1. air, gases...

$p_B = 760\ mmHg \approx 100\ kPa$

21% O_2, 79% N_2

in air we get together with water vapour

at 37°C $p_{H_2O} = 47\ mmHg$

$\rightarrow 760 = 47 + \underbrace{p_{O_2} + p_{N_2}}_{21:79}$

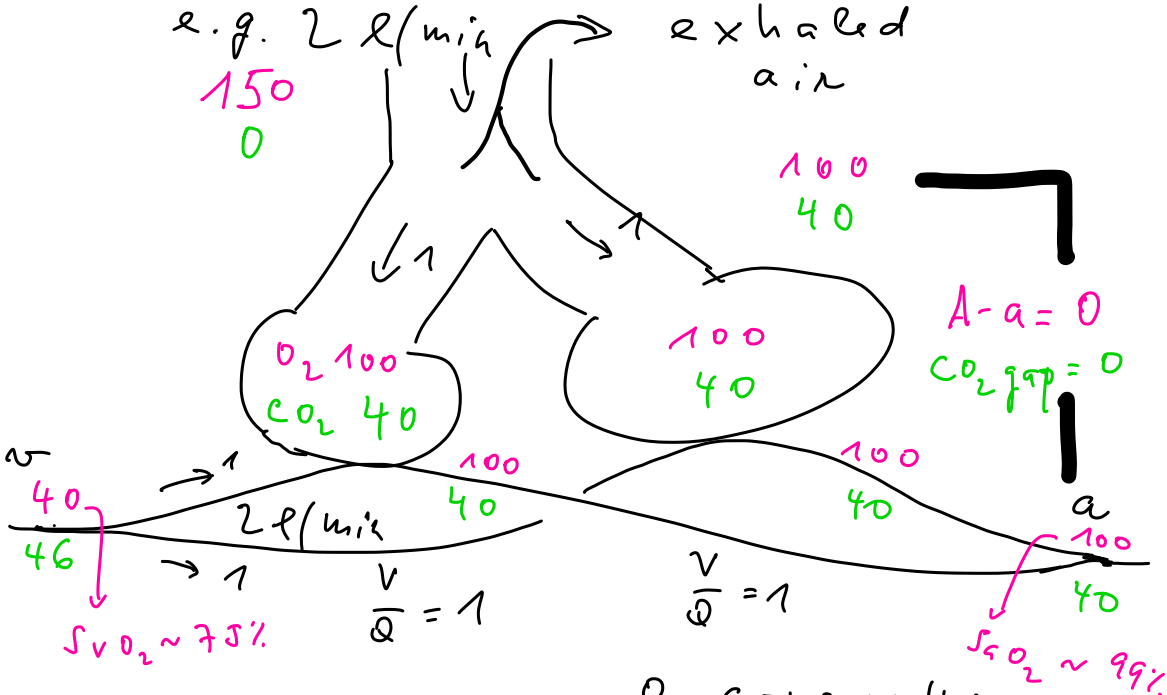
↳ in inhaled air:

$p_{O_2} = 150\ mmHg$

$p_{N_2} = 563\ mmHg$

$p_{H_2O} = 47\ mmHg$

2. ideal lungs



ventilation-perfusion ratio
- homogeneous
- $V/Q = 1$

O_2 consumption

$2 \cdot (150 - 100) = 100 / min$

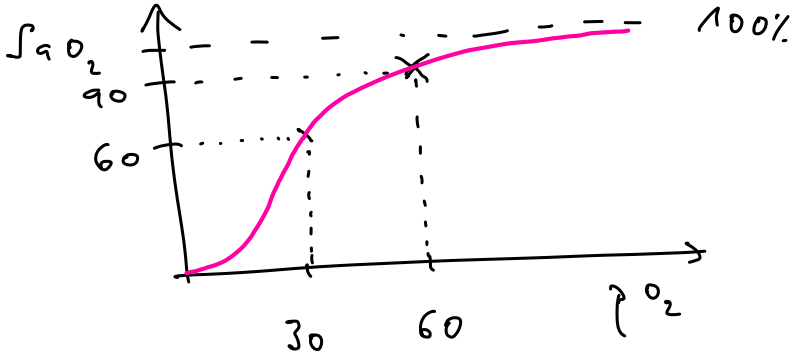
CO_2 production

$2 \cdot 40 = 80 / min$

$$R = \frac{80}{100} = 0.8 \ldots$$

respiratory quotient

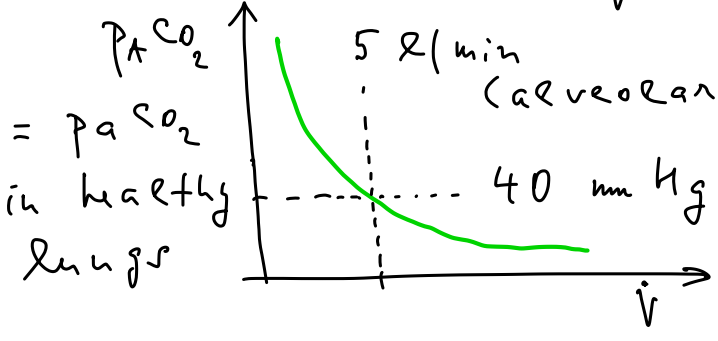
rule 30 - 60 - 90



hyperventilation

elimination of CO_2 = $P_A CO_2 \cdot \dot{V}$ = production of CO_2

↳ $P_A CO_2 = \frac{\text{const}}{\dot{V}}$ = const



3. $p_A N_2$ ... partial pressure of N_2

Since nitrogen is not metabolised, its alveolar pressure $p_A N_2$ should be the same as in the inhaled air,

i.e. 563 mmHg.

Since less CO_2 is produced than O_2 consumed ($R < 1$), the expiratory volume is lower than inspiratory and N_2 is more concentrated $\Rightarrow$ $p_A N_2 > 563$.

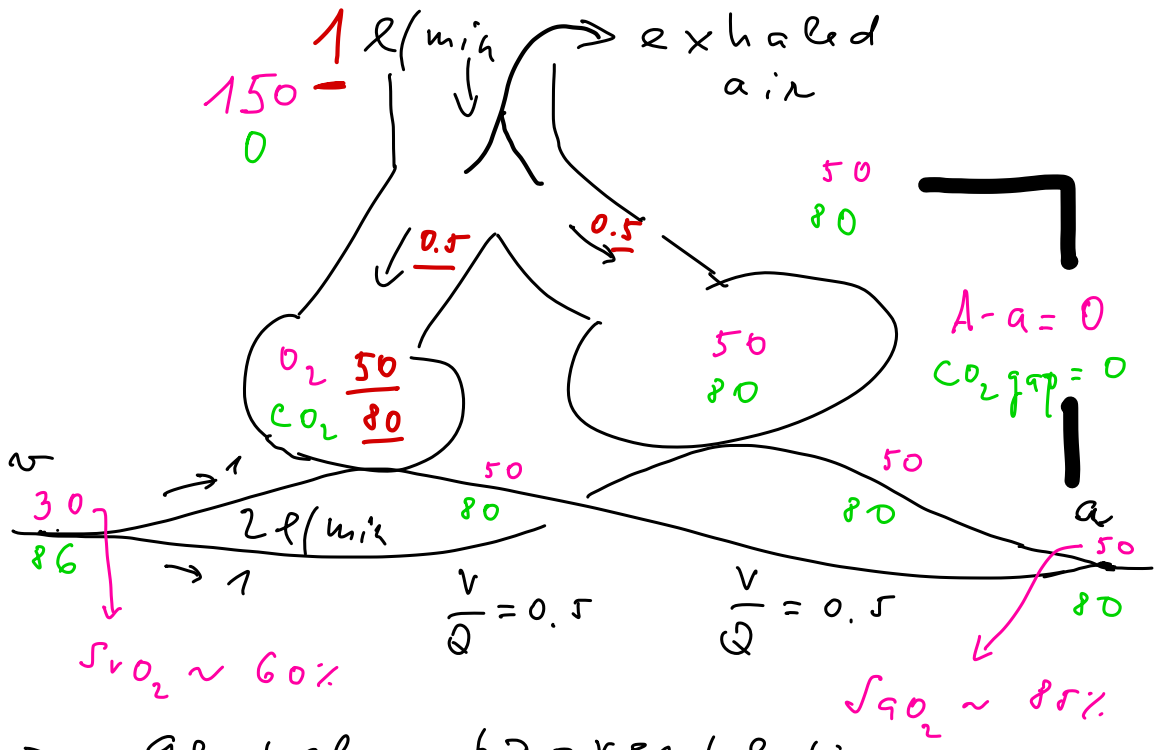
Moreover, this depends on V/Q ratio of individual alveoli $\rightarrow$ different alveoli have little different $p_A N_2$.

Agreement: These differecies will be mostly ignored in the following text.

mechanisms of respiratory insufficiency

- global hypoventilation
- impaired diffusion
- right to left shunt
- alveolar dead space
- ventilation – perfusion inequality

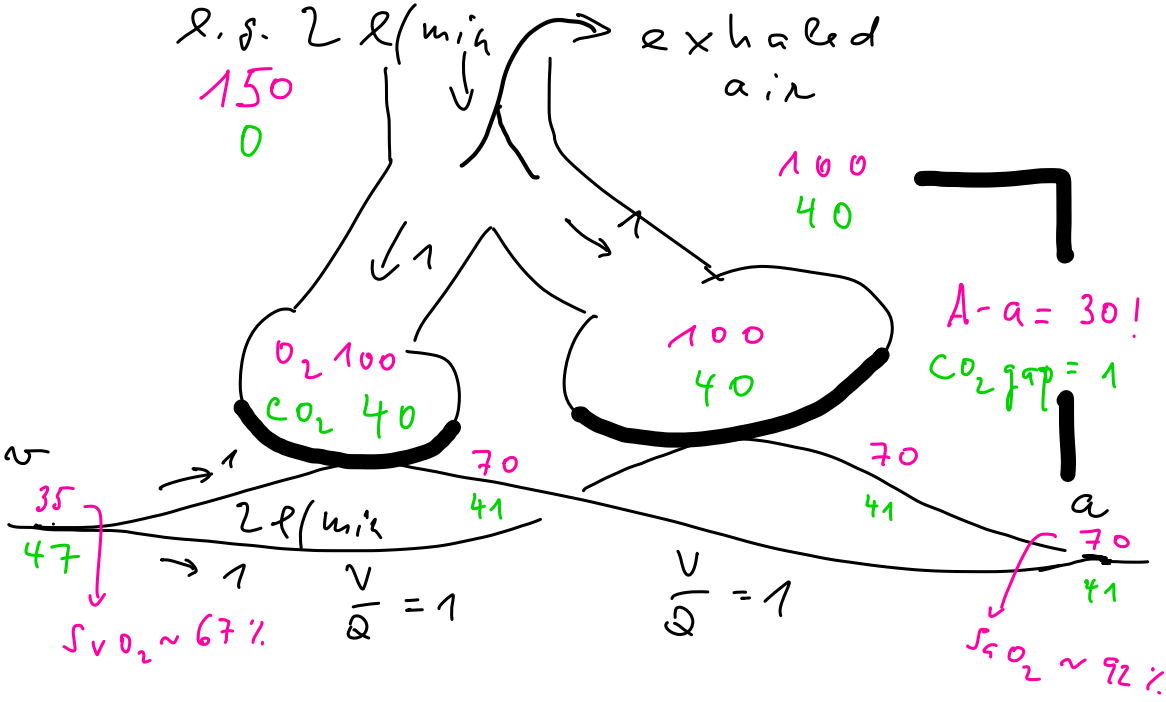
4. global hyperventilation



⇒ global hyperventilation leads to:

1) hypercapnia
2) hypoxemia
3) no increase in A-a gradient nor CO_2-gap
4) total O_2 consumption and production of CO_2 is unchanged (given by metabolic rate

5. impaired diffusion



principle: decreased venous pvO_2 leads to increased gradient on alv.-cap. membrane, which compensate for impaired diffusion

? Can it be corrected by hyperventilation 2

Can it be corrected by administration of O_2?

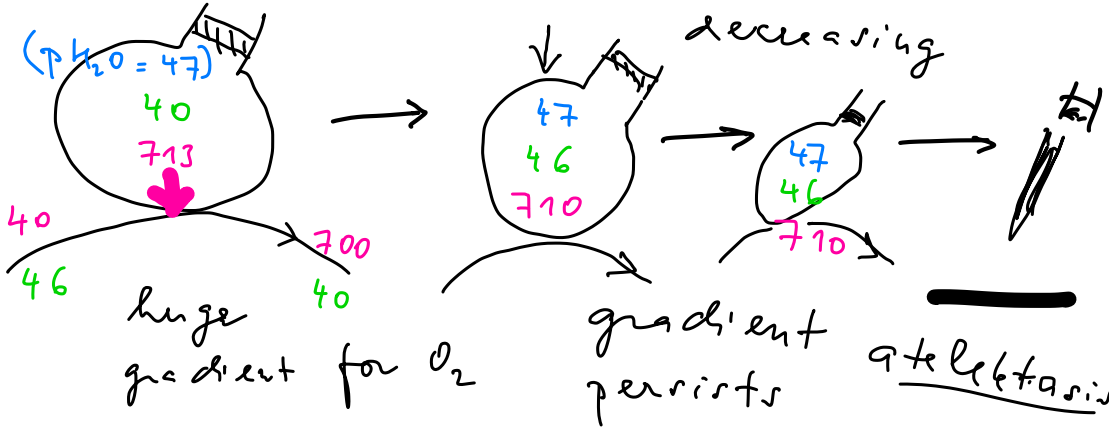
What will change on exertion?

6. Oxygenotherapy and resorption atelektases

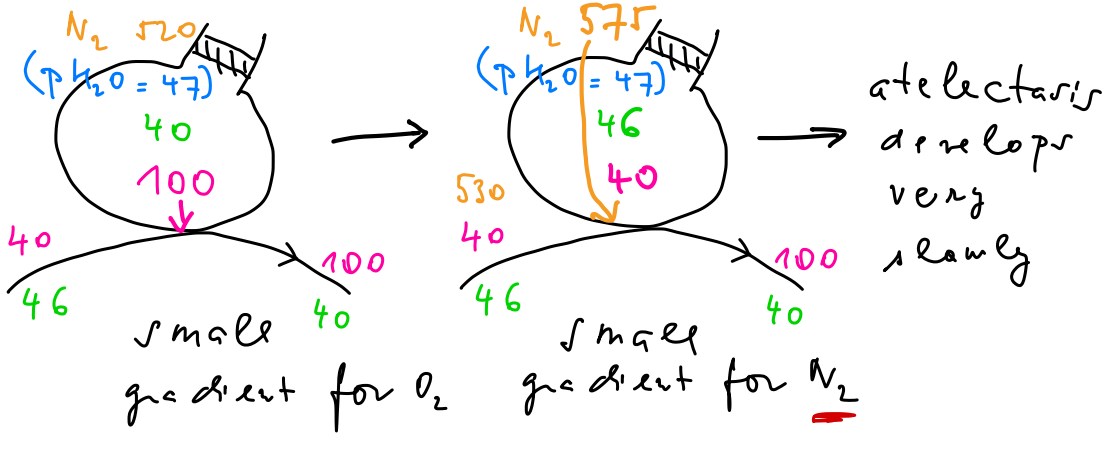
a) administration of „pure" O_2 (FiO_2 100%)

→ pO_2 = 713 mHg

let's imagine now an obstruction of airways to alveoli for a short time

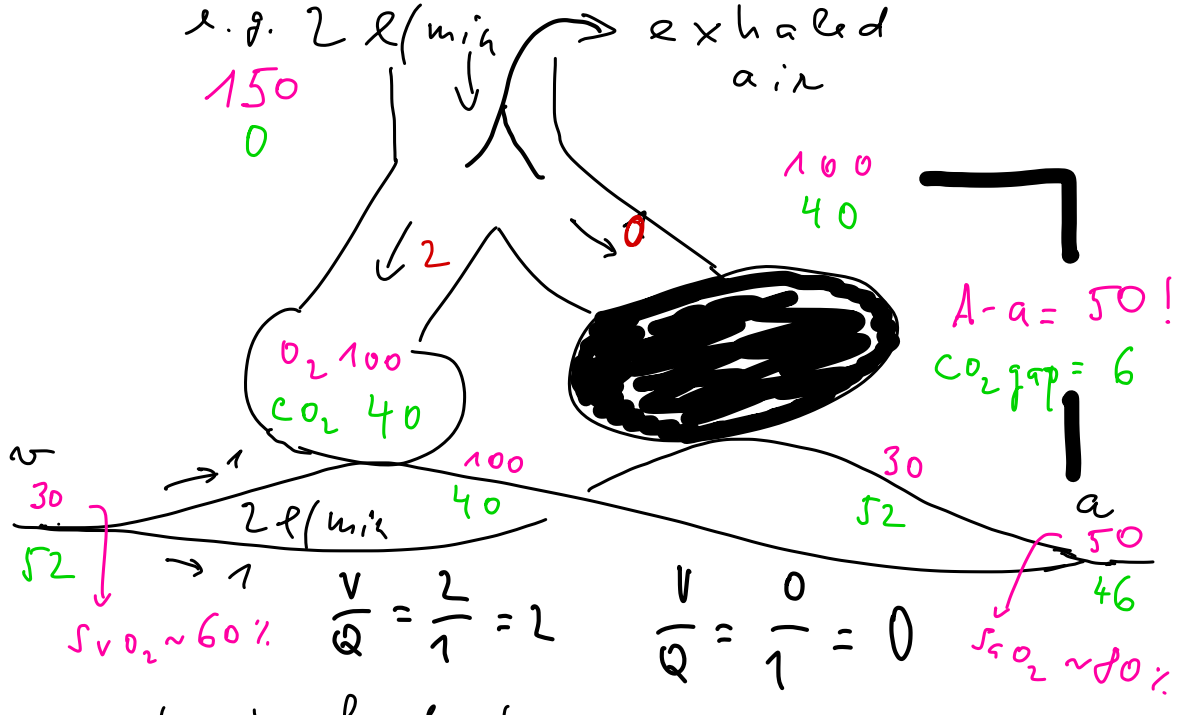


b) FiO_2 21%



7) right to left shunt

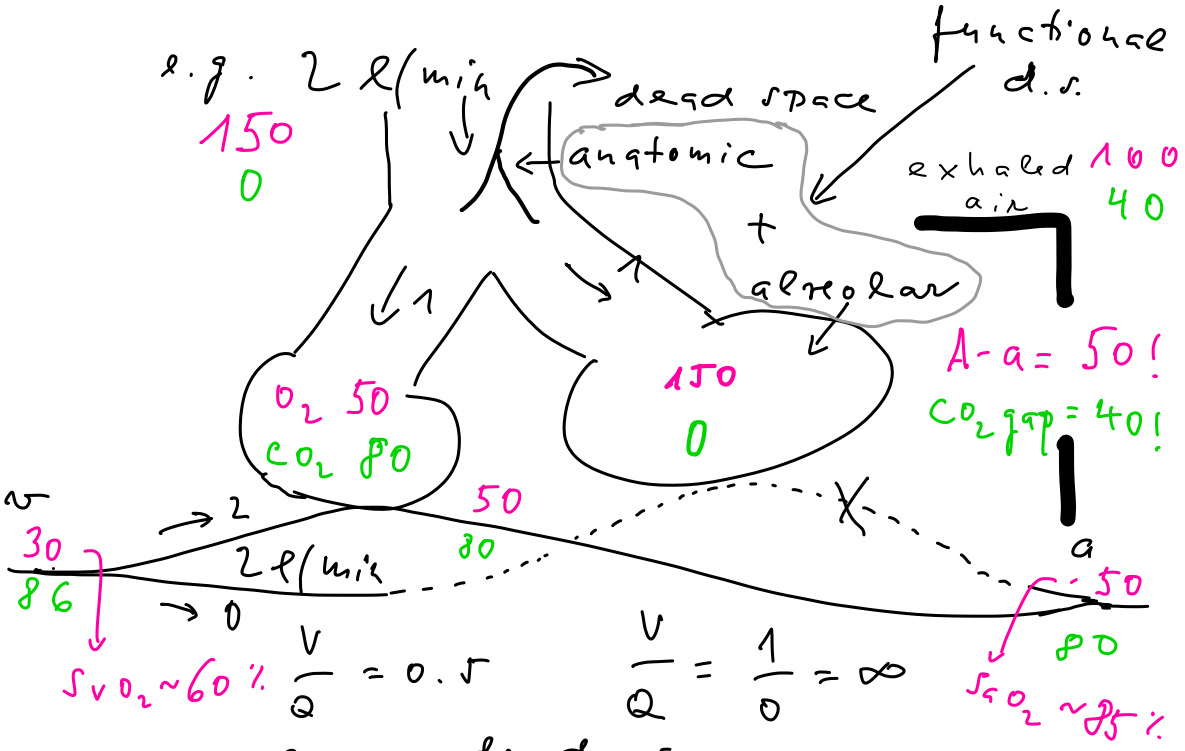
- etiology: atelektasis, infiltration, pulm. edema, ARDS...



shunt leads to:

1) hypoxemia, high A-a gradient
   - impossible to correct with hyperventilation nor with administration of O_2
2) mild hypercapnia, little CO_2 gap
   - possible to correct with hyperventilation

8. alveolar dead space



⇒ alveolar dead space leads to:

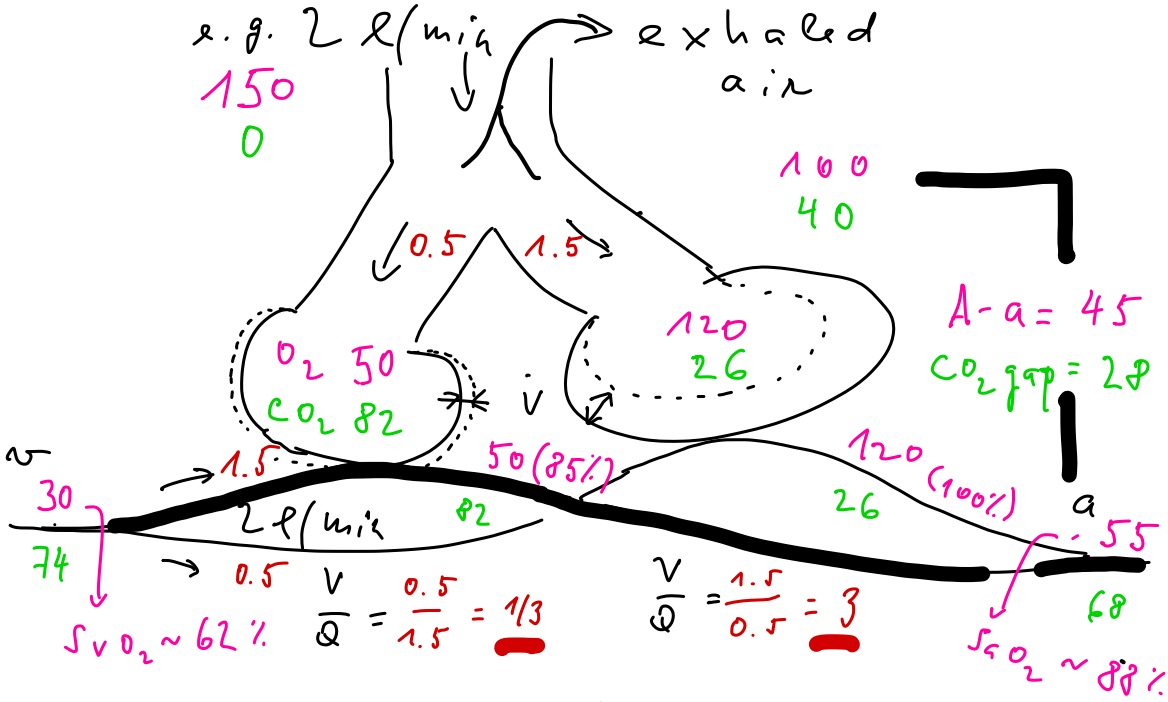
1) hypercapnia } possible to correct
2) hypoxemia } with hyperventilation
3) increased A-a gradient and CO_2-gap

|| calculated A-a gradient is related to so called ideal alveolus and does not increase with dead space

$$p_{O_2 id} = 150 - \frac{p_a CO_2}{R} = 150 - \frac{80}{0.8} = 50$$

$A-a\ gr. = p_{O_2 id} - p_a O_2 = 50 - 50 = 0$

9. ventilation - perfusion inequality



⇒ ventilation - perfusion inequality leads to:

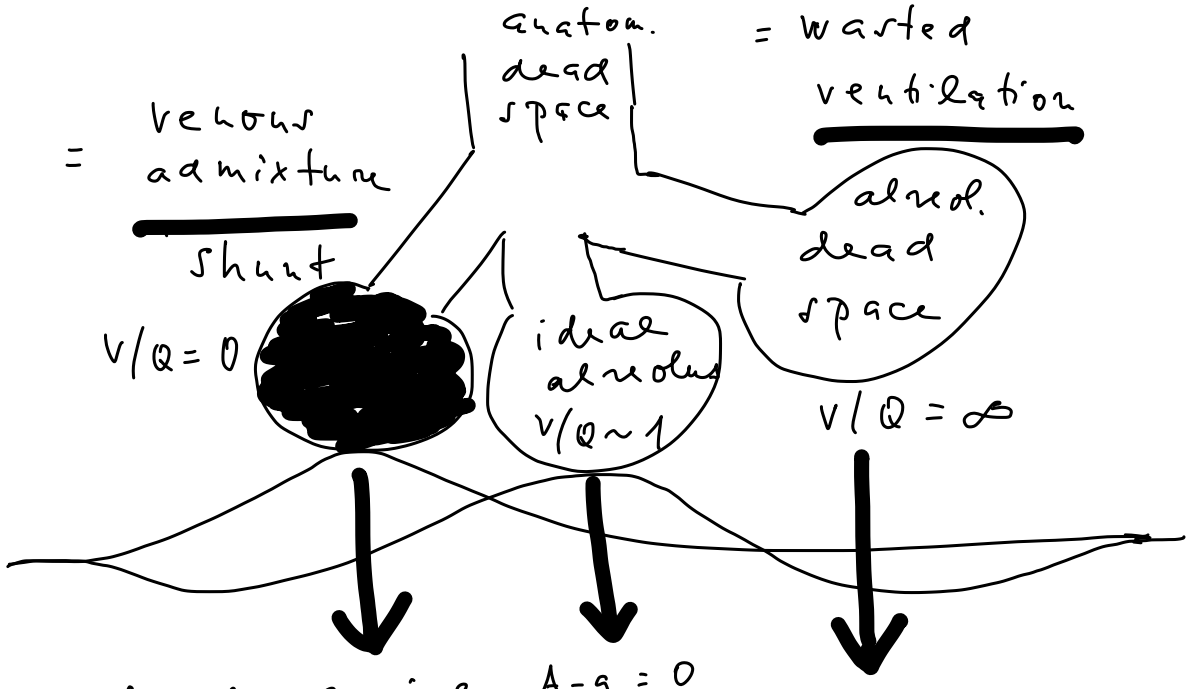
1) hypercapnia - possible to correct with
2) hypoxemia - impossible hyperventilation
   ↪ possible to correct with external O_2
3) increased A-a gradient and CO_2-gap

!! calculated A-a gradient increases as well

$$pO_{2id} = 150 - \frac{paCO_2}{R} = 150 - \frac{68}{0.8} = 65$$

A-a gr. = $pO_{2id} - paO_2 = 65 - 55 = 10$

10. 3 - compartment model



leads maing to hypoxemia

A-a = 0
CO_2 gap = 0

leads maing to hypercaphia

A - a grad. > 0

CO_2 gap ≈ 0

- pheumonia, pulm. edema, ARDS

A - a ≈ 0

CO_2 gap > 0

- pulm. embolism
- COPD

!! 3 - comp. model is a simplification, does not explain ventil.-perfusion inequality exactly e.g. does not explain effect of O_2 administration completely