Myocardial Viability Testing to Guide Coronary Revascularization

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KEYWORDS

• Viability • Hibernation • Revascularization • Chronic total occlusion

KEY POINTS

- Left ventricular dysfunction remains one of the best prognostic determinants of survival in patients with coronary artery disease and revascularization improves survival.
- Patients with myocardial viability have increased mortality if treated medically and do not undergo revascularization.
- Out of the most commonly used modalities to assess viability, CMR and PET offer the highest sensitivity and specificity.
- Patients undergoing CMR-guided CTO intervention have been shown to have improvement in LV ejection fraction, myocardial perfusion reserve, and symptoms.

INTRODUCTION

Left ventricular (LV) dysfunction remains one of the best prognostic determinants of survival in patients with coronary artery disease (CAD).[1,2] It was originally thought that dysfunctional myocardium after an infarction was irreversibly damaged.[3] However, it was later recognized that some of the involved tissue remained viable and contractility may be restored with revascularization.[4,5] Given that worsening LV systolic function secondary to ischemia has been shown to be associated with worse outcomes, but not all myocardium improves with revascularization, viability testing has since been well studied and used. This article reviews the pathophysiology and mechanism of myocardial viability, the most commonly used noninvasive modalities to assess myocardial viability and their strengths and weaknesses, the utility of viability testing for chronic total occlusion (CTO) interventions, and the STICH trial.[6]

PATHOPHYSIOLOGY AND MECHANISM OF MYOCARDIAL VIABILITY

After a myocardial infarction, the myocardium usually demonstrates one of five pathophysiologies: (1) normal myocardial perfusion and function, (2) myocardial ischemia, (3) stunned myocardium, (4) myocardial hibernation, and (5) nonviable infarction.[3] Prompt reperfusion or the presence of collateral vessels and intact coronary microvasculature function may preserve myocardial perfusion. Ischemia occurs as a result of decreased blood flow resulting in low ATP production and subsequent LV dysfunction.[3]

Myocardial Stunning

Myocardial stunning is a reversible state of regional contractile dysfunction that occurs after transient ischemia without ensuing necrosis.[7] Myocardial stunning is believed to play an important role in persistent contractile dysfunction seen in patients with acute myocardial

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infarction after successful reperfusion.[8] In general, myocardial perfusion is normal and function recovers quickly.

Myocardial Hibernation

More than 40 years ago physicians noticed that chronic myocardial dysfunction before coronary bypass often improved after revascularization.[4,5] Myocardial hibernation is a state of persistent LV dysfunction that results from chronically reduced blood flow or repetitive stunning without infarction and necrosis. A downregulation in contractile function at rest is thought to represent a protective mechanism to reduce myocardial oxygen requirements and ensure myocyte survival. When severe cellular hypoperfusion and damage occurs, only cellular function that is essential for survival, such as membrane integrity, is preserved. Preserved or increased myocardial glucose metabolism also occurs during this state.

Nonviable Myocardium

If myocardial perfusion is not restored, irreversible myocardial necrosis can occur. The goal of viability testing, detailed in the next section, is to determine if a large portion of dysfunctional myocardium is nonviable in which case the risks would likely outweigh benefit of revascularization.

VIABILITY AND NONINVASIVE IMAGING METHODS OF ASSESSMENT

Viability testing can predict improvement of heart failure symptoms and exercise capacity after revascularization.[9,10] The ability to distinguish viable from nonviable myocardium that is able to recover contractile function following revascularization presents a clinical challenge in current practice.[11] Furthermore, viability testing can have a lower specificity because not all patients with viable myocardium improve function after revascularization.[12]

Medical therapy with revascularization in patients with ischemic cardiomyopathy has been shown to decrease mortality compared with medical therapy alone.[13] The probability of reversing LV remodeling and improving LV systolic function with medical therapy and/or revascularization has been shown to be greater with increased proportions of viable myocardium on noninvasive imaging.[14,15] As shown in Fig. 1, Allman and colleagues[16] demonstrated in a meta-analysis of mostly observational studies that patients with viability treated by revascularization had a near 80% reduction in mortality. Those without viability had no difference in mortality between medical therapy or revascularization. We will next review each of the currently most commonly used modalities for viability testing.

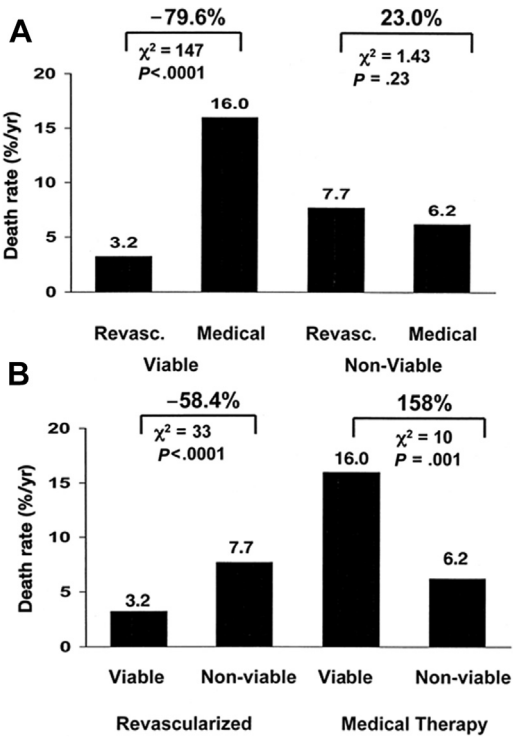

Fig. 1. (*A*) Death rates for patients with and without myocardial viability treated by revascularization or medical therapy. (*B*) Same data as A with comparisons based on treatment strategy in patients with and without viability. (*From* Allman KC, Shaw LJ, Hachamovitch R, et al. Myocardial viability 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):1155; with permission.)

Single-Photon Emission Computed Tomography

Single-photon emission computed tomography (SPECT) uses radionuclide-labeled tracer to measure regional tracer concentration in the myocardium and can measure viability by determining percentage of peak uptake of the tracer. This is interpreted with rest images only or with a stress/rest testing protocol. The most commonly used tracers are ^{99m}Tc-sestamibi or ^{201}Tl. The two tracers have been shown to have comparable results in predicting recovery of resting defects.[17] Radiotracers sequester within myocytes with intact cell membrane. Thus, myocardial viability is interpreted as an all-or-none phenomenon because SPECT cannot assess the