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Assessment of intracardiac flow and vorticity in the right heart of patients after repair of tetralogy of Fallot by flow-sensitive 4D-MRI

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Abstract

Objectives—To comprehensively and quantitatively analyze flow and vorticity in the right heart of patients after repair of tetralogy of Fallot (rTOF) compared with healthy volunteers.

Methods—Time-resolved flow-sensitive 4D MRI was acquired in 24 rTOF patients and 12 volunteers. Qualitative flow evaluation was based on consensus reading of two observers. Quantitative analysis included segmentation of the right atrium (RA) and ventricle (RV) in a 4-chamber view to extract volumes and regional hemodynamic information for computation of regional mean and peak vorticity.

Results—Right heart intra-atrial, intraventricular and outflow tract flow patterns differed considerably between rTOF patients and volunteers. Peak RA and mean RV vorticity was significantly higher in patients ($p=0.02/0.05$). Significant negative correlations were found between patients' maximum and mean RV and RA vorticity and ventricular volumes ($p<0.05$). The main pulmonary artery (MPA) regurgitant flow was associated with higher RA and RV vorticity, which was significant for RA maximum and RV mean vorticity ($p=0.01/0.03$).

Conclusion—The calculation of vorticity based on 4D flow data is an alternative approach to assess intracardiac flow changes in rTOF patients compared with qualitative flow visualization. Alterations in intracardiac vorticity could be relevant with regard to the development of RV dilation and impaired function.

Keywords

4D Flow MRI; Tetralogy of Fallot; Vorticity; Right heart; Intracardiac Flow

Introduction

Tetralogy of Fallot (TOF) is the most common cyanotic congenital heart defect with an incidence of about 10% for all congenital heart diseases (CHD)^{1, 2}. Based on the four main defects, which include pulmonary stenosis, ventricular septal defect (VSD), overriding aorta, and right ventricle hypertrophy, TOF patients usually undergo corrective surgery in their first year of life. Advances in diagnosis, surgical techniques and postoperative treatment have led to an increasing number of patients reaching adulthood with a high survival rate of almost 90% at 30 years^{3, 4}. Surgery for TOF patients consists of VSD closure and reconstruction of the right pulmonary outflow tract (RVOT)⁵. There are three main different types of RVOT reconstruction: transannular patch, homograft or a valve preserving technique. The most common postsurgical complication is pulmonary valve insufficiency which can lead to secondary right ventricular (RV) volume overload^{6, 7}.

The growing amount of long-term survivors and the need for ongoing clinical surveillance is currently driving the need for optimized cardiovascular imaging³. Cardiovascular magnetic resonance (CMR) outperforms echocardiography for assessing RV volume, quantifying pulmonary valve insufficiency and monitoring progression of pulmonary regurgitation and RV dilatation^{8, 9}. In complement to standard CMR protocols, time-resolved 3D phase contrast MRI (also called 4D Flow MRI) facilitates the visualization and quantification of blood flow in the cardiovascular system in order to obtain detailed information on vascular hemodynamics^{10, 11}. Thus, the technique may be a promising method to evaluate altered blood flow in patients with CHD with complex anatomy and hemodynamics^{12–18}.

4D flow is especially relevant given that characterization of intracardiac flow is much more challenging than flow evaluation in vessels because of the three-dimensional geometry of the cavity and varying dynamics during the heart cycle^{19, 20}. In particular, the RV has a more complex shape than the left ventricle (LV), which can complicate flow assessment. Previous studies have tried to assess the complexity of intraventricular blood flow, including the assessment of retrograde and vortical flow, by using descriptive visualization or by the calculation of kinetic energy^{21–24}. Of particular interest are recent reports which have qualitatively characterized the presence of vorticity in the RV and right atrium (RA)¹³. Vorticity (i.e. the local spinning motion of blood) is a promising hemodynamic biomarker given that it can be quantitatively computed and it has been shown to correlate to LV function²⁵. Therefore in this study, we aim to evaluate how flow disturbances are impacted in the RA and RV of TOF patients after repair (rTOF), as assessed by the computation of blood vorticity.

Methods

Patients and Volunteers

Twenty-four rTOF patients (mean age 11.7 ± 5.8 years, range 1–24 years; weight 39.8 ± 18.5 kg, range 6–71 kg; 16 male) and twelve healthy volunteers (age 23.3 ± 1.6 years, range 21–26 years; weight 65.7 ± 10.3 kg, range 54–91 kg; 7 male) were prospectively enrolled for CMR with whole heart flow-sensitive 4D Flow MRI.

The mean interval between surgery and MRI was 10.5 ± 6.1 years. Fourteen patients underwent transannular repair with consecutive pulmonary valve insufficiency, six pulmonary valve-preserving surgery, and four valve conduit implantation. 18 patients had mild (grade I), three patients moderate (grade II) tricuspid regurgitation known by echocardiography.

The study was approved by the local ethics committee. Written informed consent from each participant over 18 years of age and from parents or legal guardians for patients under age were obtained prior to MRI examinations.

Magnetic resonance imaging

Volumetric assessment—CMR examinations were performed on 1.5T (23 patients) and 3T MR (1 patient and all volunteers) systems (AVANTO or TRIO, Siemens, Germany). The standard clinical CMR protocol consisted of ECG-gated 2D CINE SSFP planes ($TE = 1.24\text{--}1.36$ ms, $TR = 41.48\text{--}45.21$ ms, slice thickness = 5 mm, field of view (FOV) = $256\text{ mm} \times 208\text{ mm}$, flip angle = $75\text{--}80^\circ$) in the ventricular short-axis and axial plane for the assessment of RV volumetry and function. End-diastolic and end-systolic RV volumes (EDV, ESV), ejection fraction (EF) and stroke volume (SV) were obtained by manual segmentation of the corresponding slices using commercially available software (Argus, Siemens). The functional parameters were normalized to the body surface area (BSA), as indicated by $ESVi$, $EDVi$ and SVi . The CINE SSFP sequences were performed in breath-hold during expiration in all patients except four who were sedated due to their young age (<6 years).

Two patients did not receive ventricular volumetry because the MRI scan was solely done to evaluate the pulmonary arteries. In healthy volunteers only flow-sensitive 4D MRI was performed.

Flow-sensitive MRI—In patients, 4D Flow MRI was performed subsequent to the clinical sequences. All acquisitions were synchronized to the heart rate and breathing using prospective ECG-gating and adaptive diaphragm navigator gating¹⁰. The MRI sequence consisted of a k-space segmented RF-spoiled gradient echo sequence with interleaved 3-directional velocity encoding. Data were acquired in a sagittal oblique 3D data volume individually adapted to include the entire heart, thoracic aorta, and main pulmonary arteries. Imaging parameters were as follows: velocity sensitivity = $150\text{--}200$ cm/s, views per segment = 2, $TE = 2.3\text{--}2.6$ ms, $TR = 4.7\text{--}5.1$ ms, field of view (FOV) = $169\text{--}315\text{ mm} \times 300\text{--}420\text{ mm}$, spatial resolution = $2.1\text{--}3.2 \times 1.6\text{--}2.6 \times 2.4\text{--}3.6\text{ mm}^3$, temporal resolution = $37.6\text{--}40.8$ ms, flip angle = 15° , scan time $\sim 10\text{--}20$ min. All patients received intravenous gadolinium administration (d-BOPTA, Prohance Bracco, dose = $0.1/0.2$ mmol/kg body weight) for the clinically indicated MR-angiography prior to 4D Flow MRI. MRI scans in volunteers were done without contrast medium; therefore the flip angle was reduced to 7° to optimize signal-to-noise ratio (SNR) as calculated via the Ernst angle.

All 4D data were preprocessed using a home-built Matlab-based software tool which provides phase unwrapping algorithms, several noise-masking functions and eddy-current correction²⁶.

Flow visualization

Data analysis included the calculation of a 3D phase contrast (PC) MR angiogram (MRA) from 4D Flow MRI data to facilitate anatomical orientation for visualization. For a visual overview of 3D blood-flow in the right heart and pulmonary arteries, visualization of 3D particles traces was performed with EnSight (CEI, Apex, NC, USA)^{27, 28}. The particle traces were emitted from manually positioned planes in the superior and inferior caval vein (SVC/IVC), the RA, the tricuspid valve (TV), the main pulmonary artery (MPA), and right (RPA) and left pulmonary arteries (LPA). Additional anatomic visualization was guided with co-registered 2D CINE SSFP images, acquired as standard of care for cardiac function assessment. Particle traces were color-coded according to their origin and local velocity. The qualitative evaluation was performed by two experienced readers (one radiologist and one pediatrician) in a consensus reading. For this purpose, images were viewed dynamically and rotated in any orientation for inspection of intra-atrial and intraventricular flow features. For RA evaluation, a defined view from the patients' right with the sinus venosus in the posterior position was particularly suitable for vortex observation.

Flow and Vorticity quantification

Blood flow volume and regurgitant fraction (RF) were quantified from 4D Flow data. Volume flow rate was measured using a cut-plane placed in the MPA at the approximate level of the pulmonary valve (PV). The RF was computed from the net retrograde flow at the PV position and reported as a percentage of the net forward flow. For vorticity computation, the 4D flow data was co-registered with a 4-chamber CINE view using EnSight. An analysis plane positioned co-planar to the 4-chamber CINE view was used to extract the 4D flow velocity fields and subsequently used to calculate vorticity throughout the cardiac cycle (Fig. 1). Since vorticity magnitude describes the local spinning motion of a fluid near a point, vorticity was computed by the formula: $\omega = \nabla \times V$, where ∇ is the curl of its velocity field V and was calculated using a compact derivative algorithm^{29, 30}, which is robust in the presence of complex 3D flow features and signal noise^{31–33}. Finally, to compute regional hemodynamic information, the RA and RV were manually segmented and the regional mean and peak vorticity were computed.

Statistical analysis

For each parameter, the assumption of a normal distribution was assessed using a Kolmogorov-Smirnov and Shapiro-Wilk test. The association between RA and RV vorticity, flow measurements, and volumes were assessed by Pearson's correlation and a 2-sided t-test. $P < 0.05$ was considered significant. Statistical analysis was performed with SPSS 17 (SPSS, Chicago, Ill, USA).

Results

General Results

4D Flow MRI datasets and right heart flow visualization were successfully acquired in all subjects. Vorticity could not be calculated in two volunteers due to poor signal caused by

motion artifacts. RV volumetry was performed in 22 patients who received CINE SSFP sequences.

Qualitative Assessment

Right atrium—12/12 healthy volunteers showed a clockwise RA vortex (Figure 2a). 9/24 rTOF patients had remarkable additional vortices in diastole besides the normal clockwise vortex (Fig. 2d), except one patient who showed a vortex with anticlockwise direction. In healthy volunteers there were also additional vortices in 5/12 examinations but far smaller compared to rTOF patients.

Right ventricle—RV flow in volunteers was similar between subjects, which consisted of a filling pattern in the region of the tricuspid valve similar to that commonly seen during LV filling (i.e. a normal inflow jet with vortex formation adjacent to the jet, as illustrated in Fig 2b)³⁴. Parts of the intraventricular flow were immediately directed towards the RVOT, followed by slight helical flow in the RVOT during systole. Very little flow was found at the apex. None of the volunteers had retrograde flow due to PV regurgitation.

In patients however, the flow features were very heterogeneous with more direct intraventricular flow during diastole which was directed toward the apex or the ventricular base. All patients had vortices in the RVOT due to regurgitation because of PV insufficiency (Fig 2f).

Quantitative Assessment

Volumetry—The results of the RV volumetry are summarized in table 1. 15 patients had RV EDV > 100ml/m² normalized to BSA, six of them impaired RV function (EF<45%).

Vorticity—Figure 3 shows that the calculated RA peak vorticity was significantly higher ($p=0.02$) in rTOF patients compared to volunteers. Values for mean vorticity were slightly higher in patients but not significant ($p=0.3$).

Peak vorticity and mean vorticity showed a trend toward higher values in the patients' RV as compared to volunteers (peak vorticity $p=0.06$; mean vorticity $p=0.06$) (Fig. 3).

Significant negative correlations were observed between the RA maximum vorticity and EDV ($r=-0.44$, $p=0.039$) and ESV ($r=-0.48$, $p=0.024$). This correlation also existed between the RV maximum vorticity and EDV ($r=-0.52$, $p=0.014$) and ESV ($r=-0.61$, $p=0.003$). The correlations were higher for the BSA normalized values ESV_i (RA maximum vorticity: $r=-0.54$, $p=0.01$; RV maximum vorticity: $r=-0.69$, $p=0.001$) and EDV_i (RA maximum vorticity: $r=-0.58$, $p=0.005$; RV maximum vorticity: $r=-0.61$, $p=0.002$).

The correlations between mean RA / RV vorticity and ventricular volumes were also significant ($p=0.021$; $p=0.023$) (table 2 and fig 4).

A significant association was detected between RA maximum vorticity and MPA backward flow ($p=0.013$), and PV regurgitant fraction ($p=0.045$). RV mean vorticity was also significantly associated with MPA backward flow ($p=0.032$) (Table 2 and Fig. 5).

Discussion

The results of this study demonstrate the use of 4D Flow MRI to evaluate and quantify intracardiac blood flow patterns in rTOF patients. The description and characterization of flow in the RV is very challenging because of the complex shape as compared to the LV^{35, 36}. In patients with CHD, RV function is frequently impaired and therefore of high diagnostic importance³⁷. Among CHD subjects, TOF represents one of the most common diseases referred for postsurgical CMR. The long-term sequelae depend partially on the type of corrective surgery, which can lead to RVOT ectasia, pulmonary or tricuspid valve insufficiency, and RV dilatation or failure^{9, 38, 39}, for which CMR plays an important diagnostic role.

Few studies have investigated intracardiac flow patterns in the right heart with 4D flow^{13, 35, 40, 41}. Descriptions of left heart flow are more numerous: 4D Flow MRI was used in a previous study for evaluation of intraventricular flow in which flow patterns showed differences related to age, gender, blood pressure, and ventricular geometry⁴². Another study demonstrated that patients with dilated cardiomyopathy and mild left ventricular remodeling had altered diastolic flow patterns and different energetics compared with healthy controls⁴³.

The first study examining intracardiac 4D Flow MRI of the RV examined 10 healthy volunteers in which the flow patterns were found to differ from those in the LV³⁵. A ring-like vortex surrounding the tricuspid inlet was initially described during diastole and then was subdivided into several flow regimes which represented functional components to describe intraventricular flow. It was postulated that diastolic flow patterns create favorable conditions for systolic ejection via diastolic and systolic coupling.

Right heart intracardiac flow was first assessed by 3D real-time echocardiography in dogs using computational fluid dynamic simulations for investigating RV flow patterns⁴⁴. It was postulated that intraventricular diastolic vortices represent energy-preserving flow structures which facilitate ventricular filling. A recent method which combines 4D echocardiography with numerical simulations was used to assess 3D RV flow³⁶. Similar to the previously reported results^{35, 44}, vortex formations downstream from the tricuspid valve were described as breaking abruptly on the initialization of systole, resulting in a slight helical systolic outflow.

Assessment of normal flow in healthy subjects is a challenging task but it is especially difficult to comprehensively evaluate the heterogeneous intracardiac flow features found in rTOF patients by qualitatively assessing streamline and pathline flow visualizations. Nonetheless, previous 4D Flow MRI studies found marked differences between rTOF patients' RVOT and pulmonary artery flow features and those of volunteers^{13, 40}. The study by Geiger et al. was focused on flow patterns in the pulmonary arteries and did not investigate intraventricular flow⁴⁰. Asymmetric flow distribution to the pulmonary arteries, severe regurgitation and pronounced vortices were the main pathological findings. The alteration of RA flow in rTOF patients resulted in more vortices in addition to the normal clockwise vortex and an increased number of intraventricular vortices¹³. Similarly, our patient group differed concerning the abnormal RA vortices as well as RVOT vortices which

often extended down to the RV due to flow regurgitation during diastole. In our study, the volunteers showed more RA vortices but they were smaller in size, compared to the patients most of whom had mild tricuspid insufficiency. Additionally, one patient also exhibited a RA vortex with anticlockwise direction; however, there were no patently different demographics or history (such as ‘type of repair’, age, gender and cardiac function) that clearly resulted in this finding. The qualitative flow results in volunteers is similar to what was described by Fredriksson³⁵. Almost no flow was detected in the RV’s apical third because of low velocities in this area (in comparison to the *venc* value). In patients, there was also marginal flow in the apex. Early diastolic flow was frequently directed to the ventricular base because of the retrograde flow, which caused inflowing portions to be re-directed away from the RVOT, where heterogeneous vortices were generated.

Given the heterogeneity among patients and the purely descriptive nature of these hemodynamic observations, significance testing is challenging which was the driving motivation to quantitatively compute vorticity. Previous quantitative approaches have been published to quantify vortex rings in the LV by using a relatively new flow analysis method, called Lagrangian Coherent Structures (LHS)⁴⁵. LHS enabled quantification of vortex ring formation downstream the mitral valve during LV filling. Another recent study used the Lambda2-method to extract 3D LV vortex ring cores from flow velocities in 4D flow MRI⁴⁶. The proposed method can characterize and quantify diastolic 3D vortex rings in vivo and might be helpful in understanding hemodynamics in patients with mitral valve or LV dysfunction.

Another study compared 4D flow MRI with retrospective valve-tracking and 2D PC for the assessment of tricuspid and pulmonary valve flow in corrected Fallot patients and controls⁴¹. 4D flow MRI showed strong correlations to 2D PC, and was found to reliably assess pulmonary regurgitation and RV diastolic function in TOF patients. A similar approach using 4D flow MRI was recently proposed by Calkoen et al.⁴⁷. In this study the standard of care was using a static orthogonal plane to the MPA for evaluating pulmonary valve flow. A recent publication applied the quantification of ventricular kinetic energy (KE) extracted from 4D flow MRI data to assess ventricular performance in rTOF patients²². Ventricular KE was higher in patients than in volunteers suggesting that this could represent a potential biomarker of ventricular impairment.

Our study aimed to overcome the limitations of qualitative flow grading by using another quantitative method: vorticity. We hypothesized that this could be an appropriate method to evaluate the intraventricular flow changes in CHD patients. Standard CINE planes were used to isolate the computed vorticity magnitudes (using 4D flow velocity fields) and the vorticity was quantified as a function of cardiac territory (i.e. RA and RV). The use of vorticity as a comparison method revealed a higher frequency of vortices in patients compared with volunteers which reached significant levels in the case of RA peak vorticity. The significantly higher RA peak vorticity might be caused by additional vortices in patients, secondary to tricuspid valve insufficiency, anatomical variations after surgical repair³⁹, or the abnormal timing of RA filling with greater diastolic than systolic filling as observed in previous studies¹³. One would expect more vortices in rTOF patients in general because of pulmonary reflux which hampers regular systolic antegrade flow and thus leads to disturbed

flow patterns. Indeed, a significant correlation existed between the MPA regurgitant flow (Fig 2e–f) and the occurrence of vorticity in the right heart (Table 2). Higher vorticity could also be caused by different shaped ventricles or the ventricular overload by enlargement after surgical repair⁴⁸. Patients' mean RV EDV were increased compared to reference values⁴⁹. This explanation seems unlikely given that a negative correlation was found between vorticity and ESVi and EDVi in patients. This negative correlation can be explained by the concept that vorticity is the circulation of fluid per unit area. Thus, if a small ventricle has the same amount of circulation as a large ventricle, the smaller one will have a higher value of circulation per unit area, or, in other words, vorticity will be higher.

A drawback of our study is that the healthy volunteers were not age-matched to the patients. The patients consisted of a very heterogeneous group with a wide range in age and different types of repair. All healthy volunteers were examined at the 3T scanner whereas the patients were examined at the 1.5T MRI, which is used for routine CMR examinations. Although the field strength causes a possible source of variability in the data, we did not observe differences in image quality between the two groups. Previous studies have shown that while exams with and without contrast agent are comparable; those with contrast agent typically have higher SNR⁵⁰. Therefore, the lack of contrast administration in volunteers was counterbalanced by the higher field strength.

Ventricular volumetry was not performed in volunteers, thus a correlation of heart volumes and vorticity is not possible. In some subjects there is incomplete coverage of the apex. This will not have an impact on the results presented here, as there is minimal flow¹³. There was little flow in the RV apical third both in patients and volunteers which could barely be detected due to slow velocities. The use of lower venc settings could optimize flow depiction in this area; however, with lower vencs the risk of flow aliasing in other RV areas increases.

Another study limitation includes the lack of data on inter- and intra-observer variability of the calculated vorticity. Further studies for validation of the proposed method will be mandatory to conclude whether vorticity is a more objective or reliable method to assess flow disorders in rTOF patients compared with the flow visualization based approach. In addition, a four-chamber plane was used to extract 4D flow velocities and calculate vorticity. This approach provided enough information to quantify flow vorticity along the plane, but it did not assess vorticity in the full 3D volume.

While vorticity itself has not been extensively studied in terms of pathological phenomena, preliminary efforts have attempted to understand its association with cardiac function and efficiency. In the cases presented here, the clockwise RA vortex and the umbrella-like pattern around the tricuspid valve were found in volunteers. The appearance of vortices seems to be necessary for optimized flow kinetics and efficiency as previously discussed^{22, 35, 46}. Thus, the sole observation of vorticity does not indicate compromised ventricular function; however, an approach using vorticity analysis may allow for the computation of physiologically normal ranges. Additionally, one should take into account the exact localization and appearance of vortices during the heart cycle to determine the quality of “physiological” and “pathological” vortices. In this context, the aspect of RV-PA coupling which was discussed by Jeong et al.²² is of great interest because turbulent

ventricular flow could lead to changes in KE which results in ventricular-vascular inefficiency in rTOF patients.

Conclusion

In this study, the observation of intracardiac vortices in rTOF patients was correlated with quantitative measurements of intraventricular and intra-atrial vorticity. The proposed approach provides a quantitative method to compute vorticity in regions where complex blood flow is known to occur. Changes in vorticity may be a cause for ventricular energy loss, progressive RV dilatation and functional impairment in many rTOF patients.

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Key points

- 4D flow MRI with vorticity calculation enables a novel approach to assess intracardiac flow
- Significantly higher intracardiac vorticity occurred in patients after repair of Tetralogy of Fallot
- Regurgitant flow in the main pulmonary artery is associated with higher right heart vorticity

Figure 1.

(a) Schematic of the analysis workflow. A PC-MRA generated from 4D flow data was co-registered with 4-chamber SSFP CINE images in order to visualize anatomical landmarks (superior vena cava (SVC), inferior vena cava (IVC), right atrium (RA), right ventricle (RV), and main pulmonary artery (MPA, white cutplane). (b) Qualitative pathline visualization of the flow in the 4-chamber view showing a vortex in the RA (white arrow, rotation axis: right-left). (c) Computation of vorticity in the RA on an analysis plane which was co-registered with the 4-chamber view.

Figure 2.

Vortex patterns in a healthy volunteer and rTOF patient visualized in a 4-chamber plane and a plane in the right ventricular outflow tract which is marked as MPA (Rotation axis: cranio-caudal) (a) A physiologically normal vortex forms in the RA during late systole, which (b) is ejected into the RV in the presence of an inflow vortex; (c) at end-diastole the RV is hemodynamically inactive. (d) A double vortex was found in the RA of a number of patients; (e) RV vortex formation downstream the tricuspid valve; (f) Vortex formation in the RA in presence of pulmonary regurgitation. RA: right atrium; RV: right ventricle; MPA: main pulmonary artery. White arrows indicate pulmonic valve regurgitation. White arrows indicate vortex formation, white cutplane corresponds to MPA location.

Figure 3.

(a) Mean and (b) peak vorticity in the right atrium (RA) and ventricle (RV) in patients and volunteers.

Figure 4.

Mean and peak vorticity in the right ventricle (RV) as it correlates to indexed end diastolic and end systolic volume (EDVi, ESVi).

Figure 5.
Regressions between main pulmonary artery (MPA) backward flow, right atrium (RA) maximum vorticity (a) and right ventricular (RV) mean vorticity (b).

Table 1

Overview of the right heart function parameters of patients with rTOF

	Mean	Std. Deviation
Male sex n (%)	16 (66%)	
Age (yrs)	12	6
EDV (mL)	147.16	63.34
ESV (mL)	74.02	33.43
SV (mL)	73.14	32.86
EF (%)	49.78	7.07
CO (L/min)	5.85	2.54
EDVi (mL/m ²)	119.24	34.40
ESVi (mL/m ²)	60.32	20.90
SVi (mL/m ²)	59.01	17.04
CI (L/min/m ²)	5.02	1.58
BSA (m ²)	1.24	0.43
MPA BF (mL)	-26.93	20.96
PV RF (%)	25.77	13.87

EDV/ESV = enddiastolic and endsystolic volumes, SV = stroke volume, EF = ejection fraction, cardiac output (CO), BSA = body surface area, ESVi/EDVi/SVi = EDV/ESV/SV indexed to BSA, CI = Cardiac index, MPA BF = main pulmonary artery backward flow, PV RF = pulmonary valve regurgitant fraction

Summary of significant correlations between functional cardiac parameters and right atrium (RA) and right ventricle (RV) vorticity. Significant values are in bold ($P < 0.05$).

Table 2

	EDV (mL) [r,P]	ESV (mL) [r,P]	SV (mL) [r,P]	EF (%) [r,P]	EDVi (mL/m ²) [r,P]	ESVi (mL/m ²) [r,P]	SVi (mL/m ²) [r,P]	MPA BF (mL) [r,P]	PV RF (%) [r,P]
Maximum vorticity RA (1/s)	-0.44, 0.039	-0.48, 0.024	-0.36, 0.094	0.21, 0.339	-0.58, 0.005	-0.54, 0.010	-0.50, 0.017	0.50, 0.013	-0.41, 0.045
Maximum vorticity RV (1/s)	-0.52, 0.014	-0.61, 0.003	-0.38, 0.081	0.41, 0.061	-0.61, 0.002	-0.69, 0.001	-0.42, 0.052	0.39, 0.059	-0.31, 0.139
Mean vorticity RA (1/s)	-0.49, 0.021	-0.56, 0.007	-0.37, 0.088	0.28, 0.207	-0.49, 0.021	-0.51, 0.015	-0.36, 0.103	0.35, 0.090	-0.20, 0.355
Mean vorticity RV (1/s)	-0.49, 0.001	-0.75, 0.000	-0.53, 0.011	0.38, 0.078	-0.48, 0.023	-0.54, 0.009	-0.31, 0.158	0.44, 0.032	-0.25, 0.242

EDV= Enddiastolic volume; ESV = endsystolic volume; SV = stroke volume; EF = ejection fraction; EDVi, ESVi, SVi = EDV, ESV, SV indexed to body surface area (BSA); MPA BF = main pulmonary artery backward flow; PV RF = pulmonary valve regurgitation fraction; r = correlation coefficient.