Nonuniform Regional Deformation of the Pericardium During the Cardiac Cycle in Dogs

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We hypothesized that local contact forces between the pericardium and the heart cause regional variation in pericardial deformation during the cardiac cycle, reflecting volume changes of the underlying cardiac chambers. To test this, we measured regional pericardial area over the right atrium (RA) and right ventricle (RV) with orthogonal pairs of sonomicrometers in six open-chest dogs. At a left ventricular end-diastolic pressure of 5 mm Hg, RV pericardial area paralleled RV volume, that is, shrinkage during ejection by 10±8% and expansion during filling. RA pericardial area was reciprocally related to RV pericardial area, with average expansion during ventricular ejection of 2±2%, thus paralleling RA volume during RV ejection. With volume loading, RV pericardial shrinkage during ejection increased to 14±6%, but the RA pericardial area change was no longer reciprocal (0±3% change during RV ejection). Elimination of contact forces by cardiac tamponade resulted in both marked attenuation of RV pericardial area changes and synchronization of the RV and RA pericardial area pattern; that is, both shrank during RV ejection. In two additional dogs, measurement of pericardial area over left ventricle and atrium showed similar results. We conclude that dynamic pericardial contact forces cause regional variation in pericardial deformation, which reflects volume changes of the underlying chambers. These findings imply that the influence of the pericardium on filling and ejection may be more complex than previously recognized, varying both by chamber and dynamically over the course of the cardiac cycle. (Circulation Research 1990;67:1107–1114)

The pericardium prevents excessive cardiac dilatation and accentuates diastolic ventricular coupling.1,2 This restraining function of the pericardium has been assessed by measuring pericardial surface pressure at a single site adjacent to a cardiac chamber.3–5 However, recent studies have shown that pericardial surface pressure measured with a flat balloon varies over different parts of the heart.6,7 This finding suggests that the contact force between the pericardium and the surface of the heart varies by region. However, the presence of a balloon in the pericardial space might itself alter these local forces. Therefore, if indeed there is regional variation in the force between the pericardium and the surface of the heart, a better approach to investigating this would use a technique that does not in and of itself alter local forces.

Accordingly, the purpose of this study was to test the hypothesis that pericardial contact forces vary by region by demonstrating nonuniform regional variation in pericardial deformation during the cardiac cycle. If the material properties of the pericardium are uniform over different regions of the heart, as suggested by Lee et al,8 demonstration of nonuniform regional deformation of the pericardium would directly indicate regional variation of the contact forces. Further, if elimination of the contact between the pericardium and the heart abolishes the nonuniform regional pericardial deformation, this would also be proof of the presence of regional contact forces. To this end, rather than using balloons, we measured regional pericardial area over the right ventricle (RV) and right atrium (RA) with orthogonal pairs of sonomicrometers in open-chest dogs under various conditions.

Materials and Methods

Surgical Preparation

Eight random source dogs (17–26 kg) premedicated with 1 mg/kg subcutaneous morphine sulfate were anesthetized with sodium pentobarbital (25 mg/kg i.v.), intubated, and ventilated with a constant-volume respirator (Harvard Apparatus, South Natick, Mass.). A median sternotomy was performed.
in each dog in the supine position. A limb lead electrocardiogram was monitored throughout the experiment. Micromanometer catheters (7F, Millar Instruments, Houston) with fluid-filled lumens were introduced into the left ventricle (LV) from the right femoral artery and into the RV from the right jugular vein. Another micromanometer catheter was introduced into the RA from the right femoral vein in six of the eight dogs and into the left atrium (LA) from the right upper pulmonary vein in two other dogs. All fluid-filled lumens were connected to pressure transducers (model P23XL, Gould Instruments, Cleveland) with zero reference at the level of the RA. Pressures from the fluid-filled and micromanometer catheters were matched at the beginning of the experiment and subsequently checked to correct for drift. A vinyl chloride catheter (2.3 mm in diameter) was inserted into the pericardial space through a small slit (5 mm in length) at the cardiac base and secured by a purse-string suture. A polyvinyl tube for intravenous fluid administration was introduced into the left femoral vein.

In the six dogs with RA micromanometers, right-sided pericardial areas were measured. After pericardial fat was gently dissected away, four pairs of specially designed ultrasonic crystals (5 MHz, 2 mm in diameter) were placed on the pericardium over the anterolateral surfaces of the RV and RA to measure circumferential and longitudinal segment lengths of the pericardium (Figure 1). Each ultrasonic crystal was mounted with its face oriented perpendicular to the electrical cable exiting from the rear of the crystal. The cable was then threaded through a 1-cm length of plastic tubing, and the latter was glued to the rear of the crystal such that the crystal and plastic tubing constituted a single fixed unit.9 Each crystal-tubing unit was fixed to the surface of the pericardium with a single suture so that each segment would be 1–2 cm in length. Ultrasonic signals were obtained by covering the crystals with a small amount of ultrasonic gel.

In the remaining two dogs with LA micromanometers, left-sided pericardial areas were measured; that is, the ultrasonic crystals were placed on the pericardial surface over the LV and LA in a similar manner to the right-sided area measurement. However, in one dog, we were not able to place all four ultrasonic crystals over the LA as a result of limited access, since most of the LA was hidden behind the LV and the great arteries. Therefore, pericardial area over the LA was measured in only one dog.

At the completion of instrumentation, superior and diaphragmatic pericardial ligaments and remaining adipose tissue were left intact, and the chest was left wide open.

**Experimental Protocol**

Measurements of the electrocardiogram, pressures, and pericardial segment lengths were made under baseline conditions at an LV end-diastolic pressure of approximately 6 mm Hg. If the LV end-diastolic pressure was lower than 6 mm Hg, it was adjusted by infusing warmed 6% dextran solution in 0.9% saline. The respirator was temporarily turned off at end-expiration so that there was no contact between the lungs and the lateral surfaces of the heart during measurements. 10 After baseline measurements, dextran was infused with a pump (Masterflex, Cole-Parmer Instrument Co., Chicago) until LV end-diastolic pressure reached approximately 12 mm Hg (volume load 1) and then 18 mm Hg (volume load 2), and measurements were repeated during steady-state conditions. Blood was then withdrawn until LV end-diastolic pressure returned to approximately 6 mm Hg, and a second set of baseline measurements was made in a new steady state (i.e., pretamponade control). Then the effect of elimination of local contact between the pericardium and the heart on the pericardial deformation pattern was assessed by creating cardiac tamponade. To produce mild (tamponade 1) and moderate (tamponade 2) cardiac tamponade, volumes of warmed saline of approximately 90 and 160 ml, respectively, were infused into the pericardial space through the pericardial catheter. Finally, after a second blood withdrawal to reduce LV end-diastolic pressure to the baseline level, measurements were made before and immediately after cutting the pericardial diaphragmatic ligament to examine whether this ligament plays a significant role in generating the pericardial deformation pattern.

**Data Analysis**

Data were recorded on a pen recorder and also converted from analog to digital on a PDP 11/73 computer (DEC, Maynard, Mass.) at a sampling interval of 5 msec. End-diastole of each ventricle was defined as the time when positive dP/dt of the ventricle increased to 10% of its peak value.10,11 The beginning of ejection of each ventricle was defined as

**Figure 1. Schematic illustration of placement of ultrasonic crystals on the pericardial surface over the right ventricle (RV) and atrium (RA). LV, left ventricle.**
the time of peak dP/dt. End-systole of each ventricle was defined as the time 30 msec before peak negative dP/dt of that ventricle. The atrioventricular pressure crossing point was defined as the time RA or LA pressure first exceeded RV or LV pressure, respectively, after peak negative dP/dt.

The dimensional data during one cardiac cycle were analyzed based on the above seven timing landmarks: RV and LV end-diastole, RV and LV beginning of ejection, RV and LV end-systole, and atrioventricular pressure crossing. Under baseline conditions with an average RR interval of 561±126 (±SD) msec, the percentage of the RR interval between RV end-diastole and each landmark was as follows: LV end-diastole, 2.8±2.5%; LV beginning of ejection, 10.0±1.0%; RV beginning of ejection, 12.7±3.4%; RV end-systole, 48.4±6.6%; LV end-systole, 49.2±7.5%; and atrioventricular pressure crossing, 62.1±11.7%. Pericardial area was calculated as the product of the circumferential and longitudinal segment lengths divided by 2 to reflect the diamond-shaped area surrounded by the four crystals. To eliminate interindividual variation, pericardial areas were normalized to each baseline pericardial area value at RV end-diastole. All data were averaged over three beats during steady-state contractions.

Statistics

Hemodynamic and dimensional data among different conditions including baseline, volume load, cardiac tamponade, and before and after cutting the ligament were compared by repeated measures analysis of variance. When this indicated a significant difference among the conditions, the least significant difference method was used to determine the significance of difference between the conditions. Comparisons of pericardial area among different time points within the cardiac cycle in each region under each hemodynamic condition were made by the same method. Values of \( p < 0.05 \) were considered statistically significant. Data are presented as the mean±SD, unless otherwise indicated.

Results

Table 1 summarizes hemodynamic and end-diastolic pericardial area data before and during volume loading and cardiac tamponade. Heart rate was little changed during interventions, except for cardiac tamponade 2, during which it increased significantly. Other hemodynamic variables changed as expected during both volume loading and tamponade.

Figure 2 shows an example of recordings of LV and RV pressures and pericardial segments and areas in the baseline steady state. The circumferential and longitudinal pericardial segment lengths over the RV and RA did not show any consistent excursion patterns related to the orientation of the segment. However, RV and RA pericardial area recordings consistently showed a reciprocal relation. During ventricular ejection, RV pericardial area shrank, whereas RA pericardial area expanded.

Figure 3 shows representative recordings of LV and RV pressures and RV and RA pericardial areas during the baseline period, volume load 1, volume load 2, and cardiac tamponade 2. Under baseline conditions, ventricular and atrial pericardial areas again show a reciprocal relation during ventricular ejection. With moderate volume loading (volume load 1), both RV and RA end-diastolic pericardial areas increased, and ventricular pericardial area shrinkage during ventricular ejection increased, while the atrial pericardial area excursion decreased. With further volume loading (volume load 2), end-diastolic areas of both the RV and RA pericardium increased further, whereas the excursions of both areas decreased. During cardiac tamponade, both pericardial area excursions were markedly attenuated, despite the fact that the end-diastolic pericardial areas were similar to those present during volume load 2.

### Table 1. Hemodynamic and Dimensional Data

<table>
<thead>
<tr>
<th></th>
<th>Baseline</th>
<th>Volume load 1</th>
<th>Volume load 2</th>
<th>Pretamponade</th>
<th>Tamponade 1</th>
<th>Tamponade 2</th>
</tr>
</thead>
<tbody>
<tr>
<td>HR (beats/min)</td>
<td>111±23</td>
<td>101±30</td>
<td>103±32</td>
<td>107±31</td>
<td>113±28</td>
<td>128±21*</td>
</tr>
<tr>
<td>LVESP (mm Hg)</td>
<td>117±18</td>
<td>122±15</td>
<td>132±21</td>
<td>121±16</td>
<td>120±18</td>
<td>96±34†</td>
</tr>
<tr>
<td>LVEDP (mm Hg)</td>
<td>5.5±1.4</td>
<td>11.2±1.0‡</td>
<td>17.5±1.0‡</td>
<td>5.6±1.3</td>
<td>7.2±3.1</td>
<td>11.7±7.1*</td>
</tr>
<tr>
<td>RVESP (mm Hg)</td>
<td>13±4</td>
<td>23±169</td>
<td>29±12‡</td>
<td>20±5</td>
<td>18±8</td>
<td>27±9</td>
</tr>
<tr>
<td>RVEDP (mm Hg)</td>
<td>3.2±1.9</td>
<td>7.3±2.2‡</td>
<td>11.3±2.0‡</td>
<td>4.1±1.5</td>
<td>5.6±1.8</td>
<td>10.4±5.2*</td>
</tr>
<tr>
<td>Normalized RV pericardial area at RV end-diastole (% of baseline)</td>
<td>100</td>
<td>125±8‡</td>
<td>134±13‡</td>
<td>109±19‡</td>
<td>126±15*</td>
<td>133±14*</td>
</tr>
<tr>
<td>Normalized RA pericardial area at RV end-diastole (% of baseline)</td>
<td>100</td>
<td>118±12‡</td>
<td>123±15‡</td>
<td>102±6</td>
<td>117±10*</td>
<td>123±15*</td>
</tr>
</tbody>
</table>

Values are mean±SD. HR, heart rate; LVESP, left ventricular end-systolic pressure; LVEDP, left ventricular end-diastolic pressure; RVESP, right ventricular end-systolic pressure; RVEDP, right ventricular end-diastolic pressure; RA, right atrial.

* \( p < 0.01 \) vs. pretamponade value.
† \( p < 0.05 \) vs. pretamponade value.
‡ \( p < 0.01 \) vs. baseline value.
§ \( p < 0.05 \) vs. baseline value.
Figure 4 depicts average pericardial deformation data during one cardiac cycle under baseline conditions, volume loading, and cardiac tamponade. The upper and lower panels show RV and RA pericardial areas, respectively. Under baseline conditions, both RV \((p=0.08)\) and RA \((p<0.05)\) pericardial areas increased during the isovolumic contraction period (from RV end-diastole to the beginning of RV ejection). During RV ejection, the ventricular pericardium shrunk significantly. In contrast, the atrial pericardium expanded significantly during RV ejection. During isovolumic relaxation (from RV end-systole to atrioventricular pressure crossing), there was no significant pericardial area change. Between the atrioventricular pressure crossing and RV end-diastole, that is, during the period of ventricular filling, RV pericardial area increased significantly while RA pericardial area decreased significantly, although the area changes during this period were not as obvious as during ejection in the examples in Figures 2 and 3, because the dynamic patterns were more complex.

Group values under the other conditions were similar to those shown in Figure 3. Thus, with volume loading, both ventricular and atrial end-diastolic pericardial areas increased significantly from the baseline values (Table 1). Although RV pericardial shrinkage during RV ejection was maintained during volume loading, RA pericardial expansion during RV ejection virtually disappeared (Figure 4). In contrast, during cardiac tamponade, both ventricular and atrial area shrinkage during ejection was markedly attenuated. This is especially apparent when compared with volume loads 1 and 2, when end-diastolic pericardial areas were virtually identical to those present during tamponades 1 and 2 (Figure 4).

Table 2 summarizes area changes of the RV and RA pericardium during RV ejection under various conditions. Under baseline conditions, ventricular and atrial...
pericardial areas were once again reciprocally related during the ejection period. With volume loading, ventricular pericardial area shrinkage initially increased (during volume load 1, $p=0.06$) and then returned to the baseline value during volume load 2. Atrial pericardial area expansion virtually disappeared with volume loading. During cardiac tamponade, RV pericardial area shrinkage markedly decreased. Interestingly, the RA pericardial area also began to shrink slightly during ejection, resulting in a synchronization of ventricular and atrial pericardial deformation.

Figure 5 displays the dynamic changes in pericardial deformation as the relation between LV pressure and pericardial area during one cardiac cycle, that is, as LV pressure–pericardial area loops. These loops were constructed because they provide useful information about the timing of pericardial area changes during the cardiac cycle. Under baseline conditions and volume load 1, the LV pressure–pericardial area loop over the RV is very similar to a ventricular pressure–volume loop, suggesting that the RV pericardial area change corresponds to RV volume. In contrast, the RA loops rotate in the opposite direction, reflecting right atrial volume.

Figure 6 shows the effect of cutting the diaphragmatic ligament on the pericardial deformation pattern. Cutting the ligament did not change heart rate or LV and RV pressures. Although RV pericardial area at RV end-diastole increased significantly after cutting ($p<0.05$), the basic pattern of RV and RA pericardial area deformation remained unchanged.

Pericardial area measured over the LV and LA showed deformation patterns basically similar to those over the right heart, although the magnitude of LV pericardial area shrinkage during LV ejection was smaller than that of the RV pericardium. In these two dogs, LV pericardial area shrank by 2.5% and 4.0% during the LV ejection period under baseline conditions. During this period, LA pericardial area increased by 16.7% in one of these dogs. During volume load 2, LV pericardial area shrinkage during LV ejection was unchanged (4.4% and 2.8%), whereas LA pericardial area expansion decreased to 2.1%. During cardiac tamponade, both LV and LA pericardial area excursions markedly decreased, and synchronization was observed during ventricular ejection, changes that were similar to those observed over the RV and RA. Finally, cutting the pericardial diaphragmatic ligament did not cause major changes in the left-sided pericardial area deformation patterns.

**Discussion**

This study has clearly demonstrated regional variation in pericardial deformation during the cardiac cycle. The most important finding is that under baseline conditions, pericardial area over the ventricle parallels ventricular volume, whereas atrial pericardial area is reciprocally related to ventricular pericardial area. In addition, elimination of a direct contact between the pericardium and the heart by

**Table 2. Percent Area Shrinkage During Right Ventricular Ejection**

<table>
<thead>
<tr>
<th></th>
<th>Baseline</th>
<th>Volume load 1</th>
<th>Volume load 2</th>
<th>Pretamponade</th>
<th>Tamponade 1</th>
<th>Tamponade 2</th>
</tr>
</thead>
<tbody>
<tr>
<td>RV pericardium (%)</td>
<td>9.7±7.5</td>
<td>13.7±6.2*</td>
<td>9.5±3.9</td>
<td>10.0±3.1</td>
<td>8.9±3.1</td>
<td>2.4±1.3†</td>
</tr>
<tr>
<td>RA pericardium (%)</td>
<td>-2.4±2.1</td>
<td>-0.1±2.8†</td>
<td>0.6±2.6†</td>
<td>-3.0±3.5</td>
<td>0.8±1.3†</td>
<td>1.3±1.4†</td>
</tr>
</tbody>
</table>

Values are mean±SD. RV, right ventricular; RA, right atrial.

$p<0.05$ vs. baseline value.

$\dagger p<0.01$ vs. baseline value.

$\ddagger p<0.05$ vs. baseline value.

![Figure 4](http://circres.ahajournals.org/) Changes in normalized pericardial area over the right ventricle (RV, upper panel) and right atrium (RA, lower panel) during one cardiac cycle. RVED, right ventricular end-diastole; LVED, left ventricular end-diastole; LVBEj, left ventricular beginning of ejection; RVBEj, right ventricular beginning of ejection; RVES, right ventricular end-systole; LVES, left ventricular end-systole; AVPX, atrioventricular pressure crossing. Mean±SEM is indicated. *$p<0.05$; **$p<0.01$ compared with the value of the preceding point.
cardiac tamponade resulted in both marked attenuation of pericardial area changes and synchronization of the ventricular and atrial pericardial area patterns. These findings indicate that there is, in fact, a contact force between the pericardium and the heart since the present study was not performed with measuring devices (i.e., balloons), which themselves could influence such contact forces by virtue of being interposed between the two surfaces. Thus, dynamic pericardial contact forces cause regional variation in pericardial deformation, which reflects the volume changes of the underlying chambers.

Because the pericardium is a biological tissue with viscoelastic properties, its behavior should obey an exponential, passive stress-strain relation, as schematically illustrated in Figure 7. Therefore, to assess pericardial behavior in situ, pericardial stress or strain needs to be measured. One approach has been to measure pericardial surface pressure as an index of pericardial stress. Measurement of pericardial surface pressure in vivo has been made with either open-ended, fluid-filled catheters or flat balloons, although recent studies have shown that a flat balloon is more accurate than an open-ended catheter in the absence of a significant amount of pericardial fluid. However, pericardial surface pressure may be imperfect as a measure of pericardial stress because it reflects pericardial stress only in the radial direction. Furthermore, the presence of the balloon itself may artifactually distort local stress and resultant strains.

Another approach is to measure pericardial strain from pericardial dimensions. Pericardial dimensions have been assessed in both in vitro and in vivo studies. However, all of these studies assessed static or end-diastolic properties rather than the dynamic behavior of the pericardium. In contrast, the present study has assessed the dynamic behavior of the pericardium in vivo throughout the cardiac cycle. Furthermore, this study has also assessed regional varia-

**Figure 5.** Dynamic changes in pericardial deformation displayed as the relation between left ventricular pressure and pericardial area during one cardiac cycle, that is, left ventricular pressure—pericardial area loops, over the right ventricle (upper panel) and right atrium (lower panel). The loops over the right ventricle rotate counterclockwise, indicating that right ventricular pericardial area parallels ventricular volume. In contrast, the loops over the right atrium rotate clockwise, indicating that right atrial pericardial area corresponds with atrial volume.
tions in pericardial strain over the ventricle and the atrium.

Many of the previous studies that measured pericardial dimensions have shown that the canine pericardium is slightly anisotropic both in vitro\(^8,15\) and in vivo.\(^2\) This suggests that unidirectional measurement of a pericardial dimension may not accurately reflect pericardial strain. Our pericardial area measurements with two orthogonal segment lengths reflect multidirectional pericardial strain. Thus, the pericardial area measurement is a new, direct method to assess pericardial behavior in situ.

Our findings that pericardial deformation varies depending on both the site over the heart and the timing within the cardiac cycle are in accordance with recent reports of Smiseth et al\(^8\) and Hoit et al,\(^7\) which have shown regional variation in pericardial surface pressure measured with flat balloons over the ventricles. These findings, together with marked attenuation and synchronization of the RV and RA pericardial deformation during cardiac tamponade, constitute direct proof of the existence of local contact forces between the pericardium and the heart. However, this does not mean that these are the only types of forces acting between the surface of the heart and the parietal pericardium. Indeed, it is entirely possible that at other sites the predominant force might be hydrostatic in nature.

In the present study, pericardial area changes of both RV and RA appeared to parallel the volume changes of the underlying cardiac chambers under baseline conditions. However, the percent atrial pericardial area excursion was less than the ventricular area excursion (Table 2, Figures 4 and 5). In addition, the atrial pericardial area excursion disappeared during volume loading, while ventricular pericardial shrinkage during the ejection period was maintained. This difference between the ventricular and atrial pericardial areas may be related to differences in the filling patterns of the two chambers, because ventricular filling is cyclic, whereas atrial filling is continuous except during atrial contraction. Thus, when average atrial inflow was augmented by volume loading, the relative amount of cyclic excursion may have been diminished. Alternatively, if atrial chamber compliance were less than ventricular chamber compliance, this could also result in attenuated volume variation of the atrium at higher filling pressures. A third explanation is the possibility that there is regional variation in the material properties of the pericardium over the ventricle and the atrium. None of the in vitro studies of pericardial behavior have addressed this issue, although Lee et al\(^8\) demonstrated similar material properties of the pericardium over the left and right ventricles.

In relation to pericardial area excursion, several reasons for the decrease in RV pericardial area shrinkage after volume load 2 (Figure 3, Table 2) can be proposed. The first is that during marked volume loading, the increase in RV end-systolic volume exceeded the increase in RV end-diastolic volume, resulting in decreases in both RV stroke volume and pericardial area excursion. The second possibility is that at higher filling conditions, the pericardium becomes too stiff to demonstrate much dynamic motion because of the nonlinear stress-strain relation (Figure 7). The third possibility is that other factors become more important than regional contact forces under these conditions. Thus, the exact mechanism remains to be determined. However, because there is no reason to think that pericardial deformation is
determined exclusively by regional contact forces, it is not surprising that there may not be a perfect qualitative or quantitative relation between deformation and chamber volume.

We measured pericardial areas mainly over the right heart in the present study. Placement of ultrasonic crystals over the LA was technically difficult because in the normal dog, only a portion of the LA is covered by the pericardium, and most of it is hidden behind the LV and great arteries. Although the number of experiments was small, LV pericardial area excursion was smaller than that of the RV. This quantitative difference between the RV and LV might be explained by either differences in hydrostatic forces or simply regional variation in the contact force. A priori, there is no reason to expect that a contact force would be regionally uniform. Nonetheless, our results indicate that the deformation patterns of the local pericardium are qualitatively similar over the right and left sides of the heart.

The dynamic deformation of the local pericardium could have potential physiological significance in ventricular filling and ejection mechanics in terms of atrioventricular mechanical coupling, especially in the low-pressure chambers such as the RV and the atria. At the time of atrioventricular pressure crossing, that is, the beginning of ventricular filling, the local pericardium over the atrium is maximally stretched under normal conditions (Figure 4). This stretched local pericardium may facilitate atrial emptying or ventricular filling by its elastic force. The ventricular pericardium would impede ventricular filling least during early diastole, because it is minimally stretched at the time when ventricular filling begins, and might prevent overfilling of the ventricle near end-diastole by means of its elastic force. On the other hand, the ventricular pericardium is maximally stretched at the time of beginning of ventricular ejection. The potential energy stored in the elastic pericardium may facilitate, in turn, ventricular ejection, particularly that of the RV, in which the systolic developed force is much smaller than that in the LV. Thus, the dynamic behavior of pericardial deformation suggests that the influence of the pericardium on ventricular filling and ejection mechanics may be more complex than previously recognized, varying both by chamber and dynamically over the course of the cardiac cycle. Furthermore, the pericardium may affect atrioventricular coupling not only through a steady-state mechanism, but also through a dynamic mechanism. Further study will be needed to elucidate the dynamic nature of the role of the pericardium in ventricular and atrial filling and ejection mechanics.

Another point of physiological relevance of the present study is that if regional contact forces between the pericardium and the heart vary by region, the external pressure around the heart cannot be represented by a single number measured at a single site (with the pericardium intact). This is also the conclusion of the study by Hoit et al. Thus, the concept of the transmural filling pressure may be much more complicated than the usual thinking, based on the assumption of a global, uniform pericardial surface pressure, would suggest.

Acknowledgments

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References

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