Shock in the ICU: Implications for Physical Therapy

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Amy Pawlik, PT, DPT, CCS
CSM 2017: San Antonio, TX
February 16, 2017
Where we come in...
Learning Objectives

• Correctly describe various causes of systemic shock.

• Competently describe the role of oxygen delivery and vitals.

• Competently discuss various changes in lab values and vitals.

• Appropriately discuss possible positive and negative outcomes of therapy interventions for patients in shock.
A problem…

Functional Disability 5 Years after Acute Respiratory Distress Syndrome

Margaret S. Herridge, M.D., M.P.H., Catherine M. Tansey, M.Sc., Andrea Matte, B.Sc., George Tomlinson, Ph.D., Natalia Díaz Granados, M.Sc., Andrew Cooper, M.D., Cameron B. Guest, M.D., C. David Mazer, M.D., Sangeeta Mehta, M.D., Thomas E. Stewart, M.D., Paul Kudlow, B.Sc., Deborah Cook, M.D., Arthur S. Slutsky, M.D., and Angela M. Cheung, M.D., Ph.D., for the Canadian Critical Care Trials Group

Acute Skeletal Muscle Wasting in Critical Illness

Zudin A. Puthucheary, MRCP; Jaiiktry Rawal, MRCS; Mark McPhail, PhD; Bronwen Connolly, BSc; Gamunu Ratnayake, MRCP; Pearl Chan, MBBS; Nicholas S. Hopkinson, PhD; Rahul Padhke, PhD; Tracy Dew, MSc; Paul S. Sidhu, PhD; Cristiana Velloso, PhD; John Seymour, PhD; Chibesa C. Agley, MSc; Anna Selby, PhD; Marie Limb, PhD; Lindsay M. Edwards, PhD; Kenneth Smith, PhD; Anthea Rowlerson, PhD; Michael John Rennie, PhD; John Moxham, PhD; Stephen D. R. Herridge, PhD; Nicholas Hart, PhD; Hugh E. Montgomery, MD

Physical Complications in Acute Lung Injury Survivors: A Two-Year Longitudinal Prospective Study

Eddy Fan, MD, PhD; David W. Dowdy, MD, PhD; Elizabeth Colantuoni, PhD; Pedro A. Mendez-Tellez, MD; Jonathan E. Sevransky, MD, MHSc; Carl Shanholm, MD; Cheryl R. Dennison Himmelfarb, RN, PhD; Sanjay V. Desai, MD; Nancy Ciesla, DPT; Margaret S. Herridge, MD, MPH; Peter J. Pronovost, MD, PhD; Dale M. Needham, MD, PhD

Critical Care Medicine • April 2014 • Volume 42 • Number 4

Shock in the ICU
Solutions!

Feasibility of physical and occupational therapy beginning from initiation of mechanical ventilation*

Mark C. Pohlm (MD); William D. Schweickert, MD; Anne S. Pohlm, RN, MSN; Celerina Nigos, RN; Amy J. Pawlik, PT; Cheryl L. Esbrook, OTRL; Linda Spears, PT; Megan Miller, OTRL; Mietka Franczyk, PT; Deanna Deprizio, OTRL; Gregory A. Schmidt, MD; Amy Bowman, RN, BSN; Rhonda Barr, PT; Kathryn McCallister, BS; Jesse B. Hall, MD; John P. Kress, MD

Crit Care Med 2010 Vol. 38, No. 11

Receiving Early Mobility During an Intensive Care Unit Admission Is a Predictor of Improved Outcomes in Acute Respiratory Failure

Peter E. Morris, MD, Leah Griffin, MS, Michael Berry, PhD, Clif Thompson, RN, R. Duncan Hite, MD, Chris Winkelmann, PhD, Ramona O. Hopkins, PhD, Amelia Ross, MSN, Luz Dixon, RN, Susan Leach, RN and Edward Haponik, MD

The American Journal of the Medical Sciences • 2011

The effect of increased mobility on morbidity in the neurointensive care unit

Clinical article


Early mobilization out of bed after ischaemic stroke reduces severe complications but not cerebral blood flow: a randomized controlled pilot trial

Karin Diserens, Tiago Moreira, Lorenz Hirt, Mohamed Faouzi, Jelena Grujic, Gilles Bieler, Philippe Vuadens and Patrik Michel

Clin Rehabil 2012 26: 451 originally published online 2 December 2011

Effectiveness of an Early Mobilization Protocol in a Trauma and Burns Intensive Care Unit: A Retrospective Cohort Study

Diane E. Clark, John D. Lowman, Russell L. Griffin, Helen M. Matthews and Donald A. Reiff

PHYS THER. 2013; 93:186-196.
Why discuss shock in terms of critical illness?

- There is a growing body of evidence supporting safety and effectiveness of interventions
- But there are still many physiological considerations prior to us intervening
- It all comes down to oxygen…
Shock

- A life threatening condition of circulatory failure

- Initially reversible, but rapidly becomes irreversible, resulting in multi-organ failure (MOF) and death

- Definition: A state of *cellular* and *tissue hypoxia* due to *reduced oxygen delivery* and/or *increased oxygen consumption* or inadequate utilization
**CaO₂** – amount of O₂ bound to Hgb + amount of O₂ dissolved in arterial blood

\[ \text{CaO₂} = (1.34 \times \text{Hgb} \times \text{SaO₂}) + (0.0031 \times \text{PaO₂}) \]

**Delivery**

- The rate at which O₂ is transported from the lungs to the tissues
  - \( \text{DO₂} = \text{Cardiac Output (Q)} \times \text{CaO₂} \)

**Consumption**

- The rate at which O₂ is removed from the blood for use by the tissues
  - \( \text{VO₂} = \text{Q} \times (\text{CaO₂} – \text{CvO₂}) \)

**Extraction**

- \( \text{SvO₂} – \text{mixed venous oxyhemoglobin saturation} \)
- **Normal SvO₂ – 75%**
- OE ratio = \( \frac{\text{VO₂}}{\text{DO₂}} = 25\% \)
## Common Causes of Increased Consumption

<table>
<thead>
<tr>
<th>Condition</th>
<th>% Increase</th>
</tr>
</thead>
<tbody>
<tr>
<td>Fever (each degree C)</td>
<td>10</td>
</tr>
<tr>
<td>Fracture</td>
<td>10</td>
</tr>
<tr>
<td>Agitation</td>
<td>16</td>
</tr>
<tr>
<td>Increased WOB</td>
<td>40</td>
</tr>
<tr>
<td>Severe Infection</td>
<td>60</td>
</tr>
<tr>
<td>Burns</td>
<td>100</td>
</tr>
<tr>
<td>Sepsis</td>
<td>50-100</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Procedure</th>
<th>% Increase from rest</th>
</tr>
</thead>
<tbody>
<tr>
<td>Dressing Change</td>
<td>10</td>
</tr>
<tr>
<td>Visitors</td>
<td>22</td>
</tr>
<tr>
<td>Bath</td>
<td>23</td>
</tr>
<tr>
<td>Endotracheal Suctioning</td>
<td>27</td>
</tr>
<tr>
<td>Chest PT</td>
<td>35</td>
</tr>
<tr>
<td>Getting out of bed</td>
<td>39</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Medication</th>
<th>% Increase</th>
</tr>
</thead>
<tbody>
<tr>
<td>Norepinephrine</td>
<td>10-21</td>
</tr>
<tr>
<td>Dopamine</td>
<td>6</td>
</tr>
<tr>
<td>Dobutamine</td>
<td>19</td>
</tr>
<tr>
<td>Epinephrine</td>
<td>23-29</td>
</tr>
</tbody>
</table>
### Causes of Failure of Oxygen Delivery

<table>
<thead>
<tr>
<th>Decline in CaO2</th>
<th>Decline in Cardiac Output (Q)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Anemia</td>
<td>Hypovolemia</td>
</tr>
<tr>
<td>Lung Disease</td>
<td>Cardiac Disease</td>
</tr>
</tbody>
</table>
Hypoperfusion

Cell Hypoxia

Energy Deficit

Anaerobic Metabolism

Lactic Acid Accumulation

Metabolic Acidosis

Cell Membrane Ion Dysfunction

Intracellular Edema

Extracellular Leakage

Hypotension

Hypoxia

Activation of WBCs

Inflammatory Response

Microvascular Blood Clots

Inflammatory Response

Release of Nitric Oxide

Cytokines

Vasodilation

Shock in the ICU
Hypoperfusion causes Inflammation

Inflammation causes Hypoperfusion

Shock in the ICU 13
Is it safe?
Tissue Hypoperfusion

Cardiac Output (Q)

Systemic Vascular Resistance (SVR)
Perfusion

Cardiac Output

The volume of blood ejected from the L side of the heart in one minute

SV

HR

HR x SV

Systemic Vascular Resistance

The resistance to blood flow by all of the systemic vasculature

MAP

CVP

Q

(MAP - CVP)/Q

Blood Pressure

Q

SVR

Q x SVR

Shock in the ICU
## Cardiac Index

An indicator of pump performance in individuals of various sizes

\[
CI = \frac{CO}{BSA}
\]

<table>
<thead>
<tr>
<th>Mr. Johnson</th>
<th>Mr. Smith</th>
</tr>
</thead>
<tbody>
<tr>
<td>52 yo male</td>
<td>52 yo male</td>
</tr>
<tr>
<td>5’10”</td>
<td>5’10”</td>
</tr>
<tr>
<td>60 kg</td>
<td>110 kg</td>
</tr>
<tr>
<td>CO = 5.0</td>
<td>CO = 5.0</td>
</tr>
<tr>
<td>BSA = 1.75</td>
<td>BSA</td>
</tr>
<tr>
<td>CI = 2.86</td>
<td>CI = 2.21</td>
</tr>
</tbody>
</table>

Mr. Johnson's Cardiac Index (CI) is 2.86, which is within the normal range for an individual of his size.

Mr. Smith's Cardiac Index (CI) is 2.21, which is also within the normal range for an individual of his size.
Stroke Volume

Preload

Afterload

Stroke Volume

Contractility
Preload

- SV increases proportionally to the volume of blood filling the heart (EDV)
- The increased volume stretches the ventricle to allow for a more forceful contraction
Afterload

- There is an inverse relationship with afterload and SV
- Elevated afterload decreases SV
Contractility

Effects of Preload
Greater volume > Greater stretch > Greater contract

Effects of Afterload
Greater resistance > Greater inotropy (compensation)

Contractility

Greater volume > Greater stretch > Greater contract
Contractility

An indicator of pump performance in individuals of various sizes

\[ \text{EF} = \frac{\text{SV}}{\text{EDV}} \quad (60-70\%) \]

**HFrEF**

- \( \text{SV} = 50 \text{ mL} \)
- \( \text{EDV} = 120 \text{ mL} \)
- \( \text{ESV} = 70 \text{ mL} \)
- \( \text{EF} = \frac{50}{120} = 0.42 = 42\% \)

**HFpEF**

- \( \text{SV} = 50 \text{ mL} \)
- \( \text{EDV} = 80 \text{ mL} \)
- \( \text{ESV} = 30 \text{ mL} \)
- \( \text{EF} = \frac{50}{80} = 0.63 = 63\% \)
Mean Arterial Pressure (MAP)

Normal 70-110 mmHg

Perfusion pressure to end organs

MAP = (CO x SVR) + CVP

MAP = DBP + 1/3 (SBP - DBP)
Factors Effecting MAP

- Cardiac Output
  - Heart Rate
    - Sympathetic
    - Parasympathetic
  - Stroke Volume
- Preload
- Blood Volume
- Total Peripheral Resistance
  - Vessel Length/Radius
  - Blood Viscosity

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## Normal Vitals

<table>
<thead>
<tr>
<th></th>
<th>HR</th>
<th>BP</th>
<th>RR</th>
<th>SpO₂</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Normal Resting:</strong></td>
<td>60-100 bpm</td>
<td>SBP &lt;120 mmHg DBP &lt;80 mmHg</td>
<td>12-20 breaths/min</td>
<td>&gt;90%</td>
</tr>
<tr>
<td><strong>Response to activity:</strong></td>
<td>Should rise with increased workload</td>
<td>Should rise with increased workload</td>
<td>Should correspond to HR changes</td>
<td>No change or minimal decrease</td>
</tr>
<tr>
<td></td>
<td>• Submax effort is nearly linear</td>
<td>• Rise is &gt; in UE vs LE activity</td>
<td>• Remains unchanged or decreases with activity</td>
<td>• Increases with heavy resistance activity</td>
</tr>
<tr>
<td></td>
<td>• 8-12 bpm rise with each MET of activity</td>
<td>• Rise is &gt; in UE vs LE activity</td>
<td>• &gt; during static vs dynamic activity</td>
<td>• 8-12 mmHg rise with each MET of activity</td>
</tr>
</tbody>
</table>
# Common Activities (MET)

<table>
<thead>
<tr>
<th>Activity</th>
<th>MET</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sitting Quietly</td>
<td>1.0</td>
</tr>
<tr>
<td>Walking (2mph)</td>
<td>2.4</td>
</tr>
<tr>
<td>Taking out the trash</td>
<td>3.0</td>
</tr>
<tr>
<td>Cycling (leisurely)</td>
<td>3.3</td>
</tr>
<tr>
<td>Raking lawn</td>
<td>4.0</td>
</tr>
<tr>
<td>Walking (4mph)</td>
<td>4.5</td>
</tr>
<tr>
<td>Cycling (moderately)</td>
<td>5.7</td>
</tr>
<tr>
<td>Stair climbing</td>
<td>8.6</td>
</tr>
<tr>
<td>Jogging (6mph)</td>
<td>10.2</td>
</tr>
</tbody>
</table>
# Abnormal Response to Exercise

<table>
<thead>
<tr>
<th>Deconditioned Individuals</th>
<th>Rapid Rate of HR</th>
</tr>
</thead>
<tbody>
<tr>
<td>Untrained Individuals</td>
<td>Slow Rate of HR rise</td>
</tr>
<tr>
<td>Sign of CAD</td>
<td>Blunted HR response, unable to achieve near predicted HR max on stress test</td>
</tr>
</tbody>
</table>
| Arrhythmias               | Decrease HR with increased work  
With arrhythmias at rest, a normal response is a lack of change in frequency of type of arrhythmia |
## Abnormal Response to Exercise

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Description</th>
</tr>
</thead>
</table>
| SBP       | • Rapid Rate of Rise  
           • Blunted Rise  
           • Exertional hypotension (>10 mmHg decrease with activity) |
| DBP       | • Progressive rise (>10 mmHg decrease with activity)  
           • Decrease (>10 mmHg) (okay with athletes) |
| RR        | • Will severely change with a drop in SpO2  
           • Resting RR > 24 may indicate clinical instability |
| SpO2      | • Decreases with increased activity  
           • Severely abnormal <88% |
Monitoring

Non-Invasive
- Blood Pressure
- ECG
- SpO2

Invasive
- Arterial Catheter
- Central Venous Catheter (TLC)
- Pulmonary Artery Catheter (Swan)
- Intracranial Pressure Monitor
- Intra-Abdominal Pressure
The Big Three

1.

2.

3.
The Bottom Line…

MAP

Cardiac Index

Cardiac Output

SvO2

BP

EF
What to expect

• An introduction to various types of shock
• Discuss specific considerations of each type and how to determine if therapy is safe as well as how to intervene early.
• Minimal answers → initiate discussion on how we can begin to approach some of these topics
Types of Shock

- Distributive
- Cardiogenic
- Hypovolemic
- Obstructive
- Combined

What will I see?

Shock

Shock in the ICU 33
Distributive Shock

Due to severe peripheral vasodilation (afterload)

<table>
<thead>
<tr>
<th>SIRS</th>
<th>Sepsis</th>
<th>Drug/Toxicity</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pancreatitits</td>
<td>Gram (+) (Pneumococcus)</td>
<td>Long Acting Narcotics</td>
</tr>
<tr>
<td>Burns</td>
<td>MRDOs (MRSA)</td>
<td>Snake/Insect bites</td>
</tr>
<tr>
<td>Hypoperfusion due to trauma/crush injury</td>
<td>Gram (-) (Pseudomonas, Klebsiella, Enterococcus)</td>
<td>Transfusion Reactions</td>
</tr>
<tr>
<td>Embolism</td>
<td>Fungi (Candida)</td>
<td>Metal Poisoning</td>
</tr>
<tr>
<td>ROSC after cardiac arrest</td>
<td></td>
<td>Toxic Shock Syndrome</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Neurogenic</th>
<th>Endocrine</th>
<th>Anaphylactic</th>
</tr>
</thead>
<tbody>
<tr>
<td>Severe TBI</td>
<td>Addisonian Crisis</td>
<td>Allergic Reactions</td>
</tr>
<tr>
<td>Severe SCI</td>
<td>Thyrotoxicosis</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Myxedema</td>
<td></td>
</tr>
</tbody>
</table>

SIRS: Systemic Inflammatory Response Syndrome
Sepsis: Severe Infection
Drug/Toxicity: Drug-Related or Toxic Effects
Endocrine: Hormonal Imbalance
Anaphylactic: Allergic Reaction
Sepsis Statistics

- Annual rate in the US between 1979-2000: >1.66 million cases
- Increased rates between 1998-2000: from 13 to 78 cases/ 100,000
- Global incidence from 1995-2015: 437 per 100,000 (not reflective of low and middle income countries)
- Reasons for rise of incidence:
  - Older population
  - Immunosuppression
  - MDR infections
  - Earlier detection
- Highest in African-American males
- Highest in older adults (>65) (60-85%)
- Greatest during winter months
Morbidity and Mortality

• Rate of Mortality
  – Ranges from 10-52%
  – Responsible for 6% of all deaths
  – Increase proportionally to severity
    • SIRS: 7%
    • Sepsis: 16%/>10%
    • Septic Shock: 46%/>40%

• Decreasing Rates
  – 50% reduction (from 35% to 18%) from 2000-2012

• Long Term
  – Despite survival from initial hospitalization, risk of death persists (up to 20%)
    • Most occur within the first 6 months, remains elevated at 2 years
    • Those that survived were younger, had better measures of sepsis scores, lower lactate, less renal support, less use of steroids
Terms

- Systemic Inflammatory Response Syndrome (SIRS)
- Early Sepsis
- Sepsis
- Severe Sepsis
- Septic Shock
- Multi Organ Dysfunction Syndrome (MODS)
- Death

Shock in the ICU
Definitions

Shock in the ICU

A life-threatening organ dysfunction caused by a dysregulated host response to infection. Organ dysfunction is considered a SOFA score of an increase in two or more.

Sepsis

Hypotension requiring vasoactives to maintain MAP >65+ and having lactate >2 despite adequate fluid resuscitation.

Septic Shock

Homeostasis cannot be maintained without intervention. Greater number of organs, greater risk of mortality.

MODS
**Measures of Sepsis**

**Acute Physiologic and Chronic Health Evaluation (APACHE)**

- Sum of the variables predicts mortality
- Versions II-IV
- Administered within 24 hours of admission to ICU
- 0-71
  - Higher scores associated with increased severity and higher risk of death

**Sequential Organ Failure Assessment (SOFA)**

- Sequential scores predict trajectory
- Calculated 24 hours after admission to the ICU and every 48 after
- Higher score are most predictive of mortality
  - Sepsis: An increase in score >2 predictive of mortality >10%
  - Septic Shock: Score >2 who also require vasopressors and elevated lactate >2 mmol/L predictive mortality >40%

- Organ
  - Respiratory
  - Cardiovascular
  - Hepatic
  - Coagulation
  - Neurological
  - Renal

### Risk Factors

<table>
<thead>
<tr>
<th>ICU Admission</th>
<th>Bacteremia</th>
<th>&gt;65 years</th>
</tr>
</thead>
<tbody>
<tr>
<td>Immunosuppression</td>
<td>Diabetes</td>
<td>Cancer</td>
</tr>
<tr>
<td>Community Acquired Pneumonia</td>
<td>Previous hospitalization</td>
<td>Genetic Factors</td>
</tr>
</tbody>
</table>
Lactate

- Organ hypoperfusion
  - \( >2 \text{ mmol/L} \) independently of hypotension
  - \( >4 \text{ mmol/L} \) is associated with poor prognosis (78%)
    - Consistent with septic shock

- Elevated due to:
  - Impaired clearance
  - Overproduction

- Indicates organ dysfunction
  - Impaired clearance (hepatic, renal)
  - Overproduction (anaerobic)
Lactate & Mortality

- 830 patients diagnosed with severe sepsis or septic shock
  - Stratified into low (<2 mmol/L), intermediate (2-3.9 mmol/L) and high (>4 mmol/L)
  - Further divided into shock or nonshock groups
    - Hypotension <90 mmHg or use of vasoactives

- Results
  - *Elevated lactate was associated with higher APACHE II scores*

  - Initial lactate levels were *higher in non survivors* compared to survivors at 28 days (3.4 vs 2.6) in nonshock and (5.2 vs 3.3) in shock
  - All patients with initial lactate <1 mmol/L survived

- 28 days
  - *Intermediate and High lactate levels were associated increased mortality* in both groups as compared to low lactate levels

- 60 days
  - High lactate levels was associated with increased mortality in both groups as compared to intermediate and low
Lactate Clearance

- A prospective observational case series to evaluate the value of lactate clearance in 6 hours
  - 111 patients
  - 52.3% patients presented to the ED in septic shock
  - In hospital mortality was 42.3%

- Vasopressors
  - Utilized less in those with high clearance in 6 hours

- APACHE II scores
  - High clearance had lower scores throughout the 72 hour study period

- Mortality
  - High clearance, 52% relatively lower in hospital mortality rate
  - Similar at 60 days follow up
  - Severe sepsis – those with higher clearance had less mortality
  - Septic shock – less patients with high clearance, but of those, there was lower mortality
QOL and Sepsis

• A systematic review reporting quality of life data (>3 months) in patients with sepsis
  – Herridge et al, reported persistent functional disability in patients who survived ALI at 1 and 5 years.
  – **Sepsis is the most common cause of ALI**, but no studies have controlled for it.

• Twelve studies using validated measures such as the SF-36 and EuroQOL-5D were analyzed.
  – All studies found **decreased QOL was lower than general population** at 3-120 months (most assessed 6 months) after the onset of septic shock.
Functional & Cognitive Impact of Sepsis

- Health and Retirement Study (HRS) is an ongoing national cohort of community dwelling Americans over 50 years.

- Analyzed patients who were hospitalized with severe sepsis from 1998-2004

- Functional Status
  - 6 ADLs (walking, dressing, bathing, eating, getting in and out of bed, toileting)
  - 5 IADLs (preparing a hot meal, grocery shopping, making phone calls, taking medication, and managing finances)
  - **No functional limitations** before sepsis resulted in the development of **1.57 new** ADL/IADL limitations
  - **Mild/moderate limitations** before sepsis resulted in the development of **1.50 new** ADL/IADL limitations

- Cognition
  - Biennial interviews with validated cognitive tests
  - **6.1%** of survivors had moderate/severe cognitive impairments when surveyed before severe sepsis
  - Increased to **16.7%** after
Sepsis Induced Myopathy

- Sepsis has been found to increase the risk of developing persistent acquired weakness affecting both respiratory and peripheral muscles.
  - Decrease in force generating capacity
  - Atrophy

- Mechanisms
  - Systemic inflammation results in increased circulating cytokines
  - Free radicals generated from cytokines can cause mitochondrial derangements
  - Proteolysis occurs with inflammation
  - Impairs protein synthesis
Sepsis Induced Myopathy

13,14,15

• Physical Activity, Muscle Strength, and Exercise Capacity
  – No difference in 6MWD or walking time between patients with severe sepsis and septic shock
  – At 3 months after hospital discharge 6MWD, MIP, quad and hand strength all were lower than % predicted of normal populations

• Physical Therapy
  – A prospective double-blinded RCT 50 patients with sepsis syndromes
  – Lactate measures were taken pre and post exercise (no increase post exercise)
  – Improved HR QOL (SF-36) at 6 months post hospital discharge
  – No change in physical function (ACIF) or exercise capacity (PFIT)
Early Goal Directed Therapy

- Established in 2001
- Adopted by the Surviving Sepsis Campaign
- 6 hour resuscitation algorithm
- Administration of fluids
- Goals:
  - MAP ≥ 65
  - Urine output ≥ 0.5 mL/kg/hr
  - CVP 8-12
  - ScVO2 ≥70% or SvO2 ≥65
  - Lactate clearance >10%

- ProCESS, ProMISe, and ARISE
  - EGDT had no improvement in outcomes
  - Although early detection and management is likely the reason
Medical Management

**Stabilize Airway**
- Supplemental O2
- Monitor oxygenation
- Intubation and MV may be required
- Sedation considerations
- Chest Xray (ARDS)
- ABGs

**Assess Perfusion**
- Hypotension (SBP <90 mmHg, MAP <70 mmHg, drop in SBP >40 mmHg)
- Hypoperfusion can occur in the absence of hypotension (early)
- Elevated lactate (>2 mmol/L) (>4 indicates worse prognosis)

**Establish Venous Access**
- Central venous access
- Infusion of IV fluids
- Medications
- Blood products
- CVP monitoring
- ScVO2
- PAC no longer considered a predictor of fluid responsiveness

**Restore Perfusion**
- Source Control
- Removal
- Antibiotics/Anti fungals
- IV Fluids
- Crystalloids (normal saline, Ringer’s lactate) vs Colloids (albumin)
- Vasopressors
- Inotropes
- Transfusion
- Glucocorticoids
- Cooling, antipyretics
Considerations for Therapy

• Stage of sepsis

• Hemodynamic stability
  – $SvO_2$
  – Lactate
  – MAP
  – Medications (vasoactives, inotropes, corticosteroids)

• Myopathy development

• Discussion

• Cases
# Cardiogenic Shock

Due to intracardiac causes of pump failure (contractility)

<table>
<thead>
<tr>
<th>Cardiomyopathic</th>
<th>Mechanical</th>
<th>Arrhythmic</th>
</tr>
</thead>
<tbody>
<tr>
<td>MI (&gt;40% of LV)</td>
<td>Severe aortic or mitral valve insufficiency</td>
<td>Atrial</td>
</tr>
<tr>
<td>MI of any size if ischemia is severe (multivessel CAD)</td>
<td>Acute valvular defects</td>
<td>Ventricular</td>
</tr>
<tr>
<td>Severe RV infarction</td>
<td>Severe VSD</td>
<td>Bradyarrhythmia</td>
</tr>
<tr>
<td>ADHF</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Advanced septic or neurogenic shock</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Myocarditis</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
Cardiogenic Shock Statistics

- Acute MI is the most common cause (RV or LV failure)
- Cardiogenic shock is the leading cause of death in patients with acute MI
  - Hospital mortality rates approx 50%
- Reduced mortality now seen due to early revascularization

- SHOCK Trial
  - Inflammation
    - 18% (54) had fever and/or leukocytosis
    - All had lower SVR
    - 40 of those developed positive cultures
Predictors of Cardiogenic Shock

- Advanced Aged
- HR on admission (75 bpm)
- Diabetes
- Hx of MI
- Hx of CABG
- Signs of heart failure on admission
- Anterior location of infarction
Criteria for Diagnosis

1. • SBP < 80 – 90 mmHg for 30 min OR
   • MAP < 65 mmHg for 30 min OR
   • Use of vasoactives to achieve SBP > 90 OR
   • MAP > 30 mmHg lower than baseline
   • Cardiac Index < 1.8 L/min/m² without support OR
   • Cardiac Index < 2 to 2.2 L/min/m² with support

2. • Pulmonary Edema OR
   • Elevated LV filling pressures

3. • Signs of impaired organ perfusion with at least one of the following:
   • AMS
   • Cold, clammy skin
   • Oliguria
   • Increased serum lactate
Medical Management

Determine cause
- ECG
- Troponins
- BNP

Revascularization
- Angiography with revascularization regardless of time interval since onset of symptoms
- Transfer to expert center
- Aspirin and heparin

Monitoring
- Arterial monitoring of DBP
- MAP > 65
- Lactate
- Kidney, liver function
- CVC – for ScVO2
- Cardiac Output and SvO2 if refractory
- Routine ECHO

Manage Hemodynamics
- MAP ≥ 65 with use of inotropes or vasoactives
- Norepinephrine (over Epinephrine)
- Dobutamine (over Dopamine)
- PDIs only if refractory

Circulatory Support
- IABP has no benefit to mortality
- VA ECMO is preferred
Early Revascularization

- SHOCK Trial (2003)
- RCT of 302 patients
  - LV failure following MI
  - Control: Medical stabilization
  - Intervention: **Emergency revascularization**
    - CABG or Angioplasty
    - Within 6 hours
  - 30 day mortality
    - No difference
  - **6 mo and 12 mo** follow up
    - Significant **survival** with revascularization (**50% vs 37%**)
    - No difference >75 yo
  - **3 and 6 year survival was higher** (almost 2/3rds)
- SHOCK Trial survivors
  - Functional status
    - **NYHA Class I or II: 87% of 1-year survivors**
QOL

- SHOCK Trial follow up
  - 2 weeks post discharge and 6 and 12 months after
  - QOL (Andrews Ladder of Life)
  - Functional Status (NYHA) (Multidimensional Index of Life Quality – MILQ)

- Results
  - NYHA
    - 2 weeks after discharge groups were similar
    - *Proportion in Class I or II* remained constant but with *higher* number of patients having received *intervention*
    - Proportion in Class III and IV decreased over time due to deaths with more in the control group
  - MILQ
    - Similar scores among 1 year survivors
  - Andrews
    - Similar scores among 1 year survivors
81 patients who underwent ECMO support for refractory cardiogenic shock

- 34 survived hospital discharge
- Outcomes
  - 28 day and 90 day survival
    - 48% and 38%
    - Death due to refractory multiorgan failure
  - 6 survived bridge to device, 8 bridge to transplant
- **HRQOL** (SF-36)
  - 28 patients completed it
  - Compared to age and gender matched controls:
    - *Physical function, Role-physical, general health, and social functioning were lower*
    - Mental health, vitality were satisfactory
Considerations for Therapy

• Was the cause corrected (valvular problem vs. MI vs. ADHF)

• Status of reperfusion
  – Heparin vs PCI vs CABG vs ECMO

• Hemodynamic stability
  – SvO2
  – MAP
  – Medications (inotropes, diuretics)
  – Mechanical support
  – Other organ dysfunction

• How may exercise impact this?

• Discussion

• Cases
## Hypovolemic Shock

Due to reduced intravascular volume (preload)

<table>
<thead>
<tr>
<th>Hemorrhagic</th>
<th>Nonhemorrhagic</th>
</tr>
</thead>
<tbody>
<tr>
<td>Trauma</td>
<td>GI loss (diarrhea, vomiting)</td>
</tr>
<tr>
<td>Upper GI Bleed</td>
<td>Skin loss (burns, SJS, heat stroke)</td>
</tr>
<tr>
<td>Intra/Perioperative Bleeding</td>
<td>Renal Loss (Diuresis, nephropathies, hypoaldosteronism)</td>
</tr>
<tr>
<td>AAA Rupture</td>
<td>Third spacing (trauma, intestinal obstruction, pancreatitis, cirrhosis)</td>
</tr>
<tr>
<td>Arteriovenous malformation (AVM)</td>
<td></td>
</tr>
<tr>
<td>Other</td>
<td></td>
</tr>
</tbody>
</table>
Considerations for Therapy

• Was the cause corrected?

• Hemodynamic stability
  – MAP
  – Hgb/Hct

• How may exercise impact this?

• Discussion

• Cases
## Obstructive Shock

**Due to extracardiac causes of pump failure**

<table>
<thead>
<tr>
<th>Pulmonary Vascular (causes RHF)</th>
<th>Mechanical (presents like hypovolemic shock)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pulmonary Embolism (PE)</td>
<td>Tension Pneumothorax</td>
</tr>
<tr>
<td>Pulmonary Hypertension (PAH)</td>
<td>Pericardial Tamponade</td>
</tr>
<tr>
<td>Severe Stenosis</td>
<td>Constrictive Pericarditis</td>
</tr>
<tr>
<td>Acute Obstruction of Pulmonary or Tricuspid Valve</td>
<td>Restrictive Cardiomyopathy</td>
</tr>
<tr>
<td></td>
<td>Abdominal Compartment Syndrome</td>
</tr>
</tbody>
</table>
Considerations for Therapy

• Very much dependent on the cause
  – PE
    • Severity
    • Stability
    • Medical Treatment
  – Pericardial Tamponade
    • Must be treated

• Discussion

• Cases
Combined Cardio-myopathy + Over Diuresis = Combined

Combined Trauma + Myocardial Depression = Combined
Effects on

<table>
<thead>
<tr>
<th>Clinical Measurement</th>
<th>Preload</th>
<th>Contractility</th>
<th>Afterload</th>
<th>SvO2</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>CVP</td>
<td>CO</td>
<td>TPR</td>
<td>&lt;65%</td>
</tr>
<tr>
<td>Hypovolemic</td>
<td>↓*</td>
<td>↓</td>
<td>↑</td>
<td>&lt;65%</td>
</tr>
<tr>
<td>Cardiogenic</td>
<td>↑</td>
<td>↓*</td>
<td>↑</td>
<td>&lt;65%</td>
</tr>
<tr>
<td>Distributive</td>
<td>↓</td>
<td>↓</td>
<td>↓*</td>
<td>&gt;65%</td>
</tr>
<tr>
<td>Obstructive (pulm)</td>
<td>↓</td>
<td>↓</td>
<td>↑</td>
<td>&gt;65%</td>
</tr>
<tr>
<td>Obstructive (card)</td>
<td>↑</td>
<td>↓</td>
<td>↑</td>
<td>&lt;65%</td>
</tr>
</tbody>
</table>
### Normal Measures

<p>| | |</p>
<table>
<thead>
<tr>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>SvO2</strong></td>
<td>60 – 75%</td>
</tr>
<tr>
<td><strong>SV</strong></td>
<td>50 – 100 mL/beat</td>
</tr>
<tr>
<td><strong>CO</strong></td>
<td>4 – 8 L/min</td>
</tr>
<tr>
<td><strong>Cl</strong></td>
<td>2.5 – 4.0 L/min/M2</td>
</tr>
<tr>
<td><strong>CVP</strong></td>
<td>2 – 6 mmHg</td>
</tr>
<tr>
<td><strong>PAP</strong></td>
<td>25/10 mmHg</td>
</tr>
</tbody>
</table>
### Normal Measures with Exercise

<table>
<thead>
<tr>
<th>Measure</th>
<th>Range</th>
<th>Changes</th>
</tr>
</thead>
<tbody>
<tr>
<td>SvO2</td>
<td>60 – 75%</td>
<td>Rapid fall within 2 minutes of exercise</td>
</tr>
<tr>
<td>SV</td>
<td>50 – 100 mL/beat</td>
<td>In conditioned individuals, up to 200 mL/beat</td>
</tr>
<tr>
<td>CO</td>
<td>4 – 8 L/min</td>
<td>Up to 21 L/min in active individuals, &gt; in elite</td>
</tr>
<tr>
<td>CI</td>
<td>2.5 – 4.0 L/min/M2</td>
<td>Based on CO</td>
</tr>
<tr>
<td>CVP</td>
<td>2 – 6 mmHg</td>
<td>At least 4 mmHg</td>
</tr>
<tr>
<td>PAP</td>
<td>25/10 mmHg</td>
<td>Rarely exceeds &gt;20 mmHg</td>
</tr>
</tbody>
</table>
Stages of Shock

1. **Event**
   - Injury or Infection

2. **Pre-shock**
   - Compensated (cryptic)

3. **Shock**
   - Compensatory mechanism no longer effective
   - Signs/Symptoms begin to present

4. **End-Organ Dysfunction**
   - Irreversible
   - Multiorgan failure (MOF)
   - Death
Signs and Symptoms

Distributive
- Hypotension
- Tachycardia
- Fever/hypothermia
- Leukocytosis
- Cool skin
- Cyanosis/mottling/decreased capillary refill
- Organ dysfunction (AMS, oliguria)
- Specific to infection (Cough, abdominal pain, purulence)

Cardiogenic
- Hypotension
- Tachycardia
- Cool/clammy skin
- Organ dysfunction (AMS, oliguria)

Hypovolemic
- Hypotension (resting, orthostatic)
- Tachycardia
- Postural dizziness
- Easily fatigued
- Thirst
- Cramps
- Abdominal pain
- Chest pain
- Lethargy
- Confusion
- Decreased skin turgor
- Necrosis

Obstructive
- Gradual or rapid onset
- Chest pain
- Lightheadedness
- Dyspnea
- Tachypnea
- Hemoptysis
- Cyanosis
- Pleural Rub
Lab Values

Distributive
- WBC: >12 or <12
- Glucose: >140 mg/dL
- CRP: > 2STD
- **Arterial Hypoxemia:** PaO2/FiO2 <300
- Urine output: <0.5mL/kg/hr (at least 2 hours)
- Creatinine: >0.5 mg/dL
- INR: > 1.5; PTT: >60 sec
- Platelets: <100K
- Bilirubin: >4 mg/dL
- **Lactate:** > normal (0-1)

Cardiogenic
- Urine – low
- Creatinine – high
- BUN -high
- Lactate: high
- CVP: > 12 mmHg
- **SvO2:** <70%

Hypovolemic
- Urine – low
- Creatinine - high
- BUN – high
- Sodium – high or low
- Potassium – high or low
- **Metabolic:** acidosis or alkalosis
- Hematocrit - low
- Albumin – low
- CVP < 8 mmHg

Shock in the ICU 70
# Expert Consensus

<table>
<thead>
<tr>
<th>Color</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>Green</td>
<td>Low risk of an adverse event. Proceed as usual according to each ICU’s protocols and procedures.</td>
</tr>
<tr>
<td>Yellow</td>
<td>Potential risk and consequences of an adverse event are higher than green, but may be outweighed by the potential benefits of mobilization. The precautions or contraindications should be clarified prior to any mobilization episode. If mobilized, consideration should be given to doing so gradually and cautiously.</td>
</tr>
<tr>
<td>Red</td>
<td>Significant potential risk or consequences of an adverse event. Active mobilization should not occur unless specifically authorized by the treating intensive care specialist in consultation with the senior physical therapist and senior nursing staff.</td>
</tr>
</tbody>
</table>
# Expert Consensus

## Other Considerations

<table>
<thead>
<tr>
<th></th>
<th>In-Bed Exercises</th>
<th>Out-of-Bed Exercises</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Surgical</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Unstable/unstabilized major fracture</td>
<td>Yellow</td>
<td>Red</td>
</tr>
<tr>
<td>Pelvic</td>
<td>Yellow</td>
<td></td>
</tr>
<tr>
<td>Spinal</td>
<td>Yellow</td>
<td></td>
</tr>
<tr>
<td>Lower limb long bone</td>
<td>Yellow</td>
<td>Red</td>
</tr>
<tr>
<td>Large open surgical wound</td>
<td>Green</td>
<td></td>
</tr>
<tr>
<td>Chest/sternum</td>
<td>Yellow</td>
<td></td>
</tr>
<tr>
<td>Abdomen</td>
<td>Yellow</td>
<td></td>
</tr>
<tr>
<td><strong>Medical</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Known uncontrolled bleeding</td>
<td>Red</td>
<td>Red</td>
</tr>
<tr>
<td>Suspected active bleeding or increased bleeding risk</td>
<td>Yellow</td>
<td>Green</td>
</tr>
<tr>
<td>Patient is febrile with a temperature exceeding acceptable maximum despite physical or pharmacological cooling management</td>
<td>Yellow</td>
<td>Yellow</td>
</tr>
<tr>
<td>Active hypothermia management</td>
<td>Yellow</td>
<td>Yellow</td>
</tr>
<tr>
<td><strong>Other Considerations</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>ICU-acquired weakness</td>
<td>Green</td>
<td>Yellow</td>
</tr>
<tr>
<td>Continuous renal replacement therapy (including femoral dialysis catheters)</td>
<td>Green</td>
<td>Green</td>
</tr>
<tr>
<td>Venous and arterial femoral catheters</td>
<td>Green</td>
<td>Green</td>
</tr>
<tr>
<td>Femoral sheaths</td>
<td>Yellow</td>
<td>Red</td>
</tr>
<tr>
<td>All other drains and attachments, e.g., nasogastric tube, central venous catheter, pleural drain, wound drain, intercostal catheter, urinary catheter</td>
<td>Green</td>
<td>Green</td>
</tr>
</tbody>
</table>

* Patients with large open wounds who have a prolonged stay in ICU may be able to commence mobilization after consultation with the treating surgeon.
* The suspicion of active bleeding is not just about bleeding risk, but the likelihood of an adverse event that will be compounded by an increased bleeding risk, e.g., fall or line displacement.

## Respiratory Considerations

<table>
<thead>
<tr>
<th></th>
<th>In-Bed Exercises</th>
<th>Out-of-Bed Exercises</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Intubation</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Endotracheal tube</td>
<td>Yellow</td>
<td></td>
</tr>
<tr>
<td>Tracheostomy tube</td>
<td>Yellow</td>
<td></td>
</tr>
<tr>
<td><strong>Respiratory parameters</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Fraction of inspired oxygen</td>
<td></td>
<td></td>
</tr>
<tr>
<td>≤ 0.6</td>
<td>Yellow</td>
<td></td>
</tr>
<tr>
<td>&gt; 0.6</td>
<td>Yellow</td>
<td></td>
</tr>
<tr>
<td>Percutaneous oxygen saturation</td>
<td>Yellow</td>
<td>Yellow</td>
</tr>
<tr>
<td>≥ 90%</td>
<td>Yellow</td>
<td></td>
</tr>
<tr>
<td>&lt; 90%</td>
<td>Yellow</td>
<td></td>
</tr>
<tr>
<td>Respiratory rate</td>
<td></td>
<td></td>
</tr>
<tr>
<td>≤ 30 bpm</td>
<td>Yellow</td>
<td></td>
</tr>
<tr>
<td>&gt; 30 bpm</td>
<td>Yellow</td>
<td></td>
</tr>
<tr>
<td><strong>Ventilation</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mode HFOV</td>
<td>Yellow</td>
<td></td>
</tr>
<tr>
<td><strong>PEEP</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>≤ 10 cmH₂O</td>
<td>Yellow</td>
<td></td>
</tr>
<tr>
<td>&gt; 10 cmH₂O</td>
<td>Yellow</td>
<td></td>
</tr>
<tr>
<td><strong>Ventilator dysynchrony</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Rescue therapies</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Nitric oxide</td>
<td>Yellow</td>
<td></td>
</tr>
<tr>
<td>Prostacyclin</td>
<td>Yellow</td>
<td></td>
</tr>
<tr>
<td>Prone positioning</td>
<td>Yellow</td>
<td></td>
</tr>
</tbody>
</table>

## Known or Suspected Severe Aortic Disease

- Cardiac ischemia (defined as ongoing chest pain and/or dynamic EKG changes)
- Transvenous or epicardial pacemaker insertion
- Not requiring pharmacological treatment and not awaiting emergency pacemaker insertion
- Dependent rhythm
- Stable underlying rhythm

* IABP = intra-aortic balloon pump, ECMO = extracorporeal membrane oxygenation, bpm = beats per minute, MAP = mean arterial pressure, PAP = deep vein thrombosis, PE = pulmonary embolus.
* This may be a yellow (easy) for in-bed activities of the blood pressure is within target range as documented by the medical team.
* Experienced ICU practitioners were considered to have good judgment about the impact of cardiovascular morbidity and/or accident or medication or high levels of hemodynamic support, on the ability to intervene. However, if the case of uncertainty or lack of experience, it is recommended that the decision to mobilize a patient is discussed with appropriate experienced ICU staff. The target mean arterial pressure is determined by the treating ICU team.
* Cyclic and hip flexion may be contraindicated in the leg where the IABP/ECMO is inserted. If so, in-bed exercises might need to be modified to limit hip flexion.
Barriers to Therapy

- Timing of Procedures
- Femoral vascular access
- Agitation/low GCS
- Hemodynamic instability
- Neurological instability
- Respiratory instability
Early Mobilization

• Barriers – most common is oxygen desaturation
• Most mobilization studies are on ARDS – often caused from sepsis.
• No research demonstrating effect on Lactate changes
• No research demonstrating effect on SvO2 changes
• Both lactate and SvO2 change quickly with exercise. How do we determine if this is transient?
Where to start?

- Start small
  - Stable patients who present in earlier stages
  - Familiarize yourself with the terminology/labs/measures/symptoms
  - Consider how exercise impacts these values

- Multidisciplinary communication
  - Ask questions!
  - Prioritization of patients
  - Might I make this situation worse?
  - Plan of care, goals should reflect expectations based on whole patient picture

- Documentation
  - Vitals
  - Impact medical status may have on function
References


References


