Thermal Heterogeneity Within Human Atherosclerotic Coronary Arteries Detected In Vivo

A New Method of Detection by Application of a Special Thermography Catheter

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Background—Activated macrophages play an important role in the pathogenesis of acute ischemic syndromes. It has been postulated that detection of heat released by activated inflammatory cells of atherosclerotic plaques may predict plaque rupture and thrombosis. Previous ex vivo studies have shown that there is thermal heterogeneity in human carotid atherosclerotic plaques.

Methods and Results—To measure the temperature of human arteries in vivo, we developed a catheter-based technique. Ninety patients (45 with normal coronary arteries, 15 with stable angina [SA], 15 with unstable angina [UA], and 15 with acute myocardial infarction [AMI]) were studied. The thermistor of the thermography catheter has a temperature accuracy of 0.05°C, a time constant of 300 ms, and a spatial resolution of 0.5 mm. Temperature was constant within the arteries of the control subjects, whereas most atherosclerotic plaques showed higher temperature compared with healthy vessel wall. Temperature differences between atherosclerotic plaque and healthy vessel wall increased progressively from SA to AMI patients (difference of plaque temperature from background temperature, 0.106 ± 0.060°C in SA, 0.683 ± 0.347°C in UA, and 1.472 ± 0.691°C in AMI). Heterogeneity within the plaque was shown in 20%, 40%, and 67% of the patients with SA, UA, and AMI, respectively, whereas no heterogeneity was shown in the control subjects.

Conclusions—Thermal heterogeneity within human atherosclerotic coronary arteries was shown in vivo by use of a special thermography catheter. This heterogeneity is larger in UA and AMI, suggesting that it may be related to the pathogenesis. (Circulation. 1999;99:1965-1971.)

Key Words: ischemia ■ coronary disease ■ plaque ■ heat

Activated macrophages play an important role in the pathogenesis of acute ischemic syndromes by promoting plaque rupture or thrombosis and vasoconstriction in nonruptured but inflamed plaques. In addition, recent evidence suggests a possible role for infection in the pathogenesis of atherosclerosis. Furthermore, relative blood viscosity rises and aggregation of red cells increases with temperature increases. Previous ex vivo studies have shown that there is thermal heterogeneity in human carotid atherosclerotic plaques. However, the temperature of coronary arteries has not been investigated in humans in vivo.

To measure the temperature of human coronary arteries in vivo, we developed a catheter-based technique that was applied in clinical practice.

Methods

Thermography Catheter

Design and Construction

A thermistor probe (Microchip NTC Thermistor, model 100K6 MCD368, BetaTHERM), 0.457 mm in diameter, was attached to the distal end of a long, 3F, nonthrombogenic polyurethane shaft. The gold-plated lead wires of the thermistor pass through the shaft and end in a connector at the distal part of the thermography catheter. At the distal 20 cm, the catheter has a second lumen for insertion of a guide wire; thus, the catheter can be inserted into the coronary artery over a standard guide wire (rapid exchange system; Figures 1 and 2). Opposite the thermistor is a hydrofoil specially designed to ensure contact of the thermistor on the vessel wall (thickness of the catheter at this site, 4F).

The technical characteristics of the polyamide thermistor include (1) temperature accuracy, 0.05°C; (2) time constant, 300 ms; (3) spatial resolution, 0.5 mm; and (4) linear correlation of resistance versus temperature over the range of 33°C to 43°C.

In Vitro Testing

Hydraulic in vitro testing with a special setup based on a glass coronary model and circulating heparinized donor whole blood proved that the bloodstream drives the thermistor against the wall (Figure 1). A Doppler-tip guide wire (FloWire Cardiometrics, Inc) and a catheter-tip micromanometer (Millar Instruments) were used to measure flow and pressure. Contact of the thermistor with the vessel wall was also verified by experimental testing (see below) and angiography in the first 10 patients of our series.
Experimental Testing

Twelve nonatherosclerotic pigs (either sex; weight, 15 to 25 kg) were premedicated, anesthetized, and mechanically ventilated as previously described. The investigation conforms with institutional guidelines. The thermography catheter was inserted through an 8F hockey-stick guiding catheter and positioned in the coronary arteries (6 left anterior descending, 3 left circumflex, and 3 right coronary arteries) under fluoroscopic control. Luminal surface temperature was measured at 10 different locations in each vessel. After measurements, 6 pigs were killed, and samples of transverse blocks of coronary segments were obtained and processed for light (hematoxylin and eosin and Masson's trichrome stain) and scanning electron microscopy as previously described. Contact of the device with the arterial wall was tested in the remaining 6 pigs. After temperature measurements in the left anterior descending artery, the pigs were thoracotomized with a midline sternotomy, and the pericardium was opened and suspended in a pericardial cradle. A 20-MHz ultrasound connector; GW, guide wire; and Th, thermistor.

Data Acquisition and Processing

Thermistor leads were connected to a Wheatstone bridge (a type of null comparator), which is used to correlate the change of thermistor resistance (which varies with temperature) to voltage changes. Subsequently, voltage changes were fed into a personal computer (200-MHz Intel Pentium) with a multichannel 12-bit analog-to-digital converter (Data Translation Inc) and displayed in real-time mode. Voltage changes were correlated with temperature values with commercially available software (Dataflow, Crystal Biotech). Calibration was made against beakers of water at temperatures varying from 33°C to 43°C (balancing the Wheatstone bridge to 0.00 V at 33°C).

Clinical Studies

Study Population

Ninety patients made up the study population. Forty-five patients were catheterized for investigation of valvular heart disease (30 patients) or chest pain (15 patients) and were found to have normal coronary arteries (control subjects). Fifteen patients suffered from stable angina (SA) and were catheterized for elective coronary angioplasty. Fifteen patients suffered from unstable angina (UA) and were selected for emergency angioplasty on the basis of angiographic findings after a 2-day hospitalization during which they were unresponsive to maximal medical treatment. The remainder suffered from acute myocardial infarction (AMI) and were catheterized for primary angioplasty within 6 hours after the onset of pain. In the UA and AMI patients, only the culprit lesion was studied by thermography.

None of the patients was under medication with corticosteroids or nonsteroid anti-inflammatory drugs, except for aspirin (Table 1). No patient had intercurrent inflammatory or neoplastic condition likely to be associated with an acute-phase response. The patients with normal coronary arteries and valvular heart disease were excluded from C-reactive protein analysis.

The study protocol was approved by the institutional ethical committee, and each patient provided written informed consent.

Lipid and Protein Measurements

Venous blood samples were obtained before catheterization. Lipid measurements were determined routinely when the blood samples were obtained. For protein measurements, coded plasma samples were stored at −70°C and analyzed in a single batch at the end of the study. C-reactive protein was assayed by immunonephelometry (Behring NA latex CRP mono, code No. OQIV210; sensitivity, 0.0175 mg/dL; upper limit of the reference interval for healthy nonpregnant adults, 0.5 mg/dL). Fibrinogen was determined by immunonephelometry with a BNA 100 analyzer.

Procedure

After cannulation of the coronary ostium with an 8F guiding catheter, mapping of the coronary vessel at the area of interest was made with a 20-MHz intravascular ultrasound (IVUS) catheter (Visions Five-64 F/XTM, Endosonics Corp). In the AMI patients, IVUS imaging was performed after patency restoration of the occluded artery.

Coronary Artery Disease Patients

The lesion of interest was outlined in ≥2 well-opacified views with biplane angiography. Quantitative angiographic measurements were obtained with electronic digital calipers (DCI-S, Automated Coronary Analysis, Philips). Thereafter, the thermography catheter was advanced through the guiding catheter, and blood temperature was measured when the thermistor had just emerged from the tip of the guiding catheter so that it would not be in contact with the vessel wall. Subsequently, temperature measurements at 5 locations over a length of ∼1 cm of normal (verified by IVUS) vessel wall near the lesion were made. The dominant (most frequent) temperature of these measurements was designated the background temperature. In addition, measurements at 5 different lesion sites (region of interest [ROI] for the atherosclerotic coronary arteries) were made, scanning the whole lesion both longitudinally and circumferentially (Figure 3). One measurement was made in the proximal part of the lesion, 1 at the distal, and 1 at the center. The other 2 measurements were made in areas between the center and the ends of the plaque. In the SA or UA patients in whom the thermography catheter could not cross the lesion, measurements scanning the whole lesion were
obtained again ~5 minutes after successful balloon angioplasty. In the AMI patients, measurements were obtained only ~5 minutes after angioplasty and verification by IVUS of mechanical lysis of thrombus. In all patients, measurements were obtained 5 minutes after any contrast infusion. For coronary angioplasty, the balloon was dilated with a mixture of contrast medium and normal saline at 37°C.

**Control Subjects**

Absence of atherosclerosis was verified by IVUS. After blood temperature measurement, 5 wall temperature measurements in a region 1 cm long were obtained. This region was designated the control region and its dominant temperature background temperature. Subsequently, 5 temperature measurements were obtained in another region of the same length (randomly selected distally or proximally to the first lesion), which was designated the ROI. For control subjects, the absolute values of the differences between ROI and background temperature were taken for analysis.

Simultaneous with vessel wall measurements, mouth temperature was measured with a separate thermistor with the same specifications as the thermistor of the thermography catheter. All angiograms were examined by investigators who were unaware of the results of temperature measurements.

**Statistical Analysis**

Data are expressed as mean±SD. Variables were tested for normal distribution with the Kolmogorov-Smirnov 1-sample test. Because of nonnormal distributions, to detect significant differences regarding the difference between ROI temperature and background temperature (all 5 differences in ROI measurements from background temperature in each subject were taken into account in the analysis), the difference between maximum ROI temperature and background temperature, C-reactive protein, and fibrinogen between all groups, the Kruskal-Wallis 1-way ANOVA was used. Pairwise testing for significant differences between the groups regarding these variables was performed with Dunn’s test for multiple comparisons. To detect significant differences between the groups regarding age, total cholesterol, and ratio of total cholesterol to HDL cholesterol, 1-way ANOVA was used. Pairwise testing for significant differences between the groups regarding these variables was performed with an independent-samples t test. Frequency of the peak difference from background temperature regarding its distribution in the 5 sites of measurement in the SA, UA, and AMI patients was performed with the χ² goodness-of-fit test. Bivariate correlation coefficients were calculated with Pearson’s product-moment method (continuous versus continuous variables) or the Spearman’s rank method (continuous versus discrete variables) when appropriate. Heterogeneity within the ROI was defined as the presence of ≥1 measurement outside the range; mean of the 5 differences from background temperature ±0.1°C. Differences in the heterogeneity within the plaque between SA, UA, and AMI groups were tested with the χ² test. Pairwise testing between these 3 groups of patients regarding heterogeneity was done with Fisher’s exact test. The independent relations of the difference between maximum ROI temperature and background temperature to its potential predictors (age, C-reactive protein, fibrinogen, total cholesterol, ratio of total cholesterol to HDL cholesterol, time of day [day divided into 3 periods: midnight to 8 AM, 8 AM to 4 PM, and 4 PM to midnight], and time elapsed from onset of symptoms to temperature measurement [AMI patients only]).

**TABLE 1. Demographic, Treatment, and Biochemical Characteristics in the 4 Study Groups**

<table>
<thead>
<tr>
<th>Characteristic</th>
<th>Control Subjects</th>
<th>SA</th>
<th>UA</th>
<th>AMI</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age, y</td>
<td>60±11</td>
<td>60±12</td>
<td>62±11</td>
<td>63±14</td>
</tr>
<tr>
<td>Aspirin intake (Y/N), n</td>
<td>0/45</td>
<td>15/0</td>
<td>15/0</td>
<td>8/7</td>
</tr>
<tr>
<td>Total Ch, mg/dL</td>
<td>179.8±27.9</td>
<td>211.7±11.8*</td>
<td>217.9±22.2*</td>
<td>222.7±31.9*</td>
</tr>
<tr>
<td>Total Ch/HDL Ch, mg/dL</td>
<td>3.87±0.79</td>
<td>4.64±0.91‡</td>
<td>4.77±1.17§</td>
<td>4.96±1.26</td>
</tr>
<tr>
<td>C-Reactive protein, mg/dL</td>
<td>0.10±0.08</td>
<td>0.28±0.12†</td>
<td>2.03±2.16†</td>
<td>2.56±2.48†</td>
</tr>
<tr>
<td>Fibrinogen, mg/dL</td>
<td>271.9±63.8</td>
<td>290.3±51.1</td>
<td>301.3±65.3</td>
<td>303.6±63.3</td>
</tr>
</tbody>
</table>

Ch indicates cholesterol.

*P<0.001 control vs SA, UA, or AMI; †P<0.01 control vs SA, UA, AMI; ‡P<0.005 control vs SA; §P<0.05 control vs UA; ‖P<0.01 control vs AMI.

Figure 3. Left anterior oblique projections of SA patient. A, Arrowheads indicate stenosis at distal segment of right coronary artery. B, Thermistor (Th) is positioned at site of lesion (distal marker indicates catheter tip).
were analyzed with stepwise multiple regression analysis. To detect differences in the differences between maximum lesion temperature and background temperature values before and after angioplasty, multiple linear regression analysis was performed with the use of the intervention (angioplasty) and 29 dummy variables as independent variables to adjust for intersubject variability. Values of $P<0.05$ were considered statistically significant. Analyses were performed with SPSS for Windows (version 8.0) statistical software.

Results

In Vitro and Experimental Testing

In in vitro testing, the thermistor was not in contact with the glass wall at resting conditions, but when blood was infused, it was driven against the wall and contacted its internal surface at any combination of flow velocity (range, 10 to 50 cm/s) and driving pressure (range 50 to 140 mm Hg).

In all experimental procedures, the catheter could be inserted and positioned without difficulties or complications. In gross inspection after each procedure, no thrombi were observed on the catheter. No embolic events were observed in any of the experimental pigs. Scanning electron and light microscopy disclosed no endothelial denudation, no thrombus formation, and no internal elastic lamina or deep wall damage.

Coronary wall temperatures in each pig were constant, varying by only 0.05°C. The SD of the 10 measurements for each pig ranged from 0°C to 0.0258°C.

In 4 of the 6 pigs in which the contact of the thermistor on the artery wall was tested, the thermistor was close to but not in direct contact with the intima during ligation of the coronary artery origin; during unobstructed flow, however, the thermistor was in direct contact with arterial intima. In 2 cases, the thermistor was in contact both during ligation and unobstructed flow.

Clinical Studies

Age and fibrinogen did not differ among the 4 groups. Total cholesterol, ratio of total cholesterol to HDL cholesterol, and C-reactive protein were different in the 4 groups ($P<0.001$ for all). For pairwise comparisons, see Table 1.

Surface wall temperature was measured in 90 ROIs, 1 in each patient. Two left main coronary arteries, 37 left anterior descending arteries, 18 left circumflex arteries, and 33 right coronary arteries were studied. In the first 10 patients of our series in whom contact of the thermistor on the artery was tested, frame-by-frame analysis in 2 biplane views during washout of the contrast medium revealed that the radio-opaque thermistor was in contact with the edge of the vessel. The temperature of healthy vessel wall was 0.36±0.11°C higher than mouth temperature. Mean coronary artery stenosis was 83±8% for the SA patients and 81±7% for the UA patients ($P=NS$).

The 5 measurements obtained for determination of background temperature were constant in each subject of the total study population, varying by only 0.05°C (SD for each of the subjects ranged from 0 to 0.0263). Temperature of blood and healthy vessel wall did not differ ($P=NS$; in 78 patients, temperature was identical; in the remainder, it ranged within the accuracy of the thermistor [in 7 patients, blood temperature was higher by 0.05°C; in 5, blood temperature was lower by 0.05°C]). Coronary wall temperatures in the ROI of each control subject were constant, varying by only 0.05°C. SDs of ROI measurements for each control subject ranged from 0°C to 0.0274°C. In the SA or UA patients, there was no difference in the differences between maximum and background temperatures before and after angioplasty (0.470±0.418°C versus 0.458±0.415°C, respectively; $P=NS$). Most atherosclerotic plaques showed higher surface temperatures compared with normal vessel wall (Figure 4). The difference between maximum plaque and background temperatures did not correlate with coronary artery stenosis in both SA and UA patients ($r=−0.10$ and $−0.09$, respectively; $P=NS$ for both). Greater values in the differences between maximum plaque and background temperatures were observed in UA (maximum, 1.55°C) and in AMI (maximum, 2.60°C) patients. Differences between ROI and background temperatures and between maximum ROI and background temperatures were different among the 4 groups, increasing progressively from SA to AMI patients ($P<0.001$ for both parameters; for pairwise comparisons, see Table 2). Mean temperature differences between ROI and background temperatures in each group were 0.004±0.009°C in the control subjects, 0.106±0.110°C in SA patients, 0.683±0.347°C in UA patients, and 1.472±0.691°C in AMI patients; mean temperature differences between maximum ROI and background temperatures were 0.010±0.020°C in normal subjects, 0.153±0.134°C in SA patients, 0.787±0.360°C in UA patients, and 1.593±0.704°C in AMI patients (Figure 5).

There was no statistical difference in the frequency of maximum plaque difference from background temperature in terms of its distribution in the 5 sites of measurement in the coronary artery disease patients.

Heterogeneity within the ROI was shown in 20%, 40%, and 67% of the patients with SA, UA, and AMI, respectively, whereas no heterogeneity was shown in the control subjects.

Heterogeneity within the plaque between SA, UA, and AMI patients was different ($P<0.05$), and pairwise comparisons revealed significant differences in heterogeneity between AMI and SA patients ($P<0.05$).

Multiple regression analysis revealed that C-reactive protein was the only factor significantly associated with the differences between maximum ROI temperature and background temperature values ($F=70.2$, multiple $r^2=0.55$, $B=0.28$, $P<0.001$; Figure 6).

Aspirin intake did not correlate with the difference between maximum ROI and background temperature values in the AMI group ($r=−0.37$, $P=NS$).

Discussion

Plaque Rupture

Atherosclerotic plaque rupture is the main mechanism of acute ischemic syndromes, but despite improvements in established diagnostic techniques and the advent of new imaging modalities, neither plaque rupture nor plaque erosion can be predicted reliably in clinical practice. Inflammation at the immediate site of plaque rupture plays an important role in the destabilization of plaque because macrophages release enzymes that digest extracellular matrix and
weaken the overlying fibrous cap. Furthermore, recent evidence suggests a possible role for infection in the pathogenesis of atherosclerosis. In addition, relative blood viscosity rises and aggregation of red cells increases with increases in temperature.

**Thermography of Arteries**

Previous ex vivo studies by Casscells et al introduced the concept that detection of heat released by activated inflammatory cells of atherosclerotic plaques may predict plaque rupture and thrombosis. Consistent with the development of other cardiac and vascular catheters, we have developed a catheter-based technique for the temperature measurement of human arteries in vivo and demonstrated that there is thermal heterogeneity within human atherosclerotic coronary arteries. This heterogeneity is larger in UA and AMI patients, implying that it may be related to the pathogenesis of these syndromes.

The heterogeneity within the plaque that we observed in SA, UA, and AMI patients is in accordance with previous reports. Although detection of heterogeneity was not the main target of the study and optimally more measurements within the lesion should have been obtained, this finding is in accordance with the involvement of a localized process, such as clustering of mononuclear infiltrates, in the pathogenesis of plaque rupture.

Our technique or similar techniques, such as infrared thermography (which may provide additional information about the microstructure of the plaque), enhance the diagnostic approach for the detection of plaques that are prone to

**TABLE 2. Comparison of Temperature Differences Between Groups**

<table>
<thead>
<tr>
<th></th>
<th>Control Subjects</th>
<th>SA</th>
<th>UA</th>
<th>AMI</th>
</tr>
</thead>
<tbody>
<tr>
<td>Control subjects</td>
<td>…</td>
<td>&lt;0.05†</td>
<td>&lt;0.01†</td>
<td>&lt;0.01†</td>
</tr>
<tr>
<td>SA patients</td>
<td>&lt;0.01*</td>
<td>…</td>
<td>&lt;0.05†</td>
<td>&lt;0.01†</td>
</tr>
<tr>
<td>UA patients</td>
<td>&lt;0.001*</td>
<td>&lt;0.01*</td>
<td>…</td>
<td>NS†</td>
</tr>
<tr>
<td>AMI patients</td>
<td>&lt;0.001*</td>
<td>&lt;0.01*</td>
<td>&lt;0.05*</td>
<td>…</td>
</tr>
</tbody>
</table>

*Probability values indicate comparisons regarding differences between ROI and background temperatures; †Probability values indicate comparisons regarding differences between maximum ROI and background temperatures.

**Figure 4.** Individual temperature wall differences from background temperature in 4 groups. S1 through S5 indicate sites of measurement from proximal (S1) to distal (S5) parts of ROI.

**Figure 5.** Differences in maximum ROI temperature from background temperature in 4 study groups. Temperature differences increase progressively from SA to AMI patients.
rupture and may prove useful in evaluating existing or future treatment modalities, thus intensifying effectiveness in avoiding potentially life-threatening plaque rupture. Similarly, thermal detection of atherosclerotic plaques may be useful in predicting inflamed lesions at high risk for restenosis after interventional procedures. Detection of temperature heterogeneity may also provide useful information in other cardiovascular conditions, such as myocarditis, valvulitis, aortitis, or even arrhythmogenic foci. Furthermore, the concept of thermal heterogeneity may be applied by other specialties with the detection of inflamed or malignant cells in other organs.

Specific Comments
Ideally, temperature should have been measured just before the acute event. Indeed, temperature changes might result from the structural changes of the plaque, such as fissuring or fracturing, because the temperature might be different within the arterial wall. Moreover, a possible incomplete or intermittent contact of the thermistor with the vessel wall might contribute to the temperature differences observed. In addition, altered flow patterns (decreased flow may lead to decreased heat transfer with the bloodstream and consequent local increase in temperature at the lesion), the formation of thrombus, and the possibility that the inflammatory response (which leads to the increased temperature at the site of the lesion) is caused by the rupture of the plaque rather than being a process preceding the rupture of the plaque may also be considered confounding factors. However, if temperature increase is merely a secondary phenomenon and not a finding related to the process that leads to plaque rupture, then the SA patients would be expected to have no temperature difference compared with control subjects. On the contrary, the SA patients exhibited increased plaque temperature, implying that the process that leads to increased temperature is under- way and precedes the occurrence of acute syndromes. In cases of incomplete or intermittent contact of the thermistor with the vessel wall because of the structure of the plaque, one might expect equal temperature differences in SA, UA, and AMI patients because all these syndromes have approximately the same degree of atherosclerosis (and consequently anatomic irregularity), as indicated by previous studies and the similar percent stenosis of these patient groups in the present study. On the contrary, we observed a progressive temperature increase from SA to AMI patients. Moreover, even if the whole surface of the thermistor was not in direct contact with a hard, irregular, stable plaque, then the thermistor would underestimate temperature differences because of either cooling by the bloodstream or averaging of very small areas of higher temperature with areas of lower temperature. In that case, the actual temperature differences would be ever greater, thus reinforcing our findings. In addition, the effectiveness of the thermography catheter in measuring vessel wall temperature reliably is emphasized by the results that show no differences between blood and healthy wall and differences between blood and certain areas of the inflamed vessel. Regarding the possible temperature released by thrombus formation and its contribution to the findings of our study in the AMI group, it should be noted that in all these patients, measurements were obtained after angioplasty and verification by IVUS of thrombus lysis. In addition, as regards the effect of flow patterns, we should stress that no differences in temperature were observed before and after successful angioplasty. Moreover, as demonstrated, this lack of difference before and after angioplasty cannot be attributed to intersubject variability. Thus, temperature increase in the lesion is not likely to result from structural variation after angioplasty. In addition, the positive correlation between levels of C-reactive protein (a sensitive indicator of inflammation increasing in acute ischemic syndromes) and increased lesion temperature supports the notion that an inflammatory process is underlying heat release. Therefore, although our results cannot be directly extrapolated to unstable plaques before rupture, they are supportive of the concept that increased temperature precedes plaque rupture. Nevertheless, larger prospective studies are required.

An interesting point is that the AMI patients were not significantly different from the UA patients in terms of total cholesterol, ratio of total cholesterol to HDL cholesterol, C-reactive protein, fibrinogen, and age, indicating that these factors do not account for the unequal temperature differences observed in these 2 groups.

The temperature differences we found in our study were not as high would be expected according to previous studies. Several reasons can explain this discrepancy. First, the presence of heat-delivering blood in vivo may act as a buffering pool that tends to decrease heat differences. Second, the original measurements in these studies were made 10 to 15 minutes after removal of the samples at room temperature. Therefore, areas with fewer heat-producing cells may have cooled faster than monocyte-populated areas that continued, even for a short period after removal, to release heat, thus magnifying an in vivo difference. A finding supportive of this explanation is the higher temperature differences observed in a hypothermic patient suffering from cardiogenic shock secondary to AMI.

There is a theoretical possibility that a hard plaque containing relatively large amounts of collagen and calcium would have a lower temperature than the metabolically active normal wall. However, this was not the case in any of our SA patients, rendering this possibility unlikely.
Flow through the rich vasa vasorum network in the atherosclerotic plaque in vivo might affect the temperature. However, a positive correlation exists between angiogenesis and inflammation, and both are considered to predispose to plaque rupture. Therefore, the concept of higher temperature indicating a higher risk of rupture is reinforced.

The negative correlation between the intake of aspirin before the event and lesion temperature in the AMI group was not statistically significant. This may be due to the small number of patients participating in the study. Future, specially designed studies are needed to clarify the role of aspirin or other anti-inflammatory drugs in the process of heat release.

Conclusions
Thermal heterogeneity exists within the coronary arteries of coronary artery disease patients, whereas temperature is constant within normal coronary arteries. This heterogeneity increases progressively from SA to UA and then to AMI patients, thus supporting the involvement of temperature heterogeneity in the natural history of these syndromes. This technique or other techniques that can localize heat in clinical practice may prove useful in identifying plaques that are prone to rupture or thrombosis.

Acknowledgments
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