effects of chronic oral administration of quinapril (40 mg/day), captopril (150 mg/day) and placebo on vascular angiotensin (Ang) I conversion in humans.

Methods: Patients (n = 187) scheduled for coronary bypass surgery were randomized at least 7 days before surgery. Segments of the internal mammary artery were obtained and contracted to increasing doses of Ang I and II (0.1 nM to 1 μM) in organ baths. Ang I conversion was defined as (1) the difference between pEC50 of the dose-response curves to Ang I and II and (2) as the area between curves (ABC) of Ang I and II.

Results: Baseline characteristics were comparable between groups. Quinapril and captopril treated patients had a comparable and significant reduction in mean blood pressure compared to placebo (p = 0.04).

	Quinapril	Captopril	Placebo
delta pEC50	1.0 ± 0.12*	0.86 ± 0.1	0.04 ± 0.1
ABC	91 ± 8*	84 ± 4	67 ± 8

Mean ± SEM, * p < 0.05 (quinapril compared to placebo)

Conclusion: This is the first randomized study in patients showing tissue effects of ACE-inhibitors after pretreatment with adequate clinical doses. The results suggest functional differences between quinapril and captopril.

1090 Coronary Artery Disease II

Tuesday, March 31, 1998, 9:00 a.m.–11:00 a.m.
Georgia World Congress Center, West Exhibit Hall Level
Presentation Hour: 9:00 a.m.–10:00 a.m.

1090-6 Segmental Analysis of Coronary Artery Lesions Using 3-dimensional Echocardiography in Comparison With Coronary Angiography: A Clinical Semiquantitative Study

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Stenotic lesions of coronary artery (CA) are of clinical predictive value for ischemic heart events. The accuracy of transesophageal 3-dimensional echocardiography (3DTEE) in defining the severity of CA stenosis is not known.

Purpose: To analyze the quantitative potential of 3DTEE in defining moderate to severe CA stenosis.

Methods: A group of 20 subjects with CA angiography (A) less than 3 months scheduled for routine or intra-operative TEE was selected. 3DTEE was performed using a 5 MHz transducer with a integrated on-line 3DE acquisition system. The proximal CA was divided into 5 segments including left main (1), anterior descending (1), circumflex (2) and right (1) CA. CAA and 3DTEE were analyzed segment-by-segment by independent observers and a score of 0–3 was given to each segment according to none, mild (<50%), moderate (50–90%) and severe (>90%) stenosis located. A score was given to each patient according to distribution pattern of the stenotic lesions defined as none (0), localized (1) or diffusod (2).

Results: 11 of 20 pts had CA stenosis of proximal segments by CAA (3 localized, 8 diffused). A total of 85 segments of CA were observed by 3DTEE in all 20 subjects. Mild, moderate and severe stenotic segments were 15, 9 and 5 by 3DTEE and 14, 13 and 5 by CAA. Agreement (4 × 4 table) between these two methods was 80%. The sensitivity, specificity, positive and negative predictive value and general accuracy of 3DTEE for detecting moderate to severe CA stenosis were 61%, 95%, 79%, 90% and 88%, respectively.

Conclusion: 3DTEE provides an alternative specific method for detecting moderate to severe stenosis of proximal CA segments. In addition, 3DTEE can provide both longitudinal and cross-sectional views of the CA and plaques.

1090-7 Dynamic Systolic Coronary Blood Flow Reversal in Patients With Hypertrophic Cardiomyopathy: Relationship to Provokable Outflow Tract Obstruction

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Hypertrophic cardiomyopathy (HCM) is characterized by variable physiologic phenotypes, with a subset of patients showing only provokable left ventricular outflow tract (LVOT) obstruction. Unusual and variably abnormal patterns of resting coronary blood flow have been reported in patients with HCM. The relationship between dynamic LVOT obstruction and phasic coronary blood flow has not been examined. Therefore, we studied coronary flow velocity (0.014" Doppler guidewire) in 13 arteries (7 LAD, 4 CX, 2 RCA) in 8 patients with HCM before and during Valsalva maneuver at catheterization. Six patients had no resting systolic LVOT gradient (mean resting gradient = 5 ± 14, range = 0–50 mmHg). At peak Valsalva strain, the LVOT systolic gradient increased to 56 ± 23 mmHg (range 18–100 mmHg). Phasic coronary flow velocity pattern was normal in all arteries at rest. At peak Valsalva, there was reversal of systolic flow and a significant decrease in diastolic flow velocity in all arteries (see Table; ΔP = gradient, mmHg; SPV = systolic peak velocity, cm/s; DPV = diastolic peak velocity, cm/s; SPVi = SPV integral, units; DPVi = DPV integral, units; values are mean ± SD).

	LVOT ΔP	SPV	SPVi	DPV	DPVi
Rest	5 ± 14	25 ± 17	5 ± 4	40 ± 15	18 ± 6
Valsalva	56 ± 23	−24 ± 13	−3 ± 1	34 ± 12	13 ± 5
p	<0.0001	<0.0001	<0.0001	0.05	0.01

Systolic coronary flow velocity reversal was related to the dynamic LVOT gradient with Valsalva (r = 0.65, p = 0.01).

Conclusion: Dynamic pathophysiologic alterations in left ventricular hemodynamics seen in HCM are associated with dynamic changes in coronary blood flow.

1090-8 Injection Rate Minimally Affects the TIMI Frame Count

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Background: The Corrected TIMI Frame Count (CTFC) is a clinically important index of coronary artery blood flow. The goal of this study was to determine whether injection rate affects CTFC.

Methods: Dye injection rates were recorded during 361 *hand* injections to characterize the 10th, 50th, and 90th %-tiles for human angiographers. 273 ECG-gated *power* injections were then performed using a MedRad V power injector to deliver dye randomized to these 10th, 50th, or 90th %-tiles. The injection rate group and actual injection rate delivered (ml/sec) were recorded. Cineangiograms were analyzed for CTFC, proximal normal reference lumen diameter, and the presence of >50% stenoses (disease).

Results: In univariate analyses, CTFC was significantly affected by diameter (p < 0.0001), and disease (p < 0.001), but not injection rate group (p = 0.43) or actual injection rate (p = 0.94). Multivariate analysis revealed: CTFC = 20.9 frames + 4.1 frames × diameter (mm), (p < 0.001) + 4.6 frames if diseased, (p < 0.001) −1.6 frames if 50th %-tile injection rate, (p = 0.31) −1.9 frames if 90th %-tile injection rate, (p = 0.23) (p-values for 50th and 90th %-tiles represent comparison with 10th %-tile injection rate group, the dummy variable) (p < 0.0001 for model, r = 0.39).

Conclusion: Proximal lumen diameter and presence of >50% stenoses independently and significantly affect the Corrected TIMI Frame Count. Injection technique minimally impacts the Corrected TIMI Frame Count: an increase in injection rate from the 10th to 90th %-tile tends to decrease the CTFC by less than two frames.

1090-9 Coronary Flow Reserve in the Contralateral Artery Increases After Successful Coronary Angioplasty

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Background: Coronary flow reserve (CFR) is reduced in remote regions supplied by angiographically normal arteries. Recent studies have found that myocardial flow reserve is impaired in the myocardium in the perfusion area of a vessel with non-critical stenosis supplying collaterals to other segments. We, therefore, tested the hypothesis that CFR in the grossly normal contralateral artery could increase after successful angioplasty (PTCA).

Methods: We studied 31 patients (pts), 55 ± 2 years old, having stable angina and single artery disease, undergoing PTCA of the right coronary or the left anterior descending artery. CFR of the diseased and the contralateral artery was estimated using a Doppler flow guidewire, by giving ic adenosine, before and 15 min after the end of PTCA. All the procedures were successful and in 19 pts intracoronary stent was placed.

Results: (mean ± standard error)

	Before PTCA	After PTCA
Blood pressure (mmHg)	98 ± 3	95 ± 3
Heart rate (beats/min)	74 ± 1	76 ± 2
CFR of the contralateral artery	2.4 ± 0.1	2.8 ± 0.1*
Baseline APV (cm/sec)	21 ± 2	12 ± 1*
Maximum APV (cm/sec)	47 ± 3	34 ± 3*

*: p < 0.05; APV: average peak velocity;

The contralateral CFR after PTCA increased by 0.8 ± 0.1 in 11 pts with visible collaterals before PTCA and by 0.3 ± 0.1 in the remaining pts (p < 0.05). CFR of the diseased artery after PTCA was 2.2 ± 0.1.

TUESDAY POSTER