

# **Physiologic assessment of Nonculprit Vessel During Multivessel PCI**

**A considerable body of data supports the use of FFR to guide percutaneous coronary intervention (PCI). In the randomized, controlled FAME trial, investigators compared FFR-guided PCI to angiography-guided PCI in patients**

with multivessel coronary artery disease who were undergoing PCI and stenting. **(Ref:1)** There was a significant reduction in the primary endpoint of death, nonfatal myocardial infarction (MI), and repeat revascularization at 1 year with FFR-guided PCI.

In FAME 2, which enrolled patients with stable coronary artery disease (CAD), there was a reduction in spontaneous

MI at 5 years after FFR-guided PCI compared with medical therapy alone. **(Ref:2)**

Recently, Volz et al. analyzed a national registry that includes procedural data on all patients in Sweden treated with PCI.**(Ref:3)** Of 23,860 patients with stable CAD who underwent PCI, the 3,367 patients who received FFR-guided PCI had a significant 19% reduction in

the primary endpoint of all-cause mortality at a median follow-up of  $\approx 5$  years compared with the patients who underwent PCI guided by angiography alone.

This adds to the already considerable evidence demonstrating the safety of deferring revascularization of coronary lesions with FFR values above 0.75 to 0.80. Medical treatment of these lesions

avoids the risks of PCI while leading to equally low (if not lower) rates of major adverse cardiac events (MACE) during long-term follow-up.

Conversely, PCI for stenoses with FFR values  $\leq 0.80$  improves outcomes compared with medical therapy or PCI guided by angiography alone. (Most of the data are derived from patients with SIHD.)

**Morton Kern, MD (VA Long Beach and University of California, Irvine), said the evidence suggests clinicians should treat lesions causing ischemia and leave the others alone, adding that this is true for single-vessel or multivessel disease as well as for left main disease. With good stress testing and perfusion imaging zones, or other high-quality stress tests, it is possible to get enough information**

to move forward. But this may not be enough for certain lesions and vessels in different territories. It is in these situations that FFR is better.

Noninvasive FFR derived from coronary computed tomography (CT) – FFRCT – is not widely used today because of cost and work-flow issues, but it correlates well with invasive FFR (FFR<sub>cath</sub>) – usually  $>0.85$  – and is an

excellent tool to use before a patient comes to the cath lab to sort out who needs catheterization. Besides correlating well with invasive FFR, FFRCT provides superior information than CT angiography alone does, and it is a better predictor of outcomes.

## **ACS and FFR**

Patients with acute coronary syndrome, particularly STEMI or NSTEMI, often



require PCI, but what about the use of a diagnostic guidewire to assess FFR?

In STEMI, physiologic assessment is not needed for the culprit vessel, which is usually obvious: it is associated with the electrocardiogram, and it has its wall motion and thrombus. Plus, there are variable degrees of transient microvascular dysfunction that can occur in the culprit vessel of a patient

with STEMI. This can affect the accuracy of FFR assessment. The derivation of FFR assumes that microvascular resistance is minimized and stable, which is often not the case in recently infarcted myocardium. All of which goes to explain why FFR measurement is not recommended in the culprit vessel in acute STEMI.

In patients with NSTEMI, FFR can be used to assess nonculprit vessels; again, if the FFR is  $\leq 0.80$ , the lesion is significant. If FFR is  $> 0.80$ , it is usually *not* significant. (That is less certain if it is close to an injured myocardial zone.)

The problem was underscored in a paper by Hakeem and colleagues. In patients with NSTEMI, less transient microvascular dysfunction is expected in

the culprit vessel than in patients with STEMI. Yet Hakeem et al. noted that in NSTEMI patients, the best cutoff value for FFR is  $\leq 0.84$ , implying that transient microvascular dysfunction might be falsely elevating FFR in a portion of these patients. **(Ref:4)**

The fact that FFR may have little role to play in culprit vessels does not mean it has no role in assessing nonculprit

vessels in patients with STEMI or NSTEMI. In nonculprit vessels there is little, if any, transient microvascular dysfunction, meaning FFR can help guide revascularization decisions.

Bottom line: available data suggest that FFR of clear culprit lesions of STEMI or NSTEMI should be discouraged. On the other hand, data support the role of measuring FFR and applying a cutoff

value of 0.80 in stable lesions – that is, in stenoses in stable patients or in nonculprit stenoses in patients presenting with STEMI or NSTEMI. Then the clinical decision is whether to revascularize nonculprit vessels during the initial procedure or to defer revascularization to a later date (a staged procedure).

**Take-home Messages:**

- Current data support the role of measuring fractional flow reserve (FFR) and applying a cutoff value of 0.80 in stable lesions – that is, in patients with stable ischemic heart disease (SIHD). In this setting, it is appropriate to identify lesions that need treating and defer the others.
- Based on current evidence, FFR assessment is not appropriate for

the culprit lesion in patients with ST-segment elevation myocardial infarction (STEMI) or non-STEMI (NSTEMI).

- However, FFR can be useful for assessing *nonculprit* vessels in both settings. The clinical decision becomes whether to revascularize those nonculprit vessels during the



initial procedure or defer  
revascularization to a later date.

## **References:**

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