

Myocardial Strain by Doppler Echocardiography

Validation of a New Method to Quantify Regional Myocardial Function

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Background—Myocardial strain is a measure of regional deformation, and by definition, negative strain means shortening and positive strain, elongation. This study investigates whether myocardial strain can be measured by Doppler echocardiography as the time integral of regional velocity gradients, using sonomicrometry as reference method.

Methods and Results—In 13 anesthetized dogs, myocardial longitudinal strain was measured on apical images as the time integral of regional Doppler velocity gradients. Ultrasonic segment-length crystals were placed near the left ventricular (LV) apex and near the base. Apical ischemia was induced by occluding the left anterior descending coronary artery (LAD), and preload was increased by saline. Percentage systolic strain by Doppler correlated well with strain by sonomicrometry ($y=0.82x-1.79$, $r=0.92$, $P<0.01$). During LAD occlusion, apical myocardium became dyskinetic, as indicated by positive strain values and negative Doppler velocities. At the LV base, myocardial strain by Doppler, strain by sonomicrometry, and velocity of shortening by sonomicrometry (dL/dt) were unchanged during apical ischemia. However, myocardial Doppler velocities at the base decreased from 4.2 ± 0.7 (\pm SEM) to 2.7 ± 0.4 cm/s ($P<0.05$), probably reflecting loss of motion caused by tethering to apical segments. Volume loading increased myocardial Doppler velocities from 2.2 ± 0.3 to 4.1 ± 0.8 cm/s ($P<0.05$) and Doppler-derived strain from $-12\pm1\%$ to $-22\pm2\%$ ($P<0.05$), whereas peak LV elastance remained unchanged.

Conclusions—Myocardial strain by Doppler echocardiography may represent a new, powerful method for quantifying regional myocardial function and is less influenced by tethering effects than Doppler tissue imaging. Like myocardial Doppler velocities, strain is markedly load-dependent. (*Circulation*. 2000;102:1158-1164.)

Key Words: echocardiography ■ strain ■ ischemia ■ ultrasonics ■ contractility

Currently available routine methods for assessment of regional myocardial function are subjective and at best semiquantitative.¹⁻³ Recently, tissue Doppler imaging has been introduced as a new method to quantify regional myocardial function.⁴ This method determines myocardial tissue velocities by measuring the changes of phase shift of the ultrasound signal returning from the myocardium. The initial evaluations of tissue Doppler indicate that it may represent a significant step forward in the noninvasive assessment of myocardial function.⁵⁻⁷ Regional myocardial tissue velocities, however, represent the net effect of the contractile and elastic properties of the area under investigation and traction and tethering effects from other regions. In addition, cardiac translational artifacts influence the measured velocities. Therefore, one would rather like to measure myocardial deformation or strain, which more directly reflects local myocardial function. At present, strain can be measured noninvasively by MRI,^{8,9} but the complexity of this method limits its application in clinical routine.

Strain (ϵ) is a dimensionless quantity and is produced by application of stress. It represents the fractional or percentage

change from the original or unstressed dimension and includes both lengthening, or expansion (positive strains) and shortening, or compression (negative strains). Ideally, one would like to measure Lagrangian strain, defined as $(L-L_0)/L_0$, where L_0 is the length corresponding to zero stress and L is the instantaneous length.¹⁰ Because zero stress lengths are technically difficult to measure, L_0 is often replaced by the initial muscle length or the end-diastolic length. Strain rate is the temporal derivative of strain and is a measure of the rate of deformation, with units of 1/s. The strain rate is also equivalent to the shortening velocity per fiber length.

Previous studies have demonstrated myocardial velocity gradients by tissue Doppler echocardiography and suggest that myocardial strain rate can be calculated from velocity gradients in time and space.^{11,12} Ultrasound strain rate has recently been implemented as a real-time imaging modality and has been described by Heimdal et al.¹³

The aim of the present study was to validate Doppler-derived strain as a method to quantify regional myocardial function. In a prototype computer postprocessing program,

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tissue velocities and strain rate values from multiple points along an M-mode cursor line were extracted. By integrating strain rate over time, we obtained strain throughout the cardiac cycle. The strain estimates were expressed as instantaneous length in percentage of end-diastolic dimension. The mathematics and principles behind this analysis are described in the Appendix. The study was done in acutely anesthetized dogs in which a wide range of strains were induced by coronary occlusion and volume loading, respectively. As reference method for strain, we used myocardial segment lengths measured by sonomicrometry.¹⁴

Methods

Thirteen dogs of either sex and average body weight 22.0 kg were given thiopentone 25 mg/kg body wt and morphine 100 mg IV, followed by infusion of morphine 50 to 100 mg/h IV and pentobarbital 50 mg IV every hour. The animals were artificially ventilated through a cuffed endotracheal tube with room air with 20% to 50% oxygen. An ECG was monitored from a limb lead. A jugular and a femoral vein were cannulated. After a median sternotomy, the pericardium was split from apex to base, and after the instrumentation, the edges of the pericardial incision were loosely resutured. An inflatable vascular occluder was placed around the proximal third of the LAD. In 4 of the dogs, we placed inflatable vascular occluders around both caval veins. The dogs were placed in the right supine position during recordings. The study was approved by the National Animal Experimentation Board.

Pressure Measurements

A 5F micromanometer-tipped catheter (model MPC-500, Millar Instruments) was positioned in the left ventricle (LV) through a carotid artery. Via the appendage, a 5F micromanometer and a fluid-filled catheter were placed in the left atrium. All pressure transducers were calibrated with a mercury manometer. The pressures were zero-referenced against the fluid-filled left atrial catheter. Pressure and ECG data were processed via preamplifiers and were digitized at 200 Hz for further analysis on a PC computer station.

Sonomicrometry

A pair of ultrasonic crystals was implanted in the inner half of the myocardium in the anterior LV wall near the apex, and another pair was implanted in the anterolateral wall near the base. Both sets of crystals were aligned parallel to the LV long axis. The crystals were connected to a sonomicrometer (Triton Technology Inc or Sonometrics). By calculating the instantaneous length in percent of end-diastolic length, we obtained strain throughout the cardiac cycle, and on this trace we measured peak negative systolic strain (% ΔL). During ischemia, there was systolic lengthening, and we report peak positive strain. In 4 of the dogs, we inserted 3 pairs of endocardial diameter crystals to measure LV volume; ie, base-apex, equatorial septum-free wall, and anteroposterior diameters. LV volumes were calculated as a general ellipsoid.¹⁵

Echocardiographic Analysis

We used a System FiVe digital ultrasound machine (GE Vingmed Ultrasound) with a combined tissue imaging (2.5- to 3.5-MHz) and Doppler (2.75-MHz) transducer. The strain rate images were collected with a frame rate varying from 48 to 121 frames per second, with a mean value of 69 frames per second, and to minimize the noise level, the pulse repetition frequency was set to 250 to 300 Hz, and second harmonic imaging was used.

Recordings were done from apical views, and we oriented the 2D image planes through the regions in which we had inserted the segment-length crystals; ie, the anterolateral wall near the apex and the anterolateral wall near the base. The measurements were taken in the longitudinal direction from the inner half of the LV wall.

For the measurement of tissue velocities, we used a 2D multiregion technique, which allowed simultaneous processing of multiple

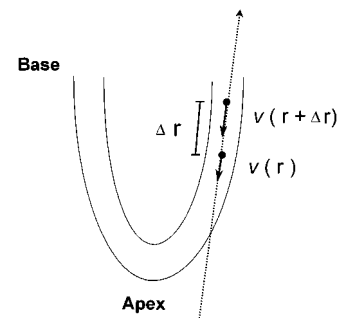


Figure 1. Schematic of how strain rate of tissue segment (Δr) is estimated from tissue velocity (v). Dashed line indicates orientation of ultrasound beam. Distance along beam is denoted r . Strain rate is calculated by subtracting $v(r + \Delta r)$ from $v(r)$ over distance Δr between these 2 points. When velocities are equal, strain rate is zero and there is no compression or expansion. If $v(r + \Delta r) > v(r)$, strain rate is negative and there is compression. When $v(r)$ exceeds $v(r + \Delta r)$, strain rate is positive, indicating expansion.

velocity traces from optional places on the 2D images. The pulse repetition frequency was set to 1.0 to 1.5 kHz. In this software, the images showing the velocity of tissue motion are superimposed on the 2D echocardiographic images for real-time display in color. These color-coded velocities are automatically decoded into numerical values for quantitative analysis. Peak systolic myocardial velocity was assessed from the apical and basal segments in the same regions as for strain rate measurements.

The strain rates were color-coded, and strain rate images were superimposed on the tissue images. An experimental application programmed in Microsoft Visual C++ was used to extract strain rate and strain along an M-mode line that was oriented in the direction from apex to base. Measurements were averaged over a region of ≈ 10 mm along the M-mode line. The measurements were taken from the inner half of the myocardium, as close as possible to the myocardial segments in which we had placed the ultrasonic crystals. Figure 1 shows schematically how strain rates are obtained, and the Appendix gives the details.

Experimental Protocol

In 8 dogs, after a baseline recording, the LAD was occluded for 1.0 to 3.5 minutes (mean, 2.3 minutes), and recordings were repeated. In 5 dogs, after a baseline recording, we increased stroke volume by infusion of 500 to 1000 mL saline IV. Four of these dogs were instrumented with LV diameter crystals and were used to determine peak LV systolic elastance (E_{\max}). E_{\max} was measured during transient caval occlusions as the slope of the end-systolic pressure-volume relationships and was used as index of LV contractility.¹⁶

Because of interference between sonomicrometry and Doppler, we first recorded pressures, ECG, and echocardiographic data during 10 seconds and then pressures, ECG, and segment lengths during the subsequent 10 seconds. Pressure and ECG data were processed via preamplifiers and were digitized at 200 Hz for further analysis by CVSOFT (Odessa Computers). Data were recorded with the respirator off.

Statistics

Values are expressed as mean \pm SEM. Statistical analysis was performed with Student's t test for paired data. The strain values obtained by the 2 different methods were compared by use of the method of Bland and Altman¹⁷ and by regression analysis with a least-squares method. We considered results significant at a value of $P < 0.05$.

Results

Figure 2 is a representative recording of myocardial strain rate and strain by Doppler echocardiography. Figure 3 dis-

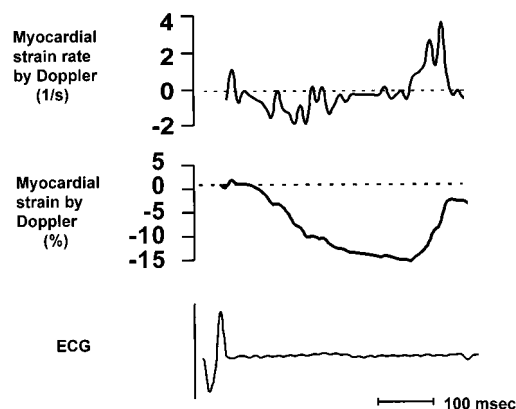


Figure 2. Myocardial longitudinal strain rate and strain by Doppler. When strain was measured, most noise in strain rate signal was canceled out, and we obtained a relatively smooth signal.

plays myocardial strain in both LV major axes during baseline, showing systolic shortening (compression) in the long axis and systolic thickening (expansion) in the short axis. For the rest of this article, all reported strain values are calculated in the long axis.

Figure 4 shows a representative experiment and displays myocardial long-axis strain by Doppler along with myocardial strain by sonomicrometry. Before LAD occlusion, peak systolic strains in the apical region were $-16 \pm 1\%$ and $-13 \pm 2\%$ ($P=NS$) by sonomicrometry and Doppler, respectively. During LAD occlusion, either technique showed systolic lengthening of ischemic myocardium, and systolic strains by sonomicrometry and Doppler were $13 \pm 2\%$ and $10 \pm 1\%$ ($P=NS$), respectively. Myocardial Doppler velocities changed from positive to negative, consistent with dyskinesia.

In the nonischemic region at the LV base, myocardial Doppler velocities decreased markedly ($P<0.05$) during

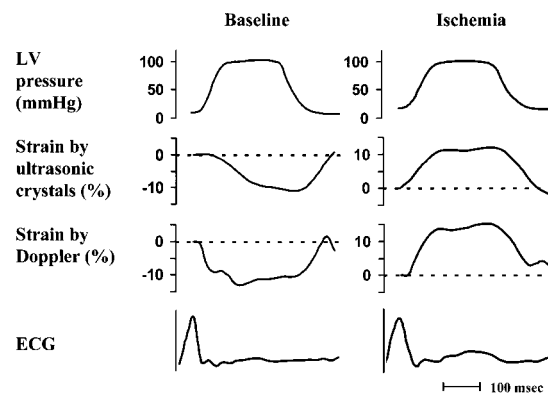


Figure 4. LV pressure, ECG, and longitudinal myocardial strain by ultrasonic crystals and by Doppler before and during ischemia. Both methods demonstrate systolic compression during baseline and systolic expansion during ischemia.

LAD occlusion, whereas there was no significant change in strain rate or in strain by either method (Table 1). Furthermore, the time derivative of the segment length (dL/dt) was unchanged, indicating no change in regional velocity of shortening.

Volume loading increased LV end-diastolic pressure from 5.6 ± 1.5 to 16.0 ± 1.2 mm Hg ($P<0.05$) (Table 2). In these animals, peak systolic strains in the LAD region by sonomicrometry and Doppler were $-13 \pm 1\%$ and $-12 \pm 1\%$, respectively, at baseline and increased to $-22 \pm 3\%$ ($P<0.05$) and $-22 \pm 2\%$ ($P<0.05$), respectively, after volume loading. There were also significant increments in myocardial strain rate and tissue velocities. Volume loading caused no change in E_{max} , indicating unchanged LV contractility (Table 2 and Figure 5).

Figure 6 displays individual data and demonstrates that peak systolic strain by Doppler correlated well with peak strain by sonomicrometry ($r=0.92$, $P<0.01$). The Bland-

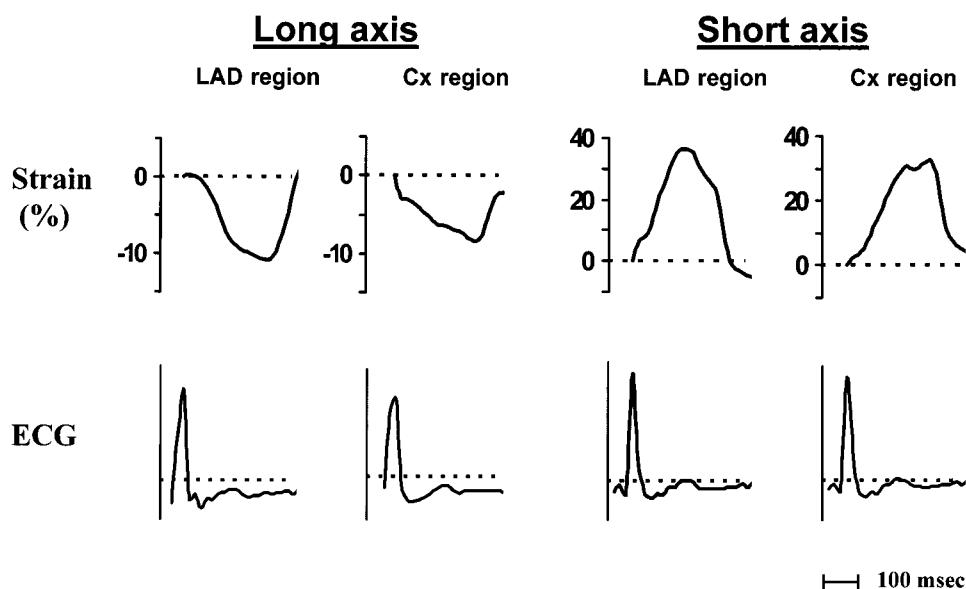


Figure 3. Myocardial strain in both LV major axes at baseline. In longitudinal axis (left), there was shortening (compression) in systole and lengthening (expansion) in diastole. In short-axis view (right), both anterior and posterior walls showed thickening (expansion) in systole and thinning (compression) in diastole.

TABLE 1. Hemodynamic Variables During Baseline and Ischemia (n=8)

	Baseline	Ischemia
Heart rate, bpm	134±8	127±5
LV peak systolic pressure, mm Hg	106±4	104±5
LV end-diastolic pressure, mm Hg	7.8±1.6	9.8±1.8
LV dP/dt _{max} , mm Hg/s	2196±202	1884±200*
End-diastolic segment length, Cx region, mm	7.6±0.9	7.8±1.1
End-diastolic segment length, LAD region, mm	10.4±1.3	10.9±1.3*
Systolic strain, Cx region, by sonomicrometry, %	-12±4	-14±4
Systolic strain, Cx region, by Doppler, %	-12±2	-11±2
Systolic strain, LAD region, by sonomicrometry, %	-16±1	13±2*
Systolic strain, LAD region, by Doppler, %	-13±2	10±1*
Systolic strain rate, Cx region, by Doppler, %	-1.4±0.3	-1.3±0.3
Systolic strain rate, LAD region, by Doppler, 1/s	-1.7±0.4	2.5±0.6*
Systolic Doppler velocities, Cx region, cm/s	4.2±0.7	2.7±0.4*
Systolic Doppler velocities, LAD region, cm/s	3.0±0.5	-0.9±0.2*
dL/dt, LAD region, cm/s	-1.3±0.2	2.1±0.2*
dL/dt, Cx region, cm/s	-0.9±0.1	-0.9±0.4

LV dP/dt_{max} indicates peak positive time derivative of LV pressure; Cx, left circumflex artery; dL/dt, peak systolic time derivative of myocardial segment length by sonomicrometry. Values are given as mean±SEM.

**P*<0.05 (ischemia vs baseline).

Altman plot in Figure 7 shows the agreement between the 2 methods.

Beat-to-beat variability was obtained in each experiment by measuring strain by Doppler in 2 randomly selected beats. The mean values±SD of differences between measurements were 3.0±2.8% and 2.6±2.4% before and during LAD occlusion, respectively.

Angle Dependency of Doppler-Derived Strain

Figure 8 illustrates the importance of the angle between the ultrasonic beam and the LV axis. In 3 dogs, we rotated the probe while measuring strain in the same myocardial region (Figure 8A). At 0° (long-axis orientation), there was systolic compression (-16% on average), and at 90° (short-axis orientation), there was systolic expansion (21% on average) of the anterior LV wall. When the angle approached 45°,

however, the measured strain was markedly reduced (3% on average) (Figure 8B).

Discussion

The present study demonstrates that myocardial strain can be measured noninvasively and in real time from regional myocardial velocities by tissue Doppler echocardiography. This was possible with an algorithm that calculates spatial differences in tissue velocities between neighboring myocardial regions. The underlying principle is that instantaneous differences in tissue velocity between 2 adjacent regions imply either compression or lengthening of the tissue in between. These differences per unit length express instantaneous regional myocardial deformation or strain rate and have units of 1/s. By integrating the velocity gradients over time, we obtained strain throughout the cardiac cycle. It

TABLE 2. Hemodynamic Variables During Baseline and Volume Loading (n=5)

	Baseline	Loading
Heart rate, bpm	96±8	89±5
LV peak systolic pressure, mm Hg	101±4	115±5*
LV end-diastolic pressure, mm Hg	5.6±1.5	16.0±1.2*
LV dP/dt _{max} , mm Hg/s	1267±95	1469±156
End-diastolic segment length, LAD region, mm	12.2±2.3	14.0±2.7*
Systolic strain, LAD region, by sonomicrometry, %	-13±1	-22±3*
Systolic strain, LAD region, by Doppler, %	-12±1	-22±2*
Systolic strain rate, LAD region, by Doppler, 1/s	-1.5±1	-4.4±1*
Systolic Doppler velocities, LAD region, cm/s	2.2±0.3	4.1±0.8*
E _{max} slope, mm Hg/mL	4.8±0.4	3.7±0.6

LV dP/dt_{max} indicates peak positive time derivative of LV pressure; E_{max} slope, slope of the LV end-systolic pressure-volume relationship. Values are given as mean±SEM.

**P*<0.05 (loading vs baseline).

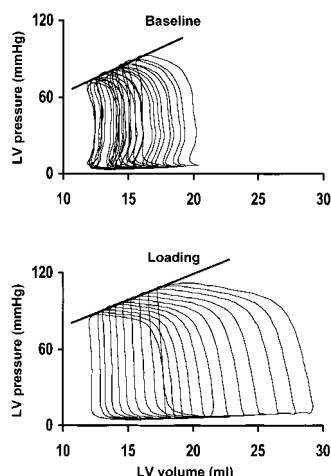


Figure 5. LV pressure-volume curves recorded during transient caval occlusions. Peak LV systolic elastance (E_{max}) did not change with volume loading.

turned out, however, that the strain rate signal had significant noise. This was mainly a result of random noise in the velocity signal, and this noise was magnified when differences were measured instead of absolute velocities. By the integration procedure, however, the random noise was cancelled out, and we obtained a relatively smooth signal.

The validity of our approach for calculating strain was confirmed by use of sonomicrometry as reference method. Over a wide range of strains induced by volume loading and coronary occlusion, respectively, the Doppler method showed strains that approximated those measured by implanted ultrasonic crystals. Taken together, these findings indicate that it is feasible to assess regional myocardial strain by Doppler echocardiography. In this model, however, with total coronary occlusion we induced marked depression of regional myocardial function. The present study did not investigate whether more subtle changes in myocardial function can be assessed by the Doppler-derived strain method. For the potential clinical application of the method, this is an important question, in particular for the application of Doppler-derived strain in the assessment of myocardial function during stress echo.

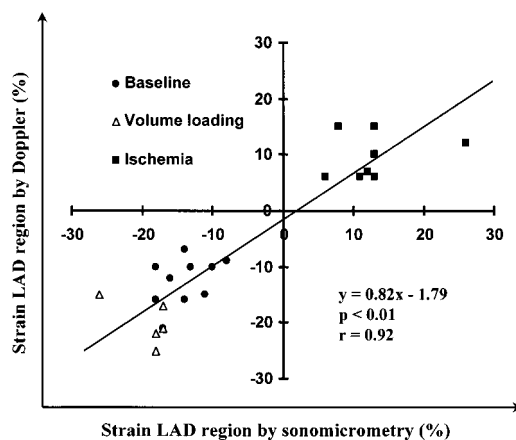


Figure 6. Correlation between systolic strain determined by Doppler and by sonomicrometry.

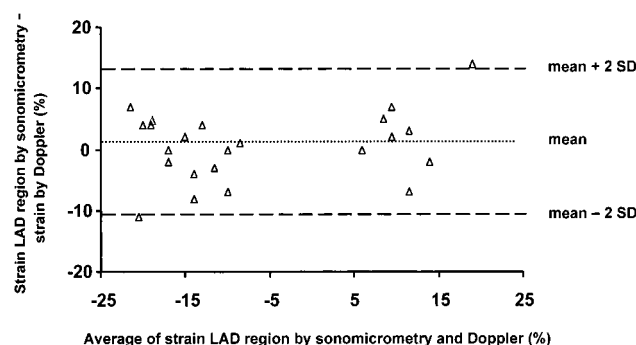


Figure 7. Agreement between systolic strain derived by Doppler and sonomicrometry. Mean difference between methods and ± 2 SD are indicated.

Volume loading markedly increased peak systolic strain rate and strain measured by Doppler. As indicated by the unchanged peak systolic elastance, this could not be attributed to increased LV contractility. This is in keeping with fundamental principles of cardiac mechanics and implies that Doppler-derived strain and strain rate are load-sensitive. Because the primary clinical potential of Doppler-derived strain may be to demonstrate regional differences in function rather than to serve as a marker of global contractility, this limitation may be less important.

Myocardial Strain and Strain Rate Versus Tissue Velocities

Myocardial velocity is a function of the local rate of deformation, and therefore, myocardial velocities and strain rate to some extent provide similar diagnostic information. This was clearly seen during LAD occlusion, in that strain rate, strain, and tissue velocities all indicated dyskinesia of the ischemic segment. However, regional myocardial velocities are also generated by tethering effects from other myocardial segments and by translational motion of the entire heart, and this could limit the ability of tissue Doppler imaging to quantify regional function. Such a limitation was suggested by Yamada et al,¹⁸ who studied myocardial velocity responses to dobutamine in patients with coronary artery disease compared with normal subjects. They demonstrated reduced velocity responses to dobutamine in nonischemic segments in the coronary patients. In the present study, myocardial velocities in the nonischemic basal portion of the LV decreased during apical ischemia, whereas regional strain and regional velocity of shortening (dL/dt) by sonomicrometry were unchanged. Therefore, the decrease in basal tissue velocities during apical ischemia could not be attributed to a decrease in function in the basal segment. Most likely, the reduction in basal myocardial velocities during LAD occlusion reflects loss of the apical contribution to long-axis shortening. The Doppler-derived strain and strain rate values at the base were also unchanged and were therefore consistent with the sonomicrometry data. Taken together, these results suggest that Doppler-derived strain and strain rate are more direct measures of regional function than tissue velocities, which are also influenced by contractile function of other myocardial regions due to tethering. This could limit the ability of

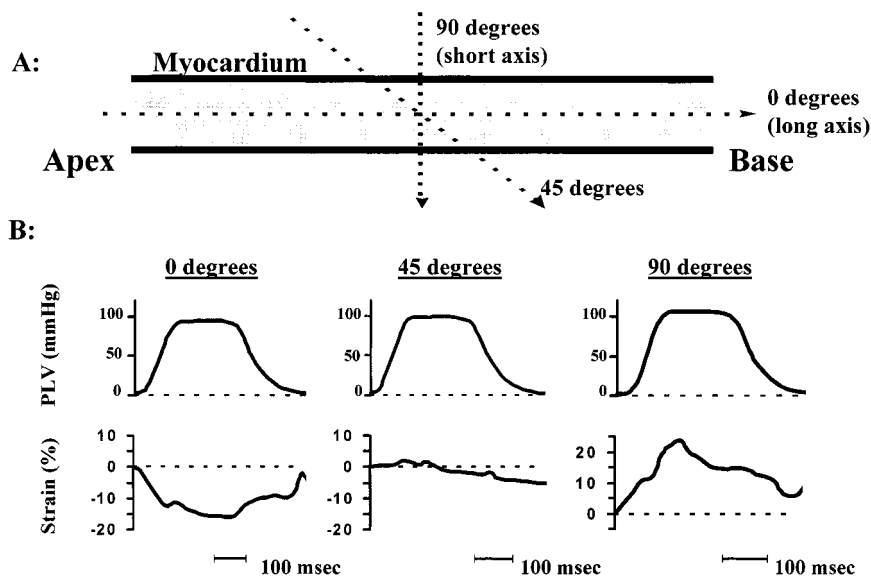


Figure 8. A, Schematic of myocardial segment in which ultrasound beam (dotted lines) was rotated from 0° (long axis) to 45° and 90° (short axis), while strain was measured in same region. B, Representative dog showing changes in systolic strain by Doppler when orientation of ultrasound beam was changed. When ultrasonic probe was rotated from an angle of ≈0° to ≈45° and then 90°, measured strain changed from negative to positive. PLV indicates LV pressure.

tissue Doppler velocities to provide quantitative data on regional function.

Because strain in principle measures the extent of shortening and strain rate, the velocity of shortening, the 2 approaches may provide complementary diagnostic information. More work is needed to establish how each of the 2 modalities may contribute to the noninvasive assessment of LV function. In our study, however, strain rate was more noisy than strain, and with the present version of the strain rate program, this appeared to be an important limitation. This problem is illustrated in Figure 2, where the strain rate, although mostly negative during systole, has spikes with positive values throughout systole. The present study did not investigate the diagnostic potential of the imaging part of Doppler-derived strain rate. It remains possible that qualitative assessment of regional strain rate may be possible as part of the imaging by Doppler echocardiography. However, more work needs to be done to determine whether gross regional differences in strain rate can be reliably assessed from color-coded images.

Limitations

With the present algorithm, the strain rate and strain were calculated directly from a spatial region of ≈5 mm, and this sets the limit for the spatial resolution. This was a compromise between demands for high spatial resolution and for high signal-to-noise ratio. When a smaller distance is used for calculating the Doppler velocity gradient, the random noise becomes relatively larger. To reduce the noise, we averaged multiple samples from a 10-mm-long segment along the M-mode beam. Improved methods for filtering may open for better spatial resolution.

A significant limitation of strain rate imaging is marked angle dependency, more so than for other Doppler modalities. This is due to the fundamental difference between measurement of fluid velocities, where particles move freely, and tissue velocities in solid structures, where deformation in one direction is always associated with deformations in other directions to keep the mass of the structure constant. The

angle dependency was demonstrated in the present study by showing directionally opposite strain values when the angle between the ultrasonic beam and LV axis was off by >45°. In the LV short-axis view, the intact myocardium shows thickening in systole and thinning in diastole. The positive systolic strain rate reflects the faster movement of inner than outer myocardial layers in systole. Negative strain rates in long-axis views reflect that the basal portion normally moves faster than the apex. As indicated in Figure 8, there is an intermediate angle of ≈45° where the present method measures strains close to zero and therefore does not reflect myocardial function. Understanding of this limitation and correct orientation of the echo beam is critical for the application of this imaging modality.

With the present method, we cannot claim to have alignment with any particular fiber orientation. We measured only net shortening of the tissue between the crystals and therefore cannot confirm that we have demonstrated the ability to directly measure fiber shortening. Furthermore, because of cardiac motion and the lack of fixed reference points in the myocardium, we could not maintain identical sampling points throughout the cardiac cycle. In this regard, the Doppler method is inferior to MR tissue tagging.

In conclusion, measurement of myocardial strain by Doppler echocardiography may represent a new, powerful method for assessing regional myocardial function, which appears to be less influenced by tethering effects than Doppler tissue imaging. Further studies are needed to determine whether this approach will be clinically useful.

Appendix

This appendix describes the concept of strain and strain rate and how these entities can be measured by ultrasound. In general terms, strain (ϵ) means relative deformation, and strain rate (SR) means rate of deformation.¹⁹ If an object has an initial length L_0 that after a certain time changes to L , strain is defined as

$$(1) \quad \epsilon = \frac{L - L_0}{L_0}.$$

The instantaneous change in length (dL) in a small time increment (dt) is related to the velocities (v_1 and v_2) of the end points of the object:

$$(2) \quad dL = (v_2 - v_1)dt.$$

By dividing Equation 2 by L , we see that the instantaneous change in length per unit length equals the velocity gradient (ie, strain rate) times the time increment:

$$(3) \quad \frac{dL}{L} = \frac{v_2 - v_1}{L} dt.$$

Because it is not feasible to track the end points of the object, the velocity gradient SR is estimated from 2 points with a fixed distance:

$$(4) \quad \frac{dL}{L} \approx \frac{v(r) - v(r + \Delta r)}{\Delta r} dt = SR \, dt.$$

Finally, by integrating this equation from time t_0 to t , we arrive at

$$(5) \quad \log \frac{L}{L_0} = \int_{t_0}^t SR \, dt,$$

where \log denotes the natural logarithm and L_0 and L denote the object length at times t_0 and t , respectively. This gives the following relation between strain rate and strain:

$$(6) \quad \varepsilon = \exp \left(\int_{t_0}^t SR \, dt \right) - 1.$$

Velocity gradients (strain rates) in the direction of the ultrasonic beam can be estimated from the spatial variation in Doppler shift frequency of the received signal. For calculation of strain, we integrated (Equation 5) strain rate throughout each cardiac cycle, starting at peak R wave on the ECG. Because strain rate equals velocity (m/s) divided by distance (m), the units are $1/s$, and strain, which is the time integral (with units $1/s \times s$), is reported as a fraction or percentage of end-diastolic dimension.

Strain rates were calculated with ≈ 5 -mm offset between the 2 sampling points (Δr in Equation 4). To get a more robust estimate, spatial averaging along the beam, as well as between neighboring beams, was performed with a prototype GE Vingmed algorithm. As a compromise between demands for low noise level and good spatial resolution, we calculated the average of ≈ 15 samples from a region that extended 10 mm along the M-mode line and < 5 mm laterally.

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