LETTERS TO THE EDITOR

Comments on
"Relationship between Myocardial Fiber Direction and Segment Shortening in the Midwall of the Canine Left Ventricle"
which appeared in
Circ. Res. 56: 31-39, 1985

In a recent paper, Freeman and colleagues (1985) studied the relation between fiber orientation and segment shortening in the middle layers of the left ventricular free wall. The authors conclude that, in these layers, myocardial segment shortening is maximal in the direction of the local fibers, and, subsequently, that functional tethering between midwall fibers and endo- or epicardial fibers does not play a major role in the pattern of wall deformation.

The first conclusion was based on the agreement between their experimental findings and their concept of the constraints of myocardial shortening. In these comments we will show that the experimental data presented by Freeman and associates (1985) can also be explained alternatively by cardiac deformation. In the study by Arts and colleagues (1979, 1982), left ventricular deformation is described by circumferential shortening, base-to-apex shortening, and torsion (defined as a rotation of the apex with respect to the base around the long axis of the left ventricle). Torsion is associated with shear deformation of the wall. Due to the presence of a shear component, segments directed at equal positive and negative angles with the fibers of a layer (AC and AD in Fig. 1) show a different amount of shortening, in this respect, our description of cardiac deformation is essentially different from the one given by Freeman and colleagues, because they assume shortening to be symmetrically distributed around the fiber direction.

The existence of shear components of deformation of the free wall of the left ventricle is demonstrated in earlier experiments (Fenton et al., 1978; Arts et al., 1982, 1984; Prinzen et al., 1984). In the latter study by Prinzen and associates, deformation of the subepicardial and subendocardial layers of the left ventricular free wall was determined in six open-chest dogs. Estimating midwall deformation by linear interpolation of the median values of epicardial and subendocardial deformation, circumferential shortening, base-to-apex shortening, and shear are calculated to be 10.5%, 8.6%, and 0.063 rad, respectively. From these values, the direction of maximum shortening can be calculated (Arts et al., 1982; Prinzen et al., 1984) and is found to be at a substantial angle (36° in this particular case) with the circumferential direction, which is the approximate fiber direction in the middle layers. Thus, the maximum of the curve, representing shortening as a function of the angle with the fiber direction, is shifted from the vertical axis (zero angle, Fig. 2). In the approach of Freeman and colleagues, no distinction is made between positive and negative angles. Therefore, as shown in Figure 2A, the part of the curve with negative angles is mirrored with respect to the vertical axis, introducing a curve with relatively low values of shortening at angles close to the fiber direction (broken line). The presence of this split branch cannot be excluded by the experimental results of Freeman and colleagues (the dots in Fig. 2), and, as a consequence, the fiber direction does not necessarily coincide with the direction of maximal shortening, as stated in their conclusion.

In our view of left ventricular mechanics, the precise value of the angle between the direction with maximum shortening, and the fiber direction (the angle of deviation) depends on the localization in the myocardial wall, as far as depth is concerned. At the epicardial surface, fiber orientation and maxi-

![Figure 1. The influence of shear deformation on the length of two segments (AC and AD) at the same angle as the fiber direction (AB). Shear causes the deformation of a square (upper paneD into a parallelogram. Consequently, segment A'C' is larger than segment AC, but segment A'D' is smaller than segment AD. Segment lengths of AB and A'B' are equal to each other.](http://circres.ahajournals.org/ by guest on September 23, 2017)
Concerning the suggestion that the fibers in the subendo- and subepicardial layers may not tether the middle layers, the following remarks can be made. Two forms of tethering have to be distinguished: a direct form, related to tight interconnections of neighboring tissues, and a global form, associated with changes in the distribution of loading within the whole structure of the heart. In the normal contracting left ventricular wall, the first form of tethering is probably of minor importance because of the strongly anisotropic properties of cardiac muscle. The second form is likely to be more important. If the subendo- and/or subepicardial layers are not active, a larger fraction of the transmural pressure has to be generated by the remaining, healthy middle layers in accordance with the mechanism of Laplace’s law. So, under those circumstances, fiber stress in the middle layers increases as a consequence of failure of other layers.

In conclusion, in our opinion, the direction of maximum shortening does not necessarily coincide with the fiber orientation, which is especially true in the subendocardial layers. When defining tethering in the broad sense of the word, i.e., including functional tethering, mutual tethering between all muscular structures occurs.

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Reply to the Preceding Letter

Prinzen and his colleagues have correctly pointed out that we did not include a shearing component in our construct of midwall deformation (Freeman et al., 1985). On the basis of their data, they feel that such shearing occurs, and their model of contraction predicts that maximum shortening is approximately 36° off the fiber direction (Prinzen et al., 1984). Our study (Freeman et al., 1985) showed that in the midwall of the left ventricle the percent maximal shortening measured in any segment fell along a relationship predicted by the angle between that segment and the local fiber orientation, with maximum shortening taking place essentially in line with the local myofibers.

Although Dr. Prinzen's studies and ours are not entirely dissimilar, there are several key differences. Whereas we directly measured uniaxial shortening at the midwall site in question, they extrapolated shortening values for midwall points from sets of gauges attached to pins placed through the heart wall. It seems likely that this technique would emphasize tethering effects, and possibly alter deformation, since there is no reason to suspect that points which are colinear across the wall at end diastole will remain so throughout contraction. Furthermore, their conclusions are based on assumed fiber geometry, as predicted from the work of Streeter and Hanna (1973). It can be seen from Figure 8 of this work that, in the middle one-third of the heart wall, there is a range of fiber orientations of greater than 30°. Whether data with such variance can be used for prediction of local fiber direction in individual animals is subject to question. We feel that it is essential to assess the fiber anatomy in each animal being studied.

In reality, both our approach and that of Dr. Prinzen and his colleagues are probably oversimplified. Although all cardiac deformation must result from the action of sarcomeres, which are uniaxial shortening units, myofiber shortening is accompanied by myofiber thickening and perhaps transmural fiber rearrangement (Spotnitz et al., 1974). Two-dimensional analysis of motion, as used by both Dr. Prinzen and ourselves, does not permit separation of active shortening from thickening and fiber rearrangement. Recent studies from this laboratory have provided complete descriptions of the three-dimensional strains present through the depth of the heart wall (Waldman et al., 1985). These studies have shown that there are substantial transverse shears at all depths through the heart wall. When principal strains and the directions of their principal axes were determined, it was shown that, although the orientation of the principal axes of shortening varies through the depth of the heart wall, it does not vary nearly as much as the corresponding fiber angle. We agree with Prinzen and colleagues that such tethering contributes to the overall pattern of myocardial deformation, particularly in the epicardial and subendocardial regions (Gallagher et al., 1982, Freeman et al., 1985). It is difficult, however, to envision that either tethering or fiber rearrangement can produce deformation out of line with myofibers which has a magnitude greater than that present in the in-line active shortening units.

We thank Dr. Prinzen and his colleagues for their interest in our study. It is clear that motion of the left ventricular wall is quite complex, and that its full description awaits further study.

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Comments on
"Myocardial Micronecrosis Produced by Microsphere Embolization: Role of an α-Adrenergic Tonic Influence on the Coronary Microcirculation" which appeared in Circ. Res. 54: 74–82, 1984

We have read with great interest the report by Eng et al. (1984). This work perfectly synthesized the possibilities inherent in morphological and pharmacological methods to obtain more complex information about the pathogenesis of the micronecrosis in the myocardium. They described the prevention of myocardial embolic lesions by α-receptor-blocking agents, using the microsphere embolization method. Therefore, α-adrenergic influence was suggested as contributing to the development of necrotic foci. In connection with these results, some aspects should be noted.

A pathological condition exists in which increased
adrenergic sensitivity can be demonstrated, both in
the myocardium and in the vasculature. In experi-
mental diabetes, adrenergic agonists produced in-
creased contractions in isolated aortic rings (Scar-
borough and Carrier, 1983). This adrenergic hyper-
sensitivity could also be observed in the myocardium
of diabetic lambs (Downing et al., 1983). In in vivo
studies, altered adrenergic responses in the coronary
arterial bed of alloxan-diabetic dogs have been de-
scribed (Palik et al., 1982a).

These data suggest that the myocardial lesions in
diabetes therefore should be larger and more severe
in course. In fact, the size of infarction under the
influence of increased circulatory levels of catechol-
amines after 48 hours of coronary artery ligation did
prove to be significantly ($P < 0.001$) larger in al-
loxan-diabetic than in metabolically healthy dogs
(Palik et al., 1982b).

Thus, the diabetic state and microembolization
may cause myocardial necrosis, in part mediated by
$\alpha$-adrenergic receptors.

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The authors of the article discussed had no further comment.
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