

ROLE OF INCREASED PRESSURE IN THE PULMONARY ARTERY IN THE DISTRIBUTION
OF PERFUSION AND GAS EXCHANGE IN BRONCHIAL OBSTRUCTION

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The connection between the extent of bronchial obstruction and changes in the pulmonary circulation and gas exchange [2] still remains largely unexplained. The aim of this investigation was to study changes in the pulmonary hemodynamics and gas exchange during the creation of increasingly widespread bronchial obstruction in experimental animals.

EXPERIMENTAL METHOD

Male dogs weighing 12-15 kg were used. Bronchial obstruction was created in 20 animals. A device consisting of a Porolon plug with tubes passing through it was introduced into the lumen of the left diaphragmatic (lower lobar) bronchus. After 6 days a similar device also was introduced into the right lower-lobe bronchus. As a result the lumen of each bronchus was constricted by 80% [3]. After 5 days, in 5 dogs, under general anesthesia (hexobarbital, 25 mg/kg) the right jugular vein and superior thyroid artery were dissected and through them a catheter was inserted into the pulmonary artery and a double-barreled catheter (with a thermistor in one barrel) into the arch of the aorta. The catheters were filled with heparin and secured to the neck. Regional ventilation of the lungs was studied in five of the 20 dogs by inhalation of ^{133}Xe . Radiometry of the lungs was carried out in zones corresponding to projections of the lower, apical, and cardiac lobes. Regional perfusion of the lungs was studied by measuring the distribution of albumin microspheres labeled with $^{99\text{m}}\text{Tc}$. The indicator was injected intravenously into five healthy animals, after unilateral (five dogs) and bilateral (five dogs) obstruction, and after injection on a ganglion blocker (0.05% solution of trimetaphan) preceded by creation of bilateral obstruction (five dogs). Using a method developed previously [1], a catheter was introduced into the pulmonary vein of the left lower lobe of seven animals, and 7 days later the left lower lobe bronchus was obstructed. Curves of pressure in the pulmonary artery and aorta were recorded on a Mingograf-34 polygraph; cardiac output was determined by the thermodilution method and blood gases by the micro-Astrup method. The parameters of the hemodynamics and gas exchange were measured in conscious animals.

EXPERIMENTAL RESULTS

The distribution of ^{133}Xe and $^{99\text{m}}\text{Tc}$ in the lungs showed that ventilation of the apical and cardiac lobes predominated in healthy animals (Table 1), and the blood flow was more intensive in the lower lobes (Table 2). Disturbance of the patency of the left lower-lobe bronchus led to a decrease (not significant) in accumulation of ^{133}Xe in this lobe, a reduction of its perfusion almost by half, and an increase in ventilation and perfusion of the right lower lobe. Bilateral obstruction was accompanied by redistribution of the ventilation and blood flow in the upper zones of the lungs and by a change in the ratio of alveolar ventilation to blood flow in them: 1.6 on the left and 1.5 on the right in the absence of bronchial obstruction, 1.3 and 1.4 respectively in the case of unilateral obstruction, and 0.9 and 1.3 after bilateral obstruction.

Direct measurements of the blood gases in the pulmonary vein revealed marked local disturbances of gas exchange in the lobe after the creation of obstruction. For instance, pO_2 in blood from the pulmonary vein was reduced by 24.2 mm Hg (Table 3). In an earlier investi-

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TABLE 1. Distribution of ^{133}Xe in Lungs of Dogs before and after Bronchial Obstruction ($M \pm m$)

Stages of experiment	Left lung, %		Right lung, %	
	lower lobe	apical + cardiac lobes	lower lobe	apical + cardiac lobes
Before obstruction of bronchi	19,7 \pm 1,3	24,7 \pm 0,8	22,8 \pm 1,1	32,7 \pm 0,9
Obstruction of left lower-lobe bronchus	18,1 \pm 1,2	22,0 \pm 1,2	26,9 \pm 1,2	33,0 \pm 1,3
Obstruction of left and right lower-lobe bronchi	13,9 \pm 1,3*	26,8 \pm 1,3*	20,4 \pm 0,9*	38,9 \pm 1,2*

Legend. Here and in Tables 3 and 4, asterisk indicates significant differences from values "before obstruction of bronchi," $p < 0.05$.

TABLE 2. Distribution of Albumin Microspheres Labeled with ^{99m}Tc in Lungs of Healthy Dogs and after Bronchial Obstruction (in % of total quantity of indicator injected, $M \pm m$)

Stages of experiment	Left lung, %		Right lung, %	
	lower lobe	apical + cardiac lobes	lower lobe	apical + cardiac lobes
Without bronchial obstruction (control), $n = 5$	29,7 \pm 0,8	14,6 \pm 0,6	33,9 \pm 0,9	21,8 \pm 0,6
Obstruction of left lower-lobe bronchus, $n = 5$	14,5 \pm 1,4	16,8 \pm 1,6	45,8 \pm 2,1	22,8 \pm 1,3
Obstruction of left and right lower-lobe bronchi, $n = 5$	13,6 \pm 0,9*	33,1 \pm 1,2*	23,4 \pm 1,1*	29,9 \pm 1,2*

Legend. Asterisk indicates significant differences from values "without bronchial obstruction," $p < 0.05$.

TABLE 3. Partial Pressure of Oxygen and Carbon Dioxide in Blood of Pulmonary Vein and in Arterial and Mixed Venous Blood of Dogs after Obstruction of Lower-Lobe Bronchus ($M \pm m$; $n = 7$)

Parameters	Stages of experiment	
	before obstruction	obstruction
Partial pressure of O_2 , mm Hg		
arterial blood	86,5 \pm 1,3	78,4 \pm 1,4*
blood from pulmonary vein of left lower lobe	91,8 \pm 1,8	67,6 \pm 1,0*
Partial pressure of CO_2 , mm Hg		
arterial blood	28,5 \pm 0,6	22,5 \pm 0,7
mixed venous blood	33,5 \pm 1,8	35,6 \pm 1,6
blood from pulmonary vein of left lower lobe	24,6 \pm 1,5	27,9 \pm 1,6

gation [3] it was shown that after constriction of the lumen of the bronchus by 80% alveolar hypoxia developed in the lobe - $p\text{O}_2$ in the alveolar gas fell from 107.6 ± 1.8 to 73.4 ± 2.1 mm Hg ($p < 0.05$). The results are evidence that disturbance of the conditions of gas exchange in the lobe was due to obstruction, which was evidently accompanied by delay of ventilation and an increase in the time constant of this region of the lungs.

It follows from the data in Table 4 that during bilateral obstruction in dogs redistribution of the blood flow in the upper zones took place against a background of raised systolic pressure in the pulmonary artery, and no significant changes of cardiac output were noted. If the unevenness of distribution of perfusion in the lungs of the healthy dogs is borne in mind, as well as data showing that pressure depends on blood flow in the pulmonary vessels [4, 5], the observed rise of pressure can be regarded as one of the basic factors leading to a change in the intensity of the blood flow and in the conditions of gas exchange in the reserve zones of the lungs. This conclusion is confirmed by the fact that after injection of the ganglion-blocker into dogs with bilateral obstruction, and consequent lowering of the systolic pressure in the pulmonary artery from 32.5 ± 1.5 to 28.4 ± 1.2 mm Hg ($p < 0.05$) and

TABLE 4. Parameters of Pulmonary Hemodynamics in Dogs after Bronchial Obstruction ($M \pm m$)

Stages of experiment	Pressure in pulmonary artery, mm Hg		Cardiac output, ml/min·kg
	systolic	mean	
Before obstruction of bronchi, n = 20	26,2±0,9	11,8±0,7	158,7±9,8
Obstruction of left lower-lobe bronchus n = 20	29,5±1,2	18,4±1,2*	140,1±7,8
Obstruction of left and right lower-lobe bronchi n = 15	32,5±1,5*	21,6±0,9*	134,2±13,4

in the aorta from 145.6 ± 4.2 to 115.4 ± 8.2 mm Hg ($p < 0.05$), perfusion of the upper zones fell from 33.1 ± 1.2 to $21.6 \pm 1.4\%$ ($p < 0.05$) on the left side and from 29.9 ± 1.2 to $26.4 \pm 1.6\%$ ($p > 0.05$) on the right side, whereas perfusion of the lower lobes rose on the left side from 13.6 ± 0.9 to $22.3 \pm 1.6\%$ ($p < 0.05$) and on the right side from 23.4 ± 1.1 to $29.8 \pm 1.3\%$ ($p < 0.05$). An increase in the blood flow in the poorly ventilated lower lobes was accompanied by an increase in arterial hypoxia: pO_2 in the arterial blood fell from 70.9 ± 1.6 (before injection of trimetaphan) to 67.2 ± 2.1 mm Hg (after injection of trimetaphan).

Local disturbance of the conditions of gas exchange caused by bronchial obstruction is thus compensated by reactions of redistribution of ventilation and of the blood flow into the reserve zones of the lungs, as a result of which the necessary conditions are maintained for oxygenation and for excretion of CO_2 from the body. There is reason to suppose that a rise of pressure in the pulmonary artery, due to the increasing spread of the effects of bronchial obstruction, is a reaction maintaining the intrapulmonary redistribution of the blood flow and improving the gas-exchange function of the lungs. A fall of pressure produced by drugs without restoration of patency of the bronchi and of alveolar ventilation leads to worsening of the gas composition of the arterial blood.

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