A Comprehensive Noninvasive Hemodynamic Assessment of Systolic Heart Failure

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The prevalence of systolic and diastolic heart failure increases with advancing age between 5% and 10% of all subjects beyond the age of 60 years. The cause of heart failure in the majority of patients is occlusive coronary artery disease, hypertension, and idiopathic dilated cardiomyopathy. The current estimated costs for the treatment of heart failure in the United States alone are approaching $40 billion per year, and for this reason, heart failure has been identified as a major health care initiative.

Some of these costs could be defrayed if recurrent hospital admissions for serial hemodynamic assessments could be replaced by noninvasive hemodynamic measurements with the use of Doppler echocardiography in a more cost-effective outpatient setting. Although right heart catheterization remains the gold standard for measurement of intracardiac pressures,1,2 enthusiasm for placement of Swan–Ganz catheters has dwindled over the past decade on account of complications that include infection, cardiac perforation, and tamponade. Doppler echocardiography is generally acknowledged to be a noninvasive alternative to Swan–Ganz catheterization for hemodynamic assessment at the bedside.3–8 Hitherto, a number of isolated noninvasive Doppler echo measurements of left ventricular (LV) filling pressure, right atrial pressure, and cardiac output and the changes following load interventions have correlated closely with measurements made by Swan–Ganz catheterization.3,8,9 What has been notable by its absence is a consensus regarding a portfolio of measurements that can be used in all patients with heart failure to provide a comprehensive hemodynamic assessment at the bedside that will determine the subsequent treatment strategy. An important prerequisite of such a panel of hemodynamic tests is that they are quick to perform, easy to interpret, previously validated, and reproducible.

Doppler echocardiography in addition to enabling numerous hemodynamic measurements to be made also provides information about LV size, architecture, myocardial composition, and the extent of remodeling that impact directly on LV function and that complements the hemodynamic data and correlates closely with clinical outcome. These include 3D real-time quantification of LV volumes or 2D LV volumes from biplane orthogonal apical images from both of which ejection fraction can be derived.

Mitral regurgitation is a frequent finding in patients with systolic heart failure that is poorly tolerated, often resulting in escalation of LV dysfunction and symptomatic deterioration. Mitral regurgitation is important to recognize early so that afterload reduction can be achieved either pharmacologically or by percutaneous or surgical mitral valve repair. The hemodynamic abnormalities and the mechanism of mitral regurgitation in individual patient with heart failure needs to be accurately determined before interventions in high-risk patients with low ejection fraction are contemplated. This is also important because in the majority of patients with heart failure, the mitral valve leaflets are anatomically normal. The mechanism of mitral regurgitation in heart failure results from either enlargement of the mitral annulus or derangement of the mitral subvalve apparatus due to LV dilation or due to ischemia. Ischemia alters the timing and coordination of contraction of the papillary muscles and may only occur on exertion. Establishing a mechanism for mitral regurgitation in heart failure is crucial so that the treatment is congruent with the mechanism, because treatment can vary from the addition of a vasodilator to percutaneous myocardial revascularization, to surgical mitral valve repair/replacement.

Fortunately, the commonly used technique of color flow Doppler velocity mapping is exquisitely sensitive to the presence of mitral regurgitation and enables a semiquantitative assessment of the severity of mitral regurgitation in terms of the length, width, or area of the mitral regurgitant jet.10 However, these metrics that describe the morphology of regurgitant mitral jets are influenced by a number of factors that limit jet propagation. These include left atrial size, left atrial compliance, and jet collision with the left atrial wall, all of which lead to underestimation of the severity of mitral regurgitation. Quantification of the severity of mitral regurgitation is best achieved by the proximal iso-velocity surface area method from which regurgitant volume, regurgitant fraction, and regurgitant orifice area can be obtained.

In this issue of Circulation: Heart Failure, Temporelli et al11 assess the accuracy and clinical applicability of Doppler echocardiography in determining the entire hemodynamic profile in individual patients with advanced heart failure and significant LV systolic dysfunction. The panel of tests that the authors advocate for a comprehensive hemodynamic assessment in systolic heart failure is shown in the Table. The study design was prospective but involved only 43 consecutive patients with advanced heart failure, New York functional class III/IV.11 All patients were in sinus rhythm with ejection...
Eighty percent had severe mitral regurgitation and cause of heart failure. Mean ejection fraction was low at 19%. Two thirds of the patients had coronary artery disease as the patients with heart failure, in that the mean age was 58 years, and there were 5 times as many males as there were females. Two thirds of the patients had coronary artery disease as the cause of heart failure. Mean ejection fraction was low at 19%. Eighty percent had severe mitral regurgitation and ∼50% had severe tricuspid regurgitation assessed by Doppler color flow velocity mapping of the regurgitant jets. Of note, the acquisition of complete Doppler echocardiographic hemodynamic profiling was obtained in every single patient because the endocardial boundaries and Doppler velocity signals were of the highest quality, which is not so unusual in large hearts with diminished endocardial motion due to poor contractile function.

The overwhelmingly important message from these investigators is the robust and highly significant correlations between the noninvasive and the invasive hemodynamic measurements of all predefined variables. Furthermore, the mean relative differences between estimated and measured variables by Bland–Altman analysis were small. The graphic display of the relationships between noninvasive estimates and direct measurement by invasive techniques for each hemodynamic variable shows close correlations with coefficients (r) ranging from 0.93 to 0.97. The formulations for estimating pulmonary capillary wedge pressure (PCWP), right atrial pressure, and pulmonary vascular resistance (PVR) have been derived and validated previously by the authors who have long-standing clinical experience with their use.3,4,6

The strong agreement between PVR estimated noninvasively and that measured invasively was unexpected because measurements are technically challenging when the requisite velocity signals are so diminutive. In addition, noninvasive estimates of PVR also entailed multiple accurate assessments that include the pre-ejection period and acceleration time, which are short-lived. However, the anticipated discrepancies and differences did not occur. There was a similar robust correlation observed between invasive and noninvasive measurement of PVR when it was indexed to unit body mass. Accurate assessment of PVR and PVR index is critically important in patients with advanced heart failure because fixed severe elevation of the PVR is a contraindication to heart transplantation alone. Furthermore, irreversibly elevated PCWP is a powerful predictor of a poor prognosis and adverse clinical outcome in patients with advanced heart failure. Not only did noninvasive assessment of PCWP and PVR correlate with invasive measurements at baseline but also the changes after controlled reduction of afterload with intravenous nitroprusside measured noninvasively paralleled the changes measured invasively. Linear regression analysis after perturbations in loading conditions confirmed the strong correlations between invasive and noninvasive measurements. An additional strength of the measurements of PCWP, PVR, and cardiac output afterload manipulations was the low (<4%) interobserver variability.

These authors have not only demonstrated that Doppler echocardiographic measurement correlate closely with Swan–Ganz catheter measurements over a range of hemodynamic parameters that are of pivotal importance in advanced heart failure but also shown that these 2 techniques are essentially interchangeable for hemodynamic assessment. They have also integrated the extensive hemodynamic profiling described in the Table, with the baseline clinical demographics that they use routinely at the bedside in all of their patients with heart failure. Furthermore, apart from PVR, which is a little complicated, the remaining Doppler echocardiographic hemodynamic data are simple and easy to do and, importantly, can be easily acquired by all sonographers and physicians. The next step is to perform the requisite serial assessment of central hemodynamics with the use of Doppler echocardiography in an outpatient setting and avoid the high costs of recurrent hospital admissions for Swan–Ganz catheterizations without compromising quality of patient care.

It is worthwhile reviewing some of the components of the panel of hemodynamic tests used to risk stratify patients with heart failure because they largely determine the treatment strategy selected for further management. First, pulmonary artery systolic pressure, which is usually calculated as the sum of right atrial pressure derived from the degree of inferior vena caval collapse and the right atrial/right ventricular systolic pressure gradient, is obtained by using the Bernoulli equation. When this algorithm was first introduced for assessing pulmonary artery systolic pressure, there was general agreement between invasive and noninvasive techniques.12,13 However, subsequent studies have demonstrated discrepancies and inconsistencies between noninvasive and invasive estimates of pulmonary artery systolic pressure, which were especially common in patients with moderate and severe pulmonary hypertension.14 Temporelli et al8,11 used an alternative method for estimating pulmonary artery systolic pressure in which right atrial pressure was estimated from the acceleration rate of early right ventricular rapid filling that correlated with the pulmonary artery pressure recorded by Swan–Ganz catheterization much more closely (r = 0.97) than did estimation of pulmonary systolic pressure using the degree of inferior vena caval collapse as an indicator of right atrial pressure.

The authors elected not to explore a panel of measurements of diastolic function beyond selecting PCWP as a surrogate of mean left atrial pressure in this patient cohort with predominantly systolic heart failure. They readily acknowledge that a single variable does not adequately describe the complex interaction between the active and the passive material
properties of the myocardium coupled with the visco-elastic properties of the extracellular matrix. PCWP was not estimated from the E/Ea ratio by tissue Doppler but from the deceleration time of early mitral filling that correlates inversely with PCWP by using the formulation:

$$PCWP = 32.16 + (\frac{-0.1045E}{A}) + (0.1345A) + (\frac{-0.17DT}{E}) + (4.95E/A).$$

PCWP estimated using the algorithm above correlated well ($r=0.93$) with measurements made invasively by Swan–Ganz catheter. PCWP is a powerful independent predictor of clinical outcome in systolic heart failure. Selection of a single measure of diastolic function is inconsistent with their rationale used for selecting a complete panel of hemodynamic measurements of systolic LV function. In addition, patients with heart failure and a mean ejection fraction of 19±5% have a high likelihood of having concomitant diastolic dysfunction. The hemodynamic assessment of LV diastolic function can also be assessed using E/Ea ratio by tissue Doppler propagation velocity by color M-mode echocardiography, but evaluation of LV diastolic function is not the major thrust of the current study.

The truly astonishing feature of this novel and interesting study is the very close correlations between measurements made noninvasively by Doppler echocardiography and invasively by Swan–Ganz catheter of a number of important hemodynamic variables including cardiac output, PCWP, and PVR measured. These unexpected but remarkable levels of correlation likely reflect the high quality of both echocardiographic images and Doppler recordings coupled with the extensive experience of the investigators. The addition of 3D real-time echocardiography for more precise volume estimation and speckle tracking to assess multi-dimensional LV global and regional strain to the authors’ current panel of noninvasive investigations will complete their diagnostic armamentarium.

In summary, Temporelli et al have shown with indelible clarity that hemodynamic monitoring in patients with systolic heart failure can be achieved noninvasively with Doppler echocardiography just as well as with right heart catheterization. What remains to be determined is whether these noninvasive measurements predict clinical outcome, whether cost savings per patient avoiding admission for invasive monitoring can be quantified in the current climate of fiscal containment, and perhaps most importantly, whether other investigators can reproduce these impressive results.

Disclosures

None.

References


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