It is essential for the management of heart failure (HF) to accurately assess hemodynamics. The gold standard of hemodynamic evaluation is right heart catheterization (RHC), but clinicians hesitate to repeat RHC in patients because of the procedure’s invasiveness, and hence the small but definite risk of the method. Therefore, a noninvasive method is required in clinical practice.

Article p2408

In the past decade, there was considerable development in the treatment of pulmonary arterial hypertension (PAH), and same therapy was also attempted for patients with HF. Recently, several randomized studies have been conducted to investigate the efficacy of PAH therapy in HF patients. However, the results were not positive in most of the trials with endothelin-1 antagonist and for HF with preserved left ventricular ejection fraction.1–3 Phosphodiesterase type 5 (PDE5) inhibitors showed improvement in hemodynamic variables and exercise tolerance; however, there is no evidence for long term outcome, and multicenter clinical trials with PDE5 inhibitors are currently ongoing.4 The negative results might be at least partially explained by the selection of patients. Only a few studies paid attention to hemodynamic status and there are no established parameters of pulmonary vascular remodeling. Patient selection is important to identify who may benefit from PAH therapy because HF patients are already receiving multi-drug therapy. Therefore, it is important to evaluate pulmonary vascular disease (PVD).

First of all, the definition of left heart disease-related pulmonary hypertension (LHD-PH) has not been standardized, but clinical use of that nomenclature may make the patient population clearer. There are 3 different hemodynamic presentations in LHD-PH proposed by Vachiery et al.5 (1) elevated

<table>
<thead>
<tr>
<th>Table. Non-Invasive Assessment of PVR</th>
<th>Patients</th>
<th>Formulae</th>
<th>Invasive PVR</th>
</tr>
</thead>
<tbody>
<tr>
<td>Echocardiography/Scapellato 200112</td>
<td>n=63</td>
<td>Severe left heart dysfunction EF=17±5% (WHO group II)</td>
<td>PEP/ActT/TT 2.5±1.5 WU</td>
</tr>
<tr>
<td>Echocardiography/Abbas 20037</td>
<td>n=44</td>
<td>Various etiology EF=54 (20–75)%</td>
<td>TRV/TVILVOT 2 (0.7–6) WU</td>
</tr>
<tr>
<td>Echocardiography/Haddad 20098</td>
<td>n=51</td>
<td>PAH (WHO group I)</td>
<td>SPAP/(HR×TVILVOT) 11.0±5.1 WU</td>
</tr>
<tr>
<td>Echocardiography/Kouzu 200911</td>
<td>n=43</td>
<td>PH (WHO group II were excluded)</td>
<td>TRPG/TVILVOT 1,294±680 dyne s−1 cm−5</td>
</tr>
<tr>
<td>Echocardiography/Kasai 201510</td>
<td>n=40</td>
<td>Chronic thromboembolic PH (WHO group IV)</td>
<td>TRPG/COLVOT 5.4±2.9 WU</td>
</tr>
<tr>
<td>Echocardiography/Kanda 201510</td>
<td>n=27</td>
<td>Heart failure (WHO group II)</td>
<td>(TRPG-PRPGedi)/3 COLVOT 2.4±1.4 WU</td>
</tr>
<tr>
<td>MRI/Bane 201514</td>
<td>n=7</td>
<td>Suspected PH (4 PAH, 3 PVH)</td>
<td>mPAPMRI-PCWPMRI/COMRI 6.1 (2.3–8.4)</td>
</tr>
<tr>
<td>Body impedance cardiography</td>
<td>n=65</td>
<td>Known or suspected PH</td>
<td>mPAPecho-10/COECHO 581±344 dyne s−1 cm−5</td>
</tr>
</tbody>
</table>

CO, cardiac output; EDV, end-diastolic volume; EF, ejection fraction; mPAP, mean pulmonary artery pressure; PAH, pulmonary arterial hypertension; PCWP, pulmonary capillary wedge pressure; PEP, pre-ejection period; PH, pulmonary hypertension; PRPGedi, pulmonary regurgitation pressure gradient at end-diastole; PVH, pulmonary venous hypertension; PVR, pulmonary vascular resistance; SPAP, systolic pulmonary artery pressure; TRPG, tricuspid regurgitation pressure gradient; TRV, tricuspid regurgitation velocity; TT, total systolic time; TVI, time velocity integral; WU, wood unit.

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pulmonary capillary wedge pressure (PCWP), but without significant PVD; (2) elevated PCWP, with PVD; (3) previously elevated but normalized PCWP, with persistent PVD. In order to assess the existence of PVD, pulmonary vascular resistance (PVR), trans-pulmonary pressure gradient, and diastolic pressure difference are used as hemodynamic parameters. Among these parameters, PVR is the most frequently analyzed when evaluating clinical stage, and there are several formulae for noninvasively evaluating PVR (Table).

Echocardiography is the main modality used to assess PVD in the clinical practice of HF, and most of the studies were certainly done using echocardiography. The formulae provided are based on empirical or experimental methods in these studies; in contrast, Kanda et al propose new theoretical formulae in this issue of the Journal. They report that the echocardiographic theoretical PVR significantly correlates with invasive PVR and their proposed method was more accurate than previously reported methods. They used 3 echocardiographic parameters of tricuspid regurgitation pressure gradient (TRPG), pulmonary regurgitation pressure gradient at end-diastole (PRPGed), and cardiac output (CO) measured from the left ventricular outflow tract. Measurement of these parameters is already well established and widely used in clinical practice. Therefore, their method may be easily applied for clinical use.

The accuracy of echocardiographic estimation for PVR is limited to patients without high PVR because Kanda et al only studied patients with mildly elevated PVR, and those with possible PVD were not included in this study; therefore, a further study including patients with markedly elevated PVR or possible PVD is required. Invasive rather than noninvasive estimation of PVR is recommended when the value is important in the guidelines. However, once we obtain the hemodynamic data invasively, then echocardiography will become the main modality for re-evaluating the hemodynamic status of patients.

References