Peripheral vascular disease currently affects as many as 10 million people in the U.S.\(^1\). Endovascular techniques are slowly but progressively replacing traditional surgical procedures for the treating patients with peripheral vascular disease. Using subintimal techniques and new devices, crossing long total occlusions has become routine. The use of newer nitinol stent designs frequently produces an acute result that looks almost normal. What has kept superficial femoral artery (SFA) and popliteal endovascular therapy from becoming the clear standard of care has been the long-term results.

Restenosis rates for stenting in most vascular trees are in the acceptable <10% to 15% range.\(^2,3\) Generally, when restenosis occurs, it is a single event and can be managed endovascularly. The one arterial tree in which long-term restenosis rates are in the 40% to 50% range, and in which restenosis is difficult to treat, has been the infrainguinal vessels.\(^4,5\) Many factors contribute to this problem. The length of lesions to be treated is often in excess of 17 cm to 20 cm.\(^6,7\) The SFA and popliteal arteries are long, with relatively few branches. They have a tendency to develop diffuse, severe atherosclerosis and often occlude for long distances.\(^8,9\) These arteries are also unique in that they move dramatically in multiple planes during limb motion.\(^10,11\) The arteries not only compress, bend, and rotate, but they also shorten and extend in response to movement.
LEG BEND ROUTINE AND INITIAL OBSERVATIONS

In our lab, the routine is to perform an on-table leg bend test of approximately 90° both before and after PTA when dealing with longer areas of disease, as commonly seen in TASC C and D disease. Using this test, we have learned a great deal about the variability in location of significant arterial angles that occur when the leg is bent. We have also noticed substantial differences in severity of arterial angulations occurring among individuals. In many patients, there is only a gentle bend starting at the adductor canal and ending just above the joint space at the knee. In other patients, the distal portion of the SFA and proximal-to-mid popliteal artery will bend acutely in more than one location.

We were surprised by the amount of compression and elongation that the SFA and popliteal artery undergo with limb motion. Measurements in our lab indicate that these larger infrainguinal vessels may lengthen and shorten by as much as 15% between the bent and straight limb positions (Figure 1). Because the length of stented artery in routine SFA and popliteal angioplasty is often close to 20 cm, a 15% change in length would result in a difference of 3 cm. Repeated changes in arterial length of this magnitude occurring with limb motion would likely place significant stresses on presently available nitinol stents. These nitinol stents are variable in their compliance to extension and compression. Generally, they all lengthen and shorten poorly when compared to the natural movement of the SFA during limb bending.

Stent designs have changed over the years. In the early Wallstent (Boston Scientific Corporation, Natick, MA) data, SFA reocclusion rates were up to 70% at 3 years. The early Wallstent was a device with weak resistance to compression, partially because it reacted to stresses placed on it by spreading those forces across the stent. If Wallstents are compressed in the middle, the ends move outward. In mobile vessels, such as the SFA and popliteal, the motion of that generation of Wallstent against the internal surface of the artery would act like sandpaper, traumatizing it from the inside. In contrast, current-generation nitinol stents have significantly improved resistance to compression and do not move laterally when squeezed in the middle. As mentioned previously, they are still relatively noncompliant in their long axis.

These stents would also be expected to fit poorly in arteries that compress, rotate, bend, shorten, and extend often, such as the SFA and popliteal. The repeated trauma to the artery caused by the negative interaction of relatively rigid stents against a more fluid artery would not be expected to be as severe as with the early Wallstents, but would still likely occur. Long-term data on SFA stenting appear to support the theory of chronic cumulative trauma creating restenosis. Mewissen showed Smart stent (Cordis Corporation, a Johnson & Johnson company, Miami, FL) primary patency in real-world SFA/popliteal disease was 76% at 1 year, and 60% by the 2-year mark, which is similar to other trials. The few trials following SFA/popliteal stenting to 3 years and beyond show further decreases in primary patency. No nitinol stent presently on the market supports the SFA/popliteal arteries adequately without distorting its architecture during motion.

LESSONS LEARNED FROM LEG BEND TESTING

As a result of present-generation nitinol stent designs, unique challenges face the interventionist performing long SFA and popliteal angioplasty. The answer to the question of how to achieve a satisfactory acute result that will not clinically restenose has eluded peripheral
There is no clear solution for this problem. On-table leg bend testing to approximately 90º before and after PTA helps us to avoid placing stents in areas of significant bend. What we have found is this only solves one problem. By not placing long stents in regions of substantial arterial bend, some restenosis and acute occlusion can be averted by avoiding traumatic compression of the normal artery against the distal end of the stent (Figure 2). Other more subtle issues were also noted.20 One of the most obvious problems we encountered was excessive arterial slack developing in the distal SFA/popliteal arteries when the stented limb was bent. The excessive slack or bunching of the artery distal to the stented region was more likely when stenting of the proximal and mid-SFA had been performed. We believe this bunching of the distal SFA/popliteal arteries occurs because the arteries are forced to achieve their natural lengthwise compression using much less artery. The stented regions in the proximal and mid-SFA are resistant to shortening because the stents that line them have poor shortening characteristics. The bending lower leg forces the popliteal and distal SFA proximal against the end of the stented region, creating kink. This iatrogenic redundancy is sometimes associated with decreased flow when the limb is bent (Figure 3A-C).

When severe enough to require therapy, our practice is to use stents that are very compliant to elongation and shortening, such as the IntraCoil (ev3, Inc., Minneapolis, MN) or the Viabahn (W.L. Gore & Associates, Flagstaff, AZ) depending on whether important collaterals exist in the area. We place the more compliant stents at the distal end of the nitinol stents, ending several centimeters into the angiographically more normal artery. Compliant stents act as a transition zone between the nitinol stents and the distal artery, allowing shortening but decreasing kinking and maintaining flow. We are presently following patients treated with this strategy to determine if it improves long-term patency rates.

**CONCLUSION**

Stenting in the SFA and popliteal arteries provides a safe, consistent, and visually pleasing result during endovascular intervention. Thus far, it has not fulfilled the promise of long-term patency or freedom from rein-
tension. Sadly, long-term (>5 years) patency may be more common with PTA only than with stenting.\textsuperscript{1,2,13,19,21,22} We believe much of the long-term restenosis is due to chronic low-level negative interaction between stents and the arterial wall. This cumulative trauma occurs as a result of stent designs that do not allow the artery to move naturally during limb motion, especially during extension and contraction. Just like restenosis in the coronary tree, a problem that once seemed insurmountable, the difficulties of SFA and popliteal intervention will one day be solved. We need only the right stent design.

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