SUDDEN CARDIAC DEATH IN YOUNG ATHLETES

Dr. Arnold M. Einhorn, MD FACC, FACP

DEMOGRAPHICS OF SUDDEN CARDIAC DEATH IN ATHLETES

- 9:1 male to female ratio
- More commonly in African Americans in the US
- Younger than 35 years
  1. Hypertrophic obstructive cardiomyopathy (~35%)
  2. Congenital coronary anomalies (15-20%)
  3. Myocarditis, aortic stenosis, aortic dissection/rupture, ion channelopathies, coronary atherosclerosis, ARVC (each 5% or less)
- Older than 35 years
  1. Coronary atherosclerosis

DEMOGRAPHICS OF SUDDEN CARDIAC DEATH IN ATHLETES

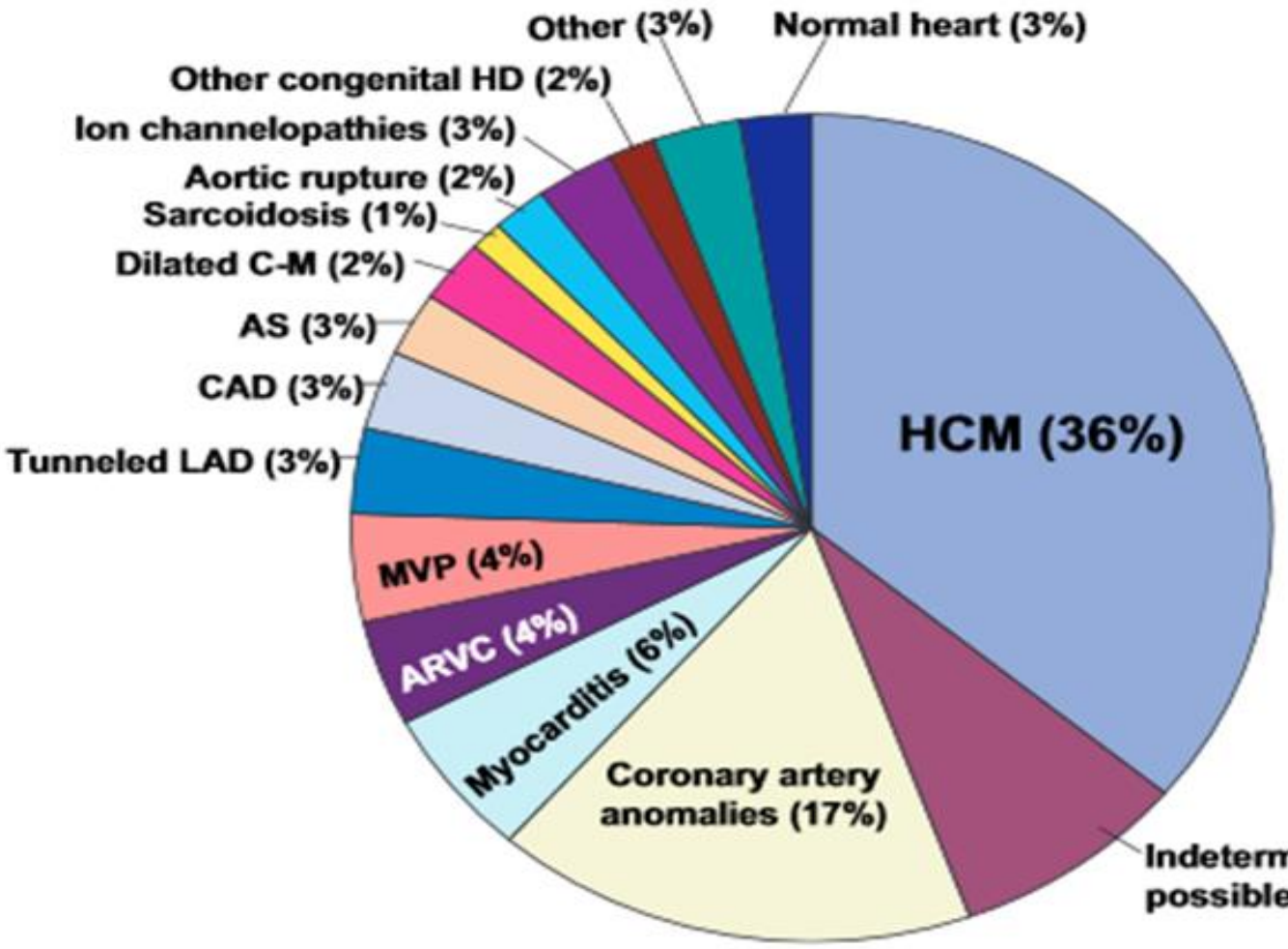


Figure. Distribution of cardiovascular causes of sudden death in 1435 young competitive athletes. From the Minneapolis Heart Institute Foundation Registry, 1980 to 2005. ARVC indicates arrhythmogenic right ventricular cardiomyopathy; AS, aortic stenosis; CAD, coronary artery disease; C-M, cardiomyopathy; HD, heart disease; LAD, left anterior descending; LVH, left ventricular hypertrophy; and MVP, mitral valve prolapse.

HYPERTROPHIC CARDIOMYOPATHY

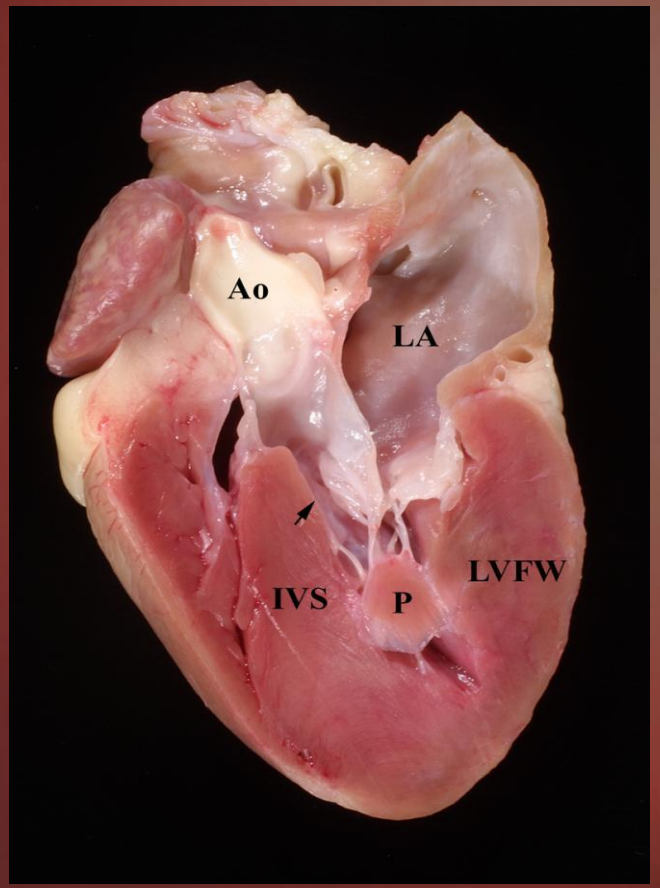
HYPERTROPHIC CARDIOMYOPATHY (HCM)

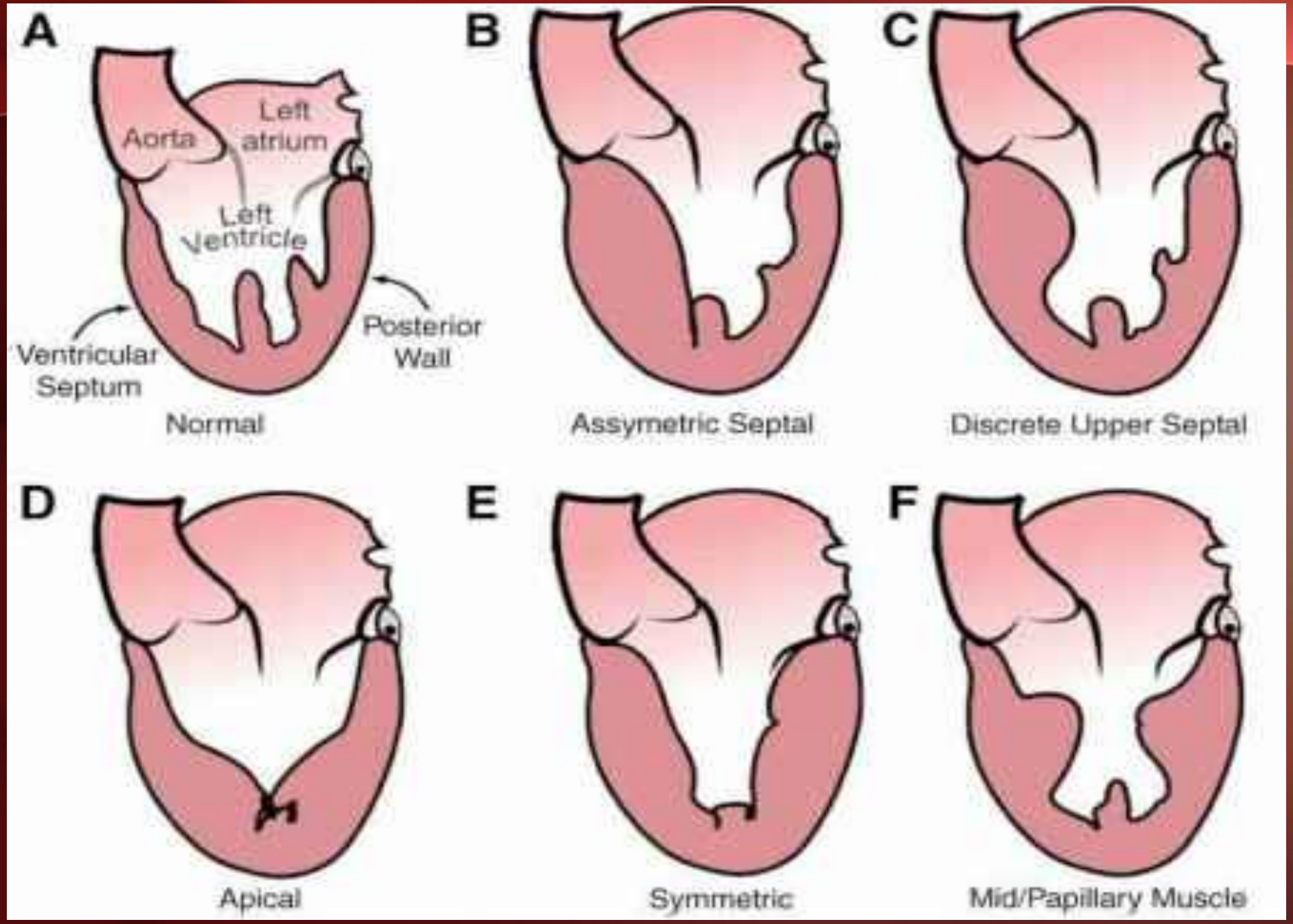
- Prevalence 1 in 500

  Leading cause of non-violent death in young people in the U.S.

HYPERTROPHIC CARDIOMYOPATHY

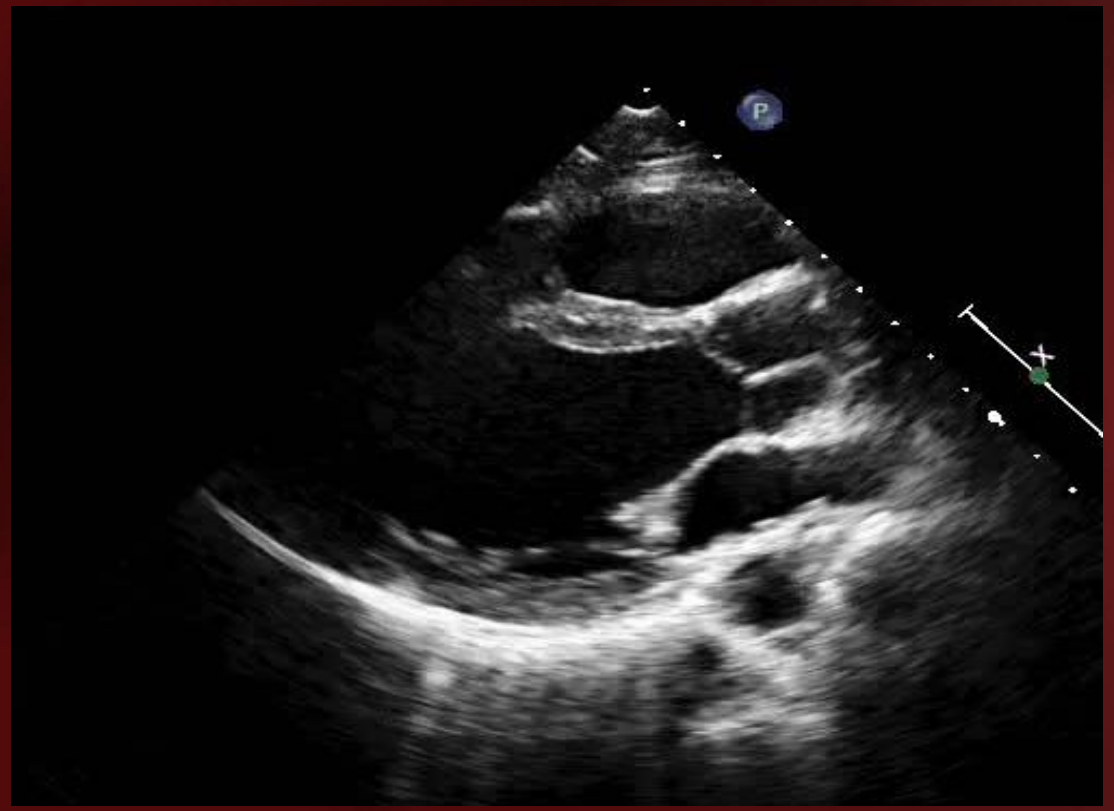
- LVH in the absence of a cause
- LV wall thickness ≥ 15mm
  - Concentric
  - Apical (Yamaguchi's disease) 25%
  - Free Wall
  - Septal 75%
    - Idiopathic hypertrophic sub aortic stenosis
    - Asymmetric septal hypertrophy



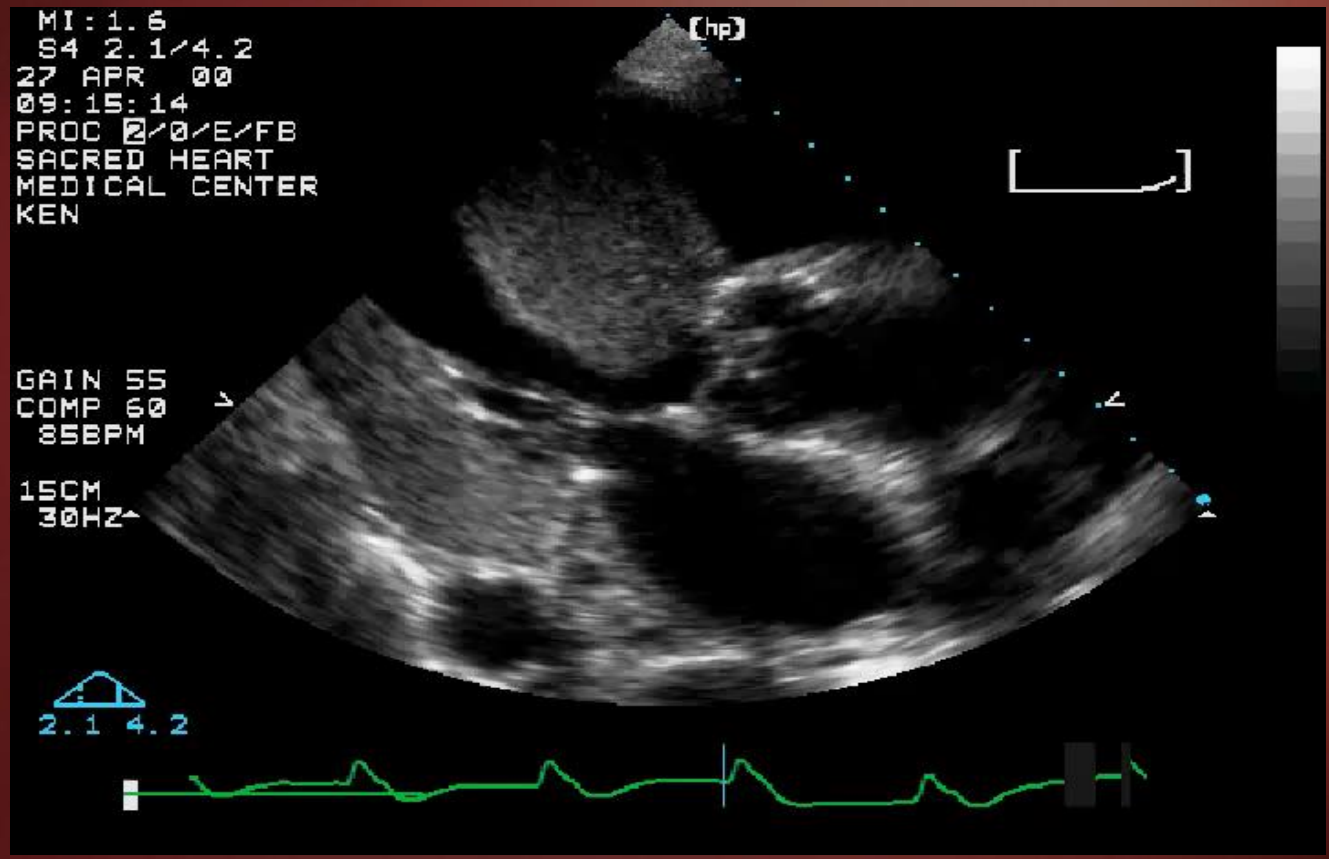

A
Aorta
Left atrium
Left Ventricle
Ventricular Septum
Posterior Wall
Normal
B
Assymetric Septal
C
Discrete Upper Septal
D
Apical
E
Symmetric
F
Mid/Papillary Muscle

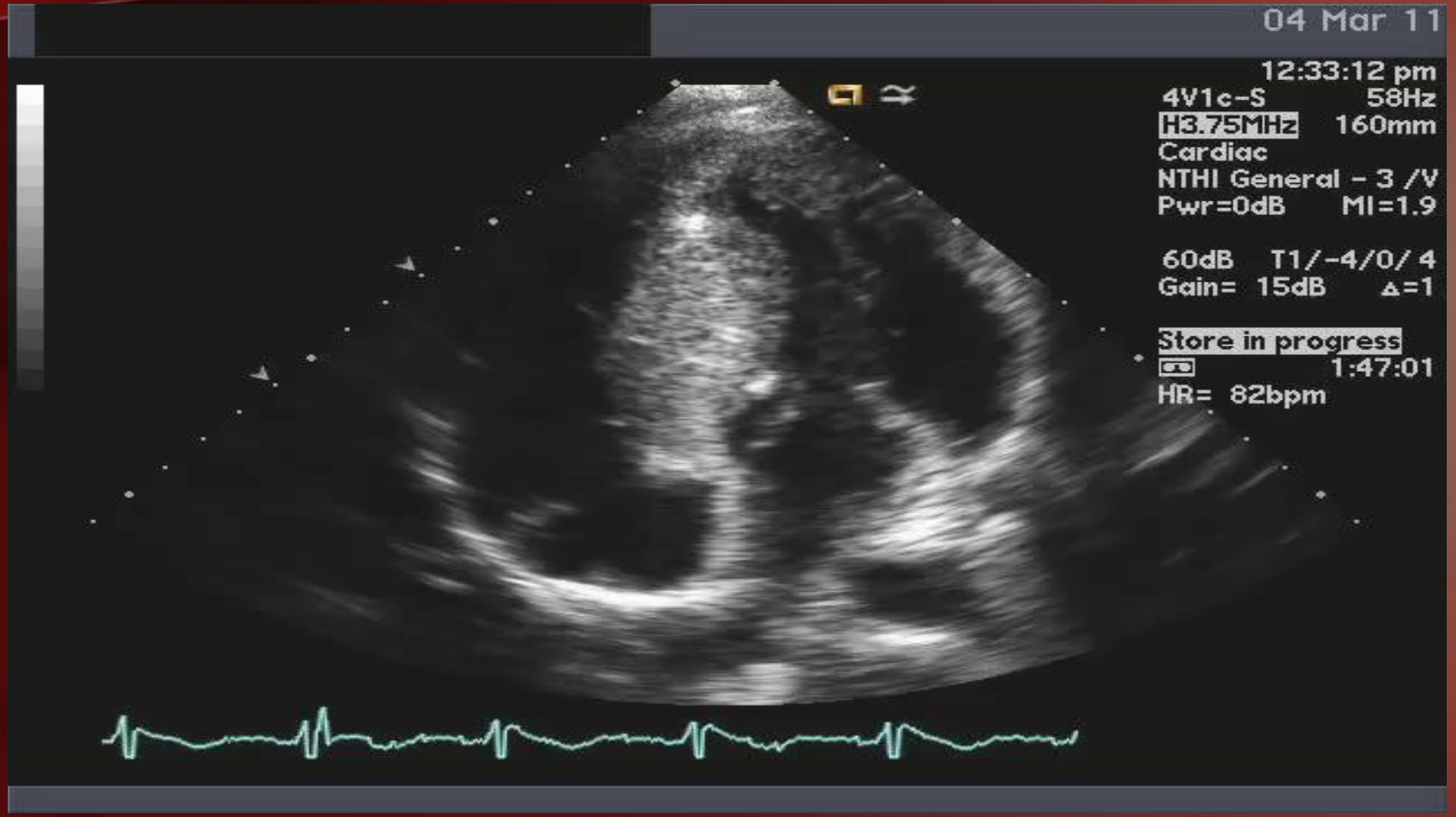
ECHOCARDIOGRAPHY

- Normal



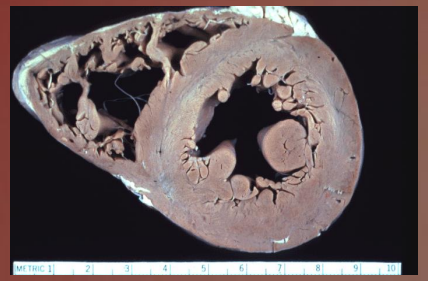
- HCM



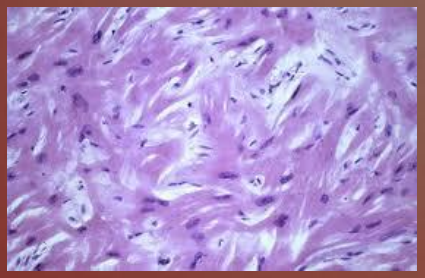

04 Mar 11
12:33:12 pm
4V1c-S 58Hz
H3.75MHz 160mm
Cardiac
NTHI General - 3 /V
Pwr=0dB MI=1.9
60dB T1/-4/0/ 4
Gain= 15dB Δ=1
Store in progress
1:47:01
HR= 82bpm

HYPERTROPHIC CARDIOMYOPATHY

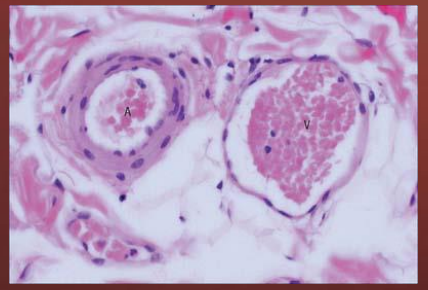
- LV hypertrophy- any pattern



- Myocyte disarray



- Vascular wall thickening of intramuscular arterioles



HCM VS "ATHLETE'S HEART"

- Regular intensive physical training in some endurance sports is associated with an increase in LV wall thickness and cavity size.
- Gender (male) and sport (rowers) specific
- Always associated with LV cavity enlargement
- LV wall is < 16mm thickness
- LVH resolves (over months) when exercise ceases

Parameter	HCM	Athlete's heart
LV wall thickness	Can be > 16 mm. Asym	< 16 mm. sym
LVDD	<45 mm	> 55 mm
LA size	Enlarged	Normal
LV filling pattern	Impaired	Normal
Deconditioning	No change	LV wall decreases
EKG	High QRS, Qs, deep neg. T	LVH but not unusual
Family Hx of LVH	Present	Absent

RISK FACTORS FOR SUDDEN DEATH

- Major Risk Factors
  - Unexplained syncope
  - Prior cardiac arrest
  - Family history of SCD
  - Sustained VT
  - Nonsustained VT (Holter)
  - LV thickness > 29 mm
  - Abnl exercise BP response
  - > 15 % late gad enhacement of LV
- Possible Risk Factors
  - Atrial fibrillation (25% have it)
  - Ischemia
  - LV outflow obstruction
  - High-risk genetic mutation
  - Intense (Competitive Physical exertion

EVALUATION

Class I- ECG yearly, Holter at baseline

Echo at baseline

Echo every 12-18 months for kids age 12 or earlier if playing sports or history of SCD

MRI if echo inconclusive

Cath if chest pain and intermediate to high risk of CAD

Class IIa- Holter yearly, echo yearly to evaluate function and gradient

Treadmill to risk stratify, stress echo if gradient <50 to assess for exercise induced obstruction

Class III- Echo more than ever 1 year

HCM: MANAGEMENT

Asymptomatic: Mange comorbidities (I)

Low intensity exercise is reasonable (IIa)

Mild symptoms: Beta-blockers to HR 60 bpm (I)

Verapamil if still having symptoms (I)

Diuretics if non obstructive (IIa)

Disopyramide if still having Sx (IIa)

III- Dlhydropyridine CCBs if gradient, verapamil if low BP/rest Sx, digoxin, disopyramide, without BB, inotropes

Obstructive: Surgical myotomy/myectomy in experience hands (I)

(grad >50): Alcohol septal ablation (IIa)

Mitral valve replacement (III)

Dual chamber pacing (IIa- if already have pacer, Iib if other options available, III if no symptoms or nothing else tried)

Nonobstructive: Cardiac transplant if symptoms (I)

WHAT ABOUT RECREATIONAL SPORTS

Recommendations not applicable to pts with syncope, angina, arrhythmias, high risk status, myectomy or septal ablation.

Avoid

"burst" exertion-basketball, tennis

Adverse programs focused on achieving higher levels of conditioning and excellence

Intense isometric exertion-free weights

Extreme sports- hang gliding, bungee jumping

"intense competitive sports regardless of age, sex, race +/_ obstruction, prior septal reduction surgery, or presence of ICD" (III)

Okay

Bowling, golf, brisk walking, skating, (not ice hockey) (IIa)

Modest biking, hiking, tennis (doubles), swimming, treadmil/stationary bike

Sports which do not require systematic training or the pursuit of excellence and are without the pressure to excel against others.

ANOMALOUS ORIGIN OF CORONARY ARTERY

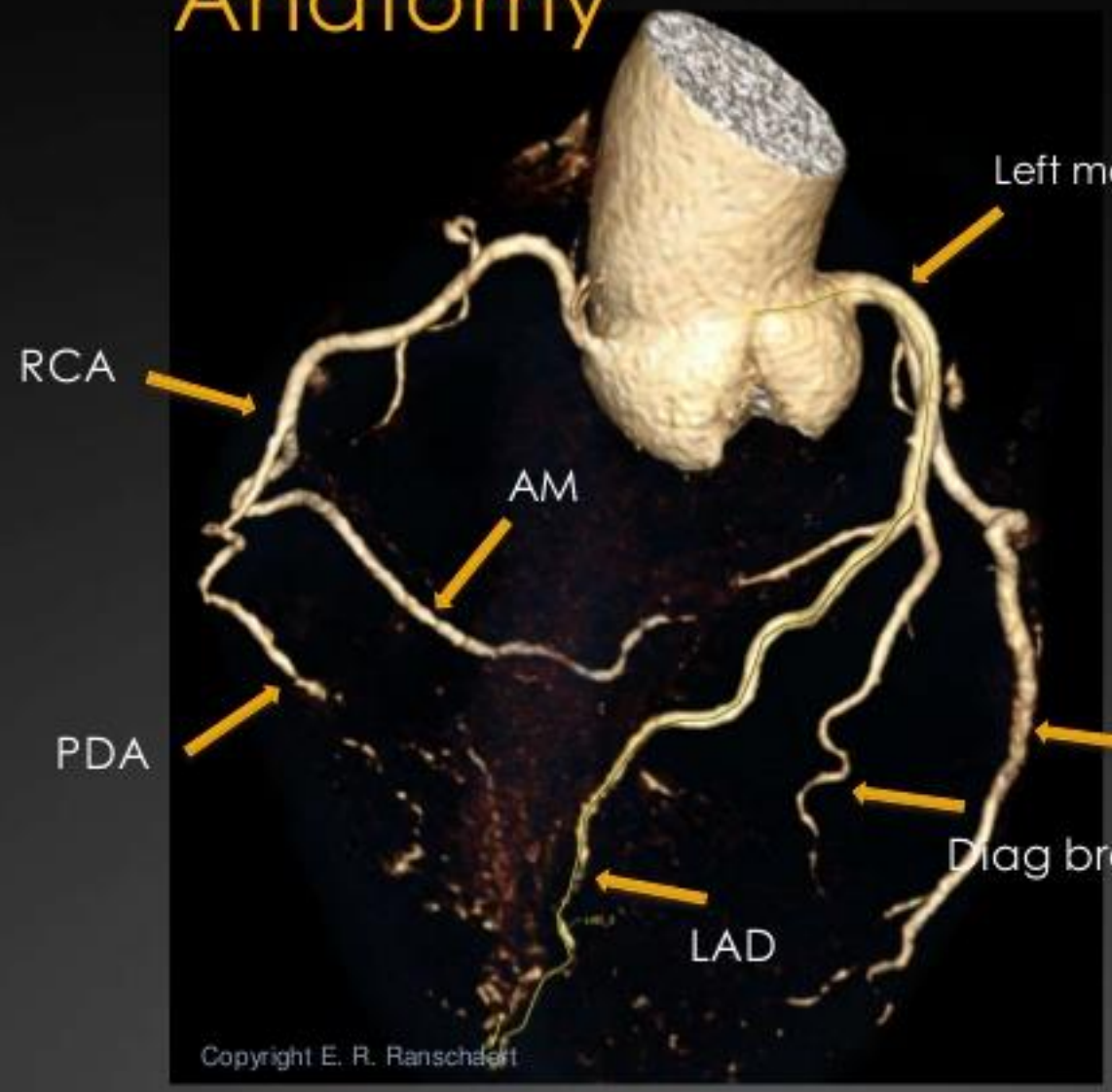
ANOMALOUS ORIGIN OF CORONARY ARTERY

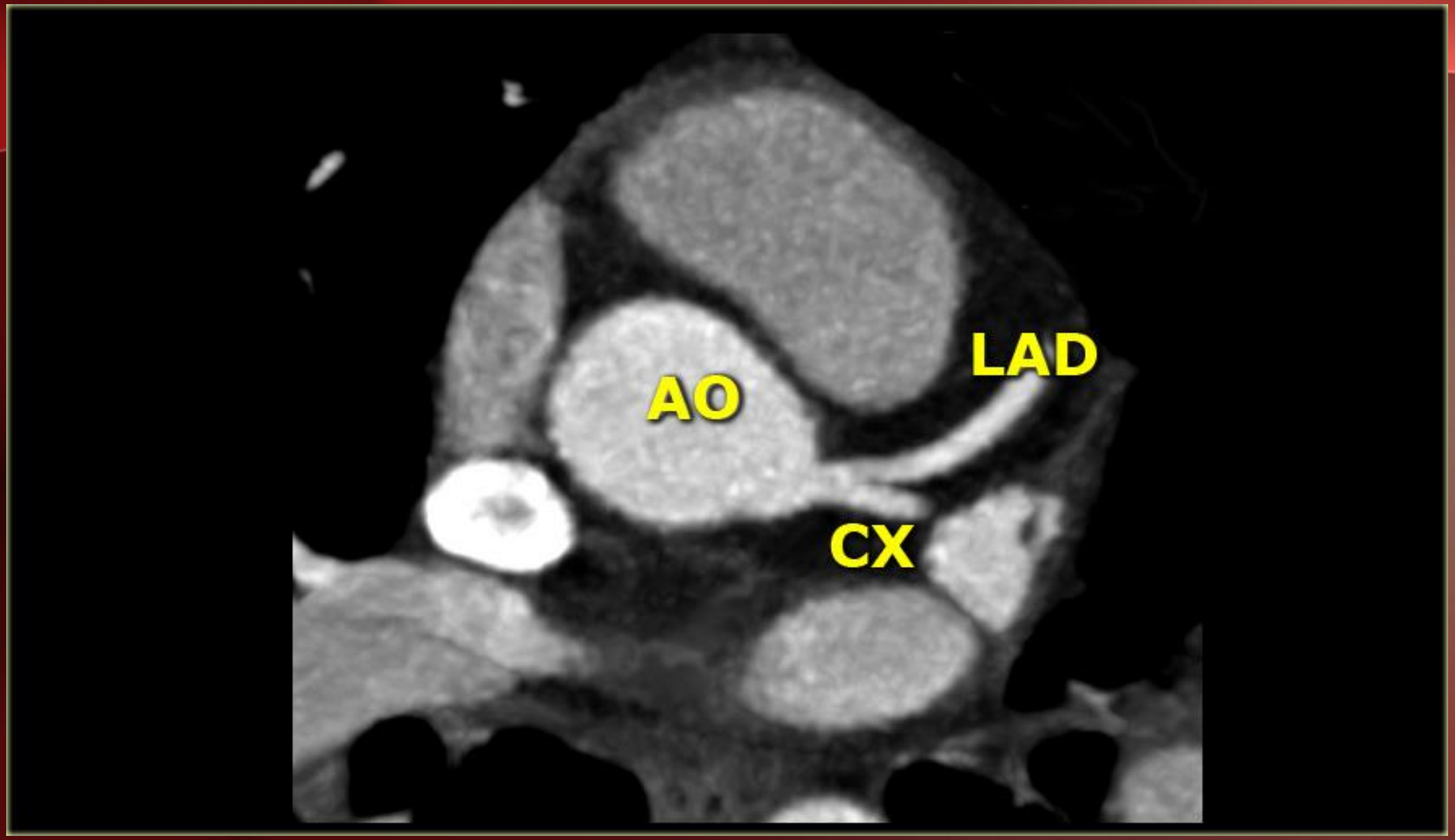
- According to the literature, coronary anomalies affect ≈1% of the general population
- It accounts for about 17% of the demographics for SCD in athletes

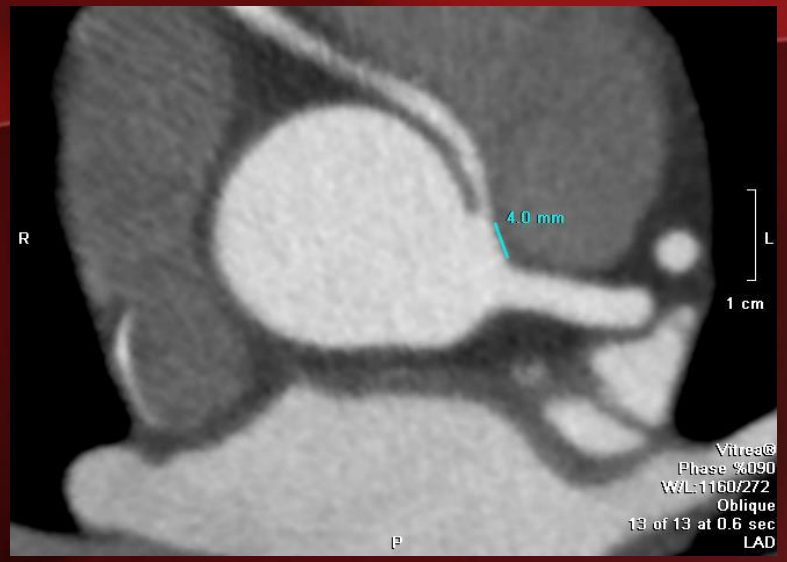
ANOMALOUS ORIGIN OF CORONARY ARTERY

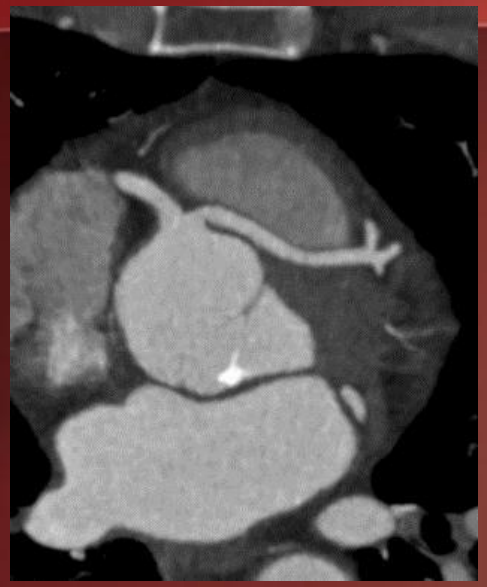
- HIGH RISK FEATURES FOR ANOMALOUS CORONARY ARTERY
- 1. RUNNING BETWEEN THE AORTA AND THE PULMONARY ARTERY
- 2. TUNNELING OF THE CORONARY ARTERY
- 3. THE SHAPE OF THE ANOMALOUS CORONARY ARTERY ORIFICE

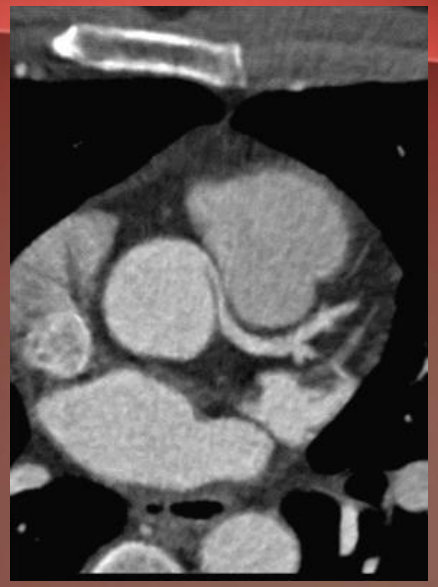
Anatomy

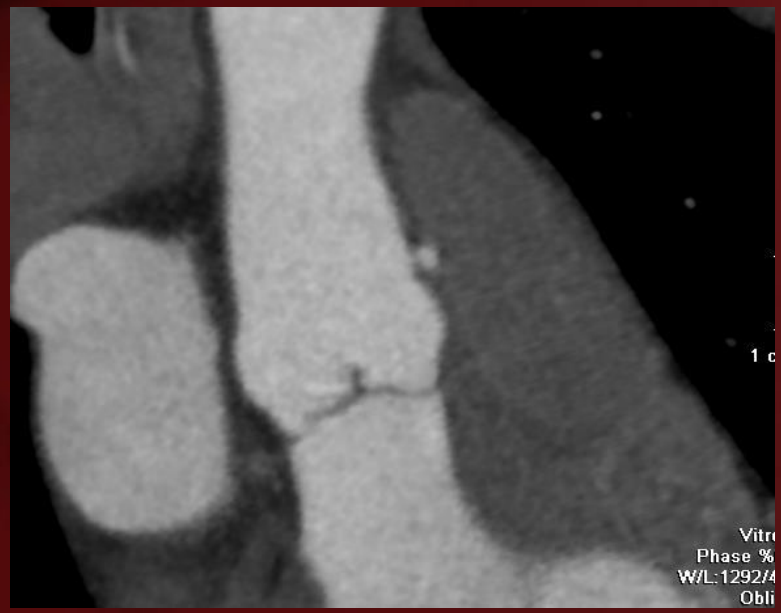

Left main stem
RCA
AM
PDA
Cx
Diag branch
LAD
Copyright E. R. Ranschaert

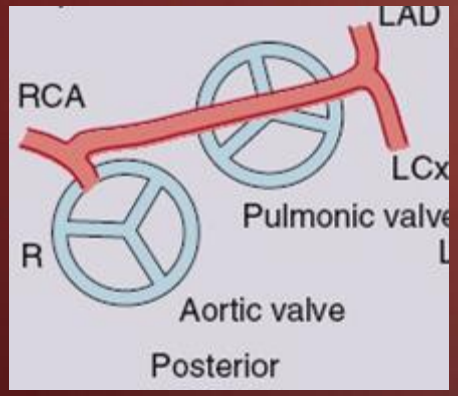

LAD
AO
CX

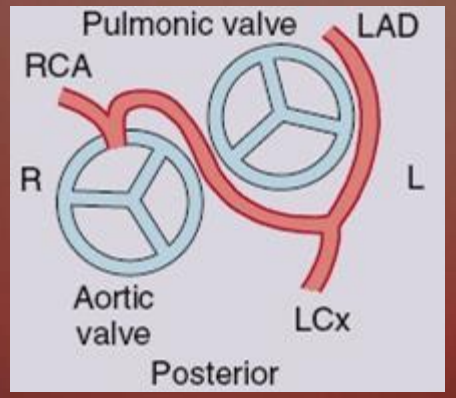

R
4.0 mm
L
1 cm
Vitrea®
Phase %090
W/L:1160/272
Oblique
13 of 13 at 0.6 sec
LAD

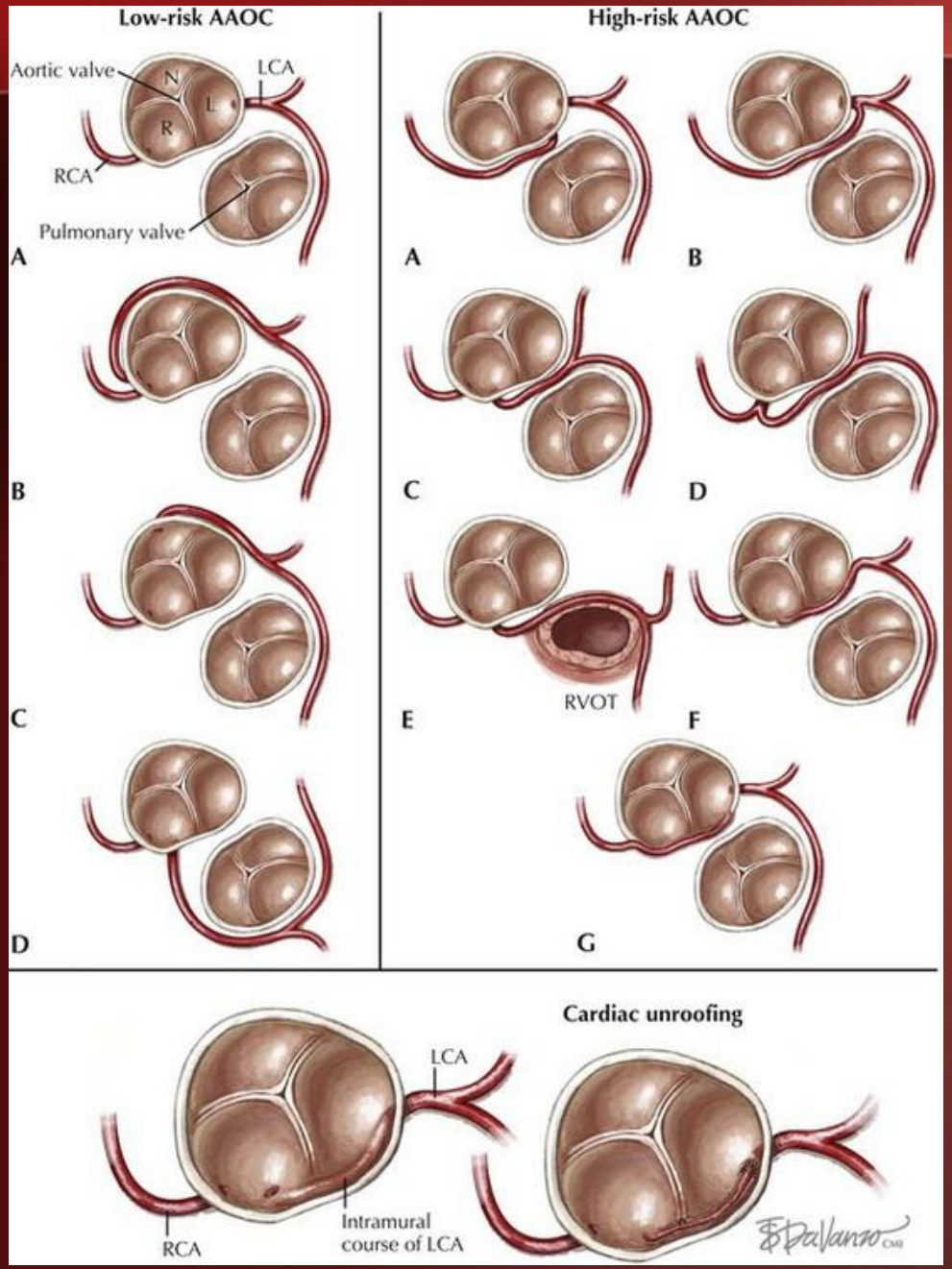





Vitr
Phase %
W/L:1292/4
Obli


LAD
RCA
LCx
Pulmonic valve
R
Aortic valve
Posterior


Pulmonic valve
LAD
RCA
R
L
Aortic valve
LCx
Posterior


Low-risk AAOC
High-risk AAOC
Aortic valve
N
L
R
LCA
RCA
Pulmonary valve
A
B
C
D
A
B
C
D
E
F
G
RVOT
Cardiac unroofing
LCA
RCA
Intramural
course of LCA

ANOMALOUS ORIGIN OF CORONARY ARTERY

- Therapy for anomalous origin of coronary artery
  - If high risk features are present then surgical intervention is usually the preferred therapy.

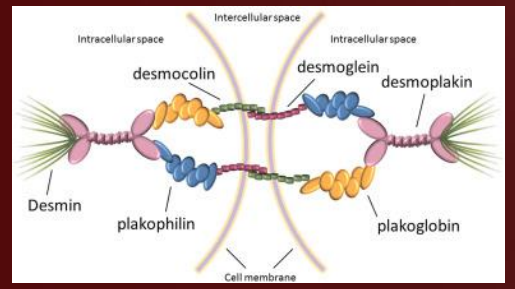
ARRHYTHMOGENIC RIGHT VENTRICULAR CARDIOMYOPATHY (ARVC)

ARVC

Disorder predominantly of the RV but can involve the LV

- Fibro-Fatty replacement of myocardium: basal TV, outflow tract, apex

Mostly autosomal dominant mutations of the cardiac demosome affecting gap junction and cell to cell adhesion (desmoplakin, plakophlin, plakoglobin and others)



Phases:

- Concealed Phase: asymptomatic but at risk for sudden death
- Electrical Phase: symptomatic arrhythmias, ventricular enlargement/dysfunction

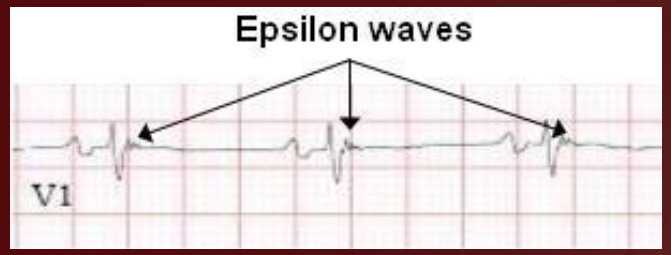
VT with LBBB morphology, triggered by exercise

Progressive heart failure, and may need heart transplant

Athletic activity precipitates the disease

ARVC: 2010 TASK FORCE CRITERIA MAJOR CRITERIA* **(2 CRITERIA FOR DIAGNOSIS)**

- Imaging (Echo, MRI, or angiography)
  - RV Akinesia, dyskinesia or aneurysm and meeting quantified thresholds
- ECG repolarization
  - T wave inversion V1, V2 and V3 without RBBB
- ECG depolarization
  - Epsilon wave

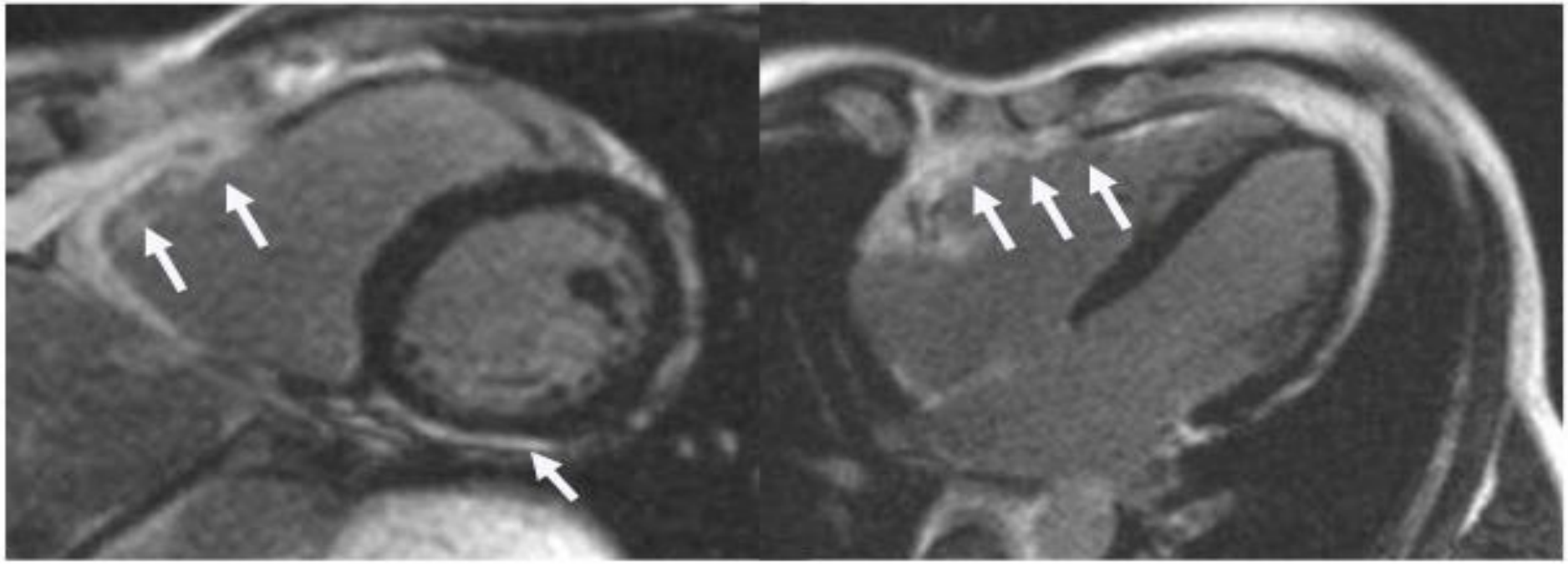


- Tissue
  - Quantified myocyte/fibrous percentages
- Ventricular arrhythmias
  - Left bundle superior axis (negative in V1, negative II, III, aVF). Meaning not RV outflow tract
- Family history
  - ARVC in 1st degree relative
  - Pathogenic mutation in the patient

IMAGING: MRI FOUR-CHAMBER VIEW

Cardiac MRI in ARVC

Late GAD enhancement indicating focal fibrosis



ARVC MANAGEMENT*

- Exercise restriction- no competitive or endurance sports, only low intensity, recreational, as may accelerate progression of disease
- Beta blockers should be considered in all patients regardless of arrhythmias
- Management of clinical heart failure (left or right): ACE-inhibitor, Angiotensin receptor blockers, beta blockers, diuretics
- Anticoagulation if prior thrombus

ARVC MANAGEMENT*

- ICD if:
  - Hemodynamically unstable VT or VF (Class I)
  - Severe RV or LV systolic dysfunction (Class I)
  - Hemodynamically stable sustained VT (Class IIa)
  - Unexplained syncope, moderate ventricular dysfunction or non-sustained VT (Class IIa)
- **DO NOT:** Implant ICD if asymptomatic patient without risk factors or healthy gene carrier (Class III)

Management of VT

- Antiarrhythmic drugs (Amiodarone + Beta blocker) are first line
- Ablation, second line, usually requires epicardial approach, but recurrence is probably about 50% at 5 years

MITRAL VALVE PROLAPSE

1. THE RELATIONSHIP WITH MVP AND TACHARRYTHMIAS

AND SCD IS CONTROVERSAL.

2. PT WITH MVP AND NO MR OR ADVANCED MITRAL APPARATUS PATH RISK OF SCD IS 2/10,000 PER YR.

3. MVP AND SIGNIFICANT MITRAL PATHOLOGY ARE AT A SIGNIFICANT INCREASE RISK OF SCD ANNUAL SCD MORTALITY RATE 0.9 TO 1.9 % ON SOME STUDIES.

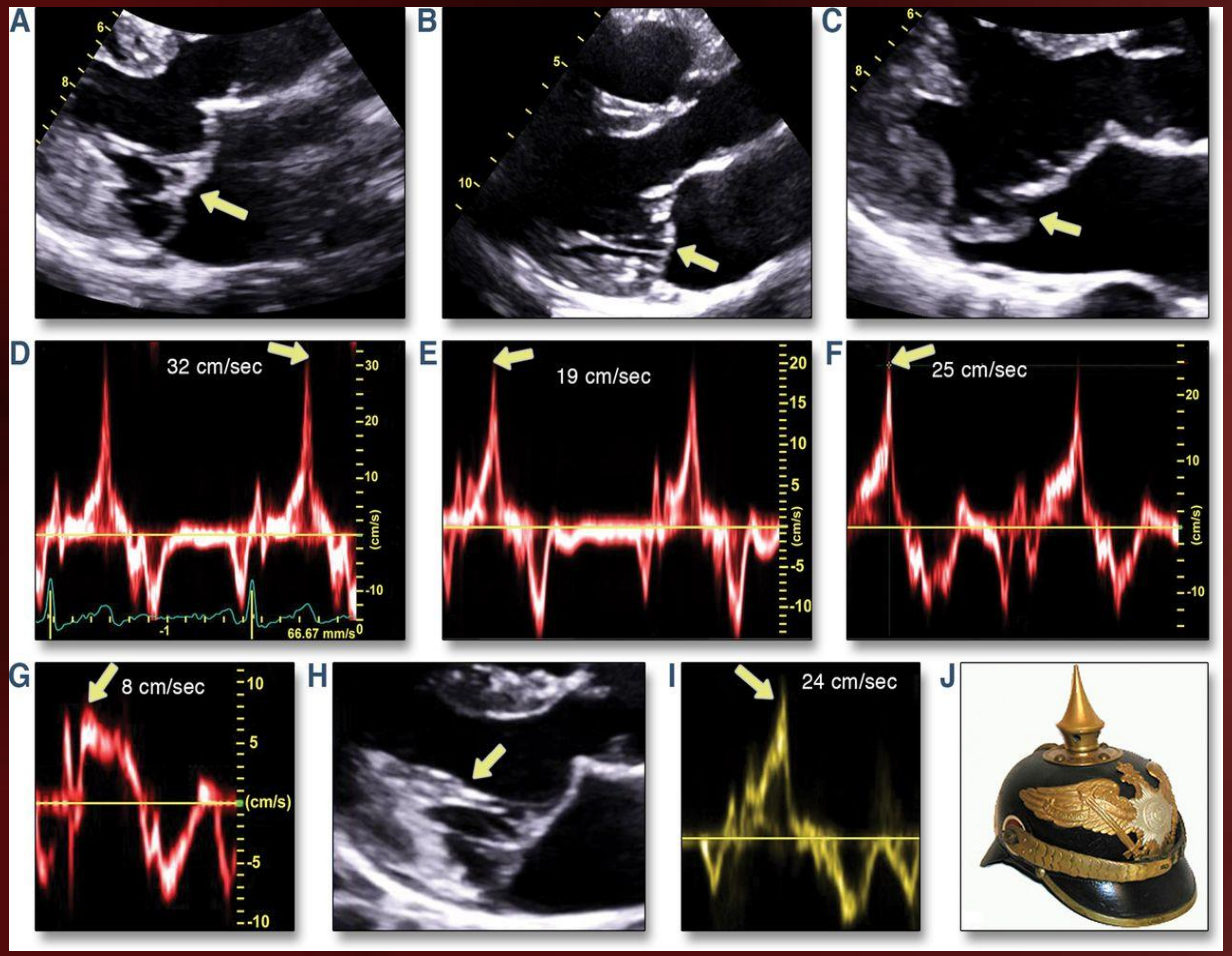
MITRAL VALVE PROLAPSE

- MVP is an under-estimated cause of arrhythmic SCD,
- mostly in young adult women.
- Fibrosis of papillary muscles and infero-basal LV wall, suggesting a myocardial stretch by the prolapsing leaflet, is the structural hallmark and correlates with ventricular arrhythmias origin.
- CE-CMR may help to identify this concealed substrate for risk stratification.

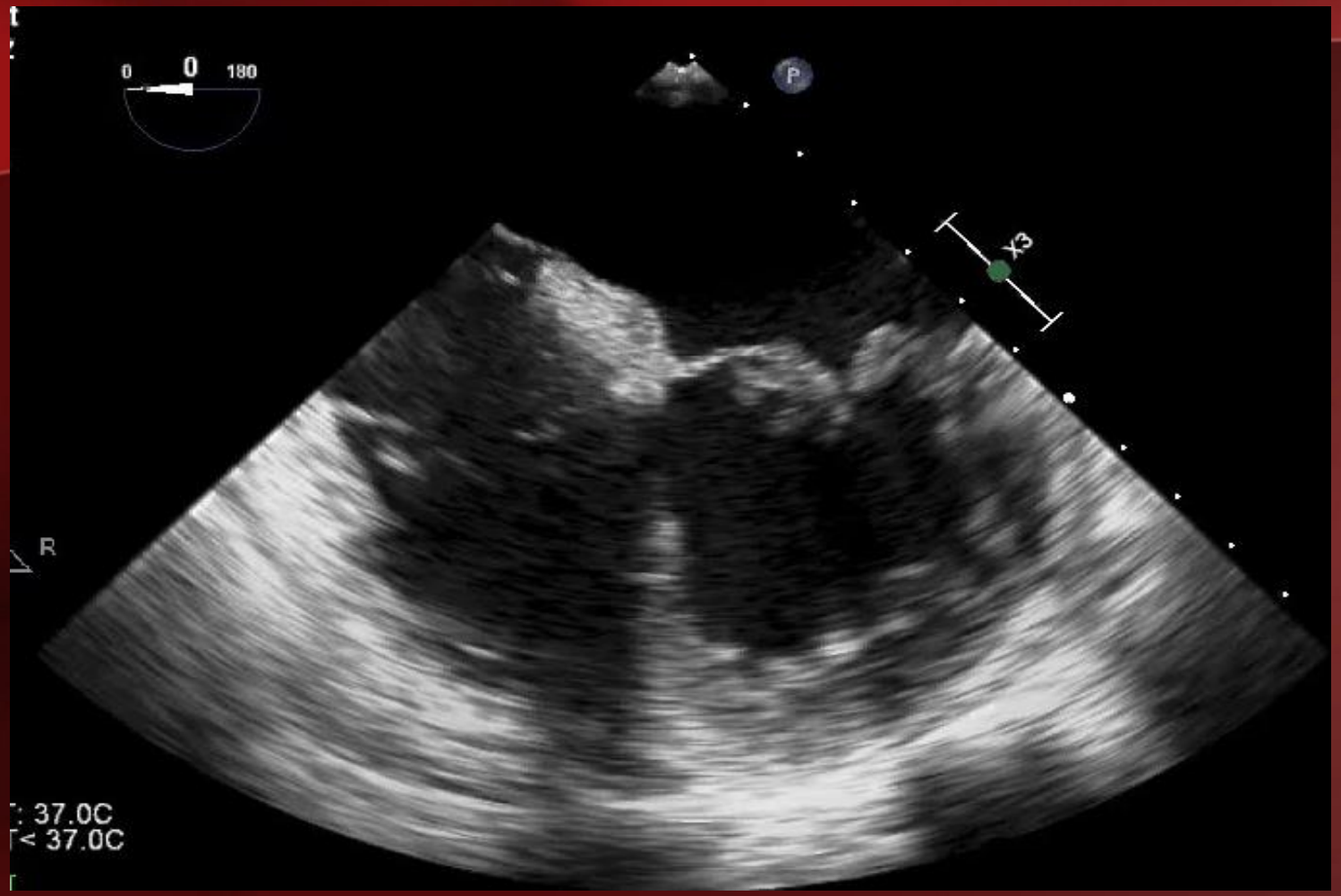
MITRAL VALVE PROLAPSE AND SCD

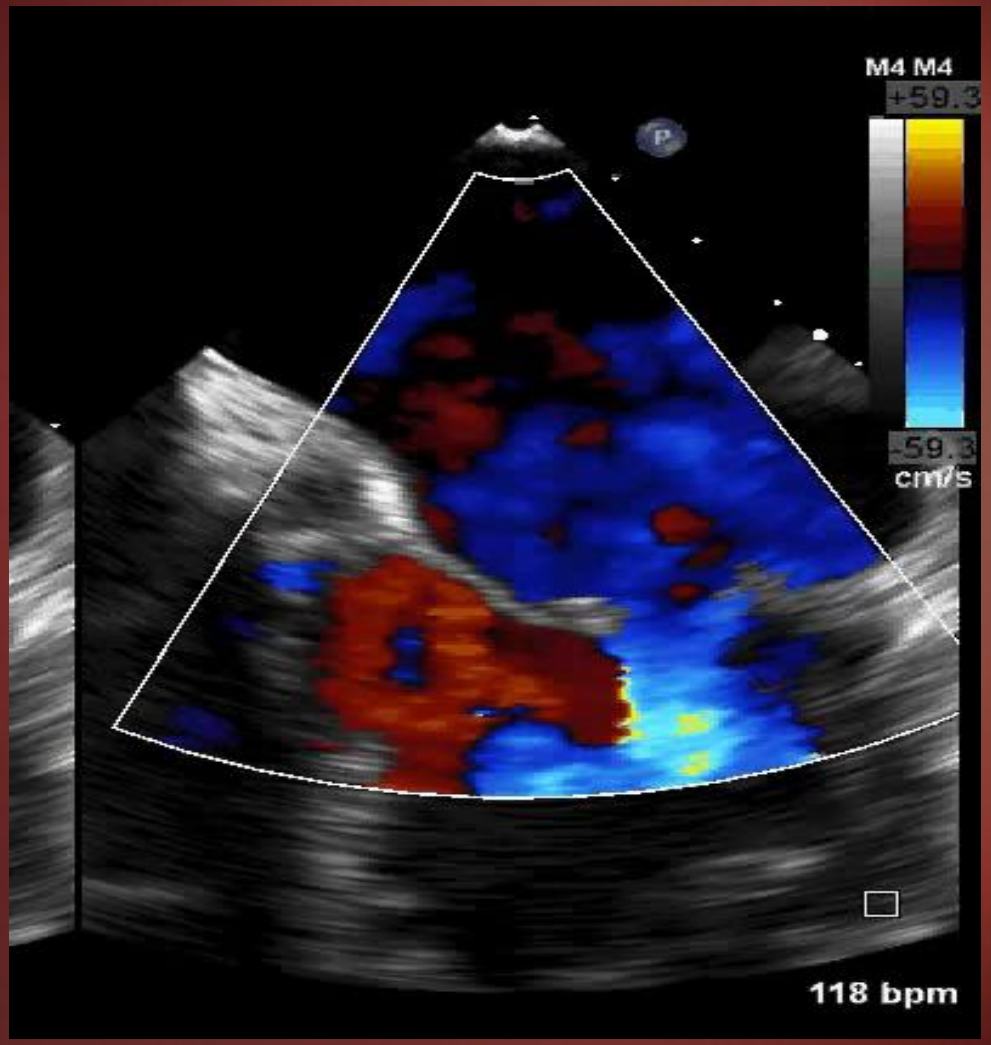
- A history of syncope associated with documented arrhythmia
- A family history of MVP and SCD
- Documented supraventricular tachycardias or complex ventricular ectopy, especially in association with exercise
- Moderate to severe associated mitral valve regurgitation
- A prior embolic event
- Associated electrical abnormalities (i.e. long QT interval)
  - If any of these is present, activity should be restricted to low intensity competitive sports
  - Given the potential for worsening of mitral regurgitation and rupture of myxomatous chordae due to the greatly increases systemic vascular resistance occurring with isometric exercise, sports with vigorous isometric components (i.e. weight lifting) should be avoided when MVP is associated with marked valvular abnormalities.

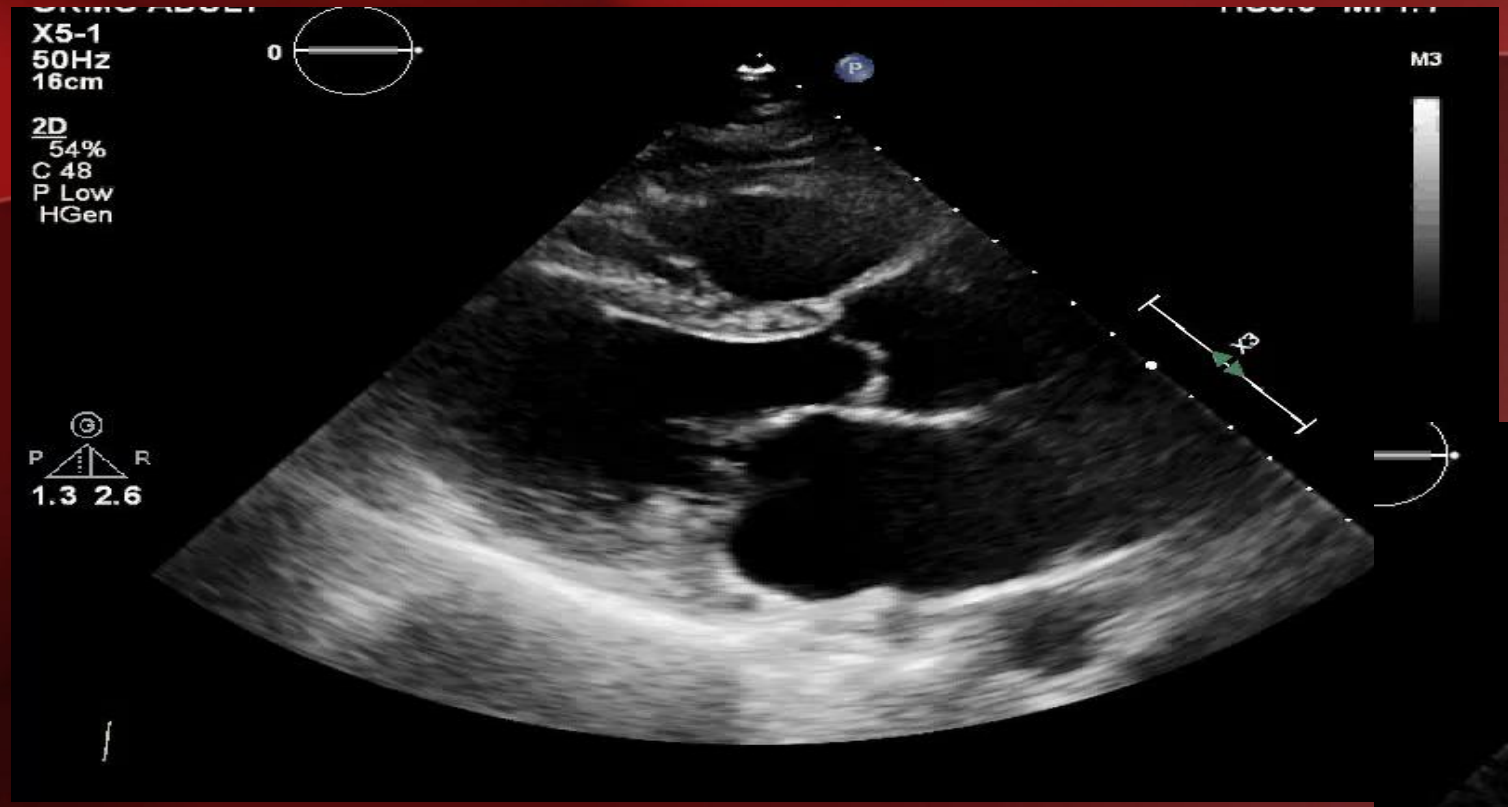
MITRAL VALVE PROLAPSE ECHOCARDIOGRAPHY

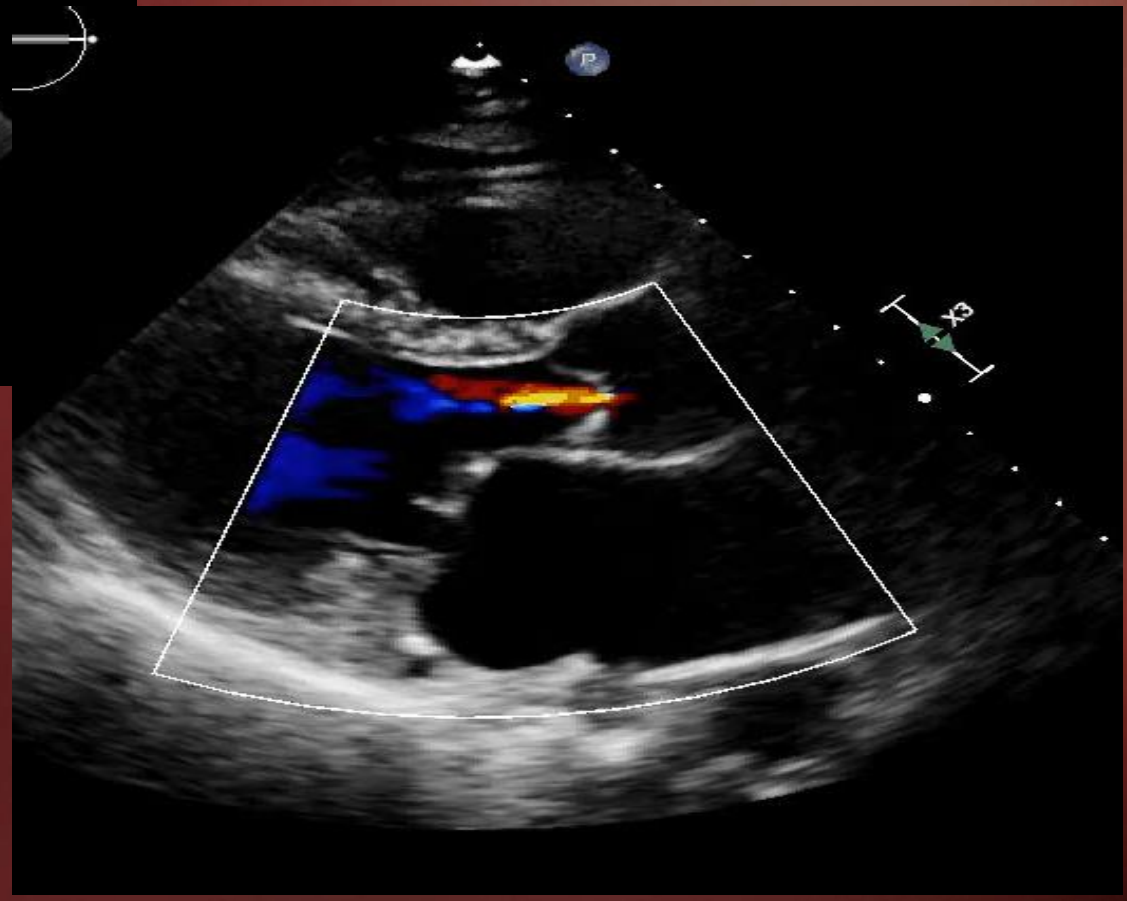


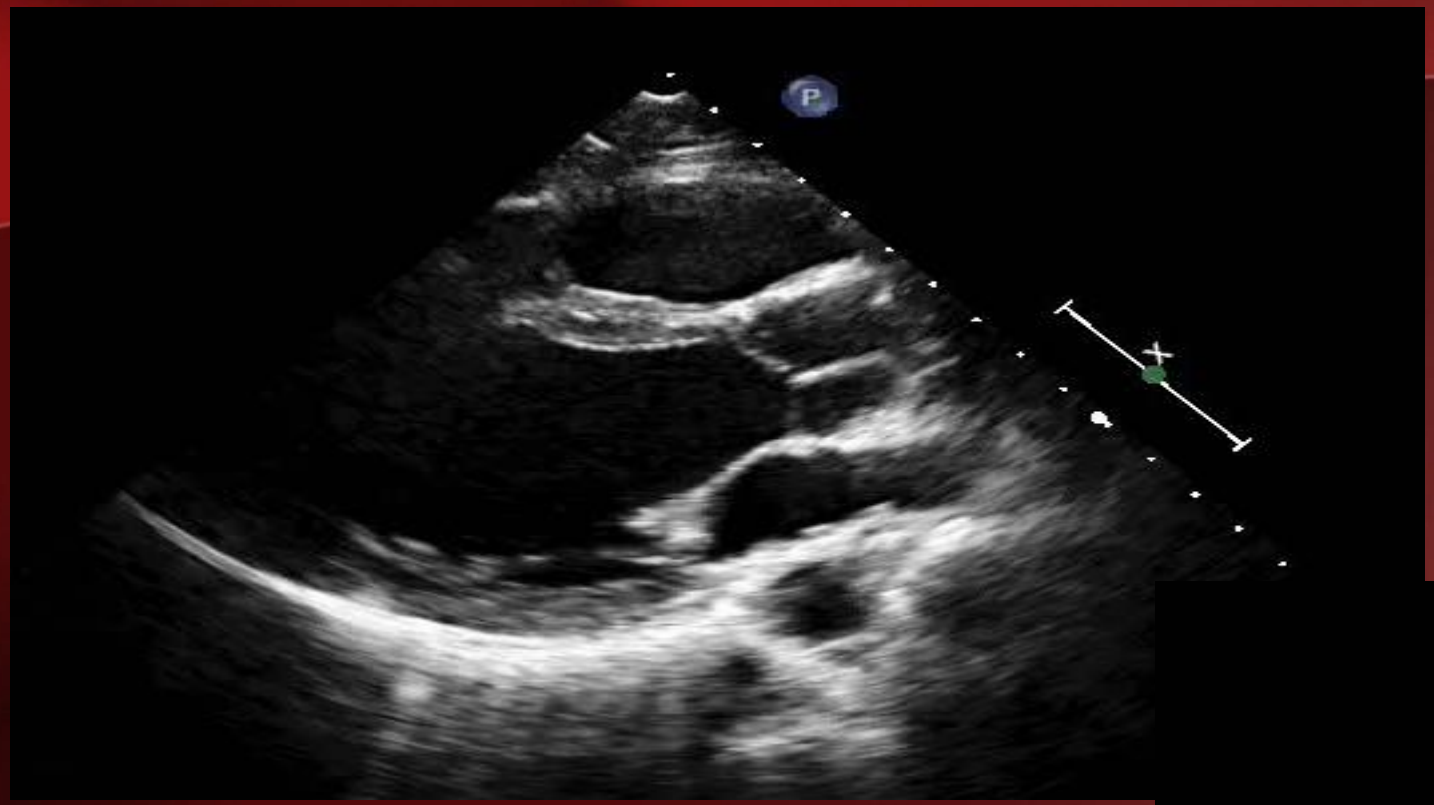
- Figure 1 Pickelhaube Sign
- Transthoracic echocardiography demonstrating myxomatous bileaflet mitral valve prolapse **(arrows)** in cases 1 **(A)**, 2 **(B)**, and 3 **(C)**. High-velocity mid-systolic spike (lateral annulus, 32 cm/s) in cases 1 **(D)**, 2 (mid-systole, 19 cm/s) **(E)**, and 3 (late systole, 25 cm/s) **(F)**. **(G)** Normal medial annulus systolic velocity, case 1. **(H)** Tugging of the posteromedial papillary muscle by prolapsing leaflets **(arrow)**, case 4. **(I)** Late-peaking systolic tissue velocity spike of 24 cm/s, case 4. **(J)** Pickelhaube, spiked German military helmet (reprinted with permission from the collection of Peter Suciu).

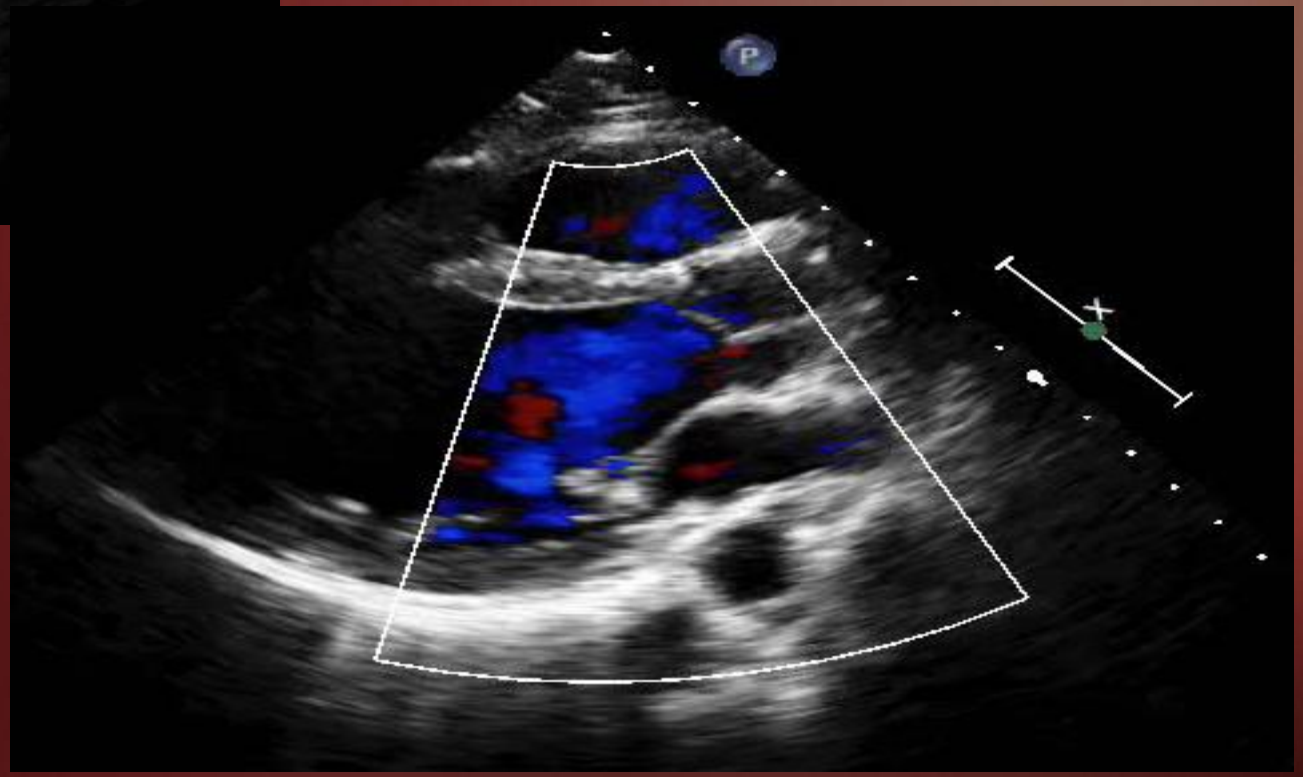

0
0
180
P
X3
R
: 37.0C
< 37.0C


M4 M4
+59.3
-59.3
cm/s
P
118 bpm


X5-1
50Hz
16cm
2D
54%
C 48
P Low
HGen
0
M3
X3
P
R
1.3 2.6


X3





ION CHANNELOPATHIES

INHERITED ELECTRICAL ARRHYTHMIAS 1 IN 1000 PEOPLE

- Long QT syndrome 1in 2000 people
- J wave syndromes
  - Brugada Syndrome
  - Early Repolarization
- Catecholaminergic polymorphic ventricular tachycardia
- Short QT syndrome

LONG QT SYNDROME- DIAGNOSIS

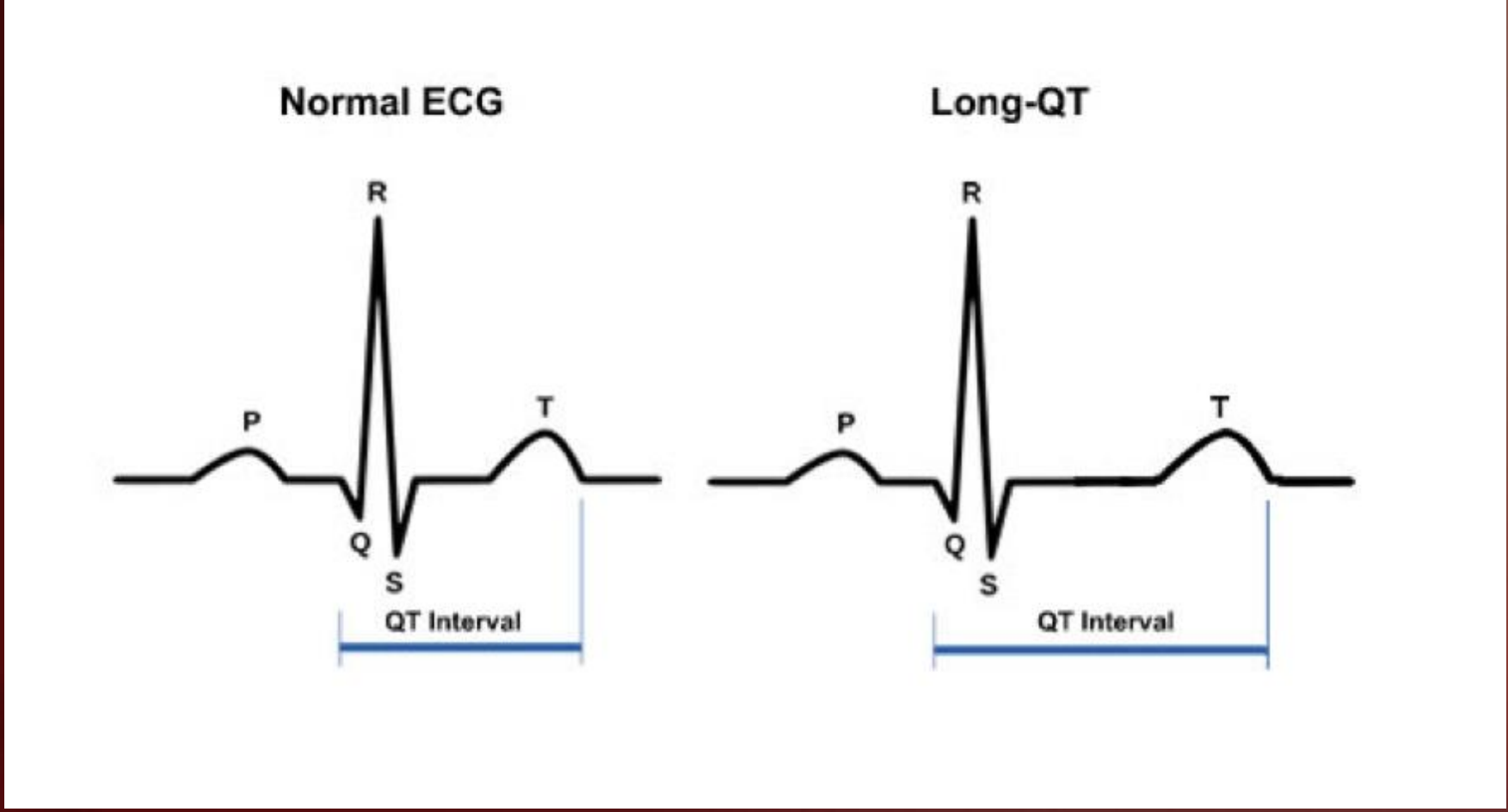
- Definite:
  - QTc ≥ 500ms (without secondary cause, recurrent)
  - LQTS risk score of at least 3.5
  - LQT mutation (unequivocal)
- Can be:
  - QTc 480-499ms, recurrent, no secondary cause AND unexplained syncope

TABLE 2. Schwartz Score for the Diagnosis of Long QT Syndrome (1993)

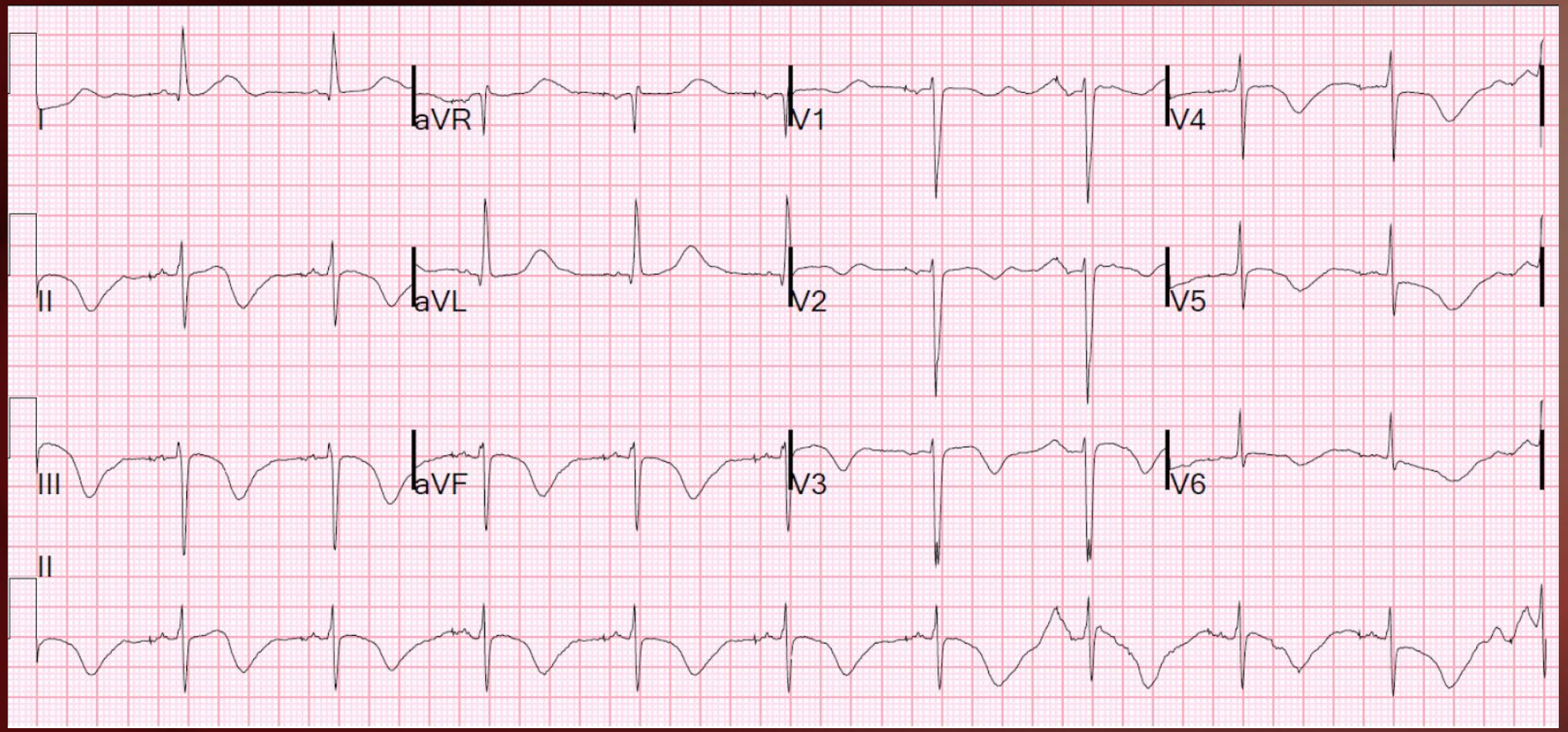
Variable		Points
Electrocardiogram		
QTc ms*	≥480	3
	460-470	2
	450 (males)	1
Torsade de pointes		2
T wave alternans		1
T wave notches in 3 leads		1
Bradycardia†		0.5
Clinical history		
Syncope		
With stress		2
Without stress		1
Congenital deafness		0.5
Family history‡		
Family members with confirmed LQTS§		1
Unexplained sudden death in first-order family members <30 years		0.5

*QTc calculated with the formula of Bazett (QTc=QT/ RR).
†Resting heart rate below the second percentile for age.
‡The same family member cannot be considered twice.
§Schwartz score ≥4: <1 point: low probability; 2-3 points: intermediate probability; ≥4 points: high probability.

ION CHANNELOPATHY



ION CHANNELOPATHY



LQT MUTATIONS

Variant	Gene	Protein	Effect of Mutation
LQT1	KCNQ1	KvLQT1 (potassium channel)	Reduced I_{Ks}
LQT2	KCNH2	HERG (potassium channel)	Reduced I_{Kr}
LQT3	SCN5A	Nav1.5 (sodium channel)	Increased I_{Na}

LQT 1, 2, 3 account for over 90%

Other LQT types:

- LQT5 – Reduced I_{Ks}
- LQT6- Reduced I_{Kr}
- LQT7 – Andersen syndrome, reduced outward I_{K1}
- LQT8: - Timothy syndrome, Increased I_{Ca}

ION CHANNELOPATHY

Table 1. Characteristics of LQTS Subtypes

Phenotype	Frequency	Trigger(s)	Cause	Mean QTc	Treatment
LQT1	60%	Exercise (e.g., swimming, running); emotion (startle, anger, fright)	Mutation in *KCNQ1*- or *KCNE1*-defective I_{Ks} channels	490 msec	Beta-blockers
LQT2	35%	Auditory stimulation causing sudden startle (e.g., alarm clock, telephone, siren)	Mutation in *hERG*- or *KCNE2*-defective I_{Kr} channels	480 msec	Beta-blockers
LQT3	4%-5%	Sleep	Mutation in *SCN5A*- I_{Na}	510-520 msec	Beta-blockers[a]; sodium channel blockers[b]; pacemaker with defibrillator

[a] *Questionable efficacy.*
[b] *A study evaluating ranolazine for LQT3 is scheduled to be completed in September 2014.*
LQTS: long QT syndrome.
Source: Reference 9.

GENETIC TESTING

Class I (is recommended)

- Strong clinical index of suspicion (history, family history, ECG, or provocative testing)
- Asymptomatic patient with QTc >500ms (adult) without predisposing factor
- Family members of a proband with defined mutation

Class IIb (may be considered)

- Asymptomatic, QTc>480ms (adult)

Highest risk for sudden death:

- QTc ≥500ms (except women with LQT3 are intermediate risk)

LONG QT SYNDROME: TREATMENT

Avoid QT prolonging drugs, avoid hypokalemia (Class I)

competitive sports - controversial

Left cardiac sympathetic denervation if high risk and cannot have ICD or beta blockade (Class I)

Potassium for LQT2 (Class IIb)

Mexiletine for LQT3 and QT> 500ms (Class IIa);

ICD if:

- Cardiac Arrest (Class I)
- Syncope despite beta blocker (Class IIa)

Do NOT implant ICD if asymptomatic and not on beta blockers

Beta blockers (for all but most effective for LQT1):

- Class I: QTc>470ms or syncope/VT.
- Class IIa: QTc<470ms
- **Nadolol** 1-2.5mg/kg or **Metoprolol** 2-4mg/kg if asthma

MEDICATIONS THAT PROLONG THE QT INTERVAL: KNOW THE KEY OFFENDERS*

Antibiotics

- Fluoroquinolones: ciprofloxacin, levofloxacin, moxifloxacin
- Macrolides: erythromycin, azithromycin, clarithromycin

Methadone

Anti-depressant and Anti-psychotic:

- SSRI's (citalopram, escitalopram)
- Anti-psychotic (haloperidol)

Note that many other drugs are suspected of prolonging the QT interval, and should not be used in patients with LQTS*

J WAVE SYNDROMES: BRUGADA SYNDROME

≥2mm ST elevation in right precordial leads

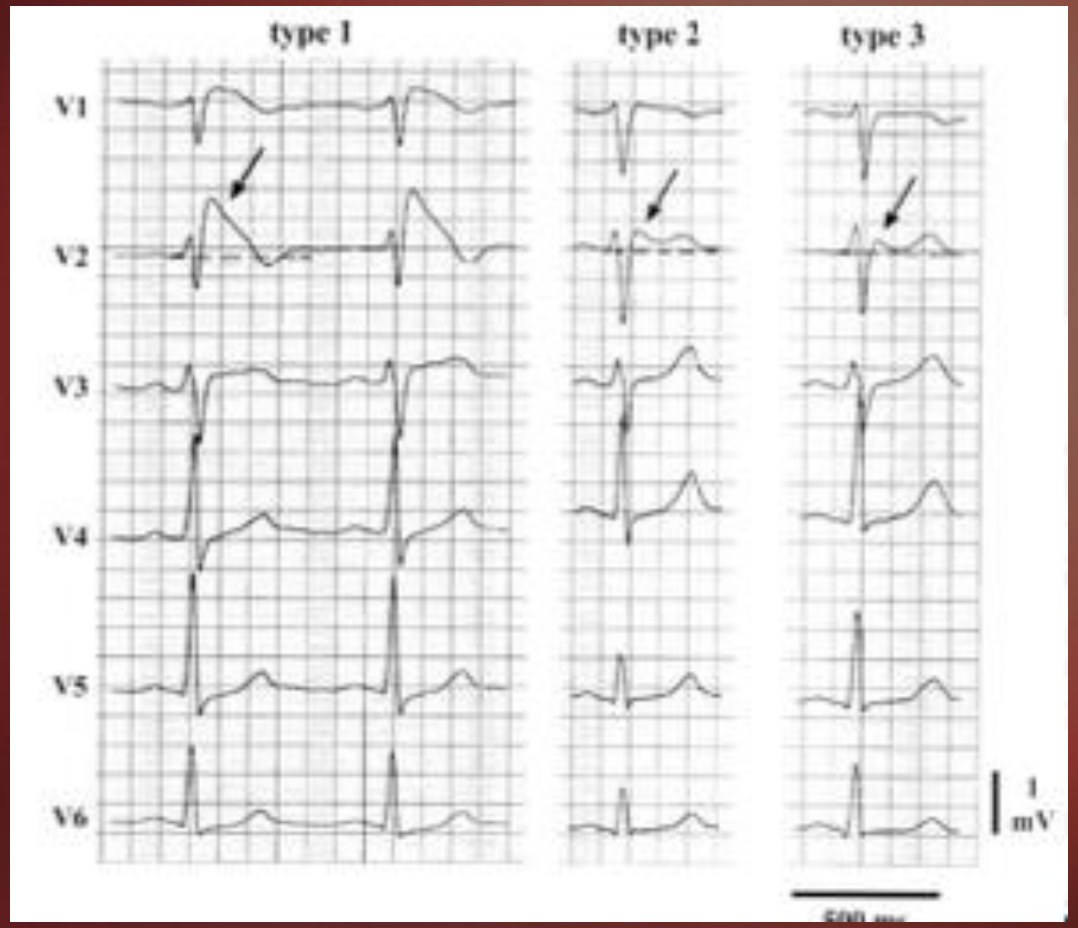
- Spontaneous or provoked with Class I antiarrhythmic drug

Only Type 1 diagnostic

2nd, 3rd or 4th intercostal space for V1, V2

What makes ST elevation worse:

- Any Na channel blockade (brugadadrugs.org)
  - Tri-cyclic antidepressants (amitriptyline)
  - Lithium
- Fever
- Large meals (vagal/sympathetic relative tone)
- In sleep
- Cocaine, alcohol



BRUGADA SYNDROME: RISK STRATIFICATION AND TREATMENT

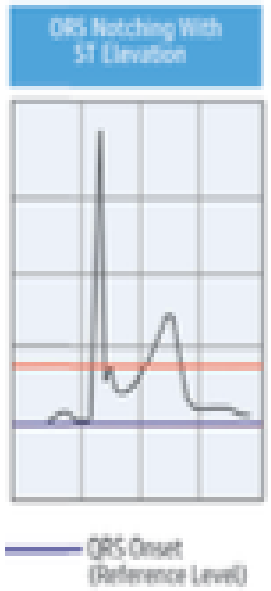
- Definite risk:
  - Spontaneous type 1 ECG
  - Prior syncope
- Not associated with risk:
  - Family history
- Avoid Na channel blocking drugs* (Class I)
- Avoid alcohol (Class I)
- Treat fevers (Class I)
- Quinidine for >2 episodes of VT/VF in 24 hrs
- Quinidine if refuse an ICD but qualify for one (Class IIa)
- Isoproterenol for VT/VF storm (Class IIa)

ICD if:

- Cardiac arrest (Class I)
- Spontaneous, sustained VT (Class I)
- Spontaneous type 1 ECG and syncope likely due to ventricular arrhythmia (Class IIa)
- Inducible VF at EP study (Class IIb)

Do NOT implant an ICD if asymptomatic with drug induced type 1 ECG on the basis of family history of sudden cardiac death alone (Class III)

OTHER INHERITED ARRHYTHMIA SYNDROMES



Early Repolarization (J wave syndrome)

- Abnormal notching, slurring at the end of the QRS in inferior/lateral leads
- ICD if cardiac arrest survivor (Class I)

Short QT syndrome (very rare, very deadly)

- QTc < 360ms

Catecholaminergic Polymorphic Ventricular Tachycardia (CPVT)

- Ventricular arrhythmias WITH EXERCISE, bidirectional
- Class I:
  - Limit exercise
  - Beta blockers
  - ICD if cardiac arrest survivor or recurrent syncope despite therapy

Bidirectional VT

ION CHANNELOPATHY

- It remains prudent for an athlete with a channelopathy, whether concealed or manifest, to exercise simple precautionary measures, including
  - (1) avoidance of QT-prolonging drugs for athletes with LQTS.
  - (2) avoidance of drugs that exacerbate the BrS in affected athletes.
  - (3) electrolyte/hydration replenishment and avoidance of dehydration for all.
  - (4) avoiding/treating hyperthermia from febrile illnesses or training-related heat exhaustion/heat stroke for athletes with either LQTS or BrS
  - (5) acquisition of a personal automatic external defibrillator as part of the athlete's personal sports safety gear.
  - (6) establishing an emergency action plan with the appropriate school/team officials.

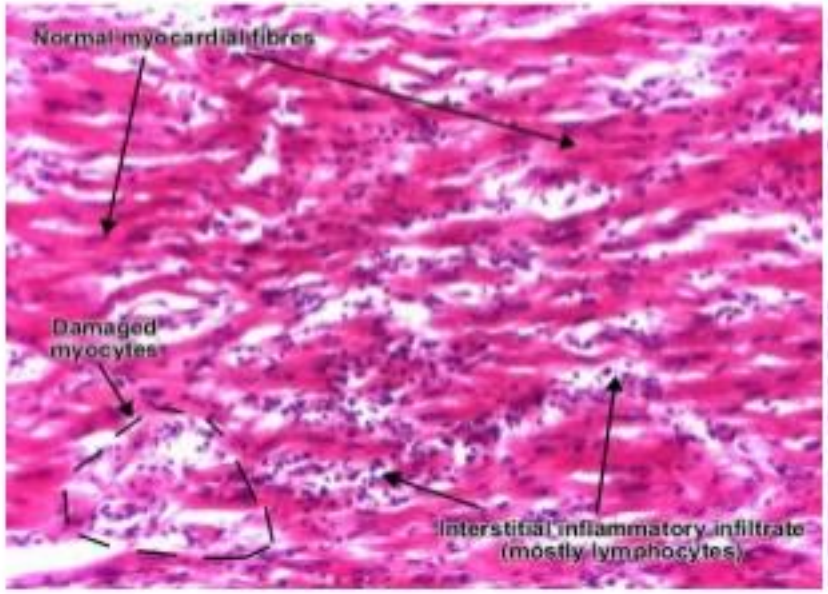
ION CHANNELOPATHY RECOMMENDATIONS

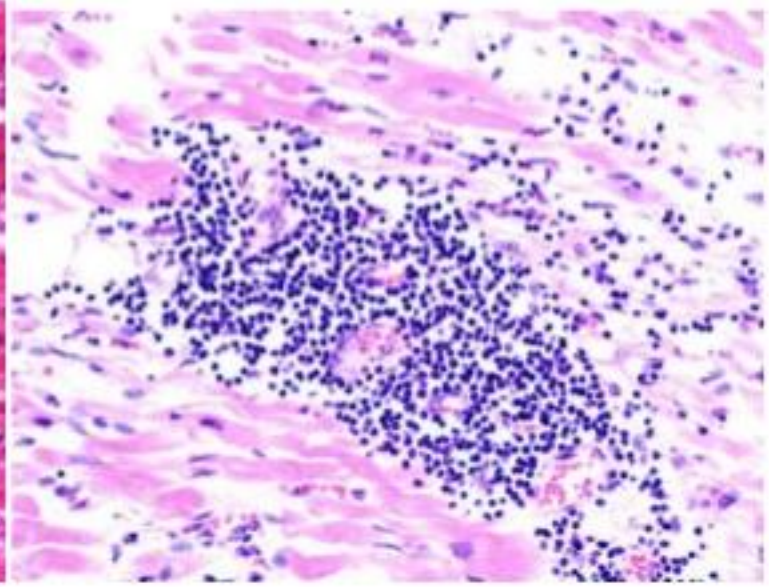
- For athletes with a suspected/diagnosed cardiac channelopathy, a comprehensive evaluation by a heart rhythm specialist or genetic cardiologist with sufficient experience and expertise with these disorders is recommended *(Class I; Level of Evidence C)*.
- It is recommended that symptomatic athletes with any suspected or diagnosed cardiac channelopathy be restricted from all competitive sports until a comprehensive evaluation has been completed, the athlete and his or her family are well informed, a treatment program has been implemented, and the athlete has been asymptomatic on therapy for 3 months *(Class I; Level of Evidence C)*.
- It is reasonable for an asymptomatic athlete with genotype-positive/phenotype-negative (ie, concealed channelopathy) LQTS, CPVT, BrS, early repolarization syndrome, idiopathic ventricular fibrillation, or short-QT syndrome to participate in all competitive sports with appropriate precautionary measures, including (1) avoidance of QT-prolonging drugs for athletes with LQTS. (2) avoidance of drugs that exacerbate the BrS in affected athletes, (3) electrolyte/hydration replenishment and avoidance of dehydration for all, (4) avoidance or treatment of hyperthermia from febrile illnesses or training-related heat exhaustion or heat stroke for athletes with either LQTS or BrS, (5) acquisition of a personal automatic external defibrillator as part of the athlete's personal sports safety gear, and (6) establishment of an emergency action plan with the appropriate school or team officials *(Class IIa; Level of Evidence C)*.
- Competitive sports participation may be considered for an athlete with either previously symptomatic or electrocardiographically evident BrS, early repolarization syndrome, or short-QT syndrome assuming appropriate precautionary measures and disease-specific treatments are in place and that the athlete has been asymptomatic on treatment for at least 3 months *(Class IIb; Level of Evidence C)*. If therapy includes an ICD, refer to the Task Force 9 report.[2]
- For an athlete with either symptomatic LQTS or electrocardiographically manifest LQTS (ie, corrected QT interval >470 ms in males or >480 ms in females), competitive sports participation (except competitive swimming in a previously symptomatic LQT1 host) may be considered after institution of treatment and appropriate precautionary measures assuming the athlete has been asymptomatic on treatment for at least 3 months *(Class IIb; Level of Evidence C)*. If treatment includes an ICD, refer to the Task Force 9 report[2] for recommendations regarding restrictions after the procedure, lead replacements, and so forth.
- For an athlete with previously symptomatic CPVT or an asymptomatic CPVT athlete with exercise-induced premature ventricular contractions in bigeminy, couplets, or nonsustained ventricular tachycardia, participation in competitive sports is not recommended except for class IA sports *(Class III; Level of Evidence C)*. Exceptions to this limitation should be made only after consultation with a CPVT specialist.

MYOCARDITIS

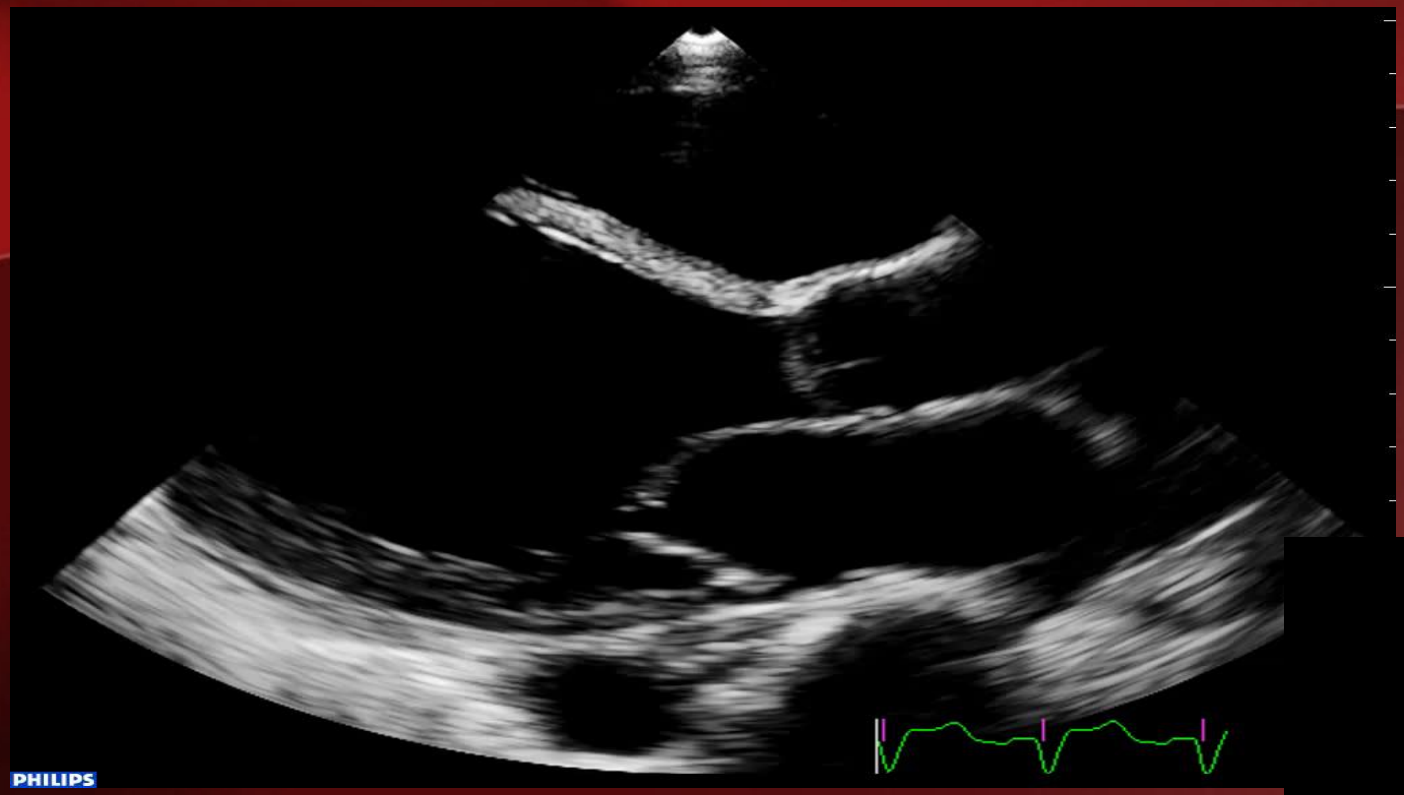
- Estimates of myocarditis related SCD varies with age
- ~2% infants
  - 5% children
  - 4 - 7.5% athletes
- Most deaths occur in males
- Most frequent cause of SCD associated with strenuous physical exertion in a cohort of US military recruits
- Risk of death does not appear to correlate with severity of myocardial inflammation in one autopsy series

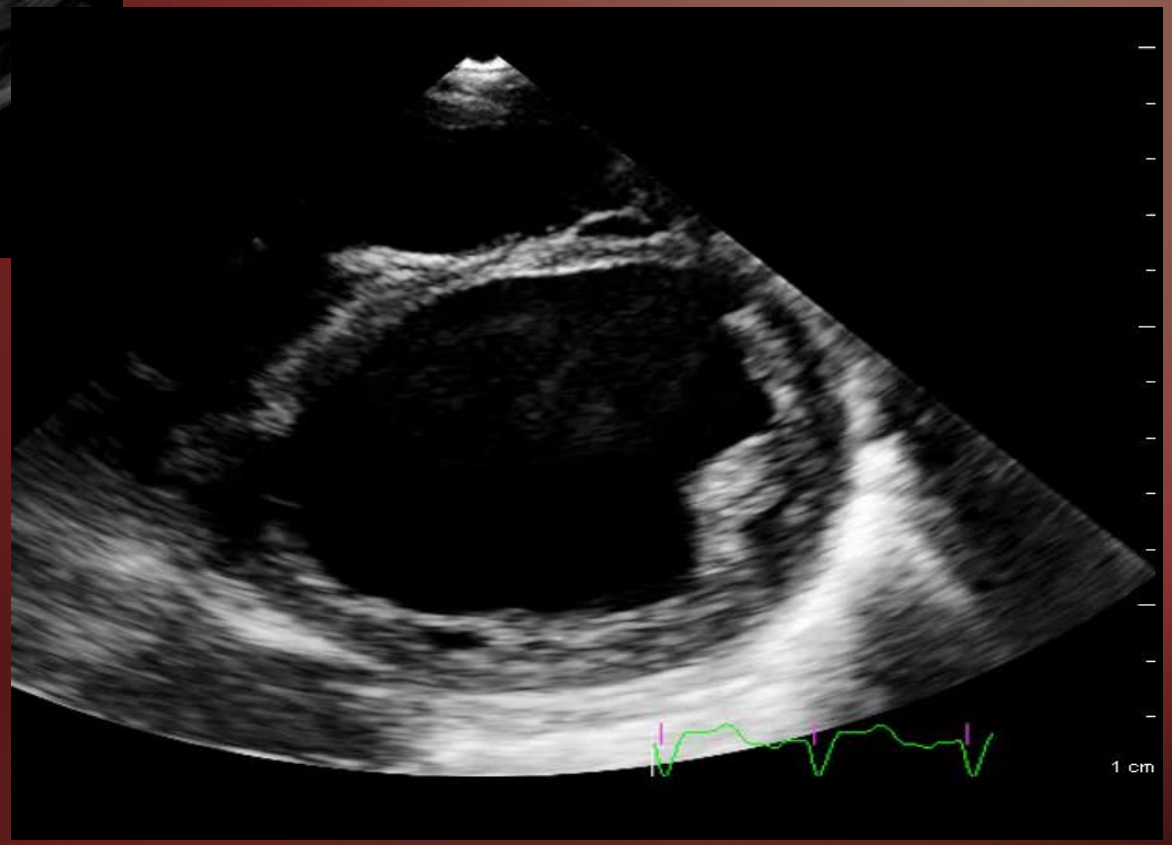
Viral myocarditis:





*N.B. established histological **Dallas criteria** defined as follows:histological evidence of inflammatory infiltrates within the myocardium associated with myocyte degeneration and necrosis of nonischaemic origin


PHILIPS


1 cm

MYOCARDITIS

- Define acute myocarditis
- Clinical syndrome that includes acute heart failure, angina-type chest pain, or myopericarditis of <3 months' duration.
  - Otherwise unexplained elevation in serum troponin; ECG features of cardiac ischemia; otherwise unexplained high-degree AV block or arrhythmias; wall motion abnormalities; pericardial effusion on echo or CMR. Additional CMR findings that suggest myocarditis in the acute clinical setting include characteristic alterations in tissue signal on T2 or T1-weighted images and the presence of late gadolinium enhancement.
- Points out that it may be impossible to distinguish active inflammation from fibrosis on LGE sequences and that there is no sensitive or specific test to determine with acute inflammation ends.
- Presence of LGE may convey increased risk for arrhythmias
- Reasonable interval for retesting based on experimental models is 3-6 months

MYOCARDITIS RECOMMENDATIONS

1. Before returning to competitive sports, athletes who initially present with an acute clinical syndrome consistent with myocarditis should undergo a resting echo, 24-hour Holter, and an exercise ECG no less than 3-6 months after the initial illness (I, Level C)
2. It is reasonable that athletes resume training and competition if all the following criteria are met (IIa, Level C)
   a. Ventricular systolic function has returned to normal
   b. Serum markers of myocardial injury, inflammation, and heart failure have normalized
   c. Clinically relevant arrhythmias such as frequent or complex repetitive forms of ventricular or supraventricular ectopy are absent on Holter monitor and graded exercise ECGs.

   At present, it is unresolved whether resolution of myocarditis-related LGE should be required to permit return to competitive sports.
3. Athletes with probable or definite myocarditis should not participate in competitive sports while active inflammation is present. This recommendation is independent of age, gender, and LV function (III, Level C)

THANK YOU

Dr. Arnold M. Einhorn, MD FACC, FACP