OXYGEN CONSUMPTION IN THE LUNGS AFTER CLOSED CHEST INJURIES IN DOGS

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The view that oxygen consumption in the lungs may be increased under pathological conditions [2, 4, 6, 10] is based on experimental data indicating intensification of the metabolic functions of the lungs in experimental models of various extremal situations [3, 6, 11-13]. However, no special study of oxygen utilization in the course of metabolic processes in the lungs was made in the investigations cited above.

The aim of this investigation was to study the dynamics of changes in oxygen consumption in the lungs in experimental thoracic trauma in dogs.

EXPERIMENTAL METHOD

Experiments were carried out on 25 dogs of both sexes weighing 8-20 kg. Experiments of series I were conducted on 15 animals with contusions of the lungs [9], in which the course of the early post-traumatic period (up to 7 days) was smooth. The experiments of series II were conducted on 10 dogs with injury to the ribs [9], of which five died during the first weeks after trauma. In preliminary operations on all dogs 2 days before trauma, cannulas were implanted into the right jugular vein and carotid artery, so that the right ventricle could be repeatedly catheterized with a "floating microcatheter" and a thermistor catheter introduced into the aorta [9]. During the same preliminary operation, holes were drilled parasternally in eight ribs on both sides to mechanically weaken the thoracic cage. Before trauma, during every hour thereafter (the first 4 h), and once during each successive day of observation (up to 7 days) the following parameters were determined in the animals: the total oxygen consumption (VO_2) through the mask on an SG-2M spirograph, the cardiac output (CO) by the thermodilution method, pO_2 of arterial and mixed venous blood on an AME-1 gas analyzer (from Radiometer, Denmark), and the hemoglobin (Hb) concentration on a Sahli hemometer. The oxygen concentration in arterial and mixed venous blood was determined by the equation [8]:

$$C_{O_2} = 1{,}34 \times Hb \times HbO_2\,\% + 0{,}003 \times pO_2.$$

The oxyhemoglobin (HbO_2) saturation in arterial and venous blood was determined with the aid of a Siggaard–Andersen nomogram, using data for the acid-base balance (ABB). The quantity of oxygen used in the system of the pulmonary circulation ($\dot{V}O_2PC$) was determined by subtracting from the total oxygen consumption of the body ($\dot{V}O_2$) the oxygen consumption in the system of the systemic circulation ($\dot{V}O_2SC$), i.e.,

$$\dot{V}O_2PC = \dot{V}O_2 = \dot{V}O_2SC.$$

$\dot{V}O_2SC$ was determined by multiplying CO by the arteriovenous oxygen difference [8]. The results were subjected to statistical analysis by Student's test.

EXPERIMENTAL RESULTS

Before chest trauma was inflicted, $\dot{V}O_2$ was the same for animals of both series. However, its components ($\dot{V}O_2PC$ and $\dot{V}O_2SC$) differed. For instance, in the animals of series II, which underwent a more traumatic preliminary operation, $\dot{V}O_2PC$ was 3-4 times higher than in the animals of series I, whereas $\dot{V}O_2SC$ was reduced almost by half compared with series (Table 1).

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TABLE 1. Oxygen Consumption of Dogs in Early Post-Traumatic Period after Closed Chest Injury (M ± m)

Series of experiments	Time after operation	$\dot{V}O_2$	$\dot{V}O_2$ PC	$\dot{V}O_2$SC	CO	Arteriovenous oxygen difference, vols. %
		ml/min·kg				
I ($n = 15$)	Before trauma	10,72±1,32	2,63±0,74	8,09±0,67	141±11,82	5,75±0,77
	1—2 h	13,54±2,72	3,09±1,25	9,65±1,21	120±11,21	8,02±1,08
	2—4 h	16,52±2,23	9,67±1,81	6,85±0,43	108±9,88	6,33±0,44
	1—3 days	12,12±1,85	4,38±1,30	7,74±1,62	163±22,33	5,01±0,77
	4—7 days	13,01±1,51	3,18±1,51	9,83±3,68	168±34,32	5,86±1,11
II:						
Convalescent ($n = 5$)	Before trauma	12,23±1,59	6,31±1,88	5,92±1,14	149±26,52	4,00±0,44
	1—2 h	13,13±2,13	5,6±0,66	7,52±2,41	120±25,81	6,20±0,93
	2—4 h	16,83±2,89	9,32±0,85	7,51±2,35	138±27,34	5,40±1,01
	1—3 days	15,79±1,61	9,90±2,41	5,89±0,69	126±11,34	4,67±0,61
	4—7 days	15,67±2,03	9,88±1,02	5,79±0,81	124±13,12	4,63±0,61
Lethal outcome ($n = 5$)	Before trauma	14,72±1,41	9,42±1,76	5,30±0,65	131±7,74	4,03±0,84
	1—2 h	15,36±2,05	8,83±2,27	6,55±0,87	107±9,37	6,08±0,93
	2—4 h	17,21±2,53	10,58±2,34	6,63±1,21	114±12,96	5,82±0,93
	1—3 days	16,49±2,18	9,13±2,32	7,36±2,52	131±7,18	5,59±0,35
	4—7 days	21,65±1,59	13,65±1,39	8,00±1,59	191±8,77	4,17±0,68
	2—4 h					
	Before death	19,16±1,62	14,93±2,85	4,23±0,83	143±18,46	2,96±0,141

During the first 4 h after chest trauma the mean values of $\dot{V}O_2$ for animals in both series of experiments showed a tendency to rise: in the animals of series I there was a rise in $\dot{V}O_2$PC and a fall in $\dot{V}O_2$SC, whereas in the animals in series II there was some tendency for both $\dot{V}O_2$PC and $\dot{V}O_2$SC to rise. Later, during the first 3 days after chest trauma the value of $\dot{V}O_2$ for animals of series I returned to the initial level, i.e., $\dot{V}O_2$PC fell and $\dot{V}O_2$SC rose. Toward the end of the week these parameters did not differ statistically significantly from their initial values. In the animals of series II 24 h after trauma and until the end of the observations a tendency for $\dot{V}O_2$ to rise continued: in the convalescent group due to an increase in $\dot{V}O_2$PC accompanied by normalization of $\dot{V}O_2$SC, but in animals with a lethal outcome due to an increase in both $\dot{V}O_2$PC and $\dot{V}O_2$SC; this last parameter, moreover, showed a tendency to fall but not until a few hours before death of the animals.

Thus in the early post-traumatic period after closed chest injury, just as in other acute pathological states [5, 7], $\dot{V}O_2$ maintained or even increased its value. However, the large dose of oxygen taken up by the body in the early post-traumatic period did not find its way into the systemic circulation, but was utilized in the system of the pulmonary circulation. Analysis of the data showed that in cases when $\dot{V}O_2$ was increased, and in those when it was unchanged (or reduced), there was a redistribution of oxygen in favor of the pulmonary circulation. This was a general rule for all the animals irrespective of the character of trauma. This redistribution evidently occurred actually during the preliminary operation and it depended on the extent of the operation and the individual response of the animal to trauma. Evidence of this was given, first, by the greater increase in $\dot{V}O_2$PC in the animals of series II, on which the extent of the surgical manipulations was greater, and second, the wide range of variation of $\dot{V}O_2$PC after infliction of injuries of equal extent.

The redistribution of oxygen in favor of the pulmonary circulation reached its maximal degree during the main experiments — during the first few hours after trauma. High values of $\dot{V}O_2$PC were maintained for a longer time in animals with injury to the thoracic cage, especially if the course of the post-traumatic period was unfavorable.

What mechanisms may lie at the basis of the rise of $\dot{V}O_2$PC in the early post-traumatic period? It can be tentatively suggested that the increase in $\dot{V}O_2$PC is due to serveral different causes. We do not, of course, regard the concept of "oxygen consumption in the lungs" as synonymous with the concept of "oxygen consumption by lung tissues." It is difficult to image that the whole volume of oxygen which, according to our data, is retained in the system of the pulmonary circulation is utilized entirely for oxidation-reduction processes in lung tissue. Evidently some part of it is used by the cells of the lung tissue itself for realization of the metabolic functions of the lungs when enhanced after trauma. It can also be postulated that some of the oxygen is dissolved in the pulmonary lymph, the volume velocity of flow of which may be increased many times over [1], in pulmonary embolism, for example, which often complicates the early post-traumatic period [5, 7, 9].

Some of the oxygen is evidently utilized for direct oxidation of metabolites contained in blood in the pulmonary capillaries. Possibly other mechanisms of the increase in $\dot{V}O_2PC$ also exist.

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EFFECT OF PSYCHOTROPIC DRUGS ON ALCOHOL MOTIVATION IN NONINBRED ALBINO RATS

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Investigations into the behavior of rats with an inclination toward spontaneous consumption of ethanol have shown that one of the most characteristic features distinguishing them in the population is the weakness of their adaptive forms of behavior, as is shown, in particular, by their weak competitiveness in the struggle for biologically meaningful goals [5]. Ethanol normalizes the adaptive behavior of these animals [5], and this evidently is responsible for their use of ethanol. At the same time, it has been shown [4] that many psychopharmacological drugs, like ethanol, can normalize the adaptive behavior of these animals.

Accordingly, in the investigation described below, interaction between the effect of several drugs of different classes on the degree of inclination of animals to develop a depression-like state (DLS) in a conflict situation, which is one form of disadaptation, and their action on the formation of alcohol motivation was studied in noninbred male rats.

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