MONITORING THE STATE OF THE ARRESTED HEART DURING OPERATIONS

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In recent years cardiac surgeons have extensively used pharmacologic cardioplegia as a method of protecting the myocardium during open heart operations. However, it is not clear why so many different formulas for a cardioplegic solution (CS) are necessary – they number more than 50. Reports of unsatisfactory results obtained by the use of this method have also been published [5, 9-12]. What is the reason for this variety of formulas for CS and for differences of opinion on the method of cardioplegia itself? In our view one of the main reasons is the absence of reliable and objective monitoring of the functional state of the patient's heart actually during the operation, of predicting recovery of its functions in the period of reperfusion, and of establishing the precise times when recovery will begin.

It was accordingly decided to develop one such method and to test it under clinical conditions. The method was based on the principle of dynamic recording of resting tension (RT). During anoxia a state of energy deficiency develops, characterized by disturbance of the ability of the mitochondria and sarcoplasmic reticulum to retain Ca^{++} ions [2]. In turn, the appearance of Ca^{++} in the sarcoplasm is accompanied by a rapid response of the myofibrils, expressed as an increase in RT.

EXPERIMENTAL METHOD

Experiments were carried out on 40 rats' and 12 dogs' hearts. For the experiments on the rats' hearts a special apparatus enabling RT of the myocardium in small laboratory animals to be recorded was devised. For this purpose the rat's heart was quickly removed and placed in physiological saline at 16-18°C. The heart was fixed by the aorta and apex to create a tension of 1-2 mm Hg and a value of RT, amplified 10 times, was led out to the recorder. A "Biomedica" (Italy) polygraph and KSP-12 instrument were used as recorders. To monitor the functional state of the preserved heart the isotonic contractility of a strip of myocardium was determined with the aid of a strain gauge on an NEK-6 polygraph (East Germany). For work with the dog's heart, strain gauge transducers developed in the Laboratory of Organ Conservation (Head, Candidate of Medical Sciences N. A. Onishchenko) of the writers' Institute were used. Changes in the value of RT were judged from deformation of a spring element mounted into the body of the transducer. The amount of deformation, directly proportional to the value of RT, was recorded by means of the TsTM-5 digital strain-gauge bridge and an "Iskra" digital analyzer. The results were subjected to statistical analysis. The clinical part of the investigation was conducted in the Department of Reconstructive Surgery of Acquired Heart Defects (Head, Dr. Med. Sci. M. L. Semenovskii) of the writers' Institute, on 11 patients undergoing operations for aortic, mitral, and tricuspid valve replacement. The heart was disconnected from the systems of the general circulation for 55-120 min.

CS of the following composition was used: K^+ 28-30 mM, Na^+ 100-110 mM, Ca^{++} 2.8-3.6 mM, Mg^{++} 4-6 mM, glucose 250-500 mg/100 ml, osmolarity 300-330 milliosmoles/liter, pH 7.5-7.55, pCO_2 30-40 mm Hg, pO_2 400-500 mm Hg. After thoracotomy and pericardiotomy the strain-gauge transducer described above was fixed to the anterior wall of the right ventricle along the muscle fibers. After restoration of cardiac activity the transducer was removed.

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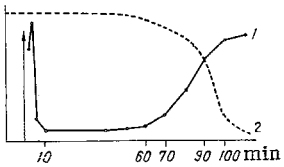

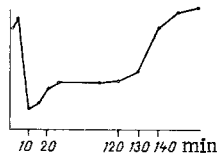

Fig. 1 Fig. 2

Fig. 1. RT of rat myocardium during period of cardioplegia (1) and myocardial contractility after reoxygenation (2).

Fig. 2. RT of dog's myocardium during period of cardioplegia.

EXPERIMENTAL RESULTS

In experiments on the rat heart the following kinetics of RT of the myocardium was obtained (Fig. 1). The value of RT rose sharply in response to injection of CS into the coronary system. After 1-2 min the heart muscle relaxed and the curve recorded flattened out on a long, isotonic plateau. Starting from the 60th-70th minutes of cardiac arrest an increase in RT of the myocardium, smooth initially, but more sudden later, was observed going beyond the high tonic plateau by the 90th and 100th minutes of anoxia. Myocardial contractility at the same time fell sharply after reoxygenation of the heart (Fig. 1).

An identical RT curve (Fig. 2) was obtained from the dog's heart during work; the only difference was that residual tension was present during the period of the isotonic plateau.

The curve obtained from the human heart during work was similar to the kinetics of RT described above. A typical curve is given in Fig. 3. Since during operations on the human heart repeated injections of CS were given, the following reactions were noted: during reperfusion the level of RT either rose sharply, similarly to the response of the myocardium to the first injection of CS, or it fell to the level of the isotonic plateau.

The general kinetics of RT obtained in all cases can be interpreted as follows: in response to injection of CS with a high potassium concentration into the coronary circulation sudden depolarization of the cytoplasm of the cardiomyocyte membranes takes place. This leads to outflow of Ca^{++} from the depots into the sarcoplasm [4], and this is accompanied by a sharp increase in RT. Subsequent relaxation of the myocardium, arising 1-2 min after cardiac arrest, is evidence of reabsorption of Ca^{++} into the depots or into the extracellular space. It is important to note that Ca^{++} reabsorption is an energy-requiring process [6], i.e., utilizing the already limited reserves of energy-yielding substrates. At the same time, stopping the cardiac contractions saves about 80% of the energy contained in the substrates [7]. Although a certain part of this energy is utilized during primary ionic redistribution, it is sufficient to maintain the myocardium in the relaxed state for a long time. This is the period of the isotonic plateau. During this period, as Fig. 1 shows, myocardial contractility remained virtually at its initial level.

Later an increase in RT was observed, conjecturally due to the outflow of Ca^{++} into the sarcoplasm on account of deficiency of the energy resources of the anoxic myocardium [2]. However, as results obtained with the model of a strip of myocardium show, these changes were reversible (Fig. 1).

The next stage in the dynamics of the myocardial RT was the stage of formation of ischemic contracture of the heart, accompanied by a sudden reduction or complete loss of cardiac function – irreversible changes.

Unfortunately, we cannot yet explain the causes of development of residual RT in experiments on the dog's heart during the isotonic plateau stage. This problem requires further study, which will undoubtedly make its role clear.

As already noted, the dynamics of RT of the heart during operation, as recorded under clinical conditions, was identical with the results of the experimental investigations (Fig. 3), which means that some idea may be obtained of the functional state of the patient's heart during the operation and considerable steps have been taken toward the development of a method of cardioplegia. In particular, further infusions of CS can be given depending on individual indications and on the level of RT, for this problem has already been widely discussed in the literature [1, 3, 8].

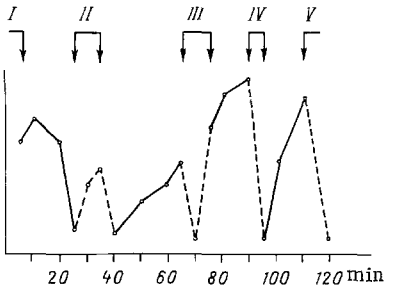

Fig. 3. RT of myocardium of patient N., a man aged 45 years, during operation under carioplegia. I) First injection of CS. Broken line – response of myocardium to repeated injection of solution (II, III, IV) and reperfusion (V).

At this stage of the investigation we were not concerned with modifying the method of preserving the heart, but simply with testing a method of monitoring cardiac function and of keeping this parameter under observation during standard conduct of the operation. As a result the following important shortcomings of the cardioplegia method were identified. The repeated washings out of the coronary circulation with CS has both favorable and extremely undesirable results. In particular, if the period of reinfusion coincides with the isotonic plateau stage, this will be accompanied by repeated outflow of Ca^{++} into the sarcoplasm and considerable energy expenditure by the heart will be required for its reabsorption (Fig. 3, the period between 25 and 40 min), which may be extremely harmful to the anoxic myocardium and evidently accelerates the process of development of ischemic contracture.

Meanwhile, carrying out repeated infusions during the period of an increase in RT (Fig. 3, periods between 65 and 80 and between 90 and 100 min) creates very favorable conditions for the heart during operation, supplying the myocardium with oxygen and, by activating processes of oxidative phosphorylation, ensuring sufficient energy formation for removing the excess of calcium ions from the cytoplasm, and so preventing the early development of ischemic contracture.

It must be pointed out that the use of repeated infusions simply to maintain the temperature regime of the heart during operation is extremely undesirable, for the protective effect of hypothermia cannot completely compensate the harmful effect of repeated ionic redistribution caused by these infusions. In this situation it is better to use surface or intraventricular irrigation of the heart.

Special attention is directed to the fact that monitoring the temperature of the arrested heart is extremely essential for the maintenance of its temperature regime. But this cannot be the sole criterion for determining the times of reinfusions, for it does not reflect the state of cardiac function, and can only give a somewhat remote idea of the intensity of metabolic processes taking place in the myocardium.

The method of recording RT of the noncontracting heart thus enables its function to be assessed objectively, restoration of its function after reoxygenation to be predicted, and the need for additional infusions of CS and the times of these infusions during the operation to be determined. Further research in this direction will lead to improvements in the technique of conduct of cardioplegic protection of the myocardium.

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