Mechanisms of respiratory insufficiency

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necessary values:

tidal volume (TV) 500 ml

breath rate (BR) 14/min

blood gases

	arterial	central venous
pCO_2	40	46
pO_2	100	40

mm Hg

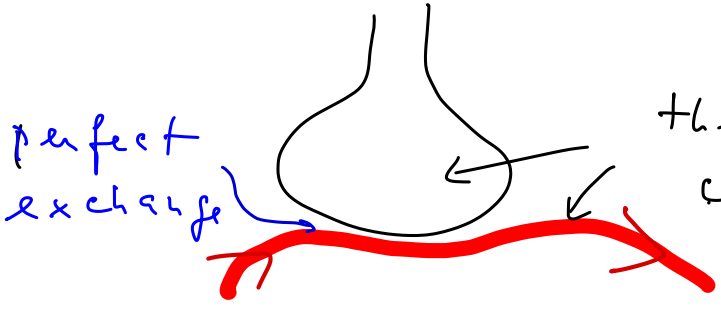
≈ alveolar

(! hypoxia
hypoxemia
hypo(hyper)capnia)

Under normal situation the gas exchange on alveolo-capillary membrane is perfect

$\Rightarrow$ composition of arterial and alveolar gasses is the same

$$p_A O_2 = p_a O_2 \quad ; \quad p_A CO_2 = p_a CO_2$$



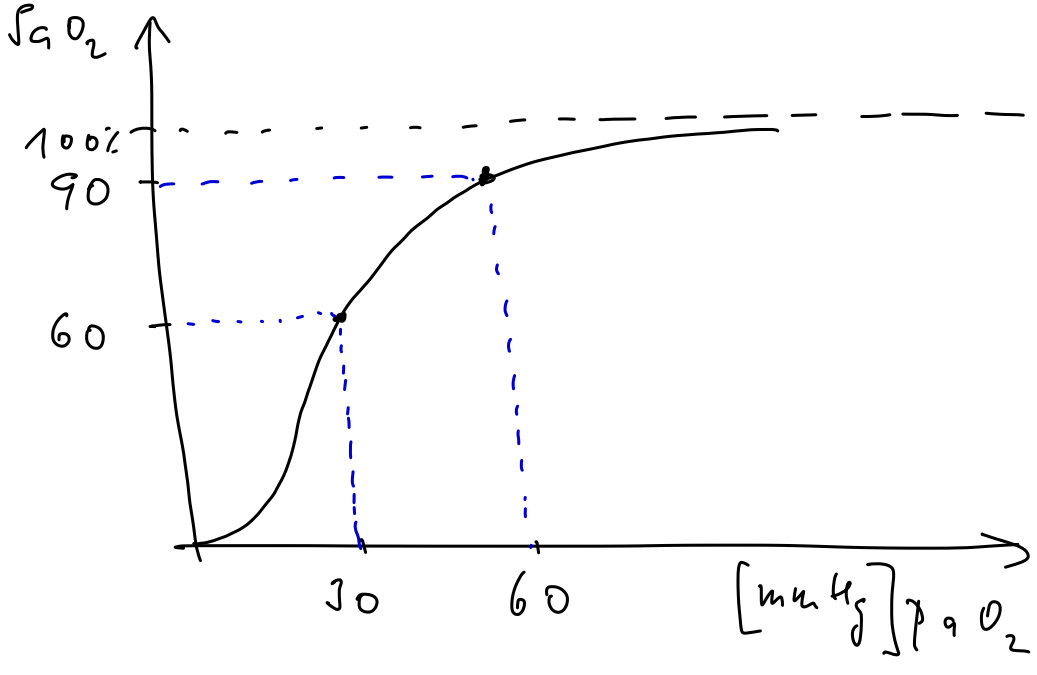
Further we will "desire" 5 mecha-nisms of respiratory insufficiency

Definition of respir. insufficiency

- $paO_2 < 60$ mmHg ($\sim SaO_2 < 90\%$)
- $paCO_2 > 50$ mmHg

Saturation curve of hemoglobin

- Mile 30 - 60 - 90



1) Fracture of 3 ribs

→ pain on inspirium

→ superficial breathing (= low TV)

→ compensatory tachypnoe

$TV \cdot BR$ = minute ventilation ($\dot{V}$)

normal: $500 \cdot 14 = 7000$ ml/min

Fx of ribs: $250 \cdot 28 = 7000$ ml/min

⇓

total ventilation is the same

Is there any difference between both situations?

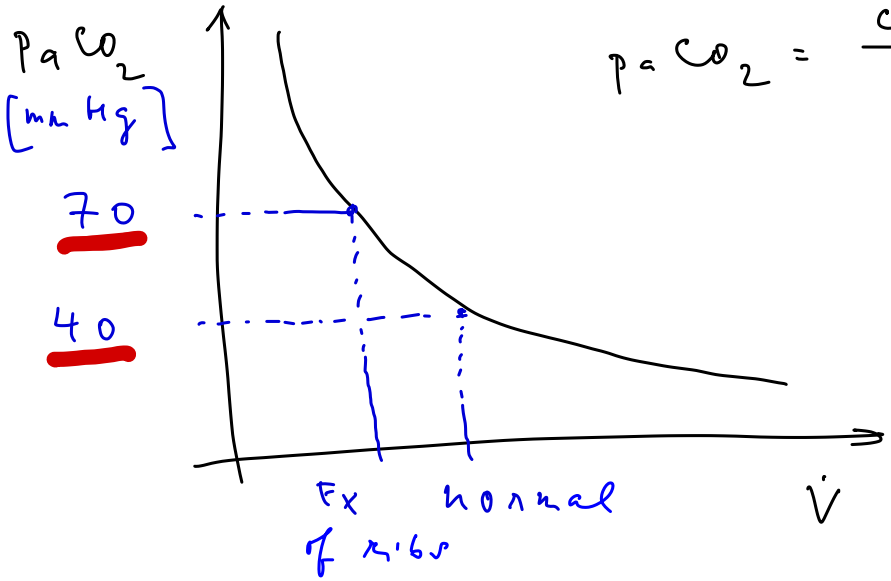
We must consider anatomic dead space, approx. 150 ml.

Real alv. ventilation is:

normal: $(500 - 150) \cdot 14 = \underline{4900}$ ml/min

Fx of risk: $(250 - 150) \cdot 28 = \underline{2800}$ ml/min

relationship between alveolar ventilation and $paCO_2$



$$paCO_2 = \frac{const}{\dot{V}}$$

In case of m/b Fx the patient has hypercapnia. This mechanism is called

① global alveolar hypoventilation

= type 2 respiratory insufficiency (= ventilatory failure = hypercaphic failure)

results:

1) ↑ $paCO_2$
2) ↓ paO_2

examples of causes

muscle weakness, myasthenia gravis, polyneuropathy, opiates, pathology of CNS, obstruction of airways, unstable chest wall, pain...

further mechanisms result usually in

Type 1 respir. insuff.

1) ↓ O_2 but 2) normal or even lower CO_2

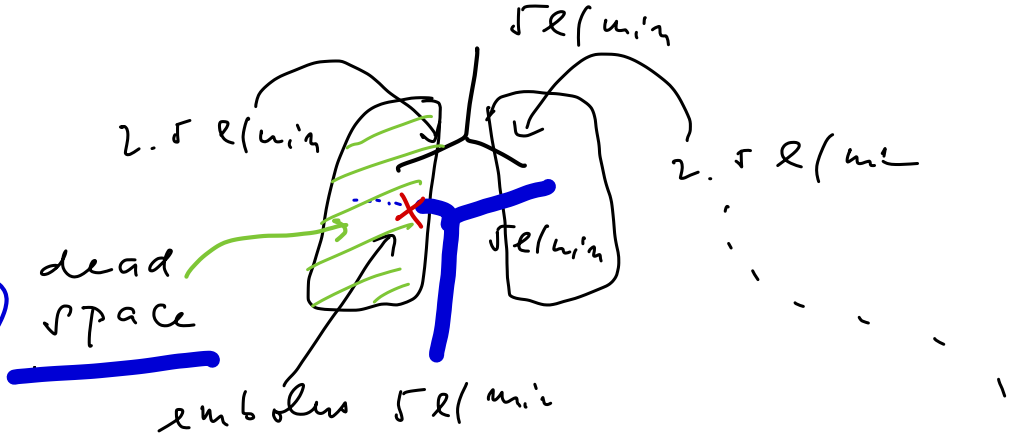
(= hypoxemic failure
= oxygenation dysfunction)

approaching

Type 1 – lung is ill

Type 2 – lung is healthy,
– or extreme illness of lung
but other respiratory components are ill

2) Pulmonary embolism



Right lung is ventilated, but not perfused with blood

= dead space

→ wasted ventilation

→ actually hyperventilation (2.5 l/min)

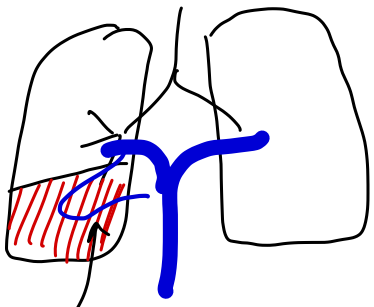
⇒ ↑ eCO_2

↓ O_2

⇒ compensatory hyperventilation

(Why does hyperventilation decrease saO_2, although total O_2 delivery is sufficient?)

3) Lobar pneumonia



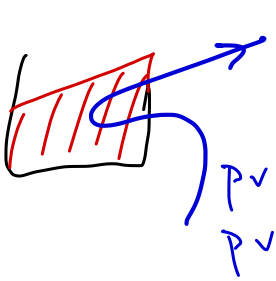
no gas exchange

preserved perfusion

(vs. extrapulmonary shunt ... e.g. open foramen ovale + pulmonary hypertension)

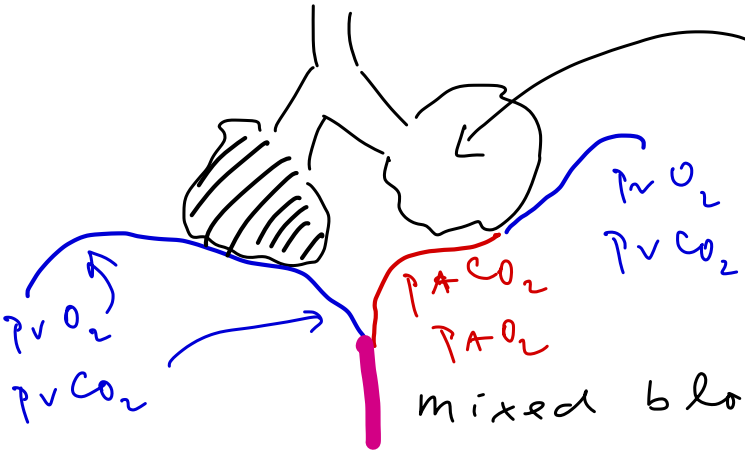
(right-to-left)

= intrapulmonary shunt (3)



$P\bar{v}O_2$
$P\bar{v}CO_2$
=
$P\bar{v}O_2$
$P\bar{v}CO_2$

← both inflow and outflow is venous blood



compensatory hyperventilation is possible

$P\bar{v}O_2$
$P\bar{v}CO_2$

P_ACO_2
P_AO_2

$P\bar{v}O_2$
$P\bar{v}CO_2$

mixed blood

$P\bar{v}CO_2 > PCO_2 > P_ACO_2$

$P\bar{v}O_2 < PO_2 < P_AO_2$

causes : pneumonia, severe lung edema, atelectasis, intra alveolar bleeding...

Is it possible to compensate for intrapulmonary shunt?

1) hyperventilation of healthy alveoli
   → will decrease CO_2
   → does not change O_2 (why?)

2) delivery of external oxygen
   → does not change O_2 (why?)
   → does not change CO_2 (why?)

⇒ actually compensation is almost impossible ⇒ necessary to treat the cause

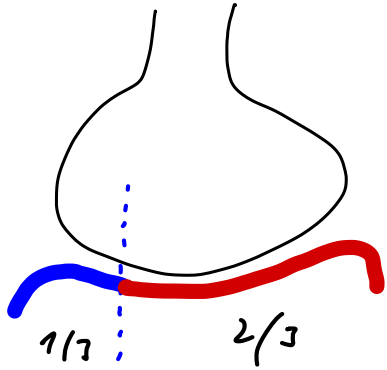
(pneumonia → ATB
atelectasis → bronchoscopy, drainage of pleural effusion)

4) Pulmonary fibrosis

→ widened alveolo-capillary membrane

→ impaired diffusion (4)

- less important mechanism



normally, the gas concentrations in alveoli and blood are equilibrated in 1/3 of the capillary length

⇓

big reserve

- problem more for O_2 than for CO_2
- exertional dyspnea and hypoxemia

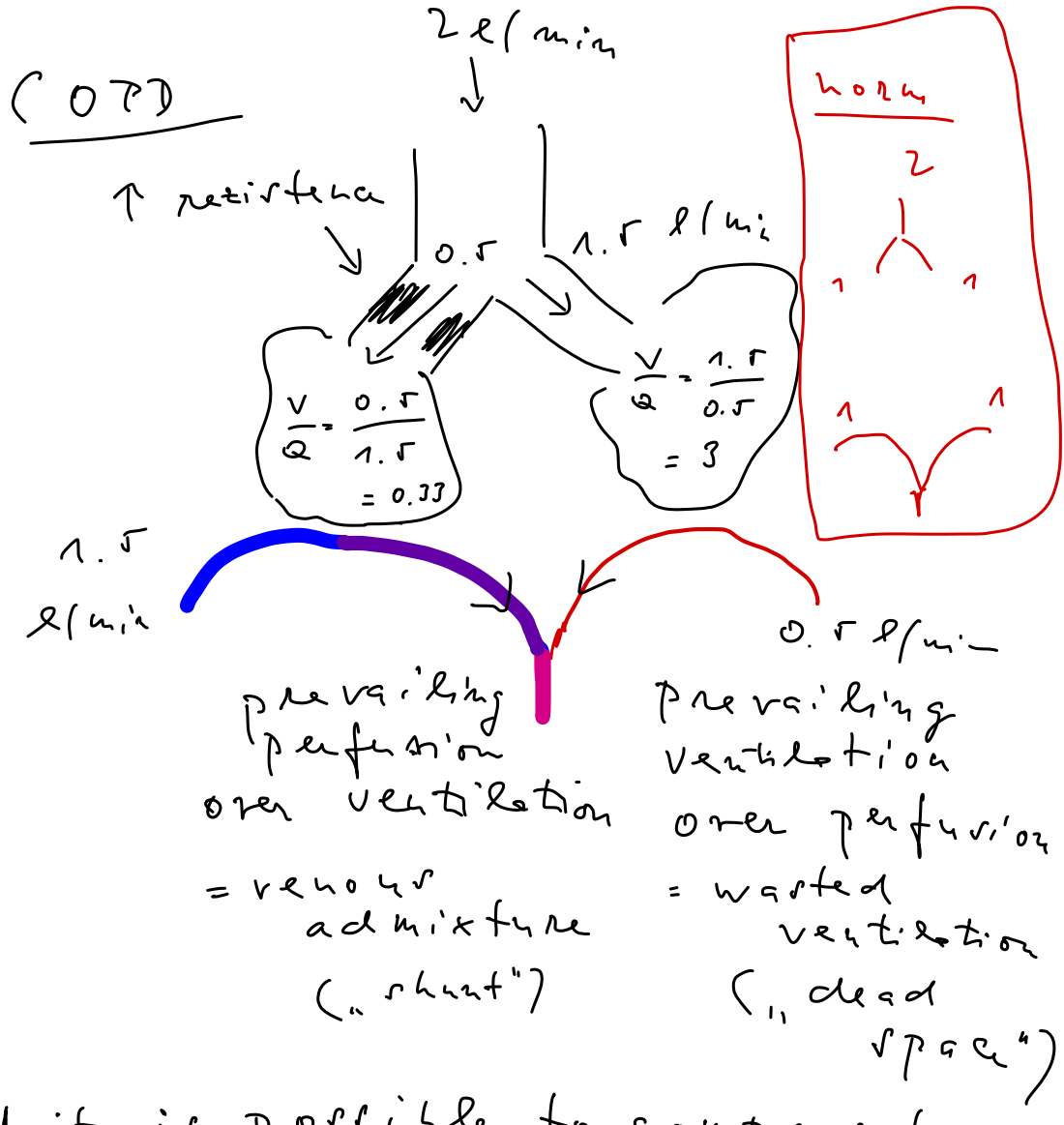
5) COPD, asthma, ♡ failure

- inhomogeneity of lung impairment
→ somewhere lower ventilation ($\dot{V}$)
somewhere lower perfusion ($\dot{Q}$)

(5)
⇒ Ventilatory–perfusion mismatch
(= $\dot{V}/\dot{Q}$ mismatch)

- the most important mechanism
- shunt and dead space are extreme variants

- hypoxic pulmonary vasoconstriction
optimizes $\dot{V}/\dot{Q}$ ratios
→ without h.p.v. the lung function would be impaired even in healthy lungs



! it is possible to compensate for it with hyperventilation or with a delivery of (why ?) oxygen