Primary percutaneous coronary intervention (pPCI) with stent implantation is an established reperfusion strategy for patients with ST-segment–elevation myocardial infarction (STEMI). However, despite achieving a sufficient flow of epicardial coronary artery after pPCI in the majority of patients, up to one third of patients do not achieve adequate tissue perfusion. It is known that the inadequate tissue perfusion occurs as a result of mechanical plugging secondary to distal embolization from the epicardial coronary arteries, external compression by edematous tissue, in situ thrombosis, vasospasm, and activation of inflammatory cascades with leukocyte stasis and extravasation. The inadequate tissue perfusion can be assessed directly in the cardiac catheterization laboratory during pPCI by different ways, ranging from simple angiographic thrombolysis in myocardial infarction (TIMI) grade score to more invasive measures of coronary blood flow (CBF).

Recently, it is possible to measure coronary pressure and estimate CBF simultaneously with a single pressure sensor/thermistor-tipped guidewire in the cardiac catheterization laboratory during pPCI. Using the thermodilution technique with this guidewire, the mean transit time of room temperature saline injected down a coronary artery can be determined and has been shown to correlate inversely with absolute flow. A previous study revealed that CBF can be measured by analyzing thermodilution curve in human coronary artery. Furthermore, an index of microcirculatory resistance (IMR)
WHAT IS KNOWN

- Primary percutaneous coronary intervention is an established reperfusion strategy for patients with ST-segment–elevation myocardial infarction.
- Microvascular obstruction is associated with poor clinical outcomes among patients treated by primary percutaneous coronary intervention.
- The index of microcirculatory resistance, which reflects microvascular coronary function, can be measured using a special pressure sensor/thermistor-tipped guidewire, at the time of cardiac catheterization.

WHAT THE STUDY ADDS

- Coronary blood flow patterns were classified into 3 groups according to the shape of the thermodilution curve: (1) a narrow unimodal pattern, (2) a wide unimodal pattern, and (3) a bimodal pattern.
- The presence of a bimodal shape of the thermodilution curve was associated with microcirculatory damage and poor midterm clinical outcomes rather than the index of microcirculatory resistance.

was recently developed for assessing the status of the microcirculation independent of the epicardial artery stenosis. It has been reported that these thermodilution-derived parameters were associated with microvascular damage after myocardial infarction and predictors of left ventricular functional recovery after 3 months. However, the relationship between thermodilution-derived parameters and clinical outcome has not been evaluated in detail. The purpose of this study is to evaluate whether the thermodilution-derived CBF parameters and IMR values immediately after pPCI predict early microvascular damage and midterm outcomes after STEMI.

Methods

Study Design and Population

This study was prospectively designed. Between November 2009 and March 2012, a total of 150 patients with STEMI were treated by pPCI with stent implantation within 12 hours from onset of symptoms. Six patients with a history of myocardial infarction in the index territory, 4 patients with left main stem culprit lesions, 13 patients with Killip class III to IV, 3 patients with a history of coronary artery bypass grafts, and 4 patients with a history of cardiomyopathy, 6 patients with significant arrhythmia rendering an invasive coronary physiological study inappropriate, and 1 patient with contraindications to adenosine and standard contraindications to contrast-enhanced cardiac MRI (ecCMR) were not enrolled to avoid bias. Ten patients who required the percutaneous cardiopulmonary support during PCI were also excluded because CBF is not physiological in this condition. Five patients in whom the sensor was positioned at the segment proximal to the stent for the assessment of CBF and IMR were also excluded. Finally, a total of 88 patients were considered to be eligible for performing postintervention physiological assessments. STEMI was defined as continuous chest pain of >30 minutes, ST-segment elevation of ≥0.1 mV in ≥2 continuous electrocardiographic leads, and an increase of serum creatine kinase (CK-MB) ≥5× the normal value. Identification of culprit lesions involved the combination of left ventricular wall motion abnormalities, ECG findings, and angiographic lesion morphology. The ethics committee at Hyogo College of Medicine approved the protocols, and written informed consent was obtained before all procedures from all patients.

Study Protocol and Angiographic Analysis

All patients received intravenous heparin administration (100 U/kg) before the procedure. Primary PCI was performed according to standard clinical practices with stent implantation. Procedural success was defined as ≤25% residual stenosis of the culprit lesion by visual assessment. Blood samples were obtained on admission and serially every 4 hours for the first 24 hours after primary PCI, and peak values of creatine phosphokinase (CPK) and CK-MB were determined. All patients received loading of dual oral antiplatelet agents with 200 mg of aspirin along with 300 mg of clopidogrel as early as possible before pPCI. During the study period, platelet glycoprotein IIb/IIIa receptor inhibitor was not used in all patients because this medicine had not been approved for clinical use in Japan. After pPCI, all patients were prescribed life-long aspirin (100 mg/d) and clopidogrel (75 mg/d) for ≥1 month.

Angiographic coronary flow was assessed based on the TIMI criteria. The corrected TIMI frame count was defined as the number of frames necessary for the dye to reach standardized distal landmarks, as previously described. Quantitative coronary angiography was analyzed using a computer-assisted, automated edge-detection algorithm (CMS, MEDIS) using standard measurements by an independent observer who was blinded to clinical and physiological information.

Coronary Physiological Measurement and Analysis

Fifteen minutes after successful pPCI, an intracoronary pressure/temperature sensor-tipped wire (Radi pressure wire Certus; St. Jude Medical, St Paul, MN) was calibrated outside the body, equalized to the pressure reading from the guide catheter with the pressure sensor positioned at the ostium of the guide catheter, and then advanced through a 6F guiding catheter to a point 7 cm distal from the ostium of each coronary artery, which in the vast majority was beyond the stented region. To position the pressure/temperature sensor to the 7 cm distal from the ostium of each coronary artery accurately, the distance from the Y-connector with the wire to fastening the torque device was 7 cm and advanced through the target artery. With commercially available software (Radi Analyzer; St. Jude Medical), the shaft of this guidewire can act as a proximal thermistor by detecting changes in temperature-dependent electric resistance. The sensor, located 3 cm proximal to the guidewire tip, simultaneously measures pressure and temperature and can thereby act as a distal thermistor. Maximal hyperemia was induced by adenosine triphosphate (150 μg/kg per minute) administered intravenously. All physiological assessments were performed after intracoronary administration of 2 mg isosorbide dinitrate. At steady-state hyperemia, the mean aortic and distal coronary pressures were measured, and 3 consecutive thermodilution curves were obtained by brisk injection of 3 mL of room temperature saline by hand into the coronary artery through the guiding catheter.

The IMR was calculated by multiplying the mean distal coronary pressure by the hyperemic mean transit times, which are inversely proportional to flow, as reported previously. CBF patterns were classified into 3 groups according to the shape of the thermodilution curve (Figure 1): (1) a narrow unimodal pattern (a rapid fall and rise of temperature–time curves), (2) a wide unimodal pattern (a gradual fall and rise of temperature–time curves), and (3) a bimodal pattern (2 populations with valley deeper than 20% of peak temperature drop). The mean transit time from the beginning of drop to the maximum drop in temperature on thermodilution curve was 0.42±0.14 in patients who were classified into narrow and wide unimodal groups. A narrow unimodal profile was characterized by a sharp peak in the CBF profile, and transit time from the beginning of drop to the maximum drop in temperature on thermodilution curve was 0.42 s. A wide unimodal profile was considered whether the top of the thermodilution curve was dull and transit time from the beginning of drop to the maximum drop in temperature on thermodilution curve was ≥0.42 s.

Intraobserver and interobserver variability were assessed by the evaluation of all thermodilution curves by 2 independent readers and by the same reader at 2 separate time points, respectively.
Definition of microvascular obstruction (MVO) on contrast-enhanced cardiac MRI (ceCMR). We defined MVO as an area of hypoenhancement within the gadolinium hyperenhanced area of infarcted tissue present early after contrast injection and persistent when reimaged 15 minutes after contrast injection.

Protocol and Imaging Analysis for ceCMR
One patient died before receiving a ceCMR scan. Patients with contraindications for ceCMR (eg, pacemaker or defibrillator), atrial fibrillation, dyspnea and an inability to hold breath for 10 to 15 seconds, or end-stage renal disease also were excluded from ceCMR scan studies. Therefore, ceCMR was performed and was evaluable in 68 patients (77%) at 7.4±3.2 days after pPCI. Scanning was performed using Intera Maste 1.5-T imaging unit (Philips Medical Systems, Best, The Netherlands) scanner by a 6-channel anterior chest coil and spinal coils within the gantry table. Early and delayed enhancement images covering the whole ventricle were acquired immediately after injection of 0.2 mmol/kg body weight of gadopentetate dimeglumine (Magnevist; Bayer Pharma). Microvascular obstruction (MVO) was defined as an area of hypo-enhancement within the gadolinium hyperenhanced area of infarcted tissue present early after contrast injection and persistent when reimaged 15 minutes after contrast injection.

Study End Points
The primary end point of this study was a composite of cardiac death, nonfatal myocardial reinfarction, and heart failure rehospitalization within 6 months (major adverse cardiovascular events [MACEs]). Six-month clinical follow-up was performed by either telephone contact or office visit using a standard questionnaire. In case of any event, these were verified by hospital charts or direct contact with the referring physician.

Statistical Analysis
Continuous variables were reported as mean±1 SD. One-way ANOVA was used to compare continuous variables. The nonparametric Kruskal–Wallis test (for 3-way comparisons) was used to compare the IMR values among 3 groups because the data were not normally distributed and to compare each group by Mann–Whitney U test with Bonferroni correction; the Bonferroni correction was used for post hoc analysis in which P<0.0167 (0.05 divided by 3) was the threshold for significance. Categorical variables were reported as frequencies and compared using χ² statistics or Fisher exact test. Survival curves were estimated using the Kaplan–Meier method and compared using the univariate log-rank test. Cox proportional hazard regression analysis was performed to determine independent predictors of MACE in the 6-month period after pPCI for patients with STEMI. All individual variables with P value <0.1 were considered for inclusion into multivariable forward stepwise models to determine the independent predictors. Variables tested included multivessel disease, peak CPK level, % MVO area, ejection fraction, IMR value, and CBF pattern. Intraobserver and interobserver variability were measured by the κ-test of concordance for classifying the CBF patterns into the unimodal or bimodal patterns. A P value of <0.05 was considered statistically significant. All statistical analyses were performed with EZR (Saitama Medical Center, Jichi Medical University), which is a graphical user interface for R (The R Foundation for Statistical Computing, Vienna; version 2.13.0). More precisely, it is a modified version of R commander (version 1.8-4) designed to add statistical functions frequently used in biostatistics.

Results
The study population consisted of 73 men and 15 women with an average age of 67±13 years. Mean time from the onset of symptoms to primary PCI was 345±193 minutes. Mean peak CPK and CK-MB values were 2604±1689 and 278±177 U/L. The shape of the thermodilution curve revealed a narrow unimodal in 41 (47%) of 88 patients and a wide unimodal in 32 (36%) patients. The remaining 15 (17%) patients were classified as having a bimodal shape (intraobserver variability and interobserver variability, κ=0.93 and κ=0.89, respectively). Baseline patient characteristics and angiographic findings are presented in Table 1. There were no linear relationships between IMR values and peak CPK and CK-MB levels. Peak CPK levels and peak CK-MB levels tended to be higher in the bimodal group than those in the narrow unimodal group (Figure 3). The TIMI frame count was significantly larger in the wide unimodal and bimodal groups than that in the narrow unimodal group (41.1±19.8, 52.9±15.2, and 25.1±8.9 frames, respectively; P<0.001).

Relationships Between CBF Patterns and IMR Values
At the end of the procedure, physiological parameters were successfully obtained in all patients. The mean IMR value was 46±38 U with a median value of 33 U (8–170 U). Although post-pPCI fractional flow reserve values were similar among the 3 groups, IMR values were significantly lower in the narrow unimodal than those in the wide unimodal and the bimodal groups (20±9, 65±41, and 76±38 U, respectively; P<0.001; Figure 4). However, no significant difference existed in IMR values between the wide unimodal and the bimodal groups (Figure 4). There was no linear relationship between post-PCI IMR and fractional flow reserve values (r=0.16; P=0.15).

Figure 1. Classification of coronary blood flow patterns. Coronary blood flow patterns were classified into 3 groups according to the shape of the thermodilution curve. A, Sharp unimodal: defined as the top of the curve was sharp. B, Dull unimodal: defined as the top of the thermodilution curve was dull. C, Bimodal: defined as 2 peak shapes.

Figure 2. Definition of microvascular obstruction (MVO) on contrast-enhanced cardiac MRI (ceCMR). We defined MVO as an area of hypo-enhancement within the gadolinium hyperenhanced area of infarcted tissue present early after contrast injection and persistent when reimaged 15 minutes after contrast injection.
The prevalence of MVO on ceCMR was significantly higher in the bimodal group when compared with that in the narrow and wide unimodal groups (100%, 78%, and 30%, respectively; \( P<0.001 \); Figure 5A), even though the mean IMR values were similar between the wide unimodal and the bimodal groups. Furthermore, percentage MVO area in the bimodal group was significantly larger than that in the narrow and wide unimodal groups (15.6±6.9, 6.0±3.4 and 7.8±2.8, respectively; \( P<0.001 \); Figure 5B).

### Relationships Between CBF Patterns and ceCMR Findings

The prevalence of MVO on the ceCMR was 59% for all patients. Mean end-diastolic volume, end-systolic volume, percentage infarct area, and percent percentage MVO area were 155±34 mL, 92±28 mL, 37.6±11.2%, and 9.9±6.0%, respectively. The IMR values were significantly higher in patients with MVO than in patients without MVO (58.2±41.8 and 28.8±30.6, respectively; \( P=0.0022 \)). The ceCMR findings are shown in Table 2. Although no significant difference existed in end-diastolic volume among the three groups, the ejection fraction was significantly lower in the bimodal group than that in the narrow unimodal group (\( P<0.05 \)). Moreover, the prevalence of MVO on ceCMR was significantly higher in the bimodal group when compared with that in the narrow and wide unimodal groups (100%, 78%, and 30%, respectively; \( P<0.001 \); Figure 5A), even though the mean IMR values were similar between the wide unimodal and the bimodal groups. Furthermore, percentage MVO area in the bimodal group was significantly larger than that in the narrow and wide unimodal groups (15.6±6.9, 6.0±3.4 and 7.8±2.8, respectively; \( P<0.001 \); Figure 5B).

### Discussion

The main finding of the present study was that bimodal shape as assessed based on the thermodilution-derived CBF pattern (hazard ratio, 17.12; 95% confidence interval, 2.96–98.91; \( P=0.00151 \)) was an independent predictor of MACE. IMR value immediately after pPCI was not independently associated with MACE. Figure 6 and Table 3 clearly indicated that the thermodilution-derived CBF pattern rather than IMR value was a critical factor of 6-month death and heart failure rehospitalization after pPCI for patients with STEMI.

**Figure 3.** Peak creatine kinase (CK) and CK-MB values for each group. **A.** Peak creatine phosphokinase value was significantly higher in the bimodal group than that in the narrow and wide unimodal groups. **B.** Peak CK-MB value in the bimodal group was significantly higher than that in the other groups. PCI indicates percutaneous coronary intervention.
technique with a pressure sensor/thermistor-tipped guidewire after pPCI in patients with STEMI was associated with the presence of MVO on ceCMR and worse midterm clinical outcome rather than IMR value. To the best of our knowledge, this is a first study reporting the relationship between CBF pattern analyzed by a pressure sensor/thermistor-tipped guidewire and the clinical prognosis after STEMI.

Despite achieving normal epicardial coronary artery flow in the majority of patients with STEMI after pPCI, up to one third of patients do not achieve adequate myocardial microvascular reperfusion.3,4 This phenomenon was first described by Ito et al13 using myocardial contrast echocardiography. This phenomenon is thought to be the result of MVO after myocardial infarction. It establishes a marker of more extensive myocardial tissue damage and is associated with poor functional recovery after STEMI.14 In addition, it has been reported that this MVO is associated with a worse clinical outcome after acute myocardial infarction successfully treated by pPCI.15

CBF can be assessed invasively by an intracoronary Doppler-tipped guidewire. A previous study showed that using a Doppler wire offers a better quantitative assessment of microvascular damage in patients with STEMI treated with pPCI.16 Kawamoto et al16 reported that low average systolic peak velocity and rapid deceleration time of diastolic flow velocity of CBF spectrum immediately after pPCI reflects a greater degree of microvascular damage. Moreover, Iwakura et al17 focused attention on characteristics of Doppler wire–derived CBF velocity pattern after STEMI. They reported that the CBF velocity pattern was characterized by the appearance of abnormal retrograde flow in early systole, and rapid deceleration of

Table 2. Contrast-Enhanced Cardiac Magnetic Resonance Findings

<table>
<thead>
<tr>
<th></th>
<th>Narrow Unimodal (n=33)</th>
<th>Wide Unimodal (n=23)</th>
<th>Bimodal (n=12)</th>
<th>P Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>MVO prevalence, %</td>
<td>10 (30)</td>
<td>18 (78)</td>
<td>12 (100)</td>
<td>0.001</td>
</tr>
<tr>
<td>Ejection fraction, %</td>
<td>45±10*</td>
<td>40±11</td>
<td>34±9*</td>
<td>0.006</td>
</tr>
<tr>
<td>EDV, mL</td>
<td>151±34</td>
<td>156±38</td>
<td>159±37</td>
<td>0.80</td>
</tr>
<tr>
<td>ESV, mL</td>
<td>85±27</td>
<td>94±28</td>
<td>106±30</td>
<td>0.09</td>
</tr>
<tr>
<td>% Infarct area</td>
<td>30.9±8.5</td>
<td>37.8±8.3</td>
<td>41.3±15.1</td>
<td>0.13</td>
</tr>
<tr>
<td>% MVO area</td>
<td>6.0±3.4</td>
<td>7.8±2.8†</td>
<td>15.6±6.9†‡</td>
<td>&lt;0.001</td>
</tr>
</tbody>
</table>

Values are given as n (%) or mean±SD. EDV indicates end-diastolic volume; ESV, end-systolic volume; and MVO, microvascular obstruction.

p<0.001; †,‡P<0.0001.
the diastolic flow velocity in patients with MVO on myocardial contrast echocardiography. In addition, Yamamuro et al described that the presence of abnormal retrograde flow in early systole is associated with a worse clinical outcome after STEMI treated with pPCI. Although the Doppler wire can be used to assess MVO immediately after pPCI for patients with STEMI in the cardiac catheterization laboratory, it may be difficult to obtain an adequate flow signal in some patients because of vessel tortuosity and the heart and breathing movements of patients. Furthermore, the Doppler flow wire interrogates the resistance of the entire vessel and may not differentiate diffuse epicardial disease or residual epicardial stenosis from MVO. Currently, simultaneous measurement of phasic distal pressure and flow velocity by a dual-sensor guidewire is available in the clinical practice. Previous study reported that microvascular resistance index that was calculated as the ratio of distal coronary pressure/average peak flow velocity using this dual-sensor guidewire may predict the transmural extent of infarction after pPCI in patients with STEMI.

Recently, a single pressure sensor/thermistor-tipped guidewire has been introduced to assess coronary pressure and estimate CBF simultaneously in the cardiac catheterization laboratory during PCI. Using the thermodilution technique with this guidewire, the mean transit time of room temperature saline injected down a coronary artery can be determined and has been shown to correlate inversely with absolute flow. Previous study revealed that CBF can be measured by analyzing thermodilution curve in human coronary artery. Moreover, the IMR, that was defined as simultaneously measured distal coronary pressure divided by the inverse of the thermodilution-derived hyperemic mean transit time, was recently introduced for quantitative assessment of the minimum microcirculatory resistance. It has been reported that it is stable in the presence of varying hemodynamic conditions. Previous clinical studies described that IMR immediately after pPCI was shown to predict the presence of microvascular obstruction and left ventricular infarct size in patients with STEMI. Our result concurs with these previous studies in showing that IMR was higher in patients with MVO than in patients without MVO. The current study extends these findings by providing a result that IMR values were significantly higher in patients with MVO when compared with patients without MVO. However, in the current study, multivariate analyses failed to reveal that IMR was an independently predictor of MACEs after pPCI in patients with STEMI.

Theoretically, the IMR is calculated as distal coronary pressure at maximum hyperemia divided by the absolute coronary flow with a single pressure sensor/thermistor-tipped guidewire. According to this theory, coronary flow equals \( V \) /mean transit time, where \( V \) represents the vascular volume between injection site of the indicator and location of the sensor. Therefore, the IMR should be measured so that the vascular volume between the tip of the guiding catheter and location is always the same. However, it is almost impossible to keep the vascular volume exactly same for all measurements because diameter and taper of vessels vary widely among individuals. This is a potential reason of discrepancy between the current study and the previous IMR study. The optimal IMR cut-off value for MACE free was found to be 37 in the current study, which is slightly different from the IMR value of 32 in a previous study.

The natural course after the development of the MVO varies based on whether the cause is capillary destruction or microembolization to small arteries. Distal embolization of atherosclerotic debris or thrombotic material might be responsible for a substantial part of clinically observed MVO. When the larger size of thrombotic material plugged in the larger artery, it will migrate distally to a small artery. Therefore, although the distal embolization of large thrombotic material could be responsible for a substantial part of clinically observed MVO, it would be transient and less likelihood of large myocardial necrosis and poor clinical outcomes. However, the microvasculature can also be obstructed by interstitial and myocardial cell edema, endothelial blistering, and microvascular spasm and plugging of capillaries by erythrocytes or leukocytes. These changes in the capillary bed can result in poor perfusion of the surrounding potentially viable myocytes with resultant cell death. A previous animal study reported that this irreversible injury can be observed only 90 minutes after temporary occlusions of a major coronary artery. Therefore, when the MVO is caused by capillary destruction, it would be associated with a large area of myocardial necrosis and poor clinical outcomes.

In patients with normal coronary circulation, myocardial impedance increases during systole, and blood is milked from the intramyocardial venules into the coronary vein. However, in patients with capillary obstruction, the myocardial blood volume is pushed back into the coronary artery because myocardial blood cannot pass the capillary bed to reach into the venous circulation in systole because of the obstruction of capillaries. As a result, bimodal shape of the thermodilution curve assessed by the thermodilution technique with a pressure sensor/thermistor-tipped guidewire can be recorded. Conversely, this reverse flow in the epicardial coronary artery cannot be observed in patients with microembolization to small arteries because, in this situation, myocardial blood is smoothly squeezed into the venous circulation during systole. Therefore, it is clinically important to distinguish between capillary destruction and microembolization to small arteries in case of MVO after pPCI for STEMI. This difference can be identified by analyzing blood flow pattern on the thermodilution curve by a pressure sensor/thermistor-tipped guidewire with high reproducibility but not by IMR values. From a technical viewpoint, blood flow pattern assessment on the thermodilution curve is relatively easy to perform and interpret with a 100% technical success rate of measurement.

### Table 3. Independent Predictors of MACE

<table>
<thead>
<tr>
<th>Variables</th>
<th>HR</th>
<th>95% CI</th>
<th>P Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>MACE</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>IMR values</td>
<td>0.99</td>
<td>0.97–1.02</td>
<td>0.59</td>
</tr>
<tr>
<td>Bimodal shape pattern</td>
<td>27.82</td>
<td>2.42–320.51</td>
<td>0.007645</td>
</tr>
<tr>
<td>Multivessel disease</td>
<td>1.20</td>
<td>0.33–4.41</td>
<td>0.79</td>
</tr>
<tr>
<td>Peak CPK</td>
<td>1.00</td>
<td>0.99–1.00</td>
<td>0.27</td>
</tr>
<tr>
<td>% MVO area</td>
<td>0.99</td>
<td>0.80–1.24</td>
<td>0.96</td>
</tr>
<tr>
<td>Ejection fraction</td>
<td>0.91</td>
<td>0.82–1.01</td>
<td>0.092</td>
</tr>
</tbody>
</table>

Cl indicates confidence interval(s); CPK, creatine phosphokinase; HR, hazard ratio(s); IMR, index of microcirculatory resistance; MACE, major adverse cardiovascular event; and MVO, microvascular obstruction.
demonstrated in the current study, and it does not increase procedural time appreciably because it is obtained from a single coronary pressure wire.

Study Limitations
This study was a single-center study with a relatively small study population. Additional multicenter studies are required to reconfirm the results in a larger number of patients. Patients with Killip class III to IV and those with hemodynamic instability or recurrent AMI were excluded from the study. Intravascular ultrasound examinations were not performed in all patients to confirm that there is no residual stenosis in the infarct-related artery after stent implantation. Twenty-one patients (23%) were not examined by ceCMR because of difficulty in holding breath caused by heart failure and other contraindications for ceCMR.

Blood flow pattern on the thermodilution curve was evaluated qualitatively, even though the reproducibility was high. An automated data acquisition and analysis system, especially defining a more quantified approach to identify the bimodal distribution, would be needed to perform more accurate and feasible assessment of CBF patterns. Despite the high specificity observed in this study, the retrograde flow pattern observed in the bimodal distribution are largely effects of systole and thus may change with degree of myocardial contractility in addition to the degree of capillary destruction. Future studies may need to investigate the effects of contraction on this functional parameter. In addition, the shape of the thermodilution curve might be influenced on the location of the sensor.

Conclusions
A bimodal shape of the thermodilution curve, which may indicate myocardial edema and consequent extrinsic compression of the capillary network, is associated with microcirculatory damage and poor midterm clinical outcomes rather than IMR of the capillary network, is associated with microcirculatory damage and poor midterm clinical outcomes rather than IMR.

Acknowledgments
We thank the staff of the catheterization laboratory in Hyogo College of Medicine for their excellent assistance during the study.

Disclosures
None.

References
Thermodilution-Derived Coronary Blood Flow Pattern Immediately After Coronary Intervention as a Predictor of Microcirculatory Damage and Midterm Clinical Outcomes in Patients With ST-Segment–Elevation Myocardial Infarction

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