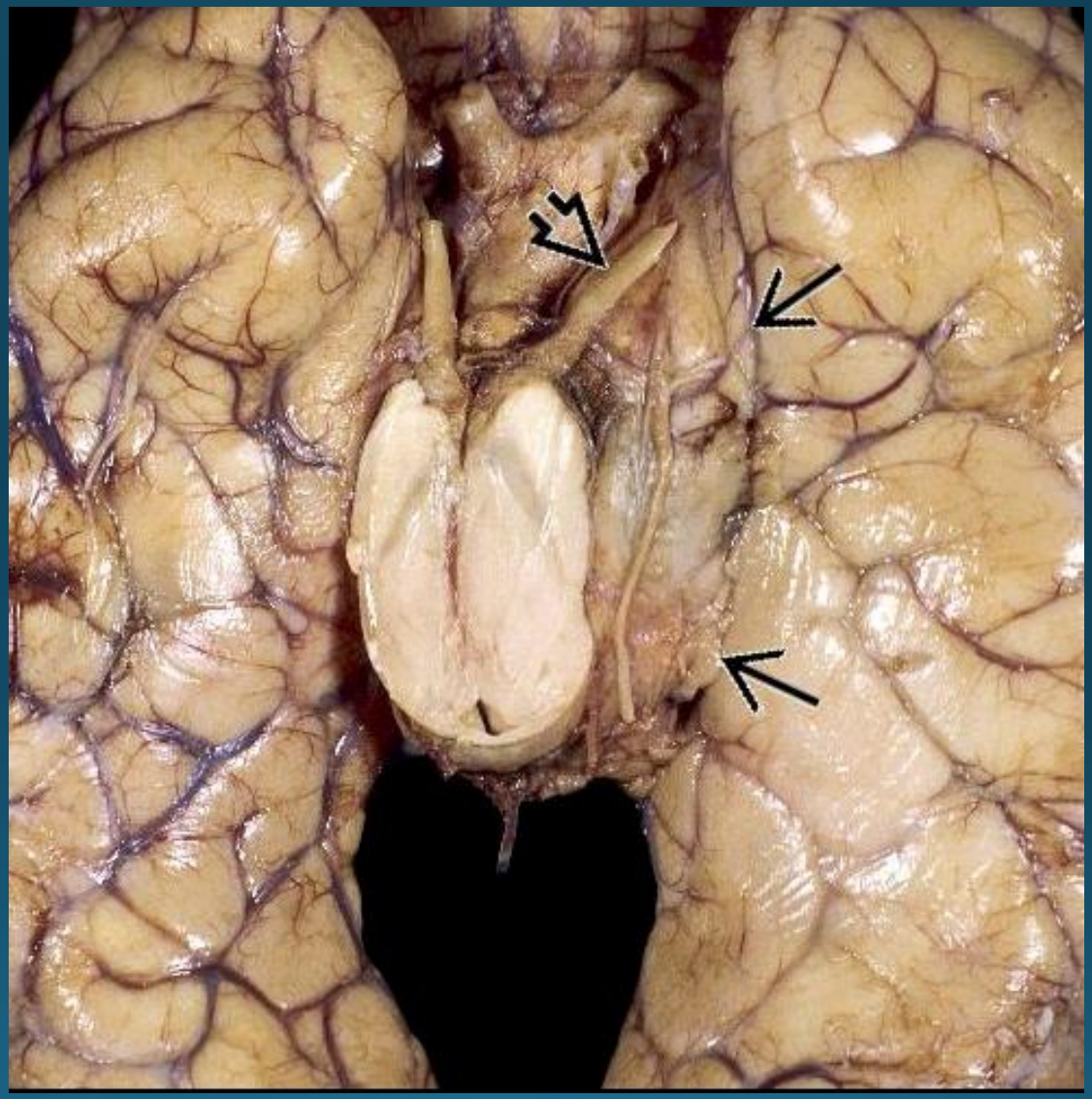
Uncal Herniation

- Features:
  - Second most common cerebral herniation
  - The uncus of the temporal lobe is pushed medially, encroaching on the supracellar cistern
  - The hippocampus follows and effaces the quadrigeminal cistern
  - With progressive mass effect, both structures herniate inferiorly though the tentorial incisura
- Complications:
  - CN III compression
  - Secondary occipital (PCA) and basal infarcts



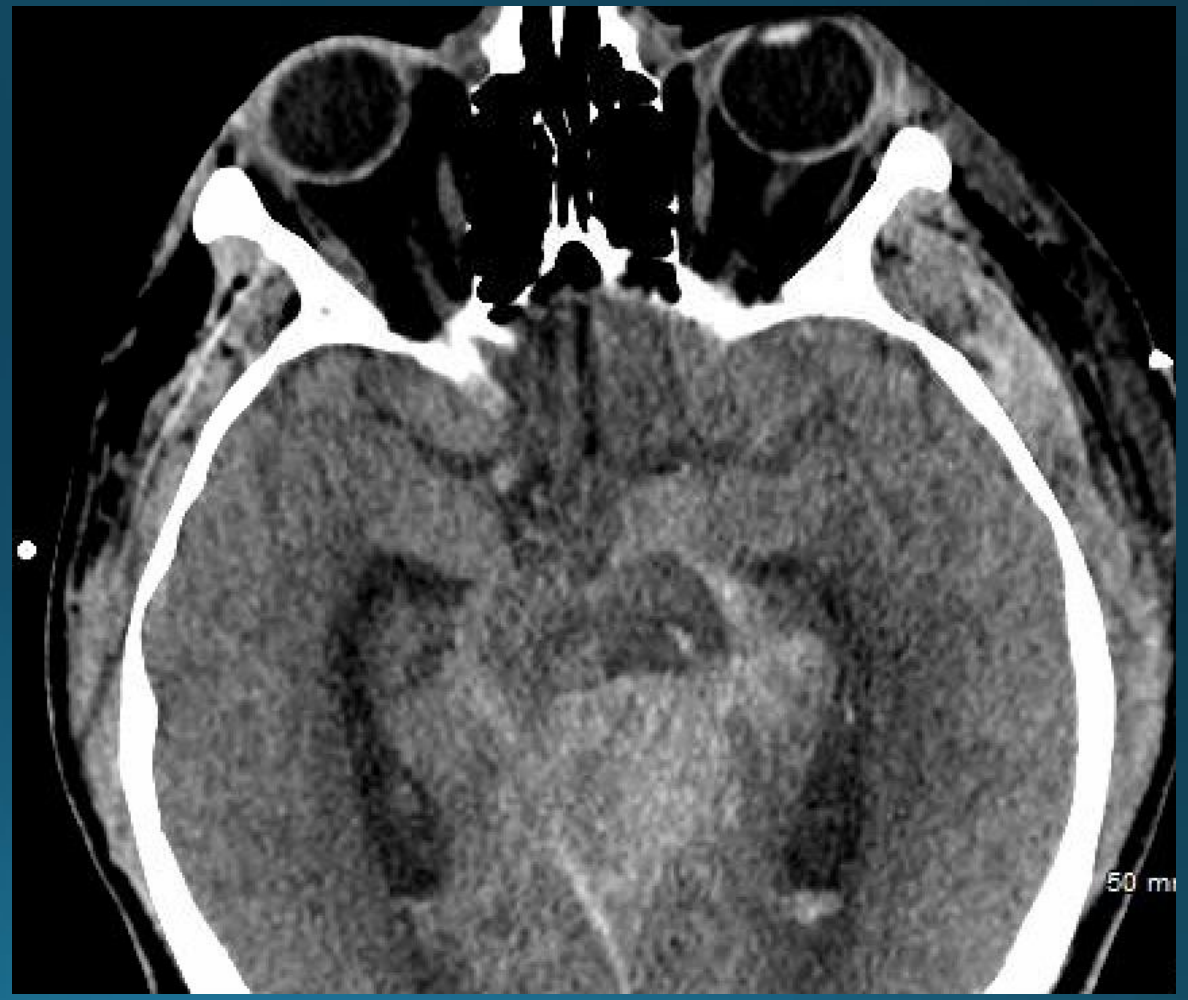
Uncal

Grooving from compression along the tentorium,
3rd nerve compression,
and midbrain compression



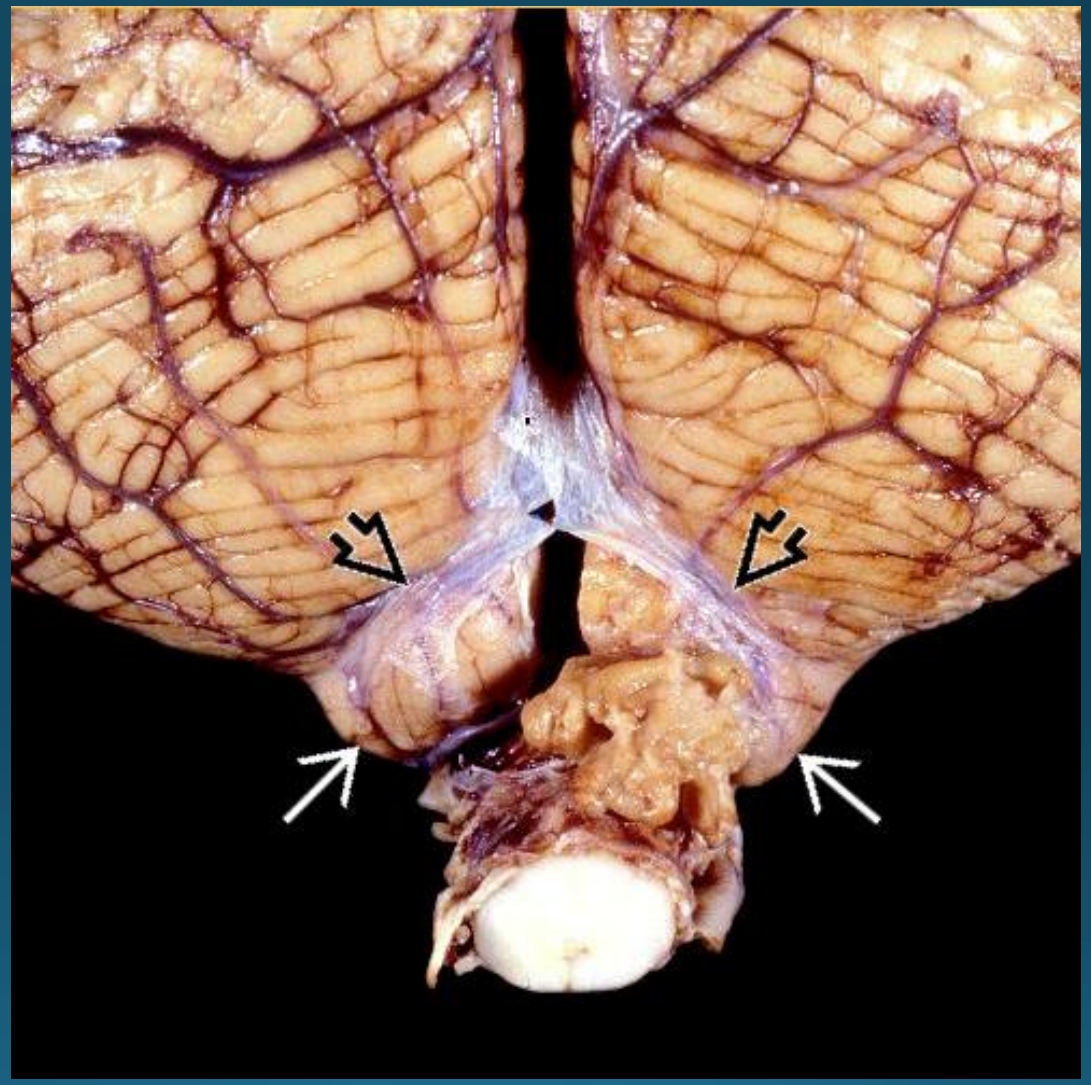
Ascending Transtentorial Herniation

- Features:
  - Caused by an expanding posterior fossa mass, more often neoplasm than trauma
  - The cerebellar vermis and hemisphere(s) are pushed upward though the tentorial incisura
  - There is flattening/effacement of the quadrigeminal cistern and compression of the midbrain
- Complications:
  - Acute obstructive hydrocephalus



Tonsillar

Grooving from compression at the foramen magnum



Thank you!