What defines vascular resistance?

If:

Q = flow

P = pressure

R = resistance

Then we can say:

$$Q = P_1 - P_2 / R$$

P_1 P_2

Where does the most flow occur?

$P_1 = 100$ and $P_2 = 90$

Or

$P_1 = 25$ and $P_2 = 10$

P_1 P_2

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?

$$R = 8nl / \pi \cdot r^4$$

Viscosity?

Vessel length?

Radius?

$$R = 8nl / \pi \cdot r^4$$

VASCULAR RADIUS

Resistance to Flow (R)

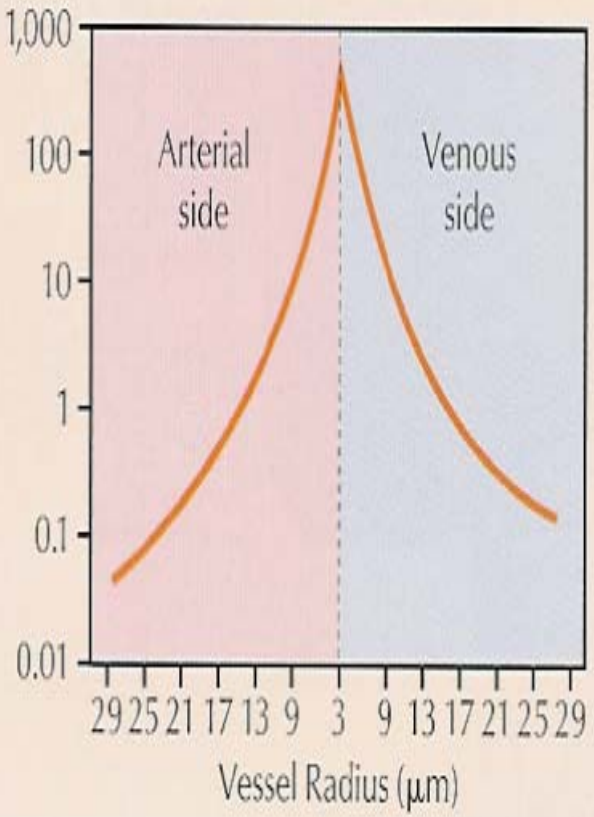
Poiseuille's Law

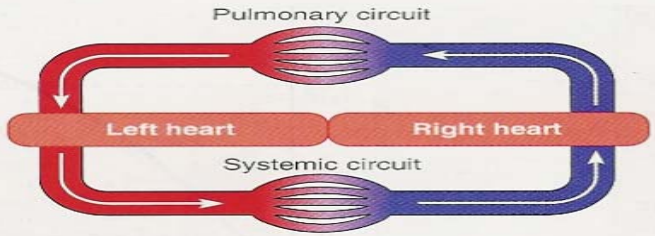
$$R = \frac{\eta \times L \times 8}{\pi r^4}$$

η = viscosity
L = vessel length
r = vessel radius

Resistance (R) per unit length

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

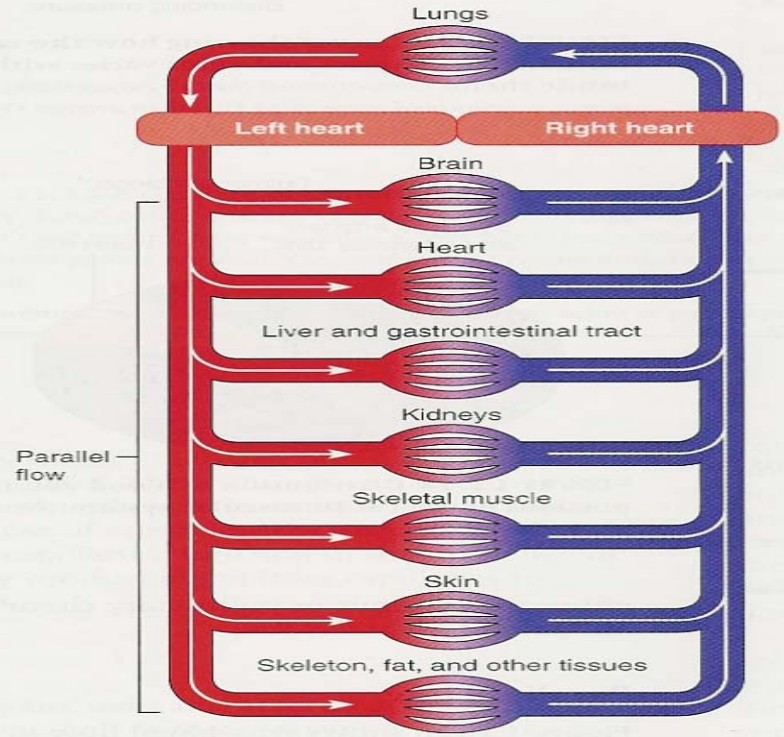




(a) Series flow

= Oxygenated blood

= Deoxygenated blood



	Rest CO = 5 l/min	Exercise CO = 25 l/min
	100% = 5 l/min	100% = 25 l/min
Brain	13–15% = 0.65–0.75 l/min	3–4% = 0.75–1.00 l/min
Heart	4–5% = 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	
Skeleton, fat, and other tissues	10–15% = 0.50–0.75 l/min	1–2% = 0.25–0.50 l/min

UNIQUE CHARACTERISTICS OF THE PULMONARY CIRCULATION

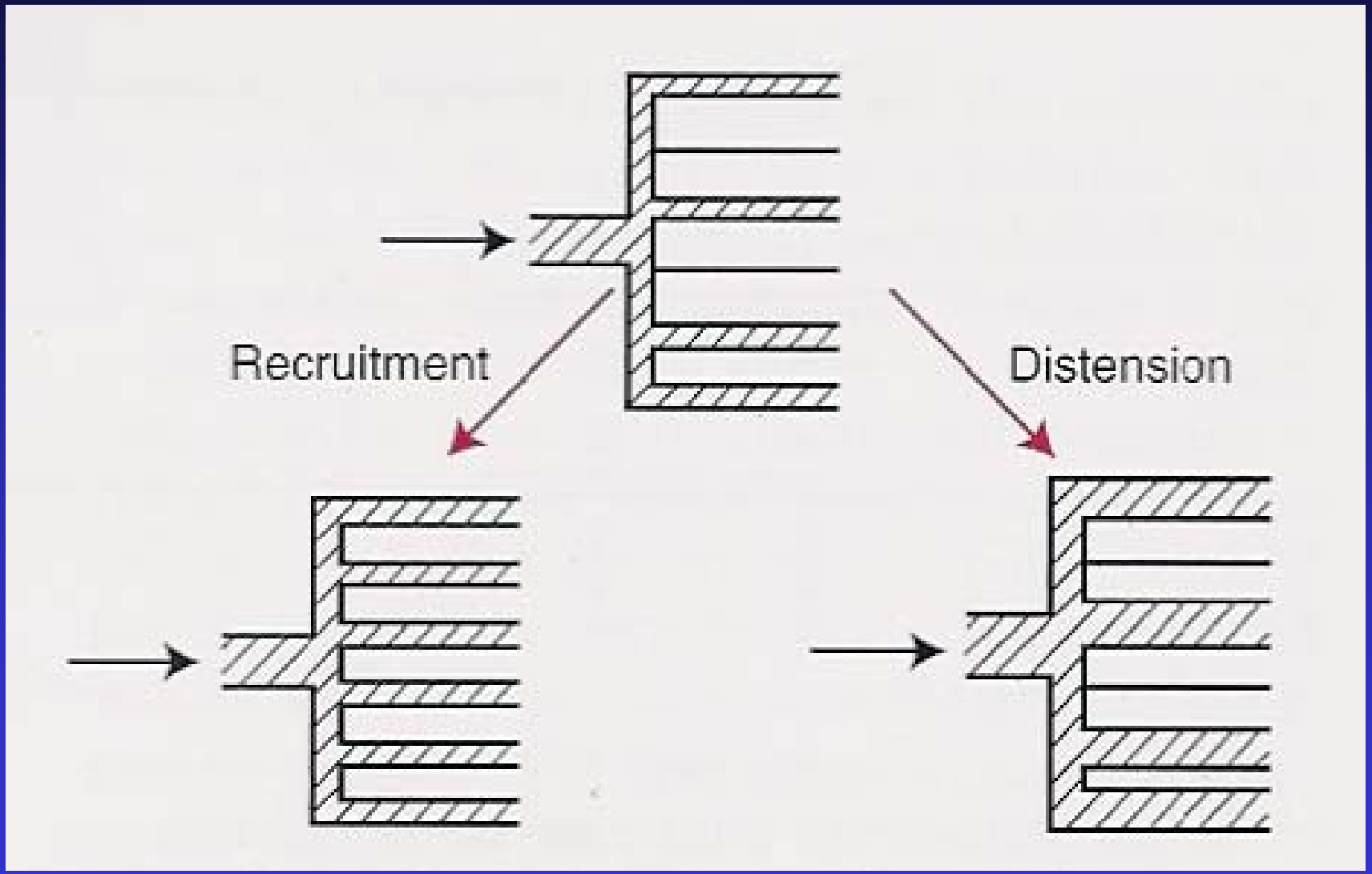
THE PULMONARY CIRCULATION MUST, AT ALL TIMES, ACCEPT THE ENTIRE CARDIAC OUTPUT

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

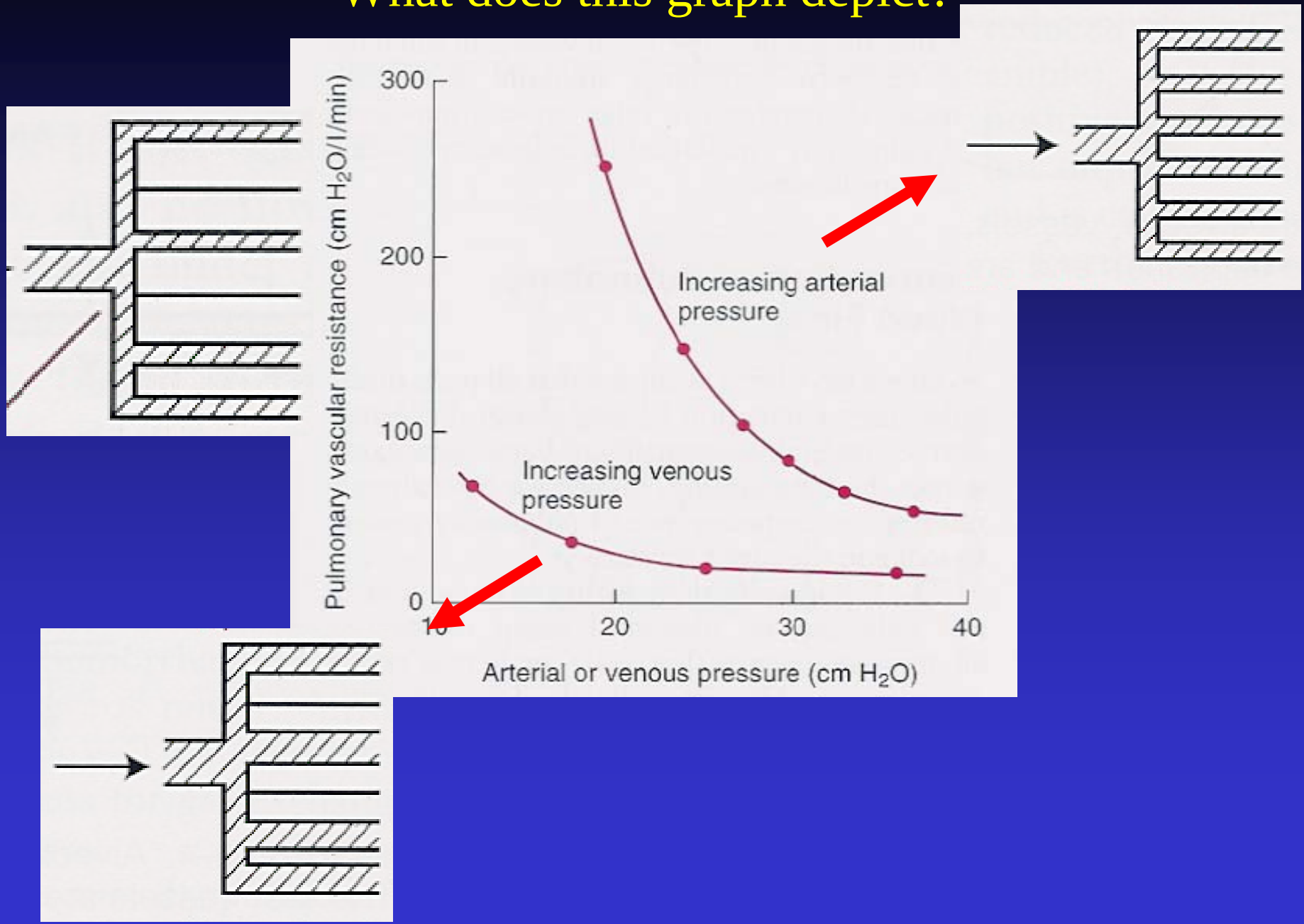
VASCULAR RESISTANCE IN THE PULMONARY CIRCULATION IS ONE-TENTH THAT OF THE SYSTEMIC CIRCULATION

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?



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?.

$$R = P_1 – P_2 / Q$$

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 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:

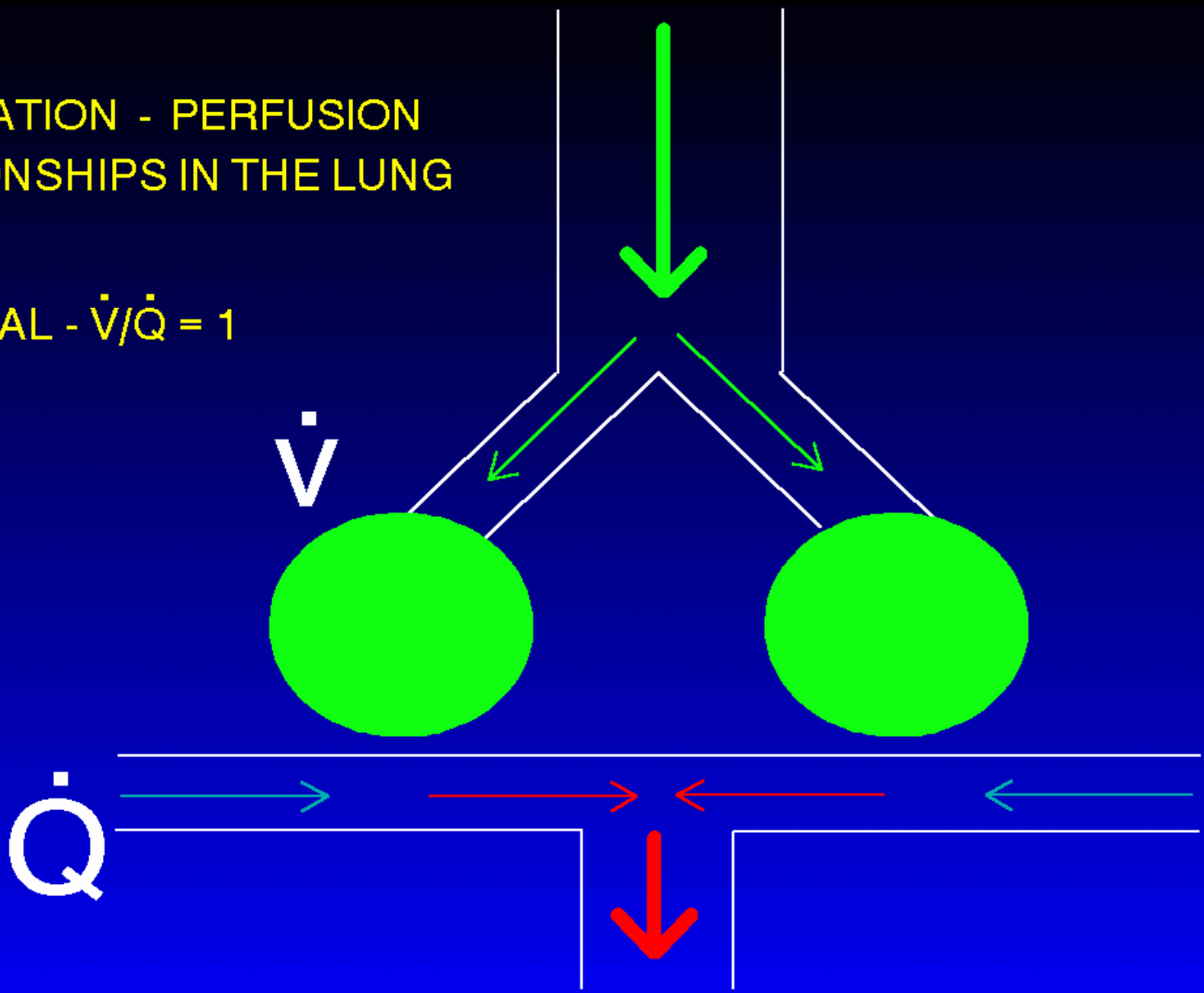
Pulmonary vasculitis

Pulmonary embolism

HPV

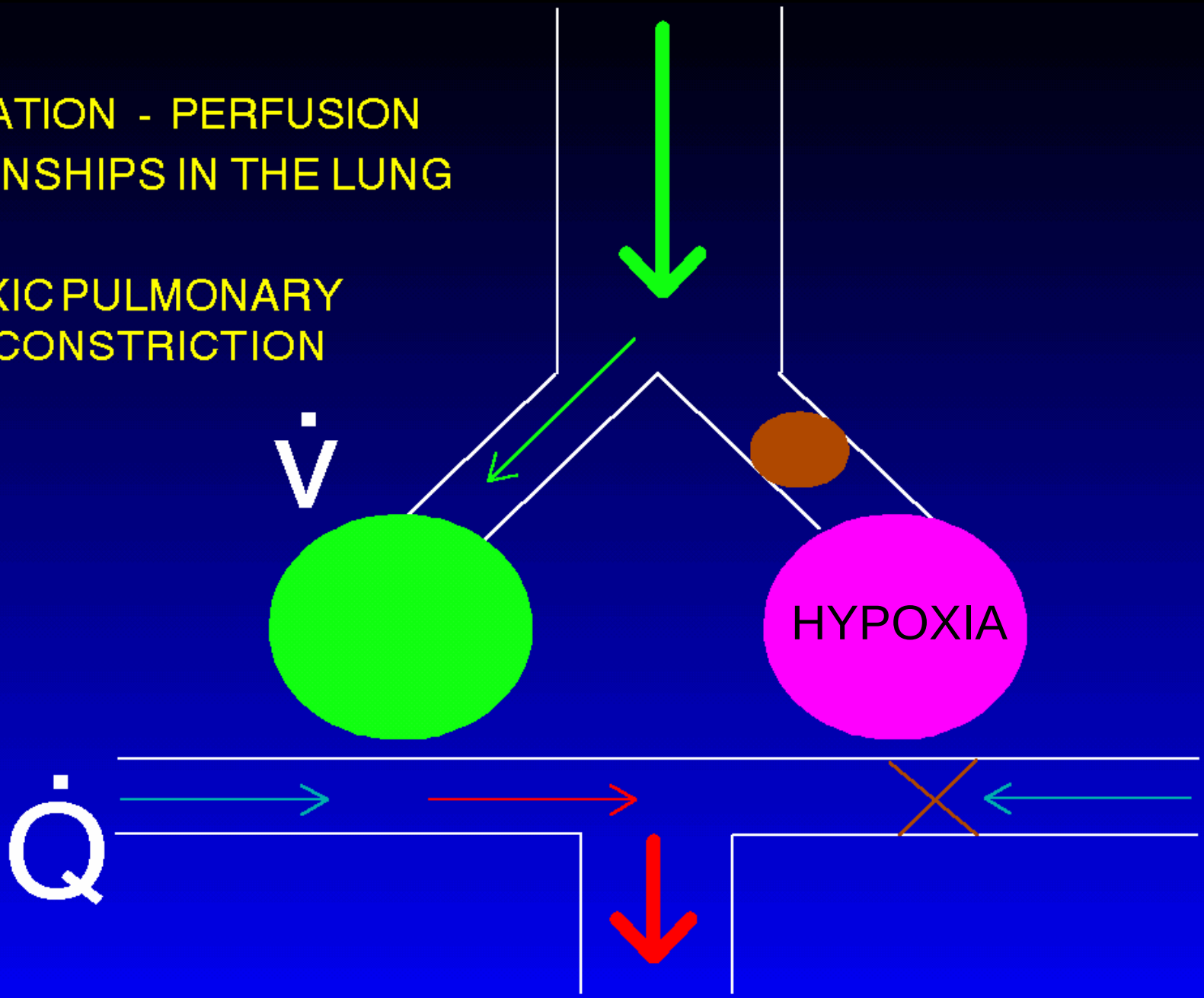
VENTILATION - PERFUSION RLEATIONSHIPS IN THE LUNG

IDEAL - $\dot{V}/\dot{Q} = 1$



VENTILATION - PERFUSION RLEATIONSHIPS IN THE LUNG

HYPOXIC PULMONARY VASOCONSTRICTION



Pulmonary Hypertension:

Pre-capillary:

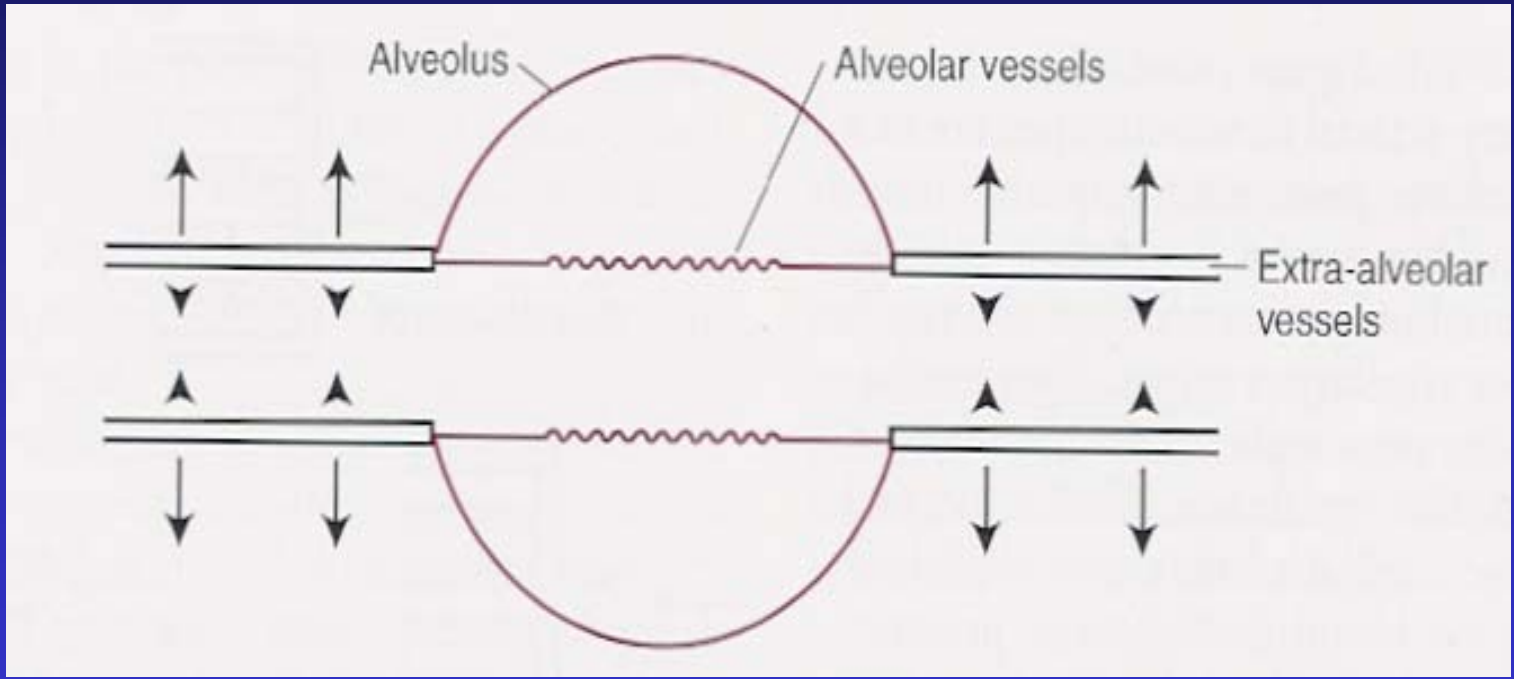
Pulmonary vasculitis

Pulmonary embolism

HPV

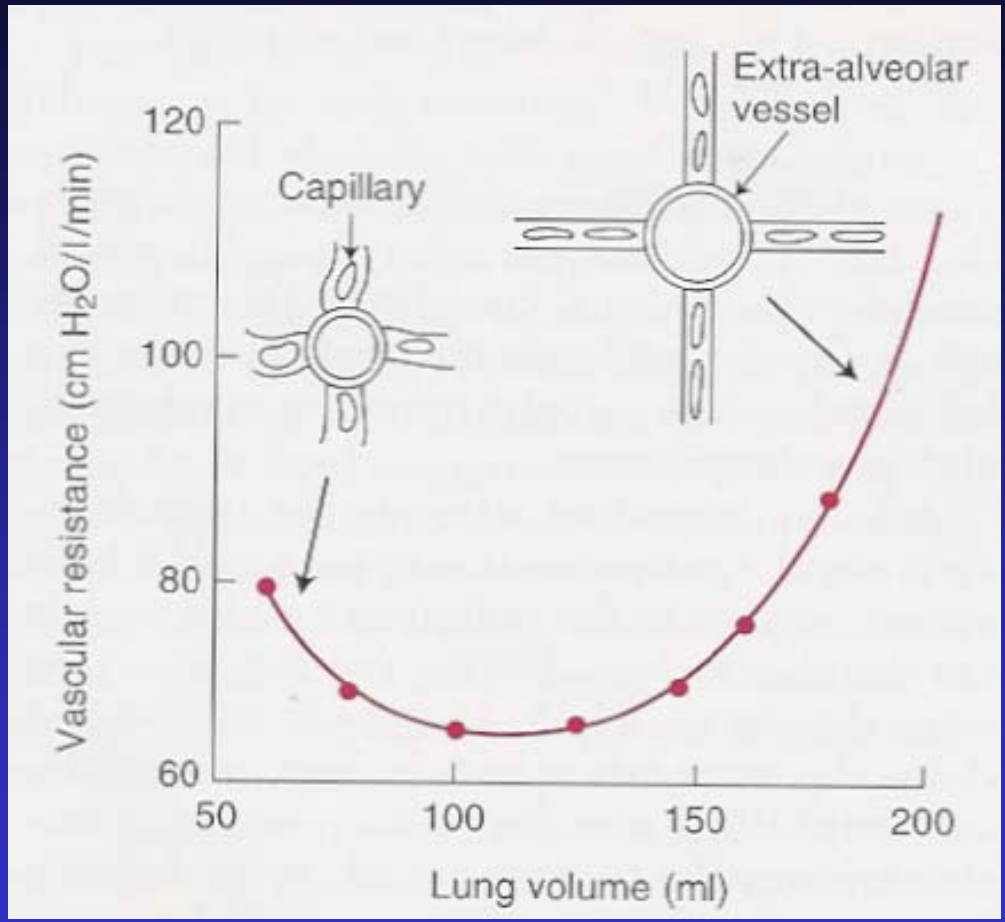
Increased Lung Volume

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 penetrence.

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?

$$R = 8nl / \pi \cdot r^4$$

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

$$R = 8nl / \pi \bullet r^4$$

Vascular remodeling:

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

$$R = 8nl / \pi \bullet r^4$$

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?

$$R = 8nl / \pi \bullet r^4$$

Vasoconstriction:

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

$$R = 8nl / \pi \bullet r^4$$

Vasoconstriction:

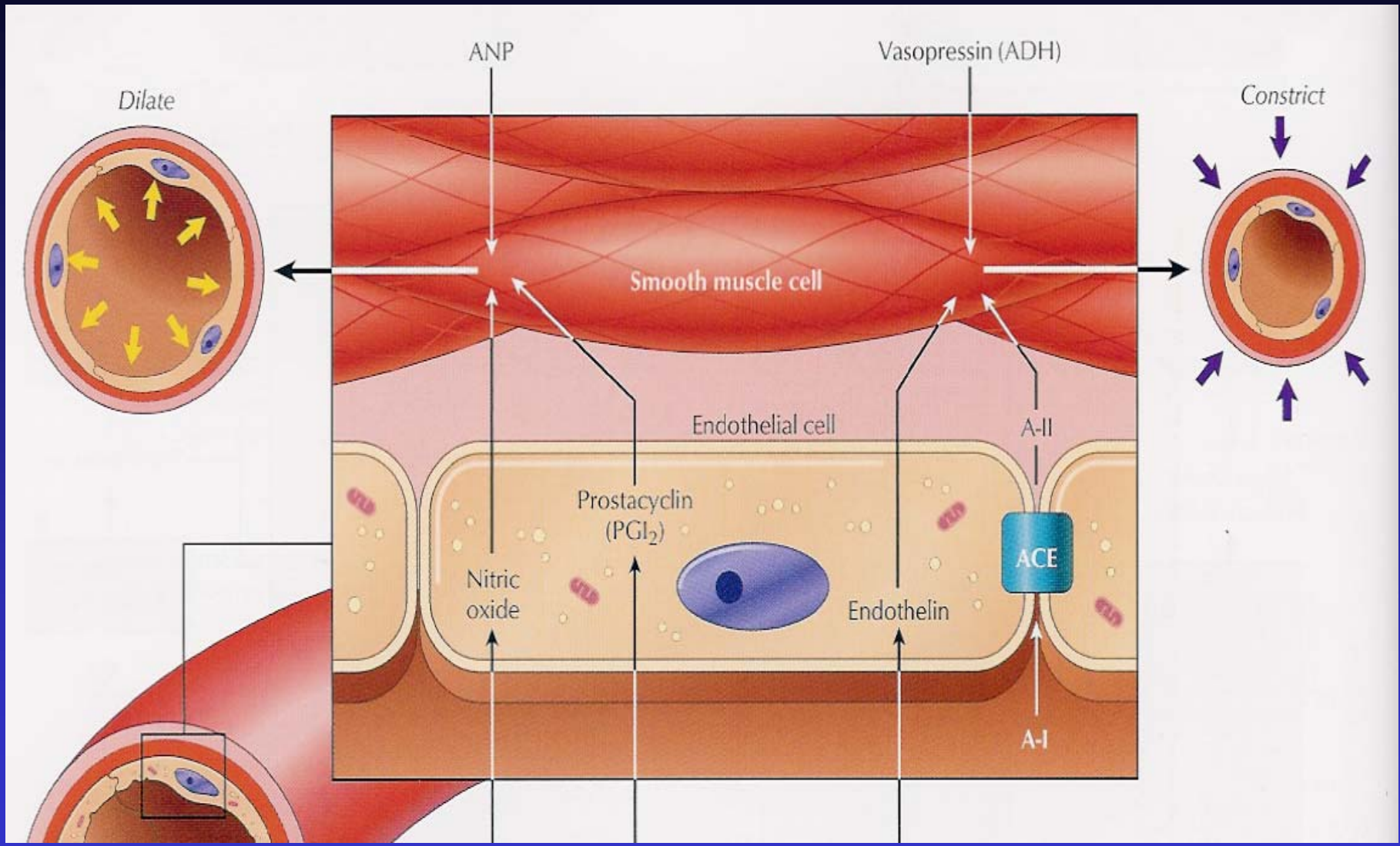
increased synthesis/activity of vasoconstrictors

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

$$R = 8nl / \pi \bullet r^4$$

Vasoconstriction:

- increased synthesis/activity of vasoconstrictors
- decreased synthesis/activity of vasodilators


ANP
Vasopressin (ADH)
Dilate
Constrict
Smooth muscle cell
Endothelial cell
A-II
Prostacyclin
(PGI_2)
ACE
Nitric
oxide
Endothelin
A-I

Were do these mediators that produce vasodilation come from?

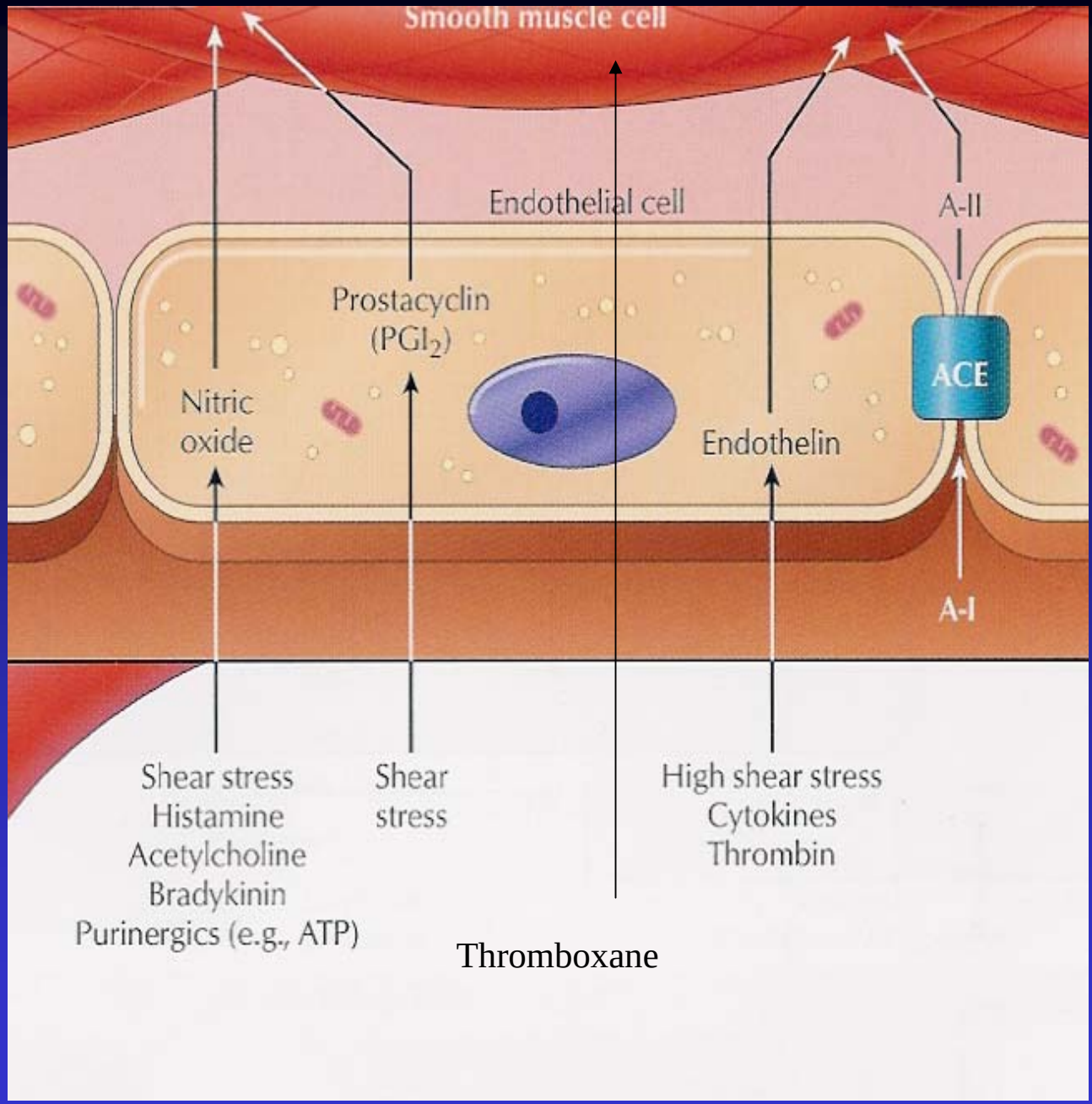
Prostacyclin: endothelial cells

Were do these mediators that produce vasodilation come from?

Prostacyclin: endothelial cells

Nitric oxide: endothelial cells

What stimulates the endothelium to produce these mediators?


Smooth muscle cell
Endothelial cell
A-II
Prostacyclin
(PGI_2)
ACE
Nitric
oxide
Endothelin
A-I
Shear stress
Histamine
Acetylcholine
Bradykinin
Purinergics (e.g., ATP)
Shear
stress
High shear stress
Cytokines
Thrombin
Thromboxane

SUMMARY

The pulmonary circulation is a low resistance circuit.

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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.