Echocardiographic Evaluation of Pulmonary Artery Pressure in Patients With Heart Failure

We read with great interest the report by Bursi et al. (1) on the prognostic value of pulmonary pressure in heart failure (HF). The investigators used Doppler-determined pulmonary artery systolic pressure (PASP), which requires an estimation of right atrial pressure by the diameter and respiratory variation of the inferior vena cava. This approach was initially followed to provide values comparable with those of right-heart catheterization. However, the almost arbitrary estimation of right atrial pressure and the use of reference values for PASP derived by right-heart catheterization worsen the reproducibility and reliability of Doppler measurements. We first identified this problem 15 years ago while studying pulmonary hypertension in β-thalassemia, and we proposed the use of tricuspid regurgitant velocity, with a threshold of 2.7 m/s, is the proposed method for the echocardiographic screening of pulmonary hypertension (3).

A tricky aspect in the echocardiographic evaluation of PASP in HF is the loading conditions at the time of examination (4). Patients with acute HF and lung congestion would have considerably high PASP reflecting the acutely increased left ventricular filling pressures, not the true, steady-state passive and active component of pulmonary hypertension. This parameter should be taken under consideration while evaluating patients with acute HF, and the examination should be repeated after clinical stabilization. Furthermore, it should be stressed that ejection fraction is a rough estimate of systolic left ventricular function, and this may account for the lack of association between PASP and systolic dysfunction severity observed by Bursi et al. (1).

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http://dx.doi.org/10.1016/j.jacc.2012.02.050

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Reply

We thank Drs. Farmakis and Aessopos for the opportunity to clarify important aspects of the prognostic use of pulmonary artery systolic pressure (PASP) measured by Doppler in patients with heart failure (1). Although Doppler is the preferred tool to measure pulmonary pressures in practice (2), we agree that the estimation of right atrial pressure has limitations (3).

The upper limit of normal of 2.7 m/s corresponding to a peak gradient of 30 mm Hg was tested in a small group of younger (mean age 38.9 ± 12.7 years), healthy, nonsmoking subjects (4). Thus, it is applicable to similar, relatively young populations such as patients with beta-thalassemia or pulmonary arterial hypertension and when right atrial pressure is thought to be normal (5). Hence, this cutoff is of less relevance in older populations with greater comorbidity, as PASP increases with age, and patients with heart failure are elderly. Because there is no universally accepted cutoff value to define pulmonary hypertension, we analyzed the entire distribution of pulmonary pressures and analyzed PASP with tertiles or continuously, rather than applying an arbitrary cutoff. We showed that the higher the PASP, the worse the prognosis, and the estimation of right atrial pressure has no bearing on this continuum of risk.

We defined heart failure by epidemiological criteria, and our goal was not to distinguish the acute increase in filling pressures from the chronic passive or active component of pulmonary hypertension. We demonstrated that Doppler estimation of PASP was feasible in most patients (91%) in the community and that when elevated, it strongly and independently predicted outcome regardless of the mechanism of pulmonary hypertension.
Finally, although ejection fraction is an imperfect tool to measure systolic function, it is the most validated method, is widely available, and is recommended by all guidelines.

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