Journal of the American College of Cardiology Vol. 53, No. 10, 2009

ISSN 0735-1097/09/$36.00

CORRESPONDENCE

Letters to the Editor

Vasodilator Therapy in Cardiac Failure

What Was New Is Old

The paper by Mullens et al. (1) and editorial comment by Yancy (2) draw attention to use of vasodilators in treatment of acute and chronic refractory cardiac failure. The importance of left ventricular (LV) afterload is stressed, but this is described only in terms of peripheral resistance as the ratio of mean arterial pressure and cardiac output, with the latter requiring and justifying right heart catheterization. There is a problem with this approach over and above the risks of Swan-Ganz catheter use; peripheral resistance is only part of LV afterload, which is best expressed as aortic input impedance (3,4). In addition to peripheral resistance, impedance also considers aortic stiffness and wave reflection, and the effects of vasodilator drugs on these (3–5). In the recent articles, brachial systolic pressure is taken as an index of LV afterload, but this is considerably higher than aortic and LV systolic pressure, especially in patients with cardiac failure and during use of vasodilators (4–6). Vascular impedance in cardiac failure during use of vasodilator drugs is not mentioned in either article, but has been described in major journals over the past 3 decades, and forms the basis for modern treatment of this condition (3,4). Central aortic pressure also can be estimated accurately through noninvasive methods (4,6), as can indices of arterial stiffness and wave reflection (4,5).

Persons wishing to apply the principles described by Mullens et al. (1) and Yancy (2) are advised to consider these issues. They can measure LV afterload better, and avoid invasive catheterization completely. They can also obtain a more accurate measure of mean arterial pressure from integration of the arterial pressure waveform using applanation tonometry, rather than estimating this from the inaccurate formula of diastolic + one-third pulse pressure (4).

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doi:10.1016/j.jacc.2008.08.082

Please note: Dr. O'Rourke is a founding director of AtCor Medical Pty Limited, manufacturer of systems for analyzing the arterial pulse.

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Reply

We thank Drs. O'Rourke and Nichols for their enthusiastic interest and insightful comments regarding our report on the potential benefits of sodium nitroprusside (SNP) in the setting of advanced decompensated heart failure (ADHF) (1). We are in complete agreement regarding the many factors that influence left ventricular (LV) afterload, including the concept that aortic impedance can be a more integrated measure of LV afterload. We would like to emphasize that throughout the article there had not been any assertion or assumptions that measuring systemic vascular resistance in ADHF better reflects LV afterload compared with aortic input impedance. It is also not the intention of our retrospective case series to compare the effectiveness or safety of administration of SNP guided by a reduction in vascular resistance or aortic input impedance. In fact, titration of SNP doses was based on achieving a measured target mean arterial blood pressure of 65 to 70 mm Hg and not on achieving a normal derived systemic vascular resistance. Nevertheless, even with this relatively crude method in the absence of specialized equipment, the substantial improvement in cardiac output secondary to sodium nitroprusside therapy was associated with more favorable (rather than adverse) long-term outcomes. Although invasive measurements were used in our protocol, it is not the intention of these data to always imply the need for invasive monitoring, but solely to understand the hemodynamic contributors and subsequent changes induced by sodium nitroprusside during the treatment of ADHF. As with interpreting the clinical utility of any biomarker, there is an important distinction between identifying individual patients who may have the appropriate hemodynamic profiles to benefit from a specific intervention versus using specific indexes of LV afterload as targets of therapeutic interventions. We agree that much promise exists regarding the use of noninvasive hemodynamic monitoring. Nevertheless, in much the same way that pharmacotherapeutics require rigorous placebo-controlled testing in the specific population with the specific treatment goals to be certain of benefit, diagnostic tools intended to guide therapy may require the same validation, especially regarding use in the acutely ill heart failure population.