SUDDEN CARDIAC DEATH OF ATHLETES

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Sudden Cardiac Death

- Sudden Cardiac Death (SCD)
- Leading medical cause of death in young athletes
- New research suggests incidence
  - 1 in 50,000 athlete-years in college athletes
  - 1 in 80,000 athlete-years in high school athletes
- Males & African Americans are at higher risk
  - Men’s basketball: 1 in 9,000 athlete-years
Sudden Cardiac Death

- Some data suggests that athletes may be at higher risk to experience SCD because of their increased level of physical activity that can lead to arrhythmias
- Other data suggests SCD is more common in young non-athletes vs young athletes
SCD

- SCD is the presenting symptom of underlying cardiovascular pathology in 50-90% of athletes
  - Significantly limits usefulness of history-based screen
SCD

- Warning symptoms
  - Exertional chest pain
  - Exertional syncope
  - Exertional near-syncope
  - Dyspnea or fatigue disproportionate to level of exertion
  - Palpitations
  - Irregular heart beats
- Family History of sudden un-explained death or SCD < 50 y/o
- Family history of cardiac disease known to cause SCD
SCD

• SCD in athletes < 35 Y/O
  • In most cases, structural heart disease was present
  • Hypertrophic Cardiomyopathy (HCM), anomalous origin of a coronary artery, arrhythmogenic right ventricular cardiomyopathy (ARVC), myocarditis, & coronary atherosclerosis
  • Similar findings in US, UK, and US Military
    • Maron BJ. Circulation 2007; 115:1643
    • Eckert RE. Ann Intern Med 2004; 141:829
    • Finocchiaro G. J Am Coll Cardiol 2016; 67;2108

• SCD in athletes < 35 Y/O in Northern Italy
  • ARVC was the most common abnormality, then coronary atherosclerosis and 3rd most common cause was anomalous origin of a coronary artery
  • Northern Italy has a known higher prevalence of ARVC
SCD

- SCD during athletics also occurs in the absence of structural heart disease known as primary electrical disease
  - Long QT Syndrome
  - Short QT Syndrome
  - Brugada Syndrome
  - Catecholaminergic polymorphic ventricular tachycardia
  - Wolf-Parkinson-White Syndrome (WPW)

- SCD precipitated from trauma
  - Commotio cordis

- SCD in athletes >35 Y/O
  - **Coronary artery disease** is the most common cause of SCD during exercise
Table 1. Causes of Sudden Death in 387 Young Athletes*

<table>
<thead>
<tr>
<th>Cause</th>
<th>No. of Athletes</th>
<th>Percent</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hypertrophic cardiomyopathy</td>
<td>102</td>
<td>26.4</td>
</tr>
<tr>
<td>Commotio cordis</td>
<td>77</td>
<td>19.9</td>
</tr>
<tr>
<td>Coronary artery anomalies</td>
<td>53</td>
<td>13.7</td>
</tr>
<tr>
<td>Left ventricular hypertrophy of indeterminate causation†</td>
<td>29</td>
<td>7.5</td>
</tr>
<tr>
<td>Myocarditis</td>
<td>20</td>
<td>5.2</td>
</tr>
<tr>
<td>Ruptured aortic aneurysm (Marfan syndrome)</td>
<td>12</td>
<td>3.1</td>
</tr>
<tr>
<td>Arrhythmogenic right ventricular cardiomyopathy</td>
<td>11</td>
<td>2.8</td>
</tr>
<tr>
<td>Tunnelled (bridged) coronary artery‡</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 coronary artery disease</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 mitral valve degeneration</td>
<td>9</td>
<td>2.3</td>
</tr>
<tr>
<td>Asthma (or other pulmonary condition)</td>
<td>8</td>
<td>2.1</td>
</tr>
<tr>
<td>Heat stroke</td>
<td>6</td>
<td>1.6</td>
</tr>
<tr>
<td>Drug abuse</td>
<td>4</td>
<td>1.0</td>
</tr>
<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 causing structural cardiac injury</td>
<td>3</td>
<td>0.8</td>
</tr>
<tr>
<td>Ruptured cerebral artery</td>
<td>3</td>
<td>0.8</td>
</tr>
</tbody>
</table>

*Data are from the registry of the Minneapolis Heart Institute Foundation (3). †Findings at autopsy were suggestive of HCM but were insufficient to be diagnostic. ‡Tunnelled coronary artery was deemed the cause of death in the absence of any other cardiac abnormality. §The long QT syndrome was documented on clinical evaluation. Source: Reproduced from Maron B.J. (3) with permission of the Massachusetts Medical Society.
Hypertrophic Cardiomyopathy (HCM)

- Most common cause of Sudden Cardiac Death < 35 y/o
- Prevalence of 1 in 500 in general population
- Reported to cause 2-36% of SCD in athletes
  - Different studies report different rates of SCD
    - Corrado 2003 (Italy): 2%
    - Maron 2007 (USA): 36%
    - Harmon 2011 (USA): 3%
HCM

- Pathologic Features
  - Symmetric LV Hypertrophy (usually involving ventricular septum)
  - **LV wall thickness ≥ 16 mm**
    - Normal ≤ 12 mm
    - Borderline 13-15 mm
  - Non-dilated left ventricle
  - Histological analysis shows disorganized cellular architecture
    - Intramural tunneling (myocardial bridging)
HCM

- Presenting symptom of HCM is SCA in 80% of cases
- Sxs may include
  - Exertional chest pain
  - Dyspnea
  - Lightheadedness
  - Syncope

- Physical Exam
  - Harsh systolic ejection murmur that increases with valsalva & decreases with maneuvers that increase venous return
    - Cardiac exam auscultation in supine, sitting & squatting positions
    - Murmur goes away or decreases when you have them squat down
HCM

- **EKG**
  - Can be abnormal in up to 90% of athletes
    - T wave inversion in lateral or inferolateral leads
    - ST segment depression
    - Pathologic Q waves
    - Complete LBBB

- **Echo**
  - Standard to confirm diagnosis
    - LV wall thickness ≥ 16 mm

- **MRI**
  - Additional value in identifying segmental hypertrophy in anterolateral free wall or at apex
HCM

- **Return to Play (RTP)**
  - Exercise increases risk of ventricular tachycardia/fibrillation
  - Exercise is a modifiable risk factor
  - Athletes with HCM should not participate in strenuous exercise
  - Which sports?
36th Bethesda Conference

Eligibility Recommendations for Competitive Athletes With Cardiovascular Abnormalities
HCM – Which Sports?

• Excluded from most competitive sports
• Can possibly participate in Class 1A
<table>
<thead>
<tr>
<th>Increasing Static Component</th>
<th>III. High (&lt;50% MVC)</th>
<th>II. Moderate (20-50% MVC)</th>
<th>I. Low (&lt;20% MVC)</th>
</tr>
</thead>
<tbody>
<tr>
<td>American football*, Field events (jumping), Figure skating*, Rodeoing*, Rugby*, Running (sprint), Surfing*, Synchronized swimming†</td>
<td>American football*, Field events (jumping), Figure skating*, Rodeoing*, Rugby*, Running (sprint), Surfing*, Synchronized swimming†</td>
<td>Basketball*, Ice hockey*, Cross-country skiing (skating technique), Lacrosse*, Running (middle distance), Swimming, Team handball</td>
<td>Baseball/Softball*, Fencing, Table tennis, Volleyball</td>
</tr>
<tr>
<td>Boxing*, Canoeing/Kayaking, Cycling*, Decathlon, Rowing, Speed-skating*, Triathlon†</td>
<td></td>
<td></td>
<td>Badminton, Cross-country skiing (classic technique), Field hockey*, Orienteering, Race walking, Racquetball/Squash, Running (long distance), Soccer*, Tennis</td>
</tr>
</tbody>
</table>

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A. Low (<40% Max \(O_2\))

B. Moderate (40-70% Max \(O_2\))

C. High (>70% Max \(O_2\))

Increasing Dynamic Component
Commotio Cordis

- Occurs after the chest wall is struck with a blunt object
  - During ventricular repolarization
    - Just before peak of T wave
  - Can lead to ventricular fibrillation

- Structurally normal heart
Commotio Cordis

• Instantaneous collapse after the blow
  • Can be right after the blow or after about 10 seconds

• Defibrillate STAT
  • 25% survival rate if defibrillation is within 3 minutes
  3% after 4 minutes
  • Animal model showed 90% survival at 2 minutes
Commotio Cordis

- Prevention?
  - Chest protectors
    - Have not been shown to be effective
  - Safety balls
    - In animal models have been shown to be associated with decreased arrhythmias

- Education
  - Avoid chest shot blocking
  - Emergency Action Plan
    - AED
Congenital Coronary Arteries (CCA)

- CCA’s lead to ischemia during exercise
- Most common CCA is abnormal origin of left coronary artery arising from the right sinus of valsalva
  - Acute angled take off
  - Hypoplastic Ostium
  - Impingement of CCA as it traverses between expanding great vessels

- Symptoms
  - Chest pain
  - Exertional syncope
  - SCA
CCA

- ECG
  - Do not typically identify CCAs

- Echo
  - Can identify in 80-97% of cases
    - Specify that you want evaluation for CCAs

- May need CT Angiography, Cardiac MRI or Coronary Angiography

- RTP
  - Excluded from competitive sports
  - After CCA correction can return after 3 months if stable with maximal exercise testing
Arrhythmogenic Right Ventricular Cardiomyopathy (ARVC)

- 1 case per 5000 people
- 3 – 27% of SCD in athletes

Pathologic Features
- Progressive fibro fatty replacement of right ventricular myocardium
  - Leads to RV thinning & dilatation

Sx
- Syncope
- Chest pain
- Palpitations
- SCA
ARVC

• P/E
  • Normal

• Echo
  • RV Dilatation & wall thinning
  • ↓ RVEF
  • RV aneurysms
  • In figure: moderator band is enlarged with attachments to septum & free wall with RV dilatation

• Cardiac MRI or CT
Myocarditis

- Acute inflammatory process involving the myocardium
- Coxsackie B Virus > 50% of time
- Echovirus
- Adenovirus
- Influenza
- Chlamydia Pneumoniae

- Lymphocytic infiltration of myocardium with necrosis of myocytes
Myocarditis

• Sx
  • Present with flu-like illness followed by progressive exercise intolerance
  • Dyspnea
  • Cough
  • Orthopnea
    • Can lead to dilated cardiomyopathy
    • → SCD can develop in active or healed myocarditis

• P/E
  • S3 gallop
  • Edema & pulmonary rales
Myocarditis

- ECG
  - Diffuse Low voltage
  - ST & T wave changes
  - Heart block
  - Ventricular arrhythmias

- Labs
  - Leukocytosis
  - ↑ ESR
  - ↑ CRP
  - ↑ Myocardial enzymes

- Echo
  - Dilated LV, global hypokinesis, ↓ LVEF
Myocarditis RTP

• Active myocarditis
  • Should not compete

• Return to play is variable after resolution (3-6 months)
  • RTP if:
    • Ventricular systolic function returned to normal
    • Serum myocardial injury markers are normal
    • Associated arrhythmias resolved
Dilated Cardiomyopathy

- 1 case per 2500 people
- 0-8% of SCD cases

Pathologic Features
- Left Ventricle (LV) Dilatation
- Systolic Dysfunction
- Normal LV wall thickness

Secondary Dilated Cardiomyopathy
- Results from untreated/suboptimal tx of HTN, ischemic heart disease, viral myocarditis, infiltrative disease (e.g., sarcoidosis), autoimmune or toxins (e.g., etoh)
Dilated Cardiomyopathy

• Sx
  • Progressive exertional intolerance
  • Dyspnea
  • Orthopnea
  • Edema
  • Fatigue

• P/E
  • S3 or S4
  • Holosystolic murmur
Dilated Cardiomyopathy

- **ECG**
  - SVT or ventricular tachyarrhythmia's, BBB or AV node block

- **Echo**
  - Dilated LV with ↓ LVEF

- **RTP**
  - Excluded from all competitive sports except possibly low intensity class 1A
Aortic Rupture/Marfan Syndrome

- Incidence of marfan syndrome
  - 1 in 5,000 - 10,000

- Pathologic Features
  - Progressive dilatation & weakness of proximal aorta
  - Myxomatous degeneration of mitral & aortic valves leading to valvular dysfunction

- Sx
  - Related to aortic root dissection
    - Chest pain
    - Thoracic pain
    - Sxs of heart failure from aortic valve incompetence

- P/E
  - Highly variable manifestation in adolescence & young adulthood
  - Ghent nosology
<table>
<thead>
<tr>
<th>System</th>
<th>Major criterion</th>
<th>Involvement</th>
</tr>
</thead>
<tbody>
<tr>
<td>Skeletal</td>
<td>At least 4 of the following features:</td>
<td>2 of the major features, or 1 major feature and 2 of the following:</td>
</tr>
<tr>
<td></td>
<td>- pectus carinatum</td>
<td>- pectus excavatum</td>
</tr>
<tr>
<td></td>
<td>- pectus excavatum requiring surgery</td>
<td>- joint hypermobility</td>
</tr>
<tr>
<td></td>
<td>- ULSR &lt; 0.86 or span:height &gt; 1.05</td>
<td>- high palate with dental crowding</td>
</tr>
<tr>
<td></td>
<td>- wrist and thumb signs</td>
<td>- characteristic face</td>
</tr>
<tr>
<td></td>
<td>- scoliosis &gt; 20° or spondylolisthesis</td>
<td></td>
</tr>
<tr>
<td></td>
<td>- reduced elbow extension (&lt;170°)</td>
<td></td>
</tr>
<tr>
<td></td>
<td>- pes planus</td>
<td></td>
</tr>
<tr>
<td></td>
<td>- protrusio acetabulae</td>
<td></td>
</tr>
<tr>
<td>Ocular</td>
<td>Lens dislocation (ectopia lentis)</td>
<td>Flat cornea</td>
</tr>
<tr>
<td>Cardiovascular</td>
<td>Dilatation of the aortic root</td>
<td>Increased axial length of globe (causing myopia)</td>
</tr>
<tr>
<td></td>
<td>Dissection of the ascending aorta</td>
<td>Hypoplastic iris or ciliary muscle (causing decreased miosis)</td>
</tr>
<tr>
<td>Pulmonary</td>
<td>None</td>
<td>Mitral valve prolapse</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Dilatation of the pulmonary artery, below age 40</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Calcified mitral annulus, below age 40</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Other dilatation or dissection of the aorta</td>
</tr>
<tr>
<td>Skin/integument</td>
<td>None</td>
<td>Spontaneous pneumothorax</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Apical blebs</td>
</tr>
<tr>
<td>Dura</td>
<td>Lumbar sacral dural ectasia</td>
<td>Striae atrophicae</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Recurrent or incisional hernia</td>
</tr>
<tr>
<td>Genetic findings</td>
<td>Parent, child or sibling meets these criteria independently</td>
<td>None</td>
</tr>
<tr>
<td></td>
<td>Fibrillin 1 mutation known to cause Marfan syndrome</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Inheritance of DNA marker haplotype linked to Marfan syndrome in the family</td>
<td></td>
</tr>
</tbody>
</table>

Having one of the features listed constitutes a major criterion or system involvement for all systems except the skeletal system, where more than one feature is needed.
ULSR, upper to lower segment ratio
Aortic Rupture/Marfan Syndrome

- I think someone has Marfan Syndrome based on Ghent, what do I do next
  - Can also use revised Ghent Criteria that provides point total
    - ≥ 7 points indicates major systemic involvement

- Echo
  - Aortic root dilatation
  - Aortic regurgitation

- Slit light ophthalmologic examination for ectopia lentis

- Lumbar MRI or CT of Lumbar Spine
  - Review for dural ectasia
Aortic Rupture/Marfan Syndrome

• Dural Ectasia on MRI
Aortic Rupture/Marfan Syndrome

• RTP
  • Based on aortic root dimensions & pathologic features of disease
  • Class IA & IIA if they do not have any of the following
    • Aortic root dilatation
      • Specific for adults & children & adolescents
      • Moderate to severe mitral regurgitation
      • Family history of dissection or sudden death in marfan relative
    • Need to have aortic root evaluated Q 6 months via Echo
Coronary Artery Disease

• Most common cause of SCD in athletes > 35 Y/O
• Incidence of exercise related sudden death in adults is 1 in 15,000 – 18,000

• Pathologic Features
  • Most often caused by atherosclerotic plaque disruption
  • Exercise may be a stimulus for plaque disruption
  • Development is progressive & related to coronary risk factors
    • HTN, DM, Dyslipidemia, Tobacco Use, ILDU, Family History of premature atherosclerotic disease

• Sx
  • Exertional chest pain, angina, palpitations, SCD
Coronary Artery Disease

- ECG
  - Evidence of prior ischemia

- Exercise stress test
  - ST segment depression

- Stress echo
  - Wall motion abnormality

- Cardiac CT, Cardiac MRI, Angiography
  - Coronary artery narrowing
Coronary Artery Disease

- RTP
  - Risk stratified based on extent of disease
    - Co-morbidities
    - Stability after medical management
    - Percutaneous coronary interventions
    - Surgery
Primary Electrical Disease

- Channelopathies
  - Diseases predisposing to potentially lethal ventricular tachyarrhythmia's
  - Characterized by abnormalities in ion-channel proteins leading to dysfunction of sodium, potassium, calcium and other ion transport across cell membranes
  - Likely that many deaths secondary to SCD with pathologically normal hearts are from channelopathies
Long QT Syndrome (LQTS)

- Most common channelopathy

- Prolonged ventricular repolarization & QT interval corrected for heart rate ($QT_c$)

- Six different types
  - LQTS-1, LQTS-2, LQTS-3 are most common

- Sx
  - Syncope
  - Pre-syncope
  - Family Hx of sudden unexplained death or sudden infant death
LQTS

- ECG
  - $QT_c \geq 470$ ms in males
  - $QT_c \geq 480$ ms in females

- RTP
  - Decisions guided by heart rhythm specialist
  - Can consider class IA sports
Short QT Syndrome

• Only recently described in 2000

• Pathologic Features
  • Hyper-functioning of potassium channel

• Sx
  • Palpitations
  • Syncope
  • A-Fib
  • SCA
  • SCD
Short QT Syndrome

• ECG
  • $\text{QT}_c < 340 \text{ ms}$

• RTP
  • Bethesda recommends restricting from all sports except possibly Class IA
Brugada Syndrome

• Rare channelopathy
• Most prevalent in males from SE Asia

• Pathologic Features
  • Abnormalities in sodium channels

• Sx
  • Syncope
  • SCA
  • SCD
  • Sudden death during sleep
Brugada Syndrome ECG

• Down-sloping ST segment elevation in V1-V3
• RBB
• Findings may not be noticed unless unmasked with sodium channel blocker (e.g., flecainide)
Brugada Syndrome

- RTP
  - Bethesda recommends restricting from all sports except possibly Class IA
Catecholaminergic Polymorphic Ventricular Tachycardia (CPVT)

- Familial disorder
- Stress-induced ventricular arrhythmias resulting in SCD in children & young adults

- Abnormalities of calcium channel function

- Sx
  - Syncope
  - SCA
  - SCD
  - Polymorphic V-Tach triggered by strenuous exercise or emotion
CPVT

- ECG
  - Often normal
  - Some patterns have prominent U waves

  - Exertion or epinephrine challenge can induce ventricular tachycardia

- Symptomatic patients have a poor prognosis unless treated with Implantable Cardioverter-Defibrillator (ICD)

- RTP
  - Do not participate in competitive sports
  - With ICD - Bethesda recommends considering Class IA sports
Wolf-Parkinson-White Syndrome (WPW)

- Approximately 1% of ECGs will show changes consistent with WPW
  - Only a small subset will go on to develop the associated arrhythmias associated with WPW

- Pathologic Features
  - Tachyarrhythmia caused by accessory pathway that directly connects the atria & ventricles & bypasses the AV node
  - Arrhythmias
    - Atrioventricular tachycardia
    - A-Fib
    - A-Flutter

- Sx
  - Palpitations, syncope, near-syncope
WPW - ECG

- Slurring of QRS upstroke (delta wave)
- ECG can also show PR < 120 ms & QRS > 120 ms
WPW

- Once diagnosed, risk stratify with exercise stress testing

- RTP
  - High Risk Pathways
    - Catheter or surgical ablation with normal repeat EP studies or 2-4 weeks without sx can undergo subsequent RTP
  - Low Risk Pathways
    - RTP without ablation is reasonable
ECG as part of PPE?
ECG ROUTINE SCREENING

• IOC/ESC (European Society of Cardiology) recommends EKG on all athletes (2004)
  • largely related to Italian data (Corrado) of death rate 1/28,000 in 1981 to 1/250,000 in 2004 (79% reduction)
ECG ROUTINE SCREENING

- AHA – response was that ECG is not recommended in the US

- Different population (less ARVD/more HCM) – 1/100,000-300,000 in U.S. (discrepancy as no national registry)
  - $330,000 per life saved (2 billion overall) due to many false-positives (40% of trained athletes have EKG abnormalities) & follow-up in addition to screening cost
  - Too many athletes in US & not enough trained physicians to read the EKG’s (Italy screens 3-5 million annually age 12-35 & 5-10.5 million in U.S.)
  - Will not identify all conditions that cause sudden death – Marfan’s, anomalous coronaries, catecholamine-induced VT
  - Lack of randomized studies – fear that screening may actually increase death rate from treatment-related procedural failures
ROUTEINE SCREENING

• ECG proponents
  • 134 sudden deaths – H & P only raised a suspicion in 3% & an accurate diagnosis was only made in <1%.
  • More is being done for the college/pro athlete (double-standard)
  • H & P being done by many inexperienced providers
ROUTINE SCREENING

• ECG Proponents
  • 70% of athletes with cardiac disease can be found by EKG
    • HCM
    • ARVD (inverted T-waves in leads V1-V3 with epsilon wave at ST segment)
    • Short or long QT
    • WPW (pre-excitation)
    • Dilated cardiomyopathy
    • Brugada Syndrome (RBBB & coved ST elevation in V1/V2)
    • Myocarditis
    • Congenital heart block
    • Rarely MVP
ROUTINE SCREENING

• ECG Proponents
  • Those at most risk (young black males) are the least likely to be able to get screened unless mandatory system
  • Should be extended to all adolescents as many die during sleep (long QT & Brugada)
12 ELEMENT AHA RECOMMENDATIONS FOR PPE

• **Personal Medical History**
  1. Exertional chest pain/discomfort
  2. Unexplained syncope/near-syncope (not vasovagal syncope)
  3. Excessive exertional and unexplained dyspnea/fatigue, associated with exercise
  4. Prior recognition of a heart murmur
  5. Elevated systemic blood pressure

• **Family History**
  6. Premature death (sudden and unexpected, or otherwise) before age 50 years due to heart disease, in ≥1 relative
  7. Disability from heart disease in a close relative <50 years of age
  8. Specific knowledge of certain cardiac conditions in family members: hypertrophic or dilated cardiomyopathy, long-QT syndrome or other ion channelopathies, Marfan syndrome, or clinically important arrhythmias

• **Physical Examination**
  9. Heart murmur‡
  10. Femoral pulses to exclude aortic coarctation
  11. Physical stigmata of Marfan syndrome
  12. Brachial artery blood pressure (sitting position)
PERSONAL MEDICAL HISTORY

1. Exertional chest pain/discomfort
2. Unexplained syncope/near-syncope (not vasovagal syncope)
3. Excessive exertional and unexplained dyspnea/fatigue, associated with exercise
4. Prior recognition of a heart murmur
5. Elevated systemic blood pressure
FAMILY HISTORY

- 6. Premature death (sudden and unexpected, or otherwise) before age 50 years due to heart disease, in ≥1 relative
- 7. Disability from heart disease in a close relative <50 years of age
- 8. Specific knowledge of certain cardiac conditions in family members: hypertrophic or dilated cardiomyopathy, long-QT syndrome or other ion channelopathies, Marfan syndrome, or clinically important arrhythmias
PHYSICAL EXAMINATION

• 9. Heart murmur
  • Listen Supine, sitting, & squatting positions
  • **Murmur of HCM resolves with squatting**

• 10. Femoral pulses to exclude aortic coarctation
  • Radial & Femoral Artery Pulse should be symmetric

• 11. Physical stigmata of Marfan syndrome

• 12. Brachial artery blood pressure (sitting position)
MEDICOLEGAL ISSUES

• The appropriate H & P is the current standard of care despite a low sensitivity & specificity for detecting CV abnormalities

• A physician who clears an athlete to play is not necessarily liable for an injury or death caused by an undiscovered cardiovascular condition unless they have deviated from the established standard of care in examining or working up the athlete
QUESTIONS
THANK YOU
References

• Dural Ectasia Case courtesy of Dr Franco Ruales, Radiopaedia.org, rID: 16114.
• Eckert RE. Sudden Death in Young Adults: A 25-year Review of Autopsies in Military Recruits. Ann Intern Med 2004; 141:829
• Maron, BJ. Comparison of the frequency of sudden cardiovascular deaths in young competitive athletes versus nonathletes: should we really screen only athletes? AM J Cardiol. 2016;117(8):1339-1341.