940-60

Discrimination Between Soft Plaque and Thrombus Based on Radiofrequency Analysis of Intravascular Ultrasound

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Prior work has shown that ultrasonic backscatter from soft tissues generally behaves as a Rayleigh scattering process for frequencies from 1–15 MHz. One necessary characteristic for Rayleigh scattering is that the ratio of the mean to standard deviation (MSR) of the radiofrequency (RF) signal envelope is 1.91. We tested if backscatter from an intravascular system (IVUS) that operates at 30 MHz also shows Rayleigh characteristics and if dicrimination between soft plaque and thrombus is possible.

Five coagulated canine blood samples and seven human coronary arteries with soft plaque accumulation were imaged with the 30 MHz IVUS system. RF signals from 82 vectors from all tissue samples were digitized at 500 MHz (in 8 bit resolution). For each vector, a region of interest was defined within the tissue sample and the RF envelope probability distribution function generated to calculate the MSR. In addition, the variance of the RF signal was computed and normalized with a perfect reflector as an index for average backscatter power (ABP).

Soft Plaque		Thrombus	
MSR	1.90 ± 0.34	p = ns	1.93 ± 0.38
ABP	-31.28 ± 9.7dB	p < 0.0001	~16.4 ± 4.7dB

Conclusion: These results suggest: 1) The RF envelopes for thrombus and soft plaque are consistent with a Rayleigh scattering process at 30 MHz. 2) The MSR is not different for the two tissue groups. 3) However, the ABP for thrombus is significantly (\approx six fold) higher than for soft plaque allowing discrimination between these tissue types.

941

Methods to Alter Ventricular Function in Coronary Artery Disease

Tuesday, March 26, 1996, 9:00 a.m.—11:00 a.m. Orange County Convention Center, Hali E Presentation Hour: 10:00 a.m.—11:00 a.m.

941-1

Angiotensin Converting Enzyme Inhibitor Therapy Promotes Remodeling in Patients With Left Ventricular Ejection Fraction > 40% After Acute Transmural Myocardial Infarction

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ACE inhibitors (I) diminish LV volume (V) increases in patients (pts) with LVEF < 40% after acute myocardial infarction (MI). Whether ACEI affects LVVs and mass in pts with LVEF > 40% after their first acute transmural MI is unknown. Accordingly, 30 pts with LVEF > 40% after their first acute transmural MI were randomized to: oral dose ramipril (R) (5-20 mg qd) (n = 17) vs. conventional therapy with beta-blockers and nitrates without R (no R)(n = 13). Magnetic resonance imaging 5-10 days after MI and at 3 months provided LVEF, LVVs and LV mass from summated short axis slices. There was no change in LVEDV (124 \pm 33 to 132 \pm 36 ml) and LVEF (51 \pm 9 to 52 \pm 10%) in the R pts as well as in the no R pts (122 \pm 33 to 136 \pm 30 ml and 51 \pm 6 to 55 \pm 8%). However, there was a significant decrease in LV mass in the R pts (163 \pm 35 to 145 \pm 32 gm, p < 0.0005) but not in the no R pts (154 \pm 32 to 151 \pm 42 gm). Thus, LV mass/EDV ratio decreased in the R pts $(1.36 \pm 0.2 \text{ to } 1.14 \pm 0.13, p = 0.006)$ but not in the no R pts $(1.33 \pm 0.33 \text{ to})$ 1.11 ± 0.24 , p = 0.06). Additionally, the decrease in LV mass corresponded to a decrease in LV wall thickness (1.15 \pm 0.13 to 1.01 \pm 0.11 cm, p < 0.001 vs 1.17 \pm 0.18 to 1.08 \pm 0.18 cm, p = 0.14) in the R vs no R pts, respectively, while systolic blood pressure was unchanged and did not differ in R (122 \pm 6 to 115 \pm 6 mmHg) and no R pts (124 \pm 6 to 126 \pm 4 mmHg). Thus, ACEI promote diastolic remodeling of the LV via a decrease in LV mass which did not occur in the pts not receiving ACEI.

941-2

The Response of Myocardium to Hyperbaric Oxygen Predicts Recovery of Function Following Revascularisation

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Hyperbaric oxygen (HBO) can induce recovery of contractile function in

hibernating myocardium. This study assessed the ability of HBO induced recovery of function to predict improvement in the same region following revascularisation.

Fifteen patients (14 men, 43 to 80 years) with demonstrable wall motion defects on echocardiography (8 inferior, 4 anterior, 3 both: infarction 2 months—14 years previously) underwent 1 half hour of HBO treatment (100% O2 at 2 atmos) with transoesophageal echocardiography being performed before and immediately after. Echocardiography was repeated following revascularisation (bypass surgery 12, angioplasty 3). Ten patients showed improvement in wall motion with HBO, mainly in the borderzone of the wall motion abnormality and five showed no change. Of the 10 responders to HBO, 9 showed improvement in the same region following revascularisation. One patient showed substantial deterioration suggestive of perioperative infarction. Of the 5 patients who did not respond to HBO, none showed improvement following revascularisation.

Conclusion: The response to HBO predicted the recovery or non-recovery of left ventricular function following revascularisation in 14 out of 15 patients. Relatively brief exposure to HBO appears useful in identifying hibernating myocardium prior to intervention.

941-3

Baseline Left Ventricular (LV) Performance and Functional Recovery After Revascularisation: Predictive Value of Absolute ¹⁸F Fluorodeoxyglucose (FDG) Uptake

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To assess the relation amongst the severity of baseline LV dysfunction, myocardial glucose utilization measured with FDG positron emission tomography (PET), and functional recovery after revascularization (R), we studied 36 patients (pts) with coronary artery disease and chronic LV dysfunction. All had radionuclide ventriculography (RNV) and FDG PET during euglycemic hyperinsulinemic clamp (EHC) and repeat RNV 6 months after R. Two Groups (G) based on baseline ejection fration (EF) were identified: G1, EF < 30%</p> (N = 15; range: 12-30%; age: 57 \pm 11) and G2, EF > 30% (N = 21; range: 32-63% age: 61 ± 6). In G1 out of 135 segments (SEG) 53 were normal (N) and 82 dysfunctional (D); in G2 out of 185 SEG, 123 were N and 62 D. The percentage of D-SEG in G1 (61%) was greater than in G2 (33%; p < 0.01). LV-EF after R improved by $5 \pm 9\%$ (p < 0.05) in G1 but not in G2 (0.43%, p = NS). Wall motion score (WMS) improved more in G1 (from 1.83 ± 0.3 to 1.5 \pm 0.32) than in G2 (from 1.4 \pm 0.32 to 1.26 \pm 0.26; δ WMS: G1 vs G2: p = 0.05). The Metabolic Rate of Glucose [MRG, (\(\mu\text{mol/min/g}\)] in N-SEG was 0.44 ± 0.18 and 0.45 ± 0.18 in G1 and G2 (p = NS). In G1 the MRG was greater than in G2 both in D-SEG (0.41 ±0.15 vs 0.34 ±0.15 , p = 0.01) and in D-SEG that IMP after R (0.46 \pm 0.11 vs 0.38 \pm 0.15, p = 0.02) whereas it was non significantly greater in unchanged SEG (0.36 \pm 0.18 vs 0.28 \pm 0.12, p = 0.08). Conclusions: 1) LV function after R is more likely to improve in pts with EF < 30%; 2) The MRG during EHC is higher in D-SEG of pts with EF < 30% suggesting that overall there is a greater mass of recoverable mvocardium.

941-4

L-Arginine and Dobutamine Modify Myocardial Contractile Response to Receptor-Mediated Coronary Endothelial Stimulation After Cardiac Transplantation

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Both vascular smooth muscle tone and myocardial contractile properties are influenced by receptor-mediated coronary (Cor) endothelial (Endo) stimulation. In normal subjects, intracoronary (IC) influsion of substance P (SP) lowers left ventricular (LV) peak systolic (PS) and end-diastolic (ED) pressures because of a direct myocardial paracrine action of NO unrelated to systemic vasodilation. In the present study, Cor Endo control of LV contractile performance was investigated in transplant (Tx) recipients during IC infusion of SP (n = 22), during IC coinfusion of SP and L-arginine(Arg)(n = 10), which reverses Cor Endo dysfunction after Tx, and during IC infusion of SP following pretreatment with intravenous(IV) dobutamine (Dob)(n = 12) to investigate concurrent myocardial β -adrenergic stimulation, which potentiates cardiodepressant contractile effects of NO in rat cardiomyocytes. Micro-tip LV pressures were recorded in Tx recipients, free of rejection or graft vasculopathy, before(baseline), during IC infusion of SP(20 pmol/min)(IC-SP), during IC coinfusion of SP and Arg (160 µmol/min) (IC-SP-Am), and during simultaneous infusion of IC-SP and IV-Dob(5 µg/kg/min) (IC-SP-IV-Dob). Compared to baseline, IC-SP induced a fall in LVPSP ($\Delta = -17 \pm 3$ mmHg; p < 0.001) and in LVEDP (Δ = -2 ± 0.5 mmHg; p < 0.01). Compared to IC-SP, IC-SP-Arg caused an additional fall in LVPSP ($\Delta = -5 \pm 1$ mmHg; p < 0.01) and in LVEDP (Δ = -1 ± 0.5 mmHg; p < 0.05). Compared to IC-SP,