Fundamentals of Critical Care:
Hemodynamics, Monitoring, Shock

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Definitions and Principles

- The measurement and interpretation of biological systems that describe performance of the cardiovascular system
- Monitoring is NOT therapy
- Clinicians must know how to interpret the data
- Very few randomized controlled trials
Oxygen Delivery is the Goal

**Oxygen Delivery**

\[
DO_2 \text{ (mL O}_2/\text{min}) = CO \text{ (L/min)} \times CaO_2 \text{ (mL O}_2/\text{dL}) \times 10
\]

\[
CO \text{ (L/min)} = HR \text{ (beats/min)} \times SV \text{ (L/beat)}
\]

\[
CaO_2 \text{ (mL O}_2/\text{dL}) = [1.34 \times (Hb)(g/dL) \times SaO_2] + [.003 \times PaO_2 \text{ mm Hg}]
\]

**Oxygen Consumption**

\[
CVO_2 \text{ (mL O}_2/\text{dL}) = [1.34 \times (Hb)(g/dL) \times SVO_2] + [.003 \times PVO_2 \text{ mm Hg}]
\]

\[
VO_2 \text{ (mL O}_2/\text{min}) = CO \times 3(CaO_2 - CVO_2) \times 10
\]
Determinants of Cardiac Performance

- **Preload**
  - Estimated by end-diastolic volume (pressure)
  - CVP for RVEDV, PAOP (wedge) for LVEDV

- **Afterload**
  - \(\text{SVR} = \frac{\text{MAP-CVP}}{\text{CO}} \times 80\)

- **Contractility**
Methods of Hemodynamic Monitoring

- Arterial Blood Pressure
  - Non-invasive
  - Direct arterial pressure measurement
- Central Venous Pressure
- The Pulmonary Artery Catheter
- Cardiac Output Measurement
- Tissue Oxygenation
Non-invasive Blood Pressure Monitoring
Non-invasive Blood Pressure Measurement

- Manual or automated devices
- Method of measurement
  - Oscillometric (most common)
    - MAP most accurate, DP least accurate
  - Auscultatory (Korotkoff sounds)
    - MAP is calculated
  - Combination
Limitations of Non-invasive Blood Pressure Monitoring

- Cuff must be placed correctly and must be appropriately sized
- Auscultatory method is very inaccurate
  - Korotkoff sounds difficult to hear
  - Significant underestimation in low-flow (i.e. shock) states
- Oscillometric measurements also commonly inaccurate (> 5 mm Hg off directly recorded pressures)
Direct Arterial Blood Pressure Measurement
Indications for Arterial Catheterization

- Need for continuous blood pressure measurement
  - Hemodynamic instability
  - Vasopressor requirement
- Respiratory failure
  - Frequent arterial blood gas assessments
- Most common locations: radial, femoral, axillary, and dorsalis pedis
Complications of Arterial Catheterization

- Hemorrhage
- Hematoma
- Thrombosis
- Proximal or distal embolization
- Pseudoaneurysm
- Infection
Pseudoaneurysm

Fig. 1 – Photography of colour Doppler result showing right axillary artery pseudoaneurysm
Limitations of Arterial Catheterization

- Pressure does not accurately reflect flow when vascular impedance is abnormal
- Systolic pressure amplification
  - Mean pressure is more accurate
- Recording artifacts
  - Underdamping
  - Overdamping
Waveform Distortion

![Diagram showing the relationship between damping coefficient, natural frequency, and dynamic response types: Overdamped, Underdamped, Adequate dynamic response, and Unacceptable. The diagram illustrates how different damping coefficients affect the response at various natural frequencies.](image)
Central Venous Catheterization

- **Central venous pressure**
  - Right atrial (superior vena cava) pressure
  - Limited by respiratory variation and PEEP

- **Central venous oxygen saturation**
  - SCVO$_2$
  - Correlates with SMVO$_2$ assuming stable cardiac function
  - Goal-directed resuscitation in severe sepsis and septic shock (Rivers, et al)
Central Venous Pressure Waveform
The Pulmonary Artery Catheter

- HJC Swan and Santa Monica Bay sailboats (NEJM 1970)
- Widespread use in critically ill patients
- Remains controversial
  - Lack of prospective, randomized trials
  - PAC data are only as good as the clinicians’ interpretation and application
- Measures CVP, PAP, PAOP, Cardiac Index and Svo₂
- Approximately 1 million PACs placed annually
Pulmonary Artery Catheter
Indications for Pulmonary Artery Catheterization

- Identification of the type of shock
  - Cardiogenic (acute MI)
  - Hypovolemic (hemorrhagic)
  - Obstructive (PE, cardiac tamponade)
  - Distributive (septic)
  - Many critically ill patients exhibit elements of more than 1 shock classification

- Monitoring the effectiveness of therapy
Normal Hemodynamic Values

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>SVO2</td>
<td>60-75%</td>
</tr>
<tr>
<td>Stroke volume</td>
<td>50-100 mL</td>
</tr>
<tr>
<td>Stroke index</td>
<td>25-45 mL/M²</td>
</tr>
<tr>
<td>Cardiac output</td>
<td>4-8 L/min</td>
</tr>
<tr>
<td>Cardiac index</td>
<td>2.5-4.0 L/min/M²</td>
</tr>
<tr>
<td>MAP</td>
<td>60-100 mm Hg</td>
</tr>
<tr>
<td>CVP</td>
<td>2-6 mm Hg</td>
</tr>
<tr>
<td>PAP systolic</td>
<td>20-30 mm Hg</td>
</tr>
<tr>
<td>PAP diastolic</td>
<td>5-15 mm Hg</td>
</tr>
<tr>
<td>PAOP (wedge)</td>
<td>8-12 mm Hg</td>
</tr>
<tr>
<td>SVR</td>
<td>900-1300 dynes·sec·cm⁻⁵</td>
</tr>
</tbody>
</table>
# Hemodynamic Profiles in Shock

<table>
<thead>
<tr>
<th>Class of Shock</th>
<th>CVP</th>
<th>PAOP</th>
<th>CO/CI</th>
<th>SVR</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cardiogenic</td>
<td>↑</td>
<td>↑</td>
<td>↓</td>
<td>↑</td>
</tr>
<tr>
<td>Hypovolemic</td>
<td>↓</td>
<td>↓</td>
<td>↓</td>
<td>↑</td>
</tr>
<tr>
<td>Hyperdynamic septic</td>
<td>↑</td>
<td>↔</td>
<td>↑</td>
<td>↓</td>
</tr>
<tr>
<td>Hypodynamic septic</td>
<td>↓</td>
<td>↓</td>
<td>↓</td>
<td>↓</td>
</tr>
</tbody>
</table>
Pulmonary Artery Catheter Placement
Complications of Pulmonary Artery Catheterization

- General central line complications
  - Pneumothorax
  - Arterial injury
  - Infection
  - Embolization
- Inability to place PAC into PA
- Arrhythmias (heart block)
- Pulmonary artery rupture
The Pulmonary Artery Catheter Controversy

- Accuracy of data affected by many conditions common in critically ill patients
- Lack of prospective randomized data supporting better outcomes with PAC
- Lack of consensus about goals of therapy
- Paucity of standard guidelines for use
- Limited by the ability of the clinician to accurately interpret PAC data
PAC-directed Supranormal Hemodynamics in Surgical Patients

- Prospective trial by Shoemaker, et al
- Observed that among high-risk surgical patients, survivors demonstrated supranormal hemodynamics
- 88 patients randomized to:
  - CVP-control group
  - PAC-control group (goal was normal hemodynamics and oxygen transport)
  - PAC-protocol group (goal was supranormal hemodynamics and oxygen transport)
- Mortality benefit in PAC-protocol group

_Chest_ 1988;94;1176-1186
PAC for Goal-Directed Therapy in High-Risk Surgical Patients

- NEJM January 2003 [348(1):5-14]
- RCT by Canadian Critical Care Clinical Trials Group
- 1994 patients, 60 or older, ASA III/IV, required ICU post-op
- No mortality benefit, No days of hospitalization benefit
- Higher rate of pulmonary embolism in PAC group
Meta-analysis of Randomized Clinical Trials of PACs

- 5051 patients in 13 RCTs
  - 8 RCTs (2667) were surgical patients
  - 3 RCTs (910) were sepsis/ARDS patients
- Results:
  - No significant change in mortality with PAC
  - No significant change in days of hospitalization with PAC
<table>
<thead>
<tr>
<th>Source</th>
<th>Total No. of Patients</th>
<th>PAC</th>
<th>No PAC</th>
<th>Odds Ratio (95% CI)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Schultz et al, 1985</td>
<td>1/35</td>
<td>10/35</td>
<td>0.11 (0.02-0.63)</td>
<td></td>
</tr>
<tr>
<td>Shoemaker et al, 1988</td>
<td>11/58</td>
<td>7/30</td>
<td>0.76 (0.27-2.15)</td>
<td></td>
</tr>
<tr>
<td>Isaacson et al, 1990</td>
<td>1/49</td>
<td>0/53</td>
<td>NA</td>
<td></td>
</tr>
<tr>
<td>Berlauk et al, 1991</td>
<td>1/66</td>
<td>2/21</td>
<td>0.18 (0.02-1.42)</td>
<td></td>
</tr>
<tr>
<td>Guyatt, 1991</td>
<td>10/16</td>
<td>9/17</td>
<td>1.10 (0.29-4.22)</td>
<td></td>
</tr>
<tr>
<td>Bender et al, 1997</td>
<td>1/51</td>
<td>1/53</td>
<td>1.04 (0.11-9.95)</td>
<td></td>
</tr>
<tr>
<td>Valentine et al, 1998</td>
<td>3/60</td>
<td>1/60</td>
<td>2.38 (0.35-16.29)</td>
<td></td>
</tr>
<tr>
<td>Bonazzi et al, 2002</td>
<td>0/50</td>
<td>0/50</td>
<td>NA</td>
<td></td>
</tr>
<tr>
<td>Rhodes et al, 2002</td>
<td>46/95</td>
<td>50/106</td>
<td>1.01 (0.58-1.75)</td>
<td></td>
</tr>
<tr>
<td>Sandham et al, 2003</td>
<td>163/997</td>
<td>155/997</td>
<td>1.06 (0.83-1.35)</td>
<td></td>
</tr>
<tr>
<td>Richard et al, 2003</td>
<td>198/338</td>
<td>208/343</td>
<td>0.93 (0.66-1.26)</td>
<td></td>
</tr>
<tr>
<td>ESCAPE, 2005</td>
<td>45/215</td>
<td>38/218</td>
<td>1.25 (0.78-2.02)</td>
<td></td>
</tr>
<tr>
<td>Harvey et al, 2005 (PAC-Man)</td>
<td>346/506</td>
<td>333/507</td>
<td>1.13 (0.57-1.47)</td>
<td></td>
</tr>
<tr>
<td>Combined</td>
<td></td>
<td>1/04</td>
<td>1.04 (0.90-1.20)</td>
<td></td>
</tr>
</tbody>
</table>

*JAMA 2005; 294(13):1664-1670*
<table>
<thead>
<tr>
<th>Source</th>
<th>PAC</th>
<th>No PAC</th>
<th>Mean Difference (95% CI)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Shoemaker et al.¹⁶ 1988</td>
<td>22.4 (58)</td>
<td>22.2 (30)</td>
<td>0.15 (~6.88 to 7.28)</td>
</tr>
<tr>
<td>Isaacson et al.¹⁷ 1990</td>
<td>10.2 (49)</td>
<td>9.4 (53)</td>
<td>0.80 (~2.20 to 3.80)</td>
</tr>
<tr>
<td>Berlauk et al.¹⁸ 1991</td>
<td>18.9 (66)</td>
<td>15.4 (21)</td>
<td>3.51 (~1.05 to 8.07)</td>
</tr>
<tr>
<td>Guyatt,¹⁹ 1991</td>
<td>10.3 (16)</td>
<td>8.1 (17)</td>
<td>2.20 (~5.60 to 10.20)</td>
</tr>
<tr>
<td>Bender et al.²⁰ 1997</td>
<td>12.5 (51)</td>
<td>12.0 (53)</td>
<td>0.50 (~3.33 to 4.33)</td>
</tr>
<tr>
<td>Valentine et al.²¹ 1998</td>
<td>13.0 (60)</td>
<td>13.0 (60)</td>
<td>0.00 (~5.65 to 5.65)</td>
</tr>
<tr>
<td>Rhodes et al.²³ 2002</td>
<td>13.0 (95)</td>
<td>14.0 (106)</td>
<td>~1.20 (~11.10 to 8.70)</td>
</tr>
<tr>
<td>Sandham et al.²⁴ 2003</td>
<td>10.0 (997)</td>
<td>10.0 (997)</td>
<td>0.00 (~0.62 to 0.62)</td>
</tr>
<tr>
<td>Richard et al.²⁵ 2003</td>
<td>14.0 (335)</td>
<td>14.4 (341)</td>
<td>~0.40 (~2.13 to 1.33)</td>
</tr>
<tr>
<td>ESCAPE,¹⁰ 2005</td>
<td>17.0 (215)</td>
<td>16.1 (218)</td>
<td>0.90 (~2.54 to 4.34)</td>
</tr>
<tr>
<td>Harvey et al.¹⁴ 2005 (PAC-Man)</td>
<td>48.9 (304)</td>
<td>52.4 (291)</td>
<td>~3.50 (~11.21 to 4.21)</td>
</tr>
</tbody>
</table>

Combined: 0.11 (~0.51 to 0.74)
Cardiac Output Measurement

- Multiple techniques
  - Thermodilution – most common
  - Transpulmonary
  - Pulse contour analysis
  - Esophageal Doppler

- Newer pulmonary artery catheters offer continuous cardiac output measurement
Thermodilution Method of Cardiac Output Measurement
Tissue Oxygenation

- Despite advances, our ability to monitor the microcirculation and tissue perfusion is limited
- Laboratory tests for metabolic acidosis are global and insensitive
- Newer technology on the horizon
  - Gastric tonometry
  - Sublingual capnometry
Shock

“The rude unhinging of the machinery of life” - Henry Gross, 1872

End-organ cellular dysfunction due to tissue hypoperfusion

Types

- Hypovolemic (hemorrhagic)
- Cardiogenic (myocardial infarction)
- Distributive (septic, neurogenic, anaphylactic)
- Obstructive (cardiac tamponade, tension pneumothorax, massive pulmonary embolism)
Cellular Pathophysiology of Shock

- Inadequate perfusion
  - Cell hypoxia
  - Energy deficit
  - Lactic acid accumulation and fall in pH
  - Anaerobic metabolism

- Metabolic acidosis
  - Vasoconstriction
  - Failure of pre-capillary sphincters
  - Peripheral pooling of blood
  - Cell membrane dysfunction and failure of 'sodium pump'
  - Intracellular lysosomes release digestive enzymes
  - Efflux of potassium
  - Influx of sodium and water
  - Toxic substances enter circulation
  - Capillary endothelium damaged
  - Further destruction, dysfunction and cell death
Pathophysiology of Shock

- Decreased cardiac output
  - Decreased venous return
  - Decreased myocardial contraction
  - Intracellular fluid loss
  - Metabolic acidosis
  - Cell hypoxia

- Decreased blood pressure
  - Decreased myocardial function
  - Decreased coronary perfusion

- Decreased tissue perfusion
  - Microcirculatory damage
  - Microcirculatory obstruction

  - Cellular aggregation
Shock in the Trauma Patient

- **Airway**
  - Hypoxia secondary to maxillofacial trauma, laryngeal injury, proximal cervical spine injury

- **Breathing**
  - Hypoxia secondary to pneumothorax, hemothorax, bronchial injury

- **Circulation**
  - Hemorrhagic
  - Cardiogenic secondary to contusion
  - Obstructive secondary to tamponade
  - Distributive secondary to spinal cord injury
# Stages of Hemorrhagic Shock

<table>
<thead>
<tr>
<th>Stage</th>
<th>I (compensated)</th>
<th>II (mild)</th>
<th>III (moderate)</th>
<th>IV (severe)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Blood loss</td>
<td>15% - 30% (750 - 1,000 ml)</td>
<td>15% - 30% (1,000 - 1,500 ml)</td>
<td>30% - 40% (1,500 - 2,000 ml)</td>
<td>&gt;40% (2,000 ml or more)</td>
</tr>
<tr>
<td>Heart rate</td>
<td>Normal (&lt;100 bpm)</td>
<td>Tachycardia (&gt;100 bpm)</td>
<td>Tachycardia (&gt;120 bpm)</td>
<td>Tachycardia (&gt;140 bpm)</td>
</tr>
<tr>
<td>BP</td>
<td>Normal; vasoconstriction redistributes blood flow; slight rise in diastolic pressure seen</td>
<td>Orthostatic changes; BP vasoconstrictions intensifies in non-critical organs (skin, muscles, gut)</td>
<td>Markedly decreased (SBP &lt;90 mm Hg); vasoconstriction decreases perfusion to kidneys, pancreas, liver, and spleen</td>
<td>Profoundly decreased (SBP &lt;80 mm Hg); decreased perfusion affects the brain and heart</td>
</tr>
<tr>
<td>Respiration</td>
<td>Normal</td>
<td>Rate mildly increased</td>
<td>Moderate tachypnea</td>
<td>Marked tachypnea; respiratory collapse</td>
</tr>
<tr>
<td>Capillary refill time</td>
<td>Normal (&lt;2 seconds)</td>
<td>&gt;2 seconds; clammy skin</td>
<td>Usually &gt;3 seconds; cool, pale skin</td>
<td>&gt;3 seconds; cold, mottled skin</td>
</tr>
<tr>
<td>Bowel sounds</td>
<td>Present, all four quadrants</td>
<td>Hypoactive</td>
<td>Absent (paralytic ileus)</td>
<td>Absent (paralytic ileus, mucosal necrosis)</td>
</tr>
<tr>
<td>Urinary output</td>
<td>&gt;30 ml/hr</td>
<td>20 - 30 ml/hr</td>
<td>&lt;20 ml/hr</td>
<td>None (anuria)</td>
</tr>
<tr>
<td>Mental status</td>
<td>Normal or slightly anxious</td>
<td>Mildly anxious or agitated</td>
<td>Confused, agitated</td>
<td>Obtunded</td>
</tr>
</tbody>
</table>
Management of Shock

1. Recognize
2. Relocate (if necessary)
3. Restore volume
4. Remedy the primary cause
5. Replace catecholamines
Vasopressors/Inotropes

- Dopamine
- Dobutamine
- Epinephrine
- Phenylephrine
- Norepinephrine
- Vasopressin
Dopamine

- Dose dependent receptor activation
  - Low dose - increases blood flow via dopamine receptors in renal, mesenteric, cerebral circulation
  - Intermediate dose - increases cardiac output via $\beta$-receptors
  - High dose - progressive vasoconstriction via $\alpha$-receptors in systemic and pulmonary circulation

- *In vivo*, receptor effects are often mixed
- Tachyarrhythmias are most common complication
- Low dose dopamine has no proven renal benefit
- Significant immunosuppressive effects through suppression of prolactin from hypothalamus
Dobutamine

- Synthetic catecholamine generally considered the drug of choice for severe systolic heart failure
- Increases cardiac output via $\beta_1$-receptor and causes vasodilation via $\beta_2$-receptor
- Inotropic and chronotropic effects are highly variable in critically ill patients
- Data supports use in septic shock when cardiac output remains low despite volume resuscitation and vasopressor support
Epinephrine

- The most potent adrenergic agent available
- Potency and high risk of adverse effects limit use to cardiac arrest (and specific situations after cardiac surgery)
- Primarily β-receptor effects at low doses and α-receptor effects at high doses
- Drug of choice in anaphylactic shock
- Arrhythmogenic
Phenylephrine

- Relatively pure $\beta$-adrenergic agonist
- Minimal inotropic effects; often causes reflex bradycardia
- Consistently decreases cardiac output
- Increased propensity to cause ischemic complications
- Limited use in shock
- Be wary in the OR
Norepinephrine

- More potent vasoconstrictor than dopamine; some inotropic effect
  - Potent $\alpha_1$ stimulation
  - Moderate $\beta_1$ activity
  - Minimal $\beta_2$ activity
- Use has changed from rescue drug in refractory septic shock to primary agent
- Nonrandomized prospective trial from France in 2000 showed mortality benefit

Vasopressin

- Acts on vascular smooth muscle via V1 receptors, independent of adrenergic receptors
- Adrenergic responsiveness typically down-regulated in septic shock
- Considered replacement therapy
- Traditionally not titrated
- Significant splanchnic vasoconstriction
Dopamine in the SOAP Study

- Sepsis Occurrence in Acutely Ill Patients
- 1058 patients in shock (3147 total) from multi-center observational trial
- 35% of patients in shock received dopamine
- Multivariate analysis identified dopamine administration as an independent risk factor for ICU and hospital mortality (20% higher)

Vasopressin vs. Norepinephrine

- RCT of 778 patients in septic shock receiving norepinephrine
- Randomized to vasopressin or norepinephrine
- No significant difference in 28-day mortality rate or overall rate of severe adverse events
- Lower mortality rate with vasopressin in less severe septic shock (no difference in more severe septic shock) but the significance of this data is uncertain

Dopamine vs. Norepinephrine

- Multicenter RCT of 1679 patients in shock
- Randomized to dopamine or norepinephrine as first-line vasopressor
- No difference in 28-day mortality
- More arrhythmic events with dopamine
- Subgroup analysis showed higher mortality with dopamine in cardiogenic shock (no difference with septic shock or hypovolemic shock)

Conclusions

- Multiple different methods of hemodynamic monitoring
- Keys to success
  - Know when to use which method
  - Technical skills for device placement
  - Know how to interpret the data
- Remember the limitations of the technology
Conclusions

- Early identification and treatment of shock is essential
- Early stages of shock are often very difficult to identify
- Understanding how the different adrenergic agents work is essential
- More prospective trials are needed to standardize the use of vasopressors and inotropes