DON’T BE SHOCKED BY SHOCK!
GOALS

• Define Shock

• Basic Physiology

• Current Concepts

• Bringing it from the Hospital to the Bedside

• Avoiding Pitfalls!
DUOMO DI FIRENZE

1296 – 1436  DOME – FILIPPO BRUNELLESCHI
Consensus on circulatory shock and hemodynamic monitoring. Task force of the European Society of Intensive Care Medicine

Circulatory Shock

Jean-Louis Vincent, M.D., Ph.D., and Daniel De Backer, M.D., Ph.D.
DEFINING SHOCK

- Diagnosis Based on Clinical, Hemodynamic, and Biological signs
  - Systemic Hypotension:
    - Typically SBP < 90 or MAP < 70
  - Clinical Signs Tissue Hypo-perfusion
    - Skin: Cold, Clammy, Cyanotic
    - Renal: Low UO
    - Neurological: AMS
  - Abnormal Cellular Oxygen Metabolism
    - High Lactate
DEFINING SHOCK

• Best defined as:
  • “A life threatening, generalized, form of acute circulatory failure associated with inadequate oxygen utilization by the cells. It is a state in which the circulation is unable to deliver sufficient oxygen to meet the demands of the tissues ... cellular dysoxia”

• Clinically:
  • Impaired Microcirculation:
    • Mottled skin
    • Acrocyanosis
    • Slow capillary refill
    • Increased Central to toe temperature gradient
PATHOPHYSIOLOGY

- Four Mechanisms
  - Preload
  - Pump Failure
  - Obstructive
  - SVR Failure
PATHOPHYSIOLOGY

• Blood Pressure

• $BP = CO \times SVR$
  - $CO = SV \times HR$
  - $SV \sim \text{Pre-load} \times \text{contractility} \times \text{afterload}$
Figure 3. Hydraulic model of the circulation.
MICROVASCULAR

- **Slow diffusion**
  - Long diffusion distance
  - Low pressure gradient
  - PaO₂ 5.3 kPa
  - Red cell

- **Rapid diffusion**
  - Short diffusion distance
  - High pressure gradient
  - PaO₂ 13 kPa
  - Capillary

- **Diffusion distance**
  - PaO₂ 1.3 kPa
  - Mitochondrial PaO₂ < 0.7 kPa
  - Intracellular PaO₂ 2.7 kPa

- **Pressure gradient**
  - 53 - 1.3 = 40 kPa
  - 13 - 2.7 = 10.3 kPa

- **Mitochondrial**
  - PaO₂ 0.7–1.3 kPa
SEPTIC SHOCK
CARDIOGENIC SHOCK
Types of shock

- 62% Distributive (septic)
- 4% Distributive (nonseptic)
- 16% Cardiogenic
- 16% Hypovolemic
- 2% Obstructive
Severe Sepsis in Pre-Hospital Emergency Care
Analysis of Incidence, Care, and Outcome

Christopher W. Seymour\textsuperscript{1,2}, Thomas D. Rea\textsuperscript{3,4}, Jeremy M. Kahn\textsuperscript{2,5}, Allan J. Walkey\textsuperscript{6}, Donald M. Yealy\textsuperscript{7}, and Derek C. Angus\textsuperscript{2,8}

\textbf{Figure 2.} Temporal changes in the rates of hospitalizations with severe sepsis among emergency medical services (EMS) encounters, adjusted for age, sex, and receiving hospital. A comparison with acute myocardial infarction (AMI) and stroke is provided for context. \textit{Error bars} represent...
RECENT U.S. TRENDS IN HOSPITALIZATIONS AND MORTALITY WITH SEVERE SEPSIS

Kumar et al. Chest 2011
Finfer, Vincent NEJM 13
Four Phases in the Treatment of Shock.

<table>
<thead>
<tr>
<th>Phase Focus</th>
<th>Salvage</th>
<th>Optimization</th>
<th>Stabilization</th>
<th>De-escalation</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Obtain a minimal acceptable blood pressure</td>
<td>Provide adequate oxygen availability</td>
<td>Provide organ support</td>
<td>Wean from vasoactive agents</td>
</tr>
<tr>
<td></td>
<td>Perform lifesaving measures</td>
<td>Optimize cardiac output, $\text{SvO}_2$, lactate</td>
<td>Minimize complications</td>
<td>Achieve a negative fluid balance</td>
</tr>
</tbody>
</table>

STAY AND PLAY? VS LOAD AND GO?
• ABCs

• Is this Patient in Shock?
  • It may be hard to know!

• Is this Patient hypotensive?
  And/or
  • Clinical Signs of Shock?
BEDSIDE SURROGATES

- **Pre-Load**
  - Neck Veins
- **SVR**
  - Skin – warm or cold, mottled or not
  - Low Diastolic pressure
- **SV/Pump**
  - Pulses – nature bounding vs thready
  - Pulse pressure narrow – wide
  - Lungs – dry or crackles
SHOCK THERAPY

- First Rule
  - Always fill the tank!
    - If the tank appears not to be full no matter the type of shock, you need to fill the tank!
POLY-TRAUMA PATIENT

• Goal:
  • Reverse Shock?
  • Reduce the deleterious effects of Shock

• Judicious Crystalloid Fluid
  • Limited to maintain pulse
  • Avoidance of hypotension in TBI patients

• Early use of Blood Products

• Avoidance of:
  • Hypothermia
  • Acidosis
  • Coagulopathy
EARLY GOAL-DIRECTED THERAPY IN THE TREATMENT OF SEVERE SEPSIS AND SEPTIC SHOCK

EMANUEL RIVERS, M.D., M.P.H., BRYANT NGUYEN, M.D., SUZANNE HAVSTAD, M.A., JULIE RESSLER, B.S., ALEXANDRIA MUZZIN, B.S., BERNHARD KNOBLICH, M.D., EDWARD PETERSON, PH.D., AND MICHAEL TOMLANOVICH, M.D., FOR THE EARLY GOAL-DIRECTED THERAPY COLLABORATIVE GROUP*

Figure 2. Protocol for Early Goal-Directed Therapy.
Goal-Directed Resuscitation for Patients with Early Septic Shock

The ARISE Investigators and the ANZICS Clinical Trials Group®

Trial of Early, Goal-Directed Resuscitation for Septic Shock

A Randomized Trial of Protocol-Based Care for Early Septic Shock

The ProCESS Investigators®
SURVIVING SEPSIS CAMPAIGN

- Administer 30 ml/kg crystalloid for hypotension or lactate >4
- Goal MAP >65, start vasopressor if not responsive to fluid bolus
- Administer Broad Spectrum antibiotics
- Obtain BC, Measure lactate
- Re-assess
TSA adjusted required information size (20% RRR) is a Two-sided graph

TSA adjusted required information size (20% RRR) = 9028
Duration of hypotension before initiation of effective antimicrobial therapy is the critical determinant of survival in human septic shock*
Tempis Fugit
Carpe Diem
Time is Flying
Seize the Day
SEPTIC SHOCK

- Establish Diagnosis
- Source Control
- Initial Appropriate Antibiotics
- Supportive care
COMMENTARY

Prehospital treatment of sepsis: what really makes the "golden hour" golden?

Sarah A Sterling, Michael A Puskarich and Alan E Jones

Has the Time for Advanced Pre-Hospital Care of Severe Sepsis Finally Arrived?

AMERICAN JOURNAL OF RESPIRATORY AND CRITICAL CARE MEDICINE VOL 186 2012
Review Article

Prehospital Emergency Ultrasound: A Review of Current Clinical Applications, Challenges, and Future Implications

Mazen J. El Sayed and Elie Zaghrini

Department of Emergency Medicine, American University of Beirut Medical Center, P.O. Box 11-0236, Riad El Solh, Beirut 1107 2020, Lebanon

- 2013 survey North America 4% EMS use US
- 20% considering
- Barriers
  - Cost
  - Evidence
Questions?
This patient is in shock...

...But, what kind?

Cardiogenic? Obstructive? Distributive? Hypovolemic?
### DEFINING SHOCK

**Table 1** Main differences between the 2006 and 2014 consensus papers in terms of definition of shock, blood pressure statements and fluid responsiveness statements

<table>
<thead>
<tr>
<th>Topic</th>
<th>ICM Antonelli 2007</th>
<th>ICM Cecconi 2014</th>
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<tr>
<td><strong>Definition</strong></td>
<td>We recommend that shock be defined as a life-threatening, generalized maldistribution of blood flow resulting in failure to deliver and/or utilize adequate amounts of oxygen, leading to tissue dysoxia. Level 1; QoE moderate (B)</td>
<td>We define circulatory as a life-threatening, generalized form of acute circulatory failure associated with inadequate oxygen utilization by the cells. Ungraded</td>
</tr>
</tbody>
</table>
| **Blood pressure statements** | - We recommend a target blood pressure during initial shock resuscitation of:  
  - For uncontrolled hemorrhage due to trauma: MAP of 40 mmHg until bleeding is surgically controlled. Level 1; QoE moderate (B)  
  - For TBI without systemic hemorrhage: MAP of 90 mmHg. Level 1; QoE low (C)  
  - For all other shock states: MAP >65 mmHg. Level 1; QoE moderate (B) | - We recommend individualizing the target blood pressure during shock resuscitation. Level 1; QoE moderate (B)  
- We recommend to initially target a MAP of ≥65 mmHg. Level 1; QoE low (C)  
- We suggest tolerating a lower level of blood pressure in patients with uncontrolled bleeding (i.e. in patients with trauma) without severe head injury. Level 2; QoE low (C)  
- We suggest a higher MAP in septic patients with history of hypertension and in patients that show clinical improvement with higher blood pressure. Level 2; QoE moderate (B) |
| **Fluid responsiveness statements** | - We do not recommend the routine use of dynamic measures of fluid responsiveness (including but not limited to pulse pressure variation, aortic flow changes, systolic pressure variation, respiratory systolic variation test and collapse of vena cava). Level 1; QoE high (A)  
- There may be some advantage to these measurements in highly selected patients. Level 1; QoE moderate (B) | - We recommend using dynamic over static variables to predict fluid responsiveness, when applicable. Level 1; QoE moderate (B)  
- When the decision for fluid administration is made we recommend to perform a fluid challenge, unless in cases of obvious hypovolemia (such as overt bleeding in a ruptured aneurysm). Level 1; QoE low (C)  
- We recommend that even in the context of fluid-responsive patients, fluid management should be titrated carefully, especially in the presence of elevated intravascular filling pressures or extravascular lung water. Ungraded best practice |

ICM, Intensive Care Medicine; QoE, Quality of experience; MAP, mean arterial pressure; TBI, traumatic brain injury
**Table 2** Main differences between the 2006 and 2014 consensus papers in terms of hemodynamic monitoring

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<tr>
<th>Topic</th>
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<th>ICM Cecconi 2014</th>
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<tr>
<td>Hemodynamic monitoring</td>
<td>- We do not recommend routine measurement of CO for patients with shock. Level 1; QoE moderate (B)</td>
<td>- We recommend further hemodynamic assessment (such as assessing cardiac function) to determine the type of shock if the clinical examination does not lead to a clear diagnosis. <em>Ungraded best practice</em></td>
</tr>
<tr>
<td></td>
<td>- We suggest considering echocardiography or measurement of CO for diagnosis in patients with clinical evidence of ventricular failure and persistent shock with adequate fluid resuscitation. Level 2; QoE moderate (B)</td>
<td>- We suggest that, when further hemodynamic assessment is needed, echocardiography is the preferred modality to initially evaluate the type of shock as opposed to more invasive technologies. Level 2; QoE moderate (B)</td>
</tr>
<tr>
<td></td>
<td>- We do not recommend the routine use of the pulmonary artery catheter for patients in shock. Level 1; QoE high (A)</td>
<td>- In complex patients we suggest to additionally use pulmonary artery catheterization or transpulmonary thermodilution to determine the type of shock. Level 2; QoE low (C)</td>
</tr>
<tr>
<td></td>
<td>- We do not recommend routine measurement of cardiac output for patients with shock responding to the initial therapy. Level 1; QoE low (C)</td>
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<td></td>
<td>- We recommend measurements of cardiac output and stroke volume to evaluate the response to fluids or inotropes in patients that are not responding to initial therapy. Level 1; QoE low (C)</td>
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<td>- We suggest sequential evaluation of hemodynamic status during shock. Level 1; QoE low (C)</td>
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<td></td>
<td>- Echocardiography can be used for the sequential evaluation of cardiac function in shock. <em>Statement of fact</em></td>
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<td>- We do not recommend the routine use of the pulmonary artery catheter for patients in shock. Level 1; QoE high (A)</td>
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<td>- We suggest pulmonary artery catheterization in patients with refractory shock and right ventricular dysfunction. Level 2; QoE low (C)</td>
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<td>- We suggest the use of transpulmonary thermodilution or pulmonary artery catheterization in patients with severe shock especially in the case of associated acute respiratory distress syndrome. Level 2; QoE low (C)</td>
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<td>- We recommend that less invasive devices are used, instead of more invasive devices, only when they have been validated in the context of patients with shock. <em>Ungraded best practice</em></td>
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# Table 1  Relative effects of changes in PaO\(_2\), haemoglobin (Hb), and cardiac output (Qt) on oxygen delivery (Do\(_2\))

<table>
<thead>
<tr>
<th></th>
<th>(\text{FiO}_2)</th>
<th>(\text{PaO}_2) (kPa)</th>
<th>(\text{SaO}_2) (%)</th>
<th>(\text{Hb}) (g/l)</th>
<th>Dissolved (\text{O}_2) (ml/l)</th>
<th>(\text{CaO}_2) (ml/l)</th>
<th>Qt (l/min)</th>
<th>Do(_2) (ml/min)</th>
<th>Do(_2) (% change)††</th>
</tr>
</thead>
<tbody>
<tr>
<td>Normal*</td>
<td>0.21</td>
<td>13.0</td>
<td>96</td>
<td>130</td>
<td>3.0</td>
<td>170</td>
<td>5.3</td>
<td>900</td>
<td>0</td>
</tr>
<tr>
<td>Patient†</td>
<td>0.21</td>
<td>6.0</td>
<td>75</td>
<td>70</td>
<td>1.4</td>
<td>72</td>
<td>4.0</td>
<td>288</td>
<td>-68</td>
</tr>
<tr>
<td>↑(\text{FiO}_2)</td>
<td>0.35</td>
<td>9.0</td>
<td>92</td>
<td>70</td>
<td>2.1</td>
<td>88</td>
<td>4.0</td>
<td>352</td>
<td>+22</td>
</tr>
<tr>
<td>↑(\text{Hb})</td>
<td>0.60</td>
<td>16.5</td>
<td>98</td>
<td>105</td>
<td>3.8</td>
<td>96</td>
<td>4.0</td>
<td>384</td>
<td>+9</td>
</tr>
<tr>
<td>↑Qt</td>
<td>0.60</td>
<td>16.5</td>
<td>98</td>
<td>105</td>
<td>3.8</td>
<td>142</td>
<td>4.0</td>
<td>568</td>
<td>+48</td>
</tr>
</tbody>
</table>

\(\text{Do}_2 = \text{CaO}_2 \times \text{Qt}\) ml/min, \(\text{CaO}_2 = (\text{Hb} \times \text{SaO}_2 \times 1.34) + (\text{PaO}_2 \times 0.23)\) ml/l where \(\text{FiO}_2\) = fractional inspired oxygen concentration; \(\text{PaO}_2\), \(\text{SaO}_2\), \(\text{CaO}_2\) = partial pressure, saturation and content of oxygen in arterial blood; Qt = cardiac output. 1.34 ml is the volume of oxygen carried by 1 g of 100% saturated Hb. \(\text{PaO}_2\) (kPa) \(\times 0.23\) is the amount of oxygen in physical solution in 1 l of blood, which is less than <3% of total \(\text{CaO}_2\) for normal \(\text{PaO}_2\) (ie <14 kPa). *Normal 75 kg subject at rest. †Patient with hypoxaemia, anaemia, reduced cardiac output, and evidence of global tissue hypoxia. ††Change in \(\text{Do}_2\) expressed as a percentage of the preceding value.
Cardiogenic shock

Arterial hypotension and usually tachycardia

Signs of tissue hypoperfusion present
- Alteration in mental state
- Mottled, clammy skin
- Oliguria
- Elevated blood lactate

Circulatory shock

Low estimated cardiac output or SvO₂

High central venous pressure

Cardiogenic circulatory shock

Symptoms
Cardiogenic shock is characterized by low cardiac output and inadequate oxygen transport. It can be the result of a myocardial infarction, end-stage cardiomyopathy, advanced valvular disease, severe myocarditis, or severe cardiac arrhythmias.

Echocardiographic signs
Large ventricles and poor contractility.
**DIAGRAM**

- **Distributive shock**
  - Vasodilatation

**SIGNS OF CIRCULATORY SHOCK PATHWAY**

1. **Arterial hypotension and usually tachycardia**
   - Altered mental state
   - Mottled, clammy skin
   - Oliguria
   - Elevated blood lactate

2. **Signs of tissue hypoperfusion present**
   - Circulatory shock
     - Normal or high cardiac output or $\text{SvO}_2$

3. **Symptoms**
   - Distributive shock is characterized by hypovolemia and hypotension. It is the result of vasodilatation and release of inflammatory mediators.

4. **Echocardiographic signs**
   - Normal cardiac chambers and (usually) preserved contractility.
**Hypovolemic shock**

- Loss of plasma or blood volume

**SIGNS OF CIRCULATORY SHOCK PATHWAY**

- **Arterial hypotension and usually tachycardia**
  - Altered mental state
  - Mottled, clammy skin
  - Oliguria
  - Elevated blood lactate

- **Circulatory shock**
  - Low estimated cardiac output or SvO₂

- **Low central venous pressure**

- **Hypovolemic circulatory shock**

**SYMPTOMS**

Hypovolemic shock is characterized by internal or external fluid loss leading to organ failure.

**ECHOCARDIOGRAPHIC SIGNS**

Small cardiac chambers and normal or high contractility.
Obstructive shock is characterized by a blockage in blood flow caused by a massive pericardial effusion, cardiac tamponade, or tension pneumothorax.

ECHOCARDIOGRAPHIC SIGNS

Depends on the cause. Pulmonary embolism or pneumothorax: dilated right ventricle, small left ventricle. Tamponade: pericardial effusion, small left and right ventricles, dilated inferior vena cava.