predicted better outcomes overall and improvement in EF, although such EF improvement did not translate into better long-term clinical outcomes.[141] Therefore, the role of viability imaging in clinical practice remains unclear.

Myocardial Viability Evaluation by Nuclear Imaging. PET myocardial FDG metabolic imaging combined with PET or SPECT perfusion imaging is the current nuclear imaging approach of choice for the evaluation of myocardial viability.[136] In the presence of repetitive ischemia and after a glucose load, viable myocardium shows an increased uptake of FDG, reflecting preferential utilization of glucose over free fatty acids. The typical viability study consists of FDG PET images paired with resting myocardial perfusion images. In regions with resting hypoperfusion a concordant reduction in both flow and metabolism ('match') represents myocardial scar while an increase in FDG uptake compared with flow ('mismatch') represents hibernating but viable myocardium.[142] PET FDG has been reported to have a high negative predictive value (mean 90%, confidence interval 86–95%) and a good positive predictive value (mean 73%, confidence interval 66–80%) for recovery of segmental contractile function after revascularization.[137] An ongoing randomized clinical trial will further test the hypothesis that ^{18}F FDG viability imaging improves patient outcomes (IMAGE-HF Project I-A).[143]

Myocardial Viability Evaluation by Dobutamine Echocardiography. The stress echocardiographic sign of myocardial viability is a stress-induced improvement of function during low levels of stress in a region that is abnormal at rest. Most of the experience is available with low-dose dobutamine stress echocardiography, the preferred stressor for assessing myocardial viability,[5,12,19] although viability may also be detected by dipyridamole stress echocardiography or during low-level bicycle exercise. In patients with CAD, proof of viability is an improvement of myocardial function, which may occur spontaneously (e.g. after stunning), on medical therapy, or after revascularization (Supplementary data online, Movie S1).

In patients with dysfunctional but viable myocardium, regional function can be improved by low-dose (5–10 μg/kg/min) dobutamine, typically worsening at higher doses (biphasic response). Dobutamine stress echocardiography has similar overall accuracy as nuclear imaging techniques.[5,23]

Myocardial Viability and Scar Evaluation by Cardiac Magnetic Resonance. Similar to echocardiography, cine CMR imaging can be performed at rest and during a low-dose infusion of dobutamine to determine viability based on improvement of wall motion.[144] This technique does not require gadolinium contrast. However, the vast majority of viability imaging by CMR utilizes the LGE method. Rather than detecting viable tissue, LGE CMR detects scar (non-viable myocardium) (Figure 15). With LGE images acquired approximately 10–12 min after the administration of gadolinium, myocardium that is infarcted, fibrotic or scarred appears bright, while viable myocardium is conventionally set to appear dark. Given its high spatial resolution, the size, location, and transmural extent of myocardial infarction by LGE CMR closely matches histopathology.[145] Myocardial segments with more than 50% transmural infarction have a low likelihood of functional recovery, while segments exhibiting less than 50% transmural enhancement are more likely to recover contractile function.[41] The presence and extent of scar detected by LGE is a powerful predictor of prognosis, independent of LVEF.[146,147]

Key Points

1. Non-invasive imaging to detect ischemia and viability is reasonable in patients presenting with heart failure who have known CAD and no angina, unless the patient is not eligible for revascularization, although its value is unclear.
2. Nuclear imaging, low-dose dobutamine echocardiography, or CMR and LGE CMR are all options to evaluate viability.
3. LGE CMR is the method of choice for scar detection.

Risk Stratification in Chronic Coronary Syndromes

All non-invasive imaging methods have demonstrated important prognostic relevance in CCS, and the choice of imaging method should be based on intended clinical assessment and the best local choice to diagnose CAD.[3]

A resting TTE assessment of LV function is important for risk stratification in CCS. Assessment of GLS will add incremental information about risk, particularly in patients with EF >35%.[37,38,148]

A resting echocardiogram should usually be followed by anatomical or functional tests for diagnostic purposes. All non-invasive imaging methods have demonstrated important prognostic relevance in chronic CAD. A normal stress echocardiogram, CMR, or myocardial perfusion scan implies excellent prognosis and coronary angiography can safely be avoided in patients with suspected CAD.[149] Results of the stress tests can be further stratified by evaluating clinical parameters (diabetes, renal dysfunction, and therapy at the time of test), resting echocardiography (global LV function), and stress echocardiography parameters (LV cavity dilatation, coronary flow reserve, and previous revascularization). The ischemic response can be further stratified with additive stress echocardiographic parameters, such as the extent of inducible WMA and the maximum workload/dose achieved. Survival rate correlates directly with ischemia-free stress time and inversely with wall motion score index. The incidence of death in patients with a negative functional test off therapy is very low. At intermediate risk are those patients with a negative test on medical therapy or a positive test off medical therapy. The findings of a negative functional test cannot, however, rule out lower degrees of coronary atherosclerosis, and patients should always receive advice and treatment according to current risk charts and recommendations.

The presence of scar by SPECT and CMR is associated with poor prognosis, and absolute quantification of MBF with PET and CMR adds prognostic information.[150]

Assessment of coronary atherosclerotic plaque burden provides powerful prediction of myocardial infarction. Numerous large-scale studies have demonstrated the long-term prognostic value of CAC testing among diverse populations, even additive to standard risk scores.[1] The absence of CAC is associated with a very good prognosis, with 1.0–1.5% risk of atherosclerotic cardiovascular disease outcomes over 10 years of follow-up, while those with advanced CAC (>300 Agatston score) have at least a six-fold increased relative risk for long-term events.[151,152]

The use of CAC scoring in symptomatic patients is more controversial. Non-calcified plaque and obstructive CAD uncommonly occur in the absence of CAC, especially in patients at higher pretest CAD risk.[153,154] Therefore, the absence of CAC has been shown to have very high negative predictive value to exclude obstructive CAD in observational and prospective studies and has been suggested as a possible gatekeeper test in low-risk patients.[155,156]