Sudden Cardiac Death in the Young Athlete

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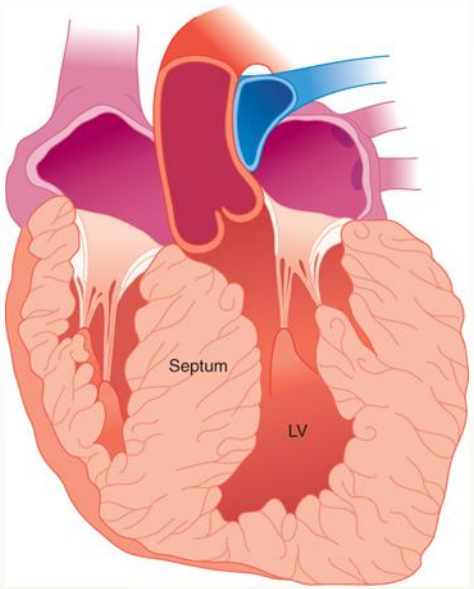
Commercial Interests/Financial disclosures

- https://www.youtube.com/watch?v=_TCbVIEtoiI&spfreload=10



Fabrice Muamba

- Retired English "soccer" player
- Played for Arsenal, Birmingham City, Bolton Wanders
- March 17th, 2012 he collapsed suffering cardiac arrest mid-game
- Resuscitated for 78 minutes
- Defibrillated multiples times
- Spent 1 month in the hospital recovering
- Diagnosed with HCM and had an ICD implanted
- Promptly retired

Outline

- Introduction
- Epidemiology
- Etiology
- AHH/ACC guidelines
- Red Flags
- Specific causes of SCD
- Covid-19
- EKG screening

Introduction

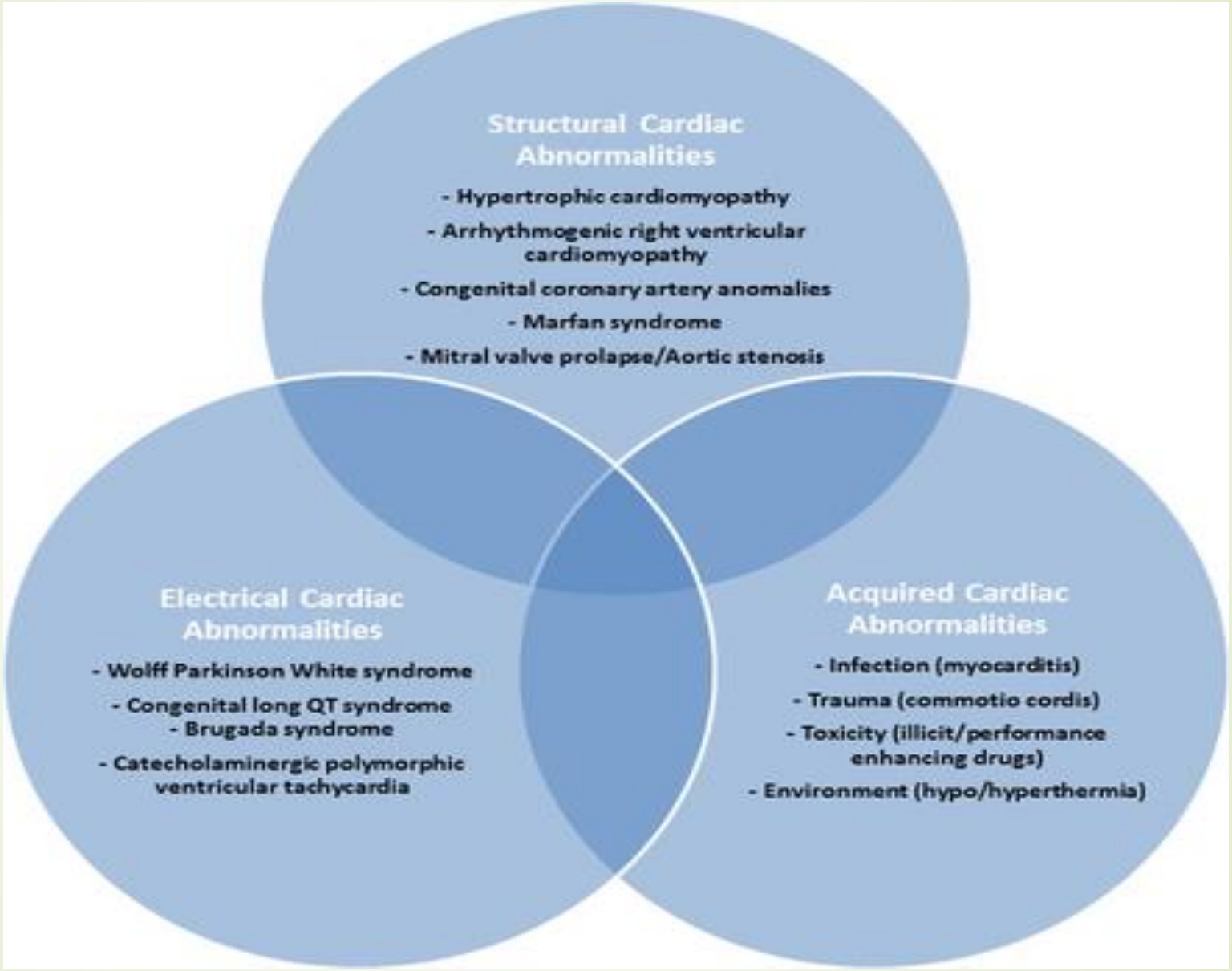
- Rare phenomenon, but generates a lot of **media attention**
- More common in athletes vs. non-athletes (2.8x increased risk)
  - Athlete= individual engaged in regular physical training and participating in official sports competition
- Ventricular arrhythmia (most classic substrate)
  - Dehydration
  - Hyperpyrexia
  - Electrolyte imbalances
  - Increased platelet aggregation

Epidemiology

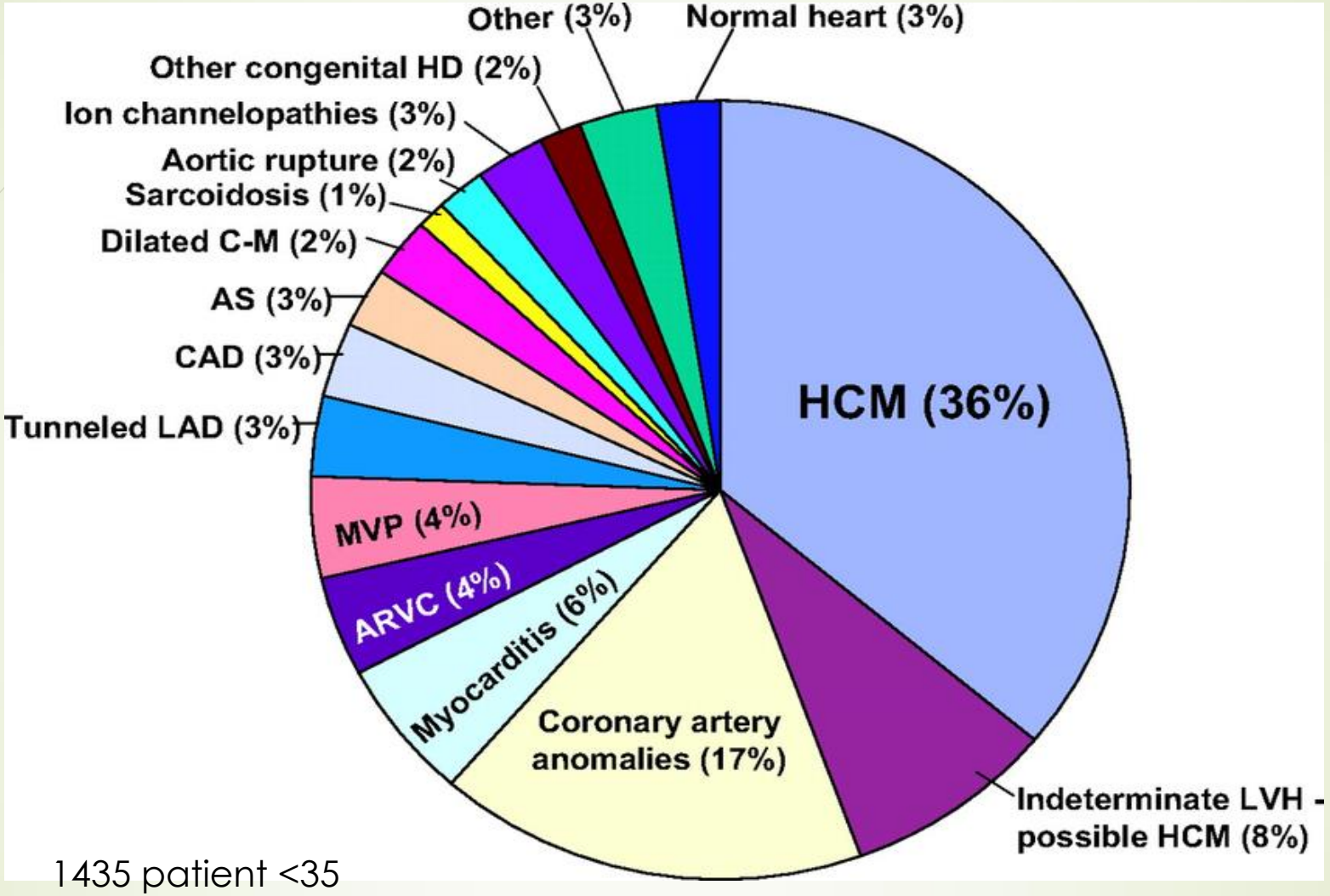
- Incidence 2.3-4.4-100,000 (varies a fair bit depending no which paper you cite)
- Males>Females
- African-American or Black athletes at highest ethnic risk (5.6 per 100,000)
  - Higher death rates from HCM
- Thought to be more common in high dynamic low isometric sports (i.e. football, **basketball**, soccer)
- Age >35 = 80% CAD
- >30% of athletes with SCD had documented symptoms such as chest pain, SOB, performance decline, palpitations, pre-syncope, or syncope

Epidemiology (NCAA athletes)

Characteristic	Increased Risk Group	Decreased Risk Group
Overall	1 in 53,703 athlete-years[14]	
Gender	Males: 1 in 37,790	Females: 1 in 121,593
Race	Black: 1 in 21,491	White: 1 in 68,354 Hispanic: 1 in 56,254
Sports	Men's Basketball: 1 in 8,978 Men's Soccer: 1 in 23,689 Men's Football: 1 in 35,951	N/A


Structural Cardiac Abnormalities
- Hypertrophic cardiomyopathy
- Arrhythmogenic right ventricular cardiomyopathy
- Congenital coronary artery anomalies
- Marfan syndrome
- Mitral valve prolapse/Aortic stenosis
Electrical Cardiac Abnormalities
- Wolff Parkinson White syndrome
- Congenital long QT syndrome
- Brugada syndrome
- Catecholaminergic polymorphic ventricular tachycardia
Acquired Cardiac Abnormalities
- Infection (myocarditis)
- Trauma (commotio cordis)
- Toxicity (illicit/performance enhancing drugs)
- Environment (hypo/hyperthermia)

Congenital/Genetic	
Structurally Abnormal Heart	**Structurally Normal Heart**
Hypertrophic cardiomyopathy	Congenital long QT syndrome
Arrhythmogenic right ventricular cardiomyopathy	Catecholaminergic polymorphic ventricular tachycardia
Dilated cardiomyopathy	Wolf-Parkinson-White syndrome or other accessory pathway
Other cardiomyopathy (i.e., left ventricular noncompaction)	Brugada syndrome
Congenital anomalies of coronary origin & course	Other ion channelopathies
Aortopathy (i.e., Marfan syndrome & ascending aortic aneurysm/ dissection)	
Valvular heart disease (i.e., congenital aortic stenosis, mitral valve prolapse)	
Acquired	
Structurally Abnormal Heart	**Structurally Normal Heart**
Atherosclerotic coronary artery disease	Commotio cordis
Kawasaki's disease	Acquired long QT (i.e., drug-induced)
Myocarditis	Other substance ingestion or environmental factors (i.e., hypo- or hyperthermia)



1435 patient <35
Maron, Thompson et al,
Circulation 2007

AHA/ACC 14 point *checklist* for pre-participation **sports** screening

- **Personal history:**
- Chest pain/discomfort/tightness/pressure related to exertion
- Unexplained syncope/near-syncope*
  - *Judged not to be of neurocardiogenic (vasovagal) origin; of particular concern when occurring *during* or *after* physical exertion.
- Excessive exertional and unexplained dyspnea/fatigue or palpitations, associated with exercise
- Prior recognition of a heart murmur
- Elevated systemic blood pressure
- Prior restriction from participation in sports
- Prior testing for the heart, ordered by a physician

- AAP checklist also includes: hx of HLP, heart infection, Kawasaki and unexplained seizure

AHA/ACC 14 point checklist for pre-participation sports screening

- **Family history:**
- Premature death (sudden and unexpected, or otherwise) before age 50 attributable to heart disease in ≥1 relative
- Disability from heart disease in close relative <50 y of age
- Hypertrophic or dilated cardiomyopathy, long-QT syndrome, or other ion channelopathies, Marfan syndrome, or clinically significant arrhythmias; specific knowledge of certain cardiac conditions in family members

- AAP additional questions: short QT syndrome, Brugada, CPVT. Heart problems, pacemakers or defibrillators. Fainting, unexplained seizures or near drowning.

AHA/ACC 14 point checklist for pre-participation sports screening

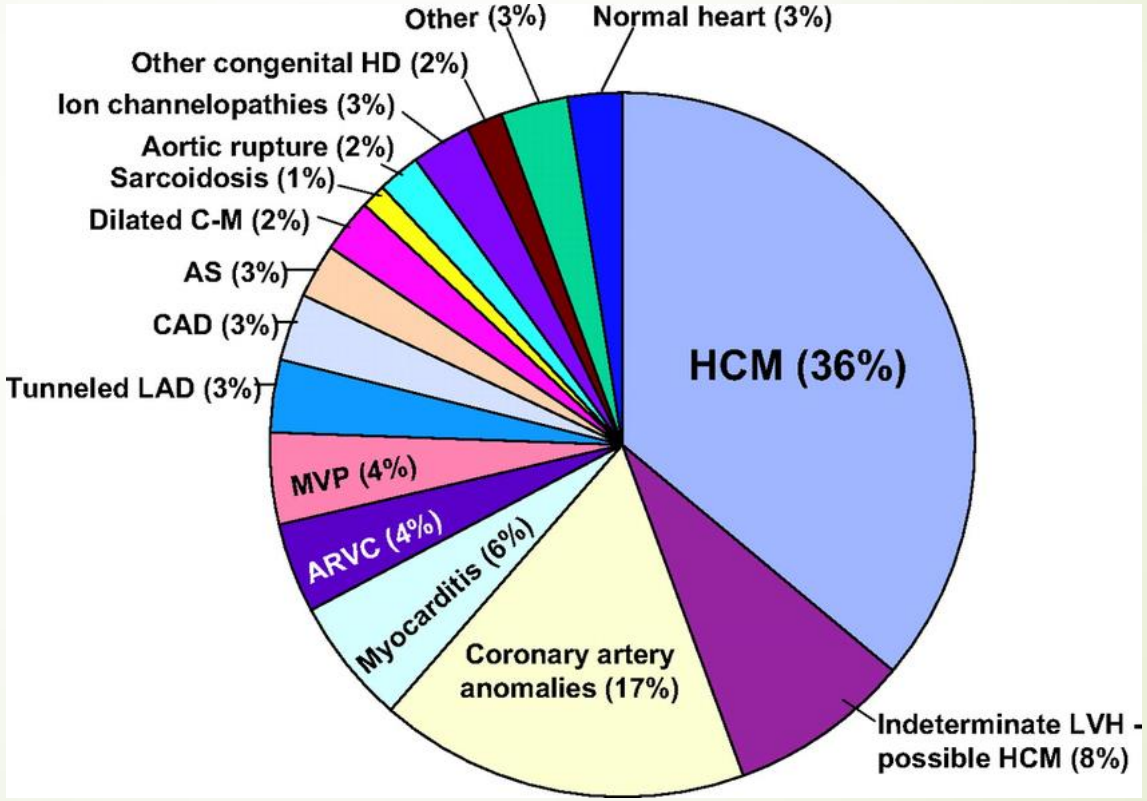
- **Physical examination:**
- Heart murmur**
  - **Refers to heart murmurs judged likely to be organic and unlikely to be innocent; auscultation should be performed with the patient in both the supine and standing positions (or with Valsalva maneuver), specifically to identify murmurs of dynamic left ventricular outflow tract
- Femoral pulses to exclude aortic coarctation
- Physical stigmata of Marfan syndrome (Ghent nosology)
- Brachial artery blood pressure (sitting position)***

  (***Preferably taken in both arms.)

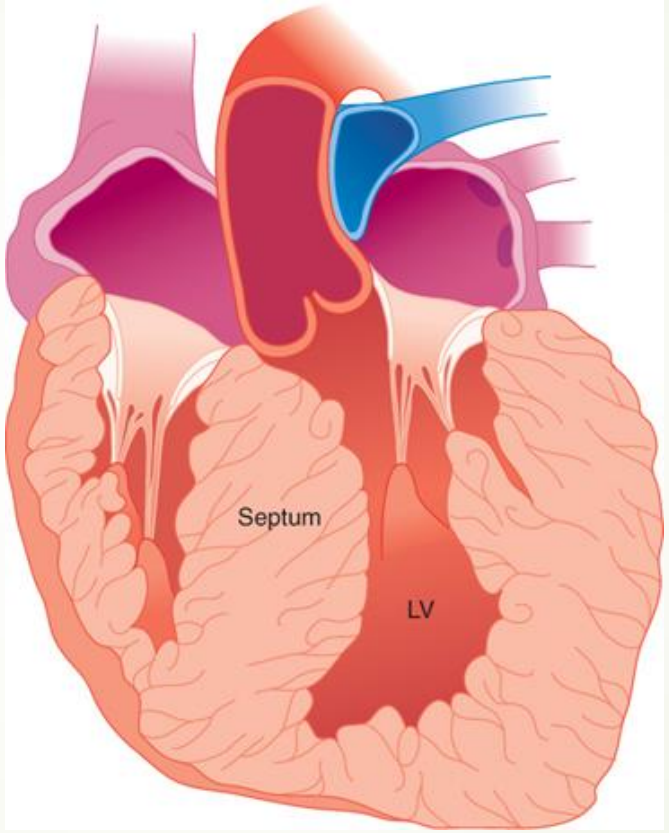
Red Flags (my personal)

- Exertional chest pain
- Chest pain secondary to a racing heart
- Syncope with exercise or shortly after
- Family history of SCD/unexplained deaths, cardiomyopathy, early pacemaker/ICD's, drownings, deafness, aortic dissection/aneurysm
- Self limiting behavior
- Decreased exercise tolerance

Specific causes of SCD

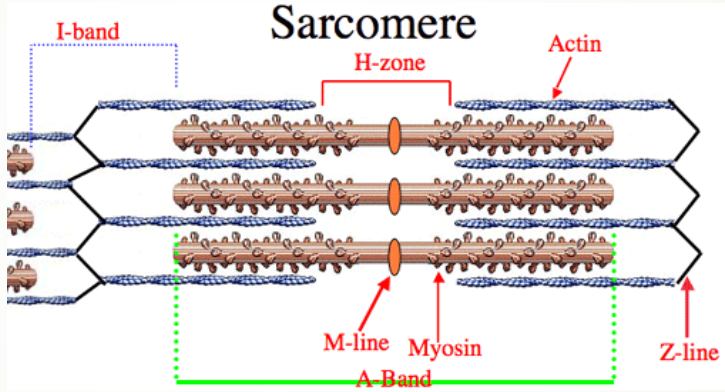


Hypertrophic Cardiomyopathy



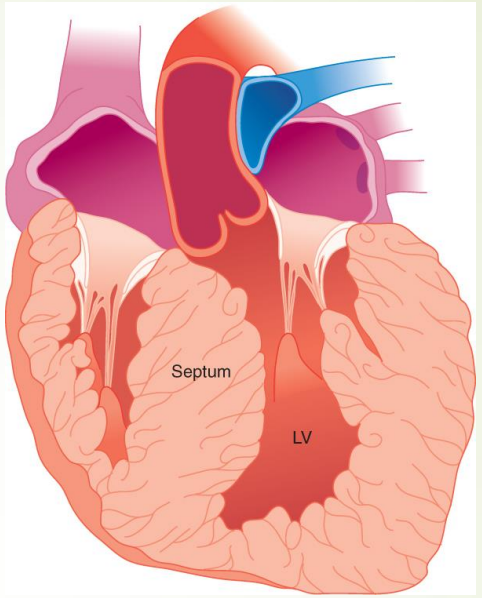
Introduction: Hypertrophic cardiomyopathy

- Heart disease caused by any one of several gene mutations coding for the **sarcomere**



- *Significant diversity in phenotypic expression and clinical course*
- >1400 mutations have been identified

Introduction



- HCM patients typically develop:
  - LV outflow tract obstruction
  - Diastolic dysfunction
  - Myocardial ischemia
  - Mitral regurgitation
  - Systolic dysfunction (end stage or “burned out”)

Epidemiology

- Likely an underestimate (clinically silent may be missed)
- Annual incidence is 0.3-0.5 cases per 100,000 *children*
- Prevalence 1:500 in the general population
- Peak incidence in infants <1 year old
- Slight male predominance
- Higher in African American children than White/Hispanic
- Accounts for 25-40% of all pediatric cardiomyopathies (most common)

Symptoms

- Presenting symptoms may include:
  - Chest pain
  - Presyncope/syncope
  - Palpitations
  - Nausea/poor appetite
  - Tachypnea
  - Easy fatigability
  - Sudden cardiac arrest
  - Death

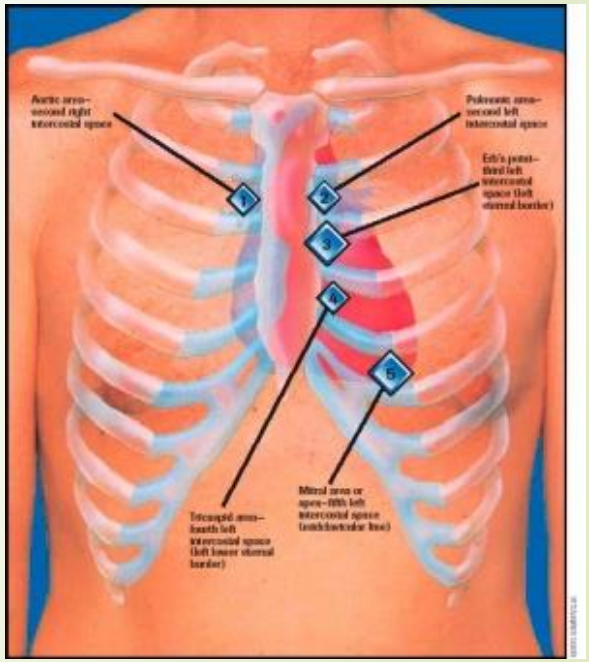
Epidemiology of cardiomyopathy - A clinical and genetic study of hypertrophic cardiomyopathy: The EPOCH-H study

Characteristics	Cases, *n* (%)
Symptomatic, *n* (%)	
Chest pain	36 (64.3)
Shortness of breath	35 (62.5)
Palpitation	30 (53.5)
Syncope	12 (21.4)
Presyncope	04 (07.1)
HNCM/HOCM ratio	2:1 (39/20)
Asymmetrical HCM	19 (32.2)
NYHA (III and IV)	14 (23.7)
Sudden death (*n*)	02 (3.4)
Undergone surgery/alcohol ablation/recommended ICD	13 (22.1)

HCM: Hypertrophic cardiomyopathy, HOCM: Hypertrophic obstructive cardiomyopathy, HNCM: Hypertrophic nonobstructive cardiomyopathy, NYHA: New York Heart Association, ICD: Implantable cardioverter defibrillator

Physical exam

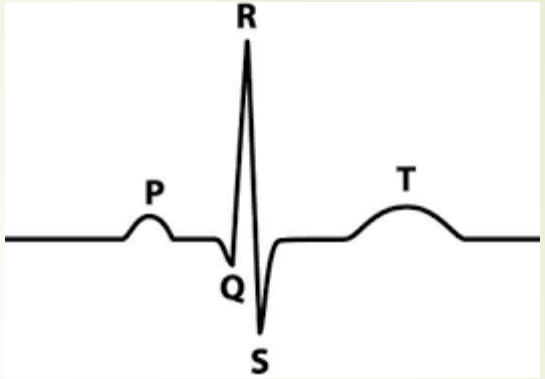
- May be normal
- Murmur
  - LVOT: due to combination of **septal hypertrophy** and **systolic anterior motion** of mitral valve
    - Degree of murmur based on amount of time mitral valve is in out Tract
  - Harsh ejection murmur usually loudest at apex/LLSB
    - 70% of patients will have at least a gradient of 30mmHg
  - May radiate to axilla and base but rarely to neck (like AS)
  - Murmur **increases** with
    - ***Valsalva*** (decreased preload)
    - ***Standing*** (decreased preload)
  - Murmur **decreases** with
    - ***Handgrip*** (increased afterload)
    - ***Squatting*** (increased afterload and preload)



Diagnosis

- **Clinical suspicion**
- ECG
- **Echocardiogram**
- Genetic testing
- Additional testing (may include)
  - Holter: assess for arrhythmias, part of routine surveillance
  - Exercise test: may be used for risk stratification
  - Cardiac MRI: may be helpful if echo diagnosis is **undetermined**, evaluate for **fibrosis**
  - Catheterization: rarely needed but may help distinguish from restrictive cardiomyopathy or constrictive pericarditis

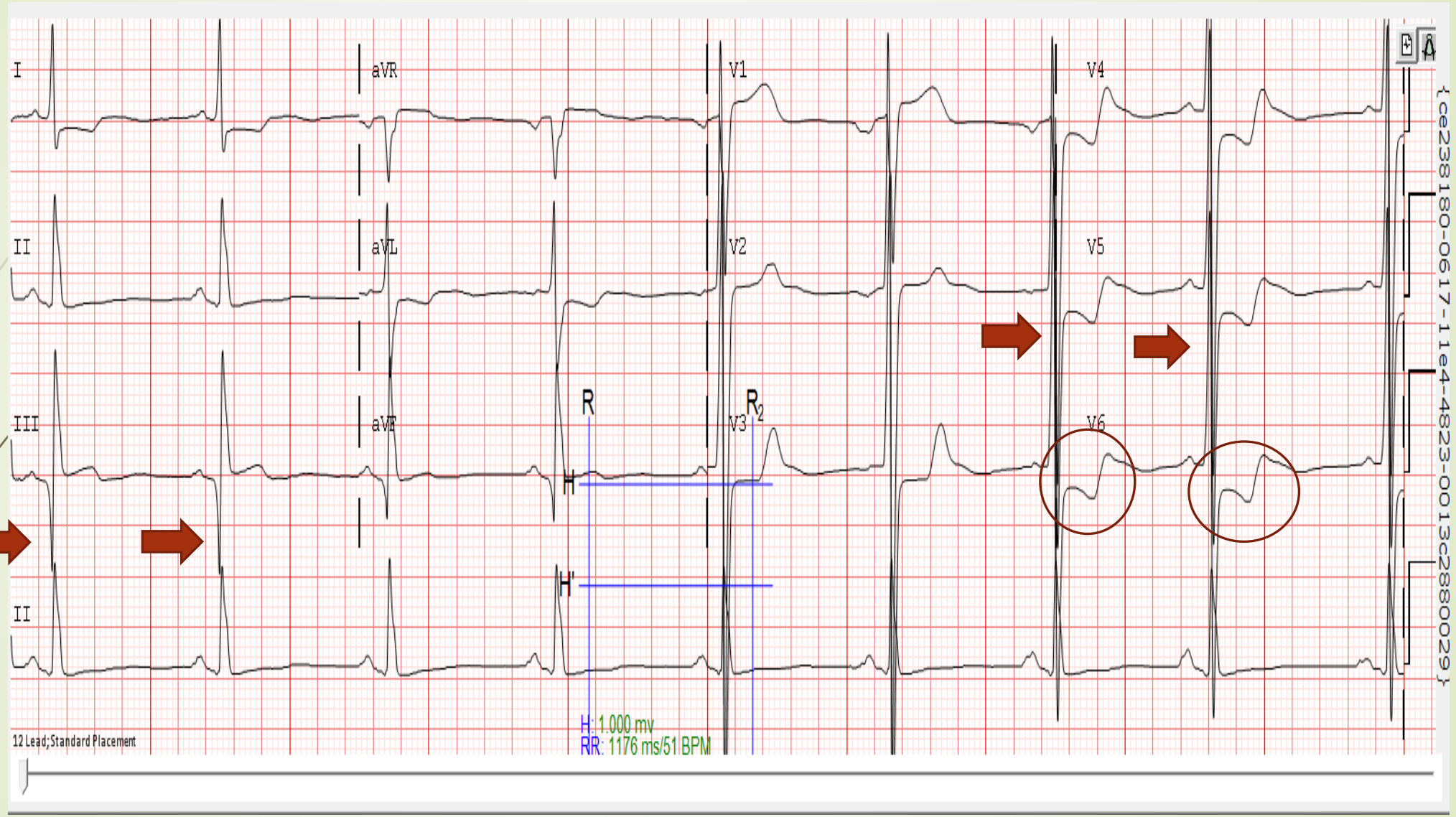
HCM- ECG

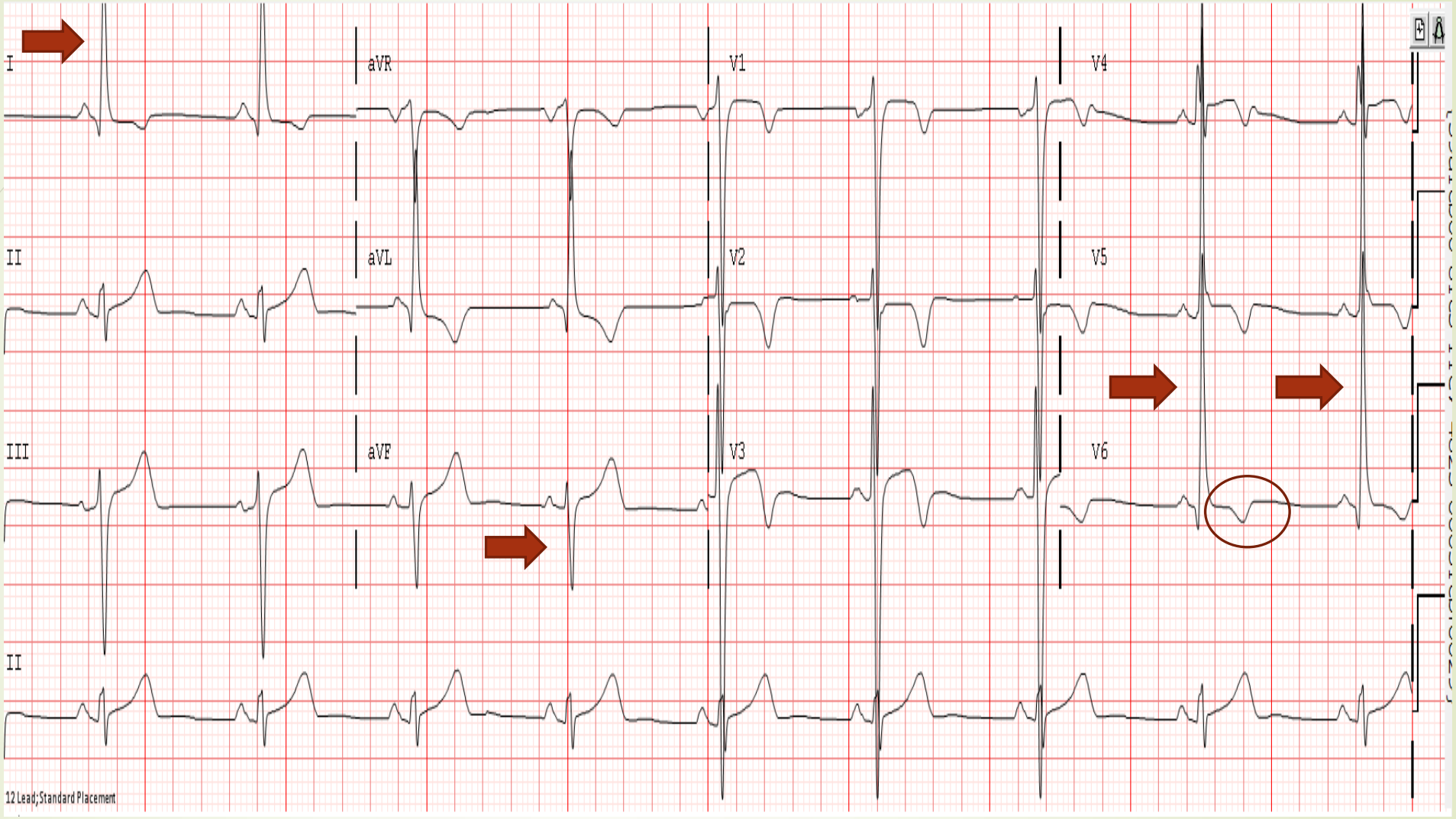


- *Most <u>sensitive</u>* but *not specific*
- One study looked at 2485 patients with HCM and **5% had a normal ECG**
- Findings include:
  - Prominent Q-waves in the inferior (II, III, aVF)and lateral leads (I, aVL, V4-V6)
    - >3mm or >40ms in duration
  - Enlarged P-waves in lead II suggesting atrial enlargement
    - Byproduct of diastolic dysfunction
  - Left axis deviation (QRS axis <0)
  - Inverted T-waves in lateral leads

Seattle Criteria for HCM

T-wave inversion (in two or more leads V2-V6, II, aVF, or I and aVL
ST depression (≥ 0.5 mm in two or more leads)
Sinus tachycardia
Pathologic Q waves (> 3 mm in depth or > 4 msec in duration in two or more leads except III and aVR)
Left bundle branch block
Left axis deviation (-30° to -90°)
Left atrial enlargement
Right ventricular hypertrophy (RV1 + SV5 > 10.5 mm and right axis deviation)
Ventricular pre-excitation
Brugada-like ECG pattern
Sinus bradycardia < 30 bpm
PVCs (≥ two PVCs per 10-second tracing or nonsustained ventricular tachycardia

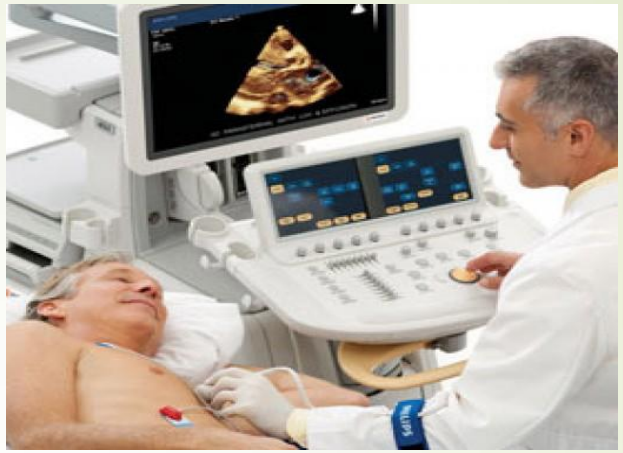

I
aVR
V1
V4
II
aVL
V2
V5
III
aVF
V3
V6
R
R2
H
H'
II
H: 1.000 mv
RR: 1176 ms/51 BPM
12 Lead;Standard Placement
{ce238180-0617-11e4-4823-0013c2880029}


I
aVR
V1
V4
II
aVL
V2
V5
III
aVF
V3
V6
II
12 Lead;Standard Placement

What about EKG screening for HCM?

- Not popular in North America due to costs of actual EKG and high false-positive rate
- Automated ECG screening has shown some promise (June ‘17)
  - Study of 128 patients with HCM and 256 controls
  - Electrophysiologist 71% sensitive, 95.7% specific
  - Non-voltage based algorithm 81% sensitive, 91% specific
- Another study from April of this 2017 looked at ECG-derived vectorcardiograophy (QRS-T angles and spatial peaks identified 84% and 94% patients respectively

Echo



- LV hypertrophy
  - Unexplained increased wall thickness >15mm (adult criteria)
  - >13mm suspicious
  - Most common area of hypertrophy is basal anterior septum
- Systolic anterior motion of the mitral valve
  - Combined with interventricular septal hypertrophy leads to outflow tract obstruction
  - Not required for diagnosis
- LVOT obstruction
  - Gradient is dynamic

Genetics

- Patients found gene+ 30-63% of probands according to the literature (obviously we don't know all the genes)
- Study of 84 children gene+ were found in 50% of sporadic and 65% of familial cases
- Conditions associated with HCM
  - **Fabry disease-** alpha galactosidase A deficiency, x-linked, treated with enzyme replacement
  - **Noonan syndrome-** characterized by dysmorphism, short status and CHD, 20% develop HCM
  - **Pompe disease-** acid maltase deficiency, excess glycogen, AR, poor muscle tone
  - **Fatty acid oxidation deficiency-** MCAD most common, AR, illness with fasting
  - **Mitochondrial-** maternal or AR, disturbances of brain and muscle function

Screening (of 1st degree relatives)

- All 1st degree relatives should undergo screening with H&P, ECG, Echo
  - Some recommend screening at any age, others at age 10
- Should be screened **q12-18** months by echo, and every 5 years after 21
- If proband found to be genetic positive, screen family for gene
- One study looked at family members who were gene**+** phenotype **–**
  - 162 patients, 18% went on to develop phenotype

Arrhythmias

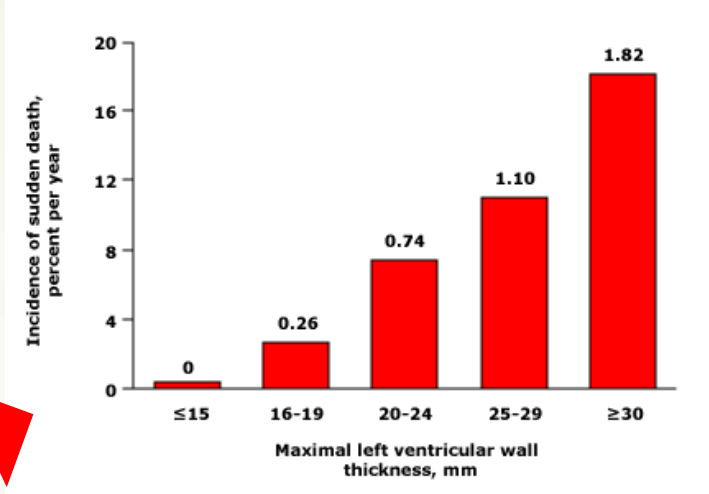
Arrhythmias

- May have both **atrial** and **ventricular** arrhythmias
- Incidence appears to be age related
- Non-sustained VT associated with significant increase risk of SCD (OR 4.4)
  - Duration, frequency and rate not associated
- SVT observed in up to 40% of patients
  - Atrial fibrillation most common (20-25% of all patients)
- Bradyarrhythmias are rare (outside of Fabry disease → HCM)
- Pharmacologic treatment aimed at symptoms/improve functional capacity/slow disease progression, not *prevention* of arrhythmias

Sudden Cardiac Death

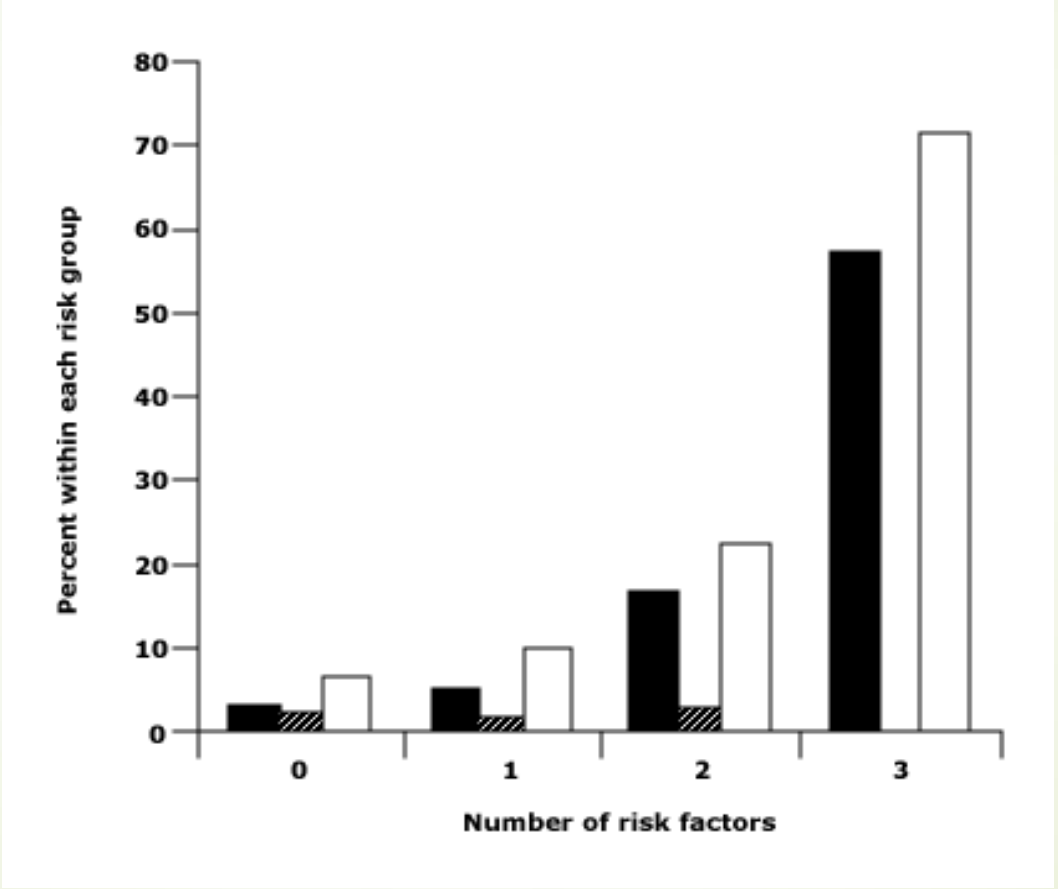
Risk factors for Sudden Cardiac Death (SCD) if you have HCM

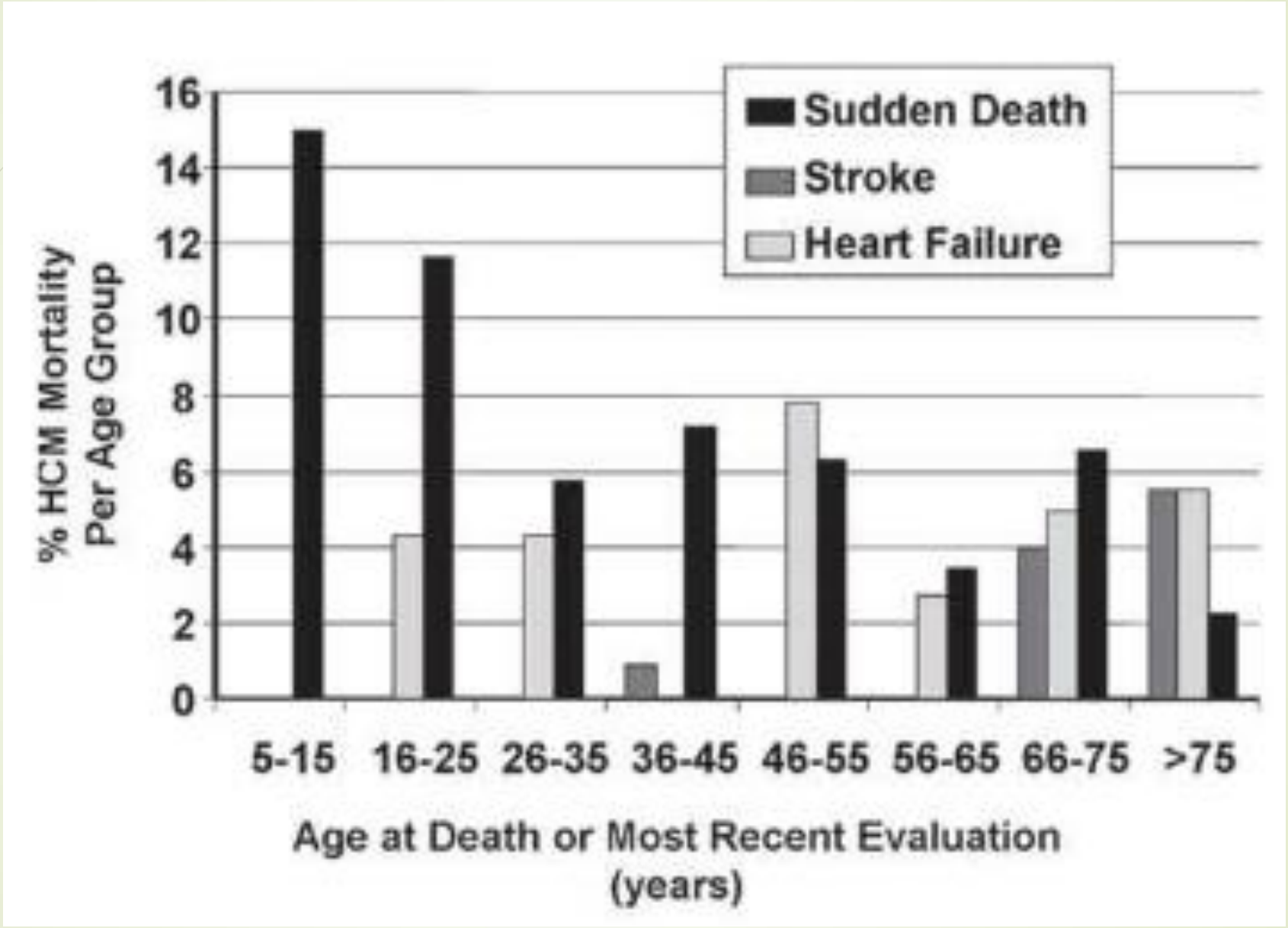
- 1. Family history of SCD
- 2. Syncope (unexplained)
- 3. Non-sustained ventricular tachycardia (NSVT)
  - ≥3 beats at rate ≥120
- 4. Massive left ventricular hypertrophy
  - LV wall thickness ≥30mm (seen in around 10% of HCM patients)
- 5. Abnormal blood pressure response to exercise
  - Failure to increase SBP by at least 20mmHg
  - Or fall >20mmHg from peak exercise BP to ongoing exercise



of high-risk factors and SCD

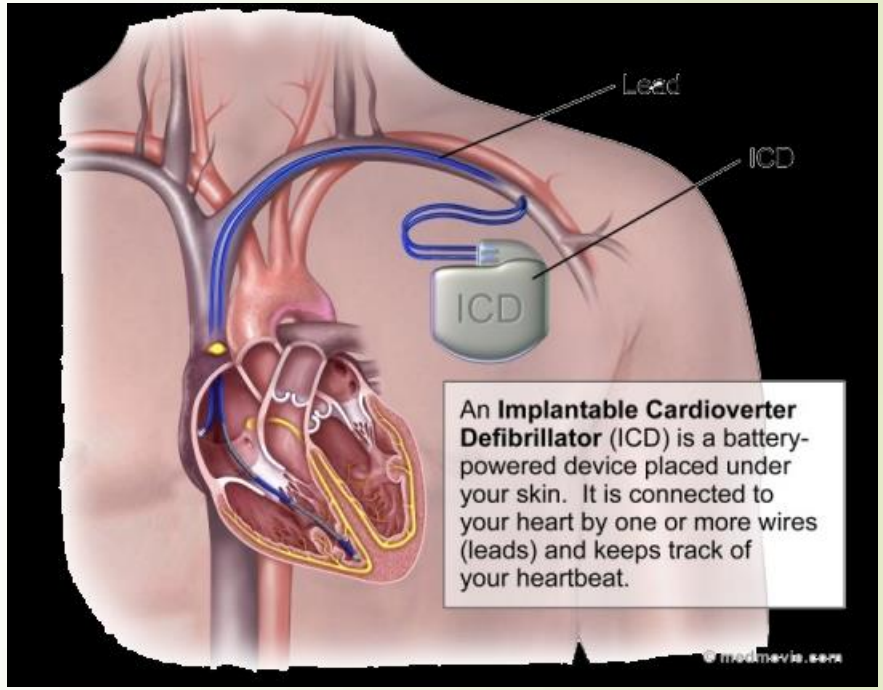
(368 patients, mean f/u 3.6 years)




Sudden Death
Stroke
Heart Failure
% HCM Mortality Per Age Group
16
14
12
10
8
6
4
2
0
5-15
16-25
26-35
36-45
46-55
56-65
66-75
>75
Age at Death or Most Recent Evaluation (years)

ICD Placement criteria

- ≥2 high risk factors =
  - Can consider for 1 HRF
- Sudden cardiac arrest =
- End-stage HCM (LVEF<50%)=
- LV apical aneurysm =



Treatment (medical therapy)

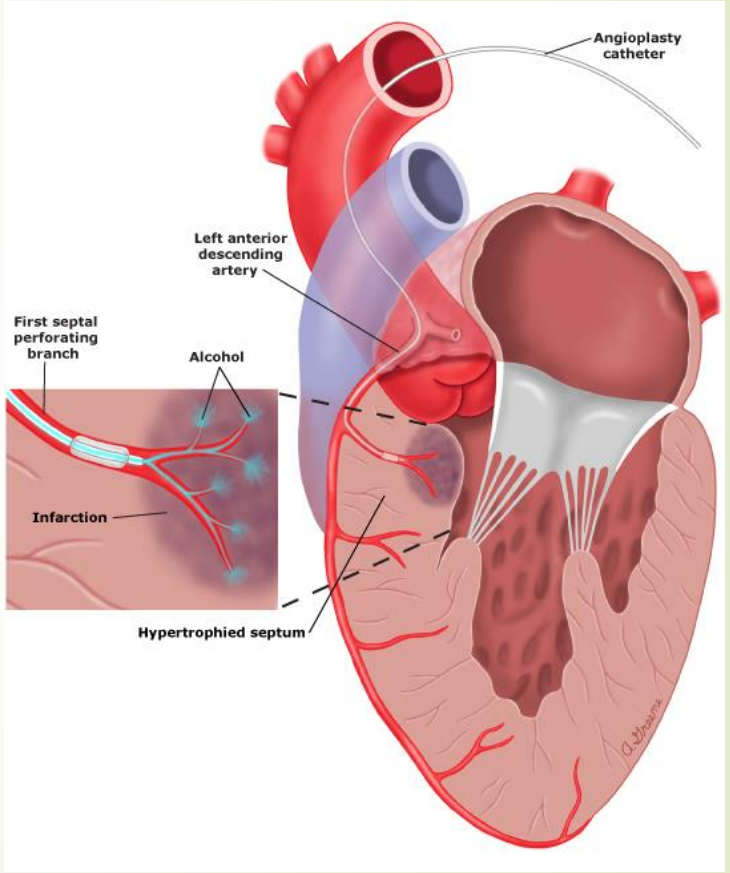
- **No RCTs**
- Goal: Treat sx, if no sx, probably don't need to treat
- Commonly used drugs
  - Beta blockers (BB): metoprolol, nadolol, atenolol, propranolol
  - Calcium channel blockers (CCB)- usually verapamil (non-dihyropyridine)
  - Disopyramide
- Ranolazine
- Judicious use of diuretics

Septal myectomy

- Involves direct removal of septal muscle
- May address abnormal mitral valve leaflets at the same time
- Complications
  - Excess septal tissue removal → VSD (2%)
  - LBBB or CHB (<5%)
- Perioperative mortality: 1-2%, 30d (3%)
  - Long term mortality: 1337 patients from Mayo who underwent septal myectomy had survival rates of 98%/96%/83% at 1/5/10 years
- Long term outcomes: 338 patients from Toronto
  - 72% of those had NYHA class III/IV → 83% NYHA I/II
  - 98% had no resting LVOTO
  - Lower rate of appropriate ICD discharges

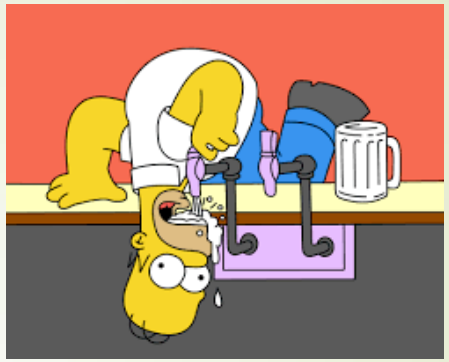
Alcohol ablation

- Creates localized infarction in basal septum
- Does not offer ability to address mitral valve
- Performed through perforator coronary artery



(Septal myectomy)

Vs.



(Alcohol ablation)

- No RCT's, data based on separate observational studies
- No difference in long-term mortality
- No difference in rates of aborted sudden cardiac death
- Need for pacemaker much higher in alcohol ablation group

Sports

Sports restrictions

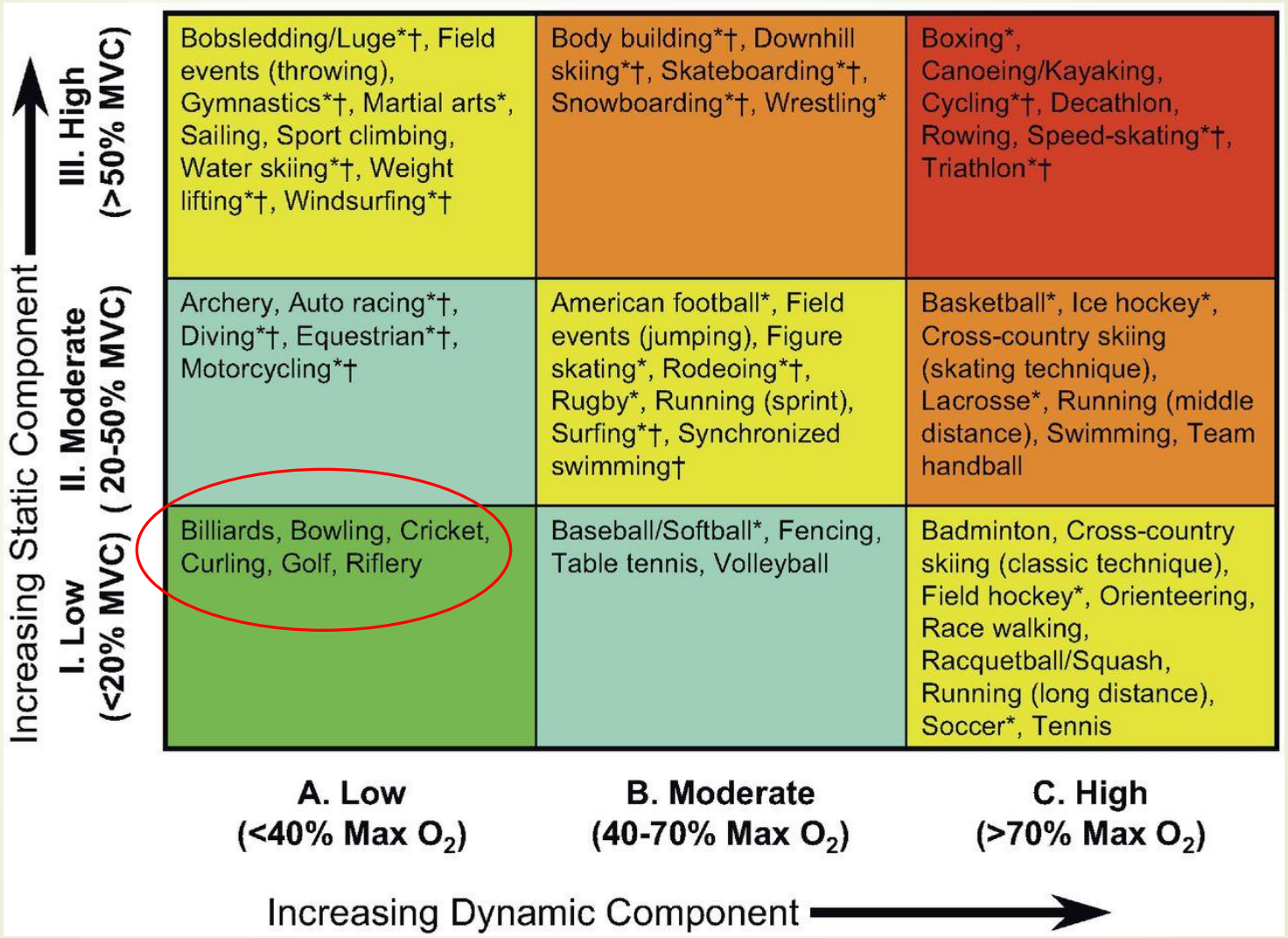
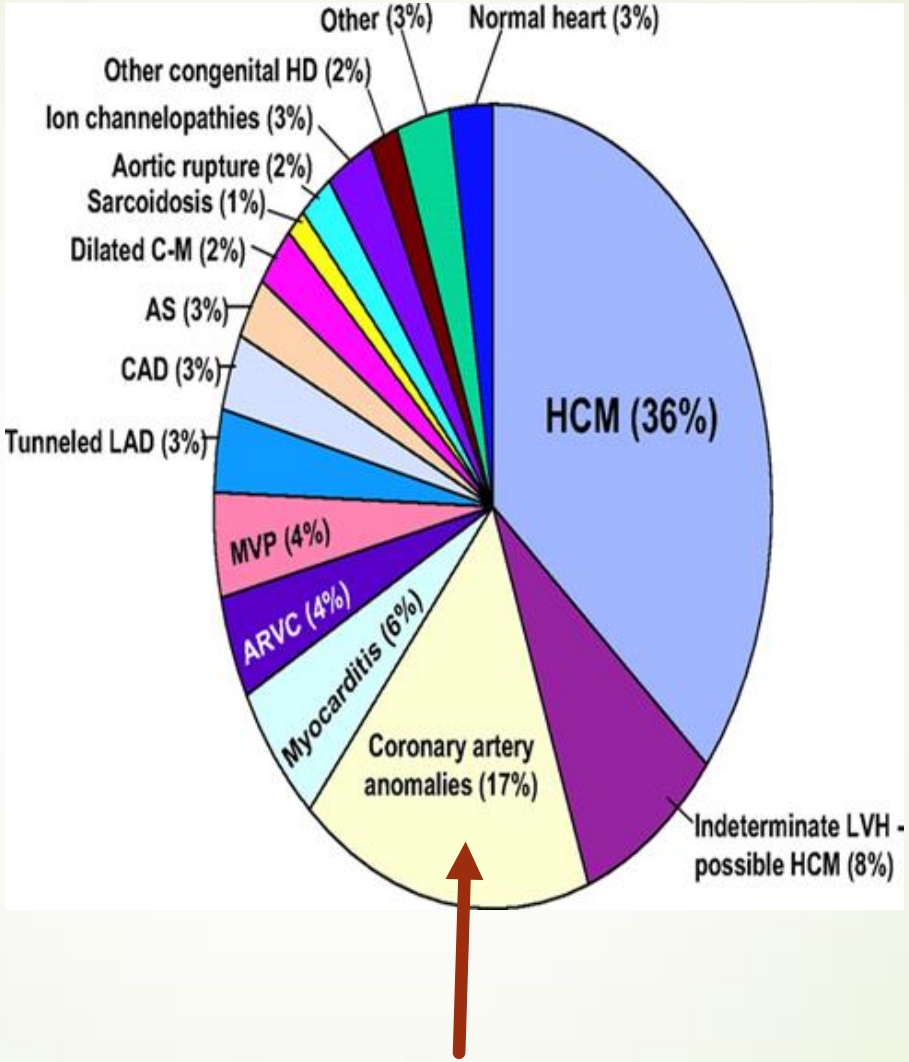


Table.

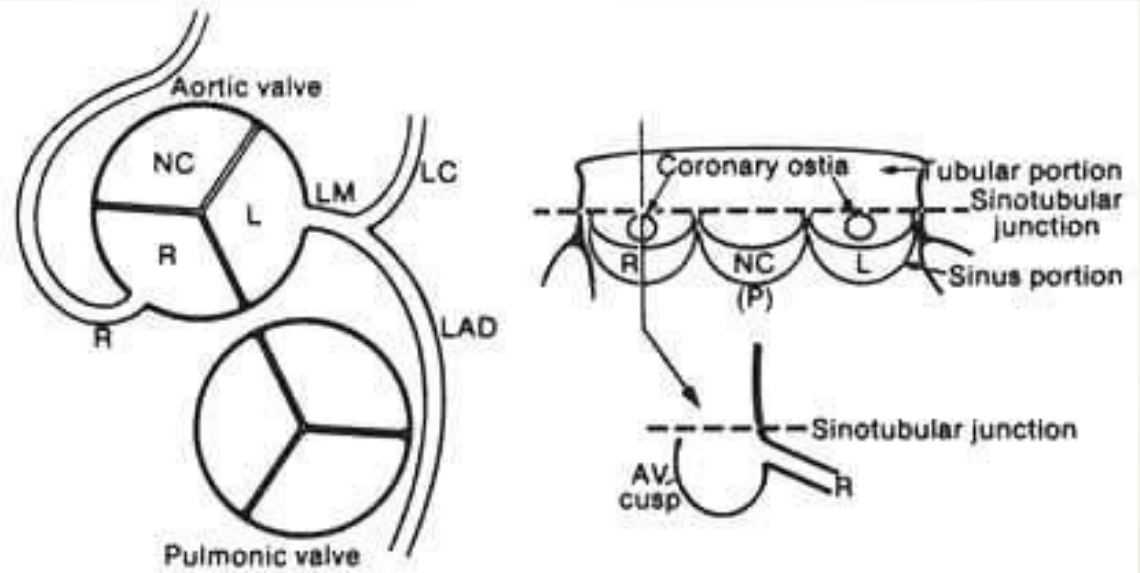
Comparison of guidelines regarding athletic participation for individuals with HCM.

Disease Severity	European Society for Cardiology	36th Bethesda Guidelines
Definitive clinical HCM	No competitive sports	Class IA sports
Definitive clinical HCM, low risk of SCD	Class IA sports	Class IA sports
Genotype positive–phenotype, negative HCM	Recreational activities only, no competitive sports	No sports restriction, close surveillance


Other (3%)
Normal heart (3%)
Other congenital HD (2%)
Ion channelopathies (3%)
Aortic rupture (2%)
Sarcoidosis (1%)
Dilated C-M (2%)
AS (3%)
CAD (3%)
Tunneled LAD (3%)
HCM (36%)
MVP (4%)
ARVC (4%)
Myocarditis (6%)
Coronary artery anomalies (17%)
Indeterminate LVH - possible HCM (8%)

Coronary Artery

- Normal anatomy



Coronary Artery Anomalies

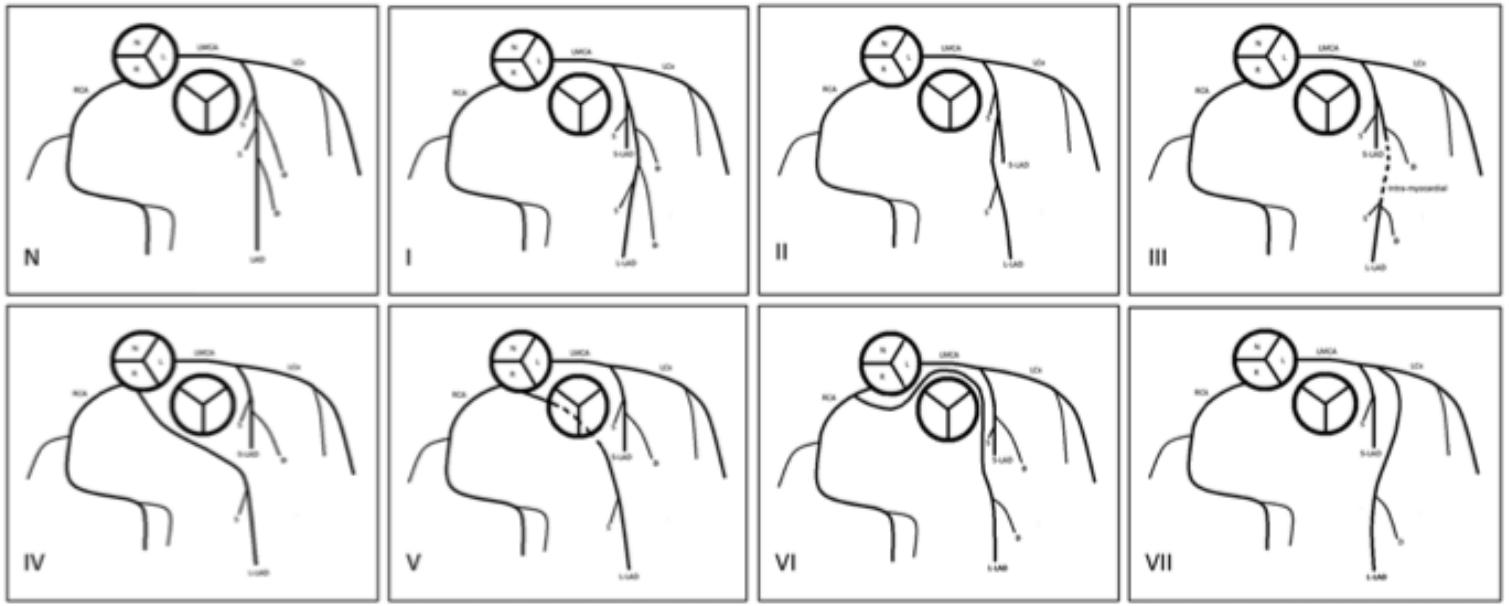
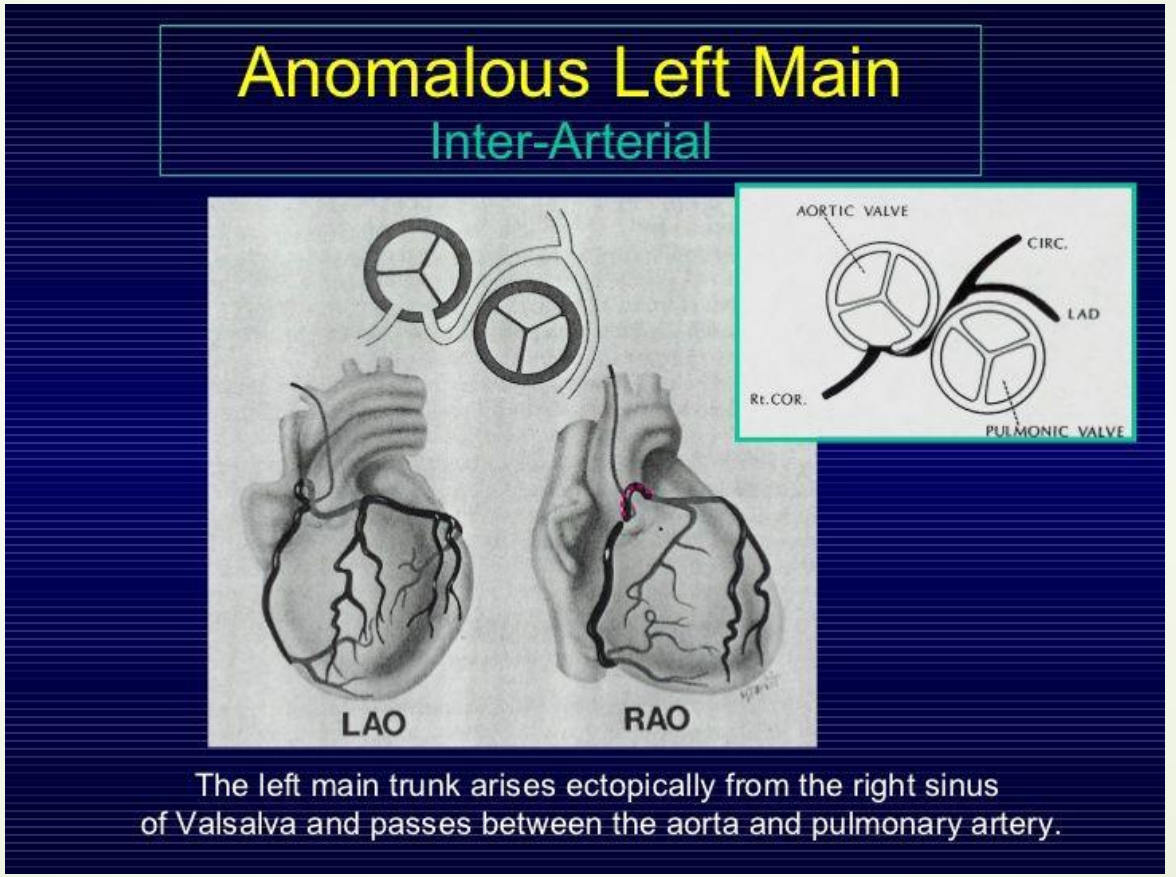


Figure 2. *Line drawing showing various types of dual left anterior descending (D-LAD) coronary artery. Normal LAD (N-LAD) is given for comparison. N-LAD is single, originates from left main coronary artery (LMCA), occupies the anterior interventricular groove and gives rise to septal (S) and diagonal (D) branches. In D-LAD types I-III, both short and long LADs (S-LAD and L-LAD) originate from a common LAD trunk. The S-LAD occupies the proximal anterior interventricular groove. The L-LAD after its proximal course along the left ventricular side (type I), right ventricular side (type II) or intra-myocardial course (type III) enters the distal interventricular groove. In D-LAD types IV–VI, S-LAD originates as a branch of LMCA and L-LAD originates from the right side (right coronary sinus or proximal right coronary artery) and reaches the anterior interventricular groove following prepulmonic (type IV), intramyocardial (type V), or interarterial course (type VI). In type VII, S-LAD originates as a separate branch from the LMCA before its bifurcation into the L-LAD and left circumflex coronary artery (LCX). (R, L, N = right, left, and non-coronary sinuses of aorta).*

Coronary Artery Anomalies



Coronary Artery Anomalies

- 2nd most common cause of SCD in the young athlete
- Occurs in ~0.6% of births (not all are pathologic)
- Most common LMCA from the right

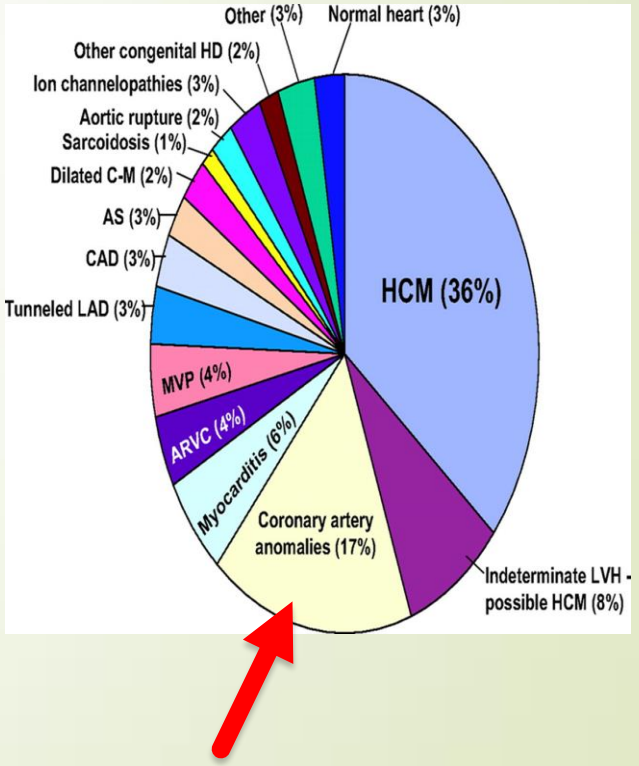
Sinus of Valsalva (pictured previously)

- LMCA and LAD arising from RCA are dangerous
- Exercise increases blood flow through aorta

And pulmonary artery causing compression

- Most common pathologic finding LMCA from

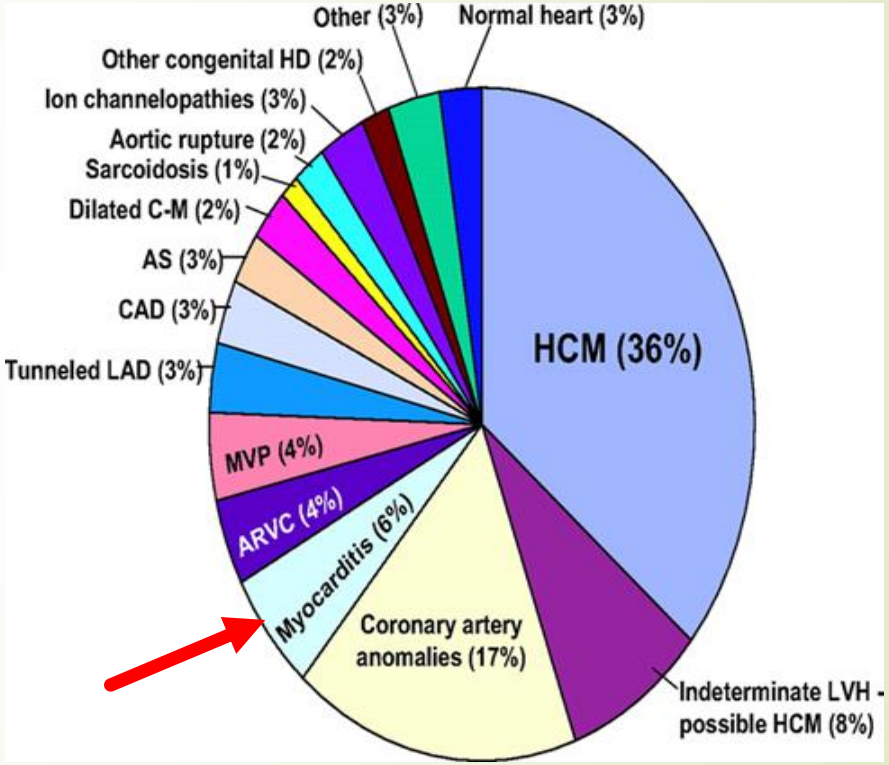
the right Sinus of Valsalva (pictured previously)



Coronary Artery Anomaly

- Presenting signs: angina, syncope, SCD
- Diagnosis:
  - Echocardiogram- easy to perform, can be difficult to visualize
  - CT scan- excellent views, quick, radiation, heart rate dependent
  - cMRI- decent views, no radiation, longer (sedation?)
  - Angiography- excellent views, invasive, radiation

Myocarditis



Myocarditis

- Inflammation of the myocardium
- Children more often acute, adults more often chronic
- Etiology: infection (viral), toxic, autoimmune (more often adults)
  - Viral- enterovirus, adenovirus, parvo, EBV, CMV, HHV-6
- Bimodal distribution (like HCM) with infants and adolescence
- Incidence <1 per 100,000 children
- Arrhythmias may occur in up to 45%= SVT, VT, heart block

Myocarditis: Clinical Manifestations

- Chest pain (45 percent)
- Respiratory distress (28 percent)
- Gastrointestinal symptoms (27 percent)
- Hepatomegaly (27 percent)
- Gallop rhythm (20 percent)
- Poor perfusion/diminished extremity pulses (16 percent)
- Viral prodrome (41 percent)
  - Fever, myalgia, malaise

Myocarditis

- Physical Exam: tachypnea, rales, **gallop rhythm**, possible MR or TR if ventricle is dilated
- EKG: ST segment changes, T-wave inversion, abnormal axis, chamber enlargement, **decreased voltages**
- Echo: LV dysfunction, abnormal chamber shape, mitral regurgitation
- cMRI: may show inflammation

Myocarditis

- Sports restrictions = 6 months after onset of disease

Covid 19 in pediatrics

- Covid 19 wide degree of sx (most asx or mildly ill)
- Most common sx: fever and cough
  - May also in clude SOB, myalgia, rhinorrhea, headache, N/V, abd pain, diarrhea, sore throat, fatigue and the infamous loss of smell or taste
- Labs may include leukopenia, lympocytopenia, elevated procalcitonin or CRP

Multi-inflammatory syndrome

- Similar features to Kawasaki and Toxic Shock Syndrome (TSS)
- Rare complication, usually in older children and adolescents (previously healthy)
- More common in Black and Hispanic Children
- Similar features to Macrophage Activation Syndrome and presumed to be 2ndary to cytokine storm
- Positive serology but negative PCR in most cases (thus likely after acute infxn)
- Sx: Fevers, GI sx, rash, conjunctivitis for 3-5 days, then potential shock and multi-organ involvement
- Labs: lymphocytopenia, elevated CSR/ESR/d-dimer, elevated cardiac markers (troponin, BNP)

Our suggested approach to the evaluation of patients with suspected COVID-19-associated multisystem inflammatory syndrome in children (MIS-C)

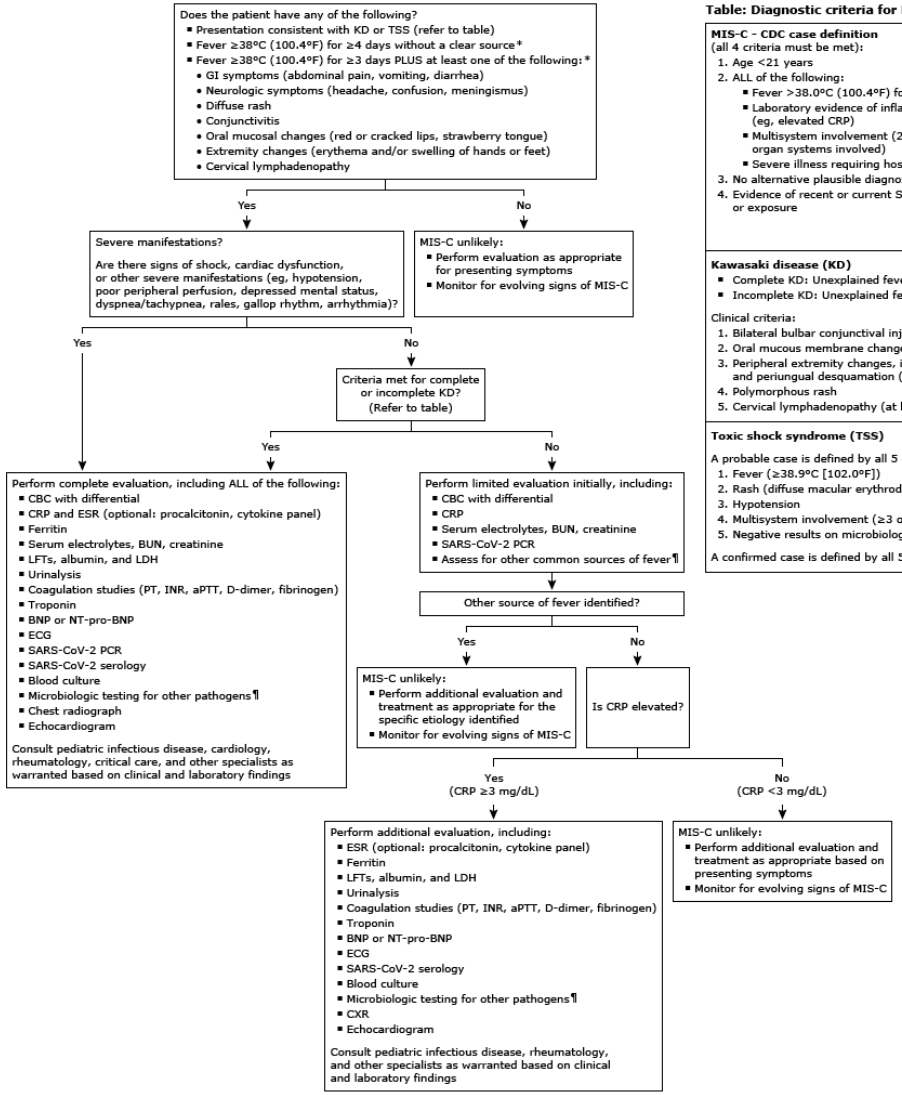


Table: Diagnostic criteria for MIS-C, KD, and TSS

MIS-C - CDC case definition (all 4 criteria must be met):	**MIS-C - WHO case definition** (all 6 criteria must be met):
1. Age <21 years 2. ALL of the following: ▪ Fever >38.0°C (100.4°F) for ≥24 hours ▪ Laboratory evidence of inflammation (eg, elevated CRP) ▪ Multisystem involvement (2 or more organ systems involved) ▪ Severe illness requiring hospitalization 3. No alternative plausible diagnoses 4. Evidence of recent or current SARS-CoV-2 infection or exposure	1. Age <20 years 2. Fever for ≥3 days 3. Signs of multisystem involvement (at least 2): ▪ Rash, conjunctivitis, mucositis, swollen hands or feet ▪ Hypotension or shock ▪ Coagulopathy ▪ Acute GI symptoms (diarrhea, vomiting, abdominal pain) 4. Elevated markers of inflammation (eg, CRP) 5. No other obvious cause of inflammation 6. Evidence of recent or current SARS-CoV-2 infection or exposure

Kawasaki disease (KD)

- Complete KD: Unexplained fever for ≥4 days PLUS at least 4 of the 5 clinical criteria below
- Incomplete KD: Unexplained fever for ≥5 days PLUS 2 to 3 of the 5 clinical criteria below

Clinical criteria:

1. Bilateral bulbar conjunctival injection
2. Oral mucous membrane changes, including injected or fissured lips, injected pharynx, or strawberry tongue
3. Peripheral extremity changes, including erythema of palms or soles, edema of hands or feet (acute phase), and periungual desquamation (convalescent phase)
4. Polymorphous rash
5. Cervical lymphadenopathy (at least 1 lymph node >1.5 cm in diameter)

Toxic shock syndrome (TSS)

A probable case is defined by all 5 of the following criteria:

1. Fever (≥38.9°C [102.0°F])
2. Rash (diffuse macular erythroderma)
3. Hypotension
4. Multisystem involvement (≥3 organ systems involved)
5. Negative results on microbiologic and/or serologic testing for other causative organisms

A confirmed case is defined by all 5 of the above criteria PLUS desquamation 1 to 2 weeks after onset of rash.

COVID-19: coronavirus disease 2019; KD: Kawasaki disease; TSS: toxic shock syndrome; GI: gastrointestinal; CBC: complete blood count; CRP: C-reactive protein; ESR: erythrocyte sedimentation rate; BUN: blood urea nitrogen; LFTs: liver function tests; LDH: lactate dehydrogenase; PT: prothrombin time; INR: international normalized ratio; aPTT: activated partial thromboplastin time; BNP: brain natriuretic peptide; NT-pro-BNP: N-terminal pro-BNP; ECG: electrocardiogram; SARS-CoV-2: severe acute respiratory syndrome coronavirus 2; PCR: polymerase chain reaction; CXR: chest radiograph; CDC: Centers for Disease Control and Prevention; WHO: World Health Organization; EBV: Epstein-Barr virus; CMV: cytomegalovirus.

* Evaluation for MIS-C may be appropriate in children with a shorter duration of fever if they have severe manifestations.

¶ In children with moderate or severe manifestations, testing for other pathogens generally includes blood, urine, stool, and throat cultures; respiratory viral panel; and testing for EBV, CMV, enterovirus, and adenovirus. In children presenting with mild symptoms, microbiologic testing should be done as clinically indicated according to the age of the child and their specific symptoms (eg, throat culture if the child has a sore throat, respiratory viral panel if there are respiratory symptoms).

UpToDate®

MIS-C vs. Kawasaki

- MIS-C tends to be in older children , Kawasaki traditionally ~6 months- 5 years of age
- More common in Black and Hispanic population
- Presents with GI sx and cardiovascular involvement
- MIS-C > Kawasaki for inflammatory markers
- Most children fully recover (8 death reports out of 363 cases)

Covid 19 & myocarditis

- Clark et al looked at 22 collegiate athletes retrospectively with cardiac MRI
- Median time from infection to study was 52 days, mean age 20.2 years
- Most athletes had mild sx (77%), remainder were asymptomatic
- None had abnormal troponin, EK or LVEF <50%
- 1 patient was found to have LGE (sign of myocarditis)

Returning to Sports after Covid-19

https://www.acc.org/latest-in-cardiology/articles/2020/07/13/13/37/returning-to-play-after-coronavirus-infection?fbclid=IwAR1d0tsq_xv-Iq7b1Ep6htf7fg0%E2%80%A6

Returning To Play After Coronavirus Infection: Pediatric Cardiologists' Perspective

Jul 14, 2020 | Peter N Dean, MD; Lanier Burns Jackson, MD; Stephen M. Paridon, MD, FACC

Expert Analysis

Quick Takes

- Returning to sports participation after a COVID infection will be a significant question posed to pediatric providers in the coming months
- The approach to sports participation clearance in pediatric patients should differ from the approach in adult patients
- Most pediatric patients will be able to be easily cleared for participation without extensive cardiac testing, but pediatric providers should ensure patients have fully recovered and have no evidence of myocardial injury

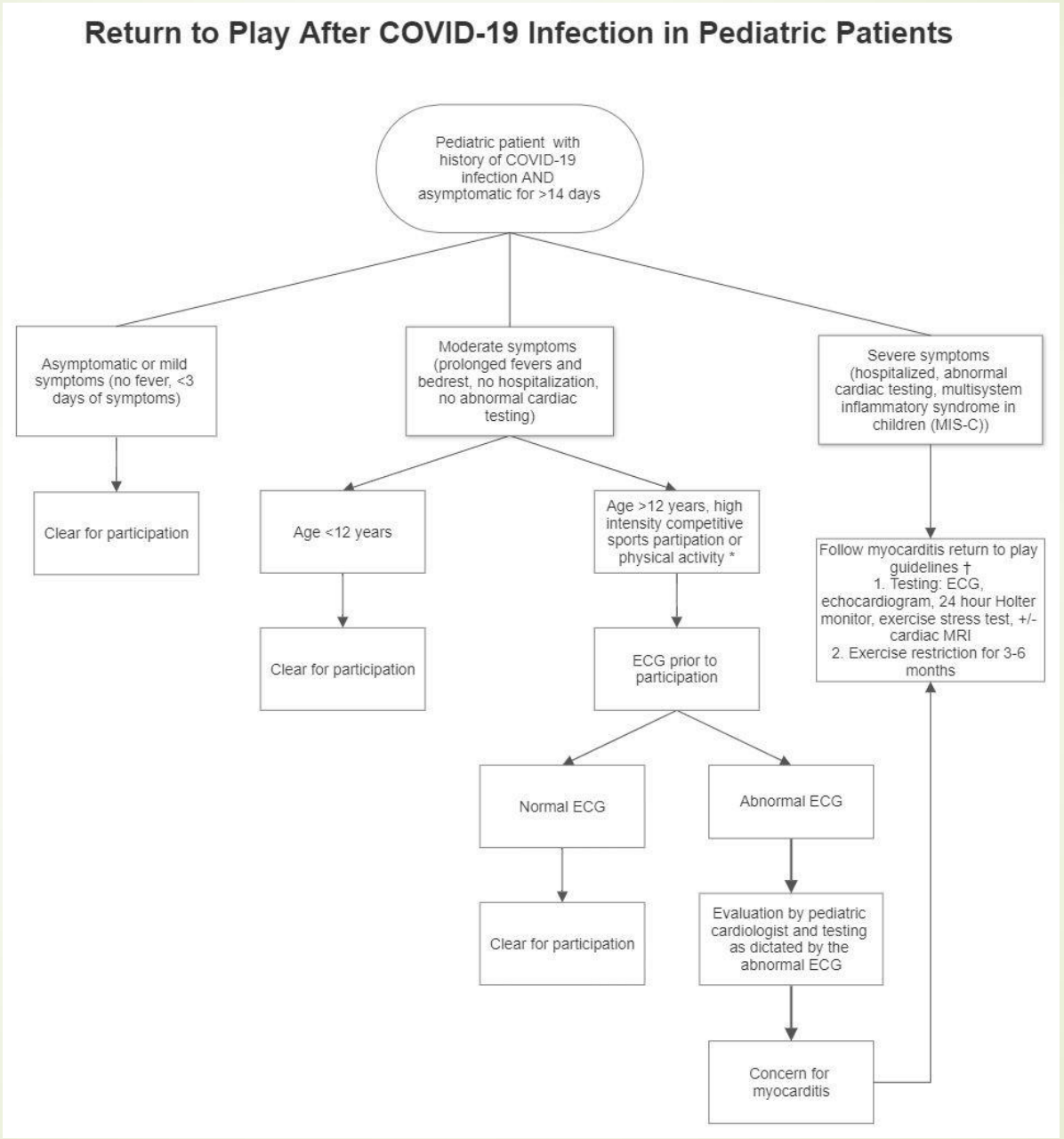
Typically, during the summer months, pediatricians and family physicians are inundated with requests for sports clearance physicals. This year, as schools and sports reopen, a new question will need to be addressed at these appointments: is it safe for my child to resume physical activity and sports after a COVID-19 infection?

There have been eloquent editorials written on the return-to-play topic from an adult cardiologist's perspective[1-3] but no statements or articles addressing return to play from a pediatric cardiologist's perspective.

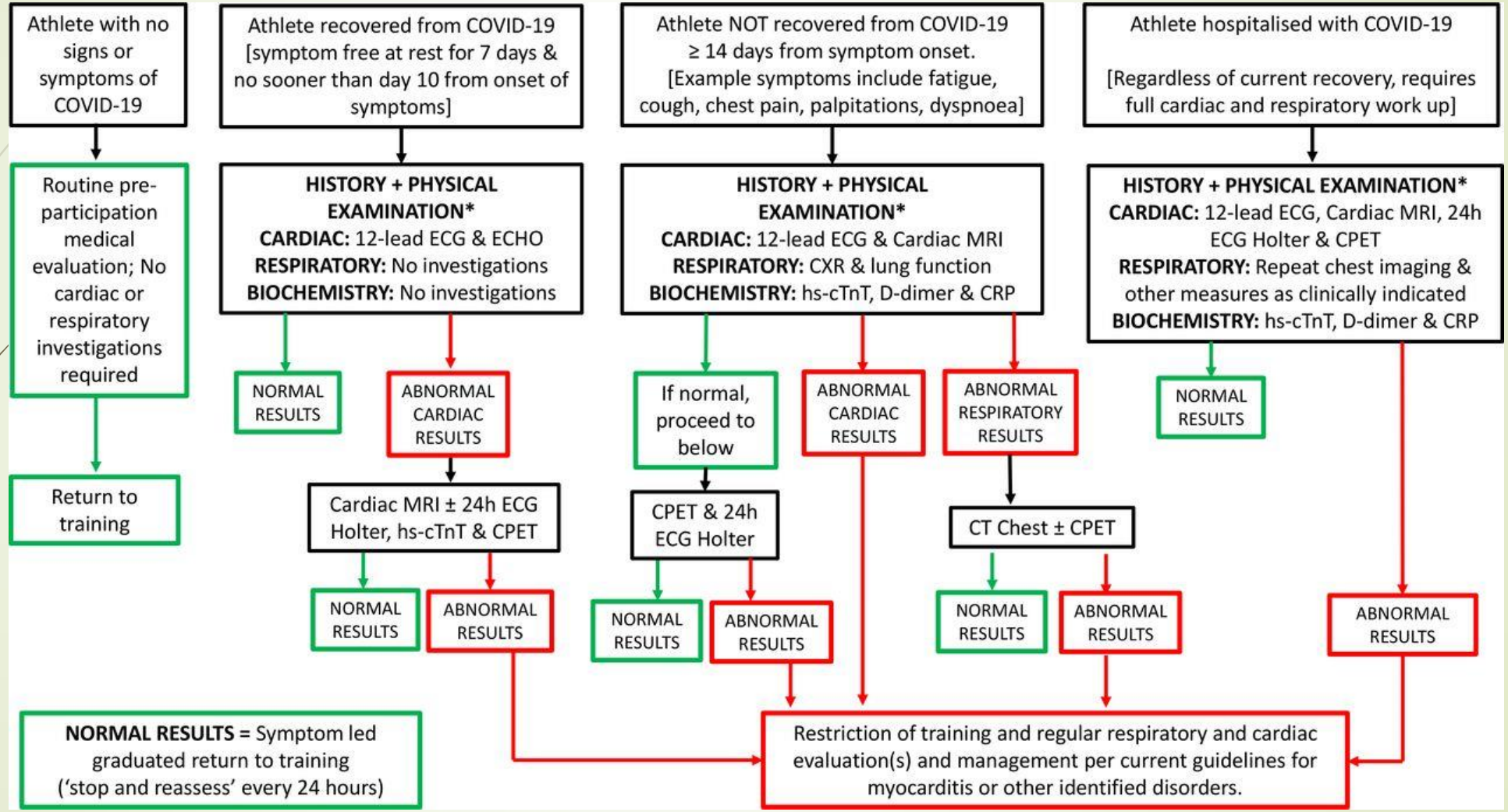
For various reasons we believe it would be wrong to universally apply the "adult return-to-play" criteria to the pediatric population. First, fortunately, COVID-19 infections in pediatric patients are more likely to be asymptomatic or mild compared to adult patients.[4,5] This means pediatric providers will frequently be presented with the return-to-play question for patients who had asymptomatic disease or very mild disease. Second, there is a significant amount of variation in intensity of youth and school-based sports. Providers should not treat the 7-year-old recreational soccer player the same as the 18-year-old varsity basketball player. Third, the pediatric population is less reliant on electrocardiograms (ECGs), echocardiograms, stress testing and troponins for general screening and for clearing patients for exercise and sports. Lastly, the recommendation to consider following the COVID-19 positive pathway algorithm if a patient develops symptoms concerning COVID-19 and testing is negative or not may be inappropriate in the pediatric population.[1] Respiratory infections in the pediatric population are highly prevalent and in the presence of reliable negative COVID-19 testing should not result in a presumptive diagnosis in the pediatric population.

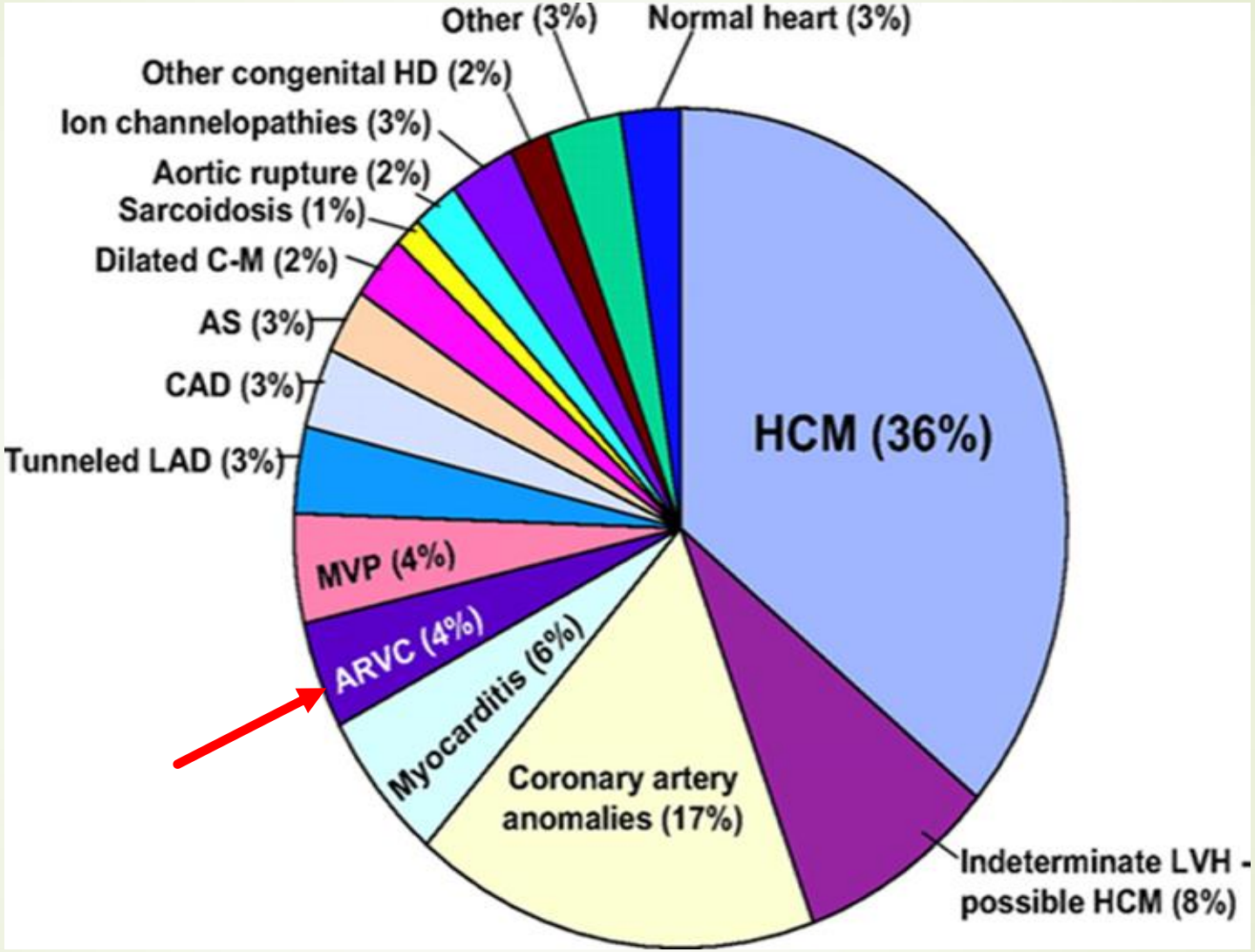
The question of returning to sports is significant because of the propensity for COVID-19 to cause cardiac damage and myocarditis.[6] While the incidence of myocarditis is lower in the pediatric population compared to the adult population, myocarditis is known to be a cause of sudden death during exercise in the young athletic populations. Similar to other forms of myocarditis, providers caring for patients who have had a COVID infection should be confident there is no myocardial injury prior to clearing athletes to participate.[7] When considering the question of return-to-play, we believe there are three variables to consider: (1) How recent was

Return to Play After COVID-19 Infection in Pediatric Patients

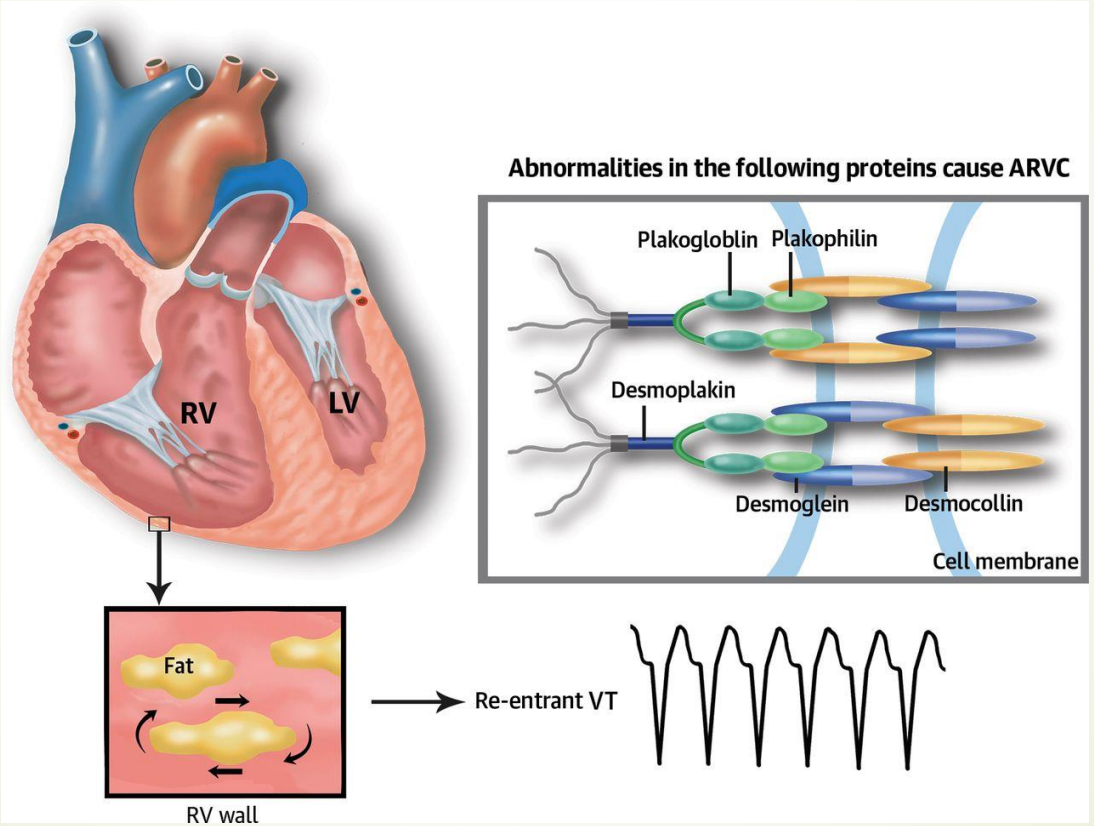


Elite Athletes




Other (3%)
Normal heart (3%)
Other congenital HD (2%)
Ion channelopathies (3%)
Aortic rupture (2%)
Sarcoidosis (1%)
Dilated C-M (2%)
AS (3%)
CAD (3%)
Tunneled LAD (3%)
HCM (36%)
MVP (4%)
ARVC (4%)
Myocarditis (6%)
Coronary artery anomalies (17%)
Indeterminate LVH - possible HCM (8%)

Arrhythmogenic Right Ventricular Cardiomyopathy (ARVC/ARVD)



Arrhythmogenic Right Ventricular Cardiomyopathy

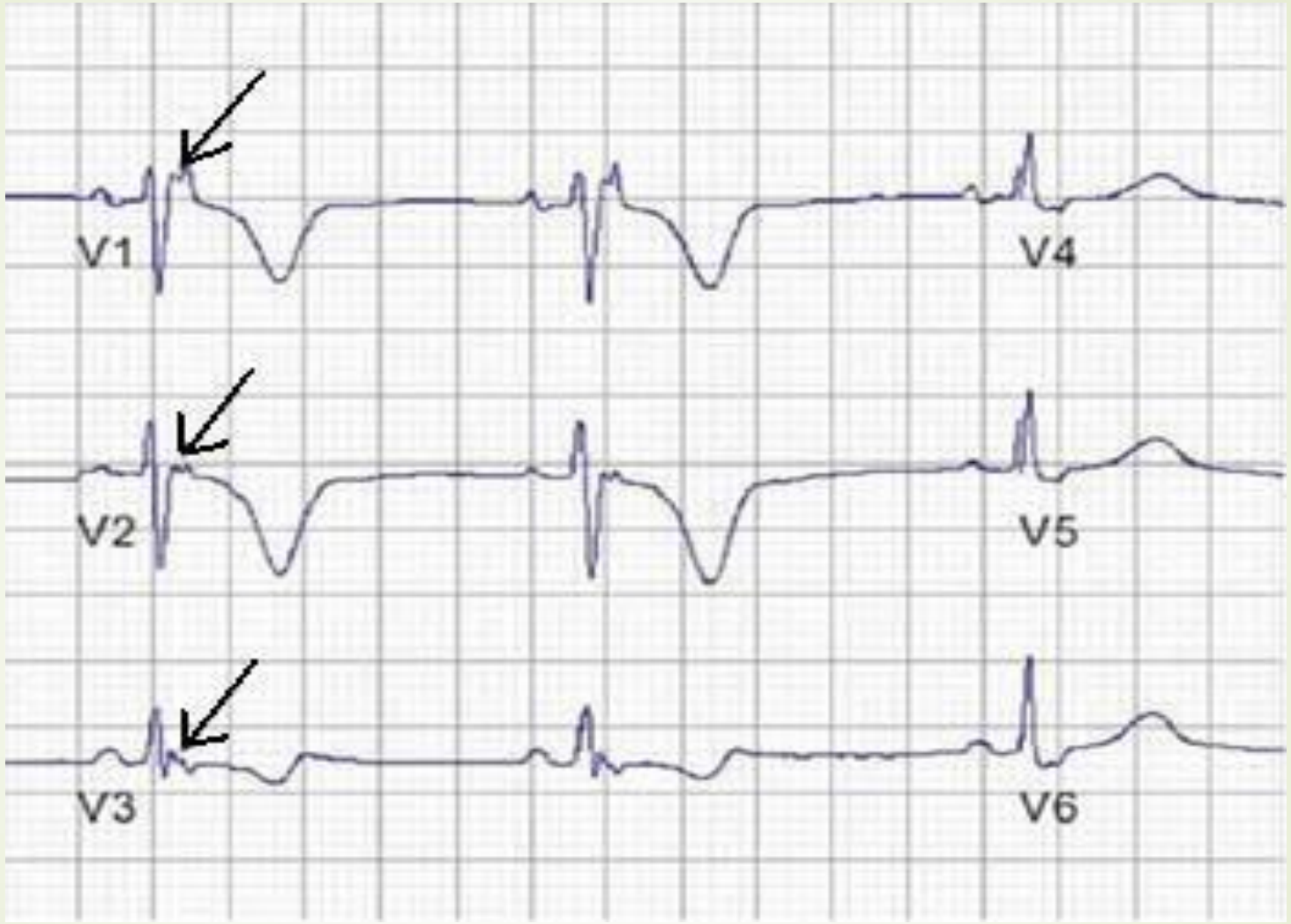
- Prevalence of 1/2000-5000 (very common in Northern Italy)
- Gene mutation in desmosomal proteins
- Focal myocarditis → fibrofatty replacement (substrate for arrhythmia) → RV dilation, dysfunction, and aneurysm
- 5x risk of SCD during exercise

Arrhythmogenic Right Ventricular Cardiomyopathy (ARVC/ARVD)

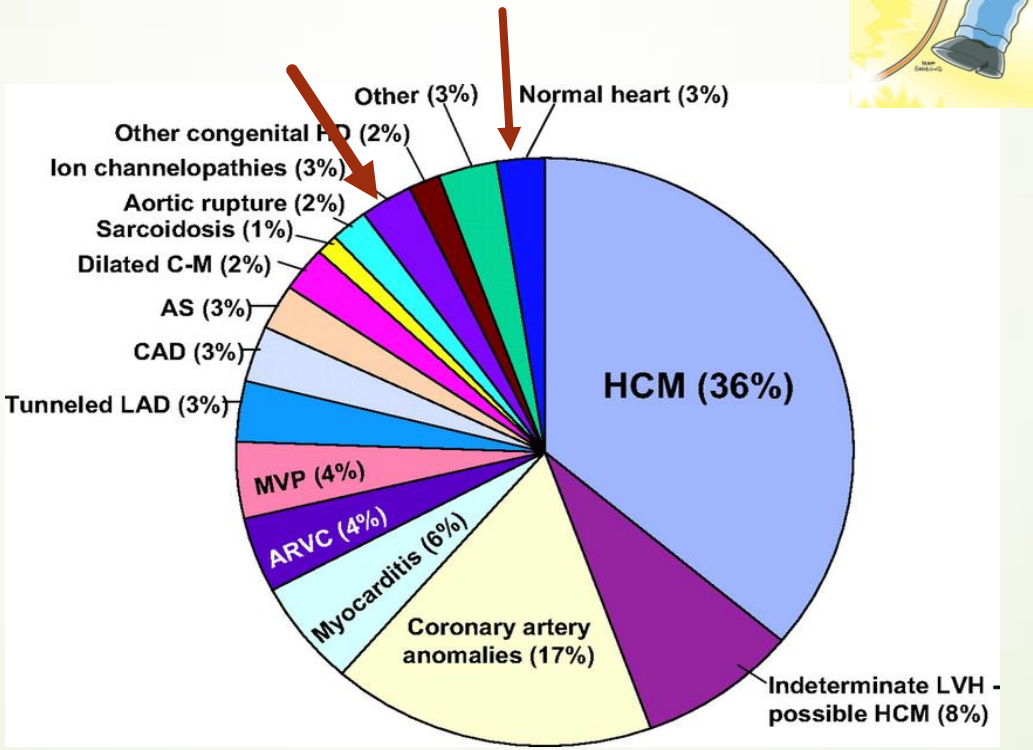
- **Symptoms:**
- Palpitations – 67%
- Syncope – 32%
- Atypical chest pain – 27%
- Dyspnea – 11%
- RV failure – 6%

Arrhythmogenic Right Ventricular Cardiomyopathy

- Dx: EKG, Echo, cardiac MRI, arrhythmia
- EKG: Inverted T-waves in V_{1-3}, Epsilon wave
- Echo: Regional akinesia, dyskinesia, or aneurysm
- MRI: see above (better imaging modality)
- Arrhythmia: Ventricular tachycardia of left bundle branch morphology with superior axis


V1
V4
V2
V5
V3
V6

Electrical Conditions

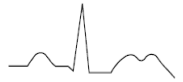
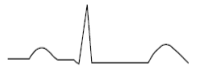


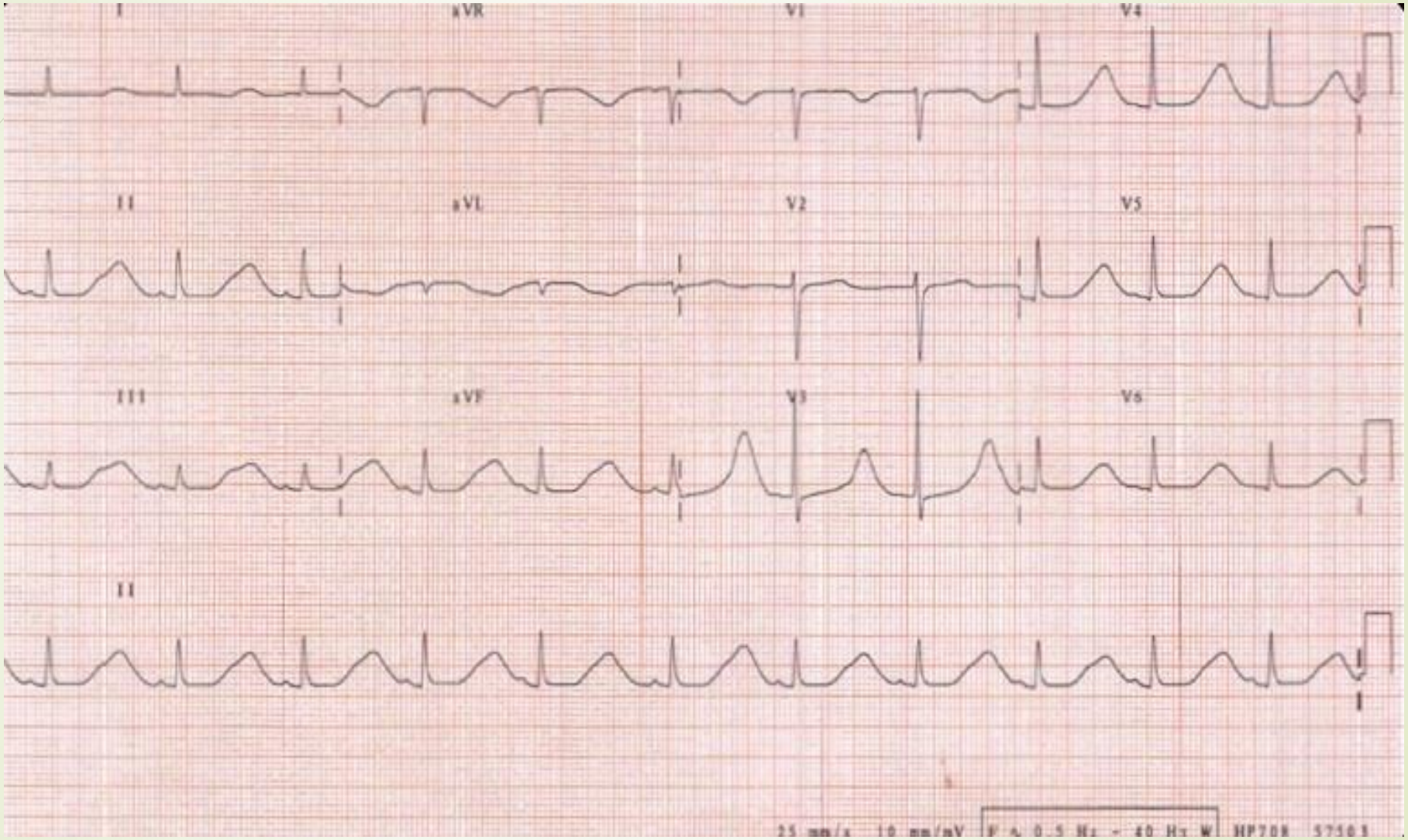
Long QT syndrome

Table 1. **Diagnostic Criteria for the Congenital Long QT Syndrome***

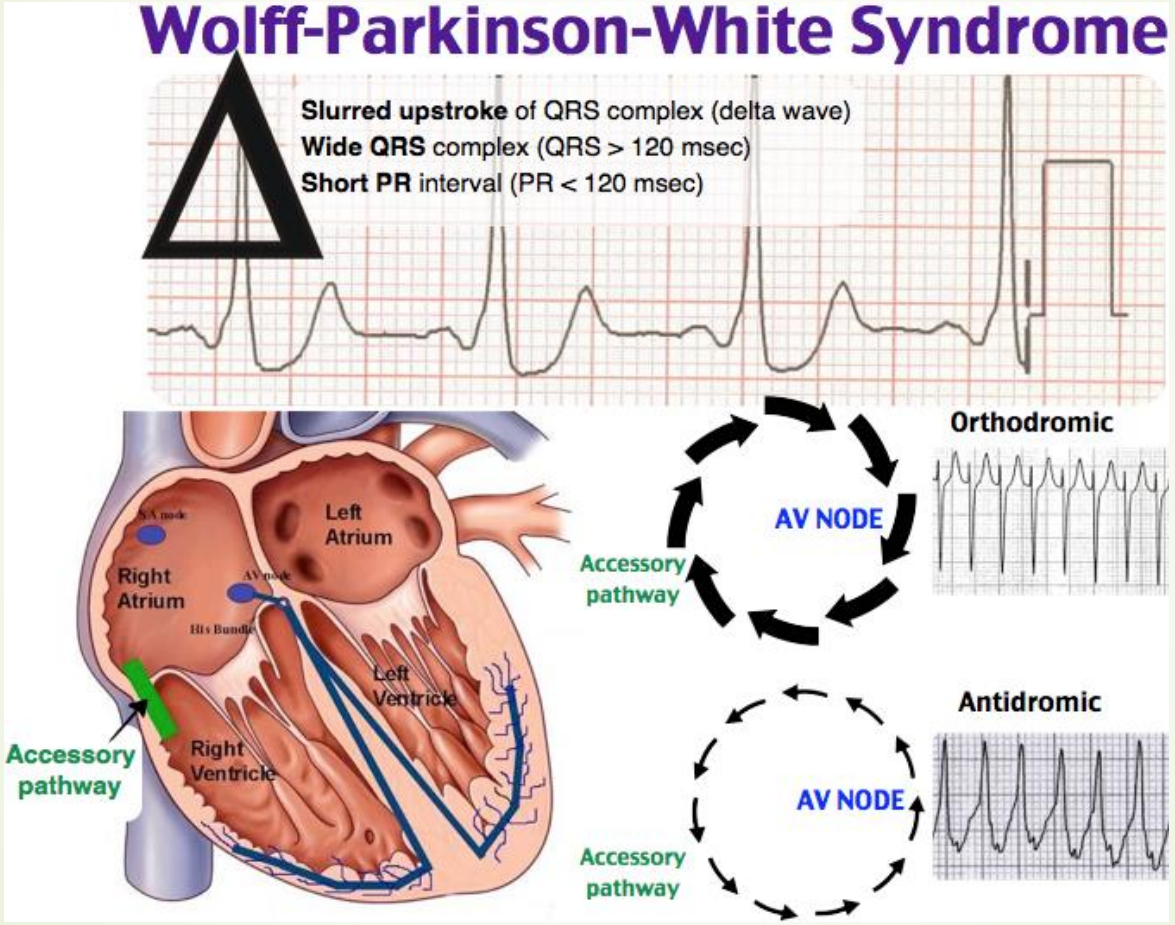
Criteria	Points
Electrocardiographic findings	
Corrected QT interval	
≥480 ms†	3
460–480 ms†	2
450–460 ms† (in males)	1
Torsade de pointes‡	2
T-wave alternans	1
Notched T wave in 3 leads	1
Low heart rate for age§	0.5
Clinical history	
Syncope	
With stress	2
Without stress	1
Congenital deafness	0.5
Family history‖	
Family members with "definite" long QT syndrome¶	1
Unexplained sudden cardiac death at age <30 y among immediate family members	0.5

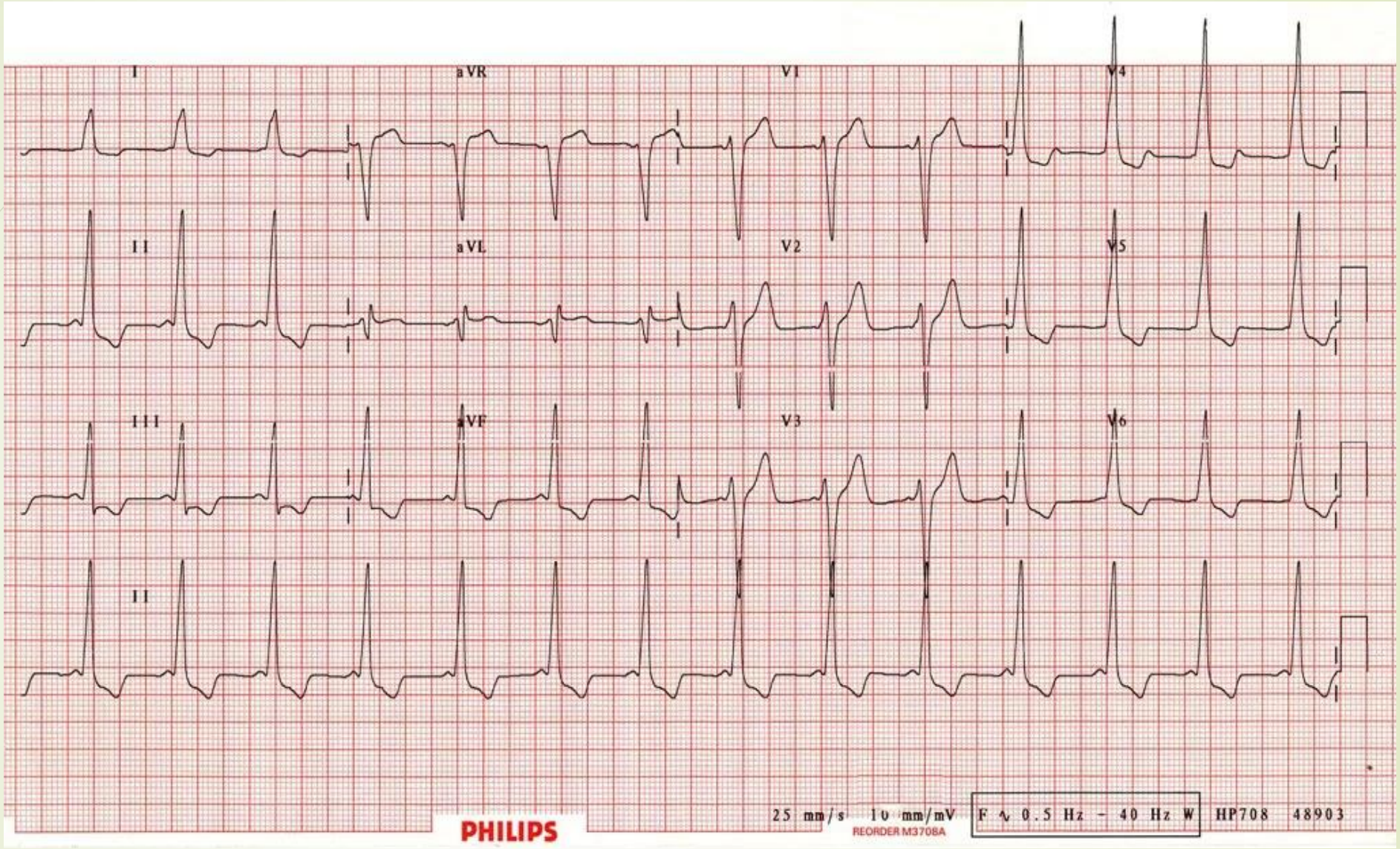
* Adapted from Schwartz PJ et al. Circulation. 1993;88:782-4 (47), with permission.
† Corrected QT calculated with Bazett formula ($QTc = QT/\sqrt{RR}$).
‡ No points if patient is taking drugs to favor QT prolongation.
§ Resting heart rate below second percentile for age (48).
‖ The same family member cannot be counted in both family history criteria.
¶ "Definite" long QT syndrome is score ≥4. Scoring: ≤1 point = low probability of long QT syndrome; 2–3 points = intermediate probability.

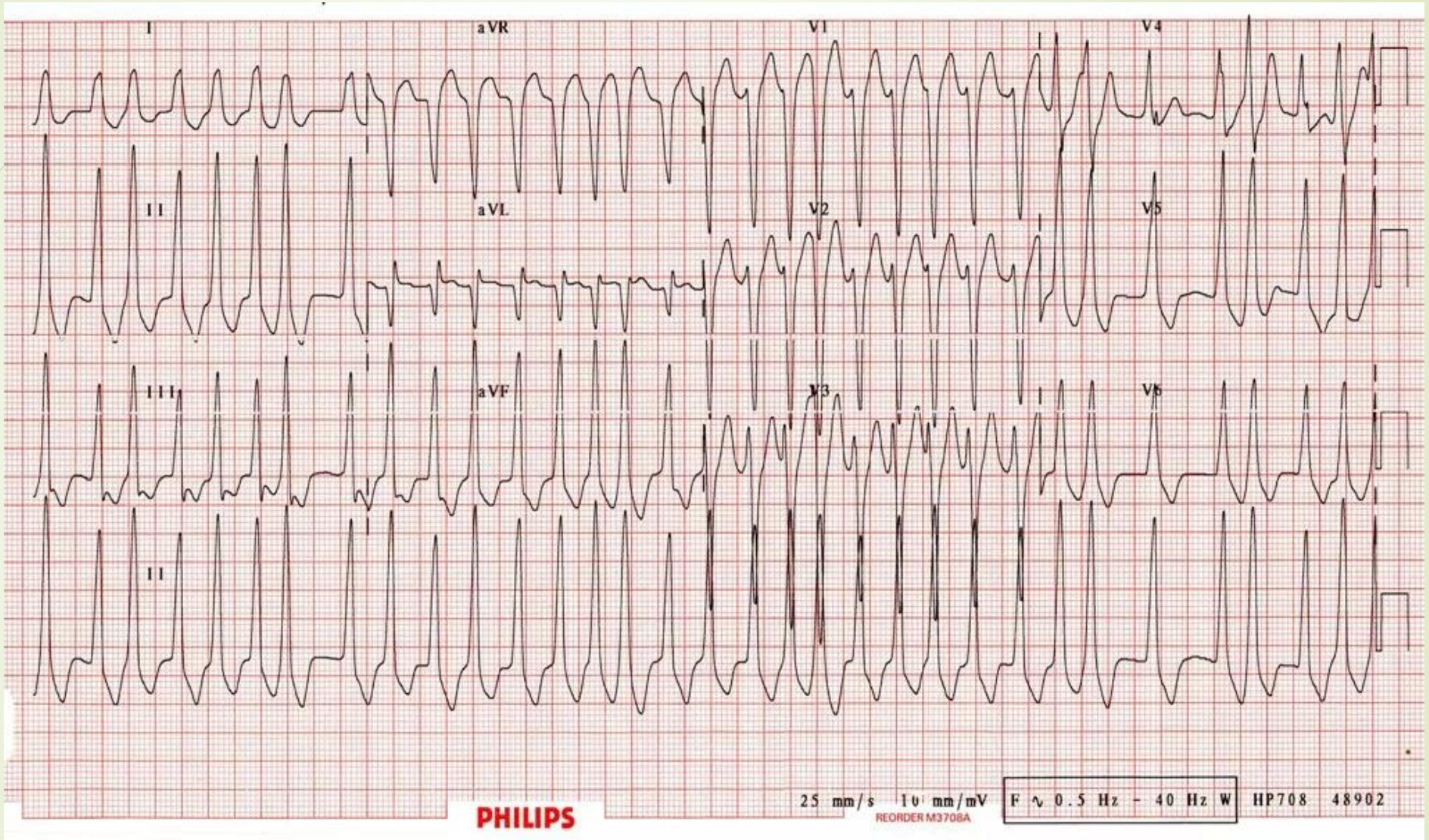
Type	Current	Functional Effect	Frequency Among LQTS	ECG[12,13]	Triggers Lethal Cardiac Event[10]	Penetrance*
LQTS1	K	↓	30%-35%		Exercise (68%) Emotional Stress (14%) Sleep, Repose (9%) Others (19%)	62%
LQTS2	K	↓	25%-30%		Exercise (29%) Emotional Stress (49%) Sleep, Repose (22%)	75%
LQTS3	Na	↑	5%-10%		Exercise (4%) Emotional Stress (12%) Sleep, Repose (64%) Others (20%)	90%


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

Wolff-Parkinson White



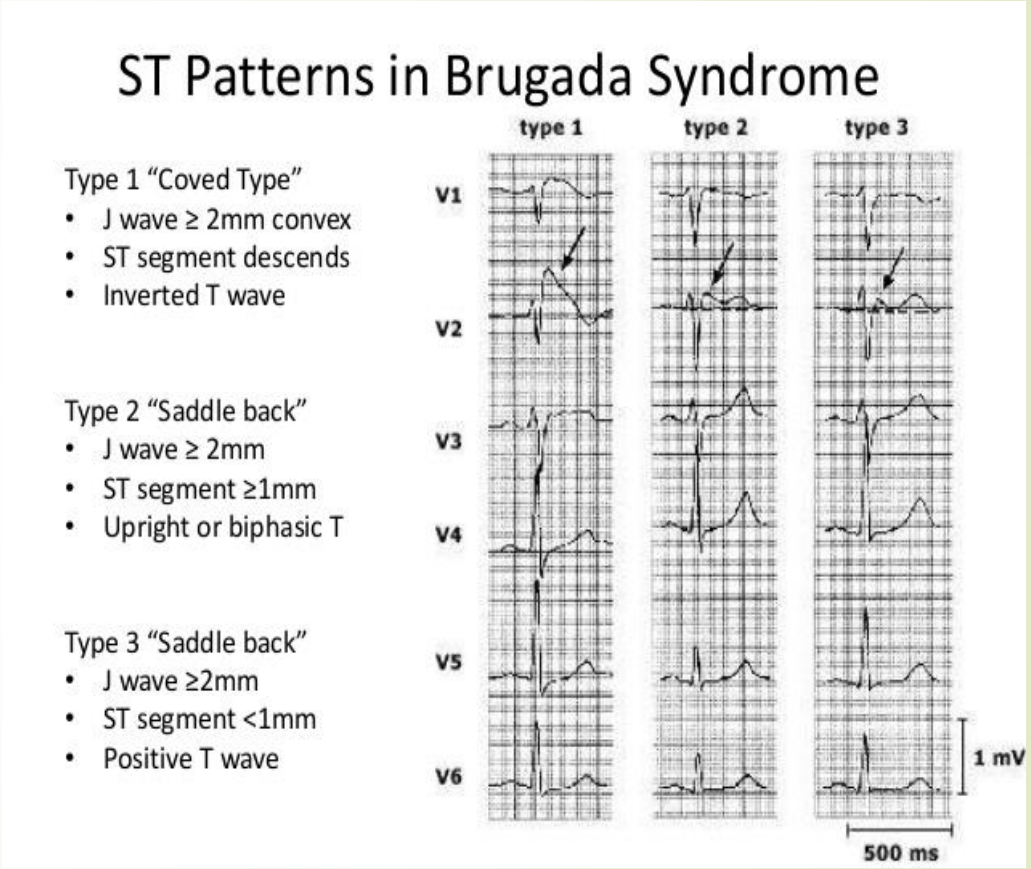

I
aVR
V1
V4
II
aVL
V2
V5
III
aVF
V3
V6
II
25 mm/s 10 mm/mV
F ∿ 0.5 Hz - 40 Hz W
HP708 48903
PHILIPS
REORDER M3708A


I
aVR
V1
V4
II
aVL
V2
V5
III
aVF
V3
V6
II
PHILIPS
25 mm/s 10 mm/mV
F ∿ 0.5 Hz - 40 Hz W
HP708 48902
REORDER M3708A

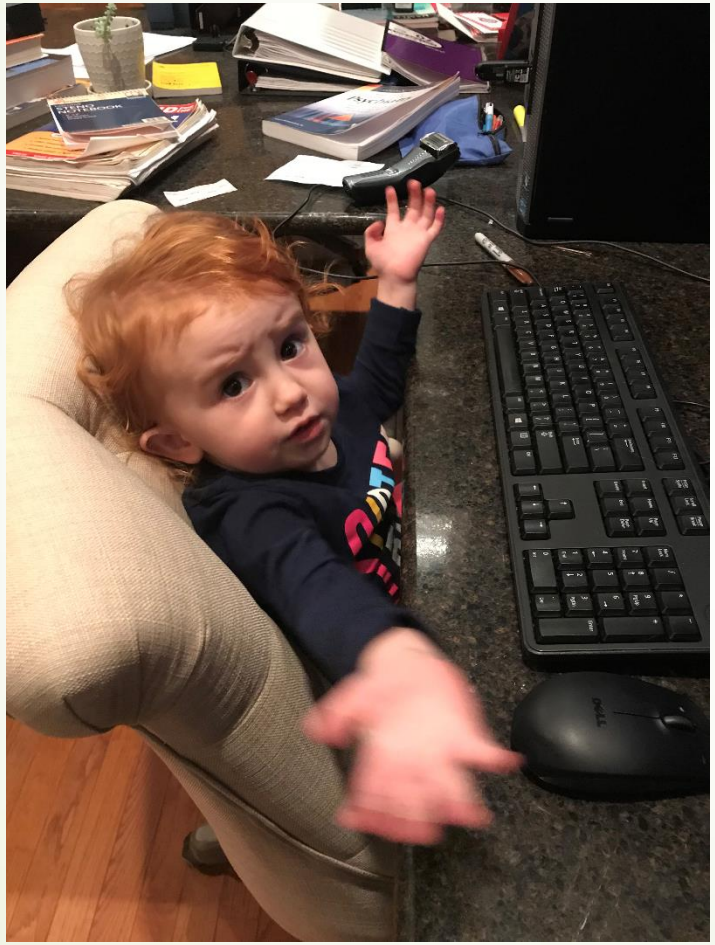
Brugada

- Presenting Sx: SCA, syncope
- Can predispose to VT/VF
- Fever may exacerbate sx
- No clear association with SCD

and exercise though has been Suggested. Restricted if phenotype Positive →



Screening



Outcomes of Cardiac Screening in Adolescent Soccer Players (NEJM)

Malhotra A, Dhutia H, Finocchiaro G, Gati S, Beasley I, Clift P, Cowie C, Kenny A, Mayet J, Oxborough D, Patel K, Pieles G, Rakhit D, Ramsdale D, Shapiro L, Somauroo J, Stuart G, Varnava A, Walsh J, Yousef Z, Tome M, Papadakis M, Sharma S.

- Study took from place from 1996-2016 looking at 11,168 adolescent football (soccer) players
- Mean age 16.4 +/- 1.2 (95% male)
- Health questionnaire, physical examination, EKG, echo
- 42 adolescents (0.4%) found to have cardiac d/o's associated with sudden death
- 225 athletes (2%) congenital or valvular abnormalities were identified
- 23 total deaths, 8 were cardiac related (7/8 cardiomyopathy)
- *6/8 had normal screening, mean time of death after screening ~7 years*

EKG Screening

TABLE 2.

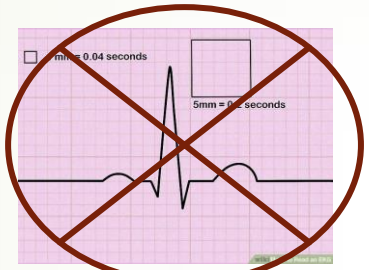
Outline of Pros and Cons for Inclusion of Electrocardiogram into the Preparticipation Examination

Pro	Con
• Reduce risk of sudden cardiac death in higher prevalence populations • Identify high-risk people that would not be discovered with history and physical examination alone • EKG screening itself is low cost, portable, and meets criteria for being cost-effective • False-positive rates may be reduced with use of athlete-specific EKG interpretation guidelines	• Actual sudden cardiac death incidence in school-aged athletes is currently thought to be too low to warrant mass EKG screening • Uncertain if EKG screening has the ability to save lives • False-positive rates may create significant financial and emotional costs •Lack of agreement on EKG interpretation may lead to additional cardiac evaluation and increased costs

Abbreviation: EKG, electrocardiogram.

EKG Screening

- AHA/ACC: H&P



- ESC/FIFA/NCAA/IOC: H&P + EKG

EKG screening

- Trends in sudden cardiovascular death in young competitive athletes after implementation of a preparticipation screening program.

  Corrado D., Basso C., Pavei A., Michieli P., Schiavon M., Thiene G. (2006)

- Looked at athletes age 12-35 from 1979 to 2004
- Annual incidence of sudden cardiac death decreased by 89% in the EKG screened population
- 2% of screened athletes were disqualified
- Risk reduction was thought to be secondary to finding *cardiomyopathies*

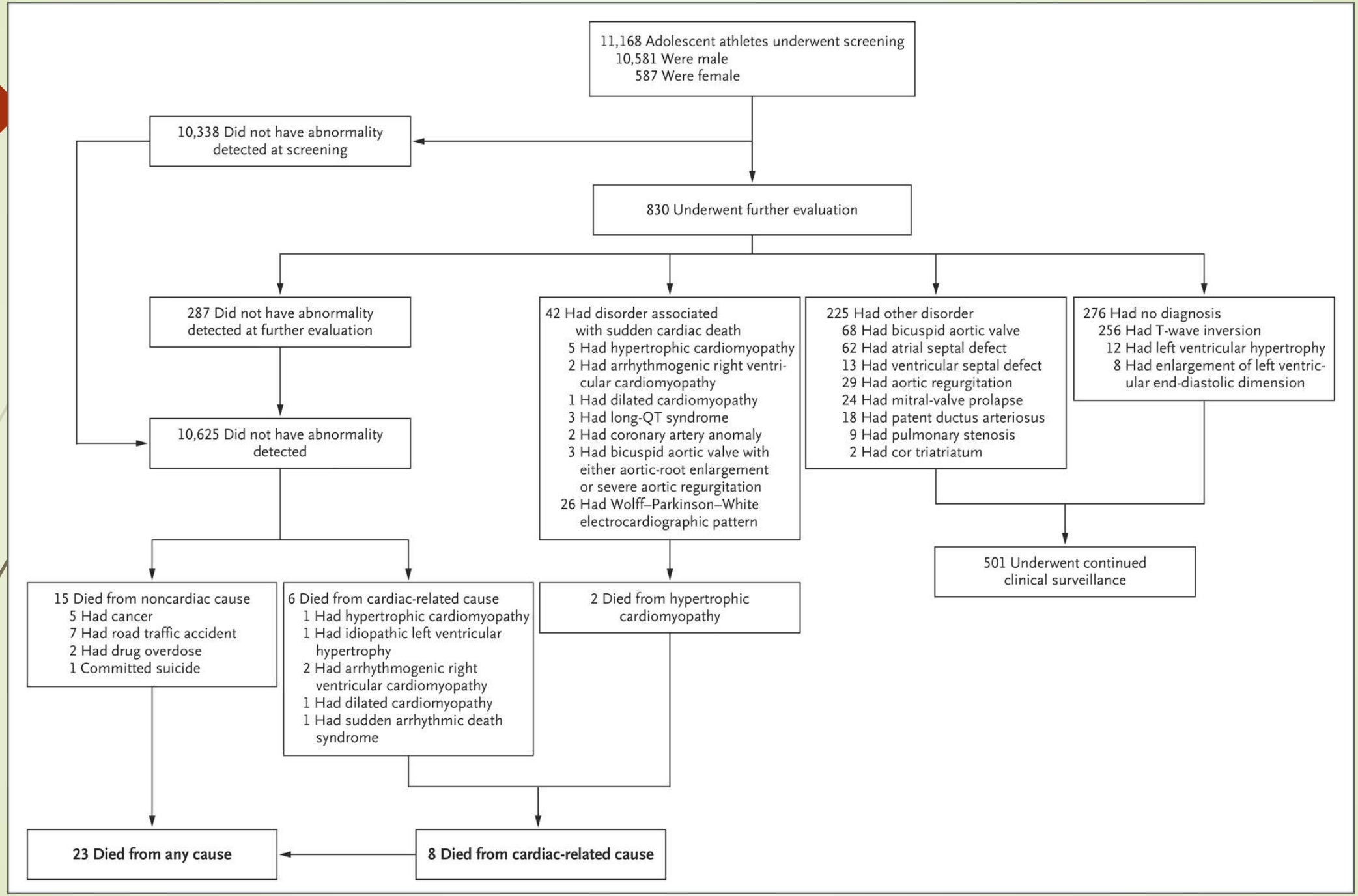

11,168 Adolescent athletes underwent screening
10,581 Were male
587 Were female
10,338 Did not have abnormality detected at screening
830 Underwent further evaluation
287 Did not have abnormality detected at further evaluation
42 Had disorder associated with sudden cardiac death
5 Had hypertrophic cardiomyopathy
2 Had arrhythmogenic right ventricular cardiomyopathy
1 Had dilated cardiomyopathy
3 Had long-QT syndrome
2 Had coronary artery anomaly
3 Had bicuspid aortic valve with either aortic-root enlargement or severe aortic regurgitation
26 Had Wolff–Parkinson–White electrocardiographic pattern
225 Had other disorder
68 Had bicuspid aortic valve
62 Had atrial septal defect
13 Had ventricular septal defect
29 Had aortic regurgitation
24 Had mitral-valve prolapse
18 Had patent ductus arteriosus
9 Had pulmonary stenosis
2 Had cor triatriatum
276 Had no diagnosis
256 Had T-wave inversion
12 Had left ventricular hypertrophy
8 Had enlargement of left ventricular end-diastolic dimension
10,625 Did not have abnormality detected
501 Underwent continued clinical surveillance
15 Died from noncardiac cause
5 Had cancer
7 Had road traffic accident
2 Had drug overdose
1 Committed suicide
6 Died from cardiac-related cause
1 Had hypertrophic cardiomyopathy
1 Had idiopathic left ventricular hypertrophy
2 Had arrhythmogenic right ventricular cardiomyopathy
1 Had dilated cardiomyopathy
1 Had sudden arrhythmic death syndrome
2 Died from hypertrophic cardiomyopathy
23 Died from any cause
8 Died from cardiac-related cause

Table 1. Clinical Features and Outcomes in Athletes with Cardiac Conditions That Are Associated with Sudden Cardiac Death.*

Condition, Sex, and Age	Race†	History and Examination	ECG Result	Echocardiography Result	LGE on Cardiac MRI	Exercise Test Result	Genetic Test Result‡	Outcome
HCM								
M, 16 yr	White	Negative	TWI (leads II, aVF, V2–V6)	LVWT 16 mm (asymmetric septal hypertrophy)	Yes	Normal	*MYBPC3* mutation	Advised not to play
M, 15 yr	White	Negative	TWI (leads V2–V6), LAD, isoelectric ST segments	LVWT 15 mm (asymmetric septal hypertrophy)	No	Normal	*MYBPC3* mutation	Advised not to play
M, 16 yr	White	Negative	TWI (leads II, III, aVF), ST depression	Apical hypertrophy	No	Normal	*MYH7* mutation	Advised not to play
M, 16 yr	Black	Negative	TWI (leads V1–V5), ST depression	LVWT 16 mm (asymmetric septal hypertrophy)	Yes	Normal	Negative	Advised not to play
M, 16 yr	Mixed	Negative	TWI (leads V4–V6), isoelectric ST segments	Apical hypertrophy	No	Normal	Negative	Advised not to play
ARVC								
M, 16 yr	White	Palpitations	TWI (leads V1–V3)	Reduced LV systolic function; dilated and aneurysmal RV	Yes	Ventricular ectopy of LBBB morphology	Negative	Advised not to play
M, 17 yr	White	Negative	Normal	Aneurysmal RV with hypokinetic free wall	No	Ventricular ectopy of LBBB morphology	*PKP2* mutation	Advised not to play
DCM								
M, 16 yr	White	Dyspnea	TWI (leads V1–V4), ST depression	LVEDD, 61 mm; EF, 45%	Yes	LV ejection fraction did not increase with exercise	Negative	Advised not to play
LQTS								
F, 16 yr	White	Negative	QTc, 510 msec	Normal	NA	QTc, >500 msec	*KCNQ1* mutation	Advised not to play
M, 15 yr	White	Negative	QTc, 503 msec	Normal	NA	QTc, >500 msec	Negative	Advised not to play
M, 16 yr	White	Negative	QTc, 490 msec	Normal	NA	Paradoxical increase in QTc during recovery	*KCNQ1* mutation	Advised not to play
CAA								
M, 16 yr	White	Negative	Normal	Left coronary artery arising from right sinus of Valsalva	NA	Positive for myocardial ischemia	NA	Underwent corrective surgery and returned to play
M, 15 yr	White	Negative	Normal	Right coronary artery arising from left sinus of Valsalva with adverse course.	NA	Normal	NA	Underwent corrective surgery and returned to play
BAV								
M, 16 yr	Black	Dyspnea, diastolic murmur	Left axis deviation	Fusion of right and left coronary cusps and severe aortic regurgitation; LVEDD, 60 mm	No	Terminated prematurely because of fatigue	NA	Underwent corrective surgery and returned to play
M, 17 yr	White	Negative	Normal	Fusion of right and noncoronary cusps with mixed aortic valve disease; diameter of aortic root at sinuses of Valsalva, 53 mm	No	Normal	NA	Underwent corrective surgery and returned to play
M, 16 yr	White	Diastolic murmur	Normal	Fusion of right and left coronary cusps and severe aortic regurgitation; LVEDD, 63 mm	No	Normal	NA	Underwent corrective surgery and returned to play

* ARVC denotes arrhythmogenic right ventricular cardiomyopathy, BAV bicuspid aortic valve, CAA coronary-artery anomaly, DCM dilated cardiomyopathy, ECG electrocardiogram, EF ejection fraction, HCM hypertrophic cardiomyopathy, LAD left axis deviation, LBBB left bundle-branch block, LGE late gadolinium enhancement, LQTS long-QT syndrome, LV left ventricular,

Table 2. Summary of Cardiac Conditions Detected According to Screening Tool.

Condition	No. of Athletes	No. of Athletes with Abnormal Result			
		History	Examination	ECG	Echocardiography
Any cardiac condition	267	6	76	84	237
Condition associated with sudden cardiac death	42	3	2	36	12
Hypertrophic cardiomyopathy	5	0	0	5	5
Arrhythmogenic right ventricular cardiomyopathy	2	1	0	1	2
Dilated cardiomyopathy	1	1	0	1	1
Coronary-artery anomalies	2	0	0	0	2
Bicuspid aortic valve–associated disease*	3	1	2	0	3
Long-QT syndrome	3	0	0	3	0
Wolff–Parkinson–White ECG pattern	26	0	0	26	0
Other cardiac condition	225	3	74	48	225
Bicuspid aortic valve	68	1	32	15	68
Atrial septal defect	62	1	6	26	62
Aortic regurgitation	29	0	16	2	29
Mitral-valve prolapse	24	0	12	3	24
Patent ductus arteriosus	18	0	1	1	18
Ventricular septal defect	13	0	3	1	13
Pulmonary stenosis	9	1	4	0	9
Cor triatriatum	2	0	0	0	2

* Bicuspid aortic valve–associated disease includes bicuspid aortic valve with either aortic-root enlargement or severe aortic regurgitation.

EKG screening

- *Inter-Rater Reliability and Downstream Financial Implications of Electrocardiography Screening in Young Athletes*

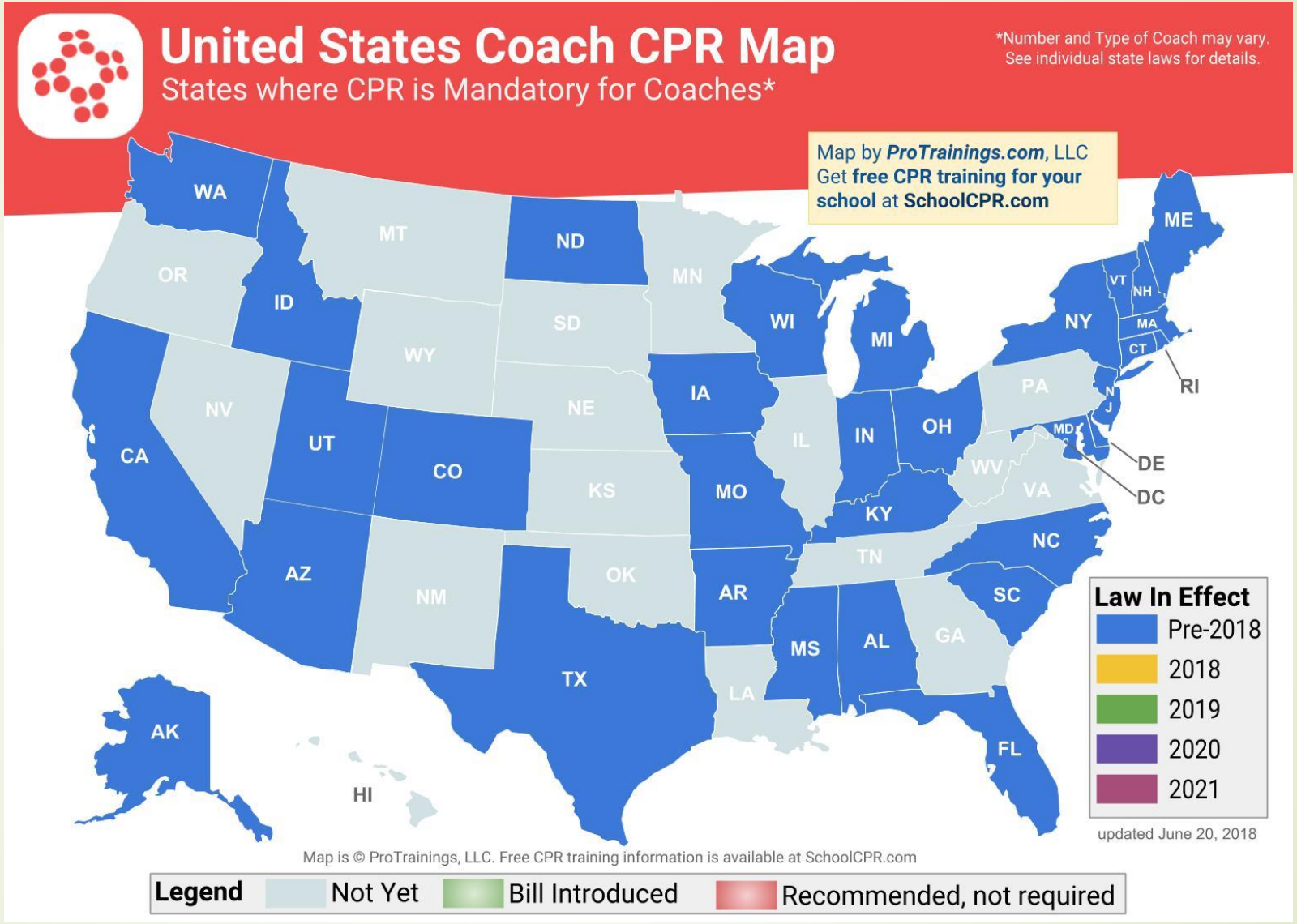
  Harshil Dhutia, Aneil Malhotra, Tee Joo Yeo, Irina Chis Ster, Vincent Gabus, Alexandros Steriotis, Helder Dores, Greg Mellor, Carmen García-Corrales, Bode Ensam, Viknesh Jayalapan, Vivienne Anne Ezzat, Gherardo Finocchiaro, Sabiha Gati, Michael Papadakis, Maria Tome-Esteban, Sanjay Sharma (2017).

  - There is only moderate interobserver reliability for ECG interpretation even among cardiologists with experience in the cardiovascular evaluation of young athletes.
  - Modification of ECG interpretation criteria improves reliability among inexperienced cardiologists.
  - The decision to propose secondary investigations after ECG interpretation varies among inexperienced and experienced cardiologists, respectively, with significant downstream financial implications.
  - The findings of this study highlight that formal training and development of standardized diagnostic pathways are essential to support cardiologists involved in cardiovascular screening of young athletes.

EKG Screening

- *Cardiac screening to prevent sudden death in young athletes.*

  Schmehil C, Malhotra D, Patel DR (2017)
- Limitations to current AHA guidelines
- 25% of population has 1/14 points on AHA checklist
- 6% had abnormal criteria based on Seattle criteria, 8% Stanford criteria, 26% ESC criteria
- None of recent study of 1596 patients were found to have a condition excluding them from sports
- If SCD is the presenting sx, checklist doesn't work
- Meta-anaylsis of AHA guidelines found 20% sensitivity and 94% specificity
- Estimated EKG screening would cost $2.5-3.5 Billion and 2 lives saved per 1,000 athletes


United States Coach CPR Map
States where CPR is Mandatory for Coaches*
*Number and Type of Coach may vary.
See individual state laws for details.
Map by ProTrainings.com, LLC
Get free CPR training for your school at SchoolCPR.com
WA
OR
CA
ID
NV
MT
WY
UT
AZ
CO
NM
ND
SD
NE
KS
OK
TX
MN
IA
MO
AR
LA
WI
IL
MS
MI
IN
KY
TN
AL
OH
WV
GA
FL
SC
NC
VA
PA
NY
MD
DE
DC
NJ
CT
RI
MA
VT
NH
ME
AK
HI
Law In Effect
Pre-2018
2018
2019
2020
2021
updated June 20, 2018
Map is © ProTrainings, LLC. Free CPR training information is available at SchoolCPR.com
Legend
Not Yet
Bill Introduced
Recommended, not required

Want to save lives?

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