

Effect of Stationary Magnetic Field on *in Vivo* Oxygen Binding by Blood

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The rates of oxyhemoglobin drop and CO₂ release in dogs with magnetized and nonmagnetized blood were compared. Stationary magnetic field enhances blood oxygen capacity by no less than 24%. A hypothesis was proposed on formation of bioxyhemoglobin molecules under the effect of magnetic field, in which hem binds two O₂ molecules.

Key Words: *oxygen; blood; magnetization; oxygen capacity of blood; bioxyhemoglobin*

We previously showed that blood magnetization with its subsequent oxygenation improved resistance to hypoxia via increasing in oxygen binding capacity of the blood [3-6]. The aim of this work is to assess the amount of additional oxygen in oxygenated magnetized blood on the basis of the balance between oxyhemoglobin utilization and CO₂ release during hypoxia.

MATERIALS AND METHODS

The study was carried out on 16 outbred dogs (body weight 7-9 kg) under ketamine (3 mg/kg) anesthesia and suxamethonium myorelaxation (1 mg/kg). After intubation the dogs were transferred to jet ventilation performed with an RO-6 apparatus. The femoral and jugular veins were shunted for blood transfer (an AT-1 pump with tygon tubes) from the inferior vena cava to the right atrium. The extracorporeal circuit contained isotonic solution (150 ml). The blood flow rate in the shunt was 20-25 ml/min/kg.

After stabilization of homeostasis indices under artificial ventilation, oxygen was supplied to the PO-6 circuit for 2 min. Then ventilation was stopped during the expiration phase and the intubation tube was clamped. In the control the blood was not magnetized. In experimental dogs magnetization was started 20 min prior to oxygen supply into PO-6 apparatus and con-

tinued during hypoxia. To magnetize the blood, extracorporeal circuit was placed between the poles of a 0.5 T magnet. Immediately after arrest of ventilation and 3, 6, and 10 min after this moment, arterial and venous blood was collected to determine So₂ and Pco₂ in AME-1 analyzer (Radiometer), Elema oxymeter, and Mingograf-82. So₂ was determined by an optical method.

The average values arterial (A) and venous (V) indices So_{2A}, So_{2B}, Pco_{2A}, and Pco_{2B}, measured in 18 control and 20 experimental dogs, were used to calculate the weighted mean parameters So₂ and Po₂ for the whole organism according to formulas:

$$\begin{aligned} \text{So}_{2m} &= \frac{1}{5} \text{So}_{2A} + \frac{4}{5} \text{So}_{2B}, \\ \text{Pco}_{2m} &= \frac{1}{5} \text{Pco}_{2A} + \frac{4}{5} \text{Pco}_{2B}, \end{aligned}$$

in which the factors $\frac{1}{5}$ and $\frac{4}{5}$ are proportional to blood volume in the pulmonary and systemic circulation, respectively.

The weighted mean concentration of oxygen in oxyhemoglobin ([O₂]_{HbO₂}) was calculated by multiplying So_{2m} by oxygen capacity of nonmagnetized blood. CO₂ saturation curve [7] and Pco_{2m} were used to determine the weighted mean CO₂ concentration.

RESULTS

Table 1 shows weighted mean blood indices and gas concentrations at different time *t* after termination of ventilation.

Table 2 shows changes in $[CO_2]$ and $[O_2]_{HbO_2}$ at t_1 and t_2 calculated from the data given in Table 1: $\Delta[CO_2] = [CO_2(t_2)] - [CO_2(t_1)]$ and $\Delta[O_2]_{HbO_2} = [O_2(t_2)]_{HbO_2} - [O_2(t_1)]_{HbO_2}$, where $[CO_2(t)]$ and $[O_2(t)]_{HbO_2}$ are the concentrations of carbon dioxide and bound oxygen at the corresponding time. This table also shows averaged respiratory coefficients for different time intervals:

$$\beta_{calc} = \Delta[CO_2] / \Delta[O_2]_{HbO_2}.$$

In the control, β_{calc} measured during 0-3 min and 3-6 min intervals considerably surpassed the normal $\beta_0 = 0.8-0.9$, although during 6-10 min interval $\beta_{calc} = 0.85$, which is close to the normal value. This can be explained by the fact that during the first 6 min after termination of ventilation utilization of oxyhemoglobin-bound O_2 is partially compensated by residual alveolar oxygen, while at $t > 6$ min alveolar oxygen is completely utilized, and $[CO_2]$ increases only due to a decrease in the concentration of in oxyhemoglobin-bound O_2 . In experimental dogs β_{calc} was higher than in controls (Table 2), which can be explained by utilization of excess oxygen stored in magnetized blood during O_2 respiration before ventilation arrest apart from oxyhemoglobin-bound and alveolar O_2 .

The change in $[CO_2]$ during 0-10 min interval divided by $\beta_0 = 0.85$ yields the total amount of O_2 utilized for 10 min of hypoxia:

$$\Delta[O_2] = \Delta[CO_2] / \beta_0.$$

The difference $\Delta[O_2] - \Delta[O_2]_{HbO_2}$ in the control determines the amount of utilized alveolar oxygen, while in experimental dogs it corresponds to the total amount of utilized alveolar and excess oxygen. The amount of excess oxygen utilized for 10 min can be

found from Table 2 as the difference $\Delta[O_2] - \Delta[O_2]_{HbO_2}$ obtained in experimental and control dogs. This difference is 4.8 vol.% or 24% oxygen capacity of non-magnetized blood.

In experimental dogs, the respiratory coefficient β_{calc} measured at 6-10 min interval is by 34% higher than in the control. It means that at $t > 6$ min excess oxygen is still utilized. Therefore, this oxygen can be not completely utilized for 10 min hypoxia. In other words, the excess oxygen capacity of magnetized blood can surpass the value determined by the amount of utilized oxygen.

The binding of excess oxygen by magnetized hemoglobin varies between individuals. In some dogs magnetization only slightly improved hypoxia resistance, *i.e.* little changed oxygen capacity of the blood, while in some experiments this parameter increased 2-fold.

The increase of oxygen capacity can be explained by binding of two O_2 molecules to the iron atom of hem ferroporphyrine in magnetic field and formation of bioxyhemoglobin $[(O_2)Hem(O_2)]$. This process is similar to the formation of hydroxyhemoglobin, hemochroms, dicyanhems, in which two ligand molecules are attached to the hem iron atom at the coordinate locations 5 and 6 [1]. These compounds are formed in the presence of a surplus of ligand molecules in the plasma, and binding of extra O_2 molecule requires oxygenation. The hemochroms of pyridine $[(Py)Hem(Py)]$ and imidazole $[(Im)Hem(Im)]$ react with O_2 to form $[(Py)Hem(O_2)]$ and $[(Im)Hem(O_2)]$, which can be considered as mixed compounds of these hemochroms and bioxyhemoglobin.

The following model explains the increased oxygen capacity of magnetized blood. Iron atom in hem is a bivalent ion with 6 electrons in open 3d shell

TABLE 1. Changes in Blood Gases after Arrest of Artificial Ventilation ($M \pm m$, $n=18-20$)

Index		Time after ventilation arrest, min							
		0		3		6		10	
		C	E	C	E	C	E	C	E
SO_2 , %	A	99.84±0.06	99.94±0.03	99.98±0.34	99.66±0.08	80.5±6.6	93.7±3.6	48.0±6.4	64.8±5.2
	V	76.8±2.4	79.2±1.7	75.6±3.0	79.6±2.7	65.4±5.4	73.3±3.7	43.8±5.1	50.0±4.5
	mean	81.4±1.9	83.3±1.2	80.5±2.5	83.6±2.1	68.4±5.6	77.0±3.7	45±5	53.0±4.6
$[O_2]_{HbO_2}$, vol.%		16.3±0.4	16.6±0.2	16.1±0.5	16.7±0.4	13.7±0.1	15.4±0.7	9±1	10.6±0.9
P_{CO_2} , mm Hg	A	24.4±6.6	22.1±4.5	52.5±2.8	48.2±2.3	67.7±2.8	65.0±3.3	80.5±3.7	81.6±1.6
	V	32.1±6.5	28.3±4.8	48.1±2.6	46.4±3.4	63.2±2.7	63.9±2.5	77.1±2.9	79.9±2.8
	mean	30.6±6.5	27.1±3.2	449.0±2.5	47.0±3.2	64.1±2.7	64.0±2.7	77.8±3.1	80.0±2.8
$[CO_2]$, vol.%		44±8	42±4	53±3	52±3	58±3	58±3	62±3	63±2

Note. Here and in Table 2: control (C), experiment (E).

TABLE 2. Changes in Blood Gases in Various Time Intervals

Index	Intervals t_1-t_2 , min							
	0-3		3-6		6-10		0-10	
	C	E	C	E	C	E	C	E
$\Delta[\text{CO}_2]$, vol. %	9	10	5	6	4	5	18	21
$\Delta[\text{O}_2]$, vol. %	-0.18	0.06	-2.42	-1.32	-4.68	-4.8	-7.26	-6.06
β	50	-16.6	2.07	4.5	0.85	1.14	—	—
$\Delta[\text{O}_2]_{\text{calc}}$	—	—	—	—	—	—	-21.6	-25.2
$\Delta[\text{O}_2]_{\text{calc}} - [\text{O}_2]_{\text{HbO}_2}$	—	—	—	—	—	—	-14.34	-19.14

[1,2]. Of these electrons, two have opposite spins, while spins of other 4 electrons are oriented in parallel. When hem is placed into magnetic field, the magnetic moments of all electrons tend to turn in parallel to magnetic induction, so in some iron atoms the spins of all 6 outer electrons become parallel. This weakens the bond between iron and histidine nitrogen and enables binding of extra O₂. The relaxation time of spin transitions is long (such transitions are forbidden by the selection rule for magnetic spin quantum numbers), and the magnetized blood retains its properties for several minutes.

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