INDICES OF CARBOHYDRATE METABOLISM IN THE KIDNEY AND PHOSPHOHEXOISOMERASE ACTIVITY IN THE PERIPHERAL BLOOD AT VARIOUS STAGES OF TRAUMATIC SHOCK

B. A. Saakov, V. I. Shepotinovskii, E. D. Dalenov, and Z. I. Garmotenko

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Traumatic shock in dogs is accompanied by marked changes in the glycogen and glucose levels and the phosphorylase, glucose-1-phosphatase, glucose-6-phosphatase, and phosphohexoisomerase activities in the kidney tissues. High activity of these enzymes was detected in the arterial and venous blood during the period of shock. Transient hypotension is accompanied by higher phosphohexoisomerase activity in the whole blood, and prolonged hypotension by its higher activity in the serum.

Considerable changes in kidney function have been described in traumatic shock [2, 7, 12]. However, few studies have been made of the dynamics of kidney metabolism during shock [14].

Accordingly, in the investigation described below, certain indices of carbohydrate metabolism in the kidney tissues and peripheral blood were studied at various stages of traumatic shock.

EXPERIMENTAL METHOD

In experiments on mongrel dogs, traumatic shock was induced by Cannon's method. Its severity was assessed on the basis of the animal's general reaction, the blood pressure in the common carotid artery, and changes in the common carotid artery, and changes in respiration recorded on a kymograph. Kidney tissue for investigation was taken intravitally under local procaine anesthesia. Kidneys of intact dogs served as the control. Arterial and venous blood were obtained by catheterization from the descending aorta and inferior vena cava. Since the intensity of glycogenolysis and gluconeogenesis in kidney tissue is high [1, 4, 6, 13], the content of glycogen and glucose and the activity of enzyme connected with their metabolism were determined in kidney biopsy material. The total glycogen content was determined by Pflüger's method, the "true" glucose by Nelson's method [9], phosphorylase activity by the method of D. L. Ferdman and E. F. Sopin, and activity of glucose-1-phosphatase and glucose-6-phosphatase by the increase in the concentration of inorganic phosphorus in samples incubated in 0.2 M acetate buffer (pH 6.5) with the addition of the corresponding substrate in a concentration of 0.006 M at 37°C for 20 min. Inorganic phosphorus was determined by the method of Lowry and Lopez. Phosphohexoisomerase activity of the kidney, whole blood, and serum was determined by Ezerskii's method [3].

EXPERIMENTAL RESULTS

Immediately after trauma, in the erectile phase of shock, the glycogen content in the kidney tissue fell sharply while the activity of the phosphorylase responsible for its breakdown was increased. The content of "true" glucose and activity of glucose-6-phosphatase also were reduced. Phosphohexoisomerase activity was almost doubled, but no glucose-1-phosphatase activity could be detected. It can be assumed

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TABLE 1. Indices of Carbohydrate Metabolism of the Kidney in the Various Phases of Traumatic Shock (M± m; results of 7 experiments)

Index	Initial data (160 mm Hg)	Erectile phase of shock (220 mm Hg)	Hypotension		
			1 h (45 mm Hg)	2 h (45 mm Hg)	24 h (45-60 mm Hg)
Glycogen (in mg %)	136,3± 11,2	74,7± 11,4	188,2± 5,2	76,6± 17,3	111,7± 5,3
P		<0,01	<0,01	<0,02	<0,02
Glucose (in mg %)	132,2± 8,4	71,2± 4,9	294,6± 45,2	76,9± 5,8	112,8± 3,9
P		<0,001	<0,01	<0,001	<0,1
Phosphorylase (in μmoles P/g)	46,6± 3,6	63,6± 3,6	26,4± 2,0	39,0± 4,2	44,2± 3,4
P		<0,01	<0,001	>0,05	>0,2
Glucose-1-phosphatase (in μmoles P/g)	No activity detected		17,1± 2,7	12,6± 2,4	15,2± 1,5
P			<0,001	<0,001	<0,001
Phosphohexoisomerase (in μmoles FR/g)	2,61± 0,26	4,0± 0,4	10,1± 1,4	5,7± 0,4	5,4± 0,3
P		<0,01	<0,001	<0,001	<0,001
Glucose-6-phosphatase (in μmoles P/g)	70,3± 3,5	55,1± 4,5	62,6± 1,2	61,7± 3,8	59,1± 1,7
P		<0,02	>0,05	0,1	<0,02

Legend: μmoles P/g denotes number of micromoles phosphorus per gram fresh tissue; μmoles FR/g – number of micromoles of fructose per gram fresh tissue; P shows significance of differences from initial data.

TABLE 2. Phosophohexoisomerase Activity in Serum and in Whole Arterial and Venous Blood (in μmoles FR/ml) of Dogs at Different Stages of Traumatic Shock (M± m; from results of 7 experiments)

Material studied	Initial data	Erectile phase	Hypotension		
			2 h	24 h	48 h
Arterial blood	0,73± 0,07	1,19± 0,1	1,45± 0,06	1,73± 0,09	1,53± 0,06
P		<0,01	<0,001	<0,001	<0,001
Venous blood	0,84± 0,05	1,08± 0,04	1,63± 0,13	1,84± 0,22	1,82± 0,09
P		<0,01	<0,001	<0,01	<0,001
Serum from arterial blood	1,13± 0,16	1,65± 0,09	1,41± 0,06	1,73± 0,24	2,21± 0,18
P		<0,02	>0,05	>0,05	<0,01
Serum from venous blood	1,34± 0,34	1,52± 0,12	1,27± 0,04	2,02± 0,27	2,07± 0,33
P		>0,5	>0,5	>0,05	>0,05

Legend: μmoles FR/ml denotes number of micromoles of fructose per ml whole blood or serum.

that the utilization of glucose-1-phosphate in the kidneys is shifted toward the formation of glucose-6-phosphate in the erectile phase of shock just as in the intact kidney.

The character of the metabolic disturbances in the hypotensive phase of shock depended on the duration of the fall in blood pressure. If the duration of hypotension was less than 1 h, the glycogen and glucose content were sharply increased, while the phosphorylase and glucose-6-phosphatase activities were reduced. The intensity of glycolysis was evidently increased, since the phosphohexoisomerase activity was significantly increased by more than 4 times (Table 1).

Hypotension under 2 h in duration, due to shock, was accompanied by a sharp decrease in the glycogen and glucose content in the kidney and a decrease in the phosphorylase and glucose-6-phosphatase activities, but the level of glucose-1-phosphatase and phosphohexoisomerase activity remained high.

These features of metabolism in the kidneys were evidently attributable to changes both in the general hemodynamics and in the local circulation in the kidney, and also to a disturbance of the neurohumoral adaptive mechanisms regulating metabolic processes in the kidney tissue [5, 6, 8, 10].

The results given in Table 2 show that traumatic shock is accompanied by sharp changes in phosphohexoisomerase activity not only in the kidney tissue, but also in the peripheral blood.

It will be noted that phosphohexoisomerase activity in whole blood was much higher after hypotension for 2 h than in the erectile phase of shock.

It is important to emphasize that during prolonged hypotension (24-48 h, arterial pressure 50-80 mm Hg) the redistribution of enzyme activity in the whole blood and serum showed some special features. Unlike in the preceding phases of shock, the phosphohexoisomerase activity in the serum (especially from venous blood) was higher than in whole blood. Presumably the level of enzyme activity in the blood during the first hours of hypotension was determined by the "flushing" of phosphohexoisomerase from the tissue and also by the character of the metabolism of the blood cells, which are marked by intensive glycolysis [11].

The results described below show that traumatic shock is accompanied by marked disturbances of carbohydrate metabolism in the kidney tissue.

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