Septal geometry in the unloaded living human heart

JOAO A. C. LIMA, M.D., PABLO A. GUZMAN, M.D., FRANK C. P. YIN, M.D., PH.D., ROBERT K. BRAWLEY, M.D., LINDA HUMPHREY, M.D., THOMAS A. TRAILL, M.R.C.P., SANDRA D. LIMA, PAOLO MARINO, M.D., MYRON L. WEISFELDT, M.D., AND JAMES L. WEISS, M.D.

ABSTRACT  Right ventricular loading leads to diastolic septal flattening in man without necessarily requiring right ventricular pressure to exceed left ventricular pressure. This observation suggested that the unstressed septal configuration is flat and that its normal concave shape is due to the left-to-right transseptal pressure gradient. To examine this hypothesis, we studied septal configuration by two-dimensional echocardiography in nine patients with normal global and regional left ventricular function during surgery for coronary artery disease. The transseptal pressure gradient was obtained from pulmonary capillary wedge pressure minus right atrial pressure. Measurements were obtained at control (open chest, intact pericardium [C]), with the pericardium open (OP), on cardiopulmonary bypass (CPB), and after cardiac arrest (CA). There were no changes in any measurements between C and OP or between CPB and CA. Left ventricular end-diastolic cavity area decreased from 16.5 ± 2.1 cm² at C to 11.1 ± 4.5 cm² after CPB, and further decreased to 8.9 ± 3.5 cm² after CA (p < .001), yet the septum flattened, as shown by an increase in its radius of curvature from 1.7 ± 0.5 cm during C to 2.5 ± 0.7 cm after CPB, and to 2.9 ± 1.0 cm after CA (p < .001), or from 0.4 ± 0.1 to 0.8 ± 0.4 to 1.1 ± 0.5 U (p < .001) when normalized for cavity area. Diastolic transseptal pressure gradient was reduced from 4.1 ± 2.3 mm Hg during C to 1.1 ± 1.8 mm Hg after CPB, and to 0.5 ± 1.4 mm Hg after CA (p < .01). Thus, in the unloaded human heart, the interventricular septum is more flat and occupies a neutral position between the left and right ventricle. Septal flattening due to a reduced but not reversed transseptal left-to-right gradient results from assumption of this unstressed shape as the right ventricular end-diastolic pressure approaches the left ventricular end-diastolic pressure. Circulation 74, No. 3, 463-468, 1986.

OWING TO its anatomic position, the interventricular septum is postulated to be a mediator of ventricular interaction.1-3 However, the mechanisms underlying the changes in septal position are still unclear. Previous work from our laboratory demonstrated leftward displacement of the interventricular septum with acute right ventricular loading during a Mueller maneuver in humans (a forced inspiration against a closed airway).1 In an effort to determine whether these shifts occurred as a result of right ventricular diastolic pressure exceeding left ventricular diastolic pressures, we then measured the diastolic transseptal pressure gradient during the Mueller maneuver, and correlated these changes with alterations in septal geometry. This study (again in humans) showed that the septal flattening that occurs with acute right ventricular loading is accompanied by a decrease in the diastolic left-to-right transseptal pressure gradient, without requiring a reversal of this gradient.2 This raised the possibility that the reason why the interventricular septum shifts leftward during acute right ventricular loading is that the normal septal configuration in the unloaded heart is flat, and that in the working heart it is held in its usual position concave to the left ventricular cavity by the normal and persistent left-to-right positive transseptal pressure gradient. The goal of the current study was to determine the position and configuration of the interventricular septum in the living human heart when the heart is arrested and the transseptal pressure approaches zero.

Methods

Study protocol. We studied nine patients with phased-array two-dimensional echocardiography who were undergoing open heart surgery for coronary artery disease (table 1). All patients had normal global and segmental left ventricular func-
TABLE 1
Patient characteristics

<table>
<thead>
<tr>
<th>Patient No.</th>
<th>Age/sex</th>
<th>Angiographic EF (%)</th>
<th>Coronary angiographic findings</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>52/M</td>
<td>57</td>
<td>99% proximal LAD occlusion</td>
</tr>
<tr>
<td>2</td>
<td>58/M</td>
<td>72</td>
<td>90% LAD, right and left circumflex marginal branch occlusion</td>
</tr>
<tr>
<td>3</td>
<td>59/M</td>
<td>54</td>
<td>50% to 70% proximal LAD and 70% left circumflex marginal branch occlusion</td>
</tr>
<tr>
<td>4</td>
<td>56/M</td>
<td>53</td>
<td>70% to 90% proximal LAD and 70% to 90% mid right coronary artery occlusion</td>
</tr>
<tr>
<td>5</td>
<td>52/F</td>
<td>59</td>
<td>Total proximal LAD occlusion</td>
</tr>
<tr>
<td>6</td>
<td>42/M</td>
<td>65</td>
<td>99% proximal LAD occlusion</td>
</tr>
<tr>
<td>7</td>
<td>54/M</td>
<td>50</td>
<td>70% to 90% proximal LAD occlusion</td>
</tr>
<tr>
<td>8</td>
<td>56/M</td>
<td>62</td>
<td>99% proximal LAD and distal right occlusions</td>
</tr>
<tr>
<td>9</td>
<td>45/M</td>
<td>57</td>
<td>50% to 70% mid LAD and 50% to 70% mid right coronary artery occlusion</td>
</tr>
</tbody>
</table>

**EF** = ejection fraction; **LAD** = left anterior descending coronary artery.

The size of the left ventricle (figure 1), indexed by the end-diastolic cross-sectional cavity area, decreased from 16.5 ± 2.1 cm² during control and 15.6 ± 3.2 cm² after the pericardial sac was opened to 11.1 ± 4.5 cm² during cardiopulmonary bypass, and further to 8.9 ± 3.5 cm² after cardioplegia and arrest (ANOVA, p < .001, and SNK test, p < .05, from opened pericardium to bypass and from bypass to arrest). The end-systolic cavity areas were also reduced from 9.0 ± 3.2 and 9.3 ± 3 cm² with intact and opened pericardium, respectively, to 6.5 ± 2.1 cm² during cardiopulmonary bypass (ANOVA, p < .005; figure 1). As the heart approached and achieved total arrest, the transseptal pressure gradient (figure 2) decreased from 4.1 ± 2.3 and 3.9 ± 2.3 mm Hg with intact and opened pericardium, respectively, to 1.1 ± 1.8 mm Hg during cardiopulmonary bypass, and further to 0.5 ± 1.4 mm Hg during arrest (overall ANOVA, p < .01). Both cardiac size and transseptal pressure gradient remained unchanged after the pericardial sac was opened but before cardiopulmonary bypass (SNK, p = NS). Similarly, the transseptal pressure gradient did not
FIGURE 1. End-diastolic (open bars) and end-systolic (hatched bars) left ventricular (LV) cross-sectional cavity area during cardiopulmonary bypass (CPB) and cardiac arrest (CA). Both decreased during CPB, and end-diastolic area decreased further during CA (SNK, p < .05 from OP to CPB and from CPB to CA; overall ANOVA, p < .001 for both). Between control (C) and open pericardium (OP), there were no changes in cavity area (SNK test, p = NS).

change from cardiopulmonary bypass to cardiac arrest (SNK, p = NS). Results for right atrial and pulmonary capillary wedge pressure were directionally and statistically similar to those for transseptal pressure.

The cross-sectional shape of the left ventricle changed markedly after cardiopulmonary bypass and cardiopulmonary bypass and cardioplegic arrest (figure 3). The interventricular septum flattened, as indexed by an increase in its radius of curvature, from 1.7 ± 0.5 and 1.6 ± 0.6 cm with intact and opened pericardium, respectively, to 2.5 ± 0.7 cm during bypass and 2.9 ± 1.0 cm after cardiac arrest (overall ANOVA, p < .001; SNK test, p = NS from bypass to complete arrest; figure 4). The changes in the septal radius of curvature, normalized to changes in end-diastolic cross-sectional area, were similar (from 0.4 ± 0.1 and 0.4 ± 0.2 U with intact and opened pericardium to 0.8 ± 0.4 U during bypass and 1.1 ± 0.5 U during arrest; overall ANOVA, p < .001; SNK test, p = NS from bypass to arrest; figure 4). By contrast, the free wall radius of curvature (diametrically opposite the interventricular septum) measured at end-diastole (figure 5) was unchanged throughout the entire experiment (from 1.4 ± 0.4 cm with intact pericardium to 1.1 ± 0.4 cm during arrest; ANOVA, p = NS). The free wall radius of curvature adjusted for changes in end-diastolic area also remained unchanged (from 0.3 ± 0.1 to 0.4 ± 0.2 U during arrest; ANOVA, p = NS; figure 5).

The changes in the normalized septal radius of curvature measured at end-systole (figure 6) during cardiopulmonary bypass were in the same direction as those measured at end-diastole (from 0.4 ± 0.2 and 0.4 ± 0.1 U during intact and opened pericardium, respectively, to 0.5 ± 0.1 U during bypass; ANOVA, p < .05). However, the absolute septal radius of curvature did not change significantly (from 1.2 ± 0.5 cm with intact pericardium to 1.3 ± 0.2 cm during cardiopulmonary bypass; ANOVA, p = NS). Conversely, the absolute end-systolic free wall radius of curvature (figure 7) decreased from 1.0 ± 0.2 cm with intact pericardium and 0.9 ± 0.2 cm with opened pericardium to 0.7 ± 0.3 cm during bypass (ANOVA, p < .05), while the normalized free wall radius of curvature remained unchanged (from 0.3 ± 0.1 to 0.3 ± 0.2 U, p = NS). There were no cross-sectional shape changes in the left ventricle after the pericardial sac was opened and reflected before cardiopulmonary bypass (figures 3 to 7).

Discussion

This study verifies the hypothesis that septal configuration is relatively flat in the unloaded human heart. That is, as the heart approaches a totally unloaded state and the positive left-to-right transseptal gradient approaches zero, the septum flattens from its normal configuration concave to the left ventricle.

The effects of the diastolic transseptal pressure gradient on interventricular septal position have been the subject of recent interest and investigation. Data from our institution,1,2 and other laboratories3-5 have shown that the position and shape of the septum at end-diastole is determined by the transseptal pressure gradient and that a reduction or reversal of such a gradient shifts the septum toward the left ventricle. While it is not difficult to understand leftward septal shifts in the presence of a reversed (negative with respect to the left ventricle) transseptal gradient, it is not as easy to conceptualize why there is a leftward shift in the presence of a reduction in the left-to-right gradient only. Our data suggest one possible mechanism for such a phenomenon: in the living but nonbeating human heart,
the interventricular septum is relatively flat and occupies a neutral position between the two ventricles. Therefore, the more familiar round configuration of the septum, with its concavity toward the left ventricle, is a result of the positive diastolic left-to-right transseptal pressure gradient. As this pressure gradient is reduced and approaches zero, the septum shifts toward its neutral position.

There is an alternative mechanism that could explain why the septum flattens in the presence of a reduced but not reversed transseptal gradient. If the septal surface exposed to right ventricular pressure exceeded that exposed to left ventricular pressure, the net force on the septum could be directed toward the left ventricle despite the pressure in the left ventricle being slightly higher than that in the right ventricle. This possibility, however, can only be confirmed with simultaneous measurements of right and left septal areas in situ — a very difficult undertaking.

In addition to the end-diastolic septal flattening as the heart was unloaded, we found a small increase in end-systolic radius of curvature. These changes were

FIGURE 3. Top, End-diastolic cross-sectional echocardiograms from one patient during control (open chest and intact pericardium), open pericardium, and cardiopulmonary bypass, and after cardiac arrest. Bottom, Same frames with contours outlining endocardium and epicardium for clarification. Septal flattening with cavity area reduction is evident during bypass and cardiac arrest.

FIGURE 4. Septal radius of curvature (open bars) at end-diastole increased during cardiopulmonary bypass (CPB) and cardiac arrest (CA). When normalized for end-diastolic cavity area (dividing by the square root of the area, hatched bars), this augmentation persisted (overall ANOVA, p < .001 for both). There was no change from control (C) to open pericardium (OP) or from CPB to CA for either radius of curvature or normalized radius of curvature (SNK test, p = NS).

FIGURE 5. End-diastolic left ventricular (LV) free wall radius of curvature (open bars). This remained unchanged through all stages (overall ANOVA, p = NS). Normalization for end-diastolic cavity area (hatched bars) did not alter these results. Other abbreviations are as in previous figures.
far less striking than the end-diastolic flattening seen during bypass or cardiac arrest, but are consistent with findings of previous studies that indicated persistent septal flattening throughout several cardiac cycles.\(^1\)\(^2\)

Why the septum maintains its flat configuration during systole when the left ventricular cavity pressure greatly exceeds right ventricular pressure is not known. As ventricular contraction proceeds, the stiffness of the muscle increases above the level present during diastole, when it is more flaccid.\(^9\)\(^10\) Thus, one possible explanation for the persistent systolic flattening is that the increase in stiffness in the initially flattened septum is enough to counterbalance the increase in left-to-right forces tending to push the septum toward the right ventricle. If this hypothesis is correct, then both the timing and relative extent of diastolic septal flattening are critical determinants of the systolic septal configuration. If too little diastolic flattening occurs, or if it occurs too late, the increase in septal stiffness would not be sufficient to prevent the net systolic forces from pushing the septum toward the right ventricle and persistent systolic flattening or displacement would not be observed. This could be the reason that no end-systolic shape change was observed in the dog by Kingma et al.\(^3\) during right ventricular volume or pressure overload. That group, however, did note a decrease in septal-to-free wall diameter during those interventions as well as a simultaneous decrease in left-to-right transseptal pressure gradient. Stool et al.\(^3\) also noted a decrease in the septal-to-lateral wall dimension during pulmonary hypertension in the dog. Clearly, further work is needed to clarify the mechanism of the behavior of the interventricular septum during systole.

Our study was designed to test the hypothesis that septal configuration is flat in the unloaded human liv-

![FIGURE 6. End-systolic septal radius of curvature (open bars) did not change during cardiopulmonary bypass (CPB) (overall ANOVA, p = NS). The normalized (but not the nonnormalized) septal radius of curvature (hatched bars) showed a slight increase during CPB (overall ANOVA, p < .005) that was of a lesser magnitude than the changes seen during diastole. No changes were detected between intact and opened pericardium for the normalized radius of curvature (SNK test, p = NS). Other abbreviations are as in previous figures.](image)

![FIGURE 7. End-systolic left ventricular (LV) free wall radius of curvature (open bars) decreased during cardiopulmonary bypass (overall ANOVA, p < .05), as the LV cavity became smaller. However, when normalized for end-systolic cavity area (hatched bars), the free wall radius of curvature remained unchanged throughout (overall ANOVA, p = NS). No changes were detected in the end-systolic LV free wall radius of curvature from intact to opened pericardium (SNK test, p = NS). Other abbreviations are as in previous figures.](image)
volume of the intracardiac chambers diminished. On the other hand, great care was taken to minimize these effects by use of the transducer of modified design, maintenance of internal landmarks in this image (papillary muscles), and by recording echocardiograms invariably as the transducer was being lifted off the heart, with the use for data analysis of only the echocardiographic frames immediately before the loss of the image (see Methods). We have previously validated this approach using the same transducer standoff device in the open-chest dog by documenting a lack of significant change in cross-sectional left ventricular shape as the standoff device was lifted from the heart.11

Our data showed no difference in left ventricular cross-sectional configuration or in any of the other measured parameters before and after the pericardial sac was opened and reflected during surgery. It has been reported that the pericardium mediates or potentiates ventricular interaction by restraining overall cardiac distensibility.12-15 Such evidence has been obtained by assessing the effects of selective loading or unloading of one or more cardiac chambers in the presence or absence of the pericardium. In our study, there were no changes in cross-sectional left ventricular cavity area after the pericardium was reflected before cardiopulmonary bypass. In addition, our patients had normal angiographic left ventricular cavity size and function before surgery, and none developed dysfunction during the surgical procedure. Thus, although we detected no changes in left ventricular shape attributable to the absence of the pericardium in our patients, right or left ventricular overload was not likely present before pericardiectomy, and distensibility may not have been limited by the pericardium for this reason.

In summary, we have documented that in patients undergoing cardiac surgery the interventricular septum flattens as the heart approaches and then achieves a totally unloaded state, and that under the conditions of this study in the living human heart, the position of the interventricular septum is not affected by the presence of the pericardium. Our results are further evidence in man that the diastolic position of the interventricular septum is a reflection of the left-to-right transseptal pressure gradient, and that it is through changes in this gradient that the interventricular septum, by altering its shape and/or position, may mediate diastolic ventricular interaction.

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Septal geometry in the unloaded living human heart.
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