High-fidelity, infinite time constant calibrated pressure apexcardiogram and its correlation with high-fidelity left ventricular pressure*†

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SUMMARY A technique of recording the pressure apexcardiogram by means of a high-fidelity piezo-resistive transducer has been developed in our laboratory. It permits the quantification of pressure with which the transducer is applied to the chest wall as well as the pressure changes during a cardiac cycle. In this preliminary report, apexcardiographic pressures were compared with simultaneously recorded high-fidelity left ventricular pressure in 32 patients. There were no significant differences in the timing of the left ventricular systolic upstroke and the "O" point. Peak dP/dt of the apexcardiogram occurred (10 ± 13 ms) significantly earlier than the intraventricular pressure recordings. Though there was a correlation between developed diastolic pressure (end-diastolic minus early diastolic pressure) measured by the apexcardiogram (20 ± 14 mmHg) and intraventricular pressure recordings (11 ± 6 mmHg), the former was significantly higher. Pressure overshoots in early and late diastole contributed to this overestimation by the apexcardiogram but the relative contribution of each phase varied from patient to patient. There was a similar correlation between systolic pressures, but this index tended to be underestimated by the apexcardiogram.

The data suggest that the apical impulse is not solely caused by the passive transmission of left ventricular pressure. The convenient, direct method of measuring application and developed pressure at the apex described in this report permits scientific investigation of the forces responsible for genesis of this impulse and its alteration in pathological states.

Conventional apexcardiography provides a record of relative displacement, that is, displacement of the chest wall enclosed by the transducer pick-up cup in relation to the surrounding area at the rim of the cup.^{1,2} The impulse cardiography of Mounsey³ and kinetocardiography of Eddleman⁴ record absolute chest wall movement at the point of measurement in relation to a fixed point in space. Both relative and absolute displacement recordings are significantly modified by physical properties of the chest wall and the application or loading pressure. Therefore, it has been felt for some time that the measurement of the force or pressure which causes the displacement would be more meaningful

and reliable. The basic principles underlying this technique have been elucidated by Willems *et al.*⁵ using either fluid-filled pressure transducers or Telco catheters in dogs and fluid-filled pressure transducers in man. Gleichmann *et al.*,⁶ and Witte and Heublen⁷ used strain-gauge transducers in human subjects to measure the force on the chest wall at the point of apical impulse, but, unfortunately, their technique did not permit measurement of application pressure, which must be known to calibrate the system properly.

We have developed a technique of recording the apexcardiogram by means of a high-fidelity piezo-resistive pressure transducer. It permits quantification of both application pressure as well as pressure changes during the cardiac cycle. In this preliminary report, we compare the results of external pressure recordings with intraventricular pressure recordings obtained by a similar transducer.

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Fig. 1 Prototype of pressure apexcardiographic system used in this study. The positive numbers on the amplifier are used to calibrate the pressure signal while the negative numbers on the left-hand side indicate the negative pressure applied to offset the loading pressure.

Subjects and methods

Our apexcardiographic transducer (Fig. 1) is a piezo-resistive strain gauge mounted on a thin stainless steel diaphragm approximately 2 mm in diameter. The dynamic range of the transducer is from zero to 1520 mmHg with a flat frequency response from zero to 10 000 Hz.

The four-leg active bridge is supplied with a highly regulated direct current voltage of ± 3 volts. The signal voltage is amplified by an instrumentation amplifier so that an output of 1 volt is equivalent to a pressure of 100 mmHg. The system is calibrated with air pressure using a sphygmomanometer.

The voltage generated by the total pressure (which is the sum of static pressure and dynamic pressure) is fed into the summing junction of an operational amplifier. A negative voltage is then applied to offset the output so that the "0" point of the apexcardiogram is close to the baseline. The loading pressure is determined from the negative voltage applied since the latter is calibrated (1 volt = 100 mmHg). The negative voltage opposing the loading pressure could be applied in steps of 0, 40, 80, 200, 400, and 800 mmHg. The signal is then

passed through a low-pass Butterworth filter with 40 db/decade roll-off above 25 Hz and electronically differentiated and calibrated (1 volt = 1000 mmHg/s) to measure dP/dt of the apexcardiogram. The undifferentiated signal is used to record the apexcardiographic pressure.

With the patient in the semi-left lateral recumbent position, the apexcardiograph transducer was held to the chest wall at the point of maximal apical impulse by means of a three-legged holder fastened to the patient with a three-way elastic strap. The transducer was advanced in the centre of the holder by means of a screw; thus, the loading pressure could be controlled by adjusting the advancement of the transducer. The apexcardiogram was recorded with a loading pressure of 200 and 400 mmHg. Loading pressure was increased beyond 200 mmHg until the maximal amplitude of the systolic wave was obtained. Respiration was held at the point where maximal amplitude was achieved.

Simultaneous recordings of the apexcardiogram, left ventricular pressure, and their first derivatives were recorded in 32 patients (23 men, nine women) undergoing cardiac catheterisation. Their mean age

Fig. 2 Calculation of pressures from the apexcardiogram (ACG). The signal has also been processed to yield the first derivative of pressure (dP/dt) and a phonocardiogram (phono). Developed diastolic pressure (DDP) is measured from the early diastolic nadir to the onset of the systolic wave upstroke. Systolic pressure is measured from the early diastolic nadir to the peak of the systolic wave.

Fig. 3 Identification of the onsets of the systolic wave upstroke and "0" points in simultaneous recordings of ACG and left ventricular (LV) pressure and their first derivatives (dP/dt). Systolic upstroke onsets were directly identified in the pressure traces. The "0" point was the point where a dP/dt of zero mmHg/s was reached in early diastole.

was 52 ± 14 years ($\pm$ SD). Their diagnoses were: atherosclerotic heart disease in 12, valvular aortic stenosis in six, aortic regurgitation in seven, mitral regurgitation in four, cardiomyopathy in two, and coarctation of the aorta in one. Three patients were in atrial fibrillation, and the remainder were in sinus rhythm. Left ventricular pressure was recorded with a Millar micromanometer catheter. Micromanometer pressure was corrected for the effect of gravity by superimposing its pressure over the fluid-filled pressure during slow rates of pressure change in diastole. The zero reference level for pressure measurement was 5 cm below the sternal angle.

For each patient, the following measurements represented an average of five consecutive cardiac cycles and were obtained from recordings taken at a paper speed of 100 to 200 mm/s, with 20 ms time lines. Developed diastolic pressure of the apexcardiograph was measured from the "0" point to the onset of the systolic wave upstroke (Fig. 2). Left ventricular developed diastolic pressure was left ventricular end-diastolic pressure minus mini-

mum left ventricular early diastolic pressure. Since the left ventricular early diastolic nadir was not a temporally distinct point in most patients, zero dP/dt in early diastole was selected as the "0" point for both the left ventricle and apexcardiograph in all cases (Fig. 3 and 4). Systolic pressure of the apexcardiograph was measured from the "0" point to the peak of the systolic wave (Fig. 2); apexcardiograph systolic pressure was not measured in seven patients since recordings were obtained only at high gain in these subjects.

Left ventricular and apexcardiograph pressure recordings were compared with respect to the occurrence of the onset of the systolic upstroke, "0" point and peak dP/dt . The following comparisons of pressures were made: left ventricular end-diastolic pressure versus left ventricular developed diastolic pressure; left ventricular end-diastolic pressure versus apexcardiograph developed diastolic pressure; left ventricular developed diastolic pressure versus apexcardiograph developed diastolic pressure; left ventricular systolic pressure versus apexcardiograph systolic pressure; left ventricular

Fig. 4 Simultaneous left ventricular and ACG pressure and dP/dt . The onsets of the systolic upstroke and "0" points are simultaneous. Developed diastolic pressure of the ACG is greater than LV developed diastolic pressure. ACG systolic pressure is less than the LV systolic pressure, but peak dP/dt 's are identical.

peak dP/dt versus apexcardiograph peak dP/dt. The comparison was made using paired data and Student's *t* test. The null hypothesis tested statistically was no significant difference between pairs. For these data, Pearson's correlation coefficient was derived and regression analysis performed assuming a linear relation. Only correlation coefficients significantly different from zero are reported.

Results

ONSET OF LEFT VENTRICULAR SYSTOLIC WAVE

The onsets of the systolic waves in the apexcardiogram and left ventricular pressure traces were not significantly different. They were simultaneous in 24 of the 32 patients (Fig. 4), occurred 10 to 40 ms earlier in the apexcardiogram of six patients (Fig. 3), and were earlier by 20 ms in the left ventricular pressure traces of two patients.

"0" POINT

The "0" point of the apexcardiogram was simultaneous with left ventricular pressure in 22 patients (Fig. 4), preceded it by 10 to 40 ms in eight patients, and followed it by 20 and 40 ms, respectively, in two patients (Fig. 3). The mean difference ($\pm$ SD) was 5 ($\pm$ 16) ms which was not statistically significant.

Fig. 5 Left ventricular end-diastolic pressure versus developed diastolic pressure determined from the apexcardiogram. The heavy solid line is the regression line, and the light solid line is the line of identity.

Fig. 6 Left ventricular developed diastolic pressure versus apexcardiogram developed diastolic pressure. The heavy solid line is the regression line and the light solid line is the line of identity.

OCCURRENCE OF PEAK DP/DT

Peak dP/dt of the apexcardiogram preceded that of left ventricular pressure by 5 to 35 ms in 20 patients (Fig. 3), occurred simultaneously with it in nine patients (Fig. 4), and followed it by 5 to 15 ms in three patients (mean ($\pm$ SD), 10 ($\pm$ 13) ms; $p < 0.001$).

DIASTOLIC PRESSURE

Left ventricular end-diastolic pressure in the 32 patients ranged from 6 to 27 mmHg (mean $\pm$ SD = 15 ± 6 mmHg). Developed diastolic left ventricular pressure ranged from 3 to 22 mmHg (mean $\pm$ SD = 11 ± 6 mmHg) and though this correlated with left ventricular end-diastolic pressure ($r = 0.79$), the two were significantly different ($p < 0.001$). The developed diastolic pressure of the apexcardiogram ranged from 3 to 71 mmHg (mean $\pm$ SD = 20 ± 14 mmHg) and correlated with left ventricular end-diastolic pressure ($r = 0.69$) (Fig. 5) and left ventricular developed diastolic pressure ($r = 0.62$) (Fig. 6), but was systematically higher than both ($p < 0.025$ and $p < 0.001$, respectively.)

Left ventricular and apexcardiographic pressure contours could be superimposed during mid-diastolic slow rates of pressure change in 22 patients (Fig. 7). In 16 of these 22, the developed diastolic pressure of the apexcardiogram exceeded that of the left ventricle by 1 to 18 mmHg (mean $\pm$ SD =

Fig. 7 Simultaneous tracings of apexcardiogram and left ventricular pressures with superimposition of pressures during slow rates of pressure change in mid-diastole. The tracing at the top is the electrocardiogram. The apexcardiogram demonstrates a negative "overshoot" in early diastole and a positive "overshoot" at end-diastole.

11 ± 5 mmHg). In five and four of the 16, this overestimation by the apexcardiogram was totally accounted for by early and late diastolic "overshoots", respectively. In the remaining seven patients, overshoots occurred at both points.

SYSTOLIC PRESSURE (Fig. 8)

Left ventricular systolic pressure in 25 patients ranged from 87 to 280 mmHg (mean $\pm$ SD = 160 ± 50 mmHg) and apexcardiographic systolic pressure varied from 60 to 684 mmHg (mean $\pm$ SD = 154 ± 129 mmHg) ($r = 0.69$). Compared with left

Fig. 8 Left ventricular systolic pressure versus apexcardiographic systolic pressure. The heavy solid line is the regression line and the light solid line the line of identity.

Fig. 9 Left ventricular peak dp/dt versus apexcardiographic peak dp/dt . The heavy solid line is the regression line and the light solid line the line of identity.

ventricular systolic pressure, the apexcardiograph systolic pressure was lower by 10 to 85 mmHg in 17 patients, higher by 3 to 404 mmHg in five patients, and equal in three patients. The patient with the highest left ventricular systolic pressure had an apexcardiograph systolic pressure 684 mmHg; with this patient excluded from the statistical analysis, apexcardiograph systolic pressure was significantly lower ($p < 0.025$) than intraventricular systolic pressure.

DP/DT (Fig. 9)

Left ventricular peak dp/dt ranged from 576 to

2062 mmHg/s (mean $\pm$ SD = 1309 ± 408 mmHg/s) and apexcardiographic peak dP/dt ranged from 477 to 3757 mmHg/s (mean $\pm$ SD = 1456 ± 620 mmHg/s). Correlation between the two was poor ($r=0.42$).

Discussion

Calibrated pressure apexcardiography employing piezo-resistive transducers has several distinct advantages: (1) it permits quantification of the apical impulse in mmHg and is essentially independent of chest wall displacement. The latter depends on chest wall compliance, which may vary from patient to patient. It should be noted, however, that the pressure perceived by the external transducer at the apex is caused by both active dynamic forces originating from cardiac motion, as well as passive transmission of ventricular pressure; (2) it permits quantification of the application or loading pressure. The pressure apexcardiographic transducer head must be applied to the chest wall with sufficient pressure so that proper coupling is obtained between it and the underlying ventricle. Since both displacement⁸ and pressure⁵ measured by apexcardiography have been shown to be dependent upon loading pressure, measurement of the latter is crucial for the quantification of any apexcardiographic signal; (3) the system has an infinitely long time constant (zero to 20 kHz band width), which circumvents the disadvantages of poor time-constant apexcardiographic recordings.

The method, however, has disadvantages and limitations: (a) it is not possible to record the apexcardiogram in all patients, though our experience has been that such failure occurs in patients whose apexcardiogram has not been obtainable by other techniques. (b) As with any apexcardiographic system, the position of the patient and placement of the transducer has a significant effect on the height of the signal, and both must be optimal to obtain the maximal impulse. (c) With this, our first model, we could quantify the loading pressure only in steps of 40, 80, 200, 400, and 800 mmHg. This did not allow the precise quantification which may be essential for acceptably reproducible measurements of loading pressure. (d) The transducers require careful handling since any irreversible deformation of the stainless steel diaphragm would modify the pressure calibration and any violent force applied to it (such as striking it on a hard surface) would destroy the transducer. (e) The system is relatively expensive.

When the pressure apexcardiogram was used to identify the onset of the left ventricular systolic upstroke, no significant differences in timing were found between this and internal left ventricular

micromanometer pressure tracings. Previously, using fluid-filled catheter systems for recording left ventricular pressure, Inoue *et al.*⁸ found that the apexcardiograph upstroke preceded the onset of left ventricular pressure rise by an average interval of 17 ms though Tavel *et al.*⁹ reported no significant differences in timing. Using micromanometer catheters, Fabian *et al.*¹⁰ also found that the left ventricular pressure upstroke followed that of the apexcardiogram by 20 ms but our data are in agreement with the findings of Manolas *et al.*¹¹ in humans and Willems *et al.*¹² in dogs who reported no systematic differences in timing of these two events. While there may be no significant discrepancy between groups of patients, individual variations may be based upon more than methodological error. As suggested by Tafur *et al.*¹³ and Tavel *et al.*⁹ the initial portion of ventricular mechanical systole may be associated with a change in ventricular configuration unaccompanied by a rise in pressure. If this change in configuration results in an outward thrust, the upstroke of the apexcardiogram may precede the left ventricular pressure rise, but if it results in retraction, then the initial rise in pressure may not be perceived by the external transducer and the upstroke of the apexcardiogram may follow that of the left ventricular pressure.

The early diastolic nadir of the left ventricular pressure trace represents an equilibration point where the pressure rise in the left ventricle resulting from filling cannot be compensated by the pressure fall caused by active relaxation. The point is not very sharp in the pressure traces of most patients, particularly when recordings are made at a high paper speed of 100 to 200 mm/s. Since the equilibration point should be equal to zero dP/dt in early diastole, measurements of the left ventricular pressure and the apexcardiogram were related to this reference point. They were identical or the apexcardiogram was earlier than the left ventricle except in a few cases. The earlier occurrence of the "0" point in the apexcardiogram may be a result of the relaxing but expanding heart causing a positive thrust on the chest wall while left ventricular pressure continues to fall.

Since there is no zero reference level, the pressure apexcardiogram is capable only of measuring changes in left ventricular diastolic pressure. If the externally recorded diastolic pressure changes were solely caused by passive transmission of left ventricular diastolic pressure, then both developed diastolic pressures should have been essentially equal. Though a correlation was found between apexcardiographic and left ventricular developed diastolic pressure, the former was systematically higher than

the latter. Since apexcardiographic and left ventricular pressure contours appeared to be superimposable in most patients during mid-diastolic slow rates of pressure change, we attempted to determine the relative contributions of early and late diastolic overshoots to this overestimation. Examination of data in this group of patients showed that both negative early and positive late diastolic overshoots contributed to the higher developed diastolic pressure recorded in the apexcardiogram. The relative contribution of each phase varied, however, from patient to patient. In the present study, we did not attempt to determine which factors (ventricular volume, wall thickness, tension) were important in determining the magnitude and phase of these overshoots. This investigation is now possible since the loading pressure and pressure changes sensed by the apexcardiogram can be quantified.

Although a correlation was found between left ventricular and apexcardiographic systolic pressures, the latter tended to underestimate systolic pressure in the majority of patients. This probably results from loss of proper contact with the underlying ventricle as the chamber progressively decreases in size during systole. However, the apexcardiogram was higher than the left ventricular systolic pressure in five patients and this may be because of the ballistic force exerted by the heart against the chest wall during forward ejection of the stroke volume. In a few patients, these positive and negative forces may cancel each other completely so that equal systolic pressures are recorded in the apexcardiogram and left ventricle. It is possible that there would be a better correlation between internal and external pressure if they were measured at the time of aortic valve opening as suggested by Kesteloot *et al.*¹⁴ If this were the case, there should be a good correlation between external and internal peak dP/dt . We did not find such a correlation, casting doubt on this premise.

It is clear that differences exist between left ventricular pressure and pressure measured at the apex, suggesting that the apical impulse is not simply caused by passive transmission of internal pressures. The convenient, direct method of measuring application and developed pressures at the apex described in this report permits the scientific investigation of forces responsible for the genesis of this impulse and its alteration in pathological states.

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