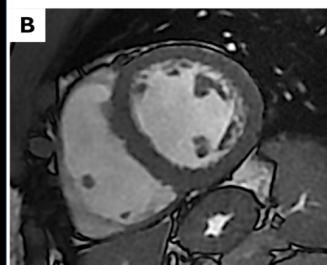
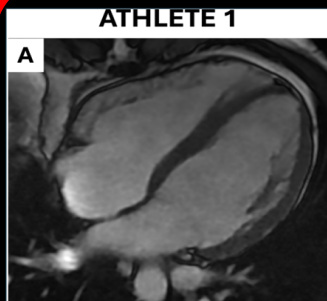


IL FENOTIPO IPERTROFICO

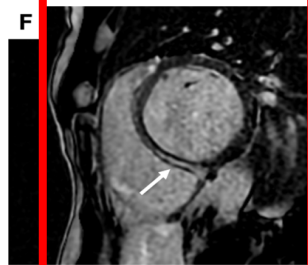
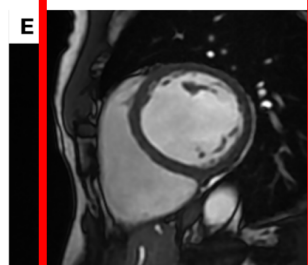
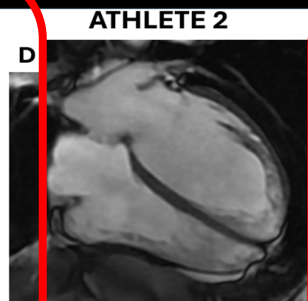
Ipertrofia, pseudoipertrofia e cuore d'atleta



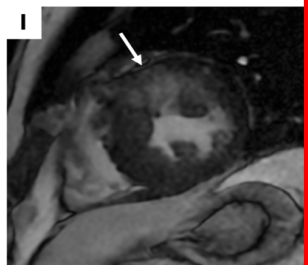
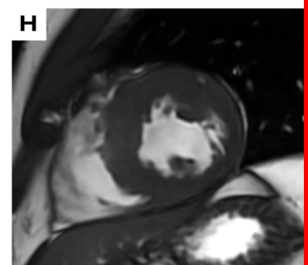
Antonio Pelliccia



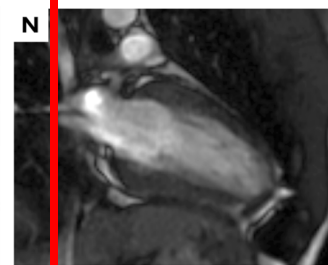
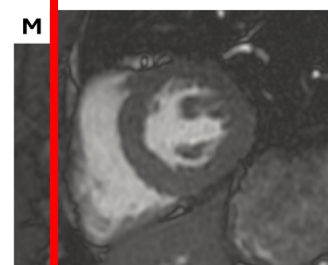
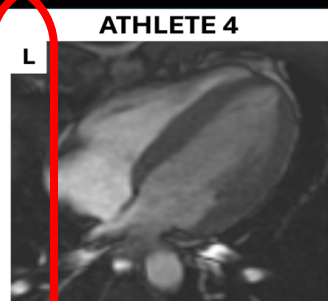
Athlete's Heart



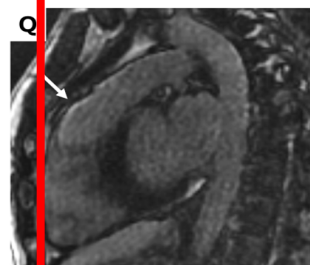
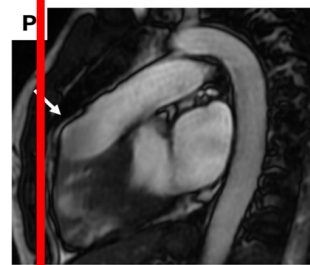
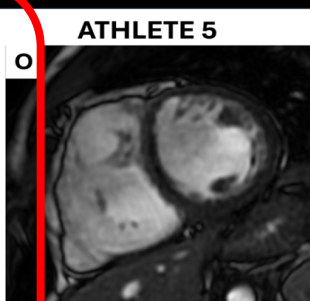
DCM



HCM



«Gray-zone»



ARVC

Impact of sport on left ventricular
remodelling



100 – 200 mt



400 mt



800 – 1.500 – 3.000 mt



5 km, 10 km, Marathon

POWER

ENDURANCE

N = 140



46 26 20

34 14 20

25 13 12

35 20 15

BSA



1.83 ± 0.20



1.76 ± 0.18



1.66 ± 0.18



1.63 ± 0.17



LVEDDi, mm/m²
LVEDVi, mL/m²
LVMI, gr/m²

27.3 ± 2
 59 ± 13.1
 86.3 ± 16.2



28.9 ± 1.8
 66.6 ± 19.1
 88.1 ± 14.3



31.4 ± 2.6
 75.6 ± 15.1
 108.9 ± 16.1



32.5 ± 2.3
 81.5 ± 16.3
 113.8 ± 19.2



HR, bpm

72.3 ± 12.3



69.3 ± 12



56.5 ± 16.5



55.7 ± 11.2



Watt/kg



2.86 ± 0.6



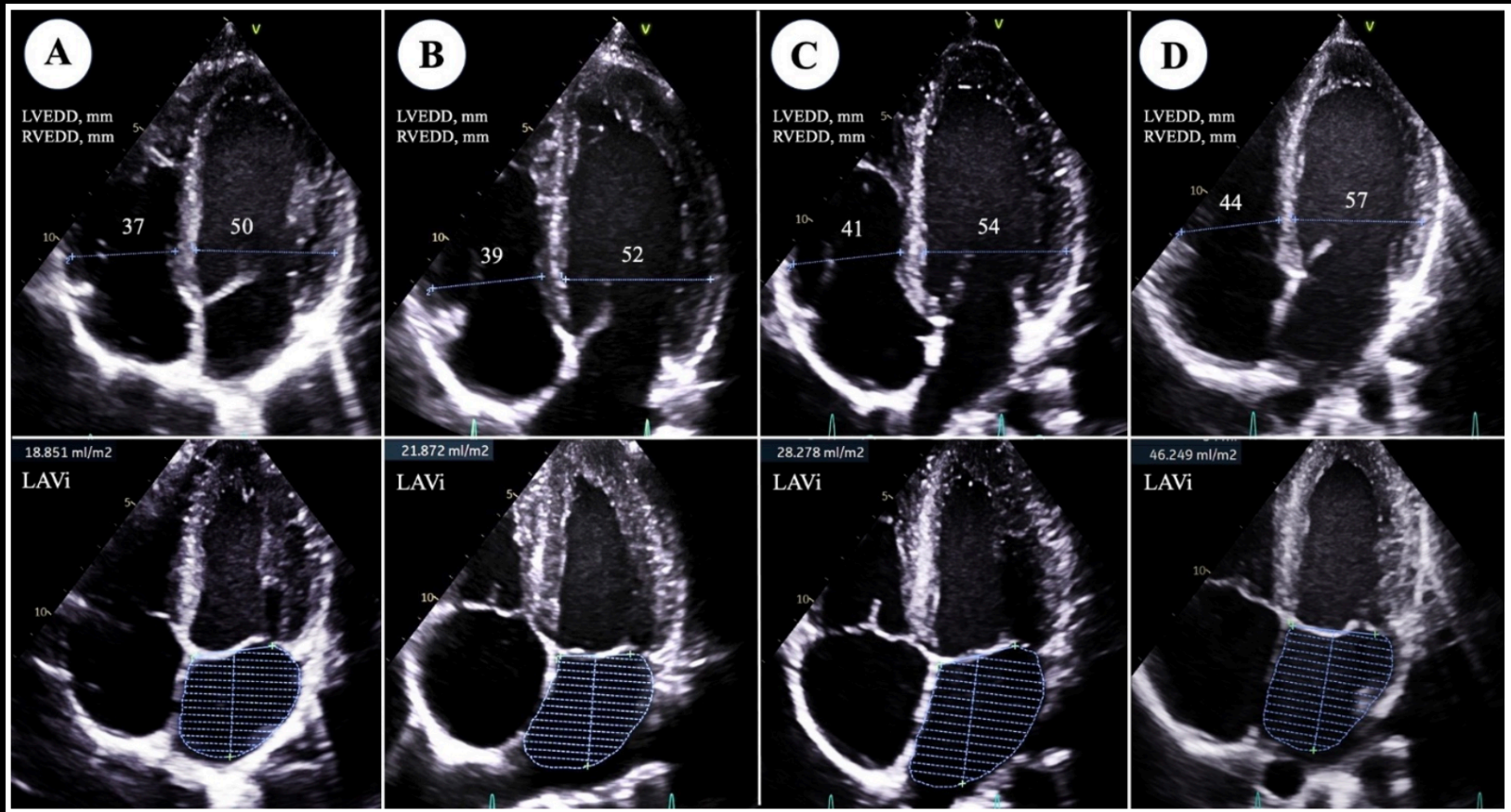
3.50 ± 0.3



4.10 ± 0.6



4.41 ± 0.7



100 – 200 mt



400 mt



800 – 1.500 – 3.000 mt

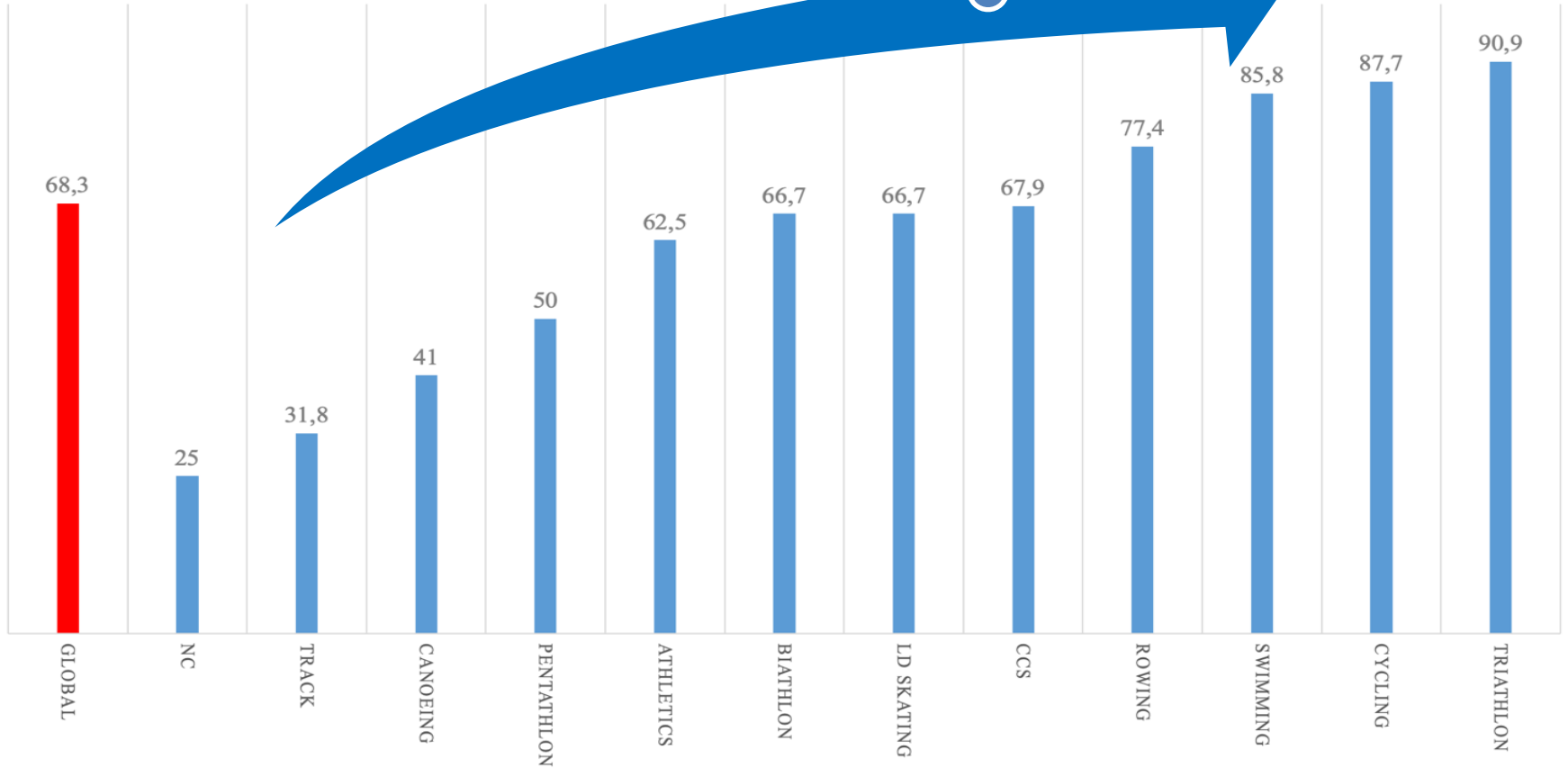
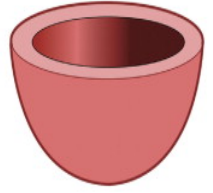


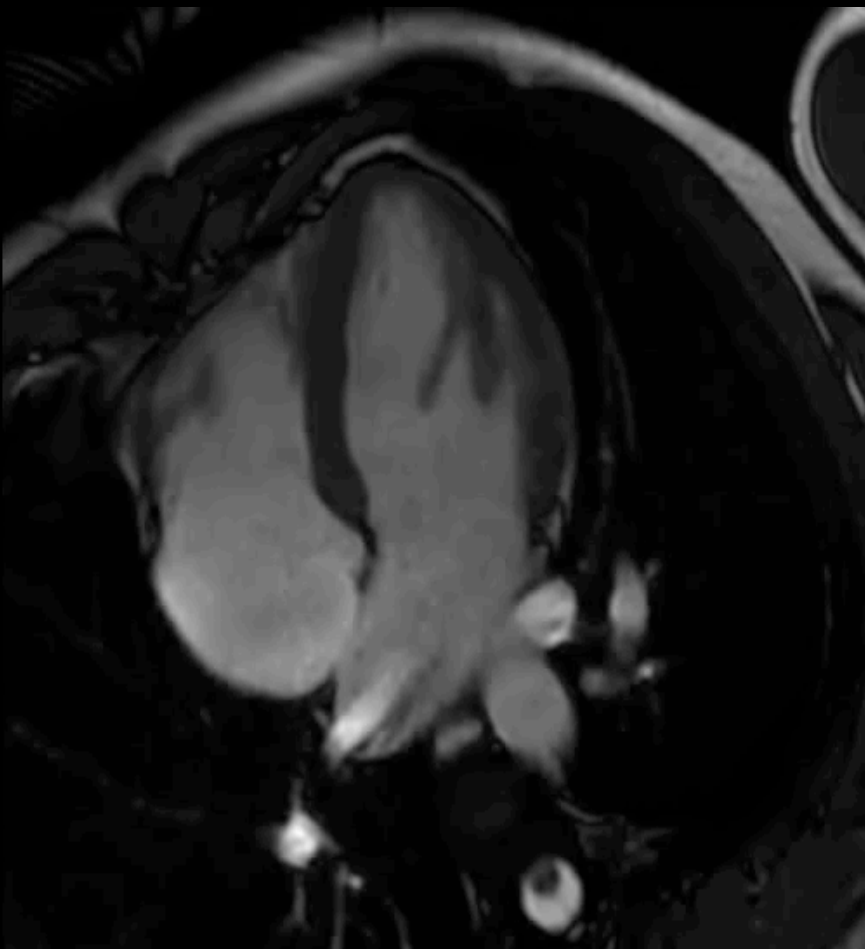
5 km, 10 km, Marathon



Normal
geometry

Eccentric
Remodeling





Physiologic remodeling with preserved
LV systolic function



The usual model:

Young age

Large body size

Endurance sport

LV cavity: 64 mm

LV Volume 208 ml

ECCENTRIC CARDIAC REMODELLING GRADING IN OLYMPIC ENDURANCE ATHLETES

LOWER



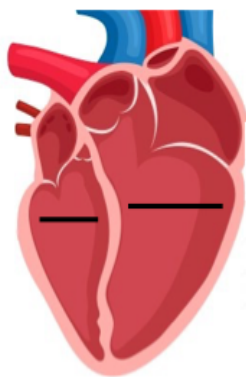
STANDARD



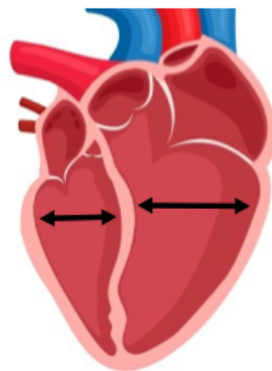
EXTREME



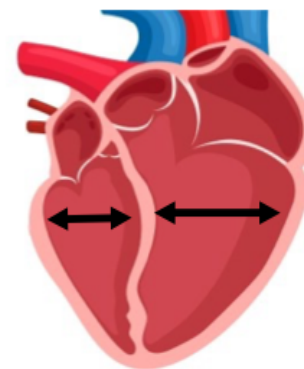
LVEDDi, mm/m²
LVEDVi, mL/m²



♂ 27,6
84,7
♀ 29,1
74,2



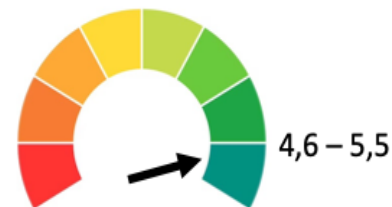
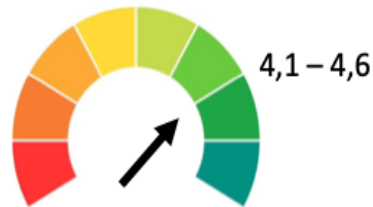
♂ 29,7 – 30,9
85 – 93,1
♀ 30,5 – 33,4
69,8 – 81,4



♂ 30,6 – 31,3
99,6 – 101,5
♀ 31,7 – 32,8
83,5 – 85,5



W/Kg

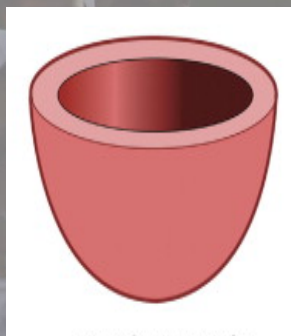


HR, bpm

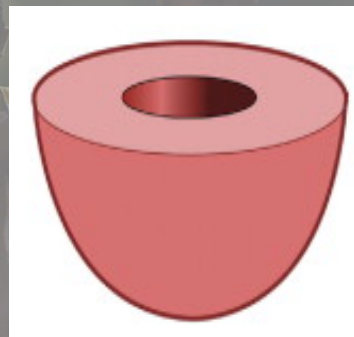


Pattern of LV remodeling

LV mass

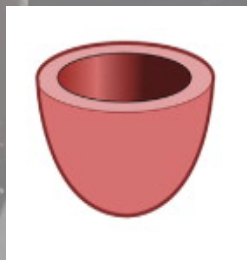


Eccentric remodeling



Concentric hypertrophy

Normal LV mass



Normal pattern



Concentric remodeling

Normal relative wall thickness



Relative wall thickness

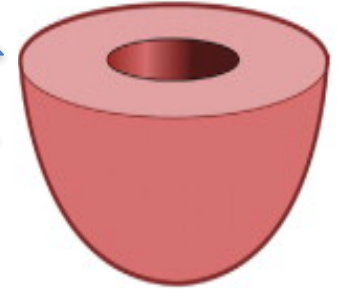
Concentric remodeling



Normal
LV mass

RWT 

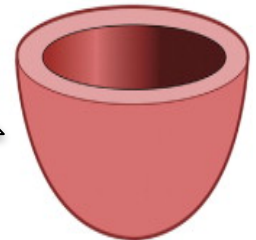
Concentric hypertrophy



LV mass 

RWT 

Eccentric remodeling



Normal
pattern

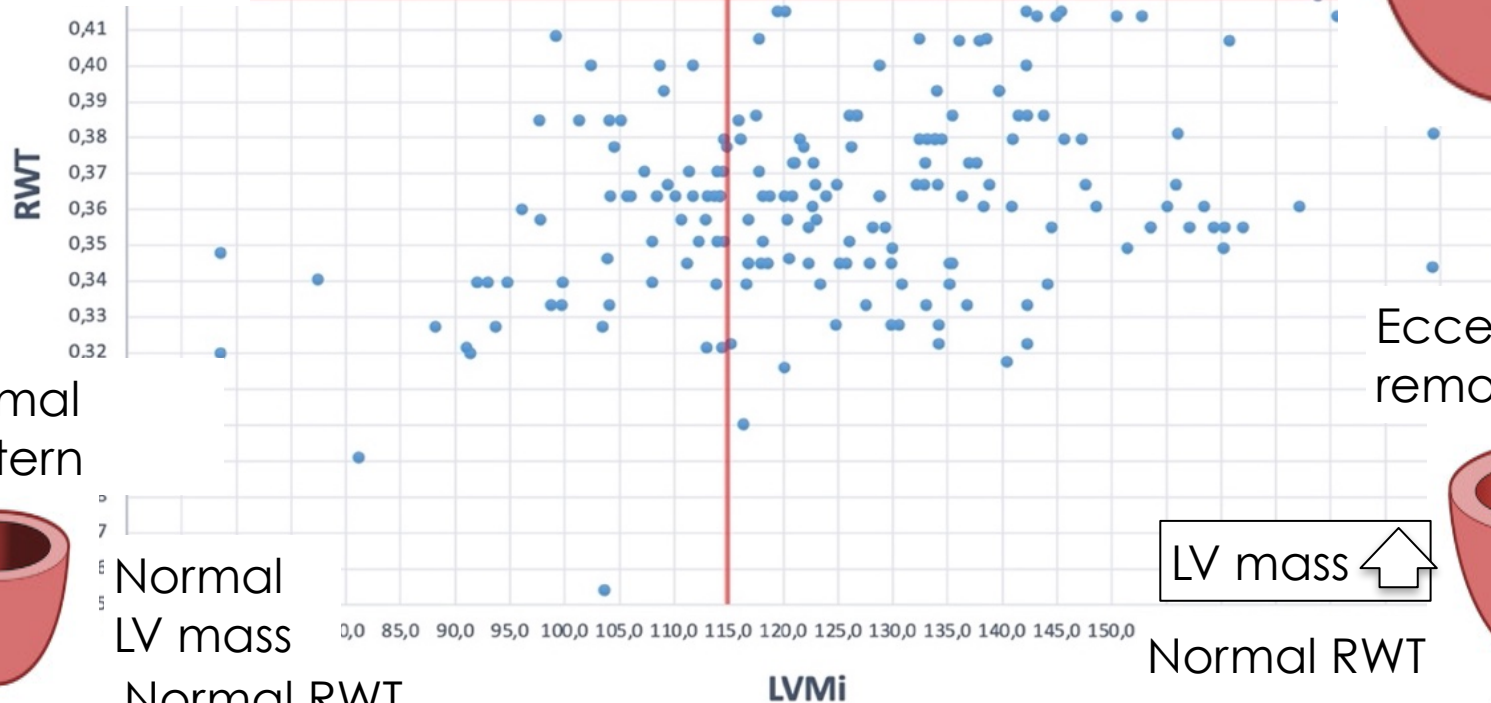


Normal
LV mass

Normal RWT

LV mass 

Normal RWT



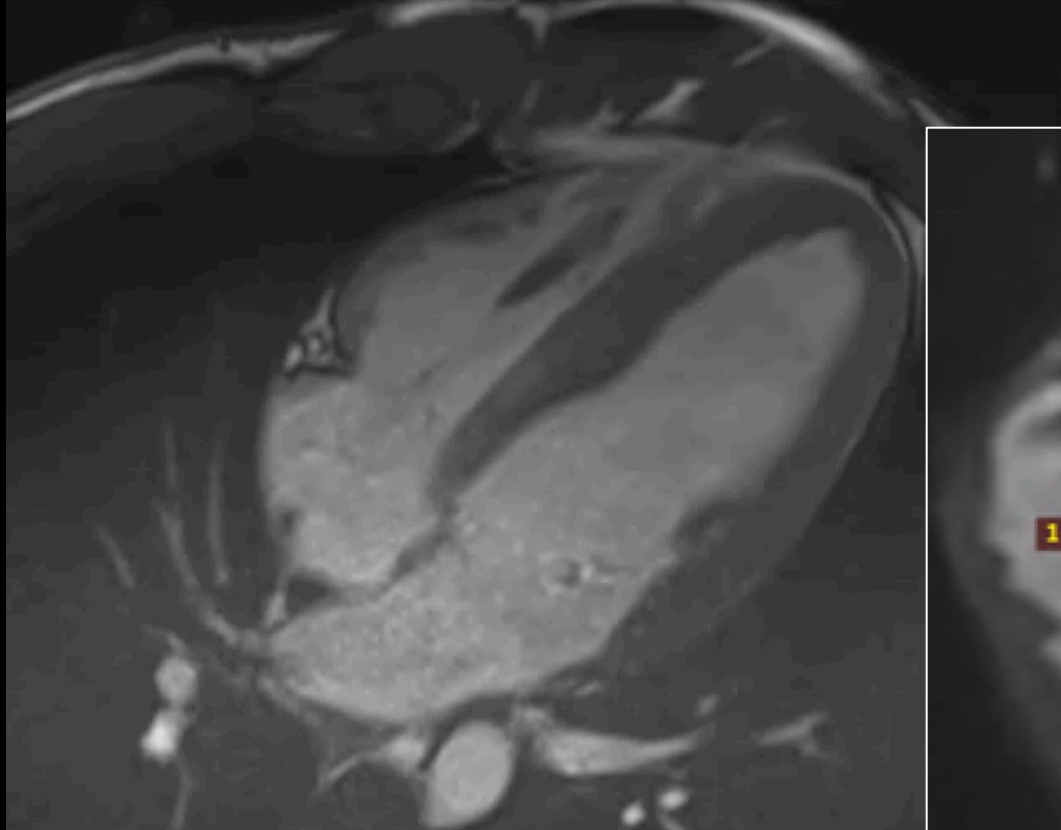
The upper limits of physiologic LV remodelling

Max LV wall thickness:

Adult Caucasian Athletes	15-16 mm ¹
Adult Afro-Caribbean Athletes	16 mm ²
Adult Female Caucasian Athletes	12 mm ³
Adult Female Afro-Caribbean Athletes	13-14 mm ^{3,6}
Adolescent/Young African Athletes	14-15 mm ⁵

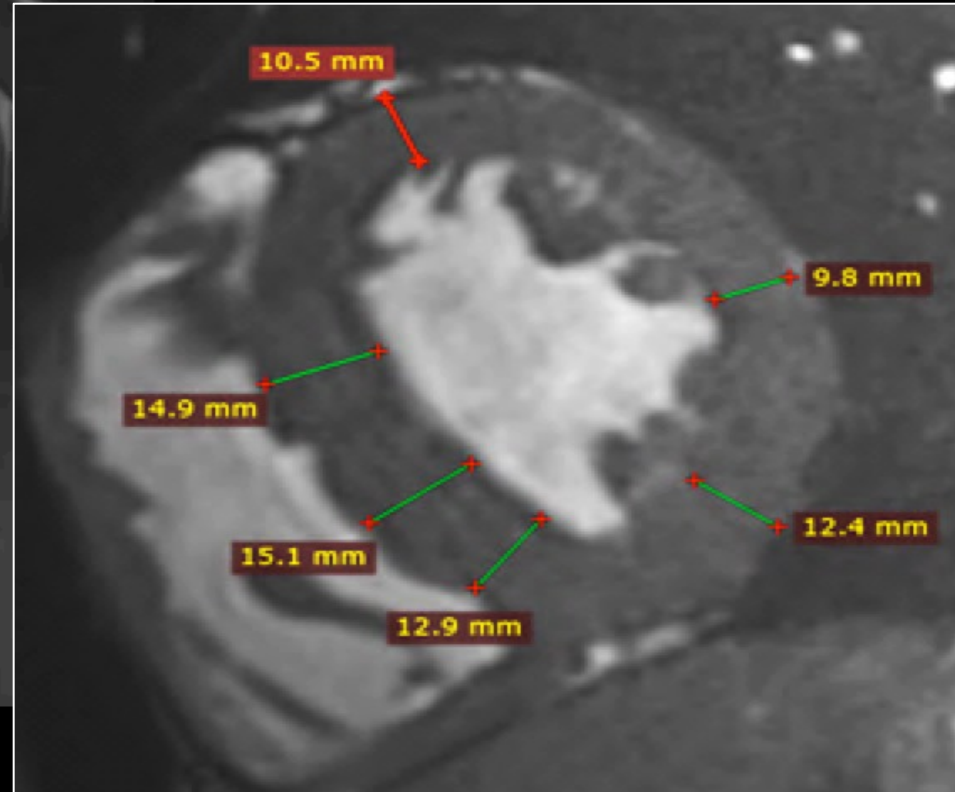
1. Pelliccia A, et al. N Engl J Med. 1991;324(5):295-301.
2. Basavarajaiah S, et al. J Am Coll Cardiol. 2008;51(23):2256-62
3. Pelliccia et al. JAMA. 1996;276(3):211-5 .
4. Basavarajaiah S, et al. J Am Coll Cardiol. 2008;51(10):1033-9.
5. Di Paolo FM, et al J Am Coll Cardiol. 2012;59(11):1029-36.
6. Quarta G, et al. Nature Reviews. Cardiology. doi:10.1038/nrcardio.2016.175.

The upper limits of physiologic LV remodelling



At peak training

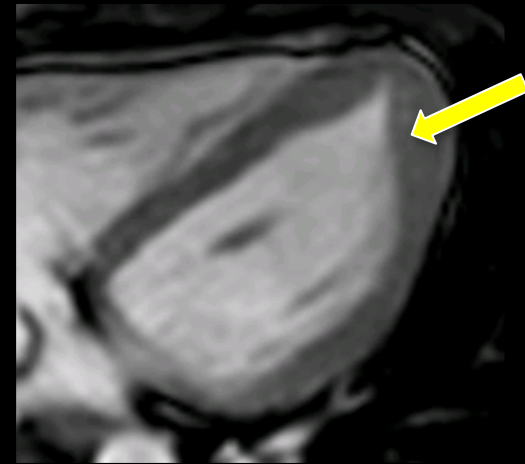
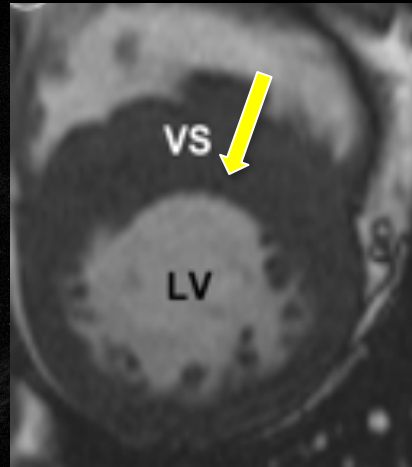
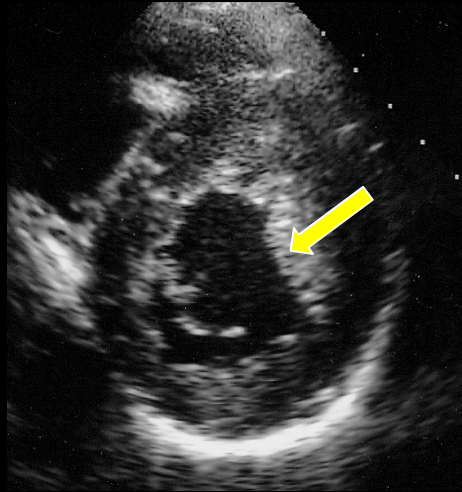
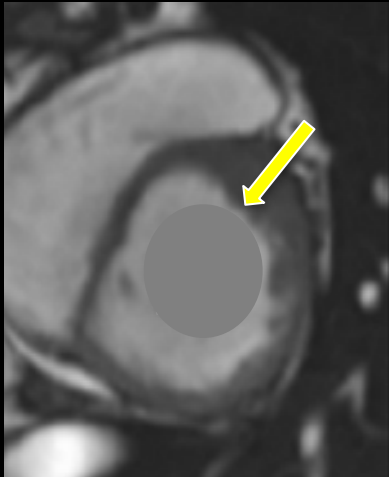
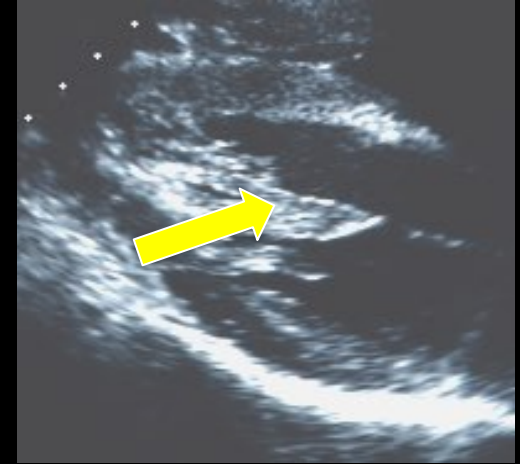
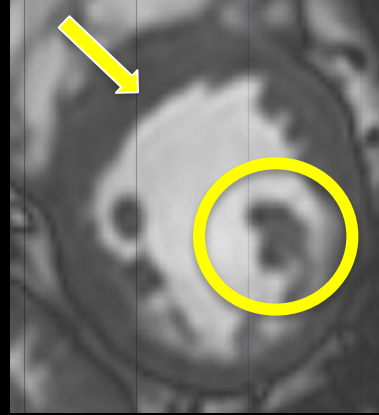
24-year, male, elite rower



Morphologic criteria
for differential diagnosis

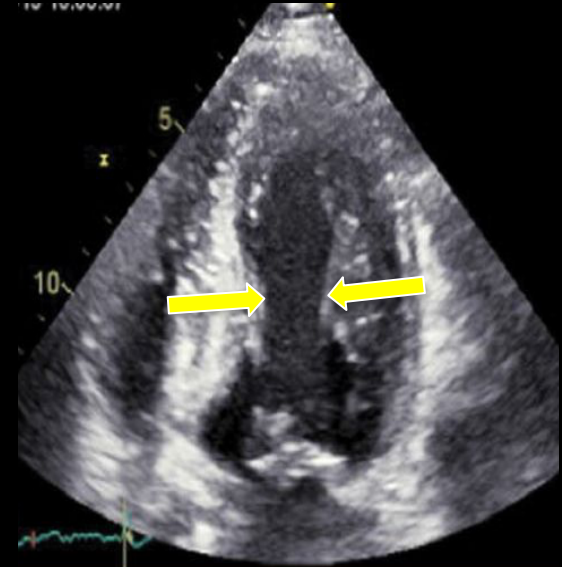
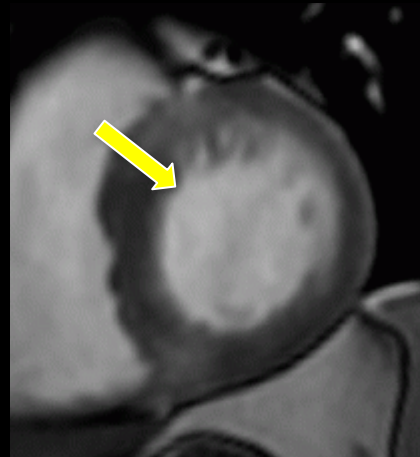
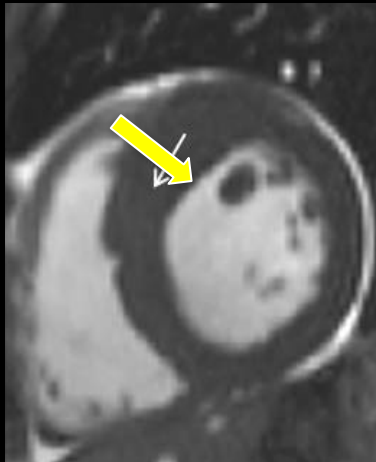
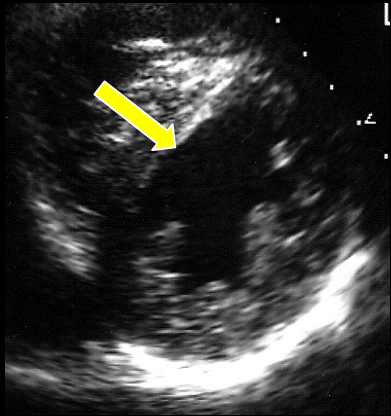
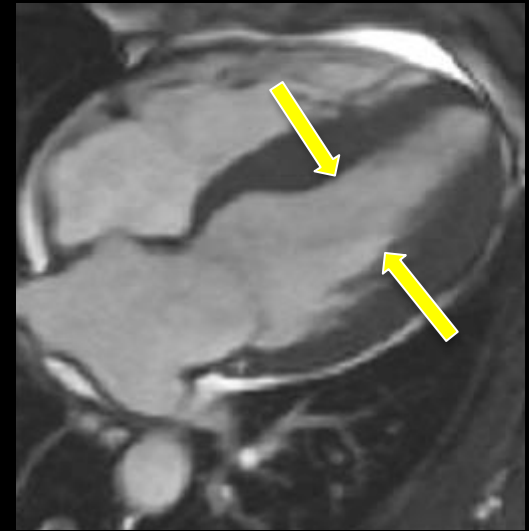
What to look for suspecting HCM

- Asymmetric/segmental LV wall thickening
- Maximum wall thickening (>15 mm)

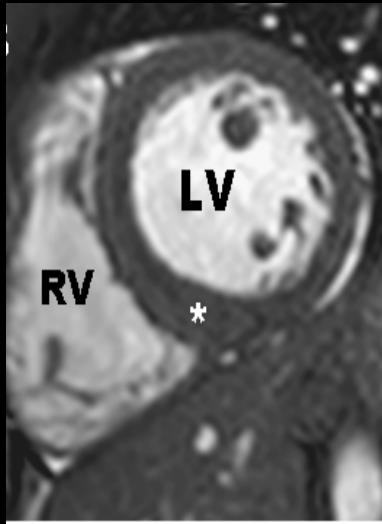


What to look for suspecting HCM

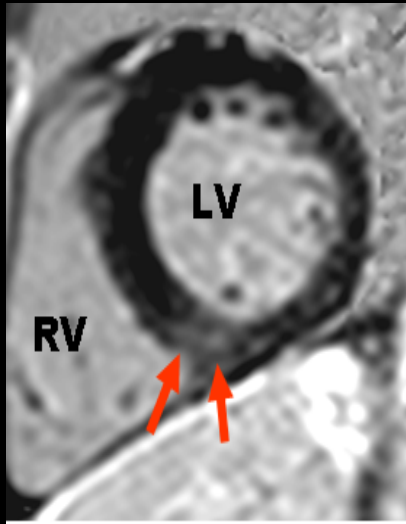
- Abnormal shape of LV cavity
- Normal (or mildly reduced) dimension of LV cavity (≤ 54 mm)



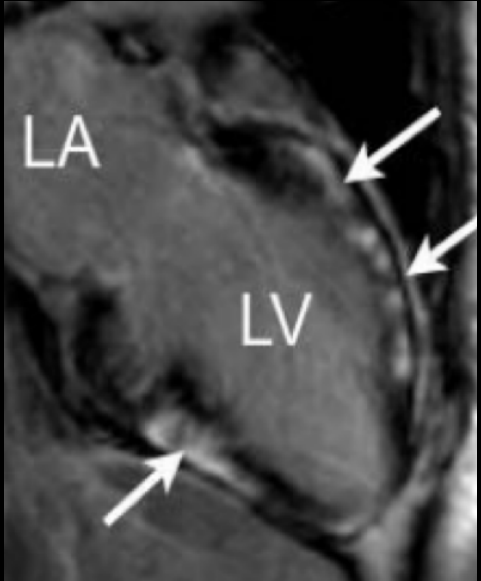
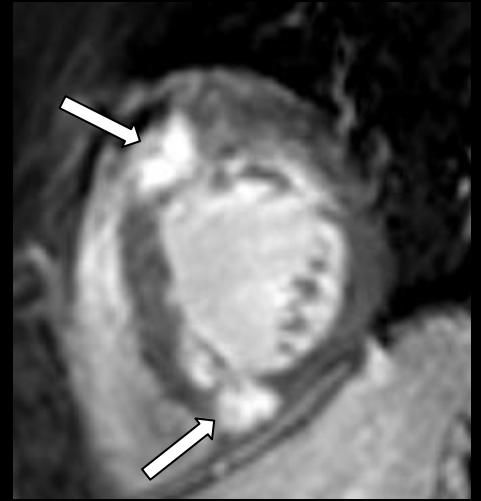
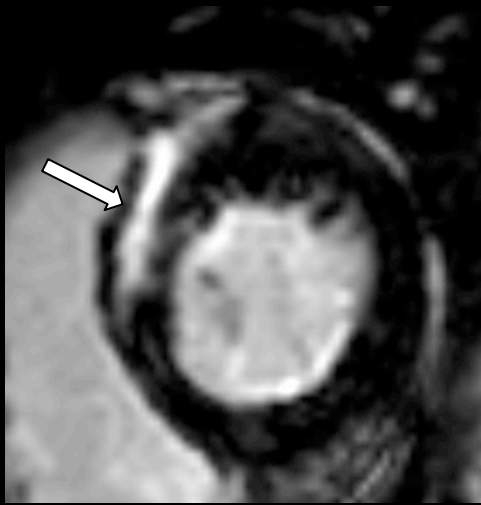
LGE at CMR



Baseline

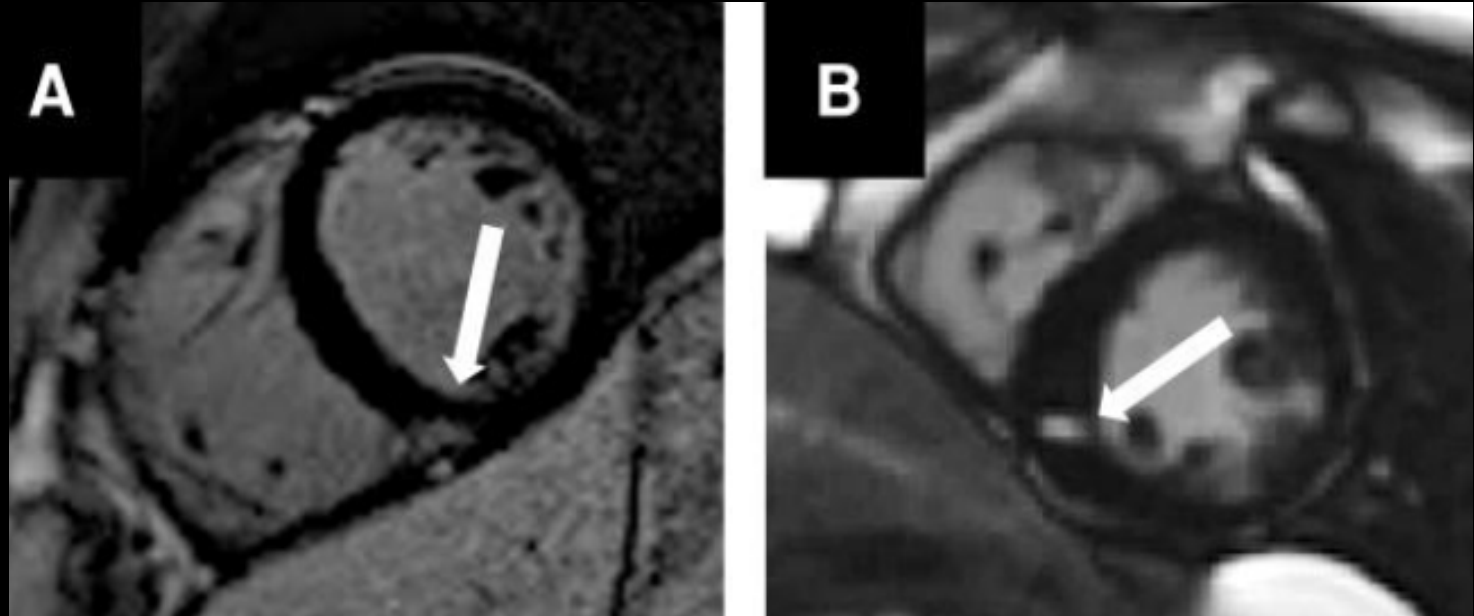


**Contrast-enhanced
CMR**



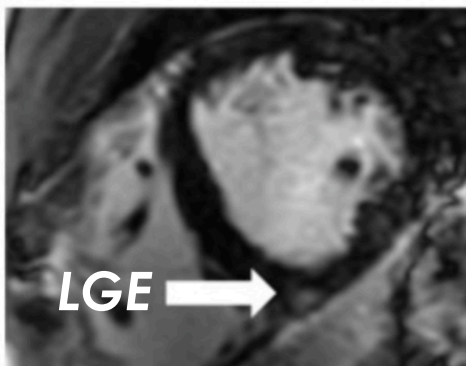
Presence of LGE
($>15\%$? Or 9% of LV
mass) is
associated with
incidence of VAs
and is an
independent risk
for SCD

LGE in AH and HCM

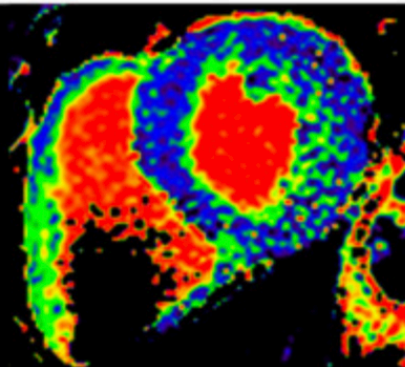


(A) LGE at the inferior hinge-point in healthy athlete (white arrow).
(B) Mid-myocardial LGE observed at the inferior RV insertion presenting with focal hypertrophy in an athlete with HCM (white arrow).

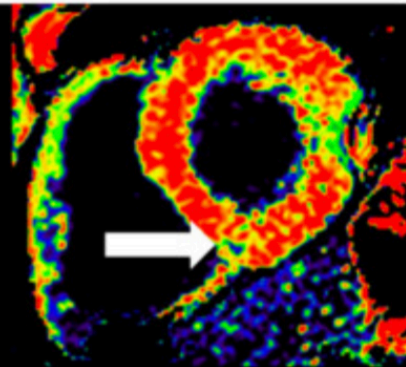
AH



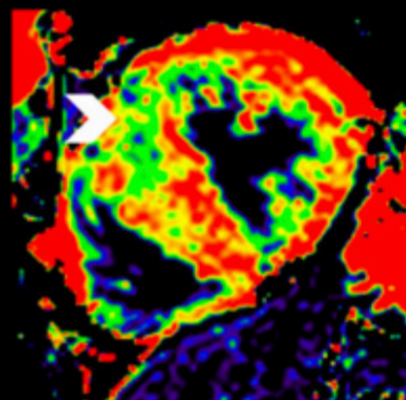
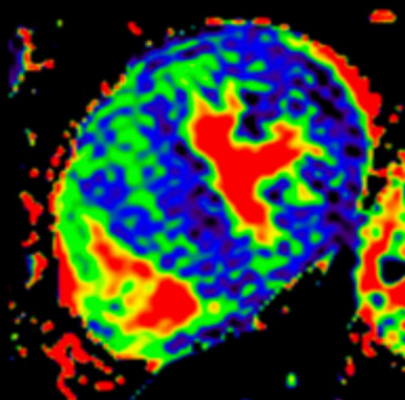
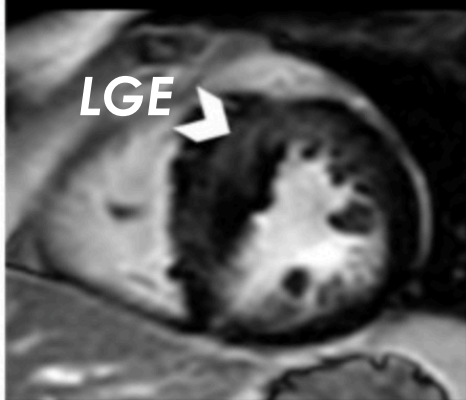
native T1 map



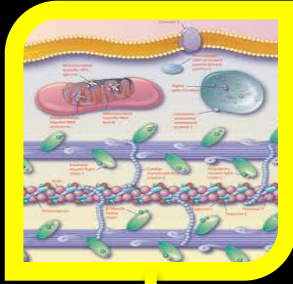
Post-contr T1 map



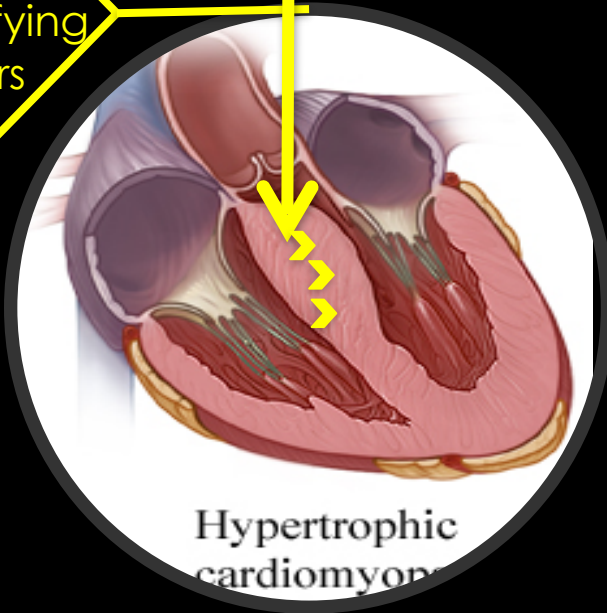
HCM



Gene anomalies



Triggers,
modifying
factors



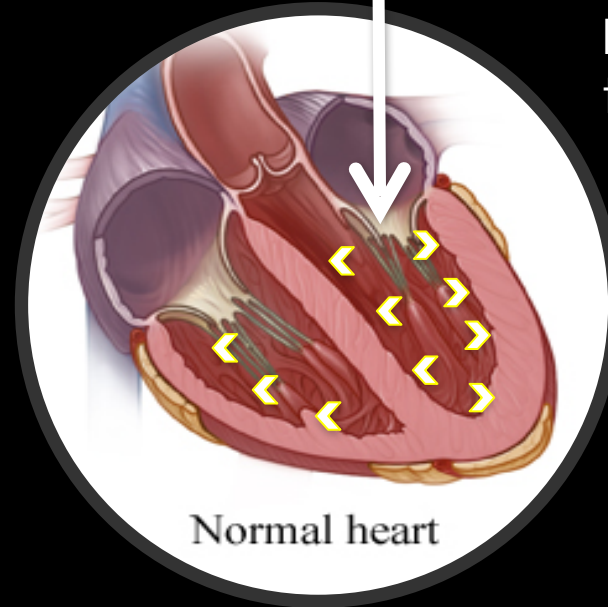
Hypertrophic
cardiomyopathy



La Place law

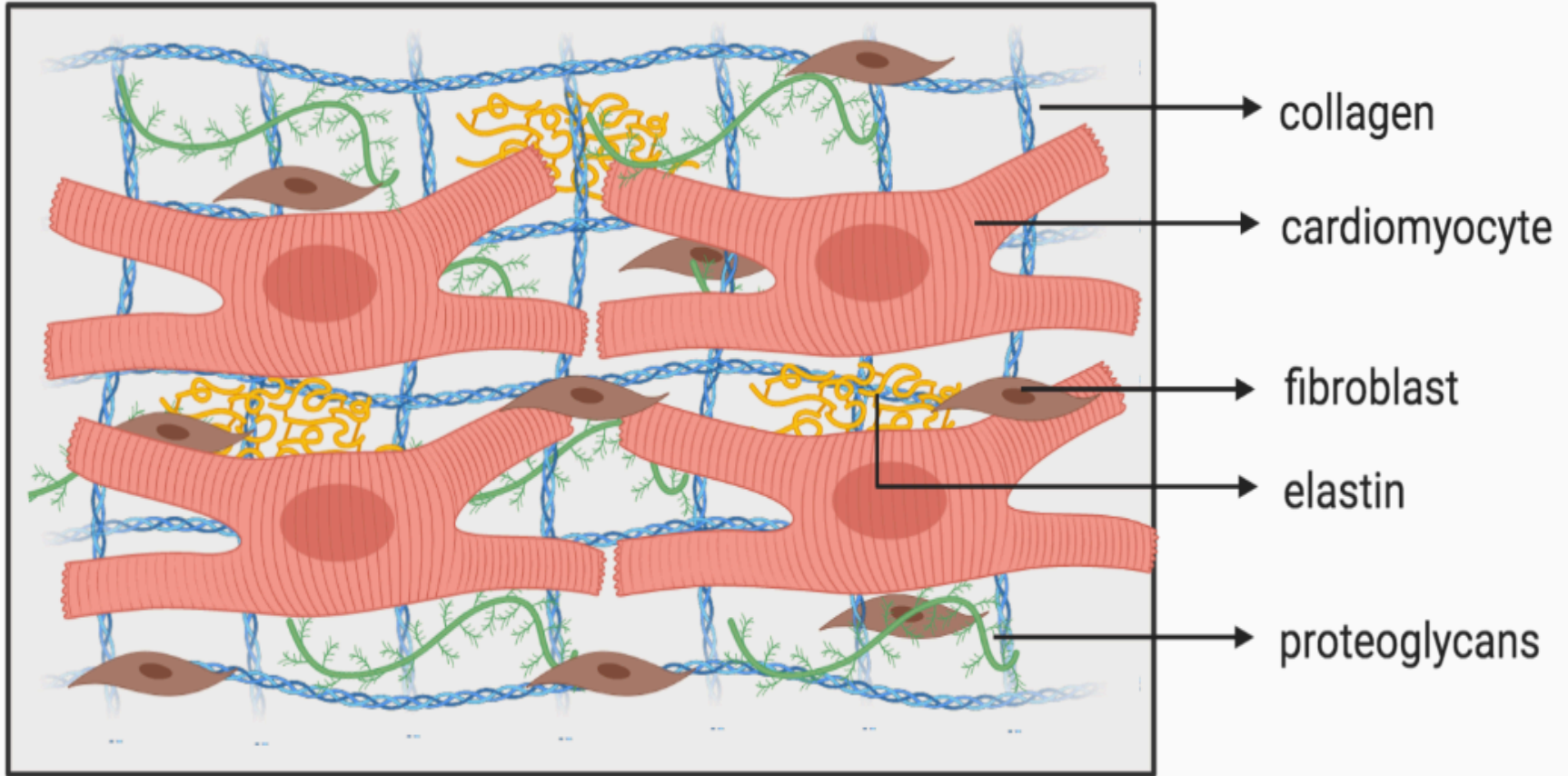
$$\tau = P \times r / 2h$$

τ = wall
stress
 P =
Pressure
 r = radius
 h =
thickness



Normal heart

Left ventricular remodeling is mostly consequence of myocyte's hypertrophy

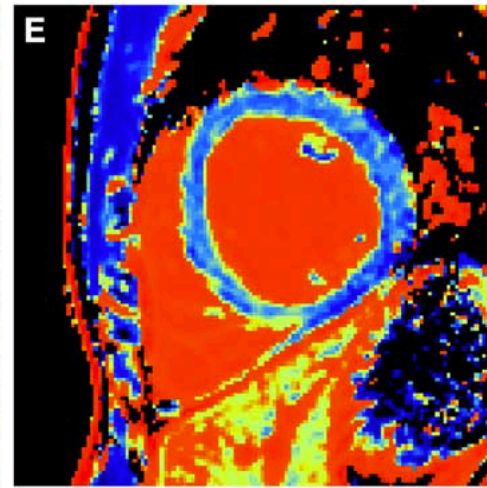
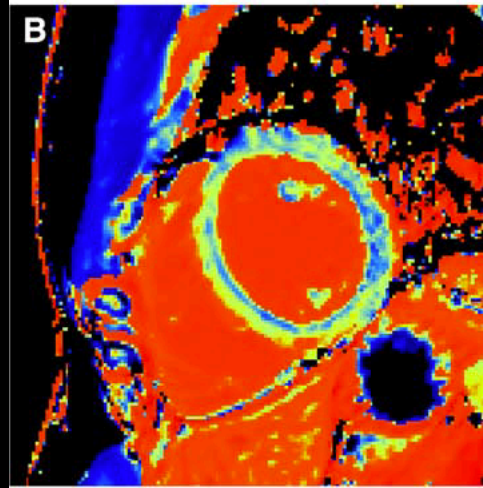
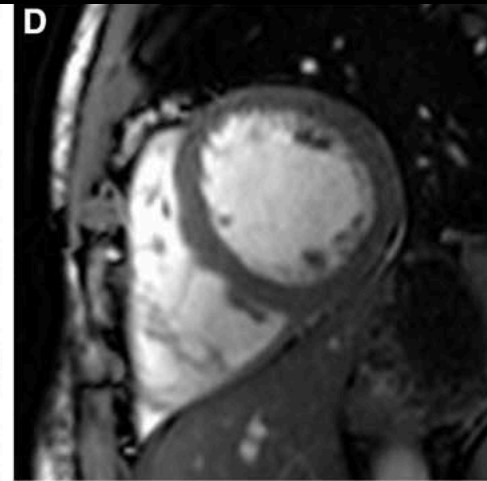
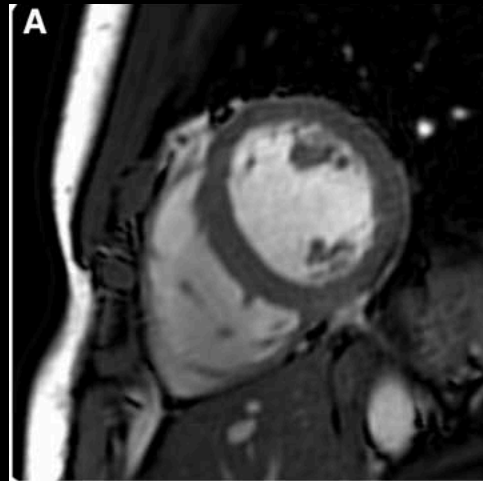


ORIGINAL ARTICLE

Athletic Cardiac Adaptation in Males Is a Consequence of Elevated Myocyte Mass

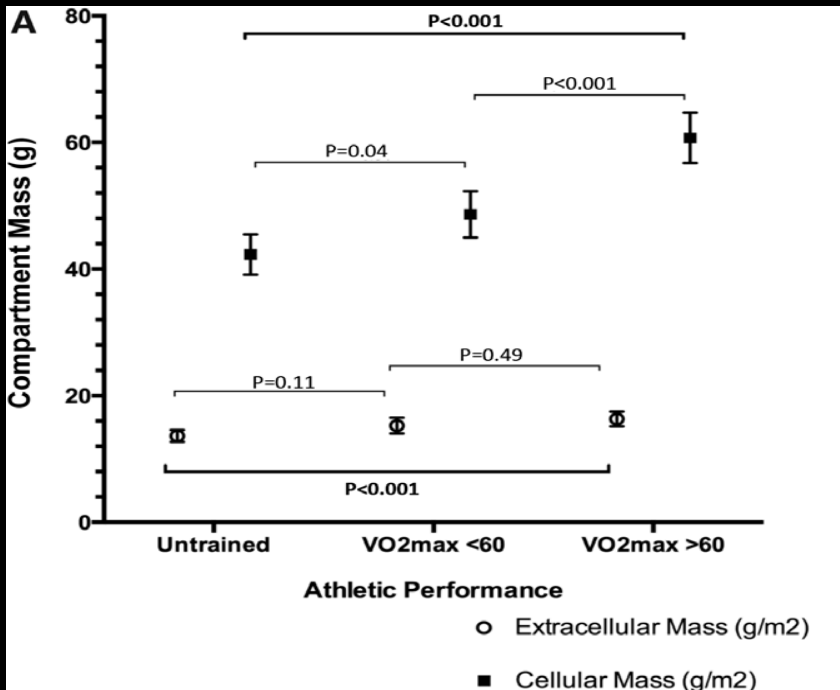
See Editorial by [Graham-Brown and McCann](#)

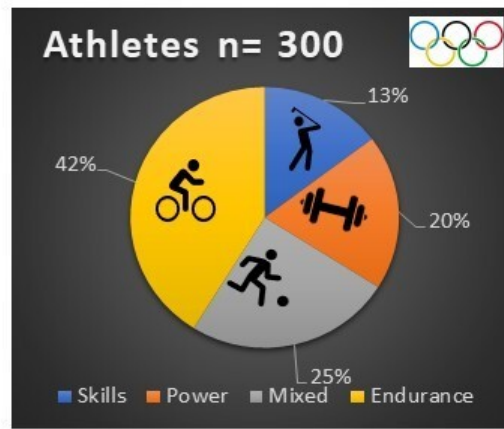
Adam K. McDiarmid, MD*, Peter P. Swoboda, MBBS*, Bara Erhayiem, BMBS, Rosalind E. Lancaster, PhD, Gemma K. Lyall, MSc, David A. Broadbent, BSc, Laura E. Dobson, MBChB, Tarique A. Musa, MBBS, David P. Ripley, MBChB, Pankaj Garg, MD, John P. Greenwood, PhD, Carrie Ferguson, PhD, and Sven Plein, PhD



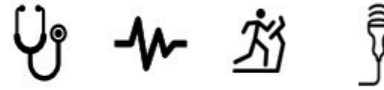
Indexed cellular mass
49 g/m²
Indexed extracellular
mass 15 g/m²

Indexed cellular mass
56 g/m²
Indexed extracellular
mass 14 g/m²





CMR-derived T1 and T2 mapping values in elite athletes

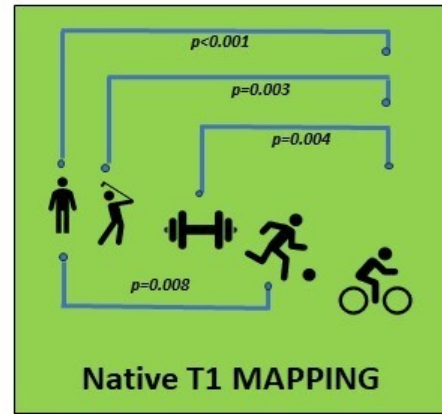


**NO
cardiac
abnormalities**



1400 ms
1200 ms
1000 ms
980 ms
960 ms
940 ms
920 ms
900 ms

90 ms
70 ms
50 ms
30 ms
10 ms



The lowest T1 mapping values are associated with the largest extent of LV remodelling and are common in athletes engaged in endurance and mixed disciplines. Instead, T2 mapping is not affected by sports type.

LV hypertrophy

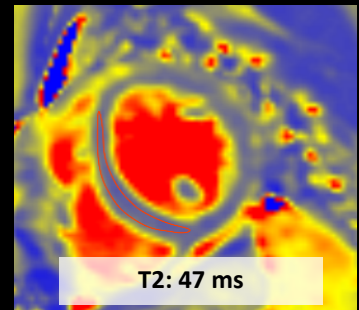
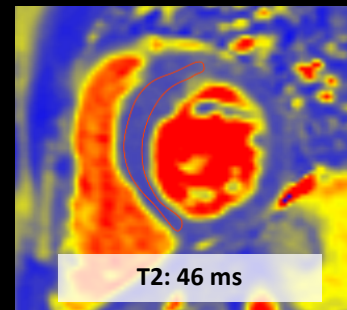
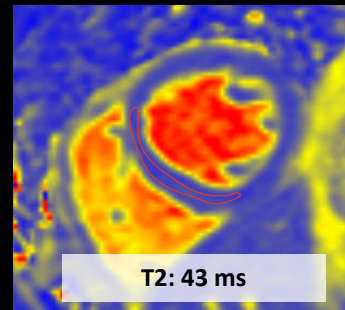
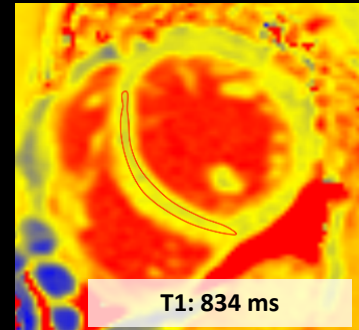
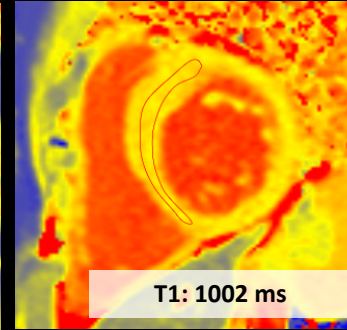
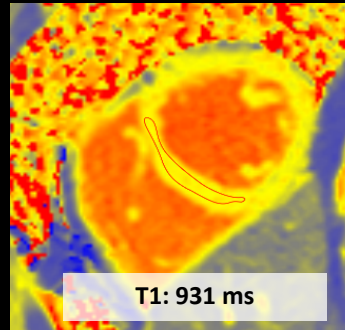
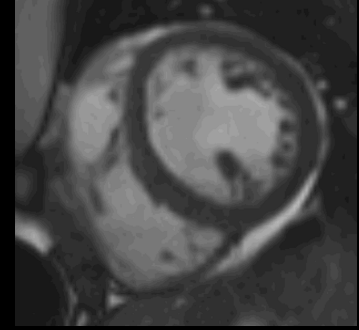
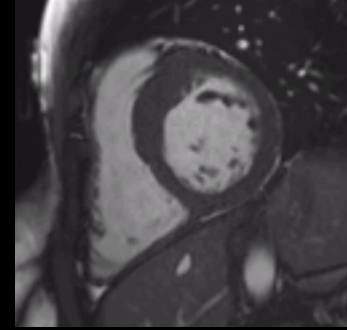
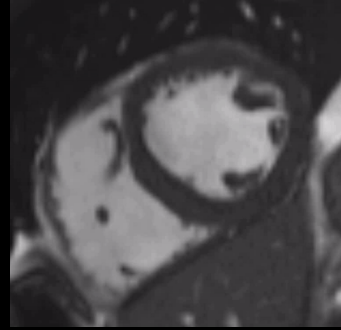
Differentiation of physiological and pathological hypertrophy

Different values in different pathology

Healthy athlete

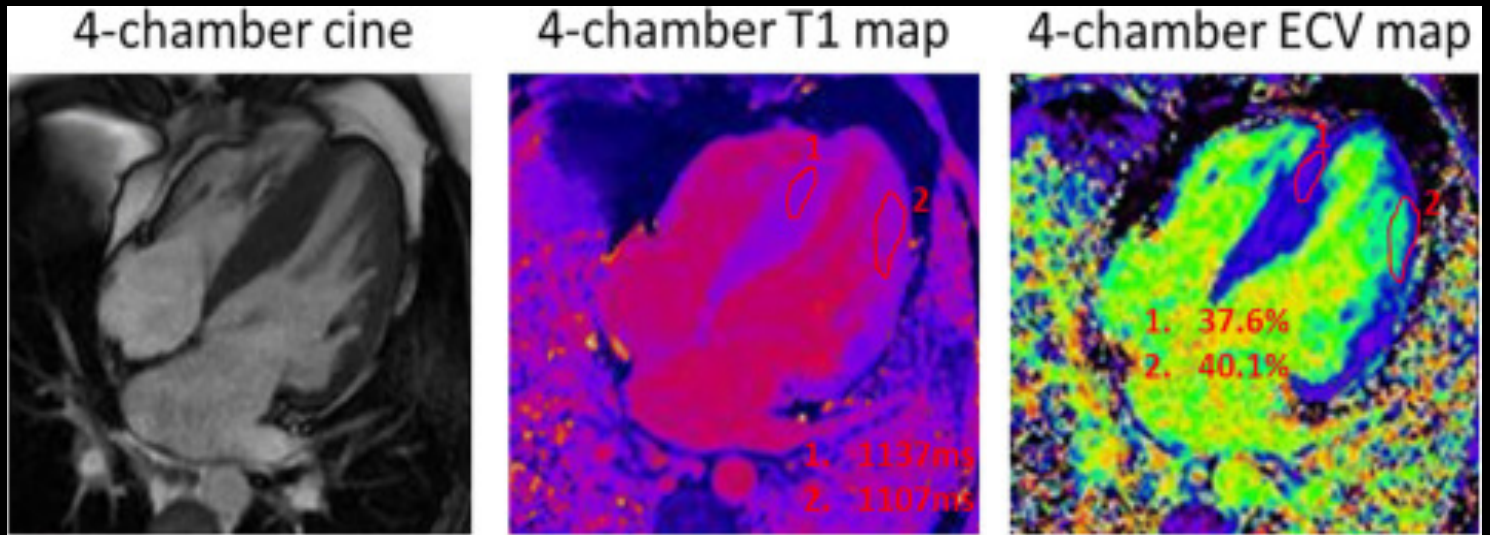
Athlete with HCM

Athlete with Fabry

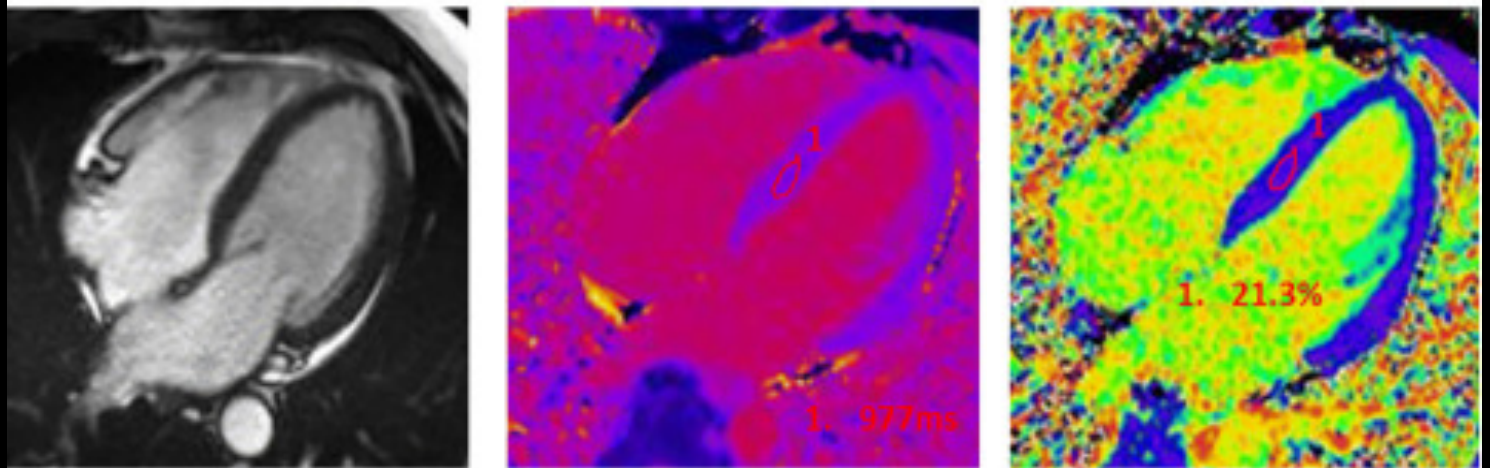


➤ ECV

HCM
Increased
ECV



Athlete's
heart
Normal/
reduced ECV



Comparison of LGE-Positive vs LGE-Negative Triathletes

45



LGE Negative (LGE-)
(n = not specified)

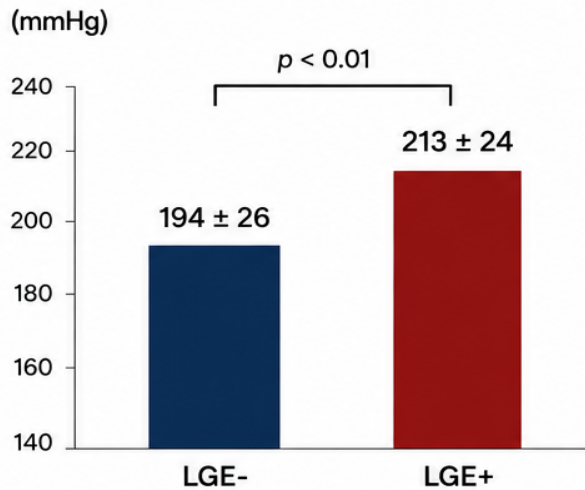
VS

9



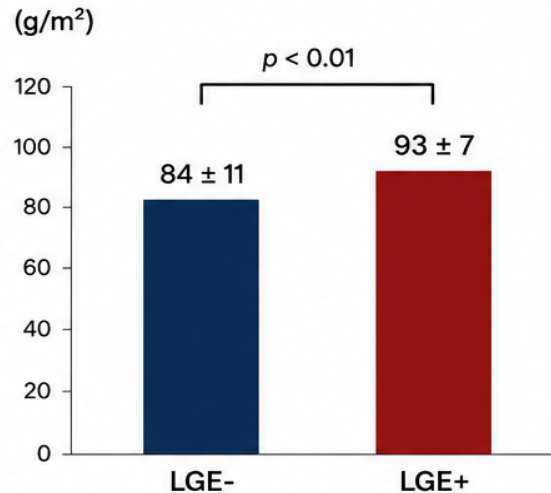
LGE Positive (LGE+)
(n = not specified)

1. Peak Systolic Blood Pressure



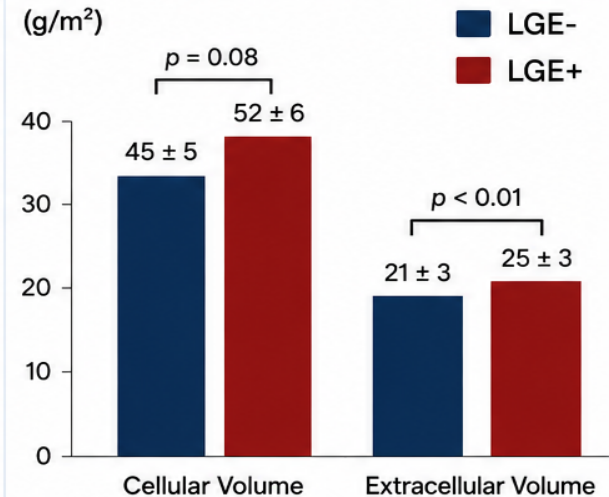
Peak systolic blood pressure is significantly higher in LGE+ subjects compared to LGE- subjects.

2. Left Ventricular Mass Index



LV mass index is higher in LGE+ subjects compared to LGE- subjects.

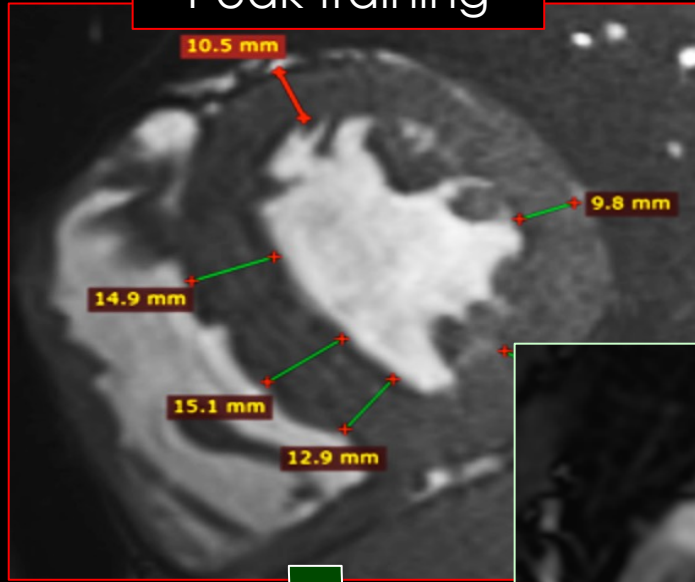
3. Cellular and Extracellular Volume



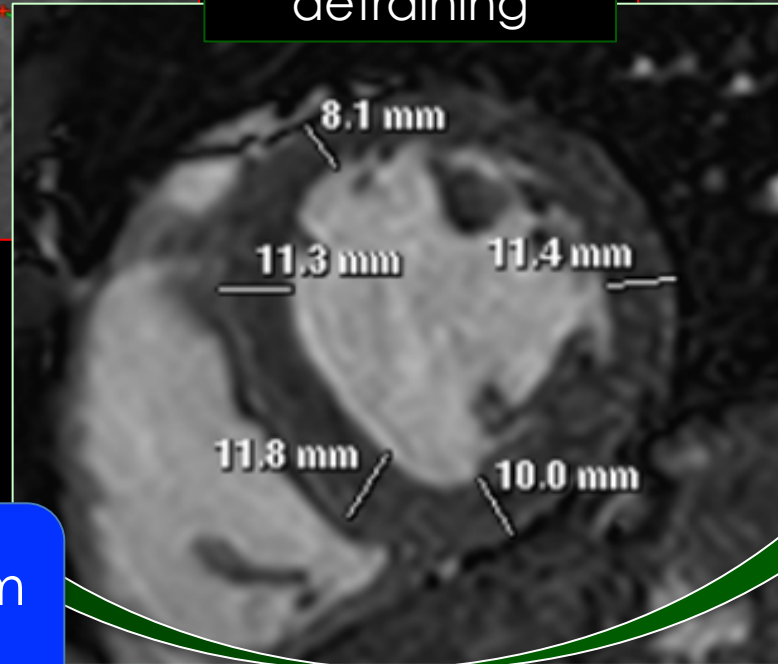
Both cellular and extracellular volumes are higher in LGE+ subjects; the difference is statistically significant for extracellular volume.

Reversibility of LVH
with short-term detraining

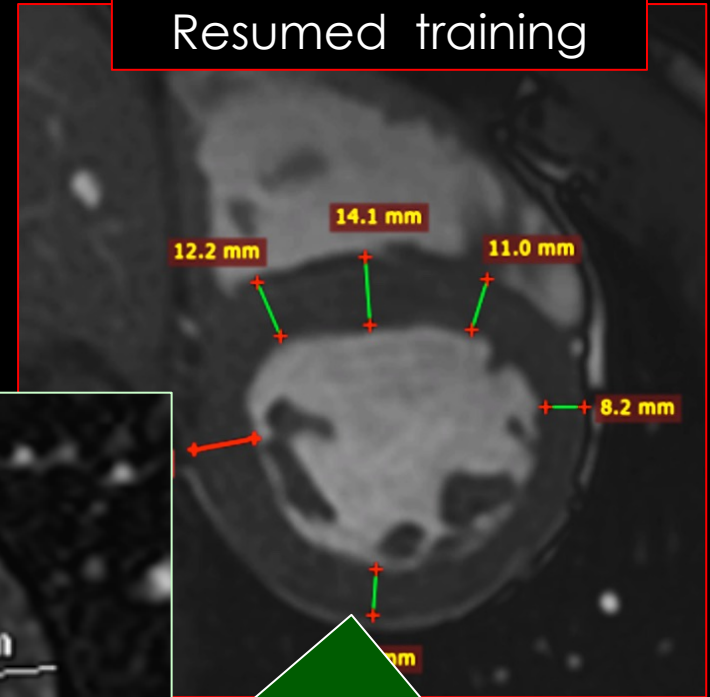
Peak training



After 6-month detraining



Resumed training



Effect of short-term detraining

Reversal of LVH in a former Olympic marathon runner

LVWT
13 mm



CARDIO
XS-1
50Hz
16cm
2D
54%
C 53
P Basso
ARis

3
P 1.6 3.2



TISO.4 MI 1.3

MI

LVWT
10 mm



CARDIO
XS-1
50Hz
16cm
2D
50%
C 53
P Basso
ARis

3
P 1.6 3.2



TISO.4 MI 1.3

MI

LVDD
57 mm

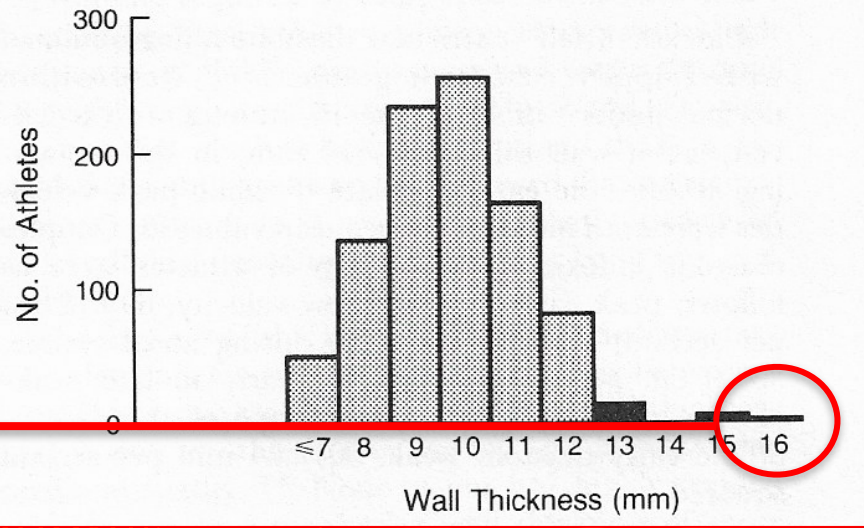
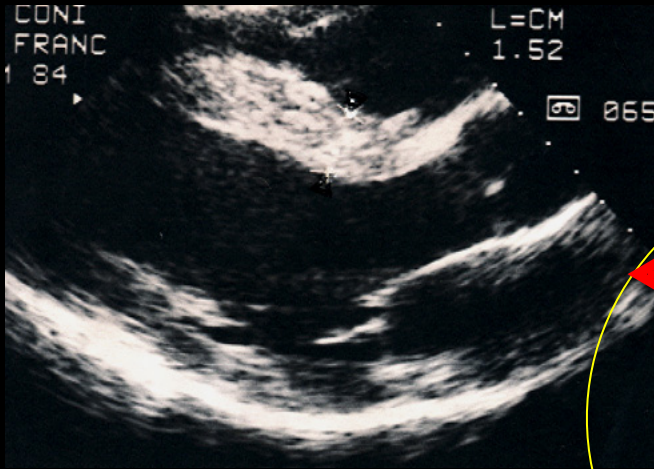


LVDD
53 mm

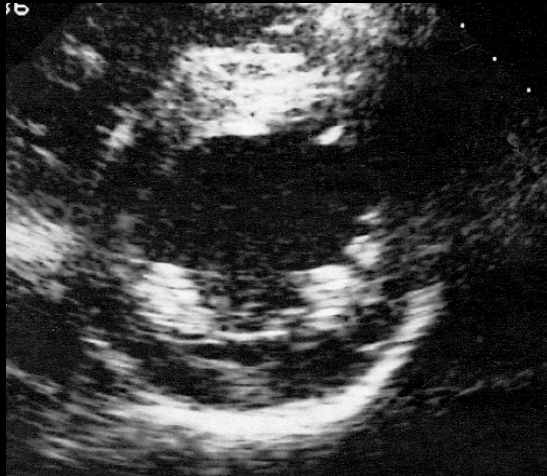


45 bpm

1988, at peak training

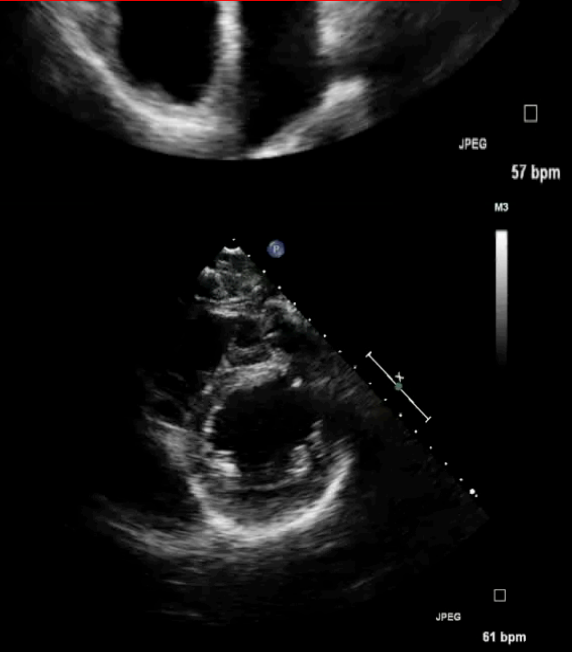


After
of
No events,
No symptoms,
No evidence of CV
abnormality



FR 47Hz
17cm
2D
66%
C 52
P Bassa
AGen

1.7 3.4





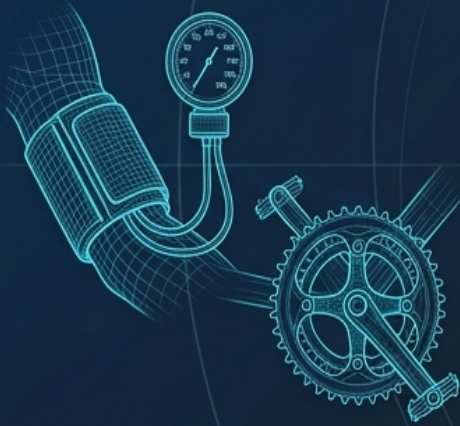
Thank you for your attention

See also :www.antonipelliccia.it

Synthesis: The Mechanics of Myocardial Scarring

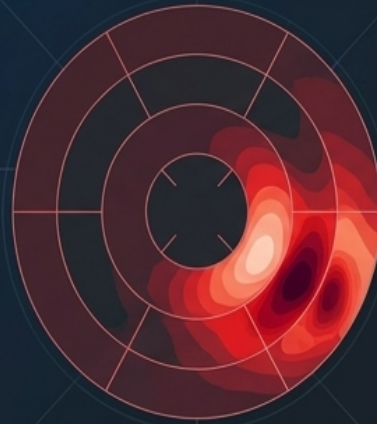
The development of nonischemic fibrosis in elite triathletes is not random. It is a predictable function of extreme mechanical overload interacting with localized anatomical vulnerability, establishing a definitive safe upper limit for endurance exercise.

1. Mechanical Overload



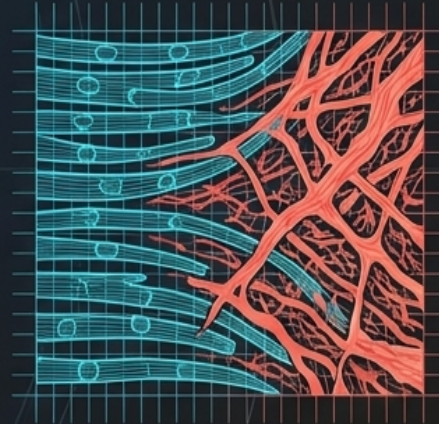
Cumulative Volume (>1,880km cycling) + **Extreme Hypertension** (>214 mm Hg peak BP).

2. Anatomical Vulnerability



Repetitive mechanical wall stress concentrated at the **basal inferolateral** myocardium.

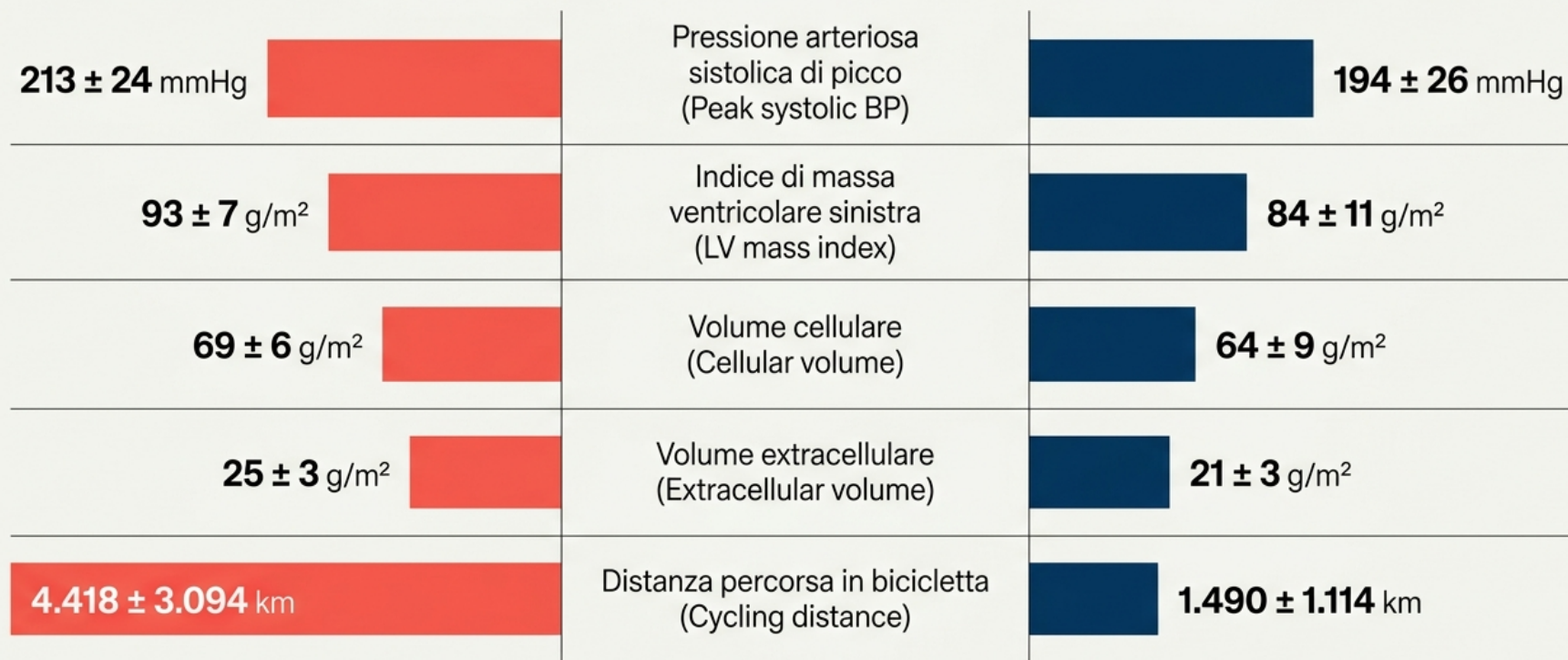
3. Pathological Remodeling



Focal scar formation (**LGE+**) and diffuse interstitial matrix expansion (**ECV >26.3%**).

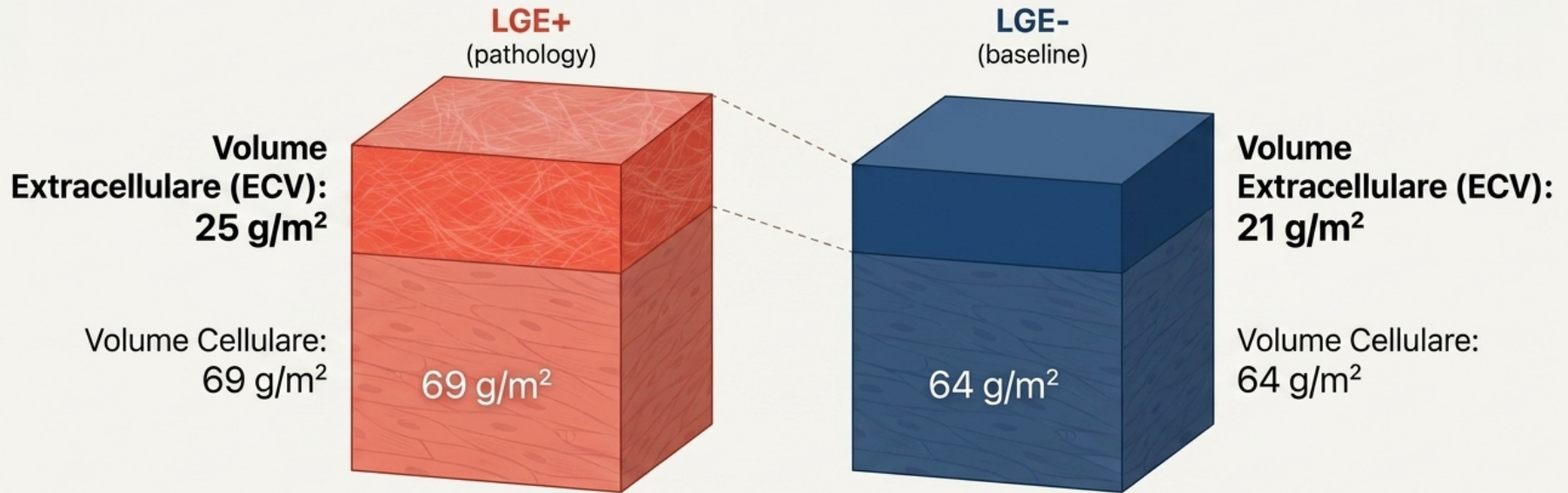
Profilo Clinico Comparativo: LGE+ vs LGE-

Analisi dei fattori di rischio emodinamici, strutturali e di carico.



Anatomia dell'Ipertrofia: Espansione dello Spazio Extracellulare

Il cuore LGE+ non ha solo miociti più grandi; presenta una fibrosi diffusa nascosta.



Insight Diagnostico: Il volume extracellulare (ECV) elevato indica che, oltre alle cicatrici focali (visibili con LGE), l'intero miocardio sta subendo una trasformazione fibrotica diffusa.

The Morganroth hypothesis

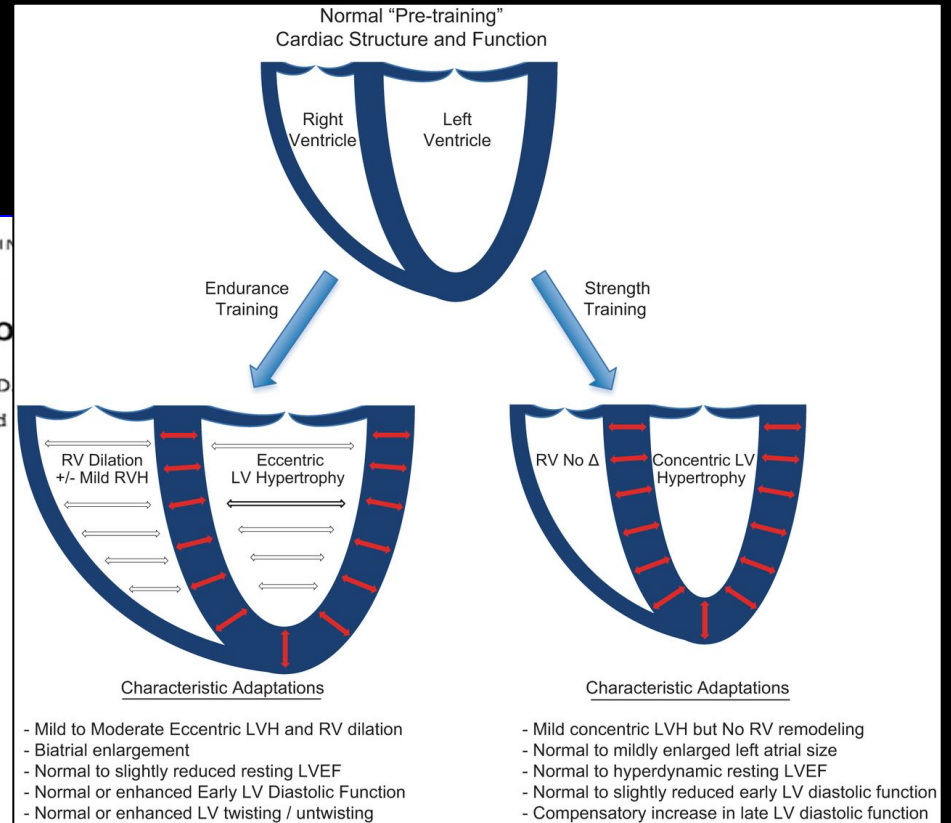


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Comparative Left Ventricular Dimensions

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

Little is known about the structure of athletes' hearts or anatomic variations associated with training. Echocardiograms of 56 active athletes were obtained. Mean left ventricular end-diastolic volume and mass were increased in athletes involved in isotonic exercise, such as swimming (181 ml, 308 g) and running (160 ml, 302 g), compared with controls (101 ml, 211 g); wall thickness was normal (≤ 12 mm). Athletes involved in isometric exercise, such as wrestling and shot putting, had normal mean left ventricular end-diastolic volumes (110 ml, 122 ml), but increased wall thickness (13 to 14 mm) and mass (330 g, 348 g). Thus, athletes participating in isotonic exercise had increased left ventricular mass with cardiac changes similar to those in chronic volume overloads. Athletes participating in isometric exercise had increased left ventricular mass with cardiac changes similar to those in chronic pressure loads. Recognizing greater left ventricular mass and volume in well-trained athletes aids in interpreting values deviating from "normal" limits.



HCM

ASYMMETRIC LVH	++	+++
NO CONCOMITANT LV DILATATION	++	+++
SUPRANORMAL LVEF	++	+++
DIASTOLIC DYSFUNCTION	+++	+
DISPROPORTIONATE LA DILATATION	++	+++
NON-ISCHEMIC LGE	-	+++
INCREASED NATIVE T1 MAPPING/ECV	-	+++

ATHLETE'S HEART

BALANCED BIVENTRICULAR DILATATION	++	+++
NO RWMA	++	+++
LV DILATATION WITH MILD LVH	++	+++
BALANCED ATRIAL DILATATION	+++	+++
HYPERTRABECULATION WITH PRESERVED COMPACTED LAYER	++	+++
NORMAL/LOW-NORMAL EF	++	+++
NORMAL/HIGH SV	++	+++
NORMAL/SUPRANORMAL DIASTOLIC FUNCTION	+++	+
NORMAL/LOW-NORMAL GLS	+++	+++
NO LGE	-	+++
LGE AT THE INFERIOR HINGE POINT	-	+++
LOW NATIVE T1 MAPPING/NORMAL ECV	-	+++

ACM

DISPROPORTIONATE RV DILATATION	++	+++
RV GLOBAL DYSFUNCTION	++	+++
RV RWMA	+	+++
RV WALL FAT INFILTRATION +/- LGE	-	++
LV GLOBAL DYSFUNCTION	++	+++
LV RWMA	++	+++
LV FAT INFILTRATION +/- NON-ISCHEMIC LGE	-	+++

GREY ZONE: DIFFERENTIAL DIAGNOSIS WITH CARDIOMYOPATHIES

DCM

DISPROPORTIONATE LV DILATATION	++	+++
LV DILATATION WITHOUT LVH	++	+++
SYSTOLIC DYSFUNCTION	++	+++
REDUCED SV	++	+++
DIASTOLIC DYSFUNCTION	+++	+
REDUCED GLS	+++	+++
NON-ISCHEMIC LGE	-	+++
INCREASED NATIVE T1 MAPPING/ECV	-	+++

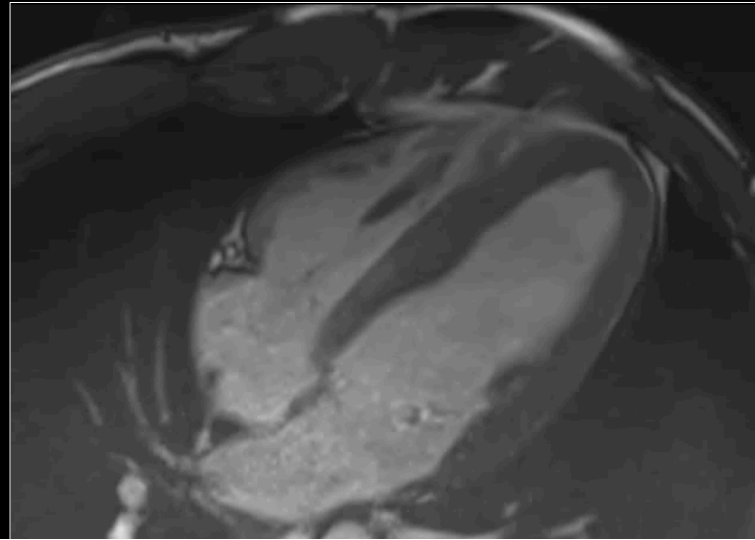
CCT TO RULE OUT CONCOMITANT CAD IN ATHLETES WITH SUSPECTED OR KNOWN HCM OR DCM

CCT WITH CONCOMITANT LATE ENHANCEMENT ANALYSIS IN ATHLETES UNABLE TO UNDERGO CMR IMAGING

NUCLEAR TECHNIQUES PLAY A CRUCIAL ROLE IN AIDING DIAGNOSIS AND RISK STRATIFICATION IN SPECIFIC INFLAMMATORY CARDIOMYOPATHY, SUCH AS SARCOIDOSIS.

Possible determinants of LV remodelling in athletes

Body
size &
compo
sition



CV risk
factors

Type

Sp

Base

European Journal of Applied Physiology
<https://doi.org/10.1007/s00421-022-05098-9>

ORIGINAL ARTICLE



Determinants of LV mass in athletes: the impact of sport, constitutional traits and cardiovascular risk factors

Antonio Pelliccia¹ · Cristian Borrazzo² · Viviana Maestrini^{1,3} · Flavio D'Ascenzi⁴ · Stefano Caselli⁵ · Erika Lemme¹ · Maria Rosaria Squeo¹ · Barbara Di Giacinto¹

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