1162-132 Intracoronary Adenosine Potentiates Coronary Flow Inhomogeneity Produced by Intravenous Dobutamine Between Stenotic and Adjacent Normal Coronary Arteries

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Background: Dobutamine (Dob) and adenosine (Ado) are widely used agents for the performance of pharmacologic stress testing, usually in conjunction with imaging techniques. The purpose of this study was to provide insights to the physiologic mechanism involving coronary flow and hemodynamic changes associated with the use of these agents in patients with coronary artery disease.

Methods: We studied 20 stenotic (ST) and 15 adjacent normal (N) coronary arteries in 20 patients using paired (15 patients) or single (5 patients) Doppler Flowires. Measurements of coronary flow velocity (CFV) and hemodynamic parameters were taken at control, after intracoronary (ic) Ado, during incremental iv Dob infusion at doses 5, 10, 20, 30 and 40 μg/kg/min (mean peak dose 38 ± 4) similar to clinically performed Dob stress tests, and after ic Ado added on peak Dob stress (Ado on Dob).

Results: Distal to ST, average peak velocity (APV) increased significantly at an intermediate dose (20 μg/kg/min) of Dob (from 11 ± 5 to 16 ± 7 cm/s, 57 ± 56%, p < 0.001) and remained relatively unchanged thereafter to peak Dob stress (to 17 ± 7 cm/s) despite the continuous increase in rate-pressure product (RPP) (23 ± 21% and 64 ± 41% respectively) even with Ado on Dob (to 18 ± 7 cm/s). In N, APV increased significantly and continuously to peak Dob stress (from 20 ± 7.5 to 42 ± 10 cm/s, 126 ± 57%, p < 0.0001) exceeding the increase in RPP (64 ± 31%, p < 0.001) and there was a significant further increase with ic Ado on Dob (to 53 ± 13 cm/s, p < 0.001). Compared to ic Ado flow measurements prior to Dob infusion CFV reserve at peak Dob stress was slightly higher than that at ic Ado in ST (1.87 ± 0.9 vs 1.43 ± 0.5, p < 0.05) and did not change with ic Ado on Dob (1.94 ± 0.9) but it was significantly lower in N (2.26 ± 0.6 vs 2.99 ± 0.5, p < 0.001), increasing significantly after ic Ado on Dob (2.87 ± 0.7, p < 0.001). Suboptimal increase of APV at peak Dob stress in N could be accounted for by a depressed systolic but an optimal diastolic APV response.

Conclusion: During incremental iv Dob stress CFV reserve is exhausted at a lower than maximal Dob dose in ST but not in N coronary arteries. Peak Dob stress is associated with worsening supply/demand ratio in ST arteries but induces less coronary flow inhomogeneity due to suboptimal vasodilation in N arteries which can be potentiated by the addition of Ado during peak Dob stress. Our findings may explain the increased sensitivity reported for combined Dob and Ado stress echocardiography.

1162-133 Regional Increases in ^{18}F-2-Deoxyglucose (FDG) Uptake in Chronically Stunned Myocardium Are Dependent Upon Coronary Flow Reserve

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We previously demonstrated that pigs instrumented with a proximal left anterior descending (LAD) coronary stenosis for 3 months develop hibernating myocardium characterized by reductions in resting flow, function and increased FDG uptake. To define the temporal development of these adaptations we studied separate groups of pigs 1 and 2 months following instrumentation in the fasted, closed-chest, anesthetized state. Regional wall motion (ventriculography; 3 = normal, 0 = akinesis), relative subendocardial perfusion (LAD/Normal, microspheres) and relative subendocardial FDG uptake are summarized below.

	Flow (LAD/Normal)		Wall Motion	Relative FDG
	Rest	Adenosine		
1 Month	1.00 ± 0.04	0.58 ± 0.07	1.8 ± 0.2	0.9 ± 0.1
2 Months	1.06 ± 0.14	0.33 ± 0.09*	1.0 ± 0.2*	2.7 ± 0.6*

Mean ± SEM; *p < 0.05 vs. 1 month

Although wall motion was depressed, resting perfusion was normal, consistent with chronically stunned myocardium. Despite reductions in resting function, there was variability in relative FDG uptake that was related to the extent to which adenosine flow reserve was reduced. These data contrast with the reductions in resting flow and increased FDG uptake found at 3 months, and support the hypothesis that there is a time-dependent transition from chronically stunned to hibernating myocardium in this model. The development of increased FDG appears to precede the reduction in resting flow.

1162-134 "Paradoxical Shrinkage" of Coronary Arterial Wall may Contribute to Luminal Narrowing in Stable Angina but Not in Acute Myocardial Infarction

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This study was done to assess how local changes in vessel size, together with plaque volume and nature, determine luminal narrowing in coronary arteries in vivo. We performed intracoronary ultrasound (ICUS) in 54 patients (pts) with stable angina (SAP, 32 pts) or acute myocardial infarction (AMI, 22 pts) prior to PTCA or thrombolytic therapy. A 3.2F or a 2.9F ICUS catheter equipped with a 30 MHz transducer was used. Motorized pullback device (1.0 mm/sec) was applied to obtain sequential 2-D ICUS images. Off-line longitudinal reconstruction of ICUS images was performed to facilitate the determination of lesion sites and quantitative measurements. Plaque characteristics were categorized as soft, fibrous, calcified, and mixed. Measurements of the vessel and the lumen were performed at every 5 mm along 20 mm length and at reference sites. There were no differences between AMI and SAP in age, sex, height, and coronary risk factors. Soft plaque was more common in AMI (67% vs 27%, p < 0.05). The total vessel area at the lesion site was smaller in SAP than in AMI (13.9 ± 4.3 mm^2 vs 19.2 ± 7.0 mm^2, p < 0.05), and the measured vessel area was significantly smaller than the calculated vessel area (mean of proximal and distal) in SA (SA: -4% vs AMI: +22%, p < 0.05).

Conclusions: Plaque characteristics and volume of the culprit lesion were different between AMI and SAP. High degree organic stenosis responsible for SAP may be caused not only by the increase of atheroma volume but also by "paradoxical shrinkage" of coronary arteries.

1162-135 Arterial Remodeling in Left Main Coronary Disease: Proof From Serial Volumetric Intravascular Ultrasound Studies

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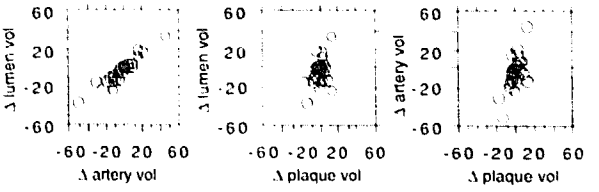
Arterial remodeling in de novo atherosclerosis has been inferred from nonsenal histopathologic and intravascular ultrasound (IVUS) studies. To confirm this finding, we performed serial IVUS studies (7.7 ± 2.4 mos apart) in untreated left main (LM) coronary arteries in 31 pts undergoing intervention of nonostial LAD or LCX lesions. All IVUS studies were performed with automatic transducer pullback @ 0.5 mm/sec. An automatic contour detection algorithm was used to measure artery, lumen, and plaque (artery - lumen) volumes (vol, mm^3) Over 7.2 ± 2.5 mm long segments. Baseline plaque burden (plaque/artery vol) was modest (33 ± 11%). Δ lumen vol correlated with A artery vol (r = 0.851, p < 0.0001), but not with Δ plaque vol (r = 0.132, p = 0.48).



Δ artery vol correlated with Δ plaque vol (r = 0.409, p = 0.0225). An increase in artery vol resulted in an increase in lumen vol despite an increase in plaque vol while a decrease in artery vol resulted in a decrease in lumen vol despite a decrease in plaque vol.

We Conclude: Serial IVUS analysis confirms both positive and negative remodeling in de novo LM disease. Changes in lumen vol resulted from changes in artery vol, not changes in plaque vol. This explains why lumenographic methods are limited in quantifying changes in atherosclerosis.

1162-136 Functional Relevance of Minimal Coronary Arteriosclerosis Myocardial Diastolic and Systolic Function During Stress-Echocardiography

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Background: Angiographically minimal manifestations of coronary arteriosclerosis (MA; severity of stenosis <50%) are accompanied by endothelial dysfunction resulting in dynamic coronary obstruction to various stimuli like