Interactive CardioVascular and Thoracic Surgery 23 (2016) 4–8
doi:10.1093/icvts/ivw054 Advance Access publication 18 March 2016

Cite this article as: Akyüz Özkan E, Khosrashahi HE, Serin Hİ, Metin B, Kılıç M, Geçit UA. Cardiac and arterial elastance and myocardial wall stress in children with pectus excavatum. Interact CardioVasc Thorac Surg 2016;23:4–8.

Cardiac and arterial elastance and myocardial wall stress in children with pectus excavatum

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Received 5 November 2015; received in revised form 23 January 2016; accepted 8 February 2016

Abstract

OBJECTIVES: Pectus excavatum (PE) is one of the most common skeletal deformities of childhood. The study was undertaken to assess cardiac functions in children with PE.

METHODS: Echocardiography was performed on 32 children with PE and 40 age-matched healthy controls. The following parameters were monitored: meridional left ventricular (LV) wall stress (WSM), arterial elastance (E_a), LV elastance at end-systole derived by single beat ($Ees_{(sb)}$), LV circumferential end-systolic wall stress (ESWSc), midwall shortening fraction (SFmid), predicted midwall fiber shortening for a measured fiber stress (midwall VCFc), myocardial fiber stress (MFS), LV end-systolic dimension (LVES), LV end-diastolic dimension (LVED) and end-systolic blood pressure (P_{es}), LV wall thickness at end-systole (h_{es}). To assess the severity of PE, Haller index (HI) was calculated by computed tomography of the thorax.

RESULTS: SFmid, ESWSc, midwall VCFc and MFS were lower in PE children than in controls. The degree to which the parameters SFmid, ESWSc, midwall VCFc and MFS were altered in PE children was 14.9, 27.5, 20.3 and 20.3%, respectively. The minimum HI value of children with PE was 2.00, the maximum value was 4.93 and the arithmetic mean was 2.62 ± 0.56. Of the 32 children, 14 (43.75%) demonstrated mild deformity, 15 (46.88%) showed moderate and only 3 (9.37%) had severe deformity. In children with PE, there was no statistically significant correlation between the cardiac data (ESWSc, midwall VCFc, MFS, Ea, $Ees_{(sb)}$, LVES, LVED, P_{es}, h_{es}) and HI.

CONCLUSION: We found ESWSc, MFS, midwall VCFc and SFmid to be lower in children with PE than in controls. We concluded that the myocardial contractility and afterload is affected in children with PE.

Keywords: Cardiac function • Children • Pectus excavatum

INTRODUCTION

Pectus excavatum (PE), or funnel chest, is one of the most common congenital skeletal deformities, seen in ∼1 in every 400 male births and characterized by an inward depression of the sternum [1].

The aetiology of PE is unknown. Most pectus deformities are seen as isolated abnormalities. The primary abnormality is dystrophic growth of the costal cartilages and sternal depression comes thereafter. Subtle abnormalities in collagen morphology in the costal cartilages of children with pectus deformities have been shown, but the causal significance is not fully understood. In about one-third of cases, there is a positive family history of chest wall deformity. Congenital heart diseases are seen more frequent in children with PE [2]. Approximately 5% of children with a pectus deformity demonstrate a concomitant scoliosis. Symptoms are variable and age-related. Young children are usually asymptomatic but older children and adolescents may complain of vague chest and back pains, especially on exertion. Fatigue and reduced exercise tolerance may be seen more frequently among adolescents. These symptoms may, however, be connected to levels of inactivity rather than to the pectus deformity directly [2].

There is some controversy over the cardiopulmonary functions in patients with PE [3]. Some studies demonstrated a significant compromise in cardiac or pulmonary functions [4], whereas others showed no changes in cardiac functions [5]. Decreased cardiac output, mitral valve prolapse (MVP) and dysrhythmias are considered to be the cardiac effects of PE. Compression of the heart by the chest wall results in incomplete filling and decreased stroke

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volume (SV) and subsequently decreased cardiac output [4]. Similarly, compression of the right heart by the chest wall can lead to symptoms including dyspnoea and chest pain with exertion [6].

There are numerous investigations into cardiac functions including SV, cardiac output, ejection fraction (EF), shortening fraction (SF) in children with PE, but not on arterial and ventricular systolic elastance, myocardial structure, myocardial fiber stress (MFS) and wall stress. The aim of this study was to assess these parameters and the relationship between the Haller index (HI) and arterial–ventricular elasticity, myocardial wall stress and fiber stress in children with PE.

METHODS

We studied 32 paediatric patients (23 males and 9 females) selected randomly and not showing any other congenital anomaly or disease and 40 healthy subjects prospectively. Patients who had congenital anomalies or comorbid diseases were excluded.

A full history was taken and a complete physical examination was performed by the physician. The body height and weight of all the children were recorded. Body surface area (BSA) was calculated by the Mosteller formula [7].

An electrocardiogram was recorded in all patients. Transthoracic echocardiography was performed by an experienced paediatric cardiologist and the following parameters were monitored to assess ventricular contractility and afterload: meridional left ventricular (LV) wall stress (WSM, g/cm^2), LV elastance at end-systole derived by single beat (Ees$_{(sb)}$), LV circumferential end-systolic wall stress (ESWSc, g/cm^2), midwall shortening fraction (SFmid), predicted midwall fiber shortening for a measured fiber stress (midwall VCFc, circ/s) and MFS (g/cm^2). To assess the afterload, arterial elastance (Ea) was measured. LV end-systolic dimension (LVES), left ventricular end-diastolic dimension (LVED), end-systolic blood pressure (P_{es}) and LV wall thickness at end-systole (h_{es}) were also evaluated [8].

$$\mathrm{ESWSc} = [(1.35)(P_{es})(\mathrm{LVES}/(2h_{es})] \times [1 - (\mathrm{LVES})^2/2(L^2)]$$

where L stands for LV diastolic length; 1.35 is the conversion factor from mmHg to g/cm^2; P_{es} is calculated as 0.9 × systolic blood pressure [9]:

$$\mathrm{MFS}\,(\mathrm{g/cm^2}) = [(1.35)(P_{es})(b_m)]/(2h_{es})$$

where b_m stands for the midwall minor semi-axis at end-systole and computed as $b_m = h_{es}/\ln(\mathrm{LVES}/2 + h_{es}) - \ln(\mathrm{LVES}/2)$ [9, 10]

$$\text{midwall-VCFc} = 0.0007 \times (\mathrm{MFS}) + 0.65$$

$$\mathrm{WSM} = (1.35)(\mathrm{Ps})(\mathrm{LVES})(h_d)(1 + h_d/\mathrm{LVES})$$

where h_d is the left ventricular end-diastolic posterior wall thickness and Ps is the systolic arterial pressure [8]

$$\mathrm{SFmid} = [(\mathrm{LVED} + h_d/2 + s_d/2) - \mathrm{LVES} - \mathrm{mwst}] /(\mathrm{LVED} + h_d/2 + s_d/2)$$

where 'mwst' stands for left ventricular end-systolic inner shell myocardial thickness

$$\mathrm{mwst} = [(\mathrm{LVED} + (h_d + s_d)/2)^3 - (\mathrm{LVED}^3 + \mathrm{LVES}^3)^{0.333} - \mathrm{LVES}]$$

where s_d is the end-diastolic septal thickness.

LV end-systolic elastance (Ees) was calculated by a modified single-beat method developed by Chen *et al.* [11], employing Ps and Pd (diastolic arterial pressure), SV, EF and an estimated normalized ventricular elastance at arterial end-diastole.

$$\mathrm{Ees}_{(sb)} = [\mathrm{Pd} - (\mathrm{ENd(est)} \times \mathrm{Ps} \times 0.9)]/(\mathrm{ENd(est)} \times \mathrm{SV})$$

where ENd(est): non-invasive estimated normalized left ventricular elastance at the onset of ejection

$$\mathrm{ENd(est)} = 0.0275 - 0.165 \times \mathrm{EF} + 0.3656 \times (\mathrm{Pd/Pes}) + 0.515 \times \mathrm{ENd(avg)}$$

where ENd(avg) is the group-averaged normalized left ventricular elastance at the onset of ejection.ENd(avg) is given by a seven-term polynomial function: ENd(avg) = Σi = 0ai × tNdi where ai are (0.35695, −7.2266, 74.249, −307.39, 684.54, −856.92, 571.95, −159.1) for i = 0 to 7, respectively. The value of tNd was determined by the ratio of pre-ejection period (R wave → flow-onset) to total systolic period (R-wave → end-flow), with the time at onset and termination of flow defined noninvasively from the aortic Doppler waveform. Our study methodology was based on the equation developed by Chen *et al.* [11]. The equation of arterial elastance (Ea = ESP/SV) was used to calculate Ea, where ESP designates end-systolic pressure and computed as (ESP = 0.9 × Ps) [12].

HI = A/B where A stands for transverse diameter at the deepest level of deformity, and B for anterior–posterior diameter of the same level. The HI of <2.5 was considered as mild, HI = 2.5–3.2 as moderate and HI of >3.2 as severe deformity. The A and B diameters of our subjects with PE were calculated by computed tomography of the thorax.

Statistical analysis

Data were analysed by the Pearson correlation, Student's t-test and analysis of covariance (ANCOVA) test. The arithmetic mean of cardiac radiographic measurements of children with PE and children with no deformity was compared according to the Student's t-test. To investigate what percentage the parameters were affected by BSA, age and pectus disease (these cardiac parameters can be affected by age and the BSA), we used ANCOVA analysis. Cardiac parameters can be affected by age and the BSA, so these two factors were taken as covariate variables in multi-ANCOVA analysis. In ANCOVA analysis, pectus and the control group were fixed factors; the variables age and BSA were taken as covariates. R^2 and the adjusted R^2: R^2 assume that every single variable explains the variation in the dependent variable, whereas the adjusted R^2 tells us the percentage of variation explained only by the independent variables that actually affect the dependent variable. In children with PE, the HI arithmetic average and its correlation with the cardiac parameters were investigated.

RESULTS

Thirty-two children (23 male, 9 female) with PE deformity and 40 (17 male, 23 female) healthy controls were included in the study. The average age of the children (11.7 ± 3.2) and BSA average (1.31 ± 0.33) were similar in both groups (Table 1).

Radiological measurements were made of the cardiac parameters; the arithmetic averages of the SF mid, ESWSc, midwall

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Table 1: Cardiac radiographic measurements of children with pectus compared with the control group

Cardiac measurements	Groups		t[a]	Sig.[b]
	Pectus (n = 32) Mean (SD)	Control (n = 40) Mean (SD)		
Age	11.16 (3.15)	12.08 (3.21)	−1.215	0.228
BSA	1.248 (0.301)	1.359 (0.347)	−1.435	0.156
SFmid	0.322 (0.065)	0.379 (0.066)	−3.672	<0.001
WSM	1135.89 (397.92)	1356.16 (562.54)	−1.871	0.066
ESWSc	−10 748.01 (−9433.92)	−99 337.51 (−108 071.82)	5.095	<0.001
Midwall VCFc	0.671 (0.005)	0.679 (0.009)	−4.248	<0.001
MFS	30.27 (7.77)	40.88 (12.29)	−4.248	<0.001
Ea	1.636 (0.582)	1.691 (0.650)	−0.371	0.711
$Ees_{(sb)}$	2.218 (0.849)	2.170 (0.939)	0.225	0.822
LVED	4.237 (0.503)	4.227 (0.528)	0.082	0.935
LVES	2.644 (0.441)	2.763 (0.466)	1.100	0.275
P_{es}	95.875 (4.100)	93.617 (8.569)	1.418	0.162
h_{es}	0.366 (0.047)	0.382 (0.056)	1.093	0.279

SD: standard deviation; BSA: body surface area; SFmid: midwall shortening fraction; ESWSc: LV circumferential end-systolic wall stress; WSM: Meridional LV wall stress; midwall VCFc: predicted midwall fiber shortening for a measured fiber stress; MFS: myocardial fiber stress; Ea: arterial elastance; $Ees_{(sb)}$: left ventricular elastance at end-systole derived by single beat; LVED: left ventricular end-diastolic dimension ; LVES: left ventricular end-systolic dimension; P_{es}: end-systolic blood pressure ; h_{es}: LV wall thickness at end-systole.
[a]Independent Student's t-test.
[b]P-value statistically significant.

VCFc and MFS in the study group and in the control group were found to be statistically different in children, whereas WSM, Ea and $Ees_{(sb)}$, LVES, LVED, P_{es} and h_{es} were not significantly different (Table 1).

According to the ANCOVA analysis, age and BSA when taken as a covariate variable; SFmid, ESWSc, midwall VCFc and MFS were lower in children with PE than in the control group. The differences such as SFmid 14.9%; ESWSc 27.5%; midwall VCFc; 20.3% and MFS; 20.3% can be attributed to the pectus deformity. Ea (27.1%), $Ees_{(sb)}$ (18.9%), LVED (40.3%) and LVES (29.8%) were only affected by BSA (Table 2).

The HI minimum value of children with PE was 2.00, the maximum value was 4.93 and the arithmetic mean was 2.62 ± 0.56. Of the 32 patients, 14 (43.75%) had mild deformity, 15 (46.88%) had moderate and 3 (9.37%) had severe deformity. There was no correlation between cardiac data (ESWSc, midwall VCFc, MFS, Ea, $Ees_{(sb)}$, LVED, LVES, P_{es} and h_{es}) and HI (Table 3). However, when divided into two groups HI <2.5 was mild deformity and 2.5–3.2 was moderate deformity, the cardiac arithmetic mean of these measured data was not different (P > 0.05). Therefore, no further analysis associated with HI was undertaken.

DISCUSSION

The most common chest wall deformity is the PE due to an inward collapse of the sternum and the lower rib cartilage [13]. The cause is unknown; however, there is a family history of chest wall deformities in 40% of the cases. PE is more common in males and our study was consistent with the literature.

PE may be considered a trivial problem but it can lead to much more than a cosmetic deformity. It causes volume reduction and cardiac chest compression and can lead to a reduction in cardiopulmonary function and physical capacity. Symptoms rarely appear in early childhood and increase with age [14].

Because the chest wall is flexible in young patients, the heart can slide to the left and this provides a modest reduction in pressure on the heart. However, with age the chest wall elasticity decreases, stiffness increases, the left deviation of the heart decreases and pressure on the heart increases with consequent symptoms. During early adolescence, when patients begin to play sport, they complain of early fatigue. In addition, exertional dyspnoea, decreased endurance, chest pain, palpitations, exercise-induced wheezing and frequent upper respiratory infections may occur. In our patients, the most frequent complaint was chest pain. As shown in the literature, the complaints were found to increase as the patients aged.

Thoracic skeletal abnormalities have been shown to have a close association with MVP [15]. In their study, Seliem *et al.* [16] found the prevalence of MVP in 55% of patients with PE, whereas Simsek *et al.* [17] found the figure to be 12.5%. In our study, the prevalence of MVP was lower (6.25%).

Congenital heart disease is relatively rare in patients with PE. In the study by Simsek *et al.* [17], 2 (2.1%) patients had atrial septal defect (ASD) and 12 (12.5%) had patent foramen ovale. In our study, 4 patients (12.5%) had ASD.

Right ventricular (RV) dysfunction can be seen in patients with PE. Ventricles share common septum and are within the same pericardial cavity. This relationship between both ventricles also causes similar changes in both systole and diastole [18]. In these children, the compression of the right side of the heart can also cause changes in the size and function of the left ventricle, resulting in a decrease in cardiovascular function. Hu *et al.* [19] looked at children with PE between 2.5 and 16 years; they determined the cardiac function by calculating left ventricular ejection fraction (LVEF), SF and the SV index. Gürsu *et al.* [20] evaluated LV heart muscle contraction capacity and found lower LVEF in the PE group than in the healthy control group. In the same study, an inverse relationship was found between LVEF and SF with HI. Krueger *et al.* [21] have shown that in transoesophageal echocardiography, the

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Table 2: The cardiac radiographic measurements were analysed by ANCOVA according to age versus groups

Cardiac measurements (dependent variables)	Independent variables	*F*	Sig.	Partial η^2	R^2	Adj. R^2
SFmid	Age	0.240	0.626	0.004		
	BSA	0.000	0.990	0.000	0.173	0.137
	Groups	11.925	0.001	0.149		
WSM	Age	8.032	0.006	0.106		
	BSA	51.570	<0.001	0.431	0.625	0.599
	Groups	1.465	0.230	0.021		
ESWSc	Age	8.348	0.005	0.112		
	BSA	36.459	<0.001	0.356	0.594	0.576
	Groups	25.036	<0.001	0.275		
Midwall VCFc	Age	3.259	0.075	0.046		
	BSA	20.871	<0.001	0.235	502	0.480
	Groups	17.353	<0.001	0.203		
MFS	Age	3.259	0.075	0.046		
	BSA	20.871	<0.001	0.235	0.502	0.480
	Groups	17.353	<0.001	0.203		
Ea	Age	0.976	0.327	0.014		
	BSA	25.219	<0.001	0.271	0.519	0.498
	Groups	3.856	0.054	0.054		
$Ees_{(sb)}$	Age	0.090	0.765	0.001		
	BSA	15.802	<0.001	0.189	0.459	0.435
	Groups	0.973	0.327	0.014		
LVED	Age	1.228	0.272	0.018		
	BSA	45.952	<0.001	0.403	0.676	0.662
	Groups	4.640	0.035	0.064		
LVES	Age	0.141	0.708	0.002		
	BSA	28.932	<0.001	0.298	0.617	0.600
	Groups	0.001	0.972	0.000		
P_{es}	Age	0.162	0.688	0.003		
	BSA	0.637	0.428	0.011	0.039	0.009
	Groups	1.794	0.186	0.029		
h_{es}	Age	0.285	0.596	0.005		
	BSA	1.548	0.218	0.026	0.066	0.018
	Groups	0.771	0.384	0.013		

Groups: Children with pectus and normal children.
ANCOVA test: Analysis of covariance. Cardiac parameters were added as dependent variables those can be affected by age and the BSA, so these two factors were taken as covariate variables to the ANCOVA analysis.
Adj. R^2: Adjusted R^2; BSA: body surface area; SFmid: midwall shortening fraction; ESWSc: LV circumferential end-systolic wall stress; WSM: meridional LV wall stress; midwall VCFc: predicted midwall fiber shortening for a measured fiber stress; MFS: myocardial fiber stress; Ea: arterial elastance; Ees(sb): left ventricular elastance at end-systole derived by single beat; LVED: left ventricular end-diastolic dimension ; LVES: left ventricular end-systolic dimension; P_{es}: end-systolic blood pressure; h_{es}: LV wall thickness at end-systole.

RV end-diastolic size and LVEF were increased after surgery. Bawazir *et al.* [22] have shown that LV cardiac output and cardiac index improve after pectus corrective surgery. Peterson *et al.* [23] documented improvements in RV end-diastolic volume and right ventricular ejection fraction (RVEF) and also increments in LV end-diastolic volume index and SV index after pectus surgery. Saleh *et al.* [24] found that both LVEF and RVEF were lower in PE patients. RV end-systolic volume was significantly higher and they did not detect any significant correlation between the RVEF and LVEF and HI.

The ventricular contractility and myocardial performance may be affected by chamber geometry which needs to be identified by measuring ESWSc, midwall VCFc and MFS. ESWS, which is dependent on both chamber shape and mass/volume ratio, demonstrates the forces opposing predominantly meridional and circumferential planes. This is an index of the total forces per unit of myocardium and thus may cause an underestimation of true afterload. MFS as representative of myofibre afterload is a more accurate index of afterload in hypertrophied or dilated LV [8]. We found ESWSc, MFS and midwall VCFc to be lower in patients with PE. This means that myocardial contractility and afterload may be affected and lower and causes decreased systolic function in patients with PE. SFmid as a systolic ejection index of deeper layers of myocardium provides more physiologically appropriate measurements of LV in wall thickness and conditions such as LV concentric hypertrophy and provides information to assess the myocardial performance [8]. In this study, SFmid was lower in patients with PE than in controls.

$Ees_{(sb)}$ is a major determinant of cardiac systolic function reflecting LV contractility. Ea, as a representative of arterial loading properties, is a more accurate parameter to assess arterial load on ventricular performance [12]. Ea reflects afterload and is sensitive to any kind of afterload changes such as blood pressure. We did not find any differences between controls and patients with PE with regard to Ea and $Ees_{(sb)}$. The possible reason for this is that Ea and Ees were affected by BSA and not pectus deformity as depicted in Table 2.

Paediatric echocardiographic evaluations of LV systolic function are usually based on the indices obtained by measurements at the endocardial level. It is suggested that in the presence of ventricular

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Table 3: The correlation of the cardiac parameters with HI in children with pectus excavatum

	HI	Age	BSA
Age	−0.007		
BSA	0.107	0.920[a]	
SFmid	−0.147	−0.235	−0.227
WSM	−0.035	0.688[a]	0.797[a]
ESWSc	0.116	−0.638[a]	−0.679[a]
Midwall VCFc	0.051	0.601[a]	0.696[a]
MFS	0.051	0.601[a]	0.696[a]
Ea	−0.148	−0.599[a]	−0.643[a]
$Ees_{(sb)}$	−0.151	−0.562[a]	−0.608[a]
LVED	−0.164	0.714[a]	0.779[a]
LVES	−0.099	0.746[a]	0.776[a]
P_{es}	−0.153	0.209	0.230
h_{es}	−0.273	0.076	0.025

Cardiac parameters can be affected by age and the BSA, so these two factors were also taken to the correlation analysis.
HI: Haller index; BSA: body surface area; SFmid: midwall shortening fraction; ESWSc: LV circumferential end-systolic wall stress; WSM: meridional LV wall stress; midwall VCFc: predicted midwall fiber shortening for a measured fiber stress; MFS: myocardial fiber stress; Ea: arterial elastance; $Ees_{(sb)}$: left ventricular elastance at end-systole derived by single beat; LVED: left ventricular end-diastolic dimension ; LVES: left ventricular end-systolic dimension; P_{es}: end-systolic blood pressure ; h_{es}: LV wall thickness at end-systole.
[a]Correlation is significant at the 0.01 level (two-tailed).

hypertrophy, the evaluation of LV systolic function based on indices obtained by measurements at the endocardial level may lead to an overestimation of systolic function. Crepaz *et al.* [25] assessed the developmental changes of LV systolic mechanics measured at the endocardial and midwall levels. They found that blood pressure, LV afterload, volume and mass increased, whereas the mass/volume ratio remained stable during growth. FS and mean VCFc at the endocardial level decreased and showed an inverse relation to afterload. SFmid and VCFc were lower during the first months and did not change during the first year of life. They concluded that LV volume and left ventricular mass (LVM) increase with age, mass/volume ratio remains almost constant while afterload increases. Endocardial systolic function indices are higher in the first stage of life.

Since there are no studies in the literature evaluating cardiac and arterial elastance and myocardial wall stress in children with PE, we could not compare our results with other studies.

In conclusion, the results of our study indicate that systolic function, contractility and afterload are modified in patients with PE.

Study limitations

In this current study, most of the children had mild or moderate forms of PE. Further studies with a larger sample size and greater number of children with a more severe form of PE are warranted to better elucidate the cardiac functions in these patients.

Conflict of interest: none declared.

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