THE APEX CARDIOGRAM AND ITS RELATIONSHIP TO HÆMODYNAMIC EVENTS WITHIN THE LEFT HEART*

BY

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Low-frequency pulsations (20 cycles a second or less) appearing on the surface of the chest over the region of the cardiac apex have been studied sporadically for many years, but recently they have attracted considerable attention (Benchimol and Dimond, 1963; Dimond, 1964; Tafur, Cohen, and Levine, 1964a; Coulshed and Epstein, 1963; Nixon and Wooler, 1963). These pulsations possess considerable amplitude in comparison with the higher frequency sound vibrations and generally can be recorded easily. Most observers would agree that motion of the left ventricle causes the major, if not the only, low-frequency deflections on the chest wall in this region; however, interpretation of the recordings has been hampered by lack of precise knowledge about how these vibrations correlate with hæmodynamic events within the left side of the heart.

Benchimol and Dimond (1963) in their recent review of the apex cardiogram (ACG) indicated the relation between chest pulsations and intracardiac tracings, but their work was confined largely to the right heart, pulmonary capillary, and left atrial pressure curves. Others (Dimond, 1964; Tafur et al., 1964a; Coulshed and Epstein, 1963; Nixon and Wooler, 1963; Benchimol and Dimond, 1962) have correlated the ACG with left heart pressure dynamics, but these studies have been limited in number and require further elucidation. Animal studies have produced some additional information about the ACG (Benchimol and Dimond, 1963), but it is impossible to extrapolate human conditions from experience with animals, inasmuch as the intrathoracic relationships and chest contour are not necessarily comparable between species. The present study was undertaken, therefore, to ascertain the relation in humans between external chest pulsations and hæmodynamic events recorded simultaneously in the left heart during cardiac catheterization.

SUBJECTS AND METHODS

Fifty-four patients undergoing left and right heart catheterization form the basis of this report. Catheterization in all instances was prompted by the usual clinical criteria, and patients were included in this study solely on the basis that technically satisfactory apex cardiograms could be made. Satisfactory tracings were most often impossible to obtain when right ventricular hypertrophy was present; unsuitability was evidenced by varying degrees of systolic retraction over the area normally occupied by the apex. All patients included in the study had typical left ventricular electrocardiographic QRS complexes overlying the apex (V4 to V6). This provided further evidence that the records obtained reflected left-sided cardiac events (Hartman, 1956).

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The apex cardiogram was recorded using the technique of Benchimol and Dimond (1963). A linear crystal microphone (Sanborn No 374) was connected by means of rubber tubing to the side outlet of the Sanborn Microphone (No. 62-1500-C13) bell. This allowed for recording of sounds and low-frequency pulsations simultaneously. Band-pass filters were set as follows: apex cardiogram, 0-1 to 20 cycles a second; sounds, 120-500 cycles a second. Attenuation of waves outside these frequency limits is at the rate of 12 decibels per octave. Owing to limitations in position imposed by the catheterization procedure, patients were allowed to lie supine or, when necessary, tilted to a 30-40° left-downward position.

A multichannel machine (Electronics for Medicine, Model DR-8) was used to record the simultaneous ACG and the intravascular pressure curves. All transducers were standardized to register pressure with equisensitive deflections. Photographic recording was done at a paper speed of 100 mm./sec., and time lines were inscribed at 0-1 sec. intervals. For each measurement, a minimum of three separate cycles was used, and, if a minor disparity existed, the results were averaged.

Catheters were placed in the aorta via the percutaneous retrograde route. Left atrial pressures were obtained by the transseptal technique (Ross, Braunwald, and Morrow, 1959). Left ventricular pressures were obtained with the retrograde catheter, but when necessary for technical reasons, the transseptal catheter was used. In 33 instances, simultaneous left ventricular and left atrial pressures could be recorded concomitantly with the ACG. Standard right heart catheterization and brachial artery cannulation also were routinely undertaken.

The delay in conduction of pressure through the cardiac catheters was calculated to be 5±1 msec. and the delay through the recording apparatus of the apex cardiogram was 2±1 msec. Therefore, the recorded intracardiac pressure-pulse lagged approximately 3 msec. behind the deflections of the ACG. Corrections were made for this delay in the results below.

![Diagram](http://heart.bmj.com)
peak of this ascent is the "e" point, and is thought to indicate initial ejection from the left ventricle into the aorta. During the isovolumetric relaxation period, which follows the second heart sound, the tracing sharply descends. It ends with a downward angulation which is called the "o" point and which is believed to represent opening of the mitral valve. This point is followed by a steep ascent, the rapid-filling wave (rf), which reaches a peak and gives way immediately to a more gradual ascent, the slow-filling wave (sf). Both these diastolic ascents are believed to reflect the pattern of the left ventricular filling during diastole, being rapid at first and slow later. In some tracings, if the cycle length is sufficiently long, the slow filling wave levels off to a horizontal orientation. During this period, termed "stasis" (Nixon and Woofer, 1963), there is thought to be no further ventricular diastolic filling.

The "a" Wave. The "a" wave of the ACG coincides closely with the rise in atrial pressure during atrial systole. Because of the absence of points of sharp demarcation, however, exact measurements of the timing of these "a" waves in the ACG and left atrium were impossible. In general, the onset of the "a" wave in the ACG appeared to follow the "a" wave in the left atrium by a small interval (approximately 10–20 msec.). When atrial systole caused a presystolic pressure rise in the left ventricle, this rise coincided closely in time with the "a" wave of the ACG.

If the "a" wave in the ACG reached a height of 15 per cent or more of the total complex, it was categorized as abnormally large. Four patients with aortic stenosis manifested these abnormally prominent waves in their apex tracings (Table). The degree of aortic stenosis was considered to be severe in these patients, the peak systolic gradient across the aortic valve being 75–95 mm. Hg. In addition, all four patients had fourth heart sounds and raised left ventricular end-diastolic pressures, and three of the four had prominent pressure rise in the left ventricle during atrial systole. Two additional patients with aortic stenosis of equal or greater severity had normal precordial "a" waves; their left ventricular end-diastolic pressures, being near normal, were lower than those observed in the group above and showed no abnormal left ventricular deflections with atrial systole. Lesser degrees of aortic stenosis were not associated with large "a" waves in the ACG, left ventricular deviations, or with fourth heart sounds. We encountered two patients who showed severe rise in left ventricular end-diastolic pressure unassociated with aortic stenosis: both had severe isolated aortic insufficiency. One of these patients manifested large "a" waves in her apical tracings together with a fourth heart sound: she had a slightly prominent "a" wave in the left ventricular curve.

Patients with moderate or severe mitral stenosis showed small or absent "a" waves in the ACG (Fig. 2).

The "c" Point. This point in the ACG coincided with the onset of the rise of left ventricular pressure. On the average, the ACG began its ascent slightly before the pressure curve, with an
average difference of 0.7 msec. Moreover, there was relatively little scatter around this mean value (SD ± 3 msec.) (Fig. 3). Very commonly, the ACG rose abruptly from its baseline at the time of beginning left ventricular contraction, whereas the left ventricular pressure curve usually began to rise with a more gradual, less clearly demarcated slope.

Fig. 2.—Apex cardiogram and left-sided pressure curves in a patient with mitral stenosis. There is no identifiable "a" wave in the ACG and the rapid-filling wave is diminutive and short in duration. BA, brachial artery; LV, left ventricle; LA, left atrium. For further discussion, see text.

Fig. 4.—Apex cardiogram in a patient with severe aortic stenosis. There is a systolic plateau, resulting in the absence of a well-demarcated "e" point. A prominent "a" wave corresponds closely to the presystolic pressure deflection in the left ventricle. [C.A. = central aorta.]

Fig. 3.—Relation between "c" point of ACG and beginning of left ventricular pressure rise.
Ejection "e" Point. This point is not always well demarcated in the ACG, frequently being broad or slurred. One finds poor demarcation most often in patients with left ventricular hypertrophy, particularly of the systolic overload type, where the "e" point is not followed immediately by a sharp collapse, but rather by a systolic plateau (Tafur, Cohen, and Levine, 1964b) (Fig. 4).

Central aortic pressures recorded in 9 patients simultaneously with the ACG disclosed that the "e" point of the ACG followed initial aortic pressure rise by a variable interval. The average delay was ±26 msec. (range, 0–70 msec.).

The "o" Point. The "o" point of the ACG coincided best in most cases with the point at which the left ventricular pressure stops its rapid descent, usually just before it falls to its lowest level in early diastole. This is not a constant feature, for occasionally the "o" point was inscribed slightly earlier or later than this point.

There was only an approximate relationship between the "o" point and the point of crossing of left ventricular and left atrial pressures in early diastole. Fig. 5 depicts the relatively wide variation of the "o" point; it occurred as early as 0 msec. to as late as 52 msec. after the left ventricular-left atrial crossing point. On the other hand, opening snaps, when present, bore a closer relationship to this crossing point (15–30 msec. delay), as depicted in Fig. 6.

Finally, the diastolic pressure gradient between the left atrium and ventricle was assessed for its effect upon the timing of the "o" point. For this purpose, patients having pure or predominant mitral stenosis and a beginning diastolic pressure gradient of greater than 10 mm. Hg were compared with patients having mild or no mitral disease and showing
early diastolic gradients of less than 10 mm. Hg. Although there was considerable overlap, the results, as seen in Fig. 7, show a significant difference (p<0.01) between the two groups. In severe mitral stenosis, the "o" point generally follows the crossing of the left atrial to left ventricular pressure curves by a greater interval than when mitral stenosis is mild or absent.

We had too few patients to assess the effect of mitral insufficiency upon the "o" point. However, one patient was encountered who had severe mitral insufficiency and large "v" waves in the left-atrial tracing (Fig. 8), resulting in a relatively early crossing of the left ventricular–left atrial pressure curves at the beginning of diastole. In this example, the "o" point also was early and followed the crossing point by only 24 msec.

Rapid-filling Wave. This wave occurred almost always during a period when the left ventricular pressure was at its lowest. Owing to frequent artefacts in the tracing during early diastole, it was impossible to measure with complete accuracy the left ventricular pressure during this time. However, at the time of the peak of the rapid-filling wave, the left ventricular pressure appeared to be slightly lower than, or the same as, its level at the time of the "o" point. This circumstance applies to cases with or without mitral stenosis, though in the former group there appeared to be a slightly greater tendency for left ventricular pressures to fall further during the rapid-filling wave (Fig. 2). When mitral stenosis was absent, the left atrial pressure was usually level or falling slightly during the rapid-filling wave; in the presence of mitral stenosis, this pressure curve was consistently falling to some extent during this period. When a pressure gradient existed across the mitral valve during early diastole, it was usually maximal during or at the peak of the rapid-filling wave (Fig. 2, 8, and 9). Six patients manifested third heart sounds which occurred at the time of the peak of the rapid-filling wave. In this group the left ventricular or left atrial pressure curves were basically the same as those in patients without such sounds, though in two instances there was a tendency for these pressures to rise more abruptly following the third sound, reaching a peak at approximately 80 msec. after the peak of the rapid-filling wave (Fig. 9).

Remainder of Diastole. The slow-filling wave is an inconstant feature of the ACG. It was often absent, with the rapid-filling wave being replaced immediately by a period of stasis. In mitral stenosis, the rapid-filling wave was short, giving way to a relatively long, gradually ascending slow-filling wave which tended to continue slanting upward as long as a significant mitral gradient existed.

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**Fig. 7.** Scatter plot demonstrating that when mitral opening is early in the cycle (group with severe mitral stenosis and early diastolic gradient of greater than 10 mm. Hg), the "o" point tends to follow this early left ventricular–left atrial crossing by a greater interval. (Mean value of group with severe mitral stenosis, 36 msec.; group with absent or minimal mitral stenosis, 19 msec.)
This relationship, however, was only approximate; many exceptions were observed in which the slow-filling wave ended before or after the gradient disappeared.

**DISCUSSION**

Our data suggest that, with only minor limitations, the ACG may be used to predict accurately the onset of left ventricular contraction. The ACG frequently showed an abrupt rise at the time of the "c" point and often appeared to precede the more gradually rising intracardiac curve. On the other hand, occasional minimal lags were observed in the ACG. Delay in conduction of the impulse to the surface of the chest may account for this lag, though, in such instances, it is possible that before outward motion begins the heart is moving for a short interval in a direction perpendicular to the sensing device. It is not surprising that there were minor disparities between the ACG and the left-ventricular pressure curve, for the ACG probably reflects only the change in configuration of the left ventricle associated with the onset of contraction. This outward change in configuration may not necessarily bear a perfect relationship to the change in intracardiac tension, as determined by pressure measurements. This probably explains why, in some instances, we and others (Tafur et al., 1964a) could detect an outward thrust from the surface of the chest shortly before the intraventricular pressure rose.

Our data indicate that the beginning of left ventricular ejection could not be predicted as accurately as the beginning of contraction could be anticipated. It is likely that the timing of the "c" point is dependent upon a diminution of left ventricular volume during the early phase of ventricular systole. If the rate of early systolic ejection is rapid and, therefore, the volume lessens abruptly, it is probable that the "c" point will be sharp and relatively early. If early ejection is slow, as might be seen in myocardial diseases or aortic stenosis, the "c" point might be more blunted and appear to be delayed following the beginning of ejection. Our findings indicate that the "c" point is variable in...
a group containing mostly patients with abnormally functioning hearts, but this finding is not necessarily valid for people without heart disease.

It is uncertain exactly what factors influence the timing of the "o" point. Our studies suggest that this point is not simply an expression of mitral valve opening, but rather a complex reflection of an interplay of intracardiac volume and dynamics within the myocardial wall and surrounding structures. In patients without significant mitral stenosis, the "o" point coincides closely with both the expected time of mitral opening (crossing of left ventricular and left atrial pressure) and the time when the falling left ventricular pressure curve first reaches the baseline. When increased left atrial pressure causes mitral valve opening to occur earlier in the cycle—as is seen with predominant mitral stenosis—we commonly did not observe commensurate prematurity of the "o" point. Increased left atrial pressure as a result of severe predominant mitral insufficiency will also cause the mitral valve to open earlier. Our limited experience with this isolated situation suggests that the "o" point can be displaced to an earlier position pari passu the prematurity of mitral opening. These observations could mean that only a large inrush of blood in early diastole is capable of altering significantly the timing of the "o" point.

The fact that the "o" point bore no close and consistent relation to mitral valve opening in predominant mitral stenosis was an unexpected finding and militates against the use of this point in predicting accurately the time of mitral opening. Our findings, therefore, do not support the conclusion that, in the absence of an opening snap, one can measure the interval between the second heart sound and "o" point for the purpose of assessing the severity of mitral stenosis (Legler, Benchimol, and Dimond, 1963). Neither do our observations support the contention (Tafur et al., 1964a) that the ACG is accurate enough to be used to differentiate between mitral and tricuspid opening snaps. The "o" point in the apex cardiogram occurs sufficiently close to the time of mitral opening, however, to justify its use in differentiating the mitral valve opening snap from the left ventricular third heart sound (Benchimol, Dimond, and Carson, 1961; Taquini, Massell, and Walsh, 1940), particularly since the latter corresponds exactly to the peak of the rapid-filling wave.

The rapid-filling wave represents relatively great motion of the apex outward, and this occurs at a time when the intraventricular pressure appears to be near its minimum during early diastole. Such an observation is consistent with the view that active ventricular expansion takes place during the rapid-filling wave. This expansion probably provides the means of maintaining the lowest possible intraventricular pressure during early diastole, that is, the ventricle is exhibiting "diastolic suction". Whether and how much such a ventricular action contributes to filling in the healthy human remains the subject of considerable debate (Brecher, 1958; Fowler, 1960). Brecher (1958) has suggested that the possibility of decreasing viscosity of the ventricular walls in early diastole could serve as an alternative explanation for a falling ventricular pressure despite rapid filling in early diastole.

The peak of the rapid-filling wave probably marks the end of active ventricular expansion and the beginning of passive ventricular enlargement, for shortly thereafter the intraventricular pressure usually begins a slow rise, during which time the slow-filling wave is being inscribed over the apex. From study of the intracardiac pressures, we could marshal no evidence for an abrupt change in the rate of ventricular filling at the time of the peak of the rapid-filling wave. At the time of this peak, the left ventricular and left atrial pressures usually were falling slowly or remaining on a plateau and usually did not begin to rise until a variable period after this point. Our finding does not agree with that of Nixon and Wooler (1963), who found that with the peak of the rapid-filling wave there was a sudden deceleration of the left atrial "y" descent ("annular ascent point") (Radner, 1957), which would suggest that flow from left atrium to left ventricle is, likewise, decelerating suddenly. We did observe a somewhat abrupt deceleration in left atrial pressure in a few patients with mitral stenosis, but the point at which the pressure changed usually coincided with the time that ventricular pressure first fell to the baseline, and this event usually preceded the peak of the rapid-filling wave in the apex cardiogram.

Patients with mitral stenosis usually show shortening or absence of the rapid-filling wave (Radner, 1957; Benchimol et al., 1960; Snellen, 1958). We have usually been able to observe a brief,
THE APEX CARDIOGRAM

837
diminutive rapid-filling wave which appears to have a normally inclined slope, even in the presence of fairly severe organic mitral obstruction (R. W. Campbell, C. Fisch, H. Feigenbaum, and M. E. Tavel, unpublished data). Since it is extremely unlikely that flow across the mitral valve is rapid during this period, this is further evidence for suggesting that the slope of the rapid-filling wave is not a true reflection of the rate of ventricular filling.

From the several preceding observations, we have arrived at the following hypothesis regarding the early diastolic events over the apex: the rapid-filling wave is initiated by active ventricular elongation or expansion and is, to a certain extent, independent of the timing of mitral valve opening. The rapid outward motion continues until the inelastic structures within the ventricles, that is, the chordae and mitral valve leaflets, become taut and thereby check this motion (or at least elongation). After this point, the ventricle is still capable of further filling and slower enlargement in a more concentric direction, but this becomes a more passive event. It is likely that the actual rate of blood flow through the mitral orifice begins with or shortly after the “o” point, but it rapidly builds to its greatest velocity around the time of and shortly after the peak of the rapid-filling wave. If there is no obstruction across the mitral orifice, rapid and voluminous blood flow into the left ventricle will allow the rapid-filling wave to progress to a relatively high peak before being checked abruptly by the inner structures. On the other hand, mitral stenosis shortens this wave possibly by two mechanisms: first, fusion of the valve cusps (with or without shortening of the chordae) shortens the length of ventricular elongation before it is checked by tautening of these structures; second, the wall of the ventricle is inhibited from continuing its rapid elongation as a result of the inadequacy of early diastolic filling. In at least the latter situation, one might expect excessive negative pressure to be built up within the ventricle during the rapid-filling wave; our data suggest this possibility, but we require more accurate measurements, entirely free of artefacts, to substantiate the theory.

If this thesis proves to be correct, then the terms rapid- and slow-“filling” waves would be inaccurate and better terms might be rapid- and slow-“expansion” or rapid- and slow-“elongation” waves.

The mechanism of production of the third heart sound is also pertinent to this discussion. We noticed that the ventricular pressure was at or near its minimum level, and below that in the left atrium, at the time when third heart sounds were recorded. This finding confirms that of Crevasse et al. (1962). We and others (Nixon and Wooler, 1963; Dock, Grandell, and Taubman, 1955) have observed that, in patients with third heart sounds, the rapid outward motion of the heart is checked abruptly at the time of the sound and retracts slightly before continuing slow expansion, or “rounding out” (Dock et al., 1955). The point at which this abrupt checking of the outward motion occurs is at the peak of the rapid filling wave, and sudden selective tension of the inelastic internal structures probably provides the origin of the third heart sound. Experimental support for this idea can be found in the work of Dock (1959) who showed that forces within a physiological range could evoke sounds from the chorda and mitral valve leaflets, but not from the ventricular myocardium. The ability of the heart to continue expanding after the third heart sound likewise tends to deny a pericardial origin for such sounds. Whether or not third sounds become manifest probably depends upon the rapidity of ventricular expansion at the time the chordae become taut, which, in turn, probably is determined by a complex interplay of rate of inflow, volume of residual blood in the ventricle at the end of systole, length of the chordae, and myocardial distensibility.

Chorda tendinea tension throughout the cardiac cycle has been directly measured in dogs by Salisbury, Cross, and Rieben (1963) and provides only equivocal support for this hypothesis. Scrutiny of Salisbury et al.’s examples indicates that small, abrupt rises in tension occasionally occur in these structures at the expected time in early diastole, but they are not always present, and third sounds or apical motion were not studied. Tension of only one chorda was measured, however, and this may not have reflected the over-all tension on all chordae. Cutting of other chordae resulted in a more abrupt rise in early diastolic tension in the measured chorda, and this is in accord with the present hypothesis.

In all likelihood, the “a” wave of the apex cardiogram reflects filling of the left ventricle in res-
ponse to atrial contraction. Evidence in support of this contention is as follows: large "a" waves are seen in aortic stenosis and in other states featuring an increased left ventricular end-diastolic pressure and large left ventricular pressure deflections in response to atrial contraction (Benchimol and Dimond, 1962). In mitral stenosis, we found "a" waves to be absent or extremely small despite large left atrial "a" waves; this has also been noticed by others (Coulshed and Epstein, 1963). It is concluded that vigorous atrial contraction with good volume is necessary for such marked external outward motion; thus, when the large "a" waves are seen in the ACG, one is probably warranted in concluding that there is increased force of left atrial contraction, with some increase in left ventricular end-diastolic pressure, and that there is little or no obstruction across the mitral valve. We found that in aortic stenosis the large apical "a" wave was closely associated with and carried the same significance as the presence of a fourth heart sound. Our results agree closely with those of Goldblatt, Aygen, and Braunwald (1962) who have found that in aortic stenosis such sounds usually indicated raised left ventricular end-diastolic pressure and an aortic systolic pressure gradient of greater than 75 mm. Hg. In aortic stenosis, left ventricular chamber size is usually relatively small; hence, a relatively great change in left ventricular diameter might occur in response to vigorous atrial contraction. Such atrial activity might not cause as much change in left ventricular diameter in states characterized by cardiac dilatation, even with increased left ventricular end-diastolic pressures of equal magnitude. This probably explains the absence of abnormally large "a" waves in one of our patients with isolated aortic insufficiency with raised left ventricular end-diastolic pressure.

The close association between large apical "a" waves and fourth heart sounds suggests that the mechanism for the production of these sounds is inextricably associated with outward thrust of the left ventricle. Thus, the origin of both the third and fourth heart sounds is probably identical (Crevasse, et al., 1962): a sudden elongation of the ventricle with abrupt tensing of the internal structures capable of producing sounds, i.e. of the chordæ and mitral valve leaflets.

**SUMMARY**

A study was undertaken to correlate the low-frequency apical vibrations of the apex cardiogram (ACG) with haemodynamic events within the left heart, and the following observations were made:

The "a" wave of the apex cardiogram correlates closely in time with left atrial contraction and is frequently abnormally large when there are large pressure deflections in the left ventricle resulting from atrial contraction.

There is an exceedingly close correlation between the initial abrupt rise of ACG and the initial rise of the left ventricular pressure curve.

The "e" point of the ACG follows initial left ventricular ejection by a variable interval, averaging 37 msec.

The "o" point of the ACG occurs near the time of mitral valve opening, but does not correlate as well with the crossing of left ventricular and left atrial pressures as does the mitral opening snap.

The "rapid-filling wave" is inscribed at a time when the left ventricular pressure remains at its lowest level in early diastole. The left atrial pressure remains low or is falling gently during this period.

During the "slow-filling wave", the left ventricular pressure usually rises gradually. This is true also for the left atrium in the absence of mitral stenosis.

From these and other observations, we have attempted to explain the underlying mechanism of these apical events and have suggested tentative theories about the origin of third and fourth heart sounds.

**REFERENCES**


