

## 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 $\mathrm{CO}_{2}$ in the expired gass and in the arterial blood

However, the measurement of the functional dead space is not carried out in practice and the alveolar ventilation is calculated by means of its effect, namely PaCO 2 .
Alveolar ventilation equation has to be constructed to this aim (Fig. $1-3$ ).
A = relating to alveoli (better: to the effective space exchanging gases)

Then, the following equation holds approximately: alveolar ventilation total ventilation ventilation of anatomical dead space


## PULMONARY GAS EXCHANGE SURVEY




## Oxygen in the alveoli and in the blood

To understand the behaviour of blood gases in various pathological conditions, we need to know the composition of the gas mixture in the alveoli, which is measured with some difficulty in the clinical practice. Fortunately, there is no large difference between the $\mathrm{PaCO}_{2}$ and $\mathrm{PACO}_{2}$; therefore, the $\mathrm{PAO}_{2}$ could be calculated from the alveolar gass equation (Fig. 4 and 5).
A.G.E. CAN BE VISUALIZED AS STRAIGHT LINE (IF R=konst.):

(2)

(3)


$\mathrm{PACO}_{2} \approx \mathrm{PaCO}_{2}$ in norm \& pathology, therefore :

$$
\mathrm{PaCO}_{2}=863 * \frac{\dot{\mathrm{~V}} \mathrm{CO}_{2}}{\ddot{\mathrm{~V}}_{\mathrm{A}}}
$$



This equation makes it possible:

- to ascertain the relationship between the partial pressures of both gases in the alveoli in pure hypoventilation and hyperventilation
- to guess the alveolo-arterial difference for oxygen (Fig. 6)


Enhanced $\mathrm{P}[\mathrm{A}-\mathrm{a}] \mathrm{O}_{2}$ 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 $\mathrm{P}[\mathrm{A}-\mathrm{a}] \mathrm{O}_{2}$ 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)

The ventilation perfusion curve for an individual alveolus could be easily deduced intuitively (Fig. 8).

$\mathrm{O}_{2}-\mathrm{CO}_{2}$ diagram showing a ventilation-perfusion ratio line. The $\mathrm{P}_{\mathrm{O}_{2}}$ and $\mathrm{P}_{\mathrm{CO}_{2}}$ of a lung unit move along this line from the mixed venous point $\overline{\mathrm{v}}$ to the inspired gas point I as its ventilation-perfusion ratio is increased (compare Figure 5.6). (From JB West: Ventilation/Blood Flow and Gas Exchange, ed 4. Oxford, Blackwell, 1985.)

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 partial pressures of both gases in the alveoli in the whole lung are situated somewhere between the equation of alveolar gases and the curve of the ventilation perfusion inequality (Fig. 6)


The ventilation perfusion ratios in the individual alveoli determine the composition of gases in them and therefore their gas exchange (Fig. 10)


The alveoli with a high ventilation perfusion ratio represent a component of the functional dead space, they are not usable for evaporation'
of $\mathrm{CO}_{2}$ and they have a tendency to produce hyperkapnia; its rise is in practice often hampered, however, by regulatory enhancement of the total ventilation $\mathrm{V}_{\mathrm{E}}$. Presence of these alveoli does not produce hypoxemia, curiously enough

The alveoli with a low ventilation perfusion ratio represent a component of the . 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 $\mathrm{CaO}_{2}$ and $\mathrm{CvO}_{2}$ 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


Regarding $\mathrm{CO}_{2}$, 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 $\mathrm{PaCO}_{2}$ 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 $\dot{V}_{A} / \dot{Q}$. It is practicable by an experimental method called ,,multiple inert gas elimination technique" (MIGET), Fig. 12-14

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

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




Figure 4.11. Distribution of ventilation-perfusion ratios in a patient with type B COPD. There is a large amount of blood flow to units with very low ventilation-perfusion ratios (physiological shunt) (From P.D. Wagner, D.R. Dantzker, R. Dueck, J.L. Clausen, and J.B. West: Ventilation-perfusion 14 inequality in chronic obstructive pulmonary disease. J. Clin. Invest. 59:203-206, 1977.)

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

## 2. Control of ventilation

Fig. 15 - a general scheme of an regulatory circuit


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A simple feedback scheme of the control of ventilation could explain mutual relationships between alveolar partial pressures (PA) of the respiratory gases and ventilation (Fig. 16)


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Fig. 17 - Scheme of the causes of the gas exchange disturbances and of the corresponding control reactions.


There are four causes of hypoxic hypoxemia: lowering of diffusion, pulmonary shunts, pure hypoventilation and ventilation perfusion inequality. Hypercapnia rises as a result of pure hypoventilation, ventilation perfusion inequality and enhanced work of breathing Now, we already know all vital components of the pathophysiology of the pulmonary failure (Fig. 6)


Clinical casuistics of the gas exchange disturbance in the lungs


2. The value of $\mathrm{PAO}_{2}$, to be able to calculate the alveolararterial oxygen difference $\mathrm{P}[\mathrm{A}-\mathrm{a}] \mathrm{O}_{2}$. The task is solved by means of alveolar gass equation calculated from ventilation equation:

$$
\mathbf{P A O}_{2} \approx \mathbf{P I O}_{2}-\mathbf{P a C O}_{2} / \mathbf{R}
$$

e.g., in the normal conditions $150 \mathrm{mmHg}-40 \mathrm{mmHg} \div 0,8=$ 100 mmHg .

Rise of the $\mathrm{P}[\mathrm{A}-\mathrm{a}] \mathrm{O}_{2}$ 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 $\mathrm{P}[\mathrm{A}-\mathrm{a}] \mathrm{O}_{2}$ is low

## 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:

1. Alveolar ventilation which could be guessed by means of $\mathrm{PaCO}_{2}$ (regardless of our ignorance of the situation in the individual alveoli). The task is solved by means of alveolar ventilation equation

$$
\dot{\mathbf{V}}_{\mathrm{A}}=863 * \dot{\mathrm{~V}} \mathrm{CO}_{2} / \mathrm{PaCO}_{2},
$$

e.g., in the normal conditions $863 * 0,23 \mathrm{~L} \mathrm{~min}^{-1} \div 40 \mathrm{mmHg}$ $=5 \mathrm{~L} \mathrm{~min}^{-1}$.

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 $\dot{V}_{A} / \dot{Q}$ inequality totally. Hyperkapnia itself may produce hypoxemia, according to the alveolar gas equation

