Phasic mitral blood flow and regional left ventricular dimensions: possible mechanism of active assist to ventricular filling

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ABSTRACT Postsystolic myocardial segment shortening (PSS) has been observed in dogs and humans by means of ultrasonic crystals but has never been studied specifically. In this study, both subendocardial and subepicardial regional function in the basal circumflex and midventricular anterior myocardium (LAD) was studied during late systole and early diastole with ultrasonic crystals. Fifteen open-chest dogs were instrumented with electrocardiographic leads; Millar catheters for measurement of left ventricular pressure, left ventricular dP/dt, and aortic blood pressure; flow probes for determination of aortic and mitral blood flow; and subendocardial and subepicardial crystal pairs to measure subendocardial segment length shortening velocity (dL/dt). Crystal pairs were placed in the subendocardial left oblique mode and the extreme subendocardial right oblique mode (−50 and +50 degrees from equator) in the lateral basal (circumflex, n = 9) and anterior midventricular myocardium (LAD, n = 6). Subendocardial segments showed PSS averaging 34 ± 7% of the total shortening distance in the circumflex bed and 21 ± 2% in the LAD bed (p = NS). The rate of subendocardial segment shortening during PSS increased 273 ± 42.6% (p < .05) relative to the rate of segment shortening during ejection in the circumflex bed and 126 ± 40% (p < .05) in the LAD bed (p = NS). The most rapid diastolic increase in subendocardial length (peak + dL/dt) occurred immediately after subendocardial PSS. Subendocardial diastolic peak + dL/dt occurred after the onset of mitral inflow during the acceleration limb of the rapid ventricular filling phase. Overlying subepicardial segments began lengthening 82 ± 12 msec before onset of subendocardial segment lengthening in the circumflex bed and 63 ± 9 msec before subendocardial lengthening in the LAD bed (p < .05), indicating that the subepicardial segment had begun to lengthen while subendocardial segment shortening continued after end-systole. Onset of early segmental subepicardial lengthening varied with respect to the point of end-systole. Early segmental subepicardial lengthening with subendocardial PSS may be a mechanism by which the rapid filling phase of the left ventricle is actively potentiated by storing potential energy released as early diastolic elastic recoil.


SINCE RUSHMER’S 1956 description of the miniature ultrasonic crystal technique for measuring myocardial dimensions in the beating heart, the technique has seen wide application in the study of several aspects of myocardial dynamics in both animals and human beings. Many of these previously published reports both in animals and in man provide evidence for regional or left ventricular short-axis shortening (in nonischemic myocardium) occurring during systole and then proceeding into the early phase of diastole before regional diastolic lengthening occurs.

Sabbah and Stein showed that the pressure-diameter relationship observed during the left ventricular rapid filling phase is inconsistent with passive filling of the ventricle. Ventricular diameter increased while pressure within the ventricle decreased. This is consistent with the concept of diastolic suction, with blood being drawn in freely from the atrium without any negative left ventricular pressures detected.

In this study we observed segmental myocardial shortening in two widely separated regions of the left ventricle after the point of aortic valve closure into early diastole in open-chest dogs in the absence of myocardial ischemia or pharmacologic maneuvers. We call this phenomenon postsystolic shortening...
(PSS) and have correlated its occurrence with other variables such as early mitral blood flow, aortic blood flow, and other functions to determine the possible significance of regional myocardial PSS to global diastolic left ventricular function and left ventricular filling.

**Materials and methods**

**Animal preparation.** Fifteen adult mongrel dogs of either sex weighing 25 to 35 kg were anesthetized with 17.5 mg/kg iv thiamylal sodium 25 min after premedication with 3 mg/kg im morphine sulfate. Mechanical ventilation after intubation was maintained by a Forreger anesthesia machine, with an Ohio positive-pressure respirator using 50% to 80% N₂O and O₂ to maintain normal blood gases. A thoracotomy was then performed through the fifth left intercostal space with the sixth rib removed for greater access to the myocardium supplied by the circumflex coronary artery. The heart was then suspended in a pericardial cradle, and the lungs were retracted, taking care not to obstruct the pulmonary veins. Studies on the effects of pericardiotomy on left ventricular diastolic pressure-volume relationships in the dog have led to the concept of “the diastolic heart as a composite shell of stiff pericardium and compliant muscle.” ⁹ The constraints on diastolic wall movement imposed by the pericardial shell would be expected to be manifested predominantly at later or greater diastolic volumes. By contrast, the events studied in these experiments occur during late systole and early diastole when restrictive pericardial effects would be at a minimum.

A short saline-filled polyethylene catheter was advanced through a carotid artery to just above the aortic valve and connected to a Statham P23Db pressure transducer for measurement of aortic blood pressure. A Millar Micro-Tip high-fidelity micromanometer was implanted in the left ventricular chamber through an apical stab incision. An electrocardiogram lead was sutured to the epicardium in the area supplied by the circumflex coronary artery (see figure 1). A polyethylene catheter was placed in the cephalic vein for infusion of normal saline and periodic supplementation of anesthesia with intravenous thiopental sodium.

Pairs of miniature ultrasonic crystals were implanted in the basal circumflex bed in nine dogs at the subendocardial and subepicardial level to assess regional dynamics. In an additional six animals regional myocardial dynamics were similarly assessed in the distribution of the left anterior descending coronary artery (LAD) toward the apex of the heart with simultaneous subendocardial crystals in the lateral base between branches of the left circumflex coronary artery in three of these animals. In five of the dogs, an incision in the left atrial appendage was made and a mitral valve flow probe (Carolina Medical Electronics, Inc.) was advanced through a purse-string suture and positioned above the mitral orifice for measuring phasic blood flow across the mitral valve as previously described. ¹⁰ Subendocardial segment length dL/dt was measured in five dogs, including four of the animals with mitral flow probes.

**Crystal placement.** Consideration of the continuous rotation of fiber directionality as the left ventricular myocardium is traversed from subendocardium to subepicardium is essential for proper placement of the ultrasonic crystals. Streeter et al. ¹¹ have shown that the smallest proportion of myofibers fall into the longitudinal or apicobasal orientation (90 ± 22.5 degrees from the circumferential line), whereas the greatest proportion of fibers is oriented in the circumferential mode (0 ± 22.5 degrees from circumferential line). Roughly equal portions, accounting for approximately 40% of the total wall thickness, are distributed into the subepicardial left oblique mode (−45 ± 22.5 degrees) and the subendocardial right oblique mode (45 ± 22.5 degrees). In this study, crystals were placed from 1.5 to 2.0 cm apart with the subepicardial pair just beneath the epicardium in the left oblique mode (−50 to −60 degrees from the circumferential line), and the subendocardial pair was always in the deepest fifth of the myocardium just off the endocardium in the right oblique mode (±50 to ±60 degrees from the circumferential line) (figure 1). We elected to implant transducers in this manner to ensure measurement of myocardial segments parallel to the myofibers at each placement depth. Accordingly, the myocardial segments chosen were not parallel to each other but rather were placed in consideration of the ventricular anatomy. This method of subendocardial placement differs substantially from those described in previous studies, which either measure circumferential midwall crystal segments or circumferentially oriented segments in the subendocardial one-third of the wall. The myocardium was carefully dissected after each experiment to ensure that selective placement of the ultrasonic transducers with respect to fiber directionality was achieved. Sites were chosen in the circumflex bed between the anterior and posterior papillary muscles because reproducibility of results and homogeneity of wall muscle distribution was seen to be maximized. The basal location of the crystal pairs also avoids the uneven subendocardial regions where the papillary muscles arise. In addition, basal regions undergo less shearing strain and torsional rotation than do more apical regions, especially in the endocardial third of the myocardium. ¹¹

In six additional animals crystals were similarly implanted in the LAD bed between the LAD and the anterior papillary muscle directly to the apical side of the midpoint between apex and base. The fiber distribution in this region is analogous to that in the basal circumflex bed. ¹¹

**Surgical implantation.** A 3 mm stab incision 0.5 mm deep was made through the epicardium between arterial branches in...
the desired region with a No. 15 scalpel blade while the heart was beating. A blunt contour-ended, 15-gauge stainless-steel ruled hypodermic needle with the crystal leads threaded through it was used to effect a secure and minimally traumatic insertion as evidenced by electrocardiographic stability and the absence of significant hemorrhage. Subendocardial crystals were implanted 1 to 2 mm from the endocardium by advancing the needle until the crystal contacted the endocardial connective tissue (by palpation) and then withdrawing slightly. The preparation was allowed to stabilize for 1 hr, as secure pockets formed around the crystals, thus eliminating the need for retaining sutures. Proper positioning of the miniature ultrasonic transducers was confirmed at autopsy in all 15 dogs. Minimal tissue damage extending less than 1 mm from the transducer was observed at the site of implantation.

Sonomicrometer. The PPI 1000 sonomicrometer (Preysz Precision Instruments, Providence, RI) generates a voltage linearly proportional to the transit time of acoustic impulses traveling at the sonic velocity of approximately 1.5 × 10⁶ mm/sec between the piezoelectric crystals (resonant at 3 MHz), thereby giving a record of instantaneous transducer separation. Thermal drift is minimal. Any drift in the measuring system from the instrument electronics or direct-writing recorder was minimized during the experiment by periodic calibrations. This involved substitution of pulses of precisely known duration from a crystal-controlled pulse generator with a basic stability of ± 0.1% (manufacturer’s specifications).

Circumflex electrocardiogram, aortic blood pressure, aortic blood flow, left ventricular dP/dt, mitral blood flow (n = 5), subendocardial myocardial segment length, subepicardial segment length, and subendocardial dL/dt (first derivative of ultrasonic crystal signal, n = 5) were recorded simultaneously on an eight-channel Brush Gould direct-writing recorder.

Data analysis. For each myocardial segment measured we determined length at end-diastole (identified at the point just before the onset of the positive dP/dt signal), segment length at end-systole (defined as the segment length at 20 msec before peak negative left ventricular dP/dt and corresponding to end ejection), and percent systolic shortening (%L) with systolic shortening defined as the quotient: [(end-diastolic segment length - end-systolic segment length (L)/end-diastolic segment length] × 100). Percent PSS was defined as: [(PSS distance/total segment shortening distance] × 100), with PSS = (end-systolic segment length - minimum segment length) and total segment shortening distance = (end-diastolic segment length - minimum segment length). While dL/dt was recorded in five dogs the slope change before and during subendocardial segment length PSS was determined in 15 animals by direct measurement of slopes on the subendocardial segment length recordings at a paper speed of 100 mm/sec and expressed as percent slope change. In addition, we determined the heart rate–dependent variables of ΔTₐ, ΔTᵣ, ΔTₑ, ΔTₒ (msec), where ΔTₐ = elapsed time from the point of end-systole to the point of minimal subendocardial segment length (maximal shortening distance), ΔTᵣ = elapsed time from onset of subepicardial segment lengthening to the onset of lengthening in the underlying subendocardial segment, ΔTₑ = elapsed time from completion of the rapid early lengthening phase (REL) in the subendocardial segments to completion of the REL phase in the subepicardium, and ΔTₒ = elapsed time from completion of the REL phase in the basal circumflex subendocardial segment to completion of the REL phase in the midventricular LAD subendocardial segment. Completion of the REL phase was determined as the time at which the point on the segment length recording first indicates regional diastasis or the plateau phase of the diastolic segment length recording (figure 2). These variables are listed in tables 1 and 2. Data are expressed as mean ± SEM. Paired groups of data were compared for significant differences by the paired t test. Independent data (tables 1 and 2) were compared by the t test for unpaired comparisons.

![Figure 2](http://circ.ahajournals.org/)

**FIGURE 2.** Record from experiment with simultaneous subendocardial and subepicardial segmental function in the anterior midventricular myocardium in the distribution of the LAD and in the lateral base of the heart in the distribution of the left circumflex artery. Vertical lines delineate systole (S) and diastole (D). Subendocardial segments in both regions show PSS while the subepicardial segment overlaying the LAD subendocardial segment shows relative early lengthening as seen in following figures in the circumflex bed. d (arrow) = the point on the myocardial segment length (MSL) recording at which the REL phase is completed (used for determination of ΔTₑ and ΔTₒ tables 1 and 2).
TABLE 1
Simultaneous subendocardial and subepicardial segmental function in lateral left ventricular base

<table>
<thead>
<tr>
<th>Dog</th>
<th>%L</th>
<th>L (mm)</th>
<th>%PSS</th>
<th>PSS % slope Δ</th>
<th>PSS (mm)</th>
<th>ΔTₐ (msec)</th>
<th>HR</th>
<th>%L</th>
<th>L (mm)</th>
<th>ΔTₐ (msec)</th>
<th>ΔTᵦ (msec)</th>
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<td>1.033</td>
<td>17.23</td>
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</table>

L = systolic shortening distance; %L = systolic segment shortening as percentage of end-diastolic length; %PSS = PSS as percentage of total segmental excursion; PSS % slope Δ = percent decrease in measured slope of subendocardial recording before and during PSS; ΔTₐ = time elapsed from end-systole to maximal subendocardial segment shortening; ΔTᵦ = time elapsed from onset of subepicardial lengthening to onset of subendocardial lengthening; ΔTᵦ = time elapsed from completion of REL phase in endocardium to end of REL in epicardium.

Comparison of %L in the paired subendocardial and subepicardial data showed the difference was not significant by the paired t test. Comparison of ΔTₐ and ΔTᵦ in the paired subendocardial and subepicardial data showed the difference was not significant by the paired t test.

Results

Subendocardial PSS. With ultrasonic crystals placed in the lateral basal aspect of the left ventricle in a right oblique orientation just off the endocardial surface in the subendocardium, we routinely observed a degree of PSS. Systolic shortening in the subendocardial segments averaged 9.35 ± 2.05% of end-diastolic length and 11.39 ± 1.29% of end-diastolic length in the overlying subepicardial segments (n = 9, p = NS) in the circumflex bed and 16.10 ± 1.65% and 10.49 ± 1.18% in the respective subendocardial and subepicardial LAD bed (p = NS). The degree of PSS observed was variable and averaged 34.02 ± 6.96% of the total segment shortening distance in the circumflex bed and 21.27 ± 2.38% in the LAD bed (p = NS). Time elapsed from end-systole to the point of maximal subendocardial segment shortening (ΔTₛ) averaged 72.22 ± 8.34 msec in the circumflex bed and 80.8 ± 6.5 msec in the LAD bed. With the present sample, no relationship was found between ΔTₛ and heart rate (tables 1 and 2).

Figure 3 shows a recording taken from one experiment with ultrasonic crystals in the circumflex myocardium. The vertical lines delineate systole and diastole, with the first line marking the onset of mechanical systole, the second line end-systole, and the last line end-diastole. Following the first vertical line, a brief plateau is observed on the ultrasonic crystal tracing, which occurs during isovolumic ventricular contraction. With the onset of systolic ejection (see aortic blood flow tracing), the segment length decreases rapidly as the ultrasonic crystals move closer together. In early diastole the segment length increases rapidly during the rapid ventricular filling phase. The diastolic increase in segment length then tapers off during diastole.
TABLE 2
Simultaneous subendocardial and subepicardial segmental function in anterior midventricular and lateral base subendocardium

<table>
<thead>
<tr>
<th>Dog</th>
<th>Region</th>
<th>%L (mm)</th>
<th>%PSS</th>
<th>PSS % slope Δ</th>
<th>PSS (mm)</th>
<th>ΔTA (msec)</th>
<th>HR</th>
<th>L (mm)</th>
<th>ΔTB (msec)</th>
<th>ΔTC (msec)</th>
<th>ΔTD (msec)</th>
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</thead>
<tbody>
<tr>
<td>10</td>
<td>LAD</td>
<td>16.13</td>
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<td>16.67</td>
<td>173.0</td>
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<td>80</td>
<td>90</td>
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<td>97±</td>
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<td>97±</td>
<td>10.49±</td>
<td>1.34±</td>
<td>63.33±</td>
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ΔTA = time elapsed from completion of REL phase in the basal circumflex subendocardial segment to completion of REL in the midventricular LAD subendocardial segment; LAD = segment between LAD and anterior papillary muscle at midventricle; CIRC = segment between anterior and posterior papillary muscle near base of heart.

Data obtained following subendocardial, subepicardial LAD run; therefore LAD subendocardial data with simultaneous CIRC subendocardial data not pooled with LAD data from the same dog.

Comparison of %L in the paired subendocardial and subepicardial data showed the difference was not significant by the paired t test (p < .10).

Comparison of ΔTa and ΔTB in the paired subendocardial and subepicardial data showed the difference was significant at p < .05.

Subendocardial PSS with simultaneous subepicardial segment shortening. During subendocardial PSS we observed simultaneous subepicardial segment shortening beginning an average of 10.67 ± 11.73 msec earlier than the onset of subendocardial segment shortening in the circumflex bed (table 1, ΔTa) and 63.33 ± 8.72 msec in the LAD bed (table 2, ΔTB). The onset of subepicardial shortening was variable with respect to the point of end-systole, occurring before end-systole in six dogs, after end-systole in two dogs, and synchronously with end-systole in one dog in the circumflex bed (table 1) and at or after end-systole in all six animals with LAD bed transducers (table 2). The difference between ΔTa and ΔTB for each experiment averaged 7.78 ± 13.8 msec (p = NS) in the circumflex bed and 17.50 ± 7.9 msec in the LAD bed (p < .05), showing the variable timing of the onset of asynchronous early subepicardial segment relaxation. Subendocardial PSS occurs in the nonischemic heart when the subepicardial segment has completed shortening or begun shortening.

Analysis of the interval between time to completion of regional REL phase in the subendocardial layers and time to completion of the REL phase in subepicardial layers (ΔTC, circumflex bed, table 1; ΔTC, LAD bed, table 2) revealed no significant differences in the circumflex bed (n = 9). A significantly shorter time to completion of the REL phase in the LAD subendocardium compared with LAD subepicardial segments was noted, averaging 23.33 ± 8.53 msec (n = 6, p < .025). The difference in time to completion of the REL phase in simultaneously measured subendocardial circumflex and LAD segments was not significant (ΔTD, table 2).

Figure 4, from a different experiment, shows aortic blood flow, left ventricular pressure, and simultaneous ultrasonic crystal records from subepicardial and subendocardial segments. The paper speed on the strip chart was increased from 100 to 200 mm/sec. The subendocardial segment exhibits PSS as in figure 3 while the overlying subepicardial segment begins to show shortening 95 msec after diastolic segment.
lengthening in the subendocardium. A similar recording is presented in figure 2 from a different experiment, with ultrasonic crystals in the subepicardial and subendocardial LAD bed and in the subendocardial circumflex bed.

The record chosen for figure 5 from a different experiment with mitral flow measurements does not show early subepicardial lengthening as in the other figures. However, subepicardial shortening in figure 5 has been completed before the onset of PSS in the underlying subendocardium. The animals studied showed variable onset of subepicardial lengthening be-

FIGURE 5. Simultaneous recording of (top to bottom) surface ECG in the circumflex bed, subepicardial segment length (subepi MSL), aortic blood pressure, mitral blood flow, subendocardial segment length (subendo MSL), left ventricular pressure, and subendocardial MSL dL/dt. Vertical lines delineate the phases of A) ventricular ejection (aortic blood pressure, A), subendocardial PSS (MSL subendo, B), ventricular rapid filling (mitral flow) and diastasis (C), and ventricular filling due to atrial contraction (second mitral flow peak, MSL subepi and MSL subendo, D). Subepicardial segment shortening is completed during ejection (A) while subendocardial PSS continues beyond the point of aortic valve closure before the onset of mitral inflow during isovolumic ventricular relaxation (B). The subendocardial segment lengthens rapidly after PSS as the mitral valve opens during the rapid filling phase (C).
peak of the atrial component of mitral flow corresponds with the increase in segment length after atrial systole.

The four subdivisions of events during the cardiac cycle in figure 5 illustrates the phases of ventricular ejection, (A), subendocardial segmental PSS (B), ventricular rapid filling and diastasis (C), and the contribution to ventricular filling by atrial contraction (D). The changes in segment length during isovolumic contraction and the ejection phase are similar to those in the previous figures. Subepicardial segment shortening is completed during ejection (A) while subendocardial PSS continues beyond the point of aortic valve closure (B). The rate of segment shortening during PSS increased an average of 273 ± 42.6% relative to the rate of shortening during ejection (n = 9, p < .05) in the circumflex bed and 126.4 ± 40.1% in the LAD bed (n = 6, p < .05). This difference between the circumflex and LAD bed was not statistically significant. Immediately after PSS (C) the subendocardial segment lengthens rapidly (dL/dt tracing) as the mitral valve opens and mitral inflow occurs during the rapid ventricular filling phase. The atrial component of mitral flow corresponds with the increases in subendocardial and subepicardial segment length as the ventricle is distended in this region during atrial systole (D).

The points of end-diastole and end-systole are not identified in the subdivisions of figure 5, which call attention to processes of filling, ejection, and wall motion without delineation of strictly isovolumic periods. Figure 7 shows comparable data from another experiment with subendocardial and subepicardial ultrasonic crystals in the LAD bed and simultaneous transmitral flow measurement. Subendocardial PSS also occurs in this anterior portion of the left ventricular wall during subepicardial segment lengthening. Rapid subendocardial segment lengthening following PSS is coincident with the acceleration limb of the first transmitral flow peak during the ventricular rapid filling phase. As seen in previous figures, the transmitral flow peak after atrial contraction coincides with a small late diastolic increase in the regional segment length measurements. Data from experiments with ultrasonic crystals placed in the LAD bed are listed in table 2. The regional segmental contraction phase of PSS did not always occur exclusively within the globally defined period of isovolumic relaxation as occurs in figures 5 and 7. Regional PSS continued at times for 10 to 20 msec after the onset of mitral blood flow. Onset of rapid segmental lengthening after PSS always occurred before or during the acceleration phase of the first transmitral flow peak.

FIGURE 6. Simultaneous recording of (top to bottom) aortic blood pressure, aortic blood flow, subendocardial MSL, mitral blood flow, left ventricular dP/dt, and left ventricular pressure. Vertical lines denote diastole (D) and end-systole (S) as determined from the dP/dt record. Marked PSS is observed in the subendocardial MSL. The predominant portion of the mitral flow rapid ventricular filling phase occurs during the rapid phase of myocardial segment lengthening, which immediately follows subendocardial PSS. An increase in segment lengthening is seen coincident with the mitral blood flow peak from atrial systole. (From same experiment as figure 4.)

tween animals and to a lesser extent during each experiment, which usually preceded subendocardial lengthening by an average of approximately 80 msec and occurred during subendocardial PSS in the circumflex bed (n = 9, table 1, ΔTb) and by approximately 60 msec in the LAD bed (n = 6, table 2, ΔTb).

PSS and relationship to subendocardial diastolic function and mitral blood flow. Figure 6 shows data from the same experiment as in figure 4 with the addition of transmitral blood flow and left ventricular dP/dt. Changes in segment length during isovolumic contraction and the onset of ejection are similar to those seen in figures 3 and 4. Once again PSS is observed in this myocardial segment. Note also an increased negative slope during PSS indicating an increase in the rate of myocardial segment shortening. PSS in the subendocardial segment begins when aortic pressure indicates closure of the aortic valve. Onset of PSS is followed by rapid PSS with subsequent rapid segment lengthening during the early filling phase of mitral blood flow. The
Discussion

Previously published reports have shown PSS in both open and awake closed-chest dogs by means of several patterns of miniature ultrasonic crystal placement.\textsuperscript{2-4} Regional and short-axis PSS have been observed in human hearts at the time of cardiac surgery.\textsuperscript{5-7} Several studies have shown PSS to occur in nonischemic myocardium while others have described PSS to be an occurrence in ischemic myocardium and after coronary occlusion and reperfusion.\textsuperscript{2-4,12} Still other studies have attributed PSS to effects of inhalant anesthetic gases in animals with coronary stenosis.\textsuperscript{13-15} We have routinely observed this behavior in healthy, anesthetized dogs with normally functioning hearts. It is already established from many previous studies that PSS occurs in most portions of the left ventricular wall in nonischemic hearts, including segments in the distribution of both the circumflex and LAD, midwall and subendocardial segments, and across the ventricular cavity short axis in man and open- and closed-chest dogs.\textsuperscript{1-7,12-16} Studies with awake chronically instrumented dogs have shown PSS by means of ultrasonic crystals in short-axis external left ventricular diameter measurements,\textsuperscript{1} short-axis internal diameters,\textsuperscript{3} and regional subendocardial measurements in the circumflex coronary artery distribution.\textsuperscript{4,12} Studies with open-chest dogs have similarly shown PSS to occur in the LAD distribution in subendocardial segments parallel to the ventricular short axis\textsuperscript{2,13-15} and in midwall segments parallel to the ventricular long axis along the line bisecting the angle formed by the LAD and circumflex branches.\textsuperscript{16}

In addition PSS has been measured in open-chest human studies with ultrasonic crystals after coronary artery bypass to measure left ventricular short-axis external diameters\textsuperscript{7} as well as regional midwall segments in the distribution of the LAD.\textsuperscript{5,6} Different regions of the left ventricular subendocardium exhibit substantial anatomic variation, particularly in areas where the papillary muscles greatly increase the local wall thickness, making placement of more apical transducer pairs difficult to standardize. More apically located segments exhibit increased shearing strain and torsional rotation than more basal segments, especially in the endocardial third of the myocardium, which may confound direct comparison of segments located in different areas.\textsuperscript{11} In addition, the fact that apical and midventricular segments complete the rapid lengthening phase significantly later than basal segments may introduce an additional variable of apicobasal delays.\textsuperscript{16} Subtle alterations of function could be imposed by damage to contractile elements and conduction pathways from additional contiguously placed pairs of crystals.

We found regional segment length dynamics in the basal circumflex region and in the distribution of the LAD near the interventricular septum and midway between apex and base to be comparable. Subendocardial PSS and early subepicardial segment lengthening relative to the onset of subendocardial segment length-

\textbf{FIGURE 7.} Similar record from another experiment with ultrasonic crystals measuring subendocardial and subepicardial segments in the LAD bed with simultaneous transmitral blood flow. Again subendocardial PSS occurs during overlying subepicardial early segment lengthening. Subendocardial REL immediately after PSS is seen coincident with the initial acceleration phase of transmitral blood flow. Transmitral flow after atrial contraction is reflected in the myocardial segment length measurements as small late diastolic increases in segment length. Vertical lines delineate systole (S) and diastole (D).
ening were observed in both regions of the left ventricle. Although only two widely separated regions of the left ventricle were studied, the similarities noted in these regional patterns may suggest a global mechanism.

We suggest that although PSS does not contribute directly to systolic ejection, it may serve to store potential energy that is released in elastic recoil as a restoring force, which rapidly returns the left ventricle to diastolic dimensions thus aiding left ventricular filling. Our data show that the onset of mitral blood flow, occurring during the rapid filling phase, corresponds very closely with the early rapid diastolic increase in subendocardial myocardial segment length. The most rapid increase in diastolic segment length occurs immediately after PSS. Therefore PSS may be a mechanism that augments the rapid ventricular filling phase in early diastole. Onset of the REL phase after subendocardial PSS occurs close to the onset of transmitral blood flow or during the acceleration phase of transmitral blood flow during the rapid ventricular filling phase. Although the significance of any myocardial recoil after PSS in augmenting mitral valve opening remains unknown, PSS with subsequent segmental rapid wall lengthening during the acceleration phase of the first mitral flow peak may provide a mechanism to augment this early rapid filling phase. Although there are inherent difficulties in extrapolating regional contraction patterns to changes in ventricular volume in relation to transmitral valve flow, studies have shown that changes in left ventricular stroke volume correlate closely with the cube of local myocardial dimension changes by means of the ultrasonic crystal method.17

We have shown in a previous study describing subendocardial and sâbepicardial segment length changes occurring with progressive coronary occlusion that a point on the subendocardial segment length recording can often be identified at end-systole where PSS begins during control coronary flow, which becomes the peak of the “paradoxical bulge” during progressively ischemic conditions.18 After the ischemic end-systolic bulge, the subendocardial segment shortens rapidly to its nadir during isovolumic relaxation before lengthening during the remainder of diastole.16, 18 Nearly identical recordings have been made by other authors using ultrasonic crystals in the distribution of the LAD.5 This phenomenon may be attributed solely to regional failure of an ischemic region to shorten throughout systole but may also be interpreted as a compensatory mechanism during ischemia that allows ischemic areas to aid ventricular filling by PSS and elastic recoil while other less ischemic or nonischemic regions shorten to a greater extent throughout systole in compensation. The latter mechanism has been described in previous studies examining segment length changes with ultrasonic crystals placed in ischemic and nonischemic regions of the left ventricular wall.2

Previous reports dating from antiquity through the 1950s to the present have addressed the degree to which the left ventricle in diastole behaves as a passively distending structure or involves restoring forces (diastolic suction) that aid ventricular volume increases and ventricular filling in early diastole.8, 19-21 Tyberg et al.19 provide two factors to explain this mechanism. The first, a geometric factor, uses the analogy of a tennis ball, which has a finite-equilibrium size that is maintained without transmural pressure and reflects the gross structure of the wall. Reduction of volume below this equilibrium volume requires active force. When this is removed, the ball will attempt to resume its previous volume, thus generating a negative pressure.19 They also suggested an ultrastructural factor based on studies showing that individual muscle fibers will shorten and with relaxation reelongate themselves22 with sarcomere lengths comparable to those in an intact ventricle at zero resting transmural pressure. Ejection of volume below that at zero filling pressure would be expected to produce restoring forces in the sarcomere that would contribute to the energy for elastic recoil and produce forces that augment early ventricular filling.19 Sabbah and Stein8 showed that the pressure-diameter relationship observed during the left ventricular rapid filling phase is inconsistent with passive filling of the ventricle. Ventricular diameter increased while pressure within the ventricle decreased. They suggested “self-enlargement” of the ventricle during the rapid filling phase, “possibly secondary to an active process of myocardial relaxation . . . . Elastic recoil or muscular contraction within the left ventricular wall during diastole may produce restoring forces which assist ventricular filling.”8 Our results may provide evidence for a regional demonstration of this concept in the intact beating dog heart.

Since the PSS observed in these studies occurs largely during the isovolumic relaxation period when the ventricle would be expected to maintain a constant volume, we would expect there to be a compensatory elongation elsewhere in the ventricle. In addition to the apparent compensatory lengthening of overlying subepicardial segments, we would expect that other areas within the ventricular wall move in concert during filling and ejection with normal impulse conduction to optimize the forces that produce ventricular filling during the early phase of mitral flow when the majority of

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ventricular filling occurs. Lew and Lewinter\textsuperscript{16} showed asynchrony of the rapid lengthening phase from apex to base with crystals placed in the midwall parallel to the myofibers in open-chest and chronically instrumented dogs. With a left ventricular end-diastolic pressure of 10 mm Hg, the time from mitral valve opening to the end of the rapid lengthening phase of the segment length recordings averaged 68 msec toward the base of the heart compared with 100 and 102 msec at the midventricle and apex, respectively. Similarly, the basal segments reached peak diastolic lengthening rates before midventricular and apical segments. Also of interest was the observation that PSS shortening clearly occurs in their basal segments that finish the rapid lengthening phase approximately 30 msec before their midventricular segment where PSS is not seen. Although the crystal placement in that study does not allow direct comparison with ours, the authors interpreted their findings to be “consistent with the concept of regional differences in elastic recoil, which may contribute to active ventricular filling.”

In this study the subendocardial LAD segments completed the REL phase significantly earlier than the overlying subepicardial LAD segments (23 \pm 9 msec, $\Delta T_C$, table 2) whereas in the circumflex bed the difference was not significant ($\Delta T_C$, table 1). The explanation for this difference in the two left ventricular regions remains speculative but may reflect the shorter interval from onset of subepicardial segment lengthening to onset of subendocardial segment lengthening after PSS (63 \pm 9 msec, $\Delta T_B$, table 2) in the LAD bed compared with that in the circumflex bed (82 \pm 12 msec, $\Delta T_B$, table 1).

We compared the point of completion of the REL phase in the basal circumflex subendocardial segments with that in the simultaneously measured mid to apical LAD segments and found no significant difference, possibly because of the small sample size of simultaneous measurements of subendocardial LAD and circumflex region segments ($n = 3$, $\Delta T_B$, table 2).

Numerous studies have demonstrated early diastolic enlargement of portions of the left ventricular cavity by means of left ventricular cineangiography and referred to the phenomenon as asynchronous segmental early relaxation (ASER).\textsuperscript{23-26} The regional ultrasonic crystal studies of Lew and Lewinter,\textsuperscript{16} which show early completion of the rapid segmental lengthening phase in basal segments compared with midwall or apical segments, may reflect asynchrony of early relaxation in areas of the ventricular wall similar to that seen in ASER. Subendocardial PSS, with compensatory subepicardial segment lengthening before the rapid lengthening phase and mitral inflow, may augment early expansion of portions of the left ventricle to assist ventricular filling. In one study ASER was observed in left ventricular cineangiograms in 45\% of a set of patients with arteriographically proven unobstructed coronary arteries as well as in 38\% of patients with significant coronary artery disease.\textsuperscript{24} Additional human studies have shown ASER in normal and ischemic hearts.\textsuperscript{23-26} ASER has been described as a normal variation of left ventricular relaxation rather than a characteristic of abnormally contracting left ventricles,\textsuperscript{23} an indication of nonuniformity of left ventricular relaxation,\textsuperscript{24} a compensatory mechanism in areas of normal contraction that offsets abnormal inward motion elsewhere, tending to maintain isovolumic status of the ventricle,\textsuperscript{26} and a very common finding among normal subjects appearing to be directly related to end-systolic volume index and wall stress.\textsuperscript{24} In a more recent review article on the subject of asynchronous left ventricular relaxation, Gaasch et al.\textsuperscript{27} suggest that, “Under some circumstances, a single mechanism may be responsible [for segmental early relaxation], but sometimes an interplay of several hemodynamic or other factors may be necessary to produce the segmental early relaxation phenomenon.”

We speculate that the pronounced asynchrony of onset of segment lengthening in the subendocardium and subepicardium observed in the present study may be related to the phenomenon of ASER described in the previous studies and that it may in fact be a normal occurrence in the intact working left ventricle. We suggest that the form of early subepicardial segmental relaxation demonstrated in these experiments is a function of the processes producing regional subendocardial PSS, which ultimately aids the early phase of left ventricular filling.

Further studies are needed to determine the effects of adrenergic activity, volume alterations, mitral stenosis, and metabolic or pharmacologic interventions on regional segmental early relaxation, mitral inflow, and segmental PSS.

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