hypertrophied LV then WHT. Future prospective studies may shed additional light on the overall incidence and prevalence of myocardial fibrosis in BHT and its relationship to adverse risk.

1235-66 Gender Differences in Hypertensive Target Organ Disease

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Background: The present study was designed to assess the prevalence of hypertensive target organ disease as assessed by the presence of left ventricular hypertrophy (LVH) or a decrease in renal blood flow (LRBF) in a population of 355 patients with essential hypertension (139 women and 216 men).

Methods: Systemic and renal hemodynamics, cardiac structure and function were assessed by invasively measuring intra-arterial pressure, cardiac output (indocyanine green), 131 I-para-aminohippuric acid clearance, and 2D-guided M-mode echocardiography after the patient had been off therapy for at least four weeks.

Results: As a measure of association, we used odds ratio ± 95% CI.

Outcomes (%)	Male	Female	P
LVH absent, LRBF absent	41	24	< 0.01
LVH present, LRBF absent	8	15	= 0.03
LVH absent, LRBF present	39	34	< 0.01
LVH present, LRBF present	12	27	= 0.16

After adjusting for differences in age, race, blood pressure, duration of hypertension and total peripheral resistance index, we found that the odds for women of having LVH was 5.9 (2.6, 13.5) of that for men and 5.3 (2.4, 11.9) for the combined presence of both. No gender differences were found with regard to LRBF alone.

Conclusion: The present data demonstrated that for any given level of arterial pressure, and after adjusting for duration of hypertension, the likelihood of women having LVH is six times greater than in men. Also, women have higher odds than men for involvement of both heart and kidney. This would indicate that compared with men, women are more susceptible to hypertensive target organ disease.

1235-67 Relationship Between Left Ventricular Mass Index and 24-hr Urinary Steroid Metabolites in Essential Hypertension

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Background: In essential hypertension, left ventricular mass index (LVMI) is not only determined by blood pressure (BP) level but also independently by neurohumoral factors. As plasma steroids have a variable diurnal pattern, we tested the hypothesis that 24-h urinary steroids metabolites are correlated with LVMI in mild to moderate essential hypertension.

Methods: Twenty-four patients with essential hypertension (48 ± 6 years, office systolic BP 163 ± 26 mm Hg, diastolic BP 100 ± 14 mm Hg, 24 hr, day-time, and night-time ambulatory systolic BP 146 ± 27 mm Hg, 151 ± 26 mm Hg, and 140 ± 30 mm Hg, 24 hr, day-time, and night-time ambulatory diastolic BP 87 ± 15 mm Hg, 92 ± 15 mm Hg and 82 ± 17 mm Hg) underwent an echocardiography to determine LVMI. A 24-hr urine collection was sampled for the determination of cortistone and cortisol metabolites excretion by HPLC technique.

Results: There was a highly significant correlation between LVMI (115 ± 31 g/m^2), and cortisone (31 ± 18 μg/24 h), r = 0.597, P = 0.009 and cortisol (23 ± 14 μg/24 h), r = 0.611, P = 0.007, independently from arterial BP (r = 0.224 and r = 0.021 for office systolic and diastolic BP; r = 0.335 and r = 0.244 for 24-h ambulatory systolic and diastolic BP; r = 0.403 and r = 0.270 for day-time systolic and diastolic BP and r = 0.272 and r = 0.063 for night-time systolic and diastolic BP).

Conclusion: These data support the hypothesis that left ventricular mass in essential hypertension is partially determined by steroids.

1235-68 Do High Growth Hormone Levels Instigate Heart Failure in Acromegaly?

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Cardiovascular disease is the major cause of morbidity and mortality in acromegaly. However, whether a specific cardiomyopathy exists, and the relative contribution and relationship of serum growth hormone (GH) and hypertension to this disorder have not been established. We assessed 25 consecutive pts with active acromegaly (age 46 ± 12, 13 men) with a cardiac exam, EKG, GH and insulin-like GF-1 levels at baseline and after 3 months of therapy.

Results: Congestive Heart Failure (CHF) ≥ NYHA class 2 was present in 7 of 25 pts, 13 of 25 had hypertension (BP > 140/90 mmHg on 3 visits) and 15 of 25 had LVH by Rhomhilt-Estes criteria. All 7 CHF pts were hypertensive vs. 6 of 18 non-CHF pts (p = 0.05). CHF pts had longer duration of acromegalic symptoms and presumably ↑GH (7 ± 3 vs. 3 ± 2 yr., p = 0.01). Even after controlling for BP, GH strongly correlated with CHF (r = 0.7), p = 0.001). Although systolic BP positively correlated with LVH (r = 0.47, p = 0.02) and ↑total 12-lead QRS amplitude (r = 0.49, p = 0.01), no such correlation with CHF (r = 0.1, p = 0.38) was evident after controlling for GH.

Baseline variables	CHF (7 pts)	Non-CHF (18 pts)	p value
Serum GH (μg/ml)	50 ± 11	26 ± 11	< 0.001
SBP (mmHg)	151 ± 8	136 ± 15	= 0.005
DBP (mmHg)	91 ± 2	79 ± 14	= 0.002
LVH score	6.6 ± 0.5	2.8 ± 3.2	< 0.001
QRS amplitude	217 ± 35	158 ± 40	= 0.003

Conclusions: CHF is frequent among pts newly diagnosed with acromegaly. High GH levels, and probably also the duration of elevated levels, correlate with CHF. This finding suggests that treating CHF with GH may have deleterious long-term consequences, despite recently demonstrated short-term benefit.

1235-69 Ventricular Geometry and Function in Healthy, Normotensive Adults With Family History of Hypertension

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Background: Left ventricular hypertrophy has been found in the normotensives with genetic predisposition for hypertension (HT). The aim of this study was to evaluate ventricular geometry and function in subjects with important family history of hypertension (HXH).

Methods: We studied a group 110 healthy normotensives, each having both parents with HT (HXH group; mean age 42 yrs, men 72, women 38) and a group of 110 healthy normotensive controls (NT group) without family history of HT, matched for age and sex. Obese subjects were excluded from both groups. 2-D, M-mode, and Doppler echo were used to assess parameters of LV and right ventricular (RV) filling: early (E)/atrial (A) filling ratio, E deceleration rate (ED), and the isovolumic relaxation time (IVRT). Endocardial fractional shortening (FS), LV mass indexed to height^2 (LVMI) and the relative wall thickness (RWT), were also calculated.

Results: HXH had increased blood pressure (128/77) compared to the NT (117/68; p < 0.05). Body mass index was comparable in the two groups (25.3 vs 24.5 kg/m^2, p = NS). The filling parameters and the LVM/were LVMI were significantly different between HXH and NT.

	E/A	ED (m/s^2)	IVRT (ms)	FS (%)	LVMI (g/m^2)	RWT
HXH	13*	4.4	80*	35	116*	0.39*
NT	1.5	4.9	72	33	101	0.32

*p < 0.05 vs NT, *p = 0.061

No significant differences in RV diastolic filling were observed between HXH and NT. We made the assumption that HXH who reached older ages were less prone to develop HT than the rest of the group with HXH. Paired differences in filling parameters and LVMI between HXH and NT were markedly smaller in the older (age > 65 yrs, n = 16; E/A 0.02, LVMI 6 g/m^2) compared with the younger subjects (age < 65 yrs; n = 94; E/A 0.3, LVMI 18 g/m^2, all p < 0.05).

Conclusions: Subjects at high genetic risk of developing HT may have abnormalities in LV filling. Out of the subjects with genetic risk of developing HT, those who are at lower risk of HT show less important alterations in LV geometry and diastolic filling.

1235-70 Endothelin Pathway and Renin-Angiotensin System in the Progression to Isolated Diastolic Heart Failure in Hypertensive Hearts

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Background: Heart failure due to diastolic dysfunction with preserved systolic function frequently occurs in hypertensive pts. However, mechanism of transition from compensated stage to isolated diastolic heart failure remains to be clarified.

Methods: The study subjects consisted of Dahl salt sensitive rats fed with 8% NaCl since 7 wks old which develop hypertension and heart failure