REACTIVITY OF THE CARDIOVASCULAR SYSTEM IN DOGS IN TRAUMATIC SHOCK AFTER PROLONGED HYPODYNAMIA

B. R. Yaremenko

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After hypodynamia lasting 14 and 28 days, the resistance of dogs to shock-producing trauma is lowered, and traumatic shock develops faster and irreversibly. In dogs whose movements were limited for 28 days, resistance to trauma itself was lowered. In order to restore the arterial pressure in the final stage of traumatic shock in animals exposed to hypodynamia, much larger volumes of blood substitutes had to be infused.

Prolonged hypodynamia considerably alters the reactivity of the body and lowers its resistance to pathogenic agents [1, 2]. Among the disorders observed in hypodynamia, particular significance is attached to the development of decompensation of the cardiovascular system, weakening of cholinergic influences on that system, and general bodily asthenia [1].

Since persons kept for long periods in a state of restricted movement can be subjected to severe mechanical trauma, an experimental investigation of the character of reactivity of the cardiovascular system in these conditions was attempted.

EXPERIMENTAL METHOD

Experiments were carried out on 31 male dogs weighing 13–24 kg. Hypodynamia was induced by keeping the dogs in special frames which severely restricted their movements throughout the experiment. Previous investigations showed that during enforced limitation of movement the course of hypodynamia was characterized by marked motor excitation of the animals for the first 1 or 2 days; this then subsided, the body weight progressively fell, and the animal developed muscular weakness, increasing hypertension, and activation of the glucocorticoid function of the adrenal cortex. The physiological changes observed in this type of restricted mobility, particularly in its early period, depend not only on the hypodynamia itself, but also on the developing manifestations of emotional and physiological stress [5].

Of the total number of dogs taking part in the experiment, 10 (series I) were kept under the conditions of hypodynamia for 14 days, and 8 (series II) under the same conditions for 28 days. After the animals had been removed from the frame they were fixed to the experimental bench and shock-producing trauma was inflicted by crushing the soft tissues of the thigh until the defensive response ceased, the animal's general condition became severely disturbed, and the blood pressure fell to 60–65 mm. Shock was produced in the same way in 13 intact dogs (series III, control). Respiration and arterial pressure were recorded on a kymograph. The magnitude of the carotid sinus pressor reflexes in response to compression of the common carotid artery for 5 and 10 sec was determined. After infliction of the trauma, the animals under observation for 6 h, during which time the duration of their survival and the effectiveness of administration of Petrov's blood substitute [4] from a high reservoir under a pressure of 100 mm Hg, by Kulagin's method [3], were noted. The dogs were all treated simultaneously in all the series of experiments, and artificial respiration was started as soon as a state of agony developed, when spontaneous

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TABLE 1. Effect of Mechanical Trauma and Prolonged Hypodynamia on Character of Carotid Sinus Reflexes in Dogs

Series of experiments	Initial state			Torpid phase of shock (5 min after trauma)			5 min after treatment		
	total number of animals	normal	phasic state	total number of animals	normal	phasic state and absence of reflexes	total number of animals	normal	phasic state and absence of reflexes
Control (III)	13	10	3	13	–	13 P < 0.025	10	8	2
After hypodynamia for 14 days (I)	10	7	3	10	–	10 P < 0.025	7	1	6 P_1 < 0.025
After hypodynamia for 28 days (II)	8	6	3	8	–	8 P < 0.025	6	1	5 P_1 < 0.025

Note. P – probability that differences between individual series of experiments are significant; P_1 – probability that differences between individual stages of investigation in control experiments and experiments with hypodynamia are significant. Statistical analysis by Fisher's method.

breathing ceased, the corneal reflex disappeared, and the arterial pressure fell to 0-10 mm Hg. At the same time, the reservoir system was connected to the right femoral artery. After the resumption of spontaneous breathing and elevation of the arterial pressure to 90-100 mm Hg, the flow of fluids in the reservoir was automatically stopped. The reservoir was disconnected 1 min later.

EXPERIMENTAL RESULTS

The mean level of the arterial pressure in the dogs exposed to hypodynamia was slightly higher (series I, 166±5.4 mm Hg, $P < 0.05$; series II; 153±4.2 mm Hg) than in the control (148±4.5 mm), evidently on account of the development of emotional and functional stress [1], while the respiration rate (28±4) was considerably lower ($P < 0.05$) after hypodynamia for 28 days (series II) than in the other series of experiments (control, 54±9; series I, 66±14).

After trauma, the transient excitation which the animal developed was followed by a serious general condition, the carotid sinus pressor reflexes disappeared or became phasic in character, the arterial pressure fell, marked tachycardia was observed, and in most cases the respiration rate was slowed. As a rule, third-order waves appeared on the arterial pressure curve after trauma; as the shock increased these waves became deeper, and 30-40 min before the animals' death they disappeared.

The results of these experiments showed that prolonged hypodynamia considerably modifies the body's response to mechanical trauma severe enough to cause shock. For instance, the mean number of blows necessary to produce shock in the dogs after hypodynamia for 28 days was 87±9 (M±m), whereas the control animals required 140±16 blows, and dogs exposed to hypodynamia for 14 days required 175±24 blows. In addition, in the animals exposed to hypodynamia, after trauma there was no definite tendency for the arterial pressure to rise, the general depression increased progressively, and as a rule the animals died 60-90 min later. By contrast, in most of the control dogs a long period of relative compensation was observed, and the period of survival in a state of shock (197±30 min) was significantly longer than in dogs exposed to hypodynamia (68±18 min after 14 days; 86±15 min after 28 days; $P < 0.01$). Two dogs in the control series recovered from shock spontaneously, but this was never observed in the series of experiments with hypodynamia.

In the dogs after hypodynamia, more profound disturbances of reflex regulation of the circulation from the carotid sinus zone than in the control series were found (Table 1).

To raise and stabilize the arterial pressure of the dogs in the control series of experiments, a much smaller volume of blood substitute had to be injected than in the series of experiments with hypodynamia (control 29.2±3.0 ml/kg, after 14 days hypodynamia 43.4±4.8 ml/kg, after 28 days 45.4±5.0 ml/kg). The mean survival of the dogs (in min) after treatment in the various series of experiments was: control 109±2, series I 42±11, series II 38 ± 13.

Prolonged limitation of the animals' movements thus reduced their resistance to the sequelae of shock-producing trauma.

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