Trauma Anesthesia

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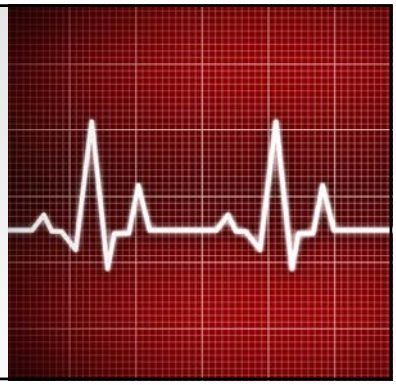

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GOLDEN HOUR

- The **first hour** following a trauma during which aggressive resuscitation can improve the chances of survival and restore the normal functions.
- Early pre-hospital care, early transport, aggressive resuscitation and interventions in ED, continued care in ICU have a definite and significant role in preventing deaths due to trauma.

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TRAUMA

- Not a biologic basis for a disease
- Results of an external force ultimately destroying/disrupting normal structure and function of the human body
- Known as a disease of human behavior
- One death every 3 minutes
- WHO prediction
  - Injury ~ leading cause of death by 2020

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Coordinated Management of Care

- *MOA?*
  - Typically warrants immediate surgery
    - Affecting multi organs
      - Co morbidities – all affect how you proceed w anesthesia different than the 'norm'
- Care cannot be postponed
  - Medical history often?
    - Patient cannot help
      - ETOH, 'street drugs', other situations ~ add to challenge
- Cannot delay
  - Work with what you have
    - Often not much

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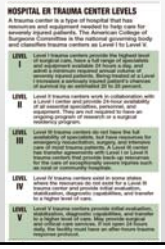
Modern Trauma Systems

- Improve patient outcomes
  - Risk of death decreased with increasing levels of system and care



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ATLS by ACS

Framework for management and evaluation from prehospital to hospital

- Phases
  - Primary Survey
    - Logical and sequential treatment strategies based on RAPID ASSESSMENT
      - ABCDE- Airway, breathing, circulation, disability (NEURO), exposure the patient (environment impact to body i.e. blast injury, burns, other)
    - GOAL
      - Identify and rapidly manage life threatening injuries
      - Vascular access, establish airway, other
  - Secondary Survey
    - GOAL
      - Continued resuscitation and stabilization efforts
      - Head to toe assessment, neurologic examination, other

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Blunt versus Penetrating Trauma

- **Blunt**
  - Direct impact, deceleration, continuous pressure, shearing, and rotary forces
  - *Factors associated with high levels of energy*
    - Result from high speed collisions, falls, other
  - Newton's first law
    - Object tends to remain in motion until it is affected by an outside force
    - Abrupt decelerations creates negative gravitational forces
    - Outer shell of body abruptly decelerates, internal organs continue forward at original velocity and torn from attachments
    - Disruption of connective tissue, blood vessels, nerves

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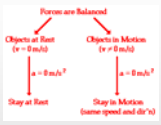
Laws of Energy and Motion

- *Newton's first law of motion* states that a body at rest will remain at rest and a body in motion will remain in motion unless acted on by an outside force.
- the vehicle hits a tree and stops →the unrestrained person continues in motion → hits the steering column, dashboard, and windshield →stops the forward motion of the torso or head →but the internal organs of the person remain in motion →hit the inside of the chest wall, abdominal wall, or skull → effect



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MVA

Blunt trauma – rarely occurs in isolated body systems
- Entire body impacted

- 5 types of MVA
  - Head on
  - Rear impact
  - Side impact
    - Motor vehicle accidents, the most common cause of blunt abdominal injuries, often result in hepatic injury to the passenger if impact is on the passenger's side
    - Rotational impact
  - Rollover

- Injuries categorized: above or below waist
- Upper body collide with front parts of car (dashboard, other)
  - Expect: head, neck chest, abdomen, upper extremities

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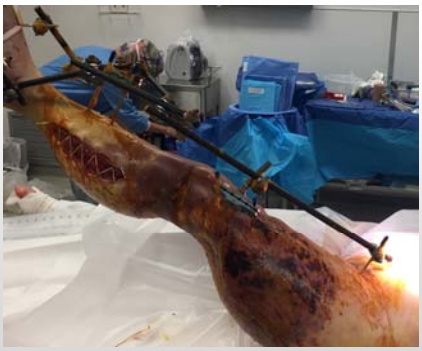
Or categorized below the waist; pedestrian versus vehicle

Knees, femurs, acetabular fractures



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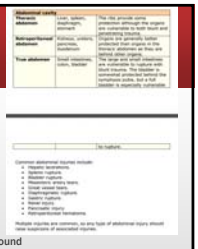
Case Scenario #1



- Rollover MVA
- Young healthy male adolescent
- Sustained Injuries:
  - Spleen, liver, hollow organs, other
  - Uncontained bleeding = hemoperitoneum

Massive hemorrhage with shock complicated by external bleeding

*** Bleeding from abdominal injury MOSTLY internal (within the abdominal cavity)

Critical combination injury - associated penetrating injury = external bleeding through the wound

- With hollow organ injury, the contents of the organ (for example, stomach acid, stool, or urine) may enter the abdominal cavity and cause peritonitis

- Remember: Blunt trauma is more difficult to assess because injuries are less obvious, so massive blood loss may occur before injuries are detected

3 types of blunt injuries:

1) crush, which results from compression,
2) shear, which involves tearing, and
3) burst, which relates to sudden increased pressure (such as from an explosion)

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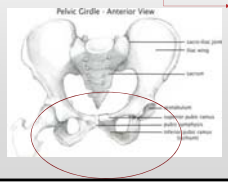
Case Scenario #2
Jump from burning building, 7 stories, young healthy obese female

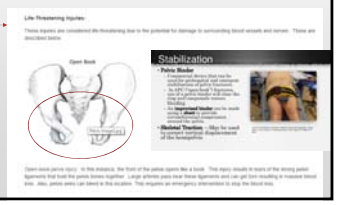
- Sustained injuries:
  - Open book pelvic fracture

THE AMERICAN ASSOCIATION FOR THE SURGERY OF TRAUMA

Injury to the pelvis occurs in approximately 8-9% percent of what is termed blunt trauma: falls, motor vehicle crashes (cars and motorcycles), bicycle wrecks, and pedestrians being struck by moving vehicles.

Normal Pelvis





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BUT ALSO, Perforated Viscus

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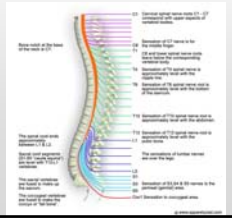
Your neck is on the line
Cervical Spine Injury – blunt trauma

- All blunt trauma victims including those ultimately w/o high risk mechanisms of cervical spine injury should be suspected of and treated as unstable neck until proven otherwise!



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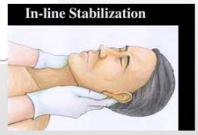
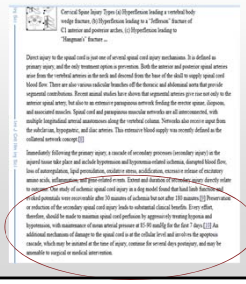
Cervical Spine Injury Patterns from Blunt Trauma

Table 2. NEXUS Criteria For Radiographic Evaluation Of The Cervical Spine Following Blunt Trauma[6]

1. Midline cervical tenderness
2. Focal neurologic deficits
3. Altered level of consciousness
4. Evidence of intoxication
5. Painful distracting injury





Cervical Spine Injury Types (a) Hyperflexion leading a vertebral body wedge fracture, (b) Hyperflexion leading to a "Jefferson" fracture of C1 anterior and posterior arches, (c) Hyperflexion leading to "Hangman's" fracture ...

Direct injury to the spinal cord is just one of several spinal cord injury mechanisms. It is defined as primary injury, and the only treatment option is prevention. Both the anterior and posterior spinal arteries arise from the vertebral arteries at the neck and descend from the base of the skull to supply spinal cord blood flow. There are also various radicular branches off the thoracic and abdominal aorta that provide segmental contributions. Recent animal studies have shown that segmental arteries give rise not only to the anterior spinal artery, but also to an extensive paraspinous network feeding the ventral spinal, dorsum, and associated muscles. Spinal cord and paraspinous muscular networks are all interconnected, with multiple longitudinal arterial anastomoses along the vertebral column. Networks also receive input from the subclavian, hypogastric, and iliac arteries. This extensive blood supply was recently defined as the collateral network concept [1]

Immediately following the primary injury, a cascade of secondary processes (secondary injury) in the injured tissue take place and include hypotension and hypoxemia-related ischemia, disrupted blood flow, loss of autoregulation, lipid peroxidation, oxidative stress, acidification, excessive release of excitatory amino acids, inflammation and pain-related events. Extent and duration of secondary injury directly relate to outcome. The study of ischemic spinal cord injury in a dog model found that hind limb function and evoked potentials were recoverable after 30 minutes of ischemia but not after 180 minutes.[2] Preservation or reduction of the secondary spinal cord injury leads to substantial clinical benefits. Every effort, therefore, should be made to maximize spinal cord perfusion by aggressively treating hypoxia and hypotension, with maintenance of mean arterial pressure at 85-90 mmHg for the first 7 days.[3] An additional mechanism of damage to the spinal cord is at the cellular level and involves the apoptosis cascade, which may be initiated at the time of injury, continue for several days postinjury, and may be amenable to surgical or medical intervention.

Int J Crit Illn Inj Sci. 2014 Jan-Mar; 4(1): 50–56.

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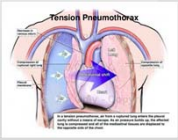
Thoracic Trauma – BLUNT

- Unique concerns
  - Not wearing seat belts
  - Injured chest structures that prevent optimal resuscitation
    - Chest wall, lungs and airways, heart and pericardium, great vessels of thorax
  - Pneumothorax
  - Take seriously; many missed

Tension pneumothorax

- What is it exactly? Lung puncture within thoracic cavity
- Creates a one way valve
  - Traps air between layers of pleura
- With each breath, more air becomes trapped, increasing intrapleural pressure until exceeds all other intrathoracic pressures
- Enlarging pleural cavity collapses ipsilateral lung and shifts structures of mediastinum into opposite hemithorax
- Size will rapidly increase during PPV



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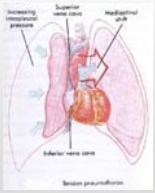
Clinical signs of tension pneumothorax

- Hypotension
- Subcutaneous emphysema, head and neck
- Unilateral decrease in BS
- Diminished chest wall expansion
- Distended neck veins
- Tracheal shift



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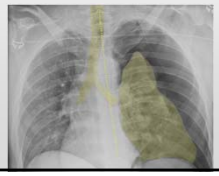
Treatment of tension pneumothorax

- Emergent situations
  - Needle decompression – 14g
  - Second intercostal space just above 3rd rib
  - Midclavicular line

  OR

  4th or 5th interspace laterally!
- Relief of pressure
  - Restore cardiac function
  - Will hear the RUSH of air
  - MOVE TO CHEST TUBE INSERTION



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Blunt Trauma

- May also see pericardial tamponade
  - Restricts filling of the cardiac chambers during diastole
  - Produces a fixed low cardiac output
  - Life threatening emergency

Clinical Manifestations of Cardiac Tamponade

- Increased central venous pressure
- Activation of the sympathetic nervous system (tachycardia and vasoconstriction)
- Equalization of right and left atrial pressures and right ventricular end-diastolic pressures at about 20 mmHg (exception may be accumulation of blood and clots over the right ventricle, as may follow cardiac surgery)
- Paradoxical pulse (decrease> 10 mmHg in systolic blood pressure during inspiration)
- Hypotension (low cardiac output)

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Pulmonary Contusions

- 70% of blunt thoracic trauma present with some degree of pulmonary contusion
- Injuries to the alveoli without gross disruption of architecture
- Bruise to the lung tissue
  - Protein rich fluid exits ruptured pulmonary capillaries
  - Enters alveolar membrane and interstitial space
  - Gas diffusion problems
  - ARDS
  - Problem?
    - High pressures and barotrauma
      - Solution
        - High frequency oscillation ventilation
        - CBP
        - Use low tidal volumes and maintain peak pressures < 32 cm h_2O
        - NOT NECESSARILY 100% FiO_2

PULMONARY CONTUSION

- It is damage to the lung tissues resulting in hemorrhage and localized edema.
- Ecchymosis at the site of the damage
- Crackels
- Cough may be present with blood-tinged sputum.
- Pulmonary contusions tend to worsen over a 24–to 48–hour period and then slowly resolve unless complications occur (infection, ARDS).
- Patients with severe contusions may require endotracheal intubation and mechanical ventilation

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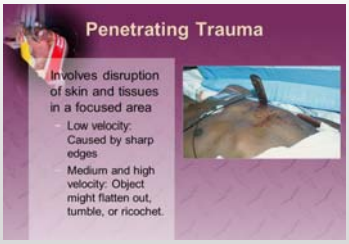
Penetrating Trauma

- High Velocity Projectile Injury



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Damage - High Velocity Projectile

3 interactive factors

1- the type of wounding instrument, knife, missile bullet or fragment, shrapnel, etc

2- the velocity of the missile at time of impact

3- the characteristics of the body tissue that the object passes through

Key point – low or high velocity penetration ~ damage ultimately results from disruption of normal anatomy and physiology. ***Velocity*** of the projectile is the most significant determinant of wound potential

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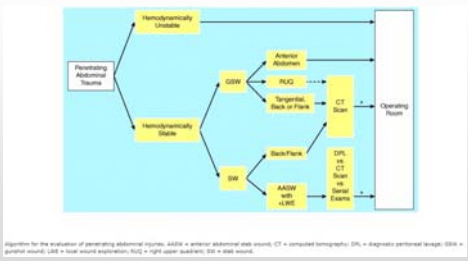

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Damage Control Surgery

- Surgical procedures – multistep
  - Early repair…not intended to be definitive but stabilization
    - ER to OR to ICU
      - Return to OR …several times and phases
- Immediate Rapid Control of:
  - Hemorrhage *****
  - *Prevention the lethal triad: acidosis, hypothermia and coagulopathy*
  - Limitations of crystalloid exist !!!
    - Blood is required to correct deficiencies
    - Rapid transfusion protocols

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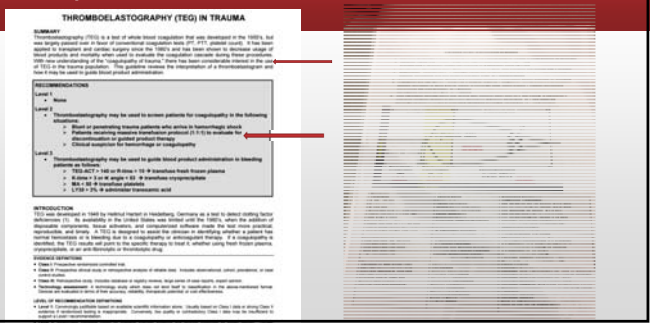
Rapid TEG


THROMBOELASTOGRAPHY (TEG) IN TRAUMA

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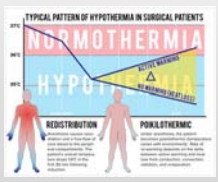
Additional Information

- Remember - blunt abdominal trauma → occult bleeding
- Immediate:
  - Abdominal ultrasound
  - FAST = Focused Assessment with Sonography for Trauma
  - CT
  - MRI
  - Angiography
  - BUT if critical – ER to OR
    - Large bore intravenous catheters
      - Above the diaphragm
      - Prior to opening the abdomen
      - Blood product in OR
      - WARM, WARM, WARM!



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The ABCDs of Trauma Anesthesia

- Anesthesia MUST facilitate rapid surgical management
- Our goal – rapidly move the patient to the OR
  - Perfusion of organ systems is directly related to TIME

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Treatment for Hemorrhagic Shock and Trauma

- Can be obvious, hidden, or a combination of the both
- Fluid Resuscitation
  - What is appropriate?
  - Often resuscitation is measured for success by BP
    - is this wrong?
  - Do view on other parameters
    - What is the state of repair of the injuries?
      - Little or no active bleeding?
        - ATLS: 2 L isotonic crystalloid
          - component therapy for a larger resuscitation with greater blood loss

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Fluid and further considerations

- Can worsen the clinical state if timing is off
- Ruptured or torn or damaged blood vessels? Blood containing nutrients, coagulation factors, catecholamines, and oxygen carrying capacity will be lost!
- Suggestion
  - Early resuscitations efforts ***before surgery***, {and fluid} should be done by 'hypotensive resuscitation'
    - What is it?
      - A clinical methodology that guides fluid therapy in a manner that avoids excessive bleeding by targeting fluid treatment toward maintaining a lower than normal systolic BP i.e. 85-90 mmHg

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Intravenous Access Catheters

- Poiseuille's Law
  - Speed is directly related to catheter diameter
    - Larger catheters decrease turbulence and increase flow
  - Rule of thumb? Short and FAT IV catheters ☺
  - Central Catheters? Not always, other access?
    - NO FEMORAL catheters used when major abdominal vascular or pelvic injury occurred
  - IV access above the diaphragm provides most assurance of staying intravascular, where needed!
  - Trauma centers
    - Typically RBCs replaced to provide oxygen carrying capacity and 1:1 with blood loss
    - Guided product administration by
      - Laboratory values
      - Vital signs
      - Blood loss
      - Perfusion to vital organs
    - NO HESPAN, NO GLUCOSE

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Coagulopathy and Trauma

- Once develops, M and M increases significantly
- Otherwise healthy people:
  - Elevated PT on admission indicates rapid hemorrhage, massive injury, and worsening perfusion state
  - Clot formation
    - Begins with vascular insult
    - Tissue injury = release of *tissue factor* from endothelium
    - Simultaneously vascular spasm causes platelets to migrate to injury
    - TF binds with activated circulation factor VII (FVIIa)
    - FVIIa interaction with TF begins converting prothrombin (II) to activated thrombin (IIa)
      - Resulting in thrombin burse on platelet surface
    - Conversion of thrombin completes the coagulation process yielding fibrin (1) connections that stabilize the clot
    - THIS IS UNDER NORMAL CONDITIONS

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In Trauma Situations….. i.e. 'abnormal conditions'

- Massive injury disrupts clotting cascade at several points in the process
- 4 mechanisms have been ID'd as primary causes of trauma-induced coagulopathy
  - 1 dilution of factors
  - 2 hypothermia/acidosis
  - 3 severe traumatic brain injury
  - 4 hemorrhagic shock

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Dilution of factors

- When massive resuscitation to replace blood volume and restore perfusion happens-
- Crystalloid dilutes coagulation factors and platelets and increases hydrostatic pressure
  - Leads to poor clot formation and non surgical bleeding
- No clear answer but the experts say
  - Administer procoagulant products to maintain an INR of <1.5 and platelet count greater than 50,000
  - Point of care testing


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Hypothermia and Acidosis = the big enemy

Table 1: Traditional classification of hypothermia & revised classification for trauma patients

Degree of hypothermia	Traditional classification (°C)	Trauma classification (°C)
Mild	32–35	34–36
Moderate	28–32	32–34
Severe	20–28	< 32
Profound	14–20	
Deep	< 14	

- Heat loss happens with trauma situation
- Hypothermia results in significant coagulopathy
  - Alters platelet function
  - Reduces fibrin enzyme kinetics
  - When mixed with acidosis – all bad things happen
- Most heat loss in trauma environment is due to ***radiant loss***
- Acidosis
  - In the presence of low temperature– contributes to - lethal triad of acidosis, hypothermia and coagulopathy
  - Acidosis
    - Impairs coagulation proteases and becomes clinically significant at pH < 7.1
    - DO NOT GIVE SODIUM BICARBONATE it does not increase clotting function!!!!!!

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TBI

{Traumatic Brain Injury}

Overall goal of all surgical treatment is to prevent secondary injury by maintaining blood flow and oxygen to the brain & minimize swelling and pressure

Closed Head Injury–

No skull penetration

Cause - direct or indirect force to the head by rotational and/or deceleration

Open Head Injury– penetration of the skull with direct injury

Diffuse Axonal Injury– diffuse cellular injury to the brain from rapid rotational movement

Cause - motor vehicle accidents or shaking injuries

Axons are damaged or torn by the rapid deceleration; injury is from the shearing force disrupting the axons (white matter)

Contusion–bruise to a part of the brain; bleeding into the tissue

Penetrating Trauma– any object that enters the brain; direct injury by impact and pushing skull fragments into the brain

Secondary Injury– swelling and release of chemicals that promote inflammation and cell injury or death

Prevent 2nd Injury

Brain swelling increases ICP and prevents drainage of CSF

Causes further increase in pressure and brain damage. If this is not controlled or prevented the brain can herniate

Only way to prevent the primary injury is to prevent the trauma. The prevention of this secondary injury is the focus of the acute medical care after injury.

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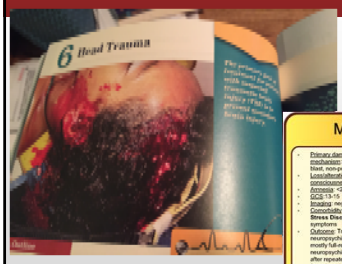
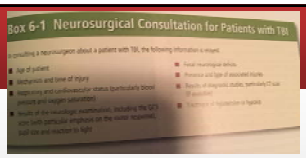
Traumatic Brain Injury





Mild	Moderate	Severe
Primary damage / Injury mechanism: predominantly blast, non-penetrating Loss/alteration of consciousness: <30 minutes Amnesia: <24 hours GCS: 13-15 Imaging: negative Comorbidity: **Post Traumatic Stress Disorder**; overlapping symptoms Outcome: Transient neuropsychiatric deficits, mostly full-recovery, long-term neuropsychiatric especially after repeated injuries are frequent	Primary damage / Injury mechanism: frequently mixed, blast + acceleration/deceleration, typically non-penetrating Loss/alteration of consciousness: >30 minutes, <24 hours Amnesia: >24 hours, <7 days GCS: 9-12 Imaging: transient changes Comorbidity: PTSD, other injuries Outcome: mild-to-moderate, typically chronic, neurological and neuropsychiatric abnormalities	Primary damage / Injury mechanism: complex, blast + acceleration/deceleration + penetration Loss/alteration of consciousness: >24 hours Amnesia: >7 days GCS: <9 Imaging: positive, lasting abnormalities Comorbidity: **Polytrauma**, such as multiple-organ injuries Outcome: death, significant, neurological and neuropsychiatric deficits, severe, chronic physical and neuropsychiatric disabilities

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The Glasgow Coma Scale

Glasgow Coma Scale		
Response	**Scale**	**Score**
Eye Opening Response	Eyes open spontaneously	4 Points
	Eyes open to verbal command, speech, or shout	3 Points
	Eyes open to pain (not applied to face)	2 Points
	No eye opening	1 Point
Verbal Response	Oriented	5 Points
	Confused conversation, but able to answer questions	4 Points
	Inappropriate responses, words discernible	3 Points
	Incomprehensible sounds or speech	2 Points
	No verbal response	1 Point
Motor Response	Obeys commands for movement	6 Points
	Purposeful movement to painful stimulus	5 Points
	Withdraws from pain	4 Points
	Abnormal (spastic) flexion, decorticate posture	3 Points
	Extensor (rigid) response, decerebrate posture	2 Points
	No motor response	1 Point
Minor Brain Injury = 13-15 points; **Moderate Brain Injury** = 9-12 points; **Severe Brain Injury** = 3-8 points		

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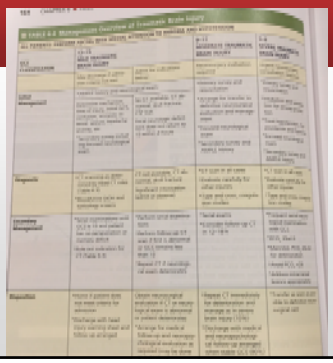
TBI



- ***GCS*** ~ Urgent neurosurgery consultation
- 1 survey/resuscitation
  - Intubation
- Treat low BP, low volume, hypoxia
- 2nd survey – AMPLE
- CT scan
- Type and Cross/Coagulation profile

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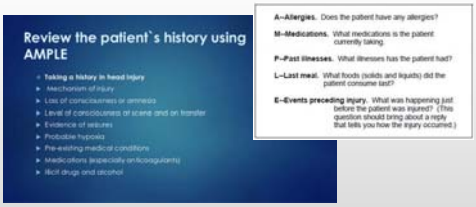
AMPLE -2nd Survey



A–Allergies. Does the patient have any allergies?

M–Medications. What medications is the patient currently taking.

P–Past illnesses. What illnesses has the patient had?

L–Last meal. What foods (solids and liquids) did the patient consume last?

E–Events preceding injury. What was happening just before the patient was injured? (This question should bring about a reply that tells you how the injury occurred.)

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Head Injury

- Anesthetic Management
  - Control of the airway
  - Maintenance of CV stability
    - Hypotension is a HUGE problem
    - Know CPP; systemic BP directly affects CBF
      - (CPP = MAP – ICP)
    - Hypoxemia and hypotension = high mortality rate
  - GCS <8 ~ INTUBATE
  - Maintain Sp02 > 90%
    - Hypercarbia and hypoxia both contribute to elevated ICP
    - Hyperventilation bad also!
  - Airway establishment
    - Induction (techniques) to prevent anything that would increase ICP
    - No nasal intubations
    - Place OGT not NGT

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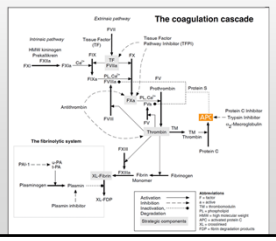
And TBI {traumatic brain injury}

- Theory
  - TBI causes a local release of TF from injured neurons, activating the protein C pathway and triggering the release of anticoagulant mediators
- Early management?
  - Plasma
- End- point of plasma administration.....normalize PT and INR
- Plasma requirements ~ unpredictable

Role in trauma induced coagulopathy?



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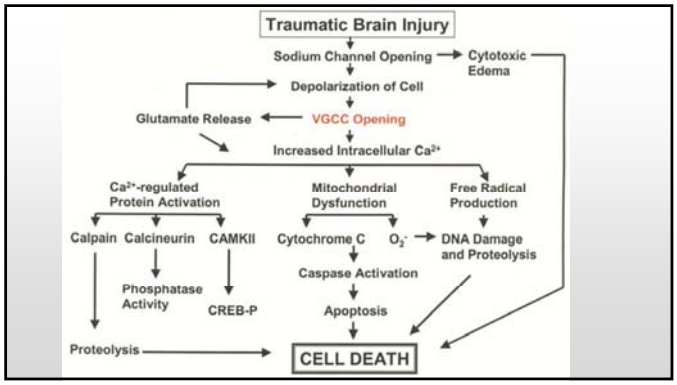

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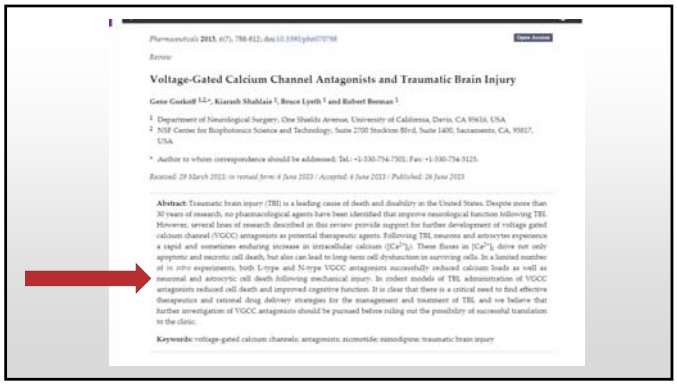

Voltage-Gated Calcium Channel Antagonists and Traumatic Brain Injury

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Abstract: Traumatic brain injury (TBI) is a leading cause of death and disability in the United States. Despite more than 30 years of research, no pharmacological agents have been identified that improve neurological function following TBI. However, several lines of research described in this review provide support for further development of voltage gated calcium channel (VGCC) antagonists as potential therapeutic agents. Following TBI, neurons and astrocytes experience a rapid and sometimes enduring increase in intracellular calcium ($[Ca^{2+}]_i$). These fluxes in $[Ca^{2+}]_i$ drive not only apoptotic and necrotic cell death, but also can lead to long-term cell dysfunction in surviving cells. In a limited number of *in vitro* experiments, both L-type and N-type VGCC antagonists successfully reduced calcium loads as well as neuronal and astrocytic cell death following mechanical injury. In rodent models of TBI, administration of VGCC antagonists reduced cell death and improved cognitive function. It is clear that there is a critical need to find effective therapeutics and rational drug delivery strategies for the management and treatment of TBI, and we believe that further investigation of VGCC antagonists should be pursued before ruling out the possibility of successful translation to the clinic.

Keywords: voltage-gated calcium channels; antagonists; ziconotide; nimodipine; traumatic brain injury

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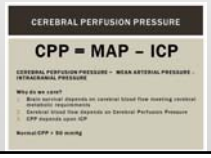
Management of ICP in the Trauma Patient

- Treatment required for ICP >20-25 mmHg
- Aggressive CPP improves outcome
- Keep CPP at least > 50mmHg (some say higher)
- Goal in treating intracranial hypertension is to promote adequate oxygenation and nutrient supply by maintaining CPP, oxygenation and glucose supply w/o hyperglycemia
  - Therapeutic options
    - Decrease cerebral blood volume
    - Decrease CSF volume ***
    - Diuretics *** 3% NS or Mannitol
    - Craniectomy ***
    - Resection of injured tissue ***
    - Evacuate hematomas



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Key components of ANESTHESIA CARE – Neurosurgery for Traumatic Brain Injury

- Hyperventilation
  - Normocarbia preferred
  - Hyperventilation = ↓ $PaCO_2$ = cerebral vasoconstriction
    - Cerebral ischemia in an already injured brain
      - <30mmHg

** Brief periods ok – will lower ICP in rapidly deteriorating situation with expanding hematoma until emergent craniotomy

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Mannitol and Hypertonic Saline

- Mannitol
  - Potent Osmotic Diuretic
  - Do not give with hypotension
  - Will NOT lower ICP in hypovolemia
    - Will worsen cerebral ischemia
- Indication for administration in euvolemic - 1 g/kg over 5 min then immediately to CT and/or OR
  - Acute neurologic deterioration
  - Dilated pupils
  - Hemiparesis
  - Loss of consciousness while observed
- Hypertonic Saline
  - Reduces high ICP
  - 3% - 23.4% used
  - Preferable if hypotension
  - Does not act as a diuretic

No difference between mannitol and hypertonic saline in lower ICP, neither will lower ICP in hypovolemic state

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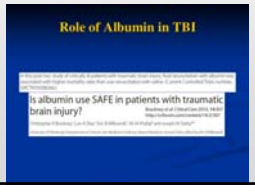
Understanding osmotic therapy

Patients with increased intracranial pressure (ICP) commonly receive bolus or continuous infusions of I.V. mannitol or hypertonic saline solution for osmotic therapy. Mannitol promotes osmotic diuresis, whereas hypertonic saline solution is more likely to maintain plasma volume. The goal of giving hypertonic saline solution is to raise the serum sodium level to 150 or 155 mEq/L, which increases serum osmolality and in turn reduces water in the brain.



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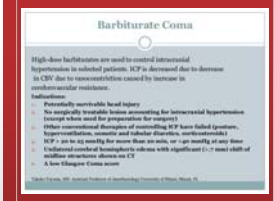
- Used in reducing ICP when refractory to other measures
- DO NOT use if hypotensive or hypovolemic
- NOT indicated in the acute resuscitation phase
- WILL prolong time to brain death determination due to long half life
- A consideration with devastating and likely non-survivable injury



Barbiturates

In acute resuscitation?

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- May inhibit brain recovery, only use when absolutely necessary
- Phenytoin and Fosphenytoin for acute phase
- Loading dose 1 gm, 50 mg/min
- Maintenance 100 mg/8 hours
- May control with GA
- PROLONGED? causes secondary brain injury

Anticonvulsants

Post traumatic epilepsy occurs in 5% w closed head injur and 15% with severe head injury

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- AND
  - Seizures are NOT controlled with NDMR
  - Prolonged seizures with NDMR on board are devastating to brain function
  - Undiagnosed and untreated if tonic clonic contractions are masked

Management

- At the site of accident
- Emergency department
- Radiological examination
- Surgical intervention
- ICU management

PITFALLS

Mannitol can have significant rebound effect

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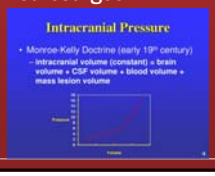
- Scalp wounds
- Depressed skull fractures
- Intracranial mass lesions
- Penetrating brain injury

All patients should be treated aggressively pending consultation with the neurosurgeon!



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Questions?

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