In addition, a non-MI control group (n = 5) was given intracoronary VEGF. All animals received heparin. Coronary angiograms, left ventricular ejection fraction, anterior wall motion scores, and left ventricular end-diastolic pressure were obtained at baseline and at 7 days post MI. Capillary and arteriolar density (vessels per square mm) was quantified histologically using periodic acid-Schiff and factor VIII antibody staining by a blinded observer.

	arteriolar/capillary density (mean ± S.D.)
NS-with MI	6.5 ± 1.4
VEGF-no MI	7.3 ± 2.6
VEGF-with MI	12.9 ± 2.9

Capillary and arteriolar density was significantly increased in MI animals given VEGF vs. NS ($p = 0.004$) and in VEGF animals with MI vs. no MI ($p = 0.016$).

Conclusions: Use of VEGF was associated with an increase in capillary and arteriolar density and was most significant after MI. Additional studies may further define a role for VEGF in promoting molecular bypass following MI.

11:45

746-6 Myocardial Stretch Preconditions Isolated Working Rat Heart

Jean-F. Obadia, Michel Ovize, Claire Abadie, Valérie Maupoil, Anne Ovize, Marc Janier, Yves Minaire, Xavier André-Fouët, Luc Rochette. *Hôpital Cardiologique, Lyon, France; University of Dijon, France*

Preconditioning (PC) is believed to be directly triggered by brief ischemia/reperfusion. However, brief ischemia results in transient dilation (or stretching) of the heart. We sought to determine whether stretch per se, induced by rapid increase in preload, could precondition the isolated working rat heart. Thirty-four rat hearts were perfused at constant pressure with Krebs-Henseleit buffer for 30 minutes and then subjected to 30 minutes of global ischemia (37°C) followed 45 minutes of reperfusion. Prior to the 30 minute sustained ischemia, all hearts underwent a 15 minute treatment period consisting of no intervention (control; n = 11), 5 minutes of zero flow global ischemia followed by 10 minutes of reperfusion (PC; n = 11) or 5 minutes of transient LV stretch (ST; n = 12). Transient stretch of the left ventricle was induced by raising the preload from 5 to 20 cm H_2O for 5 minutes, ten minutes before the sustained 30 minute global ischemia. Cumulative CPK release during reperfusion was used as an index of irreversible myocardial injury. It averaged 20 ± 12 U/g in the control group versus 1 ± 1 U/g* and 3 ± 4 U/g* in PC and ST groups respectively (* $p < 0.01$ vs control), indicating that PC and ST hearts experienced a dramatic reduction in infarct size. These data suggest that transient stretch of the left ventricle may precondition the globally ischemic working rat heart.

747 Valvular Regurgitation: Echocardiographic Evaluation

Tuesday, March 21, 1995, 10:30 a.m.–Noon
Ernest N. Morial Convention Center, Room 102

10:30

747-1 Progression of Aortic Regurgitation Assessed by Doppler Echocardiography in 127 Patients: Degree of Regurgitation

Luis R. Padial, Alvaro Oliver, Alex Sagie, Maria T. Vivaldi, Ming Hui Chen, Nelmacy Freitas, J.A. Vazquez de Prada, Arthur E. Weyman, Robert A. Levine. *Massachusetts General Hospital, Boston, MA*

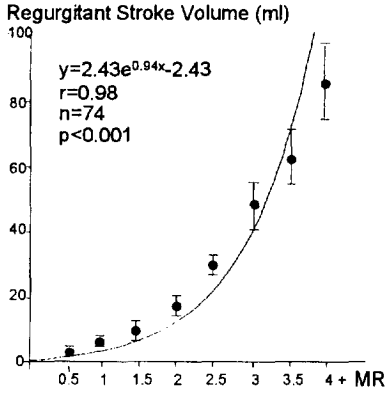
To assess the progression of chronic aortic regurgitation (AR), 127 patients (69 men; 59 ± 21 yr) with AR (59 mild, 8 mild-to-moderate, 41 moderate, 4 moderate-to-severe, 15 severe) who had ≥6 months of follow-up (6–47 months) by color Doppler and 2-D echo were studied. The degree of AR was established at entry and follow-up studies using an algorithm (semi-quantitative) that takes into account several Doppler criteria (jet area and jet height ratios, jet length, pressure half-time, reversal of flow in descending aorta) and the jet height/LV outflow tract (LVOT) height ratio (quantitative). LV volume (Simpson) and LV mass (Devereux) were calculated. A significant increase in jet/LVOT height ratio was observed in the whole population (30 ± 17 vs. 35 ± 20%; $p < 0.00001$) and in the subsets of patients with mild (18 ± 7 vs. 22.3 ± 9%; $p < 0.01$), moderate (40 ± 14 vs. 44 ± 16%; $p < 0.01$) and severe (54 ± 19 vs. 65 ± 15%; $p < 0.05$) AR. An increase in the degree of AR (semi-quantitative) during the follow-up was observed in 38 (30%) of patients: 25% with mild, 37% with mild-to-moderate, 44% with moderate and 50% with moderate-to-severe AR ($p < 0.006$). Patients were further divided according to the rank order in the rate of progression of jet/LVOT height ratio into "progressives" (n = 18; rate >5.64%/yr) and "non-progressives" (n = 109). Patients with "progressive" AR had a faster rate of progression of LV end-diastolic volume, LV end-systolic volume and LV mass than "non-progressives" ($p < 0.025$). *Conclusions:* AR is a progressive disease even in patients with mild insufficiency. The progression in the degree of AR is more frequent in patients with more severe disease. The rate of progression of regurgitation appears to play a role in LV overload in patients with AR.

10:45

747-2 Is the Semiquantitative Grading Score for Mitral Regurgitation Linearly Related to Regurgitant Flow?

Min Pu, Pieter M. Vandervoort, Brian P. Griffin, William J. Stewart, Delos M. Cosgrove, James D. Thomas. *Cleveland Clinic Foundation, Cleveland, Ohio*

Mitral regurgitation (MR) imposes a volume work load on the left ventricle (LV) that is proportional to the severity of the regurgitation. However, it is not clear how semiquantitative grading 1–4+ is related to quantitative regurgitant flow. *Method:* 74 patients with MR were investigated using multiplane transesophageal echocardiography. MR was graded semiquantitatively on a 4 point scale by two observers, integrating information on jet size and morphology in multiplane views and pulmonary venous flow patterns. Blinded to the results of the semiquantitative MR grading, regurgitant stroke volume (RSV) was calculated as the difference between thermodilution stroke volume and forward stroke volume through the mitral annulus (SV_{ma}), obtained by the mitral annulus area multiplied by mitral inflow velocity-time integral recorded by pulsed Doppler at the mitral annulus. Assuming elliptical shape, the mitral annulus area (A) was calculated as $A = \pi ab$, where a and b were radius of mitral annulus measured by 2-dimensional echocardiography at four and two chamber views. *Results:* Semiquantitative MR grading correlated best with mean RSV ($r = 0.97$ $p < 0.001$ Fig.) using an exponential fit. RSV dramatically increased when MR grading was >2+ with significantly increasing slope. 4+ MR (81 ± 36 ml) involved much more than double RSV as 2+ MR (19 ± 12 ml $p < 0.001$). *Conclusion:* Because its relationship with regurgitant flow is nonlinear, the semiquantitative MR grading score does not accurately reflect the amount of volume load on the LV, especially above MR grading 2+.



11:00

747-3 Automatic Correction of Overestimation of Mitral Regurgitation Caused by the Proximal Flow Constraint: Digital Analysis of the Proximal Flow Convergence

Min Pu, Pieter M. Vandervoort, Neil L. Greenberg, James D. Thomas. *Cleveland Clinic Foundation, Cleveland, Ohio*

We have previously shown that an adjacent proximal wall (AW) constrains the converging flow causing overestimation of mitral regurgitation calculated by the proximal flow convergence method (PFC). In this study we investigated a correction factor to overcome this overestimation. We hypothesized that the convergence angle is a function of wall distance (d) and proximal velocity contour at radius r from orifice and can be approximated as: $\pi + 2\tan^{-1}d/r$. *Method:* Steady flow rates (32–285 ml/s) were produced through circular orifices with areas of 0.1 to 1.0 cm^2 and peak velocities of 310–510 cm/s. Different degree of wall constraint were studied with the AW at 1, 3, 5, and 10 from the orifice. True flow (Q_t) was measured by timed collection. Digital color Doppler images (HP 1500) were transferred to a PC. The centerline velocity profiles were plotted against distance from the orifice. Flow rate (Q) was calculated as $Q = 2\pi r^2 v$ at velocities v between 5–10% of peak orifice velocity, assuming hemispheric flow convergence. Flow rate was also cal-