All Roads (Even Those Less Traveled) Lead to Rome

Benefits of Transradial Coronary Interventions

As he walked off the table following a transradial percutaneous coronary intervention (TRPCI), a morbidly obese patient asked me if the route from the wrist to the heart was more direct than that from the groin. He had reached this conclusion because his previous transfemoral procedure by another physician was prolonged and resulted in a large retroperitoneal hematoma, necessitating transfusion and protracted hospitalization. He was amazed at the ease and speed with which the TRPCI was performed. I replied that all roads lead to Rome; some are just easier to find and safer than others.

I applaud Dr. Kern’s recommendations in his editorial “Cardiac Catheterization on the Road Less Traveled: Navigating the Radial Versus Femoral Debate?” from the previous issue of JACC: Cardiovascular Interventions (1), that the seasoned interventionalist to be well-versed in both access sites. The virtual elimination of bleeding risk associated with TRPCI is undeniable, even by the skeptics. As the obesity epidemic appears to spread unchecked in our nation, one can only expect that physicians will encounter more patients similar to the one in the preceding example. Ironically, we trail other nations in acceptance of this technique. This patient is illustrative of the size paradox for TRPCI. In most cases, obese patients are actually excellent candidates for this approach, as they have large radial arteries, with robust pulses.

Immediate ambulation is a great benefit for individuals afflicted with back conditions. Patients not infrequently consider the post-procedural groin compression and recuperative bed rest as the most uncomfortable aspects of the procedure (2). As demonstrated by Cooper et al. (3), all quality-of-life indices favor transradial over transfemoral access. A secondary, perhaps less appreciated, difference is the intangible loss of privacy associated with instrumenta-
tion of the very personal groin area. When questioned, many patients thus perceive the transradial approach as somehow less invasive.

Another patient, who had undergone uncomplicated transfemoral stenting years ago, developed an inflammatory cyst at the access site after a subsequent TRPCI. This phenomenon is a rare hypersensitivity reaction to the hydrophilic coating on certain transradial sheaths and requires only lancing and application of antibiotic ointment. Upon hearing my apology for the adverse occurrence, the patient simply inquired: “Doc, can you please still use my wrist next time?” He preferred a (minor) transradial complication over an uncomplicated transfemoral procedure.

Importantly, elective PCI in the U.S. is now defined and reimbursed by Medicare as an outpatient procedure. TRPCI is ideally suited for early ambulation and discharge, thus allowing alignment of practice with payment. In the STRIDE (Same-day TransRadial Intervention and Discharge Evaluation) trial, we retrospectively demonstrated the potential safety and feasibility of ambulatory discharge following TRPCI (4).

Regarding concerns over radiation exposure, simple setup modifications can easily remedy the situation. By positioning the arm parallel, rather than perpendicular, to the body, the wrist is actually below the level of the groin. Moreover, the addition of an extension tubing allows the operator to stand at the level of the patient’s foot during injections.

Clearly, interventionalists, like travelers, will differ in their choice of routes to a common destination. Nonetheless, it may be preferable to have access to both the larger freeway as well as the smaller, less travelled but perhaps safer, back road.

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Reply

I thank Dr. Chen for his interest in my editorial (1) and for again highlighting the undeniable facts about the superiority of the transradial approach relative to the U.S. standard of femoral access. The issue is not whether radial access is better but why has it taken so long to be adopted in this country and what can we do about it?

As a recently converted sinner, I am intimately familiar with the rationale to delay adoption but once on the “Radial First” road, my experience and that of my patients confirms it is the right road. I gained some insight from a recent conversation with a young catheterization lab director in Springfield, Massachusetts, when asked why he was not teaching radial technique to the fellows in training. The same litany of excuses was grudgingly provided. “It was the way I was trained” was the first excuse. If we kept to that approach, I would still be doing cut-downs on the brachial artery.

I believe it is the duty of every interventionalist in charge of training fellows to take this challenge and duty to their patients to heart. I am sure Dr. Chen would agree that we should use this new
momentum, driven by the extensive datasets on better radial access outcomes to encourage the next generation of interventionalists to step to the front of the world’s stage in patient care.

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Phenotypes, Genotypes, and the 9p21 Locus for Prediction of Cardiovascular Events

I would like to commend Hoppmann et al. (1) for their thorough and well-executed paper. In this regard, I agree with the editorial by Horne and Anderson (2) that discusses the importance of the Hoppmann et al. (1) study. Thoughtfully executed prospective studies that attempt to clinically examine genomic data first identified in genome-wide association studies are at a premium.

Other remarks in the editorial by Horne and Anderson (2) are a source of disagreement. First, the authors make the argument that because replications of the 9p21 single nucleotide polymorphisms (SNPs) have been done in populations that suffer from both documented coronary artery disease (CAD) and myocardial infarction (MI), these individual phenotypes—when grouped together—confound the type of risk (phenotype) that can be attributed to these genetic factors. Although their argument is understood, it is important to point out that on the basis of experience, these genetic markers are statistically significant in both subpopulations to sometimes nearly equal extent. These data are sometimes not shown in final reports and are only rarely shown in supplemental data. The authors then attempt to differentiate the pathogenesis of CAD and that of MI as 2 distinct entities, which further precludes attribution of genetic risk to either of those 2 phenotypes individually. It is difficult to assume that the well-known progression of CAD to MI (barring less usual suspects such as spasm or dissection) can be so thoroughly extricated from each other as to invalidate the dozens of studies that have replicated the 9p21 locus as a risk marker for CAD and MI.

The authors then make the argument that because the study by Hoppmann et al. (1) finds there to be a negative association between restenosis and these genomic markers, restenosis must be a distinct pathophysiologic entity from CAD because it is not “driven” by genetic factors at 9p21.3. Though this might be the case as evidenced by much work in cell biology and immunology, an assumption based on 1 prospective clinical study that examined 4 SNPs is difficult to accept. Additionally, it should be noted that the 4 SNPs tested by Hoppmann et al. (1) are not the most frequently validated SNPs for 9p21 but rather an amalgam of SNPs from different studies that first identified the variants. This is possibly due to the initiation of the 3-year prospective study before replications of the more popular variants in better-characterized populations.

It is certainly necessary to temper our enthusiasm for direct-to-consumer genetic testing until these markers can be better understood, a point of agreement with Horne and Anderson (2). In the interim, we should encourage more studies such as the one presented by Hoppmann et al. (1) and continue our emphasis on preventative cardiovascular medicine.

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Reply

We would like to thank Dr. Abdullah for the remarks regarding our editorial (1). We appreciate the subtleties of our assertions and acknowledge that the important biological distinctions among myocardial infarction (MI), coronary artery disease (CAD), and restenosis might be unfamiliar. We appreciate the opportunity to clarify our arguments.

It was not our intent to claim that prior studies associating 9p21.3 with MI risk are invalid but to say that some erroneous conclusions about pathophysiological implications for coronary heart disease were drawn from those landmark studies. Because restenosis is a different process than CAD (2), our intent also was to note that 9p21.3 is not involved in its distinctive pathophysiology (1).

A major component of our argument is, in fact, that 9p21.3 single nucleotide polymorphisms “are statistically significant in both subpopulations to sometimes nearly the same extent.” To illustrate, consider 1 European study that showed a similar effect