



The Athlete's Heart: Part 1

Jimmy McKinney, MD., FRCP(C)

Sept 16th 2015

Outline



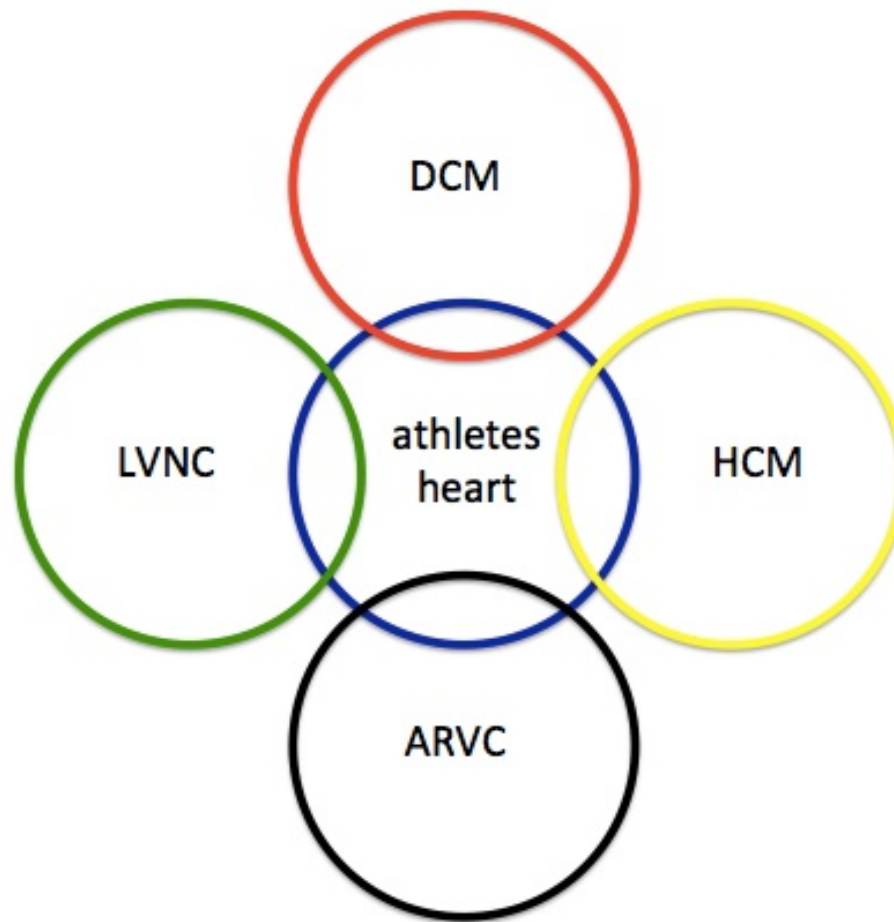
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- Brief history of the Athlete's Heart
- Physiology and mechanisms
- Defining 'normal'
- HCM vs. Athletes Heart

Outline



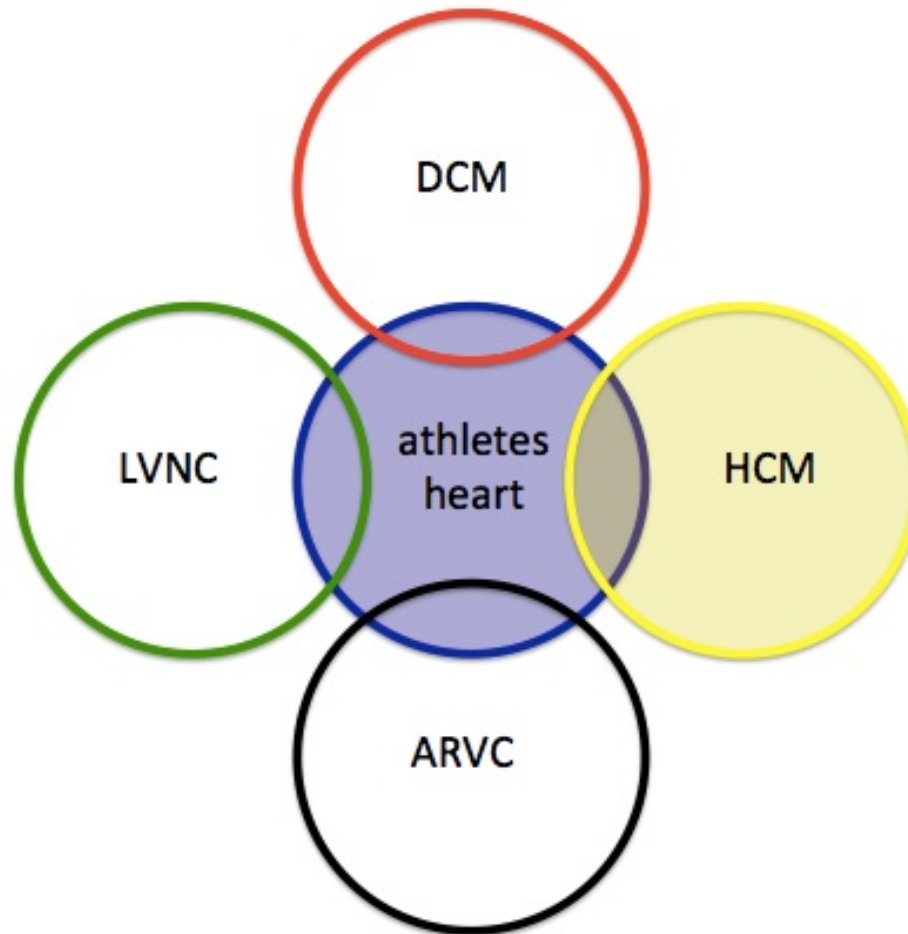
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Outline



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Sorry... Athlete's Heart vs. ARVC to be discussed later on

Canadian Journal of Cardiology 31 (2015) 502–508

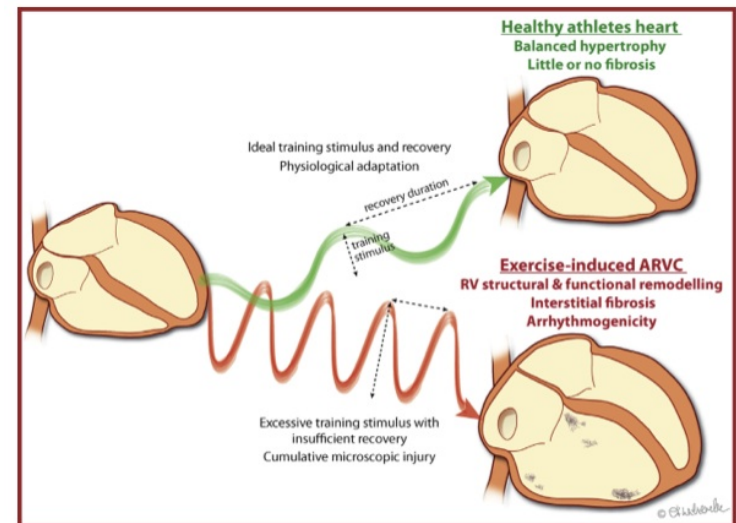
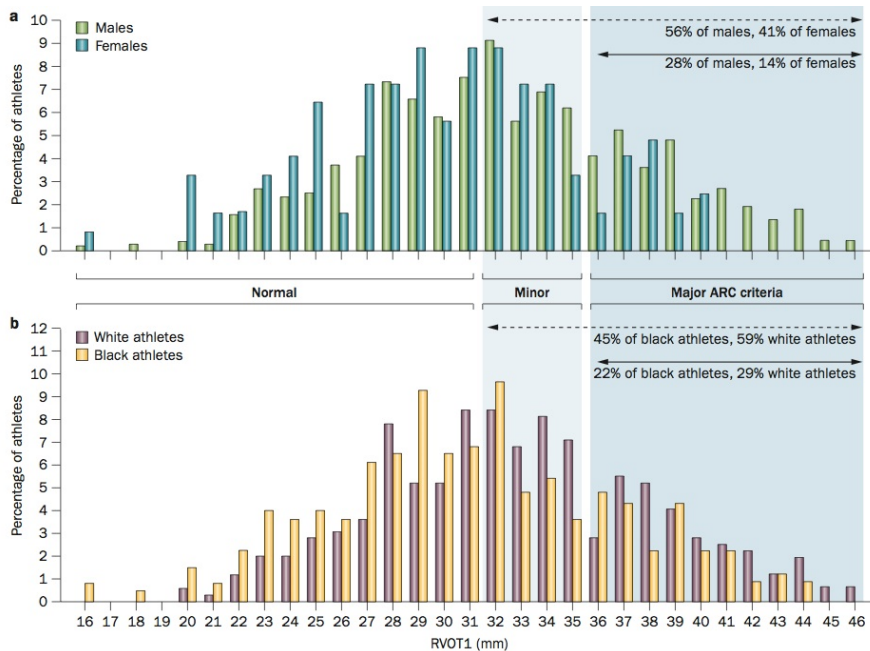


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Review

Is Exercise Good for the Right Ventricle? Concepts for Health and Disease

André La Gerche, MD, PhD,^{a,b} and Guido Claessen, MD^b



RV wall stress increases more than LV wall stress during exercise

SCBC tentative rounds schedule

<u>Topic</u>
The athletes heart PART 1
Journal Club - Afib
The athletes ECG
Journal Club - athletic HCM
SCBC research review
Too much of a good thing (part 1)
Too much of a good thing (part 2)
Journal Club
Screening - Young Athlete
SCBC research review
Screening - Masters Athlete
Journal Club
The RV ventricle
ARVC + exercise
Journal Club
Psychology of screening / disqualification
Journal Club
Marathon runners
SCBC research review
Sport eligibility review - Bethesda and European Guidelines
Journal Club
Biochemical changes of chronic exercise
Journal Club
Performance Enhancing Drugs
Cardiac risk factors in masters athletes
Journal Club
Dose of exercise / Benefits of exercise



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Athletes Heart



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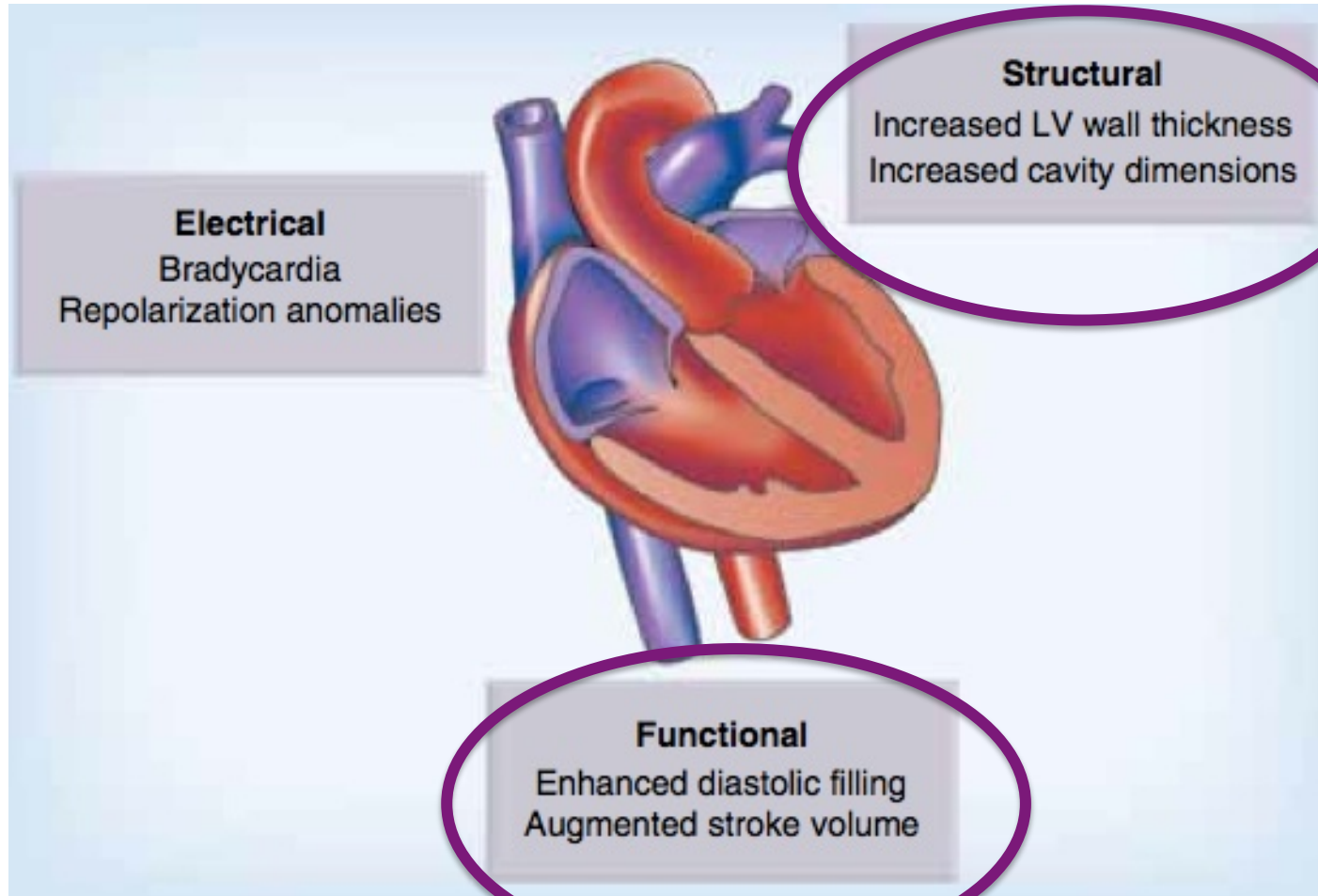
- First described in **1899** by Henschen that athletes have larger hearts than sedentary adults
 - He used the physical examination skills of auscultation and percussion to demonstrate increased cardiac dimensions in elite Nordic skiers
 - Recognized lower resting HR in athletes
- Eugene Darling of Harvard University made similar observations during the same year in university rowers



The 'Athletes Heart'



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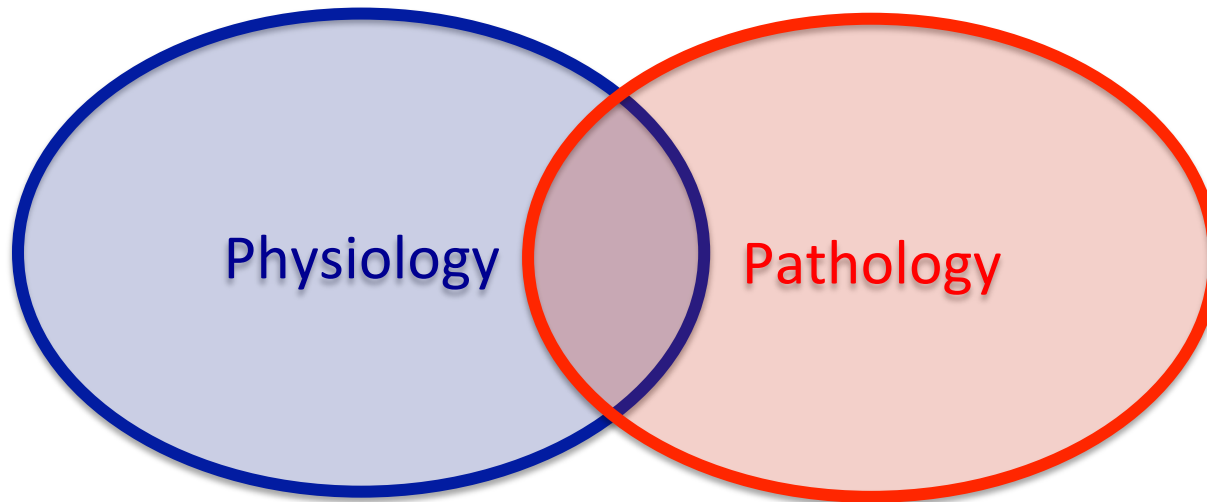


The term given to the complex of structural, functional, and electrical remodeling that accompanies regular athletic training

The Goal



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to differentiate normal findings in an athlete from the presence of cardiac pathology

A real case from SCBC



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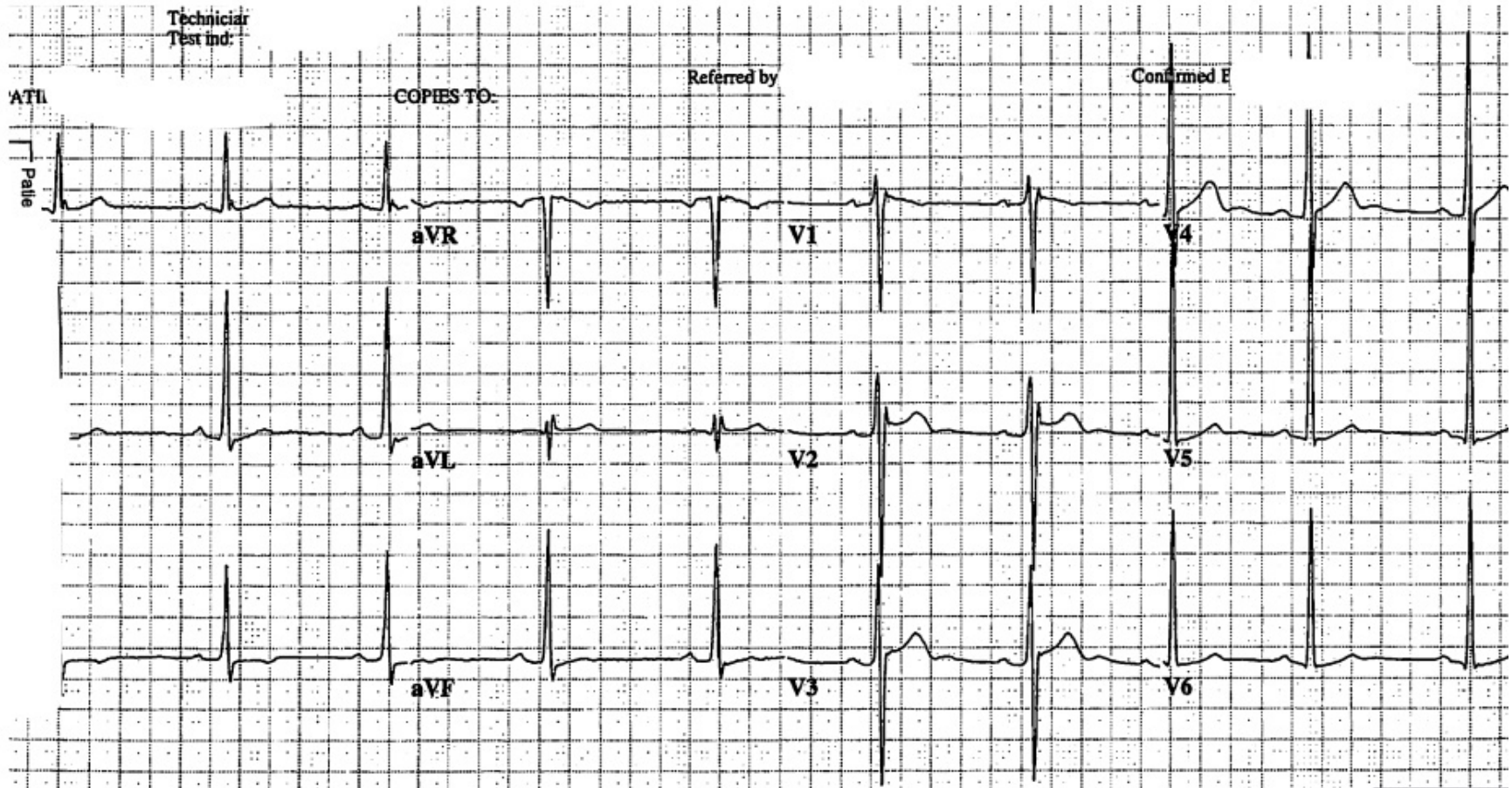
- 17M Rugby player (Provincial and Canada-West team)
- May 2014 he complained of palpitations with no syncope -> sent for ECG -> eventually saw a pediatric cardiologist in June 2015
- QUESTION: Athletes heart vs. HCM



June 17th 2015



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Holter – Aug 25th 2015



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Occasional SVE including 25 pairs and 12 runs 3 - 9 beats long and 69 - 80/min.
Rare PVCs.

Non-specific ST abnormality at higher rates. ✓
No symptoms reported.

*Sinus rhythm. Normal variability.
Rare ectopy. Normal Holter*

ECHO – June 17th 2015



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Study Date: 17-Jun-2015 02:37 PM

BSA: 2.0 m²

Height: 182 cm

Weight: 83 kg

Reason For Study: Palpitations/Tachycardia

Interpretation Summary

MMode/2D Measurements & Calculations

IVS/LVPW: 0.87

LA/Ao: 1.5

FS: 36.4 %

Doppler Measurements & Calculations

MV E max vel: 84.6 cm/sec

MV A max vel: 64.8 cm/sec

MV E/A: 1.3

Z-Score Result

Measurement Name	Value	Z-Score	Normal Range
LVIDd	5.3 cm	0.10	4.4 - 6.0
IVSd	1.2 cm	3.1	0.62 - 1.11
LVIDs	3.3 cm	0.24	2.5 - 4.0
IVSs	1.5 cm	1.3	0.72 - 1.74
LVPWd	1.4 cm	3.5	0.52 - 1.18
LVPWs	2.2 cm	4.5	1.1 - 1.8
LA dimension	4.4 cm	3.6	2.3 - 3.8
Ao root diam	3.0 cm	0.36	2.1 - 3.5

ECHO – June 17th 2015



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Doppler Measurements & Calculations

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Basic Physiology



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- All forms of exercise require an increase in skeletal muscle work
- The cardiovascular system is responsible for transporting oxygen-rich blood from the lungs to the skeletal muscles
- Exercise-induced cardiac remodeling enhances the cardiovascular system's ability to meet the demands of exercising skeletal muscle
- $CO = HR \times SV$ can increase 5-6x during max exercise

The Morganroth Hypothesis

Comparative Left Ventricular Dimensions in Trained Athletes

JOEL MORGANROTH, M.D., BARRY J. MARON, M.D., WALTER L. HENRY, M.D.,
and STEPHEN E. EPSTEIN, M.D., Bethesda, Maryland

Annals of Internal Medicine 82:521-524, 1975

- Hypothesized that morphological adaptations in athletes correspond with the type of hemodynamic overload imposed on the heart during exercise



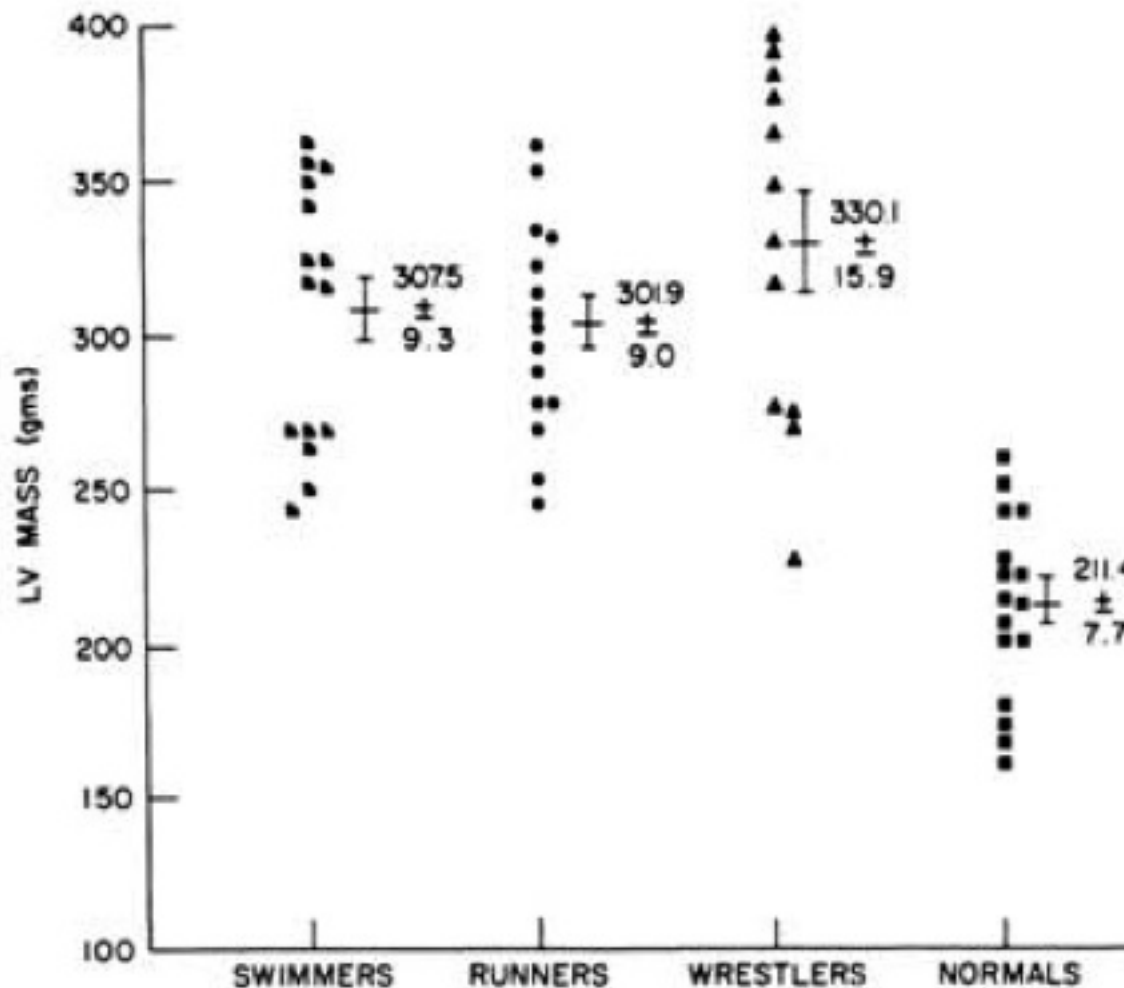
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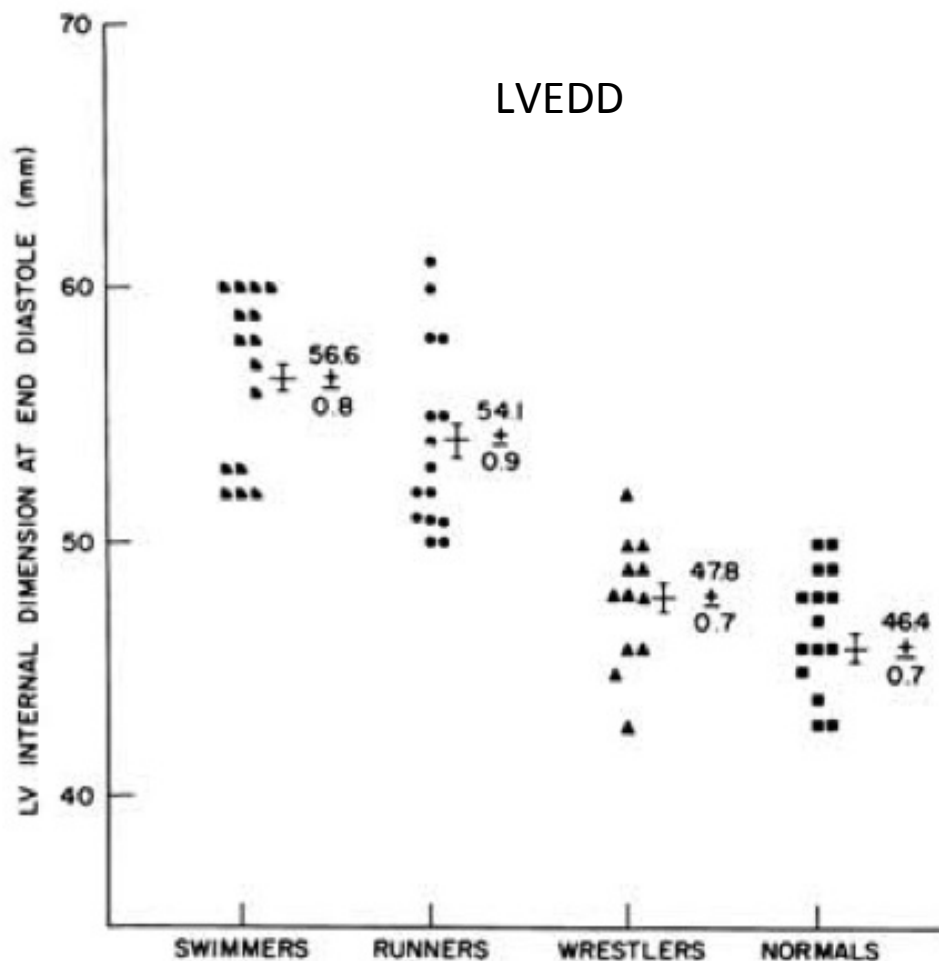
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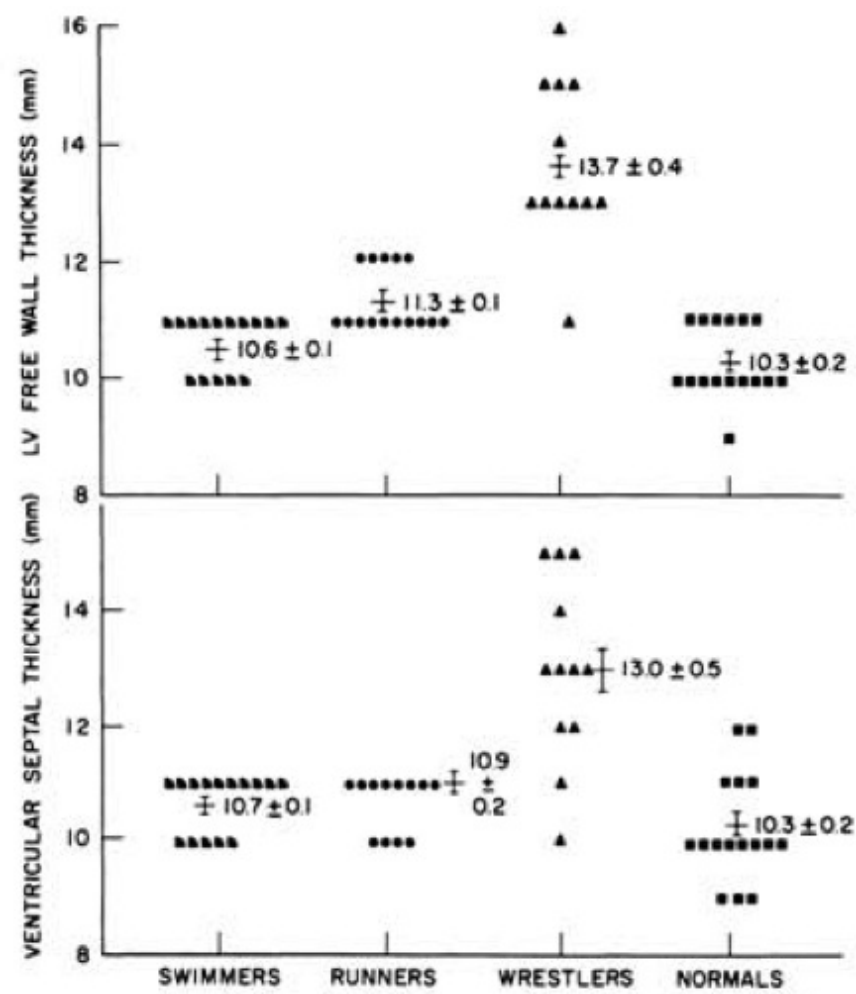
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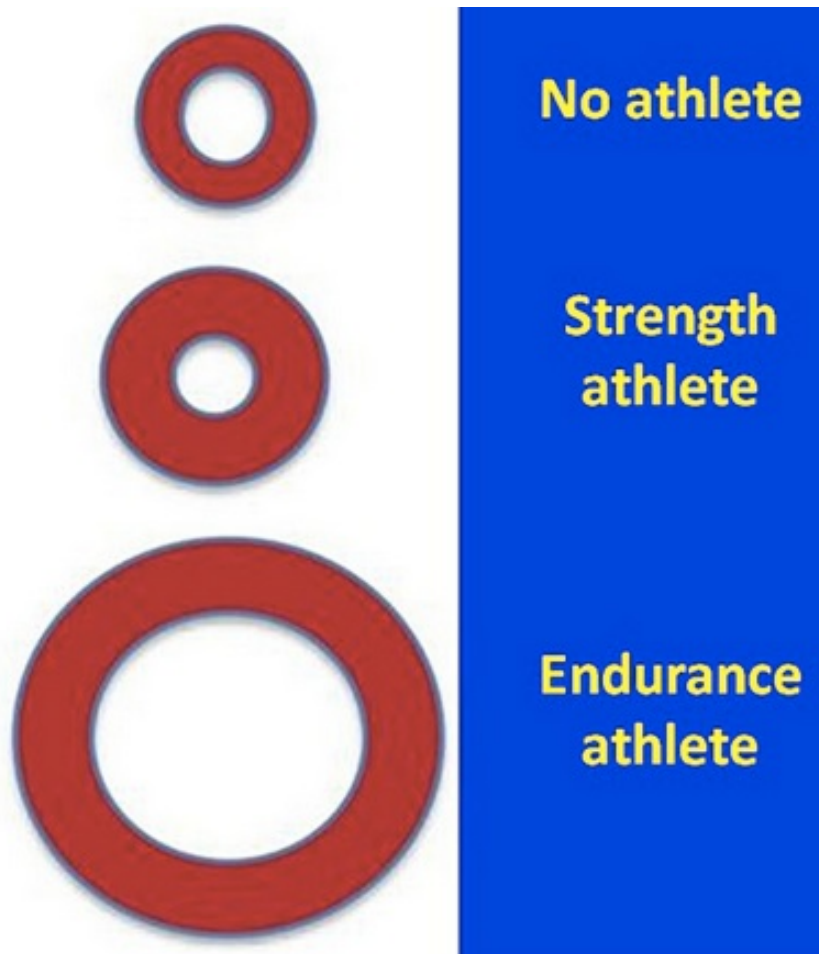


‘Morganroth hypothesis’

- states that:
 - Endurance sports, such as cycling and long- distance running, predominately place a volume load on the heart and lead to ‘eccentric hypertrophy’, characterized by an increase in LV mass, LV wall thickness, and LVEDV.
 - Sports involving strength training, such as wrestling, predominately place a pressure load on the heart, resulting in ‘concentric hypertrophy’, characterized by an increase in LV mass and wall thickness, but a normal LVEDV



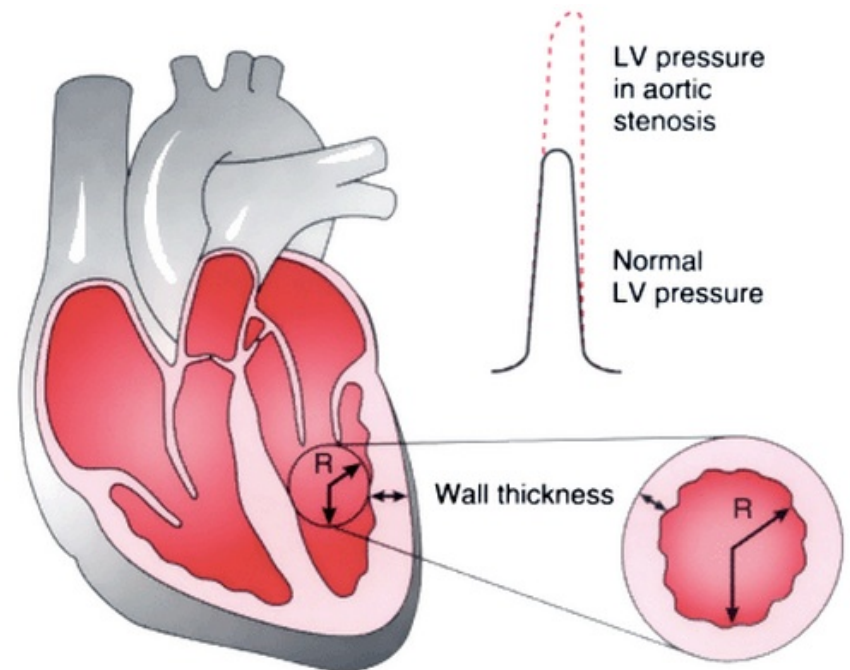
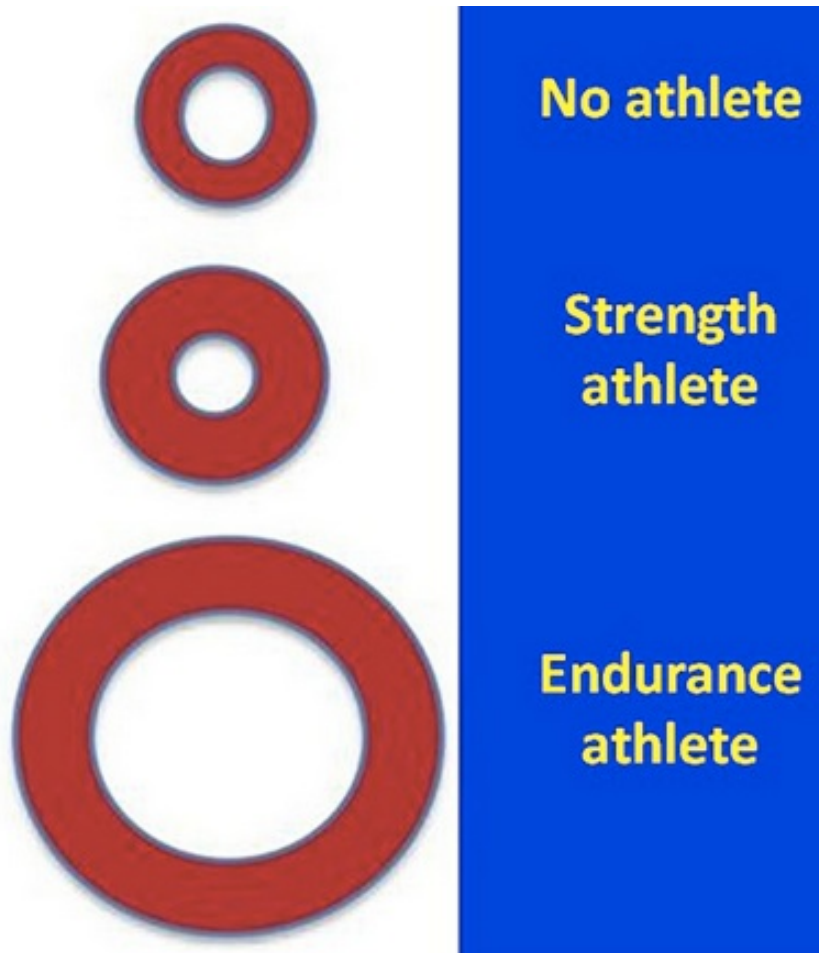
The Morganroth Hypothesis



- Hypothesized that morphological adaptations in athletes correspond with the type of hemodynamic overload imposed on the heart during exercise



The Morganroth Hypothesis



Laplace Law

$$\text{Wall stress} = \frac{\text{pressure} \times \text{radius}}{2(\text{wall thickness})}$$



Isotonic/high dynamic/ Endurance Exercise



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- **Sustained** elevations in cardiac output with normal or reduced peripheral vascular resistance
- Results in a “volume” challenge for the heart that affects all 4 chambers



Isometric exercise/High Static/ Strength training



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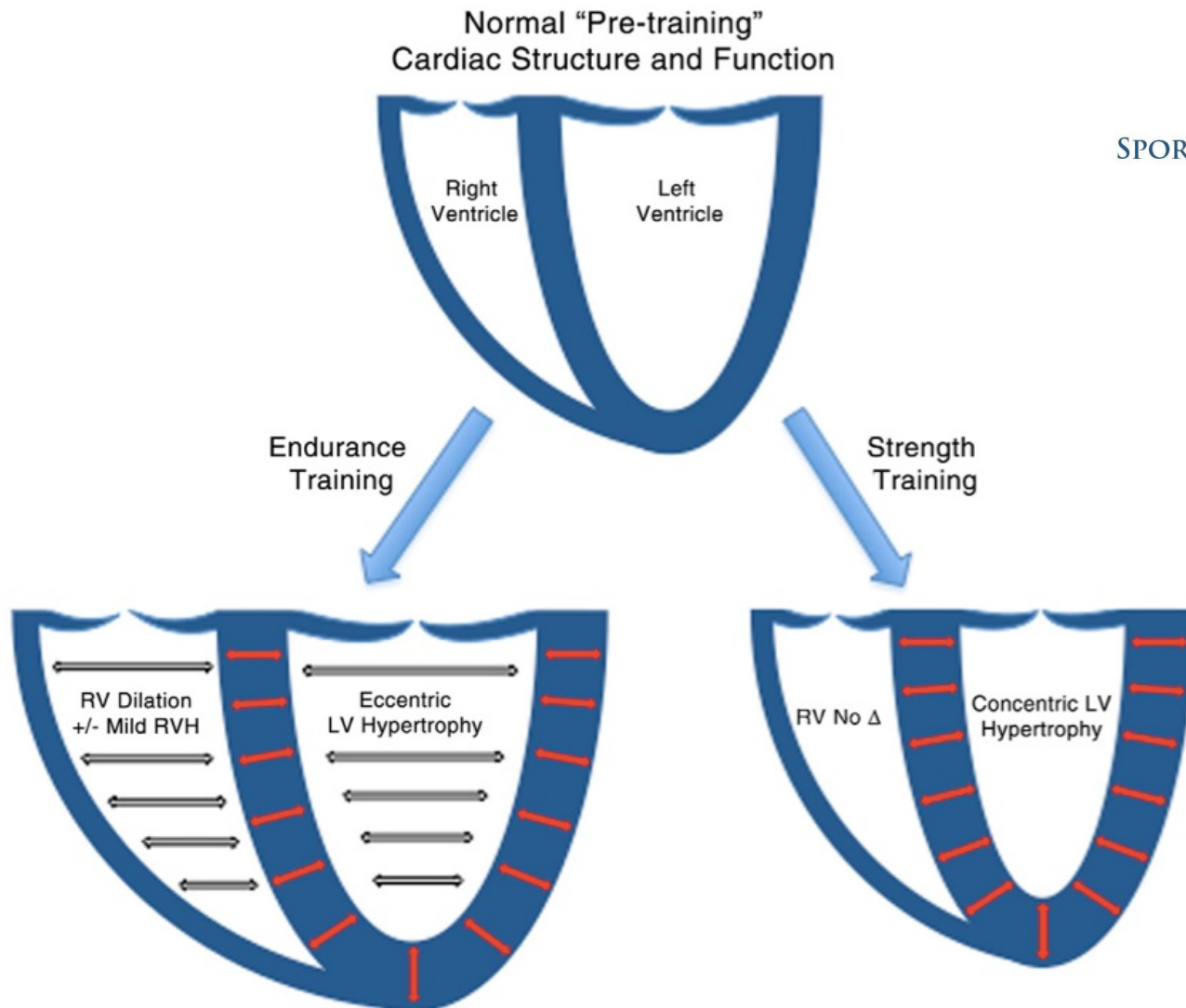
- Is characterized by **short** but **intense** bouts of increased **peripheral vascular resistance** and normal or only slightly elevated cardiac output
- Increase in peripheral vascular resistance causes transient but potentially marked systolic hypertension and LV “pressure” challenge.



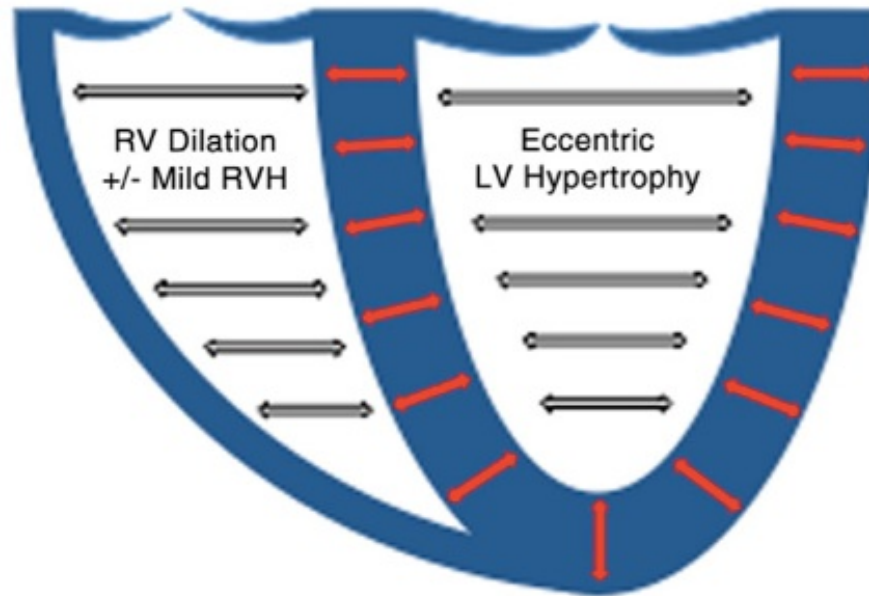
The Morganroth Hypothesis



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Isometric/Strength training



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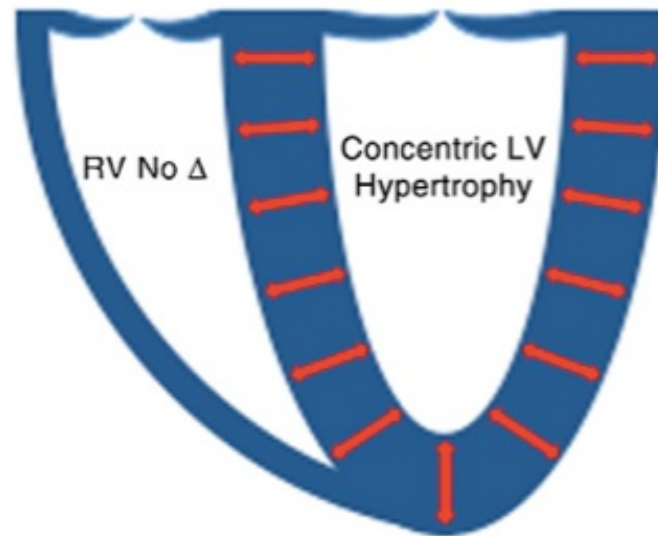
Characteristic Adaptations

- Mild to Moderate Eccentric LVH and RV dilation
- Biatrial enlargement
- Normal to slightly reduced resting LVEF
- Normal or enhanced Early LV Diastolic Function
- Normal or enhanced LV twisting / untwisting

Isotonic/Endurance training



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Characteristic Adaptations

- Mild concentric LVH but No RV remodeling
- Normal to mildly enlarged left atrial size
- Normal to hyperdynamic resting LVEF
- Normal to slightly reduced early LV diastolic function
- Compensatory increase in late LV diastolic function

The Athlete's Heart

A Meta-Analysis of Cardiac Structure and Function

Babette M. Pluim, MD; Aeilko H. Zwinderman, PhD;
 Arnoud van der Laarse, PhD; Ernst E. van der Wall, MD, PhD

TABLE 4. Cardiac Structure and Function in Endurance-Trained Athletes, Combined Endurance and Strength-Trained Athletes, Strength-Trained Athletes, and Control Subjects

	Endurance-Trained Athletes	Combined Endurance- and Strength-Trained Athletes	Strength-Trained Athletes	Control Subjects	<i>P</i> *
RWT	0.389 (0.374–0.404) (n=413)	0.398 (0.374–0.421) (n=494)	0.442 (0.403–0.480) (n=544)	0.356 (0.343–0.369) (n=813)	<0.001
LVIDd, mm	53.7 (52.8–54.6) (n=413)	56.2 (55.2–57.1) (n=494)	52.1 (50.6–53.6) (n=544)	49.6 (48.9–50.2) (n=813)	<0.001
PWTd, mm	10.3 (10.0–10.6) (n=413)	11.0 (10.3–11.6) (n=494)	11.0 (10.2–11.7) (n=544)	8.8 (8.5–9.0) (n=813)	<0.001
IVSTd, mm	10.5 (10.1–10.9) (n=413)	11.3 (10.6–12.0) (n=494)	11.8 (10.9–12.7) (n=544)	8.8 (8.6–9.1) (n=813)	<0.001
LVM, g	249 (233–264) (n=413)	288 (260–316) (n=494)	267 (234–300) (n=544)	174 (165–183) (n=813)	<0.001
LVEF, %	68.8 (65.1–72.6) (n=177)	66.1 (62.9–69.3) (n=127)	66.3 (60.7–71.9) (n=73)	67.2 (64.5–69.8) (n=296)	0.68
LVFS, %	34.4 (32.6–36.1) (n=204)	34.7 (32.7–36.8) (n=293)	35.7 (33.7–37.7) (n=276)	34.4 (33.5–35.2) (n=491)	0.50
E/A ratio	2.20 (1.49–2.91) (n=93)	1.89 (1.46–2.31) (n=126)	2.11 (1.22–2.99) (n=44)	1.84 (1.64–2.05) (n=132)	0.41

The Morganroth Hypothesis **revisited**

- The development of an endurance-trained heart (eccentric hypertrophy) and a strength-trained heart (concentric hypertrophy) should NOT be considered an absolute and dichotomous concept
- Endurance-trained athletes have **↑**LV RWT instead of a proportional increase in LVT and internal diameter with a normal relative wall thickness



Who has a thicker heart?



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1993 Absence of Left Ventricular Wall Thickening in Athletes Engaged in Intense Power Training

Antonio Pelliccia, MD, Antonio Spataro, MD, Giovanni Caselli, MD, and Barry J. Maron, MD

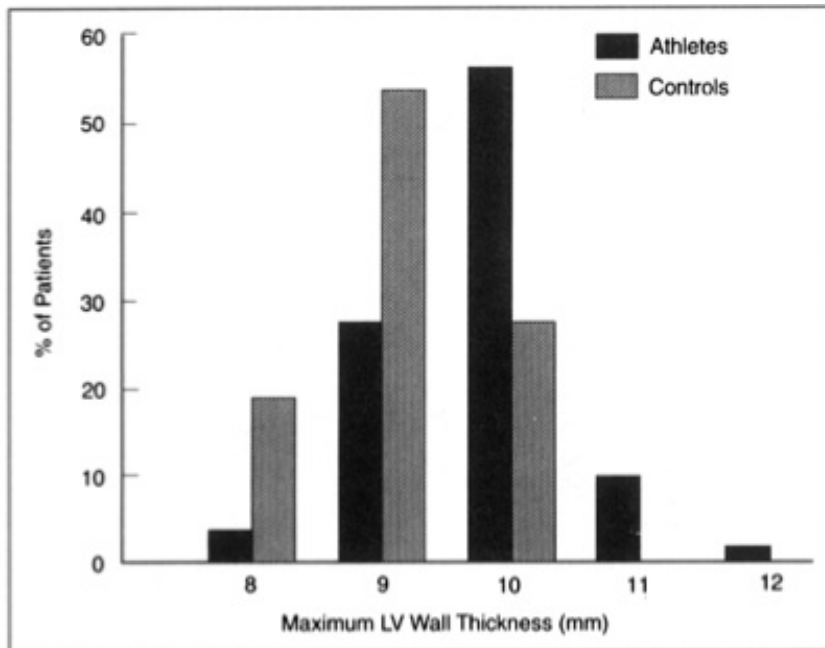


FIGURE 1. Distribution of left ventricular (LV) wall thicknesses in 100 power-trained athletes and 26 sedentary control subjects.

	Power	Controls
LVT (mm)	9.6	9.0
LVMI(g/m2)	96	82
LVEDD(mm)	55	54



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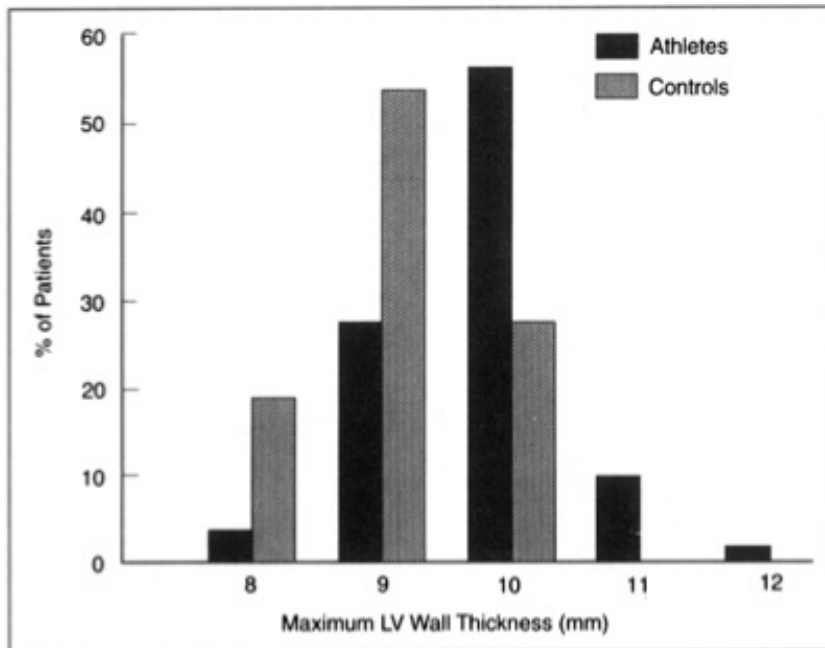


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LVMI(g/m2)	96	82
LVEDD(mm)	55	54

BOLD = statistically significant



Who has a larger LVEDD?



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Morphology of the “Athlete’s Heart” Assessed by Echocardiography in 947 Elite Athletes Representing 27 Sports*

Paolo Spirito, MD, Antonio Pelliccia, MD, Michael A. Proschan, PhD, Maristella Granata, MD, Antonio Spataro, MD, Pietro Bellone, MD, Giovanni Caselli, MD, Alessandro Biffi, MD, Carlo Vecchio, MD, and Barry J. Maron, MD

TABLE II Calculated Effects of Type of Sport on Left Ventricular Internal Diastolic Cavity Dimension (LVIDd) and Wall Thickness in 947 Athletes

Sport	Impact on LVIDd (mm)	Sport	Impact on Wall Thickness (mm)
1) Endurance cycling	5.91	Rowing	2.13
2) Cross-country skiing	5.41	Endurance cycling	2.02
3) Swimming	4.90	Swimming	1.71
4) Pentathlon	4.35	Canoeing	1.70
5) Canoeing	4.23	Long-distance track	1.49
6) Sprint cycling	3.97	Water polo	1.38
7) Rowing	3.87	Sprint cycling	1.35
8) Long-distance track	3.47	Weightlifting	1.23
9) Soccer	3.11	Wrestling/judo	1.21
10) Team handball	2.87	Tennis	1.00
11) Tennis	2.69	Pentathlon	0.98
12) Roller hockey	2.41	Cross-country skiing	0.98

1.7% of athletes had LVT>12mm

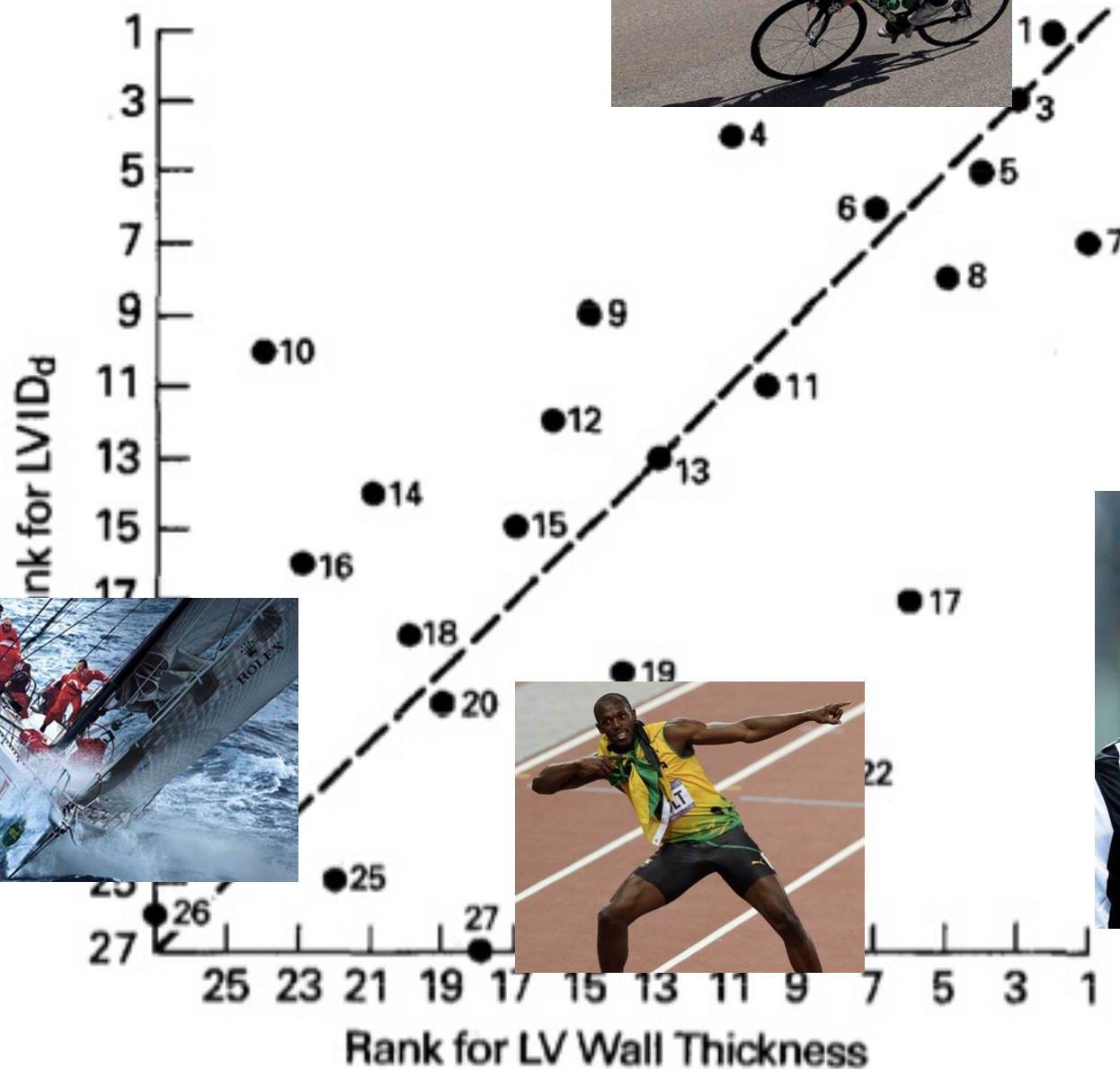
38% of athletes had LVEDD >54mm

13) Boxing	2.25	Boxing	0.94
14) Alpine skiing	2.13	Roller skating	0.88
15) Fencing	2.09	Soccer	0.76
16) Taekwondo	2.07	Roller hockey	0.69
17) Water polo	2.02	Fencing	0.63
18) Diving	1.70	Sprint track	0.54
19) Roller skating	1.68	Volleyball	0.39
20) Volleyball	1.43	Diving	0.38
21) Bobsledding	1.35	Alpine skiing	0.29
22) Weightlifting	1.32	Field weight events	0.25
23) Wrestling/judo	1.25	Taekwondo	0.23
24) Equestrian	0.43	Team handball	0.19
25) Field weight events	0.18	Equestrian	0.13
26) Yachting	0.10	Bobsledding	0.07
27) Sprint track	0.00	Yachting	0.00

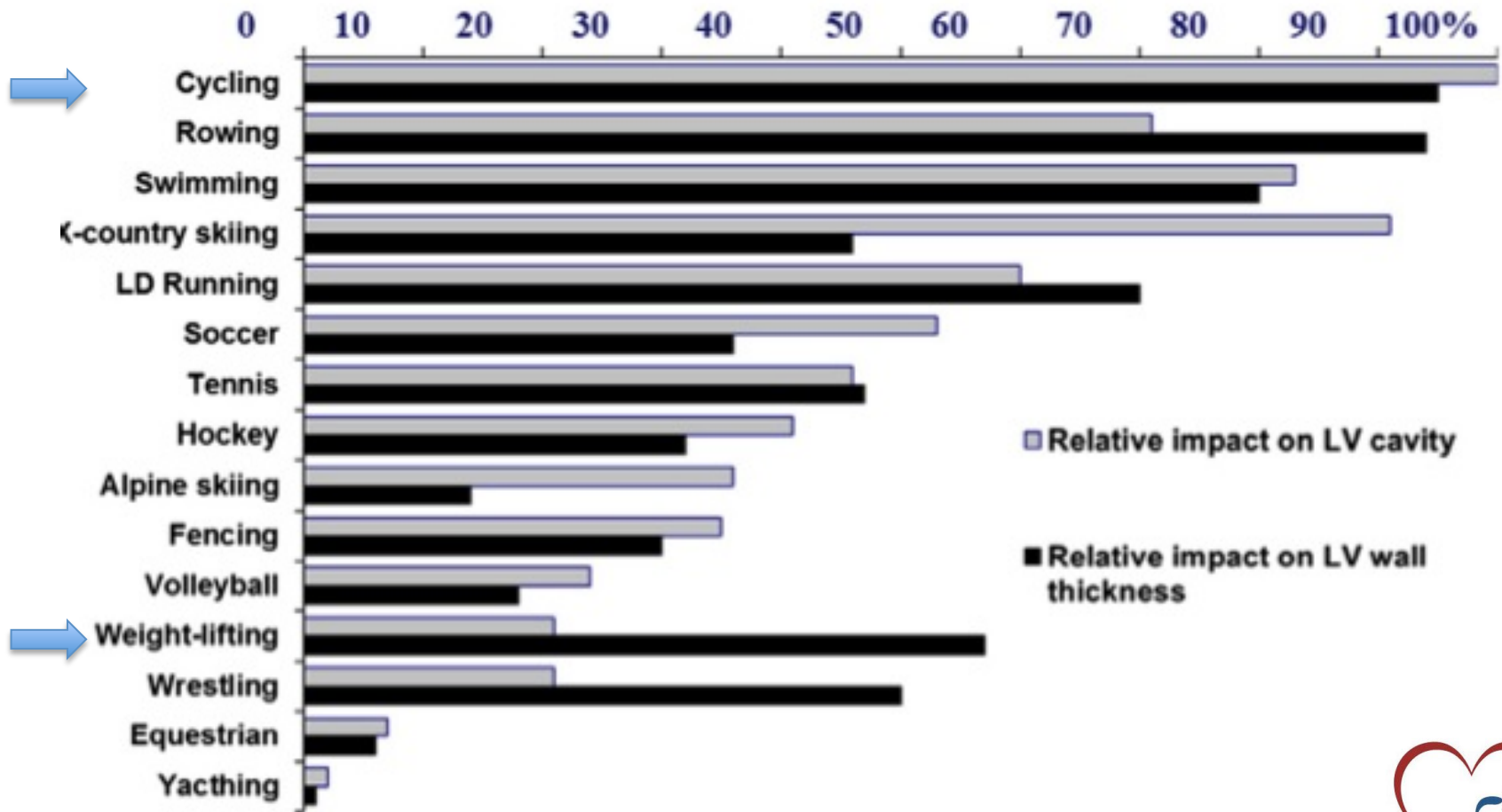




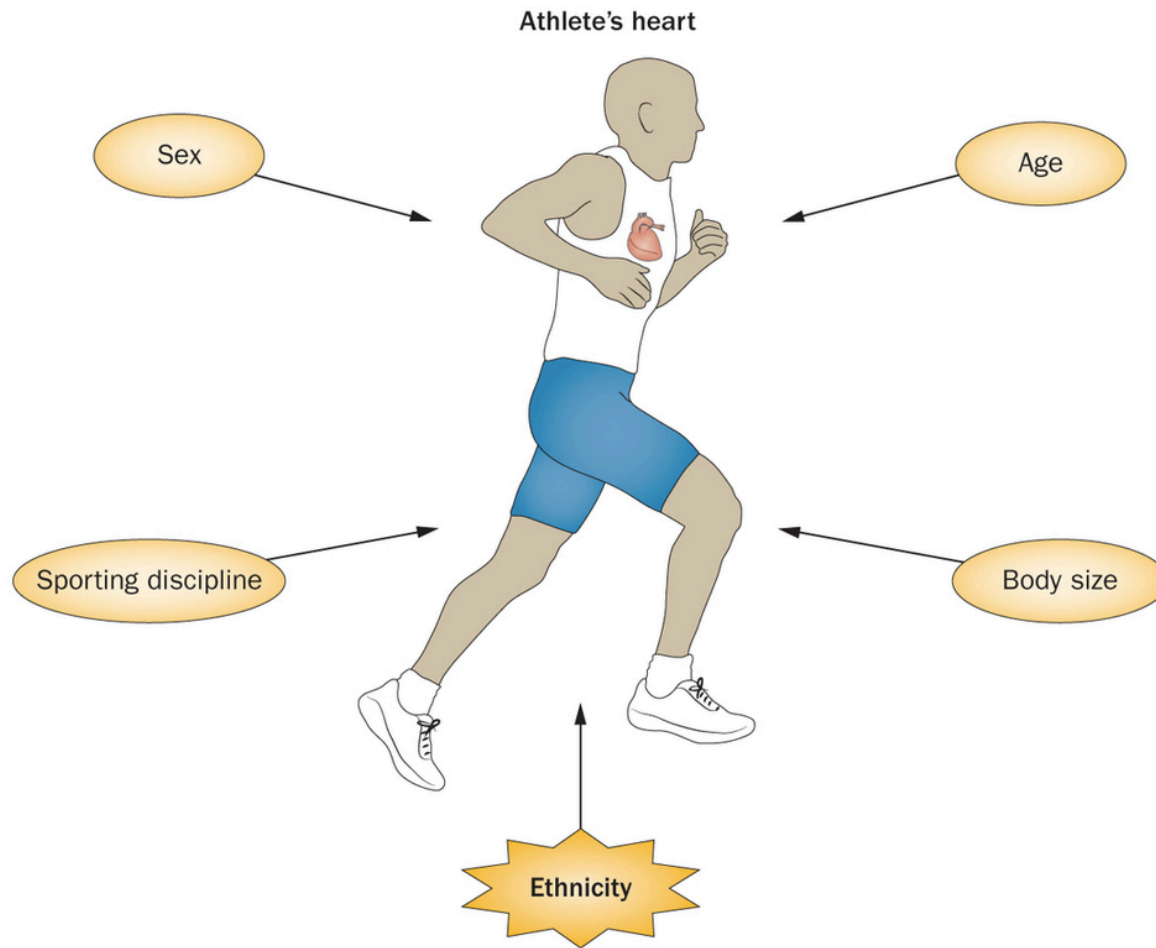
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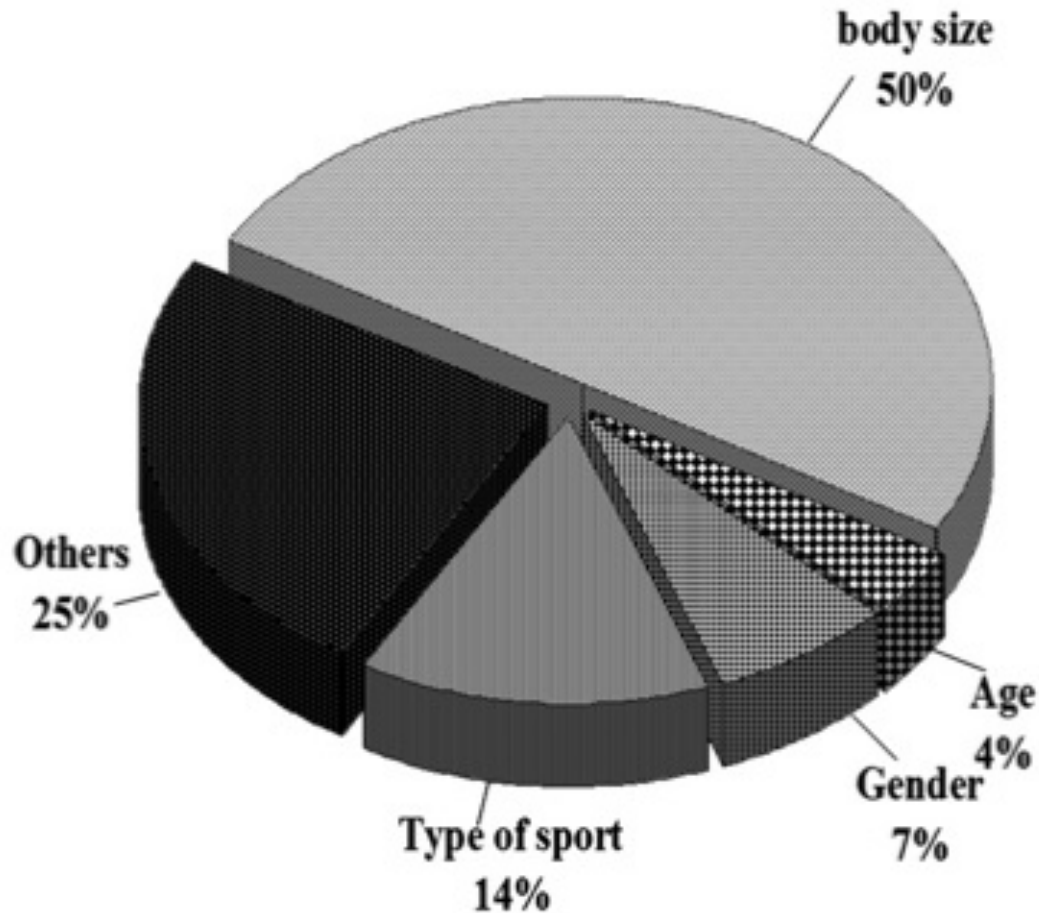
Effect of sport on LVEDD and LVT



Factors influencing the athlete's heart



Impact of different variables on LV end-diastolic cavity dimensions



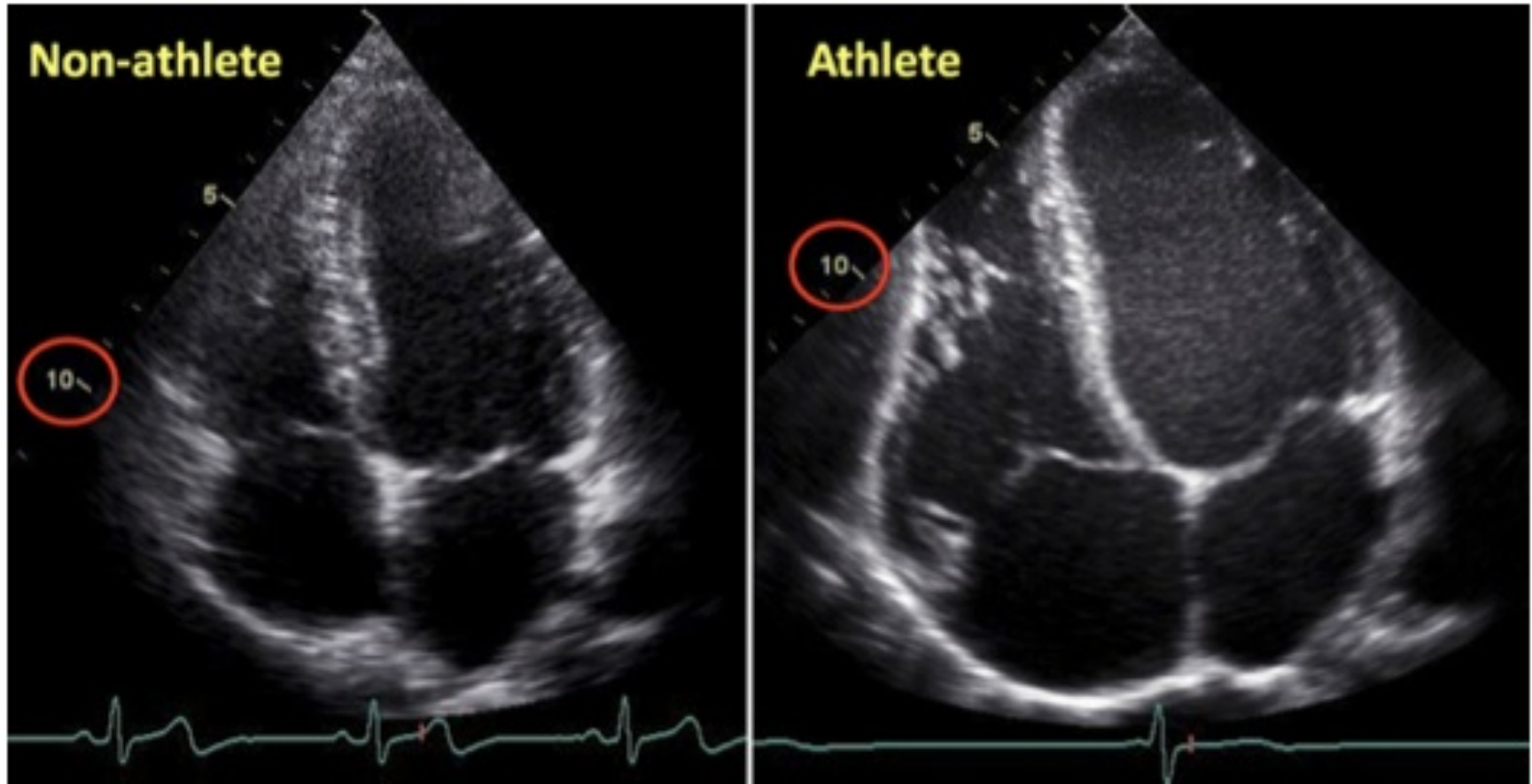
Athletes have bigger hearts





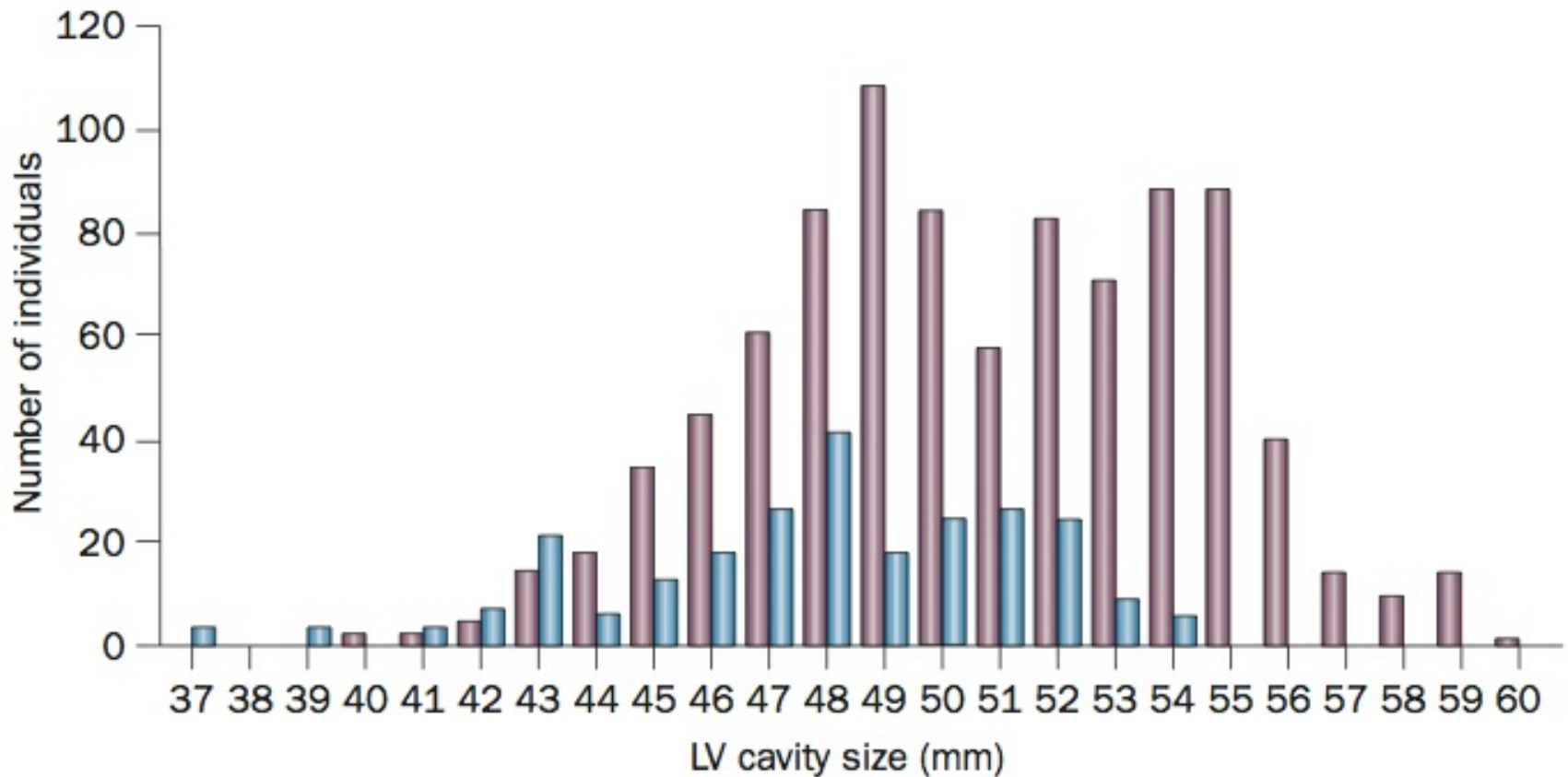
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Athletes have bigger hearts



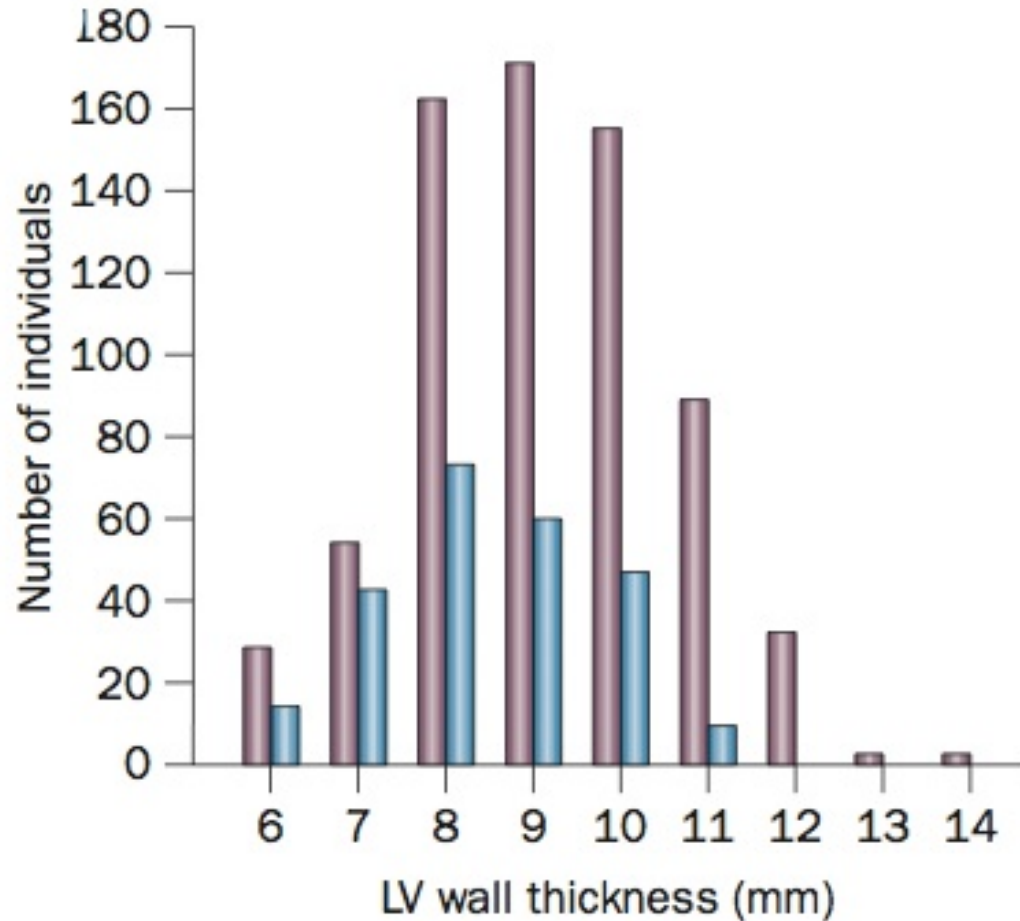


Athletes have bigger hearts





Athletes have bigger hearts



Sharma, JACC. 2002.



Athletes have bigger hearts

Table 2 | Mean absolute ventricular dimensions in athletes and nonathletes*

Echocardiographic variable	Athletes	Nonathletes	% Difference
Septal thickness (mm)	10.4	9.1	14.3
Posterior wall thickness (mm)	10.7	9.0	18.9
LV end-diastolic cavity (mm)	53.9	49.1	9.8
LV mass (gm^{-2})	256.0	175.0	46.3
RV end-diastolic cavity (mm)	22.0	17.7	24.3

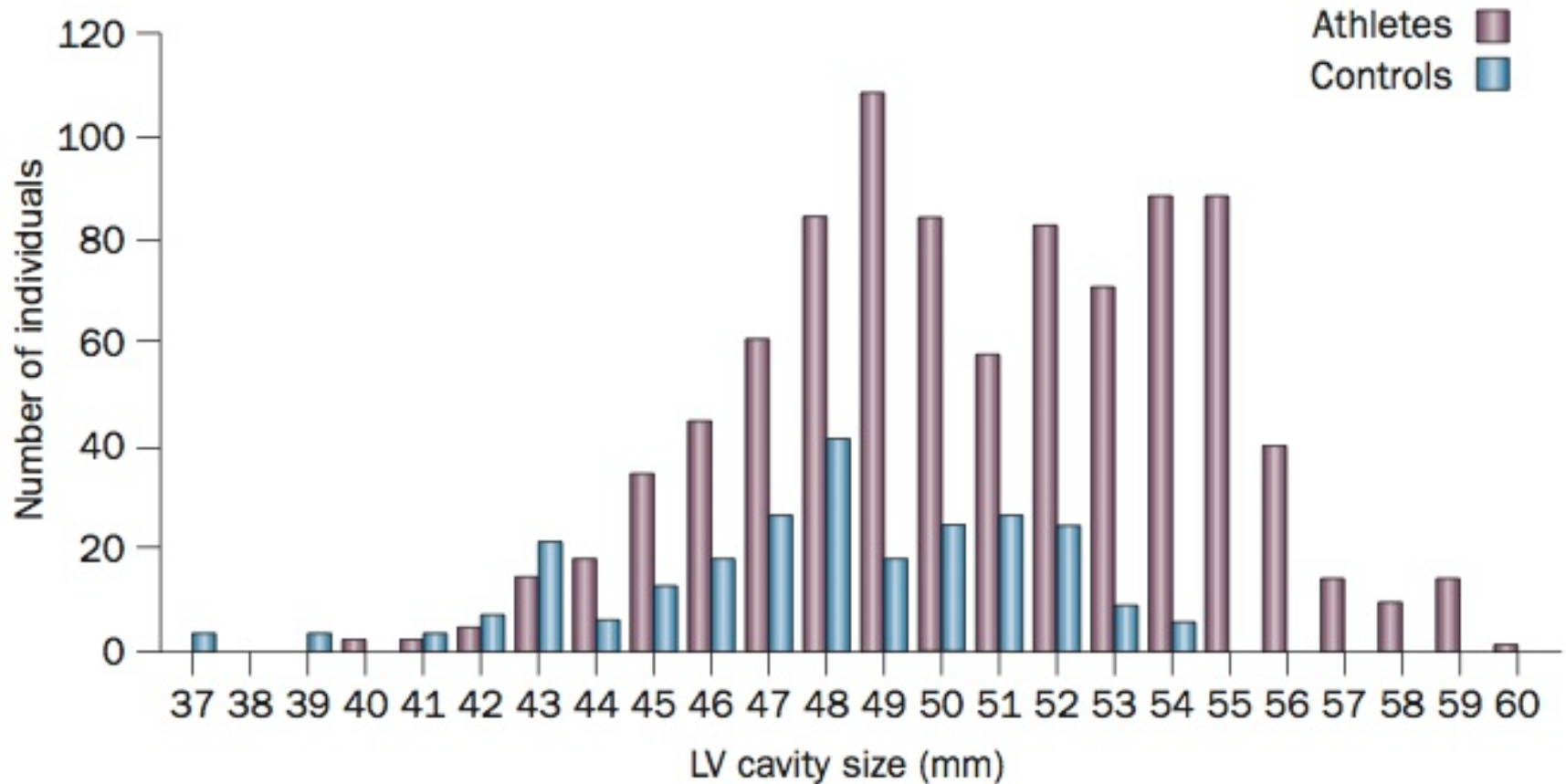
How big is too big? Wall thickness

- 25% of Caucasian non-athletes demonstrated values for LVWT exceeding reference
- >12mm was extremely uncommon (2%) and confined to male athletes
- None exhibited a LVWT >16mm



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LV end diastolic dimension

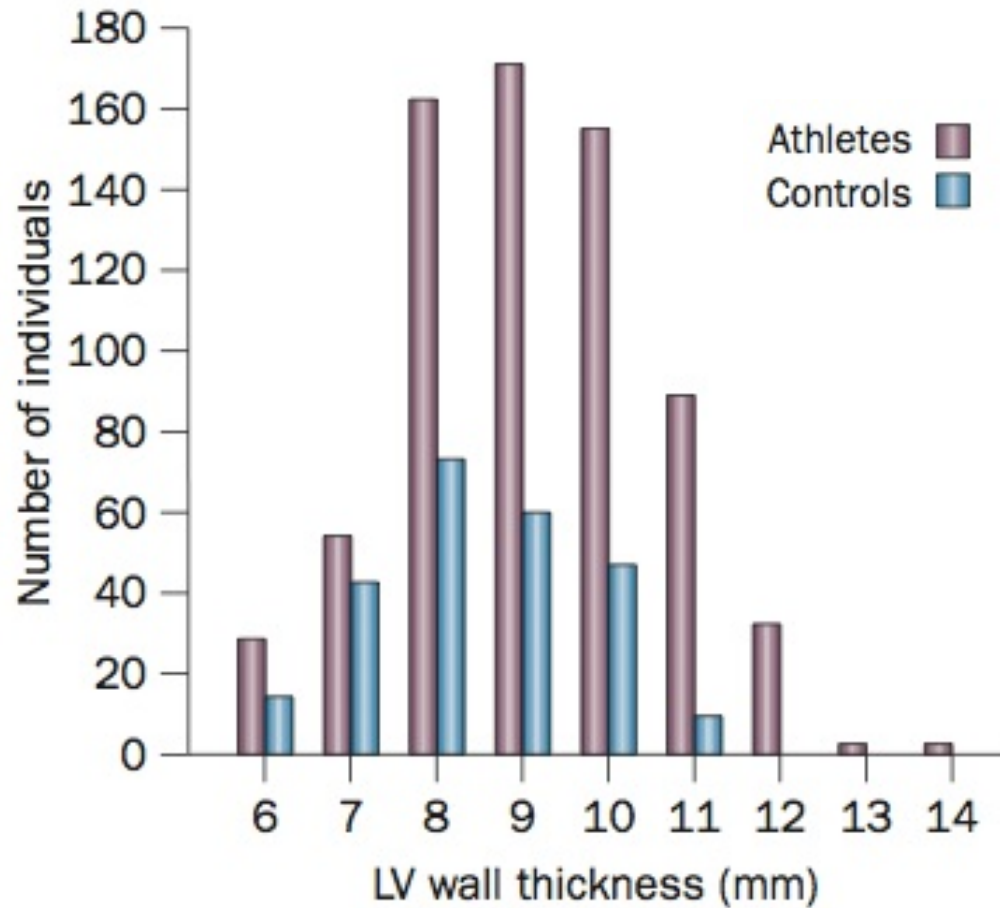


Makan, Heart. 2005.

LV wall thickness



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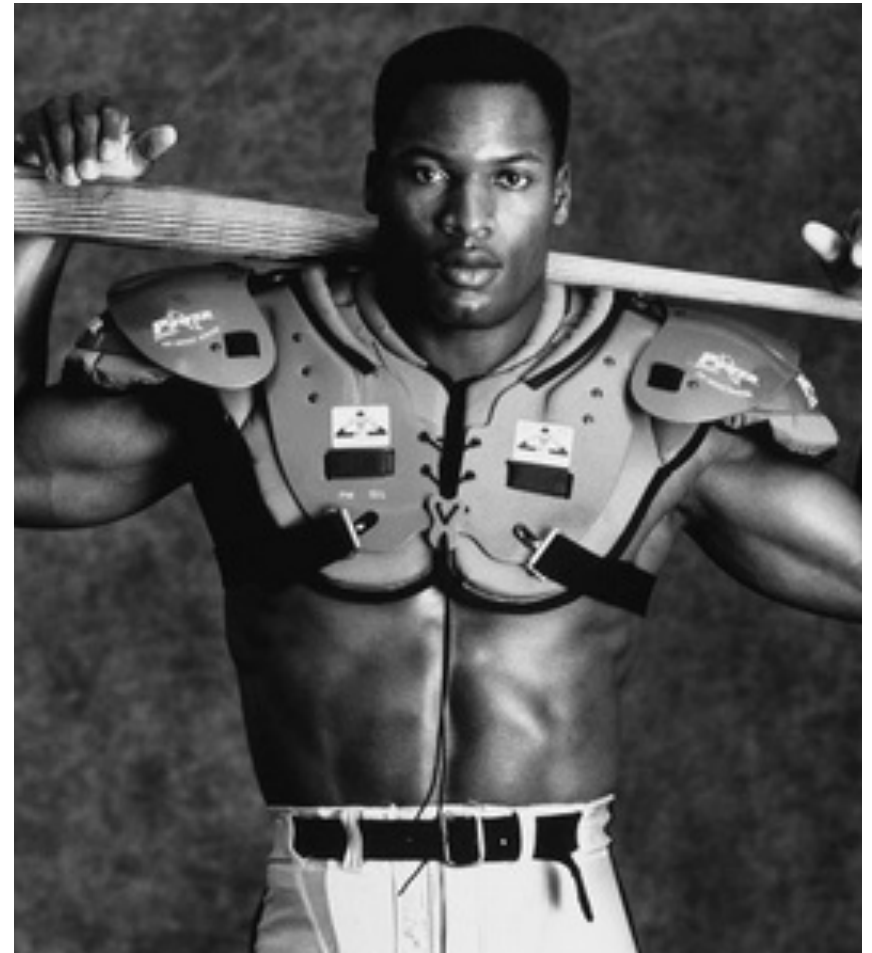


Sharma, JACC. 2002.

Black and White

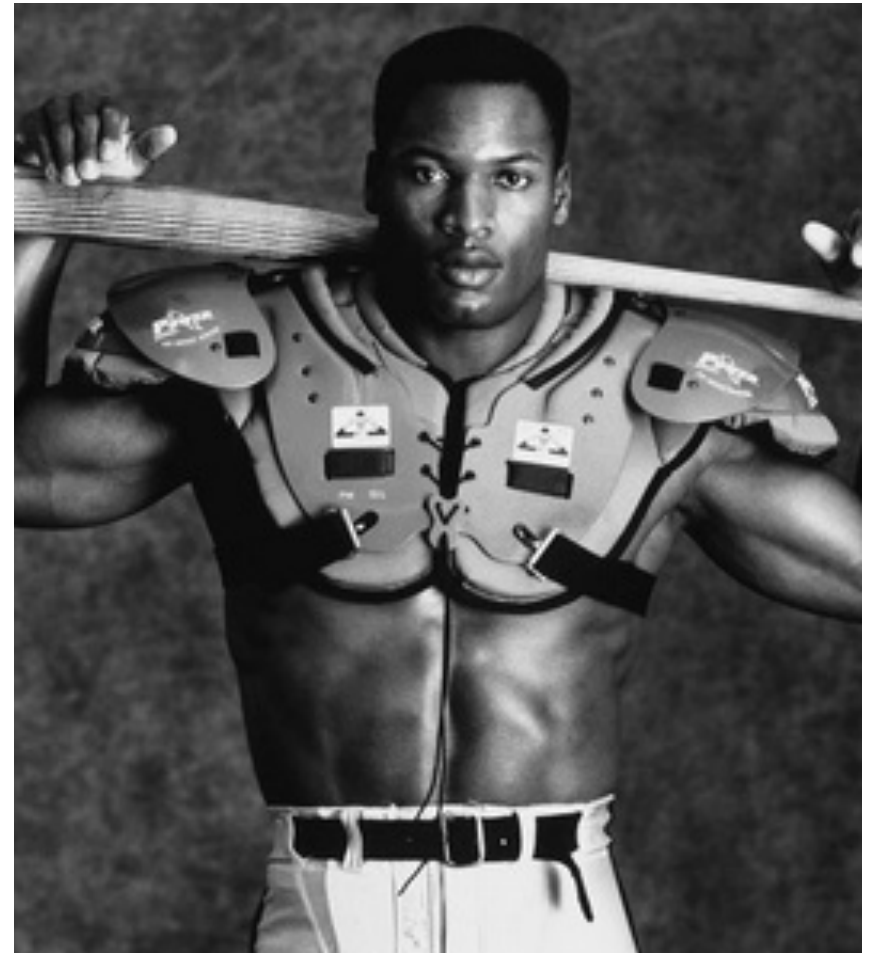


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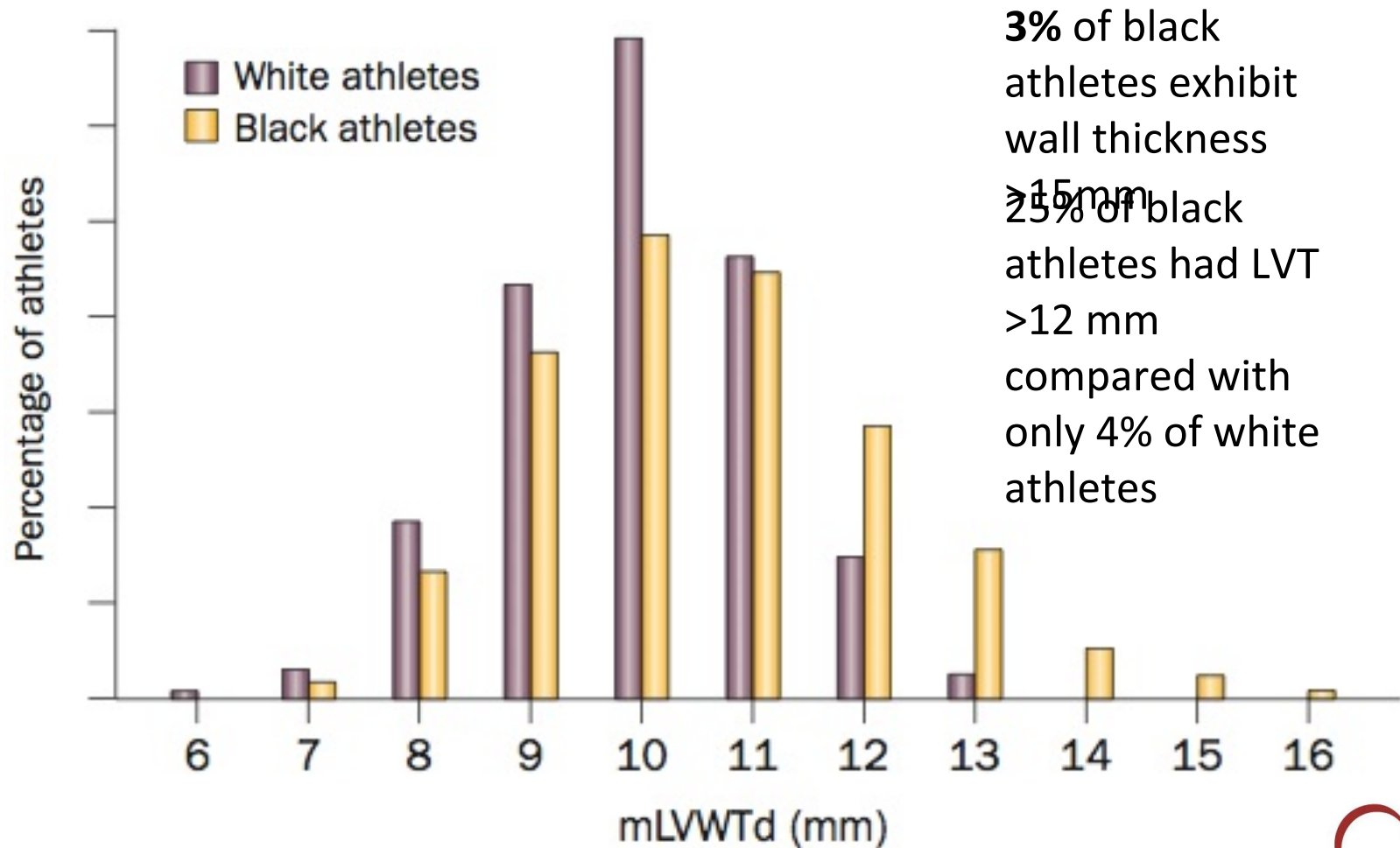




Who has the bigger heart?



Black vs. White (male) LV wall thickness

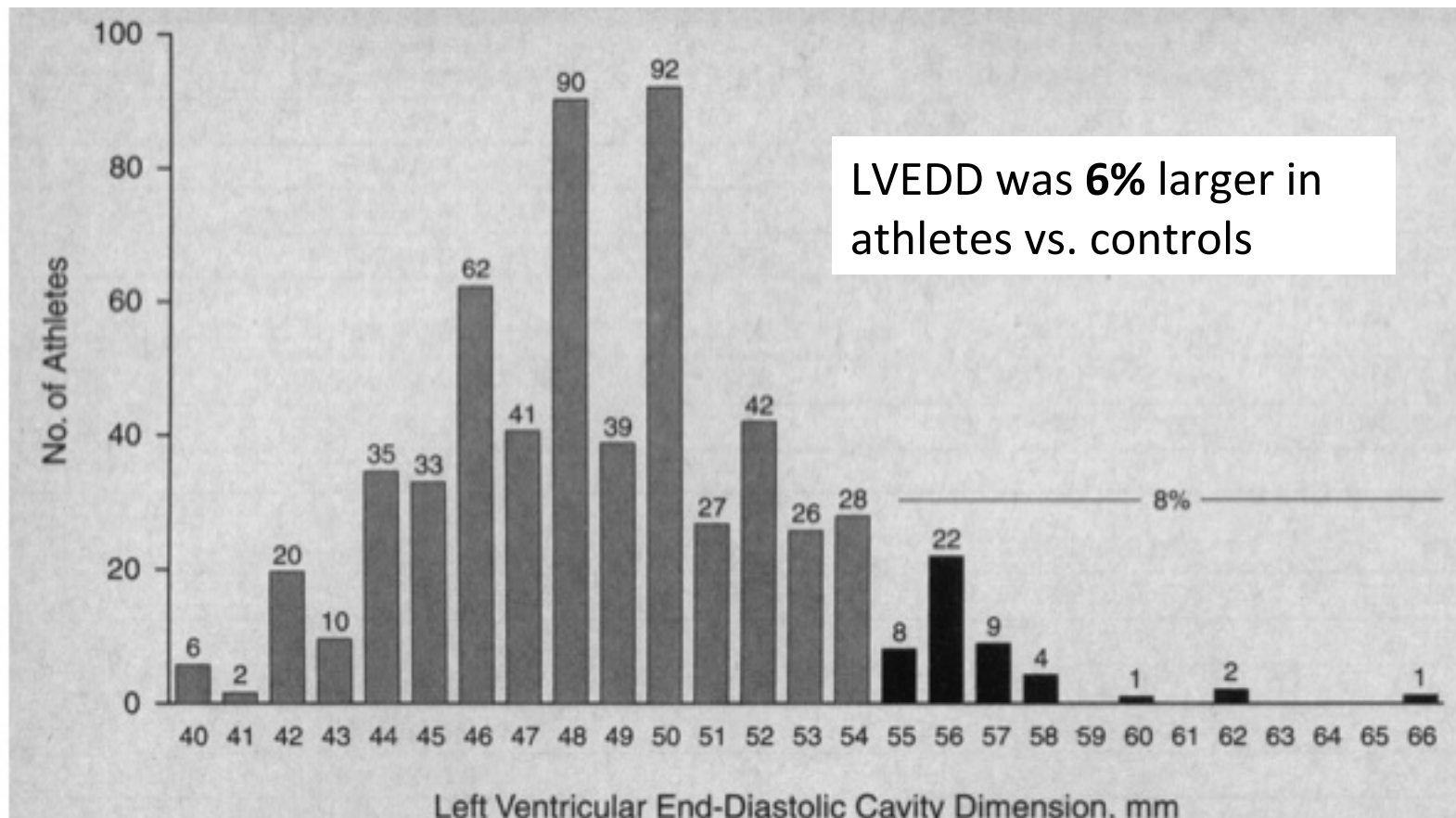




Athlete's Heart in Women

Echocardiographic Characterization of Highly Trained Elite Female Athletes

Antonio Pelliccia, MD; Barry J. Maron, MD; Franco Culasso, PhD; Antonio Spataro, MD; Giovanni Caselli, MD

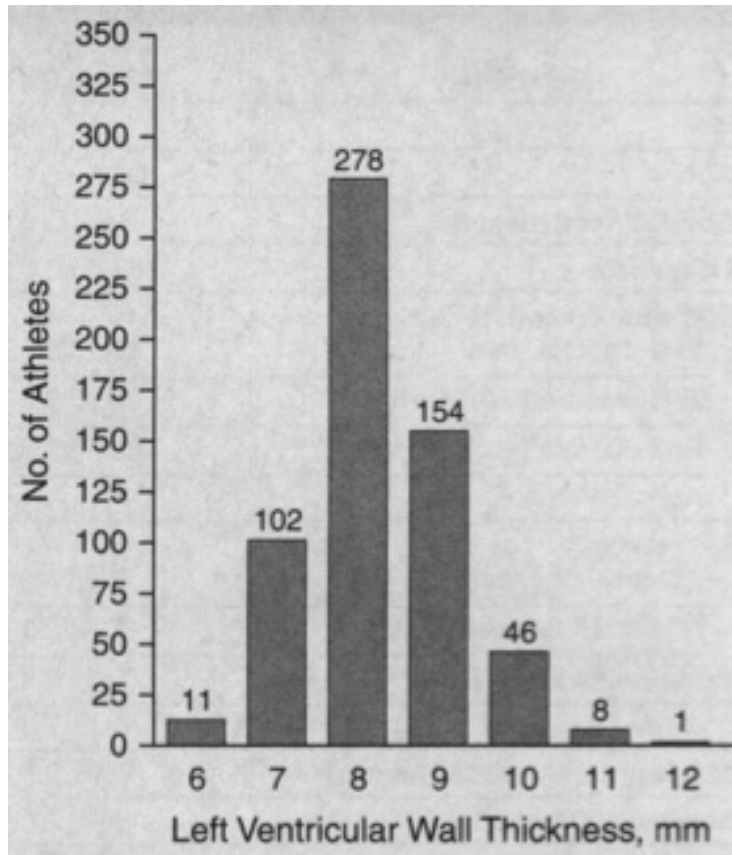


Pelliccia, JAMA 1996

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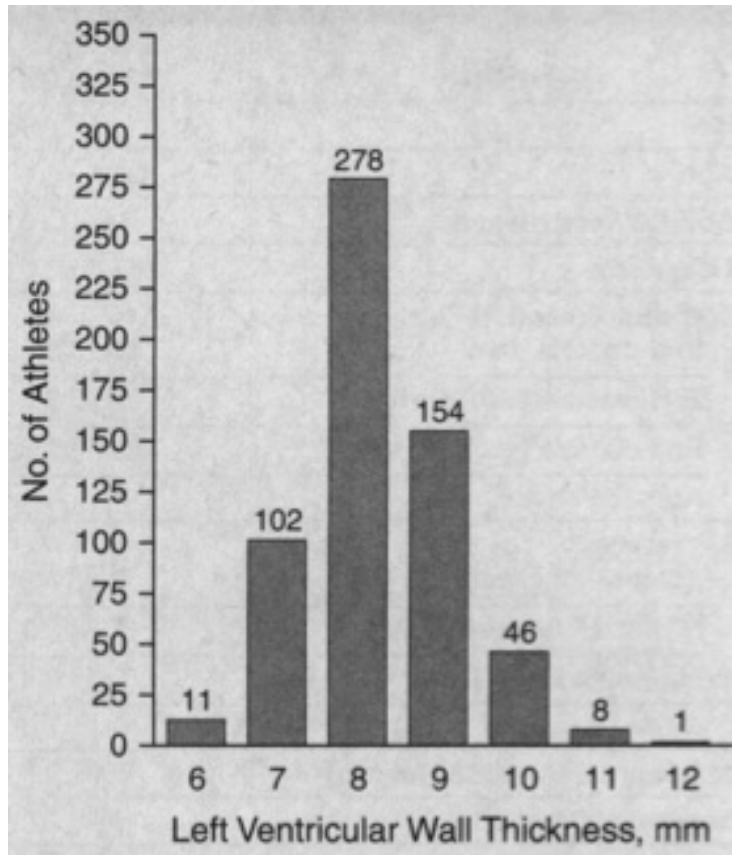
LVT was **14%** larger in athletes vs. controls

 **0** women athletes >13mm

Athlete's Heart in Women

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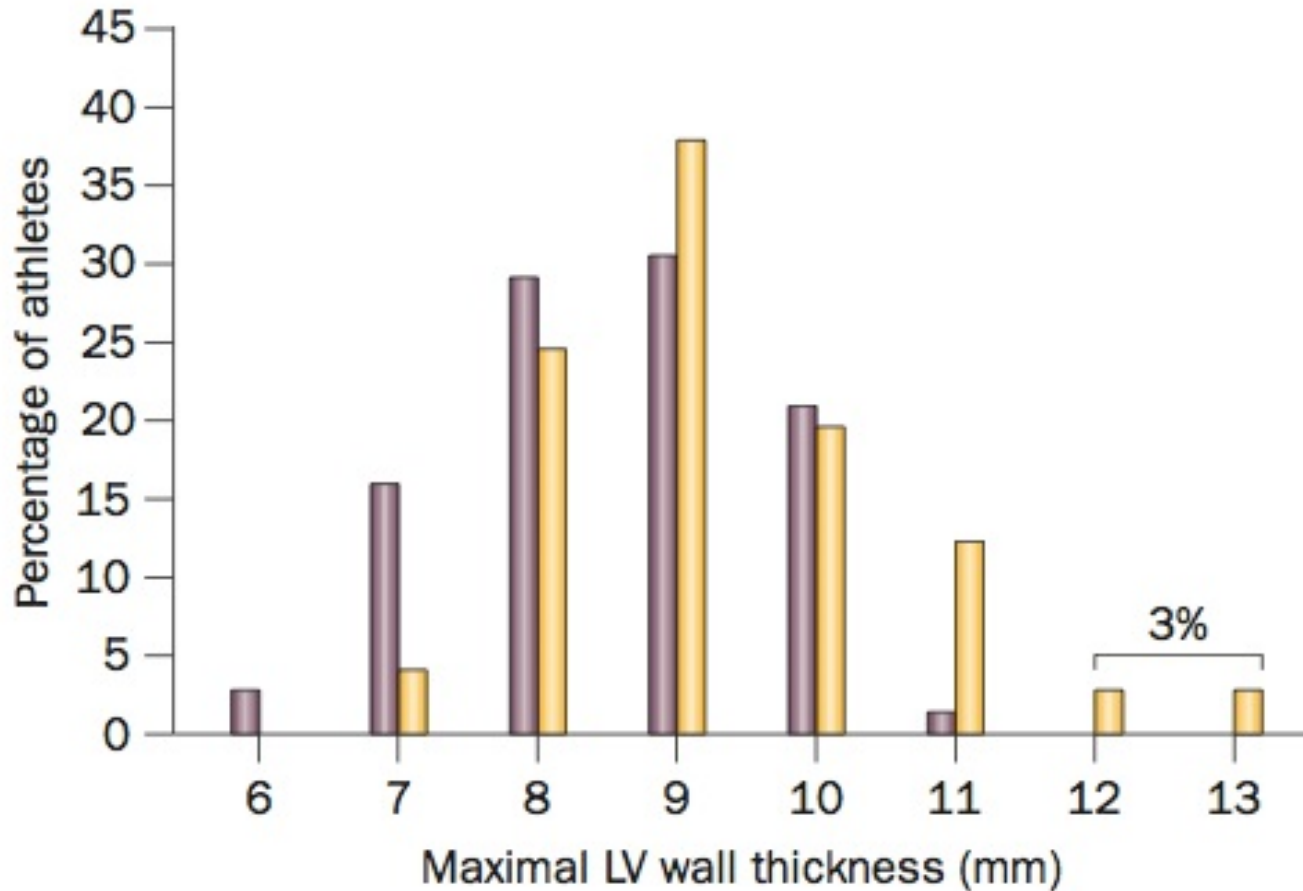


Compared with male athletes, female athletes:

-LVEDD 11% smaller

-LVT 23% thinner

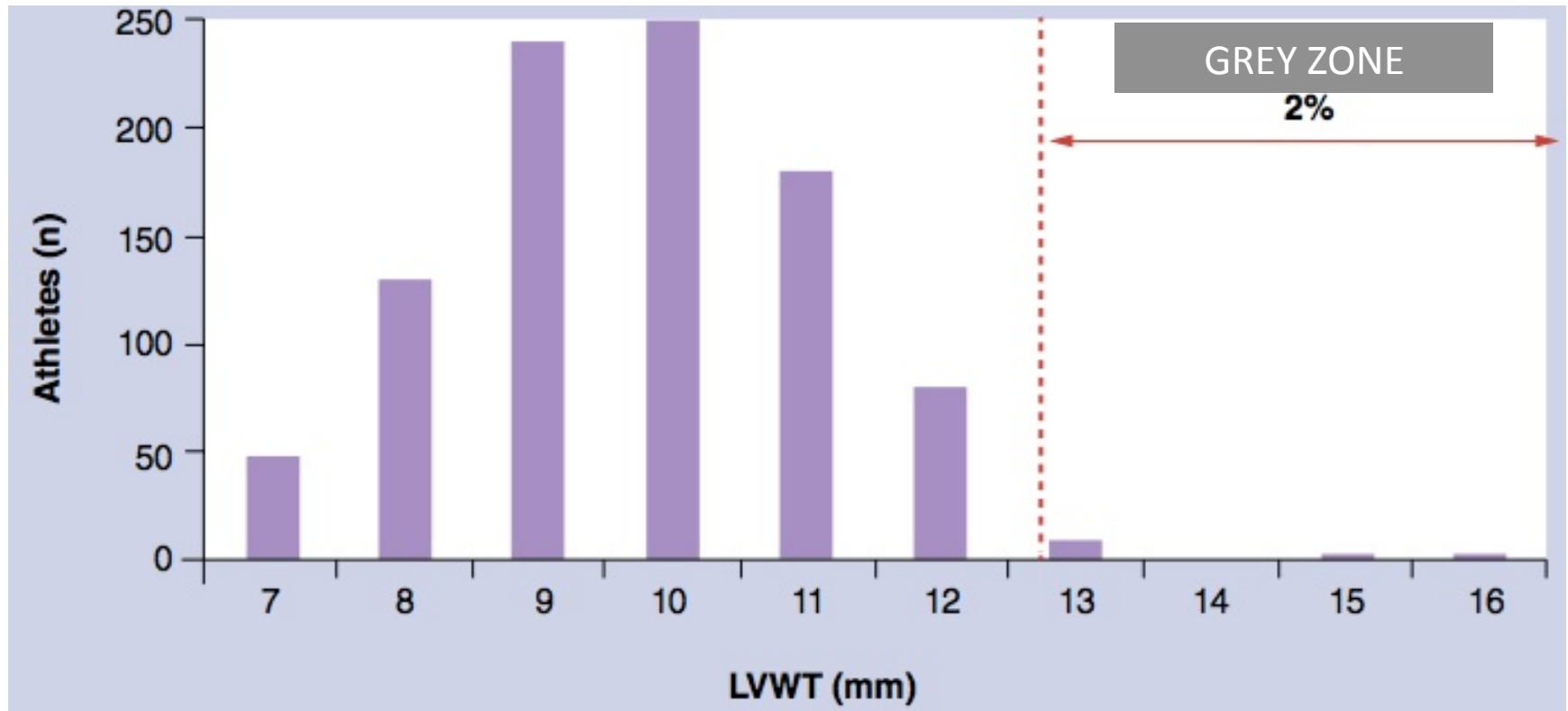
Black vs. White (female) LV wall thickness



LVWT (white athletes)



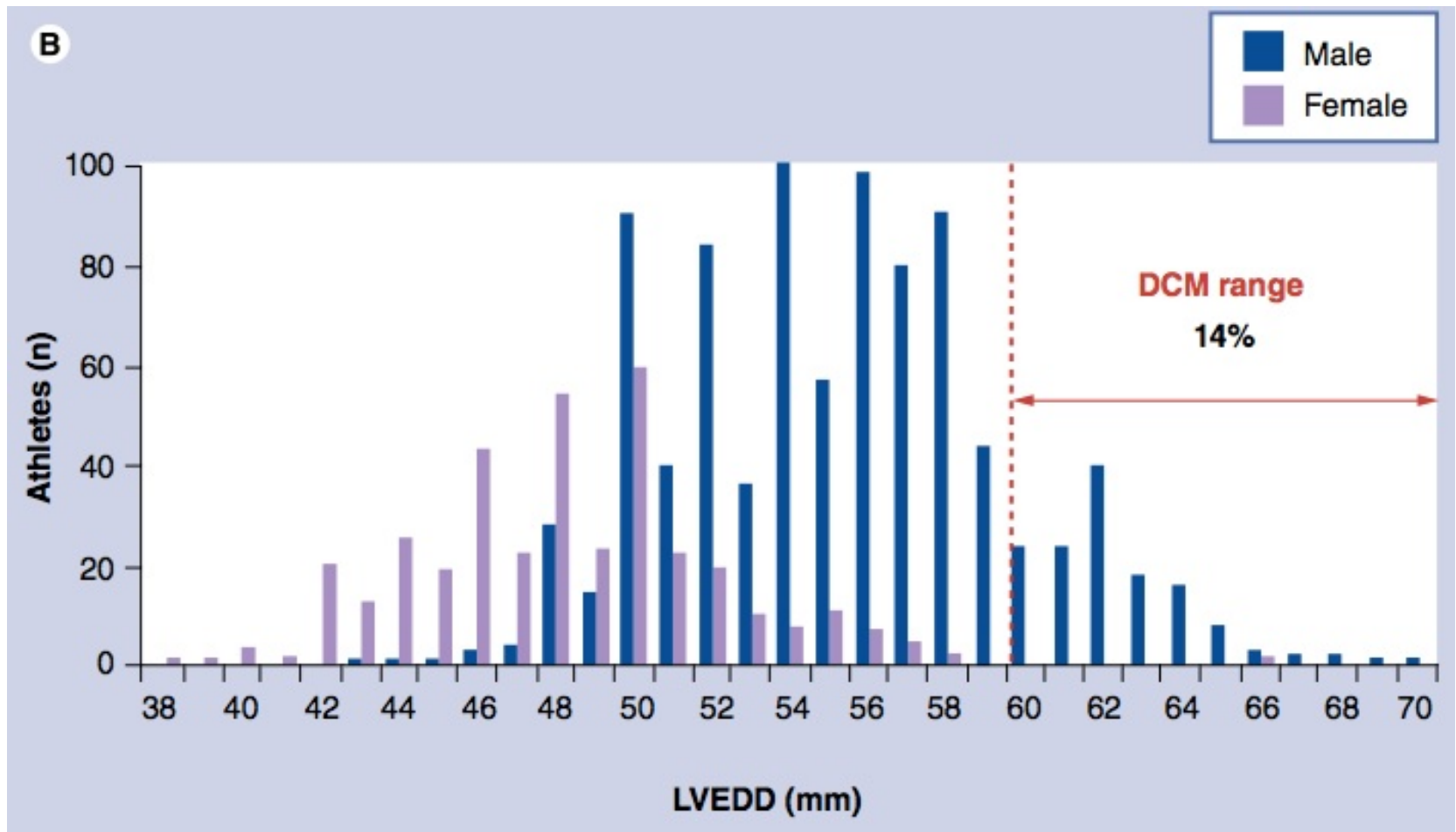
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LVEDD



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LWT for athletes (adults)

White

- Male:
 - Mean 10.1mm
 - Upper limit of normal – **12mm**
- Female:
 - Mean 8.2mm
 - Upper limit of normal – **10mm**

Only 2% of MALE Caucasian athletes show an LV wall thickness >12mm

Black

- Male:
 - Mean 11.3mm
 - Upper limit of normal – **15mm**
- Female:
 - Mean 10.2mm
 - Upper limit of normal – **12mm**

13% of black males and 3% of black female athletes show an LV wall thickness >12 mm

Upper limit (mean+2 SD) (mm)

LWT for athletes (adolescents)

White

- Male:
 - Mean $9.8 \pm 1.2\text{mm}$
 - Upper limit of normal – **12mm**
- Female:
 - Mean $8.4 \pm 1.1\text{mm}$
 - Upper limit of normal – **11mm**

Black

- Male:
 - Mean $10.3 \pm 1.6\text{mm}$
 - Upper limit of normal – **14mm**
- Female:
 - Mean $9.2 \pm 1.1\text{mm}$
 - Upper limit of normal – **11mm**

HCM should be considered strongly in any trained adolescent male athlete with LVWT 12 mm (females 11 mm) and non-dilated left ventricle.

LVEDD for athletes (adults)

White

- Male:
 - Mean $55.0 \pm 4.3\text{mm}$
 - Upper limit of normal – **64mm**
- Female:
 - Mean $48.9 \pm 3.8\text{mm}$
 - Upper limit of normal – **57mm**

Black

- Male:
 - Mean $53.0 \pm 4.4\text{mm}$
 - Upper limit of normal – **62mm**
- Female:
 - Mean $48.6 \pm 3.9\text{mm}$
 - Upper limit of normal – **56mm**

LVEDD >60 mm was exhibited by 14% of male athletes but by less than 1% of female athletes

Upper limit (mean+2 SD) (mm)



LVEDD for athletes (adolescents)

White

- Male:
 - Mean $51.6 \pm 3.3\text{mm}$
 - Upper limit of normal – **58mm**
- Female:
 - Mean 47.7 ± 3.3
 - Upper limit of normal – **54mm**

Black

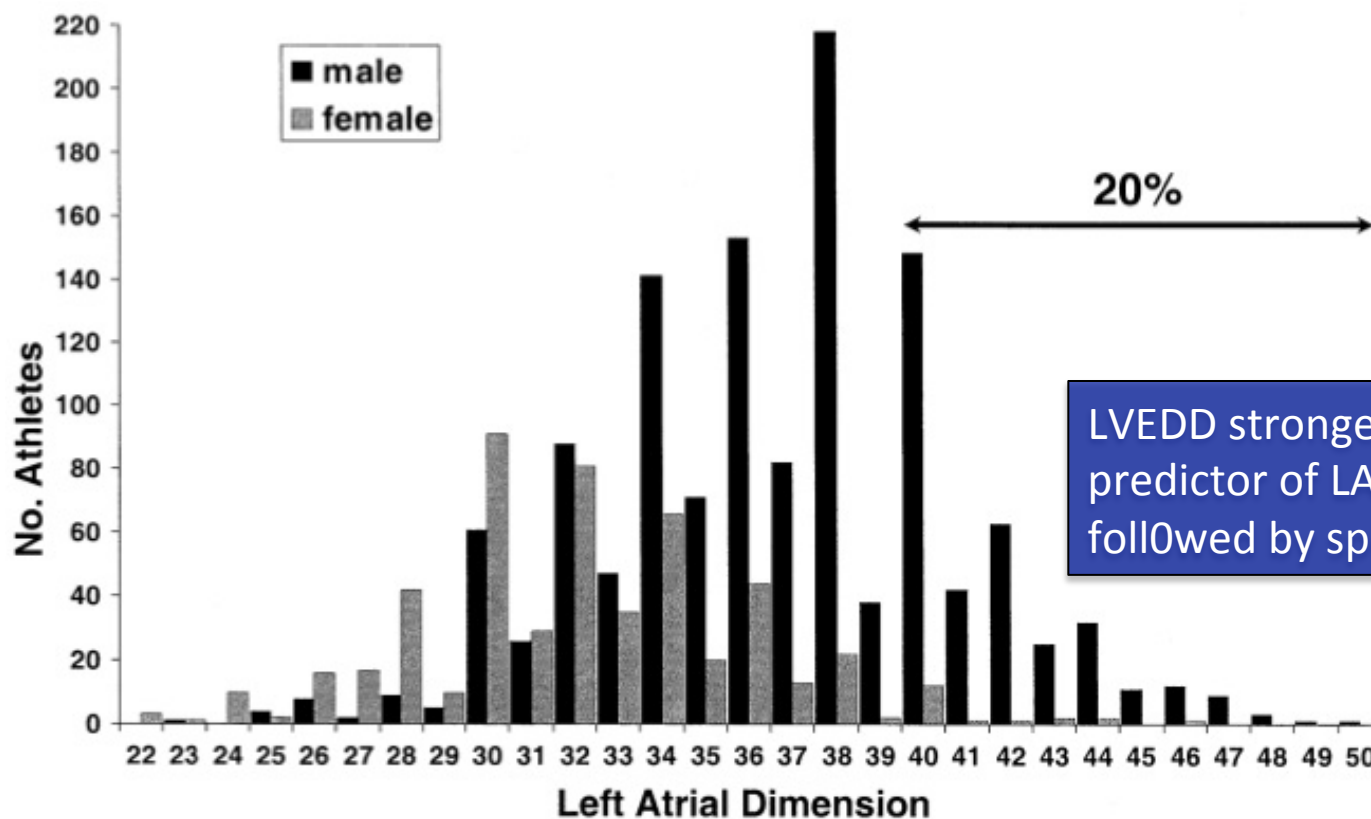
- Male:
 - Mean $52.3 \pm 5.0\text{mm}$
 - Upper limit of normal – **62mm**
- Female:
 - Mean 48.7 ± 4.2
 - Upper limit of normal – **57mm**

Upper limit (mean+2 SD) (mm)

Athletics and Cardiac Function

Prevalence and Clinical Significance of Left Atrial Remodeling in Competitive Athletes

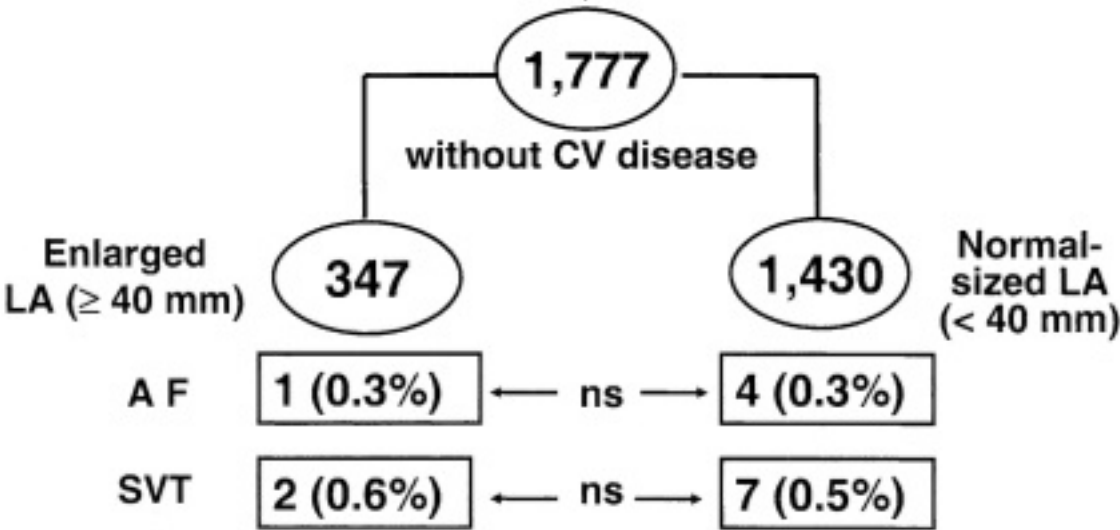
Antonio Pelliccia, MD,* Barry J. Maron, MD,† Fernando M. Di Paolo, MD,* Alessandro Biffi, MD,* Filippo M. Quattrini, MD,* Cataldo Pisicchio, MD,* Alessandra Roselli, MD,* Stefano Caselli, MD,* Franco Culasso, PhD‡



Athletics and Cardiac Function

Prevalence and Clinical Significance of Left Atrial Remodeling in Competitive Athletes

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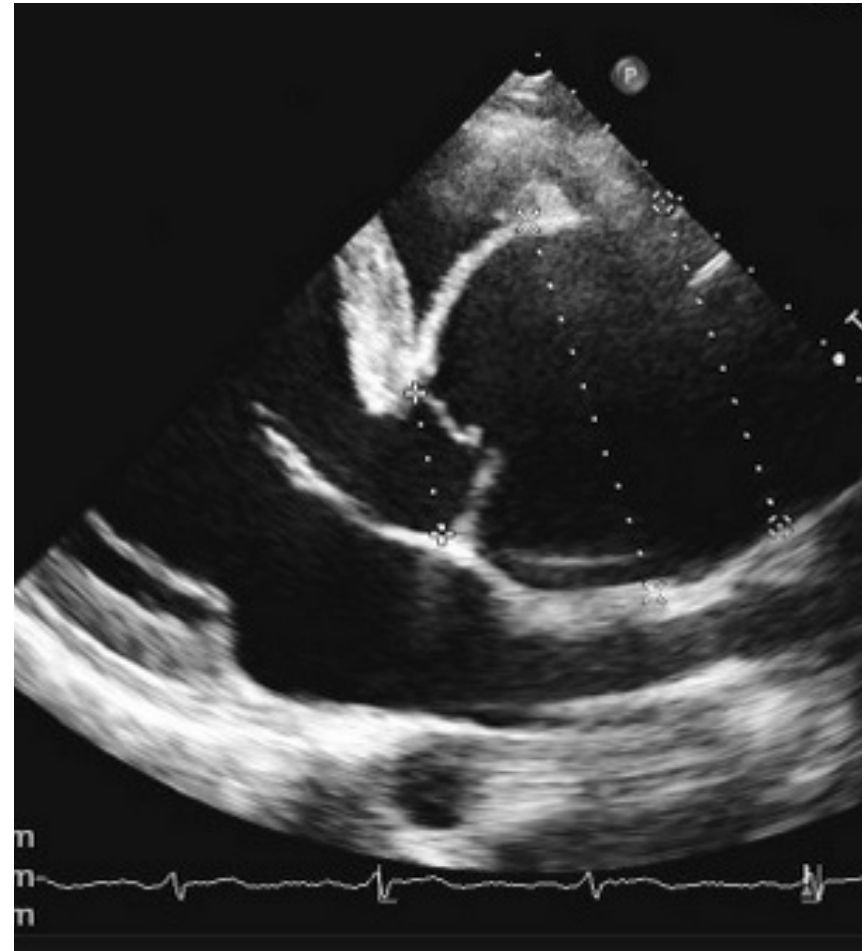
LA size failed to predict development of SVT/AF





Aorta

- Athletes can have a slightly larger aortic root compared to their age and sex matched sedentary controls but an aortic root $>40\text{mm}$ is **ABNORMAL**



What is 'normal' for an athlete?

Gieco Baby advertisement

	Non-athletes		Athletes					
	<i>Male</i>	<i>Female</i>	<i>Caucasian adults</i>		<i>Caucasian adolescents</i>		<i>Black adults</i>	
			<i>Male</i>	<i>Female</i>	<i>Male</i>	<i>Female</i>	<i>Male</i>	<i>Female</i>
LVEDD (mm)	≤59	≤53	≤63	≤56	≤58	≤54	≤62	≤56
LVWT (mm)	≤10	≤9	≤12	≤11	≤12	≤11	≤15	≤12
RVOT1 (mm)	≤35	≤35	≤38	≤37	-	-	-	-
RVD1 (mm)	≤42	≤42	≤45	≤42	-	-	-	-

An adult heart should be bigger. If an adolescent heart looks big it probably is big.

NO adolescent athletes exhibited a LVEDD >60mm (Makan, Heart 2005)

LVWT >11mm only occurred in 0.4% of adolescents (Sharma, JACC 2002)

What is 'normal' for an athlete?

Table 3 | Upper limits of normal for LV end-diastolic diameters and maximal wall thickness in athletes

Cohort	Sex	LV end-diastolic diameter				LV wall thickness			
		Study	Mean age (years) $\pm$ SD	Mean $\pm$ SD (mm)	Upper limit (mean + 2 SD) (mm)	Study	Mean age (years) $\pm$ SD	Mean $\pm$ SD (mm)	Upper limit (mean + 2 SD) (mm)
Adult white	Male	Pelliccia <i>et al.</i> ⁷⁹	24.3 $\pm$ 6.0	55. $\pm$ 4.3	$\leq$ 64	Pelliccia <i>et al.</i> ⁷⁷	22.4 $\pm$ 3.1	10.1 $\pm$ 1.1	$\leq$ 12
	Female	Pelliccia <i>et al.</i> ⁷⁸	21.1 $\pm$ 5.0	48.9 $\pm$ 3.8	$\leq$ 57	Pelliccia <i>et al.</i> ⁷⁸	21.1 $\pm$ 5.0	8.2 $\pm$ 1.2	$\leq$ 11
Adolescent white	Male	Makan <i>et al.</i> ⁸¹	15.7 $\pm$ 1.2	51.6 $\pm$ 3.3	$\leq$ 58	Sharma <i>et al.</i> ⁸⁰	15.6 $\pm$ 1.2	9.8 $\pm$ 1.2	$\leq$ 12
	Female		15.7 $\pm$ 1.2	47.7 $\pm$ 3.3	$\leq$ 54		15.4 $\pm$ 1.1	8.4 $\pm$ 1.1	$\leq$ 11
Adult black	Male	Basavarajaiah <i>et al.</i> ¹³⁵	20.5 $\pm$ 5.8	53.0 $\pm$ 4.4	$\leq$ 62	Basavarajaiah <i>et al.</i> ¹³⁵	20.5 $\pm$ 5.8	11.3 $\pm$ 1.6	$\leq$ 15
	Female	Rawlins <i>et al.</i> ¹²⁹	21 $\pm$ 4.6	48.6 $\pm$ 3.9	$\leq$ 56	Rawlins <i>et al.</i> ¹²⁹	21 $\pm$ 4.6	9.2 $\pm$ 1.2	$\leq$ 12
Adolescent black	Male	Sheikh <i>et al.</i> ¹³⁴	16.4 $\pm$ 1.3	52.3 $\pm$ 5.0	$\leq$ 62	Sheikh <i>et al.</i> ¹³⁴	16.4 $\pm$ 1.3	10.3 $\pm$ 1.6	$\leq$ 14
	Female		16.0 $\pm$ 1.3	48.7 $\pm$ 4.2	$\leq$ 57		16.0 $\pm$ 1.3	9.2 $\pm$ 1.1	$\leq$ 11
Adult Middle-Eastern	Male	Riding <i>et al.</i> ¹⁴⁹	22.7 $\pm$ 5.9	52.7 $\pm$ 4.2	$\leq$ 61	Riding <i>et al.</i> ¹⁴⁹	22.7 $\pm$ 5.9	8.9 $\pm$ 0.9	$\leq$ 11
	Female	NA	NA	NA	NA	NA	NA	NA	NA
Adult East Asian	Male	Ma <i>et al.</i> ¹⁵⁵	23.0 $\pm$ 3.8	NR	NA	Ma <i>et al.</i> ¹⁵⁵	23.0 $\pm$ 3.8	NR	NA
	Female		20.7 $\pm$ 4.1				20.7 $\pm$ 4.1		
Adolescent East Asian	Male	NA	NA	NA	NA	NA	NA	NA	NA
	Female								
Adult South Asian	Male	NA	NA	NA	NA	NA	NA	NA	NA
	Female								
Adolescent South Asian	Male	NA	NA	NA	NA	NA	NA	NA	NA
	Female								



Upper ranges for athletes

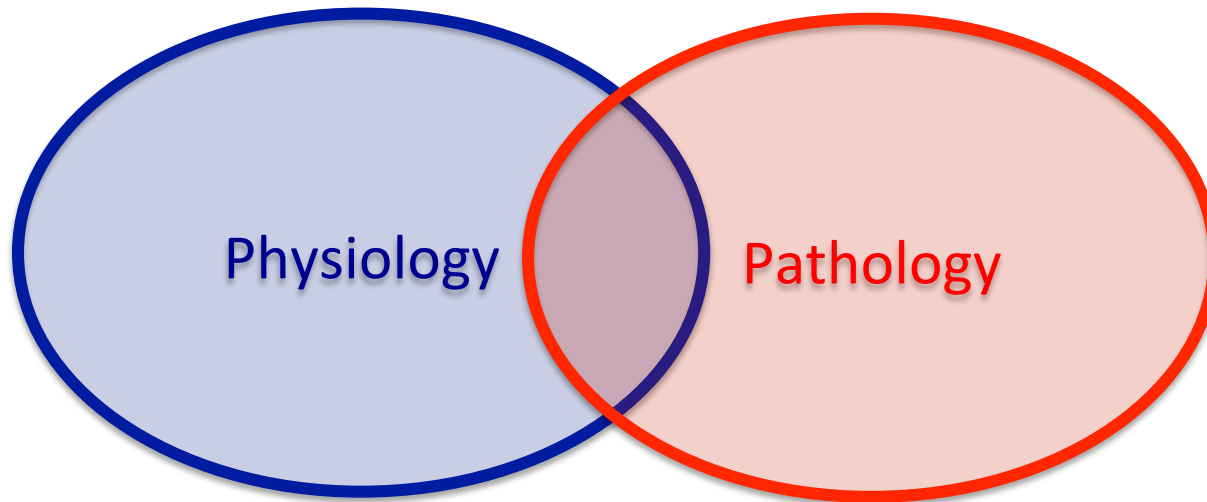
		LVEDD (mm)	LVWT (mm)	RVD1 (mm)	RVOT 1 (mm)
Non-athletes	Male	59	10	38	35
	Female	53	9	38	35
Athletes	Caucasian adult				
	Male	63	12	55	43
	Female	56	11	49	40
	Caucasian adolescent (14–18)				
	Male	58	12	–	–
	Female	54	11	–	–
	Black adult				
	Male	62	15	55	43
	Female	56	12	49	40
	Black adolescent (14–18)				
	Male	62	15	–	–
	Female	56	11	–	–

The magnitude of these adaptations is lower in adolescent athletes who are generally physically less mature and have trained for shorter periods.

The Goal

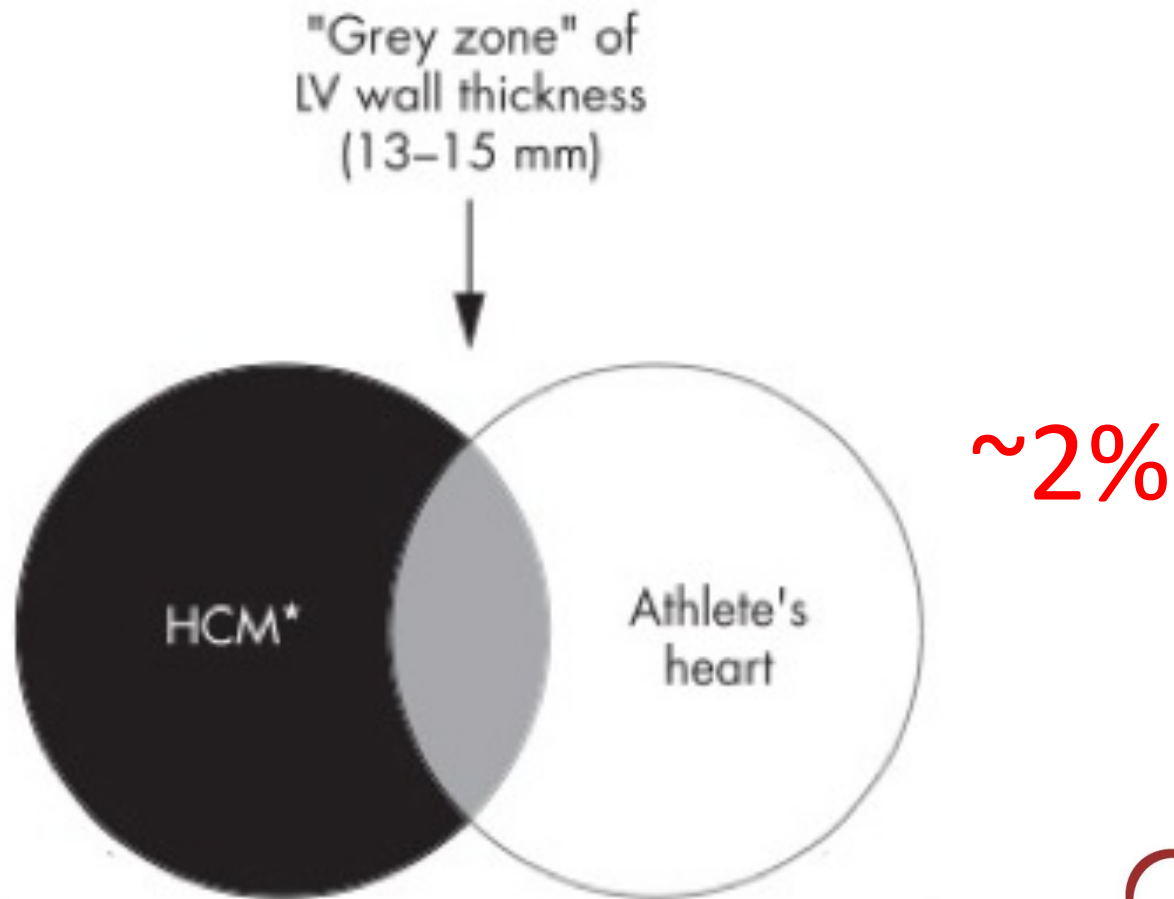


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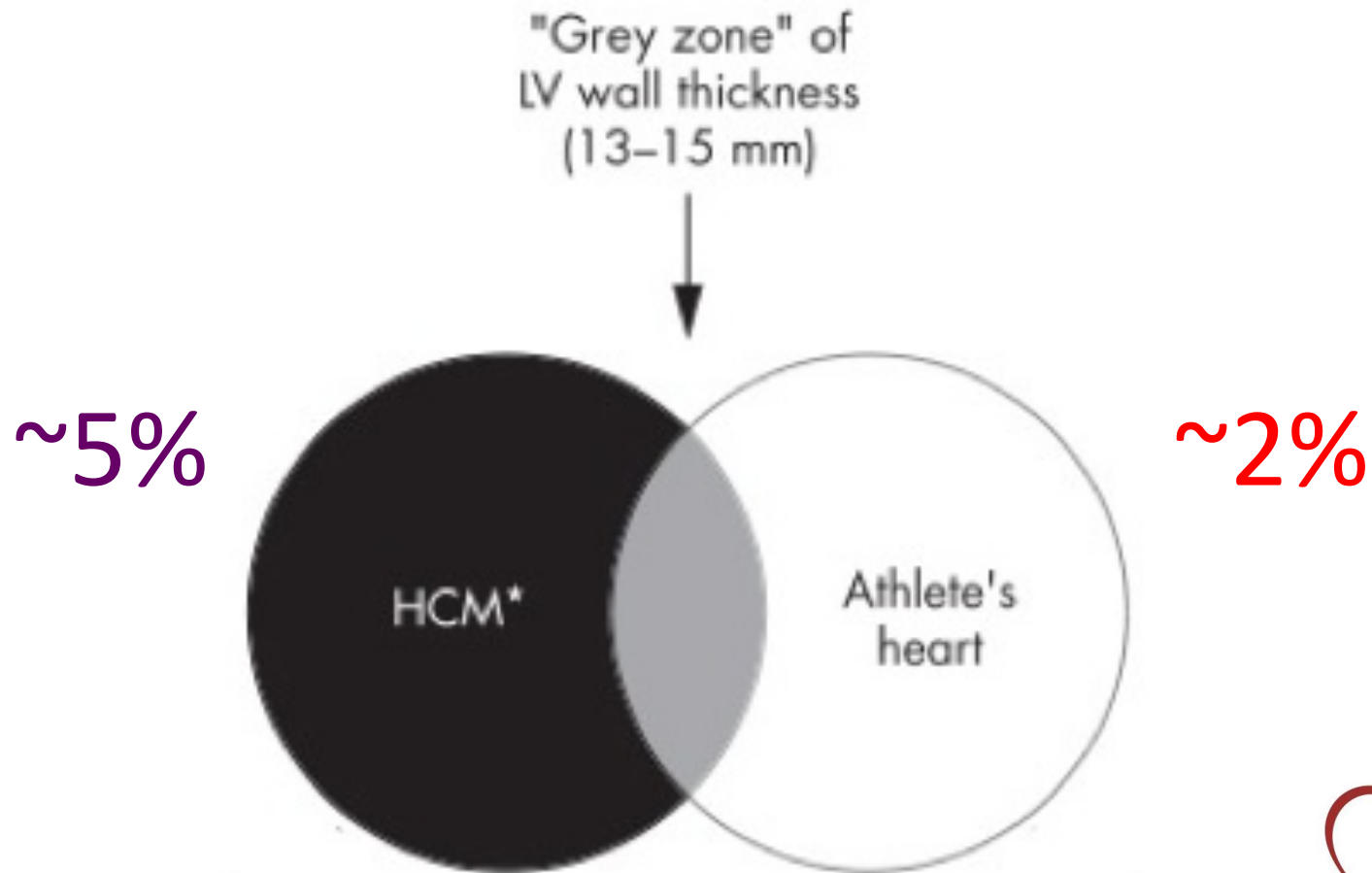


to differentiate normal findings in an athlete from the presence of cardiac pathology

What % of **athletes** fall into the 'Grey Zone'

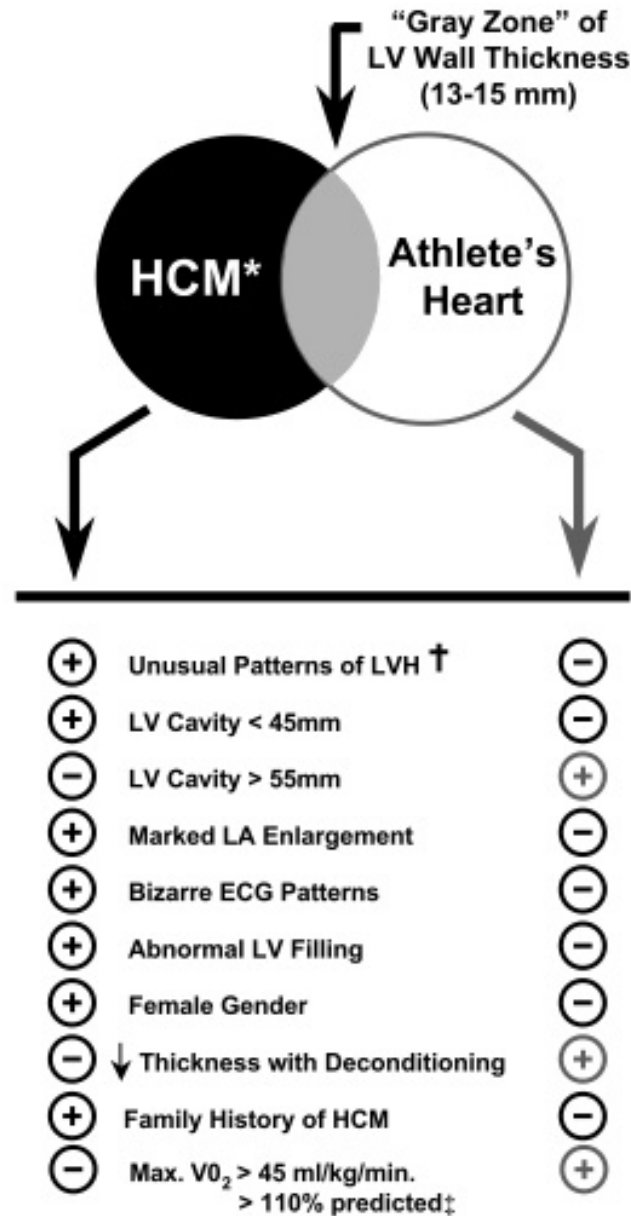


What % of **HCM** fall into the 'Grey Zone'





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Maron, Pelliccia, Circ. 1995

Differentiating Left Ventricular Hypertrophy in Athletes from That in Patients With Hypertrophic Cardiomyopathy

Stefano Caselli, MD, PhD^{a,*}, Martin S. Maron, MD^b, Josè A. Urbano-Moral, MD^b,
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- 1,191 consecutive highly trained athletes were evaluated at the Institute of Sport Medicine and Science in Rome
- 28 athletes (2.3%) had LVT 13 to 15mm in the 'grey zone' not thought to be due to HCM
- 25 non-obs HCM from Tufts University
- Followed for 4 years



Differentiating Left Ventricular Hypertrophy in Athletes from That in Patients With Hypertrophic Cardiomyopathy

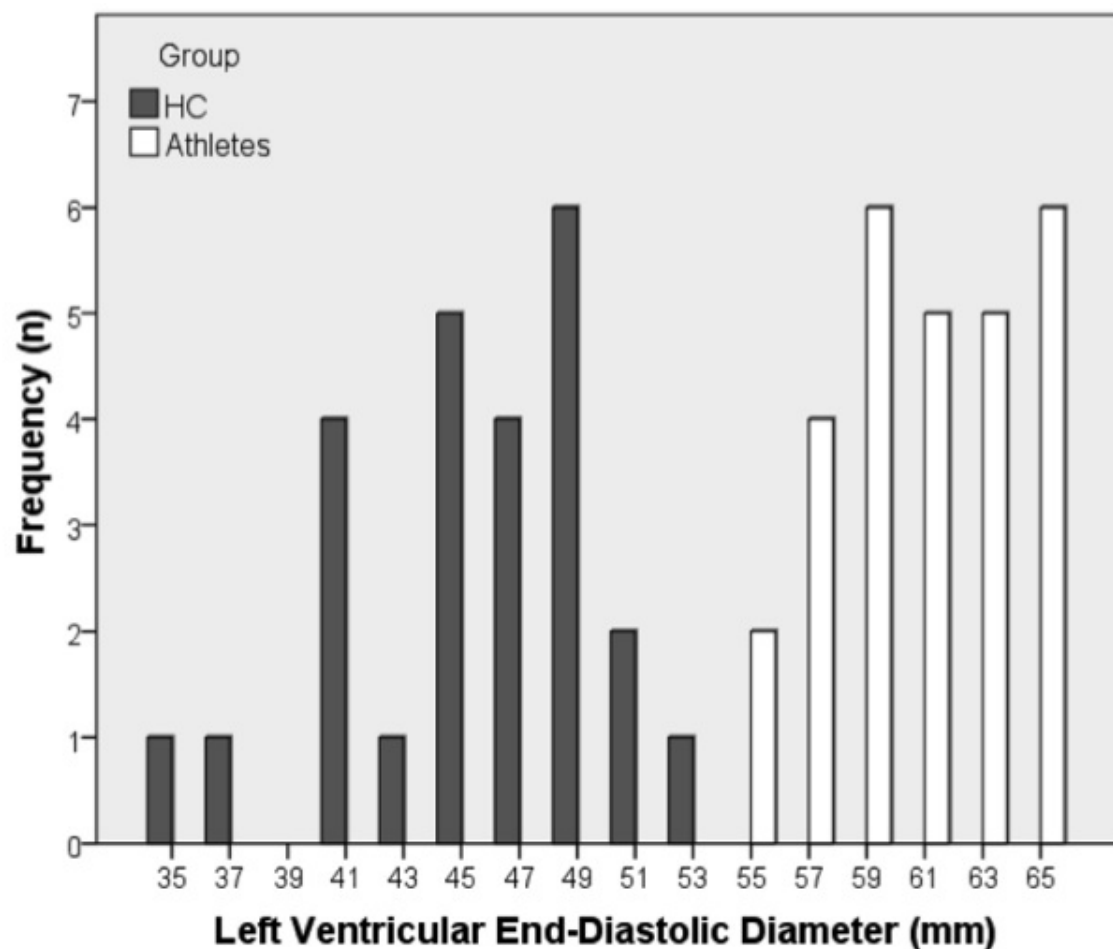
Stefano Caselli, MD, PhD^{a,*}, Martin S. Maron, MD^b, Josè A. Urbano-Moral, MD^b,
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Variable	Athletes (n = 28)	HC (n = 25)	p-Value
Anterior septum (mm)	12.5 ± 0.6	13.8 ± 1.5	<0.001
Posterior septum (mm)	13.1 ± 0.4	12.0 ± 1.7	0.002
LV Posterior free wall (mm)	11.7 ± 0.7	9.3 ± 1.5	<0.001
LV Lateral free wall (mm)	11.3 ± 0.8	8.7 ± 1.1	<0.001
LV end-diastolic diameter (mm)	60 ± 3	45 ± 4	<0.001
LV end-systolic diameter (mm)	37 ± 5	24 ± 4	<0.001
Relative wall thickness*	0.42 ± 0.03	0.62 ± 0.11	<0.001
Left atrium diameter (mm)	42 ± 4	34 ± 5	<0.001
Aortic root (mm)	34 ± 3	30 ± 3	<0.001
Ejection fraction (%)	63 ± 5	64 ± 6	0.488
E wave (cm/s)	82 ± 18	80 ± 21	0.767
A wave (cm/s)	44 ± 8	57 ± 18	0.001
E/A ratio	1.9 ± 0.5	1.6 ± 0.6	0.032
Deceleration time (ms)	207 ± 53	175 ± 30	0.011
IVRT (ms)	97 ± 16	88 ± 13	0.135
TDI e' wave (cm/s)	12.5 ± 1.9	9.1 ± 2.4	<0.001
TDI a' wave (cm/s)	7.3 ± 1.6	6.7 ± 2.5	0.452
TDI e'/a' ratio	1.77 ± 0.40	1.55 ± 0.70	0.170
TDI s wave (cm/s)	9.1 ± 1.8	8.2 ± 1.4	0.087
E/e' ratio	6.6 ± 1.2	9.2 ± 2.5	<0.001



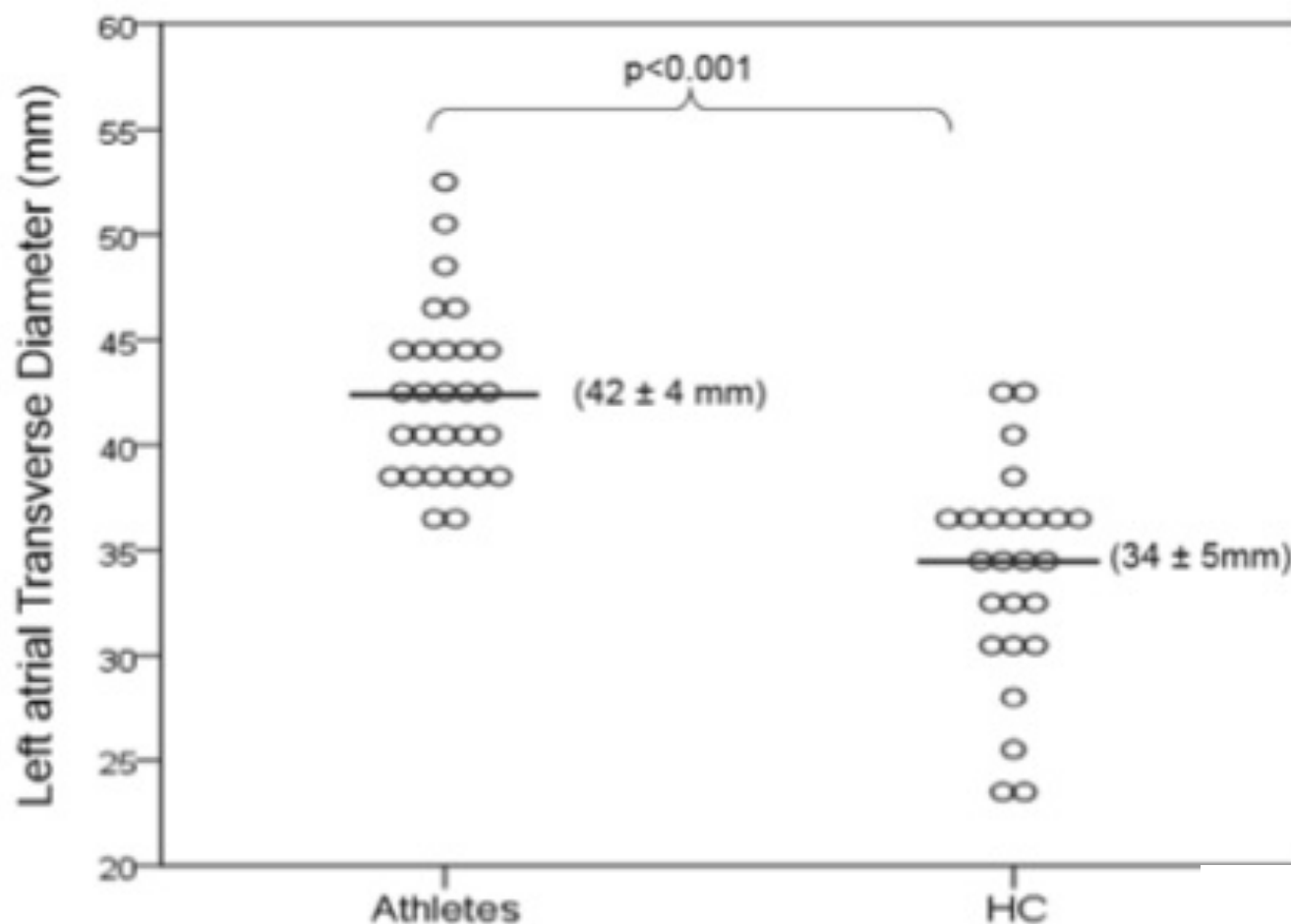
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Variable	Athletes (n = 28)	HC (n = 25)	p-Value
Age (years)	26 ± 4	28 ± 10	0.247
Body Surface Area (m ²)	2.12 ± 0.27	2.05 ± 0.21	0.335
Systolic Blood Pressure (mmHg)	128 ± 10	120 ± 13	0.015
Diastolic Blood Pressure (mmHg)	79 ± 7	74 ± 9	0.016
Heart rate (bpm)	52 ± 10	64 ± 10	<0.001
Family history for HC	0	11 (44%)	<0.001
Sokolow-Lyon score (mm)	38 ± 13	31 ± 14	0.100
Left axis deviation	1 (4%)	0	0.340
Left atrial enlargement	5 (18%)	4 (16%)	0.857
Q waves	0	3 (12%)	0.059
T-wave abnormalities	2 (7%)	13 (52%)	<0.001
Ventricular premature complexes	2 (7%)	2 (8%)	0.552



ROC analysis



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Variable	Cutoff	AUC	Sensitivity (%)	Specificity (%)	p-Value
LV end-diastolic diameter (mm)	<54	1.000	100	100	<0.001
Left atrium (mm)	<40	0.919	92	71	<0.001
TDI e' (cm/s)	<11.5	0.859	83	61	<0.001
E/e' ratio	>7.3	0.830	77	71	<0.001
A-wave (cm/s)	>46	0.741	70	64	0.003
Inverted T-waves	Present	0.724	52	92	0.005
Family Hx for HC	Present	0.720	44	100	0.006



$e' < 11.5 \text{ cm/s}$

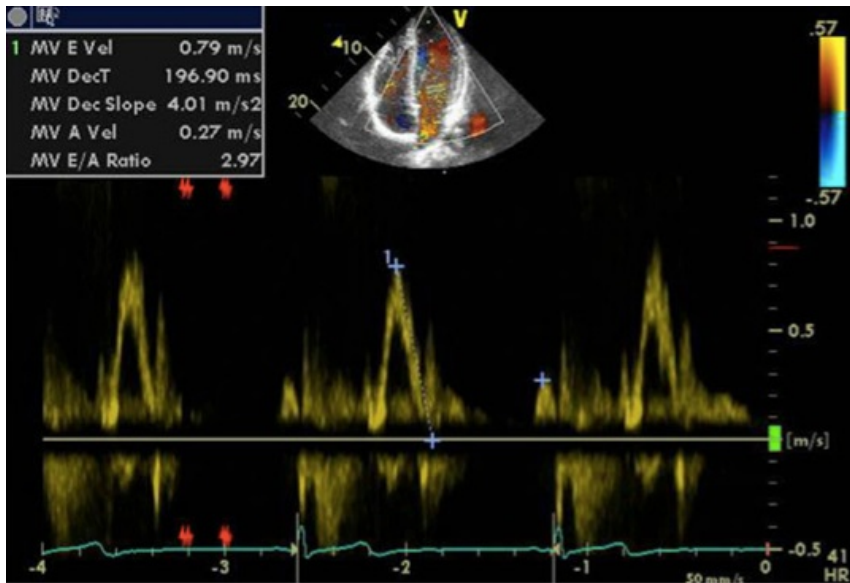
- $< 11.5 \text{ cm/s}$ TDI may be useful to raise suspicion for nonphysiologic LVH
- Athletes should have normal or supranormal LV relaxation
 - A' waves were lower in athletes (modest contribution of atria to ventricular filling)
- Diastolic dysfunction may be the first expression of HCM (in addition to ECG) and may precede the development of LV hypertrophy

E/A ratio

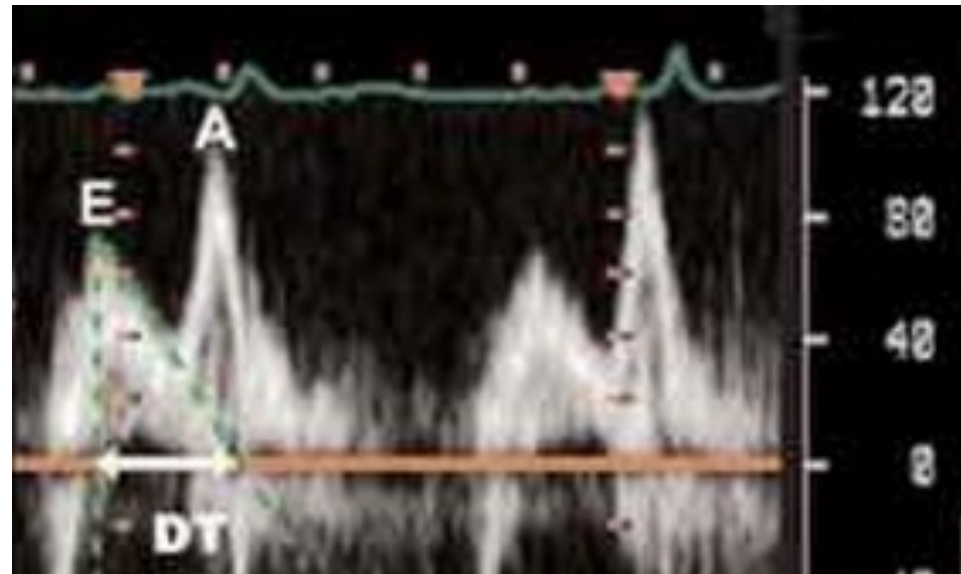


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Athlete – E/A > 2



Path LVH – E/A ~ 1 + prolonged DT



The pattern of LVH



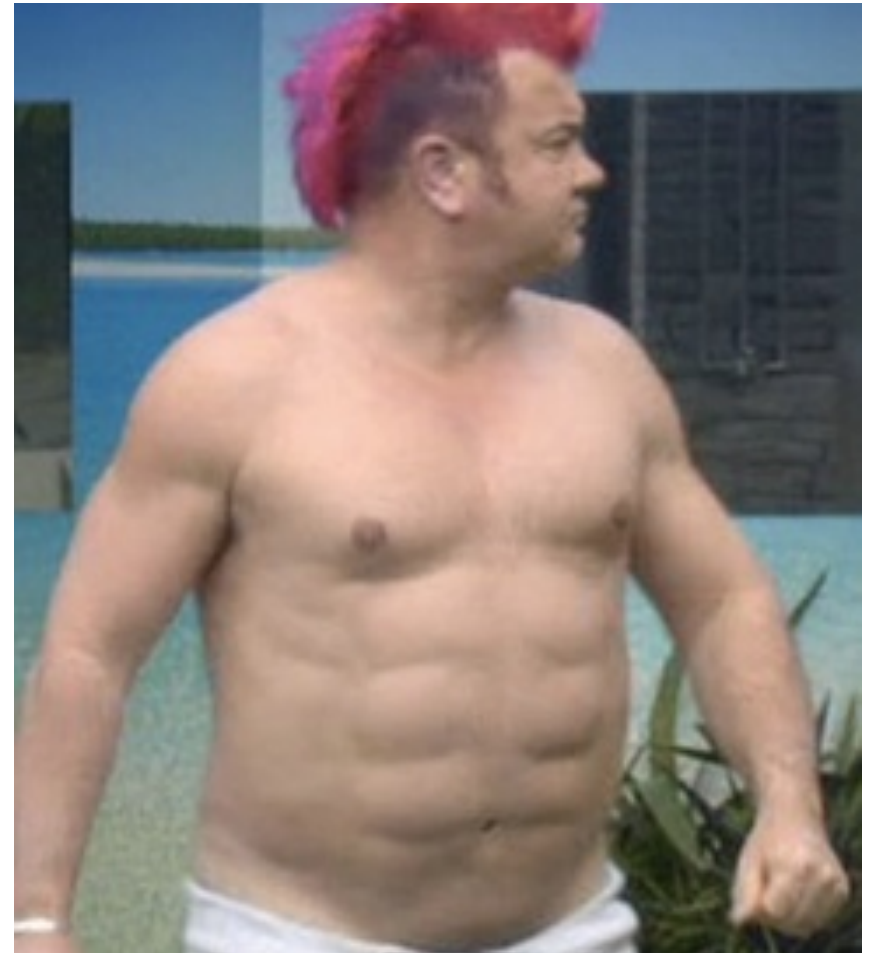
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The pattern of LVH



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The pattern of LVH

- In physiologic hypertrophy of the athlete, although the anterior ventricular septum is usually the segment of the LV wall that is maximally thickened, the overall pattern is **symmetric** and **homogeneous**, usually with a difference of **<2 mm** or less between all portions of the LV
- Its is 'not natural' to train just one part of your ventricle to get hypertrophied

Does being an elite athlete make HCM less likely?

Prevalence of Hypertrophic Cardiomyopathy in Highly Trained Athletes

Relevance to Pre-Participation Screening

Sandeep Basavarajaiah, MBBS, MRCP,*† Matthew Wilson, MSc, MPhil,‡ Gregory Whyte, PhD,‡
Ajay Shah, PhD, FRCP,* William McKenna, DSc, FRCP, FESC, FACC,§
Sanjay Sharma, BSc (HONS), MD, FRCP*†

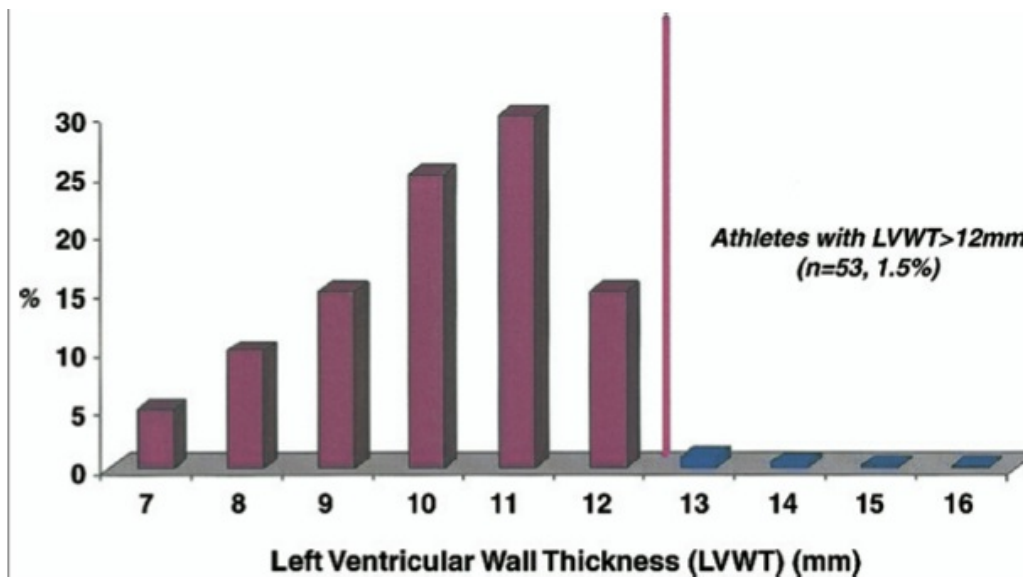


Figure 1 Distribution of LVWT in 3,500 Elite Athletes



Does being an elite athlete make HCM less likely?



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Table 2

Echocardiographic Features in Athletes With Left Ventricular Wall Thickness >12 mm

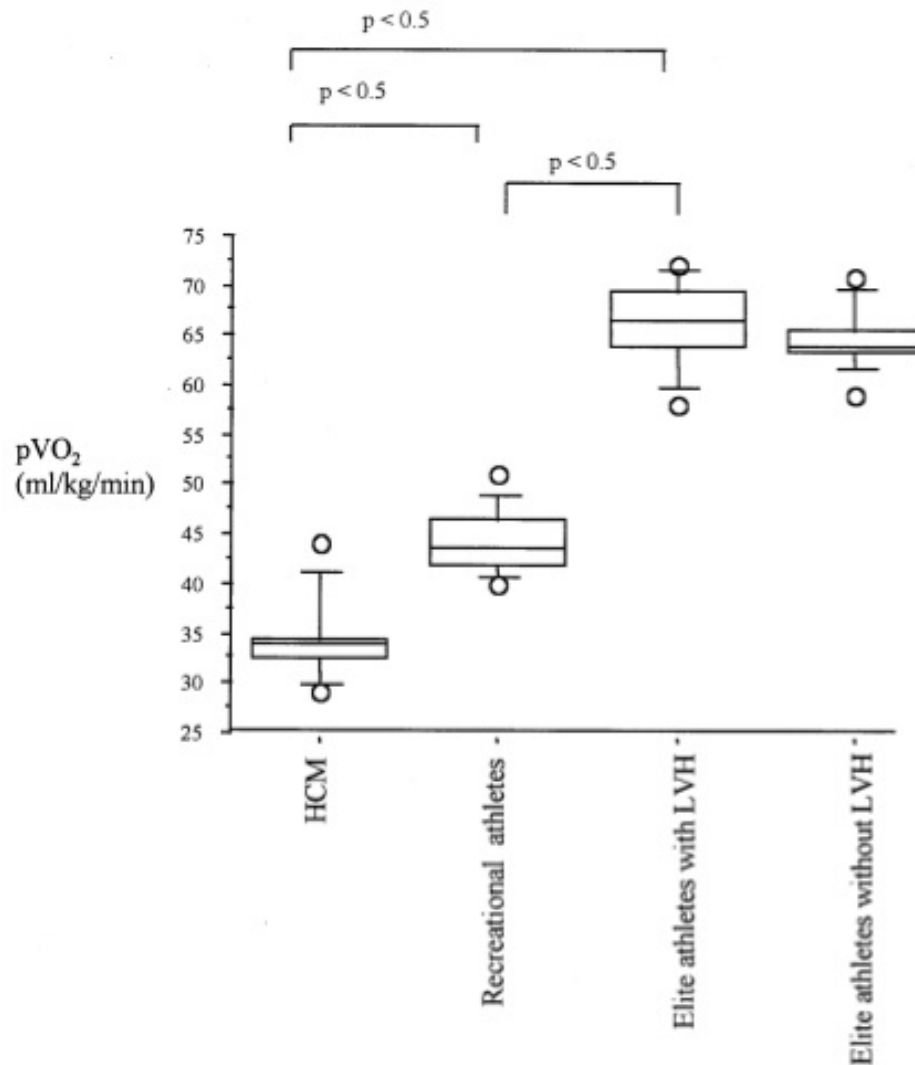
	Mean $\pm$ Standard Deviation (Range)
LVWTd (mm)	13.6 $\pm$ 0.9 (13–16)
LVIDd (mm)	58.5 $\pm$ 5.14 (45–65)
LVIDs (mm)	31.6 $\pm$ 4.1 (22–42)
Left atrial diameter (mm)	32 $\pm$ 4.8 (21–47)
E-wave (m/s)	0.87 $\pm$ 0.2 (0.5–1.5)
A-wave (m/s)	0.45 $\pm$ 0.2 (0.17–0.9)
E/A ratio	2.32 $\pm$ 0.94 (1.8–4.5)

Table 3 Echocardiographic and Cardiopulmonary Exercise Parameters in Athletes With Left Ventricular Hypertrophy and a Nondilated Left Ventricular Cavity Size

	Sport	LVWTd (mm)	LVIDd (mm)	LA (mm)	E-Wave (m/s)	A-Wave (m/s)	E/A Ratio	Peak Systolic BP	PvO ₂ (ml/kg/min)	PvO ₂ (% Predicted)	AT (% Predicted)	First-Degree Family Members With HCM
Athlete 1	Swimmer	14	46	46	0.9	0.40	2.25	240	66	148	65	None
Athlete 2	Soccer	15	45	39	0.8	0.35	2.28	245	62	120	63	None
Athlete 3	Soccer	16	45	31	1.04	0.45	2.3	260	60	116	64	None

Utility of Metabolic Exercise Testing in Distinguishing Hypertrophic Cardiomyopathy from Physiologic Left Ventricular Hypertrophy in Athletes

Sanjay Sharma, BSc, MRCP, Perry M. Elliott, MRCP, Greg Whyte, PhD,* Niall Mahon, MRCP(I), MD,
Mohan S. Virdee, MRCP, Brian Mist, PhD, William J. McKenna, FRCP, FACC, FESC



The cardiovascular response to exercise is a powerful discriminator between physiology and disease.

HCM



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voltages in hypertrophic cardiomyopathy versus athlete's heart: Relevance to athletic screening☆

Chiara Calore ^a, Paola Melacini ^a, Antonio Pelliccia ^b, Cinzia Cianfrocca ^c, Maurizio Schiavon ^d,
Fernando M. Di Paolo ^b, Francesca Bovolato ^a, Filippo M. Quattrini ^b, Cristina Basso ^a, Gaetano Thiene ^a,
Sabino Iliceto ^a, Domenico Corrado ^{a,*}

Comparison of ECG findings in HCM and athletes.

ECG	HCM (N = 247)	Athletes (N = 133)	P value
No ECG abnormalities	11 (4)	69 (52)	<0.001
Increased QRS voltage (Sokolow-Lyon) ^a n (%)	90 (36)	59 (44)	0.1
Isolated, n (%)	6/247 (2)	53/133 (40)	<0.001
Associated with non-voltage criteria, n (%)	84/247 (34)	6/133 (4)	<0.001
Left atrial enlargement, n (%)	89 (36)	4 (3)	<0.001
Left axis deviation, n (%)	30 (12)	2 (1)	<0.001
Pathologic Q waves, n (%)	86 (35)	0	<0.001
ST-T alterations, n (%)	202 (82)	6 (4)	<0.001
ST-segment depression, n (%)	164 (66)	0	<0.001
T-wave inversion, n (%)	124 (50)	6 (4)	<0.001
Complete RBBB, n (%)	16 (6)	0	0.003
Complete LBBB, n (%)	11 (4)	0	0.01

Only 4% of patients with HCM had a normal ECG

Only 2% of HCM patients had isolated ECG voltages

52% with ECHO criteria for LVH had normal ECG

40% of athletes with LVH had isolated ECG voltage.



Relation of Electrocardiographic Patterns to Phenotypic Expression and Clinical Outcome in Hypertrophic Cardiomyopathy

Julia V. Montgomery, MD, Kevin M. Harris, MD, Susan A. Casey, RN,
Andrey G. Zenovich, MSc, Barry J. Maron, MD*

Relation of normal electrocardiograms to clinical parameters

Clinical Parameter	No. of Patients	No. With Normal ECG Results (%)
Outcome*		
Survival	376	17 (4.5%)
HC-related death	40	1 (2.5%)
Sudden HC-related death only	25	1 (4.0%)
Non-HC-related death	32	1 (3.1%)
LV wall thickness (mm) [†]		
<20	179	13 (7.3%)
20–29	214	6 (2.8%)
≥30	55	0 (0)

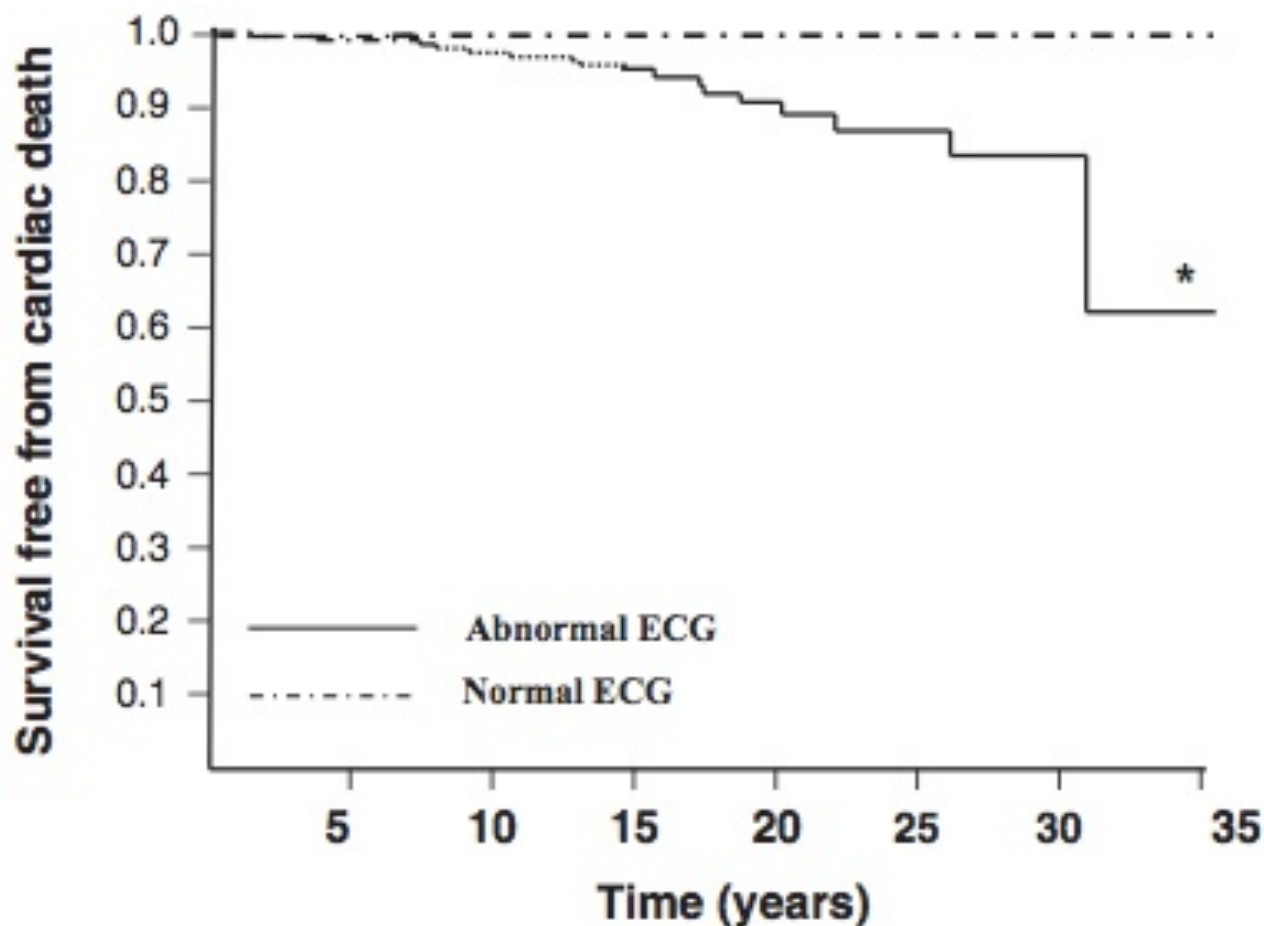
Montgomery, Am J Cardio, 2005



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Outcome of Patients With Hypertrophic Cardiomyopathy and a Normal Electrocardiogram

Christopher J. McLeod, MB, CHB, PhD, Michael J. Ackerman, MD, PhD, Rick A. Nishimura, MD, A. Jamil Tajik, MD, Bernard J. Gersh, MB, CHB, DPHIL, Steve R. Ommen, MD



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- In HCM patients with normal ECG compared to HCM with Abnormal ECG:
 - Younger
 - More likely to be female
 - Less myomectomy
 - Less ICD
 - Lower NYHA
 - Lower Resting gradient
 - Lower septal wall thickness

HCM with normal ECG
= 6%

Less severe
phenotype

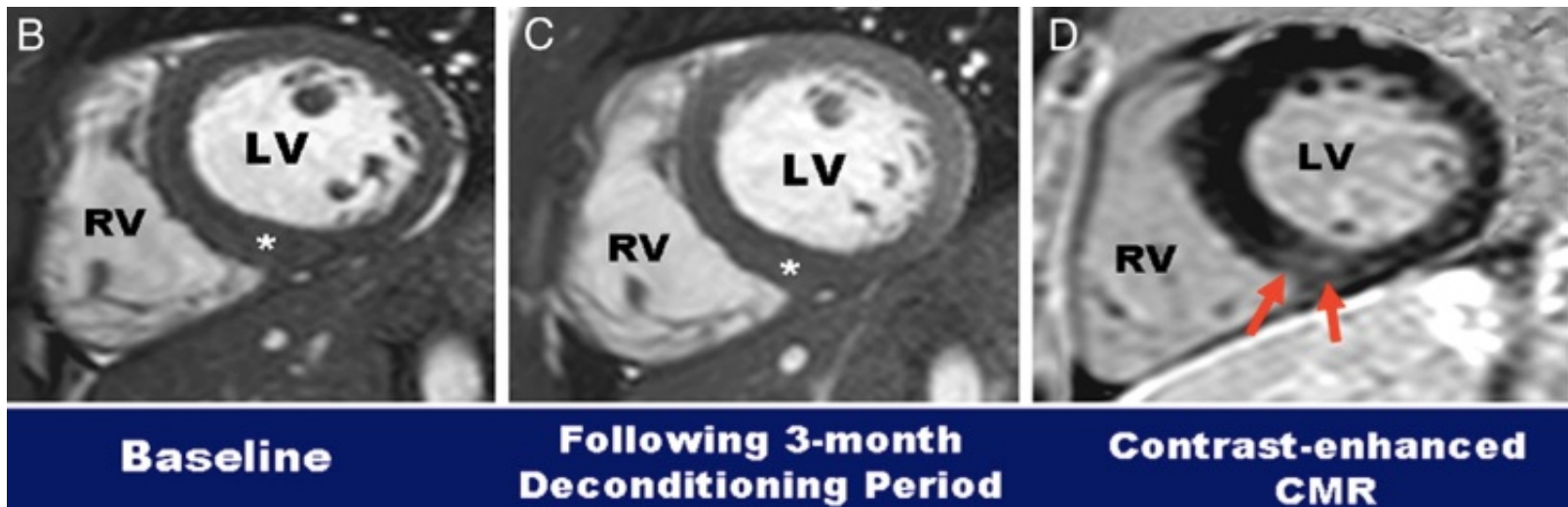
Better CV outcomes





Role of CMR

- Permits excellent visualization of the cardiac apex and lateral LV wall
- LGE helps differentiate LVH from HCM and adds prognostication value
 - The absence of LGE does not rule out HCM

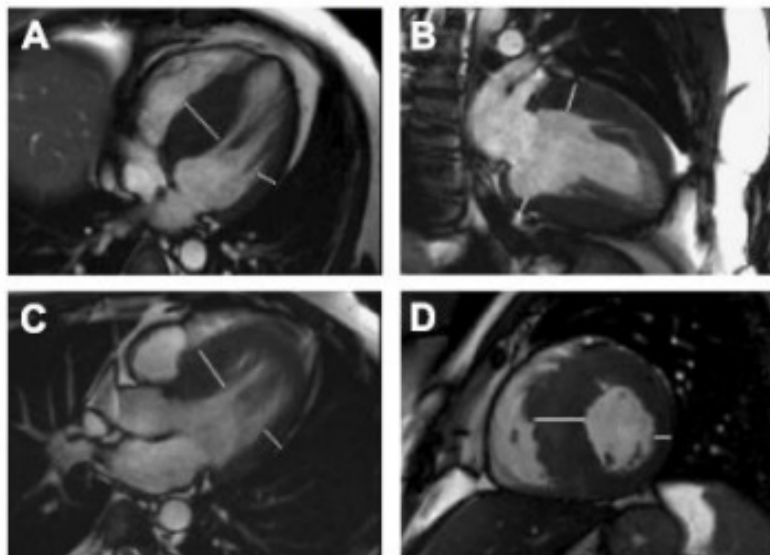




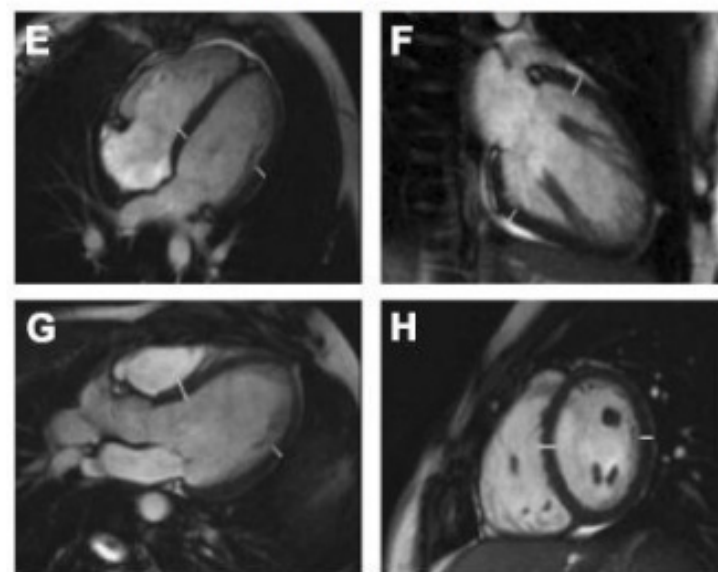
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Role of CMR

CMH



Athletes Heart



	Sens	Spec	PPV	NPV	AUC
Max Diastolic wall thickness <13 mm	40%	100%	100%	84%	0.955
diast Wtmax/ diast WT min <1.3	28%	95%	64%	95%	0.862
Diastolic WT/LVEDVI <0.15 mm.m2.ml	80%	99%	95%	94%	0.93

Petersen S, J Cardiovasc Magn Reson, 2005.

Deconditioning



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- Forced detraining may be a useful manoeuvre to resolve differential diagnosis between athlete's heart and HCM
- Reduction of LVT of 2-5 mm over 3 months
- Deconditioning requires:
 - (1) substantial compliance to interrupt the training of highly motivated athletes
 - (2) serial ECHO or CMR of technical quality suitable for comparison

Reduction in left ventricular wall thickness after deconditioning in highly trained Olympic athletes

Br Heart J 1993;69:125-128

Barry J Maron, Antonio Pelliccia, Antonio Spataro, Maristella Granata

No	Age*	Max LV thickness (mm)		LVED (mm)		LV mass (g)		LV Mass index (g/m ²)	
		Pk	De	Pk	De	Pk	De	Pk	De
1	19	15	11	56	54	330	220	153	102
2	22	14	10	57	58	288	232	129	103
3	23	13	11	60	60	331	295	153	137
4	26	15	10	56	54	330	206	162	101
5	26	13	11	57	58	288	264	134	124
6	29	13	10	61	59	323	224	145	101
Mean	24.5	13.8	10.5	57.8	57.1	315	240	146	111
SD	3.5	0.9	0.5	2.0	2.3	19.3	30.2	11.5	14.1
p		<0.005		NS		<0.01		<0.01	



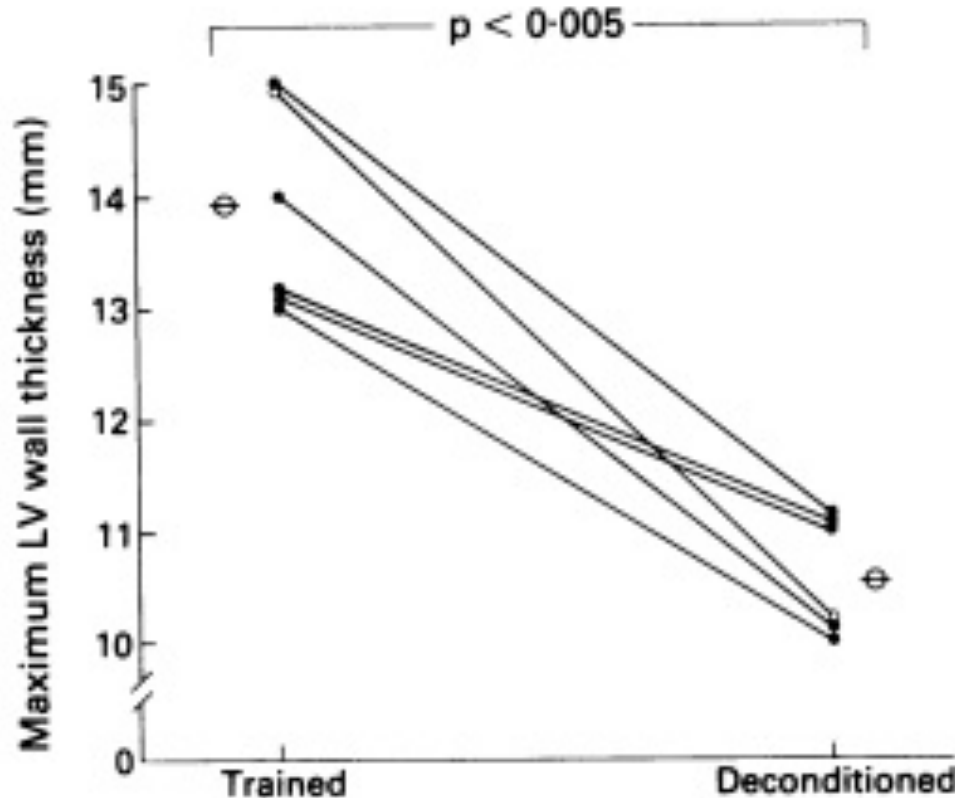
Reduction in left ventricular wall thickness after deconditioning in highly trained Olympic athletes

Br Heart J 1993;**69**:125–128

Barry J Maron, Antonio Pelliccia, Antonio Spataro, Maristella Granata



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LV thickness decreased by **15 to 33%** with deconditioning

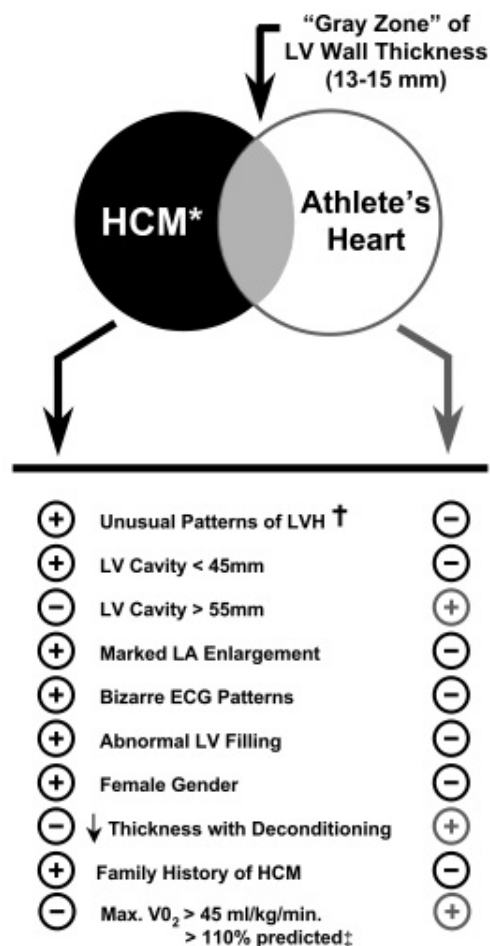


~20% of athletes will not show significant regression at 5 years

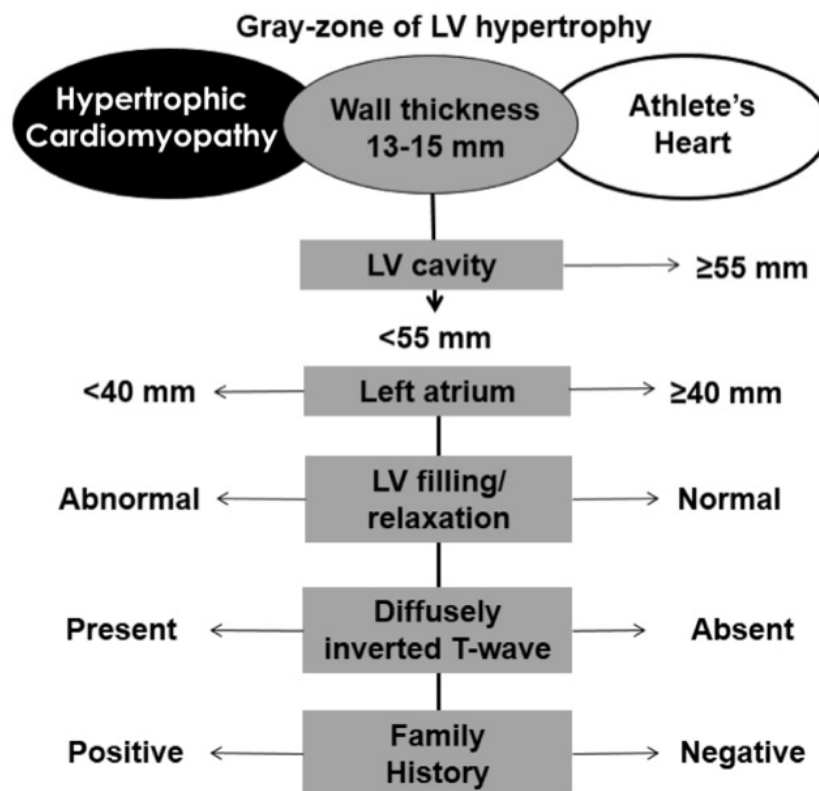
LVH second to anabolic steroids shows limited regression at 1 year



Refined 'grey zone'



Maron, Circ. 1995



Caselli, Am J Cardiol 2014

Consensus Paper



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European Heart Journal - Cardiovascular Imaging Advance Access published February 13, 2015



European Heart Journal – Cardiovascular Imaging
doi:10.1093/ehjci/jeu323

REVIEW

The multi-modality cardiac imaging approach to the Athlete's heart: an expert consensus of the European Association of Cardiovascular Imaging

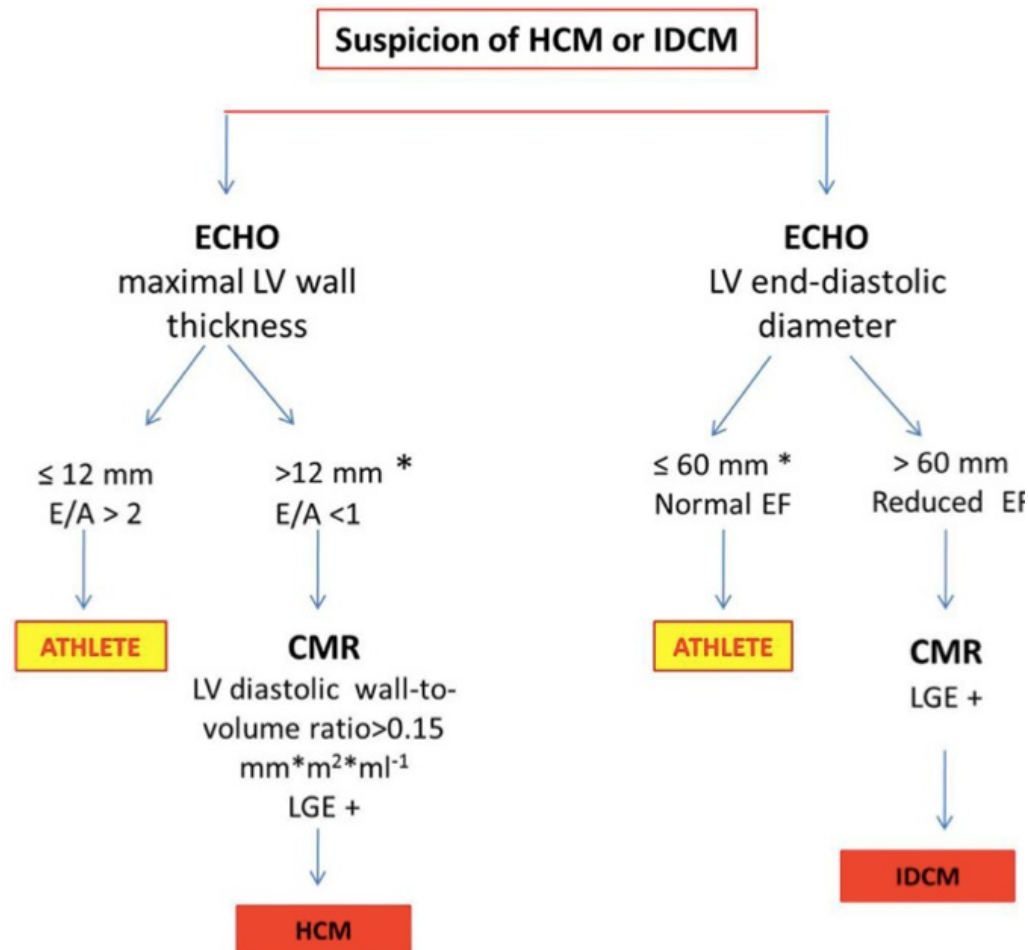
Maurizio Galderisi^{1*}, (Chair), Nuno Cardim², (Co-chair), Antonello D'Andrea³, Oliver Bruder⁴, Bernard Cosyns⁵, Laurent Davin⁶, Erwan Donal⁷, Thor Edvardsen⁸, Antonio Freitas⁹, Gilbert Habib¹⁰, Anastasia Kitsiou¹¹, Sven Plein¹², Steffen E. Petersen¹³, Bogdan A. Popescu¹⁴, Stephen Schroeder¹⁵, Christof Burgstahler¹⁶, and Patrizio Lancellotti¹⁷

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Proposed algorithm



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*persistent after deconditioning



Case Revisited

- ETT – no arrhythmia, blunted BP response, 18.7METS
- Handheld ECHO – walls and pap muscles look thick despite deconditioning since June 2015
- **PLAN:**
 - Repeat ECHO
 - CMR
 - ECHO for mother
 - Continue activity restriction and deconditioning