TRAVUA TRIAD OF DEATH: HYPOTHERMIA, ACIDOSIS AND COAGULOPATHIES

Jeff Solheim MSN RN-BC CEN CFRN FAEN

I. Hypothermia

<table>
<thead>
<tr>
<th>Classification</th>
<th>Traditional</th>
<th>Trauma Patient</th>
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</thead>
<tbody>
<tr>
<td>Mild</td>
<td>&lt; 35 - 32°C</td>
<td>35 - 34°C</td>
</tr>
<tr>
<td>Moderate</td>
<td>32 - 28°C</td>
<td>34 - 32°C</td>
</tr>
<tr>
<td>Severe</td>
<td>&lt; 28°C</td>
<td>&lt; 32°C</td>
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</tbody>
</table>

A. Predisposing factors:
1. Heat loss in the field
2. Resuscitation maneuvers
3. Injury severity (most heat loss with injuries to the extremities, pelvis, abdomen, and large blood vessels)
4. Elevated blood alcohol levels
5. Exposure of body cavities during surgery
6. Use of anesthetics/paralytics
7. Impaired thermogenesis (tissue oxygen debt, hypoxic hypothalamus
8. Transfusion
9. Age

B. Effects of Hypothermia

<table>
<thead>
<tr>
<th>System</th>
<th>Effect</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cardiovascular System</td>
<td>☑️ Mild hypothermia - ↑ sympathetic activity and catecholamines (vasoconstriction, tachycardia, increased cardiac output, atrial and ventricular arrhythmias)</td>
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<tr>
<td></td>
<td>☑️ Severe hypothermia - ↓ heart rate and cardiac output, increasing vascular resistance</td>
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<td></td>
<td>☑️ &lt; 28°C – depression of myocardial contractility</td>
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<td></td>
<td>☑️ &lt;25°C – risk of ventricular fibrillation</td>
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<tr>
<td>Pulmonary System</td>
<td>☑️ ↑ respiratory rate which becomes increasingly depressed with worsening hypothermia</td>
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<tr>
<td></td>
<td>☑️ Pulmonary edema and excessive production of bronchial secretions with rewarming</td>
</tr>
<tr>
<td>Central Nervous System</td>
<td>☑️ Progressive depression of level of consciousness</td>
</tr>
<tr>
<td></td>
<td>☑️ ↓ Cerebral blood flow</td>
</tr>
<tr>
<td>Renal System</td>
<td>☑️ Increased urinary flow despite ↓ GFR and blood flow to the kidney</td>
</tr>
<tr>
<td>Electrolyte and acid-base equilibrium</td>
<td>☑️ ↑ potassium levels</td>
</tr>
<tr>
<td></td>
<td>☑️ Acidosis</td>
</tr>
</tbody>
</table>

Gregory et al found that hypothermia developed at some point in 57% of trauma patients studied, with the greatest temperature loss occurring in the Emergency Department.
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<table>
<thead>
<tr>
<th>Gastrointestinal and endocrine system</th>
<th>☑ Mild ileus</th>
<th>☑ Depressed hepatic function</th>
<th>☑ Hyperglycemia</th>
</tr>
</thead>
<tbody>
<tr>
<td>Metabolism</td>
<td>☑ ↓ metabolic rate → ↓ O₂ uptake, ↓ CO₂ production and ↑ solubility of CO₂</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Hematological system</td>
<td>☑ ↑ blood viscosity</td>
<td>☑ ↑ hct during rewarming</td>
<td>☑ Inhibition of clotting cascade</td>
</tr>
</tbody>
</table>

THE BOTTOM LINE: Hypothermia has detrimental effects on the outcome of severely injured patients. The lower the temperature is allowed to drop, the higher mortality climbs with 100% mortality at temperatures of 32°C.

C. Rewarming strategies:
1. Passive external measures:
   a. Remove blood or saline soaked dressings and blankets
   b. Increase ambient room temperature
   c. Decrease air flow over the patient
2. Active external measures
   a. Fluid circulating, convective air and aluminum space blankets
   b. Overhead radiant warmers
   c. Head covering
   d. Avoid covering exposed bowel with saline moistened towels in the OR (dry towels or plastic bags are superior).
3. Active internal measures
   a. Humidified and warmed ventilator circuits
   b. Heated body cavity lavage (gastric, bladder, colonic, pleural)
   c. Heated IV fluids
   d. Continuous arteriovenous rewarming (CAVR)
   e. Damage Control Surgical Intervention

II. Acidosis
A. Causes
   a. Inadequate tissue perfusion leads to metabolic lactic acidosis
   b. Multiple blood transfusions
   c. Excessive normal saline administration
   d. Hypoventilation
B. Deleterious effects of acidosis
   1. Impaired renal and hepatic blood flow
   2. Decreased cardiac contractility
   3. Vasodilation leading to hypotension
   4. Increased risk for ventricular dysrhythmias and bradycardia.
   5. Prolonged clotting times and increased bleeding times.
C. The bottom line
   1. Patient’s with an average serum pH of 7.29 have the highest survival potential.
   2. Serum pH below 7.2 causes deleterious effects on cardiovascular and coagulation.
C. Treatment considerations
   1. Aimed at correcting hypoperfusion
      a. Assure adequate resuscitation volume
      b. Transfusions
      c. Resuscitate to appropriate end-points
         i. Arterial pH
         ii. Base deficit
         iii. Lactate levels
         iv. Etc.

III. Coagulopathies
   A. Causes
      a. Primary causes – due to activation of the inflammatory system and activation of coagulation factors
      b. Secondary causes
         i. Dilution
         ii. Consumption
      c. Hypothermia
      d. Blood loss
      e. Acidosis
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B. Treatment

a. Permissive Hypotension
b. Local modalities
c. Antifibrinolytics
d. Recombinant activated factor VII (rVIIa)
e. 1:1:1 Resuscitation
f. Whole blood resuscitation

References