

CHANGES IN THE STRUCTURE OF LEFT VENTRICULAR
SYSTOLE DURING COOLING

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In modern cardiology the method of phase analysis of the cardiac cycle is being used increasingly for the assessment of cardiac function [1, 2, 4]. This method, by determining the relationships between the various periods of the cycle, enables the contractile ability of the myocardium to be assessed [5, 8].

Although the action of hypothermia on the body and, in particular, on the heart has been widely investigated, insufficient attention has been paid to the study of changes in the structure of left ventricular systole under the influence of cooling. The comparatively few papers on this subject are incomplete and contain contradictory information [10, 11].

EXPERIMENTAL

In 17 experiments the effect of cooling on the structure of left ventricular systole was studied in a heart-lung preparation (HLP) of dogs, thereby excluding thermoregulatory changes in the activity of the heart during cooling, which take place during the development of hypothermia in an animal. The volume of blood reaching the preparation from the venous reservoir was maintained at a constant level by a special dosing device [7], the "venous" pressure being kept constant. To analyze the cycle of cardiac contraction, synchronized recordings were made of the ECG and of the pressure inside the left ventricle and aorta, measured by a type EM-2-01 electromanometer (Hungary), on a type N-106 oscillograph. The catheters were introduced into the left ventricle and the orifice of the aorta. The minute volume was also recorded by P. M. Starkov's method [6], the pressure in the left atrium was measured, and the mean dynamic pressure in the aorta was calculated by I. M. Sechenov's method. The preparation was cooled to 20°.

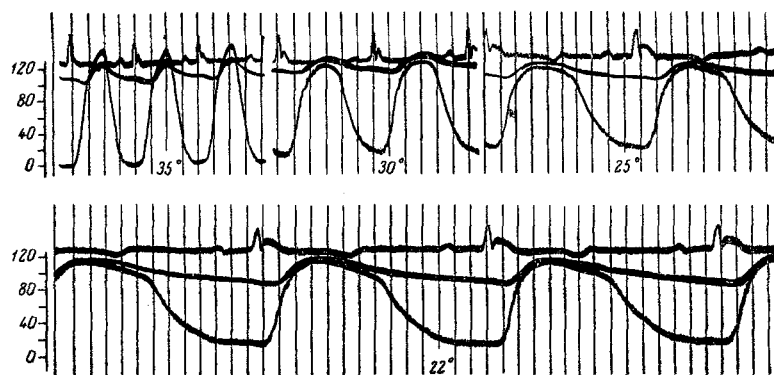
The period of concentration of the myocardium was determined from the interval between the Q wave on the ECG and the beginning of the rise of pressure in the aorta. The duration of asynchronous contraction was measured from the Q wave to the beginning of the sharp rise of pressure within the ventricle. The period of isometric contraction was measured from the beginning of the rise of pressure in the left ventricle to the beginning of the rise of pressure in the aorta, and the ejection period from the beginning of the rise of the intra-aortic pressure curve to the beginning of the incisura on this curve. The duration of total and mechanical systole and various calculated indices were also determined: the mechanical coefficient, defined as the ratio between the total time of contraction and the ejection time; the intrasy-

TABLE 1. Changes in the Duration of the Phases of Cardiac Cycle during Cooling ($M \pm m$)

	37—35°	31—29°	26—24°	22—20°
Duration of cycle	0,40±0,015	0,69±0,023	1,18±0,075	1,67±0,10
Total systole	0,26±0,01	0,44±0,02	0,66±0,03	0,82±0,05
Mechanical systole	0,20±0,009	0,36±0,018	0,55±0,028	0,69±0,047
Total period	0,14±0,004	0,18±0,005	0,21±0,013	0,20±0,015
Asynchronous contraction	0,06±0,002	0,08±0,003	0,11±0,005	0,13±0,006
Isometric contraction	0,08±0,003	0,10±0,005	0,10±0,006	0,07±0,012
Ejection	0,12±0,006	0,26±0,014	0,45±0,031	0,62±0,047
Diastole	0,14±0,019	0,25±0,033	0,52±0,065	0,85±0,079

tolic index, defined as the ratio between the ejection time and the length of mechanical systole (in percent); the rate of increase of the intraventricular pressure during isometric contraction, defined as the ratio between the difference between diastolic pressures in the aorta and ventricle and the duration of isometric contraction; the volume velocity of ejection defined as the ratio between the systolic volume and the duration of ejection. The work during systole was calculated by multiplying the difference between the mean pressure in the aorta and left atrium (in cm H₂O) by the systolic volume (in mm).

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Changes in the ECT (top curve) in the intra-aortic pressure (2nd curve from the top), and pressure within the left ventricle (bottom curve) during cooling.

TABLE 2. Changes in Certain Indices of Left Ventricular Systole during Cooling ($M \pm m$)

Index	37—35°	31—29°	26—24°	22—20°
Mechanical coefficient	$0,85 \pm 0,06$	$1,44 \pm 0,06$	$2,14 \pm 0,17$	$3,10 \pm 0,48$
Intrasystolic index	$60 \pm 2,0$	$73 \pm 0,7$	$81 \pm 1,0$	$89 \pm 1,0$
Rate of increase of intraventricular pressure (in mm Hg/sec)	$1168 \pm 68,9$	$860 \pm 54,4$	$739 \pm 53,4$	$792 \pm 96,8$
Volume velocity of ejection (in ml/sec)	$17,09 \pm 2,17$	$14,23 \pm 1,50$	$11,95 \pm 1,14$	$8,65 \pm 0,65$

EXPERIMENTAL RESULTS AND DISCUSSION

As the temperature fell, the duration of the cycle increased progressively (Table 1, figure). As the total duration of the cycle increased, so also did the duration of both systole and diastole of the left ventricle. This was accompanied by a marked internal reorganization of systole; a comparatively small increase in the period of contraction and a much larger increase in the ejection time were observed. The increase in the ejection time remained statistically significant ($P < 0.001$)* until 25°. With a further decrease in the temperature, the period of contraction no longer continued to increase, because the period of isometric contraction was shortened. The decrease in the period of isometric contraction at temperatures below 25° was associated with a fall in the mean intra-aortic pressure from 104 ($m \pm 3.94$) mm at 37–35° to 66 ($m \pm 4.8$) mm at 22–20°.

When the changes in the contractile power of the heart under the influence of cooling were assessed on the basis of the results of phase analysis of left ventricular systole, two at first sight contradictory tendencies were noticed. On the one hand, the ejection time increased, leading to an increase in the mechanical coefficient and in the intrasystolic index (Table 2). This increase in the duration of ejection, together with the change in the ratio between the ejection time and the total period of contraction, and also between the ejection time and the duration of mechanical systole is generally taken to be a sign of improvement in the working capacity of the heart and of an increase in the efficiency of cardiac contraction [3–5]. On the other hand, the rate of increase of the intraventricular pressure fell during the period of isometric contraction and the volume velocity of ejection of blood also diminished (Table 2). These facts demonstrate a reduction in the contractile power of the myocardium under the influence of cooling.

Calculation of the work done by the heart during systole showed that it amounted to 31.19 ($m \pm 4.66$) g/cm at the initial temperature, 45.36 ($m \pm 5.79$) g/cm at 31–29°, 48.97 ($m \pm 6.02$) g/cm at 26–24°, and 48.29 ($m \pm 6.60$) g/cm at 22–20°.

*The value of P was calculated by the method of direct differences.

Consequently, a considerable increase in the work of the heart was observed down to 30° ($P < 0.001$). Other investigators have reported an increase in the systolic work during cooling. On this basis, the view has been expressed in the Western literature that moderate hypothermia improves the working capacity of the isolated heart. In a survey, for instance, Badeer [9] reaches the following conclusion: hypothermia improves the contractile power of the myocardium; the total working capacity of the isolated heart is increased during moderate hypothermia. I consider that this conclusion is not justified.

As a result of hypothermia, the automatism of the heart is depressed, leading to slowing of the heart rate. The duration of systole is especially increased. If the inflow is constant, this effect is accompanied by considerable filling of the heart with blood and by an increase in the stroke volume in accordance with Starling's Law. This explains the increase in the ejection time and the increase in the ejection time and the increase in the systolic work. The Starling mechanism masks the decrease in the contractile power of the myocardium developing as the temperature falls to 30°.

The processes of automatic regulation of the cardiac activity thus compensates, up to a certain limit, the decrease in the contractile power of the myocardium taking place as a result of the direct effect of temperature on the metabolic processes.

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