Assessment of Subendocardial Function With Myocardial Contrast Echocardiography

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The classical ischemic cascade anticipates perfusion disturbances to precede the development of wall motion abnormalities, but does not account for regional heterogeneity of both perfusion and function. In this issue of *JACC: Cardiovascular Imaging*, Xie et al. (1) examine the impact of subendocardial ischemia (identified using real-time myocardial contrast echocardiography [MCE]) on myocardial thickening during dobutamine stress echocardiography (DSE). The subendocardium is the crucible of myocardial dysfunction in many diseases. This part of the myocardium is furthest from the epicardial coronary vessels, compressed by the intracavitary pressure as well as the remainder of the myocardium, and characterized by different myocardial fiber orientation (and therefore contraction vector) from the rest of the myocardium. The subendocardium is not only involved in ischemic heart disease, but also in hypertensive heart disease (2) and perhaps in small vessel disease (3,4). However, the ability to characterize subendocardial function and perfusion has—until recently—been limited.

Myocardial contrast echocardiography is 1 of 2 new imaging techniques that may address the ability to examine subendocardial perfusion in clinical practice. Previous experimental studies of MCE during dipyridamole stress have shown a transmural gradient of flow when hyperemic flow was reduced by 75%, despite preservation of wall thickening (5). This discrepancy between subendocardial malperfusion and preserved function occurred over a small window—both subendocardial and subepicardial flow were preserved at 50% reduction of hyperemic flow, and at a 90% reduction of hyperemic flow, both were reduced. The work of Xie et al. (1) represents the clinical analog of these findings. The investigators studied 94 patients with normal resting function, 55 of whom had a significant left anterior descending coronary artery stenosis. Wall thickening was visually assessed after contrast destruction and during myocardial contrast replenishment. End-systolic and -diastolic images of the subendocardial defect and entire wall thickness were chosen to measure subendocardial and transmural wall thickening, respectively. The majority of defects were subendocardial, and most of these appeared to have normal transmural wall thickening, despite reduced subendocardial wall thickening. Although quantitative transmural wall thickening failed to augment relative to baseline in those with abnormal subendocardial perfusion, the investigators showed a trend in wall thickening between those with normal through reduced subendocardial wall thickening and those with transmural abnormalities of wall thickening.

Myocardial contrast echocardiography has been shown to improve the sensitivity of stress echocardiography, as well as enhance the detection of coronary stenoses in the nonculprit territory (6). Although left ventricular opacification can be justified in patients with suboptimal image quality, the conventional wisdom is that myocardial contrast is not a useful or necessary adjunct to wall motion assessment in all stress echocardiograms. Many studies are clearly normal (no significant coronary disease) or clearly abnormal (significant disease, often in multiple vessels), and these extremes are
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Diagrams is an important question that warrants caution. The extent to which subendocardial defects may account for false negative exercise echocardiograms is an important question that warrants attention. Despite its easier performance, the sensitivity of DSE is somewhat less than that of exercise echocardiography, and defect extent is often smaller (10). Relatively lower afterload and the potent inotropic stimulus may lead to reduced left ventricular cavity size, lower wall stress, and reduced sensitivity, especially among those with concentric remodeling. Therefore, DSE may represent the “worst case” scenario for this mismatch between perfusion and function, and exercise echocardiograms, with higher wall stress and more extensive defects, may have fewer instances where ischemia is limited to the subendocardium.

Although this study has important implications for interpretation of DSE, the findings also support the use of MCE in the clinical assessment of subendocardial perfusion and function. Experimental studies have addressed these entities using microspheres and microcrystals (11), but these are not clinical tools. The spatial resolution of positron emission tomography and single photon emission tomography are not well suited to this work, although such data might be obtainable with image processing strategies. Cardiac magnetic resonance imaging assessment of perfusion may be made semiquantitatively, based on the change of signal intensity over time (similar to the principles of MCE) during the hyperenhancement of hypoperfused tissue with the first pass of gadolinium, although there are noncontrast alternatives (12). Although more difficult to perform, the echocardiographic technique is easier to apply in the acute setting, more versatile in terms of the types of stress agents, and can be obtained in real time. The combination of regional perfusion with quantification of myocardial deformation in different layers and in different dimensions may enable this tool to be used to assess both transmural perfusion and functional gradients.

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