Relationship between Myocardial Fiber Direction and Segment Shortening in the Midwall of the Canine Left Ventricle

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SUMMARY. Myocardial fiber orientation undergoes an orderly transition from the epicardium to the endocardium in the left ventricle, with circumferential fibers predominating in the middle one-third of the heart wall. How fibers lying at different depths in the myocardium, running in different directions, interact to produce local deformation is not known. To define the relationship between the orientation of uniaxial myocardial fibers and local wall motion, we placed three sets of ultrasonic dimension gauges in the middle one-third of the apex-to-base distance of the left ventricle of nine dogs. One pair was placed in line and two intentionally out of line with the presumed local fiber direction. The relative angle between the gauge and the local myofibers was determined by the use of postmortem radiography and histological techniques. Our results show that in the midwall of the left ventricle, myocardial segment shortening is maximal in the direction of local fibers; the shortening measured by gauges placed out of line with the local fiber axis by more than 30° was significantly less than the actual in-line fiber shortening which occurred. This suggests that functional tethering between midwall fibers and endocardial or epicardial fibers does not play a major role in the pattern of midwall deformation. We also documented that an external reference line can be used to predict midwall myofiber direction. Using this line as a guide, ultrasonic dimension gauges could be placed within an average of 12° (range: 0.5 to 18.5°) from the local fiber axis. (Circ Res 56: 31-39, 1985)

The left ventricle of the mammalian heart has a complex morphology which has intrigued investigators for many years. Anatomic studies have demonstrated the manner in which individual myofibers are woven together to form the ventricular chamber (McCallum, 1900; Robb and Robb, 1942; Torrent-Guasp, 1973). Streeter (1979) has demonstrated that myocardial fiber orientation in the frontal plane undergoes an orderly transition from the epicardium to the endocardium, with circumferential fibers predominating in the middle one-third of the heart wall. The way in which fibers lying at different depths in the heart wall and having different orientations interact during the cardiac cycle is not known. One theory holds that individual myocardial fibers act as "slippery skinned rods" which bear tension in only one direction and exert no force on the fibers oriented in other directions (Hort, 1957; Streeter, 1979). According to this theory, significant functional interaction does not occur between layers of myofibers lying in different depths of the heart wall. Another theory holds that such interaction, which some have termed "tethering," does occur. Using the technique of partial coronary occlusion, Gallagher et al. (1982) showed that the shortening behavior of epicardial segments is partially dependent on underlying fibers which are oriented in a markedly different direction. Meier et al. (1982) postulated a similar mechanism in the right ventricle of dogs. These and other studies (Dieudonne, 1969; Ingels et al., 1971; Arts et al., 1982) suggest an important functional linkage between successive myocardial layers. Moreover, the presence of an extensive collagenous network surrounding myocytes, collagen struts between myocytes, and microthreads (3–6 nm in diameter) between myocytes and between collagen struts demonstrates an anatomical framework for functional connections between layers (Caulfield and Borg, 1979; Robinson and Winegrad, 1981).

The net result of the complicated myofiber arrangement is to translate uniaxial sarcomere shortening into three-dimensional deformation of the ventricular cavity. In the absence of functional connections between fibers lying at different depths in the heart wall, local segment shortening would be greatest in a direction parallel to local fiber axis. This relation has been assumed by numerous investigators, who have used ultrasonic transit time-dimension gauges to study regional function, and aligned the gauges with the presumed fiber orientation. Techniques used to analyze ventricular function noninvasively, such as echocardiography, presume that, since most of the ventricular fibers are circumferential, the measurement of a linear dimension in this direction will reflect overall myofiber performance (Mitchell et al., 1969; Karliner et al., 1970;
Cooper et al., 1972). If fibers that are oriented in different directions do have important functional connections, systolic deformation will include both shortening along the axis of the fibers and motion related to such connections. The latter may be termed "lateral strain," since it is in effect sideways motion of the fibers. If lateral strain occurs, the behavior of linear segments will not depend solely on the local fiber orientation, but, to some extent, on a pattern of global ventricular deformation.

The purpose of this study was to determine how left ventricular midwall segment shortening is influenced by deformation in other layers by examining the relationship between local shortening and fiber direction. We were also interested in whether the orientation of local fibers could be predicted accurately from external landmarks in the intact, beating heart, and how closely ultrasonic dimension gauges could be aligned with the fiber axis.

Methods

Nine mongrel dogs were anesthetized with pentobarbital, 25 mg/kg, iv. The animals were intubated and respiration was supported by a Harvard ventilator; supplemental anesthesia was administered as needed. A midline sternotomy and bilateral 5th interspace thoracotomy was performed, and the heart was suspended in a pericardial cradle. The azygos vein was ligated, and sutures were positioned around the caval veins, the hila, the ascending aorta, and the innominate, left carotid, and left subclavian arteries. These were left loosely in place. A 7F micromanometer-tipped catheter (Millar Instruments) was placed in the left ventricle via the left carotid artery and a fluid-filled 8F pigtail catheter was introduced into the left ventricle via the left femoral artery. The micromanometer-tipped red catheter was calibrated to the fluid-filled catheter, and the latter was then withdrawn to the ascending aorta.

Quantitative information on the direction of fibers in the left ventricular wall stems primarily from the extensive work of Streeter (1979). In those studies, an internal reference axis system was employed which is not directly accessible in the beating heart. The current studies required that pairs of piezoelectric crystals be inserted in the midwall of the ventricle in a known relationship to the local fibers. Accordingly, we developed an empirical technique to estimate the location of the internal reference axis from an external reference axis on the surface of the beating heart. A length of 3-0 silk suture was sewn to the epicardium over the apical dimple, passed over the surface of the LV by the shortest path and secured to the epicardium just below the bifurcation of the left coronary artery. This was left in place for the duration of the study. Three sets of ultrasonic dimension gauges were then placed in the midwall of the left ventricle. Polyethylene sheathes with a flange 0.5 cm from the gauge crystals aided in insertion into the midwall at a reproducible depth. All gauges were placed in the middle one-third of the apex-to-base distance of the ventricle. The middle set of crystals was placed in the direction of the local fiber axis as predicted from prior anatomic studies (i.e., perpendicular to the external reference axis). The other gauges were intentionally placed out of line with the predicted local fiber axis (Fig. 1).

In two additional dogs, the same protocol was carried out, but the three pairs of dimension gauges were placed in the most superficial layer of the epicardium. All recordings were made on a Brush-Clevite model 2000 oscillograph, at a paper speed of 200 mm/sec. Control aortic pressure, left ventricular pressure, and segment gauge signals were recorded with respiration suspended at end-expiration, and all values were averaged for three successive beats.

The segment length at onset of ejection (LOE) was defined as the length at the time in systole when the ventricular pressure became equal to the aortic pressure. The value of pressure at the rapid upstroke of the aortic tracing was noted, and transposed to the micromanometer tracing to account for the time lag between these two catheter systems. The end-systolic length (ESL) was defined at the time when LV pressure fell below aortic pressure, using a similar time lag correction. Ejection phase shortening was equal to (LOE—ESL)/LOE.

After instrumentation, recordings were made at control left ventricular end-diastolic pressure (LVEDP) which ranged from 7 to 10 mm Hg. Measurements were then repeated after infusion of normal saline to raise the LVEDP to approximately 15 mm Hg. Left ventricular pressures were then allowed to return to the control state, and measurements were made following a 2-μg iv bolus of calcium chloride. After return to the control state once more, measurements were repeated a final time at the time of peak response to a 2-μg iv bolus of isoproterenol.

At this point, the micromanometer-tipped catheter was removed, and the pigtail catheter was reintroduced into the ventricle. The previously placed sutures were tied, isolating the heart. After injection of KCl to arrest the heart, the LV pressure was adjusted to the control value (7–10 mm Hg) by infusion or withdrawal of saline, and the heart was fixed by rapid intracoronary injection using the technique of Yoran et al. (1973). Bouin’s solution, which consists of 10 g of picric acid crystals, 50 ml of glacial acetic acid, and 250 ml of 37% formalin in 750 ml H2O, was used as the fixative agent. The heart was excised, the right ventricle and atria dissected away, and the LV was cast with latex and immersed in Bouin’s solution. When the latex was set a metal skewer (4.8 mm o.d.) was passed from the apical dimple to the mitral aspect of the

FIGURE 1. The positions of the external long axis marker and ultrasonic dimension gauges. The middle set of crystals is perpendicular to the long axis, while both the apical and basal sets are at an angle to it.
FIGURE 2. Panel A: radiograph of a ventricular slice, showing that the crystals are in the midwall (points A and B). The marker on the left shows the location of external long axis. Point C marks the center of the x-ray beam and point D shows the location of the internal long axis marker. Panel B: a radiograph showing the internal long axis marker and dimension gauges. In this example, the external long axis marker overlies the internal marker, which also obscures the position of one crystal.

FIGURE 3. Photomicrograph of histological section with the reference edge on top. The crystal defect is clearly visible.

aortic valve (the long axis used by Streeter). A fine wire marker was placed over the silk suture determining the external long axis. The heart was then positioned in a special holder, and radiographs were taken with the external marker lying in a plane perpendicular to the table (see Fig. 2). This allowed determination of angle between internal and external long axis markers.

After removal of the external marker, the ventricle was cut into rings, approximately 1 cm thick, perpendicular to the internal long axis, and radiographs were taken of these (see Fig. 2). Transmural blocks measuring approximately 1 x 1 cm which included each crystal were excised and were notched to identify their reference edge. The crystals were gently removed, and the tissue blocks were embedded in paraffin. Tissue shrinkage of 15–20% is expected using our processing technique (Humason, 1979). This was assumed to be homogeneous. Sections 10 μm thick were taken, beginning at and tangent to the epicardium, proceeding to the endocardium. Defects at the site of the crystals were clearly seen, and 15–20 sections were mounted from this region (Fig. 3).

A goniometer eyepiece (accurate ±0.25°) was used to determine the angle between the fiber direction and the reference edge in the six adjacent sections from each block in which the crystal defect had its largest diameter. An average value was calculated, and this value was used in the subsequent analysis. In two instances, sections were mounted through the entire course of the crystal (approximately 60 sections) to determine how much the local angle changed in this depth of the heart wall.

The angle between the intracystal axis and the internal long axis was determined from the radiographs (Fig. 2A). The angle between the local fiber direction and the internal long axis was determined from the microscopic slides (Fig. 3). Together, the radiographic and histological measurements allowed calculation of the relative angle between local myofiber orientation and the intracystal axis.

When the angle between the reference edge and the myofibers was different in the two crystals of a given pair, the average of these values was used.

Results

Figure 4A illustrates the construct used in our data analysis. The myofibers run in a direction parallel
FIGURE 4. Panel A: the geometric relation between the crystals and the fiber directions shown when relative angle = 0° (segment AB) and when relative angle = θ (segment AD). For clarity, all shortening is shown to occur at one end of the segment. See text for details. Panel B: effect of increasing end-diastolic segment length on geometric construct. Panel C: the ratio of measured shortening to in-line shortening as a function of the relative angle between the myofibers and the intragauge axis. Upper solid line shows the relation assuming 10% in-line shortening and no lateral strain. Lower solid line shows the relation assuming 30% in-line shortening and no lateral strain. Dashed line shows the relation assuming 10% in-line shortening and 5% lateral strain. See Appendix for the mathematical derivation of these relations, and text for details.

\[
\frac{(1 - A_n)}{(1 - A_i)} = \cos^2 \theta + \left( \frac{1 - A_n}{1 - A_i} \right) \sin^2 \theta. \tag{1}
\]

θ is the relative angle at the onset of shortening, <DAB, and Δn is the shortening resulting from lateral strain (BD - BC)/BD.

The solid lines in Figure 4C depict the theoretical relation of measured shortening as a percent of in-line shortening as the angle θ goes from 0° to 90°, assuming that no lateral strain occurs (Δn = 0). As can be seen from Equation 1, the relation of measured to in-line shortening is dependent on the amount of in-line shortening which occurs; however, this factor plays a small role. The upper solid line in Figure 4C assumes in-line shortening to be 10% and the lower solid line assures in-line shortening to be 30%. Since a 3-fold increase in shortening would alter the ratio of Δn/Δi by such a small amount, we have excluded this factor from subsequent analysis. Data on Figures 6 and 7 are plotted against the line assuming Δn = 10%, which is near the mean control value from our experiments (10.5%).

The dashed line in Figure 4C represents the relation of measured to in-line shortening, assuming that the in-line shortening is 10% and the shortening due to lateral strain is 5%. This plot demonstrates that, if lateral strain occurs, the measured shortening would fall above the line of prediction used in our construct, which assumes no lateral strain. Furthermore, the divergence of these two constructs is a function of relative angle: as relation angle increases, the influence of lateral strain becomes more pronounced.

We analyzed the relationship between the relative angle and shortening magnitude for each crystal pair. Values in each dog were normalized to the crystal pair with maximal shortening and expressed as a percent. Data from one segment gauge in each of two dogs was excluded for technical reasons.

To determine how the myofibers were oriented at the crystal site, we used the histological sections in which the crystal defect had its largest diameter. Through a 1- to 1.5-mm layer of the midwall (representing the diameter of the crystal), the fiber orientation undergoes some transition. In two instances, we analyzed sections taken every 50 μm throughout the depth of crystal defect. Figure 5 shows data from one of these. The mean fiber direction from the six sections with the maximal defect was within 2° of the average of the values of all 50 sections; in the other instance it was within...
3°. These data indicate that the method of choosing sections for analysis from the maximal defect diameter approximates the average direction of the fibers acting on the entire crystal.

**Shortening Data**

The shortening values for all segments ranged between −2.5% and 18.6%. The peak value of control shortening ranged from 7.6 to 18.6% in these animals. As can be seen from the solid lines in Figure 4C, control shortening values in this range do not substantially alter the relation between measured shortening and relative angle.

Figure 6 shows our control data and the relation predicted assuming no lateral strain. As the relative angle became larger, the measured shortening value decreased. The points are distributed on both sides of the line of prediction. A linear regression of the relation between observed and predicted values revealed a correlation coefficient of 0.74. In every case where an animal had a segment with a relative angle greater than 30°, the shortening in that segment was less than that in the more well-aligned segments.

An unpaired t-test between segments with relative angle less than 30° and greater than 30° showed that the latter had lower shortening values (P < 0.05).

Volume infusion caused an increase in the percent maximal shortening at any given relative angle in 13 of 17 segments. Isoproterenol and calcium did not systematically alter the shortening to relative angle relation.

Data from the additional two dogs in which the dimension gauges were placed in the epicardium is shown in Figure 7 (open circles). In five of the six measurements, the relationship between relative angle and shortening falls above the line of prediction.

The projection of the external and internal long axis markers in eight dogs showed that the angle formed by them ranged from 0° to 6.5° with a mean of 1.7°, indicating that the external reference system accurately reflected the internal Z axis used in earlier studies to define myocardial fiber anatomy (Streeter, 1969).

In each dog, we attempted to place one set of crystals in line with the local fibers using the external reference system as a guide. In nine dogs, the average relative angle for this gauge, representing the error in alignment of the dimension gauge, ranged from 0.5° to 18.5° with a mean value of 12°.

**Critique of the Method**

The assessment of an in situ relative angle is technically difficult. Care was taken to fix the individual hearts at a pressure corresponding to the control LVEDP. Despite the use of specialized holders and knife guides, error of 1–2° may have been introduced when reference edges were cut. Also, use of 1-cm² samples for histological determination of local fiber direction necessitates the estimation of a precise fiber orientation for an area in which there
is some geometric variability. The samples used by Streeter (1979) were considerably smaller, approximately 0.1 × 0.7 cm, ensuring less inherent variability. Use of small samples, however, would make verification of the crystal site impossible, and not allow an assessment of the average angle acting on the crystals, and thus was not practical for this study.

Another possible source of error is the difference in depth within the myocardium at which the crystals were placed. Although calibrated sheathes were used to insert each to the same depth, and although radiography showed the crystals to be in the midwall (Fig. 2B), slight differences in depth were unavoidable. When the crystals were at slightly different depths, there was a difference between the fiber angle at the center of each gauge (range 1° to 42°). The fiber angle was calculated as the average of these two values for each gauge. Despite the difference in fiber angle at each gauge, the entire gauge subtended up to a 20° range of angles, and there was always substantial overlap in fiber direction at the two gauges. However, the dispersion of fiber angles between the two gauges may have added to the scatter apparent in the data.

Figure 5 graphically depicts the fact that, even though the gauges were small in comparison to the thickness of the ventricular wall, fiber orientation was not the same at all points through the depth of the crystal. Our method of defining the local fiber direction took advantage of the spherical nature of the defect produced in the fixed tissue by quantifying the angle at the widest aspect of the defect. We demonstrated that this was within 3° of the average of the angles measured in serial sections produced for the entire depth of the crystal, and feel that it is a good estimation of the net direction of the fibers acting on the crystals. It is still possible that ±3° error was introduced by this technique, and that this added to the variance of the data shown in Figure 6.

For our analysis, we assumed that circumferential shortening was the same at the three gauge sites in any one animal. Prior studies from our laboratory have shown modest regional differences in both systolic (LeWinter et al., 1975) and diastolic (Lew and LeWinter, 1983) deformation patterns at different sites in the left ventricle. These studies have compared widely disparate regions of the ventricle—the base vs. the apex or the anterior vs. lateral wall; there is no evidence for marked differences in deformation in areas as small as those analyzed in this study. Thus, we feel this assumption is justified.

**Discussion**

Our data demonstrate that, in the midwall of the left ventricle, shortening is maximal in the direction of the local myofiber, and thus supports the hypothesis that lateral strain introduced by functional connections to overlying muscle layers does not play a dominant role in this region. Thus, although anatomic linkage in the form of collagen network has been demonstrated between myocardial layers (Caulfield and Borg, 1979; Robinson and Winegrad, 1981), epicardial and endocardial influences do not reduce the dependence of shortening patterns in the midwall on local fiber orientation. Since midwall shortening is determined primarily by local fiber orientation, placement of segment gauges markedly out of line with the local fibers in this region will result in falsely low shortening values. This theoretical relation is illustrated by the solid lines in Figure 4C, and its mathematical derivation shown in the Appendix. It is sigmoidal in nature such that measured shortening becomes increasingly less accurate as relative angle becomes larger. Our data correspond to the theoretical relation, and indicate that gauges placed more than 30° off of the local fiber axis will report shortening that is substantially less than that of the local myofibers.

The mathematical relation between measured and in-line shortening, defined by Equation 1, is of interest for several reasons. The relation is complex, being dependent on the relative angle, $\theta$, and the amounts of both in-line and lateral shortening. For our construct, we have assumed that no lateral strain occurs, or $\Delta \epsilon = 0$. As can be seen from the equation, if lateral strain does occur, the ratio of measured to in-line shortening will be larger than that predicted by our construct. The dashed line in Figure 4C is a plot of the relation when lateral shortening occurs with a magnitude equal to half of the in-line shortening. The relation falls above the line predicted by our construct, deviating more from it as the relative angle approaches 90°. In the case in which lateral shortening is equal in magnitude to in-line shortening, the ratio of measured to in-line shortening will always be 1.0, indicating a concentric decrease in size of the heart from end-diastole to end-systole. Since our midwall data indicate that gauges placed substantially out of line with local fibers measure very low shortening values, it lends support to the concept that, at this depth in the left ventricular wall, lateral strain arising from fibers in the epi- and endocardium does not alter the dependence of local deformation on local fiber alignment, or, at the least, provides a balance and opposing effect.

We limited our study to the middle one third of the ventricular apex-to-base distance. At the apex, the wall is thinner, the fiber orientation is more curvilinear and the pitch angle is steeper. At the base, the fibrous skeleton could affect deformation. Whether the relation of local fiber direction to local segment shortening persists in these regions remains to be studied.

A number of clinical techniques used to evaluate ventricular function depend on measurement of linear dimensions during the cardiac cycle. Those which measure an internal diameter such as echocardiographic or cineangiographic analysis of diameters (mean circumferential fiber velocity) must assume that the chord used for analysis is aligned with the circumferential fibers, and that no regional differences of function are present. Although these
assumptions cannot be directly verified in the individual hearts being studied, in the absence of obvious wall motion abnormalities they probably permit a reasonable estimate of the behavior of the midwall circumferential fibers. Our results suggest that other methods, particularly those which use a surface parameter to evaluate wall motion (Massie et al., 1978; Ingels et al., 1980; Karsch et al., 1980) will not directly reflect performance of the functional unit of the myocardium, the uniaxial myofiber.

Gallagher et al. (1982) showed that epicardial shortening behavior may be influenced by shortening which is occurring in the midwall fibers. These investigators directly measured the relation of local fiber orientation to intracystal axis by in situ determination of the relative angle with a protractor. In their studies, significant epicardial deformation occurred in the circumferential direction, which was approximately 70° out of line with the local epicardial fiber direction. Coronary occlusion producing midwall and subendocardial but not epicardial ischemia eliminated the epicardial circumferential deformation. The solid circles in Figure 7 show an analysis of the pre-ischemic control data from those animals, using our construct. Clearly, most of the segments have shortening values that fall above the line of prediction, which assumes no lateral strain, and more closely approximate the relation illustrated by the dashed line in Figure 4C, where lateral strain is assumed to occur. Data from the two animals in which we placed epicardial gauges and directly measured fiber angle are also shown, and similarly fall above the line of prediction. Thus, unlike the midwall, the epicardium undergoes substantial lateral strain.

The reason for this seeming paradox may be that, since a preponderance of the myofibers in the left ventricular wall are oriented in the circumferential direction, most of the force leading to deformation has a circumferential vector, whereas a smaller fraction of total force is exerted by fibers lying in a more longitudinal direction. This would suggest that lateral strain would be more apparent in the epicardial (and presumably endocardial) layers than in the midwall layers. Furthermore, it is also possible that lateral stress introduced by epicardial fibers may be balanced by the effects of subendocardial fibers which are oriented in an opposite direction. This would predict that, under conditions of strictly subendocardial ischemia, lateral strain resulting from epicardial fibers would be unmasked in the midwall region. Likewise, if the influence of epicardial fibers could be selectively removed, the influence of subendocardial fibers on midwall deformation would become apparent.

Any long axis shortening which occurs must be a result of noncircumferential fibers in the epicardium and endocardium. The importance of helical fibers in determining ventricular performance was described mathematically by Sallin (1969). If the left ventricle is assumed to be an ellipse of revolution, circumferential shortening alone will not permit development of ejection fractions comparable to those observed in vivo. Shortening of helical fibers (anatomically, predominantly the endocardial and epicardial fibers) will lead to physiological ejection fractions. Meier et al. (1982) have demonstrated that motion of epicardial markers on the canine right ventricle confirms the importance of helical shortening patterns in ventricular ejection. Although one would think that this long axis deformation would lead to lateral strain in the midwall, it's overall magnitude is small: Mitchell et al. (1969) demonstrated that long axis shortening is on the order of 8–10%. Furthermore, although mathematical analysis has assumed uniform shortening behavior along the helical pathway it is possible that regional variations of long axis deformation may occur. In any case, our data suggest that this helical shortening pattern does not introduce substantial lateral strain in the midwall during ejection.

It is of interest to note that volume infusion caused an increase in the percent maximal shortening at any given relative angle in almost all of the segments. In Figure 4B, it can be seen that an increase in end-diastolic length would cause the angle θ to become smaller (<DAB greater than <JAB). Since the hearts were fixed at control volumes, the relative angle we measured would overestimate the angle present during the acquisition of increased volume data. Shortening values from that state should thus fall above the line of prediction based on control relative angles, as was the case.

The administration of calcium and isoproterenol, on the other hand, did not consistently change the results. Inotropic stimulation causes greater segment shortening from the same or lower end-diastolic length in the intact heart. An interesting facet of our geometric construct is that, although the relation between relative angle and measured shortening is dependent on the amount of in-line shortening, this effect is quite small (Fig. 4C). It is therefore understandable that the data did not show a consistent change in the relation between shortening and relative angle after inotropic interventions.

Our secondary goal was to determine whether gauges could be accurately placed in the fiber direction in the beating heart. The description of fiber orientation generally used as a reference system for gauge placement (Streeter et al., 1969) was based on an internal long axis which passed from the apical dimple to the mitral aspect of the aortic valve. Our radiographic data illustrate that the line passing from the apical dimple to the bifurcation of the left coronary artery is an excellent predictor of the internal long axis and can be used to predict local fiber orientation.

When using this reference system, we found a wide variability in the accuracy with which gauges could be implanted. In nine attempts to align a gauge with the circumferential fibers, the relative angle ranged from 0.5° to 18.5°, with a mean value of 12°. This variability probably relates to the difficulty
in implanting crystals precisely in the moving heart, the presence of epicardial coronary arteries which limit insertion sites, and individual variations in the actual fiber geometry. Fortunately, the shortening measured by gauges falling within 18.5° of the fiber axis is reasonably close to the shortening along the fiber axis, preventing this from being a major source of error. Furthermore, in animal studies, the accurate placement of gauges in relation to the external long axis which closely predicts the internal long axis can be directly verified.

In conclusion, we have demonstrated that, in the anterior ventricular midwall, segment shortening is maximal in the orientation of the local myocardial fibers, indicating that lateral strain does not reduce the dependence of segment motion on the underlying myofiber orientation in this region during ejection. We have shown that the fiber orientation can be assessed accurately by means of a reproducible external reference system, and that segment gauges can be aligned to local myofiber direction with acceptable error.

Appendix
(see Figure 4A)

$$\theta = \angle DAB$$

In-plane shortening = $$\Delta \lambda = (AB - AC)/AB$$

Shortening due to lateral strain = $$\Delta \lambda = (BD - BC)/BD$$

Measured shortening = $$\Delta \lambda_m = (AD - AF)/AD$$

For any $$\Delta \lambda$$, $$AC = K \cdot AB$$, where $$K = 1 - \Delta \lambda$$

For any $$\Delta \lambda$$, $$BG = K \cdot BD$$, where $$K = 1 - \Delta \lambda$$

For any $$\Delta \lambda_m$$, $$AF = K \cdot AD$$, where $$K = 1 - \Delta \lambda_m$$

$$\sin \theta = BD/AD$$

$$\cos \theta = AB/AD$$

$$AF^2 = AC^2 + CF^2 = AC^2 + BG^2$$

By substitution ($$K_m = AD$$)

Rearranging terms demonstrates that

$$\frac{K_m}{K} = \left(\frac{AB}{AD}\right)^2 + \left(\frac{K}{K_{ef}}\right)^2 \cdot \left(\frac{BD}{AD}\right)$$

or

$$\frac{K_m}{K} = \cos^2 \theta + \left(\frac{K}{K_{ef}}\right)^2 \sin^2 \theta.$$

By substitution

$$\frac{\left(1 - \Delta \lambda_m\right)^2}{\left(1 - \Delta \lambda\right)^2} = \cos^2 \theta + \left(1 - \Delta \lambda\right)^2 \sin^2 \theta.$$
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