Evaluation of Methods for Quantitating Left Ventricular Segmental Wall Motion in Man Using Myocardial Markers as a Standard

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SUMMARY Radiopaque markers were implanted in the left ventricular myocardial midwall in 58 patients and studied in the 30° right anterior oblique projection by computer-aided fluoroscopy. Marker motion was used as a standard of segmental wall motion against which the accuracy of five methods for measuring left ventricular wall motion was assessed: two methods using hemiaxial measurements in rectangular coordinates, two using radial measurements in polar coordinates (all with frame-by-frame axial reindexing) and one using radial measurements in fixed external polar coordinates. The latter method showed significantly less error (25.9%, p < 10^-4) in measuring midwall marker motion than the other four methods (range 42.5-47.5%) in the group as a whole and in subgroups that had abnormalities of posterior, apical and anterior wall motion. This method also had the best correlation of marker motion and motion of adjacent endocardial border (of the overall left ventricle and the posterior, apical, and anterior walls separately) as visualized by ventriculography in 29 patients. The bulk of the reduction in error using this method was due to the use of a fixed external reference system, with a small additional increment of error removed by proper selection of the polar origin at a point 69% of the distance from the anterolateral edge of the aortic valve to the ventricular apex at end-systole.

CONTRAST VENTRICULOGRAMS are being used more often to quantify left ventricular segmental wall motion in order to assess objectively the effects of cardiovascular disease and therapeutic interventions on ventricular performance. A variety of methods has been reported to measure the excursion of the ventriculographic border. It has not been possible, however, to evaluate the measurement accuracy of these methods because no standard of wall motion has been available.

In an attempt to provide such a standard, we implanted radiopaque markers into the left ventricular midwall of patients at the time of cardiac surgery. The markers, placed so as to silhouette the left ventricle in the 30° right anterior oblique (RAO) projection, are visualized fluoroscopically, allowing computer-aided measurement of the motion of specific myocardial sites.

In the present study, the measurement accuracy of five methods for assessment of wall motion was evaluated by comparing marker motion as defined by each method with actual marker motion measured in laboratory coordinates. Contrast ventriculograms from patients with markers were analyzed by the same five methods to determine the accuracy with which midwall marker dynamics could be inferred from excursions of adjacent endocardial borders as visualized in contrast angiograms.

Patient Study Groups

Fifty-eight patients were studied, 36 of whom had myocardial markers inserted at the time of cardiac allograft transplantation and 22 during coronary artery bypass graft surgery. Patients were further categorized as having normal wall motion or abnormalities of the anterior, apical, or inferior wall. Regional wall motion was defined as abnormal if the excursion of the markers in the region fell below the tenth percentile of the motion of corresponding markers in patients who were free of coronary disease and had subjectively normal ventriculograms.

Twenty-nine patients with markers who underwent left ventriculography were studied to determine the correlation of endocardial motion as determined from left ventricular angiograms with midwall dynamics as defined by the markers.

Informed consent was obtained for marker implantation and subsequent studies in accordance with the Committees on the Use of Human Subjects in Research at both the Stanford University School of Medicine and the Palo Alto Medical Research Foundation. There were no complications from these studies.

Identification of Left Ventricular Sites

At the time of cardiac surgery, seven miniature tantalum coils (0.85 mm × 1.5 mm) were implanted to a depth of 5 mm in the midwall of the left ventricle using a pointed, flanged, insertor tool. The markers were positioned around the heart in a single plane (fig. 1), using features of the surface anatomy of the heart as landmarks, to outline the left ventricle viewed in the 30° RAO projection. Three markers were positioned within the inferior wall, three markers in the anterior wall, and one in the apex. A pair of silver tantalum...
clips was affixed to the adventitia of the ascending aorta, 2 cm above the aortic valve ring, to delineate the position of the aortic valve. For all studies, points were identified as shown in figure 1, with points 1 and 9 as the borders of the aortic valve (defined by translations of the aortic marker positions), point 10 the midpoint of the aortic valve, and points 2–8 the sites of the midwall markers.

Acquisition of Segmental Dynamics

Measurements of left ventricular marker dynamics were obtained during sustained inspiration by fluoroscopy in the 30° RAO projection using a Philips 300 MA generator with a 9” cesium iodide image intensifier. Images were recorded on an Ampex DR-10A video disc recorder at 30 frames/sec. The ECG signal (lead II) was superimposed on the video image. Magnification was determined by imaging a 1-cm grid at the level of the heart as determined echographically.

Video recordings were replayed in a frame-by-frame manner, and the X, Y coordinates of the marker images were digitized manually using a light pen coupled to a minicomputer. Marker coordinates were corrected for magnification and distortion and transmitted via telephone line to a CDC6400 computer that was used for further processing.

Twenty-nine patients had contrast ventriculography using 50 ml of Renografin-76 (meglumine diatrizoate) injected over 3 seconds through a #6.7F angio pigtail catheter into the left ventricular cavity. Ventriculograms were performed in the 30° RAO projection and were recorded during sustained inspiration. They were checked to ensure that there was no relative motion of the patient, thoracic cage or radiographic apparatus. Ectopic or postectopic beats were excluded from analysis. The projected cineventriculograms were manually traced by an experienced technician using the outermost visible margin of contrast as a guide to the endocardial surface. Particular care was taken to include the papillary muscles within the chamber tracing and to identify the most distal apical point visualized by contrast. End-diastolic and end-systolic frames were identified on the basis of maximum and minimum chamber areas, myocardial marker positions were marked on the ventriculographic tracings and these two frames were used to define systolic motion in subsequent analysis.

Terminology

A consistent terminology is needed to compare and discuss the various methods for wall motion assessment. The following terms are defined for this report.

Coordinate System

Two systems are most often used to measure the excursion of the ventricular wall: Rectangular coordinates. This system specifies the positions of points in the ventricular wall by their distances from two fixed perpendicular lines (axes) which intersect at a point termed the origin. Conventionally, the long axis of the ventricle defines one axis of a rectangular coordinate system, and points in the wall are assumed to move in a direction parallel to the other (perpendicular) axis along lines termed hemiaxes or chords.

Polar coordinates. This system specifies the positions of points in the ventricular wall by their distances from a fixed point (origin) and angles relative to a line (polar axis) from the origin. Conventionally, the ventricular long axis is the polar axis of a polar coordinate system, and points in the wall are assumed to move along radial chords at constant polar angles.

Reference System

This places the coordinate system relative to the heart and its surroundings.

External reference system. The coordinate system is not coupled to the motion of the heart. For example, the edges of lead shutters, ribs, diaphragm or external chest wall markers could be used to define an external reference system.

Internal reference system. The coordinate system is coupled to specific identifiable points within the heart; for example, valvular edges and midpoints, apex, and ventricular center of area.

Indexing System

In a sequence of ventriculographic frames, the coordinate system can be defined by a single frame or can move with successive frames. If the coordinate system moves in order to realign or superimpose images, this motion can be expressed in terms of 1) translation of the origin, which moves the origin to a new position while maintaining the new axes parallel to the old ax-
es, and 2) rotation of the axes, revolving them about the origin.

The point about which the ventricular long axis is commonly "rotated" to superimpose serial frames is not necessarily the origin of the coordinate system (i.e., the origin of hemiaxial or radial chords). However, this type of long-axis realignment can always be expressed in terms of a combination of translation of and rotation about the true origin of the coordinate system.

**Methods Evaluated**

Characteristics of the five methods for wall motion analysis are listed in Table 1. Method 1, first described by Herman et al., defines the origin of a rectangular coordinate system at the midpoint of the aortic valve. A ventricular long axis is defined from this point to the apex. Frame-by-frame indexing superimposes the origin and long axis by translation and rotation of the coordinate system.

Method 2, described by Leighton et al., defines, at the onset of ejection, an indexing line extending from the apex, bisecting the ventricular area. The origin of a rectangular coordinate system is defined for each frame at the intersection of this line with the aortic valve plane. The long axis (from origin to apex) is rotated to coincide with the indexing line on each frame.

Method 3, reported by Harris et al., defines a line from the apex to the midpoint of the aortic valve on each frame. The origin of a polar coordinate system is defined at the midpoint of this line, and the polar axis is defined from the origin to the aortic valve midpoint.

Method 4, reported by Rickards et al., defines the origin at the center of area of the end-systolic frame. The polar axis is defined from the origin to the midpoint of the aortic valve. On each frame the origin is placed at the same distance from the midpoint of the aortic valve and the polar axis at the same angle from the aortic valve plane.

Method 5, reported by Ingels et al., defines a fixed external reference system with the origin located at a point 69% of the distance from the anterolateral aortic valve edge to the apex on the end-systolic frame. The polar axis passes through the end-systolic position of the anterolateral aortic valve edge. There is no frame-by-frame indexing.

**Comparison of Standard Marker Motion With Marker Motion Measured by Each Method**

Standard marker motion (D, fig. 2) was defined to be the distance traveled by each marker from its position in the previous frame to its position in the present frame measured in a fixed external (laboratory) reference system.

Marker motion as measured by each method (d, fig. 2) was defined as the measured travel of each marker.
from its position in the previous frame to its position in the present frame using the reference system defined by each method. For each method the error (E) was defined as the absolute value of the difference between standard marker motion and marker motion measured by each method. Total error was obtained (see Appendix) by summing over all markers and all beats.

Comparison of Standard Marker Motion with Angiographic Border Motion Measured by Each Method

Angiographic endocardial border motion \((b, \text{fig. } 3)\) from end-diastole to end-systole was defined as the distance traveled by the endocardium adjacent to each marker using the reference system defined by each method. These values were correlated with corresponding values of standard marker motion \((D)\) using a zero-intercept linear regression analysis.

Results

Comparison of Standard Marker Motion with Marker Motion Measured by Each Method

The total errors computed from 173 beats in 58 patients for each of the five methods are shown in figure 4. Errors associated with methods 1–4 were not significantly different from one another. The errors from all four were significantly greater \((p < 10^{-6})\) than those computed from method 5. The absolute error as a percentage of total marker motion was 47.5%, 42.5%, 44.0%, 46.2% and 25.9% for methods 1–5, respectively.

Table 2 shows the error as a percentage of the total

![Figure 3. Schematic diagram of a region of the left ventricular wall at end-diastole and end-systole. Locations of epicardium, marker, and adjacent endocardial border \((\Delta)\) as visualized by contrast ventriculography are shown. In laboratory coordinates, marker \(m\) moves \(D_m\) (standard marker motion). Motion of the endocardial border adjacent to the marker measured along the hemiaxis or radius defined by each method is \(b\). Values of \(b\) and \(D_m\) were compared by linear regression analysis.](http://circ.ahajournals.org/)

![Figure 4. Comparison of errors in assessing marker motion associated with each method. Total error is the summation over all markers and beats (see Appendix) of the error \(E_m(n)\), shown in figure 2. This error is also expressed as a percentage of total standard marker motion (see Appendix).](http://circ.ahajournals.org/)
the anterolateral aortic valve edge to the ventricular apex at end systole (method 5). This method was suggested by the motion of ventricular wall segments as measured by implanted markers. Our previous studies of the dynamics of these markers had shown that, to good approximation, the motion of each midwall marker tracks a single line throughout the cardiac cycle. The orientation of this line is different for each marker, but during systole the markers move approximately toward a central point. For both the posterior and anterior walls, systolic excursion of basal segments is typically inward and toward the apex (fig. 5). Apical segment excursions are directed inward and toward the base. Midbase-apex segments move inward toward a ventricular long axis. The 69% point represents the best mathematical approximation to the intersection of these lines, representing that point toward which all ventricular segments move in systole and away from which the segments move in diastole.

Figure 5 shows contours of constant error that were calculated in our previous study. Using a fixed external reference system, 1400 different points in the left ventricle were tested as candidates for the optimum polar origin. Note that the error contours are gentle; thus, the location of the polar origin is not critical. The 69% point represents the optimum position (minimum error) but the selection of another point, such as the midpoint of a ventricular long axis, as shown from midaortic valve to apex in figure 5 (the 50% point), increases the error by only 2.1%. In contrast, frame-by-frame reindexing of this midpoint (as in method 3) yields an error of 44.0%, a 1.6-fold increase in error over that associated with the fixed external reference system. Thus, the bulk of the error reduction using method 5 was caused by use of a fixed external reference system, with a small additional increment of error removed by proper selection of the polar origin.

The minimum error point was not found at the center of mass of the left ventricle as suggested by Pearlman et al., but at a location anterior and apical from the center of mass. In our patients, midwall motion in the 30° RAO plane is typically asymmetric; it is of greater magnitude and more centrally directed in the posterior wall than in the anterior wall.

Although the combination of a fixed external reference system and polar coordinate system was more accurate than the four other methods in assessing midwall dynamics from ventriculograms, the correlation coefficients (table 3) were not high enough (particularly in the anterior wall) to conclude that myocardial dynamics can be measured with precision from contrast angiograms using any combination of reference and coordinate systems. The most plausible explanation for this poor correlation between midwall (marker) and endocardial (ventriculographic) dynamics is the artifact due to trabeculation and papillary muscle exclusion of dye, which is severe in late systole and extreme in left ventricular hypertrophy and idiopathic hypertrophic subaortic stenosis, uncoupling contrast border motion from wall motion in an unpredictable way. We often observe smooth translations of contrast borders in early systole,
followed by abrupt discontinuities as dye is squeezed rapidly out of enfolding regions; yet, such abrupt motions are not observable in adjacent ventricular midwall markers. The fact that the correlation coefficient is lower in the anterior than the posterior wall may result from an asymmetry in left ventricular chamber geometry during contraction due to the exclusion by anterior papillary muscles, as seen in the cast studies of Ross et al.12

Three major problems have prevented definition of optimum methods for measuring left ventricular segmental dynamics.

First, a precise method of tracking fixed sites in the myocardium has not been available. The use of intramyocardial markers, as in the present study, circumvents this problem.

Second, methods for clinical studies in man, such as angiography and echocardiography, involve recognition of either epicardial borders, which do not reflect important wall thickness changes throughout the cardiac cycle, or endocardial borders, which are confounded by trabeculation and papillary muscle artifacts. This is not to imply that important clinical information cannot be derived from such studies; only that wall motion so derived may be poorly correlated with the motion of the adjacent myocardium.

Third, and most fundamental, is that the motion of a given point in the left ventricle relative to laboratory coordinates can arise from two primary sources. The first is extracardiac motion (e.g., respiration or patient movement), which is coupled to the left ventricle with a frequency considerably less than the heart rate. In our experience, this motion can be reduced to acceptable limits by temporary suspension of respiration and maintenance of fixed x-ray system geometry and patient position relative to the recording system. One way to quantify this error is to calculate the distance translated (in laboratory coordinates) in the 30° RAO plane by the left ventricular center of mass at end-diastole in sequential beats. In the present study, this translation was 0.08 ± 0.06 cm, which is quite small relative to typical segmental motion of approximately 1 cm in this plane.

If the frequency of extracardiac motion becomes comparable to the heart rate, however, it can become indistinguishable from that of the second source of wall motion, which is intracardiac in origin. This, of course, arises primarily from stresses developed by contraction of the left ventricular myocardium itself, both locally and coupled with the contraction of other fibers in the walls of the heart. Abnormalities of segmental contraction have not only local effects on wall motion, but also on adjacent and contralateral wall segments as well.

Other sources of motion that must be considered include the acceleration of blood into and out of the chamber (ballistocardiographic motion), the motion of the major blood vessels coupled to the heart, and the motion of the atra and right ventricle, which may either deform, translate, or rotate the left ventricle as they change shape and exert forces on the heart and nearby thoracic structures.

There has been a tendency to define methods for wall motion analysis that provide relatively uniform numeric values to segmental performance for both the anterior and inferior walls.1 This approach is one of the primary reasons most authors have preferred frame-by-frame indexing of an internal reference system, to "correct" for ventricular translation and rotation. Our studies using markers, as well as those of Rickards et al. using contrast angiograms,4 suggest that posterior wall motion is significantly greater than that of the anterior wall. Further, our studies of markers implanted in apical locations show that long-axis rotation in the RAO plane is not significantly different from 0° at any time during the cardiac cycle.13 This is in contrast with the anterior apical rotations up to 10° reported in previous studies using contrast angiography.14 The inability to identify identical points on the apex in successive frames in contrast angiograms causes uncertainty in measuring long-axis rotation by angiography. We have measured small rotations of transverse diameters out of the RAO plane during systole, and have shown that errors from this source in the assessment of shortening in the 30° RAO plane are negligible.2
Methods of wall motion analysis have also been compared using repeated measurement of a set of angiograms in normal subjects to identify the method with the best reproducibility. Another approach has compared results of different methods with physician interpretation, and has revealed that a fixed external reference system is to be preferred.

We conclude that 1) since no present means exists to isolate the various intracardiac components of force underlying the motion of a given myocardial segment, 2) since anterior and posterior wall contraction is apparently asymmetric even in the absence of systolic long axis rotation, and 3) since motion of extracardiac origin can be monitored and easily reduced to acceptable limits, there is no obvious advantage to translation or rotation of the measurement coordinate system during contraction. We propose the use of the fixed external reference system using polar coordinates summarized in figure 6. This approach, one of the simplest, results in the least error and best correlation with midwall motion as measured directly from implanted markers.

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References


Appendix

Given a set of seven markers (m = 2, 3, . . . 8, as shown in fig. 1), observed in frame n of a given cardiac cycle (n = 1, 2, . . . N, where n = 1 for the first systolic frame and n = N for the end-diastolic frame of the next cycle), marker m having coordinates [Xm(n), Ym(n)], and given a point p defined inside the ventricular border by coordinates [Xp(n), Yp(n)], then the distance from p to the marker m in frame n is

\[ L_m(n) = \sqrt{(X_m(n) - X_p(n))^2 + (Y_m(n) - Y_p(n))^2}. \]  

(1)

In the preceding frame (n−1), the distance from p to the marker was

\[ L_m(n-1) = \sqrt{(X_m(n-1) - X_p(n-1))^2 + (Y_m(n-1) - Y_p(n-1))^2}. \]  

(2)

Thus, using p as a reference, the absolute value of the motion of marker m is calculated as

\[ d_m(n) = \mid L_m(n) - L_m(n-1) \mid . \]  

(3)

The actual motion of the marker, however, relative to laboratory coordinates, is

\[ D_m(n) = \sqrt{(X_m(n) - X_m(n-1))^2 + (Y_m(n) - Y_m(n-1))^2}. \]  

(4)

Thus the absolute value of the error associated with the use of reference point p in frame n for this marker (as shown in fig. 2) is given by

\[ E_m(n) = \mid D_m(n) - d_m(n) \mid . \]  

(5)

The total error for each beat is thus

\[ E = \sum_{m=2}^{8} \sum_{n=1}^{N} E_m(n). \]  

(6)

and the total actual marker motion is given by

\[ D = \sum_{m=2}^{8} \sum_{n=1}^{N} D_m(n). \]  

(7)
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