Athlete’s Heart vs. Cardiomyopathy

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No disclosures
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Official Cardiologist to the New York Jets
Questions asked of Echo

- Is increased wall thickness physiologic or pathologic?
- Are increased dimensions physiologic or pathologic?
- Is “reduced” function physiologic or pathologic?
Is this normal or abnormal?

• Can this patient play sports (make a living, take a scholarship)?
• Does this patient need a defibrillator?
• Are there genetic/family implications to my decision?
• What is the prognosis?
CAUSE OF SCA IN YOUNG ATHLETES
(N=387, BASED ON AUTOPSY REPORTS)

- Hypertrophic Cardiomyopathy: 26%
- Commotio Cordis: 20%
- Coronary Anomalies: 14%
- LVH - possible HCM: 7%
- Myocarditis: 5%
- Marfan Syndrome: 3%
- ARVC: 3%
- Aortic Stenosis: 3%
- CAD: 3%
- Dilated CM: 2%
- Long QT: 1%

Maron BJ, NEJM 2004
Causes of SCD

- Structural
- Electrical
- Other
  - Commotio cordis
  - Myocarditis, dcm
Corrado: J Am Coll Card 2003
The Changing Face of the American Athlete - Youth

Thanks to Mat Martinez, MD
The Changing Face of the American Athlete – High School

Thanks to Mat Martinez, MD
The Changing Face of the American Athlete - Collegiate

Thanks to Mat Martinez, MD
The Changing Face of the American Athlete - Masters
Atletes Come in All Shapes and Sizes
Pay attention to age, gender, race, body size and sport specific norms!
Reminder

Non-myopathic conditions affect athletes too

- CAD
- HTN
- BAV etc
The multi-modality cardiac imaging approach to the Athlete’s heart: an expert consensus of the European Association of Cardiovascular Imaging

Maurizio Galderisi¹, (Chair), Nuno Cardim², (Co-chair), Antonello D’Andrea³, Oliver Bruder⁴, Bernard Cosyns⁵, Laurent Davin⁶, Erwan Donal⁷, Thor Edvardsen⁸, Antonio Freitas⁹, Gilbert Habib¹⁰, Anastasia Kitsiou¹¹, Sven Plein¹², Steffen E. Petersen¹³, Bogdan A. Popescu¹⁴, Stephen Schroeder¹⁵, Christof Burgstahler¹⁶, and Patrizio Lancellotti¹⁷

Document Reviewers: Rosa Sicari, (Italy), Denisa Muraru, (Romania), Massimo Lombardi, (Italy), Raluca Dulgheru, (Romania), Andre La Gerche (Australia)
**Table 4**  *Average and upper limits of the main echocardiographic LV parameters in elite athletes (*sample sizes≥400*)

<table>
<thead>
<tr>
<th>Authors</th>
<th>Journal</th>
<th>Number of athletes</th>
<th>Type of sport</th>
<th>Parameter</th>
<th>Average value</th>
<th>Upper limit</th>
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<tbody>
<tr>
<td>Pelliccia et al.</td>
<td>Ann Intern Med 1999;130:23–31</td>
<td>1309</td>
<td>Endurance/strength</td>
<td>LV end-diastolic diameter (adult male) (mm)</td>
<td>55</td>
<td>70</td>
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<tr>
<td>Pelliccia et al.</td>
<td>JAMA 1996;276:211–215</td>
<td>600</td>
<td>Endurance/strength</td>
<td>LV end-diastolic diameter (adolescent) (mm)</td>
<td>51</td>
<td>60</td>
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<td>Makki et al.</td>
<td>Heart 2005;91:495–499</td>
<td>900</td>
<td>Endurance</td>
<td>LV wall end-diastolic thickness (adult male) (mm)</td>
<td>10</td>
<td>16</td>
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<tr>
<td>Spirito et al.</td>
<td>Am J Cardiol 1994;74:802–806</td>
<td>947</td>
<td>Endurance/strength</td>
<td>LV wall end-diastolic thickness (adult female) (mm)</td>
<td>9.5</td>
<td>13</td>
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<td>Rawlins et al.</td>
<td>Circulation 2010;121:1078–1085</td>
<td>440</td>
<td>Endurance/strength</td>
<td>LV wall end-diastolic thickness (adolescent) (mm)</td>
<td>9.5</td>
<td>12</td>
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<td>Sharma et al.</td>
<td>J Am Coll Cardiol 2002;40:1431–1436</td>
<td>720</td>
<td>Endurance/strength</td>
<td>LV wall end-diastolic thickness (black athlete) (mm)</td>
<td>11.5</td>
<td>16</td>
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<td>Basavarajiah et al.</td>
<td>J Am Coll Cardiol 2008;51:2256–2262</td>
<td>300</td>
<td>Endurance/strength</td>
<td>LA diameter (male) (mm)</td>
<td>37</td>
<td>50</td>
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<td>Pelliccia et al.</td>
<td>J Am Coll Cardiol 2005;46:690–696</td>
<td>1777</td>
<td>Endurance/strength</td>
<td>LA diameter (female) (mm)</td>
<td>32</td>
<td>45</td>
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<td>D'Andrea et al.</td>
<td>Am Heart J 2010;159:1155–1161</td>
<td>650</td>
<td>Endurance/strength</td>
<td>LA volume index (male) (mL/m²)</td>
<td>28</td>
<td>36</td>
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<tr>
<td>D'Andrea et al.</td>
<td>J Am Soc Echocardiogr 2010;23:1281–1288</td>
<td>650</td>
<td>Endurance/strength</td>
<td>LA volume index (female) (mL/m²)</td>
<td>26.5</td>
<td>33</td>
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<td>IVS Tissue Doppler s (cm/s)</td>
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<td>IVS Tissue Doppler e' (cm/s)</td>
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<td>LV Tissue Doppler s (cm/s)</td>
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<td>20</td>
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<td>LV Tissue Doppler e' (cm/s)</td>
<td>16</td>
<td>22</td>
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<td></td>
<td>LV Tissue Doppler e'/a' (cm/s)</td>
<td>1.45</td>
<td>1.7</td>
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</tbody>
</table>
REVIEW

Echocardiography in the evaluation of athletes [version 1; referees: 2 approved, 1 approved with reservations]

Gonzalo Grazioli*, Maria Sanz*, Silvia Montserrat, Bàrbara Vidal, Marta Sitges

Cardiology Department, Hospital Clínic, Universitat de Barcelona, IDIBAPS, Institut d’Investigacions Biomèdiques August Pi i Sunyer, Barcelona, Catalonia, Spain

* Equal contributors
Exercise Physiology Basics

- Exercise requires oxygen
- Increased pulmonary oxygen uptake
- Increased cardiac output
- Increased peripheral oxygen extraction
Exercise Physiology Basics

- Exercise requires oxygen
- Increased pulmonary oxygen uptake
- Increased cardiac output
- Increased peripheral oxygen extraction
Cardiac Output = HR X Stroke Volume

Stroke volume = End-diastolic volume minus End-systolic volume

* In the absence of valve regurgitation or intracardiac shunts
• Cardiac output may increase 5-6 X with HR responsible for the majority of the change
• Max HR does not increase with exercise training (age-related)
• Stroke volume does increase with exercise training (resting and peak exercise)
  – SV increases because EDV ↑±↓ESV
2 Forms of Exercise Training (some overlap)

• Isotonic
  – Sustained increase in CO with normal or reduced SVR

• Isometric
  – Increased SVR and normal or slightly increased CO
Athlete’s Heart

• Well recognized that repetitive physical exercise causes adaptive changes in cardiac structure and function
  “Athlete’s Heart”

• Although historically some dispute as to whether changes were harmful, consensus is that this is a favorable adaptive response rather than pre-clinical disease
Athlete’s Heart

• Anatomic changes
• Functional changes
Left Ventricular Response

• Increased cavity size
• Increased wall thickness
  – Generally associated with increased cavity size
  – More pronounced in those who are large and Afro-Caribbean
• Morganroth hypothesis
  – Isotonic -> dilatation (eccentric LVH)
  – Isometric -> increased wall thickness (concentric LVH)
Similar Changes with RV
Normative Reference Values of Right Heart in Competitive Athletes: A Systematic Review and Meta-Analysis

Flavio D’Ascenzi, MD, PhD, FESC, Antonio Pelliccia, MD, FESC, Marco Solari, MD, Pietro Piu, PhD, Ferdinando Loiacono, MD, Francesca Anselmi, MD, Stefano Caselli, MD, PhD, FASE, FESC, Marta Focardi, MD, PhD, Marco Bonifazi, MD, and Sergio Mondillo, MD, Siena and Rome, Italy
Wall thickness
Distribution of Maximal Left-Ventricular-Wall Thicknesses in the 947 Elite Athletes.


1.7% ≥ 1.3 cm
Gender Matters

Race Matters

![Graph showing comparison between White Athletes and Black Athletes in terms of Left Ventricular Wall Thickness (mm)]

- **Gray Zone** indicates the range where values for both groups overlap.
Effect of specific sports training on LV cavity dimension or wall thickness in elite athletes, representing 27 different sporting disciplines.

Barry J. Maron, and Antonio Pelliccia Circulation. 2006;114:1633-1644
NFL 2011-2013

Percentiles
75% 90% 95% 99%
1.15 1.26 1.32 1.44

LVPW > 1.6: 0 (0%)

~ 10% ≥ 1.3 cm.

NFL data Courtesy of Dr Kovacs ACC 2013
From: Athletic Cardiac Remodeling in US Professional Basketball Players: Engel et al


~10% ≥ 1.3 cm.
Impact of Gender and Race

- Less remodeling in women (even with correcting smaller baseline heart sizes)
- More remodeling in blacks
  - LV wall thickness >12mm in 20% black men vs 4% whites
- Black women have thicker walls than white women
Chamber dimensions

LVEDD
Pro Cyclists LV chamber size

LVIDd >60m (51%)
LV chamber size in the NBA


All but 5 with normal EF > 50%
Effect of Sex and Sporting Discipline on LV Adaptation to Exercise

Gherardo Finocchiaro, MD, Harshil Dhutia, MBBS, Andrew D'Silva, MBBS, Aneil Malhotra, MBBS, Alexandros Steriotis, MD, PhD, Lynne Millar, MBBS, Keerthi Prakash, MBBS, Rajay Narain, MBBS, Michael Papadakis, MD, MBBS, Rajan Sharma, MD, MBBS, Sanjay Sharma, MD, MBChB
Relative Wall Thickness

> 0.42

Concentric Remodeling

Concentric Hypertrophy

≤ 0.42

Normal Geometry

Eccentric Hypertrophy

Left Ventricular Mass (g/m²)

≤ 95 in Males

≥ 95 in Males

≤ 115 in Females

> 115 in Females
Gherardo Finocchiaro et al. JIMG 2017;10:965-972
Gherardo Finocchiaro et al. JIMG 2017;10:965-972
Cardiac Remodeling in Response to 1 Year of Intensive Endurance Training

Armin Arbab-Zadeh, Merja Perhonen, Erin Howden, Ronald M. Peshock, Rong Zhang, Beverly Adams-Huet, Mark J. Haykowsky, Benjamin D. Levine

Download PDF  DOI: https://doi.org/10.1161/CIRCULATIONAHA.114.010775
Circulation. 2014;130:2152-2161
Originally published October 3, 2014
Average training impulse (TRIMP) scores per month for all subjects over the training year.

Changes in left ventricular (LV) mass (left) and right ventricular (RV) mass (right) measured by magnetic resonance imaging every 3 months during the 1-year training program.

Mean±SD changes in left ventricular end-diastolic volume (LVEDV; left) and right ventricular end-diastolic volume (RVEDV; middle) by magnetic resonance imaging measured every 3 months during the training program.

Systolic Function
LVEF/Systolic function

- Typically normal
- But may be borderline or mildly reduced (50-55%) leading to concern about dilated cardiomyopathy
- Role for stress echocardiography in establishing contractile reserve
- Strain also helpful
Echocardiographic tissue Doppler imaging (A and C) and speckle-tracking radial strain analysis (B and D) in 2 different athletic patients presenting with left ventricular hypertrophy.

Baggish A L, Wood M J Circulation 2011;123:2723-2735
Atria
Morphological and Functional Adaptation of Left and Right Atria Induced by Training in Highly Trained Female Athletes
Flavio D'Ascenzi, Antonio Pelliccia, Benedetta Maria Natali, Valerio Zaccà, Matteo Cameli, Federico Alvino, Angela Malandrino, Paola Palmitesta, Alessandro Zorzi, Domenico Corrado, Marco Bonifazi and Sergio Mondillo

Circ Cardiovasc Imaging. 2014;7:222-229; originally published online January 27, 2014; doi: 10.1161/CIRCIMAGING.113.001345
Left atrium

• In Italian series >20% had enlarged left atria (as measured by AP diameter)
  – No volume data

• Questionable association with supraventricular arrhythmias

LA AP diameter
LA volumes

From Galderisi et al EACVI Recommendations
Diastolic Function: Myopathy or Athlete’s Heart?
Diastolic Function

• Isotonic training
  – Enhanced relaxation

• Isometric training
  – Impaired or unchanged relaxation (less well studied)
• In athlete’s heart diastolic function is normal or super-normal
• In HCM, diastolic function is variably abnormal
Diastolic Function in the Athlete

- Increased early diastolic filling
  - E/A ratio > 1
- Normal deceleration time
  - 100 - 200 ms
- Normal isovolumetric relaxation time
  - < 100 ms.
Diastolic Function in HCM

• Decreased early diastolic filling
  – E/A ratio <0.5
• Lengthened deceleration time
  – >280 ms
• Ar-Ad > 30 ms
• Decreased annular e’
Wall thickness = 1.2 cm
Decel time = 187 ms
Athlete’s Heart
IVS = 1.4, PW = 1.2
Hypertrophic CM
When left atrial pressure is elevated
Diastolic abnormalities may be present prior to hypertrophy
Tissue Doppler Imaging Predicts the Development of Hypertrophic Cardiomyopathy in Subjects With Subclinical Disease

Sherif F. Naghieh, MD; Judy McFalls, RDQS; Denise Meyer, MT (ASCP); Rita Hill, RN; William A. Zoghbi, MD; James W. Tam, MD; Miguel A. Quiñones, MD; Robert Roberts, MD; A.J. Marian, MD

Background—Systolic (Sa) and diastolic (Ea) myocardial velocities measured by tissue Doppler (TD) imaging (TDI) recently were shown to be decreased in subjects who have mutations causing hypertrophic cardiomyopathy (HCM) but who do not have left ventricular (LV) hypertrophy. By studying these subjects at a later date, we sought to determine whether TDI predicts the subsequent evolution of the HCM phenotype.

Methods and Results—Serial 2D and Doppler echocardiography were performed in 12 subjects (age range, 17 to 51 years) with HCM-causing mutations on 2 occasions: before development of hypertrophy and 2 years later. Twelve age- and gender-matched family members without mutations were included as control subjects. In those with mutations, mean septal thickness and LV mass were 1.07±0.14 cm and 103.0±11 g at baseline, respectively, and increased to 1.30±0.36 cm and 193.0±78 g at follow-up (P<0.01), with 6 subjects satisfying HCM diagnostic criteria. Sa and Ea velocities in those with mutations were lower compared with control subjects at baseline and follow-up (lateral Sa, 15±1.2 versus 8.2±2.1 cm/s; lateral Ea, 16.5±2.8 versus 8.1±2.3 cm/s; P<0.01). At 2 years, left atrial volume and pulmonary venous flow indices of LV filling pressures increased, whereas TD early and late diastolic velocities decreased (all P<0.05) in those with the mutations. Control subjects had no significant interval changes of the above parameters.

Conclusions—Subsequent development of HCM in subjects with initially reduced TD velocities establishes TDI as a reliable method for early identification of HCM mutation carriers. (Circulation. 2003;108:395-398.)
Individual data points showing septal and lateral $Sa$ and $Ea$ velocities at baseline and follow-up in both groups: the 12 subjects who had inherited the causal mutations for HCM and the 12 individuals in the control group.

Aorta

- Pathologic enlargement typically not encountered (>4 cm)
- Inconsistent data on impact of training on aortic root size
From: **Athletic Cardiac Remodeling in US Professional Basketball Players**
Engel et al.
A word about complementary modalities
EKG
Athlete’s EKG

**Vagotonia**
- Sinus bradycardia
- Sinus arrhythmia
- First degree AVB
- ST-elevation
- Tall T waves

**Increased chamber size**
- Left ventricular hypertrophy
- Incomplete RBBB
- Left atrial enlargement
- Right atrial enlargement
MRI

• Assessment of LV/RV Mass, Dimensions
• Fibrosis
• Inflammation
• Pathognomonic findings in myopathies
Proposed Approach
Galderisi EACVI Recommendation

**Suspicion of ARVC**

- ECHO: Global and regional RV function
  - Normal
  - Abnormal
    - CMR-LGE: Task Force Criteria + CLINICAL FEATURES
      - ATHLETE
    - ARVC

**Suspicion of CAD**

- Exercise ECG
  - Exercise Stress ECHO
    - Normal
    - Abnormal
    - Uncertain

- ATHLETE
- CAD

Stress CMR Stress SPECT or CCT

If myocardial ischemia is detected by a first test, coronary angiography could be proposed without the performance of other tests of inducible ischemia.

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*Major Criteria*
- Regional RV akinesia or dyskinesia or dysynchronous RV contraction
- and 1 of the following:
  - Ratio of RV end-diastolic volume to BSA ≥1.10 mL/m² (male) or ≥1.00 mL/m² (female)
  - or RV ejection fraction ≤40%

*Minor Criteria:*
- Regional RV akinesia or dyskinesia or dysynchronous RV contraction
- and 1 of the following:
  - Ratio of RV end-diastolic volume to BSA ≥1.00 to <1.10 mL/m² (male) or ≥0.90 to <1.00 mL/m² (female)
  - or RV ejection fraction >40% to ≤45%
“Gray Zone” of LV Wall Thickness (13-15 mm)

HCM*

Athlete’s Heart

+ Unusual Patterns of LVH
+ LV Cavity < 45mm
+ LV Cavity > 55mm
+ Marked LA Enlargement
+ Bizarre ECG Patterns
+ Abnormal LV Filling
+ Female Gender

Thickness with Deconditioning

Family History of HCM

Max. VO₂ > 45 ml/kg/min, > 110% predicted
Sometimes even the experts are not sure

Deconditioning
Impact of extreme endurance activity
Circulation

Circulation: Cardiovascular Imaging

Alteration in left ventricular strains and torsional mechanics after ultra-long duration exercise in athletes
Stéphane Nottin, Grégory Doucende, Iris Schuster-beck, Michel Dauzat, and Philippe Obert
CIRCULATIONAHA/2008/811273 [R2]
Figure 1. LV basal and apical circumferential and radial strains and LV longitudinal strains before (•) and after (○) the race.

Sports Cardiologist

Athlete Cardiac Assessment

Sports Medicine Team
ATC / Ex Phys

CV Imaging

EP
Take home messages

• Athlete’s heart may have altered anatomy and, to a lesser degree, function

• Published norms provide guidance but additional interventions (stress, deconditioning) may be essential
• Multimodality approach is essential
  – Advanced EKG interpretive skills
  – MRI
• Meticulous echocardiography
  – Precise measurements
  – Strain
  – Stress echocardiography
• Specialized centers important