The A Wave of the Apexcardiogram and Left Ventricular Diastolic Stiffness


SUMMARY

This study was made to determine whether the A wave of the apexcardiogram (ACG), a reflection of the late diastolic response of the left ventricle to atrial systole, corresponded in a quantifiable way to left ventricular late diastolic stiffness (LVDS). Using a combined ultrasonic and hemodynamic technique, the slope of the late diastolic left ventricular pressure/diameter relationship (ΔP/ΔD) was calculated in 25 patients and used as a measure of effective LVDS. Most patients had valvular heart disease, all were in sinus rhythm and none had regional abnormalities of contraction. An ACG was recorded in all and the ratio of the size of the A wave to the total amplitude of the ACG wave (A/H) was calculated. When A/H was more than 11%, left ventricular hypertrophy (LVH) and the presence of a fourth heart sound were the rule in the group of patients studied.

Using A/H as an independent variable, correlation coefficients were obtained for ΔP, ΔD, ΔP/ΔD, left ventricular end diastolic pressure (LVEDP), and left ventricular end diastolic volume (LVEDV). Correlation coefficients (r) were: ΔP = 0.68; ΔD = -0.05; ΔP/ΔD = 0.87; LVEDP = 0.73; LVEDV = 0.21. It is concluded that A/H corresponds best to LVDS and is a useful noninvasive measurement of this property of the left ventricle.

THE EVALUATION OF LEFT VENTRICULAR FUNCTION in man has mainly depended on data obtained by invasive methods which are often time consuming, may carry some risk to the patient, and therefore cannot be repeated frequently. Accordingly, many investigators have sought to correlate information derived from noninvasive procedures with such hemodynamic data. This approach has merit and the apexcardiogram (ACG) has been studied extensively in this context. It has been demonstrated that the ACG reflects left ventricular events accurately in a qualitative, and to a lesser extent, quantitative fashion. The recent development in this laboratory of an index of left ventricular late diastolic stiffness has led us to investigate the correlation of such an index with the A wave of the ACG, since both measurements may be regarded as reflecting the late diastolic response of the left ventricle to atrial systole. It might be expected that the magnitude of the A wave would depend on the degree of left ventricular stiffness. If this were so then the A wave of the ACG would be another useful measurement for the noninvasive evaluation of this particular facet of left ventricular function in the individual patient.

Materials and Methods

Twenty-five patients aged 17 to 62 were studied. They were selected because they were to undergo cardiac catheterization for evaluation of various cardiovascular diseases. Criteria for inclusion in this group were that 1) the patients were in sinus rhythm; 2) they were in a comparable basal state for all studies performed since no increase in blood pressure, chest
pain, or tachycardia beyond the usual resting rate occurred. This was important since the ACG was a separate procedure, usually within 24 hours of the cardiac catheterization; 3) technically adequate ACG, left ventricular cavity echocardiograms and left ventricular pressure curves were available.

ACG tracings were made with the patient in the left lateral decubitus position, usually at an angle of 20–30°. A Hellige Pulse Microphone transducer was used, held by hand at the point of maximal impulse of the apex.* Quiet respiration was usual but occasionally the optimal tracing was obtained on expiration. The ACGs were recorded with a reference phonocardiogram and ECG on a Cambridge MC IV Multichannel Data System Recorder. The oscilloscope of this recorder was used to verify the proper positioning of the transducer and the paper speed of the recording was 100 mm per second. The method of expressing the ACG data was by the ratio of the size of the A wave to the total amplitude of the ACG (A/H), and this is illustrated in figure 1. This method was originated by Benchmol and Dimond and has been used by our laboratory and others. The height of the A wave was calculated as a fraction of the total outward movement in systole (H) and expressed as a percentage. For all determinations the average of at least five complexes was used.

Cardiac catheterization was carried out in the fasting state, following diazepam (5-10 mgm i.m.) premedication. Brachial arteriotomy and retrograde left heart catheterization were performed with standard #8 French catheters in 15 patients, and with high fidelity micromanometer tipped catheters in ten patients. Micromanometer pressures were calibrated with a mercury reference and checked against the simultaneous lumen pressure of the catheter for alignment of end-diastolic and peak-systolic values. No significant baseline drift was observed in this system. All pressures were recorded on an Electronics for Medicine DR-12 recorder. Left ventricular cineangiography was performed in each study, and patients with regional disorders of contraction were excluded.

Immediately following cardiac output determination, but prior to angiography, a simultaneous photographic strip chart recording of the electrocardiogram, left ventricular pressure and ultrasonically determined left ventricular septal and posterior wall motion was made. The ultrasonoscope used was a Smith-Kline Instruments Company Ekoline 20A coupled to an Electronics for Medicine recorder via an Electronics for Medicine UDA interface channel. Paper speed was 100 mm/sec with 0.02 second timelines. The left ventricular cavity recording was obtained in a standard manner by the utilization of strip chart scanning as described by Feigenbaum. The minor axis used was that in a plane immediately below the mitral valve leaflets. Only recordings where it was possible to identify clearly the posterior wall and septal endocardial surfaces were used. Appropriate damping maneuvers were done in order to be more certain that true endocardium was being recorded. Indocyanine green or saline was injected into the left ventricular cavity if any doubt existed.

The slope of the late diastolic pressure-diameter relation at the time of left atrial systole (ΔP/ΔD) was measured in each patient, as illustrated in figure 2. The onset of left atrial systole was defined with respect to

*Fritz Hellige & Co., Freiburg, West Germany. This apparatus has a flat response at frequencies from 50Hz down to 0.3 Hz, and a time constant in excess of 3 sec.

![Figure 1](http://circ.ahajournals.org/)

**Figure 1**

The method of measurement for A/H. A = height of A wave, H = total height of the apexcardiogram; OM = outward movement; O = O point; RFW = rapid filling wave; SFW = slow filling wave. A simultaneous ECG and phonocardiogram (PCG) were taken. A/H in this example is 5%.

*Mikrotip, Millar Instruments, Houston, Texas.*

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

**Figure 2**

Photographic stripchart recording of simultaneous echocardiogram, left ventricular pressure (LVP), and ECG from patient 8. A traced form of the actual record is shown on the right for explanatory purposes. P1 and P2 represent respectively the left ventricular pressures at onset and peak of left atrial systole. D1 and D2 are shown by the lines AB and CD representing left ventricular diameters at onset (D1) and peak (D2) of left atrial systole. PW = posterior wall; LVP = left ventricular pressure.
the P wave of the ECG as a reference. The interval from onset of the P wave to onset of left atrial systole in man has been shown to be about 80 msec by Braunwald et al.\textsuperscript{12} whose method was corrected for the 5 msec transmission delay of their pressure measurement system. In this study, no correction was made for transmission delay through the standard catheters, which averages 10-14 msec in our laboratory,\textsuperscript{4} since this was felt to be less than the error in defining the onset of the electrocardiographic P wave.

Having defined P1 and D1 as the left ventricular diastolic pressures and echocardiographic diameter at the approximate onset of left atrial systole, we measured P2 and D2 at the peak of left atrial systole, taken as the peak of the atrial (a) wave in the left ventricular pressure tracing (fig. 2). When there was no clearly defined “a” wave, P2 and D2 were obtained at end diastole, defined as occurring 40 msec after the onset of the QRS in the ECG.\textsuperscript{6} Change in pressure and change in diameter (ΔP and ΔD), defined as P2-P1 and D2-D1, thus represent diastolic pressure and diameter increments occurring in association with left atrial systole. Their ratio, ΔP/ΔD, has been previously reported by us as a measure of effective left ventricular diastolic stiffness (LVDS).\textsuperscript{6, 13}

Left ventricular end diastolic pressure (LVEDP) was measured in each patient, and left ventricular end diastolic volume (LVEDV) was calculated from the echocardiographic left ventricular end diastolic diameter according to a formula proposed by Teichholz\textsuperscript{14} (personal communication): \( V = (7.0/2.4 + D) \times D^3 \), where \( V \) = volume in cc and \( D \) = left ventricular cavity diameter in cm. The presence or absence of left ventricular hypertrophy (LVH) was derived from the quantified Romhilt and Estes method using a standard 12 lead ECG.\textsuperscript{15} A score of five or more was considered to indicate left ventricular hypertrophy. The presence or absence of gallop sounds was established by means of the phonocardiogram using Leatham microphones. The statistical analyses were performed by standard techniques\textsuperscript{18} using a Wang 600 electronic calculator.

**Results**

Table 1 summarizes the over-all data obtained for the invasive and noninvasive measurements in our patients. They have been divided into two groups for the purpose of analysis. Group A consists of those patients with A/H of 11% or less, a figure we consider to be within the limits of normal, and Group B those with higher A/H. This separation is based on the level of the mean A/H obtained in our laboratory for normal controls.\textsuperscript{8}

**A/H and Left Ventricular Late Diastolic Pressure and Volume**

Correlation coefficients were calculated for the total cohort using A/H as the independent variable and ΔP, ΔD, ΔP/ΔD, LVEDP, LVEDV as the dependent variables. It can be seen from table 1 that ΔP/ΔD, LVEDP, and ΔP correlate best. The relationship between A/H and ΔP/ΔD is shown in figure 3 (\( r = 0.868 \)). A lesser correlation (\( r = 0.730 \)) was also found between A/H and LVEDP (fig. 4). The correlation between A/H and ΔP was \( r = 0.684 \) but ΔD and LVEDV did not correlate well.

**A/H and Left Ventricular Hypertrophy**

A close relationship of the degree of LVDS to the presence or absence of LVH exists,\textsuperscript{6} and the level

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of A/H should also reflect this. When Groups A and B were compared this was the case, since three of 13 patients in Group A had LVH whereas 11 of 12 of Group B had LVH. This difference was highly significant (P = 0.0003). The mean value of A/H for patients with LVH was 14.5% as compared with 7.1% for those without LVH (fig. 5).

A/H and the Fourth Sound

A fourth sound was recorded in only one patient in Group A (1/13), whereas it was the rule in Group B (11/12). The difference is again highly significant as reflected by P < 0.00003. The mean value of A/H for patients with a fourth sound was 16.6% as compared to 6.3% for those without LVH (fig. 5). In patient 17 it was difficult to identify a sound due to an Austin-Flint murmur which obscured the site where the sound might be recorded on the phonocardiogram.

Discussion

The A wave of the ACG has been used over the past decade as one indicator of left ventricular function. It has been shown to mirror diastolic events directly recorded from the left ventricle, reflecting the impact of the late atrial contribution on the filling or already filled left ventricular cavity. It is reasonable to assume that in normal man the distensibility of the left ventricle could accommodate the changes in pressure and volume brought about by this "atrial kick" phenomenon, and that the consequent precordial A wave would be of little magnitude. Similarly it might be expected that a diseased and poorly functioning left ventricle would

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Table 1

Summary of Patient Data

<table>
<thead>
<tr>
<th>Pt.</th>
<th>Age/sex</th>
<th>Diagnosis</th>
<th>A/H (%)</th>
<th>ΔP/mm</th>
<th>ΔP/Hg</th>
<th>ΔP/ΔD/mm Hg</th>
<th>LVEDP/mm Hg</th>
<th>LVEDV/cc</th>
<th>LVH</th>
<th>S1</th>
<th>S4</th>
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<tr>
<td>1.</td>
<td>MM 36 F</td>
<td>MS, AR</td>
<td>2</td>
<td>1.9</td>
<td>2.7</td>
<td>1.4</td>
<td>6</td>
<td>172</td>
<td>0</td>
<td>0</td>
<td></td>
</tr>
<tr>
<td>2.</td>
<td>GP 29 F</td>
<td>MS</td>
<td>3</td>
<td>2.0</td>
<td>3.9</td>
<td>2.0</td>
<td>9</td>
<td>148</td>
<td>0</td>
<td>0</td>
<td></td>
</tr>
<tr>
<td>3.</td>
<td>BF 40 M</td>
<td>MS</td>
<td>4</td>
<td>1.1</td>
<td>2.9</td>
<td>2.7</td>
<td>8</td>
<td>78</td>
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<tr>
<td>4.</td>
<td>RJ 50 F</td>
<td>MS</td>
<td>5</td>
<td>4.8</td>
<td>2.2</td>
<td>0.5</td>
<td>5</td>
<td>119</td>
<td>0</td>
<td>0</td>
<td></td>
</tr>
<tr>
<td>5.</td>
<td>LB 51 F</td>
<td>AR, AS, MS</td>
<td>5</td>
<td>1.5</td>
<td>5.2</td>
<td>3.4</td>
<td>13</td>
<td>179</td>
<td>LVH</td>
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<td>MR</td>
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<td>2.7</td>
<td>6.3</td>
<td>2.3</td>
<td>6</td>
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<td>0</td>
<td>0</td>
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<tr>
<td>7.</td>
<td>PM 30 F</td>
<td>VSD</td>
<td>5</td>
<td>2.3</td>
<td>4.5</td>
<td>1.9</td>
<td>12</td>
<td>148</td>
<td>0</td>
<td>0</td>
<td></td>
</tr>
<tr>
<td>8.</td>
<td>SH 25 M</td>
<td>MR</td>
<td>5</td>
<td>1.5</td>
<td>7.2</td>
<td>4.8</td>
<td>26</td>
<td>207</td>
<td>LVH</td>
<td>0</td>
<td></td>
</tr>
<tr>
<td>9.</td>
<td>EQ 44 F</td>
<td>AR, MR</td>
<td>6</td>
<td>3.4</td>
<td>11.4</td>
<td>3.9</td>
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<td>210</td>
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<tr>
<td>10.</td>
<td>VW 48 M</td>
<td>AR, MS</td>
<td>8</td>
<td>7.1</td>
<td>18.1</td>
<td>2.6</td>
<td>25</td>
<td>172</td>
<td>0</td>
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<tr>
<td>11.</td>
<td>WH 62 M</td>
<td>AR</td>
<td>10</td>
<td>1.5</td>
<td>9.0</td>
<td>5.9</td>
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<td>239</td>
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<tr>
<td>12.</td>
<td>RC 53 F</td>
<td>MR, MS</td>
<td>11</td>
<td>1.7</td>
<td>5.2</td>
<td>3.2</td>
<td>11</td>
<td>119</td>
<td>0</td>
<td>0</td>
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</tr>
<tr>
<td>13.</td>
<td>WC 40 F</td>
<td>Cardiomyopathy</td>
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<td>1.9</td>
<td>8.8</td>
<td>4.9</td>
<td>20</td>
<td>155</td>
<td>0</td>
<td>4</td>
<td></td>
</tr>
</tbody>
</table>

For Groups A and B

Correlations for both groups r = -0.051

Abbreviations: MS = mitral stenosis; MR = mitral regurgitation; AS = aortic stenosis; AR = aortic regurgitation; VSD = ventricular septal defect; CAD = coronary artery disease; LVEDP and LVEDV = left ventricular end diastolic pressure and volume; LVH = left ventricular hypertrophy; S1, S4 = third and fourth heart sounds. A/H = ratio of height of the A wave to total amplitude of apexcardiogram; ΔP, ΔD = change in left ventricular late diastolic pressure and diameter.
respond poorly to the added burden of atrial systole in that impaired distensibility leads to disproportionate intraventricular pressure-volume changes, reflected by an increase in the precordial A wave, often to the extent that it may become palpable. This has been shown in disease states where the "reserve of distensibility" of the left ventricle is impaired, usually in the presence of LVH. In aortic stenosis, aortic regurgitation, idiopathic hypertrophic subaortic stenosis, cardiomyopathy, and hypertension an abnormal increase in the height of the A wave has been described,9, 10, 20 usually associated with severe disease. In such studies some patients did not have large A waves in spite of severe disease and Tavel has shown that in patients with severe aortic stenosis the absence of a large A wave may occur.21 In severe aortic regurgitation a small or absent A wave may be due to premature closure of the mitral valve leaflets with elevation of left ventricular diastolic pressures above left atrial pressures during most of diastole.22

The use of stress with changes in preload and afterload may alter what was apparently a normal A wave in a patient at rest. Ginn et al. have shown that subjects with established coronary artery disease responded to exercise by a near doubling of the height of the A wave as measured by A/H.19 This is of interest in view of recent studies from our laboratory which have shown impairment of left ventricular relaxation and consequent increased diastolic stiffness during atrial pacing in patients with coronary artery disease.23 In normal subjects, and those thought to have cardiac ischemia but with negative coronary cineangiograms, the elevation of A/H was trivial. Ginn et al. also demonstrated that an elevated A/H during an episode of ischemia was appreciably reduced by nitroglycerin. Similar data have been obtained by Benchimol and Dimond.7

The precise mechanism whereby A wave elevation occurs is not known. Some have considered that it is an indicator, not necessarily quantitative, of changes in the level of LVEDP, and have attempted to resolve this by comparing LVEDP with A/H.24, 25 Voigt, using 58 observations in 18 patients, most of whom had coronary artery disease, was able to obtain some correlation (r = 0.57) although this was not striking. He observed that the height of the precordial A wave depended more on the height of the A wave seen in the left ventricular tracing, and stated that it therefore depended on the degree of "atrial kick." Willems, using calibrated precordial tracings in dogs, also showed a relatively weak correlation (r = 0.48) between the absolute level of the precordial A wave and LVEDP.26 Our own data show a more significant relation of A/H to LVEDP (r = 0.73), although five of the 13 patients with A/H within the limits of normal had elevated LVEDP. In any discussion of this area, LVEDV must also be considered since it is the pressure-volume relationship that determines compliance. Our data indicated that pressure change was significant whereas end diastolic volume and late diameter changes were insignificant correlates of A/H. The index of stiffness ΔP/ΔP proved to have even better correlation with A/H than ΔP.

A most important common denominator in elevations of A/H and ΔP/ΔD was LVH by ECG criteria, and the only individual with abnormal A/H but without LVH was patient 21 who had restrictive cardiomyopathy due to cardiac amyloidosis and a typical low voltage ECG. It has also been shown that there is a close association between a large A wave and the presence of a fourth heart sound; this has been related to elevated LVEDP.21 In the present study the presence of a fourth sound, together with elevation of A/H and ECG evidence of LVH, is very well correlated with an abnormal index of late diastolic stiffness of the left ventricle. This is in keeping with the data obtained by Shah26 who found that LVH was the major concomitant of a fourth sound in patients with cardiomyopathy. Both Shah26 and Craige27 have postulated that a process giving rise to LVH would result in reduced compliance and an elevated

*Figure 5*

Comparison of ratio of amplitude of A wave to total amplitude of ACG (A/H) % in patients with and without left ventricular hypertrophy (LVH) and with and without a fourth heart sound (4). The bars represent mean values and the horizontal brackets their standard errors.
resistance to filling. The atrial boost would be augmented and atrial gallop would ensue.

The relationship between an index of left ventricular stiffness and A/H makes the latter a useful noninvasive index of the ability of the left ventricle to accommodate the results of atrial systole. This relationship is to be expected since the measurements taken are of identical events. The application of A/H to disease states already exists and it has been shown to be a marker of the capability of the ventricle to respond to stress. It should be an excellent tool for the serial follow-up of patients with generalized left ventricular compromise.

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