Effect of blood pressure control on left ventricular hypertrophy in patients with essential hypertension

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Summary

1. Changes in left ventricular structure and function were assessed by echocardiography in 22 patients before and after 9 months blood pressure control.

2. Nine patients had normal baseline echocardiograms (group 1) and 13 had echocardiographic evidence of left ventricular hypertrophy (group 2).

3. Group 2 patients demonstrated significant reductions in posterior wall thickness ($P < 0.01$), septal wall thickness ($P < 0.025$) and left ventricular mass ($P < 0.005$). Only six of the 13 patients showed a reduction of $\geq 3$ mm in posterior wall thickness. The remainder showed no alteration or only a slight non-significant reduction.

4. The regression of voltage in some patients but not in others did not appear to be related to initial blood pressure, the extent of the fall in blood pressure or duration of follow-up. It was not possible to say whether any specific therapy was beneficial to regression since most of the patients were on multiple therapy.

Key words: echocardiography, hypertension, left ventricular hypertrophy, regression.

Abbreviation: LVH, left ventricular hypertrophy.

Introduction

Before the introduction of M-mode echocardiography, assessment of the effect of blood pressure control on left ventricular hypertrophy (LVH) in man was confined to electrocardiography (ECG). Improvement in ECG appearances has been previously reported with control of blood pressure (George, Breckenridge & Dollery, 1972) but since factors apart from LVH are known to affect voltages (Kilty & Lepeschkin, 1965) it is invalid to equate voltage reduction with regression of hypertrophy.

Since echocardiography permits direct measurement of ventricular wall thickness (Feigenbaum, Popp, Chip & Haine, 1968) it has advantages over the ECG in the assessment of changes in left ventricular dimensions with control of blood pressure. Information can also be obtained from derived indices of ventricular function which are also useful in sequential follow-up studies. The purpose of this study was to assess whether LVH regresses in patients with essential hypertension when blood pressure is controlled for a prolonged period of time.

Methods

Twenty-two patients with essential hypertension were studied. All patients were initially studied in the untreated state, with the exception of two, one of whom was taking a beta-blocking agent and the other a diuretic. All patients had uncontrolled hypertension initially. Patients with clinical or ECG evidence of a previous myocardial infarction or in cardiac failure were excluded from the study. Routine investigation failed to reveal any cause for the elevated blood pressure.

Echocardiograms were performed as previously described (Popp, Wolfe, Hirata & Feigenbaum, 1969). In brief, the characteristic motion of the mitral valve was obtained and the probe was then directed inferiorly and laterally so that the left ventricular dimensions were seen at the level of the chordae tendineae. At this level measurements of left ventricular systolic and diastolic dimensions and posterior and septal wall

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thickess were made (Sahn, DeMaria, Kisslo & Weyman, 1978). Each measurement was made in triplicate. From these data left ventricular mass (Bennett & Evans, 1974), systolic and diastolic volumes (Teichholz, Kreulen, Herman & Gorlin, 1976), ejection fraction and fractional fibre shortening (McDonald, Feigenbaum & Chang, 1972) were all derived. At the time of the first echocardiogram, annotation was made of the exact probe position. This was taken in reference to the interspace and to the distance from the mid-sternum. Thus in subsequent echocardiograms, the same position was used.

The patients were divided into two groups on the basis of their baseline echocardiographic findings. Group 1 consisted of patients with posterior wall thickness ≤ 1.1 cm and septal thickness ≤ 1.2 cm. Group 2 consisted of patients with posterior wall thickness ≥ 1.2 cm and a septal thickness ≥ 1.3 cm and were regarded as having LVH. Nine patients fulfilled the criteria for group 1 and 13 for group 2. There were seven males and two females in group 1 and 10 males and three females in group 2. Mean age of patients in group 1 was 41 years and in group 2 50 years. The mean follow-up time was 8-5 months in group 1 and 9-2 months in group 2. One patient in group 1 and nine in group 2 had ECG evidence of left ventricular hypertrophy.

After the initial echocardiography, the patients were examined at frequent intervals and therapy was adjusted so that blood pressure could be effectively controlled as quickly as possible. The regimen used was a step-wise approach of diuretic beta-blocker vasodilator in 17 patients (seven in group 1 and ten in group 2) and methyldopa with a diuretic in five patients (two in group 1 and three in group 2).

### Results

A reduction of at least 15 mmHg in systolic and/or diastolic pressure was achieved in all patients. In group 1 mean pressure fell from 172/115 to 145/94 mmHg (P < 0.001) and in group 2 from the higher baseline level of 200/129 to 154/98 mmHg (P < 0.001). Heart rate also fell in both groups (Table 1) but was significant only in group 2 (P < 0.05). A significant reduction in posterior wall thickness (P < 0.01) and in septal thickness (P < 0.025) was seen in group 2 only (Table 1) and in addition left ventricular mass (P < 0.01) also fell significantly (Table 1) in this group. These indices all remained unchanged in group 1. Further analysis of the alterations in wall thickness in group 2 revealed that in only six of the 13 patients was there a reduction in wall thickness of ≥ 3 mm, the remainder showing a minimal reduction or no change. It has been previously shown that week to week and inter-observer variation can account for as much as a 15% change in posterior wall thickness and 20% change in septal wall thickness (Ladipo, Dunn, Pringle, Bastian & Lawrie, 1980). Therefore, in only these six patients can the change in wall thickness be regarded as significant.

Changes in the indices of left ventricular function were also seen (Table 1). Thus fractional fibre shortening and stroke volume both increased significantly in group 2 (P < 0.025 for both). Again, no significant changes were noted in group 1.

### Discussion

Experimental animal studies have in recent years produced important information regarding regression of LVH when hypertension is controlled (Sen, Tarazi, Khairallah & Bumpus, 1974). Furthermore, it appears that some agents promote regression whereas others do not (Sen, Tarazi & Bumpus, 1977). This clearly has important implications in man but until recently studies have been hampered by the lack of an
accurate and yet non-invasive method of measuring LVH. Echocardiography fulfills such a need and previous studies have confirmed its ability to detect LVH in hypertensive patients before it is evident by ECG (Dunn, Chandraratna, DeCarvalho, Basta & Frohlich, 1977).

Before comparing the differing effects of antihypertensive agents on the left ventricle, it is important to establish whether or not regression occurs in patients with documented LVH with adequately controlled blood pressure for a prolonged period of time. We have demonstrated that regression does occur but not in all patients. Thus only six of the 13 patients with LVH showed a reduction in wall thickness which was greater than might otherwise be explained by the error of the methods (Ladipo et al., 1980). Reductions in septal wall thickness of ≥3 mm occurred in four of the six patients: one of the other two showed a reduction of 2 mm and the other showed no change. Only one of the remaining seven patients demonstrated a fall of 3 mm in septal thickness without associated fall in posterior wall thickness. Thus it appears that when significant regression occurs in posterior wall thickness a similar pattern is observed in the septum.

Left ventricular mass was reduced in all but two of the patients in group 2. Previous studies have suggested that left ventricular mass may be a more accurate indicator of LVH than wall thickness (McFarland, Alam, Goldstein, Pickard & Stein, 1978) and this may be why more clearcut changes were noted with this index. However, it should be emphasized that this is a derived measurement and therefore any dimensional or posterior wall changes will be magnified by a power of three, thus allowing differences to seem more marked.

The improvement in fractional fibre shortening in group 2 could have been due either to reduction in afterload or to regression of hypertrophy and it is not possible to assess the relative contributions of each of these factors from this study. A further factor which influences ventricular function is the type of drug therapy and this must also be kept in mind in the interpretation of these results.

In conclusion, a reduction in wall thickness and mass has been demonstrated in patients with echocardiographic evidence of left ventricular hypertrophy. The reasons for regression in some patients but not in others remain to be determined.

References


