Noninvasive Quantification of Left Ventricular Rotational Deformation in Normal Humans Using Magnetic Resonance Imaging Myocardial Tagging

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It has been postulated that rotation of the left ventricular apex with respect to the base is a component of normal systolic function in humans, but it has been difficult to measure it noninvasively. Tagging is a new magnetic resonance imaging technique that labels specific areas of myocardium by selective radio-frequency excitation of narrow planes orthogonal to the imaging plane before acquiring an image. Tags appear as black lines and persist in myocardium for 400–500 msec and, if applied at end diastole, will move with the myocardium through systole. Tagging was used to noninvasively quantify left ventricular torsion and circumferential-longitudinal shear (shearCL) in humans. Eight normal volunteers, aged 24–38 years, were imaged in a 0.38-T iron-core resistive magnet. Five short-axis left ventricular images, positioned to encompass the entire left ventricle (LV), were obtained separately at end systole. Four equiangular radial tags had been applied at end diastole, intersecting the myocardium at eight locations. We calculated the difference in angular displacement of each epicardial and endocardial tag point (a tag point being where the tag crossed the epicardium or endocardium) at end systole from the systolic position of the corresponding tag point on the basal plane. This value was called the torsion angle. From this, shearCL, the angle inscribed on the epicardial or endocardial surface between the systolic tag position, the corresponding basal tag position, and its projection onto the slice of interest could be calculated at 32 points in the left ventricular wall. The mean of the torsion angles of the eight locations on the apical slice, relative to the mean of the torsion angles of the base for endocardial points (endocardial torsion), was 19.1±2.0° (mean±SEM, p<0.001), counterclockwise when viewed from the apex. Epicardial torsion (counterclockwise, 11.2±1.3°, p<0.001) was 8±1.9° less than the endocardium (p<0.01). Torsion (mean of torsion angles between base and apex) in the posteroseptal regions was less than in anterolateral regions for both endocardium (12.4±2.9° vs. 23.1±4.4°, p<0.001) and epicardium (6.4±3° vs. 12.8±3.1°, p<0.04). The torsion angle increased with distance from the base for both epicardium and endocardium. Different amounts of torsion, however, were found to result in similar amounts of shearCL for both epicardium (5.0±0.6°) and endocardium (4.0±0.5°), which did not increase with distance from the base. Therefore, torsion varies with distance from the base and from the center of the LV but constancy of shearCL at each level of the LV is achieved. This constancy of shearCL may represent an important principle by which stress through and along the left ventricular wall is equalized during normal ejection. (Circulation 1990;81:1236–1244)
It has been postulated that relative rotation of the left ventricular apex with respect to the base, about a left ventricular long axis, is a component of normal systolic function.\textsuperscript{1,2} Indeed, mathematical models of the left ventricle (LV) suggest that this rotation might equalize end-systolic fiber stress across the left ventricular wall.\textsuperscript{3,4} Relative rotation of the apex occurs in dogs, as shown by both strain gauges and radiopaque markers,\textsuperscript{5,6} and in humans in whom similar markers have been implanted at cardiac surgery or transplantation.\textsuperscript{7,8} These methods, however, have significant drawbacks. Most require surgical implantation, which can affect the measurement of rotation by the inflammation, local hemorrhage, or fibrosis caused by the procedure or the implanted device. All these techniques are limited in the number of left ventricular sites to which they can be applied because beads and strain gauges can be implanted only in surgically accessible sites. Echocardiography, which has been used in dogs\textsuperscript{9} and humans,\textsuperscript{9} is limited to measuring the rotation of a small number of identifiable structures such as the papillary muscles. Few studies have been able to examine differences in rotation across the left ventricular wall. Apart from the limited data available from echocardiography,\textsuperscript{9} these techniques are, of course, inapplicable to normal humans. Detailed study of the rotational motion of the normal human heart, therefore, requires a noninvasive labeling technique that can be applied to any left ventricular site without interfering with the normal pattern of rotation.

The technique of magnetic resonance imaging (MRI) myocardial tagging uses selective radio-frequency saturation of narrow planes before conventional imaging to label myocardium entirely noninvasively.\textsuperscript{10} Tags can be considered magnetic markers and remain visible for 400–500 msec, a time dependent on the relaxation parameters of the tissue being imaged. Tags placed on a short-axis image at end diastole will persist through systole and rotate with the myocardium. The tag position on end-systolic images will indicate the amount and direction of myocardial movement, and from this, the rotation of the left ventricular apex with respect to the base can be measured.

Our aim in this study was to use MRI myocardial tagging to identify whether rotation of the left ventricular apex with respect to the base is a component of systolic function in normal humans, a group not previously studied. If so, we wished to quantify it and identify any variation along the length, around the circumference, or across the thickness of the normal human LV.

\section*{Methods}

\subsection*{Magnetic Resonance Imaging Myocardial Tagging}

Tagging is achieved by selective radio-frequency saturation of thin planes intersecting the myocardium in a pattern predefined by the operator.\textsuperscript{10} In the tagged regions, tissue protons will be in a different state of magnetization than those in nontagged regions. This difference in magnetization persists for a time mainly dependent on the longitudinal relaxation time (T1) of the myocardium, which for magnetic field strengths of 0.3–0.6 T is about 400–500 msec. During that time, a magnetic resonance image acquired orthogonally to the tagging plane will demonstrate a difference in the signal intensity between the tagged and nontagged regions. Additionally, any intervening displacement between the time of tagging and the time of imaging will be recorded as a change in position, shape, or orientation of the tags relative to their initial location.

In practice, a series of four tag planes parallel to a long axis of the LV, their intersection positioned at the center of the cavity, are positioned on the heart at end diastole. Short-axis images are then obtained at end systole. Figure 1 illustrates the relative position of the tagging and imaging planes, with only two tagging planes and one imaging plane shown for simplicity. Using four tag planes, eight locations on the myocardium of each short-axis image are defined at end diastole and imaged at end systole (Figure 2). Because tag lines move with the myocardium through the cardiac cycle, the tag position at end systole will indicate the amount and direction of myocardial movement (Figure 3).

\subsection*{Imaging}

Eight normal human volunteers (five men and three women, aged 24–38 years) were imaged in a 0.38-T iron-core resistive magnet (Resonex, Sunnyvale, California, RX4000). Experiments were performed using a spin-echo sequence modified as above to include tagging, with time to echo (TE) equaling 30 msec and repetition time (TR) equaling the RR interval, using 128 phase and frequency encoding steps, and averaging four excitations. End diastole was defined from the first negative deflection of the R wave of the electrocardiogram (ECG), and end systole was defined by the first high-frequency deflection on a phonographic recording of the second heart sound. Images were acquired throughout 512 cardiac cycles. After positioning the subject in the imager, a series of parallel axial scouting images was acquired,
Intramyocardial portions to apex

3. Short-axis planes centered were these planes. Five parallel slice gaps to space the image, long-axis views on apical estimates. To the left ventricular obtained. From the parallel to through the left ventricular passed septum. Locations 1 and 2 represent anterior wall, 3 and 4 represent lateral wall, 5 and 6 represent posteroseptal wall, and 7 and 8 represent septum. Two tag planes are selected to be equivalent to four-chamber (4 CH) and two-chamber (2 CH) apical views on echocardiography.

so that the orientation of the heart in each subject could be assessed and true short-axis cardiac images obtained. From the axial image that traversed the largest left ventricular cavity, a new image plane that passed through the left ventricular apex and was parallel to the septum was selected using visual estimates. This defined a left ventricular long axis. Five parallel short-axis planes, perpendicular to the long-axis image, were selected, adjusting the inter-slice gap to space the planes along the entire LV. These planes were imaged using a multislice sequence. The orientation of four equiangular tag planes centered at the center of the left ventricular cavity were then obtained (Figure 2). One plane was chosen to be equivalent to the echocardiographic apical four-chamber projection. As the four tag planes are equiangular, the positions of the other tags are, therefore, standardized from patient to patient. After this series of scouts, images of the five left ventricular short-axis planes with 4-mm-wide tags applied at end diastole could then be acquired separately. Note that although tags are applied at end diastole, no images are obtained at this time.

Analysis

The four tags intersected the myocardium at eight locations. A tag point is defined as the point where a tag crosses the epicardium or endocardium. Therefore, on each image, there are eight epicardial and eight endocardial tag points (numbered in Figure 2). The center of these points was visually estimated, and the coordinates of the center point were obtained using an on-line arbitrary coordinate system, part of the Resonex RX4000 data analysis program. Considering the epicardial and endocardial points separately, the centroid was calculated, and the slopes of the lines connecting each tag point and the centroid were calculated and expressed as angles. This was done for the epicardial and endocardial points of all five end-systolic images.

The displacement of each tag point on the systolic image from its original position depends on all the movements of the LV during systole, such as translation and rigid rotation, and includes torsion, the rotation of short-axis planes with respect to each other. Translation and rigid rotation will affect all parts of the heart equally. Only end-systolic images have been acquired, with each location equally affected by these movements. To subtract motion

Figure 2. Diagram showing short-axis section of the left ventricle, viewed from apex looking toward base, showing numbering of locations where tag planes intersect myocardium. Locations 1 and 2 represent anterior wall, 3 and 4 represent lateral wall, 5 and 6 represent posteroseptal wall, and 7 and 8 represent septum. Two tag planes are selected to be equivalent to four-chamber (4 CH) and two-chamber (2 CH) apical views on echocardiography.

Figure 3. Short-axis images of basal slice (left panel) and apical slice (right panel) of left ventricle at end systole looking from apex to base with tags applied at end diastole. Images were acquired over 512 cardiac cycles. There is counterclockwise rotation of intramyocardial portions of tags on apical slice as compared with basal slice.
because of translation and rigid rotation of the whole heart, the end-systolic image of the basal plane was used as a reference. The difference in the slope of the line connecting a tag point on the basal plane with its centroid and the line connecting the corresponding tag point on a succeeding plane with its centroid was calculated. The arctangent of this difference in slopes was calculated to derive an angle, called the torsion angle ($\Delta \Theta$) (Figure 4). This angle is the difference at end systole in the position of a basal tag point and the corresponding tag point on a succeeding slice expressed as an angle of rotation. Because there was no difference in the relative positions of these two points at end diastole, the tags being inserted as a plane in both slices simultaneously, this angle represents the rotation of one point with respect to the other point. Increasing angle (positive sign) indicates counterclockwise rotation of the apex when viewed looking toward the base.

**Definitions**

Regional torsion angle ($\Delta \Theta$) is the angle calculated as described in the preceding paragraph for every epicardial and endocardial tag point individually. Mean epicardial or endocardial torsion angle of a slice is the average of the eight values of regional torsion angle for the epicardium or endocardium. Circumferential-longitudinal shear ($\text{shear}_{CL}$) is the difference between the systolic position of a tag point and the equivalent tag point on the basal slice expressed as an angle of which the vertex is the tag point on the basal slice ($\gamma$) (Figure 4). Mean epicardial or endocardial circumferential-longitudinal shear (mean $\text{shear}_{CL}$) is the average of the eight values of epicardial or endocardial $\text{shear}_{CL}$ for the eight positions in any short-axis slice.

Assume that a point on the basal plane rotates by $\Theta_b$ and the equivalent point on plane $i$ rotates by $\Theta_i$. The rotation of that point on plane $i$, relative to the base (torsion angle, $\Delta \Theta$), is given by the following equation: $\Delta \Theta = \Theta_i - \Theta_b$. $\text{shear}_{CL}$ ($\gamma$) is calculated from the torsion angle by the relation:

$$\sin(\Delta \Theta/2) = \frac{x}{2r}$$

(1)

where $r$ is the radius of the myocardial border in question (epicardium or endocardium), $x$ is the linear displacement of the tag, and

$$\tan \gamma = \frac{x}{h}$$

(2)

where $h$ is the distance between the basal slice and the succeeding one. Therefore,

$$\gamma = \tan^{-1}[\frac{2r \sin(\Delta \Theta/2)}{h}]$$

(3)

where $r$ is calculated from the area obtained by contouring the epicardial or endocardial boundary of each slice, using an on-line contouring system (part of the Resonex RX4000 acquisition and analysis program) and assuming that area to be a circle; $h$ is calculated from the imaging plane thickness (1 cm in all cases) and the gap between planes (0.3–0.5 cm, varying between subjects so that the entire LV is included, but constant within any one subject). Figure 4 illustrates the calculation of $\text{shear}_{CL}$ from torsion for one tag point. The same calculation was performed for all epicardial and endocardial points. We have, thus, measured an angle that we have called the torsion angle and, from this, have calculated the $\text{shear}_{CL}$.

**Reproducibility of Analysis**

To assess the reproducibility of the method of image analysis used in this study, two images from each patient (slices 2 and 4) were analyzed twice by the same observer and by a second observer. The difference in the slopes of the lines connecting tag points to the centroid, converted to angles, between the two analyses of the first observer and between the analyses of the first and second observer was calculated to quantify the intraobserver and interobserver variability of the technique of analysis.

**Statistical Analysis**

The significance of variation in torsion and $\text{shear}_{CL}$, both between planes and at different locations within planes, was determined by analysis of variance (ANOVA). The difference between epicardial and endocardial torsion and $\text{shear}_{CL}$ was also determined by ANOVA. Values are expressed as mean±SEM.

**Results**

**Torsion Angle**

There was systolic counterclockwise rotation of the epicardium and endocardium of succeeding planes
TABLE 1. Epicardial and Endocardial Torsion Angles Relative to the Left Ventricular Base

<table>
<thead>
<tr>
<th>Location</th>
<th>Base</th>
<th>2</th>
<th>3</th>
<th>4</th>
<th>Apex</th>
</tr>
</thead>
<tbody>
<tr>
<td>Epicardium (°)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>1</td>
<td>0</td>
<td>1.2±1.9</td>
<td>5.2±2.1</td>
<td>6.7±1.9</td>
<td>12.3±3.4</td>
</tr>
<tr>
<td>2</td>
<td>0</td>
<td>3.6±2.6</td>
<td>7.4±2.6</td>
<td>11.9±2.6</td>
<td>15.1±3.4</td>
</tr>
<tr>
<td>3</td>
<td>0</td>
<td>2.1±1.4</td>
<td>2.3±1.8</td>
<td>3.7±1.7</td>
<td>16.0±3.4</td>
</tr>
<tr>
<td>4</td>
<td>0</td>
<td>3.1±2.0</td>
<td>1.0±2.1</td>
<td>6.9±2.0</td>
<td>14.2±3.5</td>
</tr>
<tr>
<td>5</td>
<td>0</td>
<td>3.1±1.3</td>
<td>-0.4±2.3</td>
<td>7.3±2.8</td>
<td>7.0±2.8</td>
</tr>
<tr>
<td>6</td>
<td>0</td>
<td>-1.3±1.6</td>
<td>4.2±3.5</td>
<td>5.0±2.2</td>
<td>5.7±3.2</td>
</tr>
<tr>
<td>7</td>
<td>0</td>
<td>2.2±1.5</td>
<td>4.1±1.9</td>
<td>8.6±2.0</td>
<td>10.1±2.4</td>
</tr>
<tr>
<td>8</td>
<td>0</td>
<td>2.0±1.8</td>
<td>6.0±1.9</td>
<td>6.7±1.8</td>
<td>8.8±2.5</td>
</tr>
<tr>
<td>Mean</td>
<td></td>
<td>2.0±0.5</td>
<td>3.7±0.8</td>
<td>7.1±0.8</td>
<td>11.2±1.3</td>
</tr>
<tr>
<td>Endocardium (°)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>1</td>
<td>0</td>
<td>3.4±1.8</td>
<td>11.0±2.2</td>
<td>13.9±3.2</td>
<td>27.7±7.4</td>
</tr>
<tr>
<td>2</td>
<td>0</td>
<td>6.5±4.4</td>
<td>14.1±3.0</td>
<td>21.0±3.5</td>
<td>25.2±3.3</td>
</tr>
<tr>
<td>3</td>
<td>0</td>
<td>3.1±2.1</td>
<td>1.5±2.5</td>
<td>8.9±2.6</td>
<td>18.8±2.5</td>
</tr>
<tr>
<td>4</td>
<td>0</td>
<td>3.7±1.8</td>
<td>-3.7±3.3</td>
<td>10.6±3.3</td>
<td>11.5±4.3</td>
</tr>
<tr>
<td>5</td>
<td>0</td>
<td>-0.5±1.5</td>
<td>3.2±4.7</td>
<td>10.8±4.2</td>
<td>12.6±2.0</td>
</tr>
<tr>
<td>6</td>
<td>0</td>
<td>3.5±2.2</td>
<td>5.5±1.6</td>
<td>11.0±2.9</td>
<td>13.2±2.5</td>
</tr>
<tr>
<td>7</td>
<td>0</td>
<td>4.4±2.0</td>
<td>12.0±3.1</td>
<td>17.9±2.7</td>
<td>22.1±5.7</td>
</tr>
<tr>
<td>8</td>
<td>0</td>
<td>8.9±1.8</td>
<td>12.5±2.3</td>
<td>16.9±2.0</td>
<td>21.5±3.2</td>
</tr>
<tr>
<td>Mean</td>
<td></td>
<td>4.1±0.9</td>
<td>7.0±2.1</td>
<td>13.9±1.4</td>
<td>19.1±2.0</td>
</tr>
</tbody>
</table>

n=8 and mean±SEM, for both epicardium and endocardium. Variation in torsion is significant both between slices (p<0.001 for epicardium and endocardium) and between locations (p<0.04 for epicardium, and p<0.001 for endocardium) (ANOVA).

with respect to the base, which increased with increasing distance from the base (Table 1). Mean torsion angles of the epicardium (Figure 5) for slices 2–5 were 2.0±0.5°, 3.7±0.8°, 7.1±0.8°, and 11.2±1.3°, respectively (p<0.001). Mean torsion angles of the endocardium (Figure 5) for the same slices were 4.1±0.9°, 7.0±2.1°, 13.9±1.4°, and 19.1±2.0°, respectively (p<0.001). Thus, the torsion angle is maximal at the apex.

Mean torsion angle of the endocardium was greater than that of the epicardium by approximately twofold (Figure 5) (p<0.001). Regional differences in torsion were seen, with smaller torsion angles in posteroseptal regions than in any other region around the circumference of the LV (Table 1 and Figure 6). These are positions 4–6 in the endocardium, which averaged 12.4±2.9° as compared with 23.1±4.4° in the other regions, that is, 1, 2, 3, 7, and 8, and positions 5 and 6 in the epicardium, 6.4±3.3° versus 12.8±3.1°. ANOVA showed the regional differences in torsion to be significant for both the endocardium (p<0.001) and epicardium (p<0.04).

The epicardial torsion angle does not increase in a uniform manner along the long axis of the ventricle but increases more toward the apex (Figure 5). The epicardial torsion angle was 3.7±0.9° between the base and the center plane (level 3) and 7.4±1.4° between the center plane and the apex (p<0.05). The corresponding values for the endocardium were 7.0±2.1° and for the endocardium, 12.0±1.3° (p<0.07).

Circumferential-Longitudinal Shear

There was shearCL of both the epicardium and endocardium (Table 2). The amount of shearCL was constant irrespective of the distance of a slice from the base. Mean shearCL of the epicardium (Figure 7) was 4.4±1.2°, 4.5±1.0°, 5.0±0.6°, and 5.0±0.6° for slices 2–5, respectively (differences not significant). Mean shearCL of the endocardium (Figure 7) was 5.2±1.2°, 3.8±1.2°, 4.6±0.5°, and 4.0±0.5° for the same slices (differences not significant).

ShearCL did not increase as the apex was approached, nor was there a difference between epicardium and endocardium. The regional variation of torsion angle...
within one left ventricular slice also occurs in \( \text{shear}_{\text{CL}} \). This is because \( \text{shear}_{\text{CL}} \) is calculated from the torsion angle using the radius of the slice and the distance it is from the base, which is the same for all the epicardial and all the endocardial points of that slice. (Note that no regional variation in radii was present.)

**Reproducibility of Analysis**

*Intraobserver variability.* Comparison of the slopes of the lines connecting tag points to the centroid, converted to angles, obtained from the two analyses of the first observer gave a correlation coefficient \( (r) \) of 0.99 and SEE of 1.0° for the epicardium, and an \( r \) of 0.99 and SEE of 2.2° for the endocardium. The mean differences \( (\pm \text{SD}) \) in the angles obtained were 0.82±0.7° for the epicardium and 1.75±1.42° for the endocardium.

*Interobserver variability.* Comparison of the slopes of the lines connecting tag points to the centroid, expressed as angles, obtained from the analyses of the first and the second observers gave an \( r \) of 0.99 and SEE of 1.9° for the epicardium, and an \( r \) of 0.99 and SEE of 2.2° for the endocardium. The mean differences \( (\pm \text{SD}) \) in the angles obtained were 1.57±1.28° for the epicardium and 2.23±1.83° for the endocardium between the analyses of the first and second observer.

**Discussion**

In this study, left ventricular torsion and \( \text{shear}_{\text{CL}} \) are demonstrated and quantified in the intact human using a technique for noninvasively labeling myocardium. Mathematical models of left ventricular contraction have suggested that \( \text{shear}_{\text{CL}} \) may be an important component of systolic function\(^3,4\) because it allows fiber shortening and, therefore, fiber stress to be more homogeneous across the ventricular wall. Although invasive studies in dogs and patients have suggested that these deformations do occur, it has not been possible until now to show whether they play a role in the normal human heart.

**Torsion**

Torsion of the left ventricle is a component of normal systolic function. It is of the same order, in the range of 5.7–27.7°, and in the same direction, counterclockwise torsion of the apex when viewed looking toward the base, as predicted by previous invasive studies.\(^7,8\) The ability of tagging to label both epicardium and endocardium and multiple ventricular sites, however, has enabled us to measure torsion both across and around the LV wall and to demonstrate its pattern more comprehensively.

**Table 2. Conversion of Mean Torsion to Circumferential-Longitudinal Shear**

<table>
<thead>
<tr>
<th>Slice</th>
<th>Base</th>
<th>2</th>
<th>3</th>
<th>4</th>
<th>Apex</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Epicardium</strong></td>
<td>Mean torsion angle (°)</td>
<td>0</td>
<td>2.0±0.5</td>
<td>3.7±0.8</td>
<td>7.1±0.8</td>
</tr>
<tr>
<td></td>
<td>Distance from base, ( h ) (cm)</td>
<td>...</td>
<td>1.4±0.1</td>
<td>2.8±0.2</td>
<td>4.2±0.3</td>
</tr>
<tr>
<td></td>
<td>Radius, ( r ) (cm)</td>
<td>...</td>
<td>3.3±0.2</td>
<td>3.3±0.3</td>
<td>3.0±0.3</td>
</tr>
<tr>
<td></td>
<td>( \text{shear}_{\text{CL}} ) (°)</td>
<td>0</td>
<td>4.4±1.2</td>
<td>4.5±1.0</td>
<td>5.0±0.6</td>
</tr>
<tr>
<td><strong>Endocardium</strong></td>
<td>Mean torsion angle (°)</td>
<td>0</td>
<td>4.1±0.9</td>
<td>7.0±2.1</td>
<td>13.9±1.4</td>
</tr>
<tr>
<td></td>
<td>Distance from base, ( h ) (cm)</td>
<td>...</td>
<td>1.4±0.1</td>
<td>2.8±0.2</td>
<td>4.2±0.3</td>
</tr>
<tr>
<td></td>
<td>Radius, ( r ) (cm)</td>
<td>...</td>
<td>1.8±0.2</td>
<td>1.6±0.2</td>
<td>1.4±0.2</td>
</tr>
<tr>
<td></td>
<td>( \text{shear}_{\text{CL}} ) (°)</td>
<td>0</td>
<td>5.2±1.2</td>
<td>3.8±1.2</td>
<td>4.6±0.5</td>
</tr>
</tbody>
</table>

Variation in \( \text{shear}_{\text{CL}} \) from slice to slice, for both epicardium and endocardium, was not significant.
Torsion of the endocardium is greater than that of the epicardium by approximately twofold. This suggests that shear occurs between the epicardium and endocardium (circumferential-radial shear), a strain previously detected in animal models but not in humans.

Less torsion occurs in posterior and posteroseptal regions than in anterior and anterolateral regions (Figure 6). The cause of this regional variation is unknown but could relate to local differences in the sequence of depolarization or the overall extent of thickening, two aspects of left ventricular function that might affect torsion. Torsion does not increase linearly from base to apex. The amount of torsion between the base and the center slice (i.e., slice 3), is less than between the center slice and the apex. This difference was significant for the epicardium and approached significance for the endocardium. The predominant fiber direction in the basal portion of the LV is circular but this layer diminishes towards the apex, where most fibers are oblique. The increasing proportion of oblique fibers at the apex might explain this nonuniformity of torsion.

**Torsion Versus Shear**

Shear is one of the shearing strains of the LV that occurs during systole and relates the displacement of a point to the corresponding point at the base (see Figure 4). The noninvasive measurement of torsion angle combined with the precise spatial and geometrical information provided by MRI has allowed calculation of shear in humans for the first time. Despite the differences in torsion from base to apex and between epicardium and endocardium, these areas are subject to similar amounts of mean shear, that is, the normally functioning LV maintains constant mean shear both across the ventricular wall and from apex to base. From the formula for converting torsion to shear (Equation 3), shear of a point is dependent on the torsion angle, the distance between the point and the center of the left ventricular cavity (r), and the distance between the point and the base (h). Constancy of mean shear is maintained in the LV by variation in the torsion angle of a point to compensate for its h or r (Figure 8). Thus, torsion increases as h increases and as r decreases. Also, the nonlinear increase in torsion, with greater increments between slices at the apex than at the base, might be because of the tapering of the LV at the apex. This constancy of shear might represent an important principle by which stress through and along the left ventricular wall is equalized during normal ejection.

The mechanism that produces shear is not known. It has been suggested that an imbalance between fibers with lefthand and righthand obliquity might produce torsion and shear. Other LV deformations, however, have little relation to the local fiber arrangement. Waldman et al found uniform orientation of the direction of greatest shortening at all left ventricular wall depths despite great variation in fiber angle. The direction of greatest shortening corresponded to the fiber direction in the epicardium; however, large strains perpendicular to the fiber direction were seen in the endocardium. They suggested that myofiber rearrangement or shape change, or the extensive collagen network of the interstitium, might be responsible for this. Ingels et al suggested that the transmural twist is in the direction of the epicardial rather than endocardial fibers because epicardial fibers are further from the center of the LV and, thus, have a mechanical advantage.

**Comparison With Previous Studies**

Previous studies of torsion and shear have produced results compatible with ours. Arts et al measured shear in the dog noninvasively by echocardiography and found it to be about 7°, similar to the values we have obtained. Using a technique by...
which radiopaque markers were implanted in the midwall of hearts at the time of cardiac surgery for transplantation, Ingels et al\textsuperscript{7} and Hansen et al\textsuperscript{8} described counterclockwise torsion of 13.3–23.4°. These values lie between and, hence, are consistent with the values of epicardial and endocardial torsion we have described.

We have shown that average shear\textsubscript{CL} of each short-axis plane is constant across the left ventricular wall because of the difference in the amount of torsion of the epicardium and endocardium compensating for their different radii. This difference in torsion results from shearing between the endocardium and the epicardium. Although many previous studies have only examined one wall depth, some have been able to address this point. Waldman et al\textsuperscript{2} used multiple beads across the ventricular wall in dogs, whereas Osakada et al\textsuperscript{15} used a triangulation method with one subendocardial and three epicardial sonomicrometers, and Feigl and Fry\textsuperscript{8} used a strain gauge that could be placed at different depths within the myocardium. All have shown that transverse shearing forces are greater in deeper layers of myocardium. All three, however, could only examine small areas of left ventricular free wall. Further studies using myocardial tagging will enable us to identify and measure any transmural differences in shearing strain at all left ventricular sites.

We have shown both torsion and shear\textsubscript{CL} to be least in posteroseptal regions; however, few of the invasive studies of torsion have identified this. Indeed, our findings are at variance with the one previous study addressing this issue, which involved transplanted hearts in which the least torsion was in anterior areas and nowhere was as small as we have shown in posterior regions.\textsuperscript{8} This difference has two possible explanations. First, the previous study implanted the markers in the midwall. We have shown endocardial torsion to be greater than epicardial, and therefore, if markers were not implanted precisely at the midwall, differences because of depth in the wall and not position could be seen. Second, the effect of surgery, postoperative fibrosis, the ischemic period, and bouts of rejection on the normal dynamics of these hearts is uncertain. Hansen et al\textsuperscript{8} used a floating coordinate system to prevent motions that affect the whole LV from corrupting the results. For the same reason, we compared the systolic tag positions not with their diastolic positions but with the systolic position of the equivalent point on the base because motions affecting the whole heart will affect both points. Both these methods succeed in eliminating bias because of motions affecting the whole LV, and this does not explain the difference between the results of Hansen et al\textsuperscript{8} and those of ourselves.

Most previous studies of torsion and shear\textsubscript{CL} have used implanted strain gauges, sonomicrometers, or radiopaque markers in dogs in a variety of experimental conditions. Torsion has been studied in patients after cardiac surgery or transplantation, and only pure rotation has been demonstrated in normal subjects.\textsuperscript{9} Results obtained can be affected by 1) the surgical procedure, 2) the presence of the implanted object, 3) edema or fibrosis caused by the implanted object, or 4) in the human studies, the effect of the underlying cardiac abnormality. Tagging, being non-invasive and, thus, applicable to normals, does not share these problems. Also, implantation techniques are limited to the number and location of sites in which they can be used, the septum being relatively inaccessible and the number of devices that can be implanted at one time, restricted. Tagging allows examination of all locations in all left ventricular levels during the one experimental series.

**Limitations**

A limitation of this study is that torsion and shear\textsubscript{CL} have been measured at only one time point, that is, end systole. We were, thus, not able to plot the time course of the development of torsion and shear\textsubscript{CL} or, indeed, confirm that they are maximum at end systole. The combination of tags with an acquisition technique involving gradient-recalled echoes would allow measurement of torsion and shear\textsubscript{CL} at multiple points through the cardiac cycle.

There are several errors that could potentially affect the accuracy of the coordinates of the tag points. The thickness of the tag (4 mm) can make the center difficult to identify. Respiratory motion and beat-to-beat variation in the cardiac cycle length can cause blurring of the image although, in practice, are a minor problem. Because of the thickness of the imaging slice (1 cm), some torsion will occur from top to bottom, and the tag will intersect the slice slightly obliquely, making the border between the tag and the myocardium less clear. To assess the effect of these factors on the results, interobserver and intraobserver variability of analysis was calculated. This shows that although analysis errors are small (1–2°), they are large enough to affect the smallest of our measured angles, that is, the torsion for slice 2. While we therefore cannot be confident of these smallest angles, they do fit the pattern produced by the other slices in which the angles measured are large enough to be unaffected by these potential errors.

Torsion and shear\textsubscript{CL} are integral parts of normal systolic function and are probably a way of increasing the efficiency of transmural myocardial function by equalizing fiber stress and function across the left ventricular wall.\textsuperscript{3,4} This might, therefore, increase the efficiency of energy use by the myocardium and lead to more efficient ejection of blood during systole. Constancy of mean shear\textsubscript{CL} is effected by variation in torsion at different left ventricular sites to compensate for differing distances from the base and left ventricular radii. It can be hypothesized that disruption of shear\textsubscript{CL} might compromise normal ejection and alter the normal pattern of wall stress and energy demand. The study of left ventricular torsion and shear\textsubscript{CL} can greatly increase our understanding of disease states that affect left ventricular function, and MRI tagging is an excellent tool for this purpose.
References


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