Concentric or Eccentric Hypertrophy: How Clinically Relevant Is the Difference?

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Different combinations of volume and pressure overloads cause different left ventricular (LV) geometric adaptations. Whereas this cause-effect relationship is obvious for valve disease, because clear-cut types of overloads are easily recognizable, in systemic hypertension the type of cardiac load is less evident but at least as important for LV adaptation. Human arterial hypertension encompasses a large range of interactions between volume and pressure overloads, therefore producing a very large spectrum of possible LV adaptations. Based on the assumption that LV geometry is more useful than simple assessment of brachial blood pressure to identify the predominant type of overload, for convenience (and somewhat arbitrarily), we use generated cutoff points to define different LV geometric patterns according to the predominance of one hemodynamic load over the other one.1

When considering together the prevalence of concentric remodeling and concentric LV hypertrophy,1 we can easily conclude that pressure overload is the fundamental abnormality in arterial hypertension, although this is, in most circumstances, associated with some volume component. Thus, under chronic antihypertensive treatments that reduce pressure overload, a consistent reduction of concentric LV geometry can be expected when reduction of LV hypertrophy occurs.

Whether modifications of LV geometry from concentric to eccentric are beneficial beyond the reduction of LV mass has been debated; however, the conclusion has been, in general, that concentric LV hypertrophy is also characterized by greater LV mass than eccentric LV hypertrophy; therefore, these 2 features (ie, concentric geometry and LV hypertrophy) are so interrelated that they cannot to be easily discriminated.2,3 In contrast to this argument, in the presence of normal LV mass, Verdecchia et al4 demonstrated that concentric LV remodeling (ie, with normal LV mass) reflecting a nearly pure pressure overload1 was associated with poor prognosis. However, even in this study,4 although the measurements were in the range of arbitrarily defined normal LV mass, hypertensive patients with concentric LV remodeling exhibited higher values of LV mass than hypertensive patients with normal LV geometry, making it difficult to discriminate the type of LV geometry from the magnitude of LV mass.

Another argument of this issue is now proposed by Muiesan et al,5 who convincingly demonstrated that the normalization of the relation between chamber size and wall thickness has a favorable prognostic value that adds to the recognized positive effect of reducing LV mass. This finding stimulates 2 considerations.

In the Muiesan study,5 and in other studies on LV geometry and prognosis, LV hypertrophy has been assessed by traditional normalization of LV mass for body surface area, a method that increases the proportion of patients with concentric LV remodeling and apparently “normal LV mass,” caused by the exaggerating effect of body fat included in the computation of body surface area.6,7 Normalization for height2,8 reduces the proportion of overweight/obese subjects in the concentric LV remodeling cluster and increases the proportion of subjects with recognized concentric LV hypertrophy.8 Thus, the favorable change from concentric to normal (eccentric) LV geometry found by Muiesan et al5 probably included a number of patients initially classified in the group with concentric LV remodeling. These patients were not considered among those in whom regression of LV hypertrophy could have occurred because of their initial classification in the group without LV hypertrophy (by body surface area index). Thus, the effect of regression of LV concentric geometry independent of regression of LV hypertrophy could have been attenuated by using normalization for height2,8,6 which increases the proportion of individuals with concentric LV hypertrophy and reduces the prevalence of concentric LV remodeling.8

Even taking the potential body size adjustment limitations into account, the Muiesan study5 clearly indicates that when cardiovascular disease has not yet occurred, the natural prognostically adverse LV geometric pattern in hypertensive patients is concentric (ie, because of pressure overload). This finding is in line with an impressive series of experimental and clinical data.

Physiologically, there is little adaptive advantage in the concentric LV geometry. The perception that concentric LV geometry in pressure overload is a compensatory mechanism to reduce wall stress to maintain systolic performance should be probably revised, because LV function may be normally maintained even in the presence of elevated wall stress.9,10 A scenario alternative to the stress-correction theory is that cardiomyocyte hypertrophy and structural disarray are produced by pressure-overload neurohormonal activation, causing abnormal contractility and distensibility, which is at least
partly compensated by increasing wall thickness, which preserves LV function at the chamber level. Although myocardial afterload is the prime stimulus that promotes the cascade of biological events leading to LV hypertrophy, reduction of wall stress induced by concentric hypertrophy would, therefore, be a consequence more than the final result of this adaptive mechanism. In humans, reduced wall stress, caused by concentric LV hypertrophy, is associated with increased cardiovascular risk. Moreover, an excessive prognostically adverse development of LV mass beyond the need to compensate for increased cardiac load is found when LV geometry is concentric.

Structurally, pressure-overload LV hypertrophy is characterized by myocardial alterations at the sarcomeric and extracellular scaffold levels more than is observed in volume overload. When a volume component coexists with the predominant pressure overloads, as in most cases of arterial hypertension with concentric LV hypertrophy, the type of collagen accumulation and the consequent negative mechanical effect are similar to the pressure overload pattern.

Thus, concentric LV geometry is the phenotypic condition associated with more severe hemodynamic and structural abnormalities and represents an unfavorable LV adaptation. This might be the rule in most cases of arterial hypertension when a direct ischemic myocardial insult has not yet occurred.

When progression of LV adaptation is associated with myocardial infarction, LV remodeling occurs and concentric geometry might disappear. Cross-sectionally, hypertensive patients with more eccentric LV geometry are more likely to have coronary artery disease than patients with concentric LV geometry. Thus, there is a high prevalence of eccentric LV geometry during the natural progression of arterial hypertension toward cardiovascular disease, and a high prevalence of eccentric LV geometry once cardiovascular disease has occurred, as a consequence of postinfarction remodeling and impending systolic dysfunction.

Muiesan’s article demonstrates that concentric LV geometry is the predominant and most dangerous adaptive pattern in arterial hypertension before any cardiovascular event has occurred. Progression of the disease can revert to “normal” eccentric LV geometry when post-myocardial infarction remodeling occurs or as a consequence of efficacious antihypertensive therapy. Reverting to normal geometry by antihypertensive therapy reduces the risk of adverse outcome further than can be attributed to regression of LV hypertrophy. However, it remains to be established whether reversal to normal LV geometry can be obtained independently of reduction of LV mass.

References

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