Angiographic assessment of left ventricular function in patients with ischaemic heart disease without clinical heart failure

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Quantitative assessment of left ventricular function was performed by single plane cineangiography in 13 patients with ischaemic heart disease without clinical heart failure. As compared with normal controls, 7 of 13 patients had a reduced ejection fraction and ‘normalized’ ejection rate, suggesting impaired left ventricular function. These patients could not be differentiated clinically in any way from the remaining 6 patients who had a normal ejection fraction or ejection rate. There was also no correlation between the ejection fraction or ejection rate and the angiographically determined severity of the coronary artery disease. Though the number of patients studied was small, the ejection fraction and ejection rate were found to provide a good correlation with the postoperative and follow-up results. All 3 patients who died, and another 3 patients who developed heart failure, had a reduced ejection fraction and ejection rate. In contrast, of 7 patients who remain alive and well, all but one had normal or near normal ejection fraction and ejection rate. Unlike ejection fraction and ejection rate, there was no correlation between left ventricular end-diastolic pressure and postoperative or follow-up results.

Assessment of left ventricular function in patients with ischaemic heart disease is of considerable importance for their medical and surgical management. However, left ventricular function is difficult to assess by conventional clinical criteria in the absence of overt heart failure. The ejection fraction and the ejection rate, derived from left ventricular volume analysis by angiography (Chatterjee et al., 1971) used during routine cardiac catheterization, provide methods of quantitative assessment of left ventricular function. Abundant evidence now exists to show that these measurements are sensitive indices of left ventricular ‘pump function’ (Bartle, Sanmarco, and Dammann, 1965; Miller and Swan, 1964; Miller, Kirklin, and Swan, 1965; Bunnell, Grant, and Greene, 1965; Dodge and Baxley, 1968, 1969; Hood, Rackley, and Rolett, 1968).

In this communication, the ejection fraction and ejection rate have been used for quantitative assessment of left ventricular function in group of 13 patients with proven coronary artery disease but without clinical heart failure at the time of study. An attempt has been made to relate left ventricular function so determined to the clinical progress and, in those who had surgical treatment, to the postoperative result.

Patients
Clinical details of the patients studied are summarized in the Table. In all patients coronary artery disease was confirmed by selective coronary arteriography. This revealed severe disease (complete occlusion or obvious stenosis and irregularity) of all 3 major coronary vessels in 6 patients, of 2 vessels in 6, and of 1 vessel in 1 patient. No patient had ventricular aneurysm as shown by left ventricular angiography. All patients had angina of effort and 8 had dyspnoea associated with angina. Seven patients gave a history of previous myocardial infarction. Physical signs were normal in all but 2 patients, one of whom had an atrial and the other a third heart sound. There was no evidence of any valve lesion in any patient; all had normal blood pressures and were in sinus rhythm at the time of study. Heart size was normal both clinically and radiologically in all but one patient who had a slightly increased cardio-thoracic ratio (52%) on plain chest x-ray. At the time of investigation no patient had any objective evidence of clinical heart failure (paroxysmal nocturnal dyspnoea, significantly raised jugular venous pressure, hepatomegaly, or peripheral oedema).

The electrocardiogram showed characteristic changes of old myocardial infarction in 8 patients.
and 7 of these had additional ischaemic ST-T changes. Three patients had ischaemic ST-T changes only and in 2 the electrocardiograms were normal. Six patients were taking propranolol, 3 digoxin, and 2 both propranolol and digoxin before investigation. All drugs were discontinued 48 hours before investigation.

Of the 13 patients, a revascularization procedure was performed in 7 in whom there was no symptomatic improvement after a trial of medical treatment including the use of beta blockers (propranolol).

All patients except 1 who died in the immediate postoperative period were followed up for an average of 15-6 months (range 1 week to 32 months—see Table).

### Methods

Left ventricular volumes were calculated from single plane cineangiography in the anteroposterior (AP) projection (Chatterjee et al., 1971). Cineangiography for volume analysis was performed before selective coronary arteriography was undertaken, to exclude the possibility of this latter procedure affecting left ventricular function (Kay et al., 1970).

Left ventricular function was assessed by deriving the following measurements from the beat-to-beat left ventricular volume curve: end-diastolic volume (ml/m²), end-systolic volume (ml/m²), both read directly from the volume curve, left ventricular stroke volume (ml/m²) = end-diastolic volume minus end-systolic volume; ejection fraction = left ventricular stroke volume/end-diastolic volume, and the normalized ejection rate in vol/sec obtained by dividing the ejection rate in ml/sec by the end-diastolic volume (ml/m²). The volume data in these patients were compared with data similarly obtained in normal controls.

The resting left ventricular end-diastolic pressure was measured in all patients, either immediately after the 'a' wave or, if the 'a' wave was not clearly visible, at 0-05 sec after the Q wave of the cardiogram (Braunwald, Fishman, and Courand, 1956).

### Results

Clinical, haemodynamic, coronary arteriographic, and left ventricular volume data together with postoperative and follow-up data are summarized in the Table.

### Table

<table>
<thead>
<tr>
<th>Case No.</th>
<th>Age (yr)</th>
<th>History of angina</th>
<th>Duration of angina (yr)</th>
<th>Dyspnoea with angina</th>
<th>Electrocardiography</th>
<th>Chest x-ray</th>
<th>Coronary arteriography</th>
<th>LVEDP (mmHg)</th>
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</thead>
<tbody>
<tr>
<td>1</td>
<td>55 M</td>
<td>Nil</td>
<td>3</td>
<td>Yes</td>
<td>Ischaemic changes</td>
<td>CTR 44% PV N</td>
<td>0 + + 0</td>
<td>19</td>
</tr>
<tr>
<td>2</td>
<td>54 M</td>
<td>Nil</td>
<td>1</td>
<td>Yes</td>
<td>Normal</td>
<td>CTR 49% PV N</td>
<td>+ + 0</td>
<td>17</td>
</tr>
<tr>
<td>3</td>
<td>58 M</td>
<td>Nil</td>
<td>15</td>
<td>Yes</td>
<td>Ischaemic changes</td>
<td>CTR 43% PV N</td>
<td>+ + +</td>
<td>12</td>
</tr>
<tr>
<td>4</td>
<td>48 M</td>
<td>Nil</td>
<td>9</td>
<td>Yes</td>
<td>Old inferior infarct and ischaemic changes</td>
<td>CTR 47% PV N</td>
<td>+ + +</td>
<td>24</td>
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<tr>
<td>5</td>
<td>46 M</td>
<td>Nil</td>
<td>8</td>
<td>No</td>
<td>Normal</td>
<td>CTR 48% PV N</td>
<td>+ + +</td>
<td>9</td>
</tr>
<tr>
<td>6</td>
<td>48 M</td>
<td>Nil</td>
<td>2</td>
<td>No</td>
<td>Old inferior infarct and ischaemic changes</td>
<td>CTR 45% PV N</td>
<td>+ + +</td>
<td>22</td>
</tr>
<tr>
<td>7</td>
<td>44 M</td>
<td>Yes</td>
<td>5</td>
<td>No</td>
<td>Old inferior infarct and ischaemic changes</td>
<td>CTR 45% PV N</td>
<td>+ + +</td>
<td>15</td>
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<td>42 M</td>
<td>Yes</td>
<td>8</td>
<td>Yes</td>
<td>Old inferior infarct and ischaemic changes</td>
<td>CTR 39% PV N</td>
<td>+ 0 +</td>
<td>12</td>
</tr>
<tr>
<td>9</td>
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<td>Yes</td>
<td>2</td>
<td>Yes</td>
<td>Old inferior infarct and ischaemic changes</td>
<td>CTR 45% PV N</td>
<td>+ + +</td>
<td>35</td>
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<tr>
<td>10</td>
<td>46 M</td>
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<td>Yes</td>
<td>Old inferior infarct and ischaemic changes</td>
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<td>16</td>
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<td>45 M</td>
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<td>2</td>
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<td>CTR 43% PV N</td>
<td>+ + +</td>
<td>6</td>
</tr>
</tbody>
</table>

CTR, cardiothoracic ratio; PV N, pulmonary veins normal; PV +, pulmonary veins dilated; LVEDP, left ventricular end-diastolic pressure; EDV, end-diastolic volume; LVSV, left ventricular stroke volume; EF, ejection fraction; ER, normalized ejection rate.
diastolic volume (Fig. 1a). No patient had a significantly reduced left ventricular stroke volume. In 4 patients (Cases 1 to 4) the ejection fraction was in the normal range; in the remaining 9 patients the ejection fraction was significantly reduced – severely so in 4 patients (Cases 10 to 13: ejection fraction 0.42–0.31) (Fig. 1b). The normalized ejection rate was normal in 6 patients (Cases 1 to 6) and significantly reduced in the remaining 7 patients (Cases 7 to 13) (Fig. 2a).

Patients with low values for ejection fraction and ejection rate (Cases 5 to 13) did not differ clinically in any way from those with normal values. However, with one exception (Case 4), patients with either a history or cardiographic evidence of previous myocardial infarction (Cases 7 to 13) all had low values for ejection fraction and ejection rate. Ejection fraction and ejection rate were similar in patients taking propranolol or digoxin before investigation to values in those who were not on these drugs.

**Relation between ejection fraction, ejection rate, and postoperative and follow-up results**

All 3 patients who died (Cases 10, 11, and 12) had much reduced ejection fraction and ejection rate and the 3 patients who developed heart failure either postoperatively or during the follow-up period (Cases 8, 9, and 13) also had reduced ejection fraction and ejection rate. By contrast, of the 7 patients (Cases 1 to 7) who remain alive and well, without any complications, both the ejection fraction and ejection rate were normal in 4, and in 2, though the ejection fraction was slightly reduced, the ejection rate was normal. Only one patient with a reduced ejection fraction and slightly reduced ejection rate has remained well during the follow-up period. Unlike ejection fraction or ejection rate, left ventricular end-diastolic pressure bore no relation to the postoperative or follow-up results (Fig. 2b). There was no correlation between ejection fraction or ejection rate and the angiographically determined severity of the coronary artery disease, nor was any correlation found between angiographically determined severity and the clinical and haemodynamic findings or the postoperative or follow-up results.

**Discussion**

Knowledge of the state of left ventricular function in patients with ischaemic heart disease is highly desirable for appropriate management. If surgical treatment is carried out in patients with much impaired left ventricular function, the immediate and postoperative complications may be formidable and the chances of survival may be reduced. Similarly, knowledge of left ventricular function may help in the choice of drug therapy,
the usual clinical signs of heart failure. In this study 8 of 13 patients, all without clinical heart failure, had dyspnoea associated with angina. Similarly, left ventricular function may be impaired in the absence of clinical and radiological evidence of cardiac enlargement. Three patients in this study with much reduced left ventricular function had no cardiac enlargement. Rackley et al. (1970) reported similar findings.

Left ventricular end-diastolic pressure has traditionally been used as an index of left ventricular function. Left ventricular end-diastolic pressure of more than 15 mmHg has also been regarded as a contraindication for revascularization surgery in patients with ischaemic heart disease (Sewell, 1966). In this study, however, there was no correlation between it and postoperative results, suggesting that the left ventricular end-diastolic pressure is probably not a reliable index for selection of patients with ischaemic heart disease for surgical treatment. Lack of correlation between left ventricular end-diastolic pressure and postoperative results has also been reported by others (Johnson and Lepley, 1970).

Abundant evidence now exists to show that left ventricular volume analysis by angiographic methods may provide useful information about left ventricular function (Dodge, Hay, and Sandler, 1962; Dodge et al., 1966; Dodge and Baxley, 1968, 1969; Miller and Swan, 1964; Miller et al., 1965; Arvidsson, 1966; Bunnell et al., 1965; Kennedy et al., 1968; Ellison et al., 1970). Measurement of intracavity dimensions and pressure and the wall thickness of the left ventricle during the whole cardiac cycle permits calculation of parameters of left ventricular function such as the maximum velocity of shortening of the contractile element at zero load (Hugenbaltz et al., 1970; Falsetti et al., 1970), the relation between the isovolumic rate of rise of pressure and the integrated isometric stress (Hermann, Singh, and Damann, 1969), and the velocity of the contractile element at peak stress (Vogel, Horgan, and Strahl, 1970; Gaul, Ross, and Braunwald, 1968). Though it has recently been suggested that these parameters may give more direct information about the inotropic and contractile state of the myocardium, their use in routine clinical practice is likely to be restricted because of the more complicated and time-consuming methods involved in their determination. Moreover, in patients with ischaemic heart disease who are likely to have some form of left ventricular asynergy (Herman and Gorlin, 1969) the modulus of the series elastic element may not

FIG. 1 (a) Left ventricular end-diastolic volume in patients with ischaemic heart disease as compared with normal controls. End-diastolic volume was normal in 8 and raised in 5 patients. (b). Ejection fraction in patients with ischaemic heart disease as compared with normal controls and in relation to the postoperative and follow-up results.

since, in the presence of left ventricular dysfunction, a negative inotropic drug (e.g. propranolol) may induce or aggravate heart failure, as occurred in one patient in this study.

Unfortunately, it is often difficult to assess left ventricular function clinically in patients with ischaemic heart disease unless overt heart failure is present. Dyspnoea accompanying angina may be present without

FIG. 2 (a) Normalized ejection rate in patients with ischaemic heart disease as compared with normal controls and in relation to the postoperative and follow-up results. (b) No relation is shown between left ventricular end-diastolic pressure and postoperative and follow-up results in patients with ischaemic heart disease.
remain constant (Hugenholtz et al., 1970) and hence may influence the derived value of maximum velocity of shortening of the contractile element in patients with segmental coronary heart disease. The ejection fraction and the normalized ejection rate, on the other hand – the two parameters most easily derived from volume analysis by the angiographic technique – have been found useful for the evaluation of left ventricular function as a pump in patients with various cardiac disorders (Bartle et al., 1965; Dodge and Baxley, 1968, 1969; Miller et al., 1965). Furthermore, it has been shown that these easily derived indices are as discriminating in assessing left ventricular function as the other parameters, such as the velocity of contractile element shortening at peak stress (W. P. Hood, C. E. Rackley, and E. L. Rolett, 1970; personal communication).

Though the number of patients studied was small, the ejection fraction and the normalized ejection rate were found to provide good correlation with the postoperative and follow-up results. All 3 patients who died and another 3 patients who developed heart failure had reduced ejection fraction and ejection rate. In contrast, of 7 patients who had remained alive and well, all but one had normal or near normal ejection fraction and ejection rate.

That some patients with ischaemic heart disease may have a normal ejection fraction has also been reported by other workers (Rackley et al., 1970; Hermann et al., 1969). In this study, the patients with a normal ejection fraction and ejection rate did not differ significantly in their clinical presentation from those with reduced fraction and rate. There was also no correlation between the ejection fraction or the ejection rate and the severity of the coronary artery disease as judged by selective coronary arteriography. This is in agreement with Linhart et al. (1969) who also failed to find any correlation between the angiographically determined severity of the coronary artery disease and left ventricular function as determined by the response of left ventricular work to a graded infusion of angiotensin.

Though the ejection fraction and rate in this study correlated well with the postoperative and follow-up results, it is fully appreciated that the number of patients studied is too small to draw any conclusion regarding the prognostic value of the ejection fraction and rate in patients with ischaemic heart disease. Nevertheless, it does suggest that some patients with proven coronary artery disease and without the usual clinical signs of heart failure may have much reduced left ventricular function as judged by a low ejection fraction and rate and, as these patients have a worse prognosis, knowledge of left ventricular function is desirable and is of help in planning the management of such patients.

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References


