Athletic Heart

Anthony DeMaria
Cardiac Remodeling with Exercise

Normal "Pre-training"
Cardiac Structure and Function

Endurance Training
- RV Dilation +/- Mild RVH
- Eccentric LV Hypertrophy
- Characteristic Adaptations
  - Mild to Moderate Eccentric LVH and RV dilation
  - Biplaral enlargement
  - Normal to slightly reduced resting LVEF
  - Normal or enhanced Early LV Diastolic Function
  - Normal or enhanced LV twisting / untwisting

Strength Training
- RV No Δ
- Concentric LV Hypertrophy
- Characteristic Adaptations
  - Mild concentric LVH but No RV remodeling
  - Normal to mildly enlarged left atrial size
  - Normal to hyperdynamic resting LVEF
  - Normal to slightly reduced early LV diastolic function
  - Compensatory increase in late LV diastolic function
LV and LA Dimensions in Athletes
LV and LA Dimensions in Athletes

A. Pelliccia et al. / Progress in Cardiovascular Diseases 54 (2012) 387–396

A. Male

B. Female

LVDd

LAd
LV Wall Thickness in Athletes

The diagram shows a histogram of maximum wall thickness (mm) for athletes, comparing men (blue) and women (orange). The x-axis represents maximum wall thickness in millimeters, while the y-axis shows the number of athletes. The bars indicate that the majority of athletes fall within the normal range, with a few cases above the upper limits. The notation 'above upper limits (2%)' is mentioned, indicating that 2% of the athletes have wall thicknesses exceeding the normal range.
LV Wall Thickness in Athletes

![Graphs showing LV wall thickness distribution for white and black athletes.](image)
Aortic Root Size in Athletes
Ao Size vs Normal for Sports
Aortic Size in Athletes: Meta-analysis

Aortic size in athletes is 3.2 mm greater at Sinus of Valsalva and 1.6 mm greater at Aortic Annulus than controls.
Morphologic Changes in Athletic Heart

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Athlete</th>
<th>Non-athlete</th>
</tr>
</thead>
<tbody>
<tr>
<td>Left ventricular morphology (Echo)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>IVSd (mm)</td>
<td>8–16</td>
<td>6–10</td>
</tr>
<tr>
<td>LVIDd (mm)</td>
<td>49–73</td>
<td>42–59</td>
</tr>
<tr>
<td>LVM (g)</td>
<td>113–618</td>
<td>88–224</td>
</tr>
<tr>
<td>Left ventricular volumes/EF (Echo)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>LVEDV (ml)</td>
<td>130–260</td>
<td>67–155</td>
</tr>
<tr>
<td>EF (%)</td>
<td>41–77</td>
<td>&gt;55</td>
</tr>
<tr>
<td>Tissue Doppler</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Sm (cm/s)</td>
<td>6.5–14</td>
<td>&gt;6</td>
</tr>
<tr>
<td>Em (cm/s)</td>
<td>7.5–16</td>
<td>&gt;8</td>
</tr>
<tr>
<td>LA size (mm)</td>
<td>22–55</td>
<td>30–40</td>
</tr>
<tr>
<td>Right ventricular function</td>
<td></td>
<td></td>
</tr>
<tr>
<td>RVFAC (%)</td>
<td>26–60</td>
<td>32–60</td>
</tr>
<tr>
<td>Volumes/EF</td>
<td></td>
<td></td>
</tr>
<tr>
<td>RVEDV (ml)</td>
<td>130–260</td>
<td>60–150</td>
</tr>
<tr>
<td>RVEF (%)</td>
<td>&gt;45</td>
<td>&gt;50</td>
</tr>
</tbody>
</table>

LVII and LVIDd, left ventricular internal dimension at diastole; LVM, left ventricular mass; LVIDd, left ventricular end-diastolic volume; EF, ejection fraction; Sm, peak systolic velocity; Em, peak early diastolic velocity; LA, left atrium; RVFAC, right ventricular functional area change; RVEDV, right ventricular end-diastolic volume; RVEF, right ventricular ejection fraction.
Determinants of Athletic Heart
Impact of Sport on Athletic Heart

![Bar chart showing the impact of various sports on LV dimensions.](chart_image)
ECG in Specific Sports

![Bar chart showing percentages of athletes with ECG patterns in various sports: Distinctly Abnormal, Mildly Abnormal, Normal or minor alterations.]
Arrhythmias in Athletic Heart
Sudden Death in Athletes: Causes

- Coronary artery anomalies (14%)
- LVH – Indeterminant (7%)
- Myocarditis (5%)
- Ruptured Ao Aneurysm (3%)
- ARVC (3%)
- Tunnelled coronary artery (3%)
- AS (2.5%)
- CAD (2.5%)
- DCM (2%, 9pts)
- Myxomatous MV (2%)
- Asthma (2%)
- Heat Stroke (1.5%)
- Drug Abuse (1%)
- LQTS (1%)
- Sarcoidosis (1%)
- Trauma (1%)
- Other (1.5%)
- Ruptured cerebral art. (1%)
Arrhythmogenic RV Cardiomyopathy
Athletic Trabeculations vs Non-compaction
Athletic Heart vs Cardiomyopathy

Figure 10. Differential diagnosis between athlete’s heart and cardiac disease. Gray zone of overlap between physiological hypertrophy and pathological cardiomyopathies (including myocarditis, HCM, and ARVC). Adapted from Maron1 with permission of the Massachusetts Medical Society. Copyright 2003.
# HCM vs Athletic Heart: Distinction

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<tr>
<th>Parameter</th>
<th>HCM</th>
<th>Athletic heart</th>
</tr>
</thead>
<tbody>
<tr>
<td>LV wall thickness and morphology</td>
<td>Can be &gt;12 mm; can be concentric or asymmetric across segments</td>
<td>Typically &lt;12 mm, especially in women; concentric</td>
</tr>
<tr>
<td>Diastolic LV cavity</td>
<td>&lt;45 mm (except in late, dilated phase)</td>
<td>&gt;55 mm</td>
</tr>
<tr>
<td>LA size</td>
<td>Enlarged</td>
<td>Normal</td>
</tr>
<tr>
<td>LV diastolic filling pattern</td>
<td>Impaired relaxation (E:A ratio &lt;1, prolonged diastolic deceleration time)</td>
<td>Normal</td>
</tr>
<tr>
<td>Response to deconditioning</td>
<td>None</td>
<td>LV wall thickness decreases</td>
</tr>
<tr>
<td>Family history of HCM</td>
<td>Present (except de novo mutations)</td>
<td>Absent</td>
</tr>
<tr>
<td>ECG findings</td>
<td>Very high QRS voltages; Q waves; deep negative T waves</td>
<td>Criteria for LVH but without unusual features</td>
</tr>
</tbody>
</table>

LA, left atrium; LV, left ventricular; LVH, left ventricular hypertrophy.
Echo in Athlete vs HCM
# CMR in Cardiomyopathy

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<th>Cardiomyopathy</th>
<th>Typical pattern of fibrosis seen on CMR which allows differentiation from Athletes Heart</th>
</tr>
</thead>
<tbody>
<tr>
<td>HCM</td>
<td>Classically, fibrosis at the junction of the right ventricle and interventricular septum</td>
</tr>
<tr>
<td>Ischaemic DCM</td>
<td>Subendocardial extending to transmural fibrosis, generally restricted to the perfusion territory of one coronary artery</td>
</tr>
<tr>
<td>Non-ischaemic DCM</td>
<td>Patchy, mid-wall distribution in 28%. Sub-endocardial pattern indistinguishable from ischaemic cardiomyopathy in 13%</td>
</tr>
<tr>
<td>ARVC</td>
<td>Differentiated from Athlete’s Heart as RV and LV show disproportionate changes.</td>
</tr>
<tr>
<td>LVNC</td>
<td>Non-compacted myocardium</td>
</tr>
<tr>
<td>Myocarditis</td>
<td>Differentiated from Athlete’s Heart as significant fibrosis in 55% of patients, which may occupy up to 5% of LV myocardium</td>
</tr>
<tr>
<td></td>
<td>Most commonly fibrosis has been shown to involve the epicardium of the inferior lateral wall.</td>
</tr>
<tr>
<td></td>
<td>Differentiated from Athlete’s Heart due to lack of overt arrhythmias or classical symptoms (palpitations, presyncope or syncope)</td>
</tr>
</tbody>
</table>

CMR, cardiovascular magnetic resonance; HCM, hypertrophic cardiomyopathy; DCM, dilated cardiomyopathy; ARVC, arrhythmogenic right ventricular cardiomyopathy; LVNC, left ventricular non-compaction.
CMR of Athletic Heart

Steady State

Late Enhancement
Detraining Effects on Athletic Heart
Detraining to Detect HCM

Fig 6. Role of CMR in the evaluation of athletes with borderline LV wall measurements. An asymptomatic 19-year-old US collegiate basketball player was identified on preparticipation evaluation to have an abnormal 12-lead ECG (A). During the initial cardiovascular evaluation, CMR demonstrated a focal area of increased LV wall thickness (ie, 14 mm) in the posterior septum at mid-LV level (B; asterisk). After a 3-month period of deconditioning from competitive sport and training, a repeated CMR showed no change in the posterior septal thickness (C). In addition, on contrast-enhanced CMR images (D), an area of LGE was present in the area of mild LV wall thickening. The presence of LGE and an area of LV hypertrophy confined to posterior septum, unchanged in thickness after athletic deconditioning, support the diagnosis of HCM in this athlete. Abbreviations: VS, ventricular septum; PW, posterior free wall; LV, LV cavity; LA, left atrium.
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