Trauma: A Worldwide Crisis

- Leading cause of death between the ages of 15–44 years of age.
- Cost in US due to MVC per year approximately 230.6 billion representing 2.3% of the gross domestic product.
- According to the most recent 7th World Conference on Injury Prevention and Safety Promotion sponsored by the World Health Organization, violence–related injuries account for a tremendous financial burden to society with expenditures nearing 4% of the gross domestic product.
How does a human respond to stress?

- Physician and physiologist (Harvard, ~1900)
- 1930, coined "homeostasis" implied controls that restore abnormal states to normal
- Used Starling’s phrase *Wisdom of the Body* as title of a popular book (1932) describing same
- First use of "stress" in a biological (vs. engineering) context

Shock

...failure to deliver and/or utilize adequate amounts of oxygen may include, but is not limited to the presence of hypotension.

- **Cellular level:**
  - Reduction of mitochondrial oxygen
  - Anaerobic glycolysis of ATP
  - Accumulation of pyruvate  
    - Lactatic Acidosis
Shock

“A rude unhinging of the machinery of life”

“A brief pause in the act of dying”

Stabilize (Golden Hour)

- First described by R. Adams Cowley in 1963
- Restore circulating blood volume
- Ensure adequate supply of oxygen to tissue
- Recover normal temperature
- Replenish coagulation factors

Shock Decompensation
Multiorgan Dysfunction Syndrome (MODS)

- Progression of physiologic effects as shock ensues
- Cardiac depression
- Respiratory distress
- Renal failure
- DIC
- Result is end organ failure

Epidemiology of Trauma Deaths

Initial Approach to Injured Patient

- ABCs
  - Cardiorespiratory monitor
  - Pulse oximetry
  - Supplemental oxygen
  - IV access
  - Foley catheter
  - Vital signs including temperature
### Diagnosis and recognition of shock

- Physical exam (VS, mental status, skin color, temperature, pulses, neurological exam)
- EKG
- Labs:
  - CBC
  - Chemistries
  - Lactate
  - Coagulation studies
  - ABC
  - Type and cross

### Shock Classification

<table>
<thead>
<tr>
<th>Class</th>
<th>Class I</th>
<th>Class II</th>
<th>Class III</th>
<th>Class IV</th>
</tr>
</thead>
<tbody>
<tr>
<td>Circulating Volume Loss %</td>
<td>&lt;15</td>
<td>15-30</td>
<td>&gt;30-40</td>
<td>&gt;40</td>
</tr>
<tr>
<td>Heart Rate (beats/min)</td>
<td>&lt;100</td>
<td>Tachycardia</td>
<td>Tachycardia</td>
<td>Marked Tachycardia</td>
</tr>
<tr>
<td>Pulse Pressure</td>
<td>Normal</td>
<td>Narrowed</td>
<td>Narrowed</td>
<td>Unobtainable or Very Narrowed</td>
</tr>
<tr>
<td>Systolic Blood Pressure</td>
<td>Normal</td>
<td>Minimal Decrease</td>
<td>Decrease</td>
<td>Significant decrease</td>
</tr>
<tr>
<td>Hourly Urine Output</td>
<td>≥ 0.5 cc/kg</td>
<td>≤ 0.5 cc/kg</td>
<td>≤ 0.5 cc/kg</td>
<td>Minimal</td>
</tr>
<tr>
<td>Mental Status</td>
<td>Normal</td>
<td>Anxious</td>
<td>Confused and anxious</td>
<td>Markedly depressed or lethargic</td>
</tr>
</tbody>
</table>

### Further Evaluation

- CXR, Pelvis X-ray
- Lateral C-spine X-ray
- eFAST and/or DPA/DPL
- CT Head, CT Chest, CT Abd/pelvis
- Extremity X-rays
- Evaluation of other shock etiologies (cardiogenic, septic)
Goals of Treatment

- ABCDE of shock resuscitation
  - Airway
  - control work of Breathing
  - optimize Circulation
  - assure adequate oxygen Delivery
  - achieve End points of resuscitation

Airway

- Determine need for intubation but remember: intubation can worsen hypotension
  - Sedatives can lower blood pressure
  - Positive pressure ventilation decreases preload
  - May need volume resuscitation prior to intubation to avoid hemodynamic collapse

Control Work of Breathing

- Respiratory muscles consume a significant amount of oxygen
  - Tachypnea can contribute to lactic acidosis
  - Mechanical ventilation and sedation decrease WOB and improves survival
Optimizing Circulation

- Isotonic crystalloids
- Titrated to:
  - CVP 8–12 mm Hg
  - Urine output 0.5 mL/kg/hr (30 mL/hr)
  - Improving heart rate
- May require 4–6 L of fluids
- No outcome benefit from colloids

- In patient with un–compensated shock consider early blood transfusion

Maintaining Oxygen Delivery

- Decrease oxygen demands
  - Provide analgesia and anxiolytics to relax muscles and avoid shivering
  - Maintain arterial oxygen saturation/content
    - Give supplemental oxygen
    - Maintain Hemoglobin > 10 g/dL (initially)
  - Serial lactate levels, base deficit or central venous oxygen saturations to assess tissue oxygen extraction

End Points of Resuscitation

- Goal of resuscitation is to maximize survival and minimize morbidity
- Use objective hemodynamic and physiologic values to guide therapy
- Goal directed approach
  - Urine output > 0.5 mL/kg/hr
  - CVP 8–12 mmHg
  - MAP 65 to 90 mmHg
  - Central venous oxygen concentration > 70%
Practically Speaking....

- Consider shock in all patients
- Frequent vitals signs:
  - Monitor success of therapies
  - Watch for decompensated shock
- Until shock ruled out, carefully monitor patient and do not perform unnecessary studies.
- Physical signs of shock may not be reliable

Validity and Reliability of Physical Signs in Shock Assessment

<table>
<thead>
<tr>
<th>Finding</th>
<th>Sensitivity</th>
<th>Specificity</th>
<th>PPV</th>
<th>NPV</th>
</tr>
</thead>
<tbody>
<tr>
<td>Skin Elasticity</td>
<td>0.35</td>
<td>0.97</td>
<td>0.57</td>
<td>0.93</td>
</tr>
<tr>
<td>Cap refill &lt; 2 sec</td>
<td>0.48</td>
<td>0.96</td>
<td>0.57</td>
<td>0.94</td>
</tr>
<tr>
<td>General Appearance</td>
<td>0.59</td>
<td>0.91</td>
<td>0.42</td>
<td>0.95</td>
</tr>
<tr>
<td>Abnormal Breathing</td>
<td>0.43</td>
<td>0.86</td>
<td>0.37</td>
<td>0.94</td>
</tr>
<tr>
<td>Dry Mucous Membrane</td>
<td>0.80</td>
<td>0.78</td>
<td>0.29</td>
<td>0.99</td>
</tr>
<tr>
<td>Sunken Eyes</td>
<td>0.60</td>
<td>0.84</td>
<td>0.29</td>
<td>0.95</td>
</tr>
<tr>
<td>Abnormal Radial Pulse</td>
<td>0.43</td>
<td>0.86</td>
<td>0.25</td>
<td>0.93</td>
</tr>
<tr>
<td>Tachycardia</td>
<td>0.46</td>
<td>0.79</td>
<td>0.20</td>
<td>0.93</td>
</tr>
<tr>
<td>Decrease Urine Output</td>
<td>0.85</td>
<td>0.53</td>
<td>0.17</td>
<td>0.97</td>
</tr>
</tbody>
</table>

The Problem

- Traditional hemodynamic markers such as blood pressure, heart rate and urine output can guide resuscitation during "uncompensated shock".
- A condition of hemodynamic through ongoing inadeq

ick" occurs when been stabilized snisms despite ation.
Pitfalls

- Distinguishing hypovolemic shock from cardiogenic, neurogenic and septic shock
- Extremes of age and medications
- Low urine output may be due to renal or post-renal causes
- High urine output may be due to osmotic diuretics or inability of the kidney to concentrate urine
- Blood pressure changes are late signs of hypovolemia

Compensation of Child and Adult

- Graph showing compensation in children and adults.

Patient Scenerio

- 76 year old male s/p MVC
- Pre-Hospital
  - GCS 3, Intubated for airway control
  - Vitals HR 70, SBP 110
  - Two large bore IVs established, 1 liter crystalloid administered
  - C-spine and spine protected
  - Patient transferred to HMC
On HMC arrival
- Airway verified, EtCO2 20
- SaO2 91%, HR 70, SBP 100
- Decreased breath sounds left chest
- Left chest tube inserted, 200 cc blood returned.
- Is patient in shock?

Progressive relative hypotension with development of cardiopulmonary arrest.
- Resuscitative thoracotomy performed with aortic cross clamping, and open cardiac massage.
- Eventual return of spontaneous circulation...taken to OR.
- Could cardiopulmonary collapse be detected earlier? What factors played a role in under-recognition?

STOP
Hemodynamic Goals
- Optimization of oxygen delivery to tissue
- Oxygen delivery depends on:
  - Cardiac output
  - Hemoglobin
  - Oxygen saturation

Cardiac Output
- Cardiac Output = SV x HR
- Stroke Volume Components
  - Preload
  - Afterload
  - Contractility
- Cardiac Index
  - CO/BSA
  - Normal 2.5 - 4.0 L/minute/m²

Heart Rate
- Oxygen delivery depends on arterial oxygen content, hemoglobin concentration, and heart rate.
- Maximizing heart rate will result in improved oxygen delivery.
- Optimal heart rate is between 60 and 100 bpm; however, excessive heart rates may be deleterious.
**Preload**

RA — RV — PA lungs — PV — LA — LV

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**Preload Assessment: Passive Leg Raising Predicts Fluid Responsiveness In Shock**


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**Preload Assessment (Right Side)**

- Central Venous Pressure (CVP)
- Normal CVP 2 – 7
- Critically ill with low CI, may push CVP to 14 – 18
Preload Assessment (Left Side)
- Pulmonary Capillary Wedge Pressure (PCWP)
- Normal 6–12 mmHg
- In critically ill, 16–20

FRANK–STARLING LAW

Problems with CVP and PCWP
Preload determination
- Echocardiography
- Esophageal Doppler

Afterload Optimization
- The mainstay of present-day resuscitation from hemorrhagic shock is the rapid restoration of circulating blood volume.
- Controversy exists regarding this principle. Specifically, bleeding may be exacerbated as a result of a delusional coagulopathy and secondary clot disruption.

Optimal blood pressure in penetrating trauma
- Bickell et al. evaluated the benefit of delayed fluid resuscitation compared with immediate resuscitation in hypotensive patients who sustained penetrating torso injuries.
Cardiac Output

- No randomized studies to demonstrate their benefit
- Indiscriminate or in appropriate use may be associated with complications
- Non-invasive methods of monitoring:
  - The esophageal Doppler monitor

\[
\text{EtCO}_2 = 35 \\
\text{PaCO}_2 = 40 \\ CO = 4L
\]

\[
\text{EtCO}_2 = 20 \\
\text{PaCO}_2 = 50 \\ CO = 2L
\]
Cardiac Output Determined by CO₂ Production

Endtidal CO₂ Gradient in hemorrhagic shock

Utilization of EtCO₂ in initial shock assessment

Global Perfusion Endpoints

- **Oxygen delivery (DO2I)**
  - The presence of ongoing oxygen debt is the result of an imbalance between oxygen delivery and oxygen consumption at the cellular level.

- **Oxygen consumption (VO2I)**
  - Oxygen consumption (VO2I) is the difference in oxygen saturation between arterial and venous blood.

- Resuscitation to supranormal may even be harmful, whereas an increased incidence of abdominal compartment syndrome was demonstrated by Balogh et al.

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Supranormalization of Oxygen Delivery

![Graph](image)

<table>
<thead>
<tr>
<th></th>
<th>Supranormal Resuscitation</th>
<th>Normal Resuscitation</th>
</tr>
</thead>
<tbody>
<tr>
<td>Base deficit (BD)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>ACS</td>
<td>42</td>
<td>10</td>
</tr>
<tr>
<td>MODS</td>
<td>16</td>
<td>4</td>
</tr>
<tr>
<td>Mortality</td>
<td>22</td>
<td>5</td>
</tr>
</tbody>
</table>

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Global Perfusion Endpoints

- **Base deficit (BD)**
  - Base deficit is the amount of base (mEq/L) required to titrate 1 L of whole blood to a normal pH, assuming normal physiologic values of PaO2, PaCO2 and temperature.

- **Lactate**
  - Lactate accumulation is most notable under anaerobic conditions.
### Base Deficit as Indicator of Shock State

<table>
<thead>
<tr>
<th>BG Category</th>
<th>PMN x 10^9 (mm^-3)</th>
<th>Top PMN x 10^9</th>
<th>Total PMN x 10^9</th>
<th>Trend Power</th>
<th>Trend Power</th>
<th>Trend Power</th>
<th>Trend Power</th>
</tr>
</thead>
<tbody>
<tr>
<td>Base sodium</td>
<td>140 ± 12</td>
<td>130 ± 5</td>
<td>130 ± 5</td>
<td>0.01</td>
<td>0.01</td>
<td>0.01</td>
<td>0.01</td>
</tr>
<tr>
<td>Hematocrit</td>
<td>40 ± 5</td>
<td>40 ± 5</td>
<td>40 ± 5</td>
<td>0.01</td>
<td>0.01</td>
<td>0.01</td>
<td>0.01</td>
</tr>
<tr>
<td>Mean</td>
<td>30 ± 5</td>
<td>30 ± 5</td>
<td>30 ± 5</td>
<td>0.01</td>
<td>0.01</td>
<td>0.01</td>
<td>0.01</td>
</tr>
<tr>
<td>Hematocrit</td>
<td>0.6 ± 0.1</td>
<td>0.6 ± 0.1</td>
<td>0.6 ± 0.1</td>
<td>0.01</td>
<td>0.01</td>
<td>0.01</td>
<td>0.01</td>
</tr>
</tbody>
</table>

* * * * *


### Patient Scenario

- 21 year old cyclist s/p struck by MVC
- Brought in to ER with HR 120, SBP 100, RR 24
- Airway: Intact, talking
- Breathing: Slightly tachypnic, complaining of severe pelvic pain
- Circulation: Two large bore IVs with one liter crystalloid admixture

Is this patient in shock?

### Patient Scenario

- Sent to CT of Abd/pelvis
- Arreets in CT scan, and despite resuscitative thoracotomy, aortic cross clamping and open cardiac massage, patients dies in CT scanner.
- Initial ABG which was not reviewed prior to transfer to CT was 6.91/521/12/24/12/17.6 with lactate of 20.3

Why was shock underappreciated?
Regional Resuscitation Endpoints:
Gastric Tonometry

- Gastric tonometry takes advantage of the fact that the splanchnic vascular bed, as reflected by the gastric intramucosal pH (pHi), is the first to be affected during the onset of shock and is the last to be corrected after resuscitation.
- The pHi decreases as splanchnic perfusion is reduced.

Regional Resuscitation Endpoints:
Sublingual Capnography

- Use is based on the premise that inadequate global tissue perfusion is reflected by systemic hypercarbia. As an extension of gastric tonometry, the sublingual mucosa serves as an excellent site to measure PCO2. Sublingual capnography is highly predictive of circulatory shock and correlates with increasing lactate levels.

Regional Resuscitation Endpoints:
Near-Infrared Spectroscopy (NIRS)

- NIRS allows for the optimization of oxygen delivery and consumption specifically at the tissue level. Tissue oxygen saturation (StO2) is derived from a complex algorithm of the ratio of absorption between the individual chromophores.
- In large animal hemorrhagic shock models, skeletal muscle StO2 as determined by NIRS showed close correlation with measurements of systemic oxygen delivery and was superior to that of lactate, base excess, or iOGD.
- McIntyre et al demonstrated changes in skeletal muscle StO2 to parallel DO2I during the resuscitation of severely injured trauma patients.
78 year old female involved in HSMVC
- Restrained driver
- Abrasion left chest/flank
- Intubated for GCS 7
- BP 136/68, HR 68

Initial ER vitals/labs:
- BP 146/88, HR 69, RR 18
- HCT: 31
- ABG: 7.36/39/101/32.4

Physical exam findings
- Large scalp laceration
- Abdomen: slightly distended, abrasion left flank

Is the patient in shock?
StO\textsubscript{2} Diagnosis of Shock

![Graph showing StO\textsubscript{2} values over time.]

StO\textsubscript{2} Diagnosis of Shock in the Elderly

- 67 year old male struck by motor vehicle on left side, presents with complaints of left sided chest pain
- Abrasion left flank
- Initial GCS 14
- BP 124/68, HR 94

StO\textsubscript{2} Diagnosis of Shock

- Initial ER vitals/labs:
  - BP 123/78, HR 83, RR 16
  - HCT: 36
  - ABG: 7.38/39/214/-1.2
- Physical exam findings
  - Crepitus and decreased breath sounds left chest
  - Abdomen: slightly distended, abrasion left flank
- Left chest tube placed, patient taken to CT.
- Is the patient in shock?
CT scan demonstrates renal laceration.
SBP decreased to 84
Patient transfused O+ PRBC.
HCT 21
Taken to angio for embolization.
StO2 response to fluid administration

<table>
<thead>
<tr>
<th>Response</th>
<th>Initial SBP</th>
<th>Resuscitated SBP</th>
<th>30 minute SBP</th>
<th>Initial StO2</th>
<th>Resuscitated StO2</th>
</tr>
</thead>
<tbody>
<tr>
<td>Responder</td>
<td>92 ± 11</td>
<td>100 ± 8</td>
<td>90 ± 5</td>
<td>72 ± 3</td>
<td>81 ± 7</td>
</tr>
<tr>
<td>Transient Responder</td>
<td>91 ± 6</td>
<td>101 ± 10</td>
<td>90 ± 5</td>
<td>72 ± 4</td>
<td>71 ± 5</td>
</tr>
<tr>
<td>Non-responder</td>
<td>89 ± 6</td>
<td>82 ± 11</td>
<td>85 ± 9</td>
<td>65 ± 9</td>
<td>62 ± 11</td>
</tr>
</tbody>
</table>

HMC Resuscitation Protocol

Treatment

- Stop the losses
  - Stop bleeding
- Temporary use of vasoactive drugs
- Fluid therapy
  - Blood and Blood Products
  - Colloids
  - Crystalloids
Blood

- If available, there is no question that autologous (self) fresh blood is the best resuscitation fluid
  - increased hemoglobin concentration will increase oxygen capacity
  - RBC will mostly remain in the intravascular space
  - In patients with active bleeding, blood cell transfusion is appropriate (not controversial)

<table>
<thead>
<tr>
<th>Blood Type</th>
<th>Typing</th>
<th>Antibody Screen</th>
<th>Crossmatch</th>
<th>Time</th>
</tr>
</thead>
<tbody>
<tr>
<td>Type O</td>
<td>No</td>
<td>No</td>
<td>No</td>
<td>Immediate</td>
</tr>
<tr>
<td>Type Specific</td>
<td>Yes</td>
<td>No</td>
<td>No</td>
<td>&lt;10 min</td>
</tr>
<tr>
<td>Type and Screen</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
<td>20–30 min</td>
</tr>
<tr>
<td>Type and crossmatch</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
<td>45–60 min</td>
</tr>
</tbody>
</table>

Not Fresh

- Storage of blood impairs RBC deformability and flow in the microcirculation
- Clog the capillaries?
- Multiple studies have demonstrated the inability of red blood cell transfusion to improve tissue hypoxia
Erythrocyte membrane deformability

- Capillary diameter: 3-5 \( \mu \)m
- RBC diameter: 6-8 \( \mu \)m

Effect of storage on RBC deformability

Colloids

- Theoretically:
  - preservation of plasma osmotic pressure
  - more efficient plasma volume expansion
  - decreased tissue and pulmonary edema
- However, clinical studies have not demonstrated a significant improvement in patient outcomes
Crystalloids

- Advantages
  - Cheap
  - Easy to store and warm
  - Established safety
  - Predictable rise in cardiac output
- Disadvantages
  - Large volumes needed
  - Dilutional coagulopathy
  - Increase cytokine activation
  - No oxygen carrying capacity
  - May increase ICP

Crystalloid Composition

<table>
<thead>
<tr>
<th></th>
<th>Na</th>
<th>Cl</th>
<th>K</th>
<th>Ca</th>
<th>Buffer</th>
<th>pH</th>
</tr>
</thead>
<tbody>
<tr>
<td>Plasma</td>
<td>141</td>
<td>103</td>
<td>4-5</td>
<td>5</td>
<td>Bicarb</td>
<td>7.4</td>
</tr>
<tr>
<td>0.9% NS</td>
<td>154</td>
<td>154</td>
<td>----</td>
<td>----</td>
<td>----</td>
<td>5.7</td>
</tr>
<tr>
<td>LR</td>
<td>131</td>
<td>111</td>
<td>2</td>
<td>3</td>
<td>Lactate</td>
<td>6.4</td>
</tr>
</tbody>
</table>

LR vs. NS

- No mortality difference
- LR
  - Lower overall volume
  - More buffering capacity
- NS
  - Metabolic acidosis
  - Dilutional coagulopathy
  - Preferred fluid outside of US
  - Probably no difference for prehospital or early fluid resuscitation.
Coagulopathy
- Multiple injury with multiple sites of bleeding
- Severe traumatic brain injury
- Dilution due to rapid administration of non-clotting fluids (crystalloid and blood)
- Rebleeding due to fluctuations in blood pressure and vasoconstriction
- Hypothermia and acidosis (with coagulopathy = “lethal triad”)

Prevention and treatment of coagulopathy
- Easier to prevent than to treat once it starts
- Keep patient warm and perfused
- Transfusion therapy: pay close attention to blood composition
  - Minimize use of non-blood fluid with massive hemorrhage
  - 1:1 administration of PRBCs and FFP

1:1 PRBC to FFP Ratio

University of Pennsylvania evaluation of 22 severely injured trauma patients.

11 of 22 developed AVP deficiency within 48 hours.
Patient Scenario

- 23 year old female involved in front end crash.
- Restrained passenger with prolonged extrication.
- Initial awake and alert.
- C-spine protected, placed on long board, and taken to outside hospital.

Case Study: Outside Facility

- HR: 110, SBP 100 mmHg, Temp 34.4
- Obvious deformity to right arm
- Underwent CT head, C-spine, chest, abd/pelvis.

- Is this patient in shock? When should transfer be initiated or considered?

Case Study: Radiographs
Case Study: Transport/Arrival

- HR 120, SBP 100 mmHg
- 3 liters of crystalloid administered with single episode of hypotension.
- Immediately taken to OR.

Case Study: Operative Intervention

- Initial End Tidal CO2 20, ABG 7.01 /8.29/16, 98
- Arrested following induction
- Grade V liver laceration underwent left partial hepatectomy and repair of retro-caval injury.
- Pancreatic transection and Grade III splenic laceration underwent distal pancreatectomy and splenectomy.
- Abdomen left open with Vac-Pac dressing.
- OR blood...10 liters of crystalloid, 36 units pRBCs, 30 units FFP, 12 units platelets, and 6 units cryoprecipitate

Conclusion

- Limit aggressive resuscitation until hemorrhage control obtained.
- Early concern for continued occult shock despite normal vital signs leads to improvement in outcome.
- New strategies including serial metabolic determinants and StO2 and EtCO2 may lead to early determination of uncompensated shock.
- Prevent and treat coagulopathy
- New therapies need to be based on early ability to demonstrate potential immune dysfunction