Regional Differences in Myocardial Performance in the Left Ventricle of the Dog

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ABSTRACT

To determine whether significant regional differences in shortening exist in the canine left ventricle, the shortening characteristics of small segments of the circumferentially oriented hoop axis fibers and the more longitudinally oriented fibers near the epicardium were examined using pairs of ultrasound crystals placed at three levels of the left ventricular free wall in the open-chest dog. Mean control shortening of the hoop axis fibers near the apex of the left ventricle averaged 20% of the end-diastolic length, significantly greater than shortening at the midventricular (13%) or basal (14%) levels. During transient periods of aortic constriction, end-diastolic length increased significantly and the extent of shortening was maintained for the hoop axis fibers at the apical and midventricular levels; end-diastolic length did not change and shortening decreased at the basal level. The epicardial fibers shortened an average of 5.6% of their end-diastolic length during control conditions at all three sites and showed small, parallel changes in shortening and end-diastolic length during aortic constriction. We conclude that significantly greater hoop axis shortening occurs near the apex of the left ventricle and that at this level a uniformly contracting model is inappropriate. In addition, the response of the hoop axis fibers to increased aortic impedance is not homogeneous, with a significant reduction in shortening occurring only at the base of the left ventricle where end-diastolic length does not increase.

Methods

For the studies of the circumferentially oriented midwall fibers, six adult mongrel dogs were anesthetized with an intravenous injection of 25 mg/kg of sodium pentobarbital and given small additional injections as needed. The dogs were intubated, and respiration was maintained with a Harvard respirator. The heart was exposed through a median sternotomy and suspended in a pericardial cradle. Polyethylene catheters were inserted into a jugular vein for administration of fluids and in most experiments into the right femoral artery for ventricular shortening characteristics. Kong et al. (14), using coronary artery bifurcations as markers, have noted an increased extent of shortening toward the apex of the left ventricle. Liedtke et al. (15), in an angiographic analysis of circumferential shortening, have obtained similar results.

To examine whether left ventricular fiber shortening is uniform and can be predicted from measurements of radial or long axis dimension changes, we utilized a modified ultrasound technique (16–19) to examine segmental shortening at several sites along the major axis of the left ventricle. At each site, pairs of ultrasound crystals were implanted in the circumferentially oriented fibers in the middle 60% of the wall and in the more longitudinally oriented fibers located at the epicardial surface (13). Shortening characteristics were then analyzed before and during altered ventricular performance produced by changes in aortic impedance.
measurement of abdominal aortic pressure utilizing a Statham P23Db transducer. A Statham SF-1 micromanometer tip, fluid-filled catheter was inserted into the left ventricle via the left atrial appendage for measurement of left ventricular pressures. The lumen of this catheter was connected to a Statham P23Db transducer, and the lumen and high-fidelity pressures were matched before each run.

Techniques for determining myocardial dimensions and segment lengths using ultrasound crystals have recently been described in detail (18, 19). The system used in the present study provided a flat frequency response from 0 to 100 Hz. The ultrasound crystals were constructed using 6 MHz piezoelectric disks (resolution 0.13 mm). The completed crystals had a diameter of 2.5 mm and a thickness of 2.0 mm. Three pairs of crystals were inserted into the left ventricular myocardium through small stab wounds to an approximate depth of 5 mm as shown in Figure 1A; all crystals were placed in the free wall of the left ventricular myocardium, one pair near the base, one near the apex, and one approximately halfway between the basal and apical pairs at the midventricular level. All pairs were perpendicular to the long axis of the ventricle and, therefore, parallel to the direction of the hoop fibers, as described by Streeter et al. (13). It should be noted that the positioning of the basal, midventricular, and apical crystal pairs was designed to approximate the position of the upper, middle, and lower aspects of the “leg” of the T-shaped area of left ventricular free wall examined by Streeter et al. (13). Five other dogs were utilized for studies of the epicardial fibers. Pairs of crystals were sewn to the epicardium at locations on the left ventricular free wall similar to those for the midwall studies but oriented in the direction of the epicardial fibers according to the data of Streeter et al. (13). The locations of these crystals are illustrated in Figure 1B. In these five dogs, left ventricular pressures were monitored through a fluid-filled polyethylene catheter introduced from the left atrial appendage and attached to a Statham P23Db transducer. This catheter-manometer system had a flat frequency response from 0 to 40 Hz. After each study, the heart was removed and the precise positions of the crystals were determined. For this purpose, measurements were made of the location of the crystals with respect to the long axis of the ventricle (measured from the point of bifurcation of the left main coronary artery into its anterior descending and circumflex branches to the apex), and the position of the segment was expressed as a percent of the base-to-apex distance. The distance between the crystal pairs along the epicardium and the wall thickness at the site of crystal location were also noted. For the midwall studies the depth of each crystal expressed as a percent of wall thickness from the epicardium to the endocardium was determined, and for the epicardial studies the angle made by a line between the crystals and a line perpendicular to the long axis of the ventricle was measured.

During each study, measurements of aortic pressure, left ventricular pressure, and crystal segment lengths were made, and a suitable electrocardiographic lead was monitored. Malalignment of the crystals due to rotation could easily be detected from the morphology of the received signal and corrected during the course of the study. All data write-out was done on a polygraph (Brush Clevite, model 2000) and analyzed at a paper speed of 100 mm/sec with respiration suspended at end-expiration. Ten cardiac cycles were averaged for each measurement. Control measurements were made before and after three or four constrictions of the descending thoracic aorta performed by gradual tightening of a length of umbilical tape looped around the vessel. Measurements were made as soon as pressures and segment lengths appeared to have stabilized but at least 20 seconds after peak left ventricular pressure had been reached. The aortic constrictions were regulated to produce an average increase in peak left ventricular pressure of as close to 30 mm Hg as possible.

In four additional dogs, simultaneous control epicardial and midwall measurements were made at the apical or the midventricular levels. These studies were used to compare qualitative aspects of shortening at the two sites simultaneously. Finally, two dogs were instrumented with midwall crystals and studied in the conscious, resting state after 1–2 weeks of recovery from surgery.

Tests for significance were performed using Student’s t-distribution for paired samples (20).
Results

CRYSTAL PLACEMENT

For the midwall crystals, the mean percent distance from the base of the left ventricle to the apex for the basal crystals was 23 ± 1.6% (SE), for the midventricular crystals 52 ± 0.9%, and for the apical crystals 79 ± 1.2%. The mean distance between crystal pairs at all three levels was similar (basal 1.6 ± 0.16 cm, midventricular 1.5 ± 0.10 cm, and apical 1.6 ± 0.19 cm). The mean depth of the crystals in the three locations ranged from 52% to 55% of wall thickness, measured from epicardium to endocardium, and all of the crystals were located within the middle 30% of the wall. There was less than a 10% difference in depth between individual crystals in any pair. Mean wall thickness at the basal location was 1.15 cm (range 1.0 to 1.3 cm), at the midventricle 1.18 cm (range 1.1 to 1.3 cm), and at the apex 1.02 cm (range 0.9 to 1.2 cm).

For the epicardial studies, the mean percent distance from the base of the left ventricle to the apex for the basal crystals was 26 ± 1.2%, for the midventricular crystals 54 ± 0.0%, and for the apical crystals 75 ± 1.8%. The mean distance between crystal pairs was once again similar (basal 1.3 ± 0.10 cm, midventricular 1.3 ± 0.11 cm, and apical 1.2 ± 0.12 cm). Finally, the mean angle between the crystals and a line perpendicular to the long axis of the ventricle (Fig. 1B) was 70 ± 3° for the basal crystals, 72 ± 3° for the midwall crystals, and 71 ± 3° for the apical crystals (range 60 to 77° for all locations).

MIDWALL SEGMENTAL SHORTENING CHARACTERISTICS

A typical example of shortening at the apical, midventricular, and basal levels is shown in Figure 2. The control extent of shortening for the three segments is presented in Table 1 and illustrated graphically in Figure 3A. The mean extent of shortening of the apical segment (20 ± 1.9%), expressed as a percent of the end-diastolic length, was significantly greater than that of the midventricular segment (13 ± 0.8%, P < 0.004) and the basal segment (14 ± 1.1%, P < 0.006). There was no significant difference in the extent of shortening between the midventricular and basal segments.

Similar relative differences in the extent of shortening were observed in the two conscious dogs, although the absolute amount of shortening was somewhat less. In one dog, apical shortening was 16%, midventricular 11%, and basal 10%. In the other dog, apical shortening was 14% and midventricular shortening 10%.

The effects of aortic constriction on left ventricular peak systolic pressure, end-diastolic pressure, and heart rate for these studies are presented in Table 1. Mean left ventricular peak systolic pressure increased from the control value of 112 ± 4.7 mm Hg to 166 ± 7.3 mm Hg during aortic constriction. Left ventricular end-diastolic pressure increased from 4.8 ± 1.1 to 8.3 ± 1.1 mm Hg (P < 0.003). Heart rate was not significantly changed (124 ± 4.5 beats/min during the control period and 124 ± 4.1 beats/min during aortic constriction).

Mean segment length changes during aortic constriction are presented in Figure 4A. Mean end-diastolic length of the apical segment increased by 7% from 1.49 ± 0.19 cm during the control period to 1.59 ± 0.19 cm during aortic constriction (P < 0.003). The midventricular end-diastolic length increased by 3% from 1.52 ± 0.10 cm to 1.57 ± 0.10 cm (P < 0.02). End-diastolic length of the basal segment did not change significantly (control 1.38 ± 0.15 cm, constriction 1.40 ± 0.15 cm). Mean end-systolic length increased in all three segments; the apical segment increase was 8% from 1.19 ± 0.15 cm to 1.28 ± 0.17 cm (P < 0.05), the midventricular increase was 4% from 1.32 ± 0.09 cm to 1.37 ± 0.09 cm, and the basal segment...
increase was 7% from 1.20 ± 0.14 cm to 1.28 ± 0.14 cm.

The extent of shortening during aortic constriction is presented in Table 1 and illustrated graphically in Figure 5. Shortening was maintained in the apical and midventricular segments following aortic constriction. However, the extent of shortening of the basal segment decreased significantly during aortic constriction from 14 ± 1.1% to 9 ± 0.05% (P < 0.02).

EPICARDIAL SEGMENTAL SHORTENING CHARACTERISTICS

A representative tracing from an epicardial segment with a simultaneously obtained tracing from a midwall segment is shown in Figure 6. The control extent of shortening for the epicardial segments is presented in Table 2 and illustrated graphically in Figure 3B. The mean extent of shortening of the apical segment was 6 ± 2.3%, of the midventricular segment 5 ± 0.8%, and of the basal segment 6 ± 0.9%.

The effects of aortic constriction on left ventricular peak systolic pressure and heart rate for the epicardial studies are presented in Table 2. Mean left ventricular peak systolic pressure increased from 123 mm Hg to 179 mm Hg. Mean heart rate before constriction was 147 ± 8.4 beats/min; during constriction it was 134 ± 5.7 beats/min. During aortic constriction, increases in mean end-diastolic length of 3%, 4%, and 4% were seen in the apical, midventricular, and basal locations, respectively. Only the change in end-diastolic length at the base attained statistical significance (P < 0.02). Significant parallel increases in mean end-systolic length of 6% at the apex, 6% at the midventricular segment, and 2% at the base were also observed (P < 0.005 for each site). These segment length changes during aortic constriction are represented in Figure 4B. The extent of shortening was not significantly changed during constriction compared with that during control conditions at any of the three epicardial sites (Table 2).

SIMULTANEOUS MIDWALL AND EPICARDIAL SHORTENING CHARACTERISTICS

Simultaneous studies of midwall and epicardial shortening at the apical location in three dogs and at the midventricular level in three dogs disclosed consistent differences with respect to the timing of shortening. This finding is illustrated in Figure 6, in this case at the apical location. Note that as left ventricular pressure begins to rise the epicardial segment lengthens. At the same time, the midwall segment begins to shorten at a relatively slow rate. Shortly before the plateau in left ventricular pressure is reached, the epicardial segment first begins
Mean control percent extent of shortening at the three crystal sites for the midwall (A) and the epicardial (B) studies. Apical shortening was significantly greater than midventricular (P < 0.004) or basal (P < 0.006) shortening for the midwall studies, but there were no significant differences in the three sites for the epicardial studies.

Discussion

The results of the current study indicate that the shortening characteristics of the midwall hoop fibers of the free wall of the canine left ventricle vary along its major axis both in the open-chest and in the conscious dog. The extent of shortening of these fibers may also be differentially influenced by alterations in preload and afterload. It is possible that these regional differences in shortening are related to the changes in left ventricular volume and shape occurring in an open-chest preparation; however, the studies performed in conscious dogs also showed similar regional differences in shortening. Thus, it is unlikely that such changes are responsible for the regional variations.

These results are at variance with the assumptions employed by a variety of techniques for the evaluation of left ventricular function. In general, these techniques utilize geometric models for an estimate of minor axis shortening or the cineangiographic or echocardiographic determination of one chord or diameter for the evaluation of function throughout the ventricle (9-11, 21-23). The ellipsoid of evolution, a commonly used geometric model, does reflect a lessening ventricular diameter as one approaches the apex. However, even this model would predict uniform midwall hoop fiber shortening at all levels along the major axis (see Appendix 1 for this analysis).

If the left ventricle does not behave as a uniformly contracting chamber and the assumption is made that the contractile state of all fibers in a normal left ventricle is the same, then differences in the extent of shortening in different areas of the left ventricle may relate to regional variations in either preload or afterload. With regard to regional differences in preload, Laks et al. (24) have published data demonstrating significantly longer sarcomere lengths at the left ventricular apex. At the same afterload, this type of regional sarcomere length distribution would be consistent with the greater degree of shortening observed at the apex. Direct measurements of regional variations in wall stress at different apex-to-base levels are not available. However, according to the LaPlace relationship, as one approaches the apex wall tension...
should decrease as a result of the smaller radius. In contrast, it also has been shown that wall thickness is proportional to radius throughout the left ventricular wall (25, 26). Using variations of the LaPlace relationships for a thick-walled ellipsoid of revolution (9), it is apparent that any reduction in wall tension which might derive from the smaller radius near the apex might be offset by reduced wall thickness. Thus, it would seem unlikely that major regional differences in afterload exist which would explain the increased shortening we observed at the apex.

Our data indicate that the shortening in the direction of the epicardial fibers at the basal, midventricular, and apical ventricular levels was uniform, averaging 5.6 ± 0.8% at these sites. Streeter et al. (13) have noted that fiber directions change about 20° during systole at the apical epicardial level only. Since these crystals are insensi-

**FIGURE 4**

Segment length data under control conditions (open bars) and during aortic constriction (hashed bars) for the midwall (A) and the epicardial (B) studies at all three sites. The top portion of each bar represents end-diastolic length, the bottom portion end-systolic length, and the distance between the absolute amount of shortening in centimeters. C = controls, and Ao = aortic constriction.

**FIGURE 5**

Mean percent extent of shortening for the midwall studies at each crystal site under control conditions (C) and during aortic constriction (Ao). The only significant change during aortic constriction was the reduction in shortening at the base (P < 0.02). NS = not significant.

Simultaneous epicardial and midwall tracings obtained at the apical location. Arrows on the left indicate the onset of lengthening of the epicardial segment and simultaneous shortening of the midwall segment, and those on the right indicate the onset of epicardial shortening simultaneous with an increase in the rate of midwall shortening. ECG = electrocardiogram, and LV = left ventricular.
relative to rotational effects of this magnitude (19), it is unlikely that this change would have influenced the observed shortening at this location. At all levels, the epicardial extent of shortening was substantially less than that observed in the hoop fibers. The difference could account for the higher levels of systolic shear observed by Fiegl and Fry (27) at the midwall. In addition, the timing differences noted in simultaneous epicardial and midwall studies suggest that during isovolumic systole shear forces result from bulging of the epicardial fibers at the same time that the midwall fibers are shortening but that during ejection the production of shear forces is the result of the differential extent and velocity of shortening at the two levels.

The effects of increased aortic impedance in the intact, innervated left ventricle are dependent on the magnitude of the rise in systolic pressure, the associated increase in end-diastolic pressure, and the timing of measurements with respect to the onset of the intervention (28-30). Our own measurements were made as soon as possible after both left ventricular peak systolic pressure and segment length appeared to have reached steady-state levels but at least 20 seconds after the onset of the constriction (range 20 to 50 seconds). In the present study, apical and midventricular midwall shortening was maintained during aortic constriction, but basal shortening decreased. Epicardial shortening did not change significantly with aortic constriction. From these results it would appear that the major change in left ventricular geometry during aortic constriction of this degree is an increase in end-diastolic segment length at the midventricular and apical locations with little change at the base. The observed changes in end-diastolic segment length suggest that shortening is maintained at the apical and midventricular levels via the Frank-Starling mechanism but that this mechanism cannot be utilized at the base to maintain shortening. The lack of change in end-diastolic length at the basal portion of the left ventricle may be related to the proximity of these fibers to the relatively inelastic fibrous portion of the cardiac skeleton comprising the mitral and aortic valve rings.

Previous investigators have demonstrated that stroke volume either remains unchanged or decreases coincident with an increase in aortic impedance while ventricular volume increases (30-32). Based on such changes in cavity size and stroke volume, it would be expected that the regional extent of shortening would decrease. In fact, for the midwall fibers, we observed a rather large decrease in the extent of shortening only at the base, with no statistically significant change in

### Table 2

<table>
<thead>
<tr>
<th>Segment Length (cm)</th>
<th>Midventricular-segment</th>
<th>Base</th>
</tr>
</thead>
<tbody>
<tr>
<td>C</td>
<td>Ao</td>
<td>C</td>
</tr>
<tr>
<td>Ext. no.</td>
<td></td>
<td></td>
</tr>
<tr>
<td>C</td>
<td>Ao</td>
<td>EDL</td>
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<tr>
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<td>12</td>
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<td>175</td>
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<tr>
<td>Mean</td>
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<td>165</td>
</tr>
<tr>
<td>± SE</td>
<td>5.7</td>
<td>5.1</td>
</tr>
</tbody>
</table>

**Abbreviations are the same as in Table 1.**

*Adverse effects from the apical and midventricular locations could not be obtained in this experiment.
other areas. The epicardial fibers showed no significant changes at any site. Our observations may be in contrast with those of Bugge-Asperheim and Kiil (33), who used a similar technique and noted a decrease in the extent of shortening with increased aortic impedance. However, in their study only one segment was examined, and no information was provided as to the placement of the ultrasonic gauge with respect to the long axis, fiber orientation, or wall thickness. Thus, if the gauge was placed near the basal left ventricle, our results may not be in disagreement. The significant implication of these results may be that the response of the left ventricle to an increase in aortic impedance within the context of this particular experimental setting is not homogeneous. Such a nonhomogeneous response may be a further indication of significant dynamic regional differences in left ventricular performance during various stresses, in addition to those observed under control conditions.

Several advantages offered by the use of ultrasound crystals for the study of local myocardial function deserve mention (16–19). They are small, relatively atraumatic, and highly accurate; they provide excellent resolution and are well suited for chronic implantation. The crystals themselves interfere little, if at all, with myocardial function and exert essentially no tension on the myocardium. These properties are in contrast to those of a variety of other devices which have been used to study myocardial segment length or circumference (6, 7, 34). Most importantly, as demonstrated by Bugge-Asperheim et al. (35), devices such as the mercury-in-Silastic strain gauge may introduce significant artifacts by virtue of the degree to which they exert prestretch on the length of myocardium to be studied while their construction precludes measurements of small distances.

By utilizing ultrasound crystals to examine short segments of myocardium with very little curvature, it is possible to analyze regional shortening in a manner that minimizes the effects of overall geometric changes and curvature within the ventricle. Another potentially important advantage in evaluating local myocardial shortening is that shortening is measured along fibers which are oriented in parallel to the measuring device, thus minimizing the effects of variations in fiber orientation. For example, using our postmortem placement data and the fiber angle data of Streeter et al. (13), the midwall gauges used in the present study would have subtended fibers with no more than a 10–15° variation in orientation.

From a technical standpoint, analysis of regional left ventricular shortening in humans must of necessity be less precise than that in animal models. Nonetheless, the reasonably good agreement between our own results in the dog and those obtained in humans utilizing other techniques (14, 15) suggests that the phenomenon of increased apical shortening on a local segmental level probably exists in the human left ventricle. Although a uniformly contracting ellipsoidal model for both the human and the canine left ventricle appears to be both a useful and a practical approach (1) for the assessment of left ventricle performance, our observations indicate that significant differences in regional performance do exist both at rest and in response to increased aortic impedance. It is thus likely that such a model represents an oversimplification of the actual geometric changes occurring during systole.

**Appendix 1**

The analysis showing that the ellipsoid of revolution model predicts a uniform midwall hoop fiber shortening at all levels along the major axis was made as follows. (1) The left ventricle can be represented as an ellipsoid of revolution whose projection in the frontal or the lateral plane is an ellipse. (2) \( l_s/L_s = l_d/L_d \), where \( L_s \) and \( L_d \) are the major (base-to-apex) semiaxes at end-systole and end-diastole, respectively, and \( l_s \) and \( l_d \) represent the position of a single point along the \( L \) axis at end-systole and end-diastole, respectively. This condition implies that the point retains its relative position on the \( L \) axis during contraction. (3) \( R_s \) and \( R_d \) are the minor semiaxes (at the base of the ventricle) during systole and diastole respectively, and \( r_s \) and \( r_d \) represent the radius in systole and diastole, respectively, of a parallel circle of the ellipsoid with centers at \( l_s \) and \( l_d \), respectively. From the general formula for an ellipse:

\[
r_a = R_a \sqrt{1 - \left( \frac{l_a}{L_a} \right)^2},
\]

\[
r_s = R_s \sqrt{1 - \left( \frac{l_s}{L_s} \right)^2}
\]

From point 2 above,

\[
\frac{r_e}{R_e} = \frac{r_s}{R_s} = \sqrt{1 - \left( \frac{l_s}{L_s} \right)^2}
\]

Solving for \((r_e - r_s)/r_s\),

\[
\frac{r_e - r_s}{r_s} = \frac{R_e - R_s}{R_s}
\]

Thus, the percent radial or the percent circumferential shortening at any \( l \) is equal to the percent radial (circumferential) shortening at the minor axis \( R \).

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