Cardiac Imaging

Exercise-Induced Post-Ischemic Left Ventricular Delayed Relaxation or Diastolic Stunning

Is it a Reliable Marker in Detecting Coronary Artery Disease?

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Objectives	The aim of this study was to determine whether post-ischemic left ventricular (LV) delayed relaxation could be detected by using strain imaging (SI) derived from 2-dimensional speckle-tracking echocardiography in patients with stable effort angina.
Background	Regional LV delayed relaxation during early diastole is a sensitive sign of acute myocardial ischemia and may persist beyond recovery of exercise-induced ischemia.
Methods	Regional LV transverse strain changes during the first one-third of diastole duration (strain imaging diastolic index [SI-DI]) were determined at baseline and 5 and 10 min after the exercise test in 162 patients with stable effort angina. The ratio of SI-DI before and after exercise (SI-DI ratio) was used to identify regional LV delayed relaxation.
Results	A total of 117 patients had significant ($\geq$50% of luminal diameter) coronary stenoses. The mean SI-DI decreased from 78.0 $\pm$ 9.7% to 27.6 $\pm$ 16.0% (p < 0.0001) in 191 territories perfused by coronary arteries with significant stenoses 5 min after the treadmill exercise, whereas it remained unchanged in 280 territories perfused by arteries with nonsignificant stenoses. Ten minutes after exercise, regional delayed relaxation was still observed in 85% of territories perfused by stenotic coronary arteries. An SI-DI ratio with a cutoff value of 0.74 had a sensitivity of 97% and a specificity of 93% to detect significant coronary stenosis in the receiver-operator characteristic curve.
Conclusions	Detection of post-ischemic regional LV delayed relaxation or diastolic stunning after treadmill exercise using SI is a sensitive and reliable method for the detection of coronary artery disease. (J Am Coll Cardiol 2009;53: 698–705) © 2009 by the American College of Cardiology Foundation

Diastolic left ventricular (LV) abnormalities are sensitive early signs of myocardial ischemia and have the additional advantage of persisting longer than systolic disturbance (1–4). Ischemia-induced diastolic tardorelaxation or delay in the onset of regional relaxation has been shown in the perfusion territory of the involved coronary region in animal and clinical models (5–10). Quantitative analysis of prolonged regional LV diastolic dysfunction may provide a more sensitive estimation of the coronary region involved compared with detection of systolic dysfunction to detect differences between ischemic and nonischemic myocardium more accurately. Recently, we have shown that impaired or stunned regional diastolic function with delayed outward wall motion persisted beyond recovery after ischemia in patients with effort angina during the treadmill exercise

stress test (11). Tissue Doppler imaging and derived strain and strain rate measurements have been proposed to better quantify regional myocardial deformation. However, these techniques depend on the Doppler angle and lack reproducibility (12). Strain imaging (SI) derived from 2-dimensional (2D) speckle-tracking echocardiography enables quantification of regional myocardial function without

tethering effect and Doppler-angle dependency with high temporal resolution (13–15). The SI measures the extent of regional myocardial shortening and lengthening. Because of its high temporal and spatial resolution, SI may facilitate the application of novel quantitative parameters in ischemia detection. High-resolution tracking of regional myocardial activity by SI may provide a valuable and quantifiable means of detecting inducible ischemia with implications for stress echocardiography (16,17). The current study was designed

to determine whether post-ischemic regional LV delayed relaxation or diastolic stunning occurring after treadmill exercise stress testing could be detected by using SI in patients with stable effort angina and coronary artery disease (CAD). If so, CAD could be diagnosed and stenotic coronary arteries could be identified noninvasively and accurately by the detection of post-ischemic regional LV delayed relaxation after treadmill exercise stress testing by using SI.

Methods

Patient population. One hundred sixty-seven consecutive patients who had Canadian Cardiovascular Society Classification II (18) stable effort angina and normal LV wall motion detected by standard echocardiography were recruited into this study. Consent was obtained to receive both treadmill exercise echocardiography including SI derived from 2D speckle tracking technology and coronary angiography. Patients with unstable angina, left main trunk disease, previous myocardial infarction, previous cardiac surgery, artificial pacemaker, nonsinus rhythm, significant valvular heart disease, chronic obstructive pulmonary disease, or congestive heart failure were not included. None had any apparent abnormalities in echocardiographic parameters including the LV end-diastolic diameter, fractional shortening, LV hypertrophy (wall thickness at end diastole >11 mm), or calculated LV mass index. Five patients were excluded from the analysis because of inadequate ultrasound images. The remaining 162 patients (ages 63 ± 10 years, 60% men) were studied. The study protocol was approved by the institutional ethics committee, and all patients gave written informed consent.

Exercise stress test and SI study. All patients underwent the treadmill exercise test with SI study within 1 week of coronary angiography. The SI was analyzed by an experienced echocardiography specialist who was blinded to the coronary angiography findings. Symptom-limited treadmill exercise stress testing was performed by using the standard Bruce protocol (19). Exercise was terminated in the presence of severe chest pain or other symptoms limiting further exercise, severe arrhythmia, ≥2 mm ST-segment elevation or depression, systolic blood pressure ≥250 mm Hg, diastolic blood pressure ≥130 mm Hg, or a decrease in systolic blood pressure ≥20 mm Hg. Heart rate, blood pressure, 12-lead electrocardiography, and symptoms at rest, during exercise, and for the first 10 min after exercise were recorded. Electrocardiographic findings during or after exercise were compared with those obtained at rest. The ST-segment depression ≥0.1 mV at 80 ms after the R-wave was considered to be significant for myocardial ischemia (20). If the chest pain persisted for 2 min or was too severe to be endured by the patient, sublingual nitroglycerin was administered. For the control group, 30 subjects (18 men and 12 women with a mean age of 57 ± 8 years) without chest pain or any cardiovascular disease who had had

negative findings on treadmill exercise tests were also studied.

The SI study with the patients lying in the left lateral decubitus position was performed at baseline, and also 5 and 10 min after the treadmill exercise stress test. The SI was obtained by using a ultrasound system (Aplio SSA-770A, Toshiba Medical Systems, Tokyo, Japan) with a 2.5-MHz phased-array transducer in the 3 standard LV apical views (apical 4-chamber, 2-chamber, and long-axis) and high frame rate (45 ± 5 frames/s). A novel software program was used to measure the transverse strain of myocardial segments. Two stable and well-defined consecutive cardiac cycles were acquired digitally for each view and stored on a magneto-optical disk for off-line analysis.
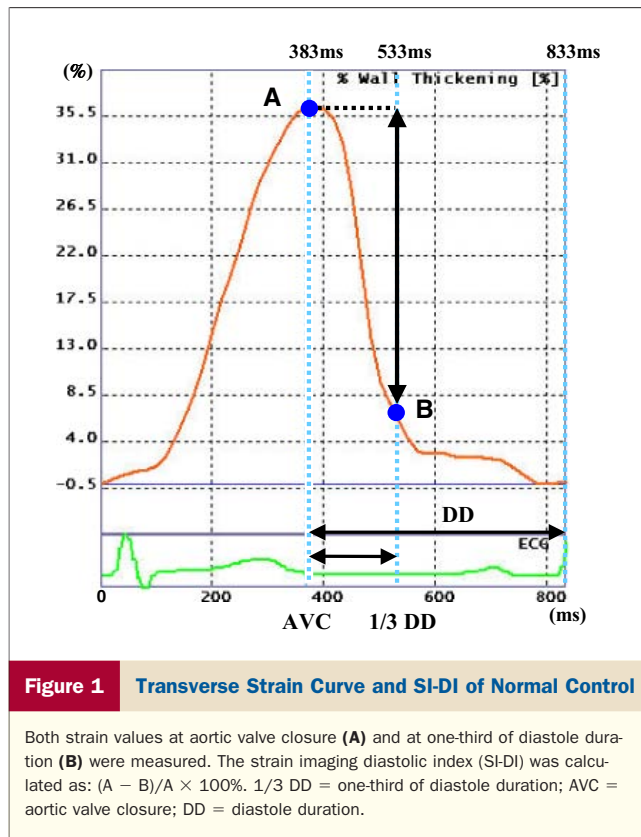
Abbreviations and Acronyms

2D = two-dimensional

CAD = coronary artery disease

LV = left ventricle

SI = strain imaging

SI-DI = strain imaging diastolic index

Analysis of SI. Transverse strain images were obtained in each segment by using 2D speckle-tracking software (Toshiba Medical Systems). End-diastole and -systole were defined to occur at the R peak on the electrocardiographic trace and aortic valve closure on the 2D-mode profile, respectively. Cardiac cycles associated with atrial and/or ventricular extrasystolic beats, post-extrasystolic cycle, or any other rhythm abnormalities were excluded. The end systolic values of strain at the closure of the aortic valve (A) and at the one-third point of diastole duration (B) were measured. The strain imaging diastolic index (SI-DI) was determined as $(A - B)/A \times 100\%$ (Fig. 1) to assess the regional LV active relaxation (21,22). The ratio of SI-DI before and after exercise was defined as the SI-DI ratio and was used to identify regional LV delayed relaxation. Three major coronary perfusion territories were assigned as defined in the American Society of Echocardiography guidelines (23): 1) the mid-anteroseptal segment to the left anterior descending coronary artery; 2) the mid-anterolateral segment to the left circumflex branch; and 3) the mid-inferior segment to the right coronary artery. The mean SI-DI at baseline in 30 control subjects was 84 ± 7%, 86 ± 9%, and 85 ± 8% in the anteroseptal, anterolateral, and inferior segments, respectively. Interobserver variability for measurement of SI-DI was 7.2 ± 2.3% (n = 30), and intraobserver variability was 4.3 ± 1.5% (n = 30).

Quantitative coronary angiography. The coronary angiograms were analyzed by using the Cardiovascular Angiography Analysis System (CAAS II, PIE Medical Imaging, Maastricht, Limburg, the Netherlands) (24). End-diastolic frames were selected for edge-detection analysis, and the tip of the catheter was used as calibration. Reference diameter, minimal lumen diameter, and diameter stenosis were calculated. Significant coronary artery stenosis was considered present when ≥50% diameter stenosis of at least 1 major coronary artery was observed.

Statistical analysis. Values are presented as the mean ± standard deviation. Comparison of the SI-DI was performed with repeated measures of analysis of variance followed by the

Figure 1 Transverse Strain Curve and SI-DI of Normal Control

Both strain values at aortic valve closure **(A)** and at one-third of diastole duration **(B)** were measured. The strain imaging diastolic index (SI-DI) was calculated as: (A − B)/A × 100%. 1/3 DD = one-third of diastole duration; AVC = aortic valve closure; DD = diastole duration.

Student paired *t* test. Values of p < 0.05 were considered statistically significant. Receiver-operator characteristics curve of the SI-DI ratio for the detection of ≥50% and ≥70% coronary stenosis were created, and the area under the curves for the ratio was created.

Results

The baseline clinical and cardiac characteristics of the 162 patients (mean age 63 ± 10 years, 60% men) with effort angina are listed in Table 1. A total of 117 patients had ≥50% coronary stenosis and 45 had normal coronary anatomy or <50% stenosis. Of the patients with significant coronary stenosis, 1-vessel disease was present in 60, 2-vessel disease in 37, and 3-vessel disease in 20 patients. There was significant left anterior descending artery stenosis in 87, circumflex artery stenosis in 53, and right coronary artery stenosis in 54 patients. Mean stenosis severity by coronary angiography was 72 ± 8.1% in the left anterior descending coronary artery, 71 ± 6.5% in the left circumflex artery, and 70 ± 7.3% in the right coronary artery.

Peak physiological response to treadmill exercise. Ninety-five patients (59%) had significant ST-segment depression during the treadmill exercise test. Sensitivity and specificity of the treadmill exercise test in the study patients were 62% and 73%, respectively. At the end point, the heart rate was 141 ± 16 beats/min, representing 89 ± 11% of the age-predicted maximum; the systolic blood pressure, 210 ± 17 mm Hg; and peak

rate-pressure products, $28.7 \pm 4.8 \times 10^3$ beats/min × mm Hg. In 12 patients, sublingual nitroglycerin was administered to suppress angina attacks persisting for more than 2 min after treadmill exercise. Both angina and electrographic ST-segment depression subsided within 5 min of the termination of the exercise in all patients. Stress-induced regional systolic wall motion abnormality (hypokinesis or akinesis) was noted in some of the segments in the 94 (80%) patients with significant coronary stenosis immediately after exercise by visual estimation. However, 5 min after the treadmill exercise test, systolic abnormality was detected in 9 (8%) patients, and 10 min afterward, no systolic abnormality was detected in any of the patients with significant coronary stenosis.

Post-ischemic LV delayed relaxation after treadmill exercise. In 162 patients for whom data were analyzed, there were 486 potentially analyzed segments. Of these, 15 segments (3%) were excluded from SI analysis because of an uninterpretable signal. The heart rate was 77 ± 11 beats/min at 5 min after the treadmill exercise and 69 ± 10 beats/min at 10 min after the exercise. The systolic blood pressure was 149 ± 13 mm Hg at 5 min after the exercise and 137 ± 15 mm Hg at 10 min after the exercise.

Figures 2 and 3 show the changes of SI-DIs in the territories perfused by coronary arteries with significant and nonsignificant stenosis after treadmill exercise test.

Table 1 Baseline Clinical Characteristics

No. of patients	162
Age (yrs)	63 ± 10
Male/female	97/65
BSA (m²)	1.6 ± 0.2
HR (beats/min)	67 ± 9
SBP (mm Hg)	139 ± 11
Coronary risk factors	
Diabetes mellitus	52 (32%)
Hypertension	92 (57%)
Hypercholesterolemia	69 (43%)
Current smoking	83 (51%)
Family history of heart disease	57 (35%)
Medications	
Aspirin	123 (76%)
Nitrates	86 (53%)
Beta-blockers	83 (51%)
ACE inhibitors/ARB	70 (43%)
Calcium-channel blockers	91 (56%)
Statins	62 (38%)
Echocardiographic characteristics	
LVDd (mm)	46 ± 5
LVDs (mm)	27 ± 4
FS (%)	32 ± 6
IVSth (mm)	9 ± 1
LVPWth (mm)	9 ± 1

Values are presented as n (%) or mean ± SD.

ACE = angiotensin-converting enzyme; ARB = angiotensin receptor blocker; BSA = body surface area; FS = fractional shortening of left ventricular long-axis diameter; HR = heart rate; IVSth = wall thickness of interventricular septum; LVDd = left ventricular diameter at end diastole; LVDs = left ventricular diameter at end systole; LVPWth = left ventricular posterior wall thickness; SBP = systolic blood pressure.
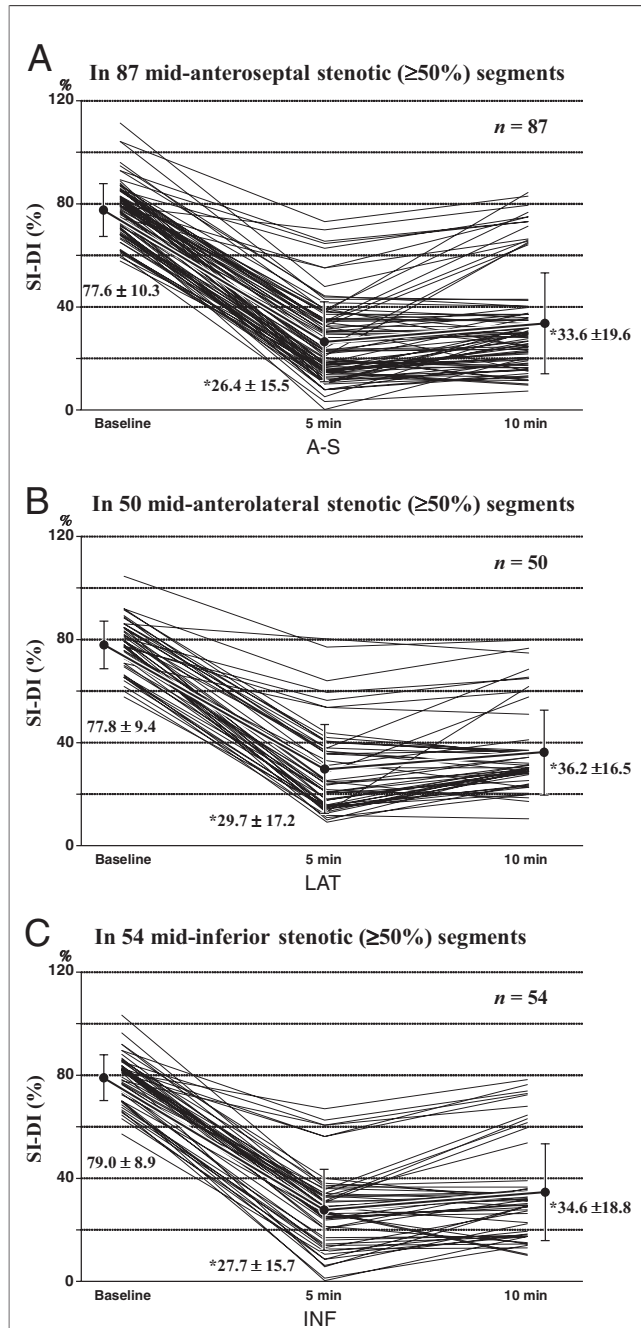
Figure 2   **Comparison of SI-DI Values at Baseline and 5 and 10 Min After Exercise**

Comparisons of strain imaging diastolic index (SI-DI) values at baseline and 5 and 10 min after treadmill exercise in segments perfused by coronary arteries with significant (≥50% of luminal diameter) stenosis. Changes in SI-DI were measured at **(A)** mid-anteroseptal (A-S), **(B)** mid-anterolateral (LAT), and **(C)** mid-inferior (INF) segments. The SI-DI significantly decreased 5 min after exercise as compared with that at baseline. Mean index was recovered but still depressed 10 min after exercise. All values are mean ± SD. *p < 0.0001.
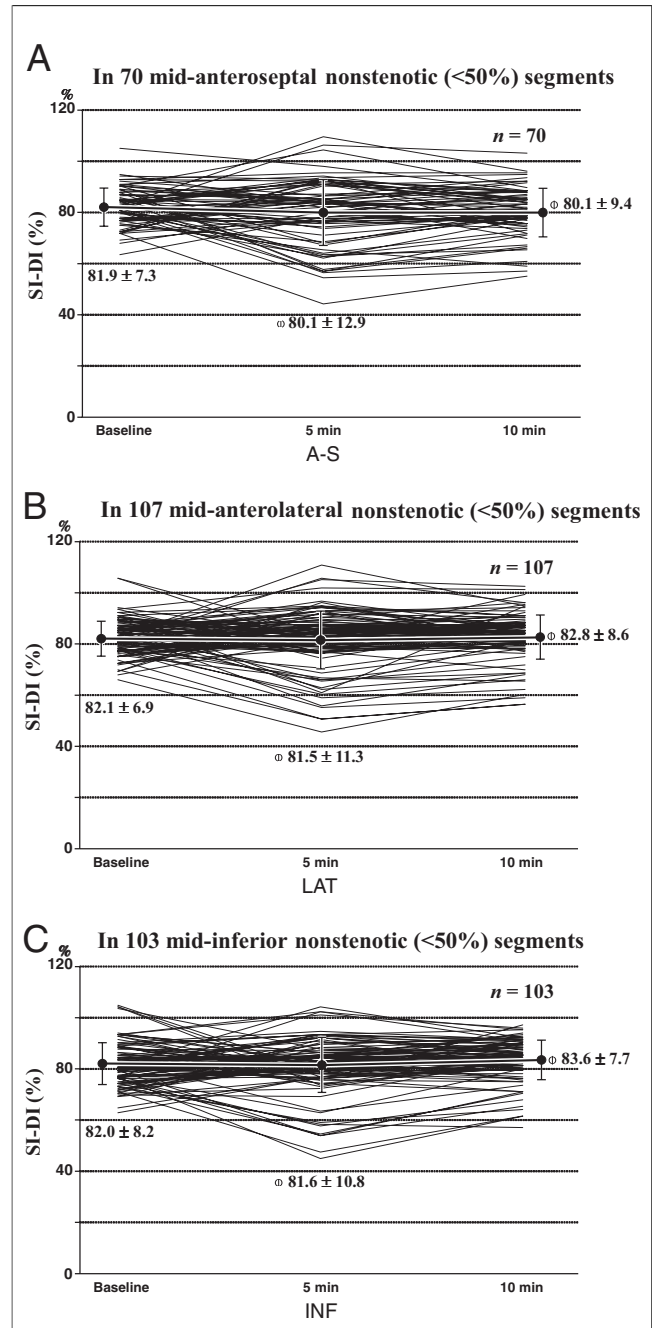

Figure 3   **Comparison of SI-DI Values at Baseline and 5 and 10 Min After Exercise**

Comparisons of SI-DI values at baseline and 5 and 10 min after exercise in segments perfused by coronary arteries with nonsignificant (<50% of luminal diameter) stenosis. Changes in SI-DI were measured at **(A)** A-S, **(B)** LAT, and **(C)** INF segments. The SI-DI remained essentially unchanged after exercise as compared with that at baseline. All values are mean ± SD. ⓪ p = NS. Abbreviations as in Figure 2.

The mean SI-DI decreased from 78.0 ± 9.7% to 27.6 ± 16.0% (p < 0.0001) in 191 territories perfused by coronary arteries with a significant stenosis 5 min after the treadmill exercise stress test, whereas it remained essentially un-

changed from 82.0 ± 7.5% to 81.2 ± 11.5% (p = NS) in 280 territories perfused by normal coronary anatomy or <50% stenosis (Table 2). The SI-DI ratio was significantly decreased in territories perfused by stenotic arteries, but not in territories perfused by nonstenotic arteries (0.34 ± 0.18

vs. 1.0 ± 0.15, $p < 0.0001$) 5 min after the treadmill exercise. Ten minutes after the treadmill exercise, regional delayed relaxation was still observed in 162 (85%) of 191 territories perfused by stenotic coronary arteries. The mean SI-DI increased to $34.6 \pm 18.6\%$ but was still significantly lower ($p < 0.0001$) than that at baseline. Receiver-operator characteristic analysis of SI-DI ratio at 5 min after treadmill exercise yielded optimal cutoff values of 0.74 for the detection of $\geq 50\%$ coronary stenosis. Sensitivity and specificity of the SI-DI ratio were 97% and 93%, respectively. Similarly, the best cutoff value of the SI-DI ratio for the detection of $\geq 70\%$ coronary stenosis was 0.39, which yielded a sensitivity and specificity of 97% and 88%, respectively (Fig. 4). An example of 2D speckle-tracking images and serial change of transverse strain curves from the study patients is shown in Figure 5.

Discussion

Post-ischemic regional LV delayed relaxation after exercise-induced myocardial ischemia. In the current study, regional LV delayed relaxation that probably developed during exercise was detected by the SI method both at 5 and 10 min after exercise-induced myocardial ischemia in patients with stable effort angina and CAD. Sensitivity and specificity for the prediction of $\geq 50\%$ coronary stenosis were 97% and 93%, respectively. Underwood et al. (25) examined 79 studies with 8,964 patients using single-photon emission tomography in the diagnosis of CAD and found a sensitivity of 86% and specificity of 74%. With regard to stress echocardiography, Schuijf et al. (26) pooled 15 studies with 1,849 patients and found a weighted mean sensitivity and specificity of 84% and 82% for the detection of CAD, and a weighted mean sensitivity and specificity of 80% and 84% in 28 studies with 2,246 patients using dobutamine echocardiography. Our data suggest that detection of exercise-induced post-ischemic delayed relaxation using SI has the potential to improve the diagnostic accuracy of stress echocardiography for the detection of significant coronary disease. Techniques such as tissue Doppler-derived strain echocardiography and strain echocardiography that quantify regional mechanics are being increasingly investigated as a means of objectively identifying myocardial ischemia, and are able to distinguish ischemic from nonischemic segments (12,16,27). With conventional echocardiography, detection of ischemia is predicted on the demonstration of systolic abnormalities. However, diastole is an energy-dependent process (2,28), and ischemia-related diastolic abnormalities have been previously shown with several techniques. Visual wall motion analysis is unable to reliably assess diastolic events. Clinical studies have previously shown diastolic abnormalities using Doppler-derived strain echocardiography in CAD (29). However, the relationship of post-ischemic regional diastolic mechanical abnormalities to significant CAD in patients with effort angina has not been clearly elaborated. Knowledge of this relationship may allow the use of diastolic abnormalities to

Table 2 Comparison of Strain Values and SI-DI at Baseline and 5 and 10 Min After Exercise in 191 Stenotic (≥50%) and 280 Nonstenotic (<50%) Segments

	Stenotic (≥50%) Segments						Nonstenotic (<50%) Segments					
	n	Baseline	5 min	p Value	10 min	p Value	n	Baseline	5 min	p Value	10 min	p Value
Strain values at AVC												
Total	191	35.2 ± 5.9	33.5 ± 9.3	0.0102	34.8 ± 5.6	NS	280	36.5 ± 4.7	38.8 ± 6.6	<0.0001	36.9 ± 5.8	NS
Mid-anteroseptum	87	35.1 ± 5.9	33.5 ± 10.2	NS	34.1 ± 5.8	NS	70	36.1 ± 4.7	38.6 ± 7.2	0.0005	36.2 ± 6.5	NS
Mid-anterolateral	50	35.4 ± 7.1	34.6 ± 8.8	NS	35.4 ± 5.6	NS	107	37.0 ± 4.9	39.2 ± 6.5	<0.0001	36.6 ± 5.4	NS
Mid-inferior	54	35.0 ± 4.6	32.6 ± 8.3	0.0269	35.4 ± 5.2	NS	103	36.4 ± 4.6	38.5 ± 6.4	<0.0001	37.6 ± 5.6	0.0232
Strain values at one-third DD												
Total	191	7.6 ± 3.2	23.9 ± 8.1	<0.0001	22.4 ± 6.3	<0.0001	280	6.5 ± 2.7	7.0 ± 4.0	0.0230	6.3 ± 2.9	NS
Mid-anteroseptum	87	7.8 ± 3.4	24.3 ± 8.8	<0.0001	22.1 ± 6.2	<0.0001	70	6.4 ± 2.4	7.3 ± 4.2	NS	6.9 ± 2.8	NS
Mid-anterolateral	50	7.7 ± 3.2	24.1 ± 8.3	<0.0001	22.4 ± 6.2	<0.0001	107	6.6 ± 2.5	7.1 ± 4.1	NS	6.3 ± 3.2	NS
Mid-inferior	54	7.2 ± 2.9	23.2 ± 6.8	<0.0001	22.8 ± 6.6	<0.0001	103	6.5 ± 3.0	6.8 ± 3.6	NS	6.0 ± 2.6	NS
SI-DI												
Total	191	78.0 ± 9.7	27.6 ± 16.0	<0.0001	34.6 ± 18.6	<0.0001	280	82.0 ± 7.5	81.2 ± 11.5	NS	82.4 ± 8.6	NS
Mid-anteroseptum	87	77.6 ± 10.3	26.4 ± 15.5	<0.0001	33.6 ± 19.6	<0.0001	70	81.9 ± 7.3	80.1 ± 12.9	NS	80.1 ± 9.4	NS
Mid-anterolateral	50	77.8 ± 9.4	29.7 ± 17.2	<0.0001	36.2 ± 16.5	<0.0001	107	82.1 ± 6.9	81.5 ± 11.3	NS	82.8 ± 8.6	NS
Mid-inferior	54	79.0 ± 8.9	27.7 ± 15.7	<0.0001	34.6 ± 18.8	<0.0001	103	82.0 ± 8.2	81.6 ± 10.8	NS	83.6 ± 7.7	NS

Values are expressed as mean ± SD. The p values were based on repeated measurement analysis of variance with Dunnett adjustment (vs. baseline).
AVC = aortic valve closure; DD = diastole duration; NS = not significant; SI-DI = strain image diastolic index.
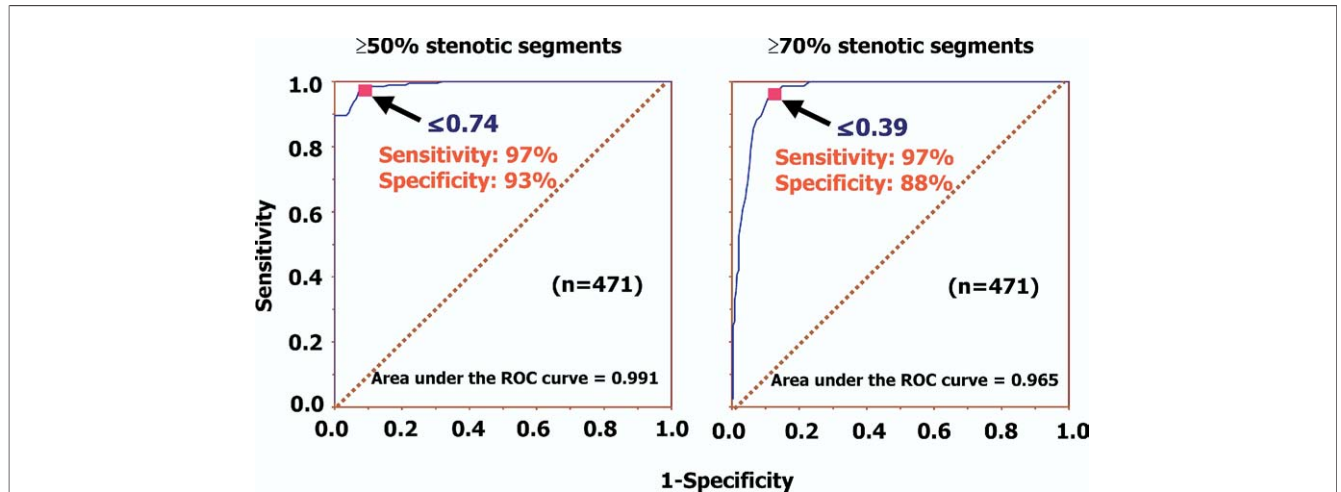
Figure 4 Sensitivity and Specificity of SI-DI Ratio 5 Min After Treadmill Exercise for ≥50% and ≥70% Stenotic Segments

Sensitivity and specificity of the strain imaging diastolic index (SI-DI) ratio 5 min after treadmill exercise for the detection of ≥50% and ≥70% coronary stenosis. Sensitivity and specificity were derived from the receiver-operator characteristic (ROC) curve.
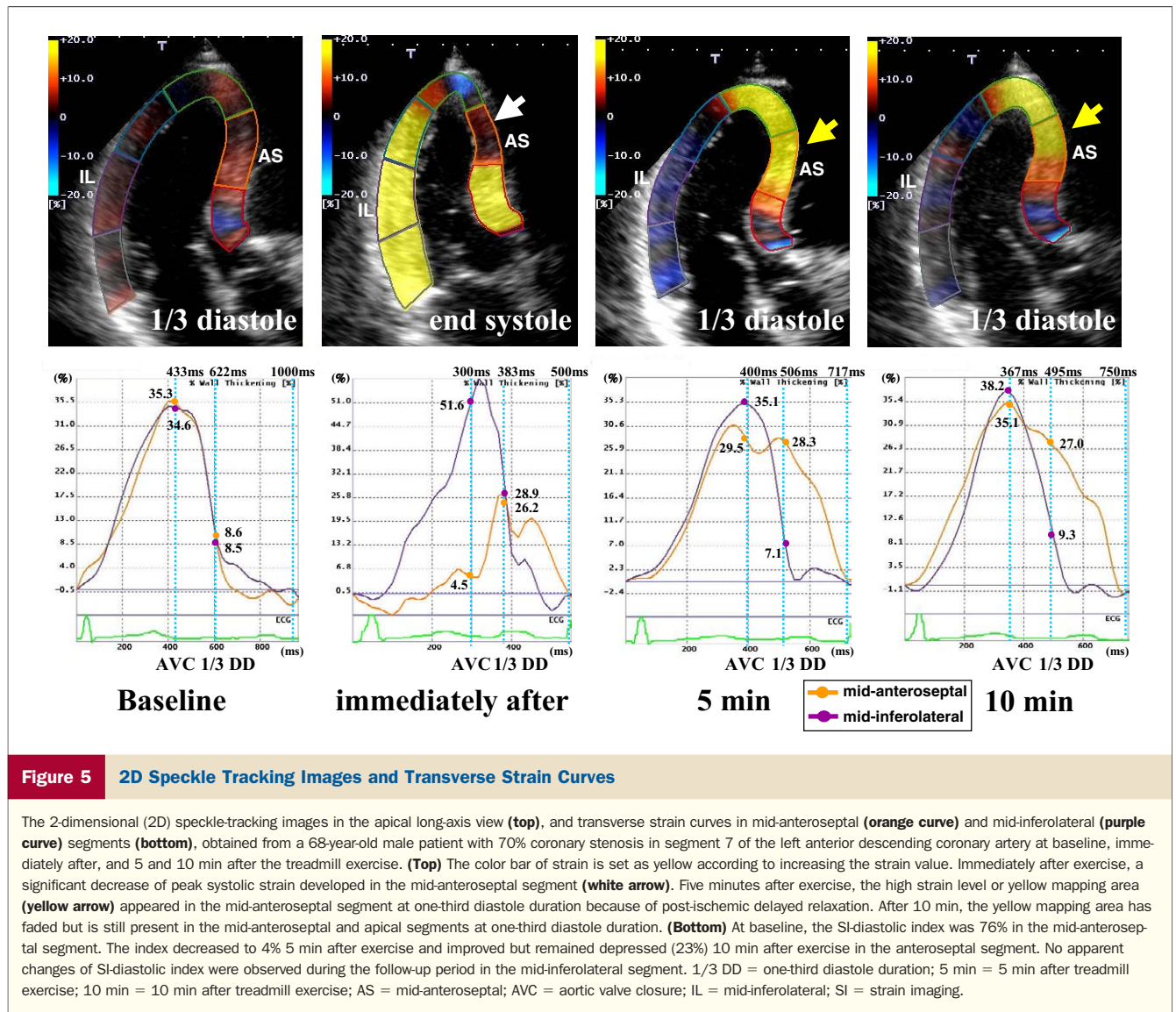
detect CAD. Recently, we reported that regional delayed relaxation or tardorelaxation was observed after a treadmill exercise stress test in patients with stable effort angina and CAD, and this impairment persisted even 1 h after exercise, when normal regional systolic motion is completely restored (11).

Regional myocardial changes in motion and deformation after acute ischemia. Acute regional ischemia causes rapid, predictable, and reproducible change in deformation in a segment. The magnitude of these changes is proportional to the severity of the acute ischemic insult and is flow related (1,30). Systolic deformation decreases with increasing stenosis, whereas post-systolic thickening becomes more prominent. Reperfusion restores deformation to near normal, but some bulging and post-systolic thickening remain in the early phase as result of stunning (31). Jamal et al. (32) reported the typical sequence of changes in M-mode images of the at-risk wall after an acute coronary occlusion. Deformation and motion return to near normal after reperfusion (after a short initial period of hypercontractility caused by hyperemia). When myocardium is ischemic for a prolonged period and subsequently reperfused, deformation remains abnormal despite full restoration of perfusion (33). Monnet et al. (34) showed that, in stunned myocardium, deformation returns to normal within a period of 24 h of reperfusion and that this is paralleled by a decrease in post-systolic thickening.

The mechanisms underlying the phenomenon of ischemia-related post-ischemic deformation have been the subject of much debate. Three different explanations have been proposed, either delayed active contraction, late passive thickening, or elastic recoil overshoot after bulging. Turschner et al. (35) reported that with Thrombolysis In Myocardial Infarction flow grade 3 infarct reperfusion, end systolic wall thickness returned to baseline within 5 min

whereas end diastolic wall thickness increased above baseline level after 1 min. Both wall thickness parameters then continued to increase logarithmically during the 60-min reperfusion period. Bragadeesh et al. (36) have reported that a major mechanism for post-ischemic regional LV dysfunction is myofibrillar edema, and as myofibrillar edema subsides with time, regional LV dysfunction improves.

Study limitations. The present study has several limitations with regard to application of the results to the clinical setting. First, it remains undetermined how long the regional delayed relaxation or stunning actually persists after the demand-ischemia. The duration of the observed diastolic stunning after the demand-ischemia is probably influenced by the total ischemic burden during the treadmill exercise testing and the severity of the coronary stenosis as well as endothelial function in the microvessels of the regional coronary circulation (11,17). Second, the success of this novel tracking algorithm depends on the quality of 2D echocardiographic images (13). Third, asynchronous LV relaxation may exist in patients with other cardiac diseases, such as idiopathic cardiomyopathy and hypertensive heart disease (37). A differential diagnosis may be required in these cases, although the abnormal regional LV relaxation may not be influenced by exercise in nonischemic heart disease as much as in cases with CAD. Fourth, in the present study, only the transverse strain component was assessed. Currently, transverse and longitudinal myocardial deformation can be analyzed in the apical view by using 2D speckle-tracking echocardiography (17), so it would be necessary to show that myocardial ischemia affects transverse and longitudinal function similarly. Thus, the effect of these factors on the relaxation index should be assessed in future studies before applying this method to differentiate patients with CAD from those without it.

Figure 5   2D Speckle Tracking Images and Transverse Strain Curves

The 2-dimensional (2D) speckle-tracking images in the apical long-axis view **(top)**, and transverse strain curves in mid-anteroseptal **(orange curve)** and mid-inferolateral **(purple curve)** segments **(bottom)**, obtained from a 68-year-old male patient with 70% coronary stenosis in segment 7 of the left anterior descending coronary artery at baseline, immediately after, and 5 and 10 min after the treadmill exercise. **(Top)** The color bar of strain is set as yellow according to increasing the strain value. Immediately after exercise, a significant decrease of peak systolic strain developed in the mid-anteroseptal segment **(white arrow)**. Five minutes after exercise, the high strain level or yellow mapping area **(yellow arrow)** appeared in the mid-anteroseptal segment at one-third diastole duration because of post-ischemic delayed relaxation. After 10 min, the yellow mapping area has faded but is still present in the mid-anteroseptal and apical segments at one-third diastole duration. **(Bottom)** At baseline, the SI-diastolic index was 76% in the mid-anteroseptal segment. The index decreased to 4% 5 min after exercise and improved but remained depressed (23%) 10 min after exercise in the anteroseptal segment. No apparent changes of SI-diastolic index were observed during the follow-up period in the mid-inferolateral segment. 1/3 DD = one-third diastole duration; 5 min = 5 min after treadmill exercise; 10 min = 10 min after treadmill exercise; AS = mid-anteroseptal; AVC = aortic valve closure; IL = mid-inferolateral; SI = strain imaging.

Conclusions

Exercise-induced regional myocardial ischemia impairs regional diastolic wall motion in patients with CAD, and this impairment persists for 10 min after exercise. Echocardiographic evaluation of regional myocardial wall motion or delayed relaxation by SI is a useful noninvasive method for detection of prolonged post-ischemic diastolic dysfunction or stunning and for identification of the angina-provoking vessel. Detection of post-ischemic regional LV delayed relaxation after treadmill exercise using SI is a sensitive and reliable method for the diagnosis of CAD.

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