PCR webinars

Coronary physiology in a modern catheter laboratory

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Question 1: What will happen to FFR if we park the wire in small branch like septals in LAD?

If the pressure wire lays with its sensor in very small branch, often you see artifacts like spikes or a very high pressure. In that case, the wire should be pullback a little bit until the sensor is in the main artery again.

Generally, this will only occur in arteries with a diameter less than 1 mm.

Question 2: Which one is preferred choice among Intracoronary vs Intravenous Adenosine? Is there any significant difference?

For purpose of creating maximum hyperemia, there is no difference. Both drugs (if administered in an adequate doses) are equivalent in creating maximum hyperemia. Which one you choose will also depend on what you want: if you have a smooth coronary artery with one single stenosis, intracoronary adenosine is perfect. If you have an artery with diffuse disease or several focal lesions, you like to make a hyperemic pullback recording to investigated what part of the coronary artery contributes most to the disease. In such case intravenous adenosine with steady-state hyperemia is mandatory.

Question 3: Do you think the temperature of intracoronary injected saline can influence the FFR/iFR results?

No. Theoretically there will be a minimal influence but in practice this is neglectable.

Question 4: How often do you encounter microvascular disease?

Depending on the literature, microvascular disease plays a role in 20-50% of all patients presenting with angina pectoris. You should keep in mind that microvascular disease is most likely a very heterogonous collection of abnormalities (ranging from purely morphologic microvascular disease to functional microvascular disease) and that in different studies with different inclusion criteria, the prevalence of microvascular disease will, as a matter of fact, be different.

New methodology, enabling measurement of absolute blood flow and absolute microvascular resistance and the new parameter MRR (Microvascular Resistance Reserve), will hopefully bring new insights in the pathophysiology and severity of microvascular disease.

Question 5: What parameter do you use to know you have the maximum hyperemia?

If you use the correct dosage of adenosine, you can trust that you have maximum hyperemia in 99% of all patients. Might you have any doubt, you can either increase the dosage of IC adenosine and see what happens. And in case of IV adenosine, you can easily give additional IC adenosine on top of the IV to see what happens. The ultimate proof of the presence of maximum hyperemia is given by the fact that you see no further increase of hyperemia (i.e. further decrease of distal pressure) by such additional drug administration.

Question 6: If you are interested in a LM lesion and LCX and LAD are normal, where would you park your pressure wire sensor?

Personally, I measure then in both of the main branches (LCX and LAD). In case of an ostial left main or mid left main lesion, one of the two big branches would be significant. But in particular in case of bifurcation lesions in the left main, sometimes the hemodynamic significance for the LAD can be different from the LCX. And anyway, it takes little time and increases your insights in the nature of the disease.

Question 7: How do you choose between Intravenous or Intracoronary?

See the answer to the second question above.

Question 8: In case of RCA-CTO with good collaterals from LAD, which is mildly and diffusely diseased (no PCI target) and low …?

I guess because the question is incompletely presented to me: probably you mean if that there are good collaterals from an artery and that artery itself has a moderate stenosis, the perfusion territory of that artery is larger than it should be originally and the FFR of that artery might be lower. In practice, this effect is limited. In case that your LAD stenosis has an FFR of 0.75 – 0.080, you can consider first to open the CTO of the RCA and repeat the measurement of FFR in the LAD.

Question 9: Why is it that Dipyridamole is not used to assess FFR?

Dipyridamole creates maximum hyperemia if administered in a sufficiently high dosage (mostly higher than usually used in MIBI SPECT). But if you use that dosage, the side effects are considerable. Hypotension will develop and that limit its use.

Question 10: Can you get away with IA nitro instead of IC nitro?

I am not aware what IA nitro means. Do you mean sublingual nitro? Normally, it works. But I would rely upon intracoronary nitro. More general, I personally believe that in every coronary angiography and in every intracoronary diagnostic manipulation, it is mandatory to administer 200-300 µg of nitroglycerine in the coronary artery before doing the assessment. I do not need to tell how a coronary artery can change by IC nitro.

Question 11: What should be the further strategy for patients with discordance between hyperemic and non-hyperemic indices?

Just trust FFR (hyperemic index). FFR has been validated in thousands of studies and related to outcome, whereas the literature for NHPR is much more limited. And believe in a general principle in science in general: " If you like to know how strong something is, test it under stress conditions".

Question 12: Does pharmacological hyperemia substantially differ from stress-induced hyperemia?

Yes. Although in most patients, there is a very good correlation, in patients for example who are proned to coronary spasm, adrenergic hyperemia by exercise can induce spasm where pharmalogical vasodilatory stimuli do not. Although in general, pharmalogical hyperemia in the catheterization laboratory is sufficient (in fact always induces maximum hyperemia) it prefends stress-induced coronary spasm which maybe play a role in a few patients.

Question 13: The only issue is the false negative FFR in 15-20% of patients with incomplete vasodilation.

I do not get exactly the meaning of this question. As a matter of fact, if hyperemia is not maximum, the chance for false negative FFR increases. And if there is no hyperemia at all (such as is the case for the non-hyperemic pressure ratios like iFR, DPR, FFR), indeed these values give false negative outcome in 15-20% of patients.

More in general: in long moderate lesions in moderately sized coronary arteries in older persons, there is often a gradient which only moderately increases with hyperemia and false negative iFR is rare. But in younger patients with short proximal lesions in large coronary arteries, false negative iFR is common. The explanation is that in the latter kind of lesions, the quadratic component in the Poiseuille equation is most pre-dominant.

Question 14: In case of normal FFR and discordant IMR and CFR (e.g. high IMR and normal CFR), how do you interpret the disease?

In the first place, the normal FFR tells you that there is no epicardial problem and that PCI in itself doesn't make sense. Second, it is difficult to say what "normal" CFR is.

"Normal" CFR can be 2.0 in an 80-year old man and can be 6.5 in an 30-year old athlete. IMR has been better validated and in case of discordancy between IMR and CMR I would believe the IMR.

Question 15: Why guideline use the cut point of 90% luminal stenosis for significant lesion?

In the FAME study, it has been clearly shown that almost all lesions with a visual stenosis on the angiogram of >90%, were also functionally significant. There were very few exceptions. In the group of stenosis which were 70-90% by visual estimation, quite a number of these lesions turned out to be not functionally significant and in the group of 50-70% that was even more often the case. This substudy of the FAME study was published with Pim A.L. Tonino et al in the Journal of the American College of Cardiology in 2010 ("Angiographic versus functional severity of coronary artery stenoses in the FAME study"). This study made clear that it is reasonable to measure FFR in all lesions with a visual stenosis between 30-90%.

Question 16: If we see slow flow phenomenon is it related with microvascular disturbance?

If there is a normal epicardial artery, often slow flow is related to microvascular disturbance. There are some exceptions. For example, if you have a very large wide coronary artery in a patient with a normal microcirculation, still you can see rather slow flow in that artery simply because of the fact that it is such a large artery.

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