Effects of Coronary Bypass Graft Occlusion on Left Ventricular Performance

By Richard P. Anderson, M.D.

SUMMARY

High-fidelity recordings of left ventricular (LV) pressure and its corresponding first derivative (dP/dt), electrocardiogram, and systemic arterial pressure were obtained intraoperatively following insertion of aortocoronary saphenous vein bypass grafts in eight men. During sequential 3-min periods, the bypass grafts were, in turn, open, occluded by vascular clamps, and opened by releasing the clamps.

Peak dP/dt averaged 1342 mm Hg/sec before graft occlusion, fell to 1223 mm Hg/sec during occlusion, and rose following release of occlusion to 1373 mm Hg/sec. LV end-diastolic pressure (LVEDP) averaged 14.5 mm Hg before, 17.6 mm Hg during, and 16.1 mm Hg after graft occlusion. Arterial systolic and diastolic pressure and heart rate showed little change (P > 0.05) throughout the experiments. Left ventricular performance as gauged by peak dP/dt/LVEDP decreased an average of 25% (P < 0.001) during graft occlusion and returned to control levels following release of occlusion. Total vein graft blood flow, measured immediately before the occlusion experiments, averaged 164 ml/min. Good correlation was lacking between vein graft blood flow and performance changes during graft occlusion in individual patients.

Aortocoronary vein bypass grafts are capable of participating directly in the mechanical events of the cardiac cycle. Acute graft occlusion can impair, while restoration of graft flow can improve, left ventricular performance.

Additional Indexing Words:
Coronary heart disease
Left ventricular dP/dt

DIRECT revascularization of the heart by means of aortocoronary bypass grafts is performed primarily for the relief of anginal chest pain. Thus, an important part of the evaluation of operative results is necessarily subjective in nature. Despite the widespread acceptance and application of the direct revascularization technic,1-3 critical observers have voiced concern over a lack of objective data supporting the apparent benefits of operation.4-6 Many recall that most of the indirect technics for myocardial revascularization applied in former years were without demonstrable physiologic effects.

Accordingly, several investigators have sought objective evidence of the influence of the coronary bypass graft operation upon the circulation. Rees et al.7 carried out quantitative analyses of left ventricular cineangiocardiograms before and after aortocoronary vein bypass operations for angina. Among patients without severe depression of ventricular performance, ventricular function improved when the operation was technically successful. Deterioration of function accompanied graft occlusion. Amsterdam et al.8 studied changes in an index of myocardial oxygen consumption, the product of heart rate, and mean systemic arterial pressure (RPP), during bicycle ergometer exercise before and after vein bypass surgery. Among 20 patients there was a mean increase of 24% in RPP postoperatively.

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The present study was performed to determine if the presence of aortocoronary bypass grafts had an immediate influence on the function of the left ventricle. This assessment was made by observing changes in the performance of the left ventricle brought about by temporary occlusion of the grafts at operation.

Methods

Intraoperative hemodynamic measurements were obtained in eight men following the insertion of autogenous saphenous vein grafts between the aorta and major coronary arteries. All patients had exertional angina pectoris and severe atherosclerotic coronary artery disease as defined by preoperative selective left and right coronary arteriograms. Clinical information is summarized in Table 1. Operations were performed under conditions of cardiopulmonary bypass, left ventricular apical venting, mild hypothermia, and electrical fibrillation. The technical details have been reported elsewhere. Informed consent for the intraoperative measurements to be described was obtained from each patient before operation. Measurements were made with the chest open. The circulation was stable with normal sinus rhythm and no vasopressor drugs were being given. The electrocardiogram was under continuous observation for ischemic changes.

Blood flow through the grafts was measured using a Biotronex BL-610 electromagnetic flowmeter. Zero flow was established by occlusion of the graft distal to the flow transducer, and meter readings were taken in triplicate and averaged. Flow transducers were calibrated for each patient by means of an in vitro system using blood from the pump oxygenator at 37°C and the flowmeter readings were converted to flow in ml of blood/min using the resulting calibration curves.

All other experimental data were recorded simultaneously on a four-channel Sanborn HP 7700 chart recorder. Peripheral arterial pressure was obtained by a plastic cannula inserted into the radial artery at cutdown and attached to a Statham P-23Db transducer. Left ventricular pressure was measured through a 20-gauge needle attached directly to a Statham P-37 transducer with zero pressure referred to the level of the left ventricle. This system showed an amplitude response which was linear from 0 to 30 Hz when tested in a mechanical pressure-generating chamber. The needle was directed into the left ventricular cavity through the anterior wall of the right ventricle and septum. Needle and cannula were irrigated with saline before each measurement. Transducers were calibrated with an air-pressure manometer system. The first derivative of left ventricular pressure (dP/dt) was obtained by an analog-differentiating circuit possessing an amplitude response which was linear within ±1% at 15 Hz and ±4% at 25 Hz. A precision limiter circuit permitted recording of only the positive deflection of dP/dt when maximum amplitude was desired.

Each experiment was divided into three sequential 3-min periods. During the first period the newly inserted grafts were open and control measurements were made. Next, the grafts were occluded by vascular clamps for 3 min and repeat data were obtained. The occlusion was then released and observations continued for an additional 3 min. Recordings at a paper speed of 100 mm/sec for 20 sec were taken at 1-min

### Table 1

**Clinical Data**

<table>
<thead>
<tr>
<th>Case</th>
<th>Age (yr)</th>
<th>Duration (yr)</th>
<th>Severity</th>
<th>Electrocardiogram</th>
<th>Angiographic stenosis (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>52</td>
<td>15</td>
<td>At rest</td>
<td>Old infarct, ischemia, LVH</td>
<td>RCA† 100, LAD 90* Cire 50</td>
</tr>
<tr>
<td>2</td>
<td>53</td>
<td>1</td>
<td>At rest</td>
<td>Ischemia</td>
<td>RCA† 95*, LAD 100* 100</td>
</tr>
<tr>
<td>3</td>
<td>50</td>
<td>10</td>
<td>At rest</td>
<td>Ischemia</td>
<td>RCA† 100*, LAD 100*</td>
</tr>
<tr>
<td>4</td>
<td>45</td>
<td>1/3</td>
<td>Minimal effort</td>
<td>Old infarct</td>
<td>RCA† 90*, LAD 100* 90*</td>
</tr>
<tr>
<td>5</td>
<td>47</td>
<td>1/2</td>
<td>At rest</td>
<td>Old infarct</td>
<td>RCA† 90*, LAD 80*</td>
</tr>
<tr>
<td>6</td>
<td>50</td>
<td>1/2</td>
<td>Minimal effort</td>
<td>Normal</td>
<td>RCA† 90*, LAD 80*</td>
</tr>
<tr>
<td>7</td>
<td>37</td>
<td>4</td>
<td>At rest</td>
<td>Old infarct</td>
<td>RCA† 100*, LAD 100*</td>
</tr>
<tr>
<td>8</td>
<td>49</td>
<td>2</td>
<td>Minimal effort</td>
<td>Old infarct</td>
<td>RCA† 100*, LAD 100* 60</td>
</tr>
</tbody>
</table>

Abbreviations: RCA = right coronary artery; LAD = left anterior descending coronary artery; Cire = circumflex artery.

*Coronary artery bypassed with graft.
†Dominant in all cases except 4.
The values for each variable were obtained by calculating the average five consecutive cardiac cycles at each minute throughout the experiment. Ventricular performance was assessed by the ratio of peak dP/dt to left ventricular end-diastolic pressure (LVEDP), as suggested by Reeves et al.\textsuperscript{10} For purposes of statistical comparison, the mean values for each variable during each of the 3-min periods were computed. The statistical significance of the differences before, during, and after graft occlusion was determined by a two-tailed Students t test.

Results

The mean values for all of the measured variables during the 3-min periods before, during, and after graft occlusion together with the results of the statistical analysis of the observed differences are listed in table 2. Arterial systolic and diastolic pressure and heart rate showed only minor variations throughout the experiments and these changes are not significant ($P > 0.05$).

LV peak dP/dt fell an average of 9\% during graft occlusion ($P < 0.05$) and returned to control level following release of occlusion. Figure 2 shows the average value of LV peak dP/dt at each minute of the experiment. There is a prompt decrease during the first

Table 2

<table>
<thead>
<tr>
<th>Variable</th>
<th>A Before graft occlusion ($P$ value A-B)</th>
<th>B During graft occlusion ($P$ value B-C)</th>
<th>C After graft occlusion ($P$ value A-C)</th>
</tr>
</thead>
<tbody>
<tr>
<td>LV peak dP/dt/mm Hg LVEDP</td>
<td>95.4 ± 4.0</td>
<td>72.0 ± 3.4</td>
<td>89.6 ± 5.5</td>
</tr>
<tr>
<td></td>
<td>&lt;0.001</td>
<td>&lt;0.01</td>
<td>NS†</td>
</tr>
<tr>
<td>LV peak dP/dt (mm Hg/sec)</td>
<td>1342 ± 42</td>
<td>1223 ± 45</td>
<td>1373 ± 52</td>
</tr>
<tr>
<td></td>
<td>&lt;0.05</td>
<td>&lt;0.05</td>
<td>NS</td>
</tr>
<tr>
<td>LV end-diastolic pressure (mm Hg)</td>
<td>14.5 ± 0.7</td>
<td>17.6 ± 4.3</td>
<td>16.1 ± 3.9</td>
</tr>
<tr>
<td></td>
<td>&lt;0.01</td>
<td>NS</td>
<td>NS</td>
</tr>
<tr>
<td>Radial artery systolic pressure</td>
<td>126 ± 4.5</td>
<td>122 ± 4.2</td>
<td>126 ± 4.0</td>
</tr>
<tr>
<td>(mm Hg)</td>
<td>NS</td>
<td>NS</td>
<td>NS</td>
</tr>
<tr>
<td>Radial artery diastolic pressure</td>
<td>74 ± 2</td>
<td>73 ± 2</td>
<td>75 ± 3</td>
</tr>
<tr>
<td>(mm Hg)</td>
<td>NS</td>
<td>NS</td>
<td>NS</td>
</tr>
<tr>
<td>Heart rate (cycles/min)</td>
<td>104.9 ± 3.0</td>
<td>105.4 ± 3.0</td>
<td>106.4 ± 3.5</td>
</tr>
<tr>
<td></td>
<td>NS</td>
<td>NS</td>
<td>NS</td>
</tr>
</tbody>
</table>

*Mean ± SEM.
†Probability that difference between mean values in indicated groups is due to chance alone.
‡NS ($P > 0.05$).
minute of graft occlusion which is maintained until release of occlusion. One minute after release of occlusion LV peak dP/dt has returned to near the control levels.

LVEDP increased significantly \((P < 0.01)\) during graft occlusion (table 2). Following release of occlusion LVEDP fell to an intermediate level which was not significantly different from either the preocclusion or occlusion values. The minute-to-minute changes in mean LVEDP are shown in figure 3.

An index of ventricular performance was calculated by combining peak dP/dt and LVEDP in a ratio.\(^{12}\) Figure 4 shows this index falling progressively throughout the 3 min of graft occlusion and then returning to control levels during the first minute following release of occlusion. The mean decrease in the performance index during occlusion of the grafts is 25\%, a highly significant change \((P < 0.001)\). There is no significant difference between the pre- and postocclusion values (table 2).

Table 3 lists the total vein bypass graft blood flow together with the average change in the study parameters for each patient. Blood flows ranged from 90 to 240 ml/min with a mean of 164 ml/min. The average decrease in peak dP/dt during graft occlusion ranged from 15 to 200 mm Hg/sec with a mean of 119 mm Hg/sec while LVEDP showed an increase ranging from 1 to 8 mm Hg and averaging 3 mm Hg. The decrease in performance index ranged from 6 to 52 mm Hg/sec/mm Hg LVEDP with a mean of 23 mm Hg/sec/mm Hg LVEDP. There is very little linear correlation evident between vein graft blood flow and changes in performance index \((r = -0.20)\), peak dP/dt \((r = -0.40)\), and LVEDP \((r = -0.04)\).

**Discussion**

Measurement of left ventricular performance under operating room conditions in man requires a technic which can be applied during a brief period of time with a high degree of safety. Assumption through analysis of the left ventricular pressure pulse meets these criteria.
CORONARY BYPASS GRAFT OCCLUSION

Table 3

<table>
<thead>
<tr>
<th>Case</th>
<th>Total graft flow (ml/min)</th>
<th>Decrease in peak dP/dt (mm Hg/min)</th>
<th>Increase in LVEDP (mm Hg)</th>
<th>Decrease in LV performance index (mm Hg/sec. mm Hg LVEDP)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>150</td>
<td>200</td>
<td>4</td>
<td>26</td>
</tr>
<tr>
<td>2</td>
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<td>22</td>
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<tr>
<td>4</td>
<td>90</td>
<td>100</td>
<td>1</td>
<td>18</td>
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<td>130</td>
<td>157</td>
<td>2</td>
<td>22</td>
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<td>195</td>
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<td>7</td>
<td>190</td>
<td>156</td>
<td>2</td>
<td>22</td>
</tr>
<tr>
<td>8</td>
<td>180</td>
<td>15</td>
<td>2</td>
<td>6</td>
</tr>
<tr>
<td>Average</td>
<td>164</td>
<td>119</td>
<td>3</td>
<td>23</td>
</tr>
</tbody>
</table>

*Mean change from control during 3-min period of graft occlusion.

The rate of rise of the ventricular pressure pulse (dP/dt) is a complex variable which is an expression of ventricular performance. Changes in dP/dt are directionally related to changes in the loading conditions under which the ventricle is operating as well as to changes in the inotropic state of the myocardium. Thus, increases in preload, reflected by LVEDP, and afterload, reflected by arterial diastolic pressure, tend to increase peak dP/dt. Similarly, a rise in peak dP/dt may be brought about by enhancement of the inotropic state of the heart such as occurs with catecholamine drug administration or in association with increases in heart rate (Bowditch effect).

In the present studies heart rate and arterial diastolic pressure remained virtually unchanged and were unlikely to produce the observed changes in peak dP/dt with manipulation of the grafts. The decrease in peak dP/dt with graft occlusion could have been produced by alterations in the contractile state of the ventricle and by changes in the preload. LVEDP actually rose during graft occlusion, a change directionally opposite to the observed decrease in peak dP/dt, suggesting that myocardial contractility may have fallen.

The decrease in peak dP/dt and increase in LVEDP with graft occlusion are similar to change produced in experimental animals by coronary artery constriction. Presumably these changes are brought about by the acute decrease in blood flow to the myocardium which may accompany graft occlusion and which is reversible by restoration of graft flow. Myocardial ischemia, however, may result in decreased ventricular compliance or increased stiffness. If the observed increase in LVEDP with graft occlusion reflects a decrease in ventricular compliance rather than an increase in end-diastolic volume, then the fall in myocardial contractility assumed from the decrease in peak first derivative could be slight or even nonexistent. For this reason peak dP/dt normalized for LVEDP, the performance index, should not be taken as a quantitative measure of myocardial contractility. Rather, it may be regarded as a convenient expression which quantifies and consolidates the observed performance changes.

Although all patients in this study showed an impairment in LV performance following graft occlusion this response may not always be seen. Recently Bolooki et al. reported measurements of extrapolated maximum contractile element velocity (Vmax) during coronary bypass graft occlusion. Three patients with single grafts to the right coronary artery showed no decrease in Vmax with graft occlusion while four patients with grafts to the left anterior descending artery plus a second graft showed a substantial decrease. All patients in the present study required and received grafts to the left anterior descending artery while six of eight also received a second graft, either to the right or a circumflex artery.
supplying the major posterior descending artery. This suggests that the severity of the disease and both the number and location of the bypass grafts may, in part, determine whether or not performance changes occur with graft occlusion.

A relationship between the measured blood flow through the grafts and the magnitude of performance change during graft occlusion could not be established. It is of interest, however, that the three patients showing the least change in performance index had angina only with exertion while all others had, in addition, angina at rest. None of these three showed electrocardiographic evidence of ischemia while one possessed the only normal electrocardiogram in the group. In contrast, two patients who showed the greatest performance changes had angina occurring both with effort and at rest, electrocardiographic evidence of ischemia, and angiographic findings of obstructions in all three major coronary arteries. If the clinical severity of the disease may be equated with the requirements of the heart for a supplementary blood supply then those patients with the greatest requirements tended to show the greatest performance response to graft occlusion. Perhaps when a critical level of graft flow capable of fulfilling these requirements is reached, additional increments of flow which the grafts may be carrying are redundant and have little effect upon performance changes during graft occlusion.

A conclusion that aortocoronary bypass grafts, of themselves, improve left ventricular performance is not warranted from the data of this study. Nevertheless, once inserted and functioning, the grafts have been shown to participate directly in the mechanical events of the cardiac cycle. When graft flow is interrupted, myocardial performance declines. Restoration of graft flow is followed by enhanced performance. The severity of the coronary artery disease among the patients studied, the requirements of their hearts for an additional blood supply, and the ability of their bypass grafts to meet these needs may all be important determinants of these responses to graft occlusion. Moreover, the extent of performance change appeared to be related to some aspects of the functional state of the individual patient, his electrocardiogram, and his angiographic findings.

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