Mechanical Evaluation of Cardiac Contractility in Left Ventricle with Disease Using Magnetic Resonance Tagging Technique*

Tadashi INABA**, Masataka TOKUDA**, Yasutomi KINOSADA***, Yutaka SAWAKI**, Kazuo YAGI** and Shingo KAWASAKI****

The cardiac contractility in human hearts was investigated by analyzing the deformations of the left ventricular myocardial walls during systole using a magnetic resonance tagging technique. Subjects were ten normal humans, eight patients with a hypertrophic cardiomyopathy (HCM) and seven patients with a hypertensive heart disease (HHD). The minimum principal strain, which describes the maximum contraction, was employed as an index for an evaluation of the cardiac contractility. The obtained results showed that the minimum principal strains in local regions of the patients with a HCM were significantly smaller compared with those in corresponding regions of the normal humans, while the minimum principal strains in whole regions of the patients with a HHD were similar to those in corresponding regions of the normal humans. This study suggests that to evaluate the cardiac contractility from a mechanical point of view is useful for a quantitative evaluation of heart diseases.

** Key Words **: Biomechanics, Cardiac Contractility, Human Left Ventricle, Myocardial Wall Motion and Deformation, Minimum Principal Strain, Magnetic Resonance Tagging Technique, Heart Disease

1. Introduction

Hearts, especially left ventricles, play a role as an ejection pump in the circulatory system, and ejection of blood is caused by contraction of the myocardium. It is, therefore, considered that to evaluate the cardiac contractility from a mechanical point of view by analyzing the deformation behavior of the myocardial wall is useful for a quantitative evaluation of the extent of heart disease or the effect of treatment. In order to analyze the deformation of the myocardial wall in humans, a noninvasive measurement of regional wall motion is required. A magnetic resonance tagging technique(1)-(9) is one of such measurement techniques. By applying this technique, the state of nuclear spins at specific locations in an imaging object is modified spatially, so that patterns of stripes can be formed in the imaging object. The tagging stripes move together with motion of the imaging object. As a result, the noninvasive measurement and analysis of the human myocardial wall motion and deformation become possible(10)-(19), by imaging a heart using the magnetic resonance tagging technique and tracking the movement of the tagging stripes within the myocardial wall.

From the above-mentioned point of view, we have tried to investigate mechanically the cardiac contractility by using the magnetic resonance tagging technique. In previous papers(7)(8), the two-dimensional deformations of the left ventricular walls during ejection period in normal humans and a patient with a hypertrophic cardiomyopathy were analyzed. Then, it was discussed that what kind of physical quantities is important to evaluate quantitatively the

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** Department of Mechanical Engineering, Meiji University, 1-15-1 Higashi-Matsubara, Tama-shi, Tokyo 204-8577, Japan. E-mail: inaba@meiji.ac.jp
*** Department of Medical Informatics, Gifu University, Tsugome, Gifu-city, Gifu 500-8705, Japan
**** Matsushita Memorial Hospital, Sotojima, Moriguchi 570-8540, Japan

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cardiac contractility. From the obtained results, it was suggested that the minimum principal strain, which describes the maximum contraction, could be an important and effective index for the evaluation of the cardiac contractility from viewpoints of both the physical meaning of this parameter and the ability to detect a singular region in the diseased heart. In the present paper, by applying this strain analysis method to patients with a hypertrophic cardiomyopathy and patients with a hypertensive heart disease, the cardiac contractility in each heart disease is investigated from a mechanical point of view, and usefulness of the mechanical evaluation of the cardiac contractility is discussed.

2. Image Acquisition

Ten normal humans (6 men and 4 women, aged 21–56 years), eight patients with a hypertrophic cardiomyopathy (HCM, 6 men and 2 women, aged 21–62 years), and seven patients with a hypertensive heart disease (HHD, 7 men, aged 39–69 years) were imaged with a 1.5 T superconducting magnet (SIGNA advantage, GE). The imaging procedure to obtain left ventricular short-axis images is as follows;

(1) Coronal gradient-echo images are obtained to identify a cardiac location.

(2) An oblique gradient-echo image is obtained so as to pass through the center of the aortic root and the left ventricular apex, as seen in the coronal image traversed the largest left ventricular cavity.

(3) A left ventricular long-axis is chosen so as to pass through the center of the mitral orifice and the apex, as seen in the oblique image.

(4) Multiphase tagged short-axis images at an equatorial section of left ventricle perpendicular to the chosen long-axis are obtained under the conditions of image acquisition as follows; 28 cm field of view, 256×256 image matrix, 10 mm thickness and 7 mm tagging stripe spacing.

It is assumed that the first tagged image corresponds to that at end diastole, because the first image is obtained after 20 ms from detection of the R wave of the electrocardiogram, and subsequent tagged images are obtained continuously at intervals of 25 ms.

3. Deformation Analysis

Tagging stripes form a number of intersections within the myocardial wall, and these intersections serve as material points in the myocardial wall. The deformation of the left ventricular wall was analyzed by tracking the intersections of the tagging stripes throughout systole. The components of Green’s strain tensor $E_{ij}$, $E_{ij}$, and $E_{ij}$ within a triangular region defined by three adjoining intersections were calculated by

$$ds^2 - dS^2 = 2E_{ij}dx_i dx_j \quad (i,j=1,2)$$

where $dS$ and $ds$ are lengths of line segments of the triangle in the initial state and the deformed state, respectively, and $dx_i$ and $dx_j$ are $x_i$ and $x_j$ components of the line segments in the initial state, respectively. Axes $x_i$ and $x_j$ are in horizontal and vertical directions of the image, respectively. The first image in a series of multiphase tagged images was employed as the initial state for the strain analysis. As a result, the strains calculated here are relative values based on an image at end diastole. Figures 1(a) and (b) show sample images at end diastole and end systole, respectively, in which the triangular regions for the strain analysis are constructed. Using the calculated strain tensor, the minimum principal strain $\lambda_1$ ($\lambda_1 > \lambda_5$, $\lambda_5$ is the maximum principal strain) was calculated by a following characteristic equation

$$|E_{ij} - \lambda_5 \delta_{ij}| = 0 \quad (i,j=1,2)$$

where $\delta_{ij}$ is Kronecker delta. The minimum principal strain, which is one of the eigenvalues of strain tensor, describes the maximum contraction of the region of interest. It is, therefore, considered that the minimum
principal strain is an important index for an evaluation of the cardiac contractility, especially an amount of contraction of the myocardium. Furthermore, in order to evaluate a difference of the cardiac contractility in local regions, the left ventricular wall was divided into four regions, that is, the anterior wall, the septal wall, the posterior wall, and the lateral wall, and the calculated strains in each triangular region were averaged in each wall (see Fig. 1 (b)).

4. Evaluation of Cardiac Contractility in Normal Heart

In order to clarify a difference between the cardiac contractility in normal humans and that in patients with a heart disease, it is indispensable to determine a quantitative reference of normal values. In the present paper, to provide this determination, the deformations of the left ventricular walls in ten normal humans were analyzed. Figure 2 shows time courses of the minimum principal strains during systole in the normal humans. In Fig. 2, horizontal axes indicate time passing from end diastole to end systole. From Fig. 2, it is confirmed that the minimum principal strains are almost uniform over whole regions in each subject. Figure 3 shows the minimum principal strains averaged in ten normal humans at end systole. As seen in Fig. 3, the minimum principal strains at end systole in each region are $-0.199 \pm 0.015$ (mean \pm S.D.) in the anterior wall, $-0.179 \pm 0.015$ in the septal wall, $-0.182 \pm 0.019$ in the posterior wall, and $-0.193 \pm 0.013$ in the lateral wall, respectively, and it is recognized that dispersion among ten subjects is considerably small (coefficients of variations are 7.5% in the anterior wall, 8.3% in the septal wall, 10.5% in the posterior wall, and 6.8% in the lateral wall). Figure 4 shows a typical example of a distribution of the minimum principal strains at end systole in a normal human. In Fig. 4, lengths and directions of line segments within triangular regions indicate magnitudes and directions of the minimum principal strains, respectively. From Fig. 4, it is observed that a circumferential direction is dominant for the directions of the minimum principal strains over whole regions. From these results, so far as the deformation of the myocardial wall at a short-axis section in normal humans is discussed, it is considered that the myocardial wall contracts similarly over whole regions, and that there is no significant difference among subjects about an amount of contraction of the myocardium. An index to evaluate singularity of diseases is hoped that dispersion among normal subjects is small. For this reason, the minimum principal strain could be an effective index for a quantitative evaluation of heart contractility.
Table 1 Minimum principal strains at end systole in patients with a HCM

<table>
<thead>
<tr>
<th></th>
<th>anterior</th>
<th>septal</th>
<th>posterior</th>
<th>lateral</th>
</tr>
</thead>
<tbody>
<tr>
<td>HCM A</td>
<td>-0.134</td>
<td>-0.094</td>
<td>-0.188</td>
<td>-0.223</td>
</tr>
<tr>
<td>HCM B</td>
<td>-0.092</td>
<td>-0.049</td>
<td>-0.101</td>
<td>-0.206</td>
</tr>
<tr>
<td>HCM C</td>
<td>-0.127</td>
<td>-0.059</td>
<td>-0.164</td>
<td>-0.162</td>
</tr>
<tr>
<td>HCM D</td>
<td>-0.185</td>
<td>-0.093</td>
<td>-0.164</td>
<td>-0.250</td>
</tr>
<tr>
<td>HCM E</td>
<td>-0.078</td>
<td>-0.151</td>
<td>-0.190</td>
<td>-0.154</td>
</tr>
<tr>
<td>HCM F</td>
<td>-0.155</td>
<td>-0.159</td>
<td>-0.144</td>
<td>-0.190</td>
</tr>
<tr>
<td>HCM G</td>
<td>-0.100</td>
<td>-0.072</td>
<td>-0.141</td>
<td>-0.192</td>
</tr>
<tr>
<td>HCM H</td>
<td>-0.100</td>
<td>-0.059</td>
<td>-0.148</td>
<td>-0.221</td>
</tr>
<tr>
<td>normal</td>
<td>-0.199±0.015</td>
<td>-0.199±0.015</td>
<td>-0.182±0.019</td>
<td>-0.193±0.013</td>
</tr>
</tbody>
</table>

5. Results and Discussions in Diseased Heart

5.1 Heart with HCM

Figure 5 shows time courses of the minimum principal strains during systole in the patients with a HCM. From Fig. 5, it is confirmed that the minimum principal strains are different from regions to regions in each subject. The minimum principal strains at end systole in the patients with a HCM are summarized in Table 1. In Table 1, an asterisk indicates that an absolute value of the minimum principal strain is significantly smaller than an absolute value of mean +3.29 × S.D. in corresponding region of the normal humans. As seen in Table 1, regions in which the minimum principal strain is small are observed in all subjects. Figure 6 shows a distribution of the minimum principal strains at end systole in the subject B. From Fig. 6, it is observed that the magnitudes of the minimum principal strains are small around the septal wall from the anterior wall to the posterior wall, and that the directions of the minimum principal strains in the lateral wall are similar to those of the normal humans.

Fig. 5 Time courses of minimum principal strains during systole in patients with a HCM

Fig. 6 Distribution of minimum principal strains at end systole in subject B

Fig. 7 Time courses of minimum principal strains during systole in patients with a HHD

5.2 Heart with HHD

Figure 7 shows time courses of the minimum principal strains during systole in the patients with a HHD. From Fig. 7, the minimum principal strains are almost uniform over whole regions in each subject as well as those in the normal humans. The minimum principal strains at end systole in the patients with a HHD are summarized in Table 2. As seen in Table 2, there is no region in which the minimum principal strain is significantly small. Figure 8 shows a typical example of a distribution of the minimum principal strains at end systole in a patient with a HHD. From Fig. 8, the directions of the minimum principal strains over whole regions are similar to those in the normal humans.

5.3 Discussions

In the obtained results, it was recognized that the minimum principal strains in local regions of the patients with a HCM were significantly smaller

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Table 2 Minimum principal strains at end systole in patients with a HHD

<table>
<thead>
<tr>
<th></th>
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<th>septal</th>
<th>posterior</th>
<th>lateral</th>
</tr>
</thead>
<tbody>
<tr>
<td>HHD A</td>
<td>-0.161</td>
<td>-0.158</td>
<td>-0.177</td>
<td>-0.196</td>
</tr>
<tr>
<td>HHD B</td>
<td>-0.192</td>
<td>-0.147</td>
<td>-0.193</td>
<td>-0.177</td>
</tr>
<tr>
<td>HHD C</td>
<td>-0.212</td>
<td>-0.151</td>
<td>-0.175</td>
<td>-0.201</td>
</tr>
<tr>
<td>HHD D</td>
<td>-0.232</td>
<td>-0.155</td>
<td>-0.165</td>
<td>-0.205</td>
</tr>
<tr>
<td>HHD E</td>
<td>-0.257</td>
<td>-0.163</td>
<td>-0.216</td>
<td>-0.258</td>
</tr>
<tr>
<td>HHD F</td>
<td>-0.196</td>
<td>-0.156</td>
<td>-0.177</td>
<td>-0.169</td>
</tr>
<tr>
<td>HHD G</td>
<td>-0.238</td>
<td>-0.182</td>
<td>-0.211</td>
<td>-0.231</td>
</tr>
<tr>
<td>normal</td>
<td>-0.199±0.015</td>
<td>-0.179±0.015</td>
<td>-0.182±0.019</td>
<td>-0.193±0.013</td>
</tr>
</tbody>
</table>

Fig. 8 A typical example of distribution of minimum principal strains at end systole in a patient with a HHD

compared with those in corresponding regions of the normal humans. From these results, it is considered that HCM is a heart disease accompanied by a reduction of an amount of contraction of the myocardium, and that the minimum principal strain could be an effective index for a quantitative and local evaluation of an extent of the reduction. In histology, it is known that disarray of the myocardium is brought about in a hypertrophic region of hearts with a HCM. It is, therefore, assumed that the disarray of the myocardium causes the reduction of the cardiac contractility.

On the other hand, from the results in the patients with a HHD, it was recognized that there was no region in which the minimum principal strain was significantly small, and that the directions of the minimum principal strains were similar to those in the normal humans. From these results, it is considered that an amount of contraction of the myocardium in hearts with a HHD is almost the same as that in normal hearts. It is, therefore, assumed that hypertrophy of the myocardium in this disease is not an abnormality of the myocardium, but a secondary change to compensate a reduction of the ejection fraction caused by hypertension. Furthermore, from the above results, it is considered that the cardiac contractility in HCM and that in HHD are different, although both of them are heart diseases accompanied with the hypertrophy of the myocardium. And, it is confirmed that a difference between the cardiac contractility in each heart disease is reflected in the minimum principal strain.

6. Concluding Remarks

In the present paper, to investigate usefulness of a mechanical evaluation of the cardiac contractility, the deformations of the left ventricular myocardial walls during systole in the normal humans, the patients with a HCM and the patients with a HHD were analyzed by using the magnetic resonance tagging technique. The obtained findings were summarized as follows;

1) The minimum principal strains in local regions of the patients with a HCM were significantly smaller compared with those in corresponding regions of the normal humans.

2) The minimum principal strains in whole regions of the patients with a HHD were similar to those in corresponding regions of the normal humans.

3) It was considered that the cardiac contractility in HCM and that in HHD were different, although both of them were heart diseases accompanied with the hypertrophy of the myocardium.

From these findings, it is confirmed that a characteristic of the cardiac contractility in each heart disease is reflected in the minimum principal strain. It is, therefore, suggested that to evaluate the cardiac contractility from a mechanical point of view by analyzing the deformation of the myocardial wall is useful for a quantitative evaluation of heart diseases.

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