EVALUATION OF THE ATHLETE

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NO DISCLOSURES
• Why evaluate athletes?
• What’s the problem?
• What evaluation should be done?
• How do you interpret the echo?
• What general approach is reasonable?
TENET #1

(MODERATE)
EXERCISE IS GOOD FOR EVERYONE
BENEFITS OF EXERCISE

• Blood sugar control
• Cancer prevention
• Improved cognition
• Decreased obesity
  • Increased smoking cessation
• Reduction in stress, anxiety and depression
  • Health care costs either neutral or decreased
• Decreased symptomatic gall stones
**FITNESS**

- Cardiopulmonary fitness is inversely associated with risk of death from all causes

- Increased muscular strength is inversely associated with death in men

  [Ruiz BMJ 2008]

- In patients with cardiac disease, exercise limitation is a predictor of mortality
ATHLETES
EXTREMES NOT REQUIRED FOR HEALTH BENEFIT
THE FEAR
SUDDEN CARDIAC DEATH DURING EXERCISE IS BAD AND AVOIDABLE
Sudden Cardiac Arrest (SCA)

- The abrupt and sudden loss of heart function, usually due to abnormal heart rhythms.

- Although overall rate of SCA is low, number at risk is high.
  - Estimates vary widely (0.5-4/100,000)
  - CDC: ~2000 SCD/year among those <25 y/o
  - Difficult case capture, no mandatory registry.

- Survival from SCA remains low (15-20%).
  - On site-AEDs improve survival.

EXERCISE AND SCA

• Net effect of exercise is believed to be beneficial & should be encouraged in nearly all individuals.

• SCA usually requires susceptible substrate and a trigger.

<table>
<thead>
<tr>
<th>Susceptible substrate:</th>
<th>Exercise as trigger:</th>
</tr>
</thead>
<tbody>
<tr>
<td>Subclinical CAD in older individuals</td>
<td>Adrenergic stimulation</td>
</tr>
<tr>
<td>Congenital / genetic heart disease or channelopathies in young</td>
<td>Myocardial ischemia</td>
</tr>
<tr>
<td></td>
<td>Dehydration</td>
</tr>
<tr>
<td></td>
<td>Electrolyte imbalances</td>
</tr>
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</table>
SUDDEN DEATH IN ATHLETES

Corrado J Am Coll Card 2003
**Risk of SCA in Young Athletes**

- Prospective 21-year study in Italy, age 12-35 years.
  - 29m total and 2.4m athlete-years of observation.

- **RR of SCA = 2.8** (95% CI: 1.9-3.7)

- **% of SCA occurring in setting of exercise:**
  - **Athletes:** 89%
  - **Non-athletes:** 9%

Soccer, basketball, and swimming were the most common sports activities leading to SCA.

Corrado D et al, JACC 2003
RISK OF EXERCISE
PHYSICIANS HEALTH STUDY

- 1 sudden death in 1.5 million exercise episodes
- Habitual exercisers less likely to have sudden death
- RR of SCD 16.9

No self reported heart disease

Risk of Ex SCA by Frequency of Exercise

Albert NEJM 2000
CAUSES OF SCA IN ATHLETES

**Structural Abnormalities**
- Hypertrophic Cardiomyopathy
- Nonischemic cardiomyopathy
- Hypertensive Cardiomyopathy
- Valvular Disease
- LV noncompaction
- Coronary artery anomaly/CAD
- Sarcoidosis
- Marfan Syndrome

**Inherited Arrhythmia Syndromes**
- Arrhythmogenic Right Ventricular Cardiomyopathy
- Long QT Syndrome
- Short QT syndrome
- Brugada Syndrome
- Catecholaminergic Polymorphic VT
- Early Repolarization Syndrome

**Other**
- Myocarditis
- Wolff-Parkinson-White Syndrome
- Commotio Cordis
- Performance Enhancing Drugs
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 Sudden Death by Age and Gender in Athletes and Non-Athletes

<table>
<thead>
<tr>
<th>Cause</th>
<th>Total (N = 300)</th>
<th>Athletes (n = 55)</th>
<th>Non-Athletes (n = 245)</th>
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<tbody>
<tr>
<td></td>
<td>n</td>
<td>Age (yrs)</td>
<td>Males (n = 50)</td>
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<tr>
<td>Cardiovascular</td>
<td>259</td>
<td>23.8 ± 8</td>
<td>46</td>
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<tr>
<td>Atherosclerotic CAD</td>
<td>58</td>
<td>29.1 ± 5</td>
<td>10</td>
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<tr>
<td>Arrhythmogenic RV cardiomyopathy</td>
<td>37</td>
<td>25.2 ± 7</td>
<td>12</td>
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<tr>
<td>Mitral valve prolapse</td>
<td>32</td>
<td>22.3 ± 7</td>
<td>5</td>
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<tr>
<td>Myocarditis</td>
<td>27</td>
<td>22.7 ± 6</td>
<td>4</td>
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<tr>
<td>Disease of the conduction system</td>
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<td>21.5 ± 9</td>
<td>3</td>
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<tr>
<td>Hypertrophic cardiomyopathy</td>
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<td>22.3 ± 7</td>
<td>1</td>
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<tr>
<td>Aortic rupture</td>
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<td>21.2 ± 8</td>
<td>1</td>
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<tr>
<td>Dilated cardiomyopathy</td>
<td>11</td>
<td>22.1 ± 7</td>
<td>1</td>
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<tr>
<td>Anomalous origin of CAD</td>
<td>8</td>
<td>20.2 ± 6</td>
<td>6</td>
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<tr>
<td>Non-atherosclerotic CAD</td>
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<td>21.4 ± 8</td>
<td>0</td>
</tr>
<tr>
<td>Mitral valve prolapse</td>
<td>6</td>
<td>21.7 ± 9</td>
<td>2</td>
</tr>
<tr>
<td>Aortic valve stenosis</td>
<td>4</td>
<td>20.7 ± 3</td>
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<tr>
<td>Postoperative CHD</td>
<td>4</td>
<td>13.2 ± 5</td>
<td>0</td>
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<tr>
<td>Pulmonary thromboembolism</td>
<td>4</td>
<td>23.4 ± 2</td>
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<td>Long QT syndrome</td>
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<td>20</td>
<td>0</td>
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<tr>
<td>Non-cardiovascular</td>
<td>23</td>
<td>24.1 ± 8</td>
<td>3</td>
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<tr>
<td>Asthma</td>
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<td>23.2 ± 7</td>
<td>0</td>
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<tr>
<td>Cerebral berry aneurysm</td>
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<td>27.8 ± 8</td>
<td>1</td>
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<tr>
<td>Cerebral embolism</td>
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<td>22.2 ± 6</td>
<td>2</td>
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<tr>
<td>Other</td>
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<td>24.5 ± 6</td>
<td>0</td>
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<tr>
<td>Unexplained</td>
<td>18</td>
<td>23.2 ± 8</td>
<td>1</td>
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</tbody>
</table>
EXERCISE CHANGES THE HEART
(and that confuses things)
THE ATHLETE’S HEART

- Body size considerations

- Prolonged, recurrent exercise can cause the heart to remodel
  - Structural
  - Electrical
  - Autonomic

- Morganroth Hypothesis:
  - Endurance exercise $\rightarrow$ volume overload $\rightarrow$ 4 chamber dilation
  - Strength conditioning $\rightarrow$ pressure overload $\rightarrow$ concentric LVH
CARDIAC ADAPTATION TO EXERCISE

- 12 sedentary subjects
- Age 29 ±6 years
- Year-long marathon training.
  - Progressive intensity
  - Predominantly aerobic
- Serial CPET & CMR with PA cath pre & post.

Peak VO2: 40.3 (±1.6) → 48.7 (±2.5) ml/kg/min over 1 year.

Arbab-Zadeh, A et al. Circulation 2014; 130:2152-2161
RV volumetric remodeling & LV hypertrophy at low levels of exercise.
LV volumetric remodeling occurs later and to a lesser degree.

Arbab-Zadeh, A et al. Circulation 2014; 130:2152-2161
WALL THICKNESS IN ATHLETES

[Graph showing the distribution of left ventricular wall thickness in white and black athletes. The graph indicates a gray zone where the thickness values overlap significantly.]
"GRAY ZONE" ATHLETES

- LV cavity: 56-70mm
- Frequent or complex ventricular arrhythmias
- Dilated cardiomyopathy
- Myocarditis
- Athlete's Heart
- Gray-area
- Cardio-myopathy
- HCM; ARVC
- Distinctly abnormal ECG (T-wave inversion)
- LV wall thickness: 13-15mm
- HCM
• Work up can be extensive.

• No further testing if:
  ▪ Normal ECG
  ▪ Concentric LVH
  ▪ Normal diastolic function

• Red flags:
  ▪ Bizarre ECG
  ▪ Syncope
  ▪ Family history

Maron BS, Br J Sports Med 2009
ABNORMAL ECG IN ATHLETES
LONGITUDINAL CASE-CONTROL STUDY

• 12,550 Italian athletes
• 81 identified with diffuse, deep TWI (>2mm). (78% M, 14 Olympians).
• vs. 229 athletes with normal ECGs

• After 12 +/- 5 years:
  ▪ 6% developed cardiomyopathy:
    ▪ 1 SCD from ARVC, 3 HCM, 1 Dilated CM.
    ▪ 0% of controls developed cardiomyopathy

• May merit continued clinical surveillance, but how often and how?

Cardiac MRI Comparison
Midventricular Short Axis Views

Hypertrophy of interventricular septum over 2 years
CONTEXT MATTERS
• What sport?
• Recreational or competitive?
• How often do they train, compete?
• What environmental factors?
• What pushes them?

CONTEXT MATTERS
TAKE HOME

• SCD is not common in athletes, but is ostensibly “preventable” with evaluation and restriction *when appropriate*
• Context is important
• Evaluation is multifaceted
• Longitudinal evaluation is needed to exclude disease progression
CASES

- A 20 yo Division 1 swimmer is referred for evaluation of a dilated aorta
- A 27 yo NHL player self refers for a family history of HCM
18 YO REFERRED FOR HISTORY OF DILATED AORTA

Rower – 6’5” 230 lbs

Baseball player – 5’9” 160 lbs
24 YO NFL PLAYER

• League/team standard evaluation
• Family history SCD in cousin, grandfather
• Half sister with “heart problem”
• ECG
12 YO SOCCER PLAYER

- Asymptomatic
- Screening through soccer club
- Normal ECG
- Murmur ► echocardiogram
• Data and recommendations controversial
  ▪ *Except: If ischemia or ischemic symptoms – fix*

• Evaluation:
  ▪ CT for anatomy
  ▪ Stress nuclear scan:
    ▪ 17:30 on standard Bruce
    ▪ No symptoms
    ▪ No ischemia
18 YO DIVISION 1 BASKETBALL PLAYER

- Athletic department screening
- ECG:
LV End Diastolic Diameter: 4.9 cm
Interventricular Septum: 1.5 cm
Posterior Wall: 1.3 cm
Ejection Fraction: 77%
Normal diastolic function
ECHOCARDIOGRAM

LV End Diastolic Diameter: 4.9 cm
Interventricular Septum: 1.5 cm
Posterior Wall: 1.3 cm
Ejection Fraction: 77%
Normal diastolic function
Cardiac MRI: 9/2008

Ventricular Wall Measurements
- Base: 0.9-1.2 cm
- Mid Cavity: 0.9-1.2 cm
- Apex: 0.3-0.9 cm
- Max WT: Mid-Septum 1.3 cm

4 Chamber
Inferior Septum

4 Chamber
Inferior Septum

Midventricular Short Axis

Long Axis
Anterior Septum

4 Chamber
Inferior Septum

4 Chamber
Inferior Septum

LV
IVS
RV
LA
RA
• CPET:
  ▪ Normal HR/BP response:
    ▪ 80 → 209 bpm
    ▪ 125/65 → 190/68 mmHg
  ▪ VO2 max:
    ▪ 36.2 ml/kg/min (69% predicted)
    ▪ 49.3 ml/kg/min (95% predicted) for ideal BW
  ▪ O2 pulse
    ▪ 20.8 ml/beat (91% predicted)

• Followed longitudinally