# CHANGES IN CARDIAC ACTIVITY DURING MECHANICAL STIMULATION OF THE BRAIN

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The brain is frequently exposed to mechanical stimulation (during labor, especially abnormal, during excessive straining, and as a result of trauma). However, the primary changes in the cardiovascular system which develop at the moment of stimulation of the brain have not hitherto been studied in detail.

## EXPERIMENTAL METHOD

Experiments were conducted on five dogs. A device for mechanical stimulation of the brain [2] was implanted into the motor and parieto-occipital regions of the cerebral cortex. The subjacent area of the dura was removed and the stimuli were applied directly to the brain surface (a pressure of 1-6 mm Hg).

The potentials of the heart were recorded by means of needle electrodes implanted beneath the skin of the right fore- and left hind-limbs. This corresponds approximately to lead 2 in man. Analysis of the electrocardiogram consisted of counting the cardiac contractions in each period of 6 sec.

We studied the effect of mechanical stimulation of the brain on the cardiac component of the motor-defensive conditioned reflex. No stereotype was used in the experiments, which were conducted at intervals of from 2 to 6 min. The conditioned stimulus acted alone for a period of 6 sec. To reinforce the conditioned stimulus we selected the current at which the dog reacted simply by withdrawing its paw, so far as possible without a pain reaction, accompanied by a cry.

## EXPERIMENTAL RESULTS

Mechanical stimulation of the brain caused marked and varied changes in the frequency of the cardiac contractions (Fig. 1). In most dogs these took the form of quickening of the heart beat, more noticeable if the initial heart rate was slow and less so if the initial rate was fast. In the animals in which pressure on the brain was applied during tachycardia (110-140 beats per minute), a slight slowing of the rate of the heart was observed (by 7-8%).

A relationship was thus revealed between the reaction and the functional state of the centers, like that formulated generally some time ago by N. E. Vvedenskii [4-6] in relation to the autonomic functions, and subsequently confirmed by many writers [10-14 and others].

Within the range of pressures which we used, their effect on the cardiac activity was practically independent of their magnitude. This was evidently due to the fact that the activity of the cardiovascular center is largely autonomous, and is subjected only to a slight extent to the regulating influence of the higher divisions of the brain, which were stimulated in the experiment.

The reaction of the heart always developed only at the time of mechanical stimulation of the brain. After removal of the pressure from the brain, the rate of the heart beat returned to its initial level within a few seconds. An after effect of such short duration is also found in many other cases, especially when the reaction is expressed by a slowing of the heart rate [11, 18]. This is probably due to the risk of prolonged inhibition of the cardiac activity.

Conditioned-reflex changes in cardiac activity during the formation of a motor-defensive reflex took place in the dogs at the 4th-5th combination. The rapid appearance of the cardiac component of the conditioned reflex during pain reinforcement has previously been reported by various workers [1, 22]. In contrast, however, to the motor component which appeared somewhat later but was at once consolidated, the cardiac component remained unstable for a considerable time. In our experiments it was consolidated only after 100-115 combinations.

As a rule, the conditioned-reflex reaction of the heart took the form of a quickening of its rate, i.e., it duplicated the unconditioned reaction. The extent of the changes in the heart rate was to some extent dependent on the frequency of the beat at the moment of application of the conditioned stimulus (Fig. 2). It will be seen from the graph (curve 1) that when the initial heart rate was 50-60 beats per minute, the rate rose by 20-30 beats (35-50%), and when the initial rate was 120-130 beats per minute it rose by only 10-15 beats per minute (10%).

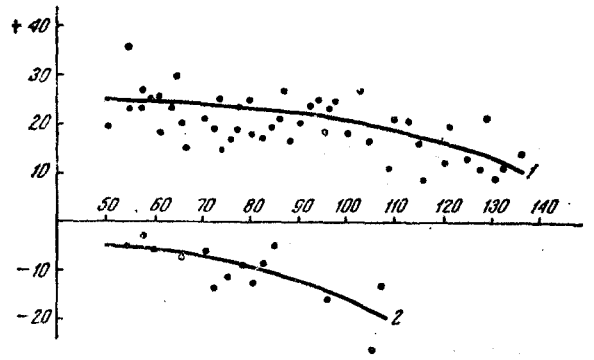


Fig. 1. Changes in the rhythm of the cardiac activity in dogs during mechanical stimulation of the brain in the region of the locomotor centers of the cortex by a pressure of 1-2 mm (1) and 4-6 mm (2). Along the abscissa – initial rate of the heart (beats per minute); along the ordinate – change in the heart rate (in beats per minute).

The changes in the conditioned reflex reactions arising under the influence of mechanical stimulation of the brain (see Fig. 2, curve 2) were not determined by a change in the underlying frequency of the cardiac contractions caused by this stimulation. They were usually qualitative in character. An important factor here was the localization of the stimulated area of the brain. If pressure was applied in the region of the locomotor centers of the cortex, the conditioned-reflex reaction was shown not as a quickening, but as a slowing of the heart beat.

During mechanical stimulation of the brain in the region of the locomotor centers of the cortex, no disinhibition of differentiation was observed. On the contrary, whereas during the application of a differential stimulus, although the motor reflex was constantly absent from the electrocardiogram a slight quickening or slowing of the heart beat was observed, during mechanical stimulation of the brain the heart as a rule failed to react to the differential stimulus. An increase in the pressure to 6 mm Hg had no significant effect on the character of these changes.

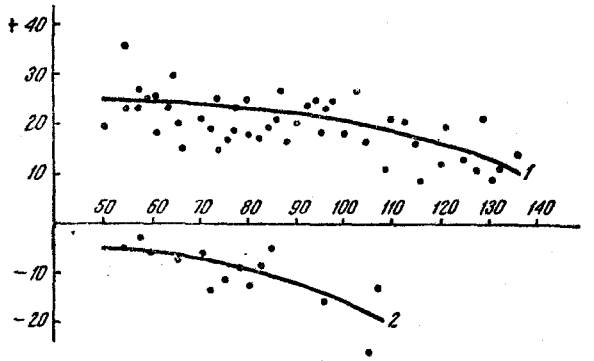


Fig. 2. Magnitude of the cardiac component of the conditioned-reflex reaction before mechanical stimulation of the brain (1) and during stimulation by a pressure of 1-2 mm Hg (2). Significance of axes as in Fig. 1.

Slight pressure in the parieto-occipital region (1-2 mm) also modified the conditioned-reflex reaction: the heart beat was not quickened but, on the contrary, slowed. However, this effect was observed less frequently than during stimulation in the region of the locomotor centers of the cortex. The application of a stronger pressure (4-6 mm), on the other hand, caused a slight increase in the conditioned-reflex effect on the heart.

The changes we have described may be explained by the development of inhibition at the site of mechanical stimulation of the brain. When pressure was applied in the region of the locomotor centers of the cortex, inhibition appeared where the central link of the reflex arc of the motor defensive reflex was situated [3, 8, 9, 13, 19, 20], so that when the conditioned-reflex stimulus was applied the rate of the heart was decreased, rather than increased.

If the site of stimulation lay outside the region of the arc of the conditioned motor-defensive reflex (for example, in the parieto-occipital region), the picture observed was different. During the application of slight pressure (1-2 mm) inhibition probably developed widely throughout the central nervous system, reached the region of the locomotor centers of the cortex, and produced the same effect as pressure on the motor area. A greater pressure (4-6 mm) evidently caused a stronger and more concentrated inhibition, around which a zone of positive induction was formed.

The appearance of inhibition in the area of the brain subjected to mechanical stimulation is also confirmed by the depression of the conditioned motor-defensive reflexes, the lowering of muscle tone, and so on [2]. Some authors, during mechanical stimulation of the brain, have observed depression of the electrical potentials [23]

and the development of "spreading depression" [24], which, according to Bures and Buresova [21], may be regarded as a manifestation of deep cortical inhibition.

Our results are also of interest to the clinician. We know that many patients after head injuries frequently develop disturbances of the rhythm of the heart [7, 15-17]. The transient character of these changes does not allow us to regard them as the result of organic changes. It is evident that these disturbances are caused by functional changes, resulting from a modification of the extracardiac regulatory influences.

Our findings contribute towards the understanding of the physiological principles lying at the basis of these changes in the heart rhythm. We know that the disturbance of the cardiac activity of an animal exposed to high accelerations is the result of the sharp change in the hemodynamics. In view of the foregoing facts, we consider that some amplification of this explanation is necessary: direct nervous influences also play a definite role in these changes in cardiac activity in these conditions, for during high accelerations it is possible for mechanical stimulation of the brain to occur.


## SUMMARY

Experiments were staged on 5 dogs. A study was made of the effect produced by mechanical stimulation of the brain by the pressure of 1 to 6 mm Hg on the cardiac activity regulation. Shifts in the rate of cardiac contractions were noted. The value and the nature of the changes depended on the initial rhythm of cardiac activity. Changes of the conditioned reflex reactions of the heart were noted. The localization of the cerebral area to be stimulated was also of significance. All the changes are explained as being the results of the inhibition process developed at the site of mechanical stimulation.


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