eling (lesion EEM CSA more than 5% greater than at the proximal reference segment) and negative remodeling (lesion EEM CSA more than 5% less than at the distal reference segment) also were evaluated. Insulin resistance was determined by homeostasis model assessment (HOMA) and defined as values above the 75th percentile (i.e. 1.71). Patients were divided into two groups: group 1: n=13, patients with ACS; group 2: n=62, patients with stable angina. Clinical and IVUS variables with a p value less than 0.1 in univariate analysis were entered into the multivariate models.
Results: The data was as follows.

The Significant Predictos for ACS

	ACS	Stable angina	Univariate p value	Multivariate p value
No. of segments	n=13	n=72		
Positive remodeling	6 (46%)	13 (18%)	0.03	0.1
Hypertension	6 (46%)	54 (75%)	0.04	0.01
Insulin resistance	10 (77%)	19 (26%)	0.0004	0.002

Conclusion: Positive remodeling, absent of hypertension and insulin resistance were the predictors for ACS. However, multivariate logistic regression analysis showed insulin resistance was the most powerful predictor.

1054-16 Distribution of Vulnerable Plaque in the Human Coronary Artery: Angiographic and Intravascular Ultrasound Analysis of Occult Atherosclerosis in Proximal Sites in the Three Major Coronary Arteries

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Objective: To use intravascular ultrasound (IVUS) to examine the distribution of vulnerable atherosclerotic plaque (VP) in human coronary arteries and clarify the morphological features of occult atherosclerosis in these sites that may develop into VP.
Methods and Results: In 307 consecutive acute coronary syndrome cases (228 acute myocardial infarction, 79 unstable angina), culprit lesion distribution, under coronary angiography, was found to be RCA 94, LAD 179, and LCx 34. We then assessed the location of these lesions to evaluate the distribution of VP. In the RCA and LAD, VPs were most frequently observed at proximal sites in the RCA, 39 lesions (41.5%) and LAD, 117 lesions (65.4%). In the LCx, VPs were more frequently observed at mid-site (20, 59.9%) than at proximal site. To assess evidence of which occult atherosclerosis might develop into VPs in these sites, we also used IVUS to examine proximal sites with no angiographic stenosis (%DS<25%) during 78 elective coronary interventions to mid or distal target lesions in each vessel. The morphological features of occult atherosclerosis in proximal sites in the three major coronary arteries were shown below (table).
Conclusion: Vulnerable plaque tends to develop at proximal sites in the RCA and LAD. Significant occult atherosclerotic lesions, associated with calcification and echolucence, may be more likely to develop in the proximal RCA and LAD than in the proximal LCx.

The morphological features of occult atherosclerosis in proximal sites of coronary arteries

	%Plaque Area	Plaque Eccentricity	Calcification	Echolucent Area
RCA (n=26)	61.3±10.1%	15/26(57.7%)	20/26(76.9%)	8/26(30.8%)
LAD (n=35)	52.1±14.5%	27/35(77.1%)	29/35(85.7%)	9/35(25.7%)
LCx (n=17)	33.1±14.6%	5/17(29.4%)	6/17(35.3%)	0/17(0%)

1054-17 Treatment With Statins Results in Decreased Heat Production From the Culprit Atherosclerotic Lesions in Patients With Stable and Unstable Angina

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Background: Previous ex vivo and in vivo studies have shown, that inflammation leads to heat production from atherosclerotic lesions. Administration of statins may further stabilize atherosclerotic plaques, due to an additional anti-inflammatory effect. The aim of our study was to investigate the effect of statins on the inflammatory process of atherosclerotic plaques by measuring the temperature of atherosclerotic plaques. **Methods:** In the study we included 62 patients (pts), 36 pts with unstable angina (UA) and 26 pts with stable angina (SA) who were hospitalized and underwent diagnostic catheterization. Balloon angioplasty was on physician's decision. Statins were administered for a period of over a month to 32 pts (18 pts with UA and 14 pts with SA). Thirty pts were not under statin treatment (18 pts with UA and 12 pts with SA). Forty-eight pts were also under aspirin treatment. Total cholesterol and low-density cholesterol were measured in all pts. During the catheterization a thermography catheter (Medispes S.W.A.G.zug-Switzerland) was used, in order to measure the temperature difference (TD) between the atherosclerotic plaque and the healthy vessel wall. **Results:** TD was higher in pts with UA compared to pts with SA (0.41 ± 0.28 vs. 0.32 ± 0.1 °C, $p < 0.04$). When we categorized the study population into pts treated with statins and pts not treated with statins, TD was greater in the untreated group (0.44 ± 0.26 vs 0.25 ± 0.23 °C, $p < 0.01$). Moreover, treated pts within each clinical syndrome had lower TD compared to untreated pts (UA: 0.29 ± 0.25 vs. 0.45 ± 0.26 °C, $p < 0.02$ and SA: 0.23 ± 0.16 vs. 0.42 ± 0.24 °C, $p < 0.03$). Multivariate analysis showed that treatment with statins was an independent factor in the assessment of temperature variation, adjusted for age, hypercholesterolemia, hypertension, smoking, aspirin intake, and clinical syndrome. **Conclusions:** Pts treated with statins have less heat production from the culprit lesion. This may be an additional anti-inflammatory effect of statins on the atherosclerotic plaque, leading to plaque stabilization.

1054-18 Volumetric Intravascular Ultrasound Quantification of the Amount of Atherosclerosis in Nonstenotic Arterial Segments

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Background: Long-term patient outcome depends on the extent and severity of atherosclerosis. The overall plaque burden rather than the number of most severe lesions may have a stronger relation to potentially adverse coronary outcomes. We used intravascular ultrasound (IVUS) to quantify the overall plaque burden in patients with de novo focal stenoses. Methods: Nineteen patients with focal stenoses in the right coronary artery and complete pre-interventional IVUS imaging from crux to ostia were studied. Measurements of the external elastic membrane (EEM) cross sectional area (CSA), lumen CSA, and plaque&media (EEM minus lumen) CSA, and arcs of calcium were obtained using computerized planimetry; and volumes were calculated. Results: Significantly more atherosclerotic plaque ($p<0.0001$) was found in the nonstenotic segments, ranging from 46% to 86% (mean = 72±12%) of the total plaque volume. There was no difference when positively remodeled stenoses (EEM CSA >mean reference) were compared to intermediately/negatively remodeled stenoses (EEM CSA ≤mean reference). The percentage of the plaque contained in the nonstenotic segments correlated inversely with lesion length ($r=0.52$, $p=0.0234$), and weakly, but directly with nonstenotic segment length ($r=0.35$, $p=0.15$). Conclusions: In arteries with single, focal stenosis, 3/4 of the atherosclerotic plaque burden is contained in the non-stenotic segments.

IVUS Measurements

	Total	Lesion	Non-Stenotic Segment	p-value (Lesion vs. Reference)
EEM,mm3	578±345	119±111	459±283	<0.0001
Lumen,mm3	257±165	29±30	228±156	<0.0001
Plaque&media,mm3	321±198	90±86	231±140	<0.0001
Length,mm	42.8±17.7	9.4±7.6	33.4±13.5	<0.0001

1054-19 Progress With the Calibration of a 3F Near Infrared Spectroscopy Fiber Optic Catheter for Monitoring the PH of Atherosclerotic Plaque: Introducing a Novel Approach for Detection of Active Vulnerable Plaque

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We hypothesized that localization of vulnerable plaque can be enhanced by physiological factors such as low pH, high temperature (T), NO, hypoxia, and oxyradicals, which have been shown to shift NIR spectra. We have previously shown that inflamed regions of plaque are lower in pH. Therefore, we chose pH to calibrate our spectroscopy catheter.
Methods: Different probe sizes were used to study variable penetration depth. Eventually, using a unique miniaturized fiber optic side-viewing catheter, we demonstrated the feasibility of performing the correlation. 10 human carotid endarterectomized plaques were collected and placed immediately in a humidified, 37°C controlled T glove-box type incubator. Optical reflectance spectra (400 - 1100 nm) were taken with the prototype catheter connected to a spectrometer. Partial Least Squares multivariate calibration techniques were used for the correlation. Results: The R2 of the determination of tissue pH from the optical NIR calibration was 0.63 and the Root Mean Squared Deviation (RMSD) was 0.14 pH units.
Conclusion: This feasibility study suggests that plaque pH can be determined with NIR spectroscopy in both ex vivo and in vivo plaque tissues. Further improvements in signal-to-noise ratio will be required to meet the long-term goal of detection of vulnerable plaques based on pH. Oue new multi-probe catheter allows multiple radial reading of NIR spectra of the vessel wall. This enabels pH spectrographic imaging of vulnerable plaque.