ANGIOCARDIOGRAMS AND HEMODYNAMICS IN IDIOPATHIC CARDIOMYOPATHY, WITH REFERENCE TO HISTOLOGY OF BIOPSIED VENTRICULAR MYOCARDIUM

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CARDIAC diseases of unknown cause are often accompanied by an enlargement or hypertrophy of the heart. These diseases are termed "idiopathic cardiomyopathies" (ICM) and have been defined as disorders of heart muscle of unknown etiology or association, by the research group on special disease¹ of the Ministry of Health and Welfare, Japan. The diseases are temporarily defined to include postpartum heart disease, alcoholic cardiomyopathy and endomyocardial disease including endocardial fibroelastosis. Goodwin's classification² was adopted for convenience. Such were roughly classified into hypertrophic cardiomyopathy (HCM) and congestive cardiomyopathy (CCM). HCM is divided into hypertrophic non-obstructive and hypertrophic obstructive cardiomyopathy (HOCM).

Hemodynamic and angiographic studies revealed that the cavity of the left ventricle is of normal size in diastole, but in systole it often appeared to be reduced in size in cases of HCM². Namely, the left ventricular end-diastolic volume (LVEDV) was normal or subnormal while the ejection fraction (EF) was above normal, especially in the obstructive type, when end-systolic volume was reduced^{3,4}. The relative wall thickness was greatly increased. The left ventricular end-diastolic pressure (LVEDP) increases in parallel and was highest in the obstructive type. Contractility indices were fairly normal and no

difference was noted between the obstructive and non-obstructive types. In CCM end-systolic and end-diastolic ventricular volumes were increased with an augmented end-diastolic pressure. The ejection fraction was markedly reduced. Distension and hypokinesis of the left ventricle were characteristic, where the relative wall thickness was reduced². Contractility indices were lower than normal^{3,5}.

Histological studies of HCM showed markedly enlarged myocardial fibers accompanied by an abnormal arrangement⁶. Muscle cells showed increased cellular branching, extensive side-to-side intercellular junctions, widened Z bands, and evidence of formation of new sarcomeres. The myofibrils were arranged annularly or perpendicularly to each other, radiating in several directions from the Z band⁷. The characteristic abnormality of hypertrophic obstructive cardiomyopathy (HOCM), i.e. idiopathic hypertrophic subaortic stenosis (IHSS)⁸ is said to be asymmetric septal hypertrophy (ASH)⁹. ASH, detected echocardiographically, is defined by a ventricular septum that is disproportionately thickened compared to the posterior-basal left ventricular free wall. The ultrastructural characteristics of the myocardium with ASH¹⁰ were disarray of myofibrils and myofilaments, and disorganization of muscle cells and abnormal cell contact. In particular, markedly enlarged, bizarrely shaped muscle cells were seen in ventricular septum. In CCM, a considerable excess of mitochondriae, the cristae of many being disrupted, lysosomes and glycogen, and interstitial fibrosis were observed. The appearance indicated severe myo-

Key Words:

Idiopathic cardiomyopathy
Left ventricular volume
Coronary cineangiogram
Biopsied ventricular muscle

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Fig.1. Diagram of left ventriculogram.
L: Long axis, D: Short axis, h: Wall thickness

cardial damage of unknown cause?

One hundred and thirty-one patients with ICM were encountered in our clinic from 1964 to 1977.¹¹ In the majority of cases, endomyocardial biopsy was performed in the right ventricle and/or the left ventricle immediately after cardiac catheterization and angiocardiology. There is apparently no documentation concerning the relationship between cardiac hemodynamics and simultaneous morphological findings of the ventricles in cases of idiopathic cardiomyopathy. The hemodynamics and angiocardiology findings were investigated herein

with special reference to correlation to the histological appearances of the biopsied ventricular myocardium in idiopathic cardiomyopathy.

MATERIALS AND METHODS

Among 131 patients with idiopathic cardiomyopathy there were 66 with HCM, including 10 with HOCM, and 65 with CCM. Chest X-ray, electrocardiography, phonocardiography, and recently echocardiography were utilized in making the diagnosis. Hypertensive or arteriosclerotic heart disease, valvular diseases, congenital heart diseases, and moreover endocrine diseases, metabolic disorders, blood dyscrasias and so forth were excluded by examinations of blood and urine and other tests. Right cardiac catheterization was performed in 109 cases and left cardiac catheterization in 79 cases. Left ventricular cineangiograms were carried out in 46 of these 79 cases, and in 40 cases, left ventricular volume was measured. The mean age in HCM of 21 cases was 32.2 years (range 19 to 50 years) and 33.8 years (range 16 to 56 years) in CCM of 19 cases. In 15 patients with HCM and 10 with CCM among the 40 cases left ventricular endomyocardial biopsy was done, the histological findings are reported herein.

The patients were administered 0.5 g of pyravital, 0.5 g of bromvalerylurea and 2 mg of diazepam 30 minutes prior to the cardiac

Fig.2. Hypertrophy on myocardial fibers. HE stain, $\times 640$

Fig.3. Vacuolization of myocardial fibers.
Grade 3+ HE stain, $\times 640$

Fig.4. Collagen proliferation (fibrosis) of interstitium.
Grade 3+ HE stain, $\times 640$

TABLE 1 ANGIOCARDIOGRAPHIC DATA

	Normal Left Ventricle (n = 9)	P Values				
		HCM (n = 21)	CCM (n = 19)	HCM vs Normal	CCM vs Normal	HCM vs CCM
End-diastolic volume (ml/m ²)	69.7 ± 10.6	64.2 ± 15.3	149.1 ± 36.6	NS	0.001	0.001
End-systolic volume (ml/m ²)	21.5 ± 6.0	16.9 ± 9.0	87.7 ± 43.3	NS	0.001	0.001
Stroke volume (ml/m ²)	48.2 ± 8.6	47.5 ± 8.5	61.6 ± 25.2	NS	NS	0.05
Ejection fraction (%)	69.2 ± 6.6	75.2 ± 8.5	43.4 ± 19.2	NS	0.001	0.001
Wall thickness (cm)	0.74 ± 0.08	1.03 ± 0.39	0.74 ± 0.24	0.01	NS	0.01
Left ventricular mass (g/m ²)	69.5 ± 12.2	105.4 ± 53.9	113.3 ± 40.1	0.02	0.01	NS
Mass/Vol (g/ml)	0.99 ± 0.20	1.73 ± 0.95	0.80 ± 0.30	0.01	NS	0.001
End-diastolic long axis (Ld)(cm)	7.88 ± 0.97	8.38 ± 0.97	8.93 ± 0.81	NS	0.02	NS
End-systolic long axis (Ls)(cm)	5.84 ± 0.82	6.26 ± 1.14	7.46 ± 0.97	NS	0.001	0.01
End-diastolic short axis (Dd)(cm)	5.27 ± 0.38	4.87 ± 0.45	6.92 ± 0.67	0.05	0.001	0.001
End-systolic short axis (Ds)(cm)	3.37 ± 0.34	2.78 ± 0.60	5.54 ± 1.22	0.01	0.001	0.001
Ld-Ls/Ld	0.26 ± 0.04	0.26 ± 0.08	0.17 ± 0.07	NS	0.001	0.001
Dd-Ds/Dd	0.36 ± 0.06	0.43 ± 0.09	0.20 ± 0.14	0.02	0.001	0.001
Mean Vcf (cir/sec)	1.31 ± 0.39	1.66 ± 0.42	0.75 ± 0.55	NS	0.01	0.001
MNSER (vol/sec)	2.49 ± 0.61	2.88 ± 0.61	1.60 ± 0.76	NS	0.01	0.001

mean Vcf: Mean velocity of circumferential fiber shortening

MNSER: Mean normalized systolic ejection rate

catheterization. Cardiac catheterization was performed utilizing standard Cournand catheters, which were inserted into the right and left ventricle from the saphenous vein and the brachial artery, respectively. Pressures were recorded by Hewlett Packard optical recording system (4588D system) or Sanborn optical recorder utilizing Statham P23D6 transducer. The mid thoracic position was utilized as the zero pressure reference. Mean pressure measurements were obtained by electrical damping. Recently, Mikro-tip pressure transducer® of Millar Instruments (PC 481 et al.) was also used to record intracardiac pressures. Mean pulmonary wedge pressure (PCm), mean pulmonary artery pressure (PAm) and right ventricular end-diastolic pressure (RVEDP) were determined from the right intracardiac pressure curves with averaged values of at least 5 beats. From the left intracardiac pressure curves, left ventricular end-diastolic pressure and "a" wave were measured. The cardiac output was determined by the direct Fick method.¹² The expired gas was collected in a Douglas bag and minute ventilation was measured by utilizing a dry gasmeter. The oxygen content of the expired air sample was

determined by the Scholander method.¹³ Blood samples from the main pulmonary artery and systemic artery obtained halfway through the air collection were analyzed for oxygen content by the method of Van Slyke and Neill.¹⁴ The heart rate, continuously recorded at a slow paper speed during the determination of cardiac output, was divided into the latter value to determine the stroke volume.

After recording of intracardiac pressures, 35–45 ml of 80% Angioconray® was injected into the left ventricular cavity with 4 kg/cm² pressure within 2–3 seconds utilizing NIH catheter No. 7 or 8 with a power injector (Gidlund). It was photographed at 45° right anterior oblique projection on 35 mm film at 100 frames per second. Magnification was set at zero level of the left chest and 5 cm of wire was placed on the position of heart. After selective left ventriculography, coronary cineangiography was performed using the technique of Sones and Shirey.¹⁵

On left ventriculograms, in rhythms other than extrasystoles and postextrasystolic beats, long axis and short axis of the left ventricle were directly measured by the method of Greene

TABLE II HEMODYNAMIC DATA

	HCM (n = 21)	CCM (n = 19)	P Values
<i>Pressures (mmHg)</i>			
Right ventricular end-diastolic	6.9 ± 1.5	5.8 ± 2.5	NS
Mean pulmonary artery	13.2 ± 1.8	17.7 ± 6.8	0.02
Mean pulmonary wedge	9.4 ± 1.5	12.3 ± 7.1	NS
Left ventricular end-diastolic	14.6 ± 6.2	14.8 ± 7.8	NS
Left artial kick	61.9%	31.6%	0.05
Cardiac index (l/min/m ²)	3.34 ± 0.92	2.96 ± 1.03	NS
Stroke index (ml/beat/m ²)	47.5 ± 13.1	44.9 ± 18.5	NS

TABLE III HISTOLOGICAL FINDINGS

		HCM	CCM	P Values
RV	Diameter (μ)	18.58 ± 4.66	19.77 ± 3.85	NS
	D-score	3.4 ± 1.2	4.4 ± 1.9	NS
	Fibrosis	1.2 ± 0.9	1.4 ± 1.0	NS
		(n = 17)	(n = 14)	
LV	Diameter (μ)	23.91 ± 4.81	23.37 ± 3.72	NS
	D-score	5.7 ± 1.9	5.8 ± 1.5	NS
	Fibrosis	1.1 ± 0.8	1.4 ± 0.7	NS
		(n = 15)	(n = 10)	

RV: Right ventricle LV: Left ventricle Diameter: Diameter of muscle fiber
D-score: Degeneration score of muscle fiber Fibrosis: Collagen fiber proliferation of interstitium

et al.¹⁶ as shown in Figure 1. Assuming the left ventricular cavity to be an ellipsoid of revolution, the volume was calculated by the following formula:

$$V = \frac{\pi}{6} LD^2 \frac{1}{f^3} \dots\dots\dots 1)$$

where L is long axis of the left ventricle, D short axis, and f magnification. The magnification (f) is ratio of 5 cm to length of the wire on image.

The distance between the point where the extended line of the short axis crossed over the cardiac shadow of the left ventricular anterior wall and inner surface was assumed to be the wall thickness.¹⁷ Left ventricular mass was determined by the method of Rackley and associates¹⁸ as follows:

$$LV \text{ mass} = \frac{\pi}{6} [(L + 2h)(D + 2h)^2 - LD^2] \frac{1}{f^3} \times 1.050 \dots\dots\dots 2)$$

L : long axis of the left ventricle
D : short axis of the left ventricle
h : wall thickness of anterior mid portion of left ventricle
f : magnification
1.050: the specific gravity of heart muscle

The calculation of mean velocity of fiber shortening (meanVcf) was made by the method of Karliner et al.⁵ The extent of shortening of the minor internal circumference between end-diastole and end-systole was divided by the time required for shortening less 50 msec. Mean normalized systolic ejection rate (MNSER) was also calculated the difference in left ventricular volume between end-diastole and end-systole, divided by the time required for shortening less 50 msec and the end-diastolic volume.^{19,20}

Left ventricular asynergy and regurgitation to the left atrium were also observed.

Fig.5. Relationship between diameter of heart muscle fiber and hemodynamics.

Fig.6. Relationship between pathological and hemodynamic findings.

As the control group 9 subjects without heart disease were selected, aged 18–64 years (averaged 43.3 years), and the mean values of hemodynamic and angiographic data was used as normal control values. As to the coronary arteries, the severity of stenosis, obstruction or the existence of other lesions were determined from the coronary arteriograms.

The right and/or left ventricle were biopsied using Konno-Sakakibara's endomyocardial biotom²¹ in order to investigate the histology of the myocardium. The obtained specimens were fixed in 10% formalin, dehydrated with a series of ethanol, embedded in paraffin and stained with hematoxylin-eosin, Mallory-azan and elastica van Gieson. As to the hypertrophy of myocardial fibers, the shortest diameter was measured only in nucleated transverse sections by ocular micrometer disc in one hundred fibers and then averaged. Hypertrophied myocardial fibers are shown in Figure 2. Degenerative changes, i.e. scarcity of myofibrils, deformity of nuclei and vacuolization of myocardial fibers were divided into four grades, i.e. 1+ to 4+, as shown in Fig. 3. The figure in total was assumed to be the score showing degenerative changes of the myocardial fibers. Collagen fiber

proliferation (fibrosis) of the interstitium was also divided into four grades (Fig. 4).

The relationship between the histological findings in myocardial fibers and parameters of hemodynamic and angiographic data was determined and the statistical significance was determined using the "t" test.

RESULTS

Angiographic findings.

The angiographic data for the 40 patients with ICM (21, HCM and 19, CCM) and for nine subjects considered to have normal left ventricles are given in Table I. LVEDV was 64.2 ± 15.3 ml/m² in HCM and 149.1 ± 36.6 ml/m² in CCM, where 69.7 ± 10.6 ml/m² in normal controls. LVESV was 16.9 ± 9.0 ml/m² in the former and 87.7 ± 43.3 ml/m² in the latter, where 21.5 ± 6.0 ml/m² in normal controls. Patients with HCM had normal or smaller end-diastolic and end-systolic volume, whereas patients with CCM had a large volume in both end-diastolic and end-systolic phases. Furthermore, the mean end-diastolic and end-systolic volumes in CCM were significantly greater than the mean value of HCM and normal control group. There was no significant difference in mean values of left

ventricular volume between HCM and normal control group. The stroke volume which was calculated from those values showed no difference from the control group.

The mean value of EF was abnormally low in those with CCM compared to that of the normal subjects. In HCM, 8 patients (38.1%) showed a larger value than normal subjects. Four of 19 patients with CCM (21.1%) had a normal ejection fraction and one showed a larger value.

The anterior wall thickness (WT) of left ventricle was 1.03 ± 0.39 cm in HCM and 0.74 ± 0.24 cm in CCM, where 0.74 ± 0.08 cm in normal controls. The thickness was greater in HCM and no difference was observed in CCM compared to that of normal subjects. Eleven cases of HCM showed thickening (52.4%), whereas only were seen in 5 cases of CCM (26.3%). In 5 cases of CCM the wall thickness was thinner than normal. Only 3 such cases were seen in HCM (14.3%).

The mean left ventricular mass was increased in both types and revealed no significant difference between the two types. The mean value of the ratio of LV-mass and volume was greater in HCM and smaller in CCM than that seen in the normal group. There was a significant difference between HCM and the normal control group. The mean values of left ventricular end-diastolic long axis (Ld) and end-systolic long axis (Ls) were larger in both types than in the normal subjects. There was a significant difference of Ld as well as Ls between CCM and normal subjects. The mean values of left ventricular end-diastolic short axis (Dd) and end-systolic short axis (Ds) were significantly greater in CCM and smaller in HCM compared to those seen in the control group. The shortening of long axis in systole ($\frac{Ld-Ls}{Ld}$) was smaller in CCM than normal subjects and HCM, and the shortening of short axis ($\frac{Dd-Ds}{Dd}$) was larger in HCM and smaller in CCM than normal subjects.

In HCM, neither parameter of myocardial contractility showed a lower value than the control group and in CCM a higher value was seen. There was a significant difference in mean values of mean Vcf and MNSER in CCM compared to HCM and the control group.

In angiocardiograms asynergy of left ventricular wall was found in 10 of 19 cases (52.6%) of CCM. There were 5 cases with hypokinesis

(26.3%), 4 with akinesis (21.1%) and one with dyskinesis (5.2%). Regurgitation to the left atrium was noted in 7 patients, i.e. one case in HOCM (4.8%) and 6 cases in CCM (31.6%).

Selective coronary cineangiography was carried out in 32 patients, i.e. 15 patients in HCM and 17 in CCM. There was neither narrowing nor occlusion of coronary arteries, rather hyper-vascularity was seen in 3 cases with HCM (20%) and 4 with CCM (23.5%). Although QS pattern in the electrocardiogram was seen in two cases with CCM, there were no abnormalities in the coronary arteriograms. One patient died suddenly and at autopsy, a marked fibrosis of the myocardium without changes of coronary arteries was noted.

Hemodynamic study

The results of the hemodynamic studies in the two groups are given in Table II. Cardiac index and stroke index were within normal ranges in both types, but lower values were seen in many patients with CCM. The mean values of cardiac index and stroke index showed no significant statistical difference. PCm and PAm were within normal range in HCM and slightly higher in CCM. A significant difference was observed in mean value of PAm between both types. RVEDP was slightly higher in HCM than in CCM, but there was no significant difference statistically. LVEDP showed high values in both types, but there was no significant difference. According to the results of 131 cases of ICM examined at our clinic,¹¹ significant differences of LVEDP, PCm and PAm were seen in both two types and such were higher in CCM. Incidence of atrial kick in left ventricular curve was significantly higher in HCM than CCM.

Histological findings of the myocardium.

Histological findings of the biopsied right and left ventricular myocardium are shown in Table III. The diameter of myocardial fibers of the right ventricle was 18.58 ± 4.66 μ m in HCM and 19.77 ± 3.85 μ m in CCM. Here there was no statistical difference. The diameter of myocardial fibers of the left ventricle was 23.91 ± 4.81 μ m in HCM and 23.37 ± 3.72 μ m in CCM. There was no difference between HCM and CCM. With respect to degenerative changes of myocardial fibers as the score, there was no difference statistically in the right or left ventricle between both types. The degree of fibrosis was also no statistical difference.

The relationships between microscopic findings of biopsied left ventricular muscle and the parameters of hemodynamic and angiocardiographic findings including EDP, Dd, EDV, EF, meanVcf, WT and LV-mass were examined. As shown in Figs. 5 and 6, there was in general no relationship except that between LV wall thickness and diameter of left ventricular myocardial fibers, which was positive in $r=0.4425$, $P<0.05$. Therefore, with an increase in diameter of the myocardial fibers, wall thickness seemed to increase.

As to individual types, there was a negative correlation between LVEDV and diameter of myocardial fibers in the left ventricle ($r=0.7900$, $P<0.05$) in CCM. Otherwise, no relationship was found in HCM as well as CCM. There were numerous cases of HCM in which there was marked elevation in LVEDP and a large diameter of the myocardial fibers.

DISCUSSION

As to the measurement of the left ventricular volume by one-plane cineangiography there are two methods, namely area length method²³ and direct axis measurement method.¹⁶ In general, the left ventricular volume has been obtained by the area-length method, where the ventricular short axis was calculated by the directly measured long axis and the area of the drawn left ventricular inner cavity obtained by planimeter. In the present study, the latter method was used, because it is a simple procedure, in which the short axis can be measured directly. Comparing the mean values of normal controls with those reported by Kennedy and associates,²⁴ the left ventricular end-diastolic, end-systolic volume and ejection fraction were almost the same, but the anterior wall thickness and mass of the left ventricle showed slightly lower values. These values were slightly less than those reported by others,^{17,25-36} except for the ejection fraction. Although there are few reports about the left ventricular short axis,^{25,35} such was estimated lower as it was calculated from the equation 1).

As the classification of the cardiomyopathies into two groups is determined mainly by means of estimation of the left ventricular volume, angiocardiograms revealed distinguished features. In this study the mean end-diastolic volume of the left ventricle in HCM was normal, though it was smaller in 8 cases (38.1%) than in normal subjects. In CCM it was much greater than that in HCM and normal subjects, and almost twice.

The left ventricular end-systolic volume of the left ventricle was normal or slightly smaller in HCM than in normal subjects, while in CCM it was about four times of normal value.

The ejection fraction which was calculated from the end-diastolic and end-systolic volumes was smaller in CCM than in HCM and normal subjects.

In more than a half cases of HCM left ventricular anterior wall was thicker than in normal group and CCM, where mean value of the wall thickness was larger by 40%. Although in CCM it was not different from normal group, it showed a little larger or smaller in some cases.

Left ventricular mass was almost the same in both types but significantly larger than in the normal control group. The ratio of the left ventricular mass and volume suggest the relative extent of left ventricular hypertrophy.³⁷ It was much larger in HCM than normal, while smaller in CCM.

From the results mentioned above, the left ventricle was hypertrophied without its dilatation in HCM, while it was not so hypertrophied with much dilatation in CCM.

There are many parameters for the assessment of myocardial contractility. Mean velocity of circumferential fiber shortening and MNSER can be assessed by cineangiocardiography, and end-diastolic pressure of the left ventricle by cardiac catheterization. In the present study mean values of meanVcf and MNSER in normal control group were closed to the lower normal range reported by other investigators.^{5,19,20} Those were normal in HCM, which showed normal PAm and PCm though end-diastolic pressure of the left ventricle was elevated. Therefore, the elevation of LVEDP did not indicate a decrease of myocardial contractility, but a decrease in diastolic compliance of the left ventricle due to thickening of the wall.³⁸ The atrial kick in left ventricular pressure curve is considered to be induced by this mechanism. In the right ventricle there appeared also an elevation of end-diastolic pressure and atrial kick.

In CCM meanVcf and MNSER were much lower than normal. Left ventricular end-diastolic pressure was elevated just like in HCM, but accompanied by an increase in PAm and PCm. These findings suggested that myocardial contractility of the left ventricle were much depressed, indicating congestive heart failure.

In HCM long axis in end-diastole (Ld) and end-systole (Ls) were normal, while short axis in

end-diastole (Dd) and end-systole (Ds) were smaller than normal. The shortening of long axis in systole was almost the same as in normal subjects, whereas the shortening of short axis was much larger. It means that the left ventricle contracts forcefully by short axis in HCM. In CCM long axes were larger as well as short axes than normal subject. The shortening of both long and short axes was smaller than in normal control, and so the contraction of the left ventricle was disturbed. By angiocardiography the long axis of the left ventricle could be determined as well as the short axis, while the long axis could not be determined by echocardiography. Therefore, it favors angiocardiography to see the contraction of the left ventricle.

By angiocardiography asynergy of the left ventricular wall and mitral regurgitation were observed frequently. Although the pathogenesis of the mitral regurgitation could not be investigated in this study, it might be included by the dilatation of the left ventricle³⁹ in CCM and by anterior movement of the anterior mitral leaflet in HCM.⁴⁰

Recently echocardiography has been popular for the diagnosis of cardiomyopathy, though it is a non-invasive method and it can detect easily the state of interventricular septum and mitral leaflets, it cannot determine the whole shape of the left ventricle, especially its long axis and mitral regurgitation. In these points angiocardiography is better than echocardiography.

In coronary cineangiograms there was neither stenosis nor dilatation of coronary arteries in cases of HCM as well as of CCM. And moreover some cases showed rather a hypervascularity. Recently, Pichard⁴¹ reported that the septal branches of the left anterior descending artery was occluded or narrowed during systole in patients with HOCM. However, it could not be observed in this study.

Histological findings of biopsied myocardium of right ventricle (31 specimens) and left ventricle (25 specimens) in 40 cases with cardiomyopathy revealed hypertrophy of myofibers, degenerative changes, i.e. scarcity of myofibrils, deformity of nucleus and vacuolization of myocardial fibers, and collagen proliferation. Those were found in the ventricular myocardium of cases with HCM as well as CCM. As shown in Table III, there were statistically no differences in intensity of the histological appearances between both groups. However, it seemed that

the degenerative changes of the myocardial fibers and interstitial fibrosis were more severe in CCM than in HCM, when observed precisely in individual case. In fact, other report in our clinic,¹¹ in which number of cases examined was increased and Wilcoxon's rank sum test and canonical discriminant analysis were used in statistics, revealed a trend of these differences.

In regard to the relationship of histological features of the biopsied left ventricle to the parameters of hemodynamics and angiocardiograms, such as LVEDP, Dd, LVEDV, meanVcf and LV-mass, there appeared no correlation between them. Only wall thickness of the left ventricle seemed to show a positive relation with the diameter of myocardial fibers. This appeared to mean that wall thickness of the left ventricle might be due to a hypertrophy of the myocardial fibers. It had been expected that hypertrophy of myocardial fibers should induce an increase in pump function of the left ventricle such as ejection fraction and meanVcf and that degenerative changes and fibrosis of the myocardium might produce the contrary. Biopsy specimens were however too small to indicate the state of the whole ventricle. This seemed to be the reason that there were no correlation between morphologic and functional findings of the heart.

SUMMARY

Cardiac catheterization, angiocardiography and ventricular muscle biopsy were performed in forty patients with idiopathic cardiomyopathy and included 21 cases of hypertrophic cardiomyopathy and 19 cases of congestive cardiomyopathy.

1) Cardiac catheterization revealed normal cardiac index and stroke index in both types, although there was a slight tendency toward decrease in cases of CCM. HCM showed slightly elevated right ventricular end-diastolic pressure and left ventricular end-diastolic pressure with a high incidence of atrial kick. CCM showed an elevated mean pulmonary artery, mean pulmonary wedge and left ventricular end-diastolic pressure.

2) Angiocardiographic findings revealed that in HCM left ventricular end-diastolic volume as well as left ventricular end-systolic volume, ejection fraction, meanVcf and MNSER were within normal range, and left ventricular anterior wall thickness, left ventricular mass and shortening of short axis in systole were increased.

In CCM left ventricular end-diastolic volume and end-systolic volume increased, and ejection fraction, mean Vcf, MNSE were decreased. The left ventricular anterior wall thickness was normal, and the left ventricular mass was smaller compared to the volume. The shortening of long and short axes in systole was slight. Left ventricular asynergy and mitral regurgitation occurred frequently. Coronary cineangiograms revealed normal patterns in both types.

3) Histological findings revealed hypertrophy of myofibers, degenerative changes, i.e. scarcity of myofibrils, deformity of nucleus and vacuolization of myocardial fibers, and collagen proliferation in both types.

4) No definite relationship was seen between parameters of left ventricular function and the findings of biopsied left ventricular muscle except for increase in wall thickness which might be apparently due to hypertrophy of the myocardial fibers.

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