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Angiographic Progression of Coronary Artery Disease and the Development of Myocardial Infarction

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There are few data on angiographic coronary artery anatomy in patients whose coronary artery disease progresses to myocardial infarction. In this retrospective analysis, progression of coronary artery disease between two cardiac catheterization procedures is described in 38 patients: 23 patients (Group I) who had a myocardial infarction between the two studies and 15 patients (Group II) who presented with one or more new total occlusions at the second study without sustaining an intervening infarction.

In Group I the median percent stenosis on the initial angiogram of the artery related to the infarct at restudy was significantly less than the median percent stenosis of lesions that subsequently were the site of a new total occlusion in Group II (48 versus 73.5%, $p < 0.05$). In the infarct-related artery in Group I, only 5 (22%) of 23 lesions were initially >70%, whereas in Group II, 11 (61%) of 18 lesions that progressed to total occlusion were initially >70% ($p < 0.01$). In Group I, patients who developed a Q wave infarction had less severe narrowing at initial angiography in the subsequent infarct-related artery (34%) than did patients who developed a non-Q wave infarction (80%) ($p < 0.05$). Univariate and multivariate analysis of angiographic and clinical characteristics present at initial angiography in Group I revealed proximal lesion location as the only significant predictor of evolution of lesions ≥50% to infarction.

This retrospective study suggests that myocardial infarction frequently develops from previously nonsevere lesions. In addition, it is often difficult to predict the location of a subsequent infarct from analysis of the first coronary angiogram. Non-Q wave infarction is usually preceded by a more severe pre-existing stenosis than is a Q wave infarction, perhaps indicating some degree of prior myocardial protection. A prospective evaluation will be necessary to confirm these findings.

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Myocardial infarction is usually caused by thrombus formation on a disrupted atherosclerotic plaque (1,2). Angiographically, this is manifested by total or subtotal occlusion of the infarct-related artery. Although there is an abundance of information on coronary anatomy immediately after myocardial infarction (3,4), there are few data on the coronary anatomy of patients who subsequently develop infarction. In other acute coronary syndromes such as unstable angina, progression of coronary artery disease often occurs in previously insignificant lesions (5,6). Therefore, the purpose of this investigation was to determine whether significant atherosclerotic lesions seen at angiography before myocardial infarction are the site of subsequent myocardial infarction or whether myocardial infarction evolves from previously insignificant lesions or normal-appearing arteries.

Methods

Selection of patients. At Mount Sinai Medical Center and Westchester Medical Center we retrospectively analyzed all identifiable patients who underwent coronary angiography both before and after a myocardial infarction (Group I). Patients underwent angiography before infarction to evaluate symptoms of angina pectoris or an abnormal exercise test, or both. These patients were restudied after infarction

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because of congestive heart failure or recurrent angina or as "standard practice" in a young patient. The decision for angiography was made by the private physician caring for the patient. Patients who underwent angioplasty or coronary bypass surgery between the two angiograms were excluded from analysis. In addition, patients without significant coronary disease (≥50% diameter reduction of a major coronary artery) at restudy or inadequate studies for comparison of coronary anatomy were excluded.

A second group of patients (Group II) was also analyzed, in whom one or more new total occlusions were present on a second angiogram, but no myocardial infarction was sustained. The diagnosis of infarction was excluded by the lack of a compatible history and by the absence of a new wall motion abnormality on ventriculography. These patients were restudied because of worsening angina pectoris or a worsening exercise tolerance test without a change in symptoms, or both.

Data Analyses

Clinical history. For each patient, the hospital records and catheterization reports were reviewed for the presence of the particular coronary syndrome and the assessment of coronary risk factors at the time of each catheterization. Unstable angina was defined as previously reported (7). Myocardial infarction was classified as Q wave or non-Q wave. The following criteria were utilized for a diagnosis of infarction:

Q wave infarction: A typical history of chest pain at rest with new significant Q waves in more than one electrocardiographic (ECG) lead or a new segmental left ventricular wall motion abnormality (severe hypokinesia or akinesia) in patients with a nondiagnostic ECG. Patients with "silent" Q wave infarction were not excluded if the preceding ECG or left ventricular wall motion abnormalities were present.

Non-Q wave infarction: A typical history of rest pain with new and persistent ST and T wave changes on ECG, lasting >48 h without the evolution of new Q waves and without a new severe segmental wall motion abnormality on left ventriculography.

Enzyme criteria for myocardial infarction included a total serum creatine kinase greater than twice the upper limits of normal. The diagnosis of infarction was made without knowledge of the change in percent lesion stenosis between angiograms.

Coronary angiography. Each pair of cineangiograms in a given patient was assessed on two Vanguard XR-35 projectors aligned side by side. The paired cine images were magnified approximately fourfold and projected in a darkened room on a clean white wall. For comparative analysis, the coronary tree was divided into 16 segments. For a dominant right coronary artery, five segments were analyzed (proximal, mid and distal right coronary artery, posterior descending coronary artery and the atrioventricular (AV) continuation artery). The left anterior descending coronary artery was also divided into five segments (proximal, mid and distal left anterior descending coronary artery and the first diagonal and second diagonal branches). The left circumflex coronary artery was divided into five segments (proximal and distal left circumflex coronary artery and the first large obtuse marginal, the second large obtuse marginal and the high lateral branch). The left main coronary artery was also analyzed. Lesion location was further classified as proximal or distal. Proximal included the proximal and mid segments of the three coronary arteries and the left main coronary artery; a distal location included the remaining segments.

Each coronary segment in the two angiograms was visually assessed by a consensus of angiographers for lesions with >30% diameter narrowing. If such a narrowing appeared on either study, it was traced on transparent paper by one individual from the magnified cine frame and compared with a "normal" segment of the same artery. The same segment was traced on the companion study, and the amount of stenosis was similarly quantified. In each film in each patient, all segments were analyzed in similar projections, anatomic locations and times of the cardiac cycle. Orthogonal views, when available, were traced, but the projection with the more severe stenosis was chosen for analysis of percent diameter stenosis. Diameter stenosis was measured from the transparent paper with a digimatic caliper (Mitutoyo, series 500). Intraobserver variability for this technique of drawing and measuring percent stenosis is ±5% in our laboratory. All drawings and measurements were made by the same individual. A significant lesion was defined as a diameter narrowing ≥50% in one or more views. If a segment had no visible narrowing, it was arbitrarily assigned as a 0% stenosis. All coronary angiographic analyses were performed independently and without knowledge of the clinical history (presence or absence of myocardial infarction) or of the analysis of left ventricular function.

In four patients with myocardial infarction, one of the two angiograms was unavailable for review. In three of these patients, the original angiogram had been reviewed by one of us at the time of catheterization. The other patient had normal coronary arteries at the time of the first angiogram. The catheterization reports contained, in detail, the coronary anatomy and were utilized for analysis. The recorded percent stenosis of only the subsequent infarct-related segment, however, was utilized for comparison with the second angiographic study.

Segmental left ventricular wall motion was qualitatively analyzed from the right anterior oblique ventriculogram. Five segments were visually assessed as normal or as showing mild hypokinesia, severe hypokinesia or akinesia/dyskinesia. Collateral vessels to the infarct-related artery were qualitatively assessed on a scale of 0 to 3; a grade of 0

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Table 1. Initial Catheterization Data in 38 Patients*

	Group I (n = 23)	Group II (n = 15)
Single vessel disease	6 (26%)	5 (33%)
Multivessel disease	15 (65%)	10 (67%)
No. of lesions ≥50%/patient	2.2	2.4
No. of totally occluded lesions	11	10

*p = NS for all comparisons.

indicated no collateral filling; a grade of 3 indicated complete reconstitution of the distal vessel.

Progression of coronary disease. Progression of coronary disease between angiograms was defined in a fashion similar to that of previous reports (6): 1) a ≥20% increase in diameter stenosis between angiograms in a lesion that on restudy was ≥50% narrowed, and 2) progression of any lesion to a new total occlusion on restudy.

Coronary morphology. Coronary morphology of lesions ≥50% and <100% was classified as previously reported (7). Lesions were divided into four categories according to the symmetry and irregularity of their borders, with special attention to asymmetric lesions with a narrow neck or irregular borders, or both (type II eccentric lesion).

Study patients. Thirty-eight patients fulfilled the selection criteria for enrollment in the study. Ten other patients were excluded because of either an unclear history or inadequate angiograms for comparison of coronary anatomy.

Group I: patients with myocardial infarction. These 23 patients had angiograms performed both before and after myocardial infarction. The myocardial infarction was a Q wave infarction in 15 (65%) and a non-Q wave infarction in 8 (35%). There were 17 men and 6 women with a mean (±SD) age of 57.8 ± 11.0 years. The time interval between the initial angiogram and infarction varied from 1 month to 7 years (median 18 months). Fifteen patients (65%) were studied within 2 years before infarction and nine (39%) were studied within 1 year of infarction. Twelve patients had unstable angina at the time of initial angiography. With two exceptions, all postinfarction angiograms were performed within 3 months of infarction. Eighty-three percent of patients underwent restudy within 1 month after myocardial infarction. No one was studied within 24 h of infarction.

Group II: patients without myocardial infarction. These 15 patients developed one or more new total coronary occlusions on restudy without evolving a myocardial infarction. There were 12 men and 3 women with a mean age of 60.2 ± 12.2 years. In this group, the time interval between angiograms varied from 3 months to 9 years (median 33 months). Eight of the 15 patients had a diagnosis of unstable angina at the time of initial angiography; 13 had this diagnosis at the time of restudy.

Statistical analysis. The Wilcoxon nonparametric test was used for comparison of timing data and degree of lesion stenosis between groups. For other continuous variables, student's *t* test was utilized. Fisher's exact test was used for comparison of discrete variables. Stepwise multivariate analysis was applied using the SAS statistical package. Significance was characterized at $p < 0.05$.

Results

There were no significant differences between the two groups in terms of age, male/female ratio and time interval between angiograms.

Initial coronary angiographic findings (Table 1). There were no significant differences between the two groups in terms of the number of patients with single or multivessel disease, number of lesions with ≥50% stenosis or number of totally occluded arteries. Two patients in Group I had no significant coronary artery disease.

Angiographic findings at restudy and progression of coronary artery stenosis (Table 2). In Group I at restudy, all patients had significant coronary artery disease. The infarct-related artery was easily identifiable in all. At restudy, 22 of the 23 patients demonstrated progression of coronary artery disease in the infarct-related artery. In the one patient without progression, percent stenosis increased by <20%, but distal coronary flow on restudy was delayed. At restudy, 14 (61%) of the 23 infarct-related arteries were totally occluded; the rest (39%) were >70% and <100% stenotic. In noninfarct related stenoses, 4 of 21 lesions were ≥50% but <100% progressed and one new lesion >50% appeared.

In Group II at restudy, all patients had significant coronary artery disease. There were 18 new total occlusions in the 15 patients. One patient had two new total occlusions and another patient, studied 7 years apart, had three new

Table 2. Angiographic Findings at Restudy: Comparison With Initial Angiogram

	Group I (n = 23)	p Value	Group II (n = 15)
Progression of coronary artery disease in IRA	22		*
Location of IRA/NTO			
LAD	7		6
LCx	6		4
RCA	9		8
Median % stenosis at initial angiogram of subsequent IRA/NTO	48	<0.05	73.5
Progression in IRA/NTO			
From <50%	11 (48%)	NS	6 (33%)
From >70%	5 (22%)	<0.01	11 (61%)

*All patients by definition had progression of coronary artery disease. IRA = infarct-related artery; LAD = left anterior descending artery; LCx = left circumflex artery; NTO = new total occlusion; RCA = right coronary artery.

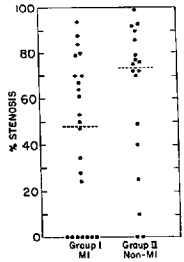

Figure 1. Initial percent stenosis of infarct-related artery at restudy in 23 Group I patients with myocardial infarction (MI) or new total occlusions in 18 Group II patients without myocardial infarction. Median values (**dashed line**) for each group are included.

total occlusions without sustaining an infarction. There were seven other coronary lesions that progressed after initial angiography. Severe hypokinesia or akinesia on the original left ventriculogram was seen in the distribution of a new total occlusion in only two angiograms.

Analysis of coronary risk factors (smoking, hypertension, diabetes, hypercholesterolemia and family history) revealed no differences between Groups I and II.

Initial percent stenoses of subsequent infarct-related arteries and new total occlusions (Fig. 1 and Table 2). In Group I, the percent stenosis of the arterial segment on initial angiography that was the site of infarction at restudy ranged from 0 to 94% (median 48%). On the initial angiogram, 11 (48%) of 23 lesions were <50% and only 5 (22%) of 23 were >70%. Of the nine Group I patients restudied within 1 year of the initial angiogram, only three had a >70% lesion in the subsequent infarct-related artery.

In Group II, the percent stenosis on the initial angiogram at the site of the new total occlusion at restudy ranged from 0 to 98% (median 73.5%). These differences between Groups I and II in the percent stenosis on initial angiography of the lesions that were the site of infarction or new total occlusion at restudy were statistically significant ($p < 0.05$). On the initial angiogram in Group II, 6 (33%) of 18 lesions were <50% stenotic, while 11 (61%) of 18 were >70% stenotic. The difference between Groups I and II in the frequency of lesions >70% was also statistically significant ($p < 0.005$). Three of the <50% lesions in Group II initially occurred in a single patient who was restudied after 84 months.

Differences between patients with Q wave and non-Q wave infarction (Table 3). In the eight Group I patients with non-Q wave infarction, the percent stenosis at initial angiography in the subsequent infarct-related artery was significantly higher than that in the 15 patients who subsequently sustained a Q

Table 3. Differences Between Q Wave and Non-Q Wave Infarction in Group I

	Q Wave (n = 15)		Non-Q Wave (n = 8)
% Stenosis at initial angiogram of subsequent IRA*	34	$p < 0.05$	80
Location of IRA			
Proximal	11 (73%)		6 (75%)
Distal	4 (27%)		2 (25%)
LAD	5 (33%)		2 (25%)
Non-LAD	10 (67%)		6 (75%)

*Median value. Abbreviations as in Table 2.

wave infarction ($p < 0.05$) (Fig. 2). At restudy, the percent stenosis of the infarct-related artery and its location were similar in those with non-Q and Q wave infarction. There was also no difference in the presence of collateral vessels to the infarct-related artery at restudy. Smoking was more frequent in patients who sustained a Q wave infarction (9 of 15) than in patients with non-Q wave infarction (1 of 18) ($p < 0.04$). There was no difference in the initial percent stenosis of the infarct-related artery between smokers and nonsmokers in the Q wave infarction group.

Prediction of subsequent infarction from analysis of the initial angiogram (Table 4). In Group I, initially there were a total of 31 lesions ≥50% and <100% in the 19 patients for whom both angiograms were available for analysis. Ten of these 31 lesions were the site of subsequent infarction at restudy. There were no differences in percent stenosis, left anterior descending coronary artery involvement or presence of the type II eccentric coronary lesion between stenoses that did and did not evolve into myocardial infarction. A proximal location was more frequent than a distal

Figure 2. Initial percent stenosis of infarct-related artery at restudy in 15 patients with Q wave infarction and 8 patients with non-Q wave infarction. Median values (**dashed line**) are included.

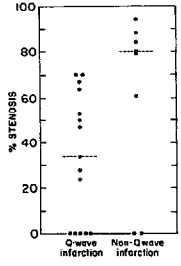

Table 4. Initial Angiographic Findings in Lesions Progressing to Infarction in Group I*

	Infarct-Related Stenosis (n = 10)		Noninfarct-Related Stenosis (n = 21)
Mean % stenosis	75.7 ± 10.9		68.7 ± 9.8
Median % stenosis	74.5		69
Type II eccentric lesion	1 (10%)		4 (19%)
Location of stenosis			
LAD	3 (30%)		4 (19%)
Proximal segment	10 (100%)	p < 0.03	13 (62%)

*p = NS for all except for proximal location. Abbreviations as in Table 2.

location in lesions that progressed to infarction ($p < 0.03$). In these same 19 Group I patients, 10 had unstable angina at the time of the initial angiography; on the initial angiogram, 4 of these 10 patients had lesions <50% in the subsequent infarct-related segment, and only 4 patients had lesions >70%. Stepwise multivariate analysis of clinical and angiographic characteristics of lesions present at the initial study (lesion location, percent narrowing, left anterior descending coronary artery involvement, type II eccentric morphology, number of diseased vessels and presence of unstable angina) revealed proximal location of the stenosis as the only significant ($p < 0.02$) predictor of evolution of a lesion to infarction.

Discussion

In acute myocardial infarction, total occlusion of the infarct-related artery is usually secondary to acute thrombus formation. As demonstrated in pathologic studies (1,2), this acute thrombotic process is frequently precipitated by disruption of an atherosclerotic plaque. Contact of the flowing blood with the undersurface of the disrupted plaque results in platelet deposition followed by activation of the clotting system and thrombus formation (8). In stable angina pectoris as well as in other acute coronary syndromes like unstable angina, total occlusion of a coronary artery may also be found. However, total occlusion of the ischemic- or infarct-related artery on angiography is less common in acute presentations of unstable angina and non-Q wave infarction than in Q wave infarction (9,10). The variable clinical presentation of patients with total occlusion suggests differences in the rate of development of total occlusion or in the amount of collateral circulation, or both.

Initial characteristics of coronary lesions predisposing to acute infarction. This retrospective analysis suggests that in many patients who subsequently developed myocardial infarction (Group I), prior angiography revealed lesions that were <50% occlusive in the infarct-related segment of the infarct artery. Thirty percent of patients in Group I had a normal appearing artery in this segment on initial angiography. Moreover, most patients did not have lesions >70% stenotic in this segment. Although the degree of narrowing in these arteries just before the onset of infarction was unknown, it is assumed that a more significant narrowing in the infarct-related artery had not slowly developed before the acute event. We suspect that this may have occurred because progression of coronary artery disease at restudy was uncommon in noninfarct-related lesions in Group I. Therefore, disruption of a mild or moderate atherosclerotic plaque with resultant thrombosis formation and total or subtotal occlusion probably explained the myocardial infarction in these patients. In patients with a previously normal-appearing infarct artery, we assume that some degree of diffuse coronary disease was indeed present, but was not detectable by these angiographic techniques.

Role of collateral flow and degree of stenosis. In other Group I patients with myocardial infarction, a significant lesion was demonstrated on angiography before myocardial infarction. Here, collateral blood flow was probably inadequate to prevent myocardial infarction after the infarct-related artery acutely occluded. Of interest, no arterial segment with a >90% stenotic lesion on the first angiogram subsequently developed Q wave myocardial infarction. These data suggest that in some cases, severe stenosis may protect the myocardium from the subsequent development of a Q wave infarction in its distribution. In non-Q wave infarction, the initial percent stenosis of the subsequent infarct-related artery was significantly higher than in patients with Q wave infarction. Therefore, the pre-existant severity of stenosis may be an important determinant of the extent of subsequent infarction, in addition to other variables such as the duration or presence of total occlusion in the infarct artery at the onset of infarction and the acuteness of the obstruction (11). Of interest, smoking was more common in patients who sustained Q wave infarction in comparison with a non-Q wave infarction. The significance of this finding is unknown.

In contrast, in Group II patients, a new total occlusion without the evolution of myocardial infarction was preceded by a significant lesion (>70% diameter reduction) on initial angiography in 61% of patients. We suspect that in most patients, total occlusion occurred more slowly than in patients in Group I, allowing time for the recruitment of collateral vessels (12). Because most Group II patients had unstable angina at restudy, collateral vessels may have contributed to the prevention of infarction, but not to the prevention of ischemia.

Comparison with previous studies. There is a similarity between this study and previous work (4,5) on progression of coronary disease in unstable angina. In patients restudied after an episode of unstable angina, progression from normal or previously insignificant coronary artery disease occurs quite commonly. We have previously shown (4) in patients with unstable angina that 19 of 26 lesions demonstrating

progression on restudy were initially <50% narrowed. Singh (13) also found new lesions in 52% of patients restudied after either myocardial infarction or unstable angina and 86% of these lesions were significant. Therefore, it appears that in myocardial infarction as well as in unstable angina, angiographic progression of coronary artery disease from less than significant lesions may occur.

Mechanism of thrombotic occlusion of mild lesions. Recent experimental studies (14–16) suggest a possible mechanism for thrombotic occlusion of mild or moderate atherosclerotic plaques. If disruption results in a large fissure through the fibrous cap of the plaque, this vessel wall injury exposes the deeper arterial elements (for example, collagen and fat) to the flowing blood. In an ex-vivo perfusion chamber, our laboratory (16) has demonstrated that platelet deposition is severe within 1 h of deep vessel injury, and it is directly related to the shear force at the site of plaque disruption. At shear rates similar to those found in mild to moderate coronary stenosis, platelet thrombi occur and remain adherent to the collagen surface. Deceleration of blood flow distal to the stenosis induces flow separations and vortices that favor the deposition of fibrin and contribute to the thrombotic occlusion. It is well known that small plaques have a high fat content, and these probably have a high frequency of plaque disruption. In our study, proximal location was predictive of lesion evolution to infarction. Perhaps the increased shear rate and turbulence in a large proximal plaque lead to mild endothelial damage followed by platelet deposition, stasis and thrombus formation.

Study limitations. This retrospective analysis represents a select group of patients who were restudied after myocardial infarction (Group I) or because of poorly controlled or unstable symptoms (Group II). These selection criteria automatically eliminated most patients admitted with myocardial infarction because of the absence of an angiogram before infarction. In addition, these data do not necessarily apply to patients with an uncomplicated course after infarction because these patients may not require angiography. Similarly, in Group II, patients with a new total occlusion but without any change in symptoms would not have been examined by the present study.

Although the finding that mild or moderate coronary artery disease progresses to infarction is probably unaffected by patient selection, one must interpret cautiously the infrequent progression of lesions >70% stenotic and especially >90% stenotic to infarction. Moise et al. (17) found that lesions >80% stenotic were a powerful predictor of subsequent occlusion on restudy, and 39% of patients sustained an interim myocardial infarction. However, the distinction between a Q and non-Q wave infarction was not made in that study. It is likely that in some patients with severe lesions in our study, a decision to subject them to revascularization was instrumental in preventing infarction in the distribution of the ischemia-producing artery; these patients were excluded from analysis. In addition, 36% of Group I patients had a patent infarct-related artery on angiography after myocardial infarction. Because there was a delay in angiography after myocardial infarction, spontaneous reperfusion of the infarct-related artery probably occurred in some of these patients.

Clinical implications. If myocardial infarction is often the result of disruption of a mild or moderate atherosclerotic coronary plaque, thrombolytic therapy given within hours of infarction might be expected to produce regression to a less than significant lesion. Under these circumstances, adjunctive measures like angioplasty would be, for the most part, unnecessary because the infarct-related artery would contain a noncritical stenosis. However, in reality, after the administration of intravenous tissue plasminogen activator or intracoronary streptokinase, significant stenoses are present in most infarct-related arteries (18,19). Recent evidence (20) from clinical trials of thrombolysis in acute myocardial infarction have shown, in a minority of cases, regression of coronary artery disease to an insignificant coronary lesion after successful thrombolytic therapy. Videodensitometric analysis of magnified angiographic images after thrombolysis in myocardial infarction have demonstrated (21) residual thrombus superimposed on mild to moderate atherosclerotic plaques. Furthermore, other investigators have also recently shown in preliminary reports (22,23) that a prolonged infusion of intravenous tissue plasminogen activator for 12 to 24 h results in progressive increases in minimal diameter and decreases in diameter stenosis of the infarct-related artery. Thus, incomplete thrombolysis after reperfusion of a totally occluded artery in acute myocardial infarction with standard doses of thrombolytic agents may be more common than previously reported. However, in some patients with disruption of a mild or moderate plaque and subsequent infarction, changes in plaque geometry after disruption rather than incomplete thrombolysis may be responsible for a significant lesion remaining after clinically successful thrombolysis.

Our data also suggest that usually one cannot predict the location of a subsequent infarction from analysis of a previous coronary angiogram. Significant lesions >50% stenotic in diameter seen on initial angiography in 31 segments of the coronary arteries in Group II patients were the site of subsequent myocardial infarction in only 10 patients. Analysis of coronary morphology, looking for the presence of the type II eccentric lesion, initial percent stenosis or the presence of unstable angina at the time of initial angiography, did not improve the predictive power for determining the site of a subsequent myocardial infarction.

Conclusions. Myocardial infarction appears to develop frequently from a coronary lesion that was less than severe on prior angiography. Because the potential implication of this finding is important, a larger number of patients must be prospectively evaluated to confirm these findings.

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