CHANGES IN GAS EXCHANGE AND OXYGEN SATURATION OF ARTERIAL BLOOD IN MAN DURING EXPOSURE TO TRANSVERSE ACCELERATION

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Several investigations have been carried out to study the function of human respiration during exposure to acceleration acting in a direction perpendicular to the longitudinal axis of the body [1-4, 6, 8-13].

The present communication (a continuation of reports of earlier studies [1, 2]) describes results indicating the function of gas exchange and the oxygen saturation of the arterial blood during exposure to acceleration.

EXPERIMENTAL METHOD

Experiments were carried out on 12 men aged 24-35 years on a centrifuge of large radius. Altogether 84 experiments were carried out, most of them with acceleration in the vertebro-sternal axis at an angle of 80° to the longitudinal axis of the human body. Accelerations of 4, 6, 8, 10, and 12 g were studied, with a specified time of action ranging from 20 sec to 2 min.

Several experiments were carried out with accelerations of 8 and 12 g, and in them the duration of exposure was determined by the subjects themselves in relation to the critical state when continued exposure to these experimental conditions was impossible.

The oxygen consumption and the excretion of carbon dioxide were determined by the Douglas–Haldane method. To obtain information of the gaseous composition of the alveolar air, the last sample of expired air was taken with the subject breathing spontaneously.

The oxygenation of the arterial blood was determined by means of the Soviet 0-38 oxyhemometer. The ferroresonance stabilization of the forces of power in relation to the anode voltage and the filament voltage of the tubes of the apparatus provided in the factory model proved inadequate for working on the centrifuge and was replaced by a system for supplying current to the apparatus with electronic stabilization of the anode and filament voltages.

EXPERIMENTAL RESULTS

The mean values of the indices of gas exchange studied during exposure to acceleration of different magnitudes are shown in Fig. 1.

Exposure to acceleration of 4, 6, 8, and 12 g caused an increase in the O_2 consumption and CO_2 excretion, and the greatest increase was observed at an acceleration of 8 g. In the experiments with acceleration of 12 g the O_2 consumption and the CO_2 excretion were lower than in the experiments with acceleration of 8 g, although they exceeded the background level.

Characteristic changes during exposure to acceleration were also observed in the gaseous composition of the mixed expired air. With an increase in the acceleration the concentration of CO_2 in the expired air fell while the O_2 concentration rose (Fig. 2).

Among the possible causes of this phenomenon, the possibility of a significant "dilution" of the alveolar air by the air of the dead space must be considered, giving rise to rapid, superficial respiration. However, the respiration rate (Table 1) in these experiments increased to relatively low figures, while the depth of respiration in most cases was actually slightly greater than the initial level, so that this factor cannot be taken as the only important cause of these changes in the gaseous composition of the expired air.

This is confirmed by the absence of any well defined relationship between the gaseous composition of the expired air and the depth and rate of respiration in each individual case.

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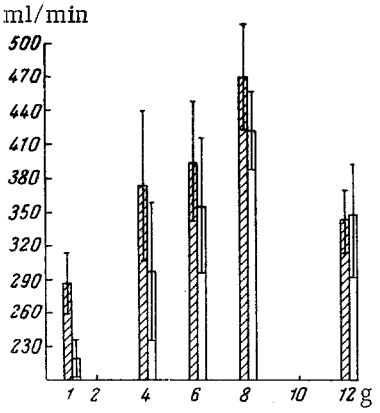

Fig. 1. Oxygen consumption (shaded columns) and excretion of CO_2 (unshaded columns) at accelerations studied.

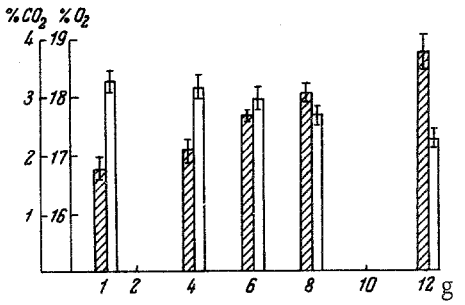

Fig. 2. Proportion of O_2 (shaded columns) and CO_2 (unshaded columns) in expired air (in %) at accelerations studied.

In connection with these remarks, the results showing the gaseous composition of the alveolar air are of great interest (Table 2). In all the experiments with exposure to acceleration the percentage of CO_2 in the alveolar air was lower, and the percentage of O_2 higher than in the samples obtained before the experiment.

Changes of this type in the composition of the alveolar air evidently reflect a disturbance of the process of gas exchange between the alveolar air and the blood of the capillaries in the lungs, possibly a result of hemodynamic changes in the pulmonary circulation arising in these conditions [3, 6, 9].

Since the marked decrease in oxygen saturation of the arterial blood during exposure to acceleration is not accompanied by any significant change in the CO_2 concentration in the arterial blood [12], while the respiratory coefficient increases, there is reason to suppose that the process of excretion of CO_2 from the body during exposure to such accelerations is disturbed relatively less than the oxygen supply.

Characteristic changes in the gas exchange were also observed in the immediate after-period. The intensity of the gas exchange in this period as a rule was higher than before exposure, and it depended on the magnitude and duration of the acceleration. In the experiments with high acceleration (with a similar duration of its action) the level of the gas exchange in the after-period was higher, while in the experiments with the same acceleration but lasting for a shorter period, it was lower.

It may, therefore, be concluded that the accelerations studied caused disturbances in the gas exchange in the lungs. In these circumstances the functional activity of several systems and organs increased simultaneously, with an accompanying increase in the oxygen consumption of the body; consequently, it is reasonable to consider that during the period of exposure to acceleration, an oxygen debt arose in the body, its size depending on the magnitude and duration of the acceleration. This debt was liquidated in the period of the after-effect.

The figures for the oxygen saturation of the blood were very demonstrative in this connection (Fig. 3).

The period of initial acceleration of the centrifuge in most cases was accompanied by a slight increase (by 0.5-1%) in the oxygen saturation of the blood, and this corresponded as a rule to increased ventilation of the lungs.

TABLE 1. Rate and Depth of Respiration at Different Accelerations

	Acceleration (in g)				
	1	4	6	8	12
Rate of respiration (per minute)	13,5±1,5	16±2,4	19±2,7	21±6,5	27±4,7
Respiratory volume (in ml)	500±60	590±110	620±110	800±80	560±100
Min. volume of respiration (in liters/min)	6,6±0,4	9,3±1,3	11,5±1,7	15,9±5,2	14,5±2,2

TABLE 2. Content of O_2 and CO_2 (in %) in Alveolar Air before Experiments and during Period of Specified Acceleration

Subject	CO_2				O_2			
	Acceleration (in g)							
	1	6	1	12	1	6	1	12
SL	5,5	4,2	5,6	2,2	15,5	17,7	15,3	18,4
T	5,2	3,8	5,3	2,7	14,5	17,6	14,9	19,3
S	5,6	4,6	5,8	4,5	14,9	16,6	14,9	17,8
A	5,3	4,5	—	—	15,7	17,4	—	—
E	5,2	5,1	—	—	15,9	16,8	—	—
U	5,1	4,1	—	—	14,7	17,0	—	—

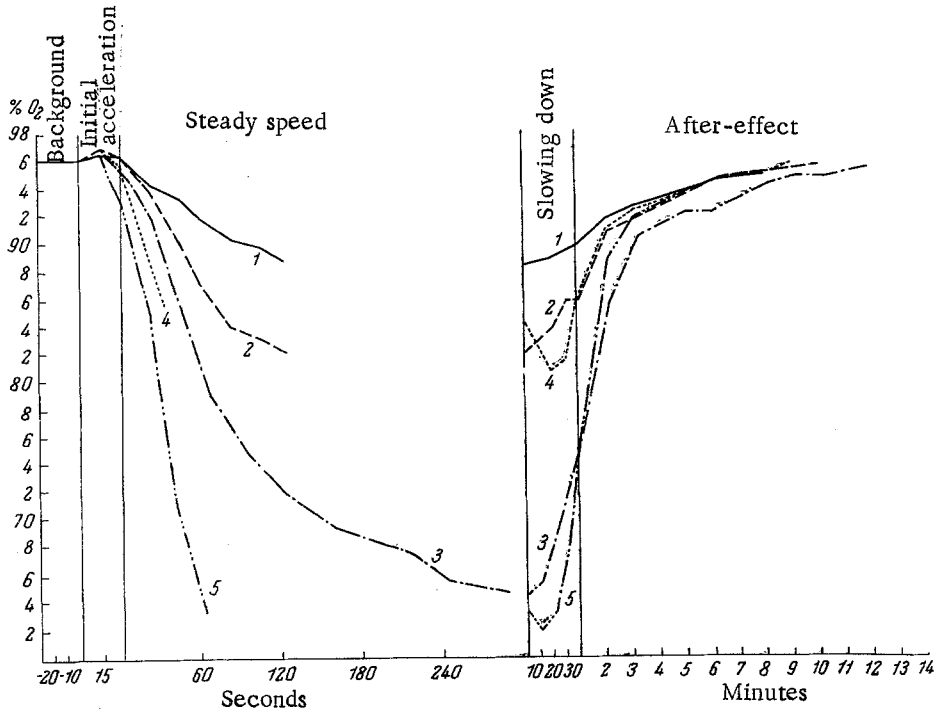

Fig. 3. Percentage saturation of arterial blood with oxygen at accelerations studied: 1) 4 g; 2) 6 g; 3) 8 g; 4) 10 g; 5) 12 g.

At the end of the initial acceleration or at the beginning of the period of a steady speed, the oxygen saturation of the blood began to fall. The rate of this fall bore a definite relationship to the magnitude of the acceleration: the higher the acceleration, the faster the rate of fall of the relative proportion of oxyhemoglobin. For instance, by the 60th second of rotation at a steady speed a statistically significant difference was observed in the oxygen saturation of the blood at different magnitudes of acceleration.

In the experiments with the maximal period of action of accelerations of 8 and 12 g the blood oxygen saturation at the end of the steady speed reached extremely low figures (60-65%). This level of oxygen saturation of the blood was only slightly higher than that at which loss of consciousness takes place, as we know from experiments carried out in a pressure chamber [5, 7].

Another interesting fact is that the level of oxygenation of the arterial blood at the end of the steady speed in the experiments with maximal duration of exposure to accelerations of 8 and 12 g was very similar. This may be confirmation of the view that one of the main factors limiting tolerance to acceleration is the state of the oxygen balance of the organism.

With the beginning of slowing down of the centrifuge in the experiments with acceleration of 4, 6, and 8 g usually the oxygenation of the blood began to increase at once. In most experiments with acceleration of 10 and 12 g the period of braking of the centrifuge (10-20 sec) was accompanied by a still further decrease of this index,

after which the relative proportion of oxyhemoglobin began to increase rapidly, and this period was accompanied by a sharp increase in the ventilation of the lungs.

This phenomenon may evidently be associated with the release into the active circulation of a large volume of blood stored in depots (including the lungs) with a very low relative proportion of oxyhemoglobin, during the period of exposure to acceleration, and this led to a still further fall in the level of oxygenation of the blood.

The oxygen saturation of the blood returned almost to its initial level 3-12 min after the centrifuge had stopped.

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