LEFT VENTRICULAR GEOMETRY AND CARDIAC RISK FACTORS DEFINE HIGH AND LOW RISK SUBGROUPS AMONG ESSENTIAL HYPERTENSIVES.

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Conventional treatment of mild hypertension achieves only modest reduction in morbidity and mortality. Better identification of hypertensive pts at high and low risk is desirable. We followed 250 essential hypertensives (66% men) for 10.2±2.0 years after an index echocardiogram used to characterize LV geometry by LV mass index (LVMI) and relative wall thickness (RWT). 147 pts had normal cardiac geometry (LVMI≤125g/m^2, RWT<0.45), 34 had concentric remodelling (LVMI<125g/m^2, RWT>0.45), 40 had eccentric hypertrophy (LVMI≥125g/m^2, RWT≤0.45) and 29 had concentric LV hypertrophy (LVH) (LVMI≥125g.m^2, RWT≥0.45). All-cause mortality was highest for pts with concentric LVH (24.1%), lowest for those with normal geometry (0.7%) and intermediate for pts with concentric remodelling (5.9%) and eccentric LVH (10.0%). Cardiovascular events (cardiac death, myocardial infarction, stroke, angina pectoris and coronary revascularization) were also most frequent in pts with concentric LVH (31%) and least in pts with normal geometry (10.8%); in contrast, there was less separation in risk between pts with and without electrocardiographic LVH (20.9% vs 15.5%). Non-smokers with serum cholesterol <240mg/dl, diastolic blood pressure≤105mmHg and normal cardiac geometry were the lowest risk subgroup with no cardiovascular deaths and only 4 morbid events in 64 pts over 10 years (6.3%, 0.6 non-fatal events/100 patient years). These data suggest that cardiac geometry determined by echocardiography stratifies risk among pts with hypertension and can be used with conventional risk factors to determine which pts are most and least likely to benefit from antihypertensive treatment.

RELATIONSHIP OF END DIASTOLIC VOLUME TO EXERCISE PERFORMANCE IN ASYMPTOMATIC HYPERTENSIVE SUBJECTS.

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We have previously noted that normal pts do not decrease end diastolic volume (edv) with exercise (ex), but that some hypertensive pts do. To determine the significance of this observation we performed cardiopulmonary ex testing on 128 hypertensive pts with <5% probability of coronary disease. The differences between 101 pts with normal (Gp I) vs 27 pts with abnormal (Gp II) edv response to ex follow: (values are mean±1 standard deviation, SV:stroke volume,VO2:oxygen uptake,EF:ejection fraction)

	Group I	Group II	p
exHeart rate	147±19	145±17	ns
exSBP(mmHg)	195±27	192±23	ns
restEF	.60±.08	.55±.08	<.01
exEF	.62±.10	.61±.10	ns
restSV(ml)	81±20	85±19	ns
exSV(ml)	99±28	78±19	<.001
peak watts	114±39	93±33	<.001
exVO2(ml/kg)	33±22	21±17	<.05

There was no difference in peak filling rate or time to peak filling rate. We conclude that group II pts have a failure of recruitment of preload with ex that is associated with an inadequate SV and CO response and impaired ex performance. This is not explained by differences in systolic or diastolic function.

CORONARY AUTOREGULATION IN HYPERTENSION: CORRELATION WITH LEFT VENTRICULAR MASS.

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Aim of the study was to evaluate if the flow(F)/pressure (P) relationship in the coronary circulation is different in normotensive(N) and in hypertensive(H)pts and, whithin these, in subjects with normal and augmented left ventricular mass index(LVMI). In 9 N subjects and in 14 H pts(7 with normal and 7 with increased LVMI,>135 g/m^2) the coronary perfusion P(aortic diastolic P) was gradually reduced(from 85 to 60 mmHg in N and from 115 to 70 mmHg in H pts)through iv infusion of nitroprusside(scaled doses from 0.6 µg/kg/min to 5.5 µg/kg/min). Sinus coronary blood F(thermodilution) was measured in basal condition and at each step (5 mmHg diastolic P reduction) during the infusion. The P/F relationship curves obtained for each pt showed that in N coronary F at first increased and then reduced towards values of 60 mmHg; in all H pts the initial increase of coronary F was not observed and in pts with normal LVMI the F was steady up to the prefixed P value of 70 mmHg, while in pts with increased LVMI the F began to decrease at 80 mmHg reaching, at 70 mmHg, values 40% lower than those observed in pts with normal LVMI. These patterns were not related to differences in oxygen myocardial consumption since changes of HR were similar in the 3 groups. Thus, increased LVMI appears,also in humans,to affect the coronary P/F relationship: therefore in pts with augmented LVMI a reduction of aortic diastolic P below 80 mmHg may induce an unfavorable decrease of coronary perfusion.

Tuesday, March 20, 1990
2:00PM–3:30PM, Room 43
Mechanisms of Arrhythmias

THE FAMILIAL LONG QT SYNDROME STUDY: PROSPECTIVE CARDIAC AND GENETIC INVESTIGATION INVOLVING 292 FAMILIES.

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This study was established in 1979 to elucidate the cardiac and genetic aspects of the familial Long QT Syndrome (LQTS) - a disorder with episodic syncope and sudden death. To date, we have enrolled 292 LQTS probands (first family member diagnosed as affected [QTc >0.44sec]) and 1882 living family members (561 affected, 748 unaffected [QTc ≤ 0.44sec], and 573 without ECGs). Probands were younger (21±15 yrs vs 33±23), had more female predominance (68% vs 60%), a higher frequency of prior cardiac syncope (82% vs 19%), slower heart rate (71 bpm vs 79), and longer QTc values (0.52±0.07sec vs 0.48±0.04) than affected family members. Risk factors significantly ($p < 0.05$) associated with syncope/death during average 54 mo./pt follow-up in 853 pts with QTc > 0.44sec were: history of syncope (odds ratio 24.0), mitral valve prolapse (2.7), QTc > 0.50sec (2.5), and heart rate < 60 bpm (1.7). The occurrence of syncope/death events (LQTS severity) showed familial patterning. Statistical genetic segregation analysis of QTc inheritance in one large LQTS pedigree (n=240) revealed that a dominant major gene contributes to lengthened QTc; in a 2nd LQTS pedigree (n=106), the mode of QTc inheritance was not so clear cut (? polygenic mechanism). These findings indicate that familial LQTS is an arrhythmogenic disorder with inherited delay in ventricular repolarization consistent with genetic heterogeneity.