

Editorial

Vorticity: At the crossroads of coronary biomechanics and physiology



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At the very beginning of interventional cardiology, coronary interventions were guided by the angiographic severity (%) of stenosis. This was a reasonable first step at that time, resulting in excellent clinical results, particularly if the angiographic information was carefully integrated with a detailed clinical history and non-invasive stress testing. Nonetheless, this early phase of anatomy-guided decision making is gradually replaced by physiology-guided strategies. The assessment of stenosis severity by angiography alone is often at variance with coronary physiology [1] and the guidance of the intervention by objective physiologic parameters has consistently proved its superiority over anatomical assessment [2–6]. Current clinical practice guidelines recommend the physiological assessment of lesion severity by fractional flow reserve (FFR) whenever an objective evidence of ischemia is not available (class I recommendation, level of evidence A) [7]. Other physiology parameters, such as the instantaneous free-wave ratio (iFR), have been recently incorporated into the clinical practice [5,6].

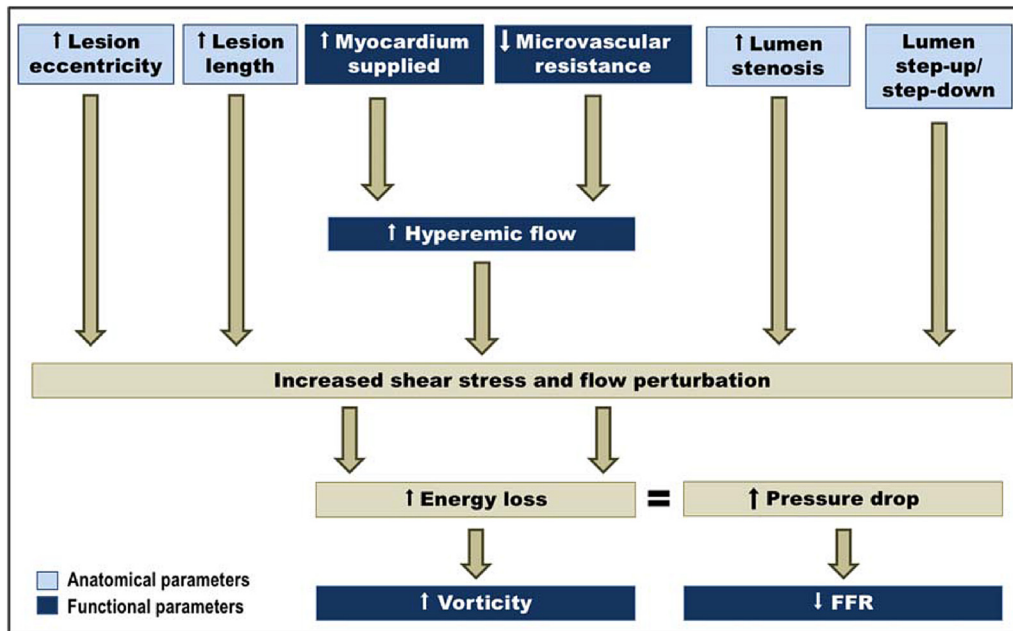
The discrepancy between morphology and physiology comes as no surprise. Fig. 1 summarises the complex interplay between anatomic and functional parameters that determine the hemodynamic relevance of a coronary stenosis. Relying on just a single anatomic parameter (e.g. % of anatomical stenosis) for decision making appears to be unrealistic. Conversely, FFR or iFR are functional parameters that stand at the point where all the anatomical and functional pathways converge (Fig. 1). Therefore, using FFR or iFR to determine the functional lesion severity and guide the coronary intervention is a strategy that integrates more information than the pure anatomical approach, reflects the clinical relevance of coronary artery disease, and potentially improves outcomes [2–6].

In this issue of *Atherosclerosis*, Chu et al. present a novel insight into coronary physiology, focusing on vorticity, a parameter which is not commonly used [8]. Vorticity is defined as the rotational vector of the flow around the longitudinal axis of the vessel. From the hemodynamics standpoint, vorticity increases in conditions that

result in loss of energy in flow, which in fluid mechanics is synonym to pressure drop. Similarly to FFR, vorticity lies at the convergence point of all the pathophysiological pathways that contribute to energy loss and pressure drop (Fig. 1). The current study found a strong inverse association between the disturbed vorticity index and FFR, as theoretically expected, suggesting that vorticity could provide similar functional information to FFR. Furthermore, vorticity can be at the crossroads between coronary anatomy and local hemodynamics, as it provides information about flow perturbations and fluid shear stresses imposed on the arterial wall (Fig. 1). Regional mapping of vorticity could represent an interesting representation of the local biomechanical forces implicated in atherogenesis and neointimal hyperplasia.

The results of this pilot study are promising, but should be interpreted with caution. The clinical success of FFR is mainly based on three unique characteristics: simplicity, reproducibility and crystal-clear cut-off value for binary decision-making. Whether vorticity can share these characteristics as well, is still unknown. It is important to highlight that the authors calculated the vorticity index using 3D-angiography, estimation of hyperemic flow from the TIMI frame count and application of computational fluid dynamics, a methodology that can be easily implemented in the cath lab for routine clinical practice. The clinical usefulness of vorticity and the identification of clinically relevant cut-off values warrant further investigation.

This work provides an interesting viewpoint that might help the interventional cardiology community to better understand the discrepancy between morphology and physiology, how for the same degree of stenosis, longer lesions result in higher loss of energy due to higher shear, and how eccentric lesions and blunt step-up/step-down transitions (i.e. abrupt changes in lumen diameter, such as in aneurysmal portions or with overexpanded stents) result in even higher energy loss due to increased shear forces and flow perturbations, and hence in increased vorticity and pressure drop (Fig. 1). In conclusion, the hemodynamic severity of a coronary stenosis depends on several factors. Even though an experienced operator could integrate many of them using compensatory fuzzy logic, plain angiography itself might not be enough to guide effective decision-making, as many clinical trials have consistently demonstrated [2–6]. Unfortunately, those in the interventional community, who feel blessed with extraordinary eyeball capabilities still prevail in real-world practice, so any effort to increase the presence of physiology in coronary interventions is timely and welcome. Overall, the current study upgrades vorticity from just a theoretical biomechanical concept to a functional parameter with useful clinical implications. The clinical and investigational potential is huge, but the way to go is still very long.



FFR, fractional flow reserve.

Fig. 1. Dynamic interplay between anatomical and functional parameters that define intracoronary physiology, fractional flow reserve and vorticity.

Conflicts of interest

The authors declared they do not have anything to disclose regarding conflict of interest with respect to this manuscript.

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