# Cardiac Conditions in Athletes

The Sports Medicine Core Curriculum Lecture Series
Sponsored by an ACEP Section Grant
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# Top Myths in Sports Medicine

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

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Thompson. Med Sci Sports Exerc, Sept 1993, 25(9): 981-4.

## Corrado et al. *Does sports activity enhance the risk of sudden death in adolescents and young adults?* J Am Coll Cardiol. 2003;42:1959-1963. *copyright Elsevier*

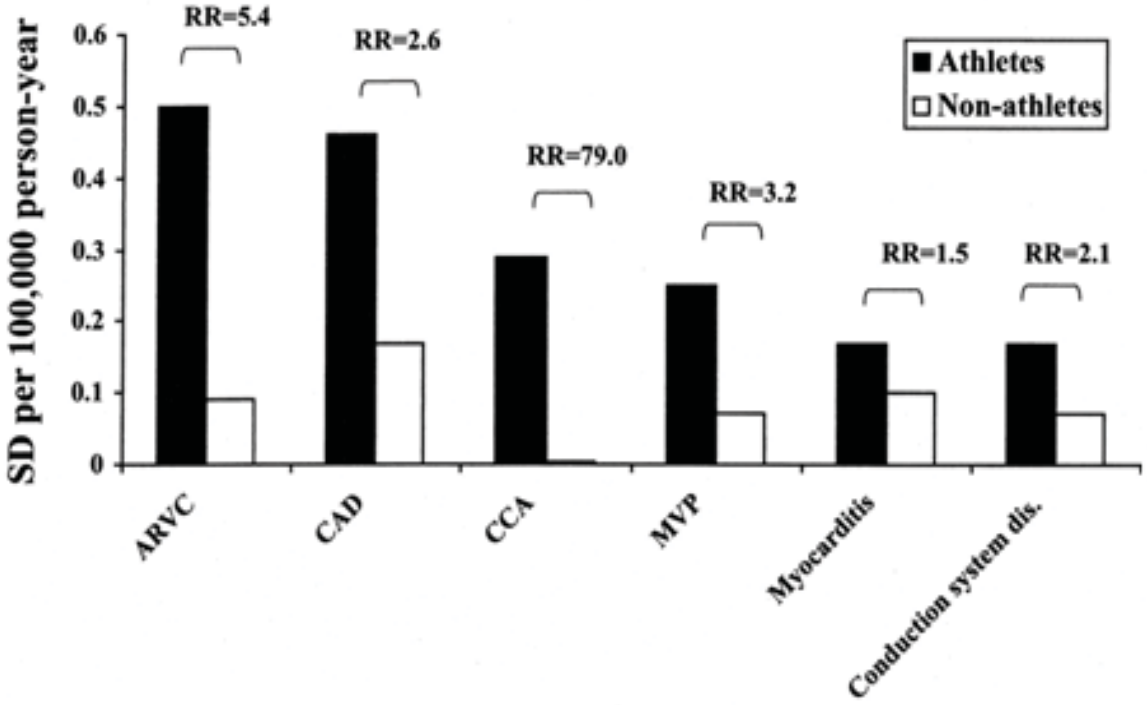


**Figure 2.** Incidence and relative risk of sudden death for specific cardiovascular causes among athletes and nonathletes.

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# 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% |

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Maron et al. NEJM *2003*

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# 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

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VanCamp et al. Med Sci Sports Exerc May 1995, 27(5): 641-7.

# 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

Maron BJ et al. Recommendations and considerations related to preparticipation screening for cardiovascular abnormalities in competitive athletes: 2007 update. A scientific statement from the American Heart Association council on nutrition, physical activity, and metabolism. Endorsed by the American College of Cardiology Foundation. Circulation 2007;115:1643-1655.

# *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

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# 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

*Fuller et al. Med Sci Sports Exerc, Sep 1997, 29(9): 1131-8.*

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**

*Shry et al. Mil Med, Oct 2002; 167 (10):831-4.*

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

*Pelliccia A, Maron BJ. Current Cardiol Rep 2001, 3(2) p147-51.*

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%**

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# *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

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Corrado D et al. Eur Heart J 2005;26:516-24.

# “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

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Zehender et al. Am Heart J, Jun 1990; 199(6):1378-91.

# “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

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Spirito et al. Am J Cardiol, Oct 15 1994, 74(8):802-6.
Pelliccia et al. NEJM Jan 31 1991;324(5):295-301.

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# 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%

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Abernethy et al. J Am Coll Cardiol, Jan 15 2003, 41(2): 280-4.

# 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

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Crouse et al. Res Q Exerc Sport, Dec 1992 63(4):393-401.

# “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

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Pelliccia et al. Cardiol Rev, Mar-Apr 2002, 10(2): 85-90.

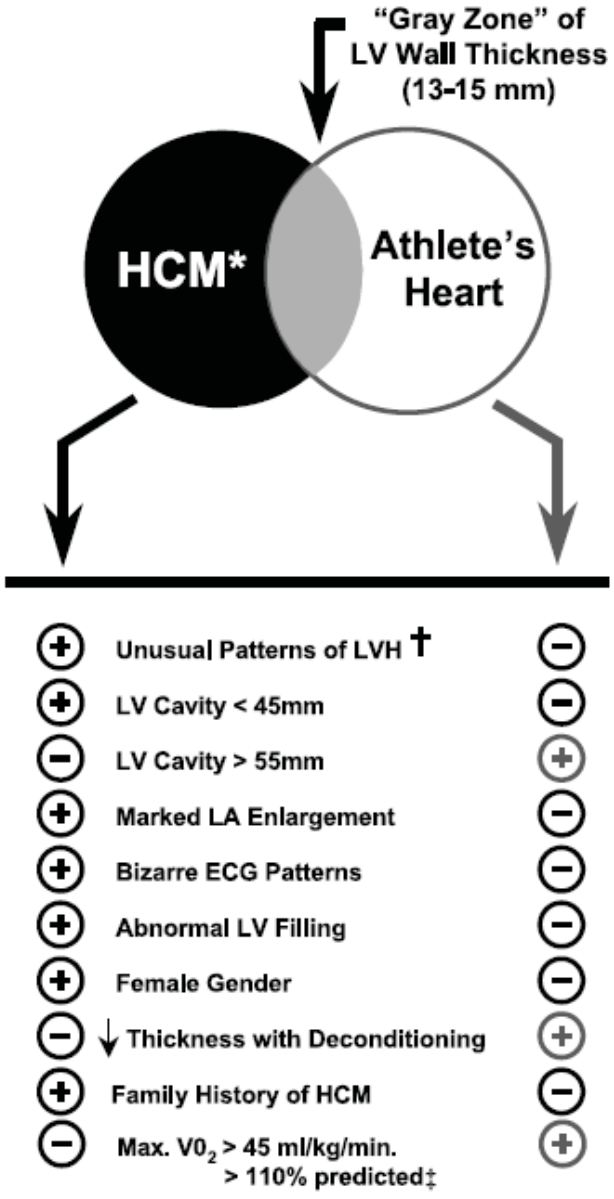


*FIGURE 2 From Maron BJ, et al. Circulation 1995;91(5):1596–601, Cardiac Disease in Young Trained Athletes: Insights Into Methods for Distinguishing Athlete's Heart From Structural Heart Disease, With Particular Emphasis on Hypertrophic Cardiomyopathy*

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# *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

*Thiene G, et al. Right ventricular cardiomyopathy and sudden death in young people. NEJM 1988;318:129-133.*

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*

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Basso et al. J Am Coll Cardiol, May 2000, 35(6): 1493-501.

# Commotio Cordis

Blunt chest wall impact, low energy

Ventricular fibrillation

NCAA ~ 10-20 cases per year in young males

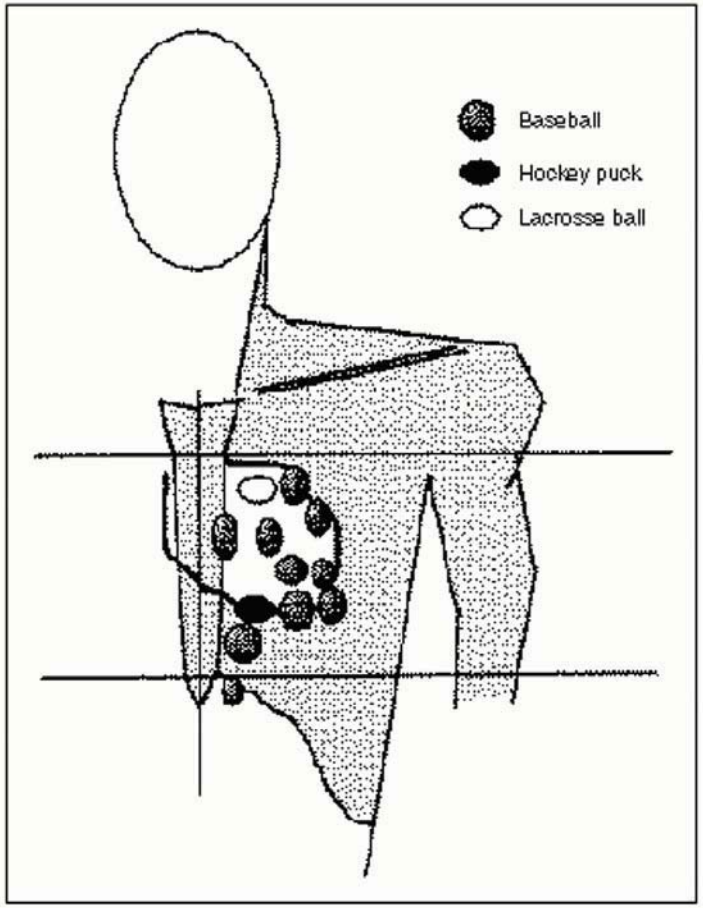


Figure 3: Maron BJ, Poliac LC, Kaplan JA, Mueller FO. Blunt Impact to the Chest Leading to Sudden Death from Cardiac Arrest During Sports Activities. N Engl J Med 333:337, 1995.



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## Kark et al. *Sickle-cell trait as a risk factor for sudden death in physical training*. NEJM 1987; 317(13):781-787.

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

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Tulloh, L et al. Raised Troponin T and Echocardiographic Abnormalities after Prolonged Strenuous Exercise-The Australian Ironman Triathalon Br J Sports Med 2006;40:605-609

**Koller, A.** Exercise-Induced Increases in Cardiac Troponins and Prothrombotic Markers. **Med Sci Sports Exercise:35(3); 2003, 444-448.**

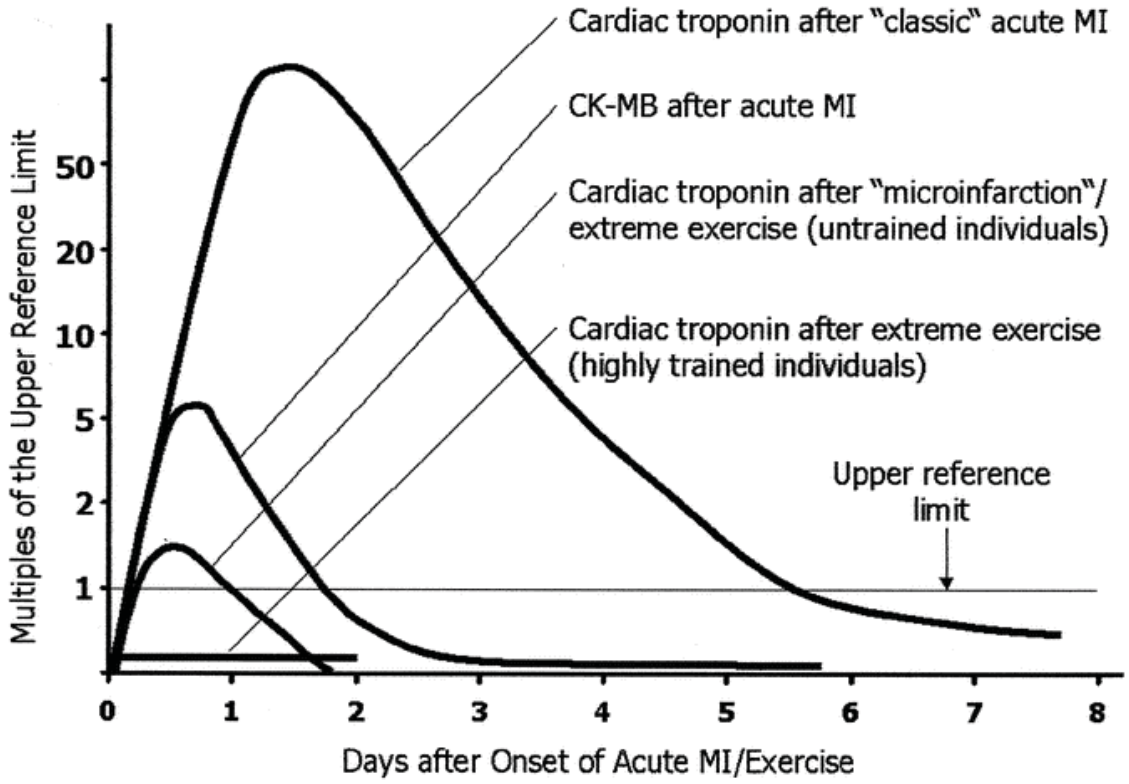


Figure 1

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# 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

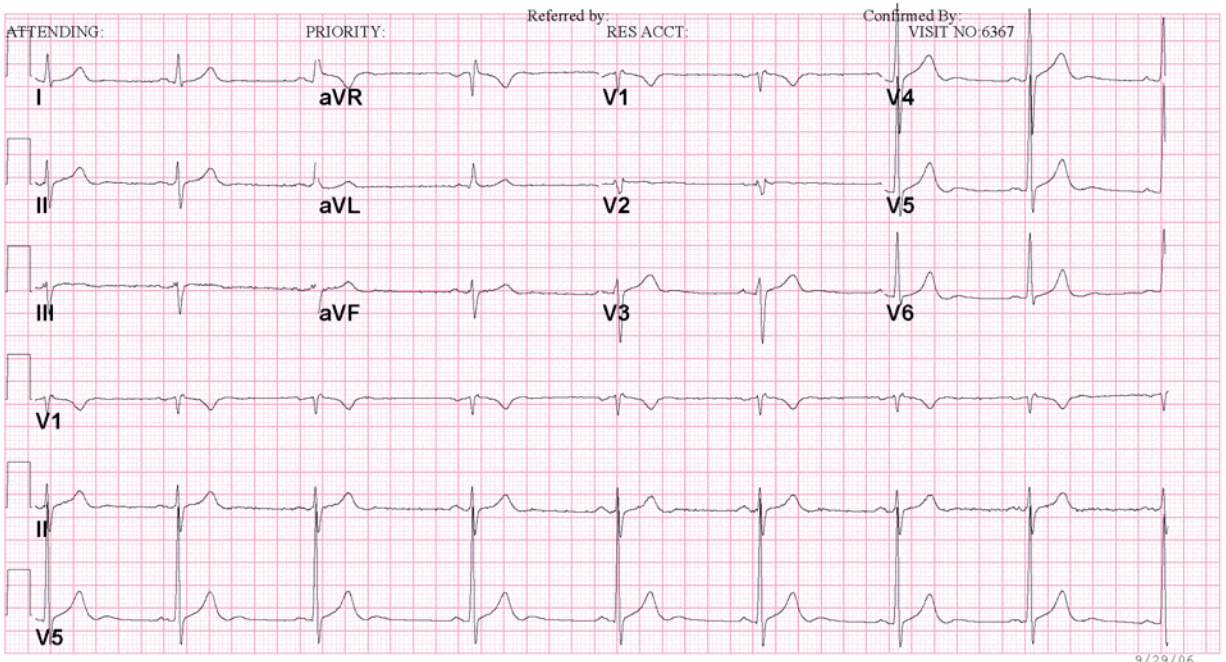
# Case: 55 yo M Chest Pain at rest 1 hr PTA
# <3 weeks after Triathalon; 6 d after 64 K bike ride

(55 yr)
Male

Loc:6

| | | |
|---|---|---|
| Vent. rate | 48 | BPM |
| PR interval | 144 | ms |
| QRS duration | 98 | ms |
| QT/QTc | 448/400 | ms |
| P-R-T axes | 39 -33 | 32 |

**Marked sinus bradycardia**
**Left axis deviation**
**Minimal voltage criteria for LVH, may be normal variant**
**Abnormal ECG**

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# Case: 55 yo M Chest Pain at rest 1 hr PTA
# <3 weeks after Triathalon; 6 d after 64 K bike ride

Labs #1:

Troponin = 0.51

CPK = 157

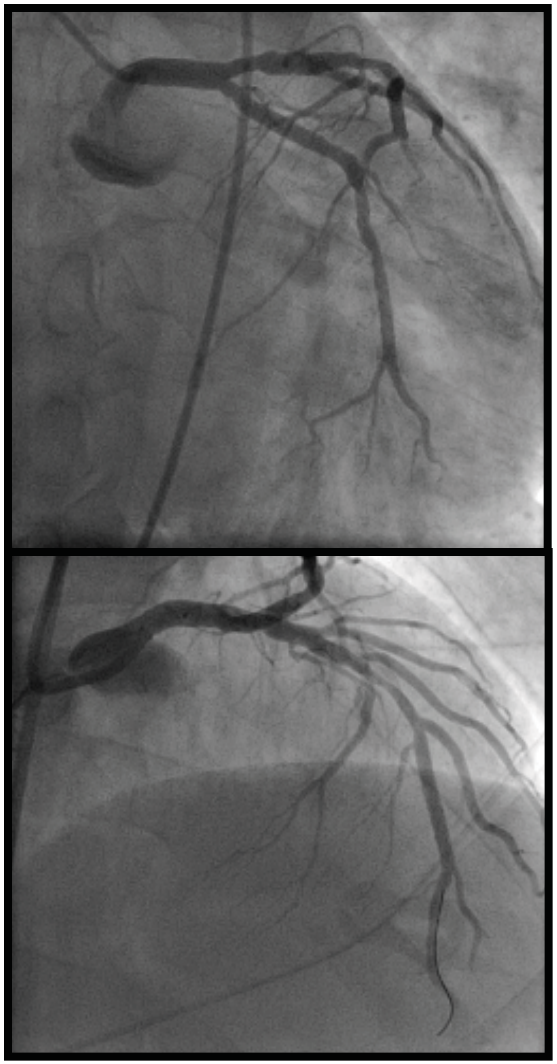
CKMB = 2.2

Cardiac cath shows:

70% D2 ostial lesion

90% mid LAD stenosis w/

plaque rupture and dissection



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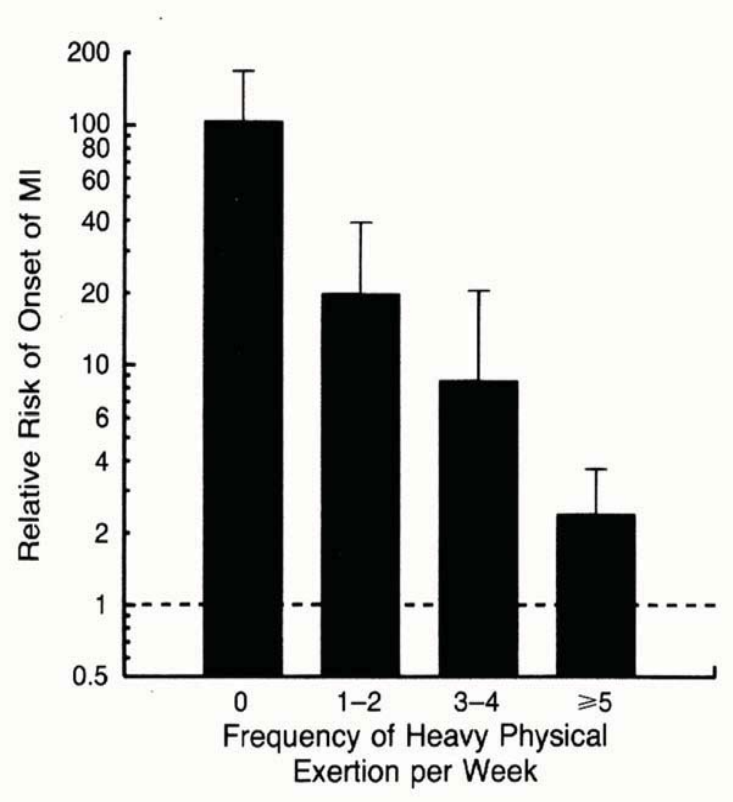
# Exercise-Induced MI

## Relative risk of MI < 1 hour after heavy physical exertion (6METS)=5.9

1228 patients w/ MI




*Mittleman MA, Maclure M, Tofler GH, Sherwood JB, Goldberg RJ, Muller JE. Triggering of acute myocardial infarction by heavy physical exertion: protection against triggering by regular exertion. N Engl J Med 1993;329:1677-1683.* Figure 2.



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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)

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## 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

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# 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."

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Knapp v. Northwestern University, No 96-3450 US Court of Appeals for the Seventh Circuit. November 22, 1996.

# 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.

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