One of the strengths of cardiac magnetic resonance (CMR) is its versatility and ability to answer important pathophysiological questions as novel pulse sequences are developed. This issue of *iJACC* is an excellent example of this phenomenon. Three papers in this issue (1–3) propose techniques that are dependent on intrinsic myocardial signal characteristics or flow properties rather than exogenous contrast to generate images and demonstrate pathophysiology in acute myocardial infarction (MI) and myocardial ischemia.

Kumar and colleagues present 2 papers (2,3). The first is a study of imaging myocardial edema as a marker of myocardial area-at-risk in acute MI (3). Although there is some controversy in regards to the utility of T2-weighted imaging as a marker of myocardial edema/area-at-risk as discussed in a recent *iForum* in this journal (4), this paper aims to validate a new approach to imaging edema using steady-state free precession (SSFP) cine imaging. The study was performed both in a mini-pig model of acute MI and patients with acute MI. SSFP is a technique that combines T1- and T2-weighting to optimize contrast-to-noise between the dark myocardium and the bright blood pool on cine imaging. This is an older magnetic resonance imaging technique, rediscovered for CMR around the turn of the millennium when scanners became fast enough to support the very short echo times required. T2-short T1 inversion recovery (STIR), a standard way of performing T2-weighted imaging, was used as the CMR technique of comparison and late gadolinium enhanced inversion recovery images as the gold standard for necrosis. SSFP correlated well with T2-STIR for demonstration of edema. In almost all patients with acute MI, SSFP showed edema. The presence of a dark zone of microvascular obstruction in a subset of patients at the subendocardial core of the infarct reduced the signal in the SSFP images within the MI, but did not harm sensitivity of the technique for edema. This suggests that SSFP could eventually replace T2-W imaging as a marker of edema. T2-weighted imaging is one of the more difficult CMR techniques as it is associated with artifacts and signal inhomogeneity. Replacement with SSFP could save a significant amount of imaging time, rendering the study more time-efficient and cost-effective.

The second study by Kumar (2) validates the use of T2* CMR imaging as a marker for reperfusion-related intramyocardial hemorrhage. Investigators have recognized hemorrhage on post-contrast imaging for a number of years, but T2* CMR is a noncontrast method for visualizing it. This pulse sequence is one that is typically used for measuring iron in the heart in the setting of iron overload from hemochromatosis or multiple transfusions due to thalassemia. They studied a small group of dogs after MI and validated their findings against histopathology; this paper has also showed the feasibility in a solitary patient after acute MI.

Combining the techniques from these 2 papers, cine SSFP for imaging edema and T2* CMR for hemorrhage, expands the CMR imager’s armamentarium for imaging acute MI. One technique speeds up the imaging session and the other identifies hemorrhage in the relatively unique group of patients who manifest this pathophysiology. With the ability to image LV volumes, function, edema, hemorrhage, and scar, CMR can comprehensively characterize the myocardial state in an MI patient.
Further studies are needed to define how these findings relate to outcome and to changes in therapy for individual patients.

The third paper in this issue of *JACC* applies a noncontrast method for measuring myocardial blood flow, arterial spin labeling (ASL), to vasodilator stress testing in patients with suspected coronary artery disease (1). ASL is a technique in which water in arterial blood is given a different longitudinal magnetization from the surrounding tissue and thus acts as a tracer wherein inflowing blood is tagged and the rate at which it enters the imaging plane is measured. Quantification is performed by subtracting control from tagged images, which leaves it susceptible to degradation by motion. In addition, the technique tends to be signal-starved. It has been used primarily for measuring cerebral perfusion. Despite the technical limitations, Zun et al. (1) demonstrate proof-of-principle in their paper, as they were able to measure differences in absolute blood flow between vasodilated and rest states. In addition, they demonstrated difference in perfusion reserve in patients between normal and diseased segments of coronary arteries. A major limitation of this study is that only 1 short-axis slice was imaged due to time limitations with the technique in its present state. Clearly, improvement will be required for this technique to achieve clinical utility. It certainly offers promise, although it is unclear as to whether it would ever replace contrast-enhanced myocardial perfusion measurement which is rather robust (5). Certainly, patients with stage IV/V chronic kidney disease, who are not candidates for gadolinium contrast, would be excellent candidates for a noncontrast technique; and it would also be ideal for studies requiring multiple perfusion measurements over time as may be necessary in clinical trials.

Taking these 3 interesting papers together, CMR continues to progress rapidly with applications of novel pulse sequences. Using intrinsic contrast of both tissue and blood flow is a major advantage of CMR and precludes the need to use contrast for these particular applications. Bringing new techniques to light is a major goal of our Journal and we are pleased to bring these papers to our readers.

**REFERENCES**


