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1037-25 **One-Year Clinical and Echocardiographic Follow-Up After Endocardial Radiofrequency Ablation for Atrial Fibrillation During Mitral Valve Surgery**

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Background : Atrial fibrillation (AF) is associated with adverse events particularly in mitral valve (MV) disease. Surgical radiofrequency (RF)ablation might be an interesting alternative to the Maze procedure for treatment of AF in MV disease. Methods: Over the past two years, 58 patients (pts, 65±10 years) operated on for mitral regurgitation (36) or stenosis (22) with either chronic (41) or paroxystic (17) AF benefited in the same time for RF ablation. RF ablation was realized on the endocardial layer using a eight tip RF probe. Cardiac rhythm was evaluated at 3, 6 and 12 months with clinical examination, patient questioning, and ECG. A 24 hours ECG record was performed after 6 months. Echocardiographic examination, including left and right atrial function, tricuspid and mitral transvalvular flow velocities was performed before operation, 7 days, 3 months, and within 1 years after operation. Results: Pts of the chronic group were significantly older, had a longer period of AF (p=0,02) and a greater left atrium (p=0,003) compared with the paroxystic group. Surgical RF ablation was performed in the left atria (55) or in both atriums (3). MV surgery consisted in MV repair in 11 pts, and MV replacement in 47 pts (mechanical prosthesis: 28, bioprosthesis: 19). Mean antiarythmic procedure duration was 19±6 min, and aortic cross clamp time 121±27 min. Post-operative complications were 1 death from multiorgan failure, 1 permanent atrioventricular block requiring a pacemaker implantation, and a circonflex artery stenosis. At 6 months 67% of pts were free of arrythmia, 33% experienced at least one access of supraventricular arrythmia, and only 12% had several recurrences. Antiarrythmic drugs were withdrawn in 25% of pts. Left and right atrial contraction were effective in 84% of pts on the basis of doppler transvalvular A wave. Conclusion: RF ablation for AF during MV surgery is an effective and simple procedure resulting in persistent sinus rhythm after 1 year. This procedure preserve atrial contraction and might allow withdrawal of antiarrythmic and anticoagulant drugs.

1037-26 **Predictors of Saphenous Vein Graft Patency: A Risk Assessment Based on a Longitudinal Analysis of 100 Consecutive Post-Coronary Bypass Angiographic Studies**

Todd K. Rosengart Eileen B. Finnin, Stacie A. Hudgens, Elizabeth A. Hahn, Ronald D. Curran, Timothy V. Votapka, Michael W. Frank, Thomas G. Frohlich, Michael H. Salinger, Timothy J. McDonough, Theodore E. Feldman, Timothy A. Sanborn, Evanston Northwestern Healthcare, Evanston, IL

Background: Saphenous vein graft (SVG) occlusion remains a persistent complication of coronary bypass grafting (CABG). Conduits such as the internal mammary and radial arteries have been promoted as useful alternatives to SVG bypasses, a risk profile useful in delineating contributors to SVG occlusion would consequently be helpful in planning for the use of alternative conduits.

Methods: Potential predictors of SVG occlusion were evaluated for all patients (M:F, 69:31; mean age = 64 ± 11 years) undergoing CABG at our institution since 1995. Post-CABG angiographic data were added to a prospectively maintained database(n=100 patients, total number of grafts=318). Patients were included in this study if the graft target territory was a vein (left anterior descending (LAD), circumflex (Cx), right coronary artery (RCA)) rather than an alternative arterial conduit (right and left internal mammaries). The generalized estimating equation method was implemented to evaluate risk factors for SVG occlusion (partial/full occlusion vs. patent). Potential graft-and patient-specific risk factors for occlusion included: target artery caliber (<=1.5 vs. 2.0), vein quality (poor/fair vs. good), graft target territory , gender, age, ejection fraction (50), diabetes, obesity, family history, hypercholesterolemia, smoking, and hypertension.

Results: Median time to post-op cath was 24 ± 22 months (interquartile range, 6-37 months). Median SVG patency was 50% (0-100%), compared to internal mammary artery patency of 91%. Target graft territory was associated with increased SVG occlusion: unadjusted odds ratio Cx vs. LAD, 1.56 (95% C.I.=0.75-3.27); RCA vs. LAD, 0.70 (0.36-1.35); Cx vs. RCA, 2.24 (1.22-4.10). The only other vein specific risk factor for occlusion was vein quality: unadjusted odds ratio, 1.6 (0.89-2.86). Patient-specific risk factors with a trend towards association (p < 0.25) with increased SVG occlusion included: gender (female vs.male), 1.87 (0.99-3.56); diabetes, 1.72 (0.88-3.35); and family history, 1.45 (0.80, 2.65).

Conclusions: Alternative conduits should be considered for saphenous vein grafts with a poor likelihood of patency.

POSTER SESSION

1059 Basic Concepts in the Pathophysiology of Valvular Heart Disease

Sunday, March 30, 2003, 3:00 p.m.-5:00 p.m.
McCormick Place, Hall A
Presentation Hour: 3:00 p.m.-4:00 p.m.

1059-21 **Increased Presence of Pathogen Burden and of Circulatory Precursor Cells in Prosthetic Compared to Native Degenerated Aortic Valves**

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Background: Based on the concept of chronic valvular infections, the present study sought to assess the presence of *Chlamydia pneumoniae* (chlamydial heat shock protein 60), *Helicobacter pylori* (HP), Cytomegalovirus (CMV), Herpes simplex virus (HSV) and Epstein-Barr virus (EBV), as well as the possible association between this pathogen burden on inflammation (CD68, CD3, CRP) in valvular degeneration of native *versus* bioprosthetic valves. In addition, circulatory precursor cells (CD34, CD133) that might contribute as repair cells were evaluated. **Methods:** Serial sections of degenerated native (n=57) and prosthetic (n=23) aortic valves were analyzed by immunohistochemistry and computer-aided morphometry for the presence of these determinants. **Results:** Degenerated aortic valves revealed prevalence of *Chlamydia penumoniae* in 65%, of CMV in 66%, of HP in 80%, of HSV in 76% and of EBV in 68%, while immunoreactive CD68 was present in 79%, CD3 in 66% and CRP in 58%. CD34 and CD133 were observed in all valves. Quantitatively, presence of pathogens (each P<0.001) as well as markers of inflammation (CD68: 34.2±29.7% *vs.* 3.4±4.3%, P<0.001; CRP: 7.8±13.8% *vs.* 2.1±4.2%; P<0.01) and precursor cells (CD34: 18.1±25.0% *vs.* 5.7±13.4%, P=0.014; CD133: 12.4±13.2 *vs.* 5.6±8.5%, P=0.011) were significantly increased in prosthetic compared to native valves. Microorganisms correlated significantly with markers of inflammation. When categorizing into valves burdend by 4 or 5 pathogens (n=49) and into valves with < 4 microorgansisms (n=31), increased signaling of CD68, CD3 and CRP were observed in the first group (P<0.001).
Conclusions: Valvular infection, inflammation and circulatory progenitor cells are frequently found in degenerated aortic valves. Their differential presence of the non-valvular cells in native and prosthetic valves suggest common pathomechanisms and a more important role for prosthetic degeneration. Correlation of pathogens with macrophages and CRP in valvular fibrosa point to regional stressor effects that might be, at least partly, attributable to cumulative pathogen burden.

1059-22 **Autocrine-Paracrine Factors Released by Cardiac Fibroblasts Stimulate Abnormal Myocardial Fibronectin Synthesis in Aortic Regurgitation**

Sharada L. Truter, Eungsuk Lee, Zhen S. Huang, Jennifer A. Lee, Jeffrey S. Borer, Weill Medical College of Cornell University, New York, NY

Background: Chronic aortic regurgitation (AR) in patients (pts) and in NZW rabbits (R) with surgically-induced experimental AR causes extensive myocardial fibrosis contributing to heart failure (CHF). We demonstrated experimentally and in pts that AR fibrosis results from abnormal expression of fibronectin (FN) and other glycoproteins, but not collagen, by cardiac fibroblasts (CF). However, FN hyperexpression continues in CF culture even after AR-strain is removed. Thus, AR-CF may propagate FN excess by secreting FN-stimulators that persist. **Methods:** To seek these, CF were cultured from NZWR without (n=3) and with surgically induced AR (n=3). NL-CF cultures (passage 6) were incubated with medium conditioned (ARCM) by AR-CF and fractionated with 30, 50 and 100kD filter columns, and in medium conditioned by NL-CF (NLCM). After 24 hrs, NL-CF media and lysates were assayed for FN. **Results:** By Western analysis, FN-expression by NL-CF incubated with <100kD ARCM fractions was upregulated [1.7:1, p=0.04] vs NL-CF in NLCM (Table), but FN upregulation was absent in NL-CF grown in <50kD or < 30kD ARCM (Table). As in our earlier reports, FN was upregulated when AR-CF were grown in their own <100kD CM (AR:NL=3.4:1, p<.01).
Conclusion: Chronic AR causes CF to release autocrine/paracrine factor(s) sized 50 to 100 kD, that stimulate FN in NL-CF and may propagate FN upregulation in culture many passages after removal of AR stresses. These factors may be targets for novel therapies to prevent myocardial fibrosis and CHF in AR.

FN Expression (NL-CF in ARCM: NL-CF in NLCM)

Cell Line	AR-RF(%)	ARCM <30kD	ARCM <50kD	ARCM <100kD
NL-CF1	14	0.3:1	0.9:1	1.3:1
NL-CF2	25	0.9:1	1.5:1	1.6:1
NL-CF3	72	0.5:1	0.8:1	2.1:1
Average		**0.6:1 (p<.04)**	**1.2:1 (NS)**	**1.7:1 (p<.04)**