Basic Cardiopulmonary Interactions

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No Disclosures
Cardiopulmonary interactions

We got you something.
I love it! I’m going to share it with EVERYONE!
Overview of heart-lung interactions

- Individual structural & dynamic properties
- Anatomically & physiologically interconnected
- Occupy the same physical space, therefore *mechanical* interactions
- Interactions are amplified in disease
The ubiquitous & pervasive nature of heart-lung interactions

Fetal life, postnatal transition

Normal, healthy activity

Critical Illness

Cardiopulmonary Interactions
Outline of Talk #1

• Review basic principles of respiratory function
• Review basic principles of cardiac function
• Discuss the effects of respiration on CV function under normal circumstances
  – Spontaneous breathing
  – Positive pressure ventilation
Reference literature on CP interactions

**Cardiopulmonary interactions in healthy children and children after simple cardiac surgery: the effects of positive and negative pressure ventilation.**

**Cardiovascular effects of mechanical ventilation.**
Shekerdemian L, Bohn D.
Arch Dis Child. 1999 May;80(5):475-80

**Cardiopulmonary Interactions.**
Bronicki RA, Penny DJ, Anas NG, Fuhrman B.

**Cardiopulmonary Interactions in Children with Heart Failure.**
Bronicki RA.

**Heart-lung interactions during mechanical ventilation: the basics.**
Mahmood SS, Pinsky MR.
Ann Transl Med. 2018 Sep;6(18):349.
Breathing
Inhalation

During inhalation (active):

- Diaphragm descends
- Rib cage expands
- The lungs expand
- The pressure in the lungs decreases (Boyle’s Law)
- Air flows into the lungs towards lower pressure
Exhalation

During exhalation (passive):

- Lung volume decreases
- Pressure within the lungs increases
- Air flows from the higher pressure in the lungs to the outside
Basic Cardiopulmonary Interactions

Negative Pressure versus Positive Pressure Breathing

- $P_{ao}$ - atmospheric pressure
- $P_A$ - airway pressure
- $P_{pl}$ - pleural (intrathoracic) pressure
- $P_L$ - transpulmonary pressure = $P_A - P_{pl}$
- ↑$P_L$ results in ↑$V_T$
Spirometry and lung capacity

Pulmonary function measures:
- Volumes
- Air flow
- Diffusion capacity
Lung compliance, tidal breath, and FRC

• Lung compliance = $\Delta V/\Delta P$
  
P - airway or alveolar pressure

• FRC- lung volume at which the lung’s recoil is balanced by the chest wall’s tendency to expand outward
Basic Cardiopulmonary Interactions

Gas exchange

• \( O_2 \) uptake and \( CO_2 \) elimination in the lungs
• Determined by:
  – Alveolar ventilation (minute ventilation - dead space ventilation)
  – Diffusing capacity across alveolar-capillary membrane
• Influenced by:
  – Ventilation-perfusion matching
  – Other factors (alveolar gas conc.; metabolic state, lung mechanics, etc.)
• ABG- \( pH/PaCO2/PaO2/HCO3^- \)
Ventilation-perfusion matching

**Venous Blood**
- $O_2 = 40$
- $CO_2 = 45$

**Inspired Air**
- $O_2 = 150$
- $CO_2 = 0$

- **V/Q = 0**
  - R-L Shunt
  - $CO_2 = 45$
  - $O_2 = 40$

- **V/Q = 1**
  - Normal
  - $CO_2 = 40$
  - $O_2 = 100$

- **V/Q = ∞**
  - Dead Space
  - $CO_2 = 0$
  - $O_2 = 150$

**Hypoxemia**

**Hypercarbia**
Ventilation-perfusion matching - West zones

↑V/Q (dead space)

↓V/Q (shunt)

Zone I
Zone II
Zone III
Cardiovascular function
Systemic $O_2$ delivery ($DO_2$) & Cardiac output (CO)

$$DO_2 = CO \times CaO_2$$

$$CO = SV \times HR$$

- Preload
- Afterload
- Contractility
- Rate
- Rhythm
Pressure-Volume relationship of the ventricle

- Stroke work
- Stroke volume
- Preload characteristics
- Contractility
- Afterload
- Ventriculo-vascular coupling (E_{es}/E_{a} ratio)

*SV= ED volume - ES volume
Pressure-volume loops: loading conditions & SV

Preload

Afterload

Contractility
Venous return and CO

- Mean systemic venous pressure ($P_{SV}$) - “pressure in the circulation if blood flow were to cease”
- $VR \propto P_{SV} - RA_p$
- $RA_p$ must be less than $P_{SV}$
- Influenced by:
  - Intrathoracic pressure
  - Circulatory volume & venous capacitance
  - Venous pathway obstruction (resistance)
  - Right heart function
  - SVR
Hydrodynamics and blood flow within a tube

Poiseuille’s Law

\[ \mathcal{F} = \frac{P_1 - P_2}{R} \]

\[ R = \frac{8 \eta L}{\pi r^4} \]

Rigid tube

Collapsible tube within a pressurized chamber

Fuhrman & Bronicki, 2016
Variables influencing venous return:

1) Intrathoracic pressure
2) Circulatory volume & venous capacitance
3) Venous pathway obstruction (resistance)
4) Right heart function
5) SVR

\[
\text{Venous return (VR)} = \frac{P_{SV} - P_{RA}}{R_{SV}}
\]
RV dysfunction and ventricular interdependence
Ventricular interdependence

- The heart sits within a confined pericardial space
- The ventricles share a septum
- A change in geometry of one ventricle affects the geometry of the other
- Diastolic & systolic interactions
- *In-series & in-parallel interactions*
Systolic ventricular interdependence

- Myofiber arrangement within and between the ventricles; shared septum
- Contractility of one ventricle contributes that of the other
- 20-40% RVp results from LV contraction
- Subject to loading conditions; intact pericardium not required

Naeije and Badagliacca. Cardiovasc Res. 2017
Effect of respiration on cardiovascular function
Intrathoracic (intrapleural) pressure & venous return:

- $\downarrow P_{\text{intrathoracic}} \rightarrow \downarrow P_{\text{RA}} \rightarrow \uparrow \text{VR}$
- $\uparrow P_{\text{intrathoracic}} \rightarrow \uparrow P_{\text{RA}} \rightarrow \downarrow \text{VR}$
  
$\uparrow R_{\text{IVC}} \rightarrow \downarrow \text{VR}$

Venous return (VR) = $\frac{P_{\text{SV}} - P_{\text{RA}}}{R_{\text{SV}}}$
Cardiac effects of changing intrapleural pressure

↓ intrapleural pressure  
(Mueller maneuver)
- ↑ Venous return
- ↑ RV filling
- ↑ LV afterload
- ↓ LV stroke volume
- ↑ LV wall stress
- Pulmonary edema

↑ intrapleural pressure  
(Valsalva maneuver)
- ↑ Atrial pressures
- ↓ Venous return
- ↓ RV filling
- ↑ RV afterload (PVR)
- ↓ LV afterload
- ↓ LV wall stress
Lung volume-PVR relationship

- Mechanical effects of lung overdistension or atelectasis
- Hypoxic vasoconstriction
- Mainly impacts the RV

Fuhrman & Bronicki, 2016
Spontaneous breathing and the right heart

Normal circulation:

- Modest reduction in intrathoracic pressure (-1→-5 cm H2O)
- Diaphragmatic descent:
  - Increased lung volumes
  - Increased intraabdominal pressure (drives venous return to the heart)
- Increased venous return (RV preload)

  \[
  \text{Increased RV stroke volume}
  \]

  “Respiratory pump”
Spontaneous breathing and the left heart

**Normal LV function:**

- Less impact with modest reduction in intrathoracic pressure (\(-1 \rightarrow -5 \text{ cm H}_2\text{O}\))
- However, large negative reduction (to \(-30 \text{ cm H}_2\text{O}\)) → acute LV dysfunction, pulmonary edema
- Increased intrathoracic blood volume
Effects of respiration on the left ventricle

Normal respiration

Airway obstruction (stridor)

expiration
inspiration
expiration
inspiration
PEEP and oxygen delivery

Granton, Can Respir J 1996
Positive pressure ventilation - impact on cardiac output

Physiological studies of the effects of intermittent positive pressure breathing on cardiac output in man.  
André Courmand, Hurley L. Motley, Lars Werko, and Dickinson W. Richards, Jr.

From the Department of Medicine, Columbia University, and the Chest and Medical Services of the Columbia University Division, Bellevue Hospital, New York, New York

Types of mask pressure curves produced by respirators.

Effect of three types of intermittent positive pressure breathing, as differentiated by the shape of the mask pressure curve, on cardiac output.

<table>
<thead>
<tr>
<th>Type of mask pressure</th>
<th>I</th>
<th>II</th>
<th>III</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mean mask pressure (mmHg)</td>
<td>7.0</td>
<td>10.6</td>
<td>5.7</td>
</tr>
<tr>
<td>Cardiac output</td>
<td>5.9</td>
<td>5.8</td>
<td>6.3</td>
</tr>
<tr>
<td>Liters per min per cent change</td>
<td>-14.5</td>
<td>-16.5</td>
<td>+6.0</td>
</tr>
</tbody>
</table>

Courmand et al., Am J Physiol 1948:152:162
# Positive pressure ventilation - loading conditions

<table>
<thead>
<tr>
<th>Afterload</th>
<th>Preload</th>
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</thead>
<tbody>
<tr>
<td>Pulmonary ventricle / circulation</td>
<td>Elevated</td>
</tr>
<tr>
<td></td>
<td>Effect:</td>
</tr>
<tr>
<td></td>
<td>↑ RVEDp</td>
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<tr>
<td></td>
<td>↑ RVp</td>
</tr>
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<td></td>
<td>↓ Antegrade PBF</td>
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<tr>
<td></td>
<td>↑ PR and/or TR</td>
</tr>
<tr>
<td>Systemic ventricle / circulation</td>
<td>Reduced</td>
</tr>
<tr>
<td></td>
<td>Effect:</td>
</tr>
<tr>
<td></td>
<td>↓ LVEDp</td>
</tr>
<tr>
<td></td>
<td>↓ LAp</td>
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<tr>
<td></td>
<td>↓ Pulmonary edema</td>
</tr>
<tr>
<td></td>
<td>↑ Increase cardiac output</td>
</tr>
</tbody>
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Positive pressure ventilation- impact on hemodynamics

**Preload-dependent condition** - VR, PVR effects dominate

**Afterload-dependent condition** - LV afterload effects dominate

Heart-lung interactions

**Autonomic tone:**
- ↑ lung volume → decreased HR
- ↑ lung volume → ↑ SVR

**Acid-base balance:**
- ↓pH, ↑pCO2 → ↑PVR
- ↓pH → ↓ myocardial dysfunction

**Neurologic function:**
- hypoventilation → cor pulmonale
- muscular → restrictive lung dz
- PNI → atelectasis, ↓ VR (Fontan)

**Renal function:**
- metabolic, electrolyte disturbance
- fluid retention → CHF, restrictive lung dz
Congenital heart diseases

- Cyanotic congenital heart disease
- Intracardiac shunts (L→R; R→L)
- Outflow obstruction
- Valvar diseases
- Myocardial dysfunction; pulmonary vascular disease

Add:
- Chronic lung disease, sepsis, nutrition insufficiency, other organ system failure
- Associated anomalies: airway, lung, chest wall, spine, muscular, etc.
Cardiovascular disease and respiratory function

Simulation scenarios:

1. HF with \( \uparrow \text{LA}_p \) and altered lung compliance
2. Intracardiac shunts (R→L)
3. Single ventricle physiology