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Editorial Comment

Flow Wire Fouls Pressure Wire: Red Card for Both

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Ruiz-Salmerón et al. exhibit a clear case of wishful writing when they start their interesting article with the sentence: "functional evaluation of stenosis severity with the pressure wire is becoming a mainstay in interventional therapy, pushing to oblivion the occulostenotic reflex." On the other hand, their caveat about a technical pitfall does not appear that relevant, after all.

In three asymptomatic patients with a follow-up angiogram performed after successful stenting for acute myocardial infarction, the introduction of a flow wire in addition to the pressure wire reduced the fractional flow reserve (FFR) from normal (> 0.75) to abnormal. In two cases, it shifted from 0.77 to 0.70; in one, from 0.76 to 0.71. The Doppler-assessed coronary flow reserve stayed normal in two and became also abnormal in one. The authors attributed this roughly 10% downward shift of the fractional flow reserve to the second wire and ignored it, i.e., they did not redilate the borderline restenoses in these three stents. The patients did well afterward, a fact the authors used to prove their point.

A 3.0 mm stent has a cross section of 7.1 mm^2. The stents were restenosed by 53% to 55%. The measured minimal luminal diameters of 1.2 to 1.3 mm indicate slightly more severe restenoses. The cross-section of a lumen with a diameter of 1.2 mm is 1.1 mm^2. The cross-section of a 0.014″ guidewire is 0.1 mm^2. Double that for two wires and you have to deduct 0.2 mm^2 from 1.1 mm^2. Thus, the two wires are reducing the cross-section from 1.1 mm^2 to 0.9 mm^2 or reverted back to minimal luminal diameters from 1.2 mm to 1.1 mm, or in percent diameter stenosis in a 3.0 mm stent from a 60% to a 63% stenosis. It is hard to believe that this should be clinically relevant and rule the decision to dilate or not to dilate.

In the situation described, the fact whether this vessel remains normally patent (or patent at all) could only play a role in the future, in case another vessel became dependent on collaterals from this vessel. The lesion itself had lost its importance during the initial infarction.

A completely different story is encountered when such measures are used to assess the necessity to dilate lesions that have not been occluded before as it was done in a study cited by the authors [1]. In that study, patients were either subjected to angioplasty or deferred based solely on the FFR of their lesions. The quantitatively assessed degree of stenosis was $< 50\%$ in half the cases, although the study included only patients with a visual estimate of $> 50\%$. Even in the patients with an abnormal FFR, the average stenosis hovered around 55%, and only 10% of the patients had a stenosis $> 70\%$. How to explain to the referring physician angioplasty of a 30% stenosis because the FFR was abnormal?

The higher freedom of angina at 2 years in deferred patients compared with patients subjected to angioplasty was produced by 20% of patients losing their angina during the second year in the deferred group compared with only about 2% in the treated group. The authors appeared as puzzled about this as I am. Otherwise, there was neither benefit nor disadvantage at 2 years of dilating hemodynamically insignificant stenoses, i.e., the patients having had their operation were as well off as those still waiting for it and this includes all complications of the operation. Which group would you want to be in?

Thoughts on plaque sealing of mild lesions by balloon angioplasty to the end of reducing the infarct potential [2] were cited but not fully understood. The fact that most infarctions originate from nonsignificant lesions does not imply that a nonsignificant lesion has a high infarct potential. Its individual infarct hazard is small but there are many mild lesions. Their aggregate infarct potential engenders that generally a mild lesion causes the next infarction rather than a significant lesion despite the higher individual infarct hazard of the latter. The plaque sealing concept pertains to mild lesions and significant lesions alike and does not pretend that mild lesions need angioplasty more than severe lesions. It merely suggests that they might benefit from angioplasty, too [3]. While normalization of blood flow provides an indication for angioplasty in significant lesions, plaque sealing would be the only indication in mild lesions. Stenting puts a question mark to the plaque-sealing concept. After balloon angioplasty, infarctions from the dilated lesions are extremely rare beyond hospital discharge. This is not entirely true for stented lesions.

One has to invest about two pressure wires plus the respective time and risk to defer one angioplasty accord-

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ing to the DEFER study [1]. The stress is on defer. Hence, economic arguments to adhere to such a policy are moot and, by the way, not claimed by the authors.

Reducing the risk of infarction or death is less discussed but more important than getting rid of angina. Admittedly, the DEFER study does not prove this effect at 2 years, but it does not disprove it. The question remains whether relying exclusively on hemodynamic measures such as FFR does not mean barking up the wrong tree when taking care of patients with coronary artery disease.

The study by Ruiz-Salmerón et al. bites its own tail. It suggests that because of some pitfalls of pressure measurements, an additional flow wire is necessary to close in on the truth. Then it concludes that this second wire makes the measurements even more unreliable. All this is a consequence of the imprecision of the measurements rather than the presence of the second wire [4].

In their three cases, the authors have spent more time and money on hemodynamic assessments of mild in-stent restenoses than if they had redilated them. Redilatation of a mild in-stent restenoses harbors practically no risk except for the remote possibility of a more important restenosis later on. The impeccable follow-up which the authors gracefully acknowledge, albeit with some anxiety, would have been a certainty had they dilated the lesions.

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