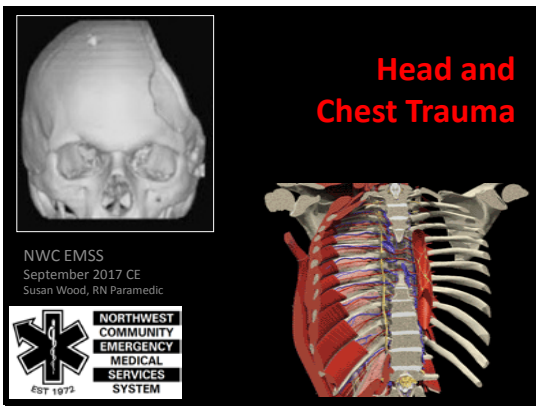
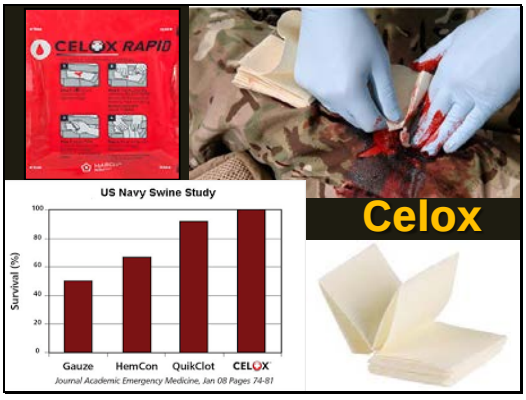
Slide 1



Slide 2

Transition before we get into our monthly CE, to discuss the plan with our hemostatic dressing change on the supply list. Of course, by attrition we will be transitioning from Quik Clot to the Celox Z fold gauze.

Slide 3

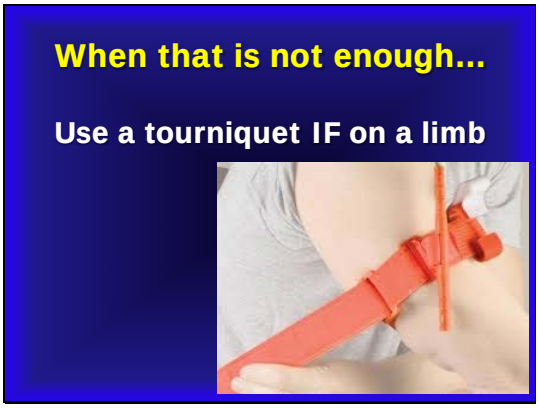


See handout for information

Slide 4

Wound Packing Essentials for EMTs and Paramedics
Sat, Apr 1, 2017
By Peter P. Taillac, MD, FACEP , Scotty Bolleter, BS, EMT-P [P] ,
A.J. Heightman, MPA, EMT P [Editor in Chief, JEMS]
(Article in handout)

Slide 5



Slide 6

We are ready with the main attraction

Slide 7

An opening slide to start the conversation about head injury
Actress Natasha Richardson succumbed to a fatal brain injury in 2009. Two days earlier, she had a fall in a skiing accident. She had not been wearing a safety helmet. The accident occurred while taking a skiing lesson on a beginners' slope. She had apparently "tumbled" down a hill, but initially seemed to be unhurt.
Tracy Morgan was in a limo bus when it was rear-ended. The crash killed comedy writer James McNair. Morgan suffered a broken leg, nose and ribs in the accident. He was also in a coma for two weeks afterward and has no memory of what happened.
Mike Tyson is a former professional boxer who competed from 1985 to 2005 as a world heavyweight champion and holds the record as the youngest boxer to win a heavyweight title at 20 yo.
In 1988, Busey was severely injured in a motorcycle accident in which he was not wearing a helmet. His skull was fractured, and doctors feared he suffered permanent brain damage. During the filming of the second season of *Celebrity Rehab* in 2008, Busey was referred to psychiatrist Dr. Charles Sophy. Sophy suspected that Busey's brain injury has had a greater effect on him than realized. He described it as essentially weakening his mental "filters" and causing him to speak and act impulsively. Makes sense…

Slide 8

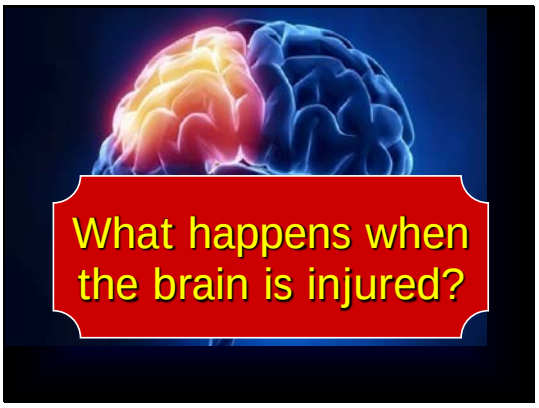
Slide 9

Children
(review: last month)

Slide 10

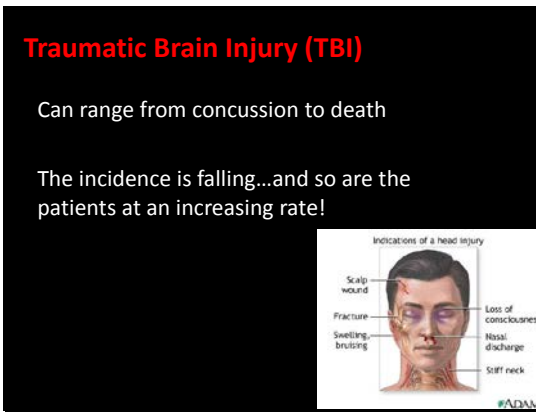
ONLY here for interest: (DO NOT DISCUSS IN CLASS) Penetrating brain injury after a suicide attempt with a speargun!~Penetrating cranial injury by mechanisms other than gunshots are exceedingly rare, and so strategies and guidelines for the management of PBI are largely informed by data from higher-velocity penetrating injuries. Here, we present a case of penetrating brain injury by the low-velocity mechanism of a harpoon from an underwater fishing speargun in an attempted suicide by a 56-year-old Caucasian male. The case raised a number of interesting points in management of low-velocity penetrating brain injury (LVPBI), including benefit in delaying foreign body removal to allow for tamponade; the importance of history-taking in establishing the social/legal significance of the events surrounding the injury; the use of cerebral angiogram in all cases of PBI; advantages of using dual-energy CT to reduce artifact when available; and antibiotic prophylaxis in the context of idiosyncratic histories of usage of penetrating objects before coming in contact with the intracranial environment. We present here the management of the case in full along with an extended discussion and review of existing literature regarding key points in management of LVPBI vs. higher-velocity forms of intracranial injury Penetrating Brain Injury after Suicide Attempt with Speargun: Case Study and Review of Literature (PDF Download Available). Available from: https://www.researchgate.net/publication/264393777_Penetrating_Brain_Injury_after_Suicide_Attempt_with_Speargun_Case_Study_and_Review_of_Literature [accessed Aug 23, 2017].

Slide 11



The goal is to take a moment here and ask questions of the group. We did discuss head injury last month in peds, therefore this is a check for knowledge retention from last month. This should allow group participation in answering the question. Feel free to elaborate on any point you find is in need of clarification for the group.

Slide 12

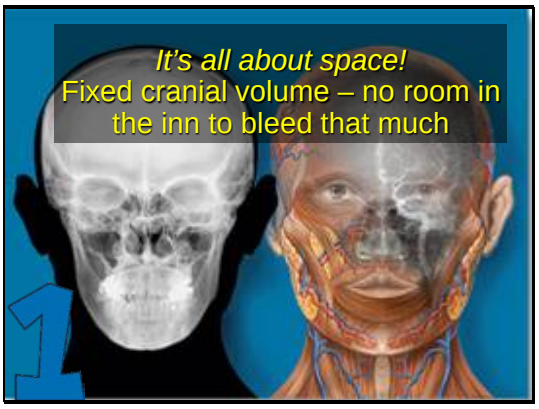


Progress has been made to prevent motor-vehicle crashes, resulting in a decrease in the number of TBI-related hospitalizations and deaths from 2007 to 2013. However, during the same time, the number and rate of older adult fall-related TBIs have increased substantially. Although considerable public interest has focused on sports-related concussion in youth, the findings in this report suggest that TBIs attributable to older adult falls, many of which result in hospitalization and death, should receive public health attention.

Slide 13

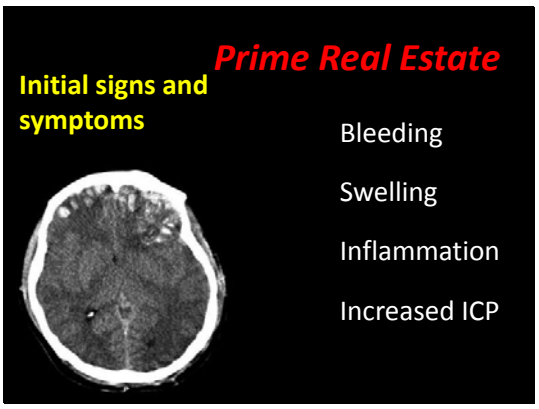
To give credit where credit is due, this is the article that the majority of work is based upon with permission of course!

Slide 14



Peds vs. adults found in handout
80% brain
10% CSF
10% blood volume

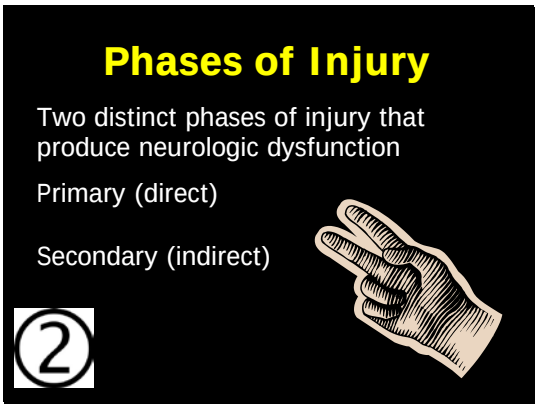
Slide 15



Traumatic brain injuries (TBI) can cause inflammation and swelling or bleeding from damage to brain tissue and blood vessels. Bleeding may occur within the brain(an intracranial hemorrhage) or the protective layers that surround it, such as an epidural, subdural or subarachnoid hemorrhage.
The brain is tightly enclosed within the skull. Bleeding and inflammation cause a rise in intracranial pressure (ICP), which squeezes and damages brain structures, within that space. As ICP increases, sections of the brain may shift to different areas within the skull, or into the opening where the spinal cord enters the cranium, through a process known as herniation. The higher and longer ICP rises, the degree of permanent disability and likelihood of death increases.
Signs of increased ICP include altered mental status, unequal or nonreactive pupils, posturing, or seizures, as well as bradycardia and hypertension (Cushing's Phenomenon).

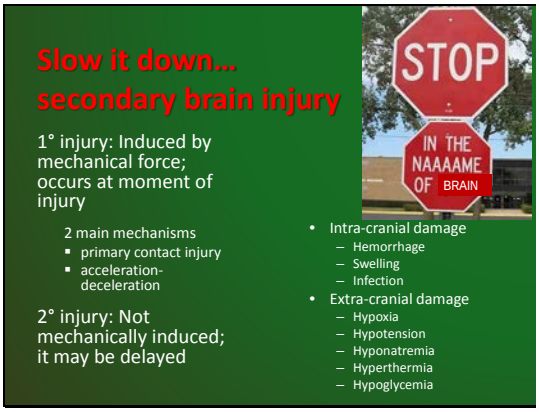
Slide 16

Slide 17



Primary=what we see
Secondary=what will cause problems for life

Slide 18



Brain tissue does not regenerate, thus damage from the initial insult is generally permanent. The subsequent edema and bleeding from the primary injury then spreads to damage other areas of the brain, and treatment is aimed at limiting this secondary injury. Through airway protection, oxygenation or ventilation, blood pressure maintenance, glucose and transport to a trauma center, EMS plays a vital role in stopping this cascade.

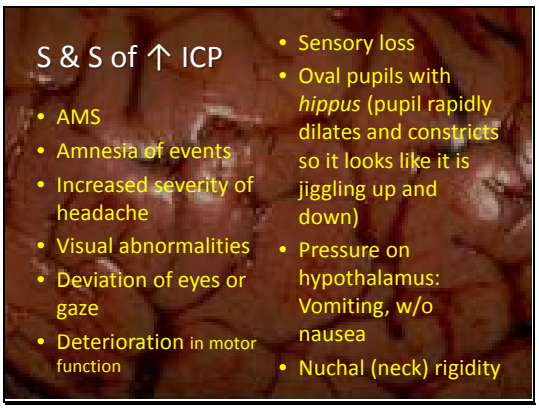
Slide 19

Patients with TBI may be confused, combative, or unresponsive. They may be unable able to follow commands or report pain, and concurrent drug or alcohol intoxication makes assessment even more difficult. Don't assume that altered mentation is caused by just one thing, a head injury instead, intoxication or glucose.
Rely on inspection, auscultation, and palpation to rule in other injuries, such as a change in the patient's facial expression when their abdomen is palpated. Remember that internal injuries cannot be ruled out, particularly those involving the spinal cord, so take spinal precautions in any patient with a head injury and altered mental status.

Slide 20

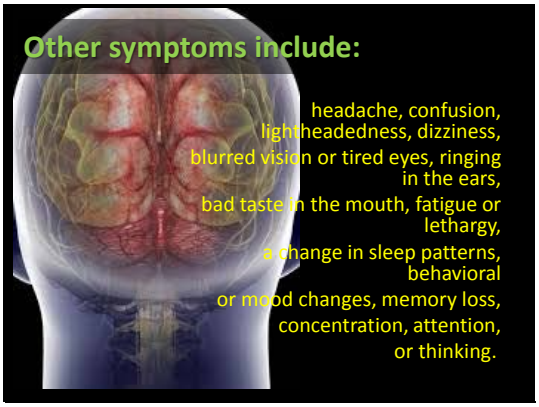
SOP Review

Slide 21



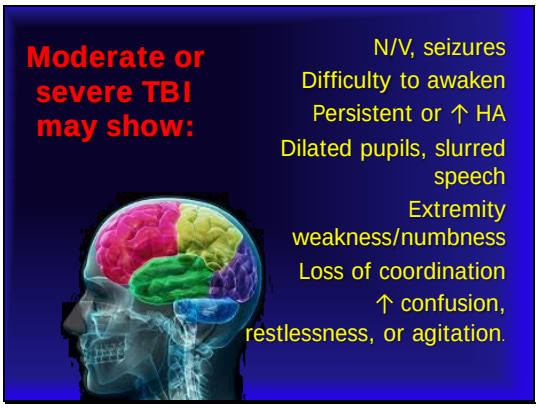
AMS; progressive restlessness, confusion, disorientation, and lethargy or combativeness; changes in speech or loss of judgment
Amnesia of events before or after the injury
Increased severity of headaches
Visual abnormalities; visual fields deficit (lose sight in part of field)
Conjugate deviation of eyes or gaze palsies
Deterioration in motor function: Monoplegia, hemiplegia: first part of body to show an increase in ICP is the wrist, will over pronate or supinate; pronator drift
Sensory loss
Oval pupils with hippus (pupil rapidly dilates and constricts so it looks like it is jiggling up and down)
Pressure on hypothalamus: Vomiting, often without nausea; temp changes
Nuchal (neck) rigidity

Slide 22



These are some subtle symptoms that strangely patients may complain of but can be overlooked… Instructor to ask the audience if there is an symptoms that their patients have oddly complained about that in hindsight was a result of a HI.

Slide 23



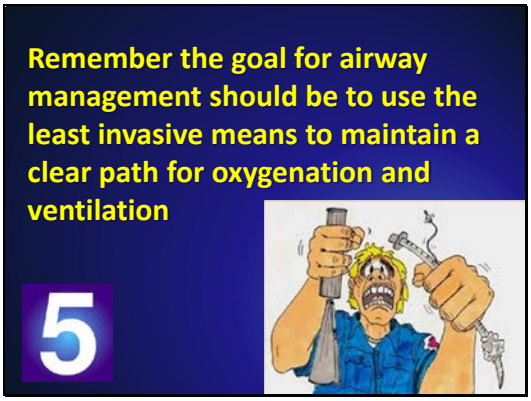
Last month the discussion included if patients (with parents) wanted to refuse care, what would be some of the discussion points to have with the family that should be relayed and documented…these are some of the symptoms.

Slide 24

Treatment for head injured patients requires rapid assessment, imaging with a CT scan, and possibly neurosurgery. Time is brain, and the chance of disability or death increases with any delay.

Transporting patients to trauma centers that offer neurological services, and early activation of teams at these centers, streamlines the process to definitive care. Know which hospitals these are, initiate transport as quickly as possible, and alert the hospital to prepare staff and equipment for your arrival.

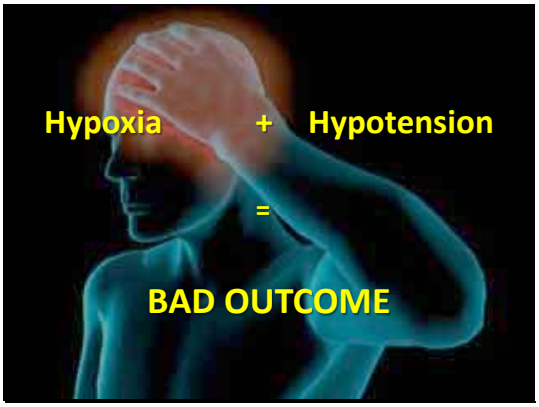
Slide 25



Unconscious head injured patients may lose muscle tone in their jaw, and their tongue may obstruct the airway. The gag reflex may also be compromised in TBI patients, which increases the risk of aspiration from vomit or blood. Clenched teeth, known as trismus, is another common finding that makes position and suctioning airway secretions difficult. Attempt to open the airway with a jaw thrust. Use an oral or nasal airway if the patient will tolerate it, position the patient on their left side to help prevent aspiration, and suction secretions.

Remember that the goal for airway management should be to use the least invasive means to maintain a clear path for oxygenation and ventilation.

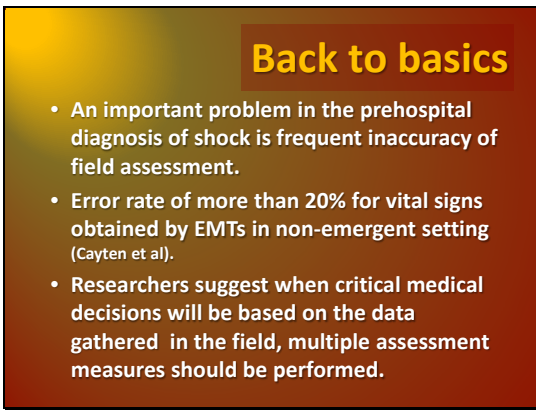
Slide 26



Irreversible brain damage can occur in TBI patients after only four minutes of anoxia, which can be caused by a compromised airway, altered respiratory patterns from the head injury, or lung injury in multi-system trauma. Use pulse-oximetry, end-tidal, skin color, and respiratory rate to assess adequate oxygenation. Administer oxygen via nasal cannula, non-rebreather mask, or bag valve to maintain a pulse-ox reading of at least 94%.

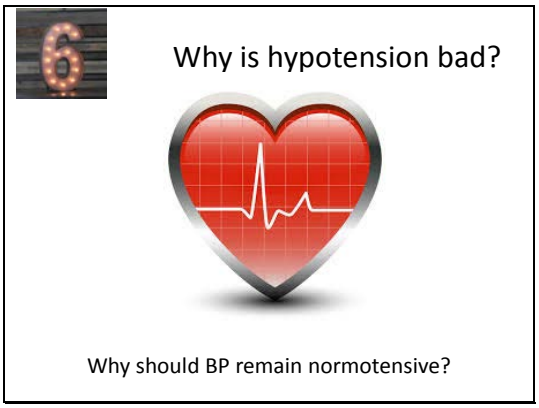
Cerebral oxygen delivery is also compromised by hypotension, which is associated with poor outcomes after even transient episodes. Hypotension caused directly by a head injury is a rare and ominous finding; it is more often caused by shock associated with other injuries. Administer IV fluids and titrate blood pressure to 110 – 120 mmHG systolic in head injured patients who are hypotensive.

Slide 27



Roth et. Al., Time/Life-Critical Events, section A., Hypotension and shock retrieved from: http://emergencymedicine.pitt.edu/sites/default/files/1_A_07_051-062_unlocked.pdf#page=8&zoom=auto,-133,269

Slide 28

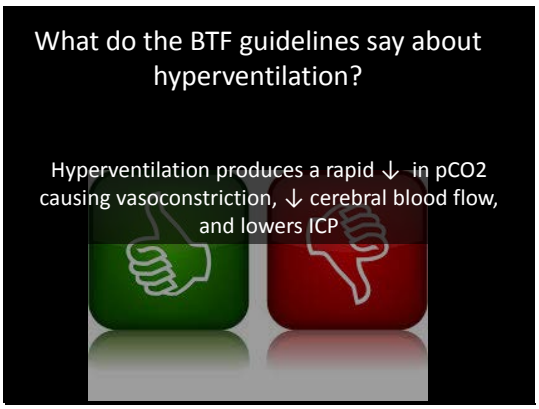


Found in secondary assessment, must fix way before that!

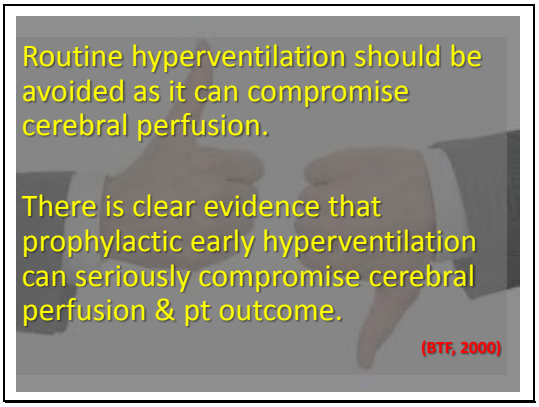
Slide 29

As harmful as hypoxia is in TBI, hypocapnea from excessive ventilation is harmful as well. A higher respiratory rate decreases the amount of CO2 in the blood, which triggers cerebral vasoconstriction and less oxygen delivery. Hyperventilation of head injuries has been associated with poor outcomes. When assisted ventilation is necessary in isolated head injuries, follow your local protocols regarding use of end-tidal CO2 (EtCO2) monitoring for a head-injured patient.
Shock from multisystem trauma can also cause a low EtCO2 reading, in which case it should not be used to guide ventilation. If capnography is not available, or if the patient may be in shock from other injuries, ventilate adults at 10 breaths per minute. There is one exception...

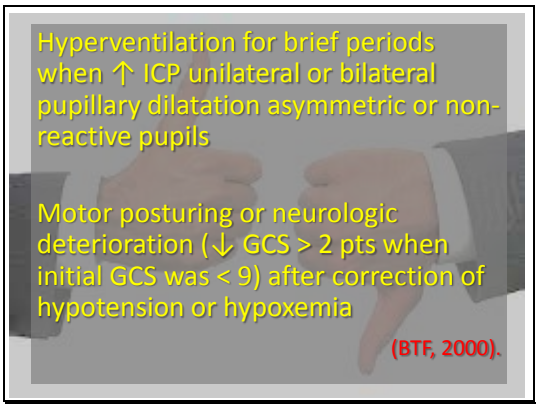
Slide 30



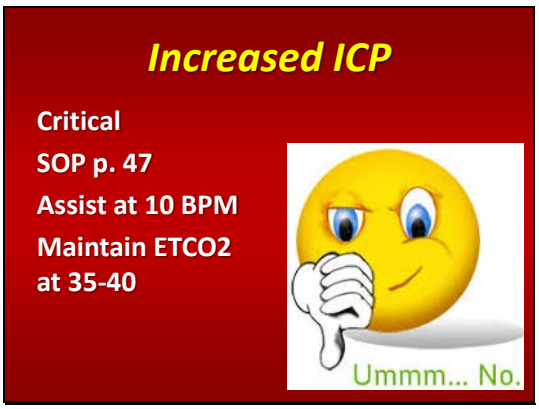
Slide 31



Slide 32



Slide 33

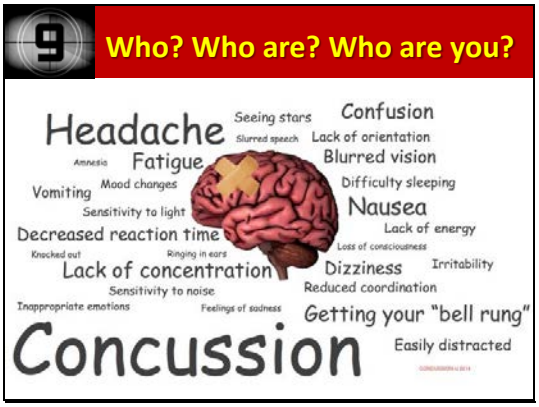


Slide 34

Patients taking blood thinners, including Coumadin and Plavix, are at risk of severe bleeding into the brain from relatively minor head injuries. Learn what medications these are, and factor the patient's medication list into your assessment. Signs and symptoms of intracranial bleeding may be delayed, especially in elderly people. SOP p. 36 same as stroke. Also note that chronic alcoholics are "thin."

Assume that an altered mental status is a change from baseline unless proven otherwise, and perform a brief neuro exam to detect subtle signs of closed head injury. Any patient taking a blood thinner who bumps their head should be evaluated at the hospital, no matter how minor their injury appears.

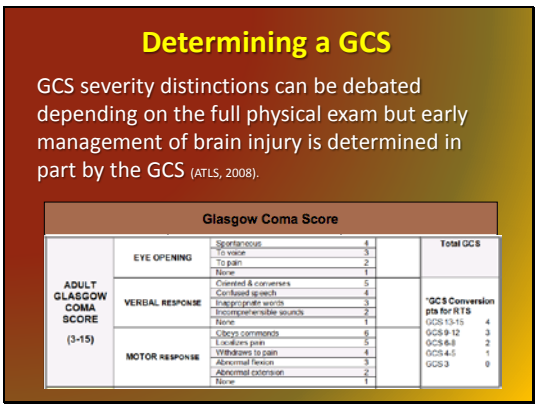
Slide 35



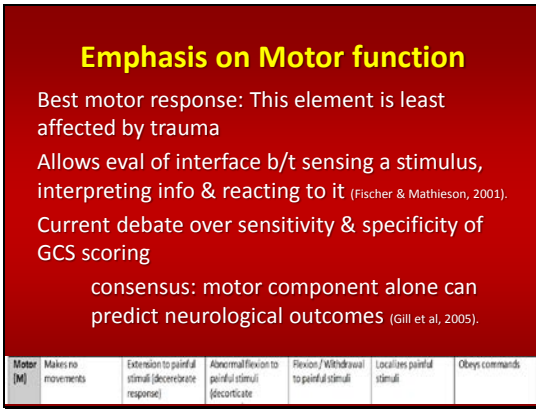
A concussion is a transient change in mentation after blunt head trauma. It can cause retrograde amnesia, and patients often repeatedly ask the same question after being given an answer. It can also cause a temporary loss of consciousness, disorientation, incoherent speech, or lack of coordination. Diagnosis of a concussion is made after a CT scan shows no structural abnormalities associated with these symptoms - not in the field.

While concussion symptoms typically resolve after a few hours or days, severe or multiple concussions can cause headaches, dizziness, depression, and difficulty concentrating for weeks. Especially worrisome is a secondary impact syndrome, where a patient sustains a second concussion before recovering from the first. This can cause rapid cerebral edema and herniation, and has a high mortality rate. Athletes with a head injury should not return to play until after symptoms have completely resolved.

Slide 36



Slide 37

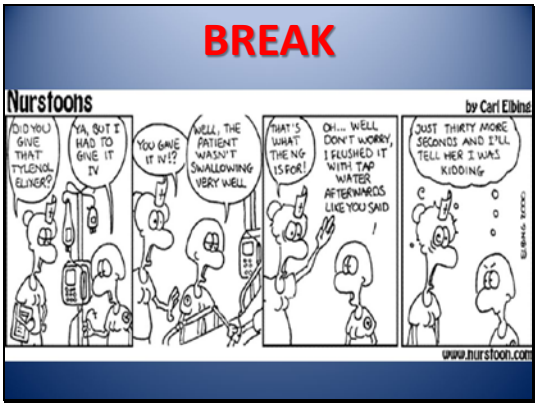


Slide 38

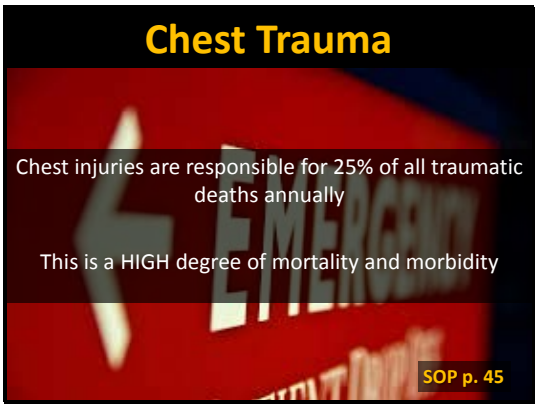
History can help understand if there was any preventative; check with what PD may have done PTA.

Slide 39

Slide 40

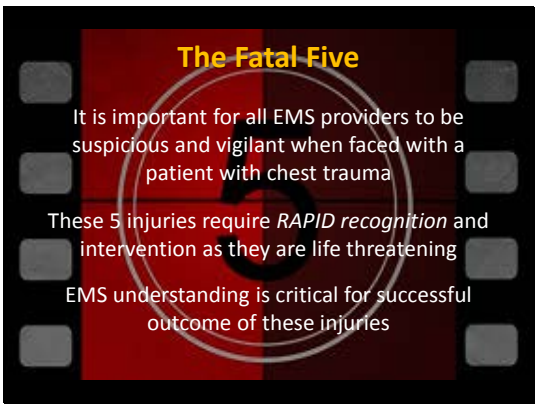


Slide 41

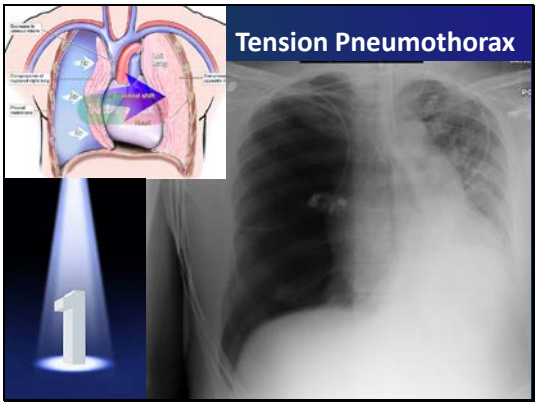


Why do you think that is? We can miss them. I want to impress that pneumothorax is not a diagnosis made after a CXR, it is made upon physical assessment.

Slide 42

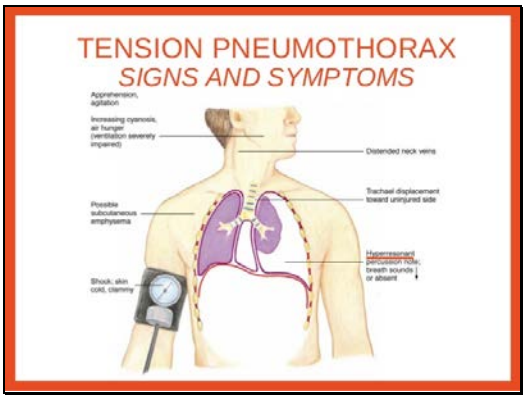


Slide 43



A tension pneumothorax can be formed from either blunt or penetrating trauma. A hole in the lung and/or a hole in the chest wall allows air to escape into the intrapleural space. If this air cannot escape to the external environment, pressure will begin to accumulate. Ventilation is compromised as the pressure builds up, causing the collapse of the ipsilateral lung and mediastinal shift. Progressively increasing pressure causes the contralateral lung to receive compression, further exacerbating the condition. Â This tension also causes circulatory compromise. The pressure compresses the vena cava, reducing venous return to the right heart. As venous return decreases, cardiac output falls. Understanding that a TPx will cause both pulmonary and cardiac collapse will give you the appropriate appreciation for its lethality.

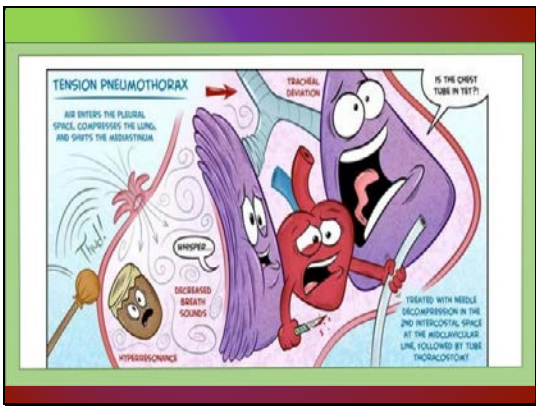
Slide 44



Assessment:

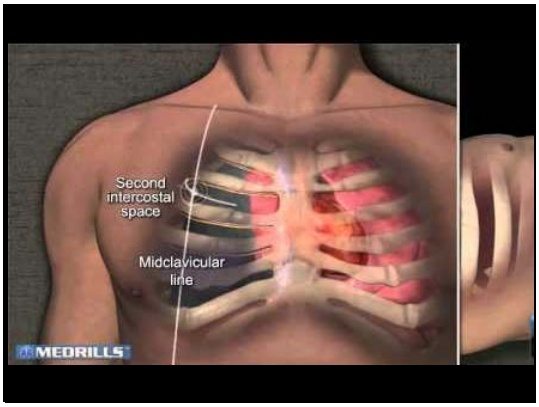
Mechanism of injury should guide your index of suspicion. The presence of blunt or penetrating trauma to the chest and a patient presenting with respiratory distress form the basis for inclusion of TPx in your differential diagnosis. In fact in Tactical Combat Casualty Care (military and TEMS) guidelines, progressive respiratory distress and known or suspected torso trauma is enough to initiate a needle thoracostomy of the affected side. Furthering your assessment…patients with massive tension build up will present with unequal chest excursion and decreased or absent breath sounds over the injured hemi-thorax. SQ emphysema may be palpated in the chest. JVD may be realized as the pressure causes central venous pressure to build. *Tracheal deviation may occur, however this sign is much debated lately as recent research suggests this is a highly unusual occurrence. If you look at the slide you will see that the deviation is truly below the area of the clavicles and cannot be visualized well.*

Slide 45

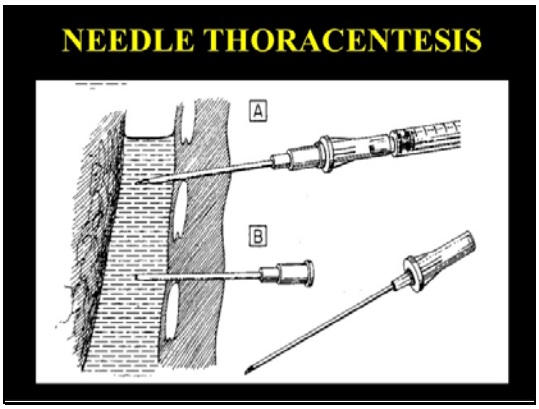


The question always becomes, when is intervention needed? We check blood pressure, without perfusion they will die.

Slide 46



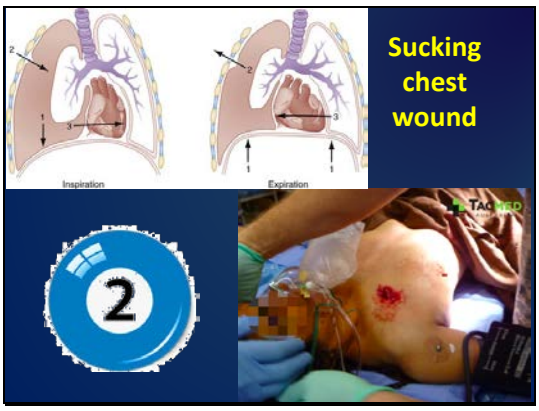
Slide 47



Intervention:

Rapid decompression of the built up pressure is the prescribed life-saving intervention. Needle decompression utilizing a large bore IV, placed into the 2nd intercostal space, midclavicular line will release the intrapleural pressure. A rush of air may be heard on insertion of the needle. But in most field environments this is not easy to hear. A better benchmark of successful decompression will be the clinical improvement of your patient, reduced dyspnea and improved hemodynamics. A chest tube should be placed as soon as feasible. Administration of fluid resuscitation for any existing hypoperfusion should be initiated. Placement of an occlusive dressing over any open chest wound is also indicated.

Slide 48



Etiology:

An open pneumothorax is caused by penetrating injury to the chest wall. Air enters the pleural space through the disturbance. If the size of the wound is greater than the diameter of the patient's trachea, air moves through the chest wound rather than the trachea. This defect results in an equalization of pleural and atmospheric pressure. This results in the loss of negative pressure inside the thoracic cavity, resulting in ventilatory failure. Additionally, pressure can continuing building inside the pleural space, creating a TPx.

Slide 49

MUST EXPOSE
Assessment:

Once again, MOI guides recognition of this injury. Presence of an open chest wound should raise the index of suspicion. These patients will present with respiratory distress. As air enters and escapes through the wound in the chest wall, a sucking noise may be heard…get it…sucking chest wound. If pressure is building up in the pleural cavity, S/S of a tension pneumothorax may be present.

Slide 50

Intervention:

In the field, placement of an occlusive dressing is indicated. Utilization of a commercially available occlusive dressing is recommended (Hyfin, HALO, Bolin, etc..). Most of these commercial chest seals have effective adhesive to stick to bloody skin. Additionally, some have a one-way valve that will allow air to escape the thoracic cavity, assisting in prevention of TPT development. If a commercially available dressing isn't available, use anything airtight (AED pad, vaseline gauze…or your gloved hand!) to seal the wound. In the past the creation of a field expedient three sided dressing was recommended. However years of lesson learned in combat has taught us this is a flawed methodology. Too much time is wasted creating a device that likely won't be effective. Cover the hole up…quickly. If your patient develops S/S of a TPT "burp" the dressing by lifting up a corner of the material, allow any pressure to escape. If that doesn't resolve the problem progress to needle decompression of the affected side

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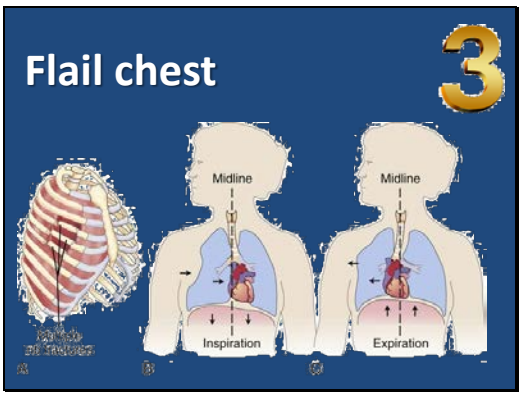

Etiology:

A flail chest typically is associated with blunt trauma. Multiple rib fractures cause a separation of the thoracic cage from the chest wall. This is typically defined by two fractures per rib, in at least two contiguous ribs. Large flail segments will cause a loss of normal ventilatory mechanics. Poor gas exchange, and painful ventilation will lead to respiratory failure. The presence of a pulmonary contusion also contributes to respiratory insufficiency.

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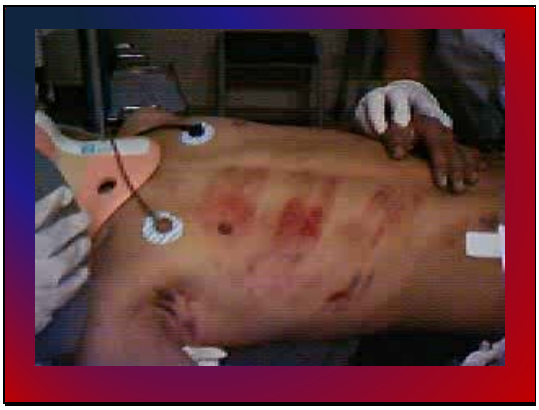

Assessment:

Bruising, discoloration, abrasions all should point to the presence of localized trauma to the torso. Chest wall motion should be observed. Instability of the flail segment will typically present with asymmetrical chest excursion. Paradoxical chest wall movement (one side goes up and one side goes down) may be tough to detect due to muscle spasms at the injury site. Palpation may be a better tool than visualization to detect this abnormal chest wall motion. Crepitus may be present on palpation of the chest. Awake patients will typically complain of pain on palpation and extremely painful breathing. (Have you ever broke a rib? Ouch…)

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Intervention:

In the presence of severe respiratory distress, BVM ventilation and/or intubation may be indicated to restore effective ventilation. Positive pressure ventilation will overcome the loss of the chest wall stability, effectively splinting the chest from the inside, however NOT if suspected Ptx.

Analgesia is important in treatment of a flail chest. Hypoventilation secondary to pain from broken ribs is a contributing factor in the respiratory insufficiency. By managing the pain involved in breathing, improvement in ventilation can be realized. The strategy is not without its problems. Opiate based narcotics can cause some pretty strong respiratory depression. Without careful observation and patient management these side effects can exacerbate the respiratory failure. Monitoring your patient for development of a PTx is also critical. Broken ribs have a tendency to puncture lungs.

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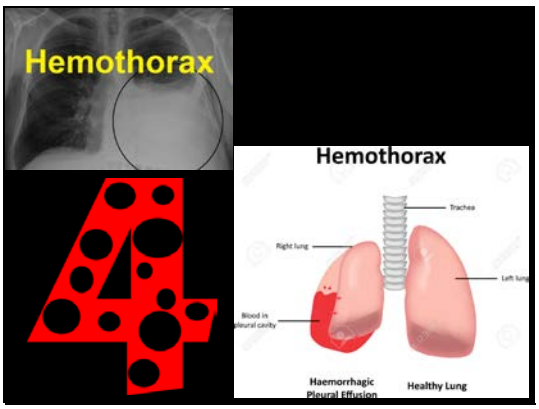

Etiology:

Massive hemothorax can be caused by blunt or penetrating trauma. Blood builds up in the pleural space due to injury to intrathoracic organs and blood vessels. The progression can be slow as with venous bleeding, or rapid in the case of arterial injury. A hemothorax is considered “massive” with the accumulation of 1500 mL of blood. As the bleeding continues, the compliant nature of the lung tissue allows a large amount of blood to become sequestered in the pleural space. Compression of the ipsilateral lung occurs effecting ventilation. Pressure can continue to build, causing mediastinal shift and the development of a tension hemothorax.

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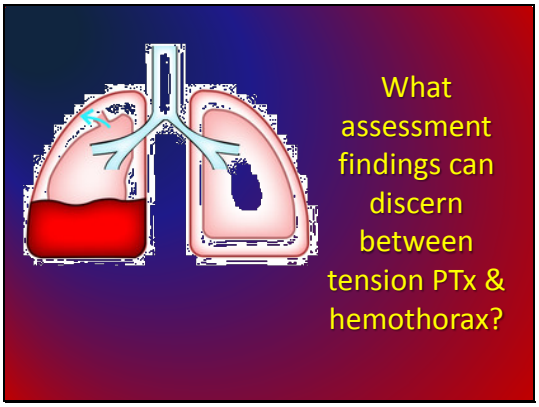

Assessment:

Clinical presentation can include dyspnea, chest pain, decreased or absent breath sounds on the affected side, dullness of percussion over the injured hemi-thorax. Signs of shock will typically become pronounced as blood loss continues. Differentiation between tension pneumo and hemothorax can be difficult in the field. JVD will not be present with a massive hemothorax due to the loss of circulating volume. Additionally, percussive differences exist. With a TPx the chest will typically be hyper resonant, while a hemothorax will typically present with dullness on percussion. Shock symptoms will present early and progress more rapidly with a hemothorax.

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Treatment:

Treatment is focused on restoration of lost blood volume and rapid transport to the level one trauma center. Much like any other hypovolemic state, large bore IV access is a must. Fluid resuscitation of any type carries the risk of worsening the hemothorax. So rapid and safe transport to the trauma center is indicated to surgically correct the injury.

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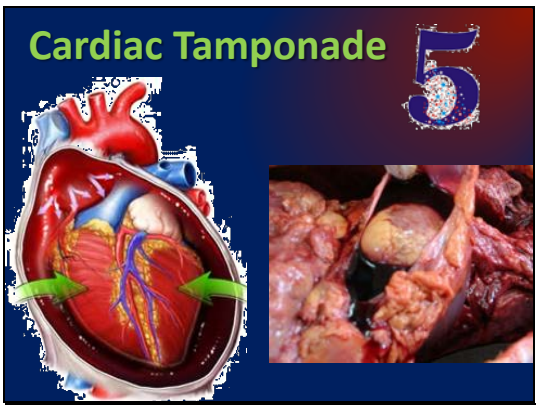

Etiology:

An acute cardiac tamponade occurs when blood accumulates in the pericardial sac. This can be a result of penetrating or blunt trauma. The effects of this tamponade are directly related to how quickly the fluid accumulates. Rapid accumulation can be fatal. If the bleeding is slow, however, the pericardium is able to stretch and accommodate much more fluid without hemodynamic consequences. Regardless, diastolic filling will eventually be compromised due to the increased pericardial pressure. Cardiac output will fall precipitously as this pressure increases.

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Assessment:

Reduced cardiac output is the primary clinical presentation of a patient with an acute cardiac tamponade. S/S of shock will progress as diastolic filling is reduced. In addition to shock, Beck's Triad will be observed (Narrowed pulse pressure, JVD, muffled heart tones). Pulse pressure will narrow due to a falling systolic B/P. Increased central venous pressure will cause JVD. Muffled heart tones will be present due to the fluid surrounding the heart. You may also notice pulsus paradoxus. This is a drop in systolic B/P greater than 15 mmHg during inspiration. Often when presented with a patient with thoracic trauma, who fails to respond to other treatment, you're dealing with a cardiac tamponade.

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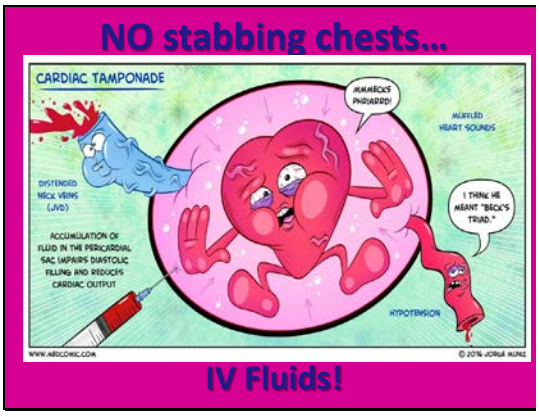

Intervention:

Field intervention of a suspected cardiac tamponade is a rapid bolus of IV fluids. This is a temporizing measure aimed at increasing preload and diastolic filling pressure, thus improving cardiac output. Pericardiocentesis is the emergent treatment of choice for a tamponade. However, this is no longer performed in the field.

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Permissive hypotension with NS WO just to achieve SBP 80 with a MAP 50-60)
Monitor for PEA…treat according to traumatic arrest protocol if needed

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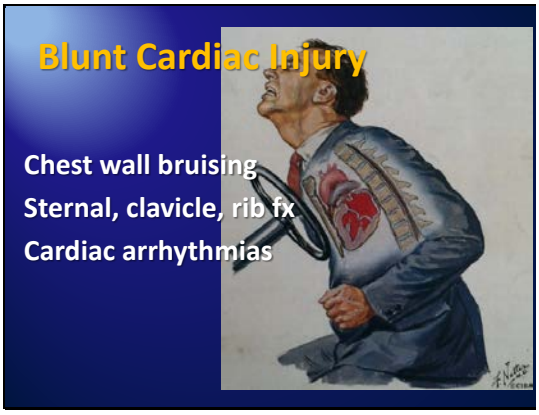

Treat what you see and maintain SBP 90 (MAP 65)

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