Hemodynamic and Metabolic Effects of Hypothermia and Extracorporeal Circulation in Experimental Myocardial Infarction and Shock

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In myocardial infarction with shock not responding to conventional therapy, the circulation is inadequate to meet the body's oxygen needs at normal temperature. Therefore, it might be advantageous to utilize hypothermia to reduce the oxygen requirement in such individuals.

There have been some data reported with this technique in experimental myocardial infarction, but there has been little experience with its use in human subjects with acute myocardial infarction. Several investigators have concluded that hypothermia is contraindicated in recent myocardial infarction because of a high incidence of ventricular fibrillation in dogs. However, in all of these experiments, a coronary vessel was ligated after the animal was hypothermic, under which circumstances the heart is irritable, particularly if manipulated. We have attempted to study this problem in a more applicable experimental setting, as far as human myocardial infarction is concerned, in which myocardial infarction with shock was first produced by plastic sphere coronary embolization, a procedure which does not require manipulation of the heart, following which hypothermia was induced.

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Methods

Myocardial infarction with shock was produced in 32 dogs anesthetized with 30 mg./Kg. of pentobarbital, injected intravenously. A previously reported modification of Agress' technique was used in which 2 to 3 mg./Kg. of plastic microspheres (325 μ in diameter) were injected under pressure through a thin-walled aortographic catheter into the coronary arteries via the ascending aorta during transient cardiac arrest produced by 0.4 mg./Kg. of acetylcholine injected rapidly intravenously. The aorta was reached in a retrograde direction, via a femoral artery, the catheter being advanced into the left ventricle with continuous monitoring of pressure. The catheter was then withdrawn from the ventricle until an aortic pressure pulse was recorded and the injection made with the catheter at this point. Thirty minutes following embolization, the animals were heparinized with 2 mg./Kg. of heparin injected intravenously, and hypothermia was induced and maintained by one of two methods.

Twenty dogs underwent blood stream cooling to the point of cardiac arrest or ventricular fibrillation and, in 8 of these, there was further cooling to profound levels of 5 to 10 C. esophageal temperature. The animals were maintained by means of extracorporeal circulation at the lowest temperatures reached for two to three hours, following which the blood was rewarmed. In 19 of these 20 animals, blood was withdrawn through Teflon tubing by gravity from the superior vena cava via the external jugular vein and, when there was circulatory arrest, from the inferior vena cava via a femoral vein as well. It was passed through a Sigmanotor pump, a prepared bubble oxygenator with helix (Abbott Laboratories, Pulmopak), and then a Brown-Harrison heat exchanger, and returned through a Bardic catheter to the abdominal aorta via a femoral artery. The total dura-
HYPOTHERMIA AND MYOCARDIAL INFARCTION

...tion of the cooling and rewarming processes occupied five to six hours. Extracorporeal circulatory flow ranged from 20 to 30 cc./Kg./min., the lower ranges being employed at temperatures below 20°C. Circulation was maintained even at profoundly hypothermic levels, during which time there was prolonged ventricular fibrillation or arrest. Oxygen flow was 2 L./min. Positive pressure artificial respiration with room air via an endotracheal tube was employed when spontaneous respirations ceased, usually at about 25°C. In one animal, venovenous cooling was employed. Blood was withdrawn by gravity from the superior vena cava via a jugular vein and returned after passage through the Sigmamotor pump and the heat exchanger to the inferior vena cava via a femoral vein.

A second group of 12 dogs was studied by a similar technique of veno-arterial blood-stream cooling in which a moderate degree of hypothermia of 28 to 30°C, ordinarily well tolerated by human subjects, was maintained for four to six hours, following which the animals were rewarmed.

A control group of six anesthetized dogs was studied at normal body temperatures for four to six hours following coronary embolization.

Of the total of 38 dogs, the chest was kept closed in all except 9 hypothermic animals in which left ventricular contractile force and left ventricular pressure were measured. In these dogs, left thoracotomy was performed at the beginning of the procedure, prior to hypothermia. Positive pressure artificial respiration with room air via an endotracheal tube was maintained while the chest was open.

Cardiac output, central aortic pressure, right atrial pressure, and, in the 9 open-chest animals, left ventricular contractile force, and left ventricular pressure were determined in the control state (after anesthesia), 30 minutes following coronary embolization at normal esophageal temperature, and then at esophageal temperatures of 30, 25, 20, and immediately after rewarining to 37°C. In these animals maintained at 28 to 30°C, these parameters were measured at hourly intervals during hypothermia and again, following rewarining.

Arterial pH, CO₂, oxygen, potassium, plasma hemoglobin, hematocrit, and central venous (right atrial) oxygen were determined at the same temperature as the esophagus by means of the Cambridge pH Research model meter. Plasma hemoglobin was measured in a Coleman spectrophotometer and plasma potassium in a Baird-Atomic flame photometer.

Esophageal temperature was measured approximately at left atrial level by a thermistor probe and a Tele-thermometer.

In those animals in which defibrillation was performed, an Electrodyne external or internal defibrillator was utilized.

Results

TEMPERATURE AT VENTRICULAR FIBRILLATION OR ARREST (FIGURE 1)

Of 20 animals with acute coronary embolization and shock brought to the level of hypothermic ventricular fibrillation or arrest (asystole, with cessation of electrocardiographic evidence of ventricular activity) by blood stream cooling, average esophageal temperature at the time of fibrillation was 19.3°C and at the time of complete asystole, 12.0°C. Effective contractions ceased at about 17.0°C, in the animals which subsequently developed complete asystole at 12.0°C, although there were electrocardiographic ventricular complexes and weak contractions between 17.0 and 12.0°C in these animals. The animal with venovenous cooling demonstrated ven-
Ventricular fibrillation at 21.0°C. All with ventricular fibrillation were successfully defibrillated on rewarming to 37.0°C, including 8 animals brought to profound hypothermic levels of 5 to 10°C. Of 12 animals maintained by blood-stream cooling at 28 to 30°C for four to five hours, there was no arrhythmia. Of the total of 32 animals with acute coronary embolization reaching 28 to 30°C, there was no instance of ventricular fibrillation at these temperatures. Figure 1 demonstrates the temperatures at which ventricular fibrillation or complete asystole occurred. As in normal animals, fibrillation occurred at somewhat higher temperatures than did arrest. The highest temperature at which ventricular fibrillation occurred in this group was 23.5°C. It was felt that ventricular fibrillation in the hypothermic animals was attributable to the hypothermia and not coronary embolization itself, as our previous experience with this technique demonstrated that when ventricular fibrillation did occur following coronary embolization, this took place within 30 minutes of embolization. Hypothermia in these experiments was not initiated until 30 minutes following coronary embolization.

HEMODYNAMIC ALTERATIONS
Animals Cooled to Levels of Circulatory Arrest or to Profound Hypothermia

Table 1 lists the hemodynamic data obtained in this group and table 2 the data in embolized animals remaining normothermic.

There was an expected fall in cardiac output after embolization and a further fall as temperature was lowered. After rewarming, there was return towards, but not quite to, the pre-embolic levels. In contrast, the cardiac outputs of embolized animals, studied for similar periods while normothermic, showed a slight progressive fall. There was a fall in stroke volume following embolization, but little further fall during hypothermia. Stroke volume returned to pre-embolic levels following rewarming, whereas in the normothermic embolized animals, it remained depressed after five to six hours. Differences in initial (control) cardiac output and aortic pressure between animals remaining normothermic and those subsequently rendered hypothermic may be attributed to the fact that there were several open-chest animals among the two hypothermic groups, which may account for initially lower cardiac outputs and aortic pressures in these animals. When the animals remaining normothermic were compared with closed-chest hypothermic animals, control values for cardiac output and aortic pressure were similar.

Mean central aortic pressure fell after embolization, but, due to an increase in systemic vascular resistance, showed little further fall to 30 mm Hg, after which progressive decline occurred. A mean pressure of 25 to 30 mm Hg was maintained by the extracorporeal circulation during cardiac arrest or ventricular fibrillation at profoundly hypothermic levels. Following rewarming, there was return towards pre-embolic levels, whereas the central aortic pressures of the normothermic group showed little change from the post-embolic level after four to five hours. There was no appreciable rise in either right atrial or left ventricular diastolic pressures in either normothermic or hypothermic animals to suggest the presence of congestive heart failure.
TABLE 1

Hemodynamic Alterations Occurring Prior To and Following Plastic Sphere Coronary Embolization in Dogs Cooled by Veno-arterial Shunting to the Level of Ventricular Fibrillation or Arrest or to Profoundly Hypothermic Levels and then Rewarmed

<table>
<thead>
<tr>
<th></th>
<th>Number of dogs</th>
<th>37°C (pre-embolic)</th>
<th>27°C (30 minutes post-embolic)</th>
<th>30°C</th>
<th>25°C</th>
<th>20°C</th>
<th>5 to 10°C (cardiac arrest or ventricular fibrillation in 5 dogs)</th>
<th>37°C (rewarm)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cardiac output (L./min.)</td>
<td>12</td>
<td>2.06</td>
<td>1.28</td>
<td>0.865</td>
<td>0.556</td>
<td>0.432</td>
<td>5.65</td>
<td>7.5</td>
</tr>
<tr>
<td>cc./Kg./min.</td>
<td></td>
<td>(4.2)</td>
<td>(2.2)</td>
<td>(4)</td>
<td>(4)</td>
<td>(4)</td>
<td></td>
<td>(1.2)</td>
</tr>
<tr>
<td>Heart rate (per minute)</td>
<td>12</td>
<td>118</td>
<td>123</td>
<td>72</td>
<td>58</td>
<td>46</td>
<td>100</td>
<td>15.6</td>
</tr>
<tr>
<td></td>
<td></td>
<td>(1.9)</td>
<td>(4.8)</td>
<td>(1)</td>
<td>(3)</td>
<td>(3)</td>
<td></td>
<td>(1)</td>
</tr>
<tr>
<td>Stroke volume (cc./beat)</td>
<td>12</td>
<td>15.4</td>
<td>10.4</td>
<td>12.3</td>
<td>9</td>
<td>9</td>
<td>9</td>
<td>86</td>
</tr>
<tr>
<td></td>
<td></td>
<td>(8)</td>
<td>(1)</td>
<td>(1)</td>
<td>(2)</td>
<td>(2)</td>
<td></td>
<td>(1)</td>
</tr>
<tr>
<td>Mean central aortic pressure (mm. Hg)</td>
<td>19</td>
<td>105</td>
<td>59</td>
<td>63</td>
<td>50</td>
<td>42</td>
<td>86</td>
<td>25-30 (15)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>(13)</td>
<td>(9)</td>
<td>(8)</td>
<td>(11)</td>
<td>(13)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Left ventricular diastolic pressure (mm. Hg)</td>
<td>6</td>
<td>5</td>
<td>5</td>
<td>5</td>
<td>5</td>
<td>5</td>
<td>5</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>(1.7)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Right atrial pressure (mm. Hg)</td>
<td>10</td>
<td></td>
<td>+1.5</td>
<td>+1.5</td>
<td>+1.5</td>
<td>+1.5</td>
<td>+1.5</td>
<td>+1.5</td>
</tr>
<tr>
<td></td>
<td></td>
<td>(0.6)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Systemic vascular resistance (dynes sec./cm.²)</td>
<td>12</td>
<td>3922</td>
<td>4313</td>
<td>5827</td>
<td>7434</td>
<td>7604</td>
<td>4200</td>
<td>(140)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>(107)</td>
<td>(208)</td>
<td>(90)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Left ventricular work (Kg.-M./min.)</td>
<td>12</td>
<td>3.61</td>
<td>1.07</td>
<td>0.62</td>
<td>0.39</td>
<td>0.27</td>
<td>1.87</td>
<td></td>
</tr>
<tr>
<td>Kg.-M./min./Kg.</td>
<td></td>
<td>(0.087)</td>
<td>(0.025)</td>
<td>(0.002)</td>
<td>(0.002)</td>
<td>(0.002)</td>
<td></td>
<td>(0.024)</td>
</tr>
<tr>
<td>Stroke work</td>
<td>0.026</td>
<td>0.009</td>
<td>0.009</td>
<td>0.009</td>
<td>0.006</td>
<td>0.02</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Left ventricular contractile force (% change from pre-embolic level)</td>
<td>4</td>
<td>-39</td>
<td>-20</td>
<td>-7</td>
<td>-43</td>
<td>41</td>
<td>41</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>(5)</td>
<td>(4)</td>
<td></td>
<td></td>
<td>(2)</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

*Average values are expressed with the standard error indicated in parentheses.

Systemic vascular resistance increased slightly following embolization and then progressively during hypothermia to 20°C, returning to pre-embolic levels after rewarming. There was a lesser, slight, persistent rise in the normothermic animals.

Left ventricular work showed a progressive fall during hypothermia.

Left ventricular contractile force fell following coronary embolization, then increased to 25°C. At 25°C and below, contractile force diminished, but at 25°C, it was still higher than the postembolic level. There was return to control levels following rewarming.

Animals Maintained at Moderate Levels of Hypothermia

In animals maintained for four to five hours at moderate levels of hypothermia (table 3), cardiac output fell as expected at 28 to 30°C, returning to control levels following rewarming. Stroke volume declined following embolization, remained constant during...
TABLE 2

Hemodynamic Alterations in Dogs Remaining Normothermic Following Plastic Sphere Coronary Embolization*

<table>
<thead>
<tr>
<th>Esophageal temperature (°C.)</th>
<th>37°C (pre-embolic)</th>
<th>37°C (post-embolic)</th>
<th>37°C</th>
<th>37°C</th>
<th>37°C</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hours</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Number of dogs</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Cardiac output (L./min.)</td>
<td>2.30</td>
<td>1.72</td>
<td>1.57</td>
<td>1.41</td>
<td>1.44</td>
</tr>
<tr>
<td>e.e./Kg./min.</td>
<td>161 (17)</td>
<td>99 (14)</td>
<td>90</td>
<td>81</td>
<td>83</td>
</tr>
<tr>
<td>Heart rate (per minute)</td>
<td>164</td>
<td>149</td>
<td>145</td>
<td>169</td>
<td>164</td>
</tr>
<tr>
<td>Stroke volume (e.e./beat)</td>
<td>11.5 (1)</td>
<td>11.5 (1.9)</td>
<td>11.3</td>
<td>(2.5)</td>
<td>(0.7)</td>
</tr>
<tr>
<td>Mean central aortic pressure (mm. Hg)</td>
<td>128 (6)</td>
<td>90 (8)</td>
<td>94 (4)</td>
<td>91 (2)</td>
<td></td>
</tr>
<tr>
<td>Left ventricular diastolic pressure (mm. Hg)</td>
<td>2 (range = 2.5)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Right atrial mean pressure (mm. Hg)</td>
<td>2 (1.1)</td>
<td>2 (range = -2 to +5)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Systemic vascular resistance (dynes sec./cm.²)</td>
<td>6 (237)</td>
<td>4130 (688)</td>
<td>4637 (642)</td>
<td>4888 (657)</td>
<td></td>
</tr>
<tr>
<td>Left ventricular work (Kg.-M./min.)</td>
<td>6 (5.5)</td>
<td>2.19 (0.942)</td>
<td>2.11 (0.012)</td>
<td>1.90 (0.001)</td>
<td></td>
</tr>
<tr>
<td>Kg.-M./min./Kg.</td>
<td>0.026 (0.042)</td>
<td>0.126 (0.012)</td>
<td>0.121 (0.002)</td>
<td>0.019 (0.001)</td>
<td></td>
</tr>
<tr>
<td>Stroke work</td>
<td>0.031 (0.013)</td>
<td>0.013 (0.014)</td>
<td>0.014 (0.001)</td>
<td>0.012 (0.001)</td>
<td></td>
</tr>
</tbody>
</table>

*Average values are expressed with the standard error indicated in parentheses.

hypothermia, and returned to control levels after rewarming.

There was a rise in systemic vascular resistance in the postembolic hypothermic state, which resulted in slight rise in aortic pressure during hypothermia, despite the fall in cardiac output, resistance returning to control levels following rewarming.

Left ventricular work declined during hypothermia. Left ventricular contractile force showed the expected fall following embolization and then rose during hypothermia. There was then a slight fall after rewarming.

The hemodynamic alterations from the control state occurring during hypothermia in this group remained constant during the period of prolonged, moderate hypothermia. There was more adequate return of cardiac output and aortic pressure towards control levels following rewarming in these animals than in those brought to deeper hypothermia, to the level of circulatory arrest.

METABOLIC DATA

There was evidence of metabolic acidosis in the more profoundly cooled dogs, more marked at the lower temperatures, the pH
TABLE 3

<table>
<thead>
<tr>
<th>Esophageal temperature (C.)</th>
<th>37 (pre-embolic)</th>
<th>37 (post-embolic)</th>
<th>28 to 30</th>
<th>28 to 30</th>
<th>37 (rewarm)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hours</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Number of dogs</td>
<td>9</td>
<td>1/2</td>
<td>2</td>
<td>3</td>
<td>5 to 6</td>
</tr>
</tbody>
</table>

Cardiac output (L./min.)
- 9
- 77 (7)
- 154 (2)
- 11 (1.5)
- 9 (13)
- 2 (3)
- 4 (0.5)
- 9 (178)
- 9 (2.61)
- 0.118 (0.023)
- 0.018
- 5

Heart rate (per minute)
- 36 (5)
- 100 (4)
- 7.96 (1.5)
- 75 (5)
- 3 (1.5)
- 115 (6)
- 10^(2.5) (range = 1 to -2.5)

Mean central aortic pressure (mm. Hg)
- 0.706 (5)
- 11 (5)
- 75 (5)
- 1.8 (4)
- 11 (6)
- 3 (1.5)
- 7 (1.5)
- 55 (5)

Right atrial mean pressure (mm. Hg)
- 0.817 (6)
- 115 (7)
- 135 (13)
- 104 (11)

Systemic vascular resistance (dynes sec./cm."^2")
- 5262 (566)
- 8928 (517)
- 8887 (280)
- 5114 (272)

Left ventricular diastolic pressure (mm. Hg)
- 0.970 (2)
- 37 (5)
- 115 (5)
- 135 (13)
- 104 (11)

Left ventricular contractile force (% change from pre-embolic level)
- 1/2
- 2
- 3
- 3
- 3
- 1
- 1
- 1
- 1

*Average values are expressed with the standard error indicated in parentheses.

Discussion

From the data obtained, it appears that an animal with acute myocardial infarction and shock is not more susceptible to fatal hypothermic arrhythmia than is a normal animal. Plasma hemoglobin increased after five hours of pumping, to an average of 160 mg. per cent in the more profoundly cooled dogs and to 90 mg. per cent in the dogs maintained at 28 to 30 C.*

*Tables containing the metabolic data in more detail will be supplied by the authors on request.
animal. This conclusion is based on previously reported studies of body temperature at the time of ventricular fibrillation or arrest in blood-stream or skin-cooled normal dogs. Therefore, temperature levels which are generally considered "safe" for normal animals without circulatory support are probably equally safe for those with recent myocardial infarction. Different conclusions from previous investigations may be attributed to the fact that coronary ligation was produced after hypothermic levels were reached during which time the heart is irritable, particularly if manipulated. It has been demonstrated in the hypothermic dog that surgically induced ventricular fibrillation may occur at higher body temperature than spontaneous ventricular fibrillation. The two investigations of which we are aware, in which cooling was produced subsequent to myocardial infarction, but more remote from the onset of the infarct than in our experiments, demonstrated that cooling of dogs to 25°C three to five days or two weeks subsequent to coronary ligation was well tolerated.

It also appears that there is relatively little immediate irreversible deleterious hemodynamic effect after five to six hours of profound or moderate hypothermia combined with low-flow extracorporeal circulation. There is evidence, however, that hemodynamic recovery is more adequate following moderate hypothermia than after similar periods of profound hypothermia. The dogs cooled to deeper levels of hypothermia demonstrated less return of cardiac output and central aortic pressure after rewarming than did dogs maintained at moderately hypothermic levels for similar periods, the cardiac output remaining about 25 per cent, and the aortic pressure 15 per cent, below control values in the more deeply cooled animals.

In addition, metabolic acidosis was evident in the profoundly cooled dogs and was not present in the dogs maintained at moderate temperatures. Whether the depression in the cardiac output following rewarming from profound levels was related to intrinsic myocardial depression attributable to the effects of cold injury, to loculation of fluid during deeper hypothermia diminishing the venous return, to slower rewarming than in the animals maintained at 28 to 30°C, to acidosis, or to the deleterious effects of prolonged extracorporeal circulation cannot be stated from the available data. The latter is unlikely as a sole explanation since similar periods of extracorporeal circulation at similar flow rates were employed in the moderately cooled animals, which did not demonstrate postwarming depression in cardiac output. Loculation of plasma in the periphery, as has been reported in normal animals made hypothermic, also seems unlikely as the principal cause of this variation between the two groups as the stroke volume in both returned to the control state following rewarming.

In both the profoundly cooled and the moderately cooled animals, there is suggestive evidence from our data that hypothermia may exert some protective effect in recent myocardial infarction, inasmuch as, following rewarming, there was more adequate recovery towards the preinfarction levels of coronary perfusion pressure, cardiac output, and systemic vascular resistance than in animals remaining normothermic for similar periods following coronary embolization.

The factors responsible for this "protective" effect have not been fully elucidated. The reduction in tissue oxygen requirement should be of benefit in any situation in which the circulation is inadequate at normal temperature. In addition to reduction in oxygen requirement, there are more specific effects of hypothermia on the cardiovascular system which may be beneficial in shock following myocardial infarction. Among these effects are reduction in cardiac work and a rise in systemic vascular resistance, by which mechanism coronary perfusion pressure is increased. It is also possible that some of the "protective" effect of hypothermia may be due to coronary artery dilatation which has been demonstrated in normal dogs at temperatures below 28°C.
In the normal animal, hypothermia increases the force of ventricular contraction. Increases (to 25 C.) were also noted in our animals with acute myocardial ischemia, but the degree of increase in left ventricular contractile force was considerably less than at similar temperatures in the previously reported studies in normal dogs. This is probably attributable to the depression in contractile force caused by the myocardial ischemia although metabolic acidosis, as was present in some of our animals, will also cause depression of myocardial contractile force.

If the procedure can be tolerated, there is experimental background from the data reported here for the belief that benefit might be derived from hypothermia in acute myocardial infarction with shock. However, our studies were acute experiments and no statement is justified as to the effects of hypothermia on ultimate survival in acute myocardial infarction or the feasibility of its application in critically ill human subjects.

Summary

The hemodynamic and metabolic effects of combined extracorporeal circulation and hypothermia induced by veno-arterial shunting were determined in 32 dogs following the production of acute myocardial infarction with shock by plastic sphere coronary embolization. Cardiac output, central aortic pressure, left ventricular work, systemic vascular resistance, left ventricular contractile force, right atrial and left ventricular pressures, arterial pH, CO₂, potassium, plasma hemoglobin, and hematocrit were determined in dogs brought to the level of ventricular fibrillation or arrest or to profoundly hypothermic levels of 5 to 10 C. esophageal temperature for four to six hours and then rewarmed; in those maintained at 28 to 30 C. esophageal temperature for four to six hours and then rewarmed; and in those remaining normothermic for four to six hours following coronary embolization. The average esophageal temperature at the time of ventricular fibrillation was 19.3 C., and at the time of complete asystole was 12.0 C., figures comparable to results in normal animals. It is concluded that an animal with acute myocardial infarction and shock is not more susceptible to fatal hypothermic arrhythmia than is a normal animal. It was also demonstrated that there was relatively little irreversible deleterious hemodynamic effect after five to six hours of profound or moderate hypothermia combined with low-flow extracorporeal circulation, although the more profoundly cooled animals did demonstrate a metabolic acidosis and less return to precooling levels of cardiac output and aortic pressure following rewarming than did those maintained at 28 to 30 C. Both the profoundly and moderately cooled animals demonstrated, following rewarming, more adequate recovery of cardiac output, aortic pressure, and systemic vascular resistance than did animals which remained normothermic for similar periods after coronary embolization, suggesting that hypothermia affords a "protective" effect in recent myocardial infarction.

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References


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