STATE OF THE HISTAMINE – HISTAMINASE SYSTEM IN THE COURSE OF TRAUMATIC SHOCK

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The histamine content and diamine oxidase activity in the blood and tissues were studied in dogs in the course of evolution of traumatic shock. Phasic changes were found in histaminase activity which led to the development of excessively high histamine levels during the development of traumatic shock. Imbalance between monoamines and histamine is considered to play an important role in the genesis of the hemodynamic and central disorders in shock.

KEY WORDS: histamine–histaminase system; traumatic shock.

The high biological activity and wide range of action of histamine determine its role in many different reactions of the body under normal physiological and pathological conditions and, in particular, in reactions to trauma [1-3, 5, 7, 10, 12-14, 16].

The object of this investigation was to study the content of histamine and the mechanism of its liberation and inactivation in the course of development of traumatic shock.

EXPERIMENTAL METHOD

Experiments were carried out on 50 adult noninbred unanesthetized male dogs. Traumatic shock was produced by crushing the soft tissues of the thigh.

The histamine content and histaminanase activity of the tissues were investigated in animals decapitated 20-30 sec and 5 min after injury, in the torpid stage and in the terminal period of shock.

Histamine was extracted from the blood and tissues (frontal cortex, hypothalamus, myocardium, liver, kidney) with butanol [15] and estimated by a chemical method [18]. Diamine oxidase activity was estimated from the decrease in the histamine added to the test serum (or tissue homogenate) after incubation for 24 h. The histaminopectic index was determined by Parrot's method [17] with a colorimetric end point.

EXPERIMENTAL RESULTS AND DISCUSSION

The mean blood histamine concentration in the intact dogs was 18.53 ± 0.82 $\mu g\%$. In the first 5 min after trauma, while the animals were in a state of marked excitation, some tendency was observed for the blood histamine level to rise. The histamine concentration continued to rise parallel with the increase in severity of the clinical picture. In the terminal period the histamine level reached its maximal values, 2.5 times higher than initially ($P < 0.001$).

One possible cause of the accumulation of histamine in the blood in shock could be a change in the activity of diamine oxidase, which increased slightly after trauma and then declined (Fig. 1).

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TABLE 1. Histamine Concentration and Diamine Oxidase Activity in the Tissues of Dogs during the Development of Traumatic Shock (M ± m)

Test object	Control	After trauma				
		immediately	5 mm	when arterial pressure 60-80 mm	when arterial pressure 30-40 mm	terminal period
Frontal cortex	2,47±0,58 18,39±1,89	2,50±0,26 19,43±0,76	2,06±0,39 20,59±0,79	2,89±0,34 16,79±0,69	4,01±0,34† 14,00±1,44*	4,58±0,48† 11,45±1,11†
Hypothalamus	6,47±0,64	6,48±0,47	6,08±0,48	7,33±0,58	8,61±1,26*	10,40±0,81†
Myocardium	7,78±0,48 10,64±0,56	7,47±0,79 10,88±0,59	6,8±0,75 12,39±0,81	8,59±0,67 9,00±0,55*	9,94±1,01* 8,46±0,62*	11,70±1,15* 6,37±0,93†
Liver	24,91±1,35 16,11±1,41	22,15±2,13 16,92±0,99	21,69±2,18 18,59±0,76	30,81±1,99* 16,02±1,13	35,44±2,06‡ 11,27±0,75†	40,11±3,19‡ 10,35±1,07†
Kidney	5,22±0,36 39,92±1,63	4,83±0,39 41,82±2,18	4,11±0,32* 46,2±3,24	6,08±0,49 34,57±2,75	8,36±0,75† 30,31±3,72*	10,30±0,57‡ 28,70±1,98†

*P ≤ 0.05 compared with control
†P < 0.01 compared with control
‡P < 0.001 compared with control
Note. Numerator gives histamine concentration in tissues (in μg/g); denominator gives histaminase activity (in μg histamine decomposed).

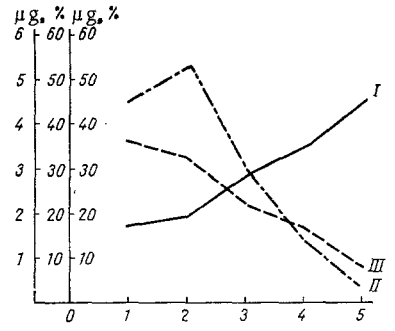

Fig. 1. Histamine concentration, diamine oxidase activity, and histaminopectic index in blood of dogs during development of traumatic shock. Abscissa: 1) initial data; 2) 5 min after trauma; 3) arterial pressure 60-80 mm; 4) arterial pressure 30-40 mm; 5) in terminal phase of shock; I) histamine concentration (in μg %); II) diamine oxidase activity (in μg histamine decomposed); III) histaminopectic index (in %).

Simultaneously with the elevation of the blood histamine concentration during the development of shock there was a marked decrease in the histaminopectic index. This observation is confirmed by data in the literature on the role of a complex formed between histamine and the γ-globulin fraction of the blood proteins in the protective and adaptive reactions of the organism [8, 20]. The decrease in the intensity of histamine binding in shock, in conjunction with inhibition of histaminase activity, must be interpreted as an important factor leading to the accumulation of free histamine in the blood.

Investigation of the tissue histamine revealed an irregular distribution of the substance in the tissues and organs of the control group of animals (Table 1). All the tissues studied had well-marked histaminolytic activity, in agreement with data in the literature [2, 6].

The initial reaction to trauma was characterized by a tendency for the tissue histamine concentration to fall; the degree of this fall varied in the different organs: the histamine level remained highest in the structures of the brain and it was lowest in the kidney.

In the torpid stage a gradual increase in the histamine concentration took place in all tissues, reaching its maximum in the terminal period of shock, when it was 1.5-2 times above the initial value.

The tissue histaminase activity, like that of the blood, underwent biphasic changes during the development of traumatic shock. The initial slight increase in histaminase activity in the torpid phase of shock was followed by a sharp decline.

The results show that the development of traumatic shock is accompanied by the accumulation of massive quantities of histamine in the blood and tissues. The changes observed in the histamine–histaminase system correlate with information in the literature showing that under the influence of extremal stimulation (trauma, ionizing radiation, radial acceleration, and so on) large quantities of tissue histamine are liberated [2, 7, 11, 14]. Together with other active substances, the histamine participates in the formation of the pathological process and is largely responsible for determining its severity and outcome.

An important role in the genesis of the hemodynamic and central disturbances in shock may be played by the upsetting of the physiological ratio between corticosteroids [4], monoamines (catecholamines and serotonin), and histamine [9, 19].

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