ROLE OF α- AND β-ADRENORECEPTORS IN SYMPATHETIC FACILITATION OF INHIBITION OF PARASYMPATHETIC BRADYCARDIA

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Experimental data have recently been obtained which are not in conformity with the traditional view of the purely antagonistic relations between the sympathetic and parasympathetic divisions of the autonomic nervous system in the regulation of cardiac activity [1-5, 10, 11]. It has been shown on representatives of various classes of vertebrates, starting with fishes and ending with warm-blooded animals, that activation of the sympathetic nervous system may not only inhibit parasympathetic bradycardia, but may also potentiate it. This potentiation of parasympathetic bradycardia against the background of sympathetic activation is evidence of the possibility of a potentiating modulating sympathetic influence of parasympathetic effects.

Although the mechanisms of sympathetic inhibition of parasympathetic bradycardia have been the subject of many investigations [13], the problem cannot be considered to have been finally solved. More especially, it is difficult to say anything definite about facilitatory sympathetic influences.

The problem of the mechanisms of inhibitory and facilitatory sympathetic influences on parasympathetic effects cannot be solved without elucidation of the role of the adrenoreceptors which are involved in these interactions.

The aim of this investigation was to study the role of α and β-adrenoreceptors in the realization of facilitatory and inhibitory sympathetic influences on parasympathetic bradycardia.

EXPERIMENTAL METHODS

Bilateral vagotomy in the neck and catheterization of the femoral artery and vein were carried out on cats under urethane-chloralos anesthesia (480 mg/kg and 48 mg/kg respectively). The blood pressure (BP), ECG, and heart rate were recorded. Parasympathetic bradycardia was induced by electrical stimulation of the peripheral end of the divided right vagus nerve (frequency of stimulation 5-20 Hz, voltage 20 V, stimulus duration 0.5 msec, total duration of stimulation 2 sec). After a stable effect of vagus nerve stimulation had been attained, the same stimulation of the nerve was applied 1 min after the end of the electrical stimulation at the exit of preganglionic sympathetic fibers from the spinal cord at the level of T1-T3 to the heart [6, 9] or 1.5-2 min after intravenous injection of tyramine, which releases noradrenalin from sympathetic terminals [14], when the adrenergic effect was at its maximum. The parameters of sympathetic stimulation were: current 60 mA, frequencies 1 and 16 Hz, stimulus duration 1 msec, total duration of stimulation 10 sec. Tyramine was injected in concentrations of 25 and 200 μg/kg. Parasympathetic bradycardia, developing against the background of sympathetic activation, differed in value from that observed initially. Potentiation of the parasympathetic effect is evidence of a facilitatory (potentiating) sympathetic influence, weakening of the effect is evidence of inhibition. The magnitude of the parasympathetic chronotropic effect was taken to be the difference between the maximal and initial durations of the cardiac cycles as a percentage of the initial duration. Besides the magnitude of the parasympathetic chronotropic effect, its percentage increase (in the case of potentiation) or decrease (in the case of inhibition) against the background of sympathetic activation was

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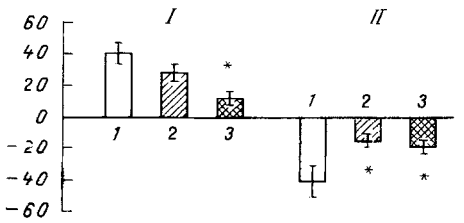

Fig. 1. Changes in modulating sympathetic influence on parasympathetic chronotropic effects after adrenoblockade. Abscissa: I) potentiation, II) inhibition. 1) Initial values, 2) β-adrenoblockade, 3) α-adrenoblockade; ordinate, modulating sympathetic influence (in %). *$p < 0.05$.

calculated relative to the initial parasympathetic bradycardia. The potentiating or inhibitory sympathetic influence was thus quantified. To explain the role of α- and β-adrenoreceptors, the magnitudes of the potentiating or inhibitory influence initially and against the background of the action of corresponding adrenoblockers was compared. The adrenoblockers were injected after several tests had been made of combined sympathetic and parasympathetic stimulation. The α-adrenoblockers used were phentolamine (2.5 mg/kg, USSR) or tropaphen (5 mg/kg, Hungary), and propranolol (2 mg/kg, East Germany) was used as the β-adrenoblocker. Completeness of the blockade was tested by injection of the corresponding mimetics (phenylephrine, isoproterenol). The significance of differences was determined by Student's test. Data are given in the M ± m form.

EXPERIMENTAL RESULTS

Statistical analysis of the results of all the experiments in which a facilitatory sympathetic effect was observed showed that the average value of the potentiating effect was 39%. α-Adrenoreceptor blockade reduced this value statistically significantly to 11% (Fig. 1). Reduction of the potentiating effect against the background of propranolol was not statistically significant. It can thus be concluded that α-adrenoreceptors are mainly involved in the development of the facilitatory sympathetic influence on parasympathetic bradycardia in anesthetized cats.

The magnitude of the inhibitory influence, namely 41%, was reduced statistically significantly by an almost equal degree both after α-adrenoblockade (to 19%) and after β-adrenoblockade (to 20%). It can be tentatively suggested that both types of adrenoreceptors participate in the realization of inhibitory sympathetic influences. Investigations now published provide evidence of the presence of adrenoreceptors on endings of both preganglionic neurons of the vagus nerve [7, 8, 12, 13, 15]. These data suggest that both facilitatory and inhibitory sympathetic influences on parasympathetic effects may be realized at the level of both intracardiac parasympathetic ganglia and of myocardial cells through pre- and postsynaptic mechanisms.

We also know that α-adrenoreceptors located on sympathetic terminals can regulate the release of noradrenalin into the heart on the negative feedback principle [7, 13]. Activation of blockade of these receptors, modulating the effect of sympathetic activity itself, may also be involved in the mechanism of the sympathetic influence on parasympathetic chronotropic effects on the heart.

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MOTIVATION OF CENTRIFUGAL "TUNING" INFLUENCES ON TASTE RECEPTORS

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Peripheral receptor formations of virtually every sensory modality are not impartial witnesses and transformers of the physical parameters of acting stimuli, but they undergo the most active tuning for sensory information perception, due to the presence of wide intersensory interactions, and for that reason a stimulus acting on one sensory system must inevitably influence the sensitivity of other sensory systems and their ability to perceive [8-10]. For instance, a phenomenon manifested as a change in the level of mobilization of the taste papillae of the tongue during stimulation of gastric receptors has been discovered and investigated in detail by a psychophysiological method [6], and it has been called the gastrolingual reflex. Taste-sensitive elements of the tongue have been shown to be in a state of "intermittent" activity, and their number at any given moment is determined both by the general sensory background of the organism and by influences from other clearly defined reflexogenic zones [1, 2, 7]. After introduction of peptone into the stomach or inflation of the stomach [3, 4] the character of the spike flow in the central end of the lingual branch of the glossopharyngeal nerve changes in a regular manner. This has been rightly interpreted by the authors cited as a manifestation of centrifugal action on the sensory apparatus of the tongue, as a neurophysiological equivalent of centrifugal "tuning." In recent years [5] an extensive series of investigations of neuronal activity in different brain regions under the influence of different dominant motivations has been undertaken. The results have shown that the stochastic structure of the pattern of activity of neurons belonging both to afferent and to efferent and association areas of the brain acquire specific features under conditions of hunger and undergo specific changes when the need is satisfied. These data were interpreted by Sudakov as a manifestation of the holographic principle of the working of the brain, which was reflected in the holographic principle of organization of a dominant motivation, enunciated by him [11, 12].

We have studied motivation-determined tuning of taste receptors.

EXPERIMENTAL METHODS

The technique developed at the P. K. Anokhin Institute of Normal Physiology for detection of dominance of interspike intervals was used, and efferent impulsation in the lingual branch of the frog glossopharyngeal nerve was analyzed. The experimental model described in [3, 4] was adopted. Experiments were carried out on 17 male frogs (Rana temporaria) immobilized by injection of curare-like drugs (tubocurarine $1 \cdot 10^{-6}$ g/ml, diplacin $1 \cdot 10^{-5}$ g/ml) into the lymph sac. Electrical activity was recorded through bipolar electrodes from the lingual branch of the glossopharyngeal nerve, which was divided distally to the point of

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