Cardiovascular Screening for Athletes

Fran Munkenbeck, MD
Oregon Cardiology
Feb 1, 2014

nothing to disclose
Sudden Cardiac Death

Exceeding 300,000 deaths annually-leading cause of death in the US.

Exercise-related Sudden Cardiac Death – 1 to 5/million athletes per year-underestimation.
Limitations of collection of data for cardiac death of athletes

- Did not account for sudden cardiac arrest survivors.
- Data base not accurate as reporting in the US is not mandatory.
Incidence of sudden cardiac death in NCAA athletes

The true incidence - not known

Until 2011 felt to be 1:23000 to 1:300000 per year in US athletes

Incidence better refined by University of Washington, Kimberly Harmon, MD – Circulation. 2011;123:1594-1600.

NCAA Memorial Resolutions, Parent Heart Watch, insurance claims
Incidence of SCD of NCAA athletes between 2004-2008

Overall there was 273 deaths.

Majority was non medical.

Leading medical cause of death was cardiac- 45 of the 273 or 16%.

Cardiac deaths accounted for 56% of medical deaths.

Of the 45 deaths- 34 deaths were in male athletes and 11 in female.
Risk of death

Higher in Division I athletes
1: 30,301/yr in Division I
1: 42,457 in Division II
1: 84,473 in Division III

Higher incidence in black athletes
1: 58,653 in white athletes
1: 17,696 in black athletes
Risk varied by sport

Basketball- highest risk sport

Overall annual death rate of 1:11,394 per year with Division I at 1:3126 per year

Greater than 3 fold risk of SCD in the black male basketball athlete over the white athlete in all divisions
Other high risk sports for SCD

Swimming 1:23,488
Lacrosse 1:23,357
Football 1:38,497
Cross-country 1:41,695
Division I football 1: 25,297 with risk varying by ethnicity
How do we accurately find the athletes at risk for sudden cardiac death?

- Without eliminating athletes who truly not at risk (false positives).
- With a reasonable cost to benefit ratio and not causing a tsunami of testing of not significant yield.
Table 1
The 12-element AHA recommendations for preparticipation cardiovascular screening of competitive athletes

**Medical history**

*Personal history*
1. Exertional chest pain or discomfort
2. Unexplained syncope or near-syncope
3. Excessive exertional and unexplained dyspnea or 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 due to heart disease, in one or more relatives
7. Disability from heart disease in a close relative less than 50 years old
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)

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*a* Parental verification is recommended for high school and middle school athletes.

*b* Judged not to be neurocardiogenic (vasovagal); of particular concern when related to exertion.

*c* Auscultation should be performed in both supine and standing positions (or with Valsalva maneuver), in particular to identify murmurs of dynamic left ventricular outflow tract obstruction.

*d* Preferably, taken initially in both arms.
**School Sports Pre-Participation Examination – Part 1: Student or Parent Completes**

**NAME:** ____________________________  **BIRTHDATE:** ____________________________

**ADDRESS:** ________________________________  **PHONE:** ________

**Athlete and Parent/Guardian:** Please review all questions and answer them to the best of your ability. Explain any YES answers on back.

**Medical Provider:** Please review with the athlete details of any positive answers.

<table>
<thead>
<tr>
<th>NO</th>
<th>YES</th>
<th>Don't Know</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Has anyone in the athlete’s family died suddenly before the age of 50 years?</td>
<td></td>
<td>✔</td>
</tr>
<tr>
<td>2. Has the athlete ever passed out during exercise or stopped exercising because of dizziness or chest pain?</td>
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<td>✔</td>
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<tr>
<td>3. Does the athlete have asthma (wheezing), hay fever, other allergies, or carry an EPI pen?</td>
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<tr>
<td>4. Is the athlete allergic to any medications or bee stings?</td>
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<td>5. Has the athlete ever broken a bone, had to wear a cast, or had an injury to any joint?</td>
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<tr>
<td>6. Has the athlete ever had a head injury or concussion?</td>
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<tr>
<td>7. Has the athlete ever had a hit or blow to the head that caused confusion, memory problems, or prolonged headache?</td>
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<td>8. Has the athlete ever suffered a heat-related illness (heat stroke)?</td>
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<td>9. Does the athlete have a chronic illness or see a physician regularly for any particular problem?</td>
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<tr>
<td>10. Does the athlete take any prescribed medicine, herbs or nutritional supplements?</td>
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<tr>
<td>11. Does the athlete have only one of any paired organ (eyes, kidneys, testicles, ovaries, etc.)?</td>
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<tr>
<td>12. Has the athlete ever had prior limitation from sports participation?</td>
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<tr>
<td>13. Has the athlete had any episodes of shortness of breath, palpitations, history of fainting, fever or swelling easily?</td>
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<tr>
<td>14. Has the athlete ever been diagnosed with a heart murmur or heart condition or hypertension?</td>
<td></td>
<td>✔</td>
</tr>
<tr>
<td>15. Is there a history of young people in the athlete’s family who have had congenital or other heart disease: cardiomyopathy, abnormal heart rhythms, long QT or Marfan’s syndrome? (You may write “I don’t understand these terms” and initial this item, if appropriate.)</td>
<td></td>
<td>✔</td>
</tr>
<tr>
<td>16. Has the athlete ever been hospitalized overnight or had surgery?</td>
<td></td>
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<tr>
<td>17. Does the athlete have weight regularly to meet the requirements for your sport?</td>
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<tr>
<td>18. Does the athlete have anything he or she wants to discuss with the physician?</td>
<td></td>
<td></td>
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<tr>
<td>19. Does the athlete cough, wheeze, or have trouble breathing during or after activity?</td>
<td></td>
<td></td>
</tr>
<tr>
<td>20. Are you unhappy with your weight?</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

**FEMALES ONLY**

<p>| | | |</p>
<table>
<thead>
<tr>
<th></th>
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</thead>
<tbody>
<tr>
<td>a. When was your first menstrual period?</td>
<td></td>
<td></td>
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<tr>
<td>b. When was your most recent menstrual period?</td>
<td></td>
<td></td>
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<tr>
<td>c. What was the largest time between menstrual periods in the last year?</td>
<td></td>
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</tbody>
</table>

**Parent/Guardian’s Statement:**

I have reviewed and answered the questions above to the best of my ability. I and my child understand and accept that there are risks of serious injury and death in any sport, including the one(s) in which my child has chosen to participate. I hereby give permission for my child to participate in sports activities.

I hereby authorize emergency medical treatment and/or transportation to a medical facility for any injury or illness deemed urgently necessary by a registered athletic trainer, coach, or medical practitioner.

I understand that this sports pre-participation physical examination is not designed nor intended to substitute for any recommended regular comprehensive health assessment.

I hereby authorize release of these examination results to my child’s school.

Signed: ____________________________  Date: ____________________________

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**OSHA 318-979, Section 1 (D):** "A school district shall require students who continue to participate in interscholastic sports in grades 7 through 12 to have a physical examination whenever two years have passed since the last examination conducted as required by subsection (B) of this section. A physical examination required by this subsection shall be conducted by an unrestricted licensee in medicine, a licensed practical nurse, a licensed physician assistant, an advanced nurse practitioner, or a pod licensed osteopathic physician who has clinical training and experience in detecting cardiopulmonary diseases and disorders."

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**Oregon School Activities Association**

Forms – Physical Examination 2020  Revised: 03/19

2013 2014 Handbook

- 135 -
### SUGGESTED EXAM PROTOCOL FOR THE PHYSICIAN

Revised May 2010

**MUSCULOSKELETAL**

Have patient:

1. Stand facing examiner
2. Look at ceiling, floor, over shoulders, touch ears to shoulders
3. Shrug shoulders (against resistance)
4. Abduct shoulders 90 degrees, hold against resistance
5. Externally rotate arms fully
6. Flex and extend elbows
7. Arms at sides, elbows 90 degrees flexed, pronate/supinate wrists
8. Spread fingers, make fist
9. Contract quadriceps, relax quadriceps
10. “Duck walk” 4 steps away from examiner
11. Sit with back to examiner
12. Knees straight, touch toes
13. Rise up on heels, then toes

To check for:

- AC joint, general laxity
- Cervical spine motion
- Trapezius strength
- Deltoid strength
- Shoulder motion
- Elbow motion
- Elbow and wrist motion
- Hand and finger motion, deformities
- Symmetry and hand/wrist girth
- Hip, knee and ankle motion
- Shoulder symmetry, scapulas
- Scoliosis, hip motion, herniations
- Calf symmetry, leg strength

**MURMUR EVALUATION** – Auscultation should be performed sitting, supine and squatting in a quiet room using the diaphragm and bell of a stethoscope.

- Auscultation findings:
  1. SS heard usually, hot holosystolic, soft, low-pitched
  2. Normal SS
  3. No murmur or mid-systolic click
  4. Continuous diastolic murmur absent
  5. No-elevated diastolic murmur
  6. Normal femoral pulses

- Rules out:
  - VSD and mitral regurgitation
  - Tetralogy, ASD and pulmonary hypertension
  - Aortic stenosis and pulmonary stenosis
  - Patent ductus arteriosis
  - Aortic insufficiency
  - Congenital

**MARFAN’S SCREEN** – Screen all men over 60° and all women over 5°10” in height with echocardiogram and slit lamp exam where any two of the following are found:

1. Family history of Marfan’s syndrome (this finding alone should prompt further investigation)
2. Cataract, murmur or mid-systolic click
3. Kyphoscoliosis
4. Anomalous thoracic deformity
5. Arm span greater than height
6. Upper to lower body ratio more than 1 standard deviation below mean
7. Myopia
8. Eccentric lens

**CONCLUSION** – When can an athlete return to play after a concussion?

After suffering a concussion, an athlete should return to play or practice on the same day. Previously, athletes were allowed to return to play if their symptoms resolved within 15 minutes of the injury. Studies have shown that the young brain does not recover that quickly, thus the Oregon legislature has established a rule that no player shall return to play following a concussion on that same day until the athlete must be cleared by an appropriate health care professional before they are allowed to return to play or practice.

Once an athlete is cleared to return to play they should proceed with activity in a derivative fashion to allow their brain to readjust to exertion. The athlete may complete a new step each day. The return to play schedule should proceed as below following medical clearance:

**Step 1:** Light exercise, including walking or riding an exercise bike. No weightlifting.

**Step 2:** Running in gym or on field. No helmet or other equipment.

**Step 3:** Non-contact training drills in full equipment. Weight training can begin. Step 4: Full contact practice or training.

**Step 5:** Game play.

If symptoms occur at any step, the athlete should cease activity and be re-evaluated by a health care provider.

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**SR1-021-0041 Form and Protocol for Sports Physical Examinations**

The State Board of Education adopts by reference the form entitled “School Sports Pre-participation Examination March 2010” that must be used to document the physical examination and test the protocol for conducting the physical examination. Medical providers conducting physicals on or after June 30, 2010 must use this form dated May 2010.

**NOTE:** The form can be found on the Oregon School Activities Association (OSAA) Website www.osaa.org.
Adaptation of a heart to certain sports

**TABLE 1. Classification of sports: based on peak dynamic and static components during competition**

<table>
<thead>
<tr>
<th>A. Low dynamic</th>
<th>B. Moderate dynamic</th>
<th>C. High dynamic</th>
</tr>
</thead>
<tbody>
<tr>
<td>Low static</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Billiards</td>
<td>Baseball</td>
<td>Badminton</td>
</tr>
<tr>
<td>Bowling</td>
<td>Softball</td>
<td>Cross-country skiing (classic technique)</td>
</tr>
<tr>
<td>Cricket</td>
<td>Table tennis</td>
<td>Field hockey</td>
</tr>
<tr>
<td>Curling</td>
<td>Tennis (doubles)</td>
<td>Orienteering</td>
</tr>
<tr>
<td>Golf</td>
<td>Volleyball</td>
<td>Race walking</td>
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<tr>
<td>Riffery</td>
<td></td>
<td>Racquetball</td>
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<tr>
<td></td>
<td></td>
<td>Running (long-distance)</td>
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<td></td>
<td></td>
<td>Soccer*</td>
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<tr>
<td></td>
<td></td>
<td>Squash</td>
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<tr>
<td></td>
<td></td>
<td>Tennis (singles)</td>
</tr>
<tr>
<td>Moderate static</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Archery</td>
<td>Fencing</td>
<td>Basketball*</td>
</tr>
<tr>
<td>Auto racing*†</td>
<td>Field events (jumping)</td>
<td>Ice hockey*</td>
</tr>
<tr>
<td>Diving*†</td>
<td>Figure skating*</td>
<td>Cross-country skiing (skating technique)</td>
</tr>
<tr>
<td>Equestrian†</td>
<td>Football (American)</td>
<td>Football (Australian rules)*</td>
</tr>
<tr>
<td>Motorcycling*†</td>
<td>Rodeoing†</td>
<td>Lacrosse*</td>
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<tr>
<td></td>
<td>Rugby*</td>
<td>Running (middle-distance)</td>
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<tr>
<td></td>
<td>Running (sprint)</td>
<td>Swimming</td>
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<tr>
<td></td>
<td>Surfing†</td>
<td>Team handball</td>
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<tr>
<td></td>
<td>Synchronized swimming†</td>
<td></td>
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<tr>
<td>High static</td>
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<tr>
<td>Bobsledding*†</td>
<td>Body building*†</td>
<td>Boxing*</td>
</tr>
<tr>
<td>Field events (throwing)</td>
<td>Downhill skiing*†</td>
<td>Canoeing/kayaking</td>
</tr>
<tr>
<td>Gymnastics*†</td>
<td>Wrestling*</td>
<td>Cycling*†</td>
</tr>
<tr>
<td>Karate/Judo*</td>
<td></td>
<td>Decathlon</td>
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<tr>
<td>Luge*†</td>
<td></td>
<td>Rowing</td>
</tr>
<tr>
<td>Sailing</td>
<td></td>
<td>Speed skating</td>
</tr>
<tr>
<td>Rock climbing*†</td>
<td></td>
<td></td>
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<tr>
<td>Water skiing*†</td>
<td></td>
<td></td>
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<tr>
<td>Weight lifting*†</td>
<td></td>
<td></td>
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<tr>
<td>Wind surfing*†</td>
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</tr>
</tbody>
</table>

*Danger of bodily collision.
†Increased risk if syncope occurs.
From ref. 4, with permission.
Dynamic(aerobic) exercise

The active muscles are rhythmically contracting during aerobic exercise. Adequate blood supply must be delivered to active muscle while blood flow is maintained to the brain, heart, lungs, other vital organs. Accomplished by increasing cardiac output.
Physiology of aerobic exercise

Cardiac output (CO) = heart rate x stroke volume (SV)

- Normally CO is 5L/min at rest
- 20 to 25 L/min in active boys + men
- 40 L/min in endurance-trained athletes
- Maximal heart rate can not be increased further with endurance training.

- SV 70-80 mL/beat at rest to 130-140 mL/beat in endurance trained athletes.
- At peak exercise 200 to 220 mL/beat in endurance-trained athletes.
The hemodynamics

With exercise systolic bp increases + diastolic bp decreases.

120/80 at rest to 250/70 w/exercise.

Dilation of blood vessels in active muscles causes the diastolic blood pressure to fall.

The increase of the stroke volume (increased ejection of the blood w/each beat) causes the systolic blood pressure to rise.
Endurance (aerobic) exercise

- Vessels dilate to increase compliance.
- To increase the stroke volume there is a compensatory increase in muscle mass of the main pumping chamber of the heart.
- Because of increased stroke volume the heart accommodates by increasing the internal diameter of the LV with slight increase in thickness of the LV walls.
- Muscle mass can increase by 23-80% w/average increase of 45-50%.
Static(nonaerobic) exercise

- Muscles do not rhythmically contract.
- Large increase in intra-muscular pressure which is compensated by large increase in blood pressure (up to 480/350 has been reported in body builders)
- Cardiac output is increased by heart rate mostly.
- To compensate the main chamber of the heart increases in wall thickness to work against the highly generated blood pressures.
The result

Normal

Eccentric hypertrophy

Concentric hypertrophy
In testing of the athlete there is a gray zone of:

Physiologic response of exercise vs. true pathology
Hypertrophic cardiomyopathy

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 LV outflow tract. FW, left ventricular free wall; Ao, aorta; LA, left atrium; RV, right ventricle; (B) Septal myocardium shows greatly disorganized architecture with adjacent hypertrophied cardiac muscle cells arranged perpendicularly and obliquely; (C) Intramural coronary artery with thickened wall, due primarily to medial hypertrophy, and narrowed lumen; (D) Demarcated area of replacement fibrosis in septum. (From Maron BJ. Hypertrophic cardiomyopathy. Lancet 1997;350:127–33; with permission.)
Gray area of overlap between athlete's heart and cardiomyopathies, including myocarditis, hypertrophic cardiomyopathy (HCM), and arrhythmogenic right ventricular cardiomyopathy (ARVC)

Arrhythmogemic right ventricular dysplasia

Figure 4. Gross and Histopathological Cardiac Findings in a 24-Year-Old Athlete Who Died Suddenly of Arrhythmogenic Right Ventricular Cardiomyopathy.

The electrocardiogram of this athlete is shown in Figure 1A. Panel A shows a cross section of the heart with a dilated right ventricular chamber, hypertrophied subendocardial trabeculae, and a diffusely thinned (1.5 mm in thickness) anterolateral wall (arrowheads), in the absence of aneurysm formation. Panel B shows a panoramic histologic section of the right ventricular outflow tract, including the pulmonary valve (arrow). Abnormalities are not evident in the myocardium (red staining) at this magnification (Heidenhain’s trichrome stain). Panel C represents the boxed area of Panel B at higher magnification, showing areas of fibrofatty replacement of atrophic myocardium. The asterisk indicates epicardial fat, which is regarded as normal. Panel D shows the boxed area of Panel C at higher magnification. Surviving myocytes (red staining) are embedded within fibrous tissue (blue staining) and fat (white staining), an acknowledged feature of arrhythmogenic right ventricular cardiomyopathy.
Cardiovascular causes of sudden cardiac death

- Hypertrophic cardiomyopathy - 36%
- Coronary artery anomalies - 17%
- Arrhythmogenic right ventricular dysplasia - 4%
- Aortic valve stenosis - 3%
- Electrical problems (channelopathies) such as long QT syndrome - 3%
- Aortic rupture such as in Marfan’s syndrome - 2%
Gray zone

Can represent up to 2% of elite athletes.

It may be hard to distinguish between mild expression of a heart disorder and extreme manifestation of physiologic hypertrophy.
Chart showing criteria used to favor or distinguish hypertrophic cardiomyopathy (HCM) from athlete's heart when maximal left ventricular (LV) wall thickness is within shaded gray zone of overlap (i.e., 13 to 15 mm), consistent with both diagnoses.

The 5 minute screening cardiac echo for athletes

Test focuses on looking for abnormalities seen in the list of structural cardiac abnormalities that can cause sudden death.

University of Wisconsin, American Society of Echocardiography
Practicality

Requires special equipment and training, less portable, expense

Quality concerns


395 athletes studied- no athlete found to have HCM.

32.9% trivial or mild regurgitation

0.5% bicuspid aortic valve
Can we get more sensitive at less cost?

Italy mandated in 1982 pre-participation EKG—w/ reported reduction of SCD from 3.6 events/100,000 person yrs. to 0.4 events/100,000 person yrs.

Israel mandated in 1997 pre-participation EKG—2.54 events/100,000 person yrs. to 2.66 events/100,000 person years
Addition of EKG screening
PAC10

Two schools require EKG prior to competitive sports participation
Stanford
University of Washington

Clinical Journal of Sport Medicine, vol 20, number 2, March 2010
Of 658 EKG’s

- 68% had normal EKG’s in females
- 38% had normal EKG’s in men
- The rest had either minor or major abnormalities.

- Sixty-five athletes (10%) had distinctly abnormal EKG’s to suggest pathology and needed further testing. 24 agreed to further testing and 21 of 24 to date of the publishing had normal f/up studies.
Relation of Abnormal and Normal ECG Patterns to Race in 1,959 Professional Football Players

Magalski, A. et al. J Am Coll Cardiol 2008;51:2250-2255
Specific ECG Abnormalities With Respect to Race

Magalski, A. et al. J Am Coll Cardiol 2008;51:2250-2255
Abnormal ECG With Respect to Race and Player Position

Magalski, A. et al. J Am Coll Cardiol 2008;51:2250-2255
Outcomes in Athletes w/marked EKG abnormalities

81 out of 12,550 trained athletes

- a. very abnormal T waves
- b. no apparent cardiac disease
- c. serial EKG’s and echoes for 1 to 27 years

5 eventually were found to have very abnormal hearts- for 3 it was not detected until 12 to 17 years after first EKG!!

NEJM 2008;358:152-61
ECG screening

Not always positive or negative

Changes can be seen as part of cardiac adaptation in athletes- false positives.

Abnormalities can vary by ethnic groups.

In original guidelines- 40% positive!
New guidelines

Uberoi et al, Circulation 2011;124:746-757

Analyzed ECG’s from 658 Stanford athletes

62% of men + 32% of women had abnml ECG by prior criteria.

10% had abnormalities warranting further testing by prior criteria.

With re-interpretation 4% remained in abnormal range + felt to need further work up.
Figure 1. Three 12-Lead Electrocardiograms (ECGs), Obtained in Trained Athletes at Initial Evaluation, Showing Evidence of Marked Repolarization Abnormalities.

The ECG from a 24-year-old national-level canoeist shows diffuse T-wave inversion (Panel A). The athlete died suddenly during exercise 12 months after evaluation, of undetected arrhythmogenic right ventricular cardiomyopathy. Panel B shows the ECG from a 26-year-old regional-level soccer player without left ventricular hypertrophy on echocardiography at the initial evaluation. The ECG shows markedly increased R- and S-wave voltage in the precordial leads (V₂ to V₅) in association with deep T-wave inversion in standard (I, II, III, aVF) and precordial (V₂ to V₅) leads. The athlete showed phenotypic evidence of nonobstructive hypertrophic cardiomyopathy 7 years later. Panel C shows the ECG from a 29-year-old national-level soccer player without evidence of left ventricular hypertrophy or other structural cardiac abnormalities on echocardiography. The ECG shows marked repolarization abnormalities, including ST-segment depression and T-wave inversion in the lateral precordial and standard inferior leads.
**Pediatric ECG Analysis**

- Normal sinus rhythm
- LVH
- Early repolarization
- Nonspecific T wave abnormality

Referred by: B. BERG M.D.

Confirmed by: [Signature]

Date: 14-AUG-2008 12:06:59

[ECG waveforms and measurements]
Defibrillation: 53 BPM
PR interval: 188 ms
QRS duration: 88 ms
QT/QTc: 456/427 ms
P-R-T axes: 8 81 -56

DRG DATE: NOW
AGE:

Referred by: PHYSIC MISCELLANEOUS

Confirmed by

29-MAY-2012 13:41:40

Sinus bradycardia
ST elevation, consider lateral injury or acute infarct
"**" ACUTE MI "**"
Abnormal ECG
No previous ECGs available
Male, Caucasian

69 yrs, 211ib

Room: Loc: 130

12-Aug-2013 11:42:10

PEACEHEALTH SACRED HEART MED CTR-CARD ROUTINE RECORD

Vent. rate
PR interval
QRS duration
QT/QTc
P-R-T axes

59 BPM
154 ms
102 ms
438/433 ms
36 86 -60

Sinus bradycardia
ST & T wave inversion inferolaterally - c/w apical hypertrophic cardiomyopathy
Abnormal ECG
When compared with ECG of 01-FEB-2013 11:48,
T wave inversion now evident in V3 and V4
T waves are deeper laterally now
Confirmed by MUNKENBECK MD, FRANCES (148351) on 8/12/2013 6:08:03 PM

Technician, TERESA ORCUTT
Text ind.

Referred by: FRANCES MUNKENBECK MD
Confirmed By: FRANCES MUNKENBECK MD

25mm/s 10mm/mV 150Hz 7 1.1 12SL 237 CID: 7

SID: E393778 EID: 14835 EDT: 18:08 12-Aug-2013 ORDER:

Page 1 of 1
Consensus on adding EKG

Jonathon Drezner MD, Vicki Vetter MD, Benjamin Levine MD

Evidence - not support mandatory EKG screening by the legal system.

Current evidence-reasonable to include. Risk of 7-8% false positive. Need people who are well versed in reading athlete EKG’s.

Reasonable not to include an EKG-accepting missing some athletes w cv disease
Commotio cordis

Blow to the chest w/hockey pucks, lacrosse balls or karate chop

Results in a chaotic heart rhythm (ventricular fibrillation)

Young athletes with thin chests are prone to modest blows (30 to 40 mph)

Older athletes prone to stronger blows (up to 90 mph)
Stop-frame images of an aborted commotio cordis event during a televised professional hockey game.
Commotio cordis

- Only approx. 15% survive
- Timely and effective CPR
- AED use
- Innovations in sports equipment design
Do you know the new CPR?

- Recognition is important!
- Agonal breathing does not count
- Chest compression only CPR during the ‘electrical phase’ (1st few minutes) until paramedics come
- 100 chest compressions/minute
- *Bee Gees “Stayin’ Alive”*
- Become familiar with an AED
What to do?

- AED availability, training and use
- New CPR
- Campaigns on no texting while driving.
- No drinking while driving.
- Assess for potential for suicide
GO DUCKS !
12 point cardiovascular screen

Table 1: The 12-element AHA recommendations for preparticipation cardiovascular screening of competitive athletes

<table>
<thead>
<tr>
<th>Medical history</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Personal history</strong></td>
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<tr>
<td>1. Exertional chest pain or discomfort</td>
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<tr>
<td>2. Unexplained syncope or near-syncope^b</td>
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<tr>
<td>3. Excessive exertional and unexplained dyspnea or fatigue, associated with exercise</td>
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<tr>
<td>4. Prior recognition of a heart murmur</td>
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<tr>
<td>5. Elevated systemic blood pressure</td>
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<tr>
<td><strong>Family history</strong></td>
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<tr>
<td>6. Premature death (sudden and unexpected, or otherwise) before age 50 due to heart disease, in one or more relatives</td>
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<tr>
<td>7. Disability from heart disease in a close relative less than 50 years old</td>
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<tr>
<td>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</td>
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<tr>
<td><strong>Physical examination</strong></td>
</tr>
<tr>
<td>9. Heart murmur^c</td>
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<tr>
<td>10. Femoral pulses to exclude aortic coarctation</td>
</tr>
<tr>
<td>11. Physical stigmata of Marfan syndrome</td>
</tr>
<tr>
<td>12. Brachial artery blood pressure (sitting position)^d</td>
</tr>
</tbody>
</table>

^a Parental verification is recommended for high school and middle school athletes.
^b Judged not to be neurocardiogenic (vasovagal); of particular concern when related to exertion.
^c Auscultation should be performed in both supine and standing positions (or with Valsalva maneuver), in particular to identify murmurs of dynamic left ventricular outflow tract obstruction.
^d Preferably, taken initially in both arms.
Sudden Cardiac Death

>35 years – atherosclerotic disease

<35 years – hereditary disorders
Why are athletes at higher risk

Arrhythmogenic right ventricular dysplasia – desmosomes that hold the cells together are not made of the right glue. The more you use the heart the looser it gets. The looser then the more irritable the heart gets.

Anomalous take off of coronary arteries- exercise compresses the coronary arteries + decreases blood to heart which causes arrhythmias.

Hypertrophic cardiomyopathy- catelcholamine surge stirs up arrhythmias/blood supply is relatively less as the there is more muscle requiring blood.

Long QT type 2- arrhythmia comes out in cold water
36 had death by exertion

- 27 of the 36 were related to cardiac causes
- 10 of the 36 involved heat stroke
- 5 of the 10 heat stroke deaths were associated with the sickle cell trait