Dynamic Hemodynamic

Alicia Williams, DNP, MBA, CRNP, CCNS
Eugene Robin:

The Cult of the Swan Ganz Catheter, Overuse & Abuse of Pulmonary Flow Catheters, Ann Int Med, 1985

The widespread use is a form of cultism.

Based on unsupported beliefs...

Fostered by an uncritical literature.....
Hemodynamics

- **Right atrial pressure (RAP)**
  - Normal range: 2-6 mmHg
  - Mean: 2-6 mmHg

- **Right ventricular pressure (RVP)**
  - Normal range:
    - Systolic: 20-30 mmHg
    - Diastolic: 0-5 mmHg
  - Mean: 2-6 mmHg

- **Pulmonary artery pressure (PAP)**
  - Normal range:
    - Systolic: 20-30 mmHg
    - Diastolic: 10-20 mmHg
  - Mean: 10-15 mmHg

- **Pulmonary artery wedge pressure (RA)**
  - Normal range:
    - Systolic: 20-30 mmHg
    - Diastolic: 10-20 mmHg
  - Mean: 4-12 mmHg
Indications for Hemodynamic Monitoring

- Assessment of cardiovascular function and response to interventions
- Evaluate and Optimize CO
- Put numbers to the concepts of preload, afterload, contractility
- Peri-op monitoring: High risk patients
- Shock States
- Assessment & treatment of pulmonary conditions
- Assessment of intravascular volume status
Principles of hemodynamic monitoring

- No HDM technique alone can improve outcome
- Monitoring requirements vary over time
- No optimal HD values apply to all patients
- We need to combine & integrate variables
- High CO is not always a good thing
- CO is estimated, not measured
- Continuous measures of HD variables is preferable
- Non-invasiveness is not the always best
- Some measured, other parameters derived

Hemodynamic Review

Preload

Afterload

Contractility

Stroke Volume

Cardiac Output

Heart Rate

O₂ Delivery

Hb (O₂ capacity)

O₂ binding (SₐO₂)

O₂ dissolved (PₐO₂)

Oxygen Content
Tissue Oxygenation

- O2 saturation
- Haemoglobin

- Venous return
- Intravascular volume
- Pump function / valves
- Heart rate / rhythm

Oxygen content

Cardiac output

Regional blood flow

Blood pressure

- Vasomotor tone and regional distribution

- Vasomotor tone (vascular resistance)
Fluid can be harmful
Fluid Responsiveness

- Stroke volume
- Preload responders
- Preload non-responders

Normal ventricular contractility
Impaired ventricular contractility

A → B
Ventricular preload
Oxygen delivery, $DO_2$

$$DO_2 = Arterial \ O_2 \ content \times \ Cardiac \ output$$

- $SaO_2$ ($PaO_2$)
- Hemoglobin
- Heart rate
- Preload
- Afterload
- Contractility
- Dobutamine
- Fluids
- Nitrites
- Oxygen PEEP
- Transfusion
- Pacemaker isoproterenol
- Nitrates
- Transfusion-related risks
- Not very effective (once $SaO_2$ is > 90%)
- $O_2$ toxicity/RV impairment (high PEEP)
- Not very effective (unless severe bradycardia)
- Tachycardia
- Increased MVO$_2$
- Tachycardia
- Increased MVO$_2$
- Lung edema
- Arterial hypotension
Static “vs” Dynamic

- CVP
- PAOP
- Transoesophageal doppler
- GEDV
- ITBV
- Echo (end-diastole diameter/area)

- SVV
- PPV
- Pleth Variability Index
- IVC collapsibility
- PLR
- Fluid challenge
Preload

Volume coming into ventricles (end diastolic pressure)

Increased in:
- Hypervolemia
- Regurgitation of cardiac values

Afterload

Resistance - left ventricle must overcome to circulate blood

Increased in:
- Hypertension
- Vasoconstriction
Preload

RH
CVP
RAP
RVEDP
RVEDV

LH
PAWP
LAP
LVEDP
LVEDV

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ASSUMES Pressure = Volume
Frank Starling Law

Increase LVEDV $\rightarrow$ increase fiber length stretch

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Frank-Starling Curve: Preload

- Patient A: Normal LV function
- Patient B: LV dysfunction
- Hypovolemia
- LVEDV
CVP: A useful but not so simple measurement.

- Lack of usefulness due to failure to consider physiologic determinants and potential errors in measurement

- CVP is there to be used by the thoughtful clinician as long as respect is paid to basic physiologic principles as well as principles of measurement…….
The Problem with pressure measurements: How to Manage PAoP=20 mmHg???

Normal LV: with high EDV

Juxtacardiac pressure with nl. or low EDV

Hypertrophied non-compliant LV with low EDV

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Increase in preload (PAWP) results in an increase in CO?

<table>
<thead>
<tr>
<th>Author</th>
<th>Journal</th>
<th>Year</th>
<th>Indicator</th>
<th>Correlation Coefficient</th>
</tr>
</thead>
<tbody>
<tr>
<td>Stites S</td>
<td>Chest 1993</td>
<td>1993</td>
<td>PAWP/CI</td>
<td>r = 0.06</td>
</tr>
<tr>
<td>Eddy V.</td>
<td>Chest 1993</td>
<td>1993</td>
<td>PAWP/CI</td>
<td>r = 0.12</td>
</tr>
<tr>
<td>Vukmir R.</td>
<td>CCM 1993</td>
<td>1993</td>
<td>PAWP/CO</td>
<td>r = 0.20</td>
</tr>
</tbody>
</table>


N=96 septic pts, 150 volume challenges

No difference in CVP or WP & CI between those who responded to volume, & those who did not.

“cardiac filling pressures are poor predictors of fluid resuscitation in septic patients”
Pressures are not reliable indicators of cardiac preload

\[ y = -0.022x + 0.056 \]
\[ r^2 = 0.005 \]
\[ r = 0.069 \]
\[ n = 299 \]

\[ y = -0.005x + 0.05 \]
\[ r^2 = 0.0003 \]
\[ r = 0.018 \]
\[ n = 199 \]

Lichtwarck-Aschoff M. et al, Intensiv Care Med 18: 1992
Opportunity to Consider Alternatives!!
# Predictive Value of Fluid Responsiveness

*Marik et al. (2011)*

## Table 2 Predictive value of techniques used to determine fluid responsiveness [15]

<table>
<thead>
<tr>
<th>Method</th>
<th>Technology</th>
<th>AUC*</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pulse pressure variation (PPV)</td>
<td>Arterial waveform</td>
<td>0.94 (0.93-0.95)</td>
</tr>
<tr>
<td>Systolic pressure variation (SPV)</td>
<td>Arterial waveform</td>
<td>0.86 (0.82-0.90)</td>
</tr>
<tr>
<td>Stroke volume variation (SW)</td>
<td>Pulse contour analysis</td>
<td>0.84 (0.78-0.88)</td>
</tr>
<tr>
<td>Left ventricular end-diastolic area (LVEDA)</td>
<td>Echocardiography</td>
<td>0.64 (0.53-0.74)</td>
</tr>
<tr>
<td>Global end-diastolic volume (GEDV)</td>
<td>Transpulmonary thermodilution</td>
<td>0.56 (0.37-0.67)</td>
</tr>
<tr>
<td>Central venous pressure (CVP)</td>
<td>Central venous catheter</td>
<td>0.55 (0.48-0.62)</td>
</tr>
</tbody>
</table>

*AUC = area under the curve with 95% confidence intervals.*
Assessment of volume responsiveness

- CVP and PAOP poor predictors of fluid status
  - Cardiac filling pressures did not predict fluid responders from non-responders. [Osman, et al. CCM 2007]

- Fluid responsiveness
  - Static markers versus dynamic markers
Dynamic parameters

- Systolic pressure variation
- Pulse pressure variation
- Stroke volume variation
- Pleth variability index
Pulsus Paradoxus

- Origin of SVV value.

- Reverse Pulses Paradoxus
  - Occurs during positive pressure ventilation.

- Clinical use of this phenomenon remains “marginal”
Stroke Volume Variation
Pulse Pressure Variation

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Pulse Wave-Contour

- Detected by the use of an arterially placed catheter with a pressure transducer, which can measure pressure tracings on a beat-to-beat basis.
Injectate temperature sensor

Central venous catheter

Central arterial thermodilution catheter

Disposable pressure transducer

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Indications for dynamic monitoring

1. Evaluate the **response** to fluid interventions

2. Determine or **predict** the patient’s potential response to fluid therapy

- Normal PPV & SVV < 10-15%
- If variability is low, need for fluid low
- If variability is high, need for fluid is high
Relationship of SVV to SV

Figure 1. SVV- SV Plot Aggregate Data. N 84 OR & ICU Patients.

SVV is inversely related to SV.

McGee W. et. Al. SCCM 2007 CCM suppl Abs 227
Preload Responsiveness

- **Patient A is preload responsive**
  - On steep portion of the curve
  - Fluid bolus produces large increase in SV
  - SVV > 10 – 15 %

- **Patient B is not preload responsive**
  - On flat portion of the curve
  - Fluid bolus does not produce the same amount of increase in SV
  - SVV < 10 – 15%

Modified Concepts from Parry-Jones, Michard, et al.

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Limitations

- Mechanical Ventilation
- Arrhythmias
- Arrhythmias
- PEEP
- Increased abdominal pressure
- Open chest
Passive Leg Raising

- 150 – 300 ml volume
- Effects < 30 sec.. Not more than 4 minutes
- Self-volume challenge; Reversible

How to:
- Pivot bed automatically (in some beds)
- Trunk is tilted supine, lower limbs raised to 45° angle
- Angle between the trunk and lower limbs remains unchanged (135°)

Monnet 2007, artwork from www.medtrng.com
PLR Effects on Starling Curve

- If the increase in preload induced by PLR → significant changes in SV (a to b), the patient will likely be fluid responsive.

- If the same changes in preload during PLR do not significantly change SV (a’ to b’), the heart is likely preload independent - fluid should not be administered.

Monnet 2007
## PLR Accurate Diagnostic Method?

<table>
<thead>
<tr>
<th>Reference</th>
<th>Index</th>
<th>Device</th>
<th>% Responders</th>
<th>$r$</th>
<th>AUC (SEM)</th>
<th>Sens</th>
<th>Spec</th>
</tr>
</thead>
<tbody>
<tr>
<td>Lamia et al(^1), 2007</td>
<td>cVTIAo</td>
<td>TTE</td>
<td>54</td>
<td>0.83</td>
<td>0.96 (0.040)</td>
<td>77</td>
<td>100</td>
</tr>
<tr>
<td>Maizel et al(^2), 2007</td>
<td>cCO</td>
<td>TTE</td>
<td>54</td>
<td>0.79</td>
<td>0.89 (0.059)</td>
<td>88</td>
<td>83</td>
</tr>
<tr>
<td>Maizel et al(^2), 2007</td>
<td>cSV%</td>
<td>TTE</td>
<td>50</td>
<td>0.56</td>
<td>0.96 (0.030)</td>
<td>100</td>
<td>80</td>
</tr>
<tr>
<td>Biais et al(^3), 2009</td>
<td>cSV% (TTE)</td>
<td>TTE</td>
<td>67</td>
<td>—</td>
<td>0.94 (0.040)</td>
<td>86</td>
<td>90</td>
</tr>
<tr>
<td>Preau et al(^4), 2010</td>
<td>cSV%</td>
<td>TTE</td>
<td>41</td>
<td>—</td>
<td>0.95 (0.02–0.97)</td>
<td>89.4</td>
<td>91.4</td>
</tr>
<tr>
<td>Overall</td>
<td>—</td>
<td>—</td>
<td>52.9</td>
<td>0.81</td>
<td>0.95 (0.92–0.97)</td>
<td>89.4</td>
<td>91.4</td>
</tr>
</tbody>
</table>

\(^1\) Lamia, P., et al. (2007). 
\(^3\) Biais, R., et al. (2009). 
IAH on Hemodynamics

- Decreased CO
  - Compression of the inferior vena cava and portal vein
  - Reduced blood return to the heart
  - Increased afterload

- Reduced Stroke volume

- Tachycardia

- Increased pressure on great vessels making hemodynamic monitoring challenging with falsely elevated and misleading pressures

- Increased risk for thromboembolic events secondary to venous stasis
Volumetric Measurements

- Right-ventricular end-diastolic volume,
- Global end-diastolic volume, and
- Intrathoracic blood volume
Key principles of hemodynamic monitoring

- No HDM technique alone can improve outcome
- Monitoring requirements vary over time, depend on available equipment & training
- No optimal HD values apply to all patients
- CO is estimated, not measured
- Monitoring HD changes over short periods of time is important
- Continuous measures of HD variables is preferable
- Non-invasiveness is not the only goal
- Some measured, other parameters derived
“No monitoring device, no matter how accurate or complete, would be expected to improve patient outcome, unless coupled to a treatment that itself improves outcome”
References


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Questions
Questions