A computer graphic–based angiographic model for normal left ventricular contraction in man and its application to the detection of abnormalities in regional wall motion

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ABSTRACT Analyzing the digitized left ventricular cineangiograms of 70 patients with no demonstrable heart disease (NDHD), we derived an angiographic model for normal contraction in the intact heart as viewed in the 30 degree right anterior oblique projection. This model was verified statistically by comparing the predicted regional stroke volumes with the measured volumes for the NDHD group. A wall motion system based on this model was compared with four other systems by examining the ventriculograms of 141 patients, all suffering from coronary artery disease but with normal volumes and ejection fractions (>0.61). Of these, 60 had normally contracting ventricles and 81 exhibited mild regional abnormalities according to two experienced angiographers. Using Cochrane’s Q test, we found significant differences among the five methods (Q = 29.5; p < .001). The new approach showed significantly better agreement with the subjective assessment than the next best method (Q = 5.3; p < .05). On a regional basis, overall sensitivity was 87.5% and specificity was 97.9%.

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THE UBIQUITOUS USE of computers to objectively analyze cardiovascular data is one indication of their importance for assessing cardiac performance in man. Of particular interest to many cardiac centers is the objective assessment of left ventricular function. The measurement of global parameters, such as volume and ejection fraction, has been widely accepted and is routinely used by most cardiac treatment centers.1 However, of growing importance for patients suffering from coronary artery disease is the measurement of regional dysfunction, since this disease has been shown to affect left ventricular contractility in a non-uniform fashion.2 This type of localized dysfunction generally manifests itself as a wall motion abnormality, which if quantified can provide clinically useful information. The straightforward measurement of this motion as visualized by ventriculography is easily accomplished by contemporary image processing equipment. However, this is generally not sufficient, since one must be able to differentiate between motion caused by simple translation and/or rotation of the left ventricle and that caused by contraction of muscle.3 It is this latter component that reflects the state of health of the myocardium and is therefore of great interest to the cardiologist. Many methods of measuring this regional myocardial contraction have been suggested and compared.4 Although some of these quantitative methods are in clinical use, none has been universally accepted; as a result, subjective assessment of wall motion by experienced angiographers continues to be the method of choice in many cardiac centers.

In this article we describe a new computer graphic–based approach to the measurement of regional wall motion abnormalities, which we believe more closely simulates the mental process that experienced observers use to assess wall motion in left ventriculograms. Specifically, we assume that they detect abnormalities by mentally comparing the real end-systolic silhouette with a conceptual image of a normal end-systolic silhouette, derived in part from the end-diastolic image. Using this premise, we have developed a computer graphic–based model of systole that replaces this conceptual image with an ideal end-systolic silhouette.

We will describe the methods we used to implement this approach and will discuss some of its advantages and limitations. Finally, the performance of this meth-
Methods

Patients. Seventy patients undergoing cardiac catheterization for suspected heart disease in whom the findings were normal form the group with no demonstrable heart disease (NDHD). There were 31 men and 39 women, ages 30 to 67 years (mean 48.1 ± 9.3).

A total of 141 patients with angiographic evidence of greater than 50% narrowing of at least one coronary artery form the group with coronary artery disease (CAD). There were 117 men and 24 women, ages 32 to 72 years (mean 52.0 ± 8.5). Thirty-eight patients had single-vessel involvement, 44 had double-vessel disease, and 59 had triple-vessel disease. There was no significant difference in these factors (p > .05) between the 60 patients judged to have normal left ventricular contraction and the 81 judged to have a localized contraction abnormality.

Data acquisition and analysis system. All cardiac data used in this study were acquired from patients undergoing routine diagnostic catheterization at the University of Ottawa Cardiac Unit. Conventional single-plane 35 mm cineangiographic techniques in the 30 degree right anterior oblique position were used to obtain the ventriculograms. A 5 cm calibration grid was also filmed to correct for x-ray magnification effects. In choosing cardiac cycles for analysis, we rejected both ventricular premature beats and the first beat after extrasystole.

Using a projector with single-frame capability, a cardiologist selected the end-diastolic and end-systolic frames (defined as those having the largest and smallest observed areas, respectively), traced each endocardial border on a sheet of paper, and identified the following four ventricular landmarks: the apex, the aortic-mitral valve junction, the superior aspect of the aortic valve, and the inferior aspect of the mitral valve.

It is clear that ventricular angiograms are graphic by nature. Therefore it seemed appropriate to analyze these data with the computer graphic tools and techniques that are widely available today at general-purpose interactive computer graphic terminals. In our own laboratory, we have used such a system as both a research and a clinical tool to study left ventricular volume, shape, and wall motion.3-5 It consists of a Mod Comp II 16 bit processor with 64K words of memory, with programs and data storage handled by a nine-track magnetic tape drive and a 1 M word moving head disc. A card reader and line printer are used for program development purposes. A refresh type vector display, an x-y plotter, a keyboard, thumbwheels, and a 10 bit 35 × 35 cm data tablet provide the graphic and textual input-output facilities that distinguish a good interactive system. The system software supplied with the computer was enriched with both general-purpose graphics software routines and special-purpose application-oriented data analysis programs. The final result is a powerful and flexible research tool that allows the user to analyze data in a simple and natural way.

With this system, digitization of the ventriculographic data is effected by tracing the silhouettes and the calibration grid with the stylus on the tablet. The distance between adjacent digitized points is under program control, which for routine analysis is between 2 to 3 mm. For this spatial resolution, an average silhouette of about 12 × 20 cm is represented by about 250 points. Under computer prompting, the ventricular landmarks are identified and entered into the computer by using the tablet stylus as a pointer. Patient identification and other pertinent data, such as body surface area for example, are added to the patient’s computer record by entering these data with the system keyboard.

During this initial acquisition phase, no correction is made for either the apical rotation of the ventricle or the descent of the aortic-mitral valve ring, which usually accompanies systolic contraction. Such corrections are deemed to form part of the scheme for wall motion analysis under study. Our software permits the investigator to analyze data using the registration system of his choice — Herman-Gorlin, junction-apex, center of gravity, etc. — by means of either a radial or rectilinear coordinate system. These options have been programmed into the system in a modular fashion. By selecting these procedures from lists displayed on the CRT, the investigator can quickly analyze the same data by many different registration and coordinate systems. This method makes evaluation of existing and/or proposed wall motion systems relatively easy.

All ventricular volume measurements are made by considering the ventricle as a three-dimensional body. In a computational sense, this is accomplished by using a scanning algorithm to convert the two-dimensional silhouette described by discrete points into a raster-type silhouette composed of a vertical array of equally spaced horizontal lines running from the inferior to the anterior endocardial borders. With the 5 cm calibration grid used as the reference, the lengths of these lines are converted to real physical dimensions. In this representation, the left ventricle is viewed as a stack of cylindrical discs. By applying Simpson’s rule to a single-plane adaptation of the Chapman formula,8 we are able to compute the overall volume of the ventricle and its sections.

Derivation of the model. Left ventricular fiber architecture in man is very intricate, causing it to move in three-dimensional space in a highly complex manner during systole.9-11 In developing a computer program to describe normal contraction-generated wall motion, as viewed angiographically in the 30 degree right anterior oblique projection, we found it useful to separate the many individual forces causing this contraction into two major components: one causing the endocardial surface of the left ventricle to shorten along some predefined long axis, the other causing the inferior and anterior walls to move toward each other in a direction perpendicular to this axis. In this model, it is the simultaneous action of these two forces that gives rise to systolic contraction. As a result, abnormalities manifest themselves as either insufficient or nonuniform motion in either one or both of these two directions.

To formulate this simple model as a computer algorithm, we determined the normal extent of the motion along both axes by analyzing the cineangigrams of the 70 patients in our NDHD group. By means of the computer graphics system’s cardiac package, the length of the long axis at the end of diastole and systole was measured for two of the long axis definitions currently in clinical use: one defined as the line running from the midpoint of the aortic valve to the apex, the other as the line joining the junction of the valves to the apex. For the latter, the frequency distribution of this ratio for these 70 normal patients is shown in figure 1. The deviation of this distribution from normality is not significant (x² = 16.25; p > .1).

To determine the relationship, if any, between the ratio of the long axes, R, and the ejection fraction, EF, we programmed the computer to calculate and plot the scatter diagrams shown in figure 2, for both long axes of interest. In both cases the correlation between these two parameters is not strong; however, it is highly significant (p < .001). The relationship between the two parameters can be expressed by the following regression equations:

Case 1. Aortic midpoint to apex long axis

\[
R = 1.14882 - 0.52664(EF) \\
\text{EF} = 0.6872 \pm 0.0505 \\
R = 0.7869 \pm 0.0462 \\
r = -.5734, \quad r^2 = .3151 \\
\text{har} = 5.8073 \\
\]

where r = correlation coefficient
FIGURE 1. Frequency distribution of ratio of long axis at end-systole to that at end-distole for 70 patients with NDHD. Long axis defined as running from valvular junction to apex.

Case 2. Junction to apex long axis

\[ R = 1.11538 \pm 0.45221 (EF) \]
\[ EF = 0.6859 \pm 0.0504 \quad R = 0.8052 \pm 0.0444 \]
\[ r = -0.5100 \quad /t = 4.92439 \]

To use the above relationships in computer graphic scaling algorithms, we have considered the ventricle as a three-dimensional body comprising circular cross sections throughout its length. Under this assumption, it is clear that

\[ V \propto s_y s_x^2 \]

where \( s_y \) = scale factor along the \( y \)-axis and \( s_x \) = scale factor along the \( x \)-axis. Using the normal ejection fraction given above, we can show from relationships derived in the Appendix that \( s_y = 0.7869 \) and \( s_x = 0.6305 \) for case 1, while for case 2 \( s_y = 0.8052 \) and \( s_x = 0.6246 \). This means that for a normal ventricle, contraction along the long axis, expressed by \( s_x \), contributes about 28.4\% of the overall stroke volume. The remaining 71.6\% is due to contraction perpendicular to the axis, expressed by \( s_y \).

The implementation of the modeling algorithm for a normal ventricle using the scaling factors for case 2 is shown in figure 3, A. In this version, the initial predicted end-systolic silhouette was generated by first scaling the end-diastolic silhouette along the long axis by an amount equal to \( s_x \) as computed by the regression equation derived earlier. This resulting silhouette was then scaled perpendicular to the long axis until our normal ejection fraction of 0.68 was obtained.

Excellent agreement is evident between the real and the predicted ideal end-systolic silhouette everywhere except near the basal regions, where the valvular ring has a constraining effect on the wall motion. To minimize this error, we applied a double correction to these regions.

First, without any loss of generality, we accepted without modification the real contour of the valves between the inferior aspect of the mitral valve and the superior aspect of the aortic valve. Second, we devised a computer algorithm to combine the real and the initial ideal silhouette to generate a final smoothed contour for the inferobasal and the anterobasal regions of the end-systolic silhouette. This blending procedure is accomplished by first dividing the real end-systolic silhouette and its initial predicted version into a number of smaller equal lengths as follows: The perimeter from the apex to the mitral valve edge was divided into 24 equal lengths as was the perimeter from the apex to the aortic valve edge. In effect we sampled the free wall of the left ventricular image at 48 points as shown in figure 3, B. Although this number of points is not critical to our method,
more points do offer higher resolution and potentially greater accuracy. Note that during contraction the points appear to move toward many separate foci located along the long axis rather than toward a single focus as occurs in most radial chord systems.

The first seven points from the valvular edge of both input silhouettes were joined by straight lines. A simple linear interpolation algorithm was used to establish the final smoothed border between the real and the initial ideal silhouette. The result of these corrections can be seen in figure 4, where the computer-generated ideal end-systolic silhouette is shown as a dotted line for a normally contracting ventricle.

**Accuracy and application of the model.** Other investigators have used a number of different methods of identifying left ventricular regions to assess their function. In our study, we elected to partition the left ventricle into five regions by dividing the long axis into three sections of 36%, 40%, and 24% as shown in figure 4. These particular percentages were obtained by having an experienced cardiologist partition the silhouettes of a group of patients with NDHD into anatomically meaningful segments in accordance with the American Heart Association angiographic classification scheme.12

This partitioning scheme was subsequently used to evaluate the accuracy of our model by measuring the real partial stroke volumes for the control group of 70 patients, and by comparing these volumes against the predicted volumes obtained with the ideal end-systolic silhouettes. The resulting data, for both long axes under consideration, are shown in table 1.

Also shown are the results of a t test for related paired samples, which indicate that for the model under consideration, the junction-to-apex long axis is clearly the better choice. Indeed, only the inferobasal region is significantly in error, while for the aortic midpoint-to-apex long axis all regions except the apical region show significant errors. This result is a reflection of the fact that our model uses the same scaling factor perpendicular to the long axis for both inferior and anterior walls. Equally good results could have been obtained with the other axis had we optimized the scaling factor for each wall. Since this would have required the use of two different scaling factors along the x-axis, resulting in a more complicated model, we decided to use the simpler junction-to-apex axis for the clinical phase of the study.

In adapting this model for the detection of abnormalities in

**FIGURE 3.** A. Normal ventricular silhouettes with uncorrected computer-generated end-systolic silhouette shown as dashed line. B. Figure outlining how the model uses points on a patient’s real end-diastolic silhouette to generate the ideal end-systolic silhouette.

**FIGURE 4.** Method of quantifying errors in representing regional wall motion. Ideal end-systolic silhouette is shown as dotted line. Normal ventricle.
TABLE 1
Comparison of real and predicted partial stroke volumes (ml/m², mean ± SD)

<table>
<thead>
<tr>
<th>Region</th>
<th>Junction-apex long axis</th>
<th>Real volume</th>
<th>Predicted volume</th>
<th>t(p)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Inferobasal</td>
<td></td>
<td>5.40 ± 2.52</td>
<td>5.62 ± 2.55</td>
<td>5.48(s)</td>
</tr>
<tr>
<td>Anterobasal</td>
<td></td>
<td>9.56 ± 2.67</td>
<td>9.69 ± 2.54</td>
<td>1.81(ns)</td>
</tr>
<tr>
<td>Inferior</td>
<td></td>
<td>9.82 ± 2.90</td>
<td>10.10 ± 3.07</td>
<td>2.28(s)</td>
</tr>
<tr>
<td>Anterior</td>
<td></td>
<td>16.57 ± 3.78</td>
<td>16.35 ± 3.65</td>
<td>0.99(ns)</td>
</tr>
<tr>
<td>Apical</td>
<td></td>
<td>4.88 ± 1.35</td>
<td>4.78 ± 1.33</td>
<td>1.75(ns)</td>
</tr>
<tr>
<td>All regions</td>
<td></td>
<td>46.22 ± 9.50</td>
<td>46.53 ± 9.83</td>
<td>0.79(ns)</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Region</th>
<th>Herman-Gorlin long axis</th>
<th>Real volume</th>
<th>Predicted volume</th>
<th>t(p)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Inferobasal</td>
<td></td>
<td>8.40 ± 3.09</td>
<td>8.60 ± 3.12</td>
<td>3.47(s)</td>
</tr>
<tr>
<td>Anterobasal</td>
<td></td>
<td>7.59 ± 2.27</td>
<td>7.10 ± 2.02</td>
<td>8.50(s)</td>
</tr>
<tr>
<td>Inferior</td>
<td></td>
<td>13.08 ± 3.29</td>
<td>14.16 ± 3.65</td>
<td>6.58(s)</td>
</tr>
<tr>
<td>Anterior</td>
<td></td>
<td>12.80 ± 3.28</td>
<td>12.40 ± 3.15</td>
<td>2.83(s)</td>
</tr>
<tr>
<td>Apical</td>
<td></td>
<td>4.50 ± 1.26</td>
<td>4.52 ± 1.30</td>
<td>0.33(ns)</td>
</tr>
<tr>
<td>All regions</td>
<td></td>
<td>46.46 ± 9.62</td>
<td>46.78 ± 9.96</td>
<td>0.80(ns)</td>
</tr>
</tbody>
</table>

s = significant; ns = not significant.

Figure 5. A, Ventricles with mild hypokinesis of the inferior wall with a compensating hyperkinetic anterior wall. Ideal end-systolic silhouette is shown as dotted line. B, Ventricles with apical and anterior wall hypokinesis with a compensating hyperkinetic inferior wall. Ideal end-systolic silhouette is shown as dotted line.

ventricles. The ventricle in figure 5, A, had been subjectively judged to have a mild abnormality in the inferior wall, while in figure 5, B, exhibited moderate-to-severe apical and anterior wall hypokinesis. Note how in both cases all regions of the left ventricle that display insufficient movement are clearly delineated. For example, in figure 5, A, it is evident that the inferior wall has not contracted as far as it should have to qualify as a normally contracting ventricle. The same is true in figure 5, B, for the apical and anterior regions of the left ventricle.

Another advantage of this mode of presentation is the clear delineation of compensatory hypercontractile regions for ventricles with a normal ejection fraction. In particular, note how the anterior wall in figure 5, A, and the inferior and upper portion of the anterior walls in figure 5, B, have moved further inward than normally expected. This additional movement compensates for the hypocontractile regions of the ventricle to maintain a normal ejection fraction of 0.70.

Many different methods of quantifying regional wall motion abnormalities have been proposed. These range from the use of chord lengths to the computation of displaced area or volume. Although any one of these parameters can be used to measure the difference between the real and the ideal end-systolic silhouette, we chose the area-based approach for this study.

In particular, the quantification of these errors was accomplished by computing the area of disagreement for each region as a percentage of the region’s area in the end-systolic silhouette. To establish reference thresholds upon which objective decisions for regional abnormality could be made, we applied the model to the control (NDHD) group and computed the mean and standard deviation for the percent hypocontractile error in each region. These results are shown in table 2.

On the basis of these data we set the error threshold for the inferobasal, inferior, anterior, and apical regions at 7%, 17%, 16%, and 24%, respectively. This is 1 SD above the mean for each region.

From previous subjective assessments of similar groups of patients with CAD we observed that, unlike the other regions, anterobasal dysfunction occurred only in the presence of anteri-
or wall abnormalities, never in isolation. The reason for this is not understood, but the regional pattern of coronary blood supply may play a role. Nonetheless, we incorporated this observation into our method by recording an abnormality in the antero-basal region only when its error exceeded its threshold of 6% and the adjacent anterior region exceeded its error threshold of 16%. In our study, this factor was not overly critical, since no anterobasal abnormalities were observed in the CAD group.

Clinical results

To evaluate the effectiveness of the described modeling concept in detecting regional wall motion abnormalities, we applied it to the analysis of a test group of 141 patients all suffering from CAD. These patients, who had all undergone routine diagnostic catheterization, were selected because they had left ventricles of normal volume and ejection fraction (>0.61). In such a data base, we believed that most abnormalities would be of a minor type, thus ensuring an exacting test for comparing wall motion systems. A summary of the global parameters for this patient population and the control group is shown in table 3. The means for the normalized end-diastolic volume, the normalized stroke volume, and the ejection fraction for the CAD group was 73.50 ± 19.18, 49.66 ± 12.74, and 0.68 ± 0.04, respectively. Only the normalized end-diastolic volume was significantly (although marginally) different (t = 2.18; p = .03) from the NDHD control group.

At this time the experienced observer is the ultimate judge of whether a ventricle is normal or not. Consequently, we asked two experienced angiographers to view the cineangiograms of each patient in this data base and to divide them into two groups: those with normally contracting ventricles, of which there were 60, and those with regional abnormalities, of which there were 81.

Using this data base as our standard, we compared the performance of the modeling concept with that of four other commonly used methods (figure 6):

Method A. Herman-Gorlin registration² with re-

gional shortening measured along five axes, four perpendicular to and one along the long axis.

Method B. Herman-Gorlin registration² with a radial coordinate system to measure regional shortening along seven radial chords.

Method C. In this method, the end-diastolic and the end-systolic silhouettes were pinned at the center of gravity of each silhouette and aligned along an axis running from the center of gravity to the midpoint of the aortic valve. In this method, regional shortening was measured along seven radial chords.

Method D. In this area-based method, each silhouette was partitioned by dividing the long axis running from the aortic-mitral valve junction to the apex by 36%, 40%, and 24% to form five regions whose areas were measured and used to calculate regional ejection fractions.

In accordance with accepted practice, the measured diagnostic parameters in methods A, B, and C were designated abnormal if their values fell more than 2 SD below the means established from the NDHD group. For these methods, this criterion also gave the best agreement with the subjective assessment results. In method D, however, better agreement was obtained with 1 SD for the threshold; consequently, for method D this value was used instead. In summary, each method was used with criteria of normality that optimized its accuracy.

Using the subjective assessment as our standard, we analyzed the data base of patients with CAD with all five methods. The resulting specificity and sensitivity figures obtained are summarized in table 4 and are shown graphically in figure 7. When Cochrane's Q-test was applied to these results, it showed that the modeling approach was significantly better than the other four methods (Q = 29.5; p < .001). Further, it was significantly better than the next best method, method D (Q = 5.3; p < .05).

Discussion

It is relatively easy to measure regional wall motion of the left ventricle during systole. Which portion of this movement is inherently caused by myocardial contraction is not obvious. Since the original study of Herman et al.² in 1967, many schemes have been proposed to objectively assess the extent of this contraction. To evaluate these methods, comparative studies have been made. Most of these methods, however, attempt to detect abnormalities by making various geometric measurements on the end silhouettes and com-

### TABLE 3

Summary of global parameters for control (NDHD) and CAD patient populations

<table>
<thead>
<tr>
<th>Parameter</th>
<th>NDHD</th>
<th>CAD</th>
<th>p value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Normalized EDV</td>
<td>67.83 ± 14.36</td>
<td>73.50 ± 19.18</td>
<td>.03</td>
</tr>
<tr>
<td>Normalized SV</td>
<td>46.33 ± 9.55</td>
<td>49.66 ± 12.74</td>
<td>ns</td>
</tr>
<tr>
<td>EF</td>
<td>0.686 ± 0.050</td>
<td>0.678 ± 0.048</td>
<td>ns</td>
</tr>
</tbody>
</table>

EDV = end-diastolic volume; SV = stroke volume; EF = ejection fraction.

*Volumes are ml/m², mean ± SD.
paring them with similar measurements made on a control group of patients with no demonstrable heart disease. Although this approach is scientifically sound, we believe that it lacks an adequate physiologic base, which is necessary to eventually understand the complex forces that cause the left ventricle to contract in a normal pattern.

Currently, subjective assessment by experienced angiographers is the only universally accepted standard. In recognizing this fact, our method attempts to simulate human expertise in quantifying abnormalities in regional wall motion. We believe that this type of "artificial intelligence" approach to the problem will assume greater importance in the future.

All known wall motion systems exhibit failure modes. Although better than the four other methods used, the modeling method is not error free. For clini-

**FIGURE 6.** Diagram showing method of registering silhouettes and measuring regional wall motion abnormalities for methods A, B, C, and D.

**TABLE 4**

<table>
<thead>
<tr>
<th>Method</th>
<th>Sensitivity (%)</th>
<th>Specificity (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>A</td>
<td>50.6</td>
<td>95.0</td>
</tr>
<tr>
<td>B</td>
<td>46.9</td>
<td>83.3</td>
</tr>
<tr>
<td>C</td>
<td>51.9</td>
<td>86.6</td>
</tr>
<tr>
<td>D</td>
<td>82.7</td>
<td>65.0</td>
</tr>
<tr>
<td>Model</td>
<td>86.4</td>
<td>80.0</td>
</tr>
</tbody>
</table>
FIGURE 7. Comparison of overall sensitivity and specificity of systems for measuring wall motion.

cal applications, these errors must be understood and taken into account. Moreover, a discussion of the type of error to which a particular method is prone can provide the basis for future improvements.

To expose potential failure modes, patient data sets used in comparative studies must have a mix of regional abnormalities as extensive as possible. In our CAD data set of 705 ventricular segments (141 for each of the five regions involved), 609 were judged to be normal. Of the remaining 96 abnormal regions, 30 occurred in the inferior region, 33 in the apical, 30 in the anterolateral, with three occurring in the inferobasal region. Unfortunately, no abnormalities were observed in the anterobasal region.

In tests for sensitivity, the modeling method failed to detect 12 abnormalities in 11 patients, resulting in an overall regional error rate of 12.5%. The breakdown of false-negative errors by region, as shown in Table 5, indicates that the largest error rates occurred in the anterior (20.0%) and the apical (12.1%) regions, with the inferior region showing a smaller error rate of 6.7%.

This result was unexpected, since in the statistical validation of the model the smallest discrepancy between the actual and the predicted stroke volumes occurred in the anterior region, with the second largest occurring in the inferior region. Moreover, all objective wall motion methods studied disagreed unanimously with the subjective standard for seven of these 11 patients, of whom six exhibited anterior wall motion abnormalities. This raises the important question of the accuracy of our standard of subjective assessment. It is well known that subjective assessment suffers from both interobserver and intraobserver variability. However, one might expect a random distribution of errors among the regions rather than a systematic error biased toward the anterior and apical regions as shown here. We considered the possibility that the anterior and apical regions are more susceptible to subjective error caused by the apical rotation of the left ventricle during systole. However, in five of the six patients noted above, the degree of apical rotation was less than 1 SD greater than the mean for the total patient population. We concluded that there was no reason to believe that apical rotation was a factor.

One of the inherent limitations of a two-frame analysis of wall motion is its inability to detect wall motion abnormalities of a temporal nature. In selecting patients for the CAD data base, no attempt was made to reject ventricles with time-based abnormalities. Certainly, some of the errors we observed may have been caused by this.

From a methodologic point of view, several types of error are possible with the modeling technique. Four ventricular landmarks must be identified by the experimenter. Two of these, the junction of the aortic and the mitral valves and the superior aspect of the aortic valve, can be located accurately. The location of the apex is not as precise but can be made without much

TABLE 5
Regional error rates for 705 ventricular segments using modeling method

<table>
<thead>
<tr>
<th>Region</th>
<th>No. of abnormal regions</th>
<th>No. of false negatives</th>
<th>Error rate (%)</th>
<th>Sensitivity</th>
<th>No. of normal regions</th>
<th>No. of false positives</th>
<th>Error rate (%)</th>
<th>Specificity</th>
</tr>
</thead>
<tbody>
<tr>
<td>Inferobasal</td>
<td>3</td>
<td>0</td>
<td>0</td>
<td></td>
<td>138</td>
<td>0</td>
<td>0</td>
<td></td>
</tr>
<tr>
<td>Inferior</td>
<td>30</td>
<td>2</td>
<td>6.7</td>
<td></td>
<td>111</td>
<td>6</td>
<td>5.4</td>
<td></td>
</tr>
<tr>
<td>Apical</td>
<td>33</td>
<td>4</td>
<td>12.1</td>
<td></td>
<td>108</td>
<td>7</td>
<td>6.5</td>
<td></td>
</tr>
<tr>
<td>Anterior</td>
<td>30</td>
<td>6</td>
<td>20.0</td>
<td></td>
<td>111</td>
<td>0</td>
<td>0</td>
<td></td>
</tr>
<tr>
<td>Anterobasal</td>
<td>0</td>
<td>0</td>
<td></td>
<td></td>
<td>141</td>
<td>0</td>
<td>0</td>
<td></td>
</tr>
<tr>
<td>All regions</td>
<td>96</td>
<td>12</td>
<td>12.5</td>
<td></td>
<td>609</td>
<td>13</td>
<td>2.1</td>
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difficulty. \(^6\) However, the inferior aspect of the mitral valve is difficult to locate precisely. This can result in errors of sufficient magnitude to push inferobasal and inferior regions back and forth across the threshold for normality depending on where an observer locates this landmark.

Another objective-subjective disagreement arises from the fact that no explicit registration of frames is used in the modeling method. As a result, ventricles exhibiting a marked systolic descent of the valvular ring would subjectively appear to have hypokinesis of the apex while presenting a normally contracted end-systolic silhouette when assessed objectively by the modeling technique.

In spite of these errors, we believe the modeling approach possesses the following important advantages over most existing methods: (1) no explicit registration of the end frames is required, (2) an easily understood and physiologically meaningful display of the left ventricle is provided for the investigator, (3) the left ventricular contraction pattern is clearly delineated into normal, hypokinetic, and hyperkinetic regions (where applicable), and (4) implementation on current computer graphic terminals is relatively easy.

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**Appendix**

The volume of a three-dimensional body with a circular cross-sectional area is directly proportional to the scale factor applied in the y-direction and to the square of the scale factor applied in the x-direction; that is,

$$ESV = (EDV) (s_y) (s_x)^2$$

where EDV = end-diastolic volume, ESV = end-systolic volume, and \(s_x\) and \(s_y\) are the respective scale factors in x and y required to generate the image at end-systole from that at end-diastole.

Substituting \(s_y = R = \text{length of long axis at end of systole/length of long axis at end of diastole and } ESV/EDV = (1 - EF)\) into the equation, we get

$$s_x = \sqrt{(1 - EF)/R}$$

where EF = ejection fraction.
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