Doppler Echocardiography in Advanced Systolic Heart Failure
A Noninvasive Alternative to Swan-Ganz Catheter

Pier Luigi Temporelli, MD; Francesco Scapellato, MD; Ermanno Eleuteri, MD; Alessandro Imparato, MD; Pantaleo Giannuzzi, MD

Background—Although several studies have demonstrated a good correlation between Doppler echocardiographic and invasive measurements of single hemodynamic variables, the accuracy of echocardiography in providing a comprehensive assessment in individual patients has not been validated. The aim of this study was to assess the accuracy and clinical applicability of Doppler echocardiography in determining the entire hemodynamic profile in stable patients with advanced systolic heart failure.

Methods and Results—Doppler echocardiography and Swan-Ganz catheterization were simultaneously performed in 43 consecutive patients with advanced heart failure. Echocardiographic data required for estimation of right atrial, pulmonary artery systolic, and pulmonary capillary wedge pressures; cardiac output; and pulmonary vascular resistance were obtained and compared with hemodynamic data. For all variables, invasive and noninvasive hemodynamic values were highly correlated ($P<0.0001$), with very low bias and narrow 95% confidence limits. In 16 patients with elevated pulmonary vascular resistance ($>3$ Wood U) and pulmonary capillary wedge pressures ($>20$ mm Hg) at baseline, hemodynamic and Doppler measurements were simultaneously repeated after unloading manipulations. Absolute values and changes of pulmonary vascular resistance and pulmonary capillary wedge pressures after unloading were still accurately predicted ($r=0.96$ and $r=0.92$, respectively).

Conclusions—Doppler echocardiography may offer a valid alternative to invasive cardiac catheterization for the comprehensive hemodynamic assessment of patients with advanced heart failure, and it may assist in monitoring and optimization of therapy in potential heart transplant recipients. (Circ Heart Fail. 2010;3:387-394.)

Key Words: catheterization ■ echocardiography ■ heart failure ■ hemodynamics

Reliable estimates of hemodynamic variables in patients with advanced heart failure (HF) are crucial for both the optimization of medical therapy and the identification of potential heart transplant recipients.1–3 Swan-Ganz catheterization remains the gold standard for the assessment of central hemodynamics, but it is invasive, not without risks, and not practical for widespread application. Doppler echocardiography is now well recognized as an accurate bedside noninvasive alternative to Swan-Ganz catheterization for various hemodynamic assessments.4–12 In particular, much attention has been given to the estimation of left ventricular filling pressures in different clinical conditions, including HF.5,6,13–17 Although several studies have demonstrated a good correlation between Doppler echocardiographic and invasive measurements of single hemodynamic variables, the accuracy of this noninvasive technique in providing a comprehensive assessment (all hemodynamic parameters taken together) in individual patients has been poorly validated. Furthermore, the highly elaborate and hardly reproducible noninvasive methods applied in some studies are not conclusive to this end. In the clinical setting, before definitive approval of Doppler echocardiography as a noninvasive alternative to Swan-Ganz catheterization can be given, a close agreement between the 2 methods systematically applied to individual patients must be demonstrated. Finally, along with reliability, easy applicability of the noninvasive method also needs to be demonstrated if it is to be recommended for widespread use at the patient’s bedside. Thus, the aim of this study was to assess the accuracy and clinical applicability of Doppler echocardiography in determining the entire hemodynamic profile in individual patients with advanced HF and significant left ventricular systolic dysfunction.

Methods

Patients
This prospective study included patients with sinus rhythm with advanced HF (New York Heart Association functional class III or...
IV) secondary to both ischemic- or idiopathic-dilated cardiomyopathy and severe systolic dysfunction (defined by echocardiographic ejection fraction ≤35%) who were undergoing Swan-Ganz catheterization for assessment of hemodynamic suitability for heart transplantation. Of 47 consecutive patients admitted to our heart failure unit for possible selection for heart transplantation, 4 patients were excluded because of mechanical mitral prosthesis (n = 1), permanent electroinduced rhythm (n = 1), or chronic atrial fibrillation (n = 2). Doppler echocardiography and right heart catheterization were simultaneously performed. Written informed consent was obtained from participants, and the study was approved by the ethics committee of our institution.

**Echocardiographic Studies**

Patients were imaged in the left lateral decubitus position with a Hewlett-Packard ultrasound unit (HP Sonos 5500) equipped with a 2.5-MHz transducer and connected to computerized hemodynamic instruments so that it was possible to record Doppler and hemodynamic traces on line. Left ventricular volumes were calculated from orthogonal apical views using the biplane area-length method. Ejection fraction was derived from the standard equation. Both mitral and tricuspid regurgitation were detected and graded by using color flow Doppler according to previous reported criteria that took into account both the width and depth of regurgitant jets in relation to the size of the receiving chamber from multiple views and the size of the jet at the regurgitant orifice.18,19 Studies were recorded for later analysis. Strip chart recordings of all spectral Doppler images were obtained for offline analysis. For each Doppler-based measurement, estimates were obtained from 3 cardiac cycles and averaged.

**Doppler Echocardiographic Data Analysis**

**Cardiac Output**

Cardiac output (CO) was calculated as the product of the heart rate, left ventricular outflow tract (LVOT), velocity time integral (VTI), and the area of the outflow tract as follows: CO = (heart rate)(LVOT VTI(\(\pi\))(outflow tract diameter/2)) \(^2\). No patients had aortic stenosis or aortic prosthesis. The aortic VTI by pulsed Doppler with the sample volume positioned in the middle of the outflow tract below the aortic cusps was assessed with the HP Sonos 5500 unit. The outflow tract diameter was measured in the parasternal long-axis view.4,20

**Pulmonary Capillary Wedge Pressure**

Pulmonary capillary wedge pressure (PCWP) was estimated from the deceleration time (DT) of early mitral filling as follows: PCWP = 32.16 + (–0.1045E) + (0.1345A) + (–0.17 DT) + (4.95 E/A). DT is the time between the peak E wave and the upper deceleration slope extrapolated to the zero baseline. The linear inverse correlation between mitral DT thus calculated and PCWP has been previously reported in patients with left ventricular systolic dysfunction and congestive HF both in sinus rhythm and in atrial fibrillation.6,14

**Mean Right Atrial Pressure**

Mean right atrial pressure (RAP) was estimated from the acceleration rate \((cm/s^2)\) of early tricuspid filling on the pulsed-wave Doppler signal as follows: RAP = –1.263 + 0.01116 × Ac (where Ac indicates acceleration rate), as previously reported.10 Acceleration rate was measured as the slope of the segment between start and the peak of the E wave on tricuspid flow.

**Pulmonary Artery Systolic Pressure**

Pulmonary artery systolic pressure (PASP) was calculated as the sum of estimated RAP and the gradient between peak right ventricular systolic pressure and the estimated RAP; this was later obtained by application of the modified Bernoulli equation to the peak velocity of the continuous-wave Doppler tricuspid regurgitation signal (VTr max) as follows: PASP = RAP + 4VTr max.2,3,11,22

**Pulmonary Vascular Resistance**

Pulmonary vascular resistance (PVR) was calculated as follows:12

\[ PVR = \frac{PAMP}{CO} \]

Where PAMP indicates mean pulmonary arterial pressure and CO, cardiac output.

**Right Heart Catheterization**

A 7F Swan-Ganz catheter (Baxter Healthcare Corp, Edwards Critical Care Division) was introduced by using the Seldinger technique through an internal jugular vein and positioned under fluoroscopic guidance in a pulmonary artery. After a 10-min rest for stabilization, consecutive standard measurement of RAP, PASP, pulmonary artery diastolic pressure, and PCWP was obtained from the digital monitor. Pulmonary artery mean pressure (PAMP) was the digital average of the PASP and pulmonary artery diastolic pressure. CO was determined by the cold saline thermodilution technique. Cardiac index was calculated as CO/body surface area. PVR is expressed in Wood units and was calculated with the following standard formula: PVR = (PAMP – PCWP)/CO.

**Statistical Analysis**

Echocardiographic and hemodynamic data were expressed as mean±SD of subject values. The Pearson r coefficient was used to compare the invasive and noninvasive hemodynamic variables. A P value <0.05 was considered statistically significant. The Bland-Altman plot23 was used to assess methodological agreement in individual patients. Interobserver variability was evaluated by calculating the mean±SD of the differences between the measurements of 2 echocardiographic observers (P.L.T and F.S.). In addition, mean percent error was estimated as the absolute difference between the means of each set of measurements divided by their average.

**Results**

This study included 43 patients (36 men) with a mean age of 54±8 years. Clinical and 2D echocardiographic data are presented in Table 1. The cause of HF was predominantly coronary artery disease (67%). The mean left ventricular ejection fraction was 19±5%. Moderate-to-severe mitral regurgitation was detected in most (80%) of the patients (severe regurgitation in 35%), whereas moderate-to-severe tricuspid regurgitation was present in ∼50% (severe regurgitation in 15%). Acquisition of a complete Doppler echocardiographic hemodynamic data set was adequate in all patients. Indeed, in patients with advanced systolic HF with a severely enlarged left ventricle, the quality of the images usually is very good, explaining why in this study no patient was excluded because of poor echocardiographic data quality.

**Relation of Noninvasive to Invasive Variables**

Table 2 shows the invasive hemodynamic variables, and the correlation between the 2 methods is presented in Table 3. For all variables, invasive and noninvasive hemodynamic values were strongly correlated (P <0.001) without significant differences for any single variable (Figure 2). On Bland-Altman plot analysis, the mean relative difference between all measured and estimated variables was very low.
for the whole population, indicating the absence of systematic error (Figure 3).

Variability in the measurement of baseline Doppler indexes was evaluated in 30 consecutive patients by 2 independent observers. Interobserver variability for all Doppler variables was small (CO, 2.4%; RAP, 3%; PCWP, 2.4%; PASP, 4.1%; PVR, 3.6%) (Table 4).

**Table 1. Clinical and 2D Echocardiographic Parameters**

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Value</th>
<th>Range</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age, y</td>
<td>54±8</td>
<td>37–65</td>
</tr>
<tr>
<td>Male/female sex</td>
<td>36/7</td>
<td></td>
</tr>
<tr>
<td>Coronary artery disease, n (%)</td>
<td>29 (67)</td>
<td></td>
</tr>
<tr>
<td>Heart rate, bpm</td>
<td>74±14</td>
<td>55–110</td>
</tr>
<tr>
<td>LVEDVI, mL/m²</td>
<td>145±41</td>
<td>85–252</td>
</tr>
<tr>
<td>LVEF, %</td>
<td>19±5</td>
<td>10–34</td>
</tr>
<tr>
<td>Left atrial diameter, mm</td>
<td>54±6</td>
<td>40–66</td>
</tr>
<tr>
<td>RVEDD, mm</td>
<td>47±8</td>
<td>29–68</td>
</tr>
<tr>
<td>MR, mild/moderate/severe</td>
<td>9/19/15</td>
<td></td>
</tr>
<tr>
<td>TR, mild/moderate/severe</td>
<td>22/15</td>
<td></td>
</tr>
</tbody>
</table>

Data are presented as mean±SD, unless otherwise indicated. LVEDVI indicates left ventricular end-diastolic volume index; LVEF, left ventricular ejection fraction; RVEDD, right ventricular end-diastolic diameter; MR, mitral regurgitation; TR, tricuspid regurgitation.

**Table 2. Invasive Hemodynamic Variables**

<table>
<thead>
<tr>
<th>Variable</th>
<th>Mean±SD</th>
<th>Range</th>
</tr>
</thead>
<tbody>
<tr>
<td>CO, L/min</td>
<td>3.7±1.0</td>
<td>2.6–6.3</td>
</tr>
<tr>
<td>Cardiac index, L/(min·m²)</td>
<td>2.1±0.6</td>
<td>1.4–3.1</td>
</tr>
<tr>
<td>PCWP, mm Hg</td>
<td>19.1±8.5</td>
<td>4–37</td>
</tr>
<tr>
<td>RAP, mm Hg</td>
<td>5.0±3.8</td>
<td>2–17</td>
</tr>
<tr>
<td>PASP, mm Hg</td>
<td>45.8±16.3</td>
<td>11–73</td>
</tr>
<tr>
<td>PVR, Wood U</td>
<td>2.5±1.7</td>
<td>0.5–7.2</td>
</tr>
<tr>
<td>PVRI, Wood U×m²</td>
<td>4.65±2.2</td>
<td>0.92–12.1</td>
</tr>
</tbody>
</table>

Data are mean±SD or number of patients. CI indicates cardiac index; CO, cardiac output; PASP, pulmonary artery systolic pressure; PCWP, pulmonary capillary wedge pressure; PVR, pulmonary vascular resistance; RAP, right atrial pressure; PVRI, PVR index.

**Figure 1.** Continuous-wave Doppler recording of tricuspid regurgitant flow (top) and pulsed-wave Doppler recording of pulmonary flow (bottom) in the same patient with invasive PVR=3.3 Wood U. AcT, acceleration time; CO, cardiac output; EjT, ejection time; PEP, pre-ejection period; PAPM, pulmonary mean arterial pressure; PCWP, pulmonary capillary wedge pressure; PVR, pulmonary vascular resistance; TT, total time.

**Figure 2.** Equation for the computation of pulmonary vascular resistance (PVR).

\[
PVR = \frac{PAPM - PCWP}{CO} = 3.3 \text{ Wood } U
\]

\[
PVR = \frac{(PEP/AcT)}{TT} = 3.2 \text{ s}^{-1}
\]

\[
TT = PEP + EjT
\]
predictor of poor prognosis, 16 patients with baseline PVR >3 Wood U and PCWP >20 mm Hg underwent unloading nitroprusside infusion on continuous Swan-Ganz and Doppler monitoring. After unloading, mean PVR decreased significantly from 4.4 \pm 1.6 mm Hg to 2.3 \pm 1.1 mm Hg, and PCWP decreased from 25 \pm 4 mm Hg to 13 \pm 3 mm Hg. Figure 4 presents graphs showing the correlation of the changes obtained by means of the 2 methods (echocardiography versus catheterization) after loading manipulations in PVR, PCWP, and CO. Linear regression analysis after unloading manipulations confirmed the strong correlation between invasive and noninvasive measures (PVR, \( r = 0.96 \); PCWP, \( r = 0.92 \)) seen at baseline. Interobserver variability for measurement of PCWP, PWR, and CO assessed immediately after unloading manipulations was 3.8%, 4.2%, and 4.0%, respectively.

### Table 3. Agreement Between Invasive and Echocardiographic Hemodynamic Measures

<table>
<thead>
<tr>
<th>Measure</th>
<th>( r )</th>
<th>Difference Between Methods</th>
<th>Limits of Agreement</th>
</tr>
</thead>
<tbody>
<tr>
<td>RAP, mm Hg</td>
<td>0.96</td>
<td>(-0.09)</td>
<td>(-0.38 \pm 0.21)</td>
</tr>
<tr>
<td>PASP, mm Hg</td>
<td>0.97</td>
<td>(-0.95)</td>
<td>(-2.08 \pm 0.17)</td>
</tr>
<tr>
<td>PCWP, mm Hg</td>
<td>0.93</td>
<td>0.23</td>
<td>(-0.76 \pm 1.21)</td>
</tr>
<tr>
<td>PVR, Wood U</td>
<td>0.96</td>
<td>0.02</td>
<td>(-0.12 \pm 0.15)</td>
</tr>
<tr>
<td>PVRI, Wood U</td>
<td>0.95</td>
<td>0.08</td>
<td>(-0.09 \pm 0.249)</td>
</tr>
<tr>
<td>CO, L/min</td>
<td>0.94</td>
<td>(-0.06)</td>
<td>(-0.81 \pm 0.07)</td>
</tr>
</tbody>
</table>

PVRI indicates PVR index (see Figure 3).

Besides basic clinical evaluation, comprehensive hemodynamic assessment plays a key role in the management of patients with HF, particularly those with advanced HF and who are potential candidates for heart transplantation. In this study, we demonstrated that a complete noninvasive Doppler echocardiographic hemodynamic evaluation is feasible for clinical application in individual patients with systolic HF (particularly in those awaiting transplantation while in stable conditions) in whom the serial assessment of central hemodynamics, chiefly PVR, is most crucial, thus avoiding the risk, cost, and discomfort associated with Swan-Ganz catheterization.

A number of studies\(^4\)\(\text{–}16\) have demonstrated a significant correlation between Doppler echocardiographic and invasive measurement in relation to single hemodynamic parameters, separately addressed, in different subsets of patients. However, very few studies have aimed to validate Doppler echocardiography as a real noninvasive alternative to Swan-Ganz catheterization for the complete hemodynamic evaluation.\(^{11}\) In addition, some studies adopted highly elaborate and poorly reproducible noninvasive methods, whereas easy applicability, along with reliability, of the method is necessary if it is to be proposed for widespread use.

### Echocardiography as a Hemodynamic Imaging Tool in the Real World

To achieve results easily applicable in the clinical setting, we precisely tested the methods routinely performed at the patient’s bedside of our heart failure unit. Some of these techniques used to estimate PCWP, RAP, and PVR were...
validated years ago in our echocardiography laboratory and are now part of our echocardiographic report for patients with systolic HF. All measures can be obtained by all trained sonographers or physicians. In fact, the acquisition of a complete Doppler echocardiographic hemodynamic data set was satisfactory in all patients. Moreover, interobserver variability for all Doppler variables was low.

The technique used to estimate CO was validated many years ago and is widely and routinely used in most echocardiographic laboratories. Similarly, the accuracy of the modified Bernoulli equation of the continuous-wave Doppler tricuspid regurgitation signal in predicting the gradient between peak right ventricular systolic pressure and RAP is well established. PASP was then calculated as the sum of estimated RAP and the gradient between peak right ventricular systolic pressure and the estimated RAP.

A number of attempts have been made to develop a noninvasive mode for estimating RAP, and echocardiography always has been considered the most reliable tool. Morphological parameters such as respiratory motion of the inferior vena cava, its respiratory diameters and percent collapse (caval index), left hepatic vein diameter, and right atrial dimension were studied initially. Later on, functional data such as left hepatic or tricuspid flow variables were considered. However, some of these indexes offer only semi-quantitative measures of RAP and have failed to demonstrate prognostic value. Others, although highly sensitive and specific, are useful only in select patient groups because of technical and clinical limitations pertaining to the mode of measurement. Moreover, most previous studies suffered from a number of limitations, such as nonsimultaneous echocardiographic and hemodynamic recordings or lack of homogeneity of study populations due to the inclusion of both patients with and patients without congestive HF or with extremely different pathological conditions (mechanical ventilation and primary hypertension). In this study, RAP was estimated from the acceleration rate of early tricuspid filling on the pulsed-wave Doppler signal, easy to measure, and more accurate than measurement of the height of the jugular venous pulse or the size and respirophasic reactivity of the inferior vena cava. Furthermore, the excellent linear correlation between Doppler-derived acceleration rate of early tricuspid filling and invasive RAP yielded an accurate quantitative estimation of PASP ($r=0.97$). The close relation we found between the acceleration rate of early tricuspid flow and RAP has a pathophysiologic explanation. In brief, it represents the acceleration of blood mass produced by all the forces inside the right atrium acting on the tricuspid valve surface during rapid right ventricular filling, which could explain why it is closely related to mean RAP.

The technique adopted to estimate PCWP as a surrogate of mean left atrial pressure relies on the well-known strong negative correlation between the DT of early mitral filling and invasive PCWP. It may be argued that a single

### Table 4. Interobserver Variability

<table>
<thead>
<tr>
<th>Measure</th>
<th>First Observation, Mean ± SD</th>
<th>Second Observation, Mean ± SD</th>
<th>Mean Error, %</th>
</tr>
</thead>
<tbody>
<tr>
<td>RAP, mm Hg</td>
<td>4.7 ± 4.1</td>
<td>4.5 ± 3.8</td>
<td>3.0</td>
</tr>
<tr>
<td>PASP, mm Hg</td>
<td>45.4 ± 16.3</td>
<td>44.1 ± 18.1</td>
<td>4.1</td>
</tr>
<tr>
<td>PCWP, mm Hg</td>
<td>17.2 ± 9.3</td>
<td>17.4 ± 8.5</td>
<td>2.4</td>
</tr>
<tr>
<td>PVR, Wood U</td>
<td>2.5 ± 1.4</td>
<td>2.3 ± 1.7</td>
<td>3.6</td>
</tr>
<tr>
<td>CO, L/min</td>
<td>4.1 ± 0.9</td>
<td>4.2 ± 0.9</td>
<td>2.4</td>
</tr>
</tbody>
</table>
variable is unlikely to reflect diastolic filling pressures that result from a complex interplay of active and passive properties of both left atrium and ventricle. Indeed, recording of Doppler mitral inflow alone may not be adequate to provide a reliable assessment of filling pressures under certain clinical conditions, for example, in patients with diastolic HF. In these patients, more recent echocardiographic techniques such as tissue Doppler, color M-mode, and myocardial strain along with left atrial volume index measurement certainly are useful. In particular, the E-wave to peak mitral annulus velocity (E/Ea) ratio by tissue Doppler and the E-wave velocity to flow propagation velocity ratio by color M-mode can estimate PCWP with reasonable accuracy over a wide range of clinical conditions and ejection fractions at rest, although the reliability of the E/Ea ratio in predicting PCWP in decompensated patients with advanced systolic HF has been challenged recently. However, it has been clearly demonstrated that in patients with systolic HF and severe left ventricular dysfunction, mitral inflow DT alone correlates extremely well with PCWP in patients with sinus rhythm or with atrial fibrillation, and even in patients with significant mitral regurgitation, this negative correlation remains very close and is why we chose this parameter to predict PCWP. Early mitral DT reflects the rate of equalization of left atrial and left ventricular pressures. As filling pressure increases, the nondistensible ventricle may induce a rapid increase in diastolic pressure with a faster equilibration of left atrial and left ventricular pressure, result-

Figure 4. Correlation of changes (Pre-Post) in PVR, PCWP and (Post-Pre) CO after unloading by catheterization (invasive) and echocardiography (ECHO). CO indicates cardiac output; PVR, pulmonary vascular resistance; PCWP, pulmonary capillary wedge pressure.
ing in premature cessation of mitral flow and short DT of early filling.

A recent study has shown a less robust correlation between early mitral DT and PCWP.29 The authors described relatively low correlation as an unexpected finding probably attributable to the confounding effects of left ventricular relaxation and stiffness, left atrial pressure, mitral valve function, and anular recoil in their advanced and mainly unstable HF population (ie, in contrast to our predominantly stable study population). Notwithstanding this study, recent metaanalyses have confirmed the independent predictive value of mitral DT in patients with chronic systolic HF or after myocardial infarction.31,32

The method used to estimate PVR is the only one that is rather elaborate,12 but it reflects a complex parameter that is laborious to calculate even invasively (PAMP−PCWP)/CO. In any case, the function for predicting PVR that we recently elaborated and have effectively validated in this study is not more laborious than that of other modes of calculation suggested by other authors, such as the transpulmonary gradient/CO ratio where the transpulmonary gradient was calculated as a difference between PAMP and left atrial pressure.11 Importantly, as the correlation between invasive and noninvasive PCWP and PVR remained excellent after unloading manipulations (ie, nitroprusside infusion) routinely performed in patients with elevated values at baseline, the noninvasive methodology can be proposed for both the optimal clinical management and the determination of hemodynamic suitability for heart transplantation. Indeed, all 5 patients unsuitable for heart transplantation on the basis of irreversible elevated PVR by right heart catheterization could have been correctly identified by Doppler echocardiography.

Study Limitations

Invasive CO was obtained by the thermodilution method. Although the Fick equation would be recommended for assessing CO in patients with HF, particularly when severe tricuspid regurgitation is present, thermodilution is in fact what is performed clinically. In our study population, no patient had trivial regurgitation. Patients with atrial fibrillation who account for up to 25% of those with advanced HF were excluded, although atrial fibrillation should not in itself pose any absolute limitation provided that several cardiac cycles are averaged. Indeed, most of the noninvasive techniques adopted in this study were validated even in the presence of atrial fibrillation.10,12,14,21 Patients with mechanical mitral prosthesis or permanent electroneinduced rhythm also were excluded (n=2 in our study population).

More sophisticated echocardiographic measurements could have been included, particularly to estimate PCWP; however, it has been clearly demonstrated that mitral DT alone, which is very easy to obtain, correlates extremely well with PCWP and is an independent predictor of outcome in systolic HF.8,13,28,30–32 Furthermore, the reliability of E/Ea ratio in predicting PCWP in decompensated patients with advanced systolic HF has been recently challenged.29

We do acknowledge that the remarkable level of correlation between echocardiographic and hemodynamic variables was much better than anticipated, and to some extent, this may be ascribed to the fact that the cardiologists of our hemodynamic and echocardiography laboratories have been involved in this kind of study for many years. Thus, it may be speculated that for widespread use of this method, such a high accuracy would not be achieved. Nevertheless, the role of Doppler echocardiography in the noninvasive assessment of central hemodynamics in stable patients with advanced HF ultimately should not be affected. Finally, further investigations are required to confirm the results of this small study and to support the potential use of these echocardiography measurements to obtain all necessary hemodynamic information.

Conclusions

Doppler echocardiography may offer today a valid alternative to invasive cardiac catheterization for the comprehensive hemodynamic assessment of predominantly stable patients with advanced systolic HF and in potential heart transplant recipients. Importantly, the reliable noninvasive measurement of PVR in stable patients awaiting transplantation in whom the serial assessment of PVR is most crucial may obviate the need for routine cardiac catheterization.

Disclosures

None.

References

Assessment of central hemodynamics is an essential component of orthotopic heart transplantation recipient evaluation, and threshold values for selected hemodynamic variables have been identified that predict unfavorable outcomes after transplantation. Swan-Ganz catheterization remains the gold standard for the assessment of central hemodynamics, but it is invasive, not without risks, and not practical for widespread application. In this study, we demonstrated that Doppler echocardiography may offer today a valid alternative to invasive cardiac catheterization for the comprehensive hemodynamic assessment of predominantly stable patients with advanced systolic heart failure and of potential heart transplant recipients. Importantly, the reliable noninvasive measure of pulmonary vascular resistance in stable patients awaiting transplantation in whom the serial assessment of pulmonary vascular resistance is most crucial may obviate the need for routine cardiac catheterization.
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