The Athlete’s Heart

Critical Role of Echo

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For a full list, visit www.EchoCoreLab.org
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Director, Sports & Performance Cardiology
MedStar Heart & Vascular Institute
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. Aortic dilatation
  4. 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
Sport Classification

Levine et al. Circulation. 2015; 131(22)

Exercise-Induced Cardiac Remodeling

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


LV Chamber Enlargement

25% of US college athletes exceed gender recommended LVIDd limit

**Olympic Athletes: LV Volumes**


![Graphs showing LV volumes for rower and lifter](image)

### Table 2: LV characteristics in athletes and nonathletes

<table>
<thead>
<tr>
<th>Characteristic</th>
<th>Athletes</th>
<th>Nonathletes</th>
</tr>
</thead>
<tbody>
<tr>
<td>Morphology</td>
<td></td>
<td></td>
</tr>
<tr>
<td>LVEDV (ml/m²)</td>
<td>20-50</td>
<td>10-30</td>
</tr>
<tr>
<td>LV1Dd (mm)</td>
<td>49-65</td>
<td>42-59</td>
</tr>
<tr>
<td>LVM (g/m²)</td>
<td>113-400</td>
<td>55-224</td>
</tr>
<tr>
<td>EF (%)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>LV EF (%)</td>
<td>130-240</td>
<td>65-155</td>
</tr>
<tr>
<td>LV ES (%)</td>
<td>45-75</td>
<td>&gt;55</td>
</tr>
<tr>
<td>Tissue Doppler Sm (cm/sec)</td>
<td>6.5-14</td>
<td>&gt;6</td>
</tr>
<tr>
<td>Mechanical parameter</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Strain/strain rate</td>
<td>Similar to nonathletes (GLS &gt; -18%)</td>
<td></td>
</tr>
</tbody>
</table>

**Note:** EDV, End-diastolic volume; EF, ejection fraction; GLS, global longitudinal strain; LV1Dd, interventricular septal thickness at diastole; LV1Dd, LV internal diameter at diastole; LVM, LV mass; Sm, tissue Doppler imaging peak velocity at systole.

Is Concentric Remodeling Adaptive?

Further work required to see if concentric remodeling is pathologic rather than adaptive

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).
Uncertainty #2: RV Chamber Enlargement/Function

102 Endurance Athletes from the UK

![Graphs showing range of values for RV inflow and outflow dimensions](image)

Figure 2: Range of values for RV inflow dimension in endurance athletes (n = 102).

Figure 3: Range of values for RV proximal outflow dimension in endurance athletes (n = 102).


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Pre-Marathon  |  Post marathon

![Images comparing pre and post marathon conditions](image)

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>Ea (cm/sec)</td>
<td>13.5±3.6</td>
<td>15.2±5.8</td>
<td>0.041</td>
</tr>
<tr>
<td>Aa (cm/sec)</td>
<td>8.6±1.5</td>
<td>9.2±3.0</td>
<td>0.47</td>
</tr>
<tr>
<td>RV Area change</td>
<td>0.35±0.13</td>
<td>0.43±0.13</td>
<td>0.007</td>
</tr>
<tr>
<td>Strain Apex (%)</td>
<td>-30±8</td>
<td>-29±7</td>
<td>0.66</td>
</tr>
<tr>
<td>SR Apex (s⁻¹)</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

## Right Ventricular Remodeling in Elite Athletes

### Table 4. Upper Reference Values for Right Ventricular Measurements in Athletes, Corrected for Sex and Body Surface Area

<table>
<thead>
<tr>
<th></th>
<th>Male Athletes</th>
<th>Female Athletes</th>
</tr>
</thead>
<tbody>
<tr>
<td>RAA, cm² (cm²/m²)</td>
<td>28 (14)</td>
<td>24 (13)</td>
</tr>
<tr>
<td>RVEDA, cm² (cm²/m²)</td>
<td>39 (19)</td>
<td>32 (18)</td>
</tr>
<tr>
<td>RVOTP, mm (mm/m²)</td>
<td>40 (20)</td>
<td>37 (21)</td>
</tr>
<tr>
<td>RVOT1, mm (mm/m²)</td>
<td>43 (22)</td>
<td>40 (23)</td>
</tr>
<tr>
<td>RVOT2, mm (mm/m²)</td>
<td>32 (17)</td>
<td>29 (16)</td>
</tr>
<tr>
<td>RVD1, mm (mm/m²)</td>
<td>55 (28)</td>
<td>49 (28)</td>
</tr>
<tr>
<td>RVD2, mm (mm/m²)</td>
<td>47 (24)</td>
<td>43 (25)</td>
</tr>
<tr>
<td>RVD3, mm (mm/m²)</td>
<td>109 (56)</td>
<td>100 (57)</td>
</tr>
<tr>
<td>RVWT, mm (mm/m²)</td>
<td>6 (3)</td>
<td>5 (3)</td>
</tr>
</tbody>
</table>

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: Aorta’s in Athletes

526 NBA basketball players
BSA 2.38 ± 0.19 m²

- 36 % LVEDD ≥59mm
- Aortic root ≥40mm in 24 (4.6%) but never >42mm

Aortic root was larger in sports with high dynamic component in both sexes but, age, left ventricular mass and BSA were main predictors of aortic dimensions.

Aortic Root In Athletes

- Aortic root does not have same physiologic adaptation to training as other cardiac structures
- Aortic Root in healthy elite athletes is within established limits for the general population
- Marked dilation of the aortic root is not explained by height, BSA or training effect
- Aortic root size in lifelong endurance masters athletes has not been studied
Uncertainty #4: 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

Thick LV Walls

Table 4 Echocardiographic findings from the study population of university athletes

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Normal (n = 200)</th>
<th>Physiologic remodeling (n = 29)</th>
<th>Normal (n = 178)</th>
<th>Physiologic remodeling (n = 19)</th>
</tr>
</thead>
<tbody>
<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>10.6 ± 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 end-systolic anterior diameter (mm)</td>
<td>37 ± 5</td>
<td>39 ± 9</td>
<td>32 ± 3</td>
<td>38 ± 4</td>
</tr>
<tr>
<td>IV end-diastolic diameter (mm)</td>
<td>30 ± 5</td>
<td>36 ± 3</td>
<td>29 ± 4</td>
<td>33 ± 3</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>Tricuspid E' (cm/sec)</td>
<td>86 ± 16</td>
<td>94 ± 13</td>
<td>84 ± 17</td>
<td>98 ± 15</td>
</tr>
</tbody>
</table>

Not a single healthy college athlete with walls > 14 mm

Weiner et al., J Am Soc Echocardiogr 2012;25:568
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)

LVIDd <54 mm differentiated HCM and Athlete’s heart

Other Distinguishing Features

Tissue Doppler (Diastolic Function)

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

Two-dimensional speckle tracking echocardiography


Left Ventricular Untwisting

**Left Atrial Size and Function**

### Echocardiographic Characteristics

<table>
<thead>
<tr>
<th></th>
<th>Controls (20)</th>
<th>Athletes (20)</th>
<th>HCM (20)</th>
<th>P (ANOVA)</th>
</tr>
</thead>
<tbody>
<tr>
<td>DD (mm)</td>
<td>51 ± 4</td>
<td>62 ± 5*</td>
<td>58 ± 5*</td>
<td>&lt;0.01</td>
</tr>
<tr>
<td>SD (mm)</td>
<td>37 ± 3</td>
<td>44 ± 4</td>
<td>39 ± 4</td>
<td>NS</td>
</tr>
<tr>
<td>SW (mm)</td>
<td>9.2 ± 1</td>
<td>15 ± 2*</td>
<td>18 ± 3*</td>
<td>&lt;0.01</td>
</tr>
<tr>
<td>LVMI (g/m²)</td>
<td>78 ± 6</td>
<td>143 ± 12*</td>
<td>157 ± 15*</td>
<td>&lt;0.01</td>
</tr>
<tr>
<td>LVEF (%)</td>
<td>60 ± 5</td>
<td>57 ± 5</td>
<td>59 ± 5</td>
<td>NS</td>
</tr>
<tr>
<td>LV GLS (%)</td>
<td>-20 ± 2</td>
<td>-18 ± 2</td>
<td>-13 ± 3*</td>
<td>0.02</td>
</tr>
<tr>
<td>LV VTI (cm)</td>
<td>22 ± 3</td>
<td>27 ± 3</td>
<td>19 ± 4</td>
<td>NS</td>
</tr>
<tr>
<td>LA volume (mL/m²)</td>
<td>25 ± 2</td>
<td>40 ± 5*</td>
<td>45 ± 6*</td>
<td>&lt;0.01</td>
</tr>
<tr>
<td>LA area (cm²)</td>
<td>18 ± 3</td>
<td>29 ± 3</td>
<td>31 ± 4*</td>
<td>&lt;0.01</td>
</tr>
<tr>
<td>LA diameter (mm)</td>
<td>35 ± 4</td>
<td>49 ± 4*</td>
<td>49 ± 4*</td>
<td>&lt;0.01</td>
</tr>
<tr>
<td>LA emptying fraction</td>
<td>0.59 ± 0.12</td>
<td>0.61 ± 0.15</td>
<td>0.42 ± 0.08*</td>
<td>&lt;0.01</td>
</tr>
<tr>
<td>E-wave (cm/sec)</td>
<td>55 ± 4</td>
<td>65 ± 5*</td>
<td>69 ± 5*</td>
<td>0.04</td>
</tr>
<tr>
<td>A-wave (cm/sec)</td>
<td>40 ± 3</td>
<td>37 ± 3</td>
<td>88 ± 5**</td>
<td>&lt;0.01</td>
</tr>
<tr>
<td>E/A ratio</td>
<td>1.3 ± 0.4</td>
<td>1.7 ± 0.4</td>
<td>0.78 ± 0.2*</td>
<td>0.02</td>
</tr>
<tr>
<td>DT (msec)</td>
<td>188 ± 11</td>
<td>178 ± 15</td>
<td>190 ± 15</td>
<td>NS</td>
</tr>
<tr>
<td>e (cm/sec)</td>
<td>13.5 ± 2</td>
<td>14.4 ± 2</td>
<td>5.5 ± 1**</td>
<td>&lt;0.01</td>
</tr>
<tr>
<td>E/e ratio</td>
<td>4.1 ± 1</td>
<td>5.2 ± 1</td>
<td>12.8 ± 2**</td>
<td>0.01</td>
</tr>
</tbody>
</table>

*P < 0.05 versus controls after significant ANOVA, **P < 0.05 versus other groups after significant ANOVA.

DD = diastolic diameter; SD = systolic diameter; SW = septal wall; LVMI = left ventricular mass index; LVEF = left ventricular ejection fraction; LV GLS = left ventricular global longitudinal strain; LV VTI = left ventricular volume-time integral; LA = left atrium; E/A = mitral inflow waves ratio; e = mean mitral annulus tissue Doppler; NS = not significant.


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**Left Atrial Size and Function**

Pathologic LVH leads to left atrial dilatation with LA dysfunction.

In contrast, this study provides more support that left atrial dilatation as a result of endurance training is an adaptive and healthy physiologic response.

Peak LA Strain during LV Systole
Marker of LA reservoir function

Peak negative LA strain rate during LA contraction
Marker of LA contractile function

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)
  - GLS and rate of untwisting may be helpful
  - Detraining may be necessary to arrive at a final diagnosis.

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. Aortic Dilatation
  4. **LV wall thickening** (gray zone hypertrophy)

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