The blood flow resistance of the coronary circulation in patients without epicardial coronary disease is governed principally by arteriolar vessels less than 300 microns in diameter. Although the majority of blood flow to the myocardium occurs in diastole, the normal coronary vascular bed is able to achieve increases in myocardial flow up to 3-5 fold higher than that seen at rest. In the presence of coronary stenosis, the microvasculature dilates to compensate for the up-stream flow limitation. When the up-stream stenosis approaches 80% of the lumen, the microvasculature is completely dilated at rest; no additional flow augmentation can be achieved. At conditions of increased myocardial oxygen demand or through the administration of vasodilator drugs, myocardium downstream from such a significant stenosis will become ischemic, visualized as a reversible perfusion defect limited to a coronary artery territory. Subendocardial perfusion defects not limited to a coronary territory can be seen with pharmacologic stress in patients with microvascular dysfunction (Syndrome X). The abnormality in these subjects relates to limitations in flow augmentation at the microvascular level, which is more evident in the subendocardial tissue.

Multiple modalities are able to perform myocardial perfusion imaging, including single photon emission computed tomography, positron emission tomography, and cardiac magnetic resonance imaging (CMR). CMR has several advantages over competing modalities, the principle advantage being spatial resolution enabling detection of subendocardial perfusion defects. Trade-offs in image quality to enable acquisition of multiple slices and identify subendocardial perfusion defects requires vigilance to detect artifacts masquerading as perfusion abnormalities. Dark-band rim-like artifacts are principally caused by a finite number of phase encoding steps at the boundary of the bright contrast-enhanced blood pool and the relatively hypointense subendocardial myocardium. Similar-appearing artifacts are caused by magnetic susceptibility differences between these tissues. Artifacts on perfusion at stress are mitigated by comparison with first pass imaging at rest and delayed enhancement imaging. Fixed perfusion abnormalities with corresponding delayed enhancement represent infarctions, while fixed defects without corresponding abnormalities on delayed enhancement are artifactual.

In an ideal situation, CMR perfusion would enable quantification of myocardial perfusion by AHA segments. By segment, perfusion could be divided into subepicardial and subendocardial components. Comparison between the stress and rest acquisitions would identify the myocardial perfusion reserve, generating quantifiable measures of myocardial blood flow augmentation to pharmacologic vasodilators. Practically, absolute quantification with first pass myocardial perfusion is not achievable as signal from the transit of gadolinium chelates through the muscle is influenced among other factors by leakage of contrast from the capillary bed into the surrounding interstitial space, leading to an increased discrepancy between myocardial enhancement at first pass imaging and absolute...
perfusion. However, semiquantitative indices can be utilized in lieu of absolute perfusion metrics. Multiple semiquantitative indices have been generated from myocardial signal intensity time curves including peak signal intensity, time to peak intensity, mean up-slope, and mean transit time. Myocardial perfusion reserve calculated through the use of the mean up-slope semiquantitative index has shown the most promise as a surrogate for absolute perfusion, when the indices are normalized to the arterial input function taken from the left ventricular blood pool.

Multiple acceleration strategies have been applied to enable acquisition of short axis slices covering the entire ventricle at first pass perfusion. Ideally, acceleration would permit whole heart coverage acquiring all perfusion slices in a single heartbeat, limiting data acquisition to the quiescent period of diastole. The majority of users perform myocardial perfusion imaging acquiring between 3 and 4 slices per heartbeat based on the heart rate, trading quantity for quality. The improved spatial resolution results in fewer artifacts at first pass perfusion imaging; as the artifacts can mimic subendocardial perfusion defects, this approach likely improves perfusion performance.

Impaired perfusion CMR image quality due to beat-to-beat variability can be addressed through the use of motion correction algorithms. In general, patients undergoing pharmacologic vasodilator stress experience mild heart-rate augmentation. Slight beat-to-beat variability persists, generating differences in ventricular filling between acquired perfusion images at each time point. Motion-correction algorithms have been developed to correct for these differences, removing the appearance of macroscopic motion from the perfusion images. Semiquantitative perfusion maps are more easily generated from motion corrected images, facilitating use in the clinical work-flow.

Perfusion imaging is a useful adjunct to viability imaging at CMR. Coupled with ventricular functional analysis, tissue characterization, evaluation of myocardial relaxation, and viability assessment, perfusion imaging enables a comprehensive myocardial evaluation at CMR imaging.