These results, together with data in the literature [1], suggest that chronic loss of bile leads to a significant disturbance of the structure and function of the outer membranes of cells of the intestinal epithelium.

LITERATURE CITED

1. Biological Membranes [in Russian], Moscow (1973).
2. S. F. Kuz'mina, "Hydrolytic and transport functions of the small intestine after ligation of the common bile and pancreatic ducts," Author's Abstract of Candidate's Dissertation, Tashkent (1974).
3. G. I. Loginov, S. F. Kuz'mina, and M. I. Talzhibaeva, Fiziol. Zh. SSSR, 62, No. 6, 913 (1976).
4. A. M. Ugolev, L. F. Smirnov, and N. M. Timofeeva, Dokl. Akad. Nauk SSSR, 221, No. 4, 984 (1975).
5. G. K. Shlygin and L. S. Vasilevskaya, Byull. Éksp. Biol. Med., No. 11, 46 (1967).

EFFECT OF OXYGEN AND HYPERGRAVITATION ON ALVEOLAR SURFACTANT

M. A. Tikhonov, O. V. Petrov, A. V. Kondakov, and E. V. Loginova

UDC 612.212.014.1.014.462.8-06: [612.273.1+612.014.47

The effect of prolonged (up to 66 h) exposures to pure oxygen and of brief (5 min) inhalation of oxygen combined with gravitational overloads produced by longitudinal acceleration (5g) on the surface tension and surface potential of the alveolar washings from the lungs of albino rats was studied. Under both experimental conditions atelectasis of the lungs was formed, with a decrease in the surface activity of the surfactant. The mechanisms of the changes in surfactant activity during hyperoxia, alone and combined with hypergravitation, are discussed.

KEY WORDS: hyperoxia; acceleration; atelectasis; surfactant.

It is now generally accepted that prolonged normobaric hyperoxia, lasting several hours, causes disorders of respiration (the Lorrain-Smith syndrome) and atelectases of the lungs as a result of the toxic action of oxygen on lung tissue [1, 3, 5]. It is also known that similar respiratory changes may arise following brief (for a few minutes) inhalation of pure oxygen, but combined with hypergravitation caused by exposure to longitudinal ($+G_Z$) or transverse ($+G_X$) acceleration [6, 8]. Atelectases of the lungs are considered to develop under these conditions on account of the increased regional inequality of ventilation of the lungs, their deformation, and the rapid absorption of oxygen by blood in the pulmonary capillaries from the unventilated alveoli [8, 14].

At the same time, absence or inactivation of the surface-active substance of the lungs (surfactant) is known to facilitate the formation of atelectases [4, 13, 15]. In particular, a decrease in alveolar surfactant activity has been found after prolonged exposures to oxygen [7, 10, 12].

Since both prolonged and brief exposures to oxygen, if combined with acceleration, lead ultimately to the development of atelectases of the lungs, changes in the surface-active properties of the surfactant are to be expected in both experimental situations. In accordance with this hypothesis the state of the surfactant was studied in the lungs of rats exposed for different times to oxygen and also in rats exposed to acceleration combined with inhalation of air and of pure oxygen.

EXPERIMENTAL METHOD

Experiments were carried out on 53 male albino rats weighing 140-170 g. In the experiments of series I (17 rats in the experimental and five in the control group) the animals were kept for between 6 and 66 h in a chamber through which pure oxygen was passed at the rate of 0.5 liter/min per rat. Carbon dioxide was absorbed by the KhPI absorber.

(Presented by Academician of the Academy of Medical Sciences of the USSR V. N. Chernigovskii.) Translated from Byulleten' Éksperimental'noi Biologii i Meditsiny, Vol. 86, No. 7, pp. 24-27, July, 1978. Original article submitted December 18, 1977.

0007-4888/78/8607-0862 $07.50

TABLE 1. Effect of Different Exposures to Oxygen on Indices of Surface Activity of Lung Surfactant (M ± m)

Parameter tested	Control	Experimental group, period of exposure to oxygen, h			
		6	22	44	66
γ_{min}, dynes/cm	22±1,5	21±0,4	20±2	25±4,8	39±1*
K	0,91±0,05	0,99±0,02	1,08±0,20	0,84±0,14	0,39±0,02*
S, conventional units	82±8	72±2	88±8	76±13	41±4*
A_φ, mV	159±10	145±3	147±16	135±12	58±7*
S_φ, conventional units	33±6	24±2	34±7	22±9	8,8±0.7*

Legend. Here and in Table 2, asterisk indicates values for which $P < 0.05$ compared with control.

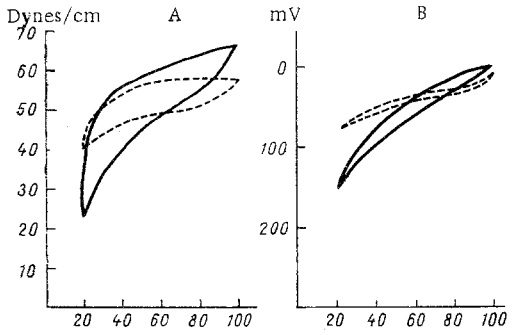

Fig. 1

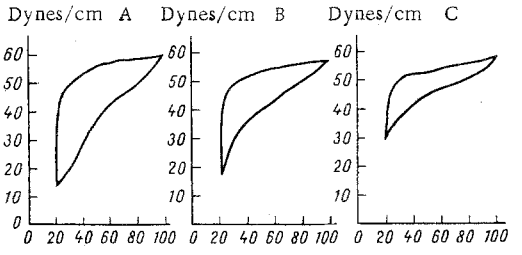

Fig. 2

Fig. 1. Changes in surface tension (A) and surface potential (B) of alveolar washings. Here and in Fig. 2: continuous lines denote hysteresis loops of surface tension and surface potential in control group; broken lines – the same after exposure for 66 h. Abscissa, values of S (in %).

Fig. 2. Changes in surface tension of alveolar washings in control (A) and after exposure to accelerations ($+G_Z$) combined with inhalation of air (B) and oxygen (C).

In the experiments of series II (24 animals in the experimental and seven in the control group) the rats were fixed to a turntable with a radius of 4.2 m, in a frame with a system for continuous supplying of oxygen, and exposed to longitudinal accelerations ($+G_Z$) with an intensity of 5 g for 5 min while inhaling air or 98.5% oxygen.

Washings were obtained from the alveolar surface, containing surfactant [15], 15-30 min after the end of exposure from the rats while anesthetized with pentobarbital. The surface tension of the alveolar washings was measured on a modified Wilhelmy's balance [2]. The surface activity of the washings was estimated from the minimal surface tension (γ_{min}), the stability index of the surfactant (K) [4], calculated by the equation: $K = 2(\gamma_{max} - \gamma_{min})/(\gamma_{max} + \gamma_{min})$, and the area of the hysteresis loop on the graph of area plotted against surface tension (S). Further information on the state of the surfactant was obtained by simultaneously recording the surface potential (A_φ) and the area of the hysteresis loop (S_φ) of the surface potential, on the grounds that the possible oxidation of phospholipids must change the dipole moment of the surface-active molecules and must lead to a change in the amplitude of the surface potential. The method of measuring surface tension and the surface potential was described previously [2]. In some of the animals investigated the lungs were examined morphologically (macroscopically and histologically).

EXPERIMENTAL RESULTS

As Table 1 shows, keeping the rats in an atmosphere of oxygen for between 6 and 44 h caused no significant changes in surface tension or surface potential of the alveolar washings. No macroscopic evidence of edema of the lung and no atelectases likewise were discovered in them.

When exposure to oxygen was prolonged to 66 h a significant ($P < 0.001$) increase in γ_{min} and a decrease in the amplitude of the surface potential A_φ and in the area of the hysteresis loop S_φ were observed (Fig. 1). Morphological examination of these animals revealed multiple subsegmental atelectases, massive transudation into the pleural cavity, and evidence of pulmonary edema.

TABLE 2. Effect of Accelerations (+G_z) during Inhalation of Air and Oxygen on Surface Activity of Lung Surfactant (M ± m)

Parameter tested	Control	Inhalation of		24 h later
		air	oxygen	
γ_{min}, dynes/cm	17,0±2,1	15,7±2,3	24,8±2,2*	15,1±2,5
K	1,11±0,08	1,13±0,10	0,85±0,08*	1,16±0,11
S, conventional units	117±13	122±12	97±9	131±18

In the experiments of series II with inhalation of pure oxygen combined with gravitational overloads, despite the short duration of exposure (5 min) massive atelectases also were observed in all the experimental animals, but without any accompanying signs of pulmonary edema or of transudation into the pleural cavity. Meanwhile, as is clear from Table 2, combined exposure for 5 min to the action of oxygen and hypergravitation was accompanied by changes in the indices of the surface-active properties of the surfactant similar in magnitude and direction to those following exposures of 66 h to pure oxygen.

However, exposure to identical levels of acceleration combined with inhalation of atmospheric air was not followed by any change in the morphological structure of the lungs or in the indices of surface activity of the surfactant (Fig. 2).

The indices of surface tension of the surfactant did not differ significantly 24 h after exposure to acceleration in an atmosphere of oxygen from the analogous values in the control group, and morphological examination revealed no atelectases in the lungs of the experimental animals. The readily reversible atelectases of the lungs found in rats after exposure to +G_z in an atmosphere of oxygen and the changes in surface tension of the alveolar surfactant thus support the hypothesis that surface-active substances participate in the formation of atelectases of the lungs.

Alveolar surfactant is known to prevent collapse of the lungs and to maintain the morphological stability of the alveoli by reducing the surface tension when the volume of the lungs decreases [4, 15]. Since compression of the lungs during hypergravitation and absorption of oxygen from unventilated alveoli cause a reduction in their volume [9], it can tentatively be suggested that compression of the alveoli and a reduction in the area of the surfactant film covering their walls cause partial desorption of molecules of surface-active substance into the hypophase, and so reduce the surface activity of the surfactant.

The possible displacement of some surfactant molecules into the hypophase of the alveolar lining may perhaps also explain the gradual collapse of the alveoli which takes place during prolonged shallow breathing [13].

By contrast to this predominantly physical mechanism of changes in surfactant activity, long exposures to oxygen are accompanied by injury of the endothelium of the pulmonary capillaries, an increase in the permeability of the alveolar-capillary membrane, and transudation of plasma into the alveoli. The intraalveolar fluid and plasma components of the blood cause washing out and inactivation of the surfactant [11]. The presence of massive transudation into the pleural cavity and pulmonary edema in rats after exposure to oxygen for 66 h can therefore be explained by the changes observed in the surface potential and surface tension of the alveolar surfactant.

Both prolonged exposures to oxygen and short exposures accompanied by hypergravitation thus lead to changes in the surface-active properties of the lung surfactant. However, the mechanisms lying at the basis of these changes are evidently different in nature. During hypergravitation they probably consist essentially of desorption of surfactant molecules into the hypophase of the alveolar film on account of its compression, whereas in the case of prolonged exposures to oxygen they consist of washing out and inactivation of the alveolar surfactant by a transudate of blood plasma.

LITERATURE CITED

1. A. M. Genin, M. A. Tikhonov, V. B. Malkin, et al., Izvest. Akad. Nauk SSSR, No. 3, 378 (1973).
2. O. V. Petrov, Byull. Éksp. Biol. Med., No. 2, 118 (1974).
3. M. A. Tikhonov and E. V. Loginova, Izvest. Akad. Nauk SSSR, No. 1, 81 (1977).
4. J. A. Clements, R. F. Hustead, and R. P. Johnson, J. Appl. Physiol., 16, 444 (1961).
5. A. B. DuBois, T. Turaids, R. E. Mammen, et al., J. Appl. Physiol., 21, 828 (1966).
6. J. Ernsting, Proc. Roy. Soc. Med., 53, 96 (1960).

7. S. T. Giammona, D. Kerner, and S. Bondyrant, J. Appl. Physiol., 20, 855 (1965).
8. D. H. Glaister, Brit. J. Hosp. Med., 3, 635 (1969).
9. J. B. Glazier and J. M. B. Hughes, Aerospace Med., 39, 282 (1968).
10. J. D. Hackeny, C. R. Collier, D. Conrad, et al., Clin. Res., 11, 91 (1963).
11. J. W. C. Johnson, S. Permutt, and J. H. Sipple, J. Appl. Physiol., 19, 769 (1964).
12. C. Lee, J. H. Lyons, et al., J. Thorac. Cardiovasc. Surg., 153, 759 (1967).
13. R. E. Pattle, Arch. Environ. Health, 14, 70 (1967).
14. H. Rahn and L. Farhi, Fed. Proc., 22, 1035 (1963).
15. E. M. Scarpelli, The Surfactant System of the Lung, Philadelphia (1968).

BLOOD GASES IN CRANIOCEREBRAL HYPOTHERMIA

Sh. D. Penner and B. N. Nikitenko

UDC 615.832.9.032.81.015.4:612.127

Experiments on dogs showed that during craniocerebral cooling from 38 to 28°C the partial pressure of oxygen in the blood increases whereas that of carbon dioxide decreases. In deep hypothermia (24°C) the concentration of the blood gases is lower than at 28°C, but is still higher than initially. These changes are due to the long persistence of adequate pulmonary ventilation in the hypothermic organism.

KEY WORDS: hypothermia; partial pressure of oxygen; partial pressure of carbon dioxide.

An important advantage of craniocerebral hypothermia is its ability to depress the level of oxidative processes considerably. In this connection it is interesting to investigate the intensity of carbon dioxide formation and also the pattern of oxygen transport and utilization in the hypothermic organism. For this purpose the partial pressures of oxygen and carbon dioxide in the blood were studied in dogs during craniocerebral hypothermia.

EXPERIMENTAL METHOD

Experiments were carried out on 25 dogs weighing 10-15 kg. After trimeperidine premedication and intravenous hexobarbital anesthesia the animals were transferred to basal ether-air anesthesia. The animal's head was placed in the Kholod-2F factory-made hypothermic apparatus. Mixed venous blood and blood from the femoral artery were taken before exposure to cold and as the body temperature fell at 38, 36, 34, 30, 28, and 24°C. The blood gases were analyzed by the AZIV-2 apparatus. The partial pressure of oxygen in the arterial (p_aO_2) and venous (p_vO_2) blood was measured by means of the polarographic attachment to the apparatus. The partial pressure of carbon dioxide (p_aCO_2 and p_vCO_2) was determined by means of the Siggaard-Andersen nomogram, with correction for temperature by Rosenthal's method [9].

The depth and frequency of the respiratory movements, the minute volume of respiration (MVR), and the oxygen consumption were measured with the META 1-25 spirograph.

The results were subjected to statistical analysis by the Minsk-32 computer.

EXPERIMENTAL RESULTS AND DISCUSSION

As the body temperature fell from 38 to 28°C the partial pressure of oxygen in the arterial and venous blood increased. At the end of cooling (24°C) the partial pressure of the blood gases was lower than at 28°C, although still higher than initially (Table 1).

The pattern of oxygen transport is closely connected with the processes of entry of the gas into the body. During local brain cooling the depth and frequency of respiration change, so that MVR in the case of superficial or moderate hypothermia (36-32°C) was higher than initially, whereas during moderate or deep hypothermia it

Department of Human Physiology, Vladimir Pedagogic Institute. (Presented by Academician V. N. Chernigovskii.) Translated from Byulleten' Éksperimental'noi Biologii i Meditsiny, Vol. 86, No. 7, pp. 27-29, July, 1978. Original article submitted July 11, 1977.

0007-4888/78/8607-0865$07.50