Relationship Between Quantitated Precordial Movement and Left Ventricular Function

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SUMMARY

The relationship between external records of precardial movement and ventricular function has been studied in 61 patients with various cardiac disorders by comparing the records of the apical impulse to the left ventricular volume. A significant correlation between external recordings and the volume data was found in patients with normal or increased ventricular volumes: Patients with a normal record in systole usually had normal end-diastolic and left ventricular stroke volumes; those with a hyperdynamic record in systole usually had an appropriate increase in stroke volume for increased end-diastolic volume; and those with sustained records usually had an increased end-diastolic volume without an appropriate increase in stroke volume, indicating poor myocardial function. Patients with a pressure-loaded left ventricle usually had a sustained record, but this appeared to be related to factors other than volume.

These studies demonstrate that records of the apical impulse may provide useful information about left ventricular function.

Additional Indexing Words:
Apexcardiography Ventricular volumes Ejection fraction

PALPATION of the cardiac apex has enabled the clinician to distinguish three main types of left ventricular impulse: normal, hyperdynamic, and sustained. This aspect of the physical examination has considerable value in the making of a cardiac diagnosis. Various technics of recording the cardiac impulse have been devised, and qualitative descriptions of the nature of the records in different cardiac disorders have been provided.

Information on the genesis of precardial movement records is limited. Agress and Fields showed that the first derivative of the left ventricular pressure pulse (dp/dt) bears some resemblance to the external apical records obtained by different technics. The similarity between dp/dt and the first derivative of apical displacement (dF/dt) was further demonstrated by Gleichmann et al. using a technic which included quantitative measurement of dF/dt. Deliyannis et al. related left ventricular movement to radiologic, surgical, and postmortem data in a number of patients with normal, overacting, and sustained cardiac impulses.

In recent years the development of methods for measurement of left ventricular volumes by angiocardiography has permitted analysis of the functional state of the left ventricle in greater detail. Ventricular volume can be determined throughout the cardiac cycle, and the systolic ejection fraction (left ventricular stroke volume divided by end-diastolic volume) can be derived.
This account describes a number of patients whose left ventricular function has been assessed by such technics and who have had the apical precordial movement recorded by a quantitative method.11 By analysis of these data, the external recording of apical precordial movement is shown to provide information about the functional state of the left ventricle in various cardiac disorders.

Methods

Selection of Patients

Sixty-one adult patients were studied both by determination of left ventricular pressure and volume and by registration of quantitated graphic records of apical precordial movement. In order that the apical record should represent only left ventricular activity, patients were excluded in whom there were grounds to suspect right ventricular enlargement either from pressure overload (e.g., severe pulmonary hypertension) or volume overload (e.g., atrial septal defect).

These 61 patients were divided into four groups as follows:

### Table 1

**Group 1: Patients with Functional Systolic Murmurs or Mitral Stenosis: Data from Analysis of Precordial Movement and Ventricular Volume Measurements**

<table>
<thead>
<tr>
<th>Case no.</th>
<th>Age (yr)</th>
<th>Sex</th>
<th>OM height (X)</th>
<th>Configuration</th>
<th>EDV (ml/m²)</th>
<th>LVSV (ml/m²)</th>
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**Mitral stenosis**

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Abbreviations: OM height (X) = height of outward movement at apex expressed in arbitrary units X; EDV = end-diastolic volume; LVSV = left ventricular stroke volume; EF = ejection fraction; LVEDP = left ventricular end-diastolic pressure; N = normal configuration of the graphic record of outward movement at the apex in systole; H = hyperdynamic configuration of this movement; S = sustained configuration of this movement. See text for explanation.

**Group 1 (14 Patients; Table 1)**

**Functional Systolic Murmur.** Six patients (age range, 17 to 32 years) in this group had systolic murmurs. The patients were asymptomatic, and cardiac catheterization findings were normal, as was the response to exercise as judged by Donald's indices.12

**Mitral Stenosis.** Eight patients (age range, 24 to 49 years) in the group had mitral stenosis as a dominant lesion. Two of these had additional trivial aortic regurgitation demonstrated by angiology, and one, additional mitral regurgitation. Pulmonary hypertension was only moderate; mean pulmonary artery pressure at rest was less than 35 mm Hg.

**Group 2 (29 Patients; Table 2)**

**Mitral Regurgitation.** This subgroup is composed of 10 patients (age range, 19 to 54 years), in whom the dominant clinical diagnosis was mitral regurgitation which was documented at cardiac catheterization by selective left ventriculography. There was slight mitral stenosis in two patients.

**Aortic Regurgitation.** In 15 patients (age range, 17 to 65 years), the clinical diagnosis of
Table 2

**Group 2: Patients with Mitral and Aortic Regurgitation and Peripheral Arteriovenous Fistula**

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Abbreviations: Same as in table 1 plus: EESV = effective stroke volume; RV = regurgitant volume.

**Group 3 (Five Patients; Table 3)**

Primary Myocardial Disease. In five patients (age range, 20 to 57 years) the clinical diagnosis was primary myocardial disease. This diagnosis was based on the finding of cardiac enlargement on plain chest x-rays disproportionate to the clinical evidence of valvular or other abnormalities. Additional findings included history of angina pectoris, incidental mitral regurgitation, and mild coarctation. The findings at cardiac catheterization in these patients supported only trivial anatomic intracardiac abnormalities.

**Group 4 (13 Patients; Table 4)**

Aortic Valve Stenosis. This section comprised

**Precordial Movement and LV Function**
eight patients (age range, 15 to 63 years) with a clinical and cardiac catheterization diagnosis of dominant aortic valve stenosis. All but one patient also had slight aortic regurgitation. The peak systolic aortic valve gradient ranged from 10 to 120 mm Hg.

**Idiopathic Hypertrophic Subaortic Stenosis (IHSS).** Three patients (age range, 21 to 51 years) were shown to have IHSS by clinical and cardiac catheterization findings. Resting intracavity gradients of 32 and 88 mm Hg were found in two patients, and no gradient was demonstrated at rest in the remaining patient.

**Systemic Hypertension.** Two patients (ages 46 and 50 years) had long-standing systemic arterial hypertension. One patient had an additional slight mitral regurgitation clinically and at cardiac catheterization.

**Recording Technic and Analysis of Apical Precordial Movement.**

Records of precordial movement over the left ventricle were made by one of us in close temporal relationship with the catheterization studies, although not simultaneously because of technical problems. Patients were studied in the left lateral decubitus position at an angle of 45° to achieve maximum definition of the apical thrust. The location of maximal discrete outward movement was determined by palpation, and the

### Table 3

**Group 3: Patients with Primary Myocardial Disease**

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<th>LVSV (ml/m²)</th>
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**Primary myocardial disease**

**Table 4

**Group 4: Patients with Left Ventricular Outflow Obstruction**

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</table>

**Aortic valve stenosis**

| 57       | 21       | M   | 47            | H             | 58          | 44           | 0.77 | 0            | 32                          | 38            |
| 58       | 51       | M   | 85            | S             | 117         | 96           | 0.82 | 46           | 50                          | 88            |
| 59       | 40       | M   | 37            | S             | 62          | 45           | 0.73 | 0            | 0                           | 0             |

**IHSS**

| 60       | 50       | M   | 68            | S             | 91          | 64           | 0.70 | 59           | 5                           | 0             |
| 61       | 46       | M   | 28            | S             | 127         | 70           | 0.55 | 31           | 39                          | 0             |

**Systemic hypertension**

**Abbreviations:** Same as in tables 1 and 2 plus IHSS = idiopathic hypertrophic subaortic stenosis.
sensing head of a Hellige transducer was carefully applied to this point by hand. The curves representing displacement of the plastic tambour of the sensing head were monitored on the oscilloscope of a multichannel Cambridge or Sanborn recorder, prior to registration of the curves by a photographic method. An ECG and a phonocardiogram were taken, usually in two locations, to assist in timing the details of the graphic records. An external carotid artery tracing with the phonocardiogram and ECG was recorded immediately before the apex tracing. Paper speed was set at 100 mm/sec.

The graphic curve representing the apical impulse consists in normal individuals of the following:

1. A very small a wave following the P wave of the ECG, resulting from atrial systole.
2. A brisk outward movement (OM) beginning a brief (0.02 to 0.03 sec) interval after the onset of the QRS complex and representing ventricular systole; the upward limb of the outward movement has a peak at approximately the time of opening of the aortic valve (E point). This has a rapid descent on termination of systole, reaching a nadir at approximately the time of the opening of the atrioventricular valves. This is followed by a rapid filling wave and, thereafter, a slower outward movement which is terminated by the a wave of the succeeding cycle.

Our present study is concerned principally with the outward movement in systole (OM), whose graphic representation was measured by using a technic previously described. This consisted of registering on the recording apparatus the excursion resulting from a standard impulse produced by a simple calibrator applied to the tambour of the Hellige transducer. The deviation from the base line resulting from this standard impulse was designated as “X.” The patient’s record was made without altering the sensitivity control and thus the height of the outward movement of the patient’s record, or of any other feature of the tracing, could be measured in terms of X.

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*Fritz Hellige & Co., Freiburg, West Germany. This apparatus has a flat response at frequencies from 50 Hz down to 0.3 Hz, and a time constant of 1.2 sec.

†Initially an electronic calibrator was employed, but more recently this has been replaced by a mechanical calibrator providing a standard impulse of four times the magnitude of the earlier model’s impulse. Thus the various curves comprising the graphic record of precordial movement have amplitudes which are smaller multiples of the standard signal.

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The temporal relationships and the amplitude of the various segments of each record were analyzed in detail and classified in terms of the height and configuration of the outward movement. Tracings were considered to be normal, hyperdynamic, or sustained. This classification is analogous to the classification used by Deliyannis et al. although the recording technics were different. As previously shown in our laboratory, normal young adults have an outward movement of less than 40X in amplitude. Furthermore, normal patients may show a fall-off or plateau after the E point but not a sustained rise after the E point (fig. 1). A tracing which was qualitatively similar to the normal one but with an amplitude of 40X or greater was classified as hyperdynamic (fig. 2). Those with a consistent rise in systole, which abolished the E point, were considered sustained (figs. 3 and 4).

**Collection of Hemodynamic Data and Measurements Made**

Routine cardiac catheterization was carried out employing retrograde arterial catheterization and

![Figure 1](image)

*Displacement record of a patient (no. 1) with a normal end-diastolic volume and left ventricular stroke volume. Outward movement (OM) measures 24X (see text for calculation) and falls off after the E point (time of aortic valve opening) indicating a normal record. PA HF = high frequency phonocardiogram in pulmonary area; A = aortic valve closure; LVDR = left ventricular displacement record; a = wave due to atrial contraction; RFW = rapid filling wave. Time markers, 0.04 sec.*
Figure 2

Displacement record of a patient (no. 19) with a high end-diastolic and left ventricular stroke volume due to mitral regurgitation. The OM measures 120X, and falls off after the E point indicating a hyperdynamic record. PCG-MA = phonocardiogram in mitral area. SM = systolic murmur. Other abbreviations as in figure 1. Time markers, 0.1 sec.

in some cases transseptal catheterization for measurement of left ventricular pressure. The technic of obtaining left ventricular volume data has been described in detail elsewhere. Injection of contrast material was made into the left ventricle, pulmonary artery, left atrium, and aortic root in different patients, and biplane angiograms of the left ventricle were made at 6 frames/sec. These exposures were timed with reference to the ECG. Forward cardiac output was measured by duplicate Fick determinations.

The standard cardiac catheterization data were analyzed in the usual manner. The left ventricular end-diastolic pressure was measured after the a wave, if one was present, or 0.05 sec after the onset of the QRS if there was no a wave. Volume determinations included the end-diastolic volume (EDV), end-systolic volume (ESV), left ventricular stroke volume (LVSV), that is, EDV minus ESV, and ejection fraction (EF), that is, LVSV/EDV. Effective stroke volume (EffSV) was calculated from Fick determinations, and regurgitant volume (RV) was derived by subtracting the EffSV from the total LVSV. All these volume measurements were corrected for body surface area and expressed as milliliters per square meter (ml/m²).

Left ventricular volume data were compared with normal values based on the results of Kennedy et al. Their results in 16 normal adults showed EDV = 70 ml/m² ± 20, LVSV = 45 ml/m² ± 13, and EF = 0.67 ± 0.08. Using these data for normal subjects, we have calculated a regression line for the relationship in their patients between EDV and LVSV. These normal values are used in the presentation of the data in this series in figures 5 to 8. This regression line has been extended to include increased volume measurements on a speculative basis for abnormal individuals permitting separation of those patients with increased EDV into those with an "appropriate..."
Precordial Movement and LV Function

Figure 3
Displacement record of a patient (no. 45) with increased end-diastolic volume, low stroke volume, and low ejection fraction. The clinical diagnosis was cardiomyopathy with secondary mitral regurgitation. OM measures 47X and has a consistent rise during systole which obliterates the E point, indicating a sustained record. PCG-LSE = phonocardiogram at left sternal edge (medium frequency). Time markers, 0.2 sec.

Figure 4
Displacement record of a patient with aortic valve stenosis (no. 49). The end-diastolic volume is normal, and stroke volume is relatively large giving a high ejection fraction. The aortic valve gradient was 102 mm Hg. OM measures 210X with a consistent rise during systole obliterating the E point and indicating a sustained record. Note the large a wave (36X). O = O point (corresponding with mitral valve opening). Time markers, 0.04 sec.

Figure 5
Ventricular volume measurements for patients in group 1 (normal and mitral stenosis). Upper limit of normal EDV is considered to be the mean value + 2 sd derived from the data of Kennedy et al. and is indicated by the vertical line at 110 ml/m². The upper limit for normal LVSV is considered to be the mean value + 2 sd from the same source and is indicated by the horizontal line at 70 ml/m². Patients are classified according to type of apical impulse.

Figure 6
Ventricular volume measurements for patients in groups 2 and 3 (valvular regurgitation and primary myocardial disease). Limits of normal and regression line relating EDV and LVSV are same as in figure 5. Patients are classified according to type of apical impulse.
Ventricular volume pulse. Group 4 from "appropriate" relating EDV and LVSV are same as in figure 5. Patients are classified according to type of apical impulse.

Results

Group 1

Among the 14 patients who had a functional murmur or mitral stenosis, 11 had a normal apical impulse, and three had a hyperdynamic impulse (table 1; fig. 5). Two patients (nos. 4 and 9, table 1) with hyperdynamic impulses had normal volume measurements; the third patient (no. 10) had an abnormally high EDV, although an "appropriate" LVSV for this increase. Although the dominant lesion was mitral stenosis, this patient had mitral regurgitation in addition.

Group 2

There were 29 patients with a volume-loaded left ventricle due to mitral or aortic regurgitation, or both, or to peripheral arteriovenous fistula (table 2; fig. 6). Twenty-five of these had hyperdynamic apical impulses, three had sustained impulses, and one, a normal impulse. Of the 25 with hyperdynamic impulses, 18 had increased EDV with an appropriate increase in LVSV; two patients (nos. 26 and 35) had abnormally low LVSV for the elevation in EDV; while five (nos. 17, 23, 34, 41, and 43) had normal volume measurements.

The patient (no. 15) with the normal apical impulse had normal volume measurements. Of the three patients with sustained impulses, one (no. 33) had an inappropriately small LVSV for the elevation in EDV; one patient (no. 22) had normal volume measurements; and the remaining patient (no. 39) had an appropriate increase in LVSV for the elevation in EDV.

Group 3

Of the five patients with primary myocardial disease (table 3; fig. 6), four had sustained apical impulses, and one had a normal impulse. Of the patients with sustained apical impulses, none had an elevation in LVSV, although three had an increase in EDV. The patient with the normal impulse (no. 44) had a relatively low LVSV although EDV was increased.
Table 5
Statistical Analysis Showing the Ventricular Volume Measurements Found with Each of the Three Types of Precordial Movement in Groups 1 to 4

<table>
<thead>
<tr>
<th>Groups</th>
<th>EDV</th>
<th>LVSV</th>
<th>EF</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Mean ± SD</td>
<td>Mean ± SD</td>
<td></td>
</tr>
<tr>
<td>Groups 1–3</td>
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<tr>
<td>N</td>
<td>13</td>
<td>78.8 ± 23.5</td>
<td>43.4 ± 10.9</td>
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<tr>
<td>H</td>
<td>28</td>
<td>148.5 ± 57.6</td>
<td>75.8 ± 20.5</td>
</tr>
<tr>
<td>S</td>
<td>7</td>
<td>159.9 ± 63.2</td>
<td>47.9 ± 27.1</td>
</tr>
<tr>
<td>Group 4</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>S</td>
<td>8</td>
<td>88.1 ± 30.6</td>
<td>56.1 ± 19.6</td>
</tr>
<tr>
<td>H</td>
<td>4</td>
<td>92.8 ± 46.9</td>
<td>50.2 ± 4.9</td>
</tr>
</tbody>
</table>

P values

| Groups 1–3 | N versus H | P < 0.001 | P < 0.001 | NS |
|           | N versus S | P < 0.005 | NS       | P < 0.02 |
| H versus S| NS         | P < 0.02  | P < 0.025 |

Abbreviations: Same as in tables 1 to 4; N = number of patients.

Group 4
Of 13 patients (table 4; fig. 7) with a pressure-loaded left ventricle, eight had sustained apical impulses, four had hyperdynamic impulses, and one had a normal impulse. No apparent relationship was seen between the configuration of the apical impulse and the ventricular volume measurements.

To determine the physiologic implications of each of the three types of apical impulse, the volume data were analyzed separately for each kind of impulse. Since there was no apparent relationship between the apical impulse and the volume measurements in the pressure-loaded group, this analysis was confined to groups 1 to 3, and the results are shown in table 5. A statistically significant variation is seen in the volume measurements of patients with normal, hyperdynamic, and sustained impulses. In groups 1 to 3 the ventricular volume measurements showed significantly higher values for EDV and LVSV in the patients with hyperdynamic movements than in those having normal movements. The EDVs in the patients with sustained impulses were also significantly larger than those with normal impulses while their LVSVs were not. The LVSVs of the patients with hyperdynamic impulses were greater than those with sustained impulses, but their EDVs were not significantly different.

In group 4 there was no relationship between the type of apical impulse and the volume measurements.

The relationship between volume measurements and types of precordial movement in the four groups is illustrated in figures 5 to 8.

Discussion
The objective assessment of the degree of cardiac dysfunction and of its potential improvement with medical or surgical therapy is a complex problem both in terms of clinical medicine and basic physiology.17–19 Left ventricular end-diastolic pressure (LVEDP) has been widely used as an index of left ventricular function in the past, and this measurement was equated with end-diastolic volume (EDV). It has been shown,20 however, that the relationship between LVEDP and LVEDV is variable, as indeed was the case in this series of patients, reported elsewhere.21 With the introduction of angiographic methods for determining ventricular volume, the relationship between EDV and LVSV (the ejection fraction, EF) can be determined. The EF has been proposed as a useful index of myocardial function and has become a clinical aid in diagnosis. It may remain normal in the
presence of an increased EDV in aortic or mitral regurgitation, and this proportionate increase in LVSV to high EDV may be termed an “appropriate” myocardial response to volume overload. If the LVSV is not increased proportionately to an increased EDV, the myocardial response may be termed “inappropriate” and indicative of impaired myocardial function. However, the nature of this response cannot be assessed in normal individuals at increased ventricular volumes, so that a definition of the “normal” EDV-LVSV relationship at high ventricular volumes is unknown. Although this relationship can be determined for normal volumes in normal individuals and in this communication is expressed as the regression line in figures 5 to 8 based on the work of Kennedy et al., extrapolation to increased volumes is conjectural and is represented by interrupted lines in figures 5 to 8.

Apexcardiography is not as precise a technic as determination of ventricular volumes, but since it is a much simpler procedure with no discomfort to the patient, our purpose was to determine whether there was a correlation between the displacement records and the physiologic measurements.

A separation of patterns of precordial movement into normal, overacting, and sustained has been provided by Deliyanis et al. These authors, using a recording device with different characteristics from those employed in the present study, have provided valuable correlations between the patterns of precordial movement and the underlying anatomic relationships with the chambers of the heart. Our observations were directed to relating the precordial graphic records to physiologic studies to see if analysis of the apical impulse might be of predictive value with respect to physiologic derangement. The availability of a calibrating device, as described in an earlier communication from this department, permits the separation of the normal systolic outward movement from the hyperdynamic impulse which is morphologically similar, but exaggerated in amplitude. The sustained thrust with its rising slope during systole is qualitatively distinct from normal and hyperdynamic impulses.

In this series, the data suggest a relationship between the type of apical impulse and the ventricular volume measurements in normal and volume overload situations (groups 1, 2, and 3) but not in pressure overload situations (group 4) as shown in table 5. In patients with a normal apical impulse, the mean values for EDV, LVSV, and EF are similar to previously reported normal data. The majority (84%) of patients with normal apical impulses had either a functional systolic murmur or mitral stenosis. That the left ventricular impulse is normal in patients with mitral stenosis relates to the normal or near-normal left ventricular function in patients with this condition.

A hyperdynamic apical impulse is associated with increased EDV and LVSV retaining a near-normal EF which can be considered an appropriate ventricular response. This kind of impulse was found in many patients with mitral or aortic regurgitation who had good ventricular function. Another cause of a hyperdynamic apical impulse is thyrotoxicosis. Although volume measurements were not obtained, 13 thyrotoxic patients were investigated by quantitated external displacement records; 12 of these had a hyperdynamic impulse as judged by our criteria. Thyrotoxicosis is regarded as a high cardiac output state, and stroke volume has usually been elevated at rest in patients with thyrotoxicosis in the absence of heart disease or heart failure, although some patients with hyperthyroidism have normal cardiac output at rest. Increased sensitivity to catecholamines may be the reason for the hyperkinetic state and the resulting hyperdynamic apical impulse.

The sustained apical impulse is associated with increased EDV, but the LVSV is relatively small and inappropriate to the increase in EDV (low EF) by contrast to that in patients who have hyperdynamic impulses. This sustained impulse was frequently seen in patients with primary myocardial disease or
valvular regurgitation and impaired myocardial function. It is similar to the systolic bulges noted by Eddleman and Harrison in patients with ischemic heart disease. It would not be appropriate, however, to extrapolate the physiologic correlations noted here to the types of patients studied by these authors because of differences in technic in the two methods. A sustained impulse may also occur in patients with pressure-loaded ventricles, although this association is not invariable, and some of these patients have hyperdynamic or even normal impulses. The character of the impulse in pressure-loaded situations may be related to the severity of the pressure-loading, as well as the state of the myocardium.

All studies using the configuration of the apical movement must be made with an understanding of the characteristics and limitations of the method used to record the apex beat. The characteristics of the method used here have been discussed, but it must be remembered that a transducer with a time constant shorter than that of the Hellige transducer used in this study may show a more rapid fall-off during systole and exaggerated negative deflections. For this reason, one cannot transfer these observations to tracings obtained, for instance, with a Sanborn transducer, in which the time constant is too short for adequate registration of precordial movement. Indeed, the criteria for normal amplitude of outward movement and other measurements described herein are applicable only to the particular calibrator-transducer system used. The same principles and methods could be employed in another laboratory, but normal standards would have to be determined.

The technic of recording precordial movement and the classification of the record obtained require careful attention, and all recordings in this series were made by one of the authors. Although repeat recordings on different occasions sometimes yielded different X values for the amplitude of outward movement, these differences were usually small and insufficient to alter the basic classification, for instance, from hyperdynamic to normal. A problem in the classification of such tracings occurs in an outward movement which rises briskly and then has a "plateau-top" configuration. Successive complexes may appear to have a slight fall-off or a slight rise during systolic ejection. Such records were placed in the classification of normal or hyperdynamic depending on amplitude, with sustained being reserved for those with a consistently rising pattern during systole.

Quantitated records of precordial movement as described herein provide useful diagnostic information, although there are some technical problems. Such records substantiate the findings of palpation and afford a means by which this portion of the physical examination may reveal certain aspects of left ventricular function.

Acknowledgment

We would like to thank Drs. William P. Hood, Jr., and Charles E. Rackley for their help in acquiring much of the ventricular volume data reported herein, and for their stimulating discussion of the results; Mrs. Lynn Evans for her technical assistance in collecting the data on external recordings; and Mrs. Pamela McLean for typing the manuscript.

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