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RESPONSE TO A COMBINATION OF SEVERE HEAD INJURY AND ACUTE MASSIVE BLOOD LOSS

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Acute experiments on dogs showed that the character of the response to a combination of craniocerebral trauma and acute blood loss depends on the order of infliction of these extremal conditions. Craniocerebral trauma inflicted in the early period of hemorrhagic shock does not significantly affect the dynamics and outcome of the underlying pathological process. Hemorrhagic shock arising after craniocerebral trauma does so as a result of a much smaller blood loss and it follows a more severe course.

KEY WORDS: *craniocerebral trauma; acute blood loss; hemorrhagic shock.*

The writers showed previously that shock does not develop in severe craniocerebral trauma [1]. Only if craniocerebral trauma is combined with mechanical trauma to the femur did shock develop, and it was marked by certain special features [2].

In this investigation the character of development of shock arising in response to a combination of craniocerebral trauma and acute massive blood loss was studied.

EXPERIMENTAL METHOD

Experiments were carried out on 54 adult dogs. The animals were bled as a one-stage procedure from the femoral artery until the blood pressure was 40-45 mm Hg.

Craniocerebral trauma consisted of infliction of 300-350 blows with an iron hammer (weight 450 g) on the vault of the skull through a rubber pad 8 mm thick. The changes in blood pressure in the femoral artery, pulse rate, and respiration, circulating blood volume (by the T-1824 dye dilution method), bioelectrical activity of the brain (unipolar recording with needle electrodes from the parietal region), ECG (standard lead II), and EMG (bipolar recording from the posterior cervical muscles) were recorded on the 4EEG-1 electroencephalograph throughout the experiment. Changes in spontaneous activity and the effect of photic (10 flashes/sec) and acoustic stimulation (2000 Hz) were investigated. To obtain more reliable data on the dynamics of the process, the arterial blood pressure was recorded simultaneously on two kymographs: on one continuously throughout the experiment with a paper-winding speed of 14 cm/h, and on the other at the most important stages of the experiment, with a paper-winding speed of 1 mm/sec. The conjunctival blood vessels were

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TABLE 1. Changes in Some Indices of the State of Dogs After Combined Craniocerebral Trauma and Acute Blood Loss (M ± m)

Series and stages of experiments	Blood pressure, mm Hg	Pulse rate, beats/min	Respiration rate, per minute	Rectal temperature, °C	Subcutaneous temperature, °C	Hematocrit index	Hemoglobin concentration, g%	Red cell count, millions	Circulating blood volume, ml/kg	Circulating plasma volume, ml/kg
I. Acute blood loss:										
Initial values	121±2	66±10	38±6	37,8±0,2	36,4±0,5	50±0,4	15±0,3	4,9±0,7	78±1,3	42±0,8
5 min after blood loss (phase of depression)	45±3*	208±12*	29±4*	37,7±0,3*	34,7±0,7	43±1,4	14±0,3	3,7±0,1	43±2,3*	23±1.4*
After 40-60 min (period of stabilization of hemorrhagic shock)	62±4*	234±10*	27±2*	37,8±0,4	33,4±0,8	44±1,2	15±0,7*	3,4±0,1	42±3,8*	23±2,1*
15-30 min before end of experiment	26±3*	180±19*	20±3*	37,8±0,6	32,2±0,9	45±1,3	15±0,5*	3,5±0,2	41±1,7*	22±2,5*
II. Craniocerebral trauma										
Initial values	135±5	85±9	49±17	38,6±0,6	36,8±0,5	47±1,2	14±0,3	3,8±0,2	78±1,6	42±0,8
5 min after trauma	137±6	122±12	50±14	38,7±0,5	36,4±0,5	44±1,0	13±0,3	3,7±0,2	76±2,3	40±0,7
After 40-60 min	139±10	118±24	61±14	38,5±0,7	35,7±0,8	43±1,3	11±0,2	3,4±0,3	76±1,8	41±0,8
15-30 min before end of experiment	132±5	121±27	44±7	38,8±0,7	35,4±0,9	42±1,9	12±0,4	3,5±0,4	74±2,1	40±0,9
III. Craniocerebral trauma superposed on acute massive blood loss										
Initial values	130±7	59±4	44±3	38,0±0,2	36,6±0,3	46±0,8	12±0,7	3,5±0,3	82±1,4	43±0,7
5 min after blood loss	48±5*	196±14*	38±4*	37,8±0,4	34,4±0,4	43±1,0	12±1,0	3,3±0,5	45±0,9*	25±0,7*
5 min after trauma	56±6*	186±20*	31±4*	37,8±0,5	33.4±0,5	45±1,2	12±0,6	2,8±0,3	41±1,2*	23±0,8*
After 40-60 min (period of stabilization of hemorrhagic shock)	66±7*	214±20*	26±4*	37,6±0,5	33,5±0,4	42±1,0	9±0,4*	3,1±0,2	39±1,1*	22±0,8*
15-30 min before end of experiment	33±4*	183±28	18±2*	37,5±0,6	31.5±0,5*	41±1,7	9±0,9*	2,6±0,3	37±1,2*	22±0,9*
IV. Acute blood loss after craniocerebral trauma										
Initial values	128±2	81±12	38±4	38,3±0,2	36,8±0,3	44±1,2	12±0,7	3,4±0,1	83±1,3	43±0,9
5 min after trauma	124±8	124±17	32±4	38,2±0,5	34,6±0,7	43±1,2	12±0,7	3,3±0,1	81±1,7	42±1,0
5 min after blood loss	50±5*	159±15	29±4*	38,0±0,6	34,2±0,7	41±1,3	12±0,9	3,3±0,2	43±0,8*	26±1,0*
40-60 min after blood loss (period of stabilization of hemorrhagic shock)	54±7*	138±16	26±4*	38,0±0,4	34,0±0,6	40±2,6	11±1,0	3,1±0,2	40±1,6*	26±2,2*
15-30 min before end of experiment	32±4*	118±20	16±3*	37,9±0,6	33,4±0,6	35±1,0	9±0,9	2,7±0,1	33±1,7*	22±2,0*

*Indices differing statistically significantly ($P < 0.05$) from those in series II.

TABLE 2. Outcome of Experiments with Combined Craniocerebral Trauma and Acute Blood Loss (M ± m)

Series of experiments	Number of animals	Severity of trauma (number of blows)	Volume of blood loss, ml/kg	Outcome	
				number surviving	length of survival of animals which died, h
I. Acute blood loss	13	—	37,2±2,0	—	2,3±0,4
II. Craniocerebral trauma	21	309±39	—	3	86,6±29,2
III. Craniocerebral trauma in early period of hemorrhagic shock	10	370±52	33,9±0,6	—	3,2±0,5
IV. Blood loss after craniocerebral trauma	10	320±52 >0,05	15,2±1,9 <0,05	—	1,8±0,2 <0,05

photographed at these same stages of the experiment (MBS-2 microscope, Zenit camera, RF-2 film) and the diameter of the microvessels was then estimated quantitatively [3]. The experiment continued until the animals died or the original values of the various parameters had been restored.

EXPERIMENTAL RESULTS

The results are given in Tables 1 and 2.

In the experiments of series I the dynamics of the posthemorrhagic reaction was studied. The results confirmed earlier data on the phasic course of this response. Immediately after bleeding there was a phase of depression, after which the typical symptom complex of hemorrhagic shock developed, in three stages (early, stabilization, and late). Next followed the phase of collapse, a terminal state developed, and the animals died. The changes in these parameters are evidence that the periods of hemorrhagic shock reflect fundamentally different functional states of the body.

In series II the dynamics of the response of the animal to severe craniocerebral trauma was investigated. During infliction of the trauma a transient phase of excitation was observed (restless movements, inhibition of the corneal reflexes, nystagmus, disturbances of the rhythm and depth of respiration, elevation of the arterial pressure, followed by a short phase of depression). The indices then gradually returned to their initial values. The dogs were taken from the table 4-6 h after trauma. They lay quietly and still, and sometimes tried to crawl or stand up. Toward the end of the first or on the second day the state of the animals gradually improved and, in their external appearance and behavior, they differed only a little from healthy animals. However, toward the end of the second or on the third day their condition again deteriorated sharply; adynamia, dyspnea, and convulsions developed. Most of the dogs died. At autopsy cerebral edema with petechial hemorrhages into the brain tissue and small epidural and subdural hematomas were found. No special changes were discovered in the other organs.

In series III craniocerebral trauma was inflicted 15 min after blood loss (in the early period of hemorrhagic shock). Under these conditions it was accompanied by a less marked phase of excitation. The motor and pressor responses were almost absent, although depression of the corneal reflexes, nystagmus, and changes in respiration were of the same intensity and duration as in the animals in series II. The changes in all the indices 15 min after trauma pointed to the typical phasic course of the posthemorrhagic reaction. Hemmorrhagic shock developed in the usual way and all the animals died.

In series IV bleeding occurred 15 min after craiocerebral trauma, immediately after disappearance of the phenomena of excitation and a subsequent depression. Under these circumstances a smaller volume of blood loss was needed to lower the blood pressure to 40-45 mm Hg. The hemorrhagic shock also followed a more severe course as shown by the shorter period of survival of the animals.

It can be concluded from these results that craniocerebral trauma superposed on developed hemmorrhagic shock does not significantly affect the course of this pathological process. Criocerebral trauma inflicted before blood loss, however, increases the sensitivity of the CNS to hypoxia and is adversely reflected in the mechanism of formation of hemorrhagic shock.

Shock therefore develops after a much smaller blood loss, its course is more severe, and the pathological changes are more marked. On the whole, when craniocerebral trauma is combined with acute blood loss the dominant picture is one of hemorrhagic shock and it is this which determines the character of the pathological process and its outcome.

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