COMPARISON OF CHANGES IN CARBOHYDRATE METABOLISM AND OXYGEN SUPPLY OF THE LIVER IN TRAUMATIC SHOCK AND STABLE HYPOTENSION

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Disturbances of carbohydrate metabolism play an important role in the pathogenesis of traumatic shock. Authors, in describing the shock syndrome, invariably have indicated hyperglycemia as one of the characteristic symptoms of this condition [1, 2, 4, 5, 12, 13]. There is no doubt of the significance of changes in the liver function in the development of disturbances of the carbohydrate metabolism [2, 5, 6, 10, 17]; however, many aspects of these phenomena remain unclear. The disclosure of a connection between the disruptions of carbohydrate metabolism during shock and the development of hypoxia is of extreme interest.

Since hypoxia during traumatic shock is mainly a result of circulatory disorders and to a lesser degree of disruptions of the external respiration, it was essential to analyze the characteristics of carbohydrate metabolism in connection with the oxygen balance, not only during traumatic shock but in hypotension, the degree and duration of which corresponded to that observed during shock.

PROCEDURE

The experiments were carried out on 25 cats of both sexes, from 3 to 4.5 kg in weight. The animals were operated upon 5-10 days before the experiment by implanting a catheter into the portal vein and in a number of cases into the vena cava [9], after which clinical and hematological tests were carried out.

Shock was induced by traumatizing the soft tissues of the femur by the application of 180-200 blows of a hammer 700 g in weight, with a rubber tip. Stable hypotension was produced according to the principle of C. Witgers [17], for which the lumen of the main vessel (caudal division of the aorta) was connected through a polyethylene catheter to a hemostat in which constant pressure of 60 mm Hg was maintained. When normovolemia was achieved (when all the blood that had passed out into the hemostat had returned to the blood vessel), the hemostat was disconnected. The instrument of [3], which we modified, was used as the hemostat.

During the experiment we observed the condition of the animals, kymographically registered the arterial pressure, pulse, and respiration. The pressure in the portal vein and posterior vena cava was measured constantly with a two-capillary salt manometer.

The acid system of the liver was judged according to the oxygenation of the blood flowing into it and out of it. The degree of saturation by oxygen was determined with the OKO-01 cuvette oxymeter produced by Biofizpribor. The sugar and lactic acid content in blood samples obtained through the catheter, aorta, portal vein, and vena cava, close to the ostia of the hepatic veins, was determined to evaluate the carbohydrate metabolism. Sugar was determined by the ferricyanide method, lactic acid according to the reaction with para-hydroxydiphenyl [11]. The hematocrit index was determined in the same blood samples.

The reliability of the difference (P) was determined according to the method of the sign criterion or the Van der Varden method [16].

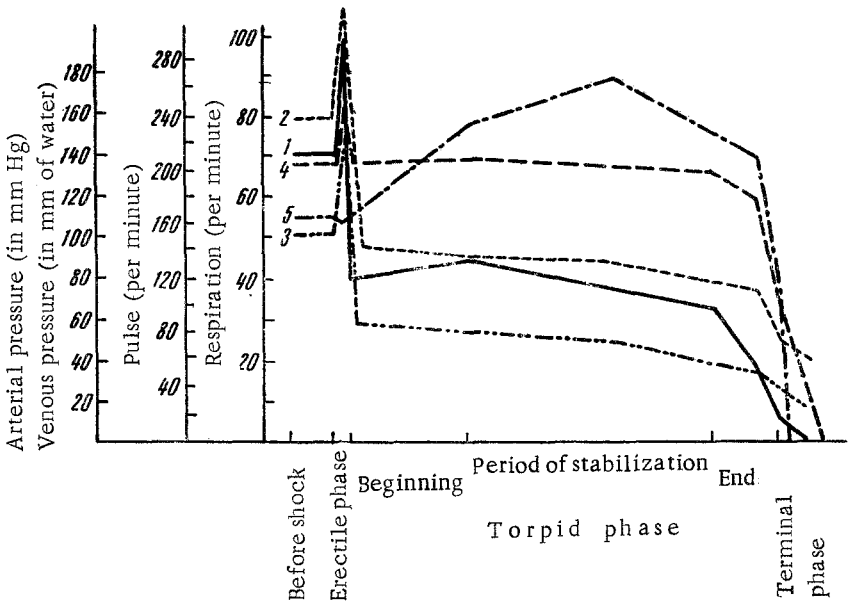

Fig. 1. Changes in the arterial and venous pressure, pulse, and respiration at various phases of traumatic shock (ratios in duration of phases are cited on the basis of average data). 1) Arterial pressure; 2) pressure in the portal vein; 3) pressure in the vena cava; 4) pulse; 5) respiration.

EXPERIMENTAL RESULTS

Traumatizing the soft tissues of the femur led to the development of severe shock, and then to death of the animals (only 3 out of 18 cats survived). Shock, which lasted for an average for 160 min, was characterized by the repeatedly described dynamics [7, 8]; in the most typical torpid phase, three periods were distinguished: initial, period of stabilization, and final (Fig. 1).

The oxygen system of the liver was characterized by the following principles in the shock experiments: low variability of the oxygenation of the arterial blood before the onset of the terminal phase of shock, a substantial and reliable decrease in the oxygenation of the blood of the portal vein ($P < 0.05$), and far less than the vena cava. These changes, appearing in the very beginning of the torpid phase, were maintained until the death of the animal (Table 1). All this indicated that with the development of hypoxia, the liver changes over to a predominant arterial blood supply.

The glucose content in the blood flowing into and out of the liver differed little from the initial content at the beginning of the torpid phase of shock, but increased sharply as the development of shock progressed (see Table 1). Beginning with the period of stabilization of the torpid phase, a substantial accumulation of lactic acid was observed in the blood, which is evidence of the predominance of the anaerobic pathway of carbohydrate metabolism. The somewhat greater lactic acid concentrarion in the blood from the portal vein in comparison with the blood from the vena cava ($P < 0.05$) may be evidence of conservation of the ability of the liver for carbohydrate resynthesis during the torpid phase of shock.

A distinct relationship was observed between the lactic acid content and oxygen saturation of the blood: the lower the degree of oxygen saturation, the more pronounced the lactacidemia. This relationship is especially patent in a comparison of the composition of blood samples obtained from the vena cava and portal vein.

In the terminal phase of shock, the lactic acid content varied extremely, reaching 600 mg % in individual cases. In the three animals that survived after suffering shock, a substantial reduction of the lactic acid content in the blood was noted. On the day following the experiment, they exhibited a further decrease in the lactic acid content in the arterial and venous blood.

Experiments on prolonged stable hypotension were undertaken to determine the significance of circulatory

TABLE 1. Sugar and Lactic Acid Contents and Oxygen Saturation of the Blood in Various Divisions of the Vascular Canal During Traumatic Shock (M +m)

Index determined	Initial state			Shock											
				torpid phase									Terminal period		
				initial period			period of stabilization			final period					
	A	VC	PV	A	VC	PV	A	VC	PV	A	VC	PV	A	VC	PV
Sugar (in mg%)	165,3 ±17,0	177,4 ±20,7	178,7 ±21,1	165,3 ±26,3	140,0 ±18,7	144,0 ±17,6	261,6 ±23,3	253,4 ±18,8	236,1 ±23,8	287,7 ±23,9	266,8 ±26,5	251,3 ±31,2	269,1 ±34,6	285,7 ±47,6	236,8 ±48,3
P				>0,05	>0,05	>0,05	<0,05	<0,05	<0,05	>0,05	>0,05	>0,05	>0,05	>0,05	>0,05
Lactic acid (in mg %)	28,2 ±3,1	30,2 ±3,8	30,1 ±5,1	37,0 ±9,1	50,5 ±13,0	52,7 ±7,0	84,6 ±9,8	93,1 ±9,5	117,7 ±18,4	129,6 ±14,7	138,6 ±19,6	145,8 ±18,7	—	—	—
P				<0,05	<0,05	<0,05	<0,05	<0,05	<0,05	<0,05	<0,05	<0,05			
Oxygen saturation (in % of oxyhemoglobin)	92 ±1,4	53 ±5,5	67 ±2,9	90 ±2,3	46 ±6,0	37 ±0,5	89 ±2,7	44 ±5,2	34 ±1,2	—	—	—	83 ±2,6	31 ±5,0	26 ±3,9
P				>0,05	>0,05	<0,05	>0,05	>0,05	>0,05				>0,05	>0,05	>0,05

Notations: A) Blood samples taken from the aorta; VC) blood samples taken from the vena cava; PV) blood samples taken from the portal vein; P) reliability of the difference in comparison with the preceding period for the same vessel.

*Data on the lactic acid content in the blood in the terminal phase of shock are not included in Table 1 as a result of the substantial individual fluctuations.

TABLE 2. Sugar and Lactic Acid Contents and Oxygen Saturation of the Blood in Various Divisions of the Vascular Canal During Stable Hypotonia (M +m)

Index determined	Initial state			Stable hypotension											
				hemostat phase						Post-hemostat phase					
				period of removal of blood	period of stabilization			period of re-entry blood			initial period	Terminal period			
	A	VC	PV		A	VC	PV	A	VC	PV		A	VC	PV	
Sugar (in mg %)	214,2 ±23,4	205,2 ±21,6	213,0 ±19,4	Insufficient data	302,5 ±18,9	301,3 ±20,1	295,4 ±22,9	244,3 ±25,0	251,5 ±27,6	248,0 ±32,2	Blood was not investigated	239,8 ±24,3	254,8 ±27,2	239,5 ±21,2
P					<0,05	<0,05	<0,05	<0,05	>0,05	>0,05		>0,05	>0,05	>0,05
Lactic acid (in mg %)	15,8 ±4,0	18,2 ±5,1	14,2 ±2,9		99,8 ±16,1	141,6 ±33,8	154,8 ±47,9	175,3 ±19,3	188,6 ±41,0	211,6 ±54,5		117,5 ±6,2	123,9 ±12,6	209,2 ±34,3
P					<0,01	<0,01	<0,01	<0,05	>0,05	>0,05		<0,05	<0,05	>0,05
Oxygen saturation (in % of oxyhemoglobin)	90 ±4,0	41 ±14,6	68 ±4,1		92 ±4,0	26 ±3,0	22 ±3,0	—	—	—		86 ±3,3	33 ±5,1	33 ±0,8
P					>0,05	>0,05	<0,01					>0,05	>0,05	<0,05

Notations the same as in Table 1.

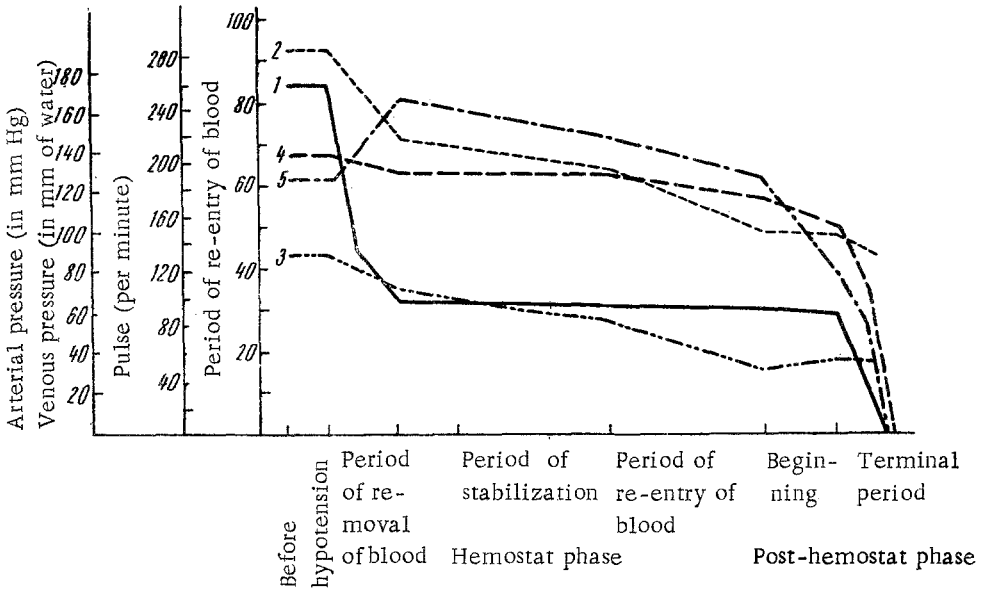

Fig. 2. Changes in the arterial pressure, pressure in the portal vein and vena cava, pulse, and respiration in stable hypotension. The conditions of compilation of the graphs and notations are the same as in Fig. 1.

hypoxia in the disturbance of carbohydrate metabolism (Table 2). Two phases were distinguished in the development of this state, arising in the use of a hemostat: the hemostat phase, characterized by a stable maintenance of the arterial pressure at a set level, and the post-hemostat phase, which sets in immediately after disconnection of the hemostat and is characterized by a progressive drop in the arterial pressure. During the hemostat phase, which is of the greatest interest to us, three periods were detected: removal of blood—development of hypovolemia; stabilization—conservation of reduced blood volume at a certain pressure; re-entry of blood—return to normovolemia.

During stable hypotension, a reduction of the pulse and respiration frequencies and a decrease in the pressure in the portal vein and vena cava were noted, with little change in the portocaval gradient, i.e., phenomena to some degree similar to shock developed (Fig. 2). The oxygenation of the arterial blood during hypotension varied little until the terminal period set in. Thus, in contrast to traumatic shock, during stable hypotension the oxygen supply of the liver was disrupted, not only through the portal vein, but also through the hepatic artery.

With the development of stable hypotension, the appearance of hyperglycemia was noted earlier (during the period of removal of the blood) (these data were not included in the table on account of their small number). During the period of stabilization, the blood sugar level remained high, in the absence of any difference in its content among the vessels. The period of re-entry of the blood was characterized by a decrease in the sugar concentration to figures approaching the initial level. At this time, signs of incipient decompensation of the blood circulation were observed, in particular, a reduction of the vascular tonus [15].

The changes in the lactic acid content in the blood during hypotension were essentially analogous to those during traumatic shock and were also independent of the degree of oxygen saturation of the blood. But since hypotonic hypoxia was more profound, the lactacidemia also was more pronounced.

In conclusion, it may be stated that traumatic shock and stable hypotension are characterized by similar changes in the carbohydrate metabolism of the liver, evidently largely determined by the disturbance of its oxygen balance [14, 18].

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