



The Society for Cardiac Angiography & Interventions

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The Donut Hole

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My eldest son, Matthew, nicknamed him “The Dunker.” Carefully slipping his finger through the donut hole, he would dunk the donut into his ice-cube-chilled coffee, lean carefully over the cup and slide the soggy donut edge into his mouth. Such was the morning ritual of my grandfather John Lapp, a.k.a. “The Dunker.” Of course, the donut hole was of paramount importance to the process. While it was possible to do with stick donuts, the hole made the procedure far easier, and much more elegant.

What, you may ask, do donuts have to do with interventional cardiology?

I propose that, as it was to my grandfather, the donut hole is critical to our rituals as well. For nearly fifteen years I have worked with intravascular ultrasound (IVUS); of course displayed in “donut” format with the hole for blood flow in the center surrounded by the fat-laden rim of plaque. For equally as long, I have struggled to teach new users that while their eyes are drawn to the abundant plaque, what is actually more important is the hole within. “We treat narrowed lumens, not plaque” I repeatedly insist.

I recently attended an international congress on coronary physiology. I was gratified that over 200 of my colleagues sat expectantly in an auditorium for two days

to learn the intricacies of physiologic measurements performed in the cath lab. I felt a sense of *deja vu*. Some of my earliest manuscripts written as a cardiology fellow dealt with the impact of intervention (PCI) on coronary physiology [1,2]. I don’t think it is coincidence that my latest abstract (albeit using different methodology) dealt with the same topic [3].

This should not surprise me. While our tools have changed, our reason for doing PCI has not: narrowed lumens restrict blood flow and cause symptoms – we fix narrowed lumens and relieve symptoms. Quite a simple concept, but one which should not be forgotten.

I am old enough to have had the honor to meet Andreas Gruentzig on several occasions. You may recall that one of his axioms was that you don’t do PTCA without objective evidence of ischemia. Somewhere along the way we have forgotten this basic principle. The oculo-stenotic reflex has become well developed. In

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fact, we now hear of placing stents over minor lesions in the name of “plaque sealing.” A slippery slope in my estimation.

What does the evidence teach us? Even in my first manuscript, the final trans-lesional pressure gradient at rest (now known to be to be less sensitive than that measured during maximal hyperemia) correlated with patient outcome [1]. Using advanced wire technology and hyperemic conditions, a recent multicenter study showed that this is still true [4]. The majority of their patients after stenting had an FFR > 0.90 which was associated with a “DES-like” event rate of < 6.2%.

In the DEBATE-II study, similar excellent results were obtained when the coronary flow reserve was > 2.5 following stenting [5].

Not only can physiology predict which patients will do well after PCI, physiology also can tell us which patients will do well *without* PCI. In the Defer Trial, patients with angiographic lesions but FFR > 0.75 (not hemodynamically significant) were randomized to PCI or medical therapy (deferral) [6]. Not only did the deferral group have fewer events, they had significantly better anginal relief at two-year follow-up. In our own experience collected over the past five years in routine clinical practice, 80% of questionable lesions were found to be hemodynamically insignificant (FFR > 0.75). Over a more than two-year follow-up they had a major adverse cardiac event (MACE) rate < 8%. Even the lesions located in the LAD (traditionally higher risk) had only a 5% “DES-like” MACE rate [3].

Lest you worry that widespread use of FFR for assessment of non-critical lesions put your interventional lab out of business, the “Tailored approach” study suggests that it may be the cardiothoracic surgeons who need to worry [7]. These investigators took patients with multivessel disease referred for CABG and studied the target lesions physiologically. In those who had only one or two hemodynamically significant (FFR < 0.75) lesions, PCI was performed. Those with multivessel significant (FFR < 0.75) lesions were sent to CABG. The physiologically guided PCI patients had an excellent outcome, no different than that achieved after CABG. So why should the surgeon worry? A full 40% of the referred patients were “converted” from CABG to PCI once their physiology was measured!

Knowledge of physiology does matter, not just for the PhD's in the medical school, but for us every day in the cath lab. Through this journal SCAI has attempted to keep you informed. Dr. Kern's “Hemodynamic Rounds” are a fantastic educational resource. I encourage you to spend time understanding and exploring the physiologic impact of the pathologic processes we treat. I have focused here on coronary artery disease, but the same is true of septal ablation for hypertrophic cardiomyopathy, closure devices for atrial septal defects, or balloon valvuloplasty for aortic stenosis.

A good cardiologist has not only an intimate knowledge of the tools of his/her trade, but also when to apply these tools, and how to assess their efficiency.

So, the next time you step into the cath lab, I encourage you to take your eyes off the donut and look intently at the donut hole instead. We treat narrowed lumens after all. Be sure the next lumen you treat is truly narrowed.

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