Cardiovascular Issues in Athletes

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Tragic Day at Olympic Trials

November 4, 2007

An ambulance carrying the runner Ryan Shay, who collapsed during the United States Olympic trials marathon on Saturday, made its way through Central Park to Lenox Hill Hospital, where Shay was pronounced dead at 8:46 a.m.

Tragic Day at Olympic Trials

November 4, 2007

Shay, left, and Ryan Hall, second from left, at the start of Saturday’s race.
Hall, who won the race, and Shay were close friends. Hall’s wife, Sara, was a bridesmaid when Ryan and Alicia Shay were married in July.
Tragic Day at Olympic Trials

Shay ran for Notre Dame in college.
"We all are devastated over Ryan's death," said Craig Masback, chief executive of USA Track & Field. "He was a tremendous champion who was here today to pursue his dreams."

Photo: Don Ryan/Associated Press

Tragic Day at Olympic Trials

Hall, center, at a news conference following the race on Saturday. During the race, Shay's ambulance whisked past Hall, who was at the front of the pack of runners. "I had no idea," Hall said. "When I heard the news, I just couldn't believe it."

Photo: Frank Franklin II/Associated Press

Tragic Day at Olympic Trials

Mary Wittenberg, president of the New York Road Runners, on Saturday. Shay's death was confirmed by Wittenberg. "It puts a knife through everybody's heart," she said.
Enlarged heart chief cause of athletes' sudden death

Ryan Shay, in red gloves, collapsed and died while running in the U.S. Olympic Team Trials Men's Marathon.

Definition of an Athlete

• “One who participates in an organized team or individual sport requiring systematic training and regular competition against others while placing a high premium on athletic excellence.”

Demographics of Athletes

- 10 to 15 million athletes participate in organized sports in the U.S.
- 4,000,000 high school (grades 9-12) athletes
- 500,000 college athletes
- 5000 professional athletes

Sudden Cardiac Death in Athletes

Incidence of Sudden Cardiac Death in Athletes

- High school athletes: 1 in 200,000
- Older athletes: 1 in 15,000 healthy male joggers
Incidence of Sudden Cardiac Death in Athletes

<table>
<thead>
<tr>
<th>Population group</th>
<th>Age</th>
<th>Incidence</th>
</tr>
</thead>
<tbody>
<tr>
<td>Organized high school/college athletes</td>
<td>High school/college age</td>
<td>7.5:1,000,000/year (male)</td>
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<tr>
<td></td>
<td></td>
<td>1.3:1,000,000/year (female)</td>
</tr>
<tr>
<td>U.S. Air Force recruits</td>
<td>17 to 28 years</td>
<td>1:735,000/year</td>
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<tr>
<td>Rhode Island joggers</td>
<td>&lt;30 years</td>
<td>1:280,000/year</td>
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<tr>
<td>Rhode Island joggers</td>
<td>30 to 65 years</td>
<td>1:5,620/year</td>
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<tr>
<td>Marathon runners</td>
<td>Mean age 37</td>
<td>1:59,000 race finishers</td>
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</tbody>
</table>


Causes of Sudden Death in 387 Young Athletes

<table>
<thead>
<tr>
<th>Cause</th>
<th>No. of Athletes</th>
<th>Percent</th>
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<tbody>
<tr>
<td>Hypertrophic cardiomyopathy</td>
<td>102</td>
<td>26.4</td>
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<tr>
<td>Commotio cordis</td>
<td>37</td>
<td>9.9</td>
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<tr>
<td>Coronary artery anomalies</td>
<td>53</td>
<td>13.7</td>
</tr>
<tr>
<td>LVH - indeterminate etiology</td>
<td>29</td>
<td>7.5</td>
</tr>
<tr>
<td>Myocarditis</td>
<td>20</td>
<td>6.2</td>
</tr>
<tr>
<td>Ruptured aortic aneurysm (Marfan's syndrome)</td>
<td>11</td>
<td>2.8</td>
</tr>
<tr>
<td>Arrhythmogenic right ventricular cardiomyopathy</td>
<td>11</td>
<td>2.8</td>
</tr>
<tr>
<td>Tunneled (bridged) coronary arteries</td>
<td>11</td>
<td>2.8</td>
</tr>
<tr>
<td>Aortic valve stenosis</td>
<td>10</td>
<td>2.6</td>
</tr>
<tr>
<td>Atherosclerotic CAD</td>
<td>10</td>
<td>2.6</td>
</tr>
<tr>
<td>Dilated cardiomyopathy</td>
<td>9</td>
<td>2.3</td>
</tr>
<tr>
<td>Myxomatous annulovalvular degeneration</td>
<td>9</td>
<td>2.3</td>
</tr>
<tr>
<td>Asthma (or other pulmonary)</td>
<td>8</td>
<td>2.1</td>
</tr>
<tr>
<td>Heat stroke</td>
<td>6</td>
<td>1.6</td>
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<tr>
<td>Drug abuse</td>
<td>4</td>
<td>1.0</td>
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<tr>
<td>Other cardiovascular cause</td>
<td>4</td>
<td>1.0</td>
</tr>
<tr>
<td>Long QT syndrome</td>
<td>3</td>
<td>0.8</td>
</tr>
<tr>
<td>Cardiac sarcoidosis</td>
<td>3</td>
<td>0.8</td>
</tr>
<tr>
<td>Trauma involving structural cardiac injury</td>
<td>3</td>
<td>0.8</td>
</tr>
<tr>
<td>Ruptured aortic aneurysm</td>
<td>3</td>
<td>0.8</td>
</tr>
</tbody>
</table>

Maron, BJ JAMA 1996; 276:199-204.

Causes of SCD in Young Athletes

Causes of SCD in >35 yr Old Athletes

- Coronary Heart Disease (~80%)
- Unexplained
- Acquired Valve Disease
- MVP
- HCM

> 36 YEARS OLD


Mechanism of Sudden Death

- Electrolyte Abnormalities
- Ischemia
- Antiarrhythmic Drugs
- Sympathetic Efferents
- Circulating Catecholamines

ANATOMIC / ELECTRICAL SUBSTRATE

Ventricular Arrhythmias

SUDDEN CARDIAC DEATH


Cause of SCD of Elite Athletes

- Jim Fixx: ASCVD
- Len Bias: Cocaine
- Reggie Lewis: Focal Myocarditis
- Flo Hyman: Marfan’s - Aortic rupture
- Hank Gathers: Hypertrophic Cardiomyopathy
- Pete Maravich: Hypoplastic Coronary Arteries
How can we prevent such tragedies?

Screening

• Disease should be:
  – prevalent
  – have significant M & M
  – be treatable
• Treatment results in a better outcome
• Good screening test must be available
• Cost-benefit ratio should be favorable

Screening

• Purpose: to provide medical clearance for participation in competitive sports.

• Assumption: intense training will increase the risk of sudden cardiac death or disease progression in trained athletes.

• Expected Outcome: to reduce the risks associated with organized sports.
Cardiovascular Issues in Athletes

Cardiac Evaluation of the Athlete: Preparticipation Screening

- Customary strategy for screening U.S. high school and college athletes is history and physical exam.
- Most trained athletes with occult cardiovascular disease are asymptomatic.
- Only 3% of trained athletes who developed SCD were suspected by routine H&P.

Bethesda Conference JACC 2005;1317-1375.

Preparticipation Screening

- AHA – pushing for a national standard for PPE to include cardiovascular screening.
  - Mandatory screening for all HS (9-12) and College athletes.
  - Repeated every 2 years with an interim history during intervening years
  - Include a history and physical designed to identify or raise suspicion of CV conditions known to increase risk of SCD
  - Conducted by healthcare worker (preferably physician) who has the appropriate training, skills and background to reliably obtain a CV history, perform a thorough CV physical and recognize heart disease.

AHA Consensus Panel Recommendations for Preparticipation Athletic Screening

- Family History
  1. Premature sudden cardiac death
  2. Heart disease in surviving relatives less than 50 years old

- Personal History
  3. Heart murmur
  4. Systemic hypertension
  5. Fatigue
  6. Syncope/near syncope
  7. Excessively/unexplained exertional dyspnea
  8. Exertional chest pain

- Physical Examination
  9. Heart murmur (supine/standing)
  10. Femoral arterial pulses (to exclude coarctation of aorta)
  11. Stigmata of Marfan syndrome
  12. Brachial blood pressure measurement (sitting)

History and Physical Findings that Require Further Investigation

• Syncope, near-syncope, frequent dizziness, or palpitations (especially if related to exertion)
• Heart murmur
• Family history of sudden cardiac death
• Exertional chest discomfort or profound dyspnea

Diagnostic Testing Strategies

• Electrocardiography
• Echocardiography
• Ambulatory ECG Monitoring
• CT Angiography
• Cardiac MRI

Electrocardiography

• Abnormal in 75% to 95% of patients with HCM
• Will identify long QT, Brugada
• Raises suspicion of myocarditis (PVCs, NAST-T)
• Raises suspicion of ARVC (T₃-V₃-V₅₃)
Preparticipation Exam – Italy

• Early 1970’s Italy passed law mandating that all athletes engaging in competitive sports will undergo a clinical evaluation and receive eligibility.
• History, Physical and ECG

Numbers and Money

• 10-12 million athletes (HS/College Athletes in US)
• 6 million athletes (all levels) Italy
• Sudden Cardiac Death Rates:
  – 1:200-300,000 athletes/year US
    • 1:135,000 males
    • 1:769,000 females
  – 2.1:100,000 athletes/year Italy
• Italy – twice as high with half the numbers

Echocardiography

• Principal imaging mode to identify HCM (LV end-diastolic wall thickness ≥ 15mm)
• Confirm MVP, AS
• Identify enlarged aortic root in Marfan’s
• Identify LV enlargement or dysfunction as in dilated cardiomyopathy or myocarditis

**Numbers and Money**

HCM 1:500 incidence  
$500/ECHO  
$250,000 to detect 1  
Case of HCM/500 athletes  
$100 million dollars to stop 1 SCD  

- 1 death  
- 1000 at risk of sudden cardiac death  
- 200,000 Athletes

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**Ambulatory ECG Monitoring**

- Useful to define the rhythm disturbance resulting in palpitations (PVCs vs PACs, etc.)
- Useful to determine if there is an electrical cause of syncope/dizziness

*36th Bethesda Conference JACC 2005:1317-1375*

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**CT Angiography**

- Useful for congenital coronary artery anomalies if clinically suspected
- Coronary angiography may still be necessary

*36th Bethesda Conference JACC 2005:1317-1375*
Cardiovascular Issues in Athletes

Cardiac MRI

- Most useful non-invasive test to identify the structural abnormalities seen with ARVC (RV enlargement, wall motion abnormalities, adipose tissue deposition, aneurysm formation)
- Not entirely sensitive nor specific for ARVC

ARVC = Arrhythmogenic Right Ventricular Cardiomyopathy


Pathology vs. Physiology

- Elite athletic training by itself causes structural changes in the normal heart ("athlete’s heart")
- Physiologic increases in cardiac mass and/or cavity dimensions are commonly seen in rowers, cross-country skiers, cyclists, and swimmers
- Isometric training as used by weight lifters and wrestlers results in a disproportionate increase in LV wall thickness in relation to the cavity size
- The above result in ECG abnormalities (R- or S-wave voltages, Q waves and repolarization abnormalities) in approximately 40% of elite athletes


The Athlete’s Heart

- Recognized in the 19th century that the hearts of cross country skiers were bigger
- CO=HR x SV
- The trained athlete improves CO by increasing SV
- Physiologic hypertrophy – an increase in LV mass due to repetitive exercise.
- Heart dimensions rarely exceed the upper limits of normal if adjusted for body size
- Detraining – physiologic v pathologic hypertrophy
Cardiovascular Issues in Athletes

**The Athlete’s ECG**

- Sinus bradycardia
- Sinus arrhythmia
- 1st degree AV block
- 2nd degree, Type 1 AV block
- Incomplete RBBB
- RVH

**Repolarization Changes**

- ST wave elevation
- Upward sloped ST segment
- T wave inversion

- LVH
- Upper limit of normal QT interval

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**The Athlete’s Heart & ECG**

38 year old male distance runner with sinus bradycardia (42 bpm) with periods of junctional rhythm (arrows)

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**The Athlete’s Heart & ECG**

41 year old male distance runner with J-point and ST-segment elevation (arrows) depicting early repolarization
The Athlete's Heart & ECG

LVH in an athlete

Pathological LVH

Note: "strain" pattern in lateral precordial leads

The Challenge


Cardiovascular Issues in Athletes
Specific Pathologic Conditions

- Hypertrophic Cardiomyopathy
- Commotio Cordis
- Congenital Coronary Artery Anomalies
- Marfan Syndrome
- Arrhythmogenic Right Ventricular Dysplasia
- Long QT Syndrome

Hypertrophic Cardiomyopathy

- Common form of genetic heart disease (1:500 in the general population)
- Most common cause of SCD in young people
- Asymmetric hypertrophy with a non-dilated LV
- LV end-diastolic wall thickness > 15 mm in adults
- Be ware, children that harbor the HCM gene may not manifest LVH until adulthood making screening echo less useful
- Should be excluded from most competitive sports even if asymptomatic with no outflow obstruction

HCM ECG
Hypertrophic Cardiomyopathy

- Murmur Characteristics:
  - Standing examination
  - Decrease venous return increases intensity of murmur
  - What do you hear?
    - Squat to stand the murmur will get louder
    - Stand to squat the murmur will get softer
    - Valsalva murmur will get louder

Commotio Cordis

- Modest, blunt trauma to the chest in the absence of structural heart disease or evidence of myocardial contusion results in cardiac arrest
- Most common in young children with compliant chest walls
- Chest impact directly over the heart
- Timing of the blow occurs precisely during repolarization, just prior to the T-wave peak

Maron, BJ NEJM 2003;349:1064-75

Congenital Coronary Artery Anomalies

- Coronary arteries arise from the wrong aortic sinus
- Not usually associated with atherosclerosis
- Diagnosis requires a high index of suspicion
- Typically normal exercise ECG because the ischemia is episodic
- History of exertional chest pain or syncope in a young athlete

Maron, BJ NEJM 2003;349:1064-75
### Anomalous Coronary Artery

- **Anomalous origin of left coronary artery from right sinus of Valsalva**

### Marfan Syndrome

- Autosomal dominant
- Disorder of the connective tissue
- Aortic root dilatation is common
- May result in aortic dissection and sudden death

### Clinical Manifestations of Marfan Syndrome

- Aortic insufficiency murmur
- Arachnodactyly
- Arm span greater than body height
- Kyphosis
- Lenticular dislocation
- Mitral valve prolapse
- Myopia
- Pectus excavatum
- Thumb sign (entire nail of the thumb projects beyond the ulnar border of the hand when clenched)
- Wrist sign (thumb overlaps last joint of 5th finger when hand is wrapped around opposite wrist)
**Arrhythmogenic Right Ventricular Dysplasia (ARVD)**

- Characterized by regional or full thickness replacement of right free ventricular wall myocardium by fat and fibrous tissue.
- Leads to electrical instability
- Not identified until late 1970s
- Incidence: 6/10,000 up to 44/10,000
- 3%-17% of SCDs

**Figure 1.** Postmortem heart in a patient with arrhythmogenic right ventricular dysplasia. Note how the right ventricular myocardium and endomyocardium have been replaced by fat.

**Triangle of dysplasia.**

**Epsilon wave** – a late positive deflection in the terminal QRS complex

Long QT Syndrome

- Infrequent 1/5000
- Defined as a corrected QT interval (QTc) of >450 msec in men and >460 in women.
- Usually present with syncope after emotional or physical stress
- “R on T Phenomenon” that can initiate a rapid polymorphic ventricular tachycardia (torsades de pointes)
- Congenital v Acquired

Long QT Syndrome

Torsades de Pointes associated with long QT syndrome (a deadly type of Ventricular Tachycardia)

Special Considerations

- Beta blockers commonly used to treat a variety of cardiac disorders cannot be regarded as a means of affording safety against arrhythmias to retain eligibility for vigorous competitive sports

- Availability of AED’s at sporting events should not be considered as justification to permit an otherwise medically ineligible athlete to participate

- ICD’s should disqualify athletes from most competitive sports

- Pacemaker-dependent athletes should not participate in most competitive sports

AHA Recommendations

• Careful, detailed history and physical exam every 2 years by qualified care providers.

• Intervening years, obtain an interim history.

• Selectively perform ETT in men (>40 yo) and women (>50 yo) having a high risk for CAD who wish to begin regular physical training.

• Develop a national standard for preparticipation medical evaluation.

Summary

• Sudden cardiac death (SCD) in the athlete is rare.

• Identifying individuals at risk for SCD (i.e., primary prevention) is very difficult.

• Guidelines for individuals with known cardiovascular disease (i.e., secondary prevention) have been published -- 36th Bethesda Conference.

Summary

• A detailed history and physical exam should be performed on all athletes.

• Noninvasive testing should be used selectively.

• Must educate the public about the limits of screening and preventing SCD in the athlete. It is not possible to achieve zero-risk in competitive sports.

• Community efforts should focus on developing and implementing systems of care that allow for prompt response and treatment.

Madden Bill

• NJ State Senator Fred H. Madden, D-Camden and Gloucester

• Requires the Department of Education to work with the Commissioner of Health and Senior Services, the American Heart Association, and the American Academy of Pediatrics to create a pamphlet about sudden cardiac death in student athletes. The pamphlet will contain a clear explanation of sudden cardiac death, statistics of cases among student athletes, warning signs, and available screening options. The literature will be distributed to local school districts to send to all parents and guardians whose children participate in interscholastic sports.

• The bill was approved by the legislature 6/18/07 and signed into law by Governor Jon Corzine 8/29/07.
Janet's Law

- Requires public and private schools to have an AED in place and to establish emergency action plans for sudden cardiac arrest. At least 5 on-site personnel must be trained for emergency response.
- The bill was signed into law September 21, 2012 by Governor Chris Christie.

New Madden Legislation

- 10/26/2012 Madden Legislation Establishing Statewide Database On Children's Sudden Cardiac Events Clears Senate
- 9/24/2012 Madden Legislation Modernizing Pre-Participation Physical For Student Athletes Clears Committee
- 9/20/2012 Madden Legislation Requiring Insurance Coverage For Annual Athletic Fitness Physicals Clears Committee

New Jersey Task Force on Preventing Sudden Cardiac Death in Student Athletes

- NJ Law has been passed which mandates special training for all physicians, physician assistants, and advanced practice nurses who will be certifying athletes for participation in sports.
- Will require a more detailed history and physical exam be documented.
- Certifying module is being prepared now.
- Once this takes effect, all pre-participation forms will have to be signed by a practitioner who has achieved this special certification.
Thank You

Any Questions?