to 80% and using a 5% significance level, to be approximately 29%, not 41%.13

In conclusion, we believe that our study14 was an excellent test of the hypothesis that unloading the reperfused ventricle reduces infarct size. Clearly, a carefully performed study of cardiopulmonary bypass on infarct size in dogs in which collateral flow is used as a covariate in the analysis of infarct size is needed to determine whether their observation is real. If that proves to be true, then carefully designed studies must be performed to exactly determine the critical variable.

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References

Left Ventricular Mass in Hypertension

I read with interest the article by Ganau et al concerning the relation of left ventricular (LV) hemodynamic load and contractile performance to left ventricular mass (LVM) in hypertension.1

Having worked in the same field, I have a particular interest in their results and agree with most of them. Despite its well-known limitations, M-mode echocardiography remains a useful method.

The influence of LV volume load on hypertensive LVM as well as its importance in LVH, whatever the cause, was described in 1984.2 In hypertensive patients, we also found a linear relation between LVM and the left end-diastolic diameter (representing LV diastolic volume). The relation led us to a new concept of the progression of hypertensive cardiomyopathy; it appeared from our experience and research that hypertrophy is a reaction that tends to keep end-diastolic dimension normal or near normal and thereby prevent dilatation that in turn reduces wall stress and oxygen consumption.

The strong dependence of left chamber size on LVM in hypertensive patients led us to search for a similar relation in normal subjects. We were able to define the relation between these two variables and found that it is a double asymptotic curve with a growth-curve appearance. The close relation of LVM and chamber size and the special growth-curve appearance of their relation strongly suggest that the observed relation reflects both physiological and pathological relations.3,4

This curve

\[
\text{Mass} = \frac{800}{1 + e^{-0.8 \times [(1.29 \times \text{diameter}) - 8.07]}}
\]

predicts the value of a theoretical mass for a given diameter, from the growing heart (e.g., infant, child) to physiological hypertrophy (e.g., trained athlete, first stage of hypertension).

The linear relation seen in the hypertensive patients of Ganau et al1 can be explained by the particular position of the hypertensive subjects in the linear portion of the normal relation.

A shift to the right defines eccentric hypertrophy as seen in left-sided volume overload and congestive cardiomyopathy.

In conclusion, I emphasize the clinical importance of the LVM relation to cardiac chamber size. The occurrence of congestive hypertension cardiomyopathy can be detected early, as soon as there is a rightward shift of the calculated mass compared with the normal curve. This relation may also be used in differential diagnosis and in the follow-up of miscellaneous pathologies.

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Reply

Dr. LeBlanc’s report of a strong, positive relation between left ventricular (LV) chamber diameter and LV muscle mass (LVM),1,2 and our finding of a significant relation (r=0.53, p<0.001) between echocardiographic LV chamber diameter and necropsy LVM—which avoids the potential tautology from using the same echocardiographic measurement in both terms in the relation—in 34
Left ventricular mass in hypertension.
H LeBlanc

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