OXYGEN REGIMEN OF THE LIVER IN SEVERE TRAUMATIC SHOCK

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Hypoxia, which plays an important role in the pathogenesis of traumatic shock, has been insufficiently studied until now [5]. There is a special need for investigations of the oxygen regimen in various organs during shock, and elucidation of its interrelationships with the total oxygen balance of the organism. In particular, it is important to have some concept of the oxygen regimen in the liver, since the metabolism of this organ depends on it, and thus, to a large degree, the energetics of the entire organism. In addition, the level of oxidative processes in the liver determines the production in that organ of vasoactive substances, which also is essential for the development of shock [18, 19, 22]. And finally, a study of the oxygen balance of the liver would provide more accurate information on its blood circulation, which is significant for an understanding of the general principles behind the hemodynamic disturbances during shock [4, 6, 8].

EXPERIMENTAL METHOD

The experiments were set up on 48 male and female cats, weighing from 3 to 5.5 kg, in which shock was caused by traumatization of the right femur, using a hammer with a rubber head, weighing 700 grams. During the experiments, we observed the condition of the animals, investigated the defense, pupillary, and corneal reflexes, and made kymographic recordings of the arterial pressure, pulse and respiration. On the basis of these indices, we determined the severity of shock.

Oxygen exchange in the liver was evaluated from the oxygenation of the inflowing and outflowing blood, and from the oxygen tension in the liver. For this purpose, repeated samples of blood were drawn from the aorta, the posterior vena cava (at the level of entrance of the hepatic veins and caudal to this site), and the portal vein. Polyethylene catheters were introduced into these vessels, according to our method [7]. The degree of blood oxygenation was determined by the use of the OKO-01 "Biofizpribor" Cuvette oxymeter. On the basis of the data obtained, we calculated the arteriovenous and cava-portal differences in blood oxygen saturation. The oxygen tension in the liver was determined by the electrochemical method [9], using a pair of electrodes (amalgamated copper-iron) and a set-up consisting of the M-21 specular galvanometer and a universal shunt.

EXPERIMENTAL RESULTS

In 10 control experiments, with fixation of the animals to a stand for 3-4 h (the same amount of time as in the experiments with shock), it was established that the hemodynamic and respiratory indices do not undergo essential changes during that time: the arterial pressure initially decreased, and then returned to its original level; the pulse quickened very slightly; respiration slowed somewhat.

The oxygen saturation of the arterial blood was almost unchanged: at the beginning of the experiment it was equal to 96 ± 1.0%, and at the end – 96 ± 0.9%, which completely corresponds to the normal indices [2]. Oxygenation of the venous blood decreased somewhat: while immediately after fixation the blood oxygen saturation in the vena cava, at the level of the hepatic veins, was equal to 66 ± 1.6%, at the end of the experiment it was 61 ± 2.2%. The oxygen tension in the liver tissue ranged within rather narrow limits -- from $1.12 \pm 0.24 \cdot 10^{-5}$ mg/cm^2/sec at the beginning of the experiment to $1.05 \pm 0.16 \cdot 10^{-5}$ mg/cm^2/sec at the end.

In order to decrease the aggravating action of repeated blood drawing on the course of shock, the animals in

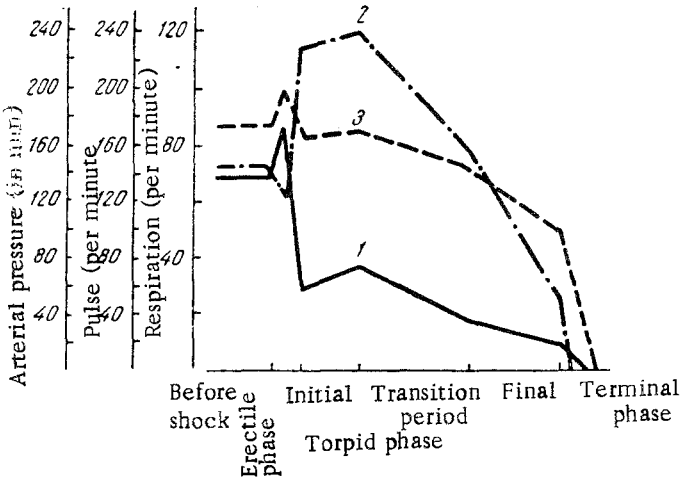

Fig. 1. Changes in the arterial pressure (1), pulse (2), and respiration (3) in different phases of traumatic shock. (The proportions in the duration of the phases are presented on the basis of the mean data of the experiments).

the experimental group were divided into three sub-groups, and blood samples from each animal were drawn from as few areas of the vascular system as possible: in the first sub-group – from the aorta and the posterior vena cava at two levels; in the second – from the aorta, the posterior vena cava at the level of the hepatic veins, and the portal vein; in the third – from the aorta and the vena cava at the level of the hepatic veins; in the aorta and the vena cava at the level of the hepatic veins; in the animals of the last sub-group, we determined the oxygen tension in the liver.

The experiments showed that with application of 180-200 blows to the femur, typical traumatic shock developed in all the animals, the course of which could be divided into three phases: erectile (usually coinciding with the period of traumatization), torpid, and terminal. In the torpid phase, most characteristic of shock, very regular dynamics were noted, and thus three periods were arbitrarily distinguished in it: initial, transition, and final.

As we know, the most striking manifestations of traumatic shock are the changes in arterial pressure, pulse, and respiration. Its phases are also normally distinguished on the basis of these indices. The data characterizing the dynamics of shock in our experiments showed that the torpid phase of shock, and its longest period – the transition period, are characterized by persistant hypotension, a stable pulse, and tachypnea (Fig. 1).

The oxygen saturation of the arterial blood changed very little throughout the course of the entire period of shock, and decreased markedly only at its terminal phase (see table). Oxygenation of the venous blood, on the other hand, decreased at the very beginning of the development of shock, and this decrease was greater in the portal vein system than in the system of the posterior vena cava almost throughout the entire course of shock. Only at the very end of the torpid phase of shock, at the moment it changed to terminal, did the opposite relationships arise (see table and Fig. 2). As pertains to the oxygen tension in the liver tissue, it already fell sharply in the erectile phase of shock and then progressively decreased throughout the entire shock course, only somewhat stabilizing in the transition period (see Fig. 2).

In a number of the animals in the original state, the oxygen saturation in the blood from the portal vein exceeded the saturation of the blood obtained from the vena cava by an average of $6 \pm 2.3\%$, while in the other animals, the blood of the portal vein was oxygenated by $12 \pm 2.1\%$ less than the blood from the vena cava. The significance of this difference was confirmed by mathematical analysis ($P < 0.01$). It is interesting that the course of shock in the animals of these two sub-groups also proved to be different: in the first sub-group, shock reached its terminal phase in an average of 142 ± 19 min, while in the second – after only 45 ± 10 min. This difference also proved to be completely significant ($P < 0.01$).

The data obtained shows that manifest hypoxia of the liver rapidly develops during severe traumatic shock, and this doubtlessly influences the course of the metabolic processes in that important organ. The hypoxia is circulatory, and thus its characteristics are determined, to a large degree, by the duality of the liver's blood supply. In the des-

Changes in the Indices for the Oxygen Regimen of the Liver Secondary to The Development of Traumatic Shock (Mean Data M ± m)

Index	Area from which blood was drawn	Before shock	Phase: erectile	torpid: initial	torpid: transition period	torpid: final	terminal
Blood oxygen saturation (in % of oxyhemoglobin)	Aorta	95± 0,52	—	96± 0,56	96± 0,45	94± 0,48	90± 0,95
	Posterior vena cava – caudal division	56± 2,31	—	44± 1,40	—	40± 1,59	35± 6,18
	Posterior vena cava – at the hepatic veins	62± 1,52	—	51± 1,54	—	43± 1,60	36± 1,23
	Portal vein	59± 3,14	—	44± 1,89	—	38± 1,24	38± 1,21
Difference in blood oxygen saturation (in % of oxyhemoglobin	Between the aorta and and hepatic vein	+33± 1,02	—	+45± 2,02	—	+51± 1,29	+54± 1,35
	Between the aorta and portal vein	+36± 2,68	—	+52± 1,77	—	+56± 1,08	+52± 1,43
	Between the hepatic and portal vein	+3± 2,88	—	+7± 1,95	—	+5± 1,82	−2± 0,87
Oxygen tension in the liver (in units of $1 \cdot 10^{-5}$ mg/cm²/sec)		1,55± 0,118	1,14± 0,072	0,71± 0,086	0,53± 0,010	0,23± 0,009	0,11± 0,012

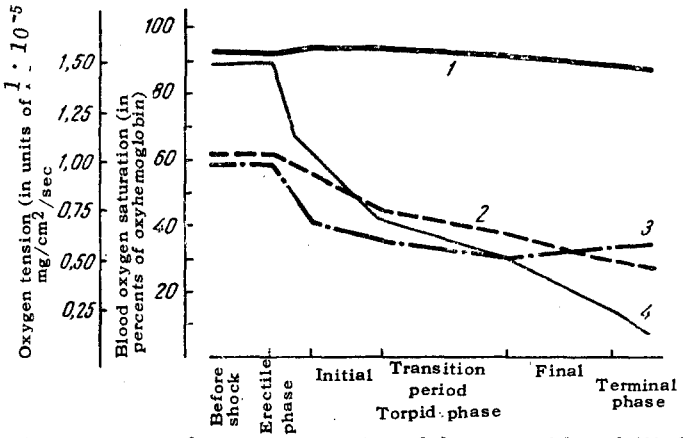

Fig. 2. Dynamics of oxygen saturation of the arterial blood (1), blood of the vena cava at the level of the hepatic veins (2), and blood of the portal vein (3), and dynamics of the oxygen tension in the liver (4) secondary to the development of traumatic shock.

cribed experiments, oxygenation of the arterial blood during shock remained essentially unchaged; other investigators have observed the same picture with the most diverse forms of shock [1, 3, 10]. The significant decrease in oxygen tension in the liver tissue, and the progressive reduction in the degree of oxygenation of the blood flowing out of the liver, testify to a slowing of the blood flow in that organ. This once again confirms the concept that hypoxia in one or another organ during shock is determined, above all, by disruptions in the circulation within the given organ [4, 5, 10, 12].

The lower blood oxygen saturation in the portal vein than in the blood flowing out of the liver, observed during the torpid phase of shock, affords a basis for postulating that during this phase, especially in its initial period, the blood supply of the liver is accomplished primarily via the hepatic artery, while under normal conditions, up to 3/4 of the blood passing through the liver gains access to it via the portal vein [21]. Obviously, the reciprocity between the portal and arterial blood supplies of the liver, which is noted under normal conditions [17], acquires even greater significance in shock.

There is an entire basis for concluding that specifically the arterial blood supply of the liver regulates its oxygen regimen, and thus, in shock, there is a transition of the liver to a predominant supply of arterial blood. This is also indirectly confirmed by the fact that the portal blood flow in the liver during shock slows markedly [6, 8, 11, 16, 20]. The mechanism for the transition of the liver to a supply of the more oxygenated arterial blood is still unclear. It is possible that, in this case, in addition to reflex acts, local humoral factors also play a role, including the production by the hypoxic liver of the vasoactive substance – ferritin [18, 19, 22]. The fact that with greater oxygenation of the blood in the portal vein shock develops markedly more slowly confirms the concept of the important role of liver hypoxia in the pathogenesis of shock [13, 14]. This fact also is in accord with the interesting experiments in which an increase was shown in the survival rate of animals in shock under conditions of perfusion of the liver with arterial blood [15].

SUMMARY

The work deals with oxygenation of the blood flowing to and from the liver and the oxygen tension in the hepatic tissue in traumatic shock. Experiments were staged on 48 cats in which shock was provoked by traumatization of the right femur. During the development of shock arterial blood oxygenation proved to change but little; as to the blood of posterior vena cava and of the portal vein – it reduces considerably. Beginning from the erectile shock phase the oxygen tension in the liver exhibits a constant decrease.

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