ratio), the cholestanol (Ch) and plant sterol (PS)/C ratios, two sterol groups reflecting synthesis and absorption of C, respectively, and fasting blood glucose were related to CE in Finnish simvastatin (n = 435) and placebo (n = 432) subgroups of 4S.

Results: The relative risk of CE was significantly reduced by simvastatin in the lowest (risk ratio for Ch 0.623;95% CI 0.395–0.982) but not in the highest quartile (risk ratio 1.166;95% CI 0.791–1.72). The relative risk was 2.2 times higher (P < 0.01) in the lowest than the highest quartile. PS and especially IS were less consistent predictors. Increasing basal Ch and PS quartiles were associated with decreasing IS, triglyceride, BMI and blood glucose (without or with diabetes), and increasing HDL cholesterol quartiles. Simvastatin-treated subjects with recurrent CE was the only subgroup not reducing blood glucose with increasing Ch quartile.

Conclusion: Coronary subjects with high basal absorption and low synthesis of cholesterol are not benefitted by reduced recurrence of coronary events during simvastatin treatment.

1116-8 Lesion Progression, Regression, and Stability With Fluvastatin in the Lipoprotein and Coronary Atherosclerosis Study

C.M. Ballantyne, J.A. Herd, J.K. Dunn, L.L. Ferlic, A.M. Gotto, Jr.. *Baylor College of Medicine, Houston, TX, USA*

Plaque rupture is thought to be the critical event leading to unstable angina and MI, but most plaque ruptures are asymptomatic and lead to lesion progression (prog). Angiographic trials with statins have shown clinical event reductions greater than the modest average Δ minimum lumen diameter (ΔMLD). Of 1215 lesions in 340 pts, only 182 (15%) lesions had prog as defined by MLD decrease >0.4 mm, and only 63 (5%) had regression (regr) as defined by MLD increase >0.4 mm; the remaining 80% were stable. Fewer fluvastatin (FL) pts categorized by maximal ΔMLD as defined above had prog (54 pts with 73 lesions with prog) than placebo (73 pts with 111 lesions with prog), and FL significantly improved the distribution of pts among categories ($p \approx 0.04$) as shown below, with small differences in mean ΔMLD within each category.

	Prog		Stable		Regr	
	n	avg ΔMLD	n	avg ΔMLD	n	avg ΔMLD
FL	54	0.28	92	+0.02	25	+0.26
Plac	73	0.29	82	0.01	14	+0.23

Other factors that affected the distribution of pts among categories by multinomial logistic regression analysis were baseline HDL-C, apo A-I, apo C-III:B, and Lp(a). FL reduced the number of pts with prog by 27%, similar to the event reduction in LCAS.

Conclusion: Because most lesions are stable, mean ΔMLD may be a less useful endpoint than lesion progression/regression.

1116-9 The LIPID Trial: Impact of Lipid Lowering Therapy With Pravastatin on the Risk of Stroke

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Epidemiological and recent clinical trial data support the potential value of cholesterol lowering in reducing the risk of ischemic stroke. The LIPID trial was designed to assess the long term effects of pravastatin versus placebo on coronary heart disease (CHD) mortality in 9,014 patients with CHD and average cholesterol levels. Secondary aims included the effect of treatment on total stroke and on non-hemorrhagic stroke. Trial patients had a baseline total cholesterol of 4.0–7.0 mmol/L (155–271 mg/dl) and a history of acute myocardial infarction or hospitalization for unstable angina. After an interim analysis showing a total mortality reduction with pravastatin ($p < 0.003$), the trial is closing early with final analysis to occur after September 30, 1997 when patient visits have been completed.

As of August 31, 1997 there have been 384 strokes reported in 344 patients. All reported strokes are classified blinded to treatment by an independent stroke assessment committee (including neurologists) as hemorrhagic, ischemic (large artery, lacunar, cardio-embolic, unknown), or of unknown type. Each stroke requires a history of sudden onset of focal neurological deficit lasting >24 hours. Ischemic stroke also requires no evidence of hemorrhage on CT/MRI brain imaging or autopsy. In the first 94 cases reviewed of confirmed stroke, 64 were ischemic, 10 hemorrhagic and 20 unknown type. The LIPID trial will provide substantially more information on the potential benefit of cholesterol lowering treatment on stroke reduction suggested from earlier trials.

1117 Myocardial Performance

Tuesday, March 31, 1998, Noon–2:00 p.m.
Georgia World Congress Center, West Exhibit Hall Level
Presentation Hour: Noon–1:00 p.m.

1117-23 Effect of Exercise on Left Ventricular Systolic Twist: An Echocardiographic Study in Normal Subjects

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Background: Left ventricular (LV) twist or counterclockwise rotation of the LV during systole is considered an important mechanism of both normal ejection dynamics and generation of restoring forces responsible for diastolic suction. Alterations in twist have been reported at rest in conjunction with depressed ventricular function. However, the effects of exercise on twist in normal humans has not been assessed.

Methods: Ten normal subjects underwent symptom limited treadmill stress tests to exhaustion. Two-dimensional echocardiography was performed prior to and immediately after exercise. End-systolic twist was determined by measuring rotation of the anterolateral papillary muscle about the center of the ventricle in a transthoracic short-axis view. Data are presented as mean ± SD.

Results: Subjects exercised for 13.4 ± 5.5 minutes. End systolic volume decreased significantly (25.72 ± 6.64 ml/m^2 vs. 16.80 ± 6.34 ml/m^2; $p = 0.0006$) with a corresponding increase in stroke volume (47.86 ± 9.35 ml/m^2 vs. 53.47 ± 11.02 ml/m^2; $p = 0.039$) and ejection fraction (0.65 ± 0.05 vs. 0.76 ± 0.06; $p < 0.0001$). LV twist increased in all subjects (6.8 ± 4.5° vs 14.7 ± 6.6°; $p < 0.0001$).

Conclusions: LV twist can be measured in patients undergoing stress echocardiography. The magnitude of LV twist increases in normal subjects in response to exercise. Thus, twist may be a mechanism of both augmentation of contractile mechanics and maintenance of diastolic filling during stress.

1117-24 Noninvasive Quantification of Intraventricular Pressure Gradients and Flow Propagation From Color Doppler M-Mode Relates to Invasive Pressures in an Ischemic Canine Model

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The purpose of the study was to investigate the relationship between noninvasive color M-mode flow propagation and pressure gradients, and invasive measured pressure changes in the left ventricle (LV) in an ischemic dog model.

Methods: 5 open-chest anesthetized dogs were instrumented by a multisensor high fidelity catheter; one sensor in left atrium (LA), one at base and one at apex. Simultaneous pressure and Doppler recordings were obtained during baseline and ischemia induced by LAD occlusion. The color coded Doppler velocity map provides spatiotemporal velocity distribution along a streamline from LA into LV, and derivatives can be directly obtained to calculate pressure gradients the Euler equation. Automated eigenvector analysis was used to compute the principal components of the E-wave, as an ellipse, with velocity weighted mean in space and time, spatial length and temporal width of the E-wave, and the angle of rotation (ϕ).

Results: The intraventricular pressure gradient obtained from catheter (IVc) correlated well with the one estimated from Doppler (IVd) ($r = 0.71$, $p < 0.01$). IVc decreased from 1.06 ± 0.10 to 0.67 ± 0.22 mmHg (ns), and IVd from 0.84 ± 0.11 to 0.61 ± 0.18 mmHg (ns) during ischemia. Tau increased from 73 ± 5 to 125 ± 20 ms ($p < 0.05$). ϕ, which is similar to flow propagation velocity, increased from 0.7 ± 0.4 to 3.5 ± 0.8° ($p < 0.05$). IVc and IVd correlated with spatial components of the E wave, while tau correlated with temporal components. Thus, distribution of velocities in time reflects changes in relaxation, while distribution in space reflects pressure changes.

Conclusion: Noninvasive quantification of IV pressure gradients and flow propagation from color Doppler M-mode is feasible; parameters correlate with invasive pressure measurements.

TUESDAY POSTER