(AEF), peak E velocity,deceleration time (dec t)and Isolumic relaxation time (IVRT), LA maximal and minimal volume, LV cardiac mass index (LVMI), pulmonary venous diastolic and systolic flows and reversal A wave.

Results. All pts had an increased LVMI (277 ± 48 vs group B 114 ± 38 g/m2;p<0.001). Diastolic function was impaired in group A: peak E vel was 0,50 ± 0.09 m/sec vs control 0,78 ± 0,10 m/sec, dec t was 278 ± 47 vs 200 ± 34, IVRT was 110 ± 14 vs 87 ± 11. Pulmonary venous diastolic was increased 57 ± 15 and systolic was decreased 43 ± 13 in group A. Peak A vel was 0,44 ± 0.12 m/sec in group A vs B 0,54 ± 0,11 m/sec, suggesting a delay in the recovery of atrial mechanical function in hypertension. Pulmonary venous reversal flow was reduced (17 ± 7 cm/sec) in group A and correlate with peak A vel. AEF increased significantly with age in normal subjects (r=0.9;p <0.001)and was strongly related to peak A velocity. In hypertensive patients the relation of AEF with age was weaker (r=0.42; p <0.05). A strong relation was reported between LVMI and AEF (r=0.75;p <0.001)in group A while a weaker relation was reported with other atrial parameters. LA size was reduced in group A after conversion. La max vol decrease from 30 ± 8 to 25 ± 6 cm3, La min vol decrease from 18 ± 5 to 12 ± 6 cm3. Comparing data with group B LA size was increase in group A. A relationship between LA max and min volumes and AEF was observed in group A (r=−0.79, p<0.01 and r=−0.68, p<0.05).

LV hypertrophy had a strong influence on atrial function and on diastolic function in pts with hypertension and atrial fibrillation.

Key Words: Hypertrophy, Left Atrium, Atrial Function

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HYPERCHOLESTEREMIA-INDUCED CHANGES IN LEFT VENTRICULAR GEOMETRY IN THE GUINEA PIG

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Purpose: Concentric remodeling of the heart is associated with increased cardiovascular risk in hypertensive patients and this change in cardiac geometry has been described in normotensive hypercholesteremic patients.The purpose of this study was to examine the effects of dietary induced hypercholesteremia on cardiac stucture in the guinea pig as no suitable model is currently available.

Methods: 7 week old male Hartley guinea pigs were fed certified guinea pig chow with (n = 6)or without (n = 6) 1% cholesterol for 13 weeks. Guinea pigs were anesthetized (ketamine (120 mg/kg) acepromazine (12 mg/kg) ip) and the jugular vein catheterized. Hearts were arrested in diastole with potassium chloride and excised, fixed in formalin and cross-sections through both ventricles were obtained by transverse sectioning 2mm parallel to the coronary groove. These sections were paraffin embedded, sectioned, and stained using hematoxylin and eosin. To ensure all sections were from a consistent location, sections were examined to ensure that neither attachment of the large ventral papillary muscle to the left ventricular wall nor valve cusps were visible within the histological sections. Sections were then digitalized and left ventricular chamber area, total left ventricular area (ventricular myocardium plus ventricular lumen), and mean ventricular thickness measured.

Results: Results are given in the Table. Cholesterol-fed guinea pigs had a significantly smaller left ventricular chamber area, left ventricular area and decreased area of left ventricular myocardium. No differences were observed in the thickness of the left ventricular myocardium between the two groups thus demonstrating an increase in relative wall thickness as confirmed by echocardiography data presented at this meeting.

Conclusion: Hypercholesteremia induces concentric remodeling of the left ventricle in the guinea pig. This is a novel model, relevant to human hypercholesteremia, which can be used to investigate the mechanisms and consequences of concentric remodeling.

Effect of hypercholesteremia on LV structure

	Control	Cholesterol
LV chamber area	30.9 ± 7.2	19.2 ± 4.0*
LV wall area	81.0 ± 5.2	68.7 ± 4.2*
Total LV area	112.0 ± 7.0	88.6 ± 6.0*
Mean LV wall thickness	2.25 ± 0.25	2.24 ± 0.17

P < 0.05; LV = left ventricle,*

Key Words: Hypercholesteremia, Cardiac Geometry, Morphometry

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DIASTOLIC DYSFUNCTION AND CENTRAL OBESITY RELATED HYPERTENSION: ROLE OF TRASFORMING GROWTH FACTOR BETA-1

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In this study the relationship between circulating growth factor b_1 ($TGFb_1$) and diastolic dysfunction has been investigated in non obese and central obese hypertensive patients. Sixthy-seven hypertensive outpatients both lean and with central obesity were enrolled and divided in three groups, according to their BMI values: Group A comprised 18 lean hypertensives (men with BMI <25 Kg/m^2 and women with BMI <24.7 Kg/m^2); Group B comprised 26 overweight hypertensives (men with BMI ≥25 Kg/m^2 and <30 Kg/m^2 and women with BMI ≥24.7 Kg/m^2 and <27.3 Kg/m^2): Group C included 23 obese hypertensives (men with BMI ≥30 Kg/m^2 and women with BMI ≥27.3 Kg/m^2). All hypertensives were further subgrouped according to their diastolic dysfunction in hypertensives with diastolic dysfunction and hypertensives without diastolic dysfunction.In all patients, circulating $TGFb_1$ by ELISA technique and. In addition, left ventricular geometry, systolic (EF%) and diastolic function (E/A, DTE, IVRT) by M-B mode echocardiography were calculated. Overweight and obese hypertensives had significantly(p<0.05)higher BMI,WHR and circulating $TGFb_1$ than lean hypertensives. Obese hypertensives had significantly (p<0.05) higher BMI and circulating $TGFb_1$than overweight hypertensives. Overweight and obese hypertensives had significantly (p<0.05) higher LVM, LVM/$h^{2.7}$, DTE, IVRT and lower E/A ratio values than lean hypertensives. Hypertensives with diastolic dysfunction had significantly higher LVM/$h^{2.7}$(p<0.001) and circulating $TGFb_1$ (p<0.04) than hypertensives without diastolic dysfunction. In all subjects $TGFb_1$ correlated directly with BMI(r=0.60;p<0.0001), LVM/$h^{2.7}$(r=0.28;p<0.03), IVRT(r=0.30; p<0.02)and negatively with E/A ratio(r=−0.38;p<0.002). Multiple regression analysis indicated that $TGFb_1$ was able to explain the 43% E/A ratio variability (R=0.65; p<0.0001).Our data suggest that $TGFb_1$ overproduction may be involved in the pathophysiology of altered ventricular filling in obese hypertensives. In these patients this finding seems to be independent by the concomitant presence of Left Ventricular Hypertrophy.

Key Words: Diastolic Dysfunction, Obesity Related Hypertension, Trasforming Growth Factor beta-1

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INCREASED OXIDATIVE STRESS IN HYPERTENSIVE PATIENTS WITH BORDERLINE LEFT VENTRICULAR MASS EVALUATED BY ELECTROCARDIOGRAPHY

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Introduction: Echocardiography and Magnetic Resonance Imaging (MRI) have moved the electrocardiogram (ECG) aside from the evalu-