A STUDY OF THE HEMODYNAMICS DURING OXYGEN RESPIRATION UNDER EXCESS PRESSURE

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Oxygen respiration under excess pressure is widely employed in the practice of medicine. Thus, in aviation it is an effective means of providing oxygen for man at high altitudes (greater than 12 km) in those instances where the hermetic seal of the airplane's cabin is disturbed.

In clinical medicine respiration under excess pressure is employed during certain regimes for artificial respiration, especially during operations on organs in the thoracic cavity; it is also successfully used therapeutically in acute emphysema and several other illnesses. There is also grounds for postulating that respiration under elevated pressure, inevitably leading to hypertension in the pulmonic circulation, can be used as a functional test for appraising the reserve potentials of the right heart.

Probably, one of the first reports of a change in the blood circulation during rapid, brief elevation of the intrapulmonic pressure was already made in the XVII century in the work of Valsalva; he noted that elevation of the intrapulmonic pressure, caused by attempting to exhale against a closed glottis (the Valsalva Maneuver), brought about a sharp drop in blood flow, up to a temporary complete stop.

In 1853, Donders [8] observed that an increase in the pressure within the lungs inhibits the normal filling of the heart during diastole, and is a reason for the development of venous congestion. The essential changes in blood circulation during oxygen respiration under elevated pressure were experimentally established in 1860 by Einbrodt [9]. In experiments on dogs he found that the pressure in the right auricle and in the veins rose in parallel to elevation of the intrapulmonic pressure. When the intrapulmonic pressure attained 25-30 mm Hg a reduction in the arterial pressure was observed in the experimental dogs, which along with disruption of respiration, appeared to be the basic reason for the death of the animals.

It is of definite interest to note Einbrodt's observation [9] pertaining to the appearance of an opposing pressure in the stomach (contraction of the stomach in the experimental animals) which permitted normalization of the blood circulation during respiration under elevation pressure. In the subsequent works of P. M. Gramenitskii et al. [2], D. I. Ivanov et al. [4], G. V. Altukhov and N. A. Agadzhanyan [1], in experiments on animals, it was shown that elevation of the pressure in the lungs inevitably leads to an increase in the intravascular pressure of the pulmonic circulatory system, the large veins, the right auricle and the right ventricle, as well as the pressure of the spinal fluid. The arterial pressure rises only with a relatively small elevation in the intrapulmonic pressure – –to 25–30 mm of Hg, and at higher levels it falls. The experiments of G. V. Altukhov and N. A. Agadzhanyan established that the involvement of a compensatory mechanism, creating a counterpressure in the body during intensification of the pressure in the lungs, leads to a greater elevation in the venous pressure and the pressure in the right and left ventricles. This makes it possible to maintain the arterial pressure at a sufficiently high level

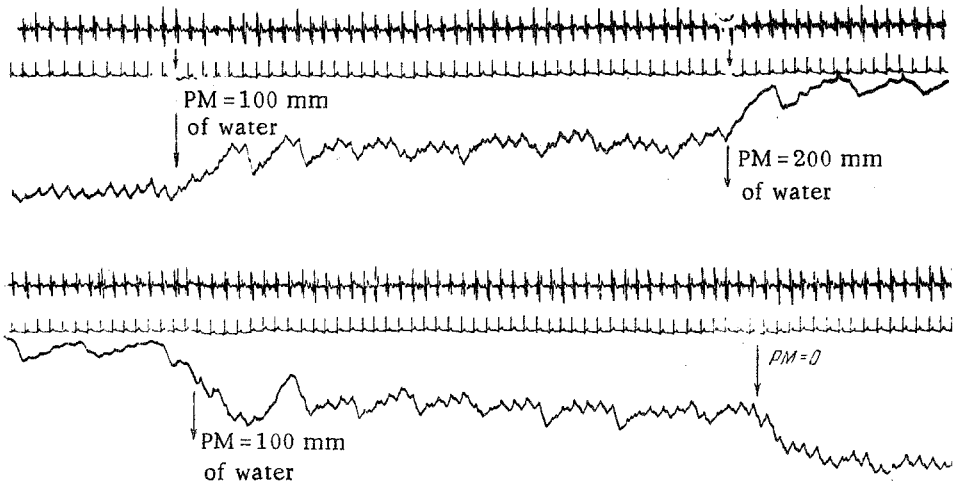

Fig. 1. Changes in the plethysmogram during respiration under excess pressure of 100 and 200 mm of water. Meaning of the curves (from above downward): BKG, EKG, plethysmogram; PM – oxygen pressure in the mask (in Figs. 2 and 3 – the same). The arrows indicate the moments when the oxygen pressure in the mask changed.

during elevated intrapulmonic pressures. According to the data in the literature, a reduction in the rate of blood flow was observed both in the pulmonic and systemic circulation during oxygen respiration under excess pressure. This was established in investigations on man using the method of decholine injection [6], and on animals by means of injections of radioactive phosphorous [4].

As can be seen from the literature presented, the presence of an excess pressure in the lungs causes essential changes in the hemodynamics. Thus, a study of the blood circulation during respiration under excess pressure has great practical significance.

The purpose of this investigation was to study the blood circulation in man during oxygen respiration under excess pressure. The work was done under terrestrial conditions, both with the use of apparati exerting counter-pressure on the surface of the body and without the use of these apparati.

METHOD

Eleven young, healthy males took part in the work; eighteen investigations were carried out. The individuals under investigation breathed oxygen under excess pressure of 8–15, 20–25 and 40 mm Hg and higher. Before the beginning of the investigation the oxygen mask was carefully fitted. The excess pressure in the lungs was created by an oxygen apparatus, permitting the generation of the necessary pressure under the mask and in the pressure system of the compensatory apparatus separately and simultaneously.

In order to evaluate the state of the blood circulation in the subjects we recorded the electrocardiogram (EKG), ballistocardiogram (BKG), pulse volume, plethysmogram, and respiratory excursions of the thoracic cavity; we also determined the levels of the minimal and maximal arterial pressure and the rate of propagation of the pulse wave.

The EKG was recorded from the three standard leads. The BKG was registered on a high-frequency ballistocardiograph (Starr table). The tracing was made during respiratory stops at inspiration and expiration.

To determine the rate of pulse wave propagation we recorded 2 sphygmograms at the same time. We placed pneumatic cuffs at the proximal portion of the thigh and the distal area of the calf of the subjects under investigation, connecting them to piezoelectric crystal datchiki. The plethysmogram recorded from the calf using a tensometric manometer.

RESULTS

It was established that even a relatively small elevation in the intrapulmonic pressure (8–15 mm Hg) causes

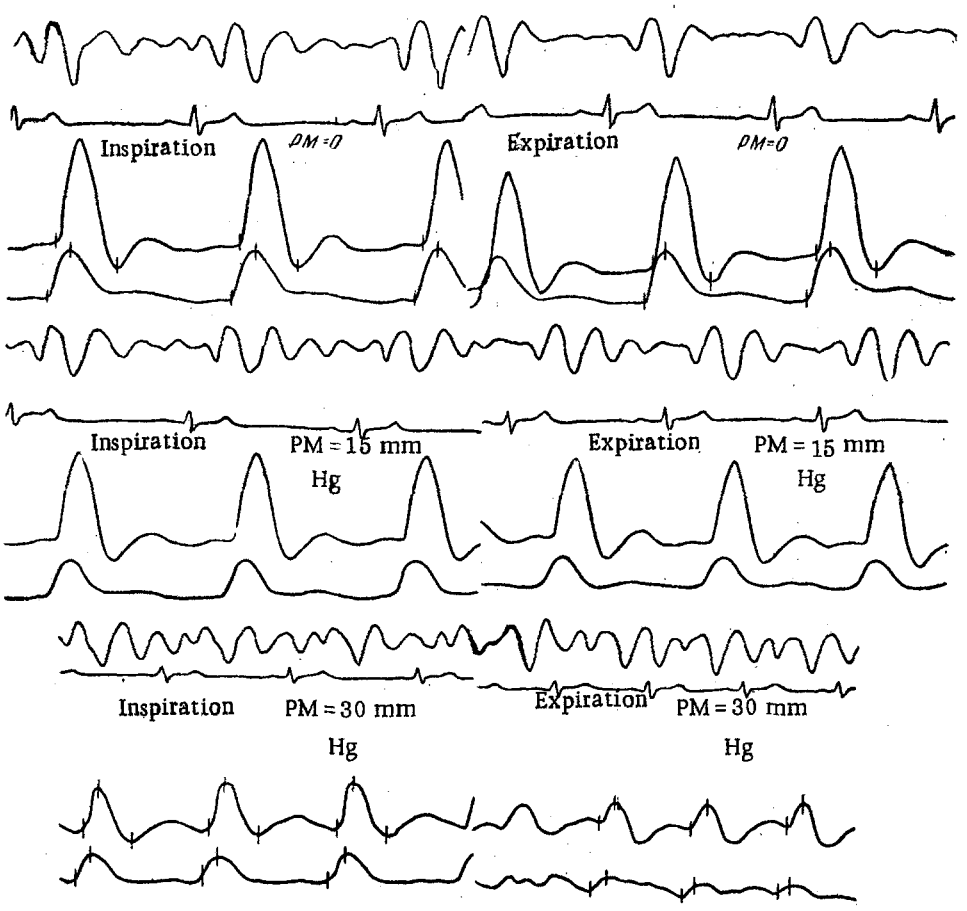

Fig. 2. Changes in the BKG, EKG, amplitude of the pulse wave, and rate of its propagation during oxygen respiration under excess pressure of 15 and 30 mm Hg, without the use of the compensatory apparatus. Meaning of the curves (from above downward): BKG, EKG, sphygmogram at the calf, sphygmogram at the thigh.

a marked shift in the hemodynamics, manifested by an increase in the volume of the extremities, a decrease in the amplitude of the pulse wave, a rise in the rate of the pulse wave propagation, and, in certain cases, a small decrease in the systolic spikes of the BKG. Evaluating these changes in the blood circulation, which in the majority of subjects were weakly demonstrated, it can be concluded that it is still possible for the organism to adapt to these conditions of respiration for a relatively long period of time. With an increase in the excess pressure within the lungs to 20–25 mm Hg, clearly manifested disturbances in the blood circulation arose in those cases where we did not employ the apparatus exerting counter-pressure on the body (Fig. 1 and 2). The volume of the extremity markedly increased (it should be noted that the plethysmographic effect exactly followed the magnitude of the excess intrapulmonic pressure), the rate of the pulse wave propagation along muscular type vessels considerably increased (by 20%), and, at the same time, the rate of pulse wave propagation in the elastic type vessels practically remained unchanged. The arterial pressure–both minimal and maximal–was somewhat elevated.

The data from the electrocardiographic and ballistocardiographic investigation testify to changes in the functional state of the heart in association with its rising load. In the majority of individuals under observation the heart contractions were noted to become more rapid; in this case the relationship of the H–K interval in the BKG to the Q–T interval in the EKG essentially did not change. Shortening of the cardiac cycle was basically caused by shortening of diastole.

Decrease in the amplitude of the systolic spikes in the BKG (assuming the durations of mechanical systole constant) was probably related to the decrease in the systolic volume (Fig. 2). The moderate elevation in the frequency of cardiac contractions already showed itself to be inadequate for the maintenance of the cardiac minute volume, as is shown by the data of our investigation and has been noted earlier by several foreign authors [7, 10, etc.].

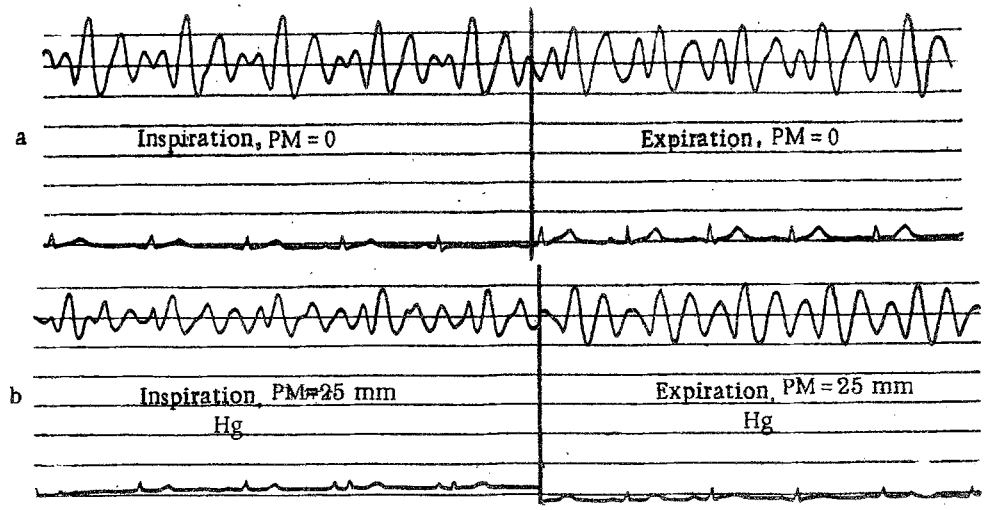

Fig. 3. Changes in the amplitude of the spikes in the systolic complex of the BKG in association with the phases of respiration. a—normal relationships; b—during respiration under excess pressure of 25 mm of Hg. Upper curve—BKG; lower curve—EKG.

It is interesting that during respiration under excess pressure the relationship between the amplitudes of the systolic spikes in the BKG during the various phases of respiration is distorted (Fig. 3). As is well known, the systolic spikes of the BKG in the inspiratory phase normally have a somewhat higher amplitude than in the phase of expiration. During respiration under excess pressure, on the other hand, the amplitude of the systolic spikes in inspiration was lower by 20—25% than in expiration; thus, the specific cardiac output during expiration was greater than during inspiration. This phenomenon can be explained by the fact that cardiac filling during the expiratory phase becomes greater than in the inspiratory phase as a result of the marked tension in the skeletal muscles and the elevation in the intraperitoneal pressure, and the sucking effect during inspiration, which exists under normal conditions of respiration, disappears.

The exertion of counter-pressure on the surface of the body in the case of oxygen respiration under excess pressure (30—40 mm Hg) normalizes the blood circulation to a significant degree. With counter-pressure the conditions of the peripheral circulation improve, and the indices characterizing the cardiac activity return to normal (BKG, EKG). The amplitude of the systolic spikes in the BKG rises, and the magnitude of the cardiac minute volume becomes close to normal. However, the character of the change in the BKG during the different phases of respiration remains the same as in oxygen respiration under excess pressure without compensation.

The dextrogram in the EKG completely or partially disappears, and the sinus tachycardia becomes less manifest.

The exertion of counter-pressure on the body leads to a marked decrease in the rate of pulse wave propagation along the muscular type vessels (femoral artery), which is related to the effect of the counter-pressure itself, tending to reduce the tone of the vessel. At higher levels of excess pressure in the lungs (50—60 mm Hg and above), despite the use of a system exerting counter-pressure on the body, the distinct shifts again appear in all the indices investigated by us characterizing the functional state of the cardio-vascular system (decrease in the spikes of the BKG and distortion of their form, sinus tachycardia, depression of the T wave, dextrogram in the EKG); this indicates the development of functional inadequacy in the cardio-vascular system.

With an elevated pressure in the lungs the increase in the intra-alveolar pressure leads to a rise in the pressure within the cappillaries of the pulmonic circulatory system. The elevated pressure in the pulmonary capillaries effects the pressure in the pulmonary artery and the right heart. In addition, the rising intrathoracic pressure is transferred to the major vessels of the thoracic cavity, modifying the tension in the wall of the vessels, although to a varying degree.

The increase in the volume of the extremity observed in the experiments, associated with a relatively small

(8 mm Hg) elevation in the intrapulmonic pressure, is apparently caused by the increased resistance to the blood flow returning to the heart, a result of the elevated pressure in the venae cavae and the right auricle.

The shifts in the blood circulation during respiration under excess pressure begin practically simultaneously with elevation of the pressure in the lungs, which suggest the important role which is played by the direct effect of mechanical factors. It is necessary to take into consideration in order to correctly understand the conditions under which the reflex regulation of the vascular tonus and cardiac function is developed. The compensatory reorganization of the circulatory reflex regulation allows the individual to adapt to the new conditions of respiration for a longer or shorter period of time only at relatively low levels of excess intrapulmonic pressure (10–15 mm Hg).

Reflexes from the pulmonic vessels probably play a definite role in the groundwork of the adaptive circulatory reorganization during oxygen respiration under excess pressure. As was shown in the investigation of V. V. Parin [5], Schwiegk [11], etc., these reflexes can provide "relief" for the pulmonic circulation.

The exertion of counter-pressure on the body leads to preservation of the effective circulating blood volume; this permits adequate blood circulation at relatively low levels of excess oxygen pressure in the lungs. This fact serves as a basis for postulating that use of "pressurized" suits can be used clinically for therapeutic purposes associated with certain cases of cardio-vascular insufficiency.

SUMMARY

To investigate the shifts occurring in hemodynamics during oxygen respiration under excessive pressure, the authors studied the electrocardiograms, ballistocardiograms, and plethysmograms and determined the pulse wave velocity. Respiration under a pressure of 8-15 mm Hg produced hemodynamic shifts manifested in the increase of the volume of extremities and pulse wave velocity and reduction of systolic complex of the ballistocardiogram; however, these changes were insignificant. With a pressure of 20-25 mm Hg, the shifts were pronounced in all the persons tested. With a pressure of 30-40 mm Hg, counter-pressure exerted on the body had a marked effect on normalization of circulation. With respiration under 50-60 mm Hg pressure or more, hemodynamic disturbances could not be completely eliminated, even by counter-pressure on the body.

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