What defines vascular resistance?

If:

\[ Q = \text{flow} \]

\[ P = \text{pressure} \]

\[ R = \text{resistance} \]

Then we can say:

\[ Q = \frac{P_1 - P_2}{R} \]
Where does the most flow occur?

$P_1 = 100$ and $P_2 = 90$

Or

$P_1 = 25$ and $P_2 = 10$
Where does the most flow occur?

$P_1 = 100$ and $P_2 = 90$

Or

$P_1 = 25$ and $P_2 = 10$
Once again, if this is the relationship that defines resistance, what is the most critical determinant of resistance in the circulation?

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

Viscosity?

Vessel length?

Radius?
R = 8nl / \pi \cdot r^4

VASCULAR RADIUS
Poiseuille's Law

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

\( \eta \) = viscosity

\( L \) = vessel length

\( r \) = vessel radius

Resistance (R) per unit length

\[ \left( \frac{\text{mm Hg}}{\left( \text{mm}^3/\text{sec} \right)/\mu \text{m}} \right) \]

Vessel Radius (\( \mu \text{m} \))

Graph showing resistance to flow (R) for arterial and venous sides, with vessel radius as the variable.
(a) Series flow

**Pulmonary circuit**

**Left heart** → **Right heart**

**Systemic circuit**

**Left heart** → **Right heart**

**Lungs**

CO = 5 l/min

<table>
<thead>
<tr>
<th>Rest</th>
<th>Exercise</th>
</tr>
</thead>
<tbody>
<tr>
<td>100% = 5 l/min</td>
<td>100% = 25 l/min</td>
</tr>
</tbody>
</table>

**Brain**

13–15% = 0.65–0.75 l/min

4–6% = 0.20–0.25 l/min

20–25% = 1.00–1.25 l/min

15–20% = 0.75–1.00 l/min

3–6% = 0.15–0.30 l/min

10–15% = 0.50–0.75 l/min

**Heart**

3–4% = 0.75–1.00 l/min

4–5% = 1.00–1.25 l/min

3–5% = 0.75–1.25 l/min

2–4% = 0.50–1.00 l/min

80–85% = 20.00–21.25 l/min

**Liver and gastrointestinal tract**

20–25% = 1.00–1.25 l/min

3–5% = 0.75–1.25 l/min

2–4% = 0.50–1.00 l/min

**Kidneys**

20% = 1.00 l/min

2–4% = 0.50–1.00 l/min

**Skeletal muscle**

80–85% = 20.00–21.25 l/min

**Skin**

3–6% = 0.15–0.30 l/min

1–2% = 0.25–0.50 l/min

**Skeleton, fat, and other tissues**

10–15% = 0.50–0.75 l/min
UNIQUE CHARACTERISTICS OF THE PULMONARY CIRCULATION

THE PULMONARY CIRCULATION MUST, AT ALL TIMES, ACCEPT THE ENTIRE CARDIAC OUTPUT

THE PULMONARY CIRCULATION IS SUBSERVED BY A VENTRICLE THAT CANNOT GENERATE HIGH PRESSURE

VASCULAR RESISTANCE IN THE PULMONARY CIRCULATION IS ONE-TENTH THAT OF THE SYSTEMIC CIRCULATION

WITHIN THE PULMONARY CIRCULATION, BLOOD FLOW MUST BE DIRECTED TO WELL VENTILATED (OXYGENATED) ALVEOLI, THAT IS, VENTILATION MUST BE MATCHED WITH PERFUSION
So – what keeps resistance low when flow is increased?
What does this graph depict?
Pulmonary Hypertension:

The normal pressure in the pulmonary circulation is 25/10 mm Hg with mean pressure of 15 mm Hg.

Pulmonary hypertension is said to be present if the pulmonary mean arterial pressure exceeds 25 mm Hg at rest and/or 30 mm Hg with exercise.
Pulmonary Hypertension:

Post-capillary:
Pulmonary Hypertension:

Post-capillary:

Left ventricular failure
REMEMBER – what defines vascular resistance?.

\[ R = \frac{P_1 - P_2}{Q} \]

So, in the pulmonary circulation, if P2 increases (venous pressure), what happens to P1 (arterial pressure) if flow is constant?

It increases – so does resistance

\[ 25-10/5 = 3 \quad 35-15/5 = 4 \]
Pulmonary Hypertension:

Post-capillary:

Left ventricular failure

Mitral valve disease
Pulmonary Hypertension:

Post-capillary:

Left ventricular failure

Mitral valve disease

Aortic valve disease
Pulmonary Hypertension:

Post-capillary:

Left ventricular failure

Mitral valve disease

Aortic valve disease

Pulmonary veno-occlusive disease
Pulmonary Hypertension:

Pre-capillary:
Pulmonary Hypertension:

Pre-capillary:

Pulmonary vasculitis
Pulmonary Hypertension:

Pre-capillary:

Pulmonary vasculitis

Pulmonary embolism
Pulmonary Hypertension:

Pre-capillary:

Pulmonary vasculitis

Pulmonary embolism

HPV
VENTILATION - PERFUSION RELATIONSHIPS IN THE LUNG

IDEAL - $\dot{V}/\dot{Q} = 1$
VENTILATION - PERFUSION RELATIONSHIPS IN THE LUNG

HYPOXIC PULMONARY VASOCONSTRICTION

HYPOXIA

Q
Pulmonary Hypertension:

Pre-capillary:

Pulmonary vasculitis
Pulmonary embolism
HPV

Increased Lung Volume
The concept of alveolar and extra-alveolar blood vessels:

So what?
Here is what!

INCREASED
Primary Pulmonary Hypertension:

Occurs in sporadic and familial forms.

The familial form is an autosomal dominant disease with incomplete penetrance.

It is more common by far in females than males.

Onset is in the 30’s.

The median survival after diagnosis is measured in years (<5).
So: if the following equation defines the determinants of vascular resistance, then what could be wrong in patients with PPH?

\[
R = \frac{8nL}{\pi \cdot r^4}
\]
What could impact on vascular caliber, i.e., what mechanisms could result in decreased vascular caliber in patients with PPH?

\[ R = \frac{8nL}{\Pi \cdot r^4} \]

Vascular remodeling:
What could impact on vascular caliber, i.e., what mechanisms could result in decreased vascular caliber in patients with PPH?

\[ R = \frac{8nl}{\Pi \cdot r^4} \]

Vascular remodeling:

- decreased vascular caliber
- smooth muscle hypertrophy
- fibrosis
What could impact on vascular caliber, i.e., what mechanisms could result in decreased vascular caliber in patients with PPH?

\[ R = \frac{8nl}{\Pi \cdot r^4} \]

Vasoconstriction:
What could impact on vascular caliber, i.e., what mechanisms could result in decreased vascular caliber in patients with PPH?

\[ R = \frac{8nl}{\Pi \cdot r^4} \]

Vasoconstriction:

- increased synthesis/activity of vasoconstrictors
What could impact on vascular caliber, i.e., what mechanisms could result in decreased vascular caliber in patients with PPH?

\[ R = \frac{8nl}{\Pi \cdot r^4} \]

Vasoconstriction:

- increased synthesis/activity of vasoconstrictors
- decreased synthesis/activity of vasodilators
Were do these mediators that produce vasodilation come from?

Prostacyclin: endothelial cells
Were do these mediators that produce vasodilation come from?

Prostacyclin: endothelial cells
Nitric oxide: endothelial cells
What stimulates the endothelium to produce these mediators?
SUMMARY

The pulmonary circulation is a low resistance circuit.
SUMMARY

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Pulmonary hypertension exists when the mean pressure in that circulation is > 25 mm Hg at rest.
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The major determinant of vascular resistance in the lung, or in any other vascular bed, is vascular radius.
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Pulmonary hypertension exists when the mean pressure in that circulation is > 25 mm Hg at rest.

The major determinant of vascular resistance in the lung, or in any other vascular bed, is vascular radius.

Pulmonary hypertension can result from pre- and post-capillary defects.
The pulmonary circulation is a low resistance circuit.

Pulmonary hypertension exists when the mean pressure in that circulation is > 25 mm Hg at rest.

The major determinant of vascular resistance in the lung, or in any other vascular bed, is vascular radius.

Pulmonary hypertension can result from pre- and post-capillary defects.

The understanding of the basic physiology is essential in considering those mechanisms that regulate vascular resistance in the lung or in any other vascular bed for that matter.