ENERGY METABOLISM IN THE RAT BRAIN IN THE COURSE OF TRAUMATIC SHOCK

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UDC 617-001.36-092.9-07:616.831-092:612.013.7

The concentration of high-energy phosphates (ATP, creatine phosphate), the total content of adenyl nucleotides, and the energy potential of the brain cells did not change significantly in experimental animals after trauma to the soft tissues of the thigh, until the terminal phase. The intensity of glycolysis was increased. In the terminal phase anaerobic processes predominated somewhat over aerobic. The absence of changes in the concentration of high-energy phosphates in the rat brain in traumatic shock is probably associated with centralization of the circulation and it is evidence that no "critical" exhaustion of energy takes place in the brain.

KEY WORDS: brain, traumatic shock.

The state of the energy metabolism in the brain is considered to play an important role in the pathogenesis of traumatic shock. However, there is little information on this subject in the literature, and such as there is, is contradictory. According to some workers [1-3],10, 11], in traumatic, tourniquet, and hemorrhagic shock the concentrations of ATP and creatine phosphate (CP) in the brain are reduced by a varied degree and the levels of inorganic phosphate and lactate are increased. Meanwhile other workers [9, 13, 14] found no change in the ATP and CP concentrations in the brain in lethal tourniquet shock until the animal was in a terminal state.

The object of the present investigation was to study the energy metabolism in the rat brain during the course of traumatic shock.

EXPERIMENTAL METHOD

Experiments were carried out on 40 male albino rats weighing 130-170 g. Shock was produced by Cannon's method by crushing the soft tissues of the thigh. The erectile, torpid, and terminal phases were distinguished in the course of shock; the torpid phase was subdivided into the beginning, the period of stabilization of the arterial blood pressure (BP), and the end of the torpid phase. The BP was recorded by a mercury manometer in the femoral artery. The energy metabolism was studied during the period of stabilization (BP 90-100 mm Hg), at the end of the torpid phase (BP about 40 mm Hg), and in the terminal phase (BP 25-30 mm Hg) of shock. The mean duration of shock was 70 min. Two groups of animals were used as the control: intact rats (control 1) and animals subjected to fixation and recording of the BP in the same manner as the experimental animals (control 2). The animals were killed under superficial ether anesthesia by immersion in liquid nitrogen. The brain was ground to a powder which was transferred quantitatively into cold 6% $HClO_4$ at the rate of 6.5 ml to 2 g of tissue and homogenized in a glass homogenizer. The samples were centrifuged in the cold for 15 min at 3000 g, the residue was washed with 6% $HClO_4$ to a final dilution of the tissue of 1:5, and they were again centrifuged under the same conditions. Protein-free tissue extracts were neutralized with 5 M K_2CO_3 to pH 3.5 for the determination of pyruvate, lactate,

I. I. Dzhanelidze Leningrad Emergency Aid Scientific-Research Institute. (Presented by Academician of the Academy of Medical Sciences of the USSR V. S. Il'in.) Translated from Byulleten' Éksperimental'noi Biologii i Meditsiny, Vol. 78, No. 11, pp. 27-29, November, 1974. Original article submitted November 2, 1973.

TABLE 1. Concentrations (in μmoles/g wet weight of tissue) of Energy Metabolites in the Rat Brain during Traumatic Shock

Experimental animals	Statistical index	ATP	ADP	AMP	ATP/ADP	Total adenyl nucleotides	Energy potential	CP	Lactate	Pyruvate	Lactate Pyruvate
Control 1 (intact animals)	$M \pm m$ n	2,30 ±0,09 12	0,52 ±0,03 11	0,16 ±0,018 11	4,81 ±0,43 11	3,06 ±0,10 11	0,86 ±0,012 11	3,31 ±0,13 13	3,07 ±0,23 13	0,174 ±0,017 12	20,1 ±1,8 12
Control 2 (fixed animals)	$M \pm m$ n P_1	2,37 ±0,12 6 >0,5	0,47 ±0,04 6 >0,2	0,20 ±0,03 6 >0,2	5,27 ±0,78 6 >0,5	3,01 ±0,12 6 >0,5	0,85 ±0,011 6 >0,5	3,11 ±0,19 6 >0,2	3,39 ±0,25 6 >0,2	0,214 ±0,026 6 >0,2	17,2 ±3,1 6 >0,2
Traumatic shock Torpid phase	$M \pm m$ n	2,19 ±0,08 9	0,54 ±0,05 9	0,18 ±0,02 9	4,32 ±0,40 9	2,92 ±0,10 9	0,84 ±0,012 9	2,73 ±0,18 10	4,61 ±0,31 10	0,252 ±0,020 10	19,8 ±2,1 10
Period of stabilization	P_1 P_2	>0,2 >>0,2	>0,5 >>0,2	>0,2 >>0,5	>0,2 >>0,5	>0,2 >>0,5	>0,1 >>0,2	<0,02 <<0,1	<0,0027 <<0,0027	<0,01<<0,02	>0,5 >>0,5
End of torpid phase	$M \pm m$ n P_1 P_2	2,04 ±0,22 6 >0,2 >>0,5	0,47 ±0,03 6 >0,2 >>0,5	0,22 ±0,05 6 >0,2 >>0,5	4,58 ±0,47 6 >0,5 >>0,5	2,73 ±0,22 6 >0,1 >>0,2	0,83 ±0,018 6 >0,2 >>0,2	3,19 ±0,36 6 >0,5 >>0,5	5,42 ±0,59 6 <0,0027 <<0,01	0,248 ±0,024 6 =0,02<<0,1	24,2 ±4,8 6 >0,2 >>0,2
Terminal phase	$M \pm m$ n P_1 P_2	2,03 ±0,23 6 >0,2 >>0,2	0,57 ±0,08 6 >0,5 >>0,5	0,36 ±0,13 6 >0,2 >>0,5	3,93 ±0,87 6 >0,5 >>0,2	2,98 ±0,28 6 >0,5 >>0,5	0,76 ±0,082 6 >0,2 >>0,2	3,05 ±0,53 6 >0,5 >>0,5	6,77 ±1,07 6 <0,01<<<0,02	0,231 ±0,044 6 >0,2<<<0,05	31,3 ±4,5 6 <<0,05

Legend: P_1 and P_2), degree of significance relative to controls 1 and 2, respectively.

ADP, and AMP, and to pH 7.5 for the determination of ATP and CP. After 30 min the residue was removed by centrifuging and the supernatant fractions were kept at 0°C and used for analysis within a few hours. Pyruvate [5], lactate [6], ATP, and CP [7], and ADP and AMP [8] were determined by enzymic methods on the SF-4A spectrophotometer at 340 nm. The energy potential of the brain cells was calculated by the method of Atkinson and Walton [4].

EXPERIMENTAL RESULTS

Fixation of the animals to the frame and catheterization of the femoral artery under local anesthesia caused no change in the energy metabolism in the rats' brain (Table 1, control 2). The concentrations of the adenyl nucleotides, their total concentration, and also the energy potential of the cells in the brain of the animals developing traumatic shock did not differ significantly from their original levels in all phases studied. The CP concentration remained unchanged during the course of the shock except for a very small (10%) but significant decrease during the period of stabilization. In the brain of the experimental rats an increase in the pyruvate concentration was observed and it was about equal in degree in all phases. During the development of shock the lactate concentration rose gradually and in the terminal phase it was almost twice as high as in the control. In the period of stabilization and at the end of the torpid phase the lactate/pyruvate ratio was virtually indistinguishable from its initial value; in the terminal phase it was significantly increased by 30% on account of a more rapid rise in the lactate concentration.

In all the phases of traumatic shock investigated the content of high-energy phosphates and the degree of phosphorylation, the total reserves, and the energy charge of the system of adenyl neucleotides thus remained substantially unchanged. Intensification of glycolysis was marked by a simultaneous increase in the concentrations of lactate and pyruvate, so that the lactate/pyruvate ratio increased a little only in the terminal phase, indicating some predominance of anerobic processes over aerobic during this period.

The results show that the energy metabolism in the rat brain during traumatic shock is maintained until the terminal state at an adequate level for keeping the content of high-energy phosphates within the original limits. This is probably achieved through centralization of the circulation in shock, as a result of which the brain and heart are provided with a better blood supply than the peripheral tissues. The absence of changes in the concentration of high-energy compounds in the brain of rats subjected to traumatic shock does not confirm the hy-

pothesis of the "critical" exhaustion of energy in this vitally important organ put forward by some authors [2, 3, 12]. However, the results described above cannot rule out the possiblity of exhaustion of energy in some local areas of the brain, its nerve centers, individual cells, or their subunits, undetectable by analysis of the whole brain [9, 13].

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