Changes of the Oxygen Capacity of the Blood under the Influence of a Constant Magnetic Field

V. I. Skorik, A. I. Zhernovoi, L. M. Sharshina, N. A. Kulikova, Z. V. Rudakova, and V. A. Chirukhin

UDC 616.152.21-02:615.847.8]-07

Translated from *Byulleten' Eksperimental'noi Biologii i Meditsiny*, Vol. 116, № 10, pp. 386-388, October, 1993
Original article submitted June 16, 1993

Key Words: *magnetic field; blood; hypoxia*

In our previous studies [6,7] we established that the time of endurance of mortal hypoxia may be prolonged by exposure of the blood circulating outside the body to a constant magnetic field (CMF). Thus, there are grounds for speaking of a mediated (via the blood) effect of the above factor on the whole organism. A relatively large number of possible mechanisms of such an effect are known [1-5, 8-11].

In the present study we attempted to pinpoint one of these mechanisms, i.e., to reveal some specificities of changes in the oxygen capacity of the blood exposed to a CMF and O_2.

MATERIALS AND METHODS

With the use of a model of apneic hypoxia in two series of experiments on dogs, and in stand tests with the use of human donor blood, the O_2 content was determined in the blood during and after exposure to the combined action of a CMF and O_2. In the control (8 dogs) and main (12 dogs) series the ketamine-anesthetized and ditiline-relaxed animals were intubated and placed on artificial lung ventilation by using an RO-6 apparatus; a veno-venous bypass was applied for pumping (volume blood flow rate 20-25 ml/min) the blood from the vena cava inferior to the left atrium. Against the background of myorelaxation, after 3 min of O_2 delivery to the RO-6 circuit, hypoxia was reproduced by switching off the artificial lung ventilation and by occluding the intubation tube. In the main series, the main line of the extracorporeal circuit was placed between magnetized plates which maintained a CMF with an induction of 0.5 T. Prior to O_2 delivery to the RO-6 circuit, the duration of exposure was 20-25 min and then 2 min during respiration with O_2. In the present study the blood gases were measured before O_2 delivery to the RO-6 apparatus was cut off. We performed 35 measurements (12 in the control and 23 in the main series). The experimental procedure for conserved donor blood (shelf life 5-7 days) was the following: a 20-25-min exposure of the blood to the CMF (induction of 0.5 T) in a closed vessel, oxygenation of the blood to attain the 100% level of HbO_2 and a pO_2 value of 313+21 mm Hg, and determination of pO_2 during exposure to the CMF and 3 and 10 min after removal from the field (34 measurements in the main series and 23 in the controls not exposed to the CMF). The content of gases and the acid-base equilibrium were determined in the blood with a Radiometer AME-1 apparatus, an Elema oximeter, and a Van Slyke apparatus. The results were statistically processed using Student's t test.

RESULTS

In previous experiments on ketamine-anesthetized, ditiline-relaxed dogs placed on controlled artificial

P. A. Kupriyanov Clinic of Cardiovascular Surgery, S. M. Kirov Academy of Military Medicine, St. Petersburg. (Presented by V. V. Kupriyanov, Member of the Russian Academy of Medical Sciences)

0007-4888/93/0010-1243$12.50

ventilation with an RO-6 apparatus, we studied the times of endurance of mortal hypoxia caused by switching off the artificial lung ventilation in animals of the control and main series. In the latter series the extracorporeal veno-venous bypass was exposed to a CMF.

The animals in the main series were shown to endure mortal hypoxia 1.5 times longer. At the same time, the levels of HbO_2 and pO_2 in the arterial and venous blood remained quite high considerably longer than those in the control experiments. The "critically low point" in the blood gases (at which the arterial and venous level of HbO_2 and pO_2 became equal) occurred in the 17th-20th min in the main series and in the 11th-13th min in the control.

We can assume that extracorporeal exposure of the blood to a CMF markedly alters the mechanism of its gas-transporting function. At the stage prior to cutting off the respiration and placing the animal in the state of hypoxia, the studies of oxygen capacity in the arterial blood during its simultaneous exposure to the CMF and O_2, which were performed using modern gas-analyzing devices and the Van Slyke apparatus, showed the following.

For exposure to the CMF, the total amount of O_2 bound to Hb and dissolved in the plasma after a 3-min respiration with O_2 was 24.65±0.80 vol.% at a Hb level of 149.1±4.7 g/liter vs. 22.13±0.55 vol.% at a Hb level of 148.3±3.2 g/liter in the control series. The difference is statistically reliable ($p<0.05$) with respect to O_2, but not to Hb. At the same time, during the first 10 min after the cessation of exposure to the CMF, the volume of plasma-dissolved O_2 increased almost 1.5-fold. For instance, after 10 min, the values of this parameter in the main and control series were 2.01±0.13 and 1.35±0.16 vol.% ($p<0.001$), respectively. Taking into account that after exposure of the blood to the CMF the increase in the volume of O_2 bound to Hb constitutes only 10% (the ratio of this value obtained on the Van Slyke apparatus to the calculated value is 1.14±0.003 in the main series and 1.04±0.008 in the control, $p<0.001$), one may assume that the additional bond between O_2 and Hb resulting from CMF exposure is highly labile. After the cessation of exposure to the magnet it is broken, and O_2 escaping Hb goes over to the soluble state.

This is confirmed by the data of stand tests of conserved blood, which show that the pO_2 of oxygenated blood exposed to a CMF and that of the same blood removed from the field differ one from another. The following experimental model was chosen. Equal amounts of venous blood and of O_2 were taken in a closed vessel (a 20-ml syringe); the syringe was placed in a CMF with induction of 0.5 T for 20-25 min; the maximum O_2 saturation of the blood was achieved by smoothly shaking the syringe; after a 100% level of HbO_2 and a pO_2 of 313±2 mm Hg were attained, the gaseous O_2 was expelled from the syringe, and without the syringe being removed from the CMF, repeated measurements of HbO_2 and pO_2 were performed. The same was done 3 and 10 min after terminating the exposure of the blood to the CMF.

It was found that along with preservation of the 100% level of HbO_2 the pO_2 values naturally decreased for some time, but the rate of this decrease was unequal in the blood exposed and not exposed to the CMF. For example, after the vessel was removed from the magnetic field, the pO_2 values in the blood samples were 1.6 and 1.4 times as high as in the control on the subsequent 3rd-10th min, respectively.

The slowed tempo of decrease of the pO_2 values after "magnetizing" may be regarded as a manifestation of an increase in its oxygen capacity caused by the transfer (a specific transmission of excessive volume) of O_2 to the soluble state.

The effect of exposure to a CMF on the oxygen capacity of the blood may be explained by the Perutz hypothesis [10] on the relationship between the oxygen-binding capacity of Hb and its spin state. Naturally, one may speculate that the effect of the magnet on the blood transforms its less active state (T form) into a more active one (S form) due to spin reorientation.

In the assessment of the role of blood "magnetization" in prolonging endurance of mortal hypoxia, the blood-mediated effect of this factor on the organism, resulting from possible changes of rheological and other properties of the blood, cannot be ruled out [1,2,4,8], as well as an indirect effect on the central neurohumoral component of regulation [3,8]. However, the above data enable us to regard the effect of CMF on the gas-transporting function of the blood as one of no lesser importance, and perhaps even as the main one.

Even a short-term increase of the affinity of Hb to O_2 results in more active binding of the latter in the lungs (or, for extrapulmonary oxygenation, in the devices for extracorporeal mass exchange) and to an increase in the total oxygen balance of the organism, this providing for longer endurance of apnea and developing hypoxia.

This phenomenon of additional O_2 binding by the blood exposed to a CMF may be used in transfusions of autologous and donor blood in surgical and reanimatological practice.

REFERENCES

1. L. S. Jackson and I. L. Brandt, in: *Trudy Kazan. Nauchno-Issled. Inst. Klin. Eksp. Khirurgii*, № 37, 76-80 (1986).
5. G. V. Lud, *Zdravookhr. Belorussii*, № 8, 47-49 (1986).
3. Z. N. Nakhil'nitskaya, *Probl. Kosm. Biol. Med.*, **37**, 116-124 (1978).
4. Yu. A. Novikov, *Anesteziol. Reanimatol.*, № 3, 21-23 (1982).
5. V. L. Semenov, *Pat. Fiziol. Eksp. Ter.*, №1, 56-61 (1987).
6. V. I. Skorik, A. I. Zhernovoi, L. M. Sharshina, *et al.*, *Byull. Eksp. Biol. Med.*, **115**, № 1, 17-20 (1993).
7. L. I. Tyvin and V. A. Gusel', *Ibid.*, № 9, 29-31 (1983).
8. M. M. Yakovleva, *Physiological Mechanisms of the Effect of Electromagnetic Fields* [in Russian], Leningrad (1973).
9. B. Grenet and R. Makowski, *Acta Physiol. Polonica*, **30**, № 1, 10-11 (1972).
10. M. Perutz, *Nature*, **237**, № 5357, 495-499 (1972).

Effect of Chorionic Gonadotropin on the Formation of the Secondary Immune Response

S. V. Shirshev

UDC 612.018.2:612.017.12

Translated from *Byulleten' Eksperimental'noi Biologii i Meditsiny*, Vol. 116, № 10, pp. 388-390, October, 1993
Original article submitted April 28, 1993

Key Words: *chorionic gonadotropin; sex steroids; antibody-producing cells; secondary immune response*

Chorionic gonadotropin (CG) synthesized by human placenta, controls hormone-dependent mechanisms of the growth and development of the fetoplacental complex from the moment of ovum implantation to birth [1]. A semiallogenic fetus and placenta are recognized by maternal lymphocytes as foreign antigenic material but are not rejected by them for a number of reasons [4]. An important role is played here by pregnancy hormones which are potent immunosuppressants [5].

CG is a highly active modulator of immune reactions. A dose-dependent suppression of the cellular [7,11] and humoral [2,9] immune response by the hormone has already been demonstrated, but as a rule only with respect to the effect of CG on the primary immune response of immunocompetent cells. Relationships between the maternal immunity system and the fetoplacental complex include not only the primary, but also a secondary, or anamnestic, immune response, which becomes dominant in repeated pregnancies [3].

We have investigated the immunomodulatory effects of CG and ovarian sex steroids as its possible mediators on the genesis of the anamnestic immune response.

MATERIALS AND METHODS

Experiments were carried out with female CBA mice weighing 18 to 22 g. One group of animals was subjected to bilateral ovariectomy under ether anesthesia. These animals were used in experiments one month after the operation.

The scheme of secondary immune response induction consisted of two stages. At stage I the animals were intraperitoneally immunized with sheep erythrocytes (SE) in a dose of 2×10^8 cells and 20 days later (stage II) were again immunized with the same dose of SE. The secondary immune response was assessed on day 5 after reimmunization by local hemolysis in agarose gel [8]. Antibody (IgG) producing cells were determined by a

Department of Biochemistry, Perm State Medical Institute (Presented by K. P. Kashkin, Member of the Russian Academy of Medical Sciences)

0007-4888/93/0010-1245$12.50