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 development 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 conditions: 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 aortic 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.

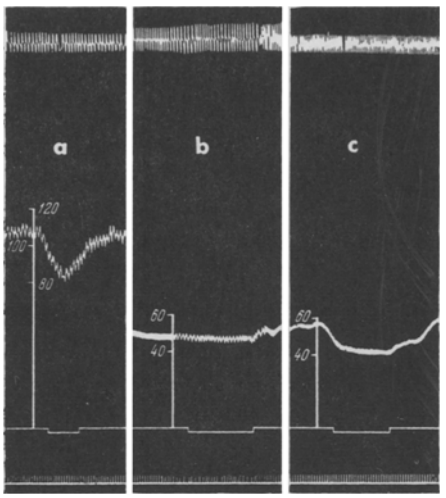

Fig. 1. Vascular depressor reflex in response to stimulation of the aortic nerve in a rabbit. a) Under normal conditions; b, c) 10 and 20 minutes after administration of the shock dose of serum respectively. Significance of the curves: (from top to bottom): respiration; arterial blood pressure; mark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

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.

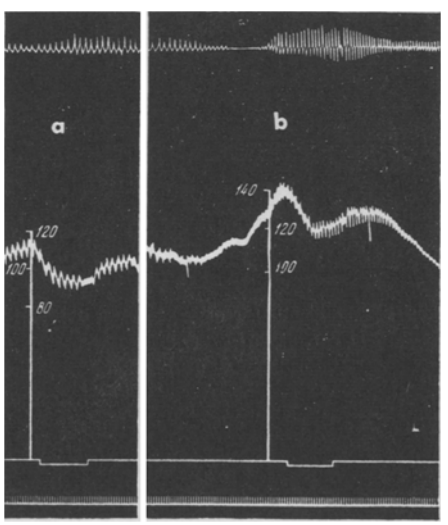

Fig. 2. Vascular depressor reflex in response to the stimulation of the aortic nerve under normal conditions (a) and during the erectile phase of shock development (b). Key as in Fig. 1.

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

normal conditions. To this purpose we investigated with the aid of a loop oscillograph the afferent impulses passing through the aortic nerve under normal conditions at the time of intravenous injection of the shock dose of serum and during the development of the shock. It appeared that the injection of the shock dose of serum has almost no influence upon the intensity of the afferent impulses passing through the depressor nerve (Fig. 3). A slight increase in these impulses could be observed immediately after the injection of the serum, but this increase was to a large extent caused not so much by the action of the shock dose of serum as rather by the increase in the arterial blood pressure caused by the development of the erectile phase of the shock.

No appreciable intensification of the afferent impulses could be observed in the aortic nerve during the state of shock. On the contrary, the electrogram (Fig. 3, c) shows that the intensity of the afferent impulses decreases correspondingly to the decrease in the general arterial blood pressure.

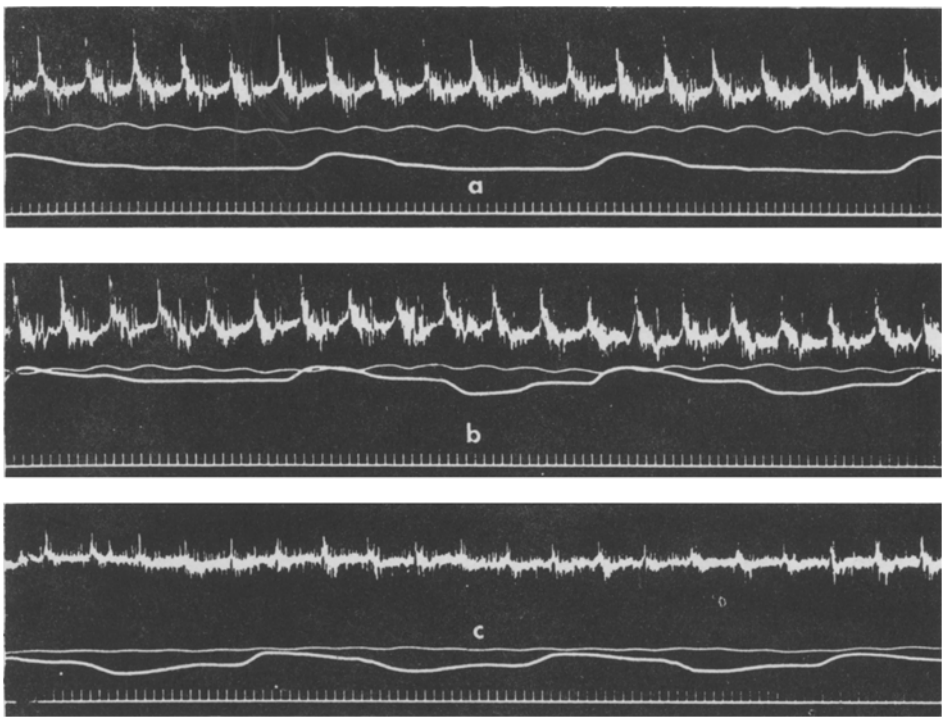

Fig. 3. Rhythmically recurring afferent impulses in the depressor nerve of a rabbit. a) Under normal conditions; b) towards the end of the intravenous injection of the shock dose of serum; c) correspondingly in the 15th minute of hypotension. Significance of the curves (from top to bottom): afferent impulses in the depressor nerve; arterial blood pressure; breathing; time mark (1/20 secs), zero level of the arterial blood pressure.

The appearance of oscillations of small amplitude in the interval between the individual discharges is probably due to the stimulation of the chemoreceptors in the aortic arch consequent to the hypoxia produced by the fall in the arterial blood pressure.

It thus appears that although the intensity of the afferent depressor impulses increases temporarily during the development of the shock state, this increase is so insignificant that it could under no circumstances produce a pessimal state in the central part of the reflex arch of the depressor reflexes.

It can be assumed that similar changes in the strength of the afferent impulses take place under the conditions described above in the nerves of the carotid sinus. In this context some authors [1, 2] were unable to obtain under normal conditions a pessimal reaction in the arterial blood pressure even after super-strong stimulation of the depressor nerves. On the other hand, reports appeared in the literature suggesting that a distortion of the depressor reflexes can be obtained even by relatively weak stimulation of the depressor mechanisms [10, 11, 12].

The distortion of the depressor reflexes after weak threshold stimulation of the aortic nerve was observed in the experiments of A. G. Sverdlov [6] under conditions of lower blood pressure produced by traumatic shock.

Finally, we wish to emphasize that both the decrease in the capacity of the vasomotor center to respond to depressor stimuli observed under conditions of shock on the one hand and the fall in the strength of the afferent impulses in the depressor nerve on the other hand have a favorable influence upon the rapid restoration of the arterial blood pressure. And, indeed, as I. R. Petrov [5] remarked: "In what state would be the animal body if any stimulation of the depressor mechanisms would lead to a fall in the arterial blood pressure which is already at a critical level." For this reason, the suppression of the depressor mechanisms under these conditions appears to be to a certain degree of adaptive character.

SUMMARY

Experiments were performed on rabbits and dogs. In conditions of anaphylactic shock of moderate intensity depressor reflexes are markedly inhibited at the beginning of shock-induced arterial hypotension. Later, with the restoration of arterial pressure these reflexes become intensified, but there is no complete normalization. Investigation of the afferent depressor impulsation during shock development, along with that of depressor reflexes against the background of shock (its erectile phase) shows that the mentioned inhibition of depressor reflexes is evidently not caused by the development of the pessimal state to depressor actions in the vasomotor center.

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All abbreviations of periodicals in the above bibliography are letter-by-letter transliterations of the abbreviations as given in the original Russian journal. *Some or all of this periodical literature may well be available in English translation.* A complete list of the cover-to-cover English translations appears at the back of this issue.
