960-66 Enalapril (EN) Improves Alveolar-Capillary Diffusion in Chronic Heart Failure (CHF) and Aspirin (AS) Contrasts This Effect

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Prostaglandin (PG) participants in the local control of transcapillary fluid flux and interstitial volume. ACE is concentrated in the lung vessels and ACE-inhibitors expose them to enhanced formation of PGs. Were PGs involved they are counteracted by a PG inhibitor.

Mean ± SD. *p < 0.05 vs PL; #p < 0.05 vs AS; $p < 0.05 vs EN + AS

Conclusions: a) Changes in DLCO and lung mechanics with EN are consistent with modulation of lung interstitial fluid, ameliorating respiratory and functional capacities; b) PG stimulation seems to underlie these actions as they are counteracted by a PG inhibitor.

960-67 C-Myc Expression Relates to Decrease in Myocardial Contractility in Patients With Chronic Aortic Regurgitation

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In patients with end-stage aortic regurgitation (AR), excess of volume-load on the left ventricle (LV) cause congestive heart failure associated with a decrease in LV contractility. Recently, proto-oncogenes have been implicated in the pathogenesis of myocardial remodeling resulting in decreases in LV contractility. However, in human heart, it has never been clarified whether C-Myc may relate to contractile impairment and structural alteration. In the present study, expression of C-Myc in myocardium was examined in patients with AR. Nine patients (7 male and 2 female) aged from 31 to 70 years (mean, 52.9 ± 13.4) with isolated chronic AR underwent aortic valve replacement. Preoperative NYHA class was III in 5 patients and IV in 4. Ejection fraction (VF) (EF) (49.2 ± 14.9%), end-systolic volume index of LV (ESVI) (87 ± 30 ml/m²) and left ventricular mass index (LVMi) (183 ± 34 g/m²) were measured before surgery. The specimens of LV muscle were obtained by endomyocardial biopsy during aortic valve replacement. Expression of C-Myc were evaluated by immunohistochemical staining. All patients showed significant higher values in myocardial cell diameter (CD) (24.2 ± 11.8 μm) and fibrous content (FC) (28.3 ± 13.2 g/m²) than control normal. C-Myc (28.3 ± 16.1%) was detected in these patients while, it has never been detected in normal control. EROSI were inversely correlated with EF(r = 0.94 p < 0.01), CD (r = 0.93 p < 0.01) and FC(r = 0.95 p < 0.01). C-Myc showed inverse correlation with ESVI(r = -0.26 p < 0.01), D (r = 0.93 p < 0.01) and FC(r = 0.92 p < 0.01). These results demonstrated that C-Myc expression relates to extensive cellular hyperplasia and interstitial fibrosis and inversely relate to ESVI in patients with chronic aortic regurgitation. This suggest that C-Myc may relate to cardiac remodeling, and its down-regulation may be associated with the decrease in LV contractility.

960-68 Differences In Temporal Risk of Sudden and Non-Sudden Deaths in Patients With End-Stage Heart Failure Listed for Cardiac Transplantation


Sudden death (SD) is a major threat to survival in patients with end-stage heart failure awaiting cardiac transplantation. Current prioritization of recipients for transplantation is based on criteria relating to hemodynamic compensation and risk of nonsudden death (NSD). Similar criteria for stratifying risk of SD do not exist and the temporal pattern of SD risk is unknown. We compared actuarial SD and NSD mortality rates in 162 patients listed for transplantation using life table analysis. There were 43/162 (26.5%) cardiac deaths (22/14.2% sudden and 20/12.3% nonsudden) during mean followup 15.8 ± 13.3 months. Hazard functions derived from life-table analysis were plotted as a function of time and the equations of the best-fit lines were calculated by linear regression (Figure).

The slope of the hazard function curve expresses the change in probability of death/month per month independent of censored events (transplantation). The SD slope was positive (+0.03 ± 0.01, R = 0.81, p = 0.04) and the NSD slope was negative (-0.05 ± 0.01, Time (months) after listing R = 0.92, p = 0.01) consistent with progressively increasing versus progressively decreasing rates of change in risk, respectively.

Conclusions: In patients with end-stage heart failure listed and awaiting transplantation, the risk of SD decreases over time, while in contrast, the risk of SD increases over time.

961 Role of Vasospasm

Tuesday, March 26, 1996, Noon-2:00 p.m.
Orange County Convention Center, Hall E
Presentation Hour: Noon-1:00 p.m.

961-69 Contribution of Coronary Vasoconstriction to Myocardial Blood Flow Changes Induced by Physiologic Stresses


Atherosclerotic coronary arteries constrict in response to exercise (EX), physical stress, and during myocardial ischemia (IS). We investigated the impact of coronary vasoconstriction on regional myocardial blood flow (MBF) in normal and ischemic zones during physiologic stresses including bicycle EX, mental stress (MS) using a video game, and compared it with adenosine (AD). Seven men (age 60 ± 10 years; 5 with 2- and 2 with 3-vessel CAD) had MBF assessed with N-13 ammonia uptake normalized to absolute flow in one sector using PET. All pts had normal left ventricular function (ejection fraction 56 ± 8%) and EX-induced wall perfusion defects. Ischemic segments were defined as regions where ADO dose-dependent MBF increase was < 90% of normal segments (supplied by unobstructed coronary arteries). MBF changes with EX and MS were compared with ADO in 171 normal and 101 ischemic segments. MBF increase to ischemic regions occurred during both EX and MS. MBF increase was < 50% of normal segments (supplied by unobstructed coronary arteries). MBF changes with EX and MS were compared with ADO in 171 normal and 101 ischemic segments. MBF increase to ischemic regions occurred during both EX and MS.

Rest ADO MS EX

MBF (Normal zones) 0.62 2.5 1.2 1.7
MBF (Ischemic zones) 0.75* 1.05* 1.1* 1.3*

p < 0.001, **p < 0.01

MBF was lower in ischemic segments at rest, and after MS and EX. Despite development of ischemia (ST depression) during EX which would be expected to maximize blood flow, MBF in the ischemic segments remained 29% below the level achieved with ADO (that measures flow reserve). Similarly, during MS, MBF in ischemic segments was lower than in normal areas, and much lower than ADO, suggesting that coronary vasoconstriction in ischemic regions occurs during both EX and MS. Thus, vasoconstriction of coronary stenoses during EX and MS causes significant attenuation in MBF increases to ischemic regions. Whether these changes can be reversed by anti-anginal medications needs to be further investigated.

961-70 Role of Coronary Tone in Vasospastic Angina

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Previous attempts to determine the role of basal coronary tone in the pathophysiology of variant angina have produced conflicting evidence. We examined the vasoconstrictor response to ergonovine and the vasodilator response to isosorbide dinitrate (basal coronary tone) at spastic and non-