by botulinus toxin, with an implanted nerve was studied. As Fig. 1 shows, after implantation of the nerve on the affected side restoration of the normal weight of muscle tissue took place more rapidly than in the previous series of experiments, and marked hypertrophy of the "poisoned" muscles was found after 100 days. Recovery of weight was regularly associated with a considerable increase in CST of the implanted nerve (Fig. 2), in the virtual absence of any significant restoration of transmission of excitation from the "poisoned" proper nerve of the muscle. A similar phenomenon was observed against the background of rapid normalization of RMP of the muscle fibers in the zone of injury on account of an increase in the number chiefly of highly polarized fibers of the reinnervated muscles compared with the control (Table 1).

As regards the phenomenon of reinnervation of the healthy muscle, implantation of the nerve into intact muscles led to slight hypertrophy (Fig. 1) of the muscles but before 45 days it had virtually no effect on the level of polarization of the muscle fibers (Table 1) and was not accompanied by the formation of functioning synapses: even in the late stages after implantation, CST did not reach significant values (Fig. 2).

It can be concluded from the experimental results that growth of additional fibrils in the zone of paralysis due to botulism may be connected with the formation of substances stimulating regeneration of nerve fibers. The synaptic apparatuses damaged by botulinus toxin, do not themselves inhibit the formation of new synapses. However, the formation of completely normal myoneural synapses extends over many months because of profound disturbances of the functions of the trophic centers of the skeletal muscles, i.e., spinal motoneurons [1, 2].

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RESPONSES OF THE SYSTEMIC CIRCULATION IN THE EARLY PERIOD OF TRAUMATIC SHOCK INDUCED BY CANNON'S METHOD

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Experiments on dogs showed that the erectile phase of shock induced by Cannon's method can be divided into two periods on the basis of changes in parameters of the systemic circulation. The first period has many features in common with "defensive reactions" in response to the appearance of danger and the action of powerful nonspecific stimuli and is characterized by an increase in the return of blood to the heart, an increase in cardiac output, and a decrease in peripheral vascular tone. The second period is characterized by commencing decompensation of functions of the cardiovascular system. The second period ends with the development of the torpid phase of shock.

KEY WORDS: cardiac output; stroke volume of the heart; total peripheral vascular resistance.

Great importance in the system of disturbances of function in shock is attached to circulatory disorders. However, in most investigations changes in the systemic hemodynamics, regional blood flow, and microcirculation have been studied under conditions of marked posttraumatic hypotension when, strictly speaking, the shock process has become established and the primary neuroendocrine and hemodynamic responses have taken effect [2, 8].

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TABLE 1. Changes in Principal Indices of Systemic Circulation in Dogs during Development of Traumatic Shock induced by Cannon's Method (M ± m)

Index	Initial state	Period of trauma		Torpid phase of shock		
		maximal rise of BP	beginning of fall of BP	BP = 60 mm Hg	BP = 40-55 mm Hg	BP = 10-20 Hg
Arterial pressure, mm Hg	127±3	207±23*	150±12†	67±3†	46±2†	16±2†
Cardiac frequency, beats/min	117±8	113±8*	192±12†	215±9*	212±13*	198±19*
Central venous pressure, cm water	67±7	240±16*	77±16†	8±7†	6±8*	16±7*
Cardiac output, ml/min/kg body wt.	326±33	528±37*	238±20†	146±17†	123±23†	80±1†
Stroke volume of heart, ml/kg body wt.	3,4±0,39	5,2±0,51*	1,4±0,16†	0,8±0,10†	0,6±0,12*	0,5±0,09*
Total peripheral vascular resistance, dynes · sec · cm^{-5}	36375±3321	23881±2373*	43828±3076†	44478±4087†	39287±5136†	19739±2482†
No. of observations	26	26	25	21	14	11

Legend. *Results differing significantly ($P < 0.01$) from initial, †results differing significantly from initial values and from those of the previous stage of the investigation.

In the present investigation, in experiments on 28 male dogs early responses of the systemic circulation in the dynamics of development of traumatic shock induced by Cannon's method were studied.

EXPERIMENTAL METHOD

Traumatic shock was induced by Cannon's method by multiple trauma to the soft tissues of the animals' thigh until the arterial blood pressure (BP) had fallen to a steady 60 mm Hg.

The following indices were studied: BP in the left femoral artery by means of a mercury manometer, the central venous pressure (CVP) by means of a Waldman's manometer and polyethylene catheter, introduced through the right external jugular vein into the right atrium, the cardiac output (CO) by a thermodilution method, the cardiac frequency (CF), the stroke volume of the heart (SV) and the total peripheral vascular resistance (TPVR). The thermodilution curve was recorded on tape by an ÉPP-09M electronic potentiometer. The sensitive element of the potentiometer was an MT-54 thermistor, mounted in a special catheter passed through the left common carotid artery into the arch of the aorta. Physiological saline, cooled to room temperature, injected in a volume of 5 ml into the right atrium, was used as indicator. The values of CO, SV, and TPVR were calculated by the usual method [4]. The animals were prepared for the experiment under local anesthesia with 0.5% procaine solution.

The indices studied were recorded at the following periods: initial (after fixation and preparation of the animals for the experiment; the period of trauma; initially, when BP had risen to its highest values, and later, when it had started to fall, but was still sufficiently close to its initial level; after the development of hypotension, when BP fell successively to 60, 40, and 20 mm Hg. The results were subjected to statistical analysis by the usual method [5].

EXPERIMENTAL RESULTS

As Table 1 shows, the initial response of the systemic circulation of the animals to trauma was a rise in BP, an increase in SV and CO (by 53 and 62% respectively), and a decrease in TPVR by 34% ($P < 0.01$). Meanwhile, a marked increase was observed in CVP ($P < 0.01$) and a decrease in CF.

When BP, after an initial rise, began to fall as a result of the continued trauma, a decrease in CBP, SV, and CO (by 31, 59, and 27% respectively compared with initially, and an increase in CF and TPVR (by 64 and 20% respectively, compared with initially, were observed. Later, during trauma and during development of the torpid phase of shock, the changes described above increased in severity. When BP had fallen to 20-10 mm Hg a sharp decrease in TPVR was found ($P < 0.001$).

As a result of this investigation, a number of new facts reflecting the dynamics of development of the initial stage of traumatic shock induced by Cannon's method were obtained, which had not previously been described by workers studying shock and which call for further detailed analysis. As a first step, however, on the basis of the results so far obtained and of data in the literature, the following suggestions can be made.

Following generalized excitation of the nervous system and activation of its autonomic components, mechanisms of nonspecific defence of the organism are stimulated at the beginning of trauma. An important role among them is played by mobilization of blood stored in depots and an increase in the circulating blood volume, an increase in tone of the capacity vessels, dilatation of the resistive vessels of the skeletal muscles, and stimulation of adrenergic fibers innervating virtually all other regions of the cardiovascular system [3]. Against the background of a considerably increased CO, which was observed at the beginning of trauma, the marked decrease in TPVR was evidently due to a systemic depressor reflex as a result of excitation of vascular β-adrenoreceptors of the skeletal muscles and an increase in tone of the muscular sympathetic vasodilators. Similar responses of the systemic circulation have been described to the action of powerful nonspecific stimuli and also to electrical stimulation of somatic nerves [6, 7].

During continued trauma the venous return to the heart is sharply limited, because of a decrease in the circulating blood volume resulting from its pathological retention in the depots and local loss of blood and plasma [1]. In this period of development of shock BP and CO remain high only because of tachycardia and maximal strain on peripheral vascular tone. Later, as the deficit of the circulating blood volume increases and the venous return to the heat continues to fall, the mechanisms of stabilization of BP and CO described above are no longer effective and profound hypotension develops progressively with development of the torpid phase of shock.

Two periods can thus be distinguished in the erectile phase of shock, induced by Cannon's method, on the basis of changes in the systemic circulation. The first period has much in common with "defensive reactions" associated with the onset of danger and exposure to powerful nonspecific stimuli. The second period is characterized by the beginning of decompensation of functions of the cardiovascular system and it ends with the development of the torpid phase of shock.

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