Pulmonary circulation

Ventilation/perfusion matching

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Specifics of the pulmonary circulation

- Function (O_2 into blood)
- ~ whole cardiac output
- Capillaries surrounded by air → no support against intravascular pressure → pressure has to be low
- High flow at low pressure → low vascular resistance

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Pulmonary circulation parameters

Flow (~cardiac output)	4-8 l/min
Pulmonary artery pressure (PAP)	25/8 mmHg (mean 13-16 mmHg)
Left atrium pressure	1-5 mmHg
Wedge pressure	5-8 mmHg

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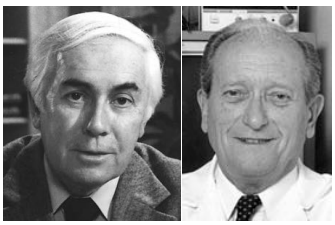
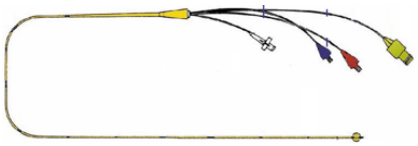
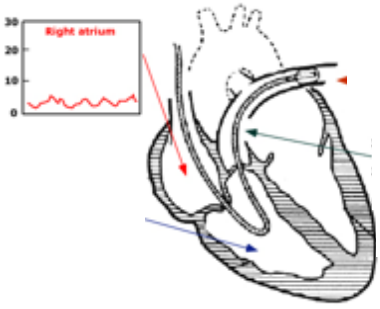
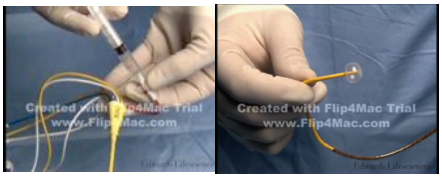
Methods

- Catheterization (Swan-Ganz)
  - pressures (incl. wedge)
  - cardiac output









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Methods

- Catheterization (Swan-Ganz)
  - pressures (incl. wedge)
  - cardiac output
  - exercise
  - vasodilatory test
- Echocardiography

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Bronchial circulation

- Systemic vascular bed
- Nutrition for airways and larger pulmonary vessels
- Some anastamoses into pulmonary veins: "physiological" shunt
  - cca 1% of cardiac output
  - lowers PaO_2 by ~2 mmHg, SaO_2 by ~0.5%
- Can partly replace pulmonary vessels in embolism

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Other functions of the pulmonary vascular bed

- Metabolic
  - ACE (1 & 2)
  - removal of BK, ET, 5-HT…
- Filtering emboli
  - PAP at rest ↑ only when >30% obstructed

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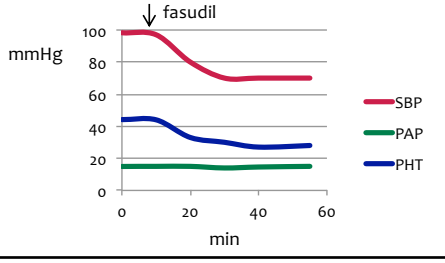
Pulmonary circulation: normally low resting vascular resistance

- Short vessels
  - Hagen–Poiseuille law
- Thin vascular wall (large compliance)
- Minimal resting tone



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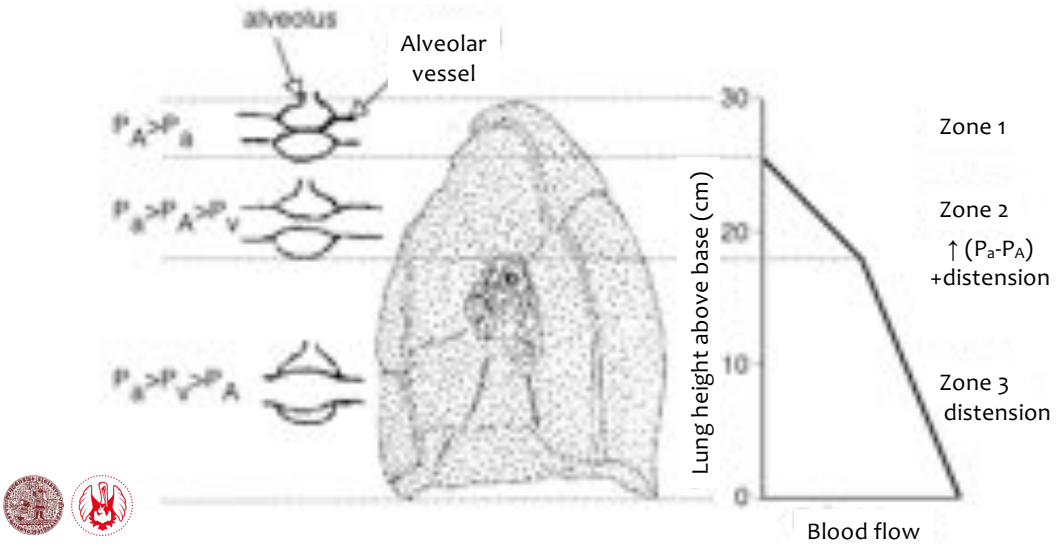
Lung blood flow rises towards bases



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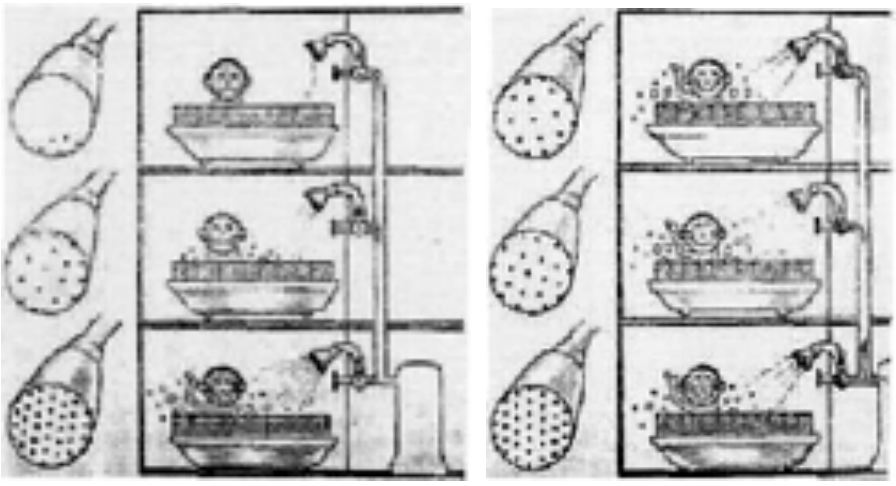
↑Pa improves perfusion of apical regions



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Consequences of lung blood flow zones

- Small ↑ Pv does not have to ↑ Pa
  - ☞ At Pv > PA, Pa rises proportionately to Pv
- Lung edema starts at the bottom (highest pressures)
- Positive pressure ventilation: zone 3 reduced, zones 1 & 2 increase

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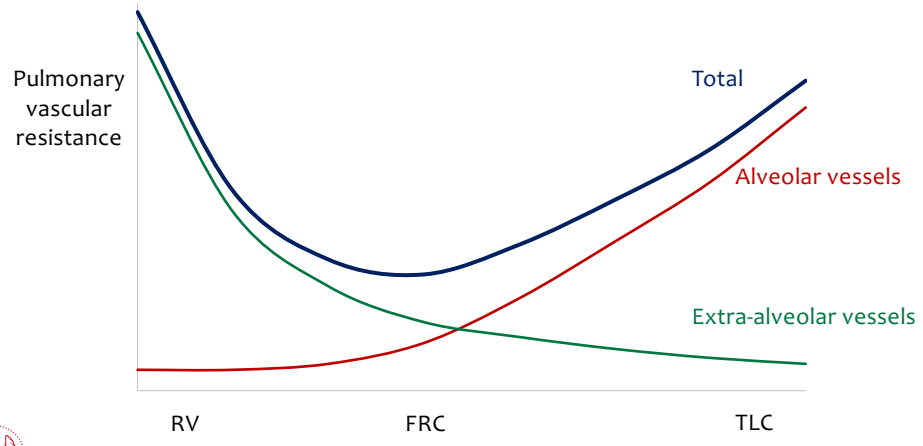
Lung volume & vascular resistance



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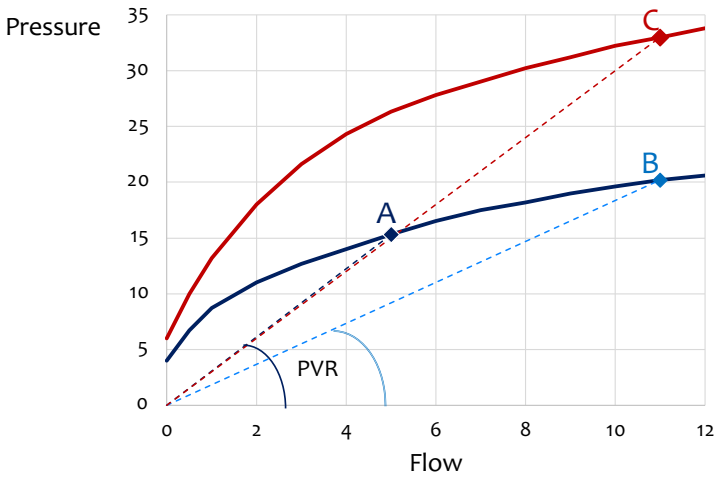
Pulmonary vascular resistance



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↑ cardiac output (e.g. exercise)

- ↓ PVR (mainly microcirculation):
  - distension
  - recruitment (zones, critical opening pressure)
- → only minimal ↑ pressure
  - saves heart work, prevents edema
  - extreme exercise: „stress failure“ of lung capillaries
  - RBC passage through lung capillary shortens from ~0.8 sec to ~0.3 sec

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Regulation of the pulmonary circulation

- Blood flow = cardiac output
- Minimal neural regulation
  - only ↑ venous return - SNS
- Humoral influences:
  - platelets, macrophages, endothelium,…
  - TxA_2, PGI_2, NO, ET, 5-HT,...
  - mainly pathology (pulmonary hypertension, edema, embolism,..)
- Local regulation
  - intra-organ flow distribution (V/Q matching)

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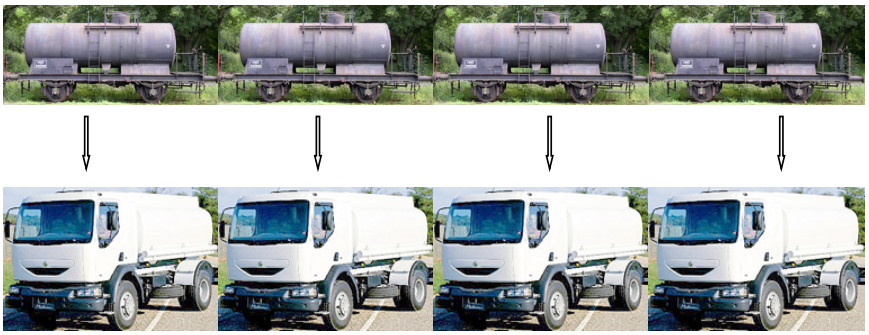
Ventilation/perfusion ratio (V/Q)



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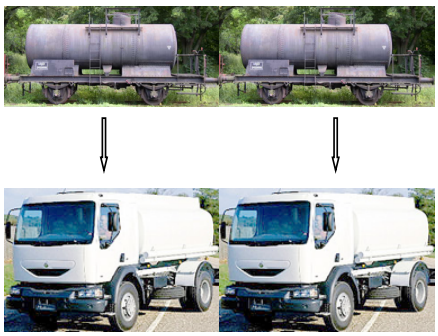
V/Q

20

V/Q

21

V/Q



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V/Q

Ventilation

↑V/Q
Dead space

Perfusion

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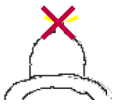
V/Q



↓V/Q
Shunt
Venous admixture

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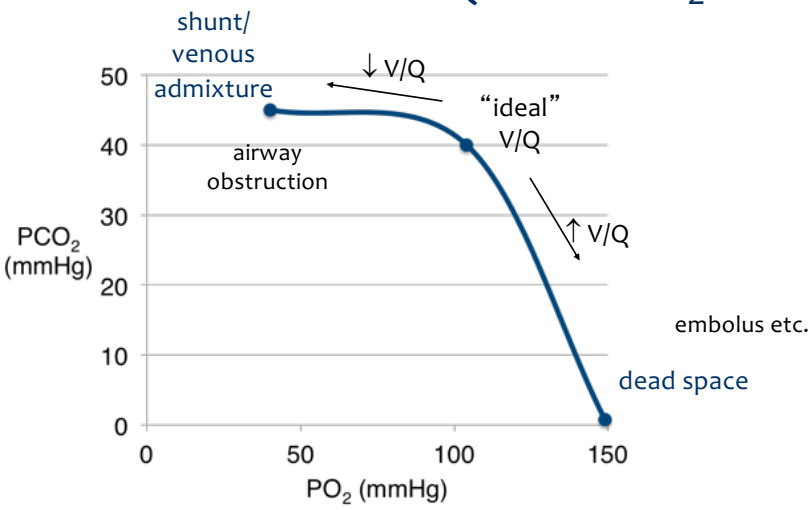
Rahn-Fenn diagram (local PO_2-PCO_2)



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Ventilation/perfusion mismatch

- Some even in health
  - effect of gravitation: Q>V
  - mucus
  - variability in resistance & compliance of airways & alveoli
- Main cause of hypoxemia in lung diseases
  - ↓ V/Q → venous admixture
  - ↑ V/Q → Q only through unaffected parts → relatively ↑ Q there → venous admixture

O_2 transport/perfusion inhomogeneity probably also in other organs

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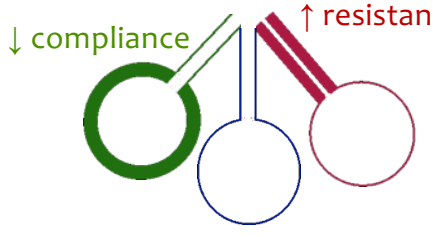
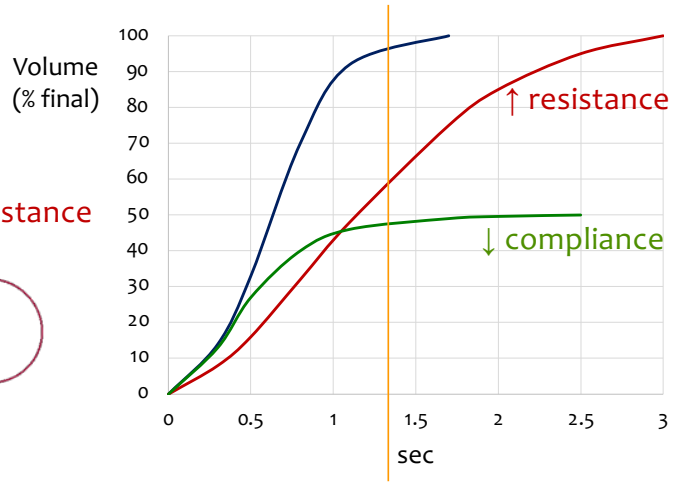
↑ resistance & ↓ compliance → ↓ventilation at normal f





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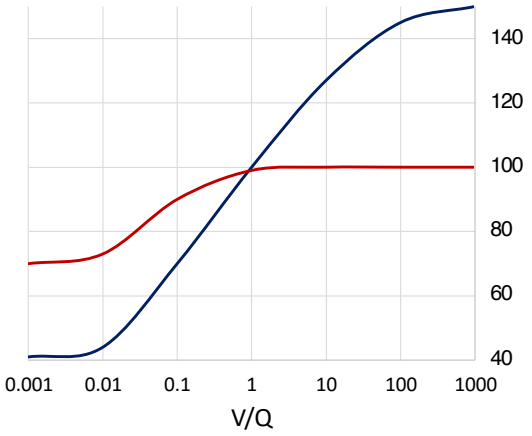
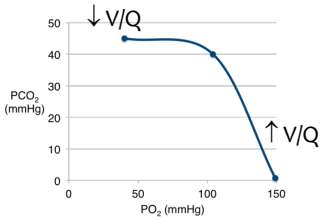
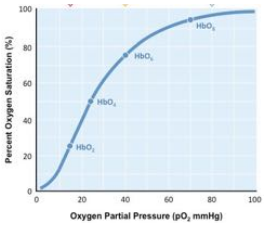
Local ↑ V/Q above ~1 has minimal effect on S_aO_2



P_aO_2 (mmHg) or S_aO_2 (%)





Thus ↑ V/Q in one part of lung can't compensate for ↓ V/Q elsewhere

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Why to detect V/Q (in)equality?

To determine the causes of hypoxemia:

- Intrapulmonary
  - shunts
  - dead space
- Extrapulmonary
  - hypoventilation
  - anemia
  - cardiac failure
  - A-B disbalance

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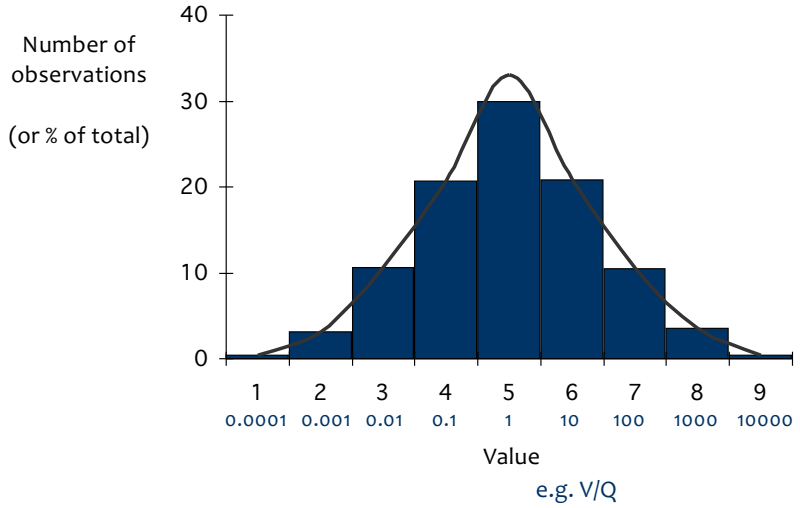
Quantification of V/Q matching: Distribution



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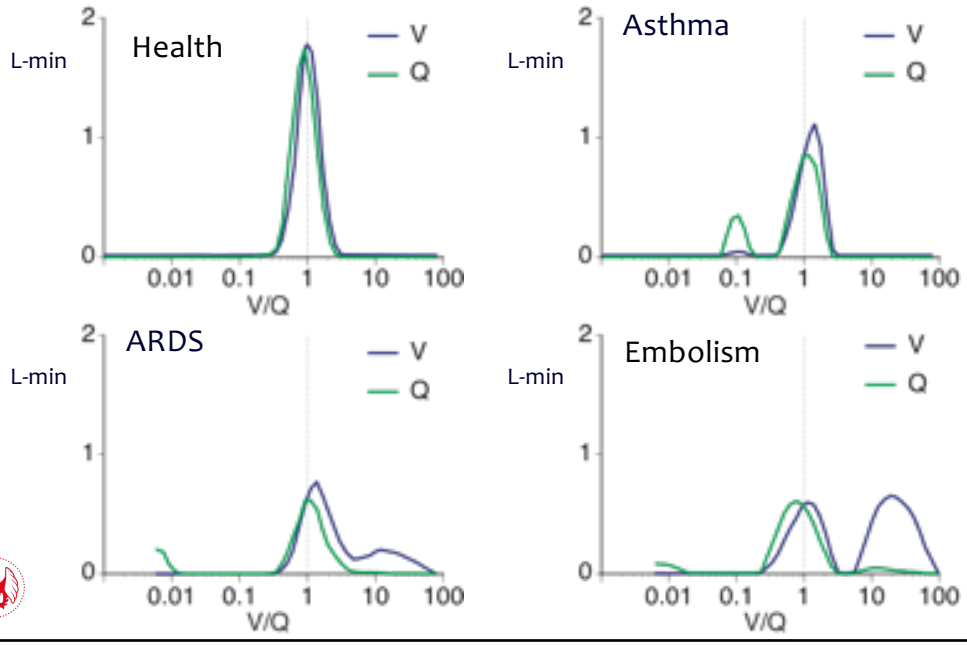
Multiple Inert Gas Elimination Technique (MIGET)



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Doing MIGET

- I.V. infusion of solutions of variably dissolving gases
  - ✓ Acetone (most dissolving)
  - ✓ Ether
  - ✓ Enflurane
  - ✓ Cyklopropane
  - ✓ Ethane
  - ✓ SF6 (least dissolving)
- Detection in exhaled air & in arterial blood

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MIGET principle

- Admixture of air <u>from dead space</u> (where the injected gas couldn't penetrate from the blood) "dilutes" the total exhaled air
  → with higher V/Q, less injected gas appears in exhaled air
- Admixture of <u>blood from shunt</u> (where the injected gas couldn't escape from blood) prevents a decrease in injected gas concentration in arterial blood
  → with lower V/Q, more injected gas appears in arterial blood

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Compensation of V/Q inequality

- ↑V/Q → local hypocapnia → ↑ pH → local bronchoconstriction - weak
- ↑V/Q → ↓ surfactant → ↓ compliance → ↓ volume
- ↓V/Q → ↑CO_2 → ↑ ventilation
  - improves CO_2 > O_2 (dissociation curves)
- ↓V/Q → hypoxic pulmonary vasoconstriction

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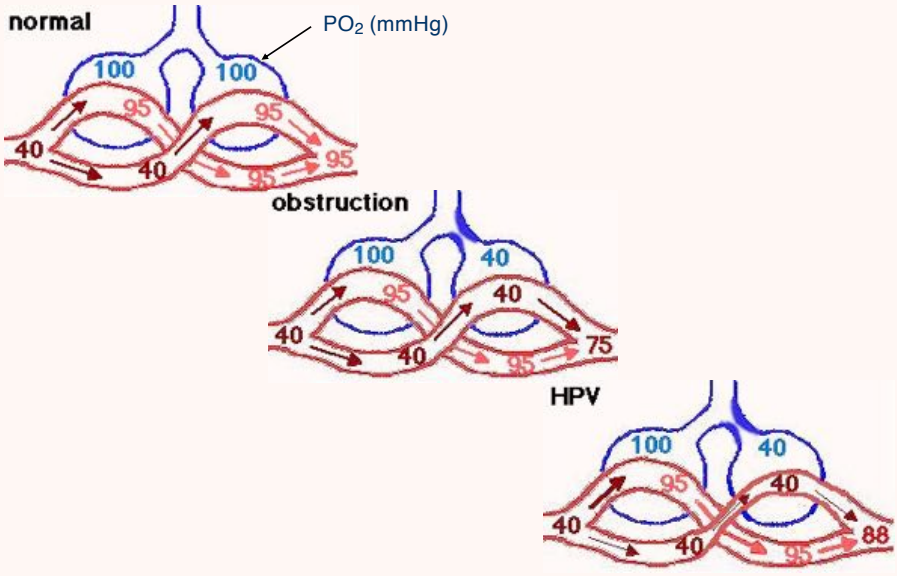
Hypoxic pulmonary vasoconstriction maintains V/Q



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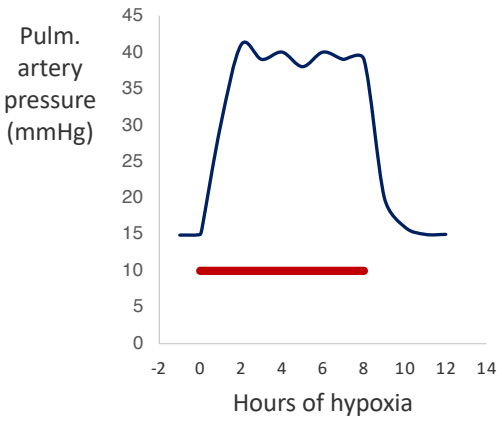
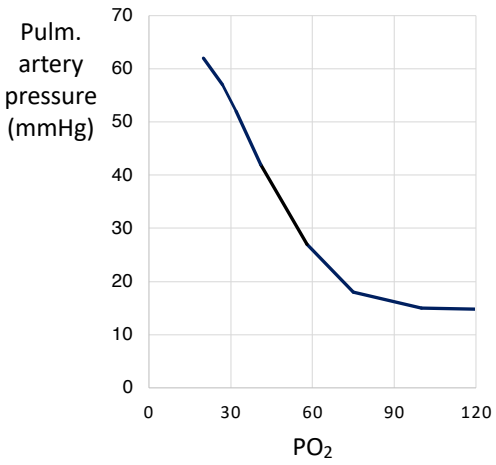
HPV: fast, sustained, reversible, depends on degree of hypoxia





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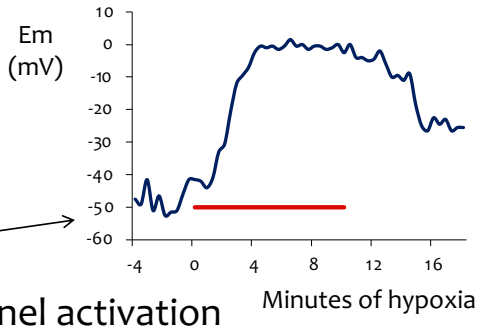
Mechanisms of HPV

- redox changes???
- K^+ channel inhibition
- depolarization
- voltage-gated Ca channel activation
- Ca^{2+} influx
- Ca^{2+} release from intracellular stores
- contractile apparatus activation



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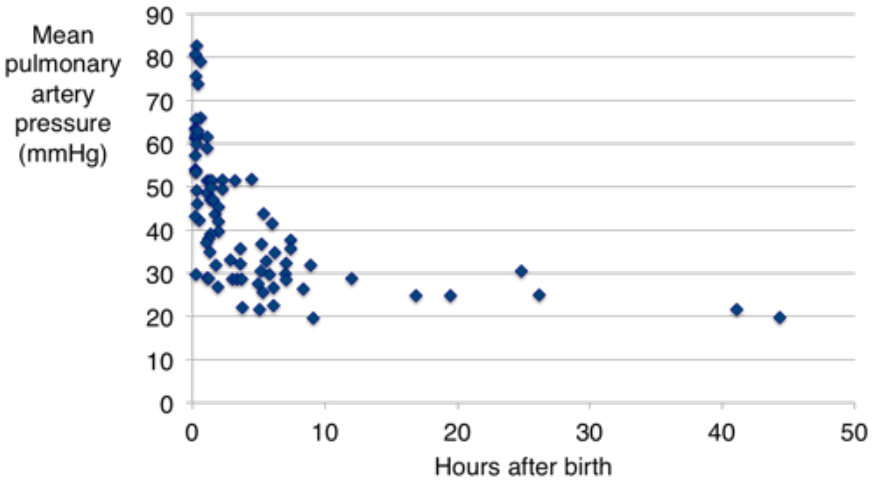
Pulmonary vasodilation at birth



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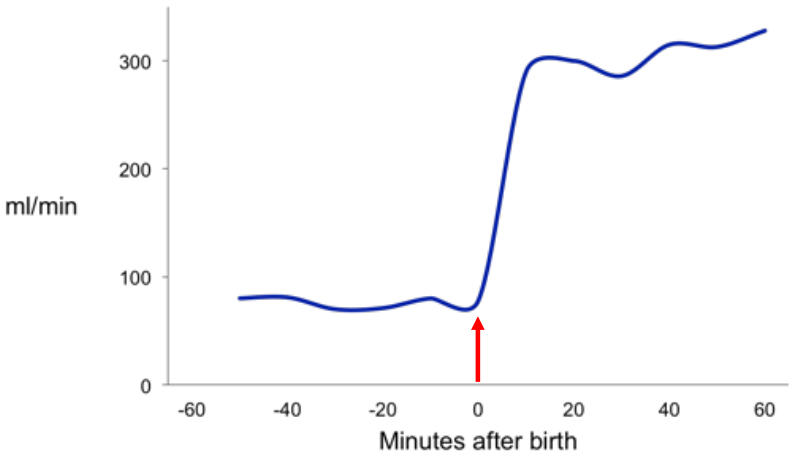
Lung blood flow at birth



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Filtration in lung capillaries

- capillary pressure: 7 mmHg
- negative interstitial pressure: 8 mmHg
- osmotic pressure of the interstitial fluid: 14 mmHg

Σ TOTAL FORCE OUTWARD: 29 mmHg

- osmotic pressure of plasma: 28 mmHg

Σ TOTAL FORCE INWARD: 28 mmHg

Σ Net filtration pressure (outward): 1 mmHg

- drained by the lymphatic system

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Diseases

- Pulmonary hypertension
  - idiopathic
  - secondary
    (L ♥ failure, hypoxia, thrombi, schistosomies, ...)
- Pulmonary edema
  - ARDS
  - cardiogenic
  - HAPE
- Pulmonary embolism

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