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M1 - Cardiovascular / Respiratory, Fall 2007

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Pulmonary Blood Flow

Thomas Sisson, M.D.
Objectives

- The student will know the structure, function, distribution and control of pulmonary blood supply
  - Compare pulmonary and bronchial circulation
  - Compare and contrast pulmonary and systemic circulation
  - Describe and explain the effects of cardiac output and lung volume on pulmonary vascular resistance
  - Describe the effects of hypoxia on pulmonary vascular resistance
  - Describe the effects of gravity of pulmonary blood flow
  - Explain Starling’s equation
  - Describe the mechanisms of pulmonary edema
Two Circulations in the Lung

• Pulmonary Circulation.
  – Arises from Right Ventricle.
  – Receives 100% of blood flow.

• Bronchial Circulation.
  – Arises from the aorta.
  – Part of systemic circulation.
  – Receives about 2% of left ventricular output.
Bronchial Circulation

Image of bronchopulmonary anastomosis removed
Pulmonary Circulation

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Source: Undetermined
Pulmonary Circulation

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Pulmonary Circulation

- In series with the systemic circulation.

- Receives 100% of cardiac output (3.5L/min/m²).

- RBC travels through lung in 4-5 seconds.

- 280 billion capillaries, supplying 300 million alveoli.
  - Surface area for gas exchange = 50 – 100 m²
Alveolar Architecture

Source: Undetermined
Functional Anatomy of the Pulmonary Circulation

• Thin walled vessels at all levels.

• Pulmonary arteries have far less smooth muscle in the wall than systemic arteries.

• Consequences of this anatomy - the vessels are:
  – Distensible.
  – Compressible.
Pulmonary Circulation Pressures

- Right Atrium: P = 2
- Left Atrium: P = 5
- Right Ventricle: P = 25/0
- Left Ventricle: P 120/0
- Systemic Artery: P = 120/80, Mean = 100
- Pulmonary Artery: P = 25/8, Mean = 15

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Pulmonary Vascular Resistance

Vascular Resistance = \( \frac{\text{input pressure} - \text{output pressure}}{\text{blood flow}} \)

\[ PVR = k \cdot \frac{\text{mean PA pressure} - \text{left atrial pressure}}{\text{cardiac output (index)}} \]

\[ \text{mean PA pressure} - \text{left atrial pressure} = 10 \text{ mmHg} \]
\[ \text{mean aorta pressure} - \text{right atrial pressure} = 98 \text{ mmHg} \]

Therefore PVR is 1/10 of SVR
Vascular Resistance is Evenly Distributed in the Pulmonary Circulation

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Reasons Why Pressures Are Different in Pulmonary and Systemic Circulations?

- Gravity and Distance:
  - Distance above or below the heart adds to, or subtracts from, both arterial and venous pressure
  - Distance between Apex and Base

<table>
<thead>
<tr>
<th>Systemic</th>
<th>100 mmHg</th>
</tr>
</thead>
<tbody>
<tr>
<td>Aorta</td>
<td></td>
</tr>
<tr>
<td>Head</td>
<td>50 mmHg</td>
</tr>
<tr>
<td>Feet</td>
<td>180 mmHg</td>
</tr>
<tr>
<td>Pulmonary</td>
<td></td>
</tr>
<tr>
<td>Main PA</td>
<td>15 mmHg</td>
</tr>
<tr>
<td>Apex</td>
<td>2 mmHg</td>
</tr>
<tr>
<td>Base</td>
<td>25 mmHg</td>
</tr>
</tbody>
</table>
Control of regional perfusion in the systemic circulation:
- Large pressure head allows alterations in local vascular resistance to redirect blood flow to areas of increased demand (e.g. to muscles during exercise).
- Pulmonary circulation is all performing the same job, no need to redirect flow (exception occurs during hypoxemia).

Consequences of pressure differences:
- Left ventricle work load is much greater than right ventricle
- Differences in wall thickness indicates differences in work load.
Influences on Pulmonary Vascular Resistance

Pulmonary vessels have:
- Little vascular smooth muscle.
- Low intravascular pressure.
- High distensibility and compressibility.

Vessel diameter influenced by extravascular forces:
- Gravity
- Body position
- Lung volume
- Alveolar pressures/intrapleural pressures
- Intravascular pressures
Influences of Pulmonary Vascular Resistance

• Transmural pressure = Pressure Inside – Pressure Outside.
  – Increased transmural pressure-increases vessel diameter.
  – Decreased transmural pressure-decreased vessel diameter (increase in PVR).
  – Negative transmural pressure-vessel collapse.

• Different effects of lung volume on alveolar and extraalveolar vessels.

$P_i$ $P_{outside}$
Effect of Transmural Pressure on Pulmonary Vessels During Inspiration

During Inspiration

Extraalveolar Space

Alveolar Space

Alveoli

Blood Vessel Elongates and Narrows

↑ Resistance

Negative Pressure

Blood Vessel Distension

↓ Resistance

Alveoli distend

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Resistance \propto \text{Length and Resistance} \propto \frac{1}{(\text{Radius})^4}
Effect of Lung Volume on PVR

Pulmonary vascular resistance (mm Hg/mL/min)

Lung volume (L)

RV  FRC  TLC

Total

Alveolar

Extraalveolar

Pulmonary Vascular Resistance During Exercise

- During exercise cardiac output increases (e.g. 5-fold), but with little change in mean pulmonary artery pressure
  - How is this possible?

  \[
  \Delta \text{Pressure} = \text{Flow} \times \text{Resistance}
  \]

  \[
  \text{Vascular Resistance} = \frac{\text{input pressure} - \text{output pressure}}{\text{blood flow}}
  \]

- \(\Delta\text{Pressure}\) = Flow \times Resistance
- If pressure does not change, then PVR must decrease with increased blood flow
  - Passive effect (seen in isolated lung prep)
    - Recruitment: Opening of previously collapsed capillaries
    - Distensibility: Increase in diameter of open capillaries.
Recruitment and Distention in Response to Increased Pulmonary Artery Pressure

## Control of Pulmonary Vascular Resistance

- **Passive Influences on PVR:**

<table>
<thead>
<tr>
<th>Influence</th>
<th>Effect on PVR</th>
<th>Mechanism</th>
</tr>
</thead>
<tbody>
<tr>
<td>↑ Lung Volume (above FRC)</td>
<td>Increase</td>
<td>Lengthening and Compression</td>
</tr>
<tr>
<td>↓ Lung Volume (below FRC)</td>
<td>Increase</td>
<td>Compression of Extraalveolar Vessels</td>
</tr>
<tr>
<td>↑ Flow, ↑Pressure</td>
<td>Decrease</td>
<td>Recruitment and Distension</td>
</tr>
<tr>
<td>Gravity</td>
<td>Decrease in Dependent Regions</td>
<td>Recruitment and Distension</td>
</tr>
<tr>
<td>↑ Interstitial Pressure</td>
<td>Increase</td>
<td>Compression</td>
</tr>
<tr>
<td>Positive Pressure Ventilation</td>
<td>Increase</td>
<td>Compression and Derecruitment</td>
</tr>
</tbody>
</table>
Regional Pulmonary Blood Flow Depends Upon Position Relative to the Heart

<table>
<thead>
<tr>
<th>Source: Undetermined</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Main PA</strong></td>
</tr>
<tr>
<td><strong>Apex</strong></td>
</tr>
<tr>
<td><strong>Base</strong></td>
</tr>
</tbody>
</table>
Gravity, Alveolar Pressure and Blood Flow

- Pressure in the pulmonary arterioles depends on both mean pulmonary artery pressure and the vertical position of the vessel in the chest, relative to the heart.

- Driving pressure (gradient) for perfusion is different in the 3 lung zones:
  - Flow in zone 1 may be absent because there is inadequate pressure to overcome alveolar pressure.
  - Flow in zone 3 is continuous and driven by the pressure in the pulmonary arteriole – pulmonary venous pressure.
  - Flow in zone 2 may be pulsatile and driven by the pressure in the pulmonary arteriole – alveolar pressure (collapsing the capillaries).
Gravity, Alveolar Pressure, and Blood Flow

Typically no zone 1 in normal healthy person

Large zone 1 in positive pressure ventilation + PEEP

Arterial Pressure, $P_a$

Venous Pressure, $P_v$

Alveolar Dead Space

Zone 1 $P_a > P_a > P_v$

Zone 2 $P_a > P_a > P_v$

Zone 3 $P_a > P_v > P_a$

Blood Flow

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Gravity Influences Pressure

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### Control of Pulmonary Vascular Resistance

**Active Influences on PVR:**

<table>
<thead>
<tr>
<th>Increase</th>
<th>Decrease</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sympathetic Innervation</td>
<td>Parasympathetic Innervation</td>
</tr>
<tr>
<td>$\alpha$-Adrenergic agonists</td>
<td>Acetylcholine</td>
</tr>
<tr>
<td>Thromboxane/PGE2</td>
<td>$\beta$-Adrenergic Agents</td>
</tr>
<tr>
<td>Endothelin</td>
<td>PGE1</td>
</tr>
<tr>
<td>Angiotensin</td>
<td>Prostacycline</td>
</tr>
<tr>
<td>Histamine</td>
<td>Nitric oxide</td>
</tr>
<tr>
<td>Alveolar Hypoxemia</td>
<td>Bradykinin</td>
</tr>
</tbody>
</table>
Hypoxic Pulmonary Vasoconstriction

- Alveolar hypoxia causes active vasoconstriction at level of pre-capillary arteriole.

- Mechanism is not completely understood:
  - Response occurs locally and does not require innervation.
  - Mediators have not been identified.
  - Graded response between pO2 levels of 100 down to 20 mmHg.

- Functions to reduce the mismatching of ventilation and perfusion.

- Not a strong response due to limited muscle in pulmonary vasculature.

- General hypoxemia (high altitude or hypoventilation) can cause extensive pulmonary artery vasoconstriction.
Barrier Function of Alveolar Wall

• Capillary endothelial cells:
  – permeable to water, small molecules, ions.
  – barrier to proteins.

• Alveolar epithelial cells:
  – more effective barrier than the endothelial cells.
  – recently found to pump both salt and water from the alveolar space.
Fluid Movement Due to Osmotic Pressure

Water moves through the semi-permeable membrane down a concentration gradient to dilute the solute.
Osmotic Pressure Gradient Can Move Fluid Against Hydrostatic Pressure
**Osmotic Gradient Counteracts Hydrostatic Gradient**

- Hydrostatic pressure in the pulmonary capillary bed > hydrostatic pressure in the interstitium
  - hydrostatic pressure drives fluid from the capillaries into the pulmonary interstitium

- Osmotic pressure in the plasma > osmotic pressure in the interstitium
  - osmotic pressure normally would draw fluid from the interstitial space into the capillaries
Starling’s Equation

\[ Q = K [(P_c - P_i) - \sigma (\pi_c - \pi_i)] \]

- \( Q \) = flux out of the capillary
- \( K \) = filtration coefficient
- \( P_c \) and \( P_i \) = capillary and interstitial hydrostatic pressures
- \( \pi_c \) and \( \pi_i \) = capillary and interstitial osmotic pressures
- \( \sigma \) = reflection (sieving) coefficient
Normally Starling’s Forces Provide Efficient Protection

- Normal fluid flux from the pulmonary capillary bed is approximately 20 ml/hr.
  - recall that cardiac output through the pulmonary capillaries at rest is ~5 l/min.
  - < 0.0066% leak.

- Abnormal increase in fluid flux can result from:
  - Increased hydrostatic pressure gradient (cardiogenic pulmonary edema).
  - Decreased osmotic pressure gradient (cirrhosis, nephrotic syndrome).
  - Increased protein permeability of the capillary wall (ARDS).