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Myocardial Viability, Coronary Physiology, and Prognosis After PCI in Ischemic Cardiomyopathy

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ABSTRACT

***Background*:** Ischemic cardiomyopathy (ICM) represents a major cause of heart failure and cardiovascular mortality worldwide, characterized by chronic coronary artery disease, left ventricular (LV) dysfunction, and a heterogeneous substrate of scarred and viable myocardium. Percutaneous coronary intervention (PCI) is frequently considered in this population to restore coronary perfusion, reduce ischemic burden, and promote ventricular recovery. However, the prognostic benefit of PCI in ICM remains uncertain, as illustrated by results from recent randomized trials such as REVIVED-BCIS2, which demonstrated no significant improvement in mortality or heart-failure hospitalization compared with optimal medical therapy despite evidence of myocardial viability. These findings highlight the evolving need to integrate myocardial viability assessment with coronary physiologic evaluation to better identify patients who may benefit from revascularization.

Myocardial viability reflects the presence of viable but dysfunctional (hibernating) myocardium with potential for recovery following revascularization. Traditional imaging modalities—including PET perfusion and metabolic imaging, cardiac magnetic resonance with late gadolinium enhancement (LGE), and dobutamine stress echocardiography—provide complementary insights into scar burden, cellular metabolism, and contractile reserve. Yet, viability alone has shown inconsistent ability to predict clinical benefit from PCI, prompting renewed focus on the contribution of ischemia, microvascular dysfunction, and coronary flow physiology. Fractional flow reserve (FFR), instantaneous wave-free ratio (iFR), quantitative flow ratio (QFR), and myocardial flow reserve (MFR) derived from PET or CMR improve delineation of flow-limiting lesions and microvascular health, offering prognostic information directly relevant to revascularization decisions.

Emerging data suggest that optimal prognostication requires integrating viability and physiologic assessment, as myocardial segments with preserved viability but absent inducible ischemia—or profound microvascular dysfunction—may not recover function after PCI. Conversely, territories demonstrating both viable myocardium and physiologically significant ischemia appear most likely to exhibit reverse remodeling and improved outcomes. Advances in imaging, including PET flow quantification, CMR tissue characterization, and machine learning–enhanced risk models, promise to refine patient selection further.

This review synthesizes current evidence linking myocardial viability and coronary physiology to prognosis after PCI in ICM, clarifying mechanisms underlying variable outcomes and providing a framework for personalized revascularization strategies aimed at improving survival, ventricular recovery, and quality of life.

Keywords: Prognosis After PCI in Ischemic Cardiomyopathy

INTRODUCTION

Ischemic cardiomyopathy (ICM) is a leading cause of heart failure with reduced ejection fraction (HFrEF), resulting from chronic myocardial ischemia, infarction, and progressive left ventricular (LV) remodeling. Despite major advances in guideline-directed medical therapy and device-based heart failure management, revascularization remains a central therapeutic consideration for patients with multivessel coronary artery disease (CAD) and impaired LV function. Percutaneous coronary intervention (PCI), in particular, has evolved substantially through improved stent technology, physiologic lesion assessment, and operator expertise. Yet, the prognostic value of PCI in ICM remains controversial. Observational studies have suggested symptomatic and functional benefits, whereas randomized trials—most notably REVIVED-BCIS2—did not demonstrate improved survival or reduced heart failure hospitalization when PCI was added to optimal medical therapy. These divergent findings underscore the need to better understand patient-specific determinants of benefit. [1–3]

Myocardial viability and coronary physiology have emerged as two of the most critical domains influencing outcomes after revascularization in ICM. Viability reflects the presence of hibernating but recoverable myocardium, identifiable through imaging modalities such as positron emission tomography (PET), cardiac magnetic resonance with late gadolinium enhancement (CMR-LGE), and dobutamine stress echocardiography. Historically, patients with substantial viability were believed to benefit most from revascularization, as restoring perfusion to hibernating myocardium could promote contractile recovery and reverse remodeling. However, randomized data—including the STICH viability substudy and REVIVED—challenge the notion that viability alone reliably predicts survival benefit from PCI, suggesting a more complex interplay between ischemia, microvascular function, and scar burden. [4–6]

Coronary physiology adds an essential complementary layer by identifying hemodynamically significant lesions and quantifying myocardial flow impairment. Tools such as fractional flow reserve (FFR), instantaneous wave-free ratio (iFR), quantitative flow ratio (QFR), coronary flow reserve (CFR), and myocardial flow reserve (MFR) from PET imaging provide insights that angiography cannot. These physiologic parameters have strong prognostic implications, as persistent ischemia or microvascular dysfunction may limit the likelihood of functional recovery even in the presence of viable tissue. Integrating viability with physiologic assessment offers a more nuanced and accurate framework for predicting which patients stand to benefit from PCI. [7–9]

The aim of this review is to synthesize the evolving evidence linking myocardial viability, coronary physiology, and clinical prognosis following PCI in ischemic cardiomyopathy. By examining mechanistic interactions, imaging and physiologic modalities, and insights from major trials, we aim to establish a contemporary framework for personalized revascularization strategies that optimize clinical outcomes in this complex patient population.

Conceptual Framework: Viability, Ischemia, and Ventricular Recovery

Myocardial viability represents the presence of dysfunctional myocardium with preserved cellular integrity and metabolic activity that may recover function following revascularization. This concept is rooted in the phenomenon of myocardial hibernation, wherein chronic hypoperfusion leads to downregulated contractility as an adaptive mechanism to prevent irreversible injury. Viable myocardium, therefore, exhibits reduced contractile performance due to insufficient flow rather than structural loss. Revascularization may restore perfusion, normalize metabolic pathways, and enable the recovery of contractile function, leading to improved left ventricular ejection fraction (LVEF) and reverse remodeling. However, the extent to which viable myocardium contributes to recovery is influenced by scar burden, microvascular dysfunction, and the presence of physiological ischemia. [10–12]

Ischemia plays a central but distinct role in determining post-PCI outcomes. While viability reflects the potential for recovery, ischemia indicates the presence of physiologically significant flow limitation capable of provoking symptoms, stunning, or progressive remodeling. Physiologic lesion assessment—via fractional flow reserve (FFR), instantaneous wave-free ratio (iFR), quantitative flow ratio (QFR), or myocardial flow reserve (MFR)—identifies lesions where restoring epicardial flow will meaningfully improve perfusion. Importantly, viable myocardium without ischemia is less likely to improve after PCI, and ischemic myocardium with extensive irreversible scar may also fail to recover. Thus, ischemia and viability must be considered

jointly, as each alone provides an incomplete picture of recovery potential. [13–15]

Ventricular recovery after PCI depends on the convergence of three critical factors: preserved myocardial substrate, physiologically significant ischemia, and sufficient microvascular integrity to permit post-revascularization perfusion enhancement. Advanced imaging and physiologic tools increasingly allow clinicians to quantify these factors with precision. PET myocardial blood flow measurements and CMR-based scar quantification help determine the balance between viable and non-viable myocardium, while physiology identifies the subset of coronary lesions that are functionally relevant. When these elements align—viable myocardium with demonstrable ischemia and preserved microvascular reserve—patients are most likely to exhibit meaningful improvements in LVEF, reduced heart failure progression, and improved survival. Conversely, mismatches among these domains explain why many patients with viability do not improve after PCI in modern trials such as REVIVED. [16–18]

Myocardial Viability Testing Modalities

Myocardial viability testing plays a central role in evaluating patients with ischemic cardiomyopathy (ICM), as it identifies dysfunctional yet recoverable myocardium with the potential to regain contractile function following revascularization. **Positron emission tomography (PET)** remains the gold standard for metabolic viability assessment, using FDG uptake to distinguish viable tissue from scar. PET viability imaging has consistently shown strong prognostic value, with early studies demonstrating improved outcomes when revascularization is directed toward territories with preserved glucose metabolism. PET can also quantify absolute myocardial blood flow and myocardial flow reserve (MFR), enabling simultaneous characterization of both viability and microvascular health. However, access, cost, and radiotracer availability limit widespread use in routine practice. [19–21]

Cardiac magnetic resonance (CMR) with late gadolinium enhancement (LGE) offers unparalleled spatial resolution for characterizing scar distribution. The degree of transmural LGE strongly predicts the likelihood of segmental functional recovery after revascularization. Segments with <25% transmural scar are highly likely to improve, while those with >50% scar rarely demonstrate functional recovery. CMR also enables the assessment of myocardial edema, microvascular obstruction, and diffuse fibrosis via T1 and extracellular volume (ECV) mapping, each carrying additional prognostic value. The precision of scar quantification makes CMR one of the most powerful tools for integrating viability into clinical decision-making, although limitations exist for patients with implantable cardiac devices or severe renal dysfunction. [22–24]

Dobutamine stress echocardiography (DSE) evaluates contractile reserve by assessing wall motion improvement at low-dose dobutamine infusion. DSE is widely available, radiation-free, and cost-effective, offering strong predictive value for segmental functional recovery. Its physiological basis lies in identifying hibernating myocardium capable of improving function when stimulated. However, image quality may be impaired in patients with poor acoustic windows, obesity, or arrhythmias. Moderately affected segments can be difficult to interpret, and reliance on visual analysis introduces operator variability. Nonetheless, DSE remains clinically valuable, especially in centers where PET or CMR resources are limited. [25–27]

Despite their strengths, viability tests demonstrate imperfect predictive accuracy when used in isolation. Randomized trials such as STICH and REVIVED revealed that viability did not reliably identify patients who would derive mortality benefit from revascularization. These findings highlight the importance of integrating viability assessment with ischemia evaluation, coronary physiology, and microvascular status. Viability may indicate potential for recovery, but without concurrent ischemia and preserved microvascular reserve, the likelihood of benefit after PCI diminishes considerably. Thus, modern clinical practice increasingly favors multimodality assessment over reliance on any single imaging technique. [28–30]

Coronary Physiology Tools in ICM (FFR, iFR, QFR, MFR)

Coronary physiology is essential for understanding the role of revascularization in ischemic cardiomyopathy (ICM), where angiographic stenosis alone often fails to predict functional significance due to diffuse disease, remodeling, and microvascular impairment. **Fractional flow reserve (FFR)**, defined as the ratio of distal coronary pressure to aortic pressure during hyperemia, remains the gold standard for identifying lesions that cause physiologically meaningful ischemia. Large randomized trials such as FAME demonstrated that FFR-guided PCI reduces major adverse cardiac events and improves resource utilization compared with angiography-guided PCI. In the context of ICM, FFR helps distinguish flow-limiting lesions that can potentially restore perfusion to viable myocardium, thereby improving the likelihood of functional recovery. However, its predictive capacity may be attenuated when microvascular dysfunction limits hyperemic flow, a common phenomenon in chronically remodeled

ventricles. [31–33]

Instantaneous wave-free ratio (iFR) offers a vasodilator-free alternative to FFR by measuring the pressure gradient during a specific portion of diastole when microvascular resistance is naturally minimized. Randomized trials—including DEFINE-FLAIR and iFR-SWEDEHEART—showed noninferiority of iFR compared with FFR for guiding PCI, making it a practical tool in patients who may not tolerate adenosine. While iFR provides efficient physiologic assessment, the presence of diffuse atherosclerosis and microvascular dysfunction in ICM may still lead to discordance between iFR and FFR. Nevertheless, iFR-guided PCI can better target lesions responsible for ischemia, potentially improving outcomes by ensuring physiologic appropriateness of revascularization. [34–36]

Noninvasive physiology-derived indices such as **quantitative flow ratio (QFR)** have expanded the ability to assess ischemia without pressure wires or pharmacologic hyperemia. QFR, calculated from routine angiographic images using computational flow modeling, correlates closely with FFR and has demonstrated strong clinical utility in multivessel disease. This technology may be particularly beneficial in ICM patients who are hemodynamically fragile or intolerant of prolonged wire manipulation. In parallel, PET-derived **myocardial flow reserve (MFR)** provides a comprehensive assessment of both epicardial and microvascular function. Reduced MFR is a powerful predictor of mortality independent of angiographic disease severity and may explain why some patients with viable myocardium fail to recover after PCI—because microvascular dysfunction prevents adequate post-procedural perfusion. Integrating MFR with viability imaging therefore offers a physiologically refined approach to prognostication in ICM. [37–39]

Integration of Viability and Physiology in Predicting PCI Benefit

Historically, myocardial viability was considered the primary determinant of functional recovery after revascularization, but emerging evidence demonstrates that viability alone is insufficient to predict which patients with ischemic cardiomyopathy (ICM) will benefit from PCI. Viable myocardium indicates the presence of living, dysfunctional tissue; however, without demonstrable ischemia or physiologic flow limitation, there is limited rationale to expect significant improvement after revascularization. The STICH viability substudy and REVIVED-BCIS2 trial both showed that even when viability is present, revascularization may not improve mortality or LV ejection fraction unless ischemia is also present. These observations reflect the interdependence of perfusion and substrate: viability represents potential, while ischemia represents the stimulus for recovery. As such, prognostic improvement depends on both factors aligning. [40–42]

Coronary physiology offers a crucial complementary evaluation by identifying flow-limiting lesions that, when relieved, are likely to improve perfusion to viable myocardium. Fractional flow reserve (FFR), instantaneous wave-free ratio (iFR), and PET-derived myocardial flow reserve (MFR) help distinguish ischemic territories that may respond to revascularization from those with fixed scar or microvascular dysfunction. When viability and physiologic ischemia overlap, the likelihood of improved contractile function and reverse remodeling is highest. Conversely, viable myocardium without ischemia—often seen in territories with collateral flow or diffuse microvascular dysfunction—may not demonstrate significant functional improvement after PCI. This mismatch provides a mechanistic explanation for why viability imaging alone has not consistently predicted outcomes in randomized trials. [43–45]

Integration of viability and physiology extends beyond detection of ischemia; it allows stratification of microvascular reserve, an increasingly recognized determinant of post-PCI recovery. PET-derived MFR and CMR-based perfusion mapping quantify microvascular health, revealing whether improved epicardial flow will translate to improved tissue-level perfusion. Low MFR (<1.5) is strongly associated with poor outcomes despite viable myocardium, reflecting an inability of the microcirculation to respond to increased flow after PCI. Thus, physiological assessment helps distinguish viable myocardium capable of functional recovery from tissue with exhausted microvascular capacity. This synergy between viability and physiology forms the foundation of modern precision revascularization and highlights why multimodal evaluation may surpass traditional approaches in identifying patients who benefit from PCI. [46–48]

Viability and LV Remodeling After PCI

Left ventricular (LV) remodeling is a major determinant of prognosis in ischemic cardiomyopathy, and myocardial viability strongly modulates the extent to which remodeling improves after PCI. Segments containing hibernating but viable myocardium can recover systolic function when perfusion is restored, leading to reductions in LV end-systolic volume (LVESV) and modest increases in ejection fraction that nonetheless translate into improved survival and fewer heart failure admissions. Multiple

imaging studies using PET and CMR have demonstrated that the burden and distribution of viable myocardium correlate with the magnitude of reverse remodeling after revascularization. However, this relationship is not absolute; patients with advanced global dilation, severe mitral regurgitation, or extensive scar may show limited functional recovery despite the presence of viable segments, highlighting that viability is necessary but not always sufficient for meaningful remodeling. [49–51]

CMR with late gadolinium enhancement (LGE) provides robust insight into how scar burden constrains remodeling potential. Segments with <25% transmural LGE have a high likelihood of functional improvement, those with 25–50% have intermediate probability, and those with >50% rarely recover. On a global level, patients with lower total scar burden and greater proportion of viable myocardium demonstrate larger reductions in LVESV and greater increases in LVEF after PCI or CABG. Conversely, diffuse or transmural scar is associated with persistent dilation and ongoing adverse remodeling despite revascularization, which may partly explain why some trials have failed to show improvement in LVEF or outcomes with PCI in ischemic LV dysfunction. These observations support the use of quantitative scar assessment to stratify expectations for post-PCI remodeling and guide discussions about prognosis. [52–54]

The temporal pattern of LV recovery after PCI is also heterogeneous and influenced by the extent of viability and microvascular integrity. Some patients experience early improvement in regional wall motion within weeks, while others show delayed recovery over several months as chronic hibernating myocardium progressively normalizes its structure and function. Biomarkers such as NT-proBNP often fall in parallel with favorable remodeling, whereas persistently elevated levels suggest ongoing wall stress and limited structural recovery. In addition, advanced strain imaging has revealed that even when global LVEF changes are modest, improvement in regional longitudinal strain in viable territories is associated with better clinical outcomes. Collectively, these data indicate that viability-informed PCI can promote beneficial remodeling in selected patients, but that response is strongly conditioned by baseline scar burden, ventricular geometry, and concomitant heart failure therapies. [55–57]

Physiologic Predictors of Outcomes After PCI in Ischemic Cardiomyopathy

Physiologic assessment of coronary circulation provides powerful prognostic information in ischemic cardiomyopathy (ICM), often exceeding that of angiographic stenosis severity alone. Lesions with reduced fractional flow reserve (FFR ≤0.80) or instantaneous wave-free ratio (iFR ≤0.89) identify territories with flow-limiting epicardial disease that are most likely to benefit from revascularization. In stable coronary disease, FFR-guided PCI has been shown to reduce major adverse cardiac events and unnecessary stenting, and similar principles apply in ICM where diffuse disease and remodeling obscure purely anatomical assessment. Importantly, patients with functionally insignificant lesions, despite angiographic severity, rarely derive prognostic benefit from PCI, emphasizing that physiologic significance—not lumen diameter—should guide revascularization decisions in this high-risk population. [58–60]

Beyond epicardial lesion assessment, microvascular function and global myocardial flow reserve (MFR) have emerged as key physiologic predictors of outcome after PCI. PET-derived MFR integrates both epicardial stenosis and microvascular health; reduced MFR (<1.5–2.0) is strongly associated with higher mortality, heart failure hospitalization, and limited capacity for LV functional recovery even when epicardial lesions are revascularized. In patients with ICM, chronic remodeling, endothelial injury, and fibrosis frequently impair microvascular reserve, meaning that restored epicardial patency may not translate to improved tissue-level perfusion. Studies have shown that patients with preserved or moderately reduced MFR experience more pronounced symptomatic and functional gains after PCI than those with severely impaired MFR, supporting its role as a gatekeeper for predicting revascularization benefit. [61–63]

Residual ischemia after PCI is another important physiologic determinant of prognosis. Quantitative perfusion imaging and post-PCI FFR measurements demonstrate that patients with minimal residual ischemia have significantly better clinical outcomes and lower rates of heart failure progression than those with persistent perfusion defects. This relationship extends to chronic total occlusion (CTO) revascularization, where successful restoration of flow to ischemic, viable territories improves LV function and symptoms, whereas unsuccessful attempts leave ischemia unresolved and offer limited prognostic advantage. Integration of physiologic indices such as FFR/iFR, noninvasive MFR, and post-PCI ischemia quantification therefore provides a comprehensive framework for predicting which ICM patients will experience meaningful clinical benefit from PCI versus those in whom optimized medical therapy alone may be more appropriate. [64–66]

Revascularization Completeness, CTOs, and Territorial Viability

Completeness of revascularization is a key modifier of prognosis after PCI in ischemic cardiomyopathy (ICM), particularly when

large viable myocardial territories are subtended by severe stenoses or chronic total occlusions (CTOs). In patients with multivessel disease and LV dysfunction, incomplete revascularization often leaves substantial residual ischemia, limiting the potential for LV reverse remodeling even when some viable segments are treated. In contrast, achieving near-complete revascularization in territories with documented viability and physiologic significance can reduce ischemic burden, improve symptoms, and favorably influence long-term outcomes. The mechanistic basis is straightforward: more ischemic, viable myocardium is salvaged, wall stress is reduced more globally, and neurohormonal activation decreases. However, achieving completeness is frequently constrained by lesion complexity, comorbidities, and procedural risk in this high-risk population. [67]

CTOs represent a particularly important intersection of anatomy, viability, and revascularization completeness. CTOs often supply large territories of dysfunctional, variably scarred myocardium; when significant viability is present, successful recanalization can restore antegrade flow, improve regional contractility, and facilitate global LV recovery. Observational data suggest that patients with LV dysfunction who undergo successful CTO PCI experience better symptom relief, improved EF, and lower event rates than those in whom the CTO remains occluded, especially when viability is demonstrated within the CTO territory. Nonetheless, CTO PCI is technically demanding, carries higher procedural risk, and may offer limited benefit when scar burden is extensive or microvascular reserve is exhausted. Therefore, decisions regarding CTO revascularization in ICM should be guided by integrated assessment of territorial viability, physiologic ischemia, and overall contribution to revascularization completeness, rather than by anatomy alone. [68]

Clinical Evidence: Lessons From STICH, PARR-2, and REVIVED

The three major bodies of evidence informing the prognostic role of viability and physiology in ischemic cardiomyopathy (ICM)—**STICH**, **PARR-2**, and **REVIVED-BCIS2**—highlight the complexity of predicting which patients benefit from PCI. The STICH viability substudy showed that although patients with viability had better overall survival, viability did *not* identify those who derived more benefit from surgical revascularization versus medical therapy. This surprising disconnect suggested that myocardial recovery requires more than the simple presence of viable tissue; global remodeling dynamics, scar burden, and ischemia also play essential roles. The STICHES 10-year extension further demonstrated significant survival benefit with CABG, but again without clear interaction by viability status, emphasizing that viability alone may be too narrow a criterion for selecting revascularization strategy in ICM. [69]

The **PARR-2** trial provided a physiology-integrated perspective by assessing whether PET-guided management improved outcomes compared with standard care. Although the overall trial narrowly missed statistical significance, patients who adhered to PET-guided recommendations—particularly those in whom PET demonstrated preserved viability and impaired flow—experienced significantly better outcomes. These findings suggest that integrating viability with perfusion or metabolic data enhances the ability to select patients who will benefit from revascularization. Importantly, PARR-2 also demonstrated that microvascular and metabolic abnormalities may override the presence of viability alone, reinforcing the concept that viability must be interpreted in a broader physiologic context. [70]

The modern era was reshaped by **REVIVED-BCIS2**, which demonstrated that PCI did not reduce mortality or heart failure hospitalization in patients with severe LV dysfunction, extensive CAD, and documented viability. REVIVED challenged long-held assumptions by showing that viability alone does not guarantee improvement and that restoring epicardial patency may have limited benefit if microvascular reserve is severely impaired or if ischemia burden is low. When viewed collectively, STICH, PARR-2, and REVIVED reveal a consistent theme: **patient selection for revascularization requires integrated assessment of viability, ischemia, coronary physiology, and microvascular health**, rather than reliance on any single factor. These trials underscore the need for multimodal evaluation and personalized decision-making in determining which patients with ICM will truly benefit from PCI.[70]

Biomarkers and Emerging Techniques

Biomarkers provide an increasingly valuable complement to imaging and physiologic assessments in predicting outcomes after PCI in ischemic cardiomyopathy (ICM). Natriuretic peptides such as NT-proBNP reflect wall stress and correlate with both symptomatic status and prognosis. When PCI successfully reduces ischemia and ameliorates ventricular loading conditions, reductions in NT-proBNP often parallel improvements in remodeling and clinical outcomes. Conversely, persistently elevated levels following revascularization suggest ongoing wall stress, incomplete recovery, or limited myocardial reserve despite technically successful PCI. High-sensitivity cardiac troponins similarly help stratify risk by identifying ongoing myocardial

injury—whether due to active ischemia, microvascular dysfunction, or chronic structural damage—and may help differentiate patients with recoverable myocardium from those unlikely to improve after revascularization.[70]

Emerging tools in advanced imaging and computational analysis offer new opportunities for refining prognosis. Strain imaging, particularly global longitudinal strain, detects subclinical myocardial dysfunction that may not be reflected in ejection fraction alone. In ICM, regional and global strain patterns can help identify segments with contractile potential and assess whether PCI is likely to yield meaningful functional gain. Cardiac MRI parametric mapping (T1 and extracellular volume mapping) adds further discriminatory ability by quantifying diffuse fibrosis—an important determinant of remodeling capacity. Incorporating such tissue characterization into viability assessment enhances the ability to distinguish reversible dysfunction from fixed injury.[70]

Technological advancements are also reshaping coronary physiology. Noninvasive or angiography-derived physiologic indices such as quantitative flow ratio (QFR) provide an opportunity to assess lesion significance without pressure wires or vasodilators, offering a safer and more feasible approach in high-risk ICM patients. Similarly, machine-learning algorithms applied to CMR or PET datasets are beginning to identify complex patterns of perfusion, scar, and microvascular injury that may escape conventional visual assessment. These tools hold the potential to generate individualized risk models that integrate viability, ischemia, microvascular reserve, and structural remodeling, allowing clinicians to more accurately predict which patients stand to benefit from PCI.[70]

Looking forward, the most promising advances will depend on merging physiology, imaging, and artificial intelligence into a cohesive decision framework. By synthesizing multiple biomarkers of myocardial health—contractile reserve, scar burden, flow reserve, and tissue composition—future models may overcome the limitations seen in STICH and REVIVED, enabling more precise and personalized revascularization strategies in ICM.[70]

Conclusion

The prognostic value of PCI in ischemic cardiomyopathy (ICM) depends greatly on the interplay among myocardial viability, physiologic ischemia, and microvascular function. Although each of these domains provides important insights independently, outcomes from major trials such as STICH, PARR-2, and REVIVED make clear that no single factor can reliably predict benefit from revascularization. Instead, meaningful improvement in survival, ventricular function, or heart-failure progression occurs only when viable myocardium is paired with physiologically significant ischemia and sufficient microvascular reserve to allow restored epicardial flow to translate into improved tissue perfusion. This integrated approach helps explain why some patients with substantial viability fail to improve after PCI, while others with moderate dysfunction may experience robust recovery when ischemia is effectively relieved.

Modern advances in imaging and coronary physiology—ranging from PET flow quantification to CMR tissue characterization and pressure-derived indices such as FFR, iFR, and QFR—now allow much more precise assessment of the pathophysiologic substrates governing ventricular performance in ICM. These tools extend beyond simple identification of stenosis severity, offering a refined understanding of how myocardial substrate and coronary flow interact to determine post-PCI remodeling potential. At the same time, the recognition of microvascular dysfunction as a central determinant of outcomes underscores the need to move beyond epicardial anatomy alone when selecting patients for PCI.

Looking ahead, the integration of multimodal data—structural, metabolic, physiologic, and computational—will be essential for developing personalized revascularization strategies in ICM. Artificial intelligence and machine-learning models capable of synthesizing viability, perfusion, flow reserve, scar burden, and clinical risk markers may reshape the way clinicians determine who benefits most from PCI. Ultimately, the future of revascularization in ischemic cardiomyopathy lies not in expanding the use of PCI broadly, but in precisely identifying the subset of patients in whom restoring flow will meaningfully improve symptoms, ventricular performance, and long-term outcomes.

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