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# Pressure Wire Assessment in Patients with An Acute Coronary Syndrome

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## Short Communication

### Abstract

Patients with stable coronary artery disease with a fractional flow reserve (FFR) of  $>0.75$  to  $0.8$  can be safely managed with medical therapy with lower major adverse cardiac events. This avoids the risks associated with percutaneous coronary intervention (PCI) including stent thrombosis and restenosis. In patients with an acute coronary syndrome (ACS) the value of FFR is unclear as maximal hyperemia is required. In patients with an ACS microvascular changes may prevent vasodilatation thus affecting the validity of FFR. Studies have shown that FFR can be safely performed with high accuracy if performed a few days after an infarct.

## Introduction

The use of FFR in patients with stable coronary disease is well established. The assessment of coronary lesions by coronary angiography is subjective and lesions can be underestimated or overestimated. This can lead to patients having PCI that is unnecessary. FFR enables the physician to identify lesions that are physiologically flow limiting and therefore avoids unnecessary PCI. The use of FFR in patients with an ACS is unclear since maximal hyperemia may not be achievable. In patients who have a lesion in a non-infarct related artery, the role of FFR is questionable.

## Discussion

FFR assesses the significance of a coronary artery lesion during maximal vasodilatation through the use of vasodilators such as adenosine [1]. The FFR is calculated as a ratio using a pressure wire to assess the distal coronary pressure divided by the mean aortic pressure. In a normal coronary artery without any obstruction the FFR is  $>0.8$ . An FFR value of  $<0.80$  indicates significant coronary artery stenosis causing ischemia with an accuracy of more than 90% [1-3]. The risk associated with performing a pressure wire assessment for a lesion of unknown significance is low and outweighs the clinical information that is obtained. This enables the clinician to avoid

an intervention that is not necessary as well as perform one that is required. The measurement of FFR is affected by the presence of small vessel disease, diffuse coronary artery disease and left ventricular hypertrophy. In these situations, there is limited blood flow once a vasodilator is administered and therefore a reduction in distal coronary pressure resulting in an inaccurate FFR measurement [1].

The annual rate of death or myocardial infarction is approximately 1% in patients with a negative FFR i.e.  $>0.80$ . This is lower than the rate after performing PCI [4]. In patients who have a positive FFR ( $<0.75$  to  $0.80$ ) outcomes are worse if PCI is deferred compared to those who undergo revascularization [5].

FFR values of  $<0.75$  have a high sensitivity (88%), specificity (100%), positive predictive value (100%) with an overall accuracy of 93% for detecting a reduction in coronary blood flow in patients with stable coronary artery disease. In patients with an ACS the use of FFR is limited. FFR measurements are not reliable when the coronary artery does not have thrombolysis in myocardial infarction flow grade 3 (TIMI) [6]. In patients who have an ST elevation myocardial infarction (STEMI) there is microvascular dysfunction as embolization of plaque occurs distally as well as inflammation and vasoconstriction. The application of FFR requires minimal microvascular resistance which is not the case in patients with STEMI therefore FFR should not be used in these patients as the FFR measurement is likely to be inaccurate. In patients with NSTEMI there is less microvascular dysfunction and the FFR maybe as reliable as stable patients. Although microvascular dysfunction maybe less in NSTEMI patients the FFR may not be significant i.e.  $<0.80$ . This is because the clot may dissolve with the initiation of medical therapy resulting in the lesion to be less flow limiting resulting in a pressure wire measurement with less of a pressure gradient [7]. Although an FFR cut off value of  $<0.75$  has been shown to be significant in patients with stable coronary disease, this value may not be appropriate in patients with a NSTEMI due to the physiological differences as described above.

Studies have assessed the validity of FFR in NSTEMI patients. FFR was performed in patients  $>6$  days after a myocardial infarction (MI). In this study the validity of FFR was compared to single-photon emission computed tomography (SPECT) myocardial perfusion imaging before and after PCI. Patients who had a positive SPECT pre-PCI had a lower FFR than those

patients how had a negative SPECT ( $p=0.0079$ ). The sensitivity and specificity of FFR of  $<0.75$  to detect a lesion on SPECT was 82% and 87% respectively [2]. In a further similar study Samady et al. assessed the use of FFR in patients with an acute MI and compared this to SPECT. SPECT was performed in 48 patients approximately 4 days after an MI with 23 patients undergoing myocardial contrast echocardiography. FFR and PCI were performed as necessary. Follow up SPECT was performed at 11 weeks to assess for reversibility when compared to baseline SPECT. The sensitivity, specificity, positive and negative predictive value of an FFR value of  $<0.75$  for detecting true reversibility on SPECT was 88%, 93%, 93% and 91% respectively. With the use of myocardial contrast echocardiography, the accuracy of detecting FFR of  $<0.75$  was 90%, 100%, 100% and 75% respectively. The FFR value for detecting significant inducible ischemia on non-invasive imaging was 0.78 [8].

FFR measurements in patients with an acute MI appear to be valid when compared to patients without an MI. 43 patients with an acute MI were matched to 25 controls without an MI. Lesion length and minimal luminal diameter were similar in both groups. Ejection fraction was lower in patients who had an acute MI than those without an MI ( $p=0.05$ ). There was a strong correlation between the percentage of diameter stenosis and FFR measurement in patients with ( $p<0.001$ ) and without an MI ( $p=0.003$ ). There was no significant difference in FFR values between patients with an acute MI and control patients [9].

Deferring PCI in patients with an MI or unstable angina with an FFR of  $>0.75$  appears to be safe. At 1 year follow up cardiac events (cardiac death, MI, revascularization) occurred in 10% of patients. There was no significant difference in event rates between patients with unstable angina, MI or stable angina (9% versus 13%  $P=0.44$ ). Furthermore, there was no significant difference in outcomes in patients with and without lesions associated with positive noninvasive test results (9% vs 10%  $p=1.00$ ). 88% of patients were free from angina or had class I angina at 1 year based on the Canadian Cardiovascular Society angina class. Therefore, patients with coronary lesions can be managed safely without revascularization if the FFR is  $>0.75$  [10]. In a further study the outcomes in 111 patients who had revascularization deferred with an FFR  $>0.75$  was assessed at 12 months. ACS was present in 35 patients. The clinical, angiographic, and coronary hemodynamics were similar amongst the patients. In the patients with an ACS there were 3 deaths, 1 MI and 6 patients required target vessel revascularization (TVR). In the 76 patients without an ACS there were 5 deaths, 1 MI and 7 TVR. Therefore, in ACS patients the event rates were low suggesting that patients with an FFR of  $>0.75$  can be managed safely with medical therapy [11].

The use of FFR in patients with an ACS appears to influence revascularization strategy. 350 patients with a non ST-segment elevation myocardial infarction (NSTEMI) were randomized to FFR guided therapy or angiography guided therapy. FFR was also performed in the angiography guided group but the result was not disclosed to the physician. Medical therapy was often

prescribed in the FFR guided group (22.4% versus 13.2% in the angiography guided group  $p=0.022$ ). Disclosure of the FFR measurement resulted in a change of revascularization therapy in 21.6% of patients. At 12 months, revascularization rates were lower in the FFR group when compared to the angiography guided group (79% versus 86.8% respectively  $p=0.054$ ) [12].

FFR may be of value in patient with an acute MI in the presence of multivessel disease. In these patients, it can be difficult to determine which lesions are ischemic. Using coronary angiography is subjective and may overestimate or underestimate the lesion severity. In the FAME trial 1005 patients with multivessel disease were randomized to angiography guided PCI or FFR guided PCI. Patients with stable angina and those with a NSTEMI were included. The primary end point was the composite of death, myocardial infarction and repeat revascularization. The primary end point occurred in 18.3% of patients in the angiography guided PCI group and in 13.2% in the FFR guided PCI group ( $P=0.02$ ). At 1 year, the all cause mortality was 3.0% and 1.8% respectively ( $P=0.19$ ). Myocardial infarction rates were significantly higher in the angiography group at 8.7% versus 5.7% in the FFR guided group ( $P=0.07$ ). Repeat revascularization was required more frequently in the angiography group; 9.5% versus 6.5% respectively ( $P=0.08$ ). Higher number of patients were free from anginal symptoms at 1 year in the FFR group; 81.3% compared to 77.9% of the patients in the angiography group ( $P=0.20$ ). Therefore, in patients with multivessel disease the use of FFR reduced the composite rate of death, myocardial infarction, and repeat revascularization at 1 year [13]. Retrospective studies have supported similar findings with FFR guided PCI resulting in better survival rates in patients who have multivessel coronary artery disease [14,15].

The assessment of non-culprit vessel disease is often delayed as it is unclear whether microvascular dysfunction occurs in the non-culprit vessel during an acute coronary event. In a prospective study the validity of FFR in a non-culprit vessels after an acute coronary event was assessed. The FFR measurement of 101 patients who had bystander coronary artery disease was assessed immediately after performing PCI of the culprit vessel in NSTEMI and STEMI patients. The FFR measurement was then repeated approximately 35 days later. The FFR value of the non-culprit vessel was not significantly different when measured acutely and during follow up. Other factors such as TIMI flow, percentage diameter stenosis and microvascular resistance was not different acutely or during follow up [16].

## Conclusion

In patients with NSTEMI and unstable angina FFR appears to be feasible and provides accurate results. Lower major adverse cardiac events have been reported when FFR is used in NSTEMI patients when compared to angiography guided therapy. Small studies also appear to favour the use of FFR in assessing the severity of non-culprit vessels during an MI.

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