LVH and hypertension: Is treating the pressure not enough?

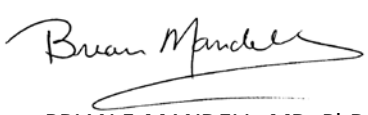
Over the past decades, aggressive antihypertensive treatment has become ingrained in clinical practice. While the optimal target pressure remains controversial, it is accepted that lowering elevated blood pressure reduces the frequency of adverse cardiovascular and cerebrovascular events and limits the progression of renal disease. The pressure itself seems a reasonable primary target of treatment, and despite a few foibles of measurement (see Rafey, *Cleve Clin J Med* 2009; 76:657–662), it is relatively easily and reliably obtained, making the management of this risk factor a cost-effective intervention.

In patients with "borderline" hypertension or those in whom the duration of blood pressure elevation is hard to ascertain, the finding of end-organ damage has traditionally been used as an argument to institute aggressive antihypertensive therapy. In this setting, retinal hypertensive disease, an S4 gallop, and left ventricular hypertrophy (LVH) are often specifically sought.

LVH and otherwise unexplained chronic kidney disease in patients with hypertension have generally been believed to be products of the elevated arterial pressure, and primary treatment has targeted pressure control. Bauml and Underwood, in this issue of the *Journal* (page 381), emphasize some published clinical trial data indicating that LVH may be an independent risk factor for poorer cardiovascular outcome. Even more provocative is the suggestion that LVH can be reversed, as can the associated increased risk of cardiovascular morbidity, independently of the hypertension.

Given our current understanding that LVH, under some conditions, can be induced by products of the renin-angiotensin system, this would suggest that pharmacologic blockade of this enzyme system should have extra benefit, above that seen from other antihypertensive agents. Conceivably, this may be true only in patients with LVH, and the time course of benefit may not directly parallel that seen with the control of hypertension. That theoretically may explain the lack of uniform advantage of angiotensin blockade over other effective antihypertensive approaches.

Since electrocardiography is a specific but not very sensitive test for LVH, the authors suggest that patients with hypertension be routinely screened for LVH using echocardiography. I am not sure the weight of the evidence supports this approach at present, particularly in the current frenzy of cost containment. Nonetheless, this concept warrants consideration, and at the least, large patient databases might be screened retrospectively to further validate or refute the concept that hypertension-associated LVH is an independent, reversible risk factor for cardiovascular morbidity.



BRIAN F. MANDELL, MD, PhD
Editor-in-Chief

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