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*The following faculty report no relevant financial interests:* Dr Catherine Wittgen.
Carotid Disease: Diagnostic Modalities

During the past 5 years, how we diagnose carotid disease has not changed significantly for surgeons. Most of us still rely on duplex ultrasonography, which gives both the US and the velocity criteria for estimating stenosis. In centers where the vascular labs are good, duplex US is very reliable, making arteriography (the gold standard) unnecessary. Most of us have gotten away from angiograms because they are associated with a real risk of stroke. In the past 5 years, our consulting physicians are relying more heavily on MR angiography (MRA) and CT angiography (CTA). Unfortunately, both of these have problems when trying to estimate a degree of stenosis.

CTA: A shortcoming of CTA is that it must be viewed carefully. In our institution, when we look at CTA on Synapse®, we joke that, when looking at it in bright whites (contrast looks same as bone), “it’s 2 inches left, 2 inches down,” which is how we want to move our mouse so that we see the contrast column separate from vessel wall calcification. Even with that, accurately estimating disease on severely calcified lesions can be difficult. This is a problem for our consultants, who will call a lesion “severely stenotic” when, on duplex US, it’s really not that bad.

MRA: MRA also has problems, especially with signal dropout. When we look at those little protons that are being excited, if we are out of phase and we get a slice that is not appropriately timed, we may not get the right estimation of disease. MRA is also much more radiology-dependent in how someone manipulates the images: it really does take someone committed to the process to put this thing back together.

Reconstructions: I stress to the residents that, for both MRA and CTA, we do not want to look at the reconstructions. We really need to look at the source images in cross section because they give us the most information on the degree and location of stenosis. Reconstructions are useful if the patient has a high lesion and you are concerned that the bifurcation is beyond your reach or that the plaque does not end until it reaches the base of the skull. In those cases, the reconstructed images within the outline of the face and the neck can provide some useful information about whether “high” is really too high.

Images vs Plaque Type: Some of these imaging modalities can give you an estimate of the type of plaque present. This can be important since some data suggest that more heterogeneous plaques are associated with an increased risk of embolism, transient ischemic attack (TIA), and stroke.

Angiograms: Although angiograms increase the risk of stroke, they are sometimes diagnostically helpful in patients with severe calcification and extensive disease and in patients being seriously considered for stenting.

Symptomatic Carotid Disease: The Decision to Operate

For the symptomatic patient with carotid disease, my ideal operative candidate would be someone who already had his heart checked out for another reason, is male, reasonably thin, has a bifurcation in the midportion of his neck, truly has a bifurcation lesion without extensive plaque or a tongue of plaque extending up his distal internal, and his internal vessels are ≥4 mm in size. The symptomatic patient who is an operative candidate should have >60% stenosis if he has a perfect heart. If he has some other heart history, some people would take that number as high as 70%.

Local Politics & Skill: The decision to make a symptomatic patient with carotid disease an operative candidate can be influenced by the local skill set at a given institution. Many surgeons and other interventionalists want to do stenting because the technology is available. In the 1980s, we had a debate about carotid endarterectomy, with Moore saying that we should not operate on asymptomatic patients unless we could prove that our periprocedural stroke rate was ≤2%. Hospitals are now starting to get onboard with that idea for carotid stenting as well. Unfortunately, the Centers for Medicare and Medicaid Services (CMS) continues to dance around this issue. In stenting versus endarterectomy trials, usually one of the big caveats in the trial is that the surgeons are well-trained and, based on analysis of their outcomes, their work is associated with exceptionally low stroke rates. While using ideally
qualified surgeons for a trial is great, we cannot extrapolate the results of those trials down to a community level where the operator’s incidence of the stroke may not be known.

Symptomatic Carotid Disease: Beware the Conclusions of the CREST-1 Trial


**Suspicious Methods:** We have been looking at stenting trials now for 10 or 15 years. These trials are industry-sponsored. The conclusions state that no significant differences were found between CAS patients than CEA patients. While not statistically significant, the absolute risk of periprocedural stroke or death and subsequent ipsilateral stroke was 37% higher for CAS than for CEA (difference statistically significant). The authors comment that the absolute rates of stroke were <7% across the trial, but that number is actually higher than what we like to see. Additionally, during their 10-year follow-up, the risk of stroke or death was higher in CAS patients than CEA patients. If you are someone who appreciates looking at the boxes with the lines for the confidence interval, you can see that the lines for the confidence intervals are somewhat wide in some cases. While not statistically significant, the article shows categories in which CEA provides clear advantage, meaning that CEA is better than CAS for some classes of patients.

**Noninferiority Trials:** I caution those who read this paper — do not be seduced by reading the abstract’s conclusions because they do not necessarily reflect the study’s data. Interpreting noninferiority studies is difficult: the statistics for noninferiority or equivalency are very different than for other types of studies. In noninferiority trials, an intent-to-treat analysis gives a statistical advantage to the thing being tested against for noninferiority. These trials need to be analyzed both on an intent-to-treat basis and based on what patients actually received. In addition, noninferiority trials must set an acceptable range or equivalence point for an outcome, such as an “acceptable” stroke rate. This value is what they will test against for noninferiority.

**Don't Be Misled:** The statistical analyses in this trial were done on an intent-to-treat basis, meaning the study may have been biased toward a favorable outcome in CAS. Also, long-term event rates were based on a Kaplan-Meier estimates because the number of strokes were very small, which is a huge confounder. In this trial, the incidence of dyslipidemia was lower in the stent group than in the CEA group. Given how we now manage dyslipidemia, we could argue that its impact may not be as significant as it was 10 years ago. More than one-third of patients in this trial were lost to follow-up. The conclusions state that no significant differences were found between CAS and CEA patients with respect to the risk of periprocedural stroke, myocardial infarction, death, or subsequent ipsilateral stroke. However, in the secondary analyses, the risk of periprocedural stroke or death and subsequent ipsilateral stroke was 37% higher for CAS than for CEA (difference statistically significant). The authors comment that the absolute rates of stroke were <7% across the trial, but that number is actually higher than what we like to see. Additionally, during their 10-year follow-up, the risk of stroke or death was higher in CAS patients than CEA patients. If you are someone who appreciates looking at the boxes with the lines for the confidence interval, you can see that the lines for the confidence intervals are somewhat wide in some cases. While not statistically significant, the article shows categories in which CEA provides clear advantage, meaning that CEA is better than CAS for some classes of patients.

Asymptomatic Carotid Disease: New Study’s Results of Questionable Value


**Questionable Methods:** I have several “cautions” for those who read this paper. This trial was highly selective in its study population: patients were aged ≤79 years, had severe carotid stenosis, and were asymptomatic. After a neurologic exam was performed to ensure that patients were indeed asymptomatic, patients were randomly assigned to CAS or CEA. Sites with very high rates of adverse events were “counseled” regarding case selection and technique. Two sites were temporarily stopped from enrolling patients. In other words, sites with data showing excess events were released from the
study. The age requirement is significant because age 80 years appears to be a break point in CAS: patients aged >80 years do not do as well with stenting compared to those <80 years.

**Medical Therapy:** The medical therapy used in this trial was also “interesting. Clopidogrel was started 3 days before the procedure and continued for 30 days after. Outside the study, clopidogrel is started 5 days in advance to provide the patient with the drug’s full impact of clopidogrel. I suspect that the 3 days used in this study was a hedge factor trying to minimize periprocedural access complications by not having them fully antiplatelet inhibited.

**Noninferiority Point:** Noninferiority trials must set an acceptable range or equivalency point for an outcome. In this study, this point was set as ≤3 percentage points difference between CAS and CEA for the primary end point. Although this does not sound like much, we are talking about a procedure that has a 3% risk of having an event. I am curious about how this number was derived.

**Power & Outcomes:** The study was designed to enroll 1658 patients, and with that they were powered to 80%. They stopped enrollment, however, subsequently making the power of the whole trial 75%. The major death or stroke rate was low in both groups (<1%), but the 30-day rate of minor stroke was higher with CAS (2.4%) than with CES (1.1%; P = 0.20). The overall 30-day rate of death and stroke was 2.9% with CAS and 1.7% with CEA (P = 0.33). Estimated 5-year survival rate was 87% for CAS and 89% for CEA (difference not significant).

**My Conclusions:** This is an okay trial. Does it change my opinion on who needs a stent versus CEA? No, not yet. For patients who are not good operative candidates, stenting may offer an acceptable option, but I think you have to look very carefully at your home institution’s numbers if you are contemplating a CAS on a patient who qualifies for CEA.

**Carotid Disease: One Surgeon’s Decision for CEA versus CAS**

**Case:** The next 100 patients come in with asymptomatic or symptomatic carotid disease, and all are operative candidates. No patient has had prior neck surgery or radiation, and no patient has severe comorbidities such as severe COPD, unstable angina, or scary heart disease. At your institution, how many of these patients will get CEA and how many will be stented?

**Response:** In this ideal group of patients, probably only 5% to 10% would undergo stenting, and would be part of an ongoing study. Generally at our institution, if patients meet criteria for an operation, they will get an operation. However, at surrounding community hospitals we are seeing more stenting done. Because we are a teaching institution, we keep these patients overnight for a number of reasons. Primarily, all go to CEA under general anesthesia. I am not a fan of trying to teach CEA to a resident while the patient is awake and blocked. Because of the general anesthesia, we keep patients at least overnight. The Society for Vascular Surgery Website contains information for patients at large, and they do not mention same-day carotid surgery. Their wording states that the patient should anticipate staying in the hospital for 1 to 2 days. It is relatively rare that we keep someone 2 days unless they are at extremes of age or they come from many miles away and have very few resources at home. Most of our patients are dismissed early the next day.

**Carotid Disease: One Surgeon’s Approach to CEA Shunting and Patches**

When performing CEA, the decision to shunt a patient is based on how I set up my CEAs. I use cerebral oximetry because it is the best method that I can get reimbursed for that has results which are very reliable and reproducible. When I set up my case, two patches on the patient’s forehead give me a starting oximetry number, which is affected by blood pressure, level of anesthesia, and bone thickness. Before I clamp, I look at is the baseline number when I have my blood pressure set at basically the high end of normal. During surgery, I consider a 25% drop in that number to be significant. If it goes down,
I already have shunts set up on the field. I use the cheapest plastic tubing shunts that are straight. I will have already tied a string in the middle on each of them that I think I might use. I select one shunt that I think will be the right size and then I select another that is one size smaller, which gives me a back-up shunt if the resident should drop one. The string allows me to get it out more easily. If the cerebral oximetry drops after clamping the internal, external, and common carotids, I put in the shunt and away we go.

**Oximetry:** I clamp the internal carotid artery (CA). Then I clamp the external CA and finally the common CA. Then I make the longitudinal arteriotomy. I have spoken with my anesthesiologist ahead of time and they know my magic number for cerebral oximetry. We do the math ahead of time. If the baseline oximetry is 60, then I instruct the anesthesiologist to alert me when it approaches 45. During surgery, this is an ongoing conversation. They will tell me, “It’s starting to drop a little bit, but he’s settling out at 50 ... it looks okay.” Or they might say, “No, he’s heading down again.”

The decision to shunt is based on this communication.

**Patch:** When performing CEA, I am a selective patcher. I am more likely to patch a female patient because women do not do as well as with CEA as do men. This may have to do with vessel size: I believe smaller vessels are more prone to recurrent stenosis. I am also more likely to patch if a trainee will be closing the vessel. Their suture bites may not be as small and as precise as mine, depending on their level of training. Finally, I am more likely to patch a male patient if their distal internal does not look like it will accept the largest size shunt. However, if the vessel looks like it will take the largest size shunt and if I have a trainee who sews very well, then we can close those primarily (no patch).

**Carotid Disease: Medical Therapy**

The ongoing Carotid Revascularization Endarterectomy vs. Stenting Trial part 2 (CREST-2) is addressing the issue of what is the best medical therapy for carotid disease and whether that medical therapy changes outcomes as we follow patients with carotid disease. Neurologists have data in their literature indicating that we are getting very aggressive with antiplatelet therapy and that our management of hyperlipidemia is getting so good that the natural history of moderate to severe carotid disease is significantly better than what was previously estimated or recorded. One of the conclusions that may come out of CREST-2 is that medical therapy is very good in 2016. Small series have looked at the question of whether good medical therapy reduces or stabilizes these carotid plaques. However, we do not have anything consistent looking at the same imaging modality (duplex US) with a large number of patients using statins at the level that we want to use in 2016 (target: LDL <100 mg/dL or, in some cases, <70 mg/dL).

**Peripheral Arterial Disease: Claudication — Diagnostics**

**Case:** A patient is referred to the surgeon because they have symptoms of claudication. They do not have a threatened limb. What’s the diagnostic workup on these patients today?

**History:** First, talk to the patient and take a reasonable history. Approximately 60% of patient referred to me with claudication truly do not have the classic description. Instead, someone in primary care suspects that the pain in the patient’s legs when they walk is claudication, so the provider refers them to the vascular surgery. Claudication should be pain in large muscle groups, buttocks, thighs, and calves. It should be reproducible at the same distance. It’s not ankle, foot, or knee pain, nor is it arthritis. I also get those patients who tell me, “Oh, it starts at my hip and it goes down my entire leg.” But asked about walking at a large grocery store with long isles, they say, “Oh, I’m fine. I get a cart, I lean over.” These patients that can walk as far as they want once they get their spines unloaded have neurogenic claudication. Therefore, taking a good history is very important in this group.
Objective Data: Next I do a physical exam. I do my own pulse exam and do not rely on that done by others. I examine the legs. Have they lost enough oxygen tension that they’re now hairless on the first toe? If the leg is hanging down, what color is it? If I elevate it, does it blanch? Next I get an ankle-brachial index (ABI). In our institution, every ABI comes with a first digit pressure as well.

Risk Factor Management: Although some provider has convinced a patient that claudication is responsible for their leg pain with exercise, aggressive risk factor management may not have been attempted before the patient presents to the surgeon. These patients need stop smoking and get their diabetes under control. If they have hyperlipidemia, they need to be on a statin.

Other: Once I have identified abnormal ABIs and agree that the symptoms are consistent with claudication, I do not get any other invasive tests. as an initial maneuver. Patients are generally seduced by their provider’s description of the ease and short intervention required to have stents placed. However, providers are not telling these patients that we measure patency on stents at that 2-year mark. What’s more, when stents occlude today, we are creating an entirely new patient population of acute limb ischemia. More than just the stent occludes — usually a significant portion of the vasculature both proximal and distal to the original lesion also occludes.

Peripheral Arterial Disease: Claudication — Nonoperative Management

Case: A patient presents to the surgeon with claudication, no tissue loss, and an abnormal ABI. What nonoperative management is recommended?

Recommendations: First, I send a note back to the primary telling them where I want the patient’s LDLs to be and that their blood pressure needs to be controlled. I try to keep that management in the primary physician’s office. Next, I outline a walking program. I tell them to find one-time access to a treadmill (gym, mall, resale shop for sports equipment, etc), and ask them to step onto the treadmill and set it at a pace of 2 miles/hour. I want them to know how fast that pace is. They should walk for a couple minutes. Nearly every patient with an ABI of 0.7 can do that. My goal with this treadmill time is to become very familiar with this walking pace. Then, I want them to walk every day at that 2 mile/hour pace. I warn them that they will get pain but I reassure that the pain is not causing any harm. I instruct them to walk until they can’t go any further and to stop and rest. I ask them to note how long they were walking before needing to stop. Once the pain eases up (usually in about 3 to 5 minutes), they should start walking again. I want them to walk 20 minutes every day. I see them again at 3 months because I want them to know that someone is watching. I have them keep a notebook in which they track every date and time they walk, as well as the location, total distance, and time walked before stopping for the pain. For committed patients, that notebook becomes a thing of pride. This also gives me a chance to identify the occasional patient with worsening symptoms due to the rupture of moderately stenotic plaques. I also want to know the patient who is not improving despite a good exercise program as I will probably intervene on sooner rather than later.

Lesion Location: Since I see referred patients, I do not see many cases of femoral popliteal disease. Because doing the types of procedures needed to treat these lesions is relatively straightforward and easy, providers in the community are treating them without referral to surgeons. Generally, the patients referred to me have multilevel disease. In the population as a whole, statistically the lesion at the adductor canal in the leg right in the mid-superficial femoral artery is where a narrowing or an occlusion is most likely to occur.

Peripheral Arterial Disease: Acute Leg Ischemia

Case: An elderly patient arrives with acute leg ischemia. They have been on medical therapy, and they have one stent in the proximal arterial tree. They have a pulse at the femoral, but have no pulses below.
The foot is pale and painful. The ischemia has been going on for about 2 hours. How do you manage this patient?

**Recommendation:** These cases generally present in the middle of the night because the patient can’t sleep. The question for us is whether to use thrombolytics versus intervention. Because this case has a previously stented leg, the bias is toward greater success with thrombolytics, but we have the issue of viability. In our initial assessment, we must determine whether this leg will tolerate the period of ischemia required for the thrombolytics to work. This issue of severity of ischemia versus time required for thrombolytics to work must be weighed for each one of these patients. Available data from the Thrombolysis Or Peripheral Arterial Surgery (TOPAS) trial and from Surgery versus Thrombolysis for Ischemia of the Lower Extremity (STILE) trial support either an open intervention or thrombolytics. However, both trials emphasize that you must consider what has already been done to the extremity and how long you think that extremity can tolerate ischemia. Some of this, at least in my group, is also hugely operator-dependent. Some have a personal preference to immediately go to thrombolytics, while others prefer a case-by-case assessment before deciding on therapy. Nonetheless, if I am concerned about how ischemic that foot is, I will probably go to the operating room to get reperfusion sooner rather than later. The subgroup that gets open intervention encompasses those patients who have had ischemia for a longer duration and who have more profound ischemia. When reperfused, these patients are much more likely to develop compartment syndrome. Therefore, the open surgical patient generally undergoes both embolectomy and fasciotomy. I rarely see a patient who is treated with thrombolytics also undergo a fasciotomy.

**Peripheral Arterial Disease: Below-Knee Fasciotomies**

For the case that presents with acute lower extremity ischemia, my recommendation is that if you find yourself considering a below-the-knee fasciotomy, then you need to go ahead and do it. You will not be sorry for having done it. Although it is written that you can do this with a single incision, I think that is ridiculous in this patient population. It requires two incisions: one on the medial aspects of the leg and one on the anterior aspect midway between the tibia and fibula. For the medial incision, no matter how carefully you plan it, the greater saphenous vein always seems to be directly underneath. Here, you want to release the superficial and the deep compartments. On the anterior leg, make the incision midway between the tibia and fibula, and you have two compartments to release (anterior and lateral). The skin incisions do not need to be huge: start with something that is four to six inches. If the muscle looks great and you don’t have a lot of bulge and this is someone that you don’t have to worry about ongoing resuscitative efforts, then you have your answer — you can probably close this in a day or two. But if there is any question at all or you start noticing the bulge, make a larger incision.

**Wound Management:** If the fasciotomy incision extends the whole length, then I use a vacuum-assisted closure (VAC). The exception to this is if the patient is still anticoagulated. Control will take a bit of time and require pressure, which the VAC will not provide. Therefore in these cases, consider packing and dressing that wound, then transition to a VAC the next day. Assuming the embolectomy goes well and the foot is well-perfused, I usually leave the VAC on for 3 to 4 days, depending on patient’s condition. If the patient does not have a fever spike, no redness is seen on the leg, and nothing concerns me, then I will remove the VAC and take a good look at the wound. This can usually be done at the bedside with a little bit of sedation and pain medications. If things look good, close the wound. If the patient has a graft, I may close one incision primarily (where the graft is or in the vicinity of the graft), and the second incision may either be delayed or require skin grafting at some point.
Peripheral Arterial Disease: Popliteal Aneurysm Treatment

For the treatment of popliteal aneurysms, open is still preferred over stent. When stents were introduced, we thought that they would obviate the need for surgery. However, this has not panned out. If interested in reading more about this, Peter Gloviczki and Ying Huang wrote a brave commentary in the Journal of Endovascular Therapy in which they say that endovascular repair of popliteal artery aneurysms is not yet ready for prime time. (Resource: J Endovasc Ther. 2015; 22 [June]: 338-40.) If a popliteal artery aneurysm has thrombosed, the major amputation rate, even in great referral centers, is high (15% to 20%). In a large series from the Mayo Clinic that assessed vein bypass for patients that were not acutely thrombosed, the 5-year limb salvage rate was 100% and the secondary patency rate was 94%. Those are incredible numbers. Stenting today cannot match that.

Popliteal Artery: When the knee bends, the popliteal artery does not stay tight to the bone making almost that right angle. Instead, it goes the other direction — it falls away from the tibia so that it is 3 to 4 inches behind that tibial plateau. It is a different set of stress and strain that people have recognized. As a result, newer stents are available that specifically address this, such as the Supera stent that has more radial force. However, the patency rate is still not as good as what the Mayo Clinic published >10 years ago. The meta-analyses of endovascular repairs in patients with great anatomy indicate that the primary patency rate is only 69% and the secondary patency rate is 77%, which is not as good as that achieved via an open procedure. However, some patients who are at high risk for open repair, and in those patients, the surgeon must have a landing zone: a normal segment of the distal superficial femoral artery or proximal popliteal artery where the stent can be seated. The same thing is also needed downstream.

Mesenteric Ischemia: 4 Major Types and General Workup

Surgeons generally encounter four types of mesenteric ischemia.

Chronic Occlusive Disease: Mesenteric ischemia can result from chronic occlusive disease, which is generally atherosclerotic. These patients are symptomatic and may not present acutely. They have the classic history of painful abdomen after eating, development of food fear, and weight loss. Patients are usually female who are often interpreted as “crazy” for a significant number of months before they actually get the care they need.

Compressive Disease: Another subgroup of patients with mesenteric ischemia is relatively young and has median arcuate ligament syndrome, where there is compression of the vessel with the ligament across the celiac. Most of these patients who are referred to me are definitely called “crazy” before they actually get stumbled to a diagnosis.

Acute Occlusive Disease: The older patient with chronic atherosclerotic disease can acutely thrombose. Their history sounds deceptively embolic. They tell you, “Oh, I was watching the 10 o’clock news and suddenly, boom, worse abdominal pain I ever had in my life.” An acute thrombotic process can be almost as bad. The clue is when you find other stigmata of significant atherosclerotic disease or when you look at their plain x-ray and see the calcification outlining their vessels.

Nonocclusive Disease: Nonocclusive mesenteric ischemia can occur via venous thrombosis or profound vasoconstriction. In patients with inflammatory bowel disease or after liver transplantation, venous thrombosis occurs, causing sufficient congestion to impair arterial and splanchnic blood flow. These patients will develop general GI malaise, distention, etc. A separate subset of patients is in the ICU due to a complicated myocardial infarction, bad congestive heart failure, or coming off a coronary artery bypass graft with a bad pump run with a low cardiac index. These patients may develop nonocclusive mesenteric ischemia due to profound vasoconstriction that does not allow sufficient blood flow to the intestines.

Diagnostic Workup: Almost all these patients undergo CT angiography before being referred to me. Occasionally, their creatinine is elevated, so no contrast is used. With a noncontrast CT, calcification can be
a clue for mesenteric ischemia. Mesenteric duplex US is not very helpful in this setting because most patients have bowel gas distention. Most of us, at this point, would say, “Let’s do an angio.” We hydrate the patient, which does work, and administer N-acetylcysteine, which has not been proven in the literature to work but is part of the routine at our institution for patients with an elevated creatinine. If I can get access, I get a wire up and I’ll set up for my first picture to be a lateral at about L1-T12. With the right table settings, I can usually get from L1 down to L2; I can go a single squirt of contrast with 10 to 20 mL; and I can see what comes off the aorta. This method greatly reduces the amount of contrast used.

Mesenteric Ischemia: Current Management Approach

How do we best manage the patient with mesenteric ischemia due to either an acute embolic event or occlusive disease related to in situ thrombosis. In the past, we have approached these in the following sequence: laparotomy, direct evaluation, and revascularization. However, a switch appears to have occurred so that the sequence is now angiography, endovascular treatment, and then laparotomy to look for dead bowel. In my opinion, it is not up to the surgeon to dictate the right sequence. Instead, the patient’s situation dictates the right sequence. For example, patients with nonocclusive mesenteric ischemia are distinguished by their history of a bad heart, failure on multiple pressors, postop myocardial infarction, etc. The advantage of angiography in someone with this history is that you can give vasodilators through that angiocatheter at that time. If, however, this patient has an incredibly high lactate, a base deficit, a twenty-thousand white count, you need to do something open as well because that bowel is beyond unhappy and is probably dead. Therefore, while you want to do that angiogram, ideally you want to be in an environment where you have the hybrid room where you can take them down, stick in the catheter, get those images, start your infusion, and then open them up. Many places do not have this luxury. OR availability and delay awaiting for arrival of the angio team, and other factors come into play, and I am not convinced that we can make an edict that there is a single right way to do this. You must first determine which type of mesenteric ischemia your patient has before deciding how to proceed. For example, the mortality for patients with nonocclusive mesenteric ischemia is approximately 70%, so for them, I might just resuscitate, get them ready as quickly as possible, do a small laparotomy, and then revascularize. I am not sure I would use a vasodilator infusion — we do not do that very much anymore. We must also realize that management is resource-dependent. Nonetheless, the current emphasis on managing mesenteric ischemia that you think needs revascularization is to do the revascularization first and then to do the laparotomy.

Dusky Bowel: The consistent knowledge deficit that bothers me most is that of managing the dusky bowel. We did a mock orals exercise for our residents during which they were asked about a patient who had an embolus that was dealt with, and they were left with dusky bowel. I was amazed by the number of young surgeons who said, “Oh, it’s been fixed. We’re going to overlook that and put them back together.” In truth, you need to give dusky bowl a little time to declare itself. You can bring the patient back for a second look and even a third look, if necessary. You do not deal with marginal bowel until you know what it is going to do.

Abdominal Aortic Aneurysms: Endovascular Treatment and Follow-Up

The management of abdominal aortic aneurysms seems to have drifted out of the realm of the general surgeon. Is endovascular repair the best way to manage these today? Endovascular repair has its advantages. You need to consider your patient, what device is being used, and who is doing the repair. For about 10 years, we had three main devices that we used. Now, other companies are developing newer types of grafts that have advantages based on theories, anatomy, and the way they set up. We know from the Veterans Affairs trial that endovascular repair is durable and efficacious, but it is also
associated with an increased incidence of secondary long-term interventions. However, their rupture-free survival rate is as good as that of open management. Open results in more acute complications: a bigger incision, longer recovery, and a greater incidence of heart attacks. Complicated anatomy is still causing issues. The required neck length (area below the renal arteries where you must seal an endovascular stent graft) used to be 2 cm. We are now down to 1.5 cm, and some available devices are saying 1 cm. These work by providing additional prongs going up into the more proximal aorta. Other devices look at that shorter neck and say that the answer is to rest the graft down on the aortic bifurcation and build it up from there, reducing the chance of the graft “falling down” into the aorta and losing that hook or that seal on the neck. The biggest challenge we have right now is patient anatomy, including short neck, small vessels, extensive calcification, and accessory aneurysms in the hypogastrics or extending down the iliacs.

**Follow-Up:** For the patient with an endovascular aortic repair, pressure sensors that detect leaks can be implanted for follow-up. However, I have no experience with these. Classic follow-up for these patients is a CT at 1 month after the procedure, then again at 6 months and yearly thereafter. That is a lot of radiation exposure in the long-term. The radiation dose of a CT cardiac angiogram is 16 times more than what you would get in an ambient year. If these patients are going to live a minimum of 10 years, then we are blasting them with radiation. Now, the radiation dose must be recorded in the patient’s chart every time they get a CT. My current follow-up protocol begins with a CT scan at 6 months. If I can demonstrate sac shrinkage at 6 months, then I put them out a year. At that year, I will get an US at the same time as the CT because measurements between CT and US do not always correspond equally. This way, I have a reference point, so I know where I am at and then I will follow with US thereafter.

**Arteriovenous Fistulas for Dialysis: DRIL Procedure and Managing Pseudoaneurysms**

In chronic renal failure patients with arteriovenous fistulas (AVFs), we sometimes talk about the DRIL procedure (distal revascularization and interval ligation). Our group has five vascular surgeons in three hospitals, and we have not done a DRIL procedure in 3 years. It is indicated in isolated circumstances. The DRIL procedure is needed because seal from the graft or fistula requires distal revascularization to resolve the extremity ischemia while trying to salvage the hemodialysis access. These are not easy procedures. **Pseudoaneurysms:** A dialysis patient with a forearm prosthetic loop graft has been doing great with no complications for several years. He refers back to you because he has developed a very large pseudoaneurysm in one limb of the graft. It is pulsatile and the skin is thinning over it. How do you manage that? Two different techniques are currently used for dialysis access grafts. (1) The stepladder technique involves selecting a distal point and you march up like the steps on a stepladder to a more proximal place. When you reach the “top,” then you start over. With this technique, you access different spots all the time. (2) With the buttonhole technique, you basically access the same spot on the graft every time the patient undergoes dialysis. This technique is largely responsible for the pseudoaneurysm complication described above. If the pseudoaneurysm is not infected and if it is large with thinning skin, you have a chance to revise it. Do this by removing the graft above and below the anastomosis and running a secondary loop around it, if the arm is large enough and you have an area that will permit this. When I open these, and the ones I open directly are usually because of infectious complications, I often find that there is no graft at all. They have stuck it often enough in the same spot that there is nothing in the place where you should see the polytetrafluoroethylene (PTFE) graft. This is one of the bigger challenges of functioning accesses. I repair these in two stages. First, I bypass around the aneurysmal area in the forearm, covering this with something like OPSITE® or Ioban™ because I do not want that aneurysm in my field. I make two separate clean incisions: sew PTFE to PTFE twice and go around the area. Then I use Dermabond® to close that, cover it, and then I open and drain that pseudoaneurysm.
Venous Disease: Venostasis Ulcers and Symptomatic Varicosities

For venous ulcers, Unna boots continue to work great. However, some patients have so much drainage that you would be changing this every 24 hours, so you cannot justify the expense when they do not have insurance. In these cases, we also a “poor man’s Unna boot,” which uses some kind of absorptive dressing directly on top of the wound. Then you wrap them with Kerlix™, and then we use Coban™. We can change this every 12 hours if we needed. I have used medical-grade honey for venous ulcers. While I think it is a good product, I do not think it is the single thing that you should put on any type of wound. I have had wounds that respond well with good debridement and some kind of foam. I have had others that, while it’s not infected, we think there is enough biofilm that needs to be controlled. New products are available, like PuraPly™ that has all this polyhexamethylene biguanide (PHMB) which knocks down those biofilms. Because I am in a clinic setting where I have a couple of medical assistants that take the bandages down, my biggest rule on dealing with venostasis ulcers is that “she who pulls down the dirty, stinking bandage has input as to what goes on next.”

Symptomatic Varicosities: The management of symptomatic varicosities has completely moved into the minimally invasive arena. We are dealing with these mainly via endovenous laser treatment (EVLT) to remove the veins. We will bring them back for stab phlebectomy on isolated areas. I do see some that have very limited reflux at the saphenofemoral junction, and for these, a very short incision with disconnection of the greater saphenous and all its branches from the deep system, is enough. You must look at what your reflux studies show. Because we are the center that we are, another problem we have is we get patients whose veins came back at 10 years after EVLT. They have more visible tributaries, and these patients are just shocked that they did not stay gone for forever. Of course, these patients have not been compliant with wearing support stockings, which is a requirement after EVLT.

Venous Disease: Iliofemoral Vein Thrombosis

Management of iliofemoral vein thrombosis (IFVT) has undergone a dramatic change now that endovascular techniques are available. Nonetheless, heparin is still a very good drug that must to be used in these patients. The question is, are you willing to accept a higher risk of bleeding and complications from thrombolysis weighed against the risk of long-term post-phlebectomy syndrome in that extremity? We know that the patient is more likely to develop a post-phlebectomy syndrome if it was an iliofemoral clot than if it was a deep femoral system. If you can clear the iliofemoral system, you can decrease reflux and symptoms by 20% to 40%, although the data on this are weak. If a patient has true phlegmasia in the threatened limb, that absolutely should be done. But the question is what to do with just a large leg in the hospital that has iliofemoral clot. Usually when I’m consulted on those patients, they are in the hospital and the clot occurred after a surgical procedure. Typically the surgical procedure is one of those worrisome contraindications, such as a craniotomy or major trauma. These are not really patients to whom I want to give thrombolytics. This includes direct infusion (low dose, catheter-directed into the clot), which supposedly has no systemic impact. According to published trials, the average amount of time that the drug is infused is 53 hours. I find it difficult to believe that direct infusion does not cause a systemic effect after 2 days of running tissue plasminogen activator (TPA). Even if the systemic coagulation profile does not change, I remain worried. Some surgeons use the mechanical thrombectomy devices. They first try the mechanical device to get some flow, and then they put the small catheter in for direct infusion. About every 12 hours, they will check the patient by moving them from the unit to IR. Patients generally get an IVC filter as part of this protocol.