Assessment of Left Ventricular Contractility from Late Systolic Stress-Volume Relations

Hubert Pouluer, M.D., Michel F. Rousseau, M.D., Christian van Eyll, Henri Van Mechelen, Lucien A. Brasseur, M.D., and André A. Charlier, M.D.

SUMMARY The aim of the study was to determine if the end-systolic stress–end-systolic volume relation of the left ventricle (i.e., its tension-length relation) could be estimated from the analysis of a single ventriculogram. For that purpose, the left ventricular (LV) wall stress and volume were computed frame by frame in 35 patients (15 controls, 11 patients with valvular disease, six with coronary artery disease and three with congestive cardiomyopathy). In all patients, a linear relation was observed between LV wall stress and volume during approximately the last two-thirds of ejection. Under basal conditions, the duration of this linear relation ranged from 100–260 msec (mean 155 msec; mean r = 0.97) with a positive slope (range 3.0–9.3 kdyn/cm²/m³ in controls) and a negative volume intercept. During a positive inotropic intervention (atrial pacing at 120 beats/min) that increased peak positive dP/dt by 24% (p < 0.0002), the slope of this linear relation also increased by 24% (p < 0.0025), but ejection fraction was unchanged. Further studies in animals indicated that this slope was insignificantly affected by changes in LV afterload within a range of 70–200% of the control values. Within the range of LV afterload encountered in the studied patients, this slope remained directly proportional to the slope of the end-systolic stress–end-systolic volume relation. Thus, during LV ejection, there is a linear stress-volume relation, which can be determined easily from a standard ventriculogram. The slope of this relation, which depends directly on the tension-length relation of the ventricle, is therefore more specific than ejection phase indexes in detecting alterations in LV inotropic state in the clinical setting.

ADEQUATE EVALUATION of the contractile state of the ventricle is important in the presence of valvular disease to predict patients at high risk for perioperative cardiac mortality and residual postoperative impairment of ventricular function.1-9 However, because of the alterations in preload and afterload associated with valvular lesions, neither the standard hemodynamic measurements nor many conventional indexes of contractility can provide such an evaluation.9, 10

The linear relation between ventricular wall stress (or pressure) and volume at the end of ejection may be the most reliable index of myocardial contractility.9-18 In animals, the slope of this relation is very sensitive to changes in inotropic state and is unaffected by alterations in preload or afterload.4, 9, 14 In man, the linearity of this relationship9-14 and its value in assessing acute10, 12, 18 or chronic10 changes in myocardial contractility have also been established. Nevertheless, practical problems still limit the clinical use of this index.

First, to obtain enough data points to determine this relation, the heart must be manipulated through a range of volumes or pressures.10, 12, 18 Such hemodynamic interventions and the multiple ventriculograms required may not be safe in patients with valvular lesions. Second, all nervous reflexes should be blocked during these interventions,18 which further complicates these studies. Finally, despite promising results reported with noninvasive techniques,12, 14 the echocardiographic determination of this linear relation remains difficult in various heart diseases because of imprecision in calculating ventricular volumes.17

We sought to determine whether an index of myocardial contractility, directly related to the end-systolic stress–volume relation, could be derived from the analysis of the stress-volume data during left ventricular (LV) ejection. We analyzed single beats from standard left ventriculograms frame by frame. During the last two-thirds of a single LV ejection, the stress-volume data were linearly related and the slope of this relation was sensitive to changes in inotropic state. Additional studies were then needed to test the effects of the changes in ventricular loading on this slope and to determine its relations with the end-systolic stress–end-systolic dimension line. We performed these studies in dogs.

Material and Methods

Patients

Thirty-five patients, mean age 45 years (range 18–59 years), were included in this study. All were in sinus rhythm and underwent diagnostic cardiac catheterization. Clinical and angiographic data are listed in table 1. Fifteen patients who had atypical chest pain and completely normal ventriculograms and coronary arteries were considered as normal control subjects. The other patients included 11 with valvular diseases, six with coronary artery disease (stenosis of 75% or greater in at least one coronary vessel) and three with congestive cardiomyopathy. All cardioactive drugs were discontinued at least 48 hours, in most cases 6–7 days, before the procedure. Each patient gave informed consent and no complication occurred as a result of this study.

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Study Protocol

Left-heart catheterization was performed using the femoral approach with the patient in the fasting state and without premedication. High-fidelity LV pressures were recorded at low (0–160 mm Hg) and high (0–40 mm Hg) gains with #5F or #6F micromanometer-tip catheters (Millar Instruments). The micromanometer system was calibrated against a mercury manometer before insertion and again after withdrawal. The rate of LV pressure change (dP/dt) was obtained by direct differentiation of the LV pressure signal using a differentiating amplifier (Physiocardiopan Philips) that has a linear response up to 60 Hz. Aortic pressure was measured through a #8F pigtail fluid-filled catheter connected to a Statham P23ID strain gauge.

Left ventriculography was performed using single-plane, 35-mm cineangiography at 50 frames/sec in the right anterior oblique projection (Philips Poly-Diagnost C). The hemodynamic variables and a cine frame marker were recorded on a multichannel recording system at a paper speed of 200 mm/sec (Honeywell 1858). The LV pressure during ventriculography and the time of the peak of the R wave were also sampled synchronously with frame exposure and displayed on the corresponding cine frame in digital form (Cine Data Philips).

In 24 patients, a bipolar electrode catheter was also introduced into the right atrium. Atrial pacing was started 10 minutes after the first ventriculography at a heart rate of 110–120 beats/min to produce a positive inotropic stimulation without changing vascular impedance; ventriculography was repeated after 5 minutes of pacing.

Measurements and Computations

To evaluate LV function, ventricular silhouettes were outlined with a light pen on a video screen. Both premature and post premature beats were excluded from analysis. A computer system (LVV Philips 100) derived the correction factor for x-ray magnification and calculated volumes every 20 msec applying Simpson's rule. Wall thickness at the LV equator was traced on the last diastolic frame and was computed for the subsequent frames assuming a constant LV mass. Midwall circumferential stress was calculated by the formula of Sandler and Dodge:

$$\frac{Pb}{H}(1 - \frac{b^2}{a^2(2b + H)}) \text{ kdyn/cm}^2$$

Where \( P \) = LV pressure, \( H \) = wall thickness, \( a \) = midwall semimajor axis and \( b \) = midwall semiminor axis. Ejection fraction and mean velocity of fiber shortening (mean Vcf) were calculated according to standard formulas. Volume data were corrected for the body surface area and end-systolic or end-diastolic volumes were defined as the smallest and largest LV volumes, respectively.

To characterize the tension-length framework of the ventricle from the stress-volume data during ejection, the stress-volume loops of each patient were plotted (figs. 1 and 2). These plots indicated that stress-volume data were linearly related during at least 100 msec before the end of ejection. An algorithm to determine the slope and intercept of this stress-volume line was developed. This algorithm first calculates the pressure/volume ratio at each frame and selects the frame in which this ratio is maximal. This frame corresponds to minimal LV systolic compliance, which is also end-systole as defined by Suga and Sagawa. Starting from this frame and going backward throughout ejection, the stress-volume data are fitted to a linear relation by least-squares regression techniques. Thus, successive relations are obtained, the first fitting the last 100 msec of ejection and the following fitting the last 120, 140, 160 ... msec of ejection. We always selected the slope and intercept of the relation with the highest correlation coefficient to characterize the tension-length relationship of the ventricle during late ejection.

Animal Experiments

For ethical reasons, we could not induce large variations in LV preload and afterload and block nervous reflex in patients. Therefore, we studied the sensitivity of the proposed index of contractility to changes in the LV loading conditions in six mongrel dogs (mean weight 28.1 kg; range 26–33 kg). The dogs were anesthetized with i.v. sodium pentobarbital (25 mg/kg, intravenously) and ventilated by a Harvard respirator.

Instrumentation and Experimental Procedures

After median sternotomy and bilateral thoracotomy, the heart was suspended in a pericardial cradle. A high-fidelity triple micromanometer (Millar Instruments, model PC772) was inserted into the left ventricle through a stab incision at the apex and its tip was advanced into the aortic root. Zero pressure levels were referenced to the middle right atrium and the LV pressures recorded by the Millar micromanometer were adjusted to match the LV pressure trace during diastole recorded by catheter connected to a Statham P23Db strain gauge. One pair of ultrasonic crystals (5-MHz piezoelectric discs, each 5 mm in diameter) was positioned at the posterior and anterior endocardial surfaces. The characteristics and calibration methods of the pressure transducers and of the sonomicrometry system have been reported.

The dogs were placed on right-heart bypass as described previously. Briefly, venous return was permitted to drain into a reservoir, and venous return to the left ventricle was maintained by a peristaltic pump through a cannula in the main pulmonary artery.

Propranolol (1 mg/kg) and atropine (0.5 mg) were given intravenously to avoid reflex changes in inotropic state during the following interventions:

All preparations were set at a venous return sufficient to keep the LV end-diastolic pressure at 6–10 mm Hg and mean aortic pressure at 80–100 mm Hg. To increase the LV afterload, the descending aorta...
was occluded completely below the left subclavian artery for 15 seconds while this level of venous return was kept constant.

When the preparation had returned to the control hemodynamic status after the aortic clamping, the peristaltic pump was stopped for 15 seconds to obtain beat-to-beat reductions in LV preload.

A bolus of calcium gluconate (2 mEq) was injected in five dogs after the two preceding maneuvers and data were recorded at the peak of action.

After completion of this protocol, the heart was arrested by an injection of potassium chloride and the LV long axis (aortic valve to apex) and LV mass was determined.

### Table 1. Clinical and Hemodynamic Data

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<th>LVEDP (mm Hg)</th>
<th>Peak + dP/dt (mm Hg/sec)</th>
<th>LVEF (ml/m²)</th>
<th>LVEDV (ml)</th>
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Mean ± SD: 50.8 ± 5.4, 76.5 ± 12.7, 10.5 ± 3.9, 1956 ± 436, 28 ± 8, 91 ± 15, 70 ± 5, 89 ± 25

Mean ± SD: 38.0 ± 12.6, 73.1 ± 11.1, 15.1 ± 5.0, 1684 ± 506, 60 ± 30, 174 ± 51, 67 ± 8, 162 ± 55

Mean ± SD: 44.3 ± 10.9, 82.7 ± 14.2, 13.5 ± 3.9, 1823 ± 315, 39 ± 7, 102 ± 4, 63 ± 9, 77 ± 25

Mean ± SD: 39.3 ± 18.7, 89.0 ± 15.8, 25.8 ± 8.3, 1172 ± 214, 153 ± 81, 226 ± 71, 35 ± 19, 185 ± 26

Abbreviations: HR = heart rate; LVEDP = left ventricular end-diastolic pressure; LVEF = left ventricular end-systolic volume; LVEDV = left ventricular end-diastolic volume; EF = ejection fraction; LVM = left ventricular mass; Vcf = velocity of fiber shortening; N = normal; MR = mitral regurgitation; MS = mitral stenosis; AR = aortic regurgitation; CAD = coronary artery disease; CMP = congestive cardiomyopathy.
end-systolic wall stress and diameter data were also determined under control conditions and during each intervention; the slope of the linear relation between end-systolic wall stress and end-systolic diameter was calculated using linear least-square regression techniques.14

During each maneuver, the slope and duration of the linear relation between stress and diameter during late ejection were also computed beat by beat by the method used to analyze angiographic data. These slopes were compared by repeated-measures analysis of variance.28

Results

Studies in Patients

The clinical data for all patients are shown in table 1. Typical stress-volume loops are shown in figures 1 and 2.

Stress-Volume Relationship During Late Ejection

LV wall stress and LV volume were linearly related during approximately the last two-thirds of ejection, both under basal conditions and during atrial pacing up to 120 beats/min (fig. 1). Under basal conditions, this linear relationship lasted 100–260 msec (mean 155 msec) with an average correlation coefficient of 0.97 (table 1). The slope was positive in all patients and the volume intercept generally negative.

When the heart rate was increased to 107–121 beats/min in 24 patients, a linear relation between stress and volume was still present for at least 100 msec (mean 130 msec, range 100–200 msec) before the end of systole (mean = 0.981, range 0.911–0.998). Thus, in control subjects and in patients with various heart diseases, late LV ejection could be characterized by the slope of this relation. In the 15 control subjects, the mean slope was 4.87 kdyn/cm²/m² (range 3–9.3 kdyn/cm²/m²). The average intercept was −24 ml/m² (range 0 to −60 ml/m²). No patient with heart disease had a slope above the control range. All the patients with congestive cardiomyopathy and seven patients with valvular disease had a slope less than the lowest control value of 3 kdyn/cm²/m² (table 1).

The volume intercepts were more negative than normal in patients with coronary artery disease (−49 ± 20 vs −24 ± 22 ml/m², p < 0.02). The seven patients with valvular disease and a slope less than 3 kdyn/cm²/m² also had a more negative intercept (−77 ± 143 ml/m², p < 0.04). The volume intercept remained normal in the patients with congestive cardiomyopathy (−23 ± 13 ml/m², NS) and in the other patients with valvular disease. Accordingly, the data were examined to test the sensitivity of this relation to a change in inotropic state and to determine its clinical usefulness.

Effects of Inotropic State

on the Late Stress-Volume Relation

To assess the effects of an enhanced contractile state on the stress-volume relation, we compared the slope and intercepts before and during atrial pacing at
107–121 beats/min. Atrial pacing had significant positive inotropic effects, as it increased peak positive dP/dt by 24% (p < 0.0002) and mean Vcf by 23% (p < 0.003), despite a decrease in LV end-diastolic volume (table 2). Concomitantly, the slope increased and the volume intercept became less negative in 21 patients; in one normal patient and one with valvular disease, the slope and intercept decreased slightly but remained constant in another normal subject. When the whole group was considered, the slope increased 24% (p < 0.0025) and the volume intercept became less negative (table 2). Because pacing had insignificant effects on LV wall stress (a 0.5% increase in peak systolic wall stress and a 9% decrease in end-systolic wall stress, NS), it is unlikely that these slope changes might have been caused by changes in afterload.

Comparison of Slopes with Ejection Fraction, Mean Vcf and End-systolic Volume Index

Ejection fraction, mean Vcf and end-systolic volume index were significantly depressed in the three patients with congestive cardiomyopathy (fig. 3). However, peak systolic wall stress (446 ± 91 vs 375 ± 60 kdyn/cm² in normals, p < 0.05) and end-systolic wall stress (336 ± 72 vs 215 ± 79 kdyn/cm², p < 0.01) were greater than normal in these patients. This increase in afterload might partially explain the alterations in ejection phase indexes.

In contrast, only the slope and the end-systolic volume were abnormal in patients with mitral or aortic regurgitation. In this group, the changes in slope and end-systolic volume cannot be explained by alterations in LV afterload, as neither the peak systolic stress (378 ± 43 vs 375 ± 60 dyn/cm² in normals, NS) nor the end-systolic stress (224 ± 77 vs 215 ± 79 kdyn/cm², NS) were altered.

Among the 11 patients with valvular disease, the four with a normal slope (4.35 ± 0.49 vs 4.87 ± 1.78 kdyn/cm²/m² in controls, NS) also had a nearly normal end-systolic volume index (35 ± 10 vs 28 ± 8 ml/m² in controls, NS). The seven patients with a depressed slope (2.06 ± 0.76, p < 0.001 vs control) also had a markedly increased end-systolic volume index (73 ± 30 ml/m², p < 0.00002). Finally, in patients with coronary artery disease, the mean Vcf and the ejection fraction remained normal despite slight increases in peak wall stress (512 ± 123 kdyn/cm², p < 0.02) and end-systolic wall stress (315 ± 99 kdyn/cm², NS).

Figure 1. Typical stress-volume loops during basal conditions (heart rate 84 beats/min) and during atrial pacing (heart rate 117 beats/min) in a normal subject. In both situations, stress and volume decrease linearly during most of the ejection. LV = left ventricular.

Figure 2. Stress-volume loops of two patients with massive mitral regurgitation. Both patients had normal ejection fractions (EF) and mean velocity of shortening (MVC). In one patient (left), the linear stress-volume decrease during late ejection remained within the normal range (delimited by the dotted lines). In the second patient, this linear relation was below the normal range and the end-systolic volume was increased.
TABLE 2. Effects of Atrial Pacing on the Slope of the Late Systolic Stress-Volume Relation

<table>
<thead>
<tr>
<th></th>
<th>HR (beats/min)</th>
<th>Peak + dP/dt (mm Hg/sec)</th>
<th>LVESV (ml/m²)</th>
<th>LVEDV (ml/m²)</th>
<th>EF (%)</th>
<th>Mean VCF (circuit/sec)</th>
<th>Slope (kdyn/cm²/m²)</th>
<th>Intercept (ml/m²)</th>
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<tr>
<td>Control (n = 24)</td>
<td>77 ± 12</td>
<td>1778 ± 456</td>
<td>43 ± 26</td>
<td>125 ± 54</td>
<td>67 ± 8</td>
<td>1.32 ± 0.31</td>
<td>4.19 ± 1.94</td>
<td>-31 ± 29</td>
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<tr>
<td>Atrial pacing (n = 24)</td>
<td>115 ± 3</td>
<td>2209 ± 592</td>
<td>38 ± 24</td>
<td>109 ± 43</td>
<td>66 ± 9</td>
<td>1.63 ± 0.53</td>
<td>5.22 ± 2.26</td>
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</tr>
<tr>
<td>p</td>
<td>&lt; 0.0001</td>
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</table>

Values are mean ± sd. Abbreviations: See table 1.

p < 0.02). The slopes were also normal, but endsystolic volume was significantly increased.

Studies in Dogs

LV diameter and LV wall stress during the last twothirds of ejection were also linearly related in dogs (fig. 4). This linear relation was close to the classic endsystolic stress-diameter line, and its slope was only slightly affected within a wide range of LV diameters.

To confirm this lack of sensitivity to changes in LV dimensions or in aortic impedance, these slopes were determined beat by beat during aortic clamping and during the decreases in venous return and were compared with the slope measured under basal conditions (fig. 5). The mean wall stress was used as an index of LV afterload because previous studies have shown that this variable best reflected the changes in ventricular afterload and changes in aortic input impedance or ventricular size. Increasing LV afterload to 200% of the control value did not significantly affect the slope of the linear relation between stress and diameter during late ejection, although the duration of the linear phase became progressively shorter (fig. 5). Above these stress values, the contraction became nearly isovolumic and it was no longer possible to determine the slope accurately. When the afterload was decreased to 70% of control, the slope tended to increase slightly but not significantly. The mean systolic wall stress of all studied patients remained 80-157% of the control values, i.e., a range of wall stress within which the slope should be constant.

Under control conditions, the diameter intercept of this late systolic line was smaller than the intercept of the end-systolic stress-diameter line (3.1 ± 3.0 mm vs 8.3 ± 2.4 mm, p < 0.05). This intercept increased from 3.1 ± 3.0 to 6.6 ± 2.8 mm when the wall stress was reduced to 70% of the control and decreased to 1.4 ± 8.0 mm when the stress was increased to 220% of the control. None of these changes were statistically significant.

When the inotropic state was enhanced by calcium, peak positive dP/dt increased significantly from 1270 ± 345 to 2652 ± 450 mm Hg/sec (p < 0.02) and the slope of the late systolic line increased from 38 ± 12 to 72 ± 16 kdyn/cm² (p < 0.05). Simultaneously, the intercept increased from 3.0 to 4.5 mm (NS). The mean systolic wall stress was not affected by calcium (77 ± 13 vs 83 ± 12 kdyn/cm², NS), as the increase in systolic pressure was attended by a decrease in LV enddiastolic dimension.

Thus, within a physiologic range of aortic impedance and LV wall stress, the slope of the late systolic stress-diameter relation was almost constant and therefore remained directly proportional to the classic end-systolic stress-diameter slope. During extreme changes in preload or afterload, late systolic stress-diameter slope tended to increase rather than decrease.

Discussion

Our data indicate that in human and canine left ventricles, LV wall stress and volume are linearly related
during the last two-thirds of ejection. The slope of this relation can be easily determined from angiographic data, is sensitive to inotropic interventions and seems to reflect myocardial contractility more directly than other ejection indexes, such as ejection fraction and mean Vcf. Experiments in animals show that this slope is insignificantly affected by changes in preload or afterload within the physiologic range. Thus, the late systolic stress-volume relation may be a clinically useful index of myocardial contractility.

**Contractile State and Stress-Volume Relations During LV Ejection**

Studies of the whole left ventricle in animals have shown that tension-length relations at the end of ejection generally approximate the isovolumic tension-length relation, although some discrepancies between isovolumic and isometric conditions may be encountered. The tension-length data at the end of ejection fell on a straight line (fig. 4), whose slope was independent of end-diastolic volume and aortic impedance but was affected by acute changes in inotropic state. Consequently, the end-systolic stress-dimension line was proposed as a clinical index of myocardial contractility. Nevertheless, the need for hemodynamic interventions and for repeated ven-triculograms to obtain a reasonable number of data points to fit this end-systolic line limits the clinical use of this superior index of contractility.

On the other hand, the present data suggest that within wide limits of ventricular afterload, the stress-volume line of a single beat during late ejection is directly proportional to the classic end-systolic line. The slope of this line is sensitive to changes in contractile state induced by changes in heart rate or calcium injection. In only three patients was a discrepancy noted between the changes in LV dP/dt and the changes in slope during atrial pacing. Technical problems (reproducibility of the frame-by-frame LV silhouette) might be responsible for these discrepancies. Preliminary data from a separate group of patients with coronary artery disease indicate that this slope increases after administration of a β agonist (ICI 118-587, 0.1 mg/kg).

The present index could therefore be used instead of the classic end-systolic relation, which is difficult to obtain. The stress-volume trajectory during ejection can vary greatly under experimental conditions, particularly when computer-controlled loadings are imposed on the left ventricle. Consequently, the clinical use of this index should be limited to a certain range of LV afterload. For ethical and practical

**Figure 4.** Beat-to-beat changes in left ventricular stress-diameter loop during a decrease in venous return in an open-chest dog. The dotted line corresponds to the end-systolic stress-end-systolic diameter line. The stress-diameter relations are linear during most of the ejection. The slope of this linear phase is much less affected by the maneuver than is the end-systolic diameter.

**Figure 5.** Average changes in the slope of the late systolic stress-diameter line during variations in mean systolic wall stress in open-chest dogs. None of these changes were statistically significant, although the slope tended to increase at low wall stress. This slope was lower than the slope of the classic end-systolic stress-end-systolic diameter relation.
reasons, we determined this range in dogs. In these experiments, this slope was reasonably constant over a wide range of LV systolic wall stress (70–200% of the normal value) (fig. 5). In our patients, the mean systolic wall stress also remained within these limits. This observation was confirmed by Urschel et al., who found that acute mitral regurgitation hardly affected the slope of the LV stress-diameter line during late ejection. In dogs, Taylor et al. showed that except for heavily afterloaded contractions, all the stress-volume trajectories were parallel during late ejection over a wide range of LV volume and stress.

Thus, the stress-volume trajectory during late ejection seems insensitive to change in outflow impedance when the heart exerts its pumping action in a physiologic circuit. The most likely explanation for this observation is that the LV wall stress during late ejection depends much more on the contractile state and instantaneous ventricular geometry than on the outflow impedance. This reemphasizes the significance of the ventricular geometry in determining the afterload of the myofibrils.

Two other objections may arise against the use of such an index. First, it is rather irksome to obtain because it requires frame-by-frame analysis of the LV angiogram. However, the increasing availability of computers and new algorithms to automatically outline the LV silhouette, should solve this problem. Second, this index requires the use of a mathematical model for quantitating wall stress. The formula used in the present study is based on the thin-wall theory, but yields values agreeing closely with those obtained using more complex thick-walled models. We also analyzed our data using Mirsky's formula for the circumferential stress of a prolate spheroid and reached similar conclusions. Nevertheless, the validity of these models has not been established when ventricular geometry is grossly nonuniform, as in hypertrophic cardiomyopathy. In such cases, the results should be interpreted cautiously.

Clinical Usefulness and Limitations

One of the main problems of clinical cardiology remains the detection of discrete impairment of ventricular function, particularly patients with aortic or mitral regurgitation. Early detection is not possible with integrated variables such as end-diastolic volume index, mean Vcf or ejection fraction. Grossman et al. reported that the end-systolic volume index could separate patients with different degrees of left ventricular dysfunction. This index correlated well with postoperative ventricular performance in patients with aortic regurgitation.

Thus, the question arises as to whether the late systolic stress-volume slope provides better information than the more easily determined end-systolic volume. End-systolic volume is more sensitive than the late systolic slope to changes in LV loading. Figure 4 shows that end-systolic volume changed by 40%, while the late systolic slope changed by 6%. Similar conclusions can be drawn from the data of Urschel et al. and Taylor et al. Further, the time-dependent shifts of the LV diastolic pressure-volume relation reported in conscious dogs are also likely to be present in all chronically dilated hearts. Reversible changes in resting length-tension relations of cardiac muscle are also induced by changes in contractile force or by increments in ejection pressure in isolated preparations. Such displacement of the LV filling relationship might induce an increase in end-systolic volume without altering myocardial contractility. In other words, this stress-relaxation phenomenon could increase the volume intercept of the end-systolic stress–end-systolic volume line without altering its slope. Such changes in apparent chamber distensibility were also observed in our animal experiments and undoubtedly contributed to the scatter of the intercept data after aortic clamping.

Thus, the marked load dependency of the end-systolic volume and the time-dependent shifts in LV dimensions suggest that the proposed index might represent myocardial contractility in the clinical setting better than the end-systolic volume or the end-systolic volume-pressure ratio. The same reasons might explain the discrepancies between the slope and the end-systolic volume index in our patients. Although a good correlation was found between the presence of an increased end-systolic volume and the depression of the slope in valvular disease and in congestive cardiomyopathy (figs. 2 and 3), the patients with coronary artery disease had normal slopes despite slightly increased end-systolic volumes (fig. 3). In addition, the presence of series viscous components in the myocardium seriously complicates the interpretation of the changes in intercept of the late systolic line evident in some patients. The effects of hypertrophy on the intercept being unknown, it would be premature to interpret any alteration of this variable in the clinical setting.

To take into account the load dependency of the ejection phase indexes, several investigators have tried to determine control regression lines between ejection fraction, end-systolic volume or mean Vcf and LV wall stress. Although this approach appears useful in confirming myocardial depression in patients with heart failure, it probably cannot detect small changes in contractility. When we applied a similar analysis to our patients with valvular disease, the individual values of mean Vcf or ejection fraction all fell within the 95% confidence interval of the normal subjects, even though many of the patients had altered slopes.

In conclusion, during the last two-thirds of ejection, the LV stress-volume data are linearly related and the slope of this relation yields information about LV tension-length relations. Because the slope of the late stress-volume line can be easily determined from a single standard ventriculogram, we propose that it be used as an index of myocardial contractility, particularly in patients with valvular disease. In such patients, the global ejection indexes as well as the first-third ejection fraction generally remain normal because of the changes in afterload. However, further studies, particularly correlations with postoperative
ventricular function, are required to assess the clinical value of the late systolic stress-volume relation as an index of myocardial contractility.

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