Effects of a Selective Bypass of the Left Ventricle

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In a series of articles in 1962, Dennis, Senning, Hall, and Moreno described a method by which the left ventricle could be bypassed without thoracotomy. A cannula passed down the right internal jugular vein perforates the atrial septum and enters the left atrium; a simple circuit then allows left atrial blood to be pumped to the femoral artery. This method of reducing the work of the left ventricle has obvious possibilities of being clinically useful. This is a report of an investigation into the effects of such a bypass on circulatory dynamics and myocardial metabolism. Attempts to assess its value in experimental coronary shock and following aortic valvular operations are also reported.

Methods

Forty-five mongrel dogs (11 to 32 Kg.) were anesthetized with Pentothal, atropine, and nitrous oxide. Both pleural cavities were entered via short intercostal incisions. Pressures were monitored in the ascending aorta, the right atrium, the pulmonary artery, and the left atrium. The arterial pressures were transmitted via strain-gauge transducers to a Grass four-channel recorder on which the dye curves and electrocardiogram were also recorded. The atrial pressures were monitored on saline manometers. All pressures were standardized to a level of 10 cm. above the operating table. Esophageal temperature was kept between 36 and 39 C. by means of a controlled temperature blanket beneath the dog and a heat exchanger in the arterial line. After preliminary dissections were completed, heparin (2 mg./Kg.) was given.

Cardiac outputs were estimated by the indiændilution technique (indocyanine-green dye, Colson densitometer, and three-cycle logarithmic paper). Two curves were used in estimating each output. For right ventricular outputs, the injection site was the superior vena cava and the sampling site the pulmonary artery; for left ventricular outputs, the injection site was the left ventricle (through a transmural catheter with its tip pointing toward the apex) and the sampling site was the ascending aorta.

In experiments in which myocardial oxygen utilization was determined, coronary flow was collected through a large catheter inserted one inch into the coronary sinus via the right atrium. A suture ligature passed around the sinus just to the right of the middle cardiac vein assured that the sampled blood was mainly from the left ventricle. A large tube returned the coronary sinus blood to the right atrium. Coronary flow was determined by occluding this tube for 30 seconds and allowing the blood to flow, via a large Y connection, to a graduated cylinder. Samples of aortic and coronary sinus blood were collected simultaneously in heparinized syringes and the oxygen content measured immediately by the Van Slyke method. An estimation of cardiac output was obtained immediately before and after the collection of each sample.

A J-shaped tube (I.D. = 8 mm.) with several end holes was inserted into the left atrium via the atrial appendage; it was secured by a thick circumferential ligature to avoid needle holes and reduce the possibility of air embolism at low left atrial pressures. Left atrial outflow was by gravity (60 to 80 cm.) to an open reservoir and was controlled by a screw clamp on the atrial line. The DeBakey-style Pemco pump was set to be occlusive to 2 ml. of blood and was calibrated as a flowmeter prior to each experiment. Heparinized blood for priming and transfusion was obtained by exsanguinating the donor dog under local anesthesia. No attempt was made to cross match the blood of the animals.

Coronary shock was produced by a slight modification of the method of Agress. Three mm./Kg. of plastic microspheres with an average diameter of 250 μ were injected into either the left ventricle or the root of the aorta. The ascending aorta was cross clamped for one minute, while simultaneous caval occlusion prevented cardiac distention.

Definitions and Calculations

Mean arterial blood pressure was calculated from the diastolic pressure plus one-third of the pulse pressure. The external work of each ventricle was calculated as the product of the minute out-
put per Kg. times the mean arterial pressure minus the atrial pressure.

Myocardial oxygen consumption, expressed as cc./100 Gm. of left ventricular muscle/min. was calculated from the product of the coronary sinus flow (as defined above) and the arteriovenous oxygen difference. The left ventricular weight was estimated as a fixed percentage of the body weight.⁸

Time-tension index was calculated in mm. Hg seconds from aortic pressure recordings at a paper speed of 100 mm./sec.⁹

The data were obtained from three series of experiments. (1) Bypass was increased in stepwise fashion from zero to near total bypass and back again. Only measured blood loss was replaced. Eleven dogs were used; six yielded useful results. (2) The bypass was set at approximately one-third of the dog's initial cardiac output and continued for 60 to 90 minutes. Only measured blood loss was replaced. Ten dogs were used; five yielded useful results. (3) Myocardial oxygen consumption before, during, and after low and high levels of bypass was measured. Transfusion was given as required to maintain a steady arterial blood pressure. Twenty dogs were used; 15 yielded useful results.

Results

Cardiac Output

In the initial series of experiments, in which pump speed was changed in a stepwise fashion, the left ventricular output was reduced as desired to near zero levels (fig. 1). The right ventricular output was not affected by the bypass. In a dog with a thoracotomy and a beating heart, it was not possible to achieve a complete bypass of the left ventricle. If complete bypass was attempted, the collapse of pulmonary veins, left atrium, and venous cannulas allowed leakage of air into the system.

The exact proportion of the systemic arterial flow being contributed by such a bypass was difficult to determine without repeated measurements of cardiac output. The cardiac output fell slowly, but steadily, in every animal in which only the measured blood loss was replaced. Thus, with constant pump speed as in the second series of experiments, a bypass of any given fraction of the initial cardiac output would slowly take over a larger proportion of the total flow. This reaction of the dog to thoracotomy and bypass was probably a result of decreased venous return from splanchnic and peripheral beds. The falling output was easily stabilized by transfusion.

A flowmeter on the pulmonary artery would probably be the most accurate method of determining the cardiac output in this bypass situation. However, as the dye-dilution method would be applicable clinically, we elected to use this technique. Several problems were immediately encountered. During bypass, the curves obtained from superior vena caval injection and ascending aorta sampling were bizarre and difficult to interpret. Left ventricular injection and aortic sampling gave accurate left ventricular outputs at low and intermediate levels of bypass, but at high bypass levels the curves were distorted by the blood from the pump, which now reached the

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ascending aorta in each period of ventricular diastole. Only right atrial injection and pulmonary arterial sampling gave accurate, repeatable curves at all levels of bypass. After the initial experiments, left ventricular outputs were determined by subtracting the known pump output from the measured right ventricular output.

**Systemic Arterial Pressure**

The proportion of the systemic arterial flow contributed by the left ventricle and by the bypass was reflected in the character of the aortic pulse wave. At complete and nearly complete left ventricular bypass, only the pattern of the pump output was seen. The shape of the pulse pattern was thus a rough guide to that proportion of the systemic output which was supported by the bypass. The rate of the heart was not affected significantly or consistently by the presence or amount of the bypass.

The mean level of the aortic pressure fluctuated at the times of change in pump speed, but was not affected by the direction in which the blood reached the aorta—either via the aortic valve or via the femoral artery. The finding of a slow fall in mean arterial pressure after 15 to 20 minutes of bypass in every experiment in which only the measured blood loss was replaced was a reflection of the fall in the effective circulating blood volume described before and to which the peripheral resistance of the animal responded with variable effectiveness. In those experiments in which a constant aortic pressure was essential, it was obtained by continuous transfusion (±10 mm. Hg, usually ± less than 5 mm. Hg; see table 1).

**Left Atrial Pressure**

With the commencement of the bypass, the left atrial pressure fell below that of the right atrium (fig. 1). At high levels of bypass, it was always very low (+2 to −2 mm. Hg), but one could never predict from the level of bypass just when the left atrial pressure would become negative. The inability to predict the left atrial pressure resulting from any given flow is shown in figures 2 and 3, in which a bypass of 90 per cent of the left atrial blood has lowered the left atrial pressure 64 per cent, and a bypass of 50 per cent has lowered it 60 per cent.

The collapse of the left atrium with negative pressures precluded any attempt at 100 per cent bypass in the dog with a thoracotomy, a beating heart, and a nonobstructed mitral valve. The 100 per cent bypass obtained by

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**Table 1**

**Mean Values Observed in Twenty-two Paired Observations Before and During Bypass**

<table>
<thead>
<tr>
<th></th>
<th>High bypass (12 observations)</th>
<th>Low bypass (10 observations)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Before</td>
<td>During</td>
</tr>
<tr>
<td>Cardiac output</td>
<td>59.6 (22.3)</td>
<td>6.1 (6.2)</td>
</tr>
<tr>
<td>(cc./Kg./min.)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Aortic blood pressure</td>
<td>90 (14)</td>
<td>89 (11)</td>
</tr>
<tr>
<td>(mm. Hg)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Cardiac rate</td>
<td>163 (33)</td>
<td>166 (20)</td>
</tr>
<tr>
<td>(beats/min.)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Coronary flow</td>
<td>55.8 (28.8)</td>
<td>40.0 (12.7)</td>
</tr>
<tr>
<td>(cc./min.)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Arteriovenous</td>
<td>11.0 (3.3)</td>
<td>10.9 (2.8)</td>
</tr>
<tr>
<td>oxygen difference (vol. %)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Oxygen consumption</td>
<td>6.7 (3.0)</td>
<td>4.6 (1.8)</td>
</tr>
<tr>
<td>(cc./100 Gm. left</td>
<td></td>
<td></td>
</tr>
<tr>
<td>ventricle/min.)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Time-tension index</td>
<td>13.2 (2.4)</td>
<td>11.2 (1.8)</td>
</tr>
<tr>
<td>(mm. Hg sec.)</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Numbers in parentheses indicate the standard deviation.
N.S. = not significant.
Moreno et al.\textsuperscript{4} suggests that the negative intrathoracic pressure of the intact dog may allow higher levels of bypass.

**External Work of the Heart**

A calculation of the external work performed by each ventricle integrates the effect of the bypass on the output of the ventricle, the pressure of the atrium which loads it, and the pressure of the artery to which it expels its contents. The external work of the left ventricle can be reduced at will, while that of the right ventricle is not basically affected.

As long as the systemic blood pressure is held constant, the external work of the left ventricle will be reduced in direct proportion to the reduction in ventricular output, apart from the slight increase in calculated work which results from a lowering of the left atrial pressure (figs. 2 and 3).

There is controversy over the exact significance of the external work of the heart as calculated here. If the work is reduced by a reduction in the pressure against which the ventricle must expel its contents, the energy required by the myocardium is much less than if the same reduction in work is achieved through a reduction in stroke volume.\textsuperscript{9} This relationship was investigated in the third series of experiments in which the oxygen consumption of the heart was measured before, during, and after ventricular bypass.

**Oxygen Consumption of the Bypassed Left Ventricle**

Twenty-two observations were obtained in animals in which the arterial blood pressure was adequately controlled. In 10 observations, the output of the left ventricle was reduced by approximately 50 per cent from its adjacent control (low bypass). In 12 observations, it was reduced by approximately 90 per cent from its adjacent control (high bypass) (see table 1). As the actual cardiac output at the time of bypass could not be accurately calculated until the completion of the experiment, the levels of bypass were determined pragmatically by the volume of the bypass, the appearance of the aortic pulse pattern, and the level of the left atrial pressure.

With a 48 per cent reduction in left ventricular volume load (49 per cent reduction in left ventricular external work), the oxygen consumption of the left ventricle was approximately 48 per cent of the control level.
consumption of the left ventricle was reduced 19 per cent (P < 0.05). With a 90 per cent reduction in volume load (89 per cent reduction in external work), the oxygen consumption was reduced 31 per cent (P < 0.01). In round figures, these results indicate that a reduction by this method of one-half the external work of the left ventricle will reduce its oxygen consumption by one-fifth; a reduction of nine-tenths of its work will reduce its oxygen consumption by one-third. The major reduction is in coronary flow rather than in arteriovenous difference.

The time-tension index was measured at the same time as the oxygen consumption. Although the index was somewhat reduced during bypass by a reduction in systolic peak pressure and a shortly reduced length of systole, it did not correlate well with the reduction in oxygen consumption. The B.P. × H.R. (arterial blood pressure times heart rate) index also did not indicate the changes in oxygen consumption as mean arterial blood pressure was held constant and heart rate did not vary consistently with the presence or absence of bypass.

Treatment of Coronary Shock and Cardiac Failure

True congestive failure is notoriously difficult to produce in the dog. The creation of acute valvular defects and the treatment of the resulting vascular imbalance by a mechanical bypass can easily deteriorate to a “juggling” of hemodynamic values. As an experimentally weakened myocardium seemed the best test object, and as the results of coronary arterial ligation were not controllable, an attempt was made to produce myocardial damage and coronary shock from microemboli.

In seven animals in which the emboli were injected, coronary shock—a state of reduced cardiac output and hypotension which responds to transfusion by failure rather than by a return to normal pressure—was produced only once. Three animals fibrillated; the other three developed hypotension, but on transfusion the pressure rose to normal levels and failure did not occur. We were thus unable to obtain a stable experimental preparation of this condition in which to test the influence of left ventricular support.

We have applied this method to two patients, both of whom were in a terminal condition from inadequate left ventricular output following prosthetic replacement of the aortic valve. In both, the arterial pressure rose to viable levels and the cardiac action became vigorous. In order to maintain the elevated blood pressure, however, overtransfusion was required, and the right side of the heart distended. Despite several hours of bypass, we were unable successfully to wean either patient from the mechanical support.

Discussion

The procedure of selective left ventricular bypass lowers left atrial pressure and reduces the stroke volume of the left ventricle while, at the same time, an adequate aortic pressure is maintained. The reduction in the oxygen consumption of the myocardium accompanying the reduction in left ventricular stroke volume is greater than expected from previous work on the heart-lung preparation. A reduction of nine-tenths of the volume load produced a reduction of one-third in the oxygen consumption; a reduction of one-half the volume load produced a reduction of one-fifth in the oxygen consumption. These reductions were not mirrored by either the time-tension index or the B.P. × H.R. index. Very similar results were reported by Dennis et al. The observations suggest that a reduction in the volume load of the left ventricle should have beneficial results clinically and is, indeed, “resting” the heart.

The myocardial wall tension is the primary determinant of cardiac oxygen consumption. The reduced volume load will affect the tension through a reduction in the ventricular radius which, it has been suggested, will vary as the cube root of the volume. The correlation between oxygen consumption and either stroke volume or radius (calculated from the stroke volume) was not significant in our experiment. It is probable, therefore, that the bypass changes the relationship between the stroke volume and the end-diastolic
volume of the ventricle. It is the end-diastolic volume which, of course, is the important determinant of the radius.

Our brief clinical experience suggests that the method must be applied before the right ventricle has also failed. An attempt to support the entire circulation by a left ventricular bypass alone requires overtransfusion and distention of the right side of the heart.

Summary and Conclusion
A selective bypass of the left ventricle is accompanied by a significant reduction in the oxygen consumption of the heart. This reduction in oxygen consumption is not mirrored by the time-tension index or the heart rate × blood pressure index. The reduction in left atrial pressure is not predictable. The method should be applied before the right side of the heart has failed.

References

BAIRD, LABROSSE, LAJOS, THOMAS
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