SOME PROBLEMS CONCERNING THE REFLEX REGULATION
OF THE VASCULAR TONUS IN ANAPHYLACTIC SHOCK

COMMUNICATION II. CHANGES OF THE DEPRESSOR REFLEXES DURING
THE LOWERING OF THE ARTERIAL BLOOD PRESSURE CAUSED
BY ANAPHYLACTIC SHOCK

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In a previous communication [9] we had shown that vascular pressor reflexes are not only restored during
the lowering of the arterial blood pressure during anaphylactic shock but become even stronger compared to the
normal level and we also showed that these changes in the pressor reflexes cannot be explained with the develop-
ment of a state of parabiosis in the vasomotor center.

These findings suggest that in anaphylactic shock of moderate severity the capacity of the vasomotor center
to respond to pressor stimuli was restored or even increases.

It was the aim of the present paper, to study the changes of vascular depressor reflexes under similar con-
ditions: the reflexes were investigated in the beginning period of the lowering of the arterial blood pressure
caused by injection of the shock dose of the serum as well as during the restoration of the arterial blood pressure.

METHOD OF EXPERIMENTS

The experiments were carried out on 28 rabbits under nembutal narcosis. During the sensitization of the
animals the injection of the shock dose of serum as well as the recording of the arterial blood pressure and the
respiration were carried out by the method described in the previous communication.

The vascular depressor reflexes were produced by electrical stimulation of rectangular impulses in the a-
ortic nerve or by stimulation of the pulmonary mechanoreceptors produced by increasing the pressure of air within
the lungs.

The afferent impulses from the aortic nerve were taken off by means of a loop oscillograph (MPO-2) with
an alternating current amplifier and recorded on a cinematographic film. Simultaneously, the arterial blood
pressure and the respiration were recorded on the same film.
EXPERIMENTAL RESULTS

Under normal conditions the strength of the depressor response after stimulation of the aortic nerve reached on the average 30-35 mm Hg. Intravenous injection of the shock dose of serum led to a marked fall in the blood pressure, the level of which reached by the 5th-10th minute 40-50 mm Hg. By that time in all experiments without exception a marked suppression of the depressor response could be observed, the strength of which reached on the average 5-11 mm Hg (Fig. 1). Later parallel to the restoration of the general blood pressure the depressor response also began gradually to gain in strength. But even by the 20th minute after the injection of the shock dose of serum when the level of the general blood pressure had reached 60-70 mm Hg, the strength of the depressor response was much lower than under normal conditions. And what is more, in the majority of experiments these reflexes did not regain their normal strength even by the 30th-40th minute. Similar changes in the depressor reflexes could be observed also in the experiments in which the pulmonary mechanoreceptors were stimulated. It must be emphasized that by that time the pressor reflexes had not only been restored but had even become stronger than the reflexes observed under normal conditions.

The data quoted above suggest that the capacity of the vasomotor center to respond to the depressor stimuli is much lower under conditions of anaphylactic shock than under normal conditions.

We were unable to find publications concerning the state of the depressor reflexes in anaphylactic shock. Investigations concerning other types of shock suggest that the majority of authors is inclined to believe that changes of the depressor reflexes under these conditions are caused by development of a state of parabiosis in the vasomotor center. For example, V. K. Sel'tser [7] observed, that simultaneously with the development of the state of shock the so-called "rule of strength" is disturbed and in many cases a narcotic levelling, and ultraparadoxical phase can be observed in the changes of the depressor reflexes.

To obtain a paradoxical response from the arterial blood pressure and above all the use of an intensive stimulus is required. For example, it is known that in this manner V. E. Delov and V. I. Filistovich [3], and V. I. Filistovich [8] caused a pessimal state in the depressor mechanisms.

Can it be assumed, however, that the changes in the depressor reflexes, described above, are connected with extreme stimulation of the depressor mechanisms strong enough to produce a state of parabiosis in the central part of the reflex arch of the depressor reflexes? A stimulation of the depressor mechanisms may well take place during the intravenous injection of the shock dose of serum as well as during the erectile phase of the shock when a temporary increase in the arterial blood pressure can be observed, a fact which will increase the stimulation of the baroreceptors in the region of the carotid sinus and the aortic region. Some experiments, however, suggest that even if such stimulation actually takes place its intensity would be so insignificant that it could never produce a pessimal state in the central part of the reflex arch of the depressor reflexes. The kymogram (Fig. 2) shows that additional stimulation of the intact aortic nerve during the erectile phase of the shock at the time when the baroreceptors undergo temporary stimulation by the increase in the arterial blood pressure, even without additional stimulation, causes a depressor response of similar strength as those observed under

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Fig. 1. Vascular depressor reflex in response to stimulation of the aortic nerve in a rabbit. a) Under normal conditions; b, c) 10 and 20 minutes after administration of the shock dose of serum respectively. Significance of the curves: (from top to bottom): respiration; arterial blood pressure; mark denoting the moment of stimulation; time mark (1 sec).

Fig. 2. Vascular depressor reflex in response to the stimulation of the aortic nerve under normal conditions (a) and during the erectile phase of shock development (b). Key as in Fig. 1.