Doppler Tissue Imaging Quantitates Regional Wall Motion During Myocardial Ischemia and Reperfusion

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Background—Quantification of regional myocardial function is a major unresolved issue in cardiology. We evaluated the accuracy of pulsed Doppler tissue imaging (DTI), a new echocardiographic technique, to quantify regional myocardial dysfunction induced by acute ischemia and reperfusion.

Methods and Results—In nine open-chest anesthetized pigs, various degrees of regional wall motion abnormalities were induced by graded reduction of left anterior descending coronary artery (LAD) blood flow. Pulsed Doppler tissue imaging was performed from an epicardial apical four-chamber view with the sample placed within the middle part of the septal wall. Peak septal velocities were calculated during systole, isovolumic relaxation, and early and late diastole. Regional myocardial blood flow and systolic and diastolic dysfunctions were assessed by radioactive microspheres and ultrasonic crystals, respectively. Ischemia resulted in a significant rapid reduction of systolic velocities and an early decrease in the ratio of early to late diastolic velocities. Both changes were detected by pulsed DTI within 5 seconds of coronary artery occlusion. The decrease in systolic velocity significantly correlated with both systolic shortening (r = .90, P < .0001) and regional myocardial blood flow (r = .96, P < .0001) during reduction of LAD blood flow.

Conclusions—These results suggest that DTI may be a promising new tool for the quantification of ischemia-induced regional myocardial dysfunction. (Circulation. 1998;97:1970-1977.)

Key Words: imaging • ischemia • echocardiography • ultrasonics

Because segmental wall motion abnormalities are the hallmark of coronary artery disease, ultrasound technique is widely used for the evaluation of regional left ventricular function because of its ability to depict endocardial excursion, myocardial thickening, and wall motion in real time.1–7 However, conventional assessment of wall motion, based on visual interpretation of endocardial excursion and myocardial thickening, suffers from the limitations of a qualitative method and is subjective and experience dependent.8 Quantitative techniques, based on the manual9–14 or automatic15,16 myocardial edge detection, have demonstrated acceptable correlations with other available techniques. However, quantitative analysis is complicated by endocardial “dropout” and trabeculae, which can impair the tracing of endocardial border. There is therefore a need for ongoing development for quantification of global and regional left ventricular function.9–16

DTI is a new ultrasound technique that is based on color Doppler imaging principles and allows quantification of intramural myocardial velocities by detection of consecutive phase shifts of the ultrasound signal reflected from the contracting myocardium.17–19 To display regional myocardial velocities, thresholding and filtering algorithms are changed to reject the low-amplitude echoes from the blood pool. DTI allows the high-intensity–low-amplitude information from the myocardium to pass to subsequent determination of the mean Doppler shift and hence mean velocity determination by use of standard autocorrelation methodology. Whereas conventional ultrasound techniques derive their information on myocardial function either from parameters measured from the blood-myocardial boundaries or from blood-pool Doppler indexes, DTI directly measures indexes of myocardial function from within the myocardial wall.

Little is known about the ability of pulsed DTI to identify and quantify wall motion alterations during regional ischemia.20 In the present study, we used a classic pig model of ischemia/reperfusion to investigate whether pulsed DTI might be a useful tool to analyze regional myocardial dysfunction. Specifically, we sought (1) to define the pattern of myocardial velocities during regional sequences of ischemia and reperfusion and (2) to compare DTI measurements to modifications in segment length measured by the conventional sonomicrometry technique.21

Methods

All experiments performed in this study conformed to the Guiding Principles in the Care and Use of Animals approved by the American Physiological Society.
incision in the middle myocardial layer of the left ventricle and assess regional contractile function, was inserted via a small scalpel positioned around the LAD. One pair of ultrasonic crystals, used to isolate just before the first diagonal branch. A micrometric constrictor was inserted in the fourth left intercostal space, and a segment of the LAD was formed when needed. Pigs were ventilated with room air through a tracheotomy tube, and tidal volume and rate were adjusted to provide physiological pH and blood gases. Body temperature was monitored with a rectal thermometer and kept constant by means of a heating pad (1 mg/kg SC) and anesthetized with pentobarbital (15 mg/kg IV).

Regional Myocardial Function

SS and LDL were used as indexes of systolic and diastolic function, respectively. To define these parameters, ESL and EDL were obtained from three well-separated cardiac cycles in each sample period. LV dP/dt was used to define the timing of the cardiac cycle for segment length measurements with ultrasonic crystals; EDL was the left anterior descending coronary artery

MBF = myocardial blood flow

SS = segment shortening

Selected Abbreviations and Acronyms

DTI = Doppler tissue imaging
EDL = end-diastolic lengthening
ESL = end-systolic lengthening
LAD = left anterior descending coronary artery
LDL = late diastolic lengthening
LV = left ventricle/ventricular
MBF = myocardial blood flow

Surgical Preparation

Nine farm pigs, weighing 28 ± 4 kg, were premedicated with droperidol (1 mg/kg SC) and anesthetized with pentobarbital (15 mg/kg IV). Additional intravenous administration of pentobarbital was performed when needed. Pigs were ventilated with room air through a tracheotomy tube, and tidal volume and rate were adjusted to provide physiological pH and blood gases. Body temperature was monitored with a rectal thermometer and kept constant by means of a heating pad and 0.5 mg/kg IV Unisperse Blue Pigment (Ciba-Geigy) was injected to delineate the in vivo area at risk as previously described. Under deep anesthesia, the heart was stopped by intravenous injection of potassium chloride (20 mEq), excised, and cut into 5- to 7-mm-thick transverse slices parallel to the AV groove. We verified that the interventricular septum in the five apical transverse slices was unstained, ie, that the Doppler sampling gate was well in the ischemic area. The correct position of the two ultrasonic crystals within the risk region was checked, and two transmural myocardial samples were excised (one from the ischemic and one from the nonischemic zone) for further measurement of regional MBF.

Data Analysis

Heart rate and arterial and LV blood pressures were measured and averaged over 5 continuous cardiac cycles in sinus rhythm at baseline, during coronary artery stenosis or occlusion, and after reperfusion.

Echographic Measurements

From the DTI tracings, we measured the peak velocity of (1) isometric contraction (V_{IC}), (2) systolic excursion (V_{S}), (3) isometric relaxation (V_{IR}), and (4) early (V_{E}) and late (V_{A}) diastolic excursion (Fig 1). Five beats were averaged for each of these measurements. By definition, velocities were encoded positive or negative when the displacement of the myocardium was directed toward or away from the transducer, respectively.

The variation of myocardial velocity during or after coronary occlusion (V_{occl}) was expressed as a percentage of baseline velocity (V_{basal}) as \( \% = \frac{V_{occl} - V_{basal}}{V_{basal}} \times 100\% \). Intraobserver variability was tested in eight pigs by repeating the measurements on two occasions under the same basal conditions. To test the interobserver variability, the measurements were repeated from the videotape recordings by a second observer who was unaware of the results of the first observer. For measurement of systolic velocity, and early and late diastolic velocities, intraobserver and interobserver variability ranged from 3.9% to 4.5%.

Regional Myocardial Function

After baseline measurements, a graded reduction of the LAD blood flow was performed by progressively (and, finally, completely) tightening the micrometric constrictor. In each animal, several degrees of constriction were adjusted to obtain various values of regional wall motion abnormalities ranging from hypokinesis to dyskinesis: this was done in a stepwise manner to reduce function by \( \sim 40\% \), 60%, and finally dyskinesis during total occlusion.

Echographic and segment length recordings were performed sequentially at the following time points: at baseline, during partial stenosis, during total occlusion, and after reperfusion of the LAD. At the end of each experiment, the LAD was briefly reocluded and 0.5 mg/kg IV Unisperse Blue Pigment (Ciba-Geigy) was injected to delineate the in vivo area at risk as previously described. Under deep anesthesia, the heart was stopped by intravenous injection of potassium chloride (20 mEq), excised, and cut into 5- to 7-mm-thick transverse slices parallel to the AV groove. We verified that the interventricular septum in the five apical transverse slices was unstained, ie, that the Doppler sampling gate was well in the ischemic area. The correct position of the two ultrasonic crystals within the risk region was checked, and two transmural myocardial samples were excised (one from the ischemic and one from the nonischemic zone) for further measurement of regional MBF.
TABLE 1. Hemodynamics and Regional MBF

<table>
<thead>
<tr>
<th></th>
<th>Baseline</th>
<th>Stenosis</th>
<th>Occlusion</th>
</tr>
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<tbody>
<tr>
<td>HR, bpm</td>
<td>146±7</td>
<td>131±4</td>
<td>130±4</td>
</tr>
<tr>
<td>SBP, mm Hg</td>
<td>94±4</td>
<td>88±5</td>
<td>84±4</td>
</tr>
<tr>
<td>DBP, mm Hg</td>
<td>61±4</td>
<td>58±5</td>
<td>56±3</td>
</tr>
<tr>
<td>MBF, mL·min⁻¹·g⁻¹</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Ischemic zone</td>
<td>0.90±0.19*</td>
<td>0.11±0.03*</td>
<td></td>
</tr>
<tr>
<td>Nonischemic zone</td>
<td>1.58±0.19</td>
<td>1.44±0.35</td>
<td></td>
</tr>
</tbody>
</table>

HR indicates heart rate; SBP, systolic blood pressure; and DBP, diastolic blood pressure. All values are expressed as mean±SE. *P<.05 vs nonischemic zone MBF.

mean±SE. A value of P<.05 was considered to indicate a statistically significant difference.

Results

Nine pigs were entered into the present study. Each animal underwent 1 to 5 episodes of either partial stenosis or total occlusion of the LAD, each separated by an intervening reperfusion period. The total duration of these ischemic events averaged 6±2 and 6±1 minutes, respectively. This design allowed us to record 59 matched measurements of DTI velocities and segment lengths among the nine animals.

Hemodynamics and Regional MBF

All pigs had similar heart rate and blood pressure at baseline (Table 1). Neither partial stenosis nor total coronary artery occlusion significantly altered heart rate or blood pressure. After partial stenosis, MBF in the risk area averaged 57±2% of that in the nonischemic bed (Table 1). As expected in this collateral-deficient species, total LAD occlusion resulted in a dramatic decrease in MBF that averaged 9±2% of MBF in the remote nonischemic zone (Table 1).

Normal Pattern of Midseptal Wall Velocities

In the open-chest pig under baseline conditions, pulsed DTI of midseptal wall velocities displayed five consecutive waves whose directions varied according to the phase of the cardiac cycle. During systole, two positive waves occurred, one positive and short wave corresponding to the isometric contraction (V_{IC}) (starting at the beginning and ending at the end of the QRS complex) and one single ogival wave corresponding to LV ejection (V_{E}) (starting at the end of the QRS complex and ending at the end of the T wave). During diastole, one positive and two negative waves were sequentially observed: a positive isometric relaxation wave (V_{IR}) followed by a negative early (V_e) rapid-filling wave and a negative late-filling (V_{LA}) wave corresponding to atrial contraction.

In the five pigs that underwent pulsed DTI analysis before thoracotomy, similar sequential events were observed, but in all cases, the IR wave was negative instead of positive (Fig 1). In addition, peak systolic velocity values were slightly but significantly higher than in open-chest preparations (Table 2). Myocardial velocities obtained in the open-chest preparations were used as control values for further comparison during LAD occlusion.
Time Sequence and Pattern of Myocardial Velocity Changes During Ischemia/Reperfusion

The time to onset of regional myocardial velocity abnormalities in the ischemic myocardium is presented for the first 60 seconds for 10 episodes of total LAD occlusion in eight pigs (Fig 3). Within 5 seconds of occlusion, systolic velocities ($V_S$) in the ischemic segment decreased to 46% of baseline values ($P < .0001$). Systolic velocities became negative at $\approx 30$ seconds and peaked at 1 minute of occlusion. These negative velocities corresponded to the paradoxical expansion of the ischemic segment observed on sonomicrometry tracings (Figs 2 and 4). This early decrease in $V_S$ was associated with a simultaneous increase in velocities during both isometric systole ($V_{IC}$) and isometric ($V_{IR}$) relaxation (Fig 3). Velocity during isometric relaxation progressively increased and peaked at 1 minute after coronary artery occlusion (Fig 3). At 1 minute after reflow, $V_S$ and $V_{IR}$ exhibited a transient positive and negative peak, respectively, corresponding to the hyperemic phase (Fig 3). Within 5 minutes of reperfusion, $V_S$ progressively decreased, whereas $V_{IR}$ increased and appeared as a positive wave as reperfused myocardium developed postischemic stunning (Figs 3 and 4).

Diastolic abnormalities also occurred very quickly after the onset of ischemia. Immediately after LAD occlusion, $V_E$ significantly decreased, and $V_A$ increased (Fig 4). As a consequence, $V_E/V_A$ ratio decreased and further remained stable until reperfusion (Fig 3). $V_E/V_A$ peaked at 1 minute after reflow (hyperemic response) and thereafter returned nearly ischemic values as diastolic alterations of myocardial stunning appeared on segment length recordings.

In five pigs, we also measured velocities in the remote nonischemic lateral wall during ischemic episodes induced by partial stenosis of LAD. Ischemia in the LAD territory did not significantly alter systolic or diastolic velocities in the lateral wall: $V_S$ averaged 8.9 ± 0.7 versus 9.1 ± 0.6 cm/s at baseline ($P = NS$) and $V_E/V_A$ averaged 1.3 ± 0.5 versus 1.3 ± 0.4 at baseline ($P = NS$).

### Table 2. Comparison of Myocardial Velocities (cm/s) Within Mid Septal Wall in Pigs Before and After Surgery

<table>
<thead>
<tr>
<th>Before Surgery</th>
<th>Open Chest</th>
<th>$P$</th>
</tr>
</thead>
<tbody>
<tr>
<td>$V_{IC}$</td>
<td>3.5 ± 1.5</td>
<td>3.6 ± 0.4</td>
</tr>
<tr>
<td>$V_S$</td>
<td>9.2 ± 0.6</td>
<td>8.4 ± 0.6</td>
</tr>
<tr>
<td>$V_{IR}$</td>
<td>-2.9 ± 1.2</td>
<td>3 ± 2.2</td>
</tr>
<tr>
<td>$V_E$</td>
<td>-17.6 ± 0.9</td>
<td>-7.9 ± 1.1</td>
</tr>
<tr>
<td>$V_A$</td>
<td>-7.2 ± 2.2</td>
<td>-9 ± 1.8</td>
</tr>
<tr>
<td>$V_E/V_A$</td>
<td>2.63 ± 0.3</td>
<td>1.2 ± 0.4</td>
</tr>
</tbody>
</table>

$V$ indicates velocity; IC, isometric contraction; S, systole; IR, isometric relaxation; E, early diastole; and A, atrial contraction. All values are expressed as mean ± SE, and velocities are given in centimeters per second.

![Figure 3](http://www.ahajournals.org/doi/fig/10.1161/HC3301.102378)

**Figure 3.** Graph of the sequential changes during a brief total occlusion of LAD on systolic and isovolumic relaxation velocities (left) and on diastolic velocities (right). Ten episodes of brief total occlusion of LAD were performed in 8 pigs. Immediately after LAD occlusion, early and significant decrease of systolic ejection velocities ($V_S$) was associated with the increase of velocities during isometric relaxation ($V_{IR}$), the decrease of early diastolic velocities ($V_E$), and the increase of late diastolic velocities ($V_A$), leading to a significant decrease in the ratio $V_E/V_A$.

After 1 minute of occlusion, $V_S$ became negative whereas $V_{IR}$ markedly increased. Immediately after reperfusion, $V_S$ returned positive and recovered to a significant higher value than at baseline. However, within 3 minutes of reperfusion, $V_S$ decreased again, whereas $V_{IR}$ increased, identifying myocardial stunning. *$P < .05$ vs baseline; **$P < .01$ vs baseline; ***$P < .001$ vs baseline.
Comparison Between DTI Velocities and Wall Motion Abnormalities

To evaluate whether the severity of ischemia could be accurately predicted by DTI, the individual systolic velocity and diastolic $V_s/V_a$ ratio (both expressed as a percentage decrease from baseline) were plotted versus corresponding modifications in segment length. There was a significant correlation between the variations of systolic velocity ($V_s\%$) and those of $SS$: $V_s\% = 0.78(\Delta SS) + 10.6$ ($r = .90, P < .0001$; Fig 5). This strongly supports that the DTI measurement of $V_s$ accurately quantifies the ischemia-related regional wall motion abnormalities. The diastolic ratio $V_s/V_a$ also showed a significant correlation with late diastolic lengthening, but the relationship was weak ($r = .39, P = .0049$) (Fig 6).

Comparison Between DTI Velocities and Regional MBF

Eleven measurements of regional MBF were obtained in eight pigs and plotted versus simultaneous myocardial systolic velocities (Fig 7).

The relationship between the decrease in MBF (MBF%) and in systolic velocity ($V_s\%$) was best fitted by a polynomial expression according to the following equation: $V_s\% = -0.004 \text{ MBF}\%^2 + 1.73 \text{ MBF}\% - 49.14$ ($r = .96$; Fig 7B).

A similar correlation existed between SS and MBF%, but this relationship was best fitted by a linear regression equation (Fig 7B).

Myocardial function became severely depressed when MBF decreased <40% of baseline values.

Discussion

In the present study, we report for the first time that pulsed DTI can identify and quantify myocardial wall velocities during regional ischemia and reperfusion. As demonstrated by concomitant measurement of wall motion by sonomicrometry and assessment of regional MBF by the radioactive microsphere technique, pulsed DTI appears to be accurate and reproducible.

DTI is a new echocardiographic method based on the Doppler principle, which provides a velocity map of the myocardial wall. 17–19 DTI velocity maps are available by use of two-dimensional imaging and M-mode and pulsed-wave Doppler. Low frame rates available from two-dimensional acquisition associated with the Doppler angle of insonation of the myocardium preclude two-dimensional DTI for measurement of rapid myocardial velocity changes. M-mode DTI interrogation of intramural velocities overcomes the temporal resolution problems inherent with the two-dimensional approach and allows the assessment of endocardial to epicardial velocity gradient.24,25 But it needs the development of special programs for off-line analysis because M-mode quantification of velocities is not yet available on our ultrasound system. Pulsed-wave DTI provides quantitative information available on-line and was therefore used in this study to analyze septal wall velocity resulting from long-axis shortening of the heart and its variations after LAD occlusion.
velocities was described by Rodriguez et al.26,27 within the recordings of midseptal velocities. The same pattern of the cardiac cycle can be described for the first time from velocity, including two systolic and three diastolic events of the Doppler beam and does not allow accurate myocardial septal or the lateral wall, moves perpendicularly toward the short-axis view, a part of the myocardium, particularly the septal wall motion and that of the Doppler beam. Indeed, in the short-axis view, a part of the myocardium, particularly the septal or the lateral wall, moves perpendicularly toward the Doppler beam and does not allow accurate myocardial velocity measurement.

In the present experimental preparation, five waves of wall velocity, including two systolic and three diastolic events of the cardiac cycle, can be described for the first time from recordings of midseptal velocities. The same pattern of velocities was described by Rodriguez et al.26,27 within the mitral annular and Isaaz et al.28 within the LV posterior wall.

Both sets of authors reported a good correlation between myocardial velocities derived from M-mode tracings and DTI measurements. However, the determination of myocardial velocities by M-mode appeared difficult, time-consuming, and poorly reproducible, which might explain why M-mode echocardiography has been considered an unreliable tool for assessment of regional wall function.29

It is worth noting that velocity of isometric relaxation shifted from negative to positive values when the chest was opened. Although we did not specifically investigate this issue, we speculate that it may be related to modifications of the transeptal interaction between the two ventricles, secondary to reduction of loading conditions and pericardectomy in the open-chest preparation.30

**Detection and Quantification of Ischemia-Related Wall Motion Abnormalities by DTI Velocities**

To investigate whether pulsed DTI could accurately identify and quantify the alterations of myocardial wall motion induced by ischemia, we compared the changes in velocities to those in segment lengths as measured by the reference method, ie, sonomicrometry.21 Within 15 seconds of coronary occlusion, systolic contraction decreased and resulted in passive bulging of the myocardium in case of severe ischemia. As expected, these modifications of systolic wall motion were also significantly correlated with the reduction of regional MBF as measured by the radioactive microsphere technique.31–33 Simultaneous diastolic dysfunction developed with a rapid increase in EDL and LDL.34

Our DTI data are in close agreement with these observations. Pulsed DTI was able to detect significant systolic and diastolic velocity changes as soon as 5 seconds after LAD occlusion, a time frame comparable to those reported when sonomicrometry was used.31–33 Systolic velocity during the ejection phase was well correlated with segment shortening, whatever the severity of ischemia. During isometric relaxation, velocity became positive and markedly increased. This paralleled post–SS observed on segment length recordings and suggests an asynchrony in myocardial contractility as previously reported by Gibson et al.35 in patients with coronary artery disease. Early diastolic velocity decreased and late diastolic velocity increased, resulting in an inversion of the \( V_d/V_A \) ratio. Its correlation with LDL, as described with sonomicrometry, was statistically significant but obviously much weaker than analysis of the systolic pattern. The reason for the poor correlation between diastolic indexes is unclear but might be related to the complex translation/rotation of the heart during the cardiac cycle. It might also be related to the fact that DTI analyzed septal wall motion, which is affected by right ventricular pressures whose influence is likely more important in diastole.

Although \( V_S \) slightly overestimated the degree of regional wall motion abnormalities, it appears to be a useful index for assessment of regional wall motion impairment related to severe, moderate, or even mild ischemia. This was also confirmed by the correlation of ejection systolic velocity and regional MBF. Even slight reductions in regional MBF were associated with a decrease in both myocardial velocities and segment shortening. We observed that ejection systolic ve-
Occult became negative for a reduction in MBF reaching 40% of baseline values, which is also associated with the onset of regional bulging as previously described.22

Interestingly, pulsed DTI was able to identify the hyperemic response after reperfusion of the ischemic myocardium. Although this transient increase in regional contractile function is usually short-lived, its identification may be useful as an indicator of reperfusion in the setting of acute myocardial infarction. When postischemic contractile dysfunction (“myocardial stunning”) developed despite restoration of a normal (or nearly normal) MBF, pulsed DTI clearly identified wall motion abnormalities similar to those observed during ischemia.36–38 In other words, as expected, pulsed DTI (as any other technique) failed to distinguish ischemia from reperfusion-induced contracture dysfunction. With respect to this, estimation of myocardial perfusion through measurement of myocardial wall velocities must be done cautiously and is valid only in situations of ischemia but not reperfusion.

Potential Clinical Implications

There are major potential clinical implications to the use of pulsed DTI. In particular, we currently lack a reliable technique to accurately quantify regional contractile function in humans. Contrast and radionuclide ventriculography and conventional two-dimensional echocardiography only allow semiquantitative evaluation of LV function. Today, only cine MRI can quantify wall motion, but it is not easily accessible for a large number of patients. Pulsed DTI appears to be a sensitive, reproducible, accurate, noninvasive echocardiographic technique that may become a very useful clinical tool for the diagnostic, follow-up, and evaluation of the prognosis of cardiac diseases. Whereas effective clinical application of DTI was hampered by low acquisition frame rates and a lack of postprocessing software, a new third generation of Doppler myocardial imaging system, with high temporal and spatial resolution, has been developed that allows real-time acquisition with subsequent on-line analysis of regional mean velocities. This new system has recently been shown to provide reproducible and accurate quantification of LV circumferential and longitudinal contraction in all myocardial segments and therefore will allow stress echocardiography to be quantified.39,40 However, further studies are needed to determine whether data in this experimental preparation can be extrapolated to human patients.

Study Limitations

Because of the version of the ultrasound machine used in this study, we could not record simultaneously DTI velocities and two-dimensional regional wall motion abnormalities. The DTI velocity measurements were performed in the middle part of the interventricular wall septum, whereas segment length data were recorded from the anterior wall. However, these two regions are supplied by the LAD; moreover, the middle part of the interventricular wall septum was clearly included in the area at risk as shown by the Unipense Blue Pigment injection in the heart slices. It is therefore likely that wall motion abnormalities in the septum were comparable to those observed in the anterior wall.

DTI measurements, as any other method assessing myocardial excursion, are affected by cardiac translation and/or rotation. Nonetheless, the correlation with segment length measurements (that are poorly influenced by cardiac translation) is good (r = .90), suggesting that movements of the heart did not dramatically alter DTI measurements.

Conclusions

DTI is a new, accurate, sensitive, noninvasive tool to quantify on-line systolic and diastolic ischemia-induced myocardial dysfunction. It appears to be a promising method to quantify regional wall motion abnormalities in the setting of ischemic heart disease.

References

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