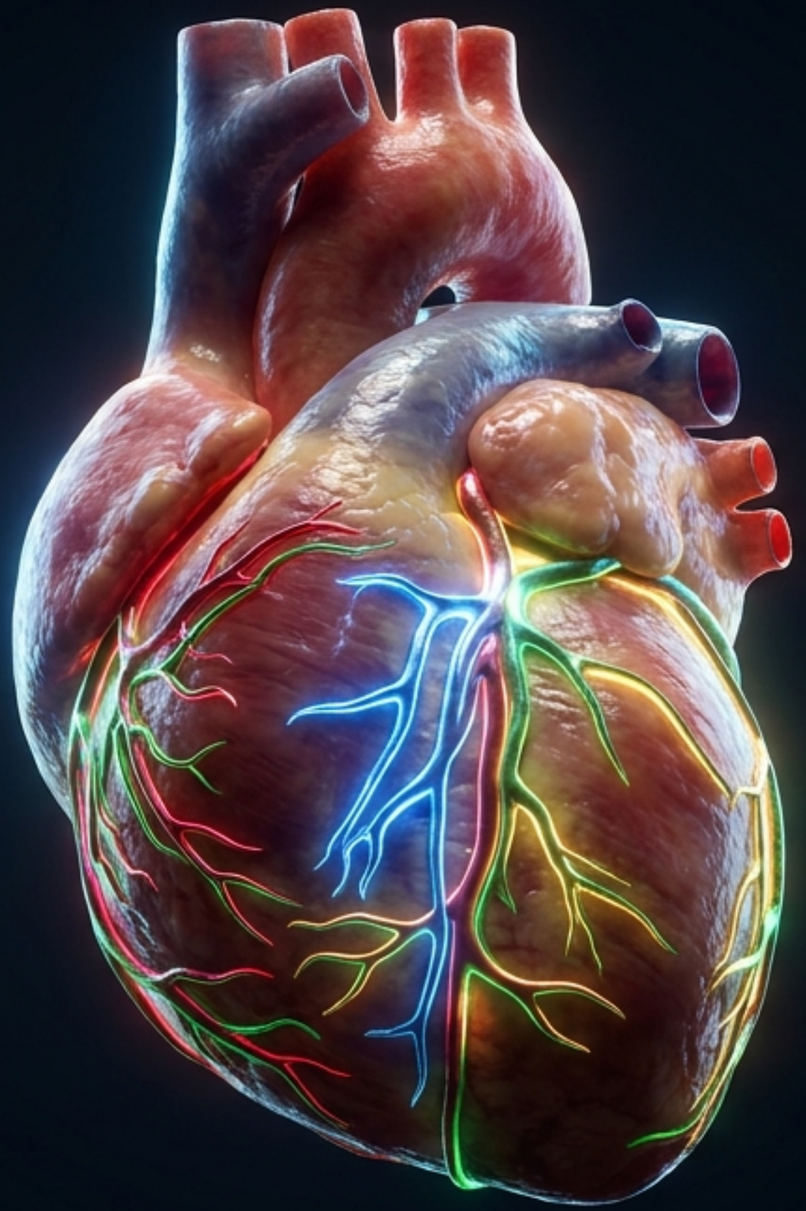
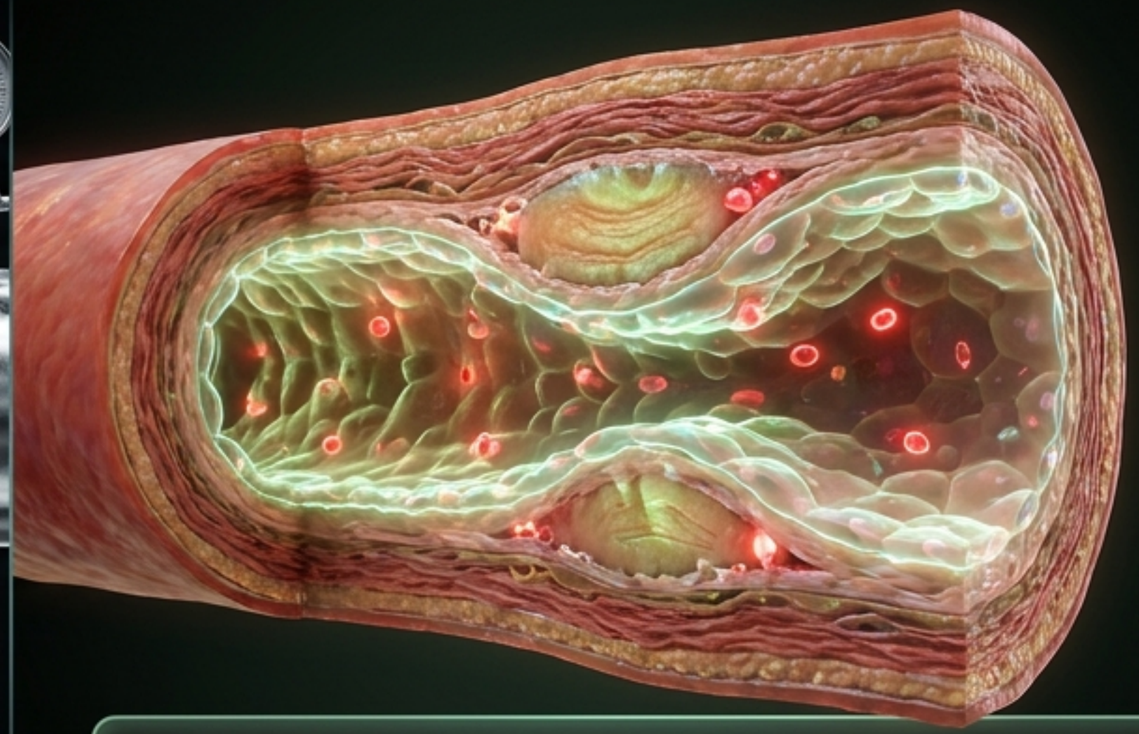
The Living Architecture of the Heart

Comparative Outcomes in Revascularizaliation and Intensive Lifestyle Medicine for Stable CAD



A Strategic Medical Explainer and Synthesis of Modern Trial Data

From Mechanical Reflex to Biological Equipoise

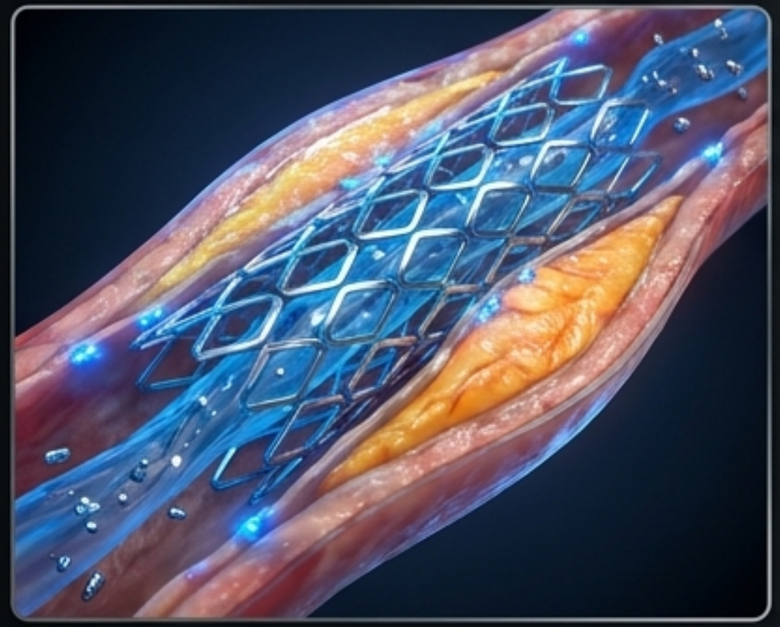
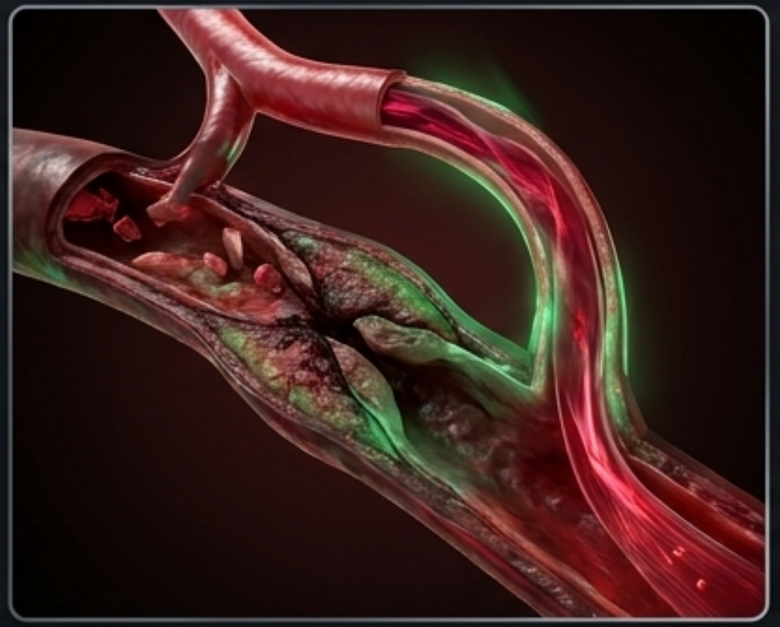
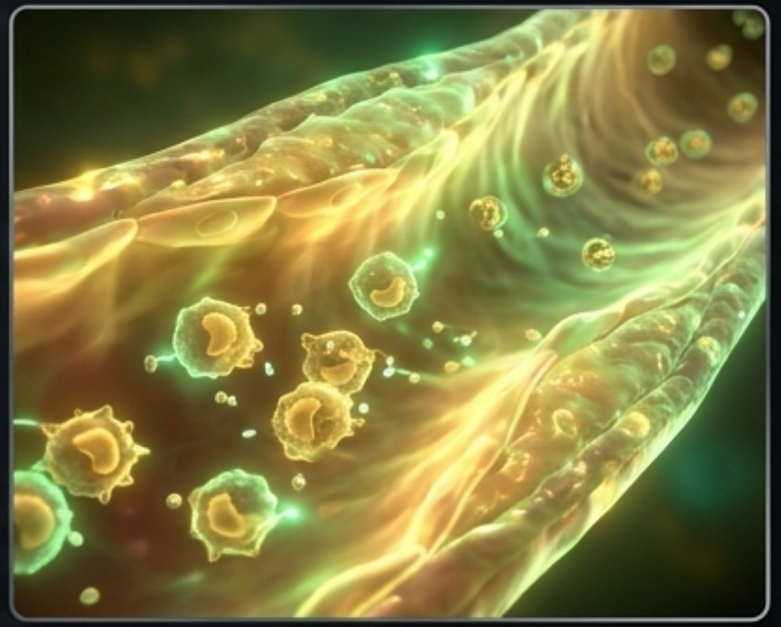


Historically: A flow-limiting epicardial stenosis demanded mechanical revascularization to prevent infarction

Modern Data: Broad clinical equipoise exists between upfront revascularization and conservative management with guideline-directed medical therapy for stable disease

Routine revascularization in stable chronic coronary syndromes does not reduce all-cause mortality relative to optimal medical therapy.

Contrasting the Interventions

Percutaneous Coronary Intervention (PCI)	Coronary Artery Bypass Grafting (CABG)	Intensive Lifestyle Therapy
		
Focal Dilation Addresses specific obstructive lesions without altering systemic substrate.	**Segmental Rerouting** Creates a new proximal artery, bypassing diffuse distal disease.	**Systemic Biological Modification** Favorably alters inflammatory, endothelial, and metabolic pathways.

The Foundation of Clinical Equipoise

Data Node 1
COURAGE Trial
(15-Year Data)

N = 2,287.
Adjusted HR for death: 1.03 (95% CI 0.83–1.21).

15 years of extended follow-up confirms persistent equipoise. Survival curves remain essentially superimposed.

Data Node 2
FAME 2 Trial
(10-Year Data)

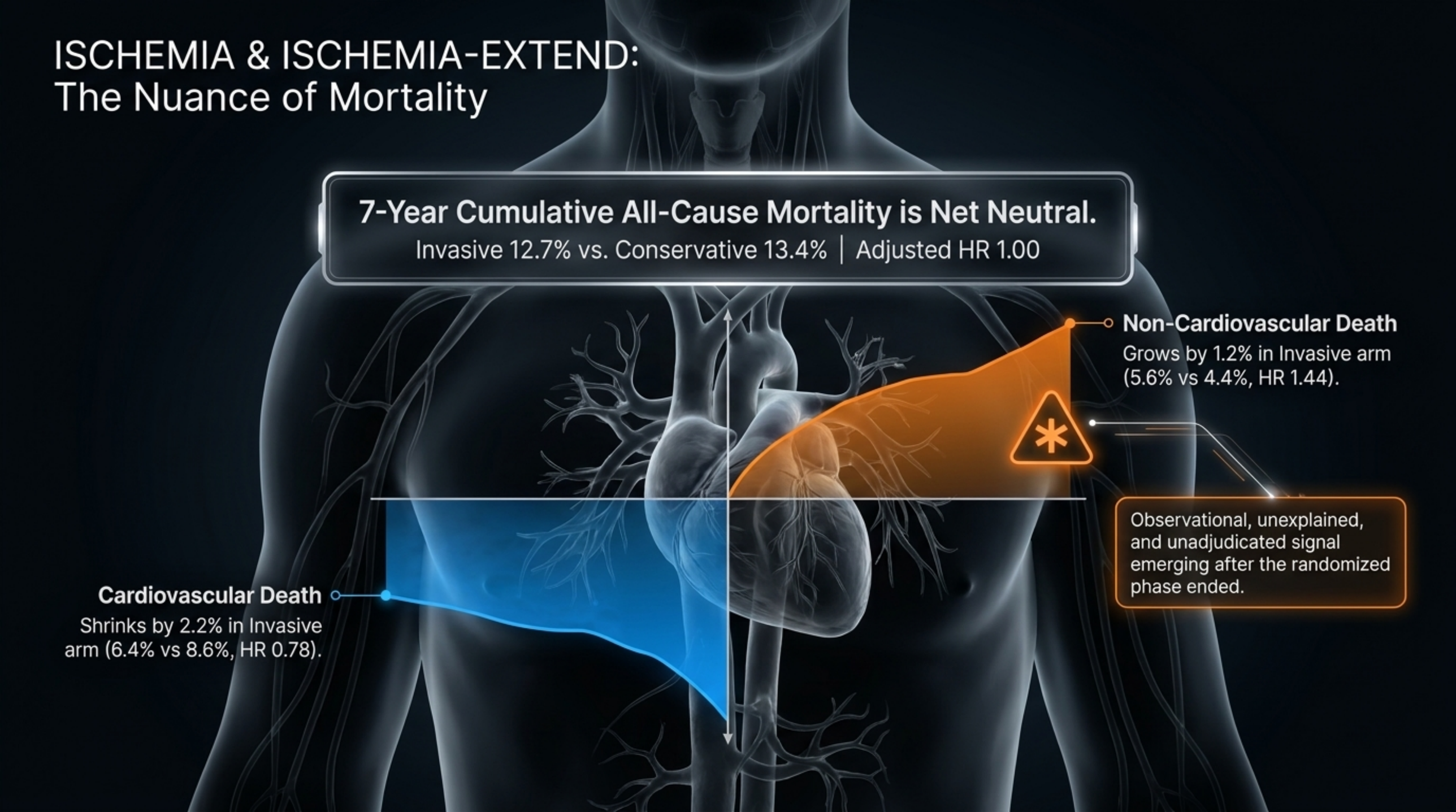
N = 1,220 (FFR ≤ 0.80).
10-year win ratio 1.25 favoring PCI strategy.

Benefit driven overwhelmingly by reductions in urgent revascularization (Component win ratio 4.57), NOT by statistically significant reductions in mortality or spontaneous MI.

ISCHEMIA & ISCHEMIA-EXTEND: The Nuance of Mortality

7-Year Cumulative All-Cause Mortality is Net Neutral.

Invasive 12.7% vs. Conservative 13.4% | Adjusted HR 1.00



Non-Cardiovascular Death

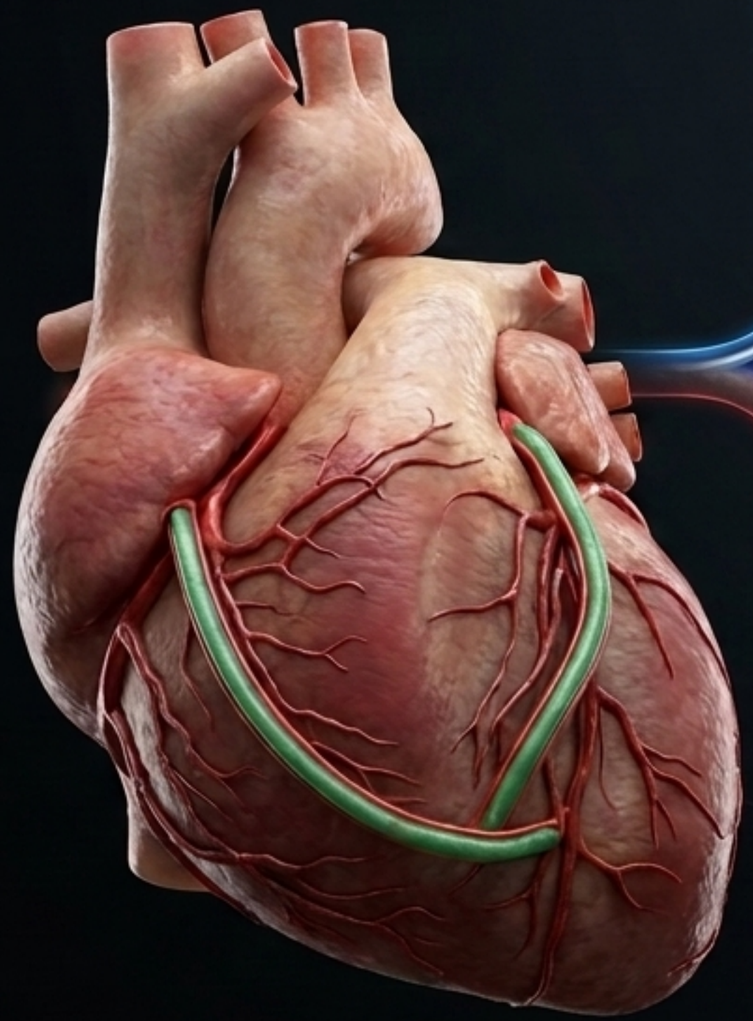
Grows by 1.2% in Invasive arm (5.6% vs 4.4%, HR 1.44).

Observational, unexplained, and unadjudicated signal emerging after the randomized phase ended.

Cardiovascular Death

Shrinks by 2.2% in Invasive arm (6.4% vs 8.6%, HR 0.78).

Head-to-Head: When the Modality Matters



Low Complexity / Focal Lesions

MASS II & FAME 3

Data: PCI and CABG remain clinically comparable for mortality here.

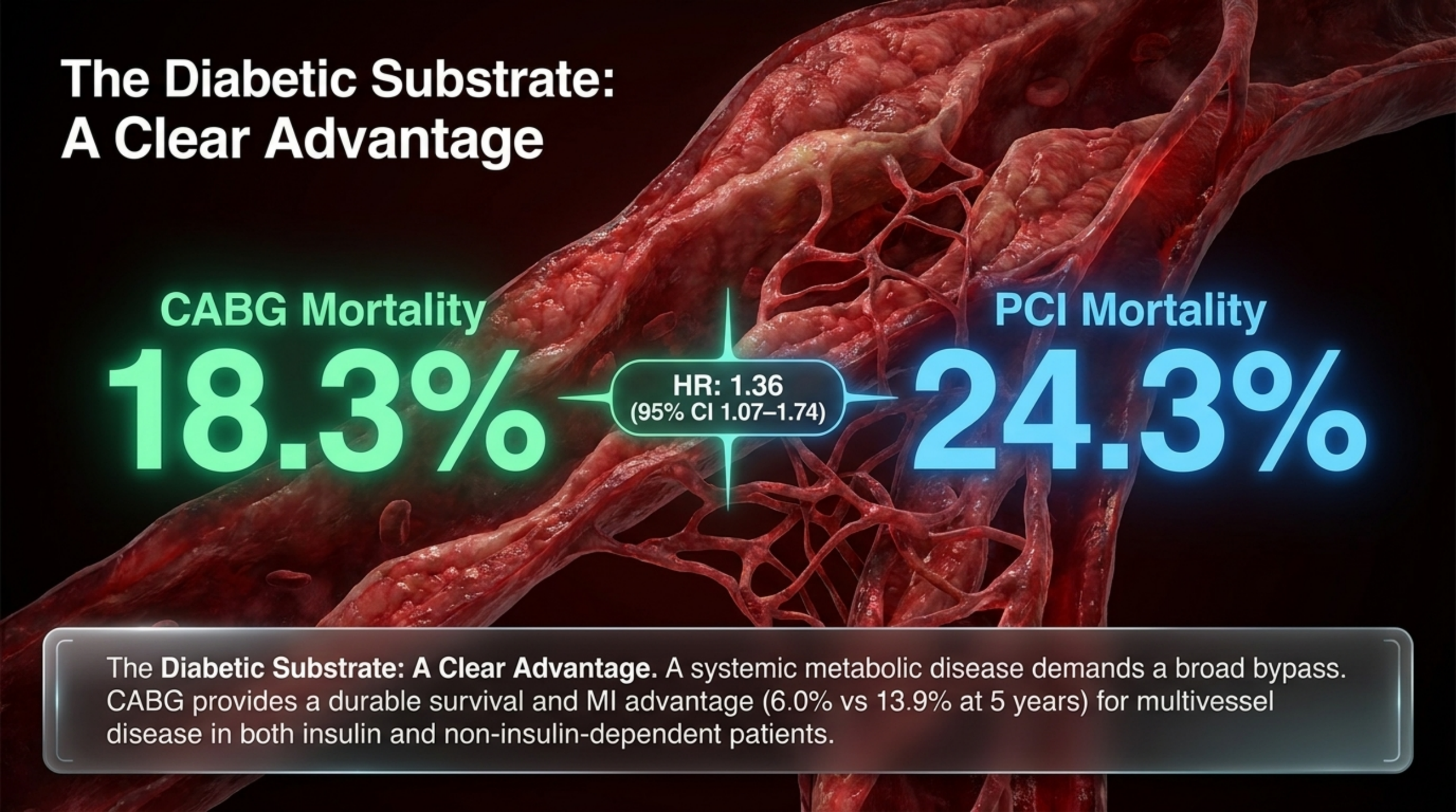
Multivessel / High Complexity (3VD)

Surgical Bypass Graft

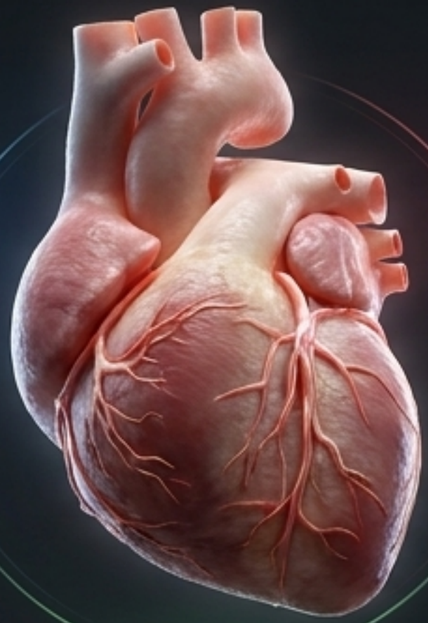
SYNTAXES 10-year mortality: A severe divergence.

CABG: 21% vs. **PCI: 28%**

Completeness of revascularization determines survival. When PCI cannot achieve complete revascularization in diffuse disease, CABG's segmental bypass confers a definitive long-term survival advantage.


The Diabetic Substrate:
A Clear Advantage
CABG Mortality
18.3%
HR: 1.36
(95% CI 1.07–1.74)
PCI Mortality
24.3%
The **Diabetic Substrate: A Clear Advantage.** A systemic metabolic disease demands a broad bypass. CABG provides a durable survival and MI advantage (6.0% vs 13.9% at 5 years) for multivessel disease in both insulin and non-insulin-dependent patients.

Navigating Left Main Coronary Artery Disease (LMCAD)



PCI

CABG

Intensive Lifestyle

1 PRECOMBAT (10-yr)

Neutral mortality
(14.5% PCI vs 13.8% CABG).

Twofold higher Target Vessel Revascularization for PCI
(16.1% vs 8.0%).

2 NOBLE (5-yr)

PCI associated with worse MACCE (28.4% vs 19.0%).

Driven by non-procedural MI and repeat revascularization.

3 EXCEL (5-yr) & The Controversy

PCI all-cause mortality higher (13.0% vs 9.9%), but cardiovascular mortality neutral.

The Crux of Dispute: Trial interpretation hinges on the definition of periprocedural vs. spontaneous myocardial infarction used to adjudicate events.

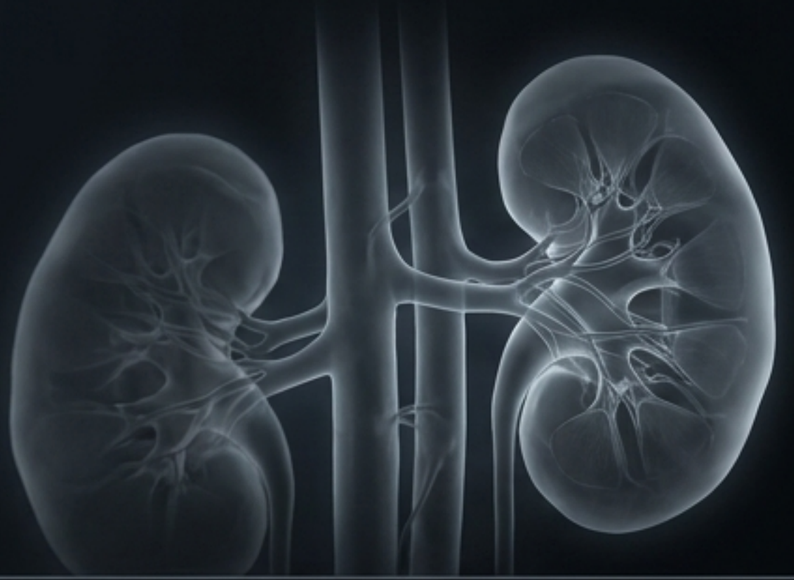
Strategic Triage: Reduced LVEF & Advanced CKD

Reduced LVEF (Ischemic Cardiomyopathy)

STICH / STICHES CABG (Surgical Green) Intensive Lifestyle

Data: For LVEF ≤ 35%, CABG demonstrates a long-term mortality benefit at 10 years (despite a neutral 5-year intention-to-treat result).

Advanced Chronic Kidney Disease



ISCHEMIA-CKD PCI (Metallic Silver) Intensive Lifestyle

Data: eGFR < 30 or dialysis. Over 2.2 years, initial invasive strategy yielded no significant difference in death/MI compared to conservative management.

Given high procedural and contrast risk, intensive medical therapy is a reasonable frontline strategy, reserving invasive management for refractory symptoms.

Reversing the Substrate: Biological Modification

Baseline: 40.7% Stenosis

72% reduction in angina frequency

Profound total cholesterol drops (~246 mg/dL down to ~137 mg/dL)

5-Year Outcome: 37.3% Stenosis (vs. progression to 51.9% in controls)

Achieved via 10-15% fat whole-food plant-based diet, stress management, and exercise, not isolated supplementation

Beyond the Lumen: Plausible Mechanisms of Healing

Plant-predominant nutrition & intensive lifestyle

1

Nitric Oxide Signaling

Nitric Oxide Signaling

Provides arginine/nitrate substrate, promoting vasodilation and limiting smooth-muscle proliferation

2

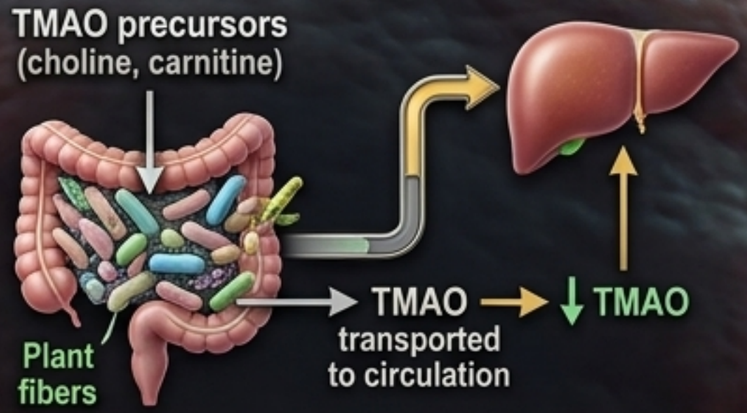
Systemic Inflammation (EVADE CAD)

Systemic Inflammation (EVADE CAD)

Randomized data shows 32% lower hs-CRP with vegan diet vs. AHA diet over 8 weeks.

3

TMAO / Microbiome Pathway

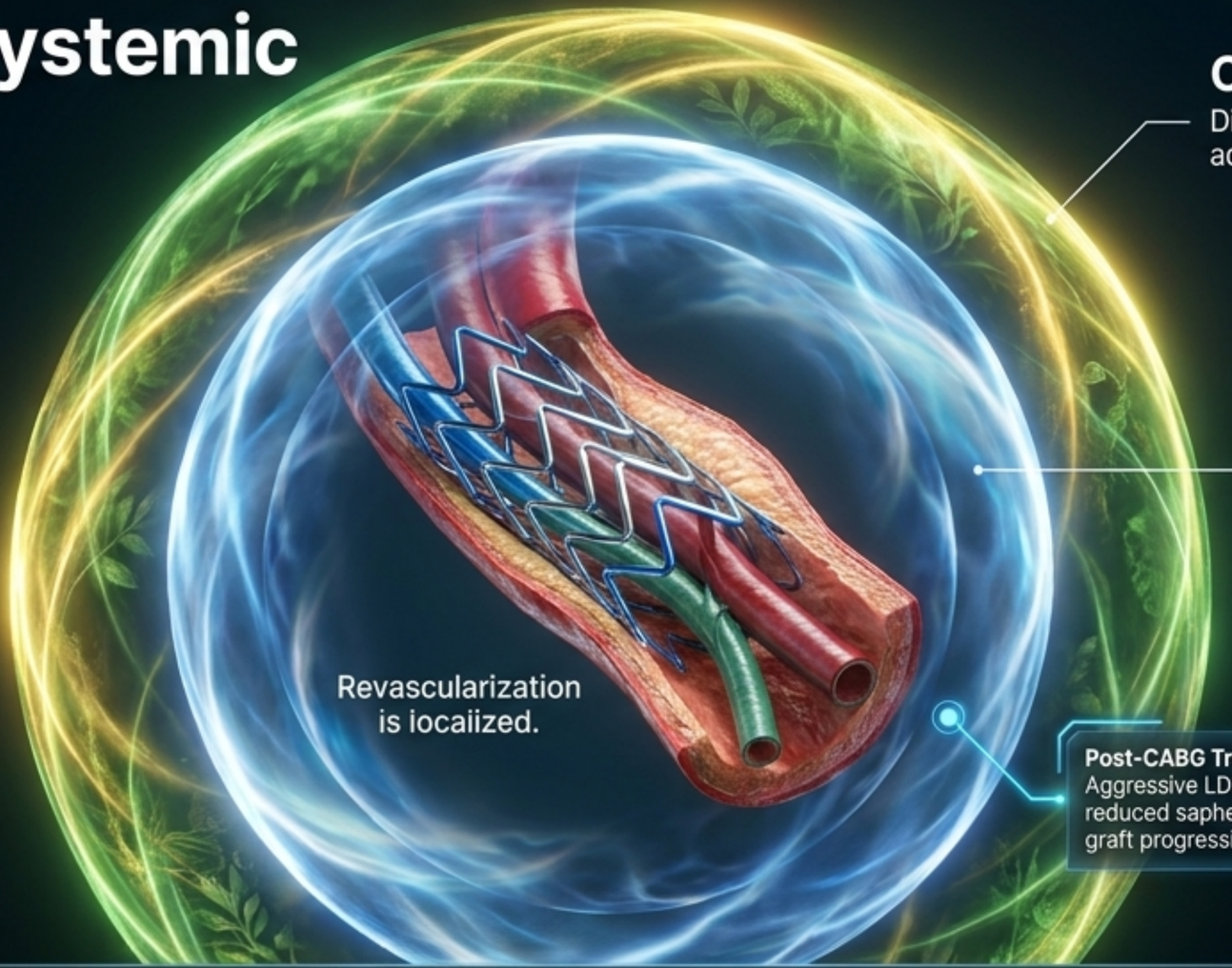


Alters microbiome to reduce TMAO, a metabolite linked experimentally to platelet hyperreactivity.

Limits of Evidence:

Claims of diet-specific reductions in in-stent restenosis or stent thrombosis are explicitly not supported by peer-reviewed controlled data.

Revascularization is Local; Disease is Systemic



Outer Shield

Diet, exercise, smoking cessation addressing systemic substrate.

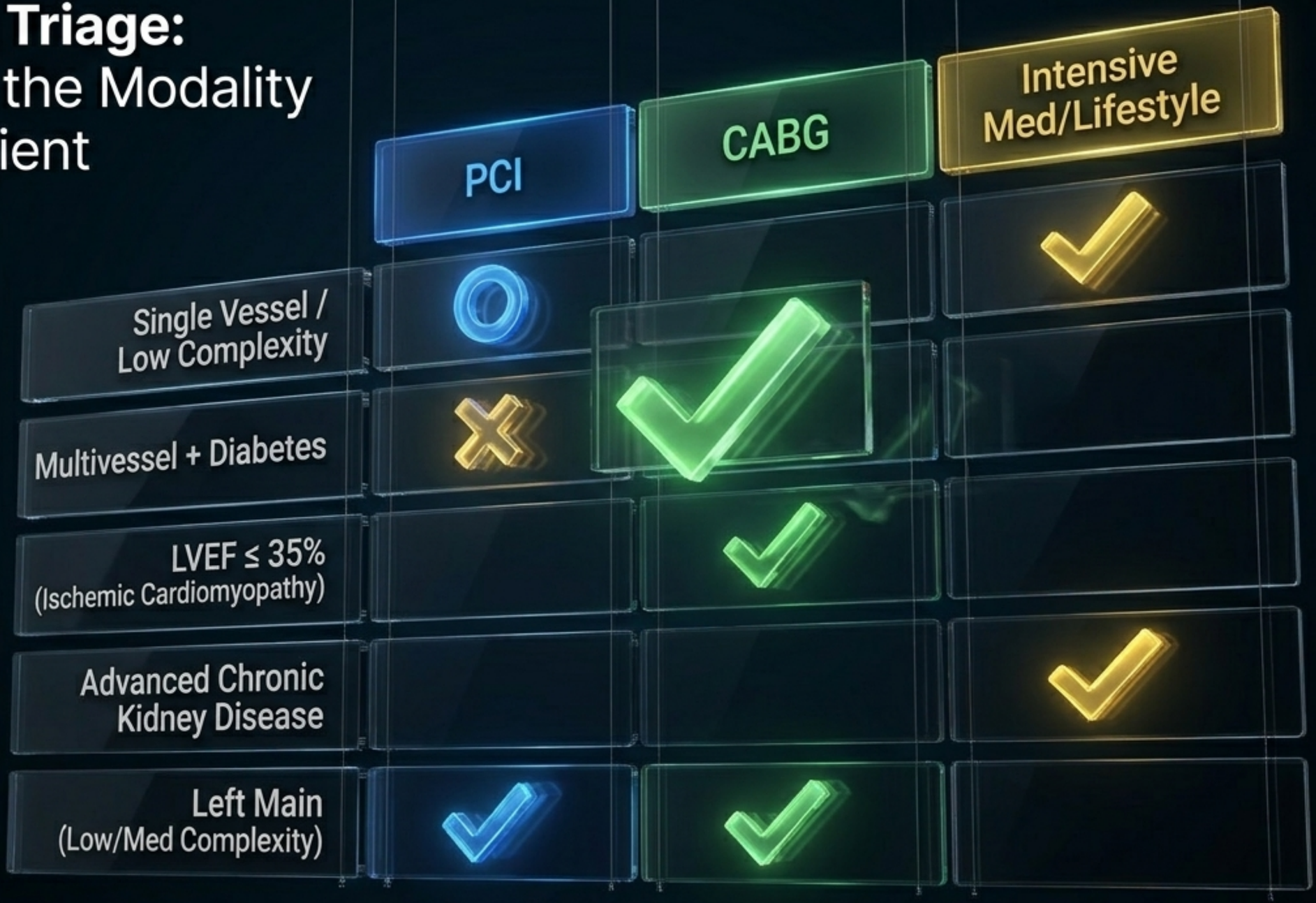
Middle Shield

Aggressive LDL Targets (< 55 mg/dL). Statins, PCSK9 inhibitors, GLP-1 RAs, Bempedoic acid.

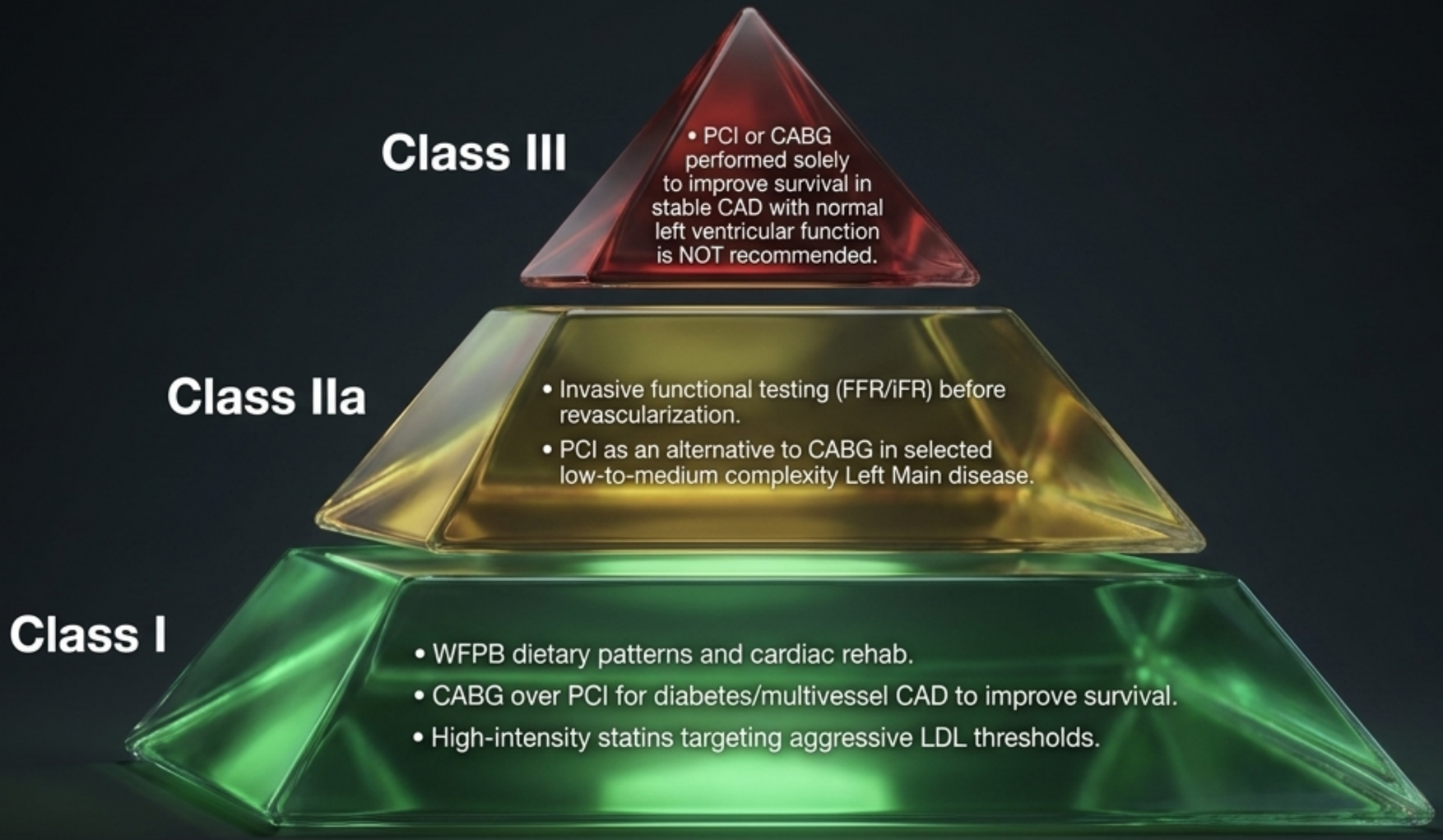
Post-CABG Trial:
Aggressive LDL lowering reduced saphenous vein graft progression by −31%.

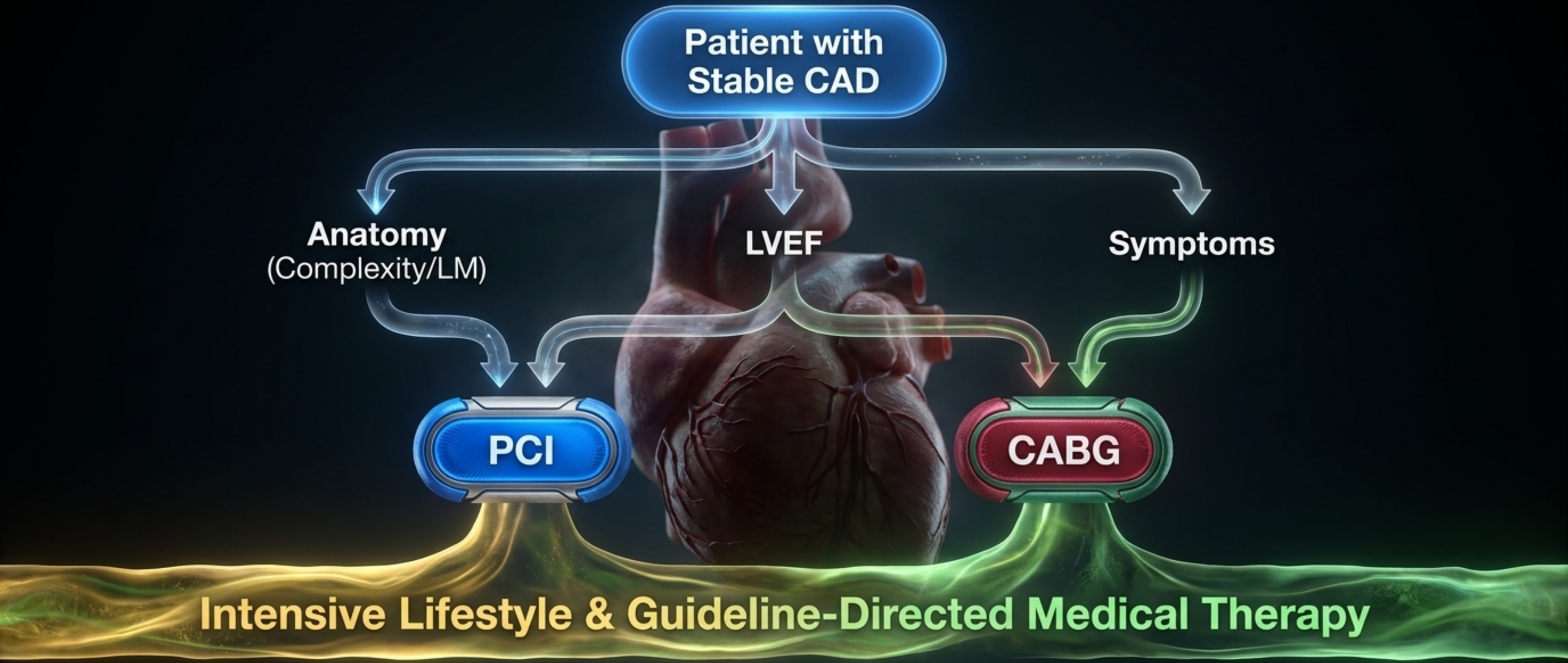
Implanting a stent or constructing a graft does not halt atherosclerosis. Contemporary secondary prevention mandates integrating pharmacotherapy with behavioral intervention.

Strategic Triage: Matching the Modality to the Patient



Translating Evidence to Guidelines





The Universal Foundation: Regardless of the requirement for mechanical intervention, biological substrate modification remains the mandatory foundation of care.