Echocardiographic assessment of left ventricular volume load

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The diastolic closure rate of the mitral echogram was measured in 46 normal subjects and in 24 patients, 13 with aortic regurgitation and 11 with a left-to-right shunt caused by ventricular septal defect (7), persistent ductus arteriosus (3), and peripheral arteriovenous fistula (2). After surgical treatment diastolic closure rate was again determined.

The effect of the increase in left ventricle volume load on diastolic closure rate was examined. The effect of surgical correction was studied and the pre- and postoperative values compared with each other and with the values for diastolic closure rate found in the normal subjects.

The diastolic closure rate was increased in all the patients before operation, the increase was proportional to the size of the shunt or valvular leak, and after corrective surgery it reverted to normal.

It is concluded that the mitral diastolic closure rate can be used as an indirect indicator of left ventricular stroke volume which can be useful in the quantitative assessment of patients with aortic reflux or shunts involving the left ventricle and also postoperatively, as an index of the success of surgical correction.

It has been shown by echocardiography that after early diastolic opening, the mitral valve partially closes in diastole after completion of rapid left ventricular filling.

Three different but closely related mechanisms dependent on the ventricular filling seem to be responsible for this diastolic closure (Fig. 1):

Firstly, there are pressure changes caused by blood flowing rapidly into the ventricle, thus reversing the polarity of the gradient across the valve (Henderson and Johnson, 1912; Little, 1951; Sarnoff, Mitchell, and Gilmore, 1961; Brockman, 1966; Bellhouse, 1972).

Secondly, vortices, which have been generated by resistance to filling, apply forces to the ventricular surfaces of the leaflets (Henderson and Johnson, 1912; Rushmer, Finlayson, and Nash, 1956; Taylor and Wade, 1970; Bellhouse, 1972).

Finally, after the rapid filling period, distension of the ventricle pulls the roots of the papillary muscles away from the ring and through the chordae tendineae attached to their edges—the mitral leaflets are drawn towards each other (Rushmer et al., 1956; Padula, Cowan, and Camishion, 1968).

When the left ventricular volume load is increased as by persistent ductus arteriosus, ventricular septal defect or aortic regurgitation, the haemodynamic fault increases the volume and speed of left ventricular filling and might increase the speed of mitral diastolic closure or diastolic closure rate.

The diastolic closure rate, which can be assessed by ultrasonic methods recording the movements of the anterior mitral leaflet, has been reported to be high in ventricular septal defect and persistent ductus arteriosus (Ultan, Segal, and Likoff, 1967), and in aortic regurgitation (Pridie, Benham, and Oakley, 1971). It was the purpose of this study to look for any quantitative correlation and to see what changes, if any, follow surgical correction.

**Subjects and methods**

Forty-six normal subjects of both sexes, aged 5 to 59 years, and 24 patients were studied. Thirteen of the patients had aortic regurgitation. In none of them was aortic stenosis or a mitral valve lesion present and all but two underwent corrective surgery. The remaining patients had a left-to-right shunt caused by ventricular...
FIG. 1 Diagram of left atrium (LA), left ventricle (LV), and aortic root (Ao) showing the mechanisms of mitral diastolic closure. I - The valve is closed until early diastole when atrial pressure exceeds the ventricular pressure. II - Blood flowing into the ventricle changes the gradient across the valve and generates vortices. III - The distension of the ventricle approximates the leaflets and helps to semiclose the valve.

FIG. 2 Anterior mitral leaflet echogram. Upward movements are opening movements; downward movements are closing movements. Left: 1 - diastolic opening; 2 - diastolic closure; 3 - atrial opening; 4 - presystolic and systolic closure. Middle: A - amplitude of diastolic opening; S - Slow component of diastolic closure; F - fast component of diastolic closure. Right: The diastolic closure rate (DCR) is measured along the faster component. As it is expressed in mm/sec it will be twice the distance indicated in the Figure.
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Semiclosed position (2 – diastolic closure). When the atrium contracts there is a second opening movement (3 – atrial opening) followed by a final closing which starts still in diastole and is completed with systole (4 – presystolic and systolic closure). During systole the echo moves slightly upward until the valve opens again. The diastolic closure (the EF slope of Edler) presents nearly always as a biphasic line (Edler, 1967; Zaky et al., 1968) where the first part is slower than the second (Fig. 2).

Since the early slow component of the diastolic closure represents the posterior movement of the mitral ring in early diastole (Zaky et al., 1968), we measured the diastolic closure rate along the fast component (Fig. 2, right) which represents cusp movement.

Multiple recordings were made applying the transducer to the 3rd, 4th, or 5th left intercostal space, in order to obtain in each subject the maximal opening amplitude of movement, measured as the vertical distance between the beginning of the fast opening upstroke and the maximal forward point (Fig. 2).

The three tracings which showed the maximal amplitude of movement were chosen for measurements of the diastolic closure rate. Measurements on the normal tracings were made by two observers in order to test the reproducibility of the method. There was no statistically significant difference between measurements made twice on the same records by one observer (P > 0.05) or between measurements made blindly by two observers on the same record (P > 0.05).

Six of the normal subjects had recordings taken during five consecutive days. Measurements of the diastolic closure rate gave consistently similar figures (Table 1). Therefore, technically satisfactory records for the remaining 6 patients, catheterization was not performed because they had acute aortic regurgitation needing urgent aortic valve replacement.

Preoperative echograms were recorded in all the patients. After a recovery period of at least 4 weeks, echograms were repeated in 16 of the 22 patients who had been operated upon (3 died and 3 went abroad shortly after operation).

Results

The diastolic closure rate in normal subjects was found to range between 115 and 225 mm/sec with a mean of 173 ± 26.

Patients with a left-to-right shunt (Table 2) had an increased preoperative closure rate ranging from 240 to 425 mm/sec. The diastolic closure rate was increased in relation to the size of the shunt except for Case 11 who will be discussed further. Cases 1 to 5, who had small or moderate shunts, had the lowest values for the diastolic closure rate (240 to 285 mm/sec), whereas Cases 6 to 10 who had large shunts (pulmonary/systemic flow ratio > 2:1) had diastolic closure rates of more than 300 mm/sec (325 to 425 mm/sec – Fig. 3). In patients with ventricular septal defects, a positive correlation was found between the measured flow and the diastolic closure rate (P = 0.017; r = 0.845).

Surgical correction resulted in reduction of the diastolic closure rate to normal (P < 0.001), the exception again being Case 11 (Fig. 4). The postoperative diastolic closure rate ranged between 135 and 250 mm/sec.

The analysis of the diastolic closure rate in patients with aortic regurgitation showed (Table 3) that they all had a raised diastolic closure rate (240 to 435 mm/sec). Cases 12 to 14 with moderate aortic regurgitation had a moderately raised diastolic closure rate (250 to 285 mm/sec). All the other 10 patients (Cases 15 to 24) had severe aortic regurgitation (8 of them had premature closure of the valve, Fig. 5) and their diastolic closure rate was (Fig. 3) close to 300 mm/sec or more (295 to 435 mm/sec).

After aortic valve replacement the diastolic closure rate dropped to normal (Fig. 4 and 6) and this change was significant (P < 0.001), the postoperative values ranging from 155 to 220 mm/sec.

Discussion

An explanation for the increased diastolic closure rate observed in the patients included in this study can be considered on the basis of the ventricular filling mechanisms earlier mentioned as responsible for mitral diastolic closure.

Table 1

<table>
<thead>
<tr>
<th>Day No.</th>
<th>1</th>
<th>2</th>
<th>3</th>
<th>4</th>
<th>5</th>
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<td>120</td>
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<tr>
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<td>6</td>
<td>160</td>
<td>160</td>
<td>160</td>
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</table>

All measurements taken to the nearest 5 mm/sec.

Patients were considered to be representative when taken on a single occasion.

The patients were assessed by clinical means with radiographs and electrocardiograms and 14 of them also had haemodynamic studies. In all the patients with ventricular septal defect, the left-to-right shunt was calculated by the Fick method during cardiac catheterization but in patients with persistent duc tus arteriosus or peripheral arteriovenous fistula it was judged only clinically and by noninvasive tests. Of the 13 patients with aortic regurgitation, 7 underwent cardiac catheterization, the severity of regurgitation being assessed by cineangiography. In the remaining 6 patients, catheterization...
TABLE 2  Size of shunt and pre- and postoperative diastolic closure rate in patients with ventricular septal defect, persistent ductus arteriosus, and peripheral arteriovenous fistula

<table>
<thead>
<tr>
<th>Case No.</th>
<th>Diagnosis</th>
<th>Size of shunt</th>
<th>Diastolic closure rate (mm/sec)</th>
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<tr>
<td></td>
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<td>Clinical</td>
<td>Measured flow</td>
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<tr>
<td>1</td>
<td>Persistent ductus arteriosus</td>
<td>Small</td>
<td>—</td>
</tr>
<tr>
<td>2</td>
<td>Post-traumatic arteriovenous fistula</td>
<td>&quot;</td>
<td>—</td>
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<tr>
<td>3</td>
<td>Ventricular septal defect + pulmonary infundibular stenosis</td>
<td>Moderate</td>
<td>Qp/Qs = 1:4:1</td>
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<tr>
<td>4</td>
<td>Ventricular septal defect + pulmonary infundibular stenosis</td>
<td>&quot;</td>
<td>Qp/Qs = 1:9:1</td>
</tr>
<tr>
<td>5</td>
<td>Persistent ductus arteriosus</td>
<td>&quot;</td>
<td>—</td>
</tr>
<tr>
<td>6</td>
<td>Pulmonary infundibular stenosis</td>
<td>Large</td>
<td>—</td>
</tr>
<tr>
<td>7</td>
<td>Ventricular septal defect + pulmonary infundibular stenosis</td>
<td>&quot;</td>
<td>Qp/Qs = 2:3:1</td>
</tr>
<tr>
<td>8</td>
<td>Ventricular septal defect</td>
<td>&quot;</td>
<td>Qp/Qs = 2:5:1</td>
</tr>
<tr>
<td>9</td>
<td>Ventricular septal defect + patent foramen ovale</td>
<td>&quot;</td>
<td>Qp/Qs = 2:7:1</td>
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<tr>
<td>10</td>
<td>Ventricular septal defect</td>
<td>&quot;</td>
<td>Qp/Qs = 3:5:1</td>
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<tr>
<td>11</td>
<td>Ventricular septal defect + pulmonary infundibular stenosis</td>
<td>&quot;</td>
<td>Qp/Qs &gt; 4:1</td>
</tr>
</tbody>
</table>

* Patient went abroad soon after operation.
† Died.

TABLE 3  Severity of regurgitation and pre- and postoperative diastolic closure rate in patients with aortic regurgitation

<table>
<thead>
<tr>
<th>Case No.</th>
<th>Degree of regurgitation</th>
<th>LV/Ao end-diastolic gradient (mmHg)</th>
<th>LV end-diastolic pressure (mmHg)</th>
<th>Echogram</th>
<th>Mitral valve closure</th>
<th>Diastolic closure rate (mm/sec)</th>
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</thead>
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<td>Clinical Aortogram</td>
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<td>7</td>
<td>Normal</td>
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<td>13</td>
<td>++</td>
<td>45</td>
<td>19</td>
<td>—</td>
<td>—</td>
<td>260</td>
</tr>
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<td>14</td>
<td>—</td>
<td>—</td>
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<tr>
<td>15</td>
<td>Severe ++</td>
<td>0-5</td>
<td>40§</td>
<td>Premature + +</td>
<td>—</td>
<td>295</td>
</tr>
<tr>
<td>16</td>
<td>++ ++</td>
<td>0-5</td>
<td>34</td>
<td>Premature + +</td>
<td>—</td>
<td>325</td>
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<tr>
<td>17</td>
<td>++</td>
<td>0-5</td>
<td>28§</td>
<td>Premature + +</td>
<td>—</td>
<td>350</td>
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<tr>
<td>18</td>
<td>++ ++</td>
<td>—</td>
<td>—</td>
<td>Normal</td>
<td>—</td>
<td>360</td>
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<td>19</td>
<td>++ ++</td>
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<td>28§</td>
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<td>385</td>
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<tr>
<td>20</td>
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<td>40</td>
<td>30§</td>
<td>Normal</td>
<td>—</td>
<td>390</td>
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<tr>
<td>21</td>
<td>++ ++ ++</td>
<td>0-5</td>
<td>34</td>
<td>Premature + +</td>
<td>—</td>
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<tr>
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<td>++ ++ ++</td>
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<td>Premature + +</td>
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<td>Premature + +</td>
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<tr>
<td>24</td>
<td>++ ++ ++</td>
<td>—</td>
<td>—</td>
<td>Premature + +</td>
<td>—</td>
<td>435</td>
</tr>
</tbody>
</table>

* Not operated.
† Patient went abroad soon after operation.
‡ Died.
§ Pressures on operating table.
Diastolic closure rate (each figure taken to the nearest 5 mm/sec).
LV = left ventricle.
Ao = aortic.
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**Fig. 3** Relation between the size of the shunt or the amount of regurgitation and the diastolic closure rate. Except for one patient (Case II) a large shunt or regurgitation assorted with a high diastolic closure rate, above 300 mm/sec. Cases with small or moderate shunt or regurgitation show lower diastolic closure rates though above the upper limit of normal.

Any changes in left ventricular filling caused either by alterations in the flow through the mitral valve, changes in the valve itself, or in left ventricular distensibility can be expected to affect these mechanisms and therefore to alter the diastolic closure.

Conditions in which the filling rate is reduced, such as mitral stenosis, aortic stenosis, and hypertrophic obstructive cardiomyopathy (Stewart, Mason, and Braunwald, 1968), have a slow diastolic closure rate (Gustafson, 1967; Moreyra et al., 1969; Popp and Harrison, 1969; Shah, Gramiak, and Kramer, 1969; Pridie and Oakley, 1970).

Patients with ventricular septal defect or persistent ductus arteriosus with a left-to-right shunt and patients with aortic regurgitation have in common the increased amount of blood flowing into the ventricle during the rapid filling period, either coming through the mitral valve (caused by the shunt) or through the aortic valve (caused by the leak). They will have an increased ventricular filling rate and predictably also a high diastolic closure rate. This high diastolic closure rate was found in the patients studied and is in accordance with the recently reported relation between ventricular filling rate and diastolic closure rate by Layton and associates (1973).

A seemingly more direct way to estimate left ventricular volume load would have been to look at the changes in the transverse dimension after the manner of Gibson (1973). Since the cube of the transverse dimension bears only a fortuitous relation to the volume measured by angiography and since the diastolic closure rate is much more easily achieved, we preferred it for the purpose.

**Fig. 4** Pre- and postoperative values for the diastolic closure rate (DCR). Only the diastolic closure rate of Case II remained above the normal limits after operation.
The correlation found between diastolic closure rate and size of the shunt or amount of regurgitation suggests that any diastolic closure rate value of 300 mm/sec or more indicates a considerable increase in left ventricle volume load. The one apparent exception in the study was Case 11. This was a child who had developed severe infundibular obstruction in the 15 months between catheterization and admission for operation when the echogram was recorded. This same patient had a residual defect and this was the one diastolic closure rate which did not revert to normal after operation.

In conclusion, measurement of the diastolic closure rate is a useful and easily repeatable non-invasive guide to the size of the shunt in ventricular septal defect and persistent ductus arteriosus and to the severity of the leak in aortic regurgitation.

References


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Requests for reprints to Dr. C. M. Oakley, Department of Medicine, Royal Postgraduate Medical School, Hammersmith Hospital, London W12.
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