Hypothermic Arrest and Potassium Arrest

METABOLIC AND MYOCARDIAL PROTECTION DURING ELECTIVE CARDIAC ARREST

By David J. Hearse, David A. Stewart, and Mark V. Braimbridge

ABSTRACT

Hypothermic arrest, potassium arrest, and ischemic arrest, either singly or in combination, with or without coronary perfusion were studied in an isolated perfused rat heart preparation. Procedures that permitted the maintenance of high cellular levels of adenosine triphosphate (ATP) and creatine phosphate during arrest, e.g., coronary perfusion with hypothermic solutions or solutions containing 16.0 mM potassium, produced a fully reversible arrest with complete cardiac recovery. Cardiac arrest and coronary flow were related to the degree of hypothermia and the concentration of potassium in the coronary perfusate, and the minimum conditions required to induce complete cardiac arrest were ascertained. The effects of hypothermia and potassium were additive; total cardiac arrest could be obtained by combining small elevations of potassium with moderate hypothermia. Under these conditions, cellular high-energy phosphates were maintained, and complete recovery was possible. Under conditions in which arrest was obtained without maintaining coronary perfusion, e.g., ischemic arrest, cellular high-energy phosphates declined rapidly, and the hearts exhibited poor recoveries. Some protection could be afforded to the ischemic myocardium by topical hypothermia or by combining the ischemia with potassium arrest. In both instances, ATP and creatine phosphate were maintained at higher levels, and improved recoveries were observed.

KEY WORDS

cardiotoplegia creatine phosphate cardiac bypass adenosine triphosphate isolated working rat heart coronary perfusion ischemic arrest

Accurate cardiac surgery demands a still and relaxed heart on which to operate. Historically, cardiac arrest (cardioplegia) was first induced by the injection of high concentrations of potassium citrate into the coronary circulation prior to the termination of coronary perfusion (1). Because reports of subsequent myocardial damage appeared (2-5), potassium citrate arrest was discontinued. Since then various cardioplegic methods for both experimental and clinical use have been reported (6-11). Currently, ischemic arrest, hypothermic arrest, and electrical fibrillation, either singly or in combination, are in widespread clinical use.

We have recently reported on a series of studies in which we investigated how various cardioplegic methods affect the survival and the recovery of the heart (12). In these studies, using a rat heart model of cardiac bypass and elective cardiac arrest, we found that the functional recovery of the heart following the termination of arrest was related to the concentrations of adenosine triphosphate (ATP) and creatine phosphate in the myocardium at the end of the period of arrest. In turn, these concentrations depended on the method used to induce arrest. Thus, although normothermic ischemic arrest reduced high-energy phosphates and gave poor functional recoveries, coronary perfusion with hypothermic solutions or solutions containing high concentrations of potassium chloride induced arrest without depleting ATP or creatine phosphate and permitted good recoveries.

Ideally, effective cardioplegia should be obtained with the least severe conditions possible, e.g., mild hypothermia or minimum elevation of potassium concentrations. The object of the studies described in this paper was to establish these conditions for potassium arrest and hypothermic arrest in the rat heart. In addition, we attempted to determine whether these cardioplegic procedures were additive and thus, by a suitable combination, to achieve effective cardiac arrest with complete myocardial protection by using very small modifications in the temperature of the myocardium and...
the potassium concentration of the coronary perfusate. As in our previous study, we related these conditions to changes in the myocardial concentration of high-energy phosphates and to the functional recovery of the heart after elective cardiac arrest.

**Methods**

Male rats (280–320 g) of the Sprague-Dawley strain maintained on a standard diet were used in these experiments. All substrates and enzymes used in the analysis of tissue extracts were obtained from the Boehringer Corporation.

**PERFUSION TECHNIQUES**

The perfusion techniques have been described previously (12). Following excision of the heart, the aorta and the left atrium were cannulated. The perfusion circuit (Fig. 1) was designed so that it could be used for two modes of perfusion which could be readily interconverted.

Nonworking Langendorff Preparation.—Hearts were perfused via the aorta as described by Langendorff (13) with a perfusion pressure of 65 cm H₂O. This mode of perfusion was used for an initial washout period and also for periods of simulated bypass in which coronary perfusion was maintained.

Working System.—Hearts were perfused via the left atrium as described by Neely et al. (14) at an atrial perfusion pressure of 20 cm H₂O. The left ventricle spontaneously ejected 40–50 ml perfusate/min against a hydrostatic pressure of 100 cm H₂O. The aortic flow and the coronary flow could be pooled and recirculated. This preparation was used for the control working period prior to elective cardiac arrest and for the recovery period following arrest.

It was therefore possible to simulate cardiac bypass with maintained coronary perfusion by converting the preparation from an atrially perfused working heart to a nonworking Langendorff preparation. During the period of simulated bypass, the heart could be subjected to cardiac arrest.

With the exception of any periods of hypothermic arrest, the hearts were maintained in the normothermic state by perfusion at 37°C. Accurate temperature control was essential in all studies, and the temperature of the perfusate was continuously monitored using a thermistor probe and a telethermometer. In periodic control checks, a microthermistor was introduced into the aortic cannula and also into the heart chamber to confirm adequate temperature control. At all times the heart was kept in a water-jacketed chamber that was maintained at the same temperature as the perfusion fluid and bubbled with the same gas mixture used to aerate the perfusate.

Prior to its introduction into the heart chamber, the gas...
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was bubbled through water that was maintained at the same temperature as the perfusate and the heart chamber. In experiments involving tissue ischemia, the hearts were maintained in a sealed, temperature-controlled heart chamber, and coronary flow was halted by clamping both the atrial and aortic cannulas.

PERFUSION MEDIUM

Krebs-Henseleit bicarbonate buffer (15). pH 7.4, containing glucose (11.1 mM) was the standard perfusion fluid. In studies with potassium-induced arrest, the concentration of potassium in the perfusion fluid was increased and the concentration of sodium was correspondingly decreased. The perfusion fluid was equilibrated with 95% O₂-5% CO₂ (aortic O₂ partial pressure was over 600 mm Hg). Precautions (16) were taken to prevent the precipitation of calcium. Before it was used, the perfusion fluid was filtered through a cellulose acetate filter with 5.0-μm pores.

PERFUSION TIME SEQUENCE

To allow an estimate of recovery and to eliminate errors resulting from variation between individual hearts, a control working perfusion period preceded all periods of cardioplegia. Immediately after mounting, the heart was perfused for a 5-minute washout period by a nonrecirculating Langendorff type of perfusion. The preparation was then converted to a working heart system for a 15-minute period. During this time, the stability of the preparation could be confirmed, and the control values for peak systolic pressure, heart rate (derived electronically from the pressure recordings), aortic flow rate (measured with a flow-through electromagnetic flowmeter in-line with the aorta), and coronary flow rate of the heart were established. As an index of external work, aortic flow rate against a hydrostatic pressure of 100 cm H₂O was monitored. At the end of the control period, the hearts were subjected to a 30-minute experimental period of simulated bypass by converting the preparation from atrial perfusion to Langendorff perfusion. By utilizing separate perfusion fluid reservoirs, the hearts could be arrested by normothermic coronary perfusion with fluid containing high concentrations of potassium or by hypothermic coronary perfusion with fluid from a refrigerated reservoir. At the end of the experimental period, cardioplegia was terminated, and the hearts were converted back to atrially perfused working preparations. The recovery of aortic flow, heart rate, and peak systolic pressure was recorded over a 15-minute period, and recovery values were expressed as percents of the control values.

TISSUE ANALYSIS

In a parallel series of studies, all experiments were repeated except that, instead of allowing the hearts to recover at the end of the period of arrest, the perfusions were terminated by freeze clamping (17) the hearts. The hearts were then analyzed to determine the percent of water, the concentration of ATP, and the concentration of creatine phosphate (12).

Results

NORMOTHERMIC CORONARY PERFUSION WITHOUT CARDIOPLE gia

As a control to all of the other conditions studied, hearts (N = 6) were subjected to 30 minutes of coronary perfusion at 37°C without cardiac arrest. The results in Figure 2 (percent recovery) and Table 1 (absolute recovery) show that when they were converted back to atrially perfused working preparations, all of the hearts recovered to 100% of the control aortic flow in less than 1 minute. The results of tissue analysis (Table 1) show that at the end of the 30-minute experimental period the concentration of ATP in the hearts (N = 8) was 26.2 ± 1.7 μmoles/g dry weight and that of creatine phosphate was 20.5 ± 1.6 μmoles/g dry weight. In a separate series of perfusions, hearts (N = 8 in each case) were perfused either for a 5-minute period (Langendorff preparation) or for a 20-minute period (working preparation) and were then freeze clamped for the determi-
<table>
<thead>
<tr>
<th>Conditions during experimental period</th>
<th>ATP (μmoles/g dry wt)</th>
<th>CP (μmoles/g dry wt)</th>
<th>Heart rate (beats/min)</th>
<th>Control</th>
<th>Recovery</th>
<th>Peak systolic pressure (cm H₂O)</th>
<th>Control</th>
<th>Recovery</th>
<th>Aortic flow rate (ml/min)</th>
<th>Control</th>
<th>Recovery</th>
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<td>t = 10</td>
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<tr>
<td>Normothermic coronary perfusion without cardioplegia</td>
<td>26.2 ± 1.7</td>
<td>20.5 ± 1.6</td>
<td>278</td>
<td>287</td>
<td>284</td>
<td>288</td>
<td>197</td>
<td>184</td>
<td>181</td>
<td>183</td>
<td>40.0</td>
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<td>Normothermic potassium chloride (16 mM) arrest with coronary perfusion</td>
<td>20.8 ± 1.2</td>
<td>35.5 ± 2.6</td>
<td>297</td>
<td>275</td>
<td>275</td>
<td>274</td>
<td>209</td>
<td>187</td>
<td>185</td>
<td>188</td>
<td>41.2</td>
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<td>Hypothermic (4°C) coronary perfusion</td>
<td>20.5 ± 1.0</td>
<td>25.3 ± 1.8</td>
<td>310</td>
<td>304</td>
<td>297</td>
<td>294</td>
<td>214</td>
<td>199</td>
<td>198</td>
<td>200</td>
<td>51.8</td>
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<td>Hypothermic (24°C) coronary perfusion with solutions containing potassium chloride (13 mM)</td>
<td>25.2 ± 0.8</td>
<td>29.6 ± 1.4</td>
<td>258</td>
<td>276</td>
<td>274</td>
<td>274</td>
<td>210</td>
<td>203</td>
<td>205</td>
<td>205</td>
<td>45.5</td>
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<td>Normothermic ischemic arrest</td>
<td>5.3 ± 0.9</td>
<td>2.8 ± 0.4</td>
<td>300</td>
<td>10</td>
<td>45</td>
<td>51</td>
<td>214</td>
<td>115</td>
<td>95</td>
<td>80</td>
<td>46.5</td>
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<td>Hypothermic (21°C) ischemic arrest</td>
<td>20.0 ± 1.0</td>
<td>5.5 ± 0.3</td>
<td>269</td>
<td>213</td>
<td>211</td>
<td>206</td>
<td>216</td>
<td>138</td>
<td>140</td>
<td>149</td>
<td>51.7</td>
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<tr>
<td>Normothermic ischemic arrest with potassium chloride (16 mM)</td>
<td>11.1 ± 4.2</td>
<td>9.4 ± 2.1</td>
<td>298</td>
<td>293</td>
<td>292</td>
<td>286</td>
<td>274</td>
<td>159</td>
<td>169</td>
<td>186</td>
<td>45.5</td>
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<tr>
<td>Normothermic ischemic arrest with potassium citrate (16 mM)</td>
<td>9.8 ± 3.5</td>
<td>9.0 ± 1.9</td>
<td>305</td>
<td>299</td>
<td>297</td>
<td>291</td>
<td>219</td>
<td>141</td>
<td>176</td>
<td>182</td>
<td>44.0</td>
</tr>
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</table>

Mean values ± SE for the myocardial concentrations of adenosine triphosphate (ATP) and creatine phosphate (CP) in hearts (n = 8 for each condition) after 30-minute experimental periods are presented. Heart rate, peak systolic pressure, and aortic flow rate control values were obtained 2 minutes prior to the onset of arrest. The duration of arrest was 30 minutes. Recovery values were obtained 5, 10, and 15 minutes after the termination of arrest. Each value represents the mean for six hearts.
nation of ATP and creatine phosphate. After 5 minutes of perfusion, ATP and creatine phosphate concentrations were 27.2 ± 0.8 and 30.2 ± 1.0 μmoles/g dry weight, respectively. After 20 minutes of perfusion, ATP levels were essentially unchanged (26.5 ± 0.8 μmoles/g dry weight), and creatine phosphate levels had declined to 25.9 ± 0.7 μmoles/g dry weight.

POTASSIUM ARREST WITH NORMOTHERMIC CORONARY PERFUSION AND HYPOTHERMIC ARREST WITH HYPOTHERMIC CORONARY PERFUSION

In agreement with our previous studies (12), we found that hearts subjected to 30 minutes of hypothermic (4°C) coronary perfusion (N = 6) or to perfusion with fluid containing a high concentration (16.0 mM) of potassium chloride (N = 6) were totally arrested but were able to recover completely at the end of the experimental period. In each instance, the recoveries (Fig. 2) reflected the high levels of ATP and creatine phosphate in the heart at the end of the period of arrest (Table 1). In the case of potassium arrest with normothermic coronary perfusion, the level of creatine phosphate (35.5 ± 2.6 μmoles/g dry weight) was greater than that observed in the control perfusions reported in the previous section, suggesting that not only was the fall of creatine phosphate, associated with normothermic perfusion, prevented but also reversed with an apparent synthesis and accumulation of this compound.

Experiments were designed to ascertain the degree of hypothermia or the concentration of potassium chloride required to achieve complete cardiac arrest, while still affording sufficient protection to the myocardium to allow complete recovery. Hearts were subjected either to normothermic coronary perfusion with solutions containing different concentrations (ranging from 6 mM to 48 mM) of potassium chloride or to hypothermic coronary perfusion at different temperatures (ranging from 2°C to 38°C). In both groups, coronary flow rate and heart rate were recorded. Potassium concentration or perfusate temperature was changed in steps using several reservoirs, and at least 5 minutes was allowed for stabilization at each stage before heart rate and coronary flow were recorded. This study was carried out using step increases in temperature (eight hearts) and potassium concentration (eight hearts) and also using step decreases in temperature and potassium concentration (again eight hearts in each case). Overall, there was no significant difference between the ascending and descending curves; therefore, they were combined and are illustrated in Figure 3. It is apparent that heart rate fell off rapidly with increasing potassium concentrations, and at approximately 20 mM complete cardiac arrest was achieved. It is of interest that over this range of 6 mM to 20 mM potassium, coronary flow was essentially constant. However, as the concentration was increased to 48 mM, the coronary flow rate declined markedly. In the hypothermic studies (Fig. 3B), the heart rate fell with temperature, and complete cardiac arrest was possible below 6°C. There was a tendency for the coronary flow rate to decline with temperature.

COMBINED ACTION OF HYPOTHERMIC AND POTASSIUM ARREST

Figure 3 allows one to determine the degree of hypothermia or the concentration of potassium required to achieve a prescribed reduction in heart rate. If the two cardioplegic effects are additive, then the possibility arises that total cardiac arrest can be obtained under relatively mild conditions. This possibility was investigated in a study in which one group of hearts (N = 8) was subjected to normothermic perfusion for 30 minutes with buffer...
containing 11 mM potassium chloride (sufficient to produce a reduction of approximately 33% in heart rate). A second group of hearts (N = 8) was subjected to hypothermic coronary perfusion for 30 minutes at 32°C (also sufficient to produce a reduction of approximately 33% in heart rate). A third group of hearts (N = 8) was subjected to hypothermic perfusion (32°C) with buffer containing 11 mM potassium chloride. The results (Table 2) indicate that the effects were additive with an overall reduction in absolute heart rate of approximately 70%. At the end of the experimental period, each of the three groups of hearts recovered to approximately 100% of the control heart rate. This experiment was repeated with a potassium concentration (13 mM) and a hypothermic temperature (24°C) which individually were sufficient to reduce heart rate by approximately 66%. When combined (Table 2), total cardiac arrest was observed, and on termination of arrest complete recovery of heart rate was found.

As shown previously (Fig. 2), hearts subjected to 30 minutes of arrest by hypothermic (4°C) coronary perfusion or normothermic perfusion with 16.0 mM potassium chloride exhibited good recoveries for aortic flow rate. The recovery of hearts subjected to the less severe condition of combined hypothermic perfusion (24°C) with fluid containing an elevated concentration of potassium chloride (13.0 mM) is also shown in Figure 2. Under these conditions, aortic flow rate recovered to greater than 100% in less than 1 minute. The recovery was greater than that with hypothermic arrest alone; so too were the myocardial levels of ATP and creatine phosphate (Table 1). Unlike potassium arrest alone, the recovery was not maintained at greater than 100%.

The results described so far show that potassium arrest and hypothermic arrest either singly or in combination allow reversible cardiac arrest with complete recovery so long as coronary perfusion is maintained for the duration of the arrest. Under clinical conditions, continuous coronary perfusion is not always possible, and hearts may be subjected to ischemic arrest. We therefore investigated whether the harmful effects of ischemic arrest (12) could be modified by combination with either hypothermic or potassium arrest.

### Table 2

<table>
<thead>
<tr>
<th>Perfusate condition during experimental period</th>
<th>Control period</th>
<th>Experimental period</th>
<th>Recovery period</th>
</tr>
</thead>
<tbody>
<tr>
<td>11 mM Potassium</td>
<td>256 ± 6</td>
<td>165 ± 8</td>
<td>256 ± 7</td>
</tr>
<tr>
<td>32°C</td>
<td>241 ± 6</td>
<td>134 ± 14</td>
<td>243 ± 5</td>
</tr>
<tr>
<td>11 mM Potassium plus 32°C</td>
<td>241 ± 8</td>
<td>68 ± 9</td>
<td>238 ± 7</td>
</tr>
<tr>
<td>13 mM Potassium</td>
<td>248 ± 7</td>
<td>108 ± 11</td>
<td>242 ± 5</td>
</tr>
<tr>
<td>24°C</td>
<td>244 ± 7</td>
<td>97 ± 12</td>
<td>247 ± 7</td>
</tr>
<tr>
<td>13 mM Potassium plus 24°C</td>
<td>244 ± 6</td>
<td>0</td>
<td>239 ± 5</td>
</tr>
</tbody>
</table>

Values are means ± SE. Eight hearts were studied for each condition. Heart rates were measured during the last 5 minutes of each 30-minute period.
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Percent recovery of aortic flow rate in the isolated working rat heart. Solid circles = after 30 minutes of normothermic ischemic arrest, open squares = after 30 minutes of ischemic arrest with topical hypothermia (21°C), solid squares = after 30 minutes of normothermic ischemic arrest in hearts which had been arrested by perfusion with a solution containing potassium chloride (16.0 mM) for 1 minute before the onset of ischemia, and open circles = after 30 minutes of normothermic ischemic arrest in hearts which had been arrested by perfusion with a solution containing potassium citrate (16.0 mM with respect to potassium) for 1 minute before the onset of ischemia. Each point represents the mean for six hearts, and the bars represent the SE.

(N = 6) recovered to 52% after 15 minutes (Fig. 4). Again, as with potassium arrest combined with ischemia, the recoveries, although poor, were substantially better than those with ischemia alone.

It is of interest to compare the preceding results obtained in the rat with the studies carried out in the dog by Berne and his colleagues (18). These workers studied the hemodynamic and metabolic changes in the heart before, during, and after normothermic ischemic arrest and hypothermic ischemic arrest. As found in this paper cooling the ischemic myocardium protected cardiac function, but in contrast it did not preserve myocardial high-energy phosphates.

POTASSIUM CITRATE AND POTASSIUM CHLORIDE ARREST

In relating our findings of complete recovery following cardiac arrest induced by normothermic perfusion with 16 mM potassium chloride or improved recoveries from ischemic arrest when the ischemia is combined with potassium chloride arrest to the reports (2–5) of the damaging effects of potassium, it is important to make several distinctions. First, in many clinical studies coronary perfusion was terminated following the administration of potassium. Under these conditions, tissue ischemia coexists with potassium arrest; it can be difficult to dissociate the metabolic effects of potassium from those of ischemia. Second, very high concentrations of potassium were used, and potassium citrate was used as opposed to potassium chloride. It could be argued that the choice of citrate as the anion could possibly be detrimental to the survival of the myocardium especially under ischemic conditions. Citrate has been shown (19) to inhibit the glycolytic pathway. This pathway, which is stimulated considerably during tissue anoxia, is responsible for the anaerobic production of ATP and as such may contribute significantly to the survival of the tissue. Any inhibition of the activity of this pathway by citrate would reduce anaerobic energy production and possibly exacerbate ischemic damage. In addition, the possibility exists that citrate may chelate calcium and thus impair cardiac function. Experiments were therefore undertaken to ascertain whether citrate has any detectable effect on the recovery after arrest. In a series of experiments, hearts (N = 6 for each group) were subjected to 30 minutes of arrest either by normothermic ischemic arrest following injection of a solution containing 16 mM potassium chloride into the coronary tree or normothermic ischemic arrest following injection of a solution containing potassium citrate (16 mM with respect to potassium) into the coronary tree. In a second series of experiments, the levels of ATP and creatine phosphate in the heart (N = 8 for each group) at the end of arrest were determined. The results (Fig. 4) illustrate that citrate reduced the mean recovery; 15 minutes after the end of arrest the potassium chloride-arrested hearts had recovered to greater than 40% of the control value, whereas the potassium citrate-arrested hearts had recovered to less than 30% of the control value. However, neither this difference nor the values obtained (Table 1) for ATP and creatine phosphate under these conditions were statistically significant (P ≥ 0.1).

Discussion

In this and our previous study (12) we have illustrated how various methods of inducing cardiac arrest can affect the recovery of the heart. Of the cardioplegic methods studied, only those that...
allowed high cellular levels of ATP and creatine phosphate to be maintained during arrest were associated with complete recovery. Continuous perfusion of the coronary system with hypothermic (4°C) solutions or solutions containing high concentrations of potassium chloride (16.0 mM) induced total cardiac arrest and allowed good and rapid recovery.

The apparent protective effects of potassium observed in these and other studies (12, 20–22) may possibly be explained simply in terms of very rapid induction of arrest and consequent conservation of cellular energy supplies. It has however also been suggested (21, 22) from studies of the metabolism of oxygen uptake of the arrested heart that potassium depresses the energy requirement and also the oxygen consumption of the heart. This possibility may explain the observation reported in this paper and also that reported by Lochner et al. (21) that during aerobic potassium arrest the cellular levels of creatine phosphate increase substantially above control values.

Hypothermic coronary perfusion or ischemic arrest with profound (4°C) topical hypothermia induces cardiac arrest that is fully reversible. Hypothermia has the advantage of conferring considerable protection on the myocardium by reducing the metabolic rate (2, 3, 23–25). In our studies, hypothermic coronary perfusion is preferable to topical hypothermia because, in addition to eliminating the element of ischemia, it allows a more rapid and uniform cooling of the heart. Our earlier study (12), which is in agreement with the findings of Bretschneider (26), has shown that in ischemic arrest with topical hypothermia there is a relationship between the recovery of the heart and the duration and the degree of the hypothermia. The shorter the duration of arrest and the deeper the degree of hypothermia, the greater is the recovery.

Elective cardiac arrest should be achieved as rapidly as possible and with the minimum interference to normal metabolic activity while ensuring complete cardiac standstill with sufficient protection to allow full recovery following the termination of arrest. Our results indicate that coronary perfusion with hypothermic solutions, solutions containing potassium chloride, or a combination of these two satisfies these criteria. It is possible to determine the minimum concentration of potassium and the minimum degree of hypothermia required to achieve arrest while still allowing complete recovery. Our results indicate that the actions of hypothermia and potassium are additive and that complete cardiac arrest can be achieved with a relatively low potassium concentration (13 mM) and moderate hypothermia (24°C). Furthermore, under these conditions coronary flow remains essentially unchanged.

Clearly, there are many differences between the model used in this study and the human heart undergoing cardiopulmonary bypass and elective cardiac arrest, and, in addition, the recovery of flow work is not a completely ideal measure of the recovery of ventricular function. However, despite these inadequacies, the results do stress the importance of attempting to obtain maximum metabolic protection during arrest. Ideal conditions for cardiac bypass surgery demand a still and relaxed heart. Cardiac arrest and relaxation can be produced by several methods, each of which has its inherent disadvantages; a balance has to be achieved between maintaining myocardial integrity and producing good operating conditions. Coronary artery perfusion without cardiac arrest preserves myocardial integrity at the cost of poor surgical conditions. Ischemia produces both cardiac arrest and muscular relaxation, but myocardial damage occurs unless the ischemic period is short or the myocardial oxygen consumption is reduced by hypothermia. A heart that is being perfused can be stopped with electrically induced ventricular fibrillation, but this procedure retains cardiac tone and gives rise to the danger of subendocardial ischemia and necrosis (27). Stopping a continuously perfused oxygenated heart in diastole with potassium would appear to offer an ideal operating field with optimum preservation of myocardial integrity. The disadvantages associated with the required high concentrations of postassium can be markedly alleviated by the coincident use of hypothermia. The latter, by virtue of its additive effect, permits a reduction in the concentration of potassium required to induce complete cardiac arrest.

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D J Hearse, D A Stewart and M V Braimbridge

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