# General pathophysiology of the respiratory system

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- 2. Control of ventilation
- 3. A survey

# 1. Gas exchange in the lungs

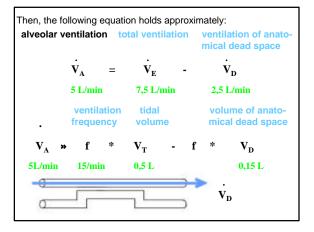
#### Measurement of alveolar ventilation

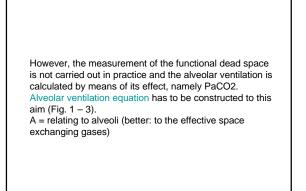
Alveolar ventilation is defined as a flow of respiratory gases through perfectly functioning (=exchanging gases) alveoli (it should be better called: "gas exchange ventilation", as not all alveoli work perfectly).

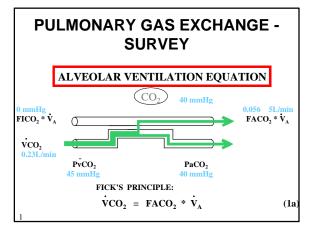
Alveolar ventilation = total ventilation minus the ventilation of the anatomical dead space, if all alveoli work approximately ideally (young healthy lungs). A fraction of alveoli exchange gases suboptimally in pathologically changed lungs. In this case, one should measure

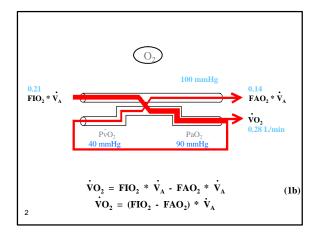
- the anatomical dead space
- plus the alveolar dead space = completely non-perfused alveoli plus the aggregative effect of badly perfused alveoli,

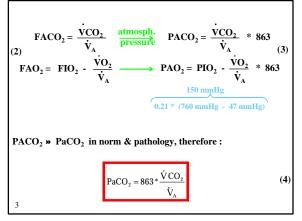
together s.c. functional dead space. However, the f.d.s. could be calculated from a relationship between the partial pressures of  $CO_2$  in the expired gass and in the arterial blood

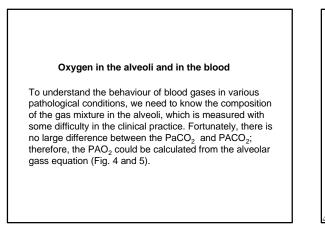


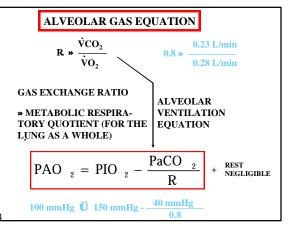


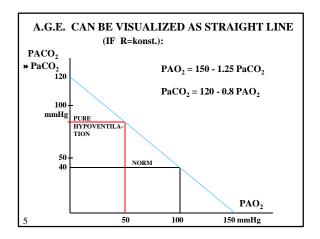


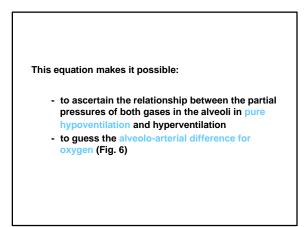


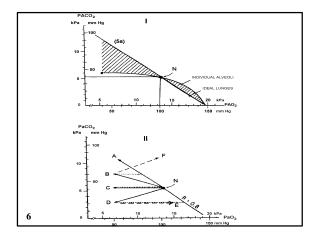








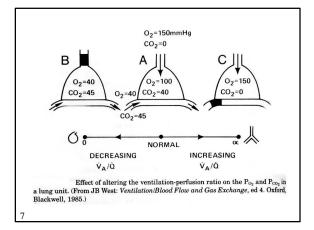


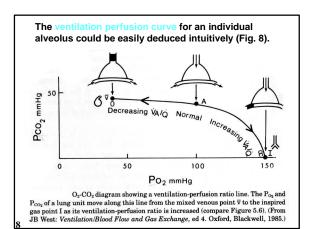


Enhanced  $P[A-a]O_2$  implies a damage of pulmonary parenchyma, it is not enhanced in pure hypoventilation. It follows from the equation that serious hypoxemia could be produced even under normal  $P[A-a]O_2$  in heavy hyperkapnia

### Ventilation perfusion inequality

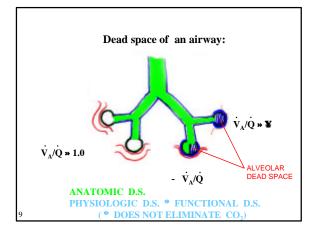
The consideration leaned so far on a unicompartmental model of the lungs. However, the main pathophysiological mechanism of the deranged gass exchange in reality is the ventilation perfusion inequality and this cannot be grasped by this model. VPI is a deviation from the optimum relationship between ventilation and perfusion of the whole lung, its various regions up to the individual alveoli. The ventilation perfusion ratio of an individual alveolus determines the composition of the gas mixture in it (Fig. 7)

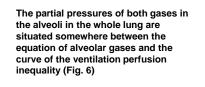


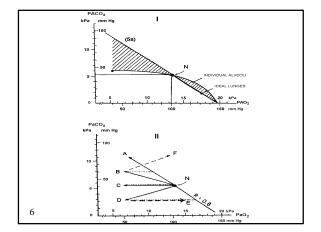


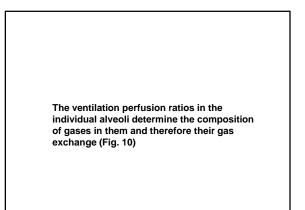
This curve could be situated into the coordinates of the alveolar gases, as the partial pressures in the alveoli depend on the ventilation perfusion ratio in the individual alveoli The functional dead space of the lungs is always

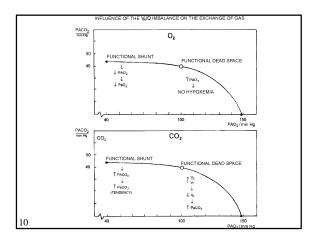
larger than the anatomical dead space, possibly much larger under pathological conditions (Fig. 9)

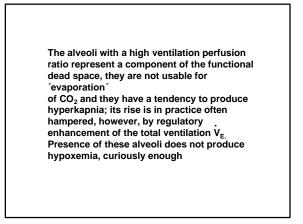






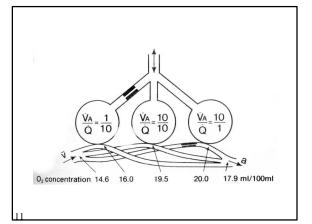






The alveoli with a low ventilation perfusion ratio represent a component of the functional shunt. Therefore, they produce hypoxemia; their mild tendency to producing hyperkapnia is normally overwhelmed by hyperventilation. The functional shunt my be calculated from the difference between  $CaO_2$  and  $CvO_2$  and this measurement is of clinical value

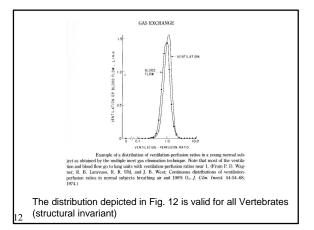
Fig. 11 illustrates in detail the influence of the ventilation perfusion inequality in the individual alveoli on the oxygen exchange

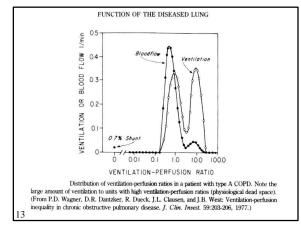


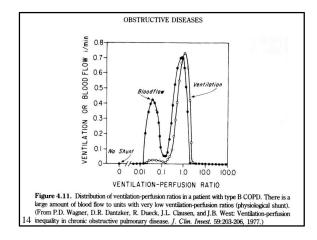
The picture explains (in combination with the known form of the oxygen dissociation curve):

- why PaO<sub>2</sub> declines in the shunt situations (in the low  $\dot{V}_{\rm A}/\dot{Q})$  alveoli)
- why this decline is not influenced substantially by hyperventilation,
- why the rise of FIO<sub>2</sub> does not help too much

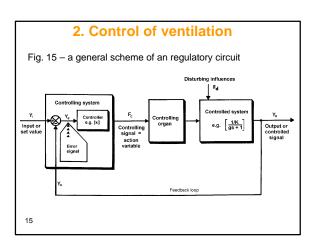
Regarding  $CO_2$ , there is a direct proportionality between its partial pressure in the alveoli and the saturation of the blood, thus the hyperventilation is very effective in lowering PaCO<sub>2</sub> To understand the pathophysiology of the disturbances of the gas exchange, we should know how many alveoli in the individual lung have this or that value of  $\dot{V}_A/Q$ . It is practicable by an experimental method called "multiple inert gas elimination technique" (MIGET), Fig. 12 - 14

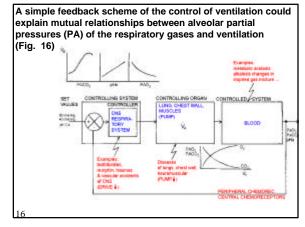


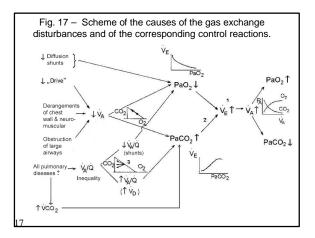


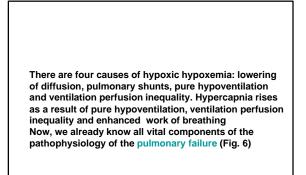


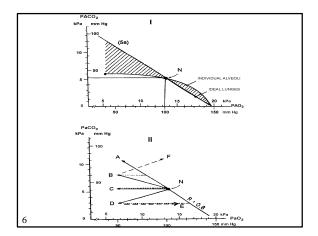
The magnitude of the ventilation perfusion inequality could only be measured with difficulties in practice (there is no encompassing method for a high and a low ventilation perfusion ratio); therefore, ventilation-perfusion inequality is guessed by exclusion of all other possible causes of gas exchange disturbances, e.g., of pure hypoventilation











Clinical ca (Fig. 18)	su	suistics of the gas exchange disturbance in the lungs								
	S E X	A G E	ANAMNESIS	PaO <sub>2</sub>	PaCO <sub>2</sub>	PAO <sub>2</sub>	P (A-a)O2	CONCUSION		
	ę	21	BARBITURA- TES	40 mmHg	80	143 - - 80 * 1.2 = <u>47</u>	47-40=	HYPOVENTILAT. NO ABNORMA- LITY OF PULM. PARENCHYMA THERAPY : FIO <sub>2</sub> MECHANICAL VENTILATION		
	õ	42	GAGGED EATING OLIVES RTG ATE- LECTASIS OF ONE LUNG	41	32	143 - - 32 * 1.2 = 105	105 - 41 =	PARENCHYAL DISTURBANCE (ATELECTASIS ↓ 		
	Ó	63	SMOKER CHROMIC DYSPNEA	50	39	143 - - 39 * 1.2= = 96	96-30= = <u>46</u>	DISTURBANCE $V_{A}$ MALDISTR		
18								Q THER.: ↑FIO2		

Hypercapnia itself loweres (according to the alveolar gas equation) $PAO_2$ and therefore $PaO_2$ (Fig. 19)								
PACO <sub>2</sub>	PAO <sub>2</sub>	PaO <sub>2</sub>	P(A-a)O <sub>2</sub>					
Norm. 40mHg	100	90	10					
Нурегс. 60 —	$ \begin{array}{c} 100 \\ \longrightarrow \\ 80 \end{array} $	60 ← ↓ 40	— 40 ← path. (40)					
<b>Hyperc. 100</b> —	$ \begin{array}{c} 100 \\ \hline \\ 100 \\ \hline \\ 25 \end{array} $	90     15!	10 norm.					

## 3. A survey

- Pathophysiology of the gas exchange should help us to evaluate the state of the respiratory functions in a patient. We are interested in:
- Alveolar ventilation which could be guessed by means of PaCO<sub>2</sub> (regardless of our ignorance of the situation in the individual alveoli). The task is solved by means of alveolar ventilation equation

$$\dot{\mathbf{V}}_{\mathrm{A}} = 863 * \dot{\mathbf{V}}_{\mathrm{CO}_2} / \text{PaCO}_2,$$

e.g., in the normal conditions  $863 * 0.23 \text{ Lmin}^{-1}$ , 40 mmHg = 5 L min<sup>-1</sup>.

2. The value of  $PAO_2$ , to be able to calculate the alveolararterial oxygen difference  $P[A-a]O_2$ . The task is solved by means of alveolar gass equation calculated from ventilation equation:

e.g., in the normal conditions  $150mmHg-40\ mmHg\_0,8=100\ mmHg.$ 

Rise of the  $P[A-a]O_2$  value testifies to the derangement of the pulmonary parenchyma. If the patient only hypoventilates ("pure" hypoventilation), his partial pressures of the respiratory gases correspond to the alveolar gas equation and the  $P[A-a]O_2$  is low

As most important pathophysiological mechanism of the gas exchange disturbances, the ventilation perfusion unequality has been recognized, producing the functional dead space and the functional shunt. We do not have, however, any direct indicator of the ventilation perfusion inequality in the clinical practice and we must deduce it from other parameters of the gas exchange. A total shunt reacts badly to hyperventilation and to the enhancement of the oxygen fraction in the inspired gas. Inhalation of 100% oxygen eliminates the influence of the V<sub>A</sub>/Q inequality totally. Hyperkapnia itself may produce hypoxemia, according to the alveolar gas equation