Let's Trauma Bond!
Resuscitation and Management of Traumatic Injuries in Small Animals

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Outline

- Common traumatic injuries
  - Hemorrhagic shock
  - Thoracic injuries – flail chest, pneumothorax, pulmonary contusions, pericardial effusion
  - Abdominal injuries – septic abdomen, hemoabdomen, uroabdomen
  - Traumatic brain injury
  - Proptosis
  - Skin wound care
- Practical triage plan for each injury

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Definitions

- Trauma = any tissue injury that occurs more or less suddenly as a result of an external force
- Classified based on injury
  - Penetrating
  - Blunt
- Minor vs. life-threatening

Hemorrhagic Shock

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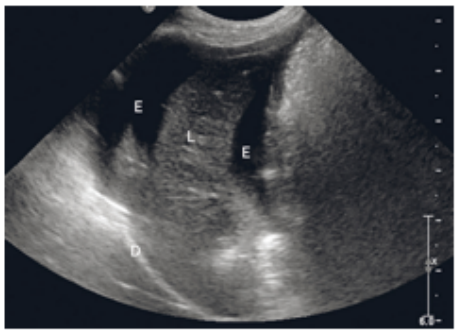
Hemorrhagic Shock

- Trauma cases can experience severe tissue injury and hemorrhage
- Therefore, most cases have some degree of hypovolemic shock (subclass traumatic hemorrhagic)



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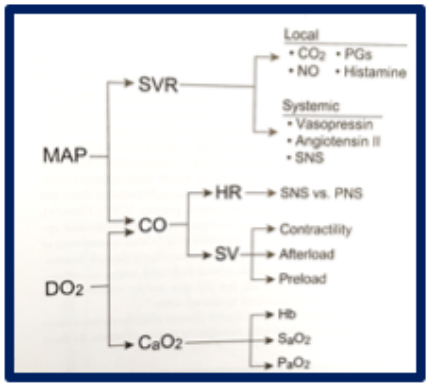
- In our severe trauma cases, patients can experience significant tissue injury leading to severe hemorrhage
- This hemorrhage can progress to shock very quickly
    - For instance, 27-43% of our blunt trauma dogs will have a hemoabdomen – which we know can result in losing liters of blood
- With hemorrhage most will present in state of hypovolemic shock
- Within the hypovolemic shock category, remember there are further subclassifications
    - Hemorrhagic = acute drop in red cells causing tissue hypoxia
        - GI bleeding, coagulopathies (not from trauma), hemoabdomen
    - Traumatic hemorrhagic =
        - This differs from just hemorrhagic bc you have the inflammatory response from soft tissue injury
    - Traumatic hypovolemic (nonhemorrhagic) = large surface burns
    - Hypovolemic without hemorrhage = v/d, renal losses, large volume fluid sequestration
    - https://todaysveterinarypractice.com/radiology-imaging/sonography-assessment-overview-of-afast-and-tfast/

Hemorrhagic Shock

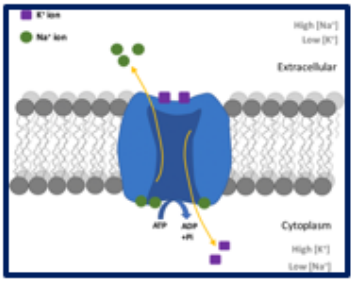
- Hemorrhage → decreases effective circulating blood volume
  - → decreased venous return (preload)
    - → decreased cardiac output
      - → decreased organ perfusion pressure
        - → decreased O2 to tissues



- Its very important for us to understand the pathophys of hemorrhagic shock in these patients because in human medicine, hemorrhage leading to exsanguination is a major cause of preventable deaths
- Bleeding leads to decreased circulating volume, leading to decreased venous return (lower preload for the heart)
- This leads to decreased cardiac output and perfusion
- Meaning decreased O2 delivery to tissues

Hemorrhagic Shock

- Aerobic metabolism → anaerobic metabolism
  - No oxidative phosphorylation
  - Inadequate energy (ATP) production
- Failure of membrane transport pumps
  - NaK ATPase
- Loss of electrical gradient → influx of Na, Ca, and water into cell → cell lysis and necrosis
- Release of inflammatory mediators → trigger systemic inflammatory response



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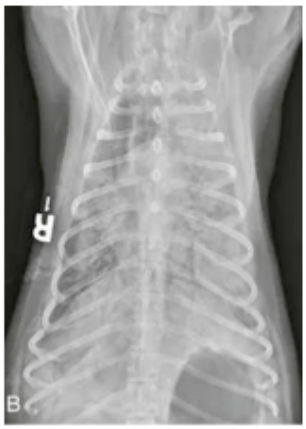
- Then in hypovolemic shock, as O2 delivery decreases, the body tries to compensate by extracting more O2 than normal from the blood
- When eventually the ratio decreases beyond critical point, usually when 50% of blood volume is lost, no longer able to compensate with extraction ratio changes
  - Remember blood volume in cats is 55-65 mL/kg and dogs is 80-90 mL/kg
- Without adequate delivery of O2 ultimately cells can't make energy and you can't have oxidative phosphorylation
  - so switch to anaerobic metabolism, which results in inadequate production of ATP
  - The Inadequate energy production that occurs in shock has several sequalae
  - First you get dysfunction of Na-K ATPase pump
  - This leads to loss of the electrical gradient and an influx of Na, Ca, and water into the cell
  - Ultimately you get cell lysis and necrosis, so cell death
  - With cell death you get release of inflammatory mediators (DAMPS) that trigger innate immune system and you get systemic inflammatory response

Hemorrhagic Shock

- Systemic complications
  - Acute lung injury (ALI) and Acute Respiratory Distress Syndrome (ARDS)
  - Acute renal tubular necrosis
  - Loss of GI mucosal barrier
  - SNS activity → Dysrhythmias
  - Reperfusion injury



- And then this inflammatory response can lead to multiple systemic complications
  - ALI and ARDs
  - Acute renal tubular necrosis
  - Loss of GI mucosal barrier → bacteremia
  - Increased sympathetic nervous system activity leading to dysrhythmias
- And keep in mind reperfusion injury too as that can be a complication as we treat these patients
  - Electrolyte abnormalities, cerebral edema, arrhythmias, MODS
  - Lifethreatening

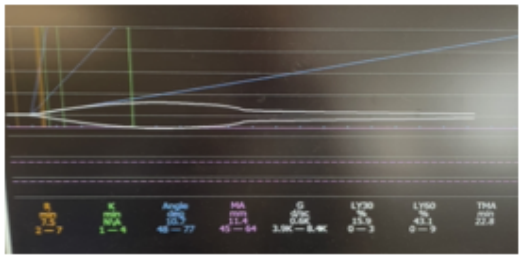
Hemorrhagic Shock

- Traumatic induced coagulopathy
  - Implicated as the cause of almost half of hemorrhagic deaths in trauma patients
    - 25-34% hypocoagulable on presentation
  - Prolongs bleeding, increases transfusion requirements, sustains hypoperfusion
  - Independent risk factor for death
  - Degree of coagulopathy positively correlated with injury severity and degree of shock

- In trauma cases, trauma induced coagulopathy can also contribute to the massive hemorrhage patients experience
  - Now we know that massive bleeding can cause a consumptive coagulopathy – essentially the body using all the platelet and coag factors to try to stop the hemorrhage
  - Trauma induced coagulopathy is NOT that, its its own separate pathophys
    - So you can have both going on!
- TIC has been determined to contribute to almost half of hemorrhagic deaths in human trauma patients
  - ¼ are determined to be hypocoagulable on presentation
  - It can prolong bleeding, require more transfusion products, and cause prolonged hypoperfusion and increase risk for complications from shock state
  - The severity of the coagulopathy does correlate with degree of injury and shock – so if you have a patient that presents in severe shock with extensive injury I think its fair to assume TIC even without coag testing

Hemorrhagic Shock

- Trauma induced coagulopathy
  - Occurs within minutes following injury, before any iatrogenic factors (fluids, etc)
  - Endogenous
  - Hyperfibrinolysis + hypocoagulable
  - 3 hypotheses for pathophysiology (see extra slides)



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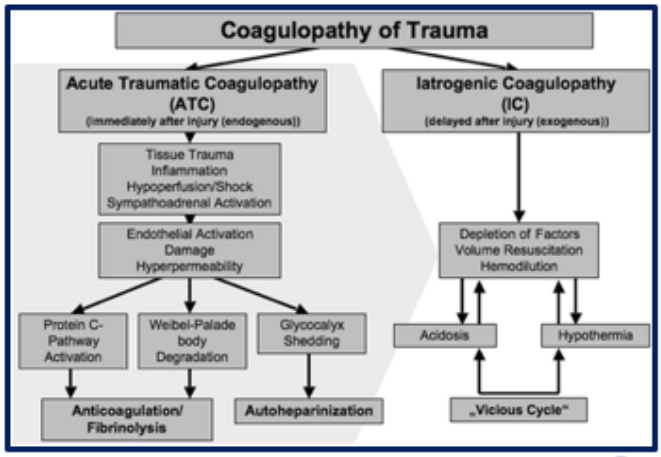
- TIC occurs within minutes of injury – so often before we get to evaluate a patient
- And important to note that it occurs even before we do anything
  - TIC is a distinct endogenous hypocoagble, hyperfibrinolytic state
    - Endogenous = occurs independently from hemodilution, acidosis, or hypothermia
      - So no contribution of a dilutional coagulopathy (when we give large volume fluid products and dilute out our plt and coag factors → essentially causing a coagulopathy)
- Currently there are 3 hypotheses for how this can occur so quickly – I added extra slides for your reference that will be included in slides sent to you but it's a bit deep to cover appropriately here

Hemorrhagic Shock

- Additional factors:
  - Tissue injury
  - Hypoperfusion
  - Inflammation
  - Metabolic acidosis
  - Hypothermia
  - Hemodilution

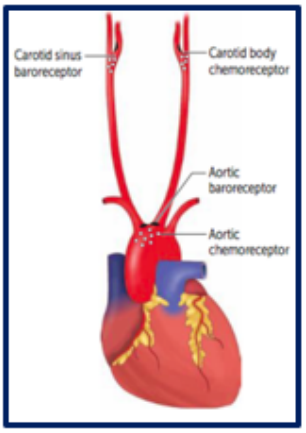


- Then there are other factors that contribute to TIC and can worsen it
- Tissue injury
  - Degree of injury related to severity of coagulopathy but only if hypoperfusion too
  - Brain, long bone, and penetrating injury may be more likely to develop ATC
  - Tissue injury exposes subendothelium collagen to vWF and exposes TF to circulating factor 7a → initiates coagulation and thrombin formation
  - Then TM binds to thrombin and activates protein C pathway via inhibition of plasminogen activator inhibitor-1 → promotes fibrinolysis
- Hypoperfusion
  - Exact mechanisms of hypoperfusion induced coagulopathy is unclear - it exerts its own effect separate of acidosis and hypothermia
  - Significant reduction in serine protease activity?
  - Increase tPa?
  - Hypoperfusion also delays clearance of thrombin so more time to bind to thrombomodulin and enhance protein C anticoagulation → hyperfibrinolysis
- Inflammation – interplay between the immune system and coagulation
  - Proinflammatory cytokines upregulate TF and Complement upregulates thrombomodulin expression → enhanced protein C → hypocoagulation and hyperfibirinolysis
  - Later, > 24 hrs, more proinflammatory cytokines downregulate protein S, heparin sulfate, thrombomodulin, → hypercoagulable due to decreased protein C and AT

- Degree of acidosis and hypothermia
    - The lethal triad causes coagulation factor and platelet dysfunction, increases fibrinogen consumption and decreased platelet count
    - The factor 10a/5a complex activity may be reduced by 50% at pH of 7.2
        - Interestingly, infusion of bicarb fails to correct hypocoagulability in these patients
    - Hypothermia decreases coagulation protease function and platelet aggregation
- Hemodilution
    - With massive blood loss, you get shift of extracellular water into vascular space (remember that intermediate phase of compensation and Starling law)
    - This leads to dilution of hemostatic factors, red cells, and platelets
    - Large volume fluid resuscitation also causes this, also reduces blood viscosity
- As you can see its quite complex and we need to be considering all these factors when assessing our patients on presentation

Hemorrhagic Shock

- Immediate compensation
  - Baroreceptors →
    - Increased HR, contractility, preferential vasoconstriction and venoconstriction
  - Chemoreceptors →
    - Increased ventilation
  - Local tissue factors →
    - Vasodilation and capillary recruitment



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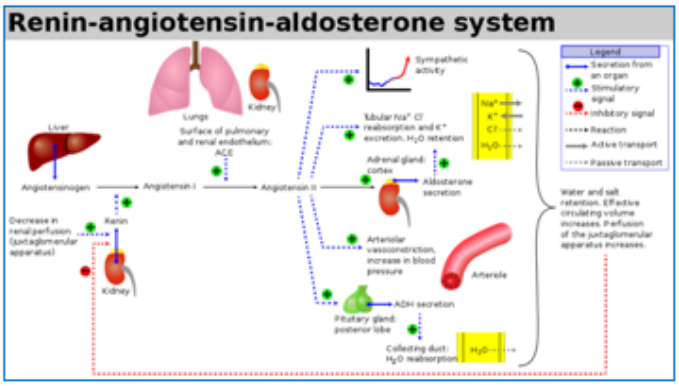
- Compensation can separated into immediate, intermediate, and late compensation phases
- The immediate phase occurs within minutes
  - Involves baroreceptor mediated stimulation of the SNS
    - Baroreceptors are located in left atrium, aortic arch, and carotid sinus
    - Stimulation leads to increased HR, increased contractility, and vasoconstriction with the goal to increase perfusion
  - You also have chemoreceptors that sense hypoxemia and metabolic acidosis
    - Their stimulation increases ventilatory drive and RR
  - Then finally you have local tissue factors
    - A decrease in blood oxygen (PaO2) causes the local metabolites to try to regulate vasomotor tone
    - Release of prostaglandins, NO, and adenosine will induce vasodilation
      - This allows capillary recruitment where its needed
      - During hemorrhage, the number of open capillaries increases proportionally to the degree of tissue hypoxia
    - This promotes oxygen diffusion by decreasing distance to tissue beds and increasing surface area
      - Diffusion rate inversely proportional to thickness and directly proportional to surface area

- This doesn’t always happen in all shock – can have microcirculatory dysfunction with septic shock bc of variation in inflammatory mediators and cytokines
    - So have to keep in mind with trauma patients bc if have significant tissue damage then may have upregulation of these factors

Hemorrhagic Shock

- Intermediate compensation
  - Transcapillary shifts (Starling's law)
  - RAAS



- Next is the intermediate phase
- In this phase you get transcapillary shifts based on Starling's law
  - But basically it refers to the fluid shifts that result in interstital and intracellular compartment fluid being pulled into the vasculature
  - Can return plasma volume up to 50% of original volume and arterial blood pressure up to 75% of its original
- Also RAAS (Renin Angiotensin Aldosterone system) plays a role in this phase
  - RAAS is triggered by: decreased renal perfusion sensed by the juxtaglomerular apparatus, decreased sodium/chloride is sensed by the macula densa, and in response to norepi release and increased renal sympathetic activity
  - Renin is secreted by the juxtaglomerular cells of the kidney in response to decreased renal perfusion (sensed by stretch receptors in the JG apparatus), decreased sodium/chloride is sensed by the macula densa and in response to norepi release and increased renal sympathetic activity.
  - Renin then cleaves angiotensinogen produced by the liver into Angiotensin I.
  - Ang I is then converted to Ang II by ACE on pulmonary and renal epithelial cells
  - Ultimately get Ang II then mediates vasomotor tone and water retention by
    - Stimulates norepi release from adrenal medulla and

sympathetic nerve terminals THEN
    - Vasopressin (ADH) released from pituitary gland (posterior lobe) → binds to arteriolar V1 receptors causing vasoconstriction
        - Binds to V2 Rc in collecting ducts of kidneys and induces insertion of aquaporin channels → reabsorb water
        - Also stimulates thirst
    - Aldosterone released from adrenal cortex and causes Na reabsorption in proximal tubule by activating Na/H antiporter
    - Causes preferential efferent arteriole vasoconstriction
- Ultimate goal is to increase effective circulating volume

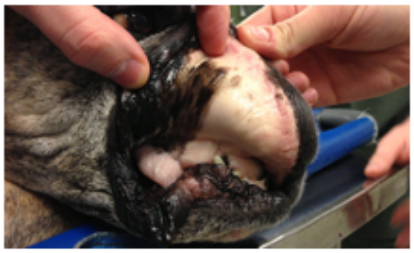
Hemorrhagic Shock

- Late compensation
  - Mainly RAAS
  - Eventually compensation fails
    - → Inadequate CO, inadequate MAP
      - → Poor tissue perfusion
        - → Inadequate oxygenation
          - This is where we start referring to decompensated shock…

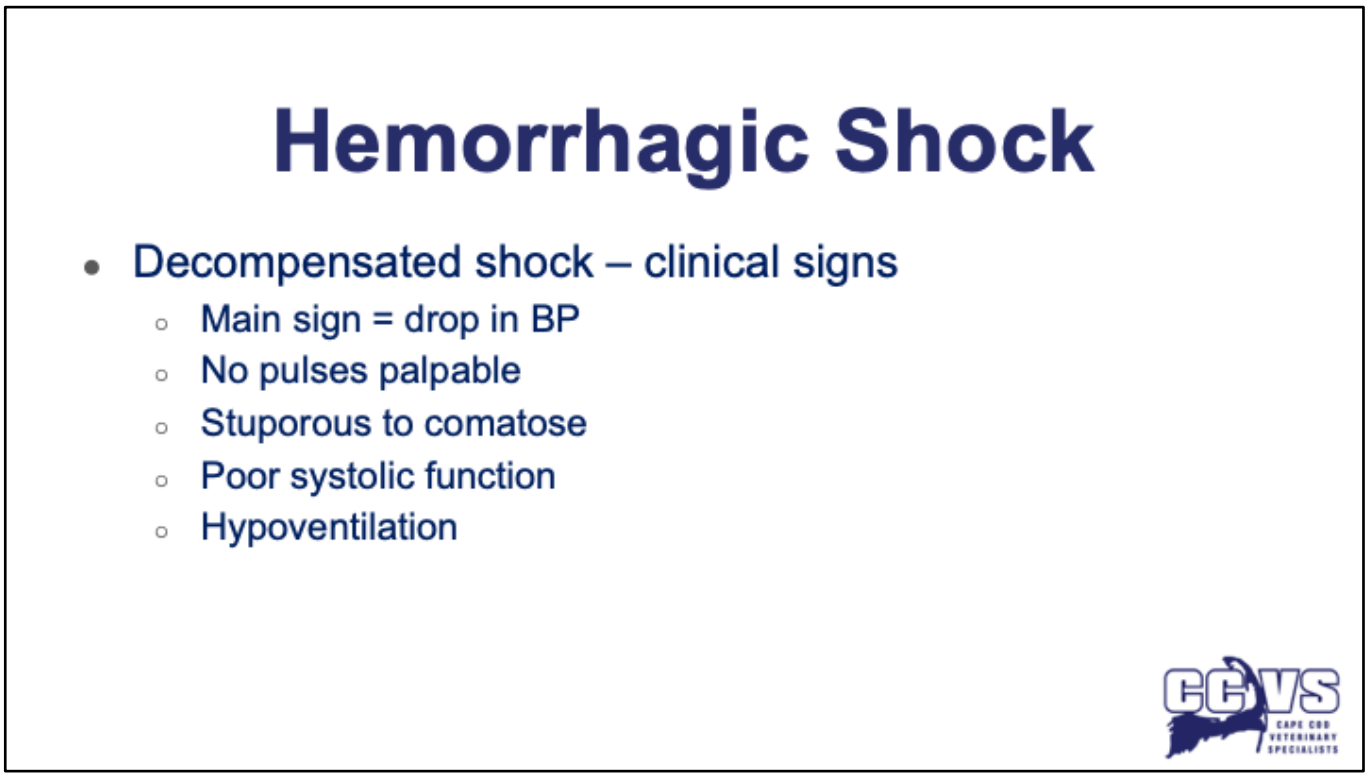
- Then finally you have the late phase
- It mainly consists of continued renal mechanisms to promote a positive fluid balance through RAAS
- If you have continued losses then eventually these compensatory mechanisms become exhausted
  - You get inadequate CO which leads to inadequate oxygenation and you get decompensated shock

Hemorrhagic Shock

- Compensated shock – clinical signs
  - Can maintain MAP
  - Tachycardia
  - Vasoconstriction → pale MM, prolonged CRT
  - Increased RR from decrease in O2 carrying capacity
  - Cats….



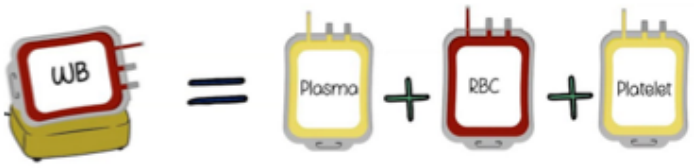
- So steering away from pathophys, Lets talk about what this looks like in our patients
  - First recognizable sign Is often tachycardia
    - So with that you also see pale MM, prolonged CRT
  - But often will have normotension due to the compensation mechanisms
  - RR increases due to decreased CaO2 (arterial oxygen content)
  - Don't forget too about splenic contraction - splenic contraction may mask decreased PCV until fluid shifts from IST and neurohormonal (RAAS, ADH) are induced or give IVF
  - Important point though is cats… tend to bypass the acute compensatory responses and commonly present with hypotension, bradycardia, and hypothermia



- As shock progresses, without any resuscitation efforts (or maybe even with efforts), you get decompensated shock
- This is where your BP starts to drop and you get all the signs that go along with that – lack of pulses, comatose, decreased systolic function, hypoventilation
- Eventually it can lead to irreversible shock
    - systemic vasodilation with decreased cardiac filling pressures, myocardial systolic depression, and widespread evidence of cell dysfunction
- At this state patient is critical and at risk of passing

Hemorrhagic Shock

- Goals = hemostasis and restore perfusion
- Strategy
  - Traditional
  - Damage control/hypotensive
- Fluid options
  - Blood products
  - Isotonic crystalloids
  - Synthetic colloids
  - Hypertonic solutions



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- We resuscitate!
  - Resuscitation strategies have continued to evolve over the years
- Obviously the first goal is to stop the bleeding if you can easily
  - Pressure, tourniquet, belly band
- With all we've discussed it makes sense that the next goal of resuscitation would be to restore tissue perfusion – obviously these are not separate goals, going on together
  - This is achieved by re-establishing vascular volume, cardiac output and arterial oxygen content
  - Also hope to mitigate coagulopathies, metabolic acidosis, and hypothermia
- We have lots of options to achieve this so let's chat about them and strategies

Hemorrhagic Shock

- Traditional resuscitation
  - Rapid large volume crystalloids
  - Restore BP back to normal as quickly as possible
  - Sometimes need 4x volume for it to remain in intravascular space
  - Risks: hyperchloremic metabolic acidosis, AKI, organ edema, dilutional coagulopathy, destruction of glycocalyx, increased risk of rebleeding
  - No longer recommended in human medicine

- First theres the Traditional approach to trauma resuscitation
  - Rapid large volume crystalloids, usually in 3:1 ratio, sometimes 8:1 for severe shock
    - need 4x volume that you give for it to remain in intravascular space
  - The goal was to restore the BP back to normal as quickly as possible
  - But large volume approach has been shown to be deleterious in cases of uncontrolled hemorrhage
  - Previous studies have linked fluids with high Cl loads with hyperchloremic metabolic acidosis and AKI via chloride-mediated renal vasoconstriction (increased Cl delivery to macula densa)
  - Also Organ edema, ***Coagulopathy,*** Alterations of inflammatory cascade, destruction of THE GLYCOCALYX
    - So can cause organ damage and increase risk of re-bleeding
  - No longer recommended in human med

Hemorrhagic Shock

- Damage control resuscitation
  - Use of hypotensive and hemostatic resuscitation
  - Blood products!!
  - Early prevention and reversal of lethal triad (now diamond), even before definitive resuscitation or surgical repair
  - Antifibrinolytics

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- The latest supported approach is damage control resuscitation – it is currently promoted as the most optimal approach
  - It involves using permissive hypotension or hypotensive resuscitation to restore perfusion without disrupting thrombus formation
- It also uses hemostatic resuscitation = use of whole blood or combined blood productions with limited crystalloids
  - the goal is to avoid causing iatrogenic hemodilution and exacerbating metabolic acidosis and hypothermia
  - also aims to treat any coagulopathy – given the premise that ATC has a hyperfibrinolysis component, this is where antifibrinolytics come into play
- Ultimately the goal is reverse the lethal triad to optimize the patient prior to pursuing definitive resuscitation or surgical repair

Hemorrhagic Shock

- Hypotensive resuscitation
  - Traditional = aim for normal systolic BP 120 mmHg
  - Instead aim for SBP 80-90 mm Hg
  - Recent literature = improved survival
    - Helps control hemorrhage and reduces risk of re-bleeding
    - But just to bridge gap until definitive hemostatic control
  - Not for TBI patients?

CPP = MAP - ICP

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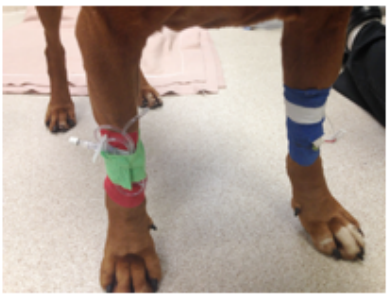
- For hypotensive resuscitation the goal for resuscitation changes
- In traditional resuscitation the goal is for systolic BP that is normal around 120 mmHg
- But for hypotensive resuscitation, you aim for 80-90 mmHg for systolic, thinking the MAP will be around 60 mmHg - which should be enough for organ perfusion
  - If BP not available then you can titrate to feeling pulses or until mental status improves
- Once you reach these goals you just maintain, there is no restoration of circulating blood volume until definitive hemostasis
  - Then use FWB or appropriate ratio of blood components to restore, again avoiding large volume of crystalloids to avoid worsening metabolic acidosis and dilutional coagulopathy
- Important to note that this may not be appropriate for TBI patients since CPP is depending on MAP
  - Some studies have shown no adverse affect on TBI patients with delayed resuscitation of circulating blood volume
  - The other difference is that HTS is recommended as the initial resuscitative fluid of choice – which is not true for other trauma patients

Hemorrhagic Shock

- Where to deliver resuscitation?
  - IV is best
  - Large gauge, short length
  - Multiple IVs, front leg if possible
  - Jugular catheter – can cut down if needed
    - Failing at the cutdown?? Use IO - faster
- How fast?
  - Over 10 to 30 minutes for first fluid challenge
  - But depends on resuscitation strategy



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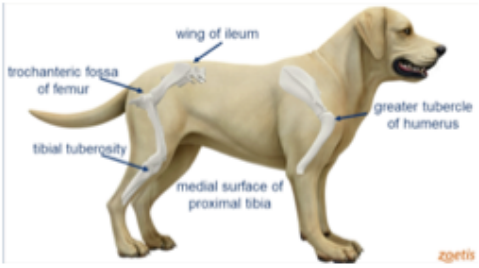
- Other important points
  - Ideally IV resuscitateion but can use IO if unable to get IV access
  - Use largest gauge, short length
  - Ideally front legs
- Which factor in Poiseuille's law is most important for increasing flow of your fluid into patient? Increasing the radius
- Give first fluid products over 10 to 30 minutes

Hemorrhagic Shock

- IO placement
  - Large gauge needle
  - Sterile prep, same as IV
  - EZ IO gun
    - https://www.youtube.com/watch?v=10twNYP1pB0



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- Again just wanted to remind that IO access can be just as valuable
  - Doesn't collapse in hypovolemia
  - Can use for fluids, boluses, blood products, drugs including CPR drugs
  - Multiple access options as you see here
  - Can use IO gun or just large gauge needle
  - This may be only option for very small patients
- Risk of pain and osteomyelitis
- https://www.zoetisus.com/petcare/blog/io-catheter-placement/
- https://www.cliniciansbrief.com/article/intraosseous-catheterization-often-underused-life-saving-tool

Hemorrhagic Shock

- Resuscitation
  - Whole blood is best!
  - Otherwise you should consider appropriate ratios of blood component products
    - FFP:pRBC:platelet (1:1:1) or FFP:2 pRBC (1:2)
    - Goal = decrease risk of dilutional coagulopathy and thrombocytopenia
  - But no standardized algorithm in vet med

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- Ok so to talk briefly on fluid choices
- Whole blood or blood component therapy is the primary therapeutic choice for replacing human trauma victims' lost blood volume
- For the blood component, there are several recommendations for ratios but ultimately the idea is to avoid dilutional coagulopathy
  - it can occur if the ratio of the blood products we are giving are significantly off
  - So are we giving multiple pRBC transfusions quickly without FFP? That can lead to a dilutional coagulopathy, hypofibrinogenemia, and thrombocytopenia
  - This is why whole blood is the first choice – but often don't have that option when patient comes in dying
  - The recommendation is 1:1:1 or 1 FFP for every 2 pRBC
    - These are based on human studies – don't have standardized algorithm in vet med yet – but hopefully with trauma registry will start to get more info
- But as we know blood products are not always available and not without risk

Hemorrhagic Shock

- pRBC
  - Replace Hgb, anemia
  - 75-80% remain in circulation for 1st 24 hours
  - But do they transport and deliver O2? i.e. do they actually help with shock state?
  - No colloidal support
- FFP
  - Replace impaired coagulation factors
  - Reduce endothelial inflammation and hyperpermeability
  - Colloidal support
  - Restore the glycocalyx
  - But takes time to thaw

- pRBC
  - Goal is to replace hemoglobin and provide O2 transport
  - Most remains in circulation for a day
  - Can give DEA 1.1 negative if don't have blood type
  - But unsure if they can actually hold onto O2 and deliver it
  - Does not provide colloidal support – which is other major part of blood loss, its not just the red cells that are the problem
  - Also often cold – contributes to hypothermia

- FFP
  - Provide colloidal support – hope to keep any crystalloids given in vessel
  - Also restores glycoalyx to hopefully have less risk of hyperpermeability and the effects on coagulation
  - Replaces impaired or lost coag factors
  - Major downside is takes time to thaw if don't keep a thawed unit – often cold so contributes to hypothermia

Hemorrhagic Shock

- Mantel Technologies' canine freeze-dried plasma
  - FDA approved Dec 2025
  - 250 mL single-use bag, 250 mL bag of sterile water for injection diluent, and an administration set
  - Administered intravenously at a dose of 10-20 mL/kg body weight
  - Longer shelf life = Shelf stable for a year
  - Don't require thawing
  - After reconstitution – can be stored at room temp for 3 hours or refrigeration for 3 days
  - $750/unit

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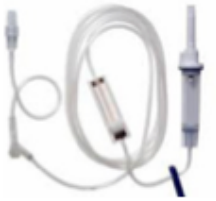
- But SUPER exciting as of Dec 2025 – there is a freeze dried product available
- This cFDP product is shelf stable for at least one year and can be reconstituted for intravenous use in acute medical emergencies in dogs when whole blood products may not be available, such as with trauma cases with military working dogs and dogs in remote locations.
  - Typically, fresh blood and plasma products require storage in refrigerators or freezers, have short shelf lives, and may require thawing before use.
- Each Mantel's cFDP kit includes 20 grams of Mantel's cFDP in a 250 mL single-use bag, 250 mL sterile water for injection to reconstitute the cFDP, and a set to administer the product to a dog intravenously.
- The product requires reconstitution before use. Once reconstituted, the cFDP bag is equivalent to 250 mL canine plasma and should be infused immediately.
- However, it may be stored at room temperature between 68-77 degrees Fahrenheit (20-25 degrees Celsius) for up to 180 minutes or stored under refrigeration between 34-43 degrees Fahrenheit (1-6 degrees Celsius) for up to three days, as long as the plasma and kit are used only once for a single patient.
- But of course it is costly…

https://www.avma.org/news/freeze-dried-plasma-now-option-canine-emergency-treatment
https://www.fda.gov/animal-veterinary/cvm-updates/fda-completes-risk-review-freeze-dried-plasma-treatment-hypovolemia-and-control-hemorrhage-dogs
https://www.aaha.org/trends-magazine/publications/canine-freeze-dried-plasma-available-for-veterinary-use/

Hemorrhagic Shock

- Autotransfusion
  - Cases to consider
    - Traumatic hemoabdomen or hemothorax
    - No/limited access to blood products
    - Unable to have quick financial conversation
  - Contraindications: uroabdomen, septic abdomen
  - No need to anticoagulate, already undergone fibrinolysis
  - Collect sterile in 60 mL syringes (small patients) or into sterile IV bag with 3 way stopcock (large patients)
  - Use blood filter to administer to patient
    - Reminder: hemonate filter change every 50 mL
    - So large patients use blood admin set with incorporated filter



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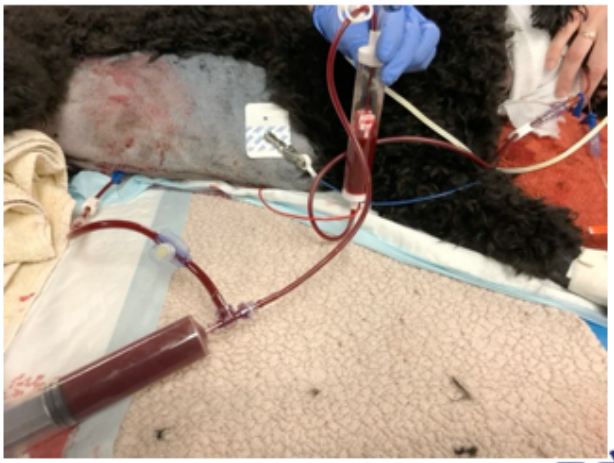
- Another option to not forget about for resuscitation is autotransfusion
  - This can be used when you have large volume hemorrhage in a cavity – typically abdomen
  - Can be lifesaving, inexpensive, fast, limited skill needed
  - We typically consider when we have to go quick and can't warm up blood, and when we don't have time to talk about financial implications for transfusions bc they are expensive
- The benefit of autotransfusion, is that it is a readily available transfusion product available from the patient.
  - As it is from the patient, it will not result in a transfusion reaction.
- However, autotransfusions should only be performed when there is no evidence of contamination from urine (e.g., uroabdomen), bile (e.g., bile peritonitis), bacteria (e.g., septic peritonitis), etc.
- As the blood has already undergone fibrinolysis, typically there is no need to add anticoagulants (such as CPDA, etc.) to autotransfusions during collection.
- Ideally, blood should be sterilely collected in 60 ml sterile syringes.
- A blood filter should be used when administering the autotransfusion back to the patient.
  - Hemonates need to be changed about every 50 mL
  - So large patients may need blood admin set with incorporated filter
- Alternatively, one can collect the blood via a three-way stopcock system and collect it directly into a sterile bag (e.g., a used IV fluid bag).
- https://vetgirlontherun.com/perform-autotransfusion-dog-vetgirl-veterinary-

continuing-education-blog/

Hemorrhagic Shock

- Autotransfusion
  - How to video: https://vetgirlontherun.com/perform-autotransfusion-dog-vetgirl-veterinary-continuing-education-blog/



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- This is a how to video for your reference

https://vetgirlontherun.com/perform-autotransfusion-dog-vetgirl-veterinary-continuing-education-blog/

Hemorrhagic Shock

- Platelet concentrate
  - Reduce platelet dysfunction and avoid dilutional thrombocytopenia from massive transfusion
  - Goal > 50K in polytrauma or > 100K if CNS injury
  - Still lack of evidence of efficacy, benefit
  - Platelet concentrate takes time to thaw
    - Freeze dried in the future??

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- Platelets
  - Reduce platelet dysfunction and avoid dilutional thrombocytopenia from MT
  - Goal > 50K in polytrauma or > 100K if CNS injury
    - Practically when I use for ITP or trauma its until bleeding stops, not goal of particular number
  - Still lack of evidence
  - Large patients require multiple units
  - Platelet concentrate takes time to thaw

Hemorrhagic Shock

- Albumin
  - Provides colloidal support after blood loss
  - Helps maintain endothelial integrity (glycocalyx), wound healing, decrease vascular leak/third spacing
  - Can't bolus
  - Canine vs human
    - Risk with human albumin Type III hypersentivity reaction
  - Canine albumin currently difficult to acquire quickly
    - So often using FFP initially and albumin later

- Next is canine albumin
- The main indication for canine albumin is to help with plasma volume in shock as well as in patients that have hypoproteinemia (such as in sepsis, trauma).
  - Low in dz from loss, vascular leak, third space, decreased hepatic production
  - The goal with the administration of any albumin product is not to achieve a normal albumin level but to improve the albumin serum level.
- It helps with endothelial integrity (glycocalyx), wound healing, metabolic and acid base function, coag, free radical scavenge
- However can't bolus
- Human 25% vs Lyophilized canine
- The product is good for 24 hours once reconstituted. It must be refrigerated at 4^0C if not used immediately.
- The suggested dose is 0.8 gram/kg. A slow infusion is recommended, at a rate of 1 ml/min. However, the rate of CSA infusion can vary based upon the product, but up to 6 hours is routinely reported.
- Human albumin
  - The main concern with giving a non-canine product, like human albumin, to a dog is not only an acute reaction (Roughly 7–10% of dogs have naturally occurring antibodies, causing acute anaphylaxis upon initial exposure) but also Type III (delayed) hypersensitivity can occur 5–16 days post-infusion

        - And if receives again fatal reaction bc will have antibodies to it
        - But is cheaper than canine albumin (human $162 while canine is $750)
- https://www.mspca.org/angell_services/canine-specific-albumin-csa/

Hemorrhagic Shock

- Massive transfusion
  - Definitions
    - Whole blood volume in 24 hours
    - 50% blood volume in 3 hours
    - 1.5 mL/kg/min for 20 minutes
  - Adverse effects: hypothermia, hypocalcemia, hypomagnesemia, transfusion reaction, TACO, TRALI, $$$, massive use of limited resources

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- Obviously massive transfusion is common risk in these patients
- There are several definitions
  - Whole blood volume in 24 hours
  - 50% blood volume in 3 hours
  - 1.5 mL/kg/min for 20 minutes
- But MT can cause adverse effects
  - the blood products are cold and may not have time to warm, contributing to hypothermia
  - low Ca and Mg due to chelation by citrate anticoagulant – treat if clinical signs of hypocalcemia
  - risk of transfusion reactions including transfusion associated circulatory overload and transfusion reaction acute lung injury
  - also costs money and use of limited resources

Hemorrhagic Shock

- Isotonic crystalloids
  - Similar to extracellular space (IV and ISF)
  - 75% rapidly redistributes to interstitial space
  - Shock dose
    - Dog 60-90 mL/kg
    - Cat 45-60 mL/kg
    - Bolus 10-20 mL/kg over 15-30 minutes
  - Easy to access, easy to give, room temperature or warmed
  - Adverse effects: organ edema (renal, pulmonary, GI tract), dilutional coagulopathy, damage to glycocalyx, increased inflammation

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- So the most common first resuscitation choice is the one we have the most accessible
- Isotonic crystalloids
  - Similar composition to ECF (IV and ISF)
  - 75% of volume is rapidly redistributed to ISF – but since most of our trauma patients aren't dehydrated, this isn't particularly helpful
    - Shortterm gain but future problems (edema)
  - Classic shock dose 60-90 mL/kg dog and 45-60 mL/kg cat over 45-60 min
    - Bolus 10-20 mL/kg over 15-30 min
    - Resuscitation end points: HR, pulse quality, CRT, temp of extremities, mentation
  - Risks
    - Volume overload = specifically for hypoproteinemia, kidney or cardiac dz
    - Organ edema
      - Pulmonary edema, ALI if increased vascular permeability to SIRS or sepsis
      - GI tract changes → decreased motility, increased permeability, bacteremia, increased risk for abdominal compartment syndrome
    - Cardiac = risk of ventricular arrhythmias, disruption of cardiac contractility, decreased CO

- Coag – dilution of factors, decreased blood viscosity
  - But less than synthetic colloids
- Alteration of inflammation (LRS)
- Negative impact on endothelial glycocalyx

Hemorrhagic Shock

- Hypertonic saline solution 3-23.4%
  - 3-5 mL/kg over 10-20 minutes; redistributes within 30 min
  - Require smaller volume
  - First recommendation for TBI patients
  - Positive immunomodulatory effects
  - Reduced edema formation, reduced endothelial cell swelling, improved regional blood flow, improved CV function
  - But no improvement in mortality
  - Adverse effects: hypernatremia or hyperchloremia, interstitial depletion if dehydrated

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- Next is HTS which is a good option for quick small volume resuscitation and head trauma cases
- Hypertonic 3-23.4%
  - Causes sudden dramatic increase in plasma osmolarity
  - Reduce ICP in TBI
  - There are also some positive Immunomodulatory – decreased neutrophil activation and adherence, stim lymphocyte proliferation, and inhibit proinflammatory cytokine production by macro
  - 3-5 mL/kg over 10-20 min
  - Redistribute within 30 min
  - Adverse
    - Hyper Na and hyper Cl
    - Hypernatremia induced osmotic demyelination if hypoNa
    - Cardiac or pulmonary dz
    - ISF depletion if dehydrated = follow additional fluids

Hemorrhagic Shock

- Synthetic colloids
  - Vetstarch, hetastarch
  - Increased and sustained vascular volume with smaller volumes of infusion
  - Reduced capillary leak and gut interstitial edema
  - Adverse effects
    - Dose related coagulopathy
    - Platelet dysfunction
    - Acute renal injury?

- And finally Synthetic colloids
  - They are Large colloid molecules suspected in isotonic crystalloid solution
    - More sustained IV expansion bc of the molecules
    - Increases colloid osmotic pressure to help hold fluid in the vessels – so may reduce capillary leak and gut interstitial edema
    - Typical = 2-5 mL/kg over 10-30 min
    - Shock = 5-20 mL/kg in dog and 2.5-10 mL/kg in cat
    - Adverse
      - AKI in sepsis
      - Can also contribute to dilutional coagulopathy
        - Colloidal plasma substitutes interfere with physiologic mechanisms of hemostasis – hemodilution or specific actions of macromolecules on plt function, coag proteins, and fibrinolytic system
      - High molecular weight can decrease vWF and FVIII, and plt dysfunction
      - Higher risk with more dose
      - Avoid if renal injury
  - Still debatable in vet med
    - Not recommended in human med bc of the risks
  - I will still use if I'm unable to do autotransfusion and can't keep stable while trying to discuss costs of large volume transfusions
    - Often debating this risk vs risk of giving human albumin (another

cheaper option for colloidal support)

- Personally haven't seen AKI in cases I've used it for – but there are many that were euthanized or passed due to severity of the trauma, so limited sample size

Hemorrhagic Shock

- Pharmacology options for TIC
  - Antifibrinolytics = synthetic lysine analogues
    - Consider within 3 hours of injury
    - Doesn't reduce transfusion requirements but reduced risk of death
    - Increase risk of vascular occlusive events?
    - Tranexamic acid: 10-15 mg/kg IV, can also do CRI 1-5 mg/kg/hr
      - Don't forget maropitant IV 10 minutes before to prevent vomiting
    - Aminocaproic acid: less potent, shorter half-life (60-75 min) so CRI; 50-100 mg/kg diluted IV then 15 mg/kg/hr CRI

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- Hyperfibrinolysis is a staple of TIC so makes sense to consider antifibrinolytics
  - Consider within 3 hours of injury – remember patients may eventually become hypercoaguable so may not make sense to continue
  - Doesn't reduce transfusion requirements but reduced risk of death
  - Risk = Increase risk of vascular occlusive events?
  - Tranexamic acid: 10-15 mg/kg IV, CRI 1-5 mg/kg/hr
    - Can cause vomiting with boluses (50 mg/kg documented) so give maropitant 10 minutes before
    - Can give diluted or undiluted
  - Aminocaproic acid: less potent, shorter half-life (60-75 min) so CRI; 100-150 mg/kg then 15 mg/kg/hr
  - Cats – use similar bolus doses as dogs (TXA 10 mg/kg and Amicar 50 mg/kg)
    - Wahldén L, Stanzani G, Cutler S, Barfield D, Manson KC, Wilson HE, Thomas EK. Evaluation of Therapeutic Use of Antifibrinolytics in Cats. J Am Anim Hosp Assoc. 2023 Jul 1;59(4):177-183. doi: 10.5326/JAAHA-MS-7349. PMID: 37432789.

So what do I do?

- Vitals – TPR, BP
- IV or IO access
- Bloodwork off of IV catheter placement (PCV/TP, Lac, BG)
- POCUS to try to assess blood loss
- Resuscitation
  - Balanced crystalloid bolus 10 mL/kg; repeated as needed
  - HTS 3-5 mL/kg bolus
  - Autotransfusion if able
  - TXA 10-15 mg/kg IV or aminocaproic acid 50-100 mg/kg IV
  - Recheck vitals

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- Ok so now practically what do we do when these cases fly through our day
  - Start with vitals
    - Vitals to determine compensated vs decompensated shock = TPR, BP
  - Blood: BG, lactate, PCV/TP is great baseline basics
    - Can do coag testing but honestly not super important at start – I'm going to assume TIC with major trauma; consider if not stabilizing
  - POCUS
    - If able to do POCUS to assess for source of bleeding and degree – also to see if you have a source for autotransfusion
  - And then for resuscitation
    - Can start with crystalloid bolus
    - HTS too or in particular for TBI cases
    - If hemoabdomen or hemothorax then can consider autotransfusion
    - If able to do antifibrinolytic great
    - Most important aspect of resuscitation is re-evaluation – did I do enough? Do I need to keep going?
      - Also important for referral – know where the pet was at after initial resuscitation

Thoracic Injuries

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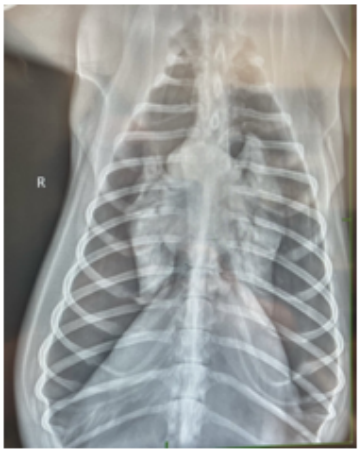
Thoracic Injuries

- Pneumothorax
- Pulmonary contusions
- Fractured ribs and flail chest
- Hemothorax
- Diaphragmatic hernia
- Pericardial effusion



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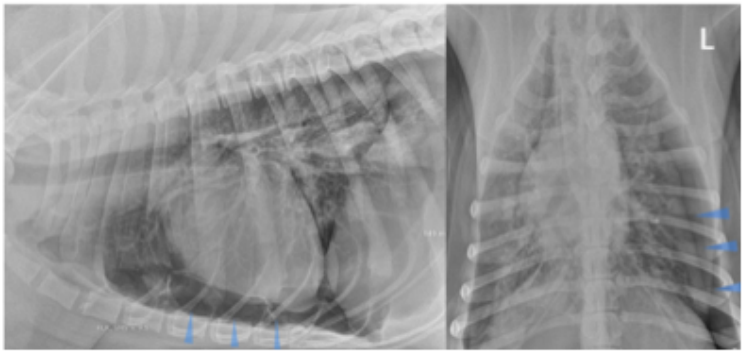
Thoracic Injuries

- Pneumothorax
  - Most common complication of blunt trauma to chest
  - Open = injury to chest wall
  - Closed = leakage of air from lesion inside
  - Tension = site of air leakage creates one way valve during inspiration → rapidly increasing pleural pressure → exceeds atmospheric pressure → decreased venous return

- Pneumo = abnormal accumulation of air in the pleural space. Air accumulation is most commonly bilateral but unilateral pneumothorax can occur.
  - It is the most common complication of blunt trauma to the chest.
  - Closed pneumothorax is seen following trauma due increased intra-thoracic pressure against a closed glottis causing rupture of alveoli or small airways, laceration of lung by fractured rib, iatrogenic, and airway or esophageal rupture causing pneumomediastinum which has progressed to pneumothorax.
    - Closed secondary to leakage of air from lesion within resp tract, esophagus, mediastinum, or diaphragm
  - Open pneumothorax may result from gunshots, dog bites, knife wounds, and stick impalement = injury to chest wall
  - Tension pneumothorax is the third type, resulting when an air leak acts as one-way valve increasing intrathoracic pressure, compressing the lungs and decreasing venous return to the heart.
    - Tension = if site of air leakage creates one way valve during inspiration and results in rapidly increasing pleural pressure that exceeds atmospheric pressure often leading to arrest

Thoracic Injuries

- Pneumothorax
  - Diagnostics
    - CXR
    - POCUS
    - Diagnostic thoracocentesis if decompensating



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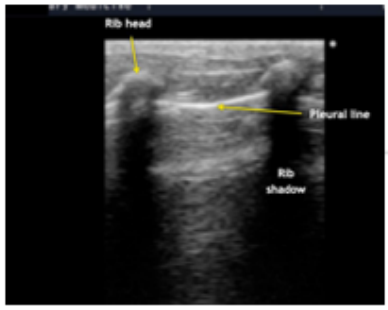
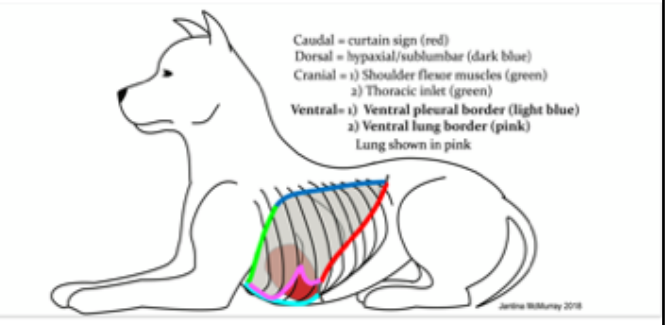
- Pneumo – how to diagnose
  - PE findings: Common examination abnormalities include an increased respiratory rate and effort characterized by a short and shallow breathing pattern, dull lung sounds dorsally, and muffled heart sounds. Less specific examination abnormalities may include pale or cyanotic mucous membranes, poor pulses, and an abnormal posture with the head and neck extended and elbows abducted.
  - CXR: elevation of the cardiac silhouette from the sternum, collapse of the lung lobes, and absence of vascular markings out to the periphery of the thorax.
  - Diagnostic tap if not stable enough – ie do this to check for pneumo in a critical trauma patient

Thoracic Injuries

- Pneumothorax
  - POCUS
  - https://youtube.com/shorts/i0w_nXRil30?si=6wfvFYwylfydWNP9

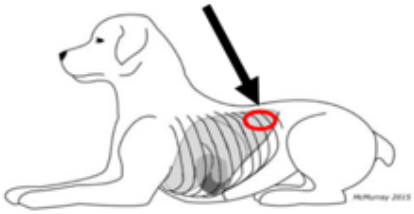


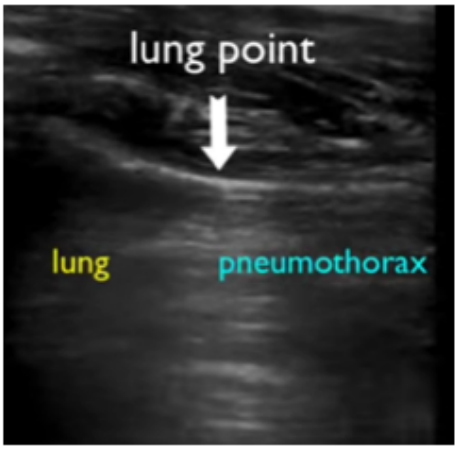


- Pneumo
  - POCUS
  - As a general rule of thumb, the authors' scan the lung and pleura in a sliding fashion in large "S" pattern) on each side of the chest, plus the subxiphoid site, to ensure adequate exploration of the lungs.
  - There are 5 key borders of PLUS:
    - 1. The caudal border, which is defined by the curtain sign (more to come on this below),
    - 2. The dorsal border, which is defined by the hypaxial muscles,
    - 3. The cranial border, which is not the true border of pleura and lung but the border that is limited by the thoracic limb,
    - 4. The ventral pleural border,
    - 5. The ventral lung border. There are two ventral borders because of the cardiac notch
  - Diagnosis of pneumothorax is based on absence of glide sign
  - https://www.canadianveterinarians.net/media/ltglcqsz/pocus-09-2022-lab-manual.pdf

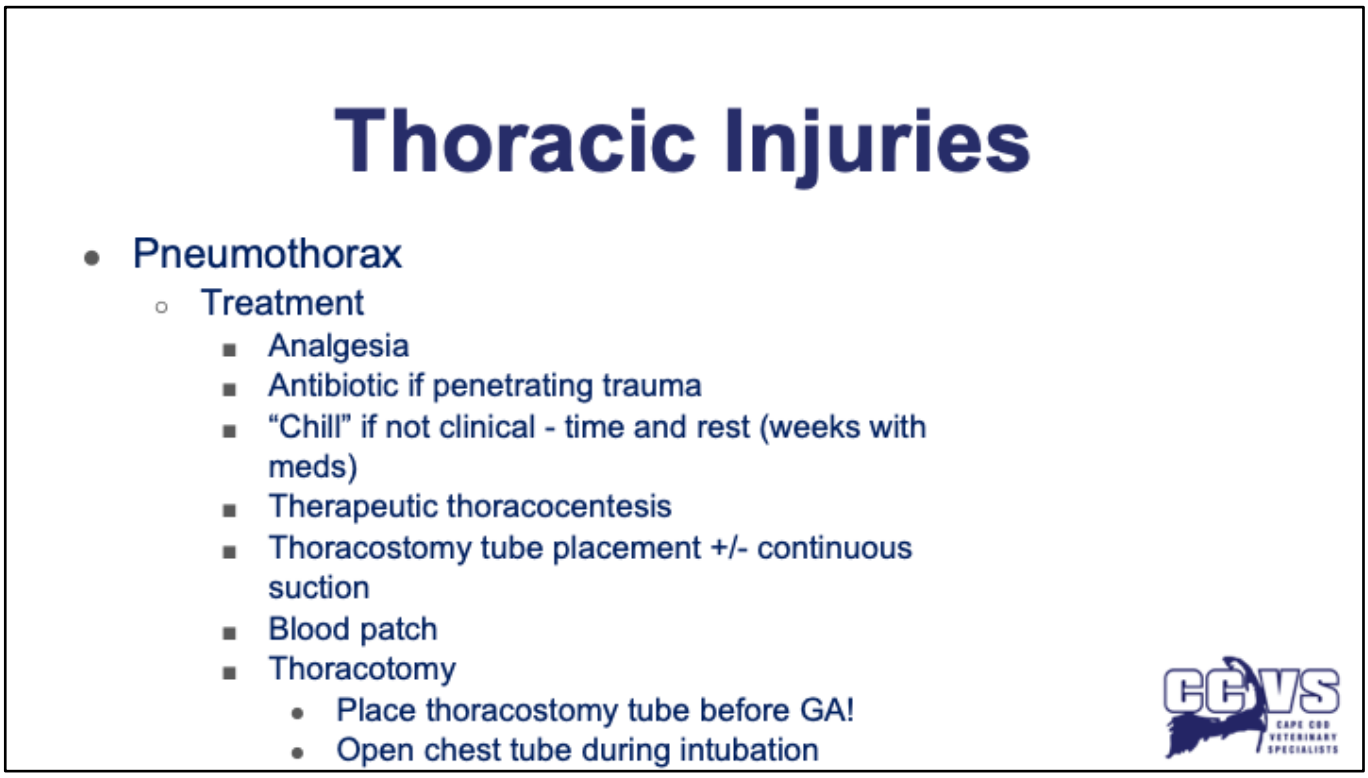
Thoracic Injuries

- Pneumothorax
  - POCUS
  - https://youtube.com/shorts/i0w_nXRil30?si=6wfvFYwylfydWNP9

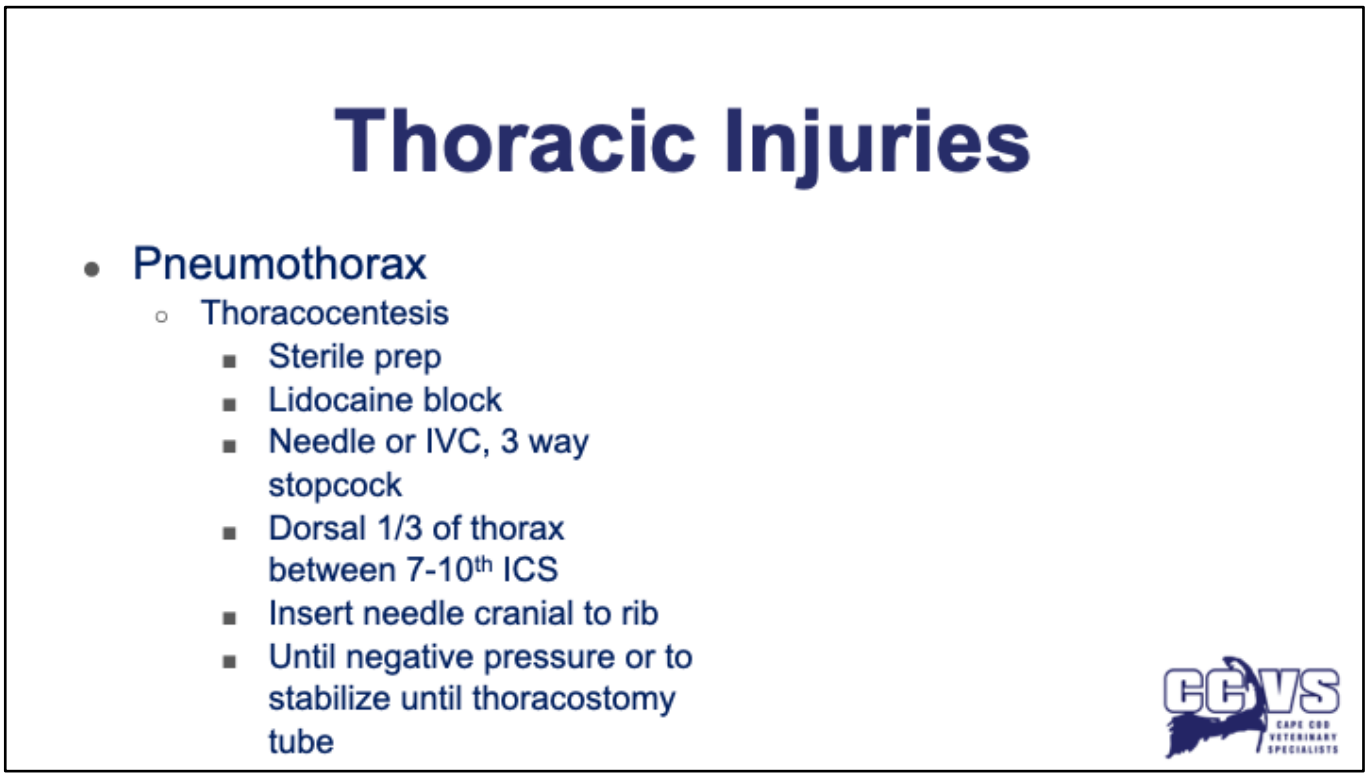




- Pneumo
  - You can also via the lung point
  - The lung point is defined as the site within the thorax where the lung recontacts the parietal pleura and creates an intermittent glide sign within half the ultrasound beam when the patient breathes. It is the exact point within the thorax where there is a return of the glide sign: movement of the probe from an area where there is no perceived glide sign, to an area where the glide sign reappears intermittently within a region of the ultrasound image
  - https://www.canadianveterinarians.net/media/ltglcqsz/pocus-09-2022-lab-manual.pdf



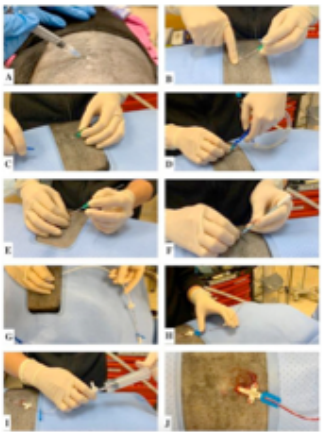
- Treatment for pneumo is dependent on severity
  - First don't forget about analgesia – they may also have rib fractures
  - If it is open then need antibiotics
  - If patient is stable and doesn't progress then may be able to take "chill" approach which is wait and watch
    - When they go home, keep them quiet for weeks
  - However if patient is unstable or worsens during stay then need to intervene
    - If becomes resp or CV unstable
- This means placing chest tube +/- thoracotomy
  - Remember if going into thoracotomy with pneumo then need chest tube before intubate and must open the chest tube on intubation
- In some cases can also consider blood patch
- https://vetgirlontherun.com/videos/how-to-place-a-mila-thoracostomy-chest-tube-via-the-seldinger-technique-in-a-dog-vetgirl-veterinary-continuing-education-videos/
- https://todaysveterinarypractice.com/emergency-medicine-critical-care/blood-patch-pleurodesis-for-small-animal-pneumothorax/
- https://www.sciencedirect.com/science/article/pii/S1938973621000751



- To perform thoracocetesis
  - The equipment needed for this procedure includes clippers, scrub, sterile gloves, a 10-60ml syringe, 3-way stop-cock, butterfly catheter or needle and extension tubing.
  - The site preparation and eventual needle placement for a patient suspected of a pneumothorax is on the dorsal 1/3 of the thorax between the 7th-10th intercostal spaces.
  - The needle is inserted cranial to the rib to avoid the intercostal artery, vein, and nerve located caudal to each rib. Air is aspirated until negative pressure is obtained
  - https://www.cliniciansbrief.com/article/thoracocentesis

Thoracic Injuries

- Pneumothorax
  - Thoracostomy tube placement +/- continuous suction
    - Indications = > 2 taps in 24 hr, tension pneumothorax, positive pressure ventilation, unable to get negative pressure
    - Sterile prep
    - Lidocaine block
    - Large bore = painful, heavy sedation or GA
    - Smaller bore = local analgesia, modified Seldinger
    - Placed at 7-10th ICS



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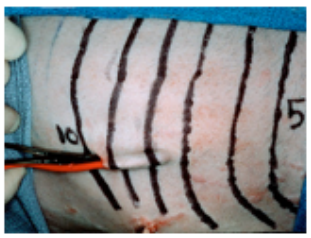
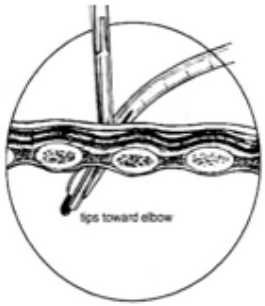
- Then if not resolved need to consider chest tube
- Indications for thoracostomy tube = more than 2 taps in 24 hr, tension pneumo, PPV, when can't get negative pressure
  - Large bore chest tubes require sedation or general anesthesia.
  - Smaller bore chest tubes are also available, placed via the modified seldinger technique with the patient awake or receiving local analgesia.
  - Equipment required for chest tube placement includes clippers, surgical scrub, surgical blade, local analgesia, suture material, the thoracostomy tube, 3-way stop-cock and syringes for initial aspiration. The chest tube can be used intermittently or attached to a suction device for continuous suction.
  - The technique for chest tube placement will depend on the type of tube used, including surgical and trocar methods for the larger bore tubes or the modified seldinger technique for the smaller bore tubes.
  - Like the thoracocentesis, surgical preparation of the site between the 7th-10th intercostal spaces is recommended.
  - Pleurovac, thora-seal, Heimlich valve
  - https://vetgirlontherun.com/videos/how-to-place-a-mila-thoracostomy-chest-tube-via-the-seldinger-technique-in-a-dog-vetgirl-veterinary-continuing-education-videos/
  - https://todaysveterinarypractice.com/emergency-medicine-critical-care/blood-patch-pleurodesis-for-small-animal-pneumothorax/

- https://www.sciencedirect.com/science/article/pii/S1938973621000751
- https://www.vin.com/apputil/content/defaultadv1.aspx?pId=20539&catId=113449&id=8506453&ind=274&objTypeID=17

Thoracic Injuries

- Pneumothorax
  - Thoracostomy tube placement +/- continuous suction
    - Red rubber
  - https://www.youtube.com/watch?v=_-wzE0zJuh4





- If don't have a MILA chest tube then can use a red rubber
- Lidocaine block
- Make a small (2-3 cm) skin incision at the 10th intercostal space.
- Incise through the subcutaneous tissue and the lattisimus dorsi muscle. Use a Carmalt or Pean forcep to create a tunnel underneath the lattisimus dorsi muscle from the skin incision to the 8th intercostal space.
- Grasp the tip of a red rubber catheter with a Carmalt forcep or use an Argyle trocar catheter.
- Insert the tip of the tube into the skin incision and advance it through the tissue tunnel to the level of the 8th intercostal space.(
- Firmly grasp the tube and insert through the chest wall with a forceful but controlled effort.
- If using a red rubber catheter and forceps, after entering the chest open the forceps, slide the catheter into the pleural cavity, then remove the forceps from the incision.
- Promptly place a clamp on the tube after entering the chest to prevent pneumonthorax.
- Place a 3-way stopcock on the end of the tube(Fig. 5); a Christmas tree adapter may be necessary to fit the stopcock to the flared end of the tube.
- Use large suture or wire to secure the stopcock and adapter to the tube and place a purse string and finger trap pattern suture to secure the tube to the skin
- A "C" clamp can also be placed on the tube for added safety in case the

stopcock should become dislodged. Apply antibiotic ointment to the tube entry site in the skin and protect the tube on the patient with a bandage.

- https://drstephenbirchard.blogspot.com/2017/12/how-to-safely-place-chest-tube-in-dogs.html

Thoracic Injuries

- Pneumothorax
  - Treatment
    - Blood patch
      - Pleurodesis
      - Autologous or allogenic blood
      - Only if patient is stable
      - Chest tube
      - 5-10 mL/kg within 2-3 minutes after collection
      - Roll left to sternal to right to distribute
      - Don't reaspirate 2-6 hours after if possible

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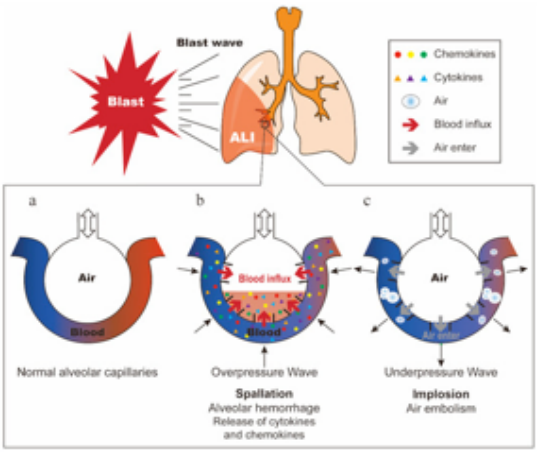
- And then in rare cases can consider blood patch
- But often not option in hypovolemic patients
- Blood patch
  - Pleurodesis as a sole method of treating pneumothorax may be considered a less invasive and less expensive option; however, it will not resolve underlying pathology.
  - Pleurodesis eliminates the pleural space by creating a permanent physical adhesion between the visceral and parietal pleura, often by inciting inflammation through physical or chemical methods.
  - When inflammation is incited, the permanent adhesion may take several days to complete.
  - it has also been theorized to result in formation of a small patch of coagulated blood over the air leak site, resulting in more rapid pneumothorax resolution.
  - BPP can be performed by using autologous or allogenic blood.
    - Autologous ABPP is collection of venous blood from the same patient that will receive the pleural infusion. The normal patient should be able to sustain, without injury or hypovolemia, the loss of 5 to 10 mL/kg used for ABPP;
    - ABPP is rarely considered an emergency procedure and should only be performed on a stable patient. In human medicine, ABPP is more commonly recommended when the air leak is persistent (>5 days).

- Allogenic BPP, in which the blood is collected from a separate donor, has also been reported.
    - Allogenic BPP carries a potential higher risk for adverse transfusion reactions; therefore, prepleurodesis diagnostics (e.g., blood typing, cross-matching, other standard prescreening), along with close postpleurodesis monitoring, using current consensus guidelines for standard allogeneic transfusion are recommended.
- Although an ideal amount to administer has not been determined, 5 mL/kg per hemithorax to be treated (maximum of 10 mL/kg) is conventionally recommended on the basis of previous retrospective evaluations in dogs and cats.
- Immediately before administration of blood for patch pleurodesis, the chest cavity should be suctioned free of any air or fluid.
- The blood for pleurodesis (5 mL/kg per hemithorax) should be administered within 2 to 3 minutes after collection to optimize blood distribution into the pleural space before clot formation.
- A small amount of saline may be used to flush the tube, but no more saline should be used than is necessary to ensure that all the blood is delivered into the pleural space. It is recommended that the clinician determine the volume of the administration set before beginning the procedure.
- After the blood is administered, it is recommended to gently, repeatedly (2 to 4 times), and safely roll the patient from sternal to both right and left lateral recumbency to help distribute the blood to all regions of the pleural space.
- Reaspiration of the pleural space or reconnection to a continuous suction device should be avoided for a minimum of 2 to 6 hours unless the patient's condition dictates otherwise.
- https://vetgirlontherun.com/videos/how-to-place-a-mila-thoracostomy-chest-tube-via-the-seldinger-technique-in-a-dog-vetgirl-veterinary-continuing-education-videos/
- https://todaysveterinarypractice.com/emergency-medicine-critical-care/blood-patch-pleurodesis-for-small-animal-pneumothorax/
- https://www.sciencedirect.com/science/article/pii/S1938973621000751

Thoracic Injuries

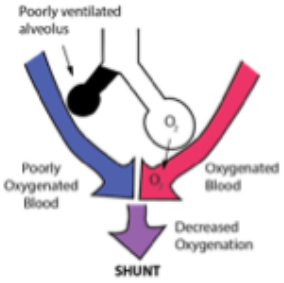
- Pulmonary contusions/hemorrhage
  - Direct or indirect injury within lung
  - Transmission of mechanical forces and energy to pulmonary parenchyma
  - Low density alveolar tissue stripped from heavier hilar structures as they accelerate at different rates
  - Hemorrhage → bronchospasm, increased mucous, alveolar collapse due to decreased surfactant production
  - Inflammatory response

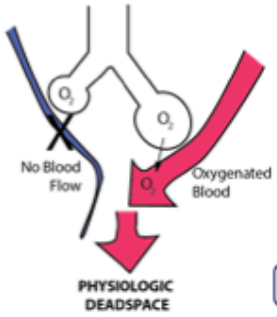


- Ok next is pulmonary contusions
- Pulmonary interstitial and alveolar hemorrhage and edema associated with blunt chest trauma, usually after compression-decompression injury of thoracic cage
  - Direct or indirect injury within lung
  - Transmission of mechanical forces and energy to pulmonary parenchyma
  - Low density alveolar tissue stripped from heavier hilar structures as they accelerate at different rates
  - Hemorrhage → bronchospasm, increased mucous, alveolar collapse due to decreased surfactant production
  - Inflammatory response

Thoracic Injuries

- Pulmonary contusions/hemorrhage
  - Ventilation perfusion mismatch
  - Decreased lung compliance due to edema/hemorrhage
  - Local hypoxia → hypoxic vasoconstriction → decreased perfusion of unventilated lung
  - Can take 24-48 hours to worsen
  - Resolve in 3-7 days unless bacterial pneumonia or ARDS

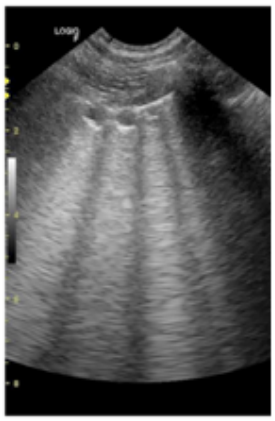


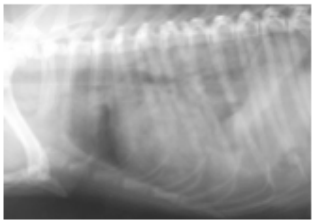


- Function changes
  - Thoracic trauma leads to blood within the alveoli, ventilation/perfusion mismatch, increased pulmonary shunt fraction, and loss of lung compliance.
  - Hypoxemia, increased work of breathing, and hypercarbia, are the physiologic results.
  - VQ mismatch when alveoli flooded
  - Increase in lung water from protein rich edema → decreased lung compliance
  - True shunt is major cause of hypoxemia
  - Local hypoxia → hypoxic vasoconstriction → vascular congestion and thrombosis
    - Can lead to decreased perfusion of unventilated lung
  - Resolve in 3-7 days but can also have delayed deterioration
    - Bacterial pneumonia, ARDS due to local or systemic inflammation
  - 48 hr healing has started and lymph vessels dilated and filled with protein
  - 7-10 days healed with little scaring

Thoracic Injuries

- Pulmonary contusions/hemorrhage
  - Diagnostics
    - CXR – changes may lag for 12-24 hours
    - CT – gold standard
    - POCUS
      - 6 B lines per ICS
      - More sensitive than radiographs





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- https://veteriankey.com/pulmonary-contusions-and-hemorrhage/
  - Dx
    - Worsen over 24 hr
    - Rads, POCUS, CT
      - 6 B lines per ICS for AIS;
      - better than rads
      - Rads may lag 12-24 hrs
      - If more severe changes, may need longer O2 and hosp
      - CT gold standard
    - Blood gas for hyoxemia – often mild to moderate

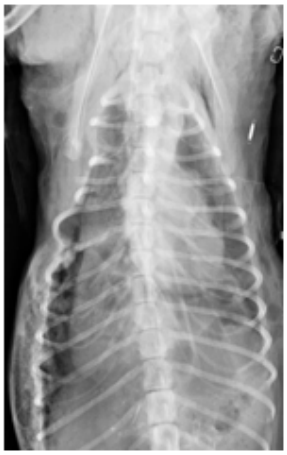
Thoracic Injuries

- Pulmonary contusions/hemorrhage
  - Treatment
    - Sternal position
    - O2 therapy
      - O2 kennel or nasal O2
      - High flow O2
      - Mechanical ventilation
    - Analgesia
    - Judicious fluid therapy
    - Often no antibiotic – low incidence of pneumonia
  - > 20% contused predictive for MV and ARDS
    - Also > 4 rib fractures

- Management
  - Sternal, O2, judicious fluid therapy, analgesia
  - High flow rate 0.4-2 L/kg/min
  - MV
  - Fluids
    - Often some hypovolemic shock
    - Increase in pulmonary capillary hydrostatic pressure may lead to extravasation
      - So could worsen hemorrhage
    - Blood products and synthetic colloids could also worsen and leak
  - Analgesia = minimal impairment of cardiac and resp functions
    - Intercostal, intrapleural, and epidural blocks can help too
  - Ab = low incidence of pneumonia so often not recommended
  - Little supportive data for steroids or furosemide
- Volume contused > 20% predictive for MV; also predictive for ARDS
  - Also more than 4 ribs fractures and GCS > 14

Thoracic Injuries

- Fractured ribs
  - Can cause direct lung injury
  - Hypoventilation due to decreased effective respiratory mechanics or pain
  - Cough reflex may be suppressed → increased risk for pneumonia?
  - Indication to look for other chest injuries: pneumothorax, contusions, hemothorax
  - Tx
    - Analgesia – systemic +/- local
    - May need MV for hypoventilation



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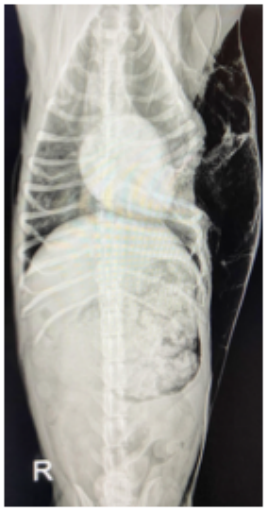
- Another common injury is Rib fractures
  - Can cause Direct lung injury, pain, hypoventilation due to decreased effective resp mechanics from flail chest segments
    - More specifically, the reduced chest wall motion and pulmonary expansion results in decreased oxygenation, ventilation, and atelectasis of the lungs
  - Cough reflex may also be suppressed
  - Tx =
    - pain management
    - May need MV for hypoventilation
    - prompting careful evaluation for additional injuries such as pulmonary contusions or a pneumothorax

Thoracic Injuries

- Flail chest
  - 3 or more rib fractures in series in which both dorsal and ventral aspect of ribs are fractured
  - Flail portions moves paradoxically inward during inspiration and outward during exhalation
  - May also have pneumothorax
  - Tx
    - Place in lateral recumbency with flail segment down to stabilize
    - Chest wrap?
    - If can't stabilize, may need surgical stabilization
  - https://youtu.be/q2AKiOYLbr8?feature=shared



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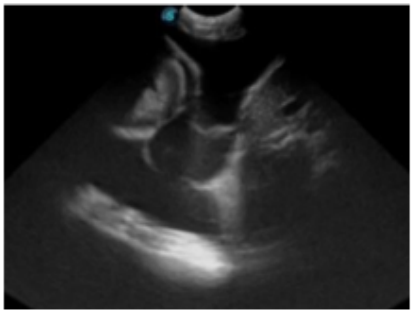
- A more serious injury is Flail chest = 3 or more rib fractures in series in which both dorsal and ventral aspect of ribs are fractured
  - Flail portion will paradoxically move inward during inspiration and outward during exhalation
    - On inspiration, the chest wall normally expands. With a flail segment, the negative intrapleural pressure causes the flail segment to collapse inward during inspiration. On expiration, the chest wall normally collapses. With a flail segment, the intrapleural pressure becomes less negative and the flail segment moves outward on expiration. Abnormal chest movement and the accompanying pain from the fractures themselves result in decreased oxygenation, ventilation, and pulmonary atelectasis
    - Penetrating chest wall injury may tear intercostal muscles with no or few associated rib fractures leading to paradoxical movement but not flail
  - Can get Crepitus and SC emphysema
  - More common in dogs than cats and more common with bite wounds
  - Pneumothorax common too
  - Tx
    - Pain control
    - Place in lateral recumbency with flail segment down to stabilize and prevent hypoventilation

        - Treatment consists of placing the patient in lateral recumbency with the flail side down, minimizing movement of the flail segment and reducing the associated fracture discomfort.
- Chest wrap
- If can’t stabilize with medical, thoracotomy indicated

Thoracic Injuries

- Hemothorax
  - Usually minimal
    - Lung laceration or laceration of pulmonary or intercostal vessel
  - If large amount then concern for rupture of large vessel
  - Diagnostics: POCUS, CXR
    - Thoracocentesis
      - PCV of fluid similar to peripheral blood
      - Therapeutic if respiratory compromise
  - Other tx
    - Resuscitation with fluids and blood products
    - Autotransfusion if needed



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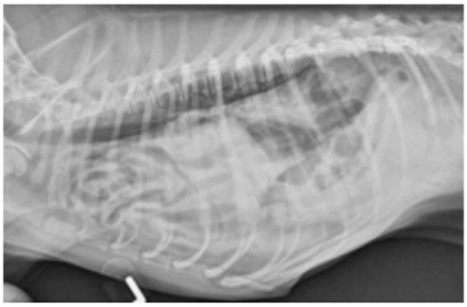
- Can also see hemothorax
  - If present, the amount of blood loss into the pleural space is usually minimal and does not contribute significantly to respiratory compromise.
  - If a large amount of hemorrhage into the pleural space is documented, there should be an increased suspicion for rupture of a large vessel.
  - More common causes for a hemodynamically insignificant hemothorax include laceration of pulmonary or intercostal vessels and/or lung laceration by a fractured rib.
- Diagnosis includes POCUS identification of pleural effusion and then thoracocentesis
  - PCV and TP of the effusion similar to that of the PCV and TP of the peripheral blood.
- Tx bleeding
  - therapeutic thoracocentesis,
  - intravenous crystalloid or synthetic colloid therapy and blood products, notably whole blood or packed red blood cell transfusions.
  - Autotransfusion can be considered if blood products are not available.

Thoracic Injuries

- Diaphragmatic hernia
  - Disruption of diaphragm allows displacement of abdominal organs into thoracic cavity
  - Intra-abdominal pressure suddenly increases causing rupture of diaphragm
  - Restriction of lung expansion and respiratory distress
  - Most common = liver, stomach, SI, spleen, omentum
  - Surgical correction
    - Immediate = stomach, strangulated bowel, inability to oxygenate or achieve CV stability, ruptured organ



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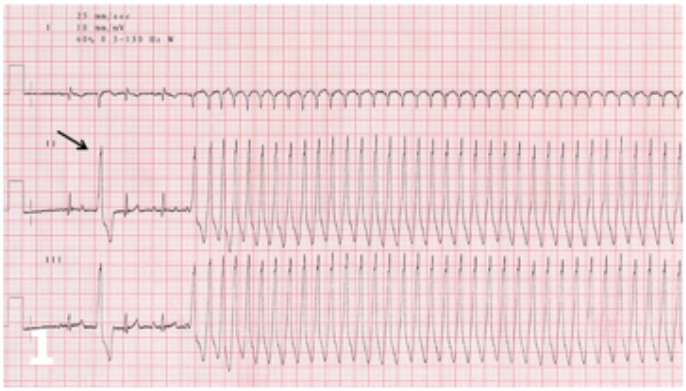
- And finally Diaphragmatic hernia
  - Diaphragmatic hernia is defined as disruption of the diaphragm, allowing displacement of abdominal organs into the thoracic cavity.
  - Diaphragmatic hernia occurs most often as a result of blunt trauma where intra-abdominal pressure is suddenly increased causing rupture of the diaphragm.
  - The resulting herniation of abdominal contents can range from a single organ or component of an organ (such as a single liver lobe) to almost all the abdominal contents moving cranially through the diaphragmatic rent into the chest cavity. The result is restriction of lung expansion and respiratory distress.
  - Most common organs = liver, stomach, SI, spleen, omentum
    - Rare = LI, GB, panc
  - Signs can be delayed
  - Treatment for diaphragmatic herniation will depend on the clinical signs of the patient with surgical repair being the definitive therapy.
    - Although there are no recent studies which outline the recommended time from stabilization to surgical correction, worsening respiratory distress or compromised blood supply to the displaced organs and ischemia would warrant a more rapid surgical correction.
    - Immediate sx indications = herniated stomach, strangulated bowel, inability to oxygenate properly or achieve CV stability,

ruptured viscera

Thoracic Injuries

- Cardiac injuries
  - Myocardial rupture
  - Pericardial laceration
  - Septal injury
  - Valve rupture
  - Myocardial contusion
  - Commotio cordis



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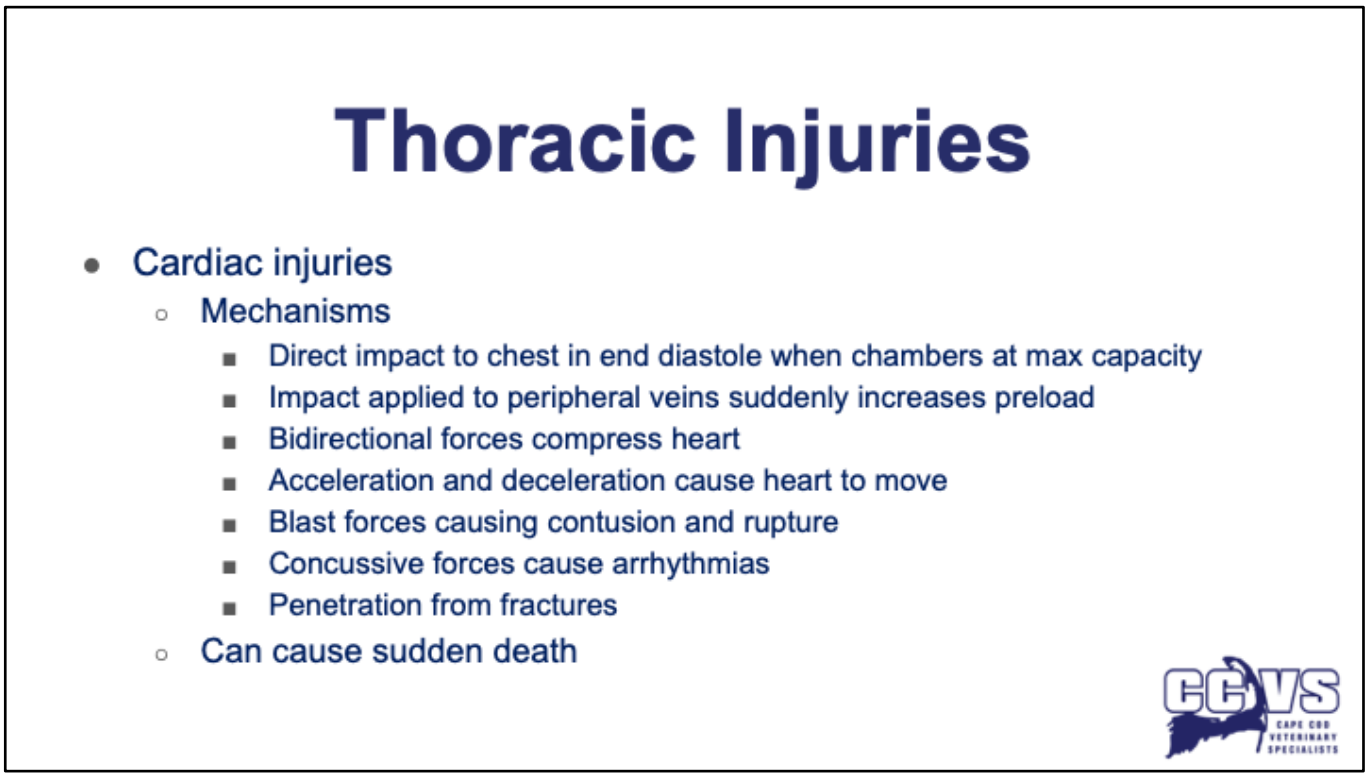
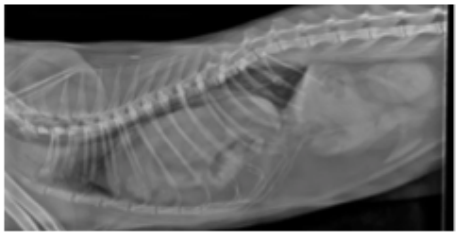
- https://www.cliniciansbrief.com/article/ventricular-tachycardia



- 7 mechanisms behind cardiac injuries and how they manifest clinically
  - Direct impact to chest in end diastole during which ventricles are at max capacity or at end systolic during which atria are at max capacity
  - Suddenly increased cardiac preload secondary to increased venous return due to impact applied to peripheral or abdominal veins
  - Bidirectional forces that compress heart within thoracic cage
  - Forces of acceleration and deceleration that cause heart to move, leading to myocardial damage/rupture and/or damage to great vessels and/or coronary arteries
  - Blast forces leading to cardiac contusion or rupture
  - Concussive forces leading to arrhythmias
  - Cardiac penetration by fractures
- Can result in sudden death

Thoracic Injuries

- Cardiac injuries
  - Myocardial rupture
    - Laceration of atrial wall, ventricular wall, or papillary muscle
    - Can lead to PCE, cardiac tamponade, and sudden death
  - Pericardial laceration
    - Can be aclinical
    - Can cause herniations of cardiac structures with strangulation
    - Can cause hemothorax
    - PPDH
    - Pleuropericardium – herniation of heart into pleural space



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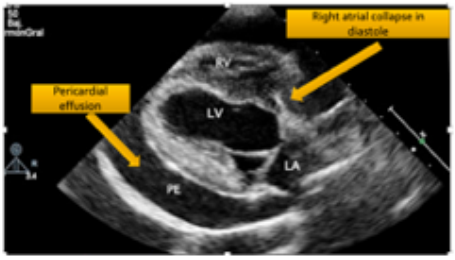
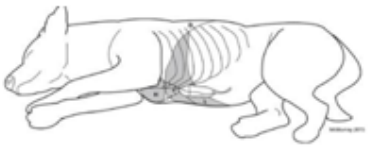
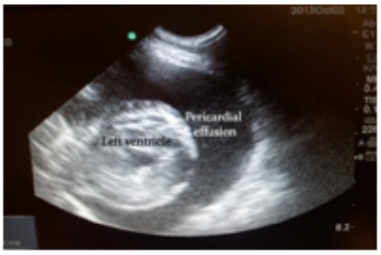
- First is Myocardial rupture
  - Laceration of atrial or ventricular walls or papillary muscles
  - Rare
  - Can get hemopericardium → sudden death
  - Can lead to obstructive shock due to cardiac tamponade
- Pericardial laceration
  - Can be clinically silent or result in herniations of cardiac structures with strangulation
  - Or hemothorax
  - Pericardial rupture can occur at diaphragm causing traumatic peritoneaopericardial diaphragmatic hernia or it can occur into pleuropericardium which can cause herniation of heart into pleural space

Thoracic Injuries

- Cardiac injuries
  - Cardiac POCUS
    - Subxiphoid vs right parasternal
    - Pericardial effusion
      - Assess for collapse of right side of heart = tamponade





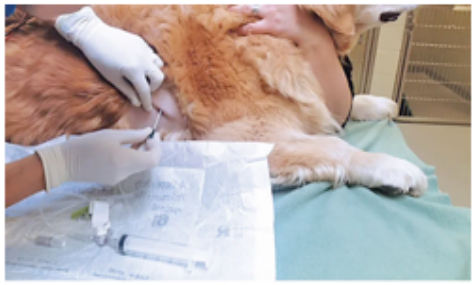


- POCUS
  - One of the best places to identify pericardial effusion is the subxiphoid view. The probe is placed in long axis in the subxiphoid and angled in a more parallel fashion to the dog. By rocking the probe until it is parallel to the patient, the ventral region of the thoracic cavity is visible
  - By getting comfortable with the different heart chambers via the right parasternal short axis approach it is possible to differentiate pericardial fluid from cardiac chambers and/or pleural effusion.
  - It is important to scan the heart from apex to base in short axis to make sure heart chambers are differentiated from pericardial effusion. Identify the chambers in short axis described above (mushroom view so that you can identify the left and right ventricles, LA:Ao view etc.).
  - If these structures are identified it then becomes apparent when pericardial effusion is present; a circular collection of fluid surrounding the heart contained within the pericardial sac
- By rotating the probe further, a 4-chamber view of the heart can be obtained; this can be useful to visualize cardiac tamponade (and pulmonary hypertension via increased right atrial and ventricular size)
- Once identify PCE, don't forget to assess for cardiac tamponade
  - This indicates emergent tap needed ASAP
- https://vetgirlontherun.com/videos/how-to-perform-a-pericardiocentesis-in-a-dog-vetgirl-veterinary-continuing-education-videos/
- https://www.cliniciansbrief.com/article/pericardiocentesis

- https://vetemcrit.com/determination-of-the-intravascular-volume-status-with-point-of-care-ultrasound/
- https://www.canadianveterinarians.net/media/ltglcqsz/pocus-09-2022-lab-manual.pdf

Thoracic Injuries

- Cardiac injuries
  - Pericardiocentesis
    - IV access
    - Light sedation, lidocaine block
    - Sterile prep
    - ECG
      - Prepare emergency lidocaine dose
    - 16 gauge catheter or centesis catheter
    - 3 way stopcock, syringe, extension lines
    - Right side vs left side, 4-6th rib
      - Cardiac notch, coronary vessels
    - Assess for nonclotting blood
    - IV fluid bolus afterwards if indicated



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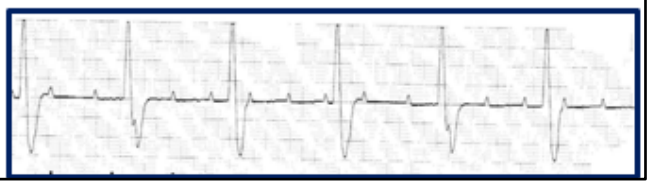
- I've included info on how to perform pericardiocentesis
  - Often need Sedation, IV
  - Local block with lidocaine = 0.5-1 mL = skin, intercostal muscle and pleura
  - R side approach = use cardiac notch so less trauma to lung, avoid major coronary vessels which are on L
  - L lateral recumbency or sternal
  - Sterile prep 3-7th rib from sternum to costochondral junction
  - Palpate for cardiac impulse between 4-6th rib just lateral to sternum
  - Cranial to rib
  - Angle towards patient's left shoulder
  - Pericardial fluid does not clot unless associated with very recent hemorrhage – which could occur in our trauma patients so don't panic if it clots
  - Complications
    - Direct myocardial injury or puncture
    - Arrhythmia = a fib, v tach
    - Coronary artery lacertation
    - Lung laceration = pneumothorax, pulmonary hemorrhage
  - And then when you are done, continue to monitor for re-effusion and may start giving resuscitation fluids
- https://vetgirlontherun.com/videos/how-to-perform-a-pericardiocentesis-in-a-dog-vetgirl-veterinary-continuing-education-videos/

- https://www.cliniciansbrief.com/article/pericardiocentesis
- https://vetemcrit.com/determination-of-the-intravascular-volume-status-with-point-of-care-ultrasound/

Thoracic Injuries

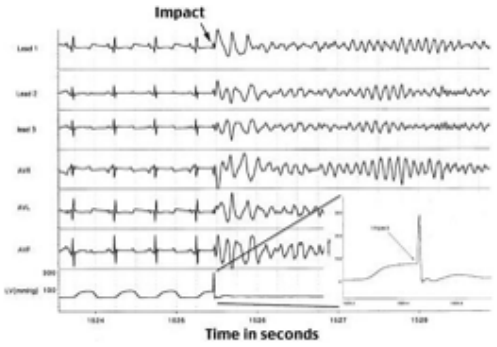
- Cardiac injuries
  - Septal injury
    - Can cause acquired intracardiac shunting → CHF
    - If damage to basilar interventricular septum, damage to AV bundle → AV block
  - Valve rupture
    - Most susceptible during time of closure
    - Rupture leads to sudden valve regurge → CHF
      - Acute or over several days



- Other injuries we may not be able to assess on triage directly include septal injury or valve rupture
- Septal injury
  - Can be delayed due to myocardial inflammation post trauma
  - Can lead to acquired intracardiac shunting lesions → CHF
  - Also since basilar interventricular septum is in location of AV bundle, septal injury can also lead to AV block
- Valve rupture
  - Most susceptible to injury during time of closure
    - Semilunar valves during diastole
    - AV valves during systole
  - Can cause papillary muscle, valve leaflet, or chordae tendineae rupture leading to sudden valve regurge which may lead to poor CO and CHF
  - Papillary muscle rupture will result in fulminant valvular regurge and likely acute CHF
  - But tear of valve leaflet or chordae tendinea may result in more insidious development of CHF over several days

Thoracic Injuries

- Cardiac injuries
  - Myocardial contusion
    - Hemorrhagic, necrotized, mixed
    - Arrhythmic effect = induction of electrically silent region of myocardium → reentrant circuit → ventricular tachyarrhythmia
    - Can cause regional myocardial wall motion abnormalities
      - Can lead to cardiogenic shock
  - Commotio cordis
    - Blunt impact during ventricular repolarization within 15-30 msec before peak of T wave
    - Leads to RonT → ventricular fibrillation → death



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- Myocardial contusion
  - Also called cardiac concussion
  - Myocardial bruising
  - Forms
    - Hemorrhagic = extravasation of blood without muscle fiber disruption
    - Necrotized = coagulation necrosis and/or contraction band necrosis of muscle fibers
    - Mixed = both
  - Arrhythmic effect proportional to kinetic energy applied to surface of myocardium
  - MOA of arrhythmia = induction of electrically silent region of myocardium which leads to initiation of reentrant circuit around this silent region → tachyarrhythmia most common ventricular
  - Can see regional myocardial wall motion abnormalities, may be clinically silent but can lead to cardiac decompensation and/or cardiogenic shock if excessive fluid resuscitation
  - The definitive Dx is histopath
- Commotio cordis = sudden cardiac death
  - Blunt impact to precordium during ventricular repolarization within 15-30 msec before peak of T wave,
  - leading to RonT which degenerates to V fib and death with no

histopath changes
- This was the condition in the news regarding the football player that arrested during the game so people more familiar with it now - Damar Hamlin 2023 NFL
- But when have a trauma patient that dies on impact this is always a possible cause

So what do I do?

- Diagnostics
  - PE, auscultation
  - POCUS
  - ECG
  - CXR
  - Blood gas if possible

- So to summarize everything into practical steps
- To assess for thoracic traumatic injuries
  - Don't forget about auscultation – that can give clues
  - POCUS to check for fluid and glide sign
  - ECG to assess for arrhythmias secondary to shock or cardiac trauma
  - CXR when stable enough
  - And for blood, a blood gas ca be helpful to assess for hypoventilation or hypoxia

So what do I do?

- Treatment
  - Analgesia
  - Antibiotic if penetrating injury
  - O2 therapy
    - Flow-by vs nasal cannulas vs intubation and PPV
  - Flail side down vs sternal
  - Thoracocentesis
    - Thoracostomy tube if needed +/- continuous suction
      - MILA
      - Red rubber
  - Pericardiocentesis if tamponade
  - Thoracotomy

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- Initial treatments you should consider
  - Pain meds – ideally opioid, understanding have to be careful about resp depression
    - Fentanyl can be titrated
    - And if worried about possible arrest have that naloxone pulled up and ready
  - Of course O2 therapy
    - Flow by
    - Nasal cannulas or high flow
    - And if severe then may need to intubate and put on 100% O2
      - If concern for pneumo then may need to be trying to tap at the same time and consider chest tube bc PPV may make it worse
      - May need to blind tap at the highest point of the chest
  - And finally if concern for PCE and tamponade then need to do pericardiocentesis
  - Ultimately you may need a thoracotomy but have to keep them alive to get there

Abdominal Injuries

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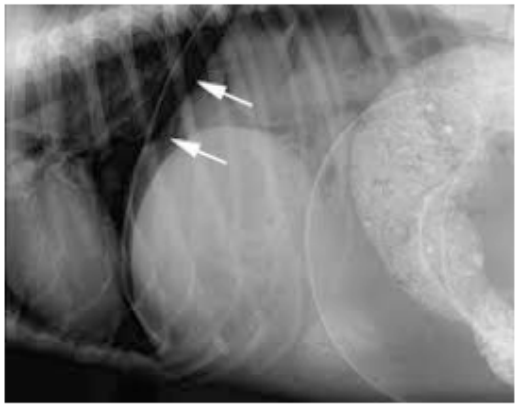
Abdominal Injuries

- Hemoabdomen
  - Liver, spleen
  - Major vessels
  - Retroperitoneal hemorrhage - Kidney
- Uroabdomen
  - Bladder rupture, ureter rupture
- Septic abdomen
  - GI tract rupture
  - Penetrating wounds
- Bile peritonitis
  - Gall bladder rupture
- Evisceration



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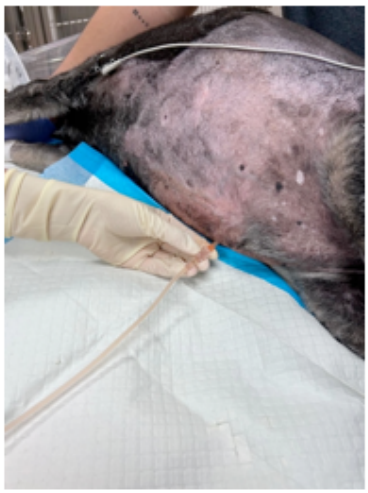
- For abdominal injuries, Injury may depend on where impact occurs, underlying conditions (mucocele, UTI)
  - Also can have more than one!
- Well talk about diagnostics to consider and each injury
- file:///Users/StephanieKline/Downloads/javma-javma.230.4.505.pdf

Abdominal Injuries

- Diagnostics
  - POCUS
    - Free fluid
    - Free gas
    - Intact gall bladder
    - Intact bladder
  - AXR
    - Free gas
  - CT with contrast
  - Abdominocentesis



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- OK starting with diagnostics
  - To assess if there is injury and what exactly it is, there are several diagnostics to consider
- POCUS is probably the most helpful – can ID free fluid or gas and assess intact walls (bladder, GB)
  - And then pairing with abdominocentesis can often get you a diagnosis
- But AXR can be helpful at catching free gas missed on POCUS so I often do a lateral AXR minimum when they are stable enough
- And finally CT can be used to assess multiple injuries, cavities, orthopedic injuries, where bleeding coming from, etc
  - But since requires GA, patient may or may not be stable enough for CT – may have to go to surgery without; sorry surgeons

Abdominal Injuries

- Diagnostics
  - POCUS
    - 5 locations
      - Subxiphoid (diphragmatico-hepatic)
      - Umbilical
      - Urinary bladder (cysto-colic)
      - Right paralumbar (hepato-renal)
      - Left paralumbar (spleno-renal)
    - Free fluid
    - Free gas

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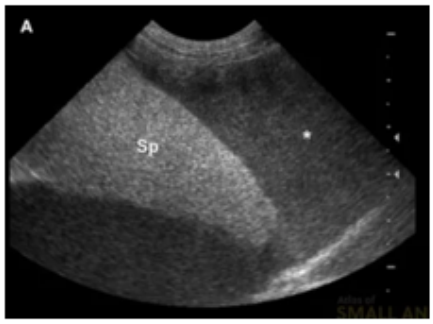
- Quick info on abdominal POCUS
- there are 5 regions to assess
  - At each site, the probe is fanned and rocked through an angle of 45° in both long and short axis views.
  - Sliding the probe 1 inch in cranial, caudal, left, and right directions will increase the area assessed at each site.
- Fanning, rocking and sliding the probe increases the likelihood that abdominal fluid will be detected
- https://www.canadianveterinarians.net/media/ltglcqsz/pocus-09-2022-lab-manual.pdf

Abdominal Injuries

- Diagnostics
  - POCUS
    - Free fluid
      - Check all 5 locations, focusing on gravity dependent regions
      - Very cellular effusions can mimic soft tissue structures
        - Try altering pressure to create "swirling"
      - Fluid within organ cavities can be mistaken for free fluid
        - Often GI contents



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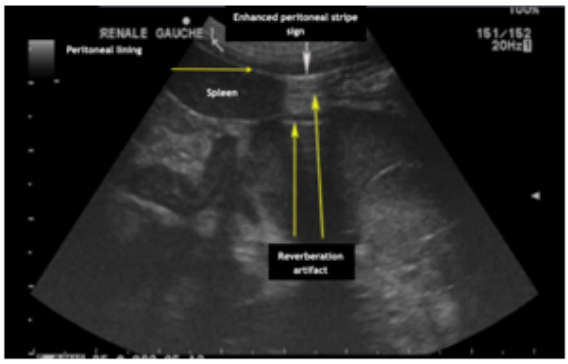
- At all sites check for fluid
- A few things to remember about assessing FF
  - Highly cellular effusions can look like organs
  - Try to create swirl if unsure
  - Fluid within organ cavities can be mistaken for FF, often occurs with stomach and intestines

- https://smallanimalultrasonography.com/echogenic-peritoneal-effusions-in-dogs-and-cats-2/

Abdominal Injuries

- Diagnostics
  - POCUS
    - Free gas = enhanced peritoneal stripe sign
      - Lateral for few minutes to allow air to rise
      - Find peritoneal lining
      - Reverberation artifact that originates at peritoneal lining



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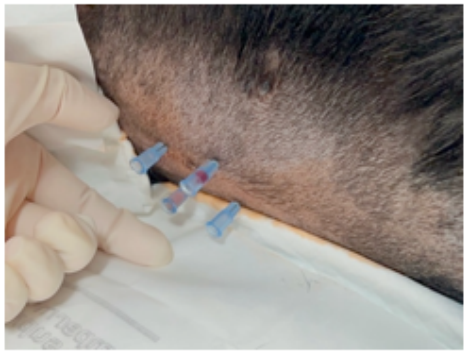
- POCUS can also be used to identify free gas
  - First The peritoneal lining must be identified.
    - This is essential so as not to confuse free air within the GI tract for free air in the abdomen.
    - Identifying the peritoneal lining can be achieved by placing the patient in lateral recumbency and identifying structures in contact with the peritoneal lining of the non-gravity dependent body wall, such as the stomach, liver or spleen.
  - Leaving the animal in lateral for a few minutes to allow air to rise to the non-gravity dependent body wall is recommended.
  - Identify the presence of reverberation artifact that originates at the peritoneal lining.
    - This is very important to differentiate from reverberation artifact contained within the GI tract, which again, emphasizes the importance of clearly identifying the peritoneal lining.
- This is the enhanced peritoneal stripe sign. This sonographic finding occurs when free abdominal air comes in contact with the peritoneal lining. At the point where free abdominal air comes in contact with the peritoneal lining it will cause the peritoneal lining to become more hyper-echoic. This is the enhanced peritoneal lining.
- https://www.canadianveterinarians.net/media/ltglcqsz/pocus-09-2022-lab-manual.pdf

Abdominal Injuries

- Abdominocentesis
  - US guided
  - 4 quadrant approach = 4 needles placed simultaneously
  - Hemoabdomen
    - PCV/TP equal to or exceeds peripheral
    - Can be diluted by urine
  - Uroabdomen
    - Potassium > 1.4:1 peripheral
    - CRE > 2:1 peripheral
  - Septic abdomen
    - Intracellular bacteria and degenerate neutrophils
    - BG < 20 mg/dL:1 (38 mg/dL on POC) difference from peripheral
    - Lactate > 2:1 mmol/L difference from peripheral
  - Gall bladder rupture
    - Bile pigments
    - Bilirubin > peripheral



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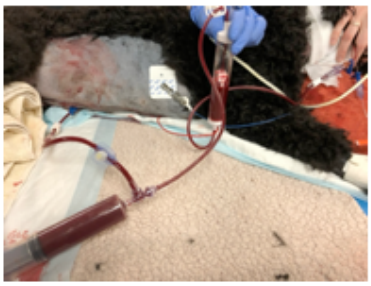
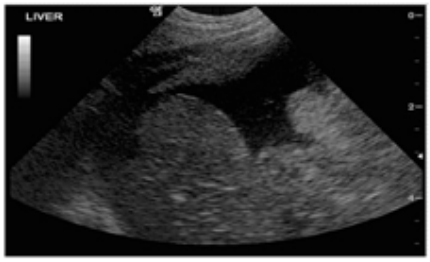
- If you do find free fluid, strongly recommend sampling it as there can be several causes in trauma cases
  - If don't have US then can use the 4 quadrant tap technique to get any fluid that is in the abdomen
- For hemoabdomen, the PCV of the fluid has to be = to or exceed peripheral
  - If also uroabdomen then may be falsely diluted
- For uroabdomen check K and CRE
    - Potassium > 1.4:1 peripheral
    - CRE > 2:1 peripheral
- For septic abdomen, perform cytology to look for degenerate neutrophils and intracellular bacteria
  - You can also look at BG and lactate
  - And use difference greater than or equal to 38 mg/dL for better specificity
    - Ok sensitivity for 20 mg/dL difference but
    - When using a lab analyzer, a glucose difference of > 20 mg/dL between peripheral blood and peritoneal effusion is diagnostic for septic peritonitis. Using a POC glucometer in place of the lab analyzer can provide 100% specificity only if the cutoff is raised to 38 mg/dL.
- For gall bladder rupture look for bile pigments or bilirubin of the fluid to be greater than peripheral
- https://www.cliniciansbrief.com/article/abdominocentesis-procedure-centesis-

lavage

Abdominal Injuries

- Hemoabdomen
  - Resuscitation with blood products
  - Autotransfusion if needed
  - Antifibrinolytic dose
  - Belly wrap?
  - Damage control surgery vs exploratory laparotomy





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- Hemoabdomen – mainly need to resuscitate and if needed try to stop bleeding
  - Prefer blood products
  - Can do autotransfusion if needed
  - Single antifibrinolytic dose
  - Debate on belly wrap efficacy
- These cases are often prime candidates for damage control surgery if you can't stabilize – we'll talk about this approach in a bit

Abdominal Injuries

- Uroabdomen
  - Manage hyperkalemia

TABLE 1 The Effects of Serum Potassium Concentration on Electrocardiogram[6,12,13]

SERUM POTASSIUM CONCENTRATION, MMOL/L	ELECTROCARDIOGRAM ABNORMALITY
≥5.5–6.5	Increased T-wave amplitude
≥6.6–7	Decreased R-wave amplitude, prolonged QRS and QPR intervals, ST segment depression
≥7.1–8.5*	Decreased P-wave amplitude, increased P-wave duration, prolongation of QT interval
≥8.6–10	Lack of P waves (atrial standstill), sinoventricular rhythm
≥10.1	Widened and biphasic QRS complex, ventricular flutter, fibrillation, or asystole

**Consider additional therapy for serum potassium ≥7.5 mmol/L.*
Modified from Stafford JR, Bartges JW. A clinical review of pathophysiology, diagnosis, and treatment of uroabdomen in the dog and cat. J Vet Emerg Crit Care (San Antonio). 2013;23(2):217-229. doi:10.1111/vec.12033

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- Ok now for uroabdomen
- Patients with uroabdomen accumulate potassium-containing urine in the abdomen, which is then resorbed into the systemic circulation, resulting in hyperkalemia.
- So hyperkalemia becomes the main focus of stabilization
  - Main sequelae is cardiac arrhythmias ultimately leading to atrial standstill, ventricular flutter, fib or asystole
  - Increased risk for arrhythmias with increasing potassium
- https://todaysveterinarypractice.com/emergency-medicine-critical-care/uroabdomen-approach-and-management/

Abdominal Injuries

- Uroabdomen
  - Manage hyperkalemia

TABLE 2 Treatment of Severe Hyperkalemia[6]

DRUG	DOSE[1]	NOTES
Insulin and 50% dextrose	▪ 0.5 units/kg regular insulin IV ▪ 2 g (4 mL) of 50% dextrose (diluted) for every unit of insulin administered	
50% dextrose alone	▪ 0.7–1 g/kg (1.4–2 mL/kg) IV diluted slowly q3–5min	Not recommended in humans[14]
10% calcium gluconate	▪ 0.5–1.5 mL/kg IV slowly q5–10min; monitor with electrocardiogram for bradycardia or exacerbation of arrhythmias	Does not lower serum potassium concentration; antagonizes the effect of hyperkalemia on the myocardium
Sodium bicarbonate	▪ 1–2 mEq/kg IV slowly over 15 minutes	May be more effective if given with other treatments for hyperkalemia

Modified from Stafford JR, Bartges JW. A clinical review of pathophysiology, diagnosis, and treatment of uroabdomen in the dog and cat. J Vet Emerg Crit Care (San Antonio). *2013;23(2):217-229. doi:10.1111/vec.12033*

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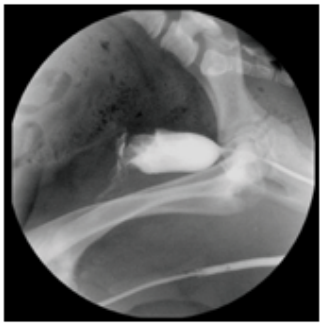
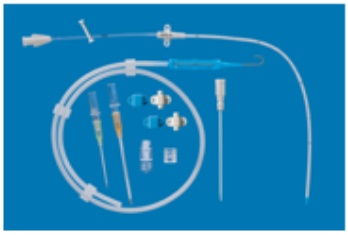
- If hyperkalemic and arrhythmias, then need to intervene
  - Calcium gluconate
    - Increases threshold potential → helps protect heart from hyperkalemia effects
    - Administer slowly IV
    - Monitor ECG when giving
  - Translocate K into cells
    - Dextrose
    - Insulin
    - Terbutaline
    - Sodium bicarbonate
- https://todaysveterinarypractice.com/emergency-medicine-critical-care/uroabdomen-approach-and-management/

Abdominal Injuries

- Uroabdomen
  - Manage hyperkalemia
  - Analgesia!
  - Sterile placement of urinary catheter
  - If still significant effusion or unable to stabilize K or azotemia, place abdominal drain
  - Urosepsis → culture sample, start antibiotic
  - When stable, may need cystourethrogram to identify location of leak before surgery





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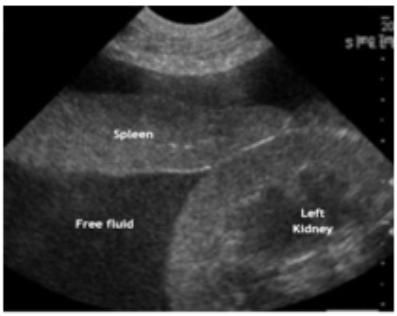
- Additional steps including analgesia (these are some of the most painful patients I've seen) and placing urinary catheter to keep bladder small and hopefully decrease leaking into peritoneum
- In some patients, urinary catheters alone do not fully drain the peritoneal cavity, and azotemia and electrolyte abnormalities do not normalize.
- In these patients, the addition of abdominal drainage to urinary catheterization can help further remove urine from the peritoneal cavity
- an indwelling drain allows for continuous drainage and often expedites stabilization of the patient.
    - Any type of long catheter or tubing can be used as an abdominal drain; however, omentum can easily obstruct many types of drains.
    - Effective options for abdominal drains include a commercially available abdominal drain with blunt insertion trocar, peritoneal dialysis catheters, active suction Jackson-Pratt drains, and guidewire-inserted thoracostomy tubes.
    - The MILA is the preference because it is effective, is simple to place, and can be placed with local anesthetic and minimal systemic sedation
- If concern for urosepsis then start antibiotic – but this is rare
- Once stable then may need to do more diagnostics to determine if surgery is needed
    - Cystourethrography is the imaging modality that provides the most practical information about the site of leakage in the bladder and/or urethra.
        - A large Foley urinary catheter is aseptically placed, and a 10% to

20% solution of water-soluble, organic, iodinated contrast medium (e.g., iohexol) is infused into the bladder (~10 mL/kg) until resistance is met.
    - Postcontrast radiographs are taken, or the procedure can be performed with fluoroscopy.
    - If a leak is not identified within the bladder, the catheter can be withdrawn while injecting contrast to highlight the urethra (10 to 15 mL for dogs, 5 to 10 mL for cats). A lateral radiograph is performed toward the end of the infusion to assess for leakage.
- https://todaysveterinarypractice.com/emergency-medicine-critical-care/uroabdomen-approach-and-management/

Abdominal Injuries

- Septic abdomen
  - Penetrating injuries
  - GI tract rupture
  - Urosepsis
  - Confirm on abdominocentesis, culture sample
  - Start antibiotics
  - Damage control surgery vs exploratory laparotomy



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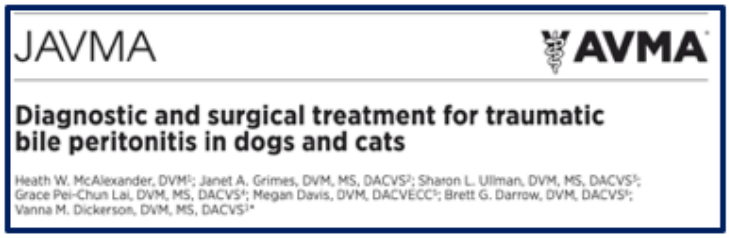
- Another common abdominal injury we see is septic abdomen
  - Most common GI tract rupture or penetrating injury, less common urosepsis
  - When confirm on cytology then save culture sample
  - Start antibiotics – often broad spectrum if unstable, I typically start with potentiated pencillin like unasyn and fluroquinolone (enrofloxacin)
    - Can de-escalate after culture result and stabilize
- In addition to hemoabdomen patients, these patients may also need a damage control surgery approach

Abdominal Injuries

- Bile peritonitis
  - Assess GB wall on POCUS
  - Abdominocentesis → elevated bilirubin 2:1, bile pigments, crystals
  - Analgesia!
  - +/- antibiotics
  - Exploratory laparotomy
  - Can present weeks later



- For patients you dx with GB rupture
  - You want to do analgesia
  - Possibly antibiotics
  - And then ex lap when stable
  - Important to remember these cases can present delayed as it may take time for the peritonitis to develop
- (2024). Diagnostic and surgical treatment for traumatic bile peritonitis in dogs and cats. *Journal of the American Veterinary Medical Association*,
  - 13 dogs, 4 cats
  - vehicular trauma and falls were the most common types of trauma precipitating biliary rupture.
  - Serum total bilirubin was not elevated in all animals despite confirmed biliary tree rupture, highlighting the importance of abdominocentesis in animals with peritoneal effusion following trauma; often 2:1 ratio
  - Also bile pigments. The presence of bile crystals or mucinous material
  - The median ratio of peritoneal bilirubin to peripheral bilirubin was 5.4 (range, 1.3 to 22.7
  - Bacterial culture of the abdominal cavity was performed in 8 dogs; 2 dogs had positive cultures, with one dog having a multidrug–resistant *Staphylococcus pseudintermedius* and the other an *Escherichia coli.* The dog that cultured positive for *E coli* had a previously undiagnosed gallbladder mucocele at the time of traumatic gallbladder rupture.
  - Animals with traumatic bile peritonitis may be asymptomatic for several

weeks after the injury.

Abdominal Injuries

- Evisceration
  - Hemodynamic stabilization
  - Antibiotics
  - Apply sterile dressing until surgery
  - +/- Extension of abdominal rent to prevent vascular compromise of eviscerated organs
  - E collar to prevent autocannibalism
  - Requires surgical intervention – damage control vs exploratory laparotomy

- And finally for patients with evisceration
  - These patients will likely also need quite a bit of stabilization
  - Definitely want to start antibiotics
  - And ideally flush off and sterile dressing
    - May need to extend rent if strangulation occurring
  - E collar so animals don't do more damage
  - And then may need damage control surgery vs ex lap – but will need surgery of course
- (2009). Major abdominal evisceration injuries in dogs and cats: 12 cases (1998–2008). Journal of the American Veterinary Medical Association
  - exposure and contamination of the abdominal viscera warrants immediate surgical intervention.
  - All animals lived
  - Major abdominal evisceration injuries can be devastating injuries, but prompt medical attention and surgical intervention may be associated with a relatively good prognosis. Animals should not be euthanatized solely on the basis of the visually shocking nature of the injury.
  - Prompt emergency care (including hemodynamic stabilization, antimicrobial treatment, and application of a sterile dressing) and surgical exploration to determine viability of abdominal organs, lavage of abdominal contents with copious amounts of fluids, reduction of eviscerated organs, and repair of defects are principles of treatment.

Abdominal Injuries

- Damage control surgery
  - Control of abdominal bleeding
  - Emergency control of abdominal contamination from bowel, biliary and urinary tract
  - Definitive repair of bowel or visceral injuries is not attempted
  - Definitive surgical repair of damaged tissues is delayed until after the patient has stabilized
  - Which patients?
    - Hemodynamic instability despite resuscitation
    - Penetrating trauma and/or complex major vascular injuries
  - Prepare owners!

- Damage control surgery is aimed at this fragile population of (mostly) trauma patients who need a modified approach to standard techniques and planning in order to maximize their chance of survival.
- The term *damage control* is borrowed from the US navy's experience with keeping damaged ships afloat with temporary measures long enough for them to get to port where definitive repair can be undertaken
  - entails a first surgery which is aimed at restoring normal physiology but not at correcting all problems.
  - This is followed by a few days of aggressive support to strengthen the patient so they can withstand a longer surgical procedure for definitive repair of injuries that were not addressed during the first damage control surgery.
- the practice has been refined to now involve the aggressive resuscitation using blood products and limited crystalloid fluid in the period between the two surgeries.
- Goal is stabilize and get out!
- Which patients?
  - Penetrating trauma and/or complex major vascular injuries
  - Hemodynamic instability
  - Systolic arterial blood pressure < 70 mm Hg
  - Persistent tachycardia or dysrhythmias
  - Coagulopathy – evidence of acute traumatic coagulopathy
  - Hypothermia

    - Metabolic acidosis with pH < 7.2
    - Inability to control bleeding, despite massive transfusion therapy
- Damage control surgery is broken down into four phases.
    - Phase 1 is the preparation of the patient for surgery by limiting hemorrhage, managing hypothermia, offering transfusions of blood and plasma to limit coagulopathy and promptly getting them into the operating room.
    - Phase 2 is the first surgery where the primary goal is to limit severe hemorrhage and control bowel leakage. Hemorrhage is limited largely by packing the abdomen with lap pads and anastomosing larger torn vessels when appropriate. Urinary diversion can be performed with indwelling urinary catheters if the urinary tract is disrupted. Tears of the bowel are closed either with sutures or stapling equipment. Severely damaged bowel is resected leaving only well perfused bowel. No attempt at this time is made to reconnect discontinuous sections of bowel – that would be done at the second definitive surgery. The abdomen is then left open with a temporary closure or barrier to limit contamination. This phase is often limited to 2 hours.
    - Phase 3 is performed in the ICU for 12-48 hour to resolve the three components of the lethal triad (acidosis, hypothermia and coagulopathy). This is a period of aggressive resuscitation with blood products, continued warming and reversal of coagulopathy. Oxygenation is maximized with supplementation or ventilation in most cases. Overhydration with aggressive crystalloid fluids is avoided since this can result in tissue edema making abdominal closure difficult and exacerbating coagulopathy and acidosis.
        - May need to keep under heavy sedation or on ventilator during recovery or until next surgery can be performed
    - Phase 4 is the definitive repair of the abdominal trauma. This phase does sometimes require more than one surgery
        - This is important to prepare owners for
- https://mycpd.veteducation.com/blog/damage-control-surgery-management-of-traumatic-haemoabdomen-in-dogs-and-cats/
- https://www.mspca.org/angell_services/damage-control-surgery-is-there-a-role-in-veterinary-medicine/

- Ok so here is a summary about interventions for each type of abdominal trauma cases
- A lot of interventions pretty straight forward, hardest part is deciding when to go to surgery
- Indications for emergency surgical intervention include:
    - Inability to medically stabilize intrabdominal hemorrhage
    - Free gas on abdominal radiographs (provided they were taken prior to abdominocentesis and there is no recent abdominal surgery)
    - Cytological evidence of intracellular bacteria or plant/food material in the abdominal fluid
    - Elevated creatinine and potassium levels compared to peripheral serum levels
    - Elevated bilirubin levels higher compared to peripheral serum levels
    - Penetrating abdominal injury
- *How soon should surgery be performed once the diagnosis is made?*
    - This depends on two factors:
        - How stable is the patient?
        - What is the underlying diagnosis?
- Clinical judgment is needed to determine the appropriate balance between stabilization before surgery and the time that passes before the patient is placed under anesthesia to surgically correct the problem.

Traumatic Brain Injury

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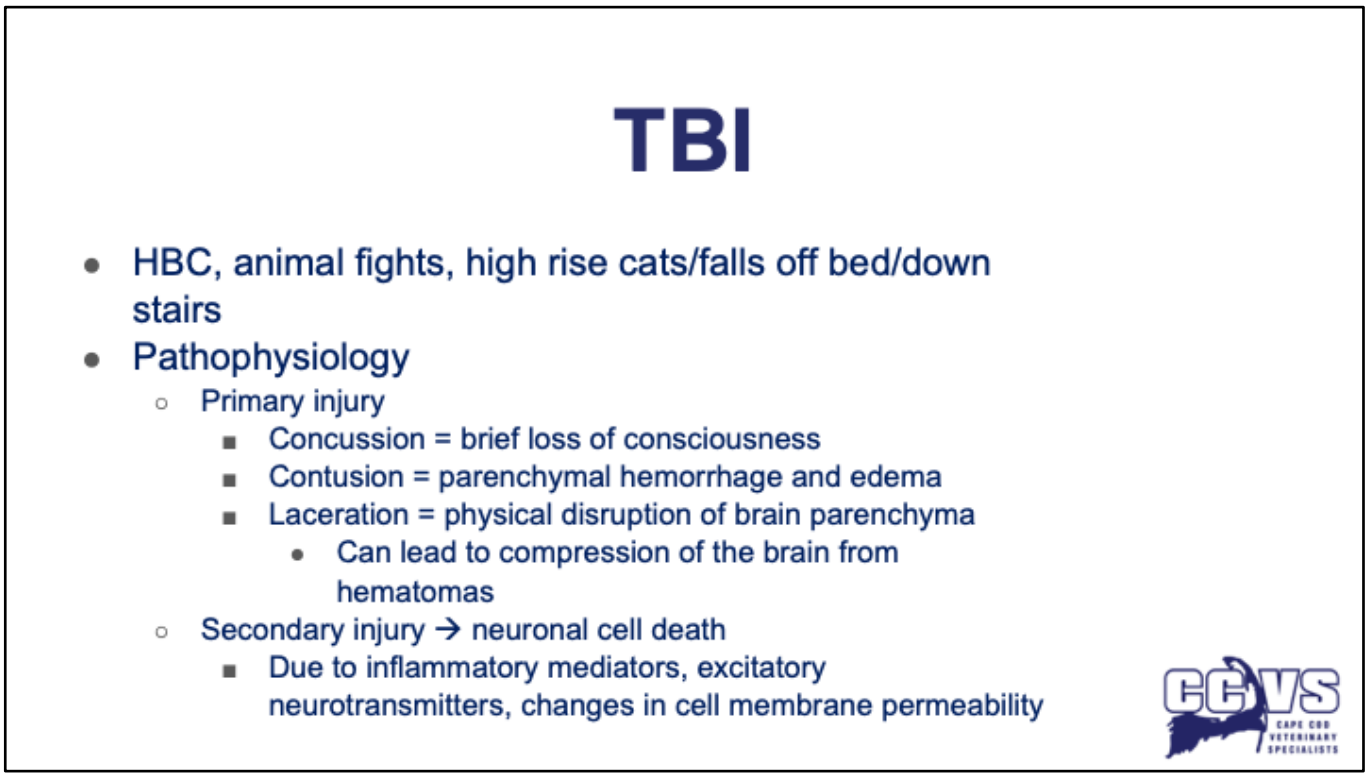
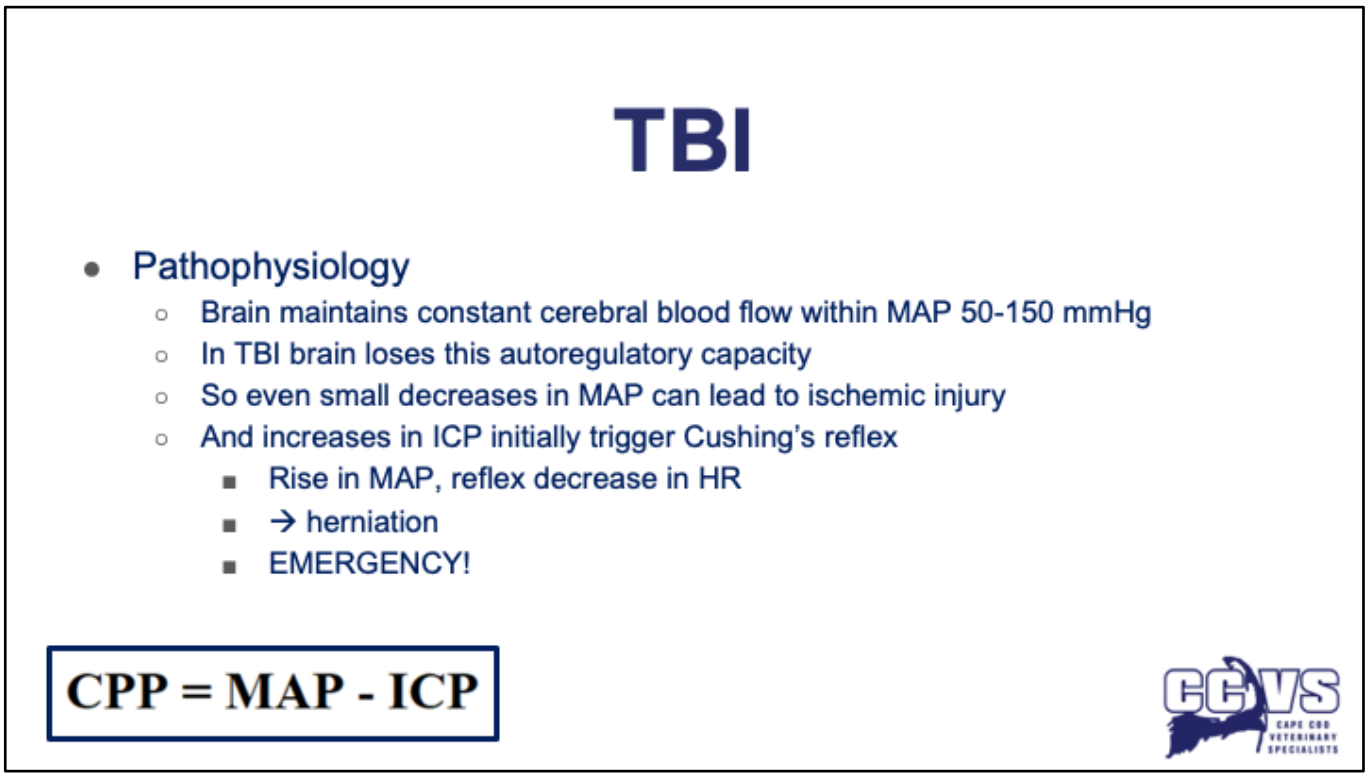


- TBIs can occur in several types of trauma cases
- Can have both primary and secondary injuries
- Primary injury
    - Concussion = brief loss of consciousness
    - Contusion = parenchymal hemorrhage and edema
    - Laceration = physical disruption of brain parenchyma
        - Can lead to compression of the brain from hematomas
- Secondary injury → neuronal cell death
    - Due to inflammatory mediators, excitatory neurotransmitters, changes in cell membrane permeability
    - Systemic insults that contribute to secondary brain injury = hypotension, hypoxemia, systemic inflammation, hyperglycemia, hypoglycemia, hypercapnia, hypocapnia, hyperthermia, electrolyte imbalances, acid base disturbances
        - Can lead to increased ICP, compromise the BBB, cause mass lesions, cerebral edema, infection, vasospasm, and seizures
        - All lead to neuronal death
- Injury also causes massive release of excitatory NT causes influx of Na and Ca into neurons → overwhelms mechanisms for removal → damage and cell death
- Also excessive metabolic activity results in depletion of brain ATP
- Ultimately with all this you get worsening of compromised CPP which worsens cerebral brain injury



- In normal physiology the brain can maintain constant cerebral blood flow within a 50-150 blood pressure
    - Autoregulatory mechanisms
        - CPP maintained via arteriolar myogenic reflexes
        - Functions at 50-150 mmHg
        - Outside this range, blood flow becomes linear with MAP = CBF passively follows systemic BP
    - Chemical autoregulation
        - PaCO2 = responsible for vascular resistance
            - CO2 in water forms H2CO3 which dissociates to H and HCO3 so you get increase in H which causes cerebral vasodilation
            - Decrease = vasoconstriction
            - Hyperventilation can cause
        - PaO2
            - Not as important as PCO2
            - Decreases cause vasodilation
- But in TBI the brain loses this autoregulatory mechanism
    - Pressure regulation generally affected first, then chemical
    - So small decreases in MAP can cause ischemic injury
        - Vasoconstriction decreases CBF and ICP but may result in hypoxia and neuronal ischemia
    - And physiologic changes that increase MAP can lead to increased cerebral

perfusion pressure
    - CBF can also be altered by changes in vascular tone or anything that impairs venous drainage (jugular compression, PPV, low head posture, increased intrathoracic pressure)
    - Vasodilation increased CBF and may lead to increased ICP
- And ultimately you can end up with the Cushing reflex
    - It indicates Late stage ICH, impending brain herniation and death
    - Clinical signs include Systemic hypertension, bradycardia, irregular resp
    - systemic hypertension bc MAP must overcome ICP to get cerebral blood flow
    - Bradycardia due to baroRc response from the systemic hypertension
    - Brainstem compression causes irregular resp
    - Ultimately this indicates an emergency

TBI

- Diagnostics – Neuro exam
  - If possible, prior to drugs
  - Modified Glascow Coma Score
  - Posture
    - Decerebrate
    - Decerebellate
  - Respiratory function
  - BP, HR
  - Papilledema

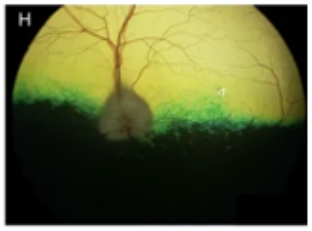


TABLE 128.1 Interpretation of Pupil Size and Pupillary Light Response in Head Trau

Pupil Size	Response to Light	Level of Lesion	Prognosis
Midposition	Normal	—	Good
Bilateral miosis	Poor to none	Cannot localize	Variable
Unilateral mydriasis	Poor to none	Cranial nerve III	Guarded to poor
Unilateral mydriasis and ventrolateral strabismus	Poor to none	Midbrain	Guarded to poor
Midposition	None	Pons, medulla	Poor to grave
Bilateral mydriasis	Poor to none	—	Poor to grave

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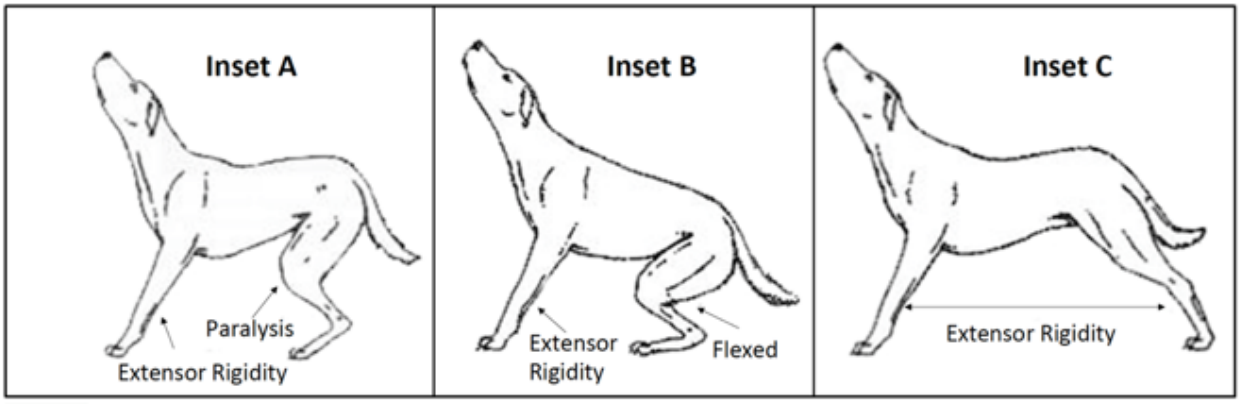
- The most important part of diagnostics for TBI is your neuro exam
  - If possible, ideal to do before pain meds or sedation but of course may not always be possible
  - Ideal to do a mGCS
    - Level of consciousness
    - Assessing brainstem reflexes
      - Brain death = deep coma, absence of spontaneous resp, loss of brainstem reflexes (fixed dilated pupils)
    - Motor activity = ambulatory, paresis, ataxia
  - Posture
    - Decerebrate rigidity = midbrain lesion
      - Unconscious
      - Recumbent
      - Opisthotonos (head thrown back)
      - Rigid extension of all limbs
    - Decerebellate = extensor rigidity in thoracic limbs and conscious
      - Acute cerebellar lesion
    - Also Schiff Sherrington?
  - Resp function
    - Breathing control = caudal brainstem = mid pons and cervicomedullary junction
  - Papilledema on fundic exam = dilated fluffy optic nerve = reliable sign of ICH

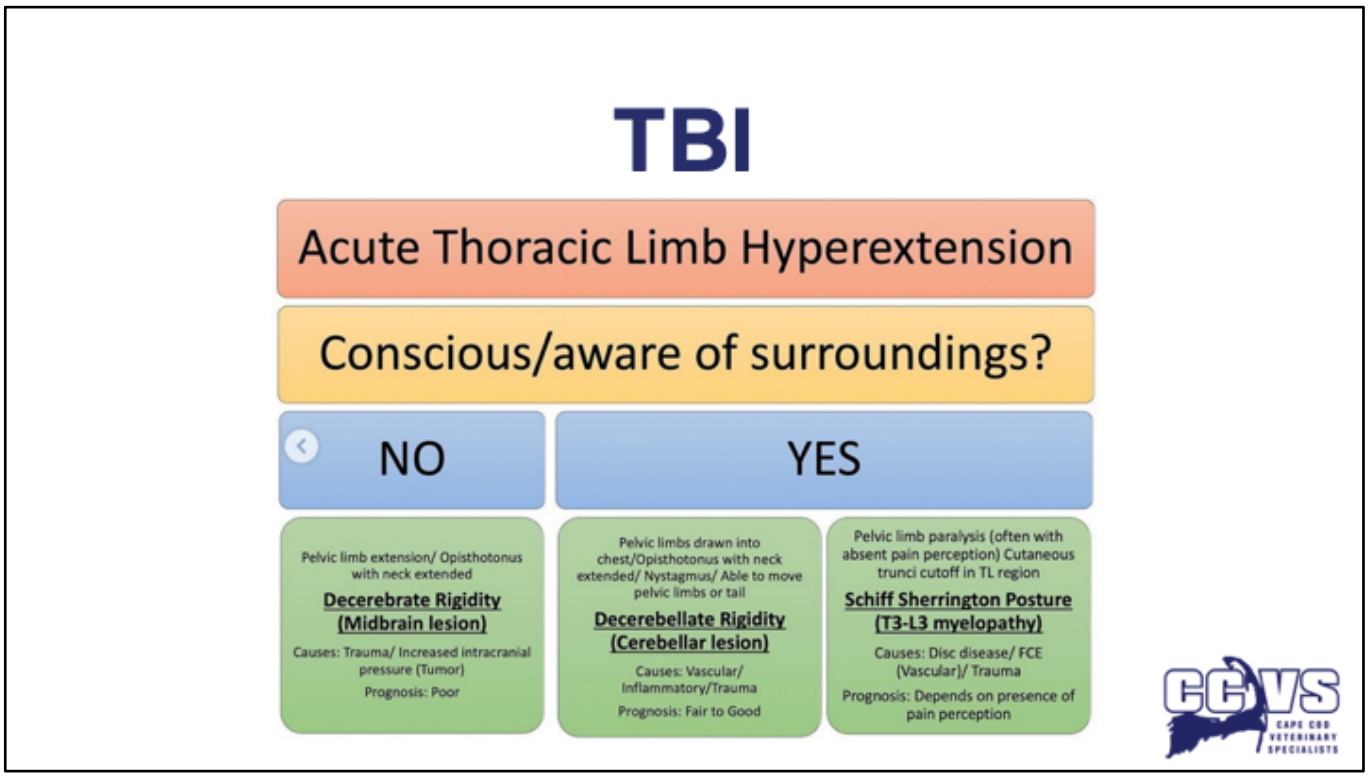
- And then don’t forget about Systemic assessment
    - They can exacerbate injuries to CNS
    - Hyper or hypoNa, hypoK
    - SIADH, transient HPA dysfunction

TBI



- Posture
    - Schiff Sherrington = T3L3 myelopathy
        - Patients with injury to the T2-L2 thoracic spine often display the Schiff-Sherrington syndrome (Figure 47, inset A), typically with normal mentation, forelimbs in extensor rigidity, and hind limbs that are flaccid. The prognosis for these patients is usually grave due to severe spinal cord trauma.
    - Decerebellate = extensor rigidity in thoracic limbs and conscious
        - Acute cerebellar lesion
        - Patients with decerebellate rigidity (Figure 47, inset B) typically are obtunded or depressed, have opisthotonus, have fore limbs in extensor rigidity, and hind limbs in active flexion.
        - These patients have a guarded prognosis due to severe injury to the cerebellum.
    - Decerebrate rigidity = midbrain lesion
        - Unconscious
        - Recumbent
        - Opisthotonos (head thrown back)
        - Rigid extension of all limbs
        - Patients with decerebrate rigidity (Figure 47, inset C) typically are obtunded, have opisthotonus, and the fore limbs and hind limbs are in extensor rigidity. The prognosis for these patients is grave due to severe injury to the midbrain.

- https://books.allogy.com/web/tenant/8/books/11511717-af9b-4844-9f65-6df25c423e24/



- Here is another diagram that takes you through flow chart to determine posture
- https://www.instagram.com/p/CfEbm52LXar/?img_index=3

TABLE 22. MODIFIED VETERINARY GLASGOW COMA SCALE[7]	
Level of Consciousness	**Score**
Occasional periods of alertness and responsive to environment	6
Depression or delirium, capable of responding but response may be inappropriate	5
Stupor – semi comatose, responsive to visual stimuli	4
Stupor – semi comatose, responsive to auditory stimuli	3
Stupor – semi comatose, responsive only to repeated noxious stimuli	2
Comatose – unresponsive to repeated noxious stimuli	1
Motor Activity	
Normal gait, normal spinal reflexes	6
Hemiparesis, tetraparesis, or decerebrate activity	5
Recumbent, intermittent extensor rigidity	4
Recumbent, constant extensor rigidity	3
Recumbent, constant extensor rigidity with opisthotonus	2
Recumbent, hypotonia of muscles, depressed or absent spinal reflexes	1
Brainstem Reflexes	
Normal PLRs and oculocephalic reflexes	6
Slow PLRs, normal to reduced oculocephalic reflexes	5
Bilateral unresponsive miosis, normal to reduced oculocephalic reflexes	4
Pinpoint pupils, reduced to absent oculocephalic reflexes	3
Unilateral unresponsive mydriasis, reduced to absent oculocephalic reflexes	2
Bilateral unresponsive mydriasis, reduced to absent oculocephalic reflexes	1

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- And then here is the modified glascow coma score with your 3 areas of assessment
- Don't go through – here for reference
- mGCS
    - Level of consciousness
        - Alert and responsive = not likely ICH
        - Obtunded = slow or inappropriate response to sensory
        - Stuporous = unresponsive except noxious stimuli
            - Suspect marked ICH
        - Comatose = unresponsive to noxious stimuli
            - Strongly suspect herniation
        - Consciousness = rostral brainstem, ascending reticular activation system, and cerebrum
        - Unconscious = likely to have global loss of autoregulatory responses
    - Assessing brainstem reflexes
        - Most info about brainstem function
        - PLR, oculocephlic reflex, pupil size
        - Size and reactivity of pupil
            - Constriction of pupil due to midbrain and efferent PNS fibers of CN 3
            - Mydriasis = lesion ipsilateral or bilateral of midbrain or CN3
            - Miosis = ipsilateral injury or part of Horners (ptosis,

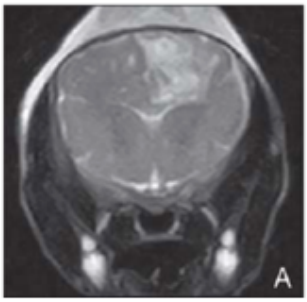
enopthalmus, third eyelid protrusion = SNS lesion)
        - Bilateral miosis = acute extensive brain disturbance
        - Unresponsive bilateral mydriasis = brain herniation
    - Resting eye position, movement, oculocephalic reflex
        - Eye abduction = CN 3 injury
        - Eye adduction = CN 6 injury or rostral medulla or pons
        - Positional nystagmus = vestibular
        - Oculocephalic reflex = moving head from side to side or vertical
            - Assess functional integrity of large segment of brainstem
            - Cervical spinal cord, medulla, CN 3, 4, 6
    - Corneal reflex
        - Absent when CN 5, 6 or 7 damaged
- Motor activity = ambulatory, paresis, ataxia
- https://books.allogy.com/web/tenant/8/books/11511717-af9b-4844-9f65-6df25c423e24/

TBI

MVGCS Score	Suggested Prognosis
3-8	Grave
9-14	Guarded
15-18	Good

- Like in human scale, lower score is worse
- Can be used serially to determine response to treatment
    - Monitor improvements, possibly improving prognosis
    - If worsening may need more advanced imaging such as CT or MRI
- https://books.allogy.com/web/tenant/8/books/11511717-af9b-4844-9f65-6df25c423e24/

TBI



- Diagnostics - Imaging
  - Goals with imaging
    - Identify depressed skull fractures
    - Info on mass lesions
  - Skull radiographs? Insensitive
  - CT = preferred imaging for bone and acute hemorrhage or edema
  - MRI = more useful as time from injury increases or if notice subtle neuro deficits
  - Strongly recommend imaging if: moderate to severe neuro signs, lateralizing signs, failure to improve significantly within first few days, acute decline in neuro status

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- And then final category of diagnostics is imaging
  - Goals with imaging
    - Identify depressed skull fractures
    - Info on mass lesions
  - Skull radiographs? Insensitive
  - CT = preferred imaging for bone and acute hemorrhage or edema
  - MRI = more useful as time from injury increases or if notice subtle neuro deficits
  - Strongly recommend imaging if: moderate to severe neuro signs, lateralizing signs, failure to improve significantly within first few days, acute decline in neuro status

TBI

- Treatment priorities
  - Maintain adequate cerebral perfusion pressure
  - Ensure adequate oxygen delivery to the brain
  - Treat acute intracranial hypertension

- So now to focus on treatment for TBI patients
  - Goals are to maintain perfusion
  - Ensure adequate O2 delivery
  - And treat any intracranial hypertension

TBI

- Treatments
  - O2 therapy
  - Volume resuscitation
  - Intracranial hypertension
    - Hyperosmotic agents for acute intracranial hypertension
      - Mannitol 0.5 to 1.5 g/kg IV with filter
      - Hypertonic saline 3-5 mL/kg IV
    - Decrease cerebral blood volume = elevate head 15-30 degrees via slant board
    - Hyperventilation
      - Only for short term to EtCO2 20 to 30 mmHg; not long term due to ischemia from vasoconstriction
  - Treat seizures

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- First, O2 indicated in any patient with TBI
- But To ensure adequate O2 delivery you need perfusion so don't forget volume resuscitation
  - Like I said earlier the permissive hypotension approach may not be appropriate for TBI patients
  - May need to aim higher than 80-90 mmHg SBP
- And finally Tx acute ICH
  - So if you note a Cushing's reflex you need to act quickly
  - First can consider a hyperosmotic agent
    - Mannitol
      - Decreases blood viscosity causing an increase in CBF and O2 delivery so can see improvement within minutes
      - Hyperosmotic effects in 15-30 min and can last 1.5-6 hours
      - May increase permeability of BBB
      - If leaks into brain parenchyma it can worsen edema – so not recommended if hemorrhage?
      - If hypovolemia/hemorrhage then must follow with crystalloids to maintain intravascular volume
    - HTS
      - Does not cross BBB
      - Also decreases blood viscosity and hyperosmotic
      - Less risk of hypotension than with mannitol bc Na is redistributed in body

- Next can try to decrease cerebral blood volume and thus ICP
    - Elevate by 15-30 degree → increases venous drainage, decreases ICP, and increases CPP without deleterious changes in cerebral oxygenation
    - Slant board instead of pillows or towels soas not to occlude jugular veins
    - If go too high with slant then may cause decrease in CPP
- And finally can do hyperventilation but only for very short while
    - Aim for EtCO2 20-30 mmHg
        - Again goal being to cause vasoconstriction
    - But if do longterm then may get ischemia from vasoconstriction
- And then of course if any seizure activity then need to treat those

TBI

- Controversial treatments
  - Steroids – not recommended, worse outcomes
  - Furosemide – not recommended
  - Seizure prophylaxis
    - Often consider in severe TBI cases
    - Usually levetiracetam 60 mg/kg IV
  - Coma induction and therapeutic hypothermia
    - Goal = decrease cerebral metabolic rate
    - Keep in induced coma around 48 hours
    - Practical approach – unless worried about CV instability, warm up more slowly

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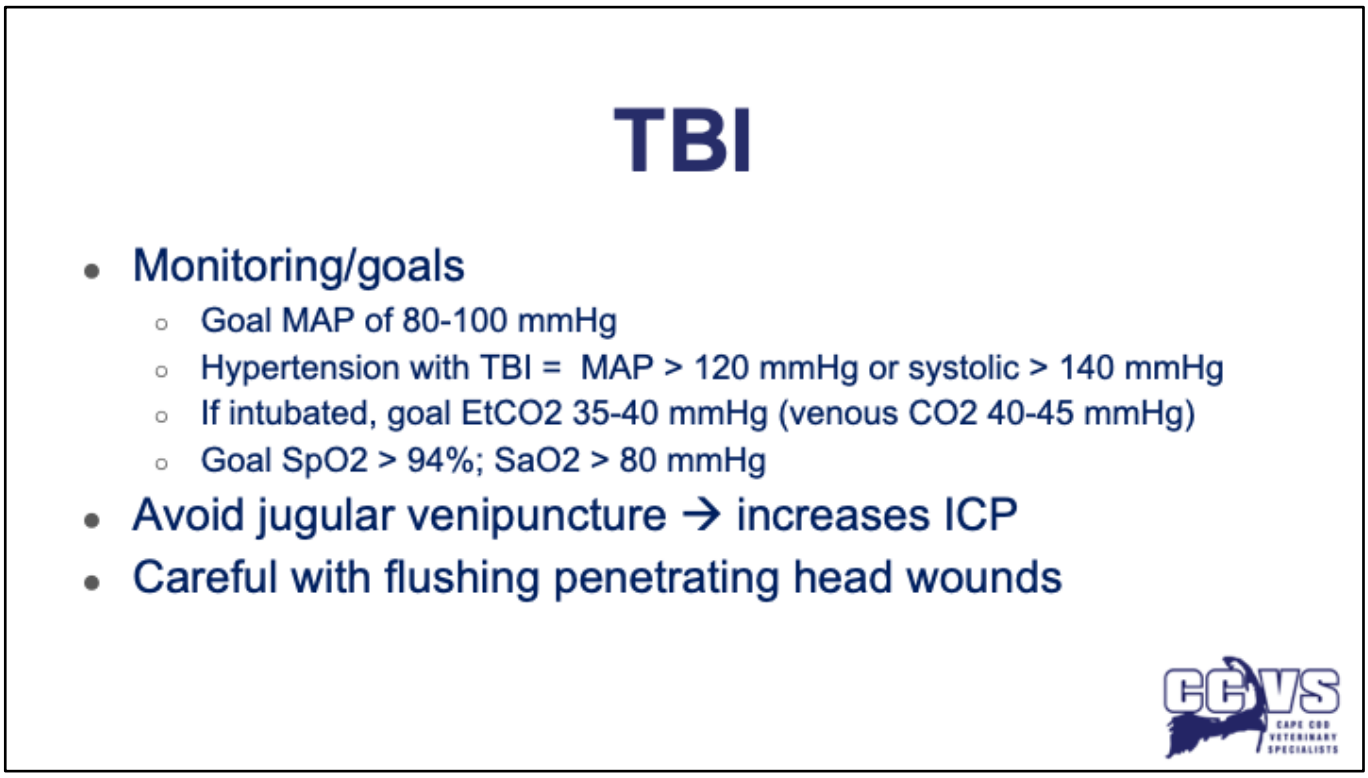
- There are some treatments that have been tried but deemed more controversial
- Steroids overall are not recommended – data shows worse outcomes
- Furosemide
  - Causes intravascular volume depletion and systemic hypotension leading to decreased CPP
  - Not recommended
  - Often in trauma patients don't want to cause intravascular volume depletion – not stable enough for that
- Seizure prophylaxis
  - Not been show to reduce development of delayed seizures in ppl after TBI
  - But most neurologists that I've talked to have recommended it for about a month after severe TBI – often just Keppra
    - There is a bit of mixed beliefs on prophylactic tx with Keppra
    - Some criticalists/neuro will recommend, others don't
    - I think it depends on the severity of TBI and my level of concern that they could develop
    - But if they do then absolutely must treat
- And then Coma induction
  - Reasoning = excitotoxicity and inflammation lead to cerebral ischemia and cellular swelling → increasing ICP
  - induce coma and hypothermia
  - Has support in human research – around 48 hr is beneficial; single case report in vet med for refractory seizures after TBI

- Consider if refractory intracranial hypertension
- I may not use this technique directly but if TBI and cold I may warm up more slowly or stop earlier – if they can tolerate it cardiovascularly



- And then throughout tx and with all TBI patients – very important to monitor closely and often and do serial neuro exams
- Our goals for vitals include
    - MAP 80-100 mmHg
    - If get MAP > 120 or systolic> 140 with TBI patient then be concerned for hypertension
        - I pair with neuro exam bc trauma patients may have other reasons to be hypertensive too
    - For end tidal, goal 35-40
        - End tidal goals
            - Increase in CO2 can lead to cerebral vasodilation and increased intracranial blood volume, worsening ICP
            - Decreased CO2 from hyperventilation can lead to cerebral vasoconstriction, decreasing CBF and leading to cerebral ischemia
            - So maintain in specific range
            - This may require mechanical ventilation
    - And then for oxygenation SPO2 > 94%
- Don't forget to avoid jugular venipuncture
    - Jug venipuncture – marked increases in ICP due to decreased venous outflow from the brain
        - Also reason to avoid neck harness or neck restraint in any neuro case

- And be careful flushing any head wounds if there is concern for communication

So what do I do?

- TBI
  - Careful with movement in case of spinal injury
  - If concern for TBI – monitor BP and HR for Cushing's reflex, slant board at 30 degree
  - O2 therapy
  - Resuscitate to normal BP if worried about TBI to maintain cerebral perfusion pressure
  - Give HTS if concern for Cushing
  - If still concern then give mannitol
  - Treat seizures

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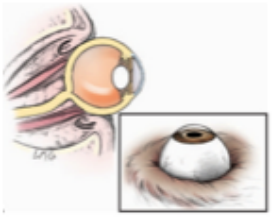
- So our short summary for what to do with TBI patients
- TBI
  - Neuro exam prior to drugs
  - Pupil size, PLR, level of consciousness - if can give modified glascow coma score baseline great!
  - Careful with movement in case of spinal injury – assess posture
  - If concern for TBI – monitor BP and HR for Cushing's reflex, slant board at 30 degree
  - O2 therapy,
  - resuscitate to normal BP if worried about TBI to maintain cerebral perfusion pressure
  - Give HTS if concern for Cushing
  - If still concern then give mannitol
  - Can also do slant board
  - Seizure meds if needed

Proptosis

Proptosis

- What is proptosis?
  - Severe, acute exophthalmos
  - Specifically involves entrapment of the eyelids behind the globe
  - = Emergency
- Leads to:
  - Compromise of globe's vascular supply
  - Peribulbar swelling
  - Stretching of optic nerve and possibly optic chiasm



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Proptosis

- What to do
  - Until surgery can be performed…
  - Keep the eye lubricated!
    - Sterile saline rinse or ophthalmic ointment q 1-2 hours
  - Analgesia
    - Parenteral opioids
  - E-collar to prevent additional trauma to the globe

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- So what do we do with these caess?
- Most important is keeping it lubricated
- Analgesia
- E collar to prevent rubbing

Proptosis

- Full ophthalmic exam – BOTH eyes!!
- Focus on
  - Globe health
    - Globe rupture?
  - Corneal health?
    - Extensive hyphema?
  - Vision
    - Dilated vs. constricted pupil
    - Lack of consensual PLR to contralateral eye?
  - Extraocular muscles

Proptosis

- Prognostic factors
  - Trauma vs. other etiologies
  - Skull conformation
    - Dolichocephalic dogs and cats require more force
    - Vs. brachycephalic dogs
  - Ruptured vs. intact globe
  - Dilated vs. constricted pupil
  - Lack of consensual PLR to contralateral eye
    - Traction on optic chiasm?
  - Corneal health
    - Moist vs. desiccated cornea
  - Corneal laceration that extends past limbus
  - Extensive hyphema
  - Owner compliance
  - Extraocular muscles

- Research has found some factors to be important in prognosis
- Favorable prognostic indicators
  - Brachycephalic
  - Positive PLR
  - Vision
- Nonfavorable
  - Cat
  - Hyphema
  - No visible pupil
  - Facial fractures
  - Optic nerve damage
  - Avulsion of 3 or more extraocular muscles
  - Eye ruptured
  - Eye completely filled with blood

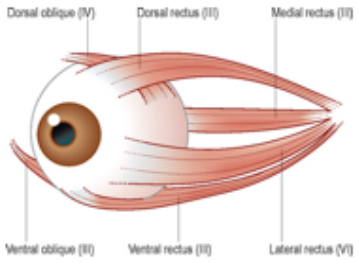
Proptosis

Table 1
Prognostic Indicators for Proptosis

Favorable Prognosis	Unfavorable Prognosis
Positive direct or consensual pupillary light reflex (a negative reflex is not necessarily poor prognosis)	Non-visible pupil (corneal desiccation or hyphema)
No extraocular muscles avulsed	Scleral rupture
Brachycephalic breed	Greater than 3 extraocular muscles avulsed
Short duration	
Vision on presentation	
Normal posterior segment	

- Cats worse prognosis than dogs
- Proptosis can lead to exposure corneal ulcers if not managed in a short amount of time → make sure to start lubrication at presentation
- Potential long term consequences of replacement: lateral strabismus (torn medial rectus)retinal degeneration, optic nerve degeneration, keratoconjunctivitis sicca
- Shallow orbits in brachycephalic dogs make it easier for proptosis to occur, but also makes it more likely that they'll require since less force is required to proptose (less muscle trauma)
- Betbeze, 2015 adapted from Gilger, 1995

Proptosis



- Extraocular muscles
  - Medial rectus muscle most common to rupture
    - → lateral strabismus
    - May improve over time
  - If >3 extraocular muscles torn → phthisis bulbi
    - Anterior ciliary arteries enter eye through these muscles
    - Negative prognostic factor

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- Assessment of extraocular muscles are particularly important for prognosis
- If 3 are ruptured then worse prognosis
- Muscles that rupture first = medial and ventral recti and ventral oblique = insert most anterior on sclera or are shortest

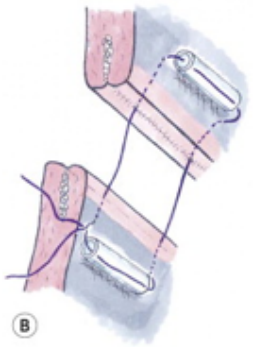
Proptosis

- Treatment options
  - Temporary tarsorrhaphy
    - Replace globe in proper position
    - Protect globe
    - Maintain ability to medicate globe
  - Enucleation
    - Remove source of pain
    - Remove threat to systemic health

- For treatment you can try to manage vs just enucleation
- With med managmenet you need temporary tarsorrhapy
    - Can still give meds

Proptosis

- Temporary tarsorrhaphy
  - May need lateral canthotomy to facilitate globe replacement
  - Place horizontal mattress sutures with tension-relieving stents
    - Place through stent → enter 5-8 mm away from upper lid margin → exit Meibomian gland
    - Enter through Meibomian gland → exit 5-8 mm away from lower lid margin → place through stent
    - Reverse needle and direction
  - Place all sutures before tying!
  - Leave space at medial canthus for medication administration
  - Lift sutures up and out while gently placing globe back into orbit



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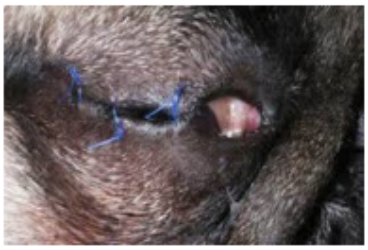
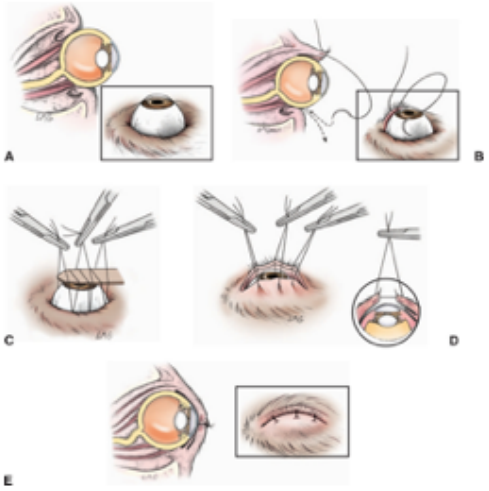
- These slides just walk through how to do

Proptosis

- Temporary tarsorrhaphy





Proptosis

- Post op medications
  - Corticosteroid tapering OR NSAID for several days
    - Goal = control periorbital swelling to decrease further damage to optic nerve
  - Topical antibiotic QID
  - Topical atropine BID
  - Oral antibiotic for 14 days
  - Additional oral analgesia
  - E-collar!

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- Following the tarrshorraphy
  - Med to help with inflammation
  - Topical antibiotic
  - Topical atronpine
  - Oral antibiotic
  - Oral analgesia
  - E collar!

Proptosis

- Recheck plan
  - Check eye about every 2 days
    - Owners able to medicate?
    - Check for purulent discharge
    - Complete PE and TPR → febrile?
  - Remove sutures in 14 days
    - Fluorescein stain to check for ulcers!!
  - Recheck 3-5 days later again to check for complications
    - Infection, Exposure keratitis, Lateral strabismus, Blindness, Dry eye disease

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- Finally for recheck you need to eval about every 2 days to see how pet is doing
- Remove stitches in 14 days
  - Check for ulcers!
- Then check again for 3-5 days later for complications

So what do I do?

- Lubricate eye
- Analgesia
- E-collar
- Temporary tarsorrhaphy vs enucleation

Skin Wounds

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Skin Wounds

- Abrasions, lacerations, degloving, punctures, open fracture
- Often classified as “contaminated”
- Usually final part of evaluation after assessing other life threatening injuries
  - Except wounds that continue to hemorrhage
    - Tourniquet
      - BP cuff up to 200 mmHg for up to 1 hour
    - Don’t remove penetrating foreign material or replace open fracture! Object or bone could be preventing hemorrhage – wait for OR

- So we see these in many trauma cases
- Clean, clean contaminated, contaminated, dirty or infected
    - Trauma wounds are often contaminated
- I’m often looking at wounds once a pet is deemed stable
    - Unless hemorrhaging
    - If so can use a tourniquet
    - For penetrating wounds or open fracture, don’t move it
        - This is where you scream at the TV don’t pull it out!
        - The object or bone could be preventing hemorrhage

Skin Wounds

- Initial goal for wounds = clean, moist, protected
  - Handle all trauma patients with gloves
  - Use sterile lube in wounds before shaving
  - Flush superficial wounds if able/safe
    - 60 mL syringe with 18g needle
  - Sterile lubricant or antibiotic ointment before dressing
  - Nonadhesive dressing
- May need to consider/discuss damage control approach to wound care
- Wound vacuum

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- Initial goals for wounds is just keeping them clean, moist, protected
- Handle with gloves
- Use sterile lube before clip and clean
- Flush wound if able/safe – ie maybe not all head wounds
- Then put sterile lubricant or antibiotic ointment before dressing with nonadhesive bandage
- If going with damage control approach then skin wounds or fractures may not be addressed
- And then some severe wounds may require days of wound vaccum until can close

Skin Wounds

- Antibiosis
  - Most common wound pathogens
    - Gram positive: Staph, Strep
    - Gram negative: E coli, Enterococcus, Proteus, Pseudomonas
    - Bites: Pasteurella multocida also common
    - Anaerobic: Bacillus, Clostridium, Corynebacterium
  - Superficial wounds: cefazolin
  - Infected, deeper wounds: ampicillin/sulbactam
  - Severe bite wounds: ampicillin + fluoroquinolone or aminoglycoside
  - Topical antibiotic ointment, SSD, Manuka honey
- Don't forget analgesia!
  - Full mu-opioid agonist recommended: methadone, hydromorphone, fentanyl

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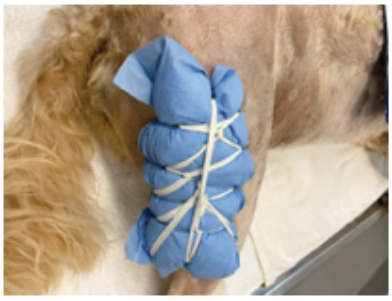
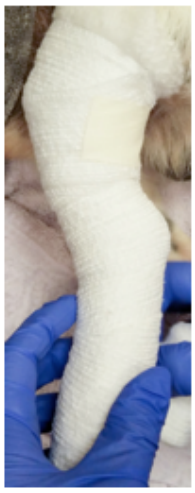
- When choosing antigbiotics it will depend on how severe or infected the wound is
- I've listed the most common trauma wound pathogens here
  - This list lends to the recommended antibiotics
- And don't forget about topical too
- Fluoroquinolone = enrofloxacin, pradofloxacin, marbofloxacin
- Aminoglycoside = amikacin, gentamycin, neomycin
- So long story short – if you can get a dose of cefazolin or unasyn in before transfer that's great
- And then again analgesia must be considered, often need opioid for control

So what do I do?

- Analgesia
- Antibiotic if indicated
- Cover with sterile nonadhesive dressing, tie-over dressing





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- In summary for wounds, give pain meds, antibiotic if indicated and cover with sterile nonadhesive bandage after cleaning it up
- And don't take out or move foreign material or bone until ready to take to OR if needed

WRAP IT UP

Practical Initial Triage Plan

- Vitals: TPR, blood pressure
- Neuro exam: level of consciousness, posture, pupil size and response to light
  - Be careful about moving for exam in case of TBI or spinal injury!
- POCUS chest and abdomen
- Initial bloodwork
  - Ideally lactate, blood sugar, PCV/TP
  - Extras: Blood gas, Renal/liver values, Platelets, PT/PTT
- Radiographs
  - Chest and abdomen – at least a lateral of each to assess for free gas

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Practical Initial Triage Plan

- Resuscitation
  - IV or IO access
  - Balanced crystalloid bolus 10 mL/kg; repeated as needed
  - HTS 3-5 mL/kg bolus
  - Blood products
  - Autotransfusion if able
  - TXA 10-15 mg/kg IV or aminocaproic acid 50-100 mg/kg IV
  - Recheck HR, BP, mentation, CRT, pulse quality
  - Aim for SBP 80-90 mmHg unless TBI

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Practical Initial Triage Plan

- Thoracic wounds
  - Analgesia
  - Antibiotic if penetrating injury
  - O2 therapy
  - Flail side down vs sternal
  - Thoracocentesis
  - Thoracostomy tube if needed +/- continuous suction
  - Pericardiocentesis if tamponade

- Analgesia
- Antibiotic if penetrating injury
- O2 therapy
  - Flow-by vs nasal cannulas vs intubation and PPV
- Flail side down vs sternal
- Thoracocentesis
  - Thoracostomy tube if needed +/- continuous suction
    - MILA
    - Red rubber
- Pericardiocentesis if tamponade
- Thoracotomy

Practical Initial Triage Plan

- Abdominal wounds
  - Abdominocentesis if free fluid
  - Hemoabdomen
    - Autotransfusion, belly wrap?, damage control surgery vs ex lap
  - Septic fluid or free gas
    - Start antibiotics, damage control surgery vs ex lap
  - Uroabdomen
    - Manage hyperkalemia
    - Place u-cath
    - Place abdominal drain if unable to manage K and azotemia
    - Contrast study, ex lap when stable
  - Evisceration
    - Sterile lubricate, cover and protect, antibiotics
    - Damage control surgery vs ex lap

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Practical Initial Triage Plan

- TBI
  - Careful with movement in case of spinal injury
  - If concern for TBI – monitor BP and HR for Cushing's reflex, slant board at 30 degree
  - O2 therapy
  - resuscitate to normal BP if worried about TBI to maintain cerebral perfusion pressure
  - Give HTS if concern for Cushing
  - If still concern then give mannitol

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Practical Initial Triage Plan

- Proptosis
  - Lubricate, pain meds, E collar!
- Skin wounds
  - Pain meds
  - Lube, clip, flush/clean if able
  - Otherwise cover with nonadhesive dressing
  - Start antibiosis if stable enough – cefazolin for superficial, Unasyn if deeper/infected

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Further Training

- Veterinary Advanced Trauma Life Support training
  - VetATLS course online modules debut on June 1, 2026!
  - Cost for the online course is $375 for veterinarians and $225 for technicians, students, and all other interested learners.
  - Check out the intro video: https://vetcot.org/vetatls/
- VetCOT (Veterinary Committee on Trauma)
  - Annual conference
  - Trauma Registry
  - https://vetcot.org/

QUESTIONS?

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NOTICE

CE credit certificates & presentation slides will be emailed to you. If you do not receive an email with this information within a week, contact Nichole - *nicholemanfredi@capecodvetspecialists.com*

Additional Info

Trauma Induced Coagulopathy

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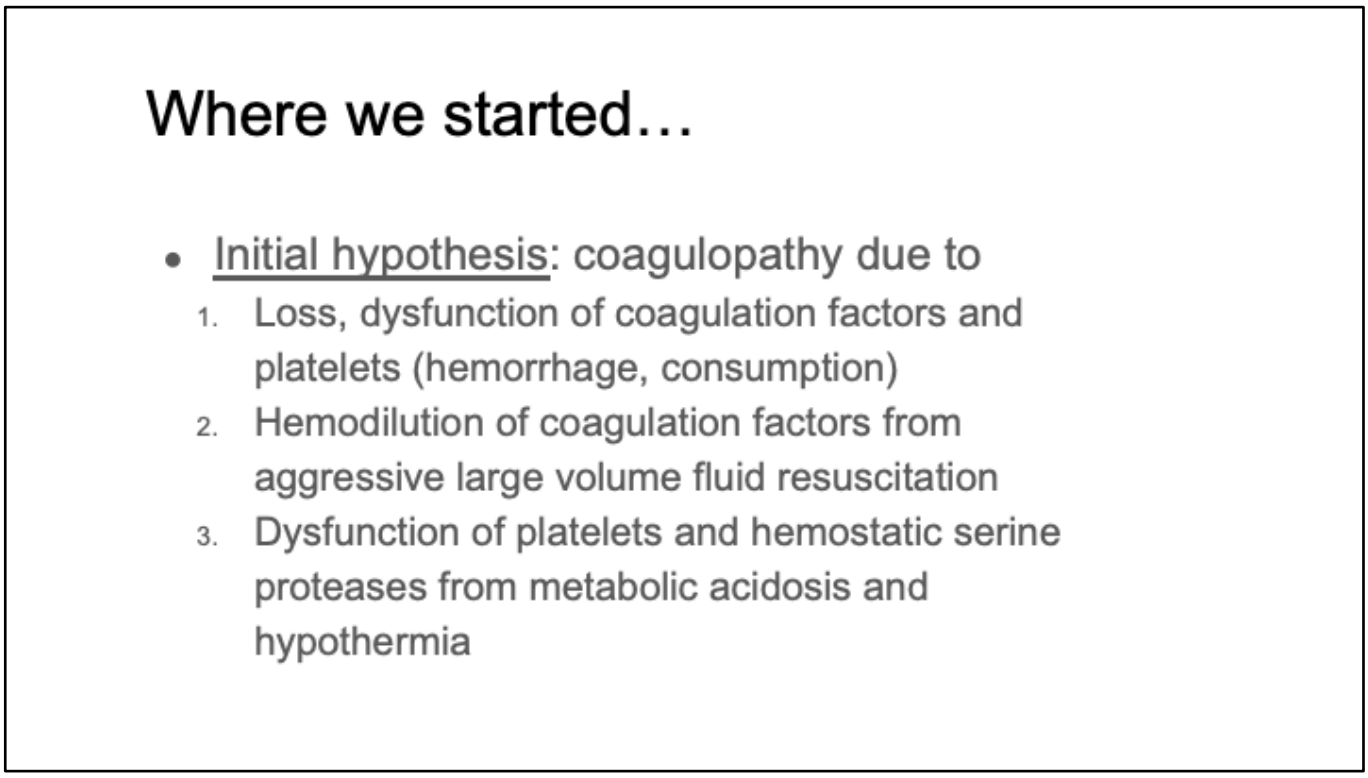
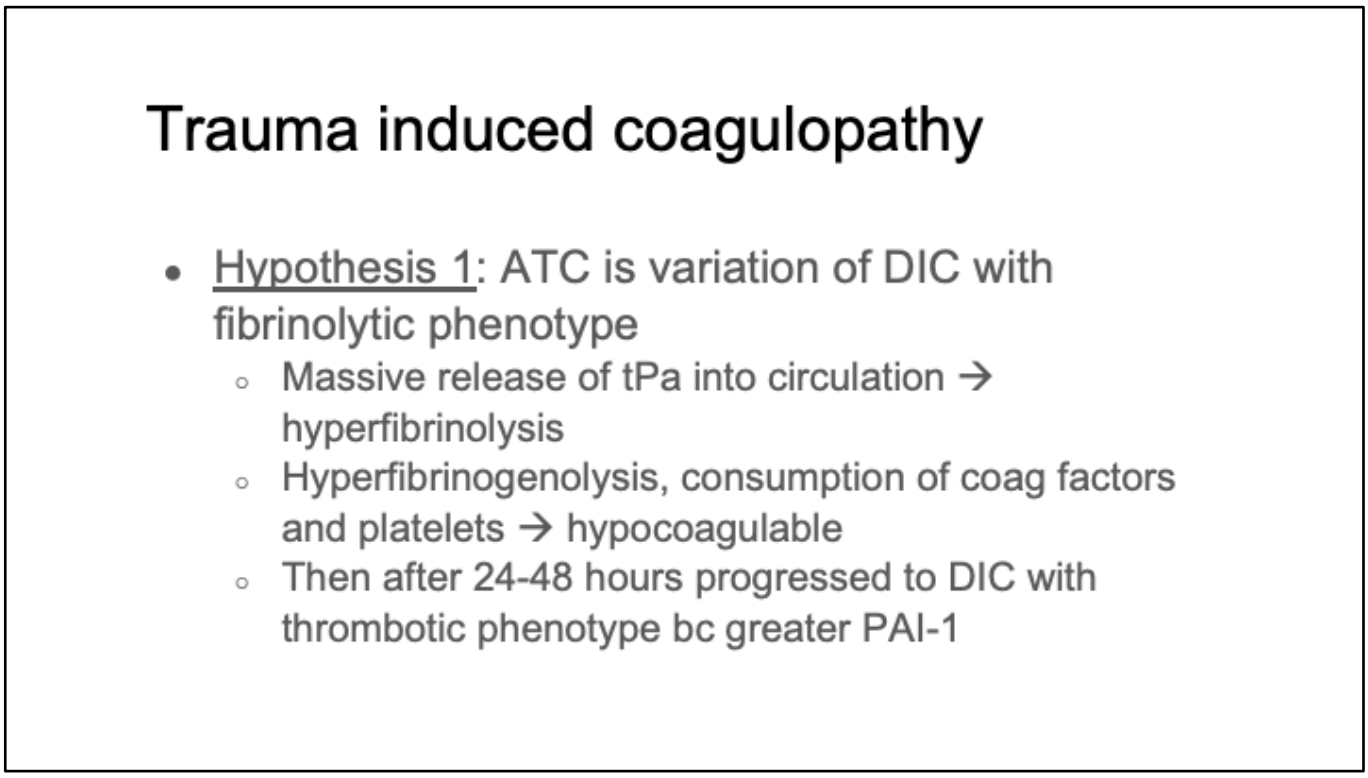


- There was recognition that trauma patients were hypocoagulable and it was hypothesized that this was due to consumption and loss, hemodilution from resuscitation and dysfunction from metabolic acidosis and hypothermia

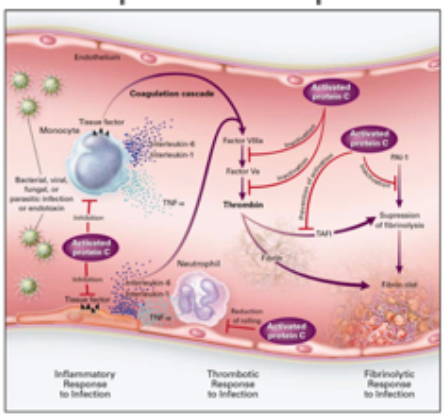
- But it was noted that patients were arriving to ER already hypocoagulable – often within 30 minutes
- So started to realize there is a distinct endogenous coagulopathy that can develop within extremely quickly, before any resuscitation or before all sequalae occur

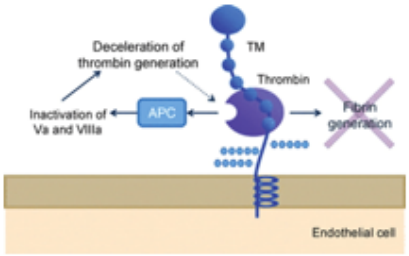


- The first hypothesis centers on ATC being a variation of DIC
- immediately following trauma and shock, you get severe endothelial injury, hypoxia, ischemia → marked generation of thrombin with systemic fibrin formation
    - So you get massive release of tissue plasminogen activator (tPa) into circulation
        - This causes conversion of large amounts of plasminogen into plasmin
        - So you get hyperfibrinolytic state
    - Hypocoag bc of primary hyperfibrinogenolysis, secondary hyperfibrinolysis, and consumption of coag factors and platelets with the marked generation of thrombin and systemic fibrin
    - As you get to later thrombotic stage, you see greater expression of plasminogen activator inhibitor-1 (PAI-1) which inhibits tPa
        - This is also important to keep in mind as these patients survive 24-48 hours after the trauma – you need to consider the notion that they may be hypercoagulable at that point

Trauma induced coagulopathy

- Hypothesis 2: Enhanced thrombomodulin-thrombin protein C pathway





- The second hypothesis differs from first hypothesis in that the hypocoagulability does not stem from a consumptive process
- Pathophys
  - It occurs because of enhanced activation of the thrombin-thrombomodulin protein C anticoagulant pathway
  - When thrombomodulin binds to thrombin it switches to anticoagulant function via the protein C activation (this activation causes inhibition of Factor 5a and 8a)
    - Also protein C enhances fibrinolysis via suppression of PAI-1 and TAFI activity and formation

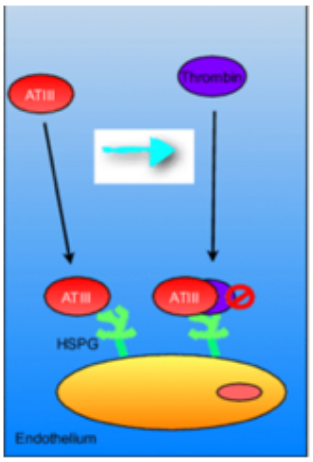
- Review
- TM (thrombomodulin) binds thrombin
  - Activates protein C → Irreversibly cleaves factor Va and VIIIa → no more thrombin can be formed
  - Inactivates PAI-1 (plasminogen activator inhibitor 1; inhibits fibrinolysis) → increases fibrinolysis
    - PAI-1 can be induced by LPS, TNF-a, TGF-b, and IL-1 → procoagulant
- TM on endothelial cell surface
  - When binds thrombin
  - Activates protein C → irreversibly cleaves factor 5a and 8a

        - So no more thrombin can be formed
        - Also inactivates plasminogen activator inhibitor 1 (which inhibits fibrinolysis) → increases lysis of any formed fibrin
            - PAI-1 is induced by LPS, TNF-a, TGF-b, and IL-1

- When activated, protein C promotes fibrinolysis and inhibits thrombogenesis and inflammation
    - Converted to active form by thrombin coupled to thrombomodulin
        - This is impaired during sepsis due to down regulation of thrombomodulin by cytokines
- Research has found reduced levels of protein C in patients with sepsis
    - Research found it was associated with increased risk of death

Trauma induced coagulopathy

- Hypothesis 3: Marked sympathoadrenal response leading to catecholamine-induced endothelial damage
  - Glycocalyx releases: heparin-sulfate, thrombomodulin, tPa
  - HSPG (heparin sulfated proteoglycans) = Binds antithrombin (AT) → inhibits factor Xa, IXa, XI, thrombin
  - Hypocoagulable + hyperfibrinolytic



- And the final hypothesis
- Sympeathoadrenal response, which is dose-dependent, and leads to catecholamine induced endothelial damage
  - The catecholamines directly damage endothelial glycocalyx in dose dependent fashion
  - The damaged endothelium changes to more prothrombotic state
    - Local hemostasis
    - But to prevent systemic coagulation there's a counter regulatory response that is anticoagulant
      - Glycocalyx sheds heparin-sulfate and soluble thrombomodulin which is anticoagulant into systemic circulation (autoheparinize)
        - HSPG on endothelial cell surface Binds antithrombin – will inhibit thrombin when produced in that area
          - Inhibits factor 10a, 9a, 11, and thrombin
      - Also sheds tPa which is fibrinolytic
- As degree of tissue trauma and endothelial damage increases, the counter regulatory response rages out of control

- review

- HSPG on endothelial cell surface
    - Binds antithrombin – will inhibit thrombin when produced in that area
        - Inhibits factor 10a, 9a, 11, and thrombin
        - Inhibits leukocyte activation (link here) via down regulation of P-sel
    - Blocks cytokine expression
    - AT can be degraded by elastase from activated neutrophils (link here)
    - HSPG can be decreased secondary to proinflammatory cytokines, decrease efficacy of AT