The Many Faces of Shock

The ABC’s of Shock and Trauma

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Objectives

- To understand the pathophysiology of shock
- To appreciate how shock influences the presentation and outcome of the trauma patient
- To discuss the role of a systematic approach to the trauma patient

History

- 1900’s: Influence of WW I / WW II
- 1940’s: Tissue anoxia defined as cause
- 1970’s: Swan Ganz catheter

Definitions

- Oxygen Delivery =
  - cardiac output x
  - (Hb g/dL x %sat x 1.36 cc/g)
  - + (Po2 x .003cc of O2/mm Hg/dL)
### Importance of Hemoglobin

- What is the ideal H/H for the critically ill patient?
- Consider complications of transfusion:
  - transmission of infection
  - immunosuppression
  - transfusion reactions
  - cost

### Transfusion Oxygen Support

**TABLE 2. Hemodynamic effects of transfusion***

<table>
<thead>
<tr>
<th></th>
<th>Before</th>
<th>After</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>MAP (mmHg)</td>
<td>83 ± 2</td>
<td>81 ± 2</td>
<td>NS</td>
</tr>
<tr>
<td>Heart rate (beats/min)</td>
<td>121 ± 4</td>
<td>115 ± 4</td>
<td>.02</td>
</tr>
<tr>
<td>Cardiac output (l/min)</td>
<td>9.9 ± 7</td>
<td>9.9 ± 6</td>
<td>NS</td>
</tr>
<tr>
<td>Cardiac index (l/min/m²)</td>
<td>5.2 ± 4</td>
<td>5.0 ± 4</td>
<td>NS</td>
</tr>
<tr>
<td>PCWP (mmHg)</td>
<td>14 ± 1</td>
<td>14 ± 1</td>
<td>NS</td>
</tr>
<tr>
<td>SVR (mmHg/l/min)</td>
<td>8 ± 8</td>
<td>7 ± 5</td>
<td>NS</td>
</tr>
<tr>
<td>PVR (mmHg/l/min)</td>
<td>1.3 ± 2</td>
<td>1.4 ± 2</td>
<td>NS</td>
</tr>
</tbody>
</table>

*Hemodynamic effects of red cell infusion in patients studied.*

### Effects of transfusion on oxygen transport

<table>
<thead>
<tr>
<th></th>
<th>Before</th>
<th>After</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hgb (g/dL)</td>
<td>6.4 ± 2</td>
<td>10.1 ± 2</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>Hct (%)</td>
<td>24 ± 5</td>
<td>29 ± 7</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>CO (l/min)</td>
<td>9.9 ± 7</td>
<td>9.9 ± 5</td>
<td>NS</td>
</tr>
<tr>
<td>CI (l/min/m²)</td>
<td>5.2 ± 4</td>
<td>5.0 ± 4</td>
<td>NS</td>
</tr>
<tr>
<td>CAO₂ (ml/O₂)</td>
<td>11.3 ± 3</td>
<td>12.7 ± 4</td>
<td>.004</td>
</tr>
<tr>
<td>VO₂ (ml/min)</td>
<td>7.8 ± 3</td>
<td>9.4 ± 4</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>DVO₂ (ml/min)</td>
<td>1047 ± 92</td>
<td>1342 ± 69</td>
<td>NS</td>
</tr>
<tr>
<td>VO₂ (ml/O₂)</td>
<td>345 ± 33</td>
<td>315 ± 16</td>
<td>NS</td>
</tr>
<tr>
<td>O₂ Ext (%)</td>
<td>32 ± 2</td>
<td>36 ± 1</td>
<td>&lt;.001</td>
</tr>
</tbody>
</table>

*Oxygen transport effects of red cell infusion in patients studied.*

### Hemodynamics

- Cardiac performance determined by:
  - preload
  - afterload
  - contractility
  - heart rate
Hypovolemic Shock

- Causes
  - external or internal bleeding
  - gastrointestinal losses
  - third space losses

Classification of Hemorrhage

<table>
<thead>
<tr>
<th>Class</th>
<th>Clinical Signs</th>
<th>% Volume Loss</th>
</tr>
</thead>
<tbody>
<tr>
<td>I.</td>
<td>Tachycardia</td>
<td>15</td>
</tr>
<tr>
<td>II.</td>
<td>Orthostatic Hypotension</td>
<td>20-25</td>
</tr>
<tr>
<td>III.</td>
<td>Supine Hypotension Oligura</td>
<td>30-40</td>
</tr>
<tr>
<td>IV.</td>
<td>Obtundation Cardiovascular Collapse</td>
<td>Over 40</td>
</tr>
</tbody>
</table>
Hypovolemic Shock

- **Crystalloids**
  - + low cost and well tolerated
  - - exits vasculature quickly
- **Colloids**
  - + remains intravascular longer
  - - cost and risk of infection transmission

Influence of Catheter Size on Infusion Rate

<table>
<thead>
<tr>
<th>Infusion Device</th>
<th>Length (inches)</th>
<th>Flowrate* (ml/min)</th>
</tr>
</thead>
<tbody>
<tr>
<td>9-French Introducer</td>
<td>5½</td>
<td>247</td>
</tr>
<tr>
<td>IV Extension Tubing</td>
<td>12</td>
<td>220</td>
</tr>
<tr>
<td>Peripheral</td>
<td></td>
<td></td>
</tr>
<tr>
<td>14-Gauge Catheter</td>
<td>2</td>
<td>195</td>
</tr>
<tr>
<td>16-Gauge Catheter</td>
<td>2</td>
<td>150</td>
</tr>
<tr>
<td>Central</td>
<td></td>
<td></td>
</tr>
<tr>
<td>16-Gauge Catheter</td>
<td>5½</td>
<td>91</td>
</tr>
<tr>
<td>16-Gauge Catheter</td>
<td>12</td>
<td>54</td>
</tr>
</tbody>
</table>

Endpoints of Resuscitation

- **Abramson et al 1993**
  - 100% survival if lactic acid levels normalized within 24 hours
  - 78% survival between 24-48 hours
  - 14% survival > 48 hours

Comparison of the three endpoints

<table>
<thead>
<tr>
<th>Base deficit</th>
<th>Lactate</th>
<th>pH</th>
</tr>
</thead>
<tbody>
<tr>
<td>Arterial blood gas</td>
<td>Yes</td>
<td>No</td>
</tr>
<tr>
<td>Invasiveness</td>
<td>No</td>
<td>No</td>
</tr>
<tr>
<td>Availability</td>
<td>Wide</td>
<td>Limited</td>
</tr>
<tr>
<td>Speed</td>
<td>Fast</td>
<td>Varies</td>
</tr>
<tr>
<td>Scientific basis</td>
<td>Retrospective</td>
<td>Studies</td>
</tr>
<tr>
<td>Cost</td>
<td>Modestly expensive</td>
<td>Least expensive</td>
</tr>
<tr>
<td>Risk</td>
<td>Minimal</td>
<td>Least expensive</td>
</tr>
</tbody>
</table>

Septic Shock

- **Infectious causes**
  - cholangitis
  - pneumonia
  - peritonitis
  - pyelonephritis
  - meningitis
- **Inflammatory causes**
  - crush injuries
  - burns
  - pancreatitis
  - anaphylaxis

Septic Shock

- **Presentation**
  - hyper or hypothermic
  - tachycardic
  - tachypneic
  - leukocytosis or leukopenia
  - thrombocytopenia
  - DIC
Septic Shock

- **SG parameters:**
  - compensated
    - CO increased
    - low wedge pressure
    - decreased SVR
  - uncompensated
    - CO decreased
    - high SVR
    - low oxygen consumption

- **Complement Cascade**
  - C3a, C5a
  - increases vascular permeability, vasodilation, chemotaxis

- **Plasma Kinins**
  - Bradykinin
  - increases vascular permeability, vasodilation, edema

Complement Cascade
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Plasma Kinins
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Cytokines

<table>
<thead>
<tr>
<th>Cytokine</th>
<th>Source</th>
<th>Activity</th>
</tr>
</thead>
<tbody>
<tr>
<td>TNFα, IL-1, IL-6, IFNγ</td>
<td>Activated T cells, monocytes, macrophages, neutrophils</td>
<td>Increases vascular permeability, neutrophil chemotaxis</td>
</tr>
<tr>
<td>IL-1β, IL-6, IL-10</td>
<td>Activated T cells, monocytes, macrophages</td>
<td>Increases vascular permeability, neutrophil chemotaxis</td>
</tr>
<tr>
<td>IL-12, IL-23</td>
<td>Activated T cells</td>
<td>Increases vascular permeability, neutrophil chemotaxis</td>
</tr>
<tr>
<td>IL-18, IFNγ</td>
<td>Activated T cells, monocytes, macrophages</td>
<td>Increases vascular permeability, neutrophil chemotaxis</td>
</tr>
<tr>
<td>IL-22</td>
<td>Activated T cells, monocytes, macrophages</td>
<td>Increases vascular permeability, neutrophil chemotaxis</td>
</tr>
<tr>
<td>IL-23</td>
<td>Activated T cells, monocytes, macrophages</td>
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</tr>
<tr>
<td>IL-27</td>
<td>Activated T cells, monocytes, macrophages</td>
<td>Increases vascular permeability, neutrophil chemotaxis</td>
</tr>
<tr>
<td>IL-28</td>
<td>Activated T cells, monocytes, macrophages</td>
<td>Increases vascular permeability, neutrophil chemotaxis</td>
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<tr>
<td>IL-29</td>
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<td>IL-30</td>
<td>Activated T cells, monocytes, macrophages</td>
<td>Increases vascular permeability, neutrophil chemotaxis</td>
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<tr>
<td>IL-31</td>
<td>Activated T cells, monocytes, macrophages</td>
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<td>Activated T cells, monocytes, macrophages</td>
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<td>IL-36</td>
<td>Activated T cells, monocytes, macrophages</td>
<td>Increases vascular permeability, neutrophil chemotaxis</td>
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<td>Activated T cells, monocytes, macrophages</td>
<td>Increases vascular permeability, neutrophil chemotaxis</td>
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<td>Activated T cells, monocytes, macrophages</td>
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<td>IL-40</td>
<td>Activated T cells, monocytes, macrophages</td>
<td>Increases vascular permeability, neutrophil chemotaxis</td>
</tr>
<tr>
<td>IL-41</td>
<td>Activated T cells, monocytes, macrophages</td>
<td>Increases vascular permeability, neutrophil chemotaxis</td>
</tr>
<tr>
<td>IL-42</td>
<td>Activated T cells, monocytes, macrophages</td>
<td>Increases vascular permeability, neutrophil chemotaxis</td>
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</tbody>
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Cardiogenic Shock

- **Presentation**
  - c/o chest pain, diaphoresis, confusion
  - appearance may be ashen or cyanotic with cool skin and mottled extremities
  - JVD and pulmonary rales
  - 3rd and 4th heart sounds
Cardiogenic Shock

- **Swan Ganz findings:**
  - A-V O$_2$ difference $> 5.5$ ml/dl
  - CI $< 2.2$ L/min/m$^2$
  - wedge $> 15$mmHg

Table 4.4  Pharmacologic Prevention and Clinical Effects of Selected Vasoactive Drugs.

<table>
<thead>
<tr>
<th>Drug</th>
<th>Prevention</th>
<th>Clinical Effect</th>
</tr>
</thead>
<tbody>
<tr>
<td>Dopamine</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>Epinephrine</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>Norepinephrine</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>Phenylephrine</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>Terlipressin</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>Vasopressin</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>Nitroprusside</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>Nitroglycerine</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
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<td>Nitroglycerine</td>
<td>-</td>
<td>-</td>
</tr>
</tbody>
</table>

Mortality Among Study Patients

- **Strengths of Hochman paper**
  - prospective randomization
  - outcomes similar for revascularized study and non-study population
- **Weaknesses of Hochman paper**
  - lack of statistical power to detect 9% difference in mortality at 30 days
  - small sample of pts $> 75$ years

Cardiogenic Shock

- **Recommendations**
  - early revascularization with CABG or angioplasty
  - further study into revascularization in pts older than 75 years
Neurogenic Shock

**Causes**
- traumatic cervical spinal cord transection
- spinal anesthesia
- acute gastric dilation
- drugs

**Presentation**
- hypotensive
- bradycardic
- pink, warm skin
- obvious neurologic deficits

**Swan parameters**
- CO may be low or normal
- decreased SVR
- decreased PVR
- decreased CVP

**Treatment**
- R/O other causes of shock
- hemodynamic stabilization
- control spine

**Role of Steroids**
- NASCIS II trial
  - randomized, controlled, double blinded
  - methylprednisolone vs naloxone vs placebo
  - treatment for 23 hours
  - blunt trauma pts only
  - outcomes at 6 weeks and 6 months
Role of Steroids

**NASCIS III**
- treated pts with methylprednisolone or tirilazad mesylate
- treatment to 23 or 48 hours
- blunt trauma pts only
- functional independence measure
- outcomes at 6 weeks and 6 months

**Shortcomings of NASCIS II**
- creation of subgroups based on data
- some subgroup sizes too small
- outcomes not correlated clinically

**Shortcomings of NASCIS III**
- no placebo group
- subgroups based on data

Role of Steroids

**Important findings of NASCIS**
- overall mortality rates similar between pts treated with steroids and control group
- FIM significantly improved in overall function, self care, and sphincter control

**Recommendations for treatment of spinal cord injuries after blunt trauma:**
- within 3 hours of injury treat with 23 hours of high dose methylprednisolone
- within 3-8 hours of injury treat with 48 hours of this steroid
- beyond 8 hours of injury do not administer steroids

Four Stages Care of Trauma Patient

- Primary survey
- Resuscitation
- Secondary survey with diagnostic evaluation
- Definitive care plan
Primary Survey
- A - airway with C-spine protection
- B - breathing
- C - circulation
- D - disability:
  - neurologic disability
- E - exposure / environment control:
  - completely undress the patient but prevent hypothermia
- It is designed to identify all immediate life threatening injuries!

Resuscitation
- Simultaneously with primary survey
- Airway is secured; ventilation is performed
- Intravenous lines are established and fluid resuscitation with balanced fluid solution is done
- Gross bleeding is controlled
- Neurologic deficit is identified
- Patient is exposed

The Secondary Survey
- Detail physical examination from head to toes
- Identify life threatening and occult injuries
- Tests:
  - CBC
  - basic chemistries
  - ABG, XR:
    - Lat C-spine
    - AP CXR, pelvic film
    - blood for type and cross match

After Secondary Survey
- Re-exam the patient
- Re-check the vital signs
- Prioritize the injuries
- Definitive plan

Airway Management
- Protecting, providing, and maintaining airway
- Failure to obtain an airway - a common cause of preventable death
- Cause: simple injuries; intoxication
- Think airway-with sudden deterioration

Risks for Airway Problems
- Injuries to the head or neck that cause obstruction or have an altered level of consciousness (injury, alcohol, drugs)
- GCS < 8 requires definitive airway
- Facial injuries
- Neck injuries (tracheal, vascular)
Identification of airway problem

- Talk to the patient
- Listen for abnormal airway: stridor, snoring, gurgling - all suggest supraglottic injuries
- Dysphonia or pain on speaking implies obstruction at laryngeal level

Airway Management

- Rule #1:
  Always assume that patient has neck injury
  - maintain immobilization in neutral position
  - avoid: hyperextension, hyperflexion or rotation of the neck
- Rule #2
  Lack of neurologic deficit does not exclude cervical spine injury

Airway Management

- Chin lift, jaw thrust, suction:
  - airway secured - oral or nasal airway:
    - no spontaneous ventilation - ventilate with bag
  - spontaneous breathing - supply oxygen
- Airway at risk: Endotracheal intubation
- Failed orotracheal intubation: surgical airway

Breathing

- Airway patency does not assure adequate ventilation
- Lung function, chest wall, diaphragm
- Expose; auscultate, palpate
- Identify pulmonary pathology
Tension Pneumothorax: Diagnosis
- Respiratory distress, subcutaneous emphysema, hypotension, distended neck veins
- Tracheal deviation away from the affected side
- Decreased breath sound on the affected side

Tension Pneumothorax: Treatment
- Needle decompression
- Tube thoracostomy

Flail Chest
- Two or more ribs fractured in at least two places with paradoxical movement
- Associated lung contusion underestimated on CXR
- Respiratory failure may not be immediate

Breathing: Pitfalls
- Dyspneic or tachypnic patient: think problem with airway
- In a ventilated patient, vigorous bagging or positive pressure ventilation may compromise ventilation
- CXR after intubation

Circulation
- Exam the pulses:
  - a SBP of 60 - palpable carotid pulse
  - a SBP of 70 - palpable femoral pulse
  - a SBP of 80 - palpable radial pulse
- Assume: hypotension from hemorrhage
- Control external bleeding before restoring circulation
- Manual compression; avoid blind clamps

The Treatment: Initial Step
- Intravascular replacement
- In adults: initial bolus 2 liters
- In children: 20 cc/kg/body weight
- Blood and blood product- type specific or O-negative
- Adjunct: Pneumatic antishock garment (PASG)- buckled into place around the legs, pelvic fractures
- Release them gradually
**Assessment of Organ Function**
- Level of consciousness
- Temperature
- Skin color
- Pulse rate and character

**Circulatory Management**
- Control hemorrhage
- Restore volume
- Reassess
- Caution:
  - elderly
  - athletes
  - children
  - medications

**Disability**
- Baseline neurologic evaluation
- GCS scoring
- Pupillary response
- Observe for deterioration

**Disability**
- Glasgow Coma Scale
  - eye (1-4)
  - verbal (1-5)
  - motor (1-6)
  - worst: 3 best: 15

**Exposure/Environment**
- Disrobe patient
  - necessary to completely evaluate patient for all injuries
  - log roll patient to look at back and spine
- Maintain normothermia
  - necessary to avoid problems with coagulopathy

**Adjuncts to primary survey**
- Vital signs
- Pulse oximeter and CO2
- Urinary and gastric catheters unless contraindicated
- Laboratory values
- Plain films
- ECG
Secondary survey

- Head to toe exam
- Finger in every orifice
- Reevaluate hemodynamics
- Determines subsequent work up and interventions
  - FAST, CT’s, Angio, OR

Case Presentation

- 50 years old male, found in the parking lot. Unknown mechanism or history. Brought to ER. Hemodynamically unstable. 2 chest tubes, 6 uPRBC, 4 Liters of crystalloid BP 120s.
- To the CT scan: Unstable again
- To OR emergently
- What might one find?
Conclusions

- Shock is fundamentally a deficiency in tissue oxygen perfusion
- Hypovolemic shock is the most commonly encountered form in the trauma patient
- A systematic approach is vital to provide efficient and effective care