Studies of Contractile Force in Man

The Effects of Myocardial Hypothermia or Coronary Perfusion during Aortic Occlusion

By W. Gerald Austen, M.D.

Here has been considerable difference of opinion regarding the relative merits of cardiac hypothermia versus unilateral or bilateral coronary perfusion as methods of protecting the heart during aortic occlusion. The present experiments in patients were undertaken to determine the degree of myocardial protection effected by these various methods.

Materials and Methods

Forty-five patients undergoing open-heart surgery for acquired aortic valve disease were included in this study. After induction with intravenous thiopental, light anesthesia was maintained with oxygen and halothane. A Walton-Brodie strain-gage arch was sutured to the left ventricle to record myocardial contractile force. Myocardial temperature was monitored with a thermistor probe, and a continuous electrocardiogram was obtained.

Cannulation for open-heart surgery was performed in the usual fashion. Initial control recordings of left ventricular contractile force were obtained at a temperature of 37 C. after a stable perfusion (approximately 60 ml./Kg./min.) had been accomplished. Then aortic occlusion with myocardial hypothermia or coronary perfusion was undertaken and the aortic valve was repaired or replaced. Following release of the aortic clamp, myocardial and body temperature was returned to 37 C. Defibrillation, if necessary, was accomplished at approximately 32 to 34 C. Fifteen minutes following release of the aortic clamp, myocardial and body temperature was returned to 37 C. Defibrillation, if necessary, was accomplished at approximately 32 to 34 C. Fifteen minutes following release of the aortic clamp, left ventricular contractile force was again recorded. Similar studies were performed 30 minutes after release of the aortic clamp. Identical perfusion flows were achieved in each patient before and after aortic occlusion and systemic pressures and resistances were essentially identical during the contractile force determinations. No inotropic drugs were employed before or during the study period.

In 15 patients cardiac hypothermia to 10 C. was employed. This was accomplished by achieving a body perfusion temperature of 28 C., clamping the aorta, and applying ice saline slush to the heart.

From the Department of Surgery, Harvard Medical School, and the General Surgical Services, Massachusetts General Hospital, Boston, Massachusetts.

Supported in part by U. S. Public Health Service Grants HE-06664 (HEFP) and HE-08021.

Figure 1

Cardiopulmonary bypass arrangement with coronary perfusion lines receiving oxygenated blood from the arterial inflow line. The femoral arterial line has a ¾-inch internal diameter and the coronary perfusion lines are ¾-inch internal diameter.

Circulation, Volume XXXII, September 1965
In 30 patients coronary perfusion at 28°C was attempted; in 15 patients only the left coronary was perfused while in 15 both coronary arteries were perfused. The coronary perfusion lines received oxygenated blood from the arterial inflow line (fig. 1). An occlusive pump was employed. The diameters of the various tubings and cannulae were so constructed that when the systemic arterial inflow was 3,000 ml/min., the flow through a coronary line approximated 300 ml/min. (fig. 2). Adequate coronary perfusion was assumed if the electrocardiogram showed continued ventricular contraction or vigorous ventricular fibrillation. If the electrocardiogram showed inadequate perfusion, the coronary cannulae were repositioned. Great care was always taken to assure continued, unobstructed systemic arterial inflow.

Results and Comments

The groups of patients undergoing hypothermia, unilateral left coronary perfusion, and bilateral coronary perfusion were essentially identical as regards severity and type of disease, length of occlusion, and type of aortic valve repair (tables 1 and 2). In addition, it should be pointed out that only two patients in each group had electrocardiographic evidence of significant coronary arterial occlusive disease.

The contractile force results are summarized in table 2. In three instances (one case of unilateral and two cases of bilateral coronary perfusion), coronary perfusion was at-

### Table 1

**Summary of Patient Material**

<table>
<thead>
<tr>
<th>Groups</th>
<th>Total no. patients</th>
<th>Average age (yr.)</th>
<th>No. of pts.</th>
<th>Pts. with aortic stenosis</th>
<th>Average aortic grad. (mm. Hg)</th>
<th>Pts. with aortic regurg.</th>
<th>Operative procedures</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
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<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Hypothermia</td>
<td>15</td>
<td>51</td>
<td>10</td>
<td>85</td>
<td>4</td>
<td>12</td>
<td>2</td>
</tr>
<tr>
<td>Unilateral perfusion</td>
<td>14</td>
<td>50</td>
<td>10</td>
<td>82</td>
<td>4</td>
<td>11</td>
<td>2</td>
</tr>
<tr>
<td>Bilateral perfusion</td>
<td>13</td>
<td>53</td>
<td>9</td>
<td>87</td>
<td>5</td>
<td>13</td>
<td>2</td>
</tr>
</tbody>
</table>

*Circulation, Volume XXXII, September 1965*
Table 2

Changes in Left Ventricular Contractile Force Associated with Aortic Occlusion and Myocardial Hypothermia or Coronary Perfusion

<table>
<thead>
<tr>
<th>Groups</th>
<th>No. of patients</th>
<th>Length of aortic occlusion (min.)</th>
<th>Average % decrease in left ventricular contractile force</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hypothermia</td>
<td>15</td>
<td>40 to 92</td>
<td>76, 46, 32</td>
</tr>
<tr>
<td>Unilateral perfusion</td>
<td>14</td>
<td>40 to 95</td>
<td>75, 22, 12</td>
</tr>
<tr>
<td>Bilateral perfusion</td>
<td>13</td>
<td>40 to 94</td>
<td>77, 16, 9</td>
</tr>
</tbody>
</table>

The length of aortic occlusion was compared with the degree of depression of left ventricular contractile force (figs. 3, 4 and 5). In the hypothermia group both at 15 minutes and 30 minutes after release of the aortic clamp the decrease in contractile force correlated in general with the length of occlusion (fig. 3). Longer periods of occlusion usually resulted in more depression of con-
Contractile force. There was somewhat less depression of contractile force at 30 minutes following release of the aortic clamp than at 15 minutes.

With unilateral left coronary perfusion, the per cent depression in left ventricular contractile force at 15 minutes and 30 minutes after release of the aortic clamp appeared to increase slightly as the time of aortic occlusion lengthened (fig. 4). Again, there was somewhat less depression of contractile force at 30 minutes after release of the aortic clamp than at 15 minutes.

With bilateral coronary perfusion, the per cent depression in left ventricular contractile force at 15 and 30 minutes after release of the aortic clamp appeared to increase slightly as the length of occlusion increased (fig. 5). Again there was somewhat less depression of contractile force at 30 minutes following release of the aortic clamp than at 15 minutes.

It should be noted that in each group there were two patients who required only 40 minutes of occlusion; in these patients hypothermia or unilateral or bilateral coronary perfusion resulted in similar degrees of left ventricular depression.

In addition, it should be pointed out that in the patients with electrocardiographic evidence of significant coronary arterial occlusive disease, bilateral coronary perfusion appeared to give better protection to the left ventricular myocardium than the other methods.

**Summary**

Forty-five patients undergoing open-heart surgery for acquired aortic valve disease were studied with measurements of left ventricular contractile force. The data presented clearly
indicate that coronary perfusion afforded, on the average, better protection to left ventricular contractility as recorded by a strain-gage arch than did hypothermia. This was particularly true in the usual case, which required an hour or longer of aortic occlusion.

Patients who required 40 minutes or less of aortic occlusion demonstrated approximately identical decreases in contractile force with hypothermia or coronary perfusion.

Bilateral coronary perfusion did not usually afford much additional left ventricular protection as compared to unilateral left coronary perfusion. In a small number of patients with significant coronary arterial occlusion, bilateral coronary perfusion appeared to give better left ventricular protection.

References

Figure 5
Comparison of the length of aortic occlusion with the degree of depression of left ventricular contractile force in the bilateral coronary perfusion group. ●, 15 minutes after release of clamp; X, 30 minutes after release of clamp.
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W. GERALD AUSTEN

Circulation. 1965;32:372-376
doi: 10.1161/01.CIR.32.3.372

Circulation is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
Copyright © 1965 American Heart Association, Inc. All rights reserved.
Print ISSN: 0009-7322. Online ISSN: 1524-4539

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