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that this imaging modality is the most exciting and the most promising technology to guide therapy. I collaborate with investigators from NYU and Mt. Sinai in using nanoparticles to image hot vascular plaques in a more detailed and accurate way than the traditionally used contrast material. I am super excited about this!

What is the role of the Peripheral Vascular Surgery Society (PVSS)?

Over the past few years, the quality of the meetings has skyrocketed with record attendance, original research presentations, industry support, and the new fellows' program a day before the winter meeting. We meet twice a year: in the winter (ski meeting) and spring (during the SVS). Many of the members of PVSS are division chiefs and leaders in the field on their own merits. It can be summarized that while PVSS maintains the same academic intensity, it is the "younger and hip" version of the SVS.

Why is this group important to young vascular surgeons?

PVSS does a good job preparing young vascular surgeons for future leadership positions within the regional and national societies. For those who choose a career in academics, PVSS is the perfect start. It offers the structure, material, and the variety (committees) without being ultra bureaucratic. I chair the fundraising committee for the PVSS, and it gives me great satisfaction to see our competitiveness today compared to when I was a fellow.

As a fellow, what was one expectation you had about being a vascular surgeon that turned out to be incorrect?

Like most fellows, I had to learn to respect experience and tame my interest in doing big and complex cases with the fact that I needed to gain the confidence of my partners and OR staff. This takes years. I also found that practice building takes time and is no small job. As a fellow, you are not really prepared for the "real thing" as much as you find out when you are an attending. Having said that, I prefer being an attending! ■

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Firas F. Mussa, MD, MS, FACS

Dr. Mussa shares his insights on the future of TEVAR for uncomplicated type B aortic dissection, percutaneous EVAR, and treatment options for CLI.

Will TEVAR be the treatment of choice for uncomplicated type B aortic dissection?

We are heading in that direction. Currently, TEVAR is associated with improved aortic remodeling at 2 years. In the near future, the 5-year results of the INSTEAD trial will be published. This will be a game changer as far as the role of TEVAR in uncomplicated type B aortic dissection. Furthermore, new imaging software dedicated to flow dynamics is going to play a critical role in selecting the appropriate patients for TEVAR. We will also witness a significant reduction in TEVAR-related complications such as stroke and retrograde dissection as a result. I am also very excited about the possible expansion of IRAD (International Registry of Aortic Dissection). This can facilitate data sharing and the development of modern treatment paradigms that can best help our patients.

As smaller devices become available, what kind of changes can we expect in terms of who is performing EVAR and how it is being performed?

I do not expect a major shift in who is performing EVAR just because the sheath size is getting smaller. This is mostly governed by referral patterns and institutional policies. With smaller devices, however, more vascular surgeons are likely to adopt percutaneous EVAR (PEVAR). I believe that vascular surgeons currently lag behind other specialists when it comes to using closure devices in general and for larger sheaths specifically. If this trend continues, I imagine that patients are going to demand the "no incision" aneurysm repair and create another shift in referral away from vascular surgeons. I also believe that PEVAR is associated with faster recovery, less blood loss, and ultra-short length of stay. I anticipate that in the near future, a cutdown will be the exception rather than the rule!

What are the current recommendations for open bypass versus endovascular repair for critical limb ischemia (CLI)?

As of today, for those who have a suitable autologous vein conduit and life expectancy that is more than 2 years, open bypass is superior, more durable, and possibly more cost effective than endovascular intervention. Unfortunately, many of our patients have less than 2 years life expectancy with no suitable vein, so endovascular intervention becomes the default first line of therapy. Having said that, I am an endovascular enthusiast and believe that the below-the-knee (BTK) vascular bed deserves special attention. We are seeing less balloon angioplasty as a standalone therapy for CLI and more drug-eluting stents, drug-coated balloons, atherectomy, retrograde tibial punctures, etc. My personal views are that vascular surgeons need to embrace and validate the newer technologies if they are to remain competitive in this field.

What can be done to avoid poor outcomes for open revascularization if an endovascular treatment has been performed previously?

It makes intuitive sense that repeat intervention is associated with biologically aggressive disease. We have shown that patients who undergo bypass after a failed initial endovascular intervention have worse limb salvage rates; this will be presented at the 2012 Eastern Vascular Society meeting. I think this might be related to the delay or reluctance in offering open bypass in favor of an endovascular approach. Not surprisingly, however, there remains a significant proportion of patients who are first treated with endovascular intervention requiring an open procedure at some point down the line. Anecdotally, the two most problematic patients are those with end-stage renal disease and/or diabetes. One is worse with endovascular (end-stage renal disease), and one is worse with open revascularization (diabetes). Furthermore, medical therapy has undergone significant transformation with high-dose statins and dual antiplatelets; the outcomes of both open and endovascular interventions have somehow improved.

How does plaque characterization guide therapy for patients with asymptomatic carotid disease?

Using FDG-PET MR/CT fusion technology to study "vulnerable" plaque in patients with carotid disease is bound to take off. It picks up inflammation in macrophages inside the plaque as increased signal uptake and superimposes the images from PET with MR or CT scan. We have also shown that statins modify this process in the carotid artery in a dose-dependent manner. With the debate over intervention for asymptomatic carotid disease (CEA and CAS) nowhere near settled, I believe

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