Detection of Hypokinesis by a Quantitative Analysis of Left Ventricular Cineangiograms

By Richard F. Leighton, M.D., Sharon M. Wilt, A.A., and Richard P. Lewis, M.D.

SUMMARY
A method for detecting left ventricular hypokinesis is proposed. It involves superimposition of left ventricular silhouettes traced from 30 degree right anterior oblique cineangiograms, correcting for thoracic cage motion, descent of the aortic valve and rotation of the apex. Normal values for the percent of systolic motion of seven endocardial segments have been established from measurements in 20 patients, permitting a statistical definition of hypokinesis. The use of this quantitative method has been compared with visual inspection of ventriculograms (qualitative method), resulting in differences in definition of hypokinetic segments by the two methods in 13 of 16 patients with coronary heart disease. When the quantitative method was used, only one hypokinetic segment was found which did not correspond to an obstructive coronary lesion while six such segments were defined using the qualitative method. In four patients segments thought to be hypokinetic (qualitative method) appeared to be akinetic (quantitative method). In six patients with cardiomyopathy, thought to have diffuse hypokinesis, all seven left ventricular segments were hypokinetic in only three patients. The use of a quantitative method appears to be essential to the proper interpretation of left ventricular wall motion and particularly to the detection of hypokinetic segments.

Additional Indexing Words:
Coronary heart disease Cardiomyopathy Left ventricular wall motion

REGIONAL ABNORMALITIES of left ventricular contraction have been shown to occur commonly in patients with coronary heart disease.1-6 These abnormalities in wall motion have been shown to correlate grossly with electrocardiographic evidence of myocardial infarction,1,2 with the demonstration of regional abnormalities in myocardial lactate production,1,4 and with the sites of coronary obstructive lesions.1,3 While left ventricular contractile abnormalities cannot be demonstrated in all patients with significant coronary artery disease, their incidence has been shown to increase with the severity of angiographically demonstrated coronary disease.8 The precise identification of regional contraction abnormalities depends on the use of a method which will separate normal motion from abnormal left ventricular wall motion more reliably than does the subjective interpretation of the left ventricular cineangiogram. While the need for such a method may be less when a gross abnormality of contraction, such as akinesis or dyskinesis is present, it is greatest in detecting decreased amplitude of wall motion or hypokinesis. The superimposition of end-diastolic and end-systolic left ventricular silhouettes traced from cineangiographic frames taken in the 30° right anterior oblique position appears to be the most satisfactory method for demonstrating the presence of regional contraction abnormalities. Although this technique has been used by previous investigators,1,3,7-9 a uniform method for superimposing left ventricular silhouettes has not been adopted. In our experience such a method should account for three types of motion which may cause misinterpretation of left ventricular contractile movements: 1) thoracic cage motion; 2) systolic movement in an apical direction of noncontracting basal structures (the aortic and mitral valves); and 3) systolic rotation of the left ventricle, usually evident in the 30°RAO projection as a lifting up of the apex.

The purpose of this study is to report the use of a method of superimposing silhouettes which we believe corrects for all three types of motion and thus permits the establishment of normal values for the extent of regional left ventricular contractile motion. This normal data may then be used in the detection of regional contraction abnormalities, particularly hypokinesis.

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Method

The patients selected for this study were taken from a larger group who underwent left ventricular and selective coronary cineangiography for evaluation of chest pain. Three groups of nonconsecutively studied patients were selected on the basis of the angiographic appearance of the left ventricle seen in the 30° right anterior oblique (RAO) position. The angiograms included in this study were ones in which the left ventricular walls were clearly outlined by contrast material so that silhouettes could be traced. The cycles analyzed were sinus beats which did not follow premature ventricular contractions. Ventriculograms which showed obvious aneurysm formation, defined as a bulge on the end-diastolic silhouette, were excluded.

Twenty patients had angiographically normal coronary arteries with normal values for left ventricular volumes, ejection fraction and end-diastolic pressure (table 1). For the purposes of this study they were considered to be normal.

Six patients with physical, electrocardiographic and radiologic evidence of cardiomyopathy had apparent diffuse hypokinesis defined as a generalized, fairly uniform decrease in the amplitude of left ventricular wall motion. Sixteen patients with angiographically proven significant coronary artery disease (at least one stenosis in a major branch of 70% or greater) had apparent regional hypokinesis defined as a localized decrease in the amplitude of left ventricular wall motion. This phenomenon has also been termed asynergy.1,4

Left ventricular cineangiography was performed during retrograde left heart catheterization with the patient in the 30° RAO position using 36 to 42 ml of 90% hypaque, injected during a 3-4 sec interval. Films were exposed at a rate of 60 frames/sec with a 35 mm Arriflex cine camera mounted on a Siemens 10-inch image intensifier. The left ventricular injection was done just prior to selective coronary cineangiography. The patient’s electrocardiogram and a cinemarker indicating each frame and the duration of the injection were recorded on high-speed photographic paper in an Electronics for Medicine DR-12 recorder.

Each ventriculogram was observed for the pattern of contraction when projected both rapidly and in a slow frame-by-frame sequence. The outlines of the ventricle at the onset of ejection (OE) and at end-systole (ES) were traced from the projected images of the cinefilm, superimposed on the same paper. OE was taken from the frame just prior to the onset of uniform inward motion of the ventricular walls. ES was taken from the last frame which showed uniform inward motion of the walls. The selection of frames was made following a review of the cineangiograms by two of the three authors. The differences in frame selection between observers has been previously tested in a blinded fashion and shown to be a difference of less than one frame for each selection.40

Since the angiographic table could be moved during filming while the image intensifier and attached camera remained stationary, the spine or a rib margin was drawn on each frame that was traced to assure that the patient’s chest had not moved during the filming. Such movements rarely occurred. The edge of the diaphragm was also traced to exclude gross respiratory movements. No attempt was made to superimpose outlines of the aortic valve or any other basal structures on the tracings.

In correcting for rotation of the ventricular apex it was assumed that no rotation of the ventricular base, aortic or mitral valves occurred in the 30° RAO plane. A longitudinal axis (L1) of the ventricular silhouette on the OE frame was drawn by trial and error through the apex so that it divided the ventricle into two equally planimetered areas (fig. 1). For each ventricle this line could be selected after two or three attempts. The line was considered to be satisfactory if the two planimetered ventricular halves came within one square centimeter of each other. A longitudinal line (L2) was then drawn through the apex on the ES tracing, intersecting L1 at the point where L1 crossed the margin of the ES ventricular silhouette (fig. 2). The point of intersection of L1 and L2 always occurred on some portion of the margin of the aortic valve complex. The angle between L1 and L2 was measured in degrees. The silhouettes were then re-aligned so that L1 and L2 were superimposed and the silhouettes were retraced (fig. 2). In order to test the agreement between different observers in selecting the apex, two of the authors marked the apex at OE and at ES in the 20 normal left ventricular tracings.

Four lines were then drawn perpendicular to the longitudinal axis, dividing L2 into five equal segments. These perpendicular lines were then extended to intersect the superimposed OE silhouette at eight points. The distance along each of the eight hemiaxes from the OE position of the ventricle to its ES position was measured and expressed as a percentage of the distance from the OE position to L1, hence as a percent of systolic motion for each endocardial segment. The distance from the OE position of the apex to the ES position of the apex was measured and expressed as a percentage of the distance from the OE position of the apex to the most apical perpendicular line (fig. 2). The percents of systolic motion for each of nine endocardial segments (four on the antero-lateral wall, four on the infero-posterior wall and one for the apex), were averaged for the

Table 1

<table>
<thead>
<tr>
<th>Parameters of Left Ventricular Function in Normal Patients (n = 20)</th>
<th>Average</th>
<th>Range</th>
</tr>
</thead>
<tbody>
<tr>
<td>End-diastolic volume</td>
<td>68 ml/m²</td>
<td>49 - 94</td>
</tr>
<tr>
<td>End-diastolic pressure</td>
<td>6 mm Hg</td>
<td>2 - 14</td>
</tr>
<tr>
<td>Ejection fraction</td>
<td>0.68</td>
<td>0.60 - 0.78</td>
</tr>
</tbody>
</table>

Figure 1

Left ventricular silhouettes from two normal patients traced at the onset of systole. On the left in a vertically oriented ventricle, a longitudinal axis drawn from apex to the mid-point of the aortic valve divides the ventricle in half. On the right in a horizontally oriented ventricle the dashed line to the mid-point of the aortic valve departs from the solid line which divides the ventricle into two equally planimetered halves.

Circulation, Volume 30, July 1974
20 normal patients and treated statistically to establish standard deviations from the means.

Ventricular volumes and ejection fractions were calculated by the area-length method as previously described. 10-13

Left ventricular pressures were measured using fluid-filled catheters and Statham strain gauge transducers (P23 Db series). End-diastolic pressure was read from photographic recordings of high-amplification pressure tracings.

Results

The angle between L1 and L2, considered to be an angle of rotation in the 30° RAO plane, averaged 2.7 degrees in the 20 normal patients (range 0 to 9.2). In the patients with coronary disease this angle averaged 5.0 degrees (range 2.2 to 8.3), and in those with cardiomyopathy 1.9 degrees (range 0 to 5.7). Thus, although there was considerable individual variation, this angle tended to be greatest in the patients with coronary heart disease and least in those patients with cardiomyopathy.

In the normal patients the aortic valve descended an average of 15.4 ± 3.5 mm during systolic ejection, a significant change, $P < 0.001$.

Diaphragmatic motion during systole occurred in only six of the 42 patients. Upward motion occurred in five of the normal patients and downward motion occurred in one patient with cardiomyopathy. Such motion was small in amplitude, averaging 2.8 mm. In the normal patients there was no correlation between degree of left ventricular rotation (av. 2.2 degrees) and the presence or absence of diaphragmatic motion.

The average differences between observers' selection of apical points for construction of longitudinal axes in all 42 patients were small, 1.4 ± 0.30 mm at OE and 0.5 ± 0.14 mm at ES.

The percent of systolic motion with two standard deviations from the mean for the apex and the eight hemiaxes, before and after correction for rotation are shown in figure 3. While systolic motion was of fairly uniform amplitude along the antero-lateral wall and in the most apical segment of the inferior wall, it diminished progressively along the inferior wall as the base was approached.

The most basal portion of the inferior wall and the apex showed such variation in amplitude of motion that they were excluded when the normal data were used to define hypokinesis in the patients with heart disease.

Correcting for rotation tended to increase the percent of systolic motion in the antero-lateral wall, while diminishing motion in the inferior wall. The most striking differences were noted in the most apical portions of both walls, where correcting for rotation tended to equalize the amplitudes of systolic motion.

The normal values for segmental systolic motion were then used to define hypokinetic segments in the patients with heart disease. Hypokinesis was originally diagnosed in these patients by a qualitative interpretation of the left ventricular cineangiogram. This method, referred to in table 2 as the qualitative method, involved superimposition of ventricular silhouettes but differed from the quantitative method in that no correction for rotation was made and hemiaxes were not drawn. Determination of the presence and location of hypokinetic segments by this qualitative method was therefore based on visual inspection of the superimposed silhouettes. By contrast, hypokinetic segments were defined with the quantitative method as areas where the percent of systolic motion was less than two standard deviations from the normal mean.

The data in table 2 compare the segments of hypokinesis defined by the two methods to the sites of

![Figure 2](image_url)

Superimposed ventricular silhouettes from a normal patient. On the left the angle of rotation between L1 and L2 is shown. On the right the silhouettes have been realigned so that L1 and L2 are superimposed. The solid hemiaxes divide the end-systolic longitudinal axis into four equal lengths and define eight segments on the antero-lateral and inferior walls. By contrast the dashed hemiaxes divide the silhouette at onset of systole into four equal lengths, defining segments which have been examined by others.

![Figure 3](image_url)

Average percent systolic motion with 2 standard deviations of each left ventricular segment for the 20 normal patients, indicated around the ventricular silhouettes of one normal patient. Values on the left are before and on the right after correction for rotation.
coronary artery obstruction in the 16 patients with coronary heart disease.

In the first five patients use of the quantitative method resulted in the addition of one or more hypokinetic segments on the inferior wall. In patient 6 an area on the antero-lateral wall previously thought to be hypokinetic appeared to contract normally. In patients 7, 8, and 9 use of the quantitative method resulted both in the addition of hypokinetic segments on the inferior wall and in the deletion of areas on the antero-lateral wall. In patients 10, 11 and 13 a similar pattern of change was observed but in addition, in these patients as well as in patient 12, areas previously thought to be hypokinetic appeared akinetic, using the quantitative method. An example of this phenomenon is shown in figure 4. In patients 14, 15 and 16 no change in hypokinetic segments determined by the two methods was noted.

Thus the segments where the presence or absence of hypokinesia changed most often by using the quantitative method were in the most apical portions of both walls (8 changes for area 5, 5 changes each for areas 3 and 4).

Patients 7, 8, 10 and 13 did not have significant left anterior coronary lesions and use of the quantitative method eliminated segments in the antero-lateral wall previously thought to be hypokinetic. Thus use of the qualitative method resulted in the definition of hypokinetic segments for which no corresponding coronary lesion could be found six times while this occurred only once using the quantitative method. In addition in patients 7 and 8 who had posterior coronary lesions hypokinetic inferior wall segments, previously thought to be normal, were defined by the quantitative method.

When the percent of systolic motion for all seven segments was averaged in these patients and correlated with ejection fraction (fig. 5), a significant correlation was found, \( r = 0.92 \).

The mean percent of systolic motion also correlated well with ejection fraction in the six patients with cardiomyopathy, \( r = 0.99 \) (fig. 5). In these patients, thought to have diffuse hypokinesia involving all seven segments, application of the quantitative

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**Table 2**

<table>
<thead>
<tr>
<th>Patient</th>
<th>Hypokinetic segments</th>
<th>Coronary artery lesions, % obstruction</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Qualitative method</td>
<td>Quantitative method</td>
</tr>
<tr>
<td>1. R.G.</td>
<td>7</td>
<td>5,6,7</td>
</tr>
<tr>
<td>2. R.B.</td>
<td>6</td>
<td>6,7</td>
</tr>
<tr>
<td>3. J.S.</td>
<td>7</td>
<td>5,6,7</td>
</tr>
<tr>
<td>4. D.L.</td>
<td>6,7</td>
<td>5,6,7</td>
</tr>
<tr>
<td>5. S.R.</td>
<td>6,7</td>
<td>5,6,7</td>
</tr>
<tr>
<td>6. G.M.</td>
<td>3,4,7</td>
<td>3,7</td>
</tr>
<tr>
<td>7. P.S.</td>
<td>4,6</td>
<td>7</td>
</tr>
<tr>
<td>8. F.M.</td>
<td>3</td>
<td>7</td>
</tr>
<tr>
<td>9. E.S.</td>
<td>2,3,6,7</td>
<td>1,2,5,6,7</td>
</tr>
<tr>
<td>10. J.W.</td>
<td>3,4,6,7</td>
<td>5,6,*7</td>
</tr>
<tr>
<td>11. W.C.</td>
<td>3,4,6,7</td>
<td>5,6,*7</td>
</tr>
<tr>
<td>12. B.J.</td>
<td>5,6,7</td>
<td>5,*6,<em>7</em></td>
</tr>
<tr>
<td>13. R.M.</td>
<td>3,4,7</td>
<td>5,6,7*</td>
</tr>
<tr>
<td>14. D.K.</td>
<td>7</td>
<td>7</td>
</tr>
<tr>
<td>15. T.B.</td>
<td>6,7</td>
<td>6,7</td>
</tr>
<tr>
<td>16. W.M.</td>
<td>5,6,7</td>
<td>5,6,7</td>
</tr>
</tbody>
</table>

*Indicate akinetic segments.

---

**Figure 4**

Superimposed left ventricular silhouettes from the 30 degree RAO position in a patient with obstructive left posterior and right coronary artery disease. The solid line indicates onset of ejection, the dashed line end-systole. With aortic valve outlines superimposed there appears to be considerable contractile motion in the inferior wall with akinesis in the high antero-lateral wall. When the aortic valve is allowed to descend and rotation is corrected, anterior motion improves and akinetic areas are evident inferiorly.

*LEIGHTON, WILT, LEWIS*
QUANTITATIVE DETECTION OF HYPOKINESIS

Figure 5

Correlation of ejection fraction with mean percent systolic motion (average of motion for segments 1 through 7), on the left in the 12 patients with coronary heart disease and regional hypokinesis; (patients with akinetic segments excluded), on the right in patients with cardiomyopathy.

The method revealed some segments of normal motion in three patients while the other three patients, who had lower ejection fractions and no apical rotation, remained hypokinetic in all seven segments (table 3). With one exception, the segments which moved normally by the quantitative method, were all on the antero-lateral wall.

Discussion

There are several methodologic points that should be considered in the proper superimposition of traced left ventricular silhouettes in order to demonstrate regional contraction abnormalities. Since the patient’s chest may move during filming, a fixed point of reference external to the heart is necessary. Such motion may result from breathing, from movement of the patient on the table or from movement of the table itself. Since the latter is possible in our angiographic system while the position of the image-intensifier and attached camera are fixed, it appeared more logical to us to adopt McDonald’s method of tracing the spine or ribs7 rather than employing radiopaque markers on the angiographic apparatus as others have done.4,8

This problem is not really answered by the positioning of a catheter in the coronary sinus as Snideman and his colleagues have done,4 since the catheter will move as a part of cardiac motion. While Chaitman and his colleagues8 may be correct in their assertion that diaphragmatic motion may cause apparent upward rotation of the left ventricular apex, this did not occur in our patients where diaphragmatic motion was of small magnitude.

The selection of angiographic frames for analysis has been a reproducible process in our hands. Thus we feel selections may be made from the projected cineangiograms alone as long as PVCs are excluded, without having to make simultaneous external graphic recordings as others have done.7 We have chosen the onset of ejection rather than end-diastole to trace the ventricular silhouette since this permits the timing of wall motion abnormalities during systolic ejection. While there is some wall motion in the isovolumic contraction period, this motion has appeared to us and to others19 to be negligible.

In superimposing left ventricular silhouettes we feel it is important not to consider any basal structure as a fixed reference point. As pointed out by McDonald7 the aortic and mitral valves move toward the apex during ejection and this motion appears to be consistently present in our normal patients. Thus while others have shown aortic valve descent in describing their method of superimposing silhouettes1,9 there has been a tendency to superimpose aortic valve outlines when contraction abnormalities are displayed.1,3,11-19 Fixation of the junction of aortic and mitral valves14 seems to us tantamount to superimposing aortic valves. While it is granted that this junction does not contract it seems apparent that it is pulled toward the apex during ejection, along with the aortic and mitral valves. Failure to fix the basal structures need not interfere with the drawing or superimposition of long axes in an attempt to correct for ventricular rotation.

The practice of drawing ventricular long axes through the mid-point of the aortic valve1,3,9,14,18 does not account for variance among normal patients in left ventricular position and hence in the angle of junction of aortic valve and ventricle. While drawing the long axis through the mid-point of the aortic valve usually divides the ventricle in half in patients with vertically oriented ventricles, it tends to divide the ventricle asymmetrically in horizontally oriented ventricles (fig. 1). Since this axis provides a reference for drawing perpendicular hemiaxes, it seemed more accurate to us to draw a longitudinal axis which would divide the ventricle into symmetrical halves as long as aneurysmal ventricles were excluded. It does require the more laborious use of a planimeter and relies on

Table 3

Patients with Cardiomyopathy

<table>
<thead>
<tr>
<th>Patients</th>
<th>Segments with normal motion</th>
<th>Degrees of rotation</th>
<th>Ejection fraction</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. T.H.</td>
<td>2,3,4</td>
<td>2.7</td>
<td>0.49</td>
</tr>
<tr>
<td>2. R.T.</td>
<td>1,2,4</td>
<td>3.1</td>
<td>0.37</td>
</tr>
<tr>
<td>3. G.N.</td>
<td>1,3,4,7</td>
<td>5.7</td>
<td>0.55</td>
</tr>
<tr>
<td>4. D.C.</td>
<td>0</td>
<td>0</td>
<td>0.15</td>
</tr>
<tr>
<td>5. F.R.</td>
<td>0</td>
<td>0</td>
<td>0.33</td>
</tr>
<tr>
<td>6. C.H.</td>
<td>0</td>
<td>0</td>
<td>0.18</td>
</tr>
</tbody>
</table>

Circulation, Volume 50, July 1974
selecting a fixed point at the apex, but our variance between observers in selecting this apical point has been small.

The average angle of ventricular rotation in the 30° RAO projection in our normal patients is rather small, in keeping with previous observations. We would agree with Sniderman and his colleagues, however, that this rotation should be corrected since it varies from patient to patient and may be large. Correction for rotation in our normal patients tended to equalize the percent of motion observed in the apical segments of the antero-lateral and inferior walls. More importantly, this correction was probably the main factor in improving the fit of hypokinetic segment to coronary artery obstruction in our patients with coronary heart disease where the superimposed silhouettes had been interpreted visually, since the usual change was an increase in apparent antero-lateral wall motion and a decrease in inferior wall motion. The finding of least apical rotation in patients with cardiomyopathy and severe left ventricular dysfunction is in keeping with the relative lack of apical motion in hypertrophied and dilated ventricles which has been ascribed to alteration in the distribution of circular and spiral myocardial fascicles.

The drawing of hemiaxes appears to us to be more appropriate than measuring planimetered regional areas since our object has been to detect differences in segmental wall motion rather than changes in area. While the selection of comparable endocardial segments in small, vigorously contracting ventricles and in large, poorly contracting ventricles has obvious limitations, a method for drawing hemiaxes should be applicable to both situations. When we attempted to use the method of dividing the longitudinal axis of the end-diastolic silhouette by three equally spaced perpendicular lines in our normal ventricles, the most apical perpendicular usually fell at the tip of the end-systolic apex (fig. 2), which would have resulted in uninterpretable values for percent of systolic motion along the two apical hemiaxes. Drawing four equally spaced perpendiculars to the end-systolic longitudinal axis avoids this problem and fortuitously provides four hemiaxes which are comparable to those previously used, while providing two additional ones (fig. 2).

This issue was avoided by Sniderman and his colleagues by dividing both end-diastolic and end-systolic longitudinal axes by three equally spaced perpendiculars and by establishing a degree of motion between the intersected endocardial segments. This method has the advantage of not assuming that the same endocardial point is intersected at end-diastole and at end-systole but seems to us to have the distinct disadvantage of not permitting a clear display of contraction abnormalities by viewing the superimposed silhouettes.

While only seven of our nine hemiaxes proved useful in defining segmental wall motion, this method still provided more points along the ventricular silhouette than have been previously examined. The extreme normal variability in shortening at the postero-basal segment may be due to the inclusion of portions of the descending mitral valve in this area. The variance of apical shortening has been noted previously. As a result the diagnosis of hypokinesis in these two segments should probably be avoided and they should be virtually akinetic in order to define a contraction abnormality.

The proposed method of superimposing silhouettes has established normal values for regional left ventricular wall motion for our laboratory which can be used to define the presence of hypokinetic segments in patients with heart disease. While this method may be too laborious to gain more widespread use, it should be possible to construct a computer program which would include its basic tenets. Without normal values the diagnosis of diffuse hypokinesis remains a subjective interpretation of the extent of wall motion, corroborated by the finding of a depressed ejection fraction. While this may not be a problem when ejection fraction is markedly depressed, it is a difficult interpretation when the ejection fraction is borderline or only slightly depressed. In this situation a statistical definition of hypokinesis may be of most value. The demonstration that the mean degree of segmental wall motion correlates with ejection fraction in diffusely hypokinetic ventricles provides an additional interrelated means of quantitating abnormal ventricular function. It is of interest that not all of the ventricular segments examined appeared to be hypokinetic in our patients with cardiomyopathy. While the significance of this finding is uncertain, it has been noted by others and has recently provided a basis for classifying patients with cardiomyopathy.

The definition of regional hypokinesis is also important since this is such a common contraction abnormality in patients with coronary heart disease. We prefer the use of this term to asynergy which has previously been used since we feel that simplification of terminology is necessary for an increased understanding of left ventricular wall motion abnormalities. The use of our method for demonstrating hypokinetic segments seems best corroborated by the improved correlation with sites of coronary artery obstruction. The significance of the finding that some of these segments appear to be actually akinetic when examined by our method is unclear. The larger question of the pathophysiologic significance of
QUANTITATIVE DETECTION OF HYPOKINESIS

hypokinetic left ventricular segments will require further elucidation. The presence of a normal ejection fraction in many of our patients with coronary disease and regional hypokinesis suggests that normal over-all ventricular function may be maintained by greater than normal motion in nondiseased segments. The high degree of correlation between mean percent motion for all segments with ejection fraction suggests that despite its theoretical limitations in patients with coronary heart disease, calculation of ejection fraction from 30° RAO cineangiocardiograms probably still has value as an expression of over-all ventricular function.

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