CONTRACTILE FUNCTION OF UNDAMAGED PARTS OF THE LEFT VENTRICLE AFTER ACUTE DISTURBANCES OF THE CORONARY CIRCULATION

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Experiments on dogs showed that after ligation of the descending branch of the left coronary artery compensatory hyperfunction develops principally in the basal portions of the myocardium of the left ventricle.

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The primary cause of circulatory disturbances in myocardial infraction is the exclusion of the ischemic portion of the heart muscle from contractile activity. As Kreuzer and Schoeppe [2] showed recently, contractions of the ischemic area of the myocardium almost cease within a few minutes after interruption of the circulation in the coronary artery. Although, according to data obtained by these workers, the contractile activity of the affected area of myocardium is restored soon after, it is obvious that a necrotic or cicatrized portion of the myocardium must lose its contractile function completely. Since in most cases the hemodynamics is restored in this situation, it has been suggested that an important factor in this compensation must be an increase in the contractile function of undamaged parts of the myocardium [1, 5]. However, hyperfunction of this type has not been studied experimentally.

In the present investigation the contractile function of undamaged portions of the myocardium of the left ventricle was studied after an acute disturbance of the coronary circulation.

EXPERIMENTAL METHOD

Experiments were performed on dogs anesthetized with urethane (1-1.5 g/kg body weight) with the chest closed and on artificial respiration.

The index used to describe the contractile function of the heart muscle was the intramyocardial pressure (IMP), recorded by the method described by Kreuzer and Schoeppe [3]. The principle underlying this method is measurement of the pressure in a hollow needle, filled with physiological saline and firmly connected to an electromanometer with low volume displacement, inserted into the myocardium. The absolutely rigid joint between the recording needle and electromanometer used by Kreuzer and Schoeppe proved unsuitable for lengthy experiments, for the heart in contact with the end of the needle became wounded as it contracted and changes in the configuration and size of the heart in the course of the experiment affected the position of the needle in the substance of the myocardium. In the present investigation, therefore, instead of a metal coupling between the needle and electromanometer, a piece of a standard catheter about 25 cm in length was used.

Depending on the place from which the IMP was to be recorded, thoracotomy was performed in the 4th-6th intercostal space. The pericardium was opened widely, and a washer was sutured to the epicardium, fixing the needle in the chosen portion of the anterolateral surface of the left ventricle.

A silk ligature was passed beneath the descending branch of the left coronary artery (together with the vein) or beneath one of its main branches, and after all the readings had been taken it was tied. The coronary artery was ligated only after no significant change occurred in all the indices recorded over a period of 15-30 min.

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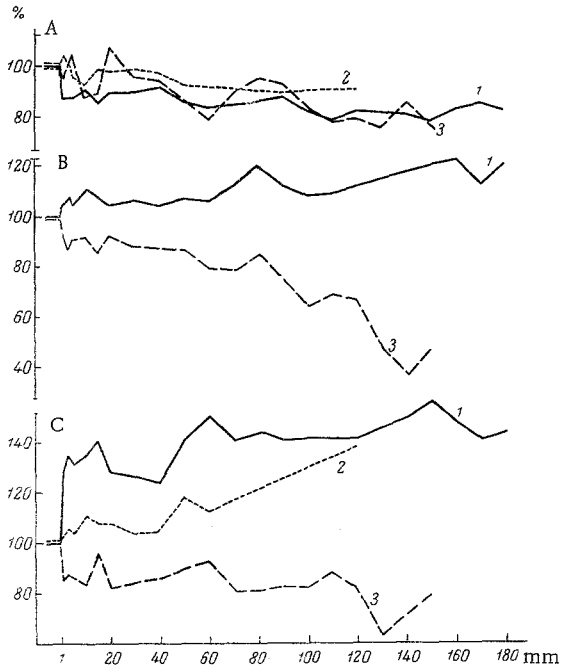

Fig. 1. Dynamics of pressure in left ventricle (A), intramyocardial pressure (B), and IMP/LV ratio (C) after acute disturbance of coronary circulation (in % of initial values). 1) Experiments with IMP recorded in basal region of left ventricle; 2) in middle third of left ventricle; 3) in apical region of left ventricle.

The ECG and the pressure inside the left ventricle and (or)in the aorta were recorded along with the IMP. Traces were drawn on a "Galileo" ink-writing recorder and simultaneously on a type N-700 loop oscillograph.

The anterolateral surface of the left ventricle was divided conventionally into three parts: base, middle third, and apical region. The dynamics of the IMP after ligation of the coronary artery was recorded in the region of the base in 21 experiments, in the middle third of the ventricle in 8, and in the apical region in 12 experiments. The needle used to record the IMP was buried in the myocardium to a depth of 6-8 mm from the outer surface of the heart. In every case the coronary artery was ligated in such a way that the region in which the IMP was recorded was protected from the region of the "infarct" by the intact coronary artery.

EXPERIMENTAL RESULTS AND DISCUSSION

In all experiments ligation of the coronary artery was accompanied by a gradual decrease in the pressure within the left ventricle, which fell on the average by 10-20% of its initial value during 2-3 h of the experiment (see Fig. 1, A). The same pattern was observed with changes in the intraaortic pressure. The figure (A) shows that the dynamics of intraventricular pressure in the experiments when the IMP was recorded in different parts of the myocardium of the left ventricle was practically the same.

In contrast to this, the dynamics of the contractile function in different parts of the myocardium after an acute disturbance of the coronary circulation was substantially different. The value of the IMP in the basal region of the left ventricle, which had a mean value of 187 mm before ligation of the coronary artery, rose progressively after a disturbance of the coronary circulation, to reach 212 mm by the end of the 1st hour of the experiment ($P < 0.05$). Three hours after ligation of the coronary artery the IMP in the basal part of the ventricle had risen to 226 mm, or 120% of its initial value (see Fig. 1, B). A clear increase in the IMP in the basal part of the left ventricle after ligation of the coronary artery was observed in 12 of the 21 experiments. In 7 experiments the IMP in this region of the heart was essentially unchanged, and it was lowered in only one experiment. An increase of 15% in the IMP was found also in the middle third of the ventricle after ligation of the coronary artery. This effect was observed in 4 of 8 experiments. In 3 experiments the IMP was unchanged, and in one it was lowered.

Hence, in more than half the experiments with an acute disturbance of the coronary circulation compensatory hyperfunction of the myocardium of the undamaged basal and middle portions of the left ventricle was observed.

The dynamics of the IMP in the apical region of the left ventricle after ligation of the coronary artery differed significantly from that in the basal and middle portions of this ventricle. The IMP in the apical region, for instance, whose mean value before ligation of the artery was 227.4 mm, fell after 2 h to 149.4 mm, or 66% of its initial value, and after 2.5 h it had fallen still further (see Fig. 1, B). A decrease in IMP in the apical part of the left ventricle after ligation of the coronary artery was observed in 6 of 12 experiments. In 1 experiment the IMP was raised, and in 5 it was substantially unchanged.

The dynamics of the ratio between the IMP in the different parts of the left ventricle and the intraventricular pressure recorded in the same experiment (the IMP/LV ratio) is interesting.

The IMP/LV ratio for the basal and middle portions of the left ventricle before ligation of the coronary artery was 0.9 and 0.99, respectively. Immediately after ligation of the coronary artery the value of this ratio was considerably increased for the base of the left ventricle, and later it remained stabilized at a level 40-55% higher than initially (see Fig. 1, C). An increase in the IMP/LV ratio, although less marked, was observed also in the middle third of the left ventricle after ligation of the coronary artery. An increase in the IMP/LV ratio was noted in 11 of 15 experiments in which the IMP was recorded at the base of the heart and in all 4 experiments when it was measured in the middle third of the left ventricle (the number of experiments in which the pressure in the left ventricle was recorded is shown). The pattern of changes in the ratio betweem IMP and the pressure in the aorta was equally definite.

The dynamics of the IMP/LV ratio in the apical region of the left ventricle was opposite to that in other parts of the left ventricle after coronary ligation. Whereas in other parts of the ventricle the ratio increased, at the apex it decreased immediately after coronary ligation, and by the end of the 2nd hour of the experiment it had fallen from 1.36-1.14, or 84% of its initial value. A decrease in the IMP/LV ratio in the apical region of the left ventricle was observed in 7 to 11 experiments after coronary ligation, it remained essentially unchanged in 3, and increased in 1 experiment. The causes of the increase in the IMP/LV ratio at the base and apex of the left ventricle were different. At the base it always increased because the IMP increased while the intraventricular pressure was unchanged or fell slightly, but at the apex the increase in the ratio was always caused by a sharp fall in the intraventricular pressure, while the IMP remained unchanged (in 2 experiments) or actually fell (in 1 experiment).

In more than half of the experiments ligation of the coronary artery was thus accompanied by an increase in the contractile function of the undamaged myocardium at the base and in the middle third of the left ventricle, but as a rule this reaction did not take place in the apical region.

The available evidence is insufficient to explain these differences. As yet only certain views may be expressed. It can be considered very schematically that the myocardium of the ventricle wall consists of three layers. In the outer and inner layers the fibers are oriented mainly longitudinally, while in the intermediate layer they lie transversely relative to the axis of the heart [4]. The circular compressor muscle described by Keith, situated in the basal and middle portions of the ventricle wall, consists chiefly of transverse fibers. It may be supposed that after ligation of the descending branch of the left coronary artery, resulting in the development of ischemia of a certain part of the lateral wall of the left ventricle, an apparent break occurs in the longitudinal fibers whereas most of the circular fibers are above the area of the lesion and stay intact. At the base of the heart, where the circular fibers of Keith's muscle remain undamaged, the conditions exist for producing compensatory hyperfunction of the myocardium. Conversely, the "break" of part of the fibers interferes with or completely prevents an increase in the contractile function of the myocardium of the apical region, where longitudinal fibers are predominant and the circular fibers are few in number.

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