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EDITORIAL COMMENT

Computed Tomography Coronary Angiography, Percutaneous Coronary Intervention, and (S)low Flow*

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"No-reflow" and its mild versions, "slow flow" and "sluggish flow," are vexing and potentially severe complications of percutaneous coronary interventions (PCIs)—vexing because this inadequate myocardial perfusion is almost unpredictable, difficult to prevent, and occurs in the absence of angiographic evidence of any epicardial obstacle, thus giving the operator a sense of powerlessness; severe because a low "Thrombolysis In Myocardial Infarction" flow grade after primary PCI for ST-segment elevation myocardial infarction is associated with a poor clinical outcome (1).

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Few data are available concerning this phenomenon when it occurs in stable patients undergoing elective stenting in native coronary arteries. In this issue of *JACC: Cardiovascular Interventions*, Kodama et al. (2) compared the plaque characteristics as defined by computed tomography angiography (CTA) as well as clinical outcome in 40 patients with stable coronary artery disease in whom an elective PCI was complicated by "slow flow," with 40 patients in whom this complication did not occur. The presence of circumferential calcium in the vicinity of a soft plaque was observed on CTA in almost two-thirds of patients who developed "slow flow" associated with transient ischemia. What we do not know is how often this CTA finding is present in the general population of stable patients scheduled for elective PCI. Therefore, the overall specificity of this plaque characteristic as revealed by CTA seems weak. Moreover, given the absence of accepted preventive measures to avoid the "slow flow" phenomenon (3), and given the relatively favorable outcome of patients in whom this phenomenon occurred in the Kodama et al. (2) study, it is unlikely that the discovery of soft plaque with a ring of calcium will change the treatment strategy of the individual patient.

Several aspects of the "slow flow" phenomenon are different when it occurs in stable patients undergoing elective PCI compared with when it happens during primary PCI. The Kodama et al. (2) data suggest that, in stable patients, the presence of circumferential calcium deposits next to a soft plaque is an ideal substrate to induce "slow flow." When squeezed between the noncompliant calcium ring and the inflated balloon catheter, the atherosclerotic material has no escape other than the longitudinal axis of the vessel with protrusion into the luminal space. In addition to making intuitive sense, the data corroborate the idea that plaque embolization is the main mechanism leading to "slow flow" in stable patients. However, in patients with ST-segment elevation myocardial infarction, on top of embolization of thrombus and plaque material, ischemia-reperfusion injury is key to the pathophysiology of "no-reflow." The duration and extent of ischemia and of subsequent reperfusion injury as well as the susceptibility of the microcirculation to injury are responsible for profound and long-lasting disturbance of the vasoregulation pathways (4). The latter mechanisms are not present during elective PCI. These differences in pathophysiological mechanisms might explain—at least in part—the differences in clinical consequences of this phenomenon. Although "no-reflow" is known to be associated with increased mortality when it complicates primary PCI, patients with stable coronary artery disease and "slow flow" after PCI reported by Kodama et al. (2) did relatively well: only 3 developed limited myocardial necrosis, and there were no deaths at 30 days. In the patients described by Kodama et al. (2), "slow flow" was well-defined and was associated with objective and persistent signs of profound ischemia. Thus there is little doubt that low tissue perfusion was present in these patients. Fortunately, this complication is relatively rare in elective PCI. "Sluggish flow"—another term in interventionalist jargon—however, is relatively common after stenting. It is characterized by a lower flow velocity of blood and contrast in the epicardial artery not associated with signs of ischemia. This phenomenon is not necessarily associated with a poor prognosis and might also be based, to a limited degree, on the process of embolization. Yet, an alternative explanation to this "sluggish flow" resides in the acute changes in diameter of the epicardial artery after revascularization of a tight stenosis. When coronary perfusion is restored, the distension pressure increases, which in turn might lead to an increase in the diameter of the vessel. In addition, at the end of an angioplasty, several episodes of balloon occlusion-induced ischemia/hyperemia cycles might

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contribute to further endothelium-dependent epicardial vasodilation (5). Because the cross-sectional area of the artery relates to the square of its radius, a small increase in diameter might be accompanied by a marked decrease in flow velocity at constant absolute flow.

"Slow flow" does not necessarily mean "low flow."

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