Differentiating Athlete’s Heart From Cardiac Pathology.

Professor Sanjay Sharma

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Background: Causes of SCD in Sport

- Congenital + Anatomic: 37%
- Cardiomyopathies: 35%
- Arrhythmias: 9%
- Infectious: 4%
- Degenerative: 1%
- Undetermined: 14%
- "Normal heart": 3%
Background: Relative Risk of SCD

- **Athletes**: SD/100,000 person yrs = 2.5
- **Non-athletes**: SD/100,000 person yrs = 1.0

Cardiac Risk in the Young Centre for Sports Cardiology
Clinical and family history
Cardiac auscultation
12-lead ECG/SAECG
Echocardiography/CMR
24 hour ECG
Exercise stress test
Pharmacological provocation tests
Electrophysiological tests

Identify most conditions
Athlete’s Heart

**ELECTRICAL**
- Bradycardia
- Repolarisation anomalies
- Voltage criteria for chamber enlargement

**FUNCTIONAL**
- Enhanced diastolic filling
- Augmentation of stroke volume

**STRUCTURAL**
- Increased chamber wall thickness and cavity size
## Absolute Cardiac Dimensions in Athletes

Results of 28 M mode echocardiographic studies in > 1000 male athletes (Maron JACC 1986)

<table>
<thead>
<tr>
<th>Echo variable</th>
<th>Controls Mean</th>
<th>Athletes Mean</th>
<th>%Diff</th>
</tr>
</thead>
<tbody>
<tr>
<td>Septal thickness (mm)</td>
<td>9.1</td>
<td>10.4</td>
<td>14.3</td>
</tr>
<tr>
<td>Post wall thickness (mm)</td>
<td>9.0</td>
<td>10.7</td>
<td>18.9</td>
</tr>
<tr>
<td>LVED cavity (mm)</td>
<td>49.1</td>
<td>53.9</td>
<td>9.8</td>
</tr>
<tr>
<td>LV Mass g/m²</td>
<td>175</td>
<td>256</td>
<td>46.3</td>
</tr>
<tr>
<td>RVED cavity (mm)</td>
<td>17.7</td>
<td>22.0</td>
<td>24.3</td>
</tr>
</tbody>
</table>
Determinants of Cardiac Dimensions in Athletes

- Age
- Size
- Ethnicity
- Gender
- Anabolic Drugs
- Type of sport
- Inherited Cardiomyopathy or ion channel disorder
Left Ventricular Cavity Dimensions in Highly Trained Athletes

45%

% 60 80 100 120
LV Cavity

Males
Females

Cardiac Risk in the Young Centre for Sports Cardiology
<table>
<thead>
<tr>
<th>Cardiac Dimension</th>
<th>Percentage</th>
</tr>
</thead>
<tbody>
<tr>
<td>LV cavity &gt; 54 mm</td>
<td>48%</td>
</tr>
<tr>
<td>LV cavity &gt; 60 mm</td>
<td>24%</td>
</tr>
<tr>
<td>RVD1 &gt; 44 m</td>
<td>57%</td>
</tr>
<tr>
<td>RVOT 1 &gt; 34 mm</td>
<td>40%</td>
</tr>
</tbody>
</table>
Prevalence of Hypertrophic Cardiomyopathy in Highly Trained Athletes

Relevance to Pre-Participation Screening

Sandeep Basavarajaiah, MBBS, MRCP,† Matthew Wilson, MSc, MPHIL,‡ Gregory Whyte, PHD,‡ Ajay Shah, PHD, FRCP,* William McKenna, DSc, FRCP, FESC, FACC,§ Sanjay Sharma, BSc (HONS), MD, FRCP*†

Figure 1 Distribution of LVWT in 3,500 Elite Athletes

We found that 1.5% of elite athletes showed a wall thickness >12 mm. LVWT = left ventricular wall thickness.
Hypertrophic Cardiomyopathy
The Challenge

Athlete’s Heart

HCM

Left Ventricular Hypertrophy 13-16 mm
Determinants of Left Ventricular Hypertrophy

History

Demographics

Echocardiography

12-lead ECG

Cardiopulmonary Exercise Testing

Cardiac Magnetic Resonance Imaging

Detraining

Evaluation of first degree relatives/Genetic testing
Determinants of Left Ventricular Hypertrophy

- Male sex
- Large BSA
- Endurance Sports
- Adult athletes
- Black Ethnicity
Role of Echocardiography

Determine the Magnitude of LVH
Assess the Pattern of LVH
Compare LVH relative to LV cavity size
Identify left ventricular outflow obstruction
Measure indices of diastolic function
Measure indices of longitudinal systolic function
The Magnitude of Left Ventricular Hypertrophy
Prevalence of Hypertrophic Cardiomyopathy in Highly Trained Athletes

Relevance to Pre-Participation Screening

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Figure 1

Distribution of LVWT in 3,500 Elite Athletes

We found that 1.5% of elite athletes showed a wall thickness >12 mm. LVWT = left ventricular wall thickness.
The Impact of Ethnicity
Distribution of Left Ventricular Wall Thickness in Black Athletes and White Athletes
Left Ventricular Wall Thickness in 240 Black and 200 White Elite Athletes

No of Athletes (%)

Maximal Left ventricular Wall Thickness (mm)

Black
White

3%

Cardiac Risk in the Young
Centre for Sports Cardiology
The Pattern of Left Ventricular Hypertrophy
Pattern of Left Ventricular Hypertrophy

HCM

Athlete’s Heart
Left Ventricular Cavity Size in Relation to Wall Thickness
Left ventricular wall thickness relative to cavity size

Physiological LVH

HCM
Indices of Diastolic Function
Diastolic Function in HCM

Normal

Impaired Relaxation

Restrictive Filling
## Diastolic Function in HCM

<table>
<thead>
<tr>
<th></th>
<th>Physiological LVH</th>
<th>HCM</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>E wave</strong></td>
<td>Increased/Normal</td>
<td>Decreased</td>
</tr>
<tr>
<td><strong>A wave</strong></td>
<td>Normal</td>
<td>Increased</td>
</tr>
<tr>
<td><strong>E/A ratio</strong></td>
<td>&gt; 1</td>
<td>&lt; 1</td>
</tr>
<tr>
<td><strong>E deceleration time</strong></td>
<td>Reduced</td>
<td>Increased</td>
</tr>
<tr>
<td><strong>IVRT</strong></td>
<td>Reduced</td>
<td>Increased</td>
</tr>
<tr>
<td><strong>E’</strong></td>
<td>Increased</td>
<td>Reduced</td>
</tr>
<tr>
<td><strong>E/E’</strong></td>
<td>&lt; 8</td>
<td>&gt; 12</td>
</tr>
<tr>
<td><strong>Pulm Vein</strong></td>
<td>S/D &gt; 1</td>
<td>S/D &lt; 1</td>
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</table>
Indices of Systolic Function
### Systolic Function in HCM

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<th>Physiological LVH</th>
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<tbody>
<tr>
<td>EF</td>
<td>Normal/Decreased</td>
</tr>
<tr>
<td>FS</td>
<td>Normal/Decreased</td>
</tr>
<tr>
<td>$E_s$</td>
<td>Increased</td>
</tr>
<tr>
<td>TDI L strain</td>
<td>Homogenous</td>
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<tr>
<td>STI</td>
<td>?</td>
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Role of the 12-lead ECG
Athlete’s ECG
Athletic Individual with HCM
ECG of a Nationally Ranked Black Rugby Player

Interpretation:

- **Age and gender specific ECG analysis***
- Sinus bradycardia
- ST & T wave abnormality, consider anterior ischemia

Unconfirmed report.
16-Year old Professional Soccer Player
ECG of a black athlete with HCM
Role of ECG in Differentiating Athlete’s Heart From HCM

- Sokolow-Lyon voltage criterion for LVH
- ST segment depression
+ Pathological q waves
+ Deep T wave inversion in any lead in Caucasians
+ Deep T wave inversion in lateral leads in blacks
+ Deep T wave inversion in inferior leads in blacks
+ Left bundle branch block
+ Combined left atrial enlargement and left axis
Determinants of Left Ventricular Hypertrophy

History

Demographics

Echocardiography

12-lead ECG

Cardiopulmonary Exercise Testing

Cardiac Magnetic Resonance Imaging

Detraining

Evaluation of first degree relatives/Genetic testing
Role of Cardio-Pulmonary Testing

\[ \text{VO}_2 = \text{CO} \times \frac{\text{SV} \times \text{HR}}{(A-V)\text{O}_2} \]

- **VO\textsubscript{2}**: Cardiac output
- **CO**: Systemic arteriovenous oxygen difference
- **SV**: Systemic arteriovenous oxygen difference
- **HR**: Heart rate
Failure to augment SV

Low peak oxygen consumption
A Peak Oxygen Consumption

> 50 ml/kg/min

or

>120% of that predicted for age, gender and size

is suggestive of physiological LVH rather than HCM
ECG Pattern in Apical HCM
Apical HCM
**Indications:**
- Inconclusive echo images (poor windows)
- Unexplained ECG (deep T waves)

**Advantages over echo:**
- Visualises the antero-lateral wall
- Diagnostic in apical HCM
- Reveals myocardial fibrosis
Role of Detraining

Detrain for 4-6 weeks

- Regression of LVH
  - Physiological

- No regression of LVH
  - Pathological
17 year old asymptomatic swimmer with abnormal ECG and LVH of 14 mm:

ECG Before and After Detraining for 8 Weeks
<table>
<thead>
<tr>
<th>HCM</th>
<th>Athlete’s Heart</th>
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<tbody>
<tr>
<td>+ Bizarre patterns of LVH</td>
<td>_</td>
</tr>
<tr>
<td>_ LV cavity &gt; 54 mm</td>
<td>+</td>
</tr>
<tr>
<td>+ LA &gt; 50 mm</td>
<td>_</td>
</tr>
<tr>
<td>+ LV outflow obstruction</td>
<td>_</td>
</tr>
<tr>
<td>+ Impaired diastolic function</td>
<td>_</td>
</tr>
<tr>
<td>- Isolated Sokolow-Lyon LVH</td>
<td>+</td>
</tr>
<tr>
<td>+ ST depression/Deep T wave inversion</td>
<td>_</td>
</tr>
<tr>
<td>+ Female gender</td>
<td>_</td>
</tr>
<tr>
<td>_ Absence of HCM in first degree rels</td>
<td>+</td>
</tr>
<tr>
<td>_ Peak VO2 &gt; 50 ml/kg/min</td>
<td>+</td>
</tr>
<tr>
<td>+ Myocardial fibrosis</td>
<td>_</td>
</tr>
<tr>
<td>_ Regression with detraining</td>
<td>_</td>
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CASE PRESENTATION

• 44 year old male

• Referred with an abnormal ECG requested for bradycardia

• Asymptomatic with no past medical history, drug history or family history

• Ultra-marathon runner for > 15 years – 3 races per year, total of > 20 races overall

• All races greater than 50km distance including mountain and ice terrain and some 24 hour events

• Physical examination normal with BP = 95/60 mmHg
12-LEAD ECG

- ST depression
- T wave inversion
Maximal LVWT = 14mm, LVEDD = 44mm
TRANS-THORACIC ECHOCARDIOGRAPHY

LA size = 37mm, E/A ratio = >1, and $E^I = 16$
SUBSEQUENT INVESTIGATIONS

• Exercise stress test
  – 21 minutes of Bruce protocol > 19.1 METS
  – Estimated peak oxygen consumption > 67 ml/kg/min
  – Max HR 161 bpm (91% predicted)
  – Normal BP response (systolic BP rising from 98mmHg to 168mm Hg)
  – No arrhythmias or ST segment shift

• 24 hour Holter monitor
  – No evidence of non-sustained ventricular tachycardia or other arrhythmias
PHYSIOLOGY vs PATHOLOGY

PHYSIOLOGY
- Ultra-endurance runner
- Normal diastolic function
- High peak VO$_2$

PATHOLOGY
- ECG
- Max LVWT – 14mm
- LVEDD – 44mm
FAMILY SCREENING

• 1st degree relatives underwent screening with ECG and echocardiography

• ECG and echocardiography were suggestive of HCM in the mother and sister

• Genetic testing was performed

• All 3 positive for MYBPC3 gene encoding myosin binding protein C
• Athletic training is associated with electrical and structural changes that may mimic morphologically mild HCM

• The differentiation between HCM and ‘Athlete’s Heart’ is possible using a methodical approach but can be challenging particularly in some athletes.