

SOME PATHOPHYSIOLOGICAL CHANGES IN THE
ACUTE PERIOD OF THE PROLONGED CRUSH
SYNDROME OF THE LIMBS

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Experiments on animals showed that in the acute period of the crush syndrome of the limbs two phases can be distinguished: 1) a phase of a transient increase in excitability of the vasomotor center, elevation of the blood pressure and dilatation of the blood vessels of the head; 2) a phase of gradual or catastrophic decrease in all indices studied.

Changes in protein metabolism, the peripheral blood, and hematopoiesis in the compression phase of the crush syndrome have been investigated sporadically [7-9].

The object of the investigation described below was to study changes in the general hemodynamics, the functional state of the vasomotor center, the cerebral circulation, and some peripheral blood indices in the acute period of the prolonged crush syndrome of the limbs.

EXPERIMENTAL

The experiments were carried out on 36 adult unanesthetized dogs of both sexes weighing 6.5-21.5 kg and on 33 rabbits weighing 3.5-4 kg.

For the first 1-3 weeks the animals were accustomed to the experimental situation. The following parameters were monitored during this period: respiration, blood pressure in the caudal artery by methods suggested by the writers [2, 3], peripheral blood count, body and skin temperature. The dogs were then prepared for the main experiment: under local anesthesia (0.5% procaine solution) the common carotid or femoral artery and vein were dissected. The two ends of the carotid artery were connected to the system of a type ÉMG two-channel electrical manometer filled with heparin solution (1:3) and recordings were obtained of the arterial pressure, respiration, and ECG (lead II).

The excitability of the vasomotor center was assessed by the response to electrical stimulation of the femoral nerve at threshold strength and at different frequencies (10, 20, and 40 Hz) and to intravenous injection of lobeline (0.015 mg/kg, 1:500 solution). The velocity of the blood flow was determined by the lobeline method. The vascular tone of the circle of Willis was studied by Hurthle's method [10] modified by Avrorov [1]. After the indices had been recorded in the initial state (5-6 recordings for 20-30 min) one of the hind limbs was crushed with a press for 4 h with a load of 1000 kg. The indices were recorded at the moment of crushing and then continuously for 10-20 min; later records were obtained every 15 min in the compression period and for 2-4 h after removal of the press. The peripheral blood was tested in the usual way every hour after application and removal of the press.

EXPERIMENTAL RESULTS AND DISCUSSION

Crushing was accompanied by marked excitation of the animals and an attempt to free themselves from the press. The rapid motor response in the dogs was accompanied by howling and yelping, and sometimes by involuntary micturition and defecation. The signs of excitation gradually subsided, and after 1-2 h

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most of the dogs exhibited a weak defensive reaction; some gave no such response, but in others there was a progressively increasing general inhibition and no response was given to external stimuli 30–45 min after application of the press.

In the initial state (before crushing) the mean pressure in the central end of the carotid artery was 143 ± 2.4 mm, and in the peripheral end it was 98 ± 3.9 mm. The ratio between the pressure in the peripheral (M_1) and central (M) ends of the carotid artery (M_1/M) varied in the initial state from 0.60 to 0.78 (mean 0.66 ± 0.25), in agreement with results published by other workers [1, 4, 5].

At the moment of crushing the arterial pressure in the central end of the carotid artery in all experiments was increased to a mean value of 200 ± 4.7 mm ($P < 0.1$), a statistically significant change when analyzed by Peters' method [6]. The pressure in the vessels of the circle of Willis at this time was increased by 10–19.3% of its initial level, but the difference in pressure was not statistically significant ($P = 11\%$). The ratio M_1/M was increased in two experiments, and the pressure rose sharply (by 42.8–72.2% over its initial level), and this, according to Hurthle et al., is evidence of vasoconstriction of the circle of Willis; in the other seven animals the ratio M_1/M fell below its initial level (dilatation of the cerebral vessels).

The circulation time from the femoral vein to the carotid zone was reduced (on the average by 2.7 sec) in 7 of the 11 dogs 5–10 min after crushing the limb. In the other animals it remained at its initial level or fell very slightly.

During the first 10 min after application of the press, electrical stimulation of the femoral nerve and injection of lobeline into the femoral vein evoked a pressure response in 8 of the 11 animals, which was 10–15 mm higher than its initial level. The latent period of the response and the duration of the aftereffect of the stimuli also were reduced. In the other three dogs no increase in excitability of the vasomotor center was found and these animals died 60–85 min after trauma with signs of a rapidly progressive hypotension.

During the 30 min to 2 h after application of the press the systemic arterial pressure of most animals fell to its original level. Meanwhile the character of the pressure curve changed. In six dogs, besides waves of the third order on the blood pressure curve, at equal time intervals (40–50 sec) slow waves (4–6/min) appeared and occupied the period of several (5 or 6) respiratory waves (Mayer's waves). It is important to emphasize that Traube–Hering and Mayer's waves appeared when the arterial pressure was relatively normal and the state of the heart perfectly satisfactory. The appearance of these waves was a poor prognostic sign during the acute period of limb crushing (5 of 6 dogs died during the 1.5–2 h after application of the press).

The fall in pressure in the vessels of the circle of Willis took place parallel to the decrease in systemic arterial pressure, but it took place more rapidly: the pressure had reached its initial level 10–60 min after application of the press. At about the same time the excitability of the vasomotor center fell to its initial level: the pressor response to stimulation did not exceed the initial values and showed a tendency to decrease further. The circulation time also reached its initial level.

Immediately after the transient increase and return to normal of the indices studied there followed a phase of gradual or catastrophic decrease. The sudden and considerable fall of blood pressure in the central end of the carotid artery was observed in experiments in which the hypertensive phase in response to limb crushing was well marked.

Despite the decrease in excitability of the vasomotor center, the law of "relative strength" applied until the systemic arterial pressure fell to 50–30 mm. Phasic changes of paralytic inhibition of the vascular unconditioned reflexes could then be observed: in most animals neither weak nor strong electrical stimulation of the femoral nerve evoked a response of the arterial pressure and respiration. Besides a decrease in the excitability of the vasomotor center, it developed inertia: application of the stimuli evoked a slow increase and decrease of arterial pressure.

Within a few minutes after application of the press and throughout the rest of the experiment the peripheral blood count showed a considerable increase in the number of red and white cells and the hemoglobin concentration. After 1 h the reticulocyte count was increased, erythroblasts appeared, and catalase activity was also increased. The increase in the reticulocyte and erythroblast count in the blood and the

increase in its catalase activity indicate that besides redistribution of the blood, stimulation of medullary hematopoiesis plays an important role in the changes in composition of the blood.

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