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ORIGINAL ARTICLE

Cardiac Function After Repair of Tetralogy of Fallot: How are the Atria Performing? Pilot Study by Cardiac Magnetic Resonance Imaging

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Abstract The atria of the heart function as reservoir, conduit, and active pump and are critical for ventricular filling and cardiac output. We sought to evaluate right (RA) and left atrial (LA) function in patients after tetralogy of Fallot (TOF) repair by using cardiovascular magnetic resonance. Twelve TOF patients, age 16.7 ± 6.1 years, weight 50.9 ± 14.9 kg, were compared to 10 healthy volunteers, age 18.8 ± 6.8 years, weight 52.3 ± 20.8 kg. Both atria and both ventricles were imaged in short-axis planes by Steady State Free Precession. Volume changes and all derived atrial functional parameters were calculated from the volume/time curves obtained after segmentation on the cine images. Blood flow across the AV valves was used to define ventricular diastolic dysfunction. TOF patients showed similar maximal RA volume compared to controls, but increased volumes at mid-diastole ($p < 0.05$), resulting in a decreased cyclic volume change and atrial filling fraction ($p < 0.01$), and a decreased passive emptying volume and fraction ($p < 0.01$). In patients with diastolic dysfunction, conduit volume was increased ($p < 0.05$), and active emptying volume and fraction tended to be increased, respectively. No significant changes were found in LA, except for a decreased passive emptying fraction ($p < 0.05$). RA function and particularly reservoir function are impaired in TOF patients. The RA conduit/reservoir ratio is increased and reflects the lost of the ability of the atrium to fill the ventricle by pulsatile flow.

Keywords Atrial function · Right atrium · Tetralogy of Fallot · Right ventricle · Cardiovascular magnetic resonance

Introduction

The atria of the heart have three major functions. First, the reservoir function is the ability to expand and receive the venous flow during ventricular systole and to passively empty during ventricular diastole. Reservoir function can be estimated with the passive emptying volume. Second, the pump function represents the atrial contraction during late diastole and is expressed by the active emptying volume. Third, the conduit function is a passive blood transfer from the veins into the ventricles without changing the size of the atrium and is expressed by the conduit volume, calculated as the difference between the ventricular stroke volume and the cyclic volume change of the atrium [6, 23].

Atrial performance is, although often underestimated or neglected, important for good ventricular filling and function. Particularly in patients with borderline ventricular function, as in cases of congenital heart disease, atrial contraction is crucial to maintain ventricular stroke volume [7, 16].

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An enlarged left atrium (LA) has been observed in mitral regurgitation, ventricular failure, myocardial infarction, and arrhythmias, and has been found to be a predictor of outcome in adults [3, 7, 17]. In contrast, knowledge on the function of the right atrium (RA) is scarce [6].

So far, the size and function of the atria have mainly been assessed by echocardiography [11, 12] and angiography [2]. The accuracy of both conventional techniques is limited by measurements that are taken in two dimensions and by the geometric assumptions used; moreover, the atrial volumes are calculated at distinct points of the cardiac cycle. Three-dimensional imaging techniques are more accurate in estimating true atrial size and allow exact characterization of the volumetric changes throughout the cardiac cycle [11, 14, 26]. More recently, Cardiovascular Magnetic Resonance (CMR) imaging has been recognized to be the most accurate imaging modality for evaluating atrial size and function [9, 27]. Reference values in children have been established by 3-dimensional echocardiography [15] and by CMR [14, 21]. However, the role of the atria, and particularly of the RA in congenital heart disease remains understudied. In patients after repair of Tetralogy of Fallot (TOF), single reports exist on evaluation of RA function by tissue Doppler imaging [8] and by CMR [13, 18, 19].

The aim of our study was to assess atrial function by using CMR in patients after TOF repair and to compare it with healthy controls. We hypothesized that in TOF patients; RA function presents specific changes compared to controls.

Materials and Methods

Patient Population

Fourteen patients after surgical TOF repair referred to CMR examination for clinical reasons were prospectively enrolled in the study. Inclusion criteria were age of 30 years or less, ability to perform repetitive breath holds written informed consent of the patients or their legal guardian of 10–15 s duration. Exclusion criteria were requirement of general anesthesia, mitral or aortic regurgitation, tricuspid insufficiency greater than grade I, arrhythmias, such as frequent supraventricular extrasystoles, ventricular extrasystoles, and history of tachycardia, as well as any additional cardiac abnormality that might potentially influence atrial size and function, including residual intracardiac shunts, systemic or pulmonary venous anomalies, or severe outflow tract obstruction. On the day of examination, the following demographic data were collected for each subject: age, height, weight, body surface area, heart rate, and blood pressure.

The control group consisted of 10 healthy young individuals without known cardiac abnormality or other systemic condition that would alter atrial size or function, including obesity, arterial hypertension, diabetes, chronic respiratory disease.

Image Acquisition

CMR was performed with a 1.5 Tesla scanner (Signa HDx, GE Medical Systems, Milwaukee WI, USA) by using an 8-channel phased-array cardiac coil. After obtaining coronal, sagittal, and axial localizers, cine Steady State Free Precession (SSFP) images were acquired in a horizontal and vertical long-axis plane showing both ventricles and both atria. The parameters of the SSFP sequence were as follows: 40 phases/cardiac cycle, TE 1.5–1.8 ms, TR 2.8–3.1 ms, flip-angle 45°, bandwidth 125 kHz, matrix 224 × 224, number of excitations 1, field of view 250–350 mm, views per segment 6–12 depending on heart rate, retrospective cardiac gating. Parameters were optimized for obtaining a temporal resolution of <25 ms.

Atria

By using the cine SSFP sequence, a stack of adjacent slices in the short-axis plane (Fig. 1a) was acquired covering both atria from the atrial base (plane of the atrioventricular valves) to the roof of the atria. A slice thickness of 6 mm without gap was chosen. All images were acquired during breath-holding in end-expiration.

Ventricles

In the patient group, ventricles were imaged in the short-axis plane from the base (plane of the atrioventricular valves) to the apex of the heart. Slice thickness was 6–8 mm with a gap of 0–2 mm as appropriate for body size for obtaining a total of 12–13 slices. All images were acquired during breath-holding.

Atrioventricular Flow

In addition to flow measurements in the main pulmonary artery, side branches, and ascending aorta, as clinically appropriate, blood flow across the atrioventricular (AV) valves was evaluated using the velocity-encoded Phase Contrast (PC) gradient echo cine sequence. A slice perpendicular to the mitral (MV) and tricuspid (TV) valves, placed midways between the tip and the insertion of the valve leaflets was acquired. The flow sensitive PC cine sequence was performed with the following parameters: echo time 5.4–5.9 ms, repetition time of 9.9–10.5 ms, flip-angle 15°, slice thickness 4 mm, velocity encoding

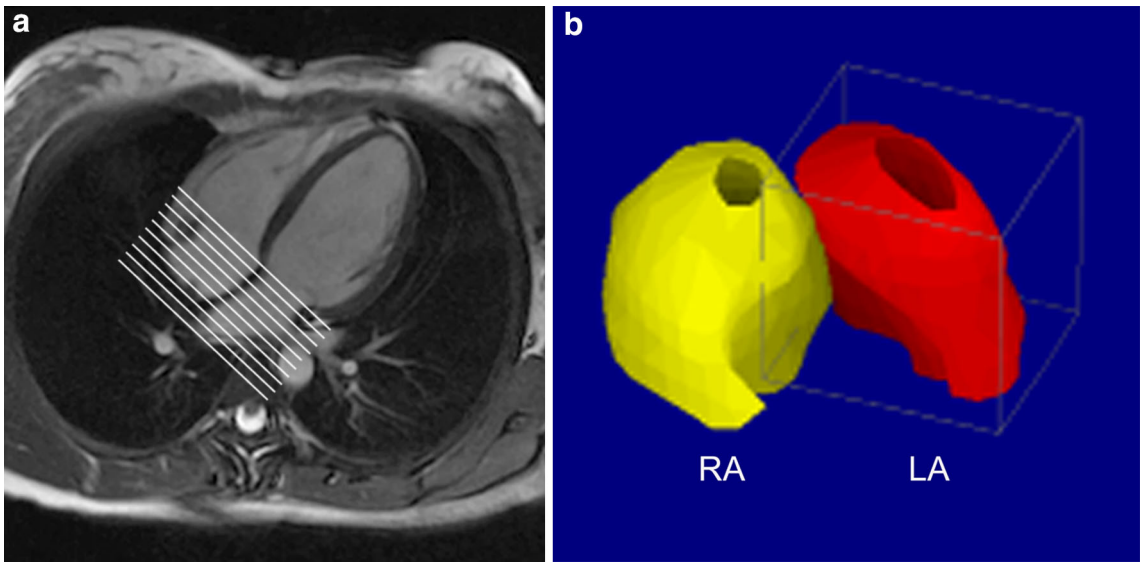

Fig. 1 Acquisition of the steady state free precession images in a short-axis plane covering both ventricles (**a**). After segmentation of each slice in every cardiac phase, the summation disk method provides the real volume of both atria (**b**)

120 cm/s, number of averages 2, views per segment 1, 40 reconstructed cardiac phases, and retrospective gating. Images were acquired during free breathing.

Image Analysis

The acquired data were analyzed offline on a separate workstation using a dedicated software (Mass plus Version 4 and FLOW Version 4, MEDIS, Medical Imaging Systems, Leiden, The Netherlands).

Atria

Endocardial contours of the left and the right atrium were traced manually in every slice and for each of the acquired 40 images (phases) in the cardiac cycle. The level of the AV valves was recognized by observing the movie loop for opposing movement of the atria and ventricles. Both atrial appendages were included in the atrial volumes. In the right atrium, the posterior part of the atrium at the confluence of the caval veins was included in the atrial volume, and the Eustachian valve was used as a boundary between the RA and the inferior caval vein (IVC). In the left atrium, the extra-pericardial segments of the pulmonary veins were carefully excluded.

In order to minimize interobserver variability, all contours were traced by one observer (CW) and carefully reevaluated and discussed with a second experienced investigator (EVB) for reaching consensus reading. Three-dimensional (3D) volumes over time were obtained by adding all the atrial areas of all slices for all 40 cardiac phases by the summation disk method. (Fig. 1b).

Maximal volume (Vol max) at end of systole, minimal and maximal volumes at mid-diastole (Vol min-diast and Vol max-diast), and minimal volume (Vol min) at the end of diastole were measured (Fig. 2). The functional parameters of both atria were calculated as shown in Table 1 and Fig. 2. Conduit volume was calculated in the patient group by subtracting cyclic volume change from the left ventricular stroke volume [1, 10]. As the RV stroke volume was increased due to the regurgitant volume from pulmonary regurgitation, we used LV stroke volume for calculation of RA conduit volume. Regurgitant volume from pulmonary valve insufficiency was used as internal validation for differences between RV and LV stroke volumes.

Ventricles

The endsystolic and the enddiastolic phase were first identified visually on a movie loop of a midventricular slice. The endocardial contours were traced manually, and stroke volume and ejection fraction were then calculated as previously described [4].

Flow Profiles

Blood flow across MV and TV was analyzed semi-quantitatively for flow profile and maximal velocity of the atrial passive emptying (*E* wave) and of the atrial active emptying (*A* wave). An altered *E*/*A* profile with *A* wave higher than *E* wave was considered as indicative for diastolic dysfunction.

The flow profile in the main pulmonary artery was also verified for presence or absence of a forward flow during late diastole as an additional sign for diastolic dysfunction.

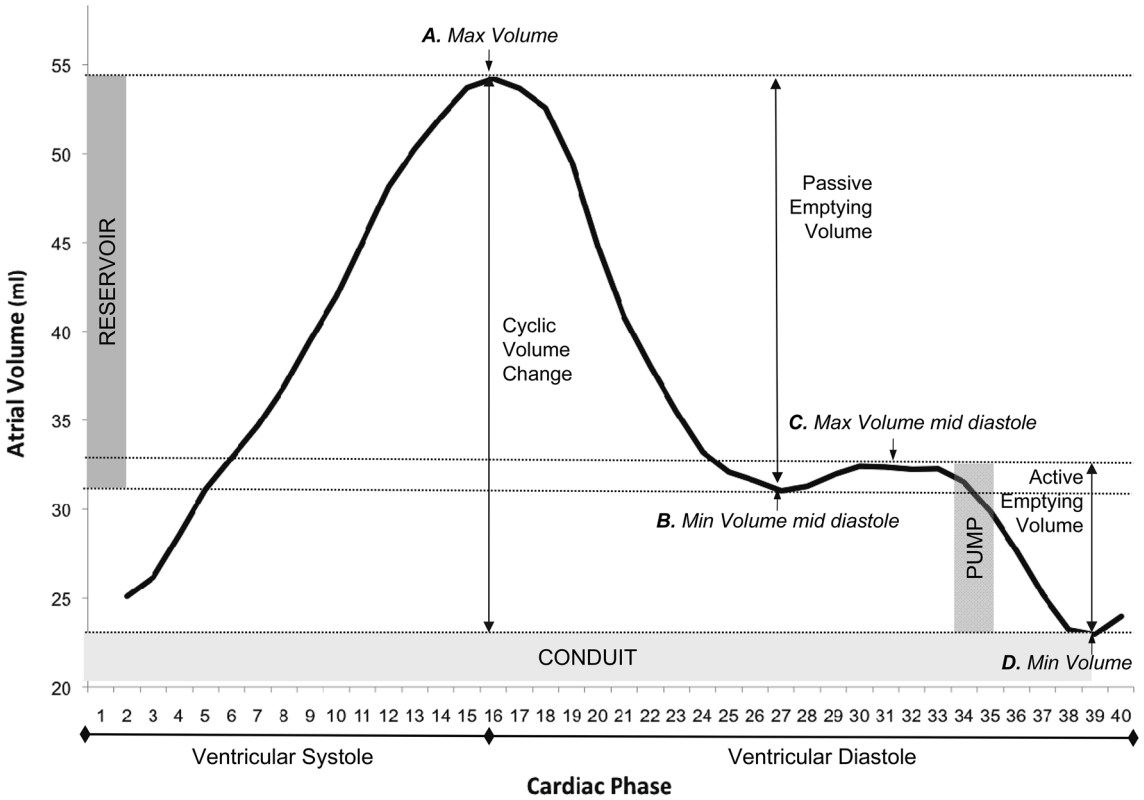

Fig. 2 Time/volume curve showing atrial function and its 3 components (reservoir, conduit, and pump function) in one sample normal subject. *A* maximal volume; *B* minimal volume during mid-diastole; *C* maximal volume during mid-diastole; *D* minimal volume

Table 1 Functional parameters calculated from the volumes/time curve of the atria (Fig. 2)

Functional parameter	Calculation
Cyclic volume change (total empting volume)	Volume max − volume min (A − D)
Total empting fraction	Total emptying volume/volume max
Passive empting volume	Volume max − volume max diast (A − B)
Passive empting fraction	Passive emptying volume/volume max
Active empting volume	Volume max diast − volume min (C − D)
Active empting fraction	Active emptying volume/volume max diast
Conduit volume	Ventricular stroke volume − cyclic volume change

Statistics

Continuous data are expressed as mean ± SD or median (range) as appropriate. All data are indexed per body surface area (BSA). Distribution was analyzed with the D'Agostino and Pearson omnibus normality test. All parameters were normally distributed. Therefore, data of different groups were compared by using an unpaired *t* test. Statistical significance was defined as $p < 0.05$. Statistics were performed using the software Prism® 4.03 (GraphPad Software, Inc., California, USA). The study was approved by the Ethics Research Board of our Institution.

Results

Twelve patients after TOF repair (9 women, 3 men) were included in the study. Two patients were excluded due to more than trivial tricuspid valve regurgitation in one and a history of atrial fibrillation in the other one. Patient characteristics are shown in Table 1. Median age at surgical repair was 1.9 (0–11) years; median time interval between surgical repair and CMR examination was 14.6 (9–23) years. TOF was repaired with a transannular patch in 8 patients. Two patients had undergone redo-surgery with pulmonary valve replacement because of severe pulmonary regurgitation at the age of 8 and 9 years, respectively. Mean time interval between redo-surgery and CMR examination was 6 years. Clinical indication for CMR consisted of quantification of pulmonary regurgitation in 11 patients, a pulmonary stenosis in 2, and an isolated stenosis of the left pulmonary artery in one.

Table 2 Characteristics of both study groups

	Patients ($n = 12$)	Controls ($n = 10$)	
Women (%)	75	70	n.s.
Age (years)	18.8 ± 6.8	16.7 ± 6.1	n.s.
Weight (kg)	50.9 ± 14.9	57.3 ± 20.8	n.s.
Height (cm)	155 ± 14.3	164.0 ± 15.6	n.s.
BSA (m^2)	1.47 ± 0.27	1.60 ± 0.36	n.s.
Heart rate	77 ± 18	76 ± 12	n.s.
RR syst	109 ± 13	111 ± 12	n.s.
RR diast	69 ± 12	67 ± 9	n.s.

BSA body surface area, *RR* blood pressure

Table 3 Right atrial size and function in TOF patients compared to healthy volunteers

	TOF patients $N = 12$	Controls $N = 10$
Volume max	50.7 ± 5.7	51.2 ± 7.7
Volume min	27.4 ± 4.9**	20.9 ± 5.8**
Volume min mid-diastole	35.9 ± 6.1*	29.9 ± 6.6*
Volume max mid-diastole	38.8 ± 6.7*	31.6 ± 5.5*
Cyclic volume change	23.3 ± 5.9*	30.3 ± 7.1*
Output (mL/min/BSA)	1,732 ± 708	2,152 ± 664
Total atrial filling fraction (%)	45.6 ± 9.3**	59.0 ± 9.6**
Passive emptying volume	11.9 ± 6.1*	19.6 ± 6.6*
Passive emptying fraction (%)	23.1 ± 12.1**	37.8 ± 10.2**
Active emptying volume	11.4 ± 4.1	10.7 ± 3.5
Active emptying fraction (%)	28.9 ± 8.2	34.3 ± 11.5
Conduit volume	24.6 ± 9.9	n.a.

Volumes expressed in mL/BSA

BSA body surface area

* $p < 0.05$; ** $p < 0.01$

Patients and controls did not differ significantly regarding demographic data or vital parameters (Table 2).

Atria

Atrial measurements and functional parameters are shown in Table 3.

In TOF patients, the RA showed an increased minimal volume, with a decreased total atrial emptying fraction and cyclic volume change. Similarly, the minimal and maximal volumes during mean diastole were significantly increased, and the passive emptying volume and passive emptying fraction were reduced. No significant difference was observed for the active emptying fraction. The volume/time curve of RA changes is shown in Fig. 3. The diminished volume changes of the right atrium during the cardiac cycle are expressed by the narrow amplitude of the curve.

No difference was found for LA size and function between TOF patients and the normal group, except for a diminished passive emptying fraction (Table 4).

Ventricles

All TOF patients presented enlarged RV volumes [26, 27]. An EF >50 % was found in only 3 of 12 patients, an EF of 45–50 % in 8, and an EF of 40–45 % in one. RV end-diastolic volume (EDV) was 135 ± 27.7 mL/m^2, RV end-systolic volume (ESV) 67.9 ± 18.2 mL/m^2, RV stroke volume 67.1 ± 18.2 mL/m^2, and RV ejection fraction (EF) 50 ± 6.6 %.

LV size and systolic function were within normal limits [4]. LV end-diastolic volume was 81.8 ± 11.9 mL/m^2 (EDV), LV end-systolic volume 33.9 ± 7.2 mL/m^2 (ESV), LV stroke volume 47.8 ± 7.8 mL/m^2, and LV EF 58.6 ± 5.5 %, respectively. Two of 12 patients had an EF <55 %.

Atrioventricular Interaction

Four patients (30 %) had a reversed *E/A* ratio of the blood flow across the tricuspid valve indicating right ventricular diastolic dysfunction (Fig. 4). In the other 8 patients, there was no abnormal late diastolic flow in the main pulmonary artery, and therefore pseudonormalization of the inflow profile due to advanced diastolic dysfunction was excluded. In the patients with a reversed *E/A* ratio, RA parameters showed a trend toward a lower passive emptying volume and fraction and a higher active emptying function, respectively (Table 5). An elevated conduit volume was the only significantly altered parameter.

Discussion

Right ventricular dilatation and dysfunction are well known sequelae during long-term follow-up after TOF repair when significant pulmonary valve regurgitation occurs. However, the RV cannot be considered an isolated entity, and the atria play an important role in global cardiac performance [24]. This prospective study demonstrates that

1. Patients after TOF repair have an abnormal RA function.
2. The RA presents with decreased volume changes during the cardiac cycle, with a globally diminished emptying capacity, i.e., an enlarged minimal RA volume.
3. The conduit/reservoir ratio is increased as a result of a decreased passive emptying volume.
4. The LA function is normal.

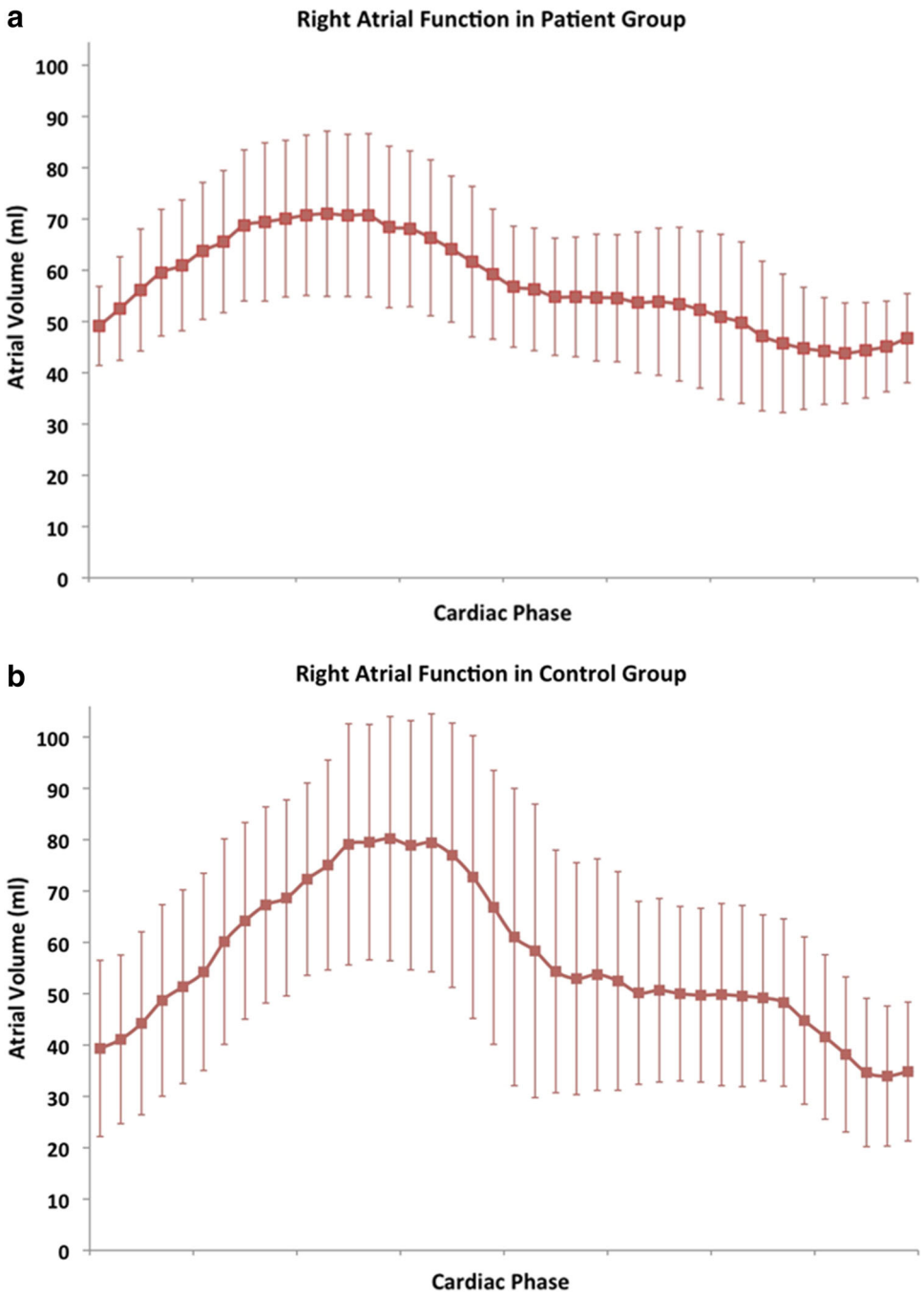

Fig. 3 Time volume curve of the right atrium in TOF patients (**a**) and in healthy controls (**b**)

The sum of the findings observed in our study documents a loss of the RA capacity to convert continuous venous return into a pulsatile ventricular filling, and therefore a loss of energetic efficiency in the atrioventricular interaction [20]. The main finding of a diminished reservoir function of the RA in patients after TOF repair is in agreement with the results published by Riesenkampff et al. [18, 19] and by Luijnenburg et al. [13].

Atrial filling and passive atrial emptying during early atrial diastole and ventricular systole, respectively, are represented by the cyclic volume changes and passive emptying volume and are both measures of the reservoir function of the atria (Fig. 2) [1, 9]. Atrial filling accounts for the largest increase in atrial volume and is determined by the relaxation and compliance of the atrial wall which recoils from the preceding atrial contraction. In a second phase, filling is modulated also by the descent of the cardiac base "sucking" blood into the atrium and by ventricular contraction "pushing" blood into the atrium by pulse wave propagation through the lungs [20]. In patients

Table 4 Left atrial size and function in TOF patients compared to healthy volunteers

	TOF patients	Controls
Volume max	33.3 ± 10.2	35.0 ± 7.7
Volume min	14.1 ± 3.4	13.9 ± 3.6
Volume min mid-diastole	20.0 ± 5.8	18.4 ± 5.0
Volume max mid-diastole	21.9 ± 6.2	20.8 ± 5.7
Cyclic volume change	19.2 ± 8.0	21.1 ± 5.3
Output (mL/min/BSA)	1,386 ± 587	1,494 ± 462
Total atrial filling fraction (%)	56.4 ± 9.4	60.0 ± 7.0
Passive emptying volume	11.4 ± 4.1	14.2 ± 3.5
Passive emptying fraction (%)	33.5 ± 9.0*	40.9 ± 7.4*
Active emptying volume	7.8 ± 3.6	6.8 ± 3.0
Active emptying fraction (%)	34.7 ± 8.8	32.2 ± 8.8
Conduit volume	28.6 ± 6.4	n.a.

Volumes in mL/BSA

BSA body surface area

* $p < 0.05$

after TOF repair, we have found a significant increase of the global minimal volume and of the minimal volume at mean diastole, both expressing an insufficient early diastolic and eventually global emptying of the atrium. This reflects an impaired reservoir function of the RA. This finding correlates well with the data reported by Riesenkampff et al. [18, 19], who described the same alterations of atrial function. In addition, they reported a correlation with decreased values of TAPSE and RV strain rate [18], data suggesting that the longitudinal systolic displacement of the tricuspid valve is a relevant extrinsic factor influencing the reservoir function of the RA, similarly as previously described for the LA [25]. A decreased longitudinal deformation of the RV in TOF patients compared to patients with an atrial septal defect has also been demonstrated in another speckle tracking study by Dragulescu et al. [5].

If the RA reservoir function during rapid emptying of the ventricle in early systole is reduced, a higher conduit flow, passing the RA without changing its volume, is necessary to fill the right ventricle adequately during diastole. This compensatory mechanism with an increased conduit/reservoir ratio has been found by other authors and is now also being confirmed by our data [18, 19]. In our cohort, the conduit volume was even more increased in patients with abnormal tricuspid inflow pattern than in patients without diastolic dysfunction.

In a sheep model, Gaynor et al. [6] showed that in acute physiological changes, such as in acute pressure overload, the healthy RA adapts and acts more as a reservoir than as a conduit; such increase in reservoir function results in a favorable low conduit/reservoir ratio and contributes to increase the cardiac output. In contrast, an elevated conduit/reservoir ratio has been associated with reduced cardiac output [6]. The negative impact on cardiac output may be even more accentuated in chronic disease states that impair RV mechanics, such as in chronic RV volume overload present in TOF patients.

The contractile function of the atria is influenced by their intrinsic mechanics and by the diastolic compliance of the connecting ventricle. One might expect that the RA active pump function is increased as an additional compensatory mechanism if the reservoir function is decreased, such as in TOF patients. However, data in the literature are not conclusive; while some studies described an increase in active emptying in patients after TOF repair [8], others found a decreased RA pump function in volume-loaded ventricles [18]. In our limited number of patients, we observed a trend toward a decreased active atrial emptying fraction in the overall group, similar to Riesenkampff et al. [18], but a trend toward an increased active atrial emptying in the subgroup of patients with restrictive RV physiology, similar to Luijnenburg et al. [13] in the patient group with end-diastolic forward flow in the main pulmonary artery. Thus, a stiff RV has a major influence on the different phases of RA function.

There are several potential reasons for the described changes in RA function. The concomitant flow from pulmonary regurgitation is considered an additional factor influencing RA function in TOF patients [13, 18]. Our data show that the total RA filling is normal, but early diastolic RA passive emptying is decreased; the subsequent conduit flow during mid-diastole is then increased. During early diastole, the regurgitant flow from pulmonary regurgitation is at its maximum and fills the RV retrogradely competing against the antegrade filling flow from the RA. Once the flow from the outflow tract decreases, the conduit flow from the RA can complete passive RV filling. This again results in an elevated conduit/reservoir ratio and in partial loss of pulsatile ventricular filling. However, the hydrodynamic effect of pulmonary regurgitation alone does not seem to be the only cause for the observed RA changes in TOF patients. In fact, Riesenkampff et al. [19] compared TOF patients with other patients with a significant pulmonary regurgitation after balloon valvuloplasty, i.e., without history of surgery, and found parameters of atrial function close to normal in the latter group. By analyzing the total heart volume and its variations during the cardiac cycle, they documented a clear correlation between pericardectomy and an elevated total heart volume variation. Thus, additional potential causes for RA dysfunction related to surgery, including lack of an intact pericardium after pericardectomy and atrial scars after atriotomy are probable additional causes of RA dysfunction after TOF repair.

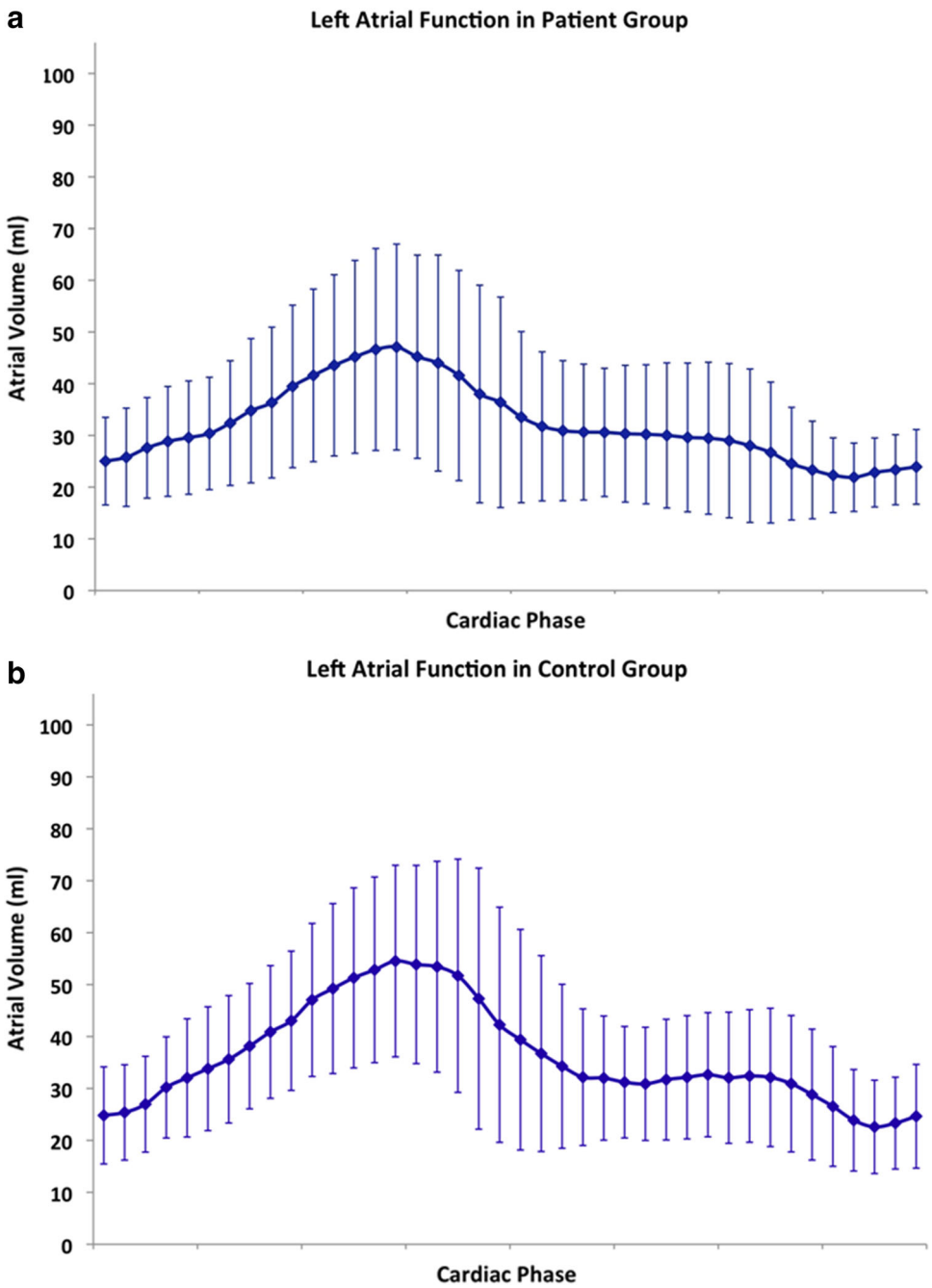

Fig. 4 Time volume curve of the left atrium in TOF patients (**a**) and in healthy controls (**b**)

Segmentation of the atria on short-axis or axial images is quite challenging due to the complex boundaries and the orifices of the systemic and the pulmonary veins. Therefore, we analyzed the images in consensus reading in order to improve accuracy and minimize interobserver variability. The tight standard deviations of our measurements document the precision of our results. Our data correlate fairly well with those published by Sievers et al. [22] for the RA. The same group demonstrated a lower variability of the measurements if they are performed in a short-axis view.

Limitations

The small number of subjects assessed is the main limitation of this study. This was our first experience in CMR assessment of atrial function in a specific congenital heart disease, and we consider the data reported as a pilot study. In spite of the small sample size, we have been able to show consistent specific and significant changes of atrial function and some trends. Our data are supported by and are similar to other observations reported recently [13, 18, 19]. Indeed, trends and interpretation of the complex

Table 5 Right atrial function in normal and abnormal RV diastolic function

	Tricuspid valve inflow	
	Preserved *E*/*A* ratio (>1)	Reversed *E*/*A* ratio (<1)
Volume max	51.8 ± 4.9	50.3 ± 19.3
Volume min	27.5 ± 3.9	27.3 ± 9.2
Volume min mid-diastole	35.1 ± 6.6	37.7 ± 10.5
Volume max mid-diastole	38.2 ± 7.5	40.5 ± 12.7
Cyclic volume change	24.31 ± 4.6	22.95 ± 14.08
Output (mL/min/BSA)	1,871 ± 774	1,563 ± 903
Total atrial filling fraction (%)	46.8 ± 6.9	43.1 ± 14.0
Passive emptying volume	13.6 ± 5.1	9.8 ± 8.7
Passive emptying fraction (%)	26.5 ± 10.2	16.3 ± 14.1
Active emptying volume	10.7 ± 4.7	13.2 ± 6.2
Active emptying fraction (%)	27.2 ± 7.9	32.5 ± 8.7
Conduit volume	20.4 ± 8.6*	32.1 ± 8.2*

Volumes in mL/BSA

BSA body surface area

* $p < 0.05$

pathophysiologic atrial mechanisms have to be confirmed in larger studies. Our preliminary data do not allow drawing conclusions on the clinical impact or outcome of the patients. In a recent paper, Luijnenburg et al. [13] correlated RA functional parameters with exercise capacity, ECG parameters, and biomarkers and found a relevant association between RA function and parameters of clinical condition. In this study, abnormal RA emptying was associated with longer QRS duration, higher NT-proBNP, lower peak workload, and peak VO_2. Specifically designed future prospective studies should add evidence to these preliminary results.

Conclusion

Right atrial function is significantly abnormal in patients after TOF repair and with pulmonary regurgitation. The reservoir function is diminished, which results in decreased early diastolic ventricular filling. As a compensatory mechanism, the conduit/reservoir ratio is increased, and the RA loses its capacity to convert continuous venous flow into an intermittent ventricular filling. These changes may be even more pronounced in the presence of right ventricular diastolic dysfunction.

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