4:30

722-3 Coronary Stenting During Acute Myocardial Infarction. Results From the Stent Without Coumadin French Registry

Thierry Lefèvre, Marie-claude Morice, Gaêtan Kamillon, Pierre Aubry, Gilles Zemour, Bernard Valeix. *ICV Paris Sud, France*

Intra coronary Thrombus and acute M.I were till recently a contraindication to coronary stenting. Ticlopidine has dramaticaly decreased the risk of subacute thrombosis after stenting to the point that some patients were stented during acute M.I in the registry. To evaluate the outcome of those Pts a retrospective analysis was conducted.

All Pts (2901) included in the registry from March 1991–1995 had Ticlopidine (250–500 mg/day) the day of PTCA for 1 month, aspirin (100–250 mg/day) for > 6 months and low molecular weight heparin (antiXa 0.5–1) for 1 month in phase II (237 Pts), 15 days in phase III (521 Pts), 7 days in phase IV (960 Pts) and not in phase V (1183 Pts).

Only 85 Pts (2.9%) had primary or rescue PTCA with stenting during acute M.I (age 59.8 ± 11.3 years, 11% female). Indication for stenting was respectively: suboptimal result (26%), non occlusive dissection (28%), occlusive dissection (11%) and de novo lesion (35%). Stented coronary arteries were LAD: 39%, RCA: 49%, Cx: 9%, L.M: 2% and Bypass: 5%. Palmaz-Schatz stents were used in 74% of cases, AVE stents in 24% and other stents in 2%. One stent was used in 80% of cases and > 1 in 20%). Balloon used for stenting was 3.38 ± 0.46 mm in diameter with an inflation pressure of 12.3 ± 3.0 atm. No vascular complication, stroke or emergency CABG occured at 1 month follow-up. Subacute closure occured in 1.2% of cases, elective CABG in 1.2%, acute M.I in 2.4% and death in 5.9%. The composite end-point of subacute closure, acute M.I., CABG and death occured in 8.2%.

In conclusion: coronary stenting is feasable in acute M.I with a low rate of subacute closure. Randomised study are necessary to evaluate mid-term outcome compared to POBA.

4:45

722-4 Intracoronary Stenting in the Setting of Myocardial Infarction

Michel R. Le May, Marino Labinaz, Rob S.B. Beanlands, Louise A. Laramée, Ed O'Brien, Jean-Francois Marquis, William L. Williams, Khalid Al-Sadoon, Richard F. Davies, Sharon Ann Kearns, Helen L. Johansen, Lyall A. Higginson. *Ottawa Heart Institute, Ottawa, Ontario, Canada*

Data on feasibility, safety and clinical outcome of intracoronary stenting (ICS) in the setting of MI are limited. Palmaz-Schatz stents were inserted in 49 patients admitted with acute MI between 01/94 and 05/95. Group A, (n = 23) were stented early, day 0 to 7 of MI and Group B, (n = 26) were stented late, 8–25 days post MI. Demographics in the two groups were similar except that all patients with cardiogenic shock (n = 4) were in Group A (P < 0.05). Only the infarct-related artery was stented. Quantitative measurements were obtained on the target lesion before and after ICS Between Group A and Group B there was no difference in minimal luminal diameter (MLD) or % diameter stenosis (DS) before or after ICS. In Group A, MLD increased from 0.43 ± 0.36 to 2.58 ± 0.47 mm (P < 0.0001); in Group B, MLD increased from 0.55 ± 0.33 to 2.61 ± 0.40 mm (P < 0.0001). Percentage DS decreased in Group A from 83 ± 14 to 15 plusmn; 13 (P < 0.0001) and in Group B from 79 ± 12 to 12 ± 12 (P < 0.0001). Visible thrombus was present in 39% of Group A and in 23% of Group B before ICS (P = 0.35) but was not associated with adverse outcome. Only one patient had visible thrombus post ICS which resolved with intracoronary urokinase. During hospitalization, no patient experienced recurrent MI or required CABG; in Group A, 2 patients required repeat PTCA and one patient died. There were no events in Group B. At follow-up of 5.9 ± 4.0 months, all Group A patients were free of recurrent MI, repeat PTCA, CABG, and death; 80% were free of angina. In Group B patients followed to 7.3 ± 4.5 months, there were 2 deaths; 3 patients required repeat PTCA and no patient required CABG; 71% of the remaining patients were free of angina.

Conclusion: ICS in the setting of MI is associated with excellent immediate angiographic success and favourable clinical outcome. Intracoronary thrombus does not preclude stenting in patients with MI.

5:00

722-5 Elective Stent Implantation in Acute Coronary Syndromes Induced by Thrombus Containing Lesions

Miguel Romero, Alfonso Medina, José Suárez de Lezo, Manuel Pan, Enrique Hernández, José Segura, Francisco Melián, Djordje Pavlovic, José R. Ortega. *Reina Sofía Hospital, University of Córdoba and Pino Hospital, University of Las Palmas, Spain*

The presence of angiographic thrombus in coronary lesions has been identified as a risk factor for adverse events during percutaneous revascularization; the use of stents could theoretically be contraindicated because of the intrinsic thrombogenicity of the stent-wires. We postulated that the achievement of the highest lumen diameter in this setting, could overcome such an adverse thrombogenic combination. From a total of 812 patients (pts) treated by stent implantation, we selected for study 87 having coronary lesions with angiographic evidence of intraluminal filling defects, and rest angina within the last 48 hours. At cardiac catheterization the responsible artery was totally occluded in 41 pts and severely stenosed in 46 (mean 82 ± 12%). Before treatment or after recanalization, intraluminal thrombus was always evidenced. A stent was implanted trying to achieve an optimal lumen size. After deployment the residual stenosis was 4 ± 9%. The filling defect decreased, but persisted in 10 and disappeared in 77. All pts became asymptomatic; 38 of them received conventional antithrombotic regimen (iv-heparin and coumadin) and 30 were treated by low-molecular-weight heparin, ticlopidine and aspirin; 3 patients had a myocardial infarction; no other major complications occurred; 41 patients (47%) were angiographically reevaluated at a mean of 7 ± 4 months; 12 of them (29%) showed restenosis.

Conclusions: Elective stent treatment may be of benefit in unstable patients with thrombus containing stenosis. The thrombogenic combination of preexisting thrombus and metal, paradoxically, does not preclude a good outcome.

5:15

722-6 Preliminary Experience With the POSSIS Coronary AngioJet Rheolytic Thrombectomy Catheter in the VeGAS I Pilot Study

Stephen R. Ramee, Richard E. Kuntz, Richard A. Schatz, Joseph P. Carrozza, Jeffrey J. Popma, Alyce S. Lanoue, Cynthia Senerchia, Robert C. Stoler, Kalon K.L. Ho, Donald S. Baim. *Ochsner Hospital, New Orleans, LA; Beth Israel Hospital, Boston, MA*

The current percutaneous treatment of thrombotic obstructions in native coronary arteries (NC) or saphenous vein grafts (SVG) is limited by distal embolization and incomplete evacuation or dissolution in many cases. The POSSIS AngioJet catheter removes intravascular thrombus bodies by rheolytic thrombectomy: a Venturi effect created by precisely directed high pressure saline jets located at the tip of the 5 French over-the-wire catheter. The VeGAS I Pilot study will evaluate the AngioJet in saphenous vein grafts and native coronary arteries. In 15 lesions treated in the first 15 patients, 8 were in SVG and 7 in NC (including 3 patients with acute myocardial infarction). Thrombotic appearing obstructions were successfully reduced in 14 of the 15 (93%) cases with AngioJet alone from 91 ± 18% diameter stenosis to 44 ± 22% (visual estimates). In the one unsuccessful case, the underlying stenosis was concluded to be non-thrombotic. In all cases, subsequent treatment included balloon angioplasty in 2, directional atherectomy in 1, and Palmaz-Schatz stenting in 12, for final residual diameter stenosis of 5 ± 12%. Complications included transient heart block requiring temporary pacing in 6, and transient "no reflow" in 2.

Conclusion: 1) The AngioJet removes intravascular thrombus in SVGs and native coronary arteries with high success and low complication rates. 2) Successful thrombus removal appears to prepare the vessel for subsequent safe and uncomplicated treatment by stenting, balloon angioplasty, or directional atherectomy. 3) The final results of this 60 patient pilot study should provide the background experience for the phase 2 randomized trial, VeGAS II.

723 Neurohormonal Factors in Heart Failure

Monday, March 25, 1996, 4:00 p.m.–5:30 p.m.
Orange County Convention Center, Room 208

4:00

723-1 The Relative Value of the Natriuretic Peptides as Markers for Detecting Abnormal Ventricular Structure and Function

Kazuhiro Yamamoto, John C. Burnett, Jr., Michihisa Jougasaki, Yoshihiko Saito, Kazuwa Nakao, David R. Holmes, Jr., Margaret M. Redfield. *Mayo Clinic, Rochester, MN*

Previous studies have suggested that brain and C- and N-terminal atrial natriuretic peptides (BNP, C-ANP, N-ANP) may have diagnostic utility in the detection of LV hypertrophy (LVH) or LV dysfunction. The current study was designed to determine the relative utility of measuring serum levels of BNP, C-ANP or N-ANP to detect LVH or systolic or diastolic dysfunction. BNP (Shionogi), C-ANP (Peninsula) and N-ANP (Phoenix) were determined by radioimmunoassay, ejection fraction (EF) and LV mass index were measured

by echocardiography and the time constant of LV relaxation (Tau) was derived from the LV pressure to assess diastolic function in 94 consecutive patients with suspected cardiac disease. The sensitivity and specificity of optimal values of each peptide for detecting abnormal LV function or LVH were:

EF < 45%	sensitivity: BNP (83%) > C-ANP (79%) > N-ANP (67%) specificity: BNP (81%) > C-ANP (67%) > N-ANP (63%)
Tau > 55 ms	sensitivity: BNP (85%) > N-ANP (70%) > C-ANP (60%) specificity: BNP (74%) > C-ANP (65%) > N-ANP (61%)
LV mass index > 120 g/m^2	sensitivity: BNP (81%) > N-ANP (73%) > C-ANP (69%) specificity: BNP (90%) > N-ANP (79%) > C-ANP (67%)

By receiver-operator-characteristic (ROC) analysis, BNP was significantly more sensitive and specific than C-ANP or N-ANP for detecting abnormal EF, Tau or LV mass index with an ROC value of 0.85, 0.82, and 0.91 respectively. Sensitivity for detecting abnormal LV structure or function employing a "natriuretic peptide panel" with an abnormal BNP *or* C-ANP *or* N-ANP exceeded that of BNP alone and was for EF < 45% (96%), Tau < 55 ms (90%) and LV mass index > 120 g/m^2 (96%). *Conclusions:* BNP is the single best test to detect abnormal systolic and diastolic function and LVH in patients with suspected cardiac disease. A "natriuretic peptide panel" has very high sensitivity and may be a useful screening test in some populations.

4:15

723-2 Attenuation of B-type Natriuretic Peptide (BNP) Secreted From an Infarcted Segment Reresents the Process of Left Ventricular Remodeling After Myocardial Infarction

Atsushi Hirayama, Fuminobu Ishikura, Naoyuki Misaki, Hisakazu Fuji, Takayoshi Adachi, Yashuhiko Sakata, Kazuhisa Kodama. *Osaka Police Hospital, Osaka, Japan*

As B-type natriuretic peptide (BNP) is known to be a marker for ventricular hypertrophy and/or dilatation, its measurement at the infarcted segment might be useful to evaluate infarct expansion after the onset of myocardial infarction (MI). Seventy five patients of first anterior MI without any cardiac event since their first attack were subjected to cardiac catheterization performed 18 ± 15 months (range 1 to 45 months) after the onset of MI. Hemodynamic parameters, left ventricular (LV) volumes and function on LV graphy and plasma BNP concentration (pg/ml) of aortic root (Ao) and anterior interventricular vein (AIV) were measured by radioimmunoassay. The difference of plasma BNP concentration between AIV and Ao (AIV-Ao), which reflect BNP secreted from the infarct segment, had significant correlations with EDVI (r = 0.67, p = 0.001), ESVI (r = 0.65, p = 0.001), EF (r = 0.71, p = 0.001), LVEDP (r = 0.53, p = 0.003), and the duration from the onset of MI to the cardiac catheterization (r = −0.75, p = 0.001). By multivariate analysis, however, EDVI (p = 0.003) and the duration from the onset of MI (p = 0.0021) among the variables were picked up as significant factors determining the plasma concentration of BNP at AIV-Ao. These results indicated that BNP released from the infarcted segment is enhanced by the ventricular dilatation and attenuated in the course of time after MI. These results suggested the secretion of BNP at the infarcted segment was stimulated by the infarct expansion at the early period after MI but decreased with the accomplishment of expansion during the late period.

In conclusion, BNP secreted from the infarct segment represent the process of LV remodeling after MI.

4:30

723-3 Repetitive Bolus Administration of Brain Natriuretic Peptide Reduces Cardiac Filling Pressures in Human Heart Failure

Robert J. Cody, for the Natrecor hBNP Multicenter Bolus Trial. *Ohio State University, Columbus, Ohio*

Endogenous natriuretic peptides (NP) produce vasodilation, in response to the myocardial remodeling of congestive heart failure (CHF). Individual NP sequences of single gene origin, confer distinct cardiovascular characteristics. In this multicenter trial, we evaluated the effect of repetitive bolus administration of brain natriuretic peptide (hBNP) in patients (pts) with CHF, during hemodynamic evaluation. 28 of 29 pts were NYHA III or IV, and all had EF < 30%. hBNP was given as 5 μg Q4H (7 pts), 10 μg Q4H (6 pts), 10 μg Q6H (6 pts), with placebo groups for Q4H (6 pts) and Q6H (4 pts) intervals. For each treatment interval, pts received repetitive doses over 24 hrs. Placebo groups were pooled for statistical analysis, considering between-/within-group differences, and dosage hierarchy, p < 0.05(*) being significant. Pulmonary wedge (PWP) and right atrial (RAP) pressures:

	Placebo	5 μg Q4H	10 μg Q4H	10 μg Q6H
PWP (mmHg)				
Baseline	27 ± 7	25 ± 6	32 ± 6	27 ± 5
hBNP + 0.5 hrs	26 ± 9	16 ± 5*	19 ± 8*	12 ± 6*
hBNP + 1 hrs	27 ± 8	15 ± 6*	20 ± 7*	13 ± 6*
hBNP + 2 hrs	26 ± 5	18 ± 8*	21 ± 9*	16 ± 6*
RAP (mmHg)				
Baseline	15 ± 6	13 ± 8	12 ± 4	12 ± 7
hBNP + 0.5 hrs	15 ± 7	10 ± 8*	8 ± 6*	7 ± 5*
hBNP + 1 hrs	16 ± 7	9 ± 9*	8 ± 6*	6 ± 5*
hBNP + 2 hrs	14 ± 6	10 ± 8*	7 ± 4*	7 ± 5*

At the common time point of 12 hours, PWP (15 ± 5) and RAP (4 ± 5,) were p < 0.05 for the 10 μg Q4H interval. With the final bolus in each group, persistent response of PWP and RAP indicated no tachyphylaxis. Changes in cardiac index and systemic resistance were concordant, consistent with vasodilation. In human CHF, exogenous hBNP improves cardiac filling pressures in the setting of vasodilation, suggesting preserved hemodynamic responsiveness of the "type A" NP receptor, while reducing the atrial tension stimulus for endogenous NP release.

4:45

723-4 Exhaled Nitric Oxide as a Measure of Response to Therapy in Patients With Heart Failure

Joshua M. Hare, Anthony F. Massaro, Jeffrey M. Drazen, Wilson S. Colucci. *Brigham and Women's Hospital, Boston, MA*

Endogenous nitric oxide (NO) can be detected in exhaled air and increases with exercise and L-arginine administration, possibly reflecting vascular release. We hypothesized that exhaled NO would be elevated in patients with heart failure (HF) as a compensatory circulatory mechanism, and would decrease as hemodynamics improved with vasodilator and diuretic therapy. Chemiluminesence was used to measure mean mixed expired NO content of a vital capacity breath in patients with HF (n = 5) and matched controls (n = 5) during inhalation of NO-free air. Exhaled NO was higher in HF than in controls (8.4 ± 3.5 vs. 6.5 ± 2.4 ppb, p < 0.04). Serial measurements were made over of period of therapy (7.3 ± 6 days) with sodium nitroprusside (SNP) and diuresis during monitoring of pulmonary arterial pressures and cardiac output in 7 additional patients with HF breathing room air.

	Pre-therapy	SNP for 2 hrs.	Post-therapy
NO (ppb)	20.4 ± 6.2	22.8 ± 4.6	11.2 ± 1.2*
PVR (dyne-sec-cm-5)	356 ± 47	287 ± 63	147 ± 32*
CO (L/min)	3 ± 0.3	4.64 ± 0.9	4.8 ± 0.3†
PCWP (mmHg)	31 ± 3	27 ± 2	21 ± 7†

*p < 0.05 vs. baseline and SNP, †p < 0.01 vs. baseline

HF therapy improves hemodynamics and is associated with reductions in exhaled NO concentrations. Thus, elevations in exhaled NO may reflect a compensatory circulatory mechanism in HF, and exhaled NO may be an easily obtainable and quantifiable measure of clinical response to therapy in HF.

5:00

723-5 Modulation of Tumor Necrosis Factor α in Advanced Heart Failure With Cachexia Is Associated With Anabolic Effects

Richard V. Milani, Stefan Endres, Mandeep R. Mehra, Julia Cook, Andreas Eigler, Frank W. Smart, Carl J. Lavie, Joseph P. Murgo, Hector O. Ventura. *Ochsner Clinic, New Orleans, LA*

Tumor Necrosis Factor (TNFα) has catabolic effects which produce cachexia in laboratory animals and humans. Indeed, patients with advanced heart failure (aHF) who demonstrate elevated levels of TNFα often suffer from cachexia, a finding characterized by reduced body fat. The purpose of this double blind, randomized, placebo-controlled study was to evaluate the impact of modulation of TNFα synthesis on alterations in body fat indices using n-3 fatty acids. 14 patients with aHF were randomly allocated to active treatment (n-3 fatty acids, 6.4 gms/d; n = 7) vs. isocaloric placebo (n = 7). TNFα production at baseline and after 18 weeks was assessed using lipopolysaccaride monocyte stimulation.

	ΔTNFα synthesis	Δ% Body Fat
n-3 Fatty Acids	−59%	+9.5%
Placebo	+44%	−4.2%

Regression analysis revealed that ΔTNFα was inversely and significantly correlated with Δ% body fat (r value = 0.60, p = 0.02). *Conclusions:* 1) N-3 fatty acid supplementation in aHF results in significant reductions in

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