The relationship between ambulatory blood pressure and echocardiographically assessed left ventricular hypertrophy


Department of Cardiovascular Medicine, East Birmingham Hospital, Bordesley Green East, Birmingham, U.K.

Summary

1. Continuous intra-arterial ambulatory monitoring of blood pressure was recorded in 46 patients with mild to moderate hypertension under standardized conditions. M-mode echocardiography was performed after recording and left ventricular mass index calculated by standard formulae.

2. Systolic blood pressure from continuous recording was significantly correlated with left ventricular mass index (mean 24 h: $r = 0.543$, $n = 45$, $P < 0.001$). Diastolic blood pressure exhibited a weaker but still significant correlation with left ventricular mass index (mean 24 h: $r = 0.318$, $n = 45$, $P < 0.05$). Casual systolic blood pressure was significantly correlated with left ventricular mass index ($I = 0.476$, $n = 46$, $P < 0.001$) but casual diastolic blood pressure did not correlate with left ventricular mass index ($r = 0.245$, $n = 46$). Awake blood pressure variability, age, resting plasma renin activity and resting plasma noradrenaline levels did not have a significant correlation with left ventricular mass index.

3. Nine patients were treated for 16 weeks with once-daily timolol and repeat ambulatory monitoring and M-mode echocardiography was performed with the same protocol.

4. Once-daily timolol provided good 24 h control of blood pressure and repeat echocardiography showed a reduction in left ventricular mass index in that group of patients ($t = 2.59$, $P < 0.05$).

Key words: echocardiography, left ventricular hypertrophy.

Introduction

Previous work has shown the value of echocardiography in the assessment of left ventricular (LV) mass in patients with electrocardiographic (ECG) and radiological (X-ray) evidence of LV hypertrophy [1] and also in assessing the effects of treatment in patients with echocardiographic features of LV hypertrophy [2].

This study was performed to assess the relationship between blood pressure (BP) recorded over prolonged periods and echocardiographically assessed LV mass in patients with mild-to-moderate hypertension and no evidence of target organ damage.

Patients and methods

Forty-six patients (18 females, 28 males) of mean age $37.5 \pm 12$ years (range 17–58 years) were studied. All patients had outpatient casual BP of greater than 140/90 mmHg on three separate occasions, the mean casual BP of the group being $160 \pm 13/101 \pm 8$ mmHg. All patients were free from clinical evidence of target organ damage and from LV hypertrophy assessed by electrocardiographic and radiological criteria. No patient had received anti-hypertensive therapy before study.

All patients underwent continuous intra-arterial ambulatory monitoring of BP [3] as hospital inpatients under standardized conditions [4]. The recording was analysed beat to beat on a computer after periods of pressure artifact and damping had been excluded. Patients recorded the time of retiring at night and early morning waking so that BP could be analysed over the whole 24 h, during sleep and during the awake period. Variability of BP was measured as previously described [5]. During this period of study resting venous blood samples were drawn.
from an indwelling venous cannula for plasma renin activity (PRA) and plasma catecholamine levels.

After the period of continuous ambulatory monitoring M-mode echocardiography was performed as previously described [6]. Only echocardiograms showing continuous endocardial echoes of the interventricular septum and LV posterior wall were included. The Teicholz formula [7] was applied to LV echocardiographic dimensions to calculate the LV cavity volume and the same formula was used to calculate total LV volume, but here 2 × LV posterior wall thickness was added to the LV diastolic dimension. From these data LV mass and LV mass index (LVMI) were calculated [8].

Nine male patients were treated with timolol once daily, mean dose 29 ± 4.6 mg (range 25–40 mg), for a 16 week period and were re-studied with similar conditions used for 24 h ambulatory BP monitoring, which was followed by repeat echocardiography.

Results

Of the 46 patients included in the study two were found to have asymmetric septal hypertrophy and the echocardiographic formulae used would underestimate their LVMI. In one further patient, the sleep BP recording was of poor technical quality and was excluded.

The mean 24 h systolic blood pressure (SBP) for the group was highly significantly correlated with LVMI \((r = 0.543, P < 0.001)\). LVMI also exhibited a strong correlation with awake SBP \((r = 0.523, P < 0.001)\), sleep SBP \((r = 0.475, P < 0.001)\) and casual SBP \((r = 0.476, P < 0.001)\). Diastolic blood pressure (DBP) from continuous ambulatory monitoring exhibited a weaker correlation with LVMI: mean 24 h, \(r = 0.318, P < 0.05\); mean awake, \(r = 0.303, P < 0.05\); mean sleep, \(r = 0.308, P < 0.05\). Casual DBP was not significantly correlated with LVMI, \(r = 0.245\).

Variability of awake SBP and DBP did not correlate with LVMI, \(r = 0.199\) and \(r = 0.168\) respectively.

No significant correlation was seen between LVMI and age \((r = 0.154)\), resting plasma renin activity \((r = 0.137)\) or with resting plasma noradrenaline levels \((r = 0.330, n = 28)\).

The group of nine patients treated with timolol showed a reduction in SBP and DBP for the 24 h period, from 142 ± 9/90 ± 8 mmHg to 120 ± 8/73 ± 7 mmHg \((P < 0.001)\). The mean LVMI for seven of this group, in whom satisfactory echocardiograms were obtainable, was 73 ± 10 g/m² pretreatment. After 16 weeks' treatment with timolol the LVMI was significantly reduced to 66 ± 9.5 g/m² \((t = 2.59, P < 0.05)\).

Discussion

Work in hypertensive children has suggested that echocardiographic assessment of cardiac hypertrophy may be valuable in detecting significant cardiac changes when the electrocardiogram and chest X-ray are normal [9]. The correlation in our study of SBP with LVMI would also indicate the usefulness of echocardiography in the complete assessment of adult hypertensive patients with no evidence of LV hypertrophy as assessed by conventional methods and providing an additional measure of LV response to hypertension.

The evolution of LV hypertrophy in response to systemic hypertension is of multifactorial aetiology [10] and from our study it would seem that SBP is a particularly important factor in this evolution. Furthermore, it would appear that sustained elevation of pressure is important as the swings of pressure associated with an increased variability are not significantly correlated. Age, plasma renin activity and plasma catecholamine levels have also been postulated to be involved in the hypertrophic process [10] but our study does not support that hypothesis.

Animal work has shown that regression of LV hypertrophy can occur with treatment of hypertension but whether this was due to a lowering of blood pressure or due to a direct effect of the drugs on the myocardium was unclear [11]. Previous studies in man using echocardiography as a means of assessing LV hypertrophy have shown a reduction in muscle mass with treatment, although this has been in patients who had previous treatment stopped, patients with evidence of target organ damage [2] or patients with asymmetric septal hypertrophy [12]. The group of patients treated with timolol all had normal resting ECG and with control of blood pressure over the full 24 h period demonstrated a fall in echocardiographically assessed LV mass index.

In conclusion, we feel that echocardiography is a useful adjunct to electrocardiography and radiology in the initial assessment of patients and also when assessing the efficacy of treatment. In this study SBP seems to be more important than DBP in the genesis of LV hypertrophy and this should be borne in mind when identifying patients who need anti-hypertensive therapy and during control of blood pressure in their follow-up.
Acknowledgments

We thank Miss A. M. Strong for secretarial assistance, Dr A. Ferraro for technical assistance and Dr M. F. Shiu for advice with echocardiograms. Dr P. Gosling performed assay of plasma renin activity and Miss B.-M. Eriksson catecholamine measurements. We are grateful to Merck, Sharp and Dohme (U.K.) for financial support and supply of Blocadren.

References


