Venous Return

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- 45+ ICU beds in a 1300 bed hospital
- Trauma center, Only center allowed to do all transplantations in adults and children
- ECMO center (last 5 mo: 20 patients)
- Admitting 2500 patients in ICU and 1000 in PACU
- 70% Mechanical Ventilation
- APACHE II: 20±9

**Research**
- Circulation: 10 PhD students
- Ventilation: 4 PhD students
- Ethics/EOL: 2 PhD students
Venous Return

- Do you measure venous return in your patients?
Cardiac Output

- Is not a physiological variable
  - Product of Stroke Volume and Heart Rate
- When cardiac function is normal this is a passive variable
  - Major determinant is the return of blood to the heart (venous return)
- Acute decreases are not well tolerated as compensation mechanisms are limited
Main functions of the heart

- Keep the right atrial pressure low
  - facilitate venous return
- Equal RV-output to LV-output
  - Frank-Starling mechanism
The Starling mechanism provides the “fine tuning” to match in-flow to out-flow

Consider a situation where the SV of the RV is 101 ml and that of the LV is 100 ml (1% difference). Heart rate is 70 /min

In 1.5 hour the total blood volume would be in the lungs!

S. Magder
Regulation of CO

- A healthy 25 year old donates one kidney to his sister
- In stable conditions CO is measured before donation
- Following complete recovery CO is measured again under stable conditions

- Is the CO following the removal of one kidney higher - similar - lower than before the donation?

PS! Don’t consider the compensation mechanisms in the remaining kidney
Distribution of cardiac output

Cardiac Output = Total Tissue Blood Flow

- Right Heart
- Lungs
- Left Heart

Venous Return (Vena Cava)

- Brain 20%
- Heart 5%
- GI 25%
- Kidneys 20%
- Muscle 20%
- Skin, etc. 10%

Cardiac Output (Aorta)
Starling's Law

CVP

Output

Output

CVP

Starling J. Physiol 1914
RAP vs CO in exercise

Cardiac output (l/min) vs Right Atrial Pressure (mmHg)

Notarius et al Am Heart J 1998
RAP vs CO in exercise
Stressed volume
Determinants of venous return

- RAP
- Vascular compliance
- Stressed volume
- Resistance to venous return
- Distribution of blood flow
5 anesthetized, hypothermic (19°C) patients
3p Thoracic arch
2p Vena caval resections

Passive drainage of the RA-catheter after cardiac arrest

Stressed volume
1290±296 ml
20.2±1.0 ml/kg

Stressed volume is 30±17% of the total blood volume
The gradient for venous return is normally very small. In this case it is only 4 mm Hg.
Restoring arterial pressure with norepinephrine improves muscle tissue oxygenation assessed by near-infrared spectroscopy in severely hypotensive septic patients


- 28 patients with septic shock
- Fluid resuscitated
- Norepinephrine to increase MAP>65 mm Hg

<table>
<thead>
<tr>
<th></th>
<th>Before norepinephrine (introduction/increase)</th>
<th>After norepinephrine (introduction/increase)</th>
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</thead>
<tbody>
<tr>
<td>SAP (mmHg)</td>
<td>86 ± 19</td>
<td>126 ± 18*</td>
</tr>
<tr>
<td>DAP (mmHg)</td>
<td>38 ± 7</td>
<td>52 ± 8*</td>
</tr>
<tr>
<td>MAP (mmHg)</td>
<td>54 ± 8</td>
<td>77 ± 9*</td>
</tr>
<tr>
<td>Heart rate (min⁻¹)</td>
<td>98 ± 25</td>
<td>101 ± 28</td>
</tr>
<tr>
<td>Temperature (°C)</td>
<td>37.5 ± 1.4</td>
<td>37.5 ± 1.3</td>
</tr>
<tr>
<td>CI (L/min/m²)</td>
<td>3.1 ± 1.0</td>
<td>3.6 ± 1.3*</td>
</tr>
<tr>
<td>GEDVI (mL/m²)</td>
<td>687 ± 117</td>
<td>730 ± 156*</td>
</tr>
<tr>
<td>EVLWI (mL/kg)</td>
<td>13 ± 9</td>
<td>13 ± 7</td>
</tr>
<tr>
<td>SaO₂ (%)</td>
<td>94 ± 5</td>
<td>93 ± 4</td>
</tr>
<tr>
<td>PaO₂ (mmHg)</td>
<td>122 ± 73</td>
<td>115 ± 63</td>
</tr>
<tr>
<td>PaCO₂ (mmHg)</td>
<td>40 ± 16</td>
<td>40 ± 15</td>
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<tr>
<td>pH</td>
<td>7.34 ± 0.10</td>
<td>7.33 ± 0.10</td>
</tr>
<tr>
<td>ScvO₂ (%)</td>
<td>68 ± 9</td>
<td>72 ± 7*</td>
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</tbody>
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Rare cause of shock
17 yr old, following liver transplantation
Admitted in other hospital with shock: hypovolemic?
Not responding to therapy
Massive amounts of vasopressors
Diagnosis: obstructive shock due to compression of the inferior vena cava

Rapid discontinuation of the vasopressors
Uneventful recovery
Importance of venous return

- Cardiac output is coupled to oxygen demand
- The primary mission of the heart is to facilitate venous return by keeping RAP low to match LV-output to the demand for oxygen delivery
- The determinant of venous return is the stressed volume
  - Increase in unstressed volume decreases CO
  - Increase in the resistance to venous return limit CO