EFFECT OF BLOOD PRESSURE IN THE PULMONARY ARTERY ON GAS EXCHANGE IN FUNCTIONALLY NONUNIFORM LUNGS

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The presence of areas in the lungs with different values of the ventilation to blood flow ratio ($\dot{V}_A/\dot{Q}$) is one of the main causes of impairment of gas transport in the lungs [2, 7]. In healthy persons nonuniformity of $\dot{V}_A/\dot{Q}$ is due mainly to the influence of gravitation [2, 3, 6]. Nonuniformity of $\dot{V}_A/\dot{Q}$ leads to the appearance of an alveolar-arterial difference in the partial pressure of oxygen (ΔpO_2) and an arterial-alveolar difference in the partial pressure of carbon dioxide (ΔpCO_2), subsequently to be designated as ΔpG [2, 3]. We know that the distribution of $\dot{V}_A/\dot{Q}$ and ΔpG depends essentially on the blood pressure in the pulmonary circulation [3, 6].

The object of this investigation was to determine the blood pressure in the common trunk of the pulmonary artery corresponding to measured values of ΔpG. The experimental conditions included different gas compositions of the mixed venous blood and different levels of ventilation of the lungs.

EXPERIMENTAL METHOD

A mathematical model of functioning of nonuniform lung was used. Only those aspects of functional nonuniformity of anatomically uniform lungs that are due to the action of gravitation with an acceleration of $+1\sigma_z$ were examined – they are considered to be the sole cause of the appearance of ΔpG. The model consists of three parts, describing the blood flow, ventilation, and gas exchange in a single lobule of the lung, and equations for averaging the gas compositions of the arterial blood and alveolar air. The first two parts of the model were described in [1]. The mechanical properties of the vessels of the human pulmonary circulation were determined from data in [4, 5]; they take into account stretching of the vessels in the presence of a positive, and compression of the vessels in the presence of a negative transmural pressure. The distance from the common trunk of the pulmonary artery to the apices of the lungs was assumed to be 18 cm, and the height of the lungs 30 cm. It was assumed that the respiratory volume is constant and that changes in ventilation take place on account of a change in the respiration rate; the distribution of the respiratory volume at different heights of the lungs is constant. The increase in ventilation was thus simulated as an increase in the mean ratio of alveolar ventilation to blood flow for the lungs as a whole $(\dot{V}_A/\dot{Q})_{mean}$ on account of a proportional increase in $\dot{V}_A/\dot{Q}$ in all parts of the lungs. For the calculations the lungs were divided into ten horizontal layers. Using calculated values of ventilation and blood flow in each layer equations describing "ideal" gas exchange [7] were solved numerically, i.e., each layer of the lungs was regarded as a uniform gas-exchange reservoir. Dependence of the concentration of gases in the blood on their partial pressure was determined by the use of known algorithms [7]. The numerical solution of the equations in the model was carried out by computer.

EXPERIMENTAL RESULTS

Qualitative and numerical analysis of the equations of the model showed that nonuniformity of distribution of the blood flow is determined by the blood pressure in the pulmonary circulation. The calculated distribution of the blood flow corresponds to the idea that three West's zones with different types of blood flow exist in the lungs. The numerical investigation of gas exchange was carried out for mean values of the blood pressure in the pulmonary artery (p_a), lying between values of 3 and 18 mm Hg. A decrease in p_a is accompanied by an increase in nonuniformity of $\dot{V}_A/\dot{Q}$ and an increase in ΔpG. The dependence of ΔpO_2 (curve 2) and ΔpCO_2

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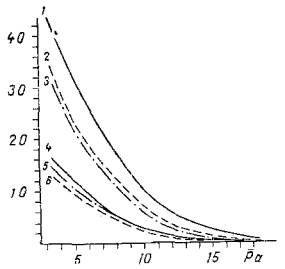

Fig. 1

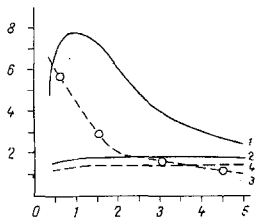

Fig. 2

Fig. 1. Dependence of ΔpO_2 (1, 2, 3) and ΔpCO_2 (4, 5, 6) on blood pressure in common pulmonary artery p_a. 2, 6) Normal gas composition of mixed venous blood (partial pressure of oxygen 40 mm Hg, partial pressure of carbon dioxide 45 mm Hg). 1, 3, 4, 5) Venous hypoxemia (20 mm Hg) and hypercapnia (55 mm Hg). 1, 2, 4, 6) $(\dot{V}_A/\dot{Q})_{mean} = 0.85$; 3, 5) $(\dot{V}_A/\dot{Q})_{mean} = 2.8$. Arteriovenous difference in blood pressure constant at 7.5 mm Hg. Abscissa, mean blood pressure in pulmonary artery (in mm Hg); ordinate, ΔpO_2 and ΔpCO_2 (in mm Hg).

Fig. 2. Dependence of ΔpO_2 (1, 3) and ΔpCO_2 (2, 4) on $(\dot{V}_A/\dot{Q})_{mean}$ in the presence of normal (3, 4) and hypoxemic-hypercapnic (1, 2) gas compositions of mixed venous blood. Distribution of $\dot{V}_A/\dot{Q}$ in the lungs calculated for blood pressure in pulmonary artery $p_a = 10.7$ mm Hg, arteriovenous blood pressure difference 7.5 mm Hg. Abscissa, $(\dot{V}_A/\dot{Q})_{mean}$; ordinate, ΔpO_2 and ΔpCO_2 (in mm Hg).

(curve 6) on p_a for a constant arteriovenous blood pressure difference and a normal gas composition of the mixed venous blood, calculated by means of the model, is shown in Fig. 1. Using these curves, and values of ΔpO_2 and ΔpCO_2 caused by the nonuniformity of $\dot{V}_A/\dot{Q}$, the pressure in the pulmonary artery can be calculated. It is preferable to use ΔpO_2, for this value can be measured relatively more accurately than ΔpCO_2. For example, in the healthy subject ΔpO_2 is 4 mm Hg and ΔpCO_2 is 1 mm Hg [6]. From curves 2 and 6 in Fig. 1 we find that these values correspond to a $\bar{p}_a$ equal to 11.5 mm Hg.

The effect of the partial pressure of gases in the mixed venous blood $p_{\bar{v}}O_2$ and $p_{\bar{v}}CO_2$) and of $(\dot{V}_A/\dot{Q})_{mean}$ are usually not determined in experiments and, if they have a significant influence on ΔpG, the task of determining p_a from ΔpG is made much more difficult. Figure 2 shows the effect of an increase in $(\dot{V}_A/\dot{Q})_{mean}$ on ΔpG with a constant pressure in the pulmonary circulation, but with different values of $p_{\bar{v}}O_2$ and $p_{\bar{v}}CO_2$. As Fig. 2 shows, an increase in $(\dot{V}_A/\dot{Q})_{mean}$ leads to a very small change in ΔpCO_2 but a significant change in ΔpO_2. The different effect of the value of $(\dot{V}_A/\dot{Q})_{mean}$ on ΔpO_2 and ΔpCO_2 can be explained on the grounds that during an increase in $\dot{V}_A/\dot{Q}$ the slope of the line of $\dot{V}_A/\dot{Q}$ on the $O_2 - CO_2$ diagram relative to the CO_2 axis is increased, but that relative to the O_2 axis is reduced [7]. Ultimately the effect of $p_{\bar{v}}O_2$, $p_{\bar{v}}CO_2$, and $(\dot{V}_A/\dot{Q})_{mean}$ on ΔpG is linked only with the properties of the blood as carrier of the respiratory gases.

Thus ΔpG depends not only on $\bar{p}_a$, but also on $p_{\bar{v}}O_2$, $p_{\bar{v}}CO_2$, and $(\dot{V}_A/\dot{Q})_{mean}$. That is why simplified decisions on nonuniformity of $\dot{V}_A/\dot{Q}$ based on values of ΔpG may lead to erroneous conclusions. However, the dependence of ΔpCO_2 on $\bar{p}_a$ changes only a little during a change in $p_{\bar{v}}O_2$, $p_{\bar{v}}CO_2$, and $(\dot{V}_A/\dot{Q})_{mean}$. This will be clear from Fig. 1: curves 4, 5, and 6 do not differ significantly. To determine $\bar{p}_a$, even during very short transition processes, when at certain values of $p_{\bar{v}}O_2$ and $p_{\bar{v}}CO_2$ different values of $(\dot{V}_A/\dot{Q})_{mean}$ are possible, it is therefore possible to use ΔpCO_2. The theoretical dependence of ΔpCO_2 on $\bar{p}_a$ is in good agreement with the experimental data [6].

Calculations show that if the gas exchange is stationary, ΔpO_2 can be used to determine $\bar{p}_a$. In fact, with a stationary gas exchange the respiratory quotient must be 0.7-1.0, and the gas composition of the mixed arterial blood ought to be close to normal. According to the calculations, this is true when $(\dot{V}_A/\dot{Q})_{mean} = 0.85$ for normal values of $p_{\bar{v}}O_2$ and $p_{\bar{v}}CO_2$, and when $(\dot{V}_A/\dot{Q})_{mean} = 2.8$ for $p_{\bar{v}}O_2 = 20$ mm Hg and $p_{\bar{v}}CO_2 = 55$ mm Hg

(an example of physical loading). With these steady-state values of $p_{\bar{v}}O_2$, $p_{\bar{v}}CO_2$, and $(\dot{V}_A/\dot{Q})_{mean}$, the dependences of ΔpO_2 and $\bar{p}_a$ are close (curves 2 and 3 in Fig. 1). Similar dependences of ΔpO_2 and $\bar{p}_a$ also were obtained for other examples of stationary gas exchange. When the gas exchange is not stationary, dependences of ΔpO_2 on $\bar{p}_a$ may differ considerably (curves 1 and 2, 3 in Fig. 1).

During stationary gas exchange, to determine $\bar{p}_a$ it is thus necessary to use ΔpO_2, whereas if the gas exchange is not stationary, ΔpCO_2 must be used. In conclusion, it will be noted that values of ΔpG depend not only on $\bar{p}_a$, but also on the blood pressure in the pulmonary veins. At high values of $\bar{p}_a$ the pressure in the pulmonary veins has a significant effect on ΔpG, but at low values of $\bar{p}_a$ this effect is negligible, as the experimental data in [6] confirm. If $\bar{p}_a$ exceeds 13 mm Hg (West's first zone is absent), ΔpG is determined by the size of the second zone, i.e., by the pressure in the pulmonary veins. In addition, at high values of $\bar{p}_a$ the slope of the curves in Fig. 1 relative to the pressure axis is shallow. These factors limit the application of the suggested method for determination of $\bar{p}_a$ to the range of normal (4 and 1 mm Hg) and high values of ΔpO_2 and ΔpCO_2. If other causes of nonuniformity of function of the lungs than gravitational are present, the blood pressure in the pulmonary arteries must be higher than that determined on the basis of the suggested dependence.

The results of this investigation thus enable the blood pressure in the pulmonary artery, an important parameter of the state of the respiratory and circulatory systems, to be determined, given certain conditions, from the values of ΔpO_2 or ΔpCO_2.

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MECHANISMS OF CARBOHYDRATE-INDUCED HYPERLIPEMIA

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KEY WORDS: hyperlipemia in rats; blood and liver lipoproteins; carbohydrate diet.

The term "carbohydrate-induced lipemia" was introduced by Ahrens in 1961 [2] to describe a hyperlipemic state caused in animals by feeding with a high carbohydrate diet. Data in the literature show correlation between the blood triglyceride level and its insulin and glucose levels [8, 9], and also with glucagon [5], which suggests that these hormones participate in the formation of the hyperlipemic state. Recent work has shown the unifying role of proteins in blood lipoprotein molecules and the importance of their composition for the normal formation, secretion, and catabolism of lipoproteins [15]. Meanwhile information on metabolism of apoproteins of the blood lipoproteins in hyperlipemic states is very scanty and the role of individual apoproteins in the mechanism of hyperlipemia is not clear.

Accordingly, the present investigation was undertaken with the aim of studying the rate of formation of the principal apoproteins of very low density lipoproteins in the liver and their ratio in the blood in carbohydrate-induced hyperlipemia and to compare these data with blood levels of insulin and glucagon.

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