



State-of-the-Art
ECHOCARDIOGRAPHY

31st
ANNUAL

Case Studies
Athlete's Heart



THE UNIVERSITY OF
CHICAGO
CARDIAC IMAGING CENTER

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HOCM




Peripartum CMP



Apical CMP



Takasubo CMP



Athlete's Heart

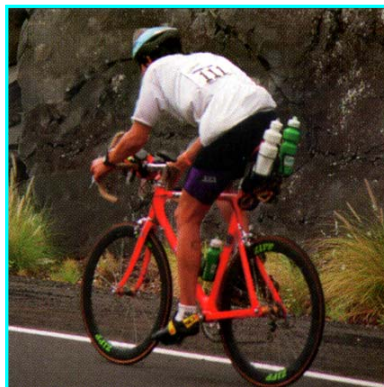
Subjects diagnosed with athlete's heart commonly display signs that would usually indicate a heart condition if they were seen in a non-athlete:

- **Bradycardia**
- **Cardiomegaly**
- **Cardiac Hypertrophy**

Athlete's Heart

Long term athletic training results in

- **↑ LV EDD**
- **↑ LV WT**
- **↑ LV Mass**
- **↑ maximal O₂ consumption**

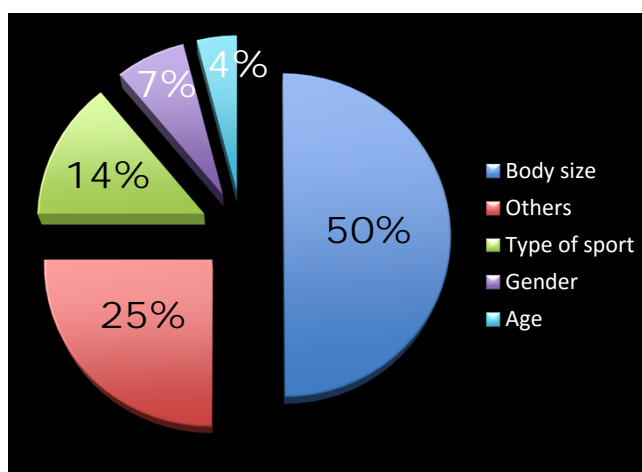


Increase extraction of oxygen, reduced blood lactate levels, maximal AV oxygen difference

LVEDD

- Large group (1309) elite athletes from 38 disciplines
- Women: (**mean 48 mm**)
- Men: (**mean 55mm**)
- **LVEDD was between 60 and 70 mm in 14%**
- Larger LVEDD seen in athletes with higher body mass and in those participating in endurance sports (cycling, canoeing)

Impact of different clinical variables on LV EDD cavity dimensions in a large population of male and female elite athletes.



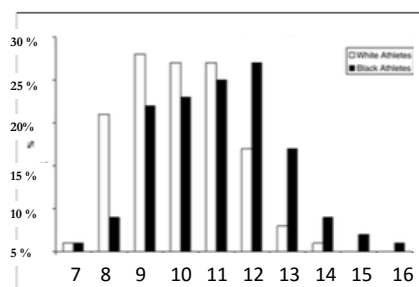
Maron B J , Pelliccia A Circulation 2006;114:1633-1644

Upper limits of Physiologic Cardiac Hypertrophy in Highly Trained Elite Athletes

- Athletes with a WT of 13-16 mm and a non-dilated LV cavity = Suspect primary pathologic hypertrophy
- All athletes with walls ≥ 13 mm also have enlarged LVED (55 to 63 mm)

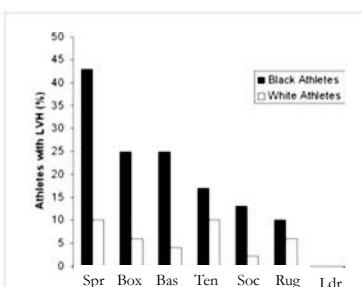
Differences in LVH between black and white athletes

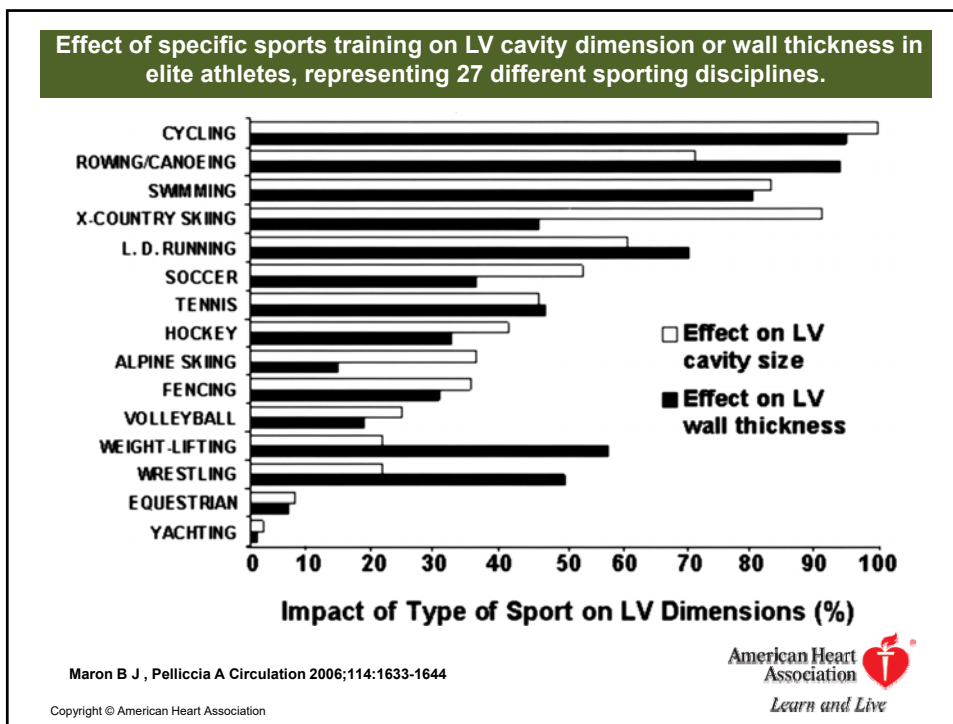
Distribution of LV WT



A minority of black athletes exhibit a LV WT ≥ 15 mm compared with none of the white athletes

LVH in relation to Sporting disciplines





RV Modeling in Olympic Athletes

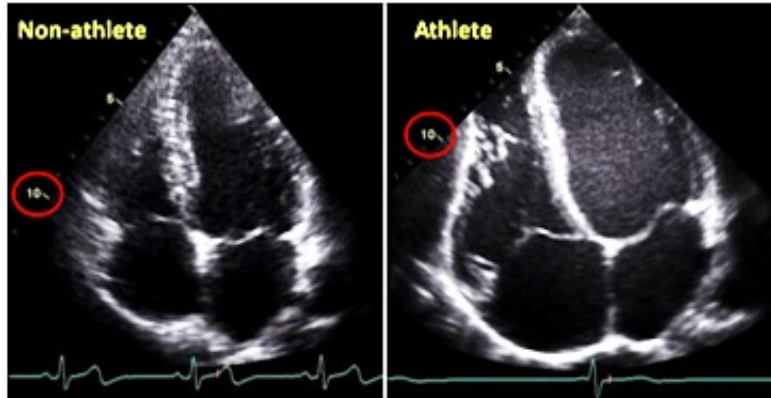
- RV modeling occurs in Olympic athletes, with male sex endurance practice playing the major impact. A significant subset (up to 32%) of athletes exceeds the normal guideline limits



LV Systolic Function in the Athlete

23 y/o non athlete, LVEF 66%

23 y/o athlete, LVEF 51%



Most athletes have normal resting LVEF
Tour de France Athletes: 11% had a calculated LVEF of \leq 52%

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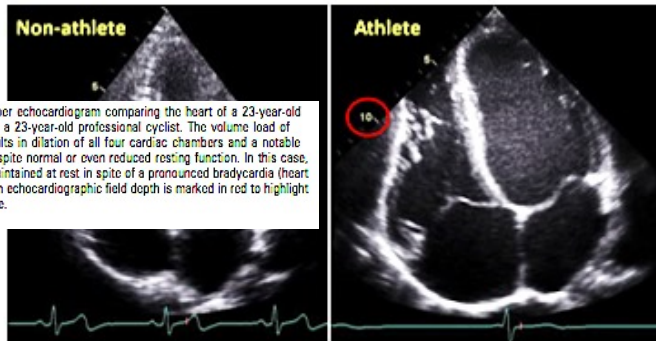
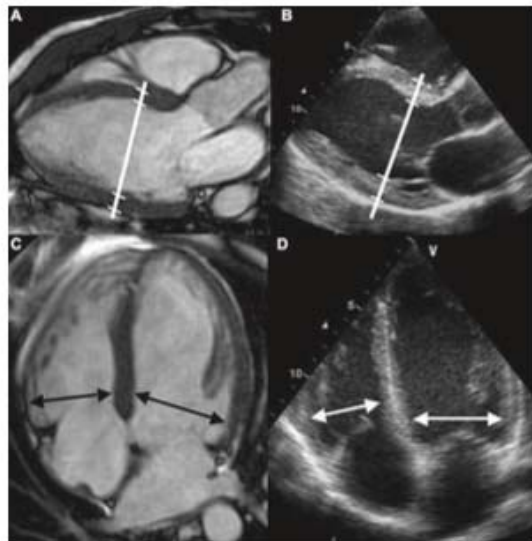


Figure 1 Apical four chamber echocardiogram comparing the heart of a 23-year-old non-athlete (left) with that of a 23-year-old professional cyclist. The volume load of endurance sport training results in dilation of all four cardiac chambers and a notable increase in stroke volume despite normal or even reduced resting function. In this case, normal cardiac output is maintained at rest in spite of a pronounced bradycardia (heart rate 28 beats/min). The 10 cm echocardiographic field depth is marked in red to highlight differences in cardiac size.

Most athletes have normal resting LVEF
Tour de France Athletes: 11% had a calculated LVEF of \leq 52%

Understand the inter-technique differences



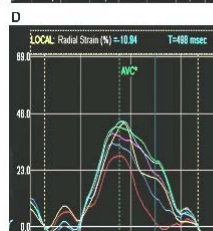
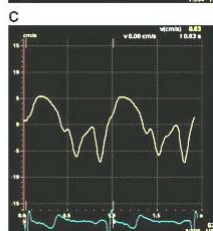
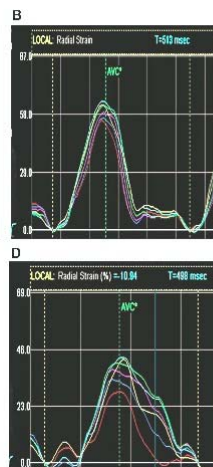
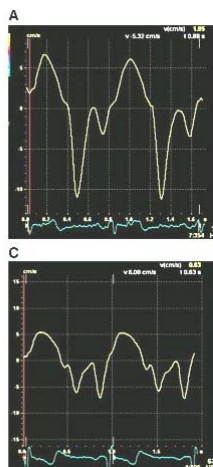
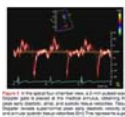
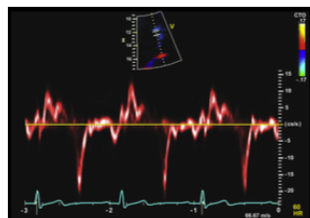
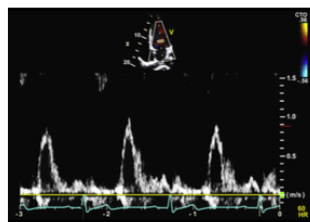
Septal WT = 11 mm
 LV Post WT = 12 mm
 LV ED = 66 mm

MRI: > Dimensions

Echo > Thickness

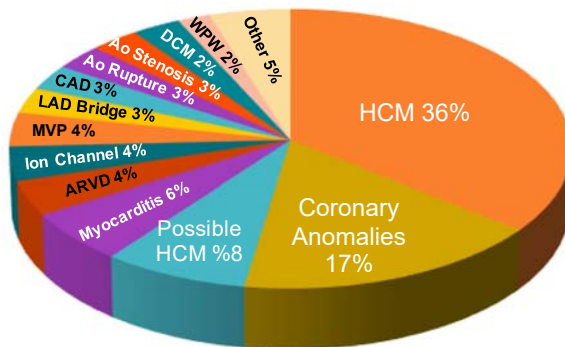
Septal WT = 14 mm
 LV Post WT = 13 mm
 LV ED = 54 mm

LV Diastolic Function in the Athlete



Endurance exercise training leads to enhanced early diastolic filling, as assessed by the E wave and mitral annular velocities

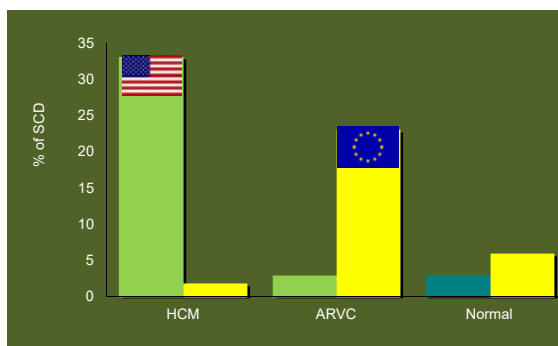
Cardiovascular causes of Sudden Death in Young Competitive Athletes



US National registry of 1866 Athlete Deaths

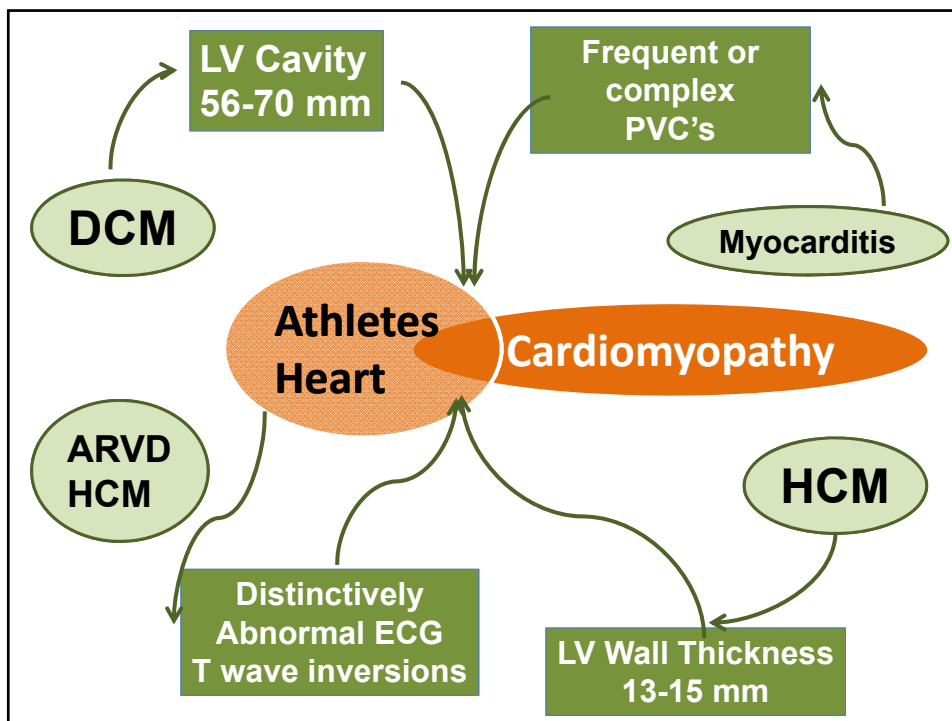
SCD (<40 y): Underlying Pathology

Athletes



	Country	Cases of SCD	n/100000/y
* Maron, 2003	US	387	0,5
Corrado, 2003	Italia	55	2,3

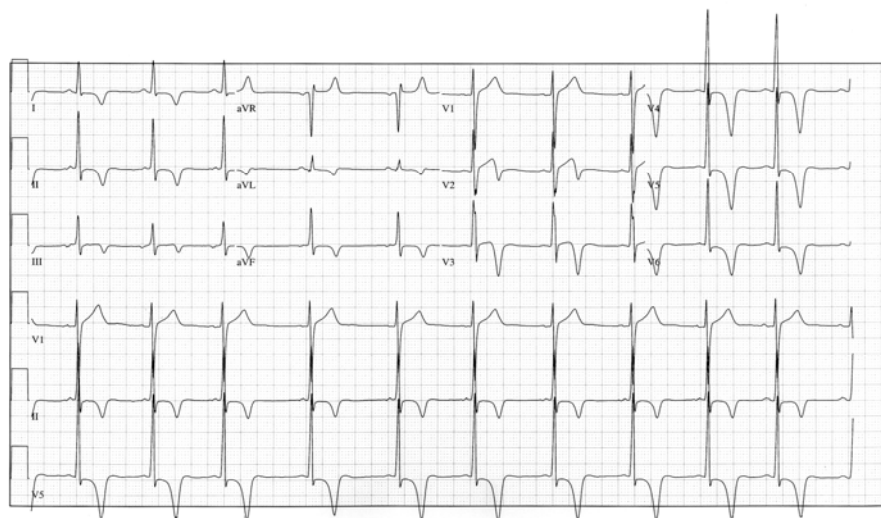
* passive surveillance, ascertainment and referral bias.



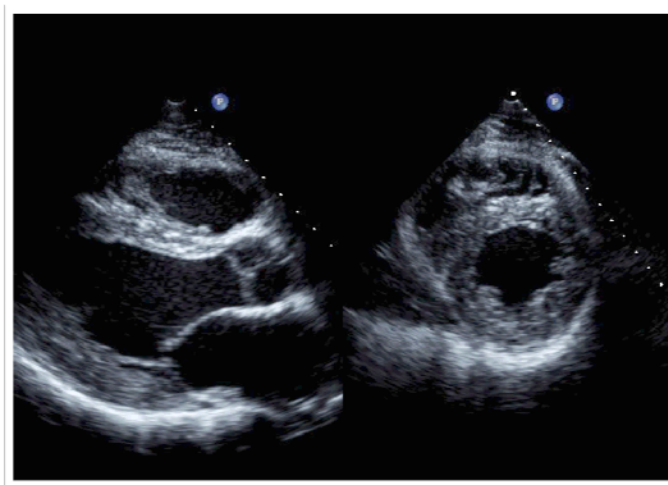
A 22 year old athlete who plays professional basketball (NBA) was evaluated for a second opinion regarding his abnormal ECG.

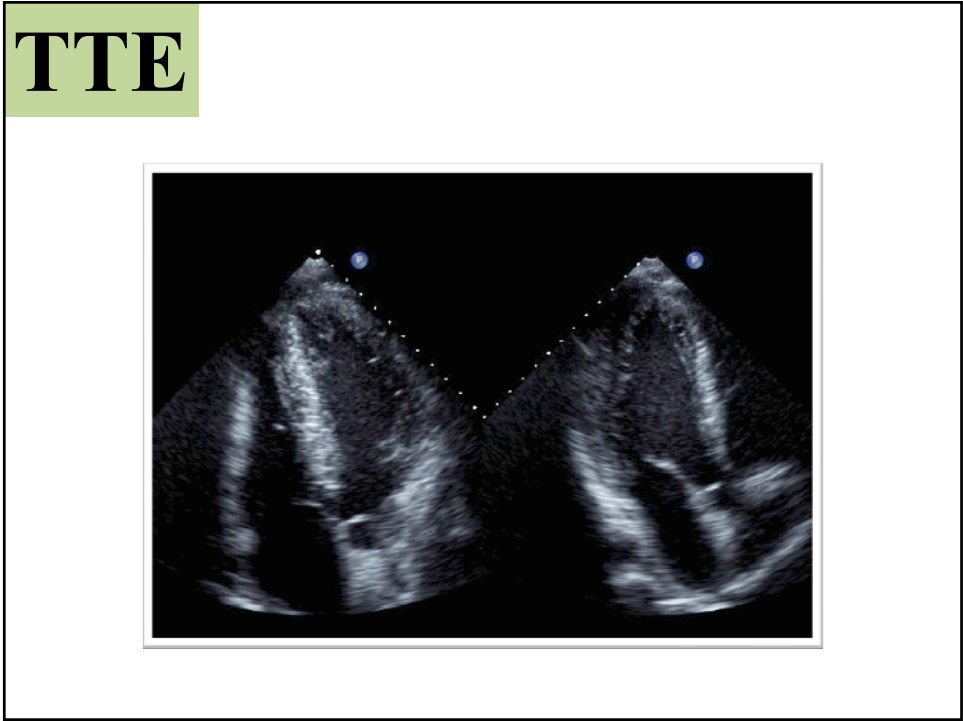
- **In the pre-season had an abnormal EKG**
- **The team physician does not allow him to continue training**
- **The player's manager sends him for a second opinion**

ECG

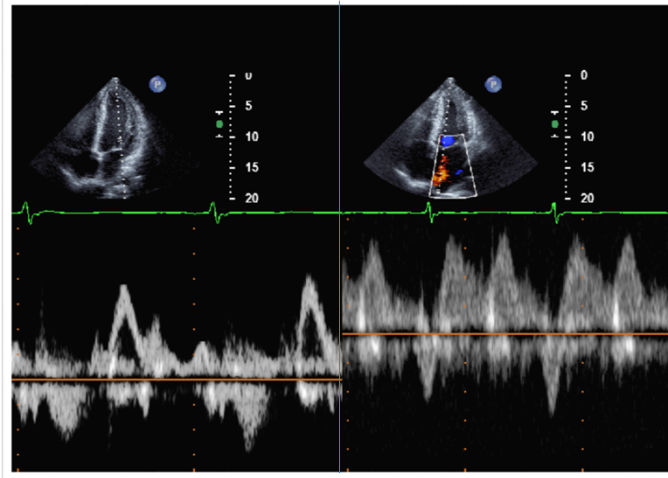


TTE: Basketball Player





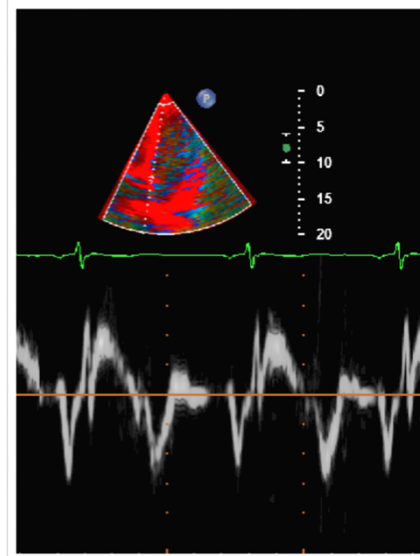
TTE



Mitral Inflow

PV Flow

TTE: Tissue Doppler

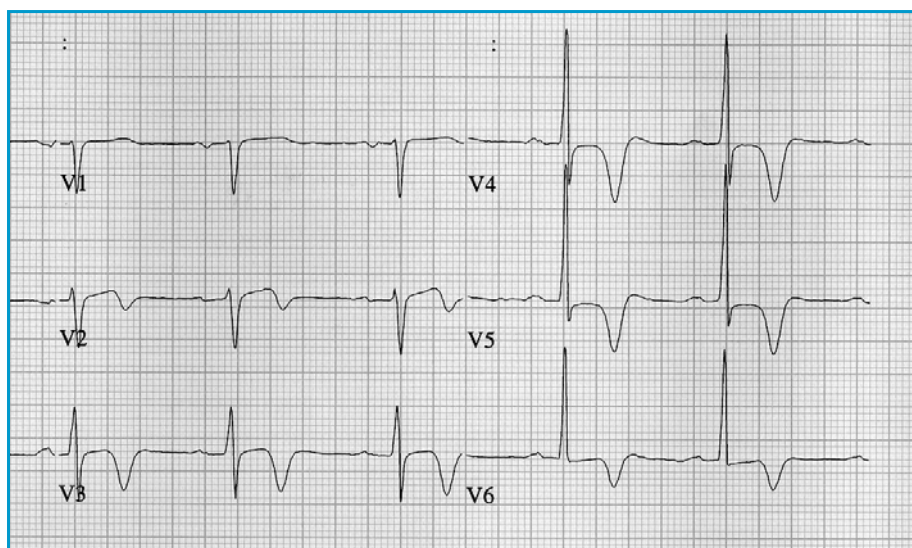


Findings: ECHO

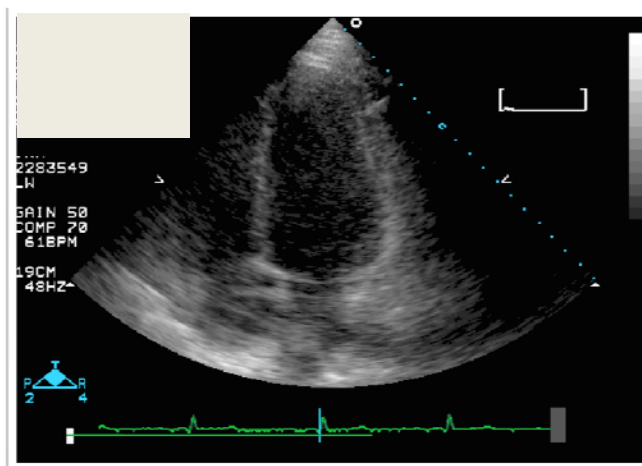
- **LV EDD = 5.2 cm**
- **LV ESD = 3.3**
- **WT = 1.33 cm**
- **LVEF = 68%**
- **Diastolic Function = normal**
- **Trivial MR**



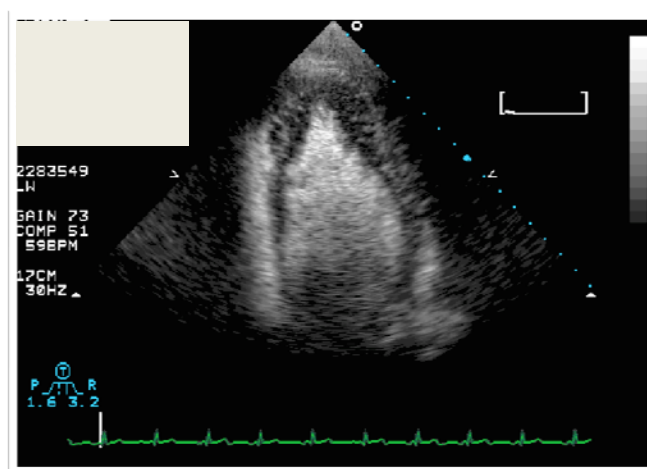
19 year old male, evaluated for an abnormal ECG

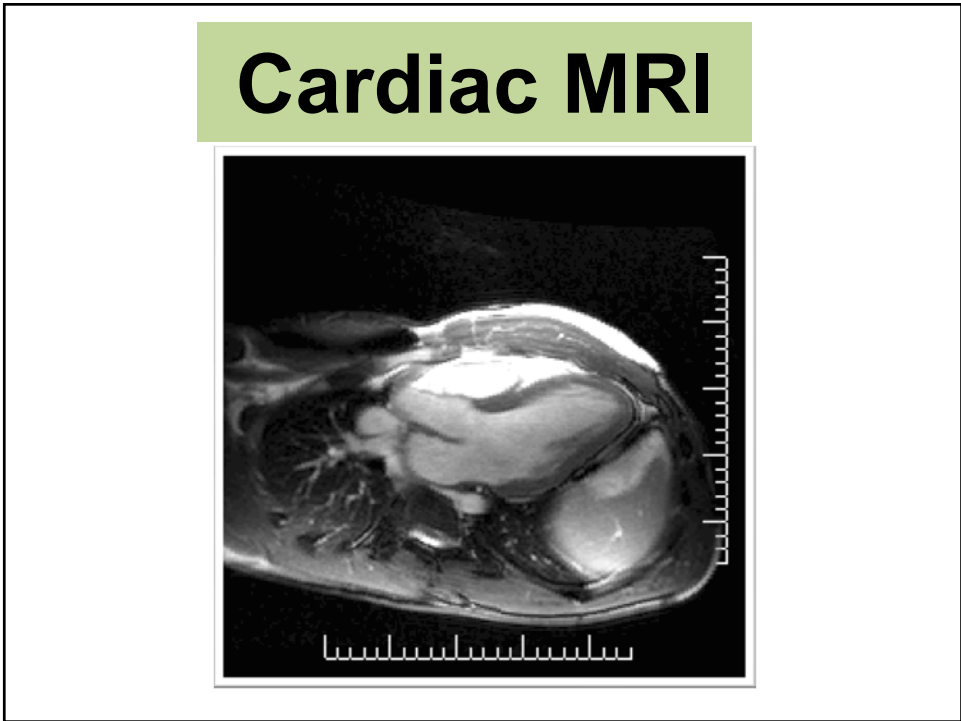
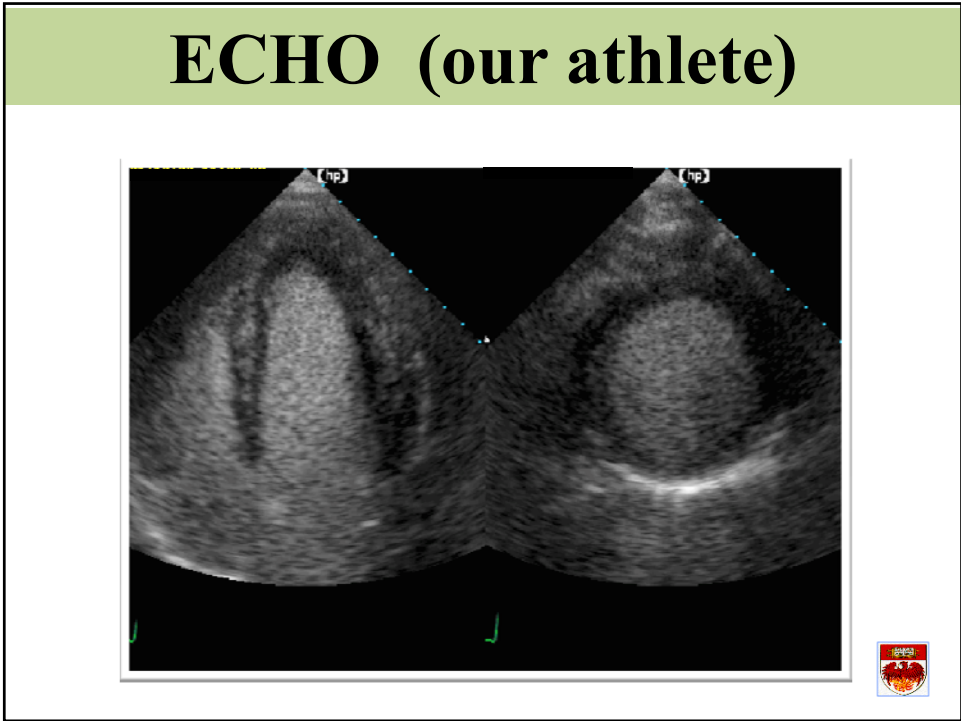


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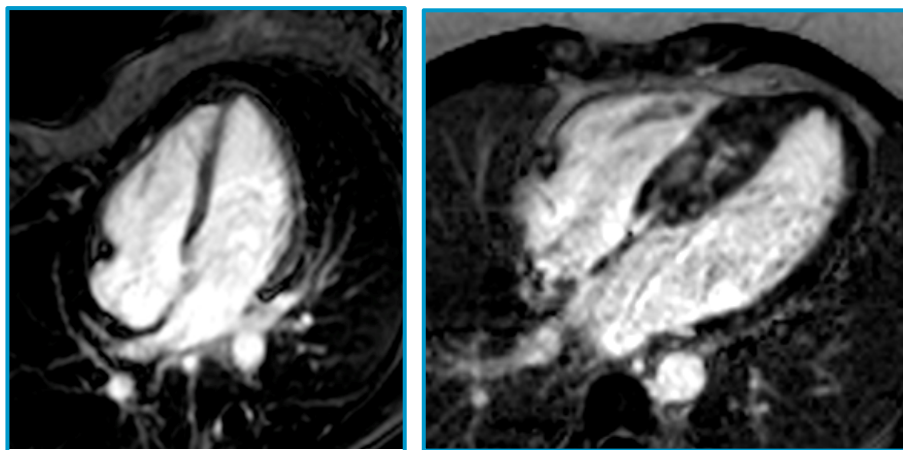
Apical CMP





Delayed Enhancement

LGE Typically Absent



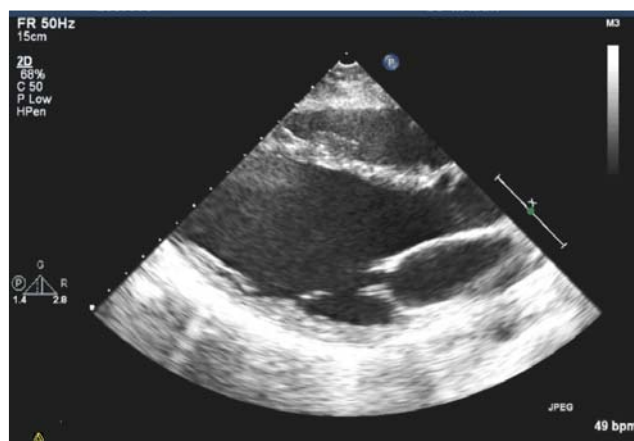
Fact:

- On the basis of this MRI, the patient was diagnosed as having localized HOCM and advised not play in the NBA.

A 24 year old athlete who plays a professional sport received his annual echocardiogram.

- **In the pre-season his physician heard an abnormal murmur**
- **The team manager does not know whether the team should sign his multi-million contract**

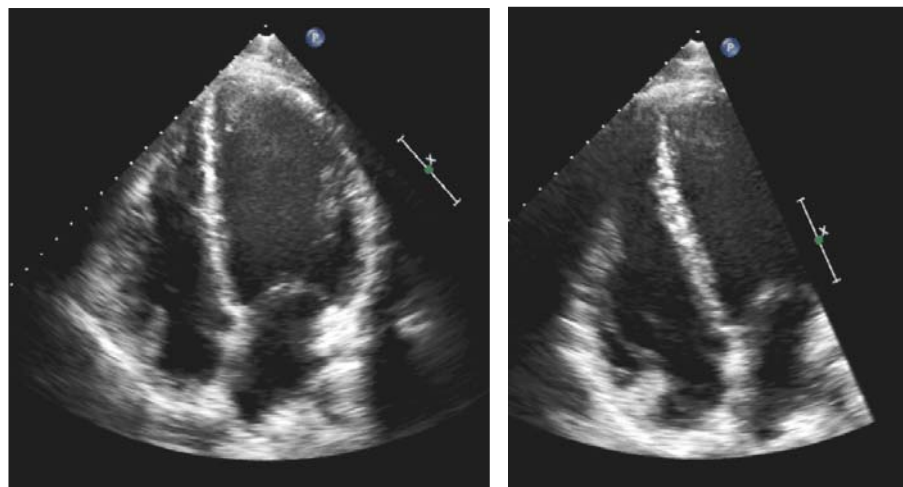
TTE: PLX



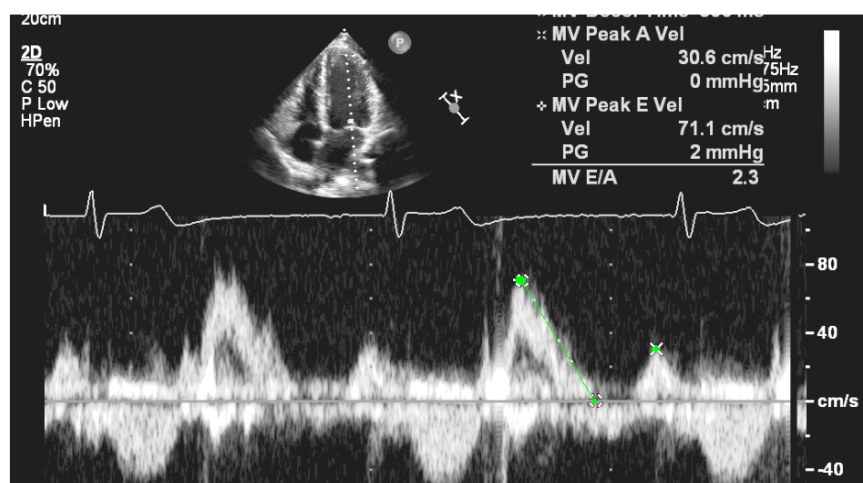
LVIDD 6.06 cm
LVIDS 4.06
PWT 1.18 cm

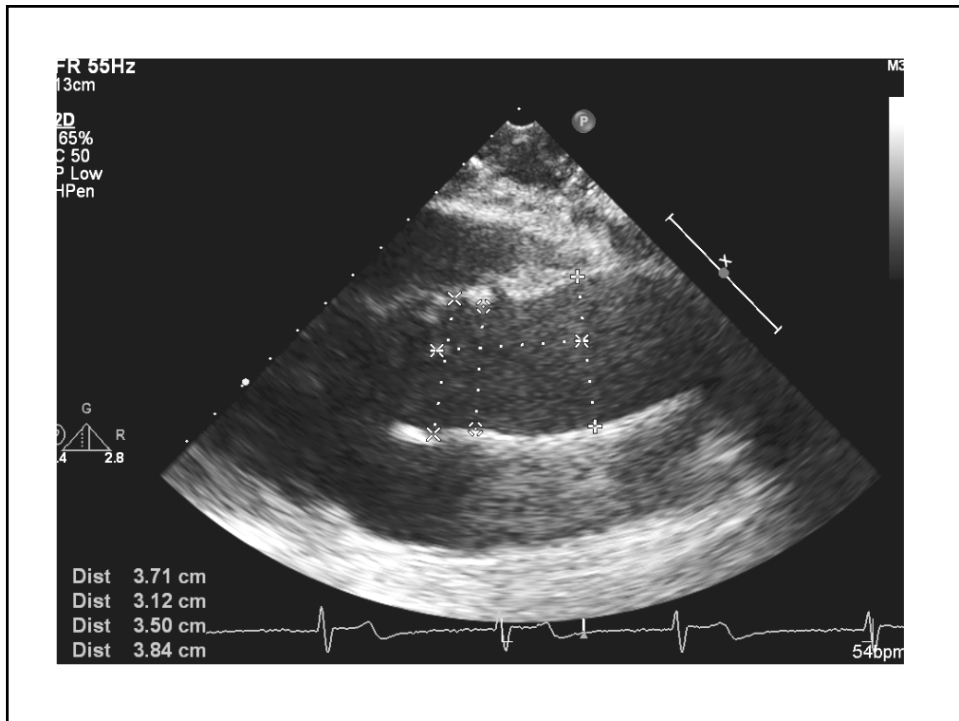
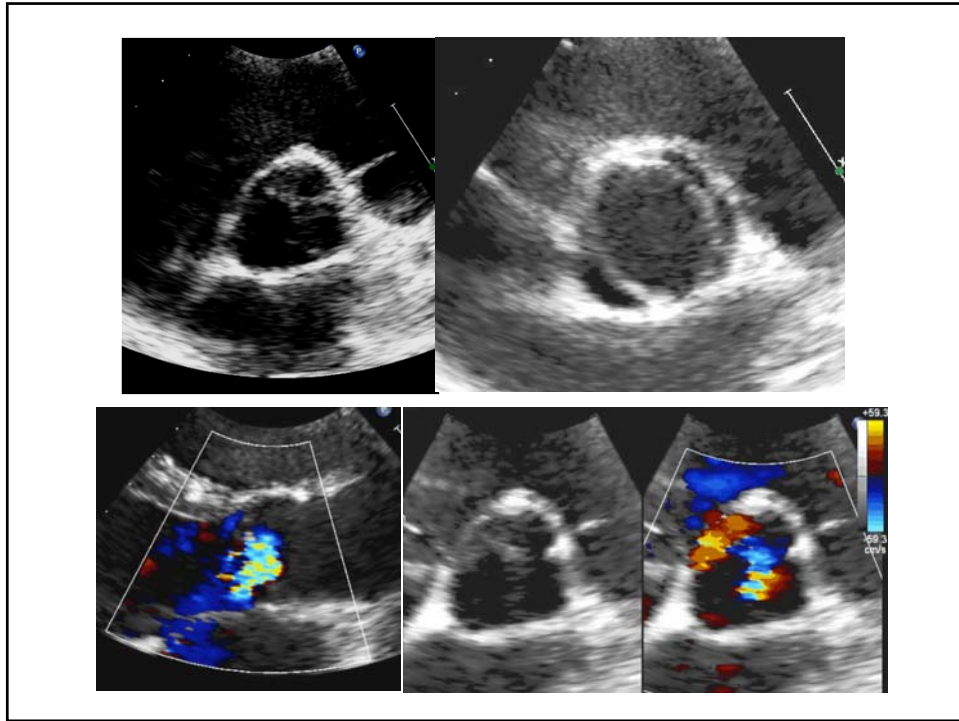
TTE: A4C

LVEF = 61%
LViD = 4.9cm



Doppler LV inflow





Guidelines: Chronic severe AR

- **Asymptomatic patients and LV systolic dysfunction (LVEF<50%)**
- **Reasonable with normal systolic function but with severe LV dilatation (LVESD >50mm)**
- **Operative intervention to repair the aortic sinuses or replace the ascending aorta is indicated in patients with a bicuspid aortic valve if the diameter of the aortic sinuses or ascending aorta is greater than 5.5 cm**

Athletes with VHD

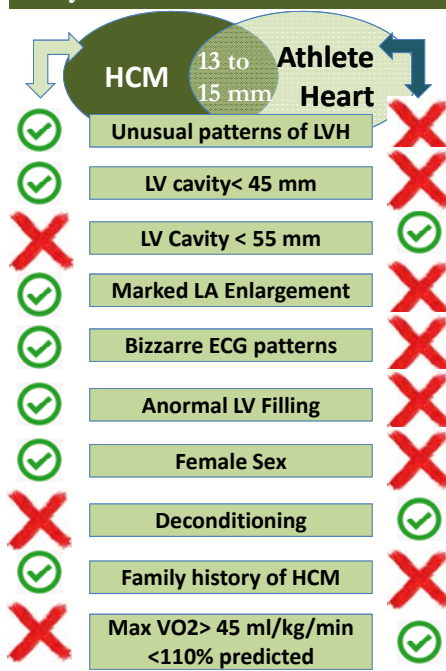
- **Moderate VHD are compatible with participation in most sports, with the exception of those with high static and high dynamic components.**
- **Athletes with severe VHD should be disqualified from participating in competitive sports until they have undergone the appropriate therapeutic procedure.**
- **Following valve repair or replacement, athletes can return to sports, although they should avoid high static and high dynamic sports.**

Athletes Heart

Pathological vs. Physiological Adaptation

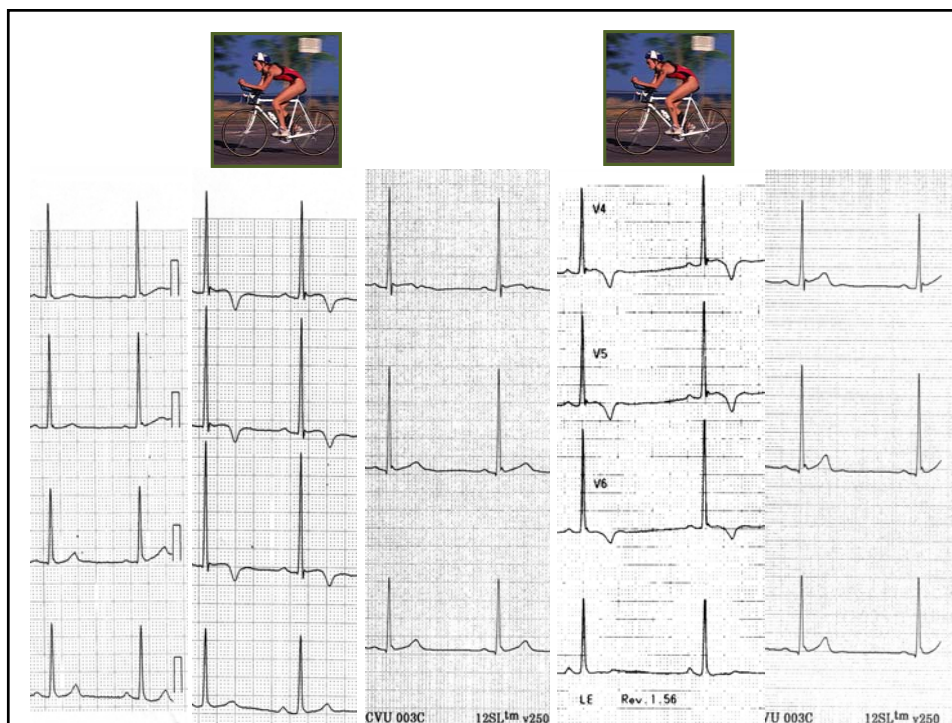
•The distinction between physiological remodeling of the athletes heart from pathological LVH in HCM is challenging with important implications for pre-participation screening

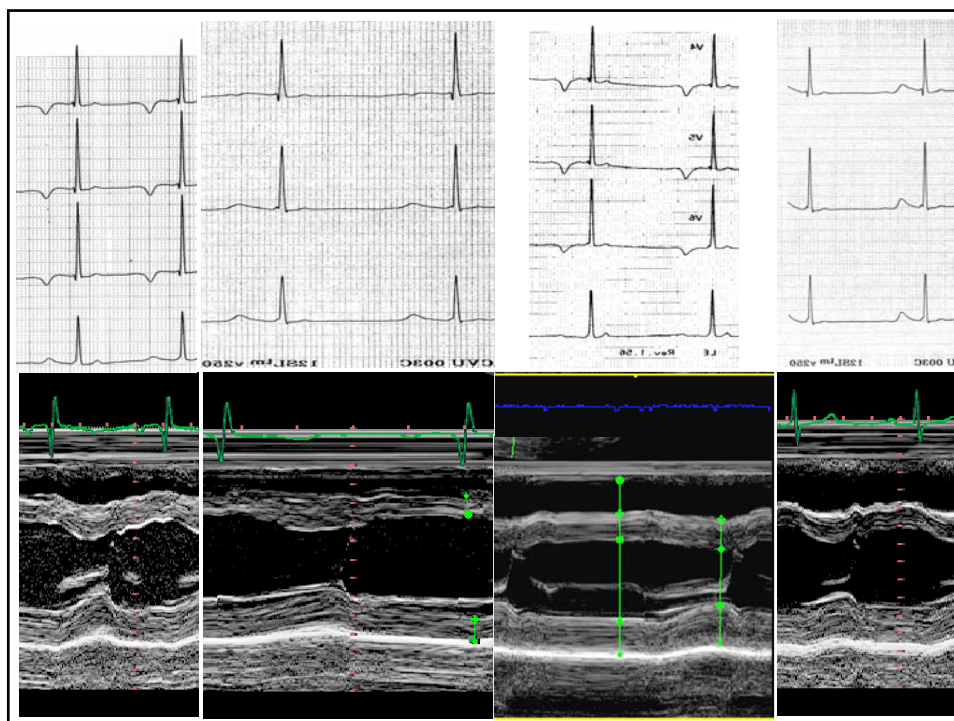
Grey Zone of LV Wall Thickness



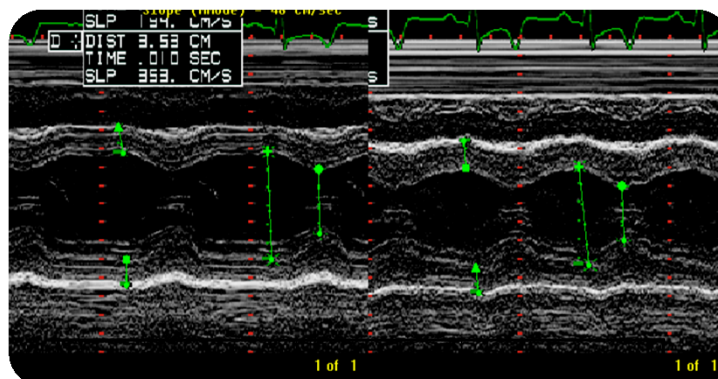
Detraining

- Complete cessation of training with intermittent imaging (6 weeks to 6 months)
- Discontinuing training affects wall thickening; cavity dilatation is not really modified
- Reversibility of LVH after detraining is in favor of athletes heart

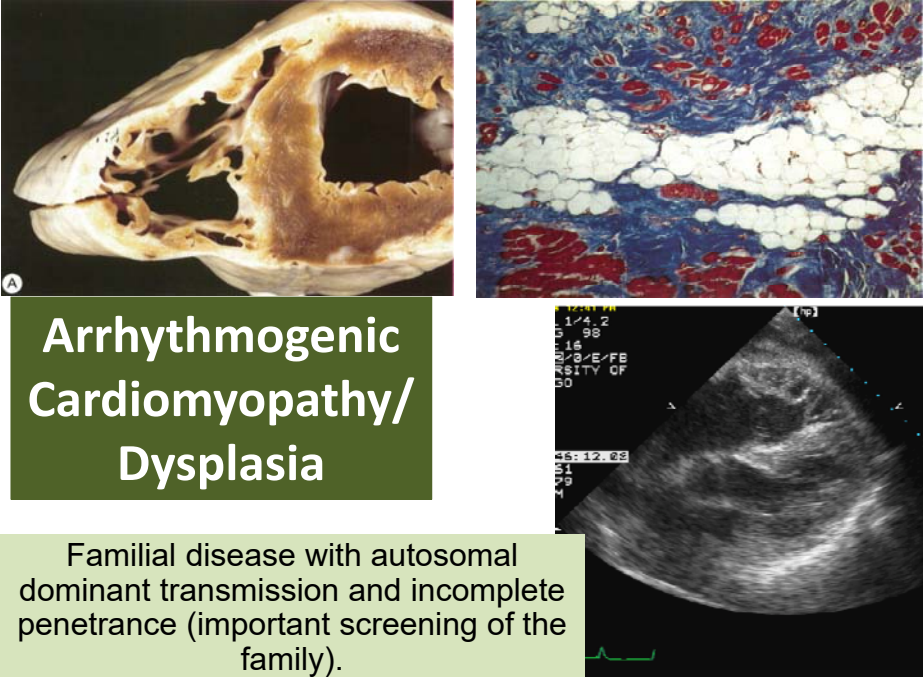




Echo Change caused by deconditioning



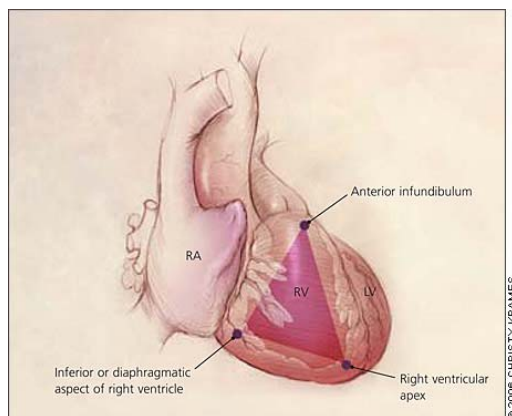
LVIDd(cm)	5.2	5.2
WT(cm)	1.32	1.10



**Arrhythmogenic
Cardiomyopathy/
Dysplasia**

Familial disease with autosomal dominant transmission and incomplete penetrance (important screening of the family).

The triangle of dysplasia

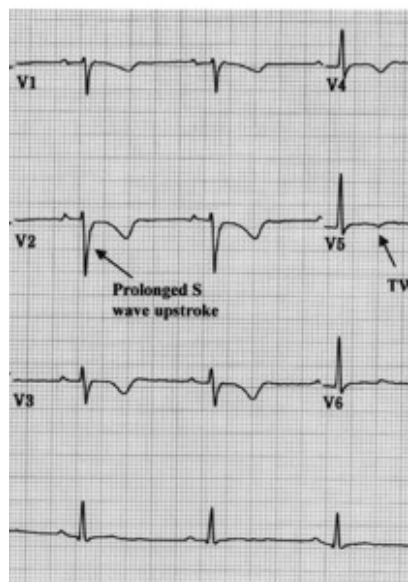


- Initially localized to the “triangle of dysplasia” then spread to the entire right ventricle, and in some cases to the left ventricle

Clinical Presentation: ARVD

- Estimated prevalence 1 in 5000
- Patients usually young and health
- Males are affected three times more than females
- Symptoms vary from lightheadedness and palpitations to syncope and sudden death
- Symptoms may be exercise induced and can occur in athletes

ARVD: ECG



Terminal slurring QRS (epsilon wave)



Task Force (Revised Criteria)	
MAJOR	
Regional RV akinesia, dyskinesia and aneurysm and one of the following	
PLAX ≥ 32 mm (≥ 19 mm/m ²)	
PSAX ≥ 36 mm (≥ 21 mm/m ²)	
FAC $\leq 33\%$	
MINOR	
Regional RV akinesia or dyskinesia and one of the following	
PLAX 29-32 mm	
PLAX 32-36 mm	
FAC 33-40 mm	

Conclusion

- A small LV cavity with increased WT should raise the suspicion of HOCM
- Disproportionate enlargement of the RV should raise the suspicion of ARVD
- Excessive dilatation of all cardiac chambers with low LVEF and failure to augment normally with exercise should raise the suspicion of DCM
- Reversal of remodeling through detraining may help to determine whether an observed structural abnormality is due to the athlete's heart.

