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ORIGINAL SCIENTIFIC PAPER

15-Year follow-up of regional right and left ventricular function after the Senning operation: a Colour-Doppler myocardial imaging study

Ana Moya[a], Pieter De Meester[b,c], Els Troost[b], Leen Roggen[b], Frank Weidemann[d], Philip Moons[e], Benedicte Eyskens[f], Piet Claus[c], Werner Budts[b,c] and Alexander Van De Bruaene[b,c]

[a]Faculty of Biomedical Sciences, KU Leuven, Leuven, Belgium; [b]Division of Structural and Congenital Cardiology, University Hospitals Leuven, Leuven, Belgium; [c]Department of Cardiovascular Sciences, KU Leuven, Leuven, Belgium; [d]Medical Clinic I, Klinikum Vest, Recklinghausen, Germany; [e]Department of Public Health and Primary Care, KU Leuven, Leuven, Belgium; [f]Division of Pediatric Cardiology, University Hospitals Leuven, Leuven, Belgium

ABSTRACT

Introduction: Despite right ventricular (RV) dysfunction being a major concern in Senning patients, long-term follow-up data is lacking. This study aimed (1) at evaluating regional (base-mid-apex) RV and left ventricular (LV) function using Colour-Doppler myocardial imaging over a 15-year follow-up period and (2) at comparing results with matched controls.

Methods: For the longitudinal analysis (2004–2019), we compared systolic and diastolic function in 10 Senning patients. For the cross-sectional analysis, we compared the subaortic RV (sRV) of Senning patients with the RV and LV of matched controls and the subpulmonary LV (spLV) of Senning patients with the LV of matched controls.

Results: The longitudinal analysis of sRV function showed a significant decrease in apical peak systolic strain ($-17 \pm 7\%$ vs $-12 \pm 4\%$; $p = 0.025$) and apical peak systolic strain rate ($-1.1 \pm 0.3 s^{-1}$ vs $-0.8 \pm 0.4 s^{-1}$; $p = 0.012$). spLV function showed a significant decrease in peak systolic velocity (mid; $p = 0.013$ and apex; $p = 0.011$) and peak systolic strain rate (mid; $p = 0.048$). The cross-sectional analysis revealed significant lower values for basal, mid and apical peak systolic velocity, peak systolic strain rate, peak systolic strain of the sRV of Senning patients when compared to both LV and RV of matched controls (all $p < 0.05$).

Conclusion: Our study showed that systolic and diastolic sRV function did not change over a 15-year follow-up period, except in the apical region. There was a decline in spLV systolic function, which may be of clinical value. On the other hand, when compared to age- and gender-matched controls, the sRV of Senning patients exhibits significantly decreased measurements of longitudinal systolic function.

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Transposition of great vessels; atrial switch procedure; senning procedure; systemic right ventricle; right ventricular dysfunction; heart failure

Introduction

Transposition of the great arteries (TGA) represents approximately 5–7% of all congenital cardiac malformations with an incidence estimated at 1 case per 3500–5000 live births and a 2:1 male predominance [1–3]. The Senning operation, or atrial switch repair, corrects blood flow at the atrial level, resulting in a biventricular physiology in which a subaortic right ventricle (sRV) supports the systemic circulation (2 V-RV). Since the atrial switch procedure has largely been replaced by the arterial switch procedure, most Senning patients have now reached adulthood [4].

Among a myriad of long-term complications, such as arrhythmias, sudden cardiac death and tricuspid valve insufficiency, patients with a systemic RV have a high likelihood to develop sRV dysfunction and heart failure (HF) earlier in life [5–7]. Facing the increased afterload of the systemic circulation, the sRV becomes hypertrophied and dilated, while atrial baffles limit preload to the sRV [8].

From a clinician's perspective, distinguishing physiologic adaptation to increased afterload [9] from pathologic remodelling using imaging modalities remains challenging. Still, early identification of sRV dysfunction is needed to identify patients who have worse prognosis [10,11] and may benefit from referral to a dedicated adult congenital heart disease (ACHD)-HF clinic [7]. This is especially important since decline is rapid when clinical HF is present [7]. Measures of sRV function are often 'abnormal' when compared to

CONTACT Alexander Van De Bruaene alexander.vandebruaene@uzleuven.be Division of Structural and Congenital Cardiology, University Hospitals Leuven, Herestraat 49, Leuven, 3000, Belgium

a healthy control group, stressing the importance of longitudinal follow-up data [12].

In 2004, Eyskens et al. [13] were among the first to evaluate deformation imaging in a group of children who had undergone a Senning procedure in childhood. Our study now aimed (1) at evaluating regional (base-mid-apex) RV and left ventricular (LV) function using Colour-Doppler myocardial imaging over a 15-year follow-up period (longitudinal analysis) and (2) at comparing results with age- and gender-matched controls (cross-sectional analysis).

Methods

Study population

Patients included in the study of Eyskens et al. in 2004, who underwent a Senning operation during childhood, were contacted and included along with 20 age-matched volunteers (11 men and 9 women, mean age 30.3 ± 4.4 years). The study was approved by the institutional review board (University Hospitals Leuven, Belgium) and informed consent was obtained in all patients

Transthoracic echography

Standard greyscale, Doppler and 2-dimensional Colour-Doppler myocardial velocity imaging examinations were performed using a Vivid E9 and E95 ultrasound system (General Electric Vingmed Ultrasound, Horten, Norway) equipped with a 3-MHz transducer and Tissue Doppler Imaging (TDI) technology. Echocardiographic studies were performed with the participant in the left lateral decubitus position. Images were stored digitally for off-line analysis using dedicated software (EchoPac, GE Vingmed Ultrasound, Horten, Norway).

Standard greyscale and Doppler echocardiographic variables

At least 3 consecutive heartbeats taken at a frame rate of 65–85 Hz were analysed with values presented as mean values. In an apical 4-chamber view, RV area at end-diastole and end-systole, fractional area change (FAC), tricuspid annular systolic excursion (TAPSE) from M-mode recordings and LV ejection fraction were obtained. Mitral and tricuspid Doppler inflow patterns were acquired to measure early (E) and late (A) diastolic waves. Early diastolic mitral and tricuspid annular velocity (e') were measured and E/e' ratio was calculated.

2-dimensional myocardial velocity imaging variables

Colour-Doppler myocardial velocity imaging data were acquired from the RV and LV free wall in an apical 4-chamber view with a frame rate of at least 100 Hz . Longitudinal velocities, strain and strain rate were analysed in the base, mid and apical segment of the RV and LV free wall, as has been previously described [13]. Three regions of interest (ROIs) of 10 mm length were positioned in the basal, mid and apical segment and manually tracked throughout the cardiac cycle in order to stay within the same part of the myocardium. Peak systolic values were calculated as peak negative strain and strain rate between aortic or pulmonary valve opening and closure, as appropriate, with baseline strain set at zero at aortic or pulmonary valve opening. All measurements were averaged over 3 consecutive beats. Linear drift compensation was applied. Timing information was measured from aorta/pulmonary and mitral/tricuspid valve Doppler traces during the same examination with a similar R-R interval (Figure 1).

Statistical analysis

Data were analysed using SPSS for Windows Version 25 (SPSS, IBM headquarters, Armonk, New York, US). Normal distribution for continuous variables was investigated and data reported as mean with standard deviation (±SD) or median with interquartile range (IQR) where appropriate. Descriptive data for discrete variables were reported as proportions with numbers and percentages (%). Mann–Whitney and Wilcoxon Signed Rank test were performed to compare unrelated and related samples respectively. All *t*-tests were two-sided and $p < 0.05$ was considered statistically significant. We previously reported adequate inter- and intra-observer variability for Colour-Doppler myocardial imaging analysis [13,14].

Results

Study population

Out of 20 patients initially included, 10 could be recruited for the follow-up study between January 2018 and April 2019. Ten patients were either lost-to-follow-up ($n = 3$), declined to participate ($n = 4$), moved abroad ($n = 1$), were pregnant ($n = 1$) or were transplanted ($n = 1$). The clinical and echocardiographic data of the TGA-Senning patients and healthy volunteers are summarised in Table 1. In 2004, there

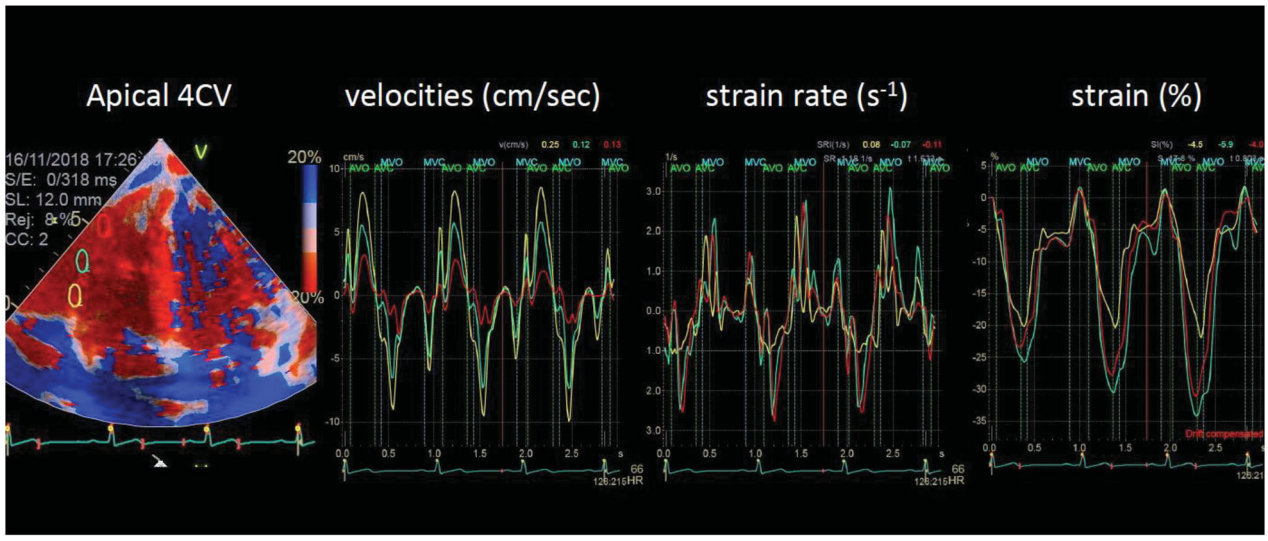

Figure 1. Representative apical 4-chamber view with velocity, strain rate and strain curves in the basal (yellow circle and line), mid (green circle and line) and apical (red circle and line) segment.

Table 1. Clinical, grey-scale echocardiographic and CMR parameters.

Variable	2004 TGA-Senning $n = 10$	2019 TGA-Senning $n = 10$	2019 Controls $N = 20$	
Male gender	5 (50%)	5 (50%)	11 (55%)	1.000
Age (years)	15 ± 3	32 ± 3	30 ± 4	0.231
Associated lesions				
ASD n(%)	1 (10%)	1 (10%)	–	–
VSD n(%)	3 (33%)	3 (33%)	–	–
PDA n(%)	3 (33%)	3 (33%)	–	–
PS n(%)	2 (20%)	2 (20%)	–	–
Comorbidities				
Diabetes n(%)	0 (0%)	0 (0%)	0 (0%)	1.000
Hypertension n(%)	0 (0%)	0 (0%)	0 (0%)	0.345
Cholesterol n(%)	0 (0%)	0 (0%)	1 (5%)	1.000
Smoking n(%)	0 (0%)	1 (10%)	2 (10%)	1.000
Medical therapy				
Beta-blocker n(%)	0 (0%)	2 (20%)	0 (0%)	0.111
ACE-I or ARB n(%)	2 (20%)	5 (50%)	0 (0%)	0.002
Diuretics n(%)	0 (0%)	0 (0%)	0 (0%)	1.000
Clinical				
NYHA I/II/ n(%)	10 (100%)/0 (0%)	9 (90%)/1 (10%)	20 (100%)/0 (0%)	*p* Value (cross-sectional)
BMI (kg/m^2)	15 ± 3	25 ± 5	23 ± 2	0.120
Systolic BP (mmHg)	119 ± 15	133 ± 23	125 ± 10	0.588
Diastolic BP (mmHg)	73 ± 16	74 ± 16	76 ± 13	0.619
QRS duration (ms)	100 ± 17	105 ± 18	95 ± 17	0.155
Echocardiography				
LVEF (%)	64 ± 5	74 ± 10	64 ± 6	0.004
MAPSE (mm)	NA	17 ± 5	18 ± 3	0.776
E/A	NA	3.0 ± 1.9	1.8 ± 0.5	0.107
E/e'	NA	8 ± 5	5 ± 1	0.055
RV EDA (cm^2)	NA	28 ± 5	19 ± 5	<0.0001
RV ESA (cm^2)	NA	19 ± 7	9 ± 3	<0.0001
RVFAC (%)	NA	35 ± 13	49 ± 10	0.008
TAPSE (mm)	NA	16 ± 3	22 ± 4	0.001
Cardiac magnetic resonance imaging				
RV EDVi (mL/m2)	NA	121 ± 48	–	–
RV ESVi (mL/m2)	NA	73 ± 45	–	–
RV EF (%)	54 ± 11	43 ± 11	–	–

NA: not available; ASD: atrial septal defect; VSD: ventricular septal defect; PDA: patent ductus arteriosus; PS: pulmonary valve stenosis; ACE-I: angiotensin converting enzyme inhibitor; ARB: angiotensinogen receptor blocker; NYHA: New York Heart Association class; BMI: body mass index; BP: blood pressure; LVEF: left ventricular ejection fraction; MAPSE: mitral annular plane systolic excursion; E: early diastolic velocity; A: late diastolic velocity; EDA: end-diastolic area; ESA: end-systolic area; RVFAC: right ventricular fractional area change; TAPSE: tricuspid annular plane systolic excursion.

were 6 (30%) patients with mild tricuspid regurgitation, compared to 1 (10%) with mild, 6 (60%) with moderate and 3 (30%) with severe tricuspid regurgitation in 2019. In 2004, 3 (15%) had mild left ventricular outflow tract obstruction, compared to 2 (20%) in the current study. No patients had baffle

Table 2. Peak systolic velocity, peak systolic strain rate and peak systolic strain in TGA-Senning patients (2004 and 2019) and controls (2019).

	Peak systolic velocity (cm/s)			Peak systolic strain rate (s^{-1})			Peak systolic strain (%)		
	Senning 2004	*Senning 2019*	*Controls 2019*	*Senning 2004*	*Senning 2019*	*Controls 2019*	*Senning 2004*	*Senning 2019*	*Controls 2019*
LV									
Base	6.7 ± 1.6	6.0 ± 1.0	7.3 ± 2.4*	−1.5 ± 0.4	−1.6 ± 0.4	−1.6 ± 0.8*	−21.1 ± 6.1	−22.4 ± 7.6	−23.9 ± 14.8
Midwall	5.6 ± 1.8	3.7 ± 1.1†	6.2 ± 2.9*	−1.7 ± 0.5	−1.0 ± 0.4†	−1.6 ± 0.6*	−22.1 ± 6.4	−16.6 ± 5.2	−19.8 ± 4.8*
Apex	5.0 ± 1.7	2.1 ± 1.2†	3.9 ± 1.9*	−1.3 ± 1.4	−0.9 ± 0.2	−1.5 ± 0.8*	−18.9 ± 8.3	−13.8 ± 4.6	−16.8 ± 3.6*
RV									
Base	3.6 ± 1.7	3.3 ± 1.5	8.7 ± 1.7*	−1.3 ± 0.5	−1.1 ± 0.4	−1.8 ± 0.5*	−17.6 ± 8.1	−17.3 ± 4.8	−26.9 ± 7.3*
Midwall	2.9 ± 1.8	3.0 + 1.7	6.1 ± 2.0*	−1.0 ± 0.3	−0.9 ± 0.4	−1.9 ± 0.5*	−11.5 ± 8.0	−13.4 ± 5.8	−30.2 ± 3.5*
Apex	2.3 ± 2.6	1.3 ± 1.0	2.9 ± 1.7*	−1.1 ± 0.3	−0.8 ± 0.4†	−1.6 ± 0.5*	−16.9 ± 7.1	−12.0 ± 4.0†	−24.4 ± 6.2*

Longitudinal analysis: †$p < 0.05$; Cross-sectional analysis: *$p < 0.05$.

stenosis or obstruction (either in 2004 or 2019). sRV ejection fraction on cardiac magnetic resonance (CMR) decreased significantly over time ($p = 0.018$).

Regional longitudinal LV and RV function

Tables 2 and 3 provide a summary of mean values of peak systolic and diastolic velocities, strain rate and strain of the LV and RV.

Longitudinal analysis

Over a 15-year time period, apical peak systolic strain rate (−1.1 ± 0.3 to −0.8 ± 0.4 s^{-1}; $p = 0.025$) and peak systolic strain (−16.9 ± 7.1 to −12.0 ± 4.0%; $p = 0.012$) significantly decreased in the sRV free wall, whereas no change was observed in the RV basal and mid segments (Figure 2). In the subpulmonary LV (spLV), a decrease in peak systolic velocity, peak systolic strain rate and peak systolic strain was observed, although not all values reached statistical significance (Table 2). There were no significant changes in diastolic function for the sRV or spLV.

Cross-sectional analysis

There were significant differences when comparing systolic and diastolic function of the sRV (subaortic RV) between TGA-Senning patients and controls (Figure 3). Basal, mid and apical peak systolic velocity, strain rate and strain of the sRV of Senning patients were significantly lower when compared to the subpulmonary RV and systemic LV of controls (except basal peak systolic strain which was not significantly different between sRV of Senning patients and sLV of controls). Basal, mid and apical peak early and late diastolic velocity and peak early diastolic strain rate of the sRV of Senning patients were significantly lower when compared to the subpulmonary RV and systemic LV of controls. The spLV (subpulmonary LV) of Senning patients had significantly lower values of mid and apical peak systolic velocity and strain rate when compared to controls.

Discussion

Our study indicated that in our group of 10 Senning patients, systolic and diastolic sRV function measured using Tissue Doppler imaging did not change over a 15-year follow-up period, except in the apical region of the sRV. We also observed a decline of spLV systolic function, which may be of clinical value. Nevertheless, when compared to an age-matched cohort, both the sRV and spLV of Senning patients exhibit significantly decreased measurements of systolic and diastolic function.

From standard greyscale imaging, the sRV of Senning is dilated, hypertrophied and exhibits mildly abnormal systolic and diastolic function when compared to healthy controls, which is confirmed in this study [15]. It is interesting to note that the proportion of patients with mild, moderate and severe tricuspid regurgitation increased from only 30% of patients having mild tricuspid regurgitation in 2004, to 90% having at least moderate tricuspid valve regurgitation in 2019. There is also an increase in patients taking ACE-inhibitors and a decrease in sRV ejection fraction indicating ventricular dysfunction. This is in contrast to the rather subtle changes in deformation imaging observed over a 15-year time period, with reductions in peak systolic strain rate and strain only observed in the apical segment of the RV free wall. Tricuspid valve regurgitation has been described as a marker of outcome [16] as well as a reflection of sRV dysfunction. Unfortunately, since peak systolic strain and strain rate are load dependent [14,17], the increase in tricuspid valve regurgitation may mask a more significant reduction in parameters reflecting systolic function of the sRV [14]. It is of interest, however, that significant changes were found in the apical segment of the sRV free wall, which is consistent with previous studies. Indeed, Eindhoven et al. [18] using speckle tracking imaging also reported a basal-to-apical gradient in peak systolic strain and strain rate of the sRV free wall in TGA after Mustard repair and similar regional

Table 3. Peak early and late diastolic velocity and peak early and late diastolic strain rate in TGA-Senning patients (2004 and 2019) and controls (2019).

	Peak diastolic velocity (cm/s)						Peak diastolic strain rate (s^{-1})					
	Early			Late			Early			Late		
	Senning 2004	*Senning 2019*	*Controls 2019*	*Senning 2004*	*Senning 2019*	*Controls 2019*	*Senning 2004*	*Senning 2019*	*Controls 2019*	*Senning 2004*	*Senning 2019*	*Controls 2019*
LV												
Base	−8.6 ± 2.7	−9.2 ± 2.7	−12.6 ± 2.9*	NR	−1.8 ± 1.2	−3.5 ± 1.7*	2.3 ± 0.6	2.7 ± 0.8	2.4 ± 0.7	NR	0.6 ± 0.6	1.0 ± 0.4
Midwall	−6.3 ± 3.3	−6.2 ± 2.0	−9.2 ± 3.2*		−0.9 ± 0.8	−2.5 ± 1.4*	2.7 ± 0.9	1.6 ± 0.8	2.4 ± 0.8		0.5 ± 0.2	1.0 ± 0.7*
Apex	−4.7 ± 2.0	−3.8 ± 1.8	−7.5 ± 7.2		−0.6 ± 0.5	−1.2 ± 0.8	1.7 ± 1.4	1.7 ± 0.8	2.8 ± 1.6*		0.3 ± 0.2	0.8 ± 0.6*
RV												
Base	−5.4 ± 2.2	−4.1 ± 2.4	−10.4 ± 2.0*	−1.5 ± 0.4	−2.0 ± 1.1	−7.4 ± 3.0*	1.5 ± 0.8	1.6 ± 0.8	2.6 ± 1.1*	NR	0.8 ± 0.5	1.2 ± 0.6
Midwall	−3.9 ± 2.8	−2.1 ± 1.5	−8.1 ± 3.0*	−1.5 ± 1.0	−1.1 ± 0.6	−5.2 ± 2.5*	1.4 ± 1.3	1.4 ± 0.6	2.7 ± 1.0*		0.7 ± 0.3	1.7 ± 0.6*
Apex	−2.0 ± 1.2	−1.0 ± 0.5	−4.5 ± 2.5*	−1.1 ± 0.8	−0.3 ± 0.2	−2.1 ± 1.8*	1.9 ± 1.3	1.3 ± 0.6	2.7 ± 1.1*		0.2 ± 0.1	1.0 ± 0.5*

Cross-sectional analysis: $^*p < 0.05$.

differences have been observed in the RV of patients with tetralogy of Fallot [19] and pulmonary arterial hypertension [20]. Differences in wall thickness [21], regional adaptation to increased afterload and/or volume load may all have influenced the reduction in apical sRV systolic function. Segmental anatomy of the right ventricle, with a trabeculated apical portion (becoming more pronounced in the setting of increased afterload) and a smooth inlet segment (with a larger capacity, but more vulnerable to increased volume load) may contribute to differential changes in the apex and basis of the sRV [22]. Although our study may certainly be subject to bias, given the small group of included patients, the relative stability of measurements underscores the difficulties in assessing changes in systolic and diastolic function over time. Lower strain values have been reported before and although a relationship with outcome has been reported [10], other studies considered the added value of strain analysis to be limited [11]. It could also be that once sRV dysfunction occurs, patients rather quickly develop overt clinical heart failure [7]. Progressive fibrosis has been reported [23] and could significantly impact diastolic function [8,16]. Although we observed a numerical reduction in peak early diastolic velocities, this did not reach statistical significance.

Of particular interest is that some changes in spLV systolic function were observed. Similarly to the RV in a biventricular circulation with a subaortic LV [24,25], the spLV in TGA patients after Senning repair may have prognostic significance. Over half of patients with sRV are reported to have pulmonary hypertension at the time of catheterisation [26,27] which related to worse outcome. Long-term increased resistive (pulmonary vascular resistance–PVR) and pulsatile (pulmonary artery compliance–PAC) load could have a detrimental effect on spLV systolic and diastolic function.

In the cross-sectional analysis, almost all segments showed lower systolic and diastolic values for TGA-Senning patients when compared to healthy controls. Although we did not assess circumferential deformation, we did not see the complete adaptation as described by Pettersen et al. [28] with the contraction pattern of the sRV resembling that of a normal LV. Our results suggest a reduction in systolic contractile function when compared to controls, which is consistent with previous studies [9,29,30], although it remains difficult to interpret to what extent they are influenced by increased afterload and preload (decreased in the presence of atrial baffles; increased due to tricuspid

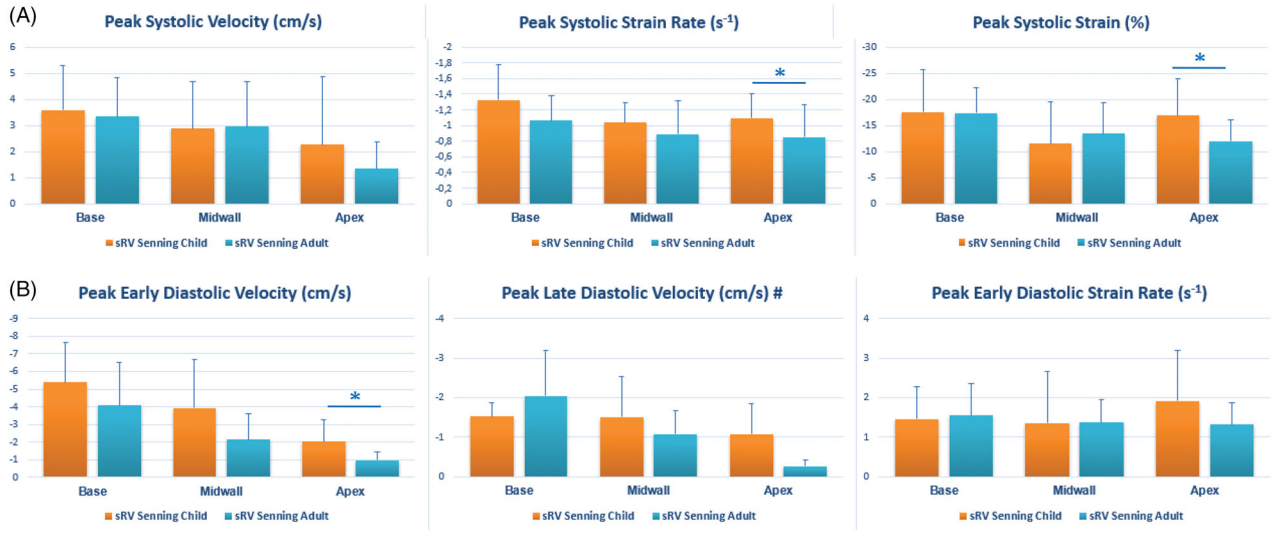

Figure 2. A. Longitudinal analysis (2004–2019) of sRV systolic function: Peak systolic velocity (cm/s), peak systolic strain rate (s^{-1}) and peak systolic strain (%). B. Longitudinal analysis (2004–2019) of sRV diastolic function: Peak early and late diastolic velocity (cm/s), peak early diastolic strain rate (s^{-1}) for the sRV of Senning patients. $^{*}p < 0.05$.

Figure 3. Cross-sectional analysis (2019) of systolic sRV function: Peak systolic velocity (cm/s), peak systolic strain rate (s^{-1}) and peak systolic strain (%) of the sRV of Senning patients compared to A1. the subpulmonary RV and A2 the systemic LV of controls. $^{*}p < 0.05$. Cross-sectional analysis (2019) of diastolic dysfunction: peak early and late diastolic velocity (cm/s) and peak early and late diastolic strain rate (s^{-1}) for the sRV of Senning patients versus B1 the spRV (green) or B2 systemic LV (red) of controls. $^{*}p < 0.05$

regurgitation). Similarly, our results suggest a reduction in diastolic function. Although fibrosis in patients with sRV has been described [31], results may be confounded by abnormal filling due to non-compliant atrial baffles. Abnormal stroke volume responses during exercise due to limited ventricular filling in the presence of non-compliant atrial baffles have been described [8,9], but a disruption of normal atrial function due to prior atrial surgery is also likely possibiliy [9].

Again, of the particular interest is that we observed decreased systolic and diastolic function of the spLV in Senning patients when compared to a normal LV [32]. Similar to changes observed in the sRV of Senning patients this is either a physiologic adaptation or subclinical dysfunction. The intuition of the congenital heart disease specialist is that a LV (with a mitral valve) in a subpulmonary position should easily tolerate the lower pressures in the pulmonary circulation. However, progressive changes in resistive and pulsatile load (driven by a struggling sRV) and ventricular-ventricular interactions may also cause progressive spLV dysfunction. On the other hand, we and others have shown that pulmonary outflow obstruction may protect against the development of tricuspid regurgitation and heart failure in ccTGA patients [33]. Whether this is also true for pulmonary hypertension in Senning patients remains to be evaluated as adaptation of the subpulmonary ventricle may depend on the type of increased afterload [34].

Conclusions

Our study showed that systolic and diastolic sRV function measured using Tissue Doppler imaging did not change over a 15-year follow-up period, except in the apical region. Interestingly, there was also a decline of spLV systolic function, which may be of clinical value. On the other hand, when compared to age- and gender-matched controls, both the sRV and spLV of Senning patients exhibit significantly decreased measurements of longitudinal systolic and diastolic function.

Limitations

Firstly, the number of patients included in the study is small, making the study subject to bias. We only measured longitudinal function from an apical 4-chamber view. Although it would have been interesting to assess circumferential strain as described by Pettersen et al., we believe it would not have changed our conclusions. Care was taken to align the RV free wall to the ultrasonic beam, making alignment unlikely to be the explanation for lower apical strain values. Second, we compared measurements available from the original study, but were unable to repeat measurements on the raw data, which could introduce bias. Third, differences in medical therapy between 2004 and 2019 could have introduced differences between both measurements.

Disclosure statement

The authors report no relationship that could be construed as a conflict of interest.

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