Echocardiographic Detection of Regional 
Myocardial Infarction

An Experimental Study

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SUMMARY
Posterior wall velocities have been advanced as a useful technic for the assessment of left ventricular function. To evaluate the echocardiographic effects of regional myocardial infarction on posterior wall motion (PWM), measurements were obtained in 22 open-chest dogs. Coronary artery ligation produced infarction of the apex in 11 dogs (group I) and the posterior wall in 11 dogs (group II). Echoes were thus received from noninfarcted myocardium in group I and from infarcted myocardium in group II. Postinfarct recordings in group I showed no significant changes in posterior wall velocities or excursion. In contrast, group II showed striking changes in the contour of PWM with a large initial posterior displacement (aneurysmal bulging) during isometric contraction, followed by a slow anterior movement during ventricular ejection and then a rapid anterior recoil motion during isometric relaxation. Mean posterior wall velocity fell from $31 \pm 3$ mm/sec preinfarct to $13 \pm 4$ postinfarct ($P < 0.01$); from $33 \pm 4$ to $21 \pm 5$ mm/sec during atrial pacing pre- and postinfarction ($P < 0.05$); from $30 \pm 3$ to $18 \pm 3$ mm/sec during volume loading with dextran $20 \text{ ml/kg} (P < 0.01)$; and from $32 \pm 4$ to $5 \pm 2$ mm/sec during aortic constriction ($P < 0.01$). Corresponding reductions in posterior wall systolic excursion were observed.

Simultaneous hemodynamic measurements revealed moderate depression of ventricular performance in group II, but there was no significant correlation between the changes in hemodynamic parameters and echocardiographic velocities following infarction.

We conclude that the marked changes in PWM following posterior infarction were due to echocardiographic detection of dyskinesis in the infarcted area rather than to generalized changes in ventricular function. Posterior wall velocities are not a reliable index of left ventricular performance when localized dyskinesis, as indicated by abnormal PWM, is present.

Additional Indexing Words:
Ultrasound Dyskinesis Ventricular aneurysm Asynergy Posterior wall velocity

Diagnostic ultrasound has been utilized in the measurement of ventricular volumes and wall thickness. More recently, attention has been directed specifically toward the left ventricular posterior wall echo. The velocity of anterior displacement of this echo during systole has been related to the rate of decrease in ventricular diameter and to the velocity of circumferential fiber shortening as determined by angiography. Echocardiographic measurements of posterior wall velocity have been advanced as a useful noninvasive clinical technic for the assessment of left ventricular function in myocardial infarction. Myocardial infarction may produce localized abnormalities of ventricular contraction as well as generalized alteration of ventricular performance. We considered the possibility that changes in the motion of one area of infarcted myocardium may not correlate well with changes in overall ventricular performance. The aims of this study were two: first, to see if localized areas of infarction would produce specific alterations in the normal left ventricular posterior wall echocardiogram, and second, to see whether such alterations, if present, affected the use of posterior wall velocity as an index of ventricular performance.
Methods

Twenty-two adult mongrel dogs, weighing from 17 to 28 kg, were anesthetized with sodium pentobarbital, 25 mg/kg i.v. Respiration were maintained by a Harvard respirator via a cuffed endotracheal tube. A thoracotomy was performed via a midsternal incision, the pericardium incised, and the heart exposed and lifted slightly in a pericardial sling. Left ventricular and subclavian artery pressures were recorded via fluid-filled polyethylene catheters. Left ventricular dp/dt was continuously determined from the left ventricular pressure trace by means of an R-C differentiating circuit. Left ventricular ejection time was measured from the subclavian artery pressure tracing. An isometric cardiac force transducer of the type described by Feigl et al. was affixed to the lateral wall of the left ventricle, and a myocardial tension curve recorded continuously. The peak systolic myocardial tension was measured from the curve; the thickness of the myocardium under the pins of the gauge was measured after the animal was sacrificed, and this times the width of the pins gave the cross-sectional area needed to calculate isometric tensile stress, in g/cm².

A commercially available ultrasound device (Smith-Kline Ekoline 20) was used to obtain the left ventricular posterior wall echocardiograms. The transducer was placed lightly on the anterior surface of the heart, directed to record the characteristic signal from the posterior wall, and fixed in place by a stationary arm. A continuous echocardiogram during several cardiac cycles was displayed on a time-motion representation and recorded either on Polaroid photographs of the oscilloscope screen, or by display of the ultrasound signals on an Electronics for Medicine DR-12 recording device. In the latter instance, pressures and echocardiograms were recorded simultaneously to permit accurate timing. Identification of the echo from the posterior wall endocardium-blood interface was verified by rapid injection of 5 cc of normal saline solution through the left ventricular catheter while an echocardiogram was being obtained. Cavitation at the catheter tip produces microbubbles from which echoes are recorded; the resulting “cloud” of echoes outlines the borders of the ventricular cavity (fig. 1). Because of the change in contour of the posterior wall echo after posterior infarction, this procedure was repeated following infarction and before the termination of each experiment.

Following instrumentation and stabilization, hemodynamic and echocardiographic measurements were made in a resting state. In an attempt to assess the correlation between hemodynamic and echocardiographic measurements in a variety of physiologic states, the response to changes in heart rate, preload, and afterload were measured. Heart rate was increased to 125% of control by pacing the right atrium. In 11 of the animals mechanical alternans developed during atrial pacing; no attempt was made to derive velocity and

![Figure 1](http://circ.ahajournals.org/)

Echocardiographic recording of the posterior heart wall. A rapid injection of saline through a catheter in the left ventricle produces a “cloud” of bubbles (arrow), outlining the borders of the ventricular cavity.
excursion measurements from echocardiographic recordings obtained during periods of alternans. The animals then underwent volume loading with intravenous dextran (molecular weight 40,000), 20 ml/kg, infused during a 2-min period. After recording an equal volume of whole blood was removed by phlebotomy; this blood was heparinized and kept warm for later reinfusion. An umbilical tape previously passed around the ascending aorta was then tightened to produce a rise in left ventricular systolic pressure to approximately 125% of control level, and further recordings were made. Following recording, the tape ligature was released.

The animals were then divided into two groups of 11 dogs each. In group I an apical myocardial infarction was created by ligating the branches of the left anterior descending and circumflex coronary arteries which supplied the cardiac apex. In group II the posterior descending coronary artery and other posterior branches of the circumflex coronary artery were ligated producing a true posterior myocardial infarction. As seen in figure 2, creation of the infarctions in this manner resulted in the posterior wall ultrasound echoes being received from noninfarcted myocardium in the apical infarction dogs (group I), and from infarcted myocardium in the posterior infarction dogs (group II).

This was verified at the conclusion of each experiment by passing a metal probe through the heart along the path of the ultrasound beam and observing where the probe intersected the posterior wall in relation to the area of infarction.

In neither group of animals did the infarction include the area to which the isometric tensile stress gauge was affixed. Following infarction the animals were allowed to stabilize for 30 min, when postinfarction hemodynamic and echocardiographic recordings were made. The stresses of atrial pacing, volume loading, and aortic constriction were then reapplied similar to the preinfarction studies, except that the previously withdrawn warm blood was now reinfused instead of dextran. Hemodynamic and echocardiographic recordings were repeated with each intervention. Following this the animals were sacrificed. The approximate percentage weight of the total left ventricle (including septum) which had been infarcted was determined by injecting Evans-blue dye into both coronary ostia of the excised, intact heart, and dissecting out the nonperfused (unstained by dye) portion of the left ventricle.

In both apex and posterior infarction groups the infarcted myocardium averaged 30% of the total left ventricular weight.

Figure 2
Location of infarctions with regard to the ultrasound beam. In apex infarction dogs (lower figure) the beam is reflected from noninfarcted myocardium, while in posterior infarction dogs it is reflected from infarcted myocardium.
Posterior wall velocities and posterior wall excursion were measured according to the conventions introduced by Krauz and Kennedy. Point B, the posterior wall position at end-diastole is approximately simultaneous with the R wave of the ECG. During isometric contraction the wall moves slightly posteriorly, from B to C. Ventricular ejection begins at C and the wall moves anteriorly, reaching its maximal forward displacement at the end of systolic ejection, D, which occurs after the T wave of the ECG. The period between D and E indicates isometric relaxation, and the wall returns to its original position between E and F during diastole. Two echocardiographic velocity measurements have been derived from this normal pattern: the mean posterior wall velocity (PWV$_m$), which is the slope of the line drawn from the onset (C) to the end (D) of left ventricular ejection, in mm/sec; and the initial or maximal posterior wall velocity (PWVi), determined by the slope of the initial, steep portion of the C-D curve. Posterior wall excursion (PWE) is the amplitude of posterior wall motion as measured by the vertical distance between C and D, in mm. The amplitude of the wall movement during isometric contraction (B-C interval) was also measured.

Statistical analysis was performed in the following manner: pre- and postinfarction variables for each condition (resting values pre- and postinfarction, atrial pacing pre- and postinfarction, volume loading pre- and postinfarction and aortic constrictor pre- and postinfarction) were compared by using a paired t test. Simple product-moment correlations of changes between hemodynamic and echocardiographic parameters during the various interventions were calculated.

**Results**

The hemodynamic and echocardiographic data are summarized in tables 1 and 2.

**Group I: Apex Infarction**

**Hemodynamic Data.** The only statistically significant change in the resting comparison (pre- vs postinfarction) was a 9% prolongation in left ventricular ejection time. No significant changes were seen comparing pre- vs postinfarction parameters during atrial pacing and volume loading. With

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**Figure 3**

Normal posterior wall echocardiogram. Ao = aortic; IV = interventricular; LV = left ventricle; PWVi = initial posterior wall velocity; PWVm = mean posterior wall velocity; PWE = posterior wall excursion. For B, C, D, E, F see text.
### Table 1

**Hemodynamic and Echocardiographic Measurements: Group I (Apex Infarction)**

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Preinfarction</th>
<th>Postinfarction</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Resting (mean ± SEM)</td>
<td>Atrial pacing (mean ± SEM)</td>
</tr>
<tr>
<td>Heart rate (beats/min)</td>
<td>143 ± 6</td>
<td>186 ± 9</td>
</tr>
<tr>
<td>Arterial systolic pressure (mm Hg)</td>
<td>143 ± 6</td>
<td>163 ± 11</td>
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<tr>
<td>Arterial diastolic pressure (mm Hg)</td>
<td>111 ± 7</td>
<td>134 ± 12</td>
</tr>
<tr>
<td>LV systolic pressure (mm Hg)</td>
<td>133 ± 6</td>
<td>134 ± 8</td>
</tr>
<tr>
<td>LV end-diastolic pressure (mm Hg)</td>
<td>10 ± 2</td>
<td>5 ± 3</td>
</tr>
<tr>
<td>LV dp/dt (mm Hg/sec)</td>
<td>2825 ± 288</td>
<td>3243 ± 472</td>
</tr>
<tr>
<td>LVET (msec)</td>
<td>138 ± 6</td>
<td>135 ± 15</td>
</tr>
<tr>
<td>Isometric tensile stress (gm/cm²)</td>
<td>580 ± 46</td>
<td>645 ± 122</td>
</tr>
<tr>
<td>PWV₁ (mm/sec)</td>
<td>56 ± 6</td>
<td>45 ± 8</td>
</tr>
<tr>
<td>PWVₑ (mm/sec)</td>
<td>32 ± 3</td>
<td>28 ± 2</td>
</tr>
<tr>
<td>PWE (mm)</td>
<td>6 ± 0</td>
<td>4 ± 1</td>
</tr>
<tr>
<td>B-C amplitude (mm)</td>
<td>1 ± 0</td>
<td>1 ± 0</td>
</tr>
</tbody>
</table>

Abbreviations: LV = left ventricle; LVET = left ventricular ejection time; PWV₁ = initial posterior wall velocity; PWVₑ = mean posterior wall velocity; PWE = posterior wall excursion; B-C = see text.

* = P < 0.01.
† = P < 0.05.

### Table 2

**Hemodynamic and Echocardiographic Measurements: Group II (Posterior Infarction)**

<table>
<thead>
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<th>Parameter</th>
<th>Preinfarction</th>
<th>Postinfarction</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Resting (mean ± SEM)</td>
<td>Atrial pacing (mean ± SEM)</td>
</tr>
<tr>
<td>Heart rate (beats/min)</td>
<td>155 ± 5</td>
<td>201 ± 4</td>
</tr>
<tr>
<td>Arterial systolic pressure (mm Hg)</td>
<td>146 ± 7</td>
<td>154 ± 10</td>
</tr>
<tr>
<td>Arterial diastolic pressure (mm Hg)</td>
<td>116 ± 6</td>
<td>122 ± 9</td>
</tr>
<tr>
<td>LV systolic pressure (mm Hg)</td>
<td>145 ± 6</td>
<td>150 ± 10</td>
</tr>
<tr>
<td>LV end-diastolic pressure (mm Hg)</td>
<td>10 ± 1</td>
<td>14 ± 4</td>
</tr>
<tr>
<td>LV dp/dt (mm Hg/sec)</td>
<td>2289 ± 170</td>
<td>2473 ± 286</td>
</tr>
<tr>
<td>LVET (msec)</td>
<td>131 ± 6</td>
<td>120 ± 6</td>
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<tr>
<td>Isometric tensile stress (gm/cm²)</td>
<td>652 ± 90</td>
<td>818 ± 157</td>
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<tr>
<td>PWV₁ (mm/sec)</td>
<td>60 ± 12</td>
<td>55 ± 9</td>
</tr>
<tr>
<td>PWVₑ (mm/sec)</td>
<td>31 ± 3</td>
<td>33 ± 4</td>
</tr>
<tr>
<td>PWE (mm)</td>
<td>5 ± 0</td>
<td>4 ± 0</td>
</tr>
<tr>
<td>B-C amplitude (mm)</td>
<td>2 ± 0</td>
<td>2 ± 0</td>
</tr>
</tbody>
</table>

Abbreviations: Same as in table 1.

* = P < 0.01.
† = P < 0.05.
aortic constriction postinfarction isometric tensile stress declined 30%.

Echoangiographic Data. There were no significant postinfarction changes in any of the echocardiographic parameters in the resting state and with the interventions of atrial pacing, volume loading, and aortic constriction. The contour of the posterior wall motion was unaffected.

**Group II: Posterior Infarction**

**Hemodynamic Data.** In the resting state, the only significant change was that the left ventricular end-diastolic pressure rose 40% postinfarction. With volume loading left ventricular end-diastolic pressure rose 30% and left ventricular ejection time increased 12%. During aortic constriction declines in systolic (19%) and diastolic (20%) arterial pressures occurred. The left ventricular dp/dt fell 16%. Isometric tensile stress fell 37% during aortic constriction.

Echoangiographic Data. A striking change in the contour of the echocardiographic posterior wall motion was seen in all the posterior infarction dogs (echoes received from infarcted myocardium). This change is illustrated in figure 4. During isometric contraction the posterior displacement of the infarcted posterior heart wall (B–C) is much greater than normal. Systolic ejection begins at C, but the normal early systolic rapid anterior motion has been replaced by a slow forward movement which persists until systolic ejection ends at point D. During isometric relaxation (D–E) the wall moves rapidly forward, and then remains displaced in an abnormal anterior position until the next systole begins. Posterior wall velocities in this group of dogs were measured as indicated in figure 4. Mean posterior wall velocity was taken as the slope of the line from C to D. PWE was again taken as the vertical distance between C and D, and the amplitude of the excursion during isometric contractions (B–C interval) was also measured. Note that the motion of the infarcted posterior wall during ventricular ejection is essentially constant; the rapid initial posterior wall velocity (PWV₁) seen in noninfarcted myocardium is no longer present.

![Figure 4](http://circ.ahajournals.org/)

*Echocardiographic changes produced by posterior infarction. Abbreviations as in figure 3.*

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Measurements made from the ultrasound recordings reflected this marked contour change. Mean posterior wall velocity fell 58% from resting preinfarction to postinfarction states. Pre- and postinfarction comparisons showed a 36% decline during atrial pacing, a 40% fall during volume loading, and a 84% decline during aortic constriction (fig. 5). Posterior wall systolic excursion (PWE) was similarly reduced, falling 60% in the resting state, 50% during atrial pacing, 67% during volume loading, and 80% during aortic constriction. B–C amplitude (excursion during isometric contraction) showed a mean increase of 100% comparing resting pre- and postinfarction states, 150% during atrial pacing, 50% during volume loading, and 100% during aortic constriction (fig. 6). All these changes were statistically significant.

Using simple linear regression equations, no significant correlation, in either group of animals, could be demonstrated between changes in echocardiographic velocities and changes in heart rate, left ventricular end-diastolic pressure, and left ventricular isometric tension.

Discussion

The change in echocardiographic contour of the infarcted posterior wall is striking, and was observed in every animal in the posterior infarction group. During isovolumetric contraction the shape of the normal left ventricle changes, resulting in a shortening of the longitudinal axis. There is a slight increase in the minor diameter as the chamber becomes more spherical. Thus the normal posterior wall echo shows a relatively small posterior movement during isovolumetric contraction (B–C interval, fig. 3). The amplitude of this motion is significantly increased in the posterior infarction animals as the infarcted myocardium displays an aneurysmal bulging during the period of rapidly rising ventricular pressure (isometric contraction). Similarly, when the intraventricular pressure falls rapidly during isometric relaxation the stretched and bulging infarcted myocardium then recoils from its abnormally posterior location and moves rapidly back to its original position, resulting in the rapid anterior displacement seen during this period (D–E interval). Of note is the observation that during ventricular ejection, when intraventricular pressure is high but peak rate of force development past, the echocardiogram reveals the infarcted ventricular wall to be moving anteriorly (C–D interval) albeit the velocity of this motion is much reduced when compared to normal. This suggests

**Figure 5**

Mean posterior wall velocity in posterior infarction dogs (Group II) before and after myocardial infarction.

**Figure 6**

B–C amplitude (excursion during isometric contraction) in posterior infarction dogs (Group II) before and after myocardial infarction.
that during this early postinfarction phase enough contractile muscle remains to generate local tension exceeding intracavitary pressure; the minor ventricular axis is then shortened to some degree. When intracavitary pressure was increased by aortic constriction, the infarcted ventricular wall moved posteriorly during the C–D period in four of the 11 group II animals, resulting in a negative value for PWVₘ in these animals. In this subgroup of animals it would seem that the contractile muscle was more severely damaged by ischemia so as to be unable to generate enough local tension to exceed the higher intracavitary pressure produced by aortic constriction, resulting in a truly “paradoxic” posterior motion during ventricular ejection. In the animals with apical infarction and undamaged posterior myocardium this response to aortic constriction was not seen.

Alterations in motion of infarcted myocardium have long been recognized, in both clinical and experimental observation. In 1935 Tennant and Wiggers observed the changes produced by coronary ligation in dogs by placing a mechanical recording arm on the anterior aspect of the left or right ventricle. The motion recorded thus portrays deflection of the epicardial, rather than endocardial surface, but nevertheless reveals the same characteristic alteration of motion of infarcted myocardium that was seen using the echocardiographic technic. Tennant and Wiggers also showed, as the present study suggests, that noninfarcted myocardium displays no alterations in motion after coronary ligation. Similar results have been obtained using slow-motion movies and radarkymography in both experimental animals and patients.

Because of the previously noted correlation of ultrasound-measured posterior wall velocity with angiographically measured circumferential fiber shortening velocity it has been proposed that this noninvasive method would provide a useful and simple way of assessing left ventricular function. In support of this it has been shown that posterior wall velocity increases with exercise, and atrial pacing and is depressed by myocardial infarction in man. However, in the latter reports only one of a total of 29 patients was thought to have a true posterior infarction. In our study the failure of changes in the echocardiographic posterior wall velocities and hemodynamic parameters to show any significant correlation in the animals with posterior infarction supports the concept that the ultrasound signal was registering a local abnormality of wall motion rather than a generalized alteration in ventricular function. In addition, it suggests that posterior wall velocity is not a reliable parameter of left ventricular function when infarction or localized dyskinesia is present.

Since the echo signal was being received from noninfarcted myocardium in group I animals any echocardiographic velocity and excursion changes would presumably reflect changes in generalized ventricular performance in this group. However, despite a substantial infarction these animals demonstrated only modest depression of ventricular function as seen in the hemodynamic parameters. Changes in the echocardiographic posterior wall velocity measurements failed to correlate with the hemodynamic parameters. It is possible that the ultrasound technic is relatively insensitive to small changes in ventricular performance, but more work needs to be done in this regard before definite conclusions can be reached.

Both groups of animals sustained infarctions of approximately equal size. The reason the dogs in group II manifested significantly more hemodynamic depression is probably to be found in the anatomic structure and behavior of the left ventricle during ejection: contraction of the circumferential muscle bundles accounts for most of the power and volume of ejection, since the volume contained in a cylinder decreases with the square of the radius. Shortening of the longitudinal axis (more affected by apex infarction) is less prominent and less effective in ejecting blood because the volume displacement is directly proportional to the change in length.

The specific motion contour of acutely infarcted myocardium may have clinical diagnostic value in the occasionally encountered true posterior infarction in man. Probably more useful is the demonstration that myocardial dyskinesia can be registered echocardiographically, and this should be suspected if a technically satisfactory ultrasound recording reveals abnormal contour of posterior wall motion. When such abnormal contour is seen, posterior wall velocities must be considered an unreliable index of overall ventricular performance. Their reliability and sensitivity as a quantitative index of ventricular function in the absence of posterior wall dyskinesia remains to be established.

Acknowledgment

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