Amelioration of the Effects of Ischemic Cardiac Arrest by the Intracoronary Administration of Cardioplegic Solutions

By George J. Todd, M.D., and G. Frank O. Tyers, M.D.

SUMMARY

 Interruption of coronary flow during cardiac surgical procedures provides a bloodless flaccid heart and allows precise and rapid correction of complex cardiac defects. However, myocardial damage occurs in direct proportion to the duration of the ischemia. As the induction of cardioplegia simultaneous with the initiation of cardiac ischemia helps to preserve cardiac energy reserves and thus myocardial integrity, the identification of a consistently reliable cardioplegic technique is desirable. Isolated perfused working rat hearts were made ischemic for one hour by aortic cross-clamping and were compared with hearts rendered cardioplegic at the onset of ischemia by the intracoronary administration of 5 ml of a hypothermic solution: 1) Krebs-Henseleit buffer, 2) Ringer's lactate, 3) tetrodotoxin, 4) potassium chloride, or 5) potassium citrate. Cardiac output, heart rate, aortic pressure and coronary flow were determined pre and post-ischemia. When compared to time-matched controls and hearts arrested with potassium or tetrodotoxin, the ischemia and ischemia-Ringer's lactate groups showed significant post cross-clamp depression of all measured parameters. Intracoronary Ringer's lactate, although often used as an adjunct to ischemic arrest, was not of significant value. In contrast, hearts arrested with tetrodotoxin, potassium chloride or potassium citrate showed no significant post-ischemic functional or histologic deficit. Perfusion with hypothermic Krebs-Henseleit buffer protected the myocardium better than did Ringer's lactate but less well than the tetrodotoxin or isotonic high potassium solutions. The induction of hypothermic metabolic arrest of the heart by briefly perfusing the coronary arteries via the aortic root with isotonic buffered solutions results in markedly improved myocardial tolerance to one hour of ischemia and avoids the problems of low cardiac output and ventricular irritability previously reported with hypertonic potassium citrate arrest.

THE RELATIVE EASE with which complex operations can be accomplished when the heart is both flaccid and unperfused has led many surgeons to induce cardiac arrest by cross-clamping the ascending aorta.1 Although this method of arresting the heart facilitates the performance of cardiac surgical procedures and shortens the duration of cardiopulmonary bypass, the heart is permitted to contract until energy reserves are depleted2 and myocardial damage may result. Because of the sporadic occurrence of irreversible myocardial damage following ischemic arrest and because techniques of coronary perfusion, perfusion with induced ventricular fibrillation and cold irrigation of the pericardial sac are inconvenient and also on occasion unreliable, there is considerable interest in the development of a safe and consistent means of preserving myocardial integrity during an interval of ischemic arrest. The immediate induction of cardioplegia at the onset of ischemia is of considerable benefit in this regard because myocardial energy reserves are spared from the rapid depletion which otherwise follows discontinuation of coronary perfusion.3,4

Despite the fact that nearly 20 years have passed since Melrose reported on potassium citrate-induced cardioplegia,4 the best means of inhibiting myocardial deterioration during induced ischemic arrest remains in question. In this paper we report on our studies of reversible hypothermic and metabolic cardioplegic agents undertaken in an attempt to develop a technique permitting prolonged intervals of ischemic cardiac arrest without functional or histological sequelae.

Methods

Rats of the Sprague-Dawley strain weighing 150–200 gm were fasted overnight before experimental use. Heparin sodium (5 mg/rat) and sodium pentobarbital (30 mg/rat) were injected intraperitoneally and when adequate anesthesia was achieved, the thoracic cavity was entered and the beating heart was removed by means of a scissors cut along its posterior-superior aspect. Upon removal from the chest, the heart was immediately dropped into a beaker containing chilled (10°C) saline. The heart was removed from the saline bath and the aorta was slipped onto the grooved cannula of the working heart apparatus (fig. 1) previously described in detail.5,6 The aorta was secured on the cannula with a silk ligature and retrograde cardiac perfusion was begun at a pressure of 60 mm

From the Department of Surgery, The Pennsylvania State University, College of Medicine, The Milton S. Hershey Medical Center, Hershey, Pennsylvania.

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Address for reprints: G. Frank O. Tyers, M.D., Department of Surgery, Division of Cardiovascular and Thoracic Surgery, The Milton S. Hershey Medical Center, Hershey, Pennsylvania 17033.

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Hg with oxygenated modified Krebs-Henseleit buffer solution warmed to 37°C. The concentrations in mM of the buffer solution constituents were as follows: NaCl 118, KCl 4.7, CaCl2 2.5, MgSO4 1.2, KH2PO4 1.2, CaEDTA 0.5, NaHCO3 25, glucose 5. The perfusate was oxygenated with a 95 O2:5 CO2 mixture which was equilibrated with water at 37°C. By bubbling the gas mixture through the buffer solution, arterial O2 tensions in excess of 500 mm Hg were obtained. The perfusate temperature was maintained at 37°C by means of a water jacket which surrounded all portions of the apparatus. Adequate oxygenation and long-term function of the control preparation have been extensively documented.

After cannulation of the aorta, retrograde perfusion was continued for 15 minutes to allow complete washout of all blood and recovery from the brief anoxic interval associated with removal of the heart from the animal. During this period all coronary venous effluent was discarded and the left atrium was cannulated. At the end of the 15 minute recovery period, the working heart preparation was established by opening the line into the left atrium and clamping the line from the retrograde perfusion reservoir to the aorta. Left atrial filling pressure was maintained constant at 10 cm H2O.

Cardiac output was pumped by the isolated heart to an overflow trap situated 78 cm above the heart where it was returned to the system except once every 15 minutes when it was collected in a graduated cylinder for determination of cardiac output. The height of this column provided the afterload necessary to maintain coronary flow which was also determined every 15 minutes by collecting the perfusate drip-off from the heart in a graduated cylinder. Between flow measurements all perfusate returned to the system for oxygenation and recirculation by a peristaltic pump. A closed bubble trap containing a 4 ml air bubble was situated halfway between the aortic valve and the top of the 78 cm column. This simulated the resiliency of the normal ascending aorta and resulted in a fairly normal pulsatile pressure tracing including a dicrotic notch (fig. 2). Although diastolic pressures are a little low with this technique, coronary flow is excellent and a smaller air bubble reduces the compliance of the system to the point where ventricular damage ensues. Aortic pressure and heart rate were continuously monitored with a recorder and pressure transducer connected into the aortic line.

Control hearts were perfused for 2½ hours but the satisfactory nature of the system has been established by the continued function of a previous set of 12 hearts for up to eight hours without significant deterioration of cardiac function. The experimental hearts were subjected to ischemic arrest by clamping both the atrial inflow and aortic outflow lines and immediately received one of the following treatments:

1) Ischemic group (I) — no further treatment;
2) Ischemia-Ringer's lactate group (I-RL) — intracoronary injection of 5 ml of an isotonic 10°C Ringer's lactate solution, pH 6.68;
3) Ischemia-Krebs-Henseleit group (I-KHB) — intracoronary injection of 5 ml of an isotonic 10°C solution containing the same constituents as the perfusing solution, pH 7.64;
4) Ischemia-Tetrodotoxin group (I-TTX) — intracoronary injection of 5 ml of an 8 µg/ml solution of tetrodotoxin dissolved in isotonic 10°C Krebs-Henseleit buffer, pH 7.70;
5) Ischemia-Potassium Chloride group (I-KCl) — intracoronary injection of 5 ml of a 10°C solution containing the following constituents (in mM): NaCl 118, KCl 25, KH2PO4 1.2, NaHCO3 25. The final solution had a pH of 7.76, an osmolality of 308 mOsm/L and a potassium concentration of 26 mEq/L;
6) Ischemia-Potassium Citrate group (I-KCIT) — intracoronary injection of 5 ml of a 10°C solution containing the following constituents (in mM): NaCl 118, K2Citrate 8.3,

![Figure 1](Isolated working heart apparatus.)

![Figure 2](Typical pulsatile pressure tracing generated by an isolated rat heart after two hours on the working apparatus and one hour of 26 mEq/L potassium arrest.)
AMELIORATION OF MYOCARDIAL ISCHEMIA

The final solution had a pH of 7.69, an osmolarity of 290 mOsm/L and a potassium concentration of 26 mEq/L.

Administration of the arrest solutions was accomplished through a self-sealing injection cap positioned just above the aortic cannula. During perfusion all hearts were maintained at 37°C within a water-jacketed heating chamber. At the time of aortic cross-clamping, the arrested hearts were moved a few centimeters above the heating chamber so that the ischemic arrest hearts drifted down towards room temperature during the 60 minute arrest while the hypothermic perfusion arrest hearts drifted up towards room temperature, both situations closely simulating the clinical setting. The control and each of the six experimental categories contained seven hearts.

After the designated ischemic interval of one hour, all experimental hearts were perfused retrograde through the aortic root with oxygenated buffer for 15 minutes and the buffer was discarded to eliminate all traces of the arresting agent from the system. The working heart preparation was then re-established for 45 minutes following which the hearts were removed from the apparatus and either dried and weighed or fixed in 10% neutral buffered formalin, sectioned and stained with hematoxylin and eosin. Ischemic damage was graded on a scale of 1–4 depending on the percentage of the slide showing degenerative changes. Student’s t-test was used for comparison between groups.

Results

Following the induction of ischemia, untreated hearts continued to contract for up to five minutes. In contrast, each of the cardioplegic solutions injected into the coronary circulation effected an almost immediate halt of all mechanical activity. No difficulties were encountered with ventricular irritability in any arrested heart when coronary perfusion was reinstituted.

Hemodynamic Results

The effects on cardiac output, coronary flow, heart rate, and aortic pressure of uninterrupted control perfusion, ischemia, and ischemia modified by hypothermic arrest solutions are shown in tables 1–4. The abbreviations used were given in the Methods section. In the pre-ischemic period, no statistically significant differences existed among any of the groups with regard to any measured parameter.

During the post-ischemic interval, the I and I-RL groups performed at similar, significantly lower levels than time-matched controls with regard to cardiac output, aortic pressure and coronary flow. On the other hand, no significant deterioration of function was observed when the I-TTX, I-KCl, and I-KCIT groups were compared with time matched controls for these same parameters.

The I-KHB group functioned well in the early post-ischemic period but subsequently deteriorated to the point where cardiac output became significantly less than time-matched controls. While the I-KHB group

<table>
<thead>
<tr>
<th>Table 1</th>
</tr>
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<tbody>
<tr>
<td><strong>Modification of the Effects of Ischemia on Cardiac Output</strong></td>
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<tr>
<td><strong>Duration (min)</strong></td>
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<tr>
<td>30</td>
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<tr>
<td>60</td>
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<td>90</td>
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<tr>
<td>120</td>
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<tr>
<td>150</td>
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<tr>
<td>180</td>
</tr>
</tbody>
</table>

*Mean cardiac output ± standard error expressed in ml/min/gm dry weight.

†Significantly less than ischemic control.

‡Significantly greater than the I-KCl group.

§Significantly greater than the I-KCIT group.

**No significant difference between the I-TTX group and the other three that comprise the group of I-TTX, I-KCl, and I-KCIT. Example — for KCl at 150 min, no significant difference between I-KCl and I-KCIT.
### Table 2
**Modification of the Effects of Ischemia on Coronary Flow* by Administration of Cardioplegic Agents**

<table>
<thead>
<tr>
<th>Duration (min)</th>
<th>Control</th>
<th>Ischemia</th>
<th>I-RL</th>
<th>I-KHB</th>
<th>I-TTX</th>
<th>I-KCl</th>
<th>I-KCIT</th>
</tr>
</thead>
<tbody>
<tr>
<td>30</td>
<td>71.5 ± 4.3</td>
<td>72.9 ± 6.2†</td>
<td>59.4 ± 8.0†</td>
<td>61.0 ± 2.9†</td>
<td>65.8 ± 7.6†</td>
<td>62.5 ± 2.4†</td>
<td>65.7 ± 5.6†</td>
</tr>
<tr>
<td>45</td>
<td>70.0 ± 4.2</td>
<td>Ischemia</td>
<td>Ischemia</td>
<td>Ischemia</td>
<td>Ischemia</td>
<td>Ischemia</td>
<td>Ischemia</td>
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<tr>
<td>60</td>
<td>71.0 ± 5.1</td>
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<td>Ischemia</td>
<td>Ischemia</td>
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<tr>
<td>75</td>
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<tr>
<td>90</td>
<td>70.0 ± 5.2</td>
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<tr>
<td>105</td>
<td>67.0 ± 4.9</td>
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<tr>
<td>120</td>
<td>65.7 ± 4.9</td>
<td>18.4 ± 9.5†, **</td>
<td>22.1 ± 10.5†</td>
<td>51.5 ± 3.5†, §, ††</td>
<td>59.2 ± 5.8†, §, ††</td>
<td>50.4 ± 5.3†, §, ††</td>
<td>58.2 ± 6.6†, §, ††</td>
</tr>
<tr>
<td>135</td>
<td>63.0 ± 6.0</td>
<td>18.1 ± 9.2†, **</td>
<td>20.4 ± 9.6†</td>
<td>41.5 ± 4.9†, §, ††</td>
<td>59.2 ± 5.1†, §, ††</td>
<td>48.3 ± 7.1†, §, ††</td>
<td>54.3 ± 6.2†, §, ††</td>
</tr>
<tr>
<td>150</td>
<td>62.5 ± 6.0</td>
<td>16.9 ± 8.7†, **</td>
<td>18.3 ± 8.6†</td>
<td>41.5 ± 4.9†, §, ††</td>
<td>56.7 ± 4.8†, §, ††</td>
<td>44.2 ± 8.3†, §, ††</td>
<td>45.7 ± 6.4†, §, ††</td>
</tr>
</tbody>
</table>

*Mean coronary flow = standard error expressed in ml/min/gm dry weight.
†Not significantly different from time matched controls.
§Significantly greater than the ischemia group.
**Significantly greater than the I-RL group.
††No significant difference between this parameter and the other three that comprise the group of I-TTX, I-KHB, I-KCl and I-KCIT. Example – for KCl at 150 min, no significant difference between it and I-KCIT, I-TTX and I-KHB at 150 min.

### Table 3
**Modification of the Effects of Ischemia on Cardiac Rate* by Administration of Cardioplegic Agents**

<table>
<thead>
<tr>
<th>Duration (min)</th>
<th>Control</th>
<th>Ischemia</th>
<th>I-RL</th>
<th>I-KHB</th>
<th>I-TTX</th>
<th>I-KCl</th>
<th>I-KCIT</th>
</tr>
</thead>
<tbody>
<tr>
<td>30</td>
<td>274.6 ± 26.7</td>
<td>247.5 ± 18.6†</td>
<td>235.0 ± 18.4†</td>
<td>265.4 ± 22.4†</td>
<td>230.0 ± 13.2†</td>
<td>220.0 ± 16.8†</td>
<td>214.7 ± 10.8†</td>
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<tr>
<td>45</td>
<td>275.6 ± 24.9</td>
<td>Ischemia</td>
<td>Ischemia</td>
<td>Ischemia</td>
<td>Ischemia</td>
<td>Ischemia</td>
<td>Ischemia</td>
</tr>
<tr>
<td>60</td>
<td>275.6 ± 24.9</td>
<td>Ischemia</td>
<td>Ischemia</td>
<td>Ischemia</td>
<td>Ischemia</td>
<td>Ischemia</td>
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<tr>
<td>75</td>
<td>277.6 ± 24.9</td>
<td>Ischemia</td>
<td>Ischemia</td>
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<td>Ischemia</td>
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</tr>
<tr>
<td>90</td>
<td>269.6 ± 25.9</td>
<td>Ischemia</td>
<td>Ischemia</td>
<td>Ischemia</td>
<td>Ischemia</td>
<td>Ischemia</td>
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<tr>
<td>105</td>
<td>271.6 ± 26.5</td>
<td>Langendorff</td>
<td>Langendorff</td>
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<td>Langendorff</td>
<td>Langendorff</td>
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</tr>
<tr>
<td>120</td>
<td>268.6 ± 26.8</td>
<td>108.8 ± 54.1†, **</td>
<td>135.0 ± 60.7†</td>
<td>257.0 ± 15.9†, §, ††</td>
<td>247.5 ± 16.3†, §, ††</td>
<td>221.0 ± 18.8†, **, ††</td>
<td>243.3 ± 13.1†, **, ††</td>
</tr>
<tr>
<td>135</td>
<td>261.0 ± 13.1</td>
<td>106.9 ± 53.7†, **</td>
<td>132.5 ± 59.6†</td>
<td>256.0 ± 19.9†, §, ††</td>
<td>242.5 ± 20.3†, §, ††</td>
<td>228.5 ± 16.3†, §, ††</td>
<td>218.1 ± 11.4†, **, ††</td>
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<tr>
<td>150</td>
<td>261.0 ± 13.1</td>
<td>97.5 ± 48.4†, **</td>
<td>127.5 ± 57.4†</td>
<td>251.0 ± 22.0†, §, ††</td>
<td>235.0 ± 19.2†, §, ††</td>
<td>221.8 ± 16.9†, §, ††</td>
<td>211.4 ± 12.1†, §, ††</td>
</tr>
</tbody>
</table>

*Mean cardiac rate = standard error expressed as beats/min.
†Not significantly different from time matched controls.
§Significantly less than the ischemia group.
**Significantly greater than the ischemia group.
††Not significantly different from the I-RL group.
†††No significant difference between this parameter and the other three that comprise the group of I-TTX, I-KHB, I-KCl and I-KCIT. Example – for KCl at 150 min, no significant difference between it and I-KCIT, I-TTX and I-KHB at 150 min.
Table 4
Modification of the Effects of Ischemia on Aortic Pressure* by Administration of Cardioplegic Agents

<table>
<thead>
<tr>
<th>Duration (min)</th>
<th>Control</th>
<th>Ischemia</th>
<th>I-RL</th>
<th>I-KHB</th>
<th>I-TTX</th>
<th>I-KCl</th>
<th>I-KCIT</th>
</tr>
</thead>
<tbody>
<tr>
<td>30</td>
<td>97.6 ± 7.4</td>
<td>96.0 ± 7.6†</td>
<td>89.8 ± 8.3†</td>
<td>89.4 ± 4.9†</td>
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<td>100.5 ± 12.4†</td>
<td>110.0 ± 9.8†</td>
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<td>36.6 ± 1.8</td>
<td>29.9 ± 2.6</td>
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<td>27.5 ± 4.3</td>
<td>27.5 ± 4.3</td>
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<tr>
<td>37.6 ± 5.9</td>
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<tr>
<td>38.0 ± 1.7</td>
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<td>Ischemia</td>
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<tr>
<td>38.4 ± 2.0</td>
<td>Ischemia</td>
<td>Ischemia</td>
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<tr>
<td>38.4 ± 1.8</td>
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<tr>
<td>90</td>
<td>92.4 ± 5.4</td>
<td>Langendorff</td>
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<tr>
<td>90.8 ± 1.9</td>
<td>27.5 ± 13.4†</td>
<td>35.8 ± 16.4†</td>
<td>81.0 ± 4.4†</td>
<td>82.7 ± 4.8†</td>
<td>99.3 ± 10.2†</td>
<td>107.5 ± 6.7†</td>
<td>107.5 ± 6.7†</td>
</tr>
<tr>
<td>120</td>
<td>38.8 ± 1.4</td>
<td>13.0 ± 6.4</td>
<td>19.3 ± 8.7</td>
<td>37.6 ± 1.7</td>
<td>30.8 ± 1.5</td>
<td>29.0 ± 5.1</td>
<td>27.5 ± 4.1</td>
</tr>
<tr>
<td>135</td>
<td>90.4 ± 5.9</td>
<td>26.8 ± 13.0†</td>
<td>35.5 ± 16.3†</td>
<td>81.0 ± 4.5†</td>
<td>81.3 ± 4.9†</td>
<td>91.5 ± 6.6†</td>
<td>101.0 ± 4.2†</td>
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<tr>
<td>150</td>
<td>39.0 ± 1.8</td>
<td>13.3 ± 6.5</td>
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<td>37.6 ± 1.7</td>
<td>31.3 ± 1.2</td>
<td>31.0 ± 4.8</td>
<td>29.5 ± 3.9</td>
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<tr>
<td>90.0 ± 5.5</td>
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<td>97.5 ± 5.6†</td>
<td>97.5 ± 5.6†</td>
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</tbody>
</table>

*Mean systolic pressure * standard error (mm Hg)/mean diastolic pressure * standard error (mm Hg).
†Peak systolic pressure (PSP) is not significantly different from time matched controls.
‡PSP significantly less than time matched controls.
§PSP significantly greater than the ischemia group.
¶PSP significantly greater than the I-RL group.
**PSP not significantly different from the I-RL group.
††No significant difference between this parameter (PSP) and the other three that comprise the group of I-TTX, I-KHB, I-KCl and I-KCIT. Example — for KCl at 150 min, no significant difference between it and I-KCIT, I-TTX and I-KHB at 150 min.
‡‡PSP is significantly less than that of the I-KCIT group.
was clearly superior to the I or I-RL groups, it did not perform as well in the 60 minute period following the ischemia as did the I-TTX, I-KCl or I-KCIT groups. Significant I-KHB reductions were noted when compared with I-KCIT with regard to aortic pressure late in the post-ischemic period and with regard to cardiac output at one point (135 min) in the post-ischemic period.

While heart rate of the I-KCIT group was less than time-matched controls at two of the post-ischemic determinations this was, in part, related to the slow pre-ischemic rate in the citrate group. No rate differences were seen when the I-KCIT, I-KCl and I-TTX groups were compared.

Histopathological Results

After one hour of normothermic ischemic arrest and one hour of recovery, hematoxylin and eosin stained sections of the left ventricular myocardium were characterized by focal necrosis, myofibrillar degeneration, loss of cross striations and pyknotic nuclei — all slides grade 3 or 4, over 50 or over 75% of cells damaged. In hearts arrested with any of the hypothermic cardioplegic solutions, hematoxylin and eosin sections revealed only mild edema and occasional myofibrillar fragmentation — all slides grade 1, less than 25% of cells damaged. There was no evidence of loss of cross striations. These changes were indistinguishable from those observed in control hearts perfused for 2½ hours.

Discussion

An hour of ischemia was studied because this duration is adequate for the performance of most cardiac operations and is uniformly injurious to the myocardium. Shorter periods of ischemia do not consistently irreversibly damage either the in situ human heart or the isolated working rat heart. Despite statements supporting aortic cross-clamping to induce cardiac arrest by Wesolowski, Nunn, Cooley and others and despite repeated attempts to estimate a safe ischemic period, there exist wide individual variations in myocardial tolerance to ischemia with the result that normothermic ischemic cardiac arrest is an unpredictable technique. In contrast, the present study shows that induced cardiac arrest with tetrodotoxin, potassium chloride or potassium citrate preserved normal cardiac functional and histological integrity after one hour of ischemia. The high potassium solutions depolarized the myocardial cells. Tetrodotoxin, on the other hand, inhibited depolarization of neuronal and cardiac conductive tissue by a selective blockade of sodium ion inflow. While tetrodotoxin and the high potassium solutions exerted their cardioplegic effects through different mechanisms, the end result was similar, preservation of myocardial high energy phosphates and maintenance of myocardial integrity. Tetrodotoxin may prove useful for protection of donor hearts while preparations are being made for cardiac transplantation, but systemic toxicity precludes its use during cardiopulmonary bypass.

The poor results obtained with intracoronary Ringer's lactate administration are significant. Urschel first used this solution for cardiac perfusion hypothermia and reported good results but subsequent studies have yielded conflicting conclusions. In previous studies in the dog on cardiopulmonary bypass, we compared 60 minutes of normothermic ischemic arrest of the myocardium, with cold Ringer's lactate perfusion of the coronary arteries followed by 60 minutes of ischemic arrest. Significant functional and structural preservation were demonstrable with the selective myocardial hypothermia, but pre-arrest status was never maintained and late arrhythmias were common. The results of the present study demonstrate only minimal benefit from intracoronary Ringer's lactate administration during induced ischemic arrest. The lack of acute functional and histological changes in tissues which go on to develop chronic arrhythmias and fibrosis after ischemia are well established, making the greater sensitivity of the isolated working rat heart preparation an asset for the screening of myocardial preservation methods. The 28 mEq/L of lactate and low pH of the Ringer's may contribute to the acidosis of ischemic arrest and thus offset the protective effect of the hypothermia. Nonlactate, metabolically inactive intracoronary hypothermic solutions were of more value in reducing ischemic myocardial damage as evidenced by the results obtained with the I-KHB group but the protective effect was less pronounced than that observed when a metabolic inhibitor and hypothermia were combined.

Melrose first used potassium to induce cardiac arrest in 1955 and promising results were reported by Lam et al. However, difficulties with ventricular fibrillation and ineffective cardiac action were reported by a number of investigators and potassium arrest was subsequently abandoned. Recent studies have indicated that hypertonicity and potassium citrate rather than potassium may have been the injurious factors with Melrose arrest. The present study indicates that there is no detrimental effect on myocardial function when a buffered isotonic solution of either potassium chloride or potassium citrate is injected into the coronaries in a concentration that produces immediate arrest. No difficulties were encountered with arrhythmias in the post-ischemic period following arrest with either potassium solution. Fur-
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eral preclinical studies to carefully assess the possible role of buffered potassium solution-induced ischemic cardiac arrest are indicated.

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