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Echocardiographic changes in the development of the athlete's heart in 9 to 20-year-old male subjects

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The purpose of this cross-sectional investigation was to estimate the age at which specific traits of the "athlete's heart" first appear and how they evolve from the beginning of regular physical training until young adulthood in healthy active males. Male athletes (n=389) and non-athletes (n=55) aged between 9 and 20 years were examined by two-dimensionally guided M-mode and Doppler echocardiography. Intragroup differences were examined by *t*-tests for independent samples between age groups of two years each. Morphologic variables were related to body size by using ratio indices in which the power terms of numerator and denominator were matched. Relative left ventricular muscle mass (LVMM) was significantly larger in the athletic males at age of 11–12, and this significant difference was maintained with advancing age. Most of this increase of LVMM could be attributed to the increase in wall thickness that became significantly manifest first in the 13- to 14-year-old athletic subjects but was demonstrable in all the other groups. A significantly larger left ventricular internal diameter was only found in the age-group of 15–16. Fractional shortening percentage (FS%) did not show any change, while resting heart rate was decreased in our athletic groups.

Keywords: echocardiography, athlete's heart, young athletes

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In adults it is an extensively investigated issue and a well-established fact that regular physical training of sufficient intensity and volume induces modifications in the structure, function and autonomic regulation of the heart (2).

These adaptive changes collectively define what is termed on “athlete’s heart” (9, 10, 15, 28, 29, 30, 34). Since its main morphologic characteristic, myocardial hypertrophy, also occurs in several pathological conditions, the modifications in morphology induced by physical training have been investigated in association with the relevant functional parameters (4, 27, 29, 37).

A great number of echocardiographic data refer to young age in connection with the athlete’s heart. In some studies young athletes (active in different branches of sports at various levels of performance) were compared to age-matched non-athletic controls (1, 14, 17, 31, 32, 35), in other studies the development of an athletic heart was followed longitudinally for some months or some years (5, 11, 18, 19, 20). Most studies found left ventricular muscle mass (LVMM) to be larger in the active group (17) or increased due to regular physical training (1, 5, 11, 18, 20). In the studies reporting no difference (19, 32, 35) either the intensity of the type of exercise can be considered insufficient. Differences were also found in respect of the two components of myocardial hypertrophy. According to some studies left ventricular internal diameter (LVID) increased more markedly (17, 19, 20), in other studies it was the left ventricular wall thickness (LVWT) that showed the greater share in the increase (1, 11), while a third group of authors (5, 18) observed increase in both components. Dissimilar results may be explained by differences of the subjects’ age, sports activity and length of observation.

In some of our previous works (24, 26) and in some other studies (6) data of young athletes and non-athletes were compared to those of adults. These results have suggested that cardiac hypertrophy does not develop to its full extent before 18 years of age.

In the present study our goal was to examine when and in what order the various characteristics of the athlete’s heart appear during the life period extending from childhood until young adult age.

Subjects and Methods

Subjects

Data of 444 male subjects aged between 9 and 20 years with no history of cardiac disease and taking no medication are presented in this study. The subjects were divided into six age groups of two years each. Table I contains some basic characteristics of the subjects.

Table I

Body surface area (BSA), systolic blood pressure (SBP) and diastolic blood pressure (DBP) in male controls and athletes: mean (SD)

Age group	N	BSA (m^2)	SBP (mm Hg)	DBP (mm Hg)
9–10c	12	1.02 (0.11)	110.83 (10.83)	66.66 (9.84)
9–10a	59	1.13 (0.11)†	112.92 (12.22)	68.02 (9.99)
11–12c	9	1.33 (0.26)	110.55 (7.68)	68.88 (7.81)
11–12a	72	1.25 (0.11)	113.84 (10.72)	72.11 (8.73)
13–14c	8	1.46 (0.20)	118.75 (8.76)	73.75 (10.60)
13–14a	39	1.52 (0.17)	120.25 (12.29)	72.94 (10.55)
15–16c	6	1.86 (0.16)	128.83 (10.20)	83.33 (9.83)
15–16a	50	1.95 (0.21)	131.46 (14.79)	78.90 (9.94)
17–18c	9	1.95 (0.18)	147.22 (18.21)	83.88 (12.69)
17–18a	54	1.95 (0.19)	127.09 (11.55)*	81.11 (10.84)
19–20c	11	1.86 (0.11)	130.90 (11.36)	82.27 (12.52)
19–20a	115	1.98 (0.16)†	128.26 (14.09)	80.40 (9.84)

N: number of echocardiographic measurements

a: athletic subjects; c: control subjects

†: $p<0.02$, *: $p<0.001$ compared to controls

Fifty-five non-athletic healthy males, matched for age, were used as controls. The control subjects were students attending primary or secondary school or a university. Apart from the obligatory curricular physical education program (2 hours per week) they did not do any physical exercise. The athletic sample (n=389) comprised various branches of sports and levels of performance. Table II indicates the distribution of the athletes by events and some basic data about their physical training (years of training history, training hours per week). In all age groups endurance athletes and ball-game players gave the highest share of the groups.

Height (BH) and weight (mass) (BW) were used to estimate BSA according to the formula of Du Bois and Du Bois (8) which has found the widest acceptance as the best general predictor (16).

Echocardiography

Investigations were carried out using a Dornier AI 4800 ultrasound imaging system with a 2.5 MHz transducer. Two-dimensionally guided M mode recordings were obtained parasternally in accordance with the recommendations of the American Society of Echocardiography (2, 33).

M-mode measurements of interventricular septal thickness and of left ventricular posterior wall thickness at end-diastole (IVSTs, LVPWTd), left ventricular internal diameter at end-systole and end-diastole (LVIDs, LVIDd) were made at or just below the mitral valve tips.

All studies were performed by the same investigator (GP). Data were obtained across several cardiac cycles; means of five to ten cardiac cycles were used in the further analysis. Investigations were always carried out with the subject at absolute rest, in a left recumbent position.

Left ventricular wall thickness (LVWTd) was obtained as the sum of IVSTd and LVPWTd (LVWTd=IVSTd+LVPWTd). Left ventricular muscle mass (LVMM) was calculated by cubing the respective diameters (33, 36) as LVMM=[(IVSTd + LVPWTd + LVIDd)^3 – EDV)] × 1.05, where EDV is end diastolic volume=LVIDd^3, and 1.05 stands for cardiac wall density.

The quotient LVWTd/LVIDd% defined as the relative wall thickness by some investigators (9, 29) and as the muscular quotient (MQ) (38) or hypertrophy index by others (6*)* was also calculated. In this presentation we term it MQ.

According to various studies (3, 7, 12, 13, 23, 24, 25), when body size varies greatly, relative cardiac measures are correct only if for the respective body dimensions the exponents of the numerator and the denominator in the formula match. Preferring to keep indices related to body surface area (BSA), in this study linear variables were related to the square root of the body surface area (BSA$^{1/2}$), while volumes and masses to the cube of its square root (BSA$^{3/2}$) (23, 24, 25). The exponent corrected indices used to relate cardiac parameters to body size were as follows: ‘LVWTd=LVWTd/BSA$^{1/2}$, ‘LVIDd=LVIDd/BSA$^{1/2}$, ‘LVMM=LVMM/BSA$^{3/2}$, ‘SV=SV/BSA$^{3/2}$, and ‘CO=CO/BSA$^{3/2}$, where the inverted quote indicates exponent correction, SV means stroke volume, CO means cardiac output.

Parameters, such as heart rate (HR), SV, CO and fractional shortening (FS%) were investigated as indicators of the function and regulation of the athletes’ heart.

Left ventricular shortening fraction percentage (FS%) was calculated as the ratio (LVIDd-LVIDs) × 100/LVIDd.

Resting CO was calculated as the product of stroke volume SV and resting HR, where SV is calculated as LVIDd^3-LVIDs^3. HR was measured during the echocardiographic examination.

Statistical analysis

The mean values for the athletes were compared with those of their age-matched controls using two-tailed *t*-tests for unpaired data.

Results

There were no significant differences in BSA between the athletic and non-athletic subjects (Table II), in any of the age groups except for those of 9–10 and 19–20 years in which BSA was found to be higher in the athletic subjects.

Table III gives means±SD of the measured values of IVSTd, LVPWTd, and LVIDd and the calculated values of LVWTd, LVMM and MQ (%); while in Table IV the exponent corrected indices are presented.

Absolute values of IVSTd, LVWTd and LVMM were found to be significantly higher in athletes earliest in the 13-to 14-year-old group ($p<0.05$). Such differences became more marked in the older age groups. Absolute LVPWTd was significantly higher in the athletes as young as 15–16 ($p<0.02$), then in those aged 17–18 ($p<0.02$) and finally in the 19–20-year-old group ($p<0.001$). LVIDd in the athletes was found to be significantly higher first at 9–10 years ($p<0.02$), then at 15–16 and 17–18 years ($p<0.02$) and finally in the 19–20-year-old athletes ($p<0.05$).

Table II

Distribution of the athletic subjects by events of sports

Age group	END	BGP	PWR	SPRJ	SPS	LTA	TOT	YRS of TRAINING M (SD)	TR.HRS/W M(SD)
9–10	21	33	3	–	–	2	59	2.48(1.76)	7.41(4.41)
11–12	31	36	5	–	–	–	72	3.72(2.02)	9.09(3.76)
13–14	23	15	–	–	1	–	39	3.92(2.7)	11.12(5.22)
15–16	8	34	7	–	–	1	50	7.58(3.37)	14.75(5.78)
17–18	11	31	9	–	1	2	54	9.88(3.08)	17.39(7.41)
19–20	26	55	19	13	2	–	115	10.4(3.79)	18.29(6.34)

END: endurance athletes
BGP: ball game players
PWR: power athletes
SPRJ: sprinters and jumpers
SPS: sportsmen of point system sports
LTA: leisure time activity sportsmen
TOT: total number of subjects
TR.HRS/W: training hours per week
M: mean

Regarding the exponent-corrected BSA-related indices (Table IV), 'LVWTd was approximately constant in the non-trained group over the studied age range, while a tendency to increase with age was observed in the athletic subjects, 13–14 years being the youngest age at which the difference proved to be significant. The difference between the athletic and non-athletic groups grew in the successive age groups (13–14 yr.: $p<0.05$, 15–16 yr.: $p<0.02$, 17–18 yr.: $p<0.02$, and 19–20 yr.: $p<0.001$).

'LVIDd tended to be higher in the physically trained subjects in all age groups. By years 11–12, it already approximated significance ($p=0.07$), by the 15th–16th years it was significantly higher ($p<0.05$).

Table III

Echocardiographic morphologic data (absolute) in the 9–20-year-old male controls and athletes in diastole: mean (SD)

Age group	IVSTd (mm)	LVPWTd (mm)	LVWTd (mm)	LVIDd (mm)	LVMM (g)	MQ%
9–10c	6.69(0.59)	6.40(0.84)	13.32(1.56)	38.44(1.8)	83.78(7.86)	34.80(5.01)
9–10a	6.88(1.05)	6.85(1.02)	13.70(1.92)	40.45(2.54)†	98.43(22.04)	34.02(5.23)
11–12c	7.52(1.05)	7.14(0.75)	14.66(1.76)	40.96(3.71)	110.84(30.03)	35.93(4.55)
11–12a	7.45(0.87)	7.33(0.78)	14.78(1.51)	41.57(3.21)	113.78(21.94)	35.78(4.58)
13–14c	7.54(0.78)	7.47(0.85)	15.01(1.61)	42.63(3.23)	121.59(27.63)	35.30(3.91)
13–14a	8.42(0.99)‡	8.10(1.09)	16.52(1.95)‡	45.38(3.92)	154.03(38.52)‡	36.56(4.28)
15–16c	8.36(1.00)	8.14(1.14)	16.56(2.13)	45.86(4.60)	157.56(43.37)	36.25(4.46)
15–16a	9.74(1.17)†	9.42(1.21)†	19.16(2.25)†	51.12(4.06)†	228.02(54.56)†	37.67(4.87)
17–18c	9.06(0.87)	8.09(0.83)	17.96(1.51)	49.05(4.20)	192.46(37.73)	36.84(4.15)
17–18a	10.28(1.38)†	9.92(1.17)†	20.21(2.39)†	50.78(4.22)†	243.81(65.11)‡	39.87(4.04)‡
19–20c	9.36(0.9)	8.52(0.65)	17.89(1.31)	48.52(3.61)	189.45(29.39)	37.04(3.68)
19–20a	10.5(1.04)*	9.97(1.08)*	20.48(1.98)*	51.34(3.90)‡	249.89(48.57)*	40.08(4.52)‡

IVSTd: interventricular septum thickness
LVPWTd: left ventricular posterior wall thickness
LVWTd: left ventricular wall thickness (IVSTd + LVPWTd)
LVIDd: left ventricular internal diameter at end-diastole
LVMM: left ventricular muscle mass
MQ, muscular quotient (LVWTd/LVIDd %)
a: athletes; c: controls
†: $p<0.02$, ‡: $p<0.05$, *: $p<0.001$ compared to controls

As a consequence, the ratio of these two parameters, LVWTd/LVIDd %, i.e. the muscular quotient (MQ) (see Table III) was significantly higher in the athletic subjects of the two oldest groups ($p<0.05$).

'LVMM, the most consistent indicator of training induced hypertrophy, was significantly higher in the athletic subjects beginning with the early age of 11 to 12 years ($p<0.05$). Differences between athletic and non-athletic subjects remained significant also in the subsequent age groups: 13–14 ($p<0.02$), 15–16 ($p<0.001$), 17–18 ($p<0.02$) and finally 19–20 ($p<0.001$).

Systolic function as assessed by the fractional shortening percentage (FS%) was unaltered in all age groups, i.e. no significant differences were observed between athletes and non-athletic subjects across the entire age span from 9 through 20 years (see Table IV).

Table IV

Body size related echocardiographic data in 9–20-year-old male controls and athletes: mean (SD)

Age group	LVWTd/BSA$^{1/2}$ (mm/m)	LVIDd/BSA$^{1/2}$ (mm/m)	LVMM/BSA$^{3/2}$ (g/m^3)
9–10c	13.14(1.18)	38.09(2.99)	83.45(10.83)
9–10a	12.89(1.61)	38.12(2.40)	81.68(13.80)
11–12c	12.76(1.12)	35.69(1.80)	71.88(6.37)
11–12a	13.25(1.33)	37.22(2.47)	81.52(13.27)‡
13–14c	12.43(1.00)	35.38(2.82)	68.91(11.47)
13–14a	13.39(1.22)‡	36.82(2.30)	81.14(11.99)†
15–16c	12.06(1.53)	33.40(3.31)	60.84(16.57)
15–16a	13.73(1.37)†	36.66(2.45)‡	83.35(13.88)*
17–18c	12.89(1.02)	35.13(1.95)	71.06(8.21)
17–18a	14.47(1.47)†	36.38(2.44)	88.80(18.19)†
19–20c	13.10(0.94)	35.52(2.46)	74.29(11.40)
19–20a	14.54(1.28)*	36.45(2.45)	89.11(13.98)*

LVWTd: left ventricular wall thickness (IVSTd + LVPWTd)
LVIDd: left ventricular internal diameter at end-diastole
LVMM: left ventricular muscle mass
CO: cardiac output
SV: stroke volume
BSA: body surface area; a: athletes; c: controls
†: $p<0.02$, ‡: $p<0.05$, *: $p<0.001$ compared to controls

On the other hand, the athletes showed a training induced bradycardia across all age groups. Resting HR was lower in the athletes in all age groups, in the age groups of 11–12, 17–18 years and finally in those of 19–20 years resting HR (Table V) was found to be significantly lower in the athletic subjects ($p<0.02$, $p<0.05$ and $p<0.001$, respectively).

The 'SV index ($SV/BSA^{3/2}$) was significantly higher in the athletes of 11–12 years ($p<0.05$) and in those of 15 to 16 years ($p<0.02$).

The resting 'CO index was found to be significantly lower only in the 19- to 20-year-old group of athletes ($p<0.05$).

Table V

Cardiac functions in 9–20-year-old male controls and athletes: mean (SD)

Age group	HR (beats.min^{-1})	FS (%)	$CO/BSA^{3/2}$ (l/min.m^3)	$SV/BSA^{3/2}$ (ml/m^3)
9–10c	78.75(9.22)	38.63(2.71)	3.43(1.02)	43.33(10.12)
9–10a	73.04(10.12)	37.23(3.95)	3.09(0.87)	42.06(8.70)
11–12c	87.06(16.96)	33.89(3.90)	2.80(0.64)	32.38(4.99)
11–12a	72.54(11.67)†	36.38(4.66)	2.77(0.66)	38.51(7.94)‡
13–14c	78.79(9.52)	35.83(4.79)	2.66(0.62)	32.55(6.01)
13–14a	71.30(10.71)	35.64(4.95)	2.62(0.61)	36.97(8.06)
15–16c	73.1(15.67)	35.01(4.27)	2.01(0.71)	27.43(8.02)
15–16a	65.01(13.17)	36.00(3.68)	2.40(0.76)	36.60(6.99)†
17–18c	69.13(8.17)	34.90(4.45)	2.18(0.50)	31.54(6.02)
17–18a	61.35(10.83)‡	34.28(4.72)	2.11(0.50)	34.89(7.77)
19–20c	79.35(14.98)	33.01(2.59)	2.51(0.73)	31.74(6.48)
19–20a	61.81(10.32)*	34.03(4.49)	2.14(0.55)‡	34.77(7.29)

HR: heart rate
FS: fractional shortening
CO: cardiac output
BSA: body surface area
SV: stroke volume
c: controls; a: athletes
†: $p<0.02$, ‡: $p<0.05$, *: $p<0.001$ compared to controls

Discussion

Cardiac hypertrophy is an unambiguous characteristic of the athlete's heart. In preadolescent and adolescent athletes, however, conflicting results have been reported about the proportion of the two components of the left ventricular hypertrophy, namely, the development of LVWTd and LVIDd with age.

Allen et al. (1) in 5 to 17-year-old swimmers, Geenen et al. (11) in 6- and 7-year-old aerobically exercised children reported a more marked increase in wall thickness than in LVIDd. Csanády et al. (6) did not find any difference between junior and senior basketball players in the LVWTd/LVIDd ratio. In contrast, Obert et al. reported that programs of long-term swimming (20) and aerobic training (19) induced a much higher increase in LVIDd than in LVWTd.

In the present study a significantly larger 'LVMM was found already in the athletes of 11–12 years and the difference became gradually more marked by the age of 19–20 years. This finding is in accordance with other published investigations dealing with the effects on cardiac morphology of systematic physical training in young athletes.

In the proportion of the two components of left ventricular hypertrophy, namely MQ (LVWT/LVID), there were no intragroup differences between athletes and non-athletic subjects from 9 to 16 years of age. It was found to be significantly higher in the older athletes (17–18 and 19–20 years), indicating thereby an approximately proportional increase of LVIDd and LVWTd during the first years of athletic training.

'LVWTd was found to be significantly higher in athletes earliest at an age of 13–14 years. and the difference grew consistently with age. 'LVIDd did not follow such a clear and proportional dynamics of development. The difference between the athletic and non-athletic subjects in 'LVIDd was near to significance ($p=0.07$) as early as at the age of 11–12 years and it was significant only between the 15–16-year-old groups ($p<0.05$). The less marked difference in LVIDd is in accordance with our earlier observation (24) according to which LVWTd shows a greater increase in athletes than LVIDd does.

As can be deduced from our material and from some other reports, morphological modifications are initiated by an increase in 'LVIDd that is either coupled with, or followed soon by, an increase in 'LVWTd. That is why MQ appears unchanged in the early period of systematic athletic training. A significant increase of this ratio was observed in our athletes in late adolescence (17–18 years) and young adult age (19–20), when 'LVIDd did not further develop appreciably, while 'LVWTd showed a further significant increase, in accordance with the report of Csanády et al. (6).

As the age group of 13–14 was the youngest to show a significant intergroup difference in 'LVWTd, it can be assumed that the more intense increase in muscular wall thickness is associated with the increased secretion of male sexual hormones.

In agreement with other data (18) our results also indicate that in the training induced myocardial hypertrophy the share of concentric and eccentric hypertrophy, i.e. whether or not the increase of LVWTd exceeds the increase of LVIDd (15, 28), cannot be clearly distinguished in athletes of 16 and younger, especially so when several sport events and different modes of training are involved in the sample.

Besides the morphological characteristics, several parameters and indices refer to the function and regulation of the athletic heart.

Resting HR is definitely lower in athletic subjects, and so is resting ejection fraction or FS. SV is a little larger in athletes, and, as a result of all these shifts, resting CO is the same as, or a little lower than in non-athletic subjects (21, 22).

Of all these parameters resting HR was the only one that showed a definite consistency in our investigations: it was lower in athletic subjects of all ages compared to controls, indicating the well established training bradycardia.

Cardiac frequency is regarded as a parameter influenced by both components of the autonomous regulation, while ventricular musculature does not get parasympathetic innervation. Parameters referring only to the ventricular musculature like FS% are indicative only of the sympathetic part of autonomous regulation. We suppose, therefore, that in the young, the increase of resting parasympathetic activity is more consistent than the decrease of the sympathetic one.

As the resting ‘CO ($CO/BSA^{3/2}$) is assumed to be a result of a refined regulation, it is not surprising that the ‘CO did not differ between athletic and non-trained subjects in the young, and it was the 19- to 20-year-old group, in which ‘CO was first found to be significantly lower in the athletic subjects.

Our results indicate that among the morphological characteristics of the athlete’s heart the increase of LVWT seems to be a more consistent feature than the increase of LV internal diameter which shows an increasing tendency only in the early period. Among the functions and regulation of the heart resting parasympathetic activity only seems to be consistently modified in this age period to result in a well-defined training bradycardia.

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