NATURE OF THE PHASE SYNDROME OF CARDIAC HYPERDYNAMIA

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Experiments on dog heart-lung preparations with constant venous inflow and peripheral resistance have shown that stimulation of the Pavlov augmentor nerve does not change the heart rate but shortens mechanical systole and the periods of isometric contraction and expulsion. The identity between the pattern of changes in the structure of left ventricular systole as described above with the phase syndrome of hyperdynamia in man suggests that this syndrome arises under the influence of impulses reaching the heart along the Pavlov augmentor nerve.

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In healthy persons doing muscular work or in emotional states and also in patients with certain types of pathology, the syndrome of cardiac hyperdynamia is observed. It is manifested by shortening of the periods of isometric contraction, expulsion, and mechanical systole [2].

The physiological mechanisms leading to such characteristic changes in the dynamics of cardiac contraction are not yet clear. The fact that the hyperdynamic syndrome is unconnected with changes in hemodynamic conditions (it is found in the presence of an unchanged venous return and diastolic pressure) means that the cause of the characteristic changes in the dynamics of cardiac contraction must be sought in direct influences on the myocardium. V. L. Karpman [2] suggests that such an influence may be transmitted by the Pavlov augmentor nerve. I. P. Pavlov [3] observed shortening of systole under the influence of the augmentor nerve and an analogous effect was described by Baxt [1] under the influence of its accelerator branches.

In the present investigation, the phase structure of systole was studied during stimulation of the augmentor nerve.

EXPERIMENTAL METHOD

Nineteen experiments were performed on dog heart-lung preparations. Such preparations provide a convenient model for the study of direct nervous influences on the heart [4, 5] and enable observations to be made during constant hemodynamic conditions. To determine the duration of the phases of the cardiac cycle, synchronized recordings were made of the ECG and also of the pressure within the left ventricle and aorta by means of an EM2-01 electromanometer (Hungary). The period of contraction at present is divided into two principal phases: the phase of change in shape or asynchronous contraction, and the phase of isometric contraction or development of pressure. The duration of asynchronous contraction was calculated from the interval between the Q wave of the ECG and the beginning of the sharp rise of the intraventricular pressure curve. The duration of isometric contraction was calculated from the interval from the beginning of increase in intraventricular pressure to the beginning of increase of intraaortic pressure, and the period of expulsion was counted from the beginning of the rise of the intraaortic pressure curve to the beginning of the incisura on the same curve. The nerves were stimulated by an induction current through 3-pole platinum electrodes.

EXPERIMENTAL RESULTS AND DISCUSSION

Of the nineteen experiments performed, the seven in which stimulation of the principal cardiac branch (the Pavlov augmentor nerve) gave an isolated inotropic effect without change in heart rate were analyzed. In the remaining 12 cases the augmentor effect was accompanied by a slight positive chronotropic effect.

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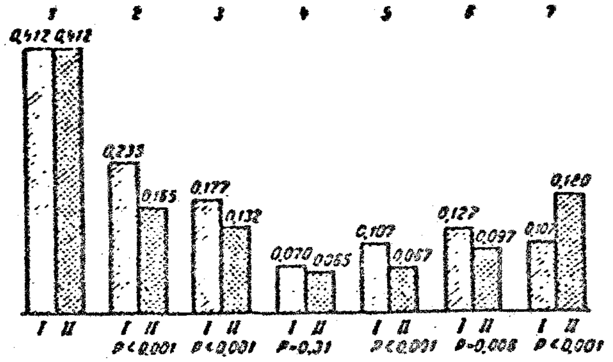

Fig. 1. Changes in duration of phases of left ventricular systole under the influence of the Pavlov augmentor nerve (mean results of seven observations). I) before stimulation of nerve; II) during stimulation. Numbers above columns denote duration of periods of cycle (in sec). 1) cycle; 2) mechanical systole; 3) period of contraction; 4) period of asynchronous contraction; 5) period of isometric contraction; 6) period of expulsion; 7) diastole.

Stimulation of the augmentor nerve caused characteristics changes in the structure of left ventricular systole (Fig. 1 and 2). The duration of total and mechanical systole was reduced, the latter to 70% of its initial value after stimulation of the nerve. The duration of systole was shortened because of shortening both of the period of contraction and of the period of expulsion. The period of contraction was shortened on account of the phase of isometric contraction, because the duration of asynchronous contraction remained practically unchanged. The duration of the period of isometric contraction was 62.6% of its value before stimulation of the nerve. The period of expulsion was shortened during stimulation of the augmentor nerve to 71.3% of its initial value. The decrease in duration of systole while the heart rate remained constant was naturally accompanied by an increase in the duration of diastole, to 168.2% of its initial value.

The changes in structure of the cardiac cycle developing under the influence of the augmentor nerve revealed an increase in the contractile power of the myocardium. This increase is confirmed by several calculated indices. For instance, the time taken by the heart to complete its external work i.e., the intrasystolic index (ratio between duration of expulsion period and duration of mechanical systole, in percent [2]), was relatively increased, from 54 to 59% ($P = 0.01$). The maximal rate of increase of pressure within the left ventricle, another index of the contractile power of the myocardium [6], was also increased. In the experiment on November 23, 1964, for example, it increased during stimulation of the augmentor nerve from 650 to 1020 mm Hg/sec.

Hence, the pattern reflecting the influence of the Pavlov augmentor during the phase syndrome of hyperdynamia in man. The fact that characteristic changes in the structure of the cardiac cycle were obtained while the heart rate was unchanged rules out any possible effect of changes in the duration of the cycle, and the absence of change in hemodynamic conditions ensured constancy of the venous inflow and peripheral resistance. This investigation did not give an answer to the question whether the augmentor nerve is the only conductor of impulses producing the phase syndrome of cardiac hyperdynamia. Shortening of the duration of systole during the action of accelerating factors cannot be accepted as proof that they play a direct part in formation of the hyperdynamic syndrome, because shortening of the phases of systole is observed during an increase in heart rate caused by various factors, not only by nervous influences. In our experiments on the heart-lung preparations, for instance, an increase in temperature of the blood flowing into the

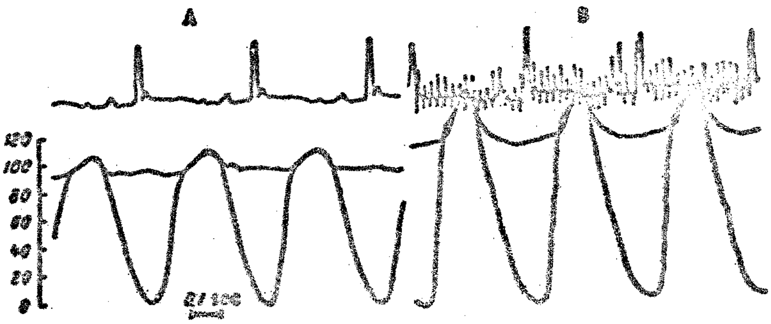

Fig. 2. Effect of stimulation of au[illegible] nerve on [illegible] of cardiac [illegible]. A) before [illegible]; B) during [illegible]lation. [illegible] ECG, [illegible] pressure in left ventricle.

heart regularly caused shortening of all the components of systole, while a fall of temperature, on the other hand, lengthened them. The results of experiments revealing shortening of systole during stimulation of the stellate ganglion with an artificially created constant rhythm [7] likewise do not provide the answer to this question, because under these circumtances both accelerator and augmentor fibers are stimulated simultaneously.

Pavlov, who made a detailed analysis of the pathway of nervous influences on the dynamics of cardiac contractions, concluded that fibers accelerating contraction run to the heart in the augmentor nerve and not in accelerator branches.

The possibility of experimental reproduction of the pattern of the phase syndrome of cardiac hyperdynamia by stimulation of the Pavlov augmentor nerve suggests that under natural conditions this syndrome may also arise under the influences of impulses reaching the heart along the augmentor nerve.

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