Assessment of left ventricular function after myocardial infarction

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The low frequency sounds and movements of the heart at the apex were recorded in 51 cases of acute myocardial infarction. Good correlation was found between the changes recorded, the clinical and radiological assessment of left heart function, and the left heart pressures. The technique is painless, safe, reproducible, and we believe gives useful additional information as to the function of the left ventricle after acute myocardial infarction.

In acute myocardial infarction the impairment of left ventricular function depends upon the severity of the acute injury and the amount of disease already present. The severity of the acute injury is usually assessed indirectly from the electrocardiogram and enzyme changes, and the impairment of left ventricular function from the presence or absence of 'failure'. 'Failure' is an unsatisfactory term because its definition and implications are open to dispute, and small changes either of improvement or deterioration are not readily apparent.

Left heart catheterization is rarely practical, ethical, or desirable.

Recording the chest wall pulsations over the apex of the left ventricle with a simultaneous phonocardiogram and electrocardiogram is an investigation which can be made at the bedside. It has been shown to give useful information about heart function in primary myocardial disease (Shah, Gramiak, and Kramer, 1968), valvular heart disease (Nixon and Wooler, 1963; Fleming, 1968; Epstein et al., 1968), hypertension (Beilin and Mounsey, 1962), and ischaemic heart disease (Benchimol and Grey Dimond, 1962).

The purpose of this paper is to describe the findings from a study of 51 cases of acute myocardial infarction and to correlate the information with the results of left heart catheterization in 13 cases of coronary heart disease.

The investigation suggests that the technique is a simple and useful method for assessing day-by-day changes in left ventricular function. It also throws light on the significance of the fourth heart sound in coronary heart disease.

FIG. 1 Diagrammatic representation of apex displacement curves showing normal and the 4 groups of abnormal recordings.

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FIG. 2 Normal pattern. adc = apex displacement curve; RFW = rapid filling wave; F = rapidly filling peak; SFW = slow filling wave; A = 'a' wave of atrial contraction.

FIG. 3 Abnormal pattern; Group 1. 3P = rapidly filling peak.

FIG. 4 Abnormal pattern: Group 1. O in low frequency phonocardiogram channel = opening snap.
Assessment of left ventricular function after myocardial infarction

FIG. 5 Abnormal pattern: Group 3.

FIG. 6 Abnormal pattern: Group 4. Simultaneous recording (reading down) — electrocardiogram lead II, phonocardiogram, apex displacement curve, left atrial pressure, electrocardiogram lead II.

FIG. 7 Left atrial pressure in Group 1, each line representing 5 mmHg, and electrocardiogram lead II. Apex displacement curve from the same patient.
Subjects and methods

The patients were 51 consecutive cases of acute myocardial infarction admitted to the intensive care ward, where a uniform pattern of clinical observation, investigation, and treatment could be followed. They were treated initially by a sleep regimen (Nixon et al., 1968) and digitalis and diuretics were given as required. The diagnosis of acute myocardial infarction was made from serial electrocardiograms and enzyme changes.

The recordings of the heart sounds and pulsations were made on the second and third day of admission and thereafter at weekly intervals, with the patient usually lying in the left lateral or semi-lateral position in arrested expiration. The site was the area of maximum outward pulsation of the apex of the left ventricle as defined by Mackenzie (1902). The aspect of motion of the chest wall selected for study was displacement, and this was recorded, together with the low frequency phonocardiogram, with a previously described (Nixon, Hepburn, and Ikram, 1964) modification of industrial apparatus.

With this apparatus it is possible to use great amplification without distorting the wave form, and this allows the diastolic portion of the curves to be selected for close study. In obtaining tracings from several thousand subjects over a 10-year period, a relatively small number of basic patterns of left ventricular diastolic wave form were encountered. These patterns may be seen in Fig. 1, from which cases of mitral valvar disease and chronic left ventricular aneurysm have been excluded. It should be noted that the patterns are not determined by the nature of the disease affecting the left ventricle but by the severity of the heart disease. Thus, movement from a group 2 pattern to a group 3 pattern is associated with the onset of heart failure. If treatment were given, movement back to group 2 would indicate that improvement was taking place, while movement to group 4 would reveal continuing deterioration. The aetiology of the heart disease could not be deduced from the wave form. Some knowledge of the patient's history might be required to distinguish a group 1 from a group 3 pattern.

The normal in Fig. 2 is recorded as physiological because it is the characteristic finding in the absence of any recognizable heart disease. In essence, the 'a' wave and the rapid filling wave bear a more or less constant relation to each other in health, both increasing or decreasing in size together with variations in heart activity. However, in pathological situations such as ischaemic heart disease without congestive failure the 'a' wave enlarges and the rapid filling wave diminishes in size as shown in Fig. 3 and 4. When congestive cardiac failure supervenes, the 'a' wave diminishes and the rapid filling wave enlarges (Fig. 5). In further deterioration in function the 'a' wave virtually disappears (Fig. 6). There is thus a waxing and waning of the 'a' wave associated with progressive deterioration in left ventricular function.

Results and discussion

In the present study of 51 cases, useful records could be obtained in 49. Normal records were obtained in 2 and abnormal records in 47. There were 31 cases in group 1, 10 cases in group 2, 4 cases in group 3, and 2 cases in group 4.

The clinical signs of heart failure were not found in those patients exhibiting a group 1 or 2 pattern, but were present in those of

FIG. 8 Left atrial pressure in Group 2. A' = aortic pressure; LA = left atrium.

FIG. 9 Left atrial pressure in Group 3.
Assessment of left ventricular function after myocardial infarction

The cardiothoracic ratio on radiological examination was less than 2:1 and the lung fields clear in groups 1 and 2, but there was always cardiac enlargement in groups 3 and 4, with evidence of raised pulmonary venous pressure in some. There appeared to be a good correlation between the clinical and radiological findings and the gradings found on apex recording (Table).

These findings were supported by the trans-septal left heart recordings that were made. The 7 cases in group 1 (Fig. 7) and 2 cases in group 2 (Fig. 8) had mean left atrial pressures below 10 mmHg. The 2 cases in group 3 had left atrial mean pressures between 11 and 20 mmHg (Fig. 9) and the 2 cases in group 4 left atrial mean pressures higher than 21 mmHg (Fig. 6).

This information suggests that movement from group 1 to 2 is unaccompanied by noteworthy change in mean left atrial pressure, but deterioration to group 3 is associated with clear-cut abnormality of the left atrial mean pressure. In group 4 the abnormality is severe. In myocardial infarction the deterioration from group 2 to 4 and its reversal with treatment might take place over a period of hours or days (Fig. 10). This contrasts with, for example, aortic valvar disease or cardiomyopathy where the time scale is measurable in months and years.

The inspection of the low frequency phonocardiogram trace showed fourth heart sounds in two-thirds of the patients (Fig. 11). All

**FIG. 10 Changing pattern over days. Group 4 pattern – 2nd October. Group 3 pattern – 4th October. Group 2 pattern – 8th October.**

**FIG. 11 Apex cardiogram and phonocardiogram results in 51 cases.**

<table>
<thead>
<tr>
<th>No.</th>
<th>4th sound</th>
<th>3rd sound</th>
<th>O.S.</th>
</tr>
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<tbody>
<tr>
<td>GROUP I</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>31</td>
<td>22</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>GROUP II</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>10</td>
<td>7</td>
<td>2</td>
<td>5</td>
</tr>
<tr>
<td>GROUP III</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>4</td>
<td>2</td>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td>GROUP IV</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>2</td>
<td>-</td>
<td>2</td>
<td>-</td>
</tr>
<tr>
<td>Normal</td>
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<td></td>
</tr>
<tr>
<td>2</td>
<td>-</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>Poor records</td>
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<td></td>
</tr>
<tr>
<td>2</td>
<td>-</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>Total</td>
<td>51</td>
<td>31(60%)</td>
<td>8(15%)</td>
</tr>
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</table>

**Table**

<table>
<thead>
<tr>
<th>Group</th>
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<th>Clinical signs of failure</th>
<th>Left atrial pressure (mmHg)</th>
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<tr>
<td>1</td>
<td>31</td>
<td>&lt;50%</td>
<td>Absent</td>
<td>&lt;10</td>
</tr>
<tr>
<td>2</td>
<td>10</td>
<td>&lt;50%</td>
<td>Absent</td>
<td>&lt;10</td>
</tr>
<tr>
<td>3</td>
<td>4</td>
<td>&gt;50%</td>
<td>Present</td>
<td>10-20</td>
</tr>
<tr>
<td>4</td>
<td>2</td>
<td>&gt;50%</td>
<td>Present</td>
<td>&gt;21</td>
</tr>
</tbody>
</table>

*Total* 51

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had abnormally large 'a' waves. Abnormal 'a' waves failed to be accompanied by fourth heart sounds under two conditions. In Fig. 12 the atrial contraction is slow and of low frequency, and thus may not produce audible vibrations, and in Fig. 13 the atrial contraction is so very close to isometric contraction that the fourth and first sounds are almost merged. This may explain the lower figure recorded for fourth sounds than other observers noted (Hill et al., 1969). We felt that the third sound gallop rhythm represented raised end-diastolic pressure and indicated more serious deterioration in function than was reported by Stock (1966).

An interesting finding was a sound transient at that time in diastole which in mitral valve disease would be designated an opening snap; its significance in this context is not clear, but it was noted in 19 per cent of cases.

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References


Requests for reprints to Dr. D. J. E. Taylor, Kent and Canterbury Hospital, Ethelbert Road, Canterbury, Kent.
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