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Revascularization in Ischemic Cardiomyopathy: From Myocardial Viability to Complete Revascularization and Contemporary Patient Selection

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Abstract

Background: Ischemic cardiomyopathy represents one of the most consequential intersections between coronary artery disease and heart failure, combining extensive atherosclerotic disease, myocardial scar, potentially recoverable dysfunctional myocardium, adverse ventricular remodeling, and a high risk of recurrent cardiovascular events. Coronary revascularization has traditionally been considered a central therapeutic strategy because restoration of myocardial blood flow may relieve ischemia, recover hibernating myocardium, improve left ventricular function, and prevent subsequent myocardial injury. The contemporary evidence base, however, has challenged a simple relationship between myocardial viability, successful revascularization, ventricular recovery, and survival. Long-term results from surgical revascularization trials support coronary artery bypass grafting in appropriately selected patients with severe ischemic left ventricular dysfunction, while randomized evidence with percutaneous coronary intervention has not demonstrated a reduction in mortality or heart-failure hospitalization when added to modern medical therapy. Similar uncertainty applies to myocardial viability testing and the concept of complete revascularization, particularly in patients with extensive scar and advanced ventricular remodeling. This review examines the pathophysiological basis of ischemic left ventricular dysfunction, contemporary methods of viability assessment, evidence supporting coronary artery bypass grafting and percutaneous coronary intervention, anatomical and functional definitions of complete revascularization, and the modifying influence of guideline-directed heart-failure therapy. A modern treatment strategy should move beyond ejection fraction or viability as isolated criteria and integrate coronary anatomy, scar burden, residual ischemia, symptoms, ventricular geometry, comorbidities, frailty, procedural feasibility, and competing clinical risks. Such multidimensional assessment provides a more rational framework for individualized revascularization in ischemic cardiomyopathy.

Keywords: ischemic cardiomyopathy, myocardial viability, coronary revascularization, patient selection

Introduction

Ischemic cardiomyopathy (ICM) is a major cause of heart failure with reduced ejection fraction (HFrEF) and represents the final clinical expression of interactions between obstructive coronary artery disease (CAD), previous myocardial infarction, recurrent ischemia, progressive myocardial fibrosis, and adverse left ventricular (LV) remodeling. Although definitions differ among studies, the clinical construct generally describes significant LV systolic dysfunction in the presence of coronary disease of sufficient severity to account for the ventricular abnormality. The population is heterogeneous: some patients have extensive transmural scar after previous infarction, others have chronically underperfused but viable myocardium, and many have mixtures of scar, hibernation, inducible ischemia, microvascular dysfunction, and nonischemic contributors to ventricular dysfunction. Contemporary European and North American guidelines therefore approach ICM as both a coronary and heart-failure syndrome rather than as an isolated stenotic coronary disease. [1]

Revascularization has an intuitive biological rationale in this setting. Restoration of coronary blood flow can relieve angina, reduce inducible ischemia, protect myocardium from recurrent infarction, and potentially recover contractile function in hibernating myocardial territories. For decades, these mechanisms encouraged an assumption that the identification of viable myocardium should identify patients most likely to obtain prognostic benefit from

revascularization. Observational investigations strongly supported this concept, but subsequent randomized trials produced a more complex picture. CABG demonstrated important long-term clinical benefit in the Surgical Treatment for Ischemic Heart Failure (STICH) program, whereas percutaneous coronary intervention (PCI) did not reduce death or hospitalization for heart failure in the REVIVED-BCIS2 trial. [6]

This divergence has transformed the clinical question. The decision is no longer simply whether viable myocardium is present or whether a stenotic vessel can technically be opened. Contemporary assessment must determine whether revascularization can alter the clinically important substrate responsible for an individual patient's risk. Scar burden, coronary complexity, jeopardized myocardium, symptoms, recurrent ischemia, the likelihood of achieving durable revascularization, surgical risk, renal function, frailty, arrhythmic substrate, associated ischemic mitral regurgitation, and the degree of implementation of guideline-directed medical therapy (GDMT) may all change the balance between benefit and procedural harm. Current revascularization guidelines consequently give CABG a central role in appropriately selected patients with severe LV dysfunction and multivessel CAD while recognizing that evidence for an equivalent prognostic role of PCI is considerably weaker. [5]

An additional challenge is that the background therapy against which revascularization is evaluated has changed substantially. Modern HFrEF therapy combines renin-angiotensin system modulation, evidence-based beta-blockade, mineralocorticoid receptor antagonism, and sodium-glucose cotransporter-2 inhibition, together with appropriate diuretics, lipid-lowering therapy, antithrombotic treatment, implantable cardioverter-defibrillators (ICDs), and cardiac resynchronization therapy (CRT). These interventions can improve ventricular remodeling and clinical outcomes independently of coronary intervention. The incremental value of revascularization therefore needs to be interpreted on top of increasingly effective medical and device therapy. [2]

The aim of this review is to integrate contemporary evidence concerning the pathophysiology of ICM, myocardial viability, CABG, PCI, and the concept of complete revascularization, with particular attention to the transition from a viability-centered model toward multidimensional patient selection. A central research gap remains the identification of the combination of anatomical, myocardial, physiological, and clinical characteristics that predicts a meaningful incremental benefit from revascularization in patients already receiving contemporary GDMT.

Pathophysiological Basis of Ischemic Left Ventricular Dysfunction

ICM develops through multiple pathways rather than a single episode of myocardial ischemia. Large myocardial infarctions produce irreversible cardiomyocyte loss and replacement fibrosis, while recurrent episodes of ischemia may generate cumulative injury, progressive chamber dilation, neurohormonal activation, and deterioration of systolic function. The surviving myocardium is exposed to increased wall stress and compensatory hypertrophy, while alterations in extracellular matrix turnover promote ventricular enlargement and a progressively more spherical geometry. Functional mitral regurgitation can emerge from annular enlargement, papillary muscle displacement, and leaflet tethering, creating additional volume overload and accelerating the remodeling process. These changes explain why reopening an epicardial coronary artery may not reverse advanced ventricular failure when the dominant pathological substrate has already become scar and structural remodeling rather than reversible ischemia. [3]

Two classic forms of reversible dysfunction are myocardial stunning and hibernation. Stunning describes persistent contractile impairment after a transient ischemic episode despite restoration of perfusion. Hibernation is a more chronic adaptive state in which contractility is downregulated in response to repetitive ischemia or reduced perfusion reserve, allowing cardiomyocytes to preserve cellular integrity at the expense of mechanical performance. Hibernating myocardium may recover after restoration of adequate blood supply, although the time course can extend over weeks or months. Long-standing hibernation may also evolve into structural dedifferentiation, cellular loss, interstitial fibrosis, and ultimately irreversible dysfunction, making viability a continuum rather than a binary biological condition. [18]

Epicardial stenosis is also only one component of the ischemic substrate. Coronary microvascular dysfunction, endothelial injury, impaired vasodilatory reserve, distal embolization after infarction or PCI, and microvascular obstruction can restrict tissue-level reperfusion even when the epicardial vessel has been successfully

opened. This phenomenon helps explain the imperfect relationship between angiographic procedural success and subsequent improvement in LV function. A vessel may be patent while its subtended myocardium remains limited by microvascular damage or established scar. Conversely, medical therapy may improve ventricular function through unloading, reverse remodeling, metabolic effects, and prevention of recurrent injury without altering a pre-existing epicardial stenosis.[1]

Scar burden is especially important because it links mechanical dysfunction with arrhythmic and prognostic risk. Replacement fibrosis reduces the amount of myocardium capable of contractile recovery and creates spatial heterogeneity in electrical conduction that facilitates ventricular re-entry. The amount, distribution, and transmural extent of scar may therefore be more informative than global LVEF when considering the recoverability of individual territories. Cardiac magnetic resonance (CMR) studies established a graded relationship between late gadolinium enhancement (LGE) and contractile recovery: segments with little or no hyperenhancement have a substantially greater probability of functional recovery after successful revascularization than segments with extensive transmural scar.[16]

This biological heterogeneity provides an important explanation for the varying outcomes of revascularization studies. A patient with recent deterioration, substantial viable jeopardized myocardium, recurrent ischemia, limited scar, and technically treatable proximal disease is biologically different from a patient with the same LVEF but extensive transmural scar, severe LV dilation, diffuse distal CAD, chronic kidney disease, and little ischemic burden. Trials based predominantly on LVEF and anatomical CAD inevitably combine these phenotypes, and the average treatment effect may not accurately represent the potential benefit or harm for an individual patient.

Guideline-Directed Medical Therapy as the Foundation of Care

Contemporary treatment of ICM begins with comprehensive GDMT rather than revascularization in isolation. Current HFrEF therapy incorporates four disease-modifying pharmacological pillars: an angiotensin receptor-neprilysin inhibitor, or an ACE inhibitor/angiotensin receptor blocker when an ARNI cannot be used; an evidence-based beta-blocker; a mineralocorticoid receptor antagonist; and an SGLT2 inhibitor. These agents reduce death and heart-failure hospitalization through complementary mechanisms involving neurohormonal blockade, reduction in sympathetic drive, antifibrotic effects, natriuresis, renal protection, and favorable ventricular remodeling. [2]

The strength of this background therapy is directly relevant to interpretation of revascularization trials. Sacubitril/valsartan reduced cardiovascular death and heart-failure hospitalization compared with enalapril in PARADIGM-HF, while dapagliflozin and empagliflozin subsequently extended disease-modifying therapy through benefits that occur irrespective of diabetes status. Mineralocorticoid receptor antagonism and evidence-based beta-blockade provide additional reductions in mortality and sudden cardiac death. A patient whose LVEF improves after initiation and titration of GDMT therefore cannot automatically be considered to have recovered because of revascularization, even when PCI or CABG was performed during the same period. [31]

Secondary prevention of coronary events is equally central. High-intensity lipid-lowering therapy, appropriate antiplatelet treatment, smoking cessation, blood-pressure control, diabetes management, exercise, weight optimization, and treatment of associated kidney disease decrease recurrent atherothrombotic risk. ICD implantation and CRT are considered in eligible patients after sufficient optimization of medical therapy, because sudden arrhythmic death and electrical dyssynchrony remain major determinants of outcome in ICM. The therapeutic question is consequently whether revascularization adds meaningful benefit to this multidimensional platform, not whether it performs better than limited historical medical treatment. [4]

Myocardial Viability: Biological Concept and Clinical Assessment

Myocardial viability refers to living myocardium with the potential to retain or recover function. Different imaging techniques identify different components of this biology. Dobutamine stress echocardiography assesses contractile reserve; SPECT evaluates cell-membrane integrity and perfusion using radiotracer uptake; FDG-PET assesses preserved glucose metabolism in relation to perfusion; and CMR characterizes myocardial scar through LGE while low-dose dobutamine CMR can evaluate contractile reserve. These techniques should therefore not be regarded as interchangeable measurements of a single variable. [18]

Dobutamine stress echocardiography is widely accessible and provides valuable information regarding contractile reserve. Improvement in wall thickening with low-dose dobutamine suggests viable myocardium, while a biphasic response during progressive stress supports viable myocardium with inducible ischemia. Its principal strengths are availability, absence of ionizing radiation, and simultaneous assessment of ventricular function and valves. Limitations include dependence on image quality, observer experience, and difficulty quantifying scar. Severe ventricular dilation and marked wall thinning reduce the probability of meaningful contractile recovery but should not automatically be interpreted as proof of complete nonviability. [17]

SPECT remains widely used because of accessibility and its established ability to evaluate perfusion, scar, and inducible ischemia. Thallium redistribution exploits preserved cellular uptake and redistribution within viable myocardium, while technetium-based tracers provide high-quality perfusion imaging. PET offers greater sensitivity for metabolic viability. The classical FDG-PET pattern of reduced perfusion accompanied by preserved glucose metabolism—the perfusion-metabolism mismatch—identifies hibernating myocardium. In PARR-2, however, a strategy of FDG-PET-assisted management did not significantly reduce the primary composite outcome in the intention-to-treat population, although exploratory analyses suggested better outcomes when PET recommendations were followed. [14]

CMR has become particularly influential because it provides high-resolution assessment of global and regional function, ventricular geometry, infarct location, scar burden, and scar transmurality in a single investigation. The landmark work by Kim and colleagues demonstrated a progressive inverse association between transmural hyperenhancement and functional recovery after revascularization. Importantly, later studies also showed that even markedly thinned myocardial regions may contain limited scar and can sometimes regain thickness and contractility, arguing against equating thinning alone with irreversible injury. [16]

The historical enthusiasm for viability-guided revascularization was driven by observational evidence. In a meta-analysis of 24 studies involving more than 3,000 patients with severe LV dysfunction, Allman and colleagues found a striking association between viability and improved survival after revascularization compared with medical treatment. Annual mortality among patients with viable myocardium was approximately 16% with medical therapy versus 3.2% after revascularization. The magnitude of this association was clinically compelling but the included studies were nonrandomized and highly vulnerable to referral, treatment-selection, and survivorship biases.[13]

Randomized evidence weakened the premise that viability should serve as a gatekeeper for prognostic revascularization. In the STICH viability substudy, the presence of viable myocardium was associated with better unadjusted prognosis but did not identify a statistically significant interaction between viability and survival benefit from CABG. Long-term analysis similarly failed to demonstrate that viability status selected a subgroup deriving differential mortality benefit from surgical revascularization. Limitations remain, including nonrandom allocation to viability testing, use of SPECT and dobutamine echocardiography rather than systematic PET or LGE-CMR, and a relatively small number of patients classified as nonviable. [9]

REVIVED-BCIS2 provided an important test of the concept in PCI. Enrollment required severe LV dysfunction, extensive CAD, and demonstrable viability in at least four dysfunctional myocardial segments amenable to PCI. Yet PCI did not improve the primary clinical outcome. In the prespecified viability analysis, the extent of viable myocardium did not identify patients who benefited from PCI. Greater nonviable myocardial burden, by contrast, was associated with a lower probability of ventricular recovery and worse prognosis. These findings suggest that scar burden may function more consistently as a marker of biological limitation than viability functions as a predictor of treatment interaction. [11]

The clinical value of viability imaging has therefore changed rather than disappeared. Viability remains useful when the clinician needs to distinguish ischemic scar from potentially recoverable myocardium, understand regional dysfunction, quantify infarct burden, or resolve uncertainty in a high-risk revascularization decision. What the randomized data do not support is the use of a simple viable/nonviable classification as the sole determinant of whether revascularization should be undertaken for prognostic benefit.

Surgical Revascularization: Lessons From STICH and STICHES

The strongest randomized evidence for prognostic revascularization in severe ICM comes from CABG. STICH enrolled 1,212 patients with CAD amenable to surgery and an LVEF of 35% or less and randomized them to CABG plus medical therapy or medical therapy alone. At the original median follow-up of approximately five years, CABG did not significantly reduce the primary endpoint of all-cause mortality, although cardiovascular mortality and the combined endpoint of death or cardiovascular hospitalization favored surgery. The trial also demonstrated a clinically important early hazard associated with CABG, reflecting the vulnerability of patients with severe LV dysfunction during major surgery. [6]

The extended STICHES follow-up changed interpretation of the trial. At a median follow-up of 9.8 years, all-cause mortality was lower among patients assigned to CABG plus medical therapy than among those receiving medical therapy alone. Cardiovascular mortality and cardiovascular hospitalization were also reduced. The delayed separation of event curves is clinically important: CABG imposes a substantial immediate physiological and procedural burden, while its benefits may accumulate over years through protection against future ischemic events and provision of durable blood supply to multiple coronary territories. [7]

This temporal pattern helps explain why short follow-up can underestimate the value of surgical revascularization. A patient needs sufficient life expectancy to survive the early hazard and accrue the downstream benefit. Severe frailty, advanced noncardiac disease, irreversible end-stage ventricular remodeling, or markedly limited life expectancy may therefore reduce the value of CABG even when the coronary anatomy itself is technically suitable. Conversely, younger patients with extensive multivessel CAD, acceptable surgical risk, diabetes, proximal disease, and substantial myocardium at risk may have a long time horizon over which durable surgical protection becomes clinically meaningful. [5]

STICH also challenged the belief that improvement in LVEF is the principal mechanism by which CABG improves survival. The long-term benefit was not confined to patients with substantial viability, and changes in LVEF did not fully explain outcome differences. CABG may prevent future infarction, reduce spontaneous ischemic events, protect against progressive ischemic myocardial loss, and provide collateralized flow beyond proximal stenoses. These mechanisms are particularly relevant in diffuse multivessel disease, where bypass grafts may protect a larger coronary territory than focal treatment of individual lesions. [9]

Current guidelines reflect this evidence. In patients with severe LV systolic dysfunction and multivessel CAD who are appropriate surgical candidates, CABG is recommended to improve survival. This recommendation is supported by randomized evidence in a population that directly represents ischemic LV dysfunction rather than extrapolation from patients with normal ventricular function. The decision still requires a Heart Team approach because operative risk, target-vessel quality, conduit availability, frailty, kidney disease, cerebrovascular disease, diabetes, pulmonary disease, and patient preferences can materially alter the net benefit. [5]

PCI in Severe Ischemic Cardiomyopathy: The REVIVED-BCIS2 Era

PCI is attractive in ICM because it avoids sternotomy and cardiopulmonary bypass, permits staged treatment, and may be feasible in patients considered unsuitable for CABG. Advances in drug-eluting stents, chronic total occlusion techniques, intracoronary imaging, calcium modification, physiological lesion assessment, and mechanical circulatory support have expanded the technical scope of complex PCI. Yet technical feasibility should not be confused with demonstrated prognostic benefit.

REVIVED-BCIS2 randomized 700 patients with an LVEF of 35% or less, extensive CAD, and viability in at least four dysfunctional segments to PCI plus optimal medical therapy or optimal medical therapy alone. At a median follow-up of 41 months, the primary endpoint of death from any cause or hospitalization for heart failure occurred in 37.2% of the PCI group and 38.0% of the medical-therapy group, producing no evidence of clinical benefit from routine PCI. LVEF also did not improve preferentially with PCI at the major follow-up time points. [10]

The neutral result is particularly informative because the trial enrolled precisely the type of patient historically thought likely to benefit from PCI: severe ischemic LV dysfunction, extensive CAD, and demonstrable viable myocardium. It therefore argues strongly against offering PCI solely because viable dysfunctional myocardium is present. It also illustrates the effectiveness of contemporary nonprocedural therapy; ventricular function improved in

both groups, demonstrating that reverse remodeling can occur through GDMT and time without mechanical revascularization. [10]

PCI did produce an early health-status advantage, particularly in patients with a greater symptom burden, although differences narrowed during longer follow-up. This distinction between symptomatic and prognostic benefit is important. A procedure may be clinically valuable because it relieves refractory angina, reduces ischemia-related limitation, or improves quality of life even when it does not reduce mortality. Similar observations have been made in broader chronic coronary disease populations, where invasive treatment has its clearest effect on symptoms among patients with frequent baseline angina. [20]

REVIVED should not be interpreted as evidence that PCI has no role in any patient with severe LV dysfunction. Patients with acute coronary syndromes, ongoing ischemia, unstable presentations, unacceptable angina despite medical therapy, or anatomies where revascularization is required for another established indication are different from the stable REVIVED population. The trial instead shows that routine PCI cannot be assumed to improve survival or prevent heart-failure hospitalization in stable severe ICM simply because anatomically treatable CAD and viable myocardium coexist. [10]

Complete Revascularization: Anatomical, Functional, and Myocardial Perspectives

Complete revascularization has traditionally been defined anatomically: treatment of all major coronary stenoses judged to be significant. Observational studies across PCI populations have consistently associated incomplete revascularization with higher residual ischemic risk. The residual SYNTAX score was developed to quantify the burden and complexity of untreated coronary disease after PCI and has been associated with subsequent mortality and ischemic events. These findings established an attractive conceptual target: if residual coronary disease predicts adverse outcomes, reducing that residual disease should improve prognosis. [47]

Yet association does not establish that completing PCI alters the outcome in severe ICM. Patients in whom complete revascularization is achievable usually have less diffuse disease, fewer chronic total occlusions, better distal target vessels, fewer comorbidities, and lower procedural risk than those left incompletely revascularized. The apparent benefit may therefore partly represent patient and anatomical selection. This issue is amplified in ICM, where opening a stenotic vessel supplying predominantly scar tissue may increase procedural exposure without restoring clinically meaningful myocardium.

The concept can be refined into anatomical, functional, and viability-guided completeness. Anatomical completeness considers whether angiographically significant lesions have been treated. Functional completeness considers whether ischemia-producing lesions remain after intervention, with fractional flow reserve or nonhyperemic pressure ratios potentially helping distinguish lesions that warrant treatment. Viability-guided completeness asks whether vessels supplying viable dysfunctional myocardium have been successfully revascularized. These definitions overlap but can produce different conclusions in the same patient. [45]

The 2024 prespecified REVIVED-BCIS2 analysis directly examined this issue. Investigators quantified anatomical completeness using changes in the British Cardiovascular Intervention Society jeopardy score and viability-guided completeness according to the proportion of viable myocardial segments supplied by diseased vessels that were revascularized. The median degree of anatomical revascularization in the PCI arm was approximately 67%, while viability-guided revascularization was approximately 85%. Neither greater anatomical nor greater viability-guided completeness was associated with improved event-free survival compared with medical therapy, and analysis using residual SYNTAX score produced a similar result. [12]

These findings distinguish established ICM from settings such as acute myocardial infarction, where randomized studies have shown benefits from staged treatment of significant nonculprit disease. In COMPLETE, complete multivessel PCI after successful culprit-lesion treatment reduced cardiovascular death or recurrent myocardial infarction compared with culprit-only treatment. That evidence cannot be directly extrapolated to chronic severe ICM, because myocardium in an acute infarction population is biologically different from a chronically remodeled ventricle containing large areas of scar and longstanding dysfunction. [25]

Complete revascularization should therefore be considered a treatment objective only when it is connected to a clinically meaningful myocardial target. The most rational question is not how many stenoses can be treated, but how much prognostically or symptomatically important myocardium can be protected at an acceptable procedural cost. A technically incomplete strategy that successfully treats the dominant ischemia-producing vessels supplying viable myocardium may be more appropriate than extensive intervention on diffuse distal lesions supplying heavily scarred territories.

PCI Versus CABG and the Importance of Coronary Anatomy

Direct randomized comparison between PCI and CABG specifically in patients with severe ICM remains a major evidence gap. Many landmark PCI-versus-CABG trials either excluded patients with severe LV dysfunction or contained relatively small numbers of such patients. In broader multivessel CAD populations, CABG has generally produced fewer spontaneous myocardial infarctions and repeat revascularizations, particularly in patients with greater anatomical complexity and diabetes, although surgery may carry greater early risk of stroke and recovery burden. [22]

The anatomical distinction matters in ICM. CABG can provide blood flow beyond long segments of proximal and diffuse coronary disease and may protect against future proximal plaque progression. PCI treats discrete lesions and is therefore more dependent on lesion-by-lesion durability and on the ability to identify all clinically important disease. A patient with complex three-vessel disease, diabetes, proximal LAD involvement, multiple chronic total occlusions, and acceptable surgical risk may therefore represent a very different revascularization problem from a patient with focal proximal disease and prohibitive surgical risk. [24]

The SYNTAX experience established the importance of coronary complexity in choosing a modality. Higher baseline anatomical complexity and greater residual disease after PCI are associated with worse outcomes, while CABG becomes increasingly attractive as disease becomes diffuse and multivessel. Modern PCI has narrowed several historical limitations through second-generation drug-eluting stents, better antiplatelet therapy, physiology, IVUS and OCT guidance, atherectomy, intravascular lithotripsy, and improved CTO techniques, but no randomized contemporary trial has yet demonstrated that these advances reproduce the long-term survival evidence of CABG in severe ICM. [23]

High-risk PCI may require temporary hemodynamic support when large myocardial territories are jeopardized and severe LV dysfunction creates little tolerance for prolonged ischemia. The PROTECT II trial compared Impella 2.5 with intra-aortic balloon support during selected high-risk PCI and demonstrated superior hemodynamic support with the microaxial pump but did not establish a definitive routine mortality advantage. BCIS-1 similarly did not support routine prophylactic intra-aortic balloon pumping for all high-risk PCI procedures. Mechanical support should therefore facilitate a carefully selected procedure rather than serve as justification for performing otherwise low-value revascularization. [25]

Contemporary Patient Selection: Moving Beyond a Viability-Only Model

The first step in contemporary patient selection is to establish the clinical indication. Refractory angina, recurrent ischemia, acute coronary instability, high-risk coronary anatomy, and recurrent ischemic ventricular arrhythmia may create reasons to consider revascularization that differ from the goal of improving chronic heart-failure prognosis. A patient referred solely because of a low LVEF should not automatically proceed to revascularization. The clinician should first ask whether CAD is causally and currently contributing to symptoms, instability, ongoing ischemia, or future myocardial jeopardy. [1]

The second step is characterization of the myocardial substrate. Echocardiography defines LVEF, chamber volumes, regional wall motion, right ventricular function, pulmonary pressure, and ischemic mitral regurgitation. CMR can add high-resolution information on infarct distribution, scar burden, scar transmurality, ventricular thrombus, and alternative myocardial diseases. PET or SPECT may be useful when perfusion-metabolic relationships, ischemic burden, or viability remain uncertain. The key purpose of imaging is to describe the substrate and resolve a decision, not to generate a binary test result that automatically mandates intervention. [18]

The third component is detailed coronary anatomical assessment. The number of diseased vessels alone is inadequate. Left main disease, proximal LAD involvement, diffuse distal disease, chronic total occlusions, heavy calcification, bifurcation disease, vessel size, quality of distal surgical targets, previous stents or grafts, and the relationship between coronary territories and viable myocardium all influence treatment. Physiological assessment can be valuable for intermediate lesions when the functional significance of a stenosis is uncertain. FFR-guided PCI has improved lesion selection in stable coronary disease, although direct evidence in advanced ICM is more limited. [25]

The fourth dimension is procedural feasibility and durability. CABG should be considered when extensive multivessel disease can be bypassed with acceptable surgical risk and durable graft targets, particularly when the patient has a sufficiently long life expectancy to realize delayed benefit. PCI becomes more attractive when surgical risk is prohibitive, coronary anatomy is focal and suitable, symptoms require treatment, or a meaningful ischemic territory can be revascularized with an acceptable contrast, bleeding, and hemodynamic burden. The objective should be clinically appropriate revascularization rather than maximal procedural activity.

The fifth dimension is competing risk. Advanced age alone should not determine treatment, but frailty, severe chronic kidney disease, cerebrovascular disease, pulmonary disease, cancer, advanced peripheral arterial disease, cognitive decline, hepatic dysfunction, and limited functional reserve can shift the balance between procedural benefit and harm. Patients with advanced HF and irreversible ventricular remodeling may remain at high risk because of pump failure and arrhythmia even after successful coronary intervention. In such patients, advanced heart-failure therapies, valve treatment, device therapy, or palliative symptom-focused strategies may have greater clinical importance than additional coronary intervention. [3]

The sixth component is the adequacy and duration of GDMT. Decisions made before initiation of foundational HFrEF therapies risk misclassifying potentially reversible dysfunction as fixed severe cardiomyopathy. Except in situations requiring urgent revascularization, optimization of heart-failure and secondary-prevention therapy provides important information about blood pressure tolerance, renal function, symptom trajectory, and ventricular recovery. Reassessment after therapy can change the indication for ICD implantation, clarify functional limitation, and identify patients whose dominant residual problem is angina or ischemia rather than congestion and neurohormonal heart failure. [4]

A practical Heart Team discussion should integrate all these domains rather than debate PCI and CABG purely from the angiogram. The interventional cardiologist contributes an assessment of lesion complexity, physiological significance, PCI completeness, contrast burden, and procedural risk. The cardiac surgeon assesses graftability, conduit strategy, and operative risk. Heart-failure and imaging specialists contribute interpretation of ventricular phenotype, scar, viability, remodeling, device indications, and competing mechanisms of symptoms. Shared decision-making can then incorporate the patient's priorities regarding longevity, symptom relief, procedural burden, recovery time, repeat intervention, stroke risk, and quality of life. [5]

Reconciling STICHES and REVIVED-BCIS2

STICHES and REVIVED are sometimes presented as conflicting trials, but they tested different interventions with different biological properties and follow-up structures. STICH evaluated CABG, which can provide extensive and durable revascularization beyond multiple proximal stenoses, while REVIVED evaluated PCI directed at amenable lesions in an older population receiving substantially more contemporary medical and device therapy. CABG also carries greater early hazard, making long-term follow-up essential to detect a delayed treatment benefit. PCI carries a smaller initial physiological burden but may not offer the same protection against diffuse future coronary events. [7]

The trials also suggest that recovery of LVEF is not an adequate surrogate for survival. Ventricular function can improve with GDMT in the absence of revascularization, and revascularization may theoretically reduce future infarction without producing a large change in resting LVEF. Conversely, an improvement in LVEF does not necessarily demonstrate a treatment-specific prognostic effect. Clinical trials should therefore prioritize death, heart-failure hospitalization, myocardial infarction, symptoms, quality of life, and treatment burden rather than assuming

that a change in ejection fraction captures the entire value of revascularization.

A coherent interpretation is that CABG has proven long-term prognostic benefit in selected surgically suitable patients with severe ischemic LV dysfunction, while routine PCI has not demonstrated an equivalent prognostic effect in stable patients resembling those enrolled in REVIVED. PCI remains relevant for established coronary indications and symptom relief, particularly when surgery is unsuitable, but the rationale should be explicit. Myocardial viability and complete revascularization can inform that decision, yet neither should be treated as an independent guarantee of survival benefit. [12]

Remaining Evidence Gaps and Future Directions

Several questions remain unresolved. A contemporary randomized comparison of CABG and PCI in severe ICM on a background of current four-pillar HFrEF therapy would be clinically valuable, but recruitment and treatment crossover present major challenges. Trials also need more refined phenotyping than a single LVEF threshold. Scar burden by CMR, inducible ischemia, coronary physiology, myocardial blood flow, ventricular volumes, right ventricular function, mitral regurgitation, renal function, frailty, and biomarker profiles could help define biologically distinct subgroups.

The future of viability assessment is likely quantitative rather than binary. The extent and distribution of scar, viable but dysfunctional myocardium, ischemic burden, and coronary territories at risk can potentially be integrated into patient-specific models. Hybrid PET/CT and PET/CMR, quantitative myocardial blood-flow imaging, CMR tissue characterization, strain imaging, and artificial-intelligence-assisted image integration may improve mapping between coronary anatomy and myocardial phenotype. Their clinical value, however, will depend on demonstrating that imaging-guided treatment changes patient outcomes rather than merely improving prediction.

The concept of complete revascularization also requires refinement in ICM. Future studies should distinguish anatomical completeness from ischemia-guided, viability-guided, and prognostically meaningful completeness. The 2024 REVIVED analysis indicates that simply increasing the proportion of angiographically or viability-defined territories treated by PCI does not guarantee improved outcomes in established severe LV dysfunction. This observation should redirect research toward identifying which myocardial territories truly represent modifiable risk. [12]

Conclusion

Revascularization in ischemic cardiomyopathy should be guided by the interaction between coronary anatomy, myocardial substrate, clinical presentation, procedural feasibility, and contemporary heart-failure therapy rather than by LVEF or myocardial viability alone. CABG has demonstrated long-term survival benefit in appropriately selected patients with severe ischemic LV dysfunction and surgically amenable coronary disease, whereas routine PCI added to modern medical therapy has not shown a comparable reduction in death or heart-failure hospitalization in stable severe ischemic cardiomyopathy. Viability imaging remains valuable for defining scar and potentially recoverable myocardium, but it should support rather than dictate treatment. Similarly, complete revascularization is meaningful only when the treated coronary territories represent clinically relevant myocardium and procedural risk remains acceptable. Multidisciplinary patient selection that integrates symptoms, scar burden, ischemia, coronary complexity, comorbidities, frailty, life expectancy, and patient preferences offers the most rational framework for contemporary care.

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