Can Coronary Angiography Predict the Site of a Subsequent Myocardial Infarction in Patients With Mild-to-Moderate Coronary Artery Disease?

William C. Little, MD, Martin Constantinescu, MD, Robert J. Applegate, MD, Michael A. Katcher, MD, Mark T. Burrows, PA, Frederic R. Kahl, MD, and William P. Santamore, PhD

To help determine if coronary angiography can predict the site of a future coronary occlusion that will produce a myocardial infarction, the coronary angiograms of 42 consecutive patients who had undergone coronary angiography both before and up to a month after suffering an acute myocardial infarction were evaluated. Twenty-nine patients had a newly occluded coronary artery. Twenty-five of these 29 patients had at least one artery with a greater than 50% stenosis on the initial angiogram. However, in 19 of 29 (66%) patients, the artery that subsequently occluded had less than a 50% stenosis on the first angiogram, and in 28 of 29 (97%), the stenosis was less than 70%. In every patient, at least some irregularity of the coronary wall was present on the first angiogram at the site of the subsequent coronary obstruction. In only 10 of the 29 (34%) did the infarction occur due to occlusion of the artery that previously contained the most severe stenosis. Furthermore, no correlation existed between the severity of the initial coronary stenosis and the time from the first catheterization until the infarction ($r^2 = 0.0005, p = \text{NS}$). These data suggest that assessment of the angiographic severity of coronary stenosis may be inadequate to accurately predict the time or location of a subsequent coronary occlusion that will produce a myocardial infarction. *(Circulation 1988;78:1157–1166)*

Coronary angiography is the clinical standard for the evaluation of patients with suspected coronary artery disease. The prognosis of patients is inversely related to the number of coronary vessels with angiographically apparent stenosis.1 Furthermore, the presence of a high-grade stenosis (>80% angiographic diameter narrowing) and extensive coronary artery disease correlates with a higher risk of subsequent coronary artery occlusion producing a myocardial infarction.2

Coronary artery bypass surgery and percutaneous transluminal coronary angioplasty are directed at areas of the coronary arteries that contain angiographic stenoses. While these interventions are effective in reducing angina, it is frequently hoped that they might also reduce the risk of a subsequent myocardial infarction. However, it has not been determined if coronary angiography can accurately predict the site of a subsequent coronary artery occlusion that will produce a myocardial infarction. Unless coronary angiography can accurately predict the site of a subsequent coronary artery occlusion, it is unlikely that therapy aimed only at sites of angiographic coronary obstruction will be effective in preventing future myocardial infarctions.

Accordingly, we evaluated the coronary angiograms of 42 patients with mild-to-moderate coronary artery disease who had undergone angiography before a myocardial infarction and again after subsequently suffering an acute myocardial infarction. The second coronary angiogram was used to determine the location of the coronary obstruction.

*From the Section of Cardiology, Department of Medicine, Bowman Gray School of Medicine, Wake Forest University, Winston-Salem, North Carolina.*

*Portions were presented at the 59th Annual Scientific Sessions of the American Heart Association, November 1987.*

*Supported in part by National Institutes of Health Grant HL-36051. W.C.L. is an Established Investigator of the American Heart Association.*

*Address for correspondence: William C. Little, MD, Section of Cardiology, Bowman Gray School of Medicine, Wake Forest University, 300 South Hawthorne Road, Winston-Salem, NC 27103.*

*Received April 8, 1988; revision accepted June 14, 1988.*
producing the infarction; the first coronary angiogram was used to determine the coronary anatomy preceding the myocardial infarction.

Patients and Methods

We reviewed the records of all patients who underwent coronary angiography at North Carolina Baptist Hospital between 1975 and 1985. Seventy-six patients had undergone two coronary angiograms; one before suffering an acute myocardial infarction, and the second within 1 month after the myocardial infarction. The diagnosis of myocardial infarction was based on electrocardiographic changes and rises in serum creatinine kinase activity and confirmed by the presence of a new wall motion abnormality on the left ventriculogram. Twenty-eight patients who had undergone coronary artery bypass surgery and one patient who had undergone coronary angioplasty were excluded. The coronary angiograms of five patients were unavailable. Finally, 13 patients who did not have a newly totally occluded coronary artery on the second catheterization were analyzed separately. Thus, the study group consisted of 29 patients, each of whom had a new abnormality of left ventricular wall motion corresponding to the distribution of a newly occluded coronary artery.

Selective coronary angiograms were performed in multiple projections. The coronary angiograms were independently reviewed by three experienced observers. Disagreements were resolved by consensus. The site of the obstruction present on the second angiogram causing the myocardial infarction (infarct-related artery) was identified. The initial coronary angiogram was then analyzed to determine the severity of the arterial disease that was present before the infarct. Lesions were analyzed with the angiographic view in which the stenosis was most severe. The most severe lesion in the infarct-related artery, as well as the most severe stenoses in each of the two other arteries, were determined. The 13 patients without a totally occluded artery after the infarction were analyzed separately because the site of occlusion producing the infarct could not be unequivocally determined. The most likely site of the infarct was determined (in most cases, a subtotal obstruction, markedly increased in severity from the first angiogram), and any lesions previously present in this artery and the other coronary arteries were determined as described above.

The coronary lesions were quantitated with a computerized analysis system. The cine coronary angiograms were projected and then cine-to-video converted (Model TV-3, Vanguard Instruments, Melville, New York). The video images were transferred to an IBM PC-XT personal computer by a frame grabber board (Matrox Electronics, Quebec, Canada). The investigator identified the center of the arterial segment. The computer then automatically detected the vessel edges with a spatial deriv-ative algorithm. Using the angiographic catheter as a scaling device, the computer calculated the dimensions every 0.5 mm along the length of the vessel. The present diameter stenosis (compared with the mean of angiographically normal segments proximal and distal to the stenosis) and minimal luminal diameter were calculated as the mean of three separate determinations. The reproducibility of these determinations was 3.7 ± 3.3% (mean ± SD). The quantitative coronary angiography system used in this study has been evaluated for accuracy by measuring dimensions from radiographic images of tubular models with known dimensions. The regression of calculated versus actual dimensions gave a slope of 1.01, a correlation coefficient of 0.996, and an SEE of 0.072 mm.

Left ventriculography was performed by the injection of 40–50 ml iodinated contrast media into the left ventricle over 4 seconds and filmed in the 30° right anterior oblique and 60° left anterior oblique projection. The end-diastolic and end-systolic left ventricular outlines were traced without knowledge of the coronary anatomy. Systolic wall motion was determined by superimposing the systolic and diastolic tracings. An abnormality of wall motion was defined as a lack of systolic motion of the anterior, inferior, septal, or lateral walls.

Data are summarized as the mean ± SD. Comparison of groups was performed by analysis of variance. If a significant difference among groups was identified, intragroup comparisons were performed by two-tailed paired t tests with correction for multiple comparisons with the Bonferroni inequality. Correlation was performed by linear least-squares regression. In determining whether the infarction occurred in the coronary artery that previously contained the highest grade stenoses, arteries that were totally occluded on the initial angiogram were excluded because they were not at risk for a new occlusion.

Results

The patient characteristics are summarized in Table 1. Seven of the patients had a history of a myocardial infarction. The first coronary angiogram was performed 706 ± 685 days (range, 4–2,298 days) before the myocardial infarction (Table 2). The indication for the first angiogram was chest pain consistent with angina in all patients. At the initial catheterization, four patients had luminal irregularities but no stenosis more than 50% in any coronary artery. Sixteen patients had a single coronary artery with a more than 50% stenosis (one-vessel disease), eight patients had two-vessel disease, and one patient had three-vessel disease. Thus, our patient population consisted predominantly of patients with mild-to-moderate coronary artery disease involving one or two vessels on the initial angiograms.

The second coronary angiogram was performed 18 ± 10 days after the infarction. Only three of the patients were taking aspirin at the time of the myocardial infarction. Representative coronary
angiograms from patients before and after an acute myocardial infarction are shown in Figures 1–3. In each of these examples, the artery that subsequently occluded did not contain a high-grade angiographic stenosis. Other arteries that did contain high-grade stenoses remained patent. The most severe stenosis existing in the infarct-related artery before the myocardial infarction was less than 50% luminal diameter narrowing in 19 (66%) of 29 patients, was less than 70% in 28 (97%) of 29 patients, and had a minimal diameter of more than 1 mm in 26 (90%) of 29 patients. Thus, angiographically severe coronary artery stenosis was infrequently present in the infarct-related artery on the initial angiogram. Some irregularity in the wall of the infarct-related artery at the site of the subsequent occlusion could be retrospectively identified in each patient. There was no correlation ($r^2=0.0005$, $p=NS$) between the severity of the stenosis in the infarct-related artery and the time from the initial catheterization to the occurrence of the myocardial infarction (Figure 4) or the severity of the highest-grade stenosis existing anywhere in the coronary arteries ($r^2=0.03$, $p=NS$).

The coronary occlusion occurred in the previously patent coronary vessel with the most severe coronary stenosis in only 10 (34%) of the 29 patients. Because previously occluded arteries are not at risk for subsequent occlusion, arteries that were totally occluded at the initial angiogram were excluded from this analysis. The interval from the initial catheterization until the development of the myocardial infarction was less ($p<0.05$) in the patients without significant coronary artery disease ($131 \pm 30$ days) compared with those with severe coronary artery disease ($291 \pm 49$ days) ($r^2=0.005$, $p=NS$).
FIGURE 1. Initial coronary angiogram (top) of the right coronary artery in patient 13 demonstrates only a luminal irregularity at the future infarct site (arrow) without an obstructive lesion. Coronary angiography after an inferior infarction 13 days later (bottom) demonstrates total occlusion of the mid-right coronary artery (arrow).
FIGURE 2. Initial coronary angiograms of the right (top left) and left coronary arteries (top right) of patient 11 demonstrate a high-grade lesion of the left anterior descending artery (arrow), while the proximal right coronary artery has less than a 50% stenosis. Five years later, the patient underwent repeat angiography of the right (bottom left) and left coronary arteries (bottom right) after an inferior infarction. The infarct was because of an occlusion of the right coronary artery (arrow), while the high-grade lesion of the left anterior descending artery remained patent (arrow).
FIGURE 3. Initial angiogram of the left coronary artery (top) of patient 20 shows occlusion of the left anterior descending coronary artery after the origin of the first septal artery and only irregularities without an obstructive stenosis of the large marginal branch (arrow). Two months later, the patient suffered a lateral infarction. Repeat angiography (bottom) demonstrated a new occlusion of the marginal branch (arrow).
days) than in patients with one-vessel (1,029 ± 719 days) or two-vessel disease (594 ± 638 days).

Ten patients suffered anterior myocardial infarctions because of occlusion of the left anterior descending coronary artery in nine and the left main coronary artery in one. In nine patients, the left circumflex coronary artery occluded producing a lateral infarction, while the right coronary artery was the site of occlusion in 10 patients. No significant differences existed in the time to infarction or severity of coronary artery disease when the patients were grouped by the site of the infarct.

The 13 patients without a new totally occluded artery after the infarction were analyzed separately because the site in the coronary arteries responsible for the infarction could not be unequivocally iden-
tified. When these patients were analyzed with the presumed site of coronary occlusion that produced the infarction, the results were similar to those of the 29 study patients. The most severe stenosis in the infarct-related artery before the infarct was less than 50% in eight (62%) of the 13 and less than a 70% stenosis in all 13.

Discussion

Acute myocardial infarction is usually produced by the sudden total occlusion of a coronary artery by thrombus, usually occurring at the site of an atherosclerotic lesion. Our study indicates that the lesion that will be the site of the thrombotic occlusion frequently is not severe when evaluated by coronary angiography weeks to years before the

<p>| Table 2. Initial Angiogram (Preinfarct) Characteristics |</p>
<table>
<thead>
<tr>
<th>Patient</th>
<th>Worst lesion in infarct vessel</th>
<th>Stenosis (%)</th>
<th>Minimum diameter</th>
<th>Worst stenosis in any vessel cath 1</th>
<th>Did MI occur in previously patent artery with worst stenosis?</th>
<th>Number vessels &gt;50% in cath 1</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td></td>
<td>77</td>
<td>0.76</td>
<td>77</td>
<td>Y</td>
<td>2</td>
</tr>
<tr>
<td>2</td>
<td></td>
<td>69</td>
<td>0.88</td>
<td>69</td>
<td>Y</td>
<td>1</td>
</tr>
<tr>
<td>3</td>
<td></td>
<td>66</td>
<td>1.00</td>
<td>66</td>
<td>Y</td>
<td>1</td>
</tr>
<tr>
<td>4</td>
<td></td>
<td>59</td>
<td>1.58</td>
<td>69</td>
<td>N</td>
<td>2</td>
</tr>
<tr>
<td>5</td>
<td></td>
<td>58</td>
<td>1.24</td>
<td>74</td>
<td>N</td>
<td>2</td>
</tr>
<tr>
<td>6</td>
<td></td>
<td>57</td>
<td>1.28</td>
<td>57</td>
<td>Y</td>
<td>1</td>
</tr>
<tr>
<td>7</td>
<td></td>
<td>57</td>
<td>1.67</td>
<td>68</td>
<td>N</td>
<td>3</td>
</tr>
<tr>
<td>8</td>
<td></td>
<td>57</td>
<td>1.94</td>
<td>57</td>
<td>Y</td>
<td>1</td>
</tr>
<tr>
<td>9</td>
<td></td>
<td>56</td>
<td>1.76</td>
<td>75</td>
<td>N</td>
<td>2</td>
</tr>
<tr>
<td>10</td>
<td></td>
<td>50</td>
<td>1.61</td>
<td>65</td>
<td>N</td>
<td>1</td>
</tr>
<tr>
<td>11</td>
<td></td>
<td>49</td>
<td>1.50</td>
<td>88</td>
<td>N</td>
<td>1</td>
</tr>
<tr>
<td>12</td>
<td></td>
<td>48</td>
<td>0.87</td>
<td>100 (56)</td>
<td>N</td>
<td>2</td>
</tr>
<tr>
<td>13</td>
<td></td>
<td>47</td>
<td>2.06</td>
<td>79</td>
<td>N</td>
<td>2</td>
</tr>
<tr>
<td>14</td>
<td></td>
<td>45</td>
<td>1.78</td>
<td>62</td>
<td>N</td>
<td>1</td>
</tr>
<tr>
<td>15</td>
<td></td>
<td>44</td>
<td>1.79</td>
<td>100 (45)</td>
<td>N</td>
<td>1</td>
</tr>
<tr>
<td>16</td>
<td></td>
<td>43</td>
<td>2.64</td>
<td>44</td>
<td>N</td>
<td>0</td>
</tr>
<tr>
<td>17</td>
<td></td>
<td>43</td>
<td>1.88</td>
<td>43</td>
<td>Y</td>
<td>0</td>
</tr>
<tr>
<td>18</td>
<td></td>
<td>45</td>
<td>1.66</td>
<td>100 (55)</td>
<td>N</td>
<td>2</td>
</tr>
<tr>
<td>19</td>
<td></td>
<td>41</td>
<td>1.60</td>
<td>100 (51)</td>
<td>N</td>
<td>2</td>
</tr>
<tr>
<td>20</td>
<td></td>
<td>37</td>
<td>2.42</td>
<td>100 (37)</td>
<td>Y</td>
<td>1</td>
</tr>
<tr>
<td>21</td>
<td></td>
<td>34</td>
<td>1.92</td>
<td>63</td>
<td>Y</td>
<td>1</td>
</tr>
<tr>
<td>22</td>
<td></td>
<td>33</td>
<td>2.27</td>
<td>55</td>
<td>N</td>
<td>1</td>
</tr>
<tr>
<td>23</td>
<td></td>
<td>32</td>
<td>1.89</td>
<td>64</td>
<td>N</td>
<td>2</td>
</tr>
<tr>
<td>24</td>
<td></td>
<td>30</td>
<td>1.44</td>
<td>100 (36)</td>
<td>N</td>
<td>1</td>
</tr>
<tr>
<td>25</td>
<td></td>
<td>29</td>
<td>2.32</td>
<td>100 (47)</td>
<td>N</td>
<td>1</td>
</tr>
<tr>
<td>26</td>
<td></td>
<td>24</td>
<td>2.72</td>
<td>25</td>
<td>N</td>
<td>0</td>
</tr>
<tr>
<td>27</td>
<td></td>
<td>24</td>
<td>2.02</td>
<td>100 (24)</td>
<td>Y</td>
<td>1</td>
</tr>
<tr>
<td>28</td>
<td></td>
<td>17</td>
<td>2.75</td>
<td>55</td>
<td>N</td>
<td>1</td>
</tr>
<tr>
<td>29</td>
<td></td>
<td>14</td>
<td>3.64</td>
<td>14</td>
<td>Y</td>
<td>0</td>
</tr>
<tr>
<td>Mean</td>
<td></td>
<td>44</td>
<td>1.82</td>
<td>71</td>
<td>1.21</td>
<td></td>
</tr>
<tr>
<td>SD</td>
<td></td>
<td>15</td>
<td>0.62</td>
<td>23</td>
<td>0.71</td>
<td></td>
</tr>
</tbody>
</table>

Worst stenosis in any vessel cath 1, most severe stenosis at any site at initial coronary angiography; number in parentheses indicates second most severe stenosis; if a 100% stenosis was present; Did MI occur in previously patent artery with worst stenosis, myocardial infarction occurred due to occlusion of previously patent artery with most severe stenosis at initial angiography (i.e., totally occluded arteries not considered); number vessels >50% at cath 1, number of arteries with more than 50% stenosis at first angiogram.
FIGURE 4. Plot showing there is no relation between the severity of the stenosis at the future infarct site and the time from initial angiography until the development of the acute myocardial infarction. Severe stenoses were infrequent in the infarct-related artery on the initial angiogram.

infarct in patients with mild-to-moderate coronary artery disease; thus, coronary angiography was not able to accurately predict the time or location of the subsequent myocardial infarction. In the majority (66%) of patients in this study, the myocardial infarction occurred because of the occlusion of a coronary artery that did not contain an obstructive (more than 50% diameter narrowing) stenosis on a previously performed coronary angiogram. A high-grade stenosis (more than 70% diameter narrowing) was initially present in the infarct-related artery in only one patient. Furthermore, the myocardial infarction did not occur because of occlusion of the previously patent artery with the most severe stenosis in two thirds of the patients. There was a large variation (4–2,298 days) in the time from the first coronary angiogram until the development of the myocardial infarction. It is possible that coronary angiograms taken a short period before a myocardial infarction would be more predictive of the site of the coronary occlusion. However, of the four patients in which the myocardial infarction occurred within 3 weeks of the coronary angiogram, only one was due to occlusion of the artery with the most severe stenosis on the initial angiogram. In addition, there was no correlation (Figure 4) between the severity of the lesion in the future infarct vessel on the initial angiogram and the time to develop the myocardial infarction. Thus, the angiographic assessment of the severity of coronary stenoses did not predict the site or the timing of the subsequent coronary occlusion in our patients.

All but four of the patients in our study had a more than 50% coronary stenosis in at least one of the coronary vessels on the initial coronary angiogram, but the artery that would subsequently occlude had an angiographically significant (≥50%) stenosis in only 34%, and only one patient initially had a more than 70% stenosis in the future infarct vessel. Thus, in our patients, the presence of angiographically apparent discrete stenoses may have indicated the presence of coronary atherosclerosis and the potential for the development of a myocardial infarction, but the coronary stenosis did not necessarily indicate the location at which the thrombotic occlusion will occur. Because the prognosis of patients with stable angina is related to the number of stenosed coronary arteries,1 it is possible that the more obstructive lesions that are present, the larger the number of potentially thrombogenic lesions; although all the potentially thrombogenic lesions may not produce angiographically apparent obstruction or produce angina.

Our observations in patients suffering an acute myocardial infarction should be compared with two previous studies3,6 of patients undergoing serial coronary angiograms. In these studies, the patients were not selected to include only individuals recently suffering an acute myocardial infarction as in our study. In a review of patients who had undergone two coronary angiograms for any reason, Moise et al2 found 116 newly occluded coronary vessels at the second angiogram. The presence of a high-grade (>80%) coronary stenosis, the extent of the coronary disease, smoking, and male sex were the factors most predictive of the development of a new coronary artery occlusion. Thus, Moise et al2 concluded that the coronary angiogram, when combined with two clinical factors, could predict the subsequent occurrence of a myocardial infarction. This conclusion differs from our results; however, analysis of the data in Table 4 of the study by Moise et al2 indicates that 72% of the occlusions in his study occurred in coronary segments that previously contained less than a 75% stenosis. These data, though not specifically commented on in this earlier study, are consistent with our conclusion that many occlusions occur in arteries that previously did not contain a high-grade angiographic stenosis. Similarly, Singh7 studied 52 patients who underwent serial coronary angiograms for any reason. Twelve of the 25 new total occlusions present in the second angiogram occurred in segments that were free of angiographic stenosis 2–108 months earlier. Furthermore, Brosius and Roberts8 found that the location of a fatal first myocardial infarction does not necessarily indicate which of the major coronary systems is most severely narrowed by atherosclerosis.

After coronary flow is restored by thrombolytic therapy in patients with myocardial infarction, there is frequently a high-grade residual stenosis.9 This may indicate that the atheroma at the site of coronary occlusion was obstructive and should have been easily identifiable by coronary angiography performed before the myocardial infarction. Alternatively, there may be residual thrombi overlying the atheroma, making the lesion appear much more severe than it was before the infarct. The study of Brown et al10 supports this latter possibility. They used angiographic magnification and computerized measurements to separate the coronary atheroma from the overlying thrombus in 32 patients who
received intracoronary streptokinase. Consistent with our observations, this technique suggested that the underlying atherosclerotic lesion was not severe in most patients with infarction and on average produced only a 56% stenosis.

Other studies provide indirect evidence consistent with our finding that a coronary atheroma need not be recognizable as a significant stenosis on coronary angiography to subsequently be responsible for an infarct. First, many patients present with myocardial infarctions without having previously experienced exertional angina. The coronary arteries of some of these patients without prodromal symptoms may not have previously contained obstructive lesions. Second, angiographically normal coronary arteries are occasionally present in patients after a myocardial infarction. Presumably, the occluding thrombus has spontaneously lysed before angiography, suggesting that angiographically normal arteries may occasionally thrombose, producing a myocardial infarction. Third, the pathophysiology of unstable angina may be similar to that of acute myocardial infarction. In patients with unstable angina, a thrombus overlying an atheroma abruptly increases the severity of the coronary obstruction without totally obstructing the artery. In agreement with our results concerning myocardial infarction, others have observed that the artery responsible for unstable angina frequently did not contain a high-grade stenosis before the onset of unstable angina. Thus, an artery does not need to contain a high-grade angiographic stenosis to subsequently be the site of thrombus formation that results in unstable angina. Finally, epicardial echocardiography has demonstrated that diffuse coronary artery disease is often present when coronary angiography demonstrates only discrete stenosis. In addition, an abnormal coronary artery response to acetylcholine has been recently reported in patients with minor luminal irregularities on coronary angiography. These latter findings indicate that coronary angiography may underestimate the extent of coronary atherosclerosis and that an abnormality in endothelial function that may help promote thrombus production can exist in coronary arteries that do not contain an angiographically significant stenosis.

The patient group in our study was highly selected. Only patients who were treated medically were included. Because patients with left main and most patients with three-vessel coronary artery disease underwent coronary artery bypass surgery, only one patient with three-vessel disease was included in the study group. Thus, our study group consisted of patients with mild-to-moderate coronary artery disease. The incidence of myocardial infarction in patients with one- and two-vessel coronary artery disease is less than 3% a year. Our results may not apply to patients with more extensive or severe coronary artery disease. It must be recognized that any myocardial infarction in a patient with three-vessel disease will occur in the distribution of an artery with an obstructive stenosis. Percutaneous transluminal coronary angioplasty was not a treatment option during much of the period of this study; however, many of the patients included in the study might presently be candidates for this procedure. The patients in this study must have survived a myocardial infarction and have undergone a second coronary angiogram, thus excluding patients with fatal myocardial infarctions, patients who did not return to our institution for their subsequent care, or patients were not referred for repeat catheterization. The possibility that the selection process may have influenced our results must be considered, and definitive assessment of the ability of coronary angiography to predict the site of a subsequent myocardial infarction would require a large prospective study.

Coronary angiography was performed 18 ± 10 days after myocardial infarction. The prevalence of complete coronary occlusion declines after the onset of an acute myocardial infarction, probably because of spontaneous lysis of the occluding thrombus. This may have been the reason that 13 of the potential patients identified for our study did not have a new total coronary obstruction on the second angiogram. These patients were excluded because the site of the obstruction causing the myocardial infarction could not be unequivocally defined. When these patients were analyzed based on the presumed site of coronary occlusion, the results were no different than the study group. Thus, exclusion of this group of patients does not appear to have biased our results.

Coronary angiography is the clinical standard for the evaluation of patients with coronary artery disease but is subject to observation bias and intraobserver and interobserver variations. We attempted to minimize these biases with a computer-based objective means of quantifying the coronary analysis. It is theoretically possible that coronary angiography may not always precisely define the site of occlusion since thrombus may propagate proximally or embolize distally. To avoid this potential problem, we analyzed the angiograms in terms of the most severe stenosis existing anywhere in the infarct vessel on the initial angiogram. Coronary angiography only images a two-dimensional projection of the arterial lumen and does not provide information on atheroma development in the arterial wall or alterations in endothelial function. In all of the patients in this study, careful retrospective inspection of the infarct-related segment on the initial angiogram disclosed at least some irregularity in the wall, probably indicating the presence of a nonobstructive atheroma.

Because it was difficult to predict the site of the subsequent occlusion in our patients from the initial coronary angiogram, coronary bypass surgery or angioplasty appropriately directed only at the angiographically significant lesions initially present in
almost all our patients would not have been effective in preventing the majority of myocardial infarctions. This does not indicate that arteries that do not have obstructive lesions should be bypassed or dilated. Instead, effective therapy to prevent myocardial infarction may need to be directed at the entire coronary tree, not just at obstructive lesions. Such therapy to prevent myocardial infarctions might rationally include avoiding smoking, reducing serum cholesterol, administering agents that alter platelet function such as aspirin or, possibly, fish oil, and pharmacological agents to prevent spasm of the coronary arteries.

In conclusion, an acute myocardial infarction occurred in the majority of our patients because of occlusion of a coronary artery that previously had less than a 50% angiographic coronary stenosis. Furthermore, in most of our patients with mild-to-moderate coronary artery disease, the infarction did not occur because of the occlusion of the artery that had previously been found to have the highest-grade stenosis. This suggests that the presence of obstructive coronary artery lesions indicates that a patient is at risk for developing a myocardial infarction but, in the majority of our patients, did not predict the timing or the location of the coronary occlusion that would subsequently produce a myocardial infarction. Thus, therapy such as percutaneous transluminal coronary angioplasty aimed solely at sites of coronary obstruction may not, by themselves, be able to prevent many subsequent myocardial infarctions.

Acknowledgments

We are grateful for Joyce Zafuto’s excellent secretarial assistance and for the critique of the manuscript and helpful suggestions by Edwin W. Rogers and Juaquin G. Arciniegas.

References


KEY WORDS: coronary angiography • myocardial infarction • coronary stenosis
Can coronary angiography predict the site of a subsequent myocardial infarction in patients with mild-to-moderate coronary artery disease?
W C Little, M Constantinescu, R J Applegate, M A Kutcher, M T Burrows, F R Kahl and W P Santamore

_Circulation_. 1988;78:1157-1166
doi: 10.1161/01.CIR.78.5.1157

_Circulation_ is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
Copyright © 1988 American Heart Association, Inc. All rights reserved.
Print ISSN: 0009-7322. Online ISSN: 1524-4539

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/78/5/1157

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/