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THE PRESENT AND FUTURE

STATE-OF-THE-ART REVIEW

Revascularization in Patients With Severe Left Ventricular Dysfunction

Is the Assessment of Viability Still Viable?

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CME Objective for This Article: After reading this article, the reader should be able to: 1) explain the physiologic basis of myocardial viability including the spectrum of its manifestation, including myocardial stunning and hibernation; 2) discuss the current noninvasive strategies to assess myocardial viability including their advantages, disadvantages, and limitations; and 3) discuss the current body of knowledge regarding the role of noninvasive viability testing in the setting of ischemic heart disease with associated left ventricular dysfunction, including the limitations of applicability and generalizability of these studies to the wider patient population.

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Is the Assessment of Viability Still Viable?

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ABSTRACT

Myocardial viability assessment is typically reserved for patients with coronary artery disease and significant left ventricular dysfunction. In this setting, there is myocardial adaptation to an altered physiological state that is potentially reversible. Imaging can characterize different parameters of cardiac function; however, despite previously published appraisals of different imaging modalities, there is still uncertainty regarding the role of these tests in clinical practice. The purpose of this review is to reflect on the physiological basis of myocardial viability, discuss the imaging tests available that characterize myocardial viability, and summarize the current published reports on the use of these tests in clinical practice. (J Am Coll Cardiol 2016;67:2874–87) © 2016 by the American College of Cardiology Foundation.

Myocardial injury occurs through varied mechanisms, most commonly in the setting of coronary artery disease (CAD). Cardiac dysfunction consequently leads to a cascade of molecular events to maintain physiological equilibrium. Clinical heart failure may ensue secondary to pump dysfunction and arrhythmias, causing significant morbidity and mortality. Therapy is multifaceted, including revascularization paralleled with ancillary pharmacological and nonpharmacological strategies. The pharmacological armamentarium focuses on altering the neurohormonal response; the nonpharmacological approaches, primarily cardiac resynchronization therapy, focus on improving electrical synchrony. The goal is to optimize cardiovascular function, prevent progressive remodeling, allay symptoms of heart failure, and improve survival.

A challenging clinical scenario remains: the patient with severe cardiac dysfunction receiving optimal medical therapy (OMT) and carrying significant surgical risk. Can we improve symptoms? Can we improve survival? Is there a role for viability testing? Is there a standalone test, or should different modalities be used complementarily?

This review aims to elaborate on these issues that are emerging in contemporary practice. We will review the pathophysiological events in ischemic cardiac dysfunction, noninvasive strategies to image viable myocardium, and the current published reports regarding patient outcomes with decisions aided by viability testing.

THE PIVOTAL IMPORTANCE OF VENTRICULAR DYSFUNCTION AND PROGNOSIS

It has been known for close to half a century that left ventricular (LV) function is of prognostic significance in cardiovascular disease. In a 10-year follow up of survival in CASS (Coronary Artery Surgery Study), admittedly in an era when advances in medical therapeutics were only in their infancy, the presence of LV dysfunction represented an important discriminant for benefit from bypass graft surgery (1). A striking finding in the same study was the relatively low 10-year mortality of patients who had a normal ejection fraction (EF), irrespective of the number of diseased coronary vessels (1). The importance of ventricular function has been borne out in multiple investigations spanning several decades; the purported mechanisms of benefit post-revascularization being manifold, extending beyond the treatment of ischemia alone (**Figure 1**) (1–10).

After injury, the heart undergoes adaptive changes with alterations in geometry and function. The process of remodeling is dictated by hemodynamic and neurohormonal factors (11), which trigger a cascade of events that alter the interaction of myocytes with each other and with the extracellular matrix. Macroscopically this manifests in changes in size, shape, and wall thickness of the heart. Although initially adaptive, if unchecked, the continued stimulus for these changes leads to deleterious effects in the setting of pre-existing CAD, including progression to

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