Hypertrophic Cardiomyopathy or « Athlete’s Heart »

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### Disclosure Statement of Financial Interest

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<thead>
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<th>Company</th>
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<tbody>
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The background

- 0.6/100,000 person-years

- Echocardiography is mandatory
  - Since February 2004
  - In high-level athletes
  - In young elite athletes

- Our responsibility
Cardiovascular causes of sudden death in young competitive athletes: USA

- 1866 athletes who died suddenly (19± 6 years)
- From 1980 to 2006
- 0.61/100.000 person-years
- **HCM = 36%** (251/690 cardiovascular death)

Cardiovascular causes of sudden death in young competitive athletes: Italy

Table 2 Causes of sudden deaths in athletes and non-athletes (aged ≤ 3)

<table>
<thead>
<tr>
<th>Cause</th>
<th>Athletes (n = 49) n (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Arrhythmogenic RV cardiomyopathy</td>
<td>11 (22.4)</td>
</tr>
<tr>
<td>Atherosclerotic coronary artery disease</td>
<td>9 (18.5)</td>
</tr>
<tr>
<td>Anomalous origin of coronary artery</td>
<td>6 (12.2)</td>
</tr>
<tr>
<td>Conduction system pathology</td>
<td>4 (8.2)</td>
</tr>
<tr>
<td>Mitral valve prolapse</td>
<td>5 (10.2)</td>
</tr>
<tr>
<td>HCM</td>
<td>1 (2)</td>
</tr>
<tr>
<td>Myocarditis</td>
<td>3 (6.1)</td>
</tr>
<tr>
<td>Myocardial bridge</td>
<td>2 (4)</td>
</tr>
<tr>
<td>Pulmonary thrombo-embolism</td>
<td>1 (2)</td>
</tr>
<tr>
<td>Dissecting aortic aneurysm</td>
<td>1 (2)</td>
</tr>
<tr>
<td>Dilated cardiomyopathy</td>
<td>1 (2)</td>
</tr>
<tr>
<td>Other</td>
<td>5 (10.2)</td>
</tr>
</tbody>
</table>

*P = 0.008 for the comparison with the athletes.
**P < 0.001 for the comparison with the athletes.
Modified from Corrado et al. 3

- 269 sudden death between 1979-1996
- 18% competitive athletes
- Aged 23,1 years


Corrado D et al. JAMA 2006
Low prevalence of HCM in athlete in Europe

3500 elite athletes caucasian
53 (1.5%) with LVH (LV thickness > 12 mm)
   50 physiological (LV dilatation, normal diastolic function)
   3 (0.8%) non dilated LV + T wave inversion
⇒ but finally physiological LVH

Pelliccia A et al. Eur Heart J 2006;27:2196-2200

Basavarajaiah S JACC 2008 51(10):1040-1
Athlete’s Heart

✓ Physiological hypertrophy depends on several factors
  ➢ training aerobic ⇒ left ventricular dilatation and harmonious walls hypertrophy
  ➢ training anaerobic ⇒ almost exclusive concentric hypertrophy

✓ Moderated modifications, close to normal values

<table>
<thead>
<tr>
<th></th>
<th>Male</th>
<th>Female</th>
</tr>
</thead>
<tbody>
<tr>
<td>LVIDd</td>
<td>63 mm (67 mm *)</td>
<td>60 mm (63 mm *)</td>
</tr>
<tr>
<td>LVWT</td>
<td>13 mm</td>
<td>&lt; 12 mm</td>
</tr>
<tr>
<td>LA</td>
<td>45 mm (47 mm *)</td>
<td>43 mm (45 mm *)</td>
</tr>
</tbody>
</table>

LVWT : +2 to 3 mm
LVIDd: +3 to 6 mm

Pluim BM et al, Circulation, 2000
Left Ventricular Hypertrophy in athletes

- Physiological LVH occurs in around 2% of highly trained athletes (Maron BJ in editorial Heart 2005;91:1380-1382)

- LVH can create diagnostic difficulty in differentiating physiologic LVH and morphological mild HCM

- > 10 hours of training / week
- No family history of HCM, no symptom
- VO2max > 50 ml/kg/min or 120% of that predicted

- Not old athlete
  - 40 elite male athletes (LVED ≥ 60 mm, IVST ≥ 13 mm)
  - Deconditionning: 5.6 ± 3.8 years
    - Maximum wall thickness by 15% (12±1.3 to 10.1±0.8 mm)
    - LV cavity dimension by 7%
  - 22% LVED ≥ 60mm
  - Thickness returned to normal in all athletes

Maron B J, Pelliccia A Circulation 2006;114:1633-1644
Distribution of left ventricular thickness in series relating to male athletes

The diagram shows the distribution of left ventricular thickness (LVWT) in athletes compared to those with hypertrophic cardiomyopathy (HCM). The "grey zone" represents the range of LVWT where differentiation between athlete’s heart and HCM is challenging.

- **Athlete’s Heart**
- **HCM**

**LVWT threshold for HCM**
- Usual clinical diagnostic: max LVWT ≥ 15 mm
- Mildly increased max LVWT of 13 to 14 mm

References:
- Pluim BM et al, Circulation, 2000, 101: 336-44

-1 mm IVS and PWT ⇒ mass index from 108 to 92 g/m²
Left Ventricular Hypertrophy in athletes

Effect of specific sports training on LV cavity dimension or wall thickness in elite athletes, representing 27 different sporting disciplines.
Distribution of LVWT in athletes: the female athlete

- 240 nationally ranked black female athletes and 200 white athletes
- 21 ± 4.6 years

- LVWT: 9.2 ± 1.2 vs 8.6 ± 1.2 mm (p < 0.001)
- Only 8 black female athletes (3%) > 11mm
- None white > 11mm

Rawlins J Circulation 2010;121:1078-1085

- 1000 Italian female athletes
- LVWT maxi :12 mm

Distribution of LVWT in athletes: the junior elite athlete

☑ 720 junior elite athletes vs 250 teenager
   ➢ 15.7 ± 1.4 years and 75% male
☑ LVWT: 9.5 ± 1.7 vs 8.4 ± 1.4
☑ 3 males (0.4%) > 12 mm
   ➢ > 16 years
☑ No female > 11 mm

Sharma S JACC 2002;40:1431-6

☑ 125 teenager athletes
   ➢ 12-16 years
☑ LVWT: 8.1 ± 1.2 mm
   ➢ None > 12 mm
   ➢ Only 1 (0.9%) 11mm
   ➢ 97% girls ≤ 9mm

Our personal serie
Distribution of LVWT in athletes: the elite male

300 nationally ranked black male athletes vs 300 highly trained white male athletes

- Our personal serie
  - 200 athletes
  - LVWT: 9.5 ± 1.5 (6.0-12.8)
  - No > 13 mm

- Basavarajaiah S J Am Coll Cardiol 2008;51:2256-62

- Kervio G et al. 2008

- 1232 elite athletes
  - 357 females
  - 875 males

- 25% of elite black athletes
- 18% of elite white athletes
- 3% of black athletes
- 4% of white athletes
## Contribution to the diagnosis of LVH in Athletes

<table>
<thead>
<tr>
<th></th>
<th>AH Athlete's Heart</th>
<th>HCM</th>
<th>References</th>
</tr>
</thead>
<tbody>
<tr>
<td>Concentric hypertrophy</td>
<td>+/-</td>
<td>+/-</td>
<td>Shapiro LM, Eur Heart J, 1985</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Symmetrical 56% vs 22%</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Asymmetrical 34% vs 78%</td>
</tr>
<tr>
<td>IVSTd/PWTd &gt; 1,5</td>
<td></td>
<td>++</td>
<td>Urhausen A, Sports Med, 1999</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>male &lt; 1,4 / female &lt; 1,3</td>
</tr>
<tr>
<td>LVEDd &lt; 45 mm</td>
<td></td>
<td>+++</td>
<td>Pluim BM et al, Circulation, 2000</td>
</tr>
<tr>
<td>LVEDd &gt; 55 mm</td>
<td>++++</td>
<td>-</td>
<td>Fagard RH, Int.J.Sports Med 1996</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Abergel. E. JACC 2004; 44:144</td>
</tr>
<tr>
<td>LA &gt; 45 mm</td>
<td></td>
<td>+</td>
<td>Lewis JF et al. Br Heart J 1992</td>
</tr>
</tbody>
</table>
## Contribution to the diagnosis of LVH in Athletes

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<th>HCM</th>
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</tr>
</thead>
<tbody>
<tr>
<td>Abnormal mitral flow</td>
<td></td>
<td>++</td>
<td><strong>Pellicia A et al. Eur Heart J 2005</strong> Normal study is consistent with either HCM and athlete’s heart</td>
</tr>
<tr>
<td>(≤ 40 years)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Out flow obstruction at rest</td>
<td></td>
<td>+</td>
<td><strong>Maron BJ, et al. Circulation 1995</strong> No consensus on the cut-off value off the intra-ventricular gradient during exercice</td>
</tr>
<tr>
<td>Reduction in wall thickness over short deconditioning period</td>
<td>+++</td>
<td></td>
<td><strong>Pellicia A, Maron BJ et al. Circulation 2002</strong> <strong>Pellicia A, N Engl J Med, 1991</strong> 6 athletes <strong>13,8 ± 0,9 mm</strong> vs <strong>10,5 ± 0,4</strong></td>
</tr>
</tbody>
</table>
Diastolic left ventricular function: Mitral e’ (pulsed DTI)

- 35 athletes (22 years) long distance competitive swimmer
  - Ea (cm/s): 28 +/- 6 vs 16 +/- 4 cm/s
  
  *Pio Caso* et al.: Am J Cardiol 2002

- 18 highly trained rowing athletes (20.7 years)
  - Ea (cm/s): 19.2 +/- 3.8 vs 14.8 +/- 3.5 cm/s
  

- 650 ATE and ATP
  - e’ 16 ± 5 cm/s ant-lateral
  - e’ 14 ± 3 cm/s infer-septal
  
  *D’Andrea A.* JASE 2010;23:1281

- 100 handball players
  - e’ 16.6 ± 3.4 cm/s ant-lateral
  - e’ 13.2 ± 2.8 cm/s infer-septal
  
  *Butz T* EJCPR 2010;17:342

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**HCM**

- Ea < 9 cm/s  
  *Vinereanu D.* et al. Am J Cardiol 2001 (1) 88:53-8

- Ea < 8-10 cm/s  

- Ea < 12.5 cm/s  
Mitral e’ (pulsed DTI) in 200 elite athletes

\[ e' : 18.6 \pm 3.0 \text{ cm/s} \ (10-27.5) \]

\[ E/e' : 5.2 \pm 1.0 \ (2.2-8.4) \]

Griffet V Arch Mal Cœur Vaiss 2007;100(10):809-15
Mitral e’/a’ and mitral s’ (pulsed DTI)

- 650 athletes
  - 350 endurance athletes (ATE)
  - 280 power athletes (ATP)

- e’ and a’ inferoseptal and anterolateral sites
- e’/a’ > 1 (both sites) in 100%
  - D’Andrea A. JASE 2010;23:1281

- 60 subjects (HCM, HTA, athletes) pathologic LVH
  - s’ mean (4 sites) < 9 cm/s, Se=87%, Sp=97%
  - Vinereanu D AJC 2001;88:53

- 100 handball players infero-septal and antero-lateral sites
  - none of the athlete with s’<9 cm/s and e’<9 cm/s at any sites of the mitral annulus
  - Cardim N. JASE 2003;16:223

Pathologic LVH

Mitral e’ (inferior or lateral) < 16 cm/s  Se=100%  Sp=95%

Mitral s’ mean (4 sites) < 9 cm/s  Se=73%  Sp=97%
Triathlete  37 years, IVWT: 15 mm
2D strain: speckle tracking
Regional and global longitudinal strain

- Regional longitudinal strain
- soccer/HCM/controls
- GLS
  -16.9% athletes vs -16.3% HCM vs -21.3% controls

Richand V. Am J Cardiol 2007; 100: 128-32

- 20 athletes, 15 HCM and 18 controls
- GLS -15.2% vs -8.1% vs -16.0%
- **Cut off -10%** Se à 86% et Sp à 95%
- Combination of DTI (s’+e’/2) and 2DS (GLS) cut off values
  - Se=100%  Sp=95%

BUTZ Th Int.J Cardiovasc Imaging 2010;27:91-100

- 650 athletes (280 ATP and 370 ATE)
- LV GLS -17% controls, -17.2% ATP, -18.1% ATE
  - 90% of athletes: GLS ≤-16% s’≥10 cm/s, e’≥16 cm/s

D’Andrea A. JASE 2010;23:1281
Rugbyman: LVWT≤13 mm, e’= 9,55 cm/s, GLS= -14%

45 bodybuilders vs controls

<table>
<thead>
<tr>
<th></th>
<th>IVWT (mm)</th>
<th>2DS (GLS%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>25 steroids +</td>
<td>12,3</td>
<td>-15 ± 5</td>
</tr>
<tr>
<td>20 steroids -</td>
<td>11,2</td>
<td>-20 ± 6</td>
</tr>
<tr>
<td>25 controls</td>
<td>9,3</td>
<td>-19 ± 6</td>
</tr>
</tbody>
</table>

D’Andréa Br. J. Sport Méd.
MRI may be useful

- Validation of LV measurements
- Segmental LV hypertrophy (apex, anterolateral, inferoseptal...)
- Late gadolinium enhancement
- Area of necrosis or fibrosis

Athlete’s Heart or HCM?

- **Male athlete**
  - ≤15 mm white
  - ≤16 mm black

- **Female athlete**
  - ≤11 mm white
  - ≤13 mm black

- **Teenager (12-18)**
  - ≤11 mm female
  - ≤12 mm male

- > 10 hours of training / week
- No symptom,
- No family history of HCM
- VO2max > 50 ml/kg/min or 110% predicted

- **HCM**
  - LVWT ≥15mm
  - or ≥13 mm (mild phenotypic expression)
Grey zone 13-15 mm
Athlete’s heart

- LV cavity > 55mm
- Normal mitral valve
  - Morphology, position
- No out flow obstruction
  - No SAM
- E/A > 1 and e’/a’>1
- Mitral e’ > 16 cm/s
- Mitral s’ > 9 cm/s
- Global Longitudinal Strain < -16%
À VOS AGENDAS

CONGRÈS
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