EFFECT OF GRAVITATIONAL ATELECTASIS OF THE LUNGS ON EXPIRATORY AIRWAY CLOSURE

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KEY WORDS: expiratory airways closure; gravitational atelectasis.

Gravitational overloads, especially if combined with brief inhalation of pure oxygen for a few minutes, cause obstructive-absorptive atelectasis of the lungs through deformation of the lung tissue accompanied by disturbance of bronchial patency, aggravation of the regional inequality of ventilation—perfusion ratios, and rapid absorption of oxygen from the unventilated alveoli by blood in the pulmonary capillaries [3, 8, 9]. The development of atelectasis of the lungs may also take place during prolonged exposure to oxygen for several hours as a result of depression of lung surfactant activity [4, 10].

Atelectasis formation is facilitated by the absence of the inert gas nitrogen in the inspired gas mixture, for nitrogen plays a role of a supporting "cushion" maintaining the morphological stability of the alveoli, and it also increases the surface tension of the liquid film covering the alveoli [4]. Atelectasis is accompanied by a decrease in compliance of the lungs [6], by hypervolemia and congestive changes in the peribronchial vascular system [10] and because of an increase in regional nonhomogeneity of the mechanical properties of the lungs disturbances of patency of the peripheral airways and aggravation of the inequality of ventilation of the lungs can be expected, with a consequent disturbance of gas exchange, increased shunting of venous blood in the lungs, and to circulatory hypoxia, which develops during exposure to overloads.

However, this hypothesis requires experimental verification by the study of the regional ventilation of the lungs in the presence of gravitational atelectasis by investigation of the dynamics of expiratory airway closure.

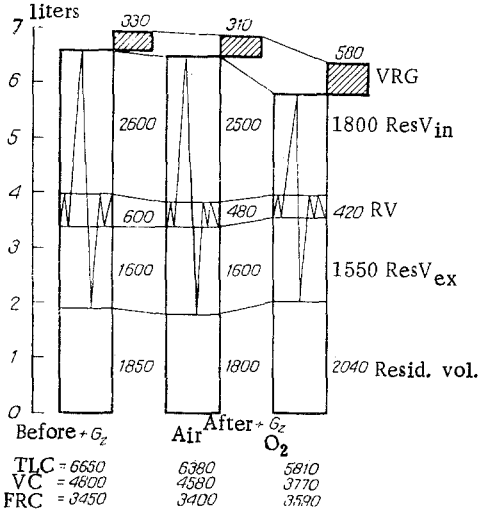

Fig. 1. Changes in fractional structure of total lung capacity (TLC) and volume of retained gas (VRG) in one subject exposed to +Gz overloads together with air or pure oxygen breathing.

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TABLE 1. Effect of +Gz Overloads Combined with Air or Pure Oxygen Breathing on Static and Dynamic Lung Volumes

Statistical index	VC, liters			$ResV_{in}$, liters			$ResV_{ex}$, liters			RV, liters			FRC, liters			TLC, liters			FEV_1, liters			FEV_1/VC, percent		
	1	2	3	1	2	3	1	2	3	1	2	3	1	2	3	1	2	3	1	2	3	1	2	3
$M \pm m$	4,8	4,8	4,0	2,6	2,6	1,8	1,6	1,6	1,6	0,6	0,6	0,6	3,6	3,6	3,5	6,8	6,8	5,9	4,1	4,0	3,5	85	83	88
	0,18	0,21	0,15	0,05	0,06	0,05	0,14	0,14	0,15	0,05	0,06	0,05	0,21	0,23	0,25	0,23	0,21	0,12	0,12	0,16	0,18	5,1	4,9	4,6
P	<0,05			<0,01												<0,05			<0,05					

Legend. Here and in Table 2: 1) before, 2) after exposure to overloads while breathing air, 3) after exposure to overloads while breathing oxygen; TLC) total lung capacity.

TABLE 2. Effect of +Gz Overloads Combined with Air or Pure Oxygen Breathing on Expiratory Airway Closure Volumes and Uniformity of Lung Ventilation

Statistical index	LCV, liters			LCC, liters			Res FRC, liters			LCV/VC, per cent			LCC/TLC, per cent			Res FRC/FRC, per cent			RGL, ml			IEM, %		
	1	2	3	1	2	3	1	2	3	1	2	3	1	2	3	1	2	3	1	2	3	1	2	3
M	0,48	0,57	0,91	2,44	2,57	2,81	1,12	1,08	0,69	10	11	23	36	37	48	32	30	19	326	358	497	92	88	75
$\pm m$	0,17	0,19	0,11	0,15	0,16	0,19	0,20	0,18	0,16	2,0	2,4	3,1	2,8	3,4	3,2	2,6	2,9	2,1	36	47	51	6,6	9,2	6,2
P	<0,05						<0,05						<0,01			<0,01			<0,01			<0,05		

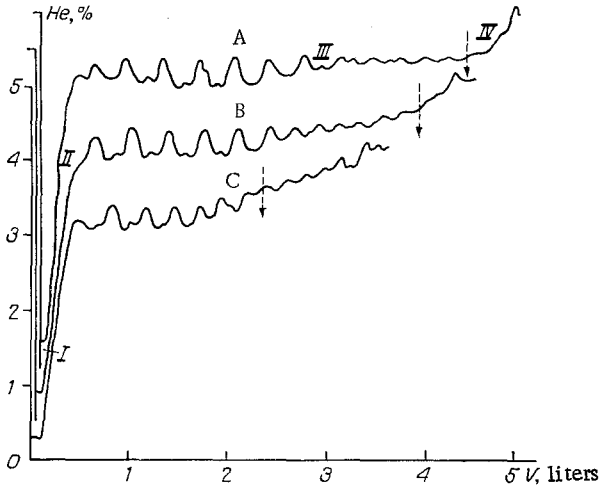

Fig. 2. Changes in parameters of expiratory airway closure in one subject before (A) and after exposure to +Gz overloads while breathing air (B) and oxygen (C). I, II, III) Phases of lung emptying before beginning of EAC; IV) the EAC phase.

EXPERIMENTAL METHODS

Tests on nine healthy men aged 18-29 years were carried out on a centrifuge with variable +Gz overloads of between 2 and 8 units for 5 min and inhalation of air or pure oxygen.

Before and 5 min after overloads the static and dynamic lung volumes, the uniformity of intrapulmonary mixing of the gases, and the indices of expiratory airway closure were determined.

The vital capacity (VC) and its fractional volumes: The inspiratory and expiratory reserve volumes ($ResV_{in}$, $ResV_{ex}$), the respiratory volumes (RV), the forced expiratory volume in the 1st second (FEV_1) were determined on a Godart spirograph. The functional residual capacity (FRC) was determined by the helium dilution method by means of a katharometer and the digital computer of the spirograph.

The uniformity of intrapulmonary gas mixing was determined from the time taken for the helium concentration to reach equilibrium in the lungs — spirograph system and the index of efficiency of mixing (IEM) [5].

Expiratory airway closure (EAC) was investigated by the bolus method [1, 8], using helium as the test gas. By synchronous recording the expiratory helium concentration and the expiratory volume on a Ricken-Denhi x-ray recorder the following parameters of EAC were determined: the lung closing volume (LCV) — that part of VC from the beginning of EAC to the residual volume level; the lung closing capacity (LCC) — the total of LCV and the residual volume; the reserve FRC (Res FRC) — the part of VC from the level of quiet expiration to the beginning of EAC. The volume of gas excluded from ventilation of the lungs as a result of EAC, the so-called retained gas of the lungs (RGL), was determined by measuring FRC twice during ordinary and forced breathing [6].

EXPERIMENTAL RESULTS

Exposure to overloads during air breathing did not lead to any subjective disorders of breathing in the subjects and all indices of lung ventilation showed no significant change (Tables 1 and 2).

Overloads combined with breathing pure oxygen caused coughing in most subjects, and in some of them sensations of retrosternal discomfort or pain in the intercostal muscles. After the end of exposure to overloads and a return to breathing atmospheric air these symptoms quickly diminished and disappeared completely in the course of 15-30 min.

Spirographic investigation of the subjects revealed a significant ($P < 0.05$) decrease in the vital capacity and total lung capacity, by 16.7 and 13.2% on average. The reduction in VC in all cases took place on account of considerable (on average by 30.8%) limitation of the inspiratory reserve volume (Tables 1 and 2; Fig. 1).

Investigation of the dynamic lung volume showed that FEV_1/VC ratio, characterizing patency mainly of the large bronchi, was unchanged and remained within normal limits (80-90%). Accordingly, the changes in the spirographic indices during the period after exposure to overloads indicated mainly the restrictive type of disturbances of pulmonary ventilation on account of the formation of transient areas of atelectasis of the lungs and the simultaneous development of peripheral bronchial obstruction.

The biomechanical hypothesis explains its appearance on the grounds that because of the vertical intrapleural pressure gradient, due to the mass of the mediastinal structures in the lungs, during prolonged exposure to +Gz overloads the intrapleural pressure in the basal portions of the lungs rises, to reach positive values, and becomes greater than the intraalveolar pressure. As a result of this excess of transpulmonary pressure over the intraalveolar pressure the small airways are compressed, with the formation of air traps. Our observations on the increase in the volume of retained gas in the lungs confirmed this hypothesis and showed that expiratory airway closure is evidently due to regional disturbances of bronchial patency and of the ventilation of individual zones of the lungs, which accompany foci of atelectasis.

The sooner EAC arises during expiration, the worse the ventilation of the lungs and the more marked the hypoxemia [2]. Accordingly the sharp increase in the fractional indices of EAC in the general structure of lung volumes must be noted (Tables 1 and 2; Figs. 1 and 2); evaluation of the FEV — LCC difference, the so-called reserve FRC, is the most interesting aspect of the problem.

Comparing FRC and LCC enables an opinion to be formed of ventilation—perfusion relations: if LCC is greater than FRC, poorly ventilated zones will exist in the lungs as a result of EAC even during quiet breathing, and part of the unoxygenated venous blood will be shunted through these zones into the arterial system.

In the present experiments a decrease in the reserve FRC was observed on average from 1.12 liter in the initial state to 0.63 liter after exposure to overloads during oxygen breathing. Changes in the ratio of the reserve FRC to FRC, which fell on average from 32 to 19% ($P < 0.01$), were even more marked.

The observed changes in EAC can evidently be interpreted as a manifestation of regional obstruction of the peripheral airways, leading to an increase in the fractional volume of the lungs with impaired ventilation.

It can thus be concluded from the results of this investigation that disturbances of ventilation of the lungs accompanying gravitatinal atelectasis are mixed restrictive-obstructive in nature. For the objective diagnosis of these disturbances, besides traditional spirographic methods, determination of the expiratory airway closure can provide significant information for it can bring to light early obstructive disturbances of lung ventilation and it allows the dynamics of recovery of this function to be studied.

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EXPERIMENTAL ACUTE HIGH ALTITUDE PULMONARY EDEMA AND THE MECHANISMS OF ITS DEVELOPMENT

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KEY WORDS: acute high-altitude pulmonary edema; oxygen saturation of arterial blood; circulating blood volume.

The syndrome of acute high-altitude pulmonary edema (AHAPE) is frequently found in the mountainous regions of our planet [2, 4-12, 14, 16]. However, the causes and mechanisms of its development have not yet been explained. The prevention of the syndrome is still less near to solution. The difference of opinion on the origin and mechanisms of development of the AHAPE syndrome can be explained by the absence of an experimental model of the syndrome in animals that corresponds adequately to the natural condition, so that the development of its various phases could be reproduced in order to discover the importance of disturbances of the various functional systems in this process and the order in which they come into operation.

The object of this investigation was to determine the possible mechanisms of development of the AHAPE syndrome.

EXPERIMENTAL METHODS

Experiments were carried out on 60 male chinchilla rabbits weighing 2.9-4.5 kg. The animals were kept in a clinical pressure chamber at an "altitude" of 6 km, where the meteorologic factors (temperature, wind velicity, air humidity, UV irradiation) corresponding to that altitude were reproduced. Under these conditions the various parameters chosen for study were determined every hour for 360 min. To assess the functional state of the cardiovascular system the ECF was recorded in standard lead II on a 6NEK-401 apparatus and the great vessels and chambers of the heart were catheterized for recording of the pressure curves

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