PULMONARY CIRCULATION IN EXPERIMENTAL PNEUMONIA

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UDC 616.24-002-092.9-07:616.24-005

KEY WORDS: pulmonary circulation; experimental pneumonia; ultrasound; oxygen; saturation.

The writers showed previously that under normal conditions, in the presence of different degrees of hypoxia and hypercapnia associated with a considerable rise of blood pressure in the pulmonary artery, the blood flow in the lobar arteries of the lungs remains relatively stable [9]. This is evidently connected with structural and functional properties of the vascular bed of the pulmonary circulation, maintaining equilibrium between the output of the right and left heart [10, 11, 15]. In pneumonias, the alveoli in the affected segments of the lungs are excluded from ventilation. Despite this, the arterial partial pressure of oxygen (p_aO_2) and the oxygen saturation of the arterial blood (S_aO_2) are frequently maintained at levels close to normal [2, 10, 12, 14]. Maintenance of a sufficiently high S_aO_2 is possible only provided that the blood flow through the nonventilated segments of the lungs is stopped or considerably restricted. This process is based on the constrictor response of the pulmonary vessels to alveolar hypoxia [4, 10, 11, 13, 15]. This conclusions is based on indirect data. A direct study of the pulmonary circulation during the development of an inflammatory process in the lungs is accordingly very important. The degree of preservation of the constrictor response of the pulmonary vessels to hypoxia during the development of inflammation in the respiratory tract and in the lungs is another problem which remains unstudied.

The aim of this investigation was to study the blood flow in unaffected and affected lobes of the lungs in acute experimental pneumonia, and also to determine the reactivity of the pulmonary vessels relative to hypoxic hypoxia and hypercapnia under these conditions.

METHODS

In acute experiments on 48 cats weighing 3-5 kg thoracotomy was performed under pentobarbital anesthesia (30-40 mg/kg), artificial ventilation of the lungs was applied, and by means of an ultrasonic method [5] the blood flow was studied in different parts of the vascular bed of the lungs: the infundibulum (output of the right heart — RHO), in the branch of the pulmonary artery carrying blood to the lower lobe of the left lung, and in the vein draining that lobe. The method of studying the pulmonary blood flow was described by the writers previously [8]. The blood pressure in the pulmonary circulation was measured by means of a microelectromanometer [6]. A cannula was introduced through the superior lobar artery into the lumen of the left pulmonary artery. In some experiments S_aO_2 was determined (blood was taken from the femoral artery), and also the degree of oxygenation of mixed venous blood taken from the infundibulum, and blood flowing from the affected and unaffected lobes of the lungs (from the corresponding pulmonary veins). Blood samples were analyzed by method of Astrup and Siggaard-Andersen. To estimate the reactivity of the pulmonary vessels and the compensatory powers of the body as a whole, samples with inhalation of gas mixtures containing a reduced concentration of oxygen (5 and 10% O_2 in nitrogen), a raised CO_2 concentration (5 and 10% CO_2 in air), and also pure oxygen, were used in the experiments. Damage to the respiratory tract and lungs was produced by preliminary injection of 0.3 ml of resinified turpentine into the trachea of the experimental animals (under ether anesthesia) toward the lungs, and the animals took part in the acute experiment 48-72 h later. After

Laboratory of Pathophysiology of Respiration and Bioengineering Laboratory, Research Institute of General Pathology and Pathological Physiology, Academy of Medical Sciences of the USSR, Moscow. (Presented by Academician of the Academy of Medical Sciences of the USSR G. N. Kryzhanovskii.) Translated from Byulleten' Éksperimental'noi Biologii i Meditsiny, Vol. 101, No. 6, pp. 671-675, June, 1986. Original article submitted August 23, 1985.

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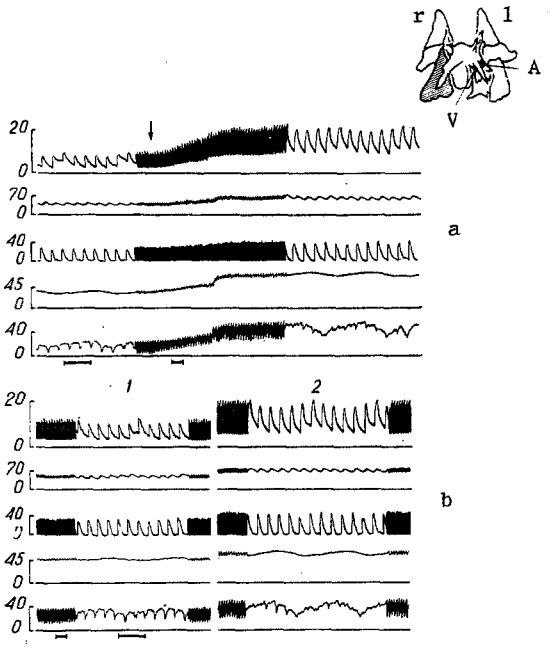

Fig. 1. Effect of hypoxia and hypercapnia on pulmonary circulation in lesion of the right lung. a) Inhalation of gas mixture containing 10% O_2 (arrow indicates beginning of exposure); b) inhalation of mixture containing 5% CO_2: 1) background, 2) 5th minute of breathing mixture. From top to bottom: blood pressure in pulmonary artery (in mm Hg), mean value of flow in inferior lobar branch of the pumonary artery (in ml/min), phased blood flow in inferior lobar branch of pulmonary artery (in cm/sec), mean flow in inferior lobar branch of pulmonary vein (in ml/min), and phased blood flow in inferior lobar branch of pulmonary vein (in cm/sec). Time scale 1 and 10 sec. Here and in Figs. 2 and 3, straight lines beneath each curve indicate zero levels; inset top right shows scheme of location of lung lesion and of transducers on inferior lobar branches of left pulmonary artery (A) and vein (V): r) right lung, 1) left lung; shaded area indicates necrosis, dotted area in Figs. 2 and 3 indicates red hepatization.

receiving the injection of turpentine the animals developed inflammatory changes in the trachea and lungs (more often on the right), which pathomorphologically resembled changes arising in spontaneous pneumonia [1].

RESULTS

In acute experimental pneumonia the absolute values of RHO and the arterial pressure in the systemic and pulmonary circulations were maintained within the limits observed in healthy animals [8, 9]. The volume velocity of the blood flow in the artery carrying blood to the intact lower lobe of the left lung (with involvement of the other lobes) averaged 18.5% of RHO (in healthy animals it averaged 21%). Meanwhile, in animals with experimental pneumonia, the character of the phases of the blood flow in the artery of the uninvolved lobe differed from that observed normally [9], mainly in the absence of diastolic flow (Fig. 1).

The character of the changes in the pulmonary blood flow during hypoxia and hypercapnia depended on the severity of the inflammation. The degree of involvement of the lungs differed in different animals, evidently in connection with their general condition during the period of action of the turpentine. In animals with mild injury to the respiratory tract and lungs (tracheitis, incomplete involvement of one lobe) the same changes were observed in hypoxia and hypercapnia as normally [9]. In the case of more severe lung damage

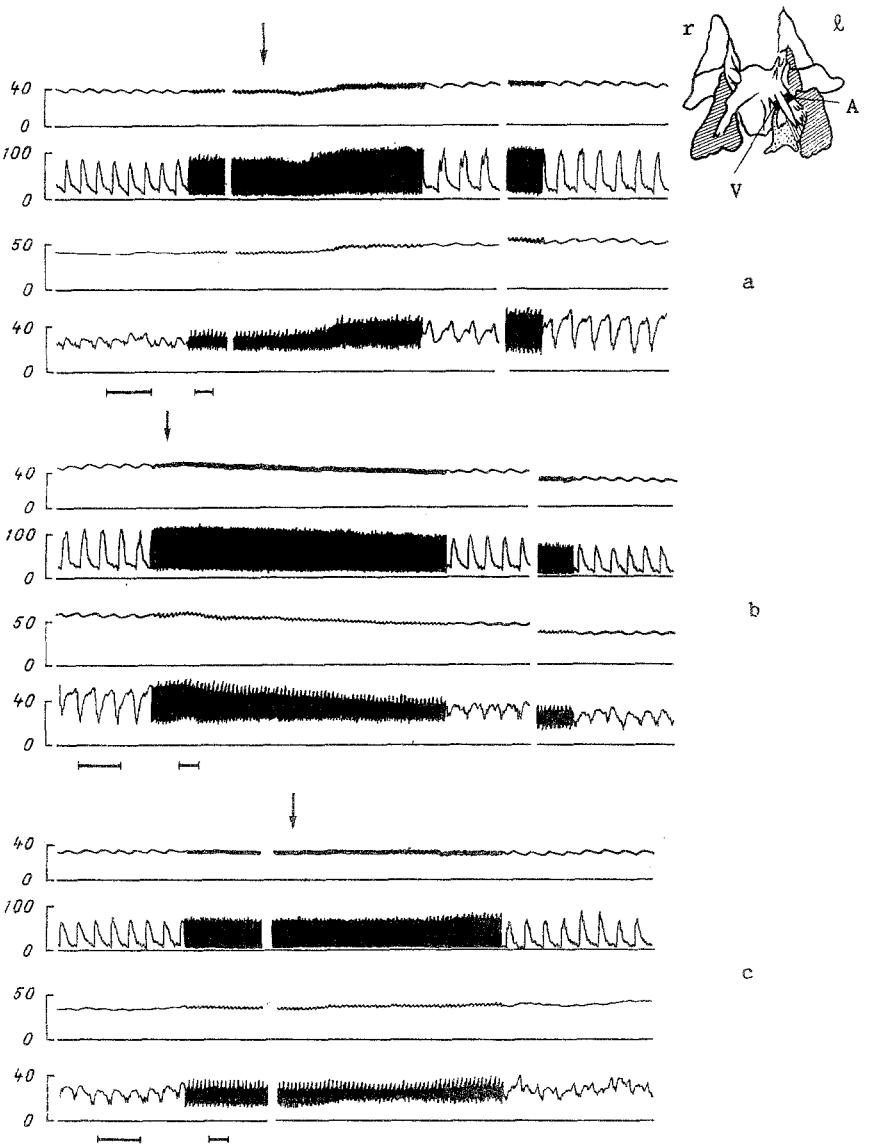

Fig. 2. Effect of hypoxia and hypercapnia in pulmonary circulation in bilateral lung disease. a) Inhalation of gas mixture containing 10% O_2 (arrow indicates beginning of inhalation of mixture); short vertical line, 5th minute of inhalation of this mixture; b) 10th minute of inhalation of hypoxic mixture (arrow indicates changes to air breathing), vertical line indicates 10th minute of recovery; c) inhalation of gas mixture containing 5% CO_2 (arrow indicates beginning of inhalation of mixture). Here and in Fig. 3, from top to bottom: mean blood flow in inferior lobar branch of pulmonary artery (in ml/min), phased blood flow in inferior lobar branch of pulmonary artery (in cm/sec), mean value of flow in inferior lobar branch of pulmonary vein (in ml/min), phased blood flow in inferior lobar branch of pulmonary vein (in cm/sec); time scale 1 and 10 sec; inset above: dotted area indicates hepatization.

(extensive involvement of one or several lobes of one lung with necrotic changes, and also bilateral lung lesions), the nature of the reactions was usually distorted. This was shown by the fact that during moderate hypoxia (10% O_2) and hypercapnia, the blood flow was increased (Fig. 1), whereas in hyperoxia the blood flow was reduced in the inferior lobar branch of the pulmonary artery (normally the opposite relations are observed). In some animals with severe pneumonia, complete reactivity of the vascular bed of the lungs was observed: the blood pressure in the pulmonary circulation and the blood flow in the pul-

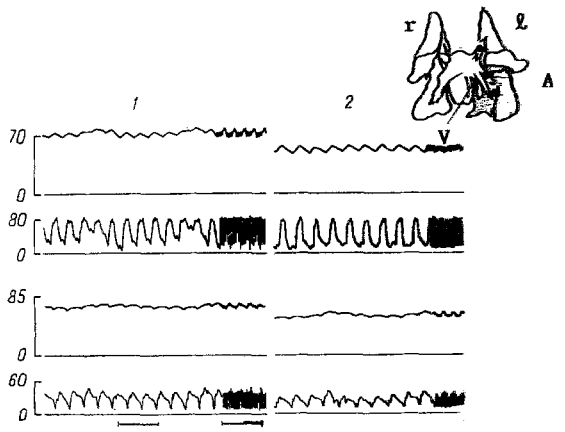

Fig. 3. Effect of hypoxia on pulmonary circulation in disease of the left lung. 1) Background; 2) 10th minute of inhalation of mixture containing 10% O_2.

monary vessels were unchanged in hypoxia and hypercapnia. Similar results were obtained by the writers previously in a study of the cerebral circulation [7].

It was also observed that the blood flow in the pulmonary artery was reduced in healthy animals during inhalation of a gas mixture containing 10% O_2, whereas the blood flow is increased during inhalation of a mixture with 5% O_2 [9]. In animals with severe pneumonia, accompanied by moderate hypoxia (10% O_2), S_aO_2 decreased approximately to the values observed in healthy animals during more severe hypoxia (5% O_2), namely to 60-80%. Distortion of the response to moderate hypoxia in animals with severe pneumonia must evidently be attributed to the greater fall in S_aO_2 during hypoxia.

In experiments on animals with a focus of inflammation in the lower lobe of the left lung unexpected results were obtained. It was found that values of the linear and volume velocity of the blood flow in the inferior lobar artery and vein in these experiments did not differ in fact in absolute terms from those observed in the uninvolved lobe in pneumonia. The pulmonary artery supplying the affected lobe was greatly enlarged in diameter, and the whole lobe itself was enlarged. Usually extensive foci of necrosis and red hepatization are found in this lobe [3], and occupy not less than half of the lobe. This part of the lobe does not expand even when the ventilation volume of the lungs is increased. Evidently there is no blood flow here. The other part of the lobe in which macroscopically visible foci of inflamation are absent, expands well on inspiration and collapses on expiration. It can be tentatively suggested that the same volume of blood as is present in analogous intact lobes passes in this case through a smaller volume of lung tissue, which continues to function. This is expressed in the character of the phased blood flow which, in these experiments, always has a large diastolic flow (Fig. 2). In some experiments the blood flow is chaotic in character: systolic ejections are unequal and the diastolic flow is particularly great (Fig. 3). To judge from the character of the blood flow, it seems that the vessels in the uninvolved part of the large lobe are in a state of intensitve dilatation as a result of a considerable reduction of their tone. In the affected lobe, during hypoxia and hypercapnia dissimilar changes are observed in the blood flow: both a decrease inthe blood flow in the inferior lobar artery (Fig. 3) and an increase (Fig. 2) may take place. In some experiments there was no response.

In the present experiments S_aO_2 of the aniamls with experimental pneumonia was quite high, 93% on average (from 84 to 97%). The degree of oxygen saturation of the mixed venous blood of healthy animals averaged 63% (from 64 to 71%), but in animals with pneumonia it averaged 51% (from 22 to 65%). The oxygen saturation of blood flowing from the affected lobes varied from 45 to 77%, and in some cases it equalled the oxygen saturation of mixed venous blood. Values of oxygen saturation of blood flowing from equally involved lobes differed significantly, even in the same animal.

Blood flowing from unaffected lobes of the lungs (with lesions present in the other lobes) was saturated with oxygen to the extent of 95-100%. In severe lung involvement these values were higher than S_aO_2 of blood taken from the femoral artery. This fact must

be explained by contamination with poorly oxygenated blood flowing into the left heart and thence into the systemic circulation from the affected lobes of the lungs.

It will be noted, however, that in most of the experiments S_aO_2 was not depressed as much as might be expected in view of the extensive foci of inflammation and the very low (in some animals) values of oxygen saturation of blood flowing from the affected lobes of the lungs. This was evidently because the blood flow in the affected parts of the lungs was considerably restricted.

It can therefore be tentatively suggested that the blood flow in a focus of inflammation is restricted and that the volume of blood which is normally present travels along the vessels of the unaffected part of the lobe, which continues to function. This is shown by the high values of the blood flow in the artery carrying blood to the affected lobe, when compared with the evidence of absence of any marked arterial hypoxemia in most of these experiments.

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