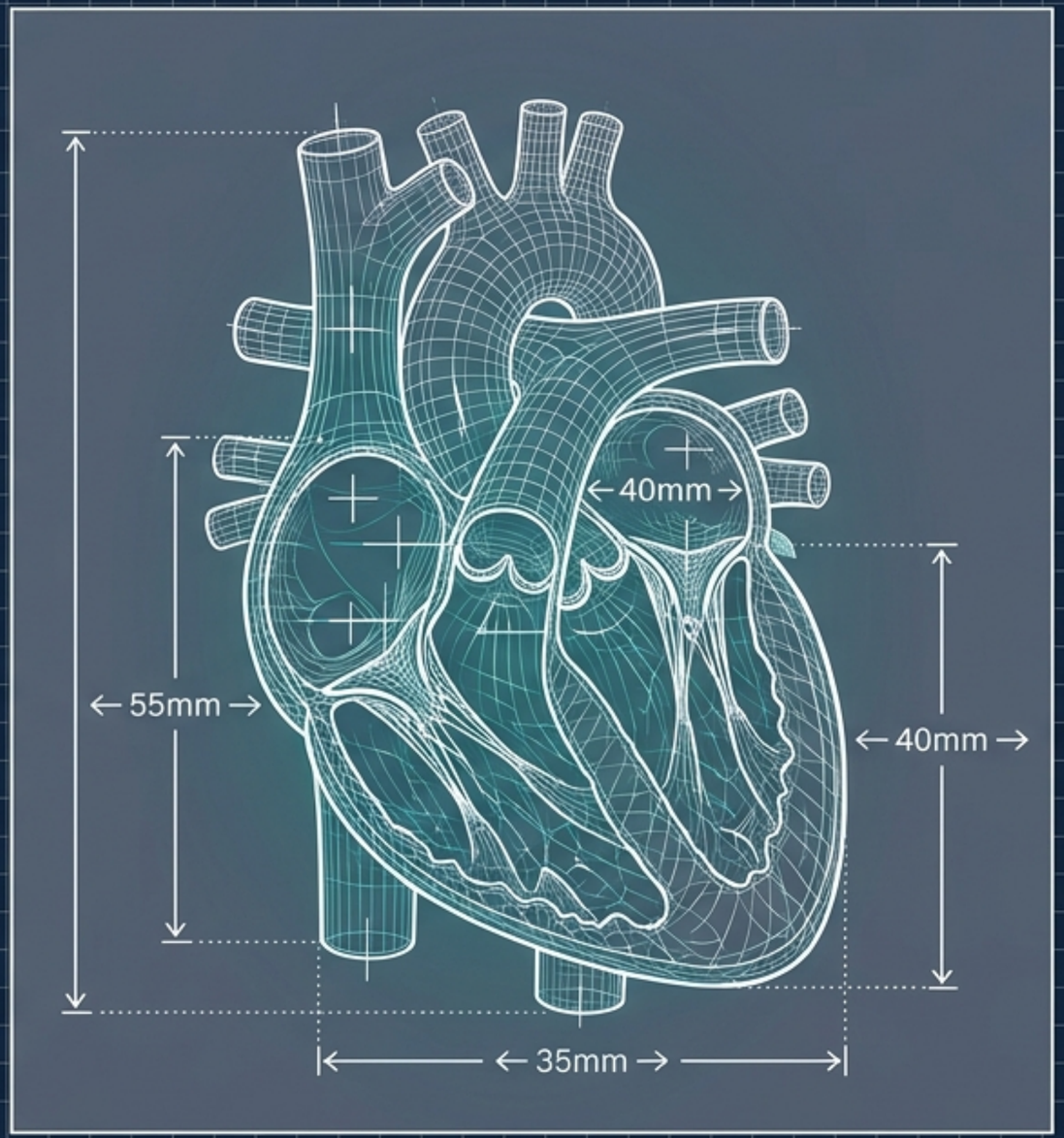
The Diagnostic Blueprint: Multimodality Imaging of the Athlete's Heart

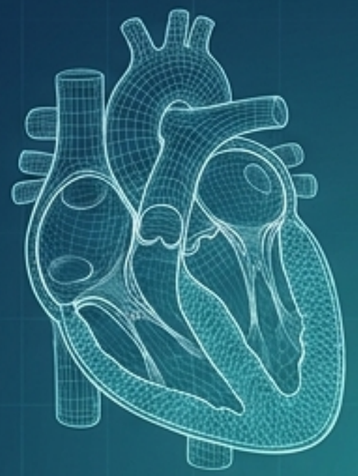
A Clinical Reference Guide for Differentiating Exercise-Induced Cardiac Remodeling from Cardiovascular Pathology in Competitive Athletes.

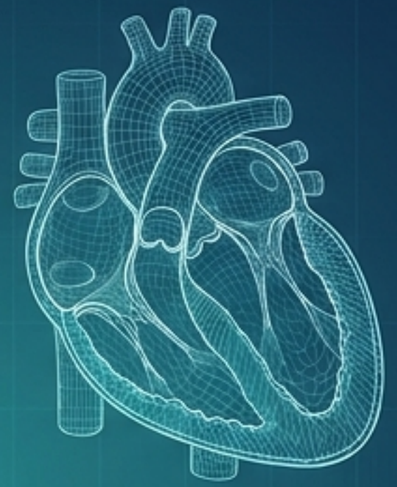


2x2 Hemodynamic Grid

Increasing **Static Component** (Pressure Load / Isometric Stress) -->

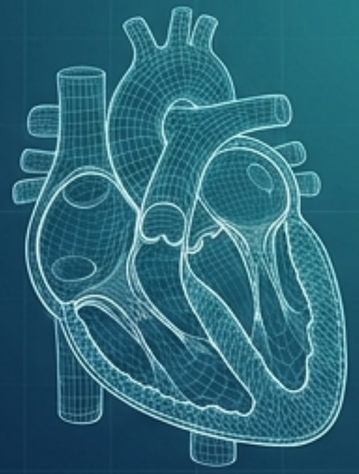
Concentric LVH
e.g., Bobsledding, Martial Arts, Weightlifting

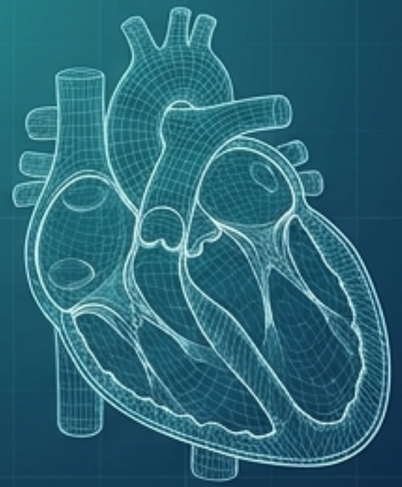




Eccentric LVH & RV Dilation
e.g., Rowing, Cycling

Normal Morphology
e.g., Bowling, Golf

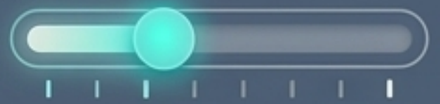




Eccentric LV Remodeling & RV Dilation
e.g., Long distance running, Soccer

Increasing Dynamic Component (Volume Load / Isotonic Stress) -->

Baseline Dimensions are Modulated by Athlete Demographics



Sex

Female athletes exhibit quantitatively less physiologic remodeling (11% smaller LV cavity, 23% less wall thickness) than male counterparts, even after correcting for body size.

Ethnicity

Athletes of Afro-Caribbean descent (Black athletes) tend to have thicker LV walls than Caucasians, shifting normal limits up to 15mm.

Training Duration

Remodeling is phasic. Acute volume overload leads to initial dilation, while chronic, extended high-intensity training drives secondary wall thickening.

Genetics

Polymorphisms (e.g., ACE-deletion/deletion) and familial polygenic traits (like familial hypertension) influence the magnitude of concentric hypertrophy.

The Multimodality Toolkit: Modality Capabilities & Selection

	Transthoracic Echocardiography (TTE)	Cardiac Magnetic Resonance (CMR)	Computed Tomography Angiography (CTA)
Cost	+++	+	++
Accessibility	+++	+	++
Portability	+++	+	+
Normative CA Data	+++	++	+
LV Morphology	++	+++	+++
RV Morphology	+	+++	++
Ventricular Tissue Composition	+	+++	+
Proximal Coronary Anatomy	+	++	+++
LV Systolic Function	+++	++	++
LV Diastolic Function	+++	+	+
Aortic Morphology	++	+++	+++
Valve Function/Morphology	+++	++	++

TTE Box

Role: First-line imaging.

CMR Box

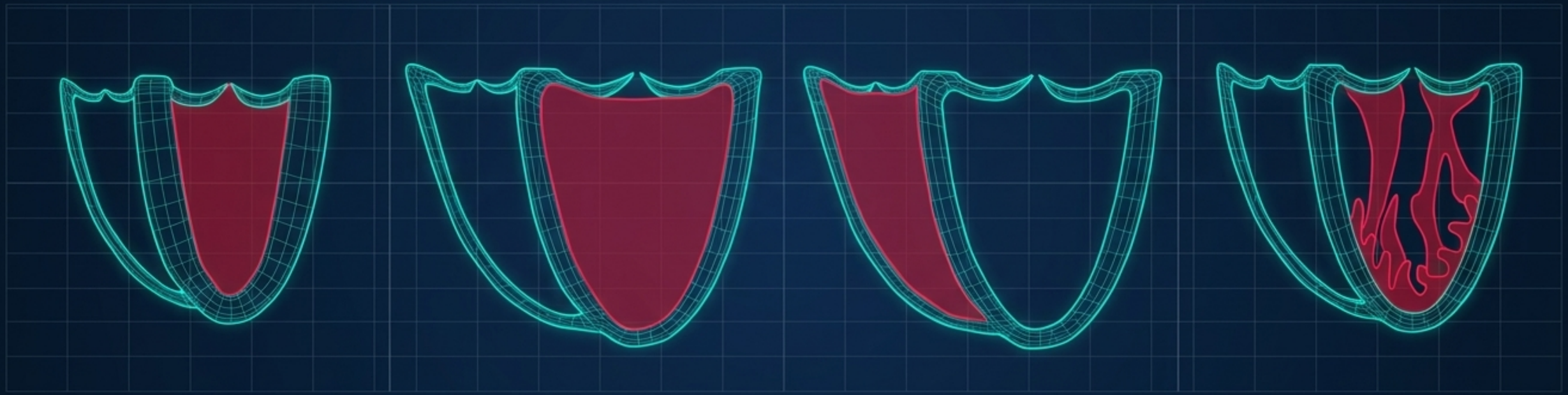
Role: Gold standard for structural/tissue confirmation.

CTA Box

Role: Definitive coronary and aortic mapping.

Decoding the Four Diagnostic ‘Gray Zones’

Vigorous exercise stimulates **adaptive structural remodeling** (EICR) that frequently overlaps with the imaging phenotypes of lethal cardiomyopathies.



Zone 1: Thick LV Walls

EICR vs. Hypertrophic Cardiomyopathy (HCM), Hypertensive Heart Disease, Infiltrative Disease

Zone 2: Dilated LV Chamber

EICR vs. Dilated Cardiomyopathy (DCM), Myocarditis, Toxic CMP.

Zone 3: Dilated RV Chamber

EICR vs. Arrhythmogenic RV Cardiomyopathy (ARVC).

Zone 4: Hypertrabeculation

EICR vs. LV Noncompaction Cardiomyopathy (LVNC).

Gray Zone 1: Differentiating Left Ventricular Wall Thickening

Indicators of EICR (Safe)

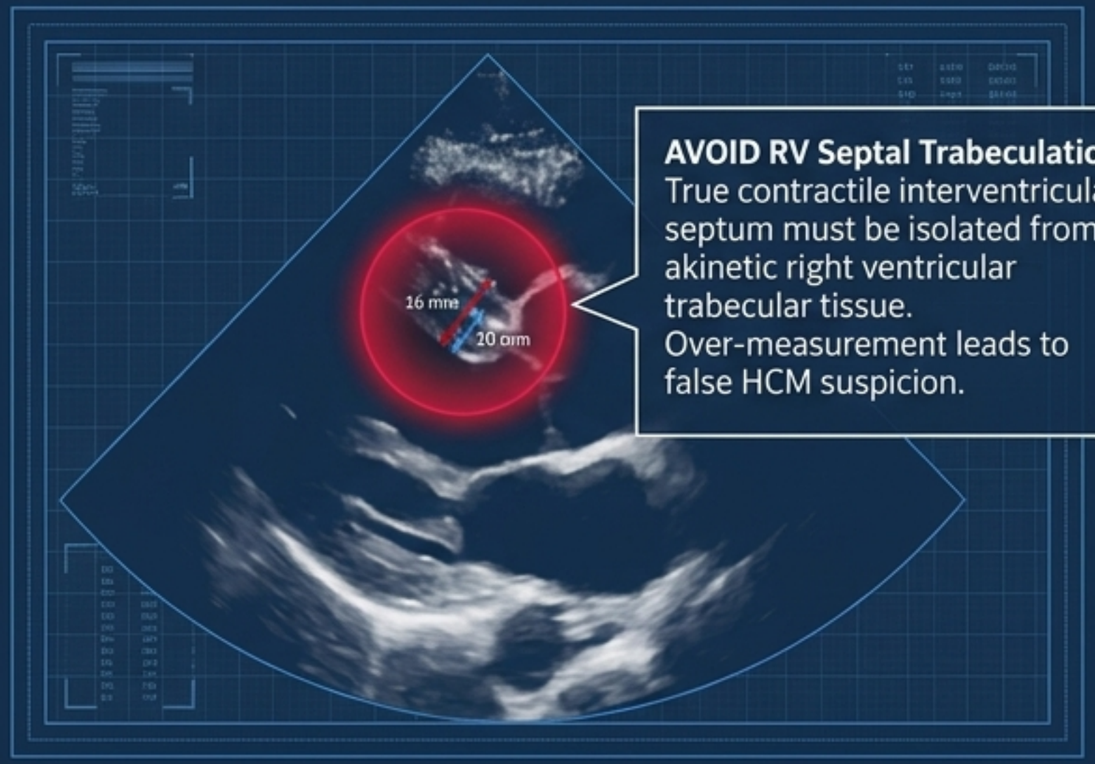
- **Morphology:** Mild, symmetric thickening. 11-13mm in Caucasians; up to 15mm in Black athletes.
- **Function:** Preserved or enhanced early diastolic relaxation velocities; normal LV longitudinal systolic strain.
- **Context:** Participates in sports with high isometric/pressure loads.

Red Flags for Pathology (HCM)

- **Morphology:** Asymmetric or focal hypertrophy; wall thickness >15mm; myocardial crypts; elongated mitral leaflets.
- **Function:** Impaired diastolic function; reduced myocardial strain.
- **Tissue (CMR):** Late Gadolinium Enhancement (LGE), interstitial expansion, patchy fibrosis at RV insertion sites.

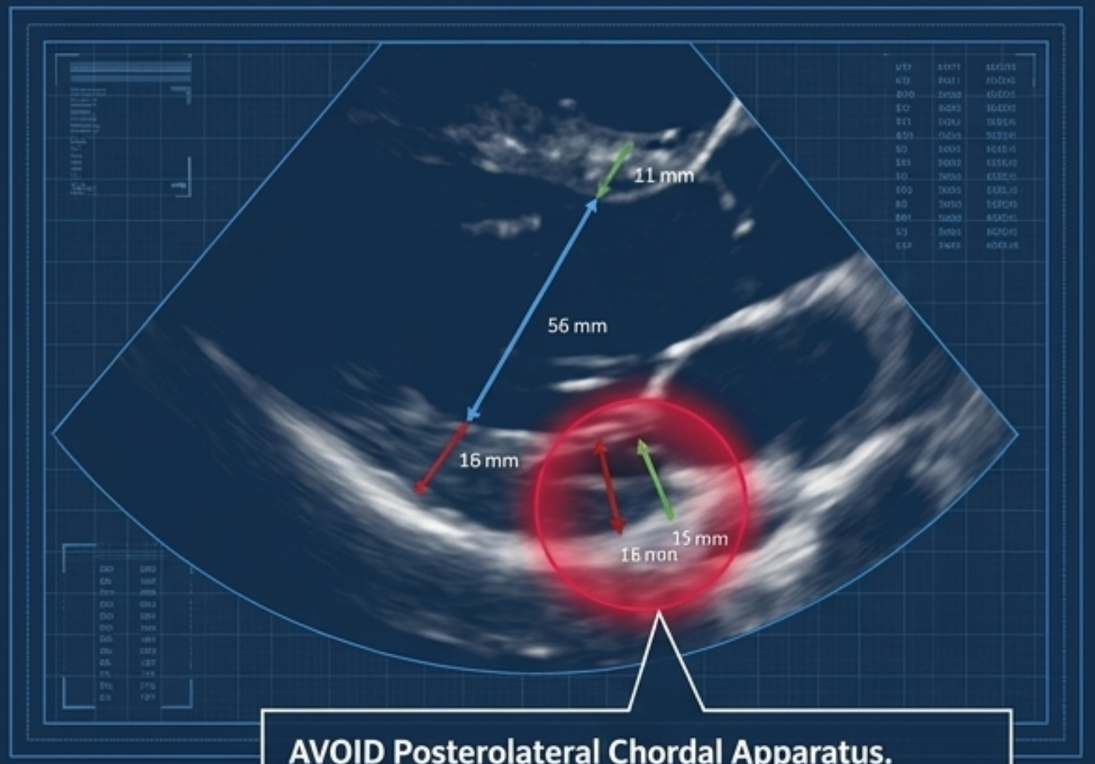
Measurement Pitfalls: False Positives in Wall Thickening

Pitfall 1: Septal Measurement



AVOID RV Septal Trabeculations.
True contractile interventricular septum must be isolated from akinetic right ventricular trabecular tissue.
Over-measurement leads to false HCM suspicion.

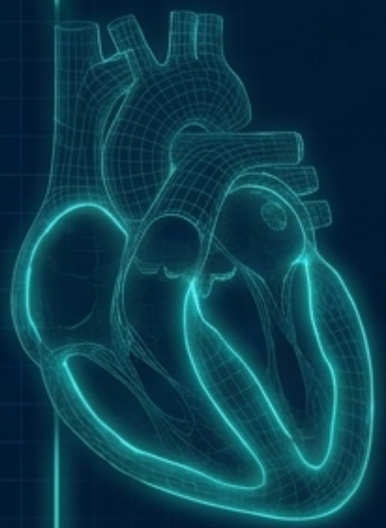
Pitfall 2: Posterior Measurement



AVOID Posterolateral Chordal Apparatus.
Erroneous inclusion of papillary muscle and chordal tissue creates the illusion of asymmetric posterior wall hypertrophy.

Gray Zone 2: Differentiating Left Ventricular Chamber Dilation

Indicators of EICR (Safe)



- **Morphology:** Dilation is accompanied by concomitant RV and biatrial dilation (global volume response).
- **Function:** Normal or supranormal diastolic indices; normal systolic strain.
- **Context:** Endurance sports (Isotonic physiology).

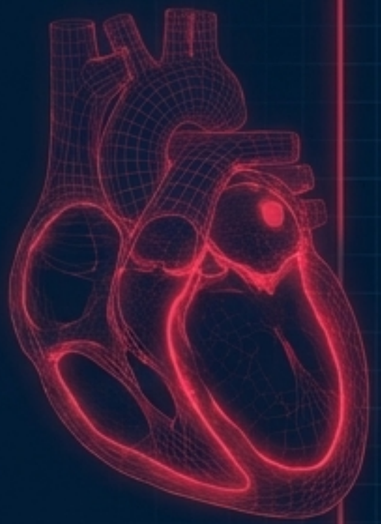
The EF Paradox

Mild reductions in resting LVEF (45-50%) can be normal in endurance athletes due to massive resting stroke volumes.

Pathology is indicated by a lack of contractile reserve during stress TTE/CMR.

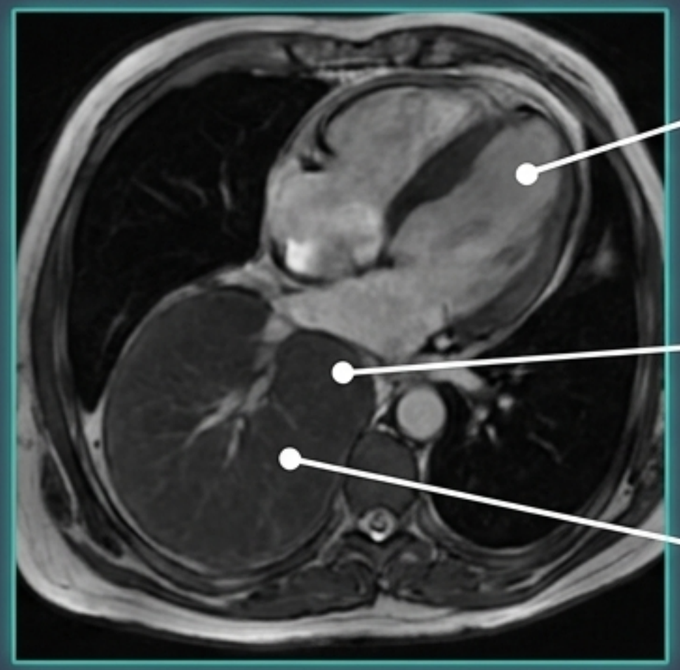
Red Flags for Pathology (DCM)

- **Morphology:** Isolated LV dilation without concomitant right-heart adaptation.
- **Function:** Regional wall motion abnormalities; impaired diastolic function; less-negative global strain.



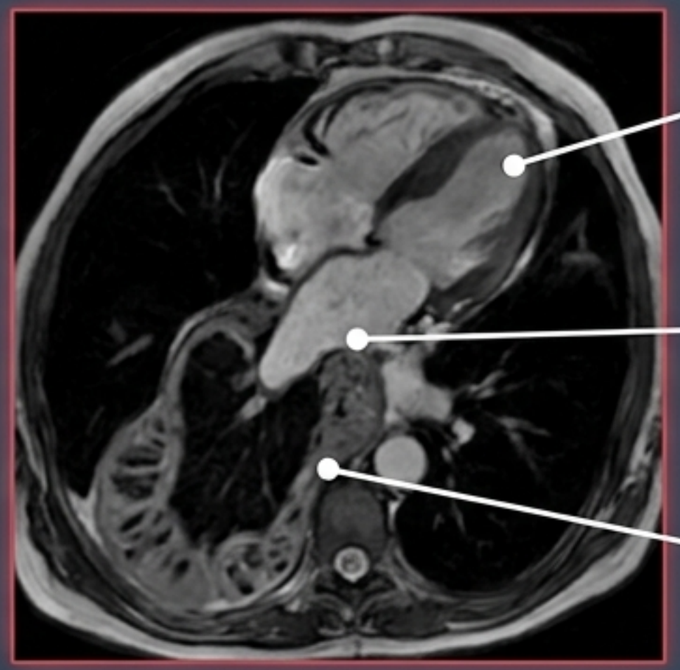
Gray Zone 3: Differentiating Right Ventricular Chamber Dilation

Healthy Endurance Athlete



- Global dilation proportional to LV
- Smooth, minimally trabeculated RV free wall
- Preserved systolic reserve.

Gene-Positive ARVC



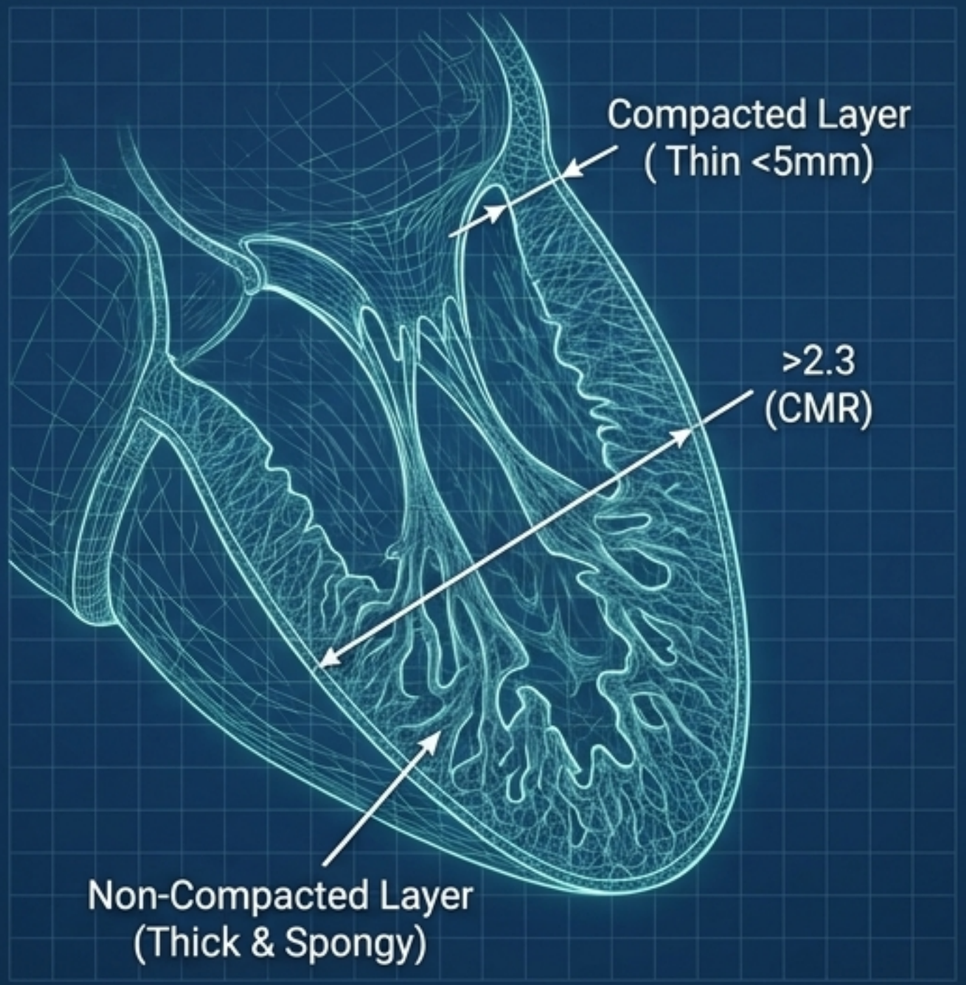
- Isolated RV dilation
- Sacculation and focal aneurysmal dilation of the mid-distal RV free wall
- Segmental hypokinesis.

Key Clinical Rule: Because TTE has significant limitations characterizing complex RV anatomy, CMR is required for almost all athletes with RV dilation of unclear etiology.

Gray Zone 4: Differentiating Left Ventricular Hypertrabeculation

Indicators of EICR (Safe)

- **Morphology:** Prominent trabeculations but with normal underlying compacted wall thickness (>5mm).
- **Function:** Normal LV ejection fraction and normal longitudinal strain.
- **Context:** Frequent in Black athletes and endurance competitors responding to chronic preload increases.

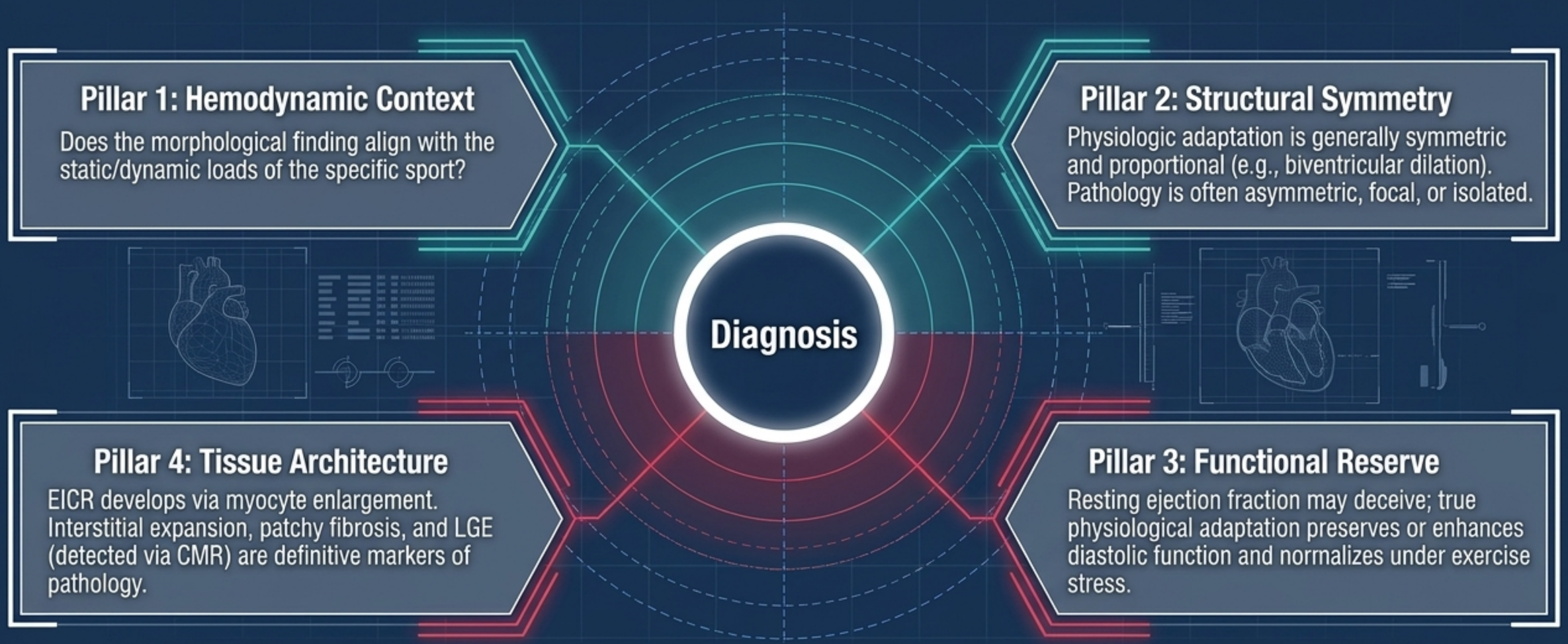


Red Flags for Pathology (LVNC)

- **Morphology:** Noncompacted to compacted layer ratio >2.0 (TTE) or >2.3 (CMR). Very thin compacted layer (<5mm).
- **Tissue:** Presence of Late Gadolinium Enhancement (LGE) on CMR.
- **Clinical Context:** Malignant ventricular arrhythmias or family history of sudden cardiac death.

The Masterclass Synthesis: A Holistic Approach to the Gray Zones

Core Principle: Differentiation of the athlete's heart from pathology is never based on a single measurement cut-off. It requires the integration of four pillars:



Post-Screening Logic: When to Initiate Multimodality Imaging

Medical History: Exertional symptoms, prior unexplained syncope, family history of SCD.

Physical Exam: Pathologic murmur, hypertension, Marfan syndrome features.

ECG Findings: Complete LBBB, T-wave inversions, pathological Q-waves, profound ST depression.

First-Line: Transthoracic Echocardiography (TTE)

Pathology Definitively Excluded → No further imaging.

Pathology Inconclusive by TTE → Escalate to CMR, CTA, or Exercise Stress Imaging.

Pathology Identified → Utilize CMR/CTA for confirmation and prognostic risk stratification.

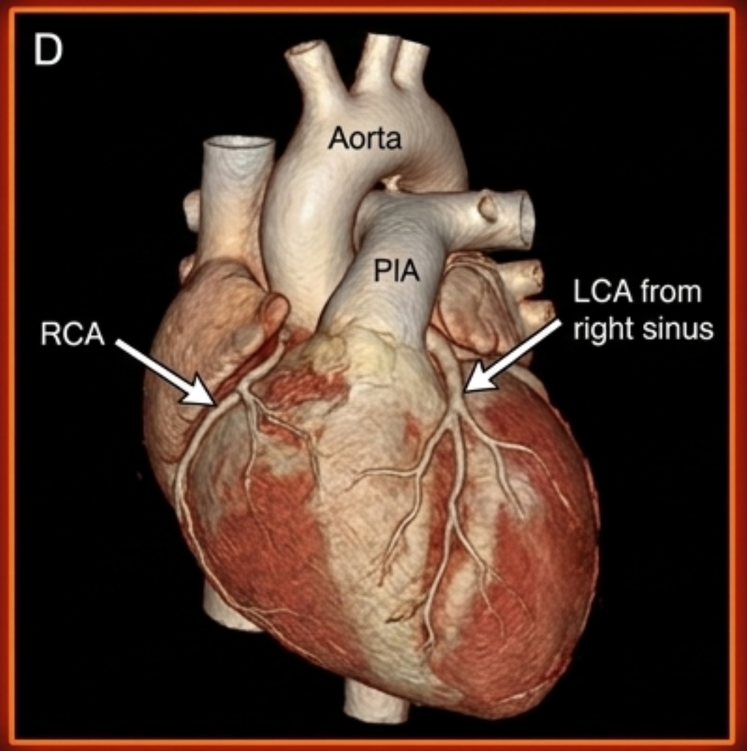
The Symptomatic Athlete: Evaluating Exertional Chest Discomfort

Clinical Rule: Chest discomfort during exertion in a CA is primarily non-cardiac, but cardiac etiologies must be excluded due to high SCD risk.

Step 1: First-Line TTE Targets.
Focus imaging specifically on ruling out: Hypertrophic cardiomyopathy (with/out obstruction), congenital/acquired valve stenosis, and anomalous proximal coronary anatomy.

Step 2: Exercise Stress.
Perform maximal effort-limited exercise testing.
Warning: Rapid heart rate recovery in highly trained athletes often renders immediate post-exercise echocardiography non-diagnostic for ischemia.

Step 3: Escalation for Coronary Anomalies.
If TTE cannot definitively confirm normal coronary origins, immediately escalate to CTA or CMR.



Cardiac CT reconstruction showing a high-risk anomalous left coronary artery originating from the right sinus.

The Symptomatic Athlete:
Evaluating Syncope & Arrhythmias

The Bifurcation: Context of the Event

Post-Exertional / Resting

Syncope occurring seconds-to-minutes after abrupt exercise termination, or resting palpitations in bradycardic athletes.

Action: Neurally-mediated/benign. No noninvasive imaging required.

Exertional / Peak Effort

Syncope abruptly during exercise (often with dramatic collapse), or palpitations that intensify during stress.

Action: Pathologic until proven otherwise.

* **Required Imaging:** Comprehensive TTE to exclude obstructive outflow pathology, arrhythmogenic cardiomyopathies, and Ebstein's anomaly (if pre-excitation is present).
* **Advanced Imaging:** CMR is mandatory if complex ventricular arrhythmias are documented, to explicitly evaluate for underlying myocardial scar.

The Symptomatic Athlete: Dyspnea & Performance Decrement

Definition: Breathlessness at previously tolerable exercise intensities without change in training regimen.

Step 1: Non-Cardiac Exclusion

Initially evaluate for reactive airway disease, vocal fold disorders, or dysfunctional breathing. If symptoms resolve with empiric respiratory therapy, imaging is not required.

Step 2: Cardiovascular Evaluation (TTE)

If symptoms persist, perform a comprehensive TTE and maximal cardiopulmonary exercise testing to exclude structural volume/pressure overloads.

Step 3: Advanced Escalation

Consider CMR/CTA only if specific myocardial or coronary pathology is myocardial or coronary pathology is suspected based on initial TTE/ECG results.

The Aortic Exception: Strict Limits to Physiologic Dilation

Core Principle: While extreme ventricular dilation can be physiologic, the aortic root and ascending aorta do not substantially dilate in response to exercise training.

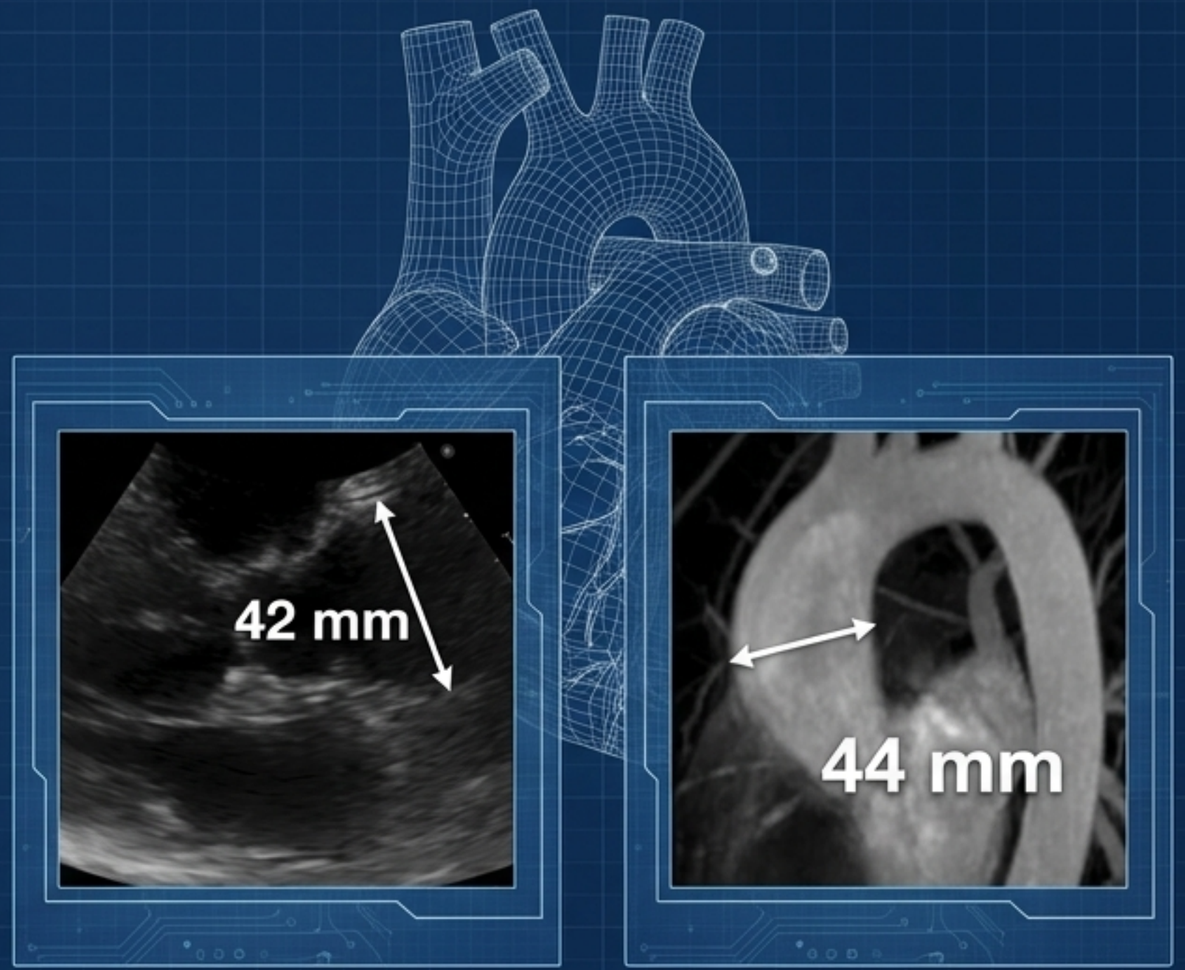
The Hard Cut-Points:

Men: >= 40 mm

Women: >= 34 mm

Clinical Protocol: Aortic dimensions exceeding these sex-specific cut-points are exceedingly rare (0.5-1.8% prevalence) and cannot be assumed to be EICR, even in athletes over 7 feet tall.

Action: Any measurement exceeding these thresholds via TTE mandates tomographic imaging (gated CTA or CMR) to evaluate for underlying aortopathy.



The Master Diagnostician’s Blueprint

Rule 1: Context Dictates Morphology

Never interpret chamber size or wall thickness in a vacuum. Always map findings against the athlete’s sport physiology, sex, ethnicity, and training phase.

Rule 2: Symmetry is Safe; Focal is Fatal

Exercise-induced remodeling causes **global, proportional changes** (biventricular dilation, symmetric mild thickening). **Asymmetry, focal aneurysms**, or **isolated regional dysfunction** signal pathology.

Rule 3: Modality Dictates Certainty

TTE is the ultimate triage tool, but it is blind to tissue characterization and complex RV/Coronary anatomy. Do not hesitate to deploy **CMR** or **CTA** to definitively separate adaptation from **lethal disease**.