Myocardial Viability in Patients Presenting with Myocardial Infarction: Pathophysiological Basis, Multimodality Assessment

Tarek Abdelmoneim Abdelaziz, Yasser Gaber Ibrahim, Mohamad Salah Abdelbasit, Mona Sabry Abd El-Rahman El-Sayed

Cardiology Department - Faculty of Medicine, Zagazig University, Egypt

Corresponding Author: Mona Sabry Abd El-Rahman El-Sayed

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Abstract

Background: Myocardial infarction initiates a dynamic continuum of ischemic injury ranging from transient contractile dysfunction to irreversible necrosis and replacement fibrosis. Although rapid reperfusion remains the principal determinant of myocardial salvage, the relationship between symptom duration and tissue viability is not absolute. Intermittent coronary occlusion, spontaneous reperfusion, residual antegrade flow, ischemic preconditioning, collateral circulation, microvascular integrity, and individual metabolic characteristics may preserve viable myocardium beyond conventional reperfusion windows. This variability becomes particularly important in patients presenting late after myocardial infarction, in whom angiographic coronary anatomy alone cannot establish whether dysfunctional myocardium remains capable of functional recovery. Myocardial viability testing therefore integrates assessment of perfusion, cellular membrane integrity, metabolism, contractile reserve, scar burden, and microvascular function. Echocardiography with low-dose dobutamine, single-photon emission computed tomography, positron emission tomography, and cardiovascular magnetic resonance interrogate different components of this biological spectrum and consequently have distinct diagnostic strengths and limitations. Angiographic indices including infarct-related artery patency, TIMI flow, corrected TIMI frame count, myocardial blush grade, collateral filling, and residual stenosis provide additional mechanistic and prognostic information but should not be considered direct substitutes for tissue characterization. Contemporary randomized evidence has also changed the interpretation of viability: recovery of dysfunctional myocardium and prognosis are related to scar burden and myocardial substrate, yet demonstration of viability alone does not reliably identify a subgroup that derives prognostic benefit from revascularization. This review examines the biological basis, invasive and non-invasive assessment, clinical interpretation, and contemporary role of myocardial viability testing after myocardial infarction, with particular attention to patients presenting beyond the conventional reperfusion window.

Keywords: myocardial viability, myocardial infarction, cardiovascular magnetic resonance, myocardial revascularization

Introduction

Acute myocardial infarction represents a competition between the duration and severity of ischemia and the capacity of the threatened myocardium to maintain cellular integrity until adequate perfusion is restored. Reperfusion as early as possible remains fundamental because cardiomyocyte death progresses with persistent coronary occlusion. The classical wavefront concept describes progression of irreversible injury from the subendocardium toward the epicardium, explaining why delay in restoring coronary flow increases infarct size. The clinical translation of this concept is the established relationship between ischemic time, myocardial salvage, left ventricular function, heart failure, and mortality. Yet the progression of necrosis is neither anatomically nor temporally identical among patients, which is central

to understanding myocardial viability after infarction. [1]

Contemporary treatment of STEMI therefore gives priority to immediate reperfusion, while also recognizing a distinct problem among late presenters. Some patients arrive after the conventional period of maximal salvage but retain substantial living myocardium because coronary occlusion was incomplete, intermittent, spontaneously reperfused, or supplemented by collateral circulation. Others have already developed extensive transmural scar despite a shorter reported symptom duration. The uploaded thesis protocol appropriately focuses on this heterogeneity, particularly among stable patients presenting after 48 hours, and proposes early angiographic characterization of a totally occluded, patent, or slow-flow infarct-related artery combined with subsequent assessment of viability and clinical outcomes.

The research gap is therefore not simply whether viable myocardium can be detected. Modern imaging can characterize viability with considerable precision. The more difficult clinical questions are whether anatomical and angiographic markers can predict the presence of recoverable myocardium, which imaging technique provides the information required for a particular patient, whether functional recovery equates to improved prognosis, and whether demonstration of viability should determine revascularization. Observational studies historically suggested marked survival advantages after revascularization when viable myocardium was present, whereas randomized data from STICH and REVIVED have weakened the concept of viability as an isolated treatment-selection test. [2]

This review examines myocardial viability specifically in the setting of myocardial infarction. It integrates infarct biology, myocardial stunning and hibernation, angiographic characteristics, microvascular reperfusion, echocardiographic and nuclear techniques, PET and CMR, and evidence relating viability to revascularization and clinical outcomes. Particular attention is given to late-presenting MI because this is the population in which conventional assumptions based on elapsed time are most likely to be inadequate.

The Biological Continuum of Ischemic Myocardial Injury

The myocardium subjected to abrupt coronary occlusion rapidly develops depletion of high-energy phosphate stores, anaerobic metabolism, intracellular acidosis, ionic disturbances, loss of contractile function, and eventually disruption of cellular membranes and irreversible myocyte death. The subendocardium is most vulnerable because of higher wall stress and greater compression of intramyocardial vessels during systole. With continued severe ischemia, necrosis expands toward the epicardium. The amount of myocardium ultimately lost reflects not only elapsed time but also the area at risk, metabolic demand, temperature, residual perfusion, collateral supply, and episodes of reperfusion. [3]

This biological heterogeneity explains why symptom-to-presentation time cannot function as an exact surrogate for tissue viability. A patient with abrupt persistent proximal LAD occlusion and poor collaterals may develop extensive necrosis rapidly, whereas another patient with intermittent occlusion, subtotal stenosis or robust collateral filling may preserve a substantial viable border zone for much longer. Experimental and clinical observations therefore support a probabilistic rather than deterministic relationship between time and irreversible injury. This principle is particularly relevant when deciding how to assess an asymptomatic and haemodynamically stable patient presenting one or several days after STEMI. [4]

Reperfusion itself introduces a second layer of injury. Restoration of epicardial patency does not guarantee normalization of microvascular perfusion because endothelial swelling, distal embolization, neutrophil accumulation, vasoconstriction, interstitial edema, capillary destruction and intramyocardial hemorrhage may produce microvascular obstruction. Consequently, two patients with angiographic TIMI grade 3 flow may have markedly different tissue-level reperfusion and subsequent functional recovery. This distinction provides the mechanistic basis for myocardial blush assessment, contrast echocardiography and CMR detection of microvascular obstruction. [5]

Myocardial Stunning

Myocardial stunning is prolonged but reversible contractile dysfunction after transient ischemia and

restoration of near-normal blood flow. The myocardium is alive and adequately reperfused but remains mechanically impaired for hours or days because restoration of perfusion precedes complete recovery of excitation-contraction coupling. Reactive oxygen species, calcium overload, altered myofilament responsiveness to calcium, mitochondrial dysfunction and transient abnormalities of cellular energetics have all been implicated. [6]

The concept has direct relevance after reperfused MI. Regional akinesia during the first hours after successful PCI does not necessarily represent transmural necrosis. Salvaged myocardium surrounding or overlying the infarct core may remain stunned and recover progressively. The degree of early LV dysfunction can consequently overestimate final permanent functional loss. Serial echocardiography, strain imaging and CMR are useful because regional recovery over subsequent days to months helps distinguish transient postischemic dysfunction from fixed scar. [7]

Stunning also explains why immediate post-reperfusion assessment of wall motion has limited specificity for nonviability. Contractile dysfunction is an endpoint shared by necrotic, stunned and hibernating myocardium. A test that measures resting contraction alone therefore cannot reliably discriminate them. Demonstration of preserved perfusion, contractile reserve, metabolism or limited scar is required to establish that an akinetic segment remains viable. [8]

Hibernating Myocardium

Hibernating myocardium traditionally describes chronically dysfunctional but viable myocardium associated with reduced coronary flow or recurrent ischemia and capable of functional improvement after effective revascularization. The concept initially proposed a protective down-regulation of contraction to match diminished oxygen supply. Subsequent work showed that chronic dysfunction can also result from repeated episodes of ischemia and stunning rather than continuously reduced resting flow. [9]

Long-standing hibernation is not a completely normal cellular state. Histological studies demonstrate progressive loss and disorganization of contractile proteins, accumulation of glycogen, mitochondrial alterations and interstitial fibrosis. The longer and more severe the ischemic burden, the less immediate and complete functional recovery may become after restoration of coronary flow. Thus, viability is better understood as a continuum rather than a binary state in which every living myocyte necessarily guarantees recovery of regional contraction. [10]

This distinction matters after MI because a dysfunctional territory may contain mixtures of dense scar, partially viable subendocardial or subepicardial tissue, stunned myocardium and chronically ischemic myocardium. An imaging result described simply as “viable” may therefore conceal important differences in the quantity, distribution and functional competence of surviving tissue. Contemporary interpretation increasingly evaluates scar burden and transmurality rather than relying on a single dichotomous viability label. [11]

Why Viability May Persist in Late-Presenting Myocardial Infarction

Late presentation does not necessarily mean persistent complete coronary occlusion since symptom onset. Thrombotic occlusion is dynamic: endogenous fibrinolysis, thrombus fragmentation, vasomotor changes, intermittent re-occlusion and spontaneous reperfusion can expose the myocardium to alternating periods of ischemia and flow. Residual antegrade flow through a subtotal lesion may also slow progression of necrosis. These mechanisms provide a pathophysiological explanation for preserved tissue despite prolonged symptom duration. [12]

Collateral circulation is particularly important. Sabia and colleagues studied patients with recent myocardial infarction and occluded infarct-related arteries and demonstrated an association between collateral perfusion of the infarct bed, persistent myocardial viability and subsequent improvement in regional function after successful angioplasty. The work remains directly relevant to late STEMI because it demonstrates that living myocardium may persist days or weeks after the index event when alternative perfusion is sufficient. [13]

Ischemic preconditioning may also reduce susceptibility to subsequent prolonged ischemia. Episodes of angina before infarction can activate endogenous protective signaling pathways and may delay cell

death, although the clinical magnitude of this protection varies. Together with collaterals and intermittent reperfusion, this means that the biological “age” of an infarct cannot always be inferred from the clock time between first pain and hospital presentation. [14]

These observations provide a rational basis for the protocol's attempt to integrate angiographic evidence of patency and collateralization with later myocardial perfusion imaging rather than assuming that all patients presenting beyond 48 hours have completed infarcts. At the same time, viability does not automatically establish an indication for PCI of a persistently occluded artery; treatment decisions must account for symptoms, ischemia, anatomy, LV function and randomized clinical evidence. [15]

Coronary Angiography as an Indirect Window on Viability

Coronary angiography defines the culprit anatomy, residual stenosis, epicardial flow, collateral channels and the distribution of multivessel disease. These observations can suggest conditions favorable or unfavorable for myocardial survival but do not directly determine whether myocytes within an abnormal segment remain alive. This distinction is crucial. A patent artery may supply myocardium that is already extensively necrotic, whereas an occluded artery may subtend viable tissue maintained by collaterals. [16]

The TIMI flow grading system established a practical method to describe epicardial reperfusion from grade 0, indicating no antegrade flow, through grades 1 and 2 to grade 3, representing normal or near-normal epicardial flow. Restoration of TIMI 3 flow is an important procedural objective, but it assesses the conduit artery rather than tissue-level perfusion. Patients may therefore demonstrate angiographic success while retaining substantial microvascular obstruction. [17]

The corrected TIMI frame count converts qualitative angiographic flow into a continuous quantitative measure by counting cine frames required for contrast to reach predefined distal landmarks. Because the LAD is longer than the other major coronary vessels, its raw frame count is corrected by a factor of approximately 1.7. The method can identify delayed epicardial contrast transit that may be overlooked by categorical TIMI grading and is particularly relevant when studying coronary slow flow or incomplete reperfusion. [18]

Myocardial blush grade adds information regarding tissue-level contrast penetration and washout. Grades 0 or 1 indicate absent or minimal myocardial blush, whereas grades 2 and 3 indicate progressively more preserved microvascular perfusion. Henriques and colleagues showed that combining TIMI 3 epicardial flow with preserved blush better characterizes effective reperfusion than epicardial flow alone. Thus, the protocol's simultaneous evaluation of TIMI flow and MBG is mechanistically justified. [19]

Collateral circulation is commonly graded angiographically using the Rentrop system, ranging from absent visible collaterals to complete opacification of the recipient epicardial vessel. Although conventional angiography visualizes only vessels above its spatial resolution and cannot directly quantify collateral conductance, high-grade filling indicates an alternative route of perfusion and can help explain preservation of myocardium distal to chronic or subacute occlusion. [20]

Angiographic variables should therefore be conceptualized as predictors or correlates of viability rather than viability tests. Combining IRA patency, quantitative flow, myocardial blush and collateral grade may improve biological characterization of a late infarct, but myocardial tissue imaging remains necessary when the clinical question is whether dysfunctional myocardium is scarred, stunned, hibernating or likely to recover after revascularization.

Resting Echocardiography and Myocardial Deformation

Transthoracic echocardiography is usually the first imaging technique used after MI because it rapidly provides LV and RV function, regional wall-motion abnormalities, chamber dimensions, filling patterns, mechanical complications and valvular abnormalities. Preserved wall thickness and systolic thickening favor viable myocardium, whereas severe thinning and persistent akinesia suggest chronic scar. Resting wall motion alone, however, has insufficient specificity because recently stunned myocardium may be completely akinetic despite preserved viability. [21]

Left ventricular ejection fraction remains a major prognostic marker but averages function across

normal, ischemic, stunned and infarcted territories. Global longitudinal strain provides additional sensitivity to regional and global systolic dysfunction and can identify impairment when EF is relatively preserved. Standardization efforts from the EACVI, ASE and industry have improved the conceptual framework for strain measurement, although vendor dependence, image quality and loading conditions remain relevant limitations. [22]

Regional strain and longitudinal strain recovery can complement conventional wall-motion assessment after MI. Severely reduced deformation in segments with preserved wall thickness does not itself prove irreversible injury, whereas improvement during follow-up can document functional recovery. Strain should consequently be interpreted alongside perfusion and scar imaging rather than as a standalone viability criterion.

Low-Dose Dobutamine Stress Echocardiography

Dobutamine stress echocardiography evaluates contractile reserve. Low doses increase myocardial inotropy with relatively modest increases in heart rate and oxygen demand. A dysfunctional segment that demonstrates improved systolic thickening at low-dose dobutamine contains sufficient functioning contractile apparatus to respond and is therefore likely to be viable. This gives DSE comparatively high specificity for predicting recovery after successful revascularization. [23]

Four response patterns can be observed. A sustained improvement suggests viable myocardium with preserved reserve; a biphasic response—initial improvement followed by deterioration at higher doses—indicates viability combined with inducible ischemia and is particularly informative. Worsening without initial augmentation suggests ischemia with limited resting reserve, while no change is more consistent with extensive scar when image quality is adequate. [24]

DSE is widely available, radiation-free and capable of simultaneously evaluating inducible ischemia. Its principal limitations are operator dependence, suboptimal acoustic windows, difficulty assessing severely remodeled ventricles, arrhythmia or inability to reach diagnostic stress, and reduced sensitivity compared with techniques that identify membrane integrity or metabolism. The uploaded review correctly highlights this general sensitivity-specificity trade-off between contractile-reserve techniques and radionuclide approaches.

Current stress echocardiography guidance supports low-dose dobutamine for evaluating viable dysfunctional myocardium when the result is expected to influence management. Quantitative strain during dobutamine may reduce subjectivity, although visual wall-motion analysis remains clinically established and requires experienced interpretation. [25]

SPECT Myocardial Perfusion Imaging

Single-photon emission computed tomography assesses viable tissue through radiotracer uptake dependent on perfusion and cellular integrity. Thallium-201 behaves as a potassium analogue and enters viable myocytes through active transport mechanisms. Redistribution therefore provides information beyond initial perfusion: a region showing reduced stress uptake but later redistribution is ischemic yet viable. [26]

Early redistribution protocols may underestimate viability in severely hypoperfused myocardium because tracer delivery is insufficient during the initial redistribution interval. Delayed imaging or reinjection can reveal uptake in regions initially appearing as fixed defects. This is why a persistent perfusion defect on a conventional early study should not automatically be equated with complete scar, particularly in severely stenosed or collateral-dependent territories. [27]

Technetium-99m sestamibi is retained within viable myocytes in relation to mitochondrial membrane potential and provides superior photon characteristics compared with thallium. Resting uptake reflects both delivery and cellular integrity. Gated SPECT additionally provides LV volumes, EF and regional wall motion, allowing perfusion and mechanical information to be integrated within a single study. [28]

The protocol's planned perfusion imaging approximately 40 days after presentation has a methodological advantage because early stunning and acute edema have had time to regress, allowing more stable assessment of residual perfusion and LV function. Its interpretation should recognize that SPECT has lower spatial resolution than CMR and may underestimate small subendocardial infarcts,

particularly when relative tracer distribution obscures globally reduced perfusion.

Positron Emission Tomography

FDG-PET interrogates myocardial metabolism and is traditionally regarded as one of the most sensitive methods for detecting viable myocardium. Ischemic but viable cardiomyocytes shift substrate utilization toward glucose, making preserved or increased ^18F-fluorodeoxyglucose uptake within a region of reduced perfusion a classic perfusion-metabolism mismatch pattern. A matched reduction in perfusion and FDG uptake is more consistent with scar. [29]

A major strength of PET is its ability to identify viable tissue despite severe contractile dysfunction and markedly reduced perfusion. Quantitative PET can also evaluate absolute myocardial blood flow and coronary flow reserve. Limitations include availability, radiation exposure, expense, the need for careful metabolic preparation, and variable FDG uptake in patients with insulin resistance or diabetes. [30]

The PARR-2 trial assessed FDG-PET-assisted management in patients with severe LV dysfunction being considered for revascularization. The overall primary endpoint was not significantly reduced by PET-guided management, although subsequent analyses highlighted the importance of adherence to imaging recommendations and experienced centers. The trial illustrates a broader principle: diagnostic accuracy does not automatically translate into improved outcomes unless imaging information changes management effectively. [31]

Cardiovascular Magnetic Resonance

CMR provides a comprehensive assessment of anatomy, LV and RV function, edema, perfusion, infarct size, microvascular obstruction, intramyocardial hemorrhage and replacement fibrosis. Late gadolinium enhancement is particularly powerful because gadolinium accumulates in areas with expanded extracellular volume after myocyte necrosis and scar formation. Ischemic injury typically begins subendocardially and extends toward the epicardium within a coronary distribution. [32]

The landmark study by Kim and colleagues demonstrated a strong relationship between transmural extent of LGE and likelihood of improvement in regional contraction after revascularization. Segments without enhancement had a high probability of recovery, while segments with extensive transmural scar had a low probability. Intermediate segments occupy a biologically mixed zone in which contractile reserve, ischemia and completeness of revascularization become important. [33]

This evidence has led to the common practical interpretation that <25% transmural scar favors recovery and >50% makes substantial recovery less likely, although these values should not be treated as rigid biological thresholds. Recovery occurs on a continuum and depends on the number of surviving myocytes, duration of dysfunction, loading conditions, revascularization quality and adjacent segment mechanics. [34]

CMR also characterizes microvascular obstruction as a hypoenhanced core within the infarct zone and can detect intramyocardial hemorrhage using T2* or susceptibility-sensitive methods. These markers identify severe reperfusion injury and are associated with adverse remodeling and worse prognosis. Thus, CMR extends the clinical question beyond “is the myocardium alive?” to “how much scar exists, how severe was the reperfusion injury, and what is the likelihood of subsequent remodeling?” [35]

Practical limitations include access, scan duration, claustrophobia, difficulty in some unstable patients or those with incompatible devices, and issues surrounding gadolinium administration in advanced renal impairment. Yet where available, CMR provides perhaps the most complete single examination for post-MI tissue characterization.

Myocardial Contrast Echocardiography

Myocardial contrast echocardiography uses intravenously administered microbubbles that remain within the vascular compartment, permitting visualization of myocardial capillary perfusion. Because microvascular integrity is necessary for sustained myocyte viability, contrast replenishment can differentiate normally perfused myocardium from regions with severe microvascular destruction. [36]

After MI, MCE may demonstrate absent tissue perfusion despite restoration of epicardial flow, providing direct visualization of the no-reflow phenomenon. Older mechanistic studies showed that preserved microvascular perfusion after recent MI was associated with myocellular viability and later

functional recovery. MCE therefore occupies an attractive physiological position between angiographic blush assessment and more elaborate CMR or PET tissue characterization. [37]

Its clinical use remains less widespread than standard echocardiography, SPECT or CMR because image acquisition and interpretation require dedicated expertise and because strong comparative outcome data are more limited. A recent systematic review from the literature supplied by the user also supports long-term prognostic value of MCE-defined viability after AMI.

Comparing the Major Viability Modalities

No viability technique measures exactly the same biological target. DSE measures contractile reserve; SPECT reflects perfusion and cellular tracer retention; FDG-PET measures glucose metabolism; LGE-CMR measures the distribution and transmurality of irreversible injury; and MCE evaluates capillary perfusion. Apparent disagreements between tests may therefore represent complementary biological information rather than diagnostic error.

Nuclear techniques generally achieve high sensitivity because preserved membrane function or metabolism may persist even when contractile reserve is limited. DSE tends to have higher specificity for subsequent mechanical recovery because it requires surviving myocardium to demonstrate an actual inotropic response. CMR offers high spatial resolution and direct scar quantification, making it particularly valuable in mixed viable-scarred territories. [38]

Selection should consequently be driven by the question being asked. If the principal concern is scar burden and transmurality after MI, CMR is highly informative. If CMR is unavailable or contraindicated, SPECT provides widely accessible perfusion-based assessment. DSE is useful when contractile reserve and inducible ischemia are central questions, while PET is valuable when severe LV dysfunction and metabolic viability require high sensitivity.

Viability, Functional Recovery, and Prognosis Are Not Identical Endpoints

Early viability research often treated improvement in regional contraction after revascularization as the decisive endpoint. This remains clinically relevant, but recovery of EF is not synonymous with improved survival. Revascularization may influence sudden ischemic events, recurrent infarction and electrical instability without producing large changes in EF, while viable myocardium may fail to recover because revascularization is incomplete, grafts fail, ischemia persists or structural dedifferentiation has become advanced. [39]

The influential meta-analysis by Allman and colleagues pooled observational studies and found a strong association between revascularization and improved survival among patients with viable myocardium and severe LV dysfunction. These findings supported viability-guided revascularization for many years. However, the constituent studies were nonrandomized and vulnerable to referral, treatment-selection and era effects. [40]

Randomized trials subsequently complicated this interpretation. In the STICH viability substudy, the presence of viability was associated with better unadjusted survival but did not identify a statistically significant interaction between viability status and the survival effect of CABG. Long-term analyses similarly failed to demonstrate that viability testing selected the patients deriving the survival benefit from surgery. [41]

The main STICH trial and extended STICHES follow-up nevertheless established a long-term benefit of CABG in appropriately selected patients with ischemic cardiomyopathy and severe LV dysfunction. The important distinction is that the benefit of surgical revascularization cannot be reduced to a binary viability result. Coronary anatomy, global ischemic substrate, scar burden, comorbidity and procedural risk remain integral. [42]

REVIVED-BCIS2 assessed PCI plus optimal medical therapy versus medical therapy alone in patients with severe ischemic LV dysfunction, extensive CAD and demonstrable viability. PCI did not reduce the primary composite of death or heart-failure hospitalization compared with medical therapy. This finding further challenged the assumption that the combination of severe dysfunction, viable myocardium and revascularizable CAD automatically predicts prognostic benefit from PCI. [43]

A prespecified REVIVED viability analysis subsequently showed that the amount of viable

dysfunctional myocardium did not identify patients who benefited from PCI. In contrast, increasing nonviable myocardium and CMR scar burden were associated with poorer prognosis and a lower likelihood of LV functional improvement. This shifts contemporary emphasis toward characterization of irreversible injury and overall substrate rather than simply counting viable segments. [44]

Late Opening of the Infarct-Related Artery

The "open artery hypothesis" proposed benefits of late restoration of infarct-related artery patency beyond immediate myocardial salvage, potentially through improved healing, remodeling, electrical stability and collateral support. Physiological and small mechanistic studies demonstrated that viable myocardium can persist within late infarct territories, providing a plausible biological rationale for delayed recanalization in selected patients. [45]

The Occluded Artery Trial provided a crucial counterpoint. Stable patients with a persistently occluded infarct-related artery identified 3 to 28 days after MI did not derive a reduction in the composite of death, reinfarction or severe heart failure from routine PCI compared with optimal medical therapy. Thus, anatomical occlusion in a stable asymptomatic patient after the acute phase is not by itself an indication for routine PCI.

This does not invalidate viability testing. OAT addressed routine opening of persistently occluded arteries in a selected stable population; it did not establish that all late presenters are biologically equivalent, nor did it eliminate revascularization for recurrent ischemia, heart failure, significant residual ischemia, left main or complex multivessel disease, or other accepted indications. The contemporary approach is therefore individualized rather than based on a universal late-open-artery strategy. [46]

Integrating Viability in the Late-Presenting MI Patient

A stable patient presenting after 48 hours should first be characterized clinically and anatomically. Ongoing ischemia, acute heart failure, cardiogenic shock, malignant arrhythmia or another instability represents a fundamentally different setting from an asymptomatic stable patient. Current ACS guidelines maintain urgent invasive management where instability or persistent ischemia is present, while the approach to stable late presentation depends more strongly on anatomy and the broader clinical context. [47]

In a stable patient, early angiography may identify a patent culprit artery with preserved flow, persistent total occlusion, slow flow, multivessel disease, left main disease or an alternative diagnosis such as MINOCA. Within an occluded territory, robust collaterals, preserved distal vessel caliber and favorable myocardial blush after restoration of flow may increase the probability that viable tissue remains but do not prove it. These are precisely the variables that make the angiographic component of the supplied research protocol clinically interesting.

When a revascularization decision remains uncertain, imaging should address the unresolved question rather than merely repeat anatomical information. CMR is particularly attractive when scar transmurality, infarct age, microvascular obstruction or alternative cardiomyopathy must be clarified. SPECT provides practical perfusion and viability information where nuclear imaging is established. DSE evaluates contractile reserve, and FDG-PET can be used when metabolic viability is especially relevant.

The result should then be integrated with coronary anatomy, technical feasibility of PCI or CABG, ischemic burden, LV function, symptoms, comorbidities, expected procedural risk and patient preferences. Viability is consequently one component of a multidimensional revascularization decision rather than an automatic trigger for intervention. This interpretation is consistent with the direction of contemporary randomized evidence.

Emerging Directions

Quantitative CMR mapping, automated scar segmentation, PET measurement of absolute myocardial blood flow, hybrid PET/MR, advanced strain analysis, radiomics and machine-learning models may refine prediction of functional recovery. Rather than categorizing an entire myocardial territory as viable or nonviable, emerging models can integrate scar heterogeneity, peri-infarct tissue, microvascular obstruction, perfusion, deformation and coronary anatomy. [48]

The most promising research direction for late MI may be multimodal prediction. Angiography provides information immediately at presentation, whereas delayed perfusion imaging or CMR defines the tissue endpoint. Linking TIMI flow, corrected frame count, myocardial blush, culprit patency and collateral grade with subsequent objective viability creates a clinically relevant opportunity to identify early invasive markers associated with myocardial preservation.

Research should also distinguish endpoints more carefully. Prediction of segmental contraction, global EF recovery, adverse remodeling, heart-failure hospitalization and mortality are related but not interchangeable. A marker can be an excellent predictor of regional recovery without identifying the patients who obtain a survival advantage from PCI. The distinction is essential when translating diagnostic studies into therapeutic recommendations. [49]

Conclusion

Myocardial viability after infarction is a dynamic biological spectrum shaped by ischemic duration, residual antegrade flow, spontaneous reperfusion, collateral circulation, microvascular integrity, stunning, hibernation and scar formation. Angiographic measures such as infarct-related artery patency, TIMI flow, corrected TIMI frame count, myocardial blush and collateral grade provide valuable mechanistic information, particularly in late-presenting MI, but do not directly replace tissue assessment. DSE, SPECT, PET and CMR interrogate different aspects of viability and should be selected according to the clinical question. Contemporary evidence supports viability imaging for myocardial characterization and prediction of functional recovery while discouraging the use of a binary viability result as the sole determinant of revascularization. Integration of anatomy, scar burden, ischemia, ventricular function, symptoms and procedural feasibility provides the strongest framework for individualized management.

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