CENTRAL NERVOUS SYSTEM TRAUMA

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Acute cell injury

- Refers to a spectrum of changes that accompany acute insults and reflect the earliest morphologic markers of neuronal cell death.
- “Red neurons” are evident by about 12 to 24 hours after an irreversible insult.
- The morphologic features consist of shrinkage of the cell body, pyknosis of the nucleus, disappearance of the nucleolus, and loss of Nissl substance, with intense eosinophilia of the cytoplasm.

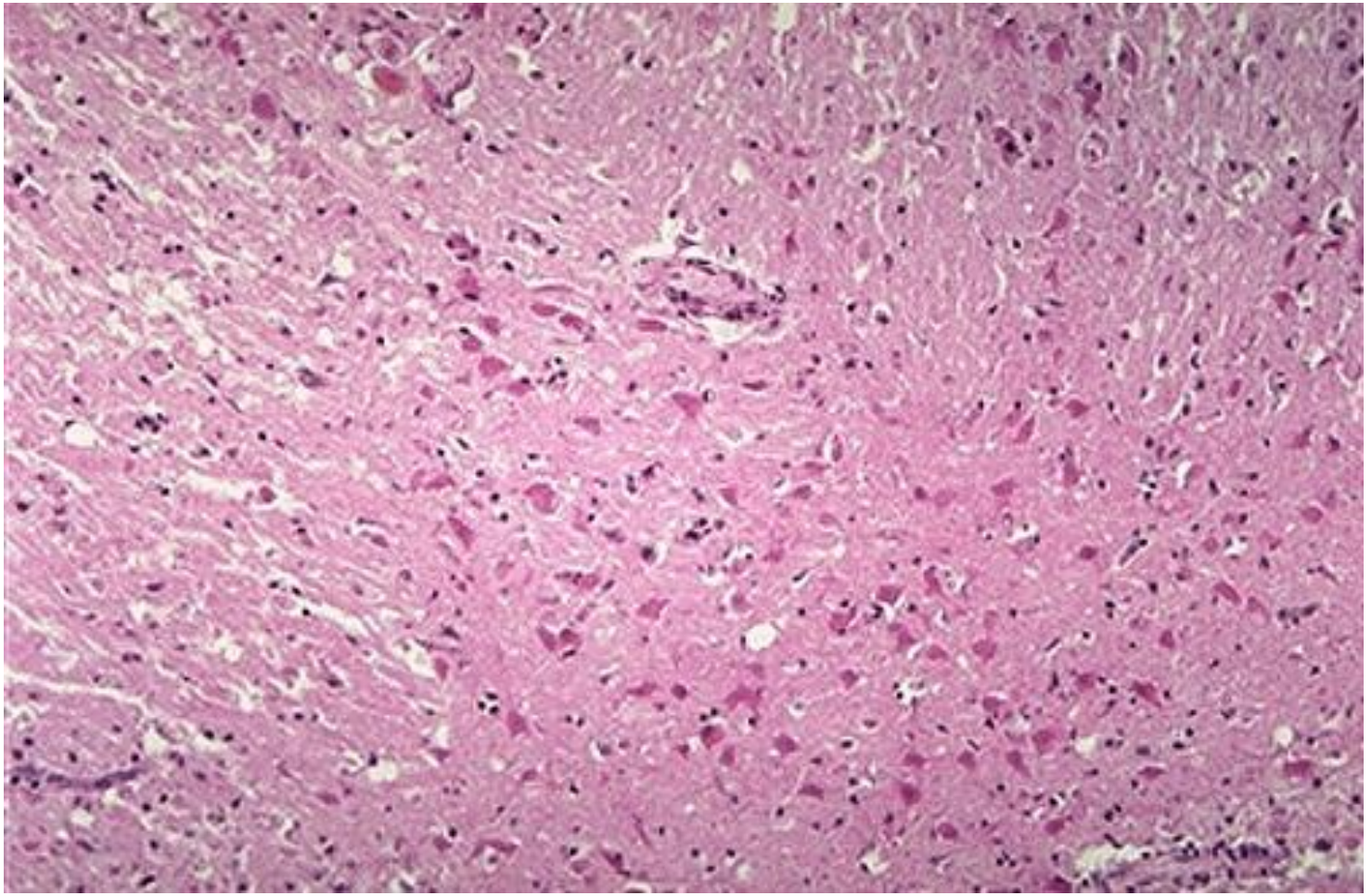
Acute cell injury

• 12 to 24 hours after an irreversible hypoxic/ischemic insult:

•The morphologic features consist of:

- Shrinkage of the neuron cell body,
- Pyknosis of the nucleus,
- Disappearance of the nucleolus,
- Loss of Nissl substance, and
- Intense cytoplasmic eosinophilia ("Red neurons").

Acute cell injury



https://webpath.med.utah.edu/CNSHTML/CNS048.html
Accessed 11/20/2019

Acute cell injury

- Astrocytes act as metabolic buffers and detoxifiers within the brain.
- Foot processes surround capillaries or extend to the subpial and subependymal zones
- Contribute to barrier functions controlling the flow of macromolecules between the blood, the cerebrospinal fluid (CSF), and the brain.

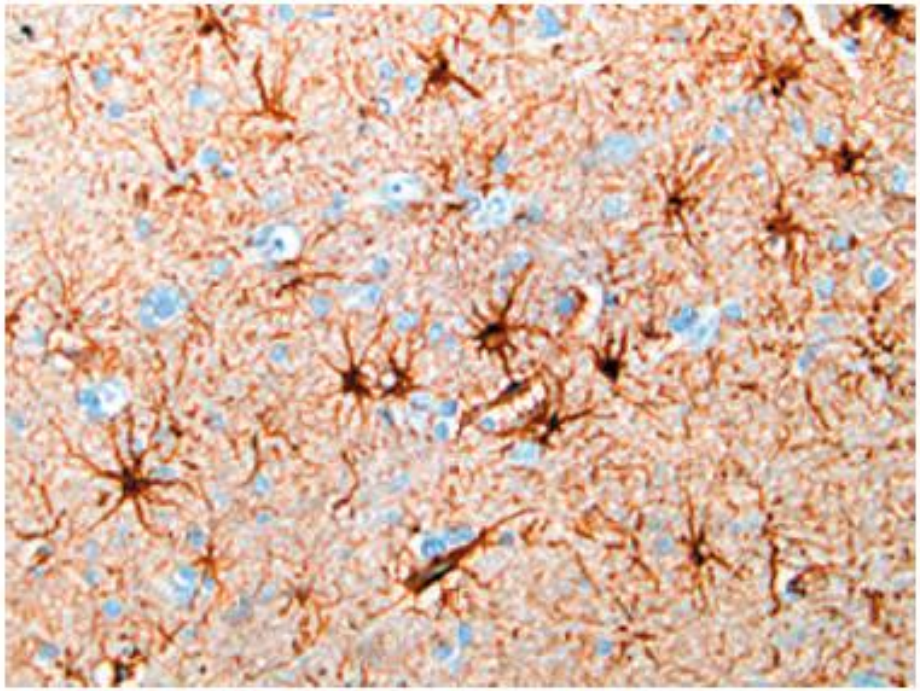


Figure 28-1 Astrocytes and their processes. Immunohistochemical staining for glial fibrillary acidic protein reveals astrocytic perinuclear cytoplasm and well-developed processes *(brown)*.

Acute cell injury

- Astrocyte nuclei become vesicular, and develop prominent nucleoli.
- Cytoplasm expands to a bright pink, somewhat irregular swath around an eccentric nucleus, from which emerge numerous stout, ramifying processes (gemistocytic change).

Acute cell injury

- Axonal reaction
- The cell body enlarges
- peripheral displacement of the nucleus,
- enlargement of the nucleolus, and
- dispersion of Nissl substance from the center to the periphery of the cell (central chromatolysis).
- Neuronal inclusions

Acute cell injury

- As many as 50% of individuals who develop coma shortly after trauma, even without cerebral contusions, are believed to have diffuse axonal injury.
- From angular acceleration alone,
- Trauma may also affect the deep white matter regions as well as the cerebral peduncles, Superior colliculi, and deep reticular formation in the brainstem.

Subacute and chronic injury

- Refers to neuronal death occurring as a result of a progressive disease of some duration
- The characteristic histologic feature is cell loss
- Involves functionally related groups of neurons
- Reactive gliosis
- Hypertrophy and hyperplasia of astrocytes
- Reactive glial changes are often the best indicator of neuronal injury
- Neuronal damage may be associated with a wide range of subcellular alterations in the neuronal organelles and cytoskeleton.

Subacute and chronic injury

- Rosenthal fibers are thick, elongated, brightly eosinophilic, irregular structures that occur within astrocytic processes
- Contain two heat-shock proteins (αB-crystallin and hsp27) as well as ubiquitin.
- Rosenthal fibers are typically found in regions of longstanding gliosis
- As well in Alexander disease (a leukodystrophy associated with mutation in GFAP gene)

Subacute and chronic injury

- Corpora amylacea are polyglucosan bodies.
- They consist primarily of glycosaminoglycan polymers, as well as heat-shock proteins and ubiquitin.
- Round, faintly basophilic, periodic acid-Schiff (PAS)-positive, concentrically lamellated structures that are located wherever there are astrocytic end processes, especially in the subpial and perivascular zones.
- Represent a degenerative change in astrocyte
- LaFora bodies of similar origin and composition

Subacute and chronic injury

- Microglia serve as the resident macrophages of the CNS (CR3 and CD68 positive).
- They respond to injury by:
- (1) Proliferating
- (2) Developing elongated nuclei *(*rod cells*)*
- (3) Forming aggregates around small foci of tissue necrosis *(*microglial nodules*)*

Subacute and chronic injury

- (4) Congregating around cell bodies of dying neurons *(*neuronophagia*)*.
- In addition to resident microglia, blood-derived macrophages may also be present in inflammatory foci.

Subacute and chronic injury

- The Alzheimer type II astrocyte is mainly seen in individuals with long-standing hyperammonemia
- A gray matter cell with a large nucleus, pale-staining central chromatin, an intranuclear glycogen droplet, and a prominent nuclear membrane and nucleolus.
- Oligodendrocytes are cells that wrap their cytoplasmic processes around axons and form myelin.

Subacute and chronic injury

- Injury or apoptosis of oligodendroglial cells is a feature of acquired demyelinating disorders and leukodystrophies.
- Oligodendroglial nuclei may harbor viral inclusions in progressive multifocal leukoencephalopathy.
- Glial cytoplasmic inclusions, primarily composed of α-synuclein, are found in oligodendrocytes in multiple system atrophy (MSA).

Subacute and chronic neuronal injury

- Axonal reaction is a change observed in the cell body during regeneration of the axon
- There is increased protein synthesis associated with axonal sprouting.
- There is enlargement and rounding up of the cell body, peripheral displacement of the nucleus, enlargement of the nucleolus, and dispersion of Nissl substance from the center to the periphery of the cell (central chromatolysis).

Subacute and chronic injury

- Ependymal cells, the ciliated columnar epithelial cells lining the ventricles, do not have specific patterns of reaction.
- Disruption of the ependymal lining is paired with proliferation of subependymal astrocytes to produce small irregularities on the ventricular surfaces *(*ependymal granulations).

Spinal cord injury

- The acute phase consists of hemorrhage, necrosis, and axonal swelling in the surrounding white matter.
- The lesion tapers above and below the level of injury.
- In time central areas of neuronal destruction becomes cystic and gliotic
- Cord sections above and below the lesion show secondary ascending and descending wallerian degeneration, respectively, involving the long white-matter tracts affected at the site of trauma.

Cerebral edema

- The brain and the spinal cord are encased and protected by the rigid skull and the bony spinal canal.
- The pressure within the cranial cavity may rise in one of three commonly observed clinical settings:
- Generalized brain edema
- Increased CSF volume (hydrocephalus)
- Focally expanding mass lesions.

Cerebral edema

- Vasogenic edema
- Normal blood-brain barrier is disrupted
- Increased vascular permeability occur
- Fluid flows predominantly into the intercellular spaces of the brain.
- The paucity of lymphatics greatly impairs the resorption of excess extracellular fluid.
- May be localized, as when it results from abnormally permeable vessels adjacent to inflammatory disease or neoplasms, or generalized.

Cerebral edema

- Cytotoxic edema
- Increase in intracellular fluid secondary to neuronal, glial, or endothelial cell membrane injury as cell unable to maintain ionic gradient.
- Generalized hypoxic/ischemic or toxic insult
- Conditions associated with generalized edema often have elements of both vasogenic and cytotoxic edema.
- In generalized edema, the gyri are flattened, the intervening sulci are narrowed, and the ventricular cavities are compressed. As the brain expands, herniation may occur.

Cerebral edema

- Interstitial edema (hydrocephalic edema)
- Seen with increased intracranial pressure
- Occurs especially around the lateral ventricles
- Abnormal flow of fluid from the intraventricular cerebrospinal fluid across the ependymal lining to the periventricular white matter
- Pseudotumor cerebri
- Absence of obstructive change
- Headache with papilledema
- No focal neurologic signs
- Associated with all-trans retinoic acid

Hydrocephalus

- The choroid plexus within the ventricular system produces cerebrospinal fluid.
- It normally circulates through the ventricular system and enters the cisterna magna at the base of the brain stem through the foramina of Luschka and Magendie.
- Subarachnoid cerebrospinal fluid bathes the superior cerebral convexities and is absorbed by the arachnoid granulations.

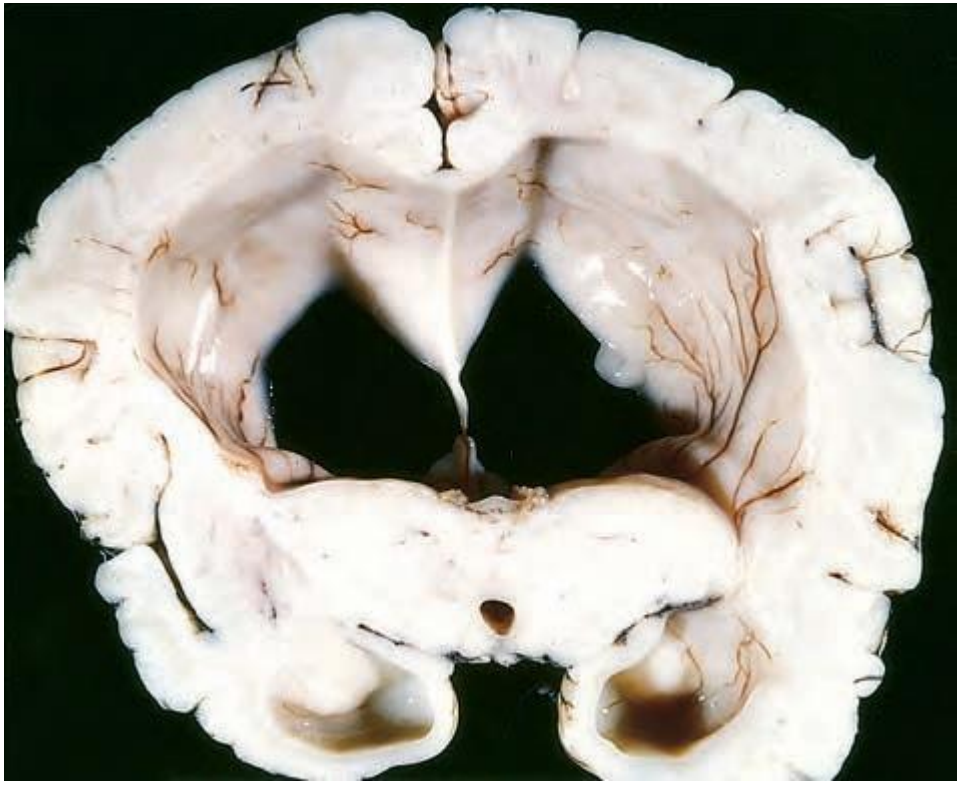
Hydrocephalus

- Hydrocephalus is the accumulation of excessive cerebrospinal fluid within the ventricular system.
- Most cases of hydrocephalus are a consequence of impaired flow and resorption of the fluid;
- Overproduction is a rare cause that can accompany tumors of the choroid plexus.
- Hydrocephalus ex vacuo refers to a compensatory increase in ventricular volume secondary to a loss of brain parenchyma.

Hydrocephalus

- Accumulation of excessive cerebrospinal fluid within the ventricular system.
- If the cranial sutures have not closed, the head enlarges.
- If the cranial sutures have not closed, neurologic changes are related to increased intracranial pressure.
- Enlargement of the ventricular system generally is seen in communicating hydrocephalus
- The ventricular system is in communication with the subarachnoid space.

Hydrocephalus



Frosch, MP, Anthony, DC, De Girolami, U, “The Central Nervous System,” in Kumar, V, Abbas, AK, Aster, JC, (eds), Robbins and Cotran Pathologic Basis of Disease (9th ed.), Elsevier. Philadelphia. (2015) Fig. 28-2 Accessed 10/25/2019

Non-communicating hydrocephalus

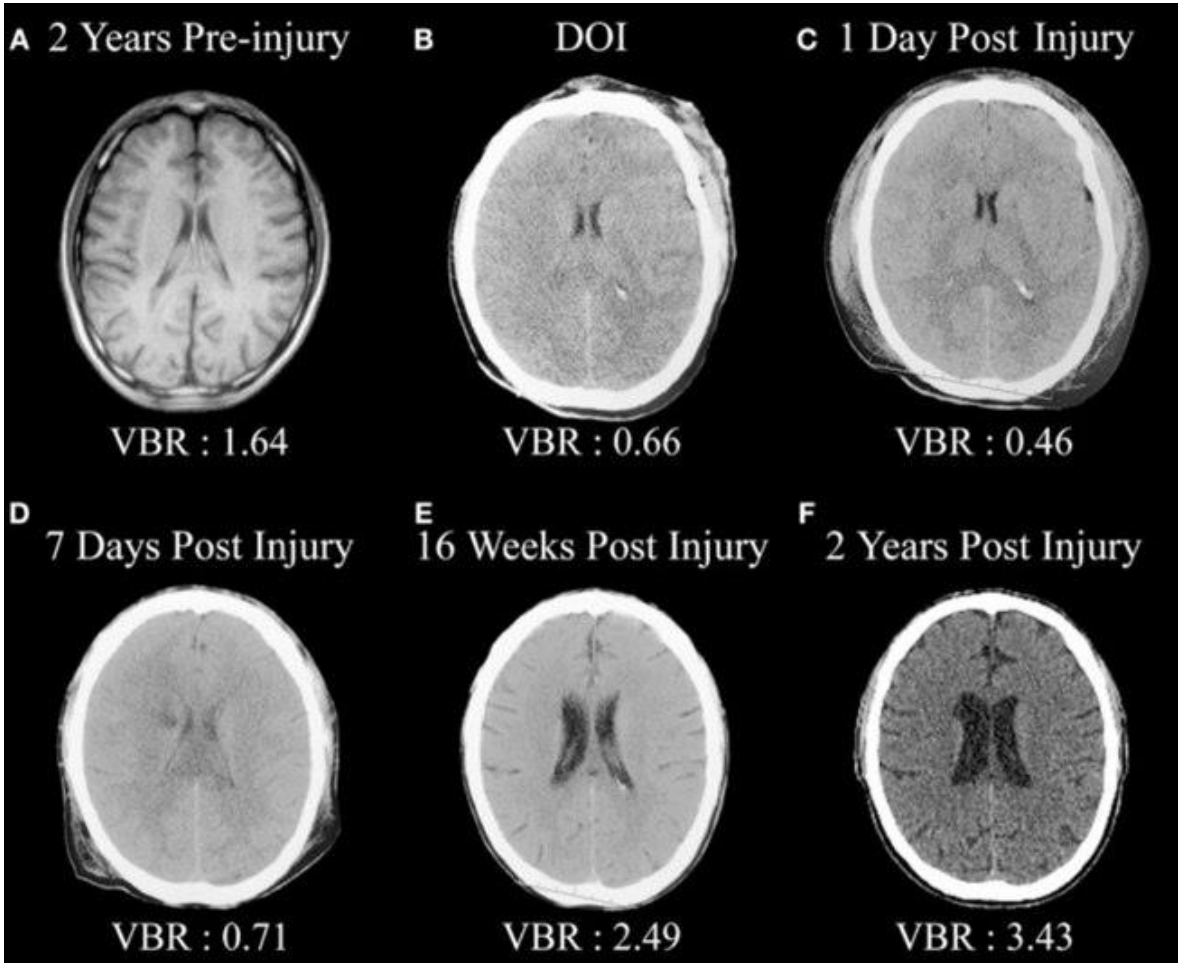
- Congenital atresia of the Aqueduct of Sylvus
- Most common cause of hydrocephalus.
- Stenosis may result after ependymitis (viral infection acquired transplacentally).
- Dandy-Walker malformation
- Cerebellar vermis rudimentary or absent
- Cystic dilatation of fourth ventricle
- May see dysplastic change of brain stem nuclei
- Tumor obstruction
- Shunt

TRAUMA

Concussion

- Transient loss of consciousness due to trauma to reticular formation in brainstem.
- Boxing knockout blow deflects head up and posteriorly; causes torque on brainstem leading to functional paralysis and temporary respiratory arrest.
- Amnesia for the event may persist.
- Blow to the temporal skull may cause fracture but not loss of consciousness as lateral movement is inhibited by the Falx.
- (Torque is Force times distance from axis of rotation times sine of the angle. Newton-meters.)

Evolution of traumatic brain injury



https://reference.medscape.com/slideshow/traumatic-brain-injuries-6009819#13
Accessed 03/20/2020 Acute subdural

Skull fracture

- Fractures occur when the elastic limit of the bone has been exceeded.
- Breaks in the inner table precede those of the outer table in a linear fracture.
- Breaks in the outer table precede those of the inner table in a depressed fracture.
- A displaced fracture involves displacement of bone into the cortical cavity by a distance greater than the length of the bone.

Skull fracture

- The formation of a skull fracture depends upon:
- The force applied,
- The point of impact (thickness of the skull),
- The direction of impact, and
- Whether the head is in motion at the time of the injury.
- Temporal bones thinnest (4mm thick).

Skull fracture

- Newton (N) is a measure of force. Mass times acceleration.
- A 70 kg (154#) man exerts around 700 N (515 foot-pounds) force against gravity, and 1000 N (737 foot-pounds) of force in a static push.
- A heavyweight boxer can deliver 5000 N (3685 foot-pounds) of force with a single punch because of the velocity of the fist.

Skull fracture

- The average adult head weighs 4.5Kg (10 pounds)
- A simple fracture can occur by walking into a fixed object (force required = 73N or 54 foot pounds)
- A simple fall through 1 m causing a frontal impact (510N or 392 foot-pounds) can also result in linear or mosaic fractures.
- Fractures have been absent when an impact force of 1314 N (or 968 foot-pounds) was recorded.
- (Torque is Force times distance from axis of rotation times sine of the angle. Newton-meters.)

Skull fracture

- The kinetic energy generated that causes a fracture is dissipated at a fused suture. Fractures that cross suture lines are termed diastatic.
- Fracture lines of subsequent injury do not extend across fracture lines of previous injury.
- Not easily diagnosed with plain x-rays.
- Bioengineering studies do not support the mechanisms proposed (rotational injury, tearing of veins) to produce the “shaken baby syndrome”.

Skull fracture

- Frontal impacts are seen following a fall in a syncopal attack as well as walking into a fixed object.
- Occipital impacts are seen following a fall off a ladder and may result is a basilar skull fracture.
- Basilar fractures are characterized by symptoms referable to the lower cranial nerves or the cervicomedullary region, and the presence of orbital or mastoid hematomas distant from the site of impact.
- Cerebrospinal fluid discharge from nose or ear.

Skull fracture

- Injury to the underlying brain may be:
- Silent if in the frontal lobe
- Severely disabling if in the spinal cord
- Fatal if in the brainstem

Contusion

- Traumatic bruise of the brain caused by acceleration of the head in an anterior-posterior direction, or, in the case of a fall, downward direction.
- Permanent neuronal damage.
- Crests are most susceptible to injury.
- Also, tips of temporal and frontal lobes.
- Contusion at site of impact is called a coup injury.
- Contusion at site opposite impact is called a contrecoup injury.

Contusion

- Brain contusions are wedge shaped with base at the site of impact.
- Edema and, later, pericapillary hemorrhage are seen.
- During the next few hours, the extravasation of blood extends throughout the involved tissue, across the width of the cerebral cortex, and into the white matter and subarachnoid space.
- Morphologic evidence of neuronal injury (pyknosis of the nucleus, eosinophilia of the cytoplasm, and disintegration of the cell) takes about 24 hours to appear, although functional deficits may occur earlier.
- Axonal swellings develop in the vicinity of damaged neurons or at great distances away in white matter.

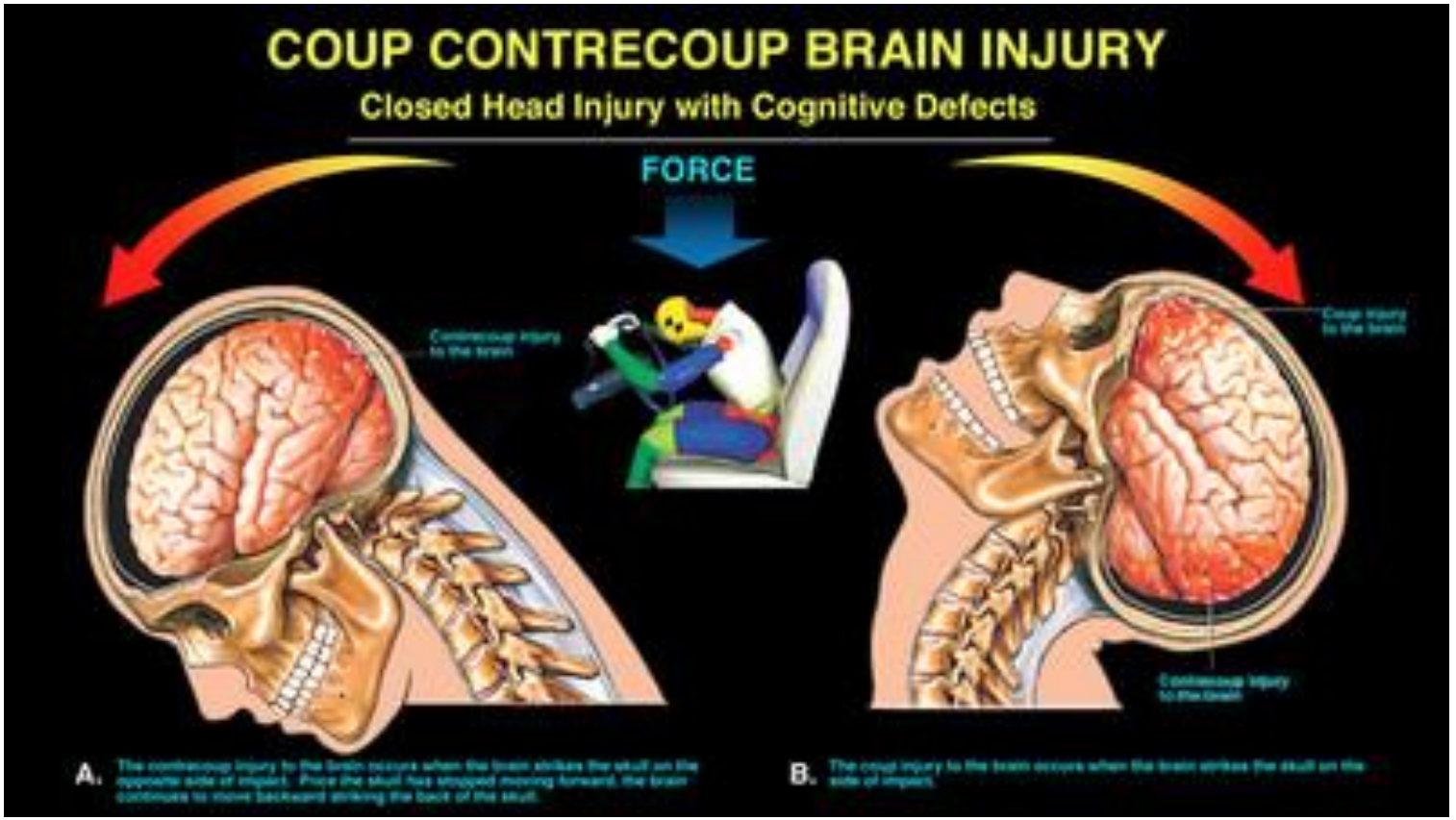
Contusion

- As many as 50% of individuals who develop coma shortly after trauma, even without cerebral contusions, are believed to have diffuse axonal injury.
- Axons are injured by the direct action of mechanical forces, with subsequent alterations in axoplasmic flow.
- Comparable mechanical disruption of axons can result from angular acceleration alone, which can cause diffuse axonal injury even in the absence of impact.
- Later, there is microgliosis and degeneration of the involved fiber tracts.

Contusion

- Old lesions are depressed, retracted, and yellow patches.
- They are found on the crests, most commonly at sites of contrecoup injury (inferior frontal cortex, temporal or occipital lobes).
- The old lesions are called “plaque jaune” and may become epileptogenic foci.

Coup contrecoup injury



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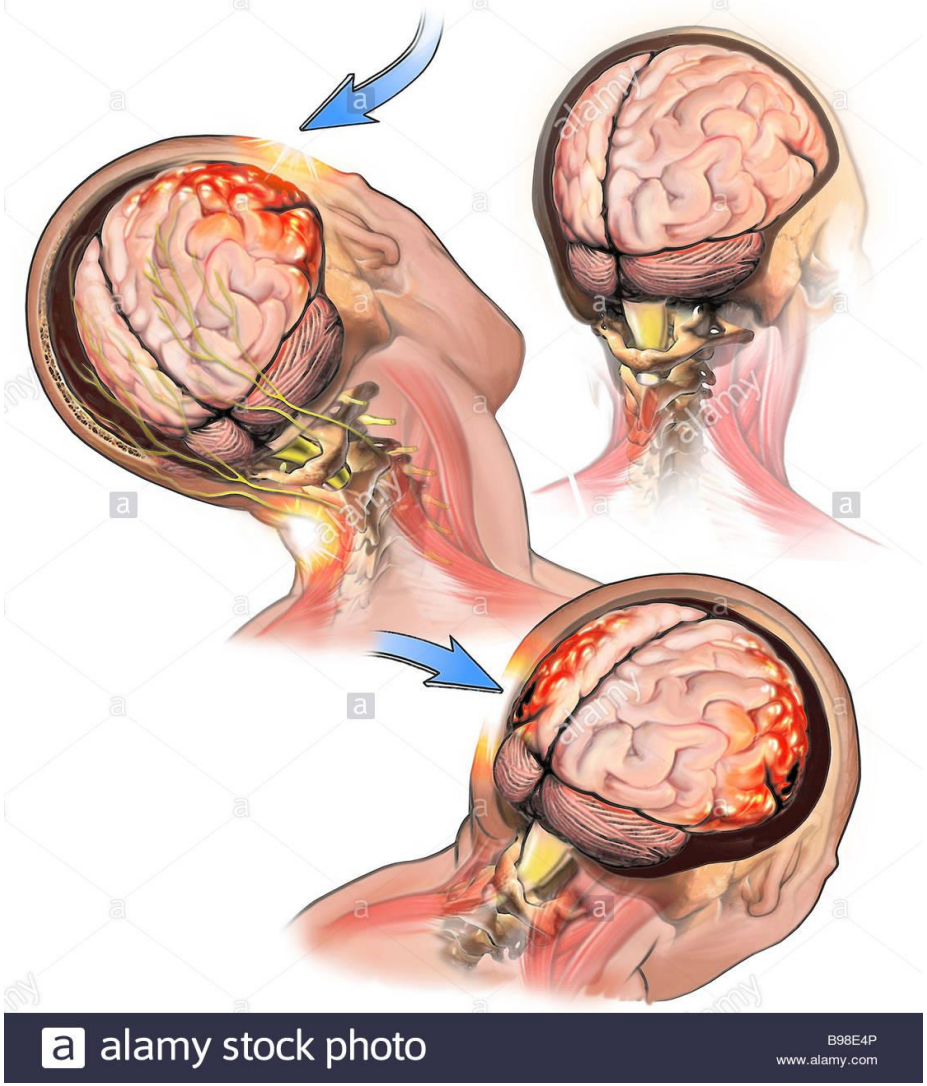
Rotational injury



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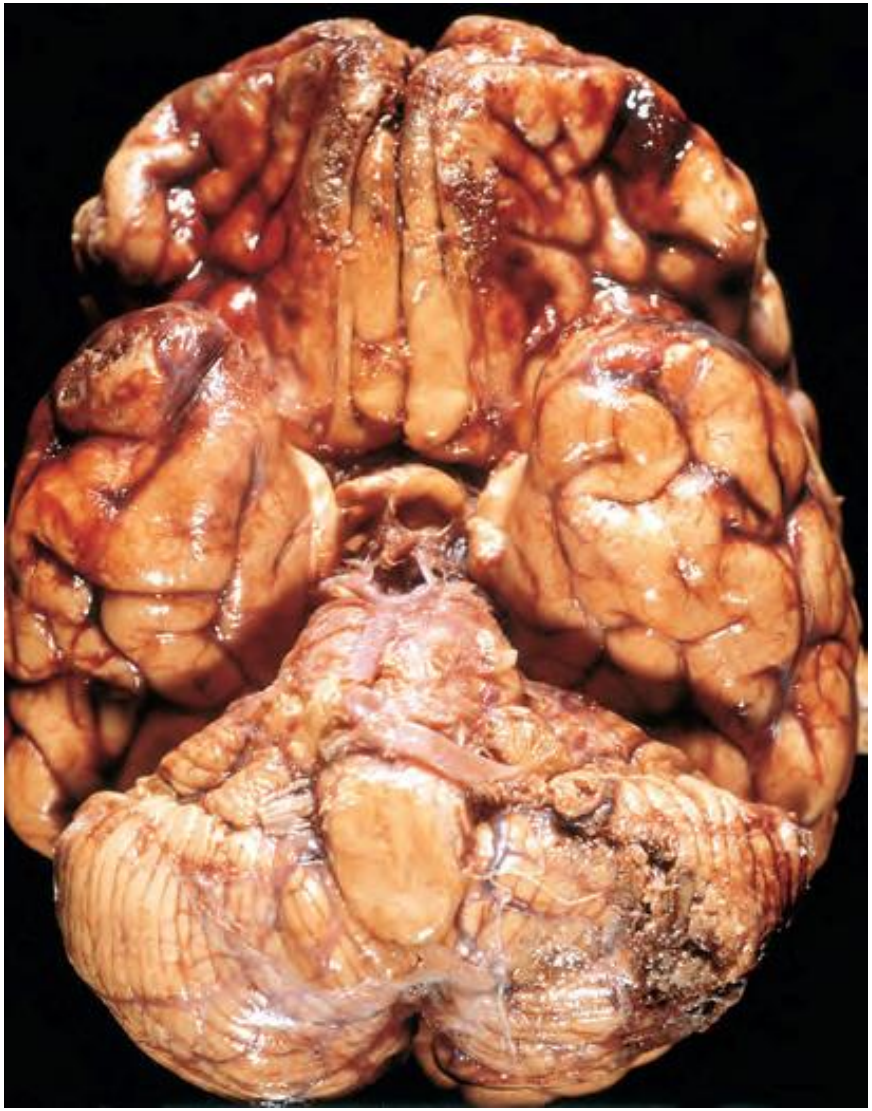
Contusion

Multiple contusions involving the inferior surfaces of frontal lobes, anterior temporal lobes, and cerebellum.



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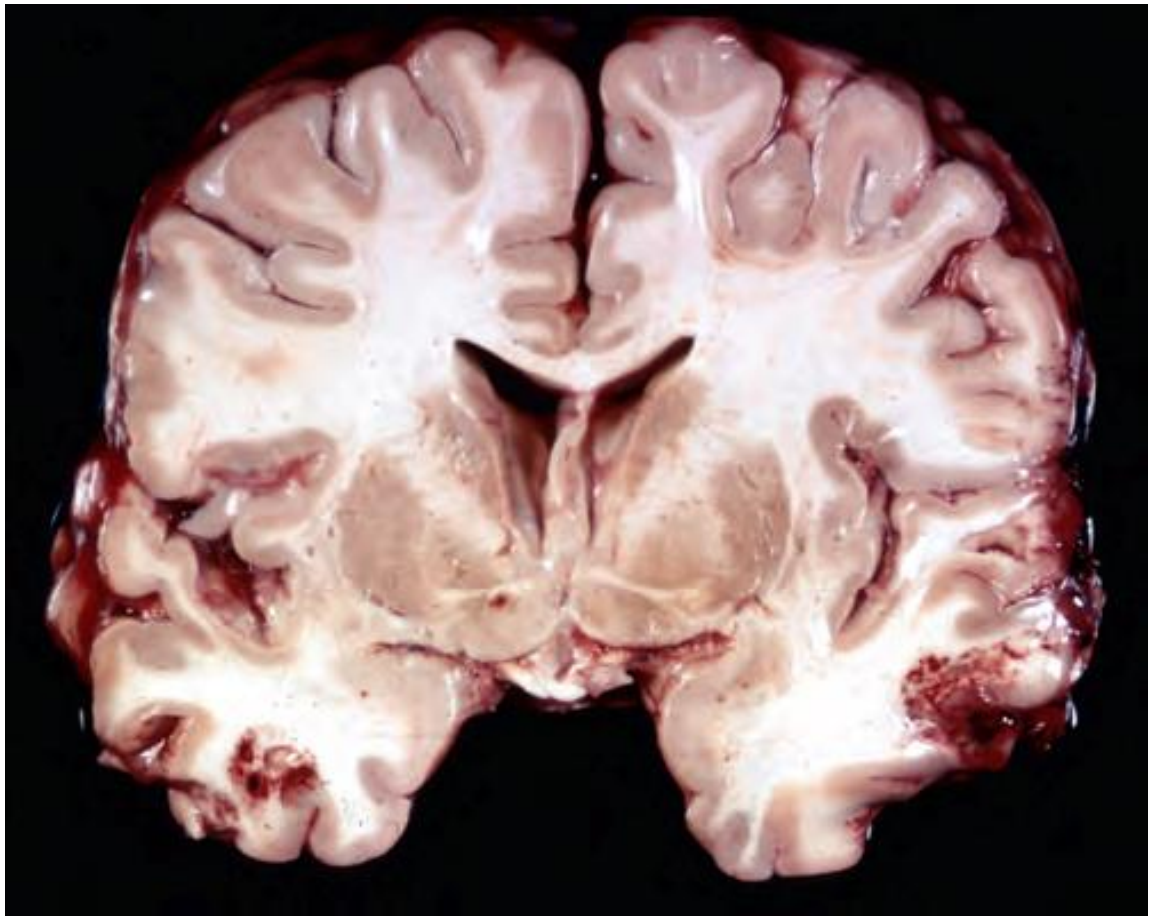
Contusion

Acute contusions are present in both temporal lobes, with areas of hemorrhage and tissue disruption.



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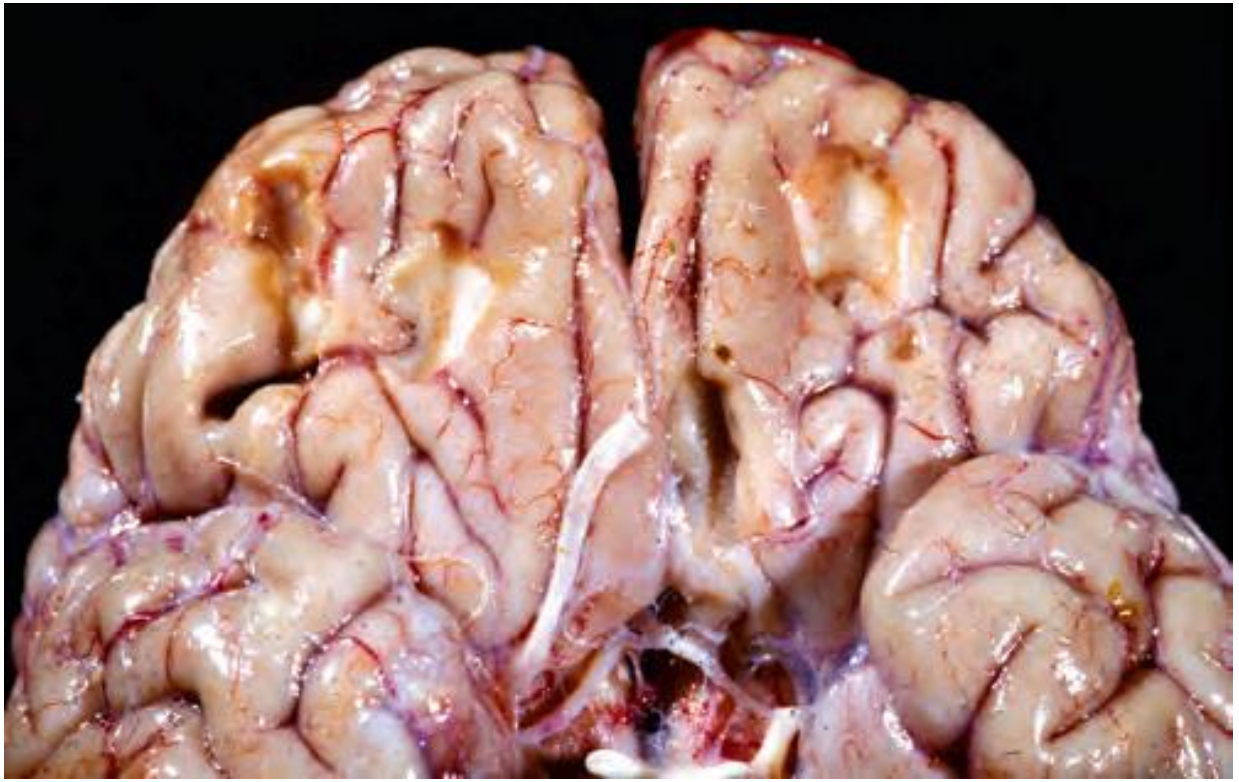
Contusion

Remote contusions are present on the inferior frontal surface of this brain, with a yellow color (associated with the term plaque jaune).



Frosch, MP, Anthony, DC, De Girolami, U, “The Central Nervous System,” in Kumar, V, Abbas, AK, Aster, JC, (eds), Robbins and Cotran Pathologic Basis of Disease (9th ed.), Elsevier. Philadelphia. (2015) Fig. 28-9AC Accessed 10/25/2019

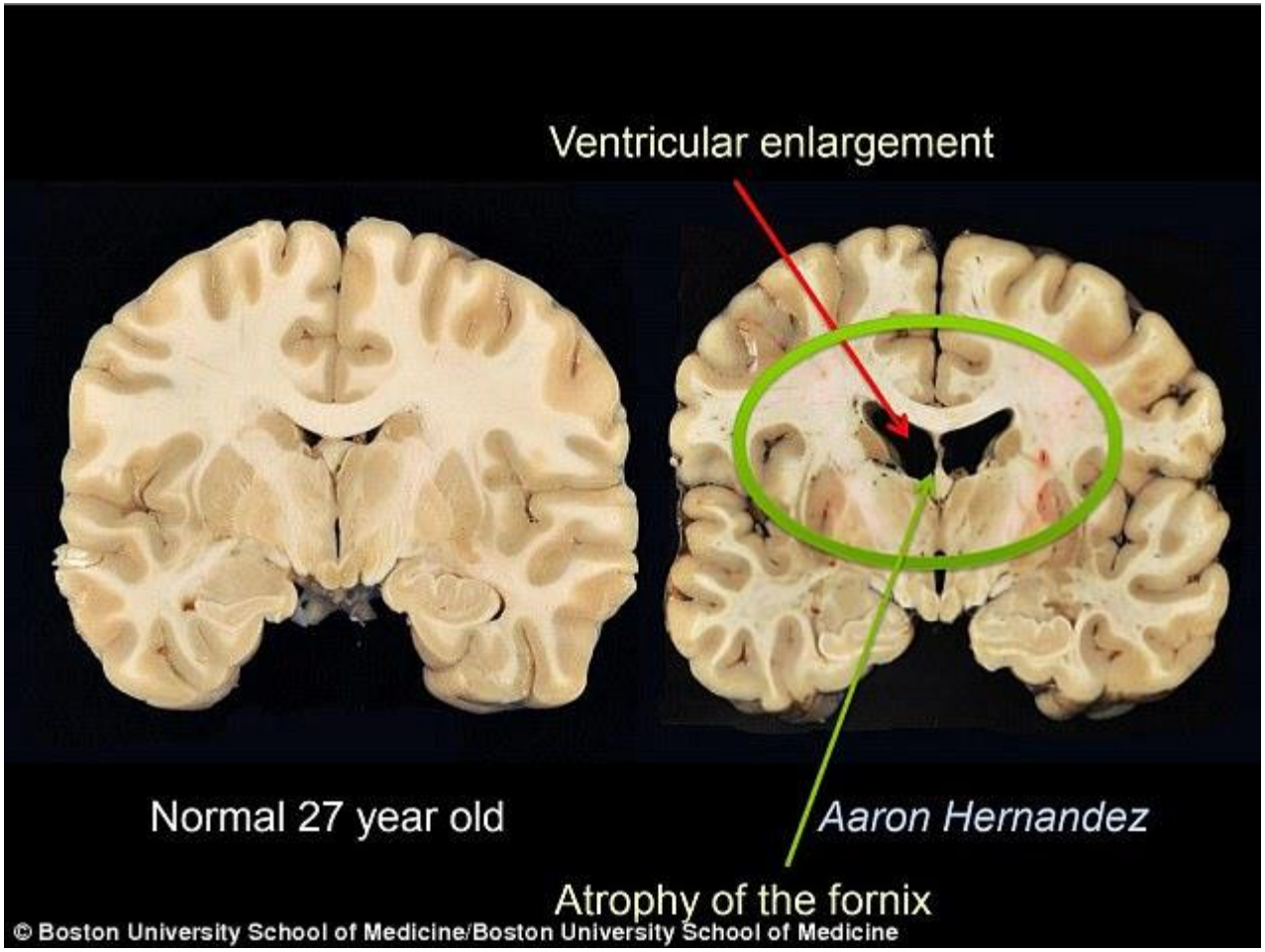
Chronic traumatic encephalopathy

- Presents with headaches, mood disturbances, memory loss.
- Results from repeated (subclinical) hits to the head.
- Affected brains are typically atrophic, with enlarged ventricles, and show accumulation of tau-containing neurofibrillary tangles in a characteristic pattern involving superficial frontal and temporal lobe cortex.

Chronic traumatic encephalopathy

- Axons rupture, releasing Tau protein (microtubule associated protein).
- Tau proteins may hyperphosphorylate, clump, and migrate independently.
- Blocks capsase and proteosome clearing of misfolded proteins.
- It is not clear whether a malformed tau protein may induce normal tau protein to misfold.

Chronic traumatic encephalopathy



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Chronic traumatic encephalopathy

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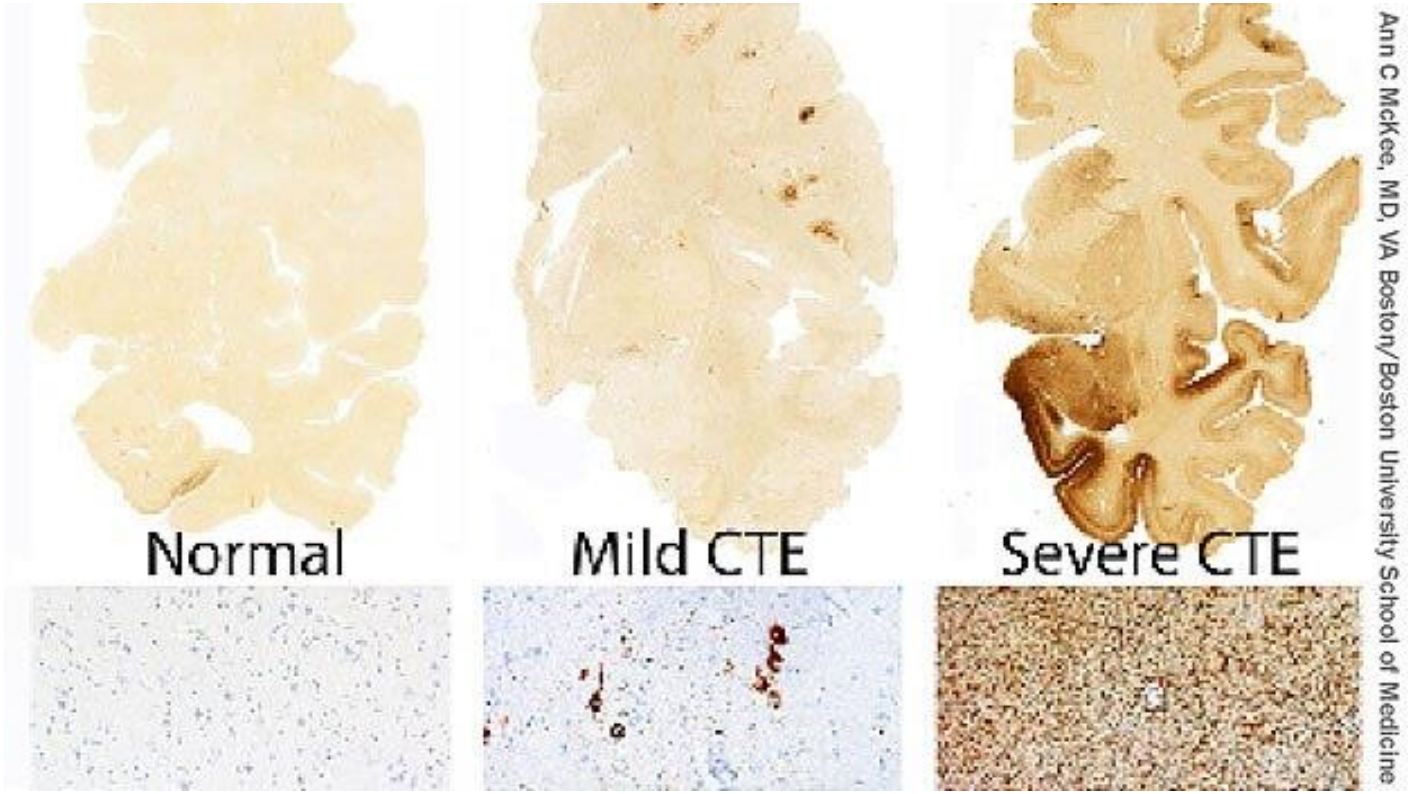


Consciousness

- Awareness is not consciousness.
- The answer may be on the “tip of my tongue”. I am aware that I do not have the necessary information. I am conscious; but that is not consciousness. That is dissociated consciousness.
- Consciousness is the ability to reflect upon being conscious. Memory is required.
- Consciousness is a cross-talk mechanism for a subset of brain functions.

Arousal and awareness

- Patients in coma cannot be aroused
- Patients who are asleep can be aroused
- Arousal in a patient with a pontine lesion is demonstrated through directed eye movements
- Patients who are awake but unresponsive to stimuli are unaware
- Patients in a vegetative state can be aroused but are unaware
- Inattention to verbal stimuli may be due to hearing loss

Coma

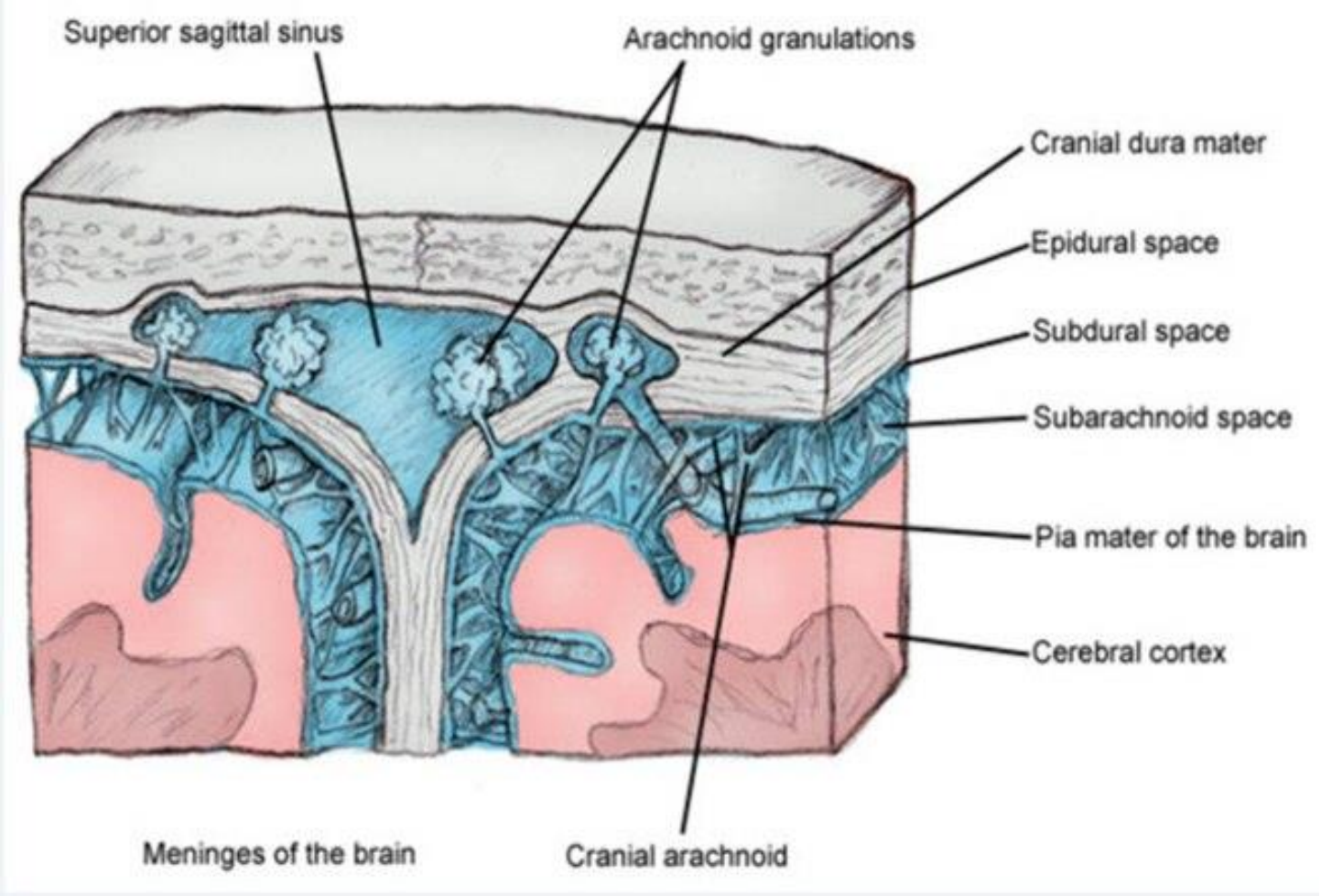
- Drug overdose, trauma, cardiac arrest are three most common causes of coma.
- 2-33% of those resuscitated outside of the hospital survive to reach the hospital.
- 80% of those are comatose.
- 10-30% have meaningful recovery.
- Cerebral O_2 stores lost within 20 seconds of the onset of cardiac arrest; glucose and ATP stores are lost by 5 minutes.

Coma outcomes

- There are no clinical findings that strongly predict a good clinical outcome.
- The findings at 24 and, then, 72 hours are more important than those immediately post-resuscitation.
- At 24 hours, a poor clinical outcome is predicted by the absence of a pupillary response (LR+, 10; LR-, 0.8) and the absence of a corneal reflex, (13; 0.6).
- At 72 hours, a poor clinical outcome is predicted by the absence of motor response (LR+, 9.2; LR-, 0.7).
- Seizures at 72 hours have minimal prognostic value.

Table 28-1 Patterns of Vascular Injury in the Central Nervous System

Location	Etiology	Additional Features
Epidural space	Trauma	Usually associated with a skull fracture (in adults); rapidly evolving neurologic symptoms, requiring intervention
Subdural space	Trauma	Level of trauma may be mild; slowly evolving neurologic symptoms, often with a delay from the time of injury
Subarachnoid space	Vascular abnormalities (Arteriovenous malformation or aneurysm)	Sudden onset of severe headache, often with rapid neurologic deterioration; secondary injury may emerge, associated with vasospasm
	Trauma	Typically associated with underlying contusions
Intraparenchymal	Trauma (contusions)	Selective involvement of the crests of gyri, where the brain may contact the inner surface of the skull (frontal and temporal tips, orbitofrontal surface)
	Hemorrhagic conversion of an ischemic infarction	Usually petechial hemorrhages in an area of previously ischemic brain, usually following the cortical ribbon
	Cerebral amyloid angiopathy	"Lobar" hemorrhage, involving cerebral cortex, often with extension into the subarachnoid space
	Hypertension	Centered in the deep white matter, thalamus, basal ganglia, or brainstem; may extend into the ventricular system
	Tumors (primary or metastatic)	Associated with high grade gliomas or certain metastases (melanoma, choriocarcinoma, renal cell carcinoma)



https://reference.medscape.com/slideshow/traumatic-brain-injuries-6009819#4
Accessed 03/20/2020

Epidural hematoma

- Normally the dura is fused with the periosteum on the internal surface of the skull.
- Dural arteries are vulnerable to injury in which the fracture lines cross the course of the vessel.
- In children, in whom the skull is deformable, a temporary displacement of the skull bones leading to laceration of a vessel can occur in the absence of a skull fracture.
- Posterior fossa lesions more common in children.

Epidural hematoma

- Results from a brief linear contact force to the calvaria that causes separation of the periosteal dura from bone and disruption of interposed vessels due to shearing stress.
- Skull fractures occur in 85–95% of adult cases
- Extension of the hematoma usually is limited by suture lines owing to the tight attachment of the dura at these locations.
- Intracranial epidural hematoma is rare in individuals younger than 2 years and older than 60 years.

Epidural hematoma

- 66% involve the temporal-parietal region
- Middle meningeal artery
- 5% in the posterior fossa (particularly in child)
- At the vertex,
- Superior sagittal sinus
- In frontal injuries,
- Ethmoidal artery
- In Occipital injuries
- Transverse or sigmoid sinus
- 2-10% bilateral

Epidural hematoma

- The extravasation of blood under arterial pressure can cause the dura to separate from the inner surface of the skull.
- The expanding hematoma has a smooth inner contour that compresses the brain surface.
- Symptoms include:
- Headache
- Nausea/vomiting
- Seizures
- Focal neurologic deficits

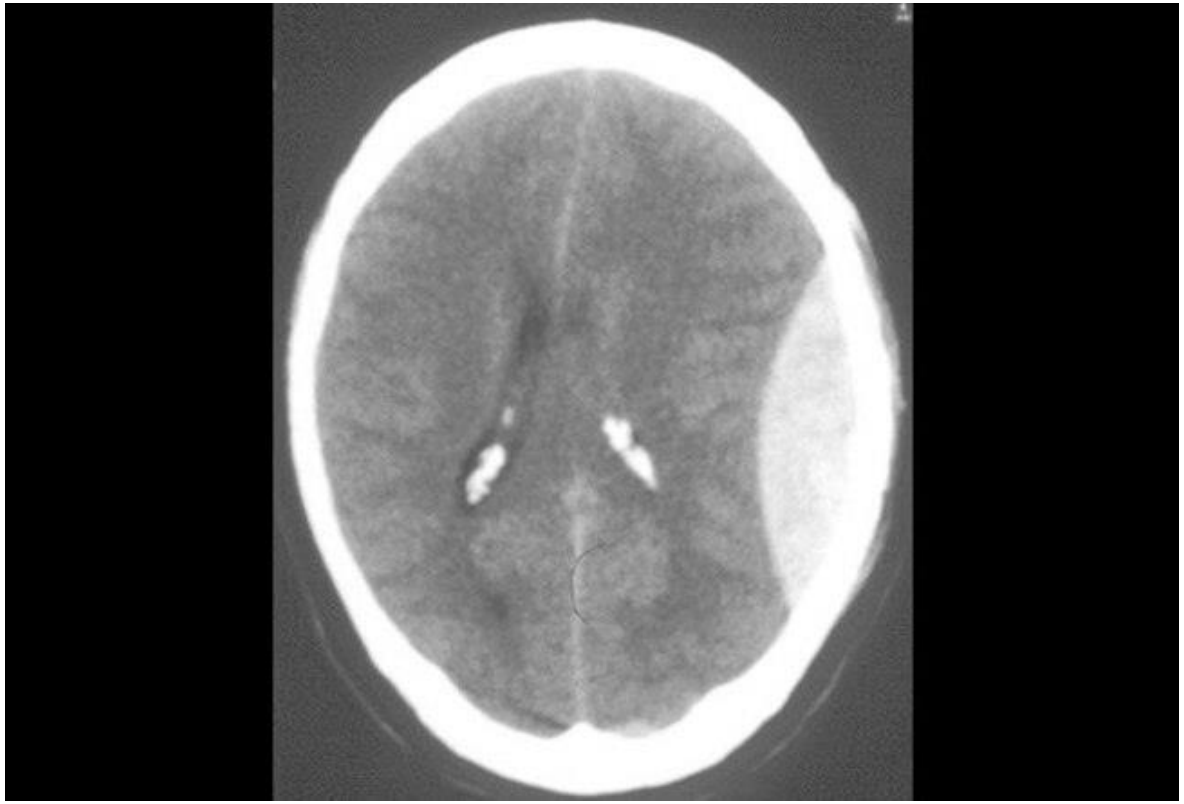
Epidural hematoma

- Alterations in the level of consciousness following impact may have a variable presentation.
- 10-33% of cases demonstrate the classic association of a lucid interval between the initial loss of consciousness at the time of impact and a delayed decline in mental status
- Posterior fossa epidural hematoma may exhibit a rapid and delayed progression from minimal symptoms to even death within minutes.

Epidural hematoma

- The level of consciousness prior to surgery has been correlated with mortality rate:
- 0% for awake patients
- 9% for obtunded patients
- 20% for comatose patients.
- Bilateral intracranial epidural hematoma has a mortality rate of 15–20%.
- Posterior fossa epidural hematoma has a mortality rate of 26%.

Epidural hematoma



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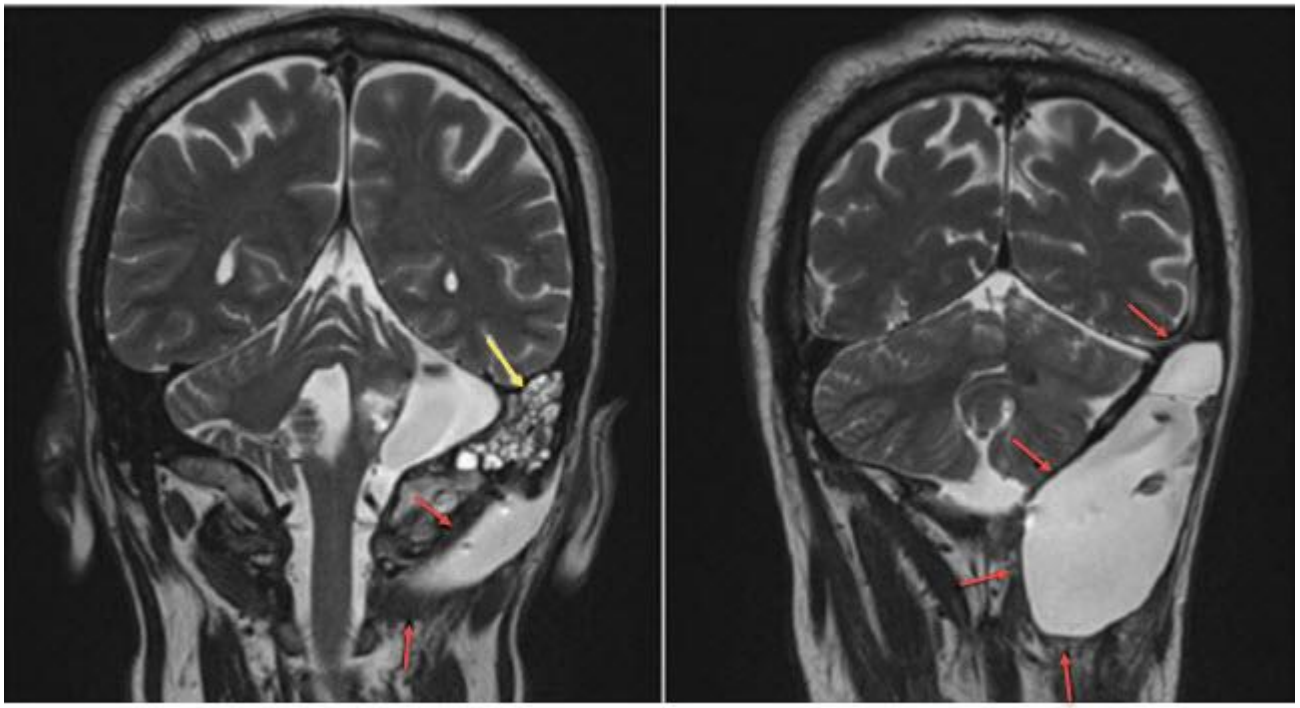
Posterior fossa



Basilar fracture with CSF leak (red arrows) and leakage in mastoid cells (yellow arrow)

Iannella G, Manno A, Pasqualitto E, et al. Case Rep Otolaryngol. 2016;2016:7521798. [Open access.] PMID: 27597915, PMCID: PMC4997066. Accessed 03/20/2020

Subdural hematoma

- The dura is composed an external collagenous and an inner border cell layer with scant fibroblasts, and abundant extracellular space devoid of collagen.
- Bridging veins travel from the convexities of the cerebral hemispheres through the subarachnoid space and the subdural space to empty into a dural sinus.

Subdural hematoma

- The brain is suspended in cerebrospinal fluid (CSF), but the venous sinuses are fixed relative to the dura.
- The displacement of the brain that occurs in trauma can tear the veins at the point where they penetrate the dura.
- Freshly clotted blood is present along the brain surface, without extension into the depths of sulci.
- The underlying brain is flattened and the subarachnoid space is often clear.

Subdural hematoma

- Most common type of intracranial mass lesion
- Occurs in 33% of severe head injuries
- 70% of patients have neurologic deterioration in the first 24 hours
- 10% bilateral
- Severe headache
- Drowsiness
- Confusion
- Contralateral weakness and positive Babinski reflex
- Delayed bleed noted in middle aged patients on anticoagulants or antiplatelet medications

Subdural hematoma

- A midline shift exceeding by 3mm the size of the hematoma on initial CT is associated with poor outcome
- 50% dead within 2 days
- If survive untreated:
- Subacute phase begins to organize at 3-7 days
- Lysis of clot
- Chronic phase begins at 2-3 weeks

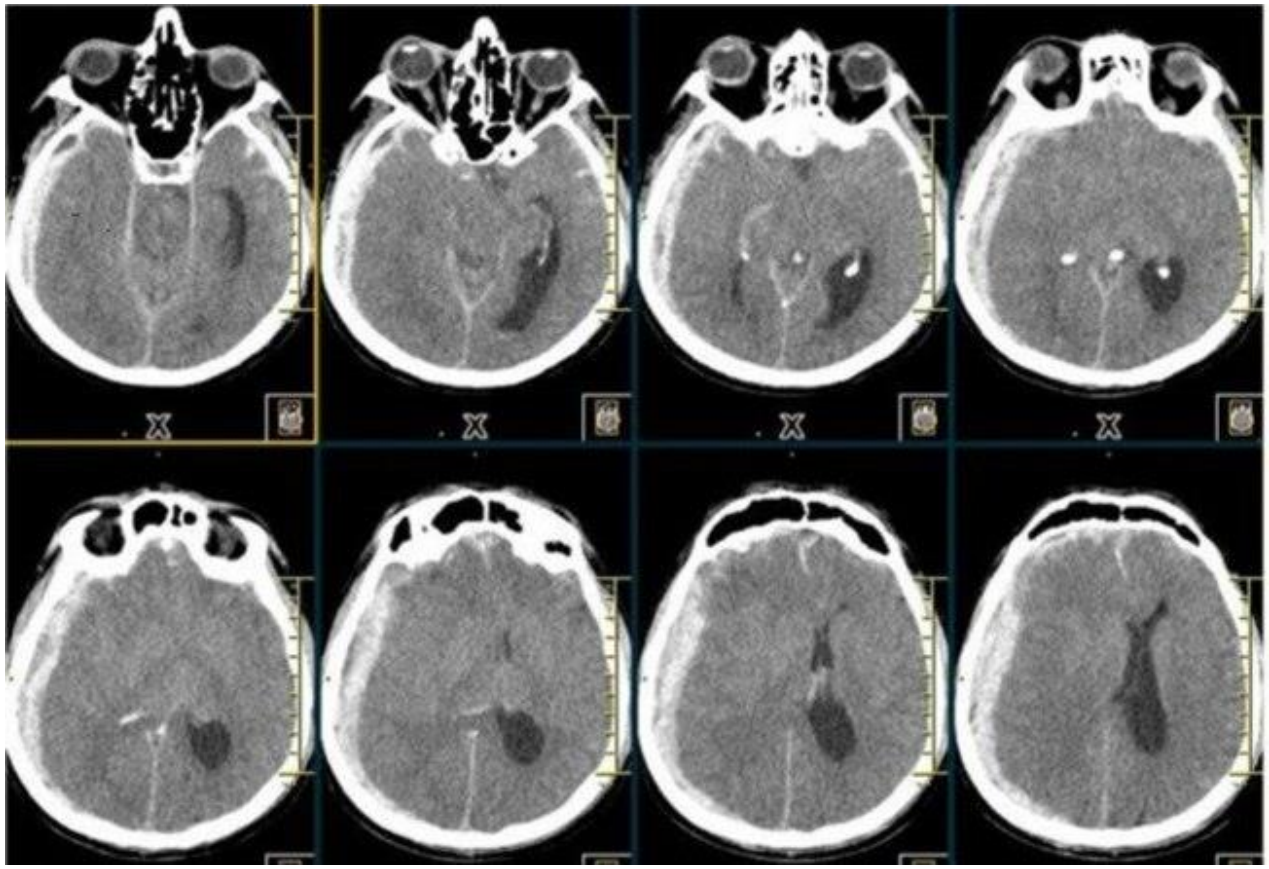
Growth of fibroblasts from the dural surface into the hematoma

Hyalinized connective tissue at 1-3 months

Subdural hematoma

- The organized hematoma is firmly attached by ingrowing fibrous tissue to the inner surface of the dura and is free of the underlying arachnoid, which does not contribute to healing.
- The lesion can eventually retract as the granulation tissue matures until only a thin layer of reactive connective tissue remains (subdural membrane)
- May re-bleed

Acute subdural hematoma



https://reference.medscape.com/slideshow/traumatic-brain-injuries-6009819#6 Accessed 03/20/2020

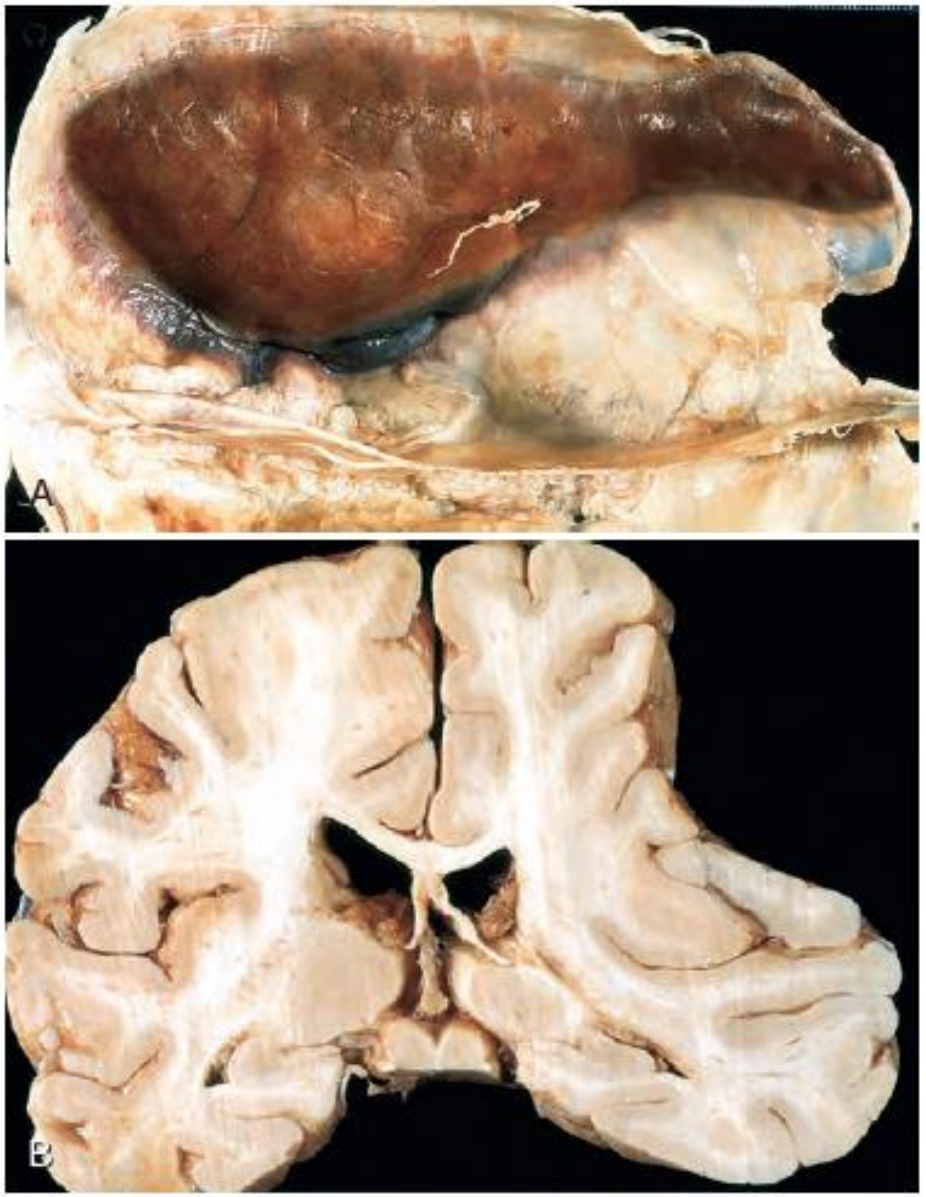


Figure 28-12 **A,** Large organizing subdural hematoma attached to the dura. **B,** Coronal section of the brain showing compression of the hemisphere underlying the subdural hematoma shown in **A.**

Extra-cerebral bleeds

Site	Vessels	Symptoms
Subdural Hematoma	Tear of a superior cerebral vein as it enters the Saggital Sinus	Severe headache within 48 hours, followed by drowsiness, confusion, contralateral weakness, positive Babinski reflex Usually lyse in 1 week and hematoma is organized. Over 3 months. If rebleed from site, is chronic subdural
Epidural Hemorrhage	Tear of Middle Meningeal Artery (usual)	Rapid onset; drowsiness, confusion; symptoms depend upon site of pressure

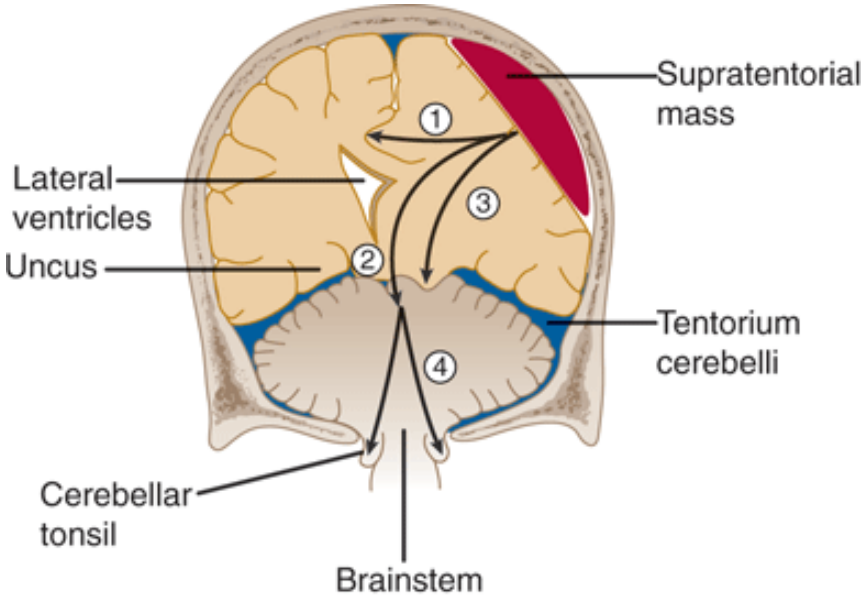
Herniation

- Displacement of brain tissue past rigid dural folds (the falx and tentorium) or through openings in the skull because of increased intracranial pressure.
- As the volume of the brain increases, CSF is displaced and the vasculature is compressed, leading to increasing pressure within the cranial cavity.
- When the increase is beyond the limit permitted by compression of veins and displacement of CSF, tissue herniates between compartments across the pressure gradient.

Herniation

- Associated with mass effect, either diffuse (generalized brain edema) or focal (tumors, abscesses, or hemorrhages).
- Elevated intracranial pressure may also reduce perfusion of the brain, further exacerbating cerebral edema.
- If the expansion is sufficiently severe, herniation may occur in multiple anatomic locations

Herniation



(Reproduced with permission from Aminoll ML, Greenberg DA, Simon RP. Clinical News 1094, 6th edn, McGraw-Hill, 2005.)
Fig. 11-8 Accessed 07/01/2010

An expanding supra-tentorial mass lesion may cause brain tissue to be displaced into an adjacent intracranial compartment, resulting in

(1) cingulate herniation under the falx,
(2) downward transtentorial (central) herniation,
(3) uncal herniation over the edge of the tentorium, or
(4) cerebellar tonsillar herniation into the foramen magnum.

Subfalcine (cingulate) herniation

- This may be associated with compression of branches of the anterior cerebral artery.
- May see a higher cerebral dysfunction such as dysphasia, dyscalculia;
- Visuospatial disorder; with homonymous visual field defect; and
- Ipsilateral motor or sensory deficits

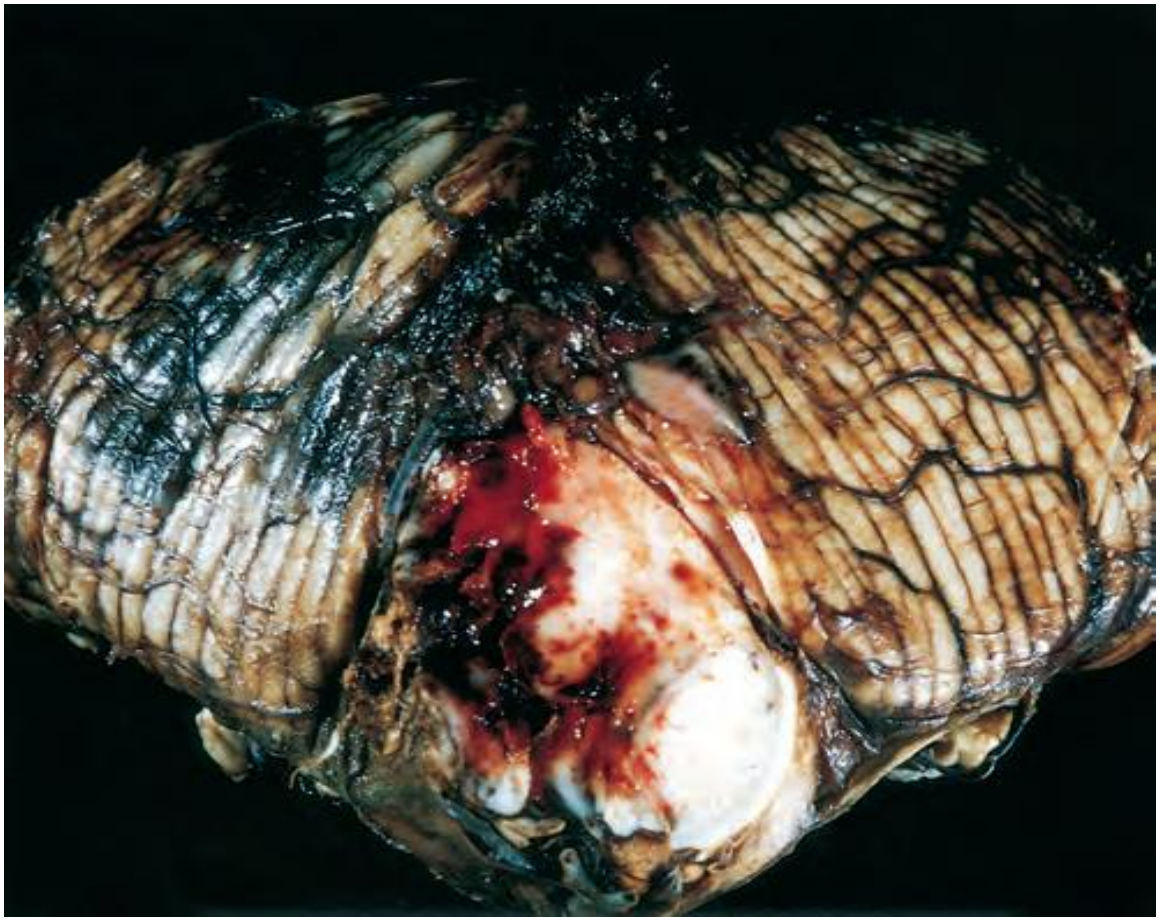
Transtentorial (uncinate, mesial temporal) herniation

- The third cranial nerve is compromised (ipsilateral fixed, dilated pupil, can only abduct eye on affected side)
- The posterior cerebral artery may also be compressed (visual cortex)

Transtentorial (uncinate, mesial temporal) herniation

- Contralateral cerebral peduncle may be compressed as herniation progresses:
- Hemiparesis ipsilateral to the side of the herniation
- Linear or flame-shaped venous hemorrhagic lesions in the midbrain and pons (Duret hemorrhages)

Duret hemorrage



Frosch, MP, Anthony, DC, De Girolami, U, “The Central Nervous System,” in Kumar, V, Abbas, AK, Aster, JC, (eds), Robbins and Cotran Pathologic Basis of Disease (9th ed.), Elsevier. Philadelphia. (2015) Fig. 28-4 Accessed 10/25/2019

Tonsillar herniation

- Displacement of the cerebellar tonsils through the foramen magnum.
- Causes brainstem compression and compromises vital respiratory and cardiac centers in the medulla oblongata.