periods did not differ significantly ($P > 0.05$). The descending phase of the second negative wave was much more gradual than that of SEP. The total duration of the three-component complex of KEP was 12 msec longer than that of SEP. The amplitude of KEP was significantly shorter than that of SEP ($P < 0.05$).

The results thus show that KEP in response to stimulation of the contralateral forelimb differ the most in the initial and final stages of development of the potential, whereas in the interval between the peak of the first positive to the peak of the second negative wave these differences were minimal, and in the last case this may reflect homogeneity of the afferent volley, arriving at that moment in the case of both kinesthetic and somatosensory stimulation. The shorter absolute latent period of KEP suggests a faster level of conduction of the modality-homogeneous afferent volley than in response to electrical stimulation, causing excitation of heteromodal receptors and mixed nerve fibers. A longer duration of KEP than of SEP can evidently be explained by the much longer time of peripheral stimulation, when kinesthetic influences continued to reach the projection cortex from the receptors for 40 msec.

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CORTICAL REPRESENTATION OF THE SINUS NODE OF THE HEART

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UDC 612.178.3:612.825.4

KEY WORDS: sinus node; afferentation; cerebral cortex

Much still remains unexplained in the problem of representation of the afferent systems of the heart at higher levels of the CNS. Most research on this theme either has been concentrated on the study of afferent impulsation arising in fibers of the vagus nerve in response to stimulation of the myocardial receptor apparatus of the atria and ventricles, the coronary vessels, and the pericardium, or has been limited to recorded evoked potentials (EP) in cerebral cortex and certain deep brain formations arising as a result of stimulation of cardiac branches of the same vagus nerve [1, 3, 4, 9, 10, 12-15]. The possibility that impulses may be conducted from receptor formations of the myocardium and pericardium to subcortical and cortical structures of the CNS by means of spinal afferent systems has received little study. Nevertheless, afferent systems have an "output" to tracts of the spinal cord via thick myelinated fibers and fibers of medium and small diameter, connecting the heart with spinal structures [2, 5, 7, 8, 11].

The aim of this investigation was to study the role of spinal pathways conducting afferent information, and to identify the distinguishing features of cortical representation of one of the most regularly important myocardial formations, the sinus nodal zone (SNZ).

METHOD

Acute experiments were carried out on 28 adult cats weighing 2.5-3.0 kg, anesthetized with chloralose (40-50 mg/kg) and curarized. Bipolar stimulating electrodes, insulated from surrounding tissues, were fixed in SNZ and to the ventricular myocardium. The heart was stimulated with square pulses (0.3 msec, 10-15 mA, frequency not more than 0.3 Hz) from an

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(Presented by Academician of the Academy of Medical Sciences of the USSR G. N. Kryzhanovskii.) Translated from Byulleten' Éksperimental'noi Biologii i Meditsiny, Vol. 100, No. 11, pp. 517-519, November, 1985. Original article submitted April 19, 1985.

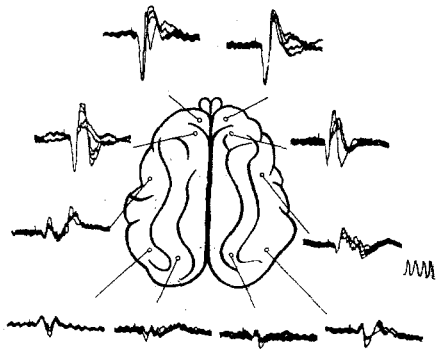

Fig. 1. EP recorded in cortex of right and left hemispheres of a cat during stimulation (0.3 msec, 10-15 mA) of sinus nodal zone of the heart. Each trace obtained by superposition of five sweeps of beam on CRO screen. Location or recording electrodes on cortex indicated by small circles. Here and in Figs. 2 and 3, calibration: 50 Hz, 50 μV.

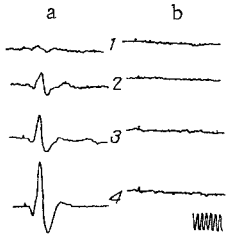

Fig. 2

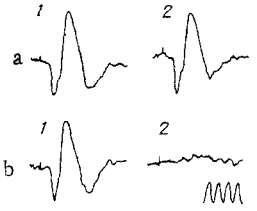

Fig. 3

Fig. 2. EP recorded in cortex (right hemisphere, anterior cruciate gyrus) in response to stimulation of increasing strength (from 5 to 20 mA, 1-4 respectively) of SNZ (A) and left ventricular myocardium (B).

Fig. 3. EP recorded in cortex of right hemisphere to stimulation of SNZ of cat heart before (a, 1) and after bilateral vagotomy (a, 2) and also before (b, 1) and after (b, 2) transection of spinal cord at level C2.

ÉSU-2 electrostimulator. EP were derived from the exposed cortical surface by silver ball electrodes, by a monopolar technique (the reference electrode was fixed in the frontal bone of the animal's skull). To amplify and record EP, a TR-4602 oscilloscope and FOR-2 motion picture camera were used. Throughout the experiment the animals' body temperature was kept constant, and the surface of the cerebral cortex was irrigated with warm (37°C) physiological saline. The ECG also was recorded in standard lead II.

EXPERIMENTAL RESULTS

Electrical stimulation of SNZ led to the formation of distinct EP in the cortex of both right and left hemispheres. The focus of maximal activity (FMA) of EP as a rule was discovered on the anterior and posterior cruciate gyri (the association area around the cruciate gyrus, namely PCA, motor-sensory area I, and part of sensomotor area I). In these areas positive-negative EP were recorded with a latent period (LP) of 19.74 ± 0.32 msec and an amplitude of the first positive phase of 60-70 μV. No significant differences were found in the configuration and duration of the latent period of EP in the left and right hemispheres. Besides this, positive-negative EP appeared in both hemispheres in small areas of the cortex belonging to sensomotor area II, with LP of 20.52 ± 0.54 msec, and with an amplitude of the first positive phase of not more than 25-35 μV. In the remaining areas of the cortex evoked potentials either did not occur at all, or long-latency negative-positive potentials with an amplitude of the first negative phase of not more than 20-30 μV were recorded (Fig. 1).

Stimulation of the myocardium of the right and left ventricles, even when pulses with a strength of 45-50 mA were used, did not induce EP in the cortex (Fig. 2).

In half of the experiments, during recording of EP in the cortex by means of special instruments, fixed immediately before the experiment on both vagus nerves at the level of the thyroid cartilage, an instant bilateral vagotomy was performed. EP continued to be recorded in the cortex after this procedure, without any appreciable changes in shape of the phases or in LP. Transection of the spinal cord at level C2, however, led to total disappearance of EP throughout the area of the cortex (Fig. 3).

The experiments thus showed that an area of right atrial myocardium, functionally defined as SNZ, projects via spinal tracts into rostral portions of the cortex of both hemispheres (PCA, motor-sensory area I, and sensomotor area I), and to a lesser degree, into the region of the anterior ectosylvian gyrus (sensomotor area II). Bilateral vagotomy, which did not lead to any evident changes in EP, confirms the fact that sensory impulses from this zone (SNZ) reach the higher levels of the CNS mainly via spinal afferent tracts. Stimulation of the ventricular myocardium did not reveal any connections between the ventricles and the cortex.

There is no doubt that SNZ is nonhomogeneous. Morphological investigations [7, 8] have shown that various receptor formations, and thick, average, and thin myelinated fibers are concentrated in this region. In the present experiments no attempt was made to study exact electrophysiological details: what concrete afferent structures in SNZ are responsible for the spread of the flow of afferent information to the cortex via spinal or vagus pathways. An attempt was made to demonstrate in principle that such connections were present with definite regions of the cortex of both hemispheres.

According to the experimental results, the latent period of EP arising in the cortex in response to stimulation of SNZ is 19-20 msec. This is evidence that afferent impulses reach the cortex via relatively slowly conducting, polysynaptic pathways. For comparison it may be noted that, according to earlier data [1], EP arising in the rostral zones of the cortex in response to stimulation of cardiac branches of the vagus nerve have an LP of 15-18 msec. This suggests that information on the state of the heart muscle reaches these areas of the cortex in two streams: the first, more rapid, along afferents of the vagus nerves, the second, slower, along spinal pathways. It was impossible to judge, on the basis of available data, the functional significance of each of the two streams. However, one thing is clear — the dual nature of the afferentation is by no means accidental. It as it were precedes the dual nature of the subsequent effector control of the heart (by sympathetic and parasympathetic divisions of the nervous system).

FMA of potentials arising in the cortex in response to stimulation of SNZ was topographically identical with or very close to the primary areas of representation of the vagus nerve as a whole, and of its cardiac branches in particular [1, 9]. Moreover, according to other investigations [6], stimulation of areas of the cortex located within the anterior orbital and premotor areas in rabbits causes definite changes in the cardiac rhythm and also in the blood pressure.

All these findings suggest that functional centers for cardiac regulation, which receive and process afferent information and take part in the control of the activity of this vital organ, are located in the rostral zones of the cortex. Another fact which undoubtedly deserves attention is that of the unique "symmetrical" representation of SNZ in the cortex of the right and left cerebral hemispheres. This type of afferent projection of an unpaired organ suggests that evolution has "made sure" of the greatest possible reliability both of pathways supplying information to the highest levels of the CNS about the functional state of this vital organ and also, evidently, of the most effective possible regulation of its activity.

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GLUTAMIC ACID INCREASES THE MAXIMAL INTENSITY OF MYOCARDIAL CONTRACTILE FUNCTION

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UDC 612.172.014.46:577.112.384.4

KEY WORDS: papillary muscle; contractile function; glutamic acid

The chronoinotropic relationship for the myocardium of most mammals is characterized by an increase in the force or amplitude of contraction up to a certain optimal frequency, and if this frequency is exceeded, the amplitude of contraction decreases and alternation develops [3, 12]. Besides contraction, relaxation also is disturbed [3, 7].

The aim of this investigation was to compare depression of contraction and relaxation of the myocardium at a high frequency when energy formation is deficient; for this purpose isolated papillary muscles of varied thickness were used, for we know that the thickness of the muscles, because of an increase in oxygen diffusion, is a factor limiting maximal contractile function [1, 8, 10]. An attempt also was made to influence these processes with glutamic acid (GA), which has the property of improving the state of the myocardium when affected by hypoxia or hypoperfusion [4, 6].

EXPERIMENTAL METHOD

Experiments were carried out on isolated papillary muscles from the guinea pig right ventricle. The muscles were contracted by electrical stimulation in Krebs' solution, saturated with 95% O_2 + 5% CO_2, pH 7.3-7.4 at 29°C. The contraction signal obtained from a photoelectric transducer and its first derivative were recorded on a "Gould Brush 2200" two-channel recorder. The thickness of the muscles was determined from the area of cross-section, using data for weight and length of the muscle. The maximal rate of contraction and relaxation was expressed as a ratio of muscle length in the usual way. Details of the technique were described previously [2]. In each experiment the frequency of contractions was increased stepwise from 0.5 to 3.5 Hz at steps of 0.5 Hz, and was maintained for 1 min at each frequency. GA (3.5 mM) was added to the perfusion fluid after the first series of increased frequency, and the second series was performed 30 min later in the presence of GA. The results were subjected to statistical analysis by Student's test and expressed in the form $M \pm m$.

EXPERIMENTAL RESULTS

The positive inotropic effect of an increase in frequency was sufficiently well marked in all experiments irrespective of the thickness of the muscles. Nevertheless, with an increase in thickness, not only the amplitude of contraction, but also the optimal frequency decreased (Fig. 1). For very thick muscles, in most experiments it was 2 Hz, compared with

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 0007-4888/85/0011-1464$09.50