The Triangle of Death: Hypothermia, Acidosis and Coagulopathy

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Objectives

At the end of the session the participant will be able to:

1. Discuss the significance of hypothermia, acidosis and coagulopathy in the survival of the critical care patient.
2. Discuss the importance of recognizing the components of the triangle of death.
3. Analyze important lab values in recognition of the triangle of death.
4. Integrate treatment options for patients in the triangle of death.

Trauma Statistics

• Ages 1-44
• 1/10 of all deaths are due to trauma Worldwide
• Uncontrollable Hemorrhage is the 2nd leading cause of traumatic death
• And, the leading cause of preventable traumatic death


In Trauma Patients:

Hypothermia accompanied with acidosis and coagulopathy increase your mortality by 90%

The Triangle of Death Facts

How does Hypothermia in trauma patients differ from patients undergoing Cardiac surgery?

Trauma Patients start with oxygen deficits, shock and hemorrhage

Triangle of Death

- Hypothermia induced by heat loss on scene
- Cold IV fluids in ED
- Temperature decrease have a greater impact on platelets reducing activation
- Thrombin generation is < 50% at 7.20
- < ph increases fibrinolysis

The Triangle of Death

- Ph 7.10
- Temp. 34
- SBP 70 for > 70 mins.

98% Chance of coagulopathy

What Fluids do we use for resuscitation?

High CF (154meq/l) solutions like NS that have a Ph of 5 may contribute to acidosis. Some attendings use LR (buffer) with a PH 6.5 for this reason. NS can exacerbate lactic acidosis.

Hypothermia

- 66% of Trauma Patient arrive to the ER Hypothermic
- Greatest loss of temperature (57%) occurred in the ED rather than in the field
- Hypothermia has protective effects but can be harmful clinically

Physiological Effects of Hypothermia with Multiple Trauma Injured Patients

- In response to hypothermia, the normal sympathetic nervous system releases catecholamines and causes alterations in the coagulation cascade.
- This is enhanced by the Systemic Inflammatory Response (SIRS), making the effects faster and more complicated.
Physiology of Temperature Control

Conduction
Transfer of heat by direct contact down the temperature gradient.

Convection
Transfer of heat by movement of heated material (i.e. wind)

Radiation
Loss of heat from non-insulated areas

H₂O Evaporation
Loss of heat through exhalation

Hypothermia
Classification

- MILD 32 – 35 degrees C (90 to 95°F)
  - thermogenesis still possible
  - Thermogenesis is the process by which the body generates heat, or energy, by increasing the metabolic rate above normal

- MODERATE 28 – 32 degrees C (82-90°F)
  - progressive failure of thermogenesis

- SEVERE less than 28 degrees C
  - spontaneous cardiac arrest

Naked in a cold room

Cold Fluids

Intoxication and Paralysis

Decreased O₂ consumption

Hypothermia: Factors affected by Temperature

- Demonstrated on animals
- In induced hypothermia:
  - Impaired hemostasis occurs at ph 7.20
  - Impaired Platelet function

Evidence Base Medicine

Hypothermia: Factors affected by Temperature (cont.)

- In 112 Trauma Patients analyzed the effects of hypothermia on trauma patients
  - Hypothermia accounts for 6 times variation in acidosis than injury severity.
  - Acidosis therefore appears to be a sequela of hypothermia rather than injury severity.
  - This study supports acidosis contributes to Coagulopathy as a result of hypothermia and not independent of it.
Hypothermia

Osborn Wave

J-wave (Osborn wave) camel hump sign
Highly suggestive of hypothermia
May appear at any temp < 32 C
Commonly seen in lead 2 or V6

Measurement Instruments

<table>
<thead>
<tr>
<th>Routes</th>
<th>Drawbacks</th>
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<tr>
<td>Tympanic</td>
<td>▲ in ambient temp.</td>
</tr>
<tr>
<td>Skin</td>
<td>▲ in ambient temp.</td>
</tr>
<tr>
<td>Oro-Nasopharynx</td>
<td>Varies based on location</td>
</tr>
<tr>
<td>Esophagus</td>
<td>Varies based on where probe is position because of gas movement in trachea</td>
</tr>
<tr>
<td>Bladder</td>
<td>Closest to PA</td>
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<tr>
<td>Rectal</td>
<td>Presence of gas and feces</td>
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<td><strong>PA Catheter</strong></td>
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Hypothermia Management

**Passive**

- Remove wet clotting
- Insulating blanket
- Raise ambient Temp.
- Keep patient dry
- Humidified Air
- Warm IV fluids and blood

**Active**

- Body Cavity Lavage
- Heated Inspired Air
- Bair Hugger
- CPB
- CAVR - MAP dependent

Hypothermia Rewarming Rates

- Warmed fluids/Heated O2/Warm Blankets – 1.2 °C/hr
- Bair Huggers 2.4° C/hr.
- Pleural and Peritoneal Lavage 6°C/hr.
- CAVR - 3 °C/hr
- CPB 0.5°C – 3.5°C/20minutes

Acidosis

Pathophysiology

Ph < 7.20 = decrease in cellular function causing failure to produce ATP (energy stores) to transport proteins across cells and slows or stops transmission of messages

Driving in a Snow Storm
Definition in Multiple Trauma Patient

**Acidosis**

- Shock induced Metabolic Acidosis - Results from inadequate tissue perfusion
- Tissue hypoxia alters cellular function shifts from aerobic to anaerobic metabolism resulting in lactic acidosis.

<7.20

**Arrhythmias**

- Poor Cardiac performance
- Vasodilatation, loss of responsiveness
- Catecholamines Hypertension which further perpetuates shock and acidosis

**Base Deficit**

Base Deficit is the amount of alkali buffer required to titrate 1 L of blood to a Ph 7.40 at normal body temperature

**Lactate vs. Base Deficits**

- **Lactic Acidosis vs. Base Deficits**
  - Lactate is a direct correlation with
    - byproduct of anaerobic metabolism
    - elevated in hypoperfused states and prevents pyruvate from entering the Krebs cycle
    - total oxygen debt (imbalance between needs and consumption)
    - Magnitude of hypoperfusion
    - Severity of Shock
    - < 4 goal for lactate
    - Base Deficit
    - good prognostic guide to resuscitation efforts after the acute phase.
    - < 6 goal for resuscitation

- **Lactic Acidosis vs. Base Deficit** (cont.)
  - If lactic acid or base deficit is worsening ---- the type of resuscitation should be re-evaluated.
  - The lactic acid remains sensitive to lactate and anaerobic metabolism
  - If normal lactate clearance occurs within 24 hours. Survival rates increase 90%
  - Bicarbonate will alter relationship
**Acidosis Intervention**

- Improve tissue oxygenation and perfusion
  - treat hypothermia
  - maintain hemodynamic stability
  - ensure patient is well oxygenated
  - Try to avoid IV bicarbonate...may cause alkalosis
  - Ph 7.20

**Oxygen Dissociation Curve**

- In the hypothermic trauma patient: O2 cannot be released from the hemoglobin to the tissue
- Shivering
- Mottling

**Acidosis Intervention**

The Oxygen Consumption and Oxygen Demand

\[(1.39 \times Hb) \times \text{art. saturation} + (PaO2 \times 0.003) = \text{ml O2 / 100 ml blood} = CaO2\]

\[CaO2 \times CO \times 10 = DO2\]

**Coagulopathy**

- Hemorrhage is the leading cause of death
  - Bleeding from wounds
  - DIC
- Hypothermia slows clotting, production of factors and platelet aggregation
- Acidosis affects platelets and fibrinolysis

**Coagulopathy Pathophysiology**

- In temperatures ~33C, pt.s clotting factor act similarly to conditions where clotting factors are 50% concentration.
- Remember Lab values are done at 37C
- Lab values may disguise coagulopathy
- In studies involving pigs decreased clotting time was observed when lactic acid was added.
Coagulopathy Pathophysiology

- Hemodilution is a cause of coagulopathy.
- Amount of volume is directly proportional to coagulopathy regardless of fluid type.
- Colloids – fibrinogen dysfunction produces poor clot stability
- Coagulopathy occurs the first half hour
- Hypoperfusion increases this process.

Coagulopathy Definition

- PT > 14 seconds (1.5 x norm)
- PTT > 34 seconds (1.5 x norm)
- Platelets < 100,000 in severely injured patients
- Raised hematocrit (@2% per degree temp drop)

There is no single reliable laboratory test to confirm Coagulopathy.

Clinical Assessment of Coagulopathy

- Ongoing blood loss
- Oozing wounds
- Oozing vascular sites
- Oozing oral mucosa

Consumption Coagulopathy Resuscitation

Treatment

- Increase INR > 2.0 plus ↑ PTT – FFP
- Increase PT and PTT – Vitamin K
- Fibrinogen Levels – Cryoprecipitate, Platelets
- D Dimer – use of fibrin products for clotting

- 1 unit of plts/kg.
- 1 unit cryo./kg.
- 1 unit pRBC’s = 3 % points

PATIENT RESUSCITATION

Massive Blood Transfusions

Biologically active cell membrane breakdown products platelet activating factors and dialkyglycerol

Neutrophil Activation Sensitive to Endothelial cells

Sensitive to Lungs and other cells

Capillary leaks

MSOF

T.R.A.L.I

Malone DL, Hess JR, Fingerhut A. Comparison of practices around the globe and suggestion for a massive transfusion protocol.

J Trauma, 2006

- Reviewed massive transfusion protocols from well-developed trauma systems in Denver, Houston, Helsinki, Sydney, and Baltimore.
- This group then presented a massive transfusion protocol based on the best data from their review.

1:1:1
**Indications for Early FFP, Cryoprecipitate, and Platelet Transfusion in Trauma**

Lloyd Ketchum, John R. Hess and Seppo Hiippala
J Trauma, 2006

- If clinically evident coagulopathy is prevented by the early use of FFP, subsequent blood product consumption is likely to be less.

- In massive transfusion, 1:1:1 PRBC: Plasma:platelets are indicated

**Massive Transfusion Tidbits**

- Monitor H/H
- Monitor Coags. – Give factors
  – Give all coags at the same time
- Warm all blood
- Autotransfusion – CPD - Phosphorous
- Monitor Calcium – Give Calcium

**Coagulopathy Treatment**

- Stop the Hemorrhage – Apply pressure
- Ligation of Bleeders
- Early Operative Management
- Damage Control Surgery
- Fluid Resuscitation
  - Permissive Hypotension in Penetrating Trauma
- Decreases mortality, Coagulopathy and survival

**Resuscitation Concepts**

- High volume pre-hospital worsens ED coagulation
- > 2000ml – worse coagulation, required more blood, higher organ failure and overall mortality.
- < 1500ml – less effects on system

**Colloid vs Crystalloid**

- Perel Systematic reviews 2011
- 65/90 Randomized controlled trials of crystalloid and colloids respectively.
  - Resuscitation with colloids reduces the risk of death compared to resuscitation with crystalloids.
  - Recommendation
    - use of crystalloids if they are effective rather than liberal use of colloid

**Hypertonic Saline**

Reduction in ICP by improving CO, oxygenation and decreasing edema administration of small volumes of hypertonic saline in TBI

- 2011 Burger – no significance difference in mortality at 28 days
- Study stopped due to mortality increases in subgroup receiving hypertonic saline but no blood transfusions within first day.
- No trials providing compelling evidence to support use of hypertonic saline
Concepts of Damage Control

• Goals
  – Stop the bleeding – surgical intervention
  • avoid prolong operative procedures – temporary
  • Paired with massive fluid resuscitation reduces mortality
  – Stop hypoperfusion
  – Correcting acidosis
  – Reversing hypothermia
  – Coagulation under control
  – Definitive corrective surgery later

Case Study

GSW to Hand

• 50 year old
• GSW to hand 9 months ago
• The patient used an ice pick to remove the bullet with Heroin as anesthesia
• No Medical Attention
• Continued to use an ice pick to debrided the wound when he felt “it looked bad”
• Came to the Hospital because of bleeding!

Clinical Assessment

• Airway - Patent
• Breathing – 24/min.
• Circulation – Pulse...Bleeding
• Disability (Neuro) – GCS 15
• Pain – 0/10!
• Temperature 35.0
• History – Heroin Addiction, Asthma
• Treated with Keflex po by relative with access to medications

Sequence of Events

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Sequence of Events (cont.)

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Case Study

Clinical Findings
- Brachial Artery Bleeding
- To OR for repair, amputation and debridement

Case Study
- Post Op bleeding occurred
- Oozing from Incision site
- Decreased BP high 70's for approx. 2 hours
- Re-intubated
- Temperature decrease to 34°C
- 2 #16 Peripheral IV,RSC Central line
- Patient Live after significant extremity lost.

Triangle of Death

Summar...