Myocardial Function in Severe Hypothermia

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The functional status of the myocardium in hypothermia was investigated by means of pulse contours. No evidence for myocardial failure was uncovered on the basis of established criteria. Artificial cardiac acceleration proved to be a dangerous procedure in the hypothermic heart.

GENERALIZED hypothermia, as employed in cardiac surgery,1–3 involves a number of factors, some of which are of benefit, others detrimental to myocardial performance. For example, the decreased arterial pressure and the increased blood viscosity tend to reduce coronary blood flow. On the other hand, the reduced metabolic rate of heart muscle, the diminished cardiac work, and the lengthened time for unimpeded coronary inflow tend to benefit the myocardium.

In the present study, the over-all effect of these various factors on myocardial function has been evaluated by pulse contour analysis. Despite the sluggish heart action and the low arterial pressure which exist in hypothermia, no evidence for a significant degree of impairment of the myocardium has been found.

METHODS

Six experiments were performed on dogs weighing between 15 and 25 Kg. The animals were anesthetized with sodium pentobarbital (30 milligrams per kilogram). The chest was opened at the third left intercostal space and respiration maintained artificially. Aortic, left ventricular, and left atrial pressures were registered by modified Gregg manometers. A Leeds and Northrup Micromax instrument was used to record temperatures in the right atrium and the rectum. Observations were made during a control period and following immersion of approximately two-thirds of the dog's body surface in ice water (no water or ice were permitted to enter the chest cavity). Cold narcosis obviated the use of supplementary anesthetic. The blood cell/plasma ratio was determined periodically and kept relatively constant during the experiments by the judicious intravenous administration of 0.9 per cent sodium chloride solution.

RESULTS

In all experiments, aortic and left ventricular pressures were recorded, and in four of these left atrial pressure was also registered. The changes observed were quite similar in each experiment; the results of a representative experiment are shown in figure 1.* With the exception of record C, which depicts the effect of vagal stimulation at a cardiac temperature of 33.5 C., records A through H show progressive changes in aortic, left ventricular, and left atrial pressures as heart blood temperature decreased from 40 C. to 20.5 C. The heart rate decreased from 158 to 13 beats per minute. The duration of systole increased from 0.18 to 1.00 second, and that of diastole from 0.20 to 3.50 seconds, resulting in a decrease of the systole/cycle ratio from 0.47 to 0.22.

Analysis of the ventricular pressure curve reveals, that in contrast to the normothermic animal (record A) in which the rise and fall of tension in the ventricle were abrupt and approximately equal in duration, hypothermia produced a less abrupt increase and a greatly prolonged decrease in tension. Comparison of records A and H discloses that the duration of isometric contraction increased about twofold (0.06 to 0.10 seconds), whereas the duration of isometric relaxation was prolonged thirteenthfold (0.04 to 0.52 second). During moderate hypothermia (fig. 1, B through E) ventricular relaxation continued until the succeeding systole, as shown by the progressive decline in intraventricular pressure throughout diastole. Only as the heart slowed markedly, as with vagal

* It should be noted that the paper speed in records G and H is half that in records A through F.
stimulation (fig. 1 C) or due to severe cold (fig. 1, F through H), was the diastolic filling accompanied by the slight rise of pressure characteristic of normal curves. These changes were not accompanied by a significant increase of initial tension.

The aortic pressure curve shows that a relatively brisk rise in pressure occurred in the later stages of hypothermia. However, the last part of rapid ejection and the entire period of reduced ejection were considerably lengthened at the lower temperatures. At normal temperatures (record A) the aortic standing wave can be observed as pre- and posticusural humps. However, as systole increased in duration with cooling, the standing wave occurred progressively earlier in the cardiac cycle and can be clearly identified in the ventricular and aortic pressure curves. The low frequency of the standing wave at reduced temperatures was due in part to a decrease in arterial pressure.

As hypothermia increased in severity, left atrial pressure became slightly elevated, atrial systole prolonged, and A-V conduction appreciably delayed.

It should be noted that many of the adventitious vibrations seen in the pressure curves at normal temperature disappeared with cooling when contractions became slower and less vigorous. The posticusural vibrations in the aortic pressure curve, produced in normothermia by the snapping shut of the aortic

Fig. 1. Effect of hypothermia on ventricular (upper curve), aortic (middle curve with incisura), and left atrial (lower curve) pressure pulses in the same dog. A, control heart blood temperature 40 C; B, 33.5 C; C, 33.5 C. with peripheral right vagus stimulation; D, 27.5 C; E, 26 C; F, 23 C; G, 22 C; H, 20.5 C. Ventricular and aortic pressures in millimeters Hg. Left atrial pressure in millimeters saline. Time scale records A through F, 0.02 second; records G and H, 0.20 second.
valves, gradually disappeared at low temperatures as the time occupied by valve closure lengthened. The ratio of the duration of systole plus isometric relaxation to total cycle length, in mild hypothermia, increased slightly from 0.53 (record A) to 0.67 (record B). The value then remained relatively constant until a temperature of 23°C was reached (record F). Further cooling decreased the ratio sharply to 0.46 (record G) and 0.34 (record H). Similar ratio changes were previously reported by Hegnauer and colleagues.4

Direct effects of cold on myocardial contractility can only be appraised by comparing the events of the cardiac cycle at equal heart rates during normothermia and hypothermia. To make this comparison, the hearts at nearly normal temperatures were slowed by vagal

Fig. 2. Effect of progressive increase in heart rate by electrical stimulation of the auricular appendage during severe hypothermia (blood temperature 20°C). From top to bottom in each record, ventricular pressure, aortic pressure (with incisura), and left atrial pressure. Ventricular and aortic pressures in millimeters Hg. Left atrial pressure in millimeters saline. Time scale, 0.20 second.
stimulation (record C, fig. 1) and cold hearts were accelerated by artificial atrial stimulation. It is apparent that only minimal increases in the duration of systole and isometric relaxation occurred with vagal slowing in normothermia. The increase in atrial pressure and initial tension and the widening of the pulse pressure are, of course, characteristic findings in vagal bradycardia.

The effect of artificial atrial stimulation in the hypothermic heart is illustrated in figure 2. Records A through D depict the changes which occurred at a heart blood temperature of 20 C. when the rate of stimulation was successively increased. In record A, before stimulation, the heart rate was 16 beats per minute. An increase in rate to 34 beats per minute (B) resulted in a slight decrease in left atrial pressure, an increase in aortic systolic and diastolic pressures, and a decrease in pulse pressure. A heart rate of 43 beats per minute (C) elevated atrial pressure and reduced aortic pressures toward control levels; pulse pressure was slightly decreased. Further cardiac acceleration to 57 beats per minute (D) brought about an increase in atrial and ventricular diastolic pressures (initial tension) and a sharp fall in aortic and pulse pressures.

In records A and B of figure 2, the end of isometric relaxation is clearly indicated by the decline in the left atrial pressure tracing when the mitral valve opened and left ventricular filling began. As can be seen in record C, however, atrial systole ensued before any such fall in pressure occurred, thus making it difficult to define accurately the point of opening of the A-V valves. However, atrial systole probably took place at the point where the A-V valves opened, since the record shows (a) that it occurred as a pressure rise on the descending limb of the ventricular pressure curve and (b) that auricular pressure just exceeded ventricular pressure at the onset of atrial systole. In record D, on the other hand, at a rate of 57 beats per minute, atrial contraction occurred early in the isometric relaxation phase and contributed little if at all to ventricular filling. Despite the elevation of left atrial pressure and the increased vigor of atrial contraction, it is only at the peak of atrial systole that the effect is noted in the ventricular pressure curve. Early in atrial systole ventricular pressure exceeded left auricular pressure. The fact that there was a rise in atrial pressure associated with a fall in aortic pressure is evidence that at this heart rate ventricular filling-time is inadequate and the contribution of atrial systole is eliminated.

In severe hypothermia, with intrinsic cardiac rhythm (fig. 2 A), the slight rise in intraventricular pressure which occurred with ventricular filling indicates that ventricular relaxation was complete before the onset of the next systole. However, with atrial stimulation (fig. 2, B through D), complete ventricular relaxation was not attained between beats since intraventricular pressure continued to decrease throughout diastole.

The increase in rate in the stimulated hypothermic heart occurred chiefly at the expense of total diastole which shortened from 2.86 seconds to 0.50 second (the systole/cycle ratio was approximately doubled). The cardiac acceleration was associated with a slight increase in the period of isometric contraction and a significant reduction in the duration of isometric relaxation.

**DISCUSSION**

These experiments demonstrate that the arterial pressure decline in severe hypothermia is not a manifestation of myocardial failure. Evidence in support of this view is that good pressure contours are maintained even with extreme degrees of cooling (fig. 1), and that the contours do not show any of the characteristics of circulatory failure.

Prec and co-workers have shown that the decrease in arterial pressure in hypothermia is primarily due to a reduction of cardiac output. The bradycardia is in part responsible for this diminished output, but these authors also observed some reduction in stroke volume. The latter they attributed to an impaired venous return, secondary to fluid loss from the vascular bed, rather than to cardiac failure.

In our studies, estimation of stroke volume based on reduced amplitude of ventricular and aortic pressure pulses is limited because of the significant prolongation of ejection. However,
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an analysis of the pressure curves presented does warrant the definite conclusion that myocardial failure does not supervene in hypothermia. Cardiac failure produced experimentally by the use of primary myocardial depressants is also characterized by a prolongation of isometric contraction and a decrease in amplitude, but the phase of ejection is abbreviated and initial tension is elevated. By contrast the period of ejection is increased more than fivefold in hypothermia and initial tension is not raised appreciably.

There is no evidence that cardiac muscle becomes hypoxic with cooling. First, Penrod found that the ability of the hypothermic heart to extract oxygen from arterial blood (despite the shift of the oxyhemoglobin dissociation curve to the left) is unimpaired, an observation we have been able to confirm. Second, a ninefold increase in the rate of blood flow in the cannulated circumflex branch of the left coronary artery (induced by artificial elevation of perfusion pressure) produced no detectable change in the aortic pressure curves (unpublished observation).

The reduction in both systolic and diastolic as well as pulse pressures consequent to artificial acceleration of the hypothermic heart indicates that the capacity of the normal heart to respond with greater output to moderate increases in rate is reversed; output is obviously decreased at rates which are considered slow at normal body temperature. The primary reason for this response is apparently the extended period of isometric relaxation that obtains in hypothermia. In the normothermic dog, isometric relaxation is very brief, whereas in the hypothermic animal it constitutes a significant portion of diastole. This in itself does not interfere with ventricular filling, since the heart rate is extremely slow during hypothermia. However, if the rate is moderately increased by artificial stimulation of the atrium, the extended period of isometric relaxation assumes major importance, since total diastole is reduced to a greater extent than is isometric relaxation. Thus, ventricular filling-time is shortened and atrial contraction does not contribute to ventricular filling, since atrial systole occurs while the A-V valves are still closed. A second important factor responsible for the decrease in arterial and pulse pressures during artificial acceleration of the cooled heart may be that coronary blood flow is reduced as a result of the proportionately long period of extravascular compression. This, however, requires experimental confirmation. The practical import of these findings is that acceleration of the heart in severe hypothermia is contraindicated.

The actual cause of death in hypothermia cannot be determined from our experimental data since we employed artificial respiration as well as cannulas and catheters which have been shown to induce arrhythmias culminating in ventricular fibrillation at temperatures which the uncatheterized dog can tolerate.

SUMMARY

Progressive generalized hypothermia was produced experimentally in the anesthetized open-chested dog by immersion in ice water. Pressure pulses recorded from the left ventricle, the left atrium, and the aorta revealed that even in severe hypothermia the heart did not exhibit typical signs of myocardial failure. The striking changes induced by hypothermia were the marked prolongation of systole and of isometric relaxation. At low temperatures, increases in heart rate from approximately 10 to 60 beats per minute produced by atrial stimulation seriously interfered with cardiac filling. This was in large part due to the fact that isometric relaxation consumed a major portion of diastole which considerably reduced ventricular filling-time and eliminated the atrial contribution to cardiac filling. Our observations support the contention that hypothermia, as employed in cardiac surgery, does not seriously interfere with myocardial competence, provided its natural slow rhythm is maintained.

REFERENCES

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