What Do You Know About Cardiac Hemodynamics?

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Overview

• Hemodynamics
  – Basic principles and definitions
  – Hemodynamic guided therapy
  – Exercise
  – Constriction and Restriction
• Endomyocardial biopsy
  • Coronary angiography
These hemodynamics represent:

a) Aortic stenosis
b) Aortic regurgitation
c) Hypertension
d) Coarctation
e) Artifact
Hemodynamics are only useful if you understand how they were obtained.
Primary Indications
- Unclear hemodynamic picture
- Pulmonary hypertension
- Consideration of Advanced Therapies
Hemodynamic Principles
Hemodynamic Principles

Pressure is only surrogate for volume

Burkoff, Maurer, Packer. Circulation 2003;107:656
systolic ejection period

diastolic filling period
Hemodynamics Change Over Time

Limitations of measurements at a single point in time

This waveform is consistent with all of the following except:

a) Tamponade
b) Constriction
c) Restriction
d) Transplantation
e) RV infarction
Central venous pressure

**Things to consider**

- Inspiratory fall of 2 – 3 mmHg
- Atrial events are “out of phase” from ventricular events
- A wave: late V diastole
  - Reflection of atrial contraction
- X descent: early V systole
  - Usually predominant in normal individuals
- V wave: late V systole
  - Determined by atrial compliance
- Y descent: early V diastole
  - Determined by ventricular compliance
Which hemodynamic parameter is correlated with cardiorenal syndrome?

- a) Cardiac index
- b) Pulmonary capillary wedge pressure
- c) Right atrial pressure
- d) Pulmonary arterial pressure
- e) Systemic vascular resistance

CVP x 2 = PCW
PCW x 2 = PA sys
This waveform is most consistent with:

a) Pulmonary artery pressure
b) Right ventricular pressure
c) Pulmonary capillary pressure
d) Left atrial pressure
Pulmonary capillary wedge pressure

Things to consider

- Do not force balloon inflation to obtain PCW
- If truly “wedged”, blood no longer moving so proximal pressure will equal distal pressure, e.g. left atrial pressure
- At diastasis, in absence of atrial or MV obstruction, PCW = LA = LVEDP (usually)
- Report A wave, V wave, and mean values
Normal 8 - 12 mmHg
Pulmonary capillary wedge pressure

Things to remember

• Thoracic, not intracardiac pressure
• Review the tracings!
• Confirm with saturation >95%
• PAD < 3 mmHg greater than LVEDP
• V wave < 2X A wave
• V wave is reflection of atrial compliance
V waves can be very dynamic
Which one is the Wedge?
The capillary wedge pressure and left ventricular end-diastolic pressure

- 472 pts with simultaneous right and left heart catheterization
- 43 without heart disease
- 429 with primarily CAD, HTN, AS
- Overall, mPCW = LVEDP
  p=0.88
- However, in 133 (28%), LVEDP > PCW by >5 mmHg
  - mPCW 13.0 ± 5.2
  - mLVEDP 20.4 ± 6.6
- 42/43 “normals” difference < 5 mmHg

Normal 25/5 mmHg
Normal 25/10/15 mmHg
Measuring Cardiac Output

• Cardiac output = Stroke volume x Heart rate
  – Ventriculography, echo, MRI, nuclear
  – Volume measurement difficult in clinical practice

• Fick principle
  – Oxygen consumption \( (V_0_2) = \text{oxygen delivery} \times \text{oxygen extraction} \)
  – Can also use other substances (e.g. green dye) or even temperature (e.g. “cold”) as the indicator
• Cardiac output = \( \frac{V_0}{[A-V\ 0_2\ difference]} \)

• \( V_0 = A-V\ 0_2\ diff \times CO \)

• \( V_0 = A-V\ 0_2\ diff \times (HR \times SV) = 3 \times 3 \times 2 = 18x \)

• \( V_0 = 125\ cc/min/m^2\ [BSA\ 2.0\ m^2] \)

• \( A-V\ 0_2\ difference = (0.99 - 0.75) \times \\
(14.0\ gm\ Hgb/dL)(1.36\ cc\ O_2/gm\ Hgb) \times \\
10\ (dL/L\ blood) \)

5.47 Liters per minute
\( (divide\ by\ BSA\ to\ get\ cardiac\ index) \)
The Fick Cardiac Output

Sources of Error

• Oxygen consumption measurement (6% error)
  • 125 cc/min/m2 vs 110 cc/min/m2 (>70 yrs)
  • $126 \pm 26$ cc/min/m2 (Dehmer GJ, Clin Card 1982;5:436)
  • Half off by >10%, some off by >25% (Kendrick AH, EHJ 1988;9:337)

• Oxygen saturation (5% error)
  • Accurate generally when >40%
  • Air bubbles, heparin dilution, site “contamination”
  • Less error with large A-V 02 differences, e.g. low output

• Total error 10% (Visscher MB, J Appl Phys 1953;5:635)
Thermodilution Cardiac Output

\[ CO = \frac{V(T_B - T_I) \times K_1 \times K_2}{\int T_B(t) \, dt} \]

Modified Stewart-Hamilton equation

\( V \) = volume of injectate
\( T_B \) = initial blood temp (C°)
\( T_I \) = initial injectate temp (C°)
\( K_1 \) = density constant
\( K_2 \) = computation constant

CO 4.95 L/min

CO 3.46 L/min
Thermodilution Cardiac Output

Sources of Error

- Tricuspid regurgitation
  - \( \text{TD CO} = 0.8 \times \text{Fick CO} \) \((\text{Hamilton MA, et al. Am J Card 1989;64:945})\)

- Other sources of warming
  - PA blood temperature changes with respiration and cardiac cycle
  - Empirical correction factor for catheter warming

- Reproducibility of injection

- Overestimates in low flow states (by as much as 35% when CO <2.5 L/min)
Cardiac Output

Thermodilution vs Fick

Difference from Fick's method (%)

Low output  Normal output  Mild-to-moderate TR  Severe TR

Courtesy of Michael Mathier, MD
Vascular Resistance

• Poiseuille equation \( R = \Delta P/Q = 8\pi l / \pi r^4 \)
• Modeled upon continuous flow (not pulsatile, e.g. impedance)
• Dependent upon length, viscosity, cross-sectional area of vessel
• Primarily at level of arterioles (60%) but other contributions (arteries 10%, capillaries 15%, small veins 15%)
• Influenced by autonomic regulation and local metabolic factors
Calculating Vascular Resistance

- Mean BP = Output x Resistance \((\text{Ohm’s law, } V=IR)\)

- SVR = \(\frac{\text{mean BP} - \text{mean RA}}{\text{Qs}}\)
- PVR = \(\frac{\text{mean PA} - \text{mean PCW}}{\text{Qp}}\)
- Generally assume \(\text{CO} = \text{Qs} = \text{Qp}\)

- Normal values
  - SVR = 800 – 1200 dynes-sec-cm-5
  - PVR = 80 – 120 dynes-sec-cm-5
  - Wood units \((x\ 80)\) = dynes-sec-cm-5
  - SVRI or PVRI obtained by dividing by CI
Hemodynamic Management

Maximizing CO, decreasing filling pressures...yet maintaining BP

\[ CO = \frac{MAP - RA}{SVR} \]

\[ MAP - RA = SVR \times CO \]

Very Bad
Which one of the following provides a measure of load independent RV function:

a) RVEF
b) TAPSE
c) CVP
d) RV width/length ratio
e) RVSWI
RVSWI = (mPAP – CVP) x [CI/HR] x 0.0136 gm-m/m²

LVSWI = (MAP - PCWP) x [CI/HR] x 0.0136 gm-m/m²

(0.0136 converts mmHg/ml to gm-m)

<table>
<thead>
<tr>
<th>Metric</th>
<th>Symbol</th>
<th>Range</th>
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<tbody>
<tr>
<td>Stroke Volume</td>
<td>SV</td>
<td>60 – 130 mL</td>
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<tr>
<td>Stroke Volume Index</td>
<td>SVI</td>
<td>30 – 65 mL/beat/m²</td>
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<tr>
<td>Cardiac Index</td>
<td>CI</td>
<td>2.5 – 4.2 L/min/m²</td>
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<tr>
<td>RV Stroke Work Index</td>
<td>RVSWI</td>
<td>5 – 10 g-m/beat/m²</td>
</tr>
<tr>
<td>LV Stroke Work Index</td>
<td>LVSWI</td>
<td>45 – 60 g-m/beat/m²</td>
</tr>
<tr>
<td>Pulmonary Vascular Resistance</td>
<td>PVR</td>
<td>20 – 120 dynes x sec x cm⁻⁵</td>
</tr>
<tr>
<td>Systemic Vascular Resistance</td>
<td>SVR</td>
<td>800 – 1500 dynes x sec x cm⁻⁵</td>
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## Assessing RV performance

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Desirable Value</th>
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<tbody>
<tr>
<td>RVSWI</td>
<td>&gt; 300-600 mmHg-ml/m²</td>
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<tr>
<td>CVP</td>
<td>&lt; 15 mmHg; 5 mmHg &lt; PCWP</td>
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<tr>
<td>Presence of TR</td>
<td>Minimal to Moderate</td>
</tr>
<tr>
<td>PVR and TPG</td>
<td>PVR &lt; 4 WU; TPG &lt; 15 mmHg</td>
</tr>
<tr>
<td>RV Size</td>
<td>RVEDV &lt; 200 mL; RVESV &lt; 177 mL</td>
</tr>
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*Slaughter MS, et al. JHLT 2010;29:S1-S39*
LVADs and post RV failure

*RV Stroke Work Index (RVSWI)*

207 pts from 1991-2002
Age 55±11.1, 14% female
All on inotropes, 76% on IABP

mPA 37±8.6, CVP 18±6.0, CI 1.85±0.52

Cr 1.75±0.90; Tbili 1.4 (0.8, 3.1), AST 45 (22, 200)

Duration of inotropic support associated with:
1) RVSWI, older age, nonischemic
2) Associated with poor pre-Tx survival

\[
\text{RVSWI (mmHg-ml/m2)} = (\text{mPAP} - \text{mCVP}) \times \text{SI}
\]

Obese man with progressive exertional dyspnea but no history of HF. No PH. BNP normal.
What next?

a) PE protocol chest CT.
b) Pulmonary function tests.
c) Nuclear stress test.
d) Holter monitor.
e) Exercise hemodynamics.
Exercise hemodynamics to unmask HFpEF

55 pts referred for dyspnea

Normal BNP
No CAD
EF > 50%

Normal resting hemodynamics
Mean PA < 25 mmHg
Mean PCW < 15 mmHg

HFpEF
Exercise PCW > 25 mmHg
Exercise mPA > 30 mmHg

Supine bicycle or arm adduction.
Femoral or internal jugular/radial.

Resting but not exertional PA pressures are age independent

Increases in PA and PCW in normal individuals varies substantially

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<tr>
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<th>Rest</th>
<th>Slight</th>
<th>Maximal</th>
</tr>
</thead>
<tbody>
<tr>
<td>$\dot{P}_{pa}$ mmHg</td>
<td>13.8 ± 3.1</td>
<td>20.8 ± 4.0</td>
<td>25.6 ± 5.6</td>
</tr>
<tr>
<td>$P_{paw}$ mmHg</td>
<td>5.9 ± 2.8</td>
<td>9.1 ± 4.2</td>
<td>14.9 ± 7.9</td>
</tr>
<tr>
<td>Heart rate min⁻¹</td>
<td>82 ± 16</td>
<td>103 ± 14</td>
<td>170 ± 14</td>
</tr>
<tr>
<td>Cardiac output L·min⁻¹</td>
<td>7.4 ± 2.2</td>
<td>14.9 ± 3.9</td>
<td>20.0 ± 3.8</td>
</tr>
</tbody>
</table>

Slight: HR 100-110 bpm, WR 50 watts, V02 1000 cc/min
Maximal: HR 160-170 bpm, WR 150-200 watts, V02 2400 cc/min

The mean PAp rises linearly with increase in CO

PH suspected by history and exam

PH at catheterization

1) LVEDP >18 mmHg?
2) PCW > 15 mmHg?
3) LAP >15 mmHg?

no

1) Exercise
2) Leg lift
3) Volume challenge
4) Nitric oxide

<18 mmHg
PAH

18-24 mmHg
Intermediate Group

>24 mmHg
PH from LHD

yes

PH from LHD

Consider

Vasodilator challenge
Invasive Hemodynamics

Take Home messages

• Phlebostatic axis and the zero reference
  – How was it determined
• Values versus waveforms
  – Patterns not apparent from values
  – Respiratory variability
• Usually obtained at rest and are not static
• Understand how cardiac output and resistance are obtained
• Are you sure it’s the wedge?
Which patient should have an endomyocardial biopsy?

a) 70 year old man with EF 25%, EDD 7.0 cm, EKG qV1-V4, NYHA III
b) 35 y/o AA man with EF 35%, EDD 6.5 cm, EKG LVH, Brother with heart transplant
c) 55 y/o woman with EF 20%, EDD 4.5 cm, EKG low volts, NSVT, NYHA IV, BP 80/65
d) 25 y/o man with EF 55%, TnI 5.0, EKG diffuse ST elevation, pleuritic chest pain
Endomyocardial Biopsy

Few Class I indications

• New onset HF < 2 wks duration
  – normal sized or dilated LV
  – hemodynamic compromise

• New onset HF 2 – 12 wks duration
  – new ventricular arrhythmias
  – advanced AV block
  – or failure to respond to usual care within 1 - 2 wks

ACC/AHA EMBx guidelines 2007
Endomyocardial Biopsy

When To Suspect a Specific Diagnosis

Class II (ACC/AHA Biopsy guidelines)

- Failure to respond to usual therapy

- Search for myocarditis or infiltrative disorders
  - Rash, eosinophilia
  - Severe HF, normal EF, no HTN
  - Systemic features (e.g. hilar adenopathy)
  - Autoimmune features

- Unexplained ventricular arrhythmias

Cooper LS, Circ 2007;5:1914
Endomyocardial biopsy

Pros and Cons

• Pros
  – Tissue diagnosis
  – Hemodynamics
  – Safe in experienced hands
  – May be definitive

• Cons
  – Invasive
  – Sampling error
  – Not uniformly available
  – May not change management

Consider Endomyocardial biopsy when specific diagnosis is suspected
Summary

• Look at the Waveforms!

• Routine use of RHC is not necessary in heart failure but useful when the hemos are unclear, assessing PH, and considering advanced therapies.

• Consider hemodynamic challenge when resting hemodynamics are not diagnostic.

• Endomyocardial biopsy is primarily indicated when you suspect inflammatory or infiltrative disease.