VENTILATION–PERFUSION RATIOS AND THE PULMONARY SHUNT

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Tests were carried out on 63 healthy persons aged 17-29 years, in the sitting position. The following parameters of lung ventilation were determined: Vd, phys/Vt = 30±0.6%, $AaDO_2$ = 10±0.9 mm Hg, $aADCO_2/p_aCO_2$ was equal to the end-expiratory pCO_2, i.e., to Pet,CO_2 = 5.0±0.4 mm Hg, $\dot{V}a/\dot{Q}t$ (air) = 2.3±0.3%, $\dot{Q}s/\dot{Q}t$ (O_2) = 1.8±0.2%. For the lungs as a whole $\dot{V}A/\dot{Q}c$ was normal (0.86±0.30). No difference in $\dot{V}A/\dot{Q}c$ could be found between men and women. The main contribution to the development of $\dot{Q}va/\dot{Q}t$ in healthy subjects belongs to the anatomical shunt and perfusion of unventilated alveoli.

KEY WORDS: ventilation–perfusion ratio; pulmonary shunt; O_2 shunt.

Local inequality of ventilation–perfusion ratios ($\dot{V}A/\dot{Q}c$) has been shown to be characteristic of healthy persons [3, 5, 9]. However, the closeness of this relationship, the mechanisms of its development, and whether there are differences between men and women are questions which have not yet been answered [4, 5, 10]. In an attempt to answer them, an investigation of $\dot{V}A/\dot{Q}c$ and the pulmonary shunt was undertaken in healthy young people.

EXPERIMENTAL METHOD

Tests were carried out on 63 healthy people aged 17-29 years (mean 22±0.3 years) 2 h after a light breakfast, under conditions of relative rest and in the sitting position. The acid–base balance of the blood and blood gases (p_aCO_2 and p_aO_2) were determined on the micro-Astrup apparatus and pO_2 by a Clark's electrode. The concentrations of CO_2 and O_2 in the expired air were investigated on the GUM-2 capnograph and MMG-7 oxygen analyzer. The minute respiratory volume was measured on the SG-1M spirograph. The following parameters were calculated during air breathing: 1) p_AO_2, or pO_2 in the alveolar air, was determined by the equation for alveolar air; by subtracting p_aO_2 from p_AO_2 the alveolo-arterial pO_2 gradient ($AaDO_2$) was determined; the value of pCO_2 in the final portion of expired air (Pet,CO_2) was taken as the value of pCO_2 in the alveolar air (p_ACO_2), and the difference between p_aCO_2 and p_ACO_2 was regarded as the arterio-alveolar pCO_2 gradient ($aADCO_2$); 3) the difference between p_aCO_2 and Pet,CO_2 after deep expiration was taken as the minimal pCO_2 gradient ($aADCO_2$)*, 4) the physiological dead space (Vd, phys) was calculated by Bohr's equation in Enghoff's modification, a correction being introduced for the dead space of the valve; 5) the anatomical dead space (Vd, anat) was calculated by Bohr's equation, Pet,CO_2 during quiet breathing being taken as p_aCO_2; the alveolar dead space (Vd, alv) was found by the equation Vd, alv = Vd, phys − Vd, anat; values calculated by this method for Vd, anat (Vd1) and for Vd, alv (Vd2) are higher than the true values (see the remarks on determination of $aADCO_2$); 7) $\dot{V}A$, the alveolar ventilation, was calculated on the basis of Vd, phys; 8) Qc, the volume of the capillary blood flow, was taken to be equal to the cardiac output ($\dot{Q}t$), measured by the rebreathing method with CO_2; $\dot{V}A/Qc$ is the ratio of ventilation to blood flow for the lungs as a whole; 10) $\dot{Q}va/\dot{Q}t$, the pulmonary shunt or venous admixture, during air breathing, was calculated in the usual way by the equation $\dot{Q}va/\dot{Q}T = (Cc'O_2 - Ca,O_2)/(Cc'O_2 - C\bar{v},O_2)$, where $\dot{Q}va/\dot{Q}T$ is the volume of admixed venous blood as a percentage of the cardiac output, and $Cc'O_2$, Ca,O_2 and $C\bar{v},O_2$ represent the O_2 concentration in the end-capillary, arterialized, and mixed venous blood respectively; when calculating $Cc'O_2$ it was assumed that pO_2 in the end-capillary blood ($pc'O_2$) was equal to p_AO_2; $C\bar{v},O_2$ was calculated from Ca,O_2, for which the arteriovenous dif-

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TABLE 1. $\dot{V}A/\dot{Q}c$ and Pulmonary Shunt in Healthy Young People

Sex	A_aDO_2	$aADCO_2$	$aADCO_2$*	Vd,phys	Vd,phys/Vt	Vd 1	Vd,1/Vt	Vd 2	Vd,2/Vt	$\dot{V}A$	$\dot{Q}c$	$\dot{V}A/\dot{Q}c$	$\dot{Q}va/\dot{Q}T$	100% O_2	
														$AaDO_2$	$\dot{Q}c/\dot{Q}r$
M.	9±1 n—30	5,4±0,6 n—32	—0,5±1,5 n—32	180±8 n—38	29±0,9 n—38	144±8 n—26	22±1 n—26	46±4 n—26	7±0,5 n—26	5,9±0,1 n—34	7,2±0,3 n—14	0,84±0,05 n—14	2,0±0,4 n—20	32±5 n—15	1,9±0,3 n—15
F.	10±1 n—19	4,6±0,7 n—18	0,8±0,6 n—18	143±10 n—25 x	31±1 n—25	103±8 n—18 x	22±1 n—18	43±6 n—18	9±1,0 n—18	4,7±0,15 n—25 x	5,4±0,17 n—18 x	0,88±0,04 n—18	2,5±0,3 n—17	27±15 n—3	1,6±0,9 n—3
Both sexes	10±0,9 n—49	5,0±0,4 n—50	0,2±0,9 n—50	162±6 n—63	30±0,6 n—63	124±6 n—44	22±07 n—44	45±3 n—44	8±0,7 n—44	5,3±0,1 n—59	6,3±0,2 n—32	0,86±0,03 n—32	2,3±0,3 n—37	30±4 n—18	1,8±0,2 n—18

Legend. $AaDO_2$) Alveolo-arterial pO_2 gradient (mm Hg); $aADCO_2$) arterio-alveolar pCO_2 gradient, during calculation of which it was assumed that p_aCO_2 = Pet,CO during quiet breathing (mm Hg); $aDCO_2$*) the same, but Pet,CO_2 during deep breathing (in mm Hg) was taken to be the same as p_aCO_2; Vd,phys) physiological dead space (in mm Hg); Vd,phys/Vt) ratio of Vd,phys to respiratory volume (RV) (in %); Vd 1) anatomical dead space (in ml), Vd 1/Vt) Vd 1 as a percentage of RV; Vd 2) alveolar dead space (in ml); Vd 2/Vt) Vd 2 as a percentage of RV; $\dot{V}a$) alveolar ventilation (in liters/min); Qc) capillary blood flow (in liters/min); $\dot{Q}A/\dot{Q}c$) ratio of ventilation to blood flow for the lungs as a whole (in units); $\dot{Q}va/\dot{Q}t$) venous admixture as a percentage of cardiac output; 100% O_2) values during breathing pure oxygen; $AaDO_2$) alveolo-arterial pO_2 gradient (mm Hg); $\dot{Q}c/\dot{Q}t$) right-left shunt as a percentage of cardiac output. Only significant differences between men and women are shown.

ference in O_2 concentration $[(a-\bar{v})O_2]$ was first found, by calculation from the venoarterial difference in CO_2 concentration $[(\bar{v}-a)CO_2]$, determined by rebreathing with CO_2, when $(a-\bar{v})O_2 = (\bar{v}-a)CO_2/R$, where R is the respiratory quotient (the ratio between CO_2 excreted per minute and O_2 consumed, i.e., $\dot{V}CO_2/\dot{V}O_2$ (it was assumed that R in the blood is equal to R in the expired air [7]). The value of the venous admixture is made up of the following sources: 1) the shunt of venous blood via venoarterial anastomoses (bronchial and Thebesius' and other natural anastomoses) — the anatomical (vascular) shunt; 2) the blood flow along capillaries of unventilated alveoli, in which $\dot{V}A/\dot{Q}c$ is zero — the capillary or parenchymatous shunt; 3) perfusion of alveoli with a low value of $\dot{V}A/\dot{Q}c$, but not down to zero — distribution (in the narrow meaning) disturbances; 4) losses of oxygenation through obstacles to O_2 diffusion. $\dot{Q}s/\dot{Q}t$ — the sum of the anatomical and capillary shunts, was measured by an O_2 method; to reduce the losses of pO_2 as much as possible, blood from the ulnar (radial) artery was directed through a needle and adaptor directly into the cuvette of the pO_2 electrode [8]; the oxygen shunt was calculated by the equation $\dot{Q}s/\dot{Q}t = (AaDO_2 \times 0.0031)/[(AaDO_2 \times 0031) - (a-\bar{v})O_2]$, where $\dot{Q}s/\dot{Q}t$ is the magnitude of the right-left shunt during breathing of pure oxygen, $AaDO_2$ is the alveolo-arterial pO_2 gradient during breathing of pure oxygen, the number 0.0031 is the coefficient of sulubility of O_2 in blood, and $(a-\bar{v})O_2$ is the arteriovenous difference in O_2 concentration during oxygen breathing (which was assumed to be equal to that during air breathing). Details of the methods used are described in the literature [1-4, 6]. The results were subjected to statistical analysis and are given in Table 1.

EXPERIMENTAL RESULTS

It will be clear from the results in Table 1 that slight inequality of distribution of $\dot{V}A/\dot{Q}c$ was found in the healthy young subjects. On the one hand, hypo- and nonperfused alveoli were functioning, as shown by the difference between Vd,phys and Vd,anat (the alveolar dead space). On the other hand, zones in which $\dot{V}A/\dot{Q}c$ was low or reduced to zero evidently existed. The blood flow along the capillaries of such alveoli and through the anatomical shunt form the venous admixture (ineffective blood flow). Moreover, judging from the values of $\dot{Q}va/\dot{Q}t$ and $\dot{Q}s/\dot{Q}t$, the venous admixture is formed almost entirely on account of the anatomical shunt and perfusion of unventilated alveoli. The presence of hypoventilated alveoli can be judged only indirectly, from the value of $aADCO_2$, which disappears after deep expiration, when $aADCO_2$* is virtually equal to zero. However, considering the insignificant contribution of perfusion of hypoventilated alveoli to the development of $\dot{Q}va/\dot{Q}t$, there is reason to suppose that $aADCO_2$ is formed mainly on account of hyperventilated alveoli.

Since losses of pO_2 because of hindrances to O_2 diffusion through the alveolo-capillary membrane are negligible and do not exceed 1 mm Hg in the structure of $AaDO_2$ during air breathing [6], it is reasonable to assume that $AaDO_2$ and the venous admixture in healthy young people are formed on account of the shunting of venous blood through the anatomical shunt and perfusion of unventilated alveoli. The main contribution belongs to the anatomical shunt — its extrapulmonary component [9].

For the lungs as a whole $\dot{V}A/\dot{Q}c$ was normal. This is evidence of the local character of the irregularity of distribution of $\dot{V}A/\dot{Q}c$ discovered.

The shunting of venous blood explains why even in healthy subjects oxygenation of arterial blood does not reach the possible upper limit. For instance, the results showed that p_aO_2 in healthy men was 94±0.9 mm Hg and in women 93±0.8 mm Hg; physiological arterial hypoxemia thus exists in healthy persons.

No significant differences were found between men and women as regards the principal parameters $\dot{V}A/\dot{Q}c$. Differences in the actual values of Vd,phys, Vd 1 (anatomical dead space), $\dot{V}A$ and $\dot{Q}c$ are attributable to anthropometric differences between men and women.

The causes and mechanisms of inequality of $\dot{V}A/\dot{Q}c$ in healthy persons are complex. They include the action of gravitation, regional differences in the mechanical properties of the principal structures of the bronchopulmonary apparatus, and certain other factors [3, 5, 6, 10]. The basic role in the regulation of $\dot{V}A/\dot{Q}c$ is played by pulmonary hypoxic vasoconstriction, hypocapnic bronchoconstriction, and collateral ventilation [3, 5, 6, 9].

*The value of $aADCO_2$ obtained by this method is somewhat overestimated, for the assumption $p_aCO_2 = Pet,CO_2$ is valid only for ideal lungs, and even in healthy subjects $Pet,CO_2 < paCO_2$ because of irregularity of $\dot{V}A/\dot{Q}c$.

The absence of differences between men and women in the character of distribution of $\dot{V}A/\dot{Q}c$ is evidence that the mechanisms of control of this parameter are the same in both cases.

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