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Time relation between apex cardiogram and left ventricular events using simultaneous high-fidelity tracings in man

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In 10 patients without left heart valvular disease and having normal function of the left ventricle, the left ventricular apex cardiogram with its first derivative (dA/dt), left ventricular pressure with its first derivative (dP/dt), aortic pressure, electrocardiogram, and phonocardiogram were recorded simultaneously during cardiac catheterization. The apex cardiographic tracings were obtained by means of a transducer with infinite time constant and very high resonant frequency and the LV and aortic pressures with catheter tip-manometers. The onset of the systolic rise of apex cardiographic and LV pressures were found to occur almost simultaneously with the upstroke of LV pressure, preceding that of the apex cardiogram by only 2±4 ms (mean±1 SD). The summit of the systolic upstroke of the apex cardiogram (called E-point) occurred 37±9 ms after opening of the aortic valve and 41±9 ms after peak dP/dt. The peak of dA/dt preceded peak dP/dt by 10±4 ms. The protodiastolic nadir of the apex cardiogram (called O-point) occurred slightly earlier (19±16 ms) than the nadir of the LV pressure curve, with considerable variation.

In conclusion, this study using external and internal transducers with similar characteristics gives a new definition of the time relation between the externally recorded apex cardiogram and the haemodynamic events within the left heart in human subjects with normal left ventricular function.

The left ventricular apex cardiogram is widely used as a guide in the interpretation of the phonocardiogram (Benchimol, Dimond, and Carson, 1961; Parker, Craige, and Hood, 1971; Spencer, Behar, and Orgain, 1973; Perosio, Silva, and Ricci, 1973). The relative height of the A wave (Voigt and Friesinger, 1970; Parker *et al.*, 1971) and various time measurements (Gadient *et al.*, 1974; Manolas *et al.*, 1974) may serve as indices of left ventricular function. Practically all studies, which have been performed in order to correlate the apex cardiogram with the intracardiac events, were conducted using conventional recording systems: pressure transducers attached to catheters or pulse transducers with variable and frequently unknown delay times and finite time constants (Benchimol and Dimond, 1963; Tafur, Cohen, and Levine, 1964; Rios and Massumi, 1965; Tavel *et al.*, 1965; Forman *et al.*, 1967; Fabian, Epstein, and Coulshed, 1972). To our knowledge there have been experimental studies only (Willems, De Geest, and Kesteloot, 1971), in which the apex cardiogram and the intracardiac pressure curves were recorded simultaneously by pulse transducers with infinite time constant and by catheter tip-manometers.

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This report analyses the relation between left ventricular pressure events and apex cardiogram obtained by high fidelity instruments, carried out during cardiac catheterization in patients with normal left ventricular performance and without or with only minimal over-load of the left ventricle.

Subjects and methods

In 10 patients (diagnosis and standard haemodynamics are given in the Table), the following tracings were recorded simultaneously in the course of cardiac catheterization: (1) left ventricular apex cardiogram, (2) left ventricular pressure, (3) aortic pressure, (4) first derivative of apex cardiogram, (5) first derivative of left ventricular pressure, (6) lead II of electrocardiogram, and (7) apical external phonocardiogram (Fig. 1).

Patients

All patients were in sinus rhythm. The duration of the

TABLE *Haemodynamic data*

Case No.	Age and sex	Diagnosis	Heart rate (beats/min)	LVEDP (mmHg) Rest	Handgrip	SAP (mmHg)	DAP (mmHg)	MAP (mmHg)	CI (l/min per m²)	max dP/dt (mmHg/s)
1	20 M	Functional murmur	61	16	17	105	64	82	4·9	1450
2	25 F	Atrial septal defect; pulm. stenosis	73	11	13	106	72	79	5·0	1680
3	19 F	Persistent ductus arteriosus	90	11	—	100	62	82	4·1	1430
4	42 F	Pulm. stenosis	84	6	9	131	82	102	4·9	2110
5	49 F	Functional murmur	110	11	11	156	74	115	4·4	2550
6	28 M	Coarctation of aorta	75	12	12	129	83	104	4·6	1680
7	19 F	Persistent ductus arteriosus	77	10	12	97	64	72	5·3	1380
8	43 F	Stenosis of innom. artery	80	11	11	148	81	110	3·1	1750
9	23 F	Ventricular septal defect	82	11	6	106	77	97	4·1	1580
10	21 M	Persistent ductus arteriosus	80	10	13	102	70	84	6·2	1360
Mean ±1 SD	29 ±10		77 ± 23	11 ± 2	12 ± 3	118 ± 21	73 ± 8	93 ± 15	4·7 ±9·8	1700 ± 370

CI=cardiac index;
DAP=diastolic aortic pressure;
LVEDP=left ventricular end-diastolic pressure;
MAP=mean aortic pressure;
SAP=systolic aortic pressure;
Conversion from Traditional to SI units: 1 mmHg≈0·133 kPa.

QRS complex did not exceed 0·11 s in any patient. Premedication consisted of 10 mg chlordiazepoxide or 5 mg diazepam given orally 1 hour before catheterization. In all 10 patients there was no clinical or haemodynamic evidence of left heart valvular or myocardial disease; and there was no or minimal pressure or volume loading of the left ventricle. Three patients had a small persistent ductus arteriosus and one had a ventricular septal defect (all 4 patients with a left-to-right shunt of less than 15 per cent of the pulmonary flow); one patient had a small atrial septal defect (left-to-right shunt: 35 per cent of the pulmonary flow) and additionally a slight valvular pulmonary stenosis (peak systolic pressure gradient: 13 mmHg (1·7 kPa)). Two patients had functional murmurs, one patient a slight valvular pulmonary stenosis (peak systolic pressure gradient: 18 mmHg (2·4 kPa)), one patient a coarctation of the aorta (systolic pressure gradient: 9 mmHg (1·2 kPa)), and one patient a stenosis of the innominate artery.

Apex cardiograms

The apex cardiograms were obtained in the left recumbent position using a pulse transducer constructed in our laboratory, consisting of a Marey capsule (diameter: 2 cm) with the interior surface of this capsule directly connected, without air leakage (Kastor *et al.*, 1970), to a Bio-Tec-transducer (BT 250-T), which had a flat frequency response from 0 to over 15 000 H_z and no time delay. The distance from the transducer to the opening of the capsule measured 0·7 cm.

Because of its configuration this system had an infinite time constant and no measureable time delay.

Left heart catheterization

Left heart catheterization was performed for diagnostic purposes using the percutaneous transfemoral technique. Pressure curves were obtained by Statham SF_1 catheter tip-manometers, introduced into the left ventricle by the transseptal or retrograde approach and into the ascending aorta by the retrograde route. The first derivative of left ventricular pressure (dP/dt) and of the apex cardiogram (dA/dt) were obtained by an analog computer; the time constant of the computer for calculating dP/dt and dA/dt was 0·8 ms. The left ventricular function was studied at rest and during isometric exercise with handgrip using standard criteria of cardiac performance: left ventricular end-diastolic pressure and maximal rate of dP/dt.

All records were made on a 16-channel Electronics for Medicine oscillograph (DR-16) at a paper speed of 200 mm/s with time lines at 0·02 s. For each measurement 5 separate heart cycles were averaged.

Results

The mean heart rate of the 10 patients was 73±7

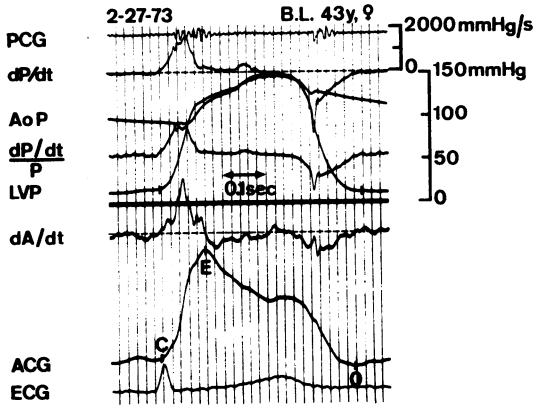

FIG. 1 *Simultaneous records of the left apex cardiogram (ACG) (lower tracing), and of the left ventricular (LVP), and of the central aortic pressure curves (AoP) (upper tracing) from Case 8. The top tracing shows an external apical phonocardiogram (PCG), the second tracing the first derivative of the left ventricular pressure (dP/dt), the fourth tracing the instantaneous quotient of dP/dt to total pressure ((dP/dt)/P) (not discussed in this paper), and the sixth tracing shows the first derivative of the apex cardiogram (ACG) (dA/dt); the lowest tracing is lead II of the electrocardiogram (ECG). C=the onset of the systolic upstroke of ACG; E=the summit of the systolic upstroke of ACG; O=the protodiastolic nadir of ACG.*

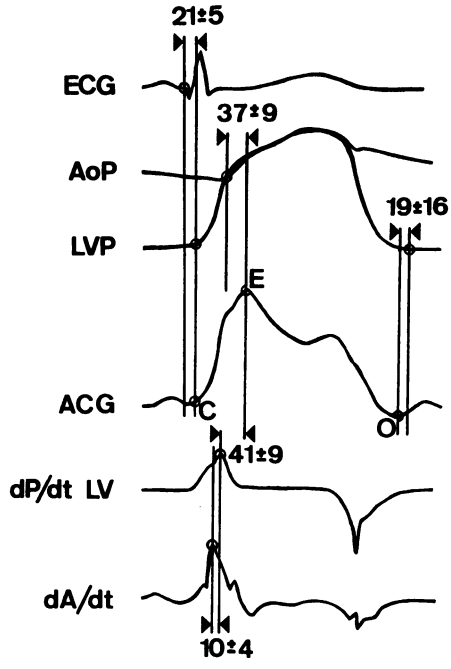

FIG. 2 *Schematic representation of mean time intervals ±1 standard deviation (in ms) of all patients demonstrating the temporal relation between apex cardiogram and left ventricular events. Abbrevations as in Fig. 1.*

(±1 SD) beats/min and the mean A wave percentage amplitude of the apex cardiogram was 9±5 per cent. The following points of the apex cardiogram were examined in respect of their time relation to LV and aortic pressure curves.

Onset of the systolic upstroke (C-point)

This point occurred almost simultaneously with the rise of left ventricular pressure curve (Fig. 1 and 2), the apex cardiogram following the left ventricular pressure by a mean time difference of 2±4 ms. The interval from the Q wave of the electrocardiogram to the rise in left ventricular pressure (the electromechanical delay) was measured in our study between the beginning of the Q wave and the C-point of apex cardiogram and averaged 21±5 ms.

Summit of the protosystolic upstroke (E-point)

This point followed in all cases both the peak dP/dt and the onset of ejection in the aorta, defined by the point of crossover of the left ventricular and aortic pressure tracings. The time difference between peak dP/dt and the E-point was 41±9 ms and that between the onset of ejection phase and the E-point 37±9 ms. The duration of the systolic upstroke of the apex cardiogram (time interval from C-point to E-point or C-E interval) measured 108±7 ms.

First derivative (dA/dt)

In only 6 of the 10 patients was a sharp peak dA/dt recognizable. The peak dA/dt preceded peak dP/dt by 10±4 ms. This finding corresponds to the more abrupt initial rise of the apex cardiogram compared to the more gradual one of the left ventricular pressure. The time interval from the C-point of apex cardiogram to the peak dA/dt (called t-dA/dt) averaged 58±6 ms.

Protodiastolic nadir (O-point)

The time difference between this point and both the C-point and the beginning of the aortic component of the second heart sound (A_2) showed a considerable variation; not only among patients with different or nearly the same heart rate but also un-

expectedly in different beats of the same patient with practically constant heart rate.

The time interval between C-point and O-point of the apex cardiogram measured 453±33 ms, and between A_2 of the phonocardiogram and O-point of the apex cardiogram 99±13 ms. There was no coincidence of the O-point with the protodiastolic nadir of the left ventricular pressure curve, the latter being the minimal value of left ventricular pressure and defined as the point where the first derivative of left ventricular pressure reaches zero after its minimal value. The time difference between the O-point of the apex cardiogram and the nadir of left ventricular pressure varied considerably, the O-point preceding the protodiastolic nadir of the left ventricular pressure by a mean interval of 19±16 ms.

Discussion

In recent years much attention has been directed toward the time relation between the apex cardiogram and the haemodynamic events in the left heart (Benchimol and Dimond, 1963; Tafur *et al.*, 1964; Rios and Massumi, 1965; Forman *et al.*, 1967; Fabian *et al.*, 1972; Willems *et al.*, 1971). Practically all these studies were conducted with the use of either conventional fluid-filled pressure recording systems with variable and mostly unknown catheter delay time and frequency response or of pulse transducers with often unknown or too short time constant, producing time shifts (Piemme, 1963; Kesteloot, Willems, and Van Vollenhoven, 1969) and distortions in the wave form (Bancroft and Eddleman, 1967; Kesteloot *et al.*, 1969). An accurate comparison of morphology and time between the apex cardiogram and intracardiac pressures is only possible when tracings obtained by highly sensitive transducers with similar characteristics are used. The different results given in numerous papers can be explained by means of different phase shifts and time delays; the resulting distortions affect significantly the exact time relations (Bancroft and Eddleman, 1967; Willems *et al.*, 1971). In the present study, these inaccuracies have been avoided by the use of a pulse transducer with an infinite time constant and by employing micromanometers with no time delay; all the transducers were strain-gauge manometers with direct-current output, which have high static sensitivity as well as high natural frequency, and are thus suitable for obtaining high fidelity tracings. To our knowledge similar tracings have been recorded only in experimental studies, by Willems *et al.* (1971); there are no corresponding studies in human subjects.

The most important and consistent finding of the present study was the synchronism of the onset of the systolic wave of the apex cardiogram and the onset of upstroke of left ventricular pressure; at times the former followed the latter by a mean time difference of 2 ms. This time difference is negligible, and therefore the apex cardiogram can be used for the exact external measurement of the electromechanical delay. This is in accordance with the findings of Willems *et al.* (1971) in dogs. The results so far reported in man are controversial (Benchimol and Dimond, 1963; Tafur *et al.*, 1964; Rios and Massumi, 1965; Tavel *et al.*, 1965; Forman *et al.*, 1967; Fabian *et al.*, 1972). However, these findings were mostly obtained with fluid-filled catheters and/or pulse transducers with too short a time constant. Tafur *et al.* (1964) found in 2 patients that the upstroke of the apex cardiogram preceded the onset of the rise in left ventricular pressure by 18 ms; Fabian *et al.* (1972) reported in 7 patients an average preceding of the upstroke of the apex cardiogram of 20 ms. Tavel *et al.* (1965) suggested 'an exceedingly close correlation' between the initial rise of the apex cardiogram and left ventricular pressure curves, but this finding was indirectly calculated using corrections for the delays in conduction of both pressure (through fluid-filled catheter) and apical pulsation (through crystal microphone transducer). The reported observations are not very valuable because first it is difficult to assess delay times in fluid-filled catheters, because of the variable, but almost inevitable presence of small air bubbles (Piemme, 1963), and secondly because the tracings recorded by pulse transducers with finite time constant are variously shifted in time and changed in form, depending on their dominant frequency (Kesteloot *et al.*, 1969).

The peak of the first derivative of the apex cardiogram has been found by Reale (1967) as mostly coinciding with the peak dP/dt and he proposed that the time from the peak of the R wave of the electrocardiogram to the peak of both derivates is identical and therefore valuable in detecting changes in myocardial contractile state. In the present communication the peak dA/dt of the apex cardiogram preceded the peak dP/dt of the left ventricular pressure by an average of 10 ms. This is somewhat in accordance with the finding of Willems *et al.* (1971) in dogs, who found an average interval of 20 ms.

The end of the upstroke of the apex cardiogram, called E-point, has been advocated by some authors (Benchimol and Dimond, 1963; Tavel *et al.*, 1965; Inoue *et al.*, 1970; Willems *et al.*, 1971), and opposed by others (Oreshkov, 1965; Spodick and Kumur, 1968), as a valid indicator of the onset of

the ejection phase of the left ventricle or of the peak dP/dt. In our study the E-point followed in all cases by a considerable interval both the onset of the ejection in the aorta and the peak dP/dt. The E-point followed the crossing point of left ventricular and aortic pressure curves by an average of 37 ms and the peak dP/dt by 41 ms. This is in contrast to the findings in dogs by Willems *et al.* (1971), who found a synchronism of a 'sharp E-point' with both the onset of the ejection in the aorta and with the peak dP/dt 'as long as the rate of early ejection was unimpaired and rapid'. According to our findings the E-point in human subjects with normal left ventricular function (and thus also rapid and unimpaired ejection) is no indicator of the above mentioned intracardiac events.

The protodiastolic nadir (O-point) of the apex cardiogram showed a considerable variation in its relation to other points (in this study its time difference to the C-point of the apex cardiogram and to the aortic component of the second heart sound was examined). The relation of the O-point to the protodiastolic nadir of left ventricular pressure curve also varied considerably, the first preceding the latter by an average of 19 ms. This is in contrast to the coincidence of these points found in dogs (Willems *et al.*, 1971).

According to our study the left ventricular apex cardiogram may serve as an indicator of the rise of the left ventricular pressure, and thus can be used to determine the electromechanical delay and, when recorded simultaneously with the carotid pulse curve, of the pre-ejection time interval; in contrast, it cannot be used to evaluate the onset of the ejection phase of the left ventricle, the peak of the first derivative, and the protodiastolic nadir of the left ventricular pressure.

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References

Bancroft, W. H., Jr., and Eddleman, E. E., Jr. (1967). Methods and physical characteristics of the kinetocardiographic and apex cardiographic systems for recording low-frequency precordial motion. *American Heart Journal,* **73,** 756.

Benchimol, A., and Dimond, E. G. (1963). The normal and abnormal apex cardiogram: its physiologic variation and its relation to intracardiac events. *American Journal of Cardiology,* **12,** 368.

Benchimol, A., Dimond, E. G., and Carson, J. C. (1961). The value of the apexcardiogram as a reference tracing in phonocardiography. *American Heart Journal,* **61,** 485.

Fabian, J., Epstein, E. J., and Coulshed, N. (1972). Duration of phases of left ventricular systole using indirect methods. I: normal subjects. *British Heart Journal,* **34,** 874.

Forman, J., Fouchard, J., Delzant, J. F., Varin, G., and Dupérier, C. (1967). Apex cardiogramme et courbes de pression. *Archives des Maladies du Coeur et Vaisseaux,* **60,** 1250.

Gadient, A., Manolas, J., Arbenz, U., Mehmel, H., Wirz, P., and Rutishauser, W. (1974). Zeitwerte im Apexkardiogramm bei Koronarsklerose. *Schweizerische Medizinische Wochenschrift,* **103,** 315.

Inoue, K., Young, G. M., Grierson, A. L., Smulyan, H., and Eich, R. H. (1970). Isometric contraction period of the left ventricle in acute myocardial infarction. *Circulation,* **42,** 79.

Kastor, J. A., Aronow, S., Nagle, R. E., Garber, T., and Walker, H. (1970). Air leaks as a source of distortion in apex cardiography. *Chest,* **57,** 163.

Kesteloot, H., Willems, J., and Van Vollenhoven, E. (1969). On the physical principles and methodology of mechanocardiography. *Acta Cardiologica,* **24,** 147.

Manolas, J., Wirz, P., Krayenbühl, H. P., and Rutishauser, W. (1974). Kontraktilitätskriterien am simultan mit dem linksventrikulären Druck registrierten, frequenz- und amplitudengetreuen Apexkardiogramm. *Schweizerische Medizinische Wochenschrift,* **104,** 1590.

Oreshkov, V. (1965). Indirect measurement of isovolumetric contraction time on the basis of polygraphic tracing. *Cardiologia,* **47,** 315.

Parker, E., Craige, E., and Hood, W. P., Jr. (1971). The Austin Flint murmur and the a wave of the apex cardiogram in aortic regurgitation. *Circulation,* **43,** 349.

Perosio, A. M. A., Silva, M. A. C., and Ricci, G. J. (1973). The first heart sound: its relation with the apex cardiogram. *American Journal of Cardiology,* **32,** 283.

Piemme, T. E. (1963). Pressure measurement: electrical pressure transducers. *Progress in Cardiovascular Diseases,* **5,** 574.

Reale, A. (1967). Evaluation of the contractile state of the human heart from the first derivative of the apex cardiogram. *Circulation,* **36,** 933.

Rios, J. C., and Massumi, R. A. (1965). Correlation between the apex cardiogram and left ventricular pressure. *American Journal of Cardiology,* **15,** 647.

Spencer, W. H., III, Behar, V. S., and Orgain, E. S. (1973). Apex cardiogram in patients with prolapsing mitral valve. *American Journal of Cardiology,* **32,** 276.

Spodick, D. H., and Kumur, S. (1968). Isovolumetric contraction period of the left ventricle. Results in a normal series and comparison of methods of calculation by atraumatic techniques. *American Heart Journal,* **76,** 498.

Tafur, E., Cohen, L. S., and Levine, H. D. (1964). The normal apex cardiogram: its temporal relationship to electrical, acoustic and mechanical cardiac events. *Circulation,* **30,** 381.

Tavel, M. E., Campell, R. W., Feigenbaum, H., and Steinmetz, E. F. (1965). The apex cardiogram and its relationship to haemodynamic events within the left heart. *British Heart Journal,* **27,** 829.

Voigt, G. C., and Friesinger, G. C. (1970). The use of apex cardiography in the assessment of left ventricular diastolic pressure. *Circulation,* **41,** 1015.

Willems, J. L., De Geest, H., and Kesteloot, H. (1971). On the value of apex cardiography for timing intracardiac events. *American Journal of Cardiology,* **28,** 59.

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