NEUROTRAUMATOLOGY

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INCIDENCE

- TRAUMATIC BRAIN INJURIES : Incidence about 150 per 100.000 per year
- SPINAL CORD INJURIES: Incidence about 4 per 100.000 per year
  Frequent cause of major disability
- occur at all ages
  - peak is in young adults between the ages of 15 and 24 years
- Men are affected three or four times as often as women
- The major cause: motor vehicle accidents
  In older adults falls

MAJOR CAUSE OF DEATH AND DISABILITY

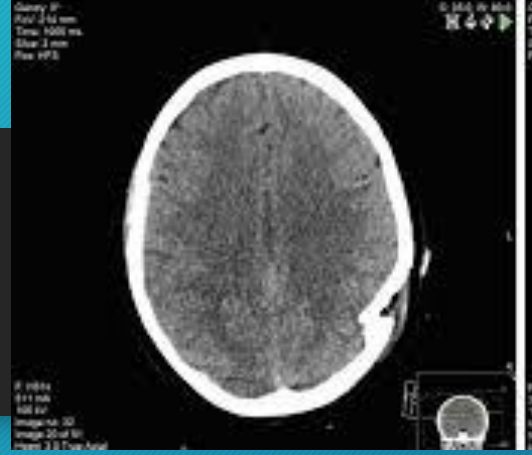
- a major cause of death:
  - especially in young adults younger than the age of 24 years)
  - trend toward an increase of mortality rates from TBI among populations aged older than 65 years.
  - deaths have increased over the past decade

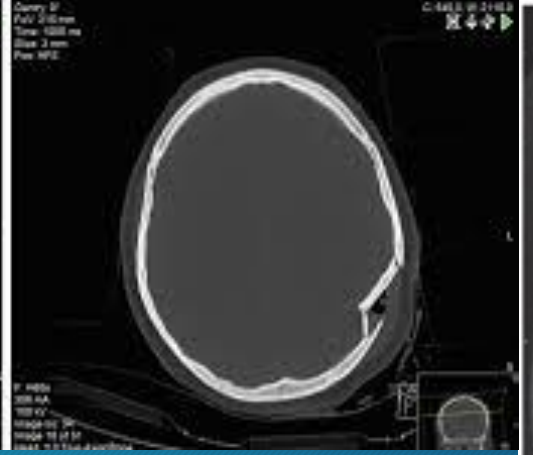
- a major cause of disability (mainly spinal cord injuries).

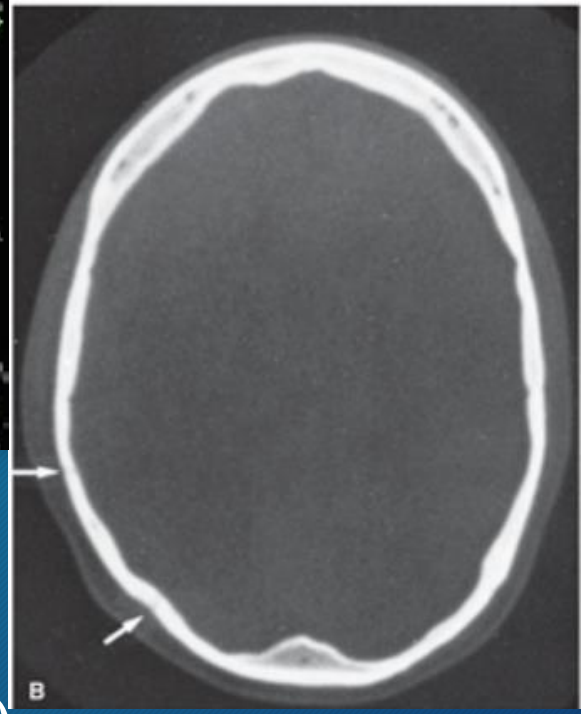
TYPES OF TBI

- skull fractures
- cerebral concussion and axonal-shearing injury (diffuse axonal injury)
- parenchymal contusion and intracerebral hematoma
- subdural hematoma (acute or chronic)
- epidural hematoma
- traumatic subarachnoid hemorrhage

SKULL FRACTURES







- linear, depressed, or comminuted types
- CLOSED (= SIMPLE, the broken bone doesn´t break the skin)
- OPEND (= COMPOUND, the scalp over the fracture is lacerated)
- = IMPORTANT MARKERS OF A POSSIBLY SERIOUS BRAIN INJURY
  - rarely cause problems by themselves (the prognosis depends more on the nature and severity of injury to the brain than on the severity of injury to the skull).
- LINEAR FRACTURES (80%) – incidate the need to perform CT (mostly otherwise normal),
- most common in the temporoparietal region, where the skull is thinnest
- Nondisplaced linear fractures are managed conservatively

DEPRESSED OR COMMINUTED FRACTURES

- IN DEPRESSED F. one or more fragments of bone are displaced inward, compressing the underlying brain
  - In 85% of cases, they are open and liable to:
    become infected
    leak cerebrospinal fluid (CSF)
    underlying brain injury
    tearing, compression, or thrombosis of underlying venous dural sinuses.
- In COMMINUTED F., there are multiple, shattered bone fragments, which may or may not be displaced
- Even when closed, most of them require surgical exploration for debridement, elevation of bone fragments, and repair of dural lacerations.

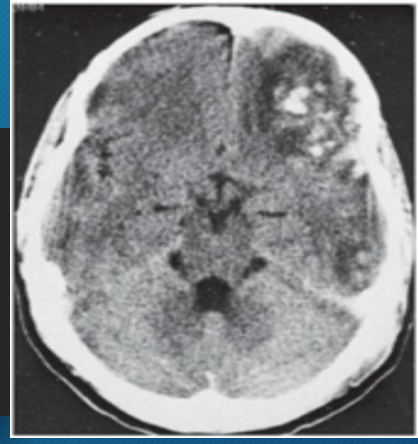
CEREBRAL CONCUSSION AND AXONAL-SHEARING INJURY

- Frequent symptom of TBI = LOSS OF CONSCIOUSNESS at the moment of impact
- It is caused by acceleration-deceleration movements of the head, which result in the stretching and shearing of axons
- The mechanism: transient functional disruption of the reticular activating system caused by rotational forces on the upper brain stem
- the term CONCUSSION is used when the alteration of consciousness is brief (e.g., <6 hours),
  - patients may be COMPLETELY UNCONSCIOUS or remain awake but appear DAZED;
  - most RECOVER SPONTANEOUSLY WITHIN SECONDS OR MINUTES, rather than hours,
  - some have RETROGRADE OR ANTEROGRADE AMNESIA surrounding the event.
  - Only 5% of patients who sustained a concussion have an intracranial hemorrhage on CT

DIFFUSE AXONAL INJURY

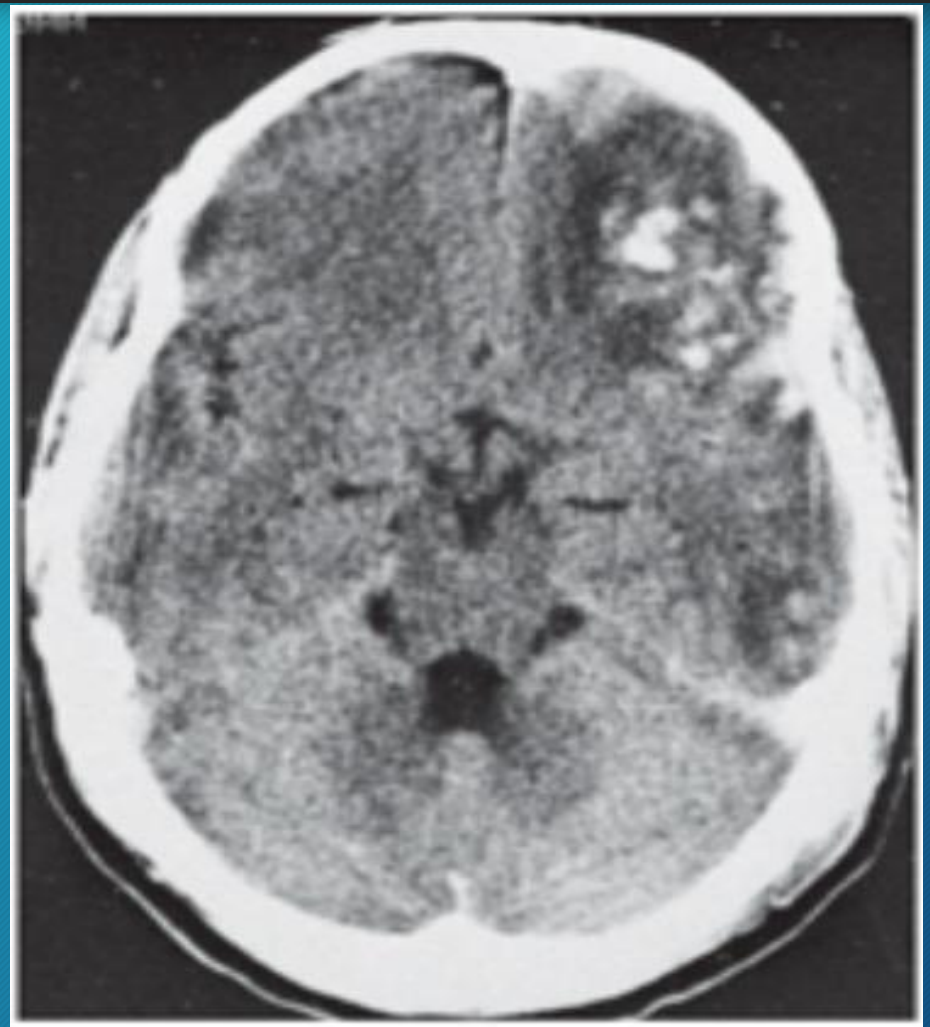
- The term DIFFUSE AXONAL INJURY (DAI) is applied to traumatic coma lasting more than 6 hours
- no other cause of coma is identified by CT or MRI
- common symptom = autonomic dysfunction (e.g., hypertension, hyperhidrosis, hyperpyrexia), which may reflect brain stem or hypothalamic injury.
- those who recover may be left with severe cognitive and motor impairment, including spasticity and ataxia
- the most important cause of persistent disability after traumatic brain damage.

PARENCHYMAL CONTUSION



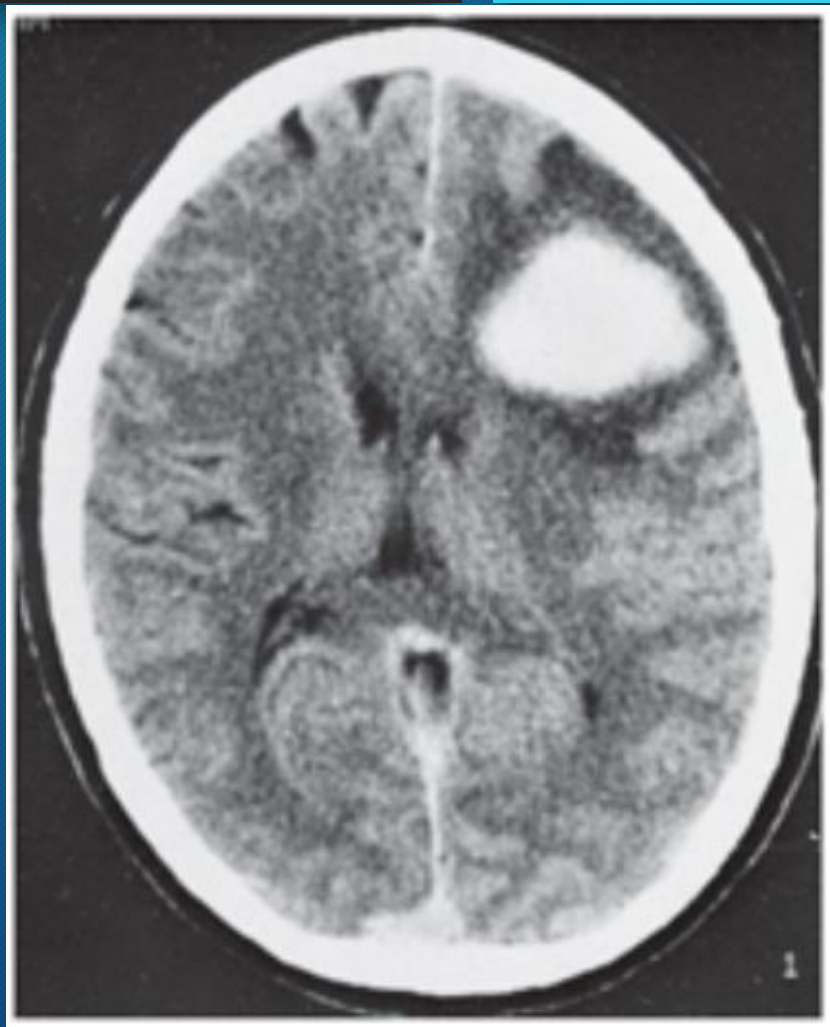
- CEREBRAL CONTUSIONS are focal parenchymal hemorrhages that result from scraping and bruising of the brain as it moves across the inner surface of the skull
  - With lateral forces, contusions may occur at the site of the blow to the head (coup lesions) or at the opposite pole as the brain impacts on the inner table of the skull (contrecoup lesions).
  - The most common sites of contusion are the inferior frontal and temporal lobes, where brain tissue comes in contact with irregular protuberances at the base of the skull
- Less frequently, the contusions may result of cuts from the sharp edges of depressed skull fragments
- Contusions are mostly small and multiple, usually cortical
- Resemble bruised and bloodied brain tissue
- Contusions are often managed conservatively, recovery from one or more small contusions may be excellent.

PARENCHYMAL CONTUSIONS

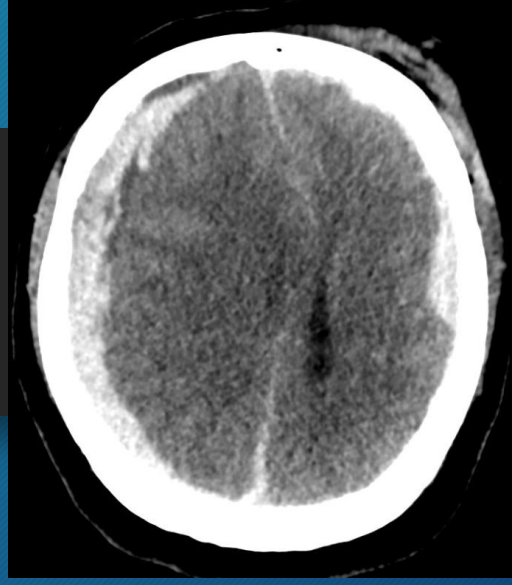


INTRACEREBRAL HEMATOMA

- May occute when rotational forces lead to tearing of small- or medium-sized vessels within the parenchyma
- Hematoma is the focal collection of blood clots that displaces the brain
- located mostly in the deep white matter
- The large, parenchymal hematomas with mass effect may require surgical evacuation.

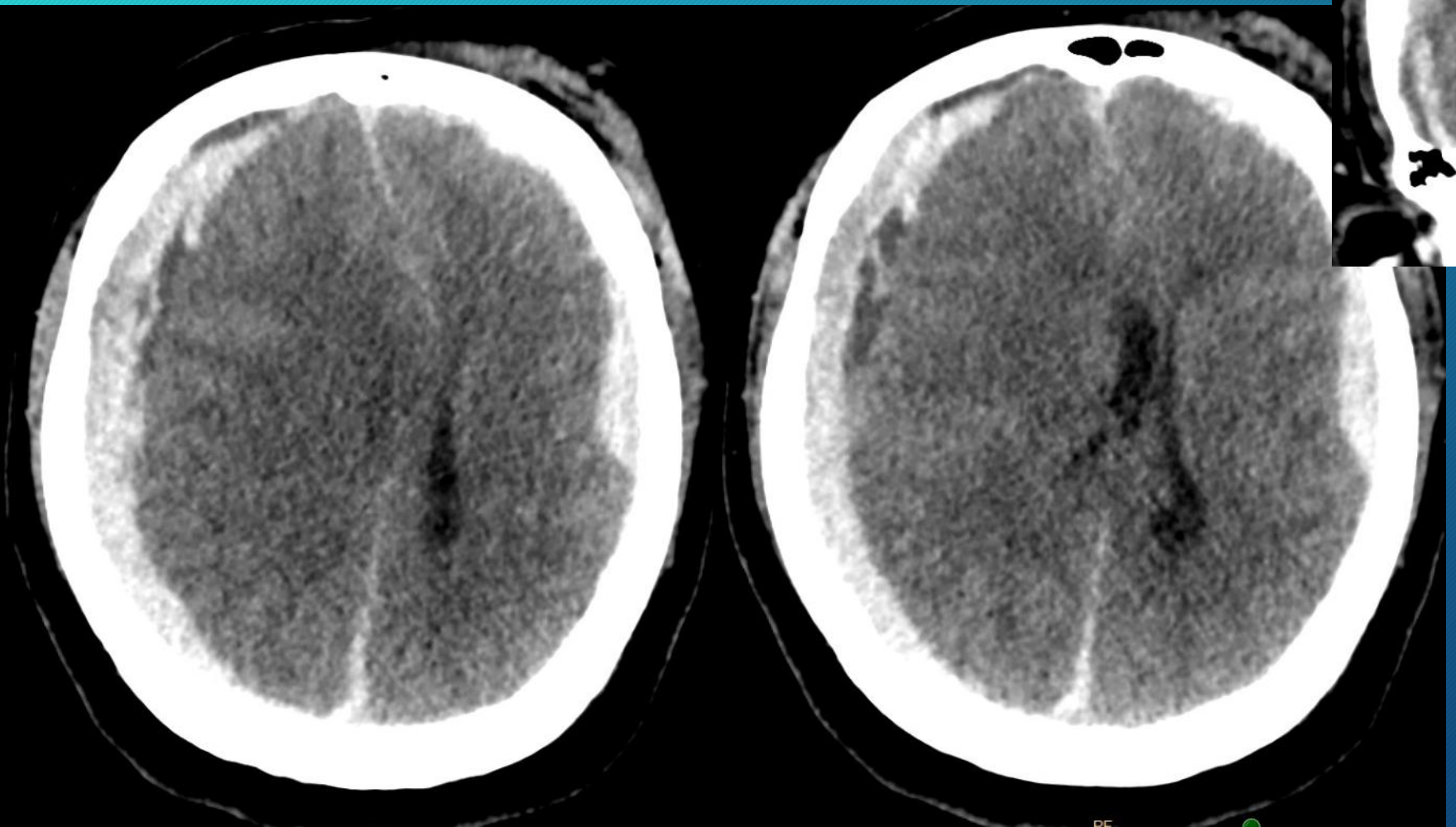


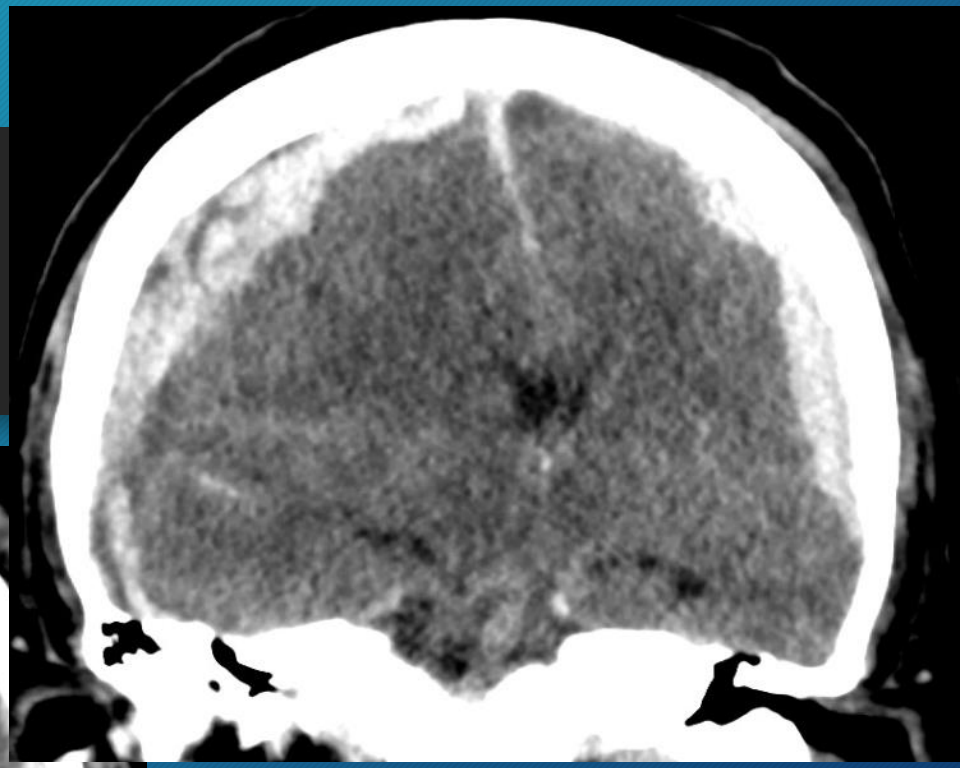
SUBDURAL HEMATOMA



- usually arise from a venous source, with blood filling the potential space between the dural and arachnoid membranes
- the bleeding is usually caused by movements of the brain within the skull that leads to stretching and tearing of “bridging” veins that drain from the surface of the brain to the dural sinuses.
- Mostly located over the lateral cerebral convexities, less frequenty in posterior fossa, between hemispheres, along tentorium usw.
- CT = high-density, crescentic collection along the hemispheric convexity
- Most important risk factors:
- elderly or alcoholic patients with cerebral atrophy (SDH may result from trivial impact or even only whiplash),
- coagulopathy (incl. oral anticoagulants) – associated with increased risk of death

SUBDURAL HEMATOMA





ACUTE SUBDURAL HEMATOMA

- ACUTE SUBDURAL HEMATOMAS - symptomatic within 72 hours of injury,
- most patients have neurologic symptoms from the moment of impact
- Most frequent after falls or assaults, relatively less common after car accidents
- Half of all patients with an acute SDH lose consciousness at the time of injury
- half of those who awaken lose consciousness for a second time after a “lucid interval” of minutes to hours as the subdural hematoma grows in size
- the most common focal neurologic signs (each occuring in 1/2 to 2/3 of patients):
- Hemiparesis (usually contralateral) and pupillary abnormalities (mostly ipslateral)
- Sometimes the other way round (because uncal herniation may lead to compression of the contralateral cerebral peduncle or third cranial nerve against the tentorial edge)

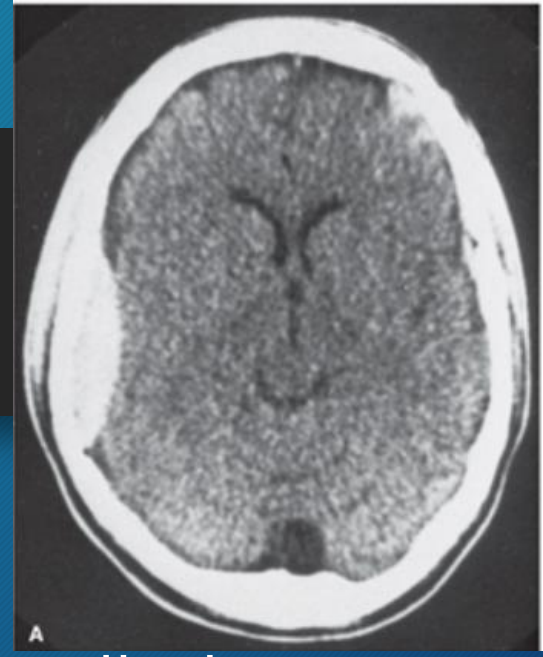
CHRONIC SUBDURAL HEMATOMA

- Become symptomatic after 21 days
- In 25% to 50% of cases, there is no recognized episode of head injury. In many cases, chronic SDH results from trivial trauma
- Risk factors same as in acute SDH (cerebral atrophy, alcoholism, bleeding disorders or use of anticoagulant medication and age - after age 50 years).
- The blood clott usually liquefies into a HYGROMA
- In most the cases, the symptoms are minimal (because the brain accommodates the gradual buildup of mass effect) and may be restricted to altered mental status
- CT typically shows an isodense or hypodense, crescent-shaped mass that deforms the surface of the brain

TREATMENT OF ACUTE X CHRONIC SDH

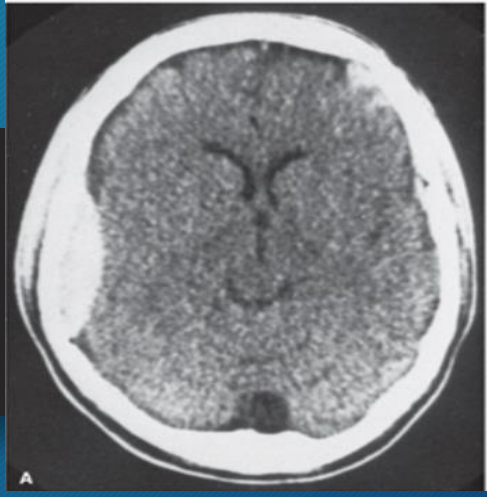
- Symptomatic acute and chronic SDHs with significant mass effect should be evacuated.
- In acute SDH, the blood is clotted and the surgical evacuation usually requires a large-window craniotomy.
  - Outcome after surgical evacuation depends primarily on the severity of the initial deficit and the interval from injury to surgery.
- Liquefied chronic SDHs are usually evacuated with drainage of the collections via a series of burr holes.
- Smaller, minimally symptomatic, subdural hematomas are managed conservatively.

EPIDURAL HEMATOMA



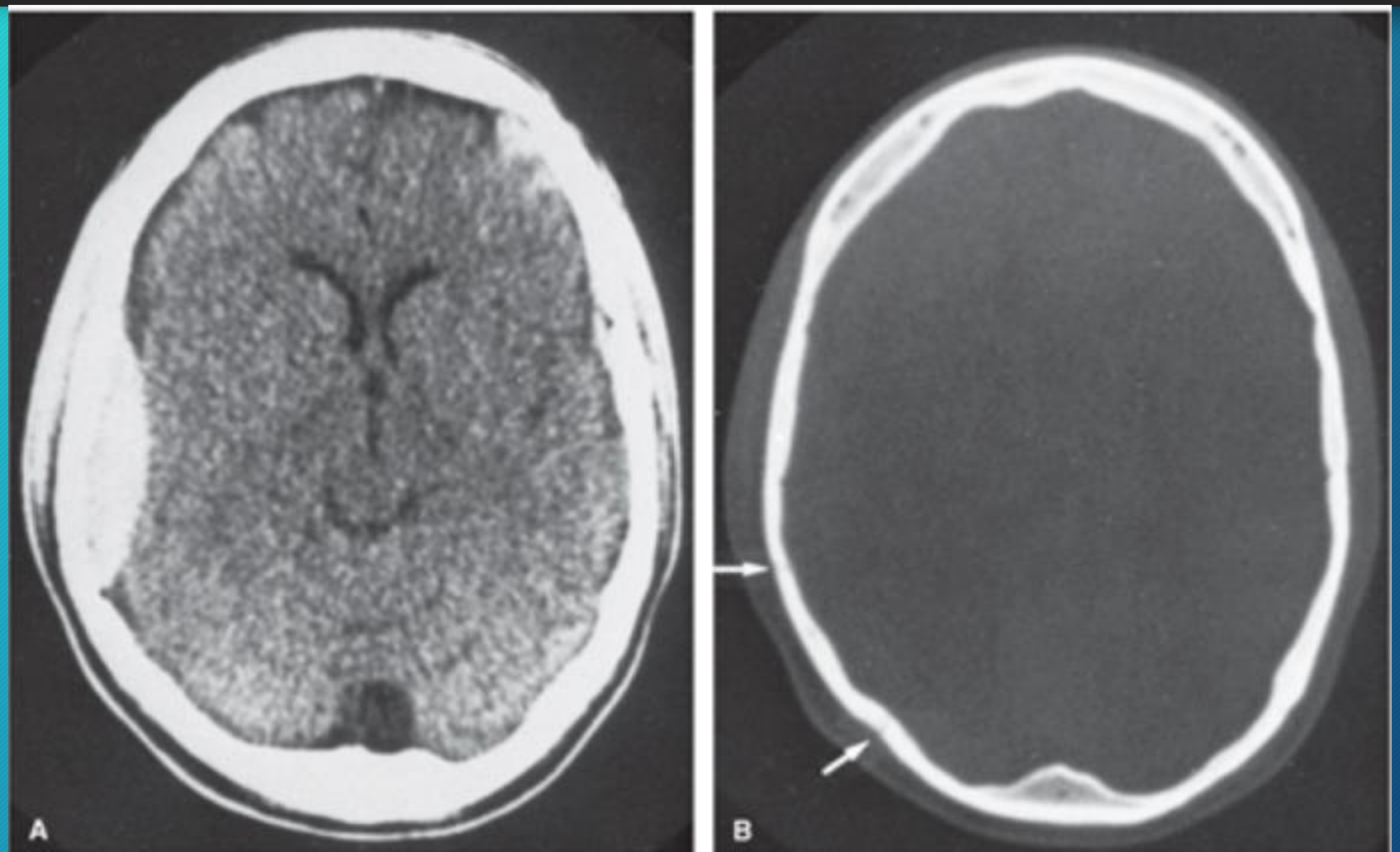
- Rare complication of TBI - occurs in less than 1% of all TBI cases
- Bleeding into the epidural space
- Generally caused by a tear in the wall of one of the meningeal arteries, usually the middle meningeal artery,
  - in 15% of patients, the bleeding arises from a dural sinus.
- Seventy-five percent of patients are associated with a skull fracture.
- Most epidural hematomas are located over the convexity of the hemisphere in the middle cranial fossa (occasionally in the anterior or posterior fossa).
- In most cases, the hematoma is ipsilateral to the site of impact.
- primarily a problem of young adults; rarely seen in the elderly because the dura becomes increasingly adherent to the skull with advanced age.

EPIDURAL HEMATOMA



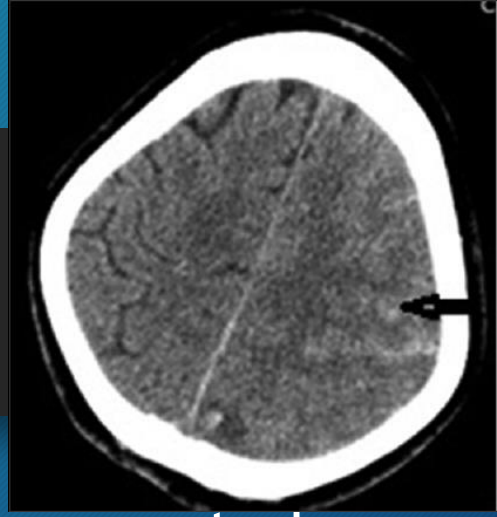
- in one-third of patients, proceeds from an immediate loss of consciousness caused by concussion to a lucid interval and then to a relapse into coma, with hemiplegia as the epidural hematoma expands.
- The ipsilateral pupil loses reactivity to light because the third cranial nerve is stretched as the midbrain is displaced contralaterally. Later, it becomes fixed and dilated as the third nerve is compressed by the hippocampal gyrus as it herniates over the free edge of the tentorium.
- On CT, epidural blood takes on a bulging convex pattern, because the collection is limited by firm attachments from the dura to the cranial sutures
- Progression (to herniation and death) may occur rapidly because the bleeding is arterial.
- The mortality rate approaches 100% in untreated patients and ranges from 5% to 30% in treated patients (the faster the treatment is, the better is a chance to recover)
- If there is little coexisting brain damage, functional recovery may be excellent.

EPIDURAL HEMATOMA



- Epidural hematoma on CT
- CT with none window shows two adjacent fractures (arrows)
- The anterior one is at the site of the groove for the middle meningeal artery

TRAUMATIC SUBARACHNOID HEMORRHAGE



- Some extravasation of blood into the subarachnoid spaces is to be expected in any patient with head injury (mostly detecable only by CSF examination).
- with more serious injuries (when larger vessels traversing the subarachnoid space are torn) focal or diffuse subarachnoid hemorrhage may be detected *by CT* (in most the cases over the convexities – note the contrast to spontaneous SAH, which is mostly distributed in basal cistern)
- Usually little clinical importance
- Large amount of blood in subarachoid space is a negative prognostic factor
- delayed complications of aneurysmal SAH, such as hydrocephalus and ischemia from vasospasm, are unusual after traumatic SAH

TRAUMATIC SPINAL CORD INJURY

- A sudden event with possibly catastrophic effects
- May create a medical, financial, and social burden for the individual and society
- primarily affects young adults aged between 16 and 30 years, 79% are male
- The most frequent cause: motor vehicle accidents (42%), followed by falls, sport or work-related injuries and violence.
- Most injuries occur on the weekends and during the summer months.
- From the neurological point of view, they may lead to:
  - Tetraplegia: incomplete (34%) or complete (18%)
  - Paraplegia: incomplete (19%) or complete (23%)
  - The level of affection identified by the sensory level. Frequent signs of dysautonomia.
- Less than 1% of persons experienced complete neurologic recovery by hospital discharge.

MECHANISM OF ACUTE SCI

- The two step process = involves primary and secondary mechanisms
- The primary mechanism results from the initial mechanical injury due to local deformation and energy transformation
- Most commonly the combination of the initial impact as well as subsequent persisting compression.
- Main primary mechanism: the impact of bone and ligament against the spinal cord from high translational forces, such as that generated by flexion, extension, axial rotation, or vertebral compression
- The spinal cord may consequently be compressed, stretched, or crushed by fracture or dislocations
- Injury can result from only the initial impact without ongoing compression (as a result of severe ligamentous injuries in which the spinal column dislocates and then spontaneously reduces or when there is preexisting cervical spondylosis or spinal stenosis)

SCI – SECONDARY MECHANISMS

- Secondary mechanisms result from biochemical cascades that occur after the initial event
- are a source of ongoing SCI and neurologic deterioration.
- cause the damage to neural tissue on the cellular level
- include the pathologic effects of microvascular changes, excitatory amino acids, cell membrane destabilization, free radicals, inflammatory mediators, and neuroglia apoptosis

SCI PATHOLOGY

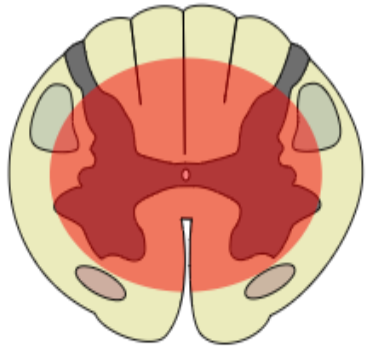
- *solid cord injury* – the spinal cord grossly appears normal without evidence of softening, discoloration, or cavity formation. However, damage to the cord can be clearly seen on histologic examination
- *contusion/cavity* - no breach or disruption in the surface anatomy, but the areas of hemorrhage and necrosis (eventually evolving into cysts) are readily identified in the cord parenchyma
- *laceration -* result in clear-cut disruption of the surface anatomy. Most often caused by penetrating missiles or sharp fragments of bone.
- *massive compression* - often accompanied by severe vertebral body fractures or dislocations

BASIC TYPES OF INCOMPLETE SCIs (spinal cord syndromes based on horizontal and partly vertical localization)

- ***central cord syndrome*** (mostly in cervical region loss of motion and sensation mainly in arms and hands, the most common)
- ***anterior cord syndrome*** (resulting in loss of function of the anterior two-thirds of the spinal cord. The region affected includes the descending corticospinal tract, ascending spinothalamic tract, and autonomic fibers. It is characterized by a corresponding loss of motor function, loss of pain and temperature sensation, and hypotension)
- ***posterior cord syndrome*** (the least common – less than 1% of SCIs, posterior column dysfunction – vibration, discrimination and proprioception is impaired)
- ***conus medullaris syndrome*** (motor and sensory deficits in lower legs + autonomic dysfunction, combination of upper and lower motor neuron signs)
- ***cauda equina syndrome***
- ***Brown-Séquard Syndrome,*** which is characterized anatomically by a hemicord injury with ipsilateral proprioceptive and motor loss and contralateral pain and temperature sensation loss below the level of the lesion

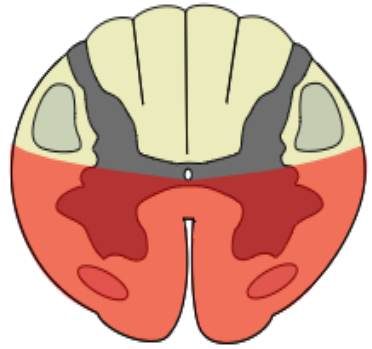
Incomplete cord injuries

Central Cord Syndrome

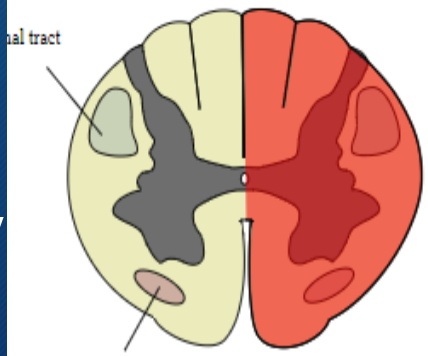


Source: wikipedia

Anterior Cord Syndrome

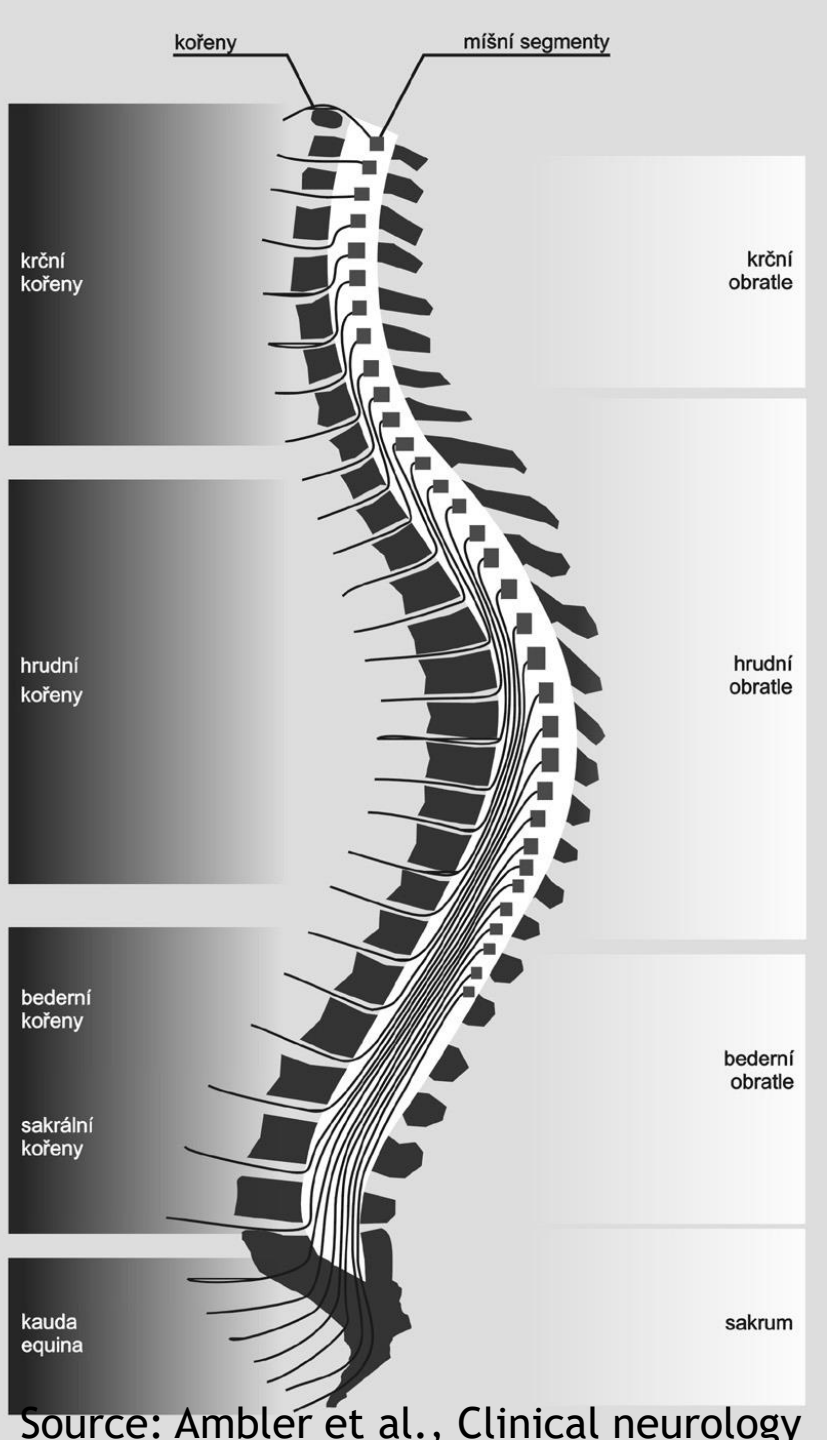


Brown-Séquard Syndrome



CLINICAL PICTURE OF SCI (based on vertical localisation)

- **Cervical - thoracic - lumbar - sacral**, particular segment
- ***Sensory level + autonomic impairment + motor dysfunction:***
- Tetraplegia (cervical segments):
  - spastic in UE if rostral C segments are affected,
  - In lesions at or above C4 ventilation is usually impaired due to diaphragm palsy
  - In lesions at or below C5, the paresis is flaccid in the affected segment (and spastiv below, while no signs of abnormity are apparent above the level of the affected segment)
- Paraplegia (thoracic or lumbar)
- Spastic in lesions affecting thoracic or rostral lumbar segments
- Flaccid at the segment of affection
- + epiconus, conus or cauda equina syndrome



Source: Ambler et al., Clinical neurology

SCI IMAGING

- There are several recommendations available allowing to decide if any imaging examination is necessary
- In general, the imaging is not necessary in case of:
  - no relevant clinical signs and symptoms (incl.neck or back pain)
  - + no risk factors (motor vehicle accidents, falls from 6 ft or more, altered mental status with an unknown mechanism of injury) are present
- In low risk patients (no neurological signs or symptoms, only neck or back pain or limited extend of spinal cord movement), the lateral and AP X-ray may be used.
- However, due to its ease of use and superior sensitivity, most centers go directly to (cervical) spine CT as the initial imaging study of choice.
- MR is the best technique for imaging the spinal cord itself. IN SCIs, it should be performed mainly in case of neurologic deficit with normal x-rays or in the lack of correlation between a neurologic deficit and x-ray findings.

SCI MANAGEMENT

- ***Prehospital*** trauma protocols are critical to prevent further injury to the spinal cord
- Any patient with suspected SCI should be immobilized with a hard cervical collar and/or a rigid head-strap/backboard until definitive neurosurgical assessment can take place.
- Treatment of hypoxia and hypotension, proper monitoring of vital signs, and transfer to an appropriate trauma center will affect ultimate outcomes positively.
- On arrival to ***the trauma center***, a rapid assessment should be undertaken to assess the status of the airway, respiratory, and circulatory (ABC) systems, followed by a cursory assessment of the neurologic status ("disability")
- The acute management of patients with SCI is primarily directed at medical stabilization to prevent secondary injury and to permit the accurate clinical and radiographic diagnosis of spinal cord and column pathology

SCI MANAGEMENT

- neural element decompression and spinal stabilization ***within 24 hours may improve neurologic recovery*** in patients with deficits and spinal cord compression
- there has been no demonstrated increased risk of neurologic deterioration from early surgery
- Management of vital functions and autonomic dysfunction
- High-dose methylprednisolone has long been considered a potential neuroprotective agent in SCI, with the potential of reducing tissue injury by inhibiting lipid peroxidase and free radical production. Several studies performed showed only small (if any) benefit. Due to a trend towards more serious complications associated with steroid use, current guidelines do not recommend administration of methylprednisolone (MP) for the treatment of acute SCI [Level 1].
- Early rehabilitation

THANKS FOR YOUR ATTENTION

- Main source of information:
- Badjatia N, Parikh GY, Mayer SA. Traumatic Brain Injury. In Lewis ED, Mayer SA, Rowland LP. Merritt's Neurology, 13th edition. LWW 2015.
- Mandigo CE, Kaiser MG, Angevine PD. Traumatic Spinal Cord Injury. In Lewis ED, Mayer SA, Rowland LP. Merritt's Neurology, 13th edition. LWW 2015