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Arterial and left ventricular end-systolic elastance in normal children

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Abstract. – OBJECTIVE: Assessing the left ventricular (LV) functions in Pediatric Intensive Care Unit, and those pediatric patients with compromised ventricular performance or enhanced systolic or diastolic load e.g. congestive heart failure, hypertension, dilated/hypertrophic cardiomyopathies is a real challenge. Currently used noninvasive methods fail giving quantitative measures to asses cardiac performance and do not allow evaluation of ventriculo-arterial interaction. Non-invasive method of cardiovascular performance determination by measuring left ventricle end-systolic elastance (Ees), arterial elastance (Ea) and the ventriculoarterial coupling (VAC), though interaction between LV and arterial network, is possible.

PATIENTS AND METHODS: Hundred and forty two otherwise normal children (1 week to 17 years old) were randomly selected. Routine transthoracic echocardiographic and Doppler studies were carried out by an experienced pediatric cardiologist. The results have been evaluated statistically.

RESULTS: We found that the Ea and Ees(sb) show powerful negative correlation with BSA (r = -0.65, -0.72 respectively) of the children.

CONCLUSIONS: We suggest that this simple measurement method may be applied at bedside to evaluate ventricular performance of the children.

Key Words:
Arterial elastance, Cardiac performance, Children, Heart failure, Left ventricular elastance.

Introduction

Assessing the left ventricular (LV) functions in Pediatric Intensive Care Unit, and those pediatric patients with compromised ventricular performance or enhanced ventricular systolic or diastolic load e.g. congestive heart failure, hypertension, dilated/hypertrophic cardiomyopathies, is a real challenge. The LV and arterial functions are influenced by all these clinical conditions. It is known that the LV performance is affected by the arterial pressure and volume load which simply can be indicated as effective arterial elastance (Ea)[1,2]. In fact, interaction between LV and Ea plays an important role in determining cardiovascular performance. Sunagawa et al[1,2], Kelly et al[3] showed that Ea as arterial load, interacts with core elements like arterial compliance, impedance, peripheral vascular resistance and cardiac systolic and diastolic intervals. Bedside quantitative evaluation of LV performance and ventriculo-arterial coupling (VAC) may help clinicians to assess the effectiveness of ongoing treatment. A noninvasive validated method developed by Chen et al[4] allows estimating left ventricular elastance at end-systole derived by single-beat technique in humans ($Ees_{(sb)}$), the major determinant of left ventricular systolic performance. Ea may be computed using the formula ESP = 0.9 × Systolic blood pressure, where the ESP stands for end systolic pressure[3]. Chen et al[4] found that the calculation of ESP from 0.9 × brachial systolic blood pressure reasonably approximated ESP measured invasively.

Currently used noninvasive methods fail giving quantitative measures to asses cardiac performance and do not allow evaluation of ventriculo-arterial interaction. Non-invasive method of cardiovascular performance determination by measuring left ventricle end-systolic elastance (Ees), arterial elastance (Ea) and the VAC, though interaction between LV and arterial network, is possible. The aim of this study is to report noninvasively measured normal values of Ea, left ventricular elastance at $Ees_{(sb)}$ and VAC among normal children.

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Patients and Methods

Study Design and Population

After approval of Ethics Committee of the institution, the study population was randomly selected from individuals of the Outpatient Clinic of our Department of Pediatric Cardiology. After complete physical examination, and taking an ECG, and color Doppler transthoracic echocardiography (TTE), normal individuals were included into the study. TTE applied by an experienced pediatric cardiologist. Individuals with history of congenital or acquired cardiac, pulmonary, renal diseases and history of diabetes, hypertension, obesity or other systemic diseases were excluded. Individuals with hypertrophic septum or dilated cardiac cavities, compromised ejection fraction or newborns with high pulmonary vascular resistance, any shunt, increased pulmonary flow velocity or any valvular stenosis or regurgitation documented by TTE were also excluded. A total of 142 otherwise normal children were randomly selected. The selected patients were divided into groups, based on their age and body surface area (BSA).

Measurements and Study Protocol

1. Brachial systolic (Ps) and diastolic (Pd) blood pressures were recorded using bilateral triplicate measurements on a rested subject using a validated oscillometric device in supine position.
2. Ejection fraction (EF) was calculated using the standard dimension cubed formula EF = $(LVDD^3\text{-}LVDS^3)/LVDD^3$, where LVDD and LVDS stands for left ventricular dimension in diastole and systole respectively.
3. LV outflow tract (LVOT) diameter was measured at the base of aortic leaflet at parasternal long axis view in echocardiography.
4. Time velocity integral for aortic valve (VTI_{Ao}) were obtained with continues wave Doppler immediately below the aortic valve in the apical long axis view.
5. Using LVOT and VTI_{Ao}, the stroke volume (SV) were calculated as: $SV = (LVOT/2)^2 \times VTI_{Ao} \times 3.141$.
6. Aortic Doppler views were used to calculate the time intervals. The pre-ejection period (PEP), i.e. the time interval from Q wave of ECG to the onset point of aortic Doppler flow, and the Q-T offset interval, i.e. the time interval from Q wave of ECG to the offset point of aortic Doppler flow.
7. To compute $Ees_{(sb)}$, the equation developed by Chen et al. (3), was used: $Ees_{(sb)} = [P_d - (E_{Nd(est)} \times P_s \times 0.9)]/(E_{Nd(est)} \times SV)$, where $E_{Nd(est)}$ is noninvasive estimated normalized elastance at the onset of ejection and was calculated as, $E_{Nd(est)} = 0.0275 - 0.165 \times EF + 0.3656 \times (Pd/Pes) + 0.515 \times E_{Nd(avg)}$, where $E_{Nd(avg)}$ is group-averaged normalized left ventricular elastance at the onset of ejection given by a seven-term polynomial function: $E_{Nd(avg)} = ai_{i=0}\ t_{Nd}^{i}$, 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. More detail can be found in report of Chen, et al[1].
8. The equation of Ea = ESP/SV was used to calculate Ea[3], where ESP designates end-systolic pressure and computed as ESP = 0.9 × Ps.

Statistical Analysis

Data analyses were carried out by IBM SPSS Statistics™ and Microsoft Excel™ programs. The parameters required to compute Ea and $Ees_{(sb)}$ were used as dependent variables, whereas age (month) and body surface area (BSA) accepted as independent variables. Mosteller formula[5], [Height(cm) × Weight (kg) ÷ 3600)^{½}] were used to calculate BSA. As the first step, multiple regression analyses were conducted between every single measurement and age and BSA. Regarding significance of age or BSA the grouping was structured. The BSA based grouping, was assessed with 0.1 m^2 intervals. The mean and 95% Confidence Interval (95% CI) of each variable were calculated. Beginning with first group, all the groups overlapped within 95% CI were unified and re-grouped. The differences between groups were analyzed by ANOVA. The homogeneity between variances of the groups was tested by Levene's test. Post ANOVA testing was done thru Tukey HSD among those groups with homogenous variances and Tamhane's T2 among those with nonhomogenous variances. $p < 0.05$ was considered as statistically significant.

Calculating the Percentiles

After converting to normal standard distribution, mean of 0 (zero) and standard deviation (SD) of 1 (one), Z scores (z = (×-mean)/standard deviation), BSA based groups of Ea and $Ees_{(sb)}$ values were calculated. Then the standard normal probability density function [$f(x) = ((1/(2\pi)^{1/2}) \times$

$e^{-(x*x/2)}$] matched with Microsoft Excel© software in order to computing percent probability (NORMSDIST(z-score) × 100). The obtained percentages, grouped in 5 percentile intervals and the percentiles were formed with mean of Ea and $Ees_{(sb)}$ values of each group. Because there was any statistically significant relation between $Ea/Ees_{(sb)}$ and BSA and age, percentiles of $Ea/Ees_{(sb)}$ values not calculated.

Results

142 individuals, M/F: 75/67, were studied. Age of children was from 8 days to 16 years (192 months). 24.6% of children were under one year of age, 17.6% between 1-< 5 years, while 9.9% were over 15 years of age (Table I). Correlation between **a**ge (month), BSA (m^2), Ea, $Ees_{(sb)}$ and the parameters required to compute Ea, $Ees_{(sb)}$ is given in Table II. There was any statistically significant relation between gender and mean values of Ea (t = 0.16, p = 0.87) and $Ees_{(sb)}$ (t = 0.64, p = 0.52). A very strong positive linear correlation (r = 0.89) between Ea and $Ees_{(sb)}$ and negative correlation with other parameters were found. The parameters which showed negative correlation with Ea were; LVOT (r = -0.77), SV (r = -0.75), Q-T offset (r = -0.70) and VTIAo (r = -0.70), and those which showed negative correlation with $Ees_{(sb)}$ were; LVOT (r = -0.85), SV (r = -0.82), Q-T offset (r = -0.75), VTIAo (r = -0.70) and PEP (r = -0.60). Although there was a strong positive correlation between LVOT and age (r = 0.91) and BSA (r = 0.92), the relationship between LVOT and age found not significant in multiple regression analysis. Based on this finding, the BSA based mean values of LVOT were compared (Table II). By increasing BSA, mean LVOT values increases and this was different among the groups (Tukey HSD, p < 0.05) (Table II)

Although Ea and $Ees_{(sb)}$ showed powerful negative correlation with age (r = -0.62, -0.69 respectively) and BSA (r = -0.65, -0.72 respectively), multiple regression analysis revealed any significant relation with the age and Ea. Because of the latter finding, grouping of Ea and $Ees_{(sb)}$ was done according to BSA (Table III). 42% of changing of Ea (R^2 = 0.42), and 51% changing of $Ees_{(sb)}$ (R^2 = 0.51) were explained with age and BSA, but the effect of age was not significant. Every 1 unite increment in BSA led to 8.71 mmHg enhancement of Ea (BSA 0.1 m^2, Ea 0.87 mmHg/ml), and 9.75 mmHg/ml decrement of $Ees_{(sb)}$ (BSA 0.1 m^2, $Ees_{(sb)}$ 0.97 mmHg/ml). The lower the BSA, the higher Ea and $Ees_{(sb)}$ mean values. The Ea and $Ees_{(sb)}$ mean values showed statistically significant difference among BSA groups (Table III).

The 5, 10, 25, 50, 75, 90 and 95 percentiles of Ea and $Ees_{(sb)}$ values standardized by BSA were computed (Table IV). Because there was any statistically significant relationship between $Ea/Ees_{(sb)}$ and BSA and age (Tables II, III), percentiles of $Ea/Ees_{(sb)}$ were not calculated. There was a moderate positive correlation between

Table I. Demographic properties distribution of the age groups.

Age groups (months)	n (%)	Height (cm) mean (Sd)	Weight (kg) mean (Sd)	BSA (m^2)[a] mean (Sd)
0-< 1	7 (4.9)	50.14 (2.04)	3.51 (0.47)	0.22 (0.02)
1-< 3	13 (9.2)	55.62 (3.25)	4.73 (1.10)	0.27 (0.04)
3-< 6	9 (6.3)	63.78 (2.22)	6.99 (0.86)	0.35 (0.03)
6-< 12	6 (4.2)	71.75 (2.82)	8.92 (0.73)	0.42 (0.02)
12-< 36	9 (6.3)	85.78 (7.08)	11.72 (1.84)	0.53 (0.06)
36-< 60	16 (11.3)	99.34 (7.59)	15.86 (3.16)	0.66 (0.09)
60-< 84	21 (14.8)	112.57 (6.27)	19.79 (3.15)	0.79 (0.08)
84-< 108	19 (13.4)	126.11 (7.53)	25.96 (7.28)	0.95 (0.15)
108-< 144	12 (8.5)	141.42 (9.22)	35.23 (7.49)	1.17 (0.15)
144-< 180	16 (11.3)	157.88 (11.37)	50.48 (8.49)	1.48 (0.17)
180-192	14 (9.9)	161.93 (8.20)	55.42 (8.67)	1.58 (0.15)
Total	**142 (100.0)**	**110.49 (37.38)**	**24.49 (17.89)**	**0.85 (0.45)**

Sd: Standard deviation.
[a]Mostelle $BSA\ (m^2) = \sqrt{\frac{\text{Body weigh (kg)} * \text{Body height (cm)}}{3600}}$

Table II. Correlation between age (month), BSA (m^2) and the parameters required to compute Ea and $Ees_{(sb)}$.

Variables	Age (month)	BSA (m^2)	Ea (mmHg/ml)	Ees(sb) (mmHg/ml)	Ea/ $Ees_{(sb)}$	PEP (msec)	Q-T offset interval	LVOT (cm)	VTI_{Ao} (cm)	Ps (mmHg)	Pd (mmHg)	EF (%)	SV (ml)
Age (month)	1	.975**	-.622**	-.694**	-.065	.690**	.743**	.912**	.530**	.575**	.558**	.132	.903**
BSA (m^2)	.975**	1	-.653**	-.720**	-.070	.667**	.728**	.921**	.551**	.591**	.576**	.101	.908**
Ea (mmHg/ml)	-.622**	-.653**	1	.894**	.417**	-.482**	-.696**	-.765**	-.703**	-.484**	-.373**	-.142	-.745**
$Ees_{(sb)}$ (mmHg/ml)	-.694**	-.720**	.894**	1	.043	-.601**	-.748**	-.848**	-.704**	-.490**	-.412**	-.064	-.816**
Ea/$Ees_{(sb)}$	-.065	-.070	.417**	.043	1	.228**	-.121	-.085	-.227**	-.052	-.014	-.269**	-.136
PEP (msec)	.690**	.667**	-.482**	-.601**	.228**	1	.724**	.630**	.372**	.389**	.387**	.004	.580**
Q-T offset (msec)	.743**	.728**	-.696**	-.748**	-.121	.724**	1	.778**	.586**	.410**	.353**	.021	.771**
LVOT (cm)	.912**	.921**	-.765**	-.848**	-.085	.630**	.778**	1	.580**	.603**	.567**	.117	.964**
VTI (cm)	.530**	.551**	-.703**	-.704**	-.227**	.372**	.586**	.580**	1	.371**	.357**	.113	.661**
Ps (mmHg)	.575**	.591**	-.484**	-.490**	-.052	.389**	.410**	.603**	.371**	1	.672**	.087	.616**
Pd (mmHg)	.558**	.576**	-.373**	-.412**	-.014	.387**	.353**	.567**	.357**	.672**	1	.042	.561**
EF (%)	.132	.101	-.142	-.064	-.269**	.004	.021	.117	.113	.087	.042	1	.158
SV (ml)	.903**	.908**	-.745**	-.816**	-.136	.580**	.771**	.964**	.661**	.616**	.561**	.158	1
N	142	142	142	142	142	142	142	142	142	142	142	142	142

BSA: Body surface area; Ea: arterial elastance; $Ees_{(sb)}$: left ventricular elastance at end-systole derived by single-beat; PEP: Pre-ejection period, LVOT: Left ventricle outflow tract, Ps: Systolic blood pressure; Pd: Daiastolic blood pressure; EF: Ejection fraction; SV: Stroke volume; N: Number of children; **Correlation is significant at the 0.01 level (2-tailed); *Correlation is significant at the 0.05 level (2-tailed).

VTI_{Ao} and the age (r = 0.53) and BSA (r = 0.55) (Table II), the relationship between VTI_{Ao} and age found to be pretty weak significant in multiple regression analysis. Due to this finding the mean values of VTI_{Ao} were compared by BSA (Table V). Any increasing in BSA, led to increase the mean value of VTI_{Ao}, but difference between group 2 and 3, and the group 5, 6 and 7 was found statistically not significant (Tukey HSD, $p > 0.05$). There was a very strong positive correlation between SV and age (r = 0.90) and BSA (r = 0.91) (Table II), the relation between SV and age showed a very weak significance in multiple regression analysis. The mean values of SV and BSA increasing together. But the difference between group 2 and 3, and the group 6 and 7 was not statistically significant (Tamhane's T2, $p > 0.05$) (Table V). There was a significant positive correlation between PEP and Q-T offset interval and age (r = 0.69, 0.74 respectively) and BSA (r = 0.67, 0.73 respectively) (Tables II, VI), but the relationship with BSA was found not significant in multiple regression analysis ($p > 0.05$). Due to this finding PEP and Q-T offset intervals were grouped by age. The PEP mean values of groups 1, 2 and 3 was not different ($p < 0.05$), but statistically significant between other groups (Tukey HSD, $p < 0.05$). The Q-T offset interval values were not statistically different between groups 2 and 3, and the groups 5 and 6 (Tukey HSD, $p < 0.05$), but significant between other groups (Tukey HSD, $p < 0.05$). There was no correlation between EF and age (r = 0.13) and BSA (r = 0.10) ($p > 0.05$) either (Table II). There was very strong positive correlation between LVOT and SV (r = 0.96) and Q-T offset interval (r = 0.78), and between VTI_{Ao} and SV (r = 0.66) (Table II).

Discussion

Assessment of the LV systolic function remains as a key point during and treatment, observation and assessment of the therapeutic interventions of different diseases like hypertension, heart failure, cardiomyopathies, repaired congenital heart diseases with compromised LV functions and other conditions concerning LV functions. We agree with Kovács[6] that we are often confronted with making decision regarding assessment of LV systolic function. Invasive methods are not practical for routine assessment of LV performance.

Table III. Ea and $Ees_{(sb)}$'s means distribution of the BSA (m^2) groups.

Groups	BSA[a] groups (m^2)	n	Ea* (mmHg/ml) mean (95% CI)	$Ees_{(sb)}$* (mmHg/ml) mean (95% CI)	$Ea/Ees_{(sb)}$ mean (95% CI)
1	0.0000-0.3999[a]	30	10.50 (8.45-12.55)	13.65 (11.69-15.62)	0.77 (0.67-0.88)
2	0.4000-0.4999	8	6.42 (5.40-7.45)	9.80 (8.44-11.15)	0.66 (0.61-0.70)
3	0.5000-0.6999	18	3.93 (3.25-4.61)	5.58 (4.54-6.61)	0.75 (0.60-0.90)
4	0.7000-0.9999	40	2.39 (2.26-2.52)	3.51 (3.31-3.72)	0.69 (0.66-0.73)
5	1.0000-1.9999	46	1.86 (1.78-1.95)	2.65 (2.54-2.75)	0.71 (0.67-0.75)
	Total	**142**	**4.35 (3.65-5.06)**	**5.99 (5.16-6.82)**	**0.72 (0.69-0.75)**
Levene's test			$p < 0.001$	$p < 0.001$	
ANOVA			F = 59.59. $p < 0.001$	F = 98.74. $p < 0.001$	F = 1.22. $p = 0.306$
Post Hoc (Tamhane's T2)			$p < 0.01$	$p < 0.01$	
Regression model			R = 0.66. $R^2 = 0.42$	R = 0.72. $R^2 = 0.51$	
Regression ANOVA			F = 52.48. $p < 0.001$	F = 75.24. $p < 0.001$	$p > 0.05$
Predictors (BSA. Age)			β = -8.714**, β = 0.019	β =-9.752**, β = 0.013	

[a]3 children with BSA less than 0.2 m^2. Ea: arterial elastance. $Ees_{(sb)}$: left ventricular elastance at end-systole derived by single-beat. 95% CI: 95% Confidence Interval. Levene test: Test of homogeneity of variances. β: Unstandardized Coefficients. R: multiple correlation coefficient. R^2: coefficient of determination; **Significant at the 0.01 level. *Significant at the 0.05 level.

Left ventricular end-systolic elastance (Ees) is a major determinant of cardiac systolic function reflecting LV contractility. Although Chen et al did not evaluate the load-sensitivity of $Ees_{(sb)}$ while proposing noninvasive single-beat determination of left ventricular end-systolic elastance in humans[4] but they declared that some other invasive investigations of the $Ees_{(sb)}$ method have demonstrated the lack of loading influences[7].

Determination of Ees generally needs invasive measurements of LV pressure and volumes recorded over the period of cardiac loading. Ea, as representative of arterial loading properties, rather than the mean arterial resistance, is more accurate parameter to assess arterial load on ventricular performance[3]. Ea reflects afterload and sensitive to any kind of afterload changes such as blood pressure. By the concept described by Sunagawa et al[1], Ees represents ventricular properties whereas Ea represents arterial loading properties. Ea/Ees ratio used as an index for assessment of cardiovascular performance and cardiac energetics and is frequently used in clinical evaluation and represents left ventricular efficiency[8-10]). Since all three parameters reflect different aspects of left ventricle hemodynamics, all should be used in LV performance assessment accordingly. We agree with Chantler and Lakatta[11] that examination of the alterations in VAC with disease can yield mechanistic insights into

Table IV. Ea and $Ees_{(sb)}$'s standard normal distribution of the BSA (m^2) groups.

Groups	BSA (m^2)	n = 142	mmHg/ml	Percentiles 5	10	25	50	75	90	95	Mean (95% CI)
1	< 0.4	30	Ea	3.98	4.78	7.33	10.87	14.76	17.71	24.95	10.5 (8.45-12.55)
			$Ees_{(sb)}$	6.34	7.48	10.24	13.9	17.96	21.87	23.75	13.65 (11.69-15.62)
2	0.4-< 0.5	8	Ea	4.85	5.05	5.75	6.51	7.42	8.19	8.25	6.42 (5.40-7.45)
			$Ees_{(sb)}$	7.35	7.84	8.87	10.01	11.2	12.12	12.42	9.80 (8.44-11.15)
3	0.5-< 0.7	18	Ea	2.56	2.60	3.05	4.02	4.95	5.95	7.66	3.93 (3.25-4.61)
			$Ees_{(sb)}$	2.86	2.96	4.34	5.6	7.02	8.25	9.77	5.58 (4.54-6.61)
4	0.7-< 1.1	40	Ea	1.79	1.88	2.15	2.43	2.70	2.97	3.41	2.39 (2.26-2.52)
			$Ees_{(sb)}$	2.60	2.75	3.13	3.56	3.96	4.45	4.85	3.51 (3.31-3.72)
5	≥ 1.1	46	Ea	1.36	1.54	1.70	1.93	2.07	2.28	2.40	1.86 (1.78-1.95)
			$Ees_{(sb)}$	2.10	2.27	2.45	2.67	2.91	3.20	3.44	2.65 (2.54-2.75)

Ea: arterial elastance; $Ees_{(sb)}$: left ventricular elastance at end-systole derived by single-beat; 95% CI: 95% Confidence Interval.

Table V. LVOT, VTIAo and SV's means distribution of the BSA (m^2) groups.

Groups	BSA (m^2)	n	Mean (95% confidence interval)		
		155	LVOT (cm)	VTI_{Ao} (cm)	SV (ml)
1	< 0.3	16	0.76 (0.72-0.81)	15.36 (13.44-17.29)	7.20 (6.03-8.36)
2	0.3-< 0.4	14	0.89 (0.84-0.95)	18.03 (16.41-19.64)	11.53 (9.79-13.27)
3	0.4-< 0.5	8	1.02 (0.93-1.11)	18.06 (15.41-20.72)	14.58 (12.38-16.77)
4	0.5-< 0.7	18	1.25 (1.18-1.32)	21.45 (19.95-22.95)	27.01 (23.55-30.47)
5	0.7-< 1.0	40	1.47 (1.43-1.51)	23.20 (22.28-24.12)	39.30 (37.36-41.24)
6	1.0-< 1.4	20	1.68 (1.62-1.74)	23.31 (22.28-24.34)	52.48 (48.74-56.22)
7	≥ 1.4	26	1.83 (1.78-1.89)	23.37 (22.01-24.74)	56.81 (53.37-60.25)
	Total	142	1.38 (1.31-1.44)	21.34 (20.66-22.02)	35.06 (31.96-38.16)
Levene's test			$p = 0.358$	$p = 0.514$	$p < 0.001$
ANOVA			F = 205.11, $p < 0.001$[a]	F = 20.93, $p < 0.001$[a]	F = 178.53, $p < 0.001$ b
Regression model			R = 0.92, $R^2 = 0.85$	R = 0.55, $R^2 = 0.30$	R = 0.91, $R^2 = 0.83$
Regression ANOVA			F = 401.9, $p < 0.001$	F = 30.51, $p < 0.001$	F = 341.41, $p < 0.001$
Predictors (BSA. Age)			β = 0.545**, β = 0.002	β = 6.327**, β = -0.010*	β = 23.139**, β = 0.106*

LVOT: left ventricular outflow tract; VTI_{Ao}: Time velocity integral for aortic valve; SV: stroke volume; 95% CI: 95% Confidence Interval. [a]Post hoc Tukey HSD: Homogeneity of variances. [b]Post hoc Tamhane's T2 test: Not homogeneity of variances. **Significant at the 0.01 level. *Significant at the 0.05 level.

the pathophysiology of the conditions and helps to increase the effectiveness of ongoing therapeutic interventions.

Chen et al[4] introduced an objective, quantitative and validated, reproducible noninvasive method for evaluation of LV systolic function based on $Ees_{(sb)}$ measurement. Chen et al[4] reported that the majority of absolute discrepancies between non-invasive estimated $Ees_{(sb)}$ and the invasive "goal-standard" value were smaller than 0.6 mm Hg/ml. They found that the $Ees_{(sb)}$ typically ranges between 2.0 mmHg/ml in normal adult heart. In failing adult heart, as cardiac ejection fraction decrease, a three to fourfold increase is observed in Ea/Ees ratio, i.e. between 0.5 and 1.2, comparing with normal adults[12].

Our data revealed that Ea and $Ees_{(sb)}$ decreasing constantly by increasing BSA (Figure 1), where Ea and $Ees_{(sb)}$ values tend to be as high as 10.50 (8.45-12.55) and 13.65 (11.69-15.62) respectively in children with BSA of less than 0.4 m^2, while as low as 1.86 (1.78-1.95) and 2.65 (2.54-2.75) respectively in children with BSA of 1-2 m^2 (Table IV). The VAC remains almost unchanged in normal children with different age groups and different BSA [0.72 (0.69-0.75, mean

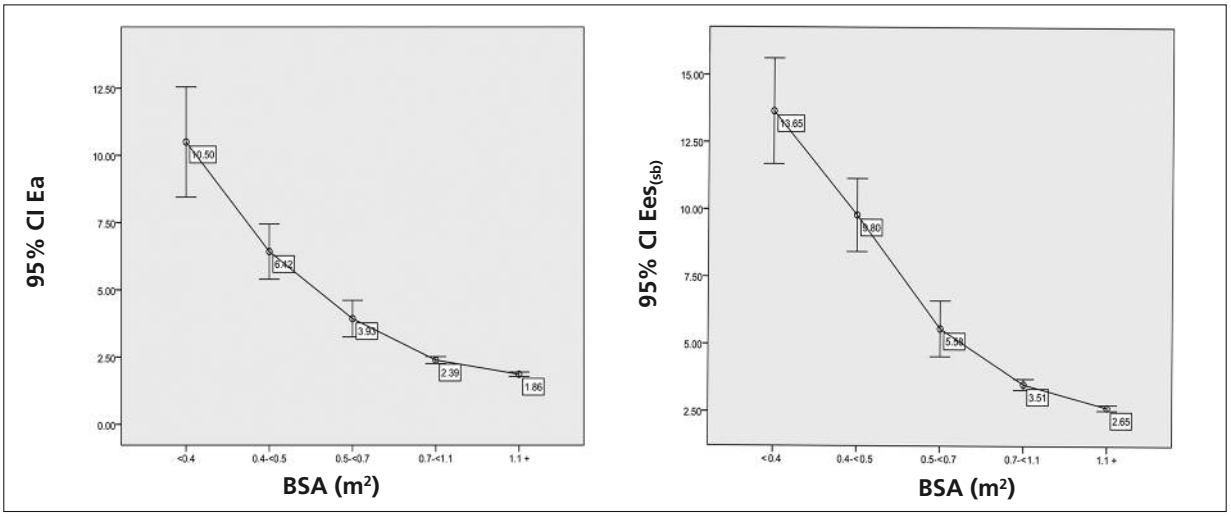

Figure 1. Ea and $Ees_{(sb)}$ decline stepwise while children grown up and body surface area increase.

95% CI] (Table III). Kiani and Shakibi[13] showed that the $Ees_{(sb)}$ tends to decline by age in normal children. Harada et al[14] studied obesity related arterioventricular stiffening in children and reported that stiffness increased by increasing BSA while the VAC remains without significant changes. The VAC ratio reported to be lower among obese children than normal control group of individuals (0.73 ± 3.2 vs 0.47 ± 0.15)[15]. Any change in afterload may increase Ea and VAC ratio. Engel et al[16] reported that a trend toward a lower arterial elastance and a higher left ventricular contractility in children with Still's murmur tends to lower VAC ratio in these children than in those without Still's murmur. Aortic root diameter shows significant negative correlation with Ea and $Ees_{(sb)}$. The conditions causing any increase LVOT and VTI_{Ao}, may decrease $Ees_{(sb)}$ significantly and lead to increase the VAC. It is also correct that diminished LV end-systolic and end-diastolic volume difference in clinical conditions like congestive heart failure, dilated cardiomyaopahties, restrictive pericarditis decrease the EF and lower $Ees_{(sb)}$ value and increase of VAC ratio if Ea value remains constant. Our data showed that by increasing the age, Q-T offset and PEP intervals increase significantly and lead to decrease in $Ees_{(sb)}$, but the VAC ratio remains with minimal changes.

The left ventricular end-systolic elastance and arterial elastance can be compute noninvasively. By running the current study we aimed to define normal values of Ea, $Ees_{(sb)}$ and $Ea/Ees_{(sb)}$ coupling among normal children of different age groups.

Conclusions

We suggest that this simple measurement method may be applied at bedside to evaluate ventricular performance of the children.

Conflict of Interest

The Authors declare that there are no conflicts of interest.

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