Automatic Processing of Cineventriculograms for Analysis of Regional Myocardial Function

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SUMMARY To evaluate disorders of regional wall motion in patients with coronary artery disease, we developed a method to detect the boundary of the cineventriculograms with the aid of minicomputers. Each image of the left ventricle was transferred to computer through a flying spot scanner and stored in the disc. The endocardial margin was automatically traced along the maximum value of spatial derivatives of the silhouette density using a new algorithm we developed. Each image was then superimposed using the external markers, and radial grids were drawn from the center of the gravity of the end-diastolic silhouette to the margin of the ventricular cavity. The length of grid lines was continuously measured throughout the cardiac cycle, which provided quantitative description of the segmental centripetal motion over the entire circumference.

In 17 patients who complained of chest pain, single-plane left ventriculograms were obtained in both the resting state and immediately after cessation of rapid cardiac pacing. Patients with normal coronary arteries revealed synchronous wall motion in both states. In patients with narrowing of either one of the major branches, pacing stress increased the magnitude and extent of hypofunction or even induced abnormal wall motion in the ischemic area that showed normal contraction at rest, while there was slight enhancement of shortening in normally perfused area. In patients with three-vessel disease, wall motion over the entire ventricular surface, which was depressed before pacing, further deteriorated. Thus, the method we describe enabled precise quantification of the extent and severity of ischemic injury and of the functional status of the uninvolved myocardium. Pacing stress should be of considerable value in estimating coronary reserve.

LOCALIZED DISTURBANCES of myocardial function occur as a result of imbalance between myocardial oxygen requirements and coronary blood flow under various experimental conditions.1–4 In a clinical setting, several techniques for detecting regional myocardial function have been described.4–6 Among these, cineangiography has been regarded as the most reliable. However, quantification of such wall motion is inadequate and subject to limitations, largely because of the tedious work required for measuring and calculating chamber dimensions in each cine film.

We developed an automatic processing system for detecting boundaries of the left ventricle in cineangiograms, using a heuristic method to search for the local maximum values of the gradient of the gray levels of the images, and expressing the segmental function of the left ventricular myocardium in quantitative terms.7 We used this method to analyze the effect of rapid cardiac pacing on the myocardial shortening to determine if the temporary ischemia, provoked by increasing the oxygen demand beyond the coronary reserve, produces a regional depression of myocardial contraction. Our primary goal was to characterize the segmental function, to define the area of ischemic myocardium in patients with coronary artery disease and to elucidate the state of the myocardium that may be subject to ischemia under states of stress.

Methods

Seventeen patients, ages 33–66 years, were studied for evaluation of chest pain. The patients were in the fasting state after premedication with 10 mg of oral diazepam. Conventional diagnostic right- and left-heart catheterization was performed. Cardiac output was measured by the Fick method. After coronary angiography by the Sones technique, the first left ventriculography was performed with the patient in the 30° right anterior oblique position, injecting 25–35 ml of iohalamate sodium (80% Angio-Conray) into the ventricle at a rate of 10 ml/sec through #8F NIH closed-tip catheter with multiple sideholes. Films were exposed at a rate of 60 frames/sec with a 35-mm Arriflex cine camera mounted on a Philips 9-inch image intensifier. A calibration was made using a grid with a known dimension at the midchest level. Two lead-impregnated letters were taped to the image intensifier as fixed reference points.

A #6F USCI bipolar pacing catheter was then positioned in the right atrium. The atrial pacing was initiated at a rate of 90 beats/min and was increased in steps of 20 beats/min every 2 minutes. In patients who developed atroventricular block during atrial pacing, right ventricular pacing was substituted. Pacing was stopped with the occurrence of chest pain or ST depression in leads II or V₅. If well tolerated, pacing was continued at a rate of 150 beats/min for 6 minutes. The second angiogram was obtained immediately after the cessation of pacing in exactly the same

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1065
manner as in the control state. An adequate recovery time (at least 30 minutes) was allowed between each angiogram to prevent the depressant effect of contrast media, and left ventricular irritability was minimized by setting the onset of injection during diastole.

Analysis of the Data

Figure 1 shows the basic survey diagram of our computer system. The left ventricular images on cine film were transferred to a computer through a flying spot scanner and were stored on a disc. Each digitized image consisted of $128 \times 128$ pixels with a gray level of 256 values, which was sufficient for exploration of cineventriculography (fig. 2). The edge tracing was usually initiated at the end-diastolic frame, which was determined by the ECG directly recorded on the cine film as the frame nearest the peak of the R wave; the detected boundary of the end-diastolic frame was most accurate and reliable because it has a smooth cavity contour (fig. 3).

First, the area of search enclosed within the window (fig. 3) was determined by the operator. This procedure was necessary to avoid the noise for tracing and to shorten the processing period. A gradient image was then obtained by spatial differentiation of gray levels. Assuming that the abrupt change of the gray levels occurred at the boundary of the ventricular silhouette, the points with the maximum gradient value were traced to delineate the ventricular bound-

![Diagram](image)

**Figure 1.** Survey diagram of the image processing system. The left ventricular image on a cine film is digitized through a flying spot scanner, which can digitize $24 \times 24$ mm film into $4096 \times 4096$ pixels (maximum) with gray levels of eight bits. Minicomputer HP 2100A (32KW) executes the principal programs and HP 2108A (24KW) executes the basic image processing, such as smoothing, cutting the threshold, image addition and subtraction and linear filtering. The scan converter is made of digital IC memories with $256 \times 256$, eight-bit elements. An image stored in the scan converter is displayed on color television. The processed data can be displayed on an oscilloscope through a DA converter.

![Sequence](image)

**Figure 2.** A sequence of tracing the boundary of an end-diastolic silhouette. (upper left) An original digitized image of the ventricle. (upper right) A gradient image obtained by the spatial differentiation of gray levels in the original image. (lower left) A detected boundary. (lower right) The detected boundary superimposed on the original image.
For the algorithm for computer tracing of edge points, two weight coefficients, denoted $\alpha$ and $\beta$ in figure 4, were introduced to multiply the corresponding derivative value. Alpha is the directional weight coefficient that enables avoidance of an abrupt change in the direction of edge tracing; $\beta$ is the depth weight coefficient by which sequential information from remote points can be added. When the detected contour was mixed into the original image (fig. 2), our technique provided the proper delineation of the end-diastolic cavity of the left ventricle.

Determining the ventricular boundary becomes increasingly difficult as the ejection proceeds during systole because of rapid wall motion. In addition, the increase in resolution is required at the apex because of a large intersegment variability caused by the irregular shape of the structure.\textsuperscript{10, 11} Thus, the single processing of a given systolic frame would be more subject to error in tracing the endocardial margins. Accordingly, we added the additional global guidance from the preceding frame in the serial processing throughout the cardiac cycle. Stroke volumes calculated from the end-diastolic and end-systolic left ventricular images, processed as described by the computer, were in close agreement with those obtained by the Fick method (table 1) (mean value of 17 patients was $82 \pm 19$ ml vs $85 \pm 19$ ml, $r = 0.72$). We felt that these results verified the feasibility of our method.

The sequential ventricular silhouettes were superimposed throughout the cardiac cycle by using two external reference markers. Though studied without breath holding, the shift of the ventricular silhouette was negligible in most of the patients. However, when a patient took a deep breath during the exposure, the respiratory change was corrected by adjusting the centers of the gravity of both end-diastolic and end-systolic cavities of the two successive beats so that they would be identical. The position of the geometric center of gravity of each ventricular silhouette was examined throughout one cardiac cycle in five normal

\begin{figure}
\centering
\includegraphics[width=\textwidth]{figure3}
\caption{Gradient image of the end-diastolic frame superimposed with the detected boundaries. The position of the aortic valve was determined at the place with shortest separation of the first and the fourth segments of the detected boundary. The apex is determined at the point with the longest distance from the middle point of the aortic valve. The square is the area of search. Starting point is set at the maximum gradient value on the horizontal line given by the cursor.}
\end{figure}

\begin{figure}
\centering
\includegraphics[width=\textwidth]{figure4}
\caption{Two weight coefficients in our algorithm for computer tracing of the endocardial margins.}
\end{figure}

\begin{table}
\centering
\begin{tabular}{|c|c|c|}
\hline
Directional Weight Coefficients & Depth Weight Coefficients \\
\hline
$P_6$ & $P_1$ & $P_2$ & $P_3$ \\
\hline
$P_0$ & $P_1$ & $P_2$ & $P_3$ \\
\hline
$P_3$ & $P_1$ & $P_2$ & $P_3$ \\
\hline
$P_7$ & $P_1$ & $P_2$ & $P_3$ \\
\hline
$\alpha_3 - \alpha_2 = \alpha_4 = \alpha_5$ & $\beta_3 - \beta_2 = \beta_1$ \\
$\alpha_2 = a_4 = 7/8$, $a_1 = a_5 = 1/8$ & $\beta_3 = 8$, $\beta_2 = 7$, $\beta_1 = 6$ \\
\hline
\end{tabular}
\end{table}
Table 1. The Averaged Data of Hemodynamic Indexes and Regional Wall Motion at Rest and After Rapid Cardiac Pacing

<table>
<thead>
<tr>
<th>Pt.</th>
<th>Age (years)</th>
<th>Previous MI</th>
<th>Coronary arteriography</th>
<th>HR (beats/min)</th>
<th>LVSP/LVEDP (mm Hg)</th>
<th>CI (l/min/m²)</th>
<th>SV (ml)</th>
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<td></td>
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<td></td>
<td>Control Post-pacing</td>
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<td></td>
<td></td>
<td></td>
<td></td>
<td>67</td>
<td>78</td>
<td>106/10</td>
<td>3.4</td>
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<tr>
<td>TK</td>
<td>43</td>
<td>None</td>
<td>Normal</td>
<td>46</td>
<td>40</td>
<td>150/7</td>
<td>3.1</td>
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<td>KO</td>
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<td>Normal</td>
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<td>80</td>
<td>93/8</td>
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<td>TY</td>
<td>66</td>
<td>None</td>
<td>Normal</td>
<td>90</td>
<td>80</td>
<td>111/7</td>
<td>3.8</td>
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<tr>
<td>Mean</td>
<td>53</td>
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<td></td>
<td>67</td>
<td>67</td>
<td>135/6</td>
<td>3.7</td>
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<td>± SD</td>
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<td></td>
<td>±17</td>
<td>±17</td>
<td>8 ± 2</td>
<td>±0.7</td>
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<tr>
<td>Group 2 (narrowing of RCA)</td>
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<tr>
<td>HK</td>
<td>66</td>
<td>None</td>
<td>90% proximal RCA</td>
<td>69</td>
<td>72</td>
<td>140/12</td>
<td>3.6</td>
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<tr>
<td>TM</td>
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<td>None</td>
<td>100% RCA, 90% LCx</td>
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<td>61</td>
<td>115/4</td>
<td>2.8</td>
</tr>
<tr>
<td>TY</td>
<td>52</td>
<td>IMI</td>
<td>90% proximal RCA</td>
<td>61</td>
<td>58</td>
<td>109/10</td>
<td>2.7</td>
</tr>
<tr>
<td>Mean</td>
<td>53</td>
<td></td>
<td></td>
<td>68</td>
<td>67</td>
<td>121 ± 13/</td>
<td>3.4</td>
</tr>
<tr>
<td>± SD</td>
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<td></td>
<td>±6</td>
<td>±8</td>
<td>9 ± 3</td>
<td>±0.8</td>
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<td>Group 3 (narrowing of LAD)</td>
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<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>SN</td>
<td>64</td>
<td>AMI</td>
<td>100% LAD</td>
<td>46</td>
<td>54</td>
<td>150/19</td>
<td>2.5</td>
</tr>
<tr>
<td>TN</td>
<td>49</td>
<td>AMI</td>
<td>90% proximal LAD</td>
<td>52</td>
<td>52</td>
<td>119/8</td>
<td>3.3</td>
</tr>
<tr>
<td>HS</td>
<td>34</td>
<td>AMI</td>
<td>90% proximal LAD</td>
<td>69</td>
<td>64</td>
<td>127/10</td>
<td>2.7</td>
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<tr>
<td>NS</td>
<td>58</td>
<td>None</td>
<td>75% proximal LAD</td>
<td>72</td>
<td>69</td>
<td>116/3</td>
<td>2.7</td>
</tr>
<tr>
<td>Mean</td>
<td>51</td>
<td></td>
<td></td>
<td>60</td>
<td>60</td>
<td>128 ± 15/</td>
<td>2.8</td>
</tr>
<tr>
<td>± SD</td>
<td>±13</td>
<td></td>
<td></td>
<td>±13</td>
<td>±8</td>
<td>10 ± 7</td>
<td>±0.3*</td>
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<td>Group 4 (narrowing of three vessels)</td>
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<td></td>
</tr>
<tr>
<td>HI</td>
<td>54</td>
<td>IMI</td>
<td>100% RCA, 100% LAD, 90% LCx</td>
<td>70</td>
<td>59</td>
<td>140/8</td>
<td>2.1</td>
</tr>
<tr>
<td>KT</td>
<td>48</td>
<td>AMI</td>
<td>90% RCA, 90% LAD, 90% LCx</td>
<td>75</td>
<td>64</td>
<td>150/9</td>
<td>3.5</td>
</tr>
<tr>
<td>IS</td>
<td>50</td>
<td>IMI</td>
<td>100% RCA, 75% LAD, 75% LCx</td>
<td>87</td>
<td>81</td>
<td>98/9</td>
<td>3.2</td>
</tr>
<tr>
<td>JN</td>
<td>65</td>
<td>IMI</td>
<td>90% RCA, 75% LAD, 90% LCx</td>
<td>75</td>
<td>73</td>
<td>151/15</td>
<td>3.1</td>
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<tr>
<td>Mean</td>
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<td></td>
<td>77</td>
<td>69</td>
<td>135 ± 25/</td>
<td>3.0</td>
</tr>
<tr>
<td>± SD</td>
<td>±8</td>
<td></td>
<td></td>
<td>±7</td>
<td>±10</td>
<td>10 ± 3</td>
<td>±0.5</td>
</tr>
</tbody>
</table>

*p < 0.05 vs control.
†p < 0.01 vs control.
‡p < 0.005 vs control.
§p < 0.001 vs control.
¶p < 0.05 vs prepping value.

Abbreviations: IMI = inferior myocardial infarction; AMI = anterior myocardial infarction; RCA = right coronary artery; LCx = left circumflex artery; LAD = left anterior descending artery; HR = heart rate; LVSP = left ventricular systolic pressure; LVEDP = left ventricular end-diastolic pressure; CI = cardiac index; LVG = left ventriculography; LVEDVI = left ventricular end-diastolic volume index; EF = ejection fraction; %ΔL = percent segment shortening during systole.

Subjects. The shift of x and y coordinates of these points was minimal in normal hearts (standard deviation from the mean value was less than four pixels; one pixel roughly corresponds to 1 mm) (table 2). Thus, we chose the geometric center of gravity of the cavity as the fixed reference points to which the inward movement of the ventricular wall can be related.

In each superimposed ventricular image, 128 radial grids were drawn from the center of gravity of the end-diastolic silhouette to the endocardial margin (figs. 5–7). Again, we selected 128 radii to match the resolution to the original image stored in the disc file with 128 words. Ninety of these 128 radii covered the outline of the left ventricular cavity, excluding the area of aortic and mitral valves, which was divided into five sections, fixing the midpoint at the apex. Accordingly, 18 radial grids were included in each section, which roughly corresponded to five segments (anterobasal, anterolateral, apical, diaphragmatic and posterobasal) defined in a reporting system described by the American Heart Association.12 Measurement of the length of each grid line throughout cardiac cycles allowed for analysis of contraction and relaxation on specific segments of the left ventricular myocardium.
TABLE 1. (Continued)

<table>
<thead>
<tr>
<th>LVEDVI (ml/m²)</th>
<th>EF (%)</th>
<th>Mean %ΔL in each area</th>
</tr>
</thead>
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<tr>
<td></td>
<td></td>
<td>1</td>
</tr>
<tr>
<td>Control</td>
<td>Post-pacing</td>
<td>Control</td>
</tr>
<tr>
<td>61</td>
<td>59</td>
<td>83</td>
</tr>
<tr>
<td>58</td>
<td>57</td>
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<td>78</td>
<td>76</td>
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<td>62</td>
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<td>77</td>
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<td>71</td>
</tr>
<tr>
<td>67</td>
<td>70</td>
<td>75</td>
</tr>
</tbody>
</table>

(Continues)

Table 2. Position of the Geometric Center of Gravity Throughout One Cardiac Cycle in Normal Subjects

<table>
<thead>
<tr>
<th>Case</th>
<th>x coordinate</th>
<th>y coordinate</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>60 ± 4</td>
<td>56 ± 1</td>
</tr>
<tr>
<td>2</td>
<td>70 ± 3</td>
<td>52 ± 1</td>
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<tr>
<td>3</td>
<td>72 ± 1</td>
<td>53 ± 3</td>
</tr>
<tr>
<td>4</td>
<td>78 ± 3</td>
<td>56 ± 1</td>
</tr>
<tr>
<td>5</td>
<td>70 ± 1</td>
<td>53 ± 0</td>
</tr>
</tbody>
</table>

Values are mean ± sd in pixels (1 pixel = 1 mm).

(Continues)

(cups, 5–7). Stroke excursion from end-diastole to end-systole was corrected by the end-diastolic length and was expressed as percent shortening. The segmental centripetal motion expressed as percent shortening was plotted over the entire circumference (figs. 5–8). The time course of each radial length was alternatively stacked from the lateral margin of the aortic cusp to the anterior margin of the mitral valve (figs. 5–7). This presentation enabled assessment of the size of the area which involved the disturbed wall motion.

The cine film processing time required for one series for a complete study was 40–60 minutes. Percent shortening was averaged in each of the five sections of the left ventricular wall and expressed as mean ± sd. All statistical comparisons were made using an unpaired t test.

Results

Of the 17 patients, five had no evidence of ischemic heart disease by coronary arteriography (group 1), four had 75% or greater intraluminal diameter reduction of the right coronary artery or left circumflex artery or both (group 2), four had 75% or greater narrowing of the left anterior descending artery (group 3) and four had a three-vessel involvement (group 4). The averaged hemodynamic data and the
measures of regional wall motion at rest and after rapid cardiac pacing in these four groups of patients are given in table 1.

Group 1

All five patients revealed a normal cardiac index, left ventricular end-diastolic pressure, volume, and ejection fraction at rest. Average percent shortening of radial grids was largest at the anterobasal wall (area 1), decreasing gradually toward the posterobasal wall (area 5) in the control state (fig. 8). At the second study, after cessation of cardiac pacing, heart rate remained the same in both states. The cardiac size and segmental shortening were also similar, with average ejection fractions of 75% and 78%, respectively.

Data from one patient are shown in figure 5. This patient showed no remarkable changes in the superimposed configuration of the left ventricular cavities at end-diastole and end-systole or instantaneous relative change of the length of the three representative radials. When centripetal wall motion expressed as the percent shortening of each radial grid was plotted over the entire wall, shortening was slightly augmented in the inferior wall after pacing. In the three-dimensional display obtained by stacking serial plots of movement against time for each radial grid, an identical synchronicity of the valleys and peaks was observed in both the basal and postpacing states.

Group 2

In the four patients in this group, heart rate did not differ in the control and postpacing studies. After pacing, the total ejection tended to decrease despite a slight increase in the size of cardiac chamber. Rapid cardiac pacing induced severe hypokinesis in the inferior wall; even when the motion was normal in the basal state, the average percent shortening was decreased by 18% in area 4 and by 7% in area 5 (fig. 6). These changes were more clearly represented in the three-dimensional configuration. Depression of the segmental wall motion was dominant in the inferior wall in the distal part, while valleys and peaks were...
Figure 6. Same format as in figure 5. Data from one patient with severe right coronary stenosis. Note the deterioration of the inferior wall motion and a compensatory augmentation of the anterior wall motion.

Figure 7. Same format as in figure 5. Data from one patient with three-vessel involvement. Note the depression of wall motion in all areas after the pacing.
cineangiograms of the left ventricle into a videomat and successfully obtained the real-time detection of the outlines. However, to achieve complete extraction of the ventricular image, these investigators still needed minor manual intervention with a writing tablet. Contrary to these earlier works, we used a flying spot scanner to obtain the digitized image and established completely automated outlining of the ventricular cavity. Chow and Kaneko also used a flying spot technique, but extracted objects from an irrelevant background through a dynamic threshold method. This method is valuable for images of low quality because thresholds can be set dynamically according to the local characteristics. Clayton et al. developed a method for detecting boundaries, based on the property that photometric intensities at or near boundaries change faster than in other regions. We also used this approach, applying spatial differentiation to images to separate objects from the background. However, this method is not feasible when noise is dominant because of its enhancement of high spatial components by the differentiation process. This disadvantage could be compensated for by improving the image quality.

The algorithm of the boundary tracing in our study consists of two major factors to multiply the derivative value. First, at a given edge point on the digitized image, a candidate for the next edge point is assumed to have the maximum value of the spatial derivative of the gray levels of the image; the edges traced are generally smooth, without any abrupt deviation of the curvature; and the edge lines do not cross. Second, the heart wall is assumed to contract homogeneously, taking into account the local maximum derivative value in the vicinity and sequential information from the remote edge points. Furthermore, the boundary of the preceding frame is always referred to as a global guidance. These procedures closely imitate a visual detection of a boundary by the human eye, and these detected boundaries are similar to those obtained by the conventional manual method.

Although direct and detailed information for the wall motion was obtained by the left ventricular cineangiogram, contractile abnormalities were originally diagnosed qualitatively, based on visual inspection of the cavity silhouettes. Several attempts have been made to express such regional wall motion abnormalities in more quantitative terms. The precise separation of normal and abnormal wall motion requires proper superimposition of sequential ventricular silhouettes throughout ventricular ejection. In the present study, each left ventricular silhouette was superimposed on the end-diastolic film using two fixed external reference markers. This method is a more reliable reference system than the conventional use of the midpoint of the left ventricular long axis or the midpoint of the aortic valve plane, in representing abnormalities in wall motion in the segments in the location of a myocardial infarction, as predicted by coronary arteriogram and ECG together with the normal function in all other segments corresponding to the normally perfused area.

FIGURE 8. Data from eight patients with normal coronary arteriograms. Data from three patients who underwent cineventriculogram only in the basal state were added. The solid line represents the mean value of percent shortening at each grid line in the basal state and the shaded area shows 1 standard deviation above and below the mean values.

more exaggerated in the anterior wall in the proximal part.

Group 3

The changes in cardiac size and total left ventricular function were directionally the same as in group 2. Anteroapical wall (areas 1, 2 and 3) motion, which had been already depressed in the basal state, further deteriorated after the rapid cardiac pacing, with the average percent shortening decreasing by 6–9%. Inferior wall (areas 4 and 5) motion was either unchanged or slightly enhanced by pacing.

Group 4

Heart rate after pacing decreased significantly, from 77 to 69 beats/min in group 4. The motion over the entire ventricle, which had been depressed before pacing, was further decreased by pacing stress (fig. 7).

Discussion

To analyze dimensional changes in the left ventricular cavity, several workers have attempted to design an automated method of boundary detection to replace the time-consuming manual input system that makes use of a light pen. Tasto developed a fully automatic approach to find an approximate boundary of the left ventricle, using a measurement for brightness change; however, the detected boundaries were irregular and far from the reality. Gaudeau et al. built an optical scanner using photocells and automatically determined the boundary of the opacified image using an adaptive filter. Slager et al. converted
Another problem in quantifying the regional motion of the left ventricular wall is the definition of reference point to which the inward movement can be related. The direction of ventricular wall motion was much closer to natural behavior for all areas if it was expressed along radial lines to the midpoint of the long axis or to the geometric center of gravity of the end-systolic frame. It is generally accepted that the spatial movement of the geometric center of gravity is minimal in normal ventricles, which move symmetrically in a concentric fashion. In the present study, the serial plots of the geometric center of gravity of all ventricular images on the single X-Y plane showed that these points actually did move in a normal heart, but the magnitude of the shift was negligible. However, in the ventricles with wall motion abnormalities, this point moves toward an akinetic or dyskinetic area. We chose the geometric center of gravity of the end-diastolic frame as the fixed reference point and measured changes of the length of related radials of all other frames to analyze the chordal shortening. Thus, with this method, asynchronous contraction was related to the shift of the geometric center of gravity during ventricular systole.

Rapid cardiac pacing provokes temporary ischemia in the myocardium potentially subjected to ischemia during a stressed state. Such pacing induces an increase in the heart rate and is considered to increase metabolic requirements of the regional myocardium exceeding the capacity of the diseased coronary arteries to augment oxygen delivery. Therefore, cardiac pacing has been widely used clinically as a valuable diagnostic test for the detection of coronary artery disease, to elicit acute reversible abnormalities in segmental contraction that are usually absent at rest but would occur when oxygen requirements exceed the fairly constant level.

Recently, the effects of rapid cardiac pacing on regional myocardial function were studied in conscious dogs instrumented with ultrasonic crystals during limited coronary blood flow by partial coronary artery constriction. Of particular interest in this study was the behavior of beats after the termination of pacing. An increase in HR results in an increase in myocardial contractility, according to the Bowditch phenomenon. After cessation of pacing, control segment shortening followed the typical dissipation pattern of this potentiation, which was improved initially with subsequent exponential decay in the sequential several beats. In the ischemic segment, the initially improved shortening deteriorated more rapidly, showing severely depressed function at 5 seconds after pacing and gradually returning to control levels before pacing over the next 5 minutes. The postpacing deterioration of ischemic segment shortening may be related to the interaction of the dissipation of poststimulation potentiation and the augmented oxygen requirements that result from sustained wall tension in the stretched ischemic myocardium and the failure of quick recovery from myocardial ischemia in coronary stenosis. In the present study, the second ventriculogram was obtained immediately after pacing was stopped and beats from 5 seconds to 1 minute were analyzed where the physiologic consequences of induced tachycardia had already disappeared; nevertheless, the ischemic myocardial shortening was considerably depressed.

We expressed the segmental function of a given radial grid as a percent shortening from the end-diastolic length. This method allows for a comparable characterization of regional segmental function, as shown in animal experiments with ultrasonic dimension gauges for the entire perimeter of the left ventricular cavity.

Compared with the control group, in which the pacing effect was no longer significant at the time of study, in those with critical coronary stenosis, the clear depression of wall motion occurred in the area corresponding to the known coronary lesions, although normal synchronous shortening was present or systolic shortening was preserved, to some extent, in the resting state. However, the segmental function of a normally perfused area in such ventricles tended to be augmented by the increased end-diastolic length. This may be related to the compensatory operation of the Frank-Starling mechanism. The generalized hypofunction was further depressed by pacing stress in cases with three-vessel disease.

Thus, the method we describe helps in precise quantification of the extent and severity of ischemic injury and enables assessment of the functional status of the uninvolved myocardium. Quantification of the responsiveness of regional myocardium to pacing stress should be of considerable value in estimating the coronary reserve and represents the potential effectiveness of subsequent therapeutic interventions.

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