Sudden Cardiac Death in the Athlete

Hutton P. Brantley, DO, FACC

CARDIOLOGY, P.C.
• No disclosures relevant to this talk
Objectives

- To discuss the magnitude of the problem of and causes of sudden cardiac death
- To provide information in differentiating physiologic adaptation from cardiac pathology.
- To discuss preventative strategies to reduce the risk sudden cardiac death during sport.
Sudden Cardiac Death

- Sudden cardiac arrest (SCA) and sudden cardiac death (SCD) refer to the sudden cessation of cardiac activity with hemodynamic collapse, typically due to sustained V TACH/FIB.
- The event is referred to as SCA (or aborted SCD) if an intervention (e.g., defibrillation) or spontaneous reversion restores circulation, and the event is called SCD if the patient dies.
- However, the use of SCD to describe both fatal and nonfatal cardiac arrest persists by convention.
The Heterogeneity of Athletes

• Younger athletes

• Masters athletes

• Recreational athletes
Definitions

• EXERCISE: Any form of physical activity, done on a regular basis, with the purpose of achieving a specific goal
  • Low level to vigorous
  • Recreational (including “play”) to competitive
• ATHLETE: Anyone who is exercising
• YOUNG ATHLETE: Less than 35 years old
• ADULT ATHLETE: Greater than 35 years old
SCA in Athletes

“The unexpected death of an athlete during exercise is tragic irony. ... much remains unknown regarding optimal screening strategies, pathophysiologic mechanisms, and prevention”

Dr Mark Link
Tufts University
The Faces of SCA
Agenda

– The athletes heart
– Epidemiology
– Risk factors
– Causes of SCD
  • Young and adult
– Prevention/Screening?
The Physiological Adaptations in the Trained Athlete

• Athletic training for competitive endurance (aerobic) or isometric (static or power) sports results in characteristic changes in cardiac structure and function.

• This physiological form of left ventricular (LVH) hypertrophy is known as the “athlete’s heart” and must be distinguished from pathological conditions that may predispose to SCD.

• Depending on the nature of the exercise training benign increases in LV mass, wall thickness, and cavity size as well as left atrial volume may be observed in healthy athletes.
The Physiological Adaptations in the Trained Athlete

Endurance Training
- Increase in LV cavity size
- Minimal increase in LV wall thickness

Isometric Training
- Increase in LV wall thickness out of proportion to increase in cavity size
The Athlete’s Heart

Gray area of overlap between the “athlete’s heart” and cardiomyopathies.

N Engl J Med 2003;349:11
Classification of sports based on peak static and dynamic components achieved during competition.
Extrinsic Risk Factors for SCD

• The risk of SCD in competitive sports increases with “burst” exertion (rapid acceleration and deceleration; common in basketball, tennis, and soccer).

• Extreme environmental conditions (temperature, humidity, and altitude) that affect blood volume and electrolyte balance also contribute to the risk.

• Progressive and systematic training to achieve higher levels of conditioning and performance may further increase the risk by resulting in a total cardiovascular demand that often exceeds that of competition.
Relative Risk of SCD

- Athletes: 2.5
- Non-athletes: 1.0

SD/100,000 person yrs

Image credit: Corrado D JACC 2003
Other Extrinsic Risk Factors for SCD

• Cocaine abuse
• Amphetamine abuse
• Performance enhancing drugs (anabolic steroids)
• Dietary and nutritional supplements (including ephedra-containing products)
How common is SCD in young competitive US athletes?

100 to 150 competitive deaths during sports in the United States annually.

On average, every 3 days in the United States a competitive athlete experiences a SCD.

Link Circulation 2012
SCD in Young Competitive Athletes
1980-2006 (1866 deaths in US)

- 1866 athlete deaths
- 38 sports
- 56% CV causes
- 3% commotio cordis
- From 2001-2006, incidence of SCD was 0.61
  / 100,000 person-yrs
  (assuming 10.9 million participants/yr)

Maron Circulation 2009
Sudden Cardiac Death in Young Athlete

- Incidence is approximately 1/50,000
- Mean age at death in athletes 23 years-old
- 40% deaths in athletes aged < 18 years old
- More common in males than females (9:1)
- 90% deaths during or immediately after exertion
Causes of Sudden Cardiac Death

• Causes of SCD in athletes vary by age and geographic location.

• Among young competitive athletes (< 35 years old) in the U.S., inherited or congenital heart conditions (such as hypertrophic cardiomyopathy) are the most common etiologies.

• Among “masters” athletes (> 35 years old), coronary artery disease (CAD) appears to be the predominant cause of SCD.
Cause of SCD in 690 confirmed events over 27 years

<table>
<thead>
<tr>
<th>CAUSE</th>
<th># of CASES</th>
<th>PERCENTAGE</th>
</tr>
</thead>
<tbody>
<tr>
<td>1) Hypertrophic cardiomyopathy (HCM)</td>
<td>251</td>
<td>36 %</td>
</tr>
<tr>
<td>2) Coronary artery anomalies</td>
<td>119</td>
<td>17 %</td>
</tr>
<tr>
<td>3) Possible HCM</td>
<td>57</td>
<td>8 %</td>
</tr>
<tr>
<td>4) Myocarditis</td>
<td>41</td>
<td>6 %</td>
</tr>
<tr>
<td>5) Arrhythmogenic RV cardiomyopathy</td>
<td>30</td>
<td>4 %</td>
</tr>
<tr>
<td>6) Ion channelopathies</td>
<td>25</td>
<td>4 %</td>
</tr>
<tr>
<td>7) Mitral valve disease</td>
<td>24</td>
<td>3 %</td>
</tr>
<tr>
<td>8) LAD myocardial bridge</td>
<td>23</td>
<td>3 %</td>
</tr>
<tr>
<td>9) Coronary artery disease</td>
<td>23</td>
<td>3 %</td>
</tr>
<tr>
<td>10) Aortic rupture</td>
<td>10</td>
<td>1 %</td>
</tr>
</tbody>
</table>

Maron Circulation 2009
Sudden death in NCAA 2004-2008 (273 athlete deaths)

Harmon Circulation 2011
SCD in 36 confirmed events in NCAA (2004-2008)

<table>
<thead>
<tr>
<th>CAUSE</th>
<th># of CASES</th>
<th>PERCENTAGE</th>
</tr>
</thead>
<tbody>
<tr>
<td>1) Autopsy Negative –sudden unexplained death</td>
<td>11</td>
<td>31%</td>
</tr>
<tr>
<td>2) Coronary artery anomalies</td>
<td>5</td>
<td>14%</td>
</tr>
<tr>
<td>3) Dilated cardiomyopathy</td>
<td>3</td>
<td>8%</td>
</tr>
<tr>
<td>4) Myocarditis</td>
<td>3</td>
<td>8%</td>
</tr>
<tr>
<td>5) Aortic dissection</td>
<td>3</td>
<td>8%</td>
</tr>
<tr>
<td>6) Possible HCM</td>
<td>3</td>
<td>8%</td>
</tr>
<tr>
<td>7) HCM</td>
<td>1</td>
<td>3%</td>
</tr>
<tr>
<td>8) Arrhythmogenic cardiomyopathy</td>
<td>1</td>
<td>3%</td>
</tr>
<tr>
<td>9) Commotio cordis</td>
<td>1</td>
<td>3%</td>
</tr>
<tr>
<td>10) Kawasaki disease</td>
<td>1</td>
<td>3%</td>
</tr>
</tbody>
</table>
Age-related risks of SCD
THE ADULT ATHLETE

• Harder to define the numbers and risk
  • Heart disease is common among adults
  • Exercise programs vary
  • No organized reporting program
• Marathoners: <1/100,000 on race day
• Tri-athletes: 1.5/100,000 on race day
• Recreational runners: 1/10,000/year or 1/396,000 hours of running
• Nordic Skiers: 1/607,000 hours
• Individuals with disease are 2 -3-X more likely to have an event during exertion.
THE ADULT ATHLETE

- Coronary Artery Disease
- Valvular Heart Disease
- Cardiomyopathy
- “Young Athlete” Disease
SCD in Senior Athletes

- CAD: 80%
- SAD: 5%
- MVP: 5%
- Valves: 5%
- HCM: 5%
Triggers for SCD in the Athlete

- Dehydration
- Adrenergic surges
- Acid/base disturbance
- Electrolyte imbalance

[Diagram showing the relationships between dehydration, adrenergic surges, electrolyte imbalance, and acid/base disturbance.]
SCD and the Young Athlete

• The “Underlying Substrate”
  – Many of these conditions predispose to lethal arrhythmia

• There can be changes in the athlete’s heart that may increase the risk
  • Hypertrophy (the “muscular heart”)
  • LV and RV dilation (the “enlarged heart”)
  • Increased demand and “adrenalin”
MECHANISM OF SUDDEN DEATH
Ventricular Tachycardia and Ventricular Fibrillation

Normal EKG

Ventricular Tachycardia

Polymorphic Ventricular Tachycardia
Ventricular Fibrillation
THE YOUNG ATHLETE and SUDDEN CARDIAC DEATH

- Rare events
- Without warning
- Devastating
- Occur in healthy individuals
- Attract attention
THE YOUNG ATHLETE

• **Structural Heart Disease**
  • Hypertrophic Cardiomyopathy
  • Anomalous Origin of the Coronary Arteries
  • Arrhythmogenic Right Ventricular Cardiomyopathy
  • Myocarditis/Cardiomyopathy
  • Valvular Disease

• **The “Channelopathies”**

• **Marfan Syndrome**
THE YOUNG ATHLETE
HANK GATHERS
1967 - 1990
1990 - Hank Gathers Tragedy

• DX: exercise related complex ventricular tachycardia
• RX: Beta Blocker- Inderal 200qd
• Return to play in three weeks
• Courtside cardiac monitor defibrillator
Hank Gathers SCA

- Medication had been decreased due to side effects
- Cause of death - HCM
- Cardiac monitor defibrillator legal issue: $32 Million lawsuit
HYPERTROPHIC CARDIOMYOPATHY
HYPERTROPHIC CARDIOMYOPATHY

- Affects 1 in 500 individuals
- Genetically determined (auto dominant)
  - Sporadic or inherited
  - At least 11 genes, 1400 mutations
- Accounts for 35 – 40% of athletic deaths
- Can be symptomatic/detectable before SCA
- Increased risk with age
- Ventricular arrhythmia is primary cause of death
Sudden Cardiac Death in HCM

- Nonsustained Ventricular Tachycardia
  - Rest
  - Exercise

- Fibrosis or Scar

- Severity of LV Hypertrophy

- Abnormal Exercise Blood Pressure Response

- Role of Isolated Myofilament Mutation?

- Family History of Sudden Death

- Unexplained Syncope

- Outflow Obstruction
Fig. 5. ECG of patient with apical hypertrophic cardiomyopathy variant with deeply inverted T waves in chest leads V₂-V₆ and limb leads II, III, and aVL.
HYPERTROPHIC CARDIOMYOPATHY

• Treatment: Beta blockers; +/- Implantable Defibrillator
• Family screening
• Genetic testing debatable

• “Disqualified” from participation in all but low effort sports (bowling, curling) regardless of symptoms, phenotype, treatment
THE 36TH BETHESDA CONFERENCE
Pete Maravich

- Starred in college at Louisiana State University
- All-time leading college scorer with an average of 44.2 pts/game
- Played for 3 NBA teams
- Forced into retirement at age 32 due to knee problems.
- Inducted into the NBA Hall of Fame.
- Sudden cardiac death (SCD) at age 40 during a recreational game of basketball.
Coronary Anomaly with Cardiomyopathy

What caused Maravich’s heart to stop

The normal human heart has two coronary arteries that feed blood to the heart muscle. Pete Maravich’s heart was missing the left coronary artery and the right one had to do the work of both. As a result, the right one, which was much larger than normal, compensated by wrapping itself around the back of the heart and opening up on the front, where the left coronary artery should have been.

Normal heart
- Right pulmonary artery
- Superior vena cava
- Right coronary artery
- Right ventricle
- Apex

Maravich’s heart
- Right pulmonary artery
- Superior vena cava
- Left coronary artery
- Right coronary artery
- Right ventricle
- Left ventricle
- Apex

The autopsy revealed Maravich’s entire heart was enlarged. A normal heart weighs 400-450 grams. His weighed 560 grams, about 30 percent larger than normal. Cardiologist Barry Maron said playing basketball might have caused Maravich’s heart to be somewhat enlarged, but not to this degree.

Source: Los Angeles Coroner’s office spokesman Robert Dambacher

By Bill Baker, USA TODAY
ANOMALOUS ORIGIN OF THE CORONARY ARTERIES
ANOMALOUS ORIGIN OF THE CORONARY ARTERIES

• Accounts for 15 – 20% of sudden death in young athletes
• Can be symptomatic (< 50%)
  • Chest discomfort
  • Shortness of breath
  • Palpitations
  • Fainting
• Treatment: Medical or Surgical
• May be “cleared” to participate if corrected
<table>
<thead>
<tr>
<th>Type of Anomaly</th>
<th>Incidence (%)</th>
<th>Anomalies (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Benign (80%)</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Separate or adjacent LCX and LAD ostia</td>
<td>0.4</td>
<td>30</td>
</tr>
<tr>
<td>Separate or adjacent LCX and LAD ostia</td>
<td>0.4</td>
<td>30</td>
</tr>
<tr>
<td>LCX from PSV</td>
<td>&lt;0.01</td>
<td>0.3</td>
</tr>
<tr>
<td>Absent LCX</td>
<td>0.0003</td>
<td>0.2</td>
</tr>
<tr>
<td><strong>Clinically Significant (20%)</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>LMCA from RSV</td>
<td>0.02</td>
<td>1</td>
</tr>
<tr>
<td>LAD from RSV</td>
<td>0.03</td>
<td>2</td>
</tr>
<tr>
<td>RCA from LSV</td>
<td>0.10</td>
<td>10</td>
</tr>
<tr>
<td>LMCA from PA</td>
<td>&lt;0.01</td>
<td>&lt;1</td>
</tr>
<tr>
<td>LAD or RCA from PA</td>
<td>&lt;0.01</td>
<td>&lt;1</td>
</tr>
</tbody>
</table>

Anomalous RCA from Left Coronary Sinus

- Most common, potentially serious anomaly
Death on the Soccer Field

- He regained consciousness and was walked to the locker room where he collapsed again.
- He was resuscitated and brought to the ICU of a nearby hospital.
- He suffered multiple prolonged cardiac arrests over the next several hours resulting in anoxic brain injury and multisystem organ failure.
- He died 3 days after his initial collapse at age 22.
- Work-up revealed arrhythmogenic right ventricular cardiomyopathy (ARVC).

Antonio Puerta (November 26, 1984 – August 28, 2007)

ARRYTHMOGENIC RIGHT VENTRICULAR CARDIOMYOPATHY
Arrhythmogenic Right Ventricular Cardiomyopathy

- Characterized by fibrofatty infiltration of the RV free wall (may affect the LV).
- Symptoms include exercise induced palpitations, presyncope, or syncope.
- SCD is due to catecholamine-sensitive ventricular arrhythmias.
- ECG findings include increased QRS duration, epsilon waves in V1-2, and T wave inversions in the right precordium.
- Imaging may demonstrate RV dilatation and aneurysms.
- Echo and cardiac MRI are the most widely used noninvasive tests for ARVC.

J Am Coll Cardiol 2001;38:1773
ARVD – Arrhythmogenic Right Ventricular Dsyplasia

• Italian Sport Federation requires school athletes to have EKG and limited stress test on an annual basis
• EKG with prolonged QRS V1-V3 110 msec and inverted T wave
• **Epsilon** wave in 50%
ARVD – Prolonged QRS, Inverted T wave V1 – V2
ARVD with fatty RV myocardium
MYOCARDITIS/CARDIOMYOPATHY

- Accounts for 5-10% of sudden cardiac arrests in young athletes
- **Causes:** “viral”, inherited/genetic, idiopathic
- **Can be symptomatic:** shortness of breath, palpitations, fatigue/weakness, fainting, chest discomfort
- **Treatment:** Medical, time, ICD, transplant
- Disqualified from most competitive sports. May return if recover. ICD = no contact sports
MARFAN SYNDROME

• Connective tissue disorder
• Genetic
  • 25% sporadic
  • Autosomal Dominant
• 1/3000 – 5000
In Marfan syndrome, the enlarged aorta can eventually lead to tears or an aneurysm.
Aortic Aneurysm and Dissection

- In young athletes, aortic aneurysm and dissection is most often associated with aortopathy.
- Closely linked with inherited connective tissue disorders (Marfan’s syndrome).
- Marfan’s is inherited in an autosomal dominant fashion with an incidence of 1 in 10,000 to 20,000.
- Characteristic morphological findings (arachnodactyly, hyperflexible joints) may be noted on examination.
- SCD occurs due to aortic aneurysm rupture or dissection.
- Diagnosed on basis of clinical criteria.
- Echo is recommended to evaluate for aortic disease (including AR).
- Cardiac MRI and CT also detect aortic pathology.
Sudden Cardiac Death in the Absence of Structural Heart Disease

• SCD in competitive athletes may also occur in the absence of structural heart disease.

• Causes of SCD in structurally “normal” hearts include inherited arrhythmia syndromes such as:
  – Long QT syndrome
  – Brugada syndrome
  – Catecholaminergic polymorphic VT
  – Wolf-Parkinson-White syndrome
  – Congenital short QT syndrome

• In addition, idiopathic VF and commotio cordis may result in SCD among competitive athletes.
INHERITED ARRHYTHMIA
and
SUDDEN CARDIAC ARREST

The “Channelopathies”
WHAT IS A CHANNEL?
THE CHANNELOPATHIES AND SCD

• Long QT Syndrome
• Brugada Syndrome
• Catecholaminergic Polymorphic Ventricular Tachycardia
• Short QT
THE CHANNELOPATHIES AND SUDDEN CARDIAC ARREST

• Inherited/genetic conditions
• Lead to Ventricular Tachycardia and Ventricular Fibrillation
• Evident (variably/intermittently) on EKG
• Cause of Sudden Cardiac Arrest in both athletes and non-athletes. Exercise does increase the risk in many of these conditions
THE LONG QT SYNDROME

Each section of an electrocardiogram (ECG or EKG) is referred to by a letter name: Q, R, S, and T.

One beat (R to R)

QRS T R

Normal

QRS T R

Long

Heart is full of blood at Q. Heart contracts at end of T.
Long QT Syndromes

• Often acquired, long QT syndrome can be inherited.
• Long QT syndromes may result in polymorphic VT (torsade de pointes) and SCD.
• Among inherited long QT syndromes, precipitants and prognosis vary.

LONG QT

• Not rare: 3000 – 4000 deaths/y in children/adolescents
• Inherited/genetic
  • 12 types/genes, hundreds of different mutations
  • Variable “lethality”
  • AR associated with deafness
• Variable expression
• Acquired form
  • Medications/drugs
  • Electrolyte changes
• Increased risk of SCA with exercise, risk variable based on type
• SCA in athletes: not rare, numbers not clear
• EKG +, gene +, symptom +: Disqualified from competitive sports
ACQUIRED LONG QT

- Medications: [www.qtdrugs.org](http://www.qtdrugs.org)
  - Antiarrhythmics
  - Antibiotics: Levaquin, Zithromax (Z pack), erythromycin
  - Antidepressants: Tricyclics, Prozac, Celexa
  - Tamoxifen
  - diuretics
  - 140 other drugs
- Methadone
- Combinations of drugs
- Electrolytes: Low K+, Mg++, Ca++
- Genetic + Drugs, ? Unmasked congenital form
- Reversible
ACQUIRED LONG QT AND EXERCISE

- Drug + exercise interaction
- Electrolyte changes with exercise
  - Dehydration
  - Excessive “free water” intake
  - Losses with sweating
  - Diuretics
  - Greater risk with endurance events
- The Perfect Storm: Congenital substrate + drugs + exercise
BRUGADA SYNDROME

- Genetic
  - Genetic testing variable
  - Na+ channel
- EKG variable
  - Provocative testing
- Multiple types
- Male > Female
- Avg age at DX: 41
- Fever/hyperthermia trigger
- Night time trigger
- Treatment: ICD, limited medications
- Caution advised for competitive sports with no history of events
- With history of events or ICD low level sports only
Brugada Syndrome

- Autosomal dominant disorder resulting in increased risk of SCD.
- Multiple mutations in the cardiac sodium channel SCN5A have been described.
- Characterized by RBBB and ST segment elevations in V1-V3 on ECG.

![Typical ECG pattern for Brugada Syndrome](image)
CATECHOLAMINERGIC POLYMORPHIC VT
CPVT

- Genetic, at least 2 gene mutations
- Inherited
- Emotional and physical triggers. Symptoms: dizziness and syncope
- Usually presents in childhood and adolescence
- Treatment: Medical therapy, ICD + medical, Sympathectomy, Medical therapy for gene + asymptomatic.
- Generally recommend against competitive sports, ICD precludes contact sports
WOLFF PARKINSON WHITE

- 1/400
- Often Incidental finding
- Can present with symptoms
- Often first diagnosed in adulthood
- Risk of V-fibrillation
- Risk stratify asymptomatic Pts
- Ablation
- OK to participate in competitive sports once treated
Wolf-Parkinson-White

- WPW syndrome has been associated with an increased risk of SCD.
- The mechanism of SCD is most often atrial fibrillation or AVNRT that degenerates to VF.
- In up to 25% of patients with SCD due to WPW, pre-excitation and arrhythmias have been previously undiagnosed.

Typical pre-excitation pattern for WPW
COMMOTIO CORDIS

- Vulnerable moment
- High force, specific area
- Baseball, hockey, karate
- Kids more vulnerable
- 20% survival
- Boys > girls
- Prevention key
  - Training to avoid impact
  - ? vests
Cardiac Concussion

Source of Blow
- Hockey puck
- Lacrosse ball
- Baseball
- Fist or elbow

Primary determinants and triggers
- Precordial impact site
- Timed during upstroke of T wave

Contributing variables
- Greater hardness of projectile
- Smaller sphere
- Direct orientation
- Thinner, more compliant chest wall

Rapid increase in intracavitary pressure

20-msec window

Upstroke of T wave

VF
“Sudden Death in Young Athletes” Maron

NEJM 2003

Sudden Death in 387 Young Athletes

1. Hypertrophic Cardiomyopathy – 34%
2. Commotio Cordis – 20%
3. Coronary-artery Anomalies – 14%
2010 Update: Cardiac Concussion

A

No. of Commotio Cordis Events

Age at Event (yr)

≤10 11–15 16–20 21–25 ≥26

Survivors
Sudden deaths
Competitive sports
Recreational sports
Routine daily activities

B

Baseball
Softball
Hockey
Football
Soccer
Lacrosse
Boxing
Cricket
Rugby
Karate
Basketball

Recreational sports (N=48)
Competitive sports (N=122)

No. of Commotio Cordis Events
2010 Update: Cardiac Concussion

- 224 Cases: NEJM, B Maron, M Estes
- Mean Age = 15: 26% < 10yo
  - Range: 6mos – 50yo
- 95% Male, 78% White

**Survival rate**

- 15% 1990-1999
- 35% 2000-2009 (2006-09 > 50%)
Myocardial Bridging

- Myocardial bridging occurs when a portion of an epicardial coronary artery “tunnels” into the myocardium.
- Systolic vessel compression and delayed diastolic relaxation impair coronary blood flow in the intramyocardial segment.
- Although usually of little clinical consequence, myocardial bridging may infrequently result in exertional angina, infarction, and SCD.
- Myocardial bridging may be diagnosed on cardiac CT, MRI, or catheterization.

Contrast-enhanced EBCT image revealing an intramyocardial segment of the LAD

N Engl J Med 2003;349:1047
Premature Coronary Artery Disease

• CAD in young patients is frequently asymptomatic. Therefore, its incidence is likely underestimated.
• In an autopsy study, advanced coronary stenoses were noted in 20% of men and 8% of women aged 30-34 years.
• 19% and 8% of men and women aged 30-34 years, respectively, had ≥ 40% stenosis of the LAD.
• Coronary artery disease is the most common cause of SCD among “masters” athletes (> 35 years old).

Circulation 2000;102:374
Athletes at Risk for SCA

- Chief complaint of syncope
- Chest Pain with or post activity
- History of palpitations
- Family History of Sudden death
- Abnormal EKG
Athlete SCA: Have We Changed the Playing Field?

Emergency Department

- **Athlete Collapse** – Assume Cardiac Etiology (Sentinel Seizure)
- **EKG Attention**: Delta and Epsilon Waves, LQT
- **Syncope, Near Syncope, Chest Pain Work Up**: Consider advanced imaging, Cardiac CT, MRI* vs ECHO
Screening

• Due to the devastating nature of SCD and the potential to prevent such deaths by diagnosing associated disorders noninvasively, clinicians have a strong incentive to screen athletes.

• However, the following obstacles prevent widespread screening with noninvasive testing:
  – Large number of competitive athletes (8 million in the U.S., including high school, collegiate, professional)
  – Low prevalence of underlying congenital heart disease
  – Number of disorders to consider, each with different optimal testing modalities
  – Impact of false-positive studies (substantial when screening for rare diseases; possible medicolegal implications)
  – No randomized trials evaluating the impact of pre-participation screening on the incidence of SCD
Screening

• In an observational series from Italy, a mandatory screening program including ECG was associated with a decrease in the annual incidence of SCD in athletes from 3.6 to 0.4 per 100,000 person-years from 1980 to 2004.

• AHA guidelines differ from those of the European Society of Cardiology (ESC) and the International Olympic Committee (IOC) such that routine noninvasive testing (including ECG) is not recommended.
AHA Screening Recommendations

• Younger competitive athletes (<35)
  – Complete personal/family history and physical exam
  – Performed by physicians or certified non-physicians
  – q2 years for high school and yearly for college/pro

• Masters athletes (>35)
  – Complete personal/family history and physical exam
  – Exercise testing for moderate-to-high risk patients (men >40, women >50 with one or more CAD risk factors; symptoms suggestive of CAD; ≥65 regardless of risk factors/symptoms)

• Recreational athletes
  – No explicit AHA guidelines; exercise testing recommended in patients at high risk for CAD

Circulation 2007;115:1643
12-Element AHA Pre-Participation Screening Recommendations

• Personal history (confirmed by parent if minor)
  – Exertional chest discomfort
  – Unexplained syncope/presyncope
  – Excessive exertional fatigue/dyspnea
  – Prior heart murmur
  – Elevated blood pressure

• Family history (confirmed by parent if minor)
  – Premature death due to heart disease before age 50
  – Disability due to heart disease in relative <50
  – Specific knowledge of certain cardiac conditions (HCM, other CM, ion channelopathy, Marfan’s, arrhythmias)

• Physical examination
  – Cardiac exam (supine and standing)
  – Femoral pulses
  – Physical stigmata of Marfan’s
  – Bilateral blood pressure readings

*Positive finding of any 1 element warrants referral to cardiovascular specialist +/- further testing
WHAT ABOUT EKGs

• Not recommended routinely in US
• Required in Europe
• Controversial
  • Not clear it helps
  • Athletes often have EKG changes that are “normal”
  • False negatives, False positives
  • Cost of EKGs, Cost of additional testing, Cost of disqualifying athletes
  • Estimated $80,000 to find one case
High False Positive Rate

False positive rate 10%

False positive rate 16.9%

False positive rate 17.3%

UNACCEPTABLY HIGH!
THE ADULT ATHLETE

CARDIOVASCULAR DISEASE IS THE PRIMARY CAUSE OF DEATH IN ADULT ATHLETES
THE ADULT ATHLETE

- Primary Cause: Coronary Artery Disease
- Cardiomyopathy
- Vascular Disease
- Arrhythmia
- Valvular Heart Disease
The adult athlete can still have almost any of the conditions of the young athlete.
CORONARY ARTERY DISEASE

STILL NUMBER ONE
JIM FIXX
1932 - 1984
WHAT IS THE RISK?

- 800,000 Heart attacks/year
- 400,000 Sudden Cardiac Death
- Sudden Death: First symptom in 50%
- 2 – 3 X as likely to suffer a cardiac event during exercise in those with disease
- Numbers during exercise unknown
  - Marathon Risk: 1/50,000 – 100,000/race
  - 2012: 550,000 finished a marathon
  - 2011: 500,00 started their first marathon
- Nobody is keeping track outside of organized events
- Odds are there are many cardiac events during unorganized exercise that are not reported
Conclusions

• SCD in competitive athletes may result from a variety of disorders that may be detected by noninvasive testing.
• Noninvasive testing must be interpreted carefully in order to distinguish the physiological effects of exercise training from pathology.
• AHA guidelines do not endorse routine pre-participation screening with noninvasive testing.
• However, noninvasive testing plays a critical role in the evaluation of competitive athletes with positive findings on screening history and physical examination.
Thank you