Pulmonary Annular Motion Velocity Assessed Using Doppler Tissue Imaging
– Novel Echocardiographic Evaluation of Right Ventricular Outflow Tract Function –
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Background: We assessed whether measuring pulmonary annular motion velocity could serve as a novel method of evaluating right ventricular outflow tract (RVOT) performance in pediatric patients with heart disease.

Methods and Results: Tissue Doppler-derived pulmonary annular motion velocity was determined from the parasternal long-axis view of the RVOT. Pulmonary annular velocity was measured in children (age, 5–10 years) with an atrial septal defect (ASD), pulmonary arterial hypertension (PAH), surgically repaired tetralogy of Fallot (TOF) and healthy children (control). Pulmonary annular velocity waveforms comprised systolic bimodal (s1' and s2') and diastolic e' and a' waves in all groups. The peak velocity of s1' and s2' was significantly higher in the ASD group than in the controls (15.0±2.4 vs. 11.2±2.1 and 6.0±0.9 vs. 4.4±1.2 cm/s; P<0.01 and P<0.001, respectively). The s1' and s2' peak velocities were significantly lower in the PAH group (8.5±1.2 and 3.2±0.4 cm/s; P<0.05 for both), and in the group with TOF (5.3±2.2 and 3.4±1.4 cm/s; P<0.001 and P<0.05, respectively). The peak velocity of e' was significantly decreased in the PAH and TOF, compared with the control group (6.8±1.6 and 8.2±2.9 vs. 11.9±1.9 cm/s; P<0.001 for both).

Conclusions: Pulmonary annular motion velocity determined using tissue Doppler imaging is a promising method of assessing RVOT function. (Circ J 2016; 80: 168–176)

Key Words: Children; Pulmonary annular motion; Right ventricular outflow tract; Tissue Doppler imaging

Right ventricular (RV) function has important prognostic and therapeutic implications in patients with various diseases, including congenital heart disease,1 ischemic heart disease,2 pulmonary hypertension3 and cardiomyopathy.4 Consequently, assessment of RV function is increasingly recognized as important in the management of patients with RV dysfunction. However, the quantitative assessment of RV function remains challenging, mainly because of the complex RV geometry and thin myocardial wall.5,6 RV function is generally evaluated using analyses of longitudinal shortening, and several reports describe analyses using tricuspid annular plane systolic excursion (TAPSE), tissue Doppler-derived tricuspid annular velocity and longitudinal strain of the RV free wall in the apical 4-chamber view.7 However, the RV morphology is complex, and some regions are not evaluable by analyses in only 1 direction. Consequently, these methods are not necessarily appropriate for functional evaluation.

The shape of the RV is triangular when viewed from the front. The tricuspid annular velocity corresponds to only 1 of 3 sides of the triangle. We considered that pulmonary annular velocity would indicate another side of the triangle and show RV outflow tract (RVOT) function. Previous investigations have not focused much on RVOT performance, although RVOT motion is important in RV ejection.8,9

Therefore the present study aimed to determine the characteristics of pulmonary annulus velocity waveforms obtained using tissue Doppler imaging (TDI), and whether tissue Doppler-derived pulmonary annular velocity can serve as a tool for RVOT functional assessment in healthy individuals and in children with RV dysfunction.

Methods

Study Population
The study group comprised 16 pediatric patients with preoperative atrial septal defect (ASD; mean age, 7.2±1.5 years; range, 5.0–9.5 years), 6 with pulmonary arterial hypertension (PAH;
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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. Guided by the 2D images, a sample volume with a fixed length of 5.0 mm was placed on the pulmonary annulus of the RV free wall side. Furthermore, the pulmonary annular motion of the septal side was also recorded for the sake of comparison. Tricuspid annular motion of the RV free wall side was recorded in the 4-chamber view. The ultrasound beam was positioned parallel to the direction of the pulmonary and tricuspid annular motions. All tissue Doppler parameters were measured during 3 consecutive heart cycles by a single physician who was blinded to the conditions of the patients, and mean values were calculated.

In addition to pulsed TDI, participants were assessed by conventional, 2D, M-mode, pulsed, continuous and color Doppler echocardiography. Transmitral and transtricuspid diastolic blood flow velocities were determined in the apical 4-chamber view by placing the pulsed Doppler sample volume at the tip of the valve leaflets. All parameters were measured during 3 cardiac cycles and then averaged.

mean age, 7.8±2.1 years; range, 6.1–10.0 years) and 34 with surgically corrected tetralogy of Fallot (TOF; mean age, 7.3±1.6 years; range, 5.0–10.0 years). We selected patients with these conditions because they had RV volume overload, RV pressure overload and surgically repaired RVOT, respectively. We also enrolled 43 age-matched healthy children without electrocardiographic or echocardiographic abnormalities (control; age, 7.3±1.7 years; range, 5.0–10.0 years). The patients underwent cardiac catheterization within 3 days of assessment by echocardiography. Data collected between December 2012 and January 2015 were analyzed. All protocols were approved by the Institutional Review Board of the Medical University of Tokushima, and conformed to the ethical guidelines of the Declaration of Helsinki (1975). The parents of all of the subjects provided 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

Table 1. Clinical Characteristics of Each Group of Pediatric Patients

|                      | Control (n=40) | ASD (n=15) | PAH (n=6) | TOF (n=32) | P value |
|----------------------|---------------|------------|-----------|------------|---------|
| Sex (M/F)            | 20/20         | 6/9        | 4/2       | 18/14      | NS      |
| Age (years)          | 7.3±1.7       | 7.2±1.6    | 7.8±2.1   | 7.3±1.6    | NS      |
| Weight (kg)          | 26.1±8.2      | 27.0±11.0  | 28.3±17.0 | 23.3±9.3   | NS      |
| Height (cm)          | 124.1±12.1    | 122.2±14.3 | 127.9±19.1| 119.4±14.1| NS      |
| BSA (m²)             | 0.94±0.14     | 0.97±0.31  | 0.98±0.21 | 0.91±0.11  | NS      |
| HR (beats/min)       | 76±12         | 74±14      | 81±16     | 69±18      | NS      |
| QRS duration (ms)    | 89±6          | 93±12      | 92±18     | 116±25†    | <0.0001 |
| LVEDD (mm)           | 36.8±3.1      | 33.9±5.1   | 32.2±6.1* | 38.2±2.9   | <0.001  |
| LVFS (%)             | 39.7±5.7      | 36.3±5.0   | 31.7±7.7* | 38.7±5.1   | <0.01   |
| LVEF (%)             | 65.5±6.4      | 63.5±10.2  | 53.3±8.2† | 66.3±13.1  | <0.001  |
| Qp/Qs                | –             | 2.30±0.51  | –         | 1.02±0.05  | –       |
| RVEDV (% of normal)  | –             | 189±38     | –         | 164±17     | –       |
| RVEDP (mmHg)         | –             | 6.9±2.9    | 9.1±5.5   | 8.1±3.1    | –       |
| RVEF (%)             | –             | 63±18      | –         | 51±11      | –       |
| RVSP (mmHg)          | –             | 24.6±8.6   | 67.6±19.6 | 46.6±18.6  | –       |
| mPAP (mmHg)          | –             | 13.6±3.6   | 38.6±14.3 | 16.1±5.6   | –       |

Transmural flow

|                      | Control (n=40) | ASD (n=15) | PAH (n=6) | TOF (n=32) | P value |
|----------------------|---------------|------------|-----------|------------|---------|
| E (m/s)              | 0.96±0.11     | 0.83±0.21* | 0.74±0.19*| 0.93±0.15  | <0.01   |
| A (m/s)              | 0.47±0.08     | 0.46±0.13  | 0.51±0.17 | 0.46±0.11  | NS      |

Transtricuspid flow

|                      | Control (n=40) | ASD (n=15) | PAH (n=6) | TOF (n=32) | P value |
|----------------------|---------------|------------|-----------|------------|---------|
| E (m/s)              | 0.73±0.13     | 0.81±0.18  | 0.53±0.14*| 0.66±0.14  | <0.01   |
| A (m/s)              | 0.36±0.05     | 0.51±0.13* | 0.66±0.12†| 0.36±0.08  | <0.001  |

Tricuspid annulus motion velocity

|                      | Control (n=40) | ASD (n=15) | PAH (n=6) | TOF (n=32) | P value |
|----------------------|---------------|------------|-----------|------------|---------|
| s' (cm/s)            | 13.2±2.4      | 16.8±1.4†  | 12.5±4.1  | 7.0±2.1†   | <0.0001 |
| e' (cm/s)            | 14.7±2.0      | 18.0±5.0†  | 10.8±1.2* | 9.7±3.2†   | <0.001  |
| a' (cm/s)            | 8.7±2.4       | 11.2±2.7   | 12.8±1.9* | 5.6±3.0*   | <0.001  |
| ICT (ms)             | 45.3±11.9     | 57.6±12.1  | 82.5±15.9*| 61.9±22.1* | <0.01   |
| IRT (ms)             | 19.6±9.3      | 12.6±16.0  | 96.0±18.8| 44.8±27.3* | <0.0001 |
| s’ duration (ms)     | 259.0±28.1    | 264.8±37.4 | 252.8±48.0| 287.4±33.8 | NS      |
| e’ duration (ms)     | 203.5±30.6    | 170.1±48.9 | 161.3±29.6| 158.7±27.8*| <0.001  |
| a’ duration (ms)     | 112.9±13.7    | 136.4±42.2 | 146.0±23.2| 129.4±23.9 | NS      |

*P<0.05, †P<0.01 and ‡P<0.001 vs. controls. BSA, body surface area; 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; NS, not significant; Qp/Qs, pulmonary to systemic blood flow ratio; RVEDP, right ventricular end-diastolic pressure; RVEDV, right ventricular end-diastolic volume; RVEF, right ventricular ejection fraction; RVSP, right ventricular systolic pressure.
Cardiac Catheterization

All patients underwent cardiac catheterization within 3 days of echocardiography, but the group with PAH was not assessed by right ventriculography. Catheterization and angiography using an Integris Allura 9 Biplane (Phillips Medical Systems, Best, The Netherlands) proceeded using 4–6Fr 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 the area-length method for the left ventricle (LV) and Simpson’s rule for the RV using quantitative CAW2000 cardiac analysis software (ELK Corporation, Osaka, Japan).

Statistical Analysis

All data are expressed as mean±standard deviation (SD) or as medians with the 5–95th percentiles. Statistical significance was determined using the Mann-Whitney U-test or the Kruskal-Wallis test followed by Dunn’s test, as appropriate. Linear regression analyses were performed for the correlations, and Pearson’s correlation coefficients were calculated. All statistical data were calculated using Prism version 6.0 (GraphPad Software, San Diego, CA, USA) installed on a desktop computer. A value of P<0.05 (2-sided) was considered statistically significant. Intra- and interobserver reproducibility of TDI measurements was assessed using Bland-Altman analysis in a blinded manner. Data were recorded at 5-min intervals by observers 1 and 2 from 20 of the participants (ASD, n=3; PAH, n=2; TOF, n=5; controls, n=10). For intraobserver variability, data were analyzed twice, 8 weeks apart. Interobserver variability was assessed by analyzing data from 2 separate observers blinded to each other’s results.
Results

Patient Characteristics

Among the 43 healthy children, 2 with arrhythmia or a small ASD and 1 with inadequate data from the long-axis view of the RVOT were excluded; 1 and 2 patients in the ASD and TOF groups, respectively, were excluded from all subsequent analyses because of suboptimal images from poor echocardiographic windows. Accordingly, the study group included 40 healthy children (mean age, 7.3±1.7 years; range, 5.0–10.0 years), 15 patients with ASD (mean age, 7.2±1.6 years; range, 5.0–9.5 years), 6 with PAH (mean age, 7.8±2.1 years; range, 6.1–10.0 years) and 32 with TOF (mean age, 7.3±1.6 years; range, 5.0–10.0 years).

Table 1 shows the clinical, echocardiographic, and hemodynamic data of the participants. Age, height, weight, body
Figure 1 shows a representative example of the color TDI and profile of the pulmonary annular motion velocity in a healthy child. The region of interest was positioned on the RV free wall side of the pulmonary annulus. The pulmonary annular motion velocity of the septal wall side was also measured for comparison in control group. Because the septal side of RVOT is part of the ventricular septum, we considered that the septal pulmonary annular motion velocity would be influenced by both RV and LV performance. Therefore, the pulmonary annular motion velocity waveform of the RV free wall side was applied to evaluate in the subsequent patient group study.

Figure 1B shows the pulmonary annular velocity curve obtained from the RV free wall side. Figure 1C is the TDI recording of the septal pulmonary annular motion. The tricuspid annular motion is shown in Figure 1D 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 the pulmonary annular motion than in the tricuspid annular velocity curve. The shapes of the e' and a' waves in diastole were similar. The comparison between the RV free wall side and the septal wall side of pulmonary annular motion is shown in Figures 1E–H. The peak velocities of s1' and e' were significantly higher on the free wall side than on the septal wall side (11.2±2.1 vs. 9.1±1.6 cm/s and 11.9±1.9 vs. 10.4±1.7 cm/s, respectively; P<0.001 for both), whereas the s2' and a' wave velocities were significantly lower on the free wall side (4.4±1.2 vs. 6.4±1.1 cm/s, P<0.001 and 4.7±2.1 vs. 5.9±1.7 cm/s, P<0.05, respectively).

Next, we examined the relationship between the pulmonary annular and tricuspid annular motion velocities in each wave of the healthy control group (Figure 2). The QRS duration was significantly increased in the TOF group. LV end-diastolic dimension, LV fractional shortening, and LV ejection fraction (EF) were significantly lower in the PAH than in the control group. Because the control group did not undergo cardiac catheterization, a statistical difference was not calculated for the hemodynamic data obtained from the invasive examination. RVEF and RV end-diastolic volume were not evaluated in the group with PAH, which was not assessed by right ventriculography.

Figure 3. Representative recording of pulmonary annular velocity and tricuspid annular velocity by tissue Doppler imaging (TDI) from each patient group. The pulmonary annular motion velocity was obtained from the RV free wall side. Pulmonary and tricuspid annular velocity waveforms from a 6-year-old girl with ASD (A,B), 10-year-old boy with PAH (C,D), and a 9-year-old girl with TOF (E,F). ASD, atrial septal defect; PAH, pulmonary arterial hypertension; TOF, tetralogy of Fallot.
Figure 4. Peak velocity of s1', s2', e', and a' obtained from pulmonary annular velocity waveforms in each group. Peak velocity of s1', s2', e' and a' waves (A–D). Boxes show distribution of peak velocity (25th and 75th percentiles; central line, median). Vertical lines represent the range between the 5th and 95th percentiles. *P<0.05, †P<0.01 and ‡P<0.001 vs. controls.

Figure 5. Comparison of ICT, IRT, s1', s2', e', and a' wave duration obtained from pulmonary annular velocity waveform in each group. ICT, IRT and duration of s1', s2', e' and a' waves are shown in (A–F). Boxes show distribution of peak velocity (25th and 75th percentiles; central line, median). Vertical lines represent the range between the 5th and 95th percentiles. *P<0.05, †P<0.01 and ‡P<0.001 vs. controls. ICT, isovolumic contraction time; IRT, isovolumic relaxation time.
than in the control group (15.0 ± 2.4 vs. 11.2 ± 2.1 cm/s, P<0.01 and 6.0 ± 0.9 vs. 4.4 ± 1.2 cm/s, P<0.001, respectively), whereas the e' and a' waves did not significantly differ between the ASD and control groups (10.9 ± 2.0 vs. 11.9 ± 1.9 and 8.3 ± 5.5 vs. 4.7 ± 2.1 cm/s, respectively). The systolic s1' and s2' waves and the diastolic waves of e' and a' were significantly lower in the PAH group than in the control group (8.5 ± 1.2, 3.2 ± 0.4, 6.8 ± 1.6 and 3.2 ± 0.7 cm/s, P<0.05, P<0.05, P<0.001, and P<0.05, respectively). The peak velocities of s1', s2', e', and a' in the TOF group were 5.3 ± 2.2, 3.4 ± 1.4, 8.2 ± 2.9, and 3.1 ± 1.4 cm/s, respectively, all of which significantly lower than in the con-

Figure 3 shows representative recordings of the pulmonary and tricuspid annular velocity waveforms in the patients. Although the configuration, peak velocity, and duration of each wave of pulmonary annular motion differed depending on the cardiac disease, systolic bimodal waves and diastolic e' and a' waves were evident in all groups. Figure 4 compares the peak velocity of each wave in the 4 groups. The peak velocity of both s1' and s2' was significantly higher in the ASD group than in the control group (15.0 ± 2.4 vs. 11.2 ± 2.1 cm/s, P<0.01 and 6.0 ± 0.9 vs. 4.4 ± 1.2 cm/s, P<0.001, respectively), whereas the e' and a' waves did not significantly differ between the ASD and control groups (10.9 ± 2.0 vs. 11.9 ± 1.9 and 8.3 ± 5.5 vs. 4.7 ± 2.1 cm/s, respectively). The systolic s1' and s2' waves and the diastolic waves of e' and a' were significantly lower in the PAH group than in the control group (8.5 ± 1.2, 3.2 ± 0.4, 6.8 ± 1.6 and 3.2 ± 0.7 cm/s, P<0.05, P<0.05, P<0.001, and P<0.05, respectively). The peak velocities of s1', s2', e', and a' in the TOF group were 5.3 ± 2.2, 3.4 ± 1.4, 8.2 ± 2.9, and 3.1 ± 1.4 cm/s, respectively, all of which significantly lower than in the con-

Inter- and intraobserver variabilities (bias±2 SD [95% limit of agreement]) are shown.

Table 2. Inter- and Intraobserver Reproducibilities in an Assessment of Pulmonary Annular Motion Velocity

| Parameter             | Interobserver variability | Intraobserver variability |
|-----------------------|---------------------------|---------------------------|
| Peak velocity of s1' (cm/s) | 0.18±2.87               | −0.14±2.44               |
| Peak velocity of s1' (cm/s) | −0.03±1.60              | −0.02±1.81               |
| Peak velocity of e' (cm/s)  | 0.62±1.92               | 1.65±1.62               |
| Peak velocity of a' (cm/s)  | 0.17±2.43               | 0.16±2.41               |
| ICT (ms)              | −6.30±16.11             | −5.31±10.12             |
| IRT (ms)              | 1.21±11.05              | −1.01±9.15              |
| s1' duration (ms)     | 3.62±10.35              | 1.62±8.33               |
| s2' duration (ms)     | −0.32±24.75             | −0.32±18.85             |
| e' duration (ms)      | 0.73±12.14              | −0.53±10.14             |
| a' duration (ms)      | 6.21±9.31               | 3.21±4.31               |

Inter- and intraobserver variabilities (bias±2 SD [95% limit of agreement]) are shown.
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Reproducibility

The inter- and intraobserver reproducibilities of the TDI analysis of pulmonary annular motion were determined from Bland-Altman analysis of 20 randomly selected participants (ASD, n=3; PAH, n=2; TOF, n=5; control, n=10). Figure 6 shows the Bland-Altman plots for interobserver variability (bias±2SD [95% limit of agreement]) and Table 2 shows the inter- and intraobserver reproducibilities.

Discussion

The present results showed that the velocity of the pulmonary annular motion appears to reflect RVOT performance. We found pulmonary annular TDI to be a simple, rapid, reproducible, and highly distinctive method for evaluating RVOT function. The differences in parameters between patients and healthy controls were obvious. Furthermore, the pulmonary annular velocity had a distinctive waveform and a different configuration from tricuspid annular motion, which indicates RV longitudinal function.

Assessment of RV function in various diseases is important but challenging because of the complex anatomy and geometry of the RV, which has limited functional evaluations. Current quantitative methods such as 2D fractional area change (FAC), TAPSE, tricuspid s’ wave of TDI, and 3D echocardiography all have limitations.7 FAC does not necessarily represent the EF of the entire RV, and TAPSE and tricuspid s’ measure only the longitudinal displacement of the lateral RV wall; 3D echocardiography is limited by the current imaging quality of RV borders.10-12 Previous studies have proposed estimating global RV function using an echocardiographic parameter such as s’, TAPSE, or longitudinal strain. However, this would be quite difficult because these parameters show RV function in only 1 direction. Furthermore, ventricular function might be inhomogeneous and have different properties depending on the region and direction, as well as the nature of the disease or type of surgery.

To the best of our knowledge, this is the first application of pulmonary annular motion velocity obtained by TDI as a tool for RVOT functional assessment. We considered the septal side of the pulmonary annular motion velocity can be affected not only by RV function, but also LV function, because the septal side of the RVOT is part of the ventricular septum, and adjacent to the LV. Therefore, we applied the pulmonary annular motion of the RV free wall side to the evaluation of RVOT performance in the patient group study. However, the methodology including the location of a sample volume should be studied in future.

We selected the 3 disease groups because they had RV volume overload (ASD), RV pressure overload (PAH) and surgically repaired RVOT (TOF), respectively. Although we did not attempt to elucidate relationships between pulmonary annular motion velocity and the entire RV performance, the parameters obtained from pulmonary annular motion clearly differed between patients and controls.

The peak velocities of s1’ and s2’ were significantly higher in the ASD group than in the control group. These patients usually have preserved RV function despite mild to moderate RV volume overload. Previous tricuspid annular motion studies in patients with ASD have shown significantly increased longitudinal contraction compared with controls.13 Our finding that pulmonary and tricuspid annular motion velocities were increased is compatible with those results. However, the pulmonary annular velocities of e’ and a’ did not significantly differ, whereas the tricuspid annular e’ was significantly higher than control values (Table 1). These results indicate that the difference in diastolic properties originated from the RV geometry.

The peak velocity of the s1’, s2’, e’, and a’ waves was significantly lower in the PAH group than in the control group. These results suggested that severe pressure overload depresses RV systolic and diastolic function. However, the tricuspid s’ wave velocity was not significantly decreased compared with the control group (Table 1). LV apical motion enhances the rocking motion of the entire heart as a result of a failing RV in patients with PAH.14 Tricuspid s’ and TAPSE are reportedly enhanced not by RV contraction, but the overall rocking motion of the heart.15 The pulmonary s1’ and s2’ might identify patients with reduced RV systolic function more accurately than the tricuspid annular s’ wave. The peak velocity of the pulmonary annular a’ was significantly reduced, whereas the tricuspid a’ was significantly increased in the PAH group compared with the controls. Annular motion is presumed to be influenced by the RV wall motion and the adjacent structures. Tricuspid annular motion might be affected by right atrial contraction and pressure in late diastole, and the pulmonary annular motion might be affected by pulmonary arterial pressure and compliance rather than by RV active motion. These conditions might contribute to the contrary results regarding the peak velocity of the a’ wave in PAH.

Patients with surgically repaired TOF have impaired systolic function of the RVOT.14 Determination of s1’ and s2’ might be useful to evaluate RVOT performance in patients with TOF. Myocardial damage induced by cardiac surgery and RVOT reconstruction might have negative effects on these parameters.8,14 Furthermore, the pressure-loaded RV induced by RVOT stenosis, pulmonary stenosis or pulmonary hypertension could influence RVOT function. On the other hand, RV volume overload caused by pulmonary regeneration in TOF could influence and augment pulmonary annular motion as in ASD.

The contraction of the RVOT and that of the RV body are important determinants of global RV systolic function in TOF. Greutmann et al found that severely decreased RVOT systolic function in TOF patients with surgically reconstructed RVOT can be compensated for by increased radial and transverse shortening of the RV body.14 Therefore, pulmonary annular motion velocity might be worth measuring in all patients with repaired TOF. Although in the normal heart it is reported that the function of the inflow and outflow components of the RV can be closely related,16 this relationship would be weak and unpredictable in patients with TOF.17 Our study also demonstrated a significant correlation between pulmonary and tricuspid annular motions in the peak velocities of the s1’ and e’
waves in the healthy control group. It would be useful to investigate the difference between pulmonary and tricuspid annular motion velocities in various cardiac diseases to provide insight into RV pathological changes. Furthermore, our comparison of the RV free wall side and the septal side of the pulmonary annulus motion was confined to the healthy control group. Further studies of patient groups are necessary to determine the utility of both sides of pulmonary annular motion.

Our results suggested that pulmonary annular velocity reflects RVOT performance, which in turn indicates RV contractility, pressure and volume overload, pulmonary hypertension and surgical damage to the myocardium. Measuring pulmonary annular motion consequently provides additional information about normal function of the healthy pediatric RVOT and together with the established longitudinal RV functional parameters, TAPSE and s’, may provide detailed assessment of global RV performance in children with cardiac diseases. Furthermore, it would be useful for the evaluation of RV performance to investigate the relationship and the difference between the pulmonary annular motion and tricuspid annular motion in various cardiac diseases.

Study Limitations
The sample cohort was relatively small, but we compared TDI parameters between patients and age-matched healthy individuals, and found distinctive waveforms in each group. We did not compare the relevance of tissue Doppler-derived pulmonary annular motion with that of more accurate and non-echocardiographic modalities such as magnetic resonance imaging. The spatial orientation of myocardial fibers and their motion have complex 3D models. Some degree of angulation between the Doppler beam and the true direction of myocardial movement might exist. Furthermore, we obtained the long-axis view of the RVOT, and made the ultrasound beam parallel to the direction of the pulmonary annular motion. We considered that this is the most appropriate cross-sectional view to obtain the shortening from the RV apex to the pulmonary annulus. Although the angulation might be small, the data presented herein are for velocity along the direction of the Doppler beam and might not indicate actual myocardial velocity. Moreover, because pulsed TDI is limited by the stationary sample volume being positioned on a moving target, the effect of translation is not eliminated. The motion of the RV free wall might be restricted by postoperative adhesions in patients with TOF. Such RV adhesion to the chest wall would affect both postoperative pulmonary and tricuspid annular motions measured using TDI. Lastly, we aimed to establish pulmonary annular motion as echocardiographic parameters of RVOT function and not as an estimation of global RV function. Thus, the study design did not allow analysis of whether pulmonary annular velocity can be an alternative index to entire RV function. Further studies are needed to determine whether pulmonary annular motion could serve as an important guideline for therapy. Further studies are needed to determine whether pulmonary annular motion could serve as an important guideline for therapy. Further studies are needed to determine whether pulmonary annular motion could serve as an important guideline for therapy. Further studies are needed to determine whether pulmonary annular motion could serve as an important guideline for therapy.

Conclusions
Pulmonary annular TDI is a promising echocardiographic tool for evaluating RVOT function.

Conflict of Interest
None.

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