



IN THE NAME OF GOD



Head Trauma

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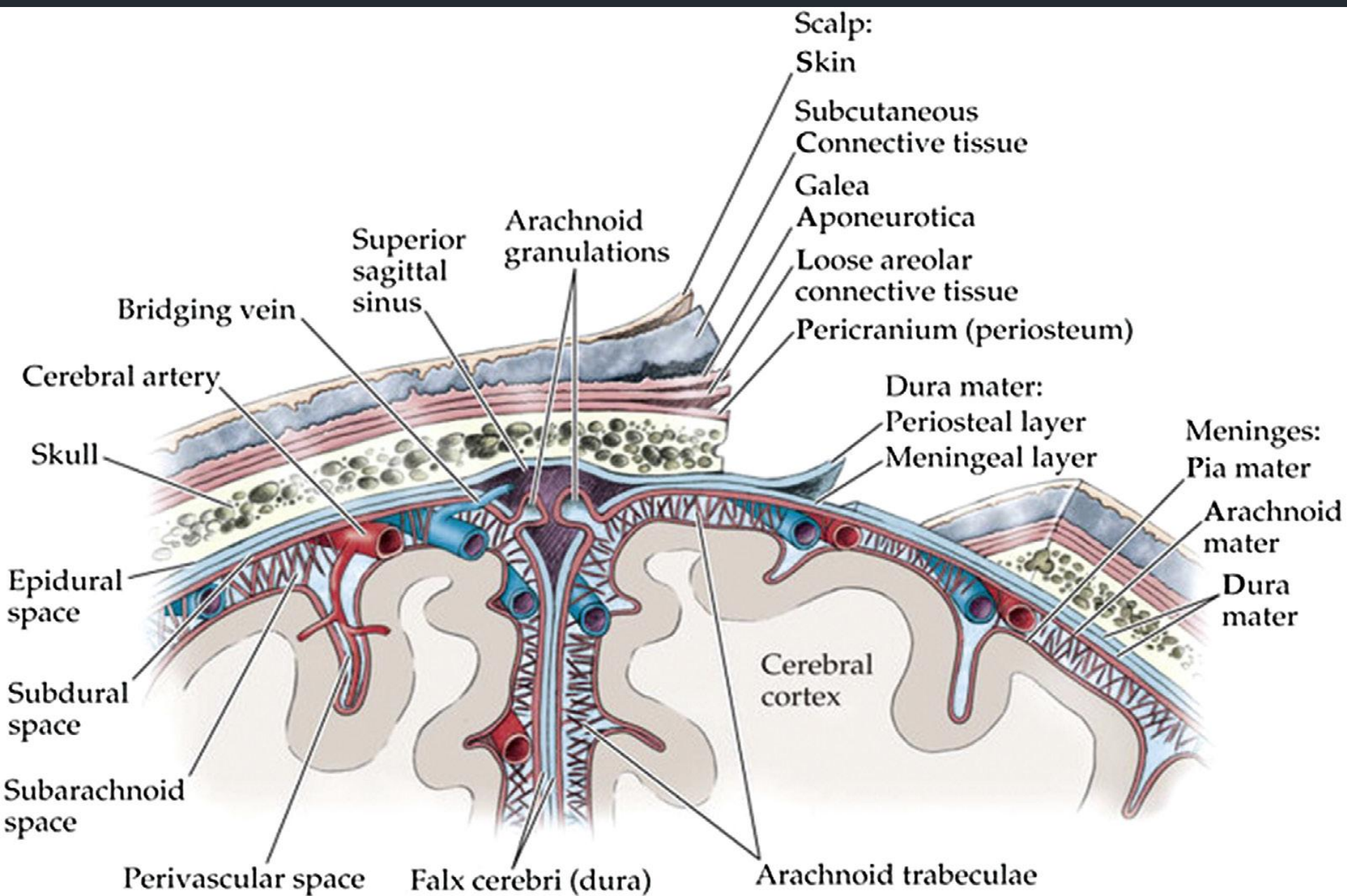
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TUMS



Mechanisms

- Blunt
- Penetrating
- Blast
- Rotational
- Acceleration-deceleration forces






Traumatic Brain Injury (TBI)

- Mild (GCS:13–15)
- Moderate (GCS:9–12)
- Severe (GCS:3–8)


TABLE 33.2 Glasgow Coma Scale		
Response	Score	Significance
Eye Opening		
Spontaneously	4	Reticular activating system intact; patient may not be aware
To verbal command	3	Opens eyes when told to do so
To pain	2	Opens eyes in response to pain
No eye opening	1	Does not open eyes to any stimuli
Verbal Stimuli		
Oriented, converses	5	Relatively intact CNS, aware of self and environment
Disoriented, converses	4	Well-articulated, organized, but disoriented
Inappropriate words	3	Random exclamatory words
Incomprehensible	2	Moaning, no recognizable words
No verbal response	1	No response or intubated
Motor Response		
Obeys verbal commands	6	Readily moves limbs when told to
Localizes to painful stimuli	5	Moves limb in an effort to remove painful stimuli
Flexion withdrawal	4	Pulls away from pain in flexion
Abnormal flexion	3	Decorticate rigidity
Extension	2	Decerebrate rigidity
No motor response	1	Hypotonia, flaccid—suggests loss of medullary function or concomitant spinal cord injury

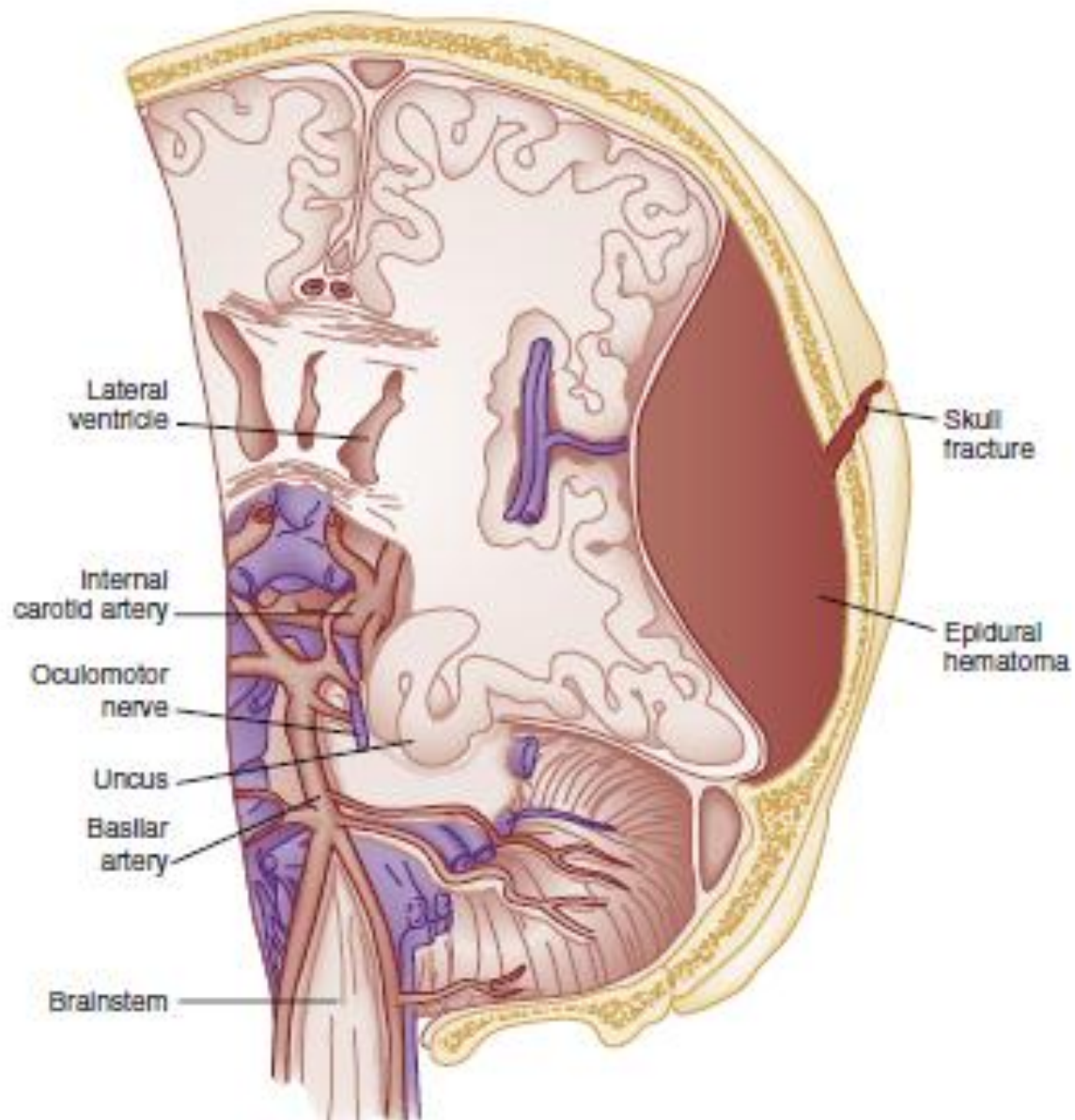
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- The motor component of the GCS is the strongest predictor of outcome following TBI.
 - Secondary systemic insults such as hypoxia and hypotension worsen neurologic outcome and should be corrected as soon as detected.`



Cushing reflex

- Progressive hypertension associated with bradycardia and diminished respiratory effort is a specific response to acute, potentially lethal increases in ICP. levels.
- Only one-third of cases of life-threatening increased ICP manifest the full triad of hypertension, bradycardia, and respiratory irregularity

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- A noncontrast head CT scan is the imaging modality of choice when TBI is suspected



BOX 33.2 Clinical Characteristics of Basilar Skull Fractures

Blood in ear canal

Hemotympanum

Rhinorrhea

Otorrhea

Battle's sign (retroauricular hematoma)

Raccoon sign (periorbital ecchymosis)

Cranial nerve deficits


Facial paralysis


Decreased auditory acuity

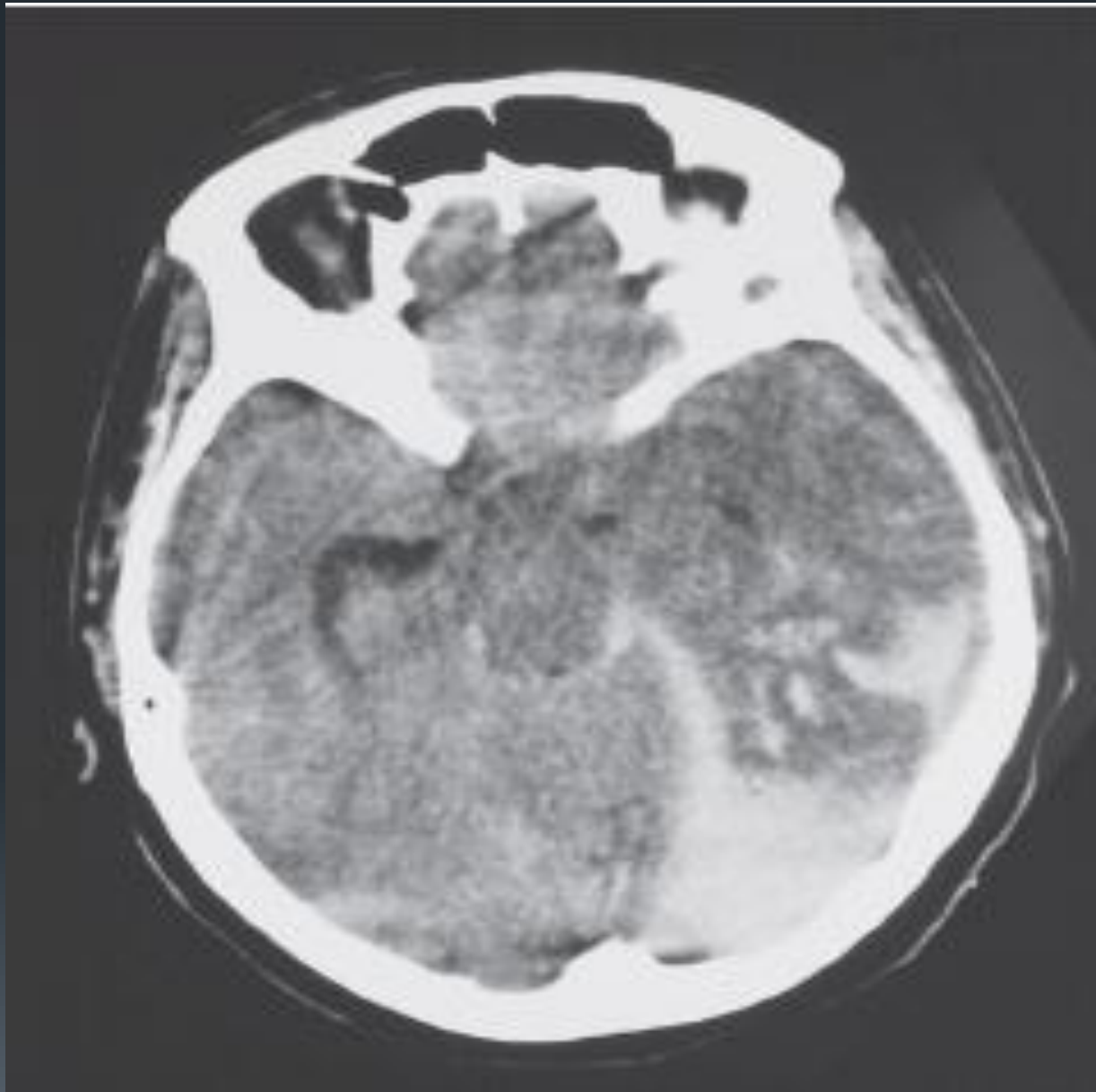
Dizziness

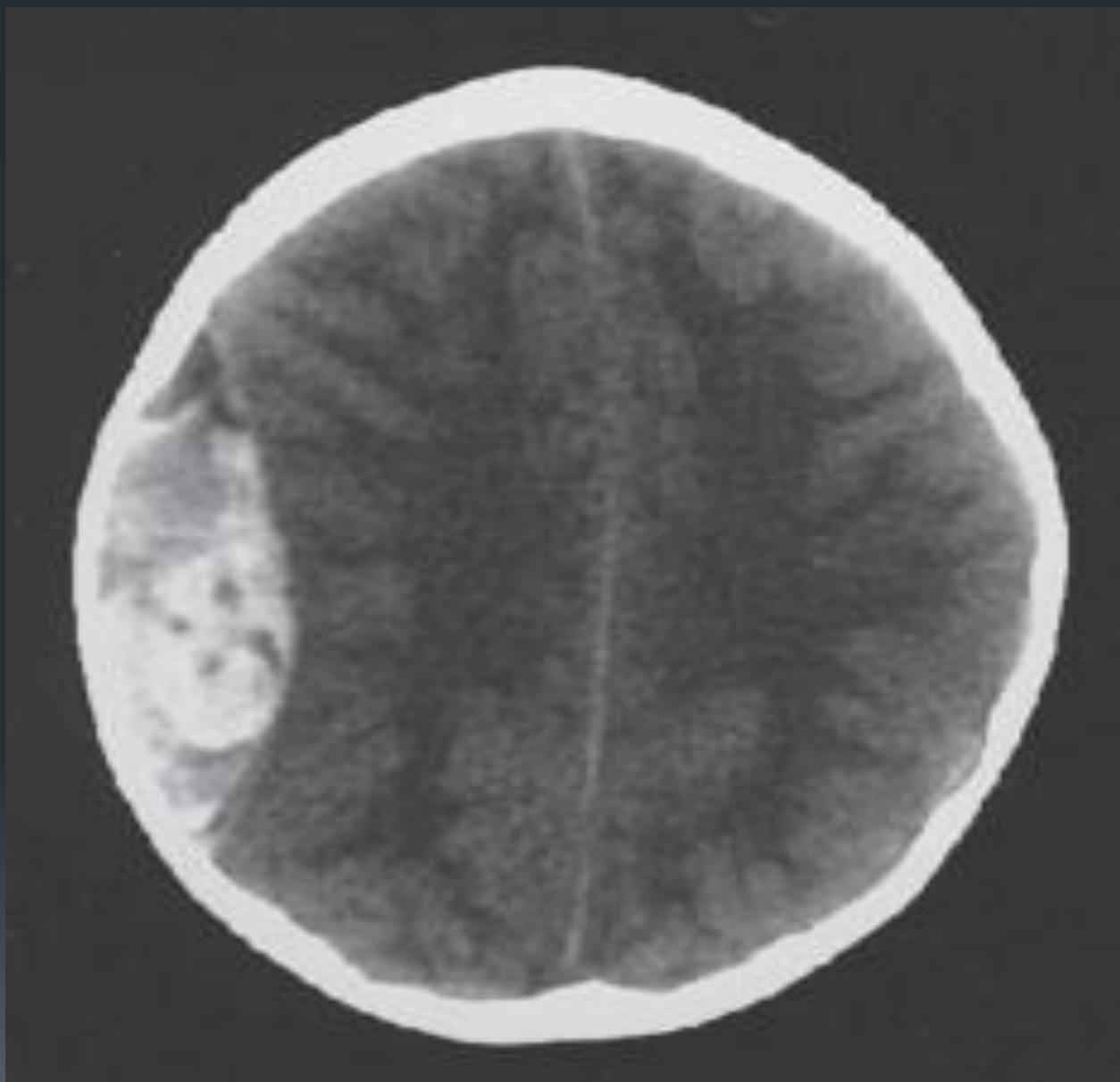
Tinnitus

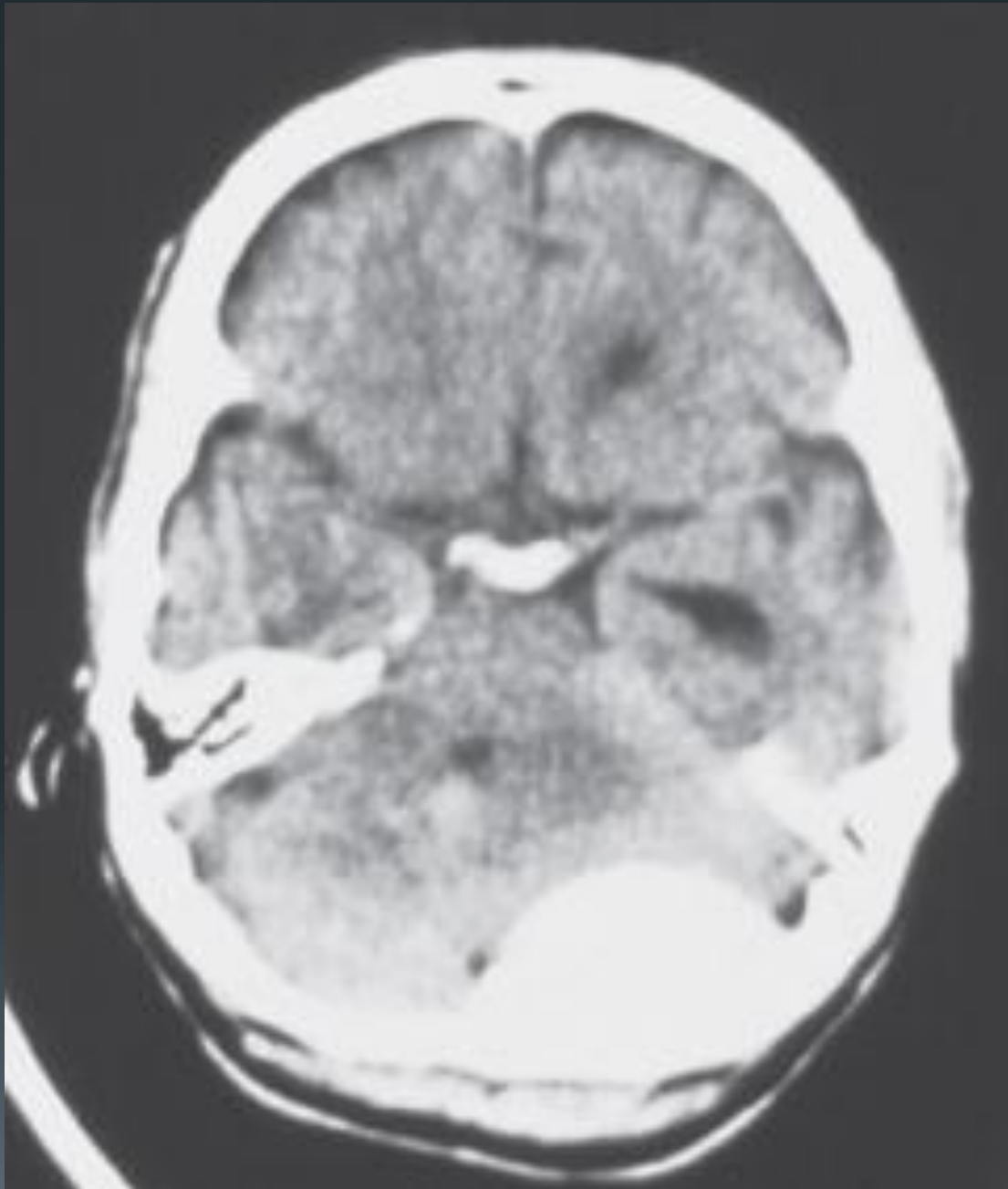
Nystagmus

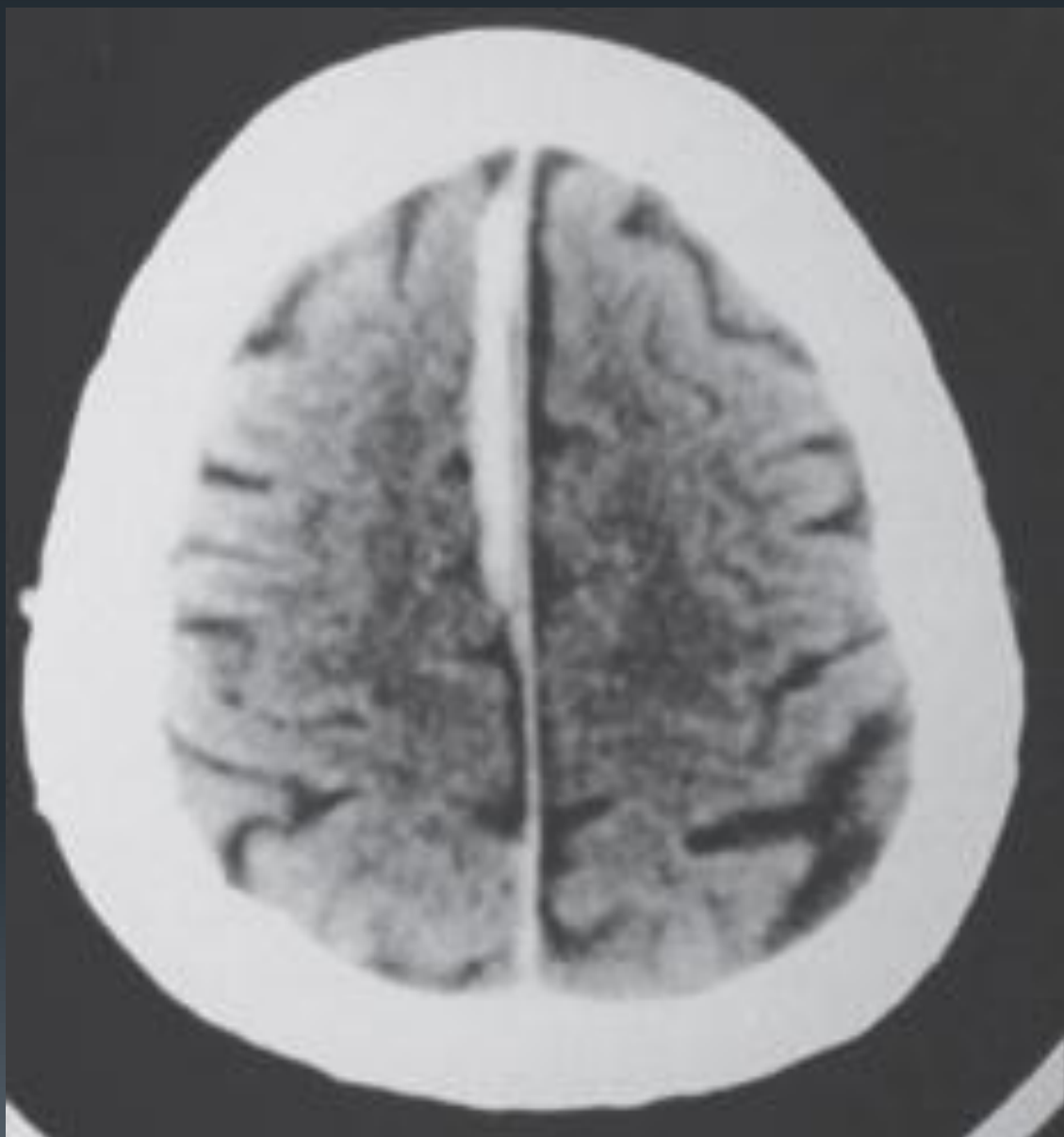
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- Patients can deteriorate from an expanding intracranial hematoma after a mild traumatic brain injury (MTBI), and should undergo serial evaluations, including GCS scoring

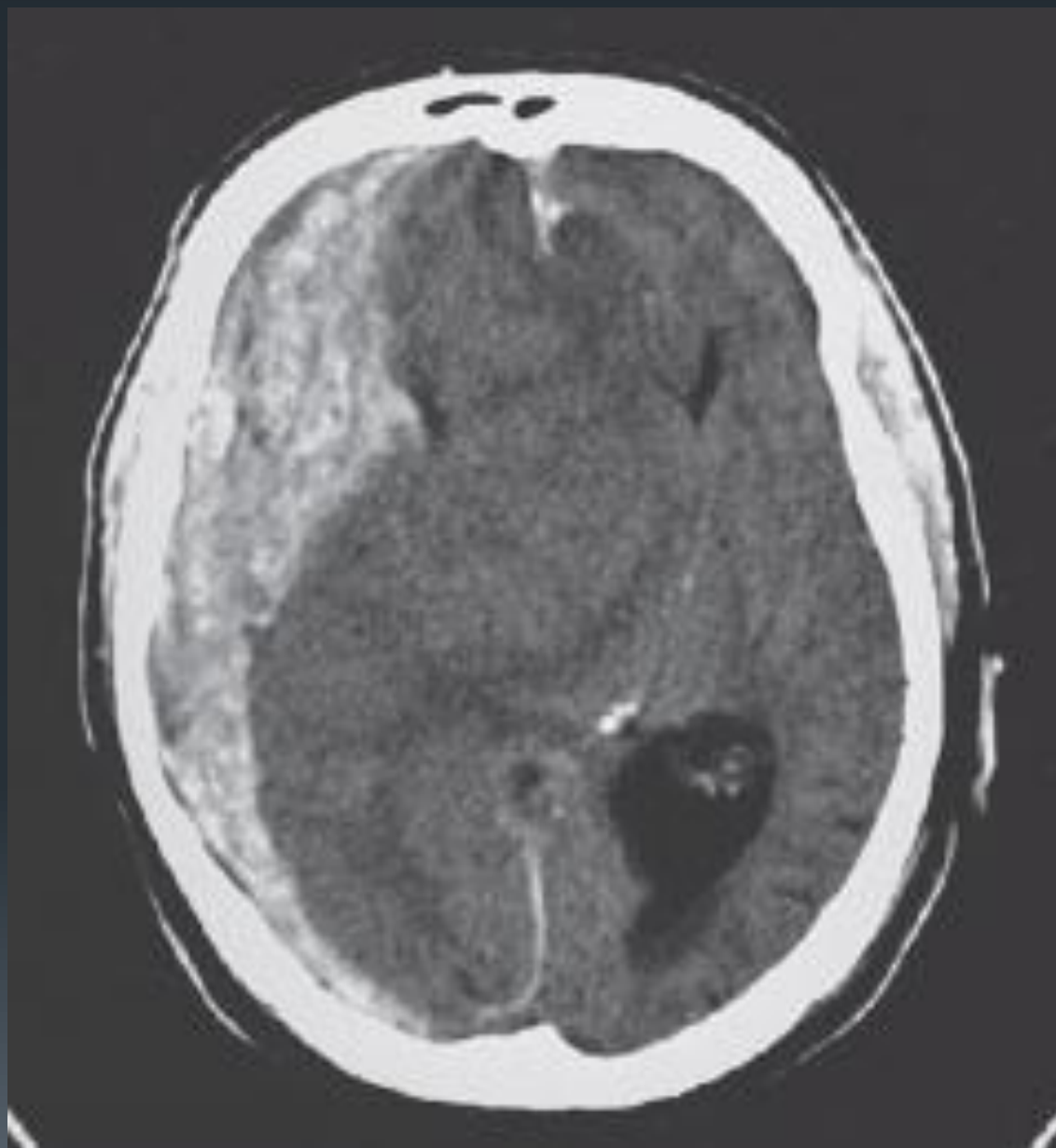
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- assess for disorientation, confusion, amnesia, or disordered awareness (with or without loss of consciousness Intoxicated individuals are high-risk patients.
 - Alcohol and drug use affect the GCS score and may significantly obscure the neurologic examination.

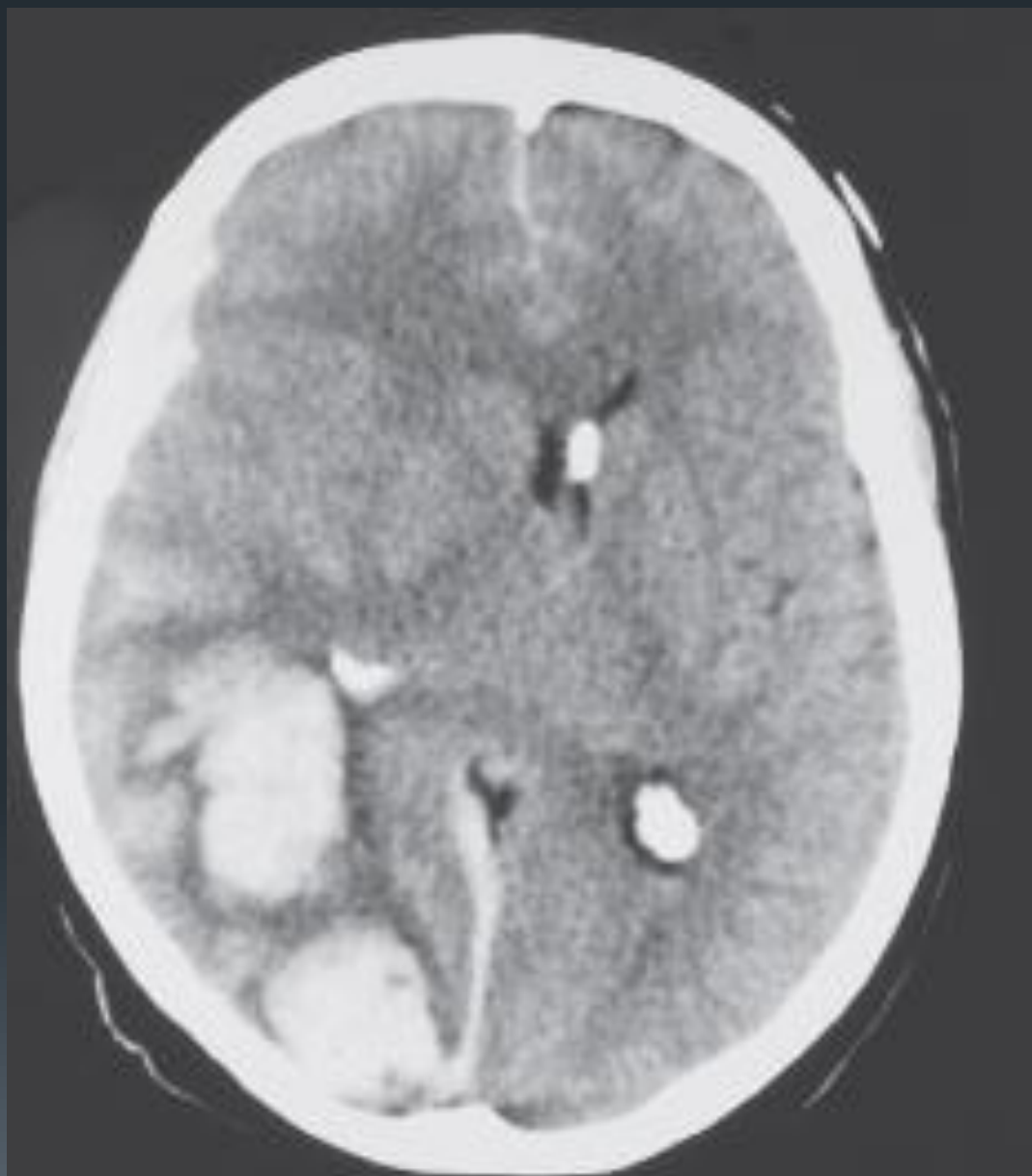


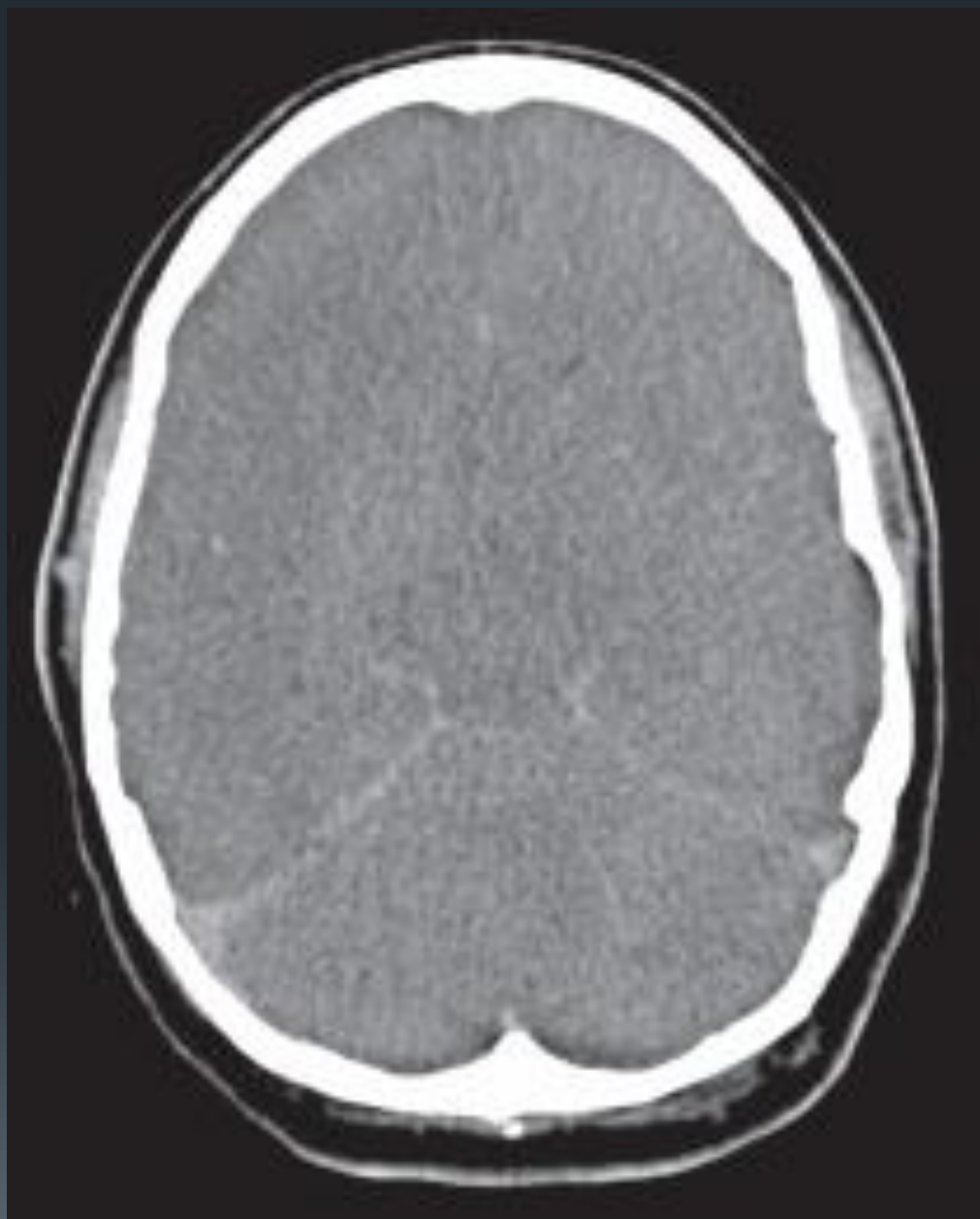












Emergency Department Management

- Monitoring of vital signs should be continuous including oxygenation (pulse oximetry) ventilation (capnography), heart rate, blood pressure, and temperature.
- Tetanus immunization status
- Pregnancy status in women of childbearing age should be verified.





■ Airway

- Primary airway compromise in the setting of head trauma may result from craniofacial or neck trauma, bleeding, or vomiting.
- Secondary airway compromise may also result from brain injury, as in the case of loss of brainstem reflexes, patient agitation, severe systemic hypotension, or alterations in mental status. In such cases, the airway should be secured early to protect against aspiration and prevent secondary brain injury as a result of hypoxia or hypercarbia

Hypotension

- Fluids or blood transfusion should be delivered to maintain a blood pressure as close to normal as possible, with a SBP of at least 110 mm Hg for those 15 to 49 and over 70 years of age, and at least 100 mm Hg for those 50 to 69 years old

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- Reversal of anticoagulation
 - Patients taking warfarin anticoagulants should have these medications reversed in the case of ongoing intracranial bleeding.
 - Intravenous (IV) vitamin K, fresh frozen plasma, or 3-factor or 4-factor prothrombin complex concentrate (PCC). While specific international
 - Target of an INR less than 1.5 is recommended

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- Inadequate evidence to support the routine use of platelet transfusion for intracranial hemorrhage for patients taking preinjury antiplatelet medications (e.g., aspirin, clopidogrel).
 - The transfusion of platelets in patients on antiplatelet medications does not reduce mortality.




Seizure prophylaxis

- The occurrence of seizures in the immediate post-trauma period is not predictive of future epilepsy
- Early seizures can cause hypoxia, hypercarbia, release of excitatory neurotransmitters, and increased ICP, potentially worsening secondary brain injury



- If the patient is actively seizing, benzodiazepines are administered as effective, rapid-acting, first-line anticonvulsants.
- If an IV is available, administer lorazepam (0.05–0.1 mg/kg IV up to 4 mg at a maximum rate of 2 mg/minute; may repeat at 3 to 5 minutes if seizures continue).

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- An effective alternative is IV diazepam (0.15 mg/kg IV, up to 10 mg per dose at a maximum infusion rate of 5 mg/minute; may repeat at 3 to 5 minutes if seizures continue, up to a total dose of 30 mg).
 - If an IV is not available, IM midazolam (0.2 mg/kg up to 10 mg) can be used




Corticosteroids

Corticosteroids have no benefit for patients with head trauma

An increase in adverse events, including infection, gastrointestinal bleeding, and mortality.


In patients with severe TBI, high-dose methylprednisolone is associated with increased mortality and is contraindicated


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- Anticonvulsant prophylaxis with phenytoin or levetiracetam and broad-spectrum antibiotics should be given to patients with penetrating brain injuries for 7 days postinjury.



Disposition

- Most patients with MTBI can be discharged from the emergency department (ED) with a normal examination and after a reasonable period of observation (4–6 h) or following a negative CT scan of the head.

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- Patients should be discharged with instructions describing the signs and symptoms of acute and delayed complications of MTBI

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- Athletes with a concussive head injury should be immediately removed from play and not return until they have been evaluated by a health care provider with expertise in concussion management.
 - There should be a gradual stepwise increase in physical activity.

TAHNK YOU

