Iodine-123-BMIPP Myocardial Washout and Cardiac Work During Exercise in Normal and Ischemic Hearts

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Iodine-123-BMIPP washout at rest and after exercise was investigated in relation to cardiac work in normal and ischemic myocardium. **Methods:** Sixteen healthy volunteers and 10 patients with ischemic heart disease were examined. After injection of 111 MBq of ^{123}I-BMIPP, successive SPECT imaging was performed to evaluate washout ratio (WR) at rest and after mild and maximal exercise. **Results:** In the healthy volunteers, the mean WR was 3.3 ± 3.5% after 1 hr of rest and increased during both mild and maximal exercise. The WR showed a better correlation with net pressure rate product (PRP) than with the peak PRP. After maximal exercise, the WR showed a distinct correlation with the net PRP in the range from 200–300 × 10^3 mmHg/min and then showed a plateau greater than 10%. In five ischemic heart disease patients with the net PRP ≥ 300 × 10^3 mmHg/min, the exercise WR values were significantly elevated in normal segments relative to ischemic segments. **Conclusion:** BMIPP washout increased with the increase in cardiac work during exercise in normal myocardium but not in ischemic myocardium. A certain amount of exercise is necessary to evaluate fatty acid metabolism in normal and ischemic myocardium.

Key Words: ^{123}I-BMIPP; washout ratio; pressure rate product; ischemic heart disease; SPECT

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Iodine-123-labeled 15-(*p*-iodophenyl)3-*R*,*S*-methylpentadecanoic acid (BMIPP) is a structurally modified fatty acid, which has been designed to reveal normal myocardial extraction but not to be readily catabolized through the oxidative pathway (*1*). Many published papers have described BMIPP's clinical usefulness in testing patients with ischemic heart disease (IHD) (*2–10*) and hypertrophic cardiomyopathy (*8,11–13*). Although BMIPP is protected from beta-oxidation by the methyl-branching, Dudczak et al. (*14*) first detected some BMIPP metabolites in blood and urine in a clinical study. Recently, several animal studies showed that BMIPP is partially metabolized in the heart through alpha-oxidation and decarboxylation and followed by beta-oxidation (*15–17*). Thus, there is a possibility that BMIPP is also partially metabolized in the human heart. The clinical investigation of BMIPP washout in the human heart is, therefore, important for the evaluation of fatty acid metabolism in normal and diseased myocardium. Although several papers evaluating the washout ratio (WR) of BMIPP at rest and after exercise in normal and ischemic myocardium have been published (*3,4*), the kinetics of BMIPP in the heart are not yet understood and the clinical usefulness of this assessment is still controversial. In the protocols they used, BMIPP was injected at peak exercise and followed by successive SPECT imaging to calculate exercise WR (*3,4*). We, however, interpret this to be postexercise WR rather than exercise WR. In this study we used a protocol in which BMIPP was injected before exercise was started. This protocol is designed based on the assumption that if BMIPP is metabolized in the myocardium, the radioactivity of BMIPP after incorporation and equilibration in the myocardium should decrease during exercise because of consumption.

The purpose of this study was to investigate: (a) how much the radioactivity of BMIPP in the heart decreases at rest and during exercise; (b) whether the WR values are increased with the increase of cardiac work during exercise; (c) whether the WR values are influenced by the type of workload (constant or multigrade); and (d) whether there is a difference in WR values between ischemic myocardium and normal myocardium.

MATERIALS AND METHODS

Subjects

Sixteen healthy volunteers and 10 patients with IHD were examined in this study. Before participation in the study, all subjects gave informed consent in accordance with the hospital Human Clinical Study Committee guidelines. The volunteers were physicians, technologists or employees in our hospitals; 15 men and one woman. Their ages ranged from 26–63 yr with a mean age of 39 yr. None of the volunteers had a history of cardiac disease or diabetus mellitus, and all showed blood chemistry within the normal range.

The 10 IHD patients included two with effort angina, two with acute myocardial infarction and six with old myocardial infarction. These patients had a ^{201}Tl stress myocardial study within 1 wk before this study and were selected because the ^{201}Tl SPECT images showed that they had abnormal myocardial perfusion.

Radiopharmaceuticals

Iodine-123-labeled BMIPP was commercially available. The formulation contained 111 MBq (3 mCi) ^{123}I-BMIPP (0.6 mg) dissolved in an aqueous solution (1 ml) of ursodeoxycholic acid (10.5 mg). The radiochemical purity was greater than 98%. The product showed a single, homogeneous radioactive component on thin-layer chromatography analysis.

Instrumentation

A three-headed SPECT system was used for SPECT imaging. For continuous data acquisition of the myocardial counts during exercise, a multicrystal gamma camera was used.

Study Protocol and Data Acquisition

Healthy Volunteers. After breakfast on the day of examination, all subjects were deprived of liquids and solid food until completion of the study. A 111-MBq dose of BMIPP was injected into the antecubital vein of each subject about 2 hr after breakfast. The initial SPECT imaging was started 30 min later. After 1 hr of rest, the second SPECT imaging was performed to calculate the resting WR in all subjects. Subsequently, 11 subjects performed mild and maximal exercise successively using a bicycle ergometer. Mild

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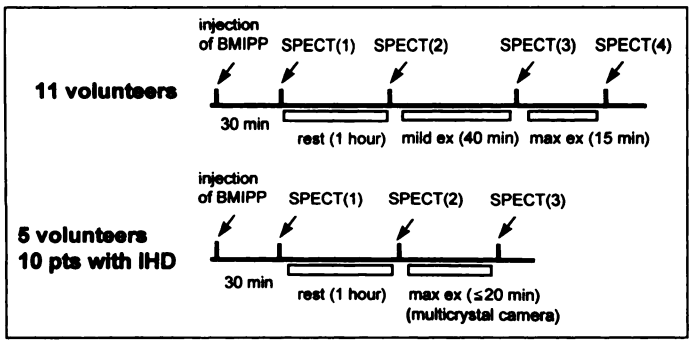

FIGURE 1. Protocols for healthy volunteers and patients with IHD. Ex = exercise; max = maximal.

exercise was performed for 40 min with a constant workload of 25 W, after which the third SPECT imaging was initiated. This was followed by maximal exercise for 15 min, starting with a workload of 50 W and increasing by 25 W every 3 min. After this, the fourth SPECT imaging was performed (Fig. 1).

The other five subjects performed only maximal exercise over a period of 20 min, starting with a workload of 50 W, increasing by 25 W every 5 min. Cardiac activity during exercise was monitored by successive 1-min data collection in these subjects using a multicrystal gamma camera.

Patient Study. The 10 patients with IHD performed only maximal exercise after the second SPECT imaging. They were divided into two groups, each with five subjects. The exercise was initiated at 50 W for both groups. For Group A, the workload increased by 25 W every 3 min, while for Group B, the workload increased by 25 W every 5 min. Exercise was terminated when chest pain, serious arrhythmia, ST depression (≥0.2 mV) and/or fatigue occurred.

The electrocardiogram, heart rate and blood pressure were monitored once per minute during exercise in the healthy volunteers as well as in the patients with IHD.

Iodine-123-BMIPP SPECT Imaging. Images were acquired in a continuous rotating mode using a high-resolution parallel-hole collimator with a 64 × 64 matrix. A total of 90 projection images was obtained over 360° in 4° increments with 20 sec per view. Projection data were collected from a main window (159 keV ± 12%) and two 3% scatter rejection windows. Scatter correction for each pixel was performed using the triple-energy window method (*18*). Construction of transaxial tomographic images was performed with a 128 × 128 matrix using Butterworth and ramp filters. The parameter of the Butterworth filter was of the order 8 for each dataset and the cutoff frequency was 0.28 cycle/pixel. Contiguous left ventricular short-axial slices, 12.8 mm in thickness, were reconstructed from apex to base.

Continuous Measurement of Myocardial Counts During Exercise. Continuous acquisition of myocardial counts was performed during exercise with the multicrystal gamma camera in the left anterior oblique view. The data were recorded by successive 1-min data collection and the time-activity curve of the left ventricle was obtained after background correction.

Thallium-201 SPECT Imaging. Stress SPECT imaging was started 10 min after 74 MBq of ^{201}Tl were injected at peak exercise. Three hours later, rest SPECT imaging was performed after reinjection of 37 MBq of ^{201}Tl.

Data Analysis

Washout Ratio of BMIPP. The myocardial BMIPP counts of each short-axial tomographic slice were measured by setting a region of interest precisely over the myocardium and total counts of the left ventricle (TCLV) were calculated by summation. TCLV was computed in each SPECT study with a physical decay correction. The WR values for BMIPP during rest, mild exercising and maximal exercising were calculated using the equation:

$$WR(\%) = \frac{(\text{TCLV before each work load change} - \text{TCLV after each work load change})}{\text{TCLV before each work load change}} \times 100. \quad \text{Eq. 1}$$

The pressure rate product (PRP) was calculated by multiplying the heart rate by the blood pressure once per minute and the peak PRP (maximum value of PRP during exercise) and the net PRP values (cumulative value of PRP during exercise) were obtained.

Statistical Analysis

The data were expressed as the mean ± s.d. A paired Student's t-test was used to compare WR at rest and after mild and maximal exercise in the healthy volunteers. For the patients with IHD, a paired Student's t-test was used to compare WR at rest and after exercise in the normal segments, and an unpaired Student's t-test was used to compare W R between normal and abnormal segments. Differences were considered significant when $p < 0.05$.

RESULTS

Results for Healthy Volunteers

Washout Ratio at Rest and After Mild and Maximal Exercise. Rest WR values per 60 min ranged from −2% to 10% with a mean of 3.3 ± 3.5% (n = 16). Mean WR values after mild and maximal exercise were 3.9 ± 3.4% (n = 11) and 8.8 ± 4.9% (n = 16), respectively. There were no significant differences among them. As compared with the mean rest WR values calculated per 40 and 15–20 min, corresponding to the period of each exercise, the mean WR after maximal exercise was significantly greater than mean rest WR values per 15–20 min (8.8 ± 4.9% vs. 0.8 ± 0.9%, $p < 0.01$).

Relationship Between Washout Ratio and Pressure Rate Product After Mild Exercise. The mean PRP at rest was 8.3 ± 1.6 × 10^3 mmHg/min and ranged from 6–12.2 × 10^3 mmHg/min. After mild exercise, the peak PRP showed a slight increase with a mean of 10.6 ± 1.5 × 10^3 mmHg/min, and correlated well with the WR ($r = 0.833$, $p < 0.01$) (Fig. 2A). Since the period of exercise was long, the net PRP showed fairly large values with a mean of 403.4 ± 71.1 × 10^3 mmHg/min and also correlated strongly with the WR ($r = 0.886$, $p < 0.01$) (Fig. 2B).

Relationship Between Washout Ratio and Pressure Rate Product After Maximal Exercise. After maximal exercise, the peak PRP showed a considerable increase with a mean of 26.8 ± 7.2 × 10^3 mmHg/min, but showed no significant correlation with the WR (Fig. 3A). The net PRP, on the other hand, showed a wide range of values with a mean of 294.6 ± 92.7 × 10^3 mmHg/min. While WR showed a nearly linear increase with the net PRP in the range from 200–300 × 10^3 mmHg/min ($r = 0.796$, $p < 0.01$, $n = 10$), only a mildly undulating plateau was observed within a 10–15% range (mean 12.5 ± 1.6%, n = 6) for net PRP values greater than 300 × 10^3 mmHg/min (Fig. 3B).

Continuous Measurement of Myocardial Counts During Exercise. The average time-activity curve of myocardial BMIPP activity during exercise and sequential change of mean net PRP values in five healthy volunteers is shown in Figure 4. Myocardial activity began to decrease rapidly around 5–6 min after initiation of the exercise (approximately 100 × 10^3 mmHg/min of the net PRP). The rapid decrease continued until 10–12 min and was followed by a slow decline with increased background activity.

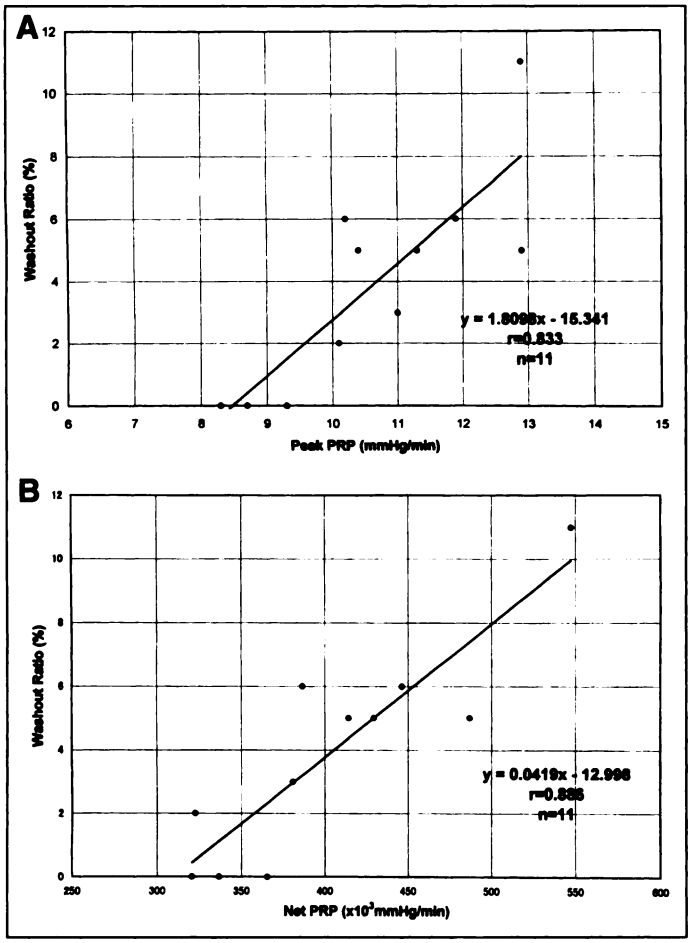

FIGURE 2. Relationship between the WR of BMIPP and (A) peak pressure rate product (PRP) and (B) net PRP after mild exercise in 11 healthy volunteers.

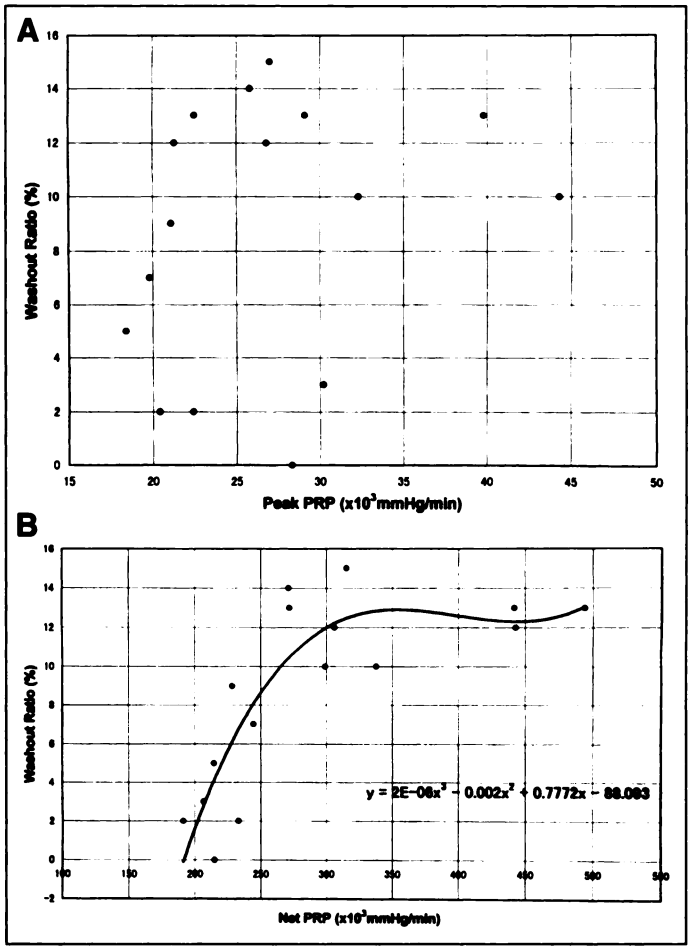

FIGURE 3. WR of BMIPP plotted against (A) peak PRP and (B) net PRP after maximal exercise in 16 healthy volunteers.

Results for Patients with Ischemic Heart Disease

Thallium-201 SPECT imaging showed 12 segments with abnormal perfusion in 10 patients (4 redistribution, 4 reversed redistribution, 4 decreased perfusion). The WRs of BMIPP at rest and after exercise in the segments with normal and abnormal perfusion are summarized in Table 1. In Group A, all patients terminated the exercise due to leg fatigue or chest pain before 15 min (mean duration of exercise was 8.2 ± 2.9 min) and thus their net PRP values were below 300×10^3 mmHg/min. The WRs of the segments with normal and abnormal perfusion did not differ significantly either at rest or after exercise. In Group B, by contrast, all patients successfully continued the exercise for 20 min and their net PRP values were greater than 300×10^3 mmHg/min. In the normal segments, the WR after exercise increased significantly greater than the rest WR (10.3 ± 2.4% vs. 3.5 ± 1.8%, $p < 0.002$), whereas in the abnormal segments the WR after exercise did not increase significantly relative to the rest WR (4.9 ± 3.7% vs. 3.6 ± 1.5%, ns). Thus, the exercise WR was significantly greater in the normal segments than in the abnormal segments (10.3 ± 2.4% vs. 4.9 ± 3.7%, $p < 0.03$).

A representative case of a 64-yr-old female patient with angina is presented in Figure 5. Thallium-201 SPECT images showed posterolateral ischemia. The BMIPP study was done 5 days later, when the patient performed maximal exercise corresponding to a net PRP of 371.8×10^3 mmHg/min after the protocol of Group B. WR at rest was similar in normal and ischemic segments (2.8% vs. 2.3%). After exercise, however, WR showed a marked increase to 7.9% in the normal segment without a corresponding increase in the ischemic segment (3.4%).

DISCUSSION

BMIPP Washout in Normal and Ischemic Myocardium

In this study, the mean WR of BMIPP after 1 hr of rest was 3.3 ± 3.5%. This is basically similar to previously reported values (13–19% over 3 hr) (*3,4,13*). During exercise, the WR values increased relative to cardiac work for constant mild exercise as well as for multigrade maximal exercise.

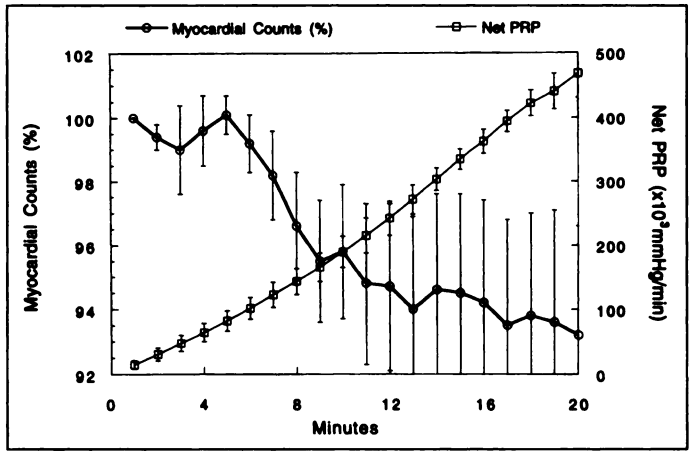

FIGURE 4. Sequential change of average myocardial BMIPP radioactivity and net PRP values during exercise in five healthy volunteers. Vertical bars represent mean ± s.d.

TABLE 1
BMIPP Washout Rate in Normal and Abnormal Perfusion Segments at Rest and After Exercise in Patients with Ischemic Heart Disease

		Washout ratio, %				
		Thallium-201 perfusion	Normal		Abnormal	
	n	Net PRP	Rest	Exercise	Rest	Exercise
Group A	5	105.3 ± 39.9	2.1 ± 1.1	4.1 ± 1.8	2.6 ± 2.0	2.9 ± 3.1
Group B	5	324.5 ± 29.3	3.5 ± 1.8	10.3 ± 2.4*	3.6 ± 1.5	4.9 ± 3.7

Net PRP: Net pressure rate product ($\times 10^3$ mmHg/min).
*$p < 0.002$ versus rest WR of normal segments in Group B and $p < 0.03$ versus WR after exercise of abnormal segments in Group B.

Peak PRP and net PRP were used as indexes of the cardiac work in this study because PRP has a close relationship with myocardial oxygen consumption (*19*). The exercise WR is consistent with the total myocardial loss of BMIPP radioactivity during the entire period of exercise. Since net PRP reflects the total oxygen consumption during the whole exercise more substantially compared with peak PRP, the correlation of net PRP with the WR of BMIPP after both mild and maximal exercise was better than that of peak PRP.

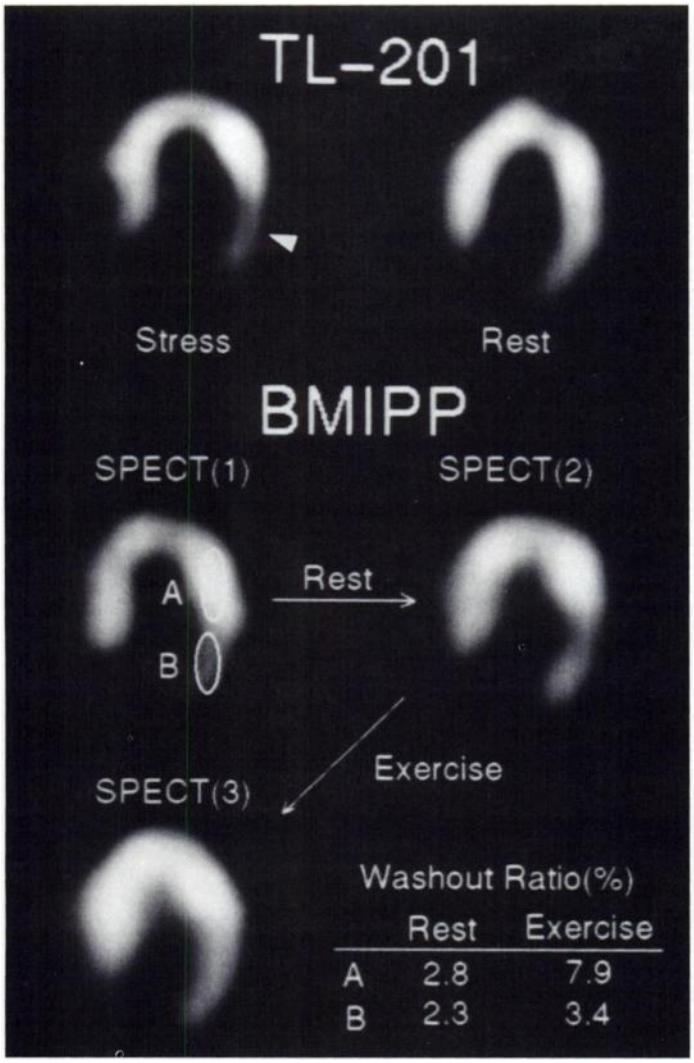

FIGURE 5. Thallium-201 and ^{123}I-BMIPP myocardial SPECT images of a 64-yr-old woman with angina. In the posterolateral wall (B), ischemia is demonstrated on a ^{201}Tl image (white arrowhead), but the WR of BMIPP does not increase relative to normal segments (A) after exercise. SPECT (1), SPECT (2) and SPECT (3) denote images obtained before rest, before exercise and after exercise, respectively.

The WR values increased relative to the net PRP but showed a plateau for net PRP greater than 300×10^3 mmHg/min at maximal exercise. Moreover, myocardial release of BMIPP radioactivity became slower 10–12 min after initiation of the exercise in contrast to the initial rapid decrease (Fig. 4). These two findings might be related to an increase of lactate in blood during exercise, which suppresses the fatty acid metabolism (*19*).

In ischemic myocardium, the WR values were similar to those of normal myocardium at rest with poor response to the exercise.

BMIPP Metabolism in Normal and Ischemic Myocardium. In normal myocardium, BMIPP is extensively incorporated into myocardial cells and substantially retained in a triglyceride pool. A certain fraction of BMIPP is washed out through the catabolism of BMIPP by alpha-oxidation followed by beta-oxidation and/or back diffusion of BMIPP itself (*14–16*). Fujibayashi et al. (*16*) reported that BMIPP was washed out mainly as alpha and beta-oxidation metabolites, with a little unchanged BMIPP released by back diffusion in normal canine hearts. In experimental cardiac ischemia, the WR of BMIPP as well as the alpha and beta-oxidation metabolites have been found to decrease, whereas back diffusion of BMIPP has been seen to increase (*20,21*). Based on these results, it seems reasonable to infer that the increase of BMIPP washout by exercise is caused by the intensified metabolism of BMIPP by alpha and beta-oxidation, which occurs in normal myocardium but not in ischemic myocardium. These findings are consistent with the fact that energy sources are converted from fatty acid to glucose or other substrates in ischemic myocardium (*19*).

Clinical Implication. Most BMIPP studies have been performed in comparison with blood flow assessment using ^{201}Tl or ^{99m}Tc-MIBI. The concordance or discordance between the two findings is an important factor in the evaluation of myocardial viability (*2–10*). However, these studies have some limitation because BMIPP uptake might be dependent on blood flow. In contrast to the uptake study, the WR is a parameter that is inherently independent of blood flow. In this study, in particular, the WR of BMIPP was evaluated after BMIPP was incorporated and equilibrated throughout the myocardium. Therefore, it seems reasonable to expect myocardial blood flow to have little effect on our assessment.

When patients continued the maximal exercise until the net PRP was more than 300×10^3 mmHg/min, the WR of BMIPP became greater than 10% in normal myocardium. This value was significantly greater than the rest WR in normal myocardium and the exercise WR in ischemic myocardium. To evaluate fatty acid metabolism in normal and ischemic myocardium, maximal exercise with net PRP $\geq 300 \times 10^3$ mmHg/min is required because the rest WR values are similar. Thus, a net PRP of 300×10^3 mmHg/min can be used as an index of the endpoint of the required exercise. To obtain a net PRP greater than 300×10^3 mmHg/min, exercise should be performed over a longer period, as in the protocol used with Group B patients. Patients with IHD might not be able to perform this protocol. However, all patients of Group B continued the exercise for 20 min, whereas nobody sustained the exercise in Group A. Therefore, even aged and/or seriously ill patients can perform this protocol more easily than the usual stress test.

CONCLUSION

In normal myocardium, BMIPP washout increased with the increase in cardiac work during exercise. In ischemic myocardium, on the other hand, the BMIPP WRs were similar to those in normal myocardium at rest and were not increased by exercise. A certain amount of maximal exercise (net PRP $\geq$ 300×10^3 mmHg/min) was thus found to be necessary to evaluate fatty acid metabolism in normal and ischemic myocardium.

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REFERENCES

1. Knapp FF Jr, Kropp J, Goodman, MM, et al. The development of iodine-123-methyl-branched fatty acids and their applications in nuclear cardiology. *Ann Nucl Med* 1993;7:SII-1–SII-14.
2. Tamaki N, Kawamoto M, Yonekura Y, et al. Regional metabolic abnormality in relation to perfusion and wall motion in patients with myocardial infarction: assessment with emission tomography using an iodinated branched fatty acid analog. *J Nucl Med* 1992;33:659–667.
3. Kropp J, Jorgens M, Glaenzer KP, Luederitz B, Biersack HJ, Knapp FF Jr. Evaluation of ischemia and myocardial viability in patients with coronary artery disease (CAD) with iodine-123-labeled 15-(p-iodophenyl)-3-R,S-methylpentadecanoic acid (BMIPP). *Ann Nucl Med* 1993;7:SII-93–SII-100.
4. Matsunari I, Saga T, Taki J, et al. Kinetics of iodine-123-BMIPP in patients with prior myocardial infarction: assessment with dynamic rest and stress images compared with stress thallium-201 SPECT. *J Nucl Med* 1994;35:1279–1285.
5. Franken PR, DeGeeter FD, Dendale P, Demoor D, Block P, Bossuyt A. Abnormal free fatty acid uptake in subacute myocardial infarction after coronary thrombolysis: correlation with wall motion and inotropic reserve. *J Nucl Med* 1994;35:1758–1765.
6. Nakajima K, Shimizu K, Taki J, et al. Utility of iodine-123-BMIPP in the diagnosis and follow-up of vasospastic angina. *J Nucl Med* 1995;36:1934–1940.
7. Tamaki N, Tadamura E, Kawamoto M, et al. Decreased uptake of iodinated branched fatty acid analog indicates metabolic alterations in ischemic myocardium. *J Nucl Med* 1995;36:1974–1980.
8. Knapp FF Jr, Franken P, Kropp J. Cardiac SPECT with iodine-123-labeled fatty acids: evaluation of myocardial viability with BMIPP. *J Nucl Med* 1995;36:1022–1030.
9. Taki J, Nakajima K, Matsunari I, et al. Impairment of regional fatty acid uptake in relation to wall motion and thallium-201 uptake in ischaemic but viable myocardium: assessment with iodine-123-labelled beta-methyl-branched fatty acid. *Eur J Nucl Med* 1995;22:1385–1392.
10. Franken PR, Dendale P, De Geeter F, Demoor D, Bossuyt A, Block P. Prediction of functional outcome after myocardial infarction using BMIPP and sestamibi scintigraphy. *J Nucl Med* 1996;37:718–722.
11. Kurata C, Tawarahara K, Taguchi T, et al. Myocardial emission computed tomography with iodine-123-labeled beta-methyl-branched fatty acid in patients with hypertrophic cardiomyopathy. *J Nucl Med* 1992;33:6–13.
12. Takeishi Y, Chiba J, Abe S, Tonooka I, Komatani A, Tomoike H. Heterogeneous myocardial distribution of iodine-123 15-(p-iodophenyl)-3-R,S-methylpentadecanoic acid (BMIPP) in patients with hypertrophic cardiomyopathy. *Eur J Nucl Med* 1992;19:775–782.
13. Chen SL, Uehara T, Morozumi T, Yamagami H, Kusuoka H, Nishimura T. Myocardial metabolism of ^{123}I-BMIPP in patients with hypertrophic cardiomyopathy: assessment by radial long-axis SPET. *Nucl Med Commun* 1995;16:336–343.
14. Dudczak R, Schmoliner R, Angelberger P, Knapp FF, Goodman MM. Structurally modified fatty acid: clinical potential as tracers of metabolism. *Eur J Nucl Med* 1986;12:S45–S48.
15. Yamamichi Y, Kusuoka H, Morishita K, et al. Metabolism of iodine-123-BMIPP in perfused rat hearts. *J Nucl Med* 1995;36:1043–1050.
16. Fujibayashi Y, Nohara R, Hosokawa R, et al. Metabolism and kinetics of iodine-123-BMIPP in canine myocardium. *J Nucl Med* 1996;37:757–761.
17. Morishita S, Kusuoka H, Yamamichi Y, Suzuki N, Kurami M, Nishimura T. Kinetics of radioiodinated species in subcellular fractions from rat hearts following administration of iodine-123-labelled 15-(p-iodophenyl)-3-(R,S)-methylpentadecanoic acid (^{123}I-BMIPP). *Eur J Nucl Med* 1996;23:383–389.
18. Ichihara T, Ogawa K, Motomura N, Kubo A, Hashimoto S. Compton scatter compensation using the triple-energy window method for single- and dual-isotope SPECT. *J Nucl Med* 1993;34:2216–2221.
19. Braunwald E, Sobel BE. Coronary blood flow and myocardial ischemia. In: Braunwald E, ed. *Heart disease*, 4th ed. Philadelphia: W.B. Saunders; 1992;1161–1199.
20. Morishita K, Shirakami Y, Kusuoka et al. Uptake and washout kinetics of [I-123]-15-p-iodophenyl-3(R,S)-methyl pentadecanoic acid (BMIPP) in rat hearts [Abstract]. *J Nucl Med* 1996;37:P94–P95.
21. Hosokawa R, Nohara R, Okuda K, et al. Can the metabolism of I-123-BMIPP reflect ischemia? [Abstract]. *J Nucl Med* 1996;37:P95.

Left Ventricular Function and Perfusion in Becker's Muscular Dystrophy

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The aim of this study was to evaluate left ventricular (LV) perfusion and function in patients with Becker muscular dystrophy (BMD). **Methods:** Fourteen male patients (age range 14–40 yr) with BMD were evaluated by ^{201}Tl SPECT and radionuclide angiography both at rest and after dipyridamole stress test. **Results:** All patients showed uptake defect demonstrated by ^{201}Tl SPECT (mean 4.1 $\pm$ 2.2 uptake defect/patient). Significant relationships ($p < 0.05$) were found between the number of uptake defects and rest LV ejection fraction (LVEF) ($r = -0.54$), peak filling rate (PFR) ($r = -0.57$) and dipyridamole LVEF ($r = -0.65$). Dipyridamole induced reversible uptake defects were found in 7/14 (50%) patients with BMD. The 14 patients were divided into two groups on the basis of the presence (Group A, n = 6) or the absence (Group B, n = 8) of severe irreversible uptake defect (i.e., < 50% ^{201}Tl uptake). Group A showed lower values of PFR and LVEF when compared to patients of Group B. **Conclusion:** In patients with BMD there is a relatively high incidence of uptake defects and LV function (both at rest and after dipyridamole) appears to be related to the number of uptake defects. Moreover, the presence of severe irreversible uptake defects identifies a subgroup of patients with BMD characterized by a severely depressed LV function.

Key Words: thallium-201-SPECT; Becker muscular dystrophy; radionuclide angiography

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Becker muscular dystrophy (BMD) is an X-linked recessive muscular disease showing a slowly progressive skeletal muscle disorder with onset in late childhood characterized by weakness of proximal limb girdle muscles and calf muscle hypertrophy (*1–3*). Although muscular symptoms are predominant, heart involvement is a relatively common occurrence with a wide range of cardiac symptoms and manifestations (*4*). Subclinical cardiomyopathy (*5*) as well as dilated cardiomiopathy may be

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