Left ventricular epicardial deformation in isolated arrested dog heart

ANDREW D. MCCULLOCH, BRUCE H. SMAILL, AND PETER J. HUNTER
Department of Physiology, School of Medicine and Department of Theoretical and Applied Mechanics, School of Engineering, University of Auckland, Auckland, New Zealand

We have developed a method for measuring epicardial deformation in the isolated arrested dog heart. A biplane video system was used to record the motion of discrete epicardial markers at midanterior sites (n = 4 hearts) and midposterior sites (n = 1) during quasi-static left ventricular (LV) filling. Experimental procedures, performed at room temperature, were completed within 20 min, and LV pressure-volume curves were repeatable and within the range of data presented by other authors. To obtain a complete description of local deformation, epicardial displacements derived from the video record were analyzed using homogeneous strain theory. Local epicardial strain was nonuniform; the mean ranges of midanterior major and minor extensions were 0-13.9 and 0-7.2%, respectively, for LV filling pressures of 0-20 mmHg. For the midanterior wall, the mean orientation of the major extension was 28-35° below the LV circumference, compared with an orientation of ~62° at the midposterior site. The results demonstrate the value of this preparation for studying passive ventricular mechanics and are not consistent with the predictions of continuum mechanics models of the passive filling left ventricle (11, 18, 21). Yin (25) has emphasized the need for test such models and validate the assumptions on which they are based. In the absence of reliable estimates of stress, this can only be achieved by comparing predicted strains with experimental measures of ventricular wall strain obtained under carefully controlled conditions.

We describe an experimental model of the diastolic ventricle developed to study factors that influence regional myocardial deformations during diastole and determine presystolic cardiac muscle fiber lengths. Accurate estimates of the distributions of stress and strain in the ventricular wall during diastole are therefore required.

Regional myocardial strains in the intact beating heart have been approximated from the displacement of markers located on the epicardium (1, 16) or within the ventricular wall (5, 6, 23). This experimental model does not lend itself to the systematic analysis of myocardial deformation during diastole, since no undeformed reference state can be identified. Furthermore, ventricular filling may be influenced by a variety of factors, including the following: variations in loading due to the interaction of right and left ventricles (10), inertial and viscous dynamic effects (10), and alterations in coronary perfusion pressure and flow (22). With in vivo preparations, it is difficult to account for the contributions that such factors make to diastolic ventricular wall deformation. Estimates of ventricular wall stress obtained from measured intramyocardial forces have been found to be unreliable (9). The only alternative to direct measurement of stress is mathematical modeling based on the laws of continuum mechanics (25), which requires experimental information on ventricular shape and dimensions, the mechanical properties of cardiac muscle, and the ventricular pressure loading conditions. Models using simplified representations of shape and material behavior have predicted passive ventricular volume changes with reasonable accuracy (12), but a precise analysis of regional myocardial stresses and strains requires the solution of equations that describe the complex three-dimensional geometry, the large deformations, and the nonlinear anisotropic constitutive properties of the ventricular wall (2). This has been made possible by the development of the finite element method, a computational technique that has been used to predict the distributions of wall stress and strain in the passive filling left ventricle (11, 18, 21). Yin (25) has emphasized the need to test such models and validate the assumptions on which they are based. In the absence of reliable estimates of stress, this can only be achieved by comparing predicted strains with experimental measures of ventricular wall strain obtained under carefully controlled conditions.
used to record the motion of discrete markers at selected epicardial sites. The measured epicardial displacements were analyzed using homogeneous strain theory to provide a complete description of local deformation.

**METHODS**

**Preparation.** Five mongrel dogs weighing 20–33 kg were anesthetized with thiopental sodium (25 mg/kg iv) and maintained with 1.5% halothane using positive-pressure ventilation. A catheter was introduced into the left ventricle (LV) via the common carotid artery and the heart exposed through a thoracotomy at the fifth intercostal space. After a monitoring period of ~1 h, the aorta was occluded and a 50-ml bolus of cold 10% potassium citrate solution was injected into the LV to induce immediate cardiac arrest. The heart was then rapidly excised, washed, and put into a bath of a chilled hyperkalemic cardioplegic solution (CPS) containing nifedipine (0.2 mg/l). The atria and pulmonary outflow tract were removed and the aorta transected below the arch.

The heart was then mounted on the stainless steel cannula assembly shown in Fig. 1. The aortic cannula consisted of a sleeve and a central vented tube with a plug attached to the end. This cannula was secured 3-4 cm above the aortic root with the plug positioned just below the aortic valves. A heavy silk suture was passed around chordae tendinae to draw mitral valve leaflets around shaft of this cannula, and a rubber seal was locked against mitral ring. Mitral cannula was supported from shaft of aortic cannula. Coronary circulation was then flushed with cold CPS and the right ventricle vented at the apex to prevent the accumulation of coronary effluent.

Three markers, each consisting of two fine silk threads sutured in a small cross to the superficial epicardium, were positioned 8–27 mm apart on the LV free wall to form an upright equilateral triangle. On three hearts, an additional marker was placed below the triangle to form a symmetrical diamond.

**Experimental recording.** The heart and cannula assembly were supported by a rigid bar and positioned in the experimental measurement system illustrated in Fig. 2. The mitral valve cannula was connected to a saline-filled loading column. This column could be raised or lowered manually to alter the LV pressure, which was measured using a strain-gauge pressure transducer (Statham P23Db) at the level of the aortic root. To determine the corresponding LV volume change, the difference between the hydrostatic pressures in the loading column and a parallel reference column was detected by a differential pressure transducer (Validyne). A reservoir 130 cm above the heart contained CPS with added dextran (70,000 mol wt, 1.5%) at room temperature for perfusing the coronary circulation. Perfusion flow was measured using an electromagnetic flowmeter (Gould SP2202) with a cannulating flow probe. The support for the heart and cannula assembly located the center line of the aortic cannula at the axis of a 45° biplane video camera arrangement. The two cameras (JVC 1K-2000, fitted with macro lenses) were fixed to racks that allowed the magnification to be increased linearly by tracking toward the heart. Camera positions, LV pressure, relative LV volume, and coronary flow rate were monitored continuously at a sampling rate of 50 Hz using a microcomputer. The computer was programmed to acquire these data on operator command and, simultaneously, to switch the camera inputs to a video cassette recorder (JVC 6600E) so that sequential biplane views of the heart could be recorded.

**Protocol.** The camera positions were adjusted for optimal magnification and then the heart was perfused briefly with room temperature CPS. The duration of hypothermia (from arrest to perfusion) ranged from 34 to 59 min. Starting at a pressure of 0 mmHg, the motion of the epicardial markers was recorded for a series of 10–20 static loads increasing to a maximum pressure of 20–30 mmHg and returning to 0 mmHg. At each load, the cameras were switched and the data were acquired. These quasi-static loading cycles were performed one to three times for each heart with or without coronary perfusion. The heart was then fixed at 0 mmHg LV pressure by perfusing 100–200 ml of glutaraldehyde (2.5%) in phosphate buffer through the coronary circulation. Biplane views of the heart and markers were recorded before and

![FIG. 1. Isolated heart cannula assembly. Outer sleeve of the aortic cannula was secured above aortic root and central tube was moved to position plug just below valve, blocking left ventricular outflow tract. Perfusion entered coronary circulation through central tube. A cannula inserted through mitral valve was used to fill left ventricle. A suture was passed around chordae tendinae to draw mitral valve leaflets around shaft of this cannula, and a rubber seal was locked against mitral ring. Mitral cannula was supported from shaft of aortic cannula.](image-url)
after fixation. The duration of each recording session (from room temperature perfusion to fixation) was always <17 min, and the total time from arrest ranged from 46 to 64 min. The fixed heart was removed from its support, and both ventricular cavities were cast with silicon rubber. The location of each marker was subsequently measured in cylindrical polar coordinates with respect to an axis between the center of the mitral valve and the apex. Epicardial fiber orientations were also determined at each marker position. After each experiment, biplane views of a calibration test grid were recorded on the video tape with the camera positions unaltered to allow the absolute accuracy of the video measurements to be checked.

Marker coordinate reconstruction. The spatial coordinates of the epicardial markers were reconstructed using a pair of horizontal and vertical cross hairs electronically imposed on the replayed video image. During stop-frame playback, a single operator manipulated the cross hairs to locate each marker in both camera views. The two sets of screen coordinates for each point were then read by the microcomputer.

To convert the screen measurements to physical coordinates, the cameras had to be calibrated. For this purpose an accurate grid (200 x 300 mm with 1-mm ruling and a cumulative accuracy of <0.02 mm) was recorded by each camera over the full range of positions permitted by the racks (50–300 mm). For each camera position, the physical coordinates of selected points on the grid were fitted independently (by least-squares linear regression) as functions of the corresponding screen positions located by the cross hairs; the coefficients of these functions were then fitted as linear functions of camera position using the same method. The form of the resulting transfer functions is given in APPENDIX A. The correlation coefficients of the fitted transfer functions were >0.9999 for both cameras, indicating that nonlinear distortions in the recording system were small.

The equations used to reconstruct the three-dimensional coordinates of the epicardial markers from biplane recordings, given the transfer functions and camera positions, are presented in APPENDIX A. The origin of the right-handed measurement coordinate system (X1, X2, X3) was at the intersection of the axes of symmetry of the camera lenses. The X2 axis coincided with the center line of the aortic cannula, and the X3 axis was defined by the lens axis of camera 1 (see Fig. 2).

Epicardial deformation analysis. To obtain a description of local epicardial deformation that was independent of the orientation of the marker triangle, the recorded motion was analyzed using homogeneous strain theory (19). Although myocardial strains vary throughout the heart and the cardiac cycle, the instantaneous deformation of a myocardial region is homogeneous if the region is sufficiently small that the deformation of every segment within it is the same. The assumption of homogeneous deformation is justified when the epicardial fiber directions and material properties do not vary significantly between the three markers. Meier et al. (16) applied a kinematic analysis to right ventricular wall motion in the intact dog heart and demonstrated that epicardial deformations throughout the cardiac cycle could be regarded as homogeneous for markers spaced <10–15 mm apart. Studies of fiber orientation in the fixed heart, by ourselves and others (20), suggest that midventricular epicardial fibers remain relatively constant over intervals the size of our marker spacings (mean 14 mm at zero load).
To obtain a mathematical description of a general homogeneous deformation, we consider the position vector $\mathbf{x}$ of a material point in the deformed heart to be a function of the pressure load $P$ producing the deformation and the corresponding position vector $\mathbf{x}$ in a zero-pressure reference state

$$\mathbf{x} = \mathbf{x}(\mathbf{x}, P)$$

A linear operator $F$ defines the correspondence between two arbitrarily small segments ($d\mathbf{x}$ and $d\mathbf{x}$) in the deformed and reference configurations

$$d\mathbf{x} = F \cdot d\mathbf{x}$$

where $F$ is a function of $P$ alone. Considering the individual components of $d\mathbf{x}$:

$$d\mathbf{x}_i = \frac{\partial \mathbf{x}_i}{\partial \mathbf{x}_j} \cdot d\mathbf{x}_j = F_{ij} \cdot d\mathbf{x}_j$$

it is evident that the linear operator $F$ is a tensor (or matrix) of deformation gradients.

To isolate the epicardial deformations from the rigid body movement of the ventricular wall and to reduce the analysis to two dimensions, we defined a local coordinate system $(x_1, x_2, x_3)$ that moved with the markers. The approach followed was similar to that described by Meier et al. (15), who defined a local cartesian coordinate system in the plane of three epicardial markers, with its origin at the centroid. The longitudinal axis was tangential to the epicardial meridian that passed through the centroid, the apex, and the aortic root. In the present study, however, we defined the ventricular meridian as the line of intersection of the epicardium and the vertical plane containing the longitudinal axis of the heart and passing through the marker centroid. This ensured that the local coordinate system was referred to a fixed global coordinate system that was independent of gross ventricular deformations.

By expressing the positions of the triangular array of markers in these local coordinates, a $2 \times 2$ matrix of deformation gradients was computed for each load, as outlined in APPENDIX B.

From the polar decomposition theorem (19), the deformation gradient tensor $F$ can be separated into two unique components

$$F = RU$$

where $R$, the orthogonal rotation tensor, describes the local myocardial twist and $U$, the right stretch tensor, defines the two-dimensional extension of the epicardial region. From $R$, we obtain a single angular measure of local epicardial shear or rotation $\alpha$ (clockwise positive) for each pressure. The stretch tensor $U$ is uniquely defined by two eigenvalues $\lambda_1$ and $\lambda_{II}$, associated with eigenvectors $e_1$ and $e_{II}$, satisfying

$$Ue_i = \lambda e_i \text{ where } i = I, II$$

The eigenvalues $\lambda_1$ and $\lambda_{II}$ represent the major and minor epicardial segment length changes (expressed as extension ratios) that are oriented in mutually perpendicular directions defined by the eigenvectors. The angle between the eigenvector $e_1$ associated with the major extension and the local circumferential axis $x_1$ is the principal angle $\Phi(-90^\circ < \Phi \leq 90^\circ)$. A third eigenvalue $\lambda_{III}$ with its eigenvector $e_{III}$ directed transmurally was derived from the principal epicardial extensions by assuming the myocardium to be incompressible, i.e., the volume of a small myocardial cube must remain constant, or

$$\lambda_1\lambda_{II}\lambda_{III} = 1, \text{ giving } \lambda_{III} = 1/(\lambda_1\lambda_{II})$$

This third eigenvalue describes the change in thickness of the epicardial layer. Although transmural deformation cannot be regarded as homogeneous (owing to the variation in fiber direction across the wall) or strictly incompressible (because of the possible redistribution of coronary perfusate), $\lambda_{III}$ provides an estimate of changes in ventricular wall thickness under load.

A physical interpretation of the rotation and stretch tensors is shown in Fig. 3. The equations used to compute the principal extension ratios ($\lambda_1$, $\lambda_{II}$, $\lambda_{III}$), the principal angle ($\Phi$), the epicardial rotation ($\alpha$), and the rigid rotation ($\Theta$) of the epicardium about the aortic axis are given in APPENDIX B. The principal extensions and unprocessed epicardial segment extensions were expressed as percent length changes

$$\Lambda = 100(\lambda - 1)$$

Hence, for each static pressure load, we obtained a record of LV volume change, rigid rotation, epicardial segment stretches, principal extensions and their directions together with epicardial shear, all referred to the zero-load
state.

**Measurement errors and data fitting.** The linear regression analysis used to calibrate the video cameras and recording apparatus provided an indication of measurement accuracy. The standard deviations of the calibration data about the fitted transfer functions were dependent on magnification and ranged from 0.09 to 0.15 mm in the horizontal direction and from 0.14 to 0.26 mm in the vertical direction, for the range of camera positions used in the five studies.

To test the absolute accuracy of the coordinate measurements, five randomly selected points were reconstructed (10 times each) from the biplane images of the calibration grid recorded after each experiment. The mean differences between the computed (observed) and known (expected) absolute coordinates \( \bar{X}_{1:0:E} \), \( \bar{X}_{2:0:E} \), \( \bar{X}_{3:0:E} \) are summarized in Table 1. Although these errors differ significantly from zero, they are approximately constant at a given magnification except at the periphery of the image field; their effect on the accuracy of the deformation analysis must therefore have been small.

To assess the variability in the epicardial measurements and to improve the reliability of the calculated strains, each marker position on the unloaded heart was reconstructed at least 10 times and the means and variances of the coordinates determined. The standard deviations \( S_{X_1}, S_{X_2}, \) and \( S_{X_3} \) of the three coordinates, which varied according to magnification, are shown in Table 1. Although these differences were slight variations in the marker coordinates amplified considerably in the computed deformations, which depended on displacement gradients. The orientation of the principal axis (which is undefined for a uniform biaxial stretch) was particularly sensitive to this variation. Therefore, to minimize the effect of scatter in the measurements without distorting the deformations, the individual marker coordinates were smoothed using a three-point filtering algorithm before the deformations were calculated. For the same reason, it was preferable to compute the epicardial strains shown in Table 2 (see RESULTS) using marker coordinates that had been interpolated from least-square polynomials fitted as functions of LV pressure rather than to interpolate from curves fitted to the derived deformations. By fitting either quadratic or cubic functions to each coordinate of each epicardial marker, another index of overall measurement consistency was provided. The standard deviations of the coordinates \( s_{X_1}, s_{X_2}, \) and \( s_{X_3} \) about the least-square polynomial fits are presented in Table 1.

**RESULTS**

Epicardial deformations were analyzed in five unperfused hearts. Epicardial markers were sutured to the midventricular anterior free wall in four cases and to the midventricular posterior wall in the fifth case.

The static pressure-volume curve for the single cycle of one experiment is shown in Fig. 4A. The arrows indicate the sequence of loading and the hysteresis reflects volume loss due to leakage. The mean pressure-volume curve (ascending limb) for all five hearts is plotted in Fig. 4B. The mean fluid loss on return to zero was 2.3 ± 2.1 ml. Deformation data for the same heart as in Fig. 4A are shown in Fig. 5. The changes in the length of each of the three arms of the marker triangle are plotted as percents of initial lengths in Fig. 5A. The positions of the markers, which were 16–17 mm apart on the anterior free wall, are shown in the inset. The absolute measurement error in the percent stretches was 1.0%, and the data in Fig. 5, B and C clearly demonstrate the nonuniform nature of the epicardial deformations. The major and minor stretches and the local wall thinning all increased with pressure in a nonlinear manner similar to that of the volume change. This is demonstrated in Fig. 5C, which shows the principal extensions varying with volume in an approximately linear fashion. In this experiment, the angle between the direction of the major extension and the local epicardial circumference was −25° (i.e., below the ventricular circumference) for most of the load cycle. The epicardial rotation (clockwise positive) and the rigid rotation of the marker triangle about the aortic axis (counterclockwise positive viewed from the fixed base) are plotted in Fig. 5D.

In three hearts, markers were sutured in a diamond arrangement to form two triplets, and two complete loading cycles were analyzed. In the two other experiments that were both another wall studies, one marker triplet was recorded for a single loading cycle only. In Table 2, the change in LV volume (\( \Delta V \)), major and minor stretches (\( \Delta_1 \) and \( \Delta_2 \)), and derived wall thinning (\( \Delta_{mil} \)) are summarized together with the orientation of the principal stretch (\( \Phi \)), the epicardial twist (\( \phi \)), and the rigid rotation (\( \Theta \)) for representative static LV filling pressures. Data for the LV anterior wall are compared with corresponding results from the single study of the LV posterior wall. These results indicate the consistency of the deformation data obtained for the midventricular region of the anterior free wall. The principal epicardial

**TABLE 1. Mean, absolute reconstruction errors, and SD in measured and fitted marker coordinates**

<table>
<thead>
<tr>
<th>Absolute Error, mm</th>
<th>Measurement Error, mm</th>
<th>Fitting Error, mm</th>
</tr>
</thead>
<tbody>
<tr>
<td>( X_{1:0:E} )</td>
<td>( X_{2:0:E} )</td>
<td>( X_{3:0:E} )</td>
</tr>
<tr>
<td>Minimum</td>
<td>0.16</td>
<td>0.08</td>
</tr>
<tr>
<td>Maximum</td>
<td>0.37</td>
<td>0.21</td>
</tr>
<tr>
<td>Mean</td>
<td>0.23</td>
<td>0.11</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>( S_{X_1} )</th>
<th>( S_{X_2} )</th>
<th>( S_{X_3} )</th>
<th>( \epsilon )</th>
</tr>
</thead>
<tbody>
<tr>
<td>Minimum</td>
<td>0.06</td>
<td>0.11</td>
<td>0.08</td>
</tr>
<tr>
<td>Maximum</td>
<td>0.11</td>
<td>0.18</td>
<td>0.14</td>
</tr>
<tr>
<td>Mean</td>
<td>0.07</td>
<td>0.15</td>
<td>0.11</td>
</tr>
</tbody>
</table>

Mean values are root mean variances for measurement and fitting errors. \( \epsilon \), overall error. See text for further explanation.
strains were clearly separated with the major stretch rising to a maximum of 14%. The major extension was directed below the ventricular circumference and coincided with the orientation of epicardial muscle fibers. Both epicardial twist and rigid rotation were small and consistently counterclockwise. In both cases the rotation was clearly load dependent. Since the major extension was aligned with the fiber axis, epicardial twist or shear represents a change in fiber orientation. The results therefore indicate that epicardial fibers become slightly more longitudinal during passive filling. Finally, the magnitude and orientation of the principal stretches for the midventricular posterior wall of the LV are seen to differ considerably from those on the anterior wall.

DISCUSSION

The aim of this work was to develop an experimental model that could be used to 1) study the factors that influence regional myocardial deformation during diastole and 2) assess the accuracy of ventricular wall strain distributions predicted by continuum mechanics models of the passive heart. To meet these objectives, it was necessary to make accurate measurements of regional ventricular deformation under carefully controlled conditions. An isolated arrested heart preparation was adopted to satisfy these requirements.

Although the loading conditions in this preparation were not strictly comparable with those in the heart during diastole, they were well defined and reproducible. The heart was suspended from a rigid structure that fixed the positions of the aortic and mitral valves; for each ventricular volume increment, measurements were

FIG. 4. Left ventricular (LV) pressure volume relationships. A: LV pressure vs. relative LV volume for experiment 2. Arrows, sequence of LV loading. B: mean ± SD (ascending limb only) for all experiments (n = 5).

FIG. 5. Midanterior epicardial deformation in response to left ventricular (LV) filling. Data from experiment 2 for a single load cycle. A: extensions of segments between markers vs. LV pressure. B: principal extensions vs. LV pressure. Major and minor extensions are clearly differentiated showing that strain was nonuniform. Major extension was oriented ~25° below ventricular circumference through most of the load cycle. C: principal extensions vs. change in LV volume showing an approximately linear variation. D: rigid rotation and epicardial twist vs. LV pressure. Marker centroid rotated counterclockwise (viewed from base) around aortic axis with LV filling. Twist was also counterclockwise.
made at steady state to exclude viscous and inertial effects, and the mechanical effects of coronary perfusion were also controlled.

The hearts were not supported metabolically, but standard procedures (3, 7) were used to preserve the myocardium and delay the onset of ischemic contracture. Electrical and mechanical activity ceased rapidly when arrest was induced; optimal hypothermia was maintained while the heart was mounted on the cannula system and prepared for study; perfusion time was brief; and nifedipine, a calcium antagonist, was added to the cardioplegic perfusate. Finally, after the hearts were brought to room temperature, the experimental studies were completed within 20 min; Laks et al. (14) showed that in excised dog hearts at room temperature there was no significant change in measured LV compliance until 40 min after arrest.

The stability of this preparation is reflected by the consistency of the results both within and between experiments. The pressure-volume curves are reproducible and lie within the range of data presented by other investigators (4, 8, 17). The slight hysteresis was due entirely to fluid loss. The epicardial strain curves did not vary significantly between loading cycles and were consistent with other estimates (4, 8, 17). The slight hysteresis was due with muscle fiber orientation across the LV wall. The local muscle fiber direction, whereas Fenton et al. (6) reported that the principal direction of shortening coincided with muscle fiber orientation across the LV wall. Waldman et al. (23) observed nonuniform principal strains in planes parallel to the epicardium. They found that the angle between the ventricular circumference and the principal axis of subepicardial shortening at end systole was in the range -25 to -60°, but reported that the direction of principal shortening varied less than the cardiac muscle fiber orientation across the LV wall. The reversal of the patterns of active ventricular deformation during relaxation and early diastole has not attracted much attention but appears to have been interpreted as postsystolic recoil. Walley et al. (24) claimed that during slow filling the principal directions of LV endocardial deformation are ill defined, with similar rates of change for major and minor extensions. They concluded that this suggests a symmetric expansion of the LV during late diastole and reflects the essentially isotropic material properties of the passive myocardium.

### Table 2. Mean LV volume change, rigid body rotation, and epicardial deformation data on anterior and posterior free walls

<table>
<thead>
<tr>
<th>LV Pressure, mmHg</th>
<th>ΔV, ml</th>
<th>Principal Extensions, %</th>
<th>Epicardial Rotation, deg</th>
<th>Principal Angle, deg</th>
<th>Rigid Rotation, deg</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>$\Delta_1$</td>
<td>$\Delta_2$</td>
<td>$-\Delta_1$</td>
<td></td>
</tr>
<tr>
<td><strong>Anterior wall (n = 4)</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
| 2                | 6.6±1.2| 2.6±0.8      | 1.1±1.1     | 3.6±1.7    | -0.5±0.5        | 0.9±0.5
| 5                | 13.7±3.0| 5.8±1.6      | 2.6±2.3     | 7.8±3.1    | -1.1±1.2        | 1.9±1.1
| 10               | 22.1±5.0| 9.9±1.9      | 4.5±3.1     | 12.8±3.9   | -1.8±2.0        | 3.3±1.9
| 15               | 27.1±6.1| 12.5±1.6     | 5.9±3.2     | 16.0±3.6   | -2.3±2.5        | 4.2±2.4
| 20               | 30.0±6.6| 13.9±1.2     | 7.2±3.2     | 18.0±3.3   | -2.6±2.8        | 4.8±2.8

| **Posterior wall (n = 1)** |        |             |             |            |                  |        |                      |                      |                     |
| 2                | 8.4    | 3.3         | 1.4         | 4.5        | -0.8            | -54    | 1.8                   |
| 5                | 15.0   | 7.3         | 3.2         | 9.7        | -1.7            | -57    | 3.7                   |
| 10               | 21.8   | 11.8        | 5.4         | 15.1       | -2.2            | -63    | 5.8                   |
| 15               | 25.3   | 14.7        | 6.3         | 17.9       | -2.1            | -68    | 6.7                   |
| 20               | 27.6   | 16.8        | 6.3         | 19.4       | -1.7            | -68    | 7.2                   |

Values are means ± SD for anterior wall. LV, left ventricular; ΔV, change in LV volume; $\Delta_1$, major stretches; $\Delta_2$, minor stretches; $-\Delta_1$, derived wall thinning.
The results of the present study demonstrate that the factors that determine regional epicardial deformation in the passive LV are more complex than has previously been suggested. For anterior and posterior midwall sites, epicardial strain was nonuniform with the principal extensions clearly differentiated and the major extension directed below the ventricular circumference. It is noteworthy that this direction coincides with the known orientation of epicardial muscle fibers (20). In addition there was a counterclockwise rigid rotation of the epicardium (viewed from the fixed base). These results are the inverse of those measured on the epicardium of the intact heart during systole (1, 6). However, they are not due to contraction, dynamic effects, or in vivo loading conditions. The observed patterns of passive deformation are therefore a fundamental property of ventricular architecture and the material properties of the passive myocardium and may indicate that the passive myocardium is most compliant in the fiber direction.

Although the measures of regional epicardial deformation obtained using this experimental model provide no direct information on strain in deeper myocardial layers, they can be readily compared with ventricular strain distributions predicted by continuum mechanics models of the passive heart. The observed strains are not consistent with the predictions of mathematical models in which it is assumed that the material properties of the passive myocardium are isotropic (11, 18, 21). It is apparent that the three-dimensional geometry and the material properties of the ventricular wall are important determinants of regional deformation and strain in the diastolic heart. We conclude that any realistic mathematical analysis of regional stress and strain should include these factors and be able to predict the results obtained using our experimental method.

We have developed an isolated, arrested heart preparation that enables us to characterize regional epicardial deformation in the LV under well-defined conditions. Using this method we have demonstrated that, for the anterior midwall region of the LV, epicardial deformation is nonuniform and that maximum extensions are in the direction of epicardial muscle fibers. The method provides the means to investigate questions that arise from the preliminary study. In particular, it is necessary to establish the extent to which the patterns of deformation at other sites correspond to those presented here for the anterior midwall LV epicardium, and to determine how regional ventricular deformation in the passive heart is influenced by viscous and inertial effects, by material inhomogeneity of the ventricular myocardium, and by variations in coronary perfusion pressure and flow.

APPENDIX A

Coordinate Reconstruction Equations

1) Definitions. For each camera $c = 1, 2$ (see Fig. 2), we define a right-handed cartesian system of physical coordinates with a common origin at the point of intersection of the axes of symmetry of the lenses where $X_1$ is the horizontal axis parallel to the lens plane of camera $c$; $X_2$ is the common vertical axis; $X_3$ is directed along the lens axis of camera $c$. $N_1$ and $N_2$ are the horizontal and vertical digitized screen coordinates of a point in the image field of camera $c$, where $-500 \leq N \leq 500$.

$D$ is the distance from the lens of camera $c$ to the origin.

The biplane angle $\beta$ is expressed as the difference in the angular positions of the two cameras: $\beta = \beta_2 - \beta_1$.

2) Transfer functions. The coordinates $\vec{x}_1$ and $\vec{x}_2$ of selected points on a calibration grid (positioned in the $X_3 = 0$ plane) were fitted independently as linear functions of $N_1$ and $N_2$ and these functions were fitted, in turn, as linear functions of $D$

$$\vec{x}_1 = [A_1(D - X_1)] + B_1N_1$$

$$\vec{x}_2 = [A_2(D - X_3)] + B_2N_2$$

where $A_1$, $A_2$, $B_1$, and $B_2$ are the constant coefficients of the least-square functions.

3) Coordinate transformation. The two camera coordinate systems differ only by a rotation about their common $X_1$ axis, thus

$$\begin{bmatrix}
X_1' \\
X_2' \\
X_3'
\end{bmatrix} = 
\begin{bmatrix}
\cos \beta & 0 & \sin \beta \\
0 & 1 & 0 \\
-\sin \beta & 0 & \cos \beta
\end{bmatrix}
\begin{bmatrix}
X_1 \\
X_2 \\
X_3
\end{bmatrix}
$$

If the global coordinates $X_1$, $X_2$, and $X_3$ are defined to coincide with $X_1'$, $X_2'$, $X_3'$, then, using Eqs. A1–A3

$$\begin{bmatrix}
\cos \beta - A_1N_1sin \beta \\
\sin \beta + A_2N_2cos \beta
\end{bmatrix}X_1 + (\sin \beta + A_2N_2cos \beta)X_2 = (D(A_1 + B_1)N_1$$

for $c = 1, 2$

Eq. A4 represents a system of two linear equations in $X_1$ and $X_2$. $X_3$ is common with $X_3'$ and $X_3''$ which, using $X_1'$ and $X_2'$, are obtained directly from Eq. A2. To account for the possibility of a small numerical difference between $X_3'$ and $X_3''$, $X_3$ is defined as the average

$$X_3 = \frac{1}{2}(X_3' + X_3'')$$

APPENDIX B

In the Lagrangian description of deformation, the position vector $\vec{x}$ of a material point in the deformed state is a function of the corresponding vector $\vec{x}$ in the undeformed state

$$\vec{x} = \hat{\vec{x}}(\vec{x})$$

A tensor of deformation gradients $F$ defines the transformation between two arbitrarily small segments in the deformed $d\vec{x}$ and reference $d\vec{x}$ states

$$d\vec{x} = F d\vec{x}$$

If the position vectors, referred to local epicardial coordinates of the three epicardial markers, are denoted by $\vec{x}^b$, $\vec{x}^a$, and $\vec{x}^c$ then any two segments, $(\vec{x}^b - \vec{x}^c)$ and $(\vec{x}^a - \vec{x}^c)$ can be used as estimates of $d\vec{x}$ in the unloaded heart and $d\vec{x}$ in the loaded heart

$$(\vec{x}^b - \vec{x}^c) = [F](\vec{x}^b - \vec{x}^c)$$

$$(\vec{x}^a - \vec{x}^c) = [F](\vec{x}^a - \vec{x}^c)$$

These two sets of $2 \times 2$ linear equations can be rearranged in terms of the unknown components of $F$

$$\begin{bmatrix}
(x^b_1 - x^c_1) \\
(x^a_1 - x^c_1)
\end{bmatrix}
\begin{bmatrix}
F_{11} & F_{12} \\
F_{21} & F_{22}
\end{bmatrix}
\begin{bmatrix}
(x^b_2 - x^c_2) \\
(x^a_2 - x^c_2)
\end{bmatrix}
$$

$F$ can be decomposed into an orthogonal rotation vector $\vec{R}$ and a symmetric right stretch tensor $U$

$$F = \vec{R}U$$

(B5)
**LEFT VENTRICULAR EPICARDIAL DEFORMATION**

\[ \mathbf{R} \text{ rotates a segment (clockwise) through an angle } \alpha \]

\[ \mathbf{R} = \begin{bmatrix} \cos \alpha & \sin \alpha \\ -\sin \alpha & \cos \alpha \end{bmatrix} \quad (B6) \]

Making use of the fact that \( \mathbf{R} \) is orthogonal (i.e., the transpose of \( \mathbf{R} \), \( \mathbf{R}^T = \mathbf{R}^{-1} \))

\[ \mathbf{U} = \mathbf{R}^{-1} \mathbf{F} = \mathbf{R}^T \mathbf{F} = \begin{bmatrix} \cos \alpha & -\sin \alpha \\ \sin \alpha & \cos \alpha \end{bmatrix} \begin{bmatrix} F_{11} \\ F_{21} \\ F_{12} \\ F_{22} \end{bmatrix} \quad (B7) \]

and that \( \mathbf{U} \) is symmetric (i.e., \( U_{21} = U_{12} \))

\[ (F_{12} - F_{21}) \cos \alpha = (F_{11} + F_{12}) \sin \alpha \]

gives

\[ \alpha = \tan^{-1} \left( \frac{F_{12} - F_{21}}{F_{11} + F_{22}} \right) \quad (B8) \]

With \( \mathbf{R} \) now known, \( \mathbf{U} \) can be found from Eq. B7. The eigenvalues \( \lambda_{1} \) and \( \lambda_{II} \) associated eigenvectors \( \mathbf{e}_{1}, \mathbf{e}_{II} \) of \( \mathbf{U} \) are related by

\[ U \mathbf{e}_{i} = \lambda_{i} \mathbf{e}_{i} \quad i = 1, II \quad (B9) \]

or the determinant \( |U - \lambda I| = 0 \), where \( I \) is the \( 2 \times 2 \) identity matrix. This yields the quadratic characteristic equation in \( \lambda \)

\[ (U_{11} - \lambda_{1})(U_{22} - \lambda_{II}) - U_{21}U_{12} = 0 \quad (B10) \]

The components of the orthogonal eigenvectors are obtained from Eq. B9 and the principal angle \( \Phi \) defines as the angle between \( \mathbf{e}_{1} \) and the unit vector \( \mathbf{i} \) of the local circumferential axis such that \(-90 < \Phi \leq 90\) (see Fig. 2)

\[ \Phi = \cos^{-1}(e_{1} \cdot i) \]

The rigid rotation \( \Theta \) is defined as the angle between the global position vectors of the marker centroid in the deformed and undeformed states.

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