The Apexcardiogram in Myocardial Asynergy

By Francis J. Lane, M.D., John M. Carroll, M.D., Harold D. Levine, M.D., and Richard Gorlin, M.D.

SUMMARY

The apexcardiogram was analyzed in 41 patients with an altered pattern of left ventricular contraction. Four types were distinguished: type I, an early sustained systolic bulge which occurred in 12 patients, all of whom had large aneurysms and large end-diastolic volumes; type II, a bulge confined to midsystole which occurred in 13 patients, 11 of whom had some form of ventricular asynergy; type III, a late or end-systolic bulge which occurred in 12 patients, seven of whom had ventricular asynergy; type IV, a prominent spike during diastolic filling which occurred in four patients who had large zones of ventricular asynergy.

Additional Indexing Words:
Aneurysm Abnormal myocardial contraction Systolic bulge

The normal pattern of the apexcardiogram and its temporal relationship to electrical, acoustical, and mechanical cardiac events has been the subject of previous communications from various laboratories, including our own. The normal pattern is uniform, reproducible, and clearly correlated with known hemodynamic events.

Lack of general agreement about the genesis of the apexcardiogram and technical difficulties in recording it have discouraged a more general use. The low frequency vibrations, which result from relative movement in the chest wall beneath the cup applicator, are thought to result from actual movement of the heart and great vessels, from changes in volume and velocity of movement of the heart, and from changes in the consistency and curvature of the heart wall. Particular difficulty arises in attempting to evaluate systolic events from the apexcardiogram.

Abnormal pressure volume relationships and chamber geometry might be expected to induce changes in the systolic configuration of the apexcardiogram. Vakil described three types of abnormal pulsations in patients with acute myocardial infarction. By means of the kinetocardiogram, Harrison, Eddleman, and co-workers described a systolic bulge of the heart wall in acute myocardial infarction and in angina pectoris. Isometric contraction has been shown to be prolonged not only in patients with ischemic heart disease but also in normal elderly individuals.

This report concerns the apexcardiogram in patients with coronary artery disease who had disturbances of left ventricular contraction as detected by cine ventriculography. We hope to show that systolic and diastolic components of the apexcardiogram are indeed related to mechanical events in contraction of the left ventricle and that certain recorded patterns are characteristic of specific abnormalities of contraction.

Methods

Forty-one patients were analyzed in whom angiography demonstrated coronary artery disease and myocardial asynergy. Seven of these had apical systolic murmurs and angiographically demonstrated mitral insufficiency. Cases in which

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the apexcardiogram was normal have been reported earlier.\(^3\)

Technique and equipment are described in a previous paper.\(^3\) A funnel-type cup applicator with diameter of 2.5 cm was attached to the pulse wave crystal microphone\(^*\) by a 5-inch length of rubber tubing. The microphone produced an electrical signal which was directly proportional to change in pressure in the tubing. The indirect carotid pulse was recorded with a similar device. Records at cardiac catheterization were obtained with an electrocardiographic pre-amplifier.\(^+\) Linearity of response is essential in the low frequency range to obtain undistorted tracings.\(^3\) Unless otherwise specified, paper speed was 75 mm/sec, and vertical lines in the tracings represented time intervals of 0.04 sec.

The technique of recording the apexcardiogram must be developed by practice; initial attempts may result in artifactual curves.\(^1\) The left lateral decubitus position was used to bring the heart close to the chest wall and to minimize changes due to shift in position of the heart and the damping effects of distance and intervening tissue on the apex beat. The left arm was extended above the heart so that the point of maximal impulse was more easily detected. The pickup bell was placed directly over the impulse and held in position either by hand or by a 1-inch rubber band so that slight-to-moderate pressure was applied at a right angle to the chest wall. In cases of questionable origin of the impulse, a precordial electrocardiographic lead was placed to confirm the presence of a left ventricular complex (qR) at the site. Oscillographic monitoring was essential to identify satisfactory tracings. Mid-expiration is the most practical respiratory phase for recording since patients tend to perform the Valsalva maneuver in full expiration. Various artifacts have been illustrated by others. If the preceding conditions are satisfied, a uniform, reproducible record is obtained.

Simultaneous electrocardiographic and phono-cardiographic tracings were obtained in all cases. In 31 of the 41 cases, indirect carotid pulse tracings were also obtained. Simultaneous intraventricular or aortic pressure curves were recorded in selected patients.

The nature and extent of coronary atherosclerosis was depicted by selective cine coronary arteriography. Pressures were recorded by means of Statham P23D strain gauges on a Sanborn 560 system. Cine left ventriculography was performed utilizing a 6 or 9-inch image intensifier system with filming at 60 frames/sec in the right anterior oblique projection. The ventriculograms were analyzed as described elsewhere.\(^15\)

Ventricular contraction was classified as follows: (1) normal or synergic—symmetrical and simultaneous inward motion of all borders of the cavity; (2) abnormal or asynergic—localized zones of altered motion categorized as: (a) akinesis, that is, lack of motion of a portion of wall, (b) dyskinesis, that is, outward motion or systolic expansion of part of the wall, (c) asyneresis, that is, uneven inward motion of part of the wall, or (d) asynchrony, that is, loss of temporal

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\(^*\)Sanborn, model 374.

\(^+\)Sanborn, model 350-3200.

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**Figure 1**

Normal apexcardiogram (ACG). Note early peak at onset of ejection (E) with rapid fall to end-systolic shoulder (ESS) and final decline to initial diastole (O). This is followed by rapid filling wave (RFW) of diastole, atrial contraction (a), and post-atrial contraction or end diastole (Z). CP = carotid pulse; dn = dicrotic notch; 1, 2, 3, 4 = components of first sound (S₁); A = aortic and P = pulmonic components of second sound S₂; os = opening snap.
### Table 1

**Apexcardiographic and Hemodynamic Measurements**

<table>
<thead>
<tr>
<th></th>
<th>a(%)</th>
<th>LVEDP (mm Hg)</th>
<th>EDV index (ml/m²)</th>
<th>Z-E (sec)</th>
<th>Δp/Δt (isovolumic) (mm Hg/sec)</th>
<th>Δp/Δt (end-diastolic) (mm Hg/sec)</th>
<th>SER (ml/sec/m²)</th>
<th>BP s/d</th>
<th>HR</th>
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<td><strong>Type I (Diffuse Holosystolic Bulge)</strong></td>
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<td></td>
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<tr>
<td>A.F.</td>
<td>—</td>
<td>19</td>
<td>303</td>
<td>0.10</td>
<td>560</td>
<td>97</td>
<td>116/66</td>
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<td>A.M.</td>
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<td>30</td>
<td>176</td>
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<td>630</td>
<td>96</td>
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<td>664</td>
<td>73</td>
<td>133/72</td>
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</tr>
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<td>30</td>
<td>187</td>
<td>0.10</td>
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<td>54</td>
<td>110/74</td>
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<td>95</td>
<td>100/60</td>
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<td>—</td>
<td>100</td>
<td>117/100</td>
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<td>600</td>
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<td>109/52</td>
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<td>27</td>
<td>—</td>
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<td>700</td>
<td>130</td>
<td>117/66</td>
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<td>—</td>
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<td>144/82</td>
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<td>53</td>
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<td>—</td>
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<td>151/85</td>
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<td>—</td>
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<td>—</td>
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<td>750</td>
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<td>136/62</td>
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<tr>
<td>P.G.</td>
<td>—</td>
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<td>179</td>
<td>0.09</td>
<td>—</td>
<td>102</td>
<td>160/90</td>
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<td>20</td>
<td>157</td>
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<td>—</td>
<td>96</td>
<td>126/69</td>
<td>64</td>
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<td>915</td>
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<td>126/87</td>
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<td>270</td>
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<td>760</td>
<td>85</td>
<td>106 mean</td>
<td>80</td>
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<td>3</td>
<td>102</td>
<td>0.13</td>
<td>1000</td>
<td>96</td>
<td>136/76</td>
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<tr>
<td>I.P.</td>
<td>12</td>
<td>35</td>
<td>103</td>
<td>0.11</td>
<td>700</td>
<td>67</td>
<td>120/83</td>
<td>90</td>
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<td>133/71</td>
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<td>57</td>
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<tr>
<td>A.P.</td>
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<td>18</td>
<td>253</td>
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<td>88/78</td>
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<tr>
<td>E.G.</td>
<td>19</td>
<td>11</td>
<td>100</td>
<td>0.13</td>
<td>950</td>
<td>104</td>
<td>143/72</td>
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<td>—</td>
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<td>750</td>
<td>130</td>
<td>104/59</td>
<td>81</td>
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<td><strong>Type IV (Diastolic Thrust)</strong></td>
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<td>27</td>
<td>25</td>
<td>141</td>
<td>0.10</td>
<td>623</td>
<td>79</td>
<td>135/72</td>
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<td>M.K.</td>
<td>45</td>
<td>25</td>
<td>60</td>
<td>0.12</td>
<td>510</td>
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<td>124/66</td>
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<td>500</td>
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<td>86/62</td>
<td>90</td>
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Abbreviations: a(%) = ratio of amplitude of a wave to amplitude of total complex; LVEDP = left ventricular end-diastolic pressure; EDV index = left ventricular end-diastolic volume; Z-E = time between Z and E points; Δp/Δt = rate of rise of isovolumic pressure; SER = systolic ejection rate; BP s/d = blood pressure, systolic/diastolic; HR = heart rate per minute.
sequence of motion of wall. The abnormality was also categorized according to the surface of the left ventricle affected. In all cases in this series, asynergy was observed on the anterior, apical, or lateral surface of the left ventricle. It is to be emphasized that this report is concerned with abnormalities of contraction localized to those surfaces of the heart which may impinge directly upon the anterior surface of the chest wall. Thus, inferior and posterior zones of abnormality were not analyzed.

End-diastolic volume was calculated as described previously. The average rate of rise of isovolumic pressure (Δp/Δt) was calculated as described previously.

**Results**

**Normal Apexcardiogram (Fig. 1)**

The normal apexcardiogram recorded by the same technique in this laboratory has been previously described. This has been seen not only in normal subjects but also in patients with coronary artery disease who exhibit normal left ventricular contraction.

The normal systolic ejection wave of the apexcardiogram commences with an initial rapid descent from the E point during the phase of maximal ventricular ejection and is followed by a mid-systolic plateau as ejection slows. A second rapid descending wave occurs at end systole. An end-systolic shoulder (ESS) is often observed approximately 0.036 sec before the aortic second sound at the onset of the second rapid decline.

**Abnormal Apexcardiogram**

The 41 patients were divided into four groups according to patterns deviating from the normal. The data are shown in figures 2 through 8 and table 1.

**Type I Abnormality (Twelve Patients; Figs. 2 and 3)**

This consisted of a sustained positive deflection (outward bulge) which commenced early in systole. This was associated with an ill-defined E point which was slurred or followed by a highly abbreviated negative wave. The Z-E interval (an estimate of duration of isovolumic contraction) was prolonged beyond 0.10 sec in 10 of 12 patients. All 12 patients in this group had large areas of ventricular asynergy estimated angio-

![Figure 2](image-url)

*Figure 2*  
Type I abnormal apexcardiogram. Note the sustained bulge following shortly after E point. The Z-E interval is normal. BA = brachial artery pressure; LV = left ventricular pressure. A large apical aneurysm was demonstrated both by ventriculography and at necropsy.
Figure 3

Type I abnormal apexcardiogram with prolonged Z-E interval, aneurysm, and severe mitral regurgitation. Note the sustained systolic bulge contiguous with the E point. SM = systolic murmur. Other symbols as in figure 1. Large apical aneurysm and fibrosed papillary muscle were demonstrated at necropsy.

Figure 4

Type II abnormal apexcardiogram with normal Z-E interval in association with small saccular aneurysm. Symbols as in preceding figures.

Figure 5

Type II abnormal apexcardiogram with prolonged Z-E intercal in association with a large antero-apical aneurysm. Symbols as in preceding figures.

Intervals were prolonged (\( \approx 0.11 \) sec). The remaining eight had smaller zones of asynergy estimated from 10 to 25% of ventricular surface and were associated with normal Z-E intervals. End-diastolic pressure was highly variable. There was no correlation between the \( \Delta p/\Delta t \) and the Z-E interval in this group.

The two patients with type II abnormality who did not have asynergy manifested systemic hypertension of 5 to 10 years’ duration and showed left ventricular hypertrophy by electrocardiogram.
Type III Abnormality (Twelve Patients; Figs. 6 and 7)

These subjects exhibited a well-defined E point and an initial systolic negative wave, absence of a mid-systolic plateau, and accentuation of the late systolic shoulder. This pattern was frequently associated with a shortened, steep rapid-filling wave and a prominent a wave of atrial contraction at end diastole. These findings have been previously described in patients with angina pectoris. At times the stress of exercise brought out a prominent a wave (fig. 6). The type III pattern was less specific than the type I and II patterns. In addition to its occurrence in seven coronary patients with asynergy, this pattern was also seen in three patients with coronary disease who had mitral insufficiency and synergic contraction, and in two patients with primary myocardial disease who had ventricular asynergy.

The Z-E interval correlated with size of the zone of asynergy in six of seven subjects. This interval tended to be normal in the patients with mitral insufficiency and synergic contraction, but was prolonged in the two patients with primary myocardial disease and asynergy.

Type IV Abnormality (Four Patients; Fig. 8)

Abnormalities were confined to diastole in this group and consisted of two components. The first was a sharp positive wave terminating the rapid-filling wave, almost equalling (79%) the total amplitude of the apexcardiogram. Normally the rapid-filling wave is only 20 to 40% of the height of the total complex. Secondly, the a wave was an average of 28% of the height of the total complex or three times the normal ratio. These four patients had large apical aneurysms, elevated left ventricular end-diastolic pressure, and a prolonged Z-E interval.

The relative amplitude of the a wave to total complex can be considered a diastolic
abnormality, although the amplitude is subject to artifactual variation.* Good measurements could be made in 37 subjects (four noted above); 27 exhibited abnormally high amplitude ratios. Twelve have type III systolic abnormalities.

**Mitral Insufficiency (Figs. 2 and 7)**

Mitral insufficiency as such produced no distinctive changes in the apexcardiogram. Severe regurgitation however was present in five patients; all had holosystolic murmurs and prolonged Z-E intervals. Mild-to-moderate regurgitation, on the other hand, was associated with a mid-systolic murmur and normal Z-E interval.

*This is due to possible nonlinearity of the recording device in the very low frequency range and to extreme respiratory maneuvers.

**Electrocardiographic Findings**

The electrocardiogram has been helpful in the detection of ventricular aneurysm. It has been implicitly assumed that persistent elevation of the R-ST segment is a clue to ventricular aneurysm only if there are changes diagnostic of myocardial infarction in the QRS complex. Twenty-eight of the 35 patients in the present series who had ventricular aneurysms showed such QRS evidence of prior infarction. Twenty-one of these 28 showed R-ST segment elevation in the resting electrocardiogram. Although persistent R-ST segment shifts can be due to other conditions, it is noteworthy that no patient in the present series had R-ST segment elevation without also having an aneurysm. Contrariwise, the remaining seven patients with aneurysm who had QRS changes of infarction
did not exhibit persistent shift of the R-ST segment.

Of the 35 patients with aneurysm, 10 showed some type of intraventricular conduction defect (one, left bundle-branch block; one, parietal block; four, diaphragmatic peri-infarction block; and three, anterolateral peri-infarction block). There seemed to be no correlation between either a conduction defect or elevation of R-ST segment in the electrocardiogram and the appearance of the recorded apexcardiogram.

Discussion

Recording of systolic precordial vibration or displacement in both the low and high frequency ranges has been carried out by several investigators, and a relatively complete listing has been published recently.20

In the case of indirect techniques, many mechanisms have been postulated to explain the observed changes. Coulshed and Epstein4 stated that movements are produced by the changes in volume, consistency, and curvature of the heart wall, and pulsations of the great vessels. Velocity of blood flow has been related to changes in the kinetocardiogram by Harrison and associates.21 In any proposed interpretation the fundamental fact must be appreciated, that no portion of the cardiohemic system may vibrate independently.22 A single cause of any given observed effect is difficult to conceive. A realistic approach considers that any forces which set up a vibrating motion will affect the entire system of blood, heart walls, and valves. Gross shifting of the position of the heart is minimized by recording the apexcardiogram in the left lateral position in mid-expiration. In this situation the forces generating vibrations are associated with acceleration and deceleration of blood flow.23 Acceleration of blood flow is provided by the force of ventricular contraction per unit mass of blood \( F = ma \); \( a = F/m \). Deceleration of blood flow occurs in the presence of impedance exceeding the forces promoting forward flow. The heart wall and tension within elastic vascular structures contribute to total impedance. The elements of mass, inductance, capacitance, and resistance are constantly changing as blood flows in and out of the heart. The latter may well be regarded as a mass of fluid-filled muscle suspended from the great vessels in the sleeve of the pericardium.

Despite the variety of recording devices employed, observations reported in patients with coronary artery disease and angina pectoris are remarkably similar. Thus, Eddleman and co-workers10,11 observed in the kinetocardiogram (1) a forward movement following the P wave (atrial contraction wave), (2) a large inward deflection at the beginning of systolic ejection, and (3) development of a large mid-systolic outward motion (bulge). Employing the same device in patients who had old myocardial infarctions, Schweizer and associates24 observed (1) large a waves, (2) outward bulges in systole which began either during isovolumic contraction, in mid or late systole, or were occasionally pansystolic, and (3) small E waves and diminished retraction.
of the apex at onset of systolic ejection. Benchimol and Dimond emphasized the relative increase in amplitude of the a wave in the apexcardiogram as well as prolongation of the a-E interval and changes in the systolic wave in patients with angina pectoris. The late systolic outward movement, which they thought was significant, was shown also to be present in some normal individuals by Rorvik. Decrease in left ventricular border movement and either partial or complete reversal of the normal inward movement during systole was noted by Dack and associates in 50 to 60% of cases of infarction by utilizing the technique of electrokyymography. A partial reversal was more common and the outward movement of the ventricular border commenced either during isovolumic contraction, during early ejection, or even at the end of ejection. If outward movement commenced during isovolumic contraction, it always persisted into the ejection phase. The phenomenon could result in the type I apexcardiogram with prolongation of the Z-E interval which we encountered in our patients with coronary disease.

The external length of the left ventricle in the dog shortens during isovolumic contraction so that a more spherical shape results from shortening of the ventricular septum and bulging of the lateral ventricular wall. Such a pattern however does not occur in man under normal conditions, although the apex of the heart per se does shift its spatial orientation within the chest cavity. Beilin and Mounsey have described a sustained apical impulse (defined as an outward movement of the cardiac impulse which persists up to or beyond the second heart sound) in left ventricular hypertrophy and in some cases of cardiac aneurysm. Employing an impulse cardiogram, Deliannis and associates correlated the apical heart beat with biplane left ventriculograms and found that failure of the apex to retract or even a forward movement of the apex during systole was responsible for the sustained type of impulse.

Our studies are in accord with those of Mounsey's group and suggest that distortions of motion of a part of the free left ventricular wall are responsible for aberrations of the systolic morphology of the apexcardiogram. The degree of abnormality is clearly related to the amount of left ventricular wall involved in the abnormal contractile process but can be influenced by hypertrophy of the wall itself.

Irrespective of the factors which govern the patterns of the apexcardiogram, a properly recorded systolic tracing can contain important diagnostic information. The type I pattern was invariably associated with a large aneurysm or asynergic zone of myocardium, anterior, apical, or anterolateral in location. The type II pattern was less specific, but if no aneurysm was present, left ventricular hypertrophy and hypertension were. The type III pattern had little specific diagnostic value other than to indicate abnormal ventricular contraction. This was just as frequently due to exaggeration of a synergic pattern of contraction as to ventricular asynergy.

The diastolic filling thrust characteristic of the type IV apexcardiogram is probably due to rapid ballooning out of the thin fibrous ventricular wall during rapid ventricular filling. To our knowledge this phenomenon has not been described previously with ventricular aneurysm. It was illustrated by Ahuja and Gutierrez in a patient with hypertensive and atherosclerotic heart disease in left ventricular failure; it is neither stated nor denied that this patient had a ventricular aneurysm. All four patients with type IV apexcardiograms had large aneurysms at ventriculography, involving from 25 to 50% of the ventricular surface.

Physical examination is an integral step in the detection of ventricular aneurysms. Thirty-five of the 41 patients in this series showed ventricular aneurysms at cineangiography. Of the 26 patients in whom either a bifid or a diffuse apical impulse was palpated in the left lateral decubitus position, 24 had a ventricular aneurysm. In 10 of the 35 patients with aneurysms, palpation of the apical impulse was actually described as normal by
experienced observers. A more painstaking physical examination might well have detected more of the described phenomena. Deliberate repeated comparisons of palpatory findings with the elaborate precision of machine recording could well enable a conscientious observer to become much more skillful at the bedside recognition of these changes.

References

The Apexcardiogram in Myocardial Asynergy
FRANCIS J. LANE, JOHN M. CARROLL, HAROLD D. LEVINE and RICHARD GORLIN

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