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# CHARACTER OF THE PULMONARY CIRCULATION IN EXPERIMENTAL PULMONARY EDEMA DATED BY ARTIFICIAL VENTILATION

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Data have been published on the efficacy of artificial ventilation of the lungs (AVL), using various schedules, during the development of pulmonary edema [7, 9, 11]. An important step in the pathogenesis of pulmonary edema is considered to be a disturbance of the circulation in the lungs [1, 7]. However, the problem of the effect of various schedules of AVL on the pulmonary hemodynamics in this form of pathology has received only little study.

The aim of this investigation was to study the parameters of the pulmonary hemodynamics during the development of acute experimental pulmonary edema, accompanied by an increase in the frequency of volume of AVL.

## EXPERIMENTAL METHOD

Experiments were carried out on 20 cats weighing 2.5-4 kg, anesthetized with pentobarbital (30-40 mg/kg, intraperitoneally), immobilized with tubocurarine (0.25-0.5 mg/kg, intravenously), with a closed chest. AVL was applied by means of a "VITA-1" respirator. Pulmonary edema was produced by intravenous injection of a mixture of fatty acids with olive oil in a dose of 0.04 ml/kg [10]. The pulmonary circulation during the development of pulmonary edema was studied during application of AVL with increased frequency (threefold) or volume (twofold) and compared with that recorded with the initial parameters of ventilation, namely frequency 19 ± 2 $min^{-1}$ and volume 40 ± 3 $cm^3$, which corresponded to the frequency and volume of the animal's natural respiration. A catheter was introduced through the superior lobar artery of the lung into the lumen of the left pulmonary artery and pressure in it was recorded by means of an electromanometer [4]. The blood flow in the left lower lobar artery and vein was studied by means of ultrasonic transducers, applied to the corresponding vessels [5]. Values of blood flow and pressure in the lobar artery of the lung were led to an analog computer, which calculated the vascular resistance in the test lobe of the lung in the course of the process. The initial values were: pressure in the pulmonary artery 14 ± 2 mm Hg, blood flow 51 ± 4 ml · $min^{-1}$, vascular resistance of the lung 0.27 ± 0.05 mm Hg · $ml^{-1}$ · $min^{-1}$. The degree of pulmonary edema was estimated

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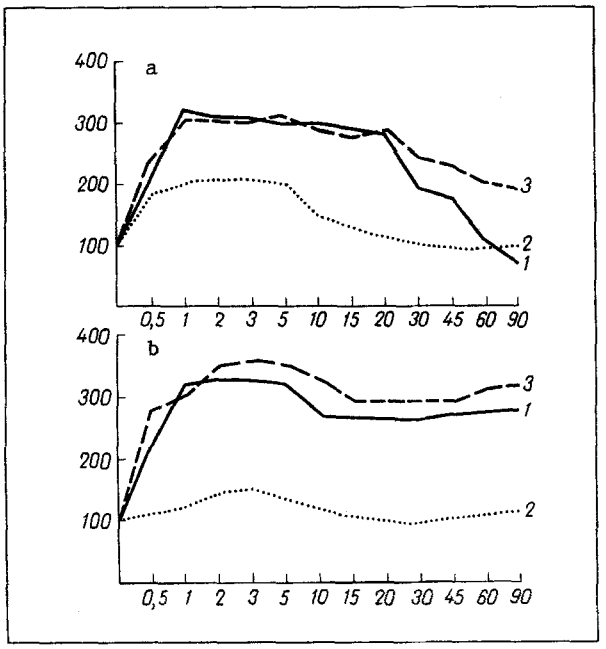


Fig. 1. Change in pressure in pulmonary artery and resistance of pulmonary vessels during development of pulmonary edema with AVL with increased frequency or volume. Abscissa, time of observation (in min); ordinate, change in parameters relative to initial values (in %); a) pressure in pulmonary artery, b) vascular resistance of the lungs. 1) Initial schedules of ventilation, 2) increase in frequency, 3) increase in volume of AVL.

as the values of the pulmonary coefficient and dry residue [3]. The parameters recorded and calculated were determined for 60-90 min after induction of edema, for the animals died in the course of this period or the parameters studied were stabilized at a steady level.

## EXPERIMENTAL RESULTS

In all the experiments basically similar changes were obtained in the parameters of the pulmonary hemodynamics. Under the initial schedules of AVL the pressure in the pulmonary artery increased by 2.8-3.2 times in the experimental animals after injection of fatty acids, and the pressure remained at that level for 20-30 min before falling until the 60th minute of the investigation to the original values (Fig. 1a). The change in pressure in the pulmonary artery against the background of increased volumes of AVL during the first 20 min after injection of fatty acids did not differ from that during ventilation according to the original schedules. Later during the experiment the pressure still remained high, twice as high as initially at the 60th minute. The development of pulmonary edema during AVL with increased frequencies was accompanied by a twofold rise of pressure in the pulmonary artery. However, it was of short duration, and having reached its initial level 20-30 min after induction of edema, thereafter it remained unchanged until the end of the experiment.

The investigations thus showed that with AVL of increased frequency the rise of pressure in the pulmonary artery during the development of toxic pulmonary edema was smaller than during ventilation with the initial schedules or with increased volume.

The vascular resistance of the lungs 1-2 min after induction of edema, and with the original AVL schedules or with increased volume, increased on average by 250% (Fig. 1b). Later during the experiment it gradually fell, while still remaining 150-220% above the initial value. When AVL with increased volume was used a tendency was noted for a greater increase in pulmonary vascular resistance than with the initial ventilation schedules. Both during ventilation with increased volume and with the initial ventilation schedule the vascular resistance of the lungs began to rise after injection of fatty acids simultaneously with the rise of pressure in the pulmonary artery or 10-15 sec later. During AVL with increased frequency the increase in resistance to the blood flow in the lungs during development of pulmonary edema began 30-40 sec later than the pulmonary arterial pressure,

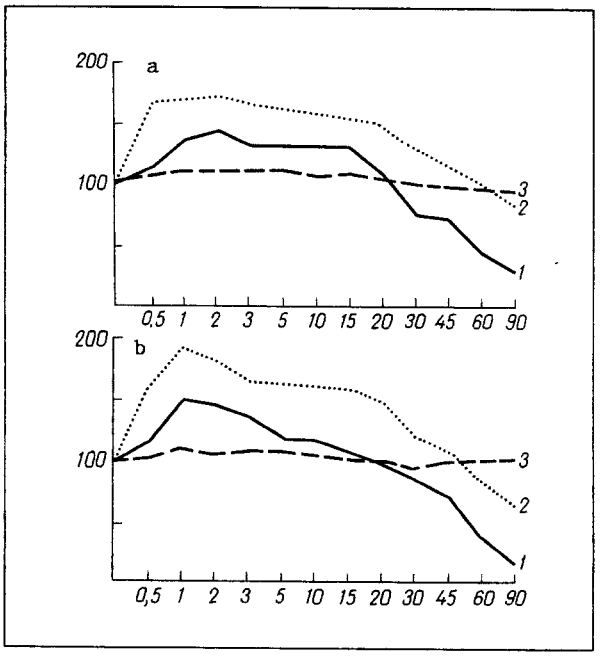


Fig. 2. Changes in volume velocity of blood flow in lower lobar artery and vein in the lung during development of pulmonary edema accompanied by AVL with increased frequency or volume. Abscissa, time of observation (in min); ordinate, change in volume velocity of blood flow relative to initial values (in %): a) in lobar artery of lung, b) in lobar vein of lung. 1) Initial ventilation schedules, 2) increase of frequency, 3) increase of volume of AVL.

and by the 3rd minute after injection of fatty acids it was increased by only 50%. At about the 15th minute of development of edema the pulmonary vascular resistance reached its initial level, and thereafter remained within these limits.

Consequently, the development of toxic pulmonary edema during AVL with increased frequency is accompanied by a decrease in the degree of growth of the vascular resistance of the pulmonary circulation, but during AVL with increased volume, on the other hand, a tendency is observed for its value to increase.

The sharp increase in pulmonary vascular resistance in this model of edema is linked with the addition of microembolism to the toxic action of the fatty acids [6, 7]. On the basis of data in the literature and our own observations it can be postulated that the reduction of the increase in vascular resistance in the pulmonary circulation during the development of pulmonary edema and against the background of AVL with increased frequency is connected with normalization of the blood flow in the pulmonary microcirculatory system, from which cell aggregates and microthrombi are flushed out, and also with a reduction of the degree of hypoxemia [2, 8].

The volume velocity of the blood flow along the lower lobar artery and vein with the initial ventilation schedules increased during the 20 min after injection of fatty acids on average by 45%, to reach a maximum in the first 2-3 min (Fig. 2a, b). Later, the blood flow along these vessels was reduced, to reach a level 80-90% lower than initially after 60-90 min of the investigation. With the development of pulmonary edema and with AVL with increased frequency, the velocity of the blood flow along the lower lobar artery and vein increased for 1-2 min on average by 80%, and then fell gradually to its initial values after 45-60 min. During the 3-5 min after induction of edema, with AVL with increased volume, the volume velocity of the blood flow in the test region increased at most by 20%. Later, until the end of the experiment, it did not differ from its initial level or fell by 10-20% below it.

During the development of pulmonary edema, against the background of AVL with increased frequency, the velocity of the blood flow along the lower lobar artery and vein exceeded that observed with the original ventilation schedules. During AVL with increased volume the blood flow along these vessels in the initial stages of edema was less than with the original ventilation, but later it exceeded it.

It was found in the course of the experiments that by the 60th-90th minute after induction of edema marked pulmonary edema developed, and its degree at this time was independent of the ventilation schedule used. The pulmonary coefficient was 12.7 ± 0.8 and the dry residue 15.8 ± 0.9 (6.7 ± 0.3 and 25.3 ± 0.6, respectively in intact animals). Previously, however, the writers showed that the use of AVL with increased frequency or volume schedules delays the increase in the degree of toxic edema of the lungs in the first 20-30 min of its development [8]. This may perhaps be associated with a fall of pressure at this period in the pulmonary artery and a fall of resistance in the pulmonary vessels, during the development of pulmonary edema against the background of an increased frequency of ventilation, and with the decrease in the velocity of the blood flow in the pulmonary vessels during ventilation with increased volume.

It can be concluded from these data that the development of toxic pulmonary edema against the background of AVL with increased frequency is characterized by reduction of the degree of rise of the pulmonary arterial pressure and of the vascular resistance of the pulmonary circulation, and also by an increase in blood flow along the lobar artery and vein of the lung compared with the original ventilation schedules. With an increased volume of AVL a tendency was noted for the increase of pressure in the pulmonary artery and the increase in resistance of the pulmonary vessels to be greater than with the original ventilation schedule. Comparison of changes in the hemodynamics with the degree of pulmonary edema suggests that the decrease of pressure in the pulmonary artery and of resistance and of the velocity of the blood flow in the pulmonary vessels observed during the use of AVL with increased frequency or volume may participate in the slowing of development of pulmonary edema in its initial stages.

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