and pre and post each apheresis session using routine laboratory assays. Features of calcified coronary plaque burden (Agatston Score, calcified plaque vol-ume, calcified plaque density) were measured using ECG- triggered fast CT at baseline and at the end of the study period. The effects of decreased blood lipoprotein exposure on coronary calcified plaque were evaluated. On average, total cholesterol, LDL- cholesterol, triglycerides and Lipoprotein (a) were substantially lowered as compared to baseline (52.9%, P<0.0001; 64.5%, P<0.0001; 48.7%, P<0.004 and; 45.3%, P<0.05), respectively. In contrast, mean HDL Cholesterol increased by 13.9% (P<0.07). Concomitantly the Agat-ston Score decreased by 26.1±14.3% (213±203, P<0.03), calcified plaque volume was reduced by 23.4±14.8% (186±199 mm3, P<0.03) and calcified plaque density increased by 17.2±10.7% (40±32 HU, P<0.01). The increase of plaque density was correlated to the reduction of total cholesterol levels (r=0.73, P<0.04).
Conclusion: Coronary calcified plaque volume decreases and mean calcified plaque density increases during long term maximal lipid lowering intervention. This regression potentially depends on the extent of lipid lowering and can be tracked quantitatively by computed tomography.

1107-88 Statin Therapy but Not Exercise Inhibits Rates of Progression of Coronary Calcification

Matthew J. Budoff, Nisha Agrawal, David S. Reiss, Tej Naik, Thuy Le, Kadisha Mayet, *Harbor-UCLA Research and Education Institute, Torrance, California.*

Background: Coronary artery calcification (CAC) by Electron Beam Tomography (EBT) is linearly associated with atherosclerotic plaque burden. The influence of different therapies to influence EBT calcium progression has not been well described.
Methods: The aim of this study was to determine the changes in CAC, under the influence of several interventions, as measured by electron beam tomography in consecutive patients presenting in our laboratory for follow-up. To evaluate the rates of progression, we evaluated 555 patients (mean age 59 +/-9 years, 78% men). The interscan period was 3.0 +/- 1.6 years. EBT scores were calculated using the Agatston method. The results were reported in % EBT score change/year. Due to high variabilities in the very low scores, patients with baseline scores >10 were included. Multivariate analysis was used to assess the independent effects of different therapies or conditions.
Results: Mean calcium score in this cohort was 257, and increased by 29.5 points per year. The mean rate of progression of the entire cohort was 18+/-7%. 241 patients were treated with statin therapy during the entire interscan period, with mean progression of 13+/-11%. In the 89 patients with high cholesterol who were not taking therapy, the rate of progression was 29+/-12% per year. There was a significant difference between the rates of progression between the groups (p=0.005). This study demonstrated statin therapy to be a significant, independent predictor of the progression of coronary calcium (odds ratio [OR] 2.41; 95% confidence interval [CI], 1.29, 4.75). Patients reporting regular exercise (n=334) had a rate of progression of 16+/-8%, while persons reporting no exercise had progression rates of 19+/-9% per year (p=n.s.). No cardiovascular risk factors were related to progression of plaque in the multivariate model.
Conclusion: Statin therapy, but not exercise, inhibited CAC progression. The prognostic significance of increasing CAC plaque remains to be determined. However, given the association of increased atherosclerosis with increased CAC, use of cholesterol-lowering therapy should be considered in patients with CAC to slow plaque progression.

1107-89 Change in Calcified Plaque Burden in Relation to Aggressiveness of Treatment and Baseline LDL Cholesterol Levels

Harvey S. Hecht, S Mitchell Harman, *Heart and Vascular Institute, Morristown, New Jersey, Kronos Research Institute, Phoenix, Arizona.*

To determine 1) whether or not progression of calcified plaque burden in the primary prevention population is significantly reduced by more aggressive LDLC lowering and 2) whether treatment will benefit pts with NCEP higher risk initial LDLC more than lower risk pts, 172 asymptomatic men (137) and women (35) without clinical CAD were evaluated by serial Electron Beam Tomography (EBT), before and 1.2±0.3 yrs after intitiation of lipid therapy. Statins were used in 163, niacin in 98, fibrates in 2, and statin+niacin in 65. Percent change/yr in EBT volume (V) and score (S) were compared in pts achieving final (F) LDLC80mg/dl and in pts with initial (I) LDLC >130 vs <130 mg/dl (Table). Conclusions: 1)After 1.2 yrs of treatment, there were no differences in calcified plaque burden progression in pts achieving LDLC80 mg/dl; i.e. lower was not better, and plaque progression was similar with initial LDLC<130 vs >130mg/dl; i.e. in pts defined as lower vs higher risk by NCEP. 2) A longer treatment period may be needed to produce differences between groups.

Changes in EBT Calcium in Relation to Risk (Initial LDLC) and Response to Treatment (Final LDLC)

Final LDL	F >80	F*80	p value	Initial LDL	I>130	I*130	p value
n	74	98	-	n	82	87	-
I-LDLC	138±46	122±28	0.007	LDLC	156±31	104±21	<0.001
I-EBT S	380±390	513±698	0.07	I-EBT S	453±501	592±829	0.16
F-LDLC	99±18	64±11	<0.001	F-LDLC	85±23	73±19	<0.001
I-EBT V	307±315	407±544	0.06	I-EBT V	353±381	468±646	0.17
%change S/yr	19.5±45.3	12.2±29.8	0.20	%change S/yr	12.8±23.2	16.6±46.6	0.52
%change V/yr	15.1±35.8	12.3±28.1	0.57	%change V/yr	11.8±24.0	14.5±36.8	0.57

1107-90 Comparison of Statin Versus Statin+Niacin Treatment in Primary Prevention: Effect on Change in Calcified Plaque Burden

Harvey S. Hecht, S Mitchell Harman, *Heart and Vascular Institute, Morristown, New Jersey, Kronos Research Institute, Phoenix, Arizona.*

The effects on calcified plaque burden by Electron Beam Tomography (EBT) of statin therapy (ST) to lower LDLC and statin+niacin therapy (ST+N) to treat combined LDLC and HDLC/Trig abnormalities were compared in 162 asymptomatic patients without clinical CAD. EBT score (S) and volume (V) were obtained before and after a mean of 1.25 yrs of ST (n=78) or 1.18 yrs of ST+N (n=84) therapy. Except for HDLC in the S group, lipid and EBT values all changed significantly after treatment (Table). Final LDLC was lower in the ST+N group (p<.03) but final HDLC and Trig did not differ significantly. There were no significant differences between groups in the change in LDLC, but changes in HDLC (p<0.0001) and Trig (p<0.006) were greater in the ST+N group. There were no differences between ST and ST+N in % change in EBT-S/yr (13.4 vs 17.2, p=0.9) or % change in EBT-V/yr (11.2 vs 16.4, p=0.77). Conclusions: ST and ST+N therapies 1) addressed different lipid profiles and produced significantly improved lipid values, and 2) did not differ in terms of rates of calcified plaque progression after 1.2 yrs of treatment.

Changes in Lipid and EBT Values in Patients on Statin and Statin + Niacin

	Statin			Statin + Niacin		
	Baseline	Final	p value	Baseline	Final	p value
LDLC	134±41	83±23	<0.001	129±30	73±18	<0.001
HDLC	58±17	60±18	0.21	44±12	54±14	<0.001
Trig	119±71	95±57	<0.001	170±121	109±63	<0.001
EBT-S	449±698	507±825	0.004	484±486	552+563	<0.0001
EBT-V	359±564	399±638	0.01	387±383	434±424	<0.001

POSTER SESSION

1108 Hypertension: Left Ventricular Hypertrophy

Monday, March 18, 2002, Noon-2:00 p.m.
Georgia World Congress Center, Hall G
Presentation Hour: Noon-1:00 p.m.

1108-69 Regression of Echocardiographic Left Ventricular Hypertrophy in Relation to Resolution Versus Persistence of Electrocardiographic Strain After One Year of Antihypertensive Therapy: The LIFE Study

Peter M. Okin, Richard B. Devereux, Vittorio Palmieri, Jennifer E. Liu, Kristian Wachtell, Markku S. Nieminen, Sverker Jern, Lasse Oikarinen, Matti Viitasalo, Sverre E. Kjeldsen, Stevo Julius, Bjorn Dahlof, for the LIFE Study Investigators, *Cornell Medical Center, New York, New York.*

Background: Presence of the ECG strain pattern (ST depression and T-wave inversion in leads V5 and/or V6) at LIFE study baseline was associated with greater left ventricular mass index (LVMI) and a higher prevalence of LV hypertrophy (LVH). However, the relation of resolution vs persistence of ECG strain to changes in LVM and LVH over time is uncertain. **Methods**: 597 patients (pts) with ECG LVH by either Cornell product and/or Sokolow-Lyon voltage, enrolled in the LIFE echocardiographic substudy, were examined at study baseline and after 1-year of blinded therapy with either losartan or atenolol based regimens. Echocardiographic LVH was defined by LVMI >116 g/m^2 in men and >104 in women. **Results**: ECG strain was absent at baseline and 1-year (S-/-) in 511 pts, present on both ECGs (S+/+) in 47 pts, and was present at baseline but had resolved by 1-year (S+/-) in 39 pts. At study baseline, compared with S-/- pts, S+/+ and S+/- pts had similarly high prevalences of LVH, but the LVH was more severe, with greater LVMI, in S+/+ than S+/- pts (p=.038); S-/- pts had significantly lower LVMI and prevalence of LVH. After 1-year, S+/+ pts had the greatest LVMI and prevalence of LVH, whereas S+/- pts had lower LVMI and prevalence of LVH. (*p<.001 vs baseline)

Variable	Strain-/- (n=511)	Strain+/- (n=39)	Strain+/+ (n=47)	p value
LVMI at baseline (g/m^2)	120±23	136±25	148±38	<.001
LVMI at 1-year (g/m^2)	107±20*	115±25*	133±31*	<.001
LVH at baseline (%)	66	90	87	<.001
LVH at 1-year (%)	39*	54*	75	<.001

Conclusions: In the setting of aggressive antihypertensive therapy and decrease in blood pressure, persistence of ECG strain identifies a subgroup of pts with greater 1-year LVMI and LVH. In contrast, resolution of strain was associated with a marked reduction in the risk of LVH at 1-year.