QUANTITATIVE ANALYSIS OF DEPENDENCE OF BLOOD ARTERIALIZATION ON INEQUALITY OF VENTILATION–PERFUSION RATIOS

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The results of a comparative quantitative analysis of dependence of blood arterialization on inequality of the ventilation-perfusion ratios for a simple two-component model of the lungs and for a model with a lognormal distribution of $\dot{V}A/\dot{Q}$ are given. The possibility that respiratory failure may exist without significant changes in the arterial blood gas composition is argued.

KEY WORDS: ventilation-perfusion ratio; arterial hypoxemia; respiratory failure.

It can be taken as established that in the presence of marked inequality of ventilation-perfusion ratios oxygenation of blood in the lungs is substantially disturbed. This is observed only in the presence of a sharp decrease in the ventilation-perfusion ratios ($\dot{V}A/\dot{Q}$) in the poorly ventilated parts of the lungs, and if a considerable proportion of the total pulmonary blood flow passes through these parts ($\dot{Q}_1/\dot{Q}$). These results, obtained by means of a two-component model of the lungs (poorly and well-ventilated parts of the lungs) agree with those of direct investigation of healthy persons and patients [2]. The investigations have shown that in subjects with some degree of inequality of ventilation-perfusion ratios the partial pressures of oxygen (p_aO_2) and the degree of oxygenation of the blood (s_aO_2) may remain at virtually the normal level, and that only if the equality of $\dot{V}A/\dot{Q}$ is sharply disturbed may arterial hypoxemia arise [1, 4].

However, a simple two-component model of the lungs enables the true phenomena to be described only approximately. It assumes that the ratio $\dot{V}A/Q$ is equally low in all poorly ventilated parts of the lungs. In reality, in the poorly ventialted parts of the lungs there may be a continuous gradation of reduced values of $\dot{V}A/\dot{Q}$ – from zero to the normal value (0.86). In the present investigation all calculations were made with this fact in mind.

EXPERIMENTAL METHOD

Calculations of s_aO_2, p_aO_2, and the other parameters were made for different values of $\dot{V}A/\dot{Q}$ and $\dot{Q}_1/\dot{Q}$ in the poorly ventilated parts of the lungs, within ranges for which significant arterial hypoxemia did not arise. It was assumed that for each discrete mean value of $\dot{V}A/\dot{Q}$ in a poorly ventilated part of the lungs (0, 0.1, 0.2, 0.3, and 0.4) there was a corresponding continuous set of separate parts with values of $\dot{V}A/\dot{Q}$ varying from zero to normal (0.86). The law of distribution of these parts was taken to be lognormal. It was thus assumed that the degree of inequality of the ventilation-perfusion ratios differed in individual poorly ventilated parts of the lungs, and was characterized by a definite value only on average. For comparison, all data (shown in the text "with dispersion") were compared with data obtained with the simple two-component model (stated "without dispersion").

It was assumed that the mean weighted respiratory quotient for expired air from the lungs remains normal (0.79) whatever the disturbance of $\dot{V}A/\dot{Q}$. In accordance with this assumption values of $\dot{V}A/Q$ were calculated for well-ventilated parts of the lungs using data from Rah and Fenn's diagram [3].

All parameters for the poorly ventilated part of the lungs are marked by the index 1, and those for the well-ventilated part by the index 2. Mean values for the lungs as a whole are given without indices.

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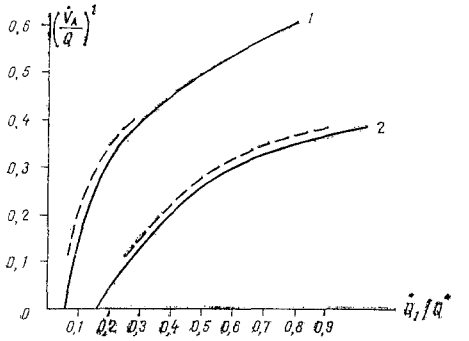

Fig. 1. Blood oxygen saturation as a function of $\dot{V}A/\dot{Q}$ and $\dot{Q}_1/\dot{Q}$. 1) p_aO_2 = 90 mm Hg; s_aO_2 = 96.4%; 2) p_aO_2 = 75 mm Hg; s_aO_2 = 93.7%. Continuous curves represent data from model without dispersion, broken curves data from model with dispersion.

EXPERIMENTAL RESULTS

The principal data are shown selectively in Table 1. It is clear that the values of $(\dot{V}A/\dot{Q})^2$ are increased, reflecting compensatory hyperventilation in the well-ventilated part of the lungs; the smaller the proportion of the total pulmonary blood flow reaching this part of the lungs, the greater this increase. Correspondingly, $p_A^2O_2$ was increased but $p_A^2CO_2$ was reduced. As a result of mixing of blood flowing from the well- and poorly ventilated parts of the lungs, p_aO_2 was reduced but p_aCO_2 was increased, with an increase in the degree of inequality of the ventilation-perfusion ratios. This led to the formation of a well-marked alveolar-arterial pO_2 gradient and a much less marked arterioalveolar pCO_2 gradient. The

TABLE 1. Parameters Showing Dependence of Blood Arterialization on Inequality of Ventilation-Perfusion Ratios

VA/Q^1	0			0,1			0,2			0,4		
$\dot{Q}_1/\dot{Q}$	0,05	0,1	0,15	0,1	0,2	0,3	0,1	0,2	0,4	0,4	0,6	0,8
$\dot{V}A/\dot{Q}^2$	0,86	0,86	0,86	0,89 0,89	0,93 0,92	0,98 0,96	0,90 0,90	0,95 0,94	1,09 1,07	1,09 1,08	1,41 1,37	2,51 2,37
pA^1O_2, pa^1O_2, mm Hg	40	40	40	51 49	51 49	51 49	58 57	58 57	58 57	73 72	73 72	73 72
Sa^1O_2, %	72,0	72,0	72,0	82,5 81,4	82,5 81,4	82,5 81,4	87,2 86,5	87,2 86,5	87,2 86,5	92,9 92,6	92,9 92,6	92,9 92,6
pA^1CO_2, pa^1CO_2, mm Hg	45,0	45,0	45,0	44,9 44,7	44,9 44,7	44,9 44,7	44,6 44,4	44,6 44,4	44,6 44,4	43,9 43,7	43,9 43,7	43,9 43,7
pA^2O_2, pa^2O_2, mm Hg	101	101	101	102 102	103 103	105 104	102 102	104 104	109 108	109 108	116 115	128 127
pA^2CO_2, pa^2CO_2, mm Hg	40,1	40,1	40,1	39,9 39,9	39,6 39,7	39,2 39,4	39,8 39,8	39,5 39,5	38,4 38,6	38,4 38,5	36,3 36,6	31,1 31,7
pAO_2, mm Hg	101	101	101	101 101	102 102	103 102	101 101	102 102	103 103	102 102	103 103	107 106
paO_2, mm Hg	90	80	75	88 87	79 78	73 72	92 91	85 84	75 74	87 86	81 80	77 76
SaO_2, %	96,3	95,0	93,8	96,1 96,0	94,7 94,4	93,2 92,8	96,6 96,6	95,6 95,5	93,7 93,4	95,9 95,8	95,1 94,9	94,1 93,9
$pACO_2$, mm Hg	40,1	40,1	40,1	39,9 40,0	39,7 39,8	39,5 39,6	39,9 40,0	39,7 39,8	39,1 39,2	39,5 39,5	38,6 38,7	36,1 36,5
$paCO_2$, mm Hg	40,4	40,8	41,1	40,4 40,5	40,9 40,9	41,2 41,3	40,3 40,3	40,6 40,6	41,1 41,2	40,5 40,5	40,8 40,8	41,0 41,1

Legend. Data obtained with the simple model are shown in the numerator, those obtained for a model with dispersion in the denominator.

values of s_aO_2 and p_aO_2 were reduced depending on the decrease in $(\dot{V}A/\dot{Q})^1$ and an increase in $\dot{Q}_1/\dot{Q}$. The calculations thus reproduced the typical picture frequently described in clinical physiological investigations of patients with disturbances of ventilation-perfusion ratios.

Comparison of results obtained with the simple model (the values given in the numerator in Table 1) and results obtained by the model with dispersion (given in the denominator) deserves special attention. As Table 1 shows, the differences between these values in many cases were nonexistent or negligible, and in the other cases they were small. The important conclusion can thus be drawn that even a simple two-component model in general approximates all parameters of the gas exchange satisfactorily. It can also be concluded from the fact that although the model with dispersion gave more accurate results, they were not much more accurate, thus the chosen law of distribution is of no great quantitative value and close results can be obtained with the assumption of different laws of distribution.

Curves reflecting the zones of practically normal p_aO_2 (90 mm Hg and over) and the initial degree of arterial hypoxemia (p_aO_2 from 90 to 75 mm Hg), depending on different combinations of reduced values of $\dot{V}A/\dot{Q}$ and $\dot{Q}_1/\dot{Q}$ are given in Fig. 1. Clearly the broken curves plotted from data of the model with dispersion are a little higher than the continuous curves plotted from the model without dispersion. This means that the model with dispersion can detect the possible onset of arterial hypoxemia a little earlier. Quantitatively speaking, however, the difference is very small.

On the basis of all these data the dependence of blood oxygenation on the degree of inequality of ventilation-perfusion ratios can be estimated with great reliability and accuracy. They confirm that the definition of respiratory failure adopted in the USSR, according to which it may occur even in patients whose s_aO_2 is virtually normal, is correct. In fact, in pathological states of the lungs when considerable inequality of ventilation-perfusion ratios arises, as the results of the present investigation show, s_aO_2 may remain close to normal. In such cases respiratory failure may develop without any significant disturbances in the arterial blood gas composition.

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