Noninvasive assessment of pulmonary arterial capacitance by pulmonary annular motion velocity in children with ventricular septal defect

Yasunobu Hayabuchi*, Akemi Ono, Yukako Homma and Shoji Kagami

Abstract

Background: We hypothesized that longitudinal pulmonary arterial deformation during the cardiac cycle reflects pulmonary arterial capacitance. To examine this hypothesis, we assessed whether tissue Doppler-derived pulmonary annular motion could serve as a novel way to evaluate pulmonary arterial capacitance in pediatric patients with ventricular septal defect (VSD).

Methods: In this prospective study, pulmonary annular velocity was measured in children (age, 6 months–5 years) with a preoperative VSD (VSD group, n = 35) and age-matched healthy children (Control group, n = 23). Pulmonary artery capacitance was calculated by two methods. Systolic pulmonary arterial capacitance (sPAC) was expressed as the stroke volume/pulmonary arterial pulse pressure. Diastolic pulmonary arterial capacitance (dPAC) was determined according to a two-element windkessel model of the pulmonary arterial diastolic pressure profile.

Results: Pulmonary annular velocity waveforms comprised systolic bimodal (s1′ and s2′) and diastolic e′ and a′ waves in all participants. The peak velocities of s1′, s2′, and e′ were significantly lower in the VSD group than in the Control group. On multiple regression analysis, sPAC was an independent variable affecting the peak velocities of the s1′, s2′, and e′ waves (β = 0.41, 0.62, and 0.35, respectively). The dPAC affected the s1′ wave peak velocity (β = 0.34). The time durations of the s1′ and e′ waves were independently determined by the sPAC (β = 0.49 and 0.27).

Conclusion: Pulmonary annular motion velocity evaluated using tissue Doppler is a promising method of assessing pulmonary arterial capacitance in children with VSD.

Keywords: Tissue Doppler imaging, Pulmonary annular motion, Pulmonary arterial compliance, Children

Abbreviations: BSA, Body surface area; dPAC, Diastolic pulmonary arterial capacitance; DPAP, Diastolic pulmonary arterial pressure; HR, Heart rate; ICT, Isovolumic contraction time; IRT, Isovolumic relaxation time; LVEDD, Left ventricular end-diastolic dimension; LVEF, Left ventricular ejection fraction; LVFS, Left ventricular fractional shortening; MPAP, Mean pulmonary arterial pressure; PA, Pulmonary artery; PAH, Pulmonary arterial hypertension; PAPP, Pulmonary arterial pulse pressure; PASV, Pulmonary artery stroke volume; PVRi, Pulmonary vascular resistance indexed for body surface area; Qp/Qs, Pulmonary to systemic blood flow ratio; RV, Right ventricle; RVEDP, Right ventricular end-diastolic pressure; RVEF, Right ventricular ejection fraction; RVOT, Right ventricular outflow tract; RVSP, Right ventricular systolic pressure; sPAC, Systolic pulmonary arterial capacitance; SPAP, Systolic pulmonary arterial pressure; TDI, Tissue Doppler imaging; VSD, Ventricular septal defect

* Correspondence: hayabuchi@tokushima-u.ac.jp
Department of Pediatrics, Tokushima University, Kuramoto-cho-3, Tokushima 770-8505, Japan
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Background
Pulmonary vascular hemodynamic assessment has traditionally evaluated pulmonary vascular resistance (PVR) [1–3]. However, PVR primarily reflects small vessel status and the static component of the pulmonary circulation [4], and it does not reflect the properties of the large and medium pulmonary vessels or account for pulsatile elements of the pulmonary circulation. Pulmonary artery (PA) capacitance reflects the dynamic component of the pulmonary circulation and is thought to be determined largely by the properties of the large proximal capacitance vessels [5]. Because it is the immediate environment encountered by the right ventricle (RV), PA capacitance is a major determinant of RV work [5]. It has recently been shown that lower PA capacitance is strongly associated with worse survival in patients with pulmonary arterial hypertension (PAH) [5–7]. Furthermore, PA capacitance is also reported to be a strong prognostic indicator in patients with left ventricular dysfunction, even in patients with normal PVR [8]. Consequently, accurate determination and serial follow-up of PA capacitance are important in the management of patients with various cardiac diseases with congestive heart failure. However, despite its potential importance for RV performance and clinical outcomes, PA capacitance is not routinely measured. Noninvasive quantitative assessment of PA capacitance remains challenging.

We have previously reported that the pulmonary annular motion velocity waveform obtained using tissue Doppler imaging (TDI) reflects right ventricular outflow tract (RVOT) performance [9]. In the present study, we hypothesized that the pulmonary annular motion would be affected not only by RVOT function, but also by PA capacitance because the pulmonary annulus is located adjacent to the RVOT and PA. In large arteries, deformation of the arterial wall during systole and diastole occurs in the radial and longitudinal directions (Additional file 1). The present study attempted to assess the relationship between the longitudinal pulmonary arterial deformation and capacitance.

The aim of this study, therefore, was to determine whether tissue Doppler-derived pulmonary annular motion velocity can be used as a noninvasive assessment of PA capacitance in children with ventricular septal defect (VSD).

Methods
Study population
The prospective study group comprised consecutive 35 pediatric patients with a preoperative VSD (VSD group; mean age, 2.3 ± 1.5 years; range, 0.6–5.0 years). These patients were scheduled for surgical closure. Twenty-three age-matched healthy children without electrocardiographic or echocardiographic abnormalities (Control group; age, 2.5 ± 1.6 years; range, 0.6–5.0 years) were also enrolled. The patients underwent cardiac catheterization within 3 days of assessment by echocardiography. Data collected between December 2012 and December 2015 were analyzed. All protocols were approved by the Institutional Review Board of the Tokushima University Hospital and conformed to the ethical guidelines of the Declaration of Helsinki (1975). The parents of all subjects provided their written, informed consent for their children to participate in the study.

Echocardiographic study
Standard and pulsed Doppler tissue echocardiography proceeded using a Preirus digital ultrasound system (Hitachi-Aloka Medical Co., Tokyo, Japan) equipped with 1–5 and 3–7 MHz sector transducers. All Doppler data were acquired from patients in the left lateral decubitus position during shallow respiration or end-expiratory apnea. Pulmonary annular motion velocity was measured using TDI in the long-axis view of the RVOT and PA. Guided by the two-dimensional images, a sample volume with a fixed length of 5.0 mm was placed on the pulmonary annulus of the RV free wall side, as indicated by the yellow arrow [9]. Figures 1a and b show a representative example of the color TDI and profile of the pulmonary annular velocity in a healthy child. Furthermore, tricuspid annular motion was recorded in the four-chamber view for the sake of comparison (Fig. 1c). The ultrasound beam was positioned parallel to the direction of the pulmonary and tricuspid annular motions. All tissue Doppler parameters were measured during three consecutive heart cycles by a single physician who was blinded to patient condition, and mean values were calculated.

In addition to pulsed TDI, participants were assessed by conventional, two-dimensional, M-mode, pulsed, continuous, and color Doppler echocardiography. The left ventricular ejection fraction (LVEF) was calculated from apical two-chamber and four-chamber images using the biplane Simpson’s technique. All parameters were measured over three cardiac cycles and then averaged.

Cardiac catheterization
All VSD patients underwent cardiac catheterization within 3 days of echocardiography. Catheterization and angiography using an Integris Allura 9 Biplane (Phillips Medical Systems, Best, The Netherlands) proceeded using 4–6 Fr catheters. All patients were intubated and examined by biplane anteroposterior and lateral projection angiography. Ventricular volume was assessed by means of ventriculography and calculated using Simpson’s rule by quantitative CAW2000 cardiac analysis software (ELK Corporation, Osaka, Japan). During cardiac catheterization, the main PA pressure was measured...
using a high-fidelity manometer-tipped 0.014-inch pressure wire (PressureWire Aeris; St Jude Medical, Inc., St. Paul, MN, USA) to compare with pulmonary annular motion velocity (Fig. 1d). Pulmonary blood flow was calculated using the Fick principle. Stroke volume indexed to body surface area (BSA) was calculated from the pulmonary blood flow in 1 min divided by the heart rate (HR), expressed in mL/m². PVR was calculated using the standard formula; that is, PVR was calculated as the mean PAP minus PA wedge pressure/pulmonary blood flow (PVR; expressed in Wood units·m²).

PA capacitance was calculated by two methods. One was designated systolic PA capacitance (sPAC), which can be determined from measures of pulse pressure and stroke volume indexed to BSA. The sPAC was expressed as the stroke volume/pulse pressure (sPAC; mL/mmHg·m²) [6–8]. Furthermore, PA capacitance was also estimated in a different way. Diastolic PA capacitance (dPAC) was approximated according to a two-element windkessel model that assumes that compliance and hemodynamic resistance are constant during the measurement [10–12]. Previous studies reported that PA capacitance can be calculated from the PA pressure profile during diastole. The time constant (tau) of the exponentially decaying curve can be obtained from the fitting curve calculated from diastolic PA pressure. The dPAC
(mL/mmHg·m\(^2\)) was calculated from PVR and the PA time constant (tau) (Fig. 1e).

### Statistical analysis
All data are expressed as means ± standard deviation (S.D.) or as medians with the 5th–95th percentiles. Statistical significance was determined using the Mann-Whitney U-test or Student’s t-test, as appropriate. Linear regression analyses were performed for correlations between the pulmonary annular motion velocity and hemodynamic parameters, and Pearson’s or Spearman’s correlation coefficients were calculated, as appropriate. Variables with \( p < 0.10 \) on univariate analysis were “candidates” for a backwards stepwise (inclusion criteria/exclusion criteria: \( p < 0.05/p > 0.1 \), respectively) multivariate analysis, and those with \( p < 0.05 \) were “retained” in the model. Multiple regression analysis was used to identify hemodynamic variables affecting the pulmonary annular motion waveform. All statistical data were calculated using Prism version 6.0 (GraphPad Software, San Diego, CA, USA) and JMP 11 (SAS Institute, Inc., Cary, NC, USA) installed on a desktop computer. A value of \( p < 0.05 \) was considered significant. Intra-observer and inter-observer reproducibilities of TDI measurements were assessed using Bland-Altman analysis in a blinded manner. Data were recorded and assessed at 5-minute intervals by observers 1 and 2 from 20 randomly selected participants (VSD, \( n = 10 \); Controls, \( n = 10 \)). For intra-observer variability, data were analyzed twice, 8 weeks apart. Inter-observer variability was assessed by analyzing data from two separate observers blinded to each other’s results.

### Results
No subjects were excluded from the subsequent analyses due to suboptimal recording from poor echocardiographic imaging. Accordingly, the study group included 23 healthy children (2.5 ± 1.6 years; range, 0.6–5.0 years) and 35 VSD patients (mean age, 2.3 ± 1.5 years; range, 0.6–5.0 years). Table 1 shows the clinical, echocardiographic, and hemodynamic data of the participants. Age, height, and HR did not differ significantly between the VSD group and the Control group, whereas body weight and BSA were significantly lower in the VSD group than in the Control group. Left ventricular end-diastolic dimension (LVEDD), left ventricular fractional shortening (LVFS), and LVEF were significantly higher in the VSD group than in the Control group.

Figure 1b shows the pulmonary annular velocity curve. The tricuspid annular motion is shown in Fig. 1c for comparison with the pulmonary annular velocity waveform. The systolic wave was monomodal (‘s’) for the tricuspid annular velocity and bimodal (s1’ and s2’) for the pulmonary annular motion velocity. The systolic waveform peaked earlier in pulmonary annular motion than in the tricuspid annular velocity curve. The shapes of the e’ and a’ waves in diastole were similar. Simultaneous recordings of pulmonary annular motion, RV pressure, and PA pressure in a patient with VSD are shown (Fig. 1d). The pulmonary s1’ wave corresponds to the steep RV pressure elevation during early systole. The s2’ wave coincides with mid to late systolic phase around the peak RV and PA pressure. Measurements of the sPAC and dPAC are shown with the PA pressure profile (Fig. 1e). The comparison between the sPAC and dPAC obtained from the VSD group is demonstrated in Fig. 1f. The sPAC was significantly higher than the dPAC (4.2 ± 1.9 vs. 1.4 ± 0.7 mL/mmHg·m\(^2\), \( p < 0.0001 \)). There was a significant correlation between these parameters (Fig. 1g: \( r = 0.65, p < 0.0001 \)).

Pulmonary annular motion was compared between the Control and VSD groups (Fig. 2). The peak velocities of the s1’, s2’, and e’ waves were significantly lower in the VSD group than in the Control group (9.9 ± 1.9 vs. 11.5 ± 2.2 cm/s, \( p = 0.0070 \); 3.4 ± 0.7 vs. 4.1 ± 1.1 cm/s, \( p = 0.0107 \); 8.8 ± 2.3 vs. 13.0 ± 2.7 cm/s, \( p < 0.0001 \), respectively). The s1’ duration, s2’ duration, e’ duration, and a’ duration were significantly shorter in the VSD group (\( p = 0.0008, <0.0001, 0.0392, and 0.0008 \), respectively), whereas isovolumic contraction time (ICT) and isovolumic relaxation time (IRT) were not significantly different between the two groups.

Next, the relationships between the parameters obtained from TDI-derived pulmonary and tricuspid annular motion and right heart performance were examined in the VSD group. Table 2 summarizes the univariate regression analysis. TDI-derived parameters were significantly correlated with and determined by RV and PA function. Figure 3 shows the relationship between the sPAC and the pulmonary annular motion waveform. The peak velocity of the pulmonary annular s1’ wave was significantly correlated with the sPAC (\( r = 0.58, p = 0.0002 \)). The s2’ and e’ velocity also showed significant correlations with the sPAC (\( r = 0.62, p < 0.0001; r = 0.66, p < 0.0001 \); respectively). ICT and IRT measured using TDI and the time duration of each wave were also assessed in terms of their correlations with the sPAC. The sPAC had significant correlations with s1’ and e’ and a’ wave duration (\( r = 0.49, p = 0.0029; r = 0.48, p = 0.0034; r = 0.44, p = 0.0088 \); respectively), whereas there were no significant correlations with ICT, IRT, and s2’ wave duration. Figure 4 shows the relationship between dPAC and TDI-derived pulmonary annular motion. The peak velocities of pulmonary annular s1’, s2’, and e’ wave were significantly correlated with the dPAC (\( r = 0.60, p = 0.0002; r = 0.46, p = 0.0051; r = 0.55, p = 0.0006 \); respectively). In regard to the timing issue, the e’ wave duration was significantly correlated with the dPAC (\( r = 0.53, p = 0.0011 \)).
Next, multiple regression analysis for predictors of the pulmonary annular motion waveform was performed (Table 3). The analysis showed that the sPAC had an effect on the peak velocities of s1′, s2′, and the e′ wave and the time duration of the s1′ and a′ waves (β = 0.41, p = 0.0131; β = 0.62, p < 0.0001; β = 0.35, p = 0.0314; β = 0.49, p = 0.0029; and β = 0.27, p = 0.0488, respectively). The dPAC affected the s1′ wave peak velocity (β = 0.34, p = 0.0354). The other right heart performance parameters, including RVEDP, RVSP, and Qp/Qs, also had an impact on pulmonary annular motion. Importantly, although RVEF had been assumed to determine pulmonary annular motion, no significant relationship was demonstrated with pulmonary annular motion in the VSD group.

Reproducibility

The inter- and intra-observer reproducibilities of the TDI analysis of pulmonary annular motion were determined from Bland-Altman analysis of 20 randomly
selected participants (VSD group, \(n = 10\); Control group, \(n = 10\)). Table 4 shows the inter- and intra-observer reproducibilities obtained from the Bland-Altman plots (bias ± 2SDs [95% limit of agreement]). They showed minimal bias and substantial agreement for reproducibility.

**Discussion**

The present results showed that the pulmonary annular motion waveform is mainly determined by PA capacitance. Assessment of pulmonary annular motion is useful for estimating PA capacitance in children with VSD. Pulmonary annular TDI was found to be a simple, rapid, reproducible, and highly distinctive method for evaluating PA capacitance.

Recent reports demonstrated that PA capacitance has great impact on the prognosis in various cardiac diseases [5–8]. Patients require lifelong follow-up that includes serial assessment of PA capacitance. Therefore, noninvasive assessment of PA capacitance is considered quite important. However, its assessment is challenging due to the difficulty of measurement and poor reproducibility. Therefore, an invasive method is necessary to accurately...
| Pulmonary annulus motion |
|--------------------------|
| s1′ (cm/sec)             |
| \( r = 0.58 \)           |
| \( p = 0.0002 \)         |
| s2′ (cm/sec)             |
| \( r = 0.62 \)           |
| \( p < 0.0001 \)         |
| e′ (cm/sec)              |
| \( r = 0.66 \)           |
| \( p < 0.0001 \)         |
| a′ (cm/sec)              |
| n.s.                    |
| ICT (msec)               |
| n.s.                    |
| IRT (msec)               |
| n.s.                    |
| s1′ duration (msec)      |
| \( r = 0.49 \)           |
| \( p = 0.0029 \)         |
| s2′ duration (msec)      |
| n.s.                    |
| e′ duration (msec)       |
| \( r = 0.48 \)           |
| \( p = 0.0034 \)         |
| a′ duration             |
| \( r = 0.44 \)           |
| \( p = 0.0086 \)         |

| Tricuspid annulus motion |
|--------------------------|
| s′ (cm/sec)              |
| \( r = 0.36 \)           |
| \( p = 0.0332 \)         |
| e′ (cm/sec)              |
| n.s.                    |
| a′ (cm/sec)              |
| n.s.                    |
| ICT (msec)               |
| n.s.                    |
| IRT (msec)               |
| n.s.                    |
| s′ duration (msec)       |
| n.s.                    |
| e′ duration (msec)       |
| \( r = 0.35 \)           |
| \( p = 0.0232 \)         |

\( sPAC \): systolic pulmonary arterial capacitance; \( dPAC \): diastolic pulmonary arterial capacitance; \( SPAP \): systolic pulmonary arterial pressure; \( DPAP \): diastolic pulmonary arterial pressure; \( MPAP \): mean pulmonary arterial pressure; \( PAPP \): pulmonary arterial pulse pressure; \( RVSP \): right ventricular systolic pressure; \( RVEDP \): right ventricular end-diastolic pressure; \( PVRi \): pulmonary vascular resistance indexed for body surface area; \( PASV \): stroke volume to pulmonary artery; \( Qp/Qs \): pulmonary to systemic blood flow ratio; \( RVEF \): right ventricular ejection fraction.
assess PA capacitance in clinical practice. Several studies have been conducted to identify noninvasive ways for estimating PA capacitance using echocardiographic parameters [13, 14]. These studies attempted echocardiographic estimation of PA capacitance from the PA pulse pressure and RV stroke volume. Systolic PA pressure was estimated from the tricuspid regurgitation velocity by the modified Bernoulli equation. RV stroke volume was calculated using the pulmonary valve diameter measured in the parasternal short-axis view and the velocity-time integral of the PA Doppler flow [13]. In other reports, the right pulmonary arterial diameter change during the cardiac cycle was used to calculate PA capacitance [14]. However, these methods are complex and time-consuming, errors can easily occur, and they have low reproducibility. Previous reports that attempted echocardiographic estimation of PA capacitance in clinical practice. Several studies have been conducted to identify noninvasive ways for estimating PA capacitance using echocardiographic parameters [13, 14]. These studies attempted echocardiographic estimation of PA capacitance from the PA pulse pressure and RV stroke volume. Systolic PA pressure was estimated from the tricuspid regurgitation velocity by the modified Bernoulli equation. RV stroke volume was calculated using
capacitance all had limitations due to poor reproducibility and difficulty, because accurate quantitative assessment by two-dimensional echocardiography is hampered by the complex geometry and difficult depiction. Pulmonary arterial wall strain evaluated by speckle tracking method can be a candidate for evaluating PA distensibility and capacitance. However, in our preliminary experimental study, the tracking was insufficient and the reproducibility was quite low. Therefore, nongeometric methods to assess PA compliance and PA deformation should be explored. One such method, TDI, allows the quantitative assessment of longitudinal PA deformation.

To the best of our knowledge, this is the first application of pulmonary annular motion velocity obtained by TDI as a tool for PA capacitance assessment. We postulated that the PA longitudinal deformation reflects stroke volume, and that the pulmonary annular motion waveform shows PA capacitance.
In the present study, PA capacitance was represented by two parameters: sPAC and dPAC. There was a significant correlation between the two parameters, although sPAC was significantly higher than dPAC. The significant difference between the two parameters was assumed to be caused by the time phase difference during the cardiac cycle. The sPAC is the arterial capacitance from end-diastole to systole, whereas the dPAC is the capacitance from end-systole to diastole.

The peak velocities of the pulmonary annular s′, s′, e′, and a′ waves were significantly lower in the VSD group than in the Control group (Fig. 2), whereas the peak velocity of the tricuspid s′ wave was significantly higher in the VSD group than in the Control group. This discrepancy between pulmonary and tricuspid annular motions might have resulted from the different RV contractility properties between RV inflow and outflow regional properties that originate from the RV geometry, including myocardial fiber orientation [15, 16]. Furthermore, we assumed that PA motion velocity might be affected by PA properties, including PA capacitance. The annular motions are presumed to be affected by RV performance and the adjacent structures [9]. The pulmonary annulus and tricuspid annulus are located adjacent to the PA and right atrium, respectively. Tricuspid annular motion would be affected by right atrial stiffness, contraction, and pressure. Pulmonary annular motion is assumed to be affected by PA properties, including arterial pressure, compliance, and stiffness. These conditions might contribute to the opposite results for the peak velocities of pulmonary and tricuspid annular motions in the VSD group.

The present data demonstrated that pulmonary annular motion parameters can be used to represent PA capacitance. On multiple regression analysis, PA capacitance independently affected the pulmonary annular motion waveform and parameters. Importantly, RVEF was not an independent variable that determined pulmonary annular motion. This is likely due to the fact that PA stroke volume is not correlated with RVEF because VSD shunt flow is directly expelled to the PA during systole in the VSD group. The present data indicate that pulmonary annular motion is determined not by RV contraction, but by PA deformation in children with VSD. Nevertheless, the

### Table 3

| Objective variable | Explanatory variables | B | SE  | β     | p values | VIF |
|--------------------|-----------------------|---|-----|-------|----------|-----|
| s1′ (cm/sec)       | sPAC                  | 0.41 | 0.16 | 0.41   | 0.0131   | 1.76 |
|                    | dPAC                  | 0.95 | 0.43 | 0.34   | 0.0354   | 1.74 |
|                    | RVEDP                | −0.37 | 0.11 | −0.39  | 0.0021   | 1.02 |
| s2′ (cm/sec)       | sPAC                  | 0.24 | 0.05 | 0.62   | <0.0001  | 1.00 |
|                    | RVSP                 | −0.05 | 0.02 | −0.28  | 0.0432   | 1.71 |
| e′ (cm/sec)        | sPAC                  | 0.37 | 0.19 | 0.35   | 0.0314   | 2.08 |
|                    | RVSP                 | −0.05 | 0.02 | −0.28  | 0.0432   | 1.71 |
| a′ (cm/sec)        | n.a.                  | -   | -   | -     | -        | -   |
| ICT (msec)         | n.a.                  | -   | -   | -     | -        | -   |
| IRT (msec)         | n.a.                  | -   | -   | -     | -        | -   |
| s1′ duration (msec)| sPAC                  | 3.76 | 1.17 | 0.49   | 0.0029   | 1.00 |
| s2′ duration (msec)| Qp/Qs                | −12.71 | 4.19 | −0.47  | 0.0047   | 1.00 |
| e′ duration (msec) | n.a.                  | -   | -   | -     | -        | -   |
| a′ duration (msec) | sPAC                  | 2.66 | 1.30 | 0.27   | 0.0488   | 1.11 |
| Qp/Qs              | -0.19                | 3.18 | 4.44 | 0.0070 | 1.48     |

### Table 4

| Parameter variability | Inter-observer variability | Intra-observer |
|-----------------------|----------------------------|----------------|
| Peak velocity of s1′  | 0.15 ± 2.12 cm/s           | −0.14 ± 2.14 cm/s |
| Peak velocity of s2′  | −0.04 ± 1.25 cm/s          | −0.05 ± 1.36 cm/s |
| Peak velocity of e′   | 0.27 ± 1.72 cm/s           | 0.29 ± 1.46 cm/s |
| Peak velocity of e′   | 0.19 ± 1.43 cm/s           | 0.18 ± 2.41 cm/s |
| ICT                   | −5.34 ± 15.14 ms           | −4.31 ± 10.22 ms |
| IRT                   | 2.21 ± 10.03 ms            | −1.31 ± 9.33 ms |
| s1′ duration (msec)   | 3.64 ± 10.31 ms            | 1.92 ± 8.13 ms |
| s2′ duration (msec)   | −0.33 ± 18.75 ms           | −0.29 ± 16.85 ms |
| e′ duration (msec)    | 0.88 ± 13.54 ms            | −0.72 ± 11.14 ms |
| a′ duration (msec)    | 5.22 ± 9.36 ms             | 3.98 ± 6.38 ms |

Inter- and intra-observer variabilities (bias ± 2 SD [95 % limit of agreement]) are shown.
present study demonstrated the significant negative correlation between the pulmonary annular s1 wave and RVEDP in the VSD group. Furthermore, RVSP and RVEDP affect the peak velocity of the e’ wave. It would be useful to investigate the relationship between pulmonary annular motion velocity and RV performance or RV overload. Further studies are necessary to determine the various factors affecting pulmonary annular motion.

Limitations
The sample cohort was relatively small, but TDI parameters were compared between patients and age-matched healthy individuals, and distinctive waveforms were found in the patient group. We postulated that PA expansion by the stroke volume and distensibility is similar in the radial and longitudinal directions. However, a directional difference might be present, which might generate systematic measurement error. The detailed characteristics of stiffness and elasticity in the PA radial and longitudinal directions should be elucidated in the future. Next, some degree of angulation between the Doppler beam and the true direction of longitudinal PA elasticity might exist. The long-axis view of the RVOT and PA was obtained, and the ultrasound beam was parallel to the direction of the pulmonary annular motions. We considered that this is the most appropriate cross-sectional view to observe the expansion and contraction of the PA.

A significant correlation between the s1 wave and RVEDP was found. Furthermore, RVSP and RVEDP had effects on the peak velocity of the e’ wave in the VSD group. These indicate that RV performance also affects pulmonary annular motion. It is necessary to note that PA capacitance is not the only factor determining pulmonary annular motion. From another point of view, it would be meaningful to investigate the relationship between pulmonary annular motion velocity and RV performance or RV overload. Further studies are needed to determine the utility of pulmonary annular motion.

Lastly, although it was demonstrated that TDI-derived pulmonary annular motion parameters reflect PA capacitance, no attempt was made to determine the most useful parameter and derive the prediction formula to evaluate PA capacitance in the present study. The correlations found between pulmonary annular motion parameter and PA capacitance are relatively weak. Further studies of larger patient populations are needed to determine the most valuable parameter and the normal range of pulmonary annular motion for the evaluation of PA capacitance. Moreover, a further study with other disease populations would be necessary to establish the importance of the TDI-derived pulmonary annular motion waveform for the estimation of PA capacitance.

Conclusions
Pulmonary annular TDI is a promising echocardiographic tool for evaluating PA capacitance in children with VSD. It can be a simple, rapid, reproducible, and highly distinctive method for evaluating PA capacitance.

Additional file

Additional file 1: The pulmonary annular motion would be affected not only by RVOT function, but also by PA capacitance because the pulmonary annulus is located adjacent to the RVOT and PA. The deformation of arterial wall during systole and diastole occurs in the radial and longitudinal directions. The longitudinal pulmonary arterial deformation can reflect the vascular capacitance. (WMV 358 kb)

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Availability of data and material
All data generated or analysed during this study are included in this published article.

Authors’ contributions
Yasunobu Hayabuchi was responsible for idea formation, study designing, performing echocardiography and reviewing the article. Akemi Ono contributed in idea formation, study designing, performing echocardiography, drafting and reviewing the article. Yukako Homma and Akemi Ono were responsible for data gathering and helped in analysis and drafting the article. Shoji Kagami was responsible for critical review and editing the manuscript. All authors read and approved the final manuscript.

Competing interests
The authors declare that they have no competing interests.

Consent for publication
Not applicable.

Ethics approval and consent to participate
We also confirm that this study complied with the principles of the declaration of Helsinki. Study protocol was approved by the Institutional Review Board of the Tokushima University Hospital. The parents of all subjects provided their written, informed consent for their children to participate in the study.

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