ANATOMICAL CONTRASTS WITH SYSTEMIC CIRCULATION

- Thinner walled vessels; main pulmonary artery VS aorta
- Pulmonary artery branches also have thinner walls and greater internal diameter branches
- Much less vascular smooth muscle
- No highly muscular vessels corresponding to system arterioles
- Rapid subdivision along short distance
- 280 x 10⁹ pulmonary capillaries
- Pulmonary vessels offer much less R to blood flow
ANATOMICAL CONTRASTS WITH SYSTEMIC CIRCULATION

- Pulmonary vessels much more
  - Compressible
  - Distensible
- Pulmonary vessels are subject to changes in
  - Alveolar pressure
  - Intrapleural pressure
  - Thoracic pressure
  - Lower intravascular pressures
  - Factors other than vascular tone affect pul. Vasc. Res (PVR)
ANATOMICAL CONTRASTS WITH SYSTEMIC CIRCULATION

- right and left ventricles are in series
- $\dot{Q}_T$ must be equal over time; otherwise: edema
- LV feeds RV; RV feeds LV
- $\dot{Q}_T$ may vary over a few beats
- pulmonary arteries (deoxygenated blood) follow the course that parallels lung branches
- pulmonary veins (oxygenated blood) do NOT
- pulmonary capillaries are described as thin sheets; systemic caps are tubular
ANATOMICAL CONTRASTS WITH SYSTEMIC CIRCULATION

BRONCHIAL CIRCULATION

- TB tree (trachea to respiratory bronchioles) is supplied with oxygenated blood fed from the systemic circulation
- 1% to 2% of $Q_T$ flows into bronchial circulation
- $\frac{1}{2}$ of bronchial venous blood enters pulmonary veins (normal anatomical shunt)
- $\frac{1}{2}$ of bronchial venous blood enters systemic venous blood (IVC).
ANATOMICAL CONTRASTS WITH SYSTEMIC CIRCULATION

PULMONARY VASCULAR RESISTANCE (PVR)

- PVR cannot be directly measured — it is calculated
  - Poiseuille’s law:
    \[ R = \frac{P_1 - P_2}{\dot{Q}} \]
  - \( PVR = \frac{PAP - PCWP}{C.O.} \)

\[ PVR = \frac{12 \text{ torr} - 5 \text{ torr}}{5 \text{ L/min}} = 1.4 \text{ torr/L/min} \]
ANATOMICAL CONTRASTS WITH SYSTEMIC CIRCULATION

SYSTEMIC VASCULAR RESISTANCE (SVR)

\[ SVR = \frac{MAP - CVP}{C.O.} \]

\[ SVR = \frac{95 \text{ torr} - 5 \text{ torr}}{5 \text{ L/min}} = 18 \text{ torr/L/min} \]

\[ \frac{SVR}{PVR} = \frac{18}{2} = \frac{9}{1} \]
ANATOMICAL CONTRASTS WITH SYSTEMIC CIRCULATION

- Pulmonary circulation has higher pulse pressure in relation to mean pulmonary vascular pressure than systemic circulation.
- Therefore, pulmonary vascular blood flow is more pulsatile.
- Systemic circulation has lower pulse pressure in relation to mean systemic vascular pressure than pulmonary circulation.
- Therefore, pulmonary vascular blood flow is more continuous.
ANATOMICAL CONTRASTS WITH SYSTEMIC CIRCULATION

- Pulmonary circulation blood volume = ~ 500 ml (10% of total circulating blood volume)
- 75 to 100 ml in pulmonary capillaries;
  ~ 200 ml in arteries and arterioles;
  ~ 200 ml in veins and venules
- RV stroke volume = 70 ml to 90 ml
- Plenty time (0.75 sec) for gas exchange
- Pulmonary vasculature serves as a reservoir for left atrium
  - If venous return to RV increases suddenly,
  - LV filling does not change for 2 or 3 beats
ANATOMICAL CONTRASTS WITH SYSTEMIC CIRCULATION

- Low PVR caused by
  - Thinner vessel walls
  - Less vascular smooth muscle
  - More distensible vessels
ANATOMICAL CONTRASTS WITH SYSTEMIC CIRCULATION

DISTRIBUTION OF PVR

- 3 major components of the pulmonary vasculature
  - Arteries and arterioles
  - Capillaries
  - Veins and venules

  1/3 PVR in arteries and arterioles
  1/3 PVR in pulmonary capillaries
  1/3 PVR in veins and venules

  - Systemically, 70% SVR is in highly muscular arterioles
CONSEQUENCES OF DIFFERENCES IN PRESSURES BETWEEN SYSTEMIC AND PULMONARY CIRCULATION

- LV must pump blood at a higher pressure to overcome higher SVR
  - Blood to pump over a longer distance
- RV must pump blood at a lower pressure to overcome lower PVR
  - Blood to pump over a shorter distance
- LV must meet varying demands for blood all over the body; different blood flow needs to different systems
CONSEQUENCES OF DIFFERENCES IN PRESSURES BETWEEN SYSTEMIC AND PULMONARY CIRCULATION

For example, exercise:
- Blood flow diverted from gut to skeletal muscles to aid in thermoregulation, oxygenation, and waste product removal
- In pulmonary vasculature low pressure occurs because redistribution of R C.O. is not a factor
- Lower pulmonary vascular pressure because of
  — recruitment
  — distention
  not caused by (1) pulmonary vascular muscle tone, (2) neural mechanism, (3) humoral agents
CONSEQUENCES OF DIFFERENCES IN PRESSURES BETWEEN SYSTEMIC AND PULMONARY CIRCULATION

- LV workload > RV workload
  - Stroke work = SV x mean arterial pressure
  - RV stroke work = SV x PAP (~ 15 torr)
  - LV stroke work = SV x MAP (100 torr)
- Metabolic demand of LV > RV
- LV > muscle mass than RV
CONSEQUENCES OF DIFFERENCES IN PRESSURES BETWEEN SYSTEMIC AND PULMONARY CIRCULATION

● Extravascular effects on PVR (passive factors)
  – Body position
  – Gravity
  – Alveolar pressure
  – Intrapleural pressure
  – Intravascular pressure
  – RV output

All affect PVR without altering tone of pulmonary vascular, small muscle
CONSEQUENCES OF DIFFERENCES IN PRESSURES BETWEEN SYSTEMIC AND PULMONARY CIRCULATION

TRANSMURAL PRESSURE GRADIENT

- Determine diameter (internal) of vessels
- Transmural $P = \text{intravascular } P - \text{intrapleural } P$
  - $\uparrow$ transmural $P = \uparrow$ intravas. $P$ and/or $\downarrow$ intrapleural $P$
    - $= \uparrow$ vessel diameter
    - $= \downarrow$ PVR
    - $= \uparrow$ blood flow
  - $\downarrow$ transmural $P = \downarrow$ intravas. $P$ and/or $\uparrow$ intrapleural $P$
    - $= \downarrow$ vessel diameter
    - $= \uparrow$ PVR
    - $= \downarrow$ blood flow
- Negative transmural $P$ may indicate vessel collapse
Because $\Delta P$ across the pulmonary vasculature remains relatively constant ($K$) despite increases in right ventricular C.O.,

$$R = \frac{\Delta P}{\dot{V}} = \frac{\Delta P}{\dot{Q}}$$

$$(\dot{V} = \dot{Q})$$

$$\Delta P = \dot{V} \times R = \dot{Q} \times R$$

C.O.

$\Delta P = K$, therefore,

$$K = \uparrow \dot{V} \times R \downarrow$$

RECRUITMENT & DISTENTION
RECRUITMENT & DISTENTION

- R must decrease as C.O. or \( \dot{Q} \) increases to maintain a constant \( \Delta P \)
  
  Caused by:
  - recruitment
  - distention

- The \( \downarrow R \) is passively caused by:
  - No vasodilatation
  - No small muscle relaxation
  - No humoral agents
RECRUITMENT & DISTENTION

- Not all pulmonary capillaries perfused
- As RV C.O. ↑, parallel vascular pathways open and R↓ = RECRUITMENT
- ↓ R C.O. = derecruitment
- As RV C.O. ↑ further, ↑ intravascular P = ↑ TMP
- ↓ R = DISTENTION
HYPOXIC PULMONARY VASOCONSTRICTION RESPONSE

- \( \downarrow PAO_2 \) cause pulmonary vasoconstriction
  - At precapillary vessels
  - Local (regional) response
  - Extrinsic neural input unnecessary
    1. Hypoxia directly stimulates vascular smooth muscle cells
    2. Release of vasoactive substances
      - prostaglandins
      - catecholamines
    - Diverts blood flow to better ventilated areas
    - Regional VS. whole lung
      • High altitude
      • Alveolar hypoventilation
Nitric oxide (NO) causes pulmonary vasodilatation

Endogenous or humoral substances:
- Bradykinin
- Acetylcholine
- Serotonin
- Histamine
- Thrombin
- Adenosine diphosphate (ADP)

Stimulates endothelial cells lining pulmonary vessels
NITRIC OXIDE AND VASODILATATION

- Stimulate endothelial cell receptors to produce constitutive – nitric oxide synthase (cNOS) (small amount) (stretching and shear stress also stimulate these receptors.) in presence of Ca^{++}

- cNOS catalyzes oxidation of L-arginine, producing release of small amount of NO
NITRIC OXIDE AND VASODILATATION

- NO stimulates pulmonary vessels to dilate
- NO highly soluble in cell membrane and diffuses instantly into vascular endothelium smooth muscle cells
- NO activates guanylate cyclase which catalyzes production of cGMP
- cGMP causes vascular smooth muscle relaxation
NITRIC OXIDE AND VASODILATATION

● SEPTIC SHOCK
  - Calcium-independent form of NOS induced in endothelial and phagocytic cells by endotoxins associated with bacterial blood infection and by various inflammatory mediators
NITRIC OXIDE AND VASODILATATION

- Calcium-independent form of NOS called inducible NOS, or iNOS
- iNOS causes continuous release NO
- Continuously released NO responsible for massive
  - Vasodilatation
  - Hypotension

- Septic shock nonresponsive to vasoconstriction
- NO given therapeutically to treat PPHN of the newborn

Associated with septic shock
Starling’s Law of the Capillaries

\[ \dot{Q}_f = K_f [(P_c - P_I) - \sigma (\pi_c - \pi_I)] \]

\( \dot{Q}_f \) = net fluid flow across capillaries
\( K_f \) = filtration coefficient (permeability characteristics of the membrane)
\( P_c \) = HP in capillaries
\( P_I \) = HP in interstitium
\( \sigma \) = reflection coefficient (ability of membrane to prevent extravasation of solutes)
\( \pi_c \) = osmotic pressure in capillaries
\( \pi_I \) = osmotic pressure in interstitium
Starling’s Law of the Capillaries

Systemic Capillary

<table>
<thead>
<tr>
<th>Arterial end</th>
<th>Venous end</th>
</tr>
</thead>
<tbody>
<tr>
<td>Capillary endothelium</td>
<td>Capillary endothelium</td>
</tr>
<tr>
<td>Intravascular space</td>
<td>Intravascular space</td>
</tr>
</tbody>
</table>

\[ Q \rightarrow \]

- CHP: 30 mm Hg
- COP: 25 mm Hg
- CHP: 10 mm Hg
- COP: 28 mm Hg
- IHP: -3 mm Hg
- IOP: 4 mm Hg
Starling’s Law of the Capillaries

Pulmonary Capillary
- Pulmonary interstitium
- Capillary endothelium
- Pulmonary vasculature

\[ Q \rightarrow \]

- CHP 7 mm Hg
- Pulmonary vasculature
- Capillary endothelium
- Pulmonary interstitium

\[-8 \text{ mm Hg}\]

- IHP

\[ Q \rightarrow \]

- COP 28 mm Hg
- IOP 14 mm Hg
Pulmonary Capillary Fluid Balance

\[ P_{\text{interstitial}} \]
\[ \pi_{\text{interstitial}} \]

**Arterial end**

**P_{\text{intravascular}}**

**Venous end**

**Hydrostatic**

**Oncotic**

**Interstitial space**

**Blood vessel**

**Lymphatic flow**