



The pipe and the plug: Is unblocking arteries enough?

It seems anachronistic that we still debate how best to fix the plumbing of clogged arteries. Our understanding of the pathogenesis of acute coronary syndromes has evolved in leaps and bounds since the first attempts at coronary revascularization. And yet, as Aggarwal et al discuss on page 515 in their analysis of the FREEDOM trial,¹ practical and technical questions about how best to open coronary blockages remain clinically relevant, even as we develop strategies to reverse the atherosclerotic processes that created those blockages.

Many acute coronary events arise not from coronary stenoses but from unstable, vulnerable plaques, which may be a distance away from the stable stenoses and thus undetectable. These unstable plaques, embedded within the remodeled arterial wall and without a protective fibrous cap, may rupture and cause an acute thrombotic occlusion. Statins, aspirin, and perhaps anti-inflammatory drugs (now including colchicine) decrease acute coronary events, likely by interfering with the chain of events initiated by plaque rupture.

So why should coronary artery bypass grafting (CABG) be superior to drug-eluting stents (with antiplatelet therapy) in some diabetic patients, as the FREEDOM trial¹ found?

Stenting and balloon dilation repair discrete areas of critical narrowing presumed to be contributing to downstream myocardial ischemia. But areas of vulnerable, non-calcified plaque (with outward remodeling of the vessel wall but generally preserved lumen integrity) may be geographically separated from the identified stenosis and thus be left untreated by stenting. On the other hand, CABG may circumvent “silent” areas of nascent vulnerable plaque that, if left in place, might later rupture and cause acute syndromes or death.

This explanation is clearly hypothetical and one of many possibilities. But paying attention to the new biology of the atherosclerotic process should lead us all to be more aggressive in using treatments shown to reduce the progression of coronary artery disease and the occurrence of acute coronary syndromes. This is especially true in patients with diabetes who are known to have diffuse coronary involvement. So even as we more fully recognize the value of CABG in these patients, perhaps if we intervene earlier—with statins, hypertension control, improved diet, smoking cessation, prevention of chronic kidney disease, antiplatelet therapy, and anti-inflammatory therapy—we will not need it.

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1. Farkouh ME, Domanski M, Sleeper LA, et al. Strategies for multivessel revascularization in patients with diabetes. *N Engl J Med* 2012; 367:2375–2384.

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