Doppler echocardiography in advanced systolic heart failure: a noninvasive alternative to Swan-Ganz catheter

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ABSTRACT

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. To assess the accuracy and clinical applicability of Doppler echocardiography in determining the entire hemodynamic profile in stable patients with advanced systolic heart failure (HF).

Methods and Results: Simultaneous Doppler echocardiography and Swan-Ganz catheterization were performed in 43 consecutive patients with advanced HF. Echocardiographic data required for estimation of right atrial, pulmonary artery systolic and pulmonary capillary wedge pressures (PCWP), and cardiac output and pulmonary vascular resistance (PVR) 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 baseline elevated PVR (>3 Wood units) and PCWP (>20 mm Hg), hemodynamic and Doppler measurements were simultaneously repeated after unloading manipulations. Absolute values and changes of PVR and PCWP after unloading were still accurately predicted (r = 0.96 for PVR and r = 0.92 for PCWP, respectively).

Conclusions: Doppler echocardiography may offer a valid alternative to invasive cardiac catheterization for the comprehensive hemodynamic assessment of patients with advanced HF, and may assist with monitoring and optimization of therapy in potential heart transplant recipients.

Keywords: 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 two methods systematically applied to individual patients must be demonstrated. Finally, along with reliability, also easy applicability of the noninvasive method needs to be demonstrated if it is to be recommended for widespread use at the patient’s bedside.

Thus, the aim of the present 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 sinus rhythm patients with advanced HF (New York Heart Association functional class III or IV) secondary to both ischemic or idiopathic-dilated cardiomyopathy, and severe systolic dysfunction, as defined by echocardiographic ejection fraction (EF) ≤ 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 with a view to selection for heart transplantation, 4 patients were excluded because of mechanical mitral prosthesis (1), permanent electro-induced rhythm (1), or chronic atrial fibrillation (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 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. EF was derived from the standard equation. Both mitral and tricuspid regurgitation were detected and graded 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 off-line analysis. For each Doppler-based measurement, estimates were obtained from three cardiac cycles and averaged. The Doppler echocardiographic data were analyzed as follows:

**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: CO = (Heart rate) (LVOT VTI) (π) (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 cups was assessed using the HP Sonos 5500 packaging. 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 on the basis of the equation: PCWP = 32.16 + (-0.1045E) + (0.1345A) + (-0.17 DT) + (4.95E/A).
DT was the time between 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 LV systolic dysfunction and CHF both in sinus rhythm and atrial fibrillation (6, 14).

**Mean right atrial pressure.** Mean right atrial pressure (RAP) was estimated from the acceleration rate (Ac, cm/s²) of early tricuspid filling on the pulsed wave Doppler signal, on the basis of the equation: RAP = -1.263 + 0.01116*Ac, as previously reported (10). Acceleration rate was measured as the slope of the segment between start and 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 latter obtained by application of the modified Bernoulli equation to the peak velocity of the continuous wave Doppler tricuspid regurgitation signal (VTr max): PASP = RAP + 4VTr max² (9,21,22).

**Pulmonary vascular resistance.** Pulmonary vascular resistance (PVR) was calculated according to the formula (12): PVR= [(PEP/AcT)/TT] where PEP, AcT and TT represent pre-ejection period, acceleration time (AcT) and total systolic time, respectively, measured on the pulsed wave Doppler pulmonary systolic flow (Figure 1).

Doppler echocardiographic studies, both at baseline and after loading manipulations, were performed by 4 different operators (PLT, FS, EE, AI) who had no knowledge of the clinical and hemodynamic data. All the Doppler echocardiographic measurements were subsequently done by a single operator (P.L.T). In 30 consecutive patients measurements were repeated by a second independent observer (F.S.). In 10 further patients interobserver variability for measurement of PCWP, PWR and CO was also assessed immediately after unloading manipulations.

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

**Statistical analysis.** Echocardiographic and hemodynamic data are expressed as mean value ± SD of subjects' 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 method of Bland and Altman (23) was used to assess methodologic agreement in individual patients. Interobserver variability was evaluated by calculating the mean ± SD of the differences between the measurements of two echocardiographic observers (P.L.T., 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 males, 7 females) with a mean age of 54 ± 8 years. Clinical and 2D-echocardiographic data are presented in Table 1. The aetiology of HF was predominantly coronary artery disease (67%). The mean left ventricular EF was 19 ± 5%. Moderate to severe mitral regurgitation was detected in most of the patients (80%, severe regurgitation = 35%), while moderate to severe tricuspid regurgitation was present in about 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 heart failure with severely enlarged left ventricle the quality of the images is usually very good and this explain why, in the present study, no patient was excluded because of poor echo data quality.
Relation of noninvasive to invasive variables. Table 2 shows the invasive hemodynamic variables, whereas the correlation between the two methods is presented in Table 3. For all variables, invasive and non-invasive hemodynamic values were strongly correlated (p<0.001), without significant differences for any single variable (Figure 2). On Bland-Altman analysis, the mean relative difference between all measured and estimated variables was very low for the whole population, indicating the absence of any 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).

Unloading manipulations. Since fixed elevated PVR is a contraindication to heart transplantation, and irreversible elevated PCWP is a strong predictor of poor prognosis, 16 patients with baseline PVR >3 Wood units 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 ± 1.6 to 2.3 ± 1.1 mm Hg, and PCWP decreased from 25 ± 4 to 13 ± 3 mm Hg. Graphs showing the correlation of the changes obtained by means of the two methods (echo vs. catheterization) after loading manipulations in PVR, PCWP and CO, respectively, are reported in Figure 4. Linear regression analysis after unloading manipulations confirmed the strong correlation between invasive and noninvasive measures (r = 0.96 for PVR and r = 0.92 for PCWP, respectively) 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.

DISCUSSION

Besides basic clinical evaluation, comprehensive hemodynamic assessment plays a key role in the management of HF patients, particularly those with advanced heart failure and potential candidates for heart transplantation. In the present 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 (4-16) 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 tested in this study precisely the methods routinely performed at the patient’s bedside of our Heart Failure Unit. Some of these techniques, i.e. that used to estimate PCWP, RAP and PVR, were validated years ago in our Echo laboratory, and are now part of our echocardiographic report for systolic HF patients. 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 (4,20). 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 (9,21,22). 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 has always been considered the most reliable tool. Morphologic parameters such as respiratory motion of the inferior vena cava, its respiratory diameters and percent collapse...
(caval index), (23) left hepatic vein diameter or right atrial dimension were initially studied. Later on, functional data such as left hepatic or tricuspid flow variables were considered (24,25). Some of these indexes, however, offer only semiquantitative measures of RAP, and have failed to demonstrate any prognostic value. Others, although highly sensitive and specific, are useful only in selected 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 non-simultaneous echocardiographic and hemodynamic recordings, or lack of homogeneity of study populations due to the inclusion of both normal and CHF patients or patients with extremely different pathologic conditions (mechanical ventilation, primary hypertension). In the present study, RAP was estimated from the acceleration rate of early tricuspid filling on the pulsed wave Doppler signal (10), 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. What is more, 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. Very briefly, 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: this 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 (6,13,14,17,26). It may be argued that a single variable is unlikely to reflect diastolic filling pressures, which 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, e.g. in patients with diastolic heart failure. In these patients, more recent echocardiographic techniques such as Tissue Doppler, color M-mode and myocardial strain along with left atrial volume index measurement are certainly useful. In particular, the E/Ea ratio by Tissue Doppler and the E/Vp ratio by color M-mode can estimate PCWP with reasonable accuracy over a wide range of clinical conditions and rest...
EFs (15,16,27,28), although the reliability of E/Ea ratio in predicting PCWP in decompensated patients with advanced systolic HF has been recently challenged (29). 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 whether in sinus rhythm (6,13,28) or with atrial fibrillation (14), and even in patients with significant mitral regurgitation this negative correlation remains very close (30). This 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, resulting 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 relatively low correlation was described by the authors as an unexpected finding, probably attributable to the confounding effects of left ventricular relaxation and stiffness, left atrial pressure, mitral valve function, and annular recoil in their advanced and mainly unstable HF population (i.e. in contrast to our predominantly stable study population). Notwithstanding this study, recent meta-analyses have confirmed the independent predictive value of mitral DT in patients with chronic systolic HF or after MI (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 minus PCWP divided by CO). In any case, the function for predicting PVR that we recently elaborated and have effectively validated in the present study is not more laborious than other modes of calculation suggested by other authors such as the transpulmonary gradient/CO ratio, where the transpulmonary gradient was calculated as PAMP minus left atrial pressure (11). Importantly, as the correlation between invasive and noninvasive PCWP and PVR remained excellent after unloading manipulations (i.e. nitroprusside infusion), routinely performed in patients with baseline elevated values, the noninvasive methodology can be proposed for both the optimal clinical management and the determination of hemodynamic suitability for heart transplantation. Indeed, all the 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 recommendable when assessing CO in patients with HF, in particular when severe tricuspid regurgitation is present, thermodilution is what in fact is performed clinically. The 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 the present study were validated even in the presence of atrial fibrillation (10,12,14, 21). Patients with mechanical mitral prosthesis or permanent electro-induced rhythm were also excluded (n = 2 in our study population). More sophisticated echo measurements could have been included, particularly to estimate PCWP; however, it has been clearly demonstrated that mitral DT alone, very easy to obtain, correlates extremely well with PCWP and is an independent predictor of outcome in systolic HF (6,13,28,30-32). Furthermore, the reliability of E/Ea ratio in predicting PCWP in decompensated patients with advanced systolic HF has been recently challenged.

We do acknowledge that the remarkable level of correlation between echocardiographic and hemodynamic variables we found was much better than anticipated. This may to some extent be ascribed to the fact that the cardiologists of our hemodynamic and echo-labs have been involved in this kind of study for many years. Thus, it may be speculated that, for a 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 advanced HF patients should not be ultimately affected.

Finally, further investigations are required to confirm the results of this small study and to support the potential of using 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 measure 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


**Figure legends**

**Figure 1.** Continuous-wave Doppler recording of tricuspid regurgitant flow *(top)* and pulsed-wave recording of pulmonary flow *(bottom)* in the same patient with invasive PVR = 3.3 Wood U.

AcT = acceleration time; EjT = ejection time; PEP = pre-ejection period; PVR = pulmonary vascular resistance.

**Figure 2.** Plots showing correlation between invasive and noninvasive (ECHO) determinations of hemodynamic variables. Lines represent the linear fitting of the regression between invasive and noninvasive determinations.

CO = cardiac output; RAP = right atrial pressure; PAPS = pulmonary artery systolic pressure; PVR = pulmonary vascular resistance; PVRI = pulmonary vascular resistance index; PCWP = pulmonary capillary wedge pressure. ECHO = echocardiography.

**Figure 3.** Bland-Altman plots of selected hemodynamic variables method.

CO = cardiac output; RAP = right atrial pressure; PAPS = pulmonary artery systolic pressure; PVR = pulmonary vascular resistance; PVRI = pulmonary vascular resistance index; PCWP = pulmonary capillary wedge pressure.

**Figure 4.** Correlation of changes (Pre-Post) in PVR, PCWP and (Post-Pre) CO after unloading by catheterization (invasive) and echocardiography (ECHO). 

CO = cardiac output; PVR = pulmonary vascular resistance; PCWP = pulmonary capillary wedge pressure.
Table 1. Clinical and Two-dimensional Echocardiographic Parameters.

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Mean ± SD</th>
<th>Range</th>
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<tbody>
<tr>
<td>Age (years)</td>
<td>54 ± 8</td>
<td>37-65</td>
</tr>
<tr>
<td>Gender (M/F)</td>
<td>36/7</td>
<td></td>
</tr>
<tr>
<td>Coronary artery disease (%)</td>
<td>29 (67%)</td>
<td></td>
</tr>
<tr>
<td>Heart rate (beats/min)</td>
<td>74 ± 14</td>
<td>55-110</td>
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<tr>
<td>LVEDVI (ml/m²)</td>
<td>145 ± 41</td>
<td>85-252</td>
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<tr>
<td>LVEF (%)</td>
<td>19 ± 5</td>
<td>10-34</td>
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<td>Left atrial diameter (mm)</td>
<td>54 ± 6</td>
<td>40-66</td>
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<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>22/15/6</td>
<td></td>
</tr>
</tbody>
</table>

Data are mean values ± SD or number of patients (%). LVEDVI, 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>
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<tr>
<td>CO (liters/min)</td>
<td>3.7 ± 1.0</td>
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<tr>
<td>CI (liters/min/m²)</td>
<td>2.1 ± 0.6</td>
<td>1.4-3.1</td>
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<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>
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<tr>
<td>PVR (Wood U)</td>
<td>2.5 ± 1.7</td>
<td>0.5-7.2</td>
</tr>
<tr>
<td>PVRI (Wood U x m²)</td>
<td>4.65 ± 2.2</td>
<td>0.92-12.1</td>
</tr>
</tbody>
</table>

Data are mean values ± SD or number of patients. CI, cardiac index; CO, cardiac output; PASP, pulmonary artery systolic pressure; PCWP, pulmonary capillary wedge pressure; PVR, pulmonary vascular resistance; RAP, right atrial pressure.
### Table 3. Agreement Between Invasive and Echocardiographic Hemodynamic Measures

<table>
<thead>
<tr>
<th></th>
<th>Difference between methods</th>
<th>Limits of Agreement</th>
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<tbody>
<tr>
<td></td>
<td>r</td>
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<tr>
<td>RAP (mm Hg)</td>
<td>0.96</td>
<td>-0.09</td>
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<tr>
<td>PAPS (mm Hg)</td>
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<td>-0.95</td>
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<tr>
<td>PCWP (mm Hg)</td>
<td>0.93</td>
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</tr>
<tr>
<td>PVR (Wood U)</td>
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<tr>
<td>PVRI (Wood Ux m²)</td>
<td>0.95</td>
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<tr>
<td>CO (liters/min)</td>
<td>0.94</td>
<td>-0.06</td>
</tr>
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</table>

Abbreviations as in Table 2. See also Figure # 3.
Table 4. Interobserver variability

<table>
<thead>
<tr>
<th></th>
<th>Mean ± SD 1st Ob</th>
<th>Mean ± SD 2nd Ob</th>
<th>Mean error %</th>
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</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>PAPS (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 (liters/min)</td>
<td>4.1 ± 0.9</td>
<td>4.2 ± 0.9</td>
<td>2.4</td>
</tr>
</tbody>
</table>

Ob = observation; other abbreviations as in Table 2.
PVR = \frac{PAPM-PCWP}{CO} = 3.3 \text{ Wood U}

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

TT = PEP + EjT
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