Regional Left Ventricular Function Assessed by Contrast Angiography in Acute Myocardial Infarction

MICHEL RIGAUD, M.D., PAULO ROCHA, M.D., JACQUES BOSCHAT, M.D., JEAN C. FARCOT, M.D., JEAN BARDET, M.D., AND JEAN P. BOURDARIAS, M.D.

SUMMARY  The relationship of segmental left ventricular (LV) wall motion abnormalities to LV function 2–6 days after acute transmural myocardial infarction (MI) was investigated in 45 patients by quantitative contrast ventriculography. Patients were divided into four classes according to the MIRU criteria. Segmental wall motion was assessed by determining the percentage of systolic shortening (ΔS) along nine hemiaxes and the extent of akinetic or dyskinetic abnormally contracting segments (% ACS) expressed as a percentage of end-diastolic perimeter.

When compared with that in 17 normal control subjects, the LV end-diastolic volume was increased only in patients in class III and class IV; the LV end-systolic volume increased progressively from normal through class IV. Ejection fraction had a negative linear correlation with %ACS (r = 0.97). The size of ACS was larger in anterior (34 ± 14%) than in inferior MIs (23 ± 7%), resulting in greater LV dysfunction. However, for a comparable size of ACS, infarct location alone did not influence LV function parameters. In the noninfarcted zone, ΔS was increased when the size of ACS was < 25% and reduced when the size of ACS was > 25%.

Thus, the size of ACS is a major determinant of LV dysfunction in acute MI. The compensatory mechanisms operate either through an augmented mechanical function of residual myocardium when the infarct is small, or through the Frank-Starling mechanism when the infarct is large.

SEGMENTAL LEFT VENTRICULAR (LV) wall motion abnormalities are a common consequence of acute myocardial infarction (MI). Depending on the magnitude of the regional dysfunction, a wide spectrum of global LV failure develops, ranging from a clinically unrecognized condition to power failure and its extreme manifestation, cardiogenic shock. Although segmental time-motion echocardiography,1–4 real-time, two-dimensional echocardiography,5–6 and radionuclide angiography1–9 might provide means to localize and quantify MI and LV function in the early stages of acute infarction, these techniques are not without potential errors.10 They have not been applied systematically, and require validation against accepted standards. Definition of the pattern of regional LV contraction may be obtained with contrast angiography, but the fear of risk that might be associated with left ventriculography has limited its use during the acute phase of MI. Recently, a number of patients with both uncomplicated11–13 and complicated14–17 acute MI have undergone coronary arteriography and left ventriculography without evidence of increased morbidity. Similarly, patients with evolving18 or recent subendocardial MI19 have been studied hemodynamically without complications.

In this study we report results of early stage contrast left ventriculography performed without complications in patients with acute MI, in most of whom surgical intervention was being considered. Special attention is focused on 1) the influence of both the extent and location of the segmental disease on the overall LV function, and 2) the tendency of noninfarcted myocardium to compensate for the depressed function of the acutely infarcted myocardium.

Materials and Methods

The study group consisted of 62 patients — 45 with acute MI and 17 normal control subjects. The diagnosis of acute MI was made on the basis of a typical history of chest pain, serial ECG and changes in cardiac enzymes. Development of Q waves (40-msec duration) in leads I, aV_{1}, V_{1}–aV_{6} was considered to represent anterior MI (24 patients); Q waves in leads II, III, aV_{F} represented inferior MI (15 patients). Six patients had both anterior and inferior MI. Seventeen patients had angiographically normal coronary arteries and normal LV volumes, ejection fraction (EF) and end-diastolic pressure (table 1) and served as control subjects. The patients with acute MI were divided into four groups according to the standard MIRU criteria: 19 patients had no signs of heart failure (class I); eight had mild-to-moderate heart failure (class II); six had overt pulmonary edema (class III) and 12 were in cardiogenic shock (class IV). Only class IV included patients who had had previous MI.

Cardiac catheterization was performed 2–6 days after the onset of MI. Selective coronary arteriography in addition to left ventriculography was obtained in 18 patients: 12 in cardiogenic shock, and six class III patients considered to be in a "pre-
<table>
<thead>
<tr>
<th>Infarct location</th>
<th>HR (beats/min)</th>
<th>EDP (mm Hg)</th>
<th>EDV (ml/m²)</th>
<th>ESV (ml/m²)</th>
<th>SI (ml/m²)</th>
<th>EF (%)</th>
<th>ACS (%)</th>
<th>Mean ΔS (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Class I</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>A</td>
<td>78</td>
<td>19</td>
<td>86</td>
<td>50</td>
<td>36</td>
<td>42</td>
<td>29</td>
<td>22</td>
</tr>
<tr>
<td>A+I</td>
<td>71</td>
<td>18</td>
<td>94</td>
<td>46</td>
<td>34</td>
<td>54</td>
<td>28</td>
<td>24</td>
</tr>
<tr>
<td>A</td>
<td>65</td>
<td>13</td>
<td>63</td>
<td>29</td>
<td>38</td>
<td>58</td>
<td>23</td>
<td>29</td>
</tr>
<tr>
<td>A</td>
<td>89</td>
<td>16</td>
<td>65</td>
<td>29</td>
<td>38</td>
<td>58</td>
<td>23</td>
<td>30</td>
</tr>
<tr>
<td>A</td>
<td>79</td>
<td>12</td>
<td>63</td>
<td>25</td>
<td>38</td>
<td>60</td>
<td>14</td>
<td>30</td>
</tr>
<tr>
<td>A</td>
<td>76</td>
<td>10</td>
<td>59</td>
<td>22</td>
<td>37</td>
<td>63</td>
<td>20</td>
<td>31</td>
</tr>
<tr>
<td>A</td>
<td>68</td>
<td>14</td>
<td>63</td>
<td>22</td>
<td>41</td>
<td>65</td>
<td>19</td>
<td>39</td>
</tr>
<tr>
<td>A</td>
<td>95</td>
<td>19</td>
<td>83</td>
<td>46</td>
<td>37</td>
<td>45</td>
<td>31</td>
<td>23</td>
</tr>
<tr>
<td>A</td>
<td>90</td>
<td>20</td>
<td>92</td>
<td>44</td>
<td>37</td>
<td>52</td>
<td>28</td>
<td>37</td>
</tr>
<tr>
<td>A</td>
<td>66</td>
<td>18</td>
<td>68</td>
<td>31</td>
<td>37</td>
<td>54</td>
<td>28</td>
<td>26</td>
</tr>
<tr>
<td>A</td>
<td>70</td>
<td>18</td>
<td>77</td>
<td>34</td>
<td>43</td>
<td>56</td>
<td>26</td>
<td>31</td>
</tr>
<tr>
<td>A</td>
<td>50</td>
<td>14</td>
<td>80</td>
<td>34</td>
<td>36</td>
<td>58</td>
<td>25</td>
<td>33</td>
</tr>
<tr>
<td>A</td>
<td>49</td>
<td>20</td>
<td>117</td>
<td>46</td>
<td>71</td>
<td>61</td>
<td>23</td>
<td>32</td>
</tr>
<tr>
<td>I</td>
<td>50</td>
<td>20</td>
<td>66</td>
<td>28</td>
<td>38</td>
<td>61</td>
<td>20</td>
<td>38</td>
</tr>
<tr>
<td>I</td>
<td>92</td>
<td>15</td>
<td>64</td>
<td>25</td>
<td>39</td>
<td>61</td>
<td>17</td>
<td>42</td>
</tr>
<tr>
<td>I</td>
<td>81</td>
<td>15</td>
<td>66</td>
<td>30</td>
<td>36</td>
<td>55</td>
<td>15</td>
<td>30</td>
</tr>
<tr>
<td>I</td>
<td>72</td>
<td>16</td>
<td>72</td>
<td>25</td>
<td>47</td>
<td>65</td>
<td>15</td>
<td>41</td>
</tr>
<tr>
<td>I</td>
<td>84</td>
<td>9</td>
<td>73</td>
<td>21</td>
<td>52</td>
<td>71</td>
<td>10</td>
<td>40</td>
</tr>
<tr>
<td>Class II</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>A</td>
<td>87</td>
<td>18</td>
<td>87</td>
<td>64</td>
<td>23</td>
<td>26</td>
<td>50</td>
<td>12</td>
</tr>
<tr>
<td>A</td>
<td>100</td>
<td>28</td>
<td>84</td>
<td>53</td>
<td>31</td>
<td>37</td>
<td>30</td>
<td>18</td>
</tr>
<tr>
<td>A</td>
<td>64</td>
<td>14</td>
<td>98</td>
<td>36</td>
<td>42</td>
<td>43</td>
<td>30</td>
<td>24</td>
</tr>
<tr>
<td>A</td>
<td>108</td>
<td>26</td>
<td>76</td>
<td>38</td>
<td>38</td>
<td>50</td>
<td>30</td>
<td>27</td>
</tr>
<tr>
<td>I</td>
<td>106</td>
<td>24</td>
<td>87</td>
<td>55</td>
<td>32</td>
<td>37</td>
<td>38</td>
<td>21</td>
</tr>
<tr>
<td>I</td>
<td>97</td>
<td>18</td>
<td>75</td>
<td>40</td>
<td>35</td>
<td>47</td>
<td>29</td>
<td>23</td>
</tr>
<tr>
<td>I</td>
<td>81</td>
<td>20</td>
<td>60</td>
<td>29</td>
<td>31</td>
<td>52</td>
<td>28</td>
<td>29</td>
</tr>
<tr>
<td>I</td>
<td>66</td>
<td>12</td>
<td>91</td>
<td>41</td>
<td>50</td>
<td>55</td>
<td>23</td>
<td>27</td>
</tr>
<tr>
<td>Class III</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>A</td>
<td>100</td>
<td>28</td>
<td>96</td>
<td>76</td>
<td>20</td>
<td>21</td>
<td>59</td>
<td>9</td>
</tr>
<tr>
<td>A</td>
<td>100</td>
<td>28</td>
<td>109</td>
<td>81</td>
<td>28</td>
<td>26</td>
<td>54</td>
<td>12</td>
</tr>
<tr>
<td>A</td>
<td>90</td>
<td>20</td>
<td>110</td>
<td>79</td>
<td>31</td>
<td>28</td>
<td>50</td>
<td>11</td>
</tr>
<tr>
<td>A</td>
<td>76</td>
<td>20</td>
<td>104</td>
<td>71</td>
<td>33</td>
<td>32</td>
<td>52</td>
<td>14</td>
</tr>
<tr>
<td>A</td>
<td>86</td>
<td>37</td>
<td>80</td>
<td>51</td>
<td>29</td>
<td>36</td>
<td>38</td>
<td>19</td>
</tr>
<tr>
<td>A</td>
<td>83</td>
<td>27</td>
<td>97</td>
<td>61</td>
<td>36</td>
<td>37</td>
<td>37</td>
<td>19</td>
</tr>
<tr>
<td>Class IV</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>A</td>
<td>103</td>
<td>35</td>
<td>184</td>
<td>178</td>
<td>6</td>
<td>7</td>
<td>85</td>
<td>1</td>
</tr>
<tr>
<td>A</td>
<td>105</td>
<td>30</td>
<td>162</td>
<td>153</td>
<td>9</td>
<td>9</td>
<td>85</td>
<td>1</td>
</tr>
<tr>
<td>A</td>
<td>110</td>
<td>27</td>
<td>188</td>
<td>168</td>
<td>20</td>
<td>16</td>
<td>73</td>
<td>1</td>
</tr>
<tr>
<td>A</td>
<td>100</td>
<td>26</td>
<td>117</td>
<td>100</td>
<td>17</td>
<td>15</td>
<td>67</td>
<td>5</td>
</tr>
<tr>
<td>A + I</td>
<td>55</td>
<td>28</td>
<td>146</td>
<td>123</td>
<td>23</td>
<td>16</td>
<td>67</td>
<td>4</td>
</tr>
<tr>
<td>A + I</td>
<td>86</td>
<td>27</td>
<td>115</td>
<td>93</td>
<td>22</td>
<td>19</td>
<td>64</td>
<td>7</td>
</tr>
<tr>
<td>A + I</td>
<td>63</td>
<td>25</td>
<td>185</td>
<td>125</td>
<td>40</td>
<td>19</td>
<td>64</td>
<td>10</td>
</tr>
<tr>
<td>A + I</td>
<td>90</td>
<td>22</td>
<td>125</td>
<td>99</td>
<td>26</td>
<td>21</td>
<td>63</td>
<td>7</td>
</tr>
<tr>
<td>A</td>
<td>112</td>
<td>20</td>
<td>122</td>
<td>97</td>
<td>25</td>
<td>20</td>
<td>60</td>
<td>11</td>
</tr>
<tr>
<td>A</td>
<td>80</td>
<td>26</td>
<td>117</td>
<td>82</td>
<td>35</td>
<td>20</td>
<td>56</td>
<td>10</td>
</tr>
<tr>
<td>A + I</td>
<td>59</td>
<td>29</td>
<td>118</td>
<td>89</td>
<td>29</td>
<td>25</td>
<td>47</td>
<td>12</td>
</tr>
<tr>
<td>A</td>
<td>64</td>
<td>30</td>
<td>128</td>
<td>89</td>
<td>39</td>
<td>30</td>
<td>48</td>
<td>13</td>
</tr>
</tbody>
</table>

| Abbreviations: HR = heart rate; EDP = end-diastolic pressure; EDV = end-diastolic volume; ESV = end-systolic volume; SI = stroke index; EF = ejection fraction; ACS = extent of abnormally contracting segments; mean ΔS = mean systolic shortening along the nine hemixaxes; A = anterior; I = inferior. |
shock” state according to the criteria suggested by O'Rourke et al. The remaining patients were requested to enter the study voluntarily; they agreed to undergo left ventriculography during the acute phase. Each patient gave informed consent. There were no complications of catheterization in the 45 patient studied. In all class IV patients, the ventriculographic study was performed after 24–36 hours of circulatory assist (intraaortic balloon pumping), at which time the shock state was reversed in most of the patients.

Left cineventriculography was performed in the 30° right anterior oblique (RAO) projection using a 35-mm film taken at 60 frames/sec, and a Philips 22.5-cm image-amplifier system. In all class IV patients and in patients with inferior MI, a second ventriculography was performed 20 minutes later in the left anterior oblique (LAO) projection. Ventricular opacification was accomplished with 50–70 ml Radioselectan, containing 76% sodium and methylglucamine diatrizoates, injected at a pressure of 4.10² KPa through a pigtail catheter. The first cardiac beat in which the LV cavity was completely opacified by the contrast medium (at least two beats after a premature ventricular depolarization) was used to draw the end-diastolic and end-systolic silhouettes of the chamber. End-systolic silhouettes were drawn on the last frame that showed inward motion of the contractile portion of the left ventricle.

Analysis of LV Contraction Abnormalities

Assessments of the location and quantification of the extent of abnormal segmental contraction was generally obtained from the RAO projection. However, in patients with cardiogenic shock or with inferior MI, the LAO projection was also analyzed for the absence of asynery in the posterobasal zone not visualized in the RAO projection. Based on the angiographic evaluation and absence of Q waves in leads V₇-V₉, no patient with involvement of the posterobasal segment was included in this study. Using the data of Feild et al., we found that in 17 patients with single infarction, the relative size of the akinetic (or dyskinetic) segment determined with biplane or single-plane angiography was not statistically different (t = 0.631).

To properly define localized segments of abnormal ventricular contraction and their extent, end-diastolic and end-systolic outlines of the LV chamber in the RAO projection were superimposed according to the technique of Leighton et al., with correction for the apical rotation. Dyskinesis (paradoxic movement) was considered to be present whenever the end-systolic silhouette extended outside the end-diastolic silhouette. Akinesis was considered to be present whenever the two silhouettes overlapped. The term “abnormally contracting segments” (ACS) signified either of these two localized disorders of LV wall motion. The cineangiographic end-diastolic perimeter and length of the akinetic or dyskinetic segment were determined, and ACS was expressed as a percentage: %ACS = (akinetic or dyskinetic length of end-diastolic circumference/total end-diastolic circumference) × 100. Four equidistant lines were constructed perpendicular to the end-systolic longitudinal axis, providing eight hemiaxes — four on the anterolateral wall and four on the inferior wall. In addition, the apical systolic shortening was measured as a percentage of the distance from the end-diastolic position of the apex to the most apical perpendicular hemiaxis. The percent of systolic motion (%ΔS) was determined for each of the nine hemiaxes (1–4, anterior wall; 5, apex; 6–9, inferior wall) and averaged (mean %ΔS). Hypokinetic (or hyperkinetic) segments were defined as zones in which %ΔS was significantly different from that observed in normal control subjects. As noted in another study, hemiaxis 9 exhibited much variability in systolic shortening among normal patients (range 16–55%), perhaps because it included portions of the descending mitral valve. Consequently, contraction abnormality at this site (akinesis excepted) should be interpreted with caution.

Left ventricular volumes were estimated in the RAO projection using Simpson's rule. EF was expressed as the ratio of ventricular stroke volume to end-diastolic volume. LV pressures were measured using fluid-filled catheters and Statham P23Db strain gauge transducers. End-diastolic pressure was read from high-amplification pressure tracings.

Statistical Analysis

Data were analyzed and treated statistically using the t test for paired and unpaired data as well as standard regression analysis. Since the relationships between several parameters were generally not linear, correlations were expressed as r² values (square of the correlation coefficients). In the cases of curvilinear or higher order polynomial relationship, the hypothesis of linearity was tested and rejected at the 0.05 level using an F test.

Results

The clinical and laboratory data (mean ± sd) are presented in table 1. The LV end-diastolic pressure (LVEDP) was higher in the MI group than in normal control subjects, and increased progressively from class I through class III. However, LVEDP was not statistically different in class III and IV. The LV end-diastolic volume (LVEDV) was within the normal range in class I and class II, but was moderately higher in class III and markedly increased in class IV. The LV end-systolic volume (LVESV) increased progressively from control through class IV, while the stroke index gradually diminished. EF was reduced in all four classes compared with normals. The %ACS was 21.4 ± 6.4% in class I, and 33.4 ± 9%, 48 ± 9.4%, 64.9 ± 12.1%, respectively, in classes II, III and IV. Similarly, the mean systolic shortening of the nine hemiaxes decreased successively from controls (45.4 ± 6.1) to class I (32.5 ± 6.3), class II (22.6 ± 5.6), class III (14 ± 4.2) and class IV (6.8 ± 4.4). Mean systolic shortening of the nine hemiaxes was highly correlated with %ACS (r = 0.95).

Figure 1 shows that LVEDP correlated with
LVEDV \( (r = 0.61), \) but correlated better with LVESV \( (r = 0.74) \) and stroke index \( (r = 0.75) \). There was a good linear correlation noted between LVEDP and EF \( (r = 0.87), \) %ACS \( (r = 0.88) \) and mean systolic shortening of the nine hemiaxes \( (r = 0.86). \) Since LVEDV and LVESV were linearly related (fig. 2), a hyperbolic function was expected to describe the relationship between EF and LVEDV \( (r^2 = 0.73). \)

Figure 3 shows a graph of EF vs %ACS and the derived linear regression. The slope of this regression line significantly exceeded the slope of the theoretical line \( (p < 0.001) \) drawn between the average normal EF \( (\%\text{ACS} = 0) \) and zero EF \( (\%\text{ACS} = 100) \). In particular, most of the patients with ACS < 25% clustered above, while those with ACS > 25% were below the theoretical line. LVEDV and LVESV had significantly parabolic correlations with ACS (fig. 2) — LVEDV = 0.0201 ACS - 0.044 ACS^2 + 76.5 \( (r^2 = 0.75); \) LVESV = 0.0203 ACS^2 + 0.0214 ACS + 23.3 \( (r^2 = 0.924). \)

In all patients with a single infarction (figs. 4 and 5), the sites of the infarction assessed by contrast ventriculography corresponded well with those derived from the ECG. Thus, in inferior MI, %ΔS was markedly decreased along the inferior hemiaxes 6–9, while systolic shortening remained in the normal range along the anterior hemiaxes 1–5. Although hemiaxis 9 may include some portion of the mitral valve, its systolic shortening was equal to or greater than the lower normal limit \( (16\%) \) in only five patients, and was lower \( (\text{range } 0–12\%) \) in the other 10 patients. In patients with anterior MI, %ΔS was markedly depressed along hemiaxes 1–5, and normal along the inferior hemiaxes 6–9. Apical involvement was noted only in the presence of anterior MI. In patients with cardiogenic shock, %ΔS was reduced in all the hemiaxes, except in the superior portion of the anterolateral wall and in the region close to the mitral valve (fig. 6). All 12 patients with cardiogenic shock had paradoxical systolic expansion in segments 3–5, compared with only four out of the 18 patients with anterior MI and none of the 15 patients with inferior MI.

The %ACS was greater in anterior \( (34 \pm 14\%) \) than in inferior MI \( (28 \pm 7\%, p < 0.02), \) and LVESV, stroke index and EF were significantly different in these groups \( (p < 0.02) \) (fig. 7). In none of the patients with inferior MI did the %ACS involve more than 40% of the end-diastolic perimeter, vs five of the 18 patients with anterior MI. When these latter five patients were excluded, two subgroups were formed with comparable %ACS, i.e., 26 ± 9% \( (\text{range } 11–39\%) \) in the anterior MI group and 23 ± 7% \( (\text{range } 10–38\%) \) in the inferior MI group. These two subsets were then equivalent in terms of basic LV function parameters. In the overall grouping of patients, regardless of size and anterior or inferior location of the infarct, systolic shortening along hemiaxes facing the noninfarcted area did not differ significantly from that of the corresponding hemiaxes in normal subjects. However,
when the patients were subdivided into two subsets depending on %ACS being less or more than 25% of the LV perimeter, the residual noninfarcted myocardium was found to be hyperkinetic when %ACS was ≤ 25%, and normokinetic or frequently hypokinetic when %ACS was > 25% (figs. 4 and 5).

Discussion

In the current cineangiographic study of segmental LV function in the early stage of acute MI, the extent of regional akinesis (or dyskinesis) correlated well with clinical indicators of the severity of heart failure.
Figure 4. Systolic wall motion in inferior myocardial infarction. $\Delta S$ = systolic shortening (mean ± SD) along each of the nine hemiaxes. The shaded area represents 1 SD. Top) In inferior myocardial infarction (closed circles) systolic shortening was severely depressed in the infarcted zone (hemiaxes 6-9), while it remained within the normal range (open circles) in the noninfarcted zone. Bottom) The normal range (open circles) is also shown. In patients with an extent of abnormally contracting segments (%ACS) ≤ 25% (closed circles), systolic shortening in the noninfarcted zone (hemiaxes 1-4) was supernormal, while it was significantly reduced in patients with a %ACS > 25% (closed triangles).

Figure 5. Systolic wall motion in anterior myocardial infarction. Top) In anterior myocardial infarction (closed circles), systolic shortening ($\Delta S$) was markedly reduced in the infarcted zone (hemiaxes 1-5), while it was within the normal range (open circles) in the noninfarcted zone. %ACS = percent abnormally contracting segments.
and the degree of altered global ventricular function. Patients usually had symptoms of heart failure whenever the %ACS exceeded 30%. The slight degree of the overlap between ACS in different clinical classes might be attributed to the subjective nature of the clinical classification. In patients without or with only mild heart failure, the finding of increased LVEDP in the presence of normal LVEDV suggests a reduction in global LV compliance, presumably due to the increased stiffness of the infarcted myocardium.\textsuperscript{24-26} However, in patients with severe pump failure, the increase in LV filling pressure was associated with an increased LVEDV. The greatest and most frequently observed geometric abnormality in LV function was an increased end-systolic volume and a decreased EF. This is obviously the result of the infarction causing a regional loss of contractile function,\textsuperscript{26} and is also consistent with the finding of a close correlation between %ACS and end-systolic volume. Our results, as well as those of other investigators,\textsuperscript{3, 7, 8, 21} suggest that diminished mean systolic fiber shortening is the earliest and primary alteration of the LV function in patients with acute MI. Detailed analysis of the regression lines showed that EF decreased when %ACS exceeded 14%, LVEDP rose above 12 mm Hg when %ACS exceeded 11% and LV volume exceeded 90 ml/m\(^2\) when the %ACS was greater than 40%.

The finding of a highly significant negative correlation between %ACS and LVEF supports the experimental work of Pairolero et al.,\textsuperscript{27} who found a close, linear relationship between the size of an akinetic segment and EF. Feild et al.\textsuperscript{21} noted a cubic relationship between EF and percentage of noninfarcted myocardium measured during the recovery phase of acute transmural infarction. More recently, Rigo et al.\textsuperscript{7} showed that in patients within 48 hours of the onset of infarction, the extent of akinesis, assessed by gated scintiphotography, correlated linearly with ventricular EF. In a basic survey, Swan et al.\textsuperscript{28} assumed a spherical model of the LV, and showed that EF is reduced theoretically in direct relation to the proportion of inert myocardium. As indicated, the slope of the calculated regression in the current clinical study was significantly higher than that of the theoretical line. Thus, in patients with a %ACS < 25%, measured EF was higher, and in patients with a %ACS > 25%, measured EF was lower than the theoretical value. The reason for this discrepancy might be that 1) the infarcted myocardium is assumed to be totally noncontractile and nondistensible, and 2) the noninfarcted myocardium is assumed to have normal contractility and compliance. Our data indicate that one or both of these assumptions might not be satisfied during the early phase of acute MI.

Significant dyskinesis should produce a greater reduction in LVEF than would akinesis;\textsuperscript{26, 28} this is consonant with the findings of Miller et al.\textsuperscript{29} In this study, dyskinesis was demonstrated in 16 of 45 patients (36%). However, except in the patients with cardiogenic shock, the extent of paradoxical motion was relatively small. The effect of the contractile and compliance state of the noninfarcted myocardium is a major factor in acute MI. Thus, after experimental coronary occlusions in dogs, several investigators found enhanced mechanical function in nonischemic zones,\textsuperscript{29-31} while others found nonoccluded segment function to be either unchanged\textsuperscript{32, 33} or depressed.\textsuperscript{34, 35} In patients with a %ACS < 25%, noninfarcted myocardial function was generally supernormal in this study. Since the LVEDV was unchanged and the LVEDP increased slightly, the augmented contractile performance of remote myocardium could not be ascribed to an increase in ventricular preload and was probably secondary to endogenous sympathodrenergic stimulation after acute MI.\textsuperscript{36} Furthermore, since end-

![Systolic shortening (ΔS, mean ± SD) in patients with cardiogenic shock (closed circles) was severely reduced along all the hemiaxies and a paradoxic systolic expansion was actually noted in the anteropapical region (hemiaxies 3–6). The shaded areas represent 1 SD.](http://circ.ahajournals.org/doi/abs/10.1161/01.CIR.60.1.136?journalIssueId=10810115)
diastolic volume remained within the normal range in these patients, myocardial fibers probably maintained their normal helical arrangement, which would facilitate maximal systolic shortening. Conversely, end-diastolic volume in patients with a %ACS > 25% was increased compared with normal patients; this might indicate that the Frank-Starling mechanism is operative in both human and experimental MI. Feild et al. observed that the upper normal limit for end-diastolic volumes was reached when 17% of the LV was contracting abnormally. This observation could lend support to a theoretical spherical model developed by Klein and associates, who imply that when 20-25% of the left ventricle is inactivated, compensatory cardiac enlargement must ensue to maintain adequate ejection of blood. However, our data also indicate that the expected volumetric compensation failed in patients who developed severe pump failure. In these cases, the residual, presumably healthy myocardium was frequently found to be hypokinetic. This finding is in accord with the observation that compensatory hyperactivity is limited to uncomplicated or moderately severe infarction, while in patients with severe pump failure the uninvolved myocardium fails to hypercontract and may have a hypocontractile response despite catecholamine infusion. It has also been shown, both in experimental animals and in man, that reduced systolic shortening was seen near an acutely ischemic area perfused by patent vessels. Other investigators have shown histologic and metabolic abnormalities in nonischemic myocardium after acute coronary occlusions. The mechanism responsible for the depressed contractile function of the noninfarcted myocardium may be related to pre-existing and unrecognized myocardial damage and/or severe and diffuse coronary artery disease limiting flow to the unaffected myocardium, even though myocardial oxygen demands are increased due to the LV dilatation. The relative importance of each mechanism cannot be evaluated from the findings of the present investigation.

When the %ACS in the present study was similar in both anterior and inferior MIs, the location of ACS alone did not influence the indices of global LV function. In this context, a source of error may be that septal involvement was underestimated in the RAO view. Biplane ventriculography would allow more accurate evaluation of anteroseptal asynergy. However, it has been reported that all patients with asynergy in the LAO view also had asynergy in the RAO view. Although some underestimation of anteroseptal contraction abnormalities cannot be excluded in some patients, this does not alter the conclusions for the
whole group. Thus, the location of infarction does not appear as a unique determinant of LV performance, and the greater hemodynamic consequence usually noted in anterior than in inferior MI is the result of the more extensive area of ACS during anterior ventricular involvement. Another factor accounting for the greater frequency of pump failure in anterior than in inferior MI is that dyskinesis occurred with anterior wall necrosis, and not with inferior wall necrosis.

The finding of a high %ACS in patients with cardiogenic shock is consistent with previous reports. In cases ending fatally, a consistent finding was that more than 40–50% of the LV myocardium had been destroyed by recent or old infarcts. In this study, although most patients improved temporarily during intraaortic balloon pumping, only the two who had a %ACS < 50% were discharged from the hospital.

In conclusion, 1) the %ACS is the major determinant of global ventricular impairment that develops after acute MI; 2) the compensatory mechanisms that tend to restore cardiac performance operate either through an increase in contractile state of the residual myocardium when the extent of asynergy is small, or through the Frank-Starling mechanism when the extent of asynergy is large; 3) the dysfunction of the noninfarcted zone may be a contributory factor in the deterioration of cardiac performance in severe MI.

Acknowledgment

We are grateful to Dr. J. C. Gaux, Dr. Frijia, and Dr. P. Mengeot for their help in the cardiac catheterization laboratory. We are also indebted to Mrs. O. Lecanu and Mrs. Autef for expert secretarial assistance.

References

30. Pashkow F, Holland R, Brooks H: Dynamic responsiveness of
distant myocardium during transient anterior wall ischemia.
(abstr) Am J Cardiol 33: 161, 1974
31. Nakano J: Effect of changes in coronary blood flow on the
myocardial contractile force. Jpn Heart J 7: 78, 1966
32. Heikkilä J, Tabakin BS, Hugenholz PG: Quantification of
function in normal and infarcted regions of the left ventricle.
Cardiovasc Res 6: 516, 1972
33. Banka VS, Helfant RH: Temporal sequence of dynamic con-
tractile characteristics in ischemic and nonischemic myocardium
after acute coronary ligation. Am J Cardiol 34: 158, 1974
Effects of acute coronary occlusion on the motion and perfusion
of the normal and ischemic interventricular septum. An experi-
35. Wyatt HL, Forrester JS, DaLuz PL, Diamond GA,
Chagrasulis R, Swan HJC: Functional abnormalities in non-
occluded regions of myocardium after experimental coronary
36. Jewitt DE, Singh BN: The role of β-adrenergic blockade in
myocardial infarction. Progr Cardiovasc Dis 16: 421, 1974
37. Sallin EA: Fiber orientation and ejection fraction in the human
ventricle. Biophys J 9: 954, 1969
38. Lewis RP, Sandler H: Relationship between change in left ven-
tricular dimensions and the ejection fraction in man. Circula-
tion 44: 172, 1971
39. Hamilton GW, Murray JA, Kennedy WA: Quantitative angio-
cardiography in ischemic heart disease. The spectrum of abnor-
mal left ventricular function and the role of abnormally con-
tracting segments. Circulation 45: 1065, 1972
40. Corya BC, Rasmussen S, Knoebel SB, Feigenbaum H:
Echocardiography in acute myocardial infarction. Am J Cardi-
ol 36: 1, 1975
41. Corday E, Kaplan L, Meerbaum S, Brash J, Constantini C,
Lang T, Gold H, Rubins S, Osher J: Consequences of coronary
arterial occlusion on remote myocardium: effects of occlusion
and reperfusion. Am J Cardiol 36: 385, 1975
42. Vikher AM, Cherpachenko NM: Changes in metabolism of un-
damaged sections of myocardium following infarction. Circ
Res 35 (suppl III): III-182, 1974
43. Bodenheimer MM, Banka VS, Trout RG, Pasdar H, Helfant
RH: Correlations of pathologic Q waves on the standard elec-
trocardiogram and the epicardial electrogram of the human
44. Cohn PR, Gorlin R, Adams DF, Chakine RA, Vokonas PS,
Herman MV: Comparison of hipline and single plane left ven-
triculograms in patients with coronary artery disease. Am J
Cardiol 33: 1, 1974
45. Miller RR, Amsterdam EA, Bogren HG, Massumi RA, Zelis
R, Mason DT: Electrocardiographic and cineangiographic cor-
relations in assessment of the location, nature and extent of ab-
normal left ventricular segmental contraction in coronary
46. Ideker RE, Behar VS, Wagner GS, Starr JW, Starmer F, Lee
HL, Hackel DB: Evaluation of asynergy as an indicator of
47. Harnarayan C, Bennett MA, Pentecost BL, Brewer DB: Quan-
titative study of infarcted myocardium in cardiogenic shock. Br
Heart J 32: 728, 1970
CA: Myocardial changes associated with cardiogenic shock. N
Regional left ventricular function assessed by contrast angiography in acute myocardial infarction.

M Rigaud, P Rocha, J Boschat, J C Farcot, J Bardet and J P Bourdarias

Circulation. 1979;60:130-139
doi: 10.1161/01.CIR.60.1.130

The online version of this article, along with updated information and services, is located on the World Wide Web at:
http://circ.ahajournals.org/content/60/1/130