Motion Versus Flow as a Possible Nibus for Atrial Fibrillation

I read with interest the editorial comment by Asirvatham and Gard (1). The authors note that atrial fibrillation is more common in athletes and long distance runners. They then posit a complex theory (fulcrum for cardiac translatory movement) to explain the tissue damage that occurs at the level of the inferior pulmonary veins, as evidenced by the findings of fibrosis and fibrofatty changes reported by Platonov et al. (2). I suggest a less complex alternative hypothesis. Recalling the zones of West (3), perhaps tissue damage occurs at the level of the inferior pulmonary veins, particularly in long distance runners, because of the increased flow that these vessels support relative to their superior counterparts.

*Christopher C. Nessel, MD
*Johnson & Johnson Pharmaceutical Research and Development
920 Route 202
Raritan, New Jersey 08869
E-mail: cnessel@its.jnj.com

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Coronary Atherosclerosis and Quantitative Myocardial Perfusion: A Relationship Beyond Stenosis

We read with great interest the study by Naya et al. (1) on the relationship between coronary atherosclerosis, detected by computed tomography angiography (CTA), and myocardial blood flow and flow reserve (MFR), measured by positron emission tomography (PET).

Naya et al. (1) studied the association between plaque morphology, composition, and severity, on the one hand, and myocardial perfusion, on the other, in a population with known or suspected coronary artery disease (CAD). They observed that the “summed stenosis score,” an indicator of vessel atherosclerotic burden, was a better predictor of depressed regional MFR than stenosis severity. Moreover, the modified Duke CAD index, an indicator of the presence and extent of significant coronary lesions, was the major determinant of global MFR. The authors concluded that MFR is influenced especially by the presence and severity of coronary luminal narrowings, underplaying the influence of CTA descriptors of coronary atherosclerotic burden.
Coincidentally, we recently reported a study that analyzed the relationships between coronary stenoses and vessel structure assessed by CTA, PET-derived MFR, and cardiovascular risk factors (2). We showed that abnormal wall structure affects regional MFR beyond the presence and severity of coronary stenoses. Specifically, coronary calcium content was the main determinant of regional MFR and a significant predictor of depressed global MFR. Interestingly, when the Framingham risk score, an indicator of overall cardiovascular risk, was considered; it remained the only significant determinant of global MFR, beyond CTA variables. Although the two investigations are similar with regard to baseline characteristics of patients and differ only slightly in their methodology, they come to apparently different conclusions. In our view, however, both studies point to the effects of diffuse coronary atherosclerosis, in addition to those of focal significant stenoses, on myocardial perfusion.

Accordingly, depressed regional MFR is closely linked to the coronary atherosclerotic burden in the related vessel, described by the “summed stenosis score” in the study by Naya et al. (1) and by the coronary calcium content in ours (2). Moreover, global MFR is consistently related to different indicators of cardiovascular risk, the Duke CAD index in the study by Naya et al. (1), and the Framingham risk score in ours (2).

Riccardo Liga, MD  
*Danilo Neglia, MD, PhD  
*Fondazione Toscana G. Monasterio and CNR  
Institute of Clinical Physiology  
Via G. Moruzzi 1  
56124 Pisa  
Italy  
E-mail: dneglia@ifc.cnr.it

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Limitations of Noninvasive Measurement of Fractional Flow Reserve From Coronary Computed Tomography Angiography

We read with interest the paper by Koo et al. (1) regarding the diagnostic accuracy of noninvasive measurement of fractional flow reserve (FFR) from coronary computed tomography angiography data (FFRCT). We do recognize the potential clinical and economic relevance of the validation of a diagnostic tool able to noninvasively determine the presence of ischemia-inducing coronary lesions because it would dramatically reduce the number of diagnostic angiograms and guide subsequent coronary revascularization. However, we have some concerns regarding the interpretation of the results of the study.

First, the major potential drawback of FFRCT relates to the fact that FFR is calculated during “simulated” and not “real” hyperemia. To this end, the authors assume that “microcirculation reacts predictably to maximal hyperemic conditions in patients with normal coronary flow.” This sentence is substantiated by a bibliographic reference that demonstrates the reproducibility of the epicardial stenosis cannot capture the effects of diffuse atherosclerosis on vasodilator function of either the epicardial coronary arteries or the microvasculature. Nonetheless, we believe that both studies add valuable insights to the literature regarding the determinants and role of MFR, which will have increasing clinical application given its powerful prognostic significance (4).

Masanao Naya, MD, PhD  
Venkatesh L. Murthy, MD, PhD  
*Marcelo F. Di Carli, MD  
*Brigham and Women’s Hospital  
ASB L1-037C  
75 Francis Street  
Boston, Massachusetts 02115  
E-mail: mdicarli@partners.org


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