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PROTECTIVE ROLE OF THE HYPOTHALAMUS AGAINST PATHOLOGICAL CARDIAC REFLEXES

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UDC 616.12-009.81-02:/616.831.31+616.831.41

KEY WORDS: hypothalamus; modulating influences; cardiocardiac reflexes; viscerocardiac reflexes; pathological reflexes; control.

It is well known that the higher levels of the brain exert modulating influences on cardiac reflexes [4, 8, 11-17]. However, the character of these influences has not been explained. Most workers consider that they are entirely inhibitory. The possibility of facilitatory modulating influences on the cardiac component of baroreflexes has received occasional mention in the literature [12-14]. Experiments by the present writer on different classes of vertebrates (bony fishes, amphibians, mammals) showed that in the early stages of phylogenetic development the higher levels of the brain are adapted to exert modulating influences in various directions on the "cardiac parasympathetic center" in the medulla [5-10], where the arcs of these reflexes are closed. These influences may produce both augmentation and inhibition of cardiocardiac reflexes or may even abolish them completely. Both augmentation and weakening of reflexes were found by the writer in response to stimulation of the hypothalamic region in fishes [5] and frogs [2, 5] and of the cerebral cortex in cats [7, 8]. Influences of both types were exhibited on reflexes developing both during and immediately after stimulation of the brain. The character of the modulating influences probably depends on many factors and, in particular, on the degree of activation of higher levels of the brain [6, 8]. The two-way influences of higher levels of the brain, including the cerebral cortex, on cardiac reflexes revealed during electrical stimulation are confirmed also by the results of an investigation of cardiocardiac reflexes before and after surgical decortication [10]. Blocking descending influences from the cortex could be accompanied by both augmentation and weakening of reflexes in cats. This indicates the possibility of dual modulating influences under natural conditions also. Duality of modulating influences from the hypothalamus has been discovered by the writer on viscerocardial reflexes also [2], which must be regarded as one variety of pathological reflexes.

It was accordingly interesting to compare correlation between facilitatory and inhibitory influences from the hypothalamus on adaptive cardiocardiac and pathological viscerocardiac reflexes.

EXPERIMENTAL METHOD

Experiments were carried out on frogs. A small balloon was introduced into the right atrium, and distension of this balloon led to activation of the cardiac receptors and to the onset of cardiocardiac reflexes. Viscerocardiac reflexes were evoked by stretching the urinary bladder [2]. The hypothalamus was stimulated by square pulses of current passed through extracellular metal microelectrodes. The parameters of stimulation used were below

Department of Physiology of Man and Animals, Faculty of Biology, M. V. Lomonosov Moscow University. (Presented by Academician of the Academy of Medical Sciences of the USSR, V. A. Negovskii.) Translated from Byulleten' Éksperimental'noi Biologii Meditsiny, Vol. 93, No. 5, pp. 32-33, May, 1982. Original article submitted February 11, 1981.

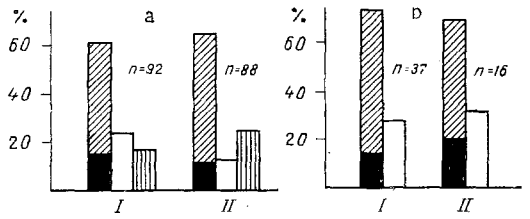

Fig. 1. Relative numbers of hypothalamic modulating influences (in both directions) on viscerocardiac (a) and cardiocardiac (b) reflexes in frogs. I) Combined stimulation, II) aftereffect. Obliquely shaded columns — inhibition; unshaded columns — facilitation; vertically shaded columns — reversal; black columns — abolition of effect. n) Number of tests, taken as 100%.

or at the threshold level relative to the heart rate. Changes in the heart rate were judged from the ECG. Modulating influences were identified from changes in magnitude of the reflex developing against the background of hypothalamic stimulation.

EXPERIMENTAL RESULTS

Viscerocardial reflexes took the form of considerable slowing of the heart rate. The development of the viscerocardiac reflex during hypothalamic stimulation, whereas hypothalamic stimulation alone in most cases caused no change in heart rate, was accompanied by a statistically significant increase or decrease in magnitude of the reflex. An increase in the reflex was evidence of facilitatory influences, a decrease of inhibitory influences.

Hypothalamic influences on cardiocardiac reflexes, manifested as quickening of the heart rate, also were found in both directions. Hypothalamic modulating influences were observed not only when the reflexes developed against the background of hypothalamic stimulation (combined stimulation), but also as an aftereffect, i.e., when the reflex developed 2-6 min after combined stimulation. Although the effect of hypothalamic stimulation on viscerocardiac reflexes, just as on cardiocardiac reflexes, could consist of weakening and facilitation of the reflexes, the ratio between the number of cases causing the various transformations of the reflex responses of the heart differed from that found during modulation of the adaptive cardiocardiac reflexes, manifested as quickening of the heart rate: In the case of viscerocardiac reflexes the number of tests with facilitatory influences was reduced (Fig. 1). This was shown particularly clearly in the after-period. Hypothalamic facilitatory influences on cardiocardiac reflexes were observed in 27% of cases (tests) with simultaneous stimulation and in 31% of cases in the after-period. By contrast with this, facilitatory influences for viscerocardiac reflexes were observed in 23% of cases with combined stimulation but only in 12% of cases in the after-period. In 16% of cases during combined stimulation and in 24% in the after-period the effects were reversed, i.e., the heart rate was quickened and not slowed. Since cardiac arrest or considerable slowing of the heart rate must be regarded as a profoundly pathological manifestation, the discovery of weakening or even reversal of the inhibitory viscerocardiac reflexes may be considered to be a physiologically appropriate form of regulation of "lower centers" by higher. These influences apparently correct the pathological character of viscerocardiac reflexes. In the absence of such control the viscerocardiac reflexes could bring about very serious disorders of cardiac activity.

This "protective" role of the higher levels of the brain is manifested particularly clearly in higher animals. Pathological forms of cardiocardiac reflexes, expressed as marked slowing of the heart rate, appeared in response to stimulation of the central end of the vagus nerve in cats [1, 6]. In none of the 12 experiments was stimulation of the cerebral cortex accompanied by an increase in reflex bradycardia. It can be tentatively suggested that modulating influences in higher animals acquire the character of a more sophisticated mechanism for weakening or abolishing pathological influences on the heart. This is confirmed also by data in the literature. It has been shown that localized injuries to the myocardium producing

temporary disturbance of functions in animals with an intact brain are accompanied in decerebrate animals by prolonged and stable disturbance of cardiac function [3]. On this basis the author cited considers that decerebration made the heart unable to adapt itself and to compensate the pathological changes.

The higher levels of the brain thus promote compensation of pathological changes in the heart itself and also, to some extent, prevent pathological influences on the heart, which are manifested when the function of other internal organs is disturbed.

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