Cardiac Conditions in Athletes

The Sports Medicine Core Curriculum Lecture Series
Sponsored by an ACEP Section Grant
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1) ‘He had a seizure’

Jiri Fischer- Detroit RedWings
Hank Gathers- Loyola Marymount
(See video of Hank Gathers arrest on YouTube. An AED was present but not used.)

Myoclonic activity occurs with cardiac arrest
Place AED and let the defibrillator decide
Be wary of “seizures” presenting to the Emergency Department
Sudden Death in Athletes

< 30 y.o. congenital abnormalities
> 30 y.o. atherosclerosis

>80% cases due to congenital or inherited cardiac disease versus
~80% general population due to CAD


**Figure 2.** Incidence and relative risk of sudden death for specific cardiovascular causes among athletes and nonathletes.
Causes of Sudden Death in Young Athletes

- Hypertrophic Cardiomyopathy 26.4%
- Commotio Cordis 19.9%
- Coronary Artery Anomalies 13.7%
- Left Ventricular Hypertrophy of Indeterminate Cause 7.5%
- Myocarditis 5.2%
- Ruptured Aortic Aneurysm (Marfan’s) 3.1%
- Arrhythmogenic Right Ventricular Cardiomyopathy 2.8%
- Tunneled Coronary Artery 2.8%
- Aortic Valve Stenosis 2.6%
- Atherosclerotic Coronary Artery Disease 2.6%
- Dilated Cardiomyopathy 2.3%
- Asthma 2.1%
- Heat Stroke 1.6%
- Drug Abuse 1.0%
- Long QT Syndrome 0.8%
- Ruptured Cerebral Artery 0.8%
Nontraumatic Sports Death

10 year review of nontraumatic sports death

Reported 126 high school deaths (115 males, 11 females
Reported 34 collegiate deaths (31 males, 3 females)
Cardiovascular etiology more common

Most common cause cardiovascular death:
IHSS and anomalous coronary arteries
Cardiac Arrest in Athletes

1971 Chuck Hughes (NFL) SCD
1990 Hank Gathers (NCAA) HCM/SCD
1998 Chris Pronger (NHL) commotio cordis
2004 Sergei Zholtok (NHL) SCD
2005 Jaxon Logan (NCAA) commotio cordis
2005 Thomas Herrion (NFL) SCD/CAD
2005 Jiri Fischer (NHL) arrhythmia – SCA
  saved with AED, CPR
2009 Russian hockey league SCD
Syncope- Cardiac Diagnostics

**History and physical:** identifies 49-85% of all causes

**EKG:** 2-11%: long QT, heart block, BBB, WPW, V-tach, etc

**Exercise treadmill testing:**
*reproduce the demands of the sport to provoke arrhythmia

**Holter monitor:**
12+ hrs, diagnostic or exclusive 21%
(4% arrhythmia, 17% symptoms with no abnormality)
>24 hrs: 15% abn 1st 24 hr, 11% 2nd 24 hr, 4% 3rd 24 hr period
Syncope - Cardiac Diagnostics

Patient activated loop monitors -

**Echocardiogram** - detect structural abnormality: AS, IHSS

**Electro Physiology Study** - useful in those w/ abnormal EKG or structural abnormality (valvular, CM, BBB, WPW)

**Stress/cath** - detect CAD/anomalous vessels

**Upright tilt table testing** - neurocardiogenic syncope
Preparticipation Screening and Diagnosis of Cardiovascular Disease in Athletes

Medical History
- Exertional chest pain
- Unexplained syncope
- Unexplained exertional dyspnea
- Heart murmur
- Elevated blood pressure

Physical Examination
- Heart murmur
- Femoral pulses asymmetric
- Marfan’s features
- Brachial artery blood pressure

Family History
- Premature sudden death
- Heart disease < age 50
- Known specific cardiac conditions

American Heart Association: Preparticipation Screening

High school athlete deaths per year ≈ 1:200,000

State high school association preparticipation screening forms ask an average of 9.7 / 12 AHA-recommended items

18 states allow chiropractors and naturopathic practitioners to perform screening

NCAA has mandated preparticipation evaluation of all collegiate athletes in Divisions I, II and III

NBA has recently (2006) mandated standardized screening with echo and ECG for all players annually

Maron et al.
Top Myths in Sports Medicine

Preparticipation history and physical exam are more sensitive than testing (EKG and echo) in detecting cardiac disease in athletes
Preparticipation Exam—
Utility of Screening Tests to Detect Cardiac Disease


5,615 high school athletes, adding EKG to H/P. +/- echo
22 athletes (1/255)

History detected 0, auscultation 1, vital signs (BP) 5, EKG 16


H/P, EKG, 2D echo performed on 95 high school athletes
10 abnormalities detected with studies (2 EKG, 8 echo)
Only 1 detected by exam
Abnormality with (13%) vs. without (9%) symptoms

EKG patterns compared with echocardiography in 1005 “elite” athletes, 38 sports
Abnormal EKGs in 40%
Subgroup 15% highly suggestive of CM w/o pathology on echo
Structural cardiac disease in 5%
European Society of Cardiology: Preparticipation Screening ‘Positive EKG’

P wave
- left atrial enlargement: negative portion of P wave V1
- right atrial enlargement: peaked P wave II/III/V1

QRS Complex
- frontal plane axis deviation R>+120, L -30 to -90
- increased voltage: amplitude of R or S wave >2mV in standard lead; S wave >3mV in V1/V2,
- R wave > 3mV in V5/V6
- abnormal Q waves >25% height of R wave or QS
- RBBB or LBBB with QRS > 0.12s

ST segment, T waves, and QT interval
- ST depression or T wave inversion 2+ leads
- prolongation of heart rate corrected QT interval >0.44 s

Rhythm and conduction abnormalities
- PVC or ventricular arrhythmias
- SVT, Atrial flutter, Atrial Fibrillation
- Short PR interval <0.12 s with or without delta wave
- Sinus bradycardia with resting HR <40
- First, second or third degree heart block

“Normal” EKG findings - Physiologic Changes in Athletes

Sinus bradycardia
AV block w/ pause < 4s
Transient 2/3 AV block
Exercise-reversible ST elevation
Exercise-reversible changes in T waves
Right and/or left ventricular hypertrophy

“Athlete’s Heart”

Physiologic adaptations to exercise:

- **Dynamic training**: increase HR+SV -> dilation+mass
  - eccentric hypertrophy-volume overload

- **Static training**: large increase arterial BP -> increase wall thickness
  - concentric hypertrophy-pressure overload
Morphology of “Athlete’s Heart”

Study of 947 athletes @ national or international level
27 different sports
Performed echocardiography
increased LV diastolic cavity size (54 mm) in 38%
increased LV wall thickness >12mm in 1.7%
endurance sports had largest diastolic cavity size and wall thickness
isometric (weight lifting) show increased wall thickness: cavity ratios
*differences with sex, sport, in/out of season

Morphology Differs w/ Sport

156 asymptomatic NFL athletes

The mean maximal wall thickness (11.2 +/- 0.2 mm) was increased over controls

23% had evidence of LV hypertrophy

The mean resting EF was 58%

Seasonal Adaptations to Exercise

15 female collegiate basketball athletes

...echo in fall, winter, spring, next fall

LVEDV, SV, LV mass, septal thickness, LV posterior wall thickness, and aortic root diameter were significantly larger (12-70%) in the athletes vs. controls

From fall to spring measurement periods: LVEDV, SV, IVS, and LVM-index increased significantly (7-18%) in the athletes

From spring to next fall: IVS, LVPW, and LVM decreased significantly (5-30%) in the athletes

“Athlete’s Heart”

Physiologic adaptations on conditioning may mimic phenotype of IHSS and ARVC

determinants of morphology: sport, gender, genetic factors

physiologic cardiac remodeling leads to EKG findings:
  increase in precordial R-wave or S-wave voltages,
  ST segment or T-wave changes, and
  deep Q waves suggestive of left ventricular hypertrophy

HCM - Epidemiology

Most common cause of sudden cardiac death < 35 yo

Prevalence of phenotype 1:500 (0.2%)
Approximately 50% of cases are familial

Annual mortality rate in overall HCM population is 1% per year, however this is higher (4-6% per year) in childhood and adolescence
HCM - Genetics

Autosomal dominant, 12 genes identified (11 encoding sarcomeric proteins)

Over 400 specific mutations in these genes

Most commonly affected proteins:
beta-myosin heavy chain and myosin-binding protein C

Others less commonly affected: troponin T and I, alpha-tropomyosin, regulatory and essential myosin light chains, titin, alpha-actin, alpha-myosin heavy chain and muscle LIM protein (MLP)
HCM – Clinical Features:
ECG Changes

Abnormal in 90-95% of patients
No particular ECG pattern is characteristic

LV hypertrophy
T-wave inversion in the lateral precordial leads
Left atrial enlargement
Deep and narrow Q-waves
Diminished R-waves in the lateral precordial leads
**HCM – Familial Screening**

History, physical exam, ECG and 2D echocardiography

DNA analysis

Repeat evaluation at 12- to 18-month intervals beginning at age 12

If there is no evidence of LV hypertrophy by age 18 to 21 years → conclude that an HCM-causing mutation is absent

Recommend to continue surveillance into adulthood at 5-year intervals
HCM - Diagnosis

Echocardiography →
otherwise unexplained and usually asymmetric hypertrophy associated with a non-dilated left ventricle

Maximal LV end-diastolic wall thickness:
> 15 mm absolute dimension
Arrhythmogenic Right Ventricular Dysplasia


12/60 sudden deaths ARVD on autopsy

Fibrolipomatous transformation of the right ventricular free wall

*Furlanello et al. Pacing Clin Electrophysiol, Jan 1998, 21(1):331-5. 20 years*

1642 competitive athletes, 101 (6%) met criteria for ARVC

Prevalence of ARVC in Italian athletes with cardiac arrest=23%, sudden death=25%
Anomalous Coronary Arteries

Sudden deaths in Italy and US
27 with improper origin of LMCA, RCA off aortic sinus-died during/immediately after exertion (age: 16 +/- 7)
15 asymptomatic, 12 symptomatic
   4 syncopal event in preceding 2 years
   5 chest pain event in preceding 2 years

All prior cardiovascular tests were within normal limits!

*EKG, echo, stress test*

Commotio Cordis

Blunt chest wall impact, low energy

Ventricular fibrillation

NCAA ~ 10-20 cases per year in young males


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2 million basic training U.S. Armed Forces 1977-1981
Deaths classified from autopsy and clinical records
Prevalence rates Hgb AS: 8 % black and 0.08 % other

**Relative risk** Black recruits w/ Hgb AS:

- 32.2 for sudden unexplained deaths,
- 2.7 for sudden explained deaths, and
- 0 for non-sudden deaths

**Exertional Rhabdomyolysis = 200X**

Mortality 8:1 sickle trait:non, recruits 18-28 y.o.
Sickle Cell Trait and Exercise

Exercise to exhaustion at sea level regularly induces reversible sickling (1%).

Altitude hypoxia increases the extent of sickling with sickle trait 2% at 4,050 ft. to 8.5% at 13,123 ft.

29/49 reported cases are Caucasian

Microscopic infarction of the renal medulla loss of maximal urine concentrating ability predispose to heat illness and induce hematuria.
Sickle Cell Trait-Related Collapse

Over 80 cases in last 30 yrs, usually all-out exertion related to conditioning drills

2/3 fatal

Deaths from arrhythmia in first hour (hyperkalemia)

Renal failure next day due to rhabdomyolysis
Troponin T/I subclinical cardiac injury in endurance athletes

~9-13% of participants: 2-7X elevation

Risk of death < 24 hours after race: 1/50,000

> 3 hours race duration risk = smoking, sedentary, beer drinker

Troponin level correlates with race time and training volume

Decreased ejection fraction

Abnormal wall motion


![Graph showing increases in cardiac troponins and prothrombotic markers](image-url)

**Figure 1**

- Cardiac troponin after "classic" acute MI
- CK-MB after acute MI
- Cardiac troponin after "microinfarction"/extreme exercise (untrained individuals)
- Cardiac troponin after extreme exercise (highly trained individuals)

Days after Onset of Acute MI/Exercise

Multiples of the Upper Reference Limit

Upper reference limit
Top Myths in Sports Medicine

Prolonged endurance exercise is equivalent to a ‘physiologic stress test’…if he/she has no chest pain with exertion cardiac disease is unlikely…

…this does not account for sudden plaque rupture and occlusion
Case: 55 yo M Chest Pain at rest 1 hr PTA <3 weeks after Triathalon; 6 d after 64 K bike ride

- (55 yr)
- Male
- Lec: 06

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- Marked sinus bradycardia
- Left axis deviation
- Minimal voltage criteria for LVH, may be normal variant
- Abnormal ECG
Case: 55 yo M Chest Pain at rest 1 hr PTA <3 weeks after Triathlon; 6 d after 64 K bike ride

Labs #1:

Troponin = 0.51
CPK = 157
CKMB = 2.2

Cardiac cath shows:

70% D2 ostial lesion
90% mid LAD stenosis w/

plaque rupture and dissection
Exercise-Induced MI
Relative risk of MI < 1 hour after heavy physical exertion (6METS)=5.9

1228 patients w/ MI


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AED Liability?

Good Samaritan law protects those who use it
If you don’t have it, get it:
$2.5 million award for not providing AED
(Chai versus Sports Fitness Clubs of America,
Circuit Court, 17th Judicial District, Broward County, Florida)
36th Bethesda Conference: Eligibility Recommendations for Competitive Athletes With Cardiovascular Abnormalities

Provides expert consensus opinion on exercise with various cardiac conditions

Use as a reference to help make decisions on participation
Exclusion from Competition
Legal Precedent

*Knapp v. Northwestern U.*

Basketball player with episode sudden cardiac arrest
- idiopathic ventricular fibrillation.

Was resuscitated and ICD placed.

Sued for excluding from play under Rehabilitation Act of 1973

Federal district court required NW to allow to return to play
- and make accommodations with standby defibrillator

Appeals court overturned, upheld that team physicians
- have the right to bar athlete from competition for medical reasons
Legal Precedent

“In the midst of conflicting expert testimony regarding the degree of serious risk of harm or death, the court’s place is to ensure that the exclusion or disqualification of an individual was individualized, reasonably made, and based upon competent medical evidence. So long as these factors exist, it will be a rare case regarding participation in athletics where a court may substitute its judgment for that of the school’s team physicians.”

Take Home Points

Be aware of causes of syncope and sudden death in young people (and athletes)
Obtain an EKG and echocardiogram if the history is suggestive of an arrhythmia
Exercise can increase risk of cardiac injury and MI, while at the same time providing cardiovascular benefits.
References


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