Myocardial Fascicle and Fiber Disarray in 25 μ-Thick Sections

Hisayoshi Fujinara, M.D., Chuichi Kawai, M.D., and Yoshihiro Hamashima, M.D.

SUMMARY We compared the histologic picture of myocardial fiber disarray in thin (4 μ) and thick (25 μ) sections of tissues obtained at autopsy from 18 adults and eight infants with clinically normal hearts, from 10 hearts with concentric hypertrophy (hypertensive patients), nine with myocardial infarction and four with hypertrophic cardiomyopathy (HCM).

In the thick sections, so-called bizarre myocardial fiber disarray in thin sections was seen as a bizarre fascicle disarray. Therefore, the usual fiber disarray reported in cases of HCM is actually a fascicle disarray with a three-dimensional complex network.

There was no marked difference in distribution and frequency of fascicle disarray among normal adult and infant hearts, and diseased hearts with hypertension and myocardial infarction. In the four hearts with HCM, diffuse bizarre fascicle disarray in the thick section was detected in the septum and anterior and posterior walls of the left ventricles in all cases, and in the lateral walls in one case. In the portions without the diffuse fascicle disarray, the distribution of disarray was the same as that in hearts with no HCM. Such fascicle disarray, including that of HCM, is probably congenital.

MYOCARDIAL FIBER DISARRAY has been described as a histologic feature of hypertrophic cardiomyopathy with or without obstruction. However, disarray is not specific for this condition. It has been found, although to a lesser extent, in many patients with or without heart disease.

Histologic examinations were done using thin (light microscopy 1–9 μ and electron microscopy 0.05–0.1 μ) sections. We speculated whether thin sections are adequate for observation of myocardial fiber disarray with a three-dimensional complex network, as each myocardial cell with disarrangement is cut by the microtome blade and the myocardial fiber architecture is necessarily lost.

We compared the histologic picture in thin (4 μ) and thick (25 μ) sections and studied the so-called myocardial fiber disarray in thick sections of clinically normal hearts and of diseased hearts, including four with hypertrophic cardiomyopathy.

Materials and Methods

Forty-nine hearts were studied, including 18 adult hearts and eight infant hearts from clinically normal subjects, 10 hearts with concentric hypertrophy due to hypertension, nine hearts with old and fresh myocardial infarction without hypertension, and four hearts with hypertrophic cardiomyopathy. The hearts of these 49 patients were selected at necropsy from a total of approximately 1500 autopsies performed between 1975 and 1977 at the Faculty of Medicine in Kyoto University and Shizuoka Rosai Hospital. One patient with hypertrophic cardiomyopathy with obstruction was autopsied in 1970. Age, sex and heart weight of these subjects are listed in table 1.

During the selection of specimens for study, a heart was judged normal if there was no evidence of clinical heart disease, the coronary arteries showed less than 75% luminal narrowing of each major coronary artery and there was no significant valvular or congenital heart disease.

The three major coronary arteries in all subjects were transversely and serially cut at 0.2–0.3-cm intervals from ostium to periphery, and the degree of luminal narrowing was recorded as a percentage. Regardless of the presence of significant atherosclerosis, a case was included in the hypertensive group whenever there was a clear-cut history of hypertension and the heart weighed over 400 g. All 10 hearts in this group had concentric hypertrophy of the left ventricle. In the group with old and fresh myocardial infarction without hypertension, the luminal narrowing in one or more major coronary arteries was greater than 90%. In four cases of hypertrophic cardiomyopathy, three had marked obstruction of the outflow tract and one had no obstruction. We used the anatomic criteria for hypertrophic cardiomyopathy as described by others. In such cases there was no hypertension and coronary narrowing was less than 50%.

The heart was incised serially and transversely at about 1-cm intervals and observed macroscopically. Two large sections (the upper third and the lower third) of the heart were divided into four and each of the eight sections per heart was used for histologic study. Each tissue block was serially sectioned at 4 μ thick (thin section) and 25 μ thick (thick section) in the adult hearts and in the specimens from infants at 4μ thick and 20 μ. These three serial sections were stained with hematoxylin-eosin and were used to compare the histologic picture in thin and thick sections.

We attempted to section the materials in various thicknesses. The arrangement of myocardial fibers could not be clearly observed in a section more than 40 μ thick because of the folding of many fibers. Sections 20–35 μ thick in adult tissues and from 15–35 μ thick
### Table 1. Summary of Cases

<table>
<thead>
<tr>
<th></th>
<th>Number of cases</th>
<th>Sex</th>
<th>Age (years)</th>
<th>Heart weight (g)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Normal adult hearts</td>
<td>18</td>
<td>13/5</td>
<td>21–80</td>
<td>200–350</td>
</tr>
<tr>
<td>Normal infant hearts</td>
<td>8</td>
<td>5/3</td>
<td>4Mo.–2</td>
<td>30–60</td>
</tr>
<tr>
<td>Hypertensive hearts weighing</td>
<td>10</td>
<td>7/3</td>
<td>45–80</td>
<td>400–600</td>
</tr>
<tr>
<td>over 400 g</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Myocardial infarction without</td>
<td>9</td>
<td>7/2</td>
<td>45–82</td>
<td>210–520</td>
</tr>
<tr>
<td>hypertension</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Hypertrophic cardiomyopathy</td>
<td>4</td>
<td>m</td>
<td>4</td>
<td>180</td>
</tr>
<tr>
<td></td>
<td></td>
<td>m</td>
<td>28</td>
<td>600</td>
</tr>
<tr>
<td></td>
<td></td>
<td>m</td>
<td>36</td>
<td>770</td>
</tr>
<tr>
<td></td>
<td></td>
<td>f</td>
<td>68</td>
<td>550</td>
</tr>
</tbody>
</table>

*These cases had obstruction of the outflow tract.

in infant tissues were adequate to observe the myocardial fiber arrangement.

**Results**

**Comparison of the Histologic Picture in Thin (4 μ) and Thick (25 μ) Sections**

In thin sections, the myocardial fiber disarray is classified into myocardial cell disarray, myocardial fiber disarray and myocardial fascicle disarray. Myocardial cell disarray means each cell has a disordered arrangement, but is artfactually cut by the blade of the microtome, and the structure of the myocardial fibers is unknown (figs. 1, 2 and 3). In myocardial fiber disarray each fiber has a disordered arrangement (fig. 4). In fascicle disarray, small bundles of normally appearing cardiac muscle cells or fibers course in a disorderly fashion (fig. 4).

![Figure 1](http://circ.ahajournals.org/)

**Figure 1.** Bizarre myocardial cell disarray in thin sections (4 μ) is shown in a₁ and b₁. The thick sections (25 μ), which revealed them to be fascicle disarrays, are shown in a₂ and b₂. V of b₁ and b₂ shows the same small vessel. a₁ is from case 4 of hypertrophic cardiomyopathy, hematoxylin-eosin stain, × 200; b₁ is from case 3 of hypertrophic cardiomyopathy, hematoxylin-eosin stain, × 50.
It was discovered that bizarre myocardial cell disarray in thin sections was mostly bizarre fascicle disarray and locally bizarre fiber disarray in the thick sections (figs. 1 and 2). Myocardial cell disarray without a bizarre pattern in thin sections was fascicle or fiber disarray without a bizarre pattern in the thick sections (fig. 3). Fascicle and fiber disarray in thin sections showed a histologic picture analogous to that seen in the thick sections (fig. 4). The area without disarrays in the thin sections had almost the same histologic picture as the thick section. Here the thick section revealed a clearer structure of myocardial fibers and fascicles.

There were two variations in the size of the myocardial cells. In one, the area had a fascicle consisting of large cells and another fascicle consisting of small cells. In the other type, the size of the cells varied within a fascicle. The former was detected in the area of fascicle disarray. The size of myocardial cells could not be measured precisely because of the folding of fibers in the thick sections when the cells were not strictly sectioned transversely.

Myocardial Fascicle and Fiber Disarray in Thick Sections: We examined the correlation between myocardial fiber and fascicle disarray in thick sections. We detected bizarre fiber disarray (fig. 2) in the area of fascicle disarray, especially in the triangular portion surrounded by several fascicles with different directions (fig. 2). Here each fiber crossed and whirled, and focal fibrosis was sometimes noted. Fiber disarray without a bizarre pattern (Y-shaped) in the thick sections was seen everywhere in the ventricles with or without fascicle disarray. Bizarre fiber disarray was not evident in areas without fascicle disarray, even in the presence of severe fibrosis, fatty infiltration, coagulation necrosis or hypertrophy of myocardial cells. This was noted in all normal adult and infant hearts, as well as in various diseased hearts with hypertensive hypertrophy, myocardial infarction and hypertrophic cardiomyopathy.

Myocardial fascicles consisting of myocardial fibers, both fused and divided, were numerous and irregular. A given fascicle had branching connections with other fascicles in several different directions. Histologic pictures of myocardial fascicle disarray were classified as type A to G, although we saw variations of each type (fig. 5). The frequency and distribution of various fascicle disarrays in tissues (except for cases of hypertrophic cardiomyopathy) were examined separately in the outer third, middle third except the septum, inner third except the septum, and inner and middle thirds of septum in the left ventricle, triangular portions of anterior and posterior septum, and the subendocardium of the septum in right ventricle.

Figure 2. Bizarre myocardial cell disarray in thin sections (4 μ) is shown in a1 and b1. The thick sections (25 μ), which revealed them to be fiber disarrays, are seen in a2 and b2. The boxed area in b2 is shown in b1. The fiber disarray of b2 is present in the triangular portion of fascicles with several different directions. These tissues were from the triangular portion of the anterior septum of normal hearts. a1) hematoxylin-eosin stain, × 200; b1) hematoxylin-eosin stain, × 400; b2) hematoxylin-eosin, × 50.
FIGURE 3. Myocardial cell disarray without a bizarre pattern in thin sections is shown in a, and b. The thick sections, which revealed a, to be a fascicle disarray and b, to be fiber disarray, are shown in a2 and b2. These tissues were from the free wall of normal hearts. Hematoxylin-eosin × 100.

The data are summarized in tables 2 and 3. Various fascicle disarrays expanded diffusely in the inner third of the left ventricle except the septum, in the subendocardium of the septum of the right ventricle and in the triangular portions of the anterior and posterior septum, although the distribution varied from case to case. Fascicle disarrays were rare and focal in the outer third, and in the inner and middle thirds of the septum of left ventricle (tables 2 and 3). Bizarre fascicle disarray frequently appeared in triangular portions of the anterior and posterior septa; in the other portions this was rare and focal. Type E was sometimes diffusely detected in the inner third and middle third of the left ventricle, especially in the lower third of the heart. There was no marked difference of frequency and distribution of fascicle disarray among normal adult and infant hearts and diseased hearts in cases of hypertension and myocardial infarction. There was no marked difference of fibrosis between areas with or without fascicle disarray, both in the normal and hypertensive hearts.

Four Hearts with Hypertrophic Cardiomyopathy

In three hearts (cases 1, 2 and 3) with obstruction of the outflow tract, diffuse bizarre fascicle disarray in the thick sections appeared in the middle third of the septa and in the subendocardium of the right ventricular septa, including the middle and outer thirds of the anterior and posterior walls in the upper one third of the left ventricles. In one case without obstruction (case 4), bizarre fascicle disarray in the thick sections had spread throughout the middle and outer thirds of the free walls and the septa in the lower third of the left ventricle. In cases 1, 3 and 4, the thick sections revealed bizarre fascicle disarray and the thin sections bizarre cell disarray (fig. 1). In case 2, both thick and thin sections showed analogous histologic pictures of fascicle disarray (fig. 4A).

In the areas without the diffuse bizarre disarray, there was almost the same frequency and distribution of fascicle disarray both in normal and diseased hearts. Marked hypertrophy of the myocardial cells was detected diffusely in areas with or without disarray in four hearts.

Discussion

Two types of histology in the so-called bizarre myocardial fiber disarray have been reported. One picture is that of myocardial cells with a bizarre pattern and disorganization; this is what we refer to as myocardial cell disarray. The other type is composed of bizarre fascicle disarray. It has been documented
FIGURE 4. Myocardial fascicle and fiber disarray in thin sections is seen in a₁, b₁, and c₁. The thick sections, which showed an analogous histologic picture in comparison with that in the thin section, are shown in a₂, b₂, and c₂. a) From case 2 of hypertrophic cardiomyopathy; b and c) from normal hearts. Hematoxylin-eosin stain, × 100.

that the latter was rare in hearts of patients with hypertrophic cardiomyopathy; studies were all done by observation of thin sections of 4–9 μm. Our observations in 25-μm-thick sections revealed that the bizarre myocardial cell disarray was mostly bizarre fascicle disarray and focally bizarre fiber disarray. The bizarre fiber disarray was noted in the areas of fascicle disarray in normal hearts, diseased hearts in cases of hypertension and myocardial infarction, and four hearts with hypertrophic cardiomyopathy. Therefore, histologic features of bizarre myocardial fiber disarray are actually myocardial fascicle disarray with a three-dimensional complex architecture.

There was no marked difference in distribution and frequency of fascicle disarray among normal adult and infant hearts, and diseased hearts in cases of hypertension and myocardial infarction. Such fascicle disarray, including that of hypertrophic cardiomyopathy, is probably congenital. This conclusion is compatible with reports that a diffuse disarray was seen in hearts of infants with congenital anomalies with middle or late systolic isometric contraction. We believe that bizarre and diffuse fascicle disarray is an anomaly that occurs in the fetus in which the architecture of fascicles is congenitally determined.

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References

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