The Use of Apexcardiography in the Assessment of Left Ventricular Diastolic Pressure

By Gustav C. Voigt, M.D., and Gottlieb C. Friesinger, M.D.

SUMMARY

A total of 58 observations of simultaneous left ventricular pressure and apexcardiograms (ACG) was made on 18 patients. An a wave percentage amplitude (aWPA) of greater than 15% of the total deflection of the ACG indicated an increase in left ventricular end-diastolic pressure (LVEDP). In 12 observations on six patients, an aWPA of less than 15% was associated with a high LVEDP. Patients with high LVEDP and aWPA of less than 15% had a high early left ventricular diastolic pressure with further rise in pressure prior to atrial contraction. These patients had small LV a waves ("atrial kick"). The aWPA of the ACG correlated better with the magnitude of the LV a wave than the absolute level of LVEDP in all patients. Correlation was good between changes in aWPA and changes in LVEDP in individual patients; but the ACG as an indirect means of evaluating left ventricular function is limited by the fact that elevations in LVEDP can exist in the presence of a normal aWPA. The ACG is a complex tracing reflecting not only intracardiac pressures, but changes in left ventricular volume, compliance, position, and perhaps left atrial function as well.

Additional Indexing Words:
Myocardial function Angina pectoris Coronary artery disease

APEXCARDIOGRAPHY is an indirect means of evaluating left ventricular function by recording the low frequency displacements of the chest wall over the apex beat of the heart. Tracings so obtained can be used to time certain cardiac events such as atrial systole, the onset of ventricular contraction, and mitral valve opening.\(^1\)\(^-\)\(^2\) It has been suggested that the relative magnitude of the a wave of the apexcardiogram (ACG), which represents the effect of atrial contraction on ventricular filling, reflects the left ventricular end-diastolic pressure (LVEDP) and in this way provides semi-quantitative information about left ventricular function.\(^3\)\(^-\)\(^9\) Despite these suggestions the correlations between simultaneous measurements of intracavitary cardiac pressures and ACGs have been examined in only a few studies. A method of evaluating left ventricular function at frequent intervals without repeated cardiac catheterization is needed. Since few studies have correlated simultaneous LV pressure and ACG, a study was designed to examine the relationships between the a wave of the ACG and the left ventricular pressures in man.

Methods

Fifty-eight observations in 18 patients undergoing cardiac catheterization in the Wellcome Research Laboratory of the Johns Hopkins University School of Medicine formed the basis of this report. These patients were being evaluated for coronary artery disease (CAD) or primary myocardial disease (PMD). Two patients had aortic stenosis coexisting with CAD and one
### Table 1

**Summary of Observations on 18 Patients**

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Abbreviations: aWPA = a wave percentage amplitude of the apexcardiogram; LVEDP = left ventricular end-diastolic pressure; LV a = left ventricular a wave or "atrial kick"; LVd = left ventricular early diastolic pressure; LVs = left ventricular systolic pressure; — = not recorded; PMD = primary myocardial disease; CAD = coronary artery disease; AS = aortic stenosis; IHSS = idiopathic hypertrophic subaortic stenosis.

patient had idiopathic hypertrophic subaortic stenosis (IHSS). Three patients had normal selective coronary arteriograms and normal hemodynamics at cardiac catheterization (table 1).

All patients were in sinus rhythm. Simultaneous left ventricular pressures, ECGs, and ACGs were recorded for each patient and repeated whenever changes in LVEDP were anticipated. Changes in LVEDP were produced by the spontaneous occurrence of angina pectoris, the administration of nitroglycerin, or the injection of contrast material for diagnostic coronary arteriography or for left ventricular angiography, or all three. In patients with primary myocardial disease, the infusion of angiotensin for evaluation of left ventricular function provided a means of obtaining simultaneous ACGs and left ventricular pressures at increments of LVEDP.13

The technics of cardiac catheterization and selective coronary arteriography have been described elsewhere. The ACGs were recorded by using a pulse-wave linear microphone (Sanborn no. 374) attached to a conical cup 2 cm in diameter with a 6-inch plastic tubing with an inside diameter of 2 mm. The linear microphone was connected to an AC amplifier of a DR-8 Electronics for Medicine oscillographic recorder. The band-pass filters were set at 0.1 Hz, lower limit, and 20 Hz, upper limit. The acoustic and electronic characteristics of this system have been described previously.16, 17

The pulse-wave linear microphone (Sanborn no. 374) was modified to provide a time constant of greater than 1.0 sec with an instantaneous and sustained mercury manometer signal of 50 mm Hg.18

The ACG was obtained by placing the cone of

the sound microphone over the apex beat of the heart. The instrument was held in place by hand, in all instances by the same person. The recordings were made with the patient in a modified left lateral decubitus position unless satisfactory tracings could be obtained in the supine position. The ACGs were usually recorded at the end of a normal expiration, although technically satisfactory tracings were occasionally obtained during quiet breathing. Simultaneous left ventricular pressures, ECG, and ACG were recorded at a paper speed of 75 mm/sec with 0.04-sec time lines. A preliminary study showed that the recording of pressure lagged behind the recording of the ACG by 0.005 sec. This difference is negligible and is ignored.

The temporal relationships of components of the ACG to intracardiac events has been detailed elsewhere and confirmed in this laboratory. The ACG to intracardiac events has been detailed elsewhere and confirmed in this laboratory. Figure 1 is a schematic representation of an apexcardiogram. For each observation of simultaneous ACG and LV pressure the following measurements were made:

1. The relative magnitude of the a wave of the ACG to the total deflection of the ACG, or the a-wave percentage amplitude (aWPA), was determined. The vertical magnitude of the a wave in millimeters was measured from the intersection of the slow filling wave (SFW) and the initial upstroke of the a wave to the peak of the a wave. This value was expressed as a percentage of the total vertical deflection of the ACG (a-O-E = aWPA; see fig. 1).

2. The a-wave amplitude was determined as a function of the total diastolic deflection of the ACG (a/D).

3. The total diastolic deflection was deter-
A simultaneous apexcardiogram (ACG) and EKG. The O point represents mitral valve opening and is followed by waves representing rapid ventricular filling (RFW) and slow ventricular filling (SFW). The a wave is produced by the effect of atrial contraction on left ventricular filling ("atrial kick"). The a wave is followed by a sharp upstroke representing the period of isometric contraction, terminating at the E point, which corresponds to aortic valve opening and the beginning of ventricular ejection.

The following measurements were made: (1) the a-wave percentage amplitude (aWPA), determined by measuring the height of the a wave in millimeters from its intersection with the SFW to its peak, and expressing this value as a percentage of the total deflection of the ACG (a/O-E = aWPA); (2) the a-wave amplitude as a function of the total diastolic deflection (a/D), and (3) the total diastolic deflection as a function of the total vertical deflection (D/O-E).

A Comparison between Left Ventricular End Diastolic Pressure and "a" wave Percentage Amplitude of the Apex Cardiogram

Comparison of the aWPA of the ACG with the LVEDP. An aWPA of greater than 15% is always associated with an LVEDP over 12 mm Hg. In many observations, however, the aWPA is less than 15% and the LVEDP is greater than 12 mm Hg (open circles).

Results

A total of 58 observations was made. Changes in LVEDP of 5 mm Hg or more were observed in 11 patients (table 1). The relationship between the aWPA of the ACG and the LVEDP for each observation is seen in figure 2. An aWPA of greater than 15% was associated consistently with an LVEDP of greater than 12 mm Hg. However, the aWPA was less than 15% and the LVEDP was greater than 12 mm Hg in many instances and as high as 30 mm Hg in some. The correlation coefficient of 0.57 indicates only fair predictability of the LVEDP from the aWPA; this is primarily due to the fact that a high LVEDP was often associated with an aWPA of less than 15%.
Simultaneous left ventricular pressures and ACGs in two patients and plots of the aWPA and LVEDP in these patients.

Patient 1, who had primary myocardial disease, was studied during an infusion of angiotensin for the evaluation of left ventricular function. The resting LVEDP was normal. As the LVEDP was increased to 28 mm Hg, the aWPA of the ACG rose from 12% to 27%, and the LV a wave from 6 to 17 mm Hg. When the LVEDP rose further to 30 mm Hg, the aWPA decreased to 17%, the amplitude of the LV a wave fell from 17 to 12 mm Hg, and the early left ventricular diastolic pressure rose from 11 to 17 mm Hg (see text).

Patient 3, with CAD and aortic stenosis, had an LVEDP of 20 mm Hg and an aWPA of 25% shortly after catheterization of the left ventricle. Before any other interventions, the LVEDP fell to 14 mm Hg and the aWPA to 16%. After a left ventricular angiogram, the LVEDP rose to 27 mm Hg and the aWPA to 29%. At high LVEDP the LV a wave is large, up to 18 mm Hg, and the early LV pressure is low, 10 mm Hg or less.
Simultaneous LV pressures and ACGs in two patients with CAD in whom the aWPA did not significantly exceed 15% in spite of LVEDPs over 20 mm Hg.

Patient 15 had an LVEDP of 26 mm Hg with an LV a wave of 11 mm Hg during a spontaneous attack of angina pectoris. Note that the early LV pressure was 14 mm Hg and the aWPA only 16%. Following sublingual nitroglycerin the LVEDP fell to 8 mm Hg with a small LV a wave and an aWPA of 5%.

Patient 16 had an LVEDP of 24 mm Hg with an aWPA of 16% during a spontaneous episode of angina. The LVEDP fell to 9 mm Hg and the aWPA to 5% after nitroglycerin. After coronary arteriography the LVEDP rose again to 20 mm Hg and the aWPA to 13%. When the LVEDP was elevated the early LV diastolic pressure was increased to 10 to 15 mm Hg and the LV a wave was small, 4 to 8 mm Hg.
APEXCARDIOGRAPHY IN LV DIASTOLIC PRESSURE

Simultaneous LV pressures and ACGs in a patient with CAD. Although the LVEDP was markedly elevated to over 30 mm Hg, the aWPA is only 10 and 15%. The early LV diastolic pressures are 19 and 13 mm Hg. The LV a waves measured 10 mm Hg. (It is not clear whether the LV a wave is truly even that large since the break near the top of the LV a wave may actually be the beginning of the a wave.) The P wave of lead II of the ECG provides evidence of left atrial enlargement.

Representative LV pressure tracings with simultaneous ACGs and plots of the LVEDP and aWPA of the ACG in two patients are shown in figure 3. The aWPA increases as the LVEDP increases, and the relationship between these two parameters approaches linearity. It can be seen that the early LV diastolic pressure is low, less than 10 mm Hg, and that the size of the LV a wave is large at a high LVEDP. Similar observations were noted in nine of the 11 patients whose LVEDP changed by 5 mm Hg or more.

In figure 4 are seen representative LV pressures and simultaneous ACGs in two patients in whom increases in LVEDP are associated with increases in the aWPA but in whom the aWPA does not significantly exceed 15%. Figure 5 shows the tracings of another patient with a resting LVEDP of 28 to 35 mm Hg in whom the aWPA is consistently 15% or less. Five patients had high LVEDPs associated with aWPAs which were not significantly above 15%. A fall in aWPA from 27% to 16% as the LVEDP was increased from 28 to 30 mm Hg with angiotensin was recorded in one patient in whom a linear relationship between increases in LVEDP and aWPA up to 28 mm Hg had been demonstrated previously (fig. 3, patient 1). Examination of the pressure tracings in these patients at the time that a high LVEDP and a relatively low aWPA were recorded revealed that the early LV diastolic pressure was high, up to 20 mm Hg, and that the contribution to LVEDP by atrial contraction as evidenced by the size of the a wave was small, less than 5 mm Hg in some instances.

The observation that high LVEDPs were associated with aWPAs of less than 15%, and
that this occurred when the LV a wave was small and the early LV diastolic pressure was high, prompted a comparison of the LV a wave to the aWPA of the ACG (Fig. 6). In contrast to figure 2, there is less scatter of points about the regression line and the correlation coefficient of 0.81 indicates that the aWPA of the ACG reflects more consistently the magnitude of the LV a wave than it does the LVEDP.

The following relationships were examined to determine whether they would provide information which would predict, from the ACG alone, which patients with an aWPA of less than 15% would have a high LVEDP and small LV a waves and which would have a normal LVEDP: (1) the relationship between the a wave amplitude of the ACG to the total diastolic deflection of the ACG (A/D) and the LVEDP, and (2) the relationship between the total diastolic deflection of the ACG as a function of the total vertical deflection of the ACG (D/O-E) and the LVEDP. No useful correlation was evident in either of these relationships.

Discussion

Apexcardiography has been used in several studies as a means of evaluating left ventricular function. It has been shown that patients with ischemic heart disease tend to have aWPAs which are greater than those of normal controls. It has also been demonstrated that in subjects with coronary artery disease the aWPA may increase significantly above base-line values after exercise and during attacks of angina pectoris. These findings have been interpreted as indicative of an increased LVEDP under these conditions. Conversely, the finding of a normal aWPA in patients with ischemic heart disease has been used as evidence of a normal LVEDP. However, although extensive experience with apexcardiography has been reported by several investigators, the number of observations of simultaneous intracardiac pressures and ACG is small.

In the present studies, an aWPA greater than 15% was associated consistently with an elevated LVEDP. The data are insufficient to establish confidently an upper limit of normal for the aWPA but do support the results of studies of large numbers of normal adults in whom aWPAs of greater than 15% are seldom seen. Left ventricular pressures were not obtained in these subjects.

The presence of an aWPA of less than 15% did not exclude significant elevation of the LVEDP. However, this is an important observation in view of the fact that a normal aWPA is often cited as evidence of a normal LVEDP. It has been stated that a normal aWPA in patients with ischemic heart disease may be an important prognostic and therapeutic observation, implying that such patients have normal LVEDP because the aWPA of the ACG is normal. These concepts must be modified in the light of the observations reported here.

Patients in the present study with high LVEDPs and aWPAs of less than 15% had small LV a waves. The early left ventricular diastolic pressure was high and often rose further before atrial systole. Decreased ventricular compliance or increased ventricular volume will produce such hemodynamic findings and could be responsible for LV a waves which are small relative to the absolute level of LVEDP.

The fact that the aWPA of the ACG predicts the magnitude of the LV a wave more accurately than the LVEDP is logical, in retrospect. The a wave of the ACG is a reflection of the left ventricular a wave which is caused by atrial systole. Both the a wave of the ACG and the left ventricular a wave begin at points on waves which represent diastolic filling of the ventricle prior to atrial systole, the slow filling wave of the ACG and the left ventricular diastolic pressure wave, respectively. That the magnitude of the a wave of the ACG would be influenced by the same factors which influence the magnitude of the left ventricular a wave, namely, the level of pressure in the left ventricle at the time atrial contraction begins, left ventricular volume, and left ventricular compliance, and not simply reflect the level of the end-
diastolic pressure, would be expected. The fact that the a wave “kick” and the aWPA may be small in the presence of an elevated LVEDP may be indicative of impaired left atrial function. In this manner the ACG may provide indirect information concerning the effectiveness of atrial contraction.\textsuperscript{24}

APEXCARDIOGRAPHY does provide information concerning left ventricular function, since in a given patient increases in LVEDP may be paralleled by increases in aWPA, and an aWPA greater than 15% indicates a high LVEDP. The fact that an aWPA of less than 15% does not necessarily indicate a normal LVEDP limits the usefulness of apexcardiography as an indirect tool and as the only means of evaluating left ventricular function.

Present information does not permit a more sophisticated analysis of the relative importance of the several factors which influence the apexcardiogram and the aWPA. The ACG is a complex tracing which indirectly records not only the effects of changes in pressure in the left ventricle but also of changes in its volume, position, and compliance. In this respect it records information which is not detectable by other means, but the ability to interpret this information is limited.

Acknowledgment
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References

1. BENCHIMOL A, DIMOND EG: The normal and abnormal apexcardiogram. Amer J Cardiol 12: 368, 1963
8. BANCHROFT WH, EDDLEMAN EE: Methods and physical characteristics of the kinetocardiographic and apexcardiographic systems for recording low-frequency precordial motion. Amer Heart J 73: 756, 1967
9. Rios JC, MASSUMI RA: Correlation between the apex cardiogram and left ventricular pressure. Amer J Cardiol 15: 647, 1965
13. Ross J, BRAUNWALD E: The study of left ventricular function in man by increasing resistance to ventricular ejection with angiotensin. Circulation 29: 739, 1964
17. MILLER A, WHITE PD: Crystal microphone for pulse wave recording. Amer Heart J 21: 504, 1941


Science and Magic

(Analogy: The Oak Host and the Mistletoe Graft)

But while science has this much in common with magic that both rest on a faith in order as the underlying principle of all things, readers of this work will hardly need to be reminded that the order presupposed by magic differs widely from that which forms the basis of science. The difference flows naturally from the different modes in which the two orders have been reached. For whereas the order on which magic reckons is merely an extension, by false analogy, of the order in which ideas present themselves to our minds, the order laid down by science is derived from patient and exact observation of the phenomena themselves. The abundance, the solidity, and the splendour of the results already achieved by science are well fitted to inspire us with a cheerful confidence in the soundness of its method. Here at last, after groping about in the dark for countless ages, man has hit upon a clue to the labyrinth, a golden key that opens many locks in the treasury of nature. It is probably not too much to say that the hope of progress—moral and intellectual as well as material—in the future is bound up with the fortunes of science, and that every obstacle placed in the way of scientific discovery is a wrong to humanity.—Frazer, JG: Golden Bough. New York, Macmillan Co., 1952, p. 825.
The Use of Apexcardiography in the Assessment of Left Ventricular Diastolic Pressure
GUSTAV C. VOIGT and GOTTLIEB C. FRIESINGER

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