-Usefulness of the Continuous-Wave Doppler-Derived Pulmonary Arterial-Right Ventricular Pressure Gradient Just Before Atrial Contraction for the Estimation of Pulmonary Arterial Diastolic and Wedge Pressures

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Conflict of interest: None to declare
ABSTRACT

We aimed to clarify the usefulness of our new echocardiographic method using the pulmonary regurgitant velocity just before the right atrial (RA) contraction to estimate pulmonary artery (PA) diastolic pressure (PADP) and mean PA wedge pressure (MPAWP) over the conventional method using that at end-diastole. We studied 55 consecutive patients who underwent echocardiography and right heart catheterization. The pulmonary regurgitant velocities just before RA contraction and at end-diastole were measured to obtain echocardiographic estimates of PADP (EPADP$_{preA}$ and EPADP$_{ED}$, respectively) by adding the pressure gradients to the echocardiographically estimated RA pressure. Compared to EPADP$_{ED}$, EPADP$_{preA}$ better correlated with PADP ($r=0.87$) and MPAWP ($r=0.80$), and direct fixed biases were detected for EPADP$_{ED}$, but not for EPADP$_{preA}$. The AUC to distinguish patients with MPAWP $\geq 18$mmHg was greater for EPADP$_{preA}$ (0.97) than for E/e’ (0.94) and E/A (0.83). EPADP$_{preA}$ is thus useful to estimate PADP and MPAWP in heart disease patients.

**Key Words:** Continuous-wave Doppler echocardiography, Pulmonary regurgitation, Pulmonary arterial diastolic pressure, Pulmonary arterial wedge pressure, Right atrial contraction
INTRODUCTION

Pulmonary arterial (PA) pressure is a fundamental and essential parameter for evaluating patients with various cardiopulmonary diseases (Galiè et al. 2016). In addition, in the absence of pulmonary or pulmonary vascular pathologies, the PA diastolic pressure (PADP) can be used as a surrogate for the mean PA wedge pressure (MPAWP), which plays an important role in the hemodynamic assessment of patients with left-sided heart failure (Falicov et al. 1970; Jenkins et al. 1970; Forsberg et al. 1971). Cardiac catheterization has been the standard technique for the measurement of PADP and MPAWP, but this is an invasive procedure with some risk and discomfort for patients (Boyd et al. 1983; Matthy et al. 1988).

It has been reported that PADP can be noninvasively estimated from the end-diastolic pressure gradient between the PA and the right ventricle (RV) derived from the pulmonary regurgitant (PR) velocity measured by continuous-wave Doppler echocardiography (Masuyama et al. 1986; Lee et al. 1989; Ge et al. 1992). For the estimation of the PADP, the guidelines of the American Society of Echocardiography (ASE) encourage the use of the sum of the end-diastolic PA-RV pressure gradient and the mean RA pressure (MRAP) estimated based on the measurements of the inferior vena cava (IVC) (Rudski et al. 2010). However, the end-diastolic PA-RV pressure gradient may be influenced by an RV pressure rise during atrial contraction, i.e., the A-wave of the RV pressure curve. Thus, the conventional method for estimating the PADP using the end-diastolic PA-RV pressure gradient may underestimate the PADP in patients with a large A-wave and with RV end-diastolic pressure (RVEDP) that is clearly higher than the MRAP. We hypothesized that the use of the PA-RV pressure gradient
just before the RA contraction, which is free from the effect of the RV A-wave, would provide a more accurate estimation of the PADP than the conventional method.

Our objective in this study was to determine the accuracy of our new method using the PA-RV pressure gradient just before the RA contraction in comparison with the standard method using the PA-RV pressure gradient at end-diastole to estimate PADP and also assess the usefulness of our method for evaluating left ventricular (LV) filling pressure in comparison with the conventional parameters such as tissue Doppler E/e’ in patients with heart disease.

PATIENTS AND METHODS

Patients
The study subjects were 55 consecutive patients (33 men and 22 women, 63±16 years old) who were admitted to Hokkaido University Hospital in the 2-year period from January 2013 to December 2014 and underwent an echocardiographic examination and right heart catheterization within a 1-week interval under stable clinical conditions. We excluded patients with an insufficient echocardiographic or catheterization pressure record and those with persistent or very frequent arrhythmia or tachycardia, which prevent correct echocardiographic or catheterization measurements such as atrial fibrillation and frequent ventricular premature beats. The underlying diseases of our study subjects were ischemic heart disease in 27 patients, valvular heart disease in 13, cardiomyopathy in 9, idiopathic arrhythmia in 2, congenital heart disease in 2, hypertensive heart disease in 1, and others in 1. They included 2 patients with the primary lesion in the right heart (1 patient with atrial septal defect and 1 with
arrhythmogenic right ventricular dysplasia), but did not include any with pre-capillary pulmonary hypertension such as pulmonary arterial hypertension or pulmonary thromboembolism.

This study was approved as a retrospective observational study by the Research Ethics Committee of Hokkaido University Hospital and the Ethics Committee of the Faculty of Health Sciences in Hokkaido University. Instead of obtaining informed consent, the program of the present study had been open to the public both through the home page and on the bulletin board of Hokkaido University Hospital.

**Echocardiographic examination**

Echocardiography was performed using an Aplio XG/Artida system equipped with a 2.5/3.0 MHz probe (Toshiba Medical Systems, Otawara, Japan), a Vivid E9 ultrasound system with an M5S probe (GE Healthcare, Little Chalfont, UK), or an iE33 ultrasound system with an S5-1 probe (Philips Medical Systems, Eindhoven, The Netherlands).

The basal RV dimension at end-diastole and the RA minor axis dimension at end-systole were measured in the apical 4-chamber image. The IVC dimension was measured in the subcostal longitudinal image just proximal to the junction of the hepatic veins. The tricuspid annular plane systolic excursion (TAPSE) was measured in the apical 4-chamber image (Kaul et al. 1984).

Using continuous-wave Doppler echocardiography, we measured the peak systolic TR velocity and calculated the peak systolic RV-RA pressure gradient using the simplified Bernoulli equation. We also measured the PR velocities just before RA contraction and at end-diastole to calculate the PA-RV pressure gradients at both timings (Fig. 1). When the dip was very small or not clearly defined, the timing just before RA
contraction was assumed to be 0.11 sec after the onset of electrocardiographic P wave based on our preliminary results (the interval of 0.11±0.02 sec in 22 patients with a very clear late diastolic dip). To minimize the incident angle of the beam to the PR jets, the color flow signal of a jet was visualized in 2 mutually orthogonal planes from several different echocardiographic windows, and the flow velocity was recorded from the window providing the least incident angle and the greatest PR velocity (Kaga et al. 2014).

The IVC dimension and its respiratory change were measured, and we estimated the RA pressure as 3 mmHg when the IVC diameter was ≤19 mm and collapsed >20% with quiet inspiration, and as 15 mmHg when the IVC diameter was >19 mm and collapsed <20% with quiet inspiration. In indeterminate cases in which the IVC diameter and collapse did not fit these criteria, the RA pressure was estimated as 8 mmHg. These criteria were almost in agreement with the ASE guidelines (Rudski et al. 2010), but we modified the cut-off value of the IVC diameter from 21 to 19 mm in light of the different body sizes between Asian and Western populations (Lee et al. 2014).

The sum of the peak systolic RV-RA pressure gradient and the estimated RA pressure (ERAP) was used as an echocardiographic estimate of the PA systolic pressure (PASP) (Rudski et al. 2010). The ERAP was also added to the PA-RV pressure gradient just before the RA contraction and to the end-diastolic PA-RV pressure gradient to obtain echocardiographic estimates of PADP (EPADP_{preA} and EPADP_{ED}, respectively).

**Catheterization examination**

Cardiac catheterization was performed for assessing coronary artery stenosis in 19 patients, for a hemodynamic assessment of heart failure in 14 patients, for the diagnosis
of structural heart disease in 12 patients, for a preoperative evaluation of valvular heart
disease in 5 patients, for a postoperative evaluation in 1 patient, and for other purposes
in 4 patients. Right heart catheterization was performed using a Swan-Ganz catheter.
From the pressure records, we measured the MRAP, RV diastolic pressure just before
RA contraction (RVpreAP), RVEDP, PASP, PADP, mean PA pressure (MPAP) and
MPAWP (Fig. 2). MPAWP was visually measured from the mean pressure curve
obtained using standard hemodynamic software (QP-400G, Nihon Kohden, Tokyo,
Japan).

Statistical analysis
Standard statistical software (SPSS ver. 22 for Windows, SPSS, Chicago, IL) was used
for the statistical analysis. Pearson’s linear correlation and a Bland-Altman analysis
were used to assess the relationship between the catheter and Doppler data (Bland and
Altman 1986). A paired t-test was used to compare the data from the echocardiographic
and catheterization examinations. We performed a receiver operating characteristic
curve (ROC) analysis to assess the diagnostic ability of the echocardiographic
parameters. For all statistical tests, a p-value <0.05 was considered to indicate
significance.

RESULTS

The clinical, echocardiographic and catheterization variables of the patients
Table 1 summarizes the clinical, echocardiographic and catheterization data of the
patients. The EPADP_{preA} was not significantly different from PADP (12.2±6.2 mmHg vs.
12.7±5.8 mmHg, n.s.), but the EPADP$_{ED}$ was significantly smaller than the PADP (10.3±6.1 mmHg vs. 12.7±5.8 mmHg, p<0.001). The EPADP$_{preA}$ was not significantly different from MPAWP (12.2±6.2 mmHg vs. 11.7±6.7 mmHg, n.s.), whereas the EPADP$_{ED}$ was significantly smaller than the MPAWP (10.3±6.1 mmHg vs. 11.7±6.7 mmHg, p<0.05).

**Different late diastolic PR velocity patterns**

Four examples of late diastolic PR velocity patterns were shown in Fig. 3. Among the 55 study patients, 20 (36%) had a late diastolic dip which terminated before end-diastole (Fig. 3a), 16 (29%) had a dip which was directly connected at an acute angle to the abrupt decline of the PR velocity during isovolumic contraction phase (Fig. 3b), 14 (26%) had a dip obtusely connected to the abrupt PR velocity decline (Fig. 3c), and 5 (9%) had a small obscure dip (Fig. 3d).

**The relationship between echocardiographically estimated PADPs and catheter-derived PADP**

The EPADP$_{preA}$ correlated well with the PADP (r=0.87, p<0.001) (Fig. 4a), and there was no fixed bias between the EPADP$_{preA}$ and the PADP in the Bland-Altman analysis (the difference between the means was −0.41 mmHg; the 95% confidence interval, −1.22 to 0.40 mmHg) (Fig. 4c). Although the EPADP$_{ED}$ also correlated well with the PADP (r=0.84, p<0.001) (Fig. 4b), there was a direct fixed bias (the difference between the means was −2.35 mmHg; the 95% confidence interval, −3.23 to −1.46 mmHg) showing an underestimation of the PADP by the EPADP$_{ED}$ (Fig. 4d).
The relationship between echocardiographically estimated PADP and catheter-derived MPAWP

The EPADP_{preA} correlated well with the MPAWP (r=0.80, p<0.001) (Fig. 5a), and there was no fixed bias in the Bland-Altman analysis (the difference between the means was 0.50 mmHg; the 95% confidence interval, −0.59 to 1.59 mmHg) (Fig. 5c). Although the EPADP_{ED} also correlated well with the MPAWP (r=0.74, p<0.001) (Fig. 5b), there was a direct fixed bias (the difference between the means was −1.44 mmHg; the 95% confidence interval, −2.67 to −0.21 mmHg) showing an underestimation of MPAWP by the EPADP_{ED} (Fig. 5d).

The relationship between the late diastolic PR velocity reduction and the late diastolic RV pressure rise

The difference between EPADP_{preA} and EPADP_{ED} was significantly correlated with the difference between RVEDP and RVpreAP (r=0.69, p<0.001), indicating that the decrease in PR velocity from the timing just before RA contraction to end-diastole was caused chiefly by the late diastolic RV pressure rise from RVpreAP to RVEDP (Fig. 6).

The correlation between other echocardiographic parameters of LV filling pressure and catheter-derived MPAWP, and the prediction of elevated MPAWP

There was a significant correlation between E/A and MPAWP (r=0.65, p<0.001) and between E/e’ and MPAWP (r=0.52, p<0.001), but each of these correlations was weaker than that between EPADP_{preA} and MAPWP (r=0.80, p<0.001). In the ROC analysis to distinguish patients with MPAWP ≥ 18 mmHg, the area under the curve was greater for EPADP_{preA} (0.97) than for E/e’ (0.94) and E/A (0.83) (Fig. 7). The sensitivity and
specificity of EPADP_{preA} in predicting MPAWP \geq 18 \text{ mmHg} using the best cutoff value of 14 \text{ mmHg} were 92\% and 91\%, respectively.

**DISCUSSION**

The results of the present study demonstrated that EPADP_{preA}, which is the sum of the PA-RV pressure gradient just before the RA contraction and the ERAP, more accurately reflects PADP and MPAWP than EPADP_{ED}, which is recommended in the ASE guidelines. Moreover, EPADP_{preA} was shown to have sufficient utility in estimating MPAWP and discriminating patients with elevated MPAWP with an accuracy comparable to or rather higher than that of the conventional parameters such as E/e’ and E/A. To our knowledge, there has been no report showing the usefulness of EPADP_{preA} for these purposes. Our method will provide a useful new tool for the estimation of PADP and LV filling pressure in patients with heart disease.

*Conventional methods to estimate PADP by echocardiography*

Masuyama et al. (1986) were the first to report that the PA-RV pressure gradient at end-diastole derived from PR velocity measured using continuous-wave Doppler echocardiography correlated well with invasive PADP, but they noted that this method underestimated the actual value. They also described that the degree of underestimation was especially greater in patients with elevated RVEDP. Lee et al. (1989) also reported that the Doppler estimate (which was the sum of the PA-RV pressure gradient from the end-diastole PR velocity and the central venous pressure directly measured using a central venous catheter) was useful to estimate the PADP. The ASE guidelines
encourage the estimation of the PADP using the sum of the end-diastolic PA-RV pressure gradient and the RA pressure estimated from the IVC measurements (Rudski et al. 2010). However, the guidelines do not comment about the possibility of the error accompanying a large RV A-wave or high RVEDP.

Ge et al. (1992) stated that the mean diastolic pressure gradient across the pulmonary valve measured by Doppler was useful to estimate the PADP. They did not add the ERAP to the mean diastolic PA-RV pressure gradient for the estimation of PADP. It might be possible that the mean diastolic PA-RV pressure gradient without the addition of the ERAP incidentally agreed with the PADP in Ge et al. study since the mean diastolic PA-RV pressure gradient is naturally higher than the PA-RV pressure gradient just before the RA contraction and that at end-diastole. Although the Ge et al. method shares a common idea with ours, our method is not only less affected by a disproportionate RVEDP rise but also simpler and easier to perform than theirs, because our method does not require the complete recording and tracing of a PR jet envelope as the Ge et al. method does. Our new method is thus more feasible for routine echocardiographic examinations.

The reason why the $EPADP_{preA}$ was more accurate than the $EPADP_{ED}$ in estimating PADP and MPAWP

A few investigators have pointed out the influence of the elevated late diastolic RV pressure on the PR velocity waveform. Kisanuki et al. (1987) reported three cases with a late diastolic PA forward flow thought to be caused by decreased RV compliance and highly elevated RVEDP. Moukarbel et al. (2009) described a case with a biphasic PR flow velocity pattern having a deep dip at the timing of the atrial contraction.
These case reports suggested that the PR velocity waveform during late- to end-diastole can be modified by the late diastolic RV pressure rise, especially during atrial systole. To our knowledge, however, there have been no systematic studies on the deformation of PR velocity pattern due to atrial contraction. Our present findings showed that the difference between EPADP_{preA} and EPADP_{ED} is significantly correlated with the difference between RVEDP and RVpreAP, suggesting that the late diastolic PR velocity pattern may be greatly influenced by the late diastolic RV pressure rise associated with RA contraction. Especially when the RV A-wave continues up to the abrupt RV pressure rise during isovolumic contraction and the late diastolic dip of the PR velocity is directly connected to the abrupt decline, the end-diastolic measurement may become inaccurate (Fig. 3). The PA-RV pressure gradient just before the RA contraction may be less influenced by the late diastolic RV pressure rise and completely free from the RV A-wave, thus enabling a more accurate estimation of the PADP and MPAWP.

**Usefulness for the assessment of LV filling pressure**

Several investigators reported direct correlations between the transmitral E/A ratio and LV filling pressure (Giannuzzi et al. 1994; Nishimura et al. 1996). However, the “U-shaped” distribution of the transmitral flow patterns from normal diastolic function to severe diastolic dysfunction often interferes with the correct interpretation (Garcia et al. 1998). In addition, the mitral flow velocity curves are influenced not only by the LA pressure (LAP), but also by the relative compliance of the LA and LV, the rate of ventricular relaxation, pericardial restraint, and atrial systolic function (Appleton et al. 1988; Choong et al. 1988; Nishimura et al. 1996).
Currently, E/e’ has been most widely used for the estimation of LV filling pressure and is listed in the ASE guidelines as one of the important parameters to assess LV filling pressure (Nagueh et al. 1997, 2016). However, Geske et al. (2007) reported that they could not find any correlation between LAP and E/e’ in patients with symptomatic hypertrophic cardiomyopathy. Moreover, Mullens et al. (2009) reported that there was no significant correlation between MPAWP and E/e’ in patients with advanced systolic heart failure. The ASE recommends that E/e’ not be used for patients with significant mitral annular calcification, surgical rings, mitral stenosis, prosthetic mitral valves, moderate to severe primary mitral regurgitation, or constrictive pericarditis (Nagueh et al. 2016). The PADP can be used as a substitute for MPAWP in patients without pulmonary or pulmonary vascular lesions (Falicov et al. 1970; Jenkins et al. 1970; Forsberg et al. 1971). The present study demonstrated that EPADP_{preA} was also useful to estimate MPAWP with a higher accuracy than E/A and E/e’ in patients with left-sided heart failure.

**Limitations**

There are several limitations in this study. First, the sample size was relatively small, from a single hospital, and the subjects were only adult patients. This might have led to some bias and influenced the results. Second, the right heart catheterization and echocardiography were not performed simultaneously; there was a time difference of 2.8 ± 1.9 days (0–7 days). Although we excluded patients with unstable hemodynamics and/or loading conditions between catheterization and echocardiography, the possibility of hemodynamic alternation might not be completely excluded. Third, although we think that the late diastolic dip of the PR velocity largely reflects the A-wave of the RV
pressure curve, we can not exclude the possibility that the late diastolic transtricuspid flow modified the PR jet velocity. Fourth, the PADP does not necessarily reflect MPAWP in patients with abnormal pulmonary vascular beds (i.e., elevated pulmonary vascular resistance) whose PADP is often substantially higher than their MPAWP (Falivov et al. 1970; Rapp et al. 2001).
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FIGURE LEGENDS

Fig. 1. Continuous-wave Doppler measurements of the pulmonary regurgitant velocities just before right atrial contraction (yellow arrow) and at end-diastole (white arrow).

(a) Parasternal long-axis color Doppler image of the right ventricular outflow tract and pulmonary artery to guide the Doppler beam direction. (b) Continuous-wave Doppler recording of pulmonary regurgitant velocity.

Fig. 2. Tracings of the right ventricular (a), pulmonary artery (b) and pulmonary arterial wedge (c) pressures.

The pressure records of the same patient as in the Figure 1 were shown. RVpreAP = right ventricular pressure just before right atrial contraction; RVEDP = right ventricular end-diastolic pressure; PADP = pulmonary arterial diastolic pressure; MPAWP = mean pulmonary arterial wedge pressure.

Fig. 3. Examples of different late diastolic PR velocity patterns.

(a) A distinct late diastolic dip which terminated before right ventricular end-diastole.

(b) A distinct dip which was directly connected at an acute angle to the abrupt decline of the PR velocity during right ventricular isovolumic contraction. (c) A distinct dip obtusely connected to the abrupt PR velocity decline. (d) A small obscure dip. Yellow arrows show pulmonary regurgitant velocity just before right atrial contraction and white arrows show pulmonary regurgitant velocity at end-diastole. PR = pulmonary regurgitation.
Fig. 4. Relationship between the echocardiographically estimated pulmonary arterial diastolic pressures just before right atrial contraction (EPADP_{preA}) and at end-diastole (EPADP_{ED}) and the actual pulmonary arterial diastolic pressure (PADP).

The results of the correlation and regression analysis (a, b) and the Bland-Altman analysis (c, d) are shown.

Fig. 5. Relationship between the echocardiographically estimated pulmonary arterial diastolic pressures just before right atrial contraction (EPADP_{preA}) and at end-diastole (EPADP_{ED}) and the actual mean pulmonary arterial wedge pressure (MPAWP).

The results of the correlation and regression analysis (a, b) and the Bland-Altman analysis (c, d) are shown.

Fig. 6. Correlation between the difference between the echocardiographically estimated pulmonary arterial diastolic pressures just before right atrial contraction and that at end-diastole (EPADP_{preA} – EPADP_{ED}) and the actual difference between the right ventricular end-diastolic pressure and that just before the atrial contraction (RVEDP – RVpreAP).

Fig. 7. Receiver operating characteristic curves for the echocardiographic estimate of pulmonary arterial diastolic pressure just before right atrial contraction (EPADP_{preA}) (solid line), E/e’ (dashed line) and E/A (dotted line) to distinguish
patients with a mean pulmonary arterial wedge pressure $\geq 18 \text{ mmHg}$. AUC = area under the curve; $E/e'$ = ratio of the early-diastolic transmitral flow velocity to the early-diastolic mitral annular velocity; $E/A$ = ratio of the early-diastolic transmitral flow velocity to the atrial-systolic velocity.
Figure 3
Figure 4

(a) $y = 0.95x + 0.29$
$n = 55$
$r = 0.87$
$p < 0.001$

(b) $y = 0.89x - 0.90$
$n = 55$
$r = 0.84$
$p < 0.001$

(c) $\text{EPADP}_{\text{preA}} - \text{PADP}$

(d) $\text{EPADP}_{\text{E}} - \text{PADP}$
Figure 5

(a) $y = 0.74x + 3.50$
$n = 55$
$r = 0.80$
$p < 0.001$

(b) $y = 0.67x + 2.45$
$n = 55$
$r = 0.74$
$p < 0.001$
$y = 0.98x + 0.37$

$n = 55$

$r = 0.69$

$p < 0.001$
Table 1. Clinical, echocardiographic and catheterization variables of the study patients

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<td>Age (years)</td>
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<td>Height (cm)</td>
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<td>Weight (kg)</td>
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<td>SBP (mmHg)</td>
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<td>DBP (mmHg)</td>
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<tr>
<td>EPADP&lt;sub&gt;preA&lt;/sub&gt; (mmHg)</td>
<td>12.2 ± 6.2</td>
<td>5.6–34.0</td>
</tr>
<tr>
<td>EPADP&lt;sub&gt;ED&lt;/sub&gt; (mmHg)</td>
<td>10.3 ± 6.1</td>
<td>3.5–30.5</td>
</tr>
<tr>
<td>ERAP (mmHg)</td>
<td>5.0 ± 3.7</td>
<td>3–15</td>
</tr>
<tr>
<td>Catheterization parameters:</td>
<td></td>
<td></td>
</tr>
<tr>
<td>MPAWP (mmHg)</td>
<td>11.7 ± 6.7</td>
<td>4.0–31.0</td>
</tr>
<tr>
<td>PASP (mmHg)</td>
<td>30.2 ± 14.2</td>
<td>15.0–88.0</td>
</tr>
<tr>
<td>PADP (mmHg)</td>
<td>12.7 ± 5.8</td>
<td>5.0–36.0</td>
</tr>
<tr>
<td>MPAP (mmHg)</td>
<td>19.3 ± 8.6</td>
<td>9.0–52.0</td>
</tr>
<tr>
<td>RVpreAP (mmHg)</td>
<td>6.2 ± 2.9</td>
<td>1.1–13.5</td>
</tr>
<tr>
<td>RVEDP (mmHg)</td>
<td>7.8 ± 3.3</td>
<td>2.9–16.0</td>
</tr>
<tr>
<td>MRAP (mmHg)</td>
<td>5.3 ± 3.2</td>
<td>0.0–16.0</td>
</tr>
</tbody>
</table>

HR, heart rate; BSA, body surface area; SBP, systolic blood pressure; DBP, diastolic blood pressure; LVDd, left ventricular end-diastolic diameter; IVST, interventricular septal thickness; PWT, left ventricular posterior wall thickness; LVEF, left ventricular ejection fraction; LAVI, left atrial volume index; E, peak early diastolic transmitral flow velocity; A, peak late diastolic transmitral flow velocity; E/A, ratio of E to A; e', averaged early-diastolic mitral annular velocities at the septal and lateral sides; E/e', ratio of E to e'; RVDd, right ventricular end-diastolic diameter; RAD, right atrial diameter; IVCD, inferior vena cava diameter; TAPSE, tricuspid annular plane systolic excursion; EPASP, echocardiographic estimate of pulmonary arterial systolic pressure; EPADP<sub>preA</sub>, echocardiographic estimate of pulmonary arterial diastolic pressure just before right atrial contraction; EPADP<sub>ED</sub>, echocardiographic estimate of pulmonary arterial diastolic pressure at end-diastole; ERA P, echocardiographic estimate of right atrial pressure; MPAWP, mean pulmonary arterial wedge pressure; PASP, pulmonary arterial systolic pressure; PADP, pulmonary arterial diastolic pressure; MPAP, mean pulmonary arterial pressure; RVpreAP, right ventricular diastolic pressure just before the atrial contraction; RVEDP, right ventricular end-diastolic pressure; MRAP, mean right atrial pressure. Data are mean ± SD.