The Athlete’s Heart and Arrhythmias: good, bad or does it exist?

Dr Andreas Wolff
The athlete’s heart: a historic perspective

• Osler 1892\(^1\): large heart - nature vs nurture
• Henschen 1898\(^2\): dilatation and hypertrophy and changes were normal and favourable
• Kirch 1935\(^3\): described hypertrophy in 35 athletes who experienced sudden death
• Kjellberg 1949\(^4\): chest radiograph. Relationship between cardiac volume and working capacity

\(^1\) Osler W. New York 1892; Appleton : pp635
\(^2\) Henschen  S. Mitt med Klin Uppsala 1898.
\(^3\) Krch E. Verh Dtsch Ges Inn Med 1935; 47: 73-98
\(^4\) Kjellberg SR et al. Acta Radiol 1949; 31;113-22
Athlete’s heart is generally regarded as a benign increase in cardiac mass, with specific circulatory and cardiac morphological alterations, that represents a physiological adaptation to systematic training.
Why are there concerns?

• Morphological changes and long-term health
• Higher incidence of sudden cardiac death in the athletic population
• Brady-arrhythmias
• Tachy-arrhythmias
What makes it difficult

- Different sports
- Different performance level
- Differences in race, sex and age
- Differentiating pathology
- Performance enhancing drugs
The athlete's heart: is big beautiful?

R J Shephard

Br. J. Sports Med. 1996;30;5-10
doi:10.1136/bjsm.30.1.5
The athlete’s heart

- Left ventricular wall thickening
- Left ventricular dilatation
- Left atrial dilatation
Figure 1  Distribution of LVWT in 3,500 Elite Athletes

We found that 1.5% of elite athletes showed a wall thickness >12 mm. LVWT = left ventricular wall thickness.
Figure 5 Differential diagnosis. Clinical criteria used to distinguish hypertrophic cardiomyopathy (HCM) from athlete’s heart when maximal left ventricular (LV) wall thickness falls within the shaded grey area of overlap, consistent with both diagnoses.

Figure 1. Distribution of left ventricular wall thicknesses in 720 junior elite athletes (black bars) and 250 controls (white bars).

Sharma S et al. JACC 2002; 40: 1431-6
A substantial minority (3%) of black athletes exhibited a left ventricular wall thickness $\geq 15$ mm, compared with none of the white athletes.

Basavarajaiah S et al. JACC 2008; 51: 2256-62
<table>
<thead>
<tr>
<th></th>
<th>Endurance-Trained Athletes</th>
<th>Combined Endurance- and Strength-Trained Athletes</th>
<th>Strength-Trained Athletes</th>
<th>Control Subjects</th>
<th>$P^*$</th>
</tr>
</thead>
<tbody>
<tr>
<td>RWT</td>
<td>0.389 (0.374–0.404 (n=413))</td>
<td>0.398 (0.374–0.421 (n=494))</td>
<td>0.442 (0.403–0.480 (n=544))</td>
<td>0.356 (0.343–0.369 (n=813))</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>LVIDd, mm</td>
<td>53.7 (52.8–54.6 (n=413))</td>
<td>56.2 (55.2–57.1 (n=494))</td>
<td>52.1 (50.6–53.6 (n=544))</td>
<td>49.6</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>PWTd, mm</td>
<td>10.3 (10.0–10.6 (n=413))</td>
<td>11.0 (10.3–11.6 (n=494))</td>
<td>11.0 (10.2–11.7 (n=544))</td>
<td>8.8</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>NSTd, mm</td>
<td>10.5 (10.1–10.9 (n=413))</td>
<td>11.3 (10.6–12.0 (n=494))</td>
<td>11.8 (10.9–12.7 (n=544))</td>
<td>8.8</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>LVM, g</td>
<td>249 (233–264 (n=413))</td>
<td>288 (260–316 (n=494))</td>
<td>288 (234–300 (n=544))</td>
<td>174</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>LVESF, %</td>
<td>68.8 (65.1–72.6 (n=177))</td>
<td>66.1 (62.9–69.3 (n=127))</td>
<td>66.3 (60.7–71.9 (n=73))</td>
<td>67.2</td>
<td>0.68</td>
</tr>
<tr>
<td>LVFS, %</td>
<td>34.4 (32.6–36.1 (n=204))</td>
<td>34.7 (32.7–36.8 (n=293))</td>
<td>35.7 (33.7–37.7 (n=276))</td>
<td>34.4</td>
<td>0.50</td>
</tr>
<tr>
<td>E/A ratio</td>
<td>2.20 (1.49–2.91 (n=93))</td>
<td>1.89 (1.46–2.31 (n=126))</td>
<td>2.11 (1.22–2.99 (n=44))</td>
<td>1.84</td>
<td>0.41</td>
</tr>
</tbody>
</table>

RWT indicates relative wall thickness; LVIDd, left ventricular end-diastolic internal diameter; PWTd, diastolic posterior wall thickness; NSTd, diastolic interventricular septum thickness; LVM, left ventricular mass; LVESF, left ventricular ejection fraction; and LVFS, left ventricular fractional shortening. Values in parentheses are 95% confidence intervals and n indicates number of studies for which the respective variable for the subsequent group of athletes was reported.

*Mixed-model ANOVA.
Figure 1. LV end-diastolic cavity dimensions at peak training and after long-term detraining shown individually in the 40 elite athletes.

Figure 2. Maximum LV wall thicknesses at peak training and after long-term detraining shown individually in the 40 elite athletes.

Figure 1. Incidence and relative risk (RR) of sudden death (SD) among athletes (solid columns) and non-athletes (open columns) from cardiovascular and non-cardiovascular causes. Athletes had a 2.8 RR of cardiovascular SD (confidence interval [CI] 1.9 to 3.7; p < 0.001), as compared with a 1.7 RR of non-cardiovascular SD (CI 0.3 to 5.7; p = 0.39).
Corrado D et al. JAMA 2006; 296: 1593-601
Some cardio-vascular diseases associated with increased risk during athletic activity

- Cardiomyopathies
- Coronary anomalies
- Long-QT syndromes
- Brugada syndrome
- CPVT
- Valvular disease
- Coronary artery disease
**Figure 2.** Incidence and relative risk (RR) of sudden death (SD) for specific cardiovascular causes among athletes and non-athletes. ARVC = arrhythmogenic right ventricular cardiomyopathy; CAD = coronary artery disease; CCA = congenital coronary artery anomaly; MVP = mitral valve prolapse.
Figure 3. Cardiovascular deaths according to race, with respect to the number of white and nonwhite athletes with each disease. ARVC indicates arrhythmogenic right ventricular cardiomyopathy; HCM, hypertrophic cardiomyopathy; CAD, coronary artery disease; and MVP, mitral valve prolapse. Analysis excludes 55 athletes for whom race could not be established.

Ambulatory ECG recordings in endurance athletes

- Sinus pauses >2s 37% (5.7%)
- 1\textsuperscript{st} degree AV block 37% (14%)
- Wenckebach AV block 22.9% (5.7%)
- Mobitz type II AV block 8.6% (0%)
- Junctional rhythm 20% (0%)

\cite{1} Viitassalo MT et al. Br Heart J 1982; 47: 213-20
<table>
<thead>
<tr>
<th>Variable</th>
<th>Total (n = 175)</th>
<th>Group A 0 (n = 40)</th>
<th>Group B 1–100 (n = 71)</th>
<th>Group C 101–1,000 (n = 33)</th>
<th>Group D &gt;1,000 (n = 31)</th>
<th>p Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (yrs)</td>
<td>23.6 ± 6</td>
<td>23.4 ± 5.4</td>
<td>24.1 ± 6.1</td>
<td>23.8 ± 6</td>
<td>23.1 ± 5.5</td>
<td>0.41</td>
</tr>
<tr>
<td>Men/women</td>
<td>108/67</td>
<td>23/17</td>
<td>42/29</td>
<td>21/12</td>
<td>22/9</td>
<td>0.01*</td>
</tr>
<tr>
<td>Heart rate at rest (beats/min)</td>
<td>48.8 ± 6</td>
<td>49.3 ± 5.5</td>
<td>47.4 ± 5.4</td>
<td>49.6 ± 7</td>
<td>48.6 ± 5.1</td>
<td>0.62</td>
</tr>
<tr>
<td>PVCs</td>
<td>802 ± 2,308</td>
<td>0</td>
<td>21 ± 27</td>
<td>364 ± 254</td>
<td>4,390 ± 4,116</td>
<td>0.001</td>
</tr>
<tr>
<td>Couplets</td>
<td>23 (13%)</td>
<td>0</td>
<td>5 (7%)</td>
<td>9 (28%)</td>
<td>9 (30%)</td>
<td>0.017</td>
</tr>
<tr>
<td>NSVT</td>
<td>8 (5%)</td>
<td>0</td>
<td>4 (5%)</td>
<td>2 (6%)</td>
<td>2 (7%)</td>
<td>0.89</td>
</tr>
<tr>
<td>Ventricular septum (mm)</td>
<td>9.4 ± 1.3</td>
<td>9.4 ± 1.4</td>
<td>9.4 ± 1.7</td>
<td>9.5 ± 1</td>
<td>9.3 ± 1.1</td>
<td>0.11</td>
</tr>
<tr>
<td>Posterior wall thickness (mm)</td>
<td>9.2 ± 1.2</td>
<td>9.3 ± 1.3</td>
<td>9.4 ± 1.3</td>
<td>9.2 ± 1.1</td>
<td>8.93 ± 1</td>
<td>0.10</td>
</tr>
<tr>
<td>LV end diastolic diameter (mm)</td>
<td>53.6 ± 5</td>
<td>52.9 ± 4.1</td>
<td>53.7 ± 5.7</td>
<td>53.6 ± 5.3</td>
<td>54.5 ± 3.9</td>
<td>0.12</td>
</tr>
<tr>
<td>LV mass (g)</td>
<td>92 ± 57</td>
<td>188 ± 56</td>
<td>195 ± 66</td>
<td>191 ± 19</td>
<td>188 ± 43</td>
<td>0.59</td>
</tr>
<tr>
<td>LV mass index (g/m²)</td>
<td>99 ± 21</td>
<td>98.5 ± 21</td>
<td>100 ± 24</td>
<td>99 ± 19</td>
<td>98 ± 19</td>
<td>0.62</td>
</tr>
</tbody>
</table>

Data are expressed as mean ± SD or as number (percentage).
* Groups A and B versus group D.
† Groups C and D versus groups A and B.
Figure 1. Relation between the frequency of ventricular tachyarrhythmias on 24-hour Holter electrocardiographic monitoring and LV mass index in 175 elite athletes without cardiovascular disease.

Biffi A et al. JACC 2008; 101: 1792-5
Figure 1. Number of premature ventricular depolarizations (PVD), ventricular couplets, and bursts of non-sustained ventricular tachycardia (NSVT) during 24-h Holter electrocardiogram recording at peak training and after the period of deconditioning in 70 trained athletes.

Biffi A. et al. JACC 2004; 44: 1053-8
Atrial Fibrillation
Lone atrial fibrillation in vigorously exercising middle aged men: case-control study¹

- 300 top Finnish orienteers vs 495 controls
- Mean age 47 vs 49 years
- 10 year follow up
- Lone AF developed in 5.3% of orienteers vs 0.9% in control group
- Lower mortality rate in orienteers: 1.7% vs 8.5% in control group


Figure 2. The Kaplan-Meier survival curves for cumulated survival free of lone atrial fibrillation in sedentary men and marathon runners.

Annual incidence rate:
Marathon runners: 0.43/100
Control subjects: 0.11/100

Log rank P-value = 0.067
Possible mechanisms

• Influence of autonomic nervous system: up to 70% of AF episodes in GIRAFA were vagally mediated

• Structural remodelling of the atrium in athletes\(^1\): structural changes in non-athletes are a known substrate for AF

\(^1\)Pellicia A et al. JACC 2005; 46: 690-6
Conclusion

- Structural changes of the heart as a result of training remain within normal limits in the vast majority of athletes.
- Deconditioning reverses the changes of the athlete’s heart.
- Increased number of sudden deaths in athletes are caused by undetected cardio-vascular disease.
- Arrhythmias associated with the athlete’s heart appear to be benign.
- Long-term endurance training might lead to a higher incidence of atrial fibrillation later in life.
Thank you.

Questions:
AndreasWolff@nhs.net