Ventriculo-arterial coupling and diastolic elastance

MasterclassIC Schiermonnikoog 2015
Ventriculo-arterial coupling

• Dynamic interaction between heart and systemic circulation (modulation of compliance / stiffness / resistance) allowing the system to provide adequate CO and arterial pressures necessary for sufficient organ perfusion under different circumstances

• Optimizes cardiovascular performance and cardiac energetics
Ventriculo-arterial coupling

Cardiac contractility
End-systolic arterial pressure
Low
Normal
High

EDV
ESV
EDV
ESV
EDV
ESV
EDV
ESV
EDV
ESV
Diastole

Baseline
LVEF 70%

Systole

Norepinephrine
LVEF 40%

Veillard-Baron A. Ann Int Care 2011;1:6
Ventriculo-arterial coupling

- Ratio of arterial- to ventricular elastance (Ea/Ees)

- Ea/Ees approximating unity is optimal for the system (adequate SV with lowest possible energetic consumption)

- Ees is the slope of the end-systolic pressure-volume relationship

- Ea represents the total afterload of LV and is the capability of the arterial vessels to increase pressure when LV stroke volume increases
Effects of increased afterload in normal and failing heart
End-systolic pressure = 0.9 × systolic blood pressure

Pressure at onset of injection = diastolic blood arterial pressure

End-Diastolic pressure = 11.96 + (0.596 × E/e')

Single beat method by Chen
(Stroke volume, SBP, DBP, isovolumic contraction time and ejection time)

Chen CH. J Am Col Cardiol 2001;38:2028-2024
V-A decoupling (Ea/Ees > 1)

• *Due to decreased Ees*
  - Acute heart failure
  - Cardiac surgery
  - Acute systemic hypotension

• *Due to decreased Ea*
  - Acute systemic hypertension

Could be an additional clue what is wrong and how it should be treated
Levosimendan and V-A coupling

Bolus Levosimendan of 12 μg followed by 0.1 μg/kg/min

Ischemic cardiomyopathy

Guarracino F. Acta Anaesthesiol Scand 2007;51:1217-1224
V-A decoupling in sepsis

Ea/Ees (sb) ratio

Septic shock
Median 1.81 (IQR 1.49-2.02)

Critically ill without septic shock
Median 1.07 (IQR 0.95-1.14)

Ea usually increases due to pharmacologic vasoconstriction

Guarracino F. Crit Care 2014;18:R80
Levosimendan and myocardial depression in sepsis

<table>
<thead>
<tr>
<th></th>
<th>Levosimendan</th>
<th>Dobutamine</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Baseline</td>
<td>24 h</td>
</tr>
<tr>
<td>EDVI (ml/m²)</td>
<td>75.8 ± 23.8</td>
<td>66.2 ± 24.6**</td>
</tr>
<tr>
<td>ESVI (ml/m²)</td>
<td>46.7 ± 21.9</td>
<td>36.9 ± 19.4**</td>
</tr>
<tr>
<td>LVEF (%)</td>
<td>37.1 ± 3.0</td>
<td>45.4 ± 8.4**</td>
</tr>
</tbody>
</table>

** Morelli A. Intensive Care Med 2005;31:638-644
Ventriculo-arterial coupling in aortic arch replacement

Wegmann A. PLOS ONE 2014;9:e103588
Right ventricle

Acute Pulmonary Hypertension Chronic

Minutes

Heterometric adaption Homeometric adaption

Hypertrophy homeometric Dilatation heterometric

Increase afterload Decrease afterload

Change in Ventricular Volume ml

Time, min
A
1: Conductance catheter
2: Occlusive balloon
3: Tricuspid valve

B
End-systolic point
Ea: End-systolic pressure
Ees: End-systolic volume

Pressure (mmHg) vs. Volume (ml)
Increase in $\frac{E_a}{E_{es}}$ ratio is invariably associated with ↑ EDV

\[ E_{es} = \frac{(P_{max} - mPAP)}{Sv} \]
\[ E_a = \frac{mPAP}{Sv} \]

Normal 0.5 - 0.7

\[ \frac{E_a}{E_{es}} = \frac{(ESP/SV)}{(ESP/ESV)} = \frac{ESV}{SV} \]
RV diastolic function

- Active relaxation
- Stiffness of the right ventricle
Right ventricular relaxation in PHT

RV $\tau$ (ms)

Controle

PHT

18.3 ± 3.2 mmHg

53.3 ± 32.4 mmHg

P < 0.05

N = 25

Murch SD. Pulm Circ 2015;5:370-375
RV $\tau$ correlates with RV DP

$R = 0.93$, $P < 0.0001$

Murch SD. Pulm Circ 2015;5:370-375
Diastolic stiffness correlates with RAP and SV

Rain S. Circulation 2013;128:2016-2025
Mild PHT decreases LV relaxation

LVEDP (mmHg) vs. LVEDV (mL)

Normal

VCO

**

**

E/E' (lateral)

Control  DD  IPAH

Change in cardiac output after VCO (%)

Control  DD  IPAH

Kasner M. Am J Respir Crit Care Med 2012;186:181-189
Ea/Ees changes with different medications

<table>
<thead>
<tr>
<th>Drug</th>
<th>Ea/Ees</th>
</tr>
</thead>
<tbody>
<tr>
<td>Dobutamine</td>
<td>Decrease</td>
</tr>
<tr>
<td>Levosimendan</td>
<td>Decrease</td>
</tr>
<tr>
<td>Milrinone</td>
<td>Decrease</td>
</tr>
<tr>
<td>Norepinephrine</td>
<td>Decrease</td>
</tr>
</tbody>
</table>
Effect of fluid challenge on EA in sepsis

Explains why some septic patients increase CO without improving blood pressure after a FC

Monge García MI. Intensive Care Med 2015;41:1247-1255
Hypotension - will a fluid challenge help?

- Increased SV not always results in a higher arterial pressure (dependent on arterial tone)
- Arterial elastance (Ea) is the best integrative parameter of overall arterial system behavior
Dynamic arterial elastance
Defined by ratio PPV/SVV during single MV breath

N = 25 - MAP responder 16, MAP non-responder 9

García MIM. Crit Care 2011;15:R15
Dynamic arterial elastance

Prediction of volume expansion on MAP

\[ R^2 = 0.8292 \]
\[ y = -16,3321 + 33,4849 \times x \]
\[ P < 0.0001 \] (95% CI: 28.9285 to 40.0412)

<table>
<thead>
<tr>
<th>Parameter</th>
<th>AUC</th>
<th>95% CI</th>
</tr>
</thead>
<tbody>
<tr>
<td>( E_{a,dyn} )</td>
<td>0.986 ± 0.02</td>
<td>0.84 - 1.00</td>
</tr>
<tr>
<td>SVR</td>
<td>0.503 ± 0.12</td>
<td>0.3 - 0.71</td>
</tr>
<tr>
<td>MAP</td>
<td>0.604 ± 0.12</td>
<td>0.39 - 0.79</td>
</tr>
<tr>
<td>PP/SV</td>
<td>0.50 ± 0.12</td>
<td>0.3 - 0.7</td>
</tr>
</tbody>
</table>

García MIM. Crit Care 2011;15:R15
All fluid responders (increase CO $\geq$ 10%)

MAP responders defined by increase MAP $\geq$ 10%}

García MIM. Crit Care 2014;18:626
Predicting MAP ↑ after fluid challenge

García MIM. Crit Care 2014;18:626
Eadyn also predicts MAP ↓ after NE ↓

N = 35 - Septic shock - decrease in NE 0.04 μg/kg/min
(13 [37%] were NE responders - ↓ MAP > 15%)

< 0.90 gives 70% change of MAP ↓

<table>
<thead>
<tr>
<th></th>
<th>AUC</th>
<th>95% CI</th>
</tr>
</thead>
<tbody>
<tr>
<td>Eadyn</td>
<td>0.87</td>
<td>0.72 - 0.96</td>
</tr>
<tr>
<td>SVR</td>
<td>0.54</td>
<td>0.54 - 0.77</td>
</tr>
<tr>
<td>Compliance</td>
<td>0.61</td>
<td>0.43 - 0.77</td>
</tr>
</tbody>
</table>

Guinot PG. Crit Care 2015;19:14
Conclusions

- Estimation of the Ea/Ees ratio may provide another clue for a rational therapeutic choice
- Early right ventricular dysbalance in Ea/Ees ratio immediately results in increased EDV with septal shift and compromise of left ventricle
- In a FR patient with hypotension a $E_{a_{\text{dyn}}} > 0.8$ predicts an increase in MAP