Nonhomogeneous Left Ventricular Regional Shortening During Acute Right Ventricular Pressure Overload

Yoichi Goto, Bryan K. Slinker, and Martin M. LeWinter

Acute right ventricular pressure overload shifts the interventricular septum leftward and decreases systolic shortening of the left ventricular (LV) septal-lateral diameter. These changes should alter regional shortening in the LV minor axis. To test this hypothesis, LV minor axis circumferential segment lengths of the septum and anterior, lateral, and posterior walls were measured during pulmonary artery or venae caval constriction in seven open-chest dogs with intact pericardia. Starting at an end-diastolic pressure of 10 mm Hg, venae caval constriction decreased LV end-systolic pressure by 19±6% and stroke volume by 40±15% and produced uniform decreases in systolic shortening and end-diastolic length around the minor axis. However, during pulmonary artery constriction resulting in similar decreases in end-systolic pressure (22±7%) and stroke volume (39±11%), decreases in systolic shortening were significantly larger in the anterior (-34±10%) and posterior (-33±21%) walls than in the septum (-10±9%) or lateral wall (-8±13%). The mechanisms of these large anterior and posterior shortening decreases differed: anterior end-diastolic length decreased more than posterior and lateral end-diastolic lengths, while posterior end-systolic length decreased less than anterior and lateral end-systolic lengths. Similar changes were seen at starting end-diastolic pressures of 5 and 15 mm Hg. Propranolol did not alter this nonuniform response, while pericardiectomy attenuated the regional variations. Thus, changes in LV geometry during acute right ventricular pressure overload are associated with nonuniform regional changes in systolic shortening in the LV minor axis that are enhanced by the pericardium. (Circulation Research 1989;65:43–54)
Materials and Methods

Experimental Preparation
Thirteen random source dogs (21–30 kg) premedicated with 1 mg/kg subcutaneous morphine sulfate were anesthetized with sodium pentobarbital (25 mg/kg i.v.), intubated, and ventilated with a respirator (Harvard Apparatus, South Natick, Massachusetts). A median sternotomy and bilateral fifth intercostal thoracotomy were performed in each dog. A limb lead electrocardiogram was monitored throughout the experiment. Micromanometer catheters (7F; Millar Instruments, Houston, Texas) with fluid-filled and micromanometer catheters were matched at the beginning of the experiment and intermittently during the experiment to correct for zero drift. A polyvinyl tube for intravenous fluid administration was introduced into the LV free wall from the right femoral vein. Both lumens were connected to pressure transducers (model P23XL, Gould Instruments, Inc, Cleveland, Ohio) with zero reference at the level of the right atrium. Pressures from the fluid-filled and micromanometer catheters were matched at the beginning of the experiment and intermittently during the experiment to correct for zero drift. A polyvinyl tube for intravenous fluid administration was introduced into the left femoral vein.

A small longitudinal slit (2 cm in length) was made in the pericardium, an electromagnetic flow probe was placed around the ascending aorta, and a snare was placed around the pulmonary artery. The azygos vein was ligated, and snares were placed around the inferior and the superior venae cavae.

Placement of ultrasonic crystals. In 11 dogs, four pairs of 5 MHz piezoelectric crystals (2 mm in diameter) were placed in the LV free wall and the interventricular septum to measure four circumferential segment lengths in the LV minor axis (Figure 1). Plastic introducer sleeves were constructed so that the free-wall crystals were placed at the same midwall depth of 5 mm. All crystals were inserted through small slits (3 mm in length) in the pericardium so that the pericardium was kept virtually intact.

The LV free-wall crystals were placed according to an external reference system similar to Freeman et al10 and Lew and LeWinter:14 The anterior long axis was the line connecting the bifurcation of the left main coronary artery and the apical dimple10; the posterior long axis was the line connecting the inferior pulmonary vein and the apical dimple and roughly parallel to the posterior interventricular branches of the left circumflex coronary artery; and the lateral long axis was the line midway between the anterior and posterior external long axes. Three pairs of free-wall segment crystals, that is, anterior, lateral, and posterior, were implanted perpendicular to the corresponding external long axes. The level at which crystals were implanted along the course of the external long axis was determined visually to be 50–60% of the distance from the apex to the base. Crystals were placed so that each segment would be 1–2 cm in length.

The septal segment crystals were placed in the same minor axis plane as the LV free-wall segments (Figure 1). They were introduced from the LV anterior and posterior walls by means of long plastic sleeves (5 cm in length) through small pericardial slits, adjacent to the left anterior descending coronary artery and the posterior descending branch of the left circumflex coronary artery.

In two other dogs, three circumferential segment lengths in the LV free wall (place as above) and the LV septal-lateral diameter were measured. To measure the LV septal-lateral diameter, the fourth pair of crystals was placed in the subendocardium of the left side of the interventricular septum and the LV lateral wall again with the pericardium intact.

Postmortem examination. At the completion of each experiment, proper crystal position was confirmed. In each LV wall region the distance from the apex to the crystals and the total apex-to-base distance were measured, and the ratio of the former distance to the latter, that is, the relative position of the crystals along the course of the external long axis, was calculated. The relative crystal positions along the course of the external long axis were not significantly different among the wall regions (septum 55.0±5.5%, anterior wall 57.9±6.3%, lateral wall 58.3±3.6%, posterior wall 58.2±4.5%). This result indicates that all of the four circumferential segments were located in the LV minor axis equatorial plane. Further, all LV free wall crystals were at a similar depth in the midwall layer. All paired septal segment crystals were in the same one-third layer of the interventricular septum: nine pairs were located in the left one third and two pairs were in the middle one third of the septum.

Experimental Protocol
In seven of the 11 dogs with four pairs of circumferential segment crystals, we assessed changes in regional systolic shortening during either superior and inferior VC or PA constriction with the pericardium intact. This we refer to as the main protocol. VC or PA constriction was initiated in random
order at a starting LV end-diastolic pressure of approximately 5 mm Hg. Aortic flow, LV pressure, right ventricular pressure, and lengths of the septal, anterior, lateral, and posterior segments were measured before and during the intervention while respiration was suspended at end-expiration. The magnitude of VC and PA constrictions was adjusted to produce a 20–25% decrease in LV systolic pressure within 5–7 seconds.

To examine the influence of changes in LV volume on regional responses to VC and PA constriction, similar measurements were repeated at higher starting LV end-diastolic pressures of approximately 10 and 15 mm Hg. LV volume was increased by infusing warmed 6% dextran solution in 0.9% saline. Measurements were made after a new steady state was reached at each LV end-diastolic pressure.

To determine the effect of the pericardium on regional responses to VC and PA constriction, measurements were repeated after pericardiectomy in six of the seven dogs. The pericardium was opened widely, and a loose pericardial cradle was made to prevent excessive cardiac movement. Because regional responses to VC or PA constriction were similar among the three different starting LV end-diastolic pressures with the pericardium intact (see “Results”), measurements were made only at a starting LV end-diastolic pressure of 10 mm Hg after pericardiectomy.

In four other dogs with four pairs of circumferential segment crystals, the effect of β-blockade on the response of regional segment shortening to PA constriction was examined. Measurements were made at a starting LV end-diastolic pressure of 10 mm Hg with the pericardium intact before and after propranolol (1 mg/kg i.v.).

In the remaining two dogs with three pairs of LV free-wall segment crystals and the septal-lateral diameter crystals, changes in LV septal-lateral diameter shortening and the correlation between the diameter and segment lengths were assessed during VC and PA constriction at starting LV end-diastolic pressures of 10 and 15 mm Hg.

Data Analysis

Data were recorded on a pen recorder and also converted from analog to digital on a DEC PDP 11/73 computer at a sampling interval of 5 msec. End diastole was defined as the time when LV dP/dt increased to 10% of its peak value. End systole was defined as the time 30 msec before peak negative dP/dt. This end-systolic timing was selected because, when we constructed LV pressure-length loops, the point 30 msec before peak negative dP/dt was consistently closer to the left upper corner of the loops than points 20 and 40 msec before peak negative dP/dt. The extent of systolic shortening was calculated as the end-diastolic segment length minus the end-systolic length, divided by the end-diastolic length, times 100. The data recorded during control periods before VC and PA constriction were called Control 1 and 2, respectively, regardless of the actual order. Changes from control during VC or PA constriction were assessed by comparing two selected beats: one from steady-state contractions obtained during control, and the other from quasi steady-state contractions obtained during VC or PA constriction.

In the seven dogs used for the main protocol, time from the control beat to the selected beat during VC or PA constriction averaged 4.4±1.9 sec (n=54), during which time reflex increases in heart rate and ventricular contractile state were expected to be minimal. Data were expressed as percent changes from the respective control values.

To assess changes in the LV end-systolic and end-diastolic pressure-segment length relations, we normalized segment length data to the end-diastolic length of each segment during VC constriction beginning at an LV end-diastolic pressure of 5 mm Hg, that is, to the length at the lowest measured LV end-diastolic pressure (this pressure averaged 1.8±1.4 mm Hg).  

Statistics

Hemodynamic and dimension data during Control 1, VC constriction, Control 2, and PA constriction at the same starting LV end-diastolic pressure were compared by repeated measures analysis of variance. When this indicated a significant difference among the four conditions, the least significant difference (LSD) method was used to determine the significance of difference between the conditions. Comparisons of dimension data among different regions were made by the same method. Decreases in LV end-systolic pressure and stroke volume during VC and PA constrictions were compared by a paired t test. Values of p<0.05 were presented as statistically significant. Data are presented as the mean±SD unless otherwise indicated.

Results

In the main protocol, control LV end-diastolic pressures before VC constriction averaged 5.1±1.5, 9.4±2.5, and 13.1±2.4 mm Hg with the pericardium intact (target pressure of 5, 10, and 15 mm Hg, respectively), and 9.8±3.3 mm Hg with the pericardium open (target pressure of 10 mm Hg). Control LV end-diastolic pressures before PA constriction averaged 5.6±1.5, 10.4±3.5, and 14.4±2.1 mm Hg with the pericardium intact and 9.7±4.4 mm Hg with the pericardium open. For convenience, these will subsequently be referred to as starting LV end-diastolic pressures of 5, 10, and 15 mm Hg.

Figure 2 shows an example of a recording made during PA constriction at a starting end-diastolic pressure of 15 mm Hg with the pericardium intact. End-diastolic length decreased in all four circumferential segments and shortening most noticeably decreased in the anterior and posterior segments during PA constriction.

Table 1 summarizes hemodynamic data before...
FIGURE 2. A sample recording of left ventricular (LV) and right ventricular (RV) pressures and four segment lengths in the LV minor axis during control (left) and pulmonary artery (PA) occlusion (right).

and during VC and PA constriction at a starting LV end-diastolic pressure of 10 mm Hg with the pericardium intact. Heart rate did not significantly change. Decreases in LV end-systolic pressure during VC (−18.5 ± 6.2%) and PA constriction (−22.4 ± 7.2%) were similar (p > 0.2), as were decreases in stroke volume (−40.1 ± 15.0% versus −38.5 ± 11.2%, p > 0.5), indicating that the effects of both VC and PA constriction on LV preload reduction were comparable. As expected, right ventricular peak systolic and end-diastolic pressures decreased during VC constriction, whereas they significantly increased during PA constriction.

<table>
<thead>
<tr>
<th></th>
<th>Control 1</th>
<th>VCC</th>
<th>Control 2</th>
<th>PAC</th>
</tr>
</thead>
<tbody>
<tr>
<td>HR (beats/min)</td>
<td>126±26</td>
<td>125±25</td>
<td>126±28</td>
<td>128±27</td>
</tr>
<tr>
<td>LVESP (mm Hg)</td>
<td>125±16</td>
<td>102±17*</td>
<td>123±17</td>
<td>96±16*</td>
</tr>
<tr>
<td>LVEDP (mm Hg)</td>
<td>9.4±2.5</td>
<td>4.1±1.4*</td>
<td>10.4±3.5</td>
<td>6.6±3.5*</td>
</tr>
<tr>
<td>RVSP (mm Hg)</td>
<td>31.7±6.6</td>
<td>17.3±4.9*</td>
<td>34.3±7.1</td>
<td>67.6±12.6*</td>
</tr>
<tr>
<td>RVEDP (mm Hg)</td>
<td>6.3±3.5</td>
<td>1.8±2.3*</td>
<td>7.2±4.2</td>
<td>10.3±4.6*</td>
</tr>
<tr>
<td>Stroke volume (ml)</td>
<td>16.9±6.9</td>
<td>10.1±5.0*</td>
<td>17.5±7.2</td>
<td>10.6±5.0*</td>
</tr>
</tbody>
</table>

VCC, venae caval constriction; PAC, pulmonary artery constriction; HR, heart rate; LVESP, left ventricular end-systolic pressure; LVEDP, left ventricular end-diastolic pressure; RVSP, right ventricular peak systolic pressure; RVEDP, right ventricular end-diastolic pressure.

All values are mean±SD.

*p<0.01 compared with the corresponding control.

Dimension data at a starting LV end-diastolic pressure of 10 mm Hg with the pericardium intact are summarized in Table 2 and Figure 3. During VC constriction, end-diastolic length and systolic shortening significantly decreased in all four segments. The percent decreases in systolic shortening during VC constriction were not significantly different (p > 0.25) among the four segments (Figure 3, left), indicating that circumferential systolic shortening in the LV minor axis decreased uniformly. During PA constriction, end-diastolic length significantly decreased in all four segments (Table 2). However, in contrast to the results during VC constriction, percent decreases in systolic shortening in anterior (−33.5 ± 10.2%) and posterior segments (−33.4 ± 20.9%) were significantly greater (p < 0.01) than those in septal (−10.5 ± 8.8%) and lateral segments (−7.9 ± 13.3%), indicating that the decreases in circumferential regional shortening were nonuniform in the LV minor axis (Figure 3, right).

To elucidate the mechanisms responsible for the greater decreases in the anterior and posterior segment shortening during PA constriction, changes in end-diastolic and end-systolic lengths were compared in the three segments in the LV free wall during PA constriction (Figure 4). The septal segment was excluded from this analysis because a significant change in radius of curvature of the septum, which should occur during PA constriction, might have caused some error in the assessment of septal segment length (see “Appendix”). As shown in Figure 4 (left), the percent decrease in end-diastolic length in the anterior segment (−5.7 ± 2.0%) was significantly greater than in the lateral (−3.6 ± 1.5%) and posterior segments (−3.0 ± 2.6%) during PA constriction. In contrast, the percent change in end-systolic length in the posterior segment (1.3 ± 4.0% insignificant change from control, p > 0.4) was significantly smaller than the other two segments (anterior −2.3 ± 1.9%, lateral −2.6 ± 2.3%), both of which decreased significantly (p < 0.05) from their control values (Figure 4, right).

Figure 5 shows the normalized end-systolic and end-diastolic pressure-segment length relations in the three LV free-wall segments during control and VC and PA constriction at a starting end-diastolic pressure of 10 mm Hg with the pericardium intact.
TABLE 2. Dimension Data at a Starting Left Ventricular End-Diastolic Pressure of 10 mm Hg With the Pericardium Intact

<table>
<thead>
<tr>
<th>Segment</th>
<th>Control 1</th>
<th>VCC</th>
<th>Control 2</th>
<th>PAC</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>EDL (mm)</td>
<td>SS (%)</td>
<td>EDL (mm)</td>
<td>SS (%)</td>
</tr>
<tr>
<td>Septal segment</td>
<td>30.4±3.6</td>
<td>22.5±8.3*</td>
<td>30.6±3.9</td>
<td>31.0±7.9</td>
</tr>
<tr>
<td>Anterior segment</td>
<td>14.2±1.6</td>
<td>7.9±2.4*</td>
<td>14.3±1.6</td>
<td>10.2±2.4</td>
</tr>
<tr>
<td>Lateral segment</td>
<td>14.9±4.3</td>
<td>9.7±3.8t</td>
<td>15.0±4.3</td>
<td>11.9±2.1</td>
</tr>
<tr>
<td>Posterior segment</td>
<td>12.4±3.9</td>
<td>11.6±4.7*</td>
<td>12.6±4.0</td>
<td>13.0±3.5</td>
</tr>
</tbody>
</table>

VCC, vena caval constriction; PAC, pulmonary artery constriction; EDL, end-diastolic length; SS, systolic shortening.

All values are mean±SD.
*p<0.01 compared with the corresponding control.
1p<0.05 compared with the corresponding control.

pressure of 10 mm Hg. To assess shifts of the end-systolic and end-diastolic pressure-length relations during PA constriction, we compared the relative position of the single data point obtained during PA constriction to the relations obtained during control and VC constriction. In the anterior segment, there was no major shift of the end-systolic pressure-length relation during PA constriction. However, the end-diastolic pressure-length relation during PA constriction shifted markedly leftward; that is, end-diastolic pressure was significantly higher during PA constriction than during VC constriction \( (>0.01) \) for a similar normalized end-diastolic segment length. In the lateral segment, neither the end-systolic nor end-diastolic pressure-length relation showed a major shift during PA constriction. In contrast, the end-systolic pressure-length relation of the posterior segment during PA constriction shifted rightward; that is, normalized end-systolic segment length was significantly greater during PA constriction than during VC constriction \( (p<0.01) \) for a similar end-systolic pressure, whereas the end-diastolic relation fell on the same curve obtained during control and VC constriction. Thus, shifts of the end-systolic and end-diastolic pressure-length relations were not uniform among the circumferential segments in the LV minor axis.

Results at starting LV end-diastolic pressures of 5 and 15 mm Hg were similar to those at a starting end-diastolic pressure of 10 mm Hg. At a starting LV end-diastolic pressure of 5 mm Hg, LV end-systolic pressure decreased by 22.3±7.0% during VC constriction and by 26.4±9.7% during PA constriction, and stroke volume decreased by 47.1±10.5% during VC constriction and by 56.9±11.2% during PA constriction. At a starting...
LV end-diastolic pressure of 15 mm Hg, LV end-systolic pressure decreased by 21.2±8.9% during VC constriction and by 25.2±8.2% during PA constriction, and stroke volume decreased by 40.9±19.1% during VC constriction and by 34.6±11.4% during PA constriction. All these decreases were not significantly different between VC and PA constriction (all $p>0.1$). Table 3 summarizes changes in systolic shortening in the four segments during VC and PA constriction at starting pressures of 5 and 15 mm Hg. At both starting pressures, VC constriction caused homogeneous decreases in systolic shortening in the four segments. In contrast, PA constriction caused greater decreases in systolic shortening in the anterior and posterior segments than the septal and lateral segments, as was also seen at a starting pressure of 10 mm Hg. Thus, changes in systolic segment shortening were more pronounced during PA constriction than during VC constriction.

![Graph showing normalized end-systolic and end-diastolic pressure-segment length relations in anterior, lateral, and posterior segments](image)

**Table 3.** Percent Decrease in Systolic Shortening at Starting Left Ventricular End-Diastolic Pressures of 5 and 15 mm Hg With the Pericardium Intact

<table>
<thead>
<tr>
<th></th>
<th>Septal segment</th>
<th>Anterior segment</th>
<th>Lateral segment</th>
<th>Posterior segment</th>
</tr>
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<tbody>
<tr>
<td>LV EDP, 5 mm Hg</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>VCC</td>
<td>$-26.1±12.8$</td>
<td>$-27.7±15.7$</td>
<td>$-16.5±10.1$</td>
<td>$-24.1±22.7$</td>
</tr>
<tr>
<td>PAC</td>
<td>$-15.5±6.1$</td>
<td>$-39.5±15.0^*$</td>
<td>$-12.9±11.9$</td>
<td>$-35.9±22.7^*$</td>
</tr>
<tr>
<td>LV EDP, 15 mm Hg</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>VCC</td>
<td>$-18.4±19.4$</td>
<td>$-21.6±12.1$</td>
<td>$-16.6±22.2$</td>
<td>$-20.7±15.0$</td>
</tr>
<tr>
<td>PAC</td>
<td>$-0.2±6.9$</td>
<td>$-28.1±14.3^*$</td>
<td>$-4.1±16.0$</td>
<td>$-25.0±18.9^*$</td>
</tr>
</tbody>
</table>

LV EDP, left ventricular end-diastolic pressure; VCC, vena caval constriction; PAC, pulmonary artery constriction. All values are mean±SD.

* $p<0.01$ compared with the lateral segment.
* $p<0.01$ compared with the septal segment.
* $p<0.05$ compared with the septal segment.
Comparisons of decreases in systolic shortening during PA constriction were similar regardless of baseline LV end-diastolic pressure, indicating that LV chamber volume has no effect on the nonuniform decrease in circumferential regional shortening during PA constriction.

Table 4 summarizes hemodynamic data before and during VC and PA constriction at a starting LV end-diastolic pressure of 10 mm Hg after pericardiectomy. Hemodynamic changes during VC and PA constriction were qualitatively similar to those observed before pericardiectomy. In addition, percent decreases in LV end-systolic pressure (-16.2±7.8%) and stroke volume (-30.3±10.2%) during VC constriction were not significantly different from those during PA constriction (end-systolic pressure -16.4±10.2%, stroke volume -24.1±17.7%; p>0.2 for both).

Figure 6 shows percent decreases in systolic shortening during VC and PA constriction after pericardiectomy. During VC constriction, percent decreases in systolic shortening were homogeneous around the LV minor axis, as was the case before pericardiectomy. However, during PA constriction the nonuniformity that was observed before pericardiectomy was no longer present. The most notable difference after pericardiectomy was observed in the anterior segment, which had behavior essentially identical to the septal and lateral segments. Although the decrease in posterior segment shortening was greater than those in the other segments, the p value was larger than 0.1 by analysis of variance. This result indicates that the nonuniform response of circumferential segment shortening during PA constriction, especially in the anterior segment, is in part related to the presence of the pericardium.

In an additional four dogs, propranolol was given intravenously to examine whether β-blockade alters the nonuniform response of circumferential segment shortening during PA constriction. Starting at similar end-diastolic pressures (before blockade 9.4±2.4 mm Hg, after blockade 10.1±3.3 mm Hg), decreases in LV end-systolic pressure (before -22.4±3.2%, after -21.5±4.7%) and decreases in stroke volume (before -36.4±5.8%, after -42.2±19.2%) were similar before and after β-blockade (all p>0.2). Before β-blockade, decreases in systolic shortening in the anterior (-23.8±13.4%) and posterior segments (-28.8±12.2%) during PA constriction tended to be greater than those in septal (-7.8±13.8%) and lateral segments (-7.5±9.3%), although the overall difference among the segments did not reach statistical significance (p=0.1 by analysis of variance). After β-blockade, however, decreases in systolic segment shortening during PA constriction were significantly different among the four segments (septal -10.7±15.5%, anterior -51.9±18.5%, lateral -19.2±15.8%, posterior -34.6±26.8%, p<0.05), indicating that β-blockade did not alter the nonuniformity.

In two dogs with septal-lateral diameter measurements, relations between LV septal-lateral diameter and free-wall segment lengths were analyzed. Figure 7 shows correlations of septal-lateral diameter versus lateral segment length (panel A), anterior segment length versus lateral segment length (panel B), posterior segment length versus lateral segment length (panel C), and anterior segment length versus posterior segment length (panel D) throughout one cardiac cycle during control, VC constriction, and PA constriction at a starting LV end-diastolic pressure of 15 mm Hg in one dog. LV end-systolic pressure and stroke volume during the two interventions were similar (end-systolic pressure, 90 versus 86 mm Hg; stroke volume, 21.0 versus 20.9

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**TABLE 4. Hemodynamic Data With the Pericardium Open**

<table>
<thead>
<tr>
<th></th>
<th>Control 1</th>
<th>VCC</th>
<th>Control 2</th>
<th>PAC</th>
</tr>
</thead>
<tbody>
<tr>
<td>HR (beats/min)</td>
<td>123±29</td>
<td>122±28</td>
<td>119±23</td>
<td>124±20</td>
</tr>
<tr>
<td>LVEDP (mm Hg)</td>
<td>130±36</td>
<td>110±34</td>
<td>116±34</td>
<td>99±39</td>
</tr>
<tr>
<td>RVSP (mm Hg)</td>
<td>9.8±3.3</td>
<td>4.1±1.6*</td>
<td>9.7±4.4</td>
<td>5.1±3.6*</td>
</tr>
<tr>
<td>RVEDP (mm Hg)</td>
<td>33.8±7.3</td>
<td>20.8±4.2*</td>
<td>32.7±5.2</td>
<td>62.3±10.4*</td>
</tr>
<tr>
<td>Stroke volume (ml)</td>
<td>27.1±11.2</td>
<td>18.9±8.3*</td>
<td>27.1±11.5</td>
<td>20.4±10.2*</td>
</tr>
</tbody>
</table>

VCC, venae caval constriction; PAC, pulmonary artery constriction; HR, heart rate; LVEDP, left ventricular end-systolic pressure; RVSP, right ventricular peak systolic pressure; RVEDP, right ventricular end-diastolic pressure.

All values are mean±SD.

* p<0.01, t p<0.05 compared with the corresponding control.

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**Figure 6. Comparisons of decreases in systolic shortening (SS) during venae caval (VC, left) and pulmonary artery (PA, right) constrictions after pericardiectomy. Mean±SEM is indicated. Sept, septal segment; ant, anterior segment; lat, lateral segment; post, posterior segment. NS, not significant.**
ml). During VC constriction, the slope of the long axis of the loop in each panel is similar to that during control, indicating that the two dimensions in the ordinate and abscissa shorten in phase and that the decreases in systolic shortening during VC constriction are similar between the two dimensions. In contrast, during PA constriction the slope of the long axis of the loop deviated downward compared with both control and VC constriction (panels A, B, and C). Although the amount of downward deviation of the septal-lateral diameter is apparently greater than those of the anterior and posterior segments, the directions of these changes are similar. These downward deviations indicate that at a given lateral segment length, septal-lateral diameter and anterior and posterior segment lengths are shorter during PA constriction than during VC constriction and the decreases in systolic shortening during PA constriction are greater in these three dimensions than in the lateral segment. In contrast, the anterior-posterior segment length loop during PA constriction (panel D) lies approximately on the same line as that obtained during both control and VC constriction. Similar findings were observed at a starting LV end-diastolic pressure of 10 mm Hg, and also in the other dog with septal-lateral diameter measurements. These findings indicate that the anterior and posterior segment lengths, but not the lateral segment length, behave qualitatively similarly to the LV septal-lateral diameter during PA constriction with the pericardium intact.

Discussion

The major findings of the present study are 1) acute right ventricular pressure overload causes nonuniform regional changes in systolic segment shortening in the left ventricular minor axis (i.e., anterior and posterior segment shortening decreases more than septal and lateral segment shortening); 2) the greater decreases in anterior and posterior segment shortening are attributable to two different mechanisms, that is, a leftward shift of the end-diastolic pressure-length relation in the anterior segment and a rightward shift of the end-systolic pressure-length relation in the posterior segment; 3) the pericardium enhances this nonuniformity; and 4) LV volume and β-adrenergic activity are not responsible for the nonuniformity. Thus, changes in loading conditions imposed on individual regions during global alterations in LV geometry and function result in nonuniform systolic segment shortening around the LV minor axis.

Nonhomogeneous LV Regional Function During Acute Right Ventricular Pressure Overload

Although there have been many studies of regional variability of segment shortening or wall thickening of the normal LV, little is known about regional myocardial function during acute right ventricular
Mechanism of Nonuniformity

In the present study, anterior and posterior segment shortening decreased more than septal and lateral segment shortening during PA constriction. Because regional variation in catecholamines levels within the LV has been reported, one possible explanation for this nonuniform response is a regional difference in sympathetic activity. However, duration of PA constriction was short, and the heart rate during constriction did not differ from control, suggesting that a reflex increase in regional ventricular contractile state did not occur. Further, β-blockade with intravenous propranolol did not suppress this nonuniformity. Thus, the possibility that the nonuniform response is due to regional difference in sympathetic activity is remote.

Another explanation is that inhomogeneous changes in regional wall stress accompanying the global change in LV geometry (leftward septal shift) cause the nonuniformity. This explanation seems reasonable if it is assumed that pericardectomy reduced nonuniformity by attenuating global changes in LV geometry resulting from direct ventricular interaction during PA constriction, thus decreasing the magnitude of changes in regional wall stress. On the other hand, if we assume a prolate ellipsoidal LV geometry, regional wall stress in the anterior and posterior walls should, on the average, become smaller than that in the lateral wall during PA constriction because the regional radius of curvature in the anterior and posterior walls should decrease due to the leftward septal shift. This decrease in regional wall stress should, in turn, result in an increase in systolic shortening of the anterior and posterior segments. However, the results we observed were opposite in direction to those predicted by this explanation. Further, this simple geometric assumption does not account for the difference in regional wall stress between the anterior and posterior walls and, hence, does not explain the difference in segment length changes between these two regions during PA constriction.

In the present study, anterior end-diastolic length decreased more than posterior and lateral end-diastolic lengths, while posterior end-systolic length decreased less than anterior and lateral end-systolic lengths. These changes are attributable to two distinct mechanisms: a leftward shift of the end-diastolic pressure-length relation of the anterior segment and a rightward shift of the end-systolic pressure-length relation of the posterior segment. This suggests that there could be some local factors other than a simple change in global LV geometry that explain our results.

One such local factor is fiber continuity of the LV anterior and posterior myocardium with the right ventricular free wall. According to Thomas, the LV posterior midwall myocardium at the midventricular level, which is oriented in the circumferential direction, has direct continuity with the right ventricular free wall. In contrast, the LV anterior midwall myocardium, which is also circumferentially oriented, is mainly connected to the interventricular septum. This architectural relation between the two ventricles may explain the finding that end-systolic posterior segment length during PA constriction did not significantly decrease despite a decrease in LV end-diastolic pressure, because the expanded right ventricular free wall during PA constriction might stretch the directly connected LV posterior wall fibers. On the other hand, the finding that end-diastolic anterior segment length during PA constriction decreased more than the lateral and posterior segments might be related to the direct connection of the anterior midwall fibers with the interventricular septum.

A second local factor is fiber orientation. Although the fiber orientation in the LV midwall is mainly circumferential, fibers in the inner and outer layers that are more longitudinally oriented could affect midwall shortening of the anterior and posterior walls differently via tethering. In this regard, the finding of Lew and LeWinter that longitudinal shortening is different between the anterior and posterior walls could explain the different behavior of these two segments during PA occlusion. How-
ever, further studies will be needed to determine the roles of these factors during PA constriction.

Regional differences in diastolic distensibility could also influence regional systolic shortening. Lew and LeWinter compared the end-diastolic pressure-segment relation of the anterior segment and rightward shift of the end-systolic pressure-length relation of the posterior segment. LV volume and β-adrenergic activity do not appear to be responsible for this nonuniformity, whereas it is enhanced by the pericardium. These results also indicate that we cannot infer global LV contractile function from the shortening of any one segment during acute right ventricular pressure overload, and regional shortening during acute right ventricular pressure overload may not reflect the intrinsic regional contractile state.

Appendix

The error in measuring septal segment length due to a change in radius of curvature of the interventricular septum during PA constriction was estimated as follows. In Figure 8 left, in which part of the latitudinal plane of the LV during control is shown, arc AB (length l mm) represents part of the interventricular septum with a latitudinal septal radius of curvature of r, mm (lines AO and BO). Straight line AB represents septal segment length measured with sonomicrometers (length 30 mm). This length is approximately the same as end-diastolic segment length during control at LV end-diastolic pressure of 10 mm Hg in the present study. When we assume the radius of curvature of the septum during control (r,) as 30 mm based on the studies of Olsen et al and Slutsky et al, angle θ, is 60° because triangle ABO is equilateral. Then, we obtain length of arc AB as

\[ l = 2\pi r_1 \times \sin \frac{\theta_1}{2} \]

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\[ l = 2\pi r_1 \times \sin \frac{\theta_1}{2} \]

Thus, during control, the relatively long septal segment length AB underestimated the true length of the septal arc by 4.5%.

Latitudinal septal radius of curvature increases by approximately 15% to 27% during PA constriction. Therefore, we assume that the radius of curvature increases, at most, by 30% during PA constriction, which yields a septal radius of curvature during PA constriction (r,) of 30\times1.3=39 (mm) (Figure 8, right).

Because length l is constant regardless of septal radius of curvature, angle θ, is calculated as

\[ \theta_2 = \sin^{-1}\left(\frac{t_2}{r_2}\right) = 46.1° \]

Then,

\[ \theta_3 = (180 - \theta_2)/2 = 66.9° \]

Because \( \frac{a}{\sin \theta_3} = t_2 \sin \theta_3 \), length a is calculated as

\[ a = t_2 \sin \theta_3 = 30.5 \text{ mm} \]

Thus, when latitudinal septal radius of curvature increases by 30% during PA constriction, septal segment length underestimated the true arc length by 2.9%. However, when compared with the control measurement of 30 mm, segment length during PA constriction is overestimated by 0.5 mm, or 1.7%.
FIGURE 8. Models used for estimation of error in measuring septal segment length due to a change in septal radius of curvature during pulmonary artery (PA) constriction. Part of LV latitudinal plane during control (left) and PA constriction (right) are schematically shown. See "Appendix" for more explanation.

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References


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