Reversible Segmental Left Ventricular Dysfunction After Coronary Angioplasty

Egerton K. van den Berg Jr., MD, Jeffrey J. Popma, MD,
Gregory J. Dehner, MD, Frank R. Snow, MD, Stephen A. Lewis, MD,
George W. Vetrovec, MD, and J.V. Nixon, MD

Patients with chronic segmental myocardial dysfunction may demonstrate improvement after coronary revascularization. To evaluate the early effects of percutaneous transluminal coronary angioplasty (PTCA) on resting left ventricular segmental function, we obtained serial two-dimensional echocardiograms 1.1±0.9 days before and 3.1±2 days after elective PTCA in 40 patients. Echocardiograms were reviewed in a blind fashion; left ventricular segmental wall motion was analyzed in four short-axis views, and a score was assigned to each region (0, normal; 1, hypokinetic; and 2, akinetic). Abnormal regional wall motion was present in 20 of the patients before PTCA. Summed segment scores in these 20 patients showed an improvement in regional wall motion from 4.5±2.5 to 1.6±2.1 (p<0.01) after successful PTCA. Similar results were obtained when the patients were divided into those with or without a previous myocardial infarction. Improvement occurred in the seven patients without a previous myocardial infarction; the summed segment score decreased from 4.2±3.4 to 0.86±1.6 (p<0.05) after PTCA. Ten of the 13 patients with a prior myocardial infarction demonstrated improvement in wall motion after PTCA; the summed segment scores decreased 54% (p<0.001). Of the 260 segments analyzed in the study, 180 were normal before and after PTCA. Forty-nine of the 69 hypokinetic segments were normal, and 10 of 12 akinetic segments were hypokinetic after successful coronary revascularization. There was no deterioration in wall motion after PTCA. These data show that two-dimensional echocardiography was able to detect improvement in abnormal resting left ventricular systolic function after successful PTCA, and they support the hypothesis that patients with ischemic heart disease experience recovery of segmental left ventricular dysfunction after coronary revascularization. (Circulation 1990;81:1210–1216)

Patients

Patients with myocardial perfusion that is chronically diminished yet adequate to maintain viable myocardium may demonstrate improvement in myocardial function after coronary revascularization.1 This phenomenon, first recognized in patients undergoing coronary artery bypass surgery, has stimulated interest in the concept of “the hibernating myocardium.”1–8 Left ventricular function has important prognostic implications in patients with ischemic heart disease.9,10 Therefore, the identification of hibernating myocardium, and appropriate referral for coronary revascularization, may improve the morbidity and mortality rates associated with coronary artery disease. Percutaneous transluminal coronary angioplasty (PTCA) is a form of coronary revascularization that may increase regional myocardial perfusion.11 Previous studies have demonstrated improvements in global systolic function during exercise and diastolic function at rest after PTCA,12–14 but changes in resting segmental function require further evaluation. Therefore, the purpose of this study was to determine whether resting segmental left ventricular function determined by two-dimensional echocardiography improves in patients after PTCA.

Methods

The study population consisted of 40 patients referred for elective PTCA of one or more major epicardial vessels. Although not consecutive, the patients were not preselected for this study, and only patients with a failed attempt at PTCA or patients with technically poor or uninterpretable echocardio-
TABLE 1. Clinical Characteristics of Patients Undergoing Percutaneous Transluminal Coronary Angioplasty

<table>
<thead>
<tr>
<th>Patient</th>
<th>Age (yr)</th>
<th>Prior infarct location</th>
<th>Interval from MI to PTCA (days)</th>
<th>Indication for PTCA</th>
<th>Vessel dilated</th>
<th>Prestenosis severity</th>
<th>Summed echocardiographic segment score</th>
<th>Dysfunctional segments</th>
</tr>
</thead>
<tbody>
<tr>
<td>Patients with prior infarctions</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>1</td>
<td>40</td>
<td>AWMI</td>
<td>240</td>
<td>Refractory angina</td>
<td>LAD</td>
<td>90%</td>
<td>8</td>
<td>4</td>
</tr>
<tr>
<td>2</td>
<td>62</td>
<td>IWMI</td>
<td>28</td>
<td>Unstable angina</td>
<td>RCA</td>
<td>99%</td>
<td>3</td>
<td>0</td>
</tr>
<tr>
<td>3</td>
<td>48</td>
<td>IWI</td>
<td>26</td>
<td>Refractory angina</td>
<td>Cx</td>
<td>99%</td>
<td>9</td>
<td>6</td>
</tr>
<tr>
<td>4</td>
<td>64</td>
<td>IWI</td>
<td>21</td>
<td>Unstable angina</td>
<td>RCA</td>
<td>90%</td>
<td>4</td>
<td>0</td>
</tr>
<tr>
<td>5</td>
<td>62</td>
<td>PLMI</td>
<td>24</td>
<td>Refractory angina</td>
<td>RCA</td>
<td>95%</td>
<td>3</td>
<td>0</td>
</tr>
<tr>
<td>6</td>
<td>41</td>
<td>AWMI</td>
<td>46</td>
<td>Unstable angina</td>
<td>LAD</td>
<td>99%</td>
<td>4</td>
<td>0</td>
</tr>
<tr>
<td>7</td>
<td>63</td>
<td>AWMI</td>
<td>15</td>
<td>Refractory angina</td>
<td>LAD</td>
<td>70%</td>
<td>5</td>
<td>2</td>
</tr>
<tr>
<td>8</td>
<td>66</td>
<td>AWMI</td>
<td>28</td>
<td>Refractory angina</td>
<td>SVG-LAD, RCA</td>
<td>99%, 95%</td>
<td>3</td>
<td>2</td>
</tr>
<tr>
<td>9</td>
<td>72</td>
<td>IWI</td>
<td>3</td>
<td>Refractory angina</td>
<td>RCA</td>
<td>95%</td>
<td>6</td>
<td>0</td>
</tr>
<tr>
<td>10</td>
<td>59</td>
<td>IWI</td>
<td>15</td>
<td>Unstable angina</td>
<td>RCA</td>
<td>90%</td>
<td>3</td>
<td>3</td>
</tr>
<tr>
<td>11</td>
<td>41</td>
<td>AWMI</td>
<td>9</td>
<td>Refractory angina</td>
<td>Cx</td>
<td>90%</td>
<td>6</td>
<td>6</td>
</tr>
<tr>
<td>12</td>
<td>70</td>
<td>NQWMI</td>
<td>10</td>
<td>Refractory angina</td>
<td>LAD, RCA</td>
<td>80%, 85%</td>
<td>3</td>
<td>3</td>
</tr>
<tr>
<td>13</td>
<td>63</td>
<td>NQWMI</td>
<td>840</td>
<td>Unstable angina</td>
<td>LAD</td>
<td>90%</td>
<td>3</td>
<td>0</td>
</tr>
<tr>
<td>Patients without prior infarction</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>14</td>
<td>55</td>
<td>Refractory angina</td>
<td>LAD</td>
<td>95%</td>
<td>6</td>
<td>0</td>
<td>6</td>
<td>PL, INF</td>
</tr>
<tr>
<td>15</td>
<td>55</td>
<td>Unstable angina</td>
<td>LAD</td>
<td>95%</td>
<td>7</td>
<td>0</td>
<td>7</td>
<td>PL, INF, AP</td>
</tr>
<tr>
<td>16</td>
<td>71</td>
<td>Refractory angina</td>
<td>RCA</td>
<td>90%</td>
<td>2</td>
<td>2</td>
<td>0</td>
<td>PL</td>
</tr>
<tr>
<td>17</td>
<td>59</td>
<td>Refractory angina</td>
<td>LAD</td>
<td>90%</td>
<td>2</td>
<td>0</td>
<td>2</td>
<td>PL</td>
</tr>
<tr>
<td>18</td>
<td>55</td>
<td>Unstable angina</td>
<td>RCA</td>
<td>99%</td>
<td>4</td>
<td>0</td>
<td>4</td>
<td>AL, S</td>
</tr>
<tr>
<td>19</td>
<td>46</td>
<td>Refractory angina</td>
<td>LAD</td>
<td>85%</td>
<td>10</td>
<td>4</td>
<td>6</td>
<td>AL, S, PL</td>
</tr>
<tr>
<td>20</td>
<td>60</td>
<td>Refractory angina</td>
<td>RCA</td>
<td>90%</td>
<td>1</td>
<td>0</td>
<td>1</td>
<td>PL</td>
</tr>
</tbody>
</table>

MI, myocardial infarction; PTCA, coronary angioplasty; AWMI, anterior wall myocardial infarction; IWI, inferior wall myocardial infarction; PLMI, posterolateral myocardial infarction; NQWMI, non-Q wave myocardial infarction; LAD, left anterior descending artery; RCA, right coronary artery; Cx, circumflex coronary artery; SVG-LAD, saphenous vein graft to the left anterior descending coronary artery; AL, anterolateral; INF, inferior; PL, posterolateral; AP, apical; S, septal.

grams were excluded from the study. All patients had demographic and clinical characteristics similar to the standard routine patients undergoing PTCA. Only two patients had echocardiograms that were obtained after a failed PTCA. Three patients were excluded because of technically poor or uninterpretable echocardiograms. Approval for the studies was obtained from the respective committees for the conduct of human research at the two institutions. Of the 40 patients in this study, 20 had normal wall motion before and after PTCA and were not considered further in this study. The clinical characteristics of the 20 patients with resting abnormal segmental left ventricular function are summarized in Table 1. There were 18 men and two women; their mean age was 57.6±10 years. Seven patients had no clinical history consistent with a previous myocardial infarction, had normal electrocardiograms, or had electrocardiograms without Q waves. Thirteen patients had a previous myocardial infarction according to accepted clinical and electrocardiographic criteria. In 11 of these patients, the myocardial infarction occurred 20.5±11.3 days (range, 3–46 days) before PTCA. In the remaining two patients, myocardial infarction occurred 240 and 840 days before echocardiography. One patient received thrombolytic therapy with intravenous streptokinase. Single-vessel coronary artery disease was present in 14 patients, and multivessel coronary artery disease was present in six patients. One patient had previously undergone coronary artery bypass grafting. PTCA was performed for relief of symptoms of refractory stable angina in 13 patients and for unstable angina in seven patients. The right coronary artery was dilated in 10 patients, the left anterior descending coronary artery was dilated in nine patients, the circumflex coronary artery was dilated in two patients, and a saphenous vein graft was dilated in one patients (Table 1). Two patients underwent multivessel PTCA; the left anterior descending and the right coronary arteries were dilated in one, and the right coronary artery and a saphenous vein graft were dilated in the other. All 20 patients were taking aspirin, 18 patients were taking a calcium antagonist, 17 patients were taking nitrates (oral or cutaneous), and five patients were taking a β-blocker at the time of selection for study. All but two patients continued on the same regimen throughout; diltiazem was added to the medical therapy of one patient, and nifedipine, aspirin, and persantine were added to the medical therapy of the other patient after PTCA.
Angiography and Angioplasty

The severity of all coronary lesions was determined by two experienced angiographers. All vessels subjected to PTCA had a greater than 70% luminal diameter narrowing. PTCA was performed with standard techniques and equipment. Generally, three to five inflations of 60–90 seconds in duration were performed until improvement in the lesion was noted on subsequent angiography. Successful PTCA was defined as the reduction of the lesion severity to a luminal diameter less than 50% compared with the adjacent normal vessel.

Echocardiography

Two-dimensional echocardiograms were obtained in patients 1.1±0.9 days before and 3.1±2.0 days after PTCA. Echocardiograms were recorded on videotape for subsequent analysis. All patients were studied in the recumbent or left lateral decubitus position.

The method of analysis of left ventricular segmental wall motion by summed segment score has been previously reported. The transducer was placed perpendicular to the chest wall, and the short axis of the left ventricle was scanned until its image was circular or almost circular, closely approximating the short axis. Serial scans were obtained by moving the transducer head inferolaterally on the chest wall to avoid extreme transducer angulation that would produce an oval rather than a circular image of the left ventricle and render interpretation of the wall asynchrony unreliable.

The four short-axis views of the left ventricle used to analyze wall motion were identified as positions IV, Va, Vb, and VI by Kisslo et al. These views permit the analysis of the anterolateral, posterolateral, apical, septal, and inferior regions of the left ventricular wall. Ventricular wall motion was graded in each wall region in each view with a modification of the terminology of Herman and Gorlin: 0, normal; 1, hypokinesia; 2, akinesia; and 3, dyskinesia. The graded scores of all regions in each echocardiographic view were totaled, and the scores obtained from all views were summed for each patient. This summed segment score was the echocardiographic estimate of the dysfunctional myocardium.

Echocardiographic recordings were analyzed by two investigators without prior knowledge of the identity and clinical status of the patient and, in the case of study after PTCA, the results of the initial study. Interobserver variability was minimal; correlations between numbers of regions and summed abnormal wall motion scores were r=0.97 and r=0.96, respectively. When discrepancies occurred, blind evaluation by a third observer was obtained. Persistent discrepancies were resolved by consensus. An echocardiographic study was considered adequate when all 13 segments could be analyzed by each observer. The 40 patients in this study represent 75% of the patients referred for echocardiography. Three patients were considered unsuitable because of limited acoustic windows. Figure 2 shows echocardiograms obtained before and after PTCA.

Statistical Analysis

Summed segment scores obtained before and after PTCA were compared with a paired t test and were considered significantly different if p was less than 0.05. All values are expressed as mean±SD.

Results

The summed segment scores in the 20 patients with resting abnormal segmental wall motion before PTCA are summarized in Figure 3. The mean summed segment score before PTCA was 4.5±2.5 and improved to 1.6±2.1 (p<0.001) after PTCA. Although the summed segment score did not improve in four patients (20%), there was no deterioration in segmental wall motion in any patient after PTCA.

The summed segment scores of the 13 patients with a history of previous myocardial infarction are shown in Figure 4. The mean summed segment score in these patients before PTCA was 4.6±2.0 and improved to 2.1±2.3 (p<0.001) after PTCA. Although there was no improvement in three patients, no patient in this group demonstrated deterioration in wall motion after PTCA.

Figure 5 displays the summed segment scores for the seven patients without a previous myocardial infarction. The mean summed segment score before PTCA was 4.2±3.4. After PTCA, there was a reduction in the summed segment score to 0.9±1.6 (p<0.02) indicating an improvement in segmental function. There was only one patient in this group who did not demonstrate improvement in wall motion after PTCA.
The fate of all 260 segments analyzed in the 20 patients of this study is shown in Figure 6. The 180 segments that demonstrated normal wall motion before PTCA were normal after PTCA. Of the 68 segments that were hypokinetic before PTCA, 49 segments improved after PTCA, and 19 segments did not change. Twelve segments were akinetic before PTCA. After PTCA, 10 of these segments improved, and two segments remained unchanged.

The improvement in summed segment scores of patients with refractory angina and unstable angina was similar (2.7±2.3 vs. 3.3±2.1, p=NS). In addition, patients with a myocardial infarction within the 28 days before PTCA had a summed segment score similar to those patients without previous infarction or whose infarction occurred more than 28 days before PTCA. The improvement in summed segment score was compared according to the vessel undergoing PTCA. The summed segment score tended to be higher in patients undergoing PTCA of the left anterior descending coronary artery than the right coronary artery but was not significant (3.6±2.3 vs. 2.0±2.0, p=0.11). The number of patients undergoing PTCA of the circumflex coronary artery was too small for subgroup analysis.

**Discussion**

The factors that influence left ventricular function in chronic ischemic heart disease and that improve myocardial dysfunction after coronary revascularization remain undefined. In experimental animal studies, small reductions in coronary blood flow and short periods of severe, transient ischemia may produce prolonged periods of myocardial dysfunction without evidence of infarction. Furthermore, Gould and Lipsomb have demonstrated impairment in resting coronary blood flow when the luminal diameter stenosis of the coronary vessel exceeded 80%. Thus, an alteration in myocardial function in the setting of diminished coronary perfusion may be protective, because reduction in myocardial oxygen demand may subsequently minimize the extent of ischemia or infarction.
Patients with chronic ischemic heart disease who demonstrate left ventricular dysfunction may do so because of persistently depressed coronary perfusion and reversible myocardial dysfunction with areas of necrosis and scarring. Patients with dysfunctional myocardium supplied by significantly stenosed coronary vessels may experience improvement in myocardial perfusion and augmentation of regional coronary blood flow after successful PTCA. Under these conditions, improvement in global or regional left ventricular function may occur. In the present study, patients with chronic myocardial ischemia demonstrated improvement in resting regional wall motion after PTCA, including patients with previous myocardial infarction.

The terms “stunned” and “hibernating” myocardium have been adopted in an attempt to characterize reversible changes in left ventricular function observed in some patients with prolonged episodes of acute ischemia and after coronary artery bypass grafting in patients with chronic ischemia, respectively. Acute ischemic heart syndromes (which may or may not be clinically silent) associated with intermittent but severe ischemia may result in “stunned” myocardium that demonstrates reversible dysfunction at rest. Patients with depressed myocardial perfusion and chronic ischemia with persistently dysfunctional but viable myocardium (“hibernating” myocardium) may demonstrate reversible dysfunction after coronary revascularization. The present study supports the hypothesis that patients with “stunned” or “hibernating” myocardium may demonstrate improvement in left ventricular function after coronary revascularization. Patients in this study with recent and remote acute myocardial infarction as well as patients with chronic myocardial ischemia showed improvement in resting regional wall motion after PTCA.

Previous studies of systolic and diastolic function at rest or during exercise have shown improvement after successful revascularization by PTCA. None of these studies, however, used two-dimensional echocardiography in their evaluation of regional wall motion. The present study identifies improvement in resting left ventricular systolic function after coronary revascularization by PTCA. In the seven patients without a history of myocardial infarction, left ventricular dysfunction at rest was presumably due to either a subclinical infarction with necrosis and scarring or chronically ischemic and dysfunctional myocardium. The resting left ventricular dysfunction in the 13 patients with a prior myocardial infarction may be attributed completely or partially to myocardial necrosis and scarring. However, as previously discussed, these patients may also have poorly perfused and chronically ischemic myocardium that is dysfunctional but viable.

Two-dimensional echocardiography is a sensitive, inexpensive, and readily accessible noninvasive technique that is capable of visualizing abnormal left ventricular wall motion in patients with acute and chronic ischemic heart syndromes. Previous reports in patients undergoing PTCA have used echocardiography to characterize the sequence of myocardial and clinical events during balloon inflation and transient coronary artery occlusion. Segmental wall motion abnormalities including hypokinesia, akinesia, and dyskinesia typically occur within 15–20 seconds of balloon inflation and precede transient electrocardiographic changes or angina pectoris. These wall motion abnormalities subsequently resolved within 20 seconds after balloon deflation. Topol et al used serial echocardiography to document the immediate and delayed improvement in regional left ventricular wall motion after coronary artery bypass grafting. Their results are consistent with the present study in which two-dimensional echocardiography detected improvement after PTCA.

Cardiac medications such as calcium antagonists and β-blockers have well-described effects on cardiovascular hemodynamics. Although medication schedules varied among the study patients, only two of 20 patients received different medications at the time of study before and after PTCA. One of these patients demonstrated improvement in wall motion, and the
other did not exhibit any change in wall motion after PTCA. It is unlikely that the improvement in regional wall motion detected in this single study can be ascribed simply to the potentially beneficial effects of cardiac medications.

Further studies are needed to prospectively identify patients with hibernating myocardium who might benefit from coronary revascularization. The present study identifies dysfunctional myocardium but does not provide information on the mechanism by which wall motion is improved. Serial evaluation of patients undergoing PTCA with positron emission tomography or other scintigraphic methods that assess myocardial metabolism may allow us to successfully predict which patients may benefit from revascularization and provide insight into the basic mechanisms that cause reversible myocardial dysfunction.30–32

Acknowledgments

We thank Arvella Peters and Cathy Guard for technical assistance, and Shelia G. Kelley and Jeanie Toombs for secretarial help.

References


**KEY WORDS** • ventricular function • myocardial ischemia • echocardiography, two-dimensional • angioplasty
Reversible segmental left ventricular dysfunction after coronary angioplasty.
E K van den Berg, Jr, J J Popma, G J Dehmer, F R Snow, S A Lewis, G W Vetrovec and J V Nixon

Circulation. 1990;81:1210-1216
doi: 10.1161/01.CIR.81.4.1210

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/81/4/1210

Permissions: Requests for permissions to reproduce figures, tables, or portions of articles originally published in Circulation can be obtained via RightsLink, a service of the Copyright Clearance Center, not the Editorial Office. Once the online version of the published article for which permission is being requested is located, click Request Permissions in the middle column of the Web page under Services. Further information about this process is available in the Permissions and Rights Question and Answer document.

Reprints: Information about reprints can be found online at:
http://www.lww.com/reprints

Subscriptions: Information about subscribing to Circulation is online at:
http://circ.ahajournals.org//subscriptions/