Assessment of left ventricular function following coronary bypass surgery: a non-invasive study

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Hardarson, T., Ziady, G. M., and Khattri, H. N. (1974). Thorax, 29, 359–365. Assessment of left ventricular function following coronary bypass surgery: a non-invasive study. In a series of 15 patients with ischaemic heart disease, systolic time intervals (STI) were measured before, and at one week, three months, and six months following coronary vein-graft surgery. Preoperatively, the left ventricular ejection time (LVET) was abnormally short in seven patients, while the pre-ejection period was abnormally long in seven patients, suggesting impaired left ventricular function. At one week after surgery LVET and total electromechanical systole (QA2) were significantly abbreviated. This may be explained by the transient fall in cardiac output or postoperative neurohumoral changes. For the group as a whole, no significant changes were found at three or six months, suggesting that cardiac function was generally preserved rather than improved. However, in individual patients changes in STI correlated with the clinical and angiographic estimate of success of the operative treatment.

While the relief of angina is achieved in the majority of patients with ischaemic heart disease who are subjected to myocardial revascularization procedures (Mitchel et al., 1970; Spencer, Green, Tice, and Glassman, 1971; Morris et al., 1972) the effects on cardiac function are less certain.

The work of Weissler, Harris, and Schoenfeld (1968, 1969) suggests that systolic time intervals (STI) are a relevant and informative measure of left ventricular (LV) function. Being non-invasive and easily derived, these methods open the possibility of serial measurements over extended periods of time in a large number of patients.

This further avoids the risk associated with repeated cardiac catheterizations and angiography.

We have followed a series of 15 patients over

<table>
<thead>
<tr>
<th>Patient</th>
<th>Age</th>
<th>Sex</th>
<th>Years</th>
<th>Exercise (0–3)</th>
<th>Rest (0–3)</th>
<th>Previous Myocardial Infarction (yr)</th>
<th>BP (mmHg)</th>
</tr>
</thead>
<tbody>
<tr>
<td>V.C.</td>
<td>50</td>
<td>F</td>
<td>6</td>
<td>2</td>
<td>0</td>
<td>6</td>
<td>165/85</td>
</tr>
<tr>
<td>M.J.</td>
<td>39</td>
<td>M</td>
<td>7</td>
<td>2</td>
<td>1</td>
<td>4</td>
<td>170/90</td>
</tr>
<tr>
<td>T.W.</td>
<td>37</td>
<td>M</td>
<td>3</td>
<td>3</td>
<td>1</td>
<td>3</td>
<td>130/90</td>
</tr>
<tr>
<td>W.D.</td>
<td>37</td>
<td>M</td>
<td>1</td>
<td>3</td>
<td>2</td>
<td>1</td>
<td>170/80</td>
</tr>
<tr>
<td>B.P.</td>
<td>31</td>
<td>M</td>
<td>4</td>
<td>3</td>
<td>3</td>
<td>3</td>
<td>130/90</td>
</tr>
<tr>
<td>G.S.</td>
<td>37</td>
<td>M</td>
<td>6</td>
<td>3</td>
<td>2</td>
<td>3</td>
<td>130/90</td>
</tr>
<tr>
<td>L.B.</td>
<td>47</td>
<td>M</td>
<td>2</td>
<td>1</td>
<td>0</td>
<td>2</td>
<td>160/90</td>
</tr>
<tr>
<td>F.B.</td>
<td>53</td>
<td>M</td>
<td>1</td>
<td>2</td>
<td>0</td>
<td>2</td>
<td>150/90</td>
</tr>
<tr>
<td>G.J.</td>
<td>54</td>
<td>M</td>
<td>3</td>
<td>3</td>
<td>2</td>
<td>1</td>
<td>120/80</td>
</tr>
<tr>
<td>K.C.</td>
<td>40</td>
<td>M</td>
<td>3</td>
<td>3</td>
<td>1</td>
<td>3</td>
<td>130/90</td>
</tr>
<tr>
<td>J.E.</td>
<td>52</td>
<td>M</td>
<td>6</td>
<td>3</td>
<td>2</td>
<td>2</td>
<td>120/70</td>
</tr>
<tr>
<td>R.C.</td>
<td>36</td>
<td>M</td>
<td>5</td>
<td>3</td>
<td>2</td>
<td>2</td>
<td>140/90</td>
</tr>
<tr>
<td>B.D.</td>
<td>55</td>
<td>F</td>
<td>1</td>
<td>3</td>
<td>2</td>
<td>2</td>
<td>160/90</td>
</tr>
<tr>
<td>T.M.</td>
<td>42</td>
<td>M</td>
<td>1</td>
<td>2</td>
<td>0</td>
<td>1</td>
<td>130/80</td>
</tr>
<tr>
<td>D.A.</td>
<td>42</td>
<td>M</td>
<td>2</td>
<td>3</td>
<td>1</td>
<td>3</td>
<td>140/85</td>
</tr>
</tbody>
</table>

1Angina pectoris was graded 0–3 according to severity: 0, no angina; 1, angina on severe exercise; 2, angina on mild exercise; 3, angina sometimes at rest. Rest angina graded according to frequency of attacks.

2BP arterial blood pressure
### Table II

**TYPE OF OPERATION, RESULTS, AND FOLLOW-UP**

<table>
<thead>
<tr>
<th>Patient</th>
<th>Type of Graft</th>
<th>Follow-up (mths)</th>
<th>Repeat Coronary Angiography and Months-post-op.</th>
<th>LVEDP (mmHg)</th>
<th>LV Angi.</th>
<th>Coronary Arteriography</th>
<th>Collat'ls</th>
</tr>
</thead>
<tbody>
<tr>
<td>V.C.</td>
<td>X</td>
<td>Good</td>
<td>13</td>
<td>—</td>
<td>20</td>
<td>—</td>
<td>N(^1)</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Marked narrowing</td>
<td>Occluded</td>
</tr>
<tr>
<td>M.J.</td>
<td>X</td>
<td>Good</td>
<td>11</td>
<td>Graft patent 7</td>
<td>5</td>
<td>Marked LV thickening</td>
<td>Mild narrowing</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>L - R</td>
</tr>
<tr>
<td>T.W.</td>
<td>X</td>
<td>Good</td>
<td>10</td>
<td>Graft thromosed ()</td>
<td>14</td>
<td>LV aneurysm</td>
<td>Complete obstruction</td>
</tr>
<tr>
<td></td>
<td>X X</td>
<td>Good</td>
<td>9</td>
<td>—</td>
<td>22</td>
<td>Mild mitral reflux</td>
<td>Moderate narrowing</td>
</tr>
<tr>
<td>B.P.</td>
<td>X</td>
<td>Good for 3 months then developed angina</td>
<td>16</td>
<td>Graft patent but increased stenosis 5</td>
<td>7</td>
<td>—</td>
<td>Total occlusion</td>
</tr>
<tr>
<td>G.S.</td>
<td>X X</td>
<td>No improvement</td>
<td>16</td>
<td>Both grafts thromosed 3</td>
<td>13</td>
<td>LV aneurysm</td>
<td>Marked narrowing</td>
</tr>
<tr>
<td>L.B.</td>
<td>X</td>
<td>Good</td>
<td>15</td>
<td>—</td>
<td>14</td>
<td>—</td>
<td>Diffuse narrowing</td>
</tr>
<tr>
<td>F.B.</td>
<td>X</td>
<td>Good</td>
<td>15</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>Total occlusion</td>
</tr>
<tr>
<td>G.J.</td>
<td>X</td>
<td>Good</td>
<td>10</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>Near total occlusion</td>
</tr>
<tr>
<td>K.C.</td>
<td>X</td>
<td>Good</td>
<td>9</td>
<td>—</td>
<td>16</td>
<td>—</td>
<td>Marked narrowing</td>
</tr>
<tr>
<td>J.E.</td>
<td>X</td>
<td>Good</td>
<td>9</td>
<td>Graft patent 3</td>
<td>15</td>
<td>—</td>
<td>Diffuse narrowing</td>
</tr>
<tr>
<td>R.C.</td>
<td>X</td>
<td>Only slight improvement</td>
<td>8</td>
<td>Graft patent 6</td>
<td>16</td>
<td>—</td>
<td>Moderate narrowing</td>
</tr>
<tr>
<td>B.D.</td>
<td>X</td>
<td>Clear improvement but still in pain</td>
<td>8</td>
<td>—</td>
<td>3</td>
<td>—</td>
<td>Minor narrowing</td>
</tr>
<tr>
<td>T.M.</td>
<td>X X</td>
<td>—</td>
<td>8</td>
<td>Both grafts thromosed ()</td>
<td>18</td>
<td>—</td>
<td>Diffuse narrowing</td>
</tr>
<tr>
<td>D.A.</td>
<td>X</td>
<td>Good</td>
<td>11</td>
<td>Graft patent 7</td>
<td>18</td>
<td>LV aneurysm</td>
<td>Mild narrowing</td>
</tr>
</tbody>
</table>

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**Result of operation was judged in terms of amelioration of angina pectoris.**

LVEDP = LV end-diastolic pressure; LAD = left anterior descending; RC = right coronary artery; LC = left circumflex.

\(^1\) Many patients showed minor segmental variations in LV contraction.

\(^1\) N = normal vessel.
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A minimum period of six months following coronary vein-graft surgery. The results of STI measurements preoperatively and at one week, three months, and six months postoperatively are presented.

PATIENTS AND METHODS
The clinical details of 15 patients selected for this study are presented in Table I. Their ages ranged from 31 to 54 and all, except two, were men. All except one had severe angina pectoris on exercise and most had had angina at rest. Nine patients had suffered myocardial infarction previously and three had evidence of an LV aneurysm on angiography. None was markedly hypertensive. Table II shows the type of operation performed, the symptomatic results, and duration of follow-up. Only one patient (G.S.) had electrocardiographic evidence of a myocardial infarct after surgery, while one (F.B.) had an infarct during coronary arteriography before the operation. The results, as regards the alleviation of angina pectoris, were mostly good. In eight patients the grafts were examined two weeks to seven months postoperatively and these were found to be patent in five patients.

The STI were recorded using a 6-channel machine (Cambridge Scientific Instruments type 72112) (Figure). The same sensitivity settings were used throughout. A phonocardiogram was recorded at the second and fourth intercostal spaces along the left sternal border with low frequency filtration (250–1000 Hz). A bipolar ECG lead showing clearly the onset of ventricular depolarization was selected. The carotid displacement curve was recorded simultaneously, using a hand-held polyethylene funnel connected to a piezo-electric transducer with a pulse amplifier (time constant more than 1-6 sec).

Two time intervals were measured: (1) the left ventricular ejection time (LVET) measured from the beginning of the steep rise of the carotid pulse to the dicrotic notch; (2) the electromechanical systole (QA2) measured from the beginning of the Q-wave of the ECG to the first high-frequency component of the second heart sound.

The pre-ejection period (PEP) was calculated by subtracting the LVET from the QA2. The intervals were then corrected for heart rate, using the indices of Weissler et al. (1969), and the subsequent discussion refers to these corrected values. None of the patients had bundle-branch block and all were in sinus rhythm.

The STI were recorded in a supine position in a postabsorptive state. The first recordings were performed two to three days before the operation and subsequently at one week, three months, and six months postoperatively. Eleven patients had been taking β-adrenergic blocking agents before surgery and four continued this medication after the operation. In every instance, however, the drug was withdrawn at least 36 hours before the non-invasive study. No patient was taking digitalis. No significant difference was noted in the arterial blood pressure pre and postoperatively.

For comparison with the patient’s STI the normal values for this laboratory are listed in Table III.

TABLE III  
NORMAL VALUES FOR STI

<p>| | |</p>
<table>
<thead>
<tr>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>QA2</td>
<td>526±6 msec (1 SD)</td>
</tr>
<tr>
<td>LVET</td>
<td>410±14 msec</td>
</tr>
<tr>
<td>PEP</td>
<td>117±11 msec</td>
</tr>
</tbody>
</table>

RESULTS
The results of the STI measurements are presented in Table IV. Comparison with the normal shows that seven patients had LVET below one standard deviation of the normal mean and seven patients had PEP above one standard deviation of the normal mean. No clear relationship was found, however, between the coronary artery involvement and the STI abnormalities. On the other hand, the three patients who had a left ventricular aneurysm all had short LVET. The only striking postoperative changes in STI in the group as a whole were found at one week after surgery when LVET and QA2 were significantly shortened. A consequent increase in PEP/LVET was found, although PEP was unaltered. No significant change in STI was found at three or six months for the group as a whole.
### TABLE IV

STI in 15 patients before and after myocardial revascularization

<table>
<thead>
<tr>
<th>Patient</th>
<th>Control Values</th>
<th>1 Week</th>
<th>3 Months</th>
<th>6 Months</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>LVET</td>
<td>PEP</td>
<td>PEP/ LVET</td>
<td>QA2</td>
</tr>
<tr>
<td>V.C.</td>
<td>430</td>
<td>138</td>
<td>0.341</td>
<td>568</td>
</tr>
<tr>
<td>M.I.</td>
<td>376</td>
<td>159</td>
<td>0.524</td>
<td>535</td>
</tr>
<tr>
<td>T.W.</td>
<td>378</td>
<td>123</td>
<td>0.370</td>
<td>502</td>
</tr>
<tr>
<td>W.D.</td>
<td>399</td>
<td>142</td>
<td>0.432</td>
<td>542</td>
</tr>
<tr>
<td>B.P.</td>
<td>382</td>
<td>127</td>
<td>0.355</td>
<td>507</td>
</tr>
<tr>
<td>G.S.</td>
<td>393</td>
<td>138</td>
<td>0.403</td>
<td>532</td>
</tr>
<tr>
<td>L.B.</td>
<td>415</td>
<td>94</td>
<td>0.225</td>
<td>509</td>
</tr>
<tr>
<td>F.B.</td>
<td>392</td>
<td>121</td>
<td>0.334</td>
<td>513</td>
</tr>
<tr>
<td>G.J.</td>
<td>411</td>
<td>136</td>
<td>0.381</td>
<td>547</td>
</tr>
<tr>
<td>K.C.</td>
<td>411</td>
<td>114</td>
<td>0.323</td>
<td>525</td>
</tr>
<tr>
<td>J.E.</td>
<td>413</td>
<td>128</td>
<td>0.328</td>
<td>542</td>
</tr>
<tr>
<td>R.C.</td>
<td>412</td>
<td>129</td>
<td>0.345</td>
<td>541</td>
</tr>
<tr>
<td>B.D.</td>
<td>381</td>
<td>156</td>
<td>0.502</td>
<td>538</td>
</tr>
<tr>
<td>T.M.</td>
<td>391</td>
<td>116</td>
<td>0.329</td>
<td>507</td>
</tr>
<tr>
<td>D.A.</td>
<td>396</td>
<td>101</td>
<td>0.379</td>
<td>526</td>
</tr>
<tr>
<td>Mean</td>
<td>399</td>
<td>128</td>
<td>0.371</td>
<td>529</td>
</tr>
<tr>
<td>SD</td>
<td>16</td>
<td>18</td>
<td>0.074</td>
<td>19</td>
</tr>
<tr>
<td>SEM</td>
<td>4</td>
<td>5</td>
<td>0.019</td>
<td>5</td>
</tr>
<tr>
<td>P</td>
<td>&lt;0.0005</td>
<td>ns</td>
<td>&lt;0.05</td>
<td>&lt;0.025</td>
</tr>
</tbody>
</table>

All values in milliseconds.
Assessment of left ventricular function following coronary bypass surgery

DISCUSSION

Several reports have appeared in recent years on the pre and postoperative haemodynamics of the left ventricle. Hamilton, Stewart, Gould, and Kennedy (1972) found in a series of 11 patients that the LV end-diastolic pressure, end-diastolic volume, stroke volume, and ejection fraction did not change significantly following bypass surgery and concluded that ventricular performance was preserved rather than improved. In a study utilizing submaximal exercise testing, Manley, Johnson, Flemma, and Lepley (1972), however, found a significant improvement in LV performance in a group of patients with good LV contraction preoperatively. Less striking improvement was found in the presence of moderate to severe impairment of LV contraction, although angina pectoris was usually relieved. Johnson, Flemma, Manley, and Lepley (1970) noted also a fall in the filling pressure of the LV after surgery and an increase in cardiac output on supine leg exercise. Amsterdam et al. (1970) found that the maximum product of heart rate and systolic arterial pressure was increased after surgery and suggested that myocardial oxygen delivery was improved. Rees et al. (1971) reported an increase in LV ejection fraction and velocity of circumferential fibre shortening in eight patients, while in six patients LV function deteriorated, usually due to graft occlusion. Bolooki, Rubinson, Michie, and Jude (1971) examined the effects on LV contractility (Vmax) of two minutes' occlusion of a newly inserted venous graft. In patients who had good LV function Vmax decreased on clamping the graft, while no such effect was noted in patients with poor LV function. Myocardial revascularization procedures have also shown little or no beneficial effect on the LV filling pressures in surviving patients who were in cardiac failure at the time of the operation (Kong, Behar, Peter, and Morris, 1971; Mundth et al., 1971; Spencer et al., 1971). A number of patients, furthermore, sustain myocardial infarction at the time of surgery, although the haemodynamic import of these lesions is still uncertain (Morris et al., 1972).

In general, a short LVET can be regarded to reflect a low cardiac output (Weissler, Peeler, and Roehill, 1961), while a long PEP is an indirect reflection of a slow rate of rise of LV pressure (Talley, Meyer, and McNay, 1971). The ratio PEP/LVET has been suggested as a useful general measure of LV performance (Weissler et al., 1969).

In the present study, all the significant findings for the group as a whole were observed one week postoperatively. Several alternative explanations could account for these changes:

1. a fall in cardiac output;
2. postoperative neurohumoral effects, including increased catecholamine secretion;
3. a temporary deterioration of myocardial function.

A fall in cardiac output could account for the shortening of LVET and, secondarily, QA2. This has been observed in patients undergoing haemodialysis (Prakash and Wegner, 1972). An increased rate of ejection due to excessive catecholamine secretion could also be responsible for the shortening of these intervals as reported following myocardial infarction (Toutouzas, Gupta, Samson, and Shillingford, 1969; Lewis, Boudoulas, Forester, and Weissler, 1972). This seems unlikely, however, in view of the constancy of PEP. Similarly, a fall in the rate of rise of LV pressure would be expected to produce a prolongation of PEP.

Therefore, although other factors cannot be ruled out, the short LVET and QA2 at one week seem to be most easily explained by a fall in output. This may be due, at least partly, to dehydration or other peripheral factors.

Improvement in STI was defined as a shortening of PEP or prolongation of LVET by at least 10 msec to within 1·5 SD of the normal mean, and deterioration in STI was conversely defined as an opposite change of at least 10 msec, resulting in values outside this range.

Using these criteria, the long-term results indicate an improvement of STI in four patients (M.J., T.W., B.D., and G.J.). In all of them angina was improved. Two were investigated postoperatively. In patient M.J. the graft was found to be patent seven months postoperatively. In patient T.W., the graft was occluded only a fortnight after surgery. However, plication of an LV aneurysm, which was done in conjunction with the bypass operation, may account for the STI improvement. Three patients showed worse STI results after surgery (F.B., B.P., and T.M.). In patient T.M. both grafts were found to be occluded two weeks postoperatively. Patient F.B. was the only subject to suffer myocardial infarction during coronary arteriography which was done after the control STI measurements. In patient B.P. the graft was patent at five months, but the arteriosclerotic lesions were found to have progressed. In only one further patient (G.S.) was a graft occlusion found three months after sur-
surgery. He had experienced no amelioration of angina pectoris and the STI were unaltered.

Johnson, O'Rourke, Karliner, and Burian (1972) reported STI changes following myocardial revascularization in a series of 11 patients. They found that LVET was significantly increased after surgery, while PEP was abbreviated. These workers did not find a transient deterioration in the STI values at one week. It is conceivable that postoperative treatment may have been different from the present series in some important aspect, but this cannot be decided from the published data. In three of their patients, a deterioration from initially improved PEP and PEP/LVET values was found corresponding to a recurrence of symptoms.

The results of Johnson et al. (1972) seemed to indicate an improvement in LV function following myocardial revascularization procedures. This was not borne out in the present study, and LV function may therefore have been preserved rather than improved in this series of patients. However, individual changes in STI corresponded with clinical and angiographic assessment of success or failure of the operation. The indications and outcome of saphenous vein bypass grafting of the coronary arteries are still widely debated. Further studies of myocardial integrity following such interventions are therefore necessary. As a supplement to clinical evaluation and invasive measurements, STI offer an attractive physiological method, whose chief advantages are easy repetitability and absence of procedural hazards.

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REFERENCES


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