What defines vascular resistance?

If:

Q = flow

P = pressure

R = resistance

Then we can say:

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

\[ P_1 = 100 \text{ and } P_2 = 90 \]

Or

\[ P_1 = 25 \text{ and } P_2 = 10 \]
Where does the most flow occur?

P₁ = 100 and P₂ = 90

Or

P₁ = 25 and P₂ = 10
Once again, if this is the relationship that defines resistance, what is the most critical determinant of resistance in the circulation?

\[ R = \frac{8n l}{\Pi r^4} \]

Viscosity?

Vessel length?

Radius?
\[ R = \frac{8nl}{\pi \cdot r^4} \]

VASCULAR RADIUS
Resistance to Flow (R)

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

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

Arterial side

Venous side

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

29 25 21 17 13 9 3 13 17 21 25 29
(a) Series flow

Pulmonary circuit

Left heart → Right heart

Systemic circuit

Lungs

Rest

CO = 5 l/min

100% = 5 l/min

Exercise

CO = 25 l/min

100% = 25 l/min

Brain

13–15% = 0.65–0.75 l/min

3–4% = 0.75–1.00 l/min

Heart

4–6% = 0.20–0.25 l/min

4–5% = 1.00–1.25 l/min

Liver and gastrointestinal tract

20–25% = 1.00–1.25 l/min

3–5% = 0.75–1.25 l/min

Kidneys

20% = 1.00 l/min

2–4% = 0.50–1.00 l/min

Skeletal muscle

15–20% = 0.75–1.00 l/min

80–85% = 20.00–21.25 l/min

Skin

3–6% = 0.15–0.30 l/min

10–15% = 0.50–0.75 l/min

Skeleton, fat, and other tissues

2–5% = 0.25–0.50 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?

The graph illustrates the relationship between pulmonary vascular resistance (in cm H_2O/L/min) and arterial or venous pressure (in cm H_2O) for both increasing arterial pressure and increasing venous pressure. As the pressure increases, the pulmonary vascular resistance decreases, indicating a hyporesistant response. The graph shows two curves, one for each type of pressure, indicating that increasing pressure leads to a decrease in resistance.
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 \( P_2 \) increases (venous pressure), what happens to \( P_1 \) (arterial pressure) if flow is constant?

It increases – so does resistance

\[
\begin{align*}
25-10/5 &= 3 \\
35-15/5 &= 4
\end{align*}
\]
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

\[ \dot{V} \]

\[ \dot{Q} \]
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?

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

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

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

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

Prostacyclin: endothelial cells
Nitric oxide: endothelial cells
What stimulates the endothelium to produce these mediators?
The pulmonary circulation is a low resistance circuit.
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 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.
SUMMARY

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.
SUMMARY

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.