

Letter by Michiels et al Regarding Article, “Diagnostic Accuracy of Combined Intracoronary Pressure and Flow Velocity Information During Baseline Conditions: Adenosine-Free Assessment of Functional Coronary Lesion Severity”

To the Editor:

We read with interest the article by van de Hoef and colleagues about the value of the coronary stenosis pressure drop-flow velocity ratio ($\Delta P/V$) in the evaluation of coronary stenosis.¹ We congratulate the authors with this meticulous and well-written investigational report; however, we have a number of comments.

Again (as with iFR in the recently published ADVISE study),² the authors claim in their subtitle that they demonstrate the diagnostic performance of an adenosine-free assessment of functional coronary lesion severity. In recent literature, there seems to be an unjustified quest for proving that on one hand (very) high doses of intracoronary adenosine are necessary to obtain maximal hyperemia and on the other hand that it would be better not to give adenosine at all.

We believe that this presumed need for high intracoronary adenosine doses can be brought back to the fact that administration of intracoronary adenosine might be inaccurate, and prone to error compared to intravenous administration. Moreover, we recently published a consensus document about fractional flow reserve (FFR) measurement, where we stressed the importance of intravenous administration as the gold standard.³ The unusually high rate of diagnostic inaccuracy for FFR encountered in the current study could be because adenosine was only administered intracoronary and at a very low dose (lower than in any other FFR-related trial with clinical end points).

Regarding the issue of not giving adenosine at all, one forgets the advantages of hyperemic versus baseline evaluation of a coronary stenosis. First, clinical ischemia is determined mainly by maximum achievable blood flow (often a coronary stenosis at rest does not induce a significant pressure drop whereas in maximum hyperemia it does). Second, since coronary flow is related to the supplied myocardial mass, hyperemic flow and thus hyperemic gradient will decrease after myocardial infarction. As a consequence, FFR will increase. As such, induction of hyperemia is an indirect indicator for viability, a prerequisite for revascularization. When measuring baseline pressure drops across a stenosis, this aspect is not evaluated. Third, since stenosis resistance is flow dependent, it should be determined at maximal hyperemia.⁴

In short, comparing a baseline stenosis resistance to a suboptimal performed FFR is illogical.

There is also an important technical and economical drawback: the method described here involves the use of 2 separate wires (pressure wire and Doppler wire), which is inconvenient and expensive. This problem could be overcome by using only a pressure wire and thermodilution to measure flow instead of the Doppler-derived flow velocity.⁵ Furthermore, with this same simple equipment we

can measure the index of microcirculatory resistance to get a precise idea of microcirculatory functioning (often explaining discrepancy between coronary flow reserve and FFR). Moreover, the use of a Doppler wire to assess flow velocity is cumbersome: poor signal acquisition is present in 10% to 15% of patients often because of the tortuosity of the coronary artery, and there is a large variability in measured values because of differing hemodynamic conditions.

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Disclosures

None.

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