# Improved Myocardial Fatty Acid Metabolism After Coronary Angioplasty in Chronic Coronary Artery Disease

Shintaro Yoshida, Makoto Ito, Kenichi Mitsunami and Masahiko Kinoshita
*First Department of Internal Medicine, Shiga University of Medical Science, Otsu, Japan*

This study assessed the utility of myocardial fatty acid imaging using $^{123}$I-labeled 15-(beta-methyl-p-iodophenyl-pentadecanic acid (BMIPP) to evaluate improvement after percutaneous transluminal coronary angioplasty in patients with chronic coronary artery disease. **Methods:** Thirty-eight patients (18 old myocardial infarction and 20 angina pectoris patients) with chronic coronary artery stenosis and 8 control subjects were enrolled in this study. All patients underwent successful angioplasty, and BMIPP SPECT was performed before and after angioplasty. SPECT images were divided into 13 segments and scored visually from 0 (normal uptake) to 4 (defect). The defect score was calculated as the summation of the total scores in each patient. The regional washout rate was calculated in both the reperfused areas and normal uptake areas using a bull's-eye map. **Results:** In nonrestenosis patients, BMIPP defect scores before and after angioplasty did not change on the initial image (9.6 ± 9.3 compared to 9.0 ± 9.2, nonsignificant p value), whereas they improved significantly on the delayed image (9.9 ± 8.8 compared to 8.2 ± 8.7, $p < 0.05$). In nonrestenosis patients, BMIPP washout rate in reperfused areas after angioplasty was significantly lower than that before angioplasty and the washout rate in control subjects (22.9% ± 8.4% compared to 31.5% ± 10.6% and 29.5% ± 8.0%, $p < 0.01$ and $p < 0.05$, respectively). In restenosis patients, BMIPP washout rate in both reperfused areas and normal uptake areas did not change after angioplasty. **Conclusion:** These data suggest that decreased BMIPP washout rate after angioplasty indicates improved fatty acid utilization in patients with chronic coronary artery disease.

**Key Words:** fatty acid metabolism; iodine-123-15-beta-methyl-p-iodophenyl-pentadecanic acid; chronic coronary artery disease; coronary angioplasty; restenosis

**J Nucl Med 1998; 39:933–938**

Although long-chain fatty acids are major substances for energy production in the normal myocardium, fatty acid oxidation is easily suppressed under ischemic conditions (*1*). Thus, radionuclide fatty acid imaging may be a useful method of assessing metabolic disorders in patients with coronary artery disease (CAD). PET using $^{11}$C-palmitate has been used to evaluate fatty acid metabolism in ischemic myocardium (*2–4*). However, PET studies are not widely performed because of the expense, and the use of $^{11}$C-palmitate is limited by the problem of extensive back diffusion in ischemic myocardium (*4*). For practical use, $^{123}$I-labeled fatty acid analogs have been developed to assess myocardial metabolism with a conventional gamma camera (*5–7*). Iodine-123-labeled 15-beta-methyl-p-iodophenyl-pentadecanic acid (BMIPP) is a branched fatty acid analog that has desirable characteristics for SPECT imaging, showing high accumulation and prolonged retention in the myocardium (*5,8,9*). According to a previous study by Tamaki et al. (*10*), decreased BMIPP uptake relative to $^{201}$Tl uptake was often observed in acute myocardial infarction. In addition, several recent studies have reported that discordance between perfusion tracer and BMIPP uptake in the acute stage is predictive for subsequent metabolic recovery and improvement of left ventricular function (*11–14*). However, in patients with chronic and stable CAD, metabolic recovery after reperfusion therapy, such as percutaneous transluminal coronary angioplasty (PTCA), has not yet been sufficiently investigated.

Although BMIPP is not rapidly metabolized by beta-oxidation (*9*), a considerable BMIPP washout rate from the myocardium has been noted (*7,8,15*). Moreover, an extensive multicenter trial of BMIPP in Japan demonstrated that some CAD patients showed significant fill-in and/or washout on delayed BMIPP images 3–4 hr after injection (*16*). These results suggest that the myocardial washout rate of BMIPP in ischemic myocardium is different from that in normal myocardium. Therefore, we hypothesized that the BMIPP washout rate would be altered when metabolic recovery occurs in ischemic myocardium. This study investigated whether the myocardial uptake and washout rate of BMIPP would be altered after PTCA in patients with chronic CAD.

## MATERIALS AND METHODS

### Patients

The study population consisted of chronic CAD patients meeting the following criteria:

1. Stable CAD without vasospastic angina pectoris or recent myocardial infarction (within 1 mo after onset);
2. No previous coronary intervention or bypass graft surgery;
3. Successful single- or two-vessel PTCA of the native coronary arteries; and
4. Absence of repeat PTCA during the follow-up period.

Thirty-eight patients with CAD (18 old myocardial infarction and 20 effort angina pectoris patients) who met all the criteria for the current analysis were enrolled in this study (28 men, 10 women; age range 39–78 yr; mean age = 65 yr). The control group consisted of 8 normal volunteers (6 men, 2 women; age range 48–65 yr; mean age = 61 yr). Each patient underwent control coronary angiography on admission, which showed coronary artery stenosis of >75% of the luminal diameter. Twenty-four patients had single-vessel disease, and 14 patients had two-vessel disease. In each patient, myocardial viability was assessed using dobutamine stress echocardiography and/or exercise $^{201}$Tl scintigraphy in selecting cases for coronary revascularization. Of the total 52 vessels, 45 vessels were dilated by PTCA (the left anterior descending artery in 18 patients, the left circumflex artery in 10 patients and the right coronary artery in 17 patients).

This study was approved by our institutional review board, and

Received Apr. 23, 1997; revision accepted Oct. 9, 1997.
For correspondence or reprints contact: Shintaro Yoshida, MD, First Department of Internal Medicine, Shiga University of Medical Science, Tsukinowa-cho, Seta, Otsu, Shiga, 520-21, Japan.

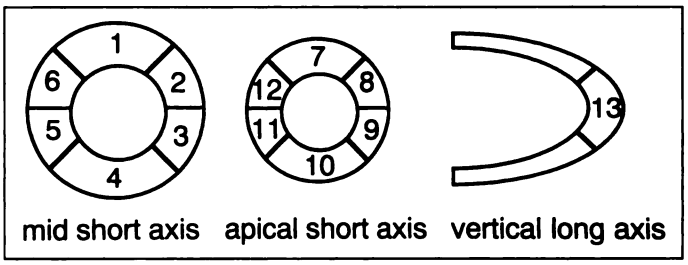


**FIGURE 1.** Schematic presentation of myocardial segments using the short-axis and vertical long-axis. The left ventricle was divided into 13 segments.

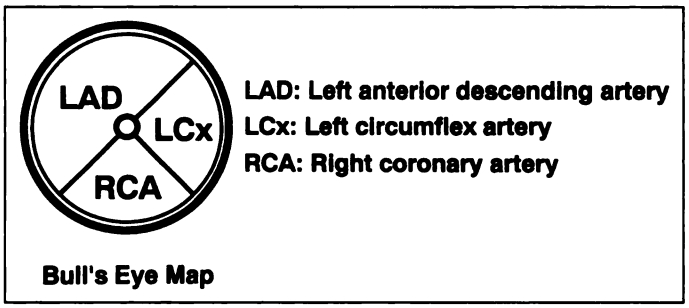


**FIGURE 2.** Schematic anatomic display of the distribution of the three major coronary arteries in the bull's-eye polar map.

informed consent was obtained from all subjects before they underwent any of the procedures.

### Coronary Intervention

Within 2 wk after control coronary angiography, PTCA was performed in all patients, each of whom showed stenosis of <50% of the luminal diameter in the target vessels. Three months after PTCA, all patients underwent follow-up coronary angiography, and findings were judged by experienced angiographers who determined whether restenosis was present. Angiographic restenosis was defined as >50% narrowing at the previous PTCA sites on follow-up coronary angiography (*17,18*).

### BMIPP Imaging Protocol

Myocardial SPECT imaging with BMIPP at rest was performed within 1 wk before PTCA in each patient and in each control. BMIPP was prepared and provided by Nihon Medi-Physics Co., Ltd. (Hyogo, Japan). The vial of BMIPP contained 74 MBq/ml (0.4 mg/ml) $^{123}$I-labeled BMIPP dissolved in ursodeoxycholic acid (7 mg/ml). Under fasting and resting conditions, 111 MBq (1.5 ml) $^{123}$I-BMIPP was injected intravenously and immediately flushed by 10 ml of saline. SPECT images were obtained 20 min (initial images) and 180 min (delayed images) after the injection of BMIPP, after the protocol of the multicenter trial of BMIPP in Japan (*16*), using a rotating gamma camera (model GCA-901A; Toshiba Co., Ltd., Tokyo, Japan) equipped with a low-energy, general-purpose collimator. The energy window was set at 159 keV ±10%. In each SPECT acquisition, 32 projection images were obtained, scanning from a 45° right anterior oblique to a 45° left posterior oblique and taking 40 sec per view. The raw data were stored in a 64 × 64 matrix on the hard disk (5.3 mm/pixel), and tomographic images (6-mm-thick slices) were reconstructed along the short, horizontal and vertical long axes of the left ventricle using the manufacturer's data processor (model GMS-550U; Toshiba, Co., Ltd., Tokyo, Japan). Three months after PTCA, follow-up BMIPP SPECT studies were performed using the same protocol as that before PTCA in all patients.

### Image Analysis

For the qualitative analysis, two short-axis (mid and apical position) slices and a long-axis slice were selected. Left ventricular myocardia were divided into 13 segments (Fig. 1). BMIPP uptake in each segment was scored visually on a scale of 0–4 (0 = normal; 1 = mild reduction; 2 = moderate reduction; 3 = severe reduction; 4 = defect) by three independent investigators (*18*). The defect score was calculated as the summation of the total regional scores in each patient. When the scores in each segment were decreased from the initial to the delayed image, the segment was regarded as showing fill-in. When the scores were increased, the segment was regarded as showing washout.

For quantitative analysis, circumferential profiles were generated to create a polar map using the short axis from apical to basal slices. The maximal count density was measured in each 9° wedge extending 360° around each short axis. Subsequently, these profiles were represented with rectangular coordinates and finally arranged as concentric circles with increasing radii (bull's-eye polar map). For the assessment of the BMIPP washout rate, the bull's-eye images were divided into three regions assigned to the distribution of the three major coronary arteries (Fig. 2). The areas reperfused by PTCA were considered regions corresponding to the angioplastic vessels, and the normal BMIPP uptake areas were determined as regions that included a maximal count density without angioplastic regions. The washout rate in the reperfused areas and in the normal uptake areas were calculated as follows:

$$\text{Washout rate (\%)} = \frac{(\text{Initial counts} - \text{Delayed counts})}{\text{Initial counts}} \times 100.$$

### Statistical Analysis

All values are presented as mean ± s.d. The differences in population were examined by chi-square test. Student's t-test was used for comparison of paired data. Scheffe's F-test for multiple comparisons was used to detect significant differences, as defined by analysis of variance. A p value of <0.05 was considered significant.

## RESULTS

### Follow-Up After Percutaneous Transluminal Coronary Angioplasty

Thirty-three of 45 vessels (74%) did not show restenosis (nonrestenosis group) in 29 of 38 patients. Twelve vessels (26%) showed restenosis (restenosis group) in 9 patients. None of the patients in the nonrestenosis group experienced recurrent angina or other cardiac events during the follow-up period. In the restenosis group, 7 patients showed recurrent chest pain, whereas 2 patients remained asymptomatic. There were no deaths among either group during the follow-up period.

### BMIPP Findings Before and After Percutaneous Transluminal Coronary Angioplasty

High-quality SPECT images were obtained in the BMIPP studies from all subjects. All control subjects showed homogeneous myocardial accumulation of BMIPP on the initial and delayed images. Before PTCA, all 38 patients showed reduced myocardial accumulation of BMIPP on the initial SPECT images in at least one myocardial segment. Of the 494 total myocardial segments, 332 (67%) were classified as segments with normal BMIPP uptake, and the remaining 187 (38%) were defined as segments with reduced BMIPP uptake. These 187 segments with reduced BMIPP uptake were evaluated visually before and after PTCA.

Eighty-eight (47%) of the total 187 segments with reduced BMIPP uptake showed fill-in (38 segments) or washout (50 segments) on the delayed SPECT images. In the nonrestenosis group, 27 segments showed fill-in, and 37 segments showed washout before PTCA. In the restenosis group, 11 segments showed fill-in, and 13 segments showed washout before PTCA.

**TABLE 1**
Comparison of Altered SPECT Segments on Delayed Images by Percutaneous Transluminal Coronary Angioplasty (PTCA)

| Group | Before PTCA* | After PTCA* |
|---|---|---|
| Restenosis (−) | | |
| Fill-in | 27 | 57† |
| Washout | 37 | 9† |
| Unchanged | 313 | 311 |
| Restenosis (+) | | |
| Fill-in | 11 | 12 |
| Washout | 13 | 14 |
| Unchanged | 93 | 91 |

*Before PTCA, within 1 wk before PTCA; after PTCA, 3 mo after PTCA.
†$p < 0.01$ compared to before PTCA.

Three months after PTCA, the number of segments with fill-in was significantly increased (from 27 to 57 segments, $p < 0.01$), and the number of segments with washout was significantly decreased (from 37 to 9 segments, $p < 0.01$) in the nonrestenosis group. In contrast, the number of segments with fill-in or washout did not change significantly after PTCA in the restenosis group (fill-in, from 11 to 12 segments; washout, from 13 to 14 segments) (Table 1).

### Defect Scores Before and After Percutaneous Transluminal Coronary Angioplasty

Changes in defect scores of BMIPP before and after PTCA in the nonrestenosis group are shown in Figure 3. Defect scores did not improve significantly 3 mo after PTCA on initial images (9.6 ± 9.3 compared to 9.0 ± 9.2, nonsignficant p value). On delayed images, however, defect scores significantly improved 3 mo after PTCA (9.9 ± 8.8 compared to 8.2 ± 8.7, $p < 0.05$). In the restenosis group, defect scores after PTCA were unchanged on both the initial images (10.2 ± 9.1 compared to 9.9 ± 9.2, nonsignficant p value) and delayed images (10.5 ± 9.9 compared to 10.4 ± 8.9, nonsignificant p value).

### Changes in BMIPP Washout Rate

Figure 4 shows the regional washout rate of BMIPP in the reperfused areas and in the normal uptake areas in the nonrestenosis group. In the 33 regions with reperfused areas, the washout rate after PTCA was significantly less than that before PTCA (22.9% ± 8.4% compared to 31.5% ± 10.6%, $p < 0.01$) and was less than that in the control group (22.9% ± 8.4% compared to 29.5% ± 8.0%, $p < 0.05$). BMIPP washout rate in the reperfused areas was also significantly less than that in the normal uptake areas before and after PTCA (22.9% ± 8.4% compared to 27.9% ± 7.4% and 28.4% ± 6.5%, respectively, $p < 0.05$). However, BMIPP washout rate in the normal uptake areas was similar before and after PTCA (27.9% ± 7.4% compared to 28.4% ± 6.5%, nonsignificant p value).

In the restenosis group, BMIPP washout rate in the reperfused areas did not change before and after PTCA (30.3% ± 9.8% compared to 31.2% ± 11.3%, nonsignificant p value), and BMIPP washout rate in the normal uptake areas also remained unchanged before and after PTCA (28.4% ± 8.3% compared to 29.9% ± 9.4%, nonsignificant p value) in 12 regions. In addition, there were no significant differences among the BMIPP washout rates in the restenosis or normal uptake areas and in the control group (Fig. 5).

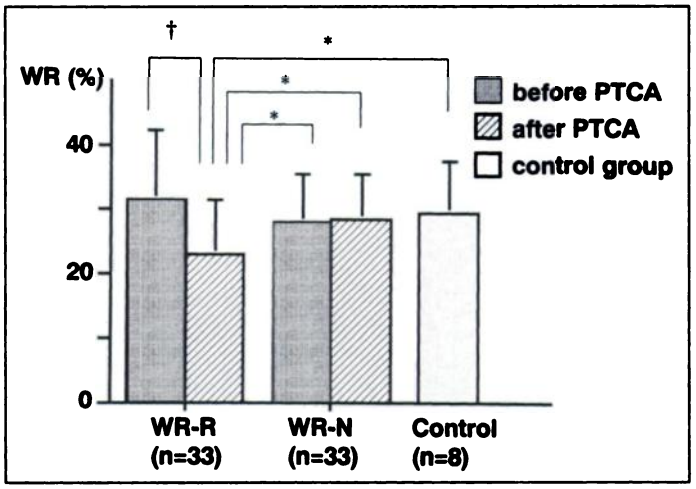


**FIGURE 4.** Changes in BMIPP washout rate the in nonrestenosis group. Before PTCA, within 1 wk before PTCA; after PTCA, 3 mo after PTCA. WR = BMIPP washout rate; WR-R = BMIPP washout rate in reperfused areas; WR-N = BMIPP washout rate in normal uptake areas. *$p < 0.05$; †$p < 0.01$.

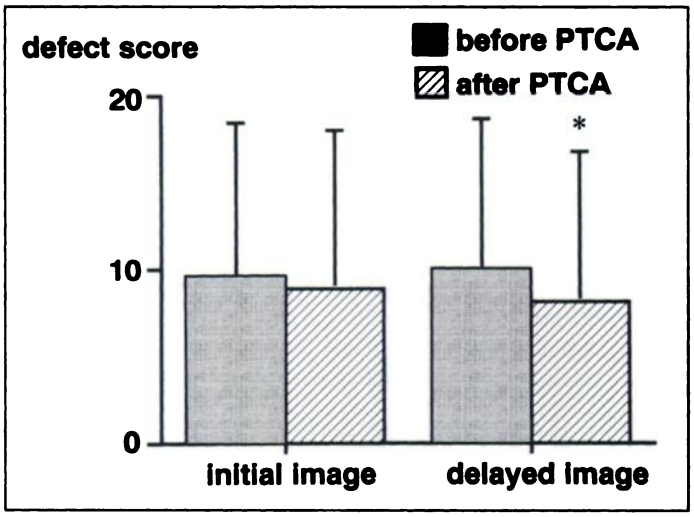


**FIGURE 3.** Comparison of BMIPP defect scores before and after PTCA in the nonrestenosis group. Before PTCA, within 1 wk before PTCA; after PTCA, 3 mo after PTCA. *$p < 0.05$ compared to before PTCA.

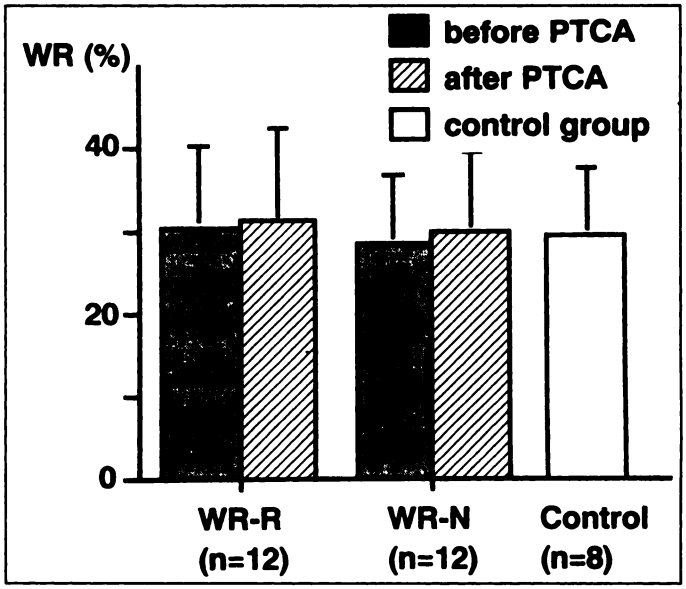


**FIGURE 5.** Changes in BMIPP washout rate in the restenosis group. Before PTCA, within 1 wk before PTCA; after PTCA, 3 mo after PTCA. WR = BMIPP washout rate; WR-R = BMIPP washout rate in reperfused areas; WR-N = BMIPP washout rate in normal uptake areas.

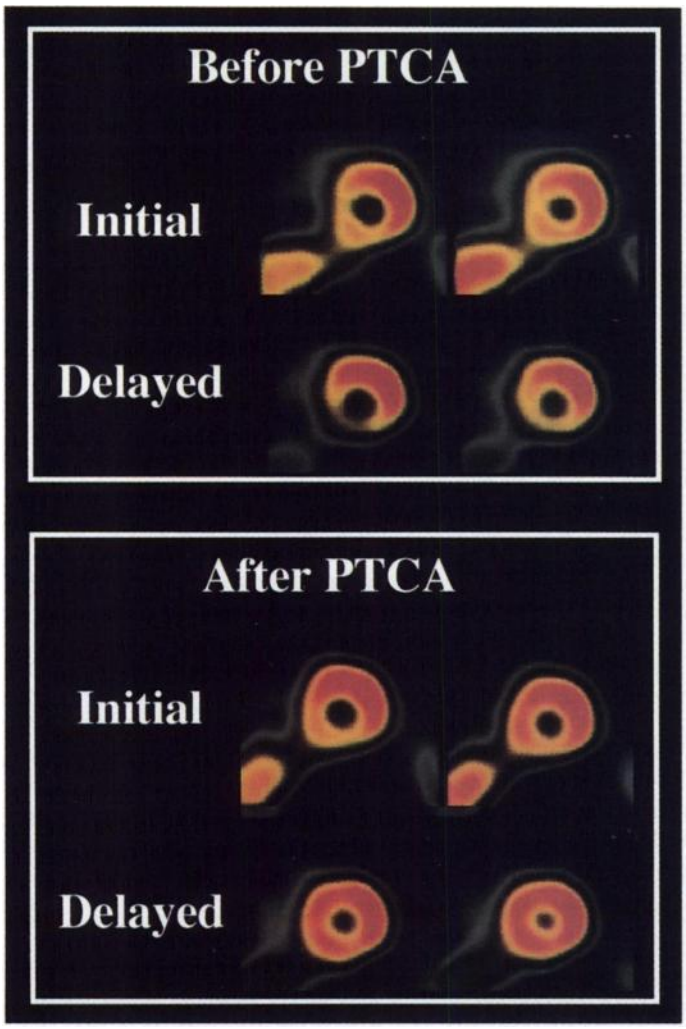


**FIGURE 6.** Representative short-axis images of BMIPP in a 48-yr-old woman with effort angina pectoris. The inferior segments showed washout before PTCA, and these segments showed fill-in after PTCA.

### Case Presentation

Figure 6 shows myocardial SPECT images with BMIPP in a 48-yr-old woman with effort angina pectoris. The patient had 90% stenosis in the right coronary artery. On BMIPP images before PTCA, washout was observed in the inferior and posterior segments. However, these segments showed fill-in 3 mo after successful PTCA. BMIPP washout rate in the right coronary artery area was 35.7% before PTCA and 19.8% after PTCA.

## DISCUSSION

### Major Findings

The results of our study demonstrated the following:

1. Myocardial accumulation of BMIPP showed different distributions on initial and delayed images in several patients with chronic CAD.
2. In the nonrestenosis group, segments with fill-in were increased, and segments with washout were decreased 3 mo after successful PTCA. The defect score after PTCA was improved significantly compared to that before PTCA.
3. In the restenosis group, segments with fill-in or washout were unchanged, and the defect score was also unchanged before and after PTCA.
4. BMIPP washout rate in the reperfused areas was reduced significantly after PTCA compared to that before PTCA in the nonrestenosis group.
5. BMIPP washout rate in the reperfused areas was unchanged after PTCA in the restenosis group.

### BMIPP Uptake and Retention

Because BMIPP is a beta-methyl branched fatty acid analog, it is not easily metabolized by beta-oxidation (*9*). BMIPP is trapped in the myocardium by regional myocardial blood flow and is incorporated into the endogenous lipid pool of triglycerides (*9,19,20*). Therefore, myocardial uptake of BMIPP may not directly reflect fatty acid metabolism but may reveal the size of the triglyceride pool. However, BMIPP requires cytosolic adenosine-5′-triphosphate (ATP) for retention in myocytes as acyl-coenzyme A (CoA). Fujibayashi et al. (*21*) reported that BMIPP accumulation was closely correlated with intracellular ATP concentration in rat hearts. Because cytosolic ATP is necessary to convert fatty acids to acyl-CoA, which is a common pathway of fatty acid metabolism (*22,23*), both the diminished size of triglyceride pool and decreased cytosolic ATP concentration may play important roles in the accumulation of BMIPP in the ischemic myocardium. Therefore, although myocardial BMIPP uptake may not directly indicate fatty acid metabolism, it seems to be an acceptable method for assessing myocardial fatty acid utilization. The segments with increased BMIPP uptake after PTCA may be associated with the recovery of fatty acid metabolism in patients with CAD.

There are only a few articles that have shown serial changes in BMIPP uptake before and after coronary reperfusion therapy in chronic CAD (*24,25*). Matsunari et al. (*24*) reported an effort angina patient who showed improved BMIPP uptake 4 mo after PTCA on the initial image. Takeishi et al. (*25*) demonstrated delayed (3 mo after PTCA) recovery of regional BMIPP uptake early after tracer administration in patients with angina pectoris. In this study, the improvement in BMIPP defect scores 3 mo after PTCA was not significant on the initial images but was significant on the delayed images in patients without restenosis (Fig. 3). However, our patient group consisted of 20 angina pectoris and 18 old myocardial infarction patients. It is possible that the improvement in the initial BMIPP uptake in patients with myocardial infarction is slower than that in patients with angina pectoris. A long-term follow-up study is required for assessing improvements in BMIPP uptake in patients with CAD after PTCA.

### BMIPP Washout Before and After Percutaneous Transluminal Coronary Angioplasty

In patients with CAD, previous studies did not demonstrate serial changes in BMIPP washout rate because BMIPP imaging has been used commonly for the early uptake after tracer administration (*24,25*). Although BMIPP is a structurally modified fatty acid showing prolonged myocardial retention, considerable washout from the myocardium has been noted (*7,8,15*). Dudczak et al. (*26*) and Knapp et al. (*7*) proposed possible pathways, such as alpha-oxidation, for the metabolism of BMIPP. In addition, according to a recent experimental study by Yamamichi et al. (*20*), BMIPP is metabolized by initial alpha-oxidation and subsequent beta-oxidation in rat myocardium. However, the retention mechanism of BMIPP has not been sufficiently clarified because of its complex metabolism in myocytes.

In this study, BMIPP uptake often showed different distributions on the initial and delayed images in patients with chronic CAD. Because BMIPP rapidly disappears from the blood (*19,20*), myocardial reuptake of BMIPP does not occur at the time of

delayed image acquisition. Therefore, observation of fill-in is not a redistribution phenomenon but a relative change in regional distribution. In the nonrestenosis group, the defect score significantly improved on delayed images because of increased fill-in and decreased washout segments after PTCA (Fig. 3). Although delayed BMIPP uptake has not been used widely in patients with CAD, BMIPP delayed images are potentially useful as a method to assess the changes in fatty acid metabolism in patients with CAD.

There are several controversial reports regarding BMIPP clearance from the myocardium (*27,28*). Kropp et al. (*28*) reported only small differences in the BMIPP turnover rate between normal and ischemic areas. Our data also indicated that the washout rate in injured areas was not significantly different from that in normal areas before PTCA. Nishimura et al. (*19*) demonstrated that the mean half-time value, which was generated from the BMIPP washout curve, was significantly larger in the reperfused canine myocardium. This experimental report is concordant with our clinical results using SPECT acquisition and the bull's-eye method in CAD patients. In the nonrestenosis group, BMIPP washout rate in the reduced uptake areas was significantly decreased 3 mo after PTCA compared to those before PTCA and those in the restenosis group. These data suggest that the decreased washout rate after PTCA might indicate metabolic recovery after improvement of coronary perfusion after PTCA.

Because BMIPP is not easily metabolized by beta-oxidation and is stored mainly in the triglyceride pool (*9,19,20*), BMIPP washout rate may not directly reflect fatty acid oxidation. Schoonderwoerd et al. (*29*) reported that the lipolysis of triglyceride pool is enhanced during low-flow ischemia in the isolated rat hearts. In this study, before PTCA, BMIPP washout rate in the reduced uptake areas was not significantly increased but was slightly increased compared to that in the normal uptake areas (Fig. 4).

After PTCA, BMIPP washout rate in the reperfused areas was significantly reduced compared to that in normal uptake areas and the control group. These results suggest that triglyceride turnover in reperfused myocardium was slower than that in normal myocardium. Slower BMIPP washout rate, at least in part, reflects improvement of fatty acid utilization and preservation of triglyceride lipolysis. Further investigation is needed to clarify the intracellular metabolism and retention mechanism of BMIPP before and after coronary reperfusion.

### Potential Study Limitations

In this study, we used BMIPP imaging alone to assess myocardial fatty acid metabolism without using myocardial perfusion agents, such as $^{201}$Tl or other $^{99m}$Tc perfusion agents. Previous studies demonstrated the ability of combined study with fatty acid analogs and perfusion agents to assess myocardial fatty acid metabolism (*10,11,30–33*). However, precise perfusion imaging at rest was not obtained in some subjects; hence, we did not refer to myocardial perfusion abnormality as an index of injured myocardium.

This study included 18 patients with old myocardial infarction, and we evaluated the myocardial BMIPP washout rate in areas with ischemia as well as infarction. Thus, the areas reperfused by PTCA included both viable and nonviable myocardium. In our institution, however, myocardial viability has been assessed using dobutamine stress echocardiography and/or exercise $^{201}$Tl scintigraphy in selecting patients for coronary revascularization. Thus, most of the reperfused areas in this study may have included more viable myocardium than necrotic tissue.

### Clinical Implications

Exercise perfusion imaging using $^{201}$Tl or other $^{99m}$Tc agents may be a useful method of detecting coronary stenosis and postangioplastic restenosis. However, a noninvasive approach at rest is more desirable than other exercise or pharmacological stress tests. In this study, we demonstrated that the decreased BMIPP washout rate at rest may indicate improved fatty acid utilization after coronary revascularization. Therefore, evaluation of the BMIPP washout rate may be a potentially useful modality for identifying postangioplastic restenosis without any stress examinations and might be useful for assessing metabolic recovery of revascularized areas.

## CONCLUSION

In patients with chronic CAD, altered regional BMIPP uptake was often observed between the initial and delayed images. On delayed images, successful PTCA without restenosis improved regional BMIPP uptake 3 mo after PTCA. A decreased BMIPP washout rate after PTCA may reflect recovery of fatty acid utilization after improvement in coronary perfusion.


## ACKNOWLEDGMENTS

We thank Drs. Tohru Inoue, Shinro Matsuo and Tuhin Haque for the SPECT studies. We also thank Nihon Medi-Physics for its support for this study.


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# First-Pass Radionuclide Angiography Using Iodine-123 Myocardial Tracers and a Multicrystal Gamma Camera

Eiji Tadamura, Haruhiro Kitano, Takashi Kudoh, Naoya Hattori, Masayuki Inubushi, Kazunobu Nishimura, Izuru Masuda, Hideo Inada, Ryohei Hosokawa, Ryuji Nohara and Junji Konishi

*Departments of Nuclear Medicine and Cardiovascular Surgery, Second and Third Divisions, and Department of Internal Medicine, Kyoto University Faculty of Medicine, Kyoto, Japan*

The purpose of this study was to validate the accuracy of the assessment of ventricular function by first-pass radionuclide angiography (FPRNA) with $^{123}$I myocardial tracers and a multicrystal gamma camera. **Methods:** Left ventricular ejection fraction (LVEF) and right ventricular ejection fraction were measured in 69 patients by FPRNA using $^{123}$I myocardial tracers (126 ± 7 MBq) and $^{99m}$Tc tracers (541 ± 141 MBq) on a multicrystal gamma camera with a high-sensitivity collimator. For 44 patients, ejection fraction values measured by $^{123}$I-FPRNA were compared to those estimated by equilibrium radionuclide angiography (ERNA). Visual wall-motion analysis was also performed to judge clinical acceptability of $^{123}$I-FPRNA images for identification of wall-motion abnormality. **Results:** Mean LVEFs (%) estimated by $^{123}$I-FPRNA and by $^{99m}$Tc-FPRNA were 49.6 ± 13.6 and 49.1 ± 14.1, respectively (nonsignificant p value). An excellent correlation was found between LVEFs estimated by $^{123}$I-FPRNA and $^{99m}$Tc-FPRNA ($r = 0.96$, s.e.e. = 1.9%). Values of LVEF measured by $^{123}$I-FPRNA also demonstrated excellent correlation with those measured by ERNA ($r = 0.95$, s.e.e. = 2.2%). A good correlation was also noted between right ventricular ejection fractions measured by $^{123}$I-FPRNA and $^{99m}$Tc-FPRNA ($r = 0.72$, s.e.e. = 4.0%). The Spearman rank correlation coefficient between $^{123}$I-FPRNA and ERNA wall-motion scores was 0.87 ($n = 135$, $p < 0.001$). **Conclusion:** Resting ventricular function can be reliably measured with $^{123}$I-FPRNA in combination with a multicrystal gamma camera. This indicates that the assessment of ventricular function is feasible in conjunction with $^{123}$I myocardial imaging without an increase in cost or radiation dose to patients.

**Key Words:** iodine-123 myocardial tracer; first-pass radionuclide angiography; ejection fraction

**J Nucl Med 1998; 39:938–944**

Noninvasive assessment of left ventricular (LV) and right ventricular (RV) performance is one of the main goals of nuclear cardiology. Continuous efforts have been made toward this objective, and the several approaches that have been reported include planar equilibrium radionuclide angiography (ERNA) (*1–3*), first-pass radionuclide angiography (FPRNA) (*4–6*), gated blood-pool SPECT (*7–9*) and gated perfusion SPECT (*10–12*). Although there were attempts to use $^{195m}$Au (*13*) and $^{191m}$Ir (*14*) in FPRNA, $^{99m}$Tc agents are now exclusively used in all of these approaches to evaluate ventricular function.

The clinical and investigational usefulness of $^{123}$I-labeled radiotracers such as 15-(p-iodophenyl)-3-(R,S)-methylpentadecanoic acid (BMIPP) (*15,16*) and metaiodobenzylguanidine (MIBG) (*17,18*) has now been widely recognized. Mainly because of the longer physical half-life of $^{123}$I (13.3 hr) compared to $^{99m}$Tc, $^{123}$I tracers are injected at limited dose (111–148 MBq), which is disadvantageous for FPRNA. However, we hypothesized that FPRNA using $^{123}$I tracers may be feasible if the images are acquired with a multicrystal camera that has higher counting capabilities than the usual single-

Received Jun. 30, 1997; revision accepted Sep. 27, 1997.

For correspondence or reprints contact: Eiji Tadamura, MD, Department of Nuclear Medicine and Diagnostic Imaging, Kyoto University Faculty of Medicine, Shogoin, Sakyo-ku, Kyoto, 606-01, Japan.