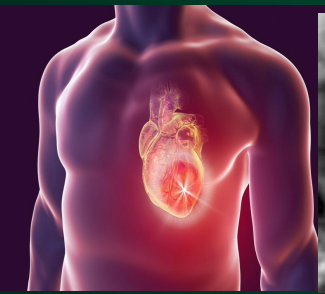
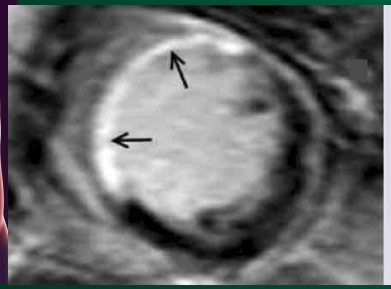
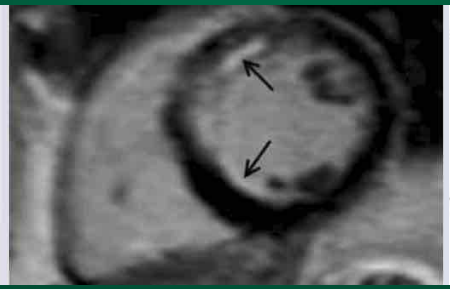
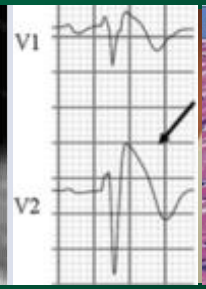
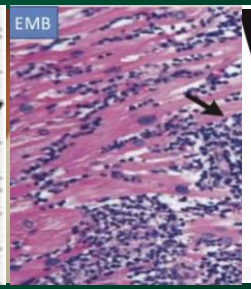
Cardiac Considerations:

Cardiac screening, Cardiac Conditions and COVID RTP



Department of Orthopedic Surgery & Department of Cardiology

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Sports Cardiology Clinic: Our team

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Objectives

1. Understand the sports cardiology components of a pre-participation physical

2. Describe medical conditions that can result in sudden cardiac death

3. Know which tests should be ordered for further cardiac work up, including post-COVID

Pre-participation Sports Screening in Pediatrics

"How to Properly Dig Up Landmines"

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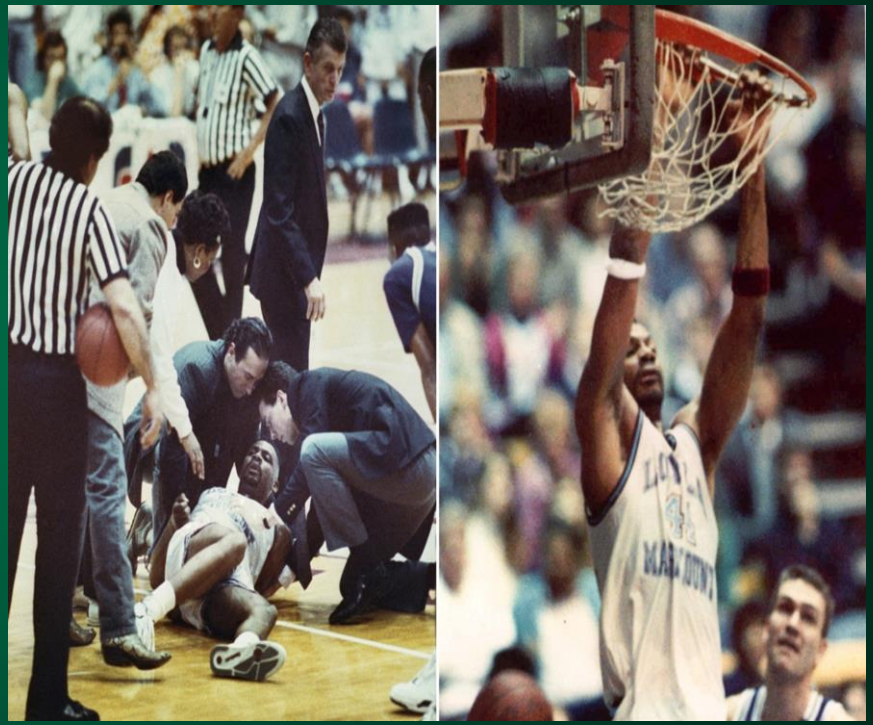
Excavation Plan

1. “Lay of the Land” – Background, Incidence, Guidelines, etc.
2. Particularly Bad Landmines – Causes of Sudden Cardiac Death in Young Athletes
3. Pre-participation Screening in the Time of COVID

Background

- Sudden cardiac death (SCD) in a young, previously “healthy” athlete is rare but devastating – family, school, community, etc.
- Some events are unexplained, however many harbor undiagnosed cardiovascular disease, with the end result being VT/VF → SCD



Background

In patients with certain CV conditions, athletics increases the risk of VT/VF in 2 theoretical ways:

1. In those with abnormal cardiac substrate, physical training can lead to maladaptive changes in cardiac structure (intracardiac fibrosis, dilation of RV/LV) → ↑arrhythmia risk
2. The demands of intense athletics (hemodynamic stress, catecholamine release, electrolyte imbalance) can trigger arrhythmias in susceptible individuals

How Should We Screen?

U.S.A. Guidelines

Emphasis on 14 point H&P screening

Routine EKG NOT recommended

- Limitations due to false (+)s – time lost, $, family angst, etc
- U.S. : approximately 4 million high school athletes
- Diverse population: “We ain’t Italy”
- Even AFTER many diagnoses, uncertainty re: restricting sports/physical activity

Maron BJ, et al. JACC 2015 Dec;66(21):2356-61

Personal History

1. Exertional chest pain/discomfort
2. Exertional syncope or near-syncope
3. Excessive exertional and unexplained fatigue/fatigue associated with exercise
4. Prior recognition of a heart murmur
5. Elevated systemic blood pressure
6. Prior restriction from participation in sports
7. Prior testing of the heart ordered by a physician

Family history

8. Premature death-sudden/unexpected < 50 yr due to heart disease, in a relative
9. Disability from heart disease in a close relative < 50 yo
10. Specific knowledge of certain cardiac conditions in family members: HCM, DCM, LQTS, other channelopathies, Marfan, other arrhythmias

Physical exam

11. Heart Murmur-exam supine and standing or with valsalva, specifically to identify murmurs of dynamic L ventricular outflow tract obstruction
12. Femoral pulses to exclude aortic stenosis
13. Physical stigmata of Marfan syndrome
14. Brachial artery blood pressure (sitting, preferrably taken in both arms)

How Americans View Europe

European Guidelines

- **Biggest difference from U.S. guidelines is that additional EKG screening is RECOMMENDED**
- **Why different? Chiefly data from Italy**
  - National EKG screening program since 1982 (data to be discussed)

Corrado D et al. Eur Heart J. 2005;26(5):516.

So, How Effective is Screening Anyway?

- **Limited data on efficacy**
- **Prevalence of abnormal EKG findings?***
  - N = 32,652 Italians who underwent routine pre-participation screening that included an ECG
  - The prevalence of an abnormal EKG was 11%, and of markedly abnormal ECG pattern < 5%
- Most frequent abnormal findings: prolonged PR interval, incomplete right bundle branch block (RBBB) and early repolarization pattern
- More likely abnormal findings: deep inverted T-waves (2.3%), LVH (0.8%), RBBB (1.0%), left anterior fascicular block (0.5%), LBBB (0.1%), pre-excitation pattern (0.1%) and prolonged QTc interval (0.03%)

*Pelliccia A, et al. Eur Heart J. 2007;28(16):2006.

So, How Effective is Screening Anyway?

Other European Perspectives*

N = 11,168 adolescent English athletes (mean age 16.4 years)

Underwent EXTENSIVE cardiac screening (including H&P, ECG, and echo)

N = 42 athletes (0.4 percent) were found to have disorders associated with sudden cardiac death

N = 8 athletes with a negative screen STILL suffered SCD (5 from a cardiomyopathy)

- SCD at a mean of 7 years after initial screen
- Six of the 8 had a normal initial screen, but no follow-up → need for serial assessment

Malhotra A, et al. N Engl J Med. 2018;379(6):524.

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What is the Impact of Screening?

- Data from Italy, again

  Annual incidence of SCD in athletes was 3.6/100,000 person-years from 1979 to 1980

- H&P + EKG screening instituted nationwide in 1982

- SCD incidence dropped to 0.4/100,000 person-years in 2003 to 2004 (89 percent reduction)

  Presumably due to H&P + EKG screening

  Notably, there was no change in the incidence of SCD among non-athletes over the same time period.

Corrado D et al. JAMA. 2006;296(13):1593.

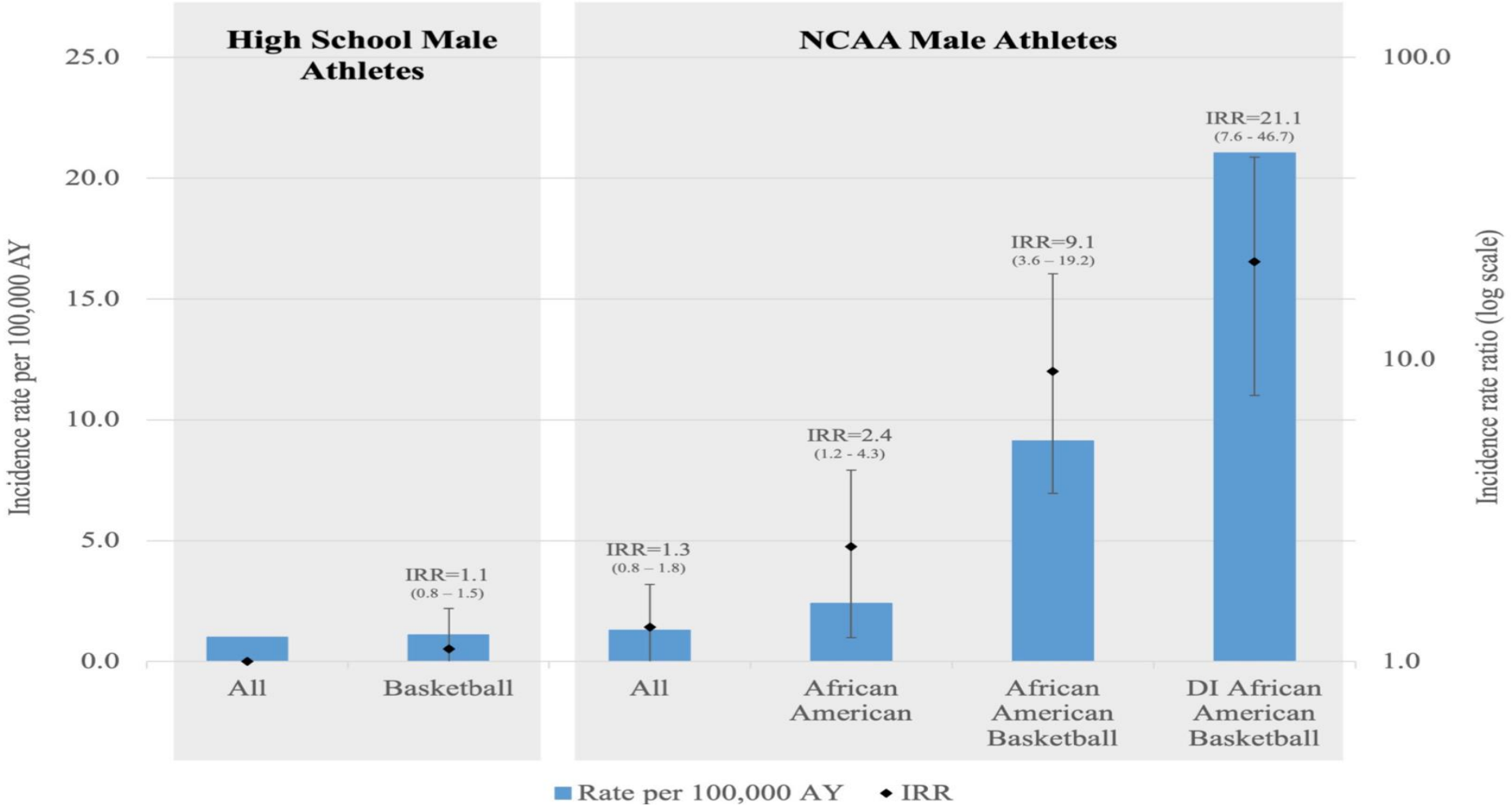

Figure 6 Incidence rate ratios (IRR) of sudden cardiac arrest and death in select athlete populations compared with all high school male athletes. DI, division I, NCAA, National Collegiate Athletic Association.

Excavation Plan

1. “Lay of the Land” – Background, Incidence, Guidelines, etc.
2. **Particularly Bad Landmines – Causes of Sudden Cardiac Death in Young Athletes**
3. Pre-participation Screening in the Time of COVID

SCD: What Are We Screening For?

Structural/Functional

1) Hypertrophic Cardiomyopathy (HCM)
2) Coronary Artery Anomalies
3) Aortic Rupture/Marfan
4) Dilated Cardiomyopathy (DCM)
5) Myocarditis
6) Left Ventricular Outflow Tract Obstruction
7) Mitral Valve Prolapse (MVP)
8) Coronary Artery Atherosclerotic Disease
9) Arrhythmogenic Right Ventricular Cardiomyopathy (ARVC)

Electrical

11) Long QT Syndrome (LQTS)
12) Wolff-Parkinson-White Syndrome (WPW)
13) Brugada Syndrome
14) Catecholaminergic Polymorphic Ventricular Tachycardia (CPVT)
15) Short QT Syndrome

Other

17) Drugs and Stimulants
18) Primary Pulmonary Hypertension (PPH)
19) Commotio Cordis

Etiology of SCD in Young Athletes

The most common structural heart diseases leading to SCD:

- **Hypertrophic cardiomyopathy (HCM)**
- **Anomalous origin of a coronary artery**

Study*: Among 1435 young U.S. athletes in a SCD registry, on post-mortem exam, the most common diagnoses were HCM (36%) and anomalous origin of a coronary artery (20%)

*Maron BJ, et al. Circulation. 2007;115(12):1643.

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U.S. Military Study

- **Six million U.S. military recruits (mean age 19) 126 nontraumatic sudden deaths occurred**
- **N = 108 [86%] deaths were related to exercise**
  - 33 % Anomalous origin of a coronary artery
  - 20% myocarditis
  - 16% coronary atherosclerosis
  - 13% HCM

Eckart RE et al. Ann Intern Med. 2004;141(11):829.

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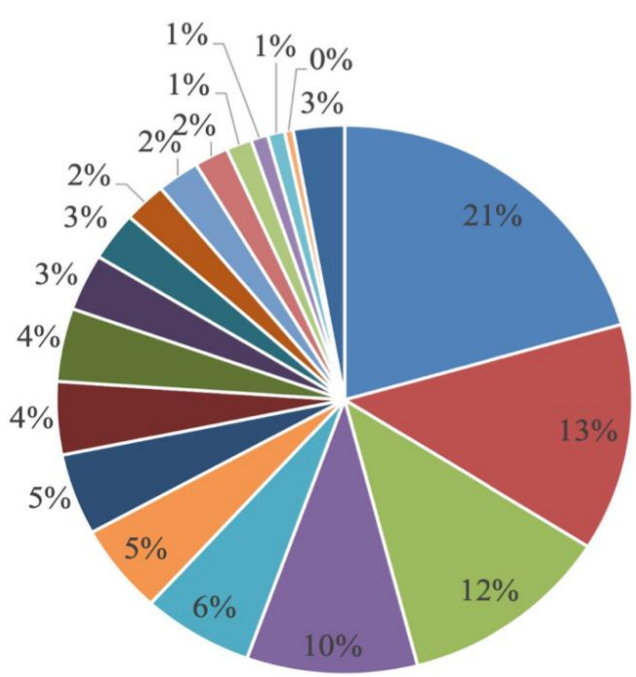
Etiology of SCD in H.S. and College U.S. Athletes, 2014-2018



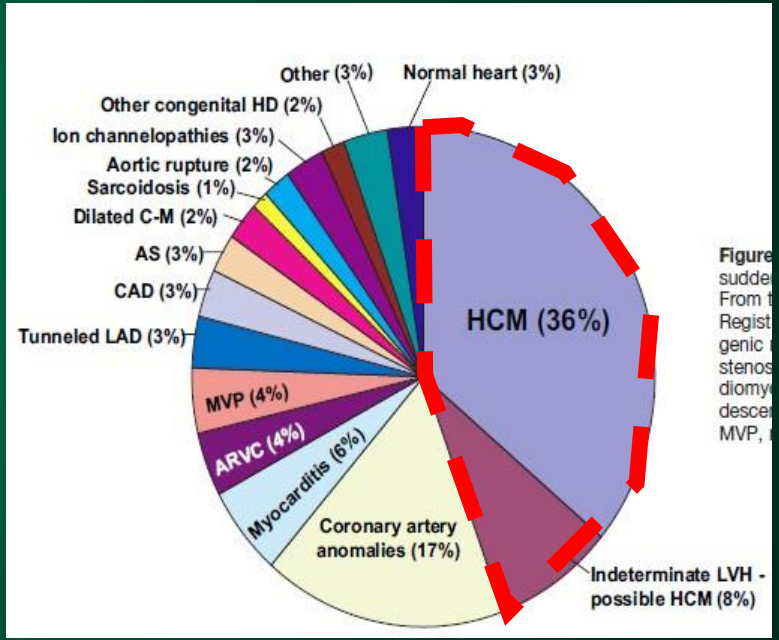
- Hypertrophic cardiomyopathy (43, 20.6%)
- Idiopathic left ventricular hypertrophy (28, 13.4%)
- Coronary artery anomalies (25, 12.0%)
- Autopsy negative sudden unexplained death (20, 9.6%)
- Arrhythmogenic cardiomyopathy (13, 6.2%)
- Long QT syndrome (11, 5.3%)
- Commotio cordis (10, 4.8%)
- Wolff-Parkinson-White (9, 4.3%)
- Myocarditis (9, 4.3%)
- Aortic dissection/rupture (7, 3.3%)
- Dilated cardiomyopathy (6, 2.9%)
- Valve disorder (5, 2.4%)
- Coronary atherosclerosis (5, 2.4%)
- Complications of a congenital heart defect (4, 1.9%)
- Catecholaminergic polymorphic ventricular tachycardia (3, 1.4%)
- Hypertensive heart disease (2, 1.0%)
- Left ventricular noncompaction (2, 1.0%)
- Restrictive cardiomyopathy (1, 0.5%)
- Other (6, 2.9%)

Figure 2 Aetiology of sudden cardiac arrest and death (n=209).

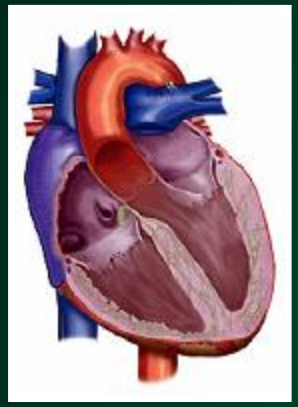
Peterson D. Br J Sports Med 2020

Hypertrophic Cardiomyopathy

- **Relatively common! 1:500 individuals in the general population**
- **The most common structural heart cause of SCD in athletes**
  - Figure: 2007 AHA statement, showing results from the Minneapolis Heart Institute registry of causes of SCD in athletes from 1980-2005
  - HCM or POSSIBLE HCM = 44% of all causes
- **In most athletes with SCD due to HCM, the diagnosis was not previously established**



Hypertrophic Cardiomyopathy



Normal

HCM Without Obstruction

HCM With Obstruction

- **Pathology: gene mutation(s) in different proteins involved in the cardiac sarcomere (50% with identifiable gene)**
  - Disorganized, fibrotic, and hypertrophied myocardium
  - Prone to arrhythmias
  - Additional problems: LV outflow obstruction, diastolic dysfunction, MR, heart failure
- **Autosomal dominant inheritance (vs. de novo mutation) → Family history!!!**
- **Typical onset of clinically significant findings (EKG, echo) → during puberty**
- **SCD can be initial presentation**

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Effect of selected maneuvers

Maneuver	Mechanism	Effect on gradient and murmur
Valsalva (strain)	Decreased LV cavity, pre and after load	Increased
Standing	Decreased LV cavity, preload	Increased
Squatting	Increased LV cavity, pre and after load	Decreased
Isometric hand grip	Increased afterload	Decreased

Effect of selected maneuvers

Maneuver	Mechanism	Effect on gradient and murmur
Valsalva (strain)	Decreased LV cavity, pre and after load	Increased
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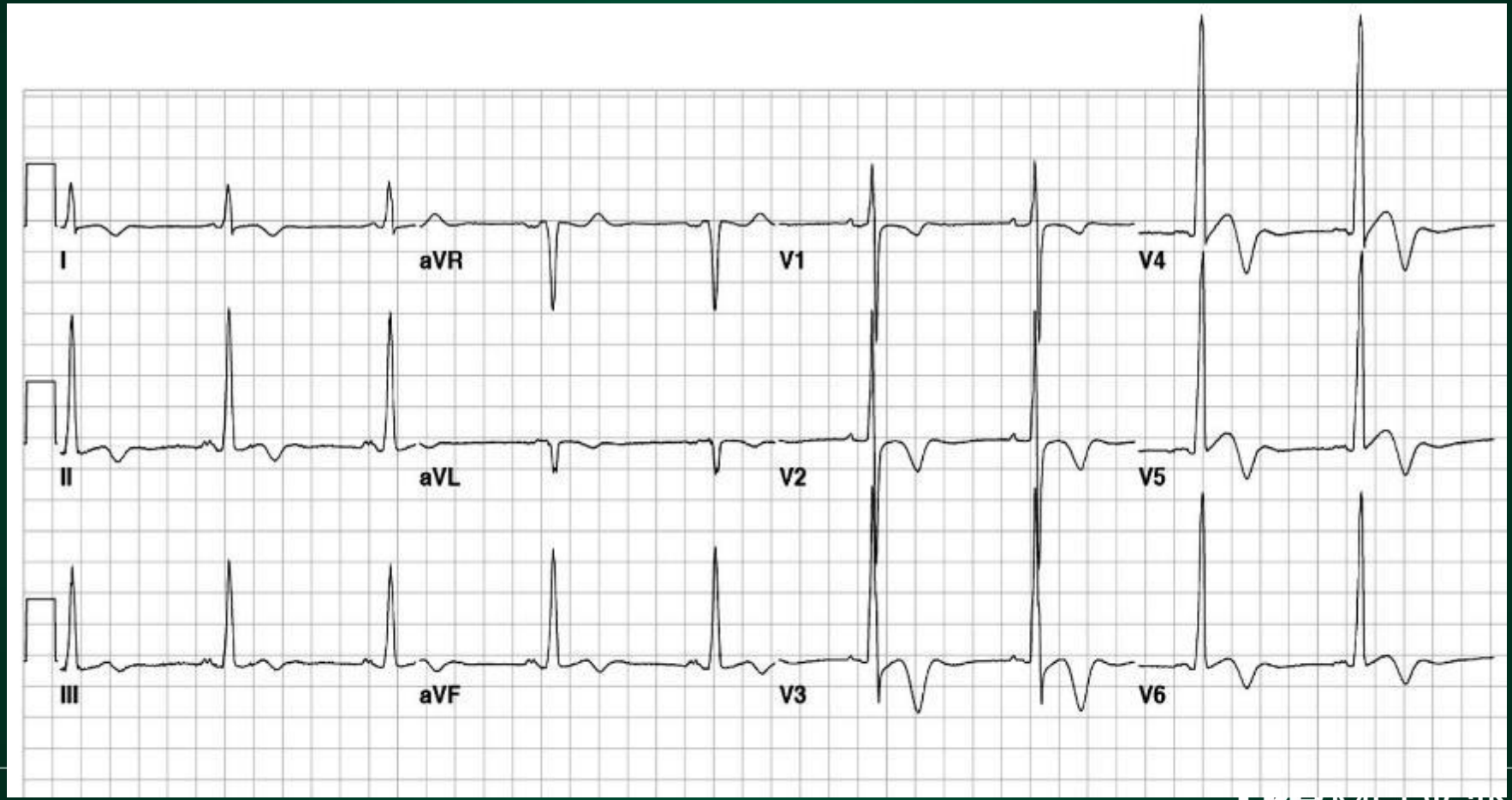

I
aVR
V1
V4
II
aVL
V2
V5
III
aVF
V3
V6

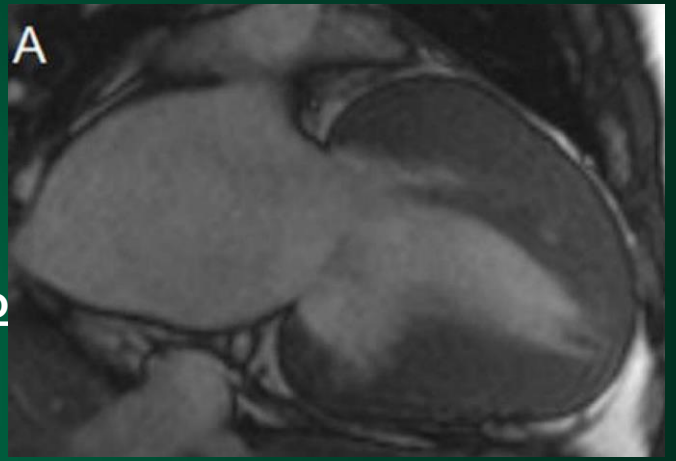
Hypertrophic Cardiomyopathy and Sports Clearance

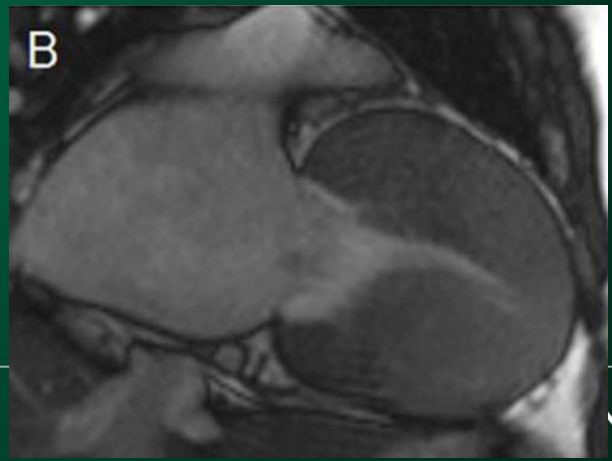
Old school: not cleared for sports, recreational/low static/dynamic sports only

Trend toward clearance of more HCM patients for more types of exercise → don't turn ALL of these patients into couch potatoes!

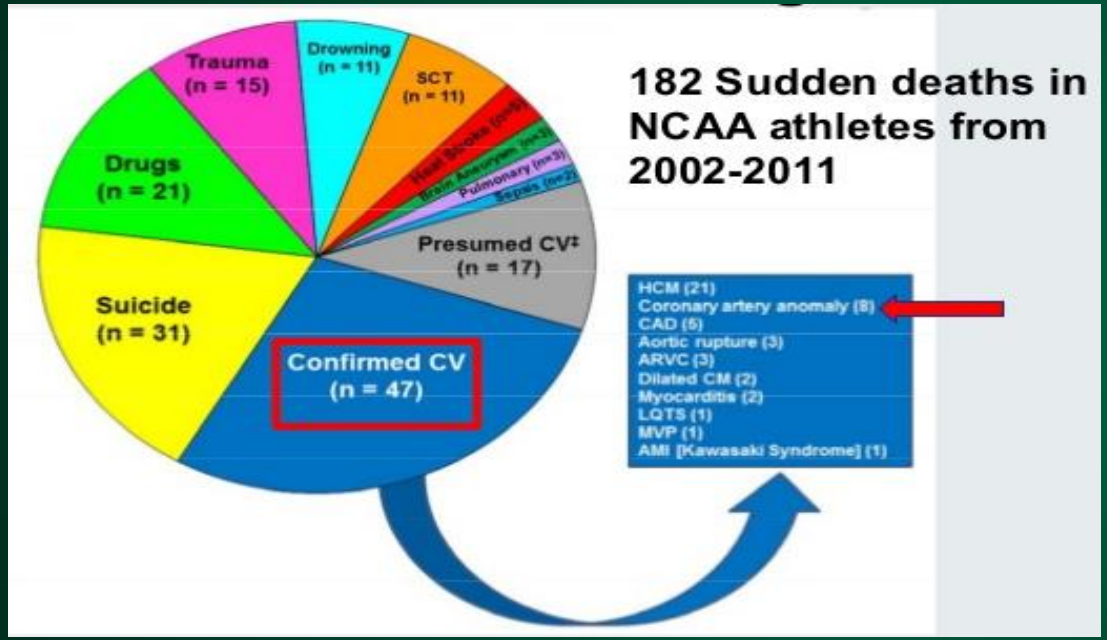
Highest Risk Patients: proceed with caution!

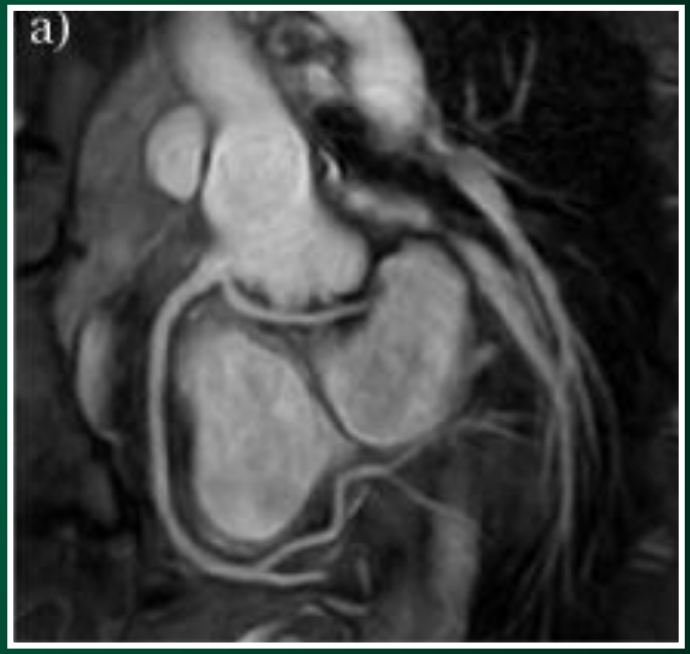
1. Family history of SCD
2. Super severe LVH – IVS >/= 30 mm
3. Sustained VT on Holter monitor
4. Non-vasovagal syncope
5. Decrease BP with exercise, or failure to augment





Coronary Anomalies

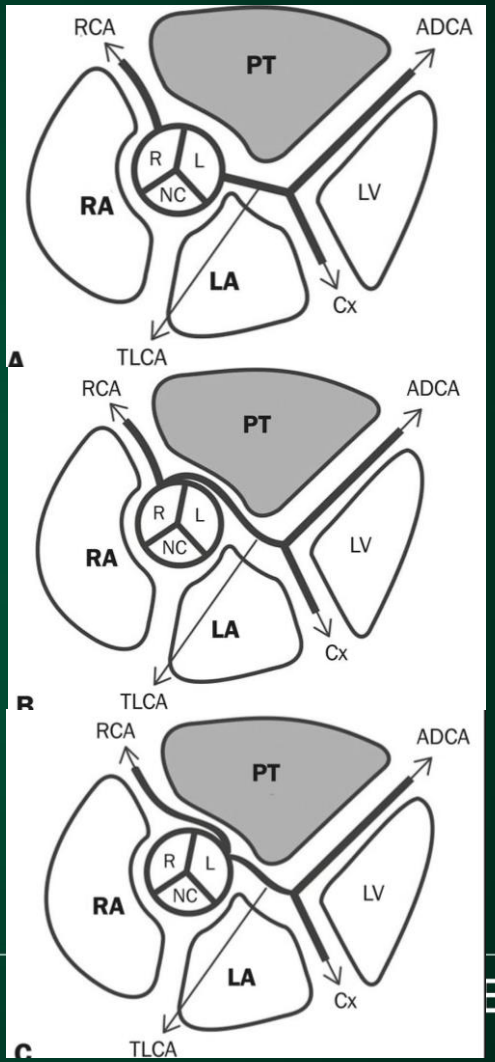




Maron BJ, et al. Incidence and Causes of Sudden Death in U.S. College Athletes. JACC, 2014.

Coronary Anomalies

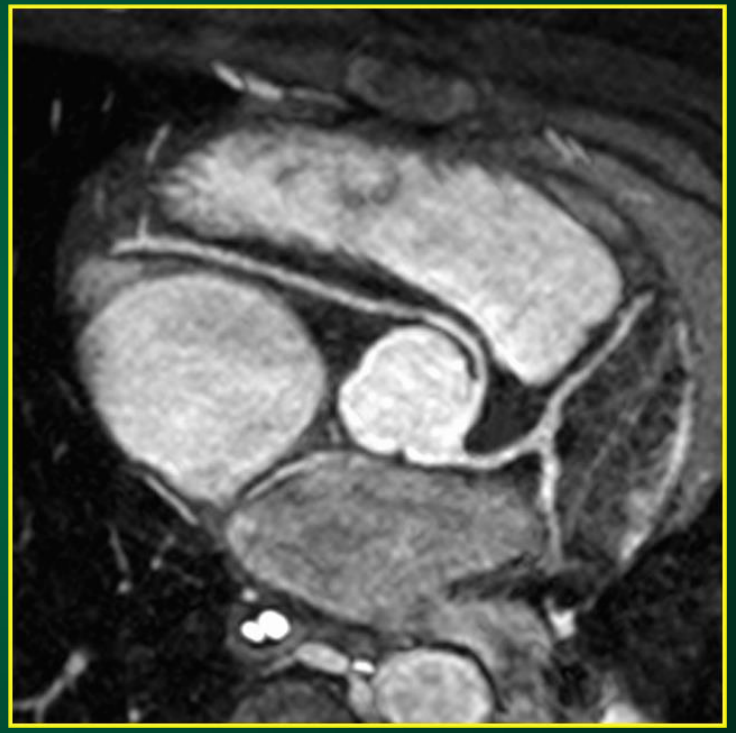
- **Incidence: 0.64% of live births***
- **Anomalous Left Coronary Artery from the Opposite Sinus is most common and when found is an indication for surgery!**
- **Anomalous Right Coronary Artery from the Opposite Sinus is less common and controversial in terms of management**
- **Other variations:**
  - Single coronaries (often left alone)
  - Anomalous origin from the pulmonary artery (typically symptomatic as infants)
  - Ectopic origin from the aorta
  - myocardial bridges



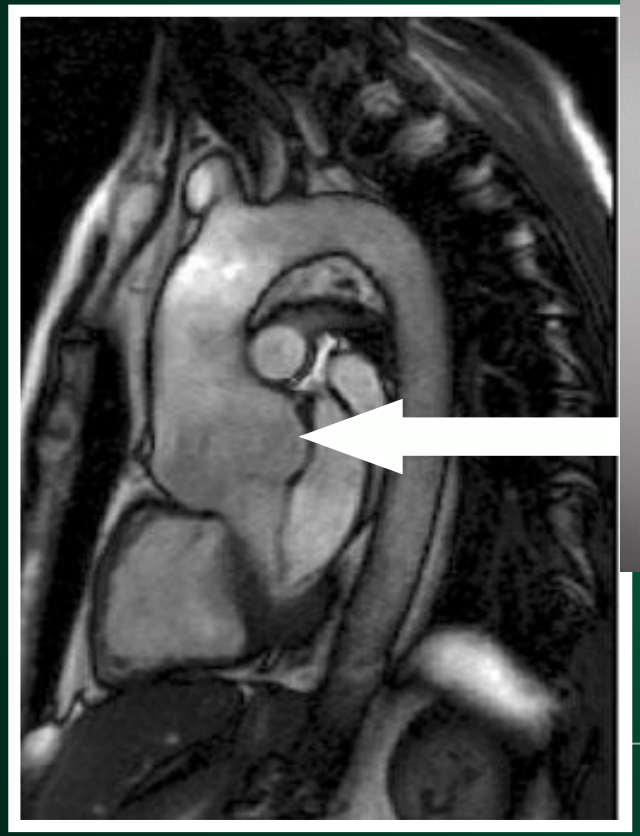
Kimbris D et al. Circulation. 1978;58(4):606.

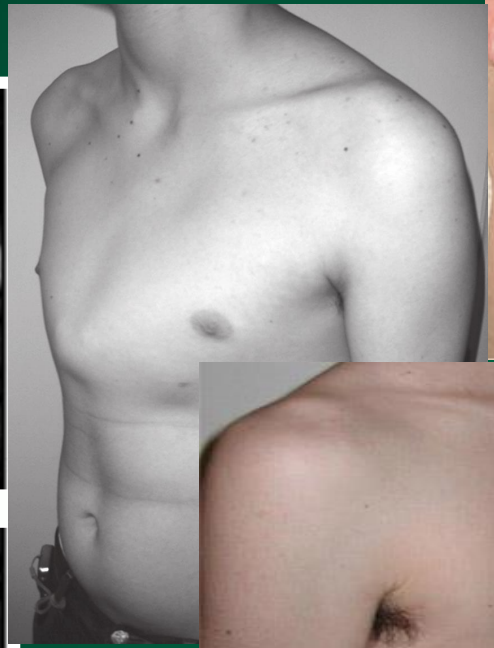
Coronary Anomalies

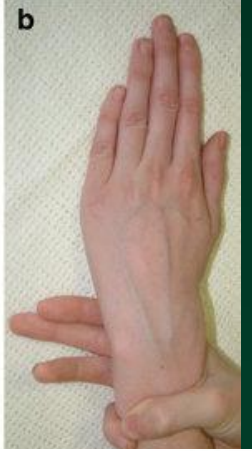
- **Presentation: asymptomatic until it is not!**
- **Chest pain with exercise – can't rule out w/o imaging**
- **Treatment: Surgery**

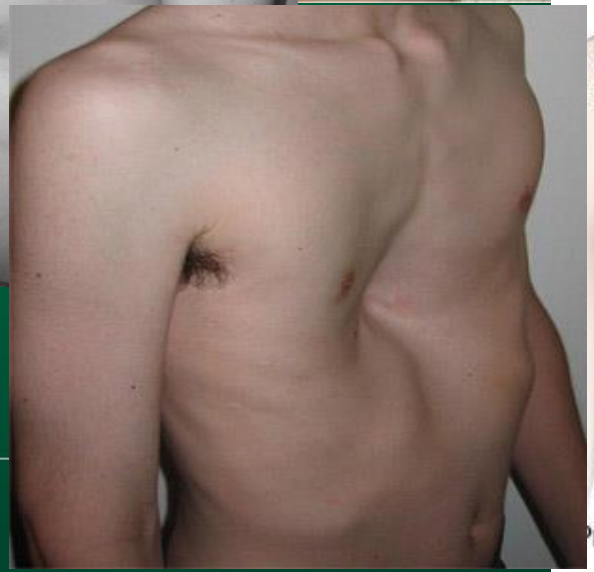


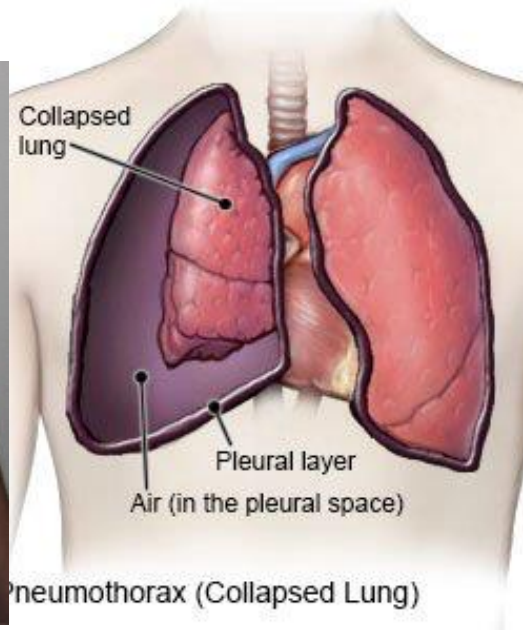
Marfan Syndrome





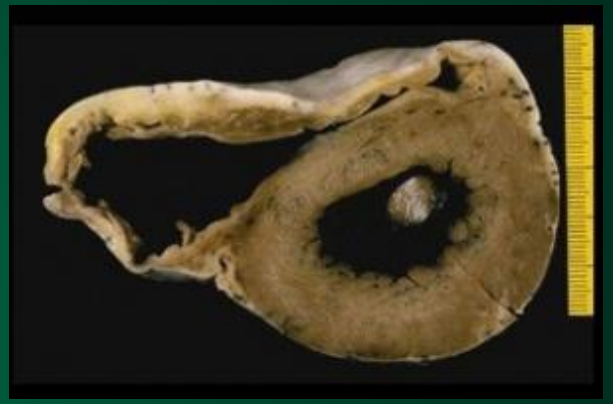


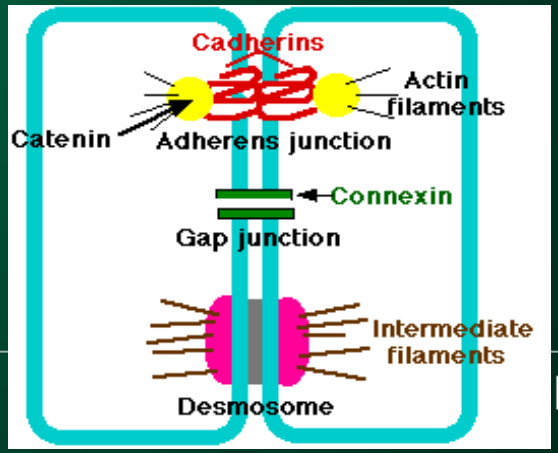




Arrhythmogenic RV Dysplasia (ARVD)

- **Generalized cardiomyopathy; principally RV fibrofatty replacement and progressive dysfunction**
  - Prevalence: 1:3000 adults
  - Cause for up to 11% of SCD in young adults
- **ARVC mutational genes most commonly encode desmosomal proteins**





Corrado D et al. Circulation. 2000;101(11):E101.

Arrhythmogenic RV Dysplasia (ARVD)

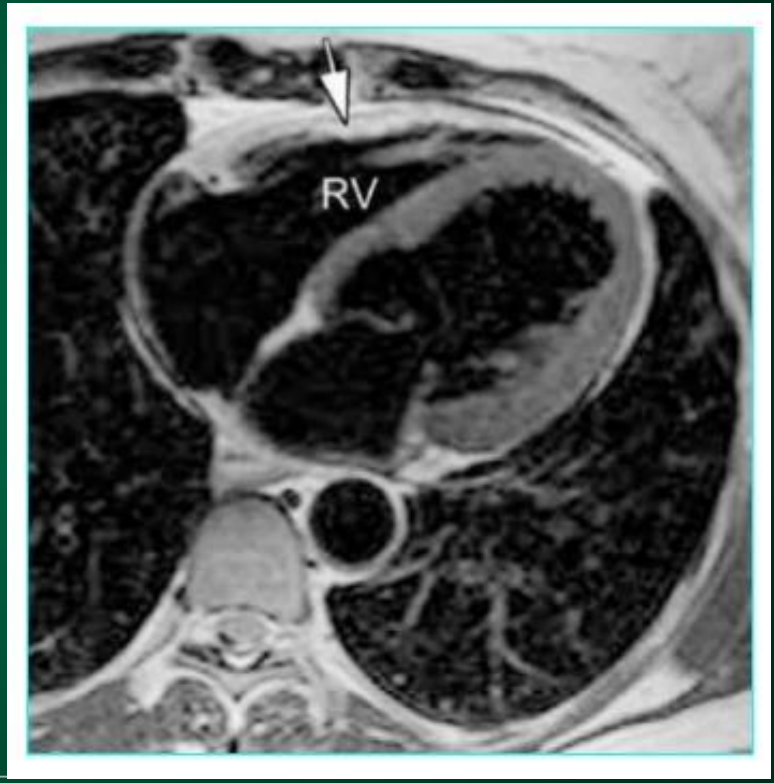
Symptoms: exercise-associated syncope/palpitations/VT/SCD

- Family history – AD form is most common
- But, 25-40% of patients are asymptomatic at time of diagnosis

Rarely diagnosed before puberty; mean age at diagnosis ~ 30 years

EKG changes

Diagnosis



Corrado D et al. Circulation. 2000;101(11):E101.

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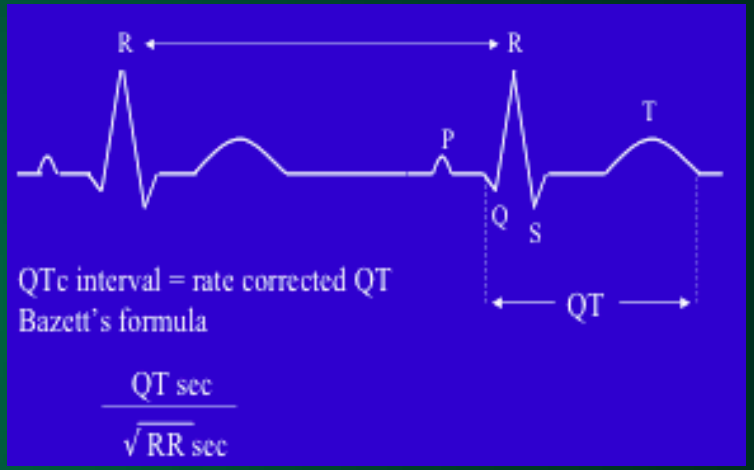
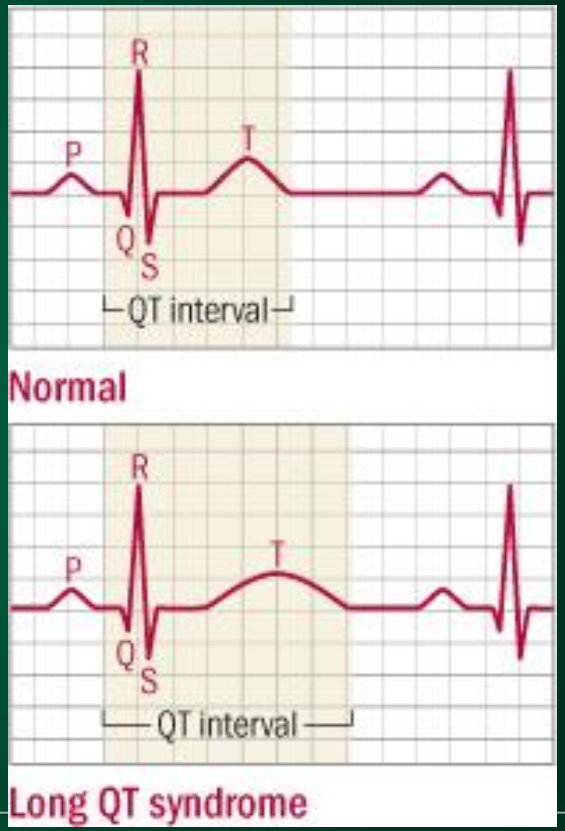
Long QT syndrome

- Problem with the way electricity travels through the heart, which can result in disorganized electrical conduction (ventricular tachycardia)
- Diagnosed by a long interval between the Q and T segments on an EKG
- A “normal” Q on a single resting EKG does not exclude the diagnosis



Long QT syndrome

- **Normal QTc < 440**
- **Borderline QTc**
  - 440 - 460 ms men
  - 440 - 470 ms women

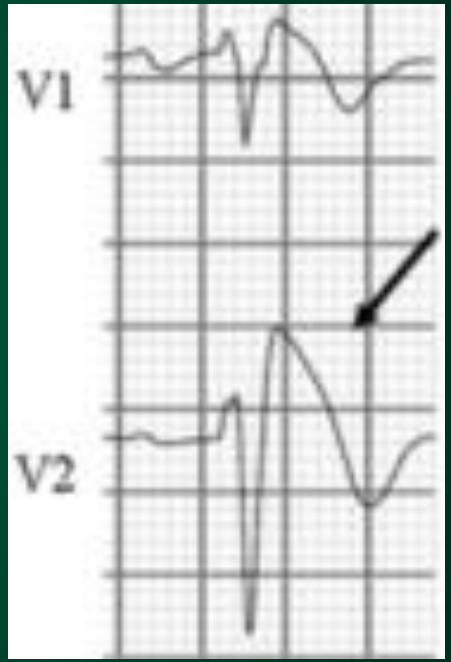


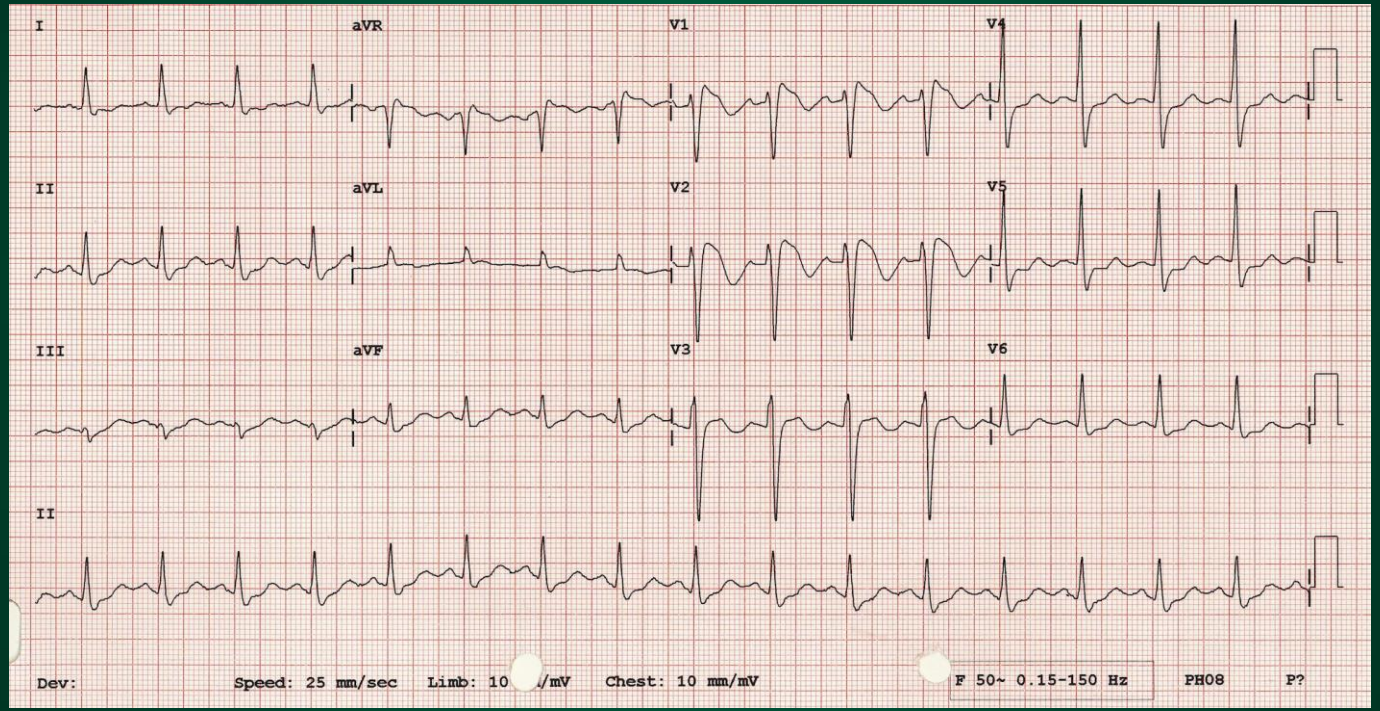
Long QT syndrome

- EKG
  - Prolonged QTc
  - T-wave changes
  - Torsades de Pointes
- Other things that can cause long QT interval – drugs, electrolytes (K, Mg, Ca), acidosis

Brugada syndrome

- Autosomal dominant arrhythmia syndrome characterized by risk of SCD and an abnormal EKG
- EKG – "pseudo-right bundle branch" pattern with ST segment elevation in leads V1/V2 (coved pattern). TWI V1-3
- Most common gene mutation is in SCN5A (Na channel, loss of function)





Children's of Alabama - University of Alabama at Birmingham

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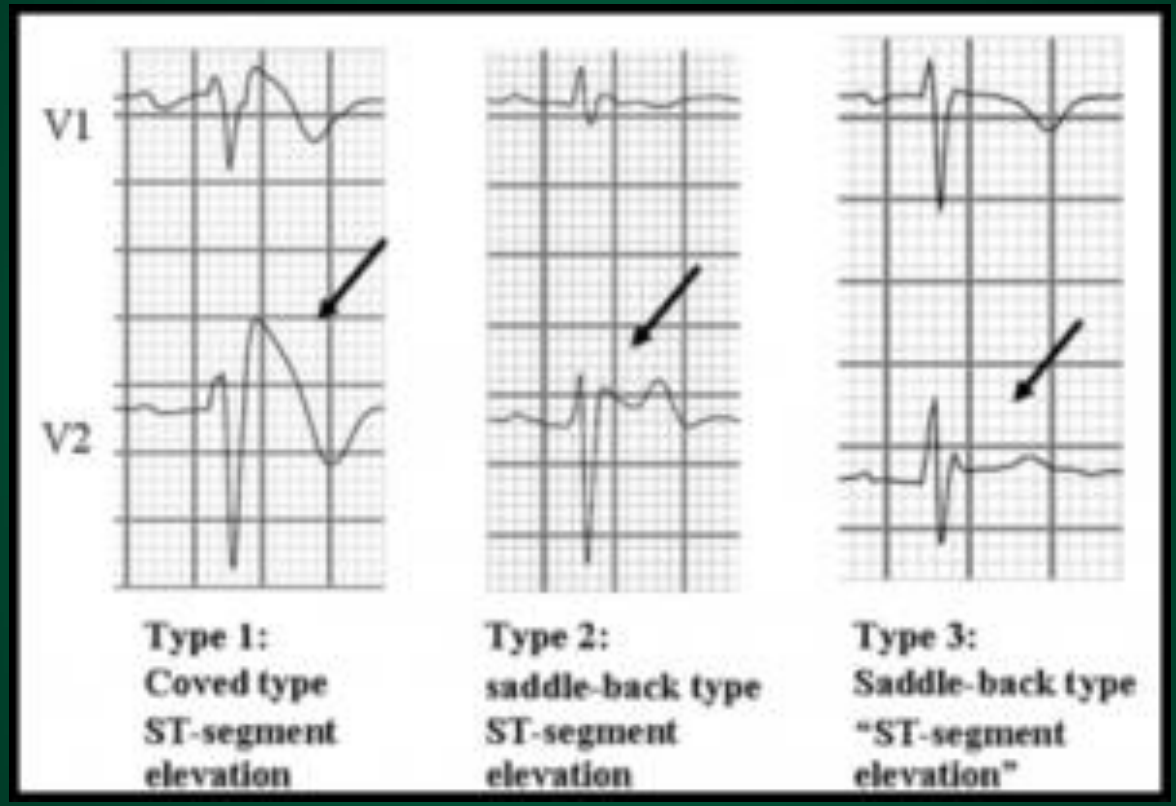

V1
V2
Type 1:
Coved type
ST-segment
elevation
Type 2:
saddle-back type
ST-segment
elevation
Type 3:
Saddle-back type
"ST-segment
elevation"

Brugada syndrome

Typical presentation at older patient age (third to fourth decade)

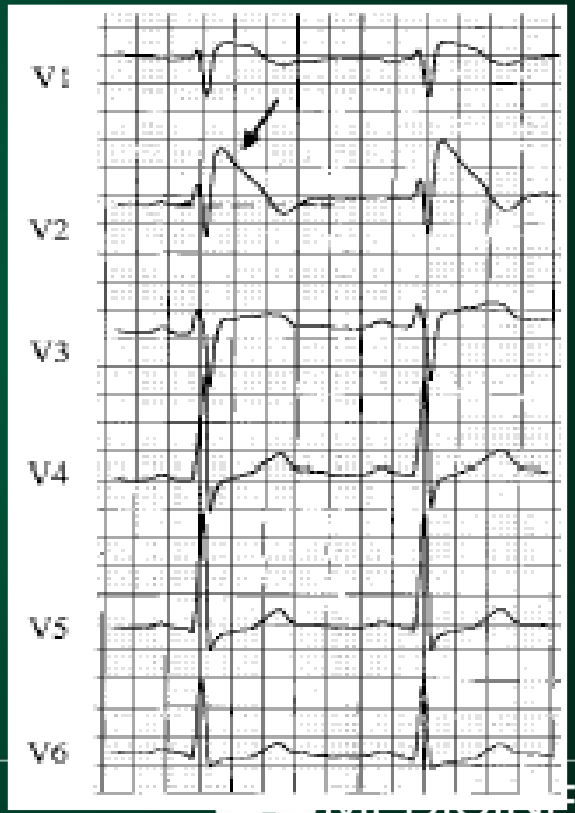
Rare pediatric presentation

- Syncope
- Febrile seizure
- SCD can be the first presentation
- Family history of Brugada syndrome, syncope, SCD

Precipitating events → fever and sleep

Treatment

- Fever treatment
- Med avoidance - www.brugadadrugs.org
- ICD



Catecholaminergic Polymorphic Ventricular Tachycardia (CPVT)

Cause

Inherited genetic code for abnormal protein which allows calcium to leak in the heart. This interferes with electrical conduction

Clinical

Young patient age

Exercise/emotion-induced seizure/syncope (can be misdiagnosed as epilepsy)

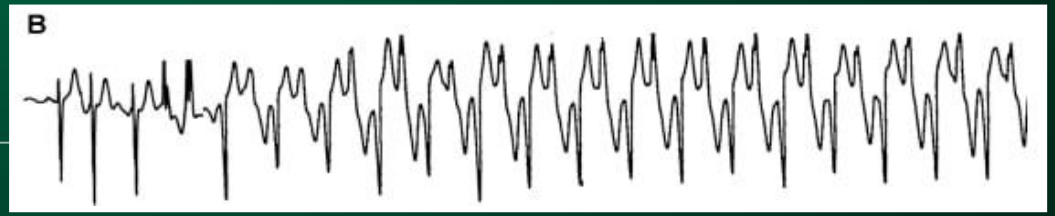
Drowning/near drowning

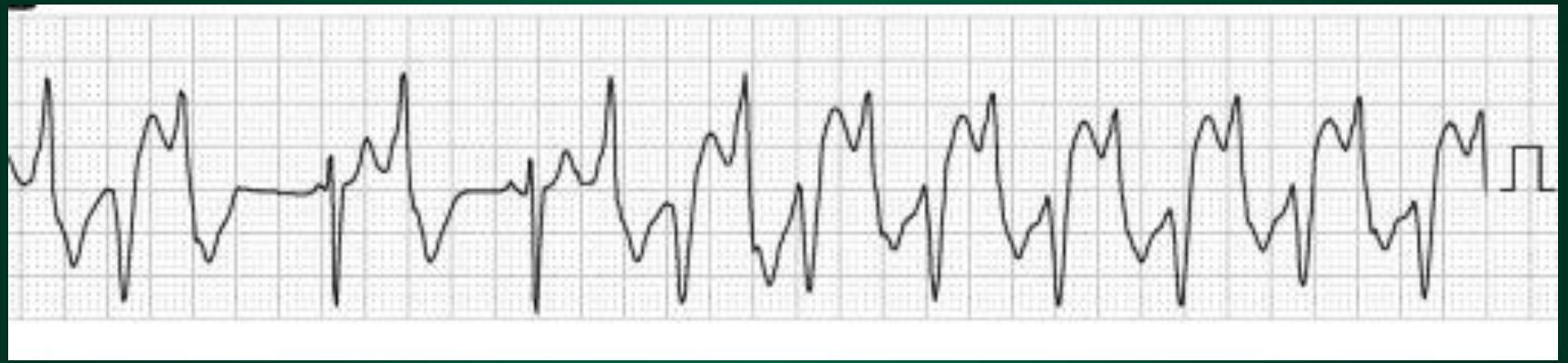
Family history of CPVT, seizure/syncope/SCD

EKG

Normal resting EKG

Exercise-induced bidirectional VT

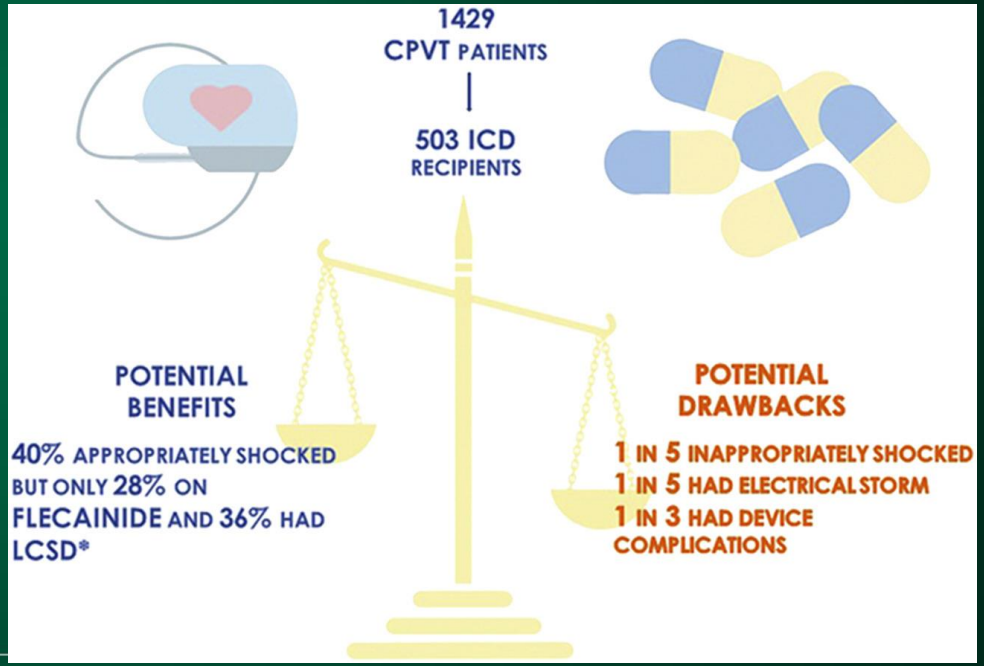




CPVT

Treatment

- Avoiding competitive sports
- Beta blockade
- ICD



Heart Rhythm. 2018

Wolff-Parkinson-White Syndrome

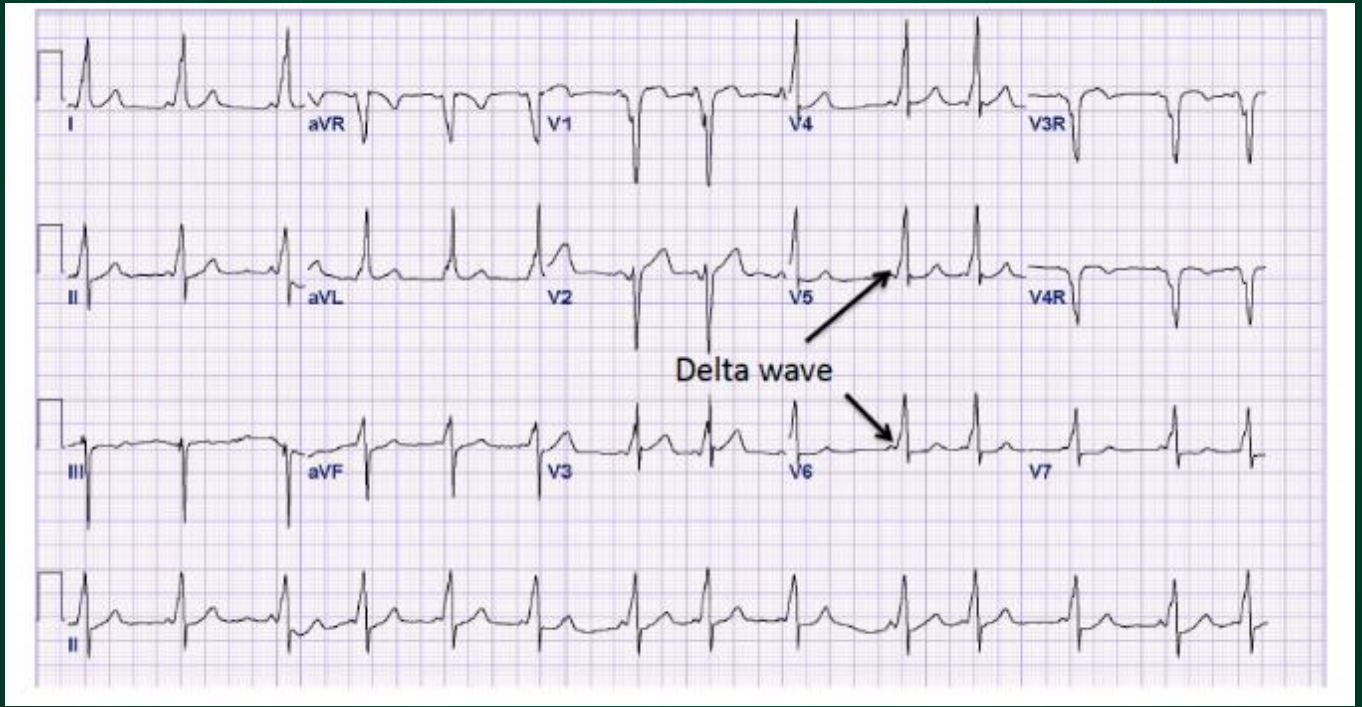
Cause

- Extra electrical pathway through the heart
- Can result in a very rapid heart rate
- Short PR <120 ms, delta wave, prolonged QRS ≥120ms

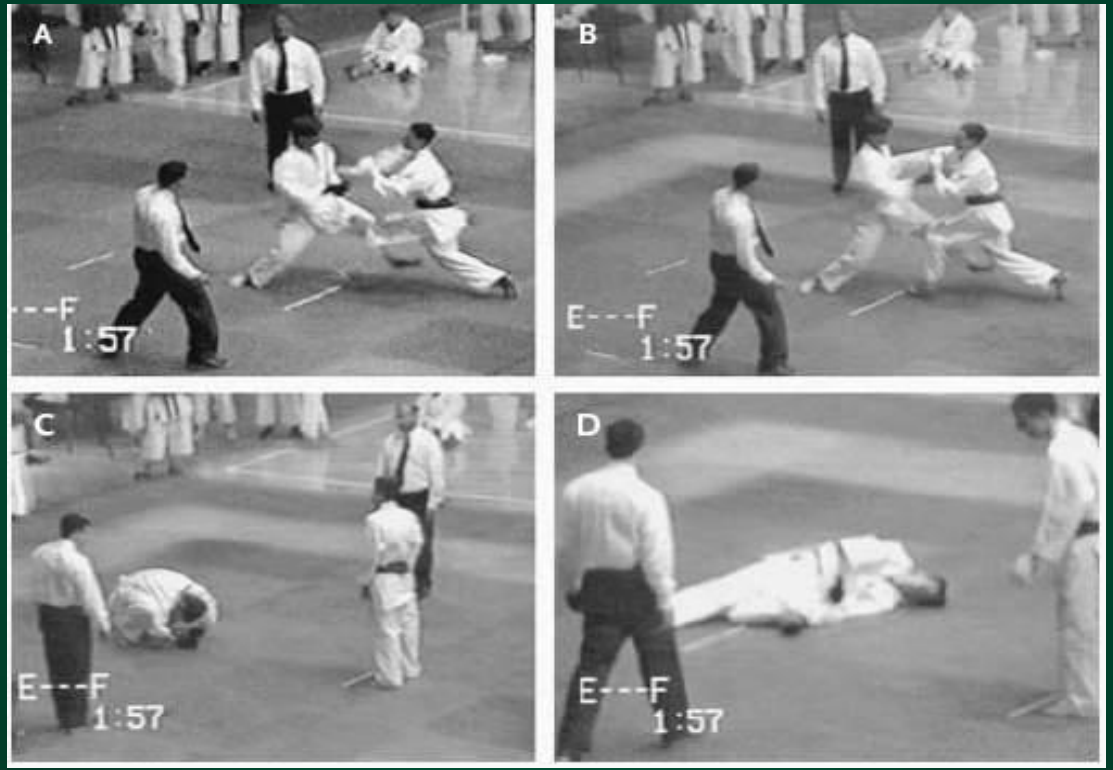
Wolff-Parkinson-White Syndrome

Evaluation

- Exercise EKG
- EP study
- Echocardiogram (Ebstein's anomaly and cardiomyopathy)


I
aVR
V1
V4
V3R
II
aVL
V2
V5
V4R
Delta wave
III
aVF
V3
V6
V7
II

Commotio Cordis



tp://www.westhertshospitals.nhs.uk/whc/archive/evidence/07%20arrhythmias/Sudden%20death%20in%20athletes-review-NEJM%202003.pdf

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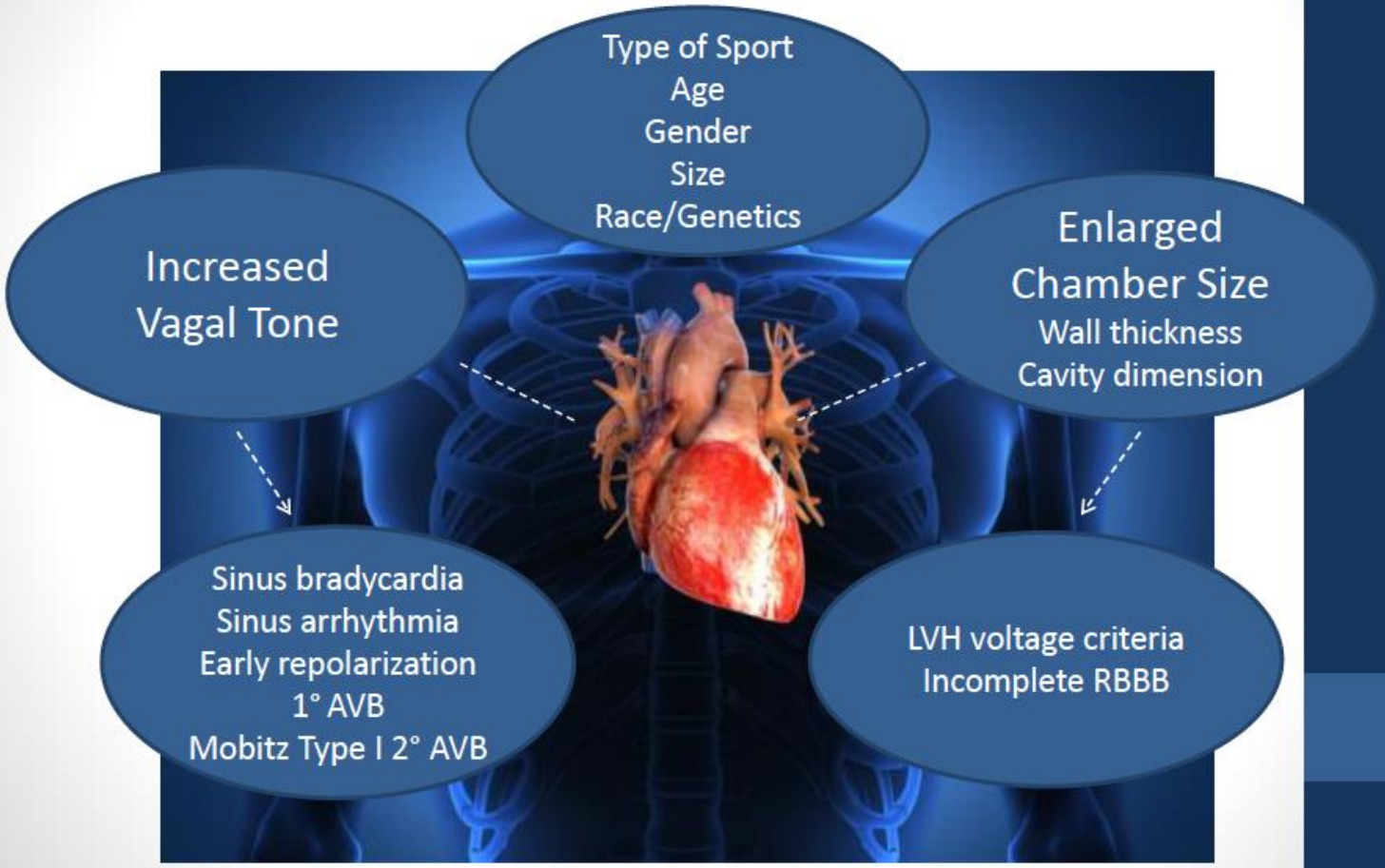
International Criteria

The “normal” athlete’s heart

- Guidelines to identify EKG changes that occur with physical conditioning
- Eliminate unnecessary further testing

Physiologic Cardiac Adaptation: 'Athlete's Heart'



***Drezner, J. AMSSM 2017**

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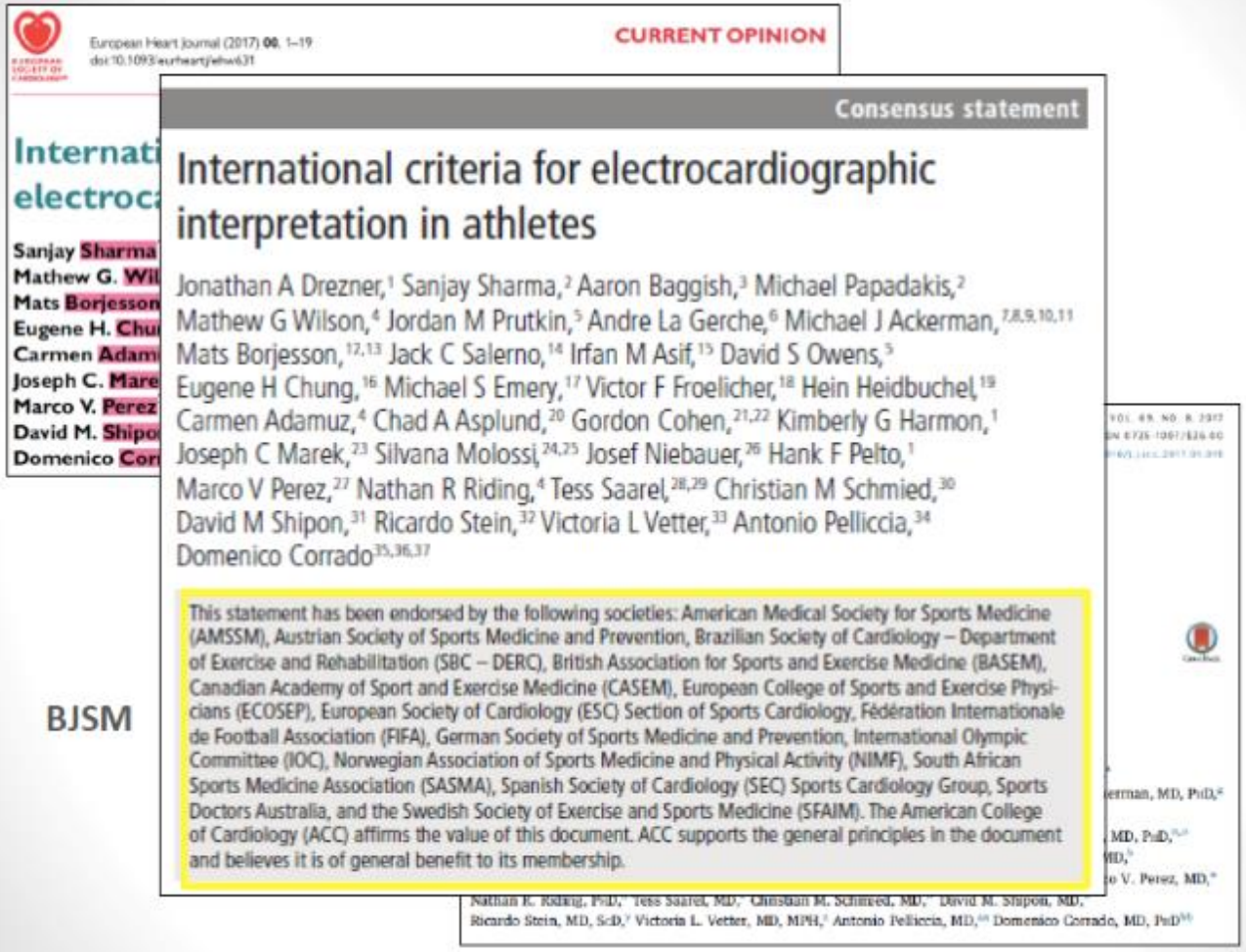

European Heart Journal (2017) 00, 1–19
doi:10.1093/eurheart/ehw631

CURRENT OPINION

Consensus statement

International criteria for electrocardiographic interpretation in athletes

Jonathan A Drezner,[1] Sanjay Sharma,[2] Aaron Baggish,[3] Michael Papadakis,[2] Mathew G Wilson,[4] Jordan M Prutkin,[5] Andre La Gerche,[6] Michael J Ackerman,[7,8,9,10,11] Mats Borjesson,[12,13] Jack C Salerno,[14] Irfan M Asif,[15] David S Owens,[5] Eugene H Chung,[16] Michael S Emery,[17] Victor F Froelicher,[18] Hein Heidbuchel,[19] Carmen Adamuz,[4] Chad A Asplund,[20] Gordon Cohen,[21,22] Kimberly G Harmon,[1] Joseph C Marek,[23] Silvana Molossi,[24,25] Josef Niebauer,[26] Hank F Pelto,[1] Marco V Perez,[27] Nathan R Riding,[4] Tess Saarel,[28,29] Christian M Schmied,[30] David M Shipon,[31] Ricardo Stein,[32] Victoria L Vetter,[33] Antonio Pelliccia,[34] Domenico Corrado[35,36,37]

This statement has been endorsed by the following societies: American Medical Society for Sports Medicine (AMSSM), Austrian Society of Sports Medicine and Prevention, Brazilian Society of Cardiology – Department of Exercise and Rehabilitation (SBC – DERC), British Association for Sports and Exercise Medicine (BASEM), Canadian Academy of Sport and Exercise Medicine (CASEM), European College of Sports and Exercise Physicians (ECOSEP), European Society of Cardiology (ESC) Section of Sports Cardiology, Fédération Internationale de Football Association (FIFA), German Society of Sports Medicine and Prevention, International Olympic Committee (IOC), Norwegian Association of Sports Medicine and Physical Activity (NIMF), South African Sports Medicine Association (SASMA), Spanish Society of Cardiology (SEC) Sports Cardiology Group, Sports Doctors Australia, and the Swedish Society of Exercise and Sports Medicine (SFAIM). The American College of Cardiology (ACC) affirms the value of this document. ACC supports the general principles in the document and believes it is of general benefit to its membership.

BJSM

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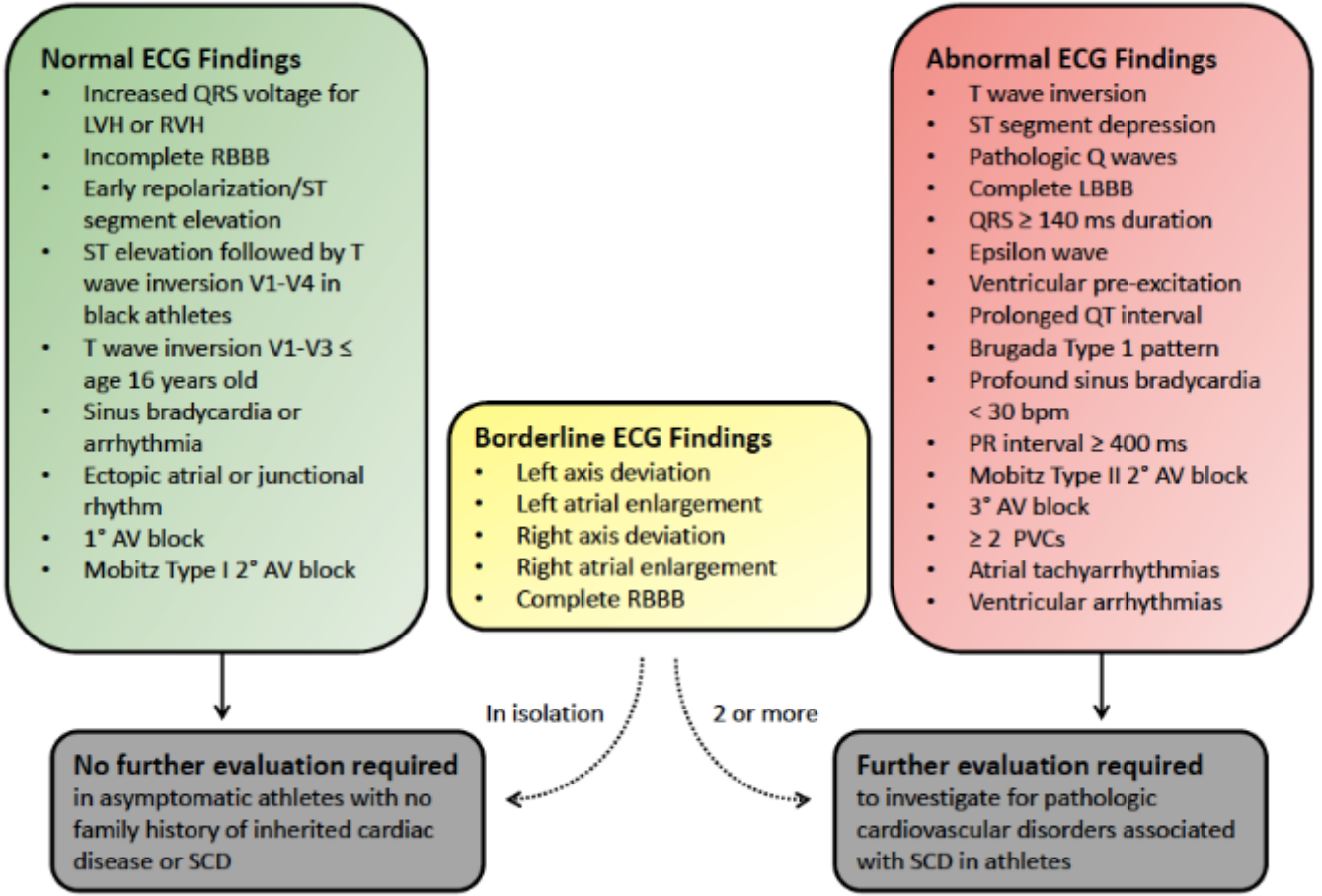
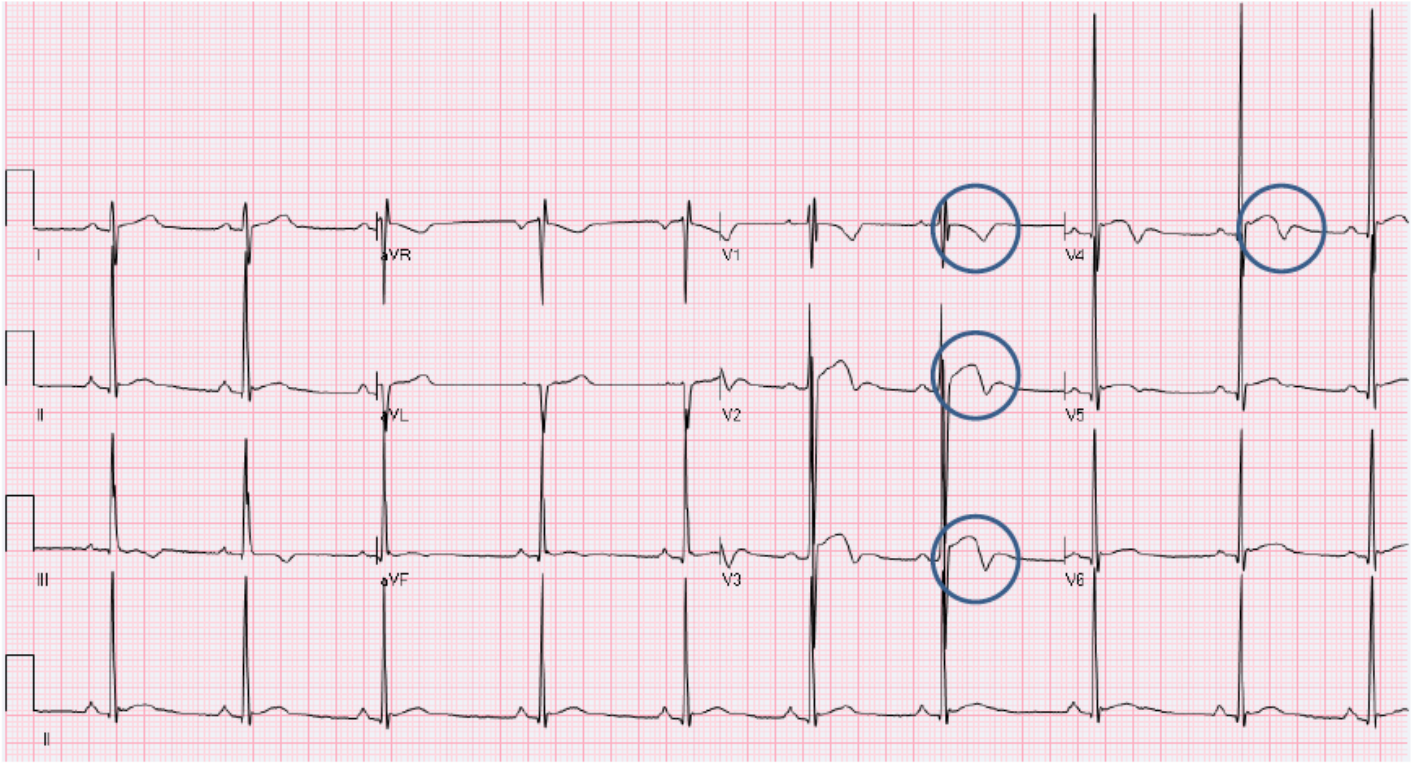
International Criteria for ECG Interpretation in Athletes



Normal Variant: Repolarization Changes in Black/African Athletes



ECG from a 24 year old asymptomatic black/African soccer player demonstrating J-point elevation, convex ('domed') ST elevation followed by T-wave inversion in leads V1-V4 (circles). This is a normal repolarization pattern in black/African athletes.

***Drezner, J. AMSSM 2017**

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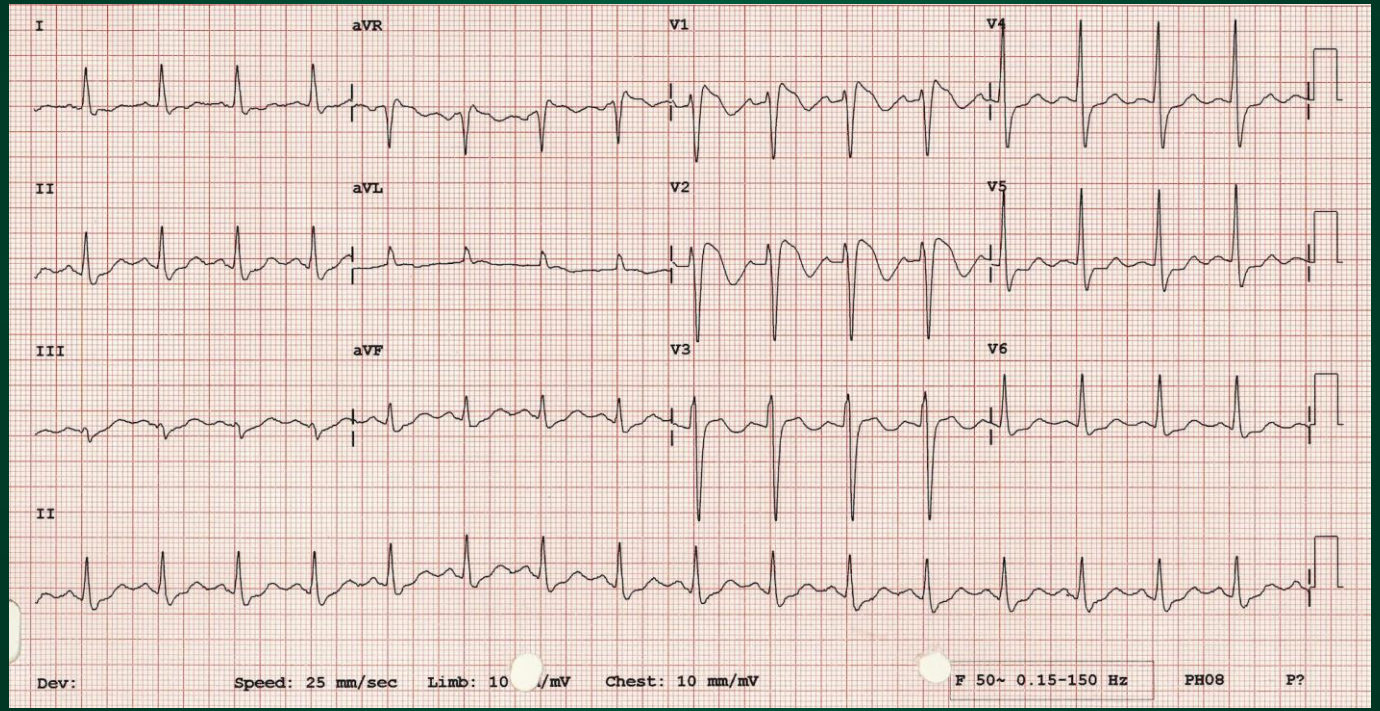
Brugada – abnormal



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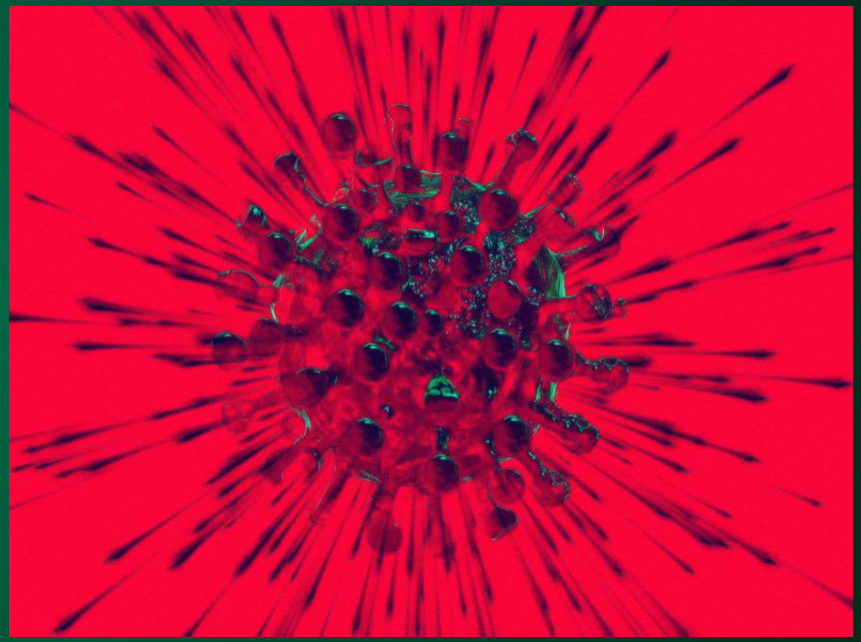
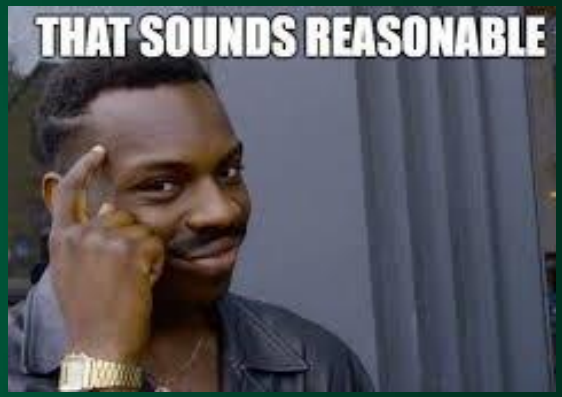
Excavation Plan

1. “Lay of the Land” – Background, Incidence, Guidelines, etc.
2. Particularly Bad Landmines – Causes of Sudden Cardiac Death in Young Athletes
3. Pre-participation Screening in the Time of COVID



COVID Guidelines: A Journey



Histrionic initial reactions

Data gathering

Current state

What is the worry about COVID and the heart?

Myocarditis – inflammatory disease of the cardiac muscle, often viral in etiology

- Presentation is variable – from fulminant to subclinical
- In pediatrics, fulminant patients can often make a quick and full recovery

Definitive diagnosis requires biopsy (Dallas Criteria) showing inflammatory cell infiltration with myocyte damage

- Cardiac MRI has become an accepted surrogate marker, with specific criteria being more accepted in adults > children
- Lake Louise criteria (involving T1/T2 mapping and LGE)

Clinical myocarditis – signs of heart failure associated with abnormalities on EKG/Echo/MRI and laboratory signs of inflammation and myocardial injury (elevated CRP/troponin/BNP)

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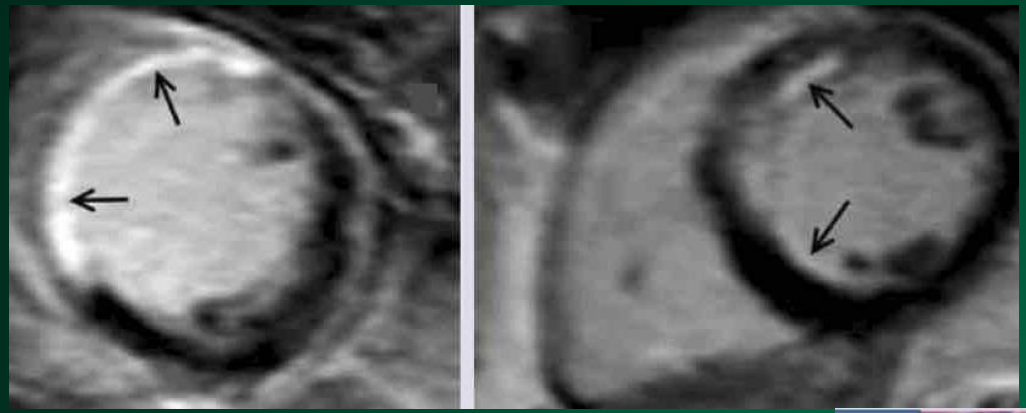

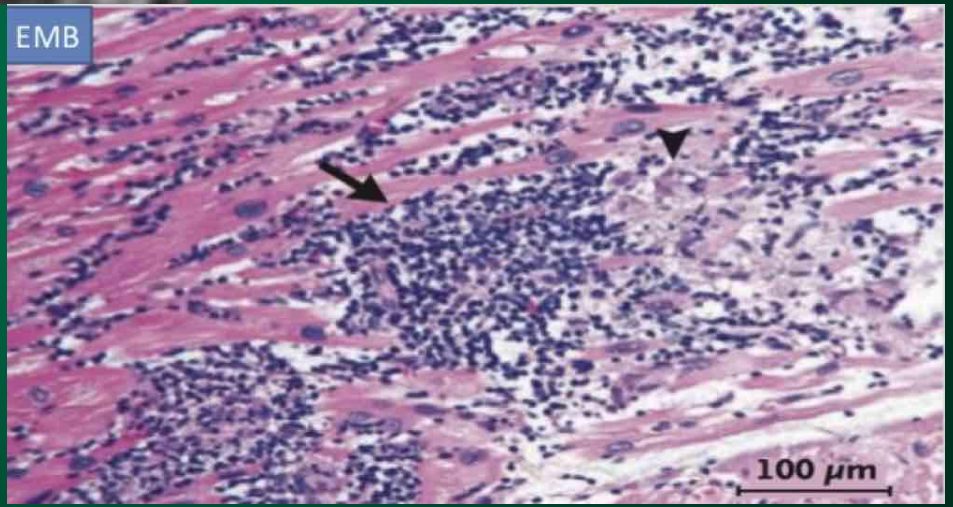

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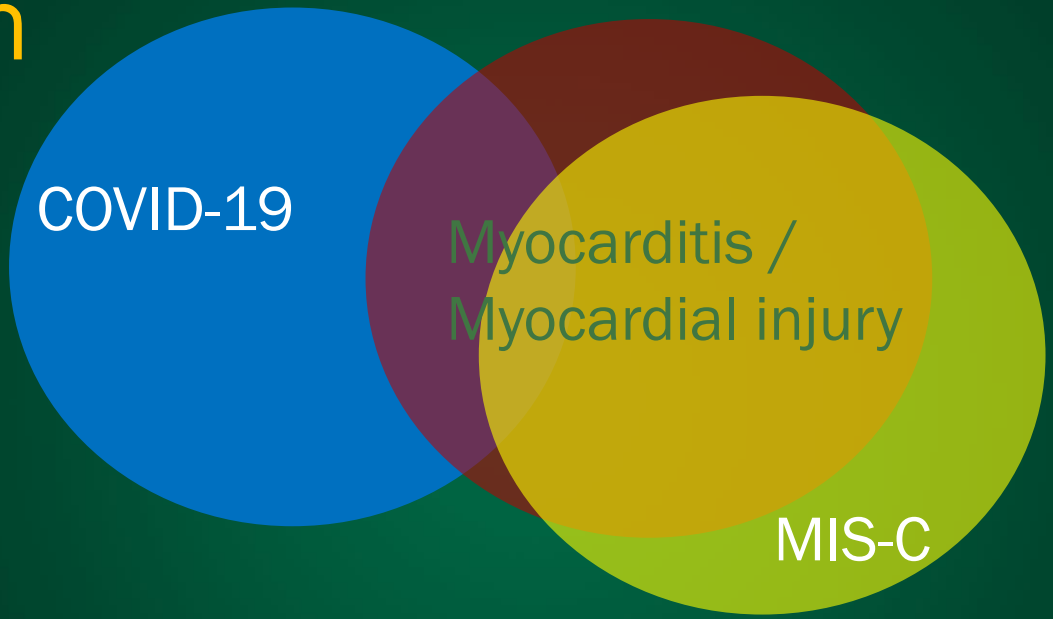
Children

Adults



How do COVID-19, myocardial injury, and MIS-C relate?

JAMA Cardiology | Original Investigation

Outcomes of Cardiovascular Magnetic Resonance Imaging in Patients Recently Recovered From Coronavirus Disease 2019 (COVID-19)

Valentina O. Puntmann, MD, PhD; M. Ludovica Carerj, MD; Imke Wieters, MD; Masia Fahim; Christophe Arendt, MD; Jedrzej Hoffmann, MD; Anastasia Shchendrygina, MD, PhD; Felicitas Escher, MD; Mariuca Vasa-Nicotera, MD; Andreas M. Zeiher, MD; Maria Vehreschild, MD; Eike Nagel, MD

- Prospective observational cohort study
- April – June 2020
- N = 100 adults, ~ 2 months after COVID-19
- Compared to risk-factor matched/non-COVID control group
- All “symptomatic” - 67% recovered at home, 33% hospitalized

CINE

Puntmann VO. JAMA Cardiology 2020

Table 1. Patient Characteristics, Cardiac Magnetic Resonance (CMR) Imaging Findings, and Blood Test Results on the Day of CMR Examination

Characteristic	COVID-19 (n = 100)	Risk factor–matched controls (n = 57)	*P* value[a]
Patient characteristics			
Age, mean (SD), y	49 (14)	49 (13)	.91
Male, No. (%)	53 (53)	28 (49)	.88
BMI, median (IQR)[b]	25 (23-28)	27 (23-29)	<.001
Hypertension, No. (%)	22 (22)	14 (25)	.003
Diabetes, No. (%)	18 (18)	12 (22)	.002
Hypercholesterolemia, No. (%)	22 (22)	13 (23)	.02
Known CAD, No. (%)	13 (13)	9 (16)	.02
Smoking, No. (%)	22 (22)	11 (19)	.54
COPD or asthma, No. (%)	21 (21)	13 (23)	.002

Conclusions and Caveats

Cardiac “involvement” in 73% of adult patients recovering from COVID-19, independent of severity of presentation

Yikes

Caveats!

- **The “risk-factor matched controls” had some MRI changes as well**
- **This study is not applicable to pediatric patients**
- **Also not applicable to asymptomatic patients**

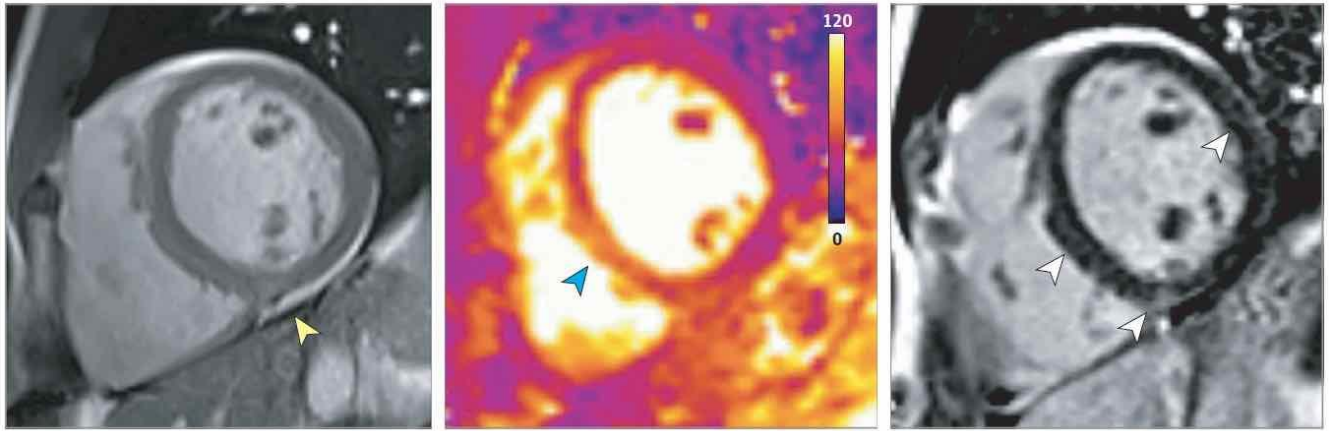
RESEARCH LETTER

Cardiovascular Magnetic Resonance Findings in Competitive Athletes Recovering From COVID-19 Infection

Myocarditis is a significant cause of sudden cardiac death in competitive athletes and can occur with normal ventricular function.[1] Recent studies have raised concerns of myocardial inflammation after recovery from coronavirus disease 2019 (COVID-19), even in asymptomatic or mildly symptomatic patients.[2] Our objective was to investigate the use of cardiac magnetic resonance (CMR) imaging in competitive athletes recovered from COVID-19 to detect myocardial inflammation that would identify high-risk athletes for return to competitive play.

Methods | We performed a comprehensive CMR examination including cine, T1 and T2 mapping, extracellular volume fraction, and late gadolinium enhancement (LGE), on a 1.5-T scanner (Magnetom Sola; Siemens Healthineers) using standardized protocols,[3] in all competitive athletes referred to the sports medicine clinic after testing positive for COVID-19 (reverse transcriptase–polymerase chain reaction) between June and August 2020. The Ohio State University institutional review board approved the study, and informed consent in writing was obtained from participating athletes. Cardiac magnetic resonance imaging was performed after recommended quarantine (11-53 days). Electrocardiogram, serum troponin I, and transthoracic echocardiogram were performed on day of CMR imaging.

Figure. Cardiovascular Magnetic Resonance Findings in Competitive Athletes Recovering From Coronavirus Disease 2019 Infection



Rajpal S. JAMA Cardiology 2020

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JAMA Cardiology | Original Investigation

Prevalence of Inflammatory Heart Disease Among Professional Athletes With Prior COVID-19 Infection Who Received Systematic Return-to-Play Cardiac Screening

Matthew W. Martinez, MD; Andrew M. Tucker, MD; O. Josh Bloom, MD, MPH; Gary Green, MD; John P. DiFiori, MD; Gary Solomon, PhD; Dermot Phelan, MD, PhD; Jonathan H. Kim, MD, MSc; Willem Meeuwisse, MD, PhD; Allen K. Sills, MD; Dana Rowe, BA; Isaac I. Bogoch, MD; Paul T. Smith, MD; Aaron L. Baggish, MD; Margot Putukian, MD; David J. Engel, MD

Study

- In May 2020, U.S. professional sports leagues adopted "Return to Play" rules in line with ACC Guidelines at the time
- Cross-sectional study of this cardiac testing on n = 789 professional athletes s/p COVID-19

Testing

- Troponin, EKG, echocardiogram on all patients
- Abnormal screening → stress testing, cMRI

JAMA Cardiology. 2021

Table 1. Return-to-Play Cardiac Screening in Professional Athletes Testing Positive for Coronavirus Disease 2019 (COVID-19)

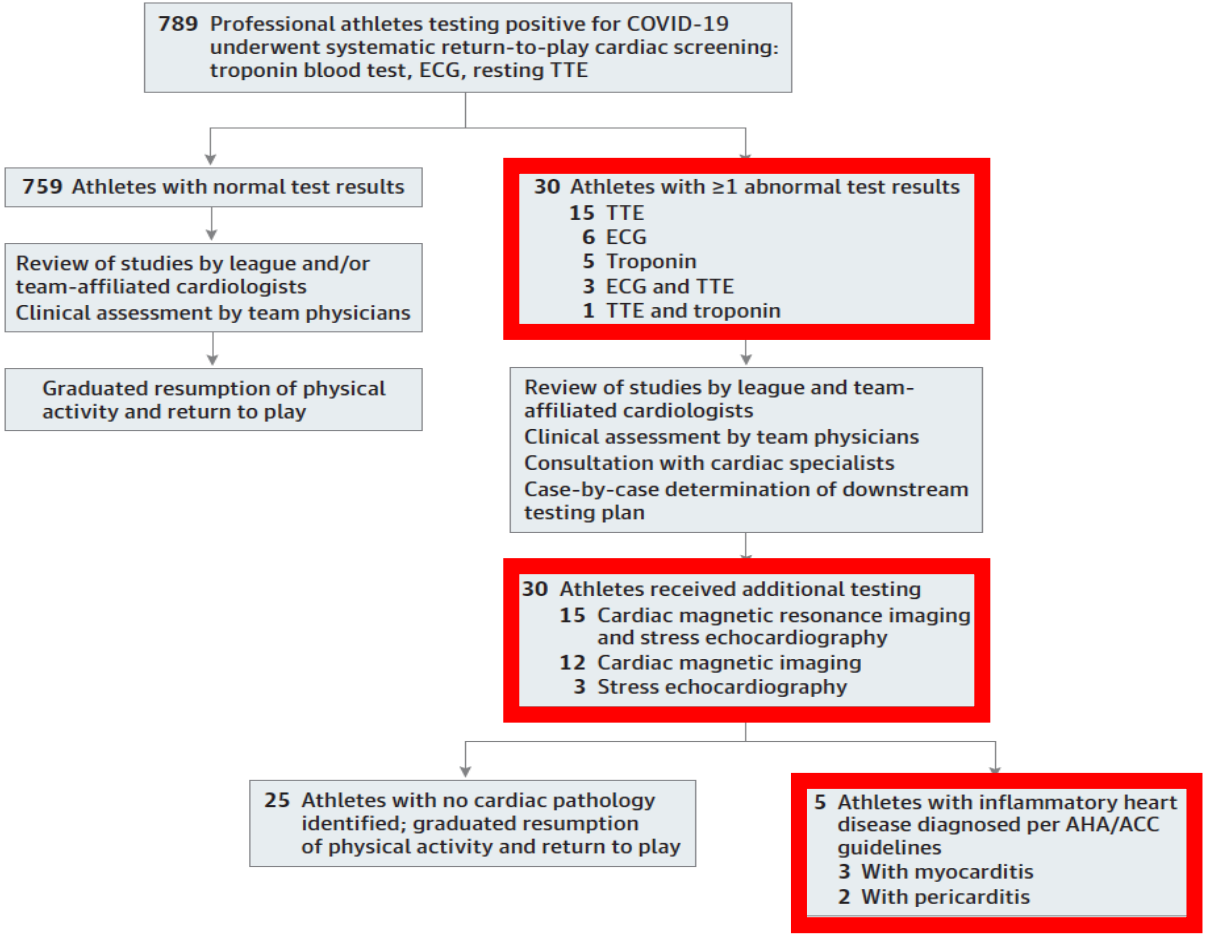
Characteristic	Individuals, No. (%)						
	National Basketball Association		Major League Soccer	Major League Baseball	National Hockey League	National Football League	Total professional athlete cohort
	Men	Women					
Total athletes positive for COVID-19, No.	109	12	70	181	68	349	789
Age, mean (range), y	25 (19-35)	27 (21-33)	25 (18-31)	25 (19-38)	25 (19-41)	25 (21-37)	25 (19-41)
COVID-19 symptom burden							
Preceding viral symptoms	71 (65.1)	8 (67)	33 (47)	109 (60.2)	51 (75)	188 (53.9)	460 (58.3)
Asymptomatic or paucisymptomatic	38 (34.9)	4 (33)	37 (53)	72 (39.8)	17 (25)	161 (46.1)	329 (41.7)
COVID-19 test							
Polymerase chain reaction	75 (68.8)	9 (75)	51 (73)	172 (95.0)	54 (79)	226 (64.8)	587 (74.4)
Antibody	34 (31.2)	3 (25)	19 (27)	9 (5.0)	14 (21)	123 (35.2)	202 (25.6)
Days between COVID-19 polymerase chain reaction test and cardiac screen, mean (range)	32 (9-124)	23 (14-60)	14 (13-16)	21 (3-90)	18 (9-97)	17 (3-156)	19 (3-156)
Abnormal cardiac testing results							
Troponin[a]	3 (2.8)	1 (8)	1 (1)	0	0	1 (0.3)	6 (0.8)
Electrocardiogram[b]	0	0	0	5 (2.8)	0	5 (1.4)	10 (1.3)
Echocardiogram[c]	2 (1.8)	3 (25)	3 (4)	6 (3.3)	1 (1)	5 (1.4)	20 (2.5)

[a] An abnormal troponin level was defined as a level greater than the 99th percentile of the reference laboratory value.

[b] An abnormal electrocardiogram was defined as meeting international recommendations[9] and demonstrating findings raising concern for potential acute cardiac injury.

[c] An abnormal echocardiogram was defined by ventricular dysfunction or another finding raising concern for potential acute cardiac injury.

Figure. Flow Diagram of the Systematic Return-to-Play Cardiac Screening Process Used for Professional Athletes Testing Positive for Coronavirus Disease 2019 (COVID-19)



Thirty of 789 athletes (3.8%) had abnormal cardiac screening test results necessitating additional evaluation and downstream testing; 5 athletes (0.6%) were detected to have findings raising concern for COVID-19–associated inflammatory heart disease that resulted in restriction from sport participation per American Heart Association (AHA)/American College of Cardiology (ACC) guidelines.[10]
ECG indicates electrocardiogram; TTE, transthoracic echocardiogram.

CONCLUSIONS AND RELEVANCE This study provides large-scale data assessing the prevalence of relevant COVID-19–associated cardiac pathology with implementation of current RTP screening recommendations. While long-term follow-up is ongoing, few cases of inflammatory heart disease have been detected, and a safe return to professional sports activity has thus far been achieved.

JAMA Cardiol. doi:10.1001/jamacardio.2021.0565

5 out of 789 players = 0.6%

Cardiac Screening and Return-to-Play following COVID-19 Infection

- All student-athletes diagnosed with a COVID-19 (SARS-CoV-2) will require isolation for 10 days with day 0 starting at the onset of symptoms or the day of testing, if asymptomatic.
- No exercise during the isolation period.
    - After the isolation period is completed, each student-athlete will undergo a medical evaluation by a team physician.
- Cardiac testing and a period of re-acclimation to exercise required prior to returning to full participation in sport.

The required cardiac testing will include:

1. Electrocardiogram (EKG)
2. Serum Troponin level
3. Echocardiogram (ECHO)

The results of these tests, medical evaluation findings, or the clinical course of the student- athlete (i.e. moderate to severe infections requiring hospitalization) may warrant further testing (such as cardiac MRI) based on the discretion of the team physician.

In addition to cardiac testing, a minimum of a 4-day period of re-acclimation to exercise will be required to monitor for any signs or symptoms of cardiac complications (i.e. chest pain, shortness of breath, presyncope, syncope). Day 1 of re-acclimation should be approximately 25% of a normal practice or conditioning session, with Day 2 being 50%, Day 3 being 75% and Day 4 being full participation.

UAB Sports and COVID-19

N = 184 UAB athletes screened to date

Football, basketball, baseball, volleyball, soccer, track, cross-country, tennis, golf, and yes . . . bowling

Patients

- S/p COVID-19, now asymptomatic, at least 10 days after infection
- Everyone receives COVID-19 IgG testing prior to clearance regardless

Clearance to play

- Normal troponin, EKG, and echocardiogram
- Cleared by team physician

No disqualifications to date

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Table 2 Difference between adults and children with COVID-19

	Adults	Children
Rates of infection	High	Low
Evidence	Chinese Center for Disease Control and Prevention reported only 2% of patients younger than 20 years of age among 44,672 cases [20] United States Center for Disease Control and Prevention (CDC) reported only 1.7% children less than 18 years of age among 149,082 reported cases [7]	
Severity of illness	High	Low
Evidence	*Chinese data:* 14% of adult patients classified as 'severe' and 5% as 'critical' disease [20] *Italian data:* 25% of adult patients classified as 'severe' and 5% as 'critical' [6]	*Chinese data:* 5% of pediatric patients classified as 'severe' and < 1% as 'critical' disease [36] *Italian data:* 2% of pediatric patients classified as 'severe' or 'critical' disease [5]
Complications and mortality	High	Low
Evidence	*United States data:* Among 2634 adults hospitalized adults in 12 hospitals in New York City, 14% required intensive care and 12% received mechanical ventilation with a case fatality rate of 21% [4] *Chinese data:* Case fatality 2.3% of overall COVID-19 affected population [20] *Italian data:* Case fatality of overall COVID-19 affected population 7% [6]	*United States data:* Among 46 North American Pediatric ICUs, only 48 children were admitted to intensive care with a case fatality rate of 4.2% [3] *Chinese data:* 1 death [36] *Italian data:* No deaths [5]
Potential risk factors leading to severe disease	Hypertension, diabetes, and obesity [4]	Infants < 1 years of age, medical complexity[a], immune suppression, obesity [3, 5]

[a]Defined as dependence on technological support (tracheostomy with or without ventilator dependence) in association with developmental delay and/or genetic anomalies

WHO case definition Multisystem Inflammatory Syndrome Children – need all 5 criteria

1. Fever for ≥3 days

2. Clinical signs of multisystem involvement (at least 2 of the following):
   - Rash, bilateral nonpurulent conjunctivitis, or mucocutaneous inflammation signs
   - Hypotension or shock
   - **CV dysfxn, pericard-/valvul-/myocarditis, coronary changes, abnormal trop/BNP**
   - Evidence of coagulopathy
   - Acute gastrointestinal symptoms

3. Elevated markers of inflammation (eg, ESR, CRP, or procalcitonin)

4. No other obvious microbial cause of inflammation

5. Evidence of SARS-CoV-2 infection (any of the following):
   - Positive SARS-CoV-2 RT-PCR
   - Positive serology
   - Positive antigen test
   - Contact with an individual with COVID-19

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American Academy of Pediatrics

DEDICATED TO THE HEALTH OF ALL CHILDREN®

COVID-19 Interim Guidance: Return to Sports

Critical Updates on COVID-19 / Clinical Guidance / COVID-19 Interim Guidance: Return to Sports

COVID(+), either asymptomatic or mildly symptomatic (< 4 days fever, short duration of mild sx)

*See PCP who performs H&P

-If possible CV symptoms or exam findings, refer to cardiology

-If screening normal, can return to gradual play after 10 days from infection

-No mandatory EKG

https://services.aap.org/en/pages/2019-novel-coronavirus-covid-19-infections/clinical-guidance/covid-19-interim-guidance-return-to-sports/

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COVID-19 Interim Guidance: Return to Sports

Critical Updates on COVID-19 / Clinical Guidance / COVID-19 Interim Guidance: Return to Sports

COVID(+), with moderate initial sx (>4 days of sx or non-ICU hospital stay.
No MIS-C

*Cardiology consult is recommended at minimum 10 days s/p (+) test result. No exercise until cleared
-Cardiologist will consider testing including labs, cMRI, stress testing
-If CV testing negative, can be allowed to return to play on similar schedule as the asymptomatic/mild sx patients

https://services.aap.org/en/pages/2019-novel-coronavirus-covid-19-infections/clinical-guidance/covid-19-interim-guidance-return-to-sports/

American Academy of Pediatrics

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COVID-19 Interim Guidance: Return to Sports

Critical Updates on COVID-19 / Clinical Guidance / COVID-19 Interim Guidance: Return to Sports

COVID(+), with severe symptoms (ICU, MIS-C)

*Cardiology consult is recommended and should be arranged prior to hospital discharge

-Recommend 3-6 months of exercise restriction

-Cardiologist will coordinate testing needed over time

https://services.aap.org/en/pages/2019-novel-coronavirus-covid-19-infections/clinical-guidance/covid-19-interim-guidance-return-to-sports/

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