1120-63 **Comparison of Intracoronary Bolus Injection and Intracoronary Continuous Infusion Methods for Inducing Hyperemia**

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Background: The method to induce coronary hyperemia with maximal and steady state is important to evaluate microvascular circulation by coronary flow reserve(CFR) and fraction flow reserve(FFR). We studied the method of induing hyperemia with intra-coronary(IC) adenosine bolus injection and continuous infusion.
Methods: CFR was evaluated in thirty-six coronary lesions(LAD 23, LCX 5, RCA 8) in 23 patients(male 18, mean 58±12 y; SA 2, UA 12, AMI 9) and FFR was evaluated in twenty lesions(LAD 13, LCX 6, RCA 1) in 10 patients(male 8, mean 60±11 y; SA 1, UA 7, AMI 2). To induce maximal coronary hyperemia, two methods were applied to same patient; Method A(IC adenosine bolus injection(RCA 24μg, LCA 48μg)) and Method B(IC adenosine continuous infusion(240μg/min). CFR was defined as a ratio of hyperemic(hAPV) to baseline APV(bAPV). FFR was defined as a ratio of mean aortic pressure to mean coronary pressure of distal part to lesion during maximal hyperemia. Hyperemic average peak velocity(hAPV, cm/sec), CFR and FFR were compared with paired t-test in two methods.
Results: All patients were tolerable during maximal hyperemic period. During maximal coronary hyperemia, heart rate and mean blood pressure were not different between two methods. Transient AV block occurred in 2 patients with method A and in 2 same patients with method B. The duration of maintenance of hyperemia was mean 24.6±5.4 seconds in method A and all patient except 2 patients were maintained stable hyperemia with method B during continuous infusion of adenosine. CFR and hAPV were significantly higher by using of method B than those of method A(3.4±2.1 vs. 2.9±1.2, p=.01 ; 52.5±23.4 vs. 45.7±19.9, p<0.01, respectively). FFR was significantly lower by using of method B than that of A(0.76±0.17 vs. 0.80±0.15, p<0.01).
Conclusion: Compared with intracoronary adenosine bolus injection method(24-48μg), intracoronary adenosine continuous infusion method(240μg/min) was more effective and stable for inducing maximal hyperemia.

1120-64 **High Left Ventricular Mass Does Not Limit the Utility of Fractional Flow Reserve for the Physiologic Assessment of Lesion Severity**

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Background: Fractional Flow Reserve (FFR) has been shown to be a useful invasive physiologic index of coronary lesion severity. Increased left ventricular mass (LVM) can impair microvascular reserve. However, whether increased LVM sufficiently impacts flow reserve to effect clinical FFR measurements is not known.
Methods: LVM was calculated from contrast left ventriculography in 84 patients using a modified Rackley method, which correlated well with LVM measured by cardiac MRI in 17 patients who had undergone both tests (r=0.80, p<0.001). The cohort was divided into normal and high left ventricular mass index (LVMI) groups based on published normal values. Cardiac risk factors and lesion FFR were compared in 22 vessels of patients with high LVMI to 62 angiographically matched lesions in vessels of patients with normal LVMI.
Results: LVMI was 84±21 g/m^2 and 126±21 g/m^2 in the normal and high LVMI groups, respectively. There were no differences in age (59 vs 58 yrs, p=ns), presence of diabetes (26% vs 27%, p=ns), hypertension (60% vs 73%, p=ns), dyslipidemia (56% vs 64%, p=ns) or angiographic LVEF (58% vs 62%, p=ns) between groups. Importantly, in lesions with similar angiographic characteristics, there was no difference in FFR between groups. (Table).

	Reference Diameter (mm)	Minimum Luminal Diameter (mm)	Percent Diameter Stenosis (%)	Lesion length(mm)	FFR
High LVMI*(n=22)*	3.3±0.5	1.3±0.6	61±13	14.2±7	0.79±0.12
Normal LVMI*(n=62)*	3.1±0.7	1.3±0.6	62±13	14.3±7	0.78±0.16
p-value	ns	ns	ns	ns	ns

Conclusion: FFR of lesions in patients with high LVM is no different to FFR of angiographically similar lesions in patients with normal LVM. These findings suggest that increased LVM should not limit the utility of FFR as a physiologic index of lesion severity.

1120-65 **Emboli Protection Improves Thrombolysis in Myocardial Infarction Perfusion Score in Saphenous Vein Graft Intervention**

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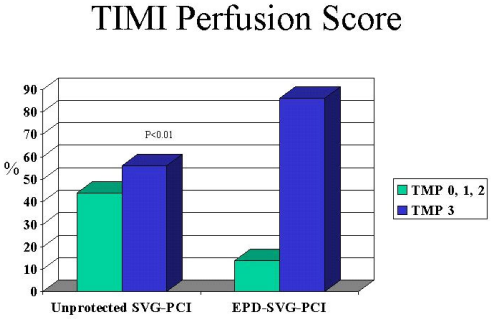
Background: Use of emboli protection devices (EPD) during saphenous vein graft percutaneous coronary intervention (SVG-PCI) has been proven to reduce major adverse cardiac events (MACE) specifically the composite of myocardial infarction, urgent target vessel revascularization, and death. However, the impact of EPD on the microcirculation using TIMI myocardial perfusion score (TMP) has not been fully characterized. We sought to analyze TMP in both unprotected- and EPD- SVG-PCI and its impact on 30-day MACE.
Methods: From August 2001 to December 2002, 305 patients had a SVG-PCI suitable for EPD; 247 (81%) had an angiogram appropriate for TMP evaluation. Of those, 49 (20%) had an EPD deployed during the coronary intervention. Both groups were similar regarding most demographic features, but use of GP IIb/IIIa inhibitors was more frequent in the EPD group (87% vs 74% P=0.007).
Results: TMP 3 score was obtained in 87% of the EPD group vs 56% of the unprotected SVG-PCI (P<0.01) (Figure 1). MACE was 4.2% in the EPD group vs. 8.1% in the unprotected SVG-PCI group (P=0.04). Unprotected SVG-PCI patients with TMP scores lower than 3 had a trend towards increased total post-procedural CK (177 U/L vs 133 U/L P=0.07), and CK-MB (21 ng/mL vs 6 ng/mL P=0.07).
Conclusions: EPD SVG-PCI improves TMP score when compared to unprotected SVG-PCI. This finding was associated with a decrease in post-procedural MACE.



1120-66 **Early Saphenous Vein Graft Failure: A Predictor of Poor Outcomes After Percutaneous Coronary Intervention**

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Background: While most saphenous vein grafts (SVGs) remain patent for approximately ten years, up to 15-20% of SVGs become occluded within the first year of implantation. The results following percutaneous coronary intervention (PCI) of early compared to late graft failure are not well known.
Methods: We identified 100 pts (121 lesions) with early (< 1yr) SVG failure and 200 pts (289 lesions) with late (> 1yr) SVG failure that underwent PCI. Pts were matched for hypercholesterolemia and diabetes. Data on procedure, post-procedure coronary angiography, and 1-year follow-up were analyzed. Angiographic measurements included reference size, final minimum lumen diameter (MLD), lesion length, and final diameter stenosis (DS).
Results: Early and late failure graft age was 6.0 ± 2.9 months and 105.4 ± 50.8 months respectively. The early group had smaller references (2.78± 0.81 vs 3.30± 0.76 mm, p< 0.01), smaller final MLD (2.33 ± 0.96 vs 2.91 ± 0.85, p< 0.01), and larger final DS (16.6 ± 24% vs 11.6 ± 18.1%, p= 0.03).
Procedural success was 96% in both groups; only 1 pt in each group had an in-hospital QMI, and 13.4% of early and 13.5% of late pts had a Non-Q MI (CKMB>5xNL). Stent use was not related to one-year target vessel revascularization (TVR) 29.8% in the stent group and 32.1% in the no stent group. However, the early SVG failure group had a significantly higher rate of TVR at one-year (38.6% vs 26.6%, p= 0.04). On multivariate analysis examining stent use, time of graft closure, and final DS; early graft closure was the strongest predictor of TVR (p= 0.06)
Conclusion: Despite a high rate of procedural success, 1-year TVR is higher in SVGs with early failure. Further investigation with novel interventional devices or techniques is necessary in this high risk population to eventually improve long term outcomes.

POSTER SESSION

1121

In-Stent Restenosis

Tuesday, March 09, 2004, 9:00 a.m.-11:00 a.m.
Morial Convention Center, Hall G
Presentation Hour: 9:00 a.m.-10:00 a.m.

1121-47 **Bone Marrow and Neural Crest Derived Cells Contribute to In-Stent Restenosis**

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Objective: In in-stent restenosis (ISR), a significant number of intimal mesenchymal cells remain unidentified. Also, there is ongoing controversy on the origin of neointimal cells. Therefore, the objective of the present study was to assess cellularity, cell type and origin of neointimal cells.