

COMPARISON OF LEFT VENTRICULAR GEOMETRY AND MASS BETWEEN BLACK AND WHITE ESSENTIAL HYPERTENSIVE PATIENTS

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Racial differences in hypertensive morbidity are incompletely accounted for by standard risk and socioeconomic factors. To determine whether race affects LV mass and geometry, we compared 380 white and 47 black essential hypertensive patients by echocardiography. Blacks and whites had similar age, systolic BP, duration and treatment of hypertension and standard risk factors, but blacks had higher diastolic BP (104 ± 18 vs 98 ± 11 mmHg; $p=0.014$) and higher body weight (82 ± 17 vs 77 ± 15 kg, $p=0.037$). LV mass index and relative wall thickness were significantly greater in blacks (119 vs 105 g/m²; $p=0.02$ and 0.46 vs 0.39 ; $p=0.003$). Black patients had nearly twice the prevalence of LV hypertrophy and increased relative wall thicknesses (42% vs 23% ; $p<0.001$ and 64% vs 33% , $p<0.0005$). In multivariate analyses, race was consistently a predictor of increased LV mass and abnormal cardiac geometry in essential hypertensive patients. We conclude that black hypertensives are more likely to develop adverse changes in left ventricular mass geometry which may contribute to higher rates of hypertensive related cardiovascular morbidity and mortality.

CLINICAL SIGNIFICANCE OF VENTRICULAR TACHYCARDIA IN HYPERTENSIVE PATIENTS WITH MARKED LEFT VENTRICULAR HYPERTROPHY

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Left ventricular hypertrophy (LVH) in pts with systemic hypertension (HBP) is associated with increased mortality. Although ventricular ectopy is more common in pts with HBP and LVH, a direct relationship between ectopy and survival has not been established. Thus, we evaluated 32 consecutive pts with HBP and marked LVH (wall thickness >16 mm) using echocardiography and 24-hour ECG monitoring (HOLTER). Pts ranged from age 20 - 86 years (mean 57); 19 (63%) were women, and 14 (37%) were men. All pts had normal systolic function (fractional shortening $=35 \pm 1\%$). Seven of the 32 pts (21%) had ventricular tachycardia (VT) (3-15 consecutive beats on HOLTER); the remaining 25 (79%) had no VT. Pts with or without VT did not differ with regard to age, sex, blood pressure or cardiac dimensions. Five of the 32 pts (15%) died over a follow-up period of 12-15 months (mean 13), including one successful resuscitation. Age at time of death ranged from 49-83 years (mean 64). Three deaths occurred in the group of 7 pts with VT (43%), and 2 occurred in the 25 pts without VT (8%); $p=0.02$. In conclusion, hypertensive pts with marked LVH experience substantial mortality (15%) at one-year followup. However, the presence of VT appears to identify a subgroup of these pts with particularly poor survival.

ASSOCIATION BETWEEN DECREASED NOCTURNAL BLOOD PRESSURE REDUCTION AND CARDIAC HYPERTROPHY IN HYPERTENSIVE WOMEN

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The effects of diurnal BP variations on LV mass in essential hypertension were assessed separately in 99 men and 122 women with 24-h noninvasive ambulatory BP monitoring and echocardiography. Patients with average daytime (6-22 h) systolic and diastolic BP decreasing by less than 10% during night (22-6 h) were classified as nondippers; the others were classified as dippers. Both among men and women, dippers and nondippers did not differ in several covariates possibly related to LV mass and nocturnal BP decline, including age, daytime BP and sleeping disturbances during ambulatory BP monitoring. In men, LV mass index was 115 g/m² (95% CI: 104-126) in dippers and 114 g/m² (95% CI: 104-125) in nondippers ($p=0.9$), and the prevalence of LV hypertrophy was 17% and 22%, respectively ($p=0.5$). In women, LV mass index was 92 g/m² (95% CI: 86-98) in dippers and 112 g/m² (95% CI: 101-122) in nondippers ($p=0.0006$), and the prevalence of LV hypertrophy was 20% and 48%, respectively ($p=0.0013$). In a multiple regression analysis, average 24-h systolic and diastolic BP and body weight showed an independent relation to LV mass in men, accounting for 28% of its variability (multiple $r=0.53$), while age, body weight, average 24-h systolic BP and per cent reduction of systolic BP from day to night were independently related to LV mass in women, accounting for 56% of its variability (multiple $r=0.74$). These data suggest that a reduction of systolic or diastolic BP by more than 10% from day to night, detected with noninvasive ambulatory BP monitoring, can result in a slower or inhibited development of LV hypertrophy in untreated hypertensive women, but not in men.

INFLUENCE OF NIGHT BLOOD PRESSURE ON LEFT VENTRICULAR HYPERTROPHY IN THE ELDERLY HYPERTENSION

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To assess the influence of nocturnal blood pressure (BP) on left ventricular hypertrophy (LVH) in the elderly, ambulatory BP was measured in 26 elderly hypertensive patients and 11 age-matched normal controls (NC, 74.7 years). The hypertensive patients were divided into 15 with LVH (H1, 73.7) and 11 without LVH (H2, 72.3). Twenty-four hour ambulatory BPs were measured by ABPM630 every 30 minutes and divided into daytime (600-2100) and nighttime (2100-600) for analysis. M-mode echocardiography was conducted before and after handgrip (HG) isometric exercise test. LVH defined as more than 130 g/m² of the LV mass index (LVMI).

	LVMI	day SBP	night SBP	d-n SBP
H1	$142.3 \pm 7.6^*$	$152.2 \pm 13.7^*$	$149.6 \pm 15.1^*$	$3.0 \pm 9.4^*$
H2	$124.4 \pm 4.2^*$	$151.3 \pm 15.1^*$	$138.2 \pm 20.1^*$	13.0 ± 16.0
NC	110.8 ± 8.9	126.2 ± 11.5	117.3 ± 15.6	8.0 ± 10.5

SBP: systolic blood pressure
d-n: difference between day and night * $p<0.05$

Night SBP in H1 was significantly higher than H2 or NC. SBP difference between day and night in H1 was significantly less compared with H2. LVMI was better correlated with night SBP ($r=0.44$, $p<0.01$) than to average SBP ($r=0.40$) or HG SBP ($r=0.37$) or day SBP ($r=0.26$).

In conclusion, blood pressure at night contribute most to the progression of LVH in the elderly hypertensive patients.