MECHANICAL TENSION IN DIFFERENT PARTS OF THE LEFT VENTRICLE UNDER THE INFLUENCE OF INOTROPIC AGENTS

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KEY WORDS: mechanical tension; wall of left ventricle; apex of heart; inotropic agent.

In the study of myocardial contractility of the left ventricle (LV) a distinction is drawn between force and velocity components, determined as intraventricular pressure and velocity of blood flow during the work cycle of the heart [2, 4, 5]. However, the degree of involvement of the different parts of the myocardium in the formation of these components is not yet clear.

In the present investigation the action of various inotropic agents on mechanical tension developed in the wall of LV and in the apex of the heart, and the contribution of these parts of the heart to the formation of hemodynamic parameters were studied.

EXPERIMENTAL METHOD

Altogether 25 experiments were carried out on seven anesthetized (trimeperidine 4 mg/kg, pentobarbital 10-15 mg/kg), mongrel dogs of both sexes weighing 12-20 kg. Thoracotomy was performed and the pericardium opened, during artificial ventilation of the lungs by the RO-2 apparatus (50 cm^3/kg, 16 inspirations/min). The pressure inside the left ventricle (P_{lv}) was recorded by means of a catheter, the velocity of the blood flow in the aortic orifice (Q) by a Nicotron-376 fluorometer (Norway), the ECG in standard lead II, and the local mechanical tension in the wall by LV (F_w) and at the apex of the heart (F_a) also were recorded. For the latter, a catheter, to the end of which a rubber balloon with a diameter of 1.2 mm, filled with liquid, was attached, was introduced into the substance of the myocardium. Tension was determined by mechanical pressure of the surrounding myocardial tissue on the balloon, and this was transformed into an electrical signal. An inotropic effect was induced by intravenous injection of 0.1-0.3 ml of a solution of adrenalin (1 : 1000) or 5 ml of a 10% solution of $CaCl_2$. P_{lv}, F_w, F_a, and the ECG were recorded on "Salyut" and 6NEK-4 (East Germany) monitors.

EXPERIMENTAL RESULTS

Data on relative changes in the amplitudes of F_w, F_a, and P_{lv} in response to injection of adrenalin and $CaCl_2$ are given in Figs. 1 and 2. Mechanical tension and left intraventricular pressure were normalized relative to the initial values of these functions. Adrenalin increased F_w, F_a, and P_{lv} by about 1.5 times (Fig. 1). There are very little difference between the rates of rise and fall of these values.

Injection of $CaCl_2$ (Fig. 2a) caused an increase in F_w by 1.4 times and in F_a by 1.8 times. Under these circumstances P_{lv} was increased by 1.3 times and the rate of its rise correlated with the rate of rise of F_w. The inotropic effects of $CaCl_2$ was manifested differently in different experiments. As Fig. 2b shows the increase in F_w, F_a, and P_{lv} was almost identical, but the fall of these values to the initial level differed significantly: F_w and P_{lv} decreased simultaneously, whereas F_w decreased about 220 sec later. In this case P_{lv} correlated with F_w (as shown in Fig. 2a).

In all the experiments constant types of myocardial response to adrenalin and $CaCl_2$ were observed, regardless of individual differences between the animals. Incidentally, when adrenalin and $CaCl_2$ were injected alternately into the same animal, the types of these responses were reproduced in different experiments. It can accordingly be concluded that the differences in developed tension in the wall of LV and in the apex after

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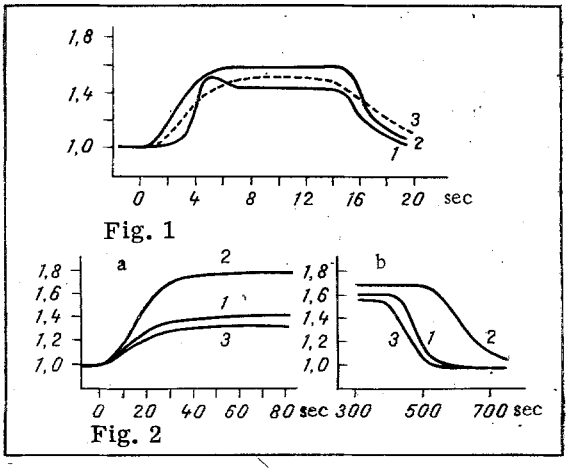

Fig. 1. Changes in F_W (1), F_a (2), and P_{lV} (3) under the influence of adrenalin. Here and in Fig. 2: F_W, F_a, and P_{lV} normalized relative to initial values of these functions.

Fig. 2. Inotropic effect in wall of LV and in apex of heart following injection of $CaCl_2$ (a) and end of inotropic effect (b). Legend as to Fig. 1.

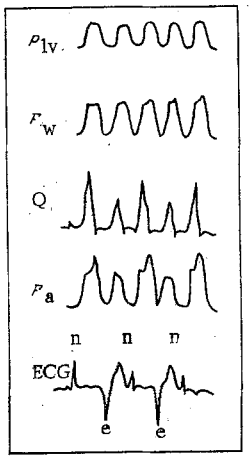

Fig. 3. Changes in F_W, F_a, F_{lV}, and velocity of blood flow Q in orifice of aorta during alternation of normal (n) and ectopic (e) ECG complexes (lead II).

injection of $CaCl_2$ were not associated with the characteristics of the coronary blood flow or the blood supply to these parts of the heart.

Changes in F_W, F_a, and P_{lV} on the appearance of ectopic excitation waves alternating with normal complexes are shown in Fig. 3. Functional dissociation of individual areas of myocardium of LV and a difference in their contributions to the formation of pressure and blood flow can be seen. Normal amplitudes of F_W, P_{lV}, and F_a, and Q curves corresponded to every complex of sinus origin. With the appearance of ectopic waves F_W and P_{lV} did not fall, F_a fell to 0.6, and Q at these same points fell to 0.5 of the initial value. This relationship between F_a and Q is due to the fact that ectopic excitation was localized in the ventricles and was determined by the order of excitation of different parts of the heart. The appearance of normal excitation restored F_a and Q to their initial values.

Changes in F_W thus correlated with changes in P_{lV} (Fig. 2a, b), whereas the change in F_a affected the value of the stroke volume but had no significant effect on P_{lV} (Fig. 3). These results agree with those of the

investigation of the shortening function [3] and with changes in configuration [1] of the wall of LV and the apex during the work cycle of the heart. Consequently the wall of LV and apex of the heart make different contributions to the formation of parameters of the central hemodynamics, namely the pressure inside LV and the stroke volume of the heart, under the influence of inotropic agents.

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INACTIVATION OF POSTURAL ASYMMETRY FACTOR AT THE STAGE OF COMPENSATION OF A POSTURAL DISTURBANCE DUE TO UNILATERAL ABLATION OF THE MOTOR CORTEX

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Unilateral injury to the cortex of the anterior lobe of the cerebellum [1], the vestibular system [2], and the motor area of the cerebral cortex [3] lead to functional modifications of the segmental apparatus manifested as fixation of postural asymmetry (PA) of the hind limbs by the lumbar segments of the spinal cord. Fixation of PA has been shown to be induced by PA factors (PAF) of peptide nature, formed in the injured CNS, as has been shown for cerebellar and vestibular factors [1, 2]. The chemical nature of PAF produced after removal of the motor cortex (cortical PAF) has not been investigated.

Comparison of the dynamics of fixation of PA with changes in PAF activity showed that PA of the hind limbs disappears (is compensated for) 3 weeks after unilateral removal of the motor cortex, against the background of a decrease in PAF activity in the CSF and brain tissue to zero [4]. The cause of the decrease in cortical PAF activity has not been explained.

The aim of this investigation was to study this problem and also to determine the chemical nature of cortical PAF.

EXPERIMENTAL METHOD

Experiments were carried out on 120 noninbred male albino rats weighing 160-180 g. Under ether anesthesia the left motor area of the necortex – the zone of cortical representation of the right hind limb, was removed. On the 3rd and 21st days after the operation the animals were decapitated, the brain removed, frozen in liquid nitrogen, and kept at −20°C; 5 g of tissue was homogenized in 0.2 M HCl as described previously [5]. The supernatant obtained after centrifugation (100,000 g, 60 min) was collected and the pH adjusted to 6.7 by the addition of 0.2 M KOH, the residue was removed by centrifugation, and the supernatant was freeze-dried. Next, 100 mg of the freeze-dried products containing 10 mg protein [10] was dissolved in 1 M acetic acid and applied to a K 26/40 column (Pharmacia, Sweden), filled with Sephadex G-25 ("fine"). Gel-filtration was carried out in 1 M acetic acid with a flow rate of 3.5 ml/h·cm^2. The fractions collected were neutralized with 1 M

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