the excitatory intercostophrenic reflex, i.e., increased activity of the diaphragm evoked by impulsation from the proprioceptors of the lower intercostal muscles [5, 6]. The two reflexes serve the same purpose: to compensate for the paucity of stretch receptors in the diaphragm, which prevents it from facilitating its own activity.

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THE USE OF HYPEROXIC MIXTURES FOR THE DIAGNOSIS OF LATENT DISTURBANCES OF EXTERNAL RESPIRATION

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The degree of oxygen saturation of the arterial blood is a very important integral parameter of external respiratory function. Under normal conditions venous blood, as it passes through the lungs, is oxygenated up to 95% with oxygen. In the presence of various pathological processes in the effector part of the external respiratory system and the regulatory systems, the level of blood oxygenation may fall. The oxygen saturation of arterial blood (SaO_2) is reduced by alveolar hypoventilation, disturbance of the diffusion properties of the alveolo-capillary membrane, disparity between ventilation and the blood flow in different parts of the lung, and shunting of blood in the lungs [4, 6, 8].

Definite correlation is known to exist between the value of SaO_2 and pO_2 of arterial blood, and this relationship is described by nomograms [5, 7]. High values of p_aO_2, close to 100 mm Hg, always correspond to a high degree of blood oxygenation.

The use of hyperoxic tests widens opportunities for analysis of the role of individual mechanisms impairing gas exchange in the lungs [2, 3, 8, 9]. A leading role in hyperoxic loading must be ascribed to possible changes in the alveolo-arterial oxygen gradient ($\Delta P_{A-a}O_2$), a composite parameter whose value is mainly determined by interaction between reduced alveolo-capillary permeability for oxygen, a disturbed distribution of air in the lungs, and the volume of venous shunting [1, 3, 8].

In the investigation described below values of $\Delta P_{A-a}O_2$ were determined in healthy subjects and patients with chronic nonspecific lung diseases, inhaling hyperoxic gas mixtures, and the characteristics of the disturbances of gas exchange in the lungs were recorded and evaluated by a graphic method, a further development of the technique of comparing p_AO_2 with SaO_2 [4].

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TABLE 1. Gas Composition of Alveolar Air and Capillary Blood and Some Parameters of Acid-base Balance in Healthy Subjects Inhaling Gas Mixtures with Increased Oxygen Concentrations (M ± m)

Parameter	PiO_2, mm Hg				
	160	230	300	460	700
$p_A O_2$, mm Hg	101,5±3,4	160,3±4,1	210,3±3,2	333,4±4,1	517,0±9,3
$p_a O_2$, mm Hg	81,8±1,2	132,5±3,4	160,0±4,1	221,2±4,4	320,1±7,4
$\Delta p_{A-a} O_2$, mm Hg	19,0±3,0	29,0±3,4	51,9±4,7	114±5,3	199±10,5
$\Delta p_{A-a} O_2$, kPa	2,53±0,4	3,86±0,45	6,9±0,63	15,2±0,7	26,5±1,4
$p_A CO_2$, mm Hg	37,4±1,1	36,4±2,0	36,8±2,0	36,9±1,9	37,6±1,5
$p_a CO_2$, mm Hg	37,6±0,71	36,8±1,1	37,1±1,3	37,6±0,9	37,9±2,0
pH	7,42±0,005	7,40±0,008	7,39±0,008	7,39±0,012	7,40±0,014

TABLE 2. Gas Composition of Alveolar Air, Blood Gases, and Some Parameters of Acid–Base Balance in Patients with Chronic Nonspecific Lung Diseases Inhaling Hyperoxic Mixtures (M ± m)

Parameter	PiO_2, mm Hg				
	160	230	300	460	700
$p_A O_2$, mm Hg	107,4±2,0	170,2±3,1	234,1±4,3	350±5,7	534±8,5
$p_a O_2$, mm Hg	79,1±2,3	132,4±4,1	162,3±5,0	213,2±5,6	296,7±7,4
$\Delta p_{A-a} O_2$,* mm Hg	28,0±3,0*	38,0±2,6*	72,0±4,2*	133±5,0*	238±9,6*
$\Delta p_{A-a} O_2$, kPa	3,72±0,399	5,05±0,346	9,58±0,559	18,35±0,665	31,65±1,277
$p_A CO_2$, mm Hg	37,4±1,2	38,5±1,3	38,9±0,9	42,3±2,1	42,3±2,2
pH	7,39±0,005	7,40±0,008	7,40±0,012	7,38±0,01	7,38±0,008

*$P < 0.05$ compared with corresponding parameter for healthy subjects.

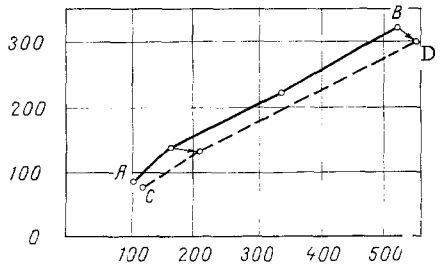

Fig. 1. Graphic estimation of efficiency of intrapulmonary gas exchange. Abscissa, $p_A O_2$ (in mm Hg); ordinate, $p_a O_2$ (in mm Hg).

EXPERIMENTAL METHOD

The parameters chosen for study were determined in 30 healthy subjects and 20 patients with chronic pneumonia (group IIA according to the VNOT* classification — without respiratory failure). The subjects inhaled a hyperoxic mixture for 5-7 min. During this period the blood was completely saturated with oxygen in the lungs [4, 7]. The gas mixtures were prepared in special bags and were carefully mixed. The alveolar air was sampled by the classical method of Haldane and Priestley. The partial pressure of oxygen in the capillary blood was studied at the same time by Clark's method in the AZIV-2 apparatus.

EXPERIMENTAL RESULTS

Under hyperoxic conditions the value of $p_A O_2$ rises, and this may lead to a sharp improvement in the conditions for oxygenation of arterial blood in the lungs. On inhalation of pure oxygen the partial pressure of oxygen in the arterialized capillary blood increases by a lesser degree than $p_A O_2$. If the value of $p_a O_2$ is calculated theoretically ($p_A O_2 - \Delta p_{A-a} O_2$), it is found that the true value of $p_a O_2$ in the presence of a low degree of hyperoxia (PiO_2 = 230 mm Hg) lies within the limits of the theoretically calculated value. Later, however, as the

*All-Union Scientific Society of Internists.

degree of hyperoxia increases, a considerable decrease takes place in p_aO_2 compared with the theoretically calculated value, evidence of changes in the conditions of gas exchange in the lungs during increasing hyperoxia. This effect limits an excessive increase of pO_2 in the blood and tissues and may weaken the toxic action of oxygen (in conjunction with peripheral defensive reactions). A quantitative reflection of the altered conditions of gas exchange in the lungs during hyperoxia is an ever-increasing value of $\Delta p_{A-a}O_2$. The clearest increase in $\Delta p_{A-a}O_2$ was observed when the oxygen concentrations in the inspired air exceeded 230 mm Hg. The maximal value of $\Delta p_{A-a}O_2$ was observed in healthy subjects at PiO = 700 mm Hg, when it was 199 mm Hg (Table 1). The value of $\Delta p_{A-a}O_2$ can be increased due to various causes, but chiefly through shunting [9].

The phenomenon of the increase in $\Delta p_{A-a}O_2$ in healthy subjects during hyperoxia also was manifested in patients with lung pathology (Table 2), but it was manifested in the patients in the presence of lower degrees of hyperoxia. This may be linked with the signs of pathological shunting observed in the presence of pneumonic foci, atelectases in the lungs, and so on.

The values of p_AO_2 and p_aO_2 can be compared by means of the graph in Fig. 1. Curve A-B reflects relations between p_AO_2 and p_aO_2 in healthy subjects and can serve as the basis for describing the characteristics of pathological processes. Curve C-D reflects relations between p_AO_2 and p_aO_2 in patients with chronic nonspecific lung diseases. Compared with the healthy subjects, the patients show a tendency for a shift of the points downward and to the right in the system of coordinates.

The technique of estimating the efficiency of the intrapulmonary gas exchange by comparing the gas composition of the alveolar air and arterialized capillary blood substantially broadens opportunities for detecting and assessing quantitatively the role and importance of the individual factors which interfere with intrapulmonary gas exchange. This method can form a useful addition to other methods of functional testing of the external respiratory system, aimed at discovering the earliest disturbances of the gas exchange function.

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