Noninvasive Assessment of Left Ventricular Function from the Mitral Valve Echogram

Relation of Final Anterior Mitral Leaflet Closing Velocity to Peak dp/dt and Aortic Velocity

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SUMMARY Since final mitral valve (MV) closure and aortic ejection velocity are mediated by the same forces in early left ventricular (LV) contraction, the rate of final MV closure (BC slope) should reflect LV performance. We first verified whether peak final closing velocity (ds/dt) of the anterior MV leaflet (AMVL) is related to peak aortic ejection velocity (V) and LV dp/dt in 18 open-chest dogs. We then checked the validity of the relations in man. Our approach was to measure peak ds/dt, peak aortic acceleration (dV/dt) and peak LV dp/dt using electronic differentiation of analog signals of the AMVL echogram, V and LV pressure. In dogs, resting ds/dt averaged 26.9 ± 9.0 (SD) cm/sec and changed significantly (P < 0.001) after isoproterenol, propranolol, coronary ligation and aortic cross-clamping. We found good (P < 0.001) correlations between ds/dt and V (r = 0.82), dV/dt (r = 0.67) and dp/dt (r = 0.73). In man, resting ds/dt averaged 25.5 ± 1.6 cm/sec in six normals. In 40 patients with coronary artery disease, resting ds/dt was lower (15.7 ± 4.4 cm/sec; P < 0.001) in the 19 with resting LV end-diastolic pressure (LVEDP) > 12 mm Hg. Resting ds/dt correlated closely with V (r = 0.82, N = 10), dp/dt (r = 0.93, N = 6), resting LVEDP (r = -0.67, N = 40), angiographic ejection fractions (r = 0.62, N = 40) as well as manually obtained BC slopes (r = 0.93, N = 40). Thus, final MV closing velocity provides a useful and simple means for the objective noninvasive assessment of LV performance.

THE MITRAL VALVE (MV) is the easiest structure to recognize, localize and record on M-mode echocardiography,1 Edler,2, 3 first pointed out the distinctive diastolic motion pattern of the anterior MV leaflet (AMVL) and its distortion in mitral stenosis. In Edler's original lettering of the MV echo,4 the AC portion denoted AMVL closure. Later, Zaky, Steinmetz and Feigenbaum5 indicated that this AC limb is made up of two components, the AB and BC portions corresponding to atrial relaxation and early left ventricular (LV) systole, respectively, with a brief interruption at B which coincides with the onset of LV systole and the R wave of the electrocardiogram. It is generally agreed that the point C,1 or C2,6, 6 represents complete MV closure. Although abnormalities of the MV echo1 are diagnostic in various forms of valvular and congenital heart disease, attempts to use various parameters from the AMVL echo (amplitude of separation of leaflets,7 DE slope,4 EF slope,9 PR-AC interval10) to quantitate LV function have not gained the same popularity as measurements of LV dimensions.11, 12 Several investigators13-18 have recently measured the AC or BC slopes of AMVL closure using a variety of methods (table 1) and some authors13, 14 have suggested that it may provide an index of cardiac function.

A consideration of cardiac mechanics suggests that the dynamics of MV closure are determined by the same forces in the early phase of LV contraction which generate LV pressure and culminate in aortic ejection. This concept is based on: 1) the proposal by Rushmer,20 in 1964, that the initial impulse of LV ejection was a useful index of myocardial performance; 2) the suggestion by Yoshitoki et al.,18 in 1965, using Doppler ultrasound, that the rate of systolic closure of the AMVL reflected LV contractility; and 3) the finding by Noble et al.,21 in 1966, that the maximum acceleration of blood ejected from the left ventricle is a "sensitive index of LV contractile function"11 in conscious dogs. Since measurement of the BC slope of the AMVL closure manually from tracings made at 200 mm/sec was tedious and susceptible to interobserver error, we used electronic differentiation of time analog signals of the AMVL echo to obtain instantaneous AMVL velocities.

The aims of this study were: 1) to determine whether peak AMVL closing velocity (ds/dt) reflects changes in LV performance in open-chest dogs, as measured by two hemodynamic variables that can be accurately measured and easily altered, namely, the maximum rate of rise of LV pressure (peak LV dp/dt) and peak aortic velocity (V); 2) to carry out an initial assessment of the applicability of peak AMVL closing velocity as a noninvasive tool for the objective evaluation of LV performance in man; 3) to compare peak AMVL closing velocity to some commonly measured indices of LV function such as cardiac output, end-diastolic pressure (EDP) and ejection frac-

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Supported in part by a grant-in-aid (MP-5544) from the Medical Research Council of Canada.

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Received December 19, 1977; revision accepted July 3, 1978.

tion as well as some other parameters from the AMVL echo suggested to reflect LV function in man.

**Methods**

**Animal Studies**

Eighteen mongrel dogs weighing 18–34 kg were anesthetized with intravenous sodium thiopental (10 mg/kg) and chloralose (80 mg/kg) and ventilated with room air. Dextrose (5%) in saline was infused at 50 ml/hr. The heart was exposed through a limited transverse thoracotomy at the sixth interspace and suspended in a pericardial cradle. Lead 2 of the ECG was monitored continuously.

**Ultrasound Recordings**

The AMVL echograms were recorded using an echograph (Ekoline 20A Ultrasonoscope, Smith Kline Instruments (SKI) Co.) with a frequency output of 2.25 MHz and repetition rate 1000 pulses/sec and equipped with a 0.5 inch diameter transducer prefocused at 10 cm. The AMVL echo signal was isolated by a time-analog pre-amplifier module (SKI) with optional adjustment of gate width. The time delay of the ultrasound system was less than 1 msec, and the inherent frequency response limitation occurs above 500 Hz (data from SKI Co). The AMVL echo was recorded from the epicardial surface of the right ventricle as done by Pohost et al. and was observed at a depth of 3–6 cm. Adequate coupling gel was used to form a cushion so as to prevent intermittent loss of contact between transducer and epicardial surface. The transducer was held with minimal pressure, but rigidly enough to minimize relative and translational motion. Continuous analog signals were recorded on the Electronics for Medicine (E for M, DR8) photographic recorder and a magnetic tape recorder together with simultaneous samples of the MV echo on the strip chart (Honeywell) at speeds of 50–200 mm/sec. The height of the analog signal was calibrated by moving the gate 1 cm and verified vs echoes from a perspex slab with a saline well.

**Velocity Recordings**

In the first six dogs, an electromagnetic catheter-tipped probe (SE Medic) was introduced via the carotid artery and its tip positioned 3 cm above the aortic valve and in the middle of the aortic root using x-ray fluoroscopy. Zero calibration was obtained with the catheter in saline before introduction into the blood stream. Peak velocity was read directly on the flowmeter (SE Medic) and phasic signals recorded on E for M and magnetic tape recorders. Linearity of response was confirmed for velocities up to 300 cm/sec.

In the remaining 12 dogs, electromagnetic external cuff probes (1–1.6 cm diameter) were placed around the aortic roots and phasic flow velocity monitored on a pulsed logic flowmeter (Biotronix) with optimal adjustments of gain and frequency. Output signals were recorded on a pen recorder (Beckmann) and the magnetic tape. Zero calibration was obtained with the probe immersed in saline or blood, and later after briefly cross-clamping the ascending aorta proximal to the cuff. Further calibration in graduated steps at flow rates of 0–4 l/min for each cuff was done postmortem by pumping blood through a section of the aortic root obtained from every dog. Linearity of response with flow velocities up to 4 l/min was confirmed. Both cuff and catheter probe velocities were recorded in dog 4, and they were in agreement (V-cuff = 1.00 V-catheter – 0.50, r = 0.99, N = 13).

**Pressure Recordings**

An SF-1 high fidelity transducer-tipped pressure catheter (Statham) was introduced via the femoral artery and positioned near the apex of the LV using x-ray fluoroscopy. The mid-heart was used as zero reference level. Signals were balanced against those of an external pressure transducer (Statham 23 PDb) connected directly to the catheter and recorded on the E for M recorder and magnetic tape. Both LV pressure and its first derivative (dp/dt) were monitored on the E for M machine and recorded at 200 mm/sec.

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**Table 1. Summary of Values of Late Closure of the Anterior Mitral Valve Leaflet from the Literature**

<table>
<thead>
<tr>
<th>Year</th>
<th>Group</th>
<th>Reference</th>
<th>Method</th>
<th>Subject</th>
<th>AMVL Closing Velocity (cm/sec)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Definition</td>
</tr>
<tr>
<td>1965</td>
<td>Yoshitoshi et al.</td>
<td>13</td>
<td>Doppler</td>
<td>Man</td>
<td>? AC</td>
</tr>
<tr>
<td>1972</td>
<td>Buynkosturk et al.</td>
<td>14</td>
<td>Echo: manual</td>
<td>Man</td>
<td>AC or BC</td>
</tr>
<tr>
<td>1974</td>
<td>Gordon et al.</td>
<td>15</td>
<td>Cineangiographic</td>
<td>Dog</td>
<td>? BC</td>
</tr>
<tr>
<td>1974</td>
<td>Emerson et al.</td>
<td>16</td>
<td>Echo: computer tracking</td>
<td>Man</td>
<td>? BC</td>
</tr>
<tr>
<td>1974</td>
<td>Upton et al.</td>
<td>17</td>
<td>Echo: manual digitization</td>
<td>Man</td>
<td>? AC</td>
</tr>
<tr>
<td>1976</td>
<td>Lalani and Lee.</td>
<td>19</td>
<td>Echo: electronic</td>
<td>Dogs</td>
<td>AC</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Man</td>
<td>BC</td>
</tr>
</tbody>
</table>

? = not specified.
Abbreviation: AMVL = anterior mitral valve leaflets.
Interventions

Simultaneous baseline recordings of ECG, AMVL echo, aortic velocity and intracardiac LV pressure were made on magnetic tape in all dogs. Recordings were then repeated in sequence after isoproterenol (1 μg/min) infusion, three boluses of propranolol (0.05 mg/kg I.V.) given 5 minutes apart, acute ligation of the left anterior descending coronary artery, and acute cross-clamping of the descending aorta. Sufficient time was allowed between interventions and before recordings to achieve "steady state" conditions.

Playback, Signal Processing and Calculations

The AMVL closure slope was measured as the mean slope of the line joining points B and C on five consecutive clear echoes on 50 mm/sec strip chart records. In some cases, the AC portion was a smooth straight line so that AC and BC slopes were identical.

The playback assembly is depicted in figure 1. The frequency response of the recorders was in excess of maximum frequencies encountered in these experiments. Mitral echo signals were filtered through "low-pass" filters (zero db gain and 12 db per octave attenuation factor and cut-off frequency of 40–60 Hz). By viewing filtered and unfiltered signals, filter cut-off was adjusted to eliminate as much noise as possible without affecting the closure slope of the AMVL echo by excessive damping. The filtered AMVL echo was then differentiated electronically and the maximal deflection corresponding to the BC slope of the AMVL echo was designated peak AMVL closure velocity or ds/dt. Pressure signals were differentiated without prior filtering and the maximum deflection of the first derivative corresponding to the rapid rise of LV pressure was designated peak dp/dt. EDPs and instantaneous LV systolic pressures corresponding to peak dp/dt were also measured and peak dp/dt normalized to instantaneous developed pressures. Aortic velocity signals were differentiated without prior filtering and the peak deflection of the derivative corresponding to the upstroke portion was read as peak aortic acceleration, or dV/dt.

For each variable, the heights of three consecutive deflections were measured directly from the zero line and multiplied by the respective calibration factors, allowing for amplification on differentiation. A beat-to-beat analysis was performed. Only consecutive beats in sinus rhythm were analyzed. A constant time delay of about 8 msec was introduced on differentiation and about 25 msec on filtering.

The variables were compared by linear regression analysis, and paired and unpaired t tests.

Clinical Studies

The AMVL echoes were recorded on magnetic tape and on strip charts (50–200 mm/sec) using standard techniques in 1) six normal subjects and 2) 52 patients at the time of diagnostic right and left heart catheterization. All subjects gave informed consent for the studies, and all were in sinus rhythm. In 35 patients, echoes were obtained immediately before Fick's cardiac output measurements just before LV angiography. Coronary angiography was done in 40 patients with coronary artery disease. The LV systolic pressures and LVEDP were measured in all patients.
before and after LV angiography using the mid-chest as zero reference.

In 10 patients, aortic ejection velocity was measured using an electromagnetic catheter-tipped probe (SE Medic) introduced via the femoral artery. In six of these patients, LV pressure was measured using an SF1 intracardiac transducer-tipped catheter. The signals were taped with simultaneous AMVL echoes.

The LV cineangiograms were replayed in 36 patients and ejection fractions calculated from end-diastolic and end-systolic outlines using the area length method of Sandler and Dodge.22

The taped echoes were processed as in the dog studies and ds/dt was compared with cardiac output, LVEDP, LV ejection fractions, peak LV dp/dt, peak V and peak dV/dt. Statistical analysis was done as for the dog studies.

Results

Dog Studies

Classical M-shaped configurations of the AMVL were observed when heart rates were less than about 100 beats/min. At greater rates, the A wave is lost and E and A points merge so that a distinct EF slope is not seen, as found by others.14,23 A drawing of the AMVL echo and its first derivative is shown in figure 2. Proper timing of components of the AMVL echo was facilitated by the simultaneous ECG and pressure tracings.5 A sample of playback signals shows a delay in the echo derivative due to filtering and differentiation (fig. 3). Hemodynamic and echo data in the 18 dogs are summarized in table 2. All values are given as mean ± 1 sd.

Over the control period, ds/dt averaged 26.9 ± 9.0 cm/sec. In some dogs, peak dp/dt remained depressed for up to 1 hour with alternans in pressure, velocity and mitral echo in spite of a normal ECG. Such beats were excluded from this analysis. Katz and Mills24 also reported alternans in aortic velocity in anesthetized dogs.

After isoproterenol, similar percent increases occurred in peak ds/dt (48%) as in dp/dt (48%), peak V (40%) and peak dV/dt (50%), with lesser increases in heart rate (7%) and LV systolic pressure (8%). After propranolol, similar percent decreases from post-isoproterenol values were seen in ds/dt (46%) as in peak LV dp/dt (53%), peak V (47%) and peak dV/dt (54%) with lesser decreases in heart rate (25%) and LV systolic pressure (22%), these absolute values being lower than baseline. After coronary ligation in six dogs, further decreases from post-propranolol values occurred in ds/dt (46%) and LV dp/dt (42%), peak V (26%) and peak dV/dt (45%) but LV systolic pressure also dropped markedly (47%) while heart rate showed less change (11%). Both peak V and acceleration decreased several seconds before peak dp/dt after coronary ligation, as found by Noble et al.21 During acute cross-clamping of the descending aorta, ds/dt decreased further to 7.2 cm/sec as did peak dp/dt, peak V and peak dV/dt but LV pressure increased (81–98 mm Hg systolic) while heart rate (96 vs 96 beats/min) did not change significantly. This suggests that changes in ds/dt were not directly due to changes in heart rate or LV pressure.

Relation Between Echo and Hemodynamic Parameters

The correlation coefficients are summarized in table 3. Directional changes in ds/dt following interventions correlated significantly (P < 0.001) with those in

![Figure 2](http://circ.ahajournals.org/)

**Figure 2.** A drawing of an anterior mitral valve leaflet (AMVL) echo and its instantaneous first derivative. Conventional lettering after Edler.3 The derivative is inverted with respect to the input signal. The arrow indicates the point from which peak ds/dt corresponding to the peak BC slope was read. The dotted vertical line indicates the timing of the B point and R wave. * = peak EF slope, ** = peak DE slope.
Table 2. Summary of Hemodynamic and Echo Data in 18 Dogs

<table>
<thead>
<tr>
<th>Group</th>
<th>Heart rate (beats/min)</th>
<th>Systolic pressure (mm Hg)</th>
<th>End-diastolic pressure (mm Hg)</th>
<th>Peak dp/dt (mm Hg/sec)</th>
<th>Peak ds/dt (cm/sec)</th>
<th>Peak V (cm/sec)</th>
<th>Peak dV/dt (cm/sec²)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Control (18 dogs)</td>
<td>127 ± 19</td>
<td>127 ± 28</td>
<td>9.4 ± 7.0</td>
<td>1963 ± 632</td>
<td>26.9 ± 9.0</td>
<td>61.8 ± 16.4</td>
<td>1634 ± 935</td>
</tr>
<tr>
<td>Isoprenaline (18 dogs)</td>
<td>136 ± 17*</td>
<td>137 ± 45</td>
<td>5.8 ± 4.0*</td>
<td>2883 ± 1122*</td>
<td>39.7 ± 14.6*</td>
<td>86.4 ± 27.2*</td>
<td>2454 ± 1333*</td>
</tr>
<tr>
<td>Propranolol (18 dogs)</td>
<td>102 ± 22*</td>
<td>107 ± 33*</td>
<td>11.2 ± 3.4*</td>
<td>1344 ± 614*</td>
<td>20.8 ± 10.2*</td>
<td>45.6 ± 16.6*</td>
<td>1131 ± 828*</td>
</tr>
<tr>
<td>LAD ligation (6 dogs)</td>
<td>91 ± 31</td>
<td>57 ± 15*</td>
<td>12.3 ± 2.3</td>
<td>780 ± 242*</td>
<td>11.3 ± 3.6*</td>
<td>33.8 ± 10.7*</td>
<td>626 ± 294*</td>
</tr>
<tr>
<td>Cross-clamped descending aorta (three dogs)</td>
<td>96 ± 20</td>
<td>81 ± 26</td>
<td>—</td>
<td>849 ± 118</td>
<td>13.2 ± 0.7</td>
<td>32.8 ± 11.0</td>
<td>656 ± 284</td>
</tr>
</tbody>
</table>

Values as mean ± sd.

†Aortic pressures.

Abbreviations: See text.

**Figure 3.** Timing of first derivative of the anterior mitral valve leaflet (AMVL) echo with pressure, velocity and ECG signals. A time delay in the echo derivative is due to filters used and to a lesser extent to differentiation. Arrows indicate points at which peak ds/dt were measured.
peak dp/dt (r = 0.73), peak V (r = 0.82) and peak dV/dt (r = 0.67). These correlations were better in individual dogs, with r values between 0.95 and 0.99, and P values < 0.001. The values of peak dp/dt and peak V are plotted for all dogs in figure 4 and for three individual dogs in figure 5. The scatter in composite plots (fig. 4) increased as dp/dt increased with isoproterenol and contractions became more vigorous, reflecting amplification of error upon differentiation. Some relative motion of aortic catheter or cuff probes also occurred after isoproterenol and may explain the scatter in velocity readings as peak dp/dt increased.

Second derivatives of the echo and LV pressure or normalization of peak dp/dt to instantaneous developed pressures and EDP did not improve correlations, so the data are not presented.

**Table 3. Summary of Correlation Coefficients in the Animal Study**

<table>
<thead>
<tr>
<th></th>
<th>Peak V</th>
<th>Peak dV/dt</th>
<th>Peak dp/dt</th>
</tr>
</thead>
<tbody>
<tr>
<td>Peak mitral closing</td>
<td>0.82</td>
<td>0.67</td>
<td>0.73</td>
</tr>
<tr>
<td>velocity (ds/dt)</td>
<td>P &lt; 0.001</td>
<td>P &lt; 0.001</td>
<td>P &lt; 0.001</td>
</tr>
<tr>
<td></td>
<td>n = 295</td>
<td>n = 288</td>
<td>n = 380</td>
</tr>
<tr>
<td>Peak aortic ejection</td>
<td>0.99*</td>
<td>0.83</td>
<td>0.79</td>
</tr>
<tr>
<td>velocity (V)</td>
<td>P &lt; 0.001</td>
<td>P &lt; 0.001</td>
<td>P &lt; 0.001</td>
</tr>
<tr>
<td></td>
<td>n = 13</td>
<td>n = 299</td>
<td>n = 302</td>
</tr>
<tr>
<td>Peak aortic acceleration</td>
<td>0.83</td>
<td>—</td>
<td>0.71</td>
</tr>
<tr>
<td>(dV/dt)</td>
<td>P &lt; 0.001</td>
<td>P &lt; 0.001</td>
<td>P &lt; 0.001</td>
</tr>
<tr>
<td></td>
<td>n = 229</td>
<td>n = 295</td>
<td></td>
</tr>
</tbody>
</table>

*Catheter vs cuff probe.

**Clinical Studies**

**Manual BC Slope vs Electronic AMVL Closing Velocity**

Values of BC slope derived manually from strip chart records correlated closely with electronically derived ds/dt from AMVL echo records in three dogs (3, 4 and 5) and 28 patients (r = 0.93, N = 40), as shown in figure 6. Manually obtained BC slopes were numerically less than corresponding electronic ds/dt as might be expected, since the former measures mean velocity and the latter the peak closing velocity.

Resting peak AMVL closing velocity, or ds/dt, averaged 25.5 ± 1.6 (range 23.6–28) cm/sec in six normal subjects. The data from 40 patients with coronary artery disease are shown in table 4. Their ds/dt averaged 21.6 ± 7.3 (range 8.8–38.5) cm/sec. On subdividing these 40 patients on the basis of whether their resting LVEDP found at catheterization was elevated (> 12 mm Hg) or not, those 21 patients with resting LVEDP < 12 mm Hg (group 1) had an average peak ds/dt of 27.0 ± 4.8 cm/sec, vs 15.7 ± 4.4 cm/sec in those 19 patients with resting LVEDP > 12 mm Hg (group 2). This difference between the two groups was significant (P < 0.001), as was that between group 2 and the normal subjects.

In 12 patients with other diseases, peak ds/dt values were: 1) 35.7 ± 13.8 (range 21.0–55.0) cm/sec in seven cardiomyopathy patients (four asymmetric septal hypertrophy, three Syndrome X — a syndrome of atypical chest pain in young women with normal coronary angiograms and ST depression induced by
Figure 5. Relation of peak anterior mitral valve leaflet (AMVL) closing velocity to peak left ventricular dp/dt (left panel) and peak aortic ejection velocity (right panel) in three dogs representative of the series.

Figure 6. Relation of manual anterior mitral valve leaflet (AMVL) closing slope (BC) to the corresponding electronic peak anterior mitral valve leaflet closing velocity (ds/dt) in dogs and man.
### Table 4. Hemodynamic, Angiographic and Echocardiographic Data in 40 Patients with Coronary Artery Disease

<table>
<thead>
<tr>
<th>Case no.</th>
<th>Sex</th>
<th>Age (min)</th>
<th>CO (l/min)</th>
<th>Heart rate (beats/min)</th>
<th>LV angiography</th>
<th>Coronary angiography</th>
<th>Site of disease (%)</th>
<th>Peak ds/dt (cm/sec)</th>
<th>PR-AC (sec)</th>
<th>EF slope (cm/sec)</th>
<th>DE slope (cm/sec)</th>
</tr>
</thead>
<tbody>
<tr>
<td>LVEDP (mm Hg)</td>
<td>Pre angio</td>
<td>Post angio</td>
<td>Ejection fraction</td>
<td>ACS</td>
<td>Mainstem LCA</td>
<td>CAB to LAD, Cx, RCA</td>
<td>CAB to LAD, Cx, RCA</td>
<td>CAB to LAD, Cx, RCA</td>
<td>CAB to LAD, Cx, RCA</td>
<td>CAB to LAD, Cx, RCA</td>
<td></td>
</tr>
<tr>
<td>1</td>
<td>55/M</td>
<td>55</td>
<td>7.6</td>
<td>8 11</td>
<td>0.06</td>
<td>0</td>
<td>LAD, Cx</td>
<td>+</td>
<td>25.7</td>
<td>0.11</td>
<td>28.5</td>
</tr>
<tr>
<td>2</td>
<td>57/M</td>
<td>68</td>
<td>4.4</td>
<td>8 11</td>
<td>0.48</td>
<td>0</td>
<td>LAD</td>
<td>26.9</td>
<td>0.09</td>
<td>24.9</td>
<td>39.1</td>
</tr>
<tr>
<td>3</td>
<td>58/F</td>
<td>73</td>
<td>3.8</td>
<td>5 20</td>
<td>0</td>
<td>0</td>
<td>LAD, Cx</td>
<td>20.0</td>
<td>0.11</td>
<td>12.5</td>
<td>25.0</td>
</tr>
<tr>
<td>4</td>
<td>56/F</td>
<td>72</td>
<td>4.5</td>
<td>5 17</td>
<td>0.59</td>
<td>0</td>
<td>LAD</td>
<td>26.7</td>
<td>0.07</td>
<td>30.0</td>
<td>55.0</td>
</tr>
<tr>
<td>5</td>
<td>55/M</td>
<td>70</td>
<td>6.9</td>
<td>8 12</td>
<td>0.68</td>
<td>+</td>
<td>CAB to LAD, Cx, RCA</td>
<td>24.4</td>
<td>0.13</td>
<td>29.3</td>
<td>40.0</td>
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<tr>
<td>6</td>
<td>58/M</td>
<td>85</td>
<td>4.1</td>
<td>6 22</td>
<td>0.57</td>
<td>0</td>
<td>CAB to LAD, Cx, RCA</td>
<td>23.0</td>
<td>0.14</td>
<td>30.1</td>
<td>22.6</td>
</tr>
</tbody>
</table>

**Group 1 (Resting LVEDP < 12 mm Hg)**

- **Total (Mean ± sd)**
  - **Group 1**
    - 55 | 74 | 5.9 | 8 16 | 0.58 | 27.0 | 0.10 | 25.2 | 35.4
    - ±7 | ±9 | ±2.0 | ±2 | ±5 | ±0.08 | ±4.8 | ±0.02 | ±4.4 | ±7.7

- **Group 2**
  - 53 | 74 | 4.9 | 18 26 | 0.46 | 15.7 | 0.06 | 17.4 | 27.8
  - ±8 | ±14 | ±1.7 | ±6* | ±7* | ±0.19† | ±4.4 | ±0.02 | ±5.4* | ±6.1†

*P < 0.001 and †P < 0.005, respectively, compared to value above.

Abbreviations: ACS = abnormally contracting segments (0 = nil; + = mild dyskinesis; ++ = aneurysm); angio = angiography; CAB = coronary artery bypass; CO = cardiac output; Cx = left circumflex coronary artery; EDP = end-diastolic pressure; LAD = left anterior descending coronary artery; LCA = left coronary artery; LV = left ventricular; RCA = right coronary artery.
atrial pacing); 2) 32.7 cm/sec in a patient with a ventricular septal defect; 3) 40 and 33.4 cm/sec, respectively, in two patients with aortic insufficiency, and 4) 18.7 and 22.5 cm/sec, respectively, in two patients with mitral stenosis. In these patients, the corresponding manual BC slopes were high (65.4 and 77.9 cm/sec, respectively). Similar high values were found by Yoshitoshi et al.18 using Doppler, and probably reflect a resolution problem.

In six patients who had intracardiac LV pressure measurements, ds/dt and dp/dt averaged 20.1 ± 1.7 cm/sec and 1372 ± 220 mm Hg/sec and showed good linear correlation (r = 0.93). In the 10 patients in whom aortic velocity was measured by the catheter probe, ds/dt and aortic ejection velocity averaged 23.6 ± 2.8 cm/sec and 36.7 ± 4.4 cm/sec, respectively, and also showed good linear correlation (r = 0.82).

Correlation coefficients between variables in the human study are tabulated in Table 5. A lesser correlation was found between ds/dt and angiographic ejection fractions (r = 0.62) and resting LVEDP (r = -0.67) but they were significant (P < 0.001).

In order to determine whether ds/dt provides similar information as the DE slope, EF slope and PR-AC interval, these were measured from the AMVL echo and the first derivative tracings in the human study and are shown in Table 4. Correlation coefficients between these parameters and ds/dt and other hemodynamic variables are summarized in Table 5.

**Discussion**

The clinical value of echocardiography lies in its noninvasive methodology, providing an opportunity to repeat observations in serial fashion. However, the use of M-mode echocardiography for the quantitative analysis of LV function requires experienced and meticulous technique. Furthermore, the conventional echo method for estimating LV function from measurements of internal LV dimensions is limited in the setting of coronary artery disease by assumptions of ellipsoid geometry, a constant relationship between the axes, and uniformity of contraction. One approach, proposed by Lalani and Lee, assumes a direct relation between the final closure slope of the AMVL echo and aortic ejection velocity and acceleration. Our results confirm that such a relation exists. In addition, we have shown that the final AMVL closing velocity provides some separation of patients with coronary artery disease (Table 4).

Conventional hemodynamic methods97-31 used to characterize LV mechanical and pump function require invasive methodology. While Noble et al.21 suggested that aortic acceleration may be a sensitive index of mechanical function, it also requires an invasive approach. In our animal studies, we found close correlations between peak AMVL closing velocity and 1) peak aortic velocity and acceleration and 2) peak dp/dt. Similar directional changes in these variables were produced by alterations of contractile state. In the human studies, we found similar correlations between these variables, but peak AMVL closing velocity showed weaker correlations with ejection fraction, resting LVEDP and cardiac output. However, both dp/dt and aortic acceleration are load-dependent and have limitations in identifying LV dysfunction. Similarly, resting LVEDP, like ejection
fraction and cardiac output, does not adequately separate normal from diseased ventricles.29, 34 We did not control loading conditions in our animal studies. It is of interest that acute aortic cross-clamping produced significant decreases in ds/dt in dogs. While further studies need to be done under different loading conditions, our results nevertheless suggest that final AMVL closing velocity is a useful noninvasive and objective index of LV performance.

Despite varied methodologies used by other investigators,13-16 the range of ds/dt in our study was similar to other studies (table 1). Other parameters from the AMVL echo have also been suggested to reflect LV function.1, 7, 9, 10 Because MV motion is influenced by MV flow7, 9 and hemodynamic factors such as the interplay between left atrial and LV pressures. Thus, Fischer et al.7 found reduced AMVL excursion and leaflet separation in the presence of LV dysfunction. The EF slope is thought to reflect changes in MV flow and cardiac output but is also influenced by LV compliance.1, 9 Decreased LV compliance leads to an early crossover of left atrial and LV pressure, with premature MV closure (A-point) and interruption of the AC limb by the B-B' shoulder so that the AC interval is prolonged.10 Under those circumstances, a PR-AC difference of 0.06 sec or less has been shown10 to indicate an LVEDP of at least 20 mm Hg. One would also expect a reduced mean AC slope and of the anterior ventricular wall motion by Doppler's method. Dig 6th Int Conf Med Elect Biol Eng 35, 1965

In conclusion, peak final AMVL closing velocity provides a means for the noninvasive estimation of aortic ejection velocity and LV performance applicable to man. The BC slope of the AMVL echo which measures mean final closing velocity provides a simple means of estimating the peak AMVL closing velocity.

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Echocardiographic Features of Constrictive Pericarditis

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SUMMARY The most characteristic echocardiographic features of 12 patients with constrictive pericarditis were compared with the findings in 10 normal volunteers. Left ventricular posterior wall (LVPW) "flatness" was quantified by measuring the diastolic change in distance from the crystal artifact to the LVPW endocardium. In 11 of 12 patients the net diastolic LVPW endocardial movement was < 1 mm. In 10 normal volunteers LVPW endocardium moved posteriorly in diastole from 1.5 to 4 mm (mean 2.2 ± 0.8). Abnormal septal motion was present in five of 12 patients with constriction. Pericardial thickness measured using standard damping techniques for both constriction and normal populations did not distinguish the two groups. The "flattening" of the left ventricular endocardium as quantified above verifies earlier qualitative observations and was the most consistent finding in this series of patients with constrictive pericarditis.

THE RECOGNITION OF constrictive pericarditis is clinically important, but often difficult. Radiographic evidence may provide a clue, but is not diagnostic and may be absent. Noninvasive tests are of limited value in identifying the impaired ventricular filling which characterizes this disorder. While echocardiography is very useful in identifying pericardial fluid, specific diagnostic criteria for identifying constriction are not widely accepted.

Feigenbaum1 and others2,3 reported a "flat" motion of the posterior left ventricular wall during diastole corresponding to the abrupt transition of rapid ventricular filling to diastasis in patients with constrictive pericarditis.4,5 To date, this observation has not been quantified and applied to a series of patients with constrictive pericarditis. In this paper we report the echocardiographic features of 12 patients with pericardial constriction. We describe a method of quantifying the relative "flatness" of the posterior left ventricular endocardium and document the validity of this early observation.

Methods

The records of all patients from our institutions with a diagnosis of constrictive pericarditis who had an echocardiogram were reviewed. The diagnosis was established in 11 of the 12 patients by right heart catheterization. Surgical or autopsy findings were confirmatory in eight patients. Surgery was offered to two additional patients, but refused. In the remaining two patients, we felt that the clinical syndrome could be controlled with medical therapy.

Hemodynamic data used to establish the diagnosis accorded with the early observations by Sawyer et al.4 and the later criteria described by Shabetai.5 All patients manifested the early diastolic dip and late high plateau in the right ventricular pressure recording and equilibration of pulmonary arterial diastolic pressure, mean pulmonary arterial wedge pressure, mean right atrial pressure, right ventricular diastolic pressure, and, if available, the left ventricular diastolic pressure (table 1). Right-sided pressures were recorded on pullback and simultaneous right and left ventricular pressures were recorded in the seven patients who underwent left heart catheterization. The 12th patient, FM, refused catheterization. He had dyspnea on exertion and the associated signs of jugular venous distension, ascites, liver engorgement, dependent edema, and a pericardial knock on auscultation. His chest x-ray had an "eggshell" calcification pattern around the heart.

Echocardiographic strip chart recordings were made on either a Kent-Cambridge multichannel...

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Noninvasive assessment of left ventricular function from the mitral valve echogram. Relation of final anterior mitral leaflet closing velocity to peak dp/dt and aortic velocity.
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Circulation. 1978;58:861-871
doi: 10.1161/01.CIR.58.5.861

Circulation is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
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Print ISSN: 0009-7322. Online ISSN: 1524-4539

The online version of this article, along with updated information and services, is located on the World Wide Web at:
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