PATHOPHYSIOLOGY AND NATURAL HISTORY
CORONARY ARTERY DISEASE

Hierarchy of levels of ischemia-induced impairment in regional left ventricular systolic function in man

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ABSTRACT We tested the hypothesis that different subsets of ischemia-induced wall motion disorders are characterized by specific patterns of abnormal regional left ventricular systolic function. Regional contraction was quantitatively assessed from two-dimensional echocardiograms by an automated integrative analysis considering the time course of wall motion during the entire contraction sequence in 20 patients with chronic myocardial infarction, in 13 patients with impending myocardial infarction (<2 hr after the onset of symptoms), and in nine patients during transient myocardial ischemia. Wall motion abnormalities were detected in all patients by the integrative analysis. In contrast, the sensitivity for detecting wall motion abnormalities was 80% during chronic infarction, 77% during impending infarction, and 56% during transient ischemia if only end-diastolic and end-systolic frames were compared for assessment of overall regional systolic function. There were distinct differences in the time course of abnormal wall excursion between the three groups. Chronic infarction was characterized by a monophasic contraction pattern, with abnormal synergy in regional contractile events occurring predominantly during early systole. In contrast, transient ischemia caused predominantly mid-to-late systolic abnormal synergy followed by late systolic shortening corresponding to a polyphasic contraction pattern. During impending infarction, an intermediate temporal contraction pattern was present with both early and mesosystolic abnormal synergy. The synergy ratio for the timing of regional contractile events—calculated by relating the extent of mid-to-late systolic asynchrony to the extent of early-to-mid systolic asynchrony—differentiated the contraction patterns of all patients with transient ischemia (synergy ratio 3.9 ± 2.2, mean ± SD) from those with chronic myocardial infarction (0.28 ± 0.24; p<.001) and was significantly lower (p<.05) in patients with impending infarction (0.89 ± 0.38) in comparison with those with transient ischemia. Impending myocardial infarction was associated with a significantly (p<.05) larger degree of functional abnormality than chronic infarction. We conclude that there is a hierarchy of levels of ischemia-induced impairment in left ventricular systolic function: ischemic injury in man first manifests itself as asynchronous polyphasic wall motion rather than any change in amplitude of wall excursion. Our results strongly suggest the need for a comprehensive analysis of the entire contraction sequence to evaluate the functional benefits of any interventions aimed at salvage of acutely ischemic myocardium in man.


FUNCTIONALLY and temporally disordered wall motion has been shown to be a characteristic feature of left ventricular contraction in response to myocardial ischemia or infarction.1–3 Experimental studies to further categorize the mechanisms of left ventricular dyssynergy have demonstrated that functional interactions of adjacent myocardial segments modulate the time course of contraction leading to an asynchronous polyphasic motion pattern during regional ischemia.4–7 Two-dimensional echocardiography is especially useful for noninvasively visualizing cardiac wall dynamics in real time. A number of studies have attempted to quantitate regional wall motion abnormalities by comparing the changes in endocardial excursion from end-diastole to end-systole.8–11 However, since myocardial function manifests itself in the amplitude and timing of wall excursion, due to the sensitivity of any two-phase (end-diastole and end-systole) analysis to the timing of normal wall motion important abnormalities may be missed when only end-diastolic and end-systolic left ventricular outlines are compared. Of more importance, because the functional interactions of adjacent myocardial segments

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will change during infarct maturation, we hypothesized that the traditional two-phase methods would demonstrate a different performance for detecting and quantitating regional abnormal left ventricular function associated with chronic myocardial infarction as compared with that associated with impending infarction or transient myocardial ischemia.

Thus, the present study was designed to test the hypothesis that a quantitative analysis that considers wall excursion throughout the entire contraction sequence provides important information not available by two-phase analysis in patients with different subsets of ischemia-induced wall motion disorders.

To quantitate regional left ventricular wall motion on a frame-by-frame basis, an automatic endocardial contour detection system was used to exclude observer bias and inconsistency in digitizing the endocardial borders.

**Methods**

**Classification of patients.** Individual patient characteristics and angiographic findings are summarized in table 1.

**Group A: chronic myocardial infarction.** Twenty patients with a well-documented history of previous myocardial infarction and pathologic Q waves were studied during the chronic period of infarction at least 2 weeks after the acute event. None of these patients had unstable angina. Left heart catheterization and coronary angiography were performed in 16 of these patients within 24 hr of the echocardiographic examination. Four patients had both an anterior and inferior previous myocardial infarction.

**Group B: impending myocardial infarction.** Thirteen patients were studied within 2 hr after the onset of their first symptoms of an acute myocardial infarction with electrocardiographic evidence of transmural injury (ST elevation greater than 2 mm at 80 msec after the J point or new pathologic Q waves in two or more involved electrocardiographic leads, or both). All patients developed infarct-specific electrocardiographic changes and enzyme release (creatine kinase—MB fraction) during follow-up. The infarct-related vessel was assessed by coronary angiography in all patients within 1 hr of the echocardiographic study.

**Group C: unstable angina.** Nine patients with angiographically documented coronary artery disease and unstable angina were studied during a spontaneous attack of angina at rest. Six of these patients had a previous myocardial infarction at least 2 months before this study. In none of these patients did persistent changes on the electrocardiogram or increases in creatine kinase (MB fraction) occur during follow-up, indicating a lack of ischemic necrosis.

Selection of patients for the study was based exclusively on the presence of high-quality echocardiograms suitable for automatic endocardial contour detection.

**Patients with intraventricular conduction abnormalities were excluded from the study.**

**Echocardiography.** The patients were examined in the 30 to 60 degree left lateral decubitus position with a commercially available mechanical sector scanner (ATL Mark 300) with a 3.5 MHz transducer. Parasternal short-axis views and the apical four-chamber view were obtained during held midinspiration to eliminate cardiac motion due to respiration, and were recorded on videotape (VHS format) simultaneously with a lead II electrocardiogram for timing purposes.

**Image analysis.** Image processing was performed on a MIPRON I image analysis computer (Kontron Electronics, Eching, F.R.G.). The echocardiographic video frames were digitized into a 512 × 512 pixel matrix with 8 bit depth and were transferred to the image memory at 25 frames/sec, as required for real-time acquisition. The image memory allowed for storage and rapid processing of 62 frames with a 512 × 512 pixel format and 256 gray levels. The images were preprocessed by histogram modification 12 and nonlinear filtering 13 to increase contrast, reduce random noise, smooth out textural variations, and sharpen the borders between the myocardium and cavity.

**Automatic contour detection.** Endocardial edge detection was performed by a previously described and extensively validated computer algorithm 14 applied to the preprocessed images. The method is based on the detection and analysis of gray level gradients by use of some a priori information about the location of the endocardial border by a radial searching process between two variably sized filters. After identification and elimination of artifacts the detected edge points are defined within the spatial as well as within the temporal domain and the endocardial contour is completed by least squares polynomial approximation. The computer-generated endocardial contours are stored on disk for subsequent analysis.

**Quantitation of regional wall excursion.** Regional left ventricular systolic function was assessed by a previously described integrative wall motion analysis, 19 as illustrated in figure 1. In brief, the computer-generated left ventricular endocardial outlines of the sequential echocardiographic images recorded at 40 msec intervals (frame by frame) are divided into 48 equiangular segments emanating from the end-diastolic center of cross-sectional area. The fractional segmental cavity area change relative to end-diastole is calculated for each frame throughout the entire cardiac cycle. The frame with the largest total cavity area is defined as end-diastole, coinciding with the peak of the R wave in the simultaneously recorded electrocardiogram, whereas end-systole is defined as the frame with the smallest total cavity area near the end of the T wave. For each of the 48 segments, regional wall excursion is quantitated by plotting the fractional segment area change as a function of time from end-diastole to end-systole. The correlation coefficients obtained by the linear regression technique reflect the amount of linearity in regional wall excursion throughout the entire contraction sequence and thus integrate spatial and temporal variations in.

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**TABLE 1**

<table>
<thead>
<tr>
<th>Patient characteristics and angiographic findings</th>
<th>Impending MI (n = 13)</th>
<th>Chronic MI* (n = 20)</th>
<th>Unstable angina (n = 9)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (years)</td>
<td>55 ± 9</td>
<td>57 ± 11</td>
<td>64 ± 8</td>
</tr>
<tr>
<td>Sex (female/male)</td>
<td>3/10</td>
<td>5/15</td>
<td>2/7</td>
</tr>
<tr>
<td>Heart rate</td>
<td>84 ± 17</td>
<td>80 ± 9</td>
<td>83 ± 18</td>
</tr>
<tr>
<td>Infarct location (anterior/inferior)</td>
<td>8/5</td>
<td>15/9</td>
<td>3/3</td>
</tr>
<tr>
<td>Infarct/ischemia-related artery</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Left anterior descending</td>
<td>8</td>
<td>15</td>
<td>6</td>
</tr>
<tr>
<td>Right coronary</td>
<td>4</td>
<td>7</td>
<td>3</td>
</tr>
<tr>
<td>Circumflex</td>
<td>1</td>
<td>2</td>
<td>—</td>
</tr>
<tr>
<td>Angiographically visible collaterals</td>
<td>4</td>
<td>3</td>
<td>5</td>
</tr>
<tr>
<td>Extent of vessel disease</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Single</td>
<td>5</td>
<td>8</td>
<td>5</td>
</tr>
<tr>
<td>Multi</td>
<td>8</td>
<td>12</td>
<td>4</td>
</tr>
</tbody>
</table>

Plus minus values are the mean ± SD.

MI = myocardial infarction.

*Four patients had both an anterior and inferior infarction.
regional left ventricular contraction. Examples for individual segments as well as their corresponding correlation coefficients are shown in figure 2.

With the use of the junction of the right ventricular free wall and the septum as an internal landmark, the short-axis views were corrected for rotational motion of the left ventricle, but not for translation during contraction. Realignment of the apical four-chamber views was performed by superimposing the images by use of the tensor of inertia of the computer-identified left ventricular long axis. The mitral valve area was excluded from analysis.

Conventional two-frame analysis was performed by calculating the relative segmental area change from end-diastole to end-systole: (end-diastolic area – end-systolic area) × 100/end-diastolic area.

The derived variables for each individual segment were plotted against segment location progressing sequentially around the left ventricular circumference (figure 3).

The circumferential extent of abnormal wall motion was defined as the span of segments outside the normal range (derived from a normal population as mean ± 2 SDs) for both the integrative and the two-phase analysis. The absolute degree of abnormal wall motion was quantitated by integrating the area under the curve outside the normal range from the correlation plots (figure 3). The values of each echographic view obtained were summed and divided by the total number of echographic views for an individual patient.

Assessment of the timing of abnormal wall excursion. As previously reported,20,21 relative outward motion never occurred in normal subjects during two consecutive echocardiographic frames (= 80 msec) during systole except for the last two frames before end-systole. Therefore, abnormal synergy in systolic wall excursion was defined as relative outward motion during at least two consecutive echocardiographic frames during the contraction period, excluding the last two frames prior to minimum total cavity area. Exclusion of the last 80 msec of systole also eliminates the false evaluation of any phases of the isovolumetric relaxation period.

Each segment was individually evaluated for the presence and timing of abnormal synergy during the contraction sequence (figure 2). After normalizing for heart rate, a histogram was generated displaying the relative frequency distribution of the timing of abnormal synergy during systole for each group of patients. To quantitate the timing of abnormal regional contractile events for each patient and to allow for comparison between individual patients, an asynery ratio was calculated. For this purpose, the number of segments demonstrating abnormal synergy during the second half of the contraction sequence was summed and expressed as a fraction of the sum of segments demonstrating abnormal synergy during the first half of systole. Thus, an asynery ratio less than 1 indicated predominantly early-to-mid systolic abnormal synergy, whereas a ratio greater than 1 represented abnormal synergy occurring predominantly during mid-to-late systole.

Statistical analysis. The statistical computations were run on a DEC minicomputer with the use of the RS/1 software package (Bolt, Beranek and Newman Inc., 1983). The least squares linear regression technique was used to compare the circumferential extent of abnormal wall motion as assessed by the integrative analysis and the circumferential extent as assessed by the two-frame method for each group of patients as well as to correlate the circumferential extent with the absolute degree in the chronic and impending infarction groups. Group differences were evaluated by the Bonferroni modified t test after analysis of variance.

Results

Analyses of patient characteristics and coronary angiographic findings (table 1) did not demonstrate significant differences between the three groups of patients. Figure 3 illustrates a wall motion plot obtained by the integrative analysis of regional systolic function.

FIGURE 1. Graphic display of the technique for quantification of regional wall excursion throughout the entire contraction sequence. A, The left ventricular cavity is divided into 48 equiangular segments (7.5 degree intervals). Standardized subdivision is obtained with use of the right ventricular (RV)–septal junction as the internal landmark in the short-axis view. B, Computer-generated three-dimensional display of fractional segmental cavity area change (FAC, y axis) for each individual left ventricular segment (1 to 48 with corresponding anatomic region, z axis) calculated frame by frame (25 frames/sec = every 40 msec, x axis) throughout one and a half cardiac cycles. ED = end-diastole; ES = end-systole (direction of x axis is reversed for illustrative purposes). C, Quantification of the homogeneity in regional wall excursion by plotting FAC (y axis) as a function of time every 40 msec (frame by frame, x axis) from ED to ES for each individual segment (here shown for segment No. 48) and calculating the correlation coefficient (r) of the linear regression line.
at three different left ventricular short-axis levels in a patient with an inferior myocardial infarction. In normal left ventricular segments, the segmental correlation coefficients are near unity and well within the tight normal range, indicating a temporal and spatial homogeneous contraction pattern. However, when the infarcted segments of the left ventricle were approached, there was an abrupt decrease in the correlation coefficients, reflecting the temporal inhomogeneous wall excursion pattern corresponding to the abnormal synergy in regional contraction in infarcted areas. Figure 3 also demonstrates the high resolution of the integrative analysis for temporal and spatial heterogeneous contraction, allowing for the exact delineation of the circumferential extent and absolute degree of abnormal regional left ventricular wall motion.

The sensitivity for the detection of wall motion disorders as well as the circumferential extent of detected abnormal regional left ventricular function are summarized in table 2 for both the integrative and the two-phase analysis. Wall motion abnormalities were detected in all patients by the integrative analysis. In contrast, the two-phase analysis demonstrated a progressive decrease in its sensitivity to detect abnormal regional left ventricular systolic function from 80% in the chronic infarction group, to 77% in the impending infarction group, and to 56% in the patients studied during transient myocardial ischemia.

The correlation between the circumferential extent of abnormal wall motion as assessed by the two-phase analysis and the circumferential extent as assessed by the integrative method demonstrated a fairly close linear relationship with a slope near unity, indicating that both methods are comparable for delineating abnormal wall motion during the chronic infarct period (figure 4, A). In contrast, in the impending infarct group there was only a weak correlation between the circumferential extent of abnormal wall motion as assessed by the two methods (figure 4, B). A similar weak relationship was found during transient myocardial ischemia (figure 4, C). Of more importance, however, the two-phase analysis considerably underestimated the circumferential extent of transient ischemia–induced abnormal systolic function during unstable angina in addition to the failure to detect wall motion abnormalities at all in more than one-third of these patients.

The assessment of the timing of abnormal synergy in systolic wall excursion demonstrated characteristic differences between the three groups of patients. In the chronic infarct period, the relative frequency in the timing of abnormal synergy was highest during early-to-mid systole (figure 5, A). In contrast, during spontaneous attacks of unstable angina the maximum extent of abnormal synergy was found during mid-to-late systole (figure 5, C). In the patients studied during impending myocardial infarction, abnormal synergy occurred during early as well as during meso-to-late systole (figure 5, B), resulting in a temporal wall excursion pattern intermediate between that in patients with

![Figure 2](http://circ.ahajournals.org/lookup/fig/2/1/5/1/7/3)
chronic infarction and transient myocardial ischemia. Figure 6 illustrates the asynergy ratios for each individual patient within the three groups. Two patients in the chronic infarction group did not demonstrate relative outward motion on two consecutive echocardiographic frames during the contraction sequence and therefore no asynergy ratio could be calculated. Abnormal synergy occurred predominantly during mid-to-late systole in all patients experiencing transient myocardial ischemia. The asynergy ratio differentiated the abnormal wall motion patterns of all patients with unstable angina from those of patients in the chronic infarct period, which was characterized by predominantly early-to-mid systolic abnormal synergy. In addition, the patients with impending infarction exhibited an intermediate contraction pattern with significantly lower (p<.05) asynergy ratios compared with the unstable angina group, but slightly increased values compared with the chronic infarction group, which represents the mixed occurrence of early-to-mid and mid-to-late systolic abnormal synergy in regional left ventricular systolic function.

The absolute degree of wall motion abnormality as quantitated by integration of the area under the curve outside the normal range of the correlation plot ranged from 0.11 to 12.47 (2.3 ± 2.6, mean ± SD) in the chronic infarction group, from 0.26 to 26.6 (5.4 ± 7.5) in the impending infarction group, and from 0.24 to 24.9 (8.5 ± 8.0) during transient ischemia. Due to the wide scatter of the individual data the differences did not prove to be significant (p>.05).

Comparison of the circumferential extent of wall motion abnormality with the absolute degree in patients with a first myocardial infarction in the chronic infarction group (n=16) and in the impending infarction group (n=11) revealed a linear relationship with r values of .85 and .87, respectively. However, the slope of the regression equation was significantly lower (p<.05), with y = 0.16x - 0.24 for the chronic infarction group as compared with y = 0.50x - 4.2 for the impending infarction group, indicating a larger degree of functional abnormality associated with impending infarction than during chronic myocardial infarction.

### Discussion

In the experimental setting, Weyman et al. demonstrated the importance of accounting for temporal heterogeneity in assessing the contraction abnormalities associated with acute myocardial ischemia. The correlation method, which integrates endocardial motion throughout the entire systolic contraction

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**TABLE 2**

Comparison between integrative and two-phase analysis for detection of wall motion abnormalities

<table>
<thead>
<tr>
<th></th>
<th>Impending MI (n=13)</th>
<th>Chronic MI (n=20)</th>
<th>Unstable angina (n=9)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sensitivity</td>
<td>100% (13/13)</td>
<td>100% (20/20)</td>
<td>100% (9/9)</td>
</tr>
<tr>
<td>Two-phase analysis</td>
<td>77% (10/13)</td>
<td>80% (16/20)</td>
<td>56% (5/9)</td>
</tr>
<tr>
<td>Circumferential extent (in segments)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Integrative analysis</td>
<td>16.1 ± 8.2</td>
<td>14.0 ± 7.7</td>
<td>24.9 ± 15.9</td>
</tr>
<tr>
<td>Two-phase analysis</td>
<td>17.5 ± 11.1</td>
<td>10.9 ± 7.5</td>
<td>12.4 ± 5.6</td>
</tr>
</tbody>
</table>

Plus minus values are the means ± 1SD.

MI = myocardial infarction.
sequence, provided better definition of ischemic left ventricular dysfunction\textsuperscript{22} and delineation of area at risk\textsuperscript{23} than any methods that consider motion at only single points in time.

Our study is the first to use an integrative wall motion analysis of two-dimensional echocardiograms by an objective means of automatic contour detection in man. The results demonstrated that regional wall mo-

\textbf{FIGURE 4.} Correlation of the circumferential extent of wall motion abnormality (expressed in number of segments) as assessed by the two-phase analysis (two-frame WMA) and as assessed by the integrative analysis (integrative WMA) for the chronic infarction group \textit{(A)}, for the impending infarction group \textit{(B)}, and during transient myocardial ischemia \textit{(C)}.

\textbf{FIGURE 5.} Histogram displaying the mean relative frequency distribution (± 1 SD) of the temporal occurrence of abnormal synergy during the normalized contraction sequence in the chronic infarction group \textit{(A)}, in the impending infarction group \textit{(B)}, and during transient myocardial ischemia \textit{(C)}.
systolic shortening period. Muscle lengths remained constant in late systole only when contractile force and the imposed force were declining at identical rates. In the hypoxic muscle with continued ability to develop tension, the imposed force decreased less rapidly relative to that of the oxygenated myocardium, resulting in persistent shortening in late systole.26

Acute coronary occlusion in experimental animals results in progressive changes in regional wall motion, with hypokinesia during the ejection period, mid-to-late systolic outward motion, and late systolic post-ejection shortening, corresponding to a polyphasic wall excursion pattern relatively early during the development of full ischemia. More severe ischemia, however, is characterized by outward motion during the early ejection period, followed by continuous inward motion during mid-to-late systole that corresponds to a monophasic wall excursion pattern.27, 28

Sasayama et al.29 constructed regional pressure-length loops by relating segmental shortening quantified by a radial coordinate system to the corresponding left ventricular pressure simultaneously recorded during angiography. They demonstrated that the systolic shortening of the potentially ischemic segment was followed by an exaggerated late additional shortening period during the decline of the ventricular pressure. Thus, wall motion in the hypoxic, but not completely ischemic, left ventricular segment is characterized by a temporal asynchronous polyphasic wall excursion pattern.

Our results have shown that in patients with chronic myocardial infarction the maximum extent of abnormal wall motion occurs during early systole, corresponding to a completely passive outward motion of the scar region, which is unable to support stress. The wall motion pattern is therefore determined for the most part by regional tension30, 31 and wall excursion is similar to a monophasic tension-length curve, which would be expected of an elastic material without any additional intrinsic contractile activities. Therefore, the extent of wall motion abnormality as quantitated by the integrated analysis correlated fairly with the extent as quantitated by the two-phase method.

In contrast, in patients with transient myocardial ischemia during spontaneous attacks of unstable angina the evaluation of overall regional systolic function by the comparison of end-diastolic and end-systolic function considerably underestimated the extent of left ventricular dysfunction and failed to detect abnormal wall motion at all in four of nine patients. In all of these patients the maximum extent of dyssnergy was found during mid-to-late systole. Thus, systolic dysfunction

![Figure 6](image-url)
during unstable angina was characterized by a polyphasic wall excursion pattern with mesosystolic lengthening followed by additional shortening during late systole. This contraction pattern is incompatible with a purely passive behavior of transient hypoxic segments and suggests the presence of continued ability to develop tension. In addition, since overall wall excursion is conventionally measured at minimum ventricular volume or cross-sectional area (especially by applying noninvasive techniques in the clinical setting), postejection shortening is also very likely to be included in an analysis of maximum regional wall motion, resulting in a finding of normal extent of regional systolic function, even though distinct abnormalities of regional contractile events are present. The patients studied during impending myocardial infarction demonstrated a temporal pattern of wall motion abnormalities intermediate between that demonstrated by patients with chronic infarction and transient ischemia. These results strongly suggest a hierarchy of levels of ischemia-induced impairment in regional left ventricular systolic function that is reflected in regional wall excursion and is noninvasively detectable by a quantitative frame-by-frame analysis of two-dimensional echocardiograms. Ischemic injury in man first manifests itself as asynchronous wall motion rather than a change in overall amplitude of wall excursion. Because experimental studies have characterized early ischemic damage as a time-dependent wavefront phenomenon, the noninvasive detection of asynchronous polyphasic wall excursion as a marker of early ischemic injury may have important clinical implications for identifying ischemic areas that have maintained the ability to develop tension, indicating potentially viable myocardium, during the early course of impending infarction in man.

Nevertheless, a method used for quantitative assessment of changes during the course of acute myocardial ischemia or infarction must be capable of detecting the maximal functional abnormality associated with ischemia-induced wall motion disturbances. Our results have demonstrated that variables of regional left ventricular function that rely primarily on the determination of the amplitude of changes in endocardial excursion between two frames arbitrarily selected as end-diastole and end-systole are very likely to be misleading. Due to the potential of additional exaggerated late systolic shortening of hypoxic segments, the quantitation of wall motion in terms of hypokinesis or akin-nesis based on an end-diastolic–end-systolic comparison will miss the maximal functional abnormality associated with acute myocardial ischemia in man. These findings might also partially explain the inconsistent results of various controlled randomized trials in which the effects of thrombolysis on left ventricular function were examined by comparing regional wall motion disturbances assessed by a two-phase analysis early in the progress of acute myocardial infarction and during the chronic infarct period. Outward movement during the early phases of systole and exaggerated shortening during late systole in the ischemic segment have been qualitatively described previously. Very recently, Gibson et al. also demonstrated that asynchronous wall motion is more common than simple hypokinesis or dyskinesis during very acute infarction. However, integrative analysis for the first time enables the quantitative assessment of the temporal abnormal wall excursion pattern. Because wall motion disturbance is considered regardless of at what time point during the entire contraction sequence it occurs, this kind of analysis should provide the standard for the assessment of functional effects of any interventions aimed at altering regional left ventricular function during acute myocardial ischemia in man.

The integrative analysis revealed a larger absolute degree of wall motion abnormality associated with impending infarction than with chronic infarction. This finding is most likely attributable to alterations in the functional interactions between ischemic and normal left ventricular regions during infarct healing. Furthermore, acute myocardial ischemia is very likely to be associated with increased autonomic activity, which affects all measures of left ventricular function. However, function appears to be an important independent marker of the effects of ischemic disease, since it is function rather than histology that determines the effect of ischemia on the organism as a whole.

In summary, simple end-diastolic and end-systolic estimates of regional left ventricular performance mask a significant amount of information available from the observation of wall excursion throughout the entire contraction sequence and thus are prone to produce misleading results. Frame-by-frame analysis of regional systolic function discloses a hierarchy of levels of impairment of left ventricular function caused by ischemic injury: temporal asynchronous polyphasic wall excursion is the first manifestation of acute myocardial hypoxia, whereas chronic myocardial infarction is characterized by a completely passive, monophasic wall motion pattern of the scar region. This critical distinction in regional temporal wall excursion suggests the need for a comprehensive analysis of the entire contraction sequence to evaluate left ventricular systolic function. The method of integrative wall motion
analysis for the first time allows quantitative assessment of regional left ventricular asynchrony and thus might provide the necessary standard for evaluation of the effects of interventions on regional performance of ischemic myocardium in man.

References

Hierarchy of levels of ischemia-induced impairment in regional left ventricular systolic function in man.
A M Zeiher, H Wollschaeger, T Bonzel, W Kasper and H Just

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