Relating myocardial laminar architecture to shear strain and muscle fiber orientation

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Received 11 August 2000; accepted in final form 18 December 2000

Arts, T., K. D. Costa, J. W. Covell, and A. D. McCulloch. Relating myocardial laminar architecture to shear strain and muscle fiber orientation. Am J Physiol Heart Circ Physiol 280: H2222–H2229, 2001.—Cardiac myofibers are organized into laminar sheets about four cells thick. Recently, it has been suggested that these layers coincide with the plane of maximum shear during systole. In general, there are two such planes, which are oriented at $\pm 45^\circ$ to the main principal strain axes. These planes do not necessarily contain the fiber axis. In the present study, we explicitly added the constraint that the sheet planes should also contain the muscle fiber axis. In a mathematical analysis of previously measured three-dimensional transmural systolic strain distributions in six dogs, we computed the planes of maximum shear, adding the latter constraint by using the also-measured muscle fiber axis. Generally, for such planes two solutions were found, suggesting that two populations of sheet orientation may exist. The angles at which the predicted sheets intersected transmural tissue slices, cut along left ventricular short- or long-axis planes, were strikingly similar to experimentally measured values. In conclusion, sheets coincide with planes of maximum systolic shear subject to the constraint that the muscle fiber axis is contained in this plane. Sheet orientation is not a unique function of the transmural location but occurs in two distinct populations.

locally myocytes are oriented approximately parallel, thus defining the local mean fiber direction. Within the left ventricular (LV) wall, fiber axes follow helical pathways (11, 20) that have a right-handed pitch in the subendocardium, a left-handed pitch near the epicardium, and follow a circumferential path near the midwall. This structure has been described quantitatively by pitch angle as a continuous function of depth in the left and right ventricular wall (19).

Analysis by mathematical models of cardiac mechanics have revealed that the fiber structure of the heart is generally consistent with a uniform distribution of strain and stress in the fiber direction during systole and diastole (2, 3, 8). This uniformity has been confirmed by experimental measurements of regional myocardial strain in animals (2, 6, 9, 16, 17, 21) despite significant transmural gradients of strain in other directions. Applying the hypothesis that fiber orientation is optimized to achieve uniformity of fiber strain during systole, the distribution of fiber direction in a thick-walled ellipsoidal model of LV mechanics has been realistically predicted (18).

Systolic myofiber work is largely determined by sarcomere length at the beginning of contraction. At this point, the passive elastic extracellular matrix forming the skeleton of the myocardium comes into focus. After diastolic filling, fiber direction and sarcomere length depend on the way the myocytes are incorporated in this matrix. Consequently, cardiac pump work is partly regulated by diastolic filling (22). The collagen matrix in the myocardial tissue (4, 10, 14, 15) is a major determinant of wall stiffness. In the ventricular walls, the collagen matrix organizes myocytes into laminar sheets about four cells thick (12). It has been suggested that this layered architecture facilitates shearing and thickening deformations during systole, thus enhancing ventricular ejection (5, 13) and filling.

In the present study, we focused on the mechanical determinants and consequences of regional sheet orientation. Because the wall shortens in plane and thickens transmurally during systole, large shear strains must occur. Layers of perimysial connective tissue, separating the myofiber sheets, could be effective in permitting large shear strains without large shear stresses. This mechanism would operate optimally if the layers were directed along the planes of maximum shear. There is, however, an important constraint to sheet orientation. Myofibers are contained within the sheets. Therefore, we tested the hypothesis that sheets are oriented along the planes of maximum shear subject to the constraint that they also contain the myofiber axis.

In a previous experimental study (5), the three-dimensional (3-D) strain tensor was measured by following the motion of radiopaque beads implanted through...
the thickness of the LV wall. Sheet orientation was predicted from measured systolic strains and fiber direction by applying the above-mentioned hypothesis and compared with histologically measured sheet orientations from the same experimental study.

MODEL OF SHEET ORIENTATION

The hypothesis used was that sheets are aligned with the direction of maximum shear strain, with the constraint that the sheets are in parallel with the fibers. In this section, it is shown how this hypothesis was used to predict sheet orientation for a given fiber direction and deformation tensor.

Interlaminar shear. Deformation of the cardiac tissue is expressed by Lagrangian strain $E_{\text{exp}}$, which is experimentally determined in the local wall coordinate base $[e_r, e_\theta, e_z]$, with $e_r$, $e_\theta$, and $e_z$ being unit vectors perpendicular to the wall (radial, $r$) and circumferentially ($\theta$) and longitudinally ($l$) directed, respectively (Fig. 1, left). Locally, the unit vector $e_l$ defines the fiber direction. According to our hypothesis on fiber sheet structure, the fibers are parallel to the plane of the sheets. So the sheet-normal vector, defined as the unit vector $e_n$ perpendicular to the sheet, is perpendicular to $e_l$. The sheet-bound coordinate base is $[e_n, e_r, e_\theta]$, where $e_n$ is the unit vector in the plane of the sheets perpendicular to the fiber direction.

In seeking the unit vector $e_n$, an orthogonal base $[e_l, e_p, e_q]$ is defined. The vector $e_n$ is a linear combination of the unit vectors $e_p$ and $e_q$. For convenience, $e_p$ is chosen to be parallel to the wall surface [c-l plane]. The sheet angle $\beta$ is the single degree of freedom in determining the direction of $e_n$. It holds that

$$e_n = \cos \beta e_r + \sin \beta e_p \quad \text{with} \quad 0 \leq \beta < +\pi$$

Note that $e_n$ coincides with $e_r$ if the fibers are parallel with the wall surface. Prediction of $e_n$ requires variation of $\beta$ until shear in the sheet plane is maximal. Lagrangian strain $E_{\text{fin}}$

$$F_{\text{obj}}(\beta) = w e^{\text{exp},l}_n(\beta) + (1 - w) e^{\text{exp},\theta}_n(\beta) \quad \text{with} \quad 0 \leq w \leq 1$$

Fig. 2. Optimization with the objective function $F_{\text{obj}}$ representing shear magnitude as a function of angle $\beta$ for the example of 30% wall thickness below the epicardium at the apex. For equal weight ($w$) of fiber shear and crossfiber shear (Eq. 3), $w = 0.5$ and $F_{\text{obj}}$ has two maxima, giving rise to two populations of $\beta$. When the weight of fiber shear is increased, only one clear optimum in $F_{\text{obj}}$ is left.

The parameter $w$ indicates the weight of the fiber shear component $e^{\text{exp},l}_n$ relative to the crossfiber shear component $e^{\text{exp},\theta}_n$. For a realistic example, at 30% of wall thickness below the epicardium at the base, the measured strain tensor $E_{\text{exp}}$ (Eq. 2) and fiber direction $e_l$ define an object function, as shown in Fig. 2 for two values of $w$.

Sheet angle probability distribution. Figure 2 shows a general property of the objective function. Commonly, there are two maxima of comparable height, rendering multiple solutions. Therefore, we considered sheet angulation as a stochastic process, characterized by a probability density function. Assuming our hypothesis, the higher the value of $F_{\text{obj}}(\beta)$, the more likely a sheet will have the related orientation. We used the following probability function

$$p(\beta) = \frac{f(\beta)}{\int_0^\pi f(\beta) \, d\beta}$$

with

$$f(\beta) = \exp(6.0 \cdot F_{\text{obj}}(\beta)/\max[F_{\text{obj}}(\beta)])$$

The structure of the probability function is inspired by similar problems in the field of physics, where the ratio of
quantum energies determines the ratio of concentrations by an exponential relationship. The more or less arbitrary factor of 6.0 has been applied for proper visualization of the probability distribution.

From the experiments (5), fiber direction and strain were determined as a function of normalized transmural depth, ranging from 0% at the epicardium to 100% at the endocardium. For a large number of equidistant depth values ($n_d = 45$), the combination of fiber direction and strain resulted in a probability function $p(b)$ according to Eq. 4. With the use of this probability function in a Monte Carlo simulation, a large number ($n_s = 44$) of $b$ values was calculated. For each $b$ value, the related sheet orientation was determined (Eq. 1). Thus a large number of sheet orientations ($n_{ds} = 1,980$) was found as a function of transmural depth.

$Laminar$ $intersection$ $angles$. Experimentally, laminar intersection angles (so-called “cleavage plane” angles in Ref. 12) were measured as the orientation of sheet intersections in histological sectioning. The orientation of the sectioning plane was given by the horizontal and vertical unit vectors $e_h$ and $e_v$, respectively (Fig. 1). Given a sheet-normal vector $e_n$, for the laminar intersection angle $b_{hv}$ it holds that

$$\tan b_{hv} = -\frac{e_n \cdot e_h}{e_n \cdot e_v}$$  \hspace{1cm} (5)

The analysis was performed on short- and long-axis slices by substituting for $[e_n, e_v]$ the vector pairs $[e_h, e_v]$ and $[e_h, e_v]$, respectively. For all selected samples of $b$, the related set of laminar intersection angles $b_{rv}$ and $b_{rl}$ were calculated. The distributions of the resulting predictions were compared with measurements.

**METHODS**

The experiments were described earlier in detail (5). The relevant details will be briefly recapitulated. All animal studies were performed according to the National Institutes of Health Guide for the Care and Use of Laboratory Animals in research. Data were obtained from six open-chest canine experiments. The long axis of the LV was defined as the line connecting the origin of the left main coronary artery with the apical dimple. Wall deformation was measured at sites ~25% (basal) and 75% (apical) down along this axis, both in the region midway between the left anterior papillary muscle and anterior interventricular sulcus (Fig. 3). At each site of deformation measurement, three columns of four to six radiopaque beads were inserted transmurally in a triangular array with sides of 10 mm.

In the experimental protocol, end-diastolic LV pressure was adjusted to 8–10 mmHg. During ventilatory arrest, biplane cineradiographic recordings of several cardiac cycles were made with 120 frames/s. Also, electrocardiograms, aortic pressure, LV pressure, and film shutter pulses were recorded on a multichannel recorder. At the end of the experiment, the heart was brought to an anoxic arrest by placing occluding ligatures around the venous inflow vessels. The LV cavity was then pressurized to 8–10 mmHg by graded saline infusion. The right ventricle was vented. The heart was fixed by retrograde aortic perfusion with buffered glutaraldehyde (2.5%). The heart was excised and stored in 10% buffered formalin for 24–48 h.

For both the apical and basal measuring site, a transmural rectangular block of tissue containing the arrays of beads was removed from the LV wall. The edges of the block were cut along the local radial, circumferential, and longitudinal direction (Fig. 3). In the analysis, these directions are denoted by the wall-bound base $[e_n, e_c, e_f]$ (Fig. 1). The transmural thickness of the wall was measured. Slices (thickness, 1 mm) were taken with $[e_n, e_c]$ and $[e_v, e_f]$ as pairs of horizontal and vertical unit vectors $[e_n, e_c]$ (Eq. 5 and Fig. 3), respectively. Microtomes (50–100 μm) of these slices were imaged using transmitted light and digitized and analyzed by computer. The transmural courses of both laminar intersection angles $b_{rv}$ and $b_{rl}$ were determined manually at 1-mm intervals, respectively (Fig. 4). The remainder of the block was sliced in 1-mm-thick sections from epicardium to endocardium with $[e_n, e_v]$ as the pair of horizontal and vertical axes. These slices were used to determine the transmural course of fiber direction $b_{rd}$. These slices were imaged with reflected light.

**Strain analysis.** Single cardiac beats were selected for each animal with an end-diastolic pressure in the range of 8–10 mmHg. The end-diastolic and end-systolic 3-D coordinates of the beads were calculated from their projections in the biplane images. End diastole and end systole were defined by the cine-frames closest to the peak R wave of the electrocardiogram and the nadir of the dichrotic notch in the aortic pressure, respectively. With the use of the 3-D bead coordinates at the latter moments, a least square fit was applied to compute the continuous transmural distribution of the 3-D Lagrangian strains in the $[e_n, e_v, e_f]$ coordinate base from end diastole to end systole. Finally, the transmural courses of all separate strain components were averaged over all animals to obtain the Lagrangian strain ($E_{exp}$) to be substituted in Eq. 2. The transmural position was defined as depth from 0% at the epicardial surface to 100% at the endocardium.

**Fiber and sheet orientation analysis.** For each animal, the transmural course of the fiber angle $b_{rv}$ was determined from the related slice images. These transmural courses were normalized to wall thickness and averaged for all animals to obtain $b_{rd}$ as a function of depth. The fibers were assumed to be parallel with the wall, neglecting the transverse component. Thus for the unit vector defining the fiber direction it holds that

$$e_f = [0, \cos b_{rv}, \sin b_{rv}]$$ with base vectors $[e_n, e_c, e_v]$  \hspace{1cm} (6)

Knowing the transmural courses of $e_f$ and $E_{exp}$, the probability of the laminar intersection angles $b_{rv}$ and $b_{rl}$ has been indicated by scatter plots showing the angle as a function of

![Fig. 3. Experimental setup. Two blocks of tissue were removed near base and apex, respectively. The arrays of beads were used to determine the transmural course of strain. Fiber direction was obtained from an array of c-f slices. The transmural courses of angles $b_{rv}$ and $b_{rl}$ were obtained from single sections.](http://apjemaj.org/h2224)
transmural depth. These plots will be compared with similar plots of pooled data of all experimentally measured laminar intersection angles \(\beta_{rl}\) and \(\beta_{rc}\), respectively. For each animal, the transmural courses of the laminar intersection angles \(\beta_{rl}\) and \(\beta_{rc}\) (Fig. 3) were determined at 1-mm intervals, with measurements repeated five times. The depth was scaled to wall thickness, as determined from the slice image (Fig. 4). Measured angles were plotted as a function of transmural depth over a principal range of 0–180° (Eq. 5).

**RESULTS**

For the deformation measurements, the hemodynamic parameters were as follows: heart rate, 100 ± 11 beats/min; LV end-diastolic pressure, 9 ± 2 mmHg; and end-systolic pressure, 117 ± 34 mmHg. LV pressure during fixation was 9 ± 1 mmHg. The position of the basal and apical tissue samples (Fig. 3) were at 23 ± 6 and 80 ± 11% distance from the base as normalized to the base to apex distance, at the anterior LV free wall, ~2–4 cm septal of the anterior papillary muscle. Basal and apical wall thicknesses were 12 ± 3 and 10 ± 2 mm, respectively. Figure 5 shows the averaged transmural course of fiber angle \(\beta_{cl}\) at the apex and base. Figure 6 shows the averaged transmural course of the components of the Lagrangian strain tensor \(\varepsilon_{exp}\) with standard deviations for apex and base, respectively. The values of the average curves were substituted in Eq. 2.

The orientation of the sheet is represented unambiguously by the sheet-normal unit vector \(e_n\) perpendicular to the local sheet plane. The probability function \(p(\beta)\) in Eq. 4 indicates whether one or two populations for the regional sheet orientation are expected. The angle \(\beta\) represents the rotation angle around the fiber axis between \(e_n\) and the radial unit vector \(e_r\), with a positive sign of \(\beta\) for a positive sign of the longitudinal component of \(e_n\) (Fig. 1). Figure 7 presents a scatter plot of this angle (modulo 180°) as a function of transmural depth normalized to wall thickness. The value of \(w\) in Eq. 3 was set to 0.5, indicating an equal weight for the n-f and n-s shear components. In the simulations over a large fraction of the wall for \(e_n\), two populations of similar size were found, centered around 45 and 135°, respectively. To relate the populations in the different figures, these populations were named P45 and P135, respectively. Toward the epicardium at the apex, P45 became somewhat more prominent. Near the epicardium, both populations fuse around 90°. At
the base, similar phenomena were seen except for the fusion near the epicardial surface, occurring here more near the P135 population around 160°, which is equivalent to ~20°.

In Fig. 8, scatter plots of the laminar intersection angles $\beta_{rl}$ and $\beta_{rc}$ are shown as functions of depth in the wall for apical and basal tissue samples. The $r-l$ plots reflect the presence of two populations, as also indicated in Fig. 7. The $r-c$ plots show crossing of populations for a laminar intersection angle of 90° at a transmural depth where the fiber angle is zero (Fig. 5). At the crossings the populations are still distinct, as shown by the $r-l$ plots. The narrow distribution of $\beta_{rc}$ at the crossing point is trivial because at that location the fibers are parallel with the vertical base vector $e_c$ in the plane of sectioning by definition (eq. 6). So, at that location, laminas always intersect the cutting plane vertically.

In Fig. 9, scatter plots of the experimentally measured laminar intersection angles are shown (the symbols refer to different animals). Between dogs, substantial variations were seen, but the pooled data in the scatter plots show a close resemblance to the simulated distributions shown in Fig. 8. Data points appeared to be grouped, leaving clear voids in between. The $r-c$ plots show a crossing point near the site where the fiber angle is zero. For the apex, the crossing occurred at an angle larger than 90° (~110°), and for the base, this angle was smaller than 90° (~70°). According to the model, both populations are similar in size, but the experiments suggested asymmetry in likelihood between the populations. At the apex the P45 population appeared dominant, whereas at the base the P135 population was somewhat more dominant.

In Fig. 10, scatter plots of the laminar intersection angles $\beta_{rl}$ and $\beta_{rc}$ are shown as functions of depth in the wall, similar to those in Fig. 8, but now with $w = 0.8$, increasing the weight of the n-f shear to fourfold that of n-s shear. Compared with the case of $w = 0.5$, the general pattern remained the same except for increasing asymmetries in the distribution over both populations. For the base, the P135 population was generally preferred except for a depth of ~25% from the epicardium and a depth of 80% near the endocardium, where an increasing preference for the P45 population was found. For the apex, there was a general preference for the P45 population.

**DISCUSSION**

In the intact canine heart, fiber direction and deformation have been determined experimentally (5). We tested the hypothesis that sheets in the LV wall are oriented along planes of maximum systolic shear strain, taking into account that the fiber direction is contained in this plane. With this hypothesis, two populations of sheet orientation were predicted, often with quite similar probabilities. Therefore, in the anal-

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Fig. 8. Predicted distribution plots of the laminar intersection angles $\beta_{rl}$ and $\beta_{rc}$ as a function of depth for base and apex ($w = 0.5$).

Fig. 9. Pooled experimental data plotting measured laminar intersection angles $\beta_{rl}$ and $\beta_{rc}$ as a function of depth for base and apex. Different symbols denote different experiments. Note the similarity with the prediction in Fig. 8.

Fig. 10. Predicted distributions of laminar intersection angles $\beta_{rl}$ and $\beta_{rc}$ as functions of depth, at base and apex, for a relatively high weight of the fiber shear component ($w = 0.8$). This distribution is a closer approximation of the experimental data in Fig. 9.
ysis, distributions of sheet orientation were described by probability functions. In the experiments, sheet orientations were assessed by the angles formed by sheet intersection lines in tissue slices cut along radial-circumferential and radial-axial planes in the wall. The measured angle distributions also appeared to occur in two populations. Quantitatively, predicted and measured distributions of laminar intersection angles appeared strikingly similar.

Deformation was assessed in the interval from end diastole to the end of ejection. One may question how the findings would be affected when choosing a different time interval. We have no data on this subject. Currently, we think that the maximum span of strain during the cycle is the most important signal related to tissue structuring. For most regions in the heart, the largest span of strain is reached in the time interval we used. After all, the heart is largest in end diastole and smallest at the end of ejection. During the isovolumic phases, however, the heart deforms with constant volume; thus some structures shorten at the cost of stretching of other structures. Therefore, the time interval with maximum deformation may vary over the isovolumic phases. In the present experiments, we have no data on the deformation components during the isovolumic phases.

In the experiments, fiber angles were defined to be parallel with the wall (Eq. 6). So, at a transmural depth where the laminar intersection angle $\beta_{rc}$ equals zero, the fiber direction was assumed to be directed along the circumference. Consequently, the laminar intersection angle $\beta_{rc}$ in the short-axis section should be equal to 90°. The angle $\beta_{rl}$ equals zero near the apex and base at a depth of 20 and 57%, respectively (Fig. 5). At these locations, the angle $\beta_{lr}$ was different from the expected 90°, namely, $\sim 110$ and 70°, respectively (Fig. 9). The latter discrepancies indicate that fiber direction at these depths is not parallel with the wall but spirals inward at the apex and outward at the base, respectively, when following the fibers from anterior to lateral. This behavior of the transverse angle is in agreement with anatomic studies (19) and with a model prediction when assuming uniformity of myofiber load during the ejection phase (18).

From the measurements, the precise values of the transverse components of fiber direction cannot be derived, thus hampering quantitative comparison on the transverse angle. We investigated the sensitivity of the current analysis to a transverse component in fiber orientation by repeating the simulation with a transverse component of ±15°. The predictions on sheet orientations changed moderately, having no impact on the general patterns and conclusions.

Without the constraint of fiber orientation being contained in the sheets, two planes of maximum shear were calculated, being exactly perpendicular and having equal shear. So no preference for one or the other shear orientation would be expected. Adding the constraint causes the planes with maximum shear to tilt. The shear maxima become unequal, which may cause a preference for one of the populations in certain regions.

With this mechanism, the preference may be explained for P45 at the apex and P135 at the base (Figs. 9 and 10, respectively).

Sheet orientation has been correlated with a weighted sum of squared shear components along the fiber direction ($\epsilon_{nf}$) and perpendicular at the fiber direction ($\epsilon_{ns}$) (Eq. 6). Changing the weight factor appears to modulate the asymmetry between the populations P45 and P135. When comparing Figs. 8 and 10 with the experimental data in Fig. 9, the correlation between prediction and measurement of the distributions appears to improve when choosing $w = 0.8$ (Eq. 6), implying a fourfold higher sensitivity for $\epsilon_{ns}$ than for $\epsilon_{ns}$. It is not yet clear whether a difference in sensitivity to shear components is the true explanation for the found asymmetry.

In Fig. 7, two populations of sheet orientations are predicted except for the region near the epicardium. The P45 population describes sheet planes, which are tilted from the basal endocardium to apical epicardium (Fig. 11). The P135 population is approximately perpendicular to P45 and is tilted from the apical endocardium to the basal epicardium [Fig. 4 (left near the endocardium) and Fig. 11]. When pooling the data on all experiments (Fig. 9), both populations were found. Within an experiment, generally, for a given transmural depth the data points belonged to one population. When following the data points transmurally, within an experiment both populations may occur. This finding raises a question as to whether, within one heart, regions can be determined of different populations and, if so, what size these regions are and how they are distributed. Current measurements do not have enough detail to answer this question. Within a single field of measurement, the five samples usually all belonged to the same population. Transmurally, transitions to different populations were often found. Observing the single micrograph in Fig. 4 carefully, patches of different orientations can be recognized, especially in the region at 15–55% from epicardium to endocardium. Comparing angular data in $rc$ and $rl$ slices in the same experiment, the measured angles $\beta_{rc}$ and $\beta_{rl}$ at

Fig. 11. A segment of the cardiac wall showing the P45 and P135 populations of sheet orientation. Left: the (P45) fiber direction is indicated. The sheets contain the fibers. Sheet planes intersect the $rc$, $rl$, and $cl$ planes, showing laminar intersection lines. The transmural courses of these line orientations are sketched as hatched patterns on the sides of the wall segment. Right: the P135 population in somewhat less detail. In a normal cardiac wall, both populations may occur in a patchy pattern.
corresponding transmural locations may belong to different populations, because both measurements could not be performed at the same location. The latter findings suggest that populations of sheet orientations may occur in patches.

In regard to the size and structure of patches belonging to a population, not much information is available. In our experiments, relatively large patches of one population were found, with some smaller regions apparently having a different sheet orientation (Fig. 4). Appearance and structure of the patches may be species dependent. Branching of cleavage planes has been discussed earlier (12). In some cases, this branching may be interpreted as a transition of one population to the other. From the pictures presented in the latter study in a long-axis \( r-c \) slice, patches can be recognized with a size of approximately one-fifth wall thickness. The authors also mentioned that branching density in \( r-c \) slices is found to be minimum near the midwall. In our study, the \( r-c \) plots at the midwall show that sheet intersection orientation is not very different for each population (Figs. 8 and 9). So, at the midwall, despite that branching and patch boundaries may be present, they may not be detected that easily in \( r-c \) sections. Thus the lower density of detected branchings may be more a matter of observation than of real physical presence.

The finding that sheets coincide with the direction of maximum shear may be the result of a general tissue property, that being strain softening. Assume that the tissue starts with a uniform random orientation of collagen fibers. If shear occurs across a plane, the fibers crossing the plane will be stretched and remain stretched due to strain softening (7). This effect will be most pronounced along the orientation of maximum shear. The process does not stop easily because softening will enhance shear deformation, thus increasing the softening stimulus for the few remaining fibers.

When assuming that shear would split the tissue, it is understandable that the P45 and P135 regions do not mix but occur in patches, separated by distinct boundaries. Generally, two mutually perpendicular planes are found with high shear values. These planes represent the centers of both populations. Now assume that, around one of the populations, the tissue splits due to high shear. This layer then serves as a lubrication layer, unloading for shear stress in the plane of the sheet, and consequently also for the plane perpendicular to the sheet, containing the fiber direction. The latter statement is elucidated as follows. Because there is no shear stress component in the plane, the sheet normal direction is a principal direction of the stress tensor. In systole, the fiber direction is also likely to be close to a principal stress direction because stress is nearly maximal for that direction. It then follows that the crossfiber insheet direction coincides with the third principal stress direction. Consequently, the plane containing the fiber direction and the sheet normal bears practically no shear stress in systole. Because the tissue is still intact along that plane, shear strain will be little in that plane too. According to the assumption that shear would split the tissue, chances are low that this occurs in planes perpendicular to the sheets already present.

It is not clear whether structure determines strain or vice versa. In a trial with a cylindrical thick-walled model of cardiac mechanics, the hypothesis tested was whether regional adaptation of the cardiac tissue to mechanical load could lead to a stable maintenance of the cardiac structure. In this model, fiber direction, sarcomere length, and tissue mass were adapted to control systolic fiber strain at a fixed level. Furthermore, the passive tissue was allowed to soften by eventual large deformations (7). As a result, a realistic transmural course of fiber direction formed automatically (1). The current results are also not contradictory to the idea that deformation modifies structure. The thus-modified structure selectively enhances certain modes of deformation. In this view, remodeling may be stable if the feedback loop of structure influencing strain and strain influencing structure converges.

In conclusion, within the ventricular wall, laminar sheets of myocytes are observed, separated by layers of perimysial connective tissue. We tested the hypothesis that these sheets are oriented along planes of maximum shear subject to the constraint that the muscle fiber axis is contained in the sheet. Applying this hypothesis in a model, it was found that sheet orientation is not a unique function of transmural position. Two distinct populations of sheet orientations were found, often with similar probability. When pooling experimental data, both populations were evident and showed close agreement with the simulated directions. Therefore, we concluded that the investigated hypothesis may be valid. A better fit between model and experiment was obtained when assigning a larger weight to the shear component along the fiber direction.

REFERENCES

8. Guccione JM, McCulloch AD, and Waldman LK. Passive material properties of intact ventricular myocardium deter-


