

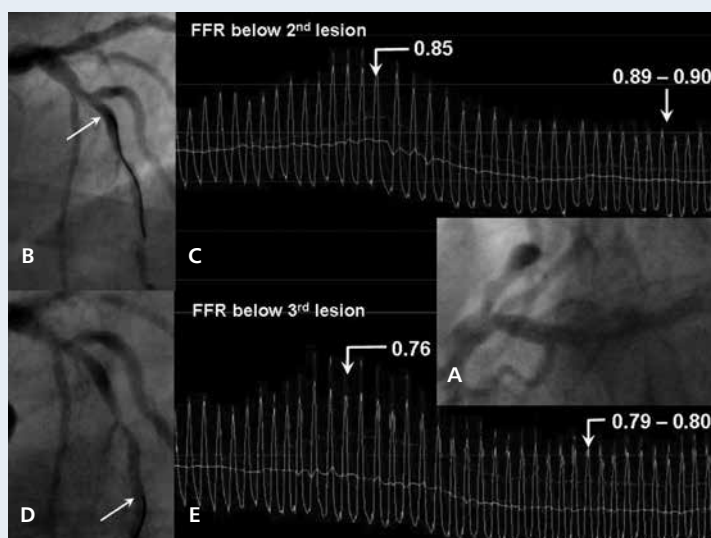
# Pitfalls of fractional flow reserve

## Pułapki cząstkowej rezerwy przepływu

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Fractional flow reserve (FFR) is obtained by the ratio of the hyperaemic distal coronary artery pressure to the aortic pressure. The lowest FFR value during steady-state hyperaemia is the base for the consideration of treatment strategy. Continuous intravenous infusion is considered to be the gold standard of adenosine administration to achieve maximal hyperaemia. We present a patient with an atypical FFR recording. Such an FFR tracing may cause confusion in interpretation. A 71-year-old male patient with stable coronary artery disease (CCS I) and metabolic syndrome was admitted to angiography due to an electrocardiographically positive exercise treadmill test. Borderline tandem lesions in proximal left anterior descending coronary artery (LAD) and 50% stenosis in left circumflex coronary artery were visualised (Figs. 1A, B, D). Thus, FFR in LAD was performed. Continuous adenosine infusion (140  $\mu\text{g}/\text{min}/\text{kg}$ ) via the forearm vein was administered. The FFR value below the third LAD lesion dropped at the beginning of hyperaemia to 0.76, followed by a rise to 0.79–0.80 during steady-state hyperaemia (Fig. 1E). The latter value did not fall despite an increase of adenosine infusion rate. Next, the FFR value below the second LAD lesion dropped at the beginning of hyperaemia to 0.85. Then similarly to before, an FFR increase to 0.89–0.90 was observed during steady-state hyperaemia (Fig. 1C). The differences between the initial, lowest FFR value and subsequent increase in these two recordings were mainly caused by an increase of aortic pressure without a simultaneous increase of distal pressure at the beginning of hyperaemia. Based on the FFR results, the patient was scheduled for further conservative treatment. Adenosine administration produces relaxation of vascular smooth muscle, resulting in reduction of blood pressure. However, Biaggioni et al. (Circ Res, 1987; 61: 779–786) reported that intravenous bolus injections of adenosine increased initially systolic and diastolic pressures, followed by its subsequent reduction. Further, Cox et al. (J Clin Invest, 1989; 84: 592–596) suggested that only intracoronary adenosine administration elicited a reflex leading to a pressure increase, in contrast to practically no pressure change after adenosine infusion into the right atrium. Interestingly, the initial pressure increase in our patient was observed after intravenous drug administration. The striking observation is the lack of simultaneous increase of distal pressure, resulting in the lowest FFR value at the beginning of adenosine infusion, and not during steady-state hyperaemia. This pattern of FFR tracing was observed twice, at different points of FFR measurement. Such an FFR pattern may lead to some confusion in interpretation, especially when the values are borderline. Based on recently published AFFECTS study (Circ Cardiovasc Interv, 2013; 6: 654–661) our interpretation is that this FFR value, obtained during shifting haemodynamics, does not fulfill the requirements considered in the theoretical framework of FFR (stable hyperaemia).



**Figure 1.** A. Borderline stenosis in proximal segment of LAD; B, C. FFR below the second stenosis in LAD. Arrow indicates the position of FFR sensor; D, E. FFR below the third stenosis in LAD. Arrow indicates the position of FFR sensor

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**Conflict of interest:** none declared