Conclusion: Unfavorable mitral valve anatomy represented by high mitral Echo score is the main cause of mitral restenosis following MBV.

1037-82 High Rate of Restenosis Following Coronary Angioplasty in Patients with Chronic Renal Failure

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Retrospective observational studies suggest a high rate of restenosis following coronary angioplasty in patients with end-stage renal disease (ESRD) undergoing chronic hemodialysis. A study which investigates the rate of restenosis in comparison to controls in respect to procedural variables and cardiovascular risk factors has not been performed.

In 20 patients on maintenance hemodialysis and in 20 age- and sex-matched patients without renal disease coronary stenosis was assessed by computer assisted quantitative coronary angiography before and after elective and primarily successful PTCA and at a follow-up coronary angiography after 6 months. Plasma concentrations of lipids and fibrinogen were measured before PTCA.

The rate of restenosis (stenosis > 50% luminal diameter) was 60% in patients with ESRD and 35% in controls. Procedural variables like the degree of stenosis before (69 ± 9 vs. 65 ± 6%) and after PTCA (37 ± 8 vs. 35 ± 11%) as well as the complexity of stenosis dilated did not differ between both groups. In patients with ESRD concentrations of fibrinogen (483 ± 101 vs. 330 ± 64 mg/dl, $p < 0.001$) and triglycerides (269 ± 163 vs. 207 ± 176 mg/dl, $p < 0.01$) were significantly elevated in comparison to controls, while concentrations of total cholesterol (262 ± 50 vs. 238 ± 39 mg/dl) LDL-cholesterol (191 ± 50 vs. 172 ± 38 mg/dl) and HDL-cholesterol (36 ± 15 vs. 42 ± 12 mg/dl) were comparable.

In this first controlled study a high rate of restenosis could be confirmed in patients with ESRD. Given the role of reactive thrombogenesis in the early phase following coronary angioplasty procoagulant factors like elevations of fibrinogen and triglycerides as a possible marker of impaired endogenous fibrinolysis may influence the process of restenosis in patients with end-stage renal disease.

1038 Novel Pharmacologic Therapies for Restenosis After PTCA

Wednesday, March 19, 1997, 9:00 a.m.–11:00 a.m.
Anaheim Convention Center, Hall E
Presentation Hour: 9:00 a.m.–10:00 a.m.

1038-70 Atherosclerosis Progression in Subjects with and without Post-Angioplasty Restenosis in QUIET

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The Quinapril Ischemic Event Trial (QUIET) randomized 1750 subjects within 72 hrs of successful PTCA to Quinapril 20 mg (Q) or placebo (P) for three years. The clinical endpoint was time to first cardiac ischemic event in the full cohort. Followup angiograms were performed in 453 subjects to assess atherosclerosis progression by QCA in non-intervened arteries. Restenosis (narrowing which was ≥ baseline pre-intervention) was also assessed visually at three years. Subjects who underwent "re-do" PTCA during the study were included in order to determine the total restenosis rate. Of the 453 angiographic substudy subjects, 164 (78 Q, 86 P, $p > 0.05$) experienced restenosis for a total rate of 36%. Sixty-one asymptomatic restenoses (13%) were found only at elective three year follow-up angiogram. Early symptomatic restenosis occurred in 103 subjects (23%). Progression, defined as a 0.40 mm diminution in minimum lumen diameter in a diseased, non-intervened artery, occurred as follows ($p > 0.05$ for all comparisons):

Angiographic Progressors

	Placebo (n = 229)	Quinapril (n = 224)	Overall (n = 453)
Restenosis (n = 164)	43/ 86 (50%)	35/ 78 (45%)	78/164 (48%)
No-Restenosis (n = 289)	64/143 (45%)	67/146 (46%)	131/ 28 (45%)

We conclude that: 1) Since atherosclerosis progression was similar in the restenosis and no-restenosis groups, the two processes appear to be pathophysiologically independent. 2) As there was no difference in restenosis rates between treated and placebo groups, the QUIET data suggests that even ACE-inhibitors with high tissue affinity do not significantly lessen restenosis.

1038-71 Local Administration of L-703,801 with a Composite Polymer Stent Reduces Platelet Deposition in Canine Coronary Arteries

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Systemic inhibition of platelet glycoprotein IIb/IIIa is effective in reducing 30 day and 6 month clinical events following high risk angioplasty. We compared the effect of L-703,801, a nonpeptide analog of tirofiban, administered locally with a drug eluting stent to that of standard metal stents on platelet deposition in canine coronary arteries. Ten mongrel dogs were given 325 mg of aspirin one day prior to stent implantation. The left anterior descending and left circumflex arteries were randomly assigned a standard 8 mm slotted metal tube stent or an 8 mm composite metal/polymer stent incorporated with L-703,801 (40% by weight). On-line quantitative angiography was use to size stents. Balloon to artery ratios of 1.2–1.3:1 were achieved with infation pressures of 10–14 ATM. Platelet deposition determined with ^{111}In-oxime was measured 2 hours after stent implantation. Platelet deposition on drug loaded stents was significantly less than metal controls: 3.43 (1.91, 3.75) vs. 7.87 (2.53, 11.3) platelets × $10^7/cm^2$, $p = 0.0166$. To determine the effect of non-drug loaded polymer, similar studies were performed on 14 animals. Composite stents without drug had slightly greater platelet deposition than metal controls but the difference was not significant: 6.71 (1.97, 12.0) vs. 4.46 (2.43, 17.8) platelets × $10^7/cm^2$, $p = 0.4326$. There was no difference in the proximal reference vessel diameter, the stent minimal lumen diameter or the stent to artery ratio. *Conclusion:* Local delivery of L-703,801 is effective in reducing 2 hour platelet deposition in the canine model of coronary stent implantation. This study is the first to show significantly reduced platelet deposition by a drug eluting stent compared to standard metal stents.

1038-72 Effect of Intracoronary ^{192}Irridium (^{192}Ir) on Late Quantitative Angiographic Outcomes After PTCA

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To evaluate the effect of gamma radiation on the late angiographic outcome after PTCA, we reviewed the serial angiograms of 21 patients (22 lesions) who received 18–20 Gy ^{192}Ir delivered via a 3 cm radioactive wire. Quantitative angiography (CMS-GFT) was performed using contrast-filled catheters before and after PTCA, 1–4 months (N = 12), and > 4 months (N = 20) later. Lesions were located in the LAD (50%), RCA (32%), and LCx (18%) arteries. Lesion length was 9.21 ± 3.77 mm. After PTCA, 2 patients had small pseudoaneurysms. Early (1–4 month) angiography showed 2 total occlusions and 2 (9%) new pseudoaneurysms (one < 2 mm). Serial quantitative angiographic findings included: *MLD = minimal diameter*

	Pre	Post	1 Month	6 Month
Normal, mm	2.90 ± 0.57	3.01 ± 60	2.95 ± 0.52	2.90 ± 0.60
MLD, mm	0.98 ± 0.52	1.94 ± 0.45	1.61 ± 0.89	1.75 ± 0.73
% Stenosis	66 ± 16	35 ± 13	46 ± 28	41 ± 24

Binary restenosis (≥ 50% follow-up diameter stenosis) occurred in 27% of lesions, including the 2 patients with early total occlusion. Late loss was 0.19 ± 0.78 mm; the loss index (late loss/acute gain) was 0.19. Ten (45%) lesions had an increase in lumen diameter during the follow-up period. *We conclude that* ^{192}Ir gamma radiation: 1) can be safely performed after PTCA without an increase in procedural complications, 2) is associated with infrequent (< 10%) development of late angiographic complications, and 3) appears to reduce the magnitude of lumen loss after PTCA (late loss = 0.19; loss index = 0.19) compared with conventional PTCA.

1038-73 Effectiveness of Probucol in Preventing Restenosis after Percutaneous Transluminal Coronary Angioplasty in Small Sized Coronary Arteries: A Subgroup Analysis of the Probucol Angioplasty Restenosis Trial (PART)

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To investigate the effectiveness of Probucol in preventing restenosis after PTCA in small sized vessels, we analysed the angiographic results of the PART study. One hundred and one patients were randomly assigned to Probucol or control group. In Probucol group, 1000 mg of Probucol was administered daily for 4 weeks prior to PTCA and continued until angiographic