Echocardiographic Detection of Left Ventricular Hypertrophy
Its Usefulness as a Prognostic Tool in Hypertensive Patients

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There is strong evidence that in hypertensive patients, electrocardiographic findings of left ventricular hypertrophy (LVH) are associated with a poor cardiovascular prognosis. In the Framingham study, the presence of defined electrocardiographic evidence of LVH was found to be associated with an increased likelihood of major cardiovascular events, independent of blood pressure level. Since the detection of such target organ damage is critical for the clinical management of hypertensive patients, an accurate identification of abnormal cardiac anatomy seems particularly important. Although the electrocardiogram (ECG) has been the conventional clinical diagnostic tool for the detection and assessment of acute ischemia, conduction abnormalities, and rhythm disturbances, it has not been highly sensitive in the detection of changes in cardiac mass such as LVH. In a large-scale study by Savage et al of patients with mild to moderate hypertension, the ECG and the chest x-ray film each identified approximately 5 percent of the patients as having met the classic criteria for a diagnosis of LVH, whereas echocardiographic criteria for LVH were demonstrated in almost 50 percent. Echocardiography, therefore, might represent the first truly sensitive and widely available noninvasive diagnostic method for critically evaluating early evidence of cardiovascular damage in patients with mild to moderate hypertension. This technique may potentially be of value in determining which patients will benefit most from long-term antihypertensive therapy, as well as in evaluating the effect of long-term antihypertensive therapy on left ventricular mass in patients with essential hypertension. So far, only a few long-term follow-up clinical trials have been reported. Therefore, there has not been sufficient time nor clinical experience as yet to evaluate definitively the prognostic significance of echocardiographic evidence for LVH or the use of antihypertensive therapy to reduce LVH, especially in those patients whose ECG and chest roentgenograms are apparently normal.

The present communication discusses some unresolved problems with echocardiographic measurements of left ventricular muscle mass, as well as its use to evaluate the efficacy of antihypertensive therapies in reducing left ventricular hypertrophy and dysfunction.

The results of early animal and human studies, using several echocardiographic methods to compare echocardiographic left ventricular mass with postmortem findings, were promising. However, recent studies using more sophisticated, complex, and divergent echocardiographic methods for the detection and quantification of LVH have yielded conflicting data which make comparisons between studies difficult and the conclusions often speculative. It remains to be shown that postmortem examination of hearts of patients who died of causes other than hypertension should be the standard for echocardiographic measurements of left ventricular mass in hypertensive patients with no, mild, or moderate LVH. In addition, despite the generally accepted importance of detecting LVH in hypertensive patients using the most sensitive and accurate methods, uncertainty persists as to whether a change in left ventricular mass is indeed the primary factor responsible for the cardiac complications seen in hypertensive patients. Factors other than a change in left ventricular mass, such as (1) a change in left ventricular diastolic function, which may precede changes in left ventricular systolic function and mass, (2) a change in the vascular smooth muscle mass that may develop earlier than, in parallel with, or after the appearance of LVH, and (3) the presence or absence of other cardiovascular diseases (eg, coronary artery disease), may reveal more important prognostic and diag-

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nastic evidence for hypertensive disease of the heart.

Studies from the literature have discussed the relationship between antihypertensive treatment-induced changes in systolic or diastolic blood pressure with the corresponding changes in echocardiographically measured left ventricular mass. These studies revealed a significant correlation (depending upon the antihypertensive agent) between blood pressure and changes in muscle mass.\(^6\) However, caution must be used when comparing data from studies in which different subgroups of patients, antihypertensive agents, and methods for measuring blood pressures, as well as different echocardiographic parameters for measuring and calculating cardiac mass, have been used.

Diverse echocardiographic indices for the recognition of LVH have been used. It is controversial as to which echocardiographic measurements, *ie*, posterior left ventricular wall thickness, ventricular septal thickness, relative wall thickness, or cross-sectional area, whether or not corrected for body mass, are the most appropriate indices of LVH. Not surprisingly, this profusion of echocardiographic methods has led to confusing and often misleading results which are difficult to compare across studies. In many studies, patients with other forms of hypertrophy (*eg*, asymmetric or disproportionate septal hypertrophy) have been included, despite the fact that these forms of hypertrophy are not typically related to hypertension.

**Prognostic Value**

Preliminary follow-up data suggest the prognostic significance of echocardiographically detected LVH in hypertensive patients.\(^7\) Patients with LVH demonstrated by echocardiography have been shown to have an increased risk of mortality, incidence of heart failure, and occurrence of cardiovascular complications, as well as higher blood pressure readings. The use of antihypertensive therapy for the regression of LVH raises several important questions. For example, based upon the severity of LVH, as determined by echocardiography, might we be able to identify a subgroup of hypertensive patients at an increased risk of sudden death?\(^8\) Also, does this regression or reduction in the mass of a hypertrophied ventricle constitute an independent therapeutic goal of antihypertensive therapy,\(^9\) influence cardiac performance and prognosis, or, in fact, constitute a risk in itself to the patient?\(^9\)

Short-term reproducibility studies of measurements of left ventricular mass have yielded poor results. Also, these results have rarely been used as “controls” in reports of studies on the effect of antihypertensive therapy on left ventricular mass. Uncertainty also persists with regard to the optimal blood pressure parameters to be used for correlating the reduction of LVH and the efficacy of various therapies. Data have been reported that the relationship between blood pressure and left ventricular mass did not improve when the averages of all blood pressure readings obtained during a 24-hour period of continuous monitoring, rather than casual blood pressures, were used to establish the degree of hypertension.\(^10\) This observation contrasts with previous reports. Devereux and others\(^11\) have suggested that blood pressure measured during normal daily physical activities is more clearly related to left ventricular mass than casual or 24-hour blood pressure readings.

The best population of patients for the determination of LVH and the efficacy of various therapies remains to be defined. It is possible that echocardiographic studies should optimally be done in newly diagnosed untreated patients with hypertension. Studies using patients previously receiving antihypertensive drugs, including diuretics, β-blockers, and/or vasodilators risk the residual effect(s) of these drugs on cardiac mass measurements and transient changes in left ventricular performance. On the other hand, although results in such a highly selected group of patients may be scientifically sound. They may not be applicable to results obtained in routine daily practice. The results of various studies can only be compared once corrected for the criteria for selecting patients, including therapeutic status, duration of therapy, severity of hypertension, and the presence or absence of concentric LVH.

A review of these nonoptimal clinical studies in hypertensive patients leads to the observation that antihypertensive therapy which reduces blood pressure and inhibits the activity of the sympathetic nervous system and/or the renin-angiotensin system (or both) may result in a decrease in left ventricular mass, whereas those agents that directly or indirectly stimulate the sympathetic and renin systems might be less efficacious in reducing left ventricular muscle mass even if they have antihypertensive actions. The effect of antihypertensive treatment on left ventricular mass is probably dependent on the primary factor(s) responsible for the hypertrophy. In patients with severe hypertension, especially when associated with marked increases in left ventricular muscle mass, it is likely that the high blood pressure itself is the dominant cause of the hypertrophy, and any form of treatment that effectively lowers blood pressure should reduce LVH. On the other hand, hypertrophy associated with earlier and milder forms of hypertension might be more dependent on the sympathetic nervous system, and reducing blood pressure levels alone may not be sufficient to alleviate the hypertrophy.\(^6\) Although these statements are in agreement with results of studies in animals and are supported by numerous studies in hypertensive patients, they will have to be confirmed in large, long-term multicenter trials in hypertensive patients.
patients. Such studies will also resolve the controversy on the benefit of regression of left ventricular mass.

**Conclusion**

Echocardiography is currently considered to be the method of choice for the diagnosis of LVH in hypertensive patients, as well as for the evaluation of the effect of antihypertensive therapies on left ventricular mass regression in patients with essential hypertension. Although the echocardiogram is more time-consuming to obtain and more expensive than the ECG or the chest x-ray film, it appears to possess overall superior sensitivity for diagnosing LVH in patients with elevated blood pressure of varying severity. The echocardiogram has also proven to be a useful tool in observing and assessing the regression of these complications as a result of pharmacotherapy. However, the true prognostic value of echocardiography awaits future clinical investigation, particularly large, long-term multicenter trials in hypertensive patients, to determine definitively the optimal criteria for its successful application.

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