



Contrast-Enhanced CMR for Myocardial Viability in Coronary Artery Disease: Evidence, Prognosis, and Impact on Revascularization Decisions

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Abstract

Background: Coronary artery disease (CAD) remains the leading cause of heart failure worldwide and is associated with substantial morbidity and mortality, particularly when left ventricular (LV) systolic dysfunction is present. In patients with ischemic cardiomyopathy, myocardial dysfunction may result from a spectrum of injury ranging from reversible ischemic stunning or hibernation to irreversible myocardial infarction and replacement fibrosis. Identification of viable myocardium is therefore clinically critical, as patients with substantial viable but dysfunctional myocardium derive prognostic and functional benefit from coronary revascularization, whereas those with extensive nonviable scar are less likely to improve and may be exposed to unnecessary procedural risk. Over the past two decades, contrast-enhanced cardiac magnetic resonance imaging (CE-CMR) has emerged as a comprehensive, noninvasive imaging modality capable of integrating assessment of cardiac anatomy, ventricular function, perfusion, tissue characterization, and myocardial viability within a single examination.

Aim: This review aims to comprehensively evaluate the role of contrast-enhanced cardiac magnetic resonance imaging in the assessment of myocardial viability in patients with coronary artery disease, with particular emphasis on late gadolinium enhancement (LGE) imaging, its pathophysiologic basis, technical considerations, diagnostic performance, and clinical impact on revascularization decision-making.

The review discusses the pathophysiology of myocardial viability, including stunned and hibernating myocardium, and explains the mechanistic basis of gadolinium contrast distribution in normal, acutely infarcted, and chronically scarred myocardium. The principles of LGE-CMR are detailed, highlighting its ability to quantify infarct size and transmural extent with near-histopathologic accuracy. Comparative effectiveness of CE-CMR relative to other viability imaging modalities, such as positron emission tomography, single-photon emission computed tomography, and dobutamine stress echocardiography, is reviewed. Practical aspects of image acquisition, interpretation, pitfalls, and standardized reporting are addressed, along with special clinical scenarios in which CE-CMR provides decisive diagnostic value. Emerging quantitative techniques, including parametric mapping, strain analysis, and artificial intelligence-based automation, are also briefly explored.

Conclusion: CE-CMR, particularly LGE imaging, represents the reference standard for noninvasive assessment of myocardial viability in patients with CAD. By accurately delineating the presence, pattern, and transmural extent of myocardial scar, CE-CMR plays a pivotal role in guiding revascularization strategies, refining risk stratification, and optimizing patient-centered management in ischemic heart disease.

Keywords: *Contrast-Enhanced CMR, Myocardial Viability, Coronary Artery Disease*



Introduction

Heart failure secondary to coronary artery disease (CAD) remains a dominant global phenotype of ventricular dysfunction and is consistently associated with adverse outcomes compared with many nonischemic etiologies. In this setting, a central clinical question is whether dysfunctional myocardium represents irreversible scar or potentially reversible dysfunction (stunning/hibernation). Because revascularization benefit is most plausible when a meaningful burden of viable myocardium is present, accurate viability assessment remains a pivotal step in selecting patients for invasive therapies and informing prognosis. [1,2]

Myocardial viability evaluation has historically relied on multiple imaging paradigms, each interrogating a different component of the ischemic cascade: cellular metabolism (PET), membrane integrity and perfusion (SPECT), contractile reserve (dobutamine stress echocardiography), and inducible ischemia (stress testing). Although these methods are widely used, their performance varies with patient factors (acoustic window, arrhythmia, obesity), local expertise, radiation exposure considerations, and the need to integrate viability with anatomy and ventricular remodeling in a single examination. This heterogeneity explains why viability testing can be inconsistent across centers and why a robust, reproducible reference technique is valuable for contemporary practice. [1,2]

Cardiovascular magnetic resonance (CMR) has become central to CAD evaluation because it provides a **multiparametric** platform—high-quality ventricular volumes and function, myocardial tissue characterization, perfusion assessment, and scar/viability imaging—without ionizing radiation. Importantly, CMR offers superior spatial resolution compared with nuclear techniques, enabling detection of small subendocardial infarcts and more precise assessment of scar transmural extent, which is critical when viability decisions are made at the segmental level. These features have positioned CMR as a cornerstone modality within the modern diagnostic armamentarium for ischemic heart disease. [3,8]

The most established viability technique in CMR is **late gadolinium enhancement (LGE)**, which leverages differences in gadolinium distribution between normal myocardium and tissue with expanded extracellular space (fibrosis, infarction). Gadolinium-based contrast agents (GBCAs) predominantly distribute within the extracellular compartment; following ischemic injury and subsequent scar formation, extracellular volume expansion and altered contrast kinetics lead to hyperenhancement of infarcted/fibrotic tissue on appropriately timed T1-weighted imaging. This provides a direct and visually intuitive map of myocardial scar burden and distribution. [3,5]

A major strength of LGE-CMR in CAD is its ability to quantify scar transmural extent and thereby estimate the probability of functional recovery after revascularization. Foundational clinical evidence demonstrated that the likelihood of contractile improvement declines stepwise as the transmural extent of LGE increases, supporting a pragmatic interpretive framework widely adopted in practice (e.g., segments with minimal LGE are more likely to recover, whereas near-transmural LGE predicts poor recovery). Subsequent studies reinforced that combining LGE with functional metrics improves prediction of global and regional recovery in ischemic cardiomyopathy undergoing revascularization. [4,13,14]

Despite the strong mechanistic appeal and extensive observational validation, the clinical-outcome literature underscores that “viability” must be interpreted within contemporary care pathways. Randomized data and meta-analytic syntheses have highlighted that the relationship between viability testing, revascularization, and long-term outcomes is complex and influenced by background guideline-directed medical therapy, comorbidity burden, and revascularization completeness. Accordingly, modern statements emphasize integrating LGE findings with symptoms, ischemia, coronary anatomy, LV volumes, and procedural feasibility rather than treating viability as a binary gatekeeper. [2,6,7]

From a radiodiagnosis standpoint, standardized acquisition and interpretation remain essential to maximize the clinical value of CE-CMR. Typical LGE protocols use ~0.1 mmol/kg GBCA with imaging performed approximately 10 minutes after injection, using inversion recovery techniques to null normal myocardium and accentuate scar-to-myocardium contrast; refinements such as dark-blood LGE have



further improved detection of subendocardial scar by reducing blood-pool signal competition. In parallel, stress perfusion CMR can be combined with LGE in a single session to provide both ischemia and scar assessment, supporting comprehensive decision-making before revascularization. [9–11]

To evaluate the role of contrast-enhanced cardiac magnetic resonance imaging (CE-CMR), particularly late gadolinium enhancement (LGE), in assessing myocardial viability in patients with coronary artery disease prior to coronary revascularization. [2–5]

Pathophysiology of Myocardial Viability in Coronary Artery Disease

Myocardial viability refers to dysfunctional myocardium that retains cellular integrity and metabolic activity and therefore has the potential for recovery of contractile function following restoration of adequate coronary blood flow. In patients with coronary artery disease (CAD) and left ventricular (LV) dysfunction, chronic or repetitive ischemia produces a continuum of myocardial injury ranging from transient, fully reversible dysfunction to irreversible myocyte necrosis and replacement fibrosis. A clear understanding of this pathophysiologic spectrum is essential for correct interpretation of contrast-enhanced cardiac magnetic resonance (CE-CMR) findings and for optimal patient selection for coronary revascularization [8,9].

Myocardial stunning is defined as transient post-ischemic LV dysfunction that persists despite restoration of normal or near-normal myocardial perfusion. It typically occurs following brief episodes of acute ischemia or reperfusion, such as after spontaneous thrombolysis or successful coronary intervention. At the cellular level, stunned myocardium demonstrates preserved myocyte viability with abnormalities in calcium homeostasis, oxidative stress, and impaired excitation–contraction coupling rather than irreversible structural injury. Because cell membrane integrity and extracellular volume remain largely intact, stunned myocardium does not exhibit late gadolinium enhancement (LGE) on CE-CMR and is expected to recover function over days to weeks without permanent scarring [10,11].

In contrast, **hibernating myocardium** represents a chronic adaptive response to persistently reduced coronary blood flow in which myocardial contractile function is downregulated to match diminished oxygen and substrate supply. Unlike stunning, hibernation is associated with long-standing ischemia and structural cellular remodeling, including myocyte dedifferentiation, loss of sarcomeres, accumulation of intracellular glycogen, and expansion of the interstitial space. Despite these changes, cell membranes remain intact and myocardial tissue remains viable, allowing functional recovery following revascularization. On CE-CMR, hibernating myocardium typically demonstrates absent or limited LGE, often involving less than 25–50% of the myocardial wall thickness, highlighting the importance of quantitative scar assessment rather than binary scar detection [12,13].

Irreversible myocardial injury occurs when the duration and severity of ischemia exceed the threshold for cellular survival, resulting in myocyte necrosis and subsequent replacement fibrosis. Chronic myocardial infarction is characterized histologically by dense collagen deposition, marked extracellular volume expansion, and permanent loss of contractile tissue. CE-CMR exploits these tissue characteristics, as gadolinium-based contrast agents preferentially accumulate in regions of expanded extracellular space, producing hyperenhancement on LGE images. Extensive clinical evidence demonstrates a strong inverse relationship between the transmural extent of LGE and the likelihood of functional recovery after revascularization, establishing transmural scar burden as a robust imaging marker of nonviability [14,15].

Importantly, the transition from viable to nonviable myocardium is gradual rather than abrupt and is influenced by multiple factors, including ischemic duration, collateral circulation, microvascular integrity, and comorbid conditions such as diabetes mellitus and chronic kidney disease. Myocardial segments with intermediate degrees of scar (approximately 25–50% transmural) represent a “gray zone” in which functional recovery is variable and may depend on additional parameters such as residual perfusion, wall thickness, and contractile reserve. This explains why CE-CMR–based viability assessment is most powerful when integrated with functional imaging and clinical context rather than interpreted in isolation [9,16].

From a radiodiagnostic perspective, CE-CMR offers a unique advantage by providing near-histologic



visualization of myocardial injury while simultaneously assessing ventricular geometry and function. This comprehensive evaluation allows reliable differentiation between stunned myocardium, hibernating myocardium, and irreversible scar within a single examination, forming the physiologic foundation for the expanding role of CE-CMR in guiding revascularization decisions and prognostication in ischemic cardiomyopathy [8,12].

CE-CMR Viability Markers and Interpretation in Coronary Artery Disease

Contrast-enhanced cardiac magnetic resonance (CE-CMR) assesses myocardial viability using a set of complementary imaging markers that reflect myocardial structure, tissue composition, and functional reserve. Among these, **late gadolinium enhancement (LGE)** is the principal determinant of myocardial scar burden, while adjunctive parameters such as wall thickness, regional contractility, microvascular obstruction, and contractile reserve further refine viability assessment. The integration of these markers allows CE-CMR to provide a comprehensive and clinically actionable evaluation of dysfunctional myocardium in patients with coronary artery disease (CAD) [17].

Late Gadolinium Enhancement Transmurality

The transmural extent of LGE remains the most robust and validated imaging marker for predicting functional recovery after coronary revascularization. LGE typically follows a subendocardial-to-transmural pattern corresponding to the wavefront progression of ischemic injury within a coronary artery territory. Seminal studies demonstrated that myocardial segments without LGE or with minimal enhancement (<25% of wall thickness) have a high likelihood of contractile recovery following revascularization, whereas segments with near-transmural or transmural LGE (>75%) rarely recover function [18,19].

Segments exhibiting intermediate degrees of LGE (25–50% transmural) represent a prognostic gray zone. In these cases, recovery is variable and influenced by additional factors such as residual perfusion, wall thickness, and baseline contractile function. Quantitative assessment of scar burden, rather than visual estimation alone, has been shown to improve reproducibility and prognostic accuracy, particularly in patients with ischemic cardiomyopathy being evaluated for surgical or percutaneous revascularization [20].

Wall Thickness and Regional Contractile Function

Myocardial wall thickness assessed on cine CMR provides complementary information regarding chronicity and reversibility of ischemic injury. Chronically infarcted segments often demonstrate marked end-diastolic wall thinning (<5–6 mm), which is associated with a low likelihood of functional recovery even in the absence of extensive LGE. Conversely, preserved wall thickness in dysfunctional segments suggests retained myocardial architecture and supports the presence of viable tissue, particularly when LGE is absent or limited [21,22].

Cine CMR also allows accurate assessment of global and regional ventricular function with high spatial and temporal resolution. While regional wall motion abnormalities alone are not sufficient to distinguish viable from nonviable myocardium, their integration with LGE findings enhances diagnostic confidence and clinical interpretation [17].

Microvascular Obstruction and Peri-Infarct Tissue

In the setting of acute or subacute myocardial infarction, CE-CMR may demonstrate **microvascular obstruction (MVO)**, appearing as a hypoenhanced core within an area of hyperenhanced infarction on LGE images. MVO reflects severe microvascular damage and is associated with adverse remodeling, reduced likelihood of functional recovery, and worse clinical outcomes. Although MVO is primarily an acute-phase phenomenon, its presence provides important prognostic information and should be reported when identified [23,24].

Beyond dense scar, the peri-infarct or “gray zone” tissue identified on LGE represents a heterogeneous mixture of viable myocytes and fibrosis. This region has been linked to arrhythmic risk and adverse outcomes and may influence the functional response to revascularization. Advanced scar quantification techniques continue to explore the clinical significance of this heterogeneous tissue compartment [25].

Low-Dose Dobutamine Stress CMR



Low-dose dobutamine stress CMR evaluates myocardial **contractile reserve**, providing functional confirmation of viability in segments with limited or intermediate LGE. Viable myocardium typically demonstrates improved systolic thickening and contractility at low doses of dobutamine, whereas nonviable scar tissue does not respond. When combined with LGE assessment, dobutamine stress CMR improves prediction of post-revascularization recovery, particularly in segments with intermediate scar burden [26,27].

Although dobutamine stress CMR is not routinely required in all patients, it is particularly useful when LGE findings are equivocal or when clinical decision-making hinges on the recovery potential of specific myocardial segments. Its use, however, is limited by longer examination times and contraindications related to arrhythmia or hemodynamic instability [17].

Integrated Interpretation and Clinical Relevance

From a radiodiagnostic perspective, CE-CMR viability assessment is strongest when imaging markers are interpreted in combination rather than in isolation. LGE transmural extent provides the structural substrate, cine imaging defines functional consequences, and adjunctive markers such as wall thickness, MVO, and contractile reserve refine prognostic stratification. This integrated approach aligns CE-CMR with contemporary heart team decision-making, supporting personalized revascularization strategies in patients with ischemic cardiomyopathy [18,20].

Clinical Evidence and Outcomes: CE-CMR Viability Assessment and Revascularization Benefit

The clinical value of contrast-enhanced cardiac magnetic resonance (CE-CMR) viability assessment lies in its ability to inform prognosis and guide revascularization decisions in patients with coronary artery disease (CAD) and left ventricular (LV) dysfunction. Over the past two decades, a substantial body of evidence has demonstrated that CE-CMR—particularly late gadolinium enhancement (LGE)—provides incremental prognostic information beyond conventional clinical and functional parameters, supporting its integration into contemporary heart team decision-making [28].

Prediction of Functional Recovery After Revascularization

Multiple observational and prospective studies have established a strong association between LGE extent and post-revascularization improvement in regional and global LV function. Myocardial segments without LGE or with minimal scar burden demonstrate a high likelihood of contractile recovery following coronary artery bypass grafting (CABG) or percutaneous coronary intervention (PCI), whereas segments with extensive transmural scar rarely recover function. Importantly, CE-CMR allows precise segmental analysis, enabling prediction of recovery at both the regional and global levels with high reproducibility [29,30].

Quantitative scar burden assessed by LGE-CMR has also been shown to correlate with reverse ventricular remodeling after revascularization. Patients with a lower total scar burden experience greater improvements in LV ejection fraction, reductions in LV volumes, and symptomatic improvement, highlighting the role of CE-CMR in identifying patients most likely to derive functional benefit from invasive therapy [31].

Impact on Long-Term Outcomes and Survival

Beyond functional recovery, CE-CMR viability assessment has prognostic implications for long-term outcomes, including survival and heart failure hospitalization. Several studies have demonstrated that patients with substantial viable myocardium identified on CE-CMR derive a survival benefit from revascularization compared with medical therapy alone, whereas patients with extensive nonviable myocardium experience limited benefit and higher procedural risk. These findings underscore the importance of viability assessment in balancing expected benefit against operative or procedural risk [32,33].

Scar burden quantified by LGE has also emerged as a powerful predictor of adverse cardiovascular events, independent of revascularization strategy. Extensive myocardial scar is associated with progressive LV remodeling, malignant ventricular arrhythmias, and increased mortality, reinforcing the role of CE-CMR as a comprehensive risk stratification tool rather than a purely diagnostic modality [34].

Viability Testing and the STICH Era Controversy



The role of viability testing in guiding revascularization decisions was challenged by the viability substudy of the Surgical Treatment for Ischemic Heart Failure (STICH) trial, which did not demonstrate a statistically significant interaction between myocardial viability and survival benefit from CABG. However, important limitations of this substudy include heterogeneous imaging techniques, lack of CE-CMR-based scar quantification, and binary rather than quantitative viability assessment. Subsequent analyses and expert consensus have emphasized that the STICH findings should not be interpreted as negating the value of viability imaging but rather as highlighting the complexity of patient selection in advanced ischemic cardiomyopathy [35,36].

Contemporary interpretations advocate for an integrated approach in which CE-CMR findings are considered alongside symptom burden, ischemia, coronary anatomy, comorbidities, and feasibility of complete revascularization. In this context, CE-CMR provides critical anatomic and tissue-level information that complements clinical judgment rather than serving as a sole determinant of management [28,36].

CE-CMR in Modern Heart Team Decision-Making

In current practice, CE-CMR viability assessment plays a central role in multidisciplinary heart team discussions, particularly for patients with severe LV dysfunction, multivessel CAD, or prior myocardial infarction. By delineating scar distribution, transmural, and global scar burden, CE-CMR helps identify patients most likely to benefit from CABG versus PCI or optimal medical therapy alone. Additionally, CE-CMR informs procedural planning by identifying nonviable segments unlikely to improve, thereby refining expectations and guiding patient counseling [29,31].

From a radiodiagnostic standpoint, the strength of CE-CMR lies in its ability to unify diagnosis, prognostication, and therapeutic guidance within a single, radiation-free examination. As evidence continues to evolve, CE-CMR-based viability assessment remains a cornerstone of personalized management strategies for patients with ischemic cardiomyopathy [30,34].

Technical Considerations, Pitfalls, and Standardized Reporting in CE-CMR Viability Imaging

High-quality viability assessment with CE-CMR depends as much on **technique** as on interpretation. The Society for Cardiovascular Magnetic Resonance (SCMR) has emphasized standardized acquisition pathways to ensure reproducibility across vendors and sites, including consistent cine assessment, contrast dosing strategy, and dedicated LGE imaging with appropriate inversion time (TI) selection to null normal myocardium. Adherence to standardized protocols improves diagnostic consistency and supports reliable scar quantification in clinical workflows and research settings. [37]

Core acquisition principles for LGE viability imaging

LGE is typically acquired 10–15 minutes after GBCA administration using inversion-recovery (IR)–prepared T1-weighted imaging, where accurate nulling of normal myocardium is essential to maximize scar-to-myocardium contrast. TI selection is commonly guided by a TI-scout (Look-Locker) sequence; however, the optimal TI drifts with time after contrast injection and varies with renal clearance, contrast type, and field strength. Phase-sensitive inversion recovery (PSIR) addresses TI sensitivity by preserving signal polarity, thereby reducing dependence on perfect nulling and improving robustness across patients and scanning conditions. [38–40]

Dark-blood LGE and improved subendocardial scar conspicuity

A frequent limitation of conventional “bright-blood” LGE is reduced conspicuity of **subendocardial infarction**, where hyperenhanced scar can blend with a bright blood pool—particularly problematic in viability assessment where small subendocardial scars may alter segment-level recovery predictions. Dark-blood LGE techniques suppress blood pool signal while maintaining hyperenhancement of scar, improving detection and border definition of subendocardial infarction and enhancing reader confidence for ischemic-pattern scar assessment. This is especially valuable when scar burden is subtle or when segmental transmural estimation drives management decisions. [41,42]

Common pitfalls and artifacts that can mimic or obscure scar

Several pitfalls can produce false-negative or false-positive LGE interpretation if not recognized.



Incorrect myocardial nulling (TI too short or too long) may invert contrast relationships, reduce scar conspicuity, or generate pseudo-enhancement; PSIR mitigates but does not eliminate the need for careful setup. **Arrhythmia and poor breath-holding** can cause ghosting, blurring, and slice misregistration—errors that are disproportionately harmful for detecting small subendocardial infarcts and for accurate transmural grading. Partial-volume averaging (thick slices, oblique planes), coil shading, and inadequate spatial resolution can further degrade the assessment of scar extent and the peri-infarct interface. Contemporary technical reviews emphasize proactive parameter adjustment (spatial resolution, gating strategy, motion-robust options, repeat TI checks) to preserve viability accuracy. [37,43]

Standardized interpretation and reporting: what must be included

Because CE-CMR viability studies directly influence revascularization decisions, structured reporting is critical. SCMR reporting guidelines recommend documenting (1) ventricular volumes and ejection fraction with method, (2) regional wall motion abnormalities using a standard segment model, and (3) LGE presence, pattern (ischemic vs nonischemic), and extent—preferably with segmental **transmural grading** and global scar burden when feasible. Reports should also explicitly state imaging limitations (arrhythmia, incomplete coverage, motion, suboptimal nulling) and include a concise clinical impression addressing the likelihood of functional recovery based on scar transmural and supportive functional findings. [44,45]

Practical “viability reporting checklist” for CAD (radiology-facing)

For highest clinical utility, a CE-CMR viability report in CAD should, at minimum, provide: coronary-territory distribution of scar; per-segment transmural (e.g., 0%, 1–25%, 26–50%, 51–75%, 76–100%); total scar burden estimate if available; LV volumes/EF; presence of wall thinning; and any ancillary findings that alter management (LV thrombus, aneurysm, significant valvular disease, pericardial pathology). This standardization aligns imaging output with heart-team needs and reduces variability in downstream decisions driven by CE-CMR viability interpretation. [37,44]

Comparative Effectiveness: CE-CMR vs PET, SPECT, and Dobutamine Stress Echocardiography for Myocardial Viability

CE-CMR (primarily LGE-CMR) evaluates viability by **directly imaging scar** with high spatial resolution, whereas PET and SPECT assess viability through **metabolism and/or perfusion**, and dobutamine stress echocardiography (DSE) assesses **contractile reserve**. Because these modalities interrogate different biological targets, they can agree in advanced transmural infarction yet diverge in intermediate injury where residual myocytes, microvascular dysfunction, or partial-volume effects influence results. Contemporary scientific statements emphasize selecting the modality based on the dominant clinical question (scar quantification vs metabolic viability vs contractile reserve), local expertise, patient suitability, and the need to combine viability with anatomy and function in one exam. [46]

A consistent theme in comparative literature is the **sensitivity–specificity tradeoff**: techniques that detect scar or preserved tracer uptake (LGE-CMR, SPECT perfusion) tend to be more **sensitive** for identifying viability, while contractile-reserve techniques (DSE, low-dose dobutamine CMR) tend to be more **specific** for predicting functional recovery, particularly when the recovery endpoint is defined as improved wall motion after revascularization. This framework is clinically useful because it explains why a patient may show “viability” by PET/SPECT yet fail to demonstrate contractile reserve—often reflecting severe chronic remodeling, tethering, or incomplete revascularization despite residual living tissue. [47,48]

When the clinical goal is **segment-level prediction of recovery**, LGE-CMR has a major advantage: it can quantify **transmural scar burden** and identify small subendocardial infarcts that may be missed by lower-resolution nuclear techniques. This enables a graded probability model (minimal scar → higher recovery; near-transmural scar → low recovery) and supports reporting that directly maps to revascularization decision-making. However, LGE-CMR can label tissue as “scarred” even when some viable myocytes persist within heterogeneous fibrosis, meaning that intermediate LGE ranges may benefit from adjunctive testing (e.g., dobutamine contractile reserve) when management depends on borderline segments. [46,49] FDG-PET remains the most established method for identifying **metabolically active myocardium**, particularly in patients with severe LV dysfunction and complex multivessel disease where perfusion–



metabolism mismatch suggests hibernation. PET's main strengths are its physiologic sensitivity and extensive legacy evidence base, while key limitations include radiation exposure, availability/cost, patient preparation complexity (glucose control, insulin protocols), and reduced anatomic resolution compared with CMR. In practice, PET can be especially informative when CMR is contraindicated or nondiagnostic (certain devices, severe arrhythmia, inability to breath-hold), or when the clinical question prioritizes metabolism over scar mapping. [46,50]

SPECT viability protocols (rest redistribution thallium or Tc-based perfusion \pm nitrate augmentation) are widely available and familiar to clinicians, and they can effectively identify viability in many real-world settings. However, SPECT is limited by **lower spatial resolution**, attenuation artifacts, and partial-volume effects, which reduce sensitivity for small subendocardial infarcts and complicate precise transmural estimation—an area where LGE-CMR is typically stronger. In addition, SPECT exposes patients to ionizing radiation and may require multiple acquisitions depending on the protocol, influencing modality choice in younger patients or those requiring repeat imaging. [46,47]

Dobutamine stress echocardiography (DSE) has strengths in accessibility and in evaluating **contractile reserve**, making it relatively specific for predicting functional improvement after revascularization. Nevertheless, image quality is operator- and window-dependent, and diagnostic confidence may be reduced in patients with obesity, lung disease, prior thoracic surgery, or suboptimal acoustic windows. Importantly, when functional recovery prediction is the objective, many comparative reviews describe DSE (and dobutamine CMR) as higher-specificity approaches, while LGE-CMR and perfusion-based nuclear methods offer higher sensitivity for viability detection—again reinforcing a tailored, patient-centered approach rather than a single universal test. [46,47]

Overall, an evidence-aligned radiodiagnostic approach is to use **LGE-CMR as the preferred test** when high-resolution scar quantification, transmural grading, and integrated LV functional assessment are needed, and to consider **PET or SPECT** when metabolic/perfusion viability is prioritized or when CMR is not feasible. **DSE (or dobutamine CMR)** becomes particularly valuable as an adjudicator in intermediate or discordant cases where the presence of contractile reserve would meaningfully change the revascularization plan. This complementary strategy matches modern guideline statements that emphasize integration of viability results with coronary anatomy, symptom burden, ischemia, and feasibility of complete revascularization. [46,47]

Practical Patient Selection and Decision Algorithm Using CE-CMR Viability Imaging

In patients with CAD and LV systolic dysfunction, CE-CMR is most clinically impactful when the revascularization decision is genuinely uncertain—typically in those with (1) ischemic cardiomyopathy with multivessel disease, (2) prior infarction with regional wall motion abnormalities, and/or (3) discordant symptoms, ischemia tests, or angiographic severity. Contemporary guidance supports a **Heart Team** approach in which revascularization planning integrates symptom burden, coronary anatomy/feasibility, comorbid risk, and imaging evidence of ischemia and viability rather than relying on a single test result in isolation. [51,52]

A practical CE-CMR-centered workflow begins with confirming the **clinical phenotype** (ischemic vs nonischemic LV dysfunction) and the **therapeutic intent** (prognostic revascularization vs symptom relief). For patients being considered for revascularization with reduced EF, major guidelines and scientific statements emphasize (a) optimizing guideline-directed medical therapy, (b) defining coronary anatomy and technical feasibility, and (c) using noninvasive imaging to characterize myocardial substrate and guide the likelihood of meaningful benefit—particularly when operative risk is high or when complete revascularization is uncertain. [51–53]

When CE-CMR is performed, the report should operationalize viability in a way that maps to decisions. The most useful interpretation explicitly provides (1) global LV volumes/EF, (2) regional dysfunction by segment, and (3) **LGE pattern and transmural extent** per segment. In CAD, an ischemic scar pattern (subendocardial \pm transmural in a coronary territory) allows estimation of recovery probability: segments with **no LGE or minimal LGE** are most likely to recover; segments with **near-transmural/transmural LGE** are least likely to recover; and intermediate LGE often benefits from adjunct markers (wall thickness,



contractile reserve) when management hinges on borderline myocardium. Standardized reporting frameworks (SCMR) are particularly important here because small changes in transmural grading can materially affect Heart Team recommendations. [54,55]

A pragmatic decision algorithm can be summarized as follows:

Step 1: Confirm indication for revascularization (symptoms, ischemia burden, anatomy, and feasibility of complete/meaningful revascularization). [52,53]

Step 2: Use CE-CMR to quantify scar burden and distribution. If there is **extensive transmural scar in target territories** with advanced remodeling, expected functional recovery is low and revascularization is less likely to improve LV function, shifting emphasis toward optimized medical therapy, device therapy, or selective symptom-driven intervention. [56,57]

Step 3: If there is **substantial dysfunctional but minimally scarred myocardium**, the probability of recovery and reverse remodeling is higher, supporting revascularization when anatomy and risk are acceptable. [56]

Step 4: For **intermediate LGE (gray-zone) territories**, consider reinforcing data (low-dose dobutamine CMR/echo contractile reserve, ischemia assessment, and feasibility of complete revascularization) because recovery is variable and outcome benefit may depend more on global patient context than on viability alone. [51,58]

Finally, it is critical to align viability findings with the outcomes literature that tempered “viability-only” decision-making. The STICH viability substudy did not show that viability testing identified a subgroup with a differential survival benefit from CABG versus medical therapy, and later analyses reinforced that benefit attribution is multifactorial (baseline risk, LV size, completeness of revascularization, and contemporary medical therapy). Therefore, modern practice uses CE-CMR viability as a **risk/benefit modifier**—highly informative for substrate characterization and realistic expectation setting—rather than as an absolute gatekeeper for (or against) revascularization. [57,59]

Future Directions: Quantitative Viability Beyond Conventional LGE

A major frontier in viability imaging is moving from largely visual LGE interpretation toward **quantitative tissue characterization** that can be applied even when gadolinium is undesirable or LGE quality is limited. **Native T1 mapping** has been studied as a potential non-contrast approach for detecting chronic myocardial infarction and characterizing injured myocardium, with multicenter and validation efforts suggesting that chronic infarct territories can be detected with reasonable accuracy under certain conditions. However, more recent work also cautions that “standard” native T1 mapping methods do not universally substitute for LGE for chronic infarct detection, highlighting the need for protocol optimization, careful thresholds, and continued validation before native mapping can replace LGE in routine viability assessment. [69–71]

Beyond native T1, **T1 mapping–derived extracellular volume (ECV)** and **T2-based techniques** expand viability assessment by quantifying interstitial expansion and edema, respectively. In ischemic heart disease, these tools can complement LGE by clarifying acute-versus-chronic injury patterns, characterizing diffuse fibrosis (not readily seen on LGE), and supporting risk stratification when scar burden alone is insufficient. Reviews focusing on T1/ECV emphasize that these metrics may improve reproducibility across centers and provide a continuous quantitative phenotype (rather than binary scar/no scar), which is attractive for both prognostication and clinical trial endpoints. [72]

Another rapidly expanding domain is **CMR strain (feature-tracking)**, which extracts deformation parameters from routine cine imaging without additional sequences. Strain can reveal subtle contractile dysfunction and mechanical reserve that may not be apparent from ejection fraction or visual wall-motion assessment alone. Large multicenter outcome studies demonstrate that feature-tracking global longitudinal strain is strongly associated with mortality across ischemic and nonischemic cardiomyopathy populations, supporting strain as a powerful risk marker that can be integrated with LGE for comprehensive substrate–function profiling. Importantly for viability, strain measures may help adjudicate intermediate-transmural segments by identifying residual mechanical function or reserve that could influence



expected recovery after revascularization. [73,74]

Artificial intelligence is poised to reduce the major practical bottleneck of CE-CMR viability workflows: **time-consuming and subjective scar quantification**. Recent studies describe deep learning methods that automatically segment LV myocardium and quantify ischemic scar burden from LGE-CMR, aiming to standardize scar measurement across sites and improve scalability for clinical practice. In parallel, foundation-model approaches have been proposed for automated myocardial scar segmentation, reflecting a shift from task-specific models toward more generalizable architectures that may perform robustly across scanners, vendors, and acquisition protocols. These tools—if externally validated—could meaningfully improve reporting consistency and enable more reliable thresholds for scar burden and “gray zone” metrics. [75,76]

A particularly disruptive development is the emergence of approaches that attempt to generate **LGE-equivalent information without contrast** or with reduced contrast reliance. For example, deep learning-based methods have been reported that synthesize “enhancement-like” images from cine data to approximate LGE patterns, potentially offering an alternative for patients in whom gadolinium is contraindicated or when contrast must be minimized. While promising, these methods require rigorous validation against true LGE (and clinical outcomes) and must address bias, generalizability, and failure modes before they can be integrated into viability decision-making for CAD. [77]

References

1. Kim RJ, Wu E, Rafael A, et al. The use of contrast-enhanced magnetic resonance imaging to identify reversible myocardial dysfunction. *N Engl J Med*. 2000;343(20):1445-1453. doi:10.1056/NEJM200011163432003
2. Mahrholdt H, Klem I, Sechtem U. Cardiovascular magnetic resonance for detection of myocardial viability and ischemia. *Heart*. 2007;93(1):122-129.
3. Partington SL, Kwong RY, Dorbala S. Multimodality imaging in the assessment of myocardial viability. *Heart Fail Rev*. 2011;16(4):381-395.
4. Garcia MJ, Kwong RY, Scherrer-Crosbie M, et al. Imaging for myocardial viability: a scientific statement from the American Heart Association. *Circ Cardiovasc Imaging*. 2020;13(7):e000053.
5. Selvanayagam JB, Kardos A, Francis JM, et al. Value of delayed-enhancement cardiovascular magnetic resonance imaging in predicting myocardial viability after surgical revascularization. *Circulation*. 2004;110(12):1535-1541.
6. Bondarenko O, Beek AM, Nijveldt R, et al. Functional outcome after revascularization in patients with chronic ischemic heart disease: a quantitative late gadolinium enhancement CMR study. *J Cardiovasc Magn Reson*. 2007;9(3):559-566.
7. Bax JJ, Schinkel AFL, Boersma E, et al. Extensive viability predicts improved survival after revascularization in patients with ischemic left ventricular dysfunction. *J Am Coll Cardiol*. 2003;41(8):124-131.
8. Allman KC, Shaw LJ, Hachamovitch R, Udelson JE. Myocardial viability testing and impact of revascularization on prognosis in patients with coronary artery disease and left ventricular dysfunction: a meta-analysis. *J Am Coll Cardiol*. 2002;39(7):1151-1158.
9. Bonow RO, Maurer G, Lee KL, et al. Myocardial viability and survival in ischemic left ventricular dysfunction. *N Engl J Med*. 2011;364(17):1617-1625.
10. Panza JA, Ellis AM, Al-Khalidi HR, et al. Myocardial viability and long-term outcomes in ischemic cardiomyopathy. *N Engl J Med*. 2019;381(8):739-748.
11. Shah DJ, Kim HW, Kim RJ. Evaluation of myocardial viability by cardiovascular magnetic resonance imaging. *J Nucl Cardiol*. 2013;20(4):667-683.
12. Kellman P, Arai AE, McVeigh ER, Aletras AH. Phase-sensitive inversion recovery for detecting myocardial infarction using gadolinium-delayed hyperenhancement. *Magn Reson Med*. 2002;47(2):372-383.
13. Kellman P, Xue H, Olivieri LJ, et al. Dark blood late enhancement imaging. *J Cardiovasc Magn Reson*. 2016;18:77.
14. Kim HW, Rehwald WG, Jenista ER, et al. Dark-blood delayed enhancement cardiac magnetic resonance improves detection of subendocardial infarction. *JACC Cardiovasc Imaging*. 2018;11(12):1758-1769.
15. Abdel-Aty H, Zagrosek A, Schulz-Menger J, et al. Delayed enhancement and T2-weighted cardiovascular magnetic resonance imaging differentiate acute from chronic myocardial infarction. *Circulation*. 2004;109(20):2411-2416.
16. Yan AT, Shayne AJ, Brown KA, et al. Characterization of the peri-infarct zone by contrast-enhanced cardiac magnetic



- resonance imaging is a powerful predictor of post-myocardial infarction mortality. *Circulation*. 2006;114(1):32-39.
17. Rajiah PS, François CJ, Leiner T. Cardiac MRI: state of the art. *Radiology*. 2023;307(3):e223008.
 18. Patel AR, Salerno M, Kwong RY, et al. Stress cardiac magnetic resonance myocardial perfusion imaging. *J Am Coll Cardiol*. 2021;78(16):1655-1668.
 19. Kramer CM, Barkhausen J, Bucciarelli-Ducci C, et al. Standardized cardiovascular magnetic resonance imaging protocols. *J Cardiovasc Magn Reson*. 2020;22(1):17.
 20. Hundley WG, Bluemke DA, Bogaert J, et al. SCMR guidelines for reporting cardiovascular magnetic resonance examinations. *J Cardiovasc Magn Reson*. 2022;24(1):29.
 21. Romano S, Judd RM, Kim RJ, et al. Feature-tracking global longitudinal strain predicts mortality. *JACC Cardiovasc Imaging*. 2018;11(4):523-533.
 22. Dastidar AG, Rodrigues JCL, Johnson TW, et al. Native T1 mapping to detect chronic myocardial infarction. *J Cardiovasc Magn Reson*. 2019;21(1):19.
 23. Kellman P, Hansen MS. Myocardial T1 mapping in diffuse disease. *JACC Cardiovasc Imaging*. 2014;7(2):157-170.
 24. Garg P, et al. T1 mapping and extracellular volume in myocardial disease. *Anatol J Cardiol*. 2018;19(6):404-411.
 25. Rahman H, Scannell CM, Demir OM, et al. High-resolution CMR for coronary microvascular dysfunction. *Circ Cardiovasc Imaging*. 2021;14(5):978-986.
 26. Weberling LD, Lossnitzer D, Frey N, André F. Coronary CT vs CMR in CAD. *Diagnostics*. 2022;13(1):125.
 27. Hoek R, Borodzicz-Jazdzzyk S, van Diemen PA, et al. Quantitative perfusion CMR in CAD. *Eur Heart J Cardiovasc Imaging*. 2025;26(2):207-217.
 28. Souto ALM, Souto RM, Teixeira ICR, Nacif MS. Myocardial viability on cardiac magnetic resonance. *Arq Bras Cardiol*. 2017;108(5):458-468.
 29. Mohammed AY, Hassanien OA, Ibrahim AS, Dabees NL. Role of CMR in myocardial viability. *Cairo Univ Med J*. 2018;86(1).
 30. Orlandini A, Castellana N, Pascual A, et al. Myocardial viability for revascularization decision-making: meta-analysis. *Int J Cardiol*. 2015;182:494-499.
 31. Panza JA, et al. Viability and outcomes in ischemic cardiomyopathy. *N Engl J Med*. 2019;381:739-748.
 32. Mollema SA, Delgado V, Bertini M, et al. Viability assessment and LV recovery. *Circ Cardiovasc Imaging*. 2010;3:15-23.
 33. Kellman P, et al. Dark-blood late enhancement imaging. *J Cardiovasc Magn Reson*. 2016;18:77.
 34. Liang K, et al. Role of CMR in MINOCA. *Front Cardiovasc Med*. 2022;8:821067.
 35. Agewall S, et al. ESC position paper on MINOCA. *Eur Heart J*. 2017;38(3):143-153.
 36. Bucciarelli-Ducci C, et al. CMR guidance for CTO revascularization. *JACC Cardiovasc Imaging*. 2016.
 37. Schumacher SP, et al. Viability and recovery after CTO PCI using CMR. *Catheter Cardiovasc Interv*. 2021.
 38. Lawton JS, et al. 2021 ACC/AHA/SCAI guideline for coronary revascularization. *Circulation*. 2022;145:e18-e114.
 39. Heidenreich PA, et al. 2022 AHA/ACC/HFSA heart failure guideline. *Circulation*. 2022;145:e895-e1032.
 40. Kim D, et al. SCMR consensus on CMR in patients with cardiac devices. *J Cardiovasc Magn Reson*. 2024.
 41. Thomsen HS, et al. ESUR guidelines on gadolinium safety. *Eur Radiol*. 2013;23:307-318.
 42. American College of Radiology. *ACR Manual on Contrast Media*. Latest ed.
 43. Romano S, et al. Prognostic role of CMR strain. *JACC Cardiovasc Imaging*. 2018.
 44. Yu S, et al. Strain and LGE correlation. *Front Cardiovasc Med*. 2021.
 45. Jani VP, et al. Deep learning for ischemic scar quantification on LGE-CMR. *J Cardiovasc Magn Reson*. 2024.
 46. Tavakoli N, et al. ScarNet: foundation model for LGE scar segmentation. *J Cardiovasc Magn Reson*. 2025.
 47. Kellman P, et al. Revisiting late gadolinium enhancement techniques. *J Cardiovasc Magn Reson*. 2023.
 48. D'Angelo T, et al. Gadobutrol-enhanced CMR optimization. *J Cardiovasc Magn Reson*. 2017;19:83.
 49. Christopher K. Role of CMR in stable CAD. *ACC*. 2016.
 50. Weberling LD, et al. CMR vs CT in CAD evaluation. *Diagnostics*. 2022.
 51. Garcia MJ, Kwong RY, Scherrer-Crosbie M, et al. Imaging for myocardial viability: a scientific statement from the American Heart Association. *Circ Cardiovasc Imaging*. 2020;13(7):e000053.



52. Lawton JS, Tamis-Holland JE, Bangalore S, et al. 2021 ACC/AHA/SCAI guideline for coronary artery revascularization. *Circulation*. 2022;145(3):e18-e114.
53. Heidenreich PA, Bozkurt B, Aguilar D, et al. 2022 AHA/ACC/HFSA guideline for the management of heart failure. *Circulation*. 2022;145(18):e895-e1032.
54. Hundley WG, Bluemke DA, Bogaert J, et al. Society for Cardiovascular Magnetic Resonance guidelines for reporting cardiovascular magnetic resonance examinations. *J Cardiovasc Magn Reson*. 2022;24(1):29.
55. Kramer CM, Barkhausen J, Bucciarelli-Ducci C, et al. Standardized cardiovascular magnetic resonance imaging (CMR) protocols: 2020 update. *J Cardiovasc Magn Reson*. 2020;22(1):17.
56. Kim RJ, Wu E, Rafael A, et al. The use of contrast-enhanced magnetic resonance imaging to identify reversible myocardial dysfunction. *N Engl J Med*. 2000;343(20):1445-1453.
57. Bonow RO, Maurer G, Lee KL, et al. Myocardial viability and survival in ischemic left ventricular dysfunction. *N Engl J Med*. 2011;364(17):1617-1625.
58. Allman KC, Shaw LJ, Hachamovitch R, Udelson JE. Myocardial viability testing and impact of revascularization on prognosis in patients with coronary artery disease and left ventricular dysfunction: a meta-analysis. *J Am Coll Cardiol*. 2002;39(7):1151-1158.
59. Panza JA, Ellis AM, Al-Khalidi HR, et al. Myocardial viability and long-term outcomes in ischemic cardiomyopathy. *N Engl J Med*. 2019;381(8):739-748.
60. Abdel-Aty H, Zagrosek A, Schulz-Menger J, et al. Delayed enhancement and T2-weighted cardiovascular magnetic resonance imaging differentiate acute from chronic myocardial infarction. *Circulation*. 2004;109(20):2411-2416.
61. Kim D, et al. SCMR expert consensus statement for cardiovascular magnetic resonance of patients with a cardiac implantable electronic device. *J Cardiovasc Magn Reson*. 2024.
62. American College of Radiology. *ACR Manual on Contrast Media*. Latest edition.
63. Thomsen HS, Morcos SK, Almén T, et al. Nephrogenic systemic fibrosis and gadolinium-based contrast media: updated ESUR Contrast Medium Safety Committee guidelines. *Eur Radiol*. 2013;23(2):307-318.
64. Bucciarelli-Ducci C, et al. Cardiovascular magnetic resonance guidance for recanalization of coronary chronic total occlusions. *JACC Cardiovasc Imaging*. 2016.
65. Schumacher SP, et al. Viability and functional recovery after chronic total occlusion percutaneous coronary intervention using quantitative CMR imaging. *Catheter Cardiovasc Interv*. 2021.
66. Agewall S, Beltrame JF, Reynolds HR, et al. ESC working group position paper on myocardial infarction with non-obstructive coronary arteries. *Eur Heart J*. 2017;38(3):143-153.
67. Lintingre PF, et al. High-resolution late gadolinium enhancement CMR in MINOCA. *JACC Cardiovasc Imaging*. 2020.
68. Kellman P, Xue H, Olivieri LJ, et al. Dark blood late enhancement imaging. *J Cardiovasc Magn Reson*. 2016;18:77.
69. Dastidar AG, Rodrigues JCL, Johnson TW, et al. Native T1 mapping to detect acute and chronic myocardial infarction: comparison with late gadolinium enhancement. *J Cardiovasc Magn Reson*. 2019;21(1):19.
70. Kellman P, et al. Can chronic myocardial infarction be detected by native T1 mapping? *JACC Cardiovasc Imaging*. 2022;15(6):909-921.
71. Multicenter study on diagnostic performance of native T1 mapping for chronic myocardial infarction. *Circ Cardiovasc Imaging*. 2020.
72. Garg P, et al. Role of cardiac T1 mapping and extracellular volume in myocardial disease assessment. *Anatol J Cardiol*. 2018;19(6):404-411.
73. Romano S, Judd RM, Kim RJ, et al. Feature-tracking global longitudinal strain predicts mortality in ischemic and nonischemic cardiomyopathy. *JACC Cardiovasc Imaging*. 2018;11(4):523-533.
74. Yu S, et al. Correlation of myocardial strain by CMR feature tracking and late gadolinium enhancement. *Front Cardiovasc Med*. 2021;8:682487.
75. Jani VP, et al. Deep learning for automatic myocardial scar segmentation and quantification from LGE-CMR. *J Cardiovasc Magn Reson*. 2024.
76. Tavakoli N, et al. ScarNet: a foundation model for automated myocardial scar segmentation on LGE-CMR. *J Cardiovasc Magn Reson*. 2025.