Chapter 9: Cardiac PET Myocardial Viability Assessment: Emerging Clinical Data

INTRODUCTION

The utility of myocardial viability imaging hinges on the premise that recovery of left ventricular (LV) function in ischemic heart disease can be achieved through coronary revascularization and results in improved outcomes and quality of life. Rahimtoola first characterized that chronic impaired coronary perfusion resulted in impaired contractility, which he termed hibernation.[1] He suggested that a heart with reduced LVEF from hibernation could be improved by revascularization. The challenge since that time has been for viability imaging to identify those patients whose hearts will recover function and who will subsequently experience improvement in quality of life. It is a challenge that remains valid and alive 30 years after those early descriptions.

Emerging data from ischemic heart failure statistics have highlighted the plight of these patients. Mortality rates remain disconcerting high; all-cause mortality at 10 years is approximately 70% of medically managed and 60% of surgically treated individuals in the younger population and even higher in older patients.[2] Mortality can also be high early in the course of the condition, depending on the treatment chosen. Mortality at surgical revascularization can be as high as 8% within 30 days even with modern operative techniques.[2] The need, therefore, remains to accurately, thoroughly, and thoughtfully evaluate patients with ischemic heart failure to optimize management decisions.

In this chapter, cardiac PET viability assessment is described and five areas of emerging data are covered:

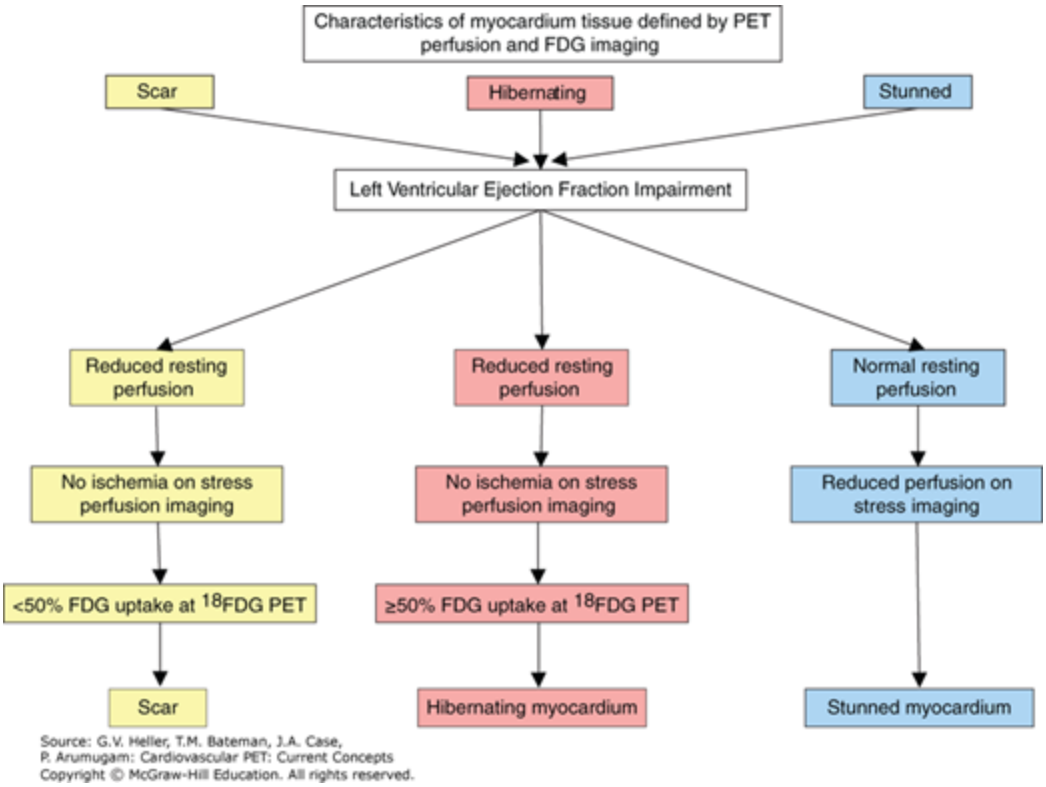
1. The STICH trial and STICH viability substudy
2. PARR-2 and Ottawa Five: has time altered the figures?
3. Quality of life outcomes: cardiac PET viability assessment can help
4. Combination of PET and MRI to refine and redefine viability definition

MECHANISMS OF PET VIABILITY IMAGING

Two types of dysfunctional but viable myocardial segments are described: stunned and hibernating. Stunned myocardium is the result of short episodes of ischemic insults that cause a temporarily depressed contractility in the corresponding area with no infarction. This phenomenon is generally associated with normal resting coronary blood flow. In contrast, hibernating myocardium reflects reduced resting perfusion and down regulation of coronary blood flow that is thought to be the result of repetitive ischemia with retained glucose metabolic activity.[3] The net result in terms of a reduction in LV contractility is observed with both stunning and hibernation. Stunning may recover spontaneously, while the persistent dysfunction of hibernating myocardium has the potential for LV functional recovery following adequate revascularization. These pathways are described in Figure 9-1.

FIGURE 9-1

Characterization of Dysfunctional Myocardium in Ischemic Heart Failure. A schematic representation of the different types of tissue present in dysfunctional myocardium: scar tissue, hibernating or stunned myocardium.



Scarred myocardium from previous myocardial infarction or chronic ischemia is to be distinguished from viable (stunned or hibernating) areas. Some myocardial segments may contain both scar and viable proportions, typically ...

Chapter 12: Current Status of Assessment of Cardiac Sarcoid and Cardiac Amyloid with PET/CT Imaging

CARDIAC SARCOIDOSIS

Clinical Need: Current Assessment and Supportive Data

Sarcoidosis is a multisystem inflammatory disease of unknown etiology that affects young men and women, typically before the age of 50 years.[1] The incidence of sarcoidosis varies based on a variety of factors including race, gender, and geography with incidence ranging from 0.73 per 100,000 in Japanese males,[2] 10 per 100,000 age- and gender-adjusted in a predominantly white cohort in the United States[1] and 71 per 100,000 in African American females.[3]

Cardiac sarcoidosis (CS) has been reported to involve approximately 2%-5% of patients with systemic sarcoidosis.[1] It can also be the only manifestation of sarcoidosis. The clinical

presentation is nonspecific and includes sudden cardiac death, ventricular arrhythmias, progressive heart failure, and conduction disturbances. Despite its low reported prevalence, CS accounts for 13%-25% of sarcoidosis-related deaths.[4]

Current diagnostic criteria for CS include the Japanese Ministry of Health and Welfare (JMWH) criteria[5] and the Heart Rhythm Society Consensus (HRS).[6] The gold standard for CS is endomyocardial biopsy, which has a low diagnostic yield owing to heterogeneous and patchy involvement of the myocardium. 2-Deoxy-2-[F-18]Fluoro-D-glucose (^{18}F-FDG) PET-CT is the best advanced imaging tool to evaluate myocardial inflammation in patients with suspected CS. It has a reported sensitivity of up to 85%-100% with more variable specificity of 39%-100%.[7–10] The low specificity is likely due in part to using the JMHW criteria as a gold standard and the difficulty of a definitive diagnosis.

In the HRS guidelines,[6] clinical diagnosis of CS is probable if there is histological proof of extra CS and one or more advanced imaging findings of CS or unexplained decreased systolic function or arrhythmias when other causes have been reasonably excluded