The Athlete's Heart

Critical Role of Echo

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Disclosures

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- **Abbott Vascular**
- **Boston Scientific**
- **Biotronic**
- **Edwards LifeScience**
- **Medtronic**
- **Sorin/ LivaNova**

For a full list, visit www.EchoCoreLab.org

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Massachusetts General Hospital

and

Ankit Shah, MD FACC FASE

Director, Sports & Performance Cardiology

MedStar Heart & Vascular Institute

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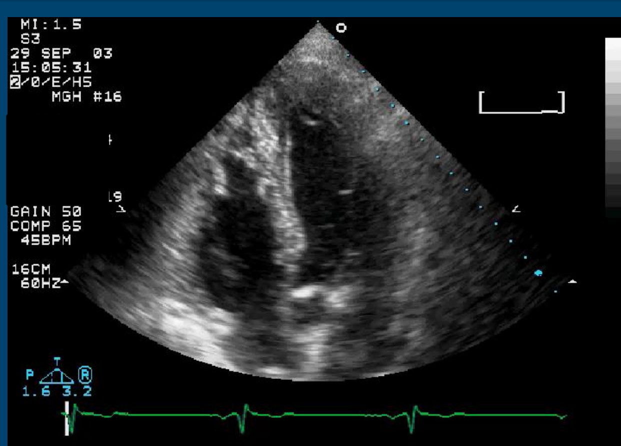

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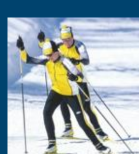
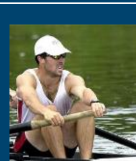
Outline

- Principles of Exercise-induced Cardiac Remodeling
  - Endurance vs. Strength training
- Healthy vs. Diseased (Athlete's heart vs. pathology)
  1. LV chamber enlargement
  2. RV chamber enlargement
  3. Aortic dilatation
  4. LV wall thickening (gray zone hypertrophy)
- Is there a role for Echo in Screening Athletes?
  - Identification and Prevention of Sudden Cardiac Death

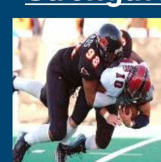
Background: Sport-Specific Physiology

Endurance Activities

Sustained ↑ CO
- 4 to 5 times rest
- ↑ ↑ ↑ HR & ↑ SV
- Vasodilation

Strength Activities




Repetitive↑ SBP
- Systolic BP > 200 mmHg
- Skeletal Muscle Contraction
- ↑ LV Afterload

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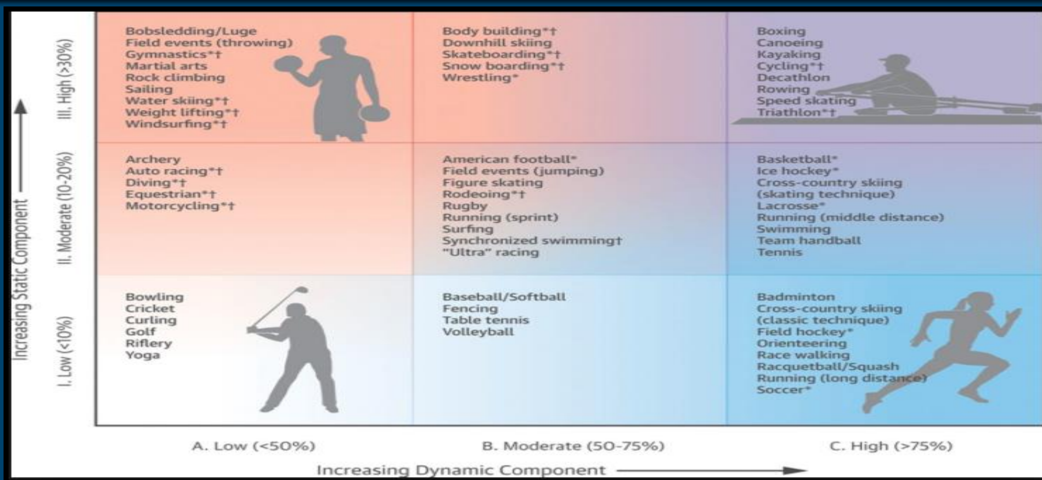
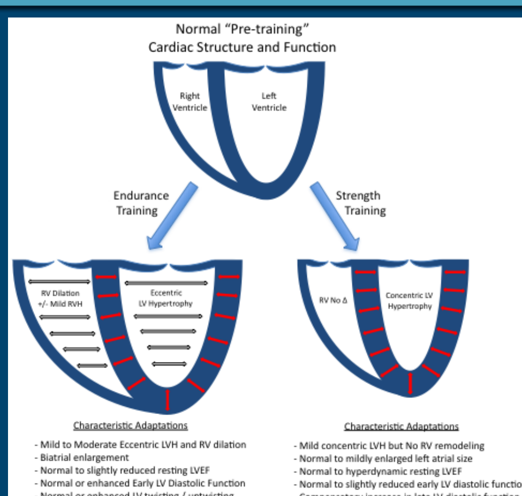
Sport Classification



Levine et al. Circulation. 2015; 131(22)

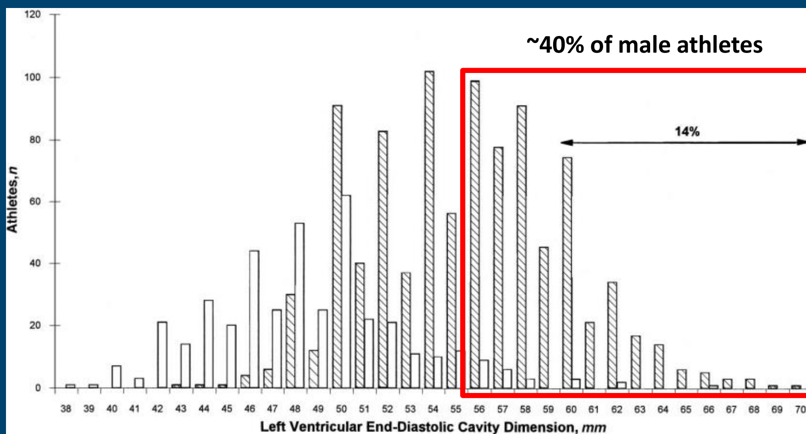
Exercise-Induced Cardiac Remodeling



Weiner & Baggish. *Prog Cardiovasc Dis* 2012;54:380.

Uncertainty #1: LV Dilatation

1309 Athletes in Diverse Sports (soccor, gymnastic, rowing)



Pelliccia et al. *Ann Intern Med* 1999;130:23.

LV Chamber Enlargement

Table 4 Echocardiographic findings from the study population of university athletes

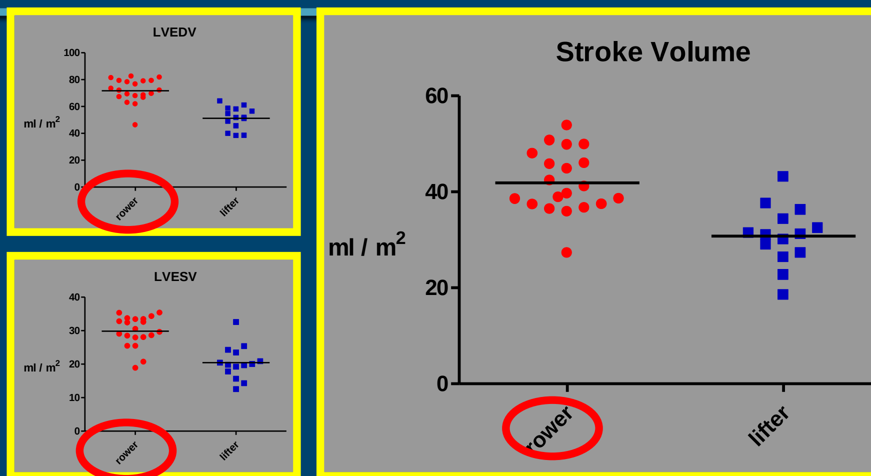
Parameter	Male (*n* = 300) Normal (*n* = 209)	Male (*n* = 300) Physiologic remodeling (*n* = 91)	Female (*n* = 197) Normal (*n* = 178)	Female (*n* = 197) Physiologic remodeling (*n* = 19)
Structural parameters				
Interventricular septal thickness (mm)	9.8 ± 0.9	11.6 ± 0.5	8.3 ± 0.7*	10.6 ± 0.5†
LV posterior wall thickness (mm)	10.0 ± 1.2	11.8 ± 1.4	8.6 ± 1.1*	10.7 ± 0.7†
LV inner dimension at end-diastole (mm)	51 ± 3	57 ± 5	42 ± 4*	54 ± 4†
LA diameter (mm)	36 ± 4	40 ± 4	32 ± 3*	38 ± 4
RV end-diastolic diameter (mm)	30 ± 5	36 ± 3	28 ± 4*	33 ± 3†
Functional parameters				
LV ejection fraction (%)	65 ± 7	58 ± 4	68 ± 6	64 ± 6†
Transmitral E wave (cm/sec)	86 ± 16	96 ± 13	81 ± 17	88 ± 12
Tra				
E′				
E′				
A′				
A′				

25% of US college athletes exceed gender recommended LVIDd limit

LA, Left atrial; *PW*, pulsed-wave.
Data are expressed as mean ± SD.
**P* < .05 for comparison with male athletes in the normal cardiac structure and function group.
†*P* < .05 for comparison with male athletes in the physiologic remodeling group.

Weiner et al. *J Am Soc Echocardiogr* 2012;25:568.

Olympic Athletes: LV Volumes



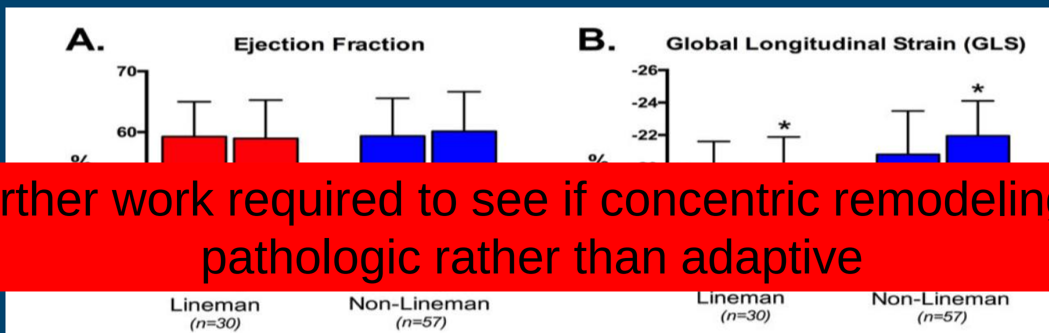
Med Sci Sports Exerc. 2010 Jun;42(6):1215-20

Table 2 LV characteristics in athletes and nonathletes

LV function	Athletes	Nonathletes
Morphology		
IVSd (mm)	8–13	6–10
LVIDd (mm)	49–65	42–59
LVM (g)	113–400	[illegible]
Volumes/EF		
LV EDV (mL)	130–240	[illegible]
LV EF (%)	45–70	>55
Tissue Doppler		
Sm (cm/sec)	6.5–14	>6
e′ (cm/sec)	7.5–16	>8
Mechanical parameter		
Strain/strain rate	Similar to nonathletes (GLS > −18%)	GLS > -18%

EDV, End-diastolic volume; *EF*, ejection fraction; *GLS*, global longitudinal strain; *IVSd*, interventricular septal thickness at diastole; LVIDd, LV internal diameter at diastole; LVM, LV mass; *Sm*, tissue Doppler imaging peak velocity at systole.
Data from Paterick TE, Jan MF, Paterick ZR, Umland MM, Kramer C, Lake P, et al. Cardiac evaluation of collegiate student athletes: a medical and legal perspective. Am J Med 2012;125:742–752.

Is Concentric Remodeling Adaptive?



Further work required to see if concentric remodeling is pathologic rather than adaptive

Figure 2. Left Ventricular Systolic Function Among ASF Participants
Left ventricular systolic function as measured by ejection fraction (A) and global longitudinal strain (B) stratified by field position before and after 90 days of American Style Football participation. * = $p<0.05$ compared to preseason values.

Lin J, et al. JACC Cardiovasc Imaging. 2016 Dec;9 (12): 1367-1376

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LV Adaptation in Endurance Athletes

- ***Physiologic:***
  - Expected with endurance training.
  - Accompanied by proportionate increase in LV mass (Eccentric LVH).
  - Accompanied by normal to low normal resting LVEF (~50%).
    - TDI / Strain assessment with preserved or enhanced function.
  - Usually accompanied by "other" chamber enlargement (RV, LA).
  - LVIDd absolute "cut-offs" are not helpful.
  - When in doubt, exercise testing is very useful (confirm LV augmentation and document supranormal exercise capacity).

Uncertainty #2: RV Chamber Enlargement/Function

102 Endurance Athletes from the UK

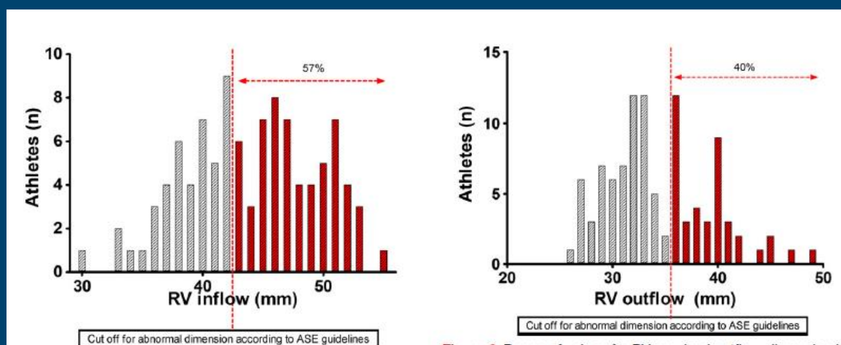
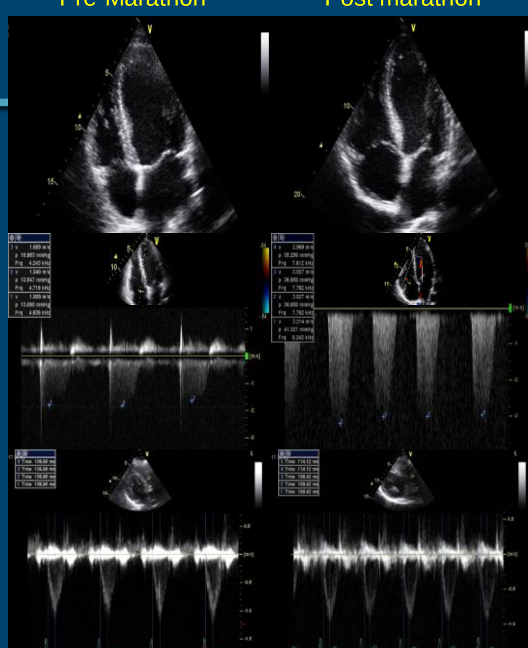


Figure 2 Range of values for RV inflow dimension in endurance athletes (n = 102).

Figure 3 Range of values for RV proximal outflow dimension in endurance athletes (n = 102).

Oxborough et al. *J Am Soc Echocardiogr* 2012;25:263.

Pre-Marathon Post marathon



Neilan, Circulation 2006

RV Function-Olympic Speedskaters

	Baseline	Post-exertion	*P* Value
Ea (cm/sec)	13.5±3.6	15.2±5.8	0.041
Aa (cm/sec)	8.6±1.5	9.2±3.0	0.47
RV Area change	0.35 ± 0.13	0.43 ± 0.13	0.007
Strain Apex (%)	-30±8	-29±7	0.66
SR Apex (s^{-1})	-1.8±0.5	-2.5±1.2	0.038

Poh KK, Int J Cardiol 2008

Right Ventricular Remodeling in Elite Athletes

37
30

Table 4. Upper Reference Values for Right Ventricular Measurements in Athletes, Corrected for Sex and Body Surface Area

	Male Athletes	Female Athletes
RAA, cm2 (cm2/m2)	28 (14)	24 (13)
RVEDA, cm2 (cm2/m2)	39 (19)	32 (18)
RVOTP, mm (mm/m2)	40 (20)	37 (21)
RVOT1, mm (mm/m2)	43 (22)	40 (23)
RVOT2, mm (mm/m2)	32 (17)	29 (16)
RVD1, mm (mm/m2)	55 (28)	49 (28)
RVD2, mm (mm/m2)	47 (24)	43 (25)
RVD3, mm (mm/m2)	109 (56)	100 (57)
RVWT, mm (mm/m2)	6 (3)	5 (3)

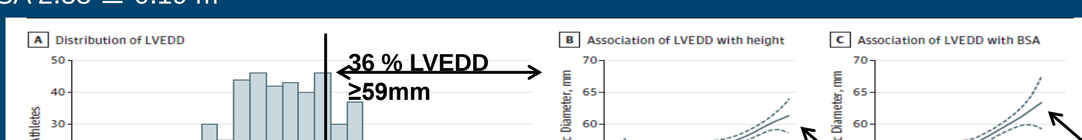
Zaidi A et al. Circulation 2013; 127: 1783-1792

RV Adaptations to training

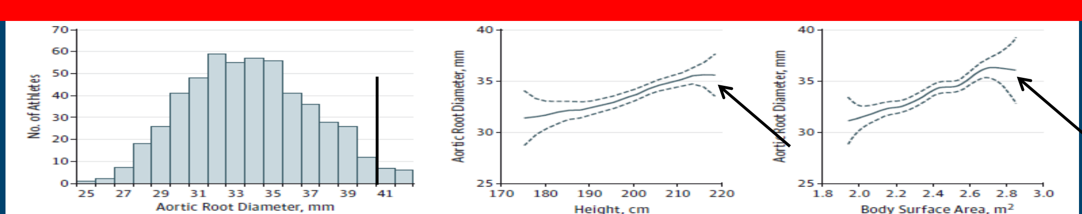
- ***Physiologic:***
  - RV enlargement expected with endurance training.
    - Global RV process without sacculation, aneurysmal dilation, segmental dysfunction, or fibrosis.
  - RV dimensions absolute “cut-offs” are not usually helpful.
  - Almost always associated with LV remodeling (concomitant LV enlargement but no RVH).
  - May be accompanied by normal to low normal resting FAC / RVEF.
    - TDI / Strain assessment should be preserved or enhanced function.
    - If in doubt, comprehensive exercise testing
    - RV demonstrates contractile reserve

Uncertainty #3: Aorta’s in Athletes

526 NBA basketball players
BSA 2.38 ± 0.19 m²



Aortic root ≥40mm in 24 (4.6%) but never >42mm



Engel DJ, Schwartz A, Homma S, JAMA Cardiol. 2016;1(1):80-87

Aortic Root Size by Sport

Table 4. Aortic Root Size According to Mitchell's Sport Classification in Males

Male	IA (n=117)		IB (n=102)		IC (n=386)		IIA (n=39)		IIB (n=222)		IIC (n=369)		IIIA (n=306)		IIIB (n=83)		IIIC (n=415)	
	Mean	P95	Mean	P95	Mean	P95	Mean	P95	Mean	P95	Mean	P95	Mean	P95	Mean	P95	Mean	P95
Aortic M mode, mm	30±3.1	36.8	29.8±3.3	35.2	29.9±2.8	34.6	29.9±3.7	36.4	29.8±3.1	36	31.6±3.7	38.9	29.5±3	35	30.1±3.3	36.1	30.6±3.1	35.9
Aortic annulus, mm	25.2±3	30.5	23.7±2.9	29.1	25.4±3	30.3	25.2±3.2	31.4	24.9±3.3	31.1	26.6±3.5	32.7	24.6±2.8	28.9	24.7±2.8	28.8	26±3.1	31.4
Sinuses of Valsalva, mm	31.3±3.4	38.9	30.6±3.6	37	31.3±3.1	36.4	31.4±3.9	37.9	31.4±3.8	38.2	32.9±3.8	39.9	30.7±3.3	36.6	31±3.1	36.5	32±3.4	37.9
Sinotubular junction, mm	26.4±3.3	32.1	25.3±3.3	30.3	26.2±2.9	31.3	26.1±3.4	31.7	26±3.5	32	27.5±3.6	33.5	25.5±3	30.7	26±3	30.7	27±3.3	32.8
Proximal ascending aorta, mm	[illegible]	[illegible]	[illegible]	[illegible]	[illegible]	[illegible]	[illegible]	[illegible]	[illegible]	[illegible]	[illegible]	[illegible]	[illegible]	[illegible]	[illegible]	[illegible]	[illegible]	[illegible]

Aortic root was larger in sports with high dynamic component in both sexes but, age, left ventricular mass and BSA were main predictors of aortic dimensions

Female	IA (n=75)		IB (n=81)		IC (n=225)		IIA (n=20)		IIB (n=121)		IIC (n=208)		IIIA (n=285)		IIIB (n=64)		IIIC (n=163)	
	Mean	P95	Mean	P95	Mean	P95	Mean	P95	Mean	P95	Mean	P95	Mean	P95	Mean	P95	Mean	P95
Aortic M mode, mm	25.5±2.5	30.2	26.5±2.6	30.2	25.5±2.4	29.8	25.2±2.7	30.9	25.8±2.5	30.2	26.9±2.8	31.8	25.1±2.5	29.8	25.4±2.2	29.6	26.2±2.2	29.8
Aortic annulus, mm	21±2.7	25.4	21.6±2.7	26	21.4±2.5	26.1	22±3.2	27.7	21.4±2.5	26.1	22.7±2.7	27.4	21±2.4	25.2	21.2±2.5	26.2	21.9±2.9	27.3
Sinuses of Valsalva, mm	26.2±2.4	30.3	27.7±3.2	33.2	26.9±2.7	31.4	26.4±2.5	30.9	27±2.8	32	28.4±2.9	33.1	26.5±2.6	30.9	26.8±2.8	32.8	27.6±2.9	32.1
Sinotubular junction, mm	22.7±2.6	26.9	23.5±3	28.6	22.8±2.8	27	22.2±2.6	26.7	22.9±2.6	27.5	24.1±2.5	28.4	22.4±2.6	26.7	23±2.7	27.4	23.2±2.8	28.5
Proximal ascending aorta, mm	22.9±3.1	28.4	22.8±3.1	29.2	23.2±3.1	28.2	22.2±2.9	28	23.4±2.9	28	24.8±3	29.7	22.9±2.9	27.4	23.3±3.1	28.1	23.8±3.2	28.8
Aortic M mode/BSA, mm/m²	15.5±1.5	18.1	15±1.6	18.4	15.8±1.7	18.7	14.9±1.4	17.9	15.2±1.5	17.7	15.2±1.6	17.8	15.4±1.6	18.3	15.6±1.4	17.8	15.8±1.5	18.6
Aortic annulus/BSA, mm/m²	12.9±2.1	16.7	12.3±1.4	15.3	13.3±1.8	16.4	13±1.7	15.4	12.7±1.6	15.6	12.9±1.6	15.4	12.9±1.6	15.7	13.1±1.6	15.8	13.2±1.9	16.7
Sinuses of Valsalva/BSA, mm/m²	16±1.8	19.2	15.7±1.9	19.4	16.7±2	20.1	15.6±1.5	18.9	16±1.7	18.9	16.1±1.7	19	16.2±1.9	19	16.5±1.7	19.2	16.6±2.1	20.6
Sinotubular junction/BSA, mm/m²	13.9±1.9	16.9	13.4±1.7	16.3	14.1±2	17.5	13.2±1.3	14.9	13.5±1.6	16.2	13.7±1.5	16.2	13.7±1.7	16.6	14.2±1.7	16.8	14±1.9	18
Proximal ascending aorta/BSA, mm/m²	14±2.1	18.3	13.5±1.8	17.1	14.4±2.1	17.8	13.1±1.6	17.1	13.8±1.7	16.4	14.1±1.8	17	14±1.9	16.9	14.4±2	18.4	14.3±2.2	18.2

Boraita et al. Circ Cardiovasc Imaging. 2016;9

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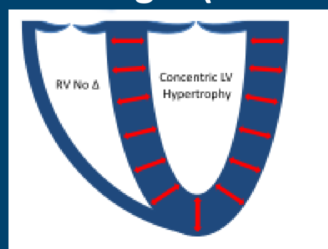
Aortic Root In Athletes

- Aortic root does not have same physiologic adaptation to training as other cardiac structures
- Aortic Root in healthy elite athletes is within established limits for the general population
- Marked dilation of the aortic root is not explained by height, BSA or training effect
- Aortic root size in lifelong endurance masters athletes has not been studied

Uncertainty #4: Thick LV Walls

Least frequent but most problematic issue.
Expected with strength (isometric) training.



Gray Zone LVH: 13 – 15 mm
Challenge: distinguish EICR from HCM
Especially since HCM is leading cause of exercise-related sudden death

Thick LV Walls

MGH 1811 Cardiovascular Performance Program

Table 4 Echocardiographic findings from the study population of university athletes

Parameter	Male (n = 300) Normal (n = 209)	Male (n = 300) Physiologic remodeling (n = 91)	Female (n = 197) Normal (n = 178)	Female (n = 197) Physiologic remodeling (n = 19)
Structural parameters				
Interventricular septal thickness (mm)	9.8 ± 0.9	11.6 ± 0.5	8.3 ± 0.7*	10.6 ± 0.5†
LV posterior wall thickness (mm)	10.0 ± 1.2	11.8 ± 1.4	8.6 ± 1.1*	10.7 ± 0.7†
LV inner dimension at end-diastole (mm)	51 ± 3	57 ± 5	42 ± 4	54 ± 4†
LA diameter (mm)	36 ± 4	40 ± 4	32 ± 3*	38 ± 4
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Transmitral E wave (cm/sec)	86 ± 16	96 ± 13	81 ± 17	88 ± 12
Tra[illegible]	[illegible]	[illegible]	[illegible]	[illegible]
E′ [illegible]	[illegible]	[illegible]	[illegible]	[illegible]
E′ [illegible]	[illegible]	[illegible]	[illegible]	[illegible]
A′ [illegible]	[illegible]	[illegible]	[illegible]	[illegible]
A′ [illegible]	[illegible]	[illegible]	[illegible]	[illegible]

LA, Left atrial; *PW*, pulsed-wave.
Data are expressed as mean ± SD.
*$P < .05$ for comparison with male athletes in the normal cardiac structure and function group.
†$P < .05$ for comparison with male athletes in the physiologic remodeling group.

Not a single healthy college athlete with walls > 14 mm

Weiner et al. *J Am Soc Echocardiogr* 2012;25:568.

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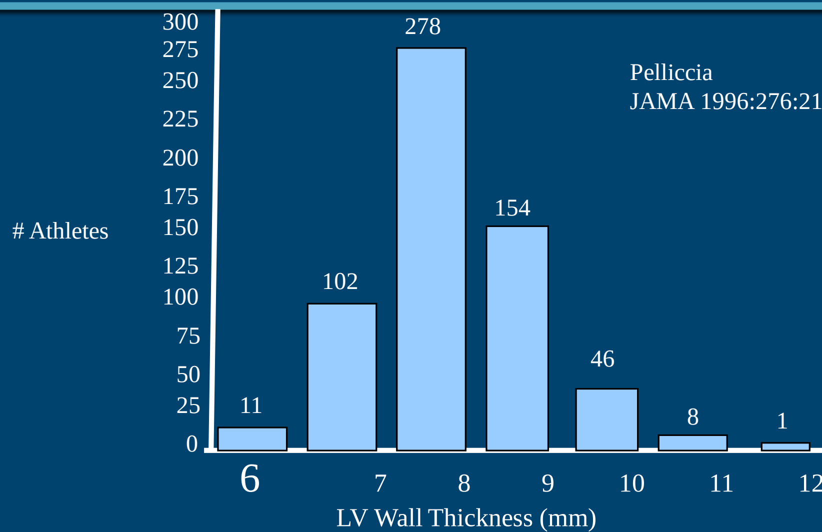
LV Wall Thickness in 600 Female Athletes

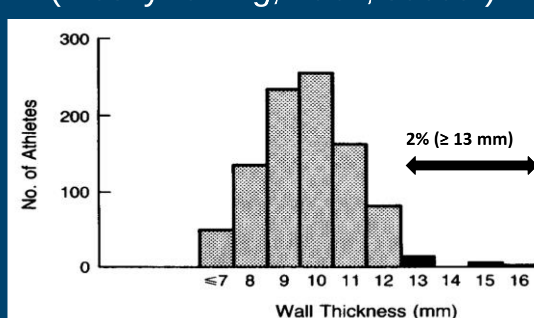


Pelliccia
JAMA 1996:276:211

Thick LV Walls

Adult Athletes

(mostly rowing, track, soccor)



Pelliccia et al. *N Engl J Med* 1991.

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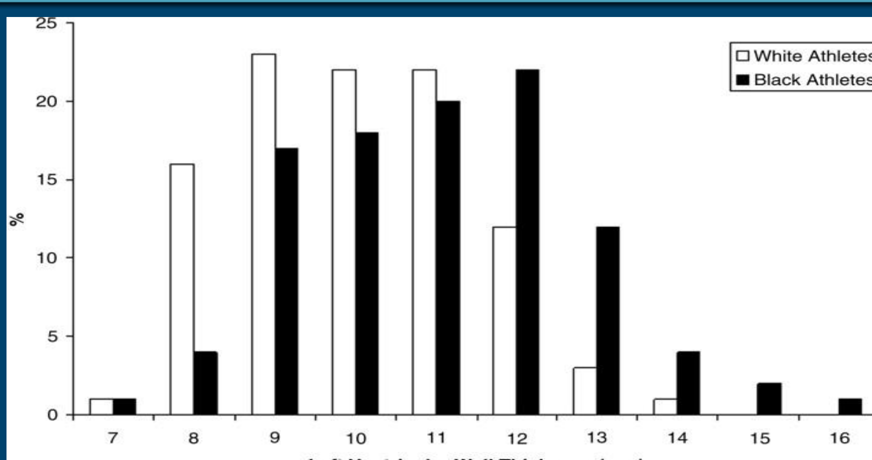
Racial Differences in LV Remodeling in Highly Trained Athletes

- **300 Nationally Ranked Black Athletes compared to 300 Matched White Athletes and 150 B & W Sedentary people**
- **Blacks Athletes had Greater LV Thickness and Cavity Size**
  - **16% BA and 4% WA had wall thickness > 12 mm**
  - **3% BA and 0% WA had wall thickness >15mm**
- **BA with LVH had enlarged LVs and normal diastolic function**

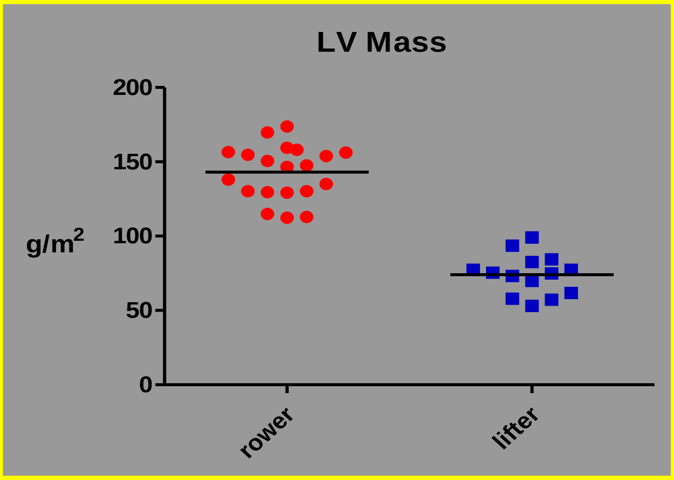
Basavarajaiah JACC 2008;51:2256-62

Racial Differences in LV Remodeling in Highly Trained Athletes



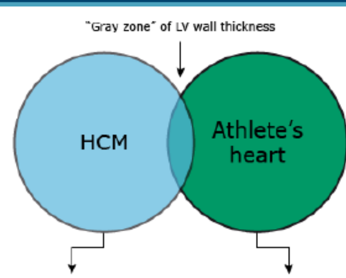
Basavarajaiah JACC 2008;51:2256-62

Olympic Athletes: LV Mass



Baggish / Wood, 2008

Pathologic LVH (HCM) vs Physiologic LVH (Athletic Heart)

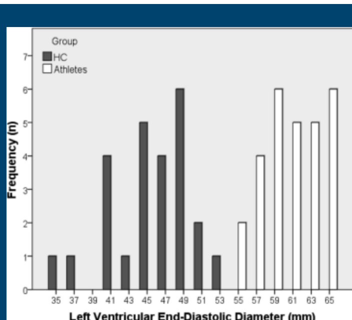


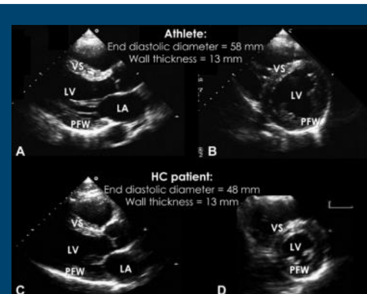
HCM		Athlete's heart
+	Focal LVH pattern	0
+	LV cavity <45 mm	0
0	LV cavity >55 mm	+
+	LA enlargement	0
+	Bizarre ECG patterns	+
+	Abnormal LV filling	0
+	Female gender	0
+	Family history of HCM	0
+	Thickness with deconditioning	0
0	VO_2 ↑ >110 percent	+
+	Late gadolinium enhancement	0
+	Pathogenic sarcomere mutation	0

Maron et al, Circz 2006;114:1633

LV Cavity Size

28 athletes without CV disease and 25 untrained patients with HCM (matched for LV wall thickness 13 – 15 mm)





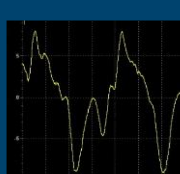
LVIDd <54 mm differentiated HCM and Athlete's heart

Caselli et al. *Am J Cardiol* 2014;114:1382-89.

Other Distinguishing Features

Tissue Doppler (Diastolic Function)

E' (septum) <11.5 cm/s (sens 81%, spec 61% for dx HCM)

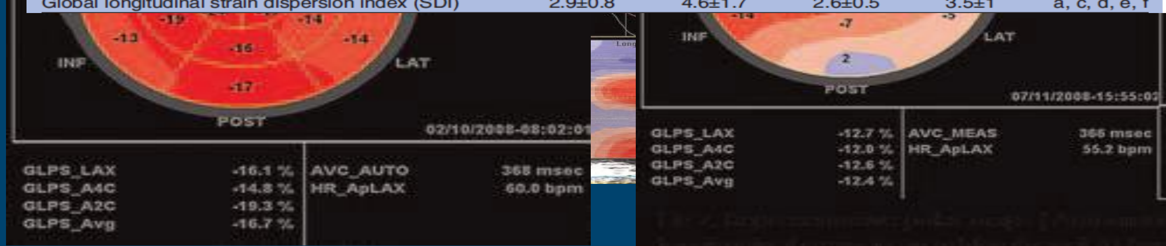


Caselli et al. *Am J Cardiol* 2014;114:1382-89.

Two-dimensional speckle tracking echocardiography

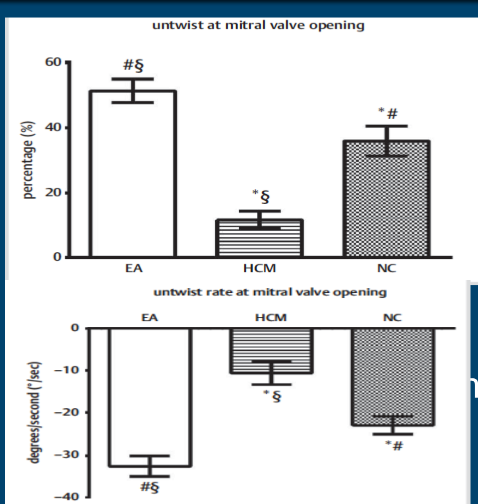
Table 2 Comparison of longitudinal strain and strain dispersion in the overall study population

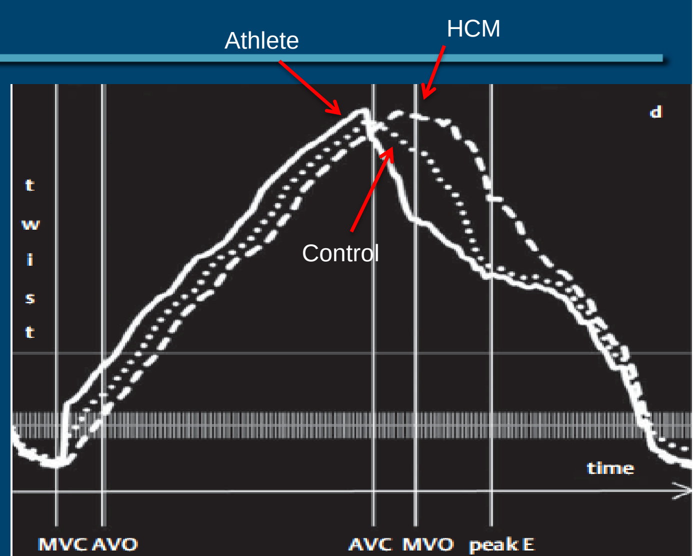
Variable	Controls N=12	HCM N=56	AT-LVH* N=27	H-LVH† N=34	p Value‡
Segmental average longitudinal strain (%)					
Basal	−18.4±2.4	−8.2±5	−16.3±2.4	−15.3±2.2	a, c, e, f
Mid-LV	−19±2	−9.2±4.8	−17.8±1.9	−17.1±3	a, e, f
Apical	−19.2±3.3	−12.3±9	−21.1±3.5	−22.1±4.9	a, e, f
Global LV longitudinal strain (%)					
LAX	−17.6±2.6	−11.2±5	−17.1±2.9	−17.7±3.2	a, e, f
4C	−18.4±1.6	−11.2±4.2	−17.3±2.5	−17.3±3.8	a, e, f
2C	−19.9±2.7	−11.1±4.2	−19±2.3	−18.5±4.2	a, e, f
Global LV longitudinal strain average (GLS avg,%)	−18.7±1.8	−11.2±4.2	−17.8±2.2	−17.8±3.1	a, e, f
Global longitudinal strain dispersion index (SDI)	2.9±0.8	4.6±1.7	2.6±0.5	3.5±1	a, c, d, e, f



Afonso L et al. BMJ Open 2012: 2:4.

Left Ventricular Untwisting





Kovacs A et al. Int J Sports Med 2014: 35: 259-264.

Left Atrial Size and Function

Echocardiographic Characteristics

	Controls (20)	Athletes (20)	HCM (20)	P (ANOVA)
DD (mm)	51 ± 4	62 ± 5*	58 ± 5*	<0.01
SD (mm)	37 ± 3	44 ± 4	39 ± 4	NS
SW (mm)	9.2 ± 1	15 ± 2*	18 ± 3*	<0.01
LVMI (g/m^2)	78 ± 6	143 ± 12*	157 ± 15*	<0.01
LVEF (%)	60 ± 5	57 ± 5	59 ± 5	NS
LV GLS (%)	−20 ± 2	−18 ± 2	−13 ± 3**	0.02
LV VTI (cm)	22 ± 3	27 ± 3	19 ± 4	NS
LA volume (mL/m^2)	25 ± 2	40 ± 5*	45 ± 6*	<0.01
LA area (cm^2)	18 ± 3	29 ± 3*	31 ± 4*	<0.01
LA diameter (mm)	35 ± 4	49 ± 4*	49 ± 4*	<0.01
LA emptying fraction	0.59 ± 0.12	0.61 ± 0.15	0.42 ± 0.08**	<0.01
E-wave (cm/sec)	55 ± 4	65 ± 5*	69 ± 5*	0.04
A-wave (cm/sec)	40 ± 3	37 ± 3	88 ± 5**	<0.01
E/A ratio	1.3 ± 0.4	1.7 ± 0.4	0.78 ± 0.2**	0.02
DT (msec)	188 ± 11	178 ± 15	190 ± 15	NS
é (cm/sec)	13.5 ± 2	14.4 ± 2	5.5 ± 1**	<0.01
E/é ratio	4.1 ± 1	5.2 ± 1	12.8 ± 2**	<0.01

*P < 0.05 versus controls after significant ANOVA, **P < 0.05 versus other groups after significant ANOVA.
DD = diastolic diameter; SD = systolic diameter; SW = septal wall; LVMI = left ventricular mass index; LVEF = left ventricular ejection fraction; LV GLS = left ventricular global longitudinal strain; LV VTI = left ventricular volume-time integral; LA = left atrium; E/A = mitral inflow waves ratio; é = mean mitral annulus tissue Doppler; NS = not significant.

Gabrielli et al. Echocardiography 2012;29:943-949.

Left Atrial Size and Function

Pathologic LVH leads to left atrial dilatation with LA dysfunction

In contrast, this study provides more support that left atrial dilatation as a result of endurance training is an adaptive and healthy physiologic response

Peak LA Strain during LV Systole
Marker of LA reservoir function

Peak negative LA strain rate during LA contraction
Marker of LA contractile function

Gabrielli et al. Echocardiography 2012;29:943-949.

Thick LV Walls

- ***Physiologic:***
  - Physiologic concentric LVH is symmetric without regional variation.
    - Marked asymmetry is pathology until proven otherwise.
  - Wall thickness "cut-offs" are VERY helpful.
    - Accurate absolute thicknesses >15 mm are pathologic until proven otherwise.
  - E' values may be helpful, but not diagnostic
  - Exercise testing may be useful discriminator (rule out other causes of LVH, i.e. hypertensive BP response)
  - GLS and rate of untwisting may be helpful
  - Detraining may be necessary to arrive at a final diagnosis.

Outline

- Principles of Exercise-induced Cardiac Remodeling
  - Endurance vs. Strength training
- Healthy vs. Diseased (Athlete's heart vs. pathology)
  1. LV chamber enlargement
  2. RV chamber enlargement
  3. Aortic Dilatation
  4. LV wall thickening (gray zone hypertrophy)
- **Is there a role for Echo in Screening Athletes?**
  - **Identification and Prevention of Sudden Cardiac Death**

Causes of Sudden Cardiac Death in Athletes

Most Common:
- Hypertrophic CMP
- Anomalous origin coronary artery

Less Common:
- Aortic Dilatation in Marfan
- Myocarditis

Uncommon:
- Arrhythmogenic RV Cardiomyopathy
- Atherosclerotic CAD
- Aortic Valve Stenosis

Utility of Screening Echo

- **Incidence of SCD during sports varies from <1/100,000 athletes* to 2/100,000#**
- **In 2688 competitive athletes, 203 (7.5%) of echos were abnormal**
  - **Only in 4 athletes did it stop athletic activity (HCM mostly)**
- **NO consensus on what type of echo to perform (handheld, limited, full, etc)**
- **Cost effectiveness is determined by**
  1) **incidence of SCD related to sports practice**
  2) **Cost of the echo**
  3) **Years of potential life saved**

  All of the above are either unknown or highly variable

*Corrado et al, JAMA 2006;296:1593
Steubvuk et al, JACC 2011;57:1291

Conclusions

1) Exercise training is a potent stimuli for cardiac remodeling and contributes to the development of "athlete's heart" morphology.
2) Understand the principles of exercise-induced cardiac remodeling
3) The nature and magnitude of cardiac remodeling depends upon sporting discipline, gender, race, level of and duration of training (Endurance vs. Strength).
4) Echocardiographic techniques can help differentiate healthy adaptation from underlying pathology
5) Echo can identify causes of SCD that are not caught with a screening ECG but the yield is still low and the cost-effectiveness is unknown