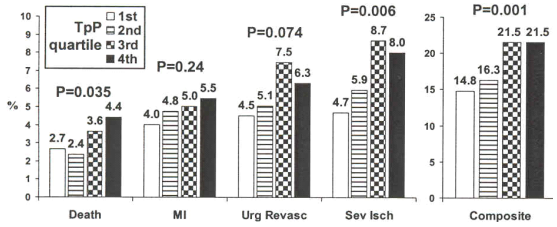
through 10 months.
Results: Median TpP levels were 8.9 ug/ml (IQR 4.8-15.9). Higher baseline levels of TpP were associated with older age, hypertension, diabetes, prior CAD, and prior CHF (p<0.05 for each). TpP levels correlated only weakly with troponin I (TnI), C-reactive protein (CRP), and B-type natriuretic peptide (BNP) (|r|<0.1 for each). The risk of death, MI, Urg Revasc, Sev Isch, and the composite increased progressively with rising levels of TpP (Fig). After adjusting for baseline differences, including TnI, CRP, and BNP, patients with TpP levels above the median remained at significantly increased risk for the composite endpoint (hazard ratio 1.47, p=0.001).
Conclusion: In patients with ACS, elevated levels of TpP at baseline are associated with an increased risk of death and ischemic complications. These data support the value of combining a marker of active thrombosis, such as TpP, with established biomarkers of necrosis, inflammation, and hemodynamic stress for risk assessment in ACS.



11:30 a.m.

884-5 **Impact of Troponin Status and Coronary Artery Disease by Angiography on Outcome in Acute Coronary Syndrome: A TACTICS TIMI-18 Substudy**

Manu Pillai, Peter DiBattiste, Sabina Murphy, David Morrow, Christopher Cannon, Nasser M. Lakkis, Baylor College of Medicine, Houston, TX, Brigham and Women's Hospital, Boston, MA

Background: Cardiac troponin (Tn) T and I elevations are powerful independent prognostic indicators in the setting of acute coronary syndrome (ACS). However, in clinical practice, seemingly "false positive" Tn's are common . ***Methods and Results***: To evaluate whether such patients are "false positives" and if there is clinical significance of an elevated Tn, we evaluated the prognosis of ACS patients enrolled in TACTICS-TIMI-18 study according to the presence or absence of CAD on angiography and Tn status on admission. In this study 1,372 patients underwent angiography. A subset of 455 patients were Tn negative, and of those, 362 (79.6%) had significant CAD (>50%stenosis in one or more vessel). Fifty-seven out of 917 patients (6.2%) with + TnT or I did not have significant CAD. The mean value of TnT in patients with angiographic CAD was 0.603 ng/ml compared to 0.281 ng/ml in those without CAD (p=0.01). The mean value of TnI was 9.39 ng/ml in patients with CAD compared to 3.32 ng/ml (in patients without CAD (p=0.005). An outcome of death or reinfarction at 6 months is shown in table.
o:p>
Conclusions: Elevated troponin in ACS confers a significantly high risk for death or MI, even among patients who are found to not have significant CAD. The mechanism of this adverse outcome deserves further study, but these data suggest that all patients presenting with a clinical history of ACS and a positive troponin at presentation deserve early *and long-term* aggressive preventive management.

	Tn neg, no CAD n=93	Tn pos no CAD n=57	Tn neg CAD n=362	Tn pos CAD n=860	p-value
Death	0%	1.75%	2.21%	4.07%	0.082
MI	0%	3.51%	4.42%	8.26%	0.003
death or MI	0%	5.26%	5.52%	11.16%	<0.001

11:45 a.m.

884-6 **Incidence of Vulnerable Plaques in Humans: Assessment With 3-D Intravascular Palpography**

Johannes A. Schaar, Evelyn Regar, Francesco Saia, Chris L. de Korte, Frits Mastik, Anton F. van der Steen, Patrick W. Serruys, Erasmus Medical Center, Rotterdam, The Netherlands, Interuniversity Cardiology Institute of the Netherlands, Utrecht, The Netherlands

Intravascular palpography (IP) can detect deformable thin-cap fibroatheroma with a high sensitivity and specificity by measuring the strain of the plaque surface. These vulnerable plaques show a typical high strain pattern. We hypothesized that it is possible to detect vulnerable plaques in-vivo in human coronary arteries by scanning the coronary artery for typical high strain patterns.
Method: IP was performed in 56 patients by using standard 20 MHz intravascular ultrasound catheters (Avanar, JOMED Inc). Analysis included the complete length of the vessel. The population was divided into 3 groups: Patients with [1] antecedent (<24h) myocardial infarction. (post MI) [2] unstable angina (ST depression, Troponin negative); (unstable). [3] Stable angina (stable). Per patient one coronary artery was investigated.
Results: IP was successful performed in all patients. The number of typical strain patterns reflecting the number of vulnerable plaques per coronary artery are given in the table. There was a significant difference between the stable vs. unstable group (p<0.001) and stable vs. post MI group (p<0.0001). No difference was seen between the unstable group and post MI group (p<0.056).

	Stable	Unstable	Post MI
n (patients)	19	19	18
High strain spots	0.7 ± 0.5	1.7 ± 0.4	2.0 ± 0.8

Conclusion:
IP can be used to assess the number vulnerable plaques in humans. This pilot study revealed a clear association between clinical presentation and the amount of vulnerable plaques. Additional validation has to be performed to assess the predictive value of the technique to identify vulnerable patients.

ORAL CONTRIBUTIONS

891

Enhanced Extracorporeal Counterpulsation: Mechanisms and Clinical Results

Wednesday, March 10, 2004, 10:30 a.m.-Noon
Morial Convention Center, Room 257

10:30 a.m.

891-1 **Enhanced External Counterpulsation Decreases Wave Reflection Amplitude and Reduces Left Ventricular Afterload and Systolic Stress in Patients With Refractory Angina**

Wilmer W. Nichols, Randy W. Braith, Rahul Aggarwal, C. R. Conti, University of Florida, Gainesville, FL

Background: Myocardial ischemia is associated with increased arterial stiffness which causes early return of reflected pressure waves from the lower body and augments aortic systolic and pulse pressures and increases left ventricular afterload and myocardial oxygen demand. The aim of this study was to determine if arterial properties and wave reflection characteristics are altered after EECP treatment in patients with chronic stable angina resistant to anti-anginal medication. Methods: High-fidelity radial artery pressure waveforms were recorded non-invasively by applanation tonometry and ascending aortic pressure waveforms generated using a mathematical transfer function. Twenty patients (age 61±7.3 years) with refractory angina taking two or more anti-anginal drugs were studied. Data were collected before and after 34 one-hour EECP sessions. Augmentation index (AIa, index of aortic stiffness) and amplitude and timing of the reflected pressure wave (index of myocardial oxygen demand) were calculated from the generated aortic pressure waveform. Results: EECP caused a significant decline in AIa from 27±10 to 19±9.8% (p<0.02) and a delay in reflected wave travel time from 68±8.3 to 74±6.8 msec (p<0.01). These modifications in wave reflection characteristics were associated with a decline in reflected wave amplitude (from 13±7.2 to 9.0±7.0 mm Hg, p<0.01) and duration (from 189±34 to 174±40 msec, p<0.01) which caused a decrease in aortic systolic (from 120±18 to 108±18 mm Hg, p<0.01) and pulse (from 48±15 to 41±16 mm Hg,p<0.01) pressure. The average number of anginal episodes per week decreased from 7.1±6.0 to 1.1±1.2 (p<0.02) while heart rate, body weight and drug regimen did not change during the course of the study. Conclusions: EECP treatment delays return of the reflected pressure wave from the periphery to the heart, reduces its amplitude and, therefore, decreases aortic blood pressure and systolic stress in patients with refractory angina taking anti-anginal medication. These changes, which are likely due to decreased arterial stiffness and pulse wave velocity of the muscular arteries in the legs, reduce myocardial oxygen demand and decrease the number of anginal episodes.

10:45 a.m.

891-2 **External Counterpulsation Therapy: Significant Clinical Improvement Without Electrophysiologic Remodeling**

Charles A. Henrikson, Nisha Chandra-Strobos, Johns Hopkins Medical Institutions, Baltimore, MD

Background: External counterpulsation therapy (ECP) non-invasively improves coronary flow, hemodynamics, and time to ischemia in patients with severe coronary artery disease (CAD). Other treatments with similar effects, e.g. left ventricular assist devices, promote electrophysiologic remodeling, with a narrowing of the QRS complex.
Methods: We studied 28 patients, who completed a 7 week 35 hour session of ECP, to assess whether such therapy would also result in electrophysiological remodeling, especially if associated with clinical improvement. All patients had class II-III angina, imaging-proven ischemia, and severe CAD.
Results: Of 28 patients, with mean age 62+/-13 years (mean +/-SD), 78% were male, 46% diabetic, 82% hypertensive, 60% had undergone angioplasty, and 67% bypass surgery. The mean ejection fraction was 44% (range 25-60%). Following ECP, most patients (82%) had at least a one full class improvement in their anginal pattern. In most patients, there was substantial baseline conduction system disease present: a mean QRS of 105 +/- 19 msec. Of note, there was no significant change in heart rate, PR, QRS, or QTc intervals before and after ECP in either clinical responders or non-responders. When