Regional Left Ventricular Epicardial Deformation in the Passive Dog Heart

Andrew D. McCulloch, Bruce H. Smaill, and Peter J. Hunter

Epicardial wall motion was measured on the left ventricular free wall in six isolated potassium-arrested dog hearts using a biplane video technique. Significant regional variations in epicardial deformations were recorded during static ventricular filling. Epicardial stretches varied linearly with cavity volume, sometimes exceeding 20% at physiological left ventricular end-diastolic pressures. The maximum component of epicardial stretch and the derived wall thinning increased substantially from the base to the apex on both the anterior and posterior free walls of the left ventricle. In five hearts, the direction of greatest epicardial stretch at moderate and high filling pressures coincided closely with the local epicardial fiber direction, suggesting that the left-handed epicardial fiber helices stretch preferentially during passive filling to maximize end-diastolic fiber lengths. Epicardial rotation was always counterclockwise, consistent with a reduction in the pitch of the fiber helix during filling. These results suggest that, on the epicardial surface, the passive myocardium is anisotropic with respect to the local fiber direction. We suggest that the resulting torsional shear acts to minimize transmural gradients of fiber stretch. (Circulation Research 1989;64:721–733)
are suitable for validating the assumptions of continuum mechanics models of the passive left ventricle and verifying the accuracy of the epicardial strains that they predict.

**Materials and Methods**

**Preparation**

Eight mongrel dogs weighing 21–30 kg were pretreated with oral nifedipine (i.e., 10 mg every 12 hours), in the 48 hours before surgery, to enhance myocardial preservation during ischemia. Anesthesia was induced with 25 mg/kg i.v. thiopental sodium and maintained with 1.5% halothane, using positive-pressure ventilation. After thoracotomy, the aorta was occluded and the heart was arrested rapidly by injecting a 50-ml bolus of cold 10% potassium chloride solution into the left ventricle. The heart was excised and transferred to a bath of cool (10°C) cardioplegic solution containing nifedipine (0.2 mg/l), and the atria and pulmonary outflow tract were removed. Hypothermia was maintained while the heart was mounted on a special cannula assembly, which we have described previously. The left ventricular outflow tract was blocked by an aortic cannula through which the coronary circulation could be perfused, and another cannula sealed the mitral valve to enable the ventricle to be filled. The coronary circulation was then flushed with the cool cardioplegic solution.

Two regions of the left ventricular free wall were studied in each heart. At both sides, three epicardial markers were positioned 10–20 mm apart in approximately the configuration of an equilateral triangle. Each marker consisted of two fine silk threads sutured in a small cross to the epicardium. In every heart, one marker triplet was located at the anterior midventricle, which served as the control region. The other triplet was placed at one of five test sites chosen in random order. The six epicardial regions were located using a linearly elastic (Silastic) template. The centerlines of the anterior and posterior marker triangles lay approximately 20% around the left ventricular circumference from the anterior and posterior borders, respectively. We chose these positions because they are generally clear of major coronary vessels. The centroids of the basal, midventricular, and apical triangles were located approximately 30%, 50%, and 75%, respectively, along the ventricular meridian between the mitral ring and the apex.

**Experimental Recording System**

The heart was transferred to the experimental recording system illustrated in Figure 1. Supported by a rigid bar, the aortic cannula was located at the axis of a 45° biplane video camera arrangement and connected to a perfusate reservoir containing room temperature cardioplegic solution and 1.5% dextran. The mitral valve cannula was connected to a loading column that could be manually raised to increase the left ventricular filling pressure. Ventricular pressure was measured with respect to the level of the aortic valve using a strain gauge pressure transducer and could be varied from −2 to 38 mm Hg (−0.3 to 5.0 kPa). The ventricular volume change was measured from the pressure difference between the loading column and a parallel reference column. The camera positions, perfusate flow rate, and ventricular pressure and volume change were monitored continuously by a microcomputer, which saved the data on command and simultaneously switched the camera inputs to a video cassette.
recorder so that sequential biplane views of the heart were recorded.

Protocol

The coronary circulation was perfused for 1 minute with the room temperature cardioplegic solution. The duration of hypothermia, from arrest to room temperature perfusion, was not permitted to exceed 40 minutes. The left ventricle was then loaded to a pressure of 30–40 mm Hg (4.0–5.3 kPa) and unloaded after any surplus fluid had been squeezed from the vascular bed. This was done to minimize possible differences between successive filling cycles due to redistribution or loss of coronary perfusate in the ventricular wall.

We recorded the control and test regions in random order without coronary perfusion. For each site, the markers were recorded at 20–30 static loads obtained by raising the fluid column to increase the ventricular volume by increments of approximately 2 ml. In each case, a final recording was made after the pressure had been returned to the reference level as a check for leakage and stability. The cannula seals were inspected for patency during filling, and hearts for which the net fluid loss exceeded 3 ml were excluded from analysis. To minimize possible changes in the material properties of the ventricular myocardium due to ischemic contracture, a maximum of 25 minutes from the time of room temperature perfusion was allowed to record both regions.

Following recording, the heart was submerged in room temperature cardioplegic solution and the loading line was clamped at zero ventricular pressure. The coronary circulation was then perfused with 3% formalin in phosphate buffer for 20–30 minutes and flushed with phosphate buffer for a further 5 minutes. Both ventricular cavities were cast using silicon rubber, and the heart was stored in phosphate buffer. Later, the position of each marker was measured on the fixed heart. Epicardial fiber angles with respect to the circumferential direction were measured at all six regions, and the volumes of the silicon rubber ventricular casts were found by water displacement.

Analysis

The three-dimensional positions of the epicardial markers were reconstructed twice from the biplane video recordings using the computer-aided technique described in McCulloch et al. We had previously seen that the mean absolute accuracy of the measurements was within 0.25 mm, provided that the markers were not on the periphery of the image; accordingly we excluded such recordings from the analysis. To assess the variability of the epicardial measurements and improve the reliability of the derived deformations, each marker position on the unloaded heart was reconstructed at least five times, and the means and variances of the coordinates were calculated.

The measured displacements were analyzed using homogeneous strain theory to obtain a complete description of local epicardial deformations that was independent of the orientation of the marker triangle. With this method, the deformed state of a small epicardial segment is represented by a linear mapping (known as the deformation gradient tensor) of the corresponding undeformed configuration. It has been shown that applying homogeneous strain theory to measurements of epicardial wall motion is valid when the markers are less than 15 mm apart; the region bounded by the markers is then sufficiently small that deformations do not vary significantly within it. A more detailed description of the homogeneous strain analysis is given in McCulloch et al.

To isolate epicardial deformations from the rigid-body translation of the region and to reduce the analysis to two dimensions, the three-dimensional marker positions were transformed to a local Cartesian coordinate system lying in the plane of the marker triangle with its origin at the centroid. The local longitudinal axis was formed by the intersection of the marker plane with the vertical plane containing the axis of the aortic cannula and passing through the centroid. The local circumferential axis was then defined to be perpendicular to the local longitudinal axis and also in the plane of the three markers.

The deformation gradient tensor was separated by polar decomposition into the orthogonal rotation tensor, which describes the rigid body rotation of an epicardial segment about the local origin, and the right stretch tensor, which describes the pure, two-dimensional extension of the epicardial region. The in-plane strain at this point on the epicardium is dependent only on the stretch tensor. From the rotation tensor we obtained the epicardial rotation angle (clockwise positive). From the stretch tensor we computed the mutually perpendicular principal epicardial extensions, which we expressed as the major (maximum) and minor (minimum) percent stretches, \( \lambda_1 \) and \( \lambda_2 \), respectively. The orientation of the major stretch to the circumferential direction is called the principal angle (counterclockwise positive). A third extension, representing the percent change in local ventricular wall thickness \( \lambda_3 \), was derived from the principal epicardial extensions by assuming the myocardium to be incompressible.

As well as the indexes of deformation, we computed the angular displacement of the centroid of the marker triangle about the aortic axis of the ventricle; viewed from the base, a positive value implies a counterclockwise ventricular twist. Although this angular translation is only one of the three components of the displacement of the marker centroid, it was sufficiently consistent to include with the results. However, it may reflect ventricular bending and other three-dimensional deformations as well as torsion or twisting about the aortic axis of the ventricle.

The ventricular volume changes, angular translation, principal extensions (\( \lambda_1, \lambda_2, \) and \( \lambda_3 \)), and...
epicardial rotation were all referred to the state at which the ventricular filling pressure was zero at the level of the aortic valve. Although measurement errors were small (SD<0.1 mm), slight variations in the reconstructed marker coordinates were amplified considerably in the analysis. Therefore, to minimize the effects of scatter on the individual results, the marker coordinates were smoothed by a three-point interpolating function prior to the deformation analysis; the reference configuration was then determined by linear interpolation of the coordinates between the two recordings spanning zero pressure. For the collected results, the deformations were calculated using marker coordinates that were interpolated independently from least-square cubic functions of left ventricular volume. The reference coordinates were then obtained directly from the fitted curves. The standard deviations of the measurements about the interpolated functions provided further information on the variability of the measurements.

Results

Epicardial motion was analyzed in six of the eight isolated hearts studied. One heart (Experiment 2-1) was excluded from the deformation analysis owing to excessive leakage through the mitral valve cannula seal during left ventricular filling, and another (Experiment 2-3) was excluded because the markers at the midanterior control site lay in the periphery of the recorded image, where measurement errors were unacceptably high. In Experiment 2-6 we were unable to record the test region within the 25-minute time limit, but since the midanterior control region had already been recorded, the control data for this heart were included with the results. The dog weights, left ventricular cast volumes, and the weights of the fixed myocardium are summarized in Table 1 together with the regions recorded and the times taken to perform the experimental procedures. The mean time to conduct one filling cycle of 20-30 recordings was 244±23 (range, 206-275) seconds.

The mean position of the centroid of the midanterior marker triplet was 55.4±8.6% of the distance along the longitudinal axis of the fixed ventricle. In all of the fixed hearts but one (Experiment 2-8), we measured epicardial fiber angles at the six sites. The angles were all negative with respect to the ventricular circumference (as the pitch of a left-handed helix). They are shown in Table 2. In general, the epicardial fiber orientations in our preparation were similar to those reported by other authors, though slightly less longitudinal.

Measurement Errors

The variances in the coordinates were averaged to provide an overall estimate, $S_x$, of the measurement error at each site. This, together with the undeformed distance between the markers, was used to calculate the corresponding standard deviation, $S_A$, in the derived percents of stretch. A summary of these errors, obtained both by sampling and from the polynomial regression, is given in Table 3.

Pressure-Volume Relations

The mean range of left ventricular pressure loads applied to the six isolated hearts was $-1.6±0.7$ to $34.4±2.9$ mm Hg ($-0.2±0.1$ to $4.6±0.4$ kPa). Referred to the volume at zero pressure, the minimum ventricular volume change averaged $-6.3±3.1$ ml, and the maximum averaged $40.2±3.3$ ml, an absolute increase of 100% above the mean ventricular cast volume. Volume changes, interpolated from least-square cubic functions of pressure, were averaged to plot the mean passive pressure-volume

TABLE 1. Summary of Weights, Volumes, and Times in Six Isolated Arrested Dog Hearts

<table>
<thead>
<tr>
<th>Experiment number</th>
<th>Dog weight (kg)</th>
<th>Myocardial weight (g)</th>
<th>LV cast volume (ml)</th>
<th>Hypothermic duration (min)</th>
<th>Total duration (min)</th>
<th>Region*</th>
</tr>
</thead>
<tbody>
<tr>
<td>2-2</td>
<td>24</td>
<td>132</td>
<td>42</td>
<td>40</td>
<td>63</td>
<td>PA</td>
</tr>
<tr>
<td>2-4</td>
<td>24</td>
<td>160</td>
<td>54</td>
<td>32</td>
<td>51</td>
<td>AM</td>
</tr>
<tr>
<td>2-5</td>
<td>25</td>
<td>126</td>
<td>38</td>
<td>32</td>
<td>49</td>
<td>AM</td>
</tr>
<tr>
<td>2-6</td>
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<td>173</td>
<td>39</td>
<td>40</td>
<td>56</td>
<td>AM</td>
</tr>
<tr>
<td>2-7</td>
<td>21</td>
<td>128</td>
<td>27</td>
<td>38</td>
<td>51</td>
<td>AM</td>
</tr>
<tr>
<td>2-8</td>
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<td>153</td>
<td>40</td>
<td>37</td>
<td>52</td>
<td>AM</td>
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<td>145</td>
<td>40</td>
<td>37</td>
<td>54</td>
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</tr>
<tr>
<td>SD</td>
<td>3</td>
<td>19</td>
<td>9</td>
<td>4</td>
<td>5</td>
<td></td>
</tr>
</tbody>
</table>

LV, left ventricular.  
*AA, anterior apex; AM, anterior midventricle; AB, anterior base; PA, posterior apex; PM, posterior midventricle; PB, posterior base.

TABLE 2. Mean Epicardial Fiber Angles at Six Sites in Five Dog Hearts

<table>
<thead>
<tr>
<th>Region</th>
<th>Anterior wall fiber angle (°)</th>
<th>Posterior wall fiber angle (°)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Base</td>
<td>$-49±5^*$</td>
<td>$-51±8$</td>
</tr>
<tr>
<td>Midventricle</td>
<td>$-42±7$</td>
<td>$-58±7$</td>
</tr>
<tr>
<td>Apex</td>
<td>$-35±16$</td>
<td>$-49±7$</td>
</tr>
</tbody>
</table>

Values are mean±SD.  
*The negative angles indicate that the fiber axes were rotated clockwise from circumferential.
The error bars indicate 1 SD. The mean ventricular cavity volume at zero pressure was 40 ml.

FIGURE 2. Mean passive left ventricular (LV) pressure-volume relation, averaged over 11 static loading cycles from six hearts. The error bars indicate 1 SD. The mean ventricular cavity volume at zero pressure was 40 ml.

Table 3. Summary of Intermarker Distances and Errors of Measurement of Marker Coordinates and Segment Extensions

<table>
<thead>
<tr>
<th>Undeformed intermarker distance (mm)</th>
<th>Sampled error</th>
<th>Fitting error</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>$S_x$</td>
<td>$S_x$</td>
</tr>
<tr>
<td></td>
<td>(mm)</td>
<td>(%)</td>
</tr>
<tr>
<td>Minimum</td>
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<td>9.7</td>
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<tr>
<td>Maximum</td>
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<td>19.6</td>
</tr>
<tr>
<td>Mean</td>
<td>14.0</td>
<td>14.0</td>
</tr>
</tbody>
</table>

*S*, standard deviations in the marker coordinates. 
†$S_x$, equivalent absolute errors in the percent of segment extensions. 
‡Mean standard deviation equals root mean variance.

The pattern of epicardial deformations was generally similar at the other test regions, but there were significant regional variations in the magnitude and nonuniformity of epicardial stretches. For example, at the posterior apex, epicardial extensions were highly nonuniform. As Figure 8 shows, the major stretch was more than four times as great as the minor stretch. The wall thinning and major stretch were greater at the posterior apex than they were at the basal and midventricular levels of the posterior wall. The extensions at the control site were comparatively uniform and typical of the midanterior results. The principal angles in this heart were well defined at both regions, decreasing with filling from approximately −20° to below −60° on the anterior midventricle and from about −40° to −60° on the posterior apex.

To compare the kinematic results from different hearts, variations due to differences in heart size were minimized by expressing the left ventricular...
volume changes as percents of the cavity cast volumes shown in Table 1. In Table 4, the mean principal extensions (\( \Delta_{\text{I}}, \Delta_{\text{II}}, \) and \( \Delta_{\text{III}} \)), principal angles, epicardial rotations, and angular translations at the midanterior control site are compared with the deformations at the five other regions.

The standard deviations in the midanterior deformations give an indication of the variability in the results and of the significance of regional differences. The small variance associated with the major stretch suggests that, by normalizing the volume changes, the effects of variations in heart size were largely eliminated. The minor stretch and derived wall thinning were considerably more variable than the major stretch. We have assumed, for the purpose of regional comparison, that the variances in the principal extensions, epicardial rotations, and translations at each test site are the same as those at the control site. However, the variability in the principal angle depends on the difference between

**Table 4. Comparisons of Kinematic Results of Regional Epicardial Deformations in Different Dog Hearts**

<table>
<thead>
<tr>
<th>Region</th>
<th>LV volume change (%)</th>
<th>Principal Extensions (%)</th>
<th>Principal angle (°)</th>
<th>Epicardial rotation (°)</th>
<th>Translation angle (°)</th>
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</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>( \Delta_{\text{I}} )</td>
<td>( \Delta_{\text{II}} )</td>
<td>(-\Delta_{\text{III}})</td>
<td></td>
</tr>
<tr>
<td>Anterior</td>
<td></td>
<td>2.5±0.5</td>
<td>1.2±0.6</td>
<td>3.6±0.3</td>
<td>-22±32</td>
</tr>
<tr>
<td>midventricle</td>
<td>(n=6)†</td>
<td>60</td>
<td>72±0.6</td>
<td>10.9±1.4</td>
<td>-22±33</td>
</tr>
<tr>
<td></td>
<td></td>
<td>60</td>
<td>7.3±1.0</td>
<td>4.6±1.3</td>
<td>-22±35</td>
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<tr>
<td></td>
<td></td>
<td>75</td>
<td>12.8±1.8</td>
<td>8.4±2.6</td>
<td>-25±1.7</td>
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<td></td>
<td>75</td>
<td>12.0</td>
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<tr>
<td>Anterior</td>
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<td>75</td>
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<td>3.9</td>
<td>-59</td>
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</table>

LV, left ventricular.

*\( \Delta_{\text{I}}, \) major stretch; \( \Delta_{\text{II}} \), minor stretch; \(-\Delta_{\text{III}}\), wall thinning.

†Values are mean±SD.

**Figure 3.** Pressure-volume data from Experiment 2-7 fitted with least-square cubic polynomials. The broken curve corresponds to the later of the two filling cycles and indicates a loss of ventricular compliance in the 9-minute interval between the cycles. The ventricular cast volume was 27 ml. LV, left ventricular.
the principal epicardial extensions. The standard deviations in the principal angles are, therefore, quite high for the relatively uniform stretches at the anterior midventricle.

In all regions, the principal extensions were generally linear with ventricular volume change. Epicardial extensions varied markedly in magnitude with region and orientation. The major stretch and wall thinning each increased significantly from the thick-walled basal regions to the thin-walled apical regions of both the anterior and the posterior walls. The longitudinal distribution of passive stretches was similar to that of epicardial shortening measured in the intact heart. Minor epicardial stretches were less consistent than major stretches, reflecting the considerable regional variation in the

**Figure 4.** Percent of epicardial segment extensions plotted against ventricular volume change for Experiment 2-7 at the midanterior control site (a) and the posterior base (b). The actual marker positions are shown at the top left, and the comparison shows that epicardial stretches at the posterior base were smaller and more nonuniform than those at the anterior midventricle. Note the different scales on the ordinates in (a) and (b). LV, left ventricular.

**Figure 5.** Principal extensions (major stretch $\mathbf{A_1}$, minor stretch $\mathbf{A_2}$, and wall thinning $-\mathbf{A_3}$) plotted against volume change for Experiment 2-7. a: Anterior midventricle. b: Posterior base. Note the different scales on the ordinates in (a) and (b). LV, left ventricular.
uniformity of epicardial stretches. Basal extensions were less uniform than midventricular stretches, while apical stretches were uniform on the anterior wall but highly nonuniform on the posterior wall. The direction of the major stretch was variable but generally in the same quadrant as the fiber angles (negative principal angle). In the dilated state (75% filling), the principal angle was within 10° of the epicardial fiber angle at all regions except the posterior midventricle, where the difference between the major and minor stretches was not statistically significant. The principal direction usually became more longitudinal during filling; the overall mean principal angle decreased from -23° (at 15–45% left ventricular volume change) to -45° (at 75% volume change).

The epicardial rigid body rotation was consistently negative, indicating a counterclockwise rotation of the epicardial segment. Rotation varied linearly with ventricular volume change and exhibited significant regional differences in magnitude. Although no clear pattern is revealed in Table 4, epicardial rotation at both apical regions was considerably greater than the corresponding control value measured at the anterior midventricle. And the rotations at the basal regions were lower than those in the control. Moreover, epicardial rotation was greatest in the regions of most uniform stretch (anterior apex and posterior midventricle) and least in the regions of most nonuniform stretch (posterior base and posterior apex).

The angular translation of the marker centroid was always counterclockwise (viewed from the base). On both the anterior and the posterior walls, it increased significantly from the base, where the heart was constrained, to the apex. It is not possible to isolate the contributions of flexural and torsional deformations to this component of the translation.

Kinematic Model

The finding that, in the dilated left ventricle, the principal angle tends to coincide with the local epicardial fiber orientation suggests that, during passive filling, the epicardium stretches preferentially along the fiber axis to maximize the end-diastolic fiber length. Although this result has not been reported before, it has been verified that epicardial systolic shortening within the free wall of the canine left ventricle tends to be greatest in the fiber direction.9,12 The description of the ventricular myocardium as a continuum of fiber helices with a smooth transmural change in pitch has been used to model the kinematics of systole.19-21 The epicardial deformations measured in this study admit of a similar interpretation: If the principal angle is the pitch of the left-handed epicardial fiber helix in the undeformed state, then the major stretch, A\textsubscript{m}, is the fiber extension and the epicardial rotation is the change in pitch of the helix as it lengthens during filling. The negative rotation angles measured at all sites therefore suggest that the pitches of the epicardial fiber helices reduced with load. The consistently counterclockwise angular translations of the epicardium, relative to the base, are readily explained by the helix interpretation: A left-handed helix follows a counterclockwise path from base to apex. Therefore, the measured translations imply that the preferential stretch of the fibers was not fully accommodated by the reduction in pitch.

To determine whether the kinematic results of this study are quantitatively consistent with the preferential extension of left-handed fiber helices, we adopt a geometric model proposed by Streeter and Hanna,22 who found that the epicardial surface of the canine left ventricle was well approximated by an ellipsoid that remained confocal throughout the cardiac cycle. A helix that inscribes a specified number of turns on an ellipsoidal surface is represented in parametric form using differential geometry.23
The rotation about the axis of symmetry and the change in pitch that result from an extension prescribed at any point on the helix are then easily derived.

We use an ellipsoid truncated halfway along the basal semiaxis, with major radius $a$ and minor radius $b$, to describe the left ventricular epicardium in the undeformed reference state. A similar ellipsoid, with dimensions $A$ and $B$, represents the deformed epicardium (see Figure 9). The position vector $x$ of a point on a general helix inscribed on the undeformed ellipsoid, which is symmetric about the $x_1$ axis, is given by the parametric equation

$$x = a \sin \theta \; i_1 + b \cos \theta \; s$$

The parameter of the helix is $\theta$, and the number of quarter-turns made by the helix between apex and equator is $w$, where a positive $w$ defines a left-handed helix with a positive pitch. On the deformed epicardial ellipsoid, the helix is similarly defined

$$X = A \sin \theta \; i_1 + B \cos \theta \; s$$

The deformed helix constant $W$ is not, in general, equal to $w$, and therefore a point on the helix is able to rotate during the deformation about the axis of symmetry.

The elements of arc length along the helix, $ds$ in the reference state and $dS$ in the deformed state, are defined by

$$ds = \left| \frac{dx}{d\theta} \right|$$

Hence, the local extension $dS/ds$ of the helix is derived by

$$dS = \frac{dX}{ds} = \left| \frac{dX}{d\theta} \right| = \frac{(A^2 + B^2)^{3/2} \cos \theta + B^2 \sin \theta}{(a^2 + b^2)^{3/2} \cos \theta + b^2 \sin \theta}$$

FIGURE 8. Percent of principal extensions vs. ventricular volume change in Experiment 2-2 at anterior midventricle (a) and posterior apex (b). At both sites, the principal angle was clearly resolved and approached $-60^\circ$ at high ventricular pressures. LV, left ventricular.

FIGURE 9. Kinematic model of the epicardial surface of the left ventricle. A helix aligned with the local epicardial fiber direction is inscribed on truncated ellipsoids representing the undeformed and deformed epicardium.
Let $dh$ and $dH$ be undeformed and deformed elements, respectively, of longitudinal arc length on the ellipsoidal surface. The pitch angles, $p$, for the undeformed helix and $P$ for the deformed helix, are then defined (with respect to the circumferential direction) by

$$\sin p = \frac{dh}{ds} = \left| \frac{dS}{dt} \right| \sqrt{\frac{a^2 \cos^2 \theta + b^2 \sin^2 \theta}{(a^2 + b^2 w^2) \cos^2 \theta + b^2 \sin^2 \theta}}$$

(5)

and

$$\sin P = \frac{dH}{dS} = \left| \frac{dS}{dt} \right| \sqrt{\frac{A^2 \cos^2 \theta + B^2 \sin^2 \theta}{A^2 + B^2 \frac{W^2}{w^2} \cos^2 \theta + B^2 \sin^2 \theta}}$$

(6)

With Equation 4, the deformed pitch resulting from an extension $\lambda = dS/ds$ may be expressed in terms of the original pitch and the position, $t$, on the helix.

$$\sin P = \sin \frac{p}{\lambda} \sqrt{\frac{A^2 \cos^2 \theta + B^2 \sin^2 \theta}{a^2 \cos^2 \theta + b^2 \sin^2 \theta}}$$

(7)

The rotation at a point along the helix given by $t$ is obtained from $W$ and $w$, which are found by rearranging Equations 5 and 6.

$$w = \frac{\sqrt{a^2 + b^2 \tan^2 \theta}}{b \tan \theta}$$

(8)

$$W = \frac{\sqrt{A^2 + B^2 \tan^2 \theta}}{B \tan P}$$

(9)

Relative to the base, where $x_1 = -a/2$ and $X_1 = -A/2$ and therefore $t = -\pi/6$, the rotation about the axis of the helix is ($W - w$) ($t + \pi/6$).

Using a constant focal length of 3.75 cm$^2$ and the mean left ventricular cast volume from Table 1 of 40 ml and assuming a constant wall volume of 100 ml, the dimensions of the undeformed ellipsoid are found to be $a = 4.52$ cm and $b = 2.52$ cm. To model the mean midanterior results, we let $dS/ds$ equal the mean measured major extension ratio ($A = 1 + \Lambda/100$) and the undeformed pitch, $p$, equal the mean magnitude of the principal angle (from Table 4). The mean location of the midventricular sites corresponds to a value for the parameter $t$ of approximately 20°. The dimensions of the deformed epicardial ellipsoid and the rotation and translation angles that are predicted by the helix model are shown in Table 5. The model predictions are similar to the mean rotations and translations measured at the anterior midventricle (see Table 4) though slightly lower at low left ventricular volumes and greater at high volumes. The coefficients of correlation between the predictions and the experimental measurements (from single degree-of-freedom fits) were 0.92 for the rotations and 0.85 for the translations.

Table 5. Predictions of Dimensions of Deformed Epicardial Ellipsoid and Rotation and Translation Angles by the Helix Model for Anterior Midventricle

<table>
<thead>
<tr>
<th>LV volume change (%)</th>
<th>Major radius (cm)</th>
<th>Minor radius (cm)</th>
<th>Epicardial rotation (°)</th>
<th>Translation angle (°)</th>
</tr>
</thead>
<tbody>
<tr>
<td>15</td>
<td>4.56</td>
<td>2.59</td>
<td>-0.36</td>
<td>0.28</td>
</tr>
<tr>
<td>30</td>
<td>4.59</td>
<td>2.65</td>
<td>-0.71</td>
<td>0.75</td>
</tr>
<tr>
<td>45</td>
<td>4.63</td>
<td>2.71</td>
<td>-1.00</td>
<td>0.89</td>
</tr>
<tr>
<td>60</td>
<td>4.66</td>
<td>2.77</td>
<td>-3.66</td>
<td>5.53</td>
</tr>
<tr>
<td>75</td>
<td>4.70</td>
<td>2.83</td>
<td>-5.22</td>
<td>8.39</td>
</tr>
</tbody>
</table>

LV, left ventricular.

Discussion

We have measured regional epicardial deformations in a controlled experimental model of the diastolic heart. The loading conditions of the preparation were carefully controlled without altering the essential mechanical and geometric properties of the passive left ventricle. The anterior midventricle served as a control site so that the variability in the results could be assessed. Homogeneous strain theory was used to provide a description of the stretch and rotation components of the epicardial deformation gradients that was independent of the orientation of the marker triangle and suitable for regional comparison. All of the computed deformations were referred to the undeformed state at which the ventricular filling pressure was zero at the level of the aortic valve. This reference state has the posterior base, and posterior apex). The angular displacements also correlated well ($r = 0.87-0.99$) at the posterior base and posterior apex, but there was a negative correlation ($r = -0.96$) at the anterior base. There was no correlation between the predicted and measured data at the anterior apex, where the measured principal angle was poorly defined and coincided with the fiber angle only at high filling pressures. Nor was there any correlation at the posterior midventricle, where the measured principal angle was also ill-defined and did not coincide with the fiber angle.

Clearly, this kinematic model is too simple to predict regional variations in epicardial rotation and angular translation accurately. However, the model was able to predict the mean midanterior rotations and translations with reasonable accuracy, and there was generally good correlation at the sites where the model assumptions were satisfied. This suggests that the observed epicardial deformations were generally consistent with a preferential extension in the direction of a left-handed epicardial fiber helix. In the following section, we discuss the implications of this kinematic interpretation for continuum mechanics models of the passive myocardium and the relevance of the results to the physiology of the heart in diastole.
advantage that when there is no transmural pressure gradient the myocardium may be approxi-
mately stress free. In mammalian hearts fixed at zero diastolic pressure, it has been found\textsuperscript{24,25} that sarcomere lengths do not vary significantly through the left ventricular wall and average 1.93 \textmu m, the resting sarcomere length at which there is no passive tension.\textsuperscript{26} The net stress in the left ventricular wall at the chosen reference state will be determined by the small hydrostatic pressure gradient between the aorta and the apex (approximately 5 mm Hg), the body force of the heart’s own weight, and any residual stress in the myocardium.\textsuperscript{27}

The analysis revealed a complex but consistent pattern of epicardial strain with significant regional variations. The magnitudes of the maximum component of stretch and the derived wall thinning increased significantly from base to apex on both the anterior and the posterior walls. Since the measured extensions were referred to an approximately stress-free reference state, this finding suggests that the increase in systolic shortening toward the apex reported by others\textsuperscript{6-14} mostly reflects an increase in end-diastolic sarcomere lengths rather than a decrease in end-systolic sarcomere lengths. Although the cavity pressure at the apex of the hanging preparation was greater than that at the base, this does not explain the longitudinal variation in epicardial extensions because the same pressure gradient existed in the undeformed state to which the deformations were referred. Nevertheless, it should be recognized that a different regional pattern of epicardial strains might be found in an intact heart. Epicardial deformations were not measured at the extremes of the ventricular long axis. The trend seen here over the central 50% of the left ventricular axis may not continue toward the apical infundibulum or the mitral ring.

The magnitude of epicardial stretch may depend on the local thickness of the ventricular wall. In an extensive study of the geometry of the canine left ventricle, Streeter and Hanna\textsuperscript{22} found that the average wall thickness at end diastole was 9.3 mm at the equator and 5.6 mm at the apex. In this study, we observed wall thicknesses exceeding 12 mm at the base and as low as 2 mm at the apex of the unloaded heart. The valve canulæ and the mitral ring may also have limited stretch at the base.

The angular translation of the epicardial marker centroid about the vertical aortic axis also increased from base to apex, presumably owing to the constraint at the base imposed by the rigid cannula system. Compared with the anterior midventricle, epicardial rotations were greater at apical regions and smaller at basal sites. Differences between the deformations on the anterior and posterior free walls were not great enough to be significant for our small sample size.

The biaxial extension of the epicardium was usually nonuniform, and the direction of the major stretch tended to coincide with the local epicardial fiber axis. This implies that, during passive filling, the left-handed epicardial fiber helix stretches preferentially to maximize end-diastolic fiber length. To interpret the experimental deformations in the context of the helical fibrous structure of the ventricular myocardium, we derived a simple kinematic model, which showed that the measured epicardial rotations and angular translations were consistent with the change in pitch and axial twist of a left-handed fiber helix. Similar analyses, proposed by Sallin,\textsuperscript{19} Streeter and Hanna,\textsuperscript{20} and Meier et al,\textsuperscript{21} have been used to interpret ventricular wall motion during systole. However, none of these models permitted torsion to occur during ejection. Recently however, Ingels et al\textsuperscript{28} measured systolic changes in chord length along an axis approximately 45° clockwise from the circumference using widely separated tantalum screws implanted in the left ventricular midwall of human transplant hearts. Using a cylindrical model, they found that this "oblique shortening" was kinematically consistent with observed systolic torsional displacements that were opposite in direction to those measured in this study for passive filling.

In most continuum mechanics models of ventricular filling, it has been assumed that the passive myocardium is an isotropic material and therefore cannot be affected by muscle fiber orientation.\textsuperscript{8} However, the results of this study are incompatible with isotropic axisymmetric models, which can predict only principal stretches aligned with the circumferential and longitudinal axes and do not predict ventricular torsion. It is unlikely that a more realistic geometric representation of the left ventri-
cle would fully account for the discrepancies. Our findings indicate that the epicardial layer of the passive left ventricle is anisotropic with respect to the fiber axis. Possibly, the epicardial muscle is most compliant along the fiber axis. This would imply that epicardial deformations are not strongly influenced by deeper fibers and, therefore, that the coupling between adjacent fiber layers is relatively weak. Alternatively, the myocardium is stiffer in the fiber direction and the deformations observed on the epicardium are influenced mainly by the right-handed endocardial fiber helices. In this case, we would expect tethering between adjacent fibers to be more significant\textsuperscript{12,13} and the modulus of shear to be higher. Although there has been no definitive measurement of the multiaxial elastic properties of cardiac muscle, biaxial testing of canine myocardium\textsuperscript{29,30} supports the conclusion that passive car-
diac muscle is anisotropic with respect to the fiber field. There is also evidence of structural anisot-
ropy: Scanning electron microscopy\textsuperscript{31,32} has revealed an extensive collagen skeleton connecting and sur-
rounding adjacent myocytes that may play an important role in governing the transverse stiffness and shear modulus of the myocardium.

The epimysial connective tissue that surrounds the heart muscle\textsuperscript{33} includes the superficial epica-
dium (visceral pericardium). Recently, it has been shown that the biaxial material properties of the epicardium differ significantly from those of the myocardium and are more like those of the pericardium. Therefore, deformations measured on the epicardium may be influenced by this relatively stiff membrane.

In addition to the consistent pattern of epicardial stretch, rotation, and translation described above, regional epicardial deformations also exhibited some other trends. The direction of the major stretch tended to become more longitudinal with filling from an approximately circumferential orientation at low pressures. In view of the foregoing interpretation, this may indicate that the degree of material anisotropy of passive myocardium increases with strain or that at low filling pressures, midwall and endocardial fibers have a greater effect on epicardial kinematics than at high pressures. The counterclockwise epicardial rotation was greatest in the regions of most uniform stretch and least in those that stretched most nonuniformly. This unusual behavior is not explained by the helix interpretation, but it may reflect regional variations in transmural coupling. If the stretch of the epicardium transverse to the fiber axis is influenced by the right-handed endocardial fiber helices, then the rotation may also be affected.

A significant physiological result of this study is that the epicardium of the passive left ventricle deforms during filling in a manner that apparently maximizes end-diastolic fiber lengths and, hence, the tension developed during systole. Since the principal angles were most commonly between −20° and −60°, there were significant epicardial shear strains with respect to the circumferential and longitudinal axes. In the mechanics of cylindrical bodies, shear strain in the plane normal to the radial axis is appropriately referred to as torsional shear. This is because pure torsion corresponds, by definition, to a constant gradient of circumferential displacement with respect to the axial coordinate. This displacement gradient gives rise to the shear strain $\gamma_{\theta\phi}$ in the Lagrangian strain tensor. Since the fiber axis changes continuously through the wall, there may also be large shear strains throughout the ventricular myocardium at end diastole. However, the role of the corresponding shear stresses in the mechanics of filling is uncertain. Although significant transmural shear strains have been measured in the beating heart, their effects on the mechanics of passive filling have not been assessed. Using a continuum mechanics model of ventricular ejection that incorporated a transmural change in fiber orientation, Arts et al concluded that “interplay between torsion and myocardial contraction” equalizes fiber stresses and fiber shortening across the wall. Therefore, it is possible that material anisotropy referred to the helical fibrous structure of the passive ventricular myocardium both maximizes end-diastolic fiber lengths and allows them to be distributed more uniformly.

To test the above hypotheses, we require a properly formulated model of the left ventricle, based on the laws of continuum mechanics. The results of this study suggest that the model should include several features: 1) the nonlinear kinematic terms associated with the large deformations, 2) the three-dimensional thick-walled geometry of the left ventricle or, at least, the longitudinal variation in wall thickness, 3) material aeolotropy referred to a fiber field with a realistic transmural distribution of fiber angle, and 4) the freedom to twist about the longitudinal axis under static pressure loading.

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**Appendix: Glossary of Continuum Mechanics Terms**

- **Anisotropy**: Material properties that depend on direction.
- **Aeolotropy**: Change of material properties consequent on change of direction.
- **Tensor**: A linear mapping that transforms one vector to another. In a given coordinate system, the components of the tensor may be represented as a matrix.
- **Deformation gradient tensor**: The linear mapping that transforms a vector in the undeformed reference state to its corresponding configuration in the deformed body. For a general, non-homogeneous deformation, this transformation will be constant for only an infinitesimally small vector.
- **Homogeneous strain theory**: The method of estimating deformations from measurements of displacement that relies on the assumption that the variation of deformation is negligible over the domain of the measurements.
- **Polar decomposition**: The decomposition that separates the deformation gradients into the matrix product of the rotation tensor and the stretch tensor.
- **Stretch tensor**: The tensor that describes the shape change of a region during deformation. It uniquely determines the strain tensor.
- **Rotation tensor**: The tensor that represents the rigid body rotation of the region during the deformation.
- **Principal extensions**: The major and minor stretches expressed as extension ratios. These are the eigenvalues of the stretch tensor.
- **Principal axes**: The mutually perpendicular axes of the major and minor stretch. These axes correspond to the eigenvectors of the stretch tensor.
- **Principal angle**: The orientation of the principal axes with respect to the reference axes.
Axisymmetric: Possessing cylindrical symmetry about a single axis.

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