

Objectives

1. Compare and contrast the clinical signs and symptoms and pathology of subdural and epidural hematomas.
2. Discuss the usual locations of and etiologic basis of diffuse axonal injury.
3. Compare and contrast coup, contrecoup and gliding contusions clinically and pathologically.

Robbins, Basic Pathology, 7th Ed, pp. 818-821.

I. Skull fracture

A. Intact scalp increases skull resistance to fracture 10X

B. Nearly 70% of skull fractures are linear, narrow

C. Basilar skull fractures can be seen from falling on ice and striking the occiput (or off a barstool); frequently seen in homicidal blows to head and in pedestrian/vehicular accidents

II. Epidural hemorrhage - hemorrhage between skull and dura

A. Cause - rupture of meningeal artery, usually branch of middle meningeal or dural venous sinus

B. Clinical

1. Peak age - 2nd and 3rd decade; "lucid interval" in only half of patients; delayed development possible (more often venous)

2. Usually associated with skull fracture, temporal bone; only found in 1 - 4% of patients imaged for craniocerebral trauma (10% of fatalities); biconvex on scans; cross dural attachments but not sutures; 95% unilateral; 95% supratentorial

3. Morbidity related to location (posterior fossa worse) and delay in diagnosis

4. Mortality age-related and ranges from 11-40%; artifactual epidurals can be caused by burning

C. Pathology - volume of blood in fatal cases usually at least 100 ml; causes compression (flattening) of adjacent gyri and displacement of brain; possible herniation and death

III. Subdural hemorrhage - between dura and brain (there is no subdural space normally)

A. Cause - disruption of bridging veins (bridging from surface of brain to dural sinuses)

B. Clinical

1. Peak age - 5th and 6th decade (atrophy, anticoagulants, alcoholics); rapid changes in head velocity

2. 10-20% of craniocerebral trauma (30% of fatalities); unilateral or bilateral (15%); cross sutures but not dural attachments; crescentic; 95% supratentorial (usually fronto-parietal, over convexities); usually more extensive than epidural hemorrhage

3. Morbidity - approximately 5% rebleed; altered mental status, maybe focal neurological deficits

4. Mortality 50-85% in acute

C. Types of presentation

1. **Acute** - usually clear history of trauma; other associated injuries
2. **Chronic** - if presenting as chronic there may not be clear history of trauma (or minor); often associated brain atrophy; slowly altered mental status (may be confused with dementia) because of tendency to enlarge progressively over weeks to months
3. **Non-accidental pediatric** - interhemispheric parafalcial; bilateral; multiple ages of lesions; may be associated with retinal hemorrhages, shearing injuries, contusions and multiple, complex, bilateral, widely-spaced, and/or depressed fractures

D. Pathology

1. Gross - no flattening of underlying gyri; may flatten gyri on other side against skull; brain edema; possible herniation and death
2. Micro - organizing neomembrane with "leaky vessels" complete by 15 to 21 days; newest bleeding usually side nearest brain; liquefaction starts at about 10 days, complete at about 21 days

IV. Traumatic Non-Missile Parenchymal Injuries

A. **Concussion** - transient loss of consciousness; sometimes seizures; recovery over hours to days; no sequelae; no pathology

B. **Diffuse Axonal Injury** ("shear injury")

1. Cause - sudden angular deceleration/acceleration; stretching and shearing nerve cell processes in white matter
2. Clinical - nearly half of all primary intra-axial traumatic brain lesions; usually severe closed head injury; typically lose consciousness at time of impact; diffuse and bilateral; primary cause of post-traumatic dementia and persistent vegetative state
3. Pathology
 - a. Locations - lobar white matter (especially grey-white interface), corpus callosum, dorsolateral upper brainstem; disruption of penetrating blood vessels causing multiple small hemorrhages
 - b. Microscopic axonal bulbs (retraction balls); chronic - nonspecific atrophic changes

C. **Contusions** - superficial foci of punctate or linear hemorrhages along gyral crests - do not conform to vascular distribution (thus are not infarcts)

1. Cause- brain striking bone or dura; depressed skull fracture
2. Clinical - second most common primary traumatic neuronal injury (45%); often coalesce and become more apparent 24 to 48 hours after injury; prognosis - source of focal seizure activity; infants usually get subcortical tears instead of contusions
3. Pathology
 - a. Gross - nearly half involve temporal lobes - poles, inferior surface and around sylvian fissure; 1/3 frontal - poles and inferior; 25% parasagittal; rarely brainstem strikes tentorium; can be associated with lacerations (tearing of superficial brain), contusions are important cause of subarachnoid hemorrhage; healed - depressed, brown, firm, may be adherent to dura (never seen in infarctions)
 - b. Micro - unlike infarcts - subpial molecular layer disrupted; edema and mass effect increases for first few days; hemorrhage, neutrophils and necrosis, followed by macrophage infiltrate, then when healed - gliosis and hemosiderin-laden macrophages
4. Types
 - a. **Coup** - immobile head, blunt force, most pronounced under point of impact
 - b. **Contrecoup** - moving head, immobile surface, severest opposite point of impact
 - c. **Gliding** - parasagittal - brain anchored to dura by arachnoid granulations, subcortical tissue glides further

D. Intracerebral hemorrhage

1. Cause - mechanical shearing of vessels
2. Clinical - depends on location
3. Pathology - usually frontal or temporal (with underlying contusions) and/or deep grey (associated with diffuse axonal injury)

E. Edema

1. Cause - increased blood volume, increased brain water content or both
2. Clinical - isolated or co-existent with other lesions, focal or diffuse (10-20% of severe brain injuries); twice as common in children as in adults; unilateral hemispheric swelling associated with subdural hematoma in 85%; severe edema usually takes 24-48 hours to develop

3. Pathology - brain soft, gyri flattened, ventricles small; more spaces and spaces enlarged on tissue sections

V. Missiles

A. Depressed - missile does not enter cranial vault - causes depressed skull fracture

B. Penetrating - enters - does not exit

C. Perforating - traverses and leaves

D. Pathology - lacerations, hemorrhage, edema, maybe radial displacement (may cause inferior frontal and temporal contusions, orbital fractures)

E. Higher incidence of infection and post-traumatic seizures with penetrating and perforating injuries

VI. Spinal cord injury

A. All the things we saw in the brain - epidural, subdural, subarachnoid hemorrhage, hematoma, contusion, laceration, etc

B. Compression, transection, ischemia

VII. Sequelae

A. Hydrocephalus

B. Organic brain syndromes

C. Seizures

Sample questions

1. During a fight at the Level 2 bar downtown Charleston, a patron is knocked backwards off a barstool and lands on the back of his head. He remains conscious and continues the fight until someone decides to stop the altercation by buying everyone a round of beers. Which of the following pathologic findings is most likely to be present as a consequence of this injury:

- A Occipital lobe contusions
- B Subarachnoid hemorrhage
- C Inferior frontal lobe contusions
- D Anterior pituitary necrosis
- E Skull fracture with epidural hematoma

2. An elderly nursing home patient has had a history of several falls, but has never lost consciousness and has remained stable. She develops increasing mental confusion associated with a mild right hemiparesis over several weeks. A CT scan demonstrates left hemispheric compression. These findings are most consistent with:

- A Epidural hematoma
- B Glioblastoma multiforme
- C Hypertensive hemorrhage
- D Cerebral abscess
- E Chronic subdural hematoma