p=0.001). Group B had more in-hospital complications (56% vs. 0%, p=0.001), such as congestive heart failure, ventricular septal rupture and pericardial effusion.
Conclusions: The evaluation of LAD flow using TCDE in pts with AMI in the ER is feasible. The assessment of CBF pattern using TCDE in pts with AMI is a rapid and promising method to predict clinical outcome, may contribute deciding the treatment strategy of AMI.

1041-95 Sildenafil Citrate (Viagra) Induces Powerful Cardioprotective Effects Following Ischemia-Reperfusion Injury in Infant Rabbits

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Background: We recently demonstrated that sildenafil citrate, a potent phosphodiesterase-5 inhibitor, induces a preconditioning-like effect through synthesis of nitric oxide and opening of mitochondrial K_{ATP} channels in adult rabbits. The purpose of this study was to demonstrate the effects of sildenafil on myocardial functional improvement and infarct size reduction during ischemia/reperfusion injury in infant rabbits.
Methods: 8-week-old rabbits were treated with sildenafil (0.7mg/kg IV) or saline 30 minutes prior to ischemia for 30 minutes and reperfusion for 3 hours. Transesophageal echocardiography was utilized to assess left ventricular cardiac output (LVCO) and aortic Velocity Time Integral (VTI). Sections of left ventricular myocardium were analyzed for infarct size using triphenyltetrazolium staining and computer morphometry.
Results: The sildenafil group had significant reduction in infarct size (15.5+/-1 versus 33+/-1.5 in controls, % risk area, mean+/-SEM, n=10-15/group, p<0.05). Sildenafil treated rabbits had a 34% decline in mean arterial pressure (MAP) and a 8% increase in heart rate (HR) compared to controls after drug administration (p<0.05) but were comparable to controls prior to ischemia. The controls had a decline in LVCO and aortic VTI after ischemia (18% and 16% lower than baseline values, respectively, p<0.05), while the LVCO and aortic VTI increased in the sildenafil group (20% and 15% higher than baseline values, respectively, p<0.05). This is a statistically significant increase in LVCO and aortic VTI in the sildenafil group compared to controls (n=6/group, p<0.05).
Conclusion: For the first time, we have shown that sildenafil citrate promotes myocardial protection in infant rabbits as evidenced by preservation and even elevation in post-ischemic LVCO and aortic VTI and reduction in infarct size. This may prove to be a viable model for myocardial ischemia in pediatric cardiac surgery involving cardiopulmonary bypass, circulatory arrest, or low flow states. As such, the mechanism of cardioprotection, either through preconditioning, enhanced inotropy, or hemodynamic effects, needs further investigation.

1041-96 Acute Myocardial Infarction Size Determines N-Terminal Pro-Brain Natriuretic Peptide Release as Measured by Contrast Enhanced Magnetic Resonance Imaging

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Background: Plasma N-terminal pro Brain Natriuretic Peptide (NTBNP) concentrations are elevated post Acute Coronary Syndromes (ACS) and predict left ventricular (LV) remodeling. It is unclear whether NTBNP release is due to myocardial necrosis or wall stress. We assessed the relationship between NTBNP and infarct size using rest contrast enhanced Magnetic Resonance imaging (ceMR).
Methods: 36 male and 18 female (median (range) age=61 (34-83) years index hospital admissions with ACS (initial treatment: 24 primary thrombolysis, 30 conservative) were consecutively recruited. Exclusions: serum creatinine >200µmol/l or history of chronic renal failure, hypertension, cardiomyopathy, significant valve disease or cor pulmonale. Blood sampling for troponin I (TnI) took place at 8-12 hours and for serum creatinine and NTBNP (Elecsys 2010, Roche NTBNP assay) prior to ceMR at a median (range) of 69 (16-120) hrs. LV dimensions were evaluated by cinematographic (TrueFISP) breath-hold sequence using a Siemens Sonata 1.5T system. ceMR was performed 10 minutes after injection of 0.2 mmol/kg gadolinium-DTPA using a breath-hold segmented turbo FLASH inversion-recovery sequence. Images were evaluated by planimetry by 2 blinded observers and the consensus used.
Results: Haemodynamic measures: mean (SD) heart rate 54 (11) bpm, systolic BP 122 (19) mmHg, diastolic BP 76 (12) mmHg. Renal function: creatinine 98 (22) µmol/l. Log NTBNP (median (range 3.03 (1.9 -3.96) pg/l) strongly correlated with late enhancement (LE) volume (mean (SD) 27 (25) ml: r=0.55, p<0.0001 but less well with TnI (median (range) 16.2 (0.3-417) µg/ml): r=0.34, p=0.01. TnI correlated strongly with LE volume: r=0.6, p<0.0001. Log NTBNP was unrelated to LV ejection fraction (mean (SD) 56 (10) %), LV mass (130 (36) g), LV end diastolic (137 (35) ml) or end systolic (60 (23) ml) volumes.
Conclusion: In patients with ACS the amount of myocardium damaged, assessed using ceMR, correlates strongly with an early increase in both plasma NTBNP and TnI. The correlation between NTBNP and TnI is not as strong suggesting that there is an additional mechanism underlying its release. There is no relationship between NTBNP and LV function and dimensions.

1041-97 Assessment of Transmyocardial Velocity Distribution During High Dose Dobutamine Stress Echocardiography Using Myocardial Velocity Profile

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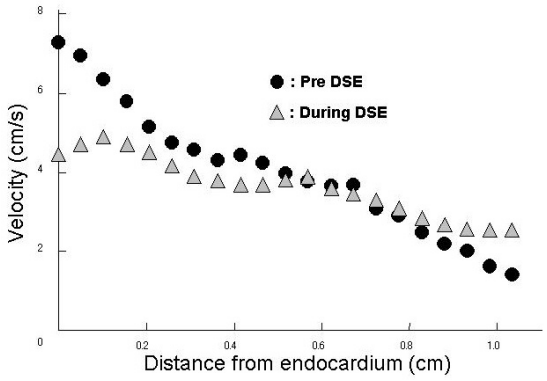
Background: High dose dobutamine stress echocardiography (DSE) has been popularly used for non-invasively detecting myocardial ischemia. Myocardial velocity profile (MVP) obtained by color tissue Doppler imaging (TDI) can optimize instantaneous distribution of myocardial velocity components from endocardium to epicardium.
Purpose: To assess the changes in transmyocardial velocity distribution during high dose DSE from an analysis of MVP.
Methods: Eighteen patients with chest pain suspected to have coronary artery disease by stress scintigraphy (99mTc-MIBI) were recruited for high dose DSE. Color TDI was recorded from parasternal short-axis view during DSE, and MVP was analyzed off-line.
Results: 1) In the region of perfusion defect by scintigraphy, the MVP at rest showed linear myocardial velocity distribution from endcardium to epicardium with the highest velocity at endocardium. 2) During high dose DSE, the peak systolic velocities at subendocardial region significantly decreased (3.99 vs 1.49 cm/s, p<0.001) resulting in the decrease in the slope of the linear regression line (myocardial velocity gradient)(5.01 vs 0.90 s^{-1}, p<0.001)
Conclusions: MVP obtained by color TDI clearly demonstrated changes in transmyocardial velocity distribution during DSE. Deterioration of the myocardial contractility in ischemic region by high dose DSE can be precisely evaluated by observing decrease in myocardial velocity gradient reflecting decrease in subendocardial velocities.



1041-98 Epigallocatechin-3-Gallate Inhibits Stat-1 Activation and Protects Cardiac Myocytes From Ischemia/Reperfusion–Induced Apoptosis

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Objectives. This study was aimed at evaluating the protective effects of epigallocatechin-3-gallate (EGCG) and green tea extract (GTE) on primary cultures of cardiac myocytes and the isolated rat heart respectively.
Background. We have previously demonstrated that STAT-1 plays a critical role in promoting apoptotic cell death in cardiac myocytes following ischemia/reperfusion (I/R) injury. EGCG, the major constituent of green tea, has recently been reported to inhibit STAT-1 activity in non-cardiac cells.
Methods and Results. Treatment of cardiac myocytes with 1µg/ml of EGCG 4 hours before simulated I/R (4 hours of hypoxia, followed by 16 hours of reoxygenation) reduced STAT-1 phosphorylation and provided protection against I/R-induced apoptotic cell death (annexin V and propidium iodide staining, followed by flow cytometer assessment) (p<0.01). Moreover, EGCG reduced the expression of pro-apoptotic Fas receptor and decreased Fas promoter activity, a known STAT-1 pro-apoptotic target gene. More interestingly, a freshly prepared solution of 0.1% GTE, supplied every day to treated animals, as the sole source of drinking water, over a period of 7 consecutive days, limited the extent of infarct size (25.4±1.7 vs 37±2.3% of I/R controls; p<0.05), and attenuated the magnitude of myocyte apoptosis (colocalization of TUNEL and cleaved caspase-3) (4.1±0.6 vs 7.1±1.4 of I/R controls; p<0.05), in the isolated rat heart exposed to 35 mins of regional I, followed by 2 hours R. Importantly, the lessening in necrotic and apoptotic cell death was associated with improved hemodynamic recovery and ventricular function in the ischemic/reperfused rat heart (p<0.05).
Conclusions. This is the first report showing that consumption of green tea is able to mediate cardioprotection and enhance cardiac function during I/R injury. Since GTE-mediated cardioprotection is achieved, at least in part, through inhibition of STAT-1 activity, we may postulate that a similar action can be implemented in the clinical setting, to minimize STAT-1 activation levels in patients with acute coronary artery disease (CAD).

1041-99 Mild Local Hypothermia Preserves High Energy Phosphate Metabolism During Regional Ischemia

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Background: Recently we showed that mild topical hypothermia without a cardioplegic component significantly decreased infarct size (by up to 65%) in the in vivo rabbit heart model of regional ischemia. The mechanism for this marked effect remains to be determined. We hypothesized that this cardioprotection was due to preservation of high energy phosphates.
Methods: The circumflex coronary artery (or a major branch) was isolated with a 4-O silk suture in anesthetized, opened-chest rabbits. Rabbits were randomized to: ischemic hypothermia (IH, n=10), or ischemic normothermia (IN, n=10) groups. In the IH group regional mild myocardial hypothermia (~32ºC) was initiated by placing an ice-water bag directly on the surface of the heart corresponding to the risk zone 15 min prior to the