There is no doubt that the superficial femoral artery (SFA) is a very troublesome vessel to treat due to the many forces acting upon it throughout the course of a normal day. Compared to some of the more “predictable” arteries, the mid- to long-term outcomes we observe in the SFA are not always as favorable as those we have come to expect in other vessel beds. As a result, some skepticism has recently been directed toward the technologies we are using to treat SFA disease. It is important, however, to recognize that numerous factors impact patient outcomes, and we must not assume that the most obvious (ie, visually noticeable) potential reason for an unfavorable outcome is the factor that caused it.

FACTORS LEADING TO FRACTURES
The incidence of stent fractures, especially in the SFA, has received a great deal of attention lately, with several major studies and increasing anecdotal evidence showing that fractures do indeed happen. As clinicians, it is incumbent on us to determine both what causes these fractures and also how they do or do not impact clinical outcomes. A popular saying is that a good craftsman never blames his tools; although we have come to expect excellence in the design, engineering, and manufacture of the devices we use, it is unreasonable for us to expect that they will never break, especially when they are not always used in a “textbook” fashion, as is often required when treating vascular disease.

My intention is not to let technologies and the companies that manufacture them off the hook for negative outcomes associated with the procedures in which they are used. If a device’s design is determined to be the cause of its failure, and that failure has the potential to outweigh the efficacy of a successful treatment, the manufacturer should be held accountable and the device removed from trials or the marketplace. However, the

Figure 1. Although a stent fracture has occurred (arrow), the patency is better in this area than in the intact portion.
treat ing physician needs to recognize two significant things about every stent fracture: (1) if a fracture occurs, it is not necessarily due to flawed device design, or the wrong material, etc.; and (2) many stent fractures do not result in negative clinical outcomes.

To briefly illustrate the first point, I ask you to consider this: What happens when you place a 6-mm balloon-expandable stent, use a 6-mm balloon, and you oversize the inflation pressure by 20%? How would the metal surface area be affected? What might this do to a stent strut? Most if not all interventionists will make slight errors (or perhaps judgment calls due to patient anatomical demands) such as this from time to time, and we cannot expect that a technology’s design and material will be strong enough to compensate for all such occurrences, yet be nimble enough to twist, turn, shorten, and lengthen with the SFA.

The second point is perhaps the most important consideration when evaluating the clinical significance or impact of stent fractures. After clinical studies such as the landmark SIROCCO trials began to show evidence of stent fractures, investigators responsibly sought to first determine what caused the fractures and whether they were associated with an increased incidence of negative outcomes, particularly restenosis.

WHAT DO THE DATA SAY?

Although the SIROCCO trials did not demonstrate superiority of drug-eluting stents over bare-metal stents, and the incidence of stent fracture received a lot of attention, the overall outcomes were actually quite encouraging. First, the results in the bare-metal arm were better than anyone expected; second, the investigators looked further than just duplex results, focusing much attention on Rutherford classification. They found that the vast majority of these patients, who had significant claudication at baseline reported minimal to no claudication at 2 years. Claudication reporting can, of course, be biased, but very good postprocedural ankle-brachial indices were also maintained at 2 years. Clearly, this represents a significant improvement for the patient. The patients were clinically doing well, and there was no concrete relationship shown between fracture and restenosis.

At the same time, our center was participating in the five-center BLASTER study of stenting in significant SFA disease. BLASTER was a randomized trial between stenting with and without abciximab. All lesions included were at least 7 cm long, unless the patient had a total occlusion. Approximately half of the patients involved in the study presented with total occlusions, and the mean lesion length was almost 12 cm. The duplex restenosis rate was 22% over the entire population at 9-month follow-up, but the assisted primary patency was excellent, at 97.6%.

Like our colleagues in the SIROCCO trials, we also wanted to look at Rutherford classification as an indication of outcome. We were encouraged to see that these patients who had significant claudication at baseline had very mild disease process at 9 months, with 88% improving by at least one Rutherford class and almost 75% being either asymptomatic or having only mild symptoms. We even went so far as to evaluate the patients on a treadmill to show increased ambulatory ability. At 9-month follow-up, we were not looking at stent fracture data, because frankly, we did not yet know they fractured. We had seen a few tine fractures, but we did not have any reason to think they were clinically relevant. The concrete patient data and outcomes we saw were favorable, regardless of whether or not there were fractures.

We have recently started collecting long-term data from the BLASTER trial. So far, we have reviewed nearly 4 years of follow-up data from 19 of the 27 (70%) of our center’s original patients; three refused follow up and five died. The patency rate we have observed is approximately 50% at 4 years, which is close to what might be expected from a PTFE bypass graft. We saw stent fractures in 26% of these cases, confirming that they are definitely a concern to be monitored. However, because there was a 50% patency rate and only a 26% rate of stent fracture, in this group, patients were more likely to have restenosis without a stent fracture than with a stent fracture.

Based on these and other clinical observations, it remains to be seen what the clinical impact of stent fractures is. Clearly, we should work toward developing technology that is as resistant to fracture as possible while maintaining the other properties necessary to maintain acceptable patency over a long period.

Many people naturally believe that a stent with a lower fracture rate will outperform one with a higher rate. We must proceed carefully when considering this assumption, however, because we have not seen concrete data to support it. An interesting set of data was recently published by the ASSURANT trial investigators. At 1 year, the reported rate of stent fracture was only about 1.5%. It is reasonable to believe that stent fracture rates may never be lower than this. However, although the stenting arm outperformed the angioplasty group, the restenosis rates are not better than what we have seen in some of the other nitinol stent trials. It is difficult and even misleading to compare results from trials that had different inclusion criteria and especially trials that were not randomized, but it is important to (Continued on page 52)
note that a look at their results at similar endpoints indicates that the hypothesis that fewer stent fractures translates into clinical benefits is at least still in question.

THE ELEPHANT IN THE ROOM

One clear truth in the discussion of stent fractures is that not all stents are created equal. For the purposes of this debate, I would rather not focus on which devices are associated with the highest fracture rate or the lowest patency rates; Biamino and Scheinert have shown this in their work, and any interventionist who has worked extensively with stents will tell you that there is a sizeable difference in the quality of stents that have been evaluated. The important thing to consider is that the incidence of stent fracture cannot be looked at as something that affects all stents equally, and as such, it should be evaluated on a device-by-device basis.

From the standpoint of clinical investigators and industry, one of our greatest and most important challenges is to determine why fractures on one stent may be shown to have more significant clinical impact than fractures on another. With this understanding, we can hopefully design next-generation devices with both lower fracture rates and, more importantly, stents that have less impact when they do fracture.

MAINTAINING A BROAD FOCUS

In conclusion, it has been shown that stents fracture more than we would like, and that this trend is greater in some devices than in others. Additionally, some stent fractures stimulate intimal hyperplasia in some patients, whereas others do not, regardless of device platform. We have even seen restenosis in stents that have fractured, but the location of the fracture was actually the best area of the stent in terms of intimal hyperplasia (Figure 1). This does not mean that stent fractures are insignificant; it simply means that there are a number of factors impacting outcome, and we should not focus our attention solely on stent fractures—or any other single factor—or we will miss the opportunity to determine and correct the others.

Gary M. Ansel, MD, is Clinical Director for Peripheral Vascular Intervention, MidOhio Cardiology and Vascular Consultants, MidWest Research Foundation, Riverside Methodist Hospital, Columbus, Ohio. No specific products or manufacturers are mentioned in this article; however, Dr. Ansel has disclosed that he has an affiliation with or financial interest in several companies that produce stenting devices. Dr. Ansel may be reached at (614) 262-6772; gansel@mocvc.com.