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CHANGES IN SOME GAS EXCHANGE INDICES IN DOGS IN THE INITIAL STAGE OF TRAUMATIC SHOCK BY CANNON'S METHOD

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Experiments in which traumatic shock was produced by Cannon's method in 35 dogs showed that metabolic acidosis during shock develops against the background of a sharply increased oxygen consumption in the initial stage of trauma when the arterial pressure is much higher than originally. The brain is under the most favorable conditions of gas exchange in traumatic shock, whereas the skeletal muscle of the limbs is less favorably situated. It is concluded that in severe trauma, despite sustained function of the respiratory and cardiovascular systems, the tissues of the brain and skeletal muscle do not receive sufficient oxygen as a result of circulatory disturbances and a sharp rise in their oxygen demand.

KEY WORDS: *gas exchange; metabolic acidosis; acid–base balance.*

Unjustifiably little attention has so far been paid to study of the functions of the main systems responsible for homeostasis in the initial period of traumatic shock. Moreover, there is no general agreement among specialists even on the meaning of the concepts of the "initial" and "late" stages of shock [1, 4, 9]. Some workers regard shock as purely a state of sharply depressed functions of the body after severe trauma or blood loss [8]. The gas exchange and, in particular, the acid–base balance have been investigated on several occasions in traumatic shock [2, 4]. However, the lack of any unified model of traumatic shock and of any combined approach to the study of its pathogenesis, coupled with neglect of the importance of the time factor and the fragmentary nature of information about the oxygen budget of the body in this severe complaint, does not permit the disturbance of the gas exchange to be analyzed with sufficient depth in the course of its development. From the writers' point of view the most important stage in the development of traumatic shock, and the one which has received least study, is the erectile stage (the stage of excitation). It is at the moment of trauma or immediately after that the vital homeostatic mechanisms are overwhelmed, with the subsequent development of marked metabolic disturbances and a serious

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TABLE 1. Changes in BP, vO_2, pH, BE, pO_2, and pCO_2 in Arterial and Venous Blood and in Blood from Sagittal Sinus of Dogs in Initial Stage of Development of Traumatic Shock by Cannon's Method (M ± m)

Index studied	Initial state	During trauma		After trauma and fall of BP to 60 mm Hg	
		1st measurement	2nd measurement	5 min	30 min
BP, mm Hg	127±3	207±23*	150±12*	59±1**	44±3
vO_2, ml/kg/min	10,6±0,8	39,4±4,4*	21,4±3,7**	7,8±0,6**	8,3±0,6
Blood from femoral artery					
pH	7,374±0,016	7,274±0,009*	7,263±0,032*	7,264±0,012	7,280±0,016
BE, meq/liter	—7,9±0,51	—12,1±0,51*	—14,5±0,73*	—14,2±0,62*	—14,3±0,86*
pO_2, mm Hg	90±2,1	97±1,7	104±2,7	101±2,0	98±2,0
pCO_2, mm Hg	28±1,58	29,2±1,22	24,7±2,10	25,5±1,45	23,8±1,73
n	35	35	35	31	22
Blood from femoral vein					
pH	7,277±0,016	7,138±0,015*	7,126±0,029*	7,074±0,021**	7,071±0,020*
BE, meq/liter	—11,8±0,92	—14,6±0,68*	—17,2±0,88*	—17,2±0,72*	—17,8±0,8*
pO_2, mm Hg	49±2	40±2*	36±4,9*	35±2*	30±2*
pCO_2, mm Hg	31,3±2,30	40,7±2,60	40,1±4,46	42,0±2,39	41,9±3,07
n	33	33	33	30	25
Blood from sagittal sinus					
pH	7,295±0,020	7,223+0,015*	—	7,157+0,029**	7,162±0,035*
BE, meq/liter	—10,9±1,62	—12,0±1,15*	—	—15,9±1,12*	—15,9±2,32*
pO_2, mm Hg	52±5	54±4	—	50±5	48±8
pCO_2 mm Hg	29,9±2,78	36,6±2,24*	—	35,7±4,53*	31,7±3,59
n	11	10	—	10	6

Note. 1. One asterisk denotes results for which difference from initial values differs statistically significantly ($P < 0.01$); two asterisks, the same compared with results of the previous stage of the investigation.
2. n) Number of observations.

general condition.

In the investigations described below some of the principal indices of the gas exchange were studied in the initial period of development of traumatic shock in dogs.

EXPERIMENTAL METHOD

Shock was induced in dogs by injury to the right thigh by Cannon's method. Injury to the animals continued until a serious general condition developed and the arterial blood pressure (BP) had fallen to a consistent level of 60-70 mm Hg. BP was determined in the left femoral artery by means of a mercury manometer, and the pH, buffer base deficit (BE), and partial pressure of carbon dioxide (pCO_2) and oxygen (pO_2) were measured in arterial and venous blood and in blood taken from a brain sinus. The oxygen demand (vO_2) also was determined on tracheotomized animals, using the Eugraph (Hungary) apparatus. The value of vO_2 was calculated from a nomogram in ml/kg body weight/min. Indices of the acid–base balance were determined by the micro-Astrup method [7] and pO_2 was measured polarographically. For obtaining venous blood from the dogs the left femoral vein was dissected beforehand and a T-shaped cannula introduced into it. Blood flowing from the brain was taken through a burr-hole in the skull (3 × 3 mm) from the sagittal sinus. It was established previously that drilling such a burr-hole and dissecting the trachea caused no significant disturbances of the general condition of the animals or of the acid–base balance of the arterial and venous blood. General anesthesia was not used. All the indices were recorded in the initial state (after fixation and preparation of the animals for the experiment), at the initial moment of trauma when BP was raised to its highest values which were steadily maintained, during the period when BP showed a tendency to fall, and also 5 and 30 min after BP had fallen to 60 mm Hg.

EXPERIMENTAL RESULTS AND DISCUSSION

The results are given in Table 1.

In the initial period of trauma the animals exhibited generalized motor excitation, accompanied by a marked rise of BP, an increase in vO_2, a decrease in pH, and an increase in the negative BE in all blood samples tested. The changes in pH and BE were most marked in blood taken from the artery and least marked in blood from the sagittal sinus. Meanwhile pCO_2 was increased in blood taken from the femoral vein and sagittal sinus.

A short time later, as trauma continued, a tendency was observed for BP and vO_2 to fall, although their levels still remained considerably higher than initially. Acidotic changes increased in the arterial and venous blood, as shown by the even greater decrease in pH and numerical increase in BE.

A progressive decrease in pH and increase in BE were observed 5 min after BP had fallen to 60 mm Hg in all the blood samples tested, and pCO_2 increased in blood taken from the sagittal sinus and femoral vein. No significant changes in pO_2 in blood from the sagittal sinus were observed at any of these times of testing.

Despite the sharp fall in BP 30 min after trauma, the indices studied remained at the same level as at the previous stage of the investigation and vO_2 was practically back to normal.

In the initial stage of trauma, while BP remained at a high level, profound changes thus occurred in the gas exchange. Despite marked activation of the external respiratory apparatus and the increase in vO_2, the pH of the blood was reduced and its base deficit increased. These changes point to the development of acidosis and to inability of the oxygen transport function of the blood to meet the sharply increased demands of the body. As was shown previously [3], it is at this time that the loss of blood and plasma into the injured limb reaches its maximum.

The metabolic reactions were most marked in blood flowing from the limb muscles and much less so in blood flowing from the brain. The increase in pCO_2 and decrease in pH in blood flowing from these organs are evidence of activation of aerobic and anaerobic oxidative processes in them.

Throughout the experiments pO_2 in blood from the sagittal sinus remained close to its initial level, in the venous blood it fell, but in the arterial blood it rose considerably. Presumably after severe mechanical trauma, accompanied by the development of shock, adaptive mechanisms protecting the functions of the brain ensure much better conditions for gas exchange in that organ than in the limb muscles.

Contrary to the widely held notion [6] that the phase of excitation in shock can be regarded as a stage of activation and of "improvement" of the function of the basic systems of the body, in that period definite signs of decompensation of some vitally important functions have already developed. Marked changes in gas exchange arising at the beginning of the erectile stage of traumatic shock lead to the development of subcompensated metabolic acidosis, which increases progressively and changes to the decompensated form long before BP falls.

The prolonged spasm of the arterioles, shunting of the blood flow, and centralization of the circulation developing in shock [5] reflects a hyperreaction of the adaptive systems of the body, brought into play in a stereotyped fashion in response to any extremal factor. Having lost their relevance to the conditions created in the erectile stage of shock, they themselves become the cause of tissue hypoxia, metabolic acidosis, hypocapnia, and other severe disorders responsible for the change in the "rigid" constants of biochemical homeostasis.

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