EFFECT OF THE INITIAL FUNCTIONAL STATE OF THE NERVOUS SYSTEM ON THE DYNAMICS OF TRAUMATIC SHOCK

D. M. Sherman

UDC 617-001.36-036.1:616.831/.832-008.1

Experiments on rabbits showed that the response to severe mechanical trauma depends on the initial functional state of the nervous system. In particular, after intravenous injection of procaine, chlorpromazine, or thiopental the late period of shock does not develop and most animals survive. Preliminary administration of adrenalin or noradrenalin hastens death of the animals after trauma.

An important role in the trigger mechanism of traumatic shock is ascribed to the development of inhibition in the central nervous system under the influence of an excessive flow of afferent impulses from the site of trauma [1, 3-7]. It can be postulated on the basis of this concept that differences in the initial functional state of the nervous system should have a significant effect on the dynamics of this pathological process. This hypothesis was tested by an experimental investigation the results of which are described below.

EXPERIMENTAL METHOD

Shock was produced in 160 adult rabbits by inflicting several hundred blows with an iron rod (covered with thick-walled rubber tube) on the inner surface of the thigh. The blood pressure (in the common carotid artery), pulse rate, respiration, temperature in the rectum and muscles ("Bioterm" electrothermometer) EEG (unipolar recording with needle electrode from the parietal regions), ECG (standard lead II) and EMG (bipolar recording with needle electrodes from the biceps femoris muscle) were recorded during the experiment. The various types of electrical activity were recorded on a fourchannel electroencephalograph (4ÉÉG-1). Responses to compression of the opposite carotid artery for 5 sec, nociceptive stimulation (square pulses, 0.25 mA, 5 sec) and acoustic stimulation (the sound of an automobile horn, duration 5 sec) were investigated. In the experimental series 2-3 min before application of trauma to the animals, various neurotropic drugs made up in 1-2 ml physiological saline were injected. The experiments lasted until it was clear whether or not the animals would survive and for how long.

EXPERIMENTAL RESULTS AND DISCUSSION

In 2 groups of experiments of the control series a distinct phasic course of the posttraumatic reaction was observed. From analysis of data in the literature [1-3, 5] and the results of these experiments it was possible to distinguish a phase of excitation (during infliction of trauma, 310 ±33 blows), and a phase of inhibition (prostration, arterial and muscular hypotonia) immediately after trauma in the first group (40 experiments). After a transient intermediate phase, the phase of typical traumatic shock appeared, during which 3 periods could easily be distinguished: early (gradual increase of arterial pressure, improvement in the electrophysiological and certain other indices), stabilization, and late (progressive worsening of all these indices). This, the longest phase, was followed by the phase of collapse (a critical fall of arterial pressure, extinction of the electrical activity of the brain). The terminal state followed within a few minutes. All the animals died.

Experimental Laboratory, L'vov Military Hospital. (Presented by Academician of the Academy of Medical Sciences of the USSR A. A. Vishnevskii.) Translated from Byulleten' Éksperimental'noi Biologii i Meditsiny, Vol. 74, No. 10, pp. 29-32, October, 1972. Original article submitted March 26, 1971.

TABLE 1. Changes in Principal Indices in Rabbits Receiving Neurotropic Drugs before Trauma (M ± m)

Index	Control group		Experimental series			Experimental series						
	shock	collapse	I Adrenalin 0.05 mg/kg	II Noradrenalin 0.5 mg/kg	III Sucinylcholine 1 mg/kg	IV Controlled respiration without succinylcholine	V Chlorpromazine 1 mg/kg	VI Procaine, 10 mg/kg intravenously	VII Thiopental 20 mg/kg	VIII Procaine anesthesia of thigh 100 mg/kg	IX Azamethonium bromide 1 mg/kg	X Atropine 0.05 mg/kg
Number of animals	40	20	10	10	10	10	10	10	10	10	10	10
Trauma (number of blows)	310±33	254±26	200±23	350±25	260±24	330±29	390±44	380±42	420±60	430±54	330±32	340±25
Number of animals surviving	1	—	1	—	4	—	7	6	6	—	1	4
Length of survival before death (in min)	114±31	9±2	83±46	93±32	127±45	132±17	471±128	98±8	138±76	287±73	112±64	115±53
Arterial pressure (in mm):												
Initial	121,0±7,2	125,0±2,3	120,0±4,1	118,0±4,3	117,0±5,2	130,0±8,2	120,5±7,9	122,0±4,7	126,0±2,4	117,0±3,3	119,0±3,4	123,0±4,7
After injection of drug	—	—	182,0±6,9	183,0±4,0	98,0±5,1	116,0±6,1	63,5±5,0	114,0±3,0	94,0±3,6	115,0±2,3	75,5±7,6	117,0±3,4
Phase of excitation	140,0±3,4	153,0±4,2	169,0±7,3	174,0±10,0	154,0±6,1	130,0±7,1	80,5±3,5	122,0±1,8	95,4±4,1	124,5±4,3	111,5±7,1	124,0±3,4
Phase of inhibition	52,0±4,3	51,0±3,4	50,0±4,1	41,0±1,7	23,5±4,7	28,0±5,8	43,0±3,5	63,5±4,3	54,0±3,8	67,5±6,3	57,0±5,8	52,0±4,6
Period of stabilization	73,0±3,2	—	—	72,0±7,9	62,0±4,2	50,4±6,3	59,5±3,9	74,5±2,7	77,0±4,1	93,0±5,1	72,0±9,2	62,5±7,6
Pulse rate (beats per minute):												
Initial	250,0±9,2	240,0±11,0	260,0±11,0	261,0±11,3	270,0±11,7	242,0±13,0	242,0±6,6	243,0±7,9	262,0±6,3	253,0±8,3	256,0±10,2	272,0±10,9
After injection of drug	—	—	307,0±13,3	275,0±10,8	264,0±13,0	240,0±5,7	257,0±11,4	241,0±7,2	266,0±10,1	241,0±7,2	256,0±9,2	268,0±11,2
Phase of inhibition	230,0±10,0	240,0±15,0	227,0±15,0	235,0±13,6	223,0±12,7	229,0±9,1	244,0±8,4	229,0±12,9	244,0±14,0	213,0±7,2	245,0±11,7	235,0±5,3
Period of stabilization	240,0±14,0	—	—	247,0±17,4	193,0±20,3	230,0±8,5	233,0±9,1	252,0±7,7	248,0±16,9	222,0±8,3	248,0±19,2	271,0±9,5
Respiration rate (frequency per minute):												
Initial	76,0±5,0	82,0±8,1	66,8±9,0	56,0±5,5	74,8±13,5	92,3±12,0	90,0±15,7	48,0±4,8	90,0±5,4	70,0±8,7	80,0±10,4	109,0±20,0
After injection of drug	—	—	70,0±10,3	64,0±7,5	71,0±7,7	72,0±7,9	61,8±6,4	53,5±3,2	57,2±4,4	68,2±3,8	82,7±11,7	86,6±15,5
Phase of inhibition	66,0±3,0	39,0±6,1	73,0±13,6	89,6±11,9	71,0±6,4	72,0±7,4	75,2±10,3	83,8±5,5	61,0±3,3	68,3±3,2	57,0±6,6	98,0±15,7
Period of stabilization	66,0±3,2	—	—	91,0±9,4	82,3±4,2	83,2±10,0	59,2±3,8	86,4±5,8	62,2±5,5	64,0±5,4	80,0±15,5	88,0±8,9
Rectal temperature:												
Initial	38,0±0,5	38,1±0,3	38,2±0,1	38,5±0,1	38,5±0,2	38,5±0,1	38,1±10,01	38,2±0,1	37,7±0,3	38,7±0,1	38,1±0,1	38,2±0,4
At end of experiment	34,0±0,4	34,7±0,4	34,1±0,6	33,3±1,1	34,3±0,7	34,3±0,7	32,8±0,7	33,6±0,5	32,4±0,4	30,6±1,1	34,7±0,8	34,7±0,5
Temperature of muscles:												
Initial	38,2±0,2	38,1±0,1	38,5±0,2	38,4±0,1	39,5±0,1	39,1±0,2	38,4±0,6	38,8±0,2	38,5±0,1	39,3±0,1	38,4±0,6	38,2±0,4
At end of experiment	32,8±0,4	36,4±0,3	34,6±1,3	32,7±1,1	34,9±0,4	34,0±0,6	35,0±1,1	33,8±0,5	32,2±0,8	30,0±1,4	35,0±1,1	35,9±0,6

Note. Values of indices after injection of drug underlined if they differ significantly from initial values, and values of indices after trauma differing significantly from control values ($P < 0.05$) are underlined.

In the second group (20 experiments) a phase of excitation also was observed during application of trauma (254 ± 26 blows). However, the subsequent phase of inhibition changed directly into the phase of collapse without the intervening development of the symptom-complex of shock. These animals died 9 ± 2 min after trauma.

The results of the next series of experiments (Table 1) showed that artificial enhancement of the phase of excitation by preliminary intravenous injection of adrenalin (series I) or noradrenalin (series II) facilitated the onset of primary collapse, and when the phase of shock developed it considerably shortened the period of survival of the animals. Preliminary injection of succinylcholine (series III) led to disappearance of the motor component of the excitation phase. Four rabbits survived, and in the rest the phase of shock was distinguished by preservation of fast waves on the EEG. By contrast with the two preceding series, after injection of succinylcholine no phenomena of collapse were observed. The use of artificial ventilation of the lungs with a mixture of air and oxygen (tracheotomy, DP-1 apparatus) did not play any significant role, as shown by the results of the experiments of series IV: the use of artificial ventilation of the lungs without administration of succinylcholine had no effect on the dynamics of the posttraumatic response and all the animals died.

Pharmacological blocking of the reticular formation with chlorpromazine (series V) or a decrease in the sensitivity of the angioreceptors induced by intravenous injection of procaine and changes in the function of the nervous system as a result of the central action of the drug (series VI) and narcotic inhibition of the cortex by thiopental (series VII) did not prevent the development of the excitation phase or of a clearly defined phase of torpid shock. However, preliminary injection of these drugs reduced the risk of development of irreversible changes, the basis of the late period of shock. This is shown by the significantly higher survival rate of the animals (7, 6, and 6 rabbits respectively in the last 3 series).

Elimination of the pain factor by preliminary infiltration of the soft tissues of the thigh with 0.25% procaine solution (80-120 ml) before trauma (series VII) did not significantly affect the outcome of the posttraumatic response, although it was accompanied by a less marked degree of arterial hypotonia. Depression of the sympathetic and parasympathetic ganglia by azamethonium bromide (series IX) led in most animals to the development of primary traumatic collapse. Finally, inhibition of the vagus reflexes by atropine (series X) significantly prolonged the survival of the animals.

The dynamics of the general response of the organism to severe mechanical trauma thus depends on the initial functional state of the vital regions of the nervous system.

LITERATURE CITED

1. É. A. Asratyan, Essays on the Etiology, Pathology, and Treatment of Traumatic Shock [in Russian] Moscow (1945).
2. I. K. Akhunbaev and G. L. Frenkel', Essays on Shock and Collapse [in Russian], Frunze (1967).
3. S. I. Banaitis, Traumatic shock in Experimental, Clinical, and Field Surgical Practice [in Russian], Kaunas (1948).
4. A. N. Gordienko, The Fundamental Mechanism of Development of Traumatic Shock [in Russian], Kiev (1956).
5. S. M. Dionesov, Pain and Its Effect on the Human and Animal Organism [in Russian], Moscow (1963).
6. V. K. Kulagin, Vestn. Khir., No. 5, 68 (1970).
7. I. R. Petrov, Traumatic Shock [in Russian], Moscow (1962).