Editorial Comment

Left Ventricular Mass Development Versus Disease

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In adults, there exists a relation between elevated systolic blood pressure and echocardiographically determined enlarged left ventricular mass. Because systolic blood pressure is a recognized risk factor for subsequent coronary heart disease and is thought to be causally related to cardiac hypertrophy, it has been hypothesized that greater left ventricular mass, that is, left ventricular hypertrophy, is itself a risk factor. There is epidemiological evidence to support such a hypothesis. In the Framingham study, patients who had hypertension and electrocardiographically determined left ventricular hypertrophy had a worsened prognosis for survival; both all-cause mortality and cardiovascular disease mortality were much higher than in a comparable group with hypertension alone. A similarly worsened prognosis has been found in hypertensive patients who have echocardiographic left ventricular hypertrophy. Moreover, recent studies have found that left ventricular hypertrophy determined by echocardiography is a reliable predictor of subsequent coronary heart disease events and affords information beyond that provided by traditional risk factors.

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In addition to the relation of left ventricular mass to systolic blood pressure, there exists an important relation of left ventricular mass to body size measures such as weight, height, and body surface area. To achieve clinical comparability, some investigators have used body surface area to index left ventricular mass. However, because in adults obesity appears to be a powerful correlate of increased left ventricular mass, Levy et al suggested that left ventricular mass should be indexed by height. To summarize, in adults, 1) larger people have larger hearts, 2) cardiac hypertrophy often occurs in patients with hypertension, and 3) cardiac hypertrophy is a predictor of subsequent coronary heart disease events beyond other more traditional risk factors.

In their article in this issue of Circulation, Daniels et al report a group of children who were considered to be hypertensive because their blood pressure remained persistently above the 95th percentile for age and sex. These investigators found that instead of the expected 5%, 38.5% of these adolescents had a left ventricular mass index (divided by height) greater than the 95th percentile. A recent analysis of the relation of cardiac mass to exponential changes in body surface area points out the weakness of indexing to the linear measure of height. Indexing by height may well introduce the bias of a higher-than-expected left ventricular mass in larger adolescents. Daniels et al, in their report, used regression methods to model the determinants of the observed increased mass. Both weight and systolic blood pressure were found to be powerful univariate determinants of left ventricular mass. However, in the multivariate models, once the effect of weight was considered in the model, systolic blood pressure no longer remained a predictor, suggesting a high correlation between these two variables.

The implication of the association of increased left ventricular mass with persistently elevated blood pressure is that these adolescents have the “disease” hypertension with the “target organ” response of cardiac hypertrophy. The dilemma is that both blood pressure and left ventricular mass increase during childhood and both variables are closely associated with increases in body weight. These two variables are so closely associated in childhood that the amount of left ventricular mass at one measurement is a predictor of resting blood pressure at future measurements.

The strong univariate relation of weight and systolic blood pressure, coupled with the failure of systolic blood pressure to remain a predictor in the multivariate model, gives rise to at least two alternative explanations for the occurrence of increased left ventricular mass in children at the upper part of the distribution for blood pressure: 1) an increase in left ventricular mass occurs in response to persistent elevations in blood pressure independent of maturational changes and therefore is a pathological consequence of hypertension, or 2) elevated blood pressure and increased left ventricular mass are linked to...
a common genetic mechanism, which regulates both growth and blood pressure.

Clearly, in adults left ventricular hypertrophy is a frequent sequela of severe hypertension and is thought to be causally linked to the subsequent occurrence of congestive heart failure.11 Studies of children and adolescents have found increased left ventricular mass with persistently elevated blood pressure.12,13 Zahka et al14 found that not only was left ventricular mass greater in hypertensive adolescents as compared with that in an age-matched, but was also greater when compared with that in a normotensive obese group of children of similar age.

However, we know that in childhood both blood pressure15 and body size16 are under strong genetic control. In 11-year-old twins, over two thirds of the variability of systolic blood pressure and over 90% of the variability of weight is genetic. Annest et al17 found that blood pressure, height, and weight were strongly influenced by genetic factors in childhood. Adams et al18 studied college twins during extensive exercise training and concluded that heart size determined both by the electrocardiogram and the echocardiogram was under the influence of “cultural familial influences” that included both genetic and environmental effects. Perhaps genes regulating body size influence both heart size and blood pressure.

Daniels et al19 make several recommendations to secondarily prevent excessive left ventricular mass in hypertensive adolescents, based on the determinants of left ventricular mass found in their models. These are to 1) reduce obesity and 2) restrict salt intake. Neither of these recommendations is without intrinsic merit; the major reservation is whether they will be successful secondary prevention strategies.

Weight loss in obese hypertensive adults has been documented to reduce both blood pressure and left ventricular mass.19 This strategy may be very effective in obese adolescent hypertensive patients.

In population studies, the amount of salt eaten by a people relates to the mean blood pressure of that population. Recently, the INTERSALT study examined sodium excretion in study populations from 32 countries.20 The investigators found a significant relation between sodium excretion and systolic blood pressure. In a group of high school children, Ellison et al21 found that manipulating the amount of salt in the diet caused changes in the students' blood pressure levels. However, these observations do not necessarily transfer directly to the clinical care of hypertensive patients. In the study by Daniels et al,8 although sodium intake continued to remain a significant determinant after the consideration of body mass index, the coefficient added only a small contribution towards understanding the variance of left ventricular mass.

I believe that the proposed strategies are most likely to succeed in older adolescent children. In younger adolescents, developmental changes associated with growth may be the most powerful determinants of both blood pressure and left ventricular mass. Perhaps it is more prudent to delay recommendations targeted toward regression of left ventricular mass until puberty is well established. Adolescents who are in the late stages of puberty may have more mature regulation of their blood pressure and left ventricular mass and may therefore be more responsive to these kinds of environmental changes.

References


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