The Pre Participation Physical Exam:
More Than Just Checking a Box

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Purpose of Pre Participation Exams

• The primary goal of CV screening of athletes is to identify underlying cardiac disorders predisposing to SCA/D with the intent to reduce morbidity and mortality by mitigating risk through individualized, patient-centered, and disease specific medical management. (AMSSM Position Statement)

• Evaluate for medical and orthopedic contraindications for participation

• Address / maximize management of chronic medical problems
When to do them?

• 2015 AHA Bethesda Conference, AMSSM, AAP
  • High school and below - 2 years (AIA=yearly)
  • College - yearly
• 4-6 weeks before the season starts to allow sufficient time for further evaluation if necessary
Where to do them?

- American Academy of Pediatrics position statement recommends that all pre-participation physical examinations occur in a physician’s office, ideally with the athlete’s primary care physician or sports medicine specialist.
AHA/ACC guidelines

• Revisited in March 2007 in response to 25 year study in Italy and in 2016
  • No changes from 2004 Bethesda conference
• Standardized medical forms recommended
• Parents should fill out for minors
• 14 element screening strategy
AHA/ACCF guidelines

1. Exertional chest pain/discomfort
2. Unexplained syncope or near-syncope (deemed not to be vasovagal)
3. Exertional dyspnea/fatigue
4. Recognition of heart murmur
5. Elevated systemic blood pressure (3 Readings)
6. Death from heart disease in relative <50 years old
7. Disability of family member from heart disease at age <50

8. Family history of Marfan’s, long QT syndrome, ion channelopathies (Brugada), clinically important arrhythmias, hypertrophic or dilated cardiomyopathies

9. Auscultation for detection of heart murmurs (LVOT obstructions)
AHA/ACCF guidelines

10. Signs of Marfan’s
11. Femoral / radial pulse examination to rule out coarctation of the aorta
12. Seated, bilateral brachial BP’s
Standard Questions

• Do you ever get chest pain, palpitations, light headed, dizziness, pass out or almost pass out during or after exercise?

• Do you have any family history of heart disease or death at a young age (<50 years old) from a heart problem?

• Do you have a history of a heart murmur or have you ever been told you could not participate in sports due to a heart problem?
Auscultation

- Any diastolic or grade II systolic murmur greater needs further evaluation
  - Provocative maneuvers
    - Murmur of HOCM increases with Valsalva or decreases with squatting and increases with standing
      - Reverse for aortic stenosis
  - EKG and Echo for any abnormalities on screening history and physical examination
Blood Pressure

• 3 separate elevated blood pressure readings to diagnose HTN

• Varies by age
  • Mild to moderate w/o end organ damage - no restriction to activity
  • Severe to very severe - restrict from high static sports until BP controlled

• All athletes with diagnosis of HTN receive cardiac work-up
Sudden Cardiac Death (SCD)
Definition:

- Unexpected death that occurs within 1 hour after the onset of symptoms or change in clinical status.
- Unexpected death occurring within 24 hours, if un-witnessed, in a previously asymptomatic person.
Sudden Cardiac Death (SCD)

- 1:200,000 US high school athletes*
- <300 deaths/year in all levels of competition (? 25 million sports participants in the US)
- 9:1 male : female
  - Exercise related death rate for females would be 1:769,000

*VanCamp et al. MSSE; 1995.
Sudden Cardiac Death

• Pete Maravich - Anomalous LCA

• Flo Jo – HCM

• Hank Gathers – HCM

• Krissy Taylor – ARVD

• Darryl Kyle - CAD
Epidemiology

• In the general adult (>35yrs) population:
  • SCD accounts for 50% of all cardiac deaths
  • ~ 300,000 events / year
  • Incidence of 1 – 2 per 1,000 patient years
  • Usually the result of occult coronary artery disease.

Maron B. JACC 32:1881-4, 1998
Epidemiology
Epidemiology

In the high school athlete population:
The risks are orders of magnitude lower!

• Risk of SCD:
  – 1 : 217,400 participants per year
  – 0.46 / 100,000 person years annually
  – 1 : 72,500 for one athlete over 4 year HS career

“It’s a very large haystack to look through…”

Epidemiology

• Most SCD’s occurred in the most popular sports.
  Maron B. JAMA 1996 Jul 17;276(3):199-204
  SCD: Epidemiology

• Most SCD’s occur during practice or competitions.
  Maron B. JAMA 1996 Jul 17;276(3):199-204
Epidemiology

• Cardiomyopathies and coronary abnormalities account for most of the cases of SCD in young athletes.

Maron B. JAMA 1996 Jul 17;276(3):199-204
Fig. 1. Distribution of cardiovascular causes of sudden death in 1,435 young competitive athletes. ARVC, arrhythmogenic right ventricular cardiomyopathy; AS, aortic stenosis; CAD, coronary artery disease; C-M, cardiomyopathy HCM, hypertrophic cardiomyopathy; HD, heart disease; LAD, left anterior descending; LVH, left ventricular hypertrophy. (Courtesy of the Minneapolis Heart Institute Registry, 1980–2005, Minneapolis, MN; with permission.)
Causes of Sudden Death in Athletes

1. Hypertrophic Cardiomyopathy
2. Congenital Coronary Artery Syndrome
3. Marfan’s Syndrome
4. Coronary Artery Disease
5. Sickle Cell Trait
6. Asthma
7. Others
Hypertrophic Obstructive Cardiomyopathy

- Most common cause of SCD in athletes in the USA
Hypertrophic Obstructive Cardiomyopathy

• Exam
  – High frequency SEM @ LUSB
  – Increased with Valsalva, decreased with squatting and increases with standing
  – S4 common
Hypertrophic Obstructive Cardiomyopathy

- Asymmetric septal hypertrophy.
- Myocardial fiber disarray and interstitial fibrosis.
Hypertrophic Obstructive Cardiomyopathy

- Myocyte hypertrophy in the absence of known stimuli for ventricular hypertrophy (AS, CoA, HTN).
- Due to mutations of sarcomeric proteins.
- Autosomal dominant inheritance, variable and age dependent penetrance.
- Sporadic cases common (new mutations).
- Prevalence: 1-2 per 1,000 persons
Fig. 3. Morphologic components of disease process in HCM. (A) Heart sectioned in cross-sectional long-axis plane. LV wall thickening is asymmetric, confined primarily to the ventricular septum (VS), which bulges prominently into small cavity. (B) Scarred myocardium
Hypertrophic Obstructive Cardiomyopathy

• Clinical Signs & Symptoms
  • Dyspnea on exertion
  • Chest pain
  • Syncope or pre-syncope
  • Palpitations
  • Sudden death
  • Mid-systolic murmur that increases in intensity with standing (dynamic auscultation)

• None of the symptoms are specific for HCM!
  Mid-systolic murmur fairly specific but insensitive
  (hypertrophic non-obstructive cardiomyopathy)
ECG in Hypertrophic Obstructive Cardiomyopathy

- >90% EKG’s abnormal
- Q III, Q aVF, Q V6, S V1, R V6
Hypertrophic Obstructive Cardiomyopathy

- Conventional ECG criteria for LVH in children lack sufficient sensitivity and specificity for diagnosis of HCM.

<table>
<thead>
<tr>
<th>TABLE I</th>
<th>Number of Patients With Absolute Value of Given Electrocardiographic Parameter &gt; 95th Percentile</th>
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<tbody>
<tr>
<td>Lead</td>
<td>HC</td>
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<tr>
<td>Q III</td>
<td>15/37 [40%]</td>
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<tr>
<td>Q aVF</td>
<td>16/37 [43%]</td>
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<tr>
<td>Q V6</td>
<td>16/37 [43%]</td>
</tr>
<tr>
<td>SV1</td>
<td>19/36 [53%]</td>
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<tr>
<td>RV6</td>
<td>7/37 [19%]</td>
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<tr>
<th>TABLE IV</th>
<th>Summed Total Number of Electrocardiographic Parameters &gt; 95th Percentile</th>
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<td>ECG Parameters &gt; 95th Percentile</td>
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<td>5</td>
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Hypertrophic Obstructive Cardiomyopathy

- Newer ECG protocols developed with better accuracy in differentiating between HCOM and athletic heart
Hypertrophic Obstructive Cardiomyopathy

- Echocardiography is the gold standard for the diagnosis of HCM.
Hypertrophic Obstructive Cardiomyopathy

Echocardiogram

- Left ventricular end diastolic septum thickness $\geq 15\text{mm}$ for adults, $<12\text{mm}$ is normal
- $>2$ standard deviations from the mean for relative to body surface area in children
Hypertrophic Obstructive Cardiomyopathy

• What if ventricular septum is 13-15mm?
  – Athletic heart or HOCM?
Athletic Heart Syndrome (AHS) vs. HOCM

• AHS
  – Reduced LV mass with short deconditioning periods
    • MRI best for assessment
  – LV end-diastolic dimension >55mm favors AHS over HOCM
Athletic Heart Syndrome (AHS) vs. HOCM

• HOCM
  – Abnormal Doppler derived LV diastolic filling or relaxation indices
  – Family member with HOCM
  – LV cavity <45 mm in diastole
  – MRI again may help measure hypertrophy or lack of
Hypertrophic Obstructive Cardiomyopathy

- B-MHC, MyBP-C, & cTn-T account for most HCM cases. There are many mutations at each locus making “rule out” genetic testing problematic.

Spirito P. NEJM 336(11):775, 1997
Hypertrophic Obstructive Cardiomyopathy

• Most patients with HCM are non-obstructive and asymptomatic. How do you find these and what do you do with them?

AHA/ACC Recommendations

• Participation in competitive athletics for asymptomatic, genotype-positive HCM patients without evidence of LV hypertrophy by 2-dimensional echocardiography and CMR is reasonable, particularly in the absence of a family history of HCM-related sudden death (Class IIa; Level of Evidence C).
Hypertrophic Obstructive Cardiomyopathy

• Death from VT / Vfib
• OR…Nothing…
• OR…
• Symptoms
  • palpitations
  • syncope
  • chest pain
  • DOE
Hypertrophic Obstructive Cardiomyopathy

- Yearly echo in those with family history of HOCM
- Disqualify from exertional sports?
- May participate in 1A sports such as golf, bowling
Fig. 1. Distribution of cardiovascular causes of sudden death in 1,435 young competitive athletes. ARVC, arrhythmogenic right ventricular cardiomyopathy; AS, aortic stenosis; CAD, coronary artery disease; C-M, cardiomyopathy; HCM, hypertrophic cardiomyopathy; HD, heart disease; LAD, left anterior descending; LVH, left ventricular hypertrophy. (Courtesy of the Minneapolis Heart Institute Registry, 1980–2005, Minneapolis, MN; with permission.)
Congenital Coronary Artery Anomalies

- Origin of LCA from right sinus of Valsalva - most common
- Single coronary artery
- Origin of coronary artery from PA
- Coronary artery hypoplasia
Congenital Coronary Artery Anomalies

• Aberrant coronary arteries compromise the second leading cause of SCD in young athletes.

• The highest risk pattern is LCA from the right Sinus of Valsalva followed by RCA from the left Sinus of Valsalva.

• The SCD event is always occurs with strenuous exercise or shortly thereafter.

• The presumed mechanism of SCD is dynamic compression of the coronary arteries during exercise leading to ischemia and lethal ventricular arrhythmia.
Congenital Coronary Artery Anomalies

- Proximal coronary artery origins as seen from the parasternal short axis view.
- Can be seen easily in most pediatric patients.

Congenital Coronary Artery Anomalies

- Highest risk pattern: LMCA from right Sinus of Valsalva

Congenital Coronary Artery Anomalies

- Second highest risk: RCA from left Sinus of Valsalva.

Congenital Coronary Artery Anomalies

• Epidemiology of Aberrant Coronaries
  One large prospective pediatric echo study
  Referral for “murmur” or “ventricular function.”
  4 cases of aberrant coronaries (0.17%) identified.
  1 case “missed” by echo.
  SCD with aberrant coronary at autopsy.
  Prevalence higher than in other reported screening studies. Referral bias?

Congenital Coronary Artery Anomalies

• The Pyramid problem: Aberrant coronaries are 2nd leading cause of SCD. The prevalence of aberrant coronaries in the general asymptomatic “healthy” population is unknown.

• The math doesn't work out: Estimated prevalence 0.1-0.2% of general population? 4,000,000 live US births annually. 4,000-8,000 affected children / yr. Observed risk of SCD (all causes) in sports 1 per 217,000 / yr. Either the prevalence of aberrant coronaries is much lower than currently reported OR the incidence of SCD in aberrant coronary arteries is quite low.
Congenital Coronary Artery Anomalies

• Symptoms
  – Death is most common
  – Rarely
    • exertional chest pain
    • syncope
    • infarction
Congenital Coronary Artery Anomalies

• Diagnosis
  – Arteriography
  – CT angiogram

• Corrective surgery if stable
Primary Electrical Disorders

- Wolff-Parkinson-White
- Long QT
- ARVD
- Brugada
- Rare disorders associated with development of malignant ventricular arrhythmias and associated sudden cardiac death.
Wolff-Parkinson-White

• Ventricular pre-excitation by an accessory bypass tract.
• Delta wave, short PR, +/- unusual T-waves on ECG. Bypass tract allows for conventional SVT.
• Bypass tract also allows for rapid conduction to ventricle with atrial fibrillation (pre-excited a fib).
Wolff-Parkinson-White

- Risk of SCD in WPW is very low (0.5% per decade).
- Risk of SVT and pre-excited a fib eliminated by successful radiofrequency ablation.
- Most patients with rapid bypass tract conduction have SVT symptoms but SCD can be the first presentation of WPW.
- Ablation of completely asymptomatic people with WPW is controversial.
- Ablation for asymptomatic WPW may be warranted in high-risk occupations (pilot, military, etc).
Long QT Syndrome

• Genetic disease involving cardiac ion channels (Na+, K+).
• Results in delayed repolarization seen on surface ECG as long QT interval.
• Risk of polymorphic VT / VF “Torsades” & SCD. Diagnosed by QTc > 460 msec on ECG along with other electrical and clinical parameters.
• Avoidance of QT prolonging drugs, exercise restriction, and Beta blockade are cornerstones of therapy.

Long QT Syndrome
Arrhythmogenic right ventricular cardiomyopathy (ARVC)

- Familial condition
- Myocyte death, replacement with fibrous or adipose tissue in right ventricle
  - Causes ventricular or supraventricular arrhythmias which can be fatal
- Most common cause of SCD in Veneto, Italy
  - <5% in USA
ARVD Diagnostic Criteria

Major Criteria
- Right ventricular dysfunction
  - Severe dilatation and reduction of RV EF with little or no LV impairment
  - Localized RV aneurysms
  - Severe segmental dilatation of the RV
Tissue characterization
- Fibrofatty replacement of myocardium on endomyocardial biopsy
Conduction abnormalities
- Epsilon waves in V1 - V3.
  - Localized prolongation (>110 ms) of QRS in V1 - V3
Family history
- Familial disease confirmed on autopsy or surgery

Minor Criteria
- Right ventricular dysfunction
  - Mild global RV dilatation and/or reduced ejection fraction with normal LV.
  - Mild segmental dilatation of the RV
  - Regional RV hypokinesis
Tissue characterization
Conduction abnormalities
- Inverted T waves in V2 and V3 in an individual over 12 years old, in the absence of a RBBB.
  - Late potentials on signal averaged EKG.
  - Ventricular tachycardia with a LBBB morphology
  - Frequent PVCs (> 1000 PVCs / 24 hours)
Family history
- Family history of sudden cardiac death before age 35
- Family history of ARVD

Corrado D, Heart. 2000 May;83(5):588-95.
Brugada Syndrome

• Rare genetic (AD) electrical myopathy
  Spike & dome ST segment in V1-V3
  Prevalence of Brugada ECG pattern 1-2 per 1,000
  Treatment: ICD

Marfan’s Syndrome

• Diagnosis (2 of 4 features):
  – Family history
  – Cardiovascular abnormality (aortic aneurysm, MVP, CHF symptoms)
  – Musculoskeletal abnormality (arm span>height, kyphoscoliosis, pectus cavus)
  – Ocular abnormality (ectopic lens, myopia)

• Usually die from aortic dissection and rupture
Coronary Artery Disease

• Consider exercise stress testing for:
  – male >45; female>55
  – diabetics, smoker, fam Hx CAD, Chol>250, HDL<30
  – anyone with exertional chest pain, syncope, or palpitations
Other causes

• Myocarditis
  – Viral illness followed by CHF symptoms
  – 50% Coxsackie B
  – Afebrile 24 hours prior to play

• Aortic stenosis - SD with or without exercise
Commotio Cordis

• Induction of VF by mechanical stimulus during “vulnerable phase” of repolarization. “R-on-T phenomenon”
  No genetic or electrical susceptibility needed.
  Requires critically timed impact directly over cardiac silhouette.
  Seen in small ball and high impact sports: baseball, hockey, lacrosse, karate.
  Preventable through use of RIF or “safety” balls and possibly rigid chest protectors.
  Rescue therapy: Early defibrillation (AEDs)
Commotio Cordis

• Induction of VF with low energy (30 mph) baseball impact 15-30 msec before t-wave peak in swine.

Screening Guidelines 1996

• Uniform Screening for all High School & College athletes
• Detailed PE & Fam Hx upon entry to sports and then q 2 years
• No testing required (ECG, echo, labs)

Screening Guidelines 2007

- Update to 1996 screening guidelines
- Addresses differences in recommendations between European Society of Cardiology and International Olympic committee guidelines.
- Emphasis on AHA 14 element screen
  - 12 element AHA screen
    - Personal history
    - Family History
    - Physical exam
- EKG and ECHO not routinely required!!!!
Does Screening Work?

- Study of 42,368 athletes in Italy.
  3 eras (1979-2004)
  Pre screening (< 82)
  Early screening (83-93)
  Late screening (93-04)

- 55 SCD in 42,368 athletes
  1.9 /100,000 person-years
  89% reduction in SCD with screening
  3,035 (7%) initially disqualified but ultimately cleared for sports
  879 (2%) of screened athletes disqualified

- Majority of cases due to ARVC rather than HOCM

JAMA 296:1593, 2006
Does Screening Work?

- 89% reduction in SCD after athlete screening started in 1982. No change in unscreened nonathletes.

JAMA 296:1593, 2006
Does Screening Work?

- European Society of Cardiology H&P screening elements
  Similar to US - AHA guidelines

- JAMA 296:1593, 2006
Does Screening Work?

• ESC EKG screening criteria. LVH criteria different (30mm).

JAMA 296:1593, 2006
Does Screening Work?

- 5,615 high school athletes screened by:
  - General H & P and BP measurement
  - Dynamic cardiac auscultation
  - **Resting ECG**

- Three year follow up period:
  - 582 / 5615 (10%) had 1 or >1 screening abnormalities
  - 115 (2%) Hx
  - 175 (3.2%) PE
  - 20 (0.3%) BP
  - 146 (2.6%) ECG
  - ECHO and exercise stress test performed if screen abnormal

Does Screening Work?

• No cases of HCM identified despite high prevalence in general population and high prevalence in SCD.
• ? Self selection bias ?
• 22 athletes disqualified after abnormal screen and follow up echo and/or exercise stress test
• 1 severe AI, 5 severe hypertension (ok)
• 1 SVT treated with ablation => “requalified”
• 15 “other arrhythmias” (6 WPW, 5 PVC’s, 4 RBBB)
• One sudden death during track practice
• Dx: aberrant right coronary artery (normal screen)

Pros of EKG screening

- Clearly shown to dramatically reduce SCD in the Italian population below that of the non athletic population (from 3.6-0.4/100,000 over 24 yrs)
- Remarkably sensitive in the Italian population for HCM as $\frac{51}{53}=96\%$ who subsequently died of HCM had positive ECGs.
- *Nevada data clearly indicate that ECGs are much better than PPE for detecting underlying cardiac disorders associated with SCD in the US
- Current US strategy has no data to support that it reduces SCD in athletes.
- ECG clearly increases detection of disorders like LQTS and ARVD that cause sudden death in the non athletic population as well.
- Once more identified, genetic/family clusters can be identified
- More money is spent on health initiatives that save fewer lives (ex. Meningococcal vaccine)
Cons to screening EKG

• Italian data not proven and potentially not applicable in US with marked greater heterogeneity of the population
• Estimated 250 to 2000/100,000 athletes would be held from competition for each life saved (estimates depend on SCD incidence figures) with no data on long term health or social effects on the disqualified athletes (DM, obesity, BP, depression, CAD)
• No gender or racial norms exist for ECG in the US at this point
• Assuming Italian data apply to US, 7% of those initially disqualified would return to play after echo, stress test, etc (which amounts to 500,000 of 7,300,000 athletes in 2006) demonstrate a normal workup
• Cost of >$2 B per year and no infrastructure like Italian exists in US
• Applying Italian data, cost per life/year saved is between $44,000 and $330,000
AHA/ACC Recommendation

• Mandatory and universal mass screening with 12-lead ECGs in large general populations of young healthy people 12 to 25 years of age (including on a national basis in the United States) to identify genetic/congenital and other cardiovascular abnormalities is not recommended for athletes and nonathletes alike. (Class III, no evidence of benefit; Level of Evidence C).
Sickle Cell Trait

• Usually benign
  – sudden death may occur in high heat or altitude
Sickle Cell Trait

- Sports participation should not be restricted but preventative measures should be taken
  - avoid dehydration
  - increase training intensity gradually
  - wear cool clothing
  - caution in high temp, humidity, or altitude
  - stop activity if cramps occur
Sickle Cell Trait

- Symptoms
  - legs weak and cramping
  - short of breath
  - collapse
  - LUQ abdominal pain from splenic infarct

- Exam
  - obtunded
  - hyperventilation
  - hypotensive
Conclusion

- Sudden cardiac death, although tragic, is very rare!
- HCM and aberrant coronary arteries are the cause of SCD in over 2/3 of cases.
- Electrical disorders, other cardiomyopathies, and aortic rupture (Marfan’s) compromise most of the remaining 1/3 of cases.
- There are well documented guidelines for pre participation screening of prospective athletes.
- Current guidelines emphasize PE & Fam Hx and do not require an ECG or ECHO evaluation.
Conclusion

- Addition of ECG screening would improve detection of HCM and allow detection of most electrical disorders but at increased cost.
- False positive ECG’s requiring echo confirmation
- False negative ECG’s in HCM (~10%)
- Benign nonspecific ECG changes requiring further evaluation / follow up (NL variants, “athlete’s heart”)
- No screening methodology other than detailed echocardiography (or CT / MRI) would allow for detection of coronary artery abnormalities.
Conclusion

• Pre Participation physical exam not just for cardiovascular screening

• Screening for sickle cell trait with appropriate education can prevent morbidity and mortality due to sickle crisis with strenuous exercise

• Screening and appropriate management of asthma can improve athletic performance and prevent morbidity from reactive airway disease
Thank You!!!!!

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