

Objectives:

1. Describe the mechanisms by which the brain is injured following closed-head injury.
2. List delayed sequelae/complications of CNS trauma.
3. Compare hemorrhage into epidural, subdural and subarachnoid spaces:
 - List arteries and/or veins and CNS structures involved.
 - Describe the pathogenesis of the lesions.
 - Describe clinical manifestations and relate the time of injury to symptoms and recovery or death.
 - Describe the gross and microscopic appearance of lesions.
 - Discuss CSF findings.
4. Define concussion: discuss associated clinical and pathological changes.
5. Discuss contusions with respect to:
 - pathogenesis
 - location
 - pathology
6. Discuss causes, most common locations, pathology and clinical signs of spinal cord trauma. Describe the types of neurological deficits and locations of lesions due to Wallerian degeneration produced by transection of the cord.

I. General Principles

Head injury may result in any combination of skull fracture, parenchymal injury, and/or vascular injury. All of these have different consequences.

A. Classification of traumatic injuries.

- Trauma may produce closed or open head injuries.
- Injuries may be penetrating or blunt. Penetrating injuries are always open head injuries. In blunt trauma nothing penetrates through the skull, but there may be severe damage.
- Skull fractures may be depressed or non-depressed

B. Mechanisms of damage in closed-head injury

1. Linear acceleration or deceleration of head

- coup lesions (directly beneath impact),
- contrecoup lesions (direct opposite the site of impact),
- intermediary coup lesions

2. Rotation of brain within cranial cavity

- shearing of bridging veins producing subdural hemorrhage
- shearing of small vessels producing petechial intracranial hemorrhages or subarachnoid hemorrhage
- shearing stresses in brain causing rupture or stretching of axons
- contusions: the orbital surfaces may be damaged by contact with the floor of the anterior fossa; the temporal lobe tips may be damaged by edges of the sphenoid ridge; the corpus callosum may be damaged by the falx cerebri; the superior surface of the cerebellum or brainstem may be injured by contact with the tentorium cerebelli.

3. Secondary damage may be produced by the space-occupying effects of edema and/or hematoma

C. Additional mechanisms of damage involved in open-head injuries.

- Direct inoculation of bacteria
- Laceration by bony fragments

D. Brain swelling

Increased intracranial pressure and herniation can be a lethal complication in head trauma. The two major factors which contribute to brain swelling are edema and increased cerebral blood volume.

E. Delayed sequelae/complications of CNS trauma

- Post-traumatic epilepsy, due to seizure activity initiated at sites of meningocerebral cicatrix (scar tissue)
- Hydrocephalus - obstruction of CSF resorption after subarachnoid hemorrhage
- Delayed intracerebral hemorrhage - may occur days or months later (probably due to partial tearing of vessels during trauma with subsequent rupture)
- Psychological deficits (post-traumatic syndrome) -symptoms include headache, dizziness, anxiety, poor concentration; morphological substrate unknown
- White matter degeneration (pathogenesis not clear, may be related to stretching or shearing of axons during trauma)

F. Mild traumatic brain injury

Mild head injury is defined as duration of loss of consciousness of less than 30 minutes. Clinically, patients may appear stunned, dazed, drowsy, indifferent, disoriented, or may have headache, nausea or vomiting. Mild traumatic brain injury is a leading cause of neurological morbidity and can have devastating effects on the lives of patients and families. Mild TBI often goes undetected and significant disability may occur. Management includes psychological counseling, pharmacological treatment and neurorehabilitation. Neurological sequelae may include:

- Cognitive impairment (e.g. memory dysfunction, impaired concentration)
- Emotional changes (e.g. depressed, anxious, mood fluctuations)
- Behavioral changes (e.g. more impulsive, more easily angered)
- Somatic changes (e.g. headache, fatigue, dizziness, sleep disturbances)

II. Lesions in meninges and ventricular system

A. Epidural Hemorrhage

Epidural hemorrhage results in most cases from tearing of the middle meningeal artery (less often may occur from tearing of other vascular branches or venous sinuses). This event is usually associated with skull fracture, often of the temporal bone

Epidural hemorrhage is a rapidly expanding space occupying lesion and death may occur 2-12 hours after injury (bleeding is slower if the middle meningeal artery is not involved). The hematoma often causes uncal herniation and/or downward displacement of brainstem structures.

The classical clinical picture involves initial unconsciousness due to concussion, a lucid interval (seconds to hours) and progression to coma. However, the lucid interval does not occur in many cases. Symptoms include focal signs and indications of increased intracranial pressure.

Pathological examination shows a fresh epidural clot at autopsy.

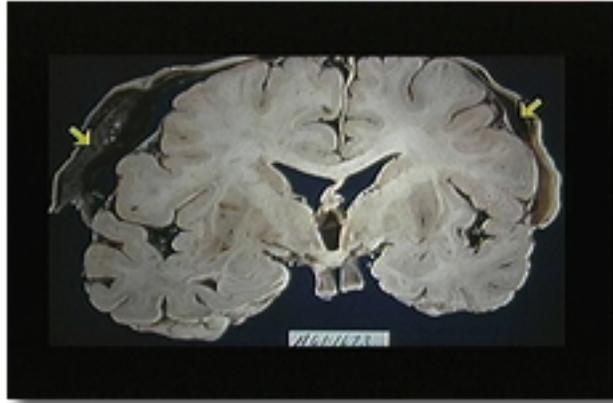
B. Subdural Hemorrhage

1. Blood accumulates between the dura and arachnoid as a result of shearing of bridging veins. Subdural hemorrhage is often associated with blunt trauma without skull fracture and results from rotation of brain.

2. The rate of progression is variable - subdural hemorrhage may be classified as acute or chronic and (and sometimes subacute) depending on the rate of accumulation of blood and thus rate of progression of symptoms. Acute and chronic conditions will be discussed separately.

- Acute subdural hemorrhage is associated with obvious trauma and is usually accompanied by contusion and cerebral artery tearing (leading to subarachnoid hemorrhage and bloody CSF). Symptoms develop within a few days (the onset of symptoms is slower than for epidural hemorrhage). The hemorrhage acts as a space-occupying lesion and symptoms may be focal and/or those of increased intracranial pressure. Consequences depend on the rapidity of surgical drainage and severity of concomitant damage to the brain.
- Chronic subdural hemorrhage is common in infants, the elderly, alcoholics, epileptics and demented individuals. Contributing factors include frequent head trauma and an enlarged subdural space (due to cerebral atrophy), providing less support for veins traversing this space. Chronic subdural hemorrhage follows mild trauma (sometimes forgotten by the patient) and symptoms may not occur for weeks to months after the trauma due to the slow rate of blood accumulation. Symptoms include seizures, headaches, confusion, behavioral changes, and signs of increased intracranial pressure; neurological signs may mimic those of degenerative disorders or neoplasms. Among diagnostic tests, CT scan or MRI is the most useful.

3. Pathology. The hematoma is encapsulated by a pseudomembrane composed of granulation tissue derived from the inflammatory reaction in the dura. The membrane forms initially at the clot surface facing the dura and is called the outer membrane. It extends around the clot to the surface facing the arachnoid, to form the thinner inner membrane. Subsequent episodes of rebleeding may occur, expanding the mass, followed by reorganization with a decrease in size. (Waxing and waning of neurological signs may correspond to the changes in hematoma size).



TRM 01

This image shows a subdural hematoma between the dura and the arachnoid.

C. Subarachnoid Hemorrhage

Accumulation of blood in the subarachnoid space to a greater or lesser degree is a consistent accompaniment of cerebral contusions. Upon lumbar puncture, grossly bloody CSF may be detected. In some cases in which a medium-sized superficial vessel is torn, subarachnoid hemorrhage may be the direct cause of symptoms; in most cases, bleeding arises from small vessels and is part of the surface bruising associated with fracture of the skull. Hydrocephalus may result if the subarachnoid bleeding or subsequent fibrosis obstructs CSF flow in the subarachnoid space.

III. Brain traumatic lesions

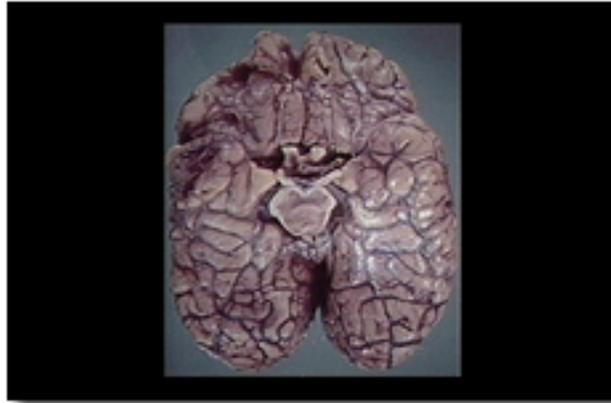
A. Concussion

Concussion is a transient neurologic dysfunction, which may include loss of consciousness, temporary respiratory arrest, and loss of reflexes, due to head injury. It is of instantaneous onset and is manifested by neurological symptoms without evidence of structural cerebral injury. A change in momentum of the head is thought to be a critical factor in producing concussion.

B. Contusions (areas of hemorrhagic necrosis)

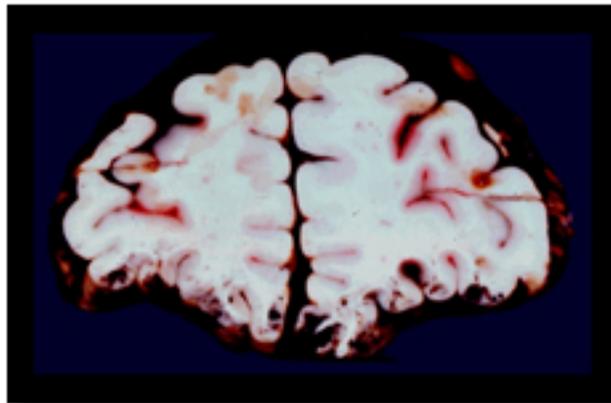
1. Pathogenesis: Following head injury, the brain strikes supporting structures (tentorium and falx cerebri), and bony projections of skull, producing superficial bruising of gyral crests.
2. Location: In general the most common sites are frontal lobe orbital surfaces and temporal lobes. Contusions can be produced by rotation of the brain or by linear forces

through the site of impact. The exact location may depend on conditions of the trauma. Contusions may be coup lesions (directly adjacent to the site of impact) or contrecoup lesions (on the opposite side of the brain from the impact). In the case of linear acceleration or deceleration injuries (i.e. a single blow to the unsupported head), contrecoup lesions are a common sequelae. A blow to the well-supported head results in severe skull fractures, often with absence of coup and contre-coup lesions (the head does not accelerate or decelerate and the skull absorbs much of the force).



TRM 02

Contusions are seen in frontal and temporal lobes, the most common sites for contusions.



TRM 03

Contusions on orbital surface of frontal lobes.

3. Pathology: In the most typical form of contusion, the summit of a cerebral gyrus is smashed, and the lesion has a wedge shape with its base toward the pia and the apex toward the white matter. All layers of cortex are regularly affected. In its early stage the hemorrhage remains bright red, and the surrounding brain tissue is edematous. When the lesion is older it becomes brick-red and finally golden orange-brown (due to deposition of hemosiderin), with a floor of glial tissue, covered by leptomenigeal fibrosis. The most chronic stage is sometimes called plaque jaune. Dura-arachnoidal adhesions

(meningocerebral cicatrix) later form on the surface, and frequently cause post-traumatic epilepsy.

C. Lacerations (rupture or tearing of brain tissue).

This physical disruption of tissue, often accompanied by contusions, is caused by a penetrating injury, e.g. by bony fragments or weapons. Meninges and cortex are both involved. The sequence of pathologic changes differs from that of contusion only with respect to the increased amount of hemorrhage, more disruptive effect and the more obvious fibroblastic proliferation and scarring of meninges in lacerations.

D. Diffuse Axonal Injury

Diffuse axonal injury is a major cause of prolonged traumatic coma. This type of lesion is present in 35% of head trauma deaths and is the most common cause of poor neurological outcome. Lesions are located in deep white matter regions. Pathological changes include axonal swellings within hours and later degeneration of fiber tracts.

IV. SPINAL CORD LESIONS

A. General comments.

The spinal levels most commonly involved with injury are the lower cervical spine (C4, C5, C6, C7, T1) and the thoracolumbar juncture (T12, L1, L2), the areas of greater mobility.

B. Pathophysiology - Mechanisms of injury

a. Direct injury results from force applied directly to the back of the neck or trunk and may cause fractures of spinous processes of laminal arches, concussion of the spinal cord, or direct compression of neural tissue by depressed bone fragments. Lacerations can result from knife or bullet wounds.

b. Indirect injury is a more common mechanism resulting from forces applied to the head and trunk or from movements that exceed the normal range, e.g. when the head is suddenly accelerated or decelerated in relation to the trunk.

C. Clinical signs

Neurological symptoms vary from complete loss of function below the injured segments to temporary loss of cord function with complete recovery. Specific symptoms depend on the

site of injury. After denervation, fibrillations, fasciculations, muscle atrophy, and groups of atrophic fibers in a muscle biopsy will be seen.

D. Pathological changes

The most common pathological changes are contusions, with necrosis, swelling and hemorrhage acutely to variable extents. Chronic changes include macrophages, gliosis and loss of architecture. Wallerian degeneration in distal axons (distal to the neuron cell body) will occur.