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Increase in Cardiac Contractility During Puberty

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A B S T R A C T

Pubertal growth of heart was analyzed. Growth rates of »diastolic variables« of heart size were compared with that of 'systolic', and both with growth intensities of some body structures and functions that are related to cardiac growth. Longitudinal echocardiographic, ergometric and anthropometric measurements were performed in 84 healthy boys, aged 11.5 years at the beginning and 14.5 at the end of study. Diastolic thickness of left ventricular walls increased by a mean rate of 15% and systolic by 36% ($p<0.001$). As a result, percent systolic wall thickening increased from 20% to 41% during the 3 years ($p<0.001$). Other measures of cardiac contractility increased in the same manner. Increase in measures of cardiac preload and afterload corresponds to the increase in heart »diastolic« and »systolic« variables, respectively. The study gave evidences for an increase in cardiac contractility during puberty. Coexistence of two simultaneous growth models for pubertal heart: diastolic and systolic, is suggested.

Key words: *growth, puberty, heart, myocardial contractility*

Introduction

Pubertal growth of human heart is not analyzed extensively. Two old cross-sectional studies[1,2] found, on small samples, synchronous growth of heart and body structures. That surprises because asynchronous growth of different body systems characterizes puberty. In a recent longitudinal study, Janz and colleagues[3] found that changes in sexual maturation were associated with changes in heart growth, but they did not pay attention to all aspects of synchronicity of cardiac growth. It is known that sexual maturation follows body growth acceleration with a mild latency[4], and that lungs grow with a delay related to body growth[5]. Cro-

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atian Growth Study[6,7] revealed a mild initial latency in pubertal heart growth acceleration when related to growth acceleration of body[8], and sexual dimorphism of heart pubertal growth[9] with temporal cardiac »growth stagnation« after menarche[10]. One of the most intriguing finding, obtained by the pilot analysis[11], was the finding of a different rate of pubertal thickening of diastolic and systolic thicknesses of left ventricular walls.

The aim of the study was to check out if the difference in the growth intensities of diastolic and systolic heart size exists or not, and if does, to explain this duality. Dual growth model could be possible if two main mechanical cardiac functions (receiving and ejecting blood) differ by an intensity of pubertal increase. That would have to results in changes of cardiac contractility. Because of all, growth intensities of cardiac diastolic and systolic variables were compared one another, and both with growth intensities of body structures and functions that are related to cardiac growth.

Material and Methods

Study design

Longitudinal echocardiographic, ergometric and anthropometric measurements were performed in 84 healthy boys. Measurements were repeated annually, always at the same period of year to avoid seasonal influences on some of observed variables, by the same team of observers (details elsewhere[12]). Personal and parental informed consent on the study protocol was obtained. All procedures were conducted in accordance with Helsinki Declaration of 1964, as revised in 1975.

Heart growth was displayed by pubertal changes in echocardiographic variables. Variables were divided into two groups, depending on the phase of cardiac cycle when they were measured; diastolic and systolic. Systolic (*s*) diameter of left ventricular cavity (LVD) and systolic thicknesses of left ventricular walls – interventricular septum (IVS) and left ventricular posterior wall (PW), were selected in the group of »systolic variables«. Diastolic (*d*) diameter of left ventricular cavity and diastolic thicknesses of left ventricular walls were selected in the group of »diastolic variables«, as well as left atrium (LA) and aortal root (AO) diameters. Percent of systolic wall thickening (100 systolic/diastolic wall thickness – 100) and shortening fraction ((100 (LVIDd-LVIDs) / (LVIDd)) were displayed as measures of cardiac contractility. For the comparison of increase in percent systolic wall thickening with the increase in body size and heart size, body surface area was used as a measure of body size and cumulative diastolic thickness of IVS and LVPW was used as a measure of heart size. These values were dichotomized by the median value of the sample.

Growth intensities of cardiac structures were compared with those of body surface area, diastolic blood pressure and physical working capacity as body structures and functions that are related to cardiac growth. Left ventricular diastolic size depends on inflow and end-diastolic blood volume (preload). The major determinant of ventricular preload is total blood volume. As blood volume correlates with body size[13], a change in body surface area was analyzed as a measure of change in preload size. Left ventricular systolic size depends on left ventricular supplying area (body) and peripheral vascular resistance, afterload determinants. A change in body surface area was again analyzed as a representative of body growth, and a change in diastolic blood pressure as a measure of vascular resistance increase. Physical working capacity reflects cardiac functional ability in a part. A change in maximum workload (a product of endurance time and body weight) was displayed as a measure of

changes in body strength and working capacity.

Sample

The sample was collected from the elementary school »Ante Kovačić«, Zagreb. The school was selected randomly. All healthy 5th-class pupils were included in the study. Health status was estimated on personal history, parental report and physical examination, and confirmed by subsequent echocardiographic and ergometric examinations. Out of 122 healthy boys included into the study, eighty four, who completed all measurements, were include into this analysis. Boys were aged 11.5 (± 0.5) at the beginning, and 14.5 (± 0.5) at the end of the study.

Measurements

Standard M-mode echocardiography was used, under the control of a 2-D imaging, from a parasternal long-axis view. The ATL Ultramark 8 device, with a 3 MHz transducer was used. All measurements were made according to the recommendation of the American Society for Echocardiography[14]. Subjects were in supine or, if needed, in left lateral decubitus position. Accuracy was 0.1 mm. Exercise test was made using the modification of Bruce's treadmill test[15], until exhaustion or until the end of 6th exercise stage (18 minutes). Accuracy was 0.1 minute. Boys were encouraged to make maximal effort. Ergometer Viagraph 200 was used. Maximum workload was calculated as a product of maximum oxygen uptake (reading from nomogram based on the value of endurance time – American Heart Association, 1982) and body weight. Blood pressure was measured with mercury sphygmomanometer. A cuff was placed over the right brachial artery. Diastolic blood pressure was determined by auscultation of the fourth Korotkoff sound. The fifth Korotkoff sound was used if the fourth sound could not be heard. Anthropometric measurements were taken according to the recommendation of the International Biological Program[16]. Boys were measured barefoot, in light underwear. Siber Hegner Machinen AG anthropometer and platform scale were used. Accuracy was 1 mm for body height, 0.5 kg for body weight. Body surface area was calculated according to formula by DuBois and DuBois[17], BSA = 71.84 height^{0,725} x weight$^{0.425}$. Two consecutive measurements were made and averaged, for all measurements presented except for ergometric testing.

Statistics

Initial (11.5 years) and final (14.5 years) values were compared to determine growth intensities of measured parameters. To enable comparison of growth rates, values of non-linear variables were transformed to linear. Square root of body surface area and cube roots of stroke volume and of maximum workload were calculated. Increments were compared by pared samples t-test. SPSS for Windows was used.

Results

All measured parameters increased during puberty ($p < 0.001$), but the rates of growth of diastolic and systolic variables differ significantly (Table 1). Cumulative systolic thickness of two left ventricular walls increased at the mean rate of 36% (difference is not significant), i.e. much more intensively ($p < 0.001$) than diastolic thickness of the same walls (15% mean, ns difference between IVS and LVPW). The difference between systolic and diastolic wall thickness (systolic wall thickening) increased by this from 20% at the age of 11.5 to 41% at the age of 14.5 (mean values; $p < 0.001$).

To determine if the increase in percent systolic wall thickening was a function of body size or heart size, percent systolic

TABLE 1
BASAL AND FINAL VALUES OF ECHOCARDIOGRAPHIC VARIABLES (X±SD, IN MM) AND PUBERTAL 3-YEAR GROWTH RATES

	IVSd	IVSs	LVPWd	LVPWs	LVIDd	LVIDs	AO	LA
11.5 years	7.8±1.1	8.7±1.6	7.0±0.8	8.8±1.9	41.1±3.6	27.3±3.0	23.9±2.0	25.3±2.7
14.5 years	9.0±1.3	11.8±2.1	8.1±1.3	12.1±2.1	47.4±4.5	29.2±3.9	26.9±2.4	28.7±3.6
Growth rate	14%	34%	16%	38%	16%	7%	13%	14%

IVSd – diastolic interventricular septum thickness; IVSs – systolic interventricular septum thickness; LVPWd – diastolic left ventricular posterior wall thickness; LVPWs – systolic left ventricular posterior wall thickness; LVIDd – diastolic left ventricular cavity diameter; LVIDs – systolic left ventricular cavity diameter; AO – enddiastolic aortal root diameter; LA – diastolic left atrial diameter

TABLE 2
PERCENT SYSTOLIC WALL THICKENING AS A FUNCTION OF BODY AND HEART SIZE

	BSA		IVSd + LVPWd	
	Below median	Over median	Below median	Over median
Percent systolic walls thickening at the age 11.5	29%	22%	37%	18%
Percent systolic walls thickening at the age 14.5	43%	40%	54%	31%

BSA – body surface area; IVSd + LVPWd – cumulative diastolic thickness of left ventricular walls

wall thickening was compared between bigger and smaller boys and, secondly, between boys with bigger and smaller hearts, at both pubertal stages: 11.5 and 14.5 years. Bigger boys had no greater percent systolic walls thickening neither at the age of 11.5, nor at the age of 14.5. In the same manner, boys with bigger hearts had no greater percent systolic walls thickening at either age; i.e. boys of the same age who had bigger body or bigger heart did not have greater percent systolic wall thickening (Table 2).

Rates of growth of diastolic thicknesses of left ventricular walls did not differ from these of other diastolic variables: left ventricular diastolic diameter, left atrial and aortic root diameters (ns). Due to the higher rate of growth of systolic walls thicknesses than the growth rate of diastolic diameter of left ventricle ($p < 0.001$), rate of growth of systolic diameter of left ventricular cavity was lower not only related to the rate of growth of systolic walls thickness ($p < 0.001$), but related to the rate of growth of heart diastolic variables ($p < 0.001$) as well. As a result of greater increase in diastolic than in systolic diameter of left ventricle, another measure of cardiac contractility – shortening fraction – increased from 34% to 38% ($p < 0.001$).

During the same period of puberty, square root of body surface area increased at the rate of 14%. At the age of 11.5, mean body surface area was 1.29 ± 0.15 m^2, while it was 1.66 ± 0.16 m^2 at the age of 14.5. The growth rate of that measure of cardiac preload did not differ from the growth rate of diastolic variables (ns). Diastolic blood pressure increased for 9%, from 71.9 ± 4.9 at the age of 11.5 to 78.2 ± 8.6 mmHg at the age of 14.5. By this, the sum of rates of increase of body surface

area and diastolic blood pressure (measure of afterload) was still lower than the growth rate of systolic wall thickness. A cube root of maximum workload increased for 21%. At the age of 11.5, maximum workload was 1.78 ± 0.35 l O_2/min, while it was 3.11 ± 0.67 at the age of 14.5.

Discussion

This study gave evidences for the increase in cardiac contractility during puberty. The increase in systolic walls thickening, in shortening fraction or in other measures of cardiac contractility were not revealed previously. The paper did not expose values of some derived variables, like left ventricular ejection fraction or stroke volume are, but their increase in this sample from 62% to 68% (9% increase) and from 48 ml to 74 ml (16% increase of the third root values), respectively, could strongly augment the findings.

One of the infrequent studies that covered cardiac pubertal changes[2] found similar growth intensity of diastolic variables as it was found in our study, but with a very slight pubertal increase in cardiac contractility. The major limitation of that study was their transversal nature, which cannot display the process of growth as longitudinal investigation can[18]. Such data collection and the small sample result in a controversially finding of a smaller heart size at the age of 18 than at the age of 16, for example. The selection of the sample (children with a two-year period of a blood pressure stability during the stormily growth process) further adds to the low level of study's realism.

Unrecognized increase in cardiac contractility surprises because findings like that on pubertal increase in functional working capacities[15] strongly suggests a need for increased cardiac contractility in that process. The observed increase in the diastolic blood pressure further suggests that systolic wall thickening have to increase to overcome an increased vascular resistance. By the increase of diastolic blood pressure from 72 to 78 mmHg, it is expected[19] that cardiac output increases from 9 to 14 l/min, i.e. for 35%. That may be hardly obtained by the 15% increase in the left ventricle diastolic dimensions. The extent of increase of myocardial mass and of diastolic left ventricle dimensions could not satisfy increase in body oxygen demand, especially during exercise, if myocardial contractility would not increase in an even greater extent.

Catastrophe theory model for pubertal increase in cardiac contractility

The increase in percent of systolic walls thickening seems to be a logical consequence of pubertal global changes. Coexistence of two pubertal growth patterns for a single cardiac structure, in accordance to its dual function – diastolic and systolic, is suggested by our findings. »Diastolic variables« follow the same (diastolic) pubertal growth pattern, and systolic variables follow different – systolic pattern of growth. The mechanism that regulates the two growth dynamics could be caused by (or mediated through) different dynamics of increase in body structures and functions that modify sizes of diastolic and systolic cardiac functions.

An equal increase of »diastolic variables« and of body surface area confirms the supposition that the growth of diastolic heart size (and myocardial mass) corresponds to the increase in cardiac preload, measured herein by a quantum of a systemic blood pool, indirectly. Systolic wall thickness probably reflects cardiac morphological size and functional capacity (myocardial contractility) at once, and its increase overcame the sum of increases in body surface area and diastolic blood pressure. That may be the caused by a task made on heart to satisfy not

only increased left ventricular supplying area and systemic vascular resistance (cardiac afterload), but an increased exercise oxygen demand as well.

Related to the mechanism of an increase in myocardial contractility during puberty, it might be a consequence of a genetically determined growth of heart on the one hand, of a physiological response as an adaptation to an increased oxygen demand (due to an increase in body size and functional working tasks) is, and a combination of both factors. The adaptation segment might be explained through a mechanism of the pubertal growth of heart which can be described by a catastrophe theory model. The model offers explanation for different other aspects of the regulation of human growth[4]. According to the catastrophe theory, certain variables can transform moving from one »behavioral level« (prepubertal growth) to another (postpubertal growth) in a continuous way of a smooth, linear growth, or in a sudden, discontinuous way known as a »catastrophic growth« (Figure 1). Diastolic variables change through puberty in a way close to the pattern of continuous growth, while systolic wall thickness changes in a way far more close to the pattern of catastrophic growth. The way

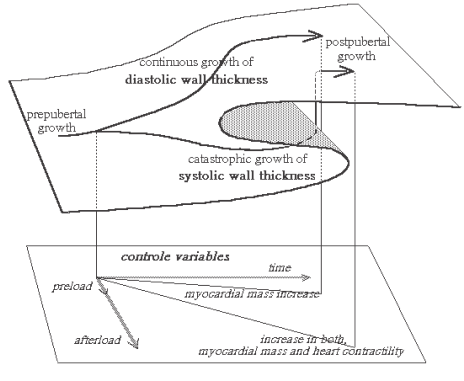

Fig. 1. Dual model of left ventricular pubertal growth.

of changes depends on control variables that regulate that growth. Growth of diastolic variables might be controlled by preload increase and it would reflect in myocardial mass increase. Growth of systolic wall thickness might be controlled by afterload increase and it would reflect in increase of both, myocardial mass and heart contractility.

Our finding that bigger body does not imply bigger percent systolic wall thickening discards the adaptation thesis as the complete explanation for the pubertal increase in cardiac contractility. So, other determinants, as above mentioned genetic factors, have to be included into the model. According to the greater increase in the systolic thickness of ventricular walls than in the afterload, it seems that cardiac systolic function prepares a reserve, probably for the future increase in myocardial energy expenditure due to an increase in functional working capacity. There might be the point for genetically triggered cardiac growth.

Beside some deficiency of the proposed dual model of growth, there are also limitations related to the study performance. Changing in the left ventricular inflow was not analyzed. Such data might add to the explanation of cardiac performance related to the preload determinants and diastolic function, but Doppler measurements were not available. Finally, it would be interesting to analyze cardiac growth by some other models, like the model of growth channels is[20], but that will be the task for another study.

In conclusion, the increase in cardiac contractility was found. Coexistence of two models for pubertal heart growth: diastolic and systolic, corresponding to the increase in cardiac preload and afterload might be suggested. Increased cardiac contractility is probably one of the most important factors that enable great increase in physical working capacity during puberty.

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POVEĆANJE KONTRAKTILNOSTI SRCA TIJEKOM PUBERTETA

SAŽETAK

Pubertetski rast srca je analiziran. Stope rasta »dijastoličkih« varijabli srca uspoređene su sa stopama rasta »sistoličkih« varijabli, a oboje s intenzitetom povećanja nekih tjelesnih morfoloških i funkcionalnih varijabli koje su vezane uz rast srca. Ehokardiografska, ergometrijska i antropometrijska longitudinalna mjerenja provedena su u 84 zdrava dječaka, u dobi od 11.5 godina na početku i 14.5 godina na kraju studije. Dijastolička debljina stijenki lijeve klijetke povećala se prosječno za 15% a sistolička za 36% ($p<0.001$). Kao rezultat, postotak sistoličkog zadebljanja stijenki povećao se tijekom 3 godine od 20% do 41% ($p<0.001$). Ostale mjere kontraktilnosti srca povećale su se na isti način. Povećanje mjera volumnog i tlakovnog opterećenja odgovaralo je stupnju povećanja »dijastoličkih« i »sistoličkih« varijabli. Studija je pružila dokaze za povećanje kontraktiliteta srca tijekom puberteta. Objašnjenje može pružiti koegzistencija dvaju simultanih modela rasta srca u pubertetu: dijastoličkog i sistoličkog.