0001) and ACE genotype (p = 0.018), and the E/A_t interacted with age (p < 0.001), heart rate (p = 0.025) and ACE genotype (p = 0.047). There was a strong correlation between E/Ap and LVMI in the DD group (r = 0.81, p < 0.0001) but not in heterozygotes (r = −0.23, p = 0.23).

These findings suggest that the DD genotype of the ACE gene is associated with impairment of left ventricular diastolic filling in patients with essential hypertension.

1007-89 Blood Pressure Changes in Acute Stroke — The West Birmingham Stroke Project

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To examine blood pressure (BP) changes in stroke and the differences between males and females, different ethnic groups and type of stroke (thrombosis, haemorrhage, transient ischaemic attack), we studied 86 patients (48 males; mean age 64.2 years ± s.d.9.2) with acute onset stroke (ictus < 12 hours) in a district general hospital. 31 patients (36.0%) had a previous history of hypertension. Manual BPs and BPs recorded using Spacelabs 09207 ambulatory blood pressure monitors (ABPM, n = 73) were studied. Mean BPs (standard deviation (s.d.) in different ethic groups and in different types of stroke are as follows:

(mmHg)	ManSBP	ManDBP	24 hr SBP	24 hr DBP	dSBP	dDBP	nSBP	nDBP
White	156.3	91.8	141.7	84.2	143.4	85.6	141.9	83.0
± s.d.	30.1	18.3	21.5	14.8	20.5	14.4	24.3	16.3
Black	166.1	86.3	173.4*	91.7	174.7*	92.3	164.2	86.2
± s.d.	38.4	22.4	30.7	13.5	29.1	13.6	31.6	10.3
Asian	162.5	88.2	141.1	85.3	141.8	85.4	139.7	84.0
± s.d.	24.3	12.5	16.6	10.6	16.5	11.6	18.4	12.6
Throm.	155.9	88.7	143.7	84.4	144.3	85.1	142.9	82.6
± s.d.	27.5	18.1	21.4	14.0	21.1	14.0	23.0	15.0
Haem.	172.6	100.0	161.6	91.7	170.5**	96.0	177.8*	98.0
± s.d.	33.5	21.7	39.0	18.0	31.4	14.1	35.2	18.4
TIAs	154.5	89.8	137.6	85.0	142.2	88.5	131.6	80.5
± s.d.	32.4	14.1	21.2	15.4	21.6	15.4	20.1	15.3

[SBP = systolic BP, DBP = diastolic BP; Man-manual; ABPM recordings: d = mean daytime, n = mean nighttime, 24 hr = mean 24 hour, TIA = transient ischaemic attack], *p < 0.001, **p < 0.05

The median percentage of successful BP readings by ABPM was 92% (IQR 72–98). SBPs as recorded by ABPM were significantly higher in black patients with acute stroke and in patients with intracerebral haemorrhage, who showed a trend towards higher nocturnal BPs. There was no difference in BPs between males and females and those who were alive or dead after 6 months follow-up (p = NS). There was also no difference between mean day and night SBP (mean difference = 1.9 mmHg, p = 0.08) although mean daytime DBP was higher than mean nighttime DBP (mean difference 24 mmHg, p = 0.01). This study demonstrates higher SBPs as recorded by ABPM (but not manually) in black patients with acute stroke in patients with intracerebral haemorrhage. ABPM recordings may be useful in the assessment of BPs in patients with stroke, who demonstrate a loss of diurnal BP rhythm, and may be considered as 'non-dippers'.

1007-90 Relationship Between Nonhemodynamic Factors and Left Ventricular Hypertrophy in Essential Hypertension

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To assess the effect of nonhemodynamic factors on left ventricular hypertrophy (LVH) 62 essential hypertension (EH) pts (32 men, 30 women, mean 55 years) and 20 normotensive health subjects (10 men, 10 women, mean 52 years) were studied. According to the Devereux formula left ventricular mass index (LVMI) was calculated, and EH pts were divided into LVH group (LVMI > 125 g/m^2 in men, and > 120 g/m^2 in women) and no LVH group. After an overnight fast, blood samples were taken for the determination of parathyroid hormone (PTH), angiotensin II (AT II) and aldosterone (ALD) by a radio-immunoassay. Results are presented in the table.

Parameters	Control (n = 20)	EH (n = 62)	LVH (n = 47)	no LVH (n = 15)
PTH (pg/ml)	25.2 ± 7.6	44.1 ± 14.8**	47.1 ± 15.2**	33.9 ± 8.4*#
AT II (pg/ml)	74.4 ± 38.6	232.7 ± 157.3**	265.3 ± 165.7**	130.1 ± 68.7*#
ALD (pg/ml)	83.7 ± 21.1	133.4 ± 46.9**	144.7 ± 47.7**	104.2 ± 24.4*#

*p < 0.005, **p < 0.001 vs control group; #p < 0.005 vs LVH group.

As shown, there was a significant difference in PTH, AT II and ALD between EH group and control group. Furthermore, in LVH group PTH, AT II and ALD elevated significantly as compared with no LVH group. In addition, we found that LVMI correlated with AT II (r = 0.342, p < 0.01) and ALD (r = 0.356, p < 0.01). There was a more significant correlation between LVMI and PTH (r = 0.422, p < 0.0025).

In conclusion, nonhemodynamic factors are important determinants of LV mass. Besides the renin-angiotensin-aldosterone system, PTH appears to play an important role in cardiac hypertrophy.

1007-91 Left Ventricular Mass and Geometric Distribution in Treated and Untreated Hypertension: Impact by Gender

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Several antihypertensive drugs can induce left ventricular hypertrophy (LVH) regression. Thus, LV mass and geometry may be significantly influenced by prior treatment. To define whether left ventricular mass and geometry differ between treated vs. untreated, and controlled vs. uncontrolled (sDBP > 90 mmHg) hypertension (HTN), 576 pts (men 329, women 247; mean age 61 yrs) with mild-to-moderate primary HTN and 107 normotensive (non-HTN) subjects (men 63, women 44; mean age 61 yrs) underwent echocardiography. LV mass index (men 134 g/m2, women 110 g/m2) and geometrical classification (concentric, eccentric, concentric remodeled and normal) were determined by widely accepted methods. LVH was more prevalent in women for both groups HTN and non-HTN (70% vs. 56%, p < 0.05; 36% vs. 29%, p = ns). Among HTN, women but not men, showed differences according to previous medication and efficacy. Compared to women controlled, those uncontrolled despite medication (58% vs. 80%, p < 0.005), and those on no medication (58% vs. 68%, p = ns), showed more LVH. Prior treatment had no effect on geometry among men, but women uncontrolled had a higher prevalence of concentric LVH than controlled (60% vs. 43%, p < 0.005) or on no medication (60% vs. 49%, p < 0.005). In conclusion, women, but not men, showed differences in LVH and in geometric distribution according to prior treatment and efficacy. Concentric is the predominant form of LVH in both HTN and non-HTN subjects.

1007-92 Sustained-Release Verapamil Blunts the Morning Hemodynamic Surge During Daily Activity: A Randomized Trial

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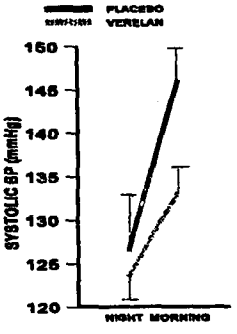
It has been hypothesized that the morning surge in blood pressure (BP) and heart rate (HR) might cause the increased morning incidence of myocardial infarction by precipitating plaque rupture. Since verapamil was found to decrease the incidence of reinfarction or death in DAVIT II, we tested whether sustained-release verapamil (Verelan), given daily at 9 am, blunted the morning hemodynamic surge. Twelve subjects with hypertension were studied using a randomized, cross-over, placebo-controlled design. Ambulatory BP and HR were recorded and the differences between the means at night (midnight to 6 am) and the means during the morning (6 am to noon) were calculated.



Verelan decreased morning systolic BP by 12 mmHg (p = 0.001), diastolic BP by 11 mmHg (p = 0.001) and HR by 6bpm (p = 0.03) but did not significantly reduce BP and HR at night. It therefore lowered the morning rise in systolic BP (by 50%, p < 0.001) as shown, diastolic BP (by 75%, p < 0.001) and tended to reduce the HR rise (by 32%, p = 0.175). Sustained-release verapamil blunted the morning hemodynamic surge. This may be a mechanism by which verapamil could reduce the risk of plaque disruption and myocardial infarction in the morning.