



# Cardiovascular Impacts of long-term endurance exercise: Implications of athlete' s heart”

*Dr. Gary Mak* 麥耀光

心臟科專科醫生

*IPP of HK Association of Sports Medicine and Sports Science*

*Director of Pro-Cardio Heart Disease and*

*Stroke Prevention center*

*Consultant Cardiologist HK Sports Institute*

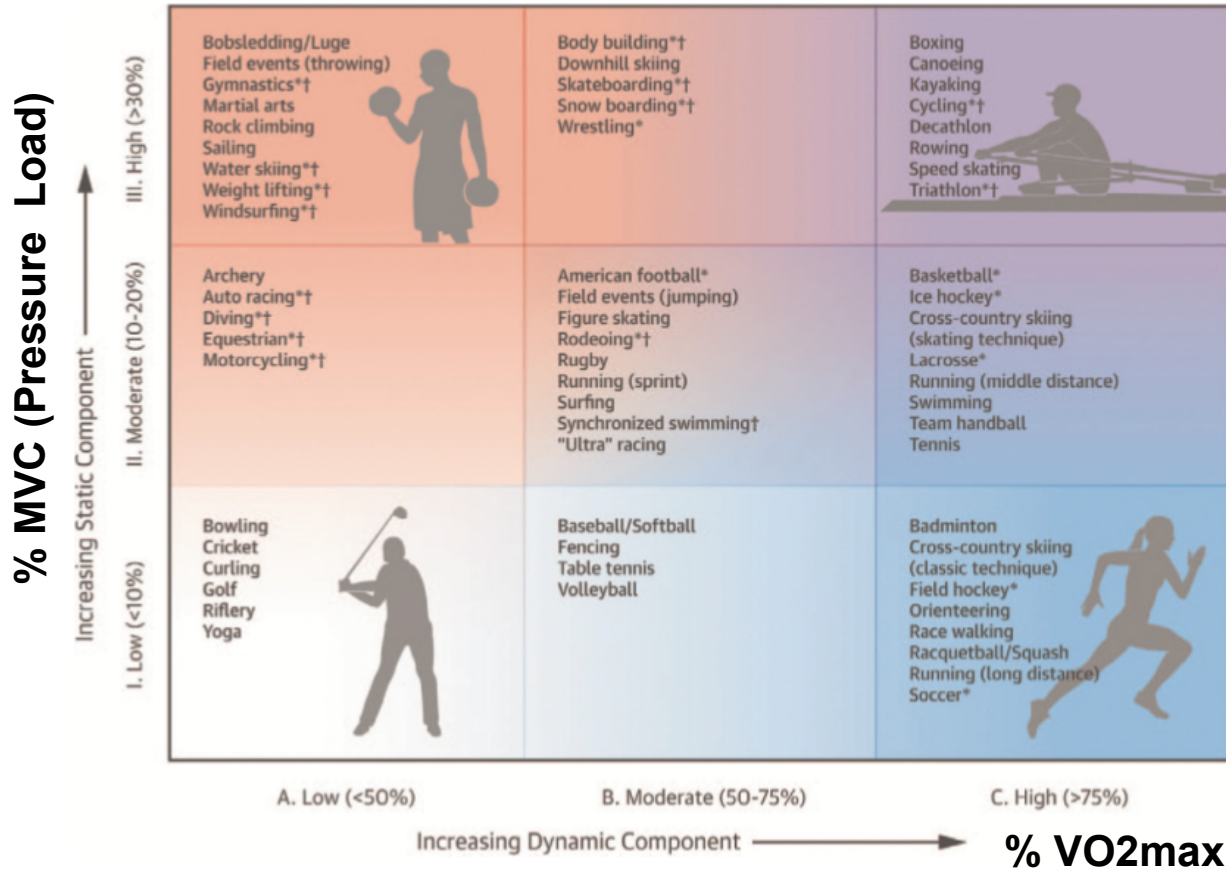
**PRO-CARDIO 心滙**

HEART DISEASE & STROKE PREVENTION CENTRE  
心臟及腦血管病檢查預防中心



# Static and Dynamic Exercise

**FIGURE** Classification of Sports

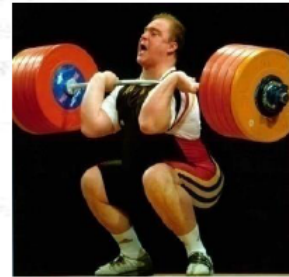
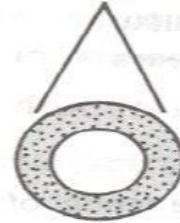
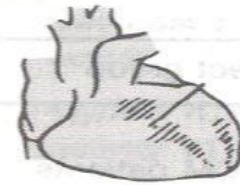
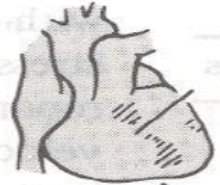


print & web 4C/FPO

This classification is based on peak static and dynamic components achieved during competition; however, higher values may be reached during training. The increasing dynamic component is defined in terms of the estimated percentage of maximal oxygen uptake ( $\dot{V}O_{2max}$ ) achieved and results in an increasing cardiac output. The increasing static component is related to the estimated percentage of maximal voluntary contraction reached and results in an increasing blood pressure load. The lowest total cardiovascular demands (cardiac output and blood pressure) are shown in the palest color, with increasing dynamic load depicted by increasing blue intensity and increasing static load by increasing red intensity. Note the graded transition between categories, which should be individualized on the basis of player position and style of play. \*Danger of bodily collision (see [Table](#) for more detail on collision risk). †Increased risk if syncope occurs. Modified from Mitchell et al. (3) with permission. Copyright © 2005, *Journal of the American College of Cardiology*.

# 'Morganroth Hypothesis'

## Endurance training leads to heart (LV) enlargement



**Endurance-trained athlete**

**Sedentary person free from heart disease**

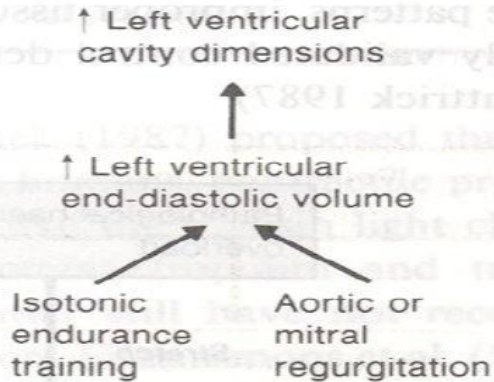
**Resistance-trained athlete**

Eccentric hypertrophy

Concentric hypertrophy

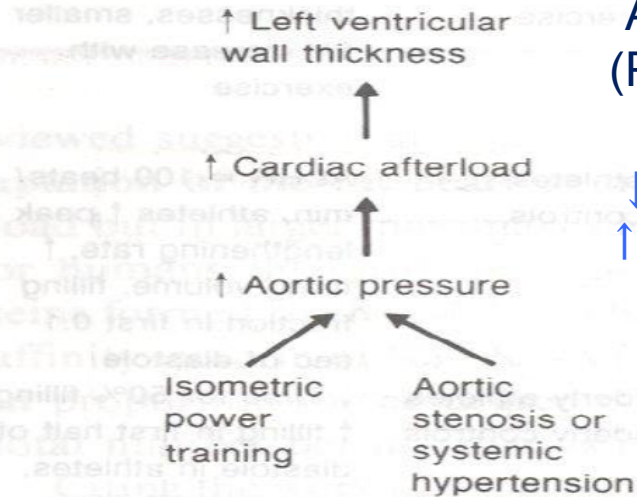
Preload (volume)

↑ ↑ LV volume  
↑ LV mass



Afterload (Pressure)

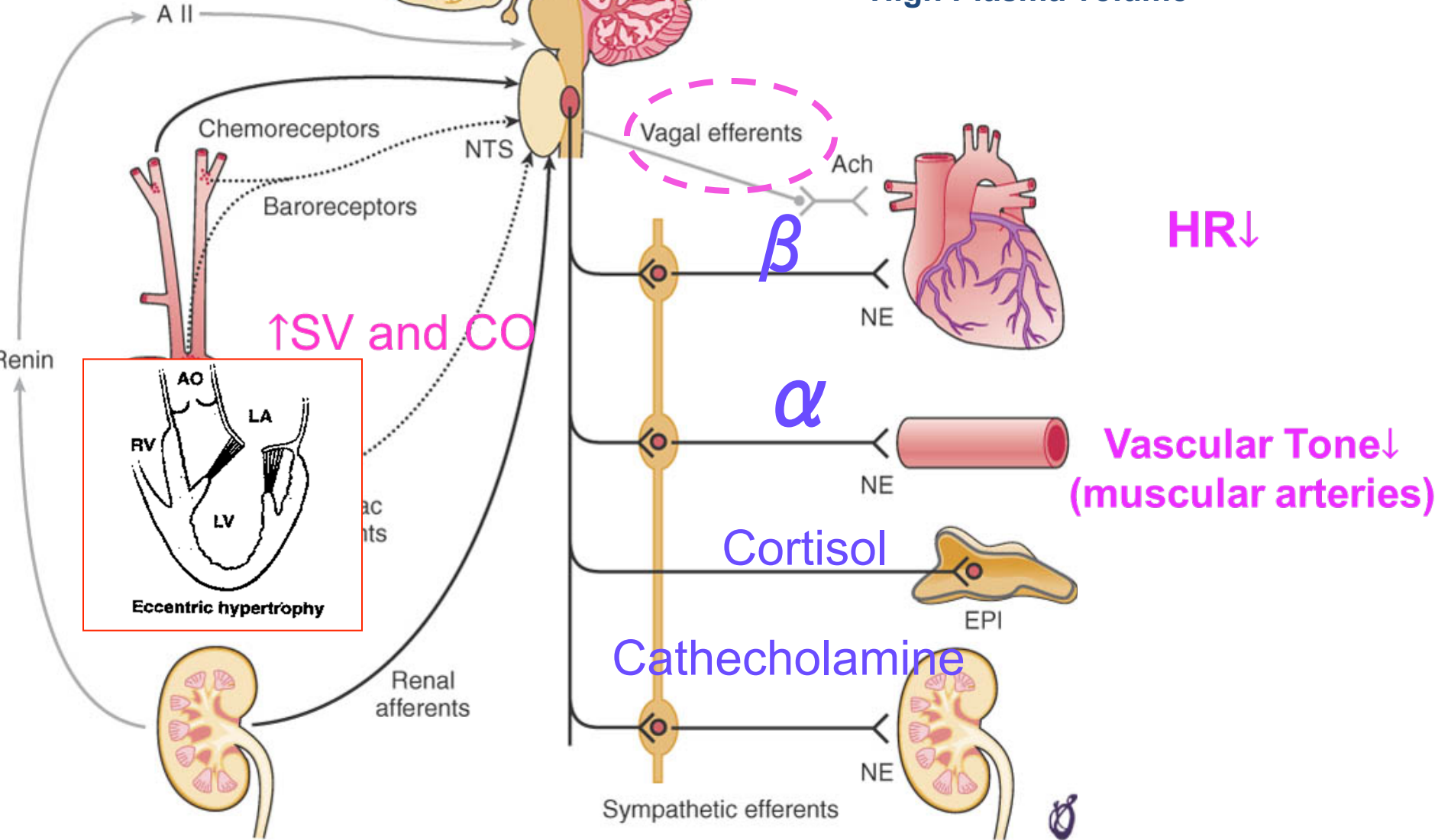
↓ LV volume  
↑ ↑ LV mass



# Endurance Training

- enhanced Vagal modulation
- Eccentric hypertrophy
- High Plasma volume

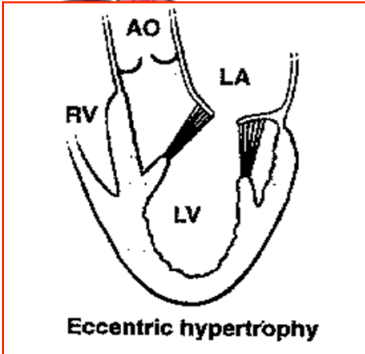
↑ plasma Volume



↑ SV and CO

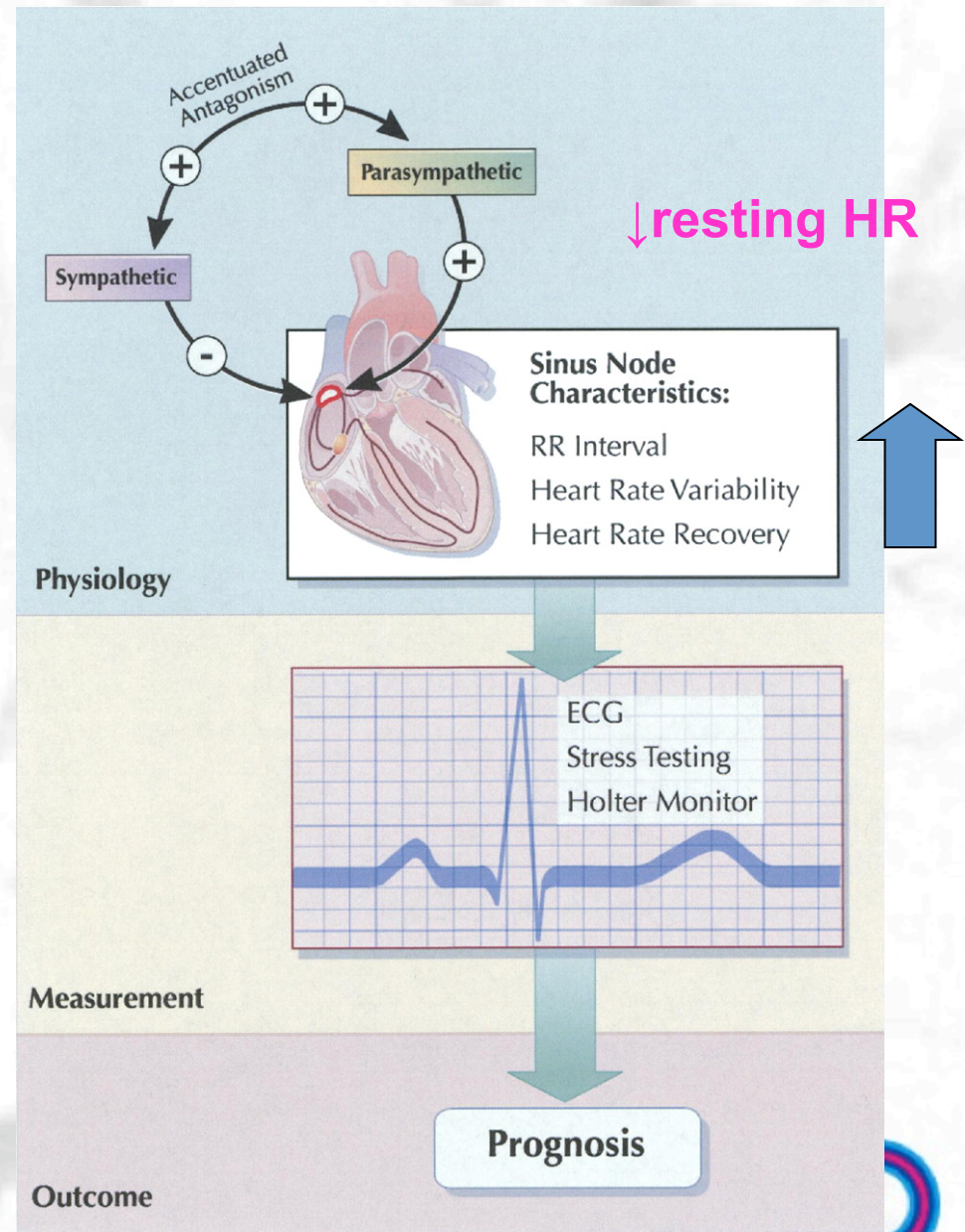
HR↓

Vascular Tone↓  
(muscular arteries)



**Regular Endurance training=>**  
**Enhanced Vagal Modulation**

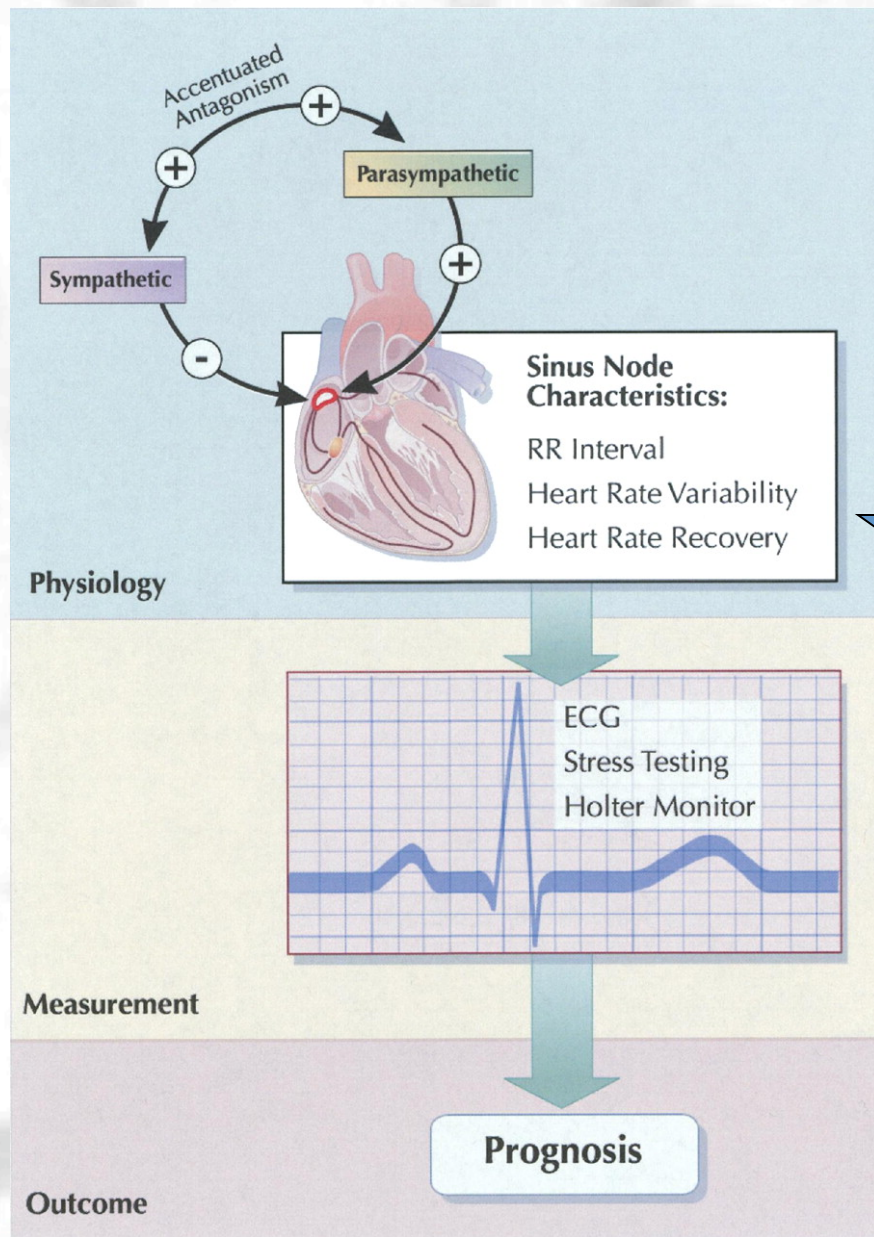
## Autonomic Nervous System & Cardiovascular Response



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Sub-optimal CP reserve /  
Excessive intensive exercise /  
overtraining =>  
**Sympathetic predominance**

## Autonomic Nervous System & Cardiovascular Response



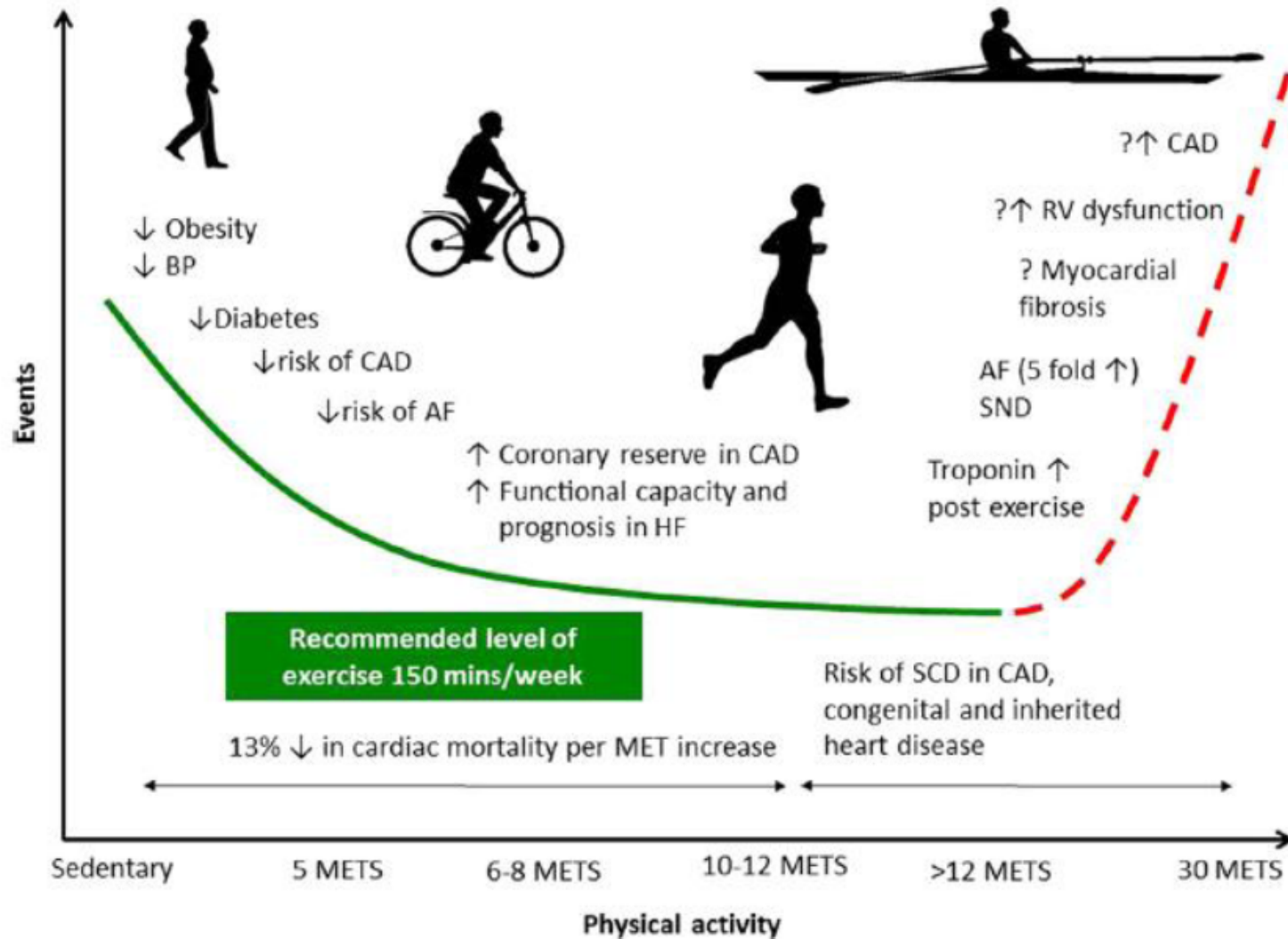
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# Athletes' Heart

- Left Ventricular Hypertrophy (eccentric)
- Effect of high vagal tone
  - Slow HR: Sinus Bradycardia at rest ,rarely < 40 bpm
  - Early repolarization pattern
  - Extra Beats: predispose to increased atrial or ventricular ectopy,
  - Conduction defects: atrioventricular (AV) block

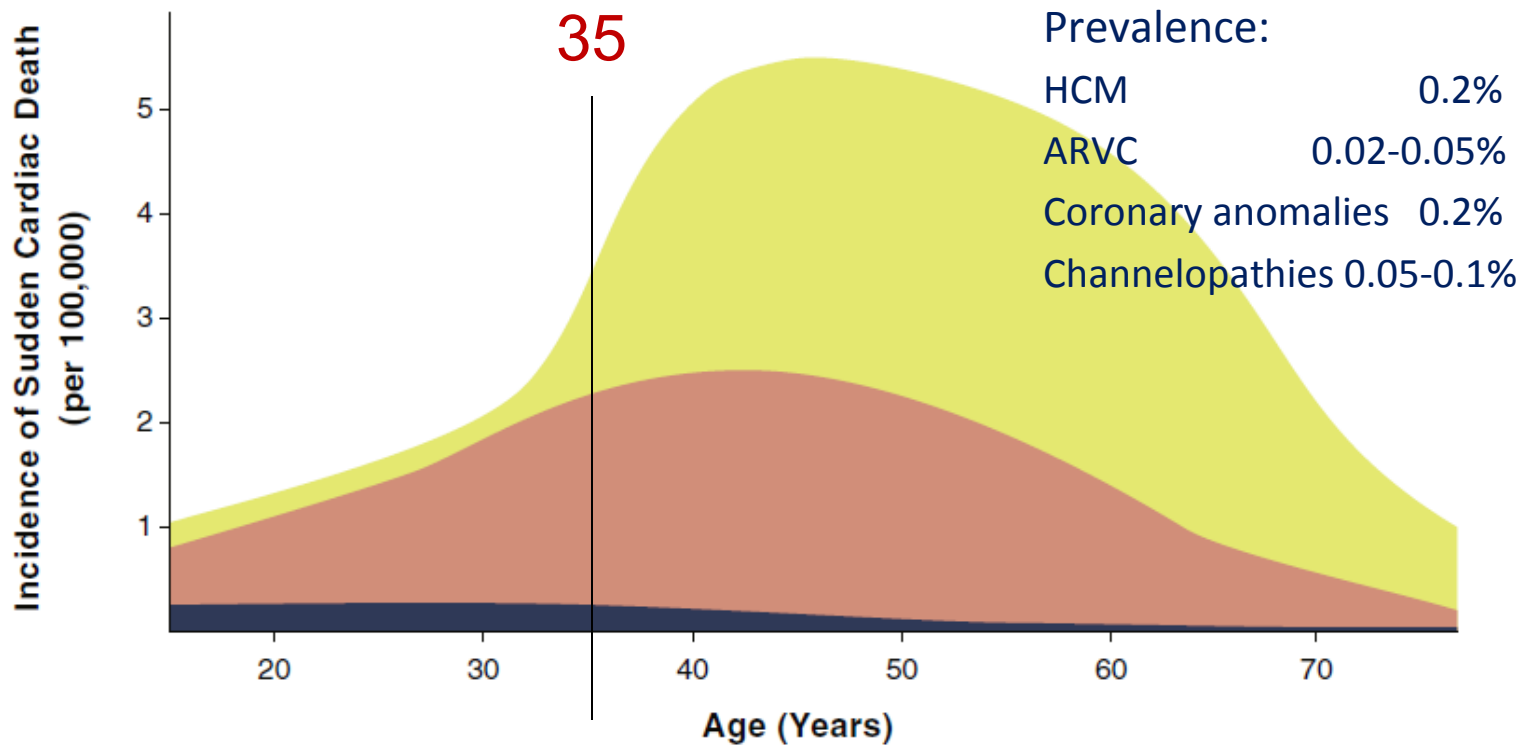
Arrhythmia, esp. Atrial Fibrillation

# The U Shaped Hypothesis



Merghani A et al: The U-shaped relationship between exercise and cardiac morbidity. Trends Cardiovasc Med. 2015 Jun 18. pii: S1050-1738(15)00171-1.





La Gerche et al. JACC: CV IMAGING, 2013

|                        |                                  |                                  |
|------------------------|----------------------------------|----------------------------------|
| <b>Channelopathies</b> | <b>Cardiomyopathies</b>          | <b>Coronary Artery Pathology</b> |
| Long QT Syndrome       | Hypertrophic Cardiomyopathy      | Atherosclerotic                  |
| Brugada Syndrome       | Arrhythmogenic RV Cardiomyopathy | Anomalous Coronary Ostia         |
| Catecholaminergic VT   | Dilated Cardiomyopathy           |                                  |

**Figure 1. Age-Dependent Changes in Incidence and Etiology of Sudden Cardiac Death**

This figure represents an interpretation of the combined experience from studies that have assessed the causes of sudden cardiac death in athletes (3,5,8,9,11–13). While the majority of deaths may be attributed to inherited cardiomyopathies and channelopathies in those younger than 30 years old, there is no absolute cutoff. Thus athletes in their thirties and forties (the median age in many competitive sports) are at greatest risk of sudden cardiac death caused by inherited and acquired causes. RV = right ventricular; VT = ventricular tachycardia.

# Pre-participation Evaluation For Young Competitive Athletes



# The 12-Element AHA Recommendations for Preparticipation Cardiovascular Screening of Competitive Athletes, 2007 Update

## Family History

1. Premature sudden cardiac death
2. Heart disease in surviving relatives less than 50 years old

## Personal History

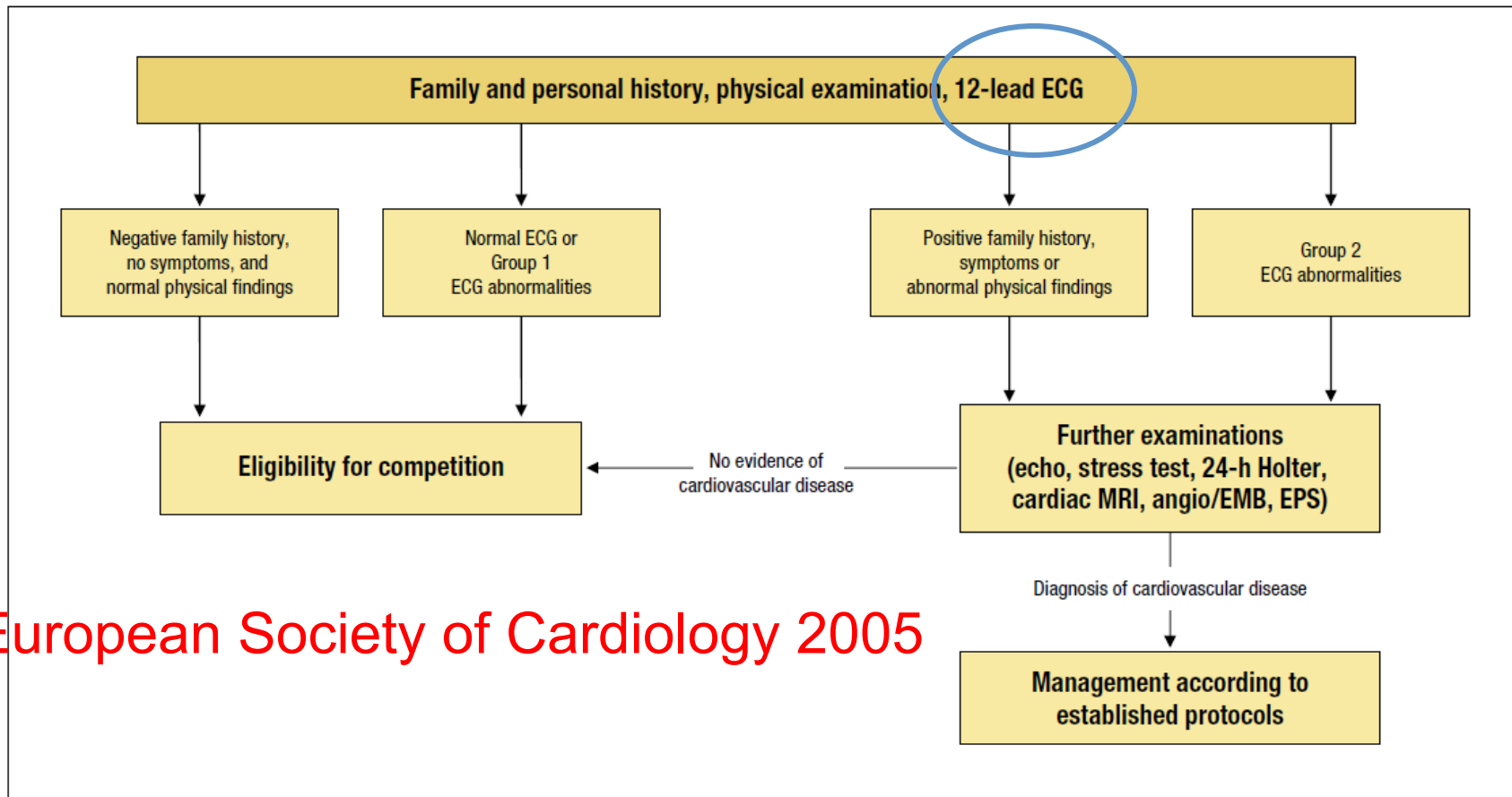
3. Heart murmur
4. Systemic hypertension
5. Fatigue
6. Syncope/near-syncope
7. Excessive/unexplained exertional dyspnea
8. Exertional chest pain

## Physical Examination

9. Heart murmur (supine/standing\*)
10. Femoral arterial pulses (to exclude coarctation of aorta)
11. Stigmata of Marfan syndrome
12. Brachial blood pressure measurement (sitting)

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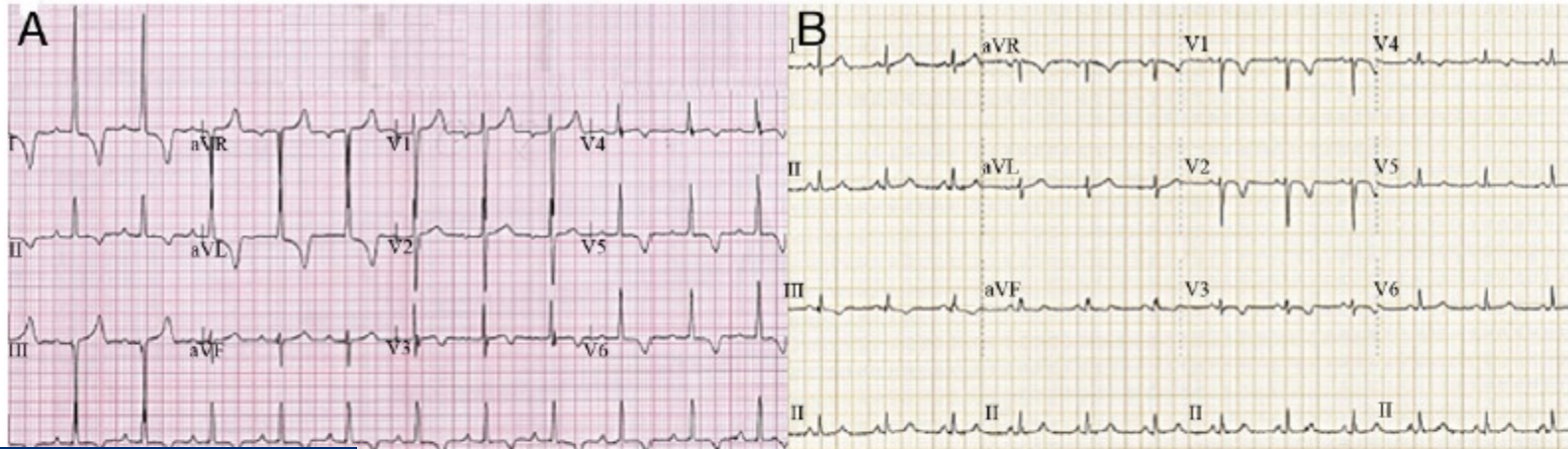
\*In particular, to identify heart murmur consistent with dynamic obstruction to left ventricular outflow. From Maron BJ, et al. *Circulation* 1996;94:850–6, reprinted with permission of the American Heart Association.



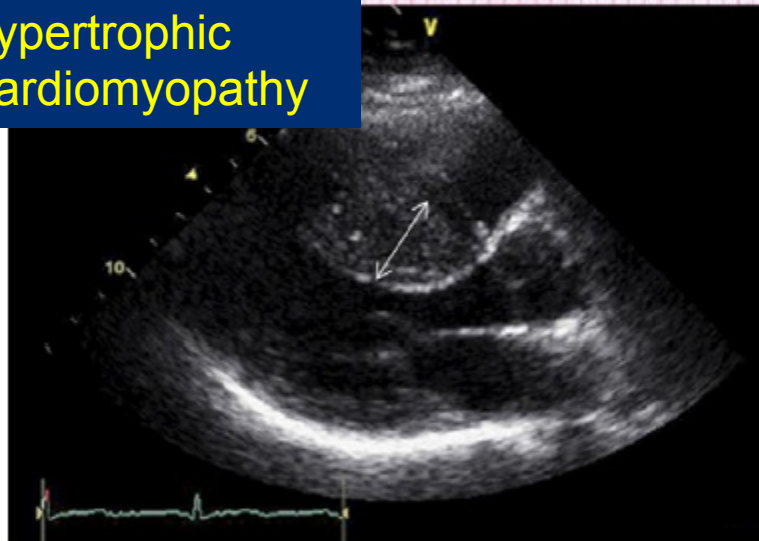
European Society of Cardiology 2005

Figure 2: Flow diagram illustrating screening work-up according to the proposed criteria for ECG interpretation in trained athletes [15].

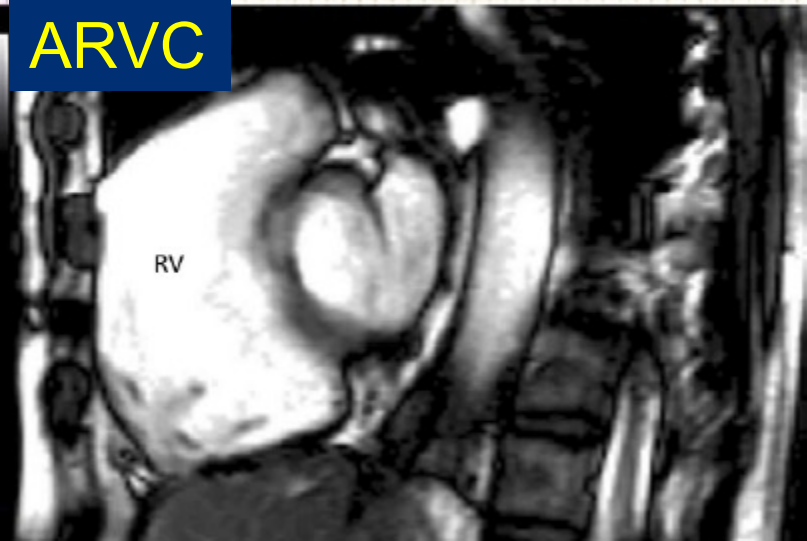
# ECG Abnormalities are present in up to 95% of Hypertrophic Cardiomyopathy and 80% of ARVC



Hypertrophic  
Cardiomyopathy



ARVC

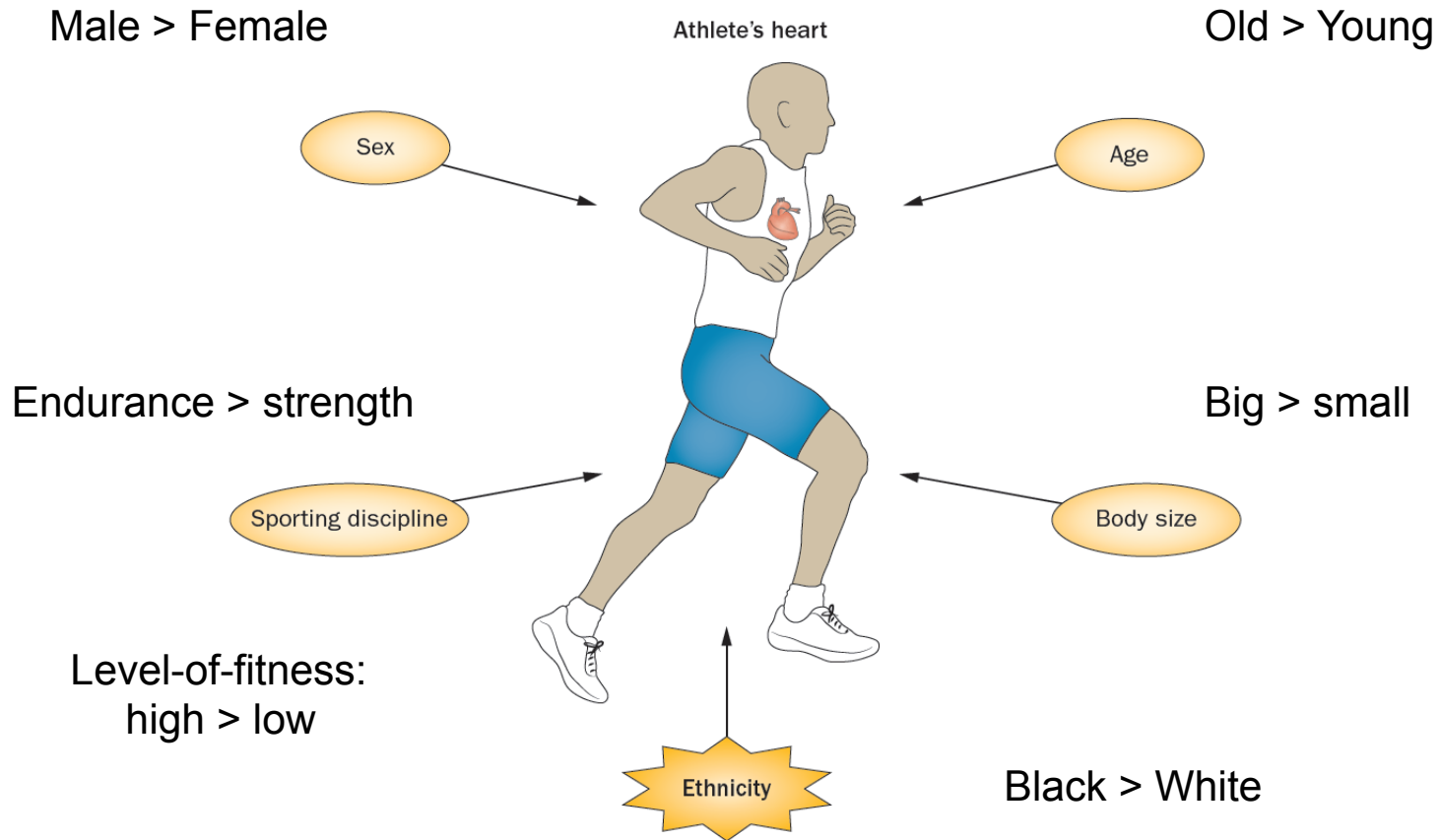


physiological adaptive ECG changes  
(Athlete's Heart)

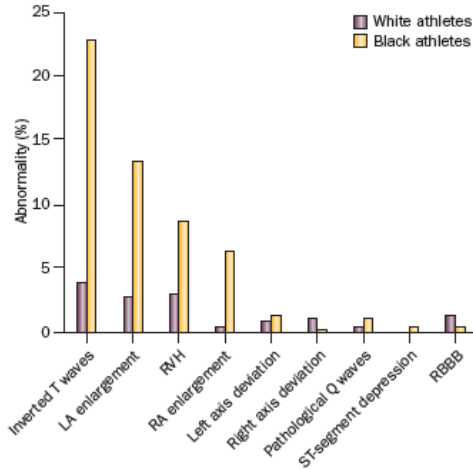
VS

pathological ECG abnormalities

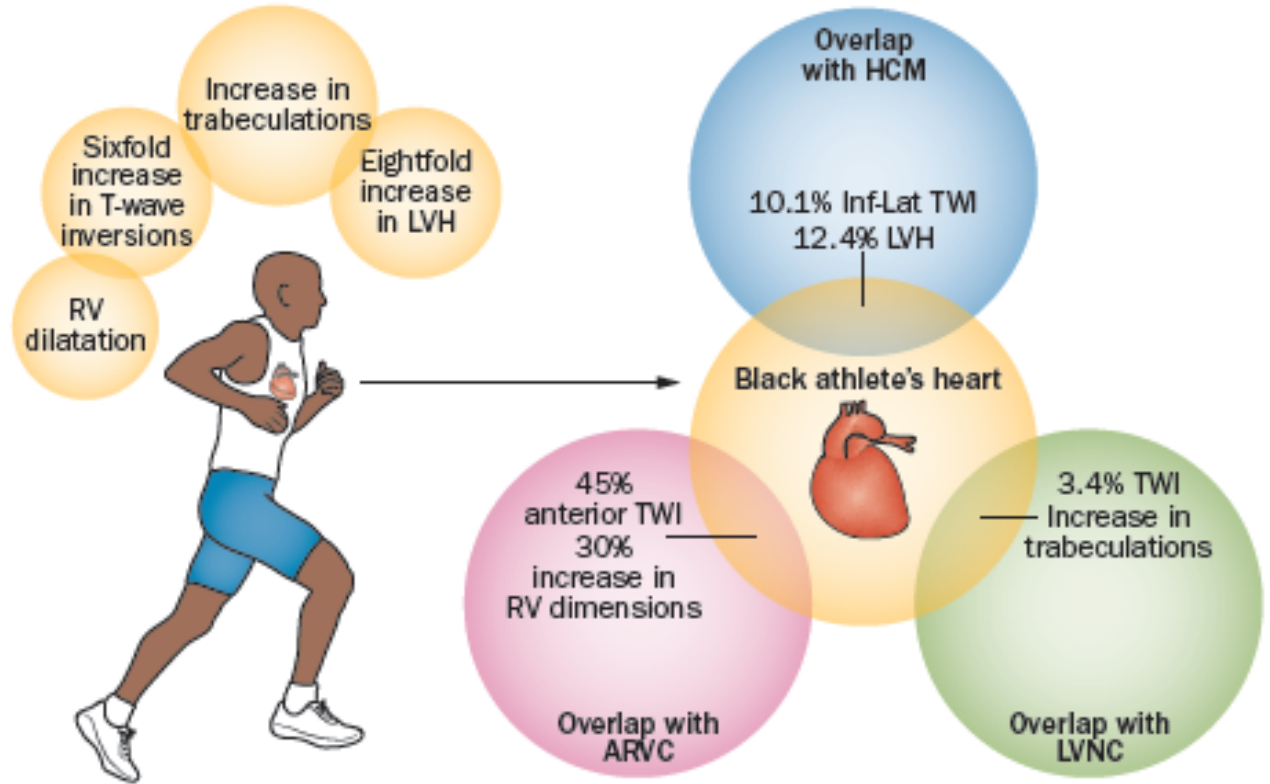
# Athlete's Heart



# Black Athletes



**Figure 7** | Prevalence of abnormal electrocardiographic patterns other than T-wave inversions in a large cohort of adult black ( $n=904$ ) or white ( $n=1,819$ ) elite athletes undergoing preparticipation evaluation.<sup>122</sup> Abbreviations: LA, left atrial; RA, right atrial; RBBB, right bundle branch block; RVH, right ventricular hypertrophy.





## Group 1: common and training-related ECG changes

Sinus bradycardia

First-degree AV block

Incomplete RBBB

Early repolarization

Isolated QRS voltage criteria for left ventricular hypertrophy

ESC 2005

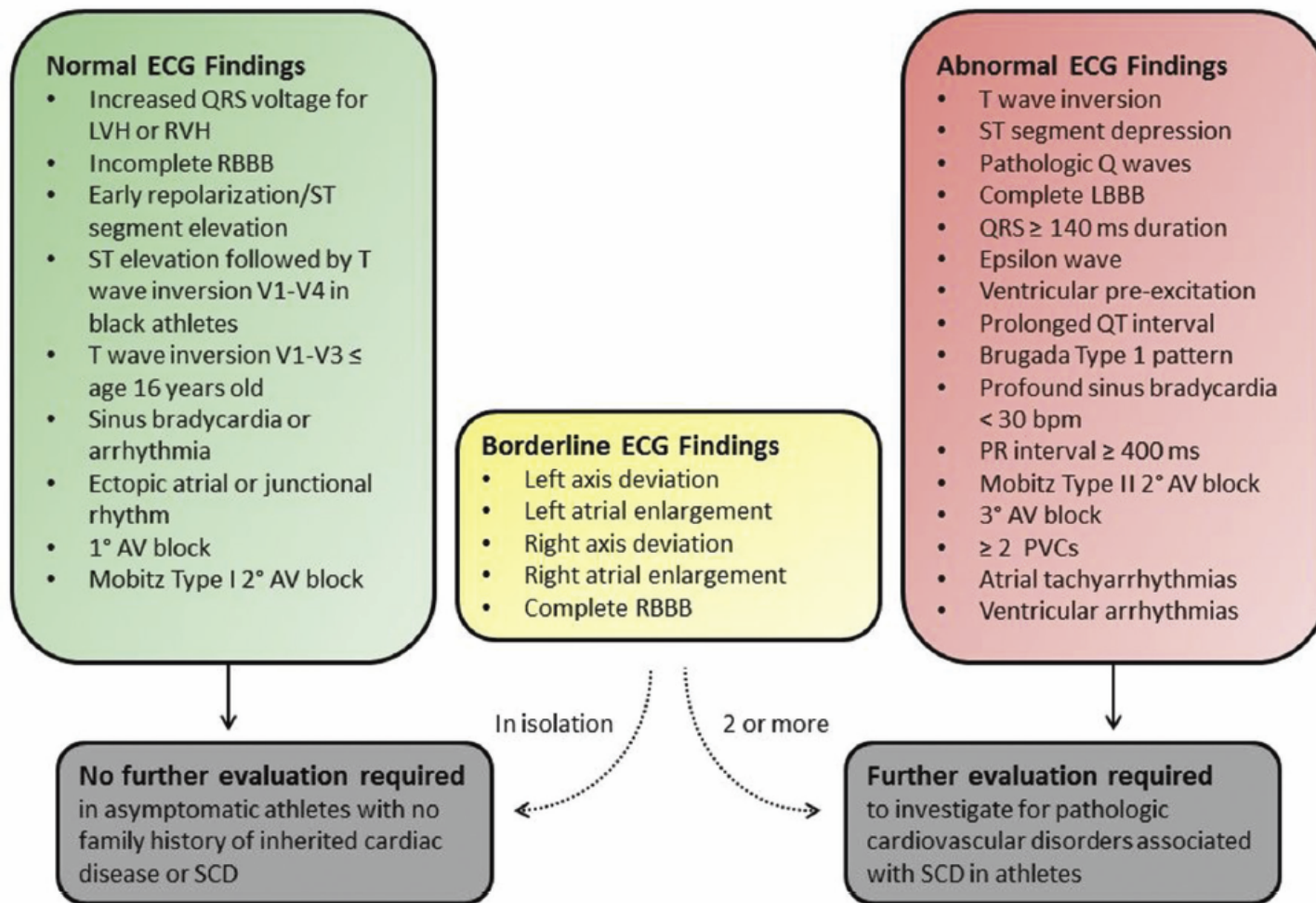
“physiological expressions” should be consistent with the gender, age, and race, the level of training and type of sports.

1. Sinus bradycardia ( $\geq 30$  bpm)
2. Sinus arrhythmia
3. Ectopic atrial rhythm
4. Junctional escape rhythm
5. 1° AV block (PR interval  $> 200$  ms)
6. Mobitz Type I (Wenckebach) 2° AV block
7. Incomplete RBBB
8. Isolated QRS voltage criteria for LVH
  - ▶ *Except:* QRS voltage criteria for LVH occurring with any non-voltage criteria for LVH such as left axis deviation, ST segment depression, T-wave inversion or pathological Q waves
9. Early repolarisation (ST elevation, J-point elevation, J- or terminal QRS slurring)
10. Convex ('domed') ST segment elevation combined with T-wave inversion in leads V1–V4 in black/African athletes

**These common training-related ECG alterations are physiological adaptations to regular exercise, considered normal variants in athletes and do not require further evaluation in asymptomatic athletes.**

AV, atrioventricular; bpm, beats per minute; LVH, left ventricular hypertrophy; ms, milliseconds; RBBB, right bundle branch block.

# International Criteria for Electrocardiographic Interpretation in Athletes 2017



**Figure 1** International consensus standards for ECG interpretation in athletes. AV, atrioventricular; LBBB, left bundle branch block; LVH, left ventricular hypertrophy; PVC, premature ventricular contraction; RBBB, right bundle branch block; RVH, right ventricular hypertrophy; SCD, sudden cardiac death.

Prevalence of an abnormal ECG was significantly reduced to 5.3% when using the 2014 Refined Criteria (International Criteria 2017) compared with the 2013 Seattle Criteria (11.6%) and the 2010 ESC recommendations (22.3%), respectively, proved 100% sensitive, identifying all cases of serious cardiac pathology

**Table 2** Performance comparison of three ECG interpretation criteria (ESC recommendations vs Seattle Criteria vs Refined Criteria)

|   | Combined<br>(n=2491) | Arabic<br>(n=1367) | Black<br>(n=748) | Caucasian<br>(n=376) |
|---|----------------------|--------------------|------------------|----------------------|
| Prevalence of an abnormal ECG using ESC recommendations | 555 (22.3%)          | 261 (19.1%)        | 224 (29.9%)      | 70 (18.6%)           |
| Prevalence of an abnormal ECG using Seattle Criteria    | 289 (11.6%)          | 133 (9.7%)         | 124 (16.6%)      | 32 (8.5%)            |
| Prevalence of an abnormal ECG using Refined Criteria    | 132 (5.3%)           | 49 (3.6%)          | 75 (10%)         | 8 (2.1%)             |
| Number of identified conditions associated with SCD     | 10 (7 HCM; 3 WPW)    | 4 (2 HCM; 2 WPW)   | 6 (5 HCM; 1 WPW) | 0                    |
| FPR when using ESC recommendations                      | 21.9%                | 18.8%              | 29.1%            | 18.6%                |
| FPR when using Seattle Criteria                         | 11.2%                | 9.4%               | 15.8%            | 8.5%                 |
| FPR when using Refined Criteria                         | 4.9%                 | 3.3%               | 9.2%             | 2.1%                 |

ESC, European Society of Cardiology; FPR, false-positive rate; HCM, hypertrophic cardiomyopathy; SCD, sudden cardiac death; WPW, Wolff-Parkinson-White syndrome.

# The Young Athlete's Heart

OX1  
S5-1  
27Hz  
18cm  
2D  
HGen  
Gn 50  
C 50  
3/2/0  
75 mm/s



10-20% increase in left ventricular wall thickness.

10% increase in LV and RV cavity.

45% increase in LV mass

## Exercise, Diet, and the Heart

### Serial Left Ventricular Adaptations in World-Class Professional Cyclists

Implications for Disease Screening and Follow-Up

Eric Abergel, MD,\* Gilles Chatellier, MD,† Albert A. Hagege, MD, PhD,\* Agnes Oblak, MD,\*  
Ales Linhart, MD,\* Alain Ducardonnet, MD,‡ Joël Menard, MD, PhD§

Paris, France

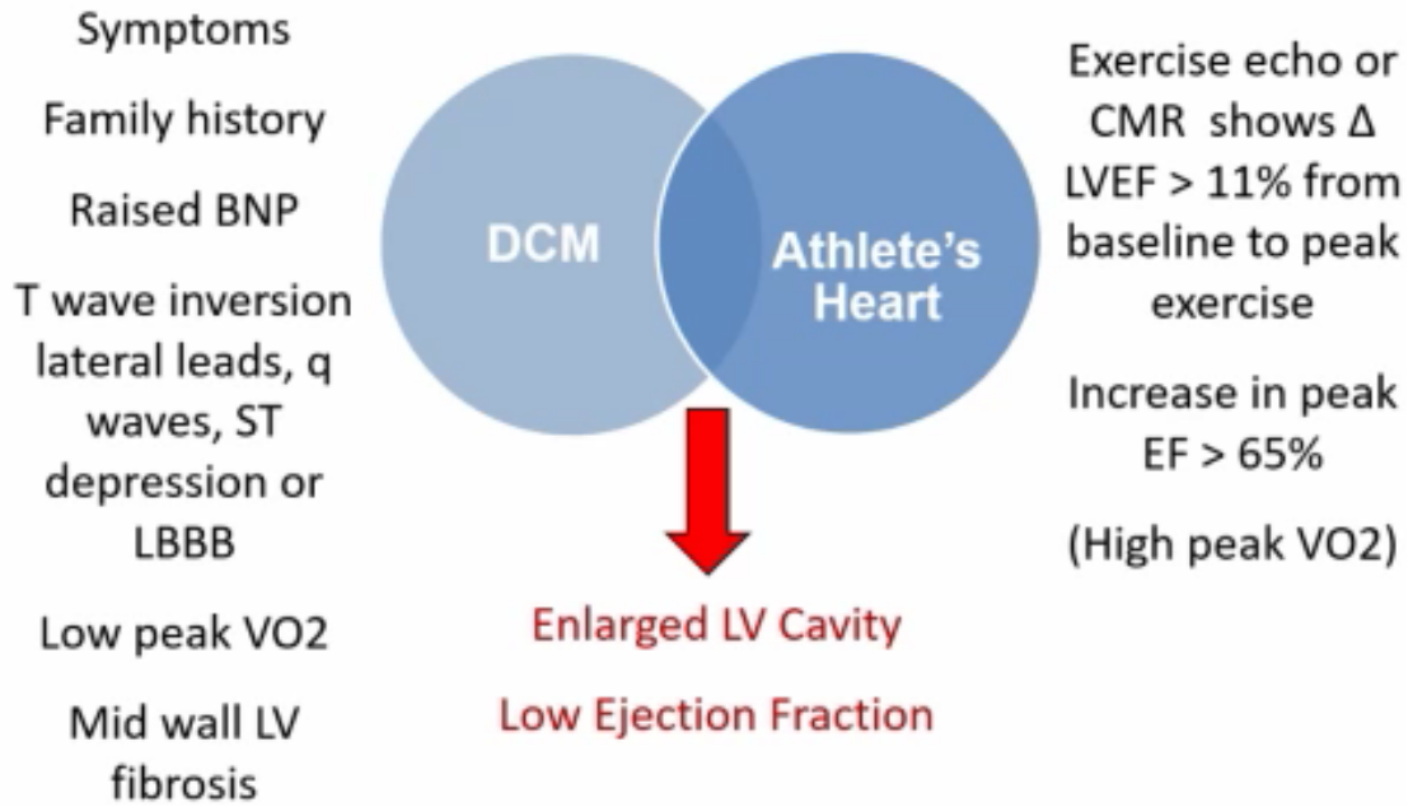


- 286 Tour de France cyclists
- 146 (51.4%) had a LVED > 60 mm
- Of these 11.7% had a LVEF < 52%

LV Dilatation with borderline low LVEF

Are these Dilated Cardiomyopathy?

## Athlete's Heart vs Dilated Cardiomyopathy



# Discriminating Variables Favouring DCM During Exercise Echocardiography

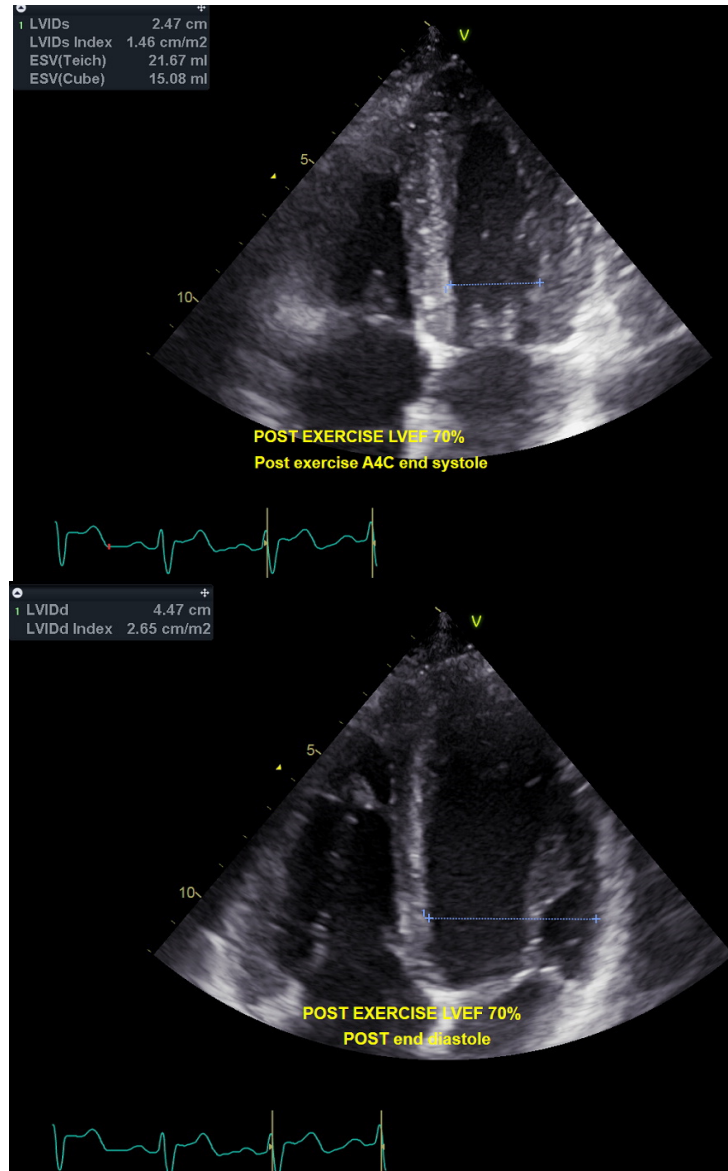
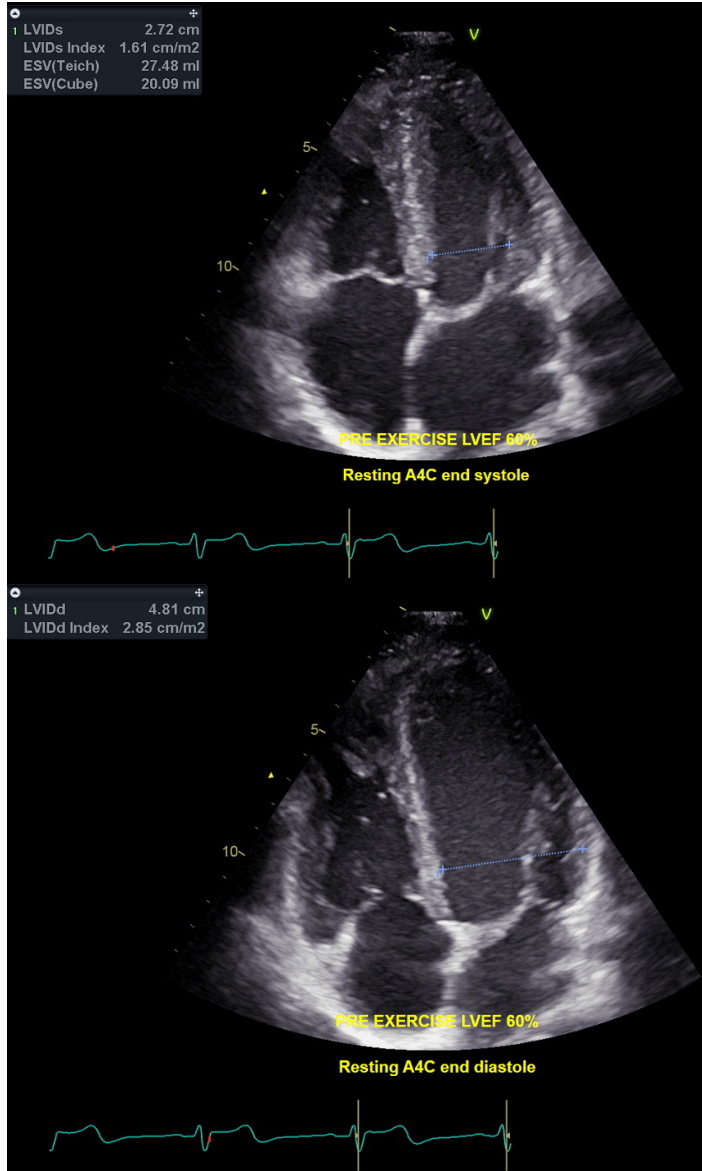
Millar L (unpublished)

| Variable                                    | AUC   | Sensitivity | Specificity | p        |
|---|-------|-------------|-------------|----------|
| E' Lateral Peak (<25cm/s)                   | 0.578 | 71%         | 50%         | p=0.401  |
| S' Lateral Peak ( $\leq$ 23cm/s)            | 0.748 | 91%         | 53%         | p<0.002  |
| Stroke Volume Peak ( $\leq$ 94ml)           | 0.754 | 61%         | 100%        | p<0.0004 |
| LV Ejection Fraction ( $\leq$ 63%)          | 0.898 | 85%         | 91%         | p<0.0001 |
| $\Delta$ LV Ejection Fraction ( $\leq$ 11%) | 0.891 | 83%         | 95%         | P<0.0001 |





# Stress Echo



# Conclusion I

- Regular endurance exercise induces significant cardiovascular adaptations including high parasympathetic tone, heart chambers enlargement with eccentric hypertrophy as well as increase of overall plasma volume resulting in a high cardiac output on exercise.
- The degree of adaptive changes varies with gender, age, body size, ethnicity, sports discipline, intensity and duration of exercise as well as the level of fitness.
- These adaptive changes would result in physiological and structural cardiovascular changes that might create confusions and mis-interpretations when one performs Pre-participation Physical Evaluation.

# Conclusion II

- Major progress in the recognition of physiological changes in resting ECG were identified and has significantly reduced the positive rate with high degree of specificity.
- LV dilatation with borderline low LVEF is common in high endurance athletes and often confused with dilatated cardiomyopathy.
- Exercise Echo is currently one of the more reliable methods for the differentiation.