ACID-BASE BALANCE IN VENOUS AND ARTERIAL BLOOD IN BLOOD TRANSFUSION SHOCK

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In shock caused by transplantation of rabbits' blood into dogs, uncompensated acidosis is observed in the venous blood, disappearing after 1 h, but reappearing after 24 h. The changes are less marked in arterial blood, and 24 h after transfusion of heterogeneous blood respiratory alkalosis is observed.

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In the comparatively numerous investigations which have been undertaken to study the pathogenesis of blood transfusion shock, insufficient attention has been paid to disturbances of acid-base balance, maintenance of which at the normal level is highly important for preservation of normal bodily activity.

In reports of a few investigations very limited information is provided only about changes in pH and blood alkaline reserve [1, 3].

Our object was to study the state of the acid-base balance in venous and arterial blood in experimental blood transfusion shock.

EXPERIMENTAL METHOD

Experiments were carried out on 23 dogs weighing from 10-20 kg. The experimental animals were transfused by the intravenous drip method (slowly) with rabbits' blood in a dose of 20-30 ml/kg body weight. The following investigations were carried out: arterial pressure, ECG, oxyhemoglobin concentration (using Brinkmann's hemoreflector), acid-base balance (using Astrup's apparatus [4]) – momentary pH, pCO_2, total CO_2 concentration, standard and actual bicarbonate, buffer bases, and base excess.

Samples were taken in syringes containing mineral oil. Venous blood was taken from a catheter inserted into the right ventricle. The investigations were carried out before transfusion of heterogeneous blood, at the height of shock, and 1 h and 24 h after transfusion.

EXPERIMENTAL RESULTS

After transfusion of heterogeneous blood a sharp decrease in arterial pressure (below 60 mm Hg) with the development of severe shock was observed in 14 animals. The fall in arterial pressure was accompanied by general excitation and dyspnea of varied degree, and subsequently the animal fell into a state of prostration. Most dogs showed significant changes in the ECG characteristic of myocardial hypoxia, disturbances of myocardial conduction, and hyperkaliemia. The duration of the fall in pressure varied from animal to animal. In some dogs arterial hypotension lasted 20-30 min, in others 2 h or more. In most dogs the pressure was restored after 24 h.

Investigation of the venous blood at the height of shock revealed uncompensated metabolic acidosis in most animals (Table 1). Only the value of pCO_2 in the blood remained unchanged, indicating the uncompensated character of metabolic acidosis at the height of shock. The concentration of oxyhemoglobin in the venous blood at the height of the shock fell sharply (from 60-80 to 35-10%). A partially compensated metabolic acidosis developed in the arterial blood at the height of shock (Table 1).

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TABLE 1. Indices of Acid-Base Balance in Venous and Arterial Blood during Transfusion Shock (M ± m)

Index of acid base balance	Initial data		Height of shock		1 h after transfusion		24 h after transfusion	
	venous blood	arterial blood	venous blood	arterial blood	venous blood	arterial blood	venous blood	arterial blood
Actual pH	7,30± 0,01	7,347± 0,01	7,22± 0,01 P<0,01	7,286± 0,01 P<0,05	7,28± 0,013 P>0,05	7,33± 0,03 P>0,05	7,23± 0,02 P<0,05	7,34± 0,01 P>0,05
pCO_2 (in mm Hg)	49,9± 1,6	38,4± 1,7	49,7± 2,29 P>0,1	30,4± 2,1 P<0,05	43,5± 3,1 P<0,05	27,2± 2,9 P<0,05	46,5± 4,03 P>0,05	32,2± 1,5 P<0,05
Total CO_2 (in meq/liter)	23,8± 1,01	19,5± 0,73	18,4± 0,95 P<0,05	12,09± 0,9 P<0,05	19,36± 1,45 P<0,05	18,3± 1,5 P<0,05	18,4± 1,55 P<0,05	18,12± 1,06 P<0,05
Standard bicarbonate (in meq/liter)	21,46± 0,86	19,15± 0,5	16,2± 0,64 P<0,05	15,3± 0,62 P<0,05	14,6± 1,55 P<0,05	16,6± 1,1 P<0,05	17,2± 1,28 P<0,05	19,16± 0,91 P<0,05
Actual bicarbonate (in meq/liter)	22,43± 0,91	18,4± 0,76	17,3± 0,9 P<0,05	13,76± 0,15 P<0,05	14,73± 1,91 P<0,05	14,64± 1,86 P<0,05	20,16± 1,6 P<0,05	17,24± 0,89 P<0,05
Base excess (in meq/liter)	—6,19± 0,71	—4,72± 0,91	—10,9± 1,1 P<0,05	—11,6± 0,63 P<0,05	—13,3± 2,56 P<0,05	—11,5± 1,86 P<0,05	—12,3± 1,4 P<0,05	—5,6± 1,12 P<0,05
Buffer bases (in meq/liter)	44,4± 2,6	45,8± 1,8	39,9± 1,7 P<0,05	38,9± 1,3 P<0,05	36,9± 2,2 P<0,05	38,38± 1,9 P<0,05	41,05± 2,22 P<0,05	44,87± 3,17 P>0,05

The following determinations were carried out 1 h after the beginning of transfusion of heterogenous blood, during elevation of the arterial pressure (in some animals at this time the pressure had almost reached its initial values). At this period a tendency toward normalization of the pH and a decrease in the standard bicarbonate level were observed in the venous blood of most animals. Consequently, respiratory compensatory mechanisms had been brought into operation, and 1 h after transfusion the acidosis was completely compensated. However, the base deficiency increased, the concentration of buffer bases was reduced, and the total CO_2 concentration was slightly increased. In other words, improvement took place on account of respiratory compensatory mechanisms and not of metabolic factors. The oxyhemoglobin concentration in the blood by this time had reached 55-30%. Completely compensated metabolic acidosis was found in the arterial blood at this period.

It is an interesting fact that 24 h after transfusion of heterogenous blood the state of the acid-base balance in the venous and arterial blood differed completely: in the venous blood at this period decompensated metabolic acidosis developed, whereas in the arterial blood a completely compensated respiratory alkalosis was found (Table 1). This shows that partial compensation (on account of respiratory components) immediately after transfusion of heterogenous blood could not completely overcome the severe disturbances arising in the tissues during blood transfusion shock.

During shock of this type profound disturbances of acid-base balance thus develop, and these are considerably more severe in venous than in arterial blood. It may be postulated that this is due to the entry of large quantities of acid metabolites into the venous blood in connection with the developing hypoxia [2, 5] resulting from the sharp decrease in arterial pressure.

The duration of the disturbances of acid-base balance in the venous blood suggests that acidosis develops not only against the background of acute circulatory disorders, because it is observed even after recovery of a stable arterial pressure. It may be that after transfusion of large doses of foreign blood significant disturbances in activity of the tissue oxidation-reduction systems take place.

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