The Rate of Change of Left Ventricular Volume in Man

II. Diastolic Events in Health and Disease

By K. E. Hammermeister, M.D., and J. R. Warbasse, M.D.

SUMMARY

Because there is little quantitative information about the hemodynamics of left ventricular diastolic events in man, single-plane cineangiographic left ventricular volume curves were quantitatively analyzed at 16.6 msec intervals to evaluate the rate and amount of left ventricular filling during the early passive stage of filling and during the time of atrial contraction. The peak rate of passive diastolic filling of the left ventricle (D dV/dt), the peak rate of filling during atrial contraction (dV/dt c 'a'), and the increment in diastolic left ventricular volume due to atrial contraction (ΔV c 'a') were analyzed for a group of 110 adult patients with a variety of cardiac lesions. Normal D dV/dt, 503 ± 171 cc/sec, was significantly depressed in patients with mitral stenosis (393 ± 109 cc/sec) and coronary artery disease (394 ± 150 cc/sec). When corrected for end-diastolic volume (EDV), the resultant D dV/dt/EDV was significantly depressed in all disease states studied. D dV/dt corrected similarly for total left ventricular stroke volume (SV) was depressed from normal in patients with coronary disease, mitral stenosis, and valvular regurgitation. These data indicate that patients with coronary disease and chronic valvular disease have an abnormality in early diastolic filling of the left ventricle and that this may have a more profound effect on ventricular performance than changes in late diastolic compliance. D dV/dt has a high correlation with peak systolic ejection rate (r = 0.71), SV (r = 0.82), and EDV (r = 0.51).

The normal ΔV c 'a' represents 21% of the stroke volume (16% of the stroke volume if corrected for passive filling during this time period). Patients with coronary artery disease, valvular regurgitation, and aortic stenosis have an increased ΔV c'a'. Atrial contraction results in a second peak in mitral valve flow in late diastole (dV/dt c 'a'). In normal patients dV/dt c 'a' is 38% of D dV/dt, but in disease states this ratio is substantially increased. In the diseased heart atrial contraction makes a greater contribution to cardiac output than in the normal heart.

This study examines the rate of change of left ventricular volume (dV/dt) during diastole and the effect of atrial contraction on dV/dt and left ventricular volume. In the prior report, a technique was described and validation provided for the calculation of the instantaneous dV/dt in man from the single-plane cineangiogram, and the peak rate of change of left ventricular volume during systole (S dV/dt) was examined in detail and related to other hemodynamic parameters in a group of 113 patients including both normal subjects and those with various cardiac lesions. In the present study, these methods are applied to diastolic events. Left ventricular dV/dt during diastole is equal to mitral valve flow in the absence of aortic regurgitation or a shunt at the ventricular level. The peak rate of passive diastolic left ventricular filling (D dV/dt), the increment in diastolic left ventricular volume with atrial contraction (ΔV c 'a'), and peak dV/dt during atrial systole (dV/dt c 'a') were calculated and analyzed. Data will be presented to support the hypotheses that 1) there are abnormalities in left ventricular filling in patients with chronic heart disease in addition to the well-established abnormalities of left ventricular contraction, and 2) that in some types
of heart disease the relative contribution of atrial contraction is increased.

Methods

Left ventricular volume and dV/dt curves on 113 patients, calculated as previously described and used in the preceding report for the analysis of S dV/dt, were evaluated in regard to diastolic events. In three patients (one with valvular regurgitation and two with coronary artery disease), the diastolic portion of the left ventricular volume curve was not suitable for analysis, making a total of 110 patients in the present study. The indications for study, technique of catheterization, and characteristics of this patient population have been previously described.

The diastolic portions of the left ventricular volume and dV/dt curves are more complex than the systolic portions, as the effect of atrial contraction must also be taken into account (fig. 1). Certain reference points have been labelled for clarity. End-diastolic volume (EDV) and end-systolic volume (ESV) are self-explanatory. D dV/dt occurs in early diastole and exceeded dV/dt c 'a' in all except two patients, both of whom had coronary artery disease. Following the period of early rapid filling there is a break in the slope of the volume curve, which point is noted as DV1. This is followed by a plateau in the volume curve (diastasis), a period of slow or no ventricular filling corresponding to a dip or nadir in the dV/dt curve. With atrial contraction there is a rapid increment in left ventricular volume (ΔV c 'a') and a second peak in the dV/dt curve (dV/dt c 'a'). The diastolic left ventricular volume just preceding this rapid increment is labelled DV2.

In 62 of the 94 (66%) patients in sinus rhythm during angiography, the effect of atrial contraction on the smoothed volume curve was discernible. These 62 patients form the basis of the second part of the present report on the effect of atrial contraction on left ventricular volume and dV/dt. Left ventricular volume and dV/dt were measured at each of the points indicated in figure 1, as well as the time in seconds following the initial EDV at which the measurement was made. To evaluate the passive filling of the left ventricle during late diastole, similar measurements were made in five patients with atrial fibrillation at the same time intervals in relation to EDV as the patients in sinus rhythm.

Means, standard deviations, correlation coefficients, and the significance of difference between means of paired and unpaired variables were calculated using standard formulae with the aid of a computer (Olivetti Programa 101). The Student's t-test was used to test for statistical significance.

Results

D dV/dt

Table 1 gives the mean ± one standard deviation of D dV/dt and D dV/dt corrected for EDV (D dV/dt/EDV) and SV (D dV/dt/SV) for each of the diagnostic categories. D dV/dt was decreased and significantly different from normal in the groups with mitral stenosis and with coronary artery disease. D dV/dt/EDV was significantly depressed in all disease groups. D dV/dt/SV was significantly depressed from normal in the groups with coronary artery disease, mitral stenosis, and valvular regurgitation. Heart rate was not significantly different from normal in any of the disease subgroups except the patients with coronary artery disease, where heart rate increased slightly (table 1).

There was no significant correlation of D dV/dt with heart rate (r = 0.12) or left ventricular end-diastolic pressure (LVEDP) (r = 0.08). The correlation coefficient of this measure with ejection fraction was 0.23 (P < 0.05 where P is the probability of r = 0). A highly significant correlation with total stroke volume was found (r = 0.82, P < 0.001), with that portion of stroke...
Table 1

<table>
<thead>
<tr>
<th>Mean Values ± sd for D dV/dt, D dV/dt/EDV, and D dV/dt/SV</th>
</tr>
</thead>
<tbody>
<tr>
<td>Group</td>
</tr>
<tr>
<td>----------------</td>
</tr>
<tr>
<td>Normal</td>
</tr>
<tr>
<td>CAD</td>
</tr>
<tr>
<td>Aortic stenosis</td>
</tr>
<tr>
<td>Mitral stenosis</td>
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<tr>
<td>Regurgitant lesions</td>
</tr>
<tr>
<td>Cardiomyopathy</td>
</tr>
</tbody>
</table>

Abbreviations: sd = one standard deviation; D dV/dt = peak diastolic filling rate; D dV/dt/EDV = D dV/dt corrected for end diastolic volume (EDV); D dV/dt/SV = D dV/dt corrected for total left ventricular stroke volume (SV); HR = heart rate; n = number of patients; CAD = coronary artery disease.

*Statistically different from normal (P < 0.05).
†Statistically different from normal (P < 0.01).

As with S dV/dt and stroke volume, there is a very high correlation (r = 0.82) between D dV/dt and stroke volume as illustrated in figure 3. There is an almost straight line relationship between these two parameters for the normal group of 28 patients (r = 0.95), as shown by the regression line in figure 3.

Figure 2
Plot of S dV/dt against D dV/dt. r = simple linear correlation coefficient.

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Note that the majority of the patients with heart disease (61/81 or 75%) fall above the normal regression line, indicating that for their left ventricular stroke volume D dV/dt is depressed below normal.

There is a similar relationship between D dV/dt and EDV (fig. 4). The correlation for the whole group is somewhat less (r = 0.51), but still significant. The correlation between D dV/dt and EDV for the normal group is again very strong (r = 0.92). Again, almost all of the patients with cardiovascular disease (70/81 or 86%) lie above the regression line for the normal group, indicating that for their EDV, D dV/dt is depressed.

**ΔV c 'a'**

In the 23 of the 29 normal patients where ΔV c 'a' was apparent on inspection of the volume curve, ΔV c 'a' and the volumes at end-diastole, end-systole, DV1, and DV2 were averaged. The time interval after end-diastole of each volume was also averaged. These data are shown in figure 5a, illustrating that for this group of normal subjects the contribution of atrial contraction to left ventricular diastolic volume, ΔV c 'a', was 20 ± 9 cc or 21% of the stroke volume. During the period between DV1 and DV2, dV/dt falls to a low of 45 ± 90 cc/sec and increases with atrial contraction to 192 ± 84 cc/sec. For five patients in atrial fibrillation the increment in diastolic volume during the same time interval was only 5.2 ± 6.3 cc or 4.7% of stroke volume (fig. 5b).

In table 2, ΔV c 'a', the percent contribution of atrial contraction to stroke volume (%SV c 'a'), and dV/dt c 'a' are shown for each of the disease groups. The mean value of ΔV c 'a' is significantly increased over normal in the groups with regurgitant lesions, coronary artery disease, and aortic stenosis.

There is no significant correlation between ΔV c 'a' and LVEDP (r = 0.16) or ejection fraction (r = 0.03). A moderate correlation of r = 0.36 (P < 0.01) exists with D dV/dt and greater correlations with stroke volume (r = 0.67, P < 0.01), EDV (r = 0.62, P < 0.01), S dV/dt (r = 0.62, P < 0.01) and the dV/dt c 'a' (r = 0.91, P < 0.01).
Table 2
Contribution of Atrial Contraction in Normal and Various Disease Groups

<table>
<thead>
<tr>
<th>Group</th>
<th>n</th>
<th>ΔV/Δt c ’a’ (cc)</th>
<th>% SV c ’a’</th>
<th>dV/dt c ’a’ (cc/sec)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Normal</td>
<td>23</td>
<td>20 ± 9</td>
<td>21 ± 7</td>
<td>192 ± 84</td>
</tr>
<tr>
<td>Coronary artery disease</td>
<td>12</td>
<td>29 ± 12*</td>
<td>33 ± 12‡</td>
<td>268 ± 103</td>
</tr>
<tr>
<td>Aortic stenosis</td>
<td>6</td>
<td>29 ± 7†</td>
<td>24 ± 7</td>
<td>270 ± 77</td>
</tr>
<tr>
<td>Mitral stenosis</td>
<td>2</td>
<td>30 ±</td>
<td>40</td>
<td>232</td>
</tr>
<tr>
<td>Regurgitant lesions</td>
<td>10</td>
<td>44 ± 21†</td>
<td>30 ± 12*</td>
<td>331 ± 167*</td>
</tr>
<tr>
<td>Cardiomyopathy</td>
<td>3</td>
<td>16 ± 9</td>
<td>19 ± 9</td>
<td>203 ± 139</td>
</tr>
</tbody>
</table>

Abbreviations: n = number of patients in each group; %SV c ’a’ = percent of total left ventricular stroke volume contributed by atrial contraction; SV = total left ventricular stroke volume; dV/dt c ’a’ = peak filling rate during atrial contraction.
*Statistically different from normal (P < 0.05).
‡Statistically different from normal (P < 0.01).

An analysis of the time intervals between various portions of the volume curve for the normal group is shown in table 3. All the intervals except the total cycle length (EDV–EDV) and DV1–DV2 vary with, in very narrow limits (±0.03–0.04 sec standard deviation). The interval DV1–DV2 correlates very highly with the interval EDV–EDV (r = 0.87), indicating that when heart rate varies it does so primarily by varying the interval DV1–DV2. The duration of ejection (EDV–ESV), early rapid diastolic filling (ESV–DV1), and atrial contraction (DV2–EDV) are relatively constant and correlate poorly with total cycle length. Whether or not ΔV c ’a’ is apparent on inspection of the left ventricular volume curve is largely a function of heart rate. If the DV1–DV2 interval becomes very short due to rapid heart rate, a break in the slope of the diastolic volume curve in mid-diastole is no longer apparent. For 94 patients in sinus rhythm, ΔV c ’a’ was apparent in 84% (42/50) of those with heart rate ≤ 80, but only 45 (20/44) of those with heart rate greater than 80. The mean heart rate for the 62 patients in whom ΔV c ’a’ was apparent was 74 ± 12 beats/min, but 88 ± 13 beats/min in the 32 patients in sinus rhythm where ΔV c ’a’ could not be seen. The difference is highly significant (P < 0.001).

dV/dt c ’a’
As with ΔV c ’a’, dV/dt c ’a’ does not correlate with LVEDP (r = 0.18) or ejection fraction (r = 0.06), but correlates significantly with D dV/dt (r = 0.41, P < 0.01), stroke volume (r = 0.63, P < 0.01), EDV (r = 0.62, P < 0.01), and S dV/dt (r = 0.64; P < 0.01).

There is a high correlation (r = 0.91) between the peak rate of flow (dV/dt c ’a’) and the volume of flow (ΔV c ’a’) during atrial contraction. If D dV/dt is related to the volume of diastolic mitral valve blood flow before atrial contraction (stroke volume minus the ΔV c ’a’ in patients in sinus rhythm) again a high correlation (r = 0.83) exists. Thus, in all three parts of the cardiac cycle (systole, early passive diastolic filling, and late diastolic filling due to atrial contraction), a consistently high correlation exists between the volume of flow and the peak velocity of flow.

Discussion

D dV/dt
Diastolic filling in patients in sinus rhythm occurs in two phases: 1) an early passive filling of the ventricle and 2) a late increment in diastolic volume and dV/dt due to atrial contraction. When the heart rate is less than 80, these two phases are generally separated by a period of little or no change in diastolic volume and low mitral valve flow (fig. 1). Our value for D dV/dt in 29 normal
patients of 503 ± 171 cc/sec is more than double the passive left ventricular filling rate for eight control subjects reported by Stott et al. While these authors do not describe their technique of calculation in detail, it appears that their value of left ventricular filling rate may represent an average rate for the period of passive filling, whereas that in the present report is a peak filling rate. Mean diastolic filling rate for our normal group determined by dividing $SV - \Delta V/\alpha'$ by duration of passive filling (ESV−DV2) is 206 cc/sec, a figure comparable to that reported by Stott et al. of 216 cc/sec. Although there is not an independent means of validating instantaneous diastolic filling rates in man, we have validated S dV/dt by simultaneous comparison with aortic root blood flow measured with an electromagnetic velocity probe. The techniques of calculation of S dV/dt and D dV/dt are identical; therefore, we conclude that our value for D dV/dt is valid.

It was not unexpected that D dV/dt was significantly depressed in the group with mitral stenosis. It might also be expected that patients with constrictive pericarditis or pericardial tamponade would have a low D dV/dt. In the single patient with constrictive pericarditis, D dV/dt was depressed to 206 cc/sec. (There were no patients with pericardial tamponade.) Of considerable interest is the finding that patients with coronary disease also have significantly depressed D dV/dt (table 1), suggesting impairment to left ventricular filling due to a restrictive noncompliant left ventricle. When D dV/dt is corrected for EDV by dividing by EDV, the resultant D dV/dt/EDV is significantly depressed in all disease groups (table 1). This may be interpreted as evidence for an abnormality in ventricular filling in chronic heart disease, regardless of the nature of the lesions. Equally plausible is that filling is not necessarily abnormal, but it is the large EDV commonly seen in all types of chronic heart disease that accounts for this depression of dV/dt/EDV. However, when D dV/dt is normalized by division by total left ventricular stroke volume (the parameter with which D dV/dt correlates best), the resultant D dV/dt/SV is significantly depressed from normal in all disease groups except the two groups with cardiomyopathy and aortic stenosis (table 1). The group of patients with cardiomyopathy had normal early diastolic filling rates. However, these are all patients with nonobstructive cardiomyopathy who have dilated, relatively thin walled ventricles. Of the three patients with idiopathic hypertrophic subaortic stenosis D dV/dt fell within the normal range in all three, and D dV/dt/EDV was normal in two and elevated in one.

The evidence for impaired early filling of the ventricle in patients with mitral stenosis and coronary disease is strong since D dV/dt and D dV/dt corrected for either EDV or SV were all significantly depressed below normal. In the mitral stenosis group the mechanism is obvious: obstruction by the stenotic mitral valve. The mechanism for impaired early filling in the coronary disease patients is not entirely clear. The slightly more rapid heart rate in the coronary disease group should not be a factor as there was no relationship between heart rate and D dV/dt ($r = 0.12$). Although left ventricular wall thickness and mass were not measured in these patients, wall thickness was normal and mass was increased only in proportion to increase in end-diastolic volume in the 66 patients reported by Hamilton et al. Those patients expected to have markedly increased left ventricular mass and thick left ventricular wall (i.e., those with aortic stenosis and IHSS) did not have depressed D dV/dt or D dV/dt/SV. Thus, increased wall thickness or mass is not the explanation for depressed early filling in coronary disease. A more likely explanation is either the presence of fibrosis or relatively ischemic myocardium which has decreased compliance and/or slowed relaxation phase. Delayed relaxation has been demonstrated in the ischemic, isolated cat papillary muscle.

Strictly speaking, decreased compliance (i.e., decreased volume change for a given increment in left ventricular diastolic pressure) cannot be proven from the present data since left ventricular pressures were not, in general, measured during angiography in the present study. Bristow et al. concluded from volume and pressure analyses in 15 patients with coronary disease that the left ventricle in this disease had an abnormality of late diastolic compliance as well as abnormal contractile performance. Our studies expand this concept and suggest that other chronic heart diseases (valvular regurgitation) have abnormal diastolic filling, and that this abnormality exists in early diastole as well as late diastole. Since 80% of ventricular filling occurs in early diastole, it is apparent that an abnormality in early diastolic filling and/or compliance will have a more profound effect on over-all ventricular performance than the previously emphasized late diastolic compliance. An abnormality in early diastolic filling may be a factor in the blunted cardiac output response to exercise seen in

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many types of chronic heart disease. Measurement of early diastolic filling rates in man during exercise have not been reported. Measurement of cardiac dimensions by ultrasound during exercise may provide a future means of assessing early filling. 

Since \( S \frac{dV}{dt} \), \( D \frac{dV}{dt} \), and \( dV/dt \) all ‘a’ correlate well with stroke volume, is \( D \frac{dV}{dt} \) depressed because stroke volume is depressed? In no disease group was mean left ventricular stroke volume depressed below normal; in fact, in the group with regurgitant lesions, it was significantly increased over normal (143 cc vs 95 cc). Figure 3 demonstrates a nearly linear relationship between stroke volume and \( D \frac{dV}{dt} \) for the normal group (\( r = 0.95 \)), and that for most patients with heart disease \( D \frac{dV}{dt} \) was depressed in relation to stroke volume.

As shown in figure 2, there is a high correlation between \( S \frac{dV}{dt} \) and \( D \frac{dV}{dt} \). This correlation holds true for the various diagnostic categories except where there was obstruction to filling (mitral stenosis) or emptying (aortic stenosis) and the small group with cardiomyopathy. Note that the absolute values of \( S \frac{dV}{dt} \) and \( D \frac{dV}{dt} \) are very similar as well. It may seem surprising that systole and diastole (the former an active energy consuming process, the latter largely passive) should have similar values for peak rate of volume change. Note, however, that 1) the volume of passive filling is about 80% of the volume of ejection, 2) that the shape of the systolic \( dV/dt \) curve is similar to the shape of the passive portion of the diastolic curve, i.e., both “bell-shaped” (fig. 1), and 3) that the time in which events occur is similar—0.36 sec for ejection and 0.37 sec for passive diastolic filling in 23 normal patients (table 3). Thus, it follows that \( D \frac{dV}{dt} \) very closely approximates \( S \frac{dV}{dt} \) except where obstruction to filling or emptying exists.

\[ \Delta V \text{ ‘}a’ \]

The analysis of atrial function in man has previously been approached by several techniques. Fifty or more years ago Straub, and then Wiggers and Katz, published curves very similar to those in figure 1, showing a rapid increment in the external displacement of the canine ventricle in late diastole due to atrial contraction. Since the first patient studied by Meakins et al. in 1923, numerous authors have reported increases in cardiac output with conversion of atrial fibrillation to sinus rhythm, the average being 31% in the 119 patients summarized by Morris et al. Leimbach et al. reported a 24% increase in cardiac output when patients with heart block underwent sequential A-V pacing. Assuming a normal ejection fraction, one can calculate from the atrial volume data of Bruns that the change in atrial volume during atrial contraction is approximately equal to 17% of the stroke volume.

Utilizing carefully drawn ventricular volume curves with sufficient points per unit time (our experience indicates 12/sec generally inadequate, 30/sec often adequate, 60/sec preferable), one can detect the effect of atrial contraction on diastolic ventricular volume in most patients with sinus rhythm with a rate less than 80 (fig. 1). A careful examination of the cineangiogram will usually show a rapid increment in volume early during atrial contraction, the so-called “atrial kick.” This increment in diastolic volume in our studies accounts for 21% of the stroke volume in the normal group (fig. 5a). In a group of five patients in atrial fibrillation at comparable heart rates, the amount of passive filling during a comparable time period in late diastole amounts to only 5% of the stroke volume (fig. 5b). Thus, the contribution to ventricular filling by atrial contraction is approximately 16% of the stroke volume or cardiac output, a figure comparable but slightly lower than those cited above using other techniques. Bristow et al., using a similar technique, arrived at an identical figure for four normal patients.

The present report indicates that for most patients with chronic heart disease, regardless of lesion, the contribution of atrial contraction to cardiac output or left ventricular stroke volume is increased (table 2). Although there was considerable overlap between groups, the mean value of \( \Delta V \text{ ‘}a’ \) was significantly increased over normal in the groups with coronary disease, aortic stenosis, and valvular regurgitant lesions. Stott et al., using a similar technique, also reported an increased value of \( \Delta V \text{ ‘}a’ \) in aortic stenosis. Bristow et al. reported an increase in \( \Delta V \text{ ‘}a’ \) in patients with coronary disease, comparable to our finding.

The correlation of \( \Delta V \text{ ‘}a’ \) with \( EDV \) again supports the hypothesis that atrial function is of greater importance in chronic heart disease, since as the heart dilates the atrial contribution to stroke volume increases. This is of particular importance in patients with valvular regurgitation, where in eight of ten patients \( \Delta V \text{ ‘}a’ \) was greater than one standard deviation above the normal mean and the average for the group was over twice the normal mean. This marked increase in \( \Delta V \text{ ‘}a’ \) in patients
with regurgitant lesions explains in part the correlation of $\Delta V \, \dot{c} \, 'a'$ with stroke volume.

Analysis of the time intervals of various portions of the volume curve indicates that variation in heart rate results largely from variations in the interval from $DV_1$ to $DV_2$ (fig. 1), with the other intervals remaining relatively constant (table 5). In the 23 normal patients in whom $\Delta V \, \dot{c} \, 'a'$ was evident and heart rate varied from 50-88 beats per minute, the period of ejection (EDV-ESV), the period of early rapid filling (ESV-DV), and the period of atrial contraction (DVx-EDV) all varied within a very narrow range and correlated poorly with the EDV-EDV (R-R) interval. In contrast, the period $DV_1$-$DV_2$ varied widely and correlated closely with the EDV-EDV interval. This accounts for the fact that $\Delta V \, \dot{c} \, 'a'$ is more often evident at slower heart rates. At faster heart rates the interval $DV_1$-$DV_2$ becomes very short or nonexistent, and atrial contraction begins at or before point $DV_1$ (fig. 1) with no break in the slope of the diastolic volume curve. This is compatible with previous animal studies. Such curves also suggest that at rapid heart rates atrial contraction may assume relatively less importance, in contrast to the conclusion of Benchimol.13 Note that D $\frac{dV}{dt}$ is substantially greater than $\frac{dV}{dt} \, \dot{c} \, 'a'$ in all subgroups (tables 1, 2). Thus atrial contraction occurring at the time of D $\frac{dV}{dt}$ (i.e., heart rates > 120/min) may not augment mitral flow and ventricular filling. Studies with atrial pacing may be compatible with this concept, since stroke volume falls as the paced rate is increased,14 although venous filling is also a factor. The maintenance of stroke volume during the tachycardia of exercise may involve other mechanisms such as increased venous return and increased sympathetic stimulation.

$\frac{dV}{dt} \, \dot{c} \, 'a'$

In about two-thirds of the patients in sinus rhythm the curve of left ventricular filling rate has two peaks, the early peak due to passive filling and the late peak due to active atrial contraction (bottom, fig. 1). In the absence of aortic regurgitation and intracardiac shunts, this curve represents mitral valve flow and is essentially identical in contour to the curve for mitral valve flow measured directly with an intracardiac electromagnetic flow meter in the calf by Nolan et al.15 In all cases except two patients with coronary disease, D $\frac{dV}{dt}$ exceeded $\frac{dV}{dt} \, \dot{c} \, 'a'$. As in the report of Stott et al.,2 the present study demonstrates an increased value for $\frac{dV}{dt} \, \dot{c} \, 'a'$ (although not statistically significant) in patients with aortic stenosis. In fact, all disease groups, except the small group with cardiomyopathy, had mean values of $\frac{dV}{dt} \, \dot{c} \, 'a'$ (table 2) increased from 24-72% over normal. (However, these were not statistically significant differences except for the valvular regurgitation group.) These data and the $\Delta V \, \dot{c} \, 'a'$ measurements indicate that atrial contraction contributes more to cardiac output in patients with chronic heart disease of several types than in normal human subjects.

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