

Quantification of Left Ventricular Systolic Function by Tissue Doppler Echocardiography

Added Value of Measuring Pre- and Postejction Velocities in Ischemic Myocardium

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Background—Tissue Doppler imaging (TDI) is a potentially powerful method for diagnosing myocardial ischemia. This study was designed to investigate how velocity patterns in ischemic myocardium relates to regional function, and to determine whether timing of velocity measurements relative to ejection and isovolumic phases may increase the diagnostic power of TDI.

Methods and Results—In 17 open-chest anesthetized dogs we measured pressures by micromanometers, myocardial longitudinal segment lengths by sonomicrometry, and velocities by TDI. Myocardial longitudinal strain rate was calculated as velocity divided by distance to the left ventricle apex. Moderate ischemia (left anterior descending coronary artery stenosis) caused parallel reductions in regional systolic shortening by sonomicrometry ($P<0.05$) and in peak systolic velocities by TDI ($P<0.05$). Severe ischemia (left anterior descending coronary artery occlusion), however, induced systolic lengthening by sonomicrometry ($P<0.001$), whereas peak TDI velocity during ejection remained positive ($P<0.05$). When velocities during isovolumic contraction (IVC) and isovolumic relaxation (IVR) were included, TDI correlated well with sonomicrometry; ie, systolic lengthening occurred predominantly during IVC and was evident as negative velocities ($r=0.70$, $P<0.001$), and postsystolic shortening during IVR ($r=0.72$, $P<0.001$) as positive velocities. In nonischemic myocardium peak systolic strain rates were more uniform than velocities.

Conclusion—The present results indicate that peak ejection velocity is an inappropriate measure of function in severely ischemic myocardium. Dyskinetic myocardium deforms predominantly during the isovolumic phases, and therefore IVC and IVR velocities are better markers of function. When isovolumic as well as ejection velocities are measured, TDI has excellent ability to quantify regional myocardial dysfunction. Longitudinal strain rates are more uniform than velocities and may further improve the diagnostic power of TDI. (*Circulation*. 2002;105:2071-2077.)

Key Words: imaging ■ echocardiography ■ ischemia ■ systole

Qualitative and semiquantitative evaluations of myocardial function by 2-dimensional and M-mode echocardiography have long been the most important noninvasive methods for diagnosing coronary artery disease. Tissue Doppler imaging (TDI) has been introduced as a method to quantify myocardial function in terms of tissue velocities, and the results so far are promising.¹⁻⁷ Ischemic regions are characterized by a decrease in systolic velocities at rest or during stress echocardiography.⁸⁻¹⁰

The clinical implementation of TDI, however, has been relatively slow, and most echocardiographic laboratories do not apply TDI as a routine diagnostic method. This may in part be attributed to a lack of established criteria for how to analyze and interpret the TDI velocity trace, which reflects the relatively limited insight into the etiology of the different

velocity components. This study was designed to determine how TDI velocity patterns in ischemic myocardium relate to regional myocardial function as measured by sonomicrometry and to determine which TDI velocity components best reflect the impairment of systolic function in ischemic myocardium. In particular, we wanted to determine whether timing of myocardial velocities relative to left ventricle (LV) ejection and isovolumic phases could increase the diagnostic power of TDI. We also investigated how velocity patterns of ischemic myocardium were altered as compared with those induced by changes in cardiac loading conditions, inotropy, and heart rate.

A limitation of TDI as a quantitative marker of myocardial function is regional heterogeneity of velocities in normal myocardium, in particular when measuring longitudinal ve-

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locities. The same limitation applies to other modalities for measuring myocardial velocity of shortening. In an attempt to establish a measure that was more specific for myocardial properties, Quinones et al¹¹ introduced the principle of calculating myocardial strain rate from echocardiographic data. This is analogous to the approach proposed by Mirsky et al¹² to calculate strain rate or "normalized velocities" from apex cardiographic tracings. A similar approach was tested in the present study by dividing the longitudinal TDI velocities by the distance from the point of measurement to the apex.

We used an animal model in which TDI velocity patterns were related to myocardial function as defined by implanted ultrasonic dimension gauges and LV micromanometer. For timing of the various phases of the cardiac cycle, we used simultaneous measurement of left atrial, aortic, and LV pressures. As models of moderate ischemia with hypokinesis and severe ischemia with dyskinesis, we used left anterior descending coronary artery (LAD) stenosis and LAD occlusion, respectively.

Methods

Animal Preparation

Seventeen mongrel dogs of either gender weighing 24.3 ± 1.3 kg were anesthetized by thiopental sodium (25 mg/kg body weight) and 100 mg IV of morphine, followed by infusion of morphine 50 to 100 mg/h IV and pentobarbital 50 mg IV every 1.5 hours. The animals were artificially ventilated.

After a median sternotomy and pericardial split, a pneumatic constrictor and an ultrasonic transit-time flow probe were placed around the proximal LAD. The edges of the pericardial incision were loosely resutured.

The study was approved by the National Animal Experimentation Board of Norway.

Instrumentation and Measurements

Micromanometer-tipped catheters and a fluid-filled catheter for zero reference were placed in the aortic arch, left atrium (LA), and LV.¹³ Regional myocardial function was measured by sonomicrometry. A pair of ultrasonic crystals was implanted with longitudinal orientation in the inner half of the anterior LV wall, in the perfusion territory of the LAD.¹³

Echocardiographic images in digital format were recorded from apical 2-chamber (n=17) and short-axis (n=7) views placed directly over the LV apex and anterior wall with a thick layer of gel interposed (GE Vingmed System Five, GE Medical Systems). The average frame rate was 86 ± 3 frames/s.

Experimental Protocol

Hemodynamic data were digitized at a rate of 200 samples/s (CVSOFT, Odessa Computers). Because of acoustic interference, recordings of myocardial segment lengths by sonomicrometry and echocardiography were performed separately within 5 seconds. Recordings were done with the dogs in the supine position and with the ventilator off.

Interventions in the Nonischemic Ventricle

In 7 animals preload was elevated by a rapid intravenous infusion with isotonic saline, raising LV end-diastolic pressure from 7.9 ± 1.7 to 17.6 ± 1.4 mm Hg. Enhancement of inotropy was obtained in 5 animals by IV isoproterenol ($0.15 \mu\text{g}/\text{kg}$ per minute) for ≈ 10 minutes, and LV dP/dt_{max} increased from 1465 ± 160 to 3207 ± 369 mm Hg/s and heart rate from 93 ± 9 to 124 ± 8 beats/min. In 6 animals we infused IV epinephrine ($10 \mu\text{g}/\text{mL}$) for 1 to 3 minutes, and LV dP/dt_{max} increased from 1449 ± 198 to 4507 ± 1029 mm Hg/sec and heart rate from 101 ± 8 to 113 ± 10 beats/min.

Induction of Ischemia

Moderate ischemia was induced in one group of 7 animals by reducing LAD flow by 30% to 40% and maintained for 2 to 4 minutes. Severe ischemia was studied in another group of 10 animals by occluding the LAD for 5 minutes.

Data Analysis

Pressures

The following pressures were calculated: peak-systolic LV pressure (PLV), end-diastolic PLV, and maximum and minimum time derivatives of PLV (dP/dt_{max} and dP/dt_{min}). Left atrial pressure was measured at first diastolic crossover with PLV. The isovolumic contraction (IVC) was defined from peak R on ECG to LV and aortic pressure crossover, and the isovolumic relaxation (IVR) was defined from dP/dt_{min} to first LA and LV pressure crossover.

Sonomicrometry

Percentage of systolic shortening was calculated as end-diastolic dimension minus minimum systolic dimension in percentage of end-diastolic dimension.

Echocardiography

A commercially available image processing and analyzing program (Echopac, GE Medical Systems) was used. Myocardial longitudinal velocity vectors were displayed as color-coded images superimposed on the 2-dimensional gray-scale echocardiographic images in real-time display. Images were recorded using an apical 2-chamber and short-axis view with imaging planes as close as possible to the ultrasonic crystals (Figure 1). The LV wall was divided into 6 segments (base, middle, and apical segments along the anterior and posterior walls). For each region the trace represents the mean values of the instantaneous velocity spectrum.

Velocities were measured during the different phases of the cardiac cycle as defined by the pressure measurements (Figure 2). During IVC and IVR, the deflections with the largest absolute velocity (V_{IVC} and V_{IVR} , respectively) were measured. During the ejection phase, peak early-ejection (V_{S1}) and mid-ejection velocities (V_{S2}) were determined. As an approximation of longitudinal myocardial strain rate, we used myocardial velocity divided by the distance from the point of measurement to the LV apex.¹¹

Statistical Analysis

Data are presented as mean \pm SEM. Comparisons were analyzed by paired Student *t* tests and when appropriate by ANOVA and the Bonferroni post hoc test. Relationship between parameters was determined using the Pearson coefficient of correlation. Probability values <0.05 were considered significant.

Results

Table 1 presents Doppler velocity data from the different myocardial segments. The ultrasonic crystals were placed in the midanterior segment. Therefore, we will refer to velocity data from this segment in the figures and in the subsequent text. Myocardial velocity traces from the short axis showed in principle the same patterns as measured from the long axis (Table 2).

Baseline

Isovolumic Contraction

Myocardial velocities were dominantly positive in all segments during IVC (Figure 2, Table 1). Sonomicrometry confirmed net shortening of the myocardial segment.

Ejection

Myocardial systolic velocity profiles were essentially similar in all regions along the anterior and posterior wall of the nonischemic ventricle. The peak ejection and mid-ejection

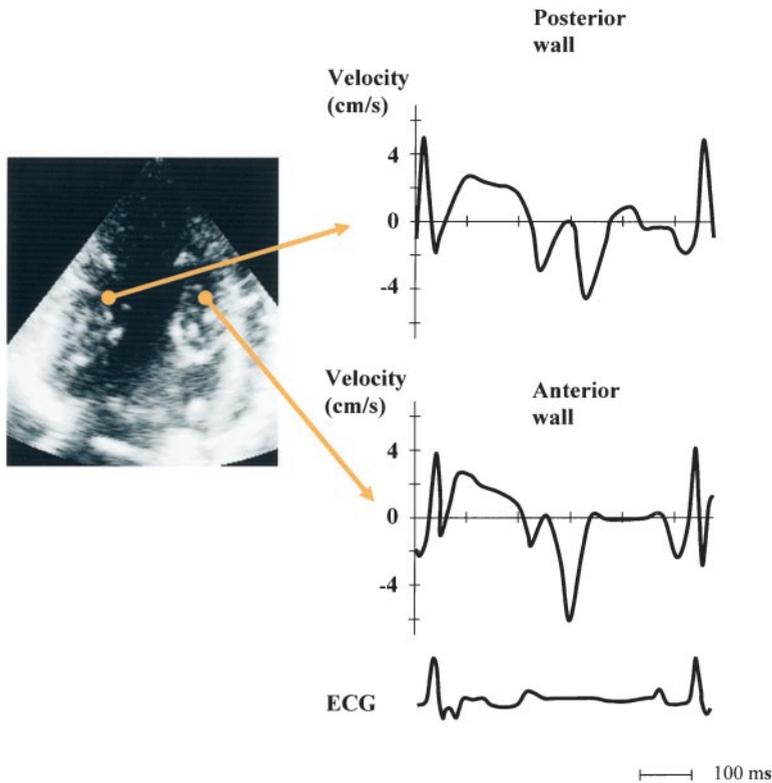


Figure 1. Velocity traces from different optional locations could be generated from the 2-dimensional loop. Upper right panel shows a velocity trace from the posterior wall; lower right panel, trace from the anterior wall.

velocities increased progressively from apex toward base ($P < 0.05$). Peak ejection strain rates, however, were essentially similar between the LV base and apex (Table 1). There were no significant differences between measurements of any velocity or strain rate variables measured from the anterior and posterior walls at comparable distances from the apex.

Isovolumic Relaxation

During IVR, the myocardial velocity pattern was dominated by a negative velocity spike (Figure 2, Table 1), indicating net elongation of the ventricle. There were no significant differences between V_{IVR} in the anterior and the posterior walls.

Ischemia

Isovolumic Contraction

Myocardial IVC velocities decreased and in some cases slightly reversed during moderate ischemia from 4.1 ± 0.6 to -0.3 ± 1.4 cm/s ($P < 0.05$). The sonomicrometry data showed a decrease in shortening during IVC from $4.0 \pm 1.2\%$ to $1.8 \pm 1.7\%$ (NS).

With onset of severe ischemia, myocardial IVC velocities decreased from 3.7 ± 0.5 to -4.5 ± 0.7 cm/s ($P < 0.001$) (Table 2). This was consistent with sonomicrometry, which showed a decrease in IVC shortening from $4.3 \pm 0.6\%$ to $-8.0 \pm 1.8\%$ ($P < 0.001$), indicating myocardial lengthening during IVC. Myocardial velocities during IVC correlated well with sonomicrometry ($r = 0.70$, $P < 0.001$) (Figure 3).

Ejection

After induction of LAD stenosis, there was a decrease in ejection velocities V_{S1} and V_{S2} in the anterior wall, and sonomicrometry showed a decrease in systolic shortening from $13.9 \pm 1.7\%$ to $7.5 \pm 1.6\%$ ($P < 0.05$).

After LAD occlusion, the mid-ejection velocity (V_{S2}) reversed. Consistent with the negative V_{S2} , sonomicrometry demonstrated systolic lengthening of the ischemic segment (Figure 2 and Table 3), $15.4 \pm 3.5\%$ to $-10.3 \pm 2.0\%$ ($P < 0.001$) at baseline and LAD occlusion, respectively. However, most of this systolic lengthening ($79 \pm 13\%$) occurred during IVC. V_{S1} , however, remained positive even during LAD occlusion in 9 out of 10 animals. Strain rates showed in principle the same changes as absolute velocities, and there was a good correlation with sonomicrometry ($r = 0.70$, $P = 0.001$).

Ejection velocities in the nonischemic posterior wall showed no significant change during anterior wall ischemia. Furthermore, in each animal the V_{S2} was lower in the ischemic segment than in the corresponding nonischemic segment.

Isovolumic Relaxation

The IVR velocity of ischemic myocardium remained dominantly negative during LAD stenosis. During LAD occlusion, however, the IVR velocity became dominantly positive with a peak value of 2.2 ± 0.2 cm/s ($P < 0.01$) (Figure 2 and Table 2), indicating postsystolic shortening. According to measurements by sonomicrometry, $76 \pm 10\%$ of the postsystolic shortening occurred during IVR. Postsystolic shortening by sonomicrometry correlated well with IVR velocities by TDI ($r = 0.72$, $P < 0.001$). As demonstrated in Figure 4, during LAD occlusion the pressure-segment length loop indicated myocardial lengthening predominantly during IVC and shortening during IVR.

In the nonischemic posterior wall segments, V_{IVR} remained unchanged. When comparing the ischemic and nonischemic segments from the same heartbeat, the V_{IVR} was higher in the ischemic wall segments in every animal ($P < 0.001$).

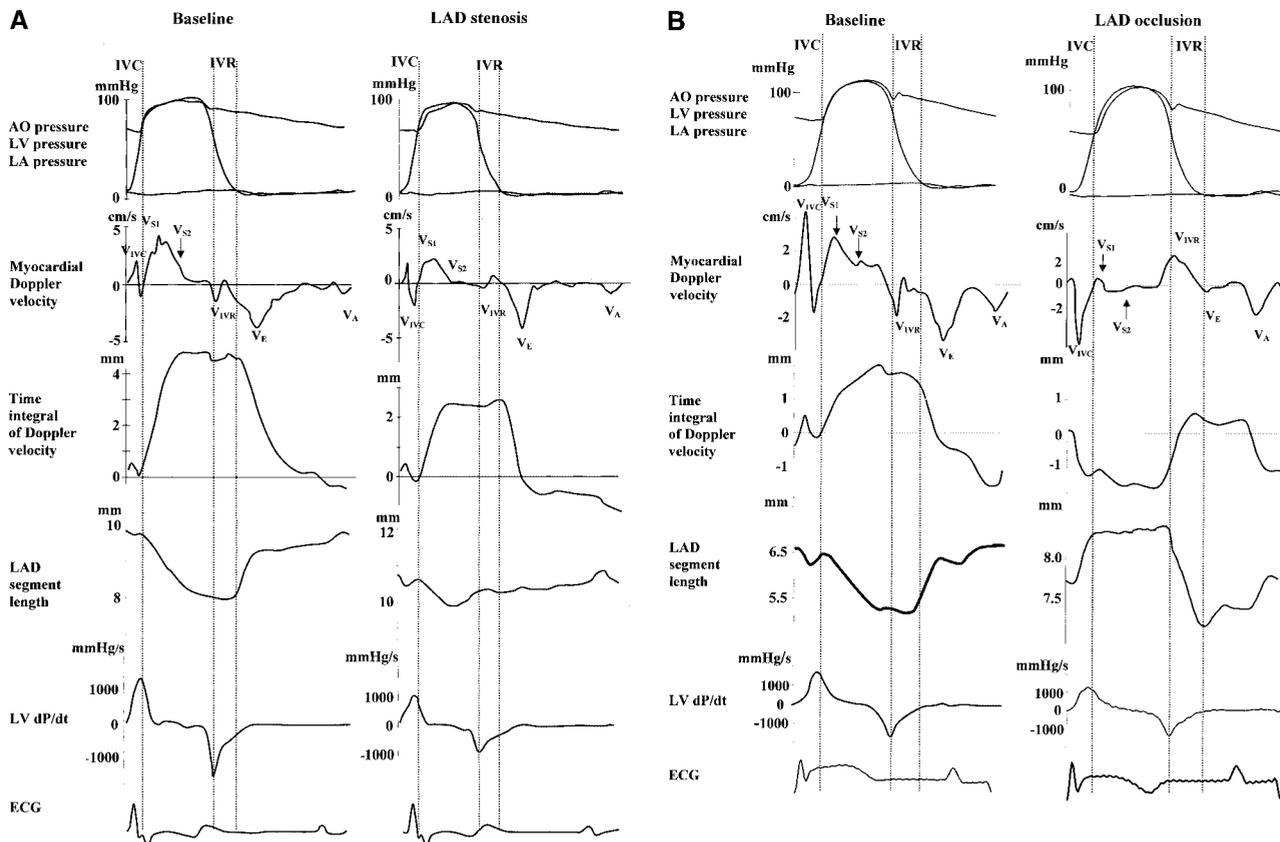


Figure 2. Representative traces at baseline and LAD stenosis (A, group with moderate ischemia) and baseline and LAD occlusion (B, group with severe ischemia), showing myocardial Doppler velocities along with other hemodynamic variables and ECG. V_{S1} indicates peak velocity during early systole; V_{S2} , tissue velocity during midsystole; V_{IVC} , peak velocity during IVC; V_{IVR} , peak velocity during IVR; V_E , early diastolic velocity; V_A , late diastolic velocity; and AO pressure, aortic pressure.

Responses in the Nonischemic Myocardium to Changes in Loading Conditions and to Catecholamines

The systolic velocities increased during epinephrine and isoprenaline infusion ($P < 0.05$), whereas no significant changes occurred during intravenous volume loading. During these interventions, changes in V_{S1} correlated well with systolic shortening by sonomicrometry ($r = 0.68$; $P < 0.01$). There were small but statistically nonsignificant changes in

V_{IVR} (Figure 5) and V_{IVC} during these interventions, except for a significant increase in V_{IVC} from 3.9 ± 0.5 to 6.2 ± 1.2 cm/s ($P = 0.04$) during isoprenaline infusion.

Discussion

A number of studies have confirmed the diagnostic potential of TDI in the assessment of regional myocardial function.^{1,2,4,14-17} As demonstrated in animal models and in

TABLE 1. Myocardial Doppler Velocities and Strain Rates in the LV Anterior Wall (Apical 2-Chamber) (n=10)

	Baseline			P	LAD Occlusion			P
	Base	Mid	Apex		Base	Mid	Apex	
Velocities, cm/s								
V_{IVC}	4.1±0.5	3.7±0.5	3.2±0.4	NS	-3.8±0.6‡	-4.5±0.7‡	-3.9±0.5‡	NS
V_{S1}	3.9±0.5	3.4±0.5	2.4±0.4	<0.001	2.7±0.4*	1.2±0.5‡	0.8±0.3‡	<0.001
V_{S2}	1.5±0.3	1.1±0.2	0.7±0.3	<0.05	0.5±0.3	-0.3±0.3‡	-0.6±0.1‡	<0.05
V_{IVR}	-1.9±0.4	-1.8±0.4	-1.2±0.3	<0.05	1.6±0.3‡	2.2±0.2‡	1.8±0.3‡	NS
Strain rates, 1/s								
SR_{IVC}	1.2±0.1	1.4±0.2	1.8±0.2	NS	-1.1±0.2‡	-1.7±0.3‡	-1.9±0.2‡	<0.05
SR_{S1}	1.2±0.2	1.3±0.2	1.3±0.2	NS	0.8±0.1	0.4±0.2‡	0.4±0.2‡	NS
SR_{S2}	0.4±0.1	0.4±0.1	0.4±0.1	NS	0.1±0.1*	-0.1±0.1‡	-0.3±0.1‡	<0.01
SR_{IVR}	-0.6±0.1	-0.7±0.2	-0.7±0.2	NS	0.4±0.1‡	0.8±0.1‡	0.9±0.2‡	<0.05

SR indicates strain rate; NS, not significant.
* $P < 0.05$ vs baseline values; † $P < 0.01$ vs baseline values; ‡ $P < 0.001$ vs baseline values.

TABLE 2. Peak Myocardial Velocities

Measure	Anterior Mid-Segment 2-Chamber View (n=10)		Posterior Mid-Segment 2-Chamber View (n=10)		Anterior Short-Axis View (n=7)	
	Baseline	LAD Occlusion	Baseline	LAD Occlusion	Baseline	LAD Occlusion
V_{IVC}	3.7±0.5	-4.5±0.7‡	3.5±0.5	3.7±1.0	-2.9±0.3	1.9±0.2‡
V_{S1}	3.4±0.5	1.2±0.5*	3.5±0.8	2.8±0.5	-4.1±0.6	-1.8±0.3*
V_{S2}	1.1±0.2	-0.3±0.3†	1.0±0.3	0.9±0.4	-1.3±0.2	0.9±0.4‡
V_{IVR}	-1.8±0.4	2.2±0.2†	-1.5±0.5	-1.2±0.7	1.7±0.4	-1.9±0.4‡

* $P < 0.05$ vs baseline values; † $P < 0.01$ vs baseline values; ‡ $P < 0.001$ vs baseline values.

patients with coronary artery disease, myocardial ischemia is characterized by a decrease in peak systolic myocardial velocity, indicating impairment of regional contractile function.^{3,9,10} The present study, however, demonstrates important limitations in the ability of peak systolic velocity to serve as a quantitative marker of regional function. Furthermore, we demonstrate how a more comprehensive analysis of the myocardial Doppler velocity signal may improve the ability of TDI to identify ischemic myocardium. This analysis includes measurement of myocardial velocities during IVC and IVR in addition to ejection velocities. Our results also

suggest that myocardial longitudinal strain rate calculated as regional velocity divided by distance to the apex provides a measure that is more specific for myocardial properties.

Systolic Velocities

Myocardial Doppler velocities correlated well with systolic shortening as measured by sonomicrometry, confirming that TDI velocities reflect regional myocardial motion. This is consistent with the observations of Derumeaux et al.¹⁰ Exact timing of myocardial Doppler velocities in relation to the different phases of the cardiac cycle was possible by simultaneous recording of ECG and pressure crossovers for the aortic and mitral valves. In the nonischemic ventricle the IVC period was dominated by a positive velocity spike of short duration, which represented slight longitudinal shortening before LV ejection. With onset of ejection, myocardial velocities accelerated rapidly, and peak velocity was reached during early systole.

During IVR there was a pattern opposite to that during IVC, with a negative velocity spike of short duration, representing slight elongation before onset of filling. Possibly, twisting and untwisting effects may have contributed to the IVC and IVR velocities.^{18,19}

The most striking change during moderate ischemia was a decrease in peak early ejection and mid-ejection velocities and was attributed to a decrease in systolic shortening as measured by sonomicrometry. The ability of ejection velocity to serve as a quantitative marker of regional function was confirmed in the nonischemic ventricle under widely different loading conditions and inotropic states and during moderate ischemia.

During severe ischemia, sonomicrometry showed systolic lengthening during IVC and during ejection, indicating dyskinesis. Contrary to this, early-ejection Doppler velocities remained positive and therefore did not reflect the marked impairment of myocardial function. The mechanism of the positive early-ejection velocity in dyskinetic myocardium is not clear, but it might represent cardiac translational motion or tethering effects resulting from contractions in other myocardial segments.

The dominant systolic velocity component during severe ischemia was a large negative velocity spike during IVC, and as confirmed by sonomicrometry it represented systolic lengthening. Furthermore, as previously reported, the mid-ejection velocity was negative and indicated dyskinesis.⁹ However, the absolute magnitude of the mid-ejection velocity was very small, on average only 7% of peak IVC velocity, and this makes it less practical as a diagnostic marker.

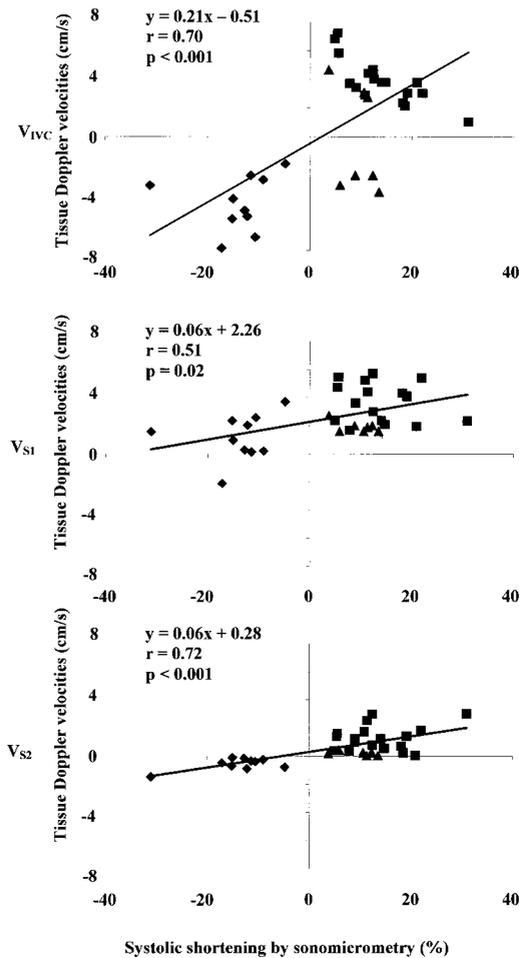


Figure 3. Regression between tissue Doppler velocities in different systolic phases and systolic shortening by sonomicrometry. Abbreviations as in Figure 2. ■ indicates baseline; ▲, LAD stenosis; and ♦, LAD occlusion.

TABLE 3. Hemodynamic and Sonomicrometry Measurements

	LAD Stenosis (n=7)		LAD Occlusion (n=10)	
	Baseline	LAD Stenosis	Baseline	LAD Occlusion
Heart rate, bpm	97±8	93±9	104±8	108±6
PLV _{max} , mm Hg	113±14	105±10	105±5	102±3
LV end-diastolic pressure, mm Hg	8.2±0.3	7.9±0.6	6.8±0.9	8.0±1.1
LV dP/dt _{max} , mm Hg/s	1741±208	1455±137	1804±136	1514±112*
PLA	7.6±0.6	7.8±0.7	6.4±1.6	7.6±0.7
SS-LAD, %	14±2	7±1*	15±4	-10±2†

Values are mean±SEM. PLA indicates pressure LA at first crossover with LV pressure; SS-LAD, systolic shortening of apical segment by sonomicrometry.

* $P<0.05$; † $P<0.001$.

To explain this dominance of IVC velocities as compared with ejection velocities, one may consider the myocardial pressure-segment length loop. During LAD occlusion, the ischemic segment was deforming predominantly during the isovolumic phases; ie, lengthening during IVC and shortening during IVR, indicating that the segment behaved like a net passive structure. The rate of lengthening during IVC and hence myocardial velocities are determined by the rate of rise

of LV pressure and by myocardial (and pericardial) elastic properties. The rate of rise of LV pressure (dP/dt) reaches a maximum during IVC and is less during ejection. In addition, because of the curvilinear shape of the myocardial pressure-segment length relation (Figure 4, right lower panel), there is less lengthening for a given pressure increment during ejection ($dl/dP=0.010\pm0.001$ mm/mm Hg) than during IVC ($dl/dP=0.014\pm0.002$ mm/mm Hg, $P=0.02$). Furthermore, most of the LV systolic pressure generation occurs during IVC. These mechanical principles explain why IVC velocity signals were larger and appeared more promising than ejection velocities as markers of severely ischemic myocardium.

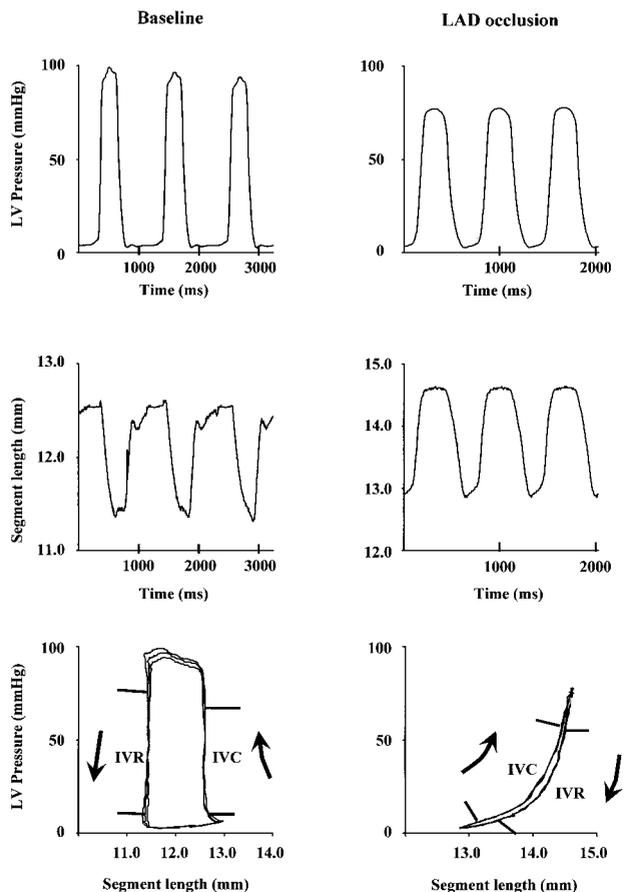


Figure 4. Left ventricular pressure and segment lengths and pressure-segment lengths loops at baseline (left) and during LAD occlusion (right). Note that during ischemia the pressure-segment length loop rotated in the direction opposite of that during baseline. Note also that during ischemia the changes in LV segment length occurred predominantly during IVC and IVR.

Strain Rates

The heterogeneous distribution of myocardial velocities is a serious limitation of the TDI method. As an alternative approach we “normalized” the velocities by calculating longitudinal strain rate as the mean velocity gradient between the point of measurement and the LV apex.¹¹ This measure showed more homogenous distribution throughout different regions in nonischemic myocardium, and therefore may represent a more characteristic measure of myocardial function than velocities. In the present study, the validity of this approach was supported by the demonstration of a good correlation between the strain rates and systolic function by sonomicrometry. Because this method averages strain rates over the entire length between the point of measurement and the apex, the ability to precisely localize the diseased region

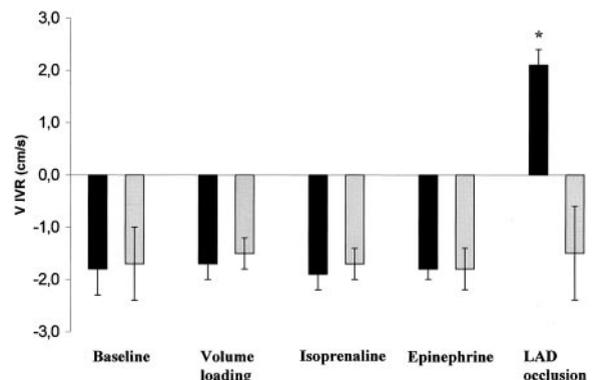


Figure 5. V_{IVR} from anterior (solid columns) and posterior (open columns) LV wall during different interventions. * $P<0.001$.

may not be as good as the method introduced by Heimdal et al²⁰ to calculate regional velocity gradients. However, the noise problem with the latter method is significant²⁰ and may be less with the present approach, which averages strain rates over a longer distance. Further studies should be done to compare the two approaches for calculating strain rate.

IVR Velocities

During severe ischemia, IVR velocities reversed and a large positive velocity component persisted throughout the entire IVR period, and in some cases continued after the early-diastolic LA/LV pressure crossover. As indicated by the LV pressure-segment length analysis, the positive IVR velocity represented postsystolic shortening of ischemic myocardium.

During a wide variety of interventions, which caused changes in LV loading and inotropy, there were only modest changes in the IVR velocities, and the magnitude of the positive velocities did not approach those found during severe ischemia. These findings suggest that a large positive IVR velocity could be a marker of dyskinesia along with the demonstration of negative IVC velocities.

Limitations

In the present study, standard 2-chamber views were used and no attempt was made to correct for angle dependency of TDI measurements. However, for important features in the temporal velocity pattern such as velocity reversal, small angle deviations are less important. Certainly, the measurements of absolute velocities are angle dependent, and one should be aware of this limitation.

The problems with cardiac translation and rotation are inherent in all TDI techniques. Also important, these motions are probably different in an open-chest model compared with humans who have not undergone surgery. The measurement of strain rate rather than velocity may resolve some of the problems related to translation and tethering effects from neighboring segments. Although some of the hemodynamic features of this model are different from those in patients with acute myocardial ischemia, we believe the model is useful for studying fundamental principles of cardiac mechanics.

Conclusions

The present findings indicate that the diagnostic power of TDI may be improved by measuring isovolumic as well as ejection velocities. In actively contracting myocardium, the peak systolic velocity measured during LV ejection reflected changes in myocardial function as measured by sonomicrometry. In severely ischemic and dyskinetic myocardium, however, IVC and IVR velocities were the strongest markers of myocardial dysfunction. Myocardial ejection velocities had very low amplitudes and appeared to be influenced by tethering effects and/or translational motion.

Timing of TDI velocities in relation to the different cardiac phases should be possible by simultaneous display of myocardial velocities, ECG, and aortic and mitral valve signals. As suggested by the present study, this would make TDI an even more powerful method for quantifying regional myocardial function. Longitudinal strain rates are more uniform than velocities, and measurement of this variable may further

improve the diagnostic abilities of TDI. Clinical studies are needed to determine the feasibility of these approaches and to investigate whether the present findings are valid for patients with coronary artery disease.

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