which different diagnostic criteria for the V/Q scan can be applied. In patients with no prior cardiopulmonary disease, fewer mismatched perfusion defects indicate a higher probability of PE than among patients with prior cardiopulmonary disease. Among patients with no prior cardiopulmonary disease, the use of fewer mismatched perfusion defects for a high probability assessment results in a higher sensitvity with no reduction of the specificity or positive predictive value (*2,3*). The same number of mismatched perfusion defects in a patient with prior cardiopulmonary disease results in a lower probability of PE.

CONCLUSION

The data presented here strengthen the validity of stratification according to prior cardiopulmonary disease for a better diagnostic interpretation of the V/Q lung scan.

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Myocardial Metabolic Changes in Hypertrophic Cardiomyopathy

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We evaluated myocardial blood flow, glucose and oxygen metabolism using PET in hypertrophic cardiomyopathy (HCM). **Methods:** PET studies using ^{18}F-fluorodeoxyglucose (FDG) and ^{11}C-acetate were performed at rest in patients with HCM and normal subjects as a control group. The metabolic rate of glucose (MRGlu), K mono value as a marker of oxidative metabolism, and myocardial blood flow were estimated from serial dynamic FDG and ^{11}C-acetate PET studies. **Results:** Myocardial blood flow (%) did not differ significantly in hypertrophic and nonhypertrophic myocardium (90.3 ± 3.1 versus 91.7 ± 3.4). The MRGlu in hypertrophic myocardium, however, was lower than that in nonhypertrophic and normal myocardium (0.44 ± 0.10 versus 0.52 ± 0.15 and 0.53 ± 0.15 μmole/min/g, respectively, $p < 0.05$). The K mono values were also lower in hypertrophic myocardium than in nonypertrophic and normal myocardium (0.05 ± 0.010 versus 0.066 ± 0.0011 and 0.065 ± 0.017 per min, respectively, $p < 0.05$). The %FDG/%perfusion values in hypertrophic myocardium did not differ significantly from those in nonhypertrophic myocardium (0.96 ± 0.10 versus 1.02 ± 0.07). **Conclusion:** Myocardial ischemia at rest is observed less frequently in patients with HCM. Impairment of oxidative and glucose metabolism may precede decreased blood flow. Primary metabolic impairment is considered to be dominant in hypertrophic myocardium.

Key Words: PET; hypertrophic cardiomyopathy; myocardial metabolism; carbon-11-acetate; fluorine-18-FDG

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Hypertrophic cardiomyopathy is a primary myocardial disease of unknown etiology and pathogenesis (*1,2*). Patients with hypertrophic cardiomyopathy often complain of chest pain. Exercise studies using ^{201}Tl have also suggested myocardial perfusion abnormalities mainly in the hypertrophied myocardium in these patients (*3,4*). Hemodynamic and metabolic evidence of pacing-induced myocardial ischemia was also demonstrated in such patients (*5,6*). Camici et al. (*7*) also suggested that coronary vasodilator reserve is impaired in patients based on ^{13}N-ammonia PET studies. Thus, it is generally accepted that flow reserve impairment is present in patients with hypertrophic cardiomyopathy.

Whether true ischemia at rest is present in patients with hypertrophic cardiomyopathy and contributes to pathogenesis remains controversial. In ischemic coronary heart disease, preserved ^{18}F-fluorodeoxyglucose (FDG) uptake in the segments with reduced myocardial perfusion, known as perfusion-metabolism mismatch, has been proposed as a marker of ischemic but viable myocardium (*8–11*). Some investigations based on PET findings using ^{13}N-ammonia and FDG suggest the presence, and others the absence, of ischemia in patients (*12,13*). In addition, metabolic alteration, including oxidative metabolism, has not been fully investigated in these patients.

Therefore, the purposes of this study were to evaluate both the presence or absence of resting ischemia and energy alterations, including glucose and oxidative metabolism, in hypertrophic myocardium.

METHODS

Subjects

The study group consisted of 20 patients (10 men, 10 women; aged 13–82 yr; mean 42.6 yr). Hypertrophic cardiomyopathy was defined as hypertrophied and nondilated left ventricle in the absence of any other cardiac or systemic disease that itself produces left ventricular hypertrophy (*14,15*). The diagnosis of hypertrophic cardiomyopathy was made based on the clinical course and the results of echocardiography, electrocardiography

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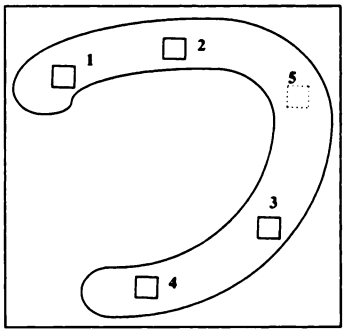

FIGURE 1. Midventricular transaxial slice illustrates the definition of four ROIs in patients with asymmetrical septal hypertrophy. Another ROI (5) was placed in the anterior segment in patients with apical hypertrophy.

(ECG), MRI and left ventriculography. Among these patients, 11 patients had a family history of hypertrophic cardiomyopathy and 10 had some symptoms, such as exertional angina or occasional shortness of breath. No patient had a history of diabetes. Patients with outflow obstruction were excluded from this study. As a control, FDG-PET studies were performed in 10 normal volunteers (9 men, 1 woman) aged 32–59 yr (mean: 44.0 yr) and ^{11}C-acetate PET studies in 7 normal male volunteers aged 23–41 yr (mean: 34.7 yr). None of the volunteers had a history of cardiac disease, hypertension, diabetes or any other cardiac symptoms. Each subject gave written informed consent and was approved by the Ethics Committee of Kyoto University.

Echocardiographic Studies

Transducers of 2.5 and 3.5 MHz were used in the echocardiographic studies. M-mode echocardiography was performed on parasternal long- and short-axis views and apical two- and four-chamber views. The M-mode echocardiographic measurements were performed according to the criteria recommended by the American Society of Echocardiography (*16*). Wall measurements of the septum and lateral wall on a plane which was thought to correspond to the midventricle were made after various views were observed.

If the end-diastolic thickness of the septum was at least 15 mm and its ratio to that of the left ventricular posterior wall was at least 1.3, asymmetrical septal hypertrophy was considered to be present (*17*). If echocardiographic apical long-axis or four-chamber views demonstrated apical hypertrophy and a spade-like configuration was also found in the right anterior oblique ventriculogram at end-diastole, apical hypertrophy was considered to be present (*18*).

Hypertrophy of the anterior septum or the posterior free wall was assessed as present when diastolic wall thickness was ≥15mm; hypertrophy of the posterior septum and lateral free wall was defined as a diastolic thickness ≥17mm (*13,19*).

MRI

MRI was performed with the use of a 1.5-T superconducting magnet in ten patients, two of whom had APH. Images were obtained in an ECG-gated spin-echo sequence. After an initial multislice scan to localize the heart in the thorax, a midventricular transaxial plane was selected. This scanning was performed with two measurements: a 128 × 256 matrix and a 5-mm slice thickness. Scans were repeated with different delay times after the R-wave to obtain systolic and diastolic frames (314 and 4 msec, respectively, after the R-wave). A double spin-echo pulse sequence with an echo delay of 25/50 msec was used for data acquisition. The repetition times were equivalent to the R-R interval of the ECG, which ranged from 600 to 1300 msec. In the two patients with APH, wall measurements, including the anterior wall, were performed based on MRI.

PET

PET studies were performed using a whole-body PET camera which provided 15 simultaneous slices at 7-mm intervals. The scanner has an effective resolution of 9 mm and an axial resolution of 7 mm FWHM after reconstruction (*20*). Each subject was positioned in the gantry of the PET camera with the aid of an ultrasound technique. At the beginning of the study, a transmission scan was obtained for 20 min using a ^{68}Ge/^{68}Ga external source to correct for photon attenuation.

Approximately 185 MBq (5 mCi) of FDG were administered at rest 40–60 min after a 75-g oral glucose load. Serial dynamic PET scans (30 sec × 8 frames, 4 min × 12 frames) were obtained after FDG administration. FDG static images were reconstructed from the last three frames (between 40 and 52 min postinjection).

On a different day, serial PET imaging was performed at rest after slow infusion of 370 MBq (10 mCi) of ^{11}C-acetate over 30–40 sec, during which 20 frames of 60 sec each for 20 min were acquired to assess oxidative metabolism and perfusion.

Data Analysis

Four regions of interests (ROIs) were placed over the left midventricular myocardium when asymmetrical septal hypertrophy was present (Fig. 1) and on the anterior wall when apical hypertrophy was present. These regions were divided into hypertrophic and nonhypertrophic segments based on the echocardiographic and MRI findings. In the analysis of ^{11}C-acetate PET studies, the time-activity curves of the corresponding four or five segments were generated from serial PET images after correction for deadtime, physical decay and cross-contamination (*20–22*). The clearance rate constant (K mono) (/min) of each segment was calculated from these time-activity curves by monoexponential fitting as a marker of oxidative metabolism (*20*). In addition, the washout rate of ^{11}C-acetate from 8 min to 20 min was calculated to generate a ^{11}C-acetate washout map. The regional perfusion was estimated from the peak activity (*23*) and perfusion images were reconstructed from the frame with the peak myocardial activities (usually 120 to 180 sec after tracer injection).

In the analysis of FDG-PET studies, regional metabolic rate of glucose (MRGlu) (μmole/min/g) in each segment was measured using Patlak graphic analysis from serial myocardial and blood-pool activities obtained from serial dynamic scans after FDG administration (*24,25*). FDG uptake between 40 and 52 min postinjection was also measured. After measuring wall thickness, the partial volume effect was corrected according to the recovery coefficient for myocardial perfusion calculation, MRGlu, and FDG uptake in each segment (*25–27*). After correcting for partial volume effects, myocardial perfusion and FDG uptake were normalized (%) to the peak values among the ROI. Furthermore, the percent FDG uptake was divided by the percent perfusion in order to calculate the FDG/perfusion percentage values. Perfusion-metabolism mismatch was considered to be present when the FDG-to-perfusion percentage ratio was above mean + 22 s.d. of the normal value determined in the normal subjects studied with FDG and ^{11}C-acetate.

Comparisons of these parameters were conducted between symptomatic and nonsymptomatic patients and between patients with and without family history of hypertrophic cardiomyopathy. In addition, these parameters were also compared according to age (i.e., patients <50 yr of age (younger group, n = 11) and those ≥50 yr (older group, n = 9).

Statistical Analysis

The mean values of all parameters such as the percent perfusion, percent FDG, MRGlu, K mono, and FDG-to-perfusion percentage ratio in hypertrophic and nonhypertrophic segments in each patient were calculated. The data are presented as mean ± s.d. The statistical significance of difference in mean values between two groups was analyzed with either the paired or unpaired Student's t-test. When the test groups were not normally distributed, the

TABLE 1
Patient Profiles

Patient no.	Age (yr)	Sex	Type	IVSth (mm)	LVPWth (mm)	LVDd (mm)	LVDs (mm)	LVEF (%)	HR (bpm)	SP (mmHg)	RPP (×0.01)	Glucose (mg/dl)	Insulin (μU/ml)	NEFA (μEq/liter)	Symp	FH
1	50	F	APH	10	11	50	33	63	60	100	60.0	136	39.7	178	–	–
2	61	M	APH	13	14	48	28	72	98	110	107.8	158	22.8	131	+	–
3	31	M	ASH	22	12	41	25	75	65	124	80.6	NP	NP	NP	+	–
4	38	M	ASH	20	12	44	29	66	55	108	59.4	157	109.1	272	–	+
5	45	M	ASH	16	11	54	37	59	55	116	63.8	161	61.7	312	+	+
6	62	M	ASH	17	10	45	28	80	62	134	83.1	157	59.4	306	–	–
7	82	M	ASH	20	12	39	17	82	60	146	87.6	139	54.8	698	+	–
8	58	M	ASH	22	13	58	26	50	60	94	56.4	128	35.5	246	–	+
9	47	F	ASH	17	12	59	46	52	62	120	74.4	117	32.6	96	–	+
10	46	F	ASH	20	11	41	25	68	58	116	67.3	117	52.5	310	+	+
11	13	F	ASH	28	11	49	31	76	71	102	72.4	158	20.3	187	–	+
12	50	M	ASH	15	11	44	25	75	56	136	76.2	148	20.1	520	–	–
13	17	M	ASH	16	12	47	29	78	82	105	86.1	142	110.2	545	–	+
14	63	F	ASH	15	11	40	23	74	54	109	58.9	157	42.0	156	+	+
15	63	F	ASH	18	8	38	22	73	64	119	76.2	143	85.8	234	+	+
16	14	M	ASH	15	9	38	21	83	70	114	79.8	126	34.6	760	–	–
17	19	F	ASH	15	8	52	25	71	78	97	75.7	130	37.8	106	–	–
18	26	F	ASH	15	10	60	44	60	69	109	75.2	101	25.6	102	+	–
19	24	F	ASH	15	9	51	36	65	44	88	38.7	99	32.6	214	+	+
20	60	F	ASH	18	10	41	22	78	54	93	50.2	183	34.7	202	+	+

APH = apical hypertrophy; ASH = asymmetrical septal hypertrophy; IVSth = end-diastolic thickness of the septum; LVPWth = end-diastolic thickness of the posterior wall; LVDd = end-diastolic left ventricular dimension; LVDs = end-systolic left ventricular dimension; LVEF = left ventricular ejection fraction; HR = heart rate; SP = systolic pressure; RPP = rate-pressure product; NEFA = non-esterified fatty acid; Symp = symptom; FH = family history; NP = not performed.

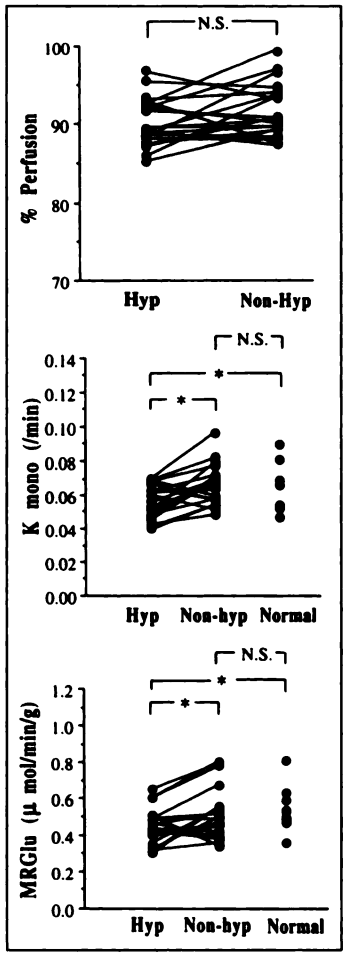

FIGURE 2. (A) Comparisons of perfusion percentages in hypertrophic and nonhypertrophic myocardium. (B, C) Comparisons of K mono (min) values and MRGlu (μmole/min/g) in hypertrophic, nonhypertrophic and normal myocardium of the healthy volunteers ($p < 0.05$).

paired or unpaired nonparametric t-test was applied. Significant differences were considered to be present when p value was less than 0.05.

RESULTS

Table 1 shows patient profiles, plasma substrate levels at the time of FDG-PET studies and hemodynamic data obtained at the time of acetate PET studies. There were no significant differences between these patients and the normal volunteers in the plasma concentrations of glucose (139.8 ± 21.9 versus 139.9 ± 15.9 mg/dl), insulin (48.0 ± 27.2 versus 50.1 ± 28.1 μU/ml), and nonesterified fatty acid (277.6 ± 162.6 versus 314 ± 241.2 μEq/l). The heart rate (65.9 ± 10.5 versus 62.0 ± 5.0 bpm), the systolic blood pressure (112.0 ± 15.1 versus 120.0 ± 5.0 mm Hg), and rate-pressure product (7360 ± 1410 versus 7500 ± 1260) also did not show any significant differences in these two groups.

Wall Measurements by Echocardiography and MRI

Wall thickness measurements in two septal and lateral regions were performed by both echocardiography and MRI in ten subjects (eight patients with ASH and two patients with APH). The wall thickness measurements (mm) by echocardiography (y) and MRI (x) were linearly well correlated with each other ($y = 1.2 \pm 0.95x$, $R = 0.88$).

Regional Perfusion, MRGlu and K mono in HCM

The regional perfusion (%) did not show a significant difference between the hypertrophic and the non-hypertrophic segments (90.3 ± 3.1 versus 91.7 ± 3.4, respectively) (Fig. 2A). The K mono value in the hypertrophic segments was lower than that in either the non-hypertrophic segments or the normal myocardium of the healthy volunteers (0.056 ± 0.010 versus 0.066 ± 0.011 and 0.065 ± 0.017, respectively, $p < 0.05$) (Fig. 2B), and MRGlu was also

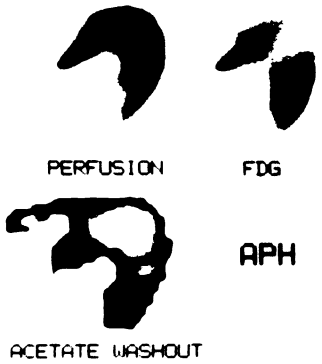

FIGURE 3. Transaxial images of perfusion, FDG and acetate washout map for Patient 2 who had apical hypertrophy. Perfusion was preserved in the hypertrophied apical region, but FDG uptake was decreased and acetate washout was also delayed, suggesting impaired glucose and oxygen metabolism in this region.

lower in the hypertrophic segments (0.44 ± 0.10 versus 0.52 ± 0.15 and 0.53 ± 0.15, respectively, $p < 0.05$) (Fig. 2C). Neither the K mono value nor MRGlu in the nonhypertrophic segments differed from that in the normal myocardium.

FDG-to-Perfusion Percentage Ratios in HCM

The mean FDG-to-perfusion percentage ratios in hypertrophic segments did not differ significantly from those in the nonhypertrophic segments (0.96 ± 0.10 versus 1.02 ± 0.07, respectively).

No patient showed perfusion-metabolism mismatch patterns which might have suggested the presence of ischemia. Perfusion-metabolism mismatch pattern was not observed in hypertrophic cardiomyopathy and, furthermore, some of the segments showed impaired glucose utilization (Figs. 3 and 4).

Symptom-Related Differences

Ten patients had cardiac symptoms such as exertional angina or occasional shortness of breath. The myocardial blood flow percentage, MRGlu, FDG-to-perfusion percentage ratio and K mono of the hypertrophic myocardium in the symptomatic patients, however, did not differ significantly from those in the nonsymptomatic patients (90.2 ± 3.2 versus 90.4 ± 3.1, 0.47 ± 0.10 versus 0.41 ± 0.10, 0.97 ± 0.11 versus 0.95 ± 0.09 and 0.060 ± 0.011 versus 0.052 ± 0.009, respectively). These parameters in the nonhypertrophic myocardium in the symptomatic patients did not differ from those in the non-symptomatic patients (91.3 ± 3.2 versus 92.1 ± 3.6, 0.53 ± 0.17 versus 0.52 ± 0.13, 1.03 ± 0.05 versus 1.00 ± 0.09 and 0.069 ± 0.011 versus 0.063 ± 0.008, respectively).

Family History-Related Differences

Eleven patients had family history of hypertrophic cardiomyopathy. The myocardial blood flow percentage, MRGlu, FDG-to-perfusion percentage ratio and K mono values of the hypertrophic myocardium in patients with family history did not show any significant differences from those in the patients without (90.1 ± 3.0 versus 90.5 ± 3.3, 0.44 ± 0.09 versus 0.45 ± 0.12, 0.94 ± 0.10 versus 0.99 ± 0.10 and 0.051 ± 0.010 versus 0.059 ± 0.010, respectively). Similarly, these parameters for the nonhypertrophic myocardium did not differ in these two patient groups (88.6 ± 4.5 versus 90.6 ± 4.2, 0.51 ± 0.17 versus 0.53 ± 0.13, 1.01 ± 0.07 versus 1.02 ± 0.08, and 0.068 ± 0.013 versus 0.063 ± 0.010, respectively).

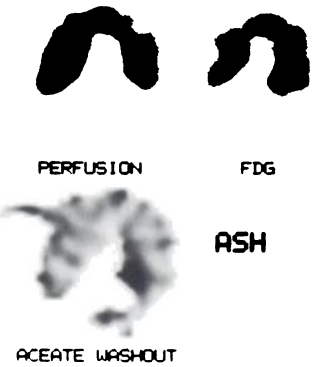

FIGURE 4. Transaxial images of perfusion, FDG and acetate washout map for Patient 8 who had asymmetrical septal hypertrophy. Perfusion was preserved in the hypertrophied septal region, while FDG uptake was slightly decreased. Acetate washout was also delayed in this region when compared to the nonhypertrophic posterolateral region.

Age-Related Differences

The myocardial blood flow percentage, MRGlu, FDG-to-perfusion percentage ratios and K mono values of the hypertrophic myocardium did not differ significantly between the younger (age <50 yr) and the older (age ≥50 yr) patient groups (90.3 ± 3.5 versus 90.2 ± 2.8, 0.45 ± 0.10 versus 0.43 ± 0.10, 0.96 ± 0.14 versus 0.96 ± 0.06, 0.056 ± 0.011 versus 0.056 ± 0.010, respectively). Similarly, these parameters for the nonhypertrophic myocardium showed no difference in these two patient groups (92.3 ± 3.7 versus 91.2 ± 3.2, 0.52 ± 0.15 versus 0.52 ± 0.15, 1.00 ± 0.07 versus 1.03 ± 0.07, 0.068 ± 0.014 versus 0.064 ± 0.010, respectively, p = ns).

DISCUSSION

The present study demonstrates that hypertrophic cardiomyopathy showed that both oxidative and glucose metabolism were often impaired in these patients, particularly in the hypertrophic myocardium, while the myocardial perfusion at rest was not significantly decreased. In the nonhypertrophic myocardium, however, myocardial metabolism was preserved compared with the normal myocardium. These data indicate that absolute metabolic changes may exist mainly in the hypertrophic myocardium.

Metabolic Alteration in Hypertrophic Cardiomyopathy

Increased glucose utilization is a hallmark of ischemic myocardium and FDG was proposed for use in delineating viable but dysfunctional myocardium. PET using ^{18}F-FDG has been used in the detection of ischemia and differentiation of ischemic but viable myocardium from irreversible myocardial necrosis in coronary artery disease (*10,28,29*). Thus, perfusion-metabolism mismatch is proposed as the criteria of PET ischemia. On the other hand, previous investigations indicated that preservation of oxidative metabolism in an abnormal region was necessary for function recovery after recanalization in chronic coronary artery disease (*30*). On the basis of these concepts, fibrotic change is considered to be dominant in the hypertrophic myocardium because both oxidative metabolism and glucose metabolism were impaired. These findings may reflect the presence of fibrosis in hypertrophic cardiomyopathy, a fact which was suggested in previous morphological investigations (*31–33*).

Myocardial Perfusion Estimated by Carbon-11-Acetate

Gropler et al. (*23*) estimated myocardial perfusion using 120-second data beginning 60 sec after bolus intravenous injection of ^{11}C-acetate. The time interval was chosen based on their observation in normal subjects of: (a) minimal myocardial clearance of myocardial activity within the first 4 min following bolus administration of ^{11}C-acetate and (b) an adequate differential ratio of myocardial activity to blood-pool activity by 60 sec postadministration of the tracer. In our study, however, ^{11}C-acetate was infused slowly over a period of 30 to 40 sec. Based on our observation in normal subjects, therefore, a frame showing peak myocardial activity (usually 120 or 180 sec after tracer injection) was selected for assessing relative myocardial perfusion. In fact, the percent myocardial perfusion obtained with our method was well correlated linearly with that obtained by ^{13}N-ammonia PET studies in nine patients with coronary artery disease (CAD) and four patients with hypertrophic cardiomyopathy. Therefore, we consider that relative myocardial perfusion can be assessed using our method not only in

patients with CAD but also in those with hypertrophic cardiomyopathy.

Myocardial Ischemia in Hypertrophic Cardiomyopathy

Chest discomfort or angina pectoris is frequently observed in patients with hypertrophic cardiomyopathy. The several possible mechanisms that have been advanced to account for this symptom include small vessel disease in the thickened ventricular septum (*34,35*), septal perforator artery compression (*36*), coronary artery spasm (*37*) and inadequate capillary density in relation to the increased myocardial mass present in hypertrophic cardiomyopathy (*5*). In fact, there are many reports indicating perfusion abnormalities after exercise (*3,4,13*), during pacing-induced tachycardia (*5,6*) and after pharmacological vasodilatation (*7*). Thus, it is generally accepted that flow reserve is impaired in patients with hypertrophic cardiomyopathy. In the resting state, however, Camici et al. reported that myocardial blood flow is increased in the hypertrophied septum compared with the nonhypertrophied myocardium (*7*). Nienaber et al. (*13*) demonstrated that the blood flow decreases in the hypertrophied myocardium. In this study, myocardial blood flow at rest was slightly decreased in the hypertrophic myocardium compared to nonhypertrophic myocardium but the difference did not show statistical significance. Thus, myocardial blood flow is relatively homogenous throughout left ventricular myocardium in hypertrophic cardiomyopathy. In addition, no patient showed perfusion-metabolism mismatch patterns. These data indicate that ischemia may less likely be observed in a resting condition. The myocardial blood flow and FDG-to-perfusion percentage ratios in the hypertrophic myocardium did not differ significantly in the symptomatic and non-symptomatic patients. From these findings, it is considered that the myocardium with hypertrophic cardiomyopathy is not always exposed to ischemia.

Nienaber et al. (*10*) indicated the presence of PET ischemia in symptomatic hypertrophic cardiomyopathy. Conversely, Grover-Mckay et al. (*12*) noted no PET ischemia in mildly symptomatic patients. The present study of symptomatic patients, however, did not indicate ischemia. There was no significant metabolic difference between symptomatic and non-symptomatic patients. The symptomatic patients may have had myocardial ischemia at the cellular or subcellular levels, but the amount of ischemic tissue may not have been large enough to be identified by PET. Such discordant findings between subjective symptoms and the metabolic alterations delineated by PET have occasionally been observed in patients with ischemic heart disease. These findings suggest that the metabolism in hypertrophic cardiomyopathy is more complicated and that such metabolic alteration cannot be explained simply in terms of ischemia. Some primary myocardial damage rather than ischemia may cause metabolic alterations as well as such symptoms as angina pectoris. Further evaluation is needed to determine the clinical significance of abnormalities in myocardial metabolism without decreased perfusion.

Metabolic Alteration in Relation to Age and Family History

In this study, metabolic alterations did not differ in the younger and the older patient groups, or in the patients with and without family history of hypertrophic cardiomyopathy. These results suggest that the metabolic alteration in hypertrophic cardiomyopathy cannot simply be explained based on genetic or aging factors. These factors, and probably some other unknown ones, may influence the myocardial metabolic alterations and symptoms in a complex manner.

Limitations

Our study has several limitations. The partial volume effect causes underestimation of regional tissue activities when the thickness of the myocardial wall is less than twice the spatial resolution of the imaging device (*26*). In this study, the partial volume effect was corrected mainly based on echocardiographic findings. In ten patients, MR images were also obtained. The wall thickness obtained from the echocardiographic measurements was correlated well with the thickness obtained with MRI. The sites of wall thickness measurements were carefully matched with the placement of the ROI. Nevertheless, slight misalignments could be observed. However, the ratio of FDG-to-perfusion percentages would eliminate the effects of regional partial volume because this index calculates values within the same region.

The variability of MRGlu is notable and is caused mainly by interpatient variability (*38*). Therefore, there might be some difficulty in comparing normal myocardium and myocardium with hypertrophic cardiomyopathy. In addition, possible changes in lumped constants in individual patients cannot be accounted for in such pathological myocardial tissue as hypertrophic cardiomyopathy.

In this study, absolute regional flow data were not available. Therefore we are not able to compare the absolute flow in hypertrophic cardiomyopathy with that in the absolute normal myocardium. A dynamic acquisition of blood pool and myocardial activities of ^{15}O-water or ^{13}N-labeled ammonia may permit absolute measurement of myocardial blood flow (*7,39–41*).

This study was performed under resting conditions. Our data, however, do not rule out metabolic abnormalities occurring during exercise.

Finally, we studied only 20 patients with hypertrophic cardiomyopathy. The number of patients and segments in this study may be too small to elucidate metabolic and perfusion abnormalities. Further investigation is needed to verify whether our results are valid.

CONCLUSION

Impairment of oxidative metabolism and glucose metabolism was observed mainly in the hypertrophic myocardium in patients with hypertrophic cardiomyopathy. Primary metabolic impairment, rather than ischemia, is therefore considered to be dominant in hypertrophic cardiomyopathy.

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Evaluation of Individual Criteria for Low Probability Interpretation of Ventilation-Perfusion Lung Scans

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The purpose of this investigation was to identify characteristics or combinations of characteristics of the ventilation-perfusion (V/Q) scan in patients with suspected acute pulmonary embolism (PE) that can be used for a "very low probability" interpretation (<10% positive predictive value). **Methods:** Data were culled from individual lungs of 532 patients in the randomized arm of the Prospective Investigation of Pulmonary Embolism Diagnosis (PIOPED) study and 205 patients in the referred arm. All patients had a <20% consensus probability estimate of PE based on V/Q scan results, and all underwent pulmonary angiography. **Results:** Nonsegmental perfusion abnormalities, perfusion defects smaller than opacities on the chest radiograph, a combination of these types of perfusion abnormalities, and matched V/Q abnormalities in two or three zones of a single lung had a positive predictive value < 10%. These criteria can therefore be used for a very low probability interpretation. A matched V/Q defect in only one zone of the lung had a positive predictive value greater than 10% and is not a criterion for very low probability classification but can be considered a criterion for low probability. Perfusion defects associated with small pleural effusions (obliteration of the costophrenic angle) had a positive predictive value of 25%–33%, depending on the group studied, and are a criterion for intermediate probability. **Conclusion:** Criteria appropriate for very low probability (<10% positive predictive value) interpretation of V/Q scans in patients with suspected acute PE have been identified.

Key Words: pulmonary embolism; thromboembolism; ventilation-perfusion lung scans

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