Echocardiographic Evaluation of Hemodynamics in Patients with Decompensated Systolic Heart Failure

Nagueh et al: Noninvasive Hemodynamics in ADHF

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Journal Subject Codes: (110): Congestive, (31): Echocardiography
Abstract

**Background**—Doppler echocardiography is currently applied for the assessment of left ventricular (LV) and right ventricular (RV) hemodynamics in patients with cardiovascular disease. However, there are conflicting reports about its accuracy in patients with unstable decompensated heart failure. The objective of this study was to evaluate the accuracy of the technique in patients with unstable heart failure.

**Methods and Results**—Consecutive patients with decompensated heart failure had simultaneous assessment of LV and RV hemodynamics invasively and by Doppler echocardiography. In 79 patients, the non-invasive measurements of stroke volume ($r=0.83, p<0.001$), pulmonary artery systolic ($r=0.83, p<0.001$) and diastolic pressure ($r=0.51, p=0.009$), and mean right atrial pressure ($r=0.85, p<0.001$) all had significant correlations with invasively acquired Doppler indices had good accuracy in identifying patients with pulmonary capillary wedge pressure $>15$ mmHg (AUC from 0.86 to 0.92). The recent ASE/EAE guidelines were highly accurate (sensitivity : 98%, specificity : 91%) in identifying patients with increased wedge pressure. In 12 repeat studies, Doppler echocardiography readily detected the changes in mean wedge pressure ($r=0.75, p=0.005$) as well as changes in pulmonary artery systolic pressure and mean right atrial pressure.

**Conclusions**—Doppler echocardiography provides reliable assessment of LV hemodynamics in patients with decompensated heart failure.

**Key Words:** diastole; doppler; echocardiography; heart failure
Patients with acute decompensated heart failure (ADHF) suffer from increased morbidity and mortality (1). Furthermore, these patients are prone to readmission, with rates ≥ 50% within the first 6 months after discharge (1, 2). The hemodynamic assessment of ADHF offers the potential of tailored therapy. However, the invasive gold standard is not without risks. Accordingly the non-invasive assessment by Doppler echocardiography can play an important role in this population. A recent study has reported that Doppler measurements, including tissue Doppler, do not provide an accurate assessment of LV filling pressures in ADHF (3). However, there were limitations to that study (4), as wedge (PCWP) pressure was measured in a supine position, but imaging was acquired in a left lateral decubitus position which weakens the relationship between Doppler and hemodynamic variables because pulmonary venous return varies with position and patients were rolled onto their left side after pressure measurement. In addition, the measured pressure is altered by a similar magnitude to the change in the level of the catheter in the vascular system, which shifts with body position. Furthermore, PCWP in pulmonary hypertension requires confirmation by oxygen saturation which was not done, and many patients in the latter study had pulmonary hypertension. Finally, some of the Doppler examples presented showed poor alignment of the Doppler beam for the lateral tissue Doppler velocities along with a broad and poorly defined spectral envelop. Aside from these issues, the clinical utility of a comprehensive echocardiographic approach for the assessment of hemodynamics in this population is unknown. Given the importance of the question, we conducted this prospective two-center study to examine the application of Doppler echocardiography for the hemodynamic assessment of patients with ADHF.
Methods

Patient Population

ADHF was defined as new-onset decompensated heart failure or decompensation of chronic heart failure with severe symptoms that warranted hospitalization. Seventy nine consecutive patients (not included in previous studies, 66 at Houston and 13 at Oslo) with a clinical indication for right heart catheterization were prospectively enrolled and underwent simultaneous imaging and cardiac catheterization. Right heart catheterization was performed for clinical reasons and not for the sole purpose of the study. Some patients were evaluated while on their oral regimen upon admission before the initiation of additional heart failure treatment. Other patients were enrolled after initiation of intravenous vasodilators, diuretics and/or pressors. This enabled us to include patients with a wide range of hemodynamic measurements. Mitral stenosis was an exclusion criterion but none of the patients had this condition. Patients with aortic valve disease and those with mitral regurgitation were not excluded. No patients were excluded due to suboptimal images as satisfactory Doppler recordings were obtained in all patients.

Echocardiographic Imaging

The patients were imaged with an ultrasound system equipped with a multi-frequency transducer. A complete echocardiographic study was performed per standard views and guidelines (5). From the apical window, PW Doppler was used to record mitral inflow for 3-5 cardiac cycles at the level of the mitral valve annulus, and at the tips level (6). Pulmonary venous flow was recorded from the right pulmonary vein, guided by color Doppler (6). Tissue Doppler (TD) was applied to record mitral annular velocities at the septal and lateral sides of the annulus (6). The resulting annular velocities by PW Doppler were recorded for 3-5 cardiac cycles at a sweep speed of 100 mm/s. Using color Doppler, the M-mode cursor was positioned within the
mitral inflow stream, and the early diastolic flow propagation velocity (Vp) was recorded (6). Baseline shift was performed as needed to obtain a distinct color border of the propagation velocity that extended well into the distal third of the LV cavity. Tricuspid and pulmonary regurgitation signals were recorded by CW Doppler from multiple windows (7). Saline contrast was used as needed and increased the feasibility of acquiring TR jets to 80%. Inferior vena caval diameter (IVC) and its collapse and hepatic venous flow were recorded in the subcostal view (7).

Echocardiographic Analysis

Measurements were performed on a computerized off-line analysis stations (Digisonics EC, and EchoPAC, GE Healthcare) without knowledge of invasively derived hemodynamic data. Mitral inflow from tips level was analyzed for peak early (E), and late (A) diastolic velocities, E/A ratio, and deceleration time (DT) of mitral E velocity. Mitral A duration was measured at the level of the mitral annulus (6). Pulmonary venous flow was analyzed for the peak velocity of the systolic (S), diastolic (D) and atrial reversal (Ar) signals. The systolic filling fraction and the difference in duration between pulmonary vein Ar velocity and mitral A velocity were derived (6). Mitral annulus early (e’) and late (a’) diastolic velocities were measured at septal and lateral mitral annulus, and E/e’ ratios were computed (6). Interobserver error (mean ± SD) for mitral E velocity was 4±3% and for annulus e’ velocity was 5±2%. Intraobserver error (mean ± SD) for mitral E velocity was 3±2% and for annulus e’ velocity was 3±2%. Measurements were averaged over 3 cardiac cycles.

The algorithm of the ASE/EAE guidelines for the estimation of LV filling pressures in patients with depressed EF was applied (6). Briefly, when the E/A ratio is <1 and with an E velocity ≤50 cm/s, a normal left atrial pressure is assumed. When the E/A ratio is ≥2, or with DT <150 ms, an increased left atrial pressure is assumed. When the E/A ratio is ≥1 but <2, or when it
is <1 but with an E velocity > 50 cm/s, the presence of abnormally elevated values in 2 of the following Doppler measurements was needed to support the conclusion that LV filling pressures are elevated: average E/e’ ratio >15, E/Vp ratio ≥2.5, pulmonary veins systolic to diastolic ratio <1, or Ar-A duration ≥ 30 ms, and PA systolic pressure >35 mmHg.

LV stroke volume was derived as the cross sectional area of the LV outflow tract x time velocity integral of LV outflow tract flow by pulse wave Doppler (8). This method was used to avoid overestimation of systemic output that occurs when 2D derived stroke volume (difference between LV end diastolic and end systolic volumes) is computed in patients with significant mitral regurgitation. Cardiac output was calculated as the product of LV stroke volume and heart rate. Pulmonary artery (PA) systolic pressure was derived using the modified Bernoulli equation as PA systolic pressure in mmHg = 4 (v)^2 of peak tricuspid regurgitation velocity in m/s + RAP in mmHg, and PA diastolic pressure was calculated as: 4 (v)^2 of end diastolic pulmonary regurgitation velocity in m/s + RAP in mmHg (7). Right atrial pressure (RAP) was estimated using the IVC diameter and its change with respiration, and hepatic venous flow (7). Valvular regurgitation was assessed using ASE guidelines (9).

**Cardiac Catheterization**

Mean right atrial pressure, pulmonary artery systolic and diastolic pressures, and pulmonary capillary wedge pressure (PCWP) were measured with a pulmonary artery catheter during right heart catheterization in 73 patients. The wedge position was verified by fluoroscopy, changes in the waveform, and when needed, with O2 saturation (O2 saturation >95%). Invasive measurements were acquired without knowledge of echocardiographic data. All were derived as an average of 5 cycles. Fluid-filled transducers were balanced before the study with the zero level at the mid-axillary line. Cardiac output (average of 3 cycles with <10% variation) was derived by
thermodilution in the majority of patients, though in some it was calculated by Fick method also. Since most patients with ADHF have pulmonary hypertension, the transpulmonary gradient was derived and was computed as mean PA pressure – mean PCWP. In 6 patients left heart catheterization only was performed and LV diastolic pressures were obtained. None of them had more than mild mitral regurgitation. In these 6 patients, LV pre-A pressure was determined and used in lieu of wedge pressure based on the very close correlation between the 2 pressures (6).

Repeat Studies

Hemodynamic and echocardiographic measurements were repeated as described above in patients who had a clinical indication for an indwelling PA catheter for ongoing hemodynamic assessment. In addition, patients who had a repeat hospitalization for ADHF and who had a clinical indication for another cardiac catheterization were included.

Statistical Analysis

Continuous data are presented as mean ± SD. Linear and polynomial regression analysis was performed to determine the correlation between non-invasive and invasive measurements of LV and RV hemodynamics showing correlation coefficient and standard error of estimate (SEE). Receiver operating characteristic curves were used to determine the threshold Doppler values that separated patients with PCWP >15 mm Hg from those with PCWP ≤15 mm Hg. Changes in LV volumes and hemodynamics on repeat studies were evaluated by paired t-tests as the data had a normal distribution. The differences between noninvasive and invasive measurements of hemodynamics were evaluated by Bland-Altman plots. \( P \leq 0.05 \) was considered significant.
**Results**

The mean age of the study sample was 57±11 (range: 24-80) years and 15 patients were females (19%). In 37 patients ADHF was due to dilated cardiomyopathy and in 42 patients due to coronary artery disease. Table 1 presents a summary of their invasive hemodynamics. There were 53 patients with pulmonary hypertension, diagnosed by invasively measured mean PA pressure > 25 mmHg, and 35 patients with a transpulmonary gradient >10 mmHg. The group had severe LV dilatation, and a mean EF of 23±9%. Mitral regurgitation of moderate severity was present in 11 patients, and was moderately severe in 3. The remaining patients had trace to mild lesions. Tricuspid regurgitation of moderate severity was present in 13 patients, and was moderately severe in 6. The remaining patients had trace to mild lesions. All patients were on diuretics in various combinations, 66 patients were receiving in addition oral/ intravenous inotropic/vasodilator drugs, and 3 had an intra-aortic balloon pump. Forty three percent had received biventricular pacing (not single site pacing).

**Feasibility of Doppler measurements**

Mitral inflow velocities were feasible in all patients. A single early diastolic mitral inflow velocity was present in 18 patients (due to atrial fibrillation in 12 patients and sinus tachycardia in 6 patients). TD velocities also had a high feasibility for septal (96%), and lateral (92%) mitral annulus velocities. Vp was feasible in 77%, but satisfactory pulmonary vein signals were obtained in only 32 patients (41%). Adequate tricuspid regurgitation signals were acquired in 63 patients (80%), and RAP could be estimated in 60 patients (76%). On the other hand, the pulmonary regurgitation jet had the lowest feasibility at 32% (adequate quality signals obtained in only 25 patients). Figures 1 to 3 show examples from 3 patients.

**Estimation of LV Stroke Volume**
A good correlation was present between stroke volume obtained by cardiac catheterization and that obtained by echocardiography in the whole group (r=0.83, p<0.001, SEE=11 ml). Likewise, the correlation was good in patients with > mild tricuspid regurgitation and/or > mild mitral regurgitation (r=0.89, p<0.01, SEE=9 ml).

**Estimation of PA Systolic and Diastolic Pressures**

Significant correlations were present between invasively obtained and Doppler derived PA systolic pressure (r=0.83, p<0.001, Figure 4). The mean difference was 3±8.6 mmHg (range: -27 to 22 mmHg). There were 49 patients with PA systolic pressure >35 mmHg; of these 46 were correctly identified by Doppler (sensitivity: 94%). The 3 cases that were not correctly identified had invasive measurements of 38, 36, and 39 mmHg, with Doppler estimates of 34, 33, and 31 mmHg, respectively. Specificity was 90%.

A significant correlation was present between PA diastolic pressure by cardiac catheterization and Doppler (r = 0.51, p=0.009, Figure 5). The mean difference was 2.6±6.7 mmHg (range: -9 to 16 mmHg). All 24 patients with PA diastolic pressure > 12 mmHg by right heart catheterization were correctly identified by Doppler. There was one patient with an invasive measurement of 10 mmHg and a Doppler estimation of 13 mmHg.

**Estimation of mean PCWP**

**A-Accuracy of Individual Doppler Measurements**

Several Doppler measurements related significantly with mean PCWP with good accuracy (Tables 2 and 3). However, LA maximum volume index >34 ml/m² had lower accuracy, due to a low specificity of 33%. The relation between mean PCWP and average E/e’ ratio was better in patients without left bundle branch block/biventricular pacing than in patients with bundle branch block or biventricular pacing (Figure 6).
Mitral inflow alone was sufficient for predicting mean PCWP (whether increased or not) in 65 patients, based on the ASE/EAE cutoff values for E/A ratio and DT (6) and summarized in the methods section above. In 14 patients with an E/A ratio >1 but <2, additional Doppler measurements were needed to predict mean PCWP. In 13 cases, all additional variables (E/e’ ratio, E/Vp ratio, and PA systolic pressure by Doppler) were available and concordant, whereas in one patient only E/e’ and E/Vp ratios could be obtained without an adequate tricuspid regurgitation signal by CW Doppler.

The ASE/EAE algorithm had the highest accuracy with a sensitivity of 98% and a specificity of 91%. There were only 3/79 patients where mean PCWP was incorrectly predicted by the algorithm. An invasively measured mean wedge pressure of 10 mmHg was incorrectly predicted by mitral inflow, E/e’ and E/Vp ratios, and there was no adequate tricuspid regurgitation signal by CW Doppler. The second patient had a mean PCWP of 8 mmHg, but an E/A ratio of 2.3 and a DT of 94 ms. This patient had an average E/e’ ratio of 15.96, E/Vp ratio of 2.38, and a PA systolic pressure by Doppler of 28 mmHg. The third patient missed by the algorithm had a mean PCWP of 20 mmHg, but an E/A ratio of 1.4, E/e’ ratio of 9.4, E/Vp of 1.4, and the PA systolic pressure by Doppler was 36 mmHg.

Estimation of Mean RAP

A strong correlation was present between the invasive and non-invasive estimates of mean RAP (r=0.85, p<0.001, Figure 7 and Table 4). The mean difference was 1±3.6 mmHg (range: -5 to 10 mmHg). There were 15 patients with RAP < 8 mmHg. RAP could be estimated non-invasively in 12 of these 15 patients (there was no adequate subcostal view in 3/15), and correctly identified
11/12 as having RAP < 8 mmHg (specificity of 92%). The exception was a patient who had a RAP of 7 mmHg, but the non-invasive estimate was 12 mmHg.

All patients with RAP ≥ 8 mmHg were correctly identified by echocardiography except for 2 patients (sensitivity of 96%). The exceptions were 2 patients with RAP of 8 and 10 mmHg, but the non-invasive estimate was 5 mmHg in both cases.

Detection of changes in hemodynamics on repeat studies

Eleven patients underwent a total of 12 repeat studies (11 patients with a single repeat study and 1 patient with 2 repeat studies). In 8 instances, the repeat measurements were obtained during the same hospitalization, but after different treatments. This included intravenous Furosemide in 2 patients, Sodium Nitroprusside in 2 patients, intravenous Milrinone in 1 patient, and intravenous pressors (Dopamine, Dobutamine, and Vasopressin) in another patient. Repeat assessment was performed after aggressive hemodialysis in 1 patient, and after DC cardioversion in another patient. In the remaining 3 patients, repeat studies were acquired during a repeat hospitalization for ADHF.

This subgroup of patients had a mean age of 56±9 years (4 females). Six patients had already received biventricular pacing prior to both studies. There were 4 patients in this group who had post infarction cardiomyopathy, and the rest had heart failure due to dilated cardiomyopathy. Three patients had moderate mitral regurgitation, while the rest had mild lesions. Two patients had severe tricuspid regurgitation, two had mild to moderate tricuspid regurgitation, while the rest had mild or trace lesions. There was no change in severity of mitral, or the severity of tricuspid regurgitation between the 2 studies. A summary of LV volumes and hemodynamics for this group with repeat studies is shown in Table 5. Of note, most patients had large increments or large decrements in their mean wedge pressure on repeat studies.
Significant correlations were observed between the changes in echo Doppler measurements and the actual hemodynamic changes measured by right heart catheterization. This included changes in PA systolic pressure ($r = 0.9$, $p = 0.005$), mean wedge pressure ($r = 0.75$, $p = 0.005$, Figure 8), mean right atrial pressure ($r = 0.87$, $p = 0.01$), and LV stroke volume ($r = 0.63$, $p = 0.05$). The results were unchanged after excluding one data set from the patient who had 2 repeat studies.

**Discussion**

This study shows that Doppler echocardiography can be readily applied to assess LV hemodynamics in patients with ADHF, including pulmonary artery pressures, mean wedge pressure and mean right atrial pressure. The study also validates the approach recommended in the recent ASE/EAE guidelines for the assessment of LV filling pressures in patients with depressed EF (6).

**Assessment of LV hemodynamics in ADHF**

ADHF patients are usually managed based on clinical assessment, though PA catheters are not infrequently used to guide management in this population. However, the invasive approach is not without risks and did not translate to a better clinical outcome (10). A noninvasive approach is appealing provided its measurements are accurate. In that regard, a comprehensive assessment of LV hemodynamics is feasible by Doppler echocardiography. The techniques were validated over the past 20-30 years by several laboratories. Furthermore, the measurements of LV stroke volume, PA pressures, and LV filling pressures are currently an integral component of echocardiographic reports in many laboratories. However, while the previous validation studies included patients with depressed EF, the critical question about the accuracy of Doppler
measurements in ADHF was not directly addressed, except for the recently published report by Mullens et al (3).

**Doppler Estimation of Mean PCWP**

The results of our study in ADHF are consistent with several previous reports showing the accuracy of mitral inflow velocities and time intervals in predicting LV filling pressures in patients with depressed EF and stable heart failure (11-21). However, we specifically included patients who were acutely decompensated and some were receiving a number of intravenous drips, including diuretics, vasodilators, positive inotropic drugs and intra-aortic balloon pump. Similar to previous studies, pulmonary vein velocities and the systolic filling fraction exhibited significant correlations with mean PCWP, albeit with a lower feasibility than mitral and TD velocities. We and others have noted the lower feasibility of adequate pulmonary vein velocity signals in patients imaged in the cardiac catheterization laboratory or in the ICU settings (17, 18).

Both E/e’ ratio and E/Vp ratio had good accuracy in predicting mean PCWP in our study. However, the correlation of E/e’ ratio with mean PCWP was lower in patients with left bundle branch block or biventricular pacing. This is probably due to the abnormal septal motion and longitudinal rotation in LBBB which can affect annular diastolic velocities (22). Of note, the weaker relation in patients with left bundle branch block was previously reported in patients with normal (23) and depressed EF (3). The current study and that by Mullens et al (3) emphasize the limitations of TD in patients with biventricular pacing, the need for satisfactory signals and the impact of technically challenging recordings on the accuracy and the clinical application of the methodology.
Interestingly, a Doppler derived PA systolic pressure >35 mmHg was a good predictor of an increased mean PCWP by cardiac catheterization in patients with ADHF. In that regard, our study using invasively measured mean PCWP extends the findings of a previous report, which showed a significant correlation between PA systolic pressure and the noninvasive estimation of mean PCWP in patients with normal LV EF (24), to patients with ADHF.

Application of ASE/EAE guidelines for the estimation of LV filling pressures in ADHF

While most of the individual Doppler parameters had good accuracy, the comprehensive approach recommended in the recent ASE/EAE guidelines had the highest accuracy (6), and only 3 patients were incorrectly classified when these guidelines were applied. Accordingly, we recommend the guidelines approach in lieu of isolated Doppler measurements in patients with ADHF. Of note, a different algorithm is applied in patients with normal EF.

The weak correlation between LA maximal volume and mean PCWP supports the ASE/EAE recommendation of not relying on LA volume for estimation of filling pressures in patients with systolic dysfunction (6). Most ADHF patients have LA enlargement due to long standing diastolic dysfunction and mitral regurgitation. However, while an increased LA volume is not necessarily helpful, patients with a normal LA volume often have a mean PCWP <15 mmHg.

Detection of Changes in mean PCWP

A clinically useful noninvasive measurement of mean PCWP should be able to detect meaningful changes in PCWP with therapy. Previous studies have shown that Doppler can detect changes in filling pressures in heart failure patients who are clinically stable (21). The current study shows that Doppler echocardiography can also be applied for that objective in patients with ADHF, including patients with an increase or a decrease in mean PCWP > 5 mmHg.
Limitations

There are a number of limitations to this study. Some are related to the lower feasibility of acquiring satisfactory pulmonary regurgitation and pulmonary venous flow signals. Fortunately, the feasibility of their acquisition can be increased with intravenous contrast agents which may be needed for LV cavity opacification. Left atrial pressure was not measured directly, as it was difficult to justify transseptal puncture for the sole purpose of the study. However, mean wedge pressure and LV pre-A pressures are good surrogates of LA pressure. We included few patients with repeat hemodynamic measurements and additional observations in this population are needed to confirm our findings.

Conclusions

We demonstrate that Doppler echocardiography can be used to reliably assess LV hemodynamics in patients with acute decompensated heart failure (25). This includes the demonstration that estimation of LV filling pressure is feasible by combining measurements of blood flow velocities and myocardial velocities.
Disclosures

None.
References


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16. Nishimura RA, Appleton CP, Redfield MM, Ilstrup DM, Holmes DR, Jr., Tajik AJ. Noninvasive Doppler echocardiographic evaluation of left ventricular filling pressures in


23. D'Souza KA, Mooney DJ, Russell AE, MacIsaac AI, Aylward PE, Prior DL. Abnormal septal motion affects early diastolic velocities at the septal and lateral mitral annulus, and


Table 1. Summary of Invasive Hemodynamics and LA/LV Volumes and EF

<p>| | |</p>
<table>
<thead>
<tr>
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</thead>
<tbody>
<tr>
<td>1-Heart rate (/min)</td>
<td>80±15</td>
</tr>
<tr>
<td>2-Systolic Blood Pressure (mmHg)</td>
<td>117±18</td>
</tr>
<tr>
<td>3-Diastolic Blood Pressure (mmHg)</td>
<td>75±15</td>
</tr>
<tr>
<td>4-Pulmonary artery systolic pressure (mmHg)</td>
<td>50±17</td>
</tr>
<tr>
<td>5- Pulmonary artery diastolic pressure (mmHg)</td>
<td>23±9</td>
</tr>
<tr>
<td>6- Pulmonary artery mean pressure (mmHg)</td>
<td>32±11</td>
</tr>
<tr>
<td>7-Pulmonary Wedge pressure (mmHg)</td>
<td>21±9</td>
</tr>
<tr>
<td>8-Transpulmonary gradient (mmHg)</td>
<td>11±6</td>
</tr>
<tr>
<td>9-Mean right atrial pressure (mmHg)</td>
<td>14±7</td>
</tr>
<tr>
<td>10-Cardiac Index (l/min/m²)</td>
<td>2.1±0.6</td>
</tr>
<tr>
<td>11-LV end diastolic volume (ml)</td>
<td>255±78</td>
</tr>
<tr>
<td>12-LV end systolic volume (ml)</td>
<td>200±73</td>
</tr>
<tr>
<td>13-LV EF (%)</td>
<td>23±9</td>
</tr>
<tr>
<td>14-Left atrium maximum volume (ml/m²)</td>
<td>48±15</td>
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</table>
### Table 2. Correlation of Doppler Measurements with Mean PCWP

<table>
<thead>
<tr>
<th>Number of patients with Satisfactory Doppler signal</th>
<th>r</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>1-Mitral E velocity</td>
<td>79</td>
<td>0.6</td>
</tr>
<tr>
<td>2-Mitral A velocity</td>
<td>61</td>
<td>-0.47</td>
</tr>
<tr>
<td>3-Mitral E/A ratio</td>
<td>61</td>
<td>0.64</td>
</tr>
<tr>
<td>4-Deceleration time of mitral E</td>
<td>79</td>
<td>-0.46</td>
</tr>
<tr>
<td>5-Isovolumetric relaxation time</td>
<td>79</td>
<td>-0.56</td>
</tr>
<tr>
<td>6-PV systolic filling fraction</td>
<td>32</td>
<td>-0.61</td>
</tr>
<tr>
<td>7-E/Vp</td>
<td>61</td>
<td>0.53</td>
</tr>
<tr>
<td>8-Average E/e’</td>
<td>79</td>
<td>0.61</td>
</tr>
<tr>
<td>9-PA systolic pressure by Doppler</td>
<td>63</td>
<td>0.57</td>
</tr>
<tr>
<td>10-PA diastolic pressure by Doppler</td>
<td>25</td>
<td>0.41</td>
</tr>
<tr>
<td>11-LA maximum volume index</td>
<td>79</td>
<td>0.25</td>
</tr>
</tbody>
</table>

E: peak mitral early diastolic velocity, A: peak mitral late diastolic velocity, PV: pulmonary veins, Vp: flow propagation velocity, e’: mitral annulus early diastolic velocity, PA for pulmonary artery. Mitral A velocity was measured in only 61 patients as 18 patients had a single mitral inflow velocity. In the patients without lateral or septal e’ velocity, the available e’ velocity was used to compute E/e’ ratio and used in the regression analysis.
Table 3. Accuracy of Individual Variables using ASE/EAE Cutoff Values in Identifying Patients with PCWP >15 mmHg (There were 56 patients with mean PCWP>15 mmHg, and 23 patients with mean PCWP≤15 mmHg)

<table>
<thead>
<tr>
<th>Variable</th>
<th>Sensitivity (%)</th>
<th>Specificity (%)</th>
<th>AUC (95% confidence intervals)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1-Mitral E/A ratio (&gt;1.37)</td>
<td>82</td>
<td>82</td>
<td>0.87 (0.79-0.96)</td>
</tr>
<tr>
<td>2-IVRT (&lt;72 ms)</td>
<td>89</td>
<td>81</td>
<td>0.86 (0.73-0.99)</td>
</tr>
<tr>
<td>3-E/Vp (≥2.5)</td>
<td>89</td>
<td>83</td>
<td>0.90 (0.81-0.99)</td>
</tr>
<tr>
<td>4-Average E/e’ (&gt;15)</td>
<td>89</td>
<td>91</td>
<td>0.92 (0.85-1)</td>
</tr>
<tr>
<td>5-PAS pressure by Doppler (&gt;35 mmHg)</td>
<td>85</td>
<td>92</td>
<td>0.89 (0.92-1)</td>
</tr>
<tr>
<td>6-LA volume index (&gt;34 ml/m²)</td>
<td>91</td>
<td>33</td>
<td>0.67 (0.52-0.83)</td>
</tr>
</tbody>
</table>

E for mitral peak early diastolic velocity, A for mitral peak late diastolic velocity, IVRT for isovolumetric relaxation time, Vp for flow propagation velocity, e’ for mitral annulus early diastolic velocity by tissue Doppler, PAS for pulmonary artery systolic, and AUC for area under the curve.
Table 4. Correlation of Pulmonary Artery Pressures, Right Atrial Pressure, and Stroke Volume by Doppler and by Right Heart Catheterization

<table>
<thead>
<tr>
<th></th>
<th>Number of patients with Satisfactory Doppler signal</th>
<th>r</th>
<th>p</th>
</tr>
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<tbody>
<tr>
<td>1-Stroke Volume (ml)</td>
<td>79</td>
<td>0.83</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>2- PA systolic pressure by Doppler (mmHg)</td>
<td>63</td>
<td>0.83</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>3- PA diastolic pressure by Doppler (mmHg)</td>
<td>25</td>
<td>0.51</td>
<td>0.009</td>
</tr>
<tr>
<td>4-Right Atrial Pressure (mmHg)</td>
<td>60</td>
<td>0.85</td>
<td>&lt;0.001</td>
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</table>
Table 5. Changes in LV Volumes and Hemodynamics in Patients with Repeat Invasive Studies

<table>
<thead>
<tr>
<th></th>
<th>Baseline</th>
<th>Repeat Study</th>
</tr>
</thead>
<tbody>
<tr>
<td>1-LV end diastolic volume (ml)</td>
<td>240±66</td>
<td>238±73</td>
</tr>
<tr>
<td>2-LV end systolic volume (ml)</td>
<td>197±53</td>
<td>183±59</td>
</tr>
<tr>
<td>3-LV EF (%)</td>
<td>18±7</td>
<td>23±5*</td>
</tr>
<tr>
<td>4-LA maximum volume index (ml/m²)</td>
<td>51±16</td>
<td>48±11</td>
</tr>
<tr>
<td>5-LV Stroke Volume (ml)</td>
<td>42±13</td>
<td>58±18*</td>
</tr>
<tr>
<td>6-Heart rate (/min)</td>
<td>81±8</td>
<td>79±14</td>
</tr>
<tr>
<td>7-Systolic blood pressure (mmHg)</td>
<td>113±20</td>
<td>100±25</td>
</tr>
<tr>
<td>8-Diastolic blood pressure (mmHg)</td>
<td>72±11</td>
<td>67±17</td>
</tr>
<tr>
<td>9-PA systolic pressure (mmHg)</td>
<td>50±20</td>
<td>42±14</td>
</tr>
<tr>
<td>10-PA diastolic pressure (mmHg)</td>
<td>22±8</td>
<td>18±8</td>
</tr>
<tr>
<td>11-Mean wedge pressure (mmHg)</td>
<td>19±8</td>
<td>18±10</td>
</tr>
<tr>
<td>12-Mean right atrial pressure (mmHg)</td>
<td>13±6</td>
<td>9±6</td>
</tr>
</tbody>
</table>

*: P < 0.05
**Figure Legends**

**Figure 1.** Mitral inflow from a patient admitted with ADHF. Data were acquired upon admission before initiation of intravenous therapy, but while she was still on oral medications. Her mean wedge pressure was 36 mmHg. Mitral inflow (upper left panel) shows restrictive filling, with a peak mitral early diastolic velocity (E) of 113 cm/s, an A (late diastolic) velocity of 35 cm/s, an E/A ratio of 3.2, and a deceleration time of 84 ms. The peak tricuspid regurgitation (TR) velocity (upper right panel) corresponds to a systolic gradient across the tricuspid valve of 38 mmHg. Both septal (lower left) and lateral (lower right) tissue Doppler (TD) early (e’) and late (a’) diastolic velocities are reduced and the average E/e’ ratio is 24.5.

**Figure 2.** Mitral inflow from a patient admitted with ADHF. Data were acquired upon admission before initiation of intravenous therapy, but while he was still on oral medications. His mean wedge pressure was 30 mmHg. Mitral inflow (upper left panel) shows restrictive filling, with a peak mitral early diastolic velocity (E) of 94 cm/s, an A (late diastolic) velocity of 27 cm/s, an E/A ratio of 3, and a deceleration time of 78 ms (vertical scale at steps of 20 cm/s). Pulmonary venous flow (upper right panel) shows reduced systolic (S) velocity and a higher diastolic (D) velocity with an S/D ratio <1. Pulmonary vein atrial velocity (Ar) had a duration that exceeded mitral A duration by 101 ms. Both septal (lower left) and lateral (lower right; vertical scale at 4 cm/s) tissue Doppler (TD) early (e’) and late (a’) diastolic velocities are reduced and the average E/e’ ratio is 18.25.
**Figure 3.** Mitral inflow from a patient admitted with ADHF. Data were obtained after initiation of intravenous therapy with Milrinone and Furosemide. His mean wedge pressure after therapy was 8 mmHg. Mitral inflow (upper left panel, vertical scale of 20 cm/s) shows an impaired relaxation pattern with an E/A ratio <1 and a deceleration time of 232 ms. The peak tricuspid regurgitation (TR) velocity (upper right panel) corresponds to a systolic gradient across the tricuspid valve of 25 mmHg. Septal (lower left), and lateral (lower right) tissue Doppler (TD) early (e’) and late (a’) diastolic velocities are reduced, and the average E/e’ ratio is 8.6.

**Figure 4.** The relation between pulmonary artery (PA) systolic pressure by Doppler versus pressure obtained by right heart catheterization is shown in the left panel (n = 63; y = 1.1x - 0.8). The graph shows the line of equality and the relation of the data points to this line. The Bland-Altman plot is shown in the right panel. The middle line corresponds to the mean difference. The upper line corresponds to the mean difference + 2 SD, whereas the lower line corresponds to the mean difference – 2SD.

**Figure 5.** The relation between pulmonary artery (PA) diastolic pressure by Doppler versus pressure obtained by right heart catheterization is shown in the left panel (n = 25; y = 0.62x + 12.2). The Bland-Altman plot is shown in the right panel. The middle line corresponds to the mean difference. The upper line corresponds to the mean difference + 2 SD, whereas the lower line corresponds to the mean difference – 2SD.

**Figure 6.** The relation between average E/e’ ratio and mean wedge pressure (PCWP) in patients without left bundle branch block (LBBB) and without cardiac resynchronization therapy (CRT)
is shown in the left panel (n=45; y = 2.1x -0.03x² -5.5; R² = 0.56). The right panel shows the relation in patients with LBBB or CRT (n=34; y = 9.4+0.76x).

**Figure 7.** The relation between mean right atrial pressure (RAP) by Doppler echocardiography versus pressure obtained by right heart catheterization is shown in the left panel (n=60; y = 0.96x + 1.69). The Bland-Altman plot is shown in the right panel. The middle line corresponds to the mean difference. The upper line corresponds to the mean difference + 2 SD, whereas the lower line corresponds to the mean difference – 2SD (there are 21 overlapping data points).

**Figure 8.** The plot shows the relation between the change in average E/e’ ratio versus the change in mean wedge pressure in the 12 repeat studies (y = 1.2x – 0.62).
Mitral Inflow

Septal TD Velocities

Lateral TD Velocities

TR by CW Doppler
Mitral Inflow

TR Jet by CW Doppler

Septal TD Velocities

Lateral TD Velocities

e’ a’ e’ a’
PA Diastolic Pressure by Doppler (mmHg)

PA Diastolic Pressure by Right Heart Catheterization (mmHg)

$\frac{\text{PA Diastolic Pressure by Cath} + \text{PA Diastolic Pressure by Doppler}}{2}$ (mmHg)

$\text{PAD by Cath} - \text{PAD by Doppler}$ (mmHg)

$r = 0.51$
$p = 0.009$
No LBBB, NO CRT

r = 0.75
p < 0.001

LBBB, or CRT

r = 0.54
p = 0.001
R.A.P. by Doppler echocardiography (mmHg)

R.A.P. by Rt Heart Catheterization (mmHg)

Average R.A.P. by Cath. + R.A.P. by Echocardiography (mmHg)

\[ r = 0.85 \]

\[ p < 0.001 \]
Average E/e' Ratio

Mean PCWP (mmHg)

r = 0.75
p = 0.005
Echocardiographic Evaluation of Hemodynamics in Patients with Decompensated Systolic Heart Failure

Sherif Nagueh, Rajat Bhatt, Ray P. Vivo, Selim R. Krim, Sebastian Imre Sarvari, Kristoffer Russell, Thor Edvardsen, Otto A. Smiseth and Jerry D. Estep

*Circ Cardiovasc Imaging*. published online March 11, 2011;

*Circulation: Cardiovascular Imaging* is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231

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Print ISSN: 1941-9651. Online ISSN: 1942-0080

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