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DYNAMICS OF CHANGE IN RAT ARTERIAL PRESSURE
UNDER CONDITIONS OF IMMOBILIZATION

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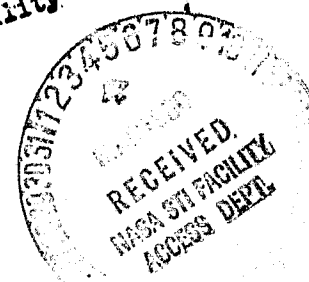
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DYNAMICS OF CHANGE IN RAT ARTERIAL PRESSURE UNDER
CONDITIONS OF IMMOBILIZATION

by

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It was shown that emotional stress accompanied by characteristic peculiarities of the changes in the regulation of arterial pressure and the frequency of cardiac contractions developed under immobilization conditions in rats. A group of adapting rats with a definite resistance to emotional stress, and a group of animals incapable of adapting to acute emotional stress which died with phenomena of cardiovascular insufficiency were revealed among the experimental animals. The mechanisms providing resistance to emotional stress in numerous conflict situations are analyzed.

It has been shown that emotional stress develops under conditions of prolonged continuous negative emotional states accompanied by disorders in behavior and self-regulation of the autonomic functions [1,5,6]. One of the leading autonomic indicators that characterize the development of emotional stress is arterial pressure (AP). In emotional stress the development of stable arterial hypertension is possible [2, 7-10, 14] which is observed, however, not in every animal and man. In this respect in a study of the mechanisms for the formation of emotional stress it is important to understand in what way in some cases individual negative emotional reactions "are accumulated" and in the final analysis create lengthy stable negative emotional excitation accompanied by the somato-autonomic disorders

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*Numbers in margin indicate pagination in original foreign text.

while in other cases thanks to the defense mechanisms a resistance to emotional stress is formed.

In order to reveal the laws governing the predisposition and resistance of animals to emotional stress the given work studied the dynamics of AP regulation and the frequency of cardiac contractions (FCC) in acute immobilization stress in rats.

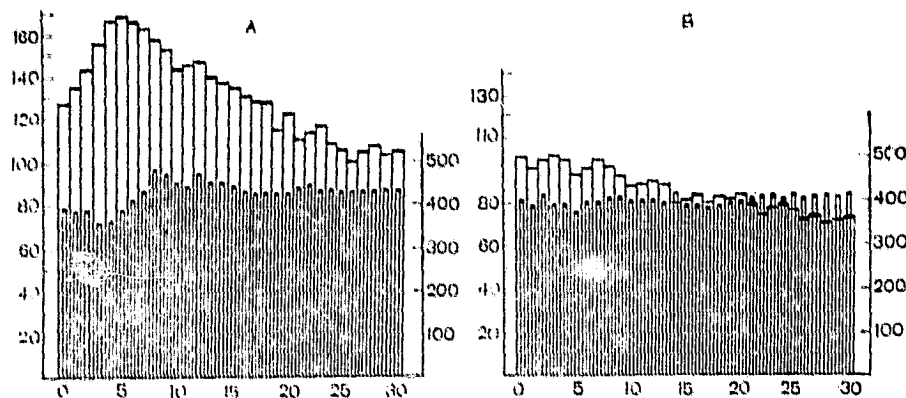
Technique

The experiments were conducted on adult male rats weighing 250 g. A total of 56 experiments were made.

The stress factor used was a single 30-hour immobilization [11, 13, 16] in a close chamber and multiple regulated immobilization for 18 weeks: for 3 h in the first 4 weeks, for 4½ h in the next 5 weeks, and for 8 h in the last 9 weeks. During the experiment the AP was recorded from the technique we developed for chronic measurement of AP in rats under conditions of free behavior [4]. For this purpose a thin catheter was implanted in the rats through the caudal artery into the abdominal aorta. The AP was measured with the help of R-37 pressure sensors (firm "Stetkem", United States), R-23 AA (firm "Galileo", Italy) and was dynamically recorded with slow sweep (2 cm/h) on a N-340 self-recorder of domestic production and with great sweep (10 mm/s) on a mingograph-81 (firm "Yelena", Sweden). The recording was made of the systolic, diastolic and mean amount of AP. The FCC was computed according to the AP recording. From the AP recording with slow sweep an hour-by-hour computation was made of the average hourly amount of AP and FCC.

Results and Discussion

As the control (8 rats) the AP was dynamically recorded in rats under normal conditions of free behavior in cages. Here it was established that the AP oscillations in the animals did not exceed 10-12 mm Hg during the entire time of recording and were mainly linked to the periodic exchange of rest and activity. The initial level of the mean amounts of AP and FCC in all the experimental animals comprised respectively 118.3 ± 1.3 mm Hg and 396 ± 7 contractions per minute.



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Figure 1. Variants (A, B) in Dynamics of Average Hourly Value of AP and FCC in Rats in 30-Hour Immobilization. Here and in fig. 2 and 3: on x-axis--time of immobilization (in hours); on y-axis--left--mean amount of AP (in mm Hg); right--FCC per minute.

The experimental observations of the AP and FCC regulation in immobilization emotional stress made it possible to separate all the animals into several groups. The initial AP and FCC levels in the animals belonging to different groups did not differ statistically.

Rats were placed in the first three groups who were able to adapt to immobilization. In 6 animals in the first group the dynamics of change in AP and FCC during immobilization did not differ significantly from that of the animals in the control group and was characterized by relative stability. However, in the majority of experiments in the indicated conditions changes were recorded in the AP and FCC. Thus, in 12 animals that we had placed in the second group, in the first 5-6 h of immobilization an increase in AP was observed by 20-40 mm Hg with a subsequent gradual decline and stabilization of it by the 23rd-26th hour on the level 20-30 mm Hg lower than the initial (fig. 1, A). In this group of animals in the first 6-8 h of immobilization oscillations in the FCC were noted in limits of 350-450 per minute, in later periods (20-24th hour) the FCC was stabilized on the level 400-450 per minute. Eight rats in the third group reacted to immobilization by a slow, gradual reduction in the AP by 20-35 mm Hg to stabilization by the 22nd-23rd hour on the level 80-100 mm Hg (fig. 1, B).

The FCC in these cases was characterized by relative stability and remained in limits 350-420 per minute for the entire time of immobilization. It is characteristic that not all the experimental animals were able to adapt to the stress situation. Fourteen rats died, apparently, as a consequence of the insufficiency of the adaptive mechanisms. Here the following dynamics of AP and FCC were noted in them, that also made it possible to divide these animals into several groups. In 7 rats placed in the fourth group, in the first 10-16 h of immobilization a gradual increase in AP was observed to 140-160 mm Hg with subsequent progressive drop (fig. 2, A). In the animals in this group sharp rises were observed in the AP (by 20-30 mm Hg) which could be maintained in the space of an hour at a high level. During immobilization in these animals FCC fluctuations were noted in the limits 300-500 per minute with the maximum during the AP rise. The animals of this group died on the background of a sharp progressive drop in FCC. In the several last hours of life the FCC in them dropped from 400 to 100 per minute and lower with a relatively high AP level (75-90 mm Hg). In these rats during autopsy massive sections were noted of myocardial infarct. For three experimental animals of the fifth group from the very beginning of the experiment a progressive drop in the AP and FCC values was characteristic. The death of these animals was observed at the 13th-17th hour of immobilization (fig. 2, B). In this time the AP was gradually reduced from 120 to 40-50 mm Hg by the last hour of the study and was 30 mm Hg 15 minutes before death. The FCC in this time was reduced from 400 to 120 per minute.

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For four rats in the sixth group a sharp increase in the AP by 45-60 mm Hg and FCC to 600 per minute and above was characteristic in the last period before death. These animals died in even earlier periods of immobilization--in the 6th-10th hour of the experiment (fig. 3). It is characteristic that the AP in animals of this group rapidly dropped from 145 to 25 mm Hg for the last several minutes before death. The FCC here all the way to the last 1-2 min. before death remained high--500-550 per minute. During the greatest rise in AP and the greatest FCC value in the animals of the given group rhythmic oscillations were noted in the mean

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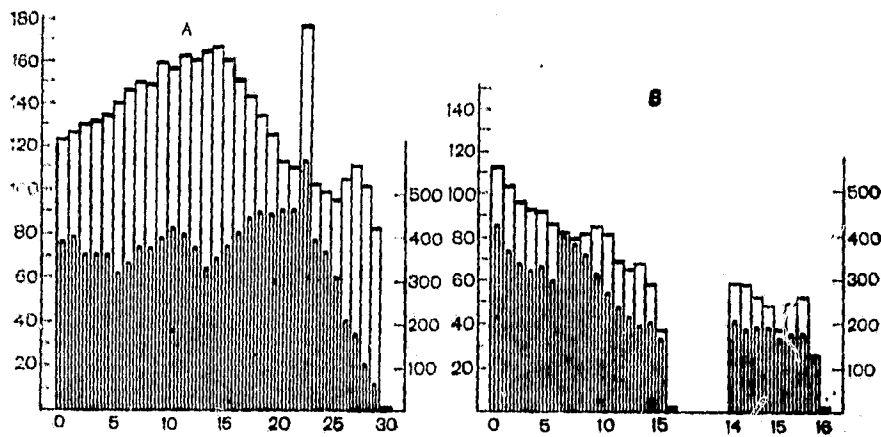


Figure 2. Dynamics of AP and FCC in Different Groups of Rats (A,B) in Immobilization
 (The average amounts of AP and FCC are presented on the graphs for 1 h and for 15 min (column B). The mark at the end of the column--death of animal).

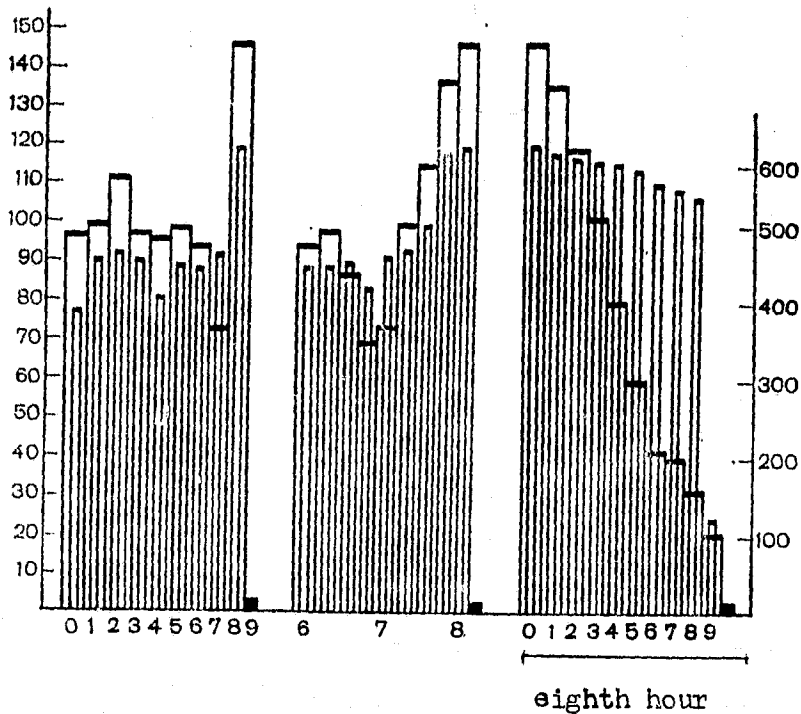


Figure 3. Variant of Dynamics of AP and FCC in Rats in Immobilization
 The averaged amounts of AP and FCC are presented on the graph for 1 h and 15 min, 1 min. The start of the eighth hour is also presented in minutes.

amount of pressure in the limits 30 mm Hg.

The experiments made it possible to find certain forecasting criteria from which one could foresee the death of the animal under conditions of immobilization emotional stress. Thus, it was established that unfavorable factors are periodic oscillations in the mean AP level in limits 15-20 mm Hg with wave duration 15-30 min., sharp increase in AP in the space of an hour by more than 40 mm Hg, progressive decline in FCC for 5-7 h, and gradual drop in AP by 30-40 mm Hg in 2-3 h.

Thus, among all the experimental animals rats were found that possessed a certain resistance to acute immobilization emotional stress, and rats that were predisposed to it who died under these conditions on the background of cardiovascular insufficiency. In this respect the question arises of whether the resistance in the rats predisposed to emotional stress can be increased by repeated immobilization of them under the same experimental conditions.

In a separate series of experiments on 8 rats after early (3-8 h) cessation of the first immobilization in several days a second was conducted. It was found that prolonged 18-week orderly (3-8 h daily at the same time) immobilization resulted in complete adaptation and preservation of the initial amount of AP and FCC in these experimental animals. Thus, in the rats predisposed to emotional stress who in all the forecasting signs in the first immobilization could die revealed an increased resistance in the second immobilization.

The experiments demonstrated that part of the animals could adapt under conditions of acute emotional-negative situation linked to immobilization. The other part, apparently, due to the insufficiency of the adaptation mechanisms died as a result of sharp cardiovascular disorders and the development of myocardial necrosis. The adaptation of the animals to the emotional stress can be increased by repeated short-term immobilizations.

The results of our experiments agree with the data of the literature [11, 12]. With repeated immobilizations of rats a restoration was also

observed [15] in the initial adrenalin level in the cerebral layer of the adrenal gland which is reduced after single immobilization and remains below the normal level more than a day. Only with prolonged negative emotional states induced by irregular immobilization for several months [11, 12], constant zoosocial conflicts [13, 14], as well as the effect of the stress situation [11, 12] do conditions occur for the development of emotional stress with stable hypertension.

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