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The coronary pressure wire for decision-making in the real world

WILLIAMS OMOROGIUWA, MICHAEL FISHER

Abstract

Coronary angiography is an imperfect tool for assessing the functional significance of lesions; while this may be determined non-invasively using myocardial perfusion scanning or stress echocardiography, it is often not done. In these circumstances the coronary pressure-derived fractional flow reserve (FFR) serves as an alternative, lesion-specific means of assessing physiological importance.

FFR is an invaluable tool not only in determining whether a lesion is functionally significant and should be tackled, but it also ensures that the appropriate physiological outcome is obtained from coronary intervention.

Key words: fractional flow reserve (FFR), coronary flow reserve (CFR), coronary pressure wire, percutaneous coronary intervention (PCI), coronary artery disease (CAD), intravascular ultrasound (IVUS).

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Introduction

Coronary blood flow is autoregulated between aortic pressures of 50–150 mmHg. Between these values an increase in coronary perfusion pressure is counterbalanced by an increase in arteriolar sphincter tone, and *vice versa*, to maintain constant coronary flow. Outside this range autoregulation will fail and changes in aortic pressure will lead to similarly directed changes in myocardial blood flow.[1] Stenosis sufficient to cause resistance to coronary blood flow will therefore induce arteriolar vasodilatation in an effort to maintain resting blood flow.

In an attempt to provide a measure of the degree of 'surplus flow' available to the myocardium by this mechanism, Gould defined coronary flow reserve (CFR) in 1974 as the ratio of maximum achievable coronary flow to resting flow.[2] A reduction in CFR could be due to a reduction in maximal flow, an increase in resting flow or a combination of both. The application of CFR in clinical practice is limited for several reasons: often increases in heart rate, contractility and blood pressure are observed in the catheter lab and these can alter CFR. In addition, pathological conditions such as myocardial infarction, valvular heart disease and left ventricular hypertrophy can affect resting flow. Furthermore, CFR is not lesion-specific as it is influenced by both epicardial and microvascular resistance, whereas only the epicardial part of the coronary circulation can be treated in the catheter lab. It follows, therefore, that CFR is significantly limited as a clinically applicable tool, in that its dependence on resting flow makes it difficult to define a threshold value for distinguishing between functionally significant and functionally insignificant stenosis.

In contrast to CFR, the pressure-derived fractional flow reserve (FFR) is based on maximal myocardial perfusion only, as assessed by coronary pressure measurement. FFR is defined as the ratio of maximum achievable blood flow in the presence of a stenosis to maximum achievable normal flow.[3-8] The exercise level at which myocardial ischaemia will occur is a function of this maximum achievable flow through the stenotic vessel, not of resting flow or CFR. The determination of FFR at maximal hyperaemia theoretically renders it independent of baseline haemodynamic changes. The advantages of FFR are listed in table 1.

This paper provides an introduction to the theory underlying the determination of FFR and gives a brief overview of the literature concerning both the theoretical and practical aspects of its application. The main objective, however, is to emphasise the real and practical problems that the fractional flow reserve helps the interventionist to address.

Table 1. Advantages of FFR_{myo}

- It is a lesion-specific index of epicardial stenosis severity
- It is independent of heart rate, blood pressure and contractility
- A value of 0.75 distinguishes functionally significant and insignificant lesions with an accuracy of 95%
- It can be applied in single vessel, multivessel and left main coronary artery disease, and there is no need for a normal coronary artery for comparison
- FFR_{myo} has an unequivocal normal value of 1.0 for every patient, every coronary artery and every myocardial distribution
- It can easily be obtained during interventional procedures

Royal Liverpool University Hospital, Prescot Road, Liverpool, L7 8XP.
Williams Omorogiuwa, Registrar in Cardiology
Michael Fisher, Consultant Cardiologist

Correspondence to: Dr Michael Fisher
(email: Michael.fisher@rlbuht.nhs.uk)

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Limitations of coronary angiography

Over the past four decades, coronary angiography has played an important role in the diagnosis and treatment of patients with coronary heart disease.[9,10] A number of well-recognised limitations do exist, however, not least inter- and intra-observer error.[11] Automated techniques do not solve this problem as studies have confirmed a poor correlation between computerised determination of coronary narrowing and physiological measures of coronary function, especially in ranges between 50–90% stenosis.[12] The angiogram has also been shown to be a poor judge of true stenosis severity, as judged by pathological findings at autopsy.[13-15] In patients with coronary disease that appeared significant on angiography, outcome was related to the extent of inducible ischaemia, not to the anatomical degree of narrowing.[16,17] Quality of life and prognosis, therefore, depend upon the functional significance of a lesion, which cannot be determined reliably from an angiogram alone.[18] A functionally significant lesion is one that limits maximum achievable coronary flow to such an extent that myocardial ischaemia can be induced in that arterial territory if the patient is sufficiently stressed.

Coronary angiography is also limited in its ability to construct complicated spatial structures from orthogonal projections.[19] In judging stenosis severity the diseased segment diameter is compared to the adjacent (apparently normal) segment diameter, which may not be normal at all, as demonstrated repeatedly by intravascular ultrasound studies.[20] Stenoses of equal angiographic severity may have different impacts on blood flow dynamics as a result of differences in stenosis shape and eccentricity.[21] The physiological impact of a stenosis on coronary blood flow is also dependent on other factors, such as aortic pressure, central venous pressure, collateral circulation, resistance and size of the dependent myocardial bed.[22,23]

In an attempt to overcome these limitations, techniques such as coronary angioscopy, intravascular ultrasound, intracoronary Doppler flow velocity measurements and intracoronary pressure measurements have been developed. These in turn have a number of significant problems, however. The limitation of the current Doppler probe method is that only changes in flow velocity, rather than absolute velocity or volumetric flow, are measurable. The change in flow velocity is directly proportional to changes in volumetric flow only when vessel dimensions are constant at the site of the sample volume. Furthermore, there is concern that changes in luminal diameter and arterial cross-sectional area during interventions are not reflected in measurements of flow velocity, thus potentially causing underestimation of the true volume flow.[24]

Intravascular ultrasound (IVUS) is a safe,[25] accurate[26] and reproducible[27,28] method of detecting vessel wall pathology[29] which lends insight into the dynamic changes before, after and late after PCI.[30,31] The two-dimensional tomographic images provided by IVUS also permit 360 degrees characterisation of arterial lumen dimensions in regions that are difficult to assess using conventional angiography, such as the left main coronary artery and the ostia of the left anterior descending, left circumflex and right coronary arteries.[32] Nevertheless, a number of factors have limited the widespread use of IVUS during PCI, including its cost, its cumbersome setup for occasional IVUS users, the steep learning curve for online IVUS interpretation, and the improved outcomes associated with the routine use of stents. Current catheter-based IVUS systems are also limited by their inability to assess lumen diameters smaller than 1.0 mm, owing to the catheter size and ring down artefact.[33,34] Also, the limited spatial resolution of a 30-MHz imaging transducer (theoretical spatial resolution, 80 µm; usual spatial resolution, 120–150 µm) makes routine IVUS usage somewhat problematic.

Coronary angioscopy complements angiography by characterising plaque composition and illuminating the presence of thrombus or endoluminal irregularities, such as ulcerations, fissures or tears. It has its limitations, though, the most significant being the need to create a blood-free field. This is achieved with a proximal occluding balloon, which itself can create complications, the most devastating of them including coronary rupture, dissection, thrombosis or arrhythmia. The alternative system uses a smaller catheter to flush saline continuously in front of the angioscope to displace blood transiently, but this technique requires removal of the guidewire before acquisition of each image. The catheter design (3.0 to 5.0F) of both systems precludes angioscopic evaluation of small vessels (< 2 mm) and renders assessment of cross-stenotic lesions difficult. Moreover, the subjectivity of colour interpretation has been criticised, so efforts have been made to develop an automated analysis system of angioscopic images. Finally, angioscopy visualises only the luminal surface, and although changes in the vessel wall are reflected on the surface, this might not be sufficiently sensitive to detect subtle alterations in plaque composition, a feature that has been raised in recent comparisons of imaging modalities.[35]

Determination of FFR

A diagram to illustrate determination of FFR is given in figure 1.

$$FFR_{myo} = \frac{\text{Maximum myocardial blood flow in the presence of a stenosis } (Q_s)}{\text{Normal maximum myocardial blood flow } (Q_n)}$$

Ohm's law states that in the presence of constant resistance (R), a pressure gradient (P) across a circuit is proportional to the flow (Q):

$$R = \frac{P}{Q} \quad \text{or} \quad Q = \frac{P}{R}$$

$$\text{Therefore} \qquad Q_s = \frac{P_d - P_v}{R} \qquad \text{and} \qquad Q_n = \frac{P_a - P_v}{R}$$

R is myocardial resistance at maximum vasodilatation, which is constant, minimal and cancels out, hence:

$$FFR_{myo} = \frac{P_d - P_v}{P_a - P_v}$$

P_v is mean central venous pressure obtained at maximal hyperaemia. It is close to zero and can be treated as such as a mathematical approximation, hence:

$$FFR_{myo} = \frac{P_d}{P_a}$$

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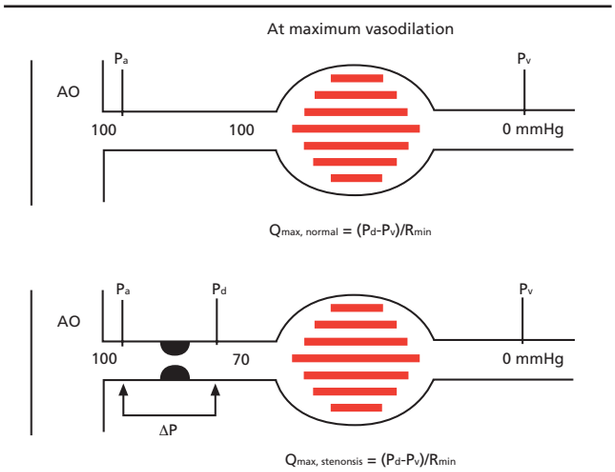
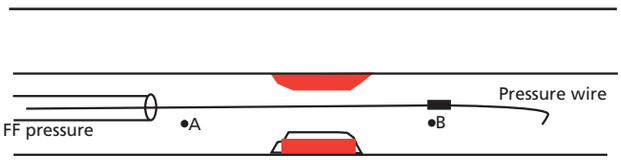
Figure 1. Determination of fractional flow reserve



P_a = mean aortic pressure
P_d = mean hyperaemic distal coronary pressure
P_v = mean hyperaemic central venous pressure. It is not zero but can be treated as such as a mathematical approximation
R = Myocardial resistance
ΔP = Translesional pressure gradient

In this example at maximal vasodilatation, myocardial perfusion pressure would be 100 mmHg if no stenosis was present. As a result of stenosis this pressure has decreased to 70 mmHg. Therefore, at maximal vasodilatation the ratio between maximum achievable flow in the presence of that stenosis and normal maximal flow is (70-0)/ (100-0).
This is the fraction of normal maximum flow that is preserved despite the presence of stenosis and is called FFRmyo. In this case it is 0.70.
It is this fraction that determines the functional effect of stenosis on myocardial blood flow, not the hyperaemic pressure gradient (ΔP).

Figure 2. Instrumentation for determination of fractional flow reserve (FFR)



0.014" guide wire - RADI medical
Piezoelectric crystal at proximal part of radio-opaque tip measures pressure
FFR is continuously derived from the ratio of the wire pressure to the guide catheter pressure
Pullback across lesion to confirm that FFR normalises

Table 2. Correlation studies between FFR and standard non-invasive tests to detect the optimal cut-off value for discrimination of ischaemia

Author Journal/year	No. of pts	Non-invasive stress test	Cut-off value	Correlation
De Bruyne *et al.* *Circulation* 1995	60	Negative exercise test	<0.72	87%
Pijls *et al.* *Circulation* 1995	60	Positive exercise test	≥0.74	98%
Bartunek *et al.* *JACC* 1996	75	Negative dobutamine echo	<0.75	95%
Pijls *et al.* *NEJM* 1996	45	Negative/positive exercise test, dobutamine echo, and SPECT	<0.75/ ≥0.75	93%

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As P_d and P_a can be measured continuously using the coronary pressure wire and guide catheter respectively (see figure 2), it follows that FFR_{myo} can be determined continuously in the catheter lab.

When the coronary wedge pressure (P_w) is known, the coronary fractional flow reserve (FFR_{cor}) can be described as:

$$FFR_{cor} = \frac{P_d - P_w}{P_a - P_w}$$

The difference between FFR_{myo} and FFR_{cor} is FFR_{coll} (FFR collateral):

$$FFR_{coll} = \frac{P_w - P_v}{P_a - P_v}$$

The FFR as a guide to practical decision-making

The decision whether to carry out percutaneous coronary intervention (PCI) on a severely or minimally occluded vessel is an easy one. The real question arises with stenoses of intermediate severity, and this is where FFR has carved its niche. How do we determine that an intermediate lesion is functionally significant and responsible for the patient's symptoms? Despite the availability of non-invasive tests to help answer this question, patients still come to the catheter laboratory without any functional data.[36] It has been shown that in a randomly selected group of asymptomatic 60-year-old men the prevalence of apparently significant coronary stenosis is 20%.[37] Therefore, one must assume that, in a number of such patients, the presence of a lesion may be coincidental and that the relation between lesion and symptoms is unclear.

FFR is a functional index of stenosis severity and it has been well demonstrated that the cut-off value of 0.75 discriminates between functionally significant and insignificant stenoses.[6,7] The diagnostic accuracy of FFR for this purpose is over 90% and is higher than for any other invasive or non-invasive test, with a specificity of 100%[4-7] (see also table 2).

That the FFR is feasible, safe, reliable and reproducible was also demonstrated in the Deferral of PTCA Versus Performance of PTCA (DEFER) trial,[38] which prospectively randomised 325 patients referred for elective percutaneous transluminal coronary angioplasty (PTCA) of an angiographically significant *de novo* stenosis (> 50% diameter stenosis by visual assessment) in a

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Figure 3. Event-free survival rates in the DEFER trial

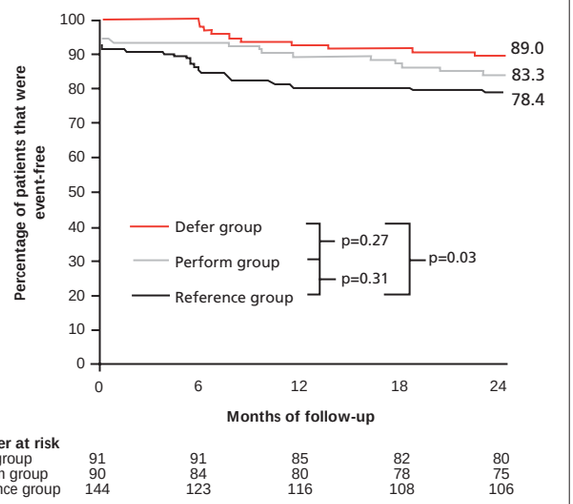
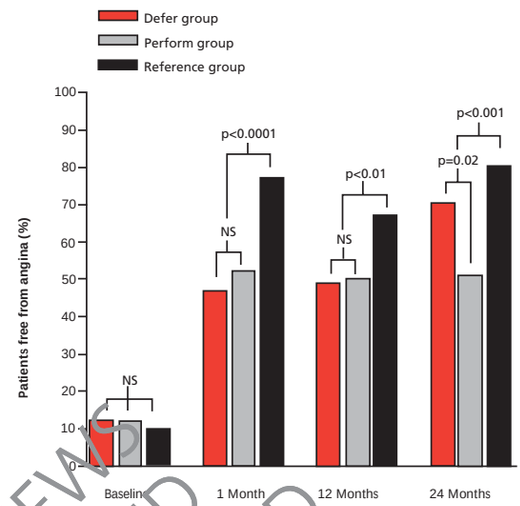


Figure 4. Rates of patients free from angina in the DEFER trial



native coronary artery with reference diameter > 2.5 mm and no prior objective evidence of ischaemia. Patients with an FFR ≤ 0.75 had PTCA, whereas those with an FFR ≥ 0.75 were randomised into a 'perform group' and a 'defer group'. The event-free survival rates were similar for all groups (figure 3) and the improvement in angina was no different (figure 4). Of particular note was the fact that not one patient with an FFR of > 0.75 who underwent angioplasty derived any symptomatic benefit. This trial demonstrates the utility of FFR in predicting those patients who will and will not benefit from PTCA.

It is generally accepted that coronary artery bypass surgery (CABG) prolongs life for patients with significant left main coronary artery (LMCA) stenosis.[39-41] However, in patients with intermediate LMCA stenosis the decision on whether to perform CABG is more difficult as early surgery could lead to the inappropriate use of available graft material and premature occlusion of either native vessel or graft.[42] Unfortunately, non-invasive tests for reversible ischaemia often fail to differentiate between ischaemia arising from LMCA stenosis and that from elsewhere in the coronaries.[43,44] It has been shown that deferring CABG in patients with LMCA stenosis and FFR > 0.75 was accompanied by excellent survival, freedom from events at up to five years of follow-up and similar outcomes compared to those patients in whom CABG was performed based on FFR < 0.75.[45]

Multivessel coronary artery disease can be of intermediate severity in all those vessels involved or can show a mixture of intermediate and severe stenoses. Whatever the case, identifying the culprit vessel(s) responsible for patients' symptoms can help to determine whether a patient should have a PCI or CABG. Non-invasive imaging is limited in its ability to localise ischaemic zones accurately in multivessel CAD.[46] In contrast, FFR, by determining which vessels have functionally significant or insignificant lesions, serves as an invaluable guide in determining which revascularisation procedure is appropriate for a particular patient and also whether revascularisation is necessary at all.[47,48]

An abstract by Botner *et al*. looked at the value of FFR in guiding revascularisation in multivessel CAD. One hundred patients referred for CABG had FFR assessment of 272 lesions. Patients with more than three vessels with haemodynamically significant zones on FFR, of which at least two were considered supplied by type C lesions, underwent CABG. Patients with fewer than two vessels showing haemodynamically significant FFR and with one or no type C lesion had targeted PCI. Follow-up at two years showed similar rates of event-free survival (74% *vs.* 72%, p=NS) and freedom from angina (76% *vs.* 77%, p=NS) in the two groups.

Long-term outcome after PCI can be predicted with the use of FFR whereas visual and quantitative coronary angiography (QCA) are poor predictors of clinical outcome before and after a PCI.[49,50] Immediately after PCI a value of FFR ≥ 0.75 will be sufficient to prevent reversible ischaemia and can be called an initially successful functional result. In the days, weeks and months following a PCI considerable changes in stenosis morphology may occur, with some loss of initial luminal gain,[51-53] hence the value of FFR immediately after a PCI should be significantly higher than 0.75 to ensure successful long-term outcome.

Pijls *et al*. investigated the relationship between optimum physiological stent implantation as assessed by post-stent FFR and outcome at six months.[54] In 750 patients who had undergone stent implantation with good angiographic results, the post-stent FFR was related to major adverse cardiac events (including target vessel revascularisation) at six months. In 36% of the patients FFR was > 0.95 (event rate 4.9%); 32% of patients had FFR 0.90–0.95 (event rate 6.2%); 32% of patients had FFR < 0.90 (event rate 20.3%); 6% of the patients had FFR < 0.80 and their event rate was 29.5% (p<0.001). The investi-

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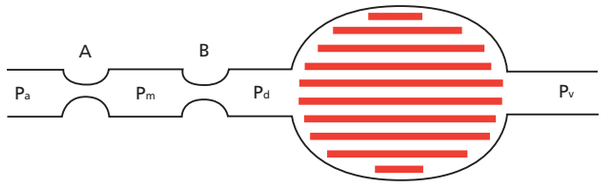
Figure 5. Calculation of predicted fractional flow reserve (FFR_{pred}) in distal stenoses



A = stenosis A
B = stenosis B
P_a = mean aortic pressure
P_d = mean hyperaemic distal coronary pressure
P_v = mean hyperaemic central venous pressure. It is not zero but can be treated as such as a mathematical approximation
P_m = mean hyperaemic pressure between stenoses
P_w = coronary wedge pressure

$$FFR(A)_{pred} = \frac{P_d - (P_m / P_a)\, P_w}{(P_a - P_m) + (P_d - P_w)}$$

$$FFR(B)_{pred} = 1 - \frac{(P_a - P_w)(P_m - P_d)}{P_a(P_m - P_w)}$$

gators concluded that FFR post stenting is a strong independent predictor of outcome at six months.

A large multicentre prospective trial in patients with current generation stents found that the best FFR cut off to correlate with IVUS was 0.96. The study concluded that FFR can be used to evaluate optimal stent deployment in the absence of diffuse CAD and serial stenosis.[55]

Slow coronary flow (SCF) is a phenomenon characterised by delayed opacification of coronary arteries in the absence of epicardial occlusive disease. It has been demonstrated that FFR in patients with SCF is significantly reduced. Upon IVUS investigation the common finding was longitudinally extended massive calcification throughout the epicardial arteries, with increased intimal thickness consistent with diffuse atherosclerotic disease.[56,57]

Multiple lesions

In multiple sequential stenoses, we have a situation where the haemodynamic significance of a stenosis is influenced by the presence of the other(s), thus confounding the calculation of FFR for each stenosis. Consequently, for stenoses in series, FFR determined by the simple ratio P_d / P_a for a single stenosis does not predict the extent to which a proximal lesion will influence myocardial flow after complete relief of the distal stenosis, and *vice versa*. The predicted FFR (FFR_{pred}) for each stenosis in serial stenoses is instead derived from a formula which also takes into account the pressure between stenoses (P_m) and coronary wedge pressure (P_w) (see figure 5). This FFR_{pred} bears a close relation to the true FFR (FFR_{true}) that will have been obtained assuming the other stenoses were physically absent, and this formula has been validated in human and animal studies.[58,59] This approach facilitates the selection of lesions for PCI and assesses the functional result of each intervention, thereby minimising unnecessary additional procedures on haemodynamically insignificant lesions with increased risk of complications but without patient benefit.

Use of this formula implies that P_w must be measured, not estimated, and it does not take into account the effect of a large side branch between stenoses, which may modify the haemodynamic influence of one stenosis on the other. Branches between serial stenoses may inhibit maximal hyperaemia because of 'branch steal' and can increase the gradient across the proximal stenosis by increasing flow while reducing the distal lesion gradient because of 'steal' by the branch. Dilating the distal stenosis may not alter the hyperaemic gradient across the proximal stenosis; dilating the proximal stenosis may subsequently increase the gradient across the distal lesion. A similar situation exists for bifurcation lesions.

Recently, the reliability of applying FFR measurement in patients with previous myocardial infarction (MI) has attracted the attention of several researchers. Claeys *et al.*[60] provide data that FFR is minimally affected (5%) in patients with severely impaired microvascular function and may still be applied to patients with recent MI. De Bruyne and colleagues have demonstrated that FFR assessment criteria are also valid in detecting reversible ischaemia in patients at least six days after an MI.[61] Even though there is a grey area of FFR measurements between 0.75–0.80, an FFR value of 0.75 is still applicable and reliable for distinguishing patients with positive from patients with negative myocardial scintigraphy, with a sensitivity of 82% and specificity of 87% in the case of prior MI. An FFR of < 0.75 invariably indicates inducible ischaemia; FFR > 0.80 excludes ischaemia in about 90% of cases, making the grey zone very limited. This is important for clinical decision-making in an individual patient.[62] Another study conducted by Usui *et al.*,[63] comparing FFR and thallium-201 myocardial imaging, also showed that pressure-derived FFR is reliable in assessing coronary artery stenosis in patients with previous MI, with a sensitivity of 79% and specificity of 79%.

In the case of prior MI, two concerns remain: (1) the mass of viable myocardium is smaller; and (2) impairment of resistance vessels might blunt pharmacologically-induced maximal hyperaemia. However, as both the decrease of viable myocardium and impairment of coronary resistance vessels are matched in the infarcted area, FFR is still a reliable indicator for predicting inducible ischaemia, even if the angiographic image of a stenosis might be more severe. In the acute phase of MI, FFR measurement should not be used due to serious microvascular impairment, and treatment should be guided by the clinical symptoms and ECG. Pressure measurements are useful only after the artery has stabilised.

The ideal means of assessing patients with microvascular disease is a combination of coronary flow reserve (CFR) and FFR measurements. A low FFR and CFR indicate significant epicardial disease, whereas a high FFR and low CFR indicate signifi-

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Table 3. Practicalities of FFR measurement

General
Interventional procedure – requires heparin
Nitrates should be used in all cases
Beware of rough handling of wire. Instances of tip detachment have rarely occurred
Company doesn't recommend radial route because of risk of tip fracture
The wire is much easier to use when disconnected from the hub
Don't use Hexabrix with papaverine – the two are incompatible
Vasodilator
Adenosine
Intracoronary bolus (42 micrograms) convenient but very short-lived[66]
Intravenous infusion (140 micrograms/kg/minute) needs to be set up but can be expensive[67]
Preferred agent in America
Contrast medium
Easy and convenient but doesn't work
Papaverine
Recommended dose is now 20 mg for left system and 16 mg for the right
Given by intracoronary route
Cheap
Good duration of action
Can get ventricular tachycardia/torsade due to QT prolongation

cant microvascular disease. Recent technological advances have made available a new pressure wire equipped with both pressure and temperature sensors to measure both FFR and CFR.

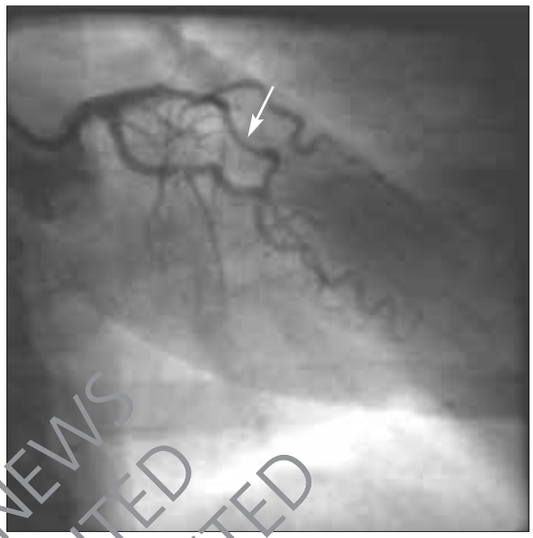
The 0.75 cut-off value of FFR has been assessed in patients with stable angina. For patients with unstable angina, it is commonly believed that maximal hyperaemic flow may be lower than in patients with stable angina. Consequently, the 0.75 cut-off value of FFR might not be valid in these patients and the appropriate value needs to be determined. However, a recent study by Leesar *et al*.[64] of patients with unstable angina or non-ST-segment elevation myocardial infarction (NSTEMI) demonstrated that the FFR assessment criteria are also valid in this patient group. A decision-making strategy based on the 0.75 cut-off is superior to a more conservative approach based on myocardial perfusion scintigraphy.

The FFR, in addition to the above, is also a cost-effective tool. In patients with intermediate coronary lesions, measuring FFR to guide the decision to perform PCI may lead to significant cost savings compared to deferring PCI to perform a nuclear stress study or with simply stenting lesions in all patients.[65]

Applications

The principal applications for the pressure wire in the catheter laboratory are: the differentiation of functionally significant from insignificant lesions in single vessel, multivessel and left main coronary disease, differentiating focal from diffuse disease to determine whether spot-stenting of a particular area is likely to be of benefit, and assessing the results of intervention. A number of practical examples arising from the authors' recent practice are given below. Some of the practicalities associated with measurement of FFR are given in table 3.

Figure 6. Coronary angiogram, showing a lesion in the left anterior descending (LAD) vessel



Determination of functionally significant lesions

Example 1

A 67-year-old woman was referred for PCI to a tight lesion of her AV circumflex going into a large obtuse marginal. It was also mentioned that she had diffuse disease of her left arterior descending (LAD), which was thought to be insignificant.

At PCI there was a suspicious-looking mid-LAD lesion (figure 6). In order to be sure, a pressure wire assessment of her mid-LAD lesion was done. This showed her FFR to be 0.92 (well above the cut-off of 0.75 for functional significance), meaning it was indeed an insignificant lesion. Her AV circumflex was then direct stented, with a good angiographic result.

Example 2

A 57-year-old man was referred for assessment of his only remaining vessel, his LAD (figure 7), with a view to doing a PCI on a mid-vessel lesion. The tortuosity of the LAD at the first diagonal made it difficult to image with any degree of confidence; there was also a region of haze and apparent discontinuity of the dye in the area of the tortuosity. A pressure wire assessment of the lesion revealed an FFR of 0.81, meaning that the lesion was not functionally significant and that his angina was most likely due to viable myocardium in the territory of the occluded vessels. PCI (a very high-risk procedure in this partic-

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Figure 7. Coronary angiogram, showing tortuosity and haziness at the first diagonal of the LAD

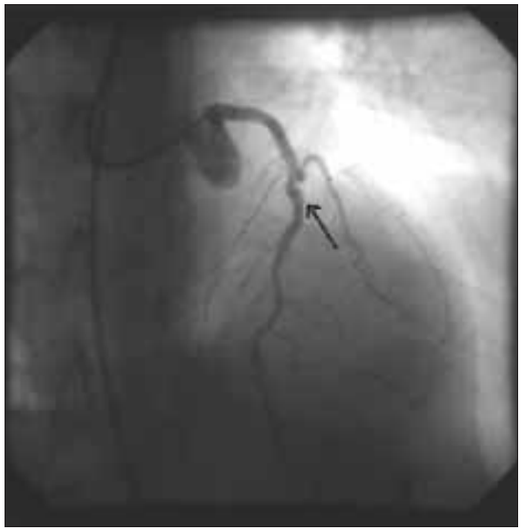


Figure 8. Coronary angiogram, showing lesions in the right coronary artery and AV circumflex

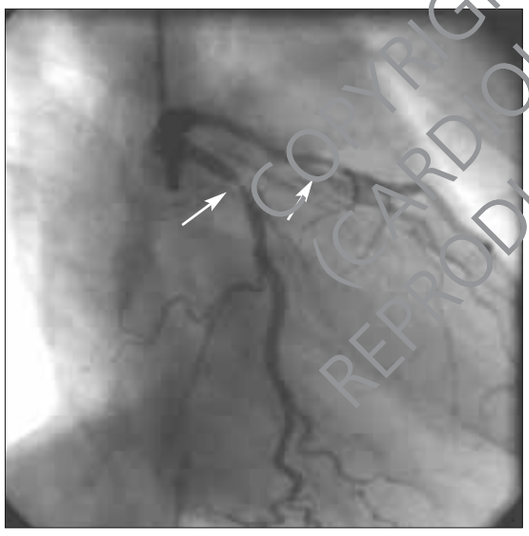


Figure 9. Angiogram (of the same patient shown in figure 8), after stenting of the RCA

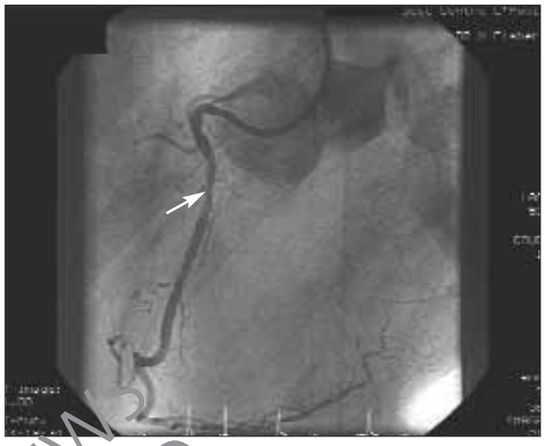
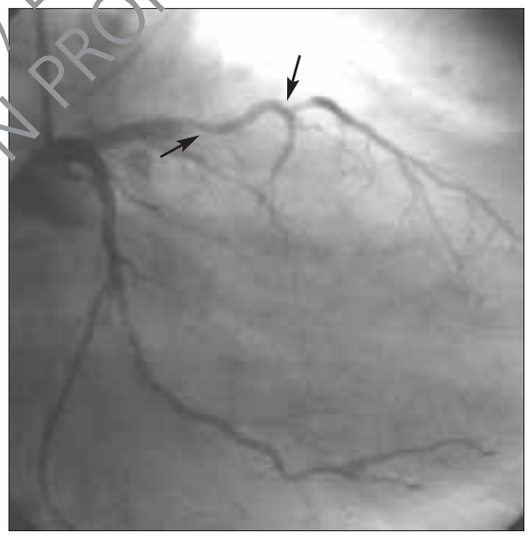


Figure 10. Coronary angiogram, showing a proximal lesion in the LAD and a distal lesion in a septal branch



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ular patient) was not done and the patient was referred to the refractory angina service.

Example 3

A 47-year-old man was admitted for PCI to his AV circumflex and right coronary artery. Pressure wire assessment of the lesion in his LAD showed an FFR of 0.88, meaning the lesion was not functionally significant and did not need a PCI since ostial disease in a small diagonal was the only significant disease in his LAD system. Direct stenting of the AV circumflex and RCA was subsequently carried out, with good angiographic result (figures 8 and 9).

Assessment of result of intervention

Example 1

A 77-year-old man was referred for PCI to his LAD. A pressure wire study was done to assess the LAD further. This was inter-

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Figure 11. Angiogram (same patient as figure 10), after direct stenting of both lesions

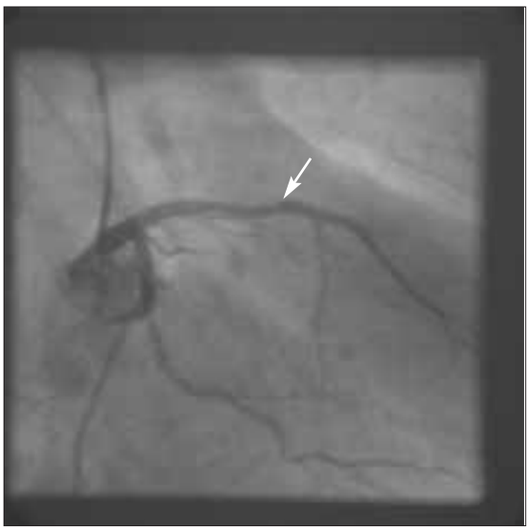


Figure 12. Angiogram (same patient as figures 10 and 11) after post dilation of both stents and the area between

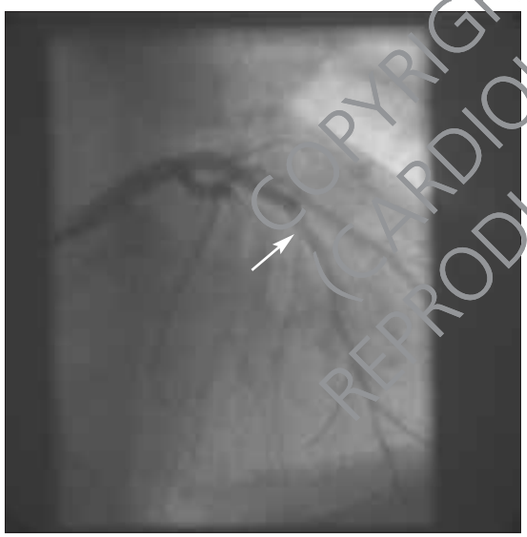


Figure 13. Coronary angiogram, showing a suspicious lesion in the proximal circumflex

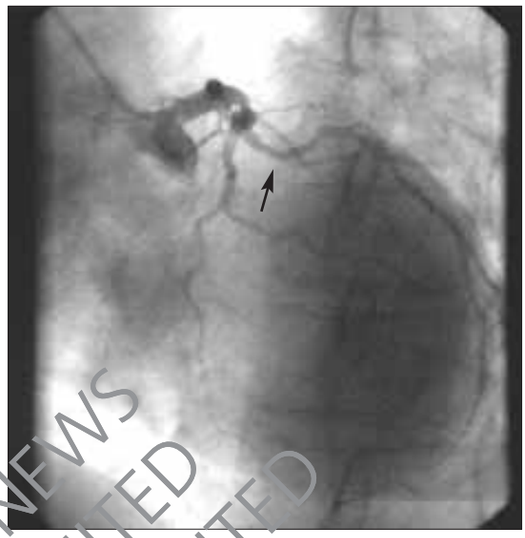
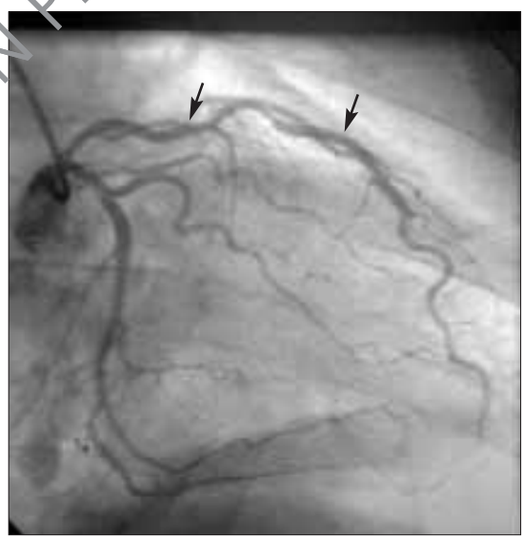


Figure 14. RAO caudal view. White arrows show rather diffuse areas of disease which do not appear significant



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esting in that it showed he had significant lesions both proximally in his LAD and distal to a large septal branch (figure 10). Both lesions were direct stented and the result checked with the pressure wire (figure 11). Despite the fact that the angiographic result looked good, the FFR was unsatisfactory at 0.84. This necessitated post dilatation of both stents and the area between (which looked slightly nipped), producing a more satisfactory FFR of 0.94 (figure 12).

Example 2

A 67-year-old woman with previous PCI to the right PDA presented with atypical chest pain, negative troponin T and no new ECG changes.

Coronary pressure wire assessment of the previous PCI and a suspicious lesion at the origin of the left ventricular wall branch showed no functionally significant lesion.

A suspicious lesion in the proximal circumflex (figure 13)

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Figure 15. LAO caudal view showing step-down after the LMS and again diffuse areas of rather non-significant disease

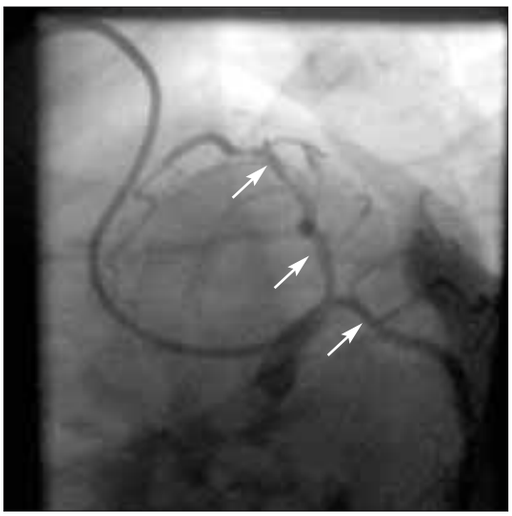

Figure 16. Pullback from the distal LAD, showing the very gradual increase in the FFR. This suggested that there was no individual focal lesion but rather a very diffusely diseased LAD, which overall produced significant stenosis

yielded an FFR of 0.74 (borderline); a stent was inserted in this lesion and then post dilated, with excellent angiographic result and an FFR of 0.98.

Application in diffusely diseased vessels

Example

A 63-year-old man presented with a history of cardiac-sounding chest pain. His MIBI scan revealed an anterior perfusion defect while the angiogram showed he had a step-down in vessel size in the LAD after the LMS and there was a more focal lesion in mid-vessel (figures 14 and 15) but overall the lesions didn't look significant.

Nevertheless, FFR in the LAD was 0.74 and the pullback (figure 16) showed there was no focal culprit lesion but a gradual normalisation of the FFR. This indicates that the whole of the proximal vessel was diffusely diseased (note it does look small – smaller than the 6F guide, which is unusual for an LAD in a man) and that stenting one segment of the vessel was unlikely to benefit the patient. In view of this and the ostial disease in the diagonal he was referred for surgery.

Key messages

- Fractional flow reserve (FFR) is an invaluable tool for assessing the functional significance of coronary artery lesions
- It is particularly helpful for stenoses of intermediate severity
- It is able to differentiate focal from diffuse disease, and to assess the results of intervention

Conflict of interest

None declared.

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