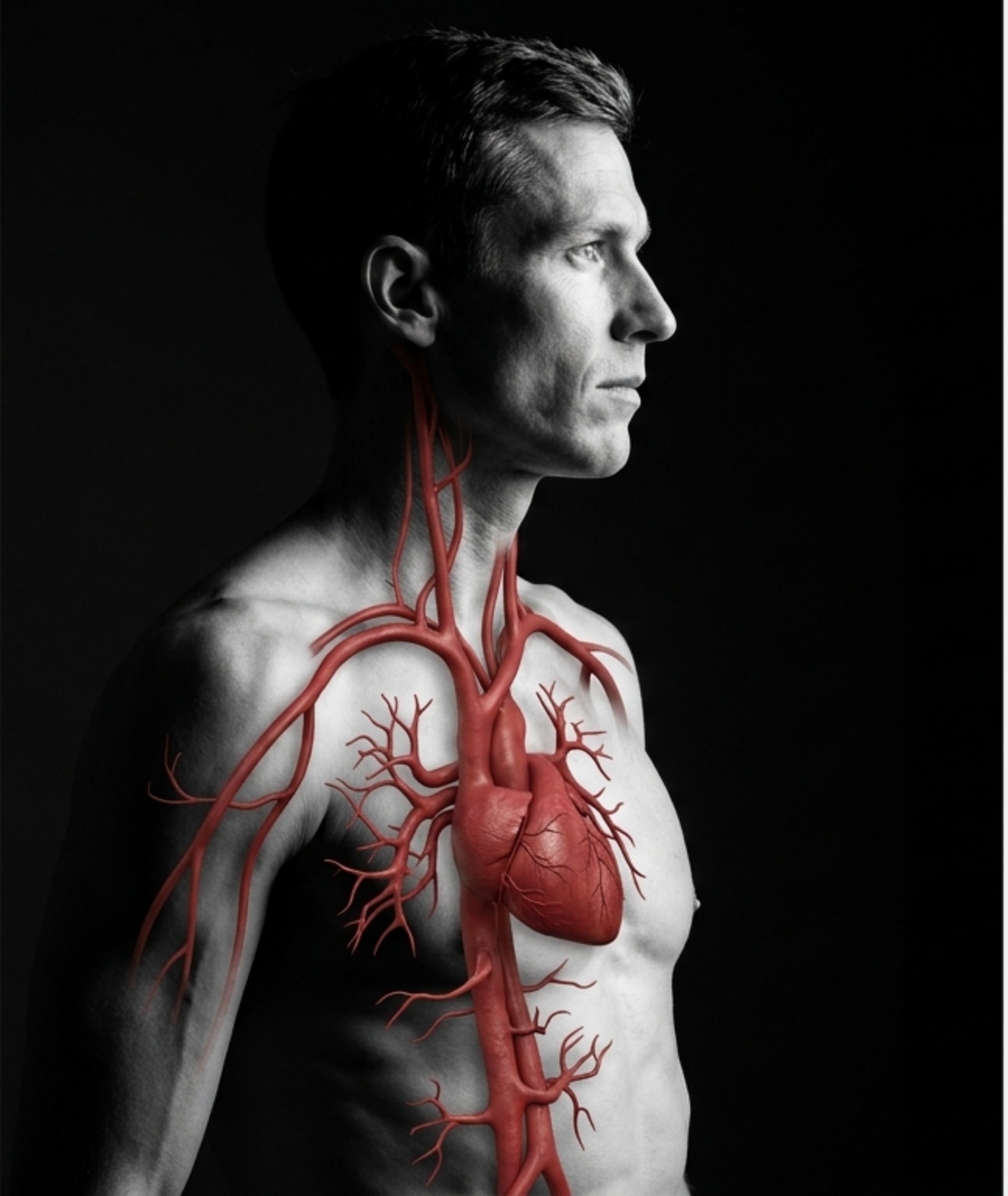


The Masked Athlete

Diagnosing Hidden Cardiovascular Risk in the Elite and Endurance Population

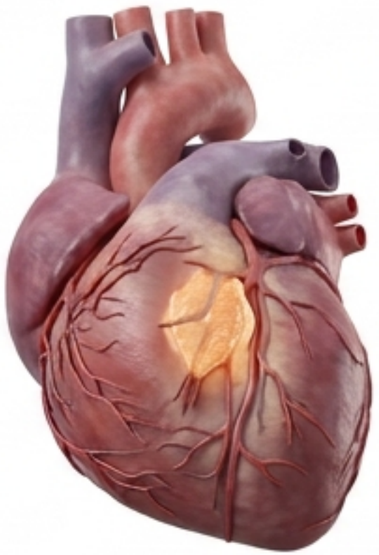
A Clinical and Mechanistic Synthesis of Pain Perception, Collateral Circulation, and Advanced Imaging

The Paradox of Athletic Adaptation

Aerobic Protection

Aerobic fitness is a powerful protector against atherosclerotic disease. Expected adaptations include:

- Enhanced vagal tone and low resting heart rate
- High stroke volume and efficient oxygen extraction
- Superior peripheral vascular conductance



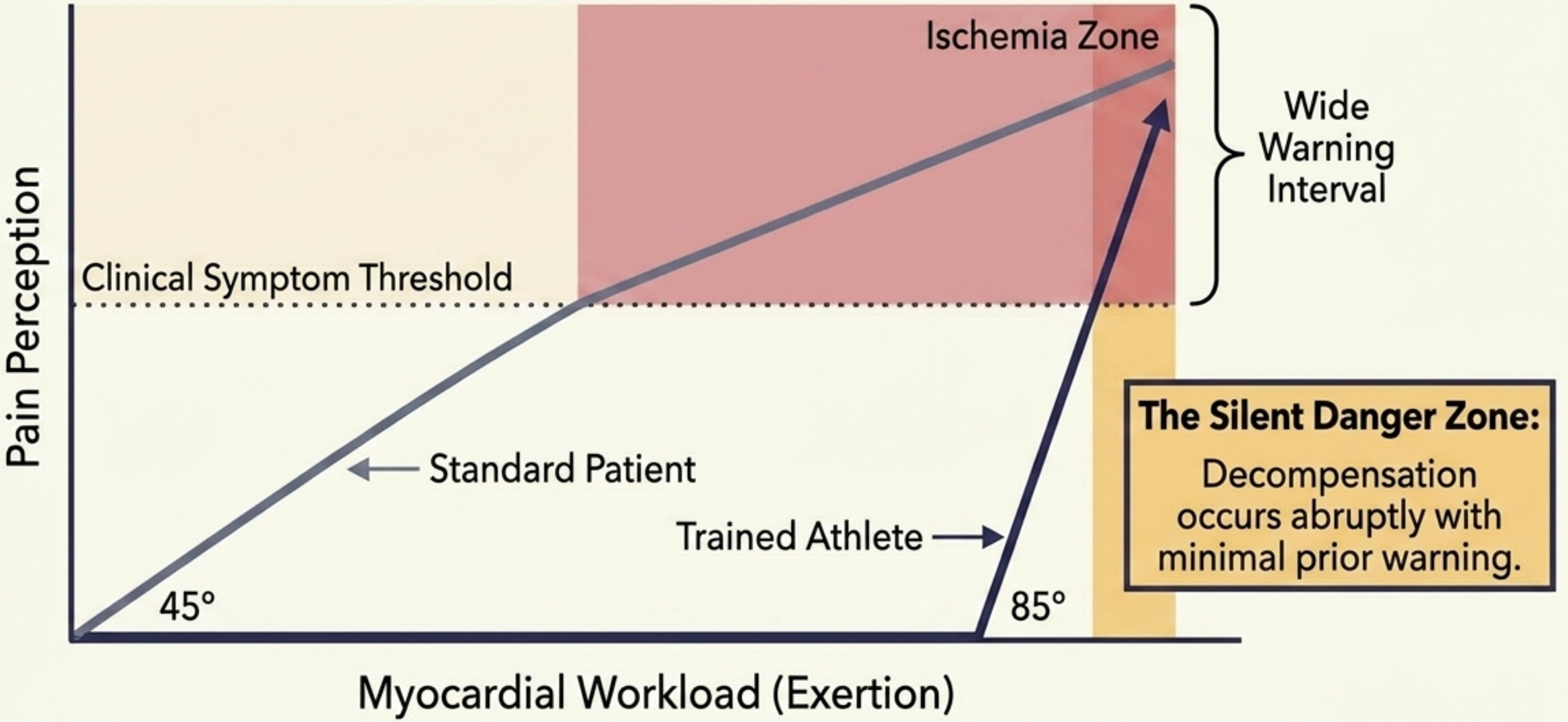
The Diagnostic Illusion

A non-trivial fraction of highly trained athletes harbor significant coronary lesions, anomalous arteries, or inherited cardiomyopathies.

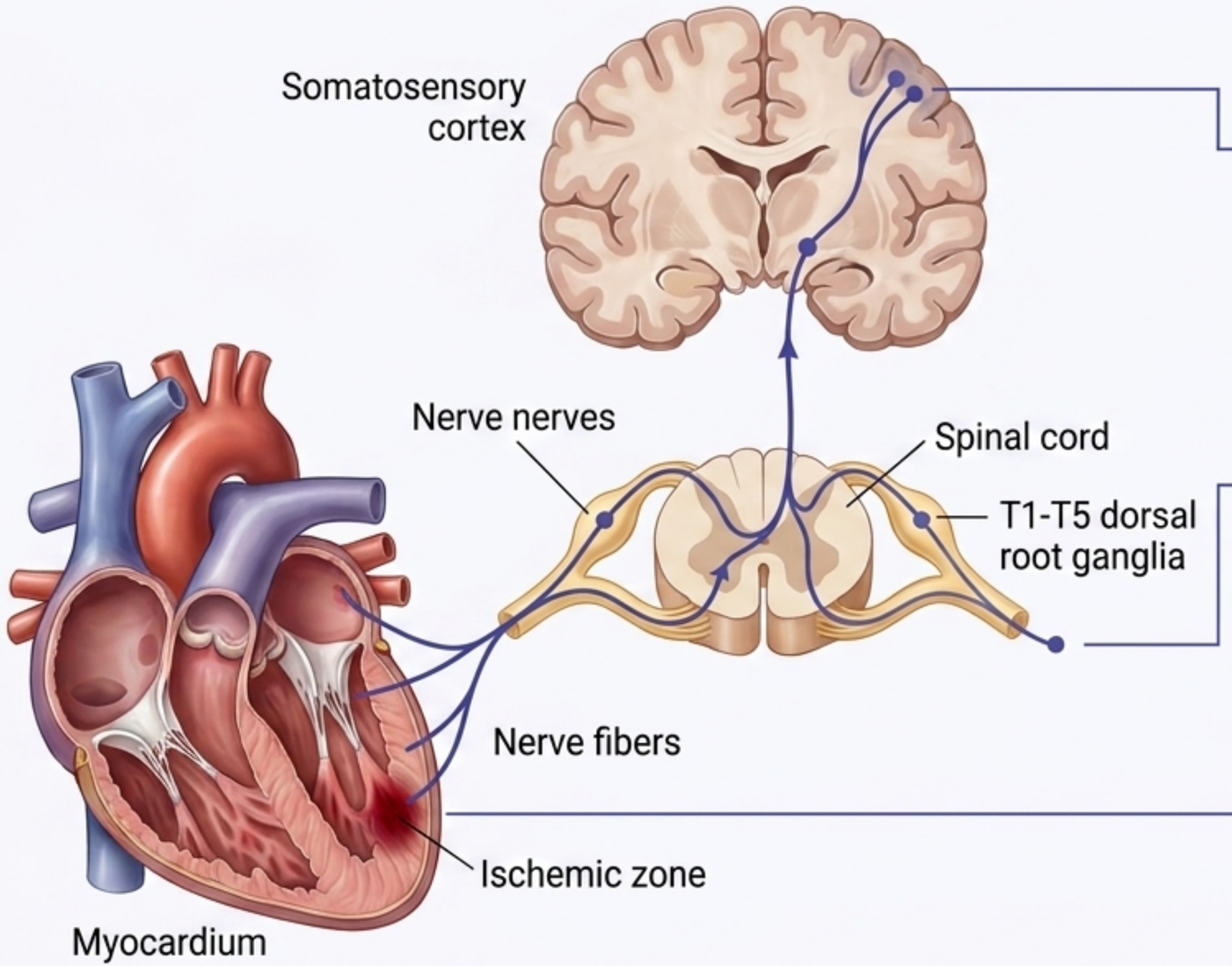
The core clinical danger: The very adaptations that improve athletic performance—autonomic modulation, enhanced cardiac efficiency, and collateral growth—artificially elevate the threshold at which ischemia becomes clinically obvious. The pathology is masked by the fitness.

Collapsing the Warning Interval

In athletes, neural and physiological inputs suppress anginal pain, narrowing the safety margin.



The Anatomy of Silent Ischemia



1. Biochemical Triggers

Ischemia triggers an anaerobic shift, releasing Adenosine (binding to A1 receptors), Bradykinin, and local acidosis (stimulating ASIC3/TRPV1 channels).

2. Neural Convergence

Sympathetic afferents converge with somatic inputs at T1-T5 spinal levels, generating classic "referred pain" in the chest, shoulder, and jaw.

3. The Disconnect

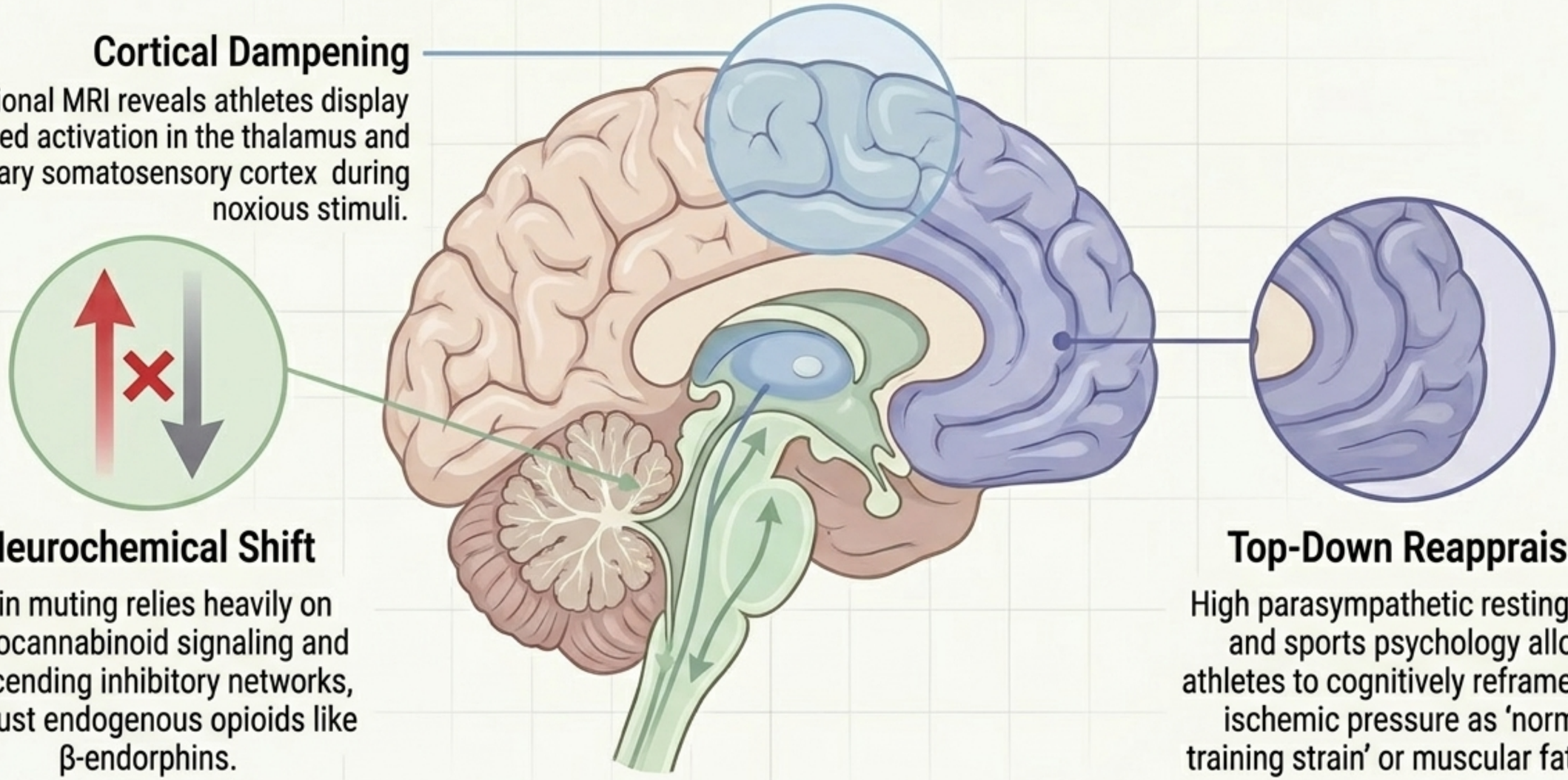
Between 50% and 75% of ischemic episodes in established CAD cohorts are clinically silent. In athletes, the subjective experience of this objective ischemia is fundamentally altered.

Exercise-Induced Hypoalgesia (EIH) & Cognitive Reappraisal

Endurance training actively intercepts and mutes ascending ischemic pain signals.

Cortical Dampening

Functional MRI reveals athletes display attenuated activation in the thalamus and primary somatosensory cortex during noxious stimuli.



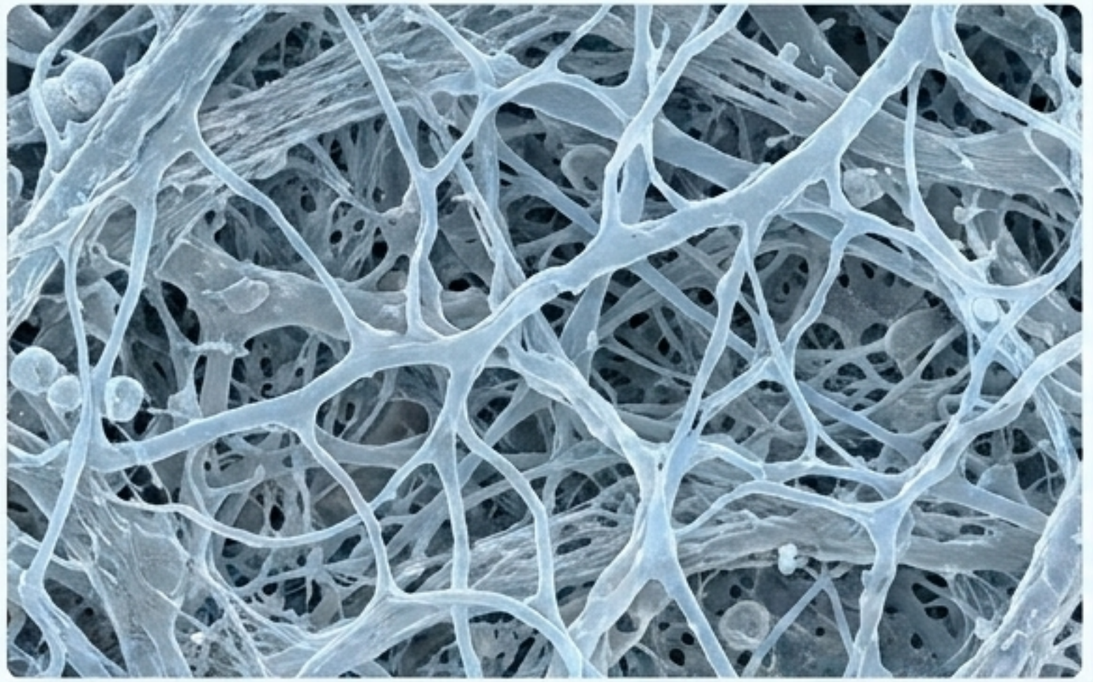
Neurochemical Shift

Pain muting relies heavily on endocannabinoid signaling and descending inhibitory networks, not just endogenous opioids like β-endorphins.

Top-Down Reappraisal

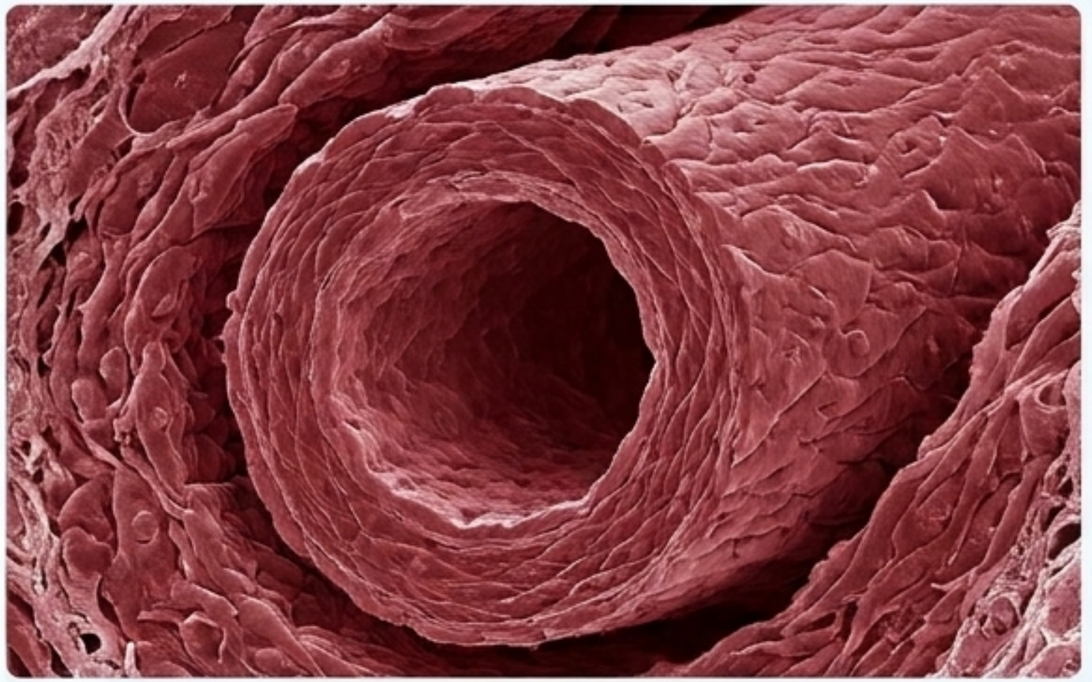
High parasympathetic resting tone and sports psychology allow athletes to cognitively reframe early ischemic pressure as 'normal training strain' or muscular fatigue.

The Collateral Illusion: Angiogenesis vs. Arteriogenesis



Angiogenesis

- Hypoxia-driven (HIF-1α / VEGF).
- Improves local capillary density.
- Lacks the muscular media required to handle bulk arterial flow.
- Cannot compensate for epicardial stenosis.

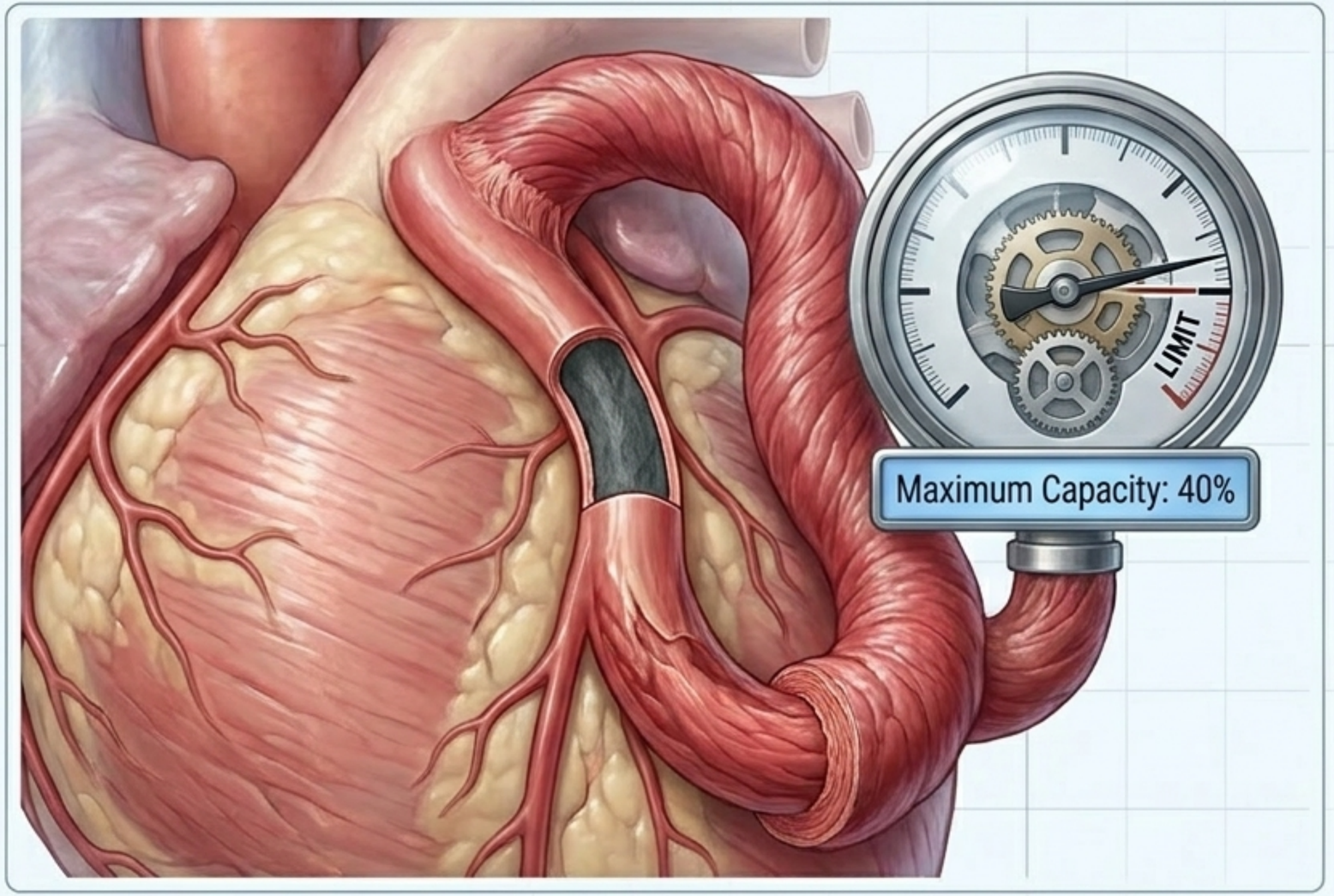


Arteriogenesis

- Shear-stress driven remodeling process.
- Pre-existing anastomoses remodel into thick, muscular conducting arteries.
- Arterioles enlarge up to 20-fold in luminal diameter.
- Intense aerobic training is a potent stimulus, increasing Collateral Flow Index.

Protective, But Not Curative

Mature collateral arteries offer significant but limited functional compensation.



The Collateral Ceiling

Even in the most extensively collateralized hearts, mature collaterals typically deliver only 25–40% of normal antegrade flow.

The Mechanistic Limit

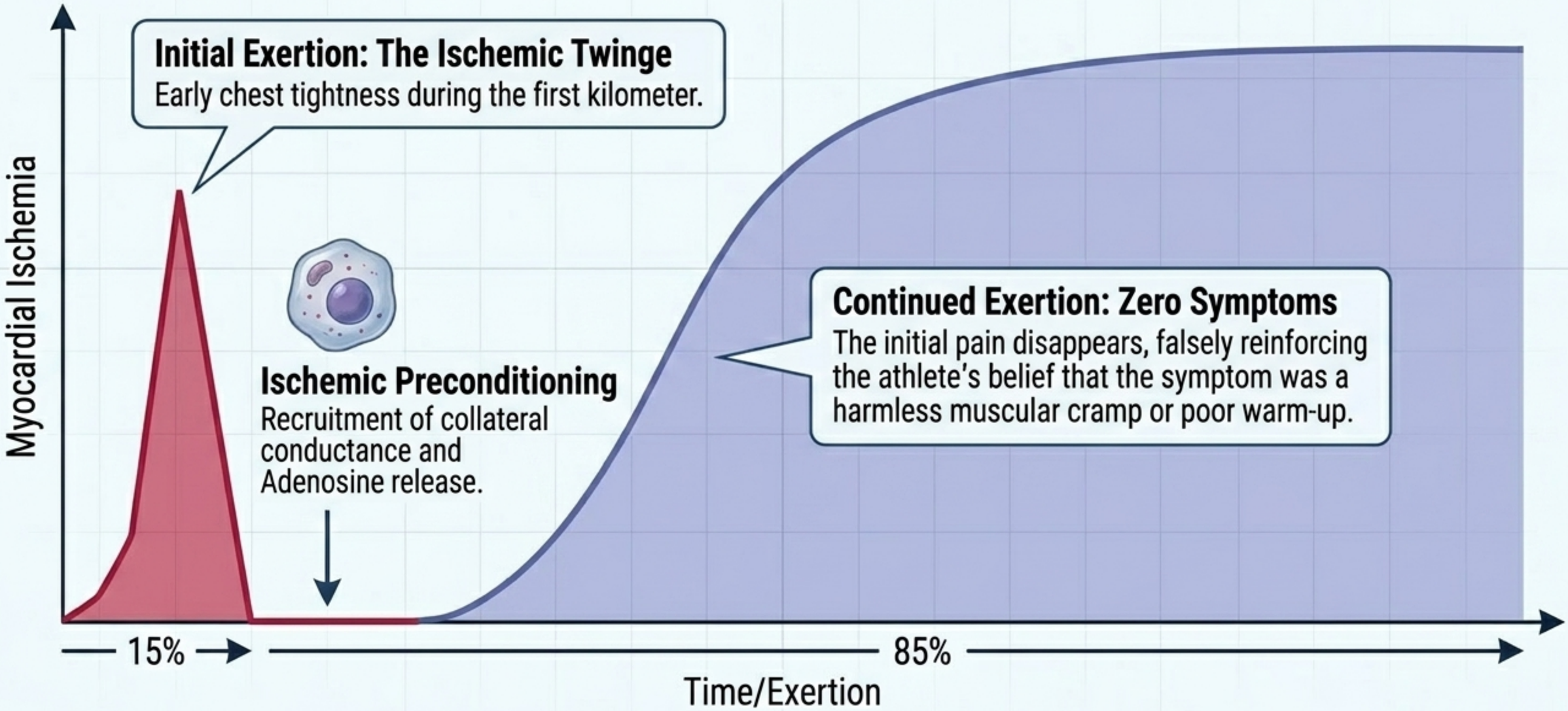
As the collateral lumen expands, the pressure gradient falls. Fluid shear stress drops below the growth threshold, and arteriogenesis permanently halts.

The Clinical Result

An athlete with critical multi-vessel disease remains completely asymptomatic at rest or moderate exertion, but suffers abrupt ischemic decompensation at peak heart rates when the 40% ceiling is breached.

The False All-Clear: Warm-Up Angina

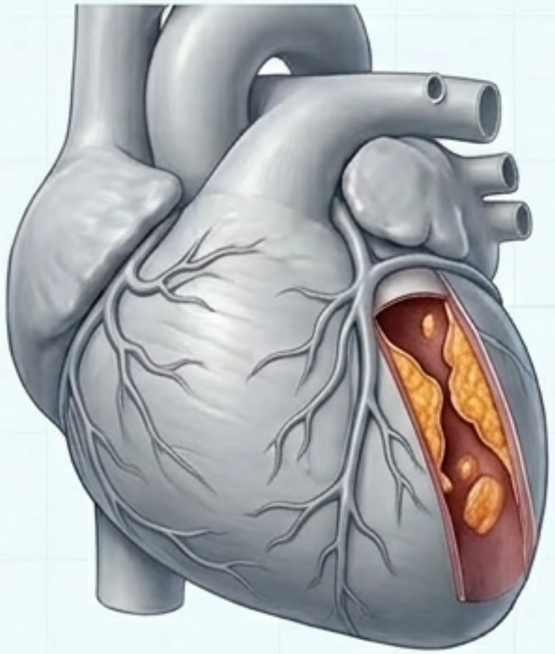
Why early warning signs vanish during continued exertion.



The Masked Phenotypes: What Lies Beneath

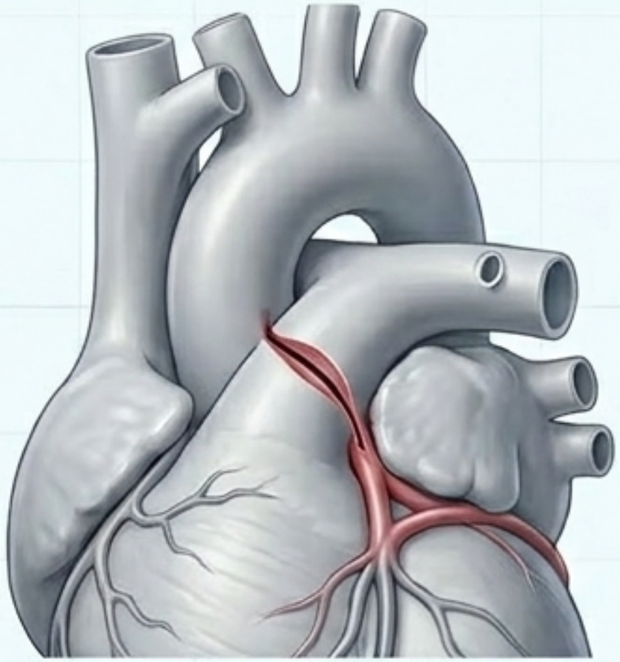
Master athletes and young competitors can harbor lethal, asymptomatic conditions.

Coronary Artery Disease (CAD)



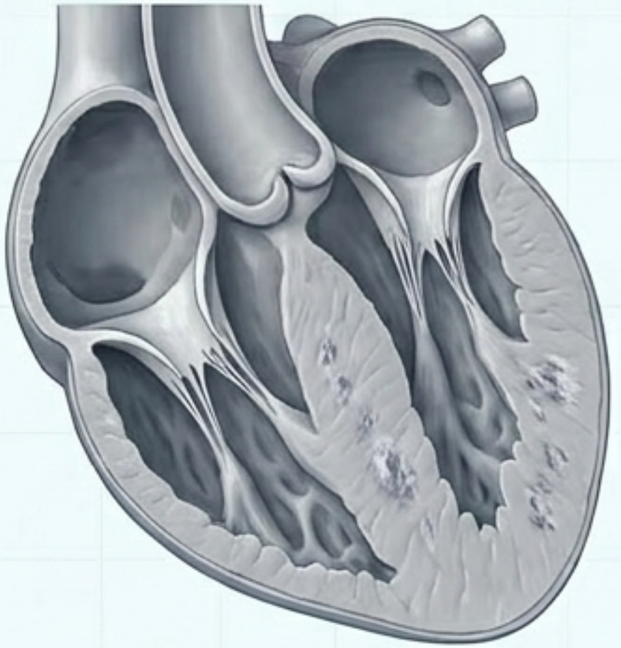
In athletes over 35, atherosclerotic CAD is the leading cause of exercise-related sudden cardiac death. Up to 16% of asymptomatic master athletes harbor silent ischemia.

Anomalous Aortic Origin (AAOCA)



Disproportionately lethal in young athletes. Expansion of great vessels during heavy exertion compresses the anomalous artery, collapsing the slit-like orifice.

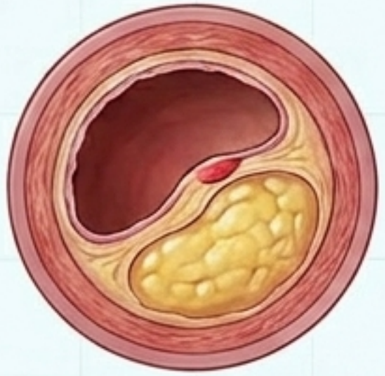
Hypertrophic Cardiomyopathy (HCM)

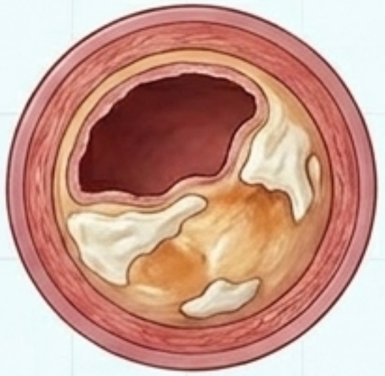


HCM and inherited arrhythmic substrates must be differentiated from physiological "athlete's heart" via MRI, identifying asymmetric hypertrophy and fibrotic scarring.

The Coronary Artery Calcium (CAC) Paradox

Lifelong male endurance athletes exhibit higher total CAC scores, requiring a paradigm shift in interpretation.





Vulnerable Plaque

- Lipid-rich, non-calcified, thin-cap.
- Highly prone to rupture.
- Associated with sedentary / high-risk populations.

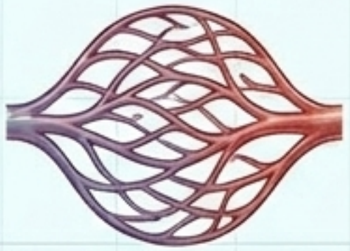
Calcified Plaque

- Dense, highly attenuated, healed.
- Stable morphology.
- Associated with lifelong male endurance athletes (≥9 METs).

Clinical Rule: Calcified plaque in the lifelong athlete is often the radiographic signature of healed atherosclerosis. Do not fixate on the total CAC score. Advanced imaging must assess non-calcified/mixed plaque burden burden and high-risk morphology.

The Female Athlete Divergence

Diagnostic frameworks derived from male cohorts risk substantial misclassification in women.



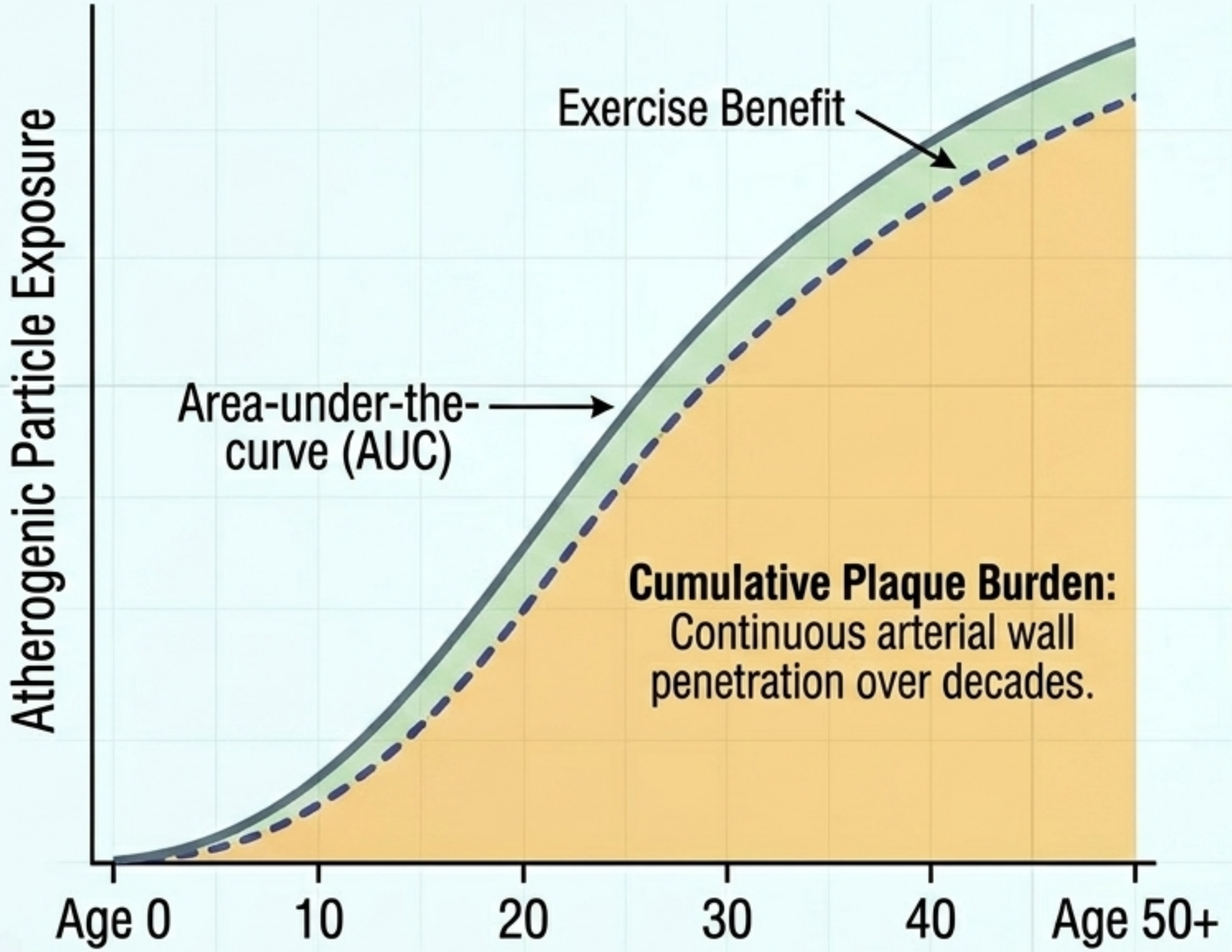
Male Athlete Phenotype

- **Macro-vascular Focus:** Disease predominantly manifests in the large epicardial coronary arteries.
- **CAC Correlation:** Strong positive association between lifelong exercise dose and increased calcified plaque (MARC cohort).

Female Athlete Phenotype

- **Micro-vascular Focus**: Disproportionately represented in INOCA and MINOCA, where microvascular dysfunction and vasospasm are central mechanisms.
- **The CAC Disconnect:** No association between lifelong exercise dose and CAC in master female endurance athletes.
- **Atypical Presentation:** Frequently present with exertional dyspnea, extreme fatigue, or epigastric discomfort rather than traditional angina.

Fit But Not Immune: Lifetime ApoB and Lp(a)



The Biological Reality

Atherosclerotic risk is a function of cumulative ApoB-particle exposure across the life course. High fitness does not eliminate lifetime risk.

Genetic Factors

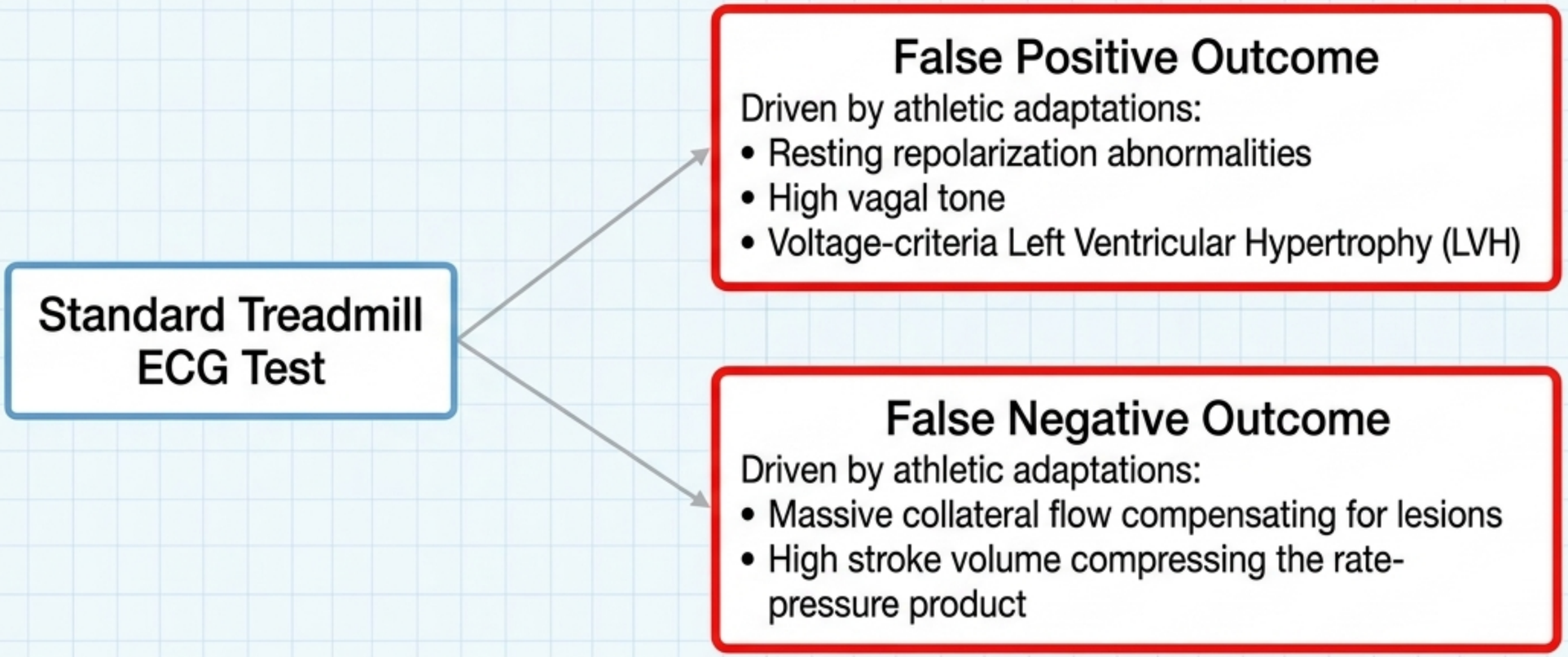
Elevated Lipoprotein(a) affects ~20% of the population and is completely unmodifiable by exercise.

Clinical Imperative

ApoB and Lp(a) must be measured at least once in master athletes. The absence of conventional risk factors does not exclude clinically significant atherosclerosis.

The Failure of Submaximal Stress Testing

Why conventional exercise ECGs actively mislead in the athletic population.



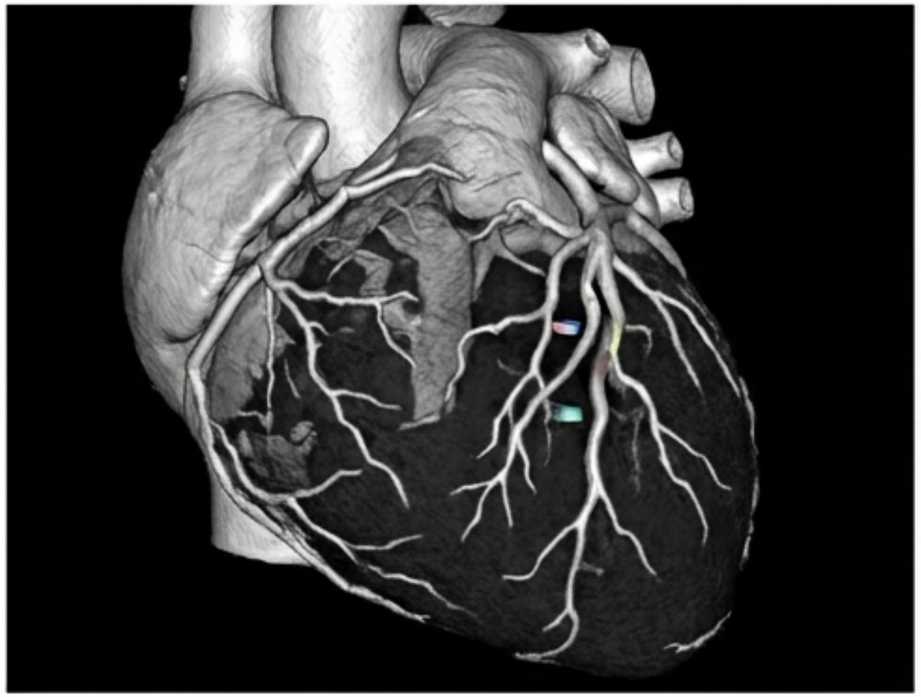
The Trap of 85%: Submaximal protocols that terminate at 85% of an age-predicted maximum systematically miss athletes. An athlete's true ischemic threshold often lies near 100% of an age-exceeding maximum.

Evaluating Modalities: The Athlete Context

Modality	Pooled Sensitivity	Pooled Specificity	The Athlete Caveat
Exercise ECG	≈ 0.58	≈ 0.62	False positives from LVH/vagal tone; False negatives from collaterals. Submaximal endpoints fail.
Stress Echo	≈ 0.85	≈ 0.82	Suboptimal acoustic windows in highly muscular individuals.
SPECT	≈ 0.73	≈ 0.83	Balanced ischemia in 3-vessel disease may falsely normalize.
Stress CMR	≈ 0.90	≈ 0.85	The gold standard for differentiating HCM from physiological athlete's heart; quantifies fibrosis.
CCTA	≈ 0.95	≈ 0.79	The anatomical gold standard for AAOCA and analyzing plaque morphology.

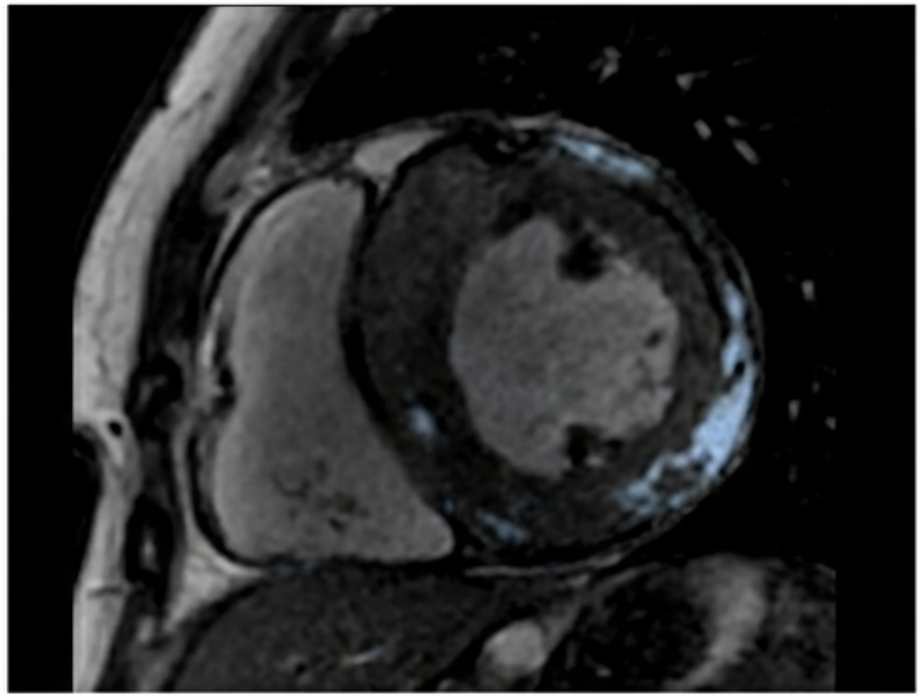
Seeing Through the Mask: Advanced Imaging

The pivot to anatomical and tissue-characterizing imaging as the definitive diagnostic solution.



CCTA (Coronary CT Angiography)

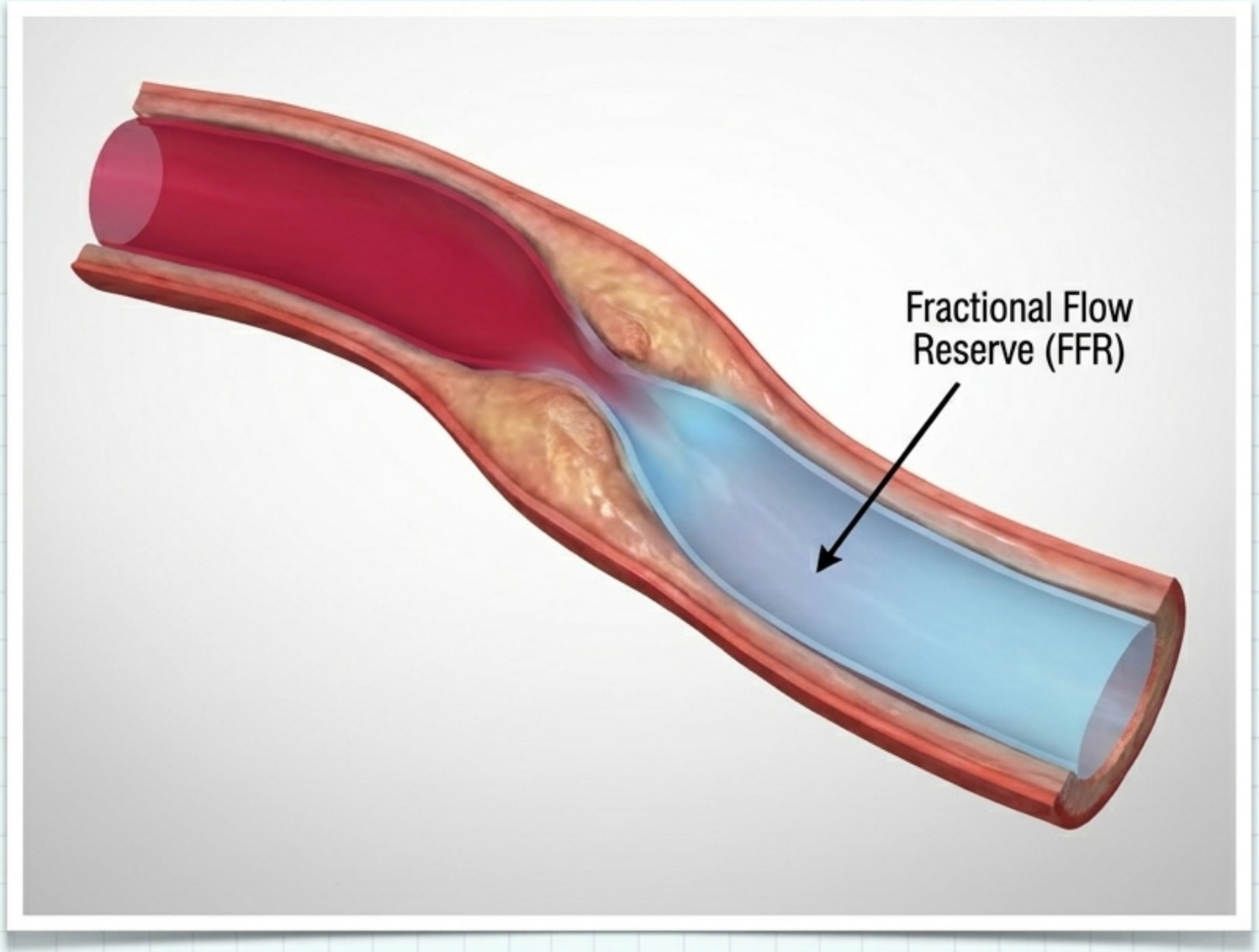
The most sensitive widely available test for obstructive epicardial disease. Accurately characterizes plaque morphology and is the absolute gold standard for diagnosing anomalous coronary arteries (AAOCA).



Stress CMR (Magnetic Resonance)

Essential for phenotype-negative gene carriers. Detects subtle myocardial crypts, focal fibrosis, or perfusion abnormalities long before frank hypertrophy appears on an echo.

Functional Lesion Assessment: Moving Beyond Anatomy



The Dilemma

When CCTA reveals an intermediate stenosis, the discrepancy between anatomical disease and functional ischemia is highly amplified in athletes due to collateral conductance.

The Solution: CT-FFR

CT-derived Fractional Flow Reserve provides pressure-derived ischemic indices non-invasively. Generates a mapped pressure gradient without a catheter.

Clinical Impact

NXT and FAME trials confirm physiology-guided assessment drastically reduces unnecessary stenting. Interventions are reserved only for lesions actively causing ischemia.

Redefining the Ischemic Equivalents

Because classic retrosternal pain is masked, clinicians must treat the following atypical signs as serious until proven otherwise:

Unexplained Performance Decline

A sustained, mysterious drop in training pace, peak power output, or inability to hit known threshold metrics.

Disproportionate Exertional Dyspnea

Shortness of breath that does not match the exercise intensity, particularly if it accelerates rapidly in onset.

Atypical Discomfort

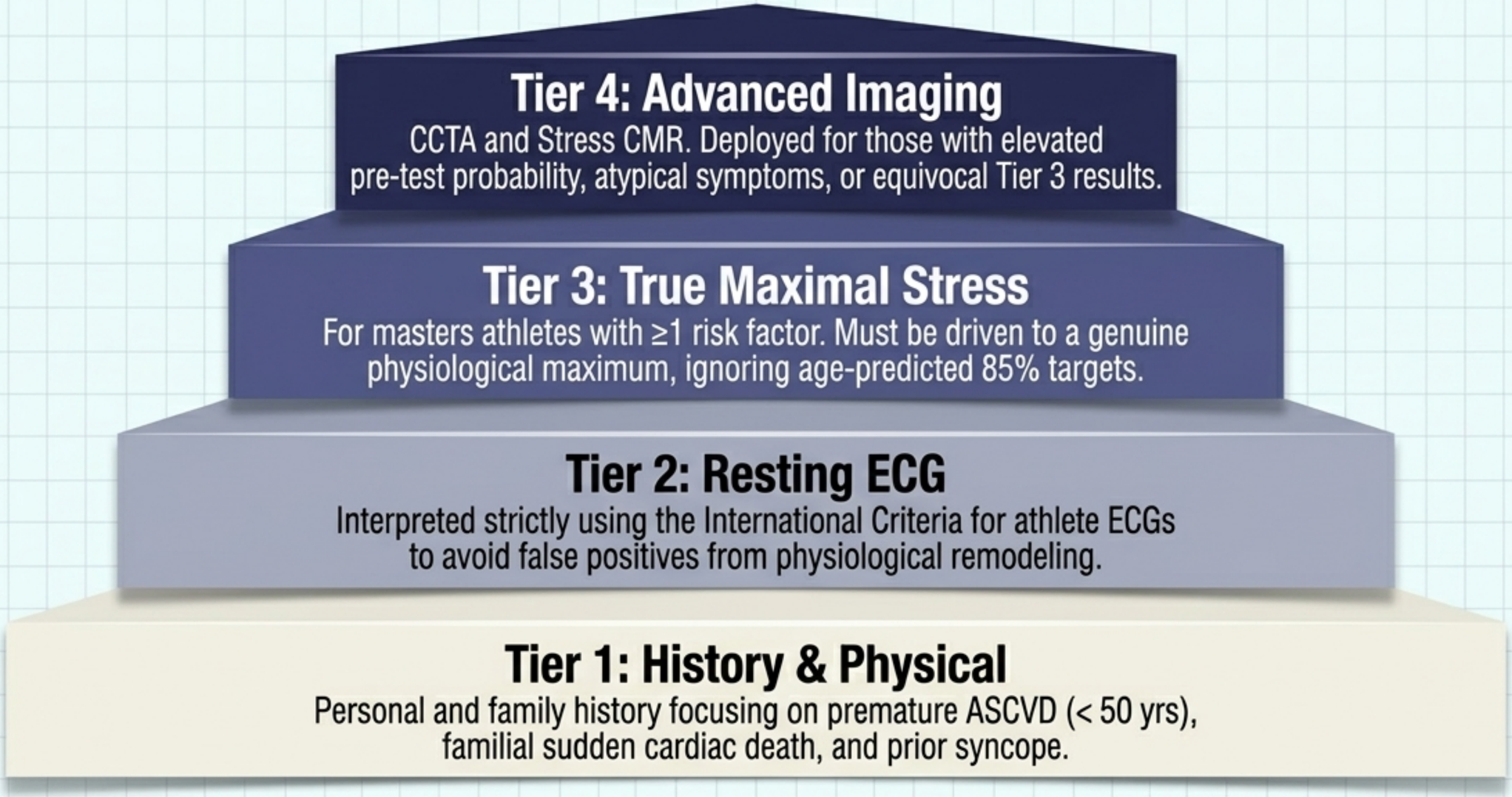
Mild epigastric, jaw, or interscapular pressure occurring specifically during high-intensity intervals that resolves upon resting.

Exertional Presyncope/Syncope

A primary, critical red flag for AAOCA, Hypertrophic Cardiomyopathy, or malignant arrhythmic substrates.

The Tiered Screening Framework (ESC/AHA 2025)

Escalating diagnostic rigor tailored to the asymptomatic athlete.



The Reversal Priority: Managing Coronary Disease

A sequential protocol prioritizing biological resolution before mechanical intervention.

Step 3: Mechanical Intervention (Secondary)

Stenting or surgical revascularization. Reserved strictly for ischemia-causing lesions that completely fail to stabilize or resolve via WFPB dietary and medical intervention.

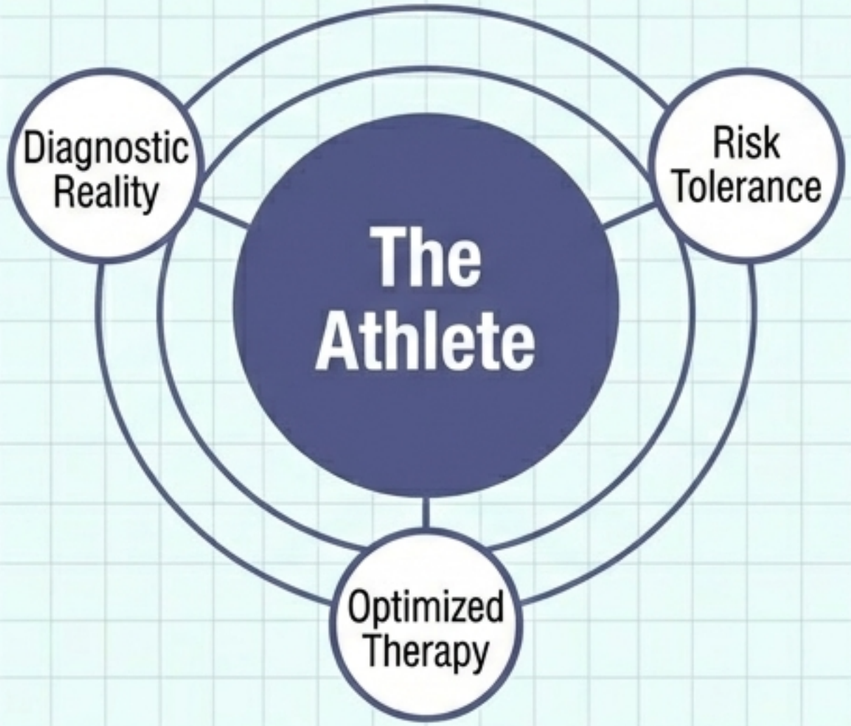
Step 2: Functional Monitoring

Repeat functional and anatomical imaging (CCTA/CMR) to verify plaque stabilization and the total absence of ischemia at a true physiological maximum exertion.

Step 1: Biological Reversal (Absolute Priority)

Implementation of a strict Whole Food Plant-Based (WFPB) diet combined with optimized ApoB-lowering pharmacotherapy. The primary clinical goal is halting lipid penetration, stabilizing morphology, and actively reversing vulnerable plaque burden.

Return to Sport: Moving Beyond Blanket Disqualification



The New Paradigm

The 2025 AHA/ACC scientific statement codifies a move away from unilateral medical gatekeeping.

Shared Decision-Making

Competitive athletes systematically display higher risk tolerance. The sports cardiologist's role is to provide transparent, evidence-based event estimates, not paternalistic bans.

Evidence of Safety

Master athletes following revascularization, and carefully selected patients with HCM or ICDs, demonstrate safety in returning to high-intensity training under close surveillance, provided ischemia is absent and LV function is preserved.

Synthesis: The Modern Playbook

1. Recognize the Mask

- Disregard the absence of classic chest pain.
- Treat unexplained performance drops and dyspnea as primary ischemic equivalents.

2. Image Anatomically

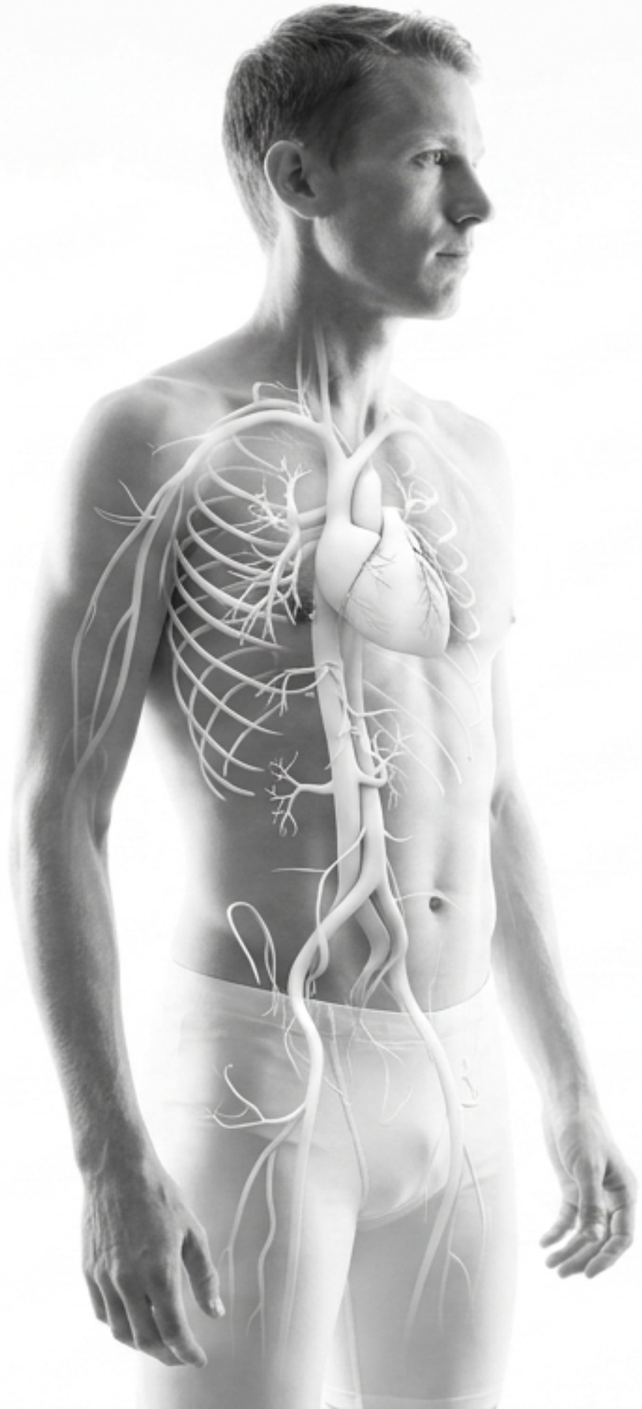
- Bypass submaximal ECGs. Rely on CCTA for plaque morphology and AAOCA, and Stress CMR for hidden cardiomyopathies.

3. Intervene Biologically First

- Prioritize Whole Food Plant-Based diets and aggressive ApoB-lowering medications to reverse plaque. Reserve mechanical stenting only for refractory ischemia.

4. Manage Collaboratively

- Utilize shared decision-making to optimize therapy, monitor via advanced imaging, and safely return the athlete to sport.



Extending the Asymptomatic Margin

The athlete’s heart is an extraordinary expression of human adaptation, but it is not invulnerable. By understanding how supreme fitness masks pathology, nothology, modern sports cardiology can shift from a mindset of disqualification to one of precision management.

Our goal is identifying the absolute limits of human adaptation with diagnostic vigilance proportionate to the dedication of the athlete.