Review

Fundamentals in vibrocardiography. Precordial accelerography and acceleration ballistocardiography

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Precordial vibrations have a frequency range from 0 to 1,500 c.p.s. The greater part of these vibrations are audible as heart sounds and murmurs. They are recorded in phonocardiography. Only a small range of vibrations below 50 c.p.s. is infrasonic. The amplitude of these infrasonic vibrations is so much greater than that of sound vibrations that no filter is needed in their recording for excluding cardiac sound.

Infrasonic vibrations may be recorded as *displacement*, *velocity*, or *acceleration* of the thoracic wall. *Precordial displacement* plays an important part in cardiac diagnosis, because it forms a link between cardiac sound and hemodynamics.[1-23] Most of the transducers employed in vibrocardiography will give a flat response to acceleration when actuated by means of a vibrator, or they transmit the higher precordial frequencies only and give tracings which closely resemble an accelerogram. For this reason, *vibrocardiography* has been more or less identified with *precordial accelerography*.[24-49]

Vibrocardiograms were shown to resemble acceleration ballistocardiograms,[38,46,48] but the initial systolic complex in vibrocardiography precedes the H-J complex in acceleration ballistocardiography. This preceding of the ballistocardiographic acceleration complex was attributed by Hollis[46] to a "time lag" in ballistocardiographic recording.

There are some other resemblances as well between acceleration ballistocardiography and vibrocardiography. Just as in acceleration ballistocardiography, alterations were found in vibrocardiography with aging,[26] smoking, and induced anoxemia,[47] with weakening of cardiac muscle[27] and coronary occlusion.[26,27,47] An abnormal reaction to exertion was found in angina pectoris.[27,49] Vibrocardiography was shown to indicate myocardial damage even before it could be found reflected in the electrocardiogram.[27,47]

These various resemblances between acceleration ballistocardiography and vibrocardiography led to the suggestion of replacing acceleration ballistocardiography with precordial accelerography in order to circumvent the errors introduced into ballistocardiographic recording by limb impedance[50] and coupling of the patient to the ballistocardiographic bed.[51,56,58,59]

Mounsey rejected this possibility be-

Received for publication Aug. 6, 1962.
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cause of the influence of thoracic build and of the thickness of the intervening tissues on the vibrocardiographic pattern.[30] Hollis pointed out the difficulty of the absence of a mass value in translating precordial acceleration into "dynamic quantities of force." He also referred to the "problem of variability of wave form in different precordial locations, more so then can be accounted for by simple phase shift."[46] Nevertheless, some authors strongly recommended replacing acceleration ballistocardiography with vibrocardiography.[38,48]

A suggestion of this kind implies the possibility of acquiring the same kind of information with vibrocardiography as with acceleration ballistocardiography.

Acceleration ballistocardiography has been used in the estimation of stroke volume,[60,62] and it has been related to cardiac "force."[57,59,60] It would seem necessary to test vibrocardiography as to this possibility of yielding a measure of stroke volume and of cardiac "force."

Exposition of the problem

Starr[60] states that "the amplitude of the ballistocardiogram is primarily related to the cardiac forces." In regard to stroke volume, he considers the relationship to be a limited one, "quite close when the ballistocardiogram is normal in form, but when the ballistocardiogram is distorted, the empirical formula giving best results when the flow is normal seriously underestimates the stroke volume."

It is evident that in cases of obstruction to the outflow, especially in aortic stenosis, there is no relationship of the ballistocardiogram to the cardiac forces at all. In regard to these estimations, ballistocardiography would seem to be of use in normal hearts only.

If it should be possible to determine cardiac "force" and stroke volume by means of precordial accelerography, this method might have the advantage of being useful in cardiac pathology as well.

It is necessary in this connection to realize exactly what precordial motion means.

If the heart were a stiff structure in which the "force" of ejection were generated in the same way as it is in a rifle, the reaction force to ejection transmitted to the thoracic wall might be comparable to cardiac "force," just as the repercussion of the rifle to the shoulder is comparable to the explosion causing the shot.

However, the heart is moved not only by repercussion, but it moves by itself, even before ejection. During the whole of systole it heaves and presses against the thoracic wall, especially by means of the apex. Precordial movement certainly is more intricate than is the movement of the body as a whole under the influence of ejection.

Analysis of precordial tracings has been attempted by means of correlation with intraventricular pressure tracings obtained during catheterization. The initial systolic complex of the vibrocardiogram has been ascribed either to isometric contraction and the first thrust of ejection[30,35,45,48] or to isometric contraction alone.[48] note on page 29; 49a

However accurate may be the determination of time relations between simultaneously recorded tracings, the fact that vibrocardiographic excursions coincide with intraventricular events does not prove them to be an expression of these events. Precordial acceleration is not acceleration in the rise of intraventricular pressure, nor yet is it acceleration of the heart moving on contraction. It is acceleration of the thoracic wall as it moves under the influence of ventricular contraction and emptying in systole, and ventricular filling in diastole.

The only way to interpret precordial acceleration correctly is by relating it to precordial displacement, of which it is the second derivative. The key to the question of the possibility of determining the "force" of cardiac contraction and stroke volume by means of precordial accelerography lies in the nature and the significance of precordial displacement.

Methods for recording precordial displacement and acceleration

Pulse tracings have been taken by means of a funnel or a tambour from the very beginning of registration on. The method was greatly improved by connecting the air tube with a crystal pickup. This combination was described for the first time in 1941, by Miller and White, who gave a detailed account of the frequency charac-

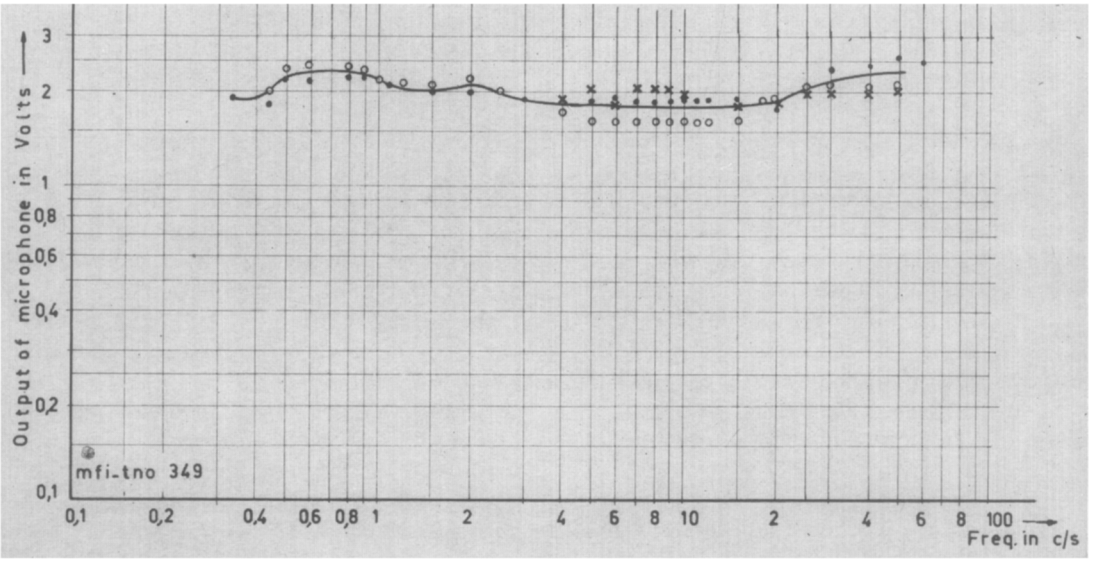

Fig. *1*. Frequency response for constant displacement of the tambour connected to a crystal pickup.

teristics of the pickup.[63] A year later, Rappaport and Sprague[64] mentioned this method in connection with precordial vibrations and called it "linear registration." Luisada and Magri[2] made use of it for taking precordial displacement tracings. It is the method employed by Holldack and Wolf[65] in their *Atlas und Kurzgefasstes Lehrbuch der Phonokardiographie.*

The "Elongationsgeber" developed by Schwarzer produces identical tracings, and so does the displacement meter by Philips adapted to precordial registration by Schneider and Klunhaar.[23]

We used a tambour in connection with a crystal pickup manufactured by Hellige Works, Freiburg, Germany. The recorder was a four-channel photographic phonocardiograph by Hellige or by the Atlas Works, Bremen, Germany. It makes no difference whether a funnel or a tambour is used, because with the funnel the skin acts as a membrane. We prefer the tambour, because with a closed system it is possible to examine the frequency response of the transducer as a whole. On actuation by means of a Goodman vibrator in which the amplitude was stablized with reference to the displacement meter by Hottinger, the tambour in connection with the crystal pickup proved to give a flat response to displacement in the region concerned, i.e., from 0.3 to 50 c.p.s. (Fig. 1). The recorder has a flat response from 0.1 c.p.s. upward.

With the use of a funnel or tambour the movement recorded is with reference to the ribs, on which the rim rests. The ribs and the intercostal space between them, the rim and the membrane, are all moved by the same impulse, but not to the same extent. The tracing is the result of subtraction, but it is not differentiated. We compared it with the tracing obtained by means of a photocell of the shadow thrown by a small cardboard rider attached to the skin. The photocell and the lamp were mounted in a unit which was attached to the patient's bed. The tracings obtained in this way give the actual movement of the thoracic wall which is not touched or influenced in any way.

The photocell was manufactured by Atlas Works, Bremen, Germany. The frequency response of this photocell in connection with the recorder proved to be flat to displacement from 0.1 to 150 c.p.s. on actuation by a vibrator.

A photocell of this kind would provide an ideal method for taking displacement tracings of the thoracic wall, if the apex did not move during contraction, and if a loose skin would not introduce disturbances of its own. The apex not only heaves during systole, it also moves upward and outward.

Because of this movement the cardboard rider on the skin is inclined to tilt and waver. With a loose skin, as for instance under the left breast in women, all kinds of secondary movements are introduced. Even in men it is necessary to tighten the skin by stretching the right arm over the head. Comparison then proved possible in young athletes with a somewhat broader apex beat. With the rider attached at the exact center of apical motion, reliable and reproducible apexcardiograms were obtained which proved to be practically the same as the apexcardiograms obtained by means of the tambour (Fig. 2). In a later paper a few slight differences will be discussed, but for our present purpose they are of no consequence. It suffices to state that the apexcardiogram as obtained by means of the tambour proves to be a displacement tracing without any differentiation.

In 1953, Hartman,[3-6] at Leiden, by means of the tambour method, started correlating mechanography with phonocardiography as a routine procedure. He took displacement tracings over the apex and the right cardiac thrust (if present), the carotid and femoral arteries, the jugular vein, and the liver, and established the normal time relations with the phonocardiogram. This pioneer work provided a firm basis for phonocardiographic interpretation. His method was adopted throughout the Netherlands. In regard to the apexcardiogram and the recordings of the right cardiac thrust, his results were confirmed by a group of American workers[7] after one of them studied the method during a stay of 3 months in Utrecht in 1956.

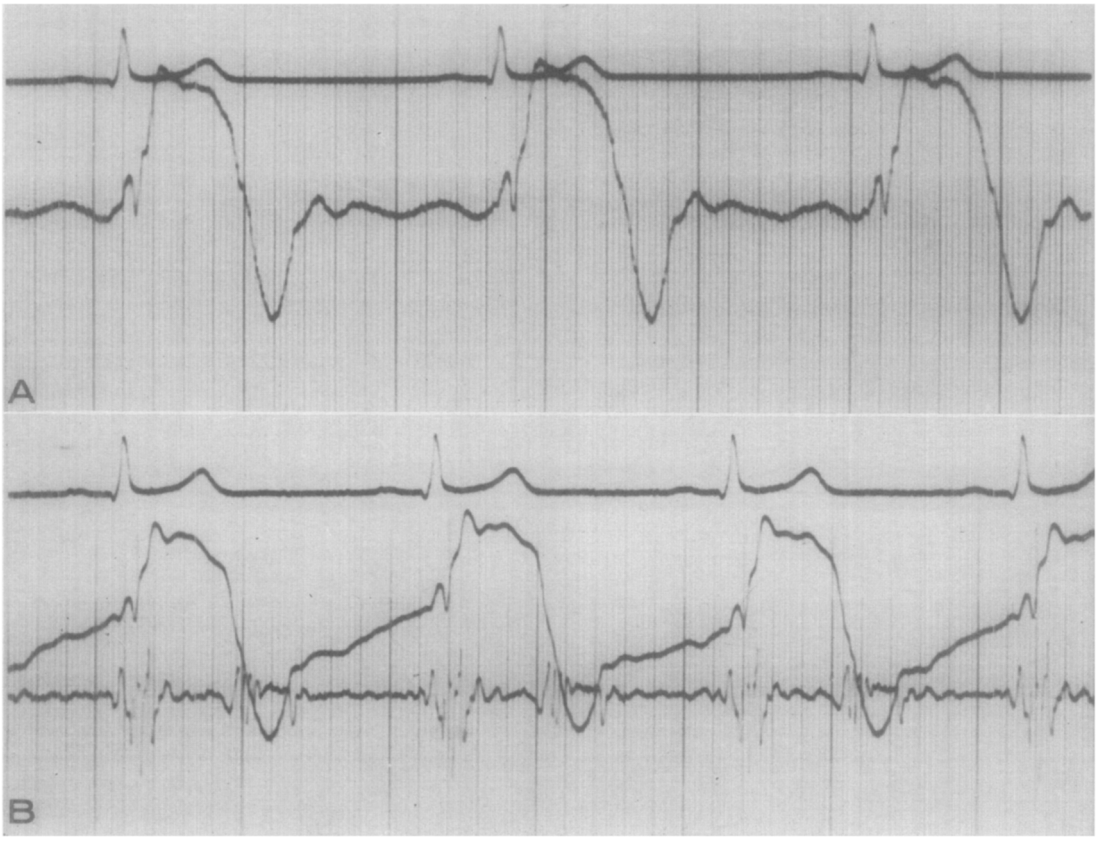

Fig. 2. Comparison of apexcardiograms obtained with the subject in the left recumbent position as taken: (*A*) by means of a *photocell unit* attached to the bed, from a shadow thrown by a cardboard rider attached to the skin over the apex, and (*B*) by means of the tambour. During systole the two tracings are almost identical. In diastole the skin fails to reflect slow ventricular filling, which in the tambour tracing is seen as a rising of the diastolic line.

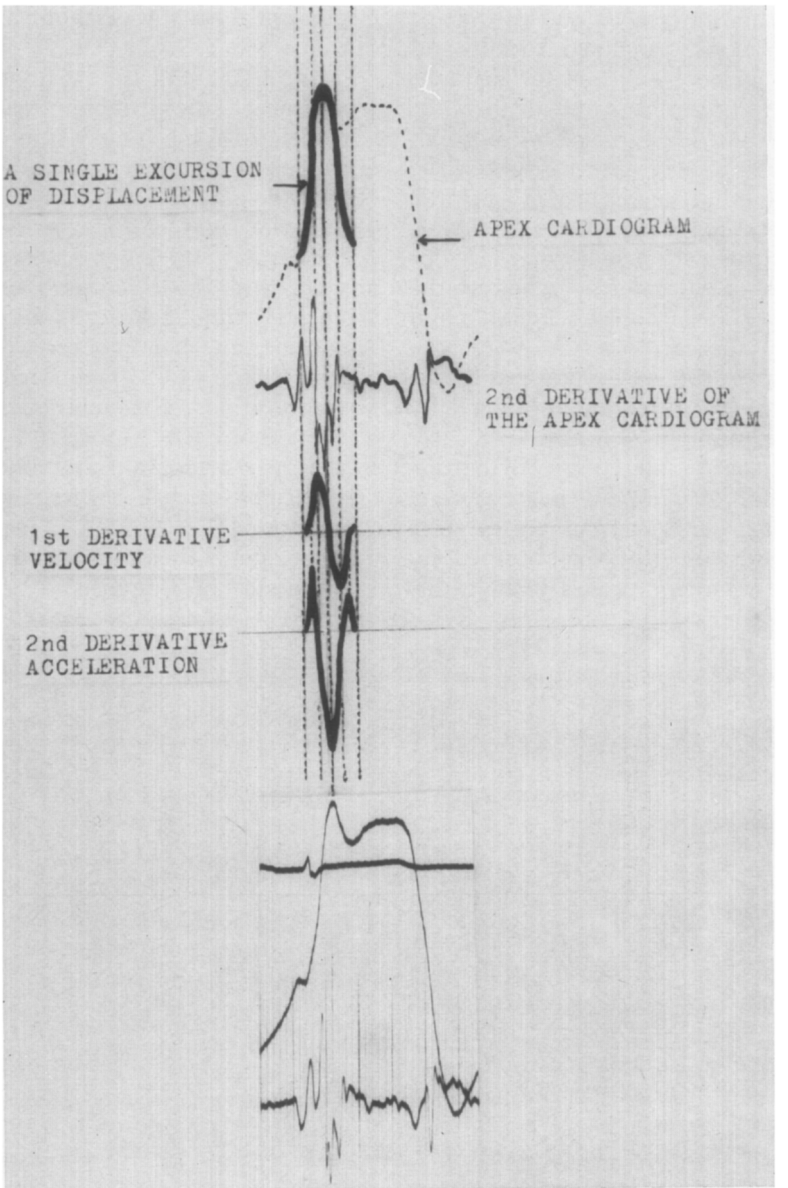

Fig. 3. A single excursion of displacement considered as forming the upstroke and the beginning of the top of the apexcardiogram. Comparison of the accelerogram which is derived theoretically from this single excursion of displacement with the second derivative of the apexcardiogram. At the bottom are the actual tracings from which the outline was taken for the comparison above.

Our own experience is based on complete phonocardiographic and mechanographic recordings in 3,300 cases, part of which were of normal people checked for sports competitions or of children with innocent murmurs. As for patients, the clinical diagnosis was confirmed in the majority by right-heart or left-heart catheterization, or both, by operation or autopsy. We added to the practice introduced by Hartman of recording both the apexcardiogram and an eventual right cardiac thrust by recording the movements over the "zone of retraction," movements low along the left sternal border and over the aortic and pulmonary orifice. These various tracings will be discussed separately farther on.

Recording is done with the subject in

the left recumbent position; in this position the contact of the heart with the thoracic wall is as close as possible, and the cardiac movements are not influenced by gravity. It is necessary in recording the apexcardiogram to choose the exact position in which the systolic plateau is completely positive. Otherwise, "artefacts" will be recorded, some examples of which Benchimol and associates[7] reproduced. The nature of these "artefacts" will be discussed in the section on "the zone of retraction."

We did not compare our results with those obtained by means of the method introduced by Eddleman,[8-17] nor with the tracings obtained with a new method by Beilin and Mounsey,[22a] since we lack experience with these methods.

In order to obtain an accelerogram at exactly the same place and at the same moment as the displacement tracing, the accelerogram was derived from it by means of double differentiation in the manner described by Burger and Noordergraaf for ballistocardiography.[51,52]

In the derivation of acceleration from displacement, however, a simple combination of resistances and capacitors proved to introduce a considerable disturbance, due to hum and noise. For this reason, operational amplifiers were used with an amplification of 30,000 and with frequency-dependent feedback. The amplifiers were stabilized in order to prevent drift. Cathode followers were used with the transducers in order to avoid hum at the input.

A few words on *the relationship between displacement and acceleration* may be of use in this connection. Acceleration may be derived from displacement either mathematically or, on recording, by means of differentiating electric filters.[52,53] The relation between a single excursion of displacement and its first and second derivatives (velocity and acceleration)[53] is shown in Fig. 3 (*heavily drawn lines*). In the ascending line of the single excursion of displacement there is a point at which the original concavity gives way to convexity. This point of greatest inflection marks the height of velocity. At the summit of the displacement tracing, velocity is nil: here the velocity tracing crosses the zero line. The nadir of velocity is reached at the point of greatest inflection of the descending line in the displacement tracing. Velocity then returns to zero again.

Acceleration is derived from velocity in exactly the same way. As may be seen from Fig. 3, a single excursion of displacement is represented by a positive and a negative excursion of velocity and by a positive, a negative, and a second positive excursion of acceleration. The nadir of acceleration coincides with the summit of the displacement tracing.

The beginning of the systolic plateau in the apexcardiogram, its upstroke and the first thrust of ejection, may be considered to be a part of just such a single excursion of displacement. We shall refer to it again in the analysis of the apexcardiogram and the accelerogram derived from it by means of double differentiation.

Precordial displacement and precordial acceleration as derived from displacement: the relation to cardiac "force" and stroke volume

In an extensive study on precordial motion, Dressler,[66] in 1933, pointed out the great difference between the movements of the exposed heart and those observed on the thoracic wall. The major movements of the exposed heart are in connection with cardiac filling and emptying. It is curious to see how little remains of these movements of filling and emptying on the thoracic wall. Here the chief movement is of the apex, which lifts as the heart contracts. In many cases it is the only movement which may be felt or seen.

In the normal heart the area taken up by the apex beat has, generally, a width of 1 inch or even less. In trained sportsmen, in thin and nervous people, in children, after exertion and with emotion it may be broader. It is widened in cardiac overloading, and in severe hypertrophy the precordial surface as a whole may be heaving.

The apex beat in the normal heart of a subject in the left recumbent position is due to left ventricular contraction.[3-7] The right ventricle is no more than a rind over the frontal face of the much stronger cone-shaped left ventricle. Its border reaches the apex and moves with the left ventricular thrust. A small thrust of right ven-

tricular origin may be observed along the left sternal border (Fig. 8,*B*). In right cardiac overloading it may be as forceful as the apex beat or even more so. In right ventricular hypertrophy and enlargement the left ventricle in many cases is forced away from the thoracic wall, and then the apex beat itself is due to the right cardiac thrust. The following description of precordial displacement and acceleration over various areas of the precordial surface deals with normal movements.

I. *The apex displacement cardiogram and the apex accelerogram.* With the subject in the left recumbent position the apex is brought nearest to the thoracic wall, and its movements are not influenced by gravity. In this position the systolic part of the apexcardiogram has the shape of a plateau with a descending top (Fig. 4,*A*). The upstroke starts 0.02 to 0.03 second after the beginning of ventricular activity in the electrocardiogram. Its summit marks the opening of the aortic valve. In Fig. 5,*A*, it is shown to precede the carotid upstroke by 0.02 second. In many cases the crest of the upstroke is somewhat broader and either rounded or notched (Fig. 5,*B* and *C*). Whatever the form of its crest, the end of the straight upstroke always precedes the upstroke of the carotid artery by as much as the lag of the carotid artery, and this crest itself adds but slightly to the apical upstroke in a normal heart. The height of the upstroke in normal people is determined by the aortic opening, i.e., by diastolic aortic pressure.

With the first thrust of ejection the pressure of the apex against the thoracic wall decreases somewhat, and because of this reduced apical pressure the plateau descends and is often more or less concave. The "force" of ejection maintains the elevation of the plateau, but cardiac emptying causes it to decline. Only with muscular relaxation at the end of systole does the "force" which maintained elevation drop off. The plateau ends with a sharp downstroke which is less steep than the upstroke and reaches the deepest point, the dip, within 0.15 to 0.20 second. The dip coincides with mitral opening.[3-7] The diastolic line goes upward, since it is an expression of ventricular filling: of rapid filling during the remainder of ventricular relaxation, of slow filling after relaxation is accomplished, and of filling by atrial contraction.[3-7]

As for precordial acceleration, we are interested in the first systolic part of the apexcardiogram. It is this beginning of systole which is of consequence in acceleration, because it is related to cardiac contraction and ejection. The downstroke of the apexcardiogram is related to ventricular relaxation only. A possible relationship between precordial acceleration and the "force" of cardiac contraction, as well as its relationship to stroke volume, must be searched for in this initial systolic complex of the precordial accelerogram.

Part of the single excursion of displacement described under a previous heading may be considered as constituting the beginning of the apexcardiogram: its upstroke and first peak (Fig. 3). In the apexcardiogram it is preceded by a small wave of atrial contraction. Generally, there is some overlapping of the final excursions due to atrial contraction and the initial excursions of ventricular systole in the acceleration pattern. With a large *a* wave the sharp transition between the *a* wave and the upstroke of the systolic plateau may by itself give rise to an excursion in the accelerogram. Moreover, every slight notch in the upstroke or in the first peak of the apex plateau may do the same.* The simple pattern of the accelerogram as derived from a single excursion of displacement is overlaid by these fluctuations of various origin. For this reason, the actual initial systolic acceleration pattern over the apex always is a more intricate structure than the theoretical accelerogram as derived from a single excursion of displacement. Yet something of the principle remains, as may be seen by a comparison of the theoretical and the actual acceleration patterns at the beginning of ventricular systole in Fig. 3. The descending line of the single excursion of displacement is not represented in the apexcardiogram. In its stead the line continues as the descending top of the apex plateau, thereby giving a more complicated sequel to the initial accelerographic excursions. The first peak of acceleration at the beginning of systole

*Part of these notches are the expression of cardiac sounds.

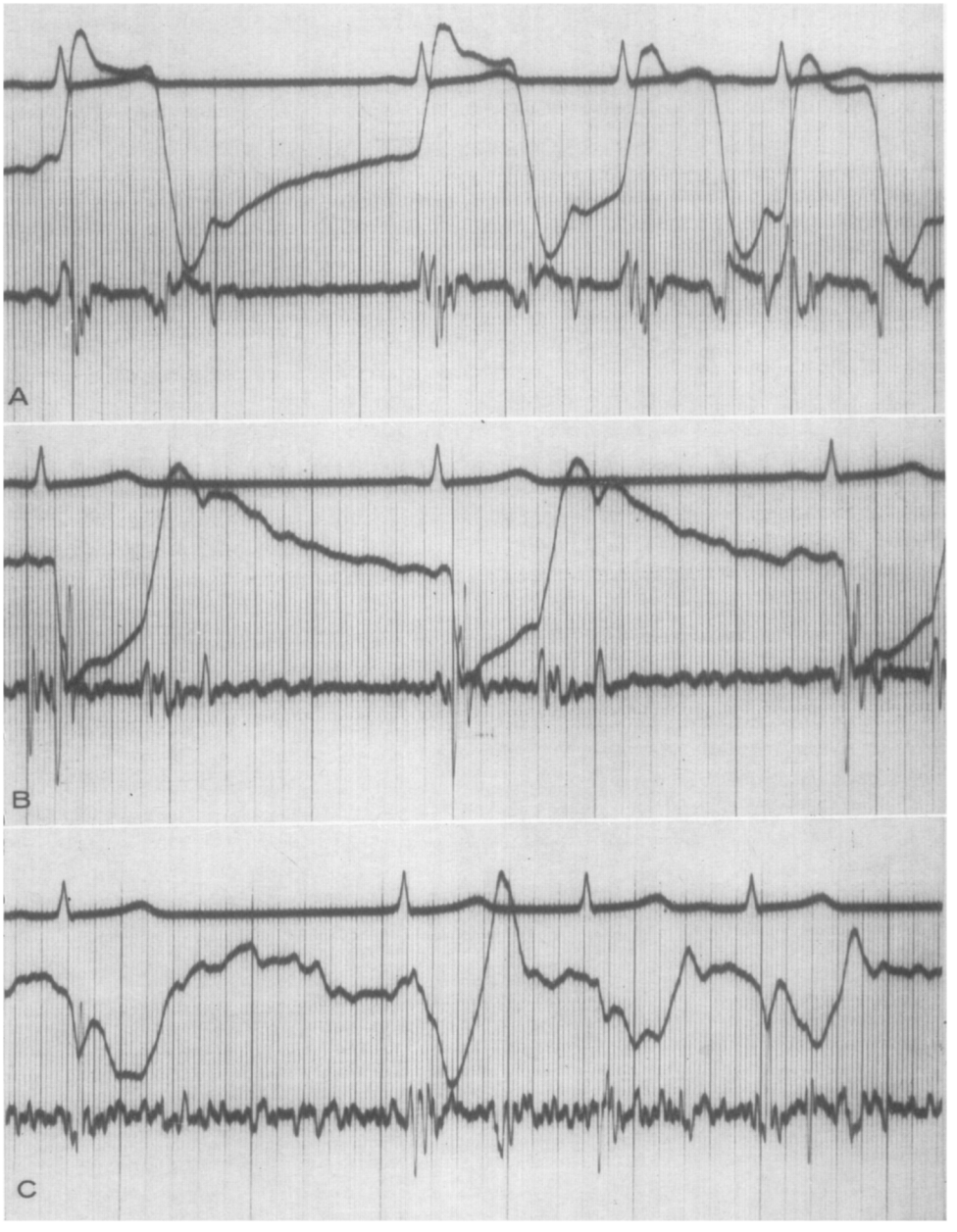

Fig. 4. Precordial displacement tracings with the accelerograms derived from them in a case of arrhythmia, with the subject in the left recumbent position. *A*, Apexcardiogram with accelerogram. *B*, Inverted apexcardiogram with accelerogram in the zone of retraction around the apex. Amplification 2.4 times the amplification of the apexcardiogram in *A*. *C*, Pulsations at 4L1, with accelerogram. Amplification 4.8 times the amplification of the apexcardiogram in *A*. The downstroke in *B* starts 0.04 second after the upstroke of the apexcardiogram in *A*, but otherwise the tracing is the exact reverse of the apexcardiogram. Not only is the systolic plateau inverted, but the diastolic filling line as well. For this reason, the inward movement around the apex cannot be due to cardiac emptying alone. In *C* the displacement tracing in systole begins with a downward movement which in the smaller beats is broken by a positive wave. In the second beat after a long diastole the inward movement is deeper and the positive wave has disappeared in this strong downward movement. The acceleration complexes are largest with the smaller beats, where they coincide with the sharp transition from downward movement to positivity.

and its nadir, which coincides with the summit of the upstroke, may be recognized in many cases. They must always be present, even though they may be deformed beyond recognition by incidental excursions due to minor irregularities of the displacement tracing. Every small notch in the apexcardiogram, provided that it is sharp enough, may give rise to relatively large excursions in the accelerogram, because in the accelerographic excursions the square of frequency of the excursions in displacement is involved.*

The nadir of acceleration at the beginning of systole coincides with the opening of the aortic valve or follows shortly after it. The acceleration pattern *precedes* this opening and *extends beyond it.* This means that the *first systolic acceleration complex over the apex is dependent on isometric contraction and on an interplay of the "force" of ejection and diminution of cardiac volume due to ventricular emptying. It is not related to cardiac "force" only. Consequently, there is no possibility of estimating the "force" of cardiac contraction from the accelerographic amplitudes over the apex.*

The highest point in the normal apexcardiogram is reached at the summit of the upstroke. This summit is conditioned by the opening of the aortic valve (Fig. 5), which depends on diastolic aortic pressure and not on stroke volume. In a case of arrhythmia the apex plateaus in the consecutive beats proved to be of equal height, although stroke volume certainly was considerably larger after a long diastole than after a short one (Fig. 4,*A*).

Since the height of the apexcardiogram depends on diastolic aortic pressure and not on stroke volume, the accelerographic excursions over the apex at the beginning of systole afford no possibility of estimating stroke volume.

II. *The zone of retraction.* Around the apex there is a zone of retraction, which has generally been ascribed to cardiac emptying[2,8-22a,66]; but tracings over this zone show a mirror image of the entire apexcardiogram. Not only is the systolic part inverted, but the diastolic line with its distinct phases of rapid and slow filling and filling by atrial contraction is inverted as well (Figs. 4,*B*, 6, and 7,*A*,*C*, and *D*).

Moreover, this mirror image may be found to the left of the apex beat, outside of any contact with the heart itself (Fig. 7,*C* and *D*), although less consistently and with smaller excursions.

Mirror images of this kind may be found likewise alongside of the arteries (see Figure 18a and b in the atlas of Holldack and Wolf[65]). A reaction of the soft tissues, which are drawn in around any point which is pushed outward by pulsation, may account for it. Whatever the explanation, it is a feature in precordial registration which must be reckoned with.

At the transition zone between the apex and the zone of the mirror image, composite tracings are found. The apex moves during contraction. In consequence of this movement the second half of the systolic plateau in the apexcardiogram becomes inverted if the tambour loses contact with the apex late in systole; a sharp inward movement at the beginning of the plateau may be caused by its gaining contact with the apex only after the systolic heaving has begun. The crest of the upstroke in systole and the dip and rapid filling in diastole would seem to be the last parts to resist the inversion in many cases (Fig. 6,*C* and *D*).

If the area of apical heaving is enlarged, the mirror image may be found along the left sternal border (Fig. 7,*A*). In severe hypertrophy, with heaving of the precordium as a whole, the mirror image may be found over the right side of the thorax.

The zone of the mirror image of the apexcardiogram, for obvious reasons, is even less suitable than is the apex itself for the derivation of cardiac "force" or stroke volume from the accelerographic excursions.

III. *The zone of relative tranquility.* In most normal hearts there is a zone of relative tranquility beyond the zone of the mirror image, especially in the third and fourth intercostal spaces along the left sternal border. Even in a normal heart a diminutive right cardiac thrust may be found somewhere within this zone, especially in children (Fig. 8,*B*). It may be recognized by its having the shape of a plateau,

*$x = \sum A_k \sin 2\pi ft.$

$$\ddot{x} = -\sum_{k=1}^{\infty} 4\pi^2 f^2 \sin 2\pi ft.$$

but its height in recording generally amounts to a few millimeters, in contrast to an apex plateau of 50 mm. In right ventricular hypertrophy it may attain the same dimensions as the apexcardiogram, and the right ventricular thrust may dominate this region completely.[3-5]

In a normal heart the right ventricular thrust, if present, is restricted to a small area. In the remainder of the zone of tranquility a shallow inward deflection is found. If this downward movement starts with isometric contraction, as in Fig. 8,*D*, cardiac emptying alone cannot account for

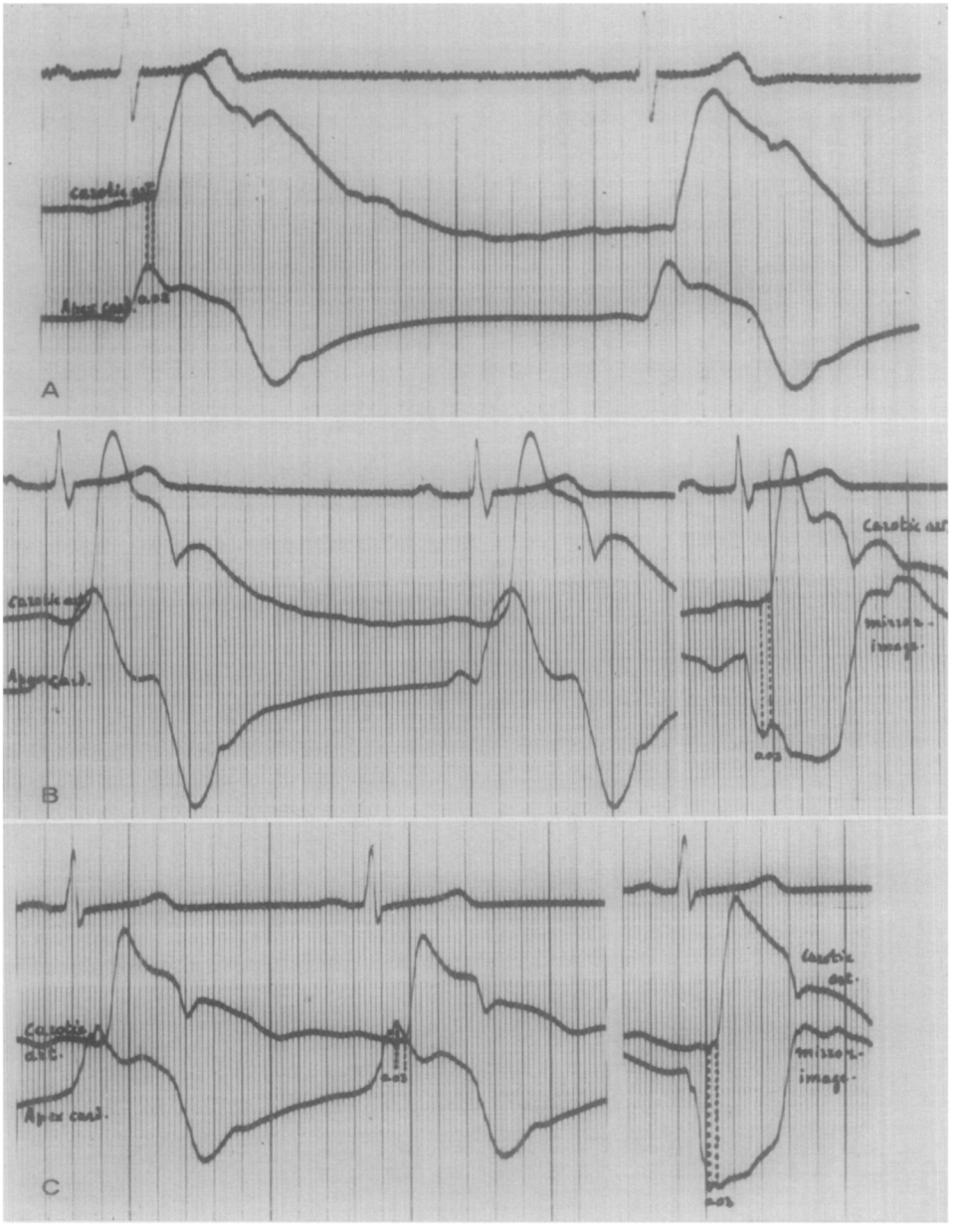

Fig. 5. Three different shapes of the upstroke in normal apexcardiograms, the summit of which may be sharp, as in *A*, or somewhat broader and either rounded (*B*) or notched (*C*). In all instances the end of the straight upstroke precedes the carotid upstroke by as much as the carotid lag. Its height is conditioned by aortic opening. If the broader crest surpasses the carotid upstroke, as in *B*, it hardly adds to the upstroke in the apexcardiogram of normal hearts.

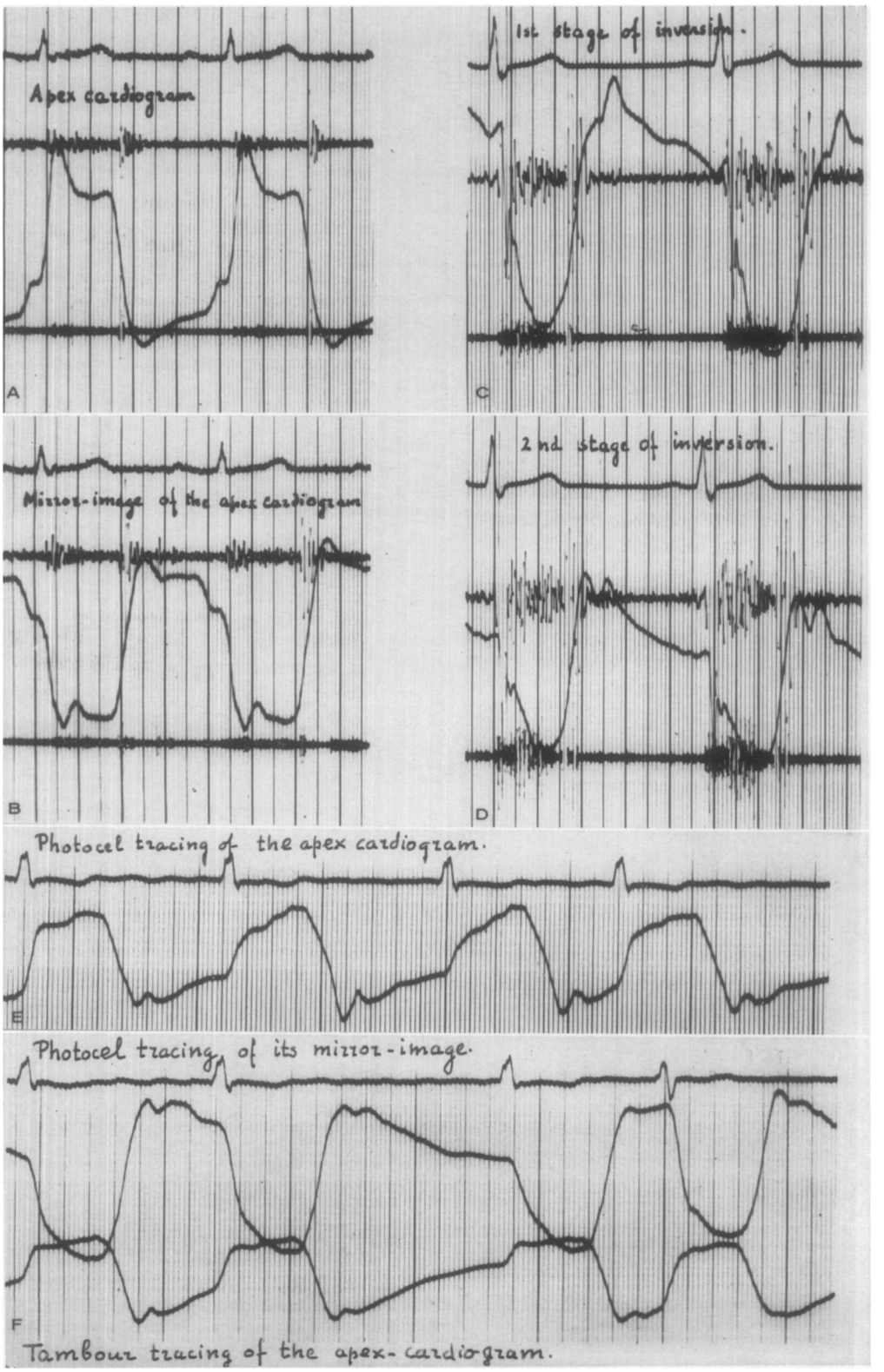

Fig. 6. *(For legend see opposite page.)*

it. Again the mirror image of the left or the right cardiac thrust may be of influence. Within this inward deflection a small positive wave is found, which resembles the summit of the upstroke in the apexcardiogram or even its cut-off top (Fig. 8,*A*). It may be due to a remnant of the left or right cardiac thrust which asserts itself within the inward deflection. It even may be due to the reaction force to cardiac ejection. In the latter case, there might be some reason for comparing precordial acceleration at this point of predilection for vibrocardiography with acceleration in ballistocardiography. The sequence of negative deflection and positivity certainly creates a spurious resemblance to the pattern of the displacement ballistocardiogram, which may account for the similarity between the vibrocardiogram taken at this point and the acceleration ballistocardiogram. Yet, in ballistocardiography the negative excursion followed by a positive wave is due to the turning of the main stream of blood in the aortic arch, which has no counterpart in precordial movement. The sequence of negative deflection and positivity on the thoracic wall arises from two movements which may change independently. This is borne out by Fig. 4,*C*. In this case of arrhythmia the positive wave is seen best with the smaller beats after a short diastole in the first and fourth complexes. With the larger second beat after a long diastole the inward deflection is deeper and the positive wave has completely disappeared in its strong downward movement. If this positive wave is due to repercussion, it certainly is not its unmitigated expression. The accelerographic excursions at the beginning of systole appear to be largest with the small first and fourth beats, where they coincide with the sharp transition from downward movement to positivity. In the larger second beat this transition is absent, and the accelerographic excursions are smaller.

In the zone of tranquility various contrasting movements are found, which may change independently. The accelerographic excursions are favored by sudden transitions between these movements, which bear no relationship to the "force" of contraction or to stroke volume.

IV. *The base of the heart.* Arterial pulsations may be recorded over the aortic or pulmonary orifices in cases of dilatation, as for instance in the poststenotic dilatation of aortic or pulmonary stenosis. These pulse waves are of the arterial type. They start with ejection, and in the downstroke an incisure is visible, which coincides with the second aortic or pulmonary sound.

In right ventricular overloading and dilatation of the right ventricle the ventricular thrust may be recorded even in the second left intercostal space.

In a normal heart, pulsations in this area are small—generally, too small for recording. The distance from the thoracic wall is greater than over the heart itself and there is a considerable damping by lung tissues. The objections by Mounsey[30] and Hollis[45] to the use of precordial accelerograms for quantitation are more stringent yet as regards pulsations over the aortic and pulmonary orifices.

To conclude, it must be stated, that *no area of the precordium would seem to be suitable for the derivation of either stroke volume or the "force" of cardiac contraction from the accelerographic excursions.*

Fig. 6. *A*, Apexcardiogram. *B*, Mirror image of the same apexcardiogram taken with 14/6 times its amplification. In systole the downward movement starts with isometric contraction; in diastole the filling line with its distinct phases of rapid and slow filling and filling by atrial contraction is inverted as well. Cardiac emptying alone cannot account for this mirror image, nor for the following ones. *C* and *D* are two stages of inversion in another case. In *C* the peak of rapid filling is still positive; in *D* the diastolic line as a whole is inverted. *E* is a photocell tracing of the apex movement in a third case. *F* is the mirror image of this apex movement taken by means of the photocell (*above*) with the simultaneously recorded tambour tracing of the apex movement (*below*). Amplification of the mirror image in *F* is 3 times the amplification of the apex cardiogram in *E*. These mirror images were all recorded over the right ventricle. During systole, cardiac emptying tends to increase the downward movement of the mirror image, but in diastole, right ventricular filling is opposed to it. The downward line of the mirror image in diastole will show only if it can prevail over the influence of cardiac filling.

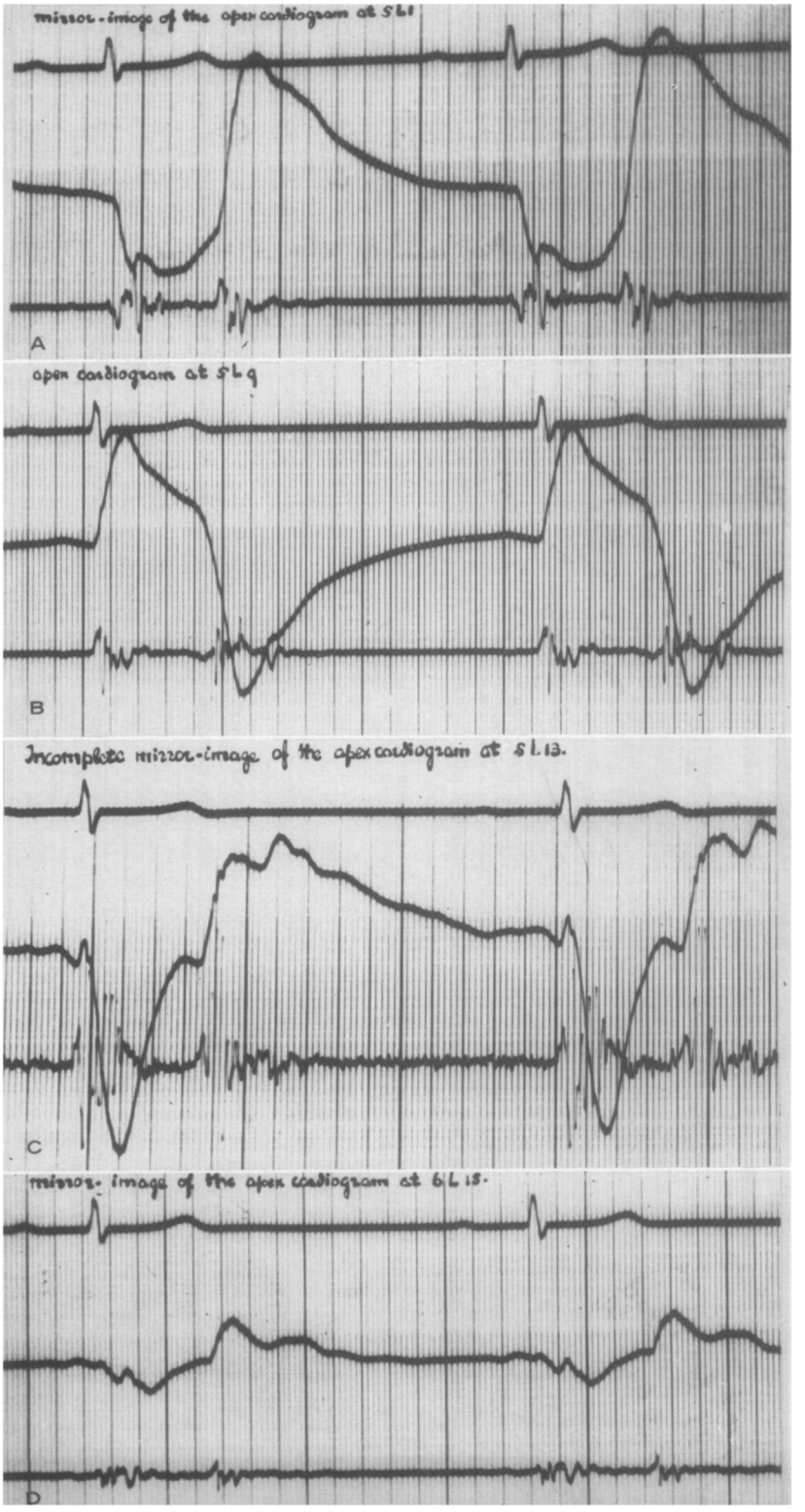

Fig. 7. (*For legend see opposite page.*)

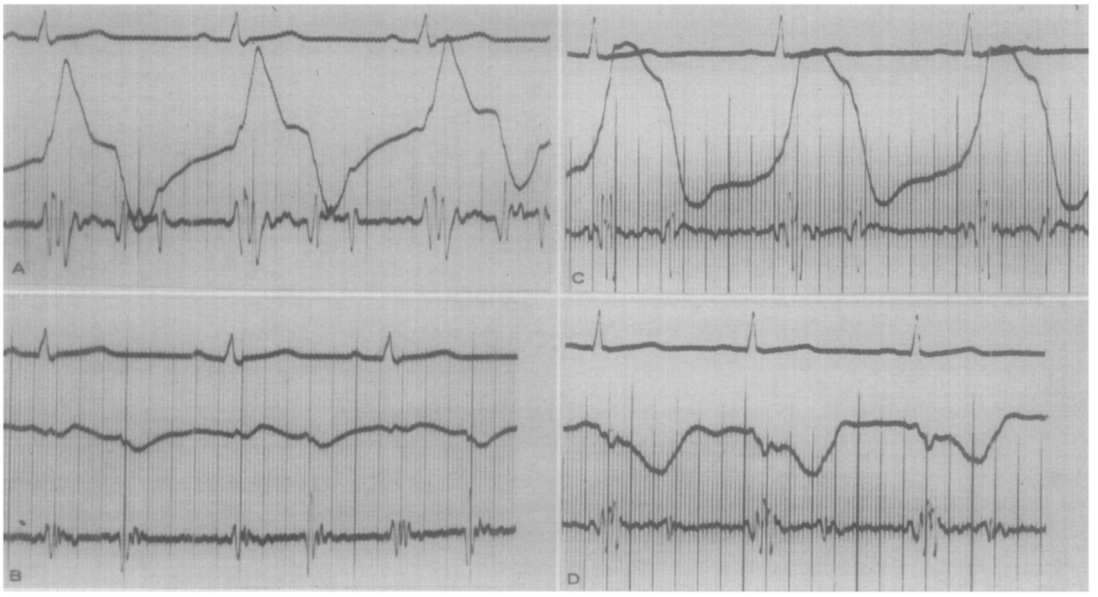

Fig. 8. Apexcardiogram (*A*) and small plateau at 4L1 of a slight right ventricular thrust (*B*) in a normal heart. *C* is another apexcardiogram and *D* is the tracing at 4L1, in which a downward movement starts with isometric contraction. Within this downward deflection a small positive wave is seen, the highest point of which coincides with the crest of the upstroke in the apexcardiogram. Constant amplification.

Comparison of the accelerographic amplitudes over the apex in mitral and aortic stenosis

In clinical practice, nevertheless, we are accustomed to estimate the 'force" of cardiac contraction by means of palpation of the apex beat.

In order to clinch the matter beyond all possibility of doubt, we proceeded to derive accelerograms from apexcardiograms in widely different hemodynamic conditions. Two cases of severe mitral stenosis were compared with two cases of severe aortic stenosis. The four patients were in the same age group and of similar build. Amplification of the apexcardiograms was the same, and the relationship between the amplification of the apexcardiograms and the accelerograms derived from them also remained the same.

The four cases selected represented the following hemodynamic conditions: Case 1: *Severe mitral stenosis*—poor ventricular filling, low resistance to ejection (Fig. 9,*A*). Case 2: *Severe aortic stenosis*—small ventricular cavity, consequently poor ventricular filling, high resistance to ejection (Fig. 9,*B*). Case 3: *Severe mitral stenosis with some regurgitation and atrial fibrillation*—poor ventricular filling, low resistance to ejection (Fig. 9,*C*). Case 4: *Severe aortic stenosis and regurgitation*—ample ventricular filling, high resistance to ejection (Fig. 9,*D*).

Existing apexcardiograms were chosen for this investigation in order to include some cases in which the diagnosis had been confirmed by autopsy. For instance, in Case 2 (Fig. 9,*B*) the aortic aperture would allow passage of a knitting needle only,

Fig. 7. *A*, Mirror image of the apexcardiogram, taken at 5L1 with 3½ times the amplification of the apexcardiogram. *B*, Apexcardiogram at 5L9. *C*, Incomplete mirror image at 5L13, taken with 9 times the amplification of the apexcardiogram. *D*, Complete mirror image at 6L15, taken with 6 times the amplification of the apexcardiogram. *C* and *D* have been taken to the left of the apex, outside of any contact with the heart. Cardiac emptying cannot account for these mirror images.

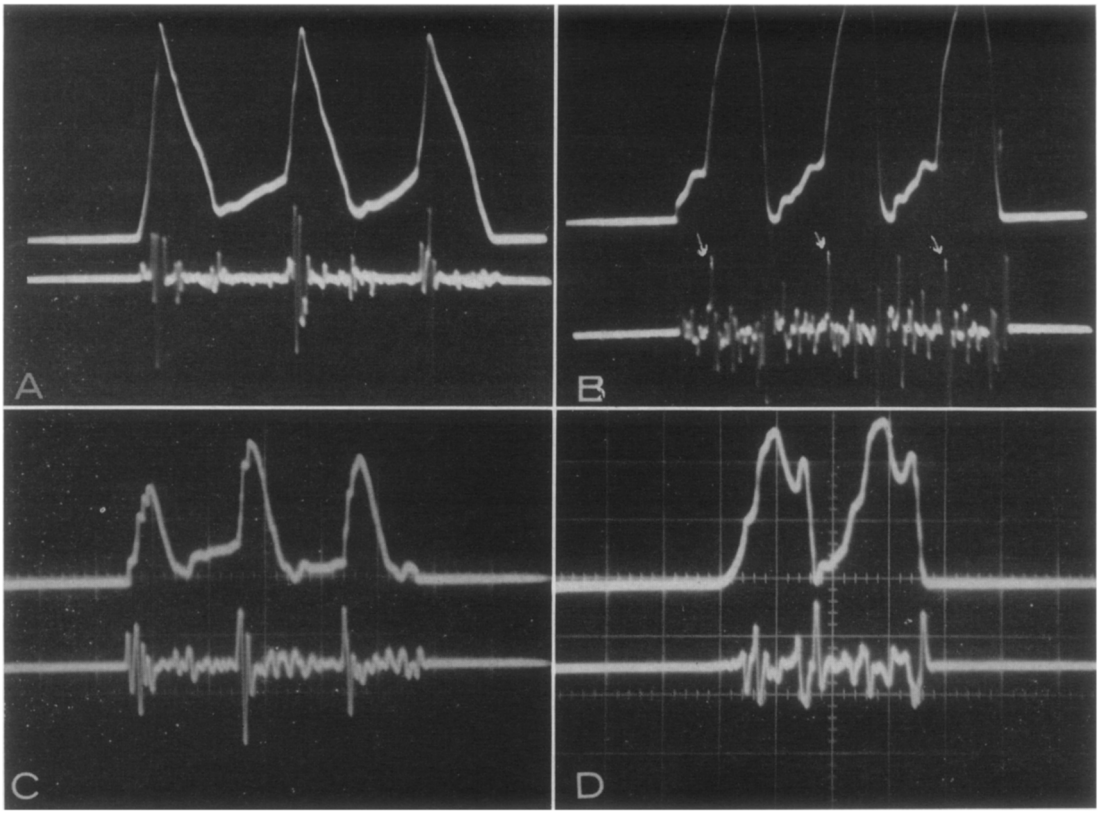

Fig. 9. Apexcardiograms and accelerograms derived from them in 4 patients in the same age group and of similar build. Constant amplification and constant relation in the amplification of displacement and acceleration. *A*, Mitral stenosis. *B*, Severe aortic stenosis. *C*, Mitral stenosis with some regurgitation and atrial fibrillation. *D*, Aortic stenosis and regurgitation.

but the mitral valve was proved to be intact. The muscle of the left ventricle had a thickness of 5 cm.

Method for obtaining accelerograms from existing apexcardiograms. An outline of the displacement tracing was cut out of cardboard, and this silhouette was mounted on a disc, which was rotated with uniform velocity by means of an electric motor. The shadow of this moving silhouette intercepted a varying quantity of light from a beam falling on a photoelectric cell. The electrical output of the photoelectric cell varies in the same manner as the shape of the original signal. These electrical variations are recorded by means of a cardioscope, and the second derivative of this signal is recorded at the same time. The shape of the apexcardiograms reproduced in this way proved to be identical to the original tracings, with the exception of the "dip," which was less pronounced. For our purpose this was of no consequence, since we were interested in the beginning of systole and not in the diastolic part of the apexcardiogram.

Results. In a comparison of the initial systolic acceleration complexes in these cases of mitral and aortic stenosis, the differences in the accelerographic amplitudes proved to be smaller than the differences in the height of the apex plateaus, especially in the beginning of systole. They certainly are not in the least comparable to the huge difference felt on palpation.* In the second case of severe aortic stenosis the

*In estimating the "force" of cardiac contraction by means of palpation of the apex beat, we evidently take into account both the measure of heaving and its duration. At the University Clinic of Utrecht it is the custom to speak of a "cleaving" apex to indicate this long duration of heaving in ventricular hypertrophy. The duration of heaving, however, is not accounted for in accelerography.

greatest accelerographic excursions are related to the sharp downstroke after the late systolic crest of the apex plateau. At the beginning of systole the excursions would seem to be partly due to the large *a* wave and the sharp transition from this *a* wave to the ventricular upstroke. The part of the initial systolic acceleration complex due to ventricular activity may be smaller yet.

The reason for the failure of relationship between the accelerographic amplitudes and cardiac "force" may be explained in the following way. In mitral stenosis the steepness of the upstroke and the immediately following decline of the apex plateau involves a greater amount of acceleration and deceleration than does the slow sustained heaving of the apex in aortic stenosis. Even with a higher plateau the accelerographic excursions in aortic stenosis may be smaller than in mitral stenosis, and even smaller, in relation to the height of the apexcardiogram, than in a normal heart. This feature is borne out by the following experiment.

Comparison of a normal apexcardiogram and its second derivative with the apexcardiogram and accelerogram in aortic stenosis

In direct recordings of apexcardiograms and the accelerograms derived from them by means of double differentiation, a fixed relation between the excursions of the apexcardiogram and of its second derivative had been obtained by using ganged potentiometers. Apexcardiograms brought up to the same dimensions by means of various degrees of amplification would give rise to accelerograms of more or less the same dimensions in normal hearts.

In Fig. 10 the upper record represents an apexcardiogram and accelerogram in a normal heart, and the lower record represents those in a case of severe aortic stenosis with some regurgitation. The apex beat in aortic stenosis proved to be the stronger one. In order to obtain an apexcardiogram of more or less the same size, twice the amplification was needed in the normal heart.

Results. This case of aortic stenosis is a special one, because the *a* wave in the apexcardiogram is not large and angular, as in most cases of aortic stenosis, nor is there a sharp transition from the descending line of the *a* to the ventricular upstroke. The displacement tracing is rounded throughout. It is one of the few cases of aortic stenosis in which the initial systolic acceleration complex may be considered as representing ventricular activity, without the disturbing aftermath of strong atrial activity overlaying its first excursions.

If the accelerographic amplitudes were related to cardiac "force," they should be greater in aortic stenosis. Since the two apexcardiograms were brought to the same dimensions, the accelerograms likewise might have the same amplitudes. However, the over-all dimensions of the accelerographic excursions in the lower record of aortic stenosis do not exceed 4 mm., whereas in the upper record of the normal heart they amount to 40 mm. They are ten times as large in the normal heart as in the hypertrophic one, with apexcardiograms brought up to the same size. Moreover, they are smaller than normal in the hypertrophic heart, as compared with the size of the apexcardiogram.

The reason for the exceedingly small accelerographic amplitudes in the hypertrophic heart can be found only in the absence of sharp transitions in the displacement tracing. The accelerographic excursions are related to the square of frequency of the excursions in displacement from which they are derived. The frequency content of the apexcardiogram proves to be of great consequence to the resulting accelerogram. Whenever the apexcardiogram is angular, the accelerographic excursions are large. When the apexcardiogram is rounded, as it tends to be in a hypertrophic heart, the accelerographic amplitudes are small as compared with the acceleration pattern derived from a normal apexcardiogram of the same size.

Even the palpable difference between the force of the apex beat from a normal heart and the apex beat in ventricular hypertrophy, and the difference between the tapping apex movement of mitral stenosis and the forceful thrust of aortic stenosis, find no counterpart in greater accelerographic excursions with the stronger apex beat, as might be expected if precordial acceleration were an expression of the "force" of contraction. The accelerographic excursions over the apex in left ven-

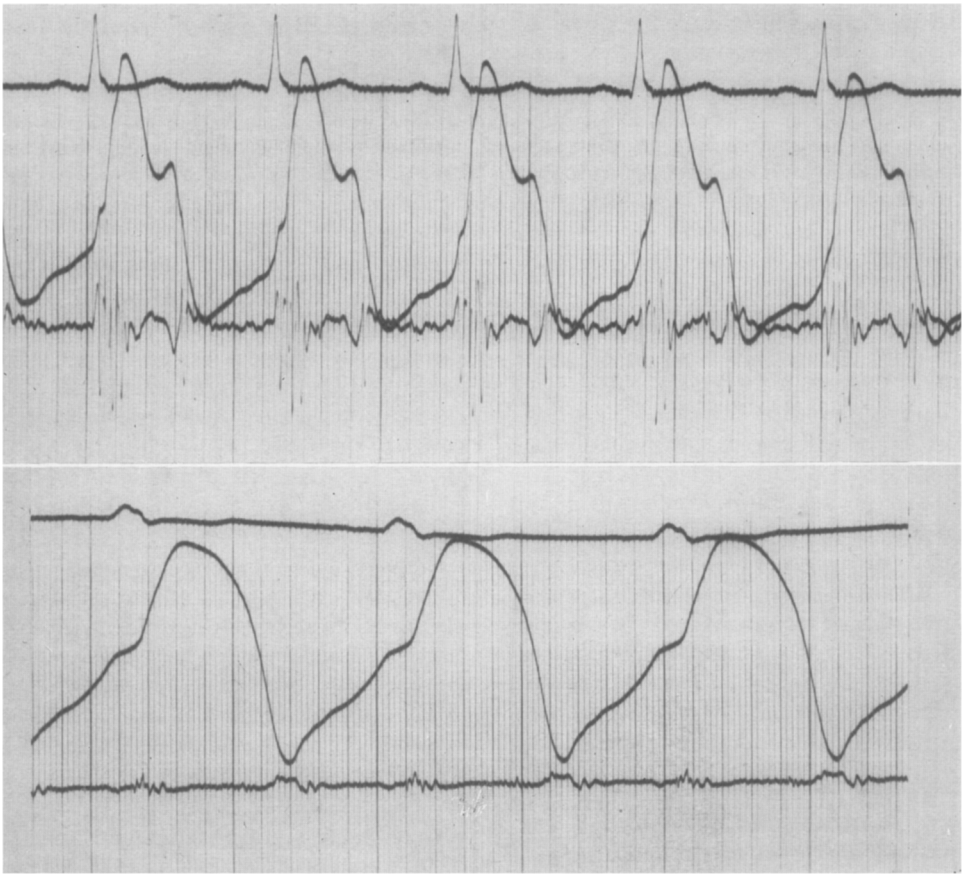

Fig. *10.* Apexcardiograms and the accelerograms derived from them, recorded with the subjects in the left recumbent position. The two apexcardiograms were brought up to more or less the same dimensions by means of double amplification of the upper tracing; the upper tracing is from a normal person, and the lower one is from a patient with severe aortic stenosis. Constant relation in the amplification of displacement and acceleration.

tricular hypertrophy even tend to be smaller than the normal ones, because of the rounding of the apexcardiogram, which is a constant feature in systolic overloading.

Conclusion

The inaccuracies of acceleration ballistocardiography have led to the suggestion of recording precordial acceleration in its stead. The estimation of stroke volume and of cardiac "force" by means of precordial accelerography, however, proves to be impossible.

In the case of myocardial damage, small amplitudes have been observed in both acceleration ballistocardiography and vibrocardiography. Small amplitudes in precordial acceleration may be caused by ventricular hypertrophy as well as by myocardial damage. The place of predilection for taking vibrocardiograms, low along the left sternal border, is by no means exempt from this influence of ventricular hypertrophy, either from left or from right cardiac origin. Further investigation will be needed in order to establish a possible difference between the acceleration pattern of ventricular hypertrophy and that of myocardial damage.

In any case, it would seem advisable to always register a displacement tracing with the accelerogram. The possibility of the simultaneous recording of them at the same place on the precordium is ensured by deriving the accelerogram from displacement by means of double differentiation.

Summary

Most transducers employed in vibrocardiography give a flat response to acceleration when actuated by means of a vibrator, or they record only the higher precordial frequencies and give tracings which closely resemble an accelerogram. For this reason, vibrocardiography has been more or less identified with precordial accelerography. Moreover, the vibrocardiographic pattern resembles the pattern of acceleration ballistocardiography. The replacement of acceleration ballistocardiography by vibrocardiography has been suggested as a means of avoiding some of the errors introduced into ballistocardiography by limb impedance and by coupling of the patient to the ballistocardiographic bed.

Acceleration ballistocardiography has been used for estimating stroke volume, and it has been related to cardiac "force." Precordial accelerography was tested with regard to the possibility of estimating both with greater accuracy. In order to do so, the precordial accelerogram was compared with the precordial displacement tracing, of which it is the second derivative.

The initial systolic acceleration complex starts with isometric contraction and continues during the first thrust of ejection. Heaving of the thoracic wall on ventricular contraction, and its deflection caused by cardiac emptying, are both involved in it. So are movements of the precordial soft tissues, which are drawn in around the apex as the tissues over the apex itself are pushed outward. This complexity of precordial movements precludes the possibility of considering precordial acceleration as a measure of the "force" of cardiac contraction.

Moreover, the accelerographic amplitudes are favored by sudden changes in the direction of precordial movement, which are more likely to occur in the case of poor ventricular filling and low resistance to ejection than in cardiac overloading. This is but another aspect of the same complexity of precordial motion, which demonstrates the impossibility of using precordial acceleration as a measure of cardiac "force."

The highest point in the normal apexcardiogram is reached at the summit of the upstroke. The height of the upstroke is conditioned by diastolic aortic pressure and not by stroke volume. In a case of arrhythmia the apex plateaus proved to be of equal height, although stroke volume certainly was larger after a long diastole. If stroke volume is not represented in displacement, it cannot be derived from its second derivative either.

Inasmuch as the purpose of acceleration ballistocardiography is the estimation of stroke volume and of cardiac "force," precordial accelerography cannot replace it.

Small amplitudes in vibrocardiography, moreover, are not a proof of myocardial damage. They may indicate ventricular hypertrophy. Along the left sternal border a strong right cardiac thrust may give rise to an abnormal vibrocardiographic pattern.* Comparison of vibrocardiographic recordings with the simultaneously recorded displacement tracings over the same precordial area would seem imperative.

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*In Fig. 4 of a recent publication by Rosa and associates,[44] this evidently is the case. Within the accelerographic pattern a remnant of the ictus plateau is seen. This feature in vibrocardiography is found only over a strong cardiac thrust, either from left or from right cardiac origin.

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