Epicardial Mapping of Segmental Myocardial Function: An Echocardiographic Method Applicable in Man

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SUMMARY A technique for epicardial mapping of segmental myocardial function at multiple sites over both right and left ventricles was developed using a high-resolution, 7.5-MHz, short-focus, miniaturized, M-mode echocardiographic transducer worn on the fingertip. Myocardial function was determined from the extent and time course of systolic thickening and diastolic thinning at each site mapped. The technique was characterized in an open-chest canine model of myocardial ischemia. Ischemia was induced by transient or permanent coronary occlusion in 17 dogs. Acute occlusions produced reduced segmental thickening within 10–15 seconds and, often, overt systolic thinning of ischemic myocardium. Rhodamine fluorescence perfusion maps were compared with echocardiographic maps in nine dogs. Segmental thickening was reduced in perfused segments adjacent to, but not involved by, ischemia, as well as ischemic segments. Reproducibility appeared satisfactory for quantitative analysis of grouped data on multiple segments, and qualitative analysis in individual segments. Initial human studies performed during coronary bypass surgery in 11 subjects showed echocardiographic abnormalities in the six patients with ventriculographic abnormalities and in four with normal ventriculograms. Transmural infarctions were akinetic, showing no change in thickness throughout the cardiac cycle. Hypokinetic segments distal to high-grade coronary stenosis were common, although most segments distal to stenosis contracted normally. Reversal of segmental contraction abnormalities by coronary bypass grafting was shown in three subjects, while worsening of function was seen in previously normal segments in two and in a previously normal segment in one subject. Epicardial echocardiographic mapping is a practical method for intraoperative assessment of myocardial function during coronary surgery in man that may enhance our understanding of the pathophysiology of coronary disease and the effects of coronary surgery.

THE ORIGINAL DESCRIPTION of the effect of coronary occlusion on segmental myocardial contraction by Tannent and Wiggers1 was based on strain-gauge recordings. Since then, improved methods have further characterized segmental myocardial dysfunction due to experimental acute and chronic myocardial ischemia. These methods include mercury-in-silastic segment-length gauges and sonomicrometry recordings of segment length and myocardial thickness.2–5 However, it has not been possible to examine the contractile effects of the ischemic process in man with the refined techniques used in experimental models. Furthermore, even in experimental ischemia, the ability to assess contractile function of multiple myocardial segments has been limited. The present study was designed to develop a method for assessing segmental myocardial function that could be applied intraoperatively to man. Our goals were to achieve the same spatial resolution obtained in experimental models and to make possible extensive topographic sampling of segmental myocardial function of the entire right and left ventricles. Therefore, we developed a high-resolution epicardial echocardiographic mapping method in an open-chest animal model and applied it during coronary bypass surgery in man.

Methods

Echocardiographic Instrumentation

Recordings were made with a miniaturized (10 × 11 × 13 mm) 7.5-MHz transducer (KB-Aerotech) with a 1–2-cm focal zone and calculated axial resolution of 0.2 mm. The transducer was small enough to be worn on an index finger (fig. 1) in a sterile fingercot, the inner surface of which was wetted with a few drops of normal saline. Transducers for human use were sterilized in cold ethylene oxide. The transducer was interfaced to a wide-band M-mode echocardiographic imaging system (Irex System II) equipped with analog channels and a strip-chart recorder. Recordings were made at 50 and 100 mm/sec paper speed with simultaneous electrocardiographic, arterial pressure and, when appropriate, pulmonary artery pressure recordings.

Epicardial Map

Recording sites were numbered according to an epicardial map used at our institution for human electrophysiologic mapping (fig. 2). The interventricular septum was imaged through the right ventricular free wall by appropriate gain adjustment and septal sites were identified by the overlying right ventricular site number.

Recording Technique

The transducer was applied lightly to the epicardium at each mapping site and held in place for 10–15 seconds to obtain a satisfactory record. Receiver gain and reject were adjusted at each site. When epicardial contact was not satisfactory, slight rotational motion of

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the transducer produced a satisfactory image in most instances.

Acute Canine Myocardial Ischemia

Studies were performed in 17 conditioned mongrel dogs that weighed 15–25 kg. They were anesthetized with sodium pentobarbital, 30 mg/kg i.v., supplemented by gallamine triethiodide, 0.5 mg/kg i.v., and Innovar, 0.025 ml/kg i.v., as needed. Ventilation was maintained through an endotracheal tube with a mechanical ventilator (Harvard Apparatus Co.). The right external jugular vein and common carotid artery were cannulated to monitor central venous and systemic arterial pressures, respectively. A left anterolateral thoracotomy was made to expose the heart, the pericardium was opened and the heart was suspended in a pericardial sling. Episodes of ischemia were produced by transient (2–4-minute) or permanent occlusion of the distal left anterior descending or a large diagonal branch in each dog, using a suture snare or ligature. Recordings were made at multiple sites before, during and after release of occlusion. In nine dogs, the relationship of perfusion to myocardial performance was assessed. For these studies, each epicardial echo recording site was identified by a 6–0 superficial suture. Recordings were taken before, during and after permanent coronary occlusion. Ten to 15 minutes after occlusion, Rhodamine B (Sigma Chemical Co.), a fluorescent dye that distributes in the pattern of perfusion, was injected intravenously and allowed to circulate for 1–2 minutes. The dog was then killed and the heart rapidly excised. The left ventricle was opened and endocardial sites immediately opposite each marked epicardial site were identified with marking sutures. Fluorescent photographs were made of both endocardial and epicardial surfaces of each heart under a Woods ultraviolet lamp. Sites that fluoresced on both surfaces were designated as normally perfused; those that did not were regarded as ischemic. Fluorescent sites immediately adjacent to ischemic sites were identified as “border” sites.

In seven dogs, epicardial echo recordings were compared to Walton-Brodie strain-gauge recordings from the same normally perfused and ischemic segments before, during and after transient occlusion. A preliminary 20-second coronary occlusion identified the ischemic myocardium to which the strain gauge was sutured in each dog. The transducer was placed next to the strain gauge and care was taken to avoid alteration of the strain-gauge recording by application of the transducer.

Reproducibility of end-diastolic thickness and percent thickening was determined by comparison of paired recordings (n = 24) obtained at the same epicardial site on separate transducer applications at least 3–5 minutes apart in the basal state.

Intraoperative Recordings

Human intraoperative echo mapping was performed during coronary bypass surgery in 11 subjects. Each subject gave informed consent. Mapping was performed after right atrial and aortic cannulation were complete, but before cardiopulmonary bypass was begun. In five subjects, mapping was repeated after coronary bypass grafting and termination of cardiopulmonary bypass. Transducer application and site numbering were similar to those used in the dog. The transducer was applied by a cardiac surgeon experienced in electrophysiologic epicardial mapping. Gentle displacement or elevation of the beating heart permitted imaging of the posterior and inferior surfaces of

**Figure 1.** A miniature 7.5-MHz echo transducer worn under a sterile fingercot on a gloved index finger.

**Figure 2.** Numerically coded topographic maps of the anterior, lateral and posterior surfaces of the heart, used to describe site of placement of echocardiographic mapping transducer. Sites 1–25 lie over the right ventricle but can also be used to image different segments of the interventricular septum, through the right ventricle, by gain adjustment. Sites 26–54 lie over the left ventricular free wall.
both ventricles in most subjects. In some subjects, however, such displacement or elevation could not be performed without immediate onset of hypotension, and thus, no attempt was made to map these sites. If a satisfactory echo image was not obtained within 15 seconds at a given site, it was abandoned. Mapping was thus completed in 15 minutes or less in each instance.

Data Analysis

Canine data were analyzed quantitatively by computer for each site. For computer analysis, the endocardial boundary was traced for three to five cardiac cycles at each site by one observer and reviewed by a second. Disagreements were resolved by consensus review. The traced echoes were then calibrated and digitized at 10-msec intervals on a Hewlett-Packard 9825A desktop computer system equipped with a high-resolution digitizer, printer, plotter, and floppy disc memory. Data were stored on disc, smoothed with a moving three-point mean smoothing routine and printed along with the first derivative, dThickness/dt, end-diastolic and end-systolic thickness and percent thickening.

No quantitative analysis of human data was attempted for this report, since uncontrolled hemodynamic, anesthetic and premedication variables could not be overcome by subgrouping in the small number of patients studied, and no normal controls were available. Instead, the myocardial thickening pattern in each segment was classified by two observers qualitatively as normal, mild-to-moderate hypokinesis and severe hypokinesis-akinesis based on the amplitude and time course of thickening. Discrepancies were resolved by consensus review. No differences of more than one class occurred.

Statistical Analyses

Canine data were analyzed to define the normal relationship between site thickness and thickening by linear regression analysis. Sites were also grouped in five tiers from base to apex, and end-diastolic thickness and percent thickening values were compared between tiers by analysis of variance.8 In rhodamine studies, percent thickening in normal, border and ischemic sites was compared by analysis of variance.

Results

Normal canine echocardiograms from anterior left ventricular site 32 and right ventricular site 23 are shown in figure 3. Epicardial motion was absent because the transducer was directly applied to the epicardium and moved with the heart.

Endocardial motion relative to the epicardium was shown distinctly and myocardial systolic thickening and diastolic thinning were well demonstrated. As expected, the right ventricular wall was much thinner than the left, but was generally well shown because of the high axial resolution of the system. Reproducibility of end-diastolic thickness and percent thickening in canine studies are shown in figure 4. Diastolic thickness values were quite reproducible, with a mean difference of 8.5 ± 8.7% (sb) between paired values from the same site. Percent thickening at individual sites was more variable, particularly in vigorously contracting segments, with a mean difference of 22.2 ± 15.4%. However, this variability was not associated with any variation in the time course of thickening and did not alter qualitative classification of any segment by the criteria used for human data.

Analysis of the relationship between local myocardial thickness and percent thickening in the normal canine left ventricle demonstrated an inverse relationship (r = −0.56), such that thicker sites showed less thickening than thinner sites. When examined in tiers with reference to the left ventricular long axis (fig. 5), a gradient of diminishing thickness was visible from base to apex, with thinner, apical sites having higher percent thickening than thicker basal sites. A similar trend was evident in human data.

The echographic effect of acute ischemia produced by transient coronary occlusion (fig. 6) was prompt

![Figure 3](http://circ.ahajournals.org/content/iupui/1/1052/article-fig3.jpg)
elimination of normal systolic thickening. Serial recordings from single left ventricular sites (fig. 7) demonstrated that normal systolic thickening ceased within 10–15 seconds of occlusion and systolic thinning rapidly developed at severely affected sites. During reperfusion, mixed systolic patterns of early systolic thinning and late systolic thickening were often observed, and diastolic function remained markedly impaired. At less severely ischemic sites, systolic thinning did not develop and some residual systolic thickening was often preserved.

Rhodamine perfusion studies in nine dogs (fig. 8) showed a close relationship between the location of marked qualitative contractile abnormalities and the nonfluorescent ischemic zone. However, quantitative analysis demonstrated that, in addition to the expected depression of contractile function in the ischemic zone during occlusion (from 56% to 21%, \( p < 0.001 \),

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**Figure 4.** (left) Segment thickness on repeated transducer placements within the same epicardial site numbers under basal conditions. There is no significant change in mean thickness, and only one segment changes by more than 2 mm. (right) Percent thickening at each site on successive transducer placements. Mean percent thickness does not change significantly. Although individual sites vary appreciably, in no instance was qualitative classification of site performance altered.

**Figure 5.** Diastolic thickness and percent thickening values are grouped in tiers of sites from apex to base. The basal tier includes sites 26, 30, 35, 40, 43, 46 and 50. The apical tier includes sites 34, 39, 54; see figure 2. (left) A significant fall in end-diastolic thickness in two most apical tiers. (right) A significant increase in mean percent thickening at the two thinnest, most apical tiers.
adjacent border sites that themselves fluoresced normally also showed a statistically significant fall in percent thickening (from 64% to 41%, \( p < 0.005 \)).

Walton-Brodie strain-gauge recordings from normal sites demonstrated the characteristic normal late systolic tension increase, which was abolished by coronary occlusion (fig. 9). However, when strain-gauge recordings were compared with echocardiograms from immediately adjacent sites in the ischemic zone, the echocardiograms showed more striking and sustained abnormalities during ischemia and reperfusion than the strain-gauge recordings. Moreover, when the ischemic zone was small, as in four of seven studies, strain gauges could not be placed with sufficient spatial selectivity to show contraction abnormalities, even when adjacent echocardiograms demonstrated marked ischemic changes.

**Human Studies**

All subjects studied underwent coronary bypass surgery for disabling angina due to two- or three-vessel coronary artery disease. Five had prior documented myocardial infarctions, but only one had residual pathologic Q waves on the ECG. Six had ventriculographic segmental contraction abnormalities of varying severity, but only one had a reduced ejection fraction. One subject with documented prior subendocardial infarction had a normal ventriculogram. No intraoperative or postoperative complications have resulted from the mapping procedure.

Echocardiographic contraction patterns in segments supplied by angiographically normal coronary arteries were qualitatively similar to those from open-chest canine studies. Normal patterns of myocardial thickening and thinning were also found in many segments supplied by coronary vessels with high-grade proximal stenosis (fig. 10). In contrast, infarcted segments corresponding to electrocardiographic Q waves were akinetic (fig. 10). A minority of segments unassociated with electrocardiographic Q waves, but supplied by severely stenotic (\( \geq 90\% \)) vessels showed abnormalities of varying severity, manifested as delayed and reduced systolic thickening and diastolic thinning (fig. 11).

All six subjects with ventriculographic abnormalities showed echocardiographic abnormalities in the same region. In addition, four subjects with normal ventriculograms showed one or more abnormal segments by echocardiography. In three of these four, the segments affected would be expected to be bordering on ventriculography (fig. 12).

Repeat echocardiographic mapping was performed 5–10 minutes after completion of cardiopulmonary bypass in five subjects. No new abnormalities were observed in segments supplied by normal vessels, and most of the segments studied showed similar contraction patterns before and after bypass. However, in two subjects, clear-cut improvement in the extent and normalization of the time course of thickening were noted in one or more segments (fig. 13). One also showed a new segmental abnormality proximal to a graft site. In two others, previously abnormal segments showed complete disappearance of systolic thickening (fig. 14). In a fifth subject, both improvement and worsening of previously abnormal segments were noted. In

**Figure 6.** (left) Normal systolic thickening pattern at site 32. (right) Recording 20 seconds after occlusion of the large diagonal branch that supplied segment 32 demonstrates elimination of the normal systolic thickening pattern.

**Figure 7.** Digitized plots of time course of myocardial thickness in single cardiac cycles at intervals after transient occlusion (ischemia) and release of occlusion (reperfusion) at a single site. Each plot begins at QRS onset and zero-reference baselines are omitted for display purposes. Upward movement of the plotted line represents thickening and downward movement represents thinning. At 15 seconds of ischemia, normal systolic thickening has disappeared. From 45–105 seconds, progressive systolic thickening of the segment is shown. Reperfusion results in progressive return of systolic thickening, but even at 105 seconds of reperfusion, there is early systolic thinning, followed by later systolic thickening. Diastolic wall thinning remains markedly reduced and delayed.
several subjects, serial recordings were obtained to assess the effect of time after cold ischemic arrest on contractile function. No changes were observed over a 20-minute period after termination of cardiopulmonary bypass.

**Discussion**

Numerous animal studies have demonstrated that analysis of segmental wall thickening, obtained from implanted sonomicrometry crystals by the pulse-transit technique, is a valuable method for assessing regional myocardial function in normal, hypertrophied or ischemic myocardium. Methods available for assessment of regional myocardial function in the human heart are less direct and have poorer spatial resolution. They rely primarily on regional cavity silhouette motion by contrast or radionuclide ventriculography, regional count changes by radionuclide ventriculography, or the limited access to wall thickness analysis and cavity dimensions provided by external echocardiography. Availability of a more precise technique for analysis of regional myocardial function in man, even if limited to intraoperative use, might greatly enhance understanding of myocardial function in ischemic heart disease. Sonomicrometry crystal implantation and alignment are time consuming and cause some tissue trauma, so that sampling of more than a few sites on a single heart would be impractical. This sampling

**Figure 8.** (left) Coded results of epicardial echo mapping at multiple sites after ligation of a large diagonal branch of the left anterior descending coronary artery in an open-chest dog. Segment performance is graded as normal, hypokinetic (mildly abnormal) and akinetic (severely abnormal). (center) Fluorescence photograph of the epicardium of the same heart after rhodamine injection and sacrifice. The numbers refer to the location of sites shown on the map. The dark area is the nonperfused zone at the epicardial surface. (right) Opened endocardial surface of the same heart is shown by fluorescence photography. The dark zone depicts the endocardial extent of the nonperfused zone. All segments showing endocardial or epicardial lack of perfusion show qualitative contraction abnormalities.

**Figure 9.** Time course of changes in contraction pattern during occlusion and reperfusion as indicated by computer plots of digitized echocardiographic (left) and strain-gauge (right) transducers placed side by side on a myocardial segment in the ischemic zone. Reduced contraction is apparent at 4 minutes as a marked reduction in thickening. A simultaneous strain-gauge record shows a subtle late systolic reduction in tension. By 1 minute of reperfusion, the strain-gauge record is back to baseline, while the echocardiographic record remains mildly abnormal on the 12-minute record, with delayed time course of thickening and relaxation. Time 0 is QRS onset on each cycle and the zero baseline for each echo thickness plot has been omitted for clarity of display.
transmural infarction reflected epicardial disease unpredictable. Kerber and co-workers showed the suitability of epicardial reflected ultrasound for assessment of segmental myocardial function in experimental models; Spotnitz and co-workers showed the feasibility of human intraoperative epicardial echocardiographic imaging in valvular heart disease. The present study describes an intraoperative echocardiographic method suitable for the topographic mapping of segmental myocardial function in canine models of acute ischemia and in human ischemic heart disease. Use of a short-focus, high-frequency miniaturized transducer permits rapid, high-resolution imaging of a large number of sites on both ventricles, as well as the interventricular septum. Image quality is sufficient to resolve right ventricular free wall thicknesses of only a few millimeters and to demonstrate the previously reported normal regional variation in left ventricular wall thickness and performance.

The initial animal studies demonstrated the sensitivity of epicardial echo recordings to the effects of acute ischemia on local myocardial function. During short (2-minute) coronary occlusions and reperusions, the echo images showed changes similar to those described with implanted sonomicrometry crystals. Furthermore, within the space of a 2-minute occlusion, an average of 16 left ventricular sites could be recorded and analyzed. A limited comparison of echocardiographic and strain-gauge recordings suggested that the echocardiographic method was more sensitive and had better spatial resolution for assessment of small areas of ischemia.

The ability to map performance topographically with the method permitted use of rhodamine, a fluorescent perfusion marker, to demonstrate that myocardial thickening can be diminished in perfused myocardium adjacent to an ischemic zone. In the rhodamine series, small ischemic zones were created by diagonal occlusion. Residual systolic function was observed at 22 of 23 ischemic sites. This was due to the known persistence of perfusion via collaterals in dog, which creates islands of functioning myocardium within the ischemic zone that are not resolvable by macroscopic fluorescence mapping. In other animals in which larger ischemic zones were created, typical systolic thinning was demonstrable in the center of the ischemic zone.

A critical component of any new method is the reproducibility of the technique. We performed reproducibility studies using each dog as its own control, repeating echocardiography under stable conditions within 5 minutes of the first recording. This eliminated fluctuations due to heart rate, blood pressure and an-

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**Figure 10.** (left) Normal pattern of myocardial thickening and relaxation at site 32, on the anterolateral wall of the left ventricle in a subject with a proximal 90% stenosis of the left anterior descending coronary artery supplying that segment via a diagonal branch. (right) Echo recording at site 50 on the inferobasal wall of the left ventricle in a subject with prior transmural inferior myocardial infarction (MI) shows no change in thickness throughout the cycle. A mural thrombus, demonstrated subsequently during endocardial excision at the site for ventricular tachycardia, contributes to the thickness of the ventricular wall.

**Figure 11.** Segmental hypokinesis at site 28 in the absence of transmural infarction by ECG. There is diminished and delayed thickening and relaxation.
esthetics. Results indicate that mean values for group data are quite reproducible, although individual data for percent thickening can show considerable variation. Despite the variability of percent thickening, no changes in qualitative classification of segment performance occurred. This is due to the marked difference in both degree and time course of thickening between normal and hypokinetic segments observed in both animal and human studies (figs. 6, 10 and 11). Further experience with the method and improved transducer designs may lead to improved reproducibility. Similar reproducibility studies in humans will take longer.

Another important problem is development of normal values for human studies. Normal values will be difficult to obtain because almost all adults who undergo cardiac surgery have coronary or valvular disease that affects the left heart. The problem is made even more complicated by the normal regional differences in function demonstrated in the animal studies, which imply that normal values must be developed for each tier of segments in the ventricle. Other problems are the large number of preoperative medications known to affect myocardial function and the numerous intraoperative anesthetic and hemodynamic variables.

Thus, human studies will probably address only very marked qualitative differences between segments and paired comparisons before and after bypass on multiple segments.

It might also be argued that the placement of the transducer directly on the myocardium could interfere with epicardial blood flow, distorting the underlying myocardial function. However, other methods of assessing segmental function that use epicardial suturing of strain gauge or sutured or implanted ultrasonic crystals are considerably more traumatic.

The initial intraoperative studies in man have shown

**Figure 12.** Comparison of echocardiographic mapping and ventriculography in a subject with subendocardial infarction and three-vessel disease. The ventriculogram is normal, but an extensive area of anteroapical hypokinesis is present on the echocardiogram in segments that should be border-forming on the ventriculogram.

**Figure 13.** Echocardiographic maps summarizing the extent of difference between pre- and postbypass maps from a subject with extensive prebypass anteroapical hypokinesis and akinesis by both echocardiography and ventriculography. The apical akinetetic zone at site 34 has improved, along with the previously hypokinetic site 39. However, site 27 is now hypokinetic. The bypass graft to the left anterior descending artery in this patient was inserted at the junction of sites 27 and 28. Uncoded zones were not mapped or yielded technically unsatisfactory recordings.

**Figure 14.** Comparison of pre- and postbypass maps shows worsening of function in previously hypokinetic segments 29 and 34, distal to a bypass graft to the left anterior descending coronary artery. Uncoded zones were not mapped or yielded technically unsatisfactory recordings.
that the echocardiographic mapping technique is safe, fast and practical. The echocardiographic features of local myocardial performance differences were readily apparent on visual inspection of the records. Areas of noncontractile transmural scar, depressed but noninfarcted myocardium and normal myocardium were easily differentiated. Echocardiographic abnormalities were found in all regions mapped that were ventriculographically normal. In addition, echocardiographic abnormalities were detected in four subjects with normal ventriculograms, three of whom had a history of documented subendocardial infarction. This is not surprising, since ventriculography can demonstrate only those segments that are border-forming in a given projection. Thus, the right oblique ventriculogram shows only 10 of 30 left ventricular free wall sites on the echocardiographic map. A "half-axial" left oblique ventriculogram would provide analysis of an additional five lateral wall and five of 24 echocardiographic septal sites. At best, biplane ventriculography might demonstrate 20 of 54 ventricular myocardial segments potentially assessable by echocardiography.

Several additional factors may contribute to discrepancies between ventriculographic and echocardiographic results. First, the two methods measure different variables. Endocardial motion on ventriculography may not always correspond to shortening of the underlying ventricular myocardium, particularly when ventricular systolic shape is abnormal. An example is the abnormal septal motion pattern seen in right ventricular volume overload, which occurs because of a ventricular shape abnormality despite normal function of the septal myocardium. In contrast to the complex relationship between endocardial motion and intrinsic myocardial function, myocardial thickening may be the best available index of segmental contribution to ventricular performance. Second, major time intervals, medication changes and effects of anesthesia also separate angiography and surgery and could permit both appearance and disappearance of areas of segmental dysfunction.

Our initial observations on the effect of coronary surgery on contractile function suggest that function of most myocardial segments is not changed acutely by the procedure. Changes we have observed have been limited to grafted segments that were abnormal before bypass. Both improvement and worsening of contractile function were seen. Differences in the contractile response to bypass grafting did not appear to be due to preexisting differences in coronary anatomy. Worsened function was not associated with overt postoperative evidence of infarction. The prompt return of contractile function in most segments after cardiopulmonary bypass and the constancy of the patterns observed when repeated records were obtained suggest that observed changes were not transient or attributable to cold ischemic arrest itself. A much larger body of data and postoperative follow-up studies will be required to clarify the significance of these initial observations. Reproducibility of the echocardiographic method must also be critically assessed in man.

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