Surface strains in the anterior leaflet of the functioning mitral valve

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Surface Strains in the Anterior Leaflet of the Functioning Mitral Valve

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Abstract—The mitral valve (MV) is a complex anatomical structure whose function involves a delicate force balance and synchronized function of each of its components. Elucidation of the role of each component and their interactions is critical to improving our understanding of MV function, and to form the basis for rational surgical repair. In the present study, we present the first known detailed study of the surface strains in the anterior leaflet in the functioning MV. The three-dimensional spatial positions of markers placed in the central region of the MV anterior leaflet in a left ventricle-simulating flow loop over the cardiac cycle were determined. The resulting two-dimensional in-surface strain tensor was computed from the marker positions using a C^0 Lagrangian quadratic finite element. Results demonstrated that during valve closure the anterior leaflet experienced large, anisotropic strains with peak stretch rates of 500%–1000%/s. This rapid stretching was followed by a plateau phase characterized by relatively constant strain state. We hypothesized that the presence of this plateau phase was a result of full straightening of the leaflet collagen fibers upon valve closure. This hypothesis suggests that the MV collagen fibers are designed to allow leaflet coaptation followed by a dramatic increase in stiffness to prevent further leaflet deformation, which would lead to valvular regurgitation. These studies represent a first step in improving our understanding of normal MV function and to help establish the principles for repair and replacement. © 2002 Biomedical Engineering Society. [DOI: 10.1114/1.1529194]

Keywords—Mitral valve, Heart valves, Valve function, Dynamic imaging, Soft tissue, Biomechanics.

INTRODUCTION

The mitral valve (MV) is a complex anatomical structure consisting of two leaflets, an annulus, chordae tendinae, and papillary muscles.^{4,16,25,33,36,42} The normal function of the mitral complex involves a delicate force balance and synchronized function of each of its

components.^{12,13} Pathological alterations affecting any one component of the complex MV structure can lead to altered MV function.²⁷ One example of an important pathological process is ischemic mitral regurgitation (IMR), caused by partial or complete obstruction of one or more coronary arteries.⁷ The incidence of IMR in the United States is estimated to be 1.2–2.1 million patients.¹⁴ Coronary artery occlusion produces IMR by altering left ventricular geometry, wall motion, the deformation of one or both papillary muscles, and possibly the septal and mural (posterior) parts of the MV annulus.

When the MV is too severely diseased for repair techniques to be effective, it is generally replaced with a mechanical or bioprosthetic valve.¹ Although over 50 specific types of replacement valves currently exist, none can be considered an ideal substitute. Mechanical prosthetic valve disadvantages include suboptimal hemodynamics, thromboembolism, and blood cell damage, while stented bioprostheses possess limited long term durability in the mitral position.⁴⁶ The development of better substitutes is an ongoing and costly process. The most ideal replacement should simulate the natural mitral complex as closely as possible.

Clearly, accurate diagnosis and treatment of MV disease, along with the development of improved surgical strategies/techniques, require a complete understanding of normal MV dynamics. A number of investigators have undertaken the task of studying the MV dynamics and left ventricular fluid mechanics.^{3,11,32,34,37,41} Due to the complexity of the MV apparatus, it is difficult to analytically or theoretically determine the functional role and importance of each individual component of the mitral complex. The most advanced efforts along these lines are the numerical simulations of Kunzelman.^{20,22,23} However, these simulations are currently difficult to validate *in vivo* with available imaging technologies. Further, *in vivo* studies suffer from lack of precise control over many variables, which may influence the final outcome and its

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FIGURE 1. (a) A dissected MV anterior leaflet showing the 16 marker array in the central region between the annulus and the coaptation line, along with the corresponding radial and circumferential axial directions. (b) SALS results for the leaflet shown in (a) along with the marker positions shown as white dots, with an enlarged view shown in (c). The SALS data indicated that marker array was aligned to the local collagen fiber preferred direction, and was located in a region where the collagen fibers are highly aligned.

interpretation. *In vitro* approaches allow each component to be precisely controlled for accurate and systematic studies that improve our fundamental understanding of the MV complex.

In the current study, an *in vitro* approach was developed to quantify both the in-surface strains of the anterior MV leaflet. We focused on a $\sim 4\text{ mm} \times \sim 4\text{ mm}$ region of the center portion of the anterior leaflet by tracking the three-dimensional (3D) motions of 16 surface markers. From the resulting marker motions the complete in-surface strain tensor was computed, along with the corresponding strain rate information. Relations of the principal strain directions to the underlying collagen fiber architecture were quantified, and the relation to other relevant structural and functional aspects discussed.

METHODS

Valve Preparation

Fresh porcine hearts were obtained from a local abattoir, and the mitral valves removed while carefully pre-

serving chordae tendinae and papillary muscles. The valves were sutured to a planar D-shaped annulus, and metal holders were attached to the papillary muscle remnants. Graphite markers were crushed into tiny particles approximately $200\text{ }\mu\text{m}$ in diameter, and 16 markers were glued in a 4×4 array to the basal surface of the anterior leaflet about 30%–70% between annulus and coaptation line [Figs. 1(a) and 2(b)].

Preliminary tests were run to refine marker size, placement, and application, with the size used providing good contrast and appeared 2–3 pixels wide for the camera resolution (see *Data and Image Acquisition*). The valve was mounted into the left heart model and papillary muscles were adjusted to normal position. After testing (see later), the anterior leaflet was removed and the collagen fiber architecture quantified for each leaflet using small angle light scattering.⁴⁰ The measured fiber directions were then compared to the placement of the markers and the local principal stretch directions (see later). A total of five valves were successfully tested, as described in the following.

FIGURE 2. (a) A schematic of the left heart simulator and flow loop. Data acquisition system recorded the transmitral pressure and flow rate. Two angled placed high speed cameras were positioned in front of mitral valve to record the 3D marker array spatial positions. Image and transmitral pressure were synchronized with trigger signal from pulse generator. (b) The pictures from camera A and B were cropped and combined in frame grabber to form one frame of image.

Physiologic Flow Loop, Data Acquisition, and Video System

A schematic of the physiological flow loop is shown in Fig. 2(a), and has been described in detail previously.¹⁸ Briefly, the flow loop comprised a Thomas air compressor and pulse programmer which controls a rubber bladder to drive flow, solid left atrium and left ventricle chambers, a compliance chamber, resistance clamp, atrial reservoir, a differential pressure transducer (model DP9-40, Validyne Engineering Corporation, CA) and an electromagnetic flow probe (model FM501D, Carolina Medical, King, NC). The loop was filled with physiologic saline as the working fluid.

The pulse programmer, which sends signals to solenoids A, B, and C is controlled by a microcomputer. The appropriate settings were adjusted in order for the loop to simulate cardiac pressure and flow waveforms. Total cycle time was set at 860 ms, corresponding to a heart rate of 70 bpm. Cardiac output and peak LV pressure (which was always maintained around 120 mm Hg) were set by adjusting the resistance clamp and the input pressure valve of compressed air. A LABVIEW program was used to acquire flow and pressure waveforms.

Two Kodak EktaPro model 1000 cameras (Eastman-Kodak, San Diego, CA) were positioned on tripods approximately 0.5 m from the valve and 30° relative to each other [Fig. 2(a)]. Both cameras were centered on the mitral valve, and light and lens were adjusted to achieve maximum contrast between markers and leaflet. Both cameras were connected to a high speed Kodak image processor, with images recorded simultaneously from both cameras at 250 frames per second [Fig. 2(b)].

3D Marker Coordinate Calibration and Reconstruction

After each valve was tested, the flow loop was disassembled. Following our established calibration methods,¹⁷ a cube 10 mm on edge was positioned within the volume occupied by the valve, and positioned so that both cameras could see seven corners. Images of the cube from both cameras were then acquired, the corner positions digitized and used for direct linear transform (DLT) calibration (see later).

Details of the marker reconstruction techniques for dynamic heart valve leaflet motion have been recently presented.¹⁷ Briefly, each image set was imported into Sigma Scan Pro (Jandel Scientific, San Rafael, CA), and contrast enhanced, then the location of each marker for each view was recorded as the position of the centroid. Corresponding marker locations in each view were identified, and a calibrated standard DLT method^{17,29} was used to convert these pixel locations into 3D spatial coordinates. The DLT parameters were obtained using calibration cube vertices as the material points with known spatial positions. The reference frame used for all subsequent strain calculations (see *Surface Strain Computations*) was taken as the first frame where all 16 markers were visible in both camera views during valve closing.

Surface Strain Computations

To compute the strain field within the marker array, we utilized an in-surface coordinate system [u - v - n , Fig. 3(a)]. Although based on the tangent plane coordinate

FIGURE 3. (a) A plot of the fitted quadratic C^0 Lagrangian finite element along with the markers (dots), along with the local u - v - n and r - s - n axes. (b) The fitted surface later in the cardiac cycle showing the major principal stretch, with gray scale indicating magnitude and vectors local direction. Here, we observed that the principal directions were always aligned to the local u - v coordinates, with generally small spatial variations in magnitude.

system developed earlier,³⁹ in the current application the u - v coordinates are projected onto the deformed surface and thus form a convective coordinate system (i.e., one that deforms with the surface). The origin of the u - v - n system was located at the center of the marker array, with corresponding unit vectors $\hat{e}_u, \hat{e}_v, \hat{e}_n$. The coordinate system was defined for the current study with $\hat{e}_u$ parallel to markers 1 and 4. The surface normal $\hat{e}_n$ was computed using $\hat{e}_n = \hat{e}_v \times \hat{e}_{m3-m15}$, where e_{m3-m15} is the unit vector parallel to markers 3 and 15, and $\hat{e}_v = \hat{e}_n \times \hat{e}_u$. Each marker x - y - z coordinate triplet for each frame was subsequently translated and rotated into the reference frame u - v - n coordinate system.

To compute the strain field within the leaflet region delimited by the markers, we used a finite element-based surface interpolation, modified from our previously described methods.⁴³ In this approach, the position of any point on in reference state is given by $R^0(u, v) = u\hat{e}_u + v\hat{e}_v + n\hat{e}_n$, where n represents the normal component. To fit the marker array, a single nine-node C^0 -continuity quadratic Lagrangian element² was used, using the isoparametric coordinates r - s , with $-1 \leq r, s \leq 1$. Since only one element was required, up to quadratic variation of the nodal variable (i.e., displacement) was allowed. In this approach, the u - v and r - s axes are coincident [Fig. 3(a)] and transformed from the u - v to the r - s systems by invertible linear functions (see the Appendix). The reference surface fit yields the initial shape of the region of the anterior leaflet defined by the 16 marker array. The metric tensor in the reference configuration, $\mathbf{g}^0$, was computed from R^0 using standard formulas (see the Appendix).

To determine the strains for each frame f and component i ($= 1, 3$) the displacements for each marker d_i^f were first computed as the difference between the reference and deformed spatial marker positions. Note that in the current approach the displacement field for each axial

component was separately fit. Thus, the position vector for frame f of any point on the deformed surface is given by $R^f(u, v) = d_1^f \hat{e}_u + d_2^f \hat{e}_v + d_3^f \hat{e}_n + R^0(u, v)$, where $d_i^f(u, v)$ is the fitted displacement field for the axial component i for the current frame f .

From R^f , the components of the metric tensor $\mathbf{g}^f$ in the deformed configuration were computed, and the resulting Almansi (i.e., Eulerian) finite strain tensor for each frame determined using $\mathbf{e} = \frac{1}{2}(\mathbf{g}^f - \mathbf{g}^0)$.⁹ This simple formulation for strain computation resulted from the use of a convective, in-surface coordinate system. The principal values of $\mathbf{e}$ were expressed as principal stretches λ_1 and λ_2 , with corresponding principal angle θ_p referred to the u axis in the reference state. We computed the shear as the angle change α between two lines originally orthogonal in the reference state. The areal stretch, representing the net change in leaflet area, was computed using $\Delta = \lambda_1 \lambda_2$. Once the principal stretches λ_1 and λ_2 were determined for each frame, the corresponding stretch rates $d(\lambda_1)/dt$, $d(\lambda_2)/dt$, $d(\Delta)/dt$ were computed using a three-point numerical derivative algorithm³⁵ and expressed as percent per second. Details of the surface fitting and strain calculations are given in the Appendix.

Surface Strain Accuracy

To determine the net resolution of the system, we prepared a precision phantom consisting of a 4×4 1.5 mm spaced grid of 200 μm diameter markers, similar to the approach used in Ref. 17. The marker array was placed in the flow loop, imaged, and reconstructed using the same process as used for the valves. Four other marker arrays were also generated, with a strain gradient of 10% applied to their original positions to a maximum

FIGURE 4. r^2 values for each displacement component d_i^f over the cardiac cycle, demonstrating a consistently high quality fit for all three components.

of 50% strain. The spatial positions of the markers from both sets were analyzed as described earlier to assess the net strain resolution.

RESULTS

Reference Surface Fitting and Marker Array Placement

The C^0 quadratic Lagrangian finite element surface fitting technique was able to fit the spatial positions of the 16 marker array very well, with $r^2 \geq 0.97$ and a mean error in the normal distance of 0.05 mm (Fig. 3). It was noted that the surface height varied by at most 0.4 mm during the cardiac cycle. Thus, this region of the anterior leaflet is fairly flat, although this is probably due to the relatively small size of the marker array. The collagen fiber architecture data indicated that the marker arrays were consistently placed in a region where the collagen fibers were highly aligned in the circumferential direction (Fig. 1). Note too that the marker array local u - v axes were also typically aligned to within $\pm 5^\circ$ of the local collagen fiber preferred direction [Fig. 1(c)].

Surface Strain Accuracy

The measured inter-marker distances were 4.499 ± 0.037 mm in the horizontal direction and 4.600 ± 0.007 mm in the vertical direction, which compared favorably to the of the actual distance of 4.500 mm. Our net spatial measurement accuracy was thus ± 0.1 mm. The resulting error in determining the principal stretches was 0.01 ± 0.005 (mean $\pm$ sd) and areal stretch 0.026 ± 0.01 . These spatial and computed strain accuracies were comparable to previous studies.^{17,43}

FIGURE 5. (a) Representative results of the principal stretches λ_1 and λ_2 over the cardiac cycle. Overall, the anterior leaflet experienced substantial and anisotropic strains during closure. Once valve closure was completed, the principal stretches maintained at nearly constant values, where they followed a similar but reversed path compared to the closing behavior. (b) Corresponding principal stretch rates which indicated peak rates of 500%/s–1000%/s. Stretch rates fell to nearly zero during closure, with a nearly symmetrical behavior during valve opening.

Strain Field Results

The strain field over the region delimited by the markers was relatively homogenous. For example, the uniformity of values and directions of the major principal stretch λ_1 were generally consistent over the cardiac cycle [Fig. 3(b)]. Note too that the major principal stretch direction was aligned to the v (radial) axis, and the minor principal stretch aligned to the u (circumferential) axis (Fig. 3). The surface fitting technique was also able to fit all three components of $d_i^f(r, s)$ well, with $r^2 \geq 0.97$ throughout the cardiac cycle (Fig. 4).

The overall behavior of the principal stretches was very consistent for all valves. The typical characteristics for the principal stretches are shown in Fig. 5(a). Here, we observed a very rapid increase in both principal stretches, with substantial differences in principal values.

FIGURE 6. (a) Representative results of the areal stretch over the cardiac cycle. The behavior of the areal followed that of the principal stretches shown in Fig. 5, including very high peak strain rates of $>1500\%/s$ as shown in (b).

Thus, the anterior leaflet of the MV experiences substantial and anisotropic strains during closure. Once valve closure was completed, the principal stretches maintained

nearly constant values until valve reopening commenced, then followed a similar but reversed path during the opening phase. Throughout the cardiac cycle values for α were less than 5° , indicating that the MV anterior leaflet experienced low shear strains.

Stretch rates fell to nearly zero during closure, with a nearly symmetrical behavior during valve opening [Fig. 5(b)]. Areal strain results were consistent with the principal stretch behavior, and indicated a nearly 75% increase in leaflet area within ~ 0.1 s [Fig. 6(a)]. Areal strain rates were also comparably high, reaching peaks of $1500\%/s$ to $2000\%/s$ [Fig. 6(b)]. Again, we observed a constant strain plateau during valve closure corresponding negligible areal strain rates.

Peak stretch values were fairly consistent between valves, with the mean values of λ_1 0.2 greater than λ_2 and statistically significant ($p < 0.05$) [Fig. 7(a)]. The mean areal strain was ~ 1.47 for all valves, indicating an average area increase of 47% from the unloaded state at a transvalvular pressure of ~ 116 mm Hg. Peak stretch rates were also consistent between valves. Here the average peak $d(\lambda_1)/dt \approx 750\%/s$ and $\sim 900\%/s$ during closing and opening, respectively. In contrast, the average peak $d(\lambda_2)/dt \approx 300\%/s$ during closing and $\approx 400\%/s$ during opening [Fig. 7(b)]. Note too that the average peak $d(\lambda_2)/dt$ was statistically smaller than the peak average peak $d(\lambda_1)/dt$ [Fig. 7(b)] for both principal axes. The average peak $d(\Delta)/dt \approx 1000\%/s$ to $1300\%/s$. It is interesting that the opening phase consistently produced slightly higher strain rates compared to the closing phase.

When the transmitral pressure against the areal strain during valve closure were compared, we observed a non-linear relationship that bore a close resemblance to the stress-strain curve for collagenous tissues (Fig. 8). In particular, we noted that small transmitral pressures were required to initially increase the anterior leaflet area, up

FIGURE 7. (a) Peak stretch and areal stretch values, showing that large and anisotropic stretch values, as evidenced by statistically different (* indicates $p < 0.05$) between the major and minor principal stretches. The mean areal strain was ~ 1.47 for all valves, indicating an average area increase of 47% from the unloaded state at a transvalvular pressure of ~ 116 mm Hg. (b) Peak stretch rates were also consistent between valves, with the average peak $d(\lambda_2)/dt$ statistically smaller than the peak average peak $d(\lambda_1)/dt$ for both stretch axes. It is interesting to note that opening phase consistently produced slightly higher strain rates compared to the closing phase.

FIGURE 8. Transmitral pressure plotted against the areal strain, which is analogous to a stress-strain curve for the leaflet, and demonstrates the classic nonlinear response.

to $\Delta \approx 1.5$. This was followed by a rapid increase in transmitral pressure to increase the leaflet area in the final closure phase from 50% to $\sim 75\%$ (Fig. 8).

DISCUSSION

In this study, we present the first known detailed study of the surface strains in the anterior leaflet in the functioning mitral valve. As important as understanding the dynamic loading behavior of heart valves, few studies have been performed due to technical difficulties. Previous studies by Thubrikar^{44,45} estimated the dynamic strains for the aortic valve *in vivo*, but used one-dimensional strain measurement techniques. More recent studies have developed complete in-surface approaches,^{17,43} but focus on the aortic valve. Thus, the current study represents an important step in improving our understanding of normal MV function, and to help establish parameters for surgical repair and functional tissue engineering.⁶

To summarize, our key findings were:

- (1) The anterior leaflet experienced large, anisotropic stretches during closure.
- (2) Once the valve is closed, further leaflet deformation ceases.
- (3) The closing deformation behavior is essentially symmetrically reversed during valve opening.
- (4) The region of the anterior leaflet studied experienced relatively little shear ($\alpha < 5^\circ$) throughout the cardiac cycle.
- (5) Peak stretch rates during the closing and opening phases were very high, reaching values 500%–1000%/s (Figs. 5 and 6).

An additional note is warranted for point (3) earlier. While we have observed very similar deformation behav-

iors during opening and closing, there were minor differences between the two phases. Presently we do not know the exact basis for these differences, but are likely related to the slightly different local hemodynamic conditions and overall leaflet shape that occur during the two phases. We have observed similar differences in bio-prosthetic valve shape during opening and closing.¹⁷ Taken as whole, these results clearly demonstrate the complex, highly dynamic nature of MV leaflet function.

Functional Structure of the MV Anterior Leaflet

Our observations of the dynamic strain behavior also have implications to MV function. First, at the macro level we noted that the strain field was relatively homogeneous over the region delimited by the markers and that this area of the leaflet remained fairly flat [Fig. 3(b)]. Large curvature changes were observed in the images (not measured) to occur only at the coaptation boundaries. These results suggest that the central region of MV anterior leaflet behaves like a membrane stretched within a plane tangent to its surface, which also undergoes a hinge-like motion at valve annulus.

Other observations can be correlated with known or hypothesized microstructural events in valvular tissues. In particular, we noted that the transmitral pressure versus areal strain graphs demonstrated a marked increase in slope at transmitral pressures exceeding ~ 20 mmHg (Fig. 8). This sudden increase in leaflet stiffness occurred at the same time point when the areal strain plateaus [Fig. 6(a)], and is most likely explained by straightening and subsequent locking of leaflet collagen fibers. Specifically, the leaflet is initially deformed into the closed position in the “toe” region of the stress-strain curve, where the collagen fibers are most highly undulated.^{10,15} As the MV fully closes and is subjected to higher trans-valvular pressures, the leaflet is stretched to the point where the collagen fiber undulations are removed. This results in substantial stiffening of the tissue, to the point where further leaflet deformations are prevented. This hypothesis is also supported by uniaxial^{19,21} and biaxial³⁰ biomechanical studies of the MV leaflets, which demonstrated the classic soft tissue stress-strain relationship. Taken as whole, our results indicate that the collagen fiber network of the valve is finely tuned to allow for sufficient initial compliance to allow for adequate leaflet coaptation, followed by a dramatic stiffening to prevent further deformation, which would lead to regurgitation.

Influence of Viscoelastic Leaflet Behavior

Like all collagenous tissues, the MV anterior leaflet has been shown to be viscoelastic.²⁸ However, the constant deformation state during full closure (Figs. 5 and 6) suggests that negligible stress relaxation occurred while the valve was in the fully closed position. If stress re-

laxation occurred, then we would see increasing strain during the full closure period. This finding is not surprising since the closing time is very short, only ~ 0.3 s (Fig. 5). Thus, any viscoelastic effects would have short relaxation time constants in order to manifest themselves when the valve is closed, which was not observed in the present study.

However, this finding does not rule out strain rate effects during closure or opening. Indeed, strain-rate sensitivity has been demonstrated for heart valve biomaterials under uniaxial²⁶ and biaxial³⁸ loading states. In these studies, peak stresses were increased by a factor of 2 when loading times were reduced from 1.0 to 0.1 s. Given the high strain rates observed in the current study [Figs. 5(b) and 7(b)], subsequent analyses of MV stresses will need to incorporate strain-rate effects.

Influence of Local Collagen Structure

We observed that the region of the valve experienced very low shear, indicating that the principal directions were closely aligned to the local collagen fiber architecture (Fig. 1). This finding is at first not surprising given the highly aligned local collagen fiber structure (Fig. 1), and is very similar to our *in vitro* findings for the porcine aortic valve under biaxial loading.⁵ However, given the fact that the MV has complex attachments to the surrounding cardiac structures (e.g., valve annulus, chordae tendinae), there is no *a priori* reason to assume that this would be the case in the functioning valve. Our current findings suggest that the central portion of the anterior leaflet behaves as a membrane-like structure, with the minor principal stretch aligned to the local collagen fiber direction.

Limitations and Recommendations

The primary limitation of the current study was the focus on only a single region of the MV anterior leaflet. Structural heterogeneity and anisotropy of the MV leaflets have been documented,^{19,24} and suggest significant regional variations in stress-strain behavior. Clearly, additional studies will have to be performed to cover as much of the leaflet surfaces as possible. Further, alterations in MV function under varying hemodynamic and papillary muscle positions (to simulate different pathological conditions and surgical repair approaches) are necessary to facilitate translation of the current *in vitro* results to the actual functioning valve and to guide surgical repair techniques. Finally, saline was used as the working fluid, which has a lower viscosity than blood (or a glycerin/water fluid analog). While it is possible that this may affect the leaflet deformation at maximum flow, it is unlikely to affect the opening/closing dynamics significantly.

Summary

In the present study, we present the first known detailed study of the surface strains in the anterior leaflet in the functioning MV. Results demonstrated that during valve closure the anterior leaflet experienced large anisotropic strains, followed by a plateau phase characterized by relatively constant strain state. We hypothesized that the presence of this plateau phase was a result of full straightening of the leaflet collagen fibers upon valve closure. This hypothesis suggests that the MV collagen fibers are designed to allow leaflet coaptation followed by a dramatic increase in stiffness to prevent further leaflet deformation, which would lead to valvular regurgitation. The present study suggests that the anterior leaflet does not function as a simple coapting membrane structure; but rather deforms in a complex, finely tuned manner. This information will be useful in furthering our understanding of MV function for both surgical repair and functional tissue engineering.⁶

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APPENDIX— C^0 SURFACE FITTING AND STRAIN COMPUTATIONS

Reference Surface Fitting

The general approach to surface fitting was based on earlier work,⁴³ and utilizes finite element functions for surface interpolation. First, we define the position vector of the i th marker in the u - v - n coordinate system point in the reference state by $R_i^0(u, v) = u\hat{e}_u + v\hat{e}_v + n\hat{e}_n$. Note that the r - s axes are coincident with the u - v axes [Fig. 3(a)], and are defined using the following invertible linear coordinate transformation:

$$r = \frac{2(u - u_{\min})}{(u_{\max} - u_{\min})} - 1, \quad s = \frac{2(v - v_{\min})}{(v_{\max} - v_{\min})} - 1, \quad (\text{A1})$$

where the subscripts min and max refer to the maximum and minimum values of the u - v coordinates for the marker array, respectively.

The local surface of MV anterior leaflet, in the region delimited by the marker array, was approximated by a nine-node C^0 quadratic Lagrangian element.² In this formulation only the n coordinate is fit, so that for an arbitrary point on the reference surface

$$n(u, v) = \sum_{j=1}^9 h_j(r, s) a_j, \quad (\text{A2})$$

where h_j are the element shape functions given in Ref. 2 and a_j the optimal nodal values. 3D surface fitting is thus reduced to a one-dimensional determination of the optimal values of a_j that minimize the distances between the fitted surface and the marker positions. This is accomplished using a standard least squares formulation,³¹ which in matrix form is represented as the following sum over all 16 markers:

$$\sum_{i=1}^{16} [A^T A] a = \sum_{i=1}^{16} [A^T b], \quad (\text{A3})$$

where A is the column vector of the values of the shape functions h_i at each marker location, a is the column matrix of optimal nodal values that minimize the error in n , and b is the column vector of experimentally determined values of n for the current marker i . The values for a in Eq. (A3) were determined using LU decomposition.³⁵ The end result of this process is the fitted surface for the reference configuration $R^0(u, v) = u\hat{e}_u + v\hat{e}_v + (\sum_{j=1}^9 h_j(r, s)a_j)\hat{e}_n$. Note that r - s used to determine the value of the shape functions h_j are computed from the u - v coordinates using Eq. (A2).

The partial derivatives of R^0 are computed using $\partial R^0 / \partial u = 1 + \sum_{j=1}^9 \partial h_j(r, s) / \partial u a_j$, $\partial R^0 / \partial v = 1 + \sum_{j=1}^9 \partial h_j(r, s) / \partial v a_j$, with the spatial derivatives of the shape functions determined using standard methods.² The components of the metric tensor in the reference configuration $\mathbf{g}^0$ are computed using⁸

$$\begin{aligned} g_{11}^0 &= \left(\frac{\partial R_u^0}{\partial u} \right)^2 + \left(\frac{\partial R_v^0}{\partial u} \right)^2 + \left(\frac{\partial R_n^0}{\partial u} \right)^2, \\ g_{12}^0 &= \frac{\partial R_u^0}{\partial u} \frac{\partial R_u^0}{\partial v} + \frac{\partial R_v^0}{\partial u} \frac{\partial R_v^0}{\partial v} + \frac{\partial R_n^0}{\partial u} \frac{\partial R_n^0}{\partial v}, \\ g_{22}^0 &= \left(\frac{\partial R_u^0}{\partial v} \right)^2 + \left(\frac{\partial R_v^0}{\partial v} \right)^2 + \left(\frac{\partial R_n^0}{\partial v} \right)^2, \end{aligned} \quad (\text{A4})$$

where R_u^0 , R_v^0 , and R_n^0 are the u , v , and n components of R^0 , respectively.

Deformed Surface Fitting

As stated in the Methods, the position vector for frame f of any point on the deformed surface is given by $R^f(u, v) = d_1^f \hat{e}_u + d_2^f \hat{e}_v + d_3^f \hat{e}_n + R^0(u, v)$, where $d_i^f(u, v)$ is the fitted displacement field for the axial component i ($1 = u, 2 = v, 3 = n$) for the current frame f . In the current approach for each frame, each displacement component is separately fitted:

$$d_i^f(u, v) = \sum_{j=1}^9 h_j(r, s) a_j, \quad (\text{A5})$$

where the a_j now represent the optimal nodal values that minimize the differences between fitted and measured marker displacements. Computation of the a_j for the displacement field and components of $\mathbf{g}^f$ follow those for the reference surface described above.

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