**Vasodilatory Capacity of the Coronary Microcirculation is Preserved in Selected Patients With Non–ST-Segment–Elevation Myocardial Infarction**

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**Background**—The use of fractional flow reserve in patients with non–ST-segment–elevation myocardial infarction (NSTEMI) is a controversial issue. We undertook a study to assess the vasodilatory capacity of the coronary microcirculation in patients with NSTEMI when compared with a model of preserved microcirculation (stable angina [SA] cohort: culprit and nonculprit vessel) and acute microcirculatory dysfunction (ST-segment–elevation myocardial infarction [STEMI] cohort). We hypothesized that the vasodilatory response of the microcirculation would be preserved in NSTEMI.

**Methods and Results**—A total of 140 patients undergoing single vessel percutaneous coronary intervention were included: 50 stable angina, 50 NSTEMI, and 40 STEMI. The index of microvascular resistance (IMR), fractional flow reserve, and coronary flow reserve were measured before stenting in the culprit vessel and in an angiographically normal nonculprit vessel in patients with SA. The resistive reserve ratio, a measure of the vasodilatory capacity of the microcirculation and calculated using the equation: baseline resistance index \( (T_mn_{\text{Base}} \times P_{a_{\text{Base}}} - P_{w_{\text{Base}}} - P_{w_{\text{Base}}})/IMR \times IMR \), where \( T_mn_{\text{Base}} \) referred to nonhyperemic transit time; \( P_{a_{\text{Base}}} \) and \( P_{d_{\text{Base}}} \) the nonhyperemic aortic and distal coronary pressures, respectively; and \( P_w \) referred to the coronary wedge pressure, was also measured. Troponin was also measured ≤24 hours after percutaneous coronary intervention. The resistive reserve ratio was significantly lower in the STEMI patients compared with the stable angina patients both culprit and nonculprit vessel (STEMI, 1.7 versus SA culprit, 2.8; \( P≤0.001 \) and SA nonculprit, 2.9; \( P<0.0001 \)) and compared with NSTEMI patients (STEMI, 2.46; \( P≤0.001 \)). The resistive reserve ratio was similar in stable angina and NSTEMI patients \( (P=0.6) \). IMR was significantly higher pre-PCI in STEMI compared with SA and NSTEMI (IMR STEMI, 36.51 versus IMR NSTEMI, 22.73 \( [P=0.01] \) versus IMR SA, 18.26 \( [P<0.0001] \)). However, there was no significant difference in IMR pre-PCI between NSTEMI and SA (IMR NSTEMI, 22.73; IMR SA, 18.26 \( [P=0.1] \)).

**Conclusions**—The vasodilatory capacity of the microcirculation is preserved in selected patients with NSTEMI. The clinical use of fractional flow reserve in the culprit vessel may be preserved in selected patients with NSTEMI. (Circ Cardiovasc Interv. 2013;6:231-236.)

**Key Words:** acute coronary syndrome • FFR • hyperemia • microcirculation

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**Fractional flow reserve (FFR)** is an increasingly important technique to guide revascularization strategies in patients with stable coronary artery disease undergoing elective percutaneous coronary intervention (PCI). The use of FFR has been associated with improved long-term outcomes and reduced healthcare costs compared with angiographic-based strategies and also a reduction in the rate of urgent revascularization when compared with contemporary medical therapy. A fundamental aspect of FFR is the ability to achieve maximal hyperemia to achieve a linear relationship between pressure and flow. Maximal coronary hyperemia is dependent on an intact microcirculation and an adequate hyperemic stimulus. Factors affecting the coronary microcirculation (eg, severe left ventricular hypertrophy) may affect the ability to achieve maximal hyperemia. Myocardial infarction (MI) can affect the distal coronary microcirculation secondary to a variety of mechanisms that include distal embolic phenomenon, microvascular stunning, and acute ischemic microvascular dysfunction. Because of the heterogeneous nature of MI, this effect may vary according to the size of myocardial infarction.
WHAT IS KNOWN

• Fractional flow reserve is a useful technique with prognostic implications to guide revascularization among stable patients. However, the use of fractional flow reserve is controversial among patients with acute coronary syndromes.
• There is a lack of data on extent of microvascular injury and microvascular vasodilatory capacity in patients with recent non–ST-segment–elevation myocardial infarction.

WHAT THE STUDY ADDS

• Microcircular injury is similar between patients with stable angina and selected patients with non–ST-segment–elevation myocardial infarction assessed after 24 hours of clinical presentation.
• The vasodilatory capacity of the microcirculation is similar between patients with stable angina and non–ST-segment–elevation myocardial infarction assessed after 24 hours of symptom onset.

and the time from infarction to FFR assessment. Thus, the validity of using FFR in patients with recent MI is not fully established.

NSTEMI is the most common form of acute myocardial infarction and is increasing in incidence. Invasive management is recommended in intermediate to high-risk patients. However, urgent coronary angiography can be associated with diagnostic uncertainty because information on ischemia may not be available because stress testing is not usually performed. Moreover, up to two thirds of patients with NSTEMI will have multi-vessel coronary disease and sometimes identifying the culprit vessel can be difficult. Thus the ability to use FFR in this population would be highly desirable. Although contemporary guidelines incorporate the use of FFR specifically in patients with non–ST-segment–elevation myocardial infarction (NSTEMI) to guide revascularization, it remains a controversial topic in interventional cardiology. Several small studies have shown that FFR assessment is reliable and valid from between 4 and 6 days after the index event and the larger, multinational Fractional Flow Reserve Versus Angiography for Multivessel Evaluation (FAME) study also included patients with NSTEMI. However, these patients were generally stable without symptoms in the previous 5 days and had unreported infarct sizes. Thus, it remains uncertain whether patients with acute NSTEMI can mount a sufficient hyperemic response to vasodilator stimuli to maintain the diagnostic accuracy of FFR. Furthermore, there is a paucity of data concerning factors involved in determining the hyperemic response in this population.

We undertook a prospective study to assess the determinants of coronary hyperemia in the culprit vessel of patients with NSTEMI and compare this response with a control group of patients with stable angina and no prior myocardial infarction and with patients with acute STEMI as a model of acute microcirculatory dysfunction.

Methods

The study population consisted of 140 patients from 2 tertiary referral centers undergoing single vessel PCI for stable angina, NSTEMI, or acute ST-segment–elevation myocardial infarction (STEMI). Patients with NSTEMI had to have pain within the preceding 5 days and an index event >24 hours from time of angiography but within the previous 7 days. Patients with STEMI undergoing primary PCI were only included if they had achieved electric and clinical reperfusion after aspiration thrombectomy. Patients were excluded if they had previous coronary artery bypass surgery or significant valvular heart disease. The Human Research Ethics Committees at St Vincent’s Hospital Melbourne and the Golden Jubilee National Hospital, Clydebank approved the study protocol.

The patients in this study had single vessel PCI of the culprit vessel only, The culprit vessel was selected on the basis of angiographic appearance, electocardiographic features, and in the stable angina cohort in conjunction with noninvasive testing. In patients with stable angina, a nonculprit vessel without significant epicardial stenosis (<30% with FFR >0.8) was also studied.

For the procedure, all patients received an initial bolus of 5000 U of intravenous heparin with additional bolus dosing to maintain an activated clotting time of 250 seconds and were receiving aspirin and clopidogrel. A 6F coronary guiding catheter was used to engage the selected coronary artery. All patients received 200 μg of intracoronary nitroglycerin in the study. A 0.014 coronary temperature and pressure-sensing guide wire was calibrated and then equalized to the guiding catheter pressure with the distal sensor placed at the ostium of the coronary artery. The wire was then passed beyond the stenosis into the distal third of the vessel.

Microvascular resistance was measured using the index of microvascular resistance (IMR) as previously described. Three milliliters of room temperature saline was injected intracoronary to produce 3 reproducible and consistent thermodilution curves. The average of the 3 values was taken as the mean baseline transit time ($T_{mn,base}$) and shown previously to be inversely proportional to coronary blood flow. Intravenous adenosine was then administered via the right femoral or an antecubital vein (140 μg/kg per minute) to achieve maximal hyperemia. Thereafter, the injection protocol was repeated to derive the hyperemic transit time ($T_{mn,hyp}$).

**IMR Calculation**

In the nonculprit vessel, a simplified method of IMR calculation was performed as previously described:

$$ IMR = \frac{P_d - P_w}{P_{hyp} - P_{aortic}} $$

In the presence of significant epicardial stenosis, IMR was calculated with the incorporation of coronary wedge pressure using the equation below:

$$ IMR = \frac{P_{aortic} - T_{mn, hyp}}{(P_{hyp} - P_{aortic})} $$

where $P_d$ is the hyperemic distal pressure, $T_{mn}$ the hyperemic mean transit time, $P_a$ the mean hyperemic aortic pressure, and $P_w$ is the coronary wedge pressure defined as the distal coronary pressure obtained during a 30-second balloon occlusion of the culprit vessel and representing recruitable collateral vessels.

**Baseline Resistance Index**

Baseline resistance (BR) index reflected resting tone in the coronary microcirculation and was calculated using the equation:

$$ BR = \frac{P_{aortic} - T_{mn, hyp}}{(P_{hyp} - P_{aortic})} $$

where $P_{aortic}$ was the resting aortic pressure, $P_{hyp}$ the resting distal pressure, and $T_{mn}$ is the transit time under resting conditions. In non-culprit vessels, a simplified method of BR index was used (BR=PD/PMN). To measure the ability of the coronary microcirculation to undergo vasodilatation in response to a pharmacological hyperemic stimulus, the resistive reserve ratio (RDR) was calculated. The RDR is given by the following equation:

$$ RDR = \frac{BR - index}{IMR} $$
The RRR is essentially a marker of the ability of the coronary microcirculation to change from baseline to minimal resistance in response to adenosine. It reflects the ability to achieve maximal hyperemia.

Coronary flow reserve was defined as the mean resting transit time divided by the mean hyperemic transit time. FFR was defined as the mean distal coronary pressure divided by the mean aortic pressure during hyperemia. Care was taken to ensure that the distal sensor was in the same position between measurements to avoid errors in transit time acquisition. All patients had a symptomatic and hemodynamic response to adenosine. Troponin was measured 12 to 24 hours after PCI.

**Study Protocol**

Physiological indices were measured in the culprit artery at baseline. In patients with stable angina, indices were also measured in a nonculprit vessel at baseline. Stenting of the culprit vessel was then performed. In the STEMI cohort, physiological measures were obtained after restoration of TIMI III flow in the epicardial vessel after aspiration thrombectomy.

**Statistical Analysis**

Data were analyzed using SPSS (SPSS, Inc, Chicago, IL) statistical software package. Normality of data was assessed with the Kolmogorov–Smirnov statistic. Nonparametric tests were used where appropriate. Continuous variables are summarized as means ± SD unless otherwise stated. Non-normally distributed data are summarized as the median and interquartile range. Between-group differences were compared using 1-way ANOVA or Kruskal–Wallis test as appropriate. Bonferroni correction was applied for multiple comparisons. Pearson χ² test was used to test for association between categorical risk factors across patient groups. On the basis of preliminary data, we hypothesized a similar mean SD of 2.18 in RRR. Using a significance level of 5%, we calculated that 32 patients in each patient group would be needed to provide a power of 90% to detect a between-group difference of 2 U of RRR. A P value of <0.05 was considered statistically significant.

**Results**

A total of 140 patients were recruited in 2 major tertiary referral centers in the United Kingdom and Australia: 50 each with stable angina (SA), NSTEMI, and 40 with STEMI. Demographics of the population are shown in Table 1 and coronary physiological values in Table 2. Importantly, there was no difference in age or sex across the 3 groups. However, the SA group had a significantly higher number of patients with hyperlipidemia than the other groups.

The mean time to PCI from symptom onset for the NSTEMI group was 4.26 ± 1.7 days and in the STEMI cohort 0.18 ± 0.16 days. In the stable angina group the mean time from symptom onset to PCI was 111.9 ± 102.6 days.

The mean post-PCI troponin concentration was higher in STEMI (33.1 µg/L) patients compared with post-PCI troponin concentrations in patients with stable angina (0.41 µg/L) and NSTEMI (1.41 µg/L; P < 0.01). Although in the sample there was a higher troponin value in the NSTEMI group than stable angina, this did not reach formal statistical significance.

**Coronary Physiology**

The RRR was significantly different across the different patient groups (ANOVA P < 0.0001; Figure 1). In a nonculprit vessel without significant stenosis the RRR was 2.9 (2.3, 3.9). There was no significant difference between this value and that of the RRR of stable angina or NSTEMI (2.8 [1.7, 4.8]; P = 0.61) and STEMI (2.46 [1.6, 3.9]; P = 0.75 and 2.46 [1.6, 3.9]; P = 0.61, respectively). However, the RRR was significantly lower in the STEMI patients (1.7 [1.2, 2.3]; P < 0.0001).

The RRR was lower in STEMI patients compared with NSTEMI patients (STEMI, 1.7; NSTEMI, 2.46; P < 0.001), yet there was no difference observed between SA and NSTEMI (2.8 [1.7, 4.8] versus 2.46 [1.6, 3.9]; P = 0.61).

In the nonculprit vessel, the IMR was 16.85 ± 9.06. There was no difference observed between IMR in stable angina in the culprit vessel compared with the nonculprit vessel (IMR nonculprit 16.85 ± 9.06 versus IMR SA, 18.26 ± 9.15; P = 0.44). However, as expected, IMR was higher in NSTEMI and STEMI compared with the nonculprit vessel (IMR NSTEMI, 22.73 ± 11.3 [P = 0.015]; IMR STEMI, 36.51 ± 35.77 [P < 0.0001]). IMR was significantly higher pre-PCI in STEMI compared with SA and NSTEMI (IMR STEMI, 36.51 versus IMR NSTEMI, 22.73 [P = 0.01] versus IMR SA, 18.26 [P < 0.0001]; Figure 2). However, there was no significant difference in IMR pre-PCI between NSTEMI and SA (IMR NSTEMI, 22.73; IMR SA, 18.26 [P = 0.1]).

Although pre-PCI coronary flow reserve values were numerically higher in the stable angina and NSTEMI populations compared with STEMI, these differences did not reach statistical significance (P = 0.3).

There was a negative correlation between IMR pre-PCI and the resistive reserve ratio (r = −0.28; P = 0.003), RRR and troponin (r = −0.36; P = 0.001), and IMR pre-PCI and troponin (r = −0.3; P = 0.04). There was no difference between baseline resistance index or FFR across the 3 groups. Ejection fraction was significantly different across the 3 groups with NSTEMI patients demonstrating the lowest values.

**Discussion**

We have demonstrated that the resistive reserve ratio, a novel marker of the vasodilatory capacity of the coronary microcirculation, is not significantly different among patients with NSTEMI when compared with a nonculprit vessel in patients with stable angina. We have also shown that the ratio in NSTEMI is similar to the culprit vessel of patients with stable angina. In STEMI patients with acute microvascular dysfunction, the resistive reserve ratio was lower in the culprit vessel compared with stable angina and NSTEMI. These findings suggest a preserved capacity of the coronary microcirculation to achieve a maximal hyperemic response in selected NSTEMI patients.

We have also shown that, as expected, microvascular injury in the culprit vessel pre-PCI is greater in patients with acute STEMI compared with stable angina and NSTEMI, but we found no difference in the degree of microvascular dysfunction pre-PCI between patients with NSTEMI and SA as highlighted by the similar IMR and coronary flow reserve values. These differences mirrored those of the resistive reserve ratio across subgroups and suggest that the use of FFR in the culprit vessel may be valid in patients with NSTEMI.

**Microvascular Dysfunction in Acute Coronary Syndromes and SA**

Because of the acuity of patients and the larger risk to the microcirculation that the STEMI group possesses, the role of the microcirculation in predicting STEMI outcomes after reperfusion has been well studied. The presence of worse microvascular function immediately after primary PCI is associated with larger infarct size and reduced left ventricular viability in...
the longer term.17 Likewise after primary PCI, the presence of microvascular dysfunction assessed using intracoronary contrast echocardiography was associated with an increase in cardiac mortality during the mean follow-up period of 48 months in a large STEMI cohort.18 Such microcirculatory dysfunction found among STEMI patients is one of the primary reasons why the attainment of maximal hyperemia is unreliable.

Microcirculatory dysfunction in the NSTEMI cohort has not been extensively investigated. However, elevation in microvascular resistance has been implicated in the genesis of ischemia in the unstable angina cohort.19 Furthermore, in the NSTEMI population and using indirect angiographic measures of microvascular function, microvascular dysfunction was associated with a higher incidence of adverse outcomes after PCI and was the strongest predictor of death, MI, or recurrent ischemia in the short term.20 However, in general, microvascular dysfunction in NSTEMI is less extensive than in STEMI, and thus attaining maximal hyperemia may be possible in this population.

**Concerns With Using FFR in Acute Coronary Syndromes**

The role of FFR in the assessment of patients with SA undergoing coronary angiography is clearly defined with a large body of work demonstrating superior outcomes when compared with a conventional angiographic-guided strategy.1,21 However, the accuracy and utility of FFR to influence revascularization decisions in patients with acute coronary syndromes is a controversial issue.22

In the setting of emergency primary PCI for acute STEMI, there is no role for FFR in the culprit artery. In the NSTEMI setting, angiography is often performed >24 hours after initial presentation. However, even at this later time point, the ability of the microcirculation to achieve maximal hyperemia and establish the critical linear relationship between pressure and flow necessary for the assessment of FFR in the culprit territory has been questioned.

The attainment of maximal hyperemia is based on the assumption of a normal distal microvascular bed. However, PET studies have demonstrated impaired microcirculatory function in the infarcted territory compared with healthy controls ≤6 months after AMI.23 Thus in patients with recent MI, microvascular injury, stunning, and edema can result in a failure to achieve minimal resistance and FFR values may be falsely elevated.23 Tamita et al24 highlighted this by demonstrating a higher post-PCI FFR in patients with STEMI compared with patients with stable angina, despite similar intravascular ultrasound parameters. Thus in patients with microvascular dysfunction, the assessment of FFR may be unreliable.

We have shown that although microvascular injury is significantly higher in patients with STEMI, our model of acute microvascular dysfunction, the observed injury is equivalent in patients with SA and NSTEMI (at a mean time from symptom onset to angiography of 4 days). Furthermore, the resistive reserve ratio was similar in SA and NSTEMI, suggesting similar potential to achieve maximal hyperemia in this population. Baseline resistance index was also similar across groups suggesting that observed differences in RRR reflect changes in vasodilatory function rather than higher baseline coronary tone.

We have also demonstrated a negative correlation between maximal troponin and the resistive reserve ratio, as well as IMR pre-PCI and the resistive reserve ratio. These findings suggest that the ability of the coronary microcirculation to vasodilate is related to the severity of the initial injury and the extent of microvascular dysfunction and explains the lower RRR values in the acute STEMI population. The lower pre-PCI IMR and lower troponin values also explain why the RRR (and thus hyperemic response) is maintained in NSTEMI. These findings are also influenced by the timing of measurements, with the STEMI group being

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<th>Table 1. Patient Demographics Across Different Subgroups</th>
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<td>Stable Angina</td>
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<tr>
<td>Age†</td>
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<tr>
<td>Men, (n)</td>
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<td>Time to PCI, d†</td>
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<td>Diabetes mellitus (n)</td>
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<td>Hypertension (n)</td>
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<td>Ever smoked (n)</td>
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<td>Hyperlipidemia (n)</td>
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<td>Troponin post†</td>
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<td>Ejection fraction (%)†</td>
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<td>BMI†</td>
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BMI indicates body mass index; NSTEMI, non–ST-segment–elevation myocardial infarction; n, number of individuals; PCI, percutaneous coronary intervention; and STEMI, ST-segment–elevation myocardial infarction.†Results expressed as mean±SD and compared using 1-way ANOVA. All other comparisons were made using Pearson χ² test. |

<table>
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<th>Table 2. Differences in Coronary Physiology Across Subgroups</th>
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<td>Nonculprit</td>
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<td>FFR pre†</td>
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<td>IMR pre†</td>
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<td>RRR*</td>
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<tr>
<td>Base res†</td>
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<tr>
<td>CFR pre†</td>
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<td>CFI</td>
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Base Res indicates baseline resistance index; CFR, coronary flow reserve; CFI, Collateral Flow Index; FFR, fractional flow reserve, IMR, index of microcirculatory resistance; NSTEMI, non–ST-segment–elevation myocardial infarction; RRR, resistive reserve ratio; and STEMI, ST-segment–elevation myocardial infarction.†Results expressed as median and interquartile range and compared using Kruskal–Wallis ANOVA. †Results expressed as mean±SD and compared using 1-way ANOVA.
evaluated within the initial 12 hours and the NSTEMI population undergoing evaluation >24 hours after the index event, thus providing time for the microcirculation to settle.

Current FFR Thresholds
The original validation studies that determined the FFR threshold for ischemia were all performed in stable patients. However, there have been several studies that have aimed to establish and validate FFR thresholds for ischemia in patients with acute coronary syndromes. In 48 stabilized patients with recent MI, Samady et al9 compared FFR in the infarct related artery with noninvasive findings using SPECT and myocardial contrast echocardiography. Patients had a mean time to angiography of 3.7 days with 73% of patients with STEMI. The group demonstrated that an $FFR \leq 0.75$ had 91% sensitivity, 93% specificity, and a diagnostic accuracy of 92% for detecting reversible ischemia. They provided an optimal cutoff FFR value of $\leq 0.78$ for detecting reversible ischemia using receiver operating characteristic analysis. De Bruyne et al10 demonstrated that an $FFR \leq 0.75$ in a culprit vessel $\geq 6$ days after an AMI was still predictive of reversible ischemia shown on noninvasive SPECT imaging. Thus, there is some evidence for the use of FFR to determine ischemia in the culprit territory 4 to 6 days after acute coronary syndromes. Our results are in accordance with these studies with the mean time from symptom onset to PCI being 4.2 days with a median time of 4 days.

Clinical Application
The results of this study suggest that in patients with NSTEMI the ability of the coronary microcirculation to achieve hyperemia sufficient to maintain the diagnostic use of FFR is similar to a nonculprit and culprit vessel of patients with SA. Furthermore, the observed correlations between IMR and troponin and the resistive reserve ratio suggest that the extent of myocardial injury and microvascular dysfunction are important determinants of the hyperemic response. Thus in patients with MI with small troponin elevations, and lower IMR values who are evaluated >24 hours from their event, FFR use may be valid. Of interest, some patients with acute STEMI demonstrated a similar resistive reserve ratio as the nonculprit control group. Although this does not advocate the use of FFR in acute STEMI, it does suggest that in patients with smaller infarcts with more rapid reperfusion, a sufficient hyperemic response may be possible. This strategy warrants conformation in a larger study.

Limitations
This is a selected population of patients with single vessel coronary artery disease, and our results may not necessarily reflect those of the general NSTEMI population. However, as this is a study examining the vasodilatory capacity of the culprit territory, this is a minor issue.

The number of patients with hyperlipidemia were significantly higher in the stable angina group. This may have possibly reduced the RRR value in the SA population so that the differences between SA and NSTEMI were attenuated in our cohort. However, both IMR and coronary flow reserve values were similar in the SA and NSTEMI populations, suggesting that any effect on the microcirculation and thus resistance reserve ratio that such differences may have would be minimal.

Ejection fraction was actually higher in the STEMI cohort than in the NSTEMI cohort in our population. This may reflect the rapid reperfusion the STEMI patients achieved in our population from symptom onset to primary PCI, but also a selection bias whereby only the more stable patients were selected to allow assessment of FFR/IMR after thrombus aspiration. However, despite this, the STEMI population in our cohort still had higher levels of myocardial and microvascular injury that would be anticipated from a general STEMI cohort.

Conclusions
We have shown that the vasodilatory capacity of the microcirculation is preserved in selected patients with NSTEMI and that baseline levels of microvascular injury are similar in SA and NSTEMI. Further randomized studies in larger groups of patients are required to assess the clinical use of FFR in NSTEMI.
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Disclosures

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