PATHOLOGICAL PHYSIOLOGY AND GENERAL PATHOLOGY

TEMPERATURE GRADIENTS OF THE HEART AND PULSE RATE IN COLD CARDIOPLEGIA

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Exclusion of the heart from the circulation in hypothermic conditions has gained general acceptance as a method of creating a bloodless operation field. However, this method cannot be used when combined defects of the heart valves require correction. A more practicable method in these conditions is that of cold cardioplegia [1, 2], based on the assumption that the metabolism of the arrested heart is close to zero. This method, although rapidly depressing the metabolism of the myocardium, does not give rise to profound changes in structure and function characteristic of chemical and anoxic cardioplegia.

The clinical application of the method of hypthermia has gone far ahead of experimental work to establish its basic principles. This is true above all of the temperature conditions of the heart and the pulse rate as indices determining the onset of asystole. For this reason these parameters were studied during local hypothermia of the heart.

EXPERIMENTAL METHOD

Experiments were carried out on 23 mongrel dogs aged 3-7 years and weighing 8-21 kg. The animals were anesthetized initially by injection of 5% thiopental-sodium solution, and later with ether and oxygen. The detectors of an electrothermometer constructed in the author's laboratory were introduced into the chamber of the heart. The brain was cooled in a craniocerebral refrigeration unit working on liquid freon-12. The revolving power of the apparatus was 26°.

After the brain had been cooled to 29-27°, the pericardium was opened widely and taken up on holding forceps. After 1.5-2 min of hyperventilation, the posterior vena cava was compressed by a tape previously passed beneath it and fixed by a tourniquet, and 30-50 sec later the procedure was repeated on the anterior vena cava. The pericardial cavity was filled with a mixture of pieces of melting ice made from sterila physiological saline. The heart was warmed with towels soaked in warm physiological saline. The activity of the heart was restored by means of direct massage and electrical stimulation.

EXPERIMENTAL RESULTS AND DISCUSSION

During cooling with a solution at a temperature of 3-5°, the temperature of the heart fell slowly and reached 17-60° only after 10-11 min. Considerable temperature gradients were observed between the atria and ventricles, particularly in the first period of cooling. At 21-19° a period of stabilization of the ventricular temperature was observed, and by the 20th minute the temperature of both portions of the heart was the same. Subsequently, the temperature differences did not exceed 1°.

The same pattern of changes in heart temperature was observed when it was incompletely separated from the circulation (part of the venous drainage was preserved) and the myocardium was incompletely isolated from the thoracic organs. However, in these cases equalization of the temperatures of the base and apex began with the onset of mechanical asystole. It follows that absence of the conditions necessary to produce adequate cooling of the heart led to a slow decrease in its temperature with the development of large gradients between the atria and ventricles.

In later experiments melting ice was used to cool the heart because its heat capacity is 81 times greater than that of water at 1° [2]. In these experiments the changes in the temperature of the heart were of a different character. In the first minutes of cooling it fell quickly, reaching 16° after 2-4 min. After

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Temperature in Various Parts of the Heart in an Experiment with Selective Cooling of the Myocardium (Experiment No. 383, December 15, 1964)

Part of the heart	Temperature						
	before exclusion of heart	minutes					
		exclusion			recovery		
		3	12	30	1	4	11
Right atrium	26,9	16,0	11,6	17,5	22,8	23,0	25,0
Right ventricle	26,5	16,4	11,2	16,9	17,7	20,2	25,4
Left atrium	26,8	15,8	11,7	17,4	17,8	20,2	23,5
Left ventricle	27,0	16,2	11,4	17,1	18,4	26,0	23,4

the temperature had reached a certain minimum, the rate of cooling was slightly reduced, leading to a small increase in temperature followed by stabilization.

In these conditions the temperature difference between the various parts of the heart was much smaller. It was greatest in the first minute of local cooling, although here also the temperature of atria was always higher. The highest gradient in this period was 4.5°, but next minute it fell to 1.3° (experiment No. 405, April 16, 1965). No significant temperature differences were found between the right and left sides of the heart, if the cooling agent surrounding all the heart uniformly. The gradients developing in this case did not exceed 0.4-0.6° (see table).

Dependence of the rate of fall of temperature on the pulse rate was seen even more clearly in this series of experiments. In the course of cold cardioplegia the temperature in the esophagus fell at the rate of 1-1.5°/30 min. When local cooling of the myocardium was stopped, its temperature rose at the rate of about 0.2-0.4°/min. Warming the heart with warm towels led to a rapid increase of its temperature. In most experiments restoration of the temperature took place faster than it fell during induction of cardioplegia. In this period the temperature of the ventricles was slightly higher than that of the atria.

The character of the pulse changes during cold cardioplegia was largely determined by the intensity of cooling and by the previous functional state of the myocardium and circulation. The principal factor determining this index was the rate of fall of the heart temperature. If it fell rapidly the pulse rate also fell sharply and cardiac arrest supervened quickly. I consider that the basis for the rapid onset of asystole is a fall of temperature at the rate of 6-8°/min. If the temperature fell slowly, cardiac arrest developed after a long period of time. In this case the initial sharp fall in the pulse rate was followed by stabilization (20-10 beats/min). In such experiments the rate of fall of the myocardial temperature was 1.5-4°/min. In all experiments a marked fall of pulse rate (on the average to 40% of its initial value) was observed in the initial cooling period. Later the pulse rate changes followed three possible courses: a continued sharp decrease parallel to the change of temperature (in this case cardiac arrest occurred after 2-3 min), a steady fall in the pulse rate with cardiac arrest after 4-5 min, and stabilization (and sometimes a slight increase) of the pulse rate followed by a steady decrease (in this variant cardiac arrest occurred after 7 min).

Asystole was preceded by fibrillation only in the last variant of cardioplegia. In some cases it arose during mechanical stimulation of the myocardium as it was cooled to 20-17°. Cardiac arrest took place at a temperature of 17°, but most commonly at 14-12°.

LITERATURE CITED

1. A. M. Malygin, Functional State of the Myocardium in Hypothermia [in Russian], Candidate Dissertation, Vladimir (1963).
2. H. G. Urschel, J. J. Greenberg, and C. A. Hufnagel, New Engl. J. Med., 261, 330 (1959).