Echocardiographic characteristics of male athletes of different age

G Pavlik, Z Olexó, P Osváth, Z Sidó, R Frenkl

Abstract

Two dimensionally guided M mode and Doppler echocardiographic data for 578 male subjects (106 non-athletic and 472 athletes) were analysed from two aspects: (a) in the young adult category (19–30 years of age), competitors in different groups of sports were studied; (b) in the different age groups (children, 10–14 years; adolescent juniors, 15–18 years; young adults, 19–30 years; adults, 31–44 years; older adults 45–60 years), data for athletes and non-athletes were compared. Morphological variables were related to body size by indices in which the exponents of the numerator and denominator were matched. Morphological signs of athletic heart were most consistently evident in the left ventricular muscle mass: in the young adult group, the highest values were seen in the endurance athletes, followed by the ball game players, sprinters/jumpers, and power athletes. A thicker muscular wall was the main reason for this hypertrophy. Internal diameter was only increased in the endurance athletes, and this increase was more evident in the younger groups. The E/A quotient (ratio of peak velocity during early and late diastole) indicated more effective diastolic function in the endurance athletes. The values for E/A quotient also suggested that regular physical activity at an older age may protect against age dependent impairment of diastolic function.

Keywords: echocardiography; heart; athletic heart; age; male athletes

To avoid such spurious trends, we attempted to use indices in which power terms match.1 2 Preferring to keep the relation to BSA, we suggested that linear variables are related to the square root of BSA, with volumes and weights related to the cube of the square root of BSA. These indices did not show any correlation with body measures, so it became possible to compare data for subjects of different age, body size, or weight. Similar suggestions have recently been made by others.3 4

In this study, these modified indices were used to analyze the echocardiographic results of a large number of male athletes and non-athletes. The results are discussed from two aspects. In the young adult athletes, the effects of their different sports are investigated; data for power athletes, sprinters/jumpers, ball game players, and endurance athletes are compared with each other and with data for young adult non-athletic subjects. A comparison is also made across the ages: from childhood to older age, data on athletes are compared with those on non-athletic healthy subjects.

As the main morphological characteristic of athletic heart—that is, myocardial hypertrophy—can also occur in several pathological states, training induced morphological modifications should be considered together with some functional parameters. In addition to the morphological variables, resting heart rate and E/A quotient—that is, the ratio of early and late transmitral flow velocity—will be given. The latter is a sensitive indicator of diastolic function—that is, left ventricular compliance. A decrease in the quotient unambiguously indicates an impairment of the diastolic function as the result of either advanced age5–7 or some pathological event.8–10

Subjects and methods

Table 1 gives some basic characteristics of the subjects. They were separated into five age groups as follows: children, 10–14 years; adolescents/juniors, 15–18 years; young adults in the age range at which most top class competition occurs, 19–30 years; adults, 31–44 years; older adults, 45–60 years.

The controls were healthy males of comparable age who were not taking any medication and had no history of cardiac disease. The athletes were competitors of variable ability.

The child athletes were soccer players and swimmers who performed 8–10 hours of physical training a week. Adolescent and junior athletes were top level middle distance and long distance runners, competitive cyclists, triathletes, waterpolo and basketball players, and weight lifters.
The group of young adults was subdivided according to the type of sport. The power athletes were top level (members of national teams or first class competitors) judo competitors and weight lifters. The sprinters/jumpers group contained second class track and field athletes and top level short track skaters. The ball game group was second level and top level waterpolo and soccer players and second level volleyball players. The endurance athletes were top level (members of national teams) road cyclists, kayak canoeists, pentathletes, and long distance runners. Data for some athletes (fencers, gymnasts, etc) who were not classified into groups were calculated in the total.

The adult group contained several pentathletes, cyclists, and canoeists still competing at the top level and lower level ball game players. Athletes in the older group were recreational athletes with a training regimen of a minimum of three hours and a maximum of 15 hours a week.

As Doppler examinations were not carried out on all the subjects, the E/A quotient column of tables 3 and 5 the number of subjects examined was smaller, indicated by column N' in table 1.

Investigations were always carried out with the subject at absolute rest using a Dornier AI 4800 echocardiograph 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 Echocardiography11; measurements of the left ventricular wall thickness and internal diameter were obtained by positioning the trackback cursor on the screen. All studies were performed by the same investigator (GP). Early and late diastolic peak filling velocities were estimated by pulse wave Doppler measurements in the four chamber apical view. Data were obtained across several cardiac cycles; means of five to ten cycles were used in the further analysis.

Left ventricular (LV) wall thickness (WT) was obtained as the sum of interventricular septum thickness (IVST) and posterior wall thickness (PWT). Of the several possibilities, left ventricular muscle mass (LVM) was calculated by cubing the respective diameters12 13 as:

\[ LVM = (IVST + PWT + EDD)^3 \]

The latter is termed relative wall thickness in many papers14 15 and muscular quotient16 or hypertrophy index17 in others.

Mean values for athletes were compared with those for age matched controls using t tests for unpaired data. Differences at p<0.05 were regarded as significant.

**Results**

Apart from some young adult groups, there was no significant difference in body size between athletic and non-athletic groups. Resting training bradycardia was evident in all of the athletic groups, the except the older adult athletes where the difference was not significant (table 1).

### Table 1

<table>
<thead>
<tr>
<th>Group</th>
<th>N</th>
<th>N'</th>
<th>Age (years)</th>
<th>BSA (m²)</th>
<th>HR (beats/min)</th>
</tr>
</thead>
<tbody>
<tr>
<td>CHLDc</td>
<td>13</td>
<td>12</td>
<td>12.38 (1.04)</td>
<td>1.34 (0.23)</td>
<td>87.11 (14.35)</td>
</tr>
<tr>
<td>CHLDa</td>
<td>88</td>
<td>40</td>
<td>11.76 (1.15)</td>
<td>1.30 (0.17)</td>
<td>72.03 (11.92)</td>
</tr>
<tr>
<td>Ajc</td>
<td>10</td>
<td>10</td>
<td>17.40 (0.84)</td>
<td>1.94 (0.18)</td>
<td>71.75 (8.99)</td>
</tr>
<tr>
<td>Alc</td>
<td>77</td>
<td>32</td>
<td>16.35 (1.10)</td>
<td>1.95 (0.21)</td>
<td>63.37 (12.44)</td>
</tr>
<tr>
<td>YADc</td>
<td>44</td>
<td>42</td>
<td>23.23 (3.38)</td>
<td>1.91 (0.13)</td>
<td>76.19 (14.58)</td>
</tr>
<tr>
<td>YADa</td>
<td>38</td>
<td>12</td>
<td>22.03 (2.99)</td>
<td>1.96 (0.2)</td>
<td>64.10 (11.93)</td>
</tr>
<tr>
<td>YAD-SPRJ</td>
<td>21</td>
<td>21</td>
<td>21.90 (2.68)</td>
<td>2.00 (0.15)†</td>
<td>61.03 (7.94)‡</td>
</tr>
<tr>
<td>YAD-BGP</td>
<td>110</td>
<td>82</td>
<td>22.34 (3.09)</td>
<td>2.08 (0.14)‡</td>
<td>58.80 (10.65)‡</td>
</tr>
<tr>
<td>YAD-END</td>
<td>90</td>
<td>43</td>
<td>23.00 (3.07)</td>
<td>1.95 (0.14)‡</td>
<td>59.34 (10.52)</td>
</tr>
<tr>
<td>ADc</td>
<td>28</td>
<td>20</td>
<td>37.11 (4.37)</td>
<td>2.00 (0.16)§</td>
<td>71.53 (13.44)</td>
</tr>
<tr>
<td>ADa</td>
<td>33</td>
<td>28</td>
<td>36.15 (4.52)</td>
<td>2.07 (0.17)§</td>
<td>61.04 (9.69)‡</td>
</tr>
<tr>
<td>OADc</td>
<td>11</td>
<td>9</td>
<td>50.45 (4.30)</td>
<td>2.03 (0.09)‡</td>
<td>70.09 (9.34)</td>
</tr>
<tr>
<td>OAda</td>
<td>12</td>
<td>12</td>
<td>50.25 (3.05)</td>
<td>2.00 (0.10)§</td>
<td>63.83 (9.70)</td>
</tr>
</tbody>
</table>

Values are mean (SD). *p<0.05, †p<0.02, ‡p<0.001 compared with controls.

**Table 2**

<table>
<thead>
<tr>
<th>Table 2</th>
<th>Body size related echocardiographic data for young adult athletes</th>
</tr>
</thead>
<tbody>
<tr>
<td>Group</td>
<td>WT/BSA (g/m²)</td>
</tr>
<tr>
<td>Control</td>
<td>12.80 (0.77)</td>
</tr>
<tr>
<td>PWR</td>
<td>14.24 (1.45)†</td>
</tr>
<tr>
<td>SPRJ</td>
<td>14.53 (1.21)†</td>
</tr>
<tr>
<td>BGP</td>
<td>14.92 (1.53)†</td>
</tr>
<tr>
<td>END</td>
<td>14.56 (1.59)†</td>
</tr>
</tbody>
</table>

Values are mean (SD). *p<0.05, †p<0.001 compared with controls.

WT, Left ventricular wall thickness; BSA, body surface area; EDD, left ventricular end diastolic diameter; LVM, left ventricular muscle mass; E/A, ratio of the early and late diastolic filling peak velocity; PWR, power athletes; SPRJ, sprinters/jumpers; BGP, ball game players; END, endurance athletes.
The morphological cardiac dimensions related to the exponent corrected body size measure (table 5) were very similar in the control subjects of different age; only the oldest group seemed to have a slightly higher "WT and LVM.

Concerning training induced LV hypertrophy, however, some differences were observed in both its extent and the share of its two components at the different ages. In the athletic children, "EDD was significantly higher than in the non-athletic ones, and "WT was slightly but not significantly larger. In the adolescent/junior groups, the difference in "WT reached significance. In young adults, differences in "WT were more pronounced than in "EDD. In the adult groups, hypertrophy was only manifest in "WT, and significant differences were absent in the older adults. As a result, differences in "LVM increased from those in children (18.7%) to those in adolescent/juniors and young adults (25.6% and 26.2% respectively), then decreased in the adult group (16.9%), and had disappeared in the older adults. "WT/EDD was not significantly different in the children and in the adolescent/junior group; the difference from controls was significant in the young adult group (11.0%), reached a maximum in the 31–44 year age group (39.8%), and disappeared again in the older groups.

Except for in children, the E/A quotient was significantly higher in the athletic groups at all ages. It should be noted that differences between the three adult age groups (young adults, 8.9%; adults, 20.4%; older adults 34.4%) consistently increased (table 5).

Discussion
In our recent reports and one from another group, it has been shown that heart indices in which the dimensional exponents of numerator and denominator match do not correlate with body size whereas simple BSA related indices do. The corrected indices would seem to be more suitable for comparing subjects of different body size.

In the pathological range, the largest IVST and LVPWT being 15.30 and 14.36 mm respectively.

Using the exponent corrected indices (table 3), athletic groups displayed increased LV measures, but the two components of hypertrophy varied. A highly significant increase was seen in the "WT in all groups, with no appreciable difference among the athletic groups. "EDD, however, was significantly larger only in endurance athletes; in the ball game players it tended to be only slightly larger (not significant).

"LVM showed a highly significant positive difference for all the athletic groups; values were found in the ball game players, and the lowest ones were found in the endurance athletes.

The E/A quotient (ratio of peak velocity during early and late diastole) was significantly higher in the endurance athletes only. In the

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Table 4 Echocardiographic parameters in men of different age

<table>
<thead>
<tr>
<th>Parameter</th>
<th>CHLDc</th>
<th>CHLDa</th>
<th>Alc</th>
<th>A1a</th>
<th>YADc</th>
<th>YADA</th>
<th>ADc</th>
<th>ADA</th>
<th>AJc</th>
<th>AJA</th>
</tr>
</thead>
<tbody>
<tr>
<td>IVST (mm)</td>
<td>7.24 (0.84)</td>
<td>7.72 (1.17)</td>
<td>8.91 (1.05)</td>
<td>10.06 (1.42)‡</td>
<td>8.97 (0.78)</td>
<td>10.63 (1.31)‡</td>
<td>9.36 (1.26)</td>
<td>10.95 (1.40)†</td>
<td>9.06 (1.42)†</td>
<td>10.06 (1.42)†</td>
</tr>
<tr>
<td>LVPWT (mm)</td>
<td>7.09 (0.70)</td>
<td>7.37 (1.03)</td>
<td>9.00 (1.06)</td>
<td>9.47 (1.27)†</td>
<td>8.68 (0.58)</td>
<td>10.16 (1.28)‡</td>
<td>9.03 (0.87)</td>
<td>10.41 (1.21)†</td>
<td>9.36 (1.27)†</td>
<td>10.16 (1.28)†</td>
</tr>
<tr>
<td>EDD (mm)</td>
<td>41.37 (3.79)</td>
<td>42.69 (3.83)*</td>
<td>48.00 (5.25)</td>
<td>51.01 (4.11)*</td>
<td>49.43 (3.68)</td>
<td>52.86 (4.43)</td>
<td>50.70 (3.67)</td>
<td>51.04 (3.98)</td>
<td>51.87 (3.67)</td>
<td>52.40 (3.82)†</td>
</tr>
<tr>
<td>LVM (g)</td>
<td>109.0 (27.8)</td>
<td>123.4 (33.4)</td>
<td>189.0 (49.1)</td>
<td>238.1 (63.9)†</td>
<td>191.5 (26.4)</td>
<td>264.6 (59.3)†</td>
<td>250.2 (46.9)</td>
<td>261.1 (55.0)†</td>
<td>255.2 (46.9)</td>
<td>252.4 (60.2)</td>
</tr>
</tbody>
</table>

Values are mean (SD).

*p<0.05, †p<0.02, ‡p<0.01, §p<0.001 compared with controls.

Table 5 Body size related echocardiographic parameters in men of different age

<table>
<thead>
<tr>
<th>Parameter</th>
<th>CHLDc</th>
<th>CHLDa</th>
<th>Alc</th>
<th>A1a</th>
<th>YADc</th>
<th>YADA</th>
<th>ADc</th>
<th>ADA</th>
<th>AJc</th>
<th>AJA</th>
</tr>
</thead>
<tbody>
<tr>
<td>WT/BSA1/2</td>
<td>12.42 (1.10)</td>
<td>13.28 (1.57)</td>
<td>12.87 (1.30)</td>
<td>14.20 (1.59)‡</td>
<td>12.80 (0.77)</td>
<td>14.65 (1.53)§</td>
<td>13.02 (1.15)</td>
<td>10.95 (1.40)†</td>
<td>10.43 (1.61)</td>
<td>10.43 (1.61)</td>
</tr>
<tr>
<td>EDD/BSA1/2</td>
<td>35.87 (2.44)</td>
<td>37.64 (2.21)‡</td>
<td>34.35 (2.58)</td>
<td>36.42 (2.59)‡</td>
<td>35.92 (2.59)</td>
<td>36.99 (2.54)‡</td>
<td>35.86 (2.12)</td>
<td>36.42 (2.59)‡</td>
<td>36.20 (2.45)</td>
<td>36.20 (2.45)</td>
</tr>
<tr>
<td>LVM/BSA3/2</td>
<td>70.23 (0.79)</td>
<td>83.38 (15.0)*</td>
<td>68.89 (13.35)</td>
<td>86.55 (17.64)‡</td>
<td>73.17 (9.31)</td>
<td>92.36 (16.86)‡</td>
<td>74.62 (10.12)</td>
<td>81.14 (17.64)‡</td>
<td>88.85 (18.33)</td>
<td>88.23 (21.35)</td>
</tr>
<tr>
<td>WT/EDD %</td>
<td>34.81 (4.35)</td>
<td>35.43 (4.35)*</td>
<td>37.56 (3.88)</td>
<td>39.15 (4.90)‡</td>
<td>35.85 (3.79)</td>
<td>39.79 (4.95)§</td>
<td>36.47 (4.27)</td>
<td>39.15 (4.90)‡</td>
<td>36.42 (4.25)</td>
<td>36.42 (4.25)</td>
</tr>
<tr>
<td>E/A</td>
<td>1.94 (0.32)</td>
<td>2.03 (0.35)</td>
<td>1.74 (0.23)</td>
<td>2.36 (0.61)‡</td>
<td>1.91 (0.38)</td>
<td>2.08 (0.49)*</td>
<td>1.47 (0.25)</td>
<td>1.77 (0.50)†</td>
<td>1.22 (0.34)</td>
<td>1.64 (0.36)†</td>
</tr>
</tbody>
</table>

Values are mean (SD).

*p<0.05, †p<0.02, ‡p<0.01, §p<0.001 compared with controls.

WT, Left ventricular wall thickness; BSA, body surface area; EDD, end diastolic diameter of the left ventricle; LVM, left ventricular muscle mass; E/A, proportion of the early and late diastolic filling peak velocity; CHLD, children; c, control, non-trained subjects; a, athletes; AJ, adolescent/junior; YAD, young adult; AD, adult; OAD, older adult.

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The present results show, however, that LVM hypertrophy and increased WT/EDD ratio and WT can no longer be regarded as unambiguous signs of an athletic heart. To characterise the condition of the heart, it is useful to estimate some functional signs, primarily the E/A quotient, which indicates diastolic function.

Two main questions arise with respect to the E/A quotient. Does regular physical training cause an increase in normal values in the young or not? Is regular physical training able to prevent the age dependent impairment of left ventricular diastolic function or not?

In answer to both questions, our results support a beneficial effect of regular physical training. However, the observations on the young adult athletes show that the type of physical training has an important role. No increase was found in the strength athletes, whereas, in the other groups, there was a more or less pronounced increase, indicating that dynamic, mostly endurance-type, training appears to be necessary to elicit a higher compliance of the left ventricle. This inference is in accordance with most other data: Pearson et al found no increase in weight lifters, and, in the studies reporting a positive effect of physical training, the athletes were mostly of the endurance type.

It is obvious that a few years of training is not enough to induce such modifications, and that is why there is no difference in childhood. In the 15–18 year and 19–30 year groups, however, an increase in the E/A quotient was evident.

With respect to public health, the most important fact seems to be that the E/A quotient is the only ratio where WT/EDD remains unchanged. A significant increase in this ratio is first observed in the young adult groups. This difference can be explained by the classic observation that regular physical training first induces ventricular dilatation, and muscular hypertrophy only begins to develop some time later.

Immediately after the competitive age range, training induced modifications in the structure and function of the heart seem to remain significant, and only the extent of the difference tends to decrease.

At an older age (45–60 years), morphological differences are likely to disappear, mostly as the result of an increase in WT even in untrained subjects. Morphological cardiac changes in the older group need a more detailed analysis, especially as few older athletes are engaged in such extensive and intense physical training as is usual at a younger age. This more detailed analysis should also be extended to the functional characteristics of the heart.

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Take home message

Numbers can lead and mislead, depending on how you use them!