Comparison of Coronary Angiographic Findings in Acute and Chronic First Presentation of Ischemic Heart Disease

Peter Bogaty, MD; Stephen J. Brecker, MD; Sarah E. White, RGN; Robert N. Stevenson, MD; Hassan El-Tamimi, MD; Raphael Balcon, MD; and Attilio Maseri, MD

Background. It is generally assumed that the clinical manifestations of ischemic heart disease occur randomly on the same underlying pathological process. Therefore, coronary angiographic findings should be similar whether the first presentation of ischemic heart disease is acute myocardial infarction or uncomplicated chronic stable angina.

Methods and Results. We studied 102 patients (men ≤60 years old, women ≤65 years old) presenting with either acute myocardial infarction as first manifestation of coronary artery disease with a concomitant coronary angiogram (55 patients; mean age, 50.2 years) or stable angina for at least 2 years with no history, ECG, or left ventriculographic evidence of any acute event and with an angiogram performed at least 2 years after initial symptoms (47 patients; mean age at symptom onset, 51.7 years). These angiograms were evaluated blindly for severity (number of vessels diseased, stenoses ≥50%, occlusions), extent of disease (with an index derived by assigning a score of 0–3 per segment, depending on the proportion of lumen length irregularity and dividing the sum by the number of visualized segments), and pattern (discrete: three or fewer loci of disease never involving more than 50% of the length of any segment or diffuse: anything exceeding this).

Patients with unheralded myocardial infarction had fewer vessels diseased, fewer stenoses and occlusions, and a lower extent index than those with uncomplicated stable angina (mean±SD of 1.3±0.8 versus 2.1±0.8, p<0.001; 2.1±1.8 versus 3.9±1.8, p<0.001; 0.6±0.6 versus 1.0±0.9, p<0.02; and 0.6±0.5 versus 1.2±0.5, p<0.001, respectively). A discrete pattern was present in 54.5% of patients with unheralded infarction and in only 8.5% of those with uncomplicated angina (p<0.001).

Conclusions. These very different angiographic findings suggest that unheralded acute myocardial infarction and uncomplicated chronic stable angina do not occur randomly on a common atherosclerotic background but rather that additional factors, such as a varying propensity to thrombosis, may predispose to one or the other of these two clinical syndromes. (Circulation 1993;87:1938–1946)

Key Words • angina • arteriosclerosis • ischemia • vessels • myocardial infarction • angiography

Isolated epidemiological reports have suggested that the possible clinical presentations of ischemic heart disease may have different risk factor profiles.1–8 More recent prospective and cross-sectional studies have implicated procoagulant markers predisposing to acute coronary disease.9–16 The traditional view is that acute and chronic clinical manifestations occur randomly on the same underlying disease process. Clinicians, however, have often been intrigued by the observation that acute coronary events can occur on a background of quite limited angiographic disease, whereas other patients with far more extensive disease seem to remain stable for a long time. Patients may present with acute myocardial infarction as the first event in their history of ischemic heart disease and may then enter a chronic phase. Other patients have an initially chronic presentation that may later be punctuated by acute coronary events.17 Acute and chronic elements thus tend to be intermeshed in most patients as a history of ischemic heart disease progresses. Consequently, if patients are studied a long time after their initial presentation, pathogenetic, clinical, and angiographic features that might be uniquely related to acute or chronic coronary syndromes will be confounded and obscured, as suggested by the postmortem studies of Rissanen.18

These considerations led to the present study. We speculated that if two pure subsets of coronary disease could be identified, one represented by unheralded acute myocardial infarction and the other by uncomplicated chronic stable angina, then a comparison of their respective angiographic features could have implications regarding the substrate of acute and chronic ischemic heart disease. Similar angiographic features

From the Cardiovascular Research Unit, Royal Postgraduate Medical School (P.B., S.E.W., H.E.-T., A.M.), Hammersmith Hospital, and the Department of Cardiology (S.J.B., R.N.S., R.B.), London Chest Hospital, London.

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Address for correspondence: Peter Bogaty, MD, Quebec Heart Institute, Hospital Laval, 2725 Chemin Ste. Foy, Ste. Foy, Quebec, Canada, G1V 4G5.

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would suggest an identical common substrate, i.e., atherosclerosis and the random occurrence of acute events on this substrate; dissimilar features would suggest interaction with another pathological process, e.g., a thrombogenic susceptibility. No study has yet addressed this question. Because the conventional evaluation of coronary angiograms based on the number of significantly narrowed vessels provides limited information on angiographic appearance, we also used novel descriptive criteria to account not only for atherosclerotic disease severity but also for its extent as well as to identify different arteriographic patterns of disease. This study reveals striking differences in the angiographic findings in these two ischemic syndromes.

**Methods**

**Selection Criteria**

The consecutive discharge summaries of all patients hospitalized in two cardiovascular units over a 13-month period were scanned. The two hospitals, in Greater London, are tertiary care centers, with referrals from outlying areas constituting about half the admissions. The patient composition reflects the heterogeneous socioeconomic and ethnic nature of the base population. The study commenced in April 1989. It included 10 months retrospectively and 3 months prospectively. The summaries of those patients undergoing coronary arteriography during that hospitalization (n = 2,605) were examined to see whether they appeared compatible with one of the following two sets of criteria.

**Un heralded acute myocardial infarction.** 1) Men had to be ≤60 years old and women ≤65 years old at the time of this presenting event. 2) The diagnosis of myocardial infarction required the presence of at least two of the following: characteristic chest pain, a cardiac enzyme rise greater than twice the upper normal limit, and compatible ECG changes lasting >48 hours. 3) Myocardial infarction had to be the first manifestation of ischemic heart disease in these patients. A proadrome of a maximum 4 weeks of new-onset chest pain was performed. 4) Coronary arteriography had to be performed within 3 months of infarction.

**Uncomplicated chronic stable angina.** This required that 1) men be ≤60 years old and women ≤65 years old at the time of first onset of angina. 2) A minimum 2-year history of typical stable angina with a positive exercise test was necessary. 3) No history of any acute event, defined as unstable angina or acute myocardial infarction, from the time of first symptom onset for at least 2 years was allowed. Unstable angina was defined as prolonged chest pain or a sharp and significant change in the pattern of established angina or new-onset angina with symptoms at rest or on minimum exertion not culminating in myocardial infarction. 4) No Q waves, no R/S >1 in lead V1 or V2, and no left bundle branch block on the resting ECG were allowed. 5) No segmental akinesis or dyskinesis on left ventriculography was allowed. 6) Coronary arteriography had to be performed at least 2 years after initial symptoms, and there had to be no history of any acute event up to and including the time of this angiogram.

The hospital records of those patients whose discharge summaries appeared compatible with one or the other of these sets of criteria were then examined (n = 412), and if they remained compatible (n = 165), these patients were then contacted either by telephone or by visit, and a structured questionnaire was administered. Four of these patients had already died, but information judged satisfactory could be obtained for three of them. Six other patients could not be reached and were excluded. Of the 158 patients reaching this last phase, 64 presented with infarction and 94 with angina. The purpose of the questionnaire was to confirm the exactitude of the clinical history, specifically to ensure that those presenting with myocardial infarction had no preceding history suggestive of angina or of a prior acute coronary event (nine of the 64 were eliminated) and that those presenting with angina had no history, however brief, compatible with an acute coronary event (47 of the 94 were eliminated). This large number of exclusions in the angina group at the interview stage was a result of the frequent finding on close questioning of symptoms highly suggestive of instability, particularly at the inaugural history (usually prolonged chest pain at rest or recurrent pain on minimal activity). Final selection of patients was carried out in committee with the participating physicians in such a way that the latter remained blinded to the patients' identity. The target recruitment figure for each subset was 50 or 60 patients. This figure was first reached for myocardial infarction, so subsequent consecutive recruitment for angina only continued until a comparable number was attained.

**Angiographic Criteria**

During this recruitment phase, coronary angiograms unrelated to the study were examined. Initially, since the conventional criterion of number of vessels diseased provides limited information on angiographic appearance, methods previously described for scoring coronary atherosclerosis were considered.19-23 Because these were found to be either too restrictive or too cumbersome and do not identify particular patterns of disease, a more comprehensive analysis of coronary angiograms based on three descriptive concepts—severity, extent, and pattern—was developed. Severity pertains exclusively to degree of narrowing (“transverse disease”) and was evaluated by counting the number of vessels diseased, stenoses, and occlusions. Extent, as distinct from severity, considers the proportion of each coronary segment that appears abnormal (“longitudinal disease”). Pattern implies a global appreciation of atherosclerotic involvement; a discrete pattern and a diffuse pattern could be identified.

Under severity, the three indexes considered were thus 1) vessels diseased, classically the number of major epicardial vessels with ≥70% narrowing of the lumen diameter. The maximum number of vessels diseased was three. A left main stem stenosis of ≥50% counted as two vessels.24 2) Stenoses, the total number of ≥50% narrowings on all the vessels of the angiogram. A long stenosis whose length attained twice the normal lumen diameter was counted as two stenoses. A maximum of three stenoses was permitted per coronary arterial segment (see below). Nonocclusive stenoses were classified as concentric, eccentric type I, or complex (eccentric type II or multiple irregularities) according to the criteria of Ambrose et al.25 3) Occlusions, which also included subtotal occlusions if there was clearly delayed...
or incomplete antegrade flow. The quality of collaterals (absent-to-poor or good-to-excellent) was noted.

The extent of atherosclerosis was quantified in a score that was normalized to an extent index. This was done simply and visually by assigning a score of 0–3 per segment of the coronary arterial tree. A segment was scored 0 if it appeared angiographically normal; 1 if \( \leq 10\% \) of its length appeared abnormal (narrowed and/or irregular); 2 if \( >10\% \) up to 50\% of its length was abnormal; and 3 if \( >50\% \) of its length was abnormal. If a segment was occluded or suboccluded with compromised antegrade flow, it was arbitrarily given a score of 2 (or 3 if any abnormality proximal to the occlusion involved more than half the nonoccluded portion of the segment), and segments distal to it were not scored. The extent score was the total score of the 15 segments of the coronary tree. The division of the angiogram into segments was a slightly modified version of the American Heart Association classification.\(^{26}\) A dominant right coronary artery had five segments: proximal, mid, distal, posterior descending artery, and posterior left ventricular. The left main stem was one segment. The left anterior descending artery had five segments: proximal, mid, distal, and two for the diagonals. The circumflex artery (of a nondominant left coronary artery) had four segments: proximal, distal, obtuse marginal, and another marginal. The extent index was the extent score divided by the number of segments that could be properly visualized by antegrade flow. Thus, the extent index could range from 0 (score of 0) to a maximum of 3 (score of 45 divided by 15 segments). An illustrative schema of stenoses and extent score appears in Figure 1.

For the pattern concept, a discrete pattern was one in which there was a maximum of three loci of disease or abnormality never involving more than half the length of any segment, with the rest of the angiogram having a wholly normal appearance. An occlusion could count as a locus of a discrete pattern provided that, if there was any abnormality just proximal to it, this did not involve more than half the nonoccluded portion of that segment. If a locus appeared astride two segments, it could count as the locus of a discrete pattern if the total length of the abnormality involved 25\% or less of the combined length of the two segments. Exceptionally, the left main stem, because of its short length, could be considered as a locus of a discrete pattern even if its entirety appeared diseased. A diffuse pattern was anything exceeding the criteria for a discrete pattern, either more than three loci of disease and/or any involvement exceeding 50\% of the length of any segment.

Once these descriptive criteria were defined and practiced, they were then applied to the constituted study group. Each angiogram was evaluated independently by two observers blinded to all clinical data including the left ventriculogram. If there had been thrombolysis or angioplasty during angiography (in case of a myocardial infarction presentation), this part of the film was removed before the angiogram was presented to the observers. After initial evaluation, each angiogram was studied jointly to reach agreement. When agreement was not possible for the criteria of severity (vessel disease \( \geq 70\% \) narrowing or stenosis \( \geq 50\% \) narrowing), a computerized contour detection system (CAAS, Pie Data Medical, Holland) was applied, as previously described.\(^ {27}\) This was done for 16 of 171 lesions of vessel disease (9\%) and 35 of 293 stenoses (12\%). Reproducibility of the extent scoring system was good, with 75\% of the scores of two observers within one point of each other, 90\% within two points, and 95\% within three points. When agreement was not possible regarding the criteria of extent of disease (13 of 102 angiograms), a third observer intervened. Regarding pattern of disease, it was found that all angiograms could be quite easily and quickly classified as having a discrete or diffuse pattern (Figure 2). The two observers initially disagreed about pattern in 12 of 102 angiograms, and a third observer intervened in nine of these. The data were analyzed only after final angiographic evaluation of the entire study group.

Risk Factor Criteria
Conventional risk factors were examined from hospital records, rechecked by direct questioning, and completed with data from personal physicians and, where appropriate, from hospital follow-up. Recent smokers were those who had been smoking five or more cigarettes a day for at least 3 months in the year preceding angiography. Past smokers were those who had consumed five or more cigarettes a day for a year or more at any time (within 20 years) up to 1 year before angiography. Serum cholesterol was measured by enzymatic assay. For myocardial infarction patients, it was determined on admission and at least 2 months later. For all subjects, the highest value found was retained. Hypertension required a satisfactory prior diagnosis or an arterial pressure \( \geq 140/90 \) mm Hg on three occasions except during angiography or an evolving infarction. Diabetes mellitus required either a satisfactory prior diagnosis or the presence of at least two fasting plasma glucose values \( \geq 140 \) mg/dL. For myocardial infarction patients, this had to be at least 1 week after infarction. A family history was positive if any first-degree relative had sustained an acute or chronic manifestation of coronary disease before the age of 65 years.
Statistical Analysis
Results are expressed as mean±SD. Comparisons were made by the unpaired two-tailed t test for normally distributed values; otherwise, the two-tailed Mann-Whitney U test corrected for ties was applied. The $\chi^2$ test with continuity correction was used to analyze differences in proportions when the smallest expected value was $>5$; otherwise, the Fisher exact test was used. Correlations between variables were tested by simple regression analysis. A value of $p<0.05$ was considered statistically significant.

Results
The study group consisted of 102 patients. This constitutes <5% of all patients with coronary artery disease undergoing catheterization during this same period. Of this group, 55 patients (80% men) fulfilled the criteria for unheralded myocardial infarction (two thirds had Q waves) and 47 (78.7% men) for uncomplicated chronic stable angina. The age of the infarction group was 50.2±8.3 years (range, 32–63 years); of the angina group, it was 51.7±5.6 years (range, 39–60 years) at the time of symptom onset and 56.7±6.2 years at the time of cardiac catheterization. The subjects with stable angina had thus been symptomatic for almost 5 years before angiography. The indications for coronary angiography in the infarction patients were residual ischemia (36%) with angiography often performed during that hospitalization, a research protocol requiring angiography before starting thrombolysis (38%), and
unrelated reasons at the discretion of the consultant cardiologist (26%).

When the angiograms of these two cohorts were evaluated in terms of the standard criteria of number of vessels diseased, the infarction group had 1.3±0.8 compared with 2.1±0.8 vessels diseased for the angina group (p<0.001) (Figure 3). The distribution of zero-, one-, two-, and three-vessel disease was 9.1%, 61.8%, 18.2%, and 10.9%, respectively, in the infarction group; it was 0%, 25.5%, 38.3%, and 36.2%, respectively, in the angina group. When the other two indexes of severity (stenoses and occlusions) were considered, the results were comparable. Patients with unheralded myocardial infarction had 2.1±1.8 stenoses and 0.6±0.6 occlusions, whereas those with uncomplicated stable angina had 3.9±1.8 stenoses (p<0.001) and 1.0±0.9 occlusions (p<0.02) (Figure 3). The distribution of lesions according to concentric, eccentric type I, and complex morphology was 25.6%, 59.8%, and 14.6%, respectively, in the infarction subset and 21.5%, 75.4%, and 3.1%, respectively, in the angina subset. Of the myocardial infarction patients, 22 (40%) had received thrombolytic therapy before angiography, and 10 of these (45.5%) had an occluded infarct-related artery; of the 33 (60%) who did not receive thrombolytic therapy at the time of the study angiogram, 19 (57.6%) had an occluded infarct-related artery. Therefore, the impact of thrombolysis on the number of occlusions in the infarction group was not very important. Of the infarction patients with occlusions, 39.5% had good-to-excellent collaterals; the corresponding figure for angina patients was 95.7%.

The findings for extent of disease were similar to those for severity (Figure 3). The extent index of those with infarction was 0.6±0.5; of those with angina, it was 1.2±0.5 (p<0.001). The indexes of severity, vessels diseased, stenoses, and occlusions correlated highly with each other (p<0.001 for all), and all correlated highly with the extent index (p<0.001 for all).

A diffuse pattern was extremely frequent in the uncomplicated stable angina subset (91.5%); a discrete pattern predominated in the unheralded infarction subset (54.5%) (Figure 3). Of the 34 patients with a discrete pattern, this involved only one locus of disease in 50%, two loci in 32.4%, and three loci in 17.6%.

The angiographic findings in patients with uncomplicated stable angina with respect to the duration of their anginal symptoms are summarized in Table 1. Subjects with a 2-year history of angina are compared with those with a history of ≥7 years. The former were 54±4.6 years old at the time of angiography; the latter, 60.7±3.7 years old. Despite these differences in age and duration of angina, the findings are not significantly different.

As for risk factors (Table 2), serum cholesterol levels and the proportion of those with a positive family history were comparable in the two groups. A recent smoking history was obtained nearly twice as often in those with myocardial infarction than in those with stable angina (60% versus 31.9%, p<0.01). When past and recent smoking history were considered, however, the proportions were comparable. Hypertension was found twice as frequently in angina patients (63.8% versus 30.9%, p<0.01). There was one patient with diabetes in the infarction group and seven such patients in the angina group (p=0.02).

None of the indexes of severity correlated with serum cholesterol either in the angina or the infarction subsets. The extent index, however, did correlate with serum cholesterol in the stable angina group (r=0.4, p=0.015) but not in the myocardial infarction group (r=0.24, p=0.11).

None of the unheralded infarction patients were taking aspirin on a regular basis before their event. Of the angina patients, fewer than half (20 of 47) were on regular aspirin therapy at the time of angiography. Only four angina patients and no infarction patients were taking lipid-lowering agents.

Discussion

The major finding of this study is the markedly different angiographic appearance of unheralded acute myocardial infarction compared with uncomplicated chronic stable angina. Whether the standard criterion of number of vessels diseased was used or the number of

<p>| Table 1. Angiographic Findings: 2-Year Versus ≥7-Year History of Uncomplicated Stable Angina |
|-----------------------------------------------|------------------|------------------|------------------|------------------|------------------|</p>
<table>
<thead>
<tr>
<th>Vessels diseased (No.)</th>
<th>2-Year history (n=12)</th>
<th>≥7-Year history (n=13)</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>Stenoses (No.)</td>
<td>2.2±0.8</td>
<td>2.1±0.8</td>
<td>0.8</td>
</tr>
<tr>
<td>Occlusions (No.)</td>
<td>3.8±1.3</td>
<td>4.2±2.0</td>
<td>0.6</td>
</tr>
<tr>
<td>Extent index</td>
<td>0.8±1.0</td>
<td>1.3±0.9</td>
<td>0.1</td>
</tr>
<tr>
<td></td>
<td>1.12±0.46</td>
<td>1.28±0.44</td>
<td>0.4</td>
</tr>
</tbody>
</table>
stenoses or occlusions or an index of disease extent, the results are concordant and discriminate clearly between the two groups. The difference is even more striking when pattern of disease is considered. A discrete pattern is frequent in and characteristic of un heralded myocardial infarction; a diffuse pattern is almost the rule in uncomplicated chronic stable angina.

These contrasting angiographic findings are intriguing. On the one hand, atherosclerosis may diffusely involve the epicardial coronary arteries and even progress to total occlusion without causing clinically apparent episodes of instability or signs of infarction. On the other, sudden occlusion can cause infarction in the presence of very limited angiographically detectable atherosclerosis. It is the study of patients at the time of first presentation of ischemic heart disease that is crucial to a recognition of these distinct patterns. The premise of this study was that if acute and chronic first clinical presentations of ischemic heart disease occurred randomly on the same underlying pathological process, similar angiographic findings would be expected. These dissimilar findings suggest that, in addition to slowly progressive coronary atherosclerosis, another pathological process and another constellation of risk factors, possibly related to increased thrombogenicity, could be involved, accounting for these different angiographic appearances and clinical presentations.

Comparison With Other Studies

It has not been generally appreciated that myocardial infarction and stable angina as pure isolated presentations might possess different angiographic features. We are not aware of any other coronary arteriographic study that has attempted to characterize patients according to rigorous criteria of exclusively acute and exclusively chronic coronary disease. As a result, with the exception of the morphological analysis of certain individual lesions,25,28,29 the angiographic impression of acute and chronic coronary disease tends to be that of an undifferentiated entity. Pathological reports as well as angiographic studies, few of which are stratified according to prior clinical status and which often have a selection bias in favor of advanced disease, have reinforced the tendency to assume that coronary artery disease is generally severe when its clinical manifestations appear, whatever they might be.20–29

It has already been suggested, however, that angiographic appearance might differ depending on clinical presentation. In a pathological study, Rissanen18 observed that atherosclerosis was more severe in patients who had had symptomatic heart disease and in old myocardial infarction and least severe in those with sudden death as the first manifestation of coronary disease. When infarction is the initial presentation of coronary disease, it appears more often associated with single-vessel involvement, whereas angina before infarction may be more frequently associated with multivessel disease.40,41 Roberts et al42 found a higher incidence of multivessel disease in chronic angina than in new-onset angina. Single-vessel disease and poor collateral flow are the most frequent angiographic findings in patients who develop a ventricular aneurysm after a first anterior myocardial infarction.43

The relatively low number of occlusions in the myocardial infarction patients can be only marginally attributed to thrombolyis (see “Results”) and more to spontaneous recanalization, because angiography was performed within 3 months of infarction. But it is the large number of occlusions in the stable angina group that is intriguing, because we had gone as far as possible to exclude acute coronary events in this cohort. The consistent presence of a developed collateral circulation and of preserved ventricular function suggests gradual rather than abrupt development of occlusion in chronic stable angina patients. Pathological studies lend support to this observation. In a pioneering work, Blumgart et al44 noted coronary occlusions in the absence of necrosis, fibrosis, or a clinical history and with evidence of a developed collateral circulation. Rissanen18 found coronary occlusions in 12% of a control group without any myocardial signs of disease. Hangartner et al45 found recanalized vessels in 10 of 16 patients with stable angina in the absence of infarction.

Nature of Cohorts and Potential Limitations

Stringent clinical criteria were applied so that the two cohorts might constitute, as far as possible, pure subsets of acute and chronic coronary disease. The interview questionnaire was structured to make doubly certain of the absence of "contaminating events" in either group. In the angina subjects, the ECG criteria and the requirement for no dyskinesia or akinesia on left ventriculography were intended to exclude, as far as possible, the occurrence of unsuspected myocardial infarction. Hypokinesia was not an exclusion criterion because it is open to interpretation variability and might also reflect
a hibernating state. In spite of these precautions, the occurrence of prior silent or inapparent acute coronary events cannot be excluded in the stable angina cohort nor prior silent ischemia in the unheralded infarction cohort. Notwithstanding these possibilities, the angiographic findings in the two groups differ quite markedly. Because this is an angiographic study, in each case the reason for this procedure was reviewed to see whether these patients are representative of unheralded infarction and chronic angina patients in general. In 36% of the infarction patients, angiography was performed because of possible residual ischemia, a percentage within the limits observed in other studies. In 38%, angiography was carried out acutely under a research protocol requiring arteriographic documentation before thrombolysis. The entry criteria of this protocol were sufficiently wide as to cover a representative sample of patients sustaining a myocardial infarction. In 26%, angiography was performed at the discretion of the attending physician for reasons present in general clinical practice, the relatively young age of a patient, a non–Q-wave infarction, left ventricular dysfunction, or an institutional policy favoring angiography after infarction. In the angina group, angiography was never performed in the context of an unstable state. Almost half of these patients were referred from areas removed from tertiary facilities. Had they originated from urban centers, it is likely that they would have come to catheterization earlier. However, this was precisely what was needed for the purposes of this study, an “undisturbed” stable subset that could give some idea of what chronic morphology might be.

It may be that two additive confounding factors, greater age and a longer clinical history, contributed to the more severe and extensive disease found in subjects with stable angina. Thus, although the ages of the two cohorts were comparable at the time of onset of symptoms, the stable angina group was 6 years older and had been symptomatic for almost 5 years at the time of angiography. An indirect way of considering this problem is to compare the ages (see “Results”) and angiographic findings of those patients with a 2-year history of stable angina with those with a history of ≥7 years of angina (Table 1). Despite the differences in age (≥6 years) and duration of angina, there is only a very weak trend to more severe and extensive disease in the latter group. Thus, although the effects of age and duration of clinical history cannot be ruled out, it is unlikely that these factors might account for the major differences in angiographic findings between the infarction and angina cohorts.

Although angiography underestimates coronary disease, especially in the presence of diffuse smooth atherosclerotic narrowing, the dissimilar findings in the two patient groups strongly suggest significantly different disease involvement.

Whereas the large number of ex-smokers in the angina group suggests risk factor modification after diagnosis, a recent smoking history is frequently elicited in the infarction group, suggesting a potential trigger for acute events. This has already been evoked in previous studies and is indirectly suggested by the link between cigarette smoking and myocardial infarction with angiographically normal coronary arteries. Although the dissimilar risk factor profiles noted in these two patient groups are provocative, this must be tempered by the limited numbers as well as by the retrospective nature of the data collection.

Conclusions

The study of patients at the ends of the clinical spectrum of ischemic heart disease has revealed marked differences in severity, extent, and pattern of coronary disease. At one end of the spectrum, diffuse atherosclerosis can progress to critical stenosis and even to occlusion without provoking infarction or clinically apparent episodes of instability. At the other end, myocardial infarction abruptly inaugurates clinical ischemic heart disease most often in coronary arteries discretely affected by angiographically detectable atherosclerosis. Although it is well accepted that the mechanisms responsible for stable and unstable coronary syndromes are different, the contrasting angiographic features (and dissimilar risk factor profiles) found in these two pure ischemic syndromes suggest that, in addition, the pathogenetic substrate on which these mechanisms come into play might not be an undifferentiated entity. These findings lend support to the concept that although atherosclerosis constitutes a strong predisposing factor for acute coronary disease, acute disease may be neither an inherent and inevitable consequence nor a random expression of atherosclerosis. Studies already suggest that the occurrence of acute coronary events in chronic ischemic heart disease patients is difficult to predict from clinical parameters or from the severity of coronary stenoses.

It is perplexing why some subjects with coronary atherosclerosis appear “protected” for at least some time from acute disease, whereas in others, myocardial infarction occurs precociously on limited angiographically detectable atherosclerosis. Factors in addition to the presence of atherosclerosis that may involve differences in genetic susceptibility and in acute (e.g., thrombogenic) as opposed to chronic coronary disease risk factors could interact in some decisive manner. Work at these two ends of the spectrum of ischemic heart disease may provide clues to these intriguing mechanisms.

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