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Diastolic ventricular interaction in patients after atrial switch for transposition of the great arteries: A speckle tracking echocardiographic study

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ABSTRACT

Background: We tested the hypothesis that diastolic ventricular interaction occurs after atrial switch operation for transposition of the great arteries (TGA) and that subpulmonary LV diastolic function is influenced by septal geometry.

Methods: Twenty-nine patients (male 19) after atrial switch operation for TGA aged 20.8 ± 4.1 years and 27 healthy controls were studied. Two-dimensional longitudinal systolic strain, systolic (SRs), early diastolic (SRe), and late diastolic (SRa) strain rates of both ventricles were determined using speckle tracking echocardiography. Early diastolic trans-atrioventricular velocity (E) and myocardial early diastolic myocardial velocity (e) at the ventricular free wall–annular junction were measured. Geometry of the morphologic left ventricle was quantified by the diastolic eccentricity index (EI).

Results: In both systemic and subpulmonary ventricles, SRe and SRa were significantly lower and trans-atrioventricular E/e ratios higher in patients than controls (all $p<0.001$). In patients, RV SRe correlated with left ventricular (LV) SRe ($r=0.49$, $p=0.008$), and RV SRa correlated with LV SRa ($r=0.46$, $p=0.01$). Significant leftward shifting of the septum in patients was reflected by the greater LV EI ($p<0.001$). In patients, LV EI correlated with age- and sex-adjusted z score of LV end-diastolic volume. As a group, LV EI correlated negatively with LV SRe ($r=-0.62$, $p<0.001$) and LV SRa ($r=-0.51$, $p<0.001$), and positively with mitral E/e ratio ($r=0.33$, $p=0.02$).

Conclusions: Systemic RV diastolic dysfunction occurs after atrial switch operation and correlates with subpulmonary LV diastolic dysfunction. The observed diastolic ventricular interaction may potentially be mediated through alteration of septal geometry.

1. Introduction

While systolic dysfunction of the systemic right ventricle is well documented in patients long term after atrial switch operation for complete transposition of the great arteries (TGA) [1,2], data on systemic right ventricular (RV) diastolic function are limited [3]. In animal models of chronic subpulmonary RV pressure overload, prolonged relaxation and increased stiffness of the right ventricle have been demonstrated [4,5]. In patients with pulmonary hypertension, the magnitude of subpulmonary RV diastolic dysfunction correlates with the extent of RV afterload [6]. The impact of chronic increase in afterload on global systemic RV diastolic function in late survivors of atrial switch operation, which has implications on systemic RV preload and hence cardiac output, has nonetheless not been systemically evaluated.

Diastolic ventricular interaction has been implicated in RV pressure and volume overloading conditions. In patients with pulmonary arterial hypertension [7] and in those with pulmonary regurgitation after repair of tetralogy of Fallot [8], shifting of the ventricular septum has been incriminated in the pathogenesis of LV diastolic dysfunction. Septal geometry is also altered after Senning and Mustard operation [9]. However, whether such diastolic ventricular interaction is operating in patients after atrial switch operation, which has implications on systemic RV preload and hence cardiac output, has hitherto not been explored.

Speckle tracking echocardiography (STE) has changed the paradigm in the evaluation of ventricular diastolic function. It offers advantages over tissue Doppler techniques given its angle independence and its ability to interrogate directly diastolic myocardial deformation [10]. In this study, we used STE to test the hypotheses that diastolic ventricular interaction occurs after atrial switch operation for TGA and that subpulmonary LV diastolic function is influenced by septal geometry.

2. Methods

Twenty-nine patients (19 males), aged 20.8 ± 4.1 years, who had undergone atrial switch operation for complete TGA were studied. Those not in sinus rhythm and those with sick sinus syndrome were excluded. Data collection from case notes included the

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following: associated ventricular septal defect (VSD) and other intracardiac abnormalities, age at and type of reparative surgery, duration of follow-up since operation, history of cardiac arrhythmias, and current cardiac medications. Twenty-seven controls (16 males), aged 18.9 ± 3.6 years ($p = 0.06$), were studied. The Institutional Review Board approved the study and all of the subjects gave informed consent.

Transthoracic echocardiography was performed using the Vivid 7 ultrasound system (General Electric, Vingmed, Horten, Norway). Measurements of 2-dimensional images were made in 3 cardiac cycles, and the average was used for statistical analyses. All echocardiographic recordings were stored in digital versatile disc for offline analyses using EchoPAC software (General Electric, Vingmed, Horten, Norway).

From the parasternal short axis view, the maximum distance from the endocardial surface of the mid-ventricular septum to that of the postero-lateral LV free wall (D1) and the distance of the orthogonal axis between the endocardial surfaces of the anterior and inferior LV free walls (D2) were measured at end-diastole. The left ventricular diastolic eccentricity index (EI) was calculated as D2/D1 [11]. The greater the EI, the more compressed is the left ventricle.

Conventional pulsed-Doppler assessment of the mitral and tricuspid inflow was performed to obtain peak early (E) and late (A) diastolic velocities and E wave deceleration time. The severity of tricuspid regurgitation was graded semi-quantitatively by colour Doppler echocardiography as the ratio of tricuspid regurgitation jet area to the right atrial area [12].

Pulsed-wave tissue Doppler imaging of the apical four-chamber view was performed with the sample volume positioned at the RV free wall-tricuspid annular and LV free wall–mitral annular junctions. The tricuspid and mitral annular peak myocardial velocities during early diastole (e), late diastole (a) and systole (s) were measured and the respective tricuspid and mitral E/e ratios were calculated accordingly.

Colour tissue Doppler imaging data were also recorded from the apical 4-chamber view. The myocardial velocities were measured at the base of the RV and LV free walls below the insertion of the atrioventricular valve leaflets. The myocardial acceleration during isovolumic contraction (IVA) was calculated as the difference between peak and baseline myocardial velocities divided by the time interval from onset of the wave during isovolumic contraction at zero-crossing to the time at peak velocity [13].

From the apical four-chamber view, 2-dimensional images were acquired for offline analysis using the 2-dimensional strain software (EchoPAC, General Electric Vingmed Ultrasound, Horten, Norway) as described previously [14]. The entire endocardial border of the respective ventricle was manually traced to determine the global longitudinal strain and strain rates. The global longitudinal strain, global systolic strain rate (SRs), and global early (SRe) and late diastolic (SRa) strain rates were then calculated. Our group has previously reported on the intra- and inter-observer reproducibilities of the speckle tracking technique [14].

All of the patients underwent cardiac magnetic resonance (CMR) imaging for assessment of ventricular diastolic and systolic volumes, stroke volume, and ejection fraction. The examination was performed using a 1.5T superconducting whole-body imager (GE Signa Horizon Echospeed; GE Medical Systems, Milwaukee, Wisconsin) with a phased-array body coil. Data on RV and LV function were obtained by fastcard spoiled gradient recalled cine on axial and short axis planes, respectively, and analyzed using the software Advantage Window version 4.2. As the controls did not undergo CMR imaging, the LV diastolic and systolic volumes and stroke volumes of patients were normalized for age and sex according to reported normal values [15] and z scores were calculated accordingly.

Data are presented as mean ± SD. Absolute values of strain and strain rates are presented. Differences in demographic and echocardiographic variables between patients and controls were compared using unpaired Student's *t*-test and Fisher's exact test where appropriate. As a whole group, Pearson correlation analysis was used to assess the relations between LV EI and LV diastolic SRs. Within the patient cohort, the relations between diastolic strain rates of the systemic right ventricle and those of the subpulmonary left ventricle were assessed using Pearson correlation analysis. Relations between z scores of CMR-derived LV volumes and echocardiographic variables were also determined using Pearson correlation analysis. A p value < 0.05 was considered statistically significant. All statistical analyses were performed using SPSS 11.0 (SPSS, Inc., Chicago, Illinois).

3. Results

3.1. Subjects

The 29 patients had atrial switch operation, 23 undergoing Senning procedure and 6 having Mustard procedure, at an age of 1.2 ± 1.3 years. They were followed up for 19.6 ± 3.7 years. Six patients required surgical closure of an associated VSD. Twenty-five patients were in the New York Heart Association functional class I and 4 in class II. One patient received digoxin, 1 enalapril, and 1 digoxin and losartan.

3.2. Diastolic parameters

Table 1 summarizes the conventional Doppler, tissue Doppler and speckle tracking parameters in patients and controls.

For the systemic ventricular diastolic inflow velocities, the E velocity was similar ($p = 0.22$) between patients and controls, while the A velocity was significantly lower ($p = 0.02$) and E/A ratio significantly greater ($p = 0.05$) in patients than controls. As for the subpulmonary ventricular diastolic inflow velocities, both the E ($p < 0.001$) and A ($p = 0.02$) velocities were greater in patients than controls, although the difference in E/A ratio did not reach statistical significance. The deceleration time of early diastolic flow across the atrioventricular valve of the subpulmonary ventricle was significantly shorter in patients than that of controls ($p < 0.001$).

The early and late diastolic myocardial velocities at the level of the free wall–annular junctions of both systemic and subpulmonary ventricles were significantly lower in patients compared with controls (all $p < 0.001$). On the other hand, systemic and subpulmonary ventricular E/e ratios were significantly higher in patients than controls (both $p < 0.001$).

The SRe and SRa of the systemic ventricle were significantly lower in patients than controls (both $p < 0.001$). Likewise, both SRe and SRa of the subpulmonary ventricle were significantly lower in patients

Table 1
Comparison of echocardiographic parameters between patients and controls.

	Systemic ventricle			Subpulmonary ventricle		
	Patients ($n = 29$)	Controls ($n = 27$)	p	Patients ($n = 29$)	Controls ($n = 27$)	p
Conventional Doppler study						
E (cm/s)	82.9 ± 17.2	82.3 ± 14.5	0.22	79.0 ± 16.6	54.6 ± 11.8	<0.001
A (cm/s)	35.0 ± 11.6	42.5 ± 11.0	0.02	31.6 ± 8.9	26.2 ± 7.2	0.02
E DT (ms)	171 ± 50	171 ± 29	1.00	139 ± 46	191 ± 48	<0.001
E/A ratio	2.64 ± 1.04	2.18 ± 0.60	0.05	2.67 ± 1.05	2.25 ± 0.62	0.08
Tissue Doppler imaging						
e (cm/s)	8.9 ± 2.6	18.1 ± 2.9	<0.001	12.7 ± 3.2	17.7 ± 2.7	<0.001
a (cm/s)	4.6 ± 1.3	7.1 ± 1.5	<0.001	4.4 ± 1.5	9.5 ± 2.0	<0.001
s (cm/s)	7.3 ± 2.0	12.4 ± 2.6	<0.001	8.8 ± 2.5	13.6 ± 1.7	<0.001
e/a ratio	2.1 ± 1.0	2.7 ± 0.8	0.02	3.2 ± 1.2	2.0 ± 0.6	<0.001
E/e ratio	10.4 ± 4.7	5.0 ± 1.0	< 0.001	7.0 ± 3.5	3.2 ± 0.7	<0.001
Speckle tracking echocardiography						
SRe (/s)	1.16 ± 0.24	1.99 ± 0.36	<0.001	1.27 ± 0.37	1.84 ± 0.39	<0.001
SRa (/s)	0.47 ± 0.18	0.81 ± 0.17	<0.001	0.50 ± 0.25	0.97 ± 0.26	<0.001
SRs (/s)	0.76 ± 0.16	1.19 ± 0.20	<0.001	1.09 ± 0.19	1.40 ± 0.29	<0.001
Strain (%)	15.9 ± 2.2	21.6 ± 2.1	<0.001	19.7 ± 3.3	26.6 ± 3.6	<0.001

A, late diastolic atrioventricular inflow velocity, a, late diastolic myocardial velocity of the atrioventricular annular-free wall junction, DT, deceleration time, E, early diastolic atrioventricular inflow velocity, e, late diastolic myocardial velocity of the atrioventricular annular-free wall junction, s, systolic myocardial velocity of the atrioventricular annular-free wall junction, SRa, global late diastolic strain rate, SRe, global early diastolic strain rate, SRs, global systolic strain rate.