Critical Myocardial Ischemia – Visually Homogeneous but Heterogeneous Through “21st Century Echo Eyes” –

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In the presence of critical myocardial ischemia, myocardial blood flow is not altered at rest, transmural thickening of myocardium is preserved¹ and visual assessment of wall motion by conventional echocardiography often fails to identify ischemia. Therefore, pharmacological or exercise stress echo, which increases myocardial oxygen requirements beyond the limits of a stenosed vessel to supply blood flow, is widely accepted as provoking a systolic wall motion abnormality. The differing behaviors of the inner/outer halves of the left ventricular (LV) wall have been documented with sonomicrometry^{1,2} and epicardial M-mode echocardiography³ in experimental animal models since the 1980s. A gradient of thickening exists across the myocardial wall, with the inner portion of the wall contributing the largest fraction to total systolic thickening.³ At rest, the fractional contribution of the inner wall to total systolic wall thickening is approximately 70%, which is significantly greater than the outer wall.² In the presence of critical coronary stenosis, reduction of inner half contraction has been documented not only during ischemia but also during exercise¹ and increased afterload.⁴

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Post ischemic shortening (PSS) is defined as a delayed ejection motion of the myocardium occurring after aortic valve closure during isovolumic relaxation time.⁵ Many investigators have described PSS by varying nomenclature, and its relation to myocardial ischemia has been discussed.^{6–8} Doyle et al described PSS as an early abnormality of wall motion where critical coronary constriction is produced by the gradual tightening of a snare.⁹ Takayama et al investigated the significance of PSS before reperfusion and early recovery after reperfusion, and they demonstrated that the PSS of dyskinetic myocardium segments was a marker of their potential for recovery.⁷ With the advent of tissue Doppler techniques, the detection of spatial and temporal heterogeneity of myocardial systolic strain has become feasible in the clinical setting.^{8,10,11} However, tissue Doppler has an unavoidable limitation based on ultrasound angle dependency. Diagnostic criteria for the presence of coronary artery disease with tissue Doppler at rest have been proposed,⁸ but the sensitivity and specificity are still unsatisfactory. To overcome this limitation, 2D echo speckle tracking imaging (STI) has been

developed and intramural strain measured by a STI system has been compared against that measured by sonomicrometry crystals.¹²

In this issue of the Journal, Ishizu et al investigate inner/outer strain, as well as PSS by STI, to detect myocardial ischemia in patients with flow-limiting coronary stenosis but visually normal LV wall motion.¹³ They focused on 2 findings: heterogeneity of inner/outer myocardial strain, and the grade of PSS at rest. They reported that impaired subendocardial thickening and PSS were present in myocardial segments perfused by the flow-limiting critical stenosis, although transmural strain was preserved. These findings may not be surprising and may be predictable, considering past experimental reports. However, combined analysis of inner/outer strain ratio and postsystolic strain index derived by 2D STI provided high sensitivity for the presence of flow-limiting stenosis in the clinical setting. Although this report provides new insight into the assessment of myocardial ischemia, showing non-uniformity of inner and outer thickening, the results must be interpreted in the light of some limitations. First, they analyzed wall motion at rest in selected patients with severely limited coronary flow and assumed myocardial ischemia was present. They defined critical stenosis as visually preserved wall thickening with 99% with delay or 100% occlusion with poor collateral flow. However, they did not confirm myocardial ischemia with SPECT or by provocation with a stress test. Second, PSS was observed even in normal subjects. In fact, PSS was visually observed in 48% of the normal segments in their study. Voigt et al also reported that more than one-third of normal LV segments showed PSS in longitudinal strain measurements, and that pathologic PSS can be detected by coexisting reduction in systolic strain and by exceeding a postsystolic strain magnitude cutoff.¹⁴ Third, their measurements differ from sonomicrometric measurements because 2D speckle tracking remains limited by the through-plane phenomenon.

The importance of this study is that it demonstrates a high-specificity method for the presence of flow-limiting stenosis, which might exclude patients from unneeded emergency coronary intervention if the presented criteria of inner/outer strain ratio and postsystolic strain index are combined. In the percutaneous coronary intervention era, coronary angiography tends to be performed for patients who complain of chest

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pain with presumptive evidence of ischemia. Therefore, under the conditions of preservation of subendocardial wall thickening and apparent PSS being absent, the probability of critical coronary stenosis may be low. Thus, an urgent invasive procedure may not be needed.

Although this report has some limitations, the study presents a new diagnostic insight of ischemic heart disease. The clinical importance of echocardiographic assessment of myocardial ischemia will be established with future larger prospective studies.

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