972-105 Afterload Reduction Selectively Improves Myocardial Deformation After Infarction in Adjacent Noninfarcted Regions

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Left ventricular remodeling post-infarction (post-MI) is accompanied by persistent dysfunction in the non-infarcted adjacent region. Chronic afterload reduction (ALR) therapy has a salutary effect on post-MI remodeling. The relative contribution of mechanical unloading to this effect remains unclear. We hypothesized that improved adjacent (ADJ) region dysfunction contributes to the beneficial effect of ALR. We therefore studied myocardial deformation in ADJ and remote (REM) non-infarcted regions in 5 sheep two months after anteroapical infarction during control (C) and after nitroprusside infusion (NP). Using magnetic resonance tissue tagging and finite element analysis techniques, we measured deformation using the orthogonal principal strains, λ_1 (1 + greatest systolic elongation), λ_2 (1 − greatest systolic shortening), and β or angular deviation of λ_1 from the radial direction. With NP, LV systolic pressure fell from 94 ± 9 to 69 ± 7 mmHg ($p < 0.05$) while end-diastolic pressure was unchanged, as measured by a high-fidelity catheter. *Results (mean ± S.E.):*

REM deformation and its orientation were similar in C and NP. In contrast:

	ADJ λ_1	ADJ λ_2	ADJ β (°)
C	1.06 ± 0.02†	0.88 ± 0.01†	45.6 ± 7.6†
NP	1.09 ± 0.03†	0.83 ± 0.01*†	25.8 ± 2.4*†
ANOVA p	NS	<0.05	<0.05

*$p < 0.05$ vs. Control, †$p < 0.05$ REM vs. ADJ

Thus in the ADJ regions, λ_1 and λ_2 were reduced compared to REM, and λ_2 decreased after NP, denoting greater systolic shortening. The most important change with NP was the normalization of ADJ β, resulting in a greater contribution to pump function. In conclusion, improved mechanical function in the ADJ non-infarcted regions, particularly the orientation of principal strains, is an important determinant of the beneficial effect of ALR on post-MI LV function.

972-106 Intramural Activation Patterns of Focal Ventricular Tachycardia Induced by Programmed Stimulation During Experimental Subacute Myocardial Infarction

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The purpose of this study was to elucidate the three dimensional (3-D) activation patterns of focal excitation during induced ventricular tachycardia (VT) in subacute myocardial infarction in canines. In 30 dogs 5 days after LAD occlusion and reperfusion, intramural electrograms were simultaneously recorded from 240 bipolar sites through 60 plunge needles inserted into both ventricles. Computerized 3-D color-coded isochronal activation maps were generated. At the given resolution, focal VT was diagnosed when the site of latest activation was anatomically remote from the site of impulse origin. In 12 of 26 experiments, reproducibly induced sustained monomorphic VTs were attributed to a focal mechanism. Activation patterns were the following: 1) Radial spread of excitation (n = 5). Ventricular activation was completed in approximately 100 ms followed by about 100 ms of electrical quiescence. 2) Progressive slow conduction (n = 2). A focal excitation pattern was associated with progressive prolongation of intramural conduction times. 3) Presystolic activity and unidirectional exit block (n = 2). The site of impulse origin was surrounded by areas of unidirectional conduction block with intramural conduction delay. 4) Pan diastolic activity (n = 3). Myocardial activation was present throughout the entire systolic and diastolic intervals without regional slow conduction or block. In conclusion, multiple intramural activation patterns are present in focal VTs induced by programmed stimulation. Delayed diastolic activity does not constitute a direct evidence for macro-reentry. Triggered activity with or without conduction delay may be the underlying mechanism of these induced arrhythmias.

972-107 L-Arginine Decreases Infarct Size in Rats Exposed to Environmental Tobacco Smoke

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We previously showed that environmental tobacco smoke (ETS) increased myocardial infarct size in a rat model of ischemia and reperfusion. If reduced reperfusion was caused by endothelial cell damage and increased vascular tone, we postulated that L-arginine (ARG) would increase nitric oxide and better protect the heart. 60 rats were randomly divided into 4 groups: ETS or Control (C) with and without ARG (2.25% ARG in drinking water). The ETS groups were exposed (4 Marlboro cigarettes per 15 minutes, 6 hours a day) for 6 weeks. During ETS-exposure, average air nicotine, carbon monoxide and total particulate concentrations were 1304 $\mu g/m^3$, 78 ppm and 31 mg/m^3, respectively. After 6 weeks, all rats were subjected to 35 min LAD occlusion (O) and 120 min reperfusion, with hemodynamic monitoring via the carotid artery. Aortic rings were harvested to evaluate vascular reactivity. Infarct size (infarct mass/risk area × 100%) decreased significantly in the ETS with ARG group compared to the ETS without ARG group. There were no significant differences among groups in heart rate (HR), systolic pressure (SP), and rate pressure product. Tlere were positive correlations between infarct size and heart rates from baseline to reperfusion 120 min (r = 0.4–0.6, p = 0.01–0.001). There was no relationship between vascular reactivity and infarct size.

Group	No. of Rats	Inf/LV (%)	Inf/RA (%)	O-35'HR (beats/m)	O-35'SP (mmHg)	Max Relax (%)
C	11	25 ± 3	51 ± 6	408 ± 11	120 ± 7	84 ± 11
C + ARG	10	25 ±2	52 ± 3	415 ± 10	103 ± 11	112 ± 15
ETS	10	34 ± 4	64 ± 6	427 ± 16	108 ± 8	128 ± 16
ETS + ARG	11	22 ± 3*	42 ± 6*	410 ± 17	106 ± 10	127 ± 18

Values are Means ± SEM (*$p < 0.05$, p values from two-way ANOVA)

Conclusion: L-arginine decreases myocardial infarct size after ischemia and reperfusion in ETS-exposed rats. This effect does not appear to be secondary to alterations in hemodynamics.

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Ernest N. Morial Convention Center, Hall E
Presentation Hour: 3:00 p.m.–4:00 p.m.

TUESDAY PM

973-108 Unstable Angina Patients Fare No Worse than Stable Patients Ten Years After Balloon Angioplasty

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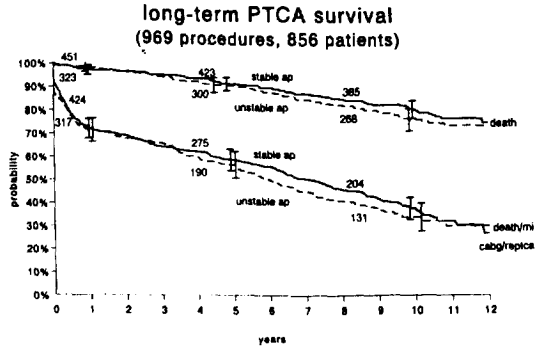
Aim. This study was designed to assess the 10 year clinical status of all 856 patients who underwent percutaneous transluminal coronary angioplasty (PTCA) in the first 5 years of our experience (1980–1985) and compare those with stable (SAP) and unstable angina (UAP).

Methods. All patients were contacted via letter, telephone or family doctor and details of cardiac events (death, infarction, rePTCA, coronary artery surgery) checked against hospital records. Actuarial survival and event-free survival curves were constructed and SAP and UAP patients compared.

Results. The overall 5 and 10 year actuarial survival was 91% and 79% respectively. The 10 year event-free survival for all patients was 35%. The outcome of SAP and UAP patients are compared and displayed in the table. There is no significant difference in survival or clinical events.



Conclusions. Ten year survival after PTCA is good although the majority of patients suffer a further cardiac event. There is no significant long-term difference in cardiac events between SAP and UAP patients.