Ventriculo-Arterial Coupling

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Presenter Disclosure Information
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Ventriculo-Arterial Coupling
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✓ Theoretical overview
✓ Assessment in experimental settings
✓ Assessment at bedside
✓ Clinical relevance and applications
✓ Conclusion
Why is VA coupling a key point in critically ill patients?

- Cardiovascular performance results from continuous interaction between ventricular and vascular systems.
- Cardiovascular system works better if ventricular pump and arterial system are perfectly matched.
- This concept may be applied in systemic as well as in pulmonary circulation.
The ventricular system can be characterized by an *elastance*: 

\[ E_{es} \text{ (mm Hg} / \text{mL)} = \text{end-systolic elastance} = \text{contractility} \]
The arterial system can also be characterized by an elastance:

\[ \text{Ea} \, (\text{mm Hg} / \text{mL}) = \text{arterial elastance} = \text{afterload} \]
VA coupling

- Intersection of elastance lines gives the working point
- **SV** and **Pes** result from this intersection

Matching between contractility and afterload

- Ventricular contractility (Ees) = driving force of the biker
- Afterload (Ea) = slope of the road

⇒ slope and driving force should be perfectly matched
Energetics

efficiency = \frac{\text{useful energy out}}{\text{total energy in}} \approx \frac{\text{SW}}{\text{Vo2}}

Ees/Ea = 2 \rightarrow \text{maximum efficiency}

Ees/Ea = 1 \rightarrow \text{maximum SW}

Ees/Ea << 1 \rightarrow \text{uncoupling}

From bench ....
1. Ventricular system: Ees

- **End-systolic points obtained from** preload reduction
- **Ees** = slope of ESPVR
- Independent of loading conditions
- = « intrinsic » contractility

2. Arterial system: Ea

- Arterial system can be modeled by an electrical circuit (Windkessel » model)
- Resistive and compliant elements can be calculated from pressure and flow waves
- Ea is precisely derived from the Windkessel model: \( Ea = \frac{Zc+R2}{ts+R2.C(1-e^{-\frac{td}{R2.C}})} \)

From bench ....

.... to bedside
1. How to assess Ees at bedside?

- Pes derived from invasive art P
- ESV derived from echocardiography
- Preload reduction is obtained from inspiratory hold maneuver of 10 cmH2O

Single beat analysis

- Isovolumic LV contraction extrapolated to determine Piso
- Piso is reported on PV graph to determine a second « fictive point »

Echocardiographic single beat method

\[ E_{Nd(\text{est})} \rightarrow E_{es(sb)} = \frac{P_d - (E_{Nd(\text{est})} \times P_s \times 0.9)}{(E_{Nd(\text{est})} \times SV)} \]

- No preload variation

2. How to assess Ea at bedside?

Ea can be approximated as:

$$\text{Ea} \approx \frac{\text{Pes}}{\text{SV}}$$

- Validation in systemic circulation
- Validation in pulmonary circulation

## Clinical relevance in ICU

<table>
<thead>
<tr>
<th></th>
<th>$\uparrow$ Ea</th>
<th>$\downarrow$ Ees</th>
</tr>
</thead>
</table>
| **Right VA uncoupling** | Pulmonary embolism  
Pulmonary HT  
High airway P  
ARDS  
Endotoxin  
Hypercapnic acidosis  
Hypoxic vasoconstriction  
... | RV infarction  
RV dysfunct° (post Tx)  
Hypercapnic acidosis  
Hypoxemia  
Sepsis  
... |
| **Left VA uncoupling**     | Vasopressors  
Correct°of mitral regurgit°  
Aortic stenosis  
.... | Myocardial infarction  
Myocarditis  
Septic cardiomyopathy  
Toxic cardiomyopathy  
.... |

- Ghuysen A et al. Shock. 2008;29(2):197-204
- Segers P, Morimont P et al. *Am Heart J* 2002; 144(4) : 568-76
- Price L. et al. *Critical Care* 2010, 14:R169
- Guarracino F. et al. *Critical Care* 2013, 17:213
Septic shock

- Depressed LV contractility (Ees ↓) is induced by cytokines, NO, acidosis ...
- Decreased arterial elastance (Ea ↓) results from vasoplegia

Septic shock + vasopressors

- Vasopressor: $\uparrow$ Ea $\Rightarrow$ $\downarrow$ $\frac{Ees}{Ea}$

LV failure

Mismatch between depressed $E_{es}$ and high $E_a$
LV failure

Combination of inotropes and vasodilators improves VA coupling: \( \frac{Ees}{Ea} \)

In ARDS, hypercapnic acidosis resulting from protective ventilation is responsible for increased PHT (↑ Ea) and decreased RV contractility: \( \frac{Ees}{Ea} \ll 1 \)

CO2 removal decreases PHT (↓ Ea), improves RV function (↑ Ees) and restores VA coupling in an experimental model of ARDS: \( \frac{Ees}{Ea} \approx 1 \)

VA coupling and VA ECMO

- Mr B. L. 54 YO
- CAD (Cx – IVA)
- Cardiogenic shock post CABP
- ECMO VA (Femoro-Femoral)
PV loops

ECMO: 5.8 L/min
ECMO: 2.9 L/min

Ees = 0.51 mm Hg/mL

SV = 17 mL
SV = 23 mL

changes in ECMO flow ➔ changes in pre- and after- load ➔ end-systolic points ➔ Ees
# Hemodynamics

<table>
<thead>
<tr>
<th></th>
<th>DAY 1</th>
<th>DAY 3</th>
</tr>
</thead>
<tbody>
<tr>
<td>Dobutamine (µg/kg/min)</td>
<td>5</td>
<td>5 =</td>
</tr>
<tr>
<td>Norepineph (µg/kg/min)</td>
<td>5</td>
<td>15 ↑</td>
</tr>
<tr>
<td>ECMO Flow (L/min)</td>
<td>5.80</td>
<td>5.50 ≈</td>
</tr>
<tr>
<td>MAP (mm Hg)</td>
<td>71</td>
<td>89 ↑</td>
</tr>
<tr>
<td>SV (mL)</td>
<td>27</td>
<td>20 ↓</td>
</tr>
<tr>
<td>LVEF (%)</td>
<td>17</td>
<td>14 ↓</td>
</tr>
<tr>
<td>Ees (mm Hg/mL)</td>
<td>0.51</td>
<td>1.09 ↑</td>
</tr>
<tr>
<td>Ea (mm Hg/mL)</td>
<td>2.63</td>
<td>4.45 ↑↑</td>
</tr>
<tr>
<td>Ees/Ea</td>
<td>0.33</td>
<td>0.24 ↓↓↓</td>
</tr>
</tbody>
</table>

- Increased vasopressors ➞ increased Ea ➞ uncoupling ➞ decreased LVEF
- However, LV contractility improved !!!
PV loops

Day 1
Day 3

Recovery is unmasked by using VA coupling
Conclusions

- Management of critically ill patients with acutely altered hemodynamic states can benefit from assessment of VA coupling.

- Treatment oriented at normalizing Ees/Ea ratio improves cardiovascular efficiency.

- Bedside evaluation of Ees/Ea is becoming available and reliable because of the improvement in echocardiographic methods
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