Advantages and applications of the centerline method for characterizing regional ventricular function

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ABSTRACT We sought to identify theoretical advantages and applications of the centerline method for quantitative assessment of regional ventricular function. Motion was measured along 100 chords constructed perpendicular to a centerline drawn midway between the end-diastolic and end-systolic contours, and normalized for heart size. Abnormality was expressed in units of standard deviations from the mean motion in a normal reference population to indicate both the severity and significance of the wall motion abnormality. The mean abnormality averaged over 100 chords correlated highly with the area ejection fraction (r = .97). The centerline method uses a "sliding window" to measure motion where it is abnormal, because assessment of wall motion in predefined regions of the ventricular contour underestimates abnormality. From the 100 data points, the extent (% of contour) of regional abnormalities can also be determined. The severity of hypokinesis at the site of acute myocardial infarction correlated better with infarct size estimated from creatine kinase release (r = -.78) than did the ejection fraction or the circumferential extent of hypokinesis. Because the centerline method measures motion along locally determined vectors, and requires no apex, origin, coordinate system, or geometric reference figure, it can be applied to contours as dissimilar as the 60 degree left anterior oblique projection of the left ventricle and the 75 degree left anterior oblique projection of the right ventricle.


IN CURRENT CLINICAL PRACTICE, wall motion is assessed subjectively from contrast ventriculograms. The poor reproducibility of such qualitative evaluations has led to the development of quantitative methods that measure the extent of wall motion at 4 to 100 points around the endocardial contour.

More recent studies have questioned the assumptions on which many of these methods are based. The validity of radial methods has been challenged by reports showing that motion proceeds toward many points rather than a single point in the ventricular chamber, and that motion measurement along radii from a single origin is erroneous. Assessments of intraobserver and interobserver variability indicate that the apex is one of the least reliably visualized parts of the contour; the dependence of rectangular and radial methods on the apex as a landmark in defining a long axis may introduce error in wall motion measure-
fraction with infarct size, and (3) compare the centerline method and methods that measure motion in predefined regions or segments of the ventricle with respect to accuracy of measuring wall motion abnormalities. We report the results of this further validation of the centerline method, and present the advantages and disadvantages of the use of the centerline method in comparison with existing methods and its applications.

Methods

Patient populations. The mean and standard deviation for normal regional ventricular function were defined with the use of data from patients who underwent angiography for diagnosis of chest pain and who were found to have normal coronary arteries, no congenital, valvular, or electrocardiographic abnormalities, no history of cardiothoracic surgery, myocardial infarction, or sudden death, a left ventricular end-diastolic volume index of less than 110 ml/m², a left ventricular end-diastolic pressure of less than 15 mm Hg, and a left ventricular ejection fraction of greater than 55%. The territory of the ventricular contour assigned to each coronary artery was defined with the use of data from patients with single-vessel disease.

Ventriculograms of patients fulfilling these criteria were obtained from the following sources: (1) 30 degree RAO left ventriculograms (n = 52 normal, 123 single-vessel disease) from the University of Washington and affiliated hospitals by data base search, (2) 30 degree RAO/60 degree LAO biplane left ventriculograms (n = 32 normal, 62 single-vessel disease) from the Seattle VA Hospital by data base search, and from Dr. Mirle Kellett (Boston University), Dr. Joachim Schofer (University Hospital Eppendorf, Hamburg, W. Germany), and Dr. Hugh Smith (Mayo Clinic), (3) 15 degree RAO/75 degree LAO biplane right ventriculograms (n = 10 normal, 10 acute right coronary artery thrombosis) courtesy of Dr. Harald Becher (University Hospital Eppendorf, Hamburg, West Germany).

Regional wall motion in acute myocardial infarction was studied in patients who underwent angiography in the course of receiving intracoronary thrombolytic therapy. All 30 degree RAO and 30 degree RAO/60 degree LAO left ventriculograms and 15 degree RAO/75 degree LAO right ventriculograms were filmed by Drs. Detlef G. Mathey and Joachim Schofer at University Hospital Eppendorf, Hamburg, West Germany.

Cardiac catheterization. Ventriculography was performed after a bolus injection of contrast through a pigtail catheter and ventriculograms were recorded on cine film. All biplane ventriculograms were simultaneously acquired.

Analysis of ventricular function

Data acquisition. The cine film of the contrast ventriculogram is projected at a magnification of about two times. A normal, nonpostmortem sinus beat is selected, and the endocardial contours at end-diastole and end-systole are traced and entered into a VAX 11/750 computer with an x-y digitizer. Contour coordinate pairs are entered at a density of eight per inch and stored in binary files that can be input into programs for calculating left ventricular chamber volume (by the area-length method),24 ejection fraction, stroke volume and cardiac output, and/or regional wall motion. Clinical data such as the patient’s name, identification number, catheterization date, height and weight, the projection angle(s), the framing rate at which ventriculography was performed, the RR interval, and comments and the identity of the operator are also stored on this file. Absolute measurements of volume and wall motion can be made if a correction factor is also entered. The correction factor is determined from the measurements of a grid of known dimension filmed at the level of the patient’s mid chest with the x-ray equipment in the same position as during ventriculography and corrects for magnification and pin-cushion distortion.25 No attempt was made to realign to correct for the translational motion of the heart within the chest, since currently known methods are arbitrary and may introduce error.17, 26, 27

Measurement of wall motion by the centerline method. Motion is measured along 100 chords drawn perpendicular to a centerline constructed midway between the end-diastolic and end-systolic contours (figure 1, appendix 1). The measured motion of the 100 chords is normalized for heart size by dividing by the length of the end-diastolic perimeter. This results in a dimensionless shortening fraction, a linear equivalent of the volume ejection fraction. Since normal motion varies from chord to chord (figure 1, C),5, 7, 8, 12, 13 the normalized motion (M) at each chord i is converted into units of normal standard deviations (SD) from the normal mean for motion at each chord (N) (figure 1, D): Z = (M - N)/SD. This standardization allows comparison of the motion of different regions of the ventricle. Positive values indicate hyperkinesis; negative values, hypokinesis. The centerline method was originally derived from the algorithm used to measure dimensions of coronary artery stenoses.28

Measurement of the severity of regional wall motion abnormality. In coronary disease patients, the severity of hypokinesis is measured in the territory of the ventricle supplied by the stenosed artery. Each artery’s territory is defined as the set of contiguous chords the motion of which is significantly depressed (p < .05, one-way analysis of variance), compared with motion in the normal group, in patients with isolated stenosis of that artery. In the 30 degree RAO projection, the territories of the left anterior descending and right coronary arteries (LAD and RCA) extend from chords 10 to 66 and 51 to 80, respectively (figure 2).20 29 The population of patients with single-vessel disease used to define these artery territories included patients with (n = 59) and without (n = 64) prior infarction. However, the artery territories were primarily determined by the extent and severity of wall motion abnormality in the patients with infarction, and the wall motion plot for the group as a whole was similar to that for the subgroup with infarction.

In patients with stenosis of both the LAD and RCA, the overlap region was halved arbitrarily (table 1). In those with stenosis of the circumflex artery (CFX), hypokinesis was measured in the LAD territory if the dominance was right or if the myocardial infarction was anterior in location, and in the RCA territory if dominance was left or the infarction was inferior in location (figure 2). Hyperkinesis was measured in the wall opposite the ischemic region (table 1).

To filter out noise in the measurement, wall motion in each territory is computed by averaging the motion of chords lying in the most abnormally contracting 50% of the territory and is expressed in standard deviations/chord (SD/chord). When measured this way, the presence of hypokinesis more severe than 2 SD below normal distinguishes patients with coronary artery disease from those with normal arteries best: averaging over 75% or 100% of the artery territory is less sensitive due to inclusion of the border zone, and specificity is low with short regions because patients with normal coronary arteries may have short stretches of hypokinesis due to variability.27, 29

Measurement of the circumferential extent of wall motion abnormality. The percent of the endocardial contour with hypokinesis more severe than 1 SD below normal was calculated as the number of contiguous chords with motion below that threshold within the artery’s maximum territory (chords 5 to 85 for the LAD; 25 to 85 for the RCA). The circumferential extent of hypokinesis more than 2 or 3 SD below normal was similarly
determined. The extent of akinesis was calculated as the number of chords with absolute motion less than or equal to 0 (figure 3).

The location of wall motion abnormality is expressed as the chord number at the beginning and end of the abnormal region.

**Studies**

**Regional wall motion and global function.** The relationship between mean regional wall motion averaged over all 100 chords and global function was evaluated. Both the mean shortening fraction (motion normalized for heart size) and mean motion abnormality (expressed in SD from normal) were calculated and compared with the area ejection fraction computed from the areas within the end-diastolic (EDA) and end-systolic (ESA) endocardial contours as (EDA − ESA)/EDA.

**Evaluation of wall motion in predefined regions of the ventricle.** This study measured the variability in the location of the most hypokinetic region in 113 patients with acute anterior or inferior myocardial infarction and the effect of this variability on measurement of hypokinesia in five predefined regions of the contour, such as those used in the Coronary Artery Surgery Study. Wall motion in each of five regions of the left ventricular contour and the mitral valve was determined by averaging the motion abnormality, expressed in SD, of chords 1 to 16, 17 to 32, 33 to 48, 49 to 64, and 65 to 80, corresponding to the anterobasal, anterolateral, apical, diaphragmatic, and posterobasal regions and the mitral valve, respectively. A sliding window with a length of 16 chords (equal to one-fifth of the contour...
FIGURE 2. Location of hypokinesis resulting from stenosis of the coronary arteries in patients with single-vessel disease. The location of hypokinesis in patients with CFX stenosis depends on the dominance of the coronary artery system.

excluding the two valve planes) was applied to the contour to select the most hypokinetic one-fifth of the ventricle. The severity of hypokinesis in this segment was measured and compared with motion in the five predetermined regions by paired t test. The centerline method per se measures motion in the most abnormally contracting 50% of the artery territory, but a 16-chord window was used here for comparison with the five predetermined regions’ motion.

Correlation between regional wall motion abnormality and infarct size. Infarct size was estimated from serial determinations of creatine kinase in 34 consecutive patients with a diagnosis of acute myocardial infarction who underwent thrombolytic therapy with intravenous urokinase. Creatine kinase release was calculated with the use of individually calculated enzyme disappearance rates (K) and correlated with parameters of regional and global left ventricular function according to results of linear regression analysis.

Application of the centerline method to 60 degree LAO left ventriculograms. Wall motion was measured from biplane 30 degree RAO/60 degree LAO ventriculograms in 32 normal subjects. Variability in the motion (shortening fraction) of each chord in the normal subjects was calculated as the ratio of the standard deviation to the mean. Variability in the 30 degree RAO and 60 degree LAO projections was compared by use of the unpaired t test. The effect of coronary artery stenosis on motion in the LAO contour, and the territories corresponding to each coronary artery, were defined by comparing the motion in the normal subjects with the motion in patients with acute thrombosis of the LAD (n = 31), RCA (n = 16), and CFX (n = 15).

Application of the centerline method to evaluation of right ventriculograms. The normal extent of right ventricular wall motion was calculated from the biplane right ventriculograms of normal subjects in the 15 degree RAO and 75 degree LAO projections. Variability in normal right ventricular motion was calculated as the ratio of the standard deviation to the mean for motion at each chord in these normal subjects. Variability in the RAO and LAO projections was compared by use of the unpaired t test. The effect of acute inferior infarction on right ventricular wall motion was evaluated by comparing the motion in the 10 normal subjects with that in 10 patients with acute RCA thrombosis (unpaired t test).

Results

Regional wall motion and global function. There was a high correlation between the mean wall motion averaged over the 100 chords and the area ejection fraction, with a coefficient of r = .97. The correlation was the same for mean abnormality in motion expressed in SDs from normal, and for the mean fractional shortening, which is absolute chord motion normalized for heart size. The correlation of mean wall motion with the volume ejection fraction had a coefficient of .95. The

FIGURE 3. Determination of the circumferential extent of akinesis (top) and of hypokinesis more than 1, 2, and 3 SDs from normal (bottom).
close correlation between mean wall motion and the ejection fraction is explained by their mathematical relationship (appendix 2).

**Evaluation of wall motion in fixed regions of the 30 degree RAO left ventriculogram.** The one-fifth of the ventricular contour selected by the sliding window as the most hypokinetic segment in patients with anterior myocardial infarction varied in location over the anterior and apical sectors (figure 4). As a result, the magnitude of hypokinesis measured in the anterolateral and apical regions (−1.8 ± 1.2 and −2.3 ± 0.9 SD/chord, respectively) underestimated abnormality compared with measurement of hypokinesis in the most hypokinetic segment (−2.8 ± 0.9 SD/chord, p < .001 vs anterolateral, p < .001 vs apical sector, n = 29).

In patients with inferior infarction, the location of the most hypokinetic segment varied to a lesser extent, but the magnitude of its hypokinesis (−2.5 ± 1.1 SD/chord) was underestimated by motion in either the diaphragmatic or posterobasal regions (−1.0 ± 1.2 and −2.3 ± 1.2 SD/chord, respectively, p < .001 and p < .001 vs most hypokinetic segment, n = 24).

**Correlation with infarct size.** The relationship between left ventricular dysfunction measured more than 2 weeks after infarction and creatine kinase release was nonlinear (logarithmic) (figure 5). In patients undergoing reperfusion, creatine kinase release correlated with the severity of hypokinesis at the infarct site with a coefficient of r = −.80, a correlation better than that with the ejection fraction (table 2). The correlations were higher when only reperfused, nonrethrombosed patients were considered, because reperfusion alters the relationship between enzyme release and infarct size.33 The ejection fraction and the severity of hypokinesis correlated with each other (r = .77), but multivariate analysis to take this into consideration revealed that the partial correlation of enzyme release with the severity of hypokinesis remained higher (r = −.55) than the partial correlation of enzyme release with the ejection fraction (r = −.29).

For the population as a whole, creatine kinase release correlated better with the severity of hypokinesis than with the circumferential extent of hypokinesis. This was because the slope and intercept of the regression relating severity of hypokinesis to enzyme release was similar for anterior and inferior infarctions. However, the relationship between the extent of hypokinesis and enzyme release differed significantly between patients with anterior and inferior infarcts, as might be expected from the larger territory supplied by the LAD as compared with the RCA. When the patients were subgrouped by infarct artery, the correlation between enzyme release and extent of hypokinesis was similar to the correlation between enzyme release and severity of hypokinesis. Indeed, the two parameters were closely related to each other (r = −.83).

**Evaluation of 30 degree RAO and 60 degree LAO ventriculograms.** Variability in normal wall motion was higher in the 60 degree LAO than in the 30 degree RAO projections. Exclusion of the outflow tract in the LAO (figure 6) view, due to its highly variable length, reduced this variability, but not to the level measured in the RAO view (0.32 ± 0.08 vs 0.38 ± 0.09 in the LAO, n = 100 chords, p < .001). In both views variability was highest in chords overlying the mitral valve or adjacent to the aortic valve. Variability was also high at the apex in the RAO projection, and along the anterosetal wall in the LAO view. Figure 6 illustrates the territories of the LAD, RCA, and CFX, as defined by the wall motion in the patients with single-
vessel disease. The territories of the RCA and CFX both lie in the posterior wall, overlapping with each other and with the LAD territory. Because of this large overlap the effect of multiple stenosis could not be distinguished, and motion in patients with multivessel disease was analyzed in the same territories as in those with single-vessel disease (table 3).

As for the RAO projection, the severity of hypokinesis at the infarct site in the study patients correlated better with the creatine kinase release than did measures of the circumferential extent of hypokinesis or akinesis (table 4).

Right ventricular wall motion. The magnitude of wall motion in the right ventricle was similar in the 15 degree RAO (2.4 ± 1.4, n = 100) and 75 degree LAO views (2.6 ± 0.9, n = 100). Right ventricular wall motion was less than wall motion in the left ventricle (2.5 ± 1.2 vs 4.0 ± 1.1, p < .001, n = 200 chords in two views). Patients with acute RCA thrombosis had significant hypokinesis in the left lateral and diaphragmatic walls of the RAO view (chords 28 to 63, p < .05 compared with normal) (figure 7). Hyperkinesis was seen in the basal aspect of the posterior wall, although it was significant in only three chords (70 to 72). In the LAO view, patients who had suffered infarction had slight hypokinesis along the diaphragmatic surface and free wall (chords 35 to 100); significant hyperkinesis was present along the basal aspect of the septum (chords 13 to 17).

Discussion

Review of previously described methods. A variety of methods have been developed for the quantitative as-

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**TABLE 2**

Correlation between left ventricular (LV) function in the RAO projection and infarct size estimated from creatine kinase (CK) release

<table>
<thead>
<tr>
<th>Severity of hypokinesis in infarct region</th>
<th>CK release in all patients</th>
<th>CK release after reperfusion</th>
<th>p value</th>
<th>p value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Circumferential extent</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Hypokinesis below -1 SD</td>
<td>.67</td>
<td>.72</td>
<td>.001</td>
<td>.001</td>
</tr>
<tr>
<td>Hypokinesis below -2 SD</td>
<td>.57</td>
<td>.61</td>
<td>.001</td>
<td>.001</td>
</tr>
<tr>
<td>Hypokinesis below -3 SD</td>
<td>.45</td>
<td>.47</td>
<td>.01</td>
<td>.05</td>
</tr>
<tr>
<td>Akinesis or dyskinesis</td>
<td>-.42</td>
<td>.49</td>
<td>.05</td>
<td>.01</td>
</tr>
<tr>
<td>Ejection fraction</td>
<td>-.65</td>
<td>-.71</td>
<td>.001</td>
<td>.001</td>
</tr>
<tr>
<td>n</td>
<td>34</td>
<td>27</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

**FIGURE 5.** Correlation between the severity of hypokinesis at the infarct site measured at follow-up and infarct size assessed from serial creatine kinase determinations in patients who underwent reperfusion. The relationship is logarithmic.
assessment of regional wall motion. Many of the methods measure motion along hemiaxes evenly spaced along a long axis drawn from the aortic valve to the apex. However, this assumes homogeneous shortening of the long axis, which may not be valid since fiber shortening is reduced by ischemia, and may result in the artifactual appearance of motion in akinetic regions. Some investigators measure motion along radii; however, the assumption that motion proceeds toward a single point may be invalid. Selection of the geometric center of mass as the origin causes abnormality to be underestimated, perhaps because (1) the center of the ventricular cavity rather than the center of muscle mass is used to define the origin, so that regional differences in wall thickening or thinning during systole are not taken into consideration, or (2) the presence of compensatory hyperkinesis in the noninfarct region may shift the center of mass, artificially causing akinetic segments to appear contractile.

Furthermore, the apex is often used to define the rectangular and radial coordinate system, even though it is not an anatomic landmark and is the least reliably visualized part of the ventricular contour. To avoid these problems, methods of measuring regional wall motion along locally defined vectors have been developed.

However, all of these methods are subject to another cause of reduced sensitivity in measuring wall motion abnormality: intraobserver and interobserver variability in tracing the endocardial contour. Variability can be lessened by calculating wall motion over a region of the ventricle rather than at discrete points.

**TABLE 3**
Coronary artery territories of the left ventricle in the 60 degree LAO projection

<table>
<thead>
<tr>
<th>Artery of interest</th>
<th>Hypokinetic region</th>
<th>Hyperkinetic region</th>
</tr>
</thead>
<tbody>
<tr>
<td>LAD</td>
<td>50–100</td>
<td>20–49</td>
</tr>
<tr>
<td>RCA</td>
<td>19–67</td>
<td>68–100</td>
</tr>
<tr>
<td>CFX</td>
<td>38–74</td>
<td>75–100</td>
</tr>
</tbody>
</table>

**TABLE 4**
Correlation between wall motion in the LAO projection and infarct size estimated from creatine kinase (CK) release

<table>
<thead>
<tr>
<th></th>
<th>CK release in all patients value</th>
<th>CK release after reperfusion value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Severity of hypokinesis in infarct region</td>
<td>-.76 .001</td>
<td>-.75 .001</td>
</tr>
<tr>
<td>Circumferential extent</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Hypokinesis below -1 SD</td>
<td>.70 .001</td>
<td>.70 .001</td>
</tr>
<tr>
<td>Hypokinesis below -2 SD</td>
<td>.60 .001</td>
<td>.63 .001</td>
</tr>
<tr>
<td>Hypokinesis below -3 SD</td>
<td>.18 NS</td>
<td>.24 NS</td>
</tr>
<tr>
<td>Akinesis or dyskinesis</td>
<td>.36 NS</td>
<td>.44 .05</td>
</tr>
<tr>
<td>n</td>
<td>33</td>
<td>26</td>
</tr>
</tbody>
</table>
points, but this solution raises a new question: How shall the regions of the left ventricle be defined? There is variability in normal coronary anatomy, and in the location of stenoses along the coronary arteries. As our data show (see figure 6), the site of hypokinesis is also variable and frequently straddles adjacent regions, in which case the wall motion of neither region is representative of the full severity of the hypokinesis. The number of regions can be increased, but the resulting multiple data are unwieldy to interpret, since the magnitude of normal motion (and hence the threshold of abnormality) varies from region to region.

Advantages of the centerline method. To address these problems, the centerline method was developed. It incorporates features that provide advantages over other methods and that make it adaptable to a variety of applications. First, the conversion of motion measurements into units of standard deviations from normal provides a measure not only of the severity of abnormality but also of its significance, and expresses abnormality in all chords in equivalent units. This is important because it allows comparison between different regions of the same heart and between different hearts. Also, the motion of adjacent chords can be averaged, which reduces variability in the measurement. From a practical standpoint, these features yield one of the most important advantages of the method — the ability to express in a single parameter the severity, significance, and direction (i.e., hypokinesis vs hyperkinesis) of a wall motion abnormality. Although a standard for assessing methods of wall motion measurement is lacking, empirical evaluations indicate that motion measurement by the centerline method not only can distinguish normal subjects from patients with coronary artery disease, but also correlates with the severity of coronary artery stenosis with enough accuracy to define the threshold of significance. The accuracy of the centerline method is further verified by our results showing that the mean wall motion abnormality correlates well with the area ejection fraction, and indicates that the motion measured at each chord represents that chord’s fractional contribution to stroke area.

Second, the centerline method applies a “sliding window” to the ventricular contour in order to focus on and measure function in the most abnormally contracting part of the infarct artery territory or of the noninfarct region. This enables more accurate evaluation of the severity of wall motion abnormality, as shown by the present results, compared with traditional analysis in fixed regions of the contour. Nor does the “window” slide blindly — the limits of each search region are predefined, and the beginning and ending chord numbers indicate the location of the selected region.
Third, since motion is measured in the direction of a locally defined vector, the lack of a geometric reference figure makes the method equally well suited to the foreshortened 60 degree LAO view in which no apex can be identified. Since no central long axis or origin need be defined, the method is also applicable to the thin curved shape of the 75 degree LAO right ventriculogram. These features also make the centerline method suitable for other imaging modalities. For example, the centerline method has been used to measure regional wall motion from two-dimensional echocardiograms obtained during normal sinus rhythm and ventricular tachycardia in the short-axis view, and during rest and exercise in the subcostal four-chamber view. The method has also been applied to analysis of radionuclide ventriculograms. Because the chords are constructed from local contour data, the centerline method can also be used to measure wall thickness and percent thickening or thinning from two-dimensional echocardiograms or from contrast studies modified with digital subtraction techniques to enhance delineation of the epicardial border.

Limitations of the centerline method. One of the unresolved questions in wall motion analysis is whether and how to correct for the translational motion of the heart within the chest and so distinguish it from the inward (or outward) motion of the ventricular walls. Many methods of realigning the end-diastolic and endsystolic contours have been devised, but all are empiric and offer no theoretical advantage over not realigning. Our own studies show that realignment by superimposition of long axes worsens variability rather than reducing it. However, if a physiologically valid method of realignment is developed, then it can be easily added to the centerline method, after redefining the normal mean and standard deviation and artery territories.

Alternate approaches to wall motion analysis. Recently, it has been suggested that wall motion be expressed in terms of its probability of belonging to the normal population, as determined by discriminant analysis. We do not use this approach because (1) the threshold for normality would then depend on both the normal reference group selected and the coronary disease population, and the mean and standard deviation values may therefore vary widely depending on the proportion of patients with myocardial infarction, and (2) it would require separate threshold determinations (and separate reference populations) for assessing hyperkinesis and hypokinesis.

The percent of the ventricular contour having wall motion abnormality appears to be a useful parameter of regional ventricular function. However, measurement of the circumferential extent of hypokinesis or akinesis has several disadvantages not present when measuring the severity of hypokinesis. First, the effects of multivessel coronary artery disease and/or previous infarction cannot be distinguished from the effect of the infarct of interest. Also, the hypokinetic segment is significantly longer for anterior than inferior infarctions, resulting in greater variability, unless these subgroups are separately studied. Third, and most importantly, measuring the length of the hypokinetic region mass provides information on the function of the border zone at the periphery of an infarct, but the information may be misleading because the function of the border zone may be influenced by changes in the function of the neighboring “normal” zone (figure 8). Finally, as demonstrated in the present report, our data indicate that the severity of hypokinesis in the stenosed artery’s territory is the parameter that most accurately reflects the degree of coronary stenosis and best correlates with infarct size as estimated from creatine kinase release. It is not valid, however, to assume from our data that regions displaying hypokinesis are infarcted, since hypokinesis sometimes develops in normally perfused myocardium adjacent to areas of ischemia. It should be noted that simply scoring wall motion by defining hypokinesis or akinesis as “present” if motion falls below an arbitrary threshold limits interpretation of the data and decreases accuracy because variability in the measurement could push a value across a threshold in the absence of real abnormality or hide a significant change because it fails to cross the threshold.

As suggested by Shepertycki et al., appreciation of abnormal motion may be enhanced by displaying the patient’s motion against the predicted normal motion. This can be easily performed by the centerline method by altering the length of the patient’s motion chords to equal the normal mean motion of that chord and displaying the chords together with the patient’s end-diastolic and end-systolic contours (figure 7). However, the method of displaying chord motion in SDs (figure 1) indicates clearly the location, severity, and significance of regional wall motion abnormalities.

One difficulty in evaluating change in ventricular function between serial studies is that alterations in the patient’s hemodynamic status may also affect function. Therefore, some investigators have normalized regional function by the ejection fraction to reduce variability due to differences in hemodynamic status. However, in the absence of hemodynamic changes this approach can cause wall motion measurement in the
region of interest to be distorted by changes in the function of the opposite wall (appendix 3).

Clinical applications. Analysis of the severity of regional wall motion abnormality is important in evaluating patients with coronary artery disease, since the not infrequent development of compensatory hyperkinesis in regions distant from the site of acute ischemia or infarction makes the ejection fraction relatively insensitive with regard to reflecting hypokinesis in the ischemic region. As a result, the effect of therapeutic interventions in salvaging ventricular function should be evaluated by measuring regional function.20, 51 Measurement of regional wall motion abnormality has also been found to be a better prognostic indicator than the ejection fraction in most studies in which they were compared.52-55 and provides additional information in patients with other cardiac diagnoses as well. For example, congestive heart failure due to chronic valvular regurgitation is associated with regional hypokinesis in the absence of coronary artery disease.56 Also, the presence of segmental wall motion abnormality indicates a better prognosis than diffuse hypokinesis in patients with congestive cardiomyopathy.57

The centerline method requires no complex programming; it can be implemented on a microcomputer. Because the centerline method requires no geometric reference figure or coordinate system, it can be applied to any cardiac chamber or imaging modality. Motion is initially measured at 100 chords, but the result of the final analysis is a single parameter that expresses both the severity and the significance of regional wall motion abnormality and that correlates highly with infarct size in patients with myocardial infarction.

Appendix 1: Computer implementation of the centerline method

The centerline method refers to the definition of a sequence of points halfway between two ventricular contours representing the endocardial surfaces defined by contrast ventriculography. Perpendiculars to the line at each point are then constructed, and the intersections of these perpendiculars with the two contours are determined. The distance between the intersections along each perpendicular is considered the extent of local wall motion.

(1) The end-diastolic and end-systolic borders (hereafter re-
ferred to as \( B1 \) and \( B2 \) are represented as a sequence of \((x, y)\) coordinate pairs, or points. The number of points are not necessarily the same, but it is assumed that the points are less than some defined distance apart (in our case, about 1.5 mm).

(2) The longer of \( B1 \) and \( B2 \) is highly smoothed to form \( S1 \). The smoothing consists of replacing each point with an arithmetic average of a maximum of 20 points on each side of the given point. \( S1 \) is used as an initial indication of direction of motion.

(3) \( S1 \) is divided evenly (by linear interpolation) into about 200 points.

(4) For each point on \( S1 \), a perpendicular \( P \) is constructed to the tangent of a circle passing through the given point and a point on each side.

(5) The intersections of \( P \) with \( B1 \) and \( B2 \) (called \( I1 \) and \( I2 \)) are determined, and the midpoint \( C \) of the line \( I1-I2 \) is determined. Points on \( B1 \) and \( B2 \) lying between digitized coordinate pairs are obtained by linear interpolation. \( P \) may intersect more than once with \( B1 \) and \( B2 \), so the program limits the search for intersections to a fraction (currently 1/6 of the entire border, beginning with the last intersection found. The goal of the centerline method is point-by-point wall motion measurement, so no perpendicular is allowed to "cross over" a previous perpendicular. If no intersection can be found with either border, the previous intersection with that border is used as the present intersection \( I1 \) or \( I2 \).

(6) The series of points \( C \) comprise centerline \( C1 \).

(7) \( C1 \) is smoothed to make \( S2 \). Half as many points are averaged during smoothing as were used to make \( S1 \).

(8) \( S2 \) is divided evenly into about 200 points.

(9) A new centerline \( C2 \) is computed by processing \( S2 \) as for \( S1 \). A sequence of motion segment lengths (\( M \)) or "chord lengths" is also computed as the length of line \( I1-I2 \) for each point on \( C2 \). Positive motion is defined as \( I1-I2 \) being inside the closed curve defined by \( B1 \). Motion is set negative if \( I2 \) is outside \( B1 \). The intersections are also saved.

The result of the above processing is the "centerline" \( C2 \), the extent of motion \( M \) for each point in \( C2 \), and the set of border intersections \( I1 \) and \( I2 \). The computer method allows processing of additional passes by renaming \( C2 \) to \( C1 \) and performing the last three steps above. Additional passes improve the results in 1% of cases. Dyskinetic regions are easily handled: the centerline passes through the intersections of the end-diastolic and end-systolic contours, and chords in the aneurysm proceed outward from the end-diastolic contour, and this motion is assigned a negative value. Since the delineation of the centerline is the rate-limiting step, the coordinates of the centerline and the 190 or so chords are saved in a binary file that is compact and inexpensive to store. A subsequent program derives 100 chords evenly spaced around the centerline using linear interpolation of \( C2 \) and \( M \).

Motion is calculated as the absolute length of each chord in centimeters after correction for magnification and distortion, and stored in a text file. These text files can be printed out, or input into a graphics program, data base, or statistical analysis package.

**APPENDIX 2.** Relationship between mean wall motion and global function.

\[
\begin{align*}
\tau_s &= \text{radius to S, the end-systolic contour} \\
\tau_D &= \text{radius to D, the end-diastolic contour} \\
\text{Dashed line} &= \text{centerline with radius } \tau_C = (\tau_s + \tau_D)/2 \\
\text{Mean chord motion} &= \tau_D - \tau_S \\
\text{Centerline length} &= 2\pi\tau_C = 2\pi(\tau_D + \tau_s)/2 = \pi(\tau_D + \tau_s) \\
\text{EDA} &= \text{end-diastolic area} = \pi\tau_D^2 \\
\text{ESA} &= \text{end-systolic area} = \pi\tau_s^2
\end{align*}
\]

**APPENDIX 3.** Effect of normalization by ejection fraction on measurement of regional wall motion. Let the end-diastolic contour of ventricles \( A \) and \( B \) be represented by circles of equal radius = \( r_D \).

1. End-diastolic area \( A = \pi r_D^2 \)
2. \( \text{ESA}_A = \pi r_s^2 \)
3. \( r_s < r_D \)
4. \( \text{ESA}_A > \text{ESA}_B \)

(5) Area ejection fraction = area EF = (EDA–ESA)/EDA

(6) Area EF \(_A\) < area EF \(_B\)

(7) Normalized motion, \( N_i = M_b/\text{area EF}_i \)

(8) If motion at chord \( i \) in \( A \) (\( M_{i,A} \)) = motion at chord \( j \) in \( B \) (\( M_{j,B} \))

(9) Then, \( N_{i,A} > N_{j,B} \)

The illustration provides one example of the results of normalization by area ejection fraction. However, equivalent results will derive from any contours in which equations 6 and 8 are true.

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