The Athlete’s Heart
Role of Echo

Neil J. Weissman, MD
MedStar Health Research Institute
&
Professor of Medicine
Georgetown University
Washington, D.C.

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For a full list, visit www.EchoCoreLab.org
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Co-director MGH Heart Center Corrigan
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Massachusetts General Hospital
Outline

• Principles of Exercise-induced Cardiac Remodeling
  – Endurance vs. Strength training

• Healthy vs. Diseased (Athlete’s heart vs. pathology)
  1. LV chamber enlargement
  2. RV chamber enlargement
  3. LV wall thickening (gray zone hypertrophy)

• Is there a role for Echo in Screening Athletes?
  • Identification and Prevention of Sudden Cardiac Death

Background: Sport-Specific Physiology

Endurance Activities

Sustained ↑ CO
  • 4 to 5 times rest
  • ↑↑↑ HR & ↑ SV
  • Vasodilation

Strength Activities

Repetitive ↑ SBP
  • Systolic BP > 200 mmHg
  • Skeletal Muscle Contraction
  • ↑ LV Afterload
Exercise-Induced Cardiac Remodeling

Uncertainty #1: LV Dilatation
1309 Athletes in Diverse Sports (soccer, gymnastic, rowing)

LV Chamber Enlargement

Table 4: Echocardiographic findings from the study population of university athletes

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Male (n = 309)</th>
<th>Physiologic remodeling (n = 91)</th>
<th>Female (n = 187)</th>
<th>Physiologic remodeling (n = 79)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Structural parameters</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Interventricular septal thickness (mm)</td>
<td>9.8 ± 0.9</td>
<td>11.6 ± 0.5</td>
<td>8.3 ± 0.7</td>
<td>10.0 ± 0.5</td>
</tr>
<tr>
<td>LV posterior wall thickness (mm)</td>
<td>10.0 ± 1.2</td>
<td>11.8 ± 1.4</td>
<td>8.6 ± 1.1</td>
<td>10.7 ± 0.7</td>
</tr>
<tr>
<td>LV inner dimension at end-diastole (mm)</td>
<td>51 ± 3</td>
<td>57 ± 5</td>
<td>42 ± 4</td>
<td>54 ± 4</td>
</tr>
<tr>
<td>LA diameter (mm)</td>
<td>30 ± 5</td>
<td>36 ± 3</td>
<td>28 ± 4</td>
<td>33 ± 3</td>
</tr>
<tr>
<td>RV end-diastolic diameter (mm)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Functional parameters</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>LV ejection fraction (%)</td>
<td>65 ± 7</td>
<td>58 ± 4</td>
<td>68 ± 6</td>
<td>64 ± 6</td>
</tr>
<tr>
<td>Transmitral E wave (cm/sec)</td>
<td>96 ± 16</td>
<td>96 ± 13</td>
<td>91 ± 17</td>
<td>88 ± 12</td>
</tr>
<tr>
<td>A'</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

LA, Left atrial; PW, pulsed-wave.
Data are expressed as mean ± SD.
*P < .05 for comparison with male athletes in the normal cardiac structure and function group.
†P < .05 for comparison with male athletes in the physiologic remodeling group.

25% of US college athletes exceed gender recommended LVIDd limit


Olympic Athletes: LV Volumes

LV Adaptation in Endurance Athletes

- **Physiologic:**
  - Expected with endurance training.
  - Accompanied by proportionate increase in LV mass (Eccentric LVH).
  - Accompanied by normal to low normal resting LVEF (~50%).
    - TDI / Strain assessment with preserved or enhanced function.
    - Usually accompanied by “other” chamber enlargement (RV, LA).
    - LVIDd absolute “cut-offs” are not helpful.
  - When in doubt, exercise testing is very useful (confirm LV augmentation and document supranormal exercise capacity).

<table>
<thead>
<tr>
<th>Morphology</th>
<th>LVSD (cm)</th>
<th>LVIDd (mm)</th>
<th>LVEDd (mm)</th>
<th>LVEF (%)</th>
<th>Tissue Doppler</th>
<th>Sm (cm/sec)</th>
<th>V (cm/sec)</th>
</tr>
</thead>
<tbody>
<tr>
<td>LV function</td>
<td>Athletes</td>
<td>Nonathletes</td>
<td>Athletes</td>
<td>Nonathletes</td>
<td>Athletes</td>
<td>Nonathletes</td>
<td>Athletes</td>
</tr>
</tbody>
</table>
| EDV, End-diastolic volume; EF, ejection fraction; GLS, global longitudinal strain; LVSD, LV internal diameter at systole; LVIDd, LV internal diameter at diastole; LVM, LV mass; Sm, tissue Doppler imaging peak velocity at systole.

Uncertainty #2: RV Chamber Enlargement/Function

102 Endurance Athletes from the UK


Pre-Marathon

Post marathon

Neilan, Circulation 2006
### RV Function-Olympic Speedskaters

<table>
<thead>
<tr>
<th></th>
<th>Baseline</th>
<th>Post-exertion</th>
<th>P Value</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Ea (cm/sec)</strong></td>
<td>13.5±3.6</td>
<td>15.2±5.8</td>
<td>0.041</td>
</tr>
<tr>
<td><strong>Aa (cm/sec)</strong></td>
<td>8.6±1.5</td>
<td>9.2±3.0</td>
<td>0.47</td>
</tr>
<tr>
<td><strong>RV Area change</strong></td>
<td>0.35±0.13</td>
<td>0.43±0.13</td>
<td>0.007</td>
</tr>
<tr>
<td><strong>Strain Apex (%)</strong></td>
<td>-30±8</td>
<td>-29±7</td>
<td>0.66</td>
</tr>
<tr>
<td><strong>SR Apex (s⁻¹)</strong></td>
<td>-1.8±0.5</td>
<td>-2.5±1.2</td>
<td>0.038</td>
</tr>
</tbody>
</table>

Poh KK, Int J Cardiol 2008

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### RV Adaptations to training

**Physiologic:**
- RV enlargement expected with endurance training.
  - Global RV process without sacculation, aneurysmal dilation, segmental dysfunction, or fibrosis.
- RV dimensions absolute “cut-offs” are not usually helpful.
- Almost always associated with LV remodeling (concomitant LV enlargement but no RVH).
- May be accompanied by normal to low normal resting FAC / RVEF.
  - TDI / Strain assessment should be preserved or enhanced function.
  - If in doubt, comprehensive exercise testing
  - RV demonstrates contractile reserve
Uncertainty #3: Thick LV Walls

Least frequent but most problematic issue. Expected with strength (isometric) training.

Gray Zone LVH: 13 – 15 mm
Challenge: distinguish EICR from HCM
Especially since HCM is leading cause of exercise-related sudden death

LV Wall Thickness in 600 Female Athletes

Pelliccia
JAMA 1996:276:211

Thick LV Walls

Adult Athletes
(mostly rowing, track, soccer)

Racial Differences in LV Remodeling in Highly Trained Athletes

• 300 Nationally Ranked Black Athletes compared to 300 Matched White Athletes and 150 B & W Sedentary people

• Blacks Athletes had Greater LV Thickness and Cavity Size
  – 16% BA and 4% WA had wall thickness > 12 mm
  – 3% BA and 0% WA had wall thickness > 15mm

• BA with LVH had enlarged LVs and normal diastolic function

Basavarajaiah JACC 2008;51:2256-62
Olympic Athletes: LV Mass

Baggish / Wood, 2008

Pathologic LVH (HCM) vs Physiologic LVH (Athletic Heart)

Maron et al, Circz 2006;114:1633
LV Cavity Size

28 athletes without CV disease and 25 untrained patients with HCM (matched for LV wall thickness 13 – 15 mm)

LVd < 54 mm differentiated HCM and Athlete’s heart

Caselli et al. Am J Cardiol 2014;114:1382-89

Other Distinguishing Features

Tissue Doppler (Diastolic Function)

E’ (septum) < 11.5 cm/s
(sens 81%, spec 61% for dx HCM)

Thick LV Walls

**Physiologic:**
- Physiologic concentric LVH is symmetric *without* regional variation.
  - Marked asymmetry is pathology until proven otherwise.
- Wall thickness “cut-offs” are VERY helpful.
  - Accurate absolute thicknesses >15 mm are pathologic until proven otherwise.
- E’ values may be helpful, but not diagnostic
- Exercise testing may be useful discriminator (rule out other causes of LVH, i.e. hypertensive BP response)
- Detraining may be necessary to arrive at a final diagnosis.

Outline

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Causes of Sudden Cardiac Death in Athletes

**Most Common:**
- Hypertrophic CMP
- Anomalous origin coronary artery

**Less Common:**
- Aortic Dilatation in Marfan
- Myocarditis

**Uncommon:**
- Arrhythmogenic RV Cardiomyopathy
- Atherosclerotic CAD
- Aortic Valve Stenosis

Utility of Screening Echo

- Incidence of SCD during sports varies from <1/100,000 athletes* to 2/100,000#
- In 2688 competitive athletes, 203 (7.5%) of echos were abnormal
  - Only in 4 athletes did it stop athletic activity (HCM mostly)
- NO consensus on what type of echo to perform (handheld, limited, full, etc)
- Cost effectiveness is determined by
  1) incidence of SCD related to sports practice
  2) Cost of the echo
  3) Years of potential life saved
  All of the above are either unknown or highly variable

*Corrado et al, JAMA 2006;296:1593
# Steubvuk et al, JACC 2011;57:1291
Conclusions

1) Exercise training is a potent stimuli for cardiac remodeling and contributes to the development of “athlete’s heart” morphology.

2) Understand the principles of exercise-induced cardiac remodeling.

3) The nature and magnitude of cardiac remodeling depends upon sporting discipline, gender, race, level of and duration of training (Endurance vs. Strength).

4) Echocardiographic techniques can help differentiate healthy adaptation from underlying pathology.

5) Echo can identify causes of SCD that are not caught with a screening ECG but the yield is still low and the cost-effectiveness is unknown.