Assisted Circulation

II. Effects of Counterpulsation on Left Ventricular Oxygen Consumption and Hemodynamics

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A NUMBER of methods utilizing extracorporeal devices to assist the circulation have been described and tested experimentally during the past five years. The two categories into which these methods may be divided are synchronous and asynchronous assistance. Both of these approaches attempt to relieve the work of the heart and to provide an adequate arterial perfusion pressure. Asynchronous partial bypass has the theoretical disadvantage that during cardiac systole the pump creates an increased pressure against which the left ventricle must contract. Dennis et al., however, report that full or near-full bypass effectively reduces myocardial oxygen consumption.

Diastolic augmentation, a form of synchronous assistance, has been shown not to lower myocardial oxygen consumption. The earliest method of synchronous assistance is counterpulsation. In this method, blood is aspirated from the arterial reservoir during cardiac systole, thereby lowering the pressure within the left ventricle. This blood is then returned during diastole. Although the theoretical benefits which may be accrued from synchronously assisted circulation appear to be self evident, it has been difficult to define these benefits experimentally. In this regard, we have chosen to examine the relationship between myocardial oxygen consumption and the altered hemodynamics induced by counterpulsation.

Materials and Methods

Counterpulsation was accomplished by cannulating the terminal abdominal aorta in dogs and connecting this cannula to a single-ended ventricle which is housed in an actuator. This actuator is triggered by a control unit capable of being synchronized with either the R wave of the electrocardiogram or the ascending limb of the arterial pressure curve. The design of this pump and its characteristics were described in a previous communication from this laboratory.

Fourteen experiments were carried out in healthy mongrel dogs anesthetized with intravenous Nembutal. Respirations were assisted, but no supplemental oxygen was given. Left ventricular and central aortic pressures were monitored by means of Sanborn 267 B pressure transducers and a six-channel recorder. Cardiac output was determined by the dye-dilution method using a Colson densitometer. A right hemithoracotomy incision was performed and an incision in the pericardium was made parallel to the right phrenic nerve. The coronary sinus was cannulated through an incision in the right atrium and secured by a purse-string suture. Measurements of coronary blood flow were made at two-minute intervals throughout the entire experiment. These measurements were greatly facilitated by the use of a specially designed cannula. Arterial and venous oxygen contents were determined by the manometric method. The experimental design is illustrated in figure 1, and the methods have been described previously.

The conduct of the experiment was as follows: during the prepump control period, measurements were made of phasic and mean arterial pressures, cardiac output, coronary sinus blood flow, and coronary arteriovenous oxygen contents. Counterpulsation was then begun and the above measurements were repeated after a period of 10 to 15
minutes. Following the period of counterpulsation, the animal was allowed to stabilize and the control measurements were repeated. Attempts were made throughout the entire procedure to maintain a constant mean arterial pressure by varying the venous return from the coronary venous reservoir. The animal was then sacrificed. The heart was removed with the cannula in place, and the position of the cannula in the coronary sinus was verified. The left ventricle was dissected free from valves, great vessels, and remaining chambers and was weighed with the interventricular septum. These weights varied from 77 to 140 Gm.

The formulas used in the calculations of the hemodynamic data have been described in the previous communication. Left ventricular mechanical efficiency index was calculated as

\[
\frac{LVW}{qO_2 \times 2.06}
\]

where \(LVW\) is left ventricular work, Kg./min.; \(qO_2\) is myocardial oxygen consumption, cc./100 Gm. of left ventricle/min.; and 2.06 is the energy equivalent of 1 cc. of oxygen.

TTI/\(qO_2\) has been used as an expression of the energy cost of pressure generation by the left ventricle and is expressed as mm. Hg sec./cc. \(O_2/100\) Gm. of left ventricle/min.

**Results**

The results of experiments in 14 dogs have been divided into two groups and are presented in tables 1 and 2. In the first group of nine dogs, proper synchronization of the pump was achieved, as demonstrated in figure 2. In each instance, a reduction in mean systolic pressure and myocardial oxygen consumption (\(qO_2\)) occurred, and pump systole was confined wholly to the period of cardiac diastole. In the second group of five dogs, there was improper synchronization of the pump. In these experiments, despite a reduction in mean systolic pressure produced by aspiration of the pump, the initiation of pump systole encroached upon the late phase of ventricular systole, and myocardial oxygen consumption increased (fig. 3).

**Proper Synchronization: Group I**

**During Counterpulsation**

The following changes were observed during counterpulsation (tables 1 and 2): mean systolic pressure fell an average of 43 mm. Hg (−39 per cent), tension-time index (TTI) decreased 39 per cent, and there was no change in cardiac output. Thus, the reduction in left ventricular work (−33 per cent) was due entirely to the decreased arterial systolic pressure. Similarly, the TTI was reduced by a selective decrease in the mean systolic pressure, and no significant reduction in the systolic ejection period was noted. Analysis of the duration of isometric contraction did not indicate a significant change, although within the experimental method employed, this measurement was difficult to assess accurately. There was little change in mean diastolic pressure (−5 per cent), but coronary flow rose an average of 50 per cent during counterpulsation. With little change in the duration of diastole (−0.8 per cent) and in the mean diastolic pressure (−5 per cent), this increase in coronary flow was due primarily to a decrease in coronary vascular resistance. The calculated coronary diastolic vascular resistance fell by 39 per cent. The mean arterial pressure fell by 20 per cent, and in the presence of an unchanged cardiac output, this indicates a fall in systemic vascular resistance.

Coronary venous oxygen content rose and myocardial arteriovenous oxygen difference narrowed in eight of the nine experiments. The fall in the arteriovenous oxygen difference averaged 4.77 volumes per cent (−48 per cent). In the single experiment that did not
Table 1

<table>
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<tr>
<th>Experiment no.</th>
<th>Coronary arteriovenous difference vol. per cent</th>
<th>Coronary flow cc./100 Gm./min.</th>
<th>(cc)./100 Gm./min.</th>
<th>Cardiac output L./min.</th>
<th>Mean systolic pressure mm. Hg</th>
<th>Mean diastolic pressure mm. Hg</th>
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C, control; P, pump; O, off; Δ, numerical difference between control and pump.
show a decrease in the arteriovenous difference, there was no significant change in the coronary flow. The myocardial qO2 fell in all nine experiments. The mean reduction was 1.69 ec./100 Gm. of left ventricle/min. (−22 per cent). Mechanical efficiency of the left ventricle decreased slightly (−13 per cent), and the TTI/qO2 ratio fell by 16 per cent. With the exception of mechanical efficiency and TTI/qO2, the changes observed between the control and pump periods are statistically significant as measured by the t-test of differences (table 3).

**Postpump Control Period**

In the postpump control period, phasic arterial pressures, tension-time index, and coronary blood flow returned toward, but never fully reached, the prepump control levels. In spite of this, myocardial arteriovenous oxygen differences remained low and myocardial qO2 actually fell an additional 0.52 ec./100 Gm. of left ventricle/min. Thus, mechanical efficiency of the left ventricle rose in the postpump control period to 19 per cent. This is 4 per cent higher than in the prepump control period. The ratio of TTI/qO2 rose 43 per cent, as compared with the prepump control levels.

**Improper Synchronization: Group II**

With improper synchronization (tables 1 and 2), mean systolic pressure and tension-time index were again both reduced, but these decreases were quantitatively less than in the group with proper synchronization. Mean diastolic pressure, however, was elevated by an average of 21 mm. Hg (+23 per cent), and there was no change in the mean arterial pressure. Although cardiac output increased slightly (+13 per cent), there was a mild fall in left ventricular work (−9 per cent) because of the decreased systolic pressure.

Coronary flow rose by 54 per cent, approximately the same degree as in the experiments with proper synchrony. The fall in coronary vascular resistance was less in this group, as compared with group I (−21 and −39 per cent, respectively). In each of the five experiments, myocardial qO2 rose: the mean increase

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**Table:**

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**Group II: Experimental data**

| 116                  | 103                   | 160                              | 146                      | 0.14                   | 0.16                             | 0.16                           |
| 103                  |                       | 138                              |                          | 22.4                   | 23                               | 23.4                           |
| 97                   | 103                   | 165                              | 128                      | 0.14                   | 0.24                             | 0.24                           |
| 107                  |                       | 130                              |                          | 23                     | 24                               | 24                             |
| 98                   | 90                    | 130                              | 146                      | 0.18                   | 0.21                             | 0.21                           |
| 107                  |                       | 136                              |                          | 23                     | 30                               | 30.7                           |
| 77                   | 88                    | 175                              | 163                      | 0.13                   | 0.23                             | 0.23                           |
| 88                   |                       |                                   |                          | 23                     | 24                               | 24                             |
| 94                   | 75                    | 188                              | 91                       | 0.12                   | 0.18                             | 0.18                           |
| 75                   |                       | 99                               |                          | 22.6                   | 16                               | 16                             |

**Group II: Experimental data**

(continued on next page)

*Circulation, Volume XXVII, April 1963*
Table 1 (continued)

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<th>Experiment no.</th>
<th>Coronary vascular resistance ( \times 10^3 ) dyne-sec. cm.(^{-5} )</th>
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<th>TTI/( q_O^2 )</th>
<th>Left ventricular work Kg. M. /min.</th>
<th>Left ventricular efficiency index per cent</th>
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C, control; P, pump; O, off; \( \Delta \), numerical difference between control and pump.
Effects of synchronous counterpulsation. Pump systole is shown to be confined wholly to ventricular diastole (dotted lines). Ventricular and aortic systolic mean pressures are reduced during counterpulsation.

Discussion

The hemodynamic effects of counterpulsation have been described previously.4,5,8 While reductions in mean systolic pressure and tension-time index have been consistently observed, the effects of counterpulsation upon cardiac output, left ventricular work, and myocardial oxygen requirements have not previously been published. A linear relationship between tension-time index and myocardial oxygen consumption has been shown in acute experiments in the isolated supported dog heart10 and in the intact dog.11 In the present study, myocardial qO₂ was reduced only when proper synchronization of the pump was achieved. Even then, the fall in myocardial oxygen consumption was not commensurate with the marked reduction in tension-time index. Although the fall in the TTI/qO₂ ratio in the group I experiments was not statistically significant (P ≥ 0.05), when the entire group of 14 experiments is examined, a significant fall in the TTI/qO₂ ratio is apparent during counterpulsation (−29 per cent, 0.01...
### Table 2

**Representative Data of Hemodynamic Alterations Produced by Counterpulsation**

<table>
<thead>
<tr>
<th></th>
<th>Control</th>
<th>Pump</th>
<th>Off</th>
<th>Control minus pump</th>
<th>Group 1*</th>
<th>Control</th>
<th>Pump</th>
<th>Off</th>
<th>Control minus pump</th>
<th>Group II†</th>
</tr>
</thead>
<tbody>
<tr>
<td>1 Coronary flow</td>
<td>87</td>
<td>130</td>
<td>117</td>
<td>+ 43 (50%)</td>
<td></td>
<td>70</td>
<td>108</td>
<td>82.0</td>
<td>+ 38 (54.3%)</td>
<td></td>
</tr>
<tr>
<td>cc./100 Gm./min.</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>2 Coronary arteriovenous</td>
<td>9.98</td>
<td>5.21</td>
<td>4.98</td>
<td>- 4.77 (47.8%)</td>
<td>9.69</td>
<td>7.94</td>
<td>7.77</td>
<td>- 1.75 (18%)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>difference, vol. per cent</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>3 Myocardial qO₂</td>
<td>7.79</td>
<td>6.1</td>
<td>5.58</td>
<td>- 1.69 (21.7%)</td>
<td>6.66</td>
<td>8.09</td>
<td>5.98</td>
<td>+ 1.43 (21.5%)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>cc./100 Gm./min.</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>4 Cardiac output</td>
<td>1.59</td>
<td>1.59</td>
<td>1.72</td>
<td>0</td>
<td>1.78</td>
<td>2.02</td>
<td>2.25</td>
<td>+ 0.24 (13.5%)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>L./min.</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>5 Aortic systolic mean</td>
<td>106</td>
<td>65</td>
<td>94</td>
<td>- 41 (38.7%)</td>
<td>104</td>
<td>75</td>
<td>98</td>
<td>- 29 (28%)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>pressure mm. Hg</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>6 Aortic diastolic mean</td>
<td>96</td>
<td>91</td>
<td>81</td>
<td>- 5 (5.2%)</td>
<td>91</td>
<td>112</td>
<td>89</td>
<td>+ 21 (23%)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>pressure mm. Hg</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
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<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>7 Aortic mean pressure</td>
<td>100</td>
<td>80</td>
<td>86</td>
<td>- 20 (20%)</td>
<td>96</td>
<td>95</td>
<td>93</td>
<td>- 1 (1.0%)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>mm. Hg</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>8 Coronary vascular</td>
<td>77.4</td>
<td>47.2</td>
<td>50.5</td>
<td>- 30.2 (39%)</td>
<td>91.6</td>
<td>72.2</td>
<td>66.8</td>
<td>- 19.4 (21.2%)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>resistance (X 10^6) dyne-sec. cm.</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>9 Left ventricular work</td>
<td>2.36</td>
<td>1.58</td>
<td>2.15</td>
<td>- 0.78 (33%)</td>
<td>2.44</td>
<td>2.21</td>
<td>3.02</td>
<td>- 0.23 (9.4%)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Kg. M./min.</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>10 Tension-time index</td>
<td>2532</td>
<td>1549</td>
<td>2270</td>
<td>- 983 (38.8%)</td>
<td>2571</td>
<td>1845</td>
<td>2512</td>
<td>- 726 (28.2%)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>mm. Hg sec./min.</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>11 TTI/qO₂</td>
<td>335</td>
<td>283</td>
<td>479</td>
<td>- 52 (15.5%)</td>
<td>391</td>
<td>228</td>
<td>466</td>
<td>- 163 (41.7%)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>12 Mechanical efficiency index %</td>
<td>15.0</td>
<td>13.04</td>
<td>19.9</td>
<td>- 1.96 (13%)</td>
<td>17.2</td>
<td>12.9</td>
<td>27.3</td>
<td>- 4.3 (33.3%)</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

*Values represent means of nine experiments.
†Values represent means of five experiments.
Figure 3
Effect of improper synchronization of the pump. Pump systole is improperly phased and encroaches upon ventricular systole (dotted lines).

Further analysis indicates that the TTI/qO₂ change in group II was also significant (0.02 > P > 0.01). When improper phasing occurred and the pump systole was initiated before the end of cardiac systole, actual increases in myocardial qO₂ were observed, despite a reduction in the measured tension-time index.

The explanation for the observed increase in the oxygen cost of pressure generation of the left ventricle during counterpulsation is not clear. Kahler et al.¹² reported that there is a dependence of myocardial oxygen consumption upon the level of the coronary blood flow. Analysis of the data revealed that there was no relationship, in these experiments, between the changes in myocardial qO₂ and coronary blood flow (P > 0.2). It is of interest, however, that a statistically significant linear relationship was observed between the

Table 3
Significance of Differences Between Control and Pump

<table>
<thead>
<tr>
<th></th>
<th>Degrees of freedom</th>
<th>d*</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>Tension-time index</td>
<td>8</td>
<td>-983.0</td>
<td>0.001</td>
</tr>
<tr>
<td>qO₂</td>
<td>8</td>
<td>-1.69</td>
<td>0.01 &gt; P &gt; 0.001</td>
</tr>
<tr>
<td>Arteriovenous difference</td>
<td>8</td>
<td>-4.76</td>
<td>0.01 &gt; P &gt; 0.001</td>
</tr>
<tr>
<td>Coronary flow</td>
<td>8</td>
<td>+42.81</td>
<td>0.01 &gt; P &gt; 0.001</td>
</tr>
<tr>
<td>Systolic mean pressure</td>
<td>8</td>
<td>-41.1</td>
<td>&lt; 0.001</td>
</tr>
<tr>
<td>Diastolic mean pressure</td>
<td>8</td>
<td>-4.44</td>
<td>0.05 &gt; P &gt; 0.01</td>
</tr>
<tr>
<td>Left ventricular work</td>
<td>7</td>
<td>-0.78</td>
<td>0.05 &gt; P &gt; 0.01</td>
</tr>
<tr>
<td>Left ventricular efficiency index</td>
<td>7</td>
<td>-1.95</td>
<td>Not significant</td>
</tr>
<tr>
<td>TTI/qO₂</td>
<td>8</td>
<td>-52.56</td>
<td>Not significant</td>
</tr>
</tbody>
</table>

* d = mean difference.
Regression line showing linear relationship between diastolic mean pressures and myocardial oxygen consumption.

myocardial qO₂ and the diastolic perfusion pressure in the entire series, irrespective of proper or improper synchrony (fig. 4). Salisbury and his colleagues¹³ have demonstrated that intramyocardial pressure may be influenced independently by coronary perfusion pressure. This intramyocardial pressure, in turn, was believed to determine the contractile strength of the heart. While the heightened perfusion pressure in the present study was limited primarily to the period of diastole, it is conceivable that this contribution to intramyocardial pressure may be costly to the heart.

The fall in the TTI/qO₂ ratio seen during counterpulsation could also be explained by an increase in the mean radius of the left ventricle.¹⁴ Left ventricular volume measurements were not made in these experiments, but in those 11 dogs in which left ventricular end-diastolic pressures were recorded, no significant change was noted during counterpulsation. In these experiments, there was no hemodynamic evidence for aortic regurgitation. However, in the presence of a heightened diastolic perfusion pressure, substantial regurgitation with chamber dilatation could occur even when the aortic regurgitant valve area was quite small.

In most of the experiments, coronary diastolic vascular resistance (11/14) and systemic vascular resistance (9/14) fell during counterpulsation. Whether this was due to the opening of collateral channels or to a direct effect on vascular tone is not currently known. The work of Jacoby et al.¹⁵ would suggest that the former mechanism may be, at least in part, responsible. Similarly, the heightened mechanical efficiency and the rise in the TTI/qO₂ ratio observed during the postpump control period are not easily explained, but again may be related to volume changes of the left ventricle.

Summary

The effect of counterpulsation upon hemodynamics and myocardial oxygen consumption was studied in 14 dogs. With properly synchronized counterpulsation, a reduction in mean systolic pressure, tension-time index, left ventricular work, and myocardial oxygen consumption was observed, with no change in effective cardiac output. The reduction in myocardial qO₂, however, did not parallel the fall in tension-time index. Mechanical efficiency fell and the oxygen cost of pressure generation by the left ventricle rose. Premature triggering of the pump systole resulted in an actual increase in myocardial qO₂, despite a fall in tension-time index. The influence of counterpulsation upon the energy requirements of the left ventricle is briefly discussed.

References


Assisted Circulation: II. Effects of Counterpulsation on Left Ventricular Oxygen Consumption and Hemodynamics

HARRY S. SOROFF, HERBERT J. LEVINE, BARRY F. SACHS, WILLIAM C. BIRTWELL, RALPH A. DETERLING, JR., Judith A. Collins and Jean Kasuba

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