

The Analysis of Left Ventricular Wall Thickness and Shear by an Ultrasonic Triangulation Technique in the Dog

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SUMMARY We developed a new ultrasonic triangulation technique to measure regional wall thickness and shear motion of the left ventricular wall under normal conditions and during complete and partial coronary occlusions. In 10 open-chest dogs, a left ventricular micromanometer was inserted, and a screw-driven arterial clamp and flowmeter were placed around the left circumflex coronary artery. A single miniature ultrasonic crystal was placed in the subendocardium of the left ventricular free wall in a region to be made ischemic, and three receiving crystals were sutured to the opposing epicardium at the corners of a 45-degree right triangle. By the trigonometric combination of three measured lengths from ultrasonic dimension gauges, the exact position of the endocardial crystal in X, Y, and Z coordinates could be displayed by computer. The endocardial surface moved in a longitudinal direction relative to the epicardial surface during normal systole [0.58 ± 0.26 (mean \pm SEM) mm]. With coronary occlusion, this myocardial shear became less marked (0.11 ± 0.12 mm), and systolic wall thickening changed to thinning. When coronary occlusion was released, end-diastolic wall thickness and percent wall thickening rapidly increased to 104.8% and 141.7% of control, respectively, concurrent with the reactive hyperemia. With partial coronary constriction, these postreperfusion changes were markedly reduced, suggesting a role for reactive hyperemia in postreperfusion wall thickness dynamics. These techniques allow measurement of shear and three-dimensional display of dimensions and thereby permit documentation of true wall thickness dynamics both during and after coronary occlusion. *Circ Res* 47: 173-181, 1980

THE regional nature of myocardial dysfunction associated with regional ischemia has been recognized since the original description of aneurysmal bulging in the area distal to an experimental coronary occlusion by Tennant and Wiggers (1935). Recently, ultrasonic crystals implanted in the left ventricular subendocardium have been used to characterize regional contractile dynamics simultaneously in several segments of the left ventricle in open-chest (Theroux et al., 1974) and chronically instrumented, unanesthetized dogs (Theroux et al., 1976a). Reduction of contractile function in the ischemic region after acute coronary occlusion usually takes place within 10 seconds, before ST segment elevation occurs on the epicardial electrocardiogram, and a holosystolic expansion develops over the subsequent 1 to 2 minutes (Theroux et al., 1974).

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Left ventricular wall thickness changes during the cardiac cycle have been measured using the metal clips attached to the wall and high-speed cineangiography (Heikkila et al., 1972), or using a strain gauge (Goldstein and Jong, 1974). The miniature ultrasonic dimension gauges also have proven to be highly suitable for the analysis of wall thickness dynamics (Sasayama et al., 1976a). However, the previous measurement of wall thickness by a single pair of ultrasonic crystals is inherently unidimensional; thus, lateral movement of the thickness crystals could be caused by shearing motion between epicardial and endocardial surfaces of the chamber wall and would be indistinguishable from radial motion. Use of a three-dimensional triangulation technique should allow measurement of true wall thickness independently of shear motion of the left ventricular chamber wall, as well as the degree of shear itself. We have employed such an ultrasonic method to study left ventricular wall thickness dynamics during and after coronary occlusion, as well as the post-reperfusion dynamics of myocardium with special reference to reactive hyperemia in the presence and absence of partial coronary stenosis.

Methods

Instrumentation

Ten mongrel dogs weighing 16-22 kg were anesthetized with sodium pentobarbital (25-30 mg/kg,

iv), and small supplemental doses were administered as required. Ventilation was provided by a Harvard pump delivering room air via an endotracheal tube. A thoracotomy was performed in the 5th intercostal space and the heart was suspended in a pericardial cradle.

The left circumflex coronary artery was dissected free distal to its site of origin, and an electromagnetic flow probe (NARCO, RT-500) and screw-driven arterial clamp were positioned around the vessel. In the region to be rendered ischemic after coronary occlusion, one miniature ultrasonic crystal was inserted subendocardially. The three receiving crystals, cemented at the corners of a right triangle made of polystyrene, were sutured to the opposing epicardial surface, where each signal between the endo- and epicardial crystal was optimal (Fig. 1). The crystals on the legs of the right angle were 10 mm apart. The subendocardial crystal was inserted tangentially through the myocardium, which allowed placement of the crystals across the left ventricular wall without damage to the intervening tissue. An additional pair of crystals was implanted subendocardially 10 mm apart in a circumferential plane adjacent to the triangulation crystals. A high-fidelity micromanometer (KONIGSBERG, P-22) and a short catheter were introduced to the left ventricular chamber through a stab wound at the apex. The latter was connected to a Statham P23Db strain gauge and used for zero reference and calibration of the micromanometer.

Data were recorded during the experiment on an

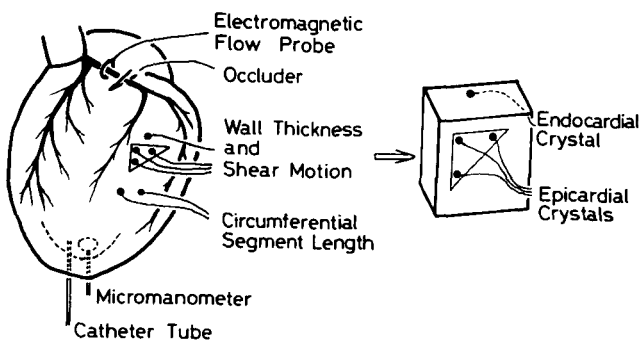


FIGURE 1 Schematic representation of the experimental preparation in the dog. The left circumflex coronary artery was dissected free and an electromagnetic flow probe and screw-driven arterial clamp were positioned around the vessel. In the ischemic region, one miniature ultrasonic crystal was inserted subendocardially and, on the opposing epicardial surface, three crystals were cemented to the corners of a 45° right triangle, as shown in the right panel. From these four crystals, wall thickness and shear motion could be obtained using the triangulation technique. Another pair of crystals was inserted close to the endocardium to measure circumferential segment shortening. A high-fidelity micromanometer was inserted in the left ventricular chamber through the apex.

eight-channel forced-ink oscillograph (SAN-EL, model 142-8) and also stored on magnetic tape (TEAC, R-260). The taped data were later played back and fed to a minicomputer system (DEC, PDP11/40) through an analog-to-digital converter.

After control recordings during the resting state, the left circumflex coronary artery was occluded briefly with a Schwartz arterial clamp. The occlusion then was released and the heart allowed to recover completely. The left circumflex coronary artery then was constricted partially by a screw-driven metal clamp to reduce the mean coronary blood flow by approximately 34%. After a stable state of hypoperfusion had been established, the left circumflex coronary artery was occluded in the same manner as in the control state.

The data were obtained before and 2 minutes after coronary occlusion, and at four stages (30 seconds, 1, 5, and 30 minutes) after reperfusion.

In each condition, segment length shortening during systole was calculated as follows: segment shortening = $[(\text{end-diastolic length} - \text{end-systolic length}) / (\text{end-diastolic length})] \times 100$.

The values then were normalized so that control values were always 100%: normalized segment shortening = $[(\text{shortening in a given condition}) / (\text{shortening during control condition})] \times 100$.

The calculations of wall thickening during systole were performed in a similar manner and normalized to the control condition (termed normalized wall thickening) in the same way. End-diastolic segment length and end-diastolic wall thickness, as well as mean coronary blood flow, also are presented as value normalized with respect to the control value.

Calculations of Wall Thickness and Shear Motion

Analog signals played back from the magnetic tapes were digitized at a sampling rate of 200 Hz and a resolution of 12 bits, and fed to the minicomputer to calculate the true wall thickness and the shear motion of the myocardium. The results then were displayed on a cathode ray tube or printed out on the terminal typewriter.

The principle of the calculation is illustrated in Figure 2.

Distances were measured from the single subendocardial crystal to each of the three epicardial crystals oriented in a fixed triangular array overlying the endocardial crystal and sutured to the epicardium. The three measured lengths between the respective crystals and the known dimensions of the fixed triangle are combined trigonometrically to determine the position of the endocardial crystal in X, Y, and Z coordinates relative to the reference epicardial plane. If line BC deviated from the longitudinal axis (drawn from the apex to the point of bifurcation of the left main coronary artery into its anterior descending and circumflex branches), the angle was corrected trigonometrically.

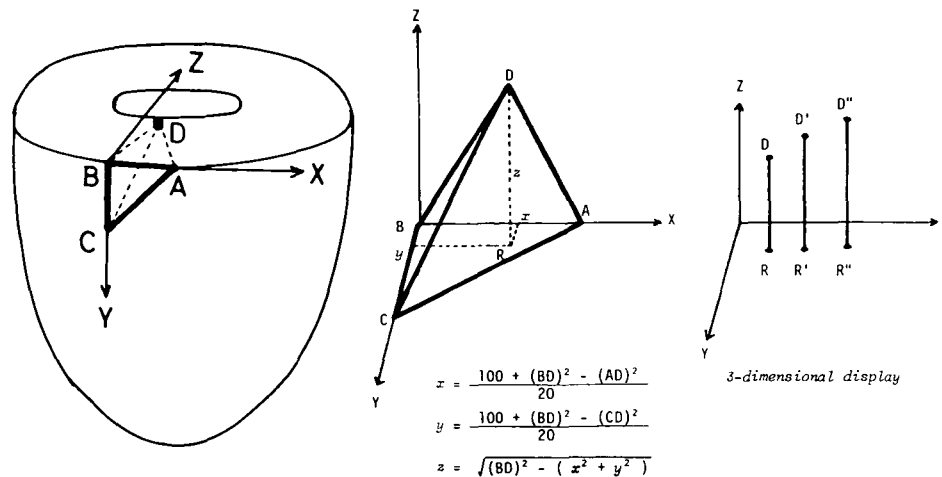


FIGURE 2 Schema of the triangulation technique. The left panel shows the notation of the crystals and axes. Of the three epicardial crystals, the one at the right angle is referred to as B, the one at the leg of right triangle in the circumferential direction as A, and the one at the leg in the longitudinal direction as C. The endocardial crystal is referred to as D. The X axis is set to the line BA, and the Y axis to the line BC. The Z axis is set to the line at point B perpendicular to the X and the Y axes. The middle panel explains the calculation of wall thickness and shear motion, the Z axis being inverted in relation to the left panel. The point R is the projection of point D on the reference epicardial plane, and the length of the line DR, that is, the Z coordinate of the point D, indicates the true wall thickness independent of the shear motion of the myocardium. The changes on the X and Y coordinates of point D express shearing movement during the cardiac cycle. These X, Y, and Z coordinates are calculated using the equations shown in the panel from the three lengths BD, AD, and CD, which are measured simultaneously by the ultrasonic dimension gauge. The right panel shows the type of instantaneous three-dimensional display of the wall thickness and the shear motion (expressed by the displacement of the perpendicular lines, DR, D'R', D''R'', ...) throughout the cardiac cycle. In this display, the true wall thickness is expressed by the length of the line, and the shear motion by the varying lateral displacement of the line.

The Three-Dimensional Display of the Wall Thickness and the Shear Motion

The displacement of the perpendicular line DR in the middle panel of Figure 2 was displayed on the cathode ray tube continuously during the cardiac cycle, to give a three-dimensional presentation of the wall thickness and shear motion as shown in the right panel of Figure 2. In this display, the true wall thickness is expressed by the changes of the length of the line, and the shear motion by the lateral displacement of the line (i.e., DR to D'R' or D''R'', ...).

Statistical Analysis

The data comparing serial changes after reperfusion were analyzed by Dunnett's test (Zar, 1974), a *P* value of <0.05 being considered significant. Comparisons were made between each resting value and those at 2 minutes after coronary occlusion, and at 30 seconds, 1, 5, and 30 minutes after reperfusion. Statistical comparisons between groups of dogs subjected to reperfusion and to reperfusion during partial coronary constriction were made using an unpaired *t*-test (Zar, 1974). All results are expressed as mean \pm SD unless otherwise stated.

Results

Representative direct recordings from which the data were derived and computed are shown in Figure 3 and illustrate the effect of a brief coronary occlusion. An example of computer-processed data during the control state which was displayed on the cathode ray tube, is shown in Figure 4. The time intervals of 5 msec correspond to the sampling rate of the analog-to-digital converter.

The effects of complete coronary occlusion and partial coronary constriction are summarized in Table 1 A and 1 B, respectively, and illustrated on Figures 5 and 6.

Wall Thickness Changes in the Resting State

In the control state, the average end-diastolic wall thickness by the triangulation technique was 9.6 ± 2.6 mm and the wall thickening during systole was $20.3 \pm 10.5\%$ of end-diastolic wall thickness before normalization to control conditions. Segment shortening during systole was $16.8 \pm 3.1\%$ of the end-diastolic length before normalization to control conditions.

TABLE 1 *Reperfusion with Intact Coronary Artery and during Partial Coronary Constriction*

Variables	Control	Coronary occlusion at 2 min	Reperfusion at			
			30 sec	1 min	5 min	30 min
A. With intact coronary artery (n = 8)						
Heart rate (beats/min)	144.9 ± 21.5	149.1 ± 20.9	145.6 ± 21.6	145.6 ± 21.9	146.3 ± 20.4	145.8 ± 21.3
P*		<0.05	NS	NS	NS	NS
Peak LVP (mm Hg)	105.0 ± 18.0	95.6 ± 16.0	107.8 ± 14.9	107.6 ± 15.0	104.7 ± 15.7	105.2 ± 14.0
P		<0.01	NS	NS	NS	NS
LVEDP (mm Hg)	8.6 ± 4.8	10.8 ± 7.5	9.7 ± 4.9	10.0 ± 5.8	8.9 ± 5.4	8.0 ± 4.3
P		NS	NS	NS	NS	NS
Norm mean cor blood flow (%)	100	0	333.6 ± 95.6	273.6 ± 71.2	102.2 ± 14.1	95.4 ± 18.2
P		<0.01	<0.01	<0.01	NS	NS
Norm end-diast seg- ment length (%)	100	117.3 ± 7.5	100.2 ± 2.4	99.4 ± 1.9	100.1 ± 1.6	100.8 ± 1.4
P		<0.01	NS	NS	NS	NS
Norm seg short (%)	100	-8.0 ± 10.2	125.1 ± 17.9	124.0 ± 18.4	105.7 ± 8.5	98.2 ± 9.2
P		<0.01	<0.01	<0.01	NS	NS
Norm end-diast wall thickness (%)	100	87.0 ± 5.6	104.8 ± 3.7	104.7 ± 2.1	99.3 ± 2.8	99.5 ± 2.1
P		<0.01	<0.01	<0.01	NS	NS
Norm wall thickening (%)	100	-25.2 ± 28.2	126.7 ± 16.6	141.7 ± 31.8	124.9 ± 28.0	96.0 ± 4.8
P		<0.01	<0.01	<0.01	<0.01	NS

Table 1 continues

Effect of Complete Coronary Occlusion and Reperfusion on Segmental Function

During coronary occlusion, end-diastolic segment length was increased by 17% and normalized segment shortening was changed to systolic elongation, being -8.0% of control. At 30 seconds after reperfusion, end-diastolic segment length returned to the control value and the normalized segment shortening was increased by 25.1% over control. This overshoot was observed by 1 minute after reperfusion; however, it was no longer apparent 55 and 30 minutes after reperfusion.

Effect of Complete Coronary Occlusion and Reperfusion on Wall Thickness Dynamics

During coronary occlusions of 2 minutes, the end-diastolic wall thickness decreased to 87.0% of control and there was systolic thinning (-25.2% of the control value, left panel, Fig. 6).

At 30 seconds after the release of coronary occlusion, end-diastolic wall thickness significantly increased by 4.8% over control in association with an increase in the normalized wall thickening of 26.7% over control value. At 1 minute after release, the normalized wall thickening was increased over control by 41.7%. These changes paralleled the reactive

hyperemic responses in the mean coronary blood flow (left panel, Fig. 5). At 30 minutes after reperfusion, no significant changes were observed in the wall thickness dynamics or coronary blood flow when compared to control.

Effect of Partial Coronary Constriction and Reperfusion on Wall Thickness Dynamics

When the mean coronary blood flow was reduced by 34% of control (control value 44.6 ml/minutes; right panel, Fig. 5) segmental function showed hypokinesia, the normalized shortening being reduced to 92.9% and normalized wall thickening to 60.7% of control, respectively. Segment length and wall thickness at end-diastole showed no significant changes (right panel, Fig. 6).

After the release of coronary occlusion during partial coronary constriction, reactive hyperemia was significantly lower than in the control reperfusion, and the overshoot in segmental function and wall thickness dynamics was not observed (right panel, Fig. 6).

Myocardial Shear Motion

A three-dimensional display of shear motion and wall thickness is shown in Figure 7. The endocardial

Table 1 continued

Variables	Partially stenosed	Coronary occlusion at 2 min	Reperfusion at			
			30 sec	1 min	5 min	30 min
<i>B. During partial coronary constriction (n = 6)</i>						
Heart rate (beats/min)	153.0 ± 20.3	156.3 ± 18.8	151.9 ± 18.8	151.4 ± 19.9	151.1 ± 20.1	154.6 ± 23.2
<i>P</i> *		<0.05	NS	NS	NS	NS
<i>P</i> †	NS	NS	NS	NS	NS	NS
Peak LVP (mm Hg)	107.8 ± 12.4	100.0 ± 9.5	108.7 ± 8.5	109.3 ± 11.3	108.1 ± 10.8	110.5 ± 13.6
<i>P</i>		<0.01	NS	NS	NS	NS
<i>P</i>	NS	NS	NS	NS	NS	NS
LVEDP (mm Hg)	8.5 ± 4.4	8.5 ± 4.4	9.1 ± 4.8	7.7 ± 3.2	8.1 ± 4.2	8.6 ± 2.9
<i>P</i>		NS	NS	NS	NS	NS
<i>P</i>	NS	NS	NS	NS	NS	NS
Norm mean cor blood flow (%)	65.8 ± 12.8	0	131.0 ± 77.7	105.5 ± 60.9	72.4 ± 24.1	62.8 ± 16.4
<i>P</i>		<0.01	<0.01	<0.05	NS	NS
<i>P</i>	<0.001	NS	<0.001	<0.001	<0.02	<0.01
Norm end-diast seg- ment length (%)	103.4 ± 5.9	118.1 ± 9.0	107.7 ± 11.8	105.4 ± 12.6	103.0 ± 6.2	104.9 ± 7.4
<i>P</i>		<0.01	NS	NS	NS	NS
<i>P</i>	NS	NS	NS	NS	NS	NS
Norm seg short (%)	92.9 ± 7.9	-9.5 ± 14.0	91.8 ± 34.0	95.6 ± 37.1	93.1 ± 13.6	95.4 ± 10.7
<i>P</i>		<0.01	NS	NS	NS	NS
<i>P</i>	<0.05	NS	<0.05	NS	NS	NS
Norm end-diast wall thickness (%)	98.3 ± 5.8	90.1 ± 5.0	98.7 ± 5.2	100.0 ± 6.4	98.4 ± 5.2	98.3 ± 5.5
<i>P</i>		<0.01	NS	NS	NS	NS
<i>P</i>	NS	NS	<0.05	NS	NS	NS
Norm wall thickening (%)	60.7 ± 27.5	-3.8 ± 52.2	62.0 ± 60.0	74.8 ± 53.5	71.8 ± 28.0	72.9 ± 30.3
<i>P</i>		<0.01	NS	NS	NS	NS
<i>P</i>	<0.01	NS	<0.02	<0.02	<0.01	NS

All values are mean ± SD. Abbreviations are as follows: LVP, left ventricular pressure; LVEDP, left ventricular end-diastolic pressure; Norm mean cor blood flow, normalized mean coronary blood flow; Norm end-diast segment length, normalized end-diastolic segment length; Norm seg short, normalized segment shortening during systole; Norm end-diast wall thickness, normalized end-diastolic wall thickness; Norm wall thickening, normalized wall thickening during systole; NS, not significant.

* Each *P* = statistically significant difference compared with the values before each coronary occlusion.

† Each *P* = statistically significant difference between control perfusion and reperfusion under partial coronary constriction.

surface moved predominantly in a longitudinal direction relative to the reference epicardial surface during systole, accompanied by the wall thickening (left panel). With coronary occlusion (right panel), the left ventricular wall showed systolic thinning, and myocardial shear between epicardium and endocardium became much less marked, being directed predominantly in the circumferential plane.

The average magnitude of myocardial shear during systole in the control state was 0.58 ± 0.26 mm (mean ± SEM) in the direction of longitudinal axis of the left ventricle (ΔY), which was significantly greater than the magnitude in the circumferential direction (ΔX), 0.08 ± 0.13 mm (Table 2).

With complete coronary occlusion, ΔY dimin-

ished to 0.11 ± 0.12 mm, and ΔX increased slightly to 0.09 ± 0.17 mm.

Discussion

Myocardial Shear and Wall Thickness

The ultrasonic dimension gauge used in the present study has proved useful in assessing subendocardial segmental shortening (Theroux et al., 1974, 1976b; Sasayama et al., 1976b) and wall thickness changes (Ross and Franklin, 1976; Sasayama et al., 1976a). For the measurement of wall thickness, the technique employs two miniature ultrasonic transducers, one embedded within the subendocardium and one placed on the epicardial surface, the sepa-

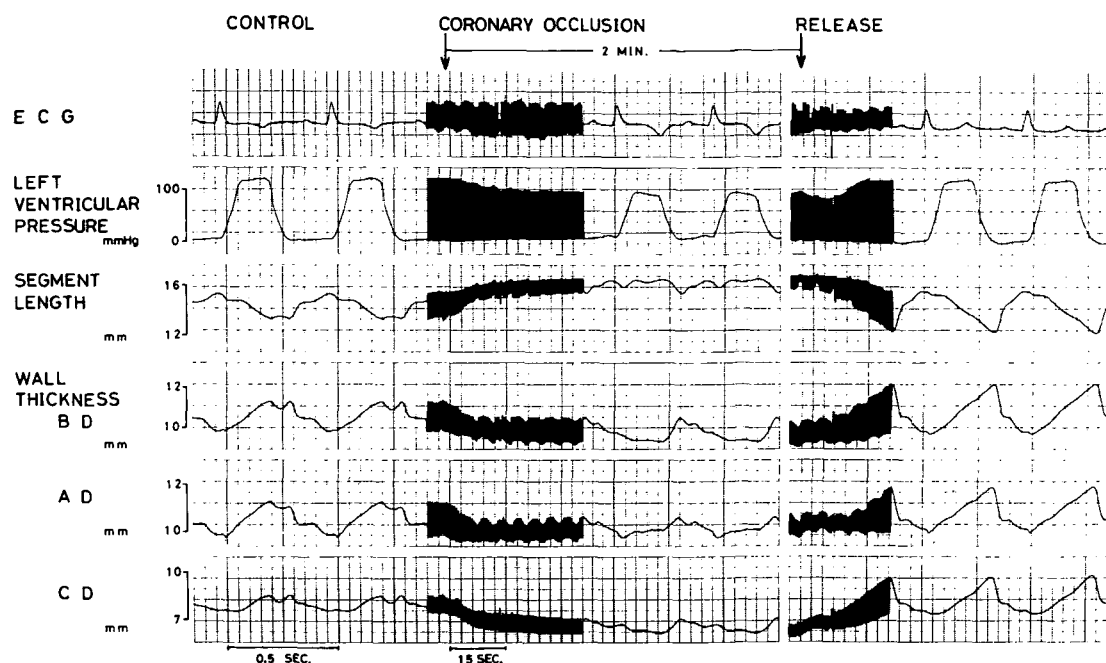


FIGURE 3 Representative original recording from which the data were derived. The electrocardiogram, left ventricular pressure, a segment length, and three lengths, BD, AD, and CD (notation same as in Fig. 2) used for computation of wall thickness were recorded simultaneously and stored on magnetic tape for further processing by computer. In the control state, the three lengths (BD, AD, CD) increased during systole as the wall thickened and the segment length decreased. At the time of coronary occlusion, indicated by the arrow, note the rapid loss of myocardial thickening in the three lengths, leading to systolic thinning, with a reciprocal change in the segment length. Immediately after the release of the occlusion, indicated by the second arrow, systolic thickening is rapidly restored above the control level.

ration of the crystals being measured by the transit time of the sound beam. The approach is inherently unidimensional, so that the translational movements of the crystals could provide a source of measurement error. Accordingly, we developed a technique for measurement of three-dimensional deformation in a small region of myocardium using an ultrasonic triangulation technique that allows measurement of myocardial wall thickness along with relative lateral motion of the inner and outer chamber walls, the shear motion.

Feigl and Fry (1964) studied myocardial shear using a strain gauge, which detected a relatively large shearing motion during the isovolumic and prediastolic portions of systole, whereas that occurring during the ejection period was relatively small. In that study, no significant difference was observed between the magnitude of longitudinal and transverse shear motion. The discrepancy between their findings and the present results may relate to the large and heavy gauge they used for the measurement of shear of a rather small magnitude. Furthermore, myocardial wall thickening might have placed tension on the gauge itself and/or damaged the tissue.

Streeter et al. (1969) studied myocardial fiber orientation across the left ventricular wall, using the sequential sections fixed in both diastole and systole. They postulated shearing strains due to

rotational displacement of adjacent fibers between diastole and systole. Subsequently, they proposed a model for stress analysis of the left ventricular wall and calculated the presence of a shearing stress in the longitudinal direction acting on a plane perpendicular to the circumferential direction (Streeter et al., 1970). According to their data, shear stress predominated near the outer portion of the wall, changing its sign at midwall. The corresponding shear strains constituted rotational displacement only, and not translational displacement of the myocardial fibers. In contrast, the shear motion measured in the present study was due mainly to translational movement of the fibers between inner and outer walls.

The phasic changes in wall thickness during the cardiac cycle observed in the present study were quite consistent with those described in the conscious animal using a pair of transducers (Sasayama et al., 1976a), although the magnitude of thickening during ejection was less in the anesthetized state. The lateral movement of the thickness crystals caused by shearing motion of the wall is relatively small (Table 2). In conventional measurements of wall thickness, we can estimate the possible error caused by shear motion by assuming that, at end-diastole, a unidimensional pair of crystals is positioned in the perpendicular line DR, as in Figure 2, whereas, at end-systole, the endocardial crystal D

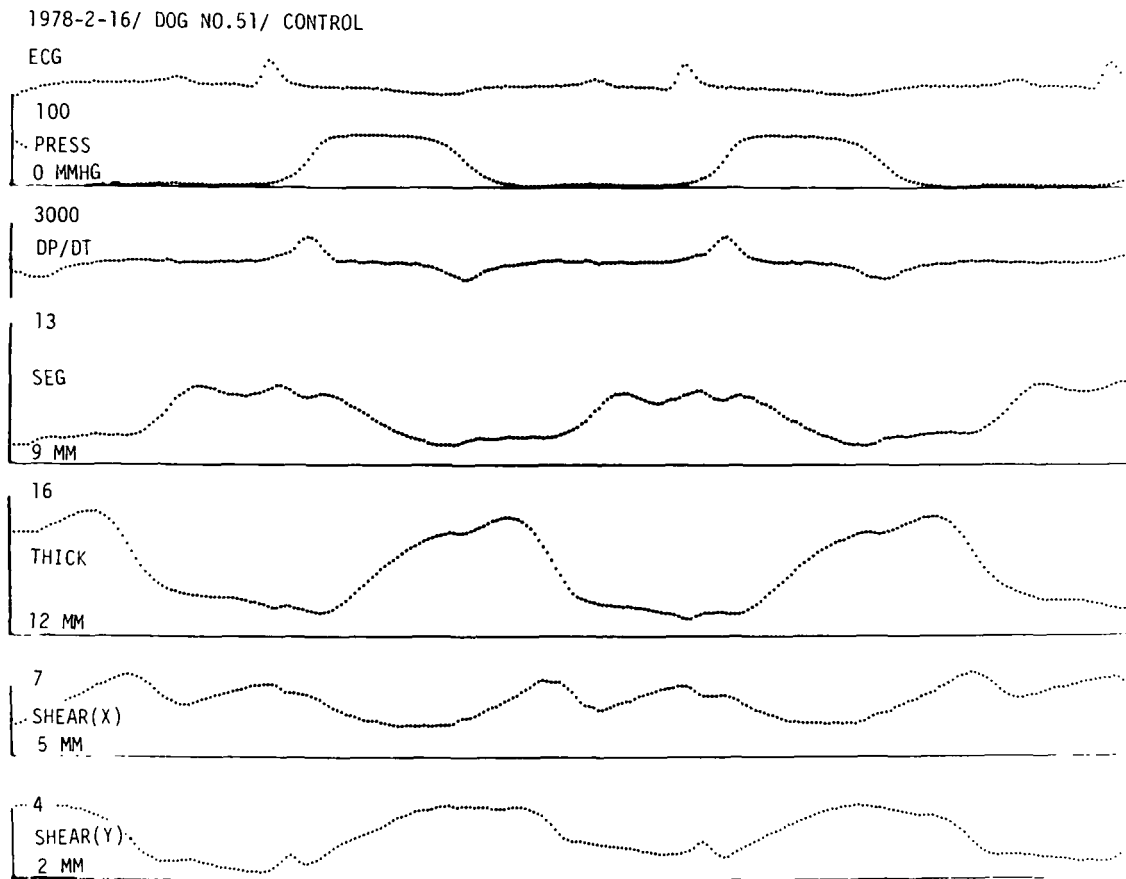


FIGURE 4 Example of computer-processed data in the control state. Data are displayed on cathode ray tube at every 5 msec corresponding with the sampling rate of A-D conversion. The traces are, from top to bottom, ECG, left ventricular pressure (PRESS), dP/dt (DP/DT), segment length (SEG), true wall thickness (THICK), and X and Y coordinates of shear motion [SHEAR (X), SHEAR (Y)].

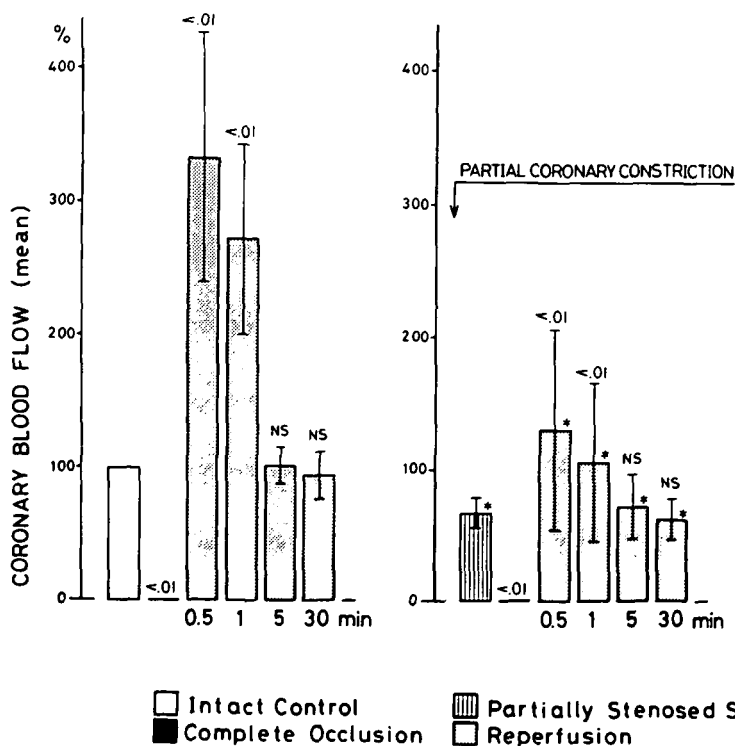


FIGURE 5 *The changes in mean blood flow in the left circumflex coronary artery compared with the control value (44.6 ± 14.6 ml/min.). Statistically significant differences compared with each resting value are indicated at the top of each bar (NS: not significant). Asterisks indicate a significant difference between each value on the control study (left panel) and during partial coronary constriction (right panel). During the control reperfusion, significant reactive hyperemia was observed in the early phase (0.5 and 1 minute). However with partial coronary constriction, control flow was significantly reduced, followed by the significantly less reactive hyperemia.*

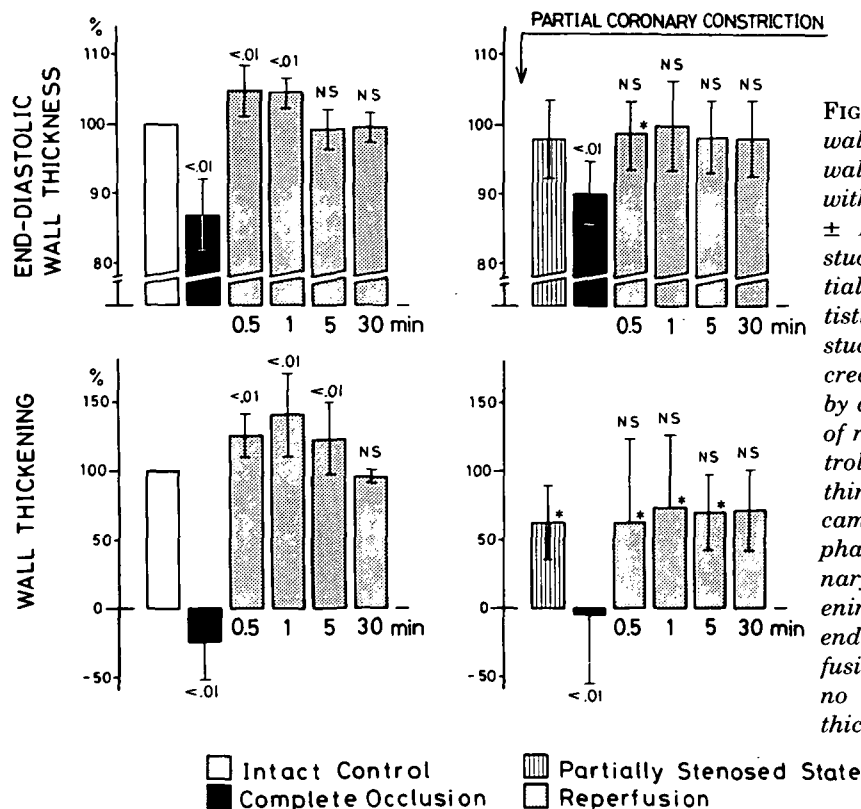


FIGURE 6 The change of end-diastolic wall thickness (upper panels) and systolic wall thickening (lower panels) compared with control values (9.6 ± 2.6 mm and $20.3 \pm 10.5\%$, respectively) during the control study (left panels) and the study during partial coronary constriction (right panels). Statistical notation as in Figure 5. In the control study, end-diastolic wall thickness decreased during coronary occlusion, followed by a significant increase in the early phase of reperfusion, which returned later to control. Systolic wall thickening showed systolic thinning during coronary occlusion and became enhanced over control during the early phase of reperfusion. During partial coronary constriction (right panels), wall thickening became hypokinetic without change in end-diastolic wall thickness. During reperfusion, end-diastolic wall thickness showed no significant change, and systolic wall thickening did not increase significantly.

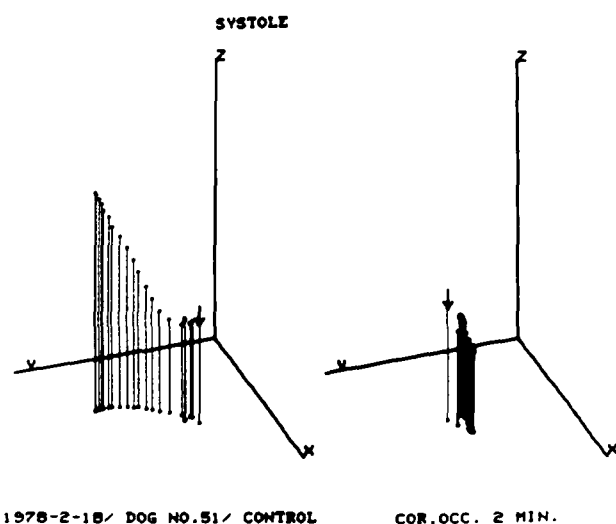


FIGURE 7 A representative three-dimensional display of wall thickness and shear motion every 10 msec during systole before (left panel) and after coronary occlusion (right panel). The height of each vertical bar denotes instantaneous wall thickness on the Z coordinate, and shear motion is expressed by lateral displacement of bar in the longitudinal direction of left ventricle (Y coordinate) and the circumferential direction (X coordinate). The length of each axis is 3 mm, and the beginning of the systole is indicated by the arrow. Shear motion was directed predominantly in the longitudinal axis as wall thickness increased during systole. After coronary occlusion, however, shear motion was much less marked, predominantly in the X-axis, as the wall thinned during systole.

moves by shear motion relative to the epicardial point R. The average estimated error of systolic wall thickening based on our experiments is less than 2%, and therefore undimensional measurement of wall thickness provides reasonable accuracy when proper placement of the thickness crystals is achieved. Furthermore, these shear strains became negligible during coronary occlusion.

The responses of regional function after brief episodes of ischemia are of interest, since they might occur during ordinary episodes of angina pectoris in patients with coronary artery disease.

TABLE 2 Summary of Shear Motion

	Control		Cor. occl. 2'		Rep. 30"	
	ΔX	ΔY	ΔX	ΔY	ΔX	ΔY
<i>Intact coronary artery (n = 8)</i>						
Mean	-0.08	0.58*	-0.09	0.11	-0.24	0.59*
SEM	0.13	0.26	0.17	0.12	0.17	0.29
	Partially stenosed state		Cor. occl. 2'		Rep. 30"	
	ΔX	ΔY	ΔX	ΔY	ΔX	ΔY
<i>Partial coronary constriction (n = 6)</i>						
Mean	-0.40	0.36*	-0.17	0.05	-0.30	0.29
SEM	0.11	0.19	0.12	0.19	0.14	0.26

Abbreviations are as follows: Cor. occl., coronary occlusion; Rep., reperfusion; NS, not significant; ΔX and ΔY , magnitude of shear motion during systole in circumferential and longitudinal direction of left ventricle, respectively (notation is the same as in Fig. 2). Unit: mm.

* $P < 0.05$ between ΔX and ΔY .

Interruptions of coronary blood flow for 5–15 minutes have been shown to be followed by reversal of changes in the epicardial electrogram after a few minutes of reperfusion, but mechanical function returned to normal only after several hours (Heyndrickx et al., 1975). Theroux et al. (1976a) showed that a 2-minute period of regional ischemia was followed by much more rapid recovery after release of the coronary occlusion. Heyndrickx et al. (1978) also used an ultrasonic dimension system to show the effect on myocardial function of reperfusion after 15 minutes of coronary occlusion and concluded that a transient increase in diastolic wall thickness occurred after release of coronary occlusion, but the systolic wall thickening remained significantly depressed for 3 hours along with reduced regional myocardial blood flow.

The present study after short (2-minute) occlusions showed an early post-reperfusion hyperfunction of the myocardium reflected both in segment shortening and wall thickening in parallel with reactive hyperemic response of coronary blood flow. With complete occlusion during partial stenosis, neither reactive hyperemia nor post-reperfusion hyperfunction was evident. Although the mechanism by which reactive hyperemia may induce supernormal function was not elucidated, several possible mechanisms can be eliminated. Changes in preload, afterload, and heart rate did not play a major role, since there were no changes in left ventricular pressure, end-diastolic segment length, and heart rate at the time of the peak rebound. Hyperfunction during reperfusion does not appear to be mediated by adrenergic mechanisms, since large doses of propranolol, or combined propranolol and reserpine, have failed to abolish the overshoot (Pagani et al., 1978). Verapamil also did not prevent the rebound (Pagani et al., 1978).

It is possible that vasodilation itself accounts in some way for post-reperfusion hyperfunction. The increase in the end-diastolic wall thickness is likely to have been caused by an increase in the amount of blood contained in the myocardium during diastole. The fact that systolic shortening was augmented from an unchanged end-diastolic segment length suggests that contractility was enhanced.

In summary, we developed a new ultrasonic triangulation technique to measure wall thickness and shear motion of left ventricular wall. Shear motion was directed in the long axis of left ventricle during systole during normal perfusion, and became unapparent during coronary occlusion. The method was applied to study wall function after release of coronary occlusion, and post-reperfusion hyper-

function of wall thickness dynamics appeared to be dependent upon the associated reactive hyperemia.

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