Regional assessment of wall curvature and wall stress in left ventricle with magnetic resonance imaging

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Balzer, Philippe, Alain Furber, Stéphane Delépine, Frédéric Rouleau, Franck Lethimonnier, Olivier Morel, André Tadéi, Pierre J allet, Philippe Geslin, and Jean-Jacques Le Jeune. Regional assessment of wall curvature and wall stress in left ventricle with magnetic resonance imaging. Am. J. Physiol. 277 (Heart Circ. Physiol. 46): H901–H910, 1999.—Left ventricular functional abnormalities are associated with regional increases of wall stress and modifications of wall curvature. This study describes the integration of the short-axis and long-axis wall curvatures for determining peak systolic wall stress. Quantification was realized with cine magnetic resonance imaging (MRI) from the location of the endocardial and epicardial borders of the left ventricle on pairs of consecutive short-axis sections. Fifteen normal volunteers were subjected to cine MRI, and different methods of calculating peak systolic wall stress were compared. A short-axis analysis showed a 55 ± 13% increase of the circumferential mean of the peak systolic wall stress between apical and basal sections. Regarding the curvature, no significant increase of wall stress was observed except on the septal wall (31 ± 18%). Short-axis studies proved to be insufficient for determining the regional variations of left ventricular wall stress and for providing normal reference values for the location of abnormal regions in patients.

Myocardial function; systolic wall stress; wall thickening; cine magnetic resonance imaging

MONITORING OF LEFT VENTRICULAR diseases requires accurate evaluation of anatomic parameters (volume, mass, wall thickness) and functional parameters (ejection fraction, wall thickening, endocardial motion, wall stress, etc.). However, identification and assessment of abnormalities of the left ventricular structures and contractility require knowledge of normal references as well as regional measurements.

The spatial and temporal resolution of the images, the contrast between tissues and blood, the absence of geometric assumptions, and the precise definition of the endocardial and epicardial borders justify the use of short-axis magnetic resonance imaging (MRI) for following up the cardiac remodeling induced by myocardial infarction or hypertension. Previous works established the reliability of a noninvasive functional study of the left ventricle with MRI in comparison with echocardiography (7, 19, 29), ventriculography (18), angiography (32), and indicator-dilution methods (10). Mass assessment, which requires location of the endocardial and epicardial borders, is well correlated with ex vivo measurements (33). The association of MRI with an automated image processing software ensures the adequacy of this examination with regard to clinical routine (17), and interobserver variability is compatible with observation of the expected functional modifications caused by cardiac disease (9, 11).

In comparison with long-axis studies (20), sets of short-axis sections are independent of geometric assumptions and minimize the partial volume effects. However, a short-axis section toward the apex of the left ventricle is no longer perpendicular to the wall. A planar short-axis analysis of the left ventricular structures consequently leads to erroneous estimation of the radius and the wall thickness. These errors are proportional to the curvature of the wall.

The heterogeneity of left ventricular function between the apex and the base, as well as between the endocardial and epicardial borders, was studied by echocardiography (3, 35) and, more recently, by tagging of magnetic resonance images (12, 37). These observations are in agreement with physiological works in which authors observed regional variations in the morphology and orientation of the myocardial fibers (21). The internal structure of the myocardium affects ventricular contractility and the equilibrium between wall resistance and blood pressure. This aspect of left ventricular function is demonstrated by the peak systolic wall stress given by the product of the peak systolic blood pressure with a geometric factor (1, 22, 26). In previous studies, the systolic wall stress was mostly determined by contrast ventriculography or echocardiography. However, the technical limitations of these methods do not permit precise regional measurements of radius and wall thickness as functions of the three-dimensional wall curvature.

The aim of this study was to assess the regional variations of wall stress in the normal left ventricle when the wall curvature is taken into account. This curvature was assessed in the short-axis plane as well as in the long-axis plane.

MATERIALS AND METHODS

Study Group

The study population used to define normal left ventricular function consisted of 15 healthy volunteer subjects (6 women and 9 men) with no history or physical finding of cardiac or pulmonary disease. All subjects gave informed consent. General data concerning this population are given in Table 1. Five
noninvasive measurements of systolic and diastolic blood pressures were recorded and averaged at the time of the MRI examination.

Imaging Technique

All volunteers were studied with a 1.5-T imager (Signa Horizon release 5.7, GE Medical Systems, Milwaukee, WI). The subjects were placed in a supine position with a phased-array coil (Torso coil). A fast-gradient echo segmented k-space sequence with radio frequency phase spoiling was used with electrocardiogram gating. Scout transversal and sagittal views ensured correct determination of the short-axis plane of the left ventricle. Each section was then acquired in a single breath hold (20–25 s) with 9–21 temporal phases per heart-beat using view sharing and uniform repetition time radio frequency excitation. Interleaved images were obtained in 8–12 planes from the apex to the base with the following parameters: 10-mm section thickness, no gap between sections, 320-mm field of view, partial echo, echo time 2.7 ms, repetition time 10.2 ms, receiver bandwidth 15.6 kHz, flip angle 30°, eight views per segment, 256 × 128 matrix, and one excitation. The total study time averaged 30 min.

Image Analysis

For analysis and computation, the magnetic resonance images were transferred to a multimodality station (HP 715–50, Hewlett-Packard, Palo Alto, CA) with a UNIX environment. Endocardial and epicardial borders of the left ventricle were drawn with an automatic segmentation method previously validated in animal subjects and patients (4). In brief, this technique is an integrated approach to segmentation by region growing, edge detection, and adaptative thresholding. The main steps are 1) a thresholding to isolate the blood pools; 2) a region-growing process to approximate the surface of the left ventricular wall; 3) construction of the epicardial borders with a gradient map and a set of a priori information; and 4) refinement of the contours in areas of slow blood flow or papillary muscles with an adaptative thresholding.

The tracing of the left ventricular borders was then controlled and possibly modified by a trained clinical observer through an interactive interface. For measurement of the wall thickness and radius of the ventricular cavity, the papillary muscles were smoothed out of the endocardial contour.

Computation of Regional Left Ventricular Function

The multisection images at the diastolic and systolic phases were determined by locating the largest and smallest areas of the left ventricular cavity on a midventricular short-axis plane. On all sections, the centroid of the left ventricle was located as the mass center of the median line between the endocardial and epicardial borders. Each short-axis section was centered on the mean position of the ventricular centroid during the cardiac cycle. The parameters of the regional left ventricular function (wall thickening, endocardial motion, peak systolic wall stress) were studied on a set of five contiguous short-axis planes (Fig. 1). For all volunteers, the set encompassed sections with closed and clearly defined endocardial and epicardial borders. Apical planes with no ventricular cavity and valvular planes with an open chamber were consequently excluded from the analysis.

Calculation of the wall curvature relies on a polar transformation of the image. This process has been widely used for segmentation algorithms (15), and it allows a radial study of the left ventricle. With a centerline approach (34), the wall curvature from base to apex is assessed on 128 radii and the left ventricular radius and wall thickness are deduced from

Table 1. Study group of normal subjects

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Mean ± SD</th>
<th>Range</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age, yr</td>
<td>28 ± 7</td>
<td>20–41</td>
</tr>
<tr>
<td>Weight, kg</td>
<td>69 ± 8</td>
<td>54–82</td>
</tr>
<tr>
<td>Height, cm</td>
<td>175 ± 7</td>
<td>164–189</td>
</tr>
<tr>
<td>End-diastolic blood pressure, mmHg</td>
<td>77 ± 7</td>
<td>70–85</td>
</tr>
<tr>
<td>End-systolic blood pressure, mmHg</td>
<td>130 ± 10</td>
<td>120–150</td>
</tr>
</tbody>
</table>

Fig. 1. Set of 5 adjacent short-axis magnetic resonance images of left ventricle at diastolic (top) and systolic (bottom) phases. For functional study of each subject, set was centered on midventricular plane (section 3), which was defined as level of papillary muscle insertions. From left to right, sections 1 and 2 were short-axis planes located 2 and 1 cm away from section 3 toward apex, respectively, and sections 4 and 5 were 1 and 2 cm away from section 3 toward base, respectively.
the geometry of the ventricle on two consecutive short-axis sections (Ref. 5; Fig. 2). The main steps of the method are summarized in the Appendix. Wall thickening and peak systolic wall stress were subsequently measured on four adjacent levels and were averaged in the anterior, lateral, inferior, and septal sectors (Fig. 3).

For each subject, the complete analysis, including edge detection and quantitative analysis, required 15 ± 5 min.

Regional ejection fraction. Diastolic volume and systolic volume were measured inside the anterior, lateral, inferior, and septal sectors. Regional ejection fractions were derived from the corresponding volumes.

Regional wall thickening and endocardial motion. Wall thickening (WT) was calculated at each level and for each sector with the following formula

\[ WT = \frac{EST - EDT}{EDT} \]

where EST and EDT are WT at end systole and end diastole, respectively.

Three-dimensional wall thickening was given by the same ratio when the wall thickness accounted for spatial curvature. Endocardial motion was defined as the variation in millimeters of the radius of the left ventricular cavity during the cardiac cycle.

Regional peak systolic wall stress. The wall stress is given by the equilibrium of forces between the left ventricular cavity and the wall. The regional peak systolic wall stress (WS) is determined, following Grossman (see Refs. 22, 28, 32), from knowledge of the inner radius (R) and wall thickness (T) at end systole

\[ WS = 0.133 \times SP \times \frac{R}{2T \times \left(1 + \frac{T}{2R}\right)} \]

where SP is peak systolic ventricular blood pressure in millimeters of mercury. In this study, SP was assessed by the systolic noninvasive blood pressure (31); 0.133 is a conversion factor to express the final results in \(10^3 \text{N/m}^2\). Peak systolic wall stress was also calculated with the radius and the wall thickness defined with the three-dimensional curvature.

Other studies (13, 16) of left ventricular wall stress in the short-axis plane with MRI or echocardiography were based on the Janz method of wall stress assessment (26). This approach relies on the measurement of the areas \(A_c\) and \(A_w\) of the blood pool and the ventricular wall, respectively. The peak systolic wall stress (AWS) in \(10^3 \text{N/m}^2\) was given by

\[ AWS = 0.133 \times SP \times \frac{A_w}{A_c} \]

Data Processing and Statistical Analysis

Automatic drawing of the ventricular borders was independently supervised and corrected by three observers to assess the interobserver variability of the functional and anatomic measurements (mass, diastolic volume, systolic volume, ejection fraction, diastolic thickness, wall thickening, endocardial motion, and wall stress). The optimal evaluation of the parameters was taken as the mean of the values obtained.
with the three observers. For determination of intraobserver variability, one of the observers controlled the tracing on two occasions, 1 mo apart. Intra- and interobserver variabilities were quantified by linear regression analysis and calculation of the standard error of estimation (SEE).

The mean value of the wall stress for each short-axis level and each sector was determined with the different formulations given in Regional peak systolic wall stress. The significance of differences between short-axis levels and sectors was evaluated first by analysis of variance. If there was a significant interaction \( P < 0.05 \) between multiple measurements, further examination of selected pairwise comparisons was undertaken. The gradient of the wall stress from the apex to the base was assessed for each formulation by applying Student’s \( t \)-test, SEE, and the correlation coefficient to define the relationship between the different formulations and each sector was determined with the different formulations given in Table 2. A good interobserver variability was observed for the anatomic parameters (mass and volumes) as well as the mean assessments of functional parameters. The interobserver variability of the wall stress measurement was given by a correlation coefficient \( r = 0.94 \) and SEE = 1 g. The wall stress measurements of all observers were highly correlated \( r = 0.93 \). The ratio between SEE (1 g) and the mean was 11%.

Evolution of Peak Systolic Wall Stress From Apex to Base

The circumferential values of the wall stress are given in Fig. 3 to highlight the regional variations of the left ventricular function and in Table 3 to correlate the results obtained with a short-axis analysis and with the calculation of the three-dimensional wall curvature.

A study of the peak systolic wall stress in the short-axis plane revealed a significant gradient between all adjacent levels from the apex to the base (Fig. 4, A and B). Wall thickness and radius of the cavity were first measured in the direction originating from the ventricular centroid. The wall stress (WS) calculated with these data was closely correlated with the wall stress (AWS) assessed with area measurements. The correlation coefficient between WS and AWS was \( r = 0.99 \). In the short-axis plane, the curvature of the two-dimensional middle line averaged 0.10 ± 0.07 rad. The planar wall stress (2DWS) accounting for this two-dimensional curvature was not significantly different from WS.

When wall thickness and radius of the cavity were calculated in the spatial direction perpendicular to the wall, the gradient of the wall stress (3DWS) from the apex to the base was no longer observed (Fig. 4C). The difference between 2DWS and 3DWS was reduced at

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**Table 2. Normal left ventricular function**

<table>
<thead>
<tr>
<th>Functional Parameter</th>
<th>Measurement by Observer 1</th>
<th>Measurement by Observer 2</th>
<th>Measurement by Observer 3</th>
<th>Mean Measurement</th>
<th>Standard Error of Estimation</th>
<th>Correlation Coefficient</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mass, g</td>
<td>119 ± 29</td>
<td>120 ± 31</td>
<td>120 ± 31</td>
<td>120 ± 31</td>
<td>7</td>
<td>0.97</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Mass index, g/m²</td>
<td>65 ± 13</td>
<td>66 ± 14</td>
<td>65 ± 14</td>
<td>65 ± 13</td>
<td>4</td>
<td>0.96</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>End-diastolic volume, ml</td>
<td>73 ± 15</td>
<td>79 ± 17</td>
<td>70 ± 14</td>
<td>74 ± 16</td>
<td>7</td>
<td>0.89</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>End-diastolic volume index, ml/m²</td>
<td>40 ± 6</td>
<td>44 ± 7</td>
<td>39 ± 6</td>
<td>41 ± 7</td>
<td>4</td>
<td>0.83</td>
<td>0.001</td>
</tr>
<tr>
<td>End-systolic volume, ml</td>
<td>27 ± 10</td>
<td>31 ± 9</td>
<td>28 ± 11</td>
<td>29 ± 11</td>
<td>6</td>
<td>0.83</td>
<td>0.001</td>
</tr>
<tr>
<td>End-systolic volume index, ml/m²</td>
<td>15 ± 5</td>
<td>17 ± 5</td>
<td>15 ± 6</td>
<td>16 ± 5</td>
<td>3</td>
<td>0.82</td>
<td>0.001</td>
</tr>
<tr>
<td>Ejection fraction, %</td>
<td>63 ± 11</td>
<td>60 ± 8</td>
<td>61 ± 11</td>
<td>61 ± 10</td>
<td>9</td>
<td>0.61</td>
<td>0.02</td>
</tr>
<tr>
<td>End-diastolic thickness, mm</td>
<td>12 ± 2</td>
<td>13 ± 2</td>
<td>12 ± 2</td>
<td>12 ± 2</td>
<td>1</td>
<td>0.92</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Wall thickening, %</td>
<td>65 ± 28</td>
<td>63 ± 23</td>
<td>65 ± 27</td>
<td>64 ± 26</td>
<td>16</td>
<td>0.78</td>
<td>0.001</td>
</tr>
<tr>
<td>Endocardial motion, mm</td>
<td>13 ± 4</td>
<td>13 ± 3</td>
<td>14 ± 4</td>
<td>13 ± 4</td>
<td>2</td>
<td>0.86</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Wall stress, N·1,000/m²</td>
<td>9 ± 4</td>
<td>8 ± 3</td>
<td>8 ± 3</td>
<td>9 ± 3</td>
<td>1</td>
<td>0.94</td>
<td>&lt;0.001</td>
</tr>
</tbody>
</table>

Measurement values are means ± SD. *Average value for 4 central short-axis sections. The 3-dimensional curvature is taken into account.
The more basal level (Table 3), when the curvature in the long-axis plane was lower. The ratio between the radius and the wall thickness was highly correlated with 3DWS ($r = 0.98$).

**Evolution of Wall Thickening and Endocardial Motion From Apex to Base**

The wall thickening (WT) determined along the short-axis radial line from the centroid of the left ventricle was constant on the three more apical levels and significantly decreased near the base (Fig. 5A). This evolution was not modified when the wall thickening (3DWT) was given in the direction perpendicular to the wall (Fig. 5B). The comparison between 3DWT and a two-dimensional assessment of the wall thickening (2DWT) accounting for the curvature in the short-axis plane did not reveal significant differences at apical or basal sections (Table 4). The correlation between wall stress and wall thickening in the short-axis plane ($r = -0.58$ between WS and WT) was similar to the correlation obtained with data accounting for the three-dimensional curvature ($r = -0.58$ between 3DWS and 3DWT).

### Table 3. Influence of lateral curvature on assessment of end-systolic wall stress

<table>
<thead>
<tr>
<th>Section</th>
<th>Lateral Curvature, rad</th>
<th>2DWS, N·1,000/m²</th>
<th>3DWS, N·1,000/m²</th>
<th>(3DWS – 2DWS)/3DWS, %</th>
<th>95% CI, N·1,000/m²</th>
<th>SEE, N·1,000/m²</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>1 (apex)</td>
<td>0.55 ± 0.12</td>
<td>3.5 ± 1.7</td>
<td>8.6 ± 5.1</td>
<td>59</td>
<td>[3.0, 7.1]</td>
<td>2.3</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>2</td>
<td>0.40 ± 0.12</td>
<td>5.2 ± 2.3</td>
<td>8.2 ± 2.8</td>
<td>36</td>
<td>[2.0, 4.0]</td>
<td>1.9</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>3</td>
<td>0.32 ± 0.13</td>
<td>6.5 ± 2.5</td>
<td>8.9 ± 3.4</td>
<td>26</td>
<td>[1.3, 3.4]</td>
<td>1.9</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>4 (base)</td>
<td>0.24 ± 0.06</td>
<td>7.5 ± 2.5</td>
<td>9.0 ± 2.7</td>
<td>16</td>
<td>[1.1, 1.9]</td>
<td>0.7</td>
<td>&lt;0.001</td>
</tr>
</tbody>
</table>

Curvature and wall stress values are means ± SD. 2DWS, end-systolic wall stress assessment in short-axis plane; 3DWS, end-systolic wall stress assessment after determination of spatial curvature; 95% CI, confidence interval of (3DWS – 2DWS); SEE, standard error of estimation; P, 2-tailed significance of paired differences.

Fig. 4. Evolution of peak systolic wall stress from apex to base of left ventricle in normal subjects. A: estimation of wall stress WS in short-axis radial direction from center of mass of ventricle. B: estimation of wall stress AWS from measurements of areas of wall and blood pool. C: estimation of wall stress 3DWS accounting for 3-dimensional curvature of wall. Values are means ± SD. P, 2-tailed significance of paired differences. N.S., not significant.

Fig. 5. Evolution of wall thickening from apex to base of left ventricle in normal subjects. A: estimation of wall thickening WT in short-axis radial direction from center of mass of ventricle. B: estimation of wall thickening 3DWT accounting for 3-dimensional curvature of wall. Values are means ± SD. P, 2-tailed significance of paired differences.
No significant evolution of the endocardial motion was observed from the apex to the base. This result was obtained in a short-axis view (mean endocardial motion on 4 sections 12 ± 2 mm) as well as in the direction perpendicular to the wall (13 ± 4 mm). There was no significant difference between methods of calculation.

Regional Quantification of Peak Systolic Wall Stress

No significant variations between the sectorial ejection fraction measurements were observed. They ranged from 58 ± 9 (lateral wall) to 63 ± 11% (septum) and were similar to the global ejection fraction (61 ± 10%).

The gradient of the wall stress for the anterior, lateral, inferior, and septal sectors from the apex to the base are given in Fig. 6. A gradient of 3DWS was observed on the septal wall between sections 1 and 4 (P < 0.01). No gradient of 3DWS was observed on the other sectors. With a short-axis analysis, the mean wall stress on the lateral sector was significantly higher than the wall stress on the septal sector regardless of the method of calculation. When the three-dimensional curvature was considered, the difference between septal and lateral walls was smaller toward the apex and was not significant on section 1. The mean radius of curvature in the short-axis plane ranged from 0.06 ± 0.05 rad on the septal wall to 0.16 ± 0.09 rad. The mean difference between the sectorial evaluations of WS and 3DWS was 3.1 ± 1.8 × 10³ N/m². The ratio (3DWS − WS)/3DWS averaged 36 ± 10% (Fig. 7).

Regional Quantification of Wall Thickening

The method of evaluation of the wall thickening did not affect the regional variations of this parameter (Fig. 8). When the three-dimensional curvature was considered, the difference of wall thickening between lateral and septal walls was 30% for all section levels, 18% for section 1 (apex), 41% for section 2, 39% for section 3, and 23% for section 4 (base). The endocardial motion in the direction perpendicular to the wall averaged 15 ± 5 mm in the anterior sector, 12 ± 4 mm in the lateral sector, 15 ± 5 mm in the inferior sector, and 13 ± 5 mm in the septal sector. No significant differences were recorded between sectors.

DISCUSSION

This work describes the normal regional variations of peak systolic wall stress with a three-dimensional approach provided by breath-hold fast-gradient echo MRI. An extensive analysis of different magnetic resonance methodologies for the assessment of peak systolic wall stress was carried out to define the most reliable calculation.

Table 4. Influence of lateral curvature on assessment of end-systolic wall thickening

<table>
<thead>
<tr>
<th>Section</th>
<th>Lateral Curvature, rad</th>
<th>2DWT, %</th>
<th>3DWT, %</th>
<th>(3DWT − 2DWT)/3DWT, %</th>
<th>95% CI, %</th>
<th>SEE, %</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>1 (apex)</td>
<td>0.55 ± 0.12</td>
<td>61 ± 20</td>
<td>64 ± 30</td>
<td>4</td>
<td>[−4, 9]</td>
<td>9</td>
<td>NS</td>
</tr>
<tr>
<td>2</td>
<td>0.40 ± 0.12</td>
<td>66 ± 27</td>
<td>74 ± 30</td>
<td>10</td>
<td>[3, 12]</td>
<td>8</td>
<td>0.03</td>
</tr>
<tr>
<td>3</td>
<td>0.32 ± 0.13</td>
<td>64 ± 22</td>
<td>67 ± 27</td>
<td>0</td>
<td>[−2, 8]</td>
<td>8</td>
<td>NS</td>
</tr>
<tr>
<td>4 (base)</td>
<td>0.24 ± 0.06</td>
<td>54 ± 18</td>
<td>54 ± 17</td>
<td>1</td>
<td>[−3, 2]</td>
<td>5</td>
<td>NS</td>
</tr>
</tbody>
</table>

Curvature and wall thickening values are means ± SE. 2DWT, end-systolic wall thickening assessment in short-axis plane; 3DWT, end-systolic wall thickening assessment after determination of spatial curvature; 95% CI, confidence interval of (3DWT − 2DWT); P, 2-tailed significance of paired differences; NS, nonsignificant.
Methodology for Measurement of Peak Systolic Wall Stress

Angiography has been widely used for the assessment of peak systolic wall stress. With left ventriculography in the right oblique projection, normal peak systolic wall stress appeared to be greater in basal inferior segments than in anterior segments (23, 30). However, angiography is invasive and requires injection of a contrast agent, the viscosity of which may alter the left ventricular geometry, blood volumes, and wall stress. Additionally, inferior segments of the epicardial borders are not accurately located for all patients. The wall stress values obtained with angiography and transthoracic echocardiography proved to be well correlated (31). Echocardiography is a simpler and widely available examination, but it relies on geometric assumptions. Moreover, the difficulty in acquiring parallel tomographic views implies that the wall stress estimations are provided only at the level of papillary muscle insertions or at the level of the tips of mitral valve leaflets (13). With an ultrafast scanner and a contrast agent, Feiring and Rumberger (14) described an increase of the wall stress from the apex to the base. These results were confirmed by other works based on the two-dimensional analysis of magnetic resonance images (16). The resolution and contrast of magnetic resonance images allows local measurements of wall stress without any geometric assumptions.

Our short-axis analysis of the magnetic resonance images did not reveal any significant differences between the methods of Grossman (see Refs. 22, 28, 32) and Janz (26) for the wall stress calculation. Pouleur et al. (30) previously suggested that the accuracy of Janz’s formulation was inversely proportional to the ventricular area. Lessick et al. (27) compared the methods of Grossman and Janz for determination of the meridional wall stress in patients with aneurysm. Significant differences between results were observed, but the qualitative conclusions were similar.

New Data on Regional Peak Systolic Wall Stress

Our bidimensional study of the short-axis sections indicates that the meridional wall stress increases from apex to base along the long axis of the left ventricle. However, few studies are available to support these observations, and direct measurements of wall stress are poorly related to short-axis calculations (36). Fujita et al. (16) reported lower values of circumferential peak systolic wall stress than in the present study or in the echocardiographic data given by Douglas et al. (13). This difference with the same method of wall stress calculation may be explained by the fact that we used the peak systolic arterial pressure, which is more similar to the peak left ventricular systolic pressure used by Douglas et al. than to the left ventricular peak systolic pressure estimated by Fujita et al. Janicki et al. (25) observed that the average ratio of radius to wall...
thickness \((R/T)\) was homogeneous over the basal half of the left ventricle and was more variable at the lower ventricular levels. This variability was attributed to possible inclusions of extraneous papillary muscles, thus increasing apparent wall thickness. In contrast, we excluded all visible papillary muscles and still found similar variations of \(R/T\).

Peak systolic wall stress is related to wall curvature by the Laplace equation: a large radius of curvature implies a large wall stress and prevents fiber shortening. This further justifies the integration of the curvature in the wall stress calculation. The long-axis curvature averaged 0.55 \pm 0.12 rad on the apical level 2 cm away from the midventricular plane. The wall curvature was important enough to cause a significant underestimation of the wall stress when \(R/T\) was measured in a short-axis direction. The conclusion concerning the axial evolution of the wall stress was consequently modified. The increase of the wall stress observed from the apex to the base with a short-axis analysis was no longer significant when the curvature of the wall was taken into account. In this case, a regional analysis of the images revealed that the peak systolic wall stress increased on the septal wall only (28% between sections 1 and 4). Beyar et al. (6) also noticed in dogs that the gradient of \(R/T\) between apex and base was smaller when the transversal curvature was considered. However, a short-axis analysis did not induce any errors on the regional wall thickening measurements for all short-axis levels.

Limitations of Study

Wall stress is the product of the ventricular peak systolic blood pressure and a geometric factor. An invasive measurement or an indirect calculation of the blood pressure only provides a global value for the whole ventricle. Consequently, regional variations of the wall stress are induced by those of the geometric factor but do not integrate the local changes in pressure. Moreover, the systolic blood pressure was 150 mmHg for two subjects participating in this study, and the subsequent mean value (130 \pm 10 mmHg) was higher than in other studies (31). In one patient, only nine temporal phases per heartbeat were obtained.

Moreover, the evaluation of the wall curvature implies the location of the endocardial and epicardial borders on two consecutive short-axis sections. The reliability of the wall stress measurement depends on the accuracy of the short-axis orientation, the section thickness, and the gap between sections. Two acquisitions in the transversal and sagittal planes were used to precisely define the short-axis view. Each section was then acquired in one breath hold. The limitation of movements between two breath holds was easier to obtain with normal volunteers than with patients. The effect of the axial twist on the determination of the wall stress between two adjacent sections was minimized by averaging the measurements for sectors encompassing 22°. This approach also reduced the influence of the axial rotation during the cardiac cycle on the value of the wall thickening. Moreover, previous studies (see Ref. 8) showed that the axial twist was maximal toward the apex and more negligible at the midventricular and basal levels.

Regional differences in cardiac twist suggest heterogeneous shear forces (24). Peak systolic wall stress may consequently be subjected to longitudinal and shear deformations as well as through-plane motion and cardiac shortening. The identification of anatomic features such as papillary muscles allowed supervision of the pairing of the sections at end diastole and end systole.

Clinical Applications

Knowledge of normal values of regional peak systolic wall stress in the left ventricle allows identification and follow-up of local functional abnormalities. Peak systolic wall stress is one of the primary determinants of myocardial oxygen consumption. This parameter is higher in ischemic areas, and the changes in its regional variations are proportional to the ventricular remodeling after myocardial infarction (30). In the case of a dilated cardiomyopathy, the wall stress is correlated with the myocardial lesions (23, 32). Wall stress has also been studied in patients with valvulopathy and volume overload. Auffermann et al. (2) described a link between global wall stress increase and the degree of regurgitation. Disproportionately high systolic wall stress relative to regurgitant volume indicates the presence of myocardial disease, and wall stress assessment is useful in timing valve replacement in patients with regurgitant lesions.

APPENDIX

Determination of Radius and Wall Thickness in Direction Perpendicular to Left Ventricular Wall

Short-axis curvature. On all short-axis sections, a set of \(N\) lines \(\gamma_n\), originating from the centroid of the left ventricle was expressed by

\[
\gamma_n = [(p \cos \theta_n, p \sin \theta_n) \theta_n = 2\pi(n - 1)/N, 0 \leq p < \infty]
\]

for \(n \in [1, N]\)

where \(\theta_n\) is the angular direction and \(p\) is the distance from the centroid. In our study \(N = 128\), so that the difference between two directions \(\theta_1\) and \(\theta_2\) was 2.8.

For each value of \(\theta_n\), the radius \(R_n\) of the left ventricular cavity and the wall thickness \(T_n\) were measured. With plane geometry, the curvature \(\alpha_n\) of the middle line of the wall in the direction \(\theta_n\) (Fig. 2A) is given by

\[
\alpha_n = \frac{\pi}{2} - A \tan \left( \frac{y_{n+1} - y_{n-1}}{x_{n+1} - x_{n-1}} \right) - \theta_n
\]

where \((x_{n-1}, y_{n-1})\) and \((x_{n+1}, y_{n+1})\) are the coordinates of the points of the middle line along the direction \(\theta_{n-1}\) and \(\theta_{n+1}\). To reduce the sensitivity of the calculation to local artifacts, the left ventricle was divided into 16 sectors in which the measurements of the radius of the blood pool, the wall thickness and the short-axis curvature were averaged. In the middle of each sector \(S\), the radius of the blood pool \(2DR_S\) and the wall thickness \(2DT_S\) in the direction of the short-axis...
curvature $\alpha_S$ were either measured on the image or deduced by geometry

$$2DT_S = T_S \times \cos(\alpha_S)$$

$$2DR_S = \left[ \frac{R_S + T_S}{2} \right] \cos(\alpha_S) - \frac{2DT_S}{2}$$

where $R_S$ and $T_S$ are the mean radius of the blood pool and the mean wall thickness in sector $S$ along the radial lines originating from the centroid of the left ventricle.

The values of $2DR_S$ and $2DT_S$ obtained by direct measurements and those given by the previous formulas were closely related; the choice of method did not significantly affect the following results.

Long-axis curvature. In the median plane between two adjacent short-axis sections, the curvature $\beta_S$ in the long-axis direction for sector $S$ is determined from

$$\beta_S = A \tan \left( \frac{dr_S}{Th} \right)$$

where $Th$ is the thickness of the short-axis sections and $dr_S$ is given by

$$dr_S = \left[ \frac{R_{1S} + T_{1S}}{2} \right] - \left[ \frac{R_{2S} + T_{2S}}{2} \right] \times \cos \left( \frac{\alpha_{1S} + \alpha_{2S}}{2} \right)$$

where $R_{1S}$, $T_{1S}$, and $\alpha_{1S}$ are the radius of the blood pool, the wall thickness, and the short-axis curvature of sector $S$ in image 1 (Fig. 2B). $R_{2S}$, $T_{2S}$, and $\beta_{2S}$ are the corresponding data for the adjacent image 2.

The radius of the blood pool ($3DR_S$) and the wall thickness ($3DT_S$) along the direction perpendicular to the ventricular wall were deduced by the same reasoning as for the short-axis curvature (Fig. 2B).

$$3DT_S = 2DT_S \times \cos(\beta_S)$$

$$3DR_S = \left[ \frac{2DR_S + 2DT_S}{2} \right] \cos(\beta_S) - \frac{3DT_S}{2}$$

The values of $R_S$, $T_S$, $3DR_S$, and $3DT_S$ were determined at end diastole and end systole for each sector $S$.

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