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Echocardiographic Analysis of Correlation Between Right Ventricle Load and Function and Left Ventricle Diastolic Malfunction in Symptomless Valvular Cardiovascular Disease Patients

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Background: Left ventricle diastolic malfunction (LVDMf) is a valvular cardiovascular disease. Here, we assessed the correlation between right ventricle (RV) load and function (L&F) and diastolic malfunction (DMf) in symptomless valvular cardiovascular disease patients.


Material/Methods: We enrolled 59 subjects who underwent right-heart catheterization, assessing their echocardiographic analysis results while performing exercises in supine position, comparing results at rest and during maximum exercise. Subjects were furthermore stratified according to resting DMf. Using cardiac resonance imaging (CRM), we assessed cardiac morphology and chamber size. RV stroke, pulmonary artery conformation, pulmonary artery elastance, pulmonary artery pulsatility, and right atrial (pRA) pressure were indexed for supine exercises.

Results: We observed that DMf grade 1 (G-1) and grade 2 (G-2) were present in 28 patients and 16 patients, respectively, while the remaining 15 patients had a normal filling pattern in the left ventricle. In comparison to patients with DMf of G-1, patients with normal diastolic filling pattern had higher volume index for RV end-diastolic (endD) (81 ± 14 mL/m² vs. 68 ± 12 mL/m², $P=0.08$) and for RV end-systolic (endS) (34 ± 11 mL/m² vs. 27 ± 8 mL/m², $P=0.07$). We also observed that in G-2 DMf pulmonary artery, pressure and elastance of the pulmonary artery were enhanced and were correlated with optimum oxygen intake and RV volume ($r=-0.69$, $P<0.001$).

Conclusions: We found that enhancement in RV afterload, which returns to normal at rest, is correlated with mild DMf. Additionally, despite maximum exercise, it is reciprocally associated with maximum oxygen intake and right atrial pressure.

MeSH Keywords: **Cardiovascular Diseases • Echocardiography • Stroke Volume • Ventricular Function, Left**

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Background

Valvular cardiovascular disease is a degenerative and slowly worsening disorder in which 1 or more of the heart valves do not operate in the right direction due to blood pressure overload. Heart failure symptoms can result due to pressure overload and can eventually lead to the need for heart valve replacement [1]. Nevertheless, symptoms of valvular cardiovascular disease are missed in some patients due to the gradual development of pressure overload. This is why maintaining adequate cardiac output is necessary to compensate for pressure overload by changing output, in which left ventricle (LV) cor pulmonale assists in conservation of end-systolic (endS) wall accentuation. It is also found that there is an enhanced diastolic malfunction (DMf) and myocardial inelasticity, and a correlation between dysfunctional coronary flow withhold and percolate fibrosis was observed, during elastance [2–4]. We observed that during DMf, the filling pressure was enhanced, especially during exertion, and, due to this activity, the right ventricle (RV) was exposed to enhanced afterload [5]. A previous physiology study found that RV diastolic possessions and pressure were affected by loading on the left side of the heart, perhaps through ventricular reciprocity [6]. No evidence was found of a correlation between RV hemodynamics and morphology and left ventricle diastolic malfunction (LVDMf) in a population with symptomless valvular cardiovascular disease [7,8]. We also found no evidence that alteration in RV load and function (L&F) is caused by LVDMf. It is also unclear whether alteration in RV L&F is due to chronic raised LV filling pressures inducing afterload mismatch or ventricle interdependency. To the best of our knowledge, this is the first study to assess the correlation between RV L&F and LVDMf in symptomless valvular cardiovascular disease (vCVD) patients.

Material and Methods

Our investigation was approved by our Institutional Review Board. Written informed consent was obtained from all enrolled subjects and they were carefully questioned to be sure they did not have symptoms. We first screened 293 subjects, and 234 were eliminated due to serum creatinine $>200 \mu\text{mol/L}$ ($n=19$), pacemaker dependency ($n=26$), chronic atrial fibrillation ($n=27$), no consent ($n=145$), and LV ejection fraction $<50\%$ ($n=17$). Eventually, 59 subjects were enrolled between July 2018 to June 2019. Inclusion criteria were having more than 50% LV ejection fraction, more than 3.5 m/s aortic peak velocity, and less than 1 cm^2 area of aortic valve. Echocardiographic analysis of all subjects was performed after right-heart catheterization, while at rest and while performing maximal exercise, both in supine position. We performed cardiopulmonary testing during exercise and cardiac magnetic resonance imaging at designated time intervals.

Echocardiographic analysis

Echocardiographic analysis was performed using the Vivid E9 ultrasound system purchased from General Electric (Horten, Norway). Echocardiographic analysis is very commonly used to assess LV function. Transthoracic echocardiographic analysis also provides extensive information on cardiac function and structure and helps guide therapy and diagnosis. In the present study, we performed Doppler recordings an average of 3 to 5 heartbeats and at a minimum of 60 s^{-1} to set the frame rate. The body surface area was standardized for measurements. In this process, continuous-wave (CW) Doppler was used for the examination of optimum aortic valve jet velocity in multiple apical views. The measurement value of diameter of LV outflow tract was 5 mm below the aortic valve. Using a continuity equation, the aortic valve area was measured. The left atrium (LA) endocardial border was traced in apical 4- and 2-chamber views. In this process, the biplane method of disks was used for the measurement of left atrium volume, standardized to body surface area according to modified Simpson's Rule [9]. Mitral E-wave deceleration time and peak early (E) and late wave velocity (A) were computed from mitral inflow. The myocardial velocity gradient was determined by 2-dimensional tissue Doppler imaging technique with pulsed wave, which is a new feature used to indicate regional LV contraction. With the help of these recordings, at the septal mitral valve annulus, tissue Doppler early diastolic velocity was calculated. On the basis of volume index of LA (ViLA), E/A, and e' , E/e' , we ranked diastolic function (DF) [10]. For normal DF patients, ViLA $<32 \text{ mL/m}^2$, E/A 0.9 to 2.1 and $e' >8 \text{ cm/s}$ were chosen as selection/inclusion criteria. In grade 1 (G-1) DMf patients, $e' \leq 8 \text{ cm/s}$ and E/A <0.9 were chosen, while in grade 2 (G-2) DMf patients the value of ViLA $\geq 32 \text{ mL/m}^2$, $e' \leq 8 \text{ cm/s}$, and E/A 0.9 to 2.1 were chosen as selection/inclusion criteria.

Cardiovascular magnetic resonance imaging (Cv-MRI)

Cardiovascular magnetic resonance imaging (Cv-MRI) is an essential medical imaging technology for assessing the structure and function of the cardiovascular system in a non-invasive manner. We performed cardiovascular magnetic resonance imaging activity on a Hitachi echelon 1.5T scanner (Hitachi Medical System, Europe). For more precise and reliable results, an expert examiner examined the images and assessed echocardiographic and invasive data on a dedicated workstation. LA mass and atrial and ventricular chamber volumes were calculated from a cine succession with short axis slices. Using the method described in a previous report [11] end-diastolic (endD) LV mass was calculated by multiplying the inter-slice gap by the difference between epicardial and endocardial areas with a myocardial density of 1.05 g/mL. In an earlier report, LV was found to have papillary muscles with the exclusion of LV mass. Additionally, LV volume includes LV appendage volume [12].

We took RV volumes and papillary muscles together while we analyzed the RV up to trabeculations and pulmonary valve. In endD and endS conditions, we measured 3 characteristics – stroke, ejection fraction, and volume – with respect to RV. We administered 0.1 mmol/kg gadoterate meglumine purchased from Dotarem (Guerbet, Aulnay-Bois, France) and then waited 15 min before late gadolinium enhancement imaging. We report “non-specific” midwall and ischemic data as late improvement pattern. We used a 17-segment cardiac model and enumerated and reported segment thickness along with longitudinal circulation and radial data [13].

Right-heart catheterization

In the cardiovascular system, for the accurate and direct measurement of hemodynamic parameters, we used right-heart catheterization, which is an invasive procedure. We used 7.5-F triple-lumen Swan-Ganz catheters purchased from Edward Lifesciences (Irvine, CA). With an increment rate of 25W/3 min, a multistage symptom-limited exercise test was performed from rest to exhaustion. We performed exercise testing on an exercise bike for echocardiographic analysis with an electronic adjustable slope in a semi-supine position, on an Echo Cardiac Stress Table purchased from Lode B.V. (Amsterdam, The Netherlands). For better and more precise results, we observed heart rate, arterial oxygen saturation, and pressure of blood on a regular basis. At an average of >10 s, we measured pressure due to pulmonary artery wedge (pPAW) at end-expiration during rest. During exercise, we measured mean pPAW. Using thermodilution, we also recorded pressure of the pulmonary artery (pPA), and pressure of the right atrium (pRA), and performed 3 successive measurements of cardiac output with <10% variability at each level of exercise. In the last phase of this activity, before and after the termination of exercise, in a composite middle venous blood sample, we measured lactate concentration and oxygen saturation.

Hemodynamic monitoring

All arithmetic measurements were made at optimum exercise and at rest conditions. The work index was calculated as RV stroke (WiRVS)=0.0136 multiplied by volume index of stroke (ViS) and (mean PAP-RAP), using a previously described method [14]. For the calculation of pulmonary artery compliance, we divided stroke volume (systolic pPA minus diastolic pPA) by pRA [15]. We divide systolic pPA by stroke volume to assess pulmonary artery elastance [16]. To determine the pulmonary artery pulsatility index and pRA/pPAW ratio, we subtracted the diastolic pPA value from the systolic pPA value and then divided this value (the value which we received after subtraction) by the pRA. To assess the value of valvular-arterial impedance, we added mean transvalvular pressure gradient and systolic arterial pressure together and then divided their sum by ViS [17].

Optimal oxygen consumption

Using the Bruce treadmill test, we subjected 33 patients to optimum oxygen consumption testing on the basis of standard cardiopulmonary function [18]. During the next step of this test, we continuously measured respiratory variables (heart rate, carbon dioxide volume, and oxygen consumption volume) using an online tool (AMIS 2001; Innovision) from Odense (Denmark). In the last part of the test, we measured optimum oxygen consumption at the highest value of more than half-minute periods.

Statistical analysis

Using ANOVA, we tested between-group differences in continuous variables with a Gaussian distribution, and we used a non-parametric rank-sum test for variables with non-Gaussian distribution. To compare the proportions of categorical variables, we used Fisher's exact test. For the assessment of linear bivariate analysis, we used Pearson's correlation coefficient. To assess the relationship between oxygen consumption and invasive hemodynamic calculations, we used multiple regression analysis.

Results

The exercise protocol activity was successfully completed by all 59 enrolled patients. Due to claustrophobia, 2 patients were unable to complete the Cv-MRI activity. According to LV diastolic filling structure, we classified patients into 3 groups: 28 patients had DMf of G-1, 16 patients had G-2 and 15 patients had a normal LV filling pattern. None of the patients had DMf of G-3. In comparison to normal filling pattern, DMf patients were found to have a longer history ($P<0.001$) (Table 1). We observed that E/A was significantly lower in moderate DMf patients during Valsalva maneuver (0.85 ± 0.30 vs. 0.65 ± 0.19 , $P<0.001$). LV DF was found to be unaffected by the severity of valvular cardiovascular disease in terms of aortic valve area ($P=0.85$) (Table 1). DMf patients with G-1 had higher Zva values in comparison to the normal patients (5.4 ± 0.7 vs. 4.5 ± 0.5 mmHg/mL/m², $P=0.005$) (Table 1). We observed that in G-2 DMf patients, the tricuspid annular plane systolic excursion (TAPSE) was higher, but we did not find any significant between-group differences. Moderate DMf patients had higher basal RV free wall (in terms of S') as compared to normal DF patients (Table 1).

Cardiovascular magnetic resonance imaging (Cv-MRI)

In comparison to patients with DMf of G-1, patients with normal diastolic filling pattern had higher volume index for RV end-diastolic (endD) (81 ± 14 mL/m² vs. 68 ± 12 mL/m², $P=0.08$)

Table 1. Clinical characteristics of echocardiographic analysis of patients with respect to diastolic malfunction.

Characteristics	In unit	Total subjects (n=59)	Normal (n=15)	Grade-1 [®] (n=28)	Grade-2 [®] (n=16)	P-value
Gender, M	%	43 (73)	9 (60)	20 (71)	12 (75)	0.58
Hbp	%	42 (71)	9 (60)	20 (71)	10 (63)	0.20
AAV	cm ²	0.95±0.22	0.90±0.20	0.95±0.23	0.99±0.23	0.85
Sugar	%	7 (12)	Nil	5 (18)	4 (20)	0.65
Vmax [#]	m/s	5.1±0.8	5.0±0.7	5.2±0.9	5.1±0.8	0.85
AG [*]	mmHg	48±15	45±13	50±16	50±13	0.80
Age	years	78±12	66±10	80±8	76±9	<0.004
Rate ^{**}	mL/min	78±18	79±17	74±19	80±16	0.52
Septal-E'	cm/s	0.08±0.04	0.9±0.04	0.08±0.02	0.08±0.02	0.008
E-e' ratio	cm/s	14±5	12±4	14±5	16±6	0.05
E/A		0.85±0.30	1.16±0.30	0.72±0.14	0.90±0.24	<0.002
Strain ^{***}	%	-18.5±2.6	-20±2.8	-18±2.2	-19±2.4	0.18
Time of retardation	ms	305±98	214±30	378±88	255±35	<0.004
Velocity of E	m/s	0.76±0.22	0.82±0.24	0.66±0.22	0.84±0.20	0.05
Velocity of A	m/s	0.98±0.32	0.75±0.16	1.07±0.36	1.04±0.24	0.04
TAPSE	mm	25±4	23±4	25±3	26±5	0.16
Zva	mmHg/mL/m ²	6.2±0.8	4.5±0.5	5.4±0.7	4.4±0.7	0.005
S'	cm/s	0.16±0.06	0.15±0.02	0.16±0.04	0.18±0.06	0.06
Area ^{##}	m ²	1.99±0.17	1.90±0.26	1.80±0.19	2.1±0.29	0.30

[®] Diastolic malfunction function type; M – male; Hbp – high blood pressure; AAV – area of aortic valve; # aortic valve; * average gradient of aortic valve; ** estimated rate of glomerular filtration in 'E' case; *** global longitudinal strain of left ventricle; TAPSE – tricuspid annular plane systolic excursion; Zva – valvulo-arterial impedance; ## with respect to body surface. All data are shown in "mean±standard deviation" format. P-value is calculated as per ANOVA model.

and for RV end-systolic (endS) (34±11 mL/m² vs. 27±8 mL/m², P=0.07) (Table 2). We also observed that in patients with DMf, the volume index of RV stroke was lower than in patients with normal filling pattern (43±9 mL/m² vs. 48±8 mL/m², P=0.29). There was no correlation between DF and ejection fraction of RV (P=0.58) and we found no significant difference in ejection fraction or in LV size or mass in DMf patients (Table 2). A total of 15 patients (26%) had evidence of replacement of myocardial fibrosis on late gadolinium enhancement, but this was not correlated with DF (P=0.40).

Invasive hemodynamics

Considerable skill is required in invasive hemodynamic monitoring. We did not find any difference in systemic vascular resistance or cardiac indices at rest. However, we found a

correlation between DMf and increased diastolic and systolic pPA (Table 3). We observed higher pulmonary artery elastance (0.40±0.11 mmHg/mL vs. 0.30±0.10 mmHg/mL, P=0.005) in DMf patients, and lower pulmonary artery compliance was observed with increased pPA (5.7±1.2 mL/mmHg vs. 6.9±2.5 mL/mmHg, P=0.09). At rest, we did not find any correlation between DF in patients with G-2 DMf and enhanced work index with respect to RV stroke (P=0.04). An average exercise load of 100 (range, 90–130) watts was successfully accomplished by the patients during supine exercise without any variation between various grades of DF (P=0.85). During exercise, there were significantly increased LV and RV filling pressures in all patients. After maximum exercise, enhancement in pPAW above 30 mmHg was observed in 45% patients. Most G-2 DMf patients (14 patients, 88%) had higher pPAW values (pPAW >30 mmHg) than patients who had normal DF (6 patients, 40%).

Table 2. Ecocardiographic analysis in terms of hemodynamic monitoring with respect to diastolic malfunction.

Characteristics	In Unit	Total subjects (n=57)	Normal (n=15)	Grade-1 [®] (n=26)	Grade-2 [®] (n=16)	P-value
Emptying fraction*		42±9	44±7	44±8	38±9	0.25
Ejection fraction**	%	64±8	67±6	63±9	64±7	0.34
Ejection fraction [#]		63±6	61±8	64±5	64±5	0.58
*Actual		53±10	56±6	51±6	54±12	0.49
*Minimum		33±8	34±7	31±8	35±13	0.40
endD**		88±19	92±16	85±17	89±18	0.59
endS**	mL/m ²	35±11	33±9	35±12	35±11	0.06
endD [#]		71±11	81±14	68±12	69±14	0.08
endS [#]		29±9	34±11	27±8	27±7	0.07
Stroke [#]		44±9	48±9	43±8	44±11	0.29
!Mass index	g/m ²	75±19	77±17	73±13	77±1	0.79

[®] Diastolic malfunction; * right atrial; ** left ventricle; # right ventricle; All data are shown in “mean±standard deviation” format. P-value is calculated as per ANOVA model.

Table 3. Measurements and calculations of hemodynamic monitoring with respect to diastolic malfunction.

Characteristics	In Unit	Optimum [#]			Reference*			P-value P _{opt} /P _{ref}
		Normal	Grade-1 [®]	Grade-2 [®]	Normal	Grade-1 [®]	Grade-2 [®]	
Measurement								
pPAW	mmHg	31±5	33±8	35±7	10±3	12±6	15±5	0.29/0.19
Heart rate	Beats/min	128±27	121±20	115±18	56±10	67±8	64±6	0.29/0.15
pRA	mmHg	13±5	15±6	17±7	9±3	7±3	8±6	0.04/0.03
pPA systolic	mmHg	62±15	68±13	77±11	23±7	31±9	37±5	0.04/0.27
Cardiac index	L/min/m ²	7.2±1.6	5.9±1.3	6.4±1.2	2.9±0.7	2.5±0.5	3.7±0.9	0.004/0.17
!Volume index	mL/m ²	65±17	56±12	60±11	50±7	41±8	51±9	0.36/0.03
Calculation								
Work index**	g/m ² /beat	30.6±8.2	25.2±6.4	30.8±7.9	7.1±2.6	7.3±2.4	9.9±3.9	0.46/0.67
PAC	mL/mmHg	2.9±0.9	1.8±0.8	2.2±0.9	6.9±2.5	5.8±2.2	5.7±1.2	0.09/0.13
PVR	wood	1.5±0.6	1.7±0.6	1.6±0.4	1.3±0.9	1.4±0.7	1.5±0.5	0.07/0.06
pPA index	mmHg	3.1±1.6	3.6±1.7	3.2±1.4	2.6±0.7	3.9±2.2	4.5±2.7	0.03/0.07
pRA index	mmHg	0.2±0.2	0.3±0.3	0.4±0.2	0.7±0.2	0.6±0.3	0.5±0.5	0.18/0.76
PAE	mmHg/mL	0.6±0.2	0.7±0.2	0.7±0.2	0.4±0.1	0.5±0.1	0.6±0.2	0.39/0.59

[#] At maximum exercise condition; * at rest condition; @ diastolic malfunction grade type; P_{opt} – P-value for optimum condition; P_{ref} – P-value for reference condition; pPAW – pressure of pulmonary artery wedge; pRA – pressure of right atrial; pPA – pressure of pulmonary artery; ** right ventricular stroke; PAC – pulmonary artery compliance; PVR – pulmonary vascular resistance; PAE – pulmonary artery elastance. All data are shown in “mean±standard deviation” format. P-value is calculated as per ANOVA model.

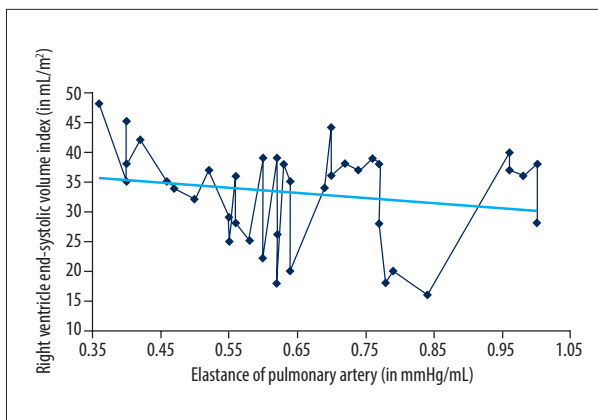


Figure 1. At optimum exercise condition, correlation between elastance of pulmonary artery and volume index of RV ends.

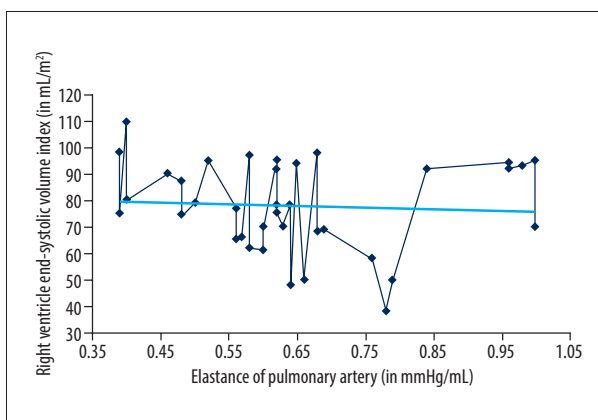


Figure 2. At optimum exercise condition, correlation between elastance of pulmonary artery and volume index of RV endD.

We did not find any significant differences in pRA/pPAW between groups. In DMf patients, systolic pPA was higher after exercise than before. At maximum exercise, enhanced pulmonary artery elastance was observed in DMf patients, and we found the opposite correlation at rest with RV size (ViRV endS $r=-0.49$, $P=0.001$) (Figure 1) as well as ViRV endD $r=-0.57$, $P<0.001$ (Figure 2). At maximum exercise, we did not find any correlation between DMf and WiRVs. We observed that compliance ($r=0.61$, $P<0.001$) and elastance of pulmonary artery ($r=-0.69$, $P<0.001$) were correlated with maximum oxygen consumption (Figure 3). After adaptations of pPAW at maximum exercise and cardiac output, there was no significant difference in correlations between maximum oxygen consumption and pulmonary artery elastance ($\beta=-0.42$, $P=0.06$).

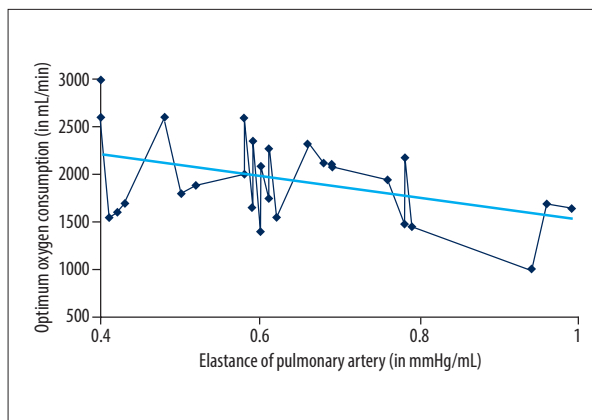


Figure 3. At optimum exercise condition, correlation between elastance of pulmonary artery and optimum oxygen.

Discussion

The present study confirmed the findings of the echocardiographic analysis of the correlation between RV L&F and LVDMf in symptomless valvular cardiovascular disease patients as follows: (a) In symptomless valvular cardiovascular disease (vCVD) patients, abnormal LV DF was very common; (b) In the case of moderate DMf, enhancement in RV afterload was observed. We found that enhancement in RV afterload, which returns to normal at rest, is correlated with mild DMf, and despite maximum exercise, it is reciprocally connected to optimal oxygen intake and correlated with right atrial pressure. Morphologically, DMf patients had smaller chamber size, which resulted in reduced RV compliance.

During LV ejection, extreme afterload is correlated with valvular cardiovascular disease. Myocardial hypertrophy is considered to be the reaction of the LV to increased systolic wall stress. In addition to this, endS wall stress and pressure are counterbalanced by increased wall thickness during myocardial hypertrophy. However, an adaptive reaction in the beginning causes relaxation deformities and ultimately leads to the increased myocardial malfunction as a result of diffuse fibrosis, causing LVDMf. In significant valvular cardiovascular disease, only 1/3 of patients had normal DF. A correlation between moderate or severe DMf and 1-year mortality was previously observed, without showing any correlation with symptomatic valvular cardiovascular disease [19]. We found a correlation between severity of DMf and poor outcome with respect to aortic valve replacement in severe valvular cardiovascular disease patients [20,21].

During exercise, an increase in LV filling pressure is an indication of DMf [22,23], which is common in valvular cardiovascular disease patients. RV afterload occurs in acute valvular cardiovascular disease due to increased postcapillary pulmonary artery hypertension enhancement. Additionally, LV consistently

affects the RV due to LV remodelling with enhanced wall depth, while the interventricular septum is shared by the LV and RV. Diastolic malfunction patients were found to have small RV size. We found that if the filling pressure of one ventricle is increased, then a significantly lower compliance subsequently occurs in the neighbouring ventricle, as also mentioned in previous reports [24,25]. A recent study of idiopathic pulmonary hypertension patients found excessive RV DMf due to myofibrillar anxiety and enhanced chamber constraint [26]. We observed that, with respect to HFpEF, LVDMf has numerous associations with valvular cardiovascular disease. Therefore, based on LVDMf physiology, we hypothesized that there is a unique phenotype in symptomless valvular cardiovascular disease patients due to small RV volume with decreased compliance.

At rest condition, with respect to RAP, RV preload was found to be normal, without consideration of the LV DF. Thus, we observed a significantly higher RV preload in moderate DMf patients during exercise. We did not find enhancement in normal DF patients due to enhancement in pRA, but we found enhancement in pRA to 15mmHg or more in 2/3 of DMf patients. On the other hand, there is a possibility that, as a result of increased pRA during exercise, moderate DMf patients did not have enhanced venous return.

We did not find any indication of RV systolic malfunction, but RV compliance, elastance, and preload were associated with the following parameters: a) increased exercise resulted in insufficient increase in RV stroke volume, b) RVEF on Cv-MRI, c) PA pulsatility index or RAP/pAWP ratio, and d) TAPSE. Therefore, obvious RV failure and central hemodynamics are not correlated with each other due to modification in central hemodynamics. A previous study found that patients with moderate DMf tend to have RV systolic function with increased RV contractility, which was correlated with RV DMf [26].

In the past decade, there have been tremendous change in the rating systems used, showing many advantages and disadvantages. In this work, for DF rating, we used Doppler echocardiography, which is widely used for rating. We selected patients who could complete supine exercise testing. Patients with cardiopulmonary disease and atrial fibrillation were excluded from this analysis, which was the result of selection from a comparatively sound valvular cardiovascular disease population. Additionally, the present investigation included completion of optimum exercise testing; therefore, it is not suitable for use in patients with more advanced co-morbidity and symptomatic valvular cardiovascular disease. Our results may have been biased due to the inclusion of subjects with co-morbidities and coronary artery disease (n=4) and diabetes (n=6), as we did not find any cases of significant anemia. Additionally, coronary angiographic analysis was done to investigate asymptomatic coronary artery disease, but it was unclear how many patients had coronary artery disease. Maximum exercise testing was performed by all patients and was assessed by 2D echocardiography, without expansion of new zonal wall motion malformation. The patients exercised in semi-supine posture and displayed the expected enhancement in preload conditions in comparison to upright posture, which is why it was not the primary factor determining differences between groups, but in some patients, enhancement in pRA was recorded. We also assessed focal fibrosis. We found no relationship between DF and focal fibrosis; but due to the small sample size, the possibility of a type 2 error is very high.

Conclusions

We found that enhancement in RV afterload, which is lower at rest, is correlated with mild DMf. Additionally, during optimal exercise, it is reciprocally correlated with optimal oxygen intake and is associated with right atrial pressure. We also found that symptomless valvular cardiovascular disease is common and significant associated with abnormal LV DF. DMf patients were tend to have smaller chamber size, which results in reduced RV compliance.

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