Regional Myocardial Systolic Function During Acute Myocardial Ischemia Assessed by Strain Doppler Echocardiography

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OBJECTIVES	We sought to evaluate if echocardiographic strain measurements could detect acute myocardial ischemia, and to compare this new method with myocardial velocity measurements and wall motion score index.
BACKGROUND	Tissue Doppler echocardiography (TDE) is a promising method for assessing regional myocardial function. However, myocardial velocities measured by tissue Doppler echocardiography (TDE) vary throughout the left ventricle (LV) because of tethering effects from adjacent tissue. Strain Doppler echocardiography (SDE) is a new tool for measuring regional myocardial deformation excluding the effect of adjacent myocardial tissue.
METHODS	Seventeen patients undergoing angioplasty of the left anterior descending coronary artery (LAD) were studied. Left ventricular longitudinal wall motion was assessed by TDE and SDE from the apical four-chamber view before, during and after angioplasty from multiple myocardial segments simultaneously.
RESULTS	Systolic strain values were uniformly distributed in the different nonischemic LV segments, whereas systolic velocities decreased from basis to apex. During LAD occlusion, strain measurement showed expansion in the apical septal segment in 16 of 17 patients ($7.5 \pm 6.5\%$ vs. $-17.7 \pm 7.2\%$, $p < 0.001$) and reduced compression in the mid-septal segment ($p < 0.05$) compared with baseline. Segments not supplied by LAD remained unchanged. Tissue Doppler echocardiography showed reduced velocities in all septal segments ($p < 0.05$) during angioplasty even though LAD does not supply the basal septal segment. Negative systolic velocities were present in 11 of 17 patients. Wall motion score index increased during ischemia (1.3 ± 0.4, $p < 0.05$).
CONCLUSIONS	The new SDE approach might be a more accurate marker than TDE for detecting systolic regional myocardial dysfunction induced by LAD occlusion. (J Am Coll Cardiol 2001;37: 726–30) © 2001 by the American College of Cardiology

Tissue Doppler echocardiography (TDE) has been introduced as a new method to quantify regional myocardial function by measuring tissue velocities (1–6). Studies of TDE indicate that this method may represent an improvement in the noninvasive assessment and comprehension of quantifying regional myocardial function (7–13). Regional Doppler tissue velocities in one area, however, are affected by motion in adjacent tissue and might therefore not differentiate between active contraction and passive motion (14–16). This might be a limitation of this technique.

Strain (change in length per unit length) reflects deformation of a structure and therefore directly describes the contraction/relaxation pattern of the myocardium (17). Strain measurements exclude the effects from adjacent regions and were first applied noninvasively using magnetic resonance imaging (MRI) (18,19). A new algorithm that calculates strain from Doppler tissue velocities has been developed (20). We have recently demonstrated, using an experimental canine model, that this Doppler algorithm correlated well with strain measurements obtained by sonomicrometry (21).

The hypothesis of this study was that regional systolic changes in myocardial function are more precisely detected by analyses of myocardial strain than by tissue velocity measurements.

METHODS

Study population. Seventeen patients, average age 58 ± 10 years (2 women, 15 men), with stable angina pectoris undergoing coronary angioplasty were included. All had significant area stenosis ($\geq 75\%$) of left anterior descending coronary artery (LAD) without any regional left ventricular (LV) dysfunction at rest (ejection fraction: 58 ± 10) or valvular heart disease, as evaluated by ventriculography or echocardiography. Significant collateral arteries should not be present in order to ensure significant myocardial ischemia. All were in regular sinus rhythm. Five patients had additional significant stenosis of the circumflex artery (CX) and eight had stenosis of the right coronary artery. Angioplasty of the LAD lesion was always performed first.

The regional ethical committee on human research ap-

Abbreviations and Acronyms

2-D	=	two-dimensional
CX	=	circumflex artery
LAD	=	left anterior descending coronary artery
LV	=	left ventricle
MRI	=	magnetic resonance imaging
SD	=	standard deviation
SDE	=	strain Doppler echocardiography
TDE	=	tissue Doppler echocardiography
WMSI	=	wall motion score index

proved the study. All individuals gave written informed consent.

Echocardiography. Studies were performed with a System Five instrument (GE Vingmed Sound, Horten, Norway) using a phased-array duplex multifrequency transducer. Two-dimensional (2-D) TDE images of the LV were obtained from the apical four-chamber view before, during and 10 min after angioplasty. All analyses were performed from three regions along the interventricular septum (apical, mid- and basal septum) and in the corresponding regions on lateral wall. Recordings were stored digitally as 2-D cineloops and analyzed with customized dedicated research software (Echopac, GE Vingmed Sound, Horten, Norway).

Strain Doppler echocardiography. Strain (ϵ) is originally defined as a dimensionless quantity produced by the application of a stress as described by Mirsky and Parmley (17). Furthermore, it represents the fractional or percentages change from the original or unstressed dimension (22–25) (Fig. 1). This equals the relative change of segmental length occurring between the reference state (l) (end-diastole) and the state of deformation (Δl) (end-systole) expressed in percentage of end-diastolic length.

Strain rate (SR) is equal with the temporal derivative of strain, and the total strain can therefore be determined by combining the strain rate values from a given time interval. Strain may thus be calculated from tissue Doppler velocities (ν) as shown in Figure 1 and equation 1:

$$\epsilon = \frac{\Delta l}{l} \approx \frac{v(r) - v(\Delta r + r)}{\Delta r} \Delta t = SR\Delta t \qquad [1]$$
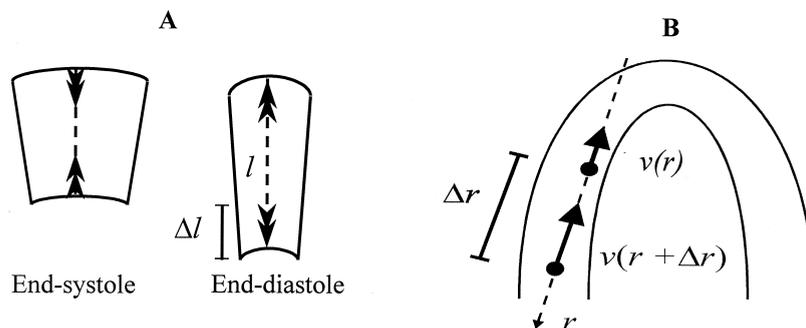
The velocity gradient or *SR* is estimated from two points (a spatial resolution) with a distance (Δr). This spatial resolution is adjustable and 8 mm gave the best signal-to-noise ratio.

The 2-D TDE and SDE techniques used in the present study allowed processing of simultaneous velocity and strain traces from different myocardial segments in the same cineloop (Fig. 2). Velocity and strain traces from the apical segments were extracted from the most proximal part of the segments to avoid a potential angle problem in measuring apical velocities. The peak systolic strain in each segment was easy to define. By definition, shortening strains are expressed as negative values and lengthening strains have positive signs. The superimposed color-coded Doppler velocities were removed and one investigator blinded for the SDE and TDE results performed visual assessment of the original gray scale 2-D loop. Wall motion was assessed by analyses of movement and thickening of the myocardium in the three septal and three lateral segments in the apical four-chamber view. A score was given to each segment: 1 = normal, 2 = hypokinesia, 3 = akinesia, 4 = dyskinesia. A wall motion score index (WMSI) was calculated as the sum of scores over number of analyzed segments.

Statistical analysis. Data are presented as mean value ± SD. Dependencies between velocities, strain and wall motion score at different time points and different segments were analyzed with two-way analysis of variance methods. A model for repeated measurements was used that allowed multiple comparisons between the levels and study of the interaction between time and segments (SPSS version 9, Chicago, Illinois). Differences were considered statistically significant if the p-value was <0.05 following Bonferroni correction. Ten patients were randomly selected and strains in 60 segments were analyzed later by the same observer (TE) and another observer (HS). Reproducibility was expressed as 95% limits of interobserver agreement (26).

RESULTS

All patients had ST-depression in their electrocardiograms (-1.7 ± 0.7 mm) during the coronary angioplasty and 14 of the 17 patients expressed chest pain. A total of 306 segments (6 segments × 17 patients × 3 interventions) were

Figure 1. Schematic illustration of how strain rate within a tissue segment (Δr) is estimated from the tissue velocity (ν). The dashed line indicates orientation of the ultrasound beam. The distance along the beam is denoted r. The strain rate is calculated by subtracting $\nu(r + \Delta r)$ from $\nu(r)$ over the distance (Δr) between these two points. Size of Δr is exaggerated for clarity. l= a given length; Δl = instantaneous change in length.
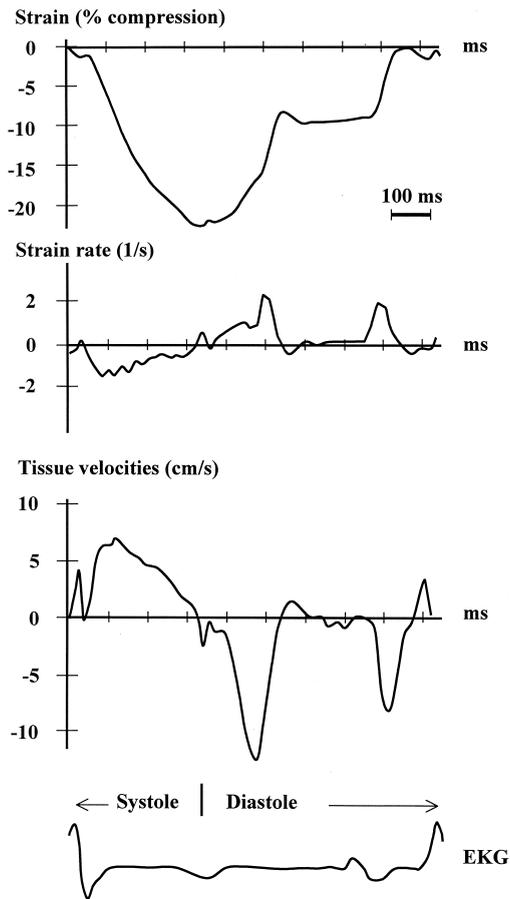
Figure 2. Representative strain **(upper)**, strain rate **(middle)** and tissue velocity **(lower)** profiles obtained from identical sample point in the longitudinal axis before coronary angioplasty. All measurements were done from the same heartbeat.

studied. The image frame rate obtained in this study was 69 ± 16 frames/s and heart rate was 57 ± 8 beats/min. Technically acceptable recordings were present in 97% of all segments studied.

Baseline. In all patients, systolic velocities measured with TDE showed for each segment a contraction pattern directed toward apex (Table 1). The highest velocities were measured in the basal segments and the lowest velocities in the apical segments ($p < 0.01$).

Table 1. Myocardial Systolic Velocities (cm/s) as Measured From Doppler Tissue Echocardiography at Baseline, During and After LAD Occlusion

	Baseline	LAD Occlusion	10 s after LAD Occlusion
Septum			
Basal segment	5.8 ± 1.5	$4.4 \pm 1.6^*$	6.0 ± 0.9
Mid-segment	4.2 ± 1.5	$1.9 \pm 1.5†$	4.2 ± 1.0
Apical segment	2.9 ± 1.2	$0.8 \pm 1.6^*$	2.5 ± 0.7
Lateral wall			
Basal segment	5.4 ± 1.5	5.2 ± 1.7	5.6 ± 1.9
Mid-segment	4.5 ± 1.4	4.1 ± 1.5	4.6 ± 1.4
Apical segment	3.4 ± 1.6	$1.8 \pm 2.5^*$	3.7 ± 1.3

$^*p < 0.05$. $†p < 0.01$. p values versus baseline. Mean values $\pm$ SD. LAD = left anterior descending coronary artery.

Table 2. Systolic Strain Measurements (%) at Baseline, During and After LAD Occlusion

	Baseline	LAD Occlusion	10 s after LAD Occlusion
Septum			
Basal segment	-20.2 ± 8.5	-19.4 ± 11.6	-21.2 ± 7.6
Mid-segment	-21.8 ± 8.2	$-13.1 \pm 4.1^*$	-17.8 ± 6.4
Apical segment	-17.7 ± 7.2	$7.5 \pm 6.5†$	-15.6 ± 7.9
Lateral wall			
Basal segment	-16.5 ± 5.4	-17.9 ± 7.2	-17.8 ± 4.7
Mid-segment	-16.0 ± 5.6	-18.4 ± 7.3	-16.1 ± 7.4
Apical segment	-16.2 ± 6.4	-19.3 ± 8.9	-20.9 ± 10.4

$^*p < 0.05$. $†p < 0.001$. p values versus baseline. Mean values $\pm$ SD. LAD = left anterior descending coronary artery.

Strain measurements demonstrated shortening (compression) during systole in all segments (Table 2) and lengthening (expansion) during diastole. Systolic strain values were similar in all segments seen from the LV four-chamber view (ns).

All patients had normal WMSI (1.0 ± 0.0) as assessed from the 2-D loops (Table 3).

Acute LAD ischemia. Peak systolic velocity measured by TDE decreased significantly in all septal segments and the apical lateral segment. No significant changes in velocities were found in mid- and basal segments of the lateral wall. Negative systolic velocities were found most frequently in the apical septal segment, where it was present in 11 of 17 patients.

Left anterior descending coronary artery occlusion resulted in a significant decrease in strain in the mid-part of the LV septum and considerable change in the apical segment with expansion (paradoxical motion) in all patients except one (Fig. 3). Significant changes were not present in basal septal segment and in the lateral wall.

The WMSI was higher during ischemia compared to baseline (1.3 ± 0.4, $p < 0.01$). Reduced wall motion was seen in the apical septal segment in nine of 17 patients during LAD occlusion. Six patients had impaired wall motion in the lateral apical segment; three of these showed considerable reduced strain.

The interaction between time of observation and segment was significant for the strain data during occlusion in mid-septal segment ($p < 0.05$) and apical septal segment ($p < 0.001$). No consistent interaction was present for the tissue velocity data and visual assessment of wall motion.

Table 3. Wall Motion Score From Each Segment at Baseline, During and After LAD Occlusion

	Baseline	LAD Occlusion	10 s after LAD Occlusion
Septum			
Basal segment	1.1 ± 0.3	1.0 ± 0.1	1.0 ± 0.0
Mid-segment	1.0 ± 0.0	1.6 ± 0.7	1.1 ± 0.3
Apical segment	1.1 ± 0.3	1.8 ± 0.7	1.1 ± 0.3
Lateral wall			
Basal segment	1.0 ± 0.0	1.0 ± 0.0	1.0 ± 0.0
Mid-segment	1.0 ± 0.0	1.1 ± 0.3	1.0 ± 0.0
Apical segment	1.0 ± 0.0	1.5 ± 0.7	1.0 ± 0.0

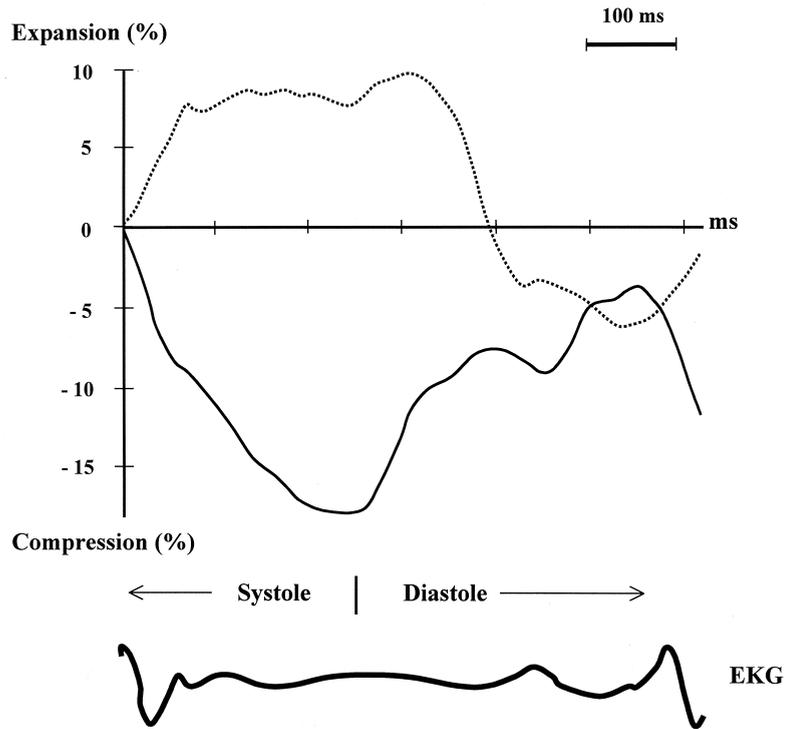Mean values $\pm$ SD. LAD = left anterior descending coronary artery.

Figure 3. Strain profiles from different segments in the same heartbeat during left anterior descending coronary artery (LAD) occlusion. The profile from basal septum **(solid line)** showing compression **(normal deformation)** and from the ischemic apical septum **(dashed line)** showing expansion **(paradoxical motion)**.

Reproducibility. The interobserver variability for peak systolic strain and velocity was $-0.8 \pm 5.5\%$ and 0.1 ± 0.7 cm/s, respectively. There was a significant correlation between strain measurements obtained by the two observers: $y = 0.9x + 2.6$, $r = 0.78$, $p < 0.001$. With respect to assessment of expansion or compression during systole, there was full agreement in 58 of 60 segments (97%). Intraobserver variability was $0.7 \pm 6.6\%$ for strain and -0.2 ± 0.8 cm/s for velocity. No systematic differences between these measurements were found.

DISCUSSION

This study shows that the strain Doppler technique detected systolic longitudinal expansion or dyskinesia in the apical septal segment in nearly all patients (94%) during controlled occlusion of LAD. Tissue Doppler echocardiography measurements revealed longitudinal expansion in only 65% of the patients, indicating that the strain technique is a more sensitive method for detecting regional ischemic wall motion abnormalities. This conclusion is also supported by the statistical interaction test, which suggests a more convincing change for strain measurements than for tissue velocities in the ischemic segments during LAD occlusion. Heimdal et al. (20) found that tissue Doppler measures positive LV velocities even in akinetic myocardial infarcts whereas SDE showed zero strains. They suggested that the TDE velocities were caused by passive movement of the adjacent normal tissue (21).

Our study shows that tissue velocities increase in the septum and lateral wall from apex to base in a nonischemic LV. With the SDE technique, however, the systolic shortening was uniformly distributed in all segments. This uniformity is in accordance with a recent MRI study (27).

Furthermore, the basal septal segment showed decreased systolic velocities by TDE, despite the fact that this segment is normally perfused by the right coronary artery (28,29). Strain measurement showed unchanged shortening in this segment. The apical lateral segment is normally perfused by LAD or from a LAD/CX overlap (28,29). Myocardial velocities in the apical lateral segment were significantly reduced during ischemia while strain values and WMSI remained unchanged. Measurements were performed in the proximal part of this segment close to or within the CX perfusion area. This probably explains the unchanged strain in this area during ischemia.

These findings strongly support the idea that the SDE technique excludes the tethering effect of adjacent myocardial tissue and explain why paradoxical movement in the ischemic area was found less frequently by the TDE technique.

Clinical implications. The results of this study indicate that strain measured by Doppler may be an important supplement to visual assessment of regional LV dysfunction. Strain Doppler echocardiography could be particularly valuable as an objective quantification to assess regional ischemic responses during stress echocardiography.

Limitations. Strain measurements are angle dependent, possibly more so than other Doppler modalities. Tissue deformation in one direction is always associated with deformations in other directions to keep the mass of the

structure constant. Therefore interpretations of strains should be performed with caution if tissue direction deviates more than 30 degrees from the beam direction. This is why measurements in this study were performed in the proximal part of the apical segments. The angle problem is a significant limitation of this technique and repositioning of the transducer must be done to avoid the problem.

All patients were examined in supine position because of the angioplasty procedure. The image quality was thus suboptimal and visual interpretation of endocardial motion difficult. Nevertheless, reliable estimations of strains could be done in nearly all segments indicating that this technique might be of great importance when 2-D image quality is poor.

Deformation of the heart through the cardiac cycle is best measured in three dimensions. In this study we assessed only the longitudinal dimension because of short imaging time during balloon inflation. Important information about radial and circumferential strain may therefore have been lost. Selective imaging from apical four-chamber view has, however, the important advantage that multiple segments of the LV could be assessed from the same heartbeat.

Conclusions. The present study demonstrates that SDE detects longitudinal dyskinesia during occlusion of LAD more frequently than does TDE. Normally, myocardial strain values are uniformly distributed in most parts of the LV. This new modality might enhance the accuracy of echocardiography for diagnosing myocardial ischemia and regional dysfunction.

Acknowledgments

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