ALTERATION OF LEFT VENTRICULAR GEOMETRY DURING PRELOAD REDUCTION AND AFTERLOAD INCREMENT

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To ascertain whether or not left ventricular geometry changes during preload reduction and afterload increment, the shortening characteristics of small segments in the left ventricular free wall were examined using 4 pairs of ultrasonic crystals in 10 dogs. Three pairs of ultrasonic crystals were circumferentially implanted in the basal, the midventricular and the apical portion of the left ventricle. Another pair of crystals were longitudinally placed in the midventricle. In the control state, the shortening at the apex was largest of all segments. During preload reduction, the end-diastolic length decreased significantly in each segment. The percent shortening decreased significantly at the apical and the longitudinal segment, but it remained unchanged at the midventricular and the basal segment. During afterload increment, the end-diastolic length increased significantly, but the percent shortening remained unchanged in each segment. We concluded that left ventricular geometry was altered during preload reduction and that the apical part is more responsive to preload change than the other portion.

The size and shape of the left ventricular chamber are major determinants of cardiac function. Therefore many investigators have been interested in the left ventricular geometry and have studied left ventricular function such as left ventricular pressure-volume relationships. Left ventricular volume is often calculated from diameters of the left ventricle because of the difficulty in measuring left ventricular volume directly. This model assumes the left ventricle to be an ellipsoid. However, Rankin et al. showed that the shape of the left ventricle changed according to the left ventricular volume. LaWinter et al. showed that there were significant differences in the dynamic geometry of the myocardium during afterload increment. Hence it is a good possibility that there are regional differences in the left ventricular geometry according to preload or afterload changes. On the other hand, Olsen et al. showed a homogeneity of left ventricular strain during ejection phase. Thus the dynamic geometry of the left ventricle is controversial. Preload reduction seems to bring about the larger change of left ventricular geometry, but this has not been examined explicitly. The aim of this study was to determine whether or not regional differences existed in left ventricular segmental shortening as a result of preload reduction or afterload increment.

METHODS

Ten mongrel dogs weighing from 9 to 20 kg (average 14.5 kg) were anesthetized with sodium pentobarbital (25 mg/kg, iv), and small supplementary doses were administered as required. The dogs were intubated, and respirated with a Harvard respiratory supplying room air. A

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Fig. 1. The positions of crystal pairs in relation to the long axis of the left ventricle. LAD = left anterior descending coronary artery, LCX = left circumflex coronary artery, BASE = basal segment, MID = midventricular segment, APEX = apical segment, LONG = longitudinal segment.

Thoracotomy was performed in the fourth left intercostal space. The heart was suspended in a pericardial cradle. A micromanometer-tipped catheter (7F, TCP1RN137F120, TOYOTA) was inserted into the left ventricle via a carotid artery. A fluid-filled catheter was inserted into the left ventricle through a small stab incision at the apex and was used to obtain zero reference pressure and to calibrate the high fidelity micromanometer. A silicone rubber hydraulic occluder was placed around the inferior vena cava to diminish venous return. An umbilical tape was placed around the descending thoracic aorta to increase aortic pressure. Segment lengths were measured by using pairs of ultrasonic crystals of 2–3 mm in diameter. Three pairs of ultrasonic crystals were placed in the free wall of left ventricular myocardium at the midwall depth; one pair at 10–15 mm from the bifurcation of the left coronary artery, one at 10–15 mm from the apical dimple, and another one approximately halfway between the basal and the apical pairs.

Fig. 2. Original tracings from experiments in a single dog showing the control state (A), vena caval occlusion (B), and aortic constriction (C).
TABLE 1  HEMODYNAMIC DATA AND LEFT VENTRICULAR SEGMENT LENGTHS IN THE CONTROL STATE, DURING VENA CAVAL OCCLUSION, AND DURING AORTIC CONSTRUCTION

<table>
<thead>
<tr>
<th></th>
<th>Apex</th>
<th>Mid</th>
<th>Long</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>HR</td>
<td>LVESP EDL</td>
<td>ESL</td>
</tr>
<tr>
<td>Control</td>
<td>128</td>
<td>114</td>
<td>16.6</td>
</tr>
<tr>
<td></td>
<td>SD</td>
<td>17.4</td>
<td>2.2</td>
</tr>
<tr>
<td>Vena cava occl</td>
<td>mean</td>
<td>111</td>
<td>81**</td>
</tr>
<tr>
<td></td>
<td>SD</td>
<td>27.2</td>
<td>2.5</td>
</tr>
<tr>
<td>Aortic constr</td>
<td>mean</td>
<td>125</td>
<td>164**</td>
</tr>
<tr>
<td></td>
<td>SD</td>
<td>25.6</td>
<td>2.9</td>
</tr>
</tbody>
</table>

HR = heart rate; LVESP = left ventricular peak systolic pressure; EDL = end-diastolic length; ESL = end-systolic length; %ΔL = (EDL/ESL) / EDL × 100.  **Different from control state, p < 0.001.  *Different from control state, p < 0.05.

Data were collected from each dog during a baseline control period, during occlusion of the inferior vena cava and during constriction of the descending thoracic aorta. Control measurements were made before and after the each manipulation. The hydraulic cuff around the inferior vena cave was inflated gradually to restrict the venous return. The systemic pressure was allowed to decline while 10–30 beats were recorded, then, the hydraulic cuff was released. Constriction of the descending thoracic aorta was done by gradually tightening an umbilical tape placed around the vessel. The aortic constriction was regulated to produce an increase of about 50 mmHg in the peak systolic left ventricular pressure. The percent shortening at each segment was expressed as percent extent of shortening to end-diastolic length.

The end-systolic pressure-length relationships were also examined. The time of end-systole was defined as 20 msec before the peak negative dP/dt. The end-systolic pressure-length relationship was derived from a linear regression analysis on 10–20 consecutive beats during the increasing pressure. The segment length was normalized so that the end-diastolic length in the control state was 10 mm. Statistical evaluation was done using analysis of variance with repeated measures and paired t-test for paired samples. When statistical significance was established through the analysis of variance, Duncan’s test was used to determine which segment was significantly different. The level of statistical significance was p < 0.05, and

namely at the midventricular level. These 3 pairs were placed perpendicularly to the longitudinal axis of the left ventricle. They were parallel to the direction of the hoop fibers, according to the description by Streeter et al. In 5 of 10 dogs, another pair of crystals was placed in the free wall of the left ventricular myocardium, parallel to the longitudinal axis (Fig. 1).

Heart rate, left ventricular peak systolic pressure, left ventricular end-diastolic pressure and segment lengths (distance of paired crystal) were studied before and after the inferior vena cava occlusion or aortic constriction. All data were recorded at a paper speed of 100 mm/sec with respiration suspended at end-expiration. These measurements were recorded on an 8-channel thermal pen recorder (RECTI-HORIZ-8K, SAN-EI) and stored on magnetic tape (FE-39A, SONY) for subsequent data analysis.

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the data are presented as the mean ± SD.

RESULTS

Representative tracings obtained from a dog are shown in Fig. 2. The average hemodynamic and segment length data characterizing these interventions are presented in Table I.

In the control state, the mean percent shortening of the apical, the midventricular, the basal, and the longitudinal segments was 25 ± 4.2%, 20 ± 3.8%, 16 ± 4.2% and 16 ± 4.2% and 16 ± 5.8%, respectively (Fig. 3). The shortening of the apical segment was significantly larger than that of the midventricular (p < 0.05), the basal (p < 0.01) or the longitudinal segment (p < 0.01). There was a significant difference in the percent shortening between the midventricular and the basal segment (p < 0.05).

During occlusion of the inferior vena cava, left ventricular peak systolic pressure decreased from the control value of 114 ± 17.4 mmHg to 81 ± 12.0 mmHg and left ventricular end-diastolic pressure also decreased from 7.3 ± 3.7 to 4.3 ± 2.5 mmHg (p < 0.05). Heart rate did not change significantly. End-diastolic length decreased in all 4 segments (Fig. 4); the decrease of the apical, the midventricular, the basal and the longitudinal segment were 8%, 6%, 5% and 6%, respectively. End-systolic length of the apical,
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Fig. 5. Mean percent shortening at each crystal site under control conditions (open bar), during vena caval occlusion (lined bar), and aortic constriction (dotted bar). *p < 0.05; **p < 0.01

Fig. 6. Left ventricular end-systolic pressure-length relationship produced by aortic constriction in one dog. LV = left ventricular. BASE = basal segment. MID = midventricular segment. APEX = apical segment

the midventricular and the basal segment decreased by 3%, 4% and 5%, respectively (Fig. 4). End-systolic length of the longitudinal segment did not change significantly. The percent shortening is presented in Fig. 5. While the inferior vena cava was occluded, the percent shortening of the midventricular and basal segments occurred to approximately the same extent as control. However, the shortening of the apical and the longitudinal segment decreased significantly, compared with control value. The apical shortening decreased from 25 ± 4.2% to 21 ± 5.3% (p < 0.01) and the longitudinal shortening decreased from 16 ± 5.8% to 12 ± 4.0% (p < 0.05).

During aortic constriction, left ventricular peak systolic pressure increased from the control value of 114 ± 17.4 mmHg to 164 ± 16.5 mmHg. However, left ventricular end-diastolic pressure and heart rate were not significantly changed. End-diastolic length increased significantly by 3% in all 4 segments (Fig. 4). End-systolic length of the apical, the midventricular and the basal segment increased significantly by 4%, 4% and 3%, respectively (Fig. 4). End-systolic length of the longitudinal segment also increased by 3%, but was not statistically significant. The percent of shortening was unchanged in all 4 segments during aortic constriction (Fig. 5).

The end-systolic pressure-length relationships at all three circumferential segments were linear except in two dogs at the apical segment; \( r = 0.96 \pm 0.03 \) (8 dogs) at the apex, \( r = 0.98 \pm 0.01 \) (10 dogs) at the midventricle and \( r = 0.97 \pm 0.01 \) (10 dogs) at the base. A representative example of the pressure-length relationship at the midventricle was shown in Fig. 6. The slope of the end-systolic pressure-length relationship at the apex, the midventricle, and the base was 106 ± 49 mmHg/mm, 138 ± 113 mmHg/mm, and 159 ± 122 mmHg/mm, respectively. The statistical level of the slopes evaluated by an analysis of variance was not significant. The extrapolated X-axis intercept (\( L_0 \)) at the apex, the midventricle, and the base were 8.70 ± 0.57 mm, 8.75 ±
0.85 mm, and 8.86 ± 1.05 mm, respectively. There was no difference in L₀.

DISCUSSION
The alterations in left ventricular geometry have been extensively investigated. Many investigators have used the pressure-volume relationship as an indicator of left ventricular contractility. The left ventricular volume was conventionally calculated from a minor axis dimension of left ventricle by using echocardiogram. They assumed that the left ventricular muscle was homogeneous and that the left ventricle was ellipsoid. However, modeling the left ventricle as a generalized ellipsoidal shell also has many limitations. Changes of left ventricular shape were reported in patients with valvular heart disease. Recent studies indicate that regional shortening and left ventricular shape are influenced differently by preload and afterload.

In the present study, the reduction of shortening of the apical and the longitudinal segment was actually greater than that of the midventricular and the basal segment during vena caval occlusion. These differences in the shortening of the segments could be accounted for by the difference between the change of end-diastolic segment length and that of end-systolic segment length during vena caval occlusion. That is, the decrement in end-diastolic length was larger than that in end-systolic length. The decreases of percent change of end-diastolic segment length in the apical and the longitudinal segment were larger than those of the midventricular and the basal segment. The decreases of percent change of end-systolic segment length in the apical and the longitudinal segment were smaller than those of the midventricular and the basal segment. But there were no statistical differences in percent change of end-diastolic and end-systolic length among each 4 segment during vena caval occlusion. These results indicated that the reduction of preload influenced the apical portion much more than the other portions of the left ventricle. This evidence supports the earlier reports that the end-diastolic left ventricular chamber becomes more spherical as the left ventricular end-diastolic volume increases.

On the other hand, during aortic constriction the end-diastolic segment length increased in all four segments. However, the shortening was almost constant in all 4 segments. These findings could signify that there was no regional difference in the percent shortening under the afterload change in our study. Probably the major change in the left ventricular geometry induced an uniform increase in both end-diastolic segment length and end-systolic segment length. The observed changes in the segment length suggested that shortening was maintained according to the Frank-Starling mechanism without ventricular shape change. LeWinter et al. observed that a significantly greater hoop axis shortening occurred near the apex of the left ventricle, and that the response of the hoop axis fibers to increase of aortic pressure was not homogeneous, with a significant reduction in shortening occurring only at the base of the left ventricle where end-diastolic length does not increase. The difference between the results of our study and theirs could be caused by the difference of the degree of afterload and preload. During aortic constriction, peak systolic left ventricular pressure was similar in both studies. However, the left ventricular end-diastolic pressure was different. At the control state, left ventricular end-diastolic pressure was 7.3 mmHg in our study, but it was 4.8 mmHg in their study. The difference of preload at control state may be attributed to the difference in the response to the increased afterload. Although left ventricular end-diastolic pressure did not change during aortic constriction in our study, it increased from 4.8 mmHg to 8.3 mmHg in their study. In their study, an increase of the afterload may have induced a compensatory increase of the ventricular end-diastolic volume or a rise in ventricular preload. These differences of the segmental shortening behavior seem to be causally related to the difference in the afterload and compensatory preload. If the left ventricular afterload was greater in our study, the basal shortening might be as small as in their study.

When the regional geometry of the left ventricle is discussed, we must consider the left ventricular contractility in the present study. But it is difficult to evaluate a regional contractility of each segment in the left ventricle. We tried to utilize the pressure-length relationship as an index of the regional contractility. The end-systolic pressure-length relationships at all 4 segment were linear. The slope of end-systolic pressure-length relationship at all 4 segment were not different statistically and also there was no difference in L₀. The end-systolic pressure-length relationship is not always equal to the end-systolic pressure-volume relationship.
However, the end-systolic pressure-length relationship is thought to be useful as a substitute for the end-systolic pressure-volume relationship when there is no regional wall motion abnormality. As intercrystal distance in our study was short, we thought the regional difference of curvature could be ignored. Therefore, the end-systolic pressure-length relationship in our study could reflect the left ventricular contractile function. Then these results may show that there was no difference in contractile function among the portions of the left ventricle.

On the other hand, Laks et al. reported that there was a significant difference between the sarcomere lengths at the base and the apex of the left ventricle. The sarcomere lengths at the left ventricular base were shorter, compared to those at the apex. They considered that the basal portion of left ventricle had the least distensibility, because it contains the largest number of muscle fibers. Since shorter sarcomere lengths result in a lesser work output on the ascending limb of the Frank-Starling curve, the sarcomeres in the base were considered to perform less mechanical work. These findings support our findings that the segmental shortening at the apex was largest and that the apical segment was more sensitive to the preload reduction than the other portion.

We considered that the apical region of left ventricle perform the largest mechanical work. Reduction of the apical wall motion was reported in patients with chronic volume overload such as aortic regurgitation and mitral regurgitation. Myocardial infarction with power failure usually extends the apex. That is, if myocardial infarction included the apex, power failure could develop easily. Not only is the size of damaged myocardium important, but the localization of damaged part is important.

In summary, we have examined the regional differences of left ventricular segment shortening during vena caval occlusion and aortic constriction. The reduction of the segment shortening was observed in the apical and the longitudinal segments during vena caval occlusion. However, the shortening was almost constantly maintained in all 4 segments during aortic constriction.

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