Percutaneous interventional treatments for superficial femoral artery (SFA) disease have long suffered from excessively high restenosis rates regardless of treatment with PTA alone or with stenting. Earlier SFA stent studies reported 1-year patency rates between 29% and 68% using the Wallstent1-3 (Boston Scientific Corporation, Natick, MA) and >50% with use of the Palmaz stent4 (Cordis Endovascular, a Johnson & Johnson company, Miami, FL). More recent SFA stent reports using self-expanding nitinol designs have shown improved 1- to 2-year primary and secondary patency rates, but secondary reintervention rates for occlusion or in-stent restenosis (ISR) remain high (20%-30% at 1 year).5,6 Longer, more complex TransAtlantic Inter-Society Consensus (TASC) type B, C, and D SFA lesions remain especially problematic, raising concerns regarding current SFA stent technology. The poor performance of early SFA stents and the complexity of the SFA have prompted the development of new stent designs, local drug delivery systems, and several new plaque debulking technologies and techniques.

In the SIROCCO trial, the only evaluation of drug-eluting stent performance in the SFA, reported no significant 6- to 12-month SFA ISR when using sirolimus-coated (Rapamycin, Wyeth-Ayerst Laboratories, St. Paul, MN) Smart stents (Cordis Endovascular) in phase I and II studies.7 The mean percent diameter stenosis was 22.6% in the sirolimus group versus 30.9% in the bare-metal group (P=.294). Recent 24-month SIROCCO follow-up disappointingly reported a significant (40%) binary ISR rate in the sirolimus group and a 47% in-stent restenosis rate in the bare-metal group. Interestingly, for the first time, nitinol stent fractures were reported in 6 of 33 (18.1%) in the SIROCCO phase I study. The SIROCCO phase II study encouraged single stent use only and reported three additional nitinol stent fractures.

In these studies, nitinol stent fractures were reported to be clinically asymptomatic and, in the phase I report, were attributed to the placement of multiple long, overlapping stents leading to splinting of the SFA at the overlaps, with the creation of abnormal hinge points. In

---

**Figure 1.** A series of images depicting the dynamic forces affecting the SFA. Note the severe torsional stent deformation at 180° twist (A). Note the multiple abnormal hinge points as the SFA attempts to elongate and contract (B). Type III nitinol stent fracture illustrating severe stent deformation several millimeters above a stent overlap. The overlap is identified at the most distal aspect of these images (C). Post SFA stent intravascular ultrasound with the knee flexed. Note the stent strut crush-like deformation (D).
the later phase II series, multiple stents and overlaps were discouraged, with a resultant decrease in nitinol stent fractures; again, these fractures were considered benign. It was not reported whether these fractures were associated with the high ISR rate reported at 24 months. Ansel et al.8 have recently cautioned that nitinol stent fractures may be of concern and note that the long-term clinical significance of these fractures remains unknown.

**STENT FRACTURES**

Stent fractures have been reported with balloon-expandable, stainless steel stents and Wallstents, and nitinol stent fractures have been reported in a variety of clinical settings, including the esophagus, biliary tract, congenital heart disease, dialysis grafts, coronary arteries, iliac arteries, popliteal arteries, and subclavian arteries and veins.9-14 Meir et al.15 and Phipp et al.16 reported a >50% stent fracture rate with subclavian vein stents placed for Paget-Schroder syndrome or exertional axillosubclavian vein thrombosis, and recommend stenting cautiously and only after first rib resection. Multiple stent fractures are reported with aortic endografts for AAA repair, and they can be associated with clinical sequelae. Babalik et al.18 reported a complete transverse linear nitinol stent fracture in the popliteal artery at 6 months after stent deployment presenting with acute occlusion requiring surgery for limb salvage. Kroger et al.19 have reported three cases of popliteal stent fracture; all were symptomatic and required reintervention.

Coronary artery stent fractures are thought to be rare, but recently, a medical device agency warning was released reporting nine cases of radial fractures of NIRoyal coronary stents (Boston Scientific Corporation) occurring between 3 and 9 months after implantation.20 All nine were symptomatic and associated with ISR (6 of 19) or occlusion (3 of 9). Recently, Sianos21 reported two cases of treatment failure resulting from stent fracture using the sirolimus-eluting coronary stent. A review of the existing stent fracture literature has consistently described clinical sequelae related to the fracture in all reports, except the SIROCCO SFA nitinol stent fracture reports by Duda et al.7

**Figure 2. Nitinol stent fracture classification.**

Type I = a single strut fracture only (A). Type II = multiple single nitinol stent fractures that can occur at different sites (B). Type III = multiple nitinol stent fractures resulting in complete transverse linear fracture but without stent displacement (C). Type IV = a complete transverse linear type III fracture with stent displacement (D).

**SFA DISEASE CHARACTERISTICS**

Several unique characteristics of SFA disease have been linked to the poor long-term patency rates reported with SFA PTA/stenting as compared to the >90% long-term patency rates in coronary and aortoiliac vessels. SFA disease is characterized by a high incidence of long complex diffuse disease, calcification, and total occlusions, and therefore a high atherosclerotic plaque burden. The resultant SFA mean luminal diameter is often very small (<2 mm to 3 mm), with flow rates significantly lower than flow rates for coronary and aortoiliac arteries. Theoretically, this sluggish flow environment would favor platelet deposition and its negative sequelae. Multilevel disease with poor infrapopliteal runoff has consistently been associated with poor surgical and endovascular results. Historically, classical intimal hyperplasia has been thought to be the etiology of the high SFA ISR rates. Only recently have other factors, such as external and internal biomechanical forces, micro and macro stent design issues, and now stent fracture, been considered as additional factors influencing failure modes with SFA stenting.

It is now clear that the SFA is a very different, dynamic vessel, unlike any other artery in the body. Flexion, torsion, compression, and elongation are but a few dynamic biomechanical forces affecting the SFA that may influence clinical results and pose a unique chal-
Challenges to endovascular therapy (Figure 1A). As the SFA traverses the adductor canal, a particularly complex combination of forces come into play, including external compression, torsion, elongation, and flexion. It is probable that more negative forces are exerted on the SFA at that point than more distal in the popliteal space. It is easy to understand why the distal SFA, after years of exposure to these repetitive negative forces, is the earliest and most often diseased and calcified SFA segment. Henry et al. reported a high incidence of distal SFA restenosis, especially if associated with stent overlap. Our experience has been consistent with the report by Henry.4

The Dynamic Free-Floating SFA

A simple anatomic analogy between the SFA and descending thoracic aorta may add insight into understanding the dynamics of the SFA and its pathophysiology as related to stenting. The transverse aortic arch is fixed, or tethered down, by large innominate and subclavian branches; likewise, the distal thoracic aorta is tethered down by the renal and visceral vessels and the diaphragm. Because the descending thoracic aorta has no major branches, a relatively mobile, free-floating descending thoracic aorta occurs between these two fixed points. This results in the descending thoracic aorta being very susceptible to dynamic forces and makes the descending thoracic aorta susceptible to traumatic aortic injury (torn aorta), which occurs just distal to the left subclavian artery after acceleration and deceleration accidents. The iliac and common femoral arteries are tethered to the pelvis and groin by large hypogastric branches and the profunda-proximal SFA bifurcation. The infrainguinal femoropopliteal artery does not become tethered again until the below-the-knee trifurcation, resulting in the SFA being free-floating, mobile, and dynamic, especially considering the motion and forces exerted at the hip and knee joint. It is reasonable to assume the SFA must perform like a rubber band, and it is likely that elongation and contraction of the SFA play a significant role in the failure mode of SFA stents (Figure 1B). Repetitive elongation of the SFA after stenting could result in stent fracture by creating abnormal metal-to-metal hinge points by a piston-like effect, especially at multiple stent overlap sites (Figure 1C).

Our Experience

Our group developed an interventional first treatment approach to SFA disease in the mid-1990s, and our SFA experience involves most interventional technology and stent designs developed since that time. Nitinol stent fractures were increasingly identified over time, prompting a nonrandomized, retrospective clinical, chart, and angiographic analysis of our nitinol stent fracture experience. Between January 2000 and July 2003, 380 patients were identified as having been treated using SFA nitinol stents. One hundred ten of 380 patients (28.9%) underwent repeat angiography and nitinol stent fracture analysis using 5-inch cine fluoroscopy magnification. The Cardiovascular Institute of the South (CIS) nitinol stent fracture classification system was developed for nitinol

Figure 3. Twenty-four-month nitinol stent fracture progression from type II to type IV. Note the irregular stent expansion at 6 months (A). Asymptomatic 1-year type III nitinol stent fracture (now transverse linear fractures without displacement). Note <50% ISR by mean luminal diameters (B). Twenty-four-month symptomatic (pseudoaneurysm) type IV stent fracture (transverse linear fracture with displacement) (C, D). Six-month angiogram after the percutaneous placement of a 6-mm Viabahn (Hemobahn, W.L. Gore & Associates, Flagstaff, AZ) covered stent (E, F).
stent fracture standardization (Figure 2). The study variables analyzed included clinical symptoms, angiographic restenosis (<50% or >50%) or occlusion, number and location of stents deployed, lesion length, and nitinol stent fracture classification. The nitinol stent fracture analysis included a correlation of the previously noted variables (Tables 1 and 2).

Analysis of Results
One hundred ten of 380 (28.9%) SFA-stented patients underwent repeat angiography during the study period. A nitinol stent fracture was identified in 72 of 110 patients (65.4%) in this angiographically selected cohort of SFA-stented patients. The average lesion length was 17.5 cm (range, 6-34 cm) and the mean time to angiography was 11 months (range, 2-34 months). The mean number of stents per case was 2.3 (range, 1-5 stents). The indications for repeat angiography and fluoroscopy were not analyzed because many of these extremities were restudied during other staged peripheral vascular disease revascularization procedures (ie, renal, celiac, contralateral extremity). During this 43-month period, multiple stent designs, lengths, and generations were deployed by several operators, oftentimes using differing techniques. Additionally, multiple stent types and designs from different members of industry were sometimes used in the same patient, especially in long, complex lesions in limb-salvage cases. Therefore, a detailed nitinol stent fracture analysis or comparison between each individual nitinol stent design would not have been reliable and was not performed.

Fifty-nine of 72 (81.9%) nitinol stent fractures were type I and II, but they were increasingly associated with angiographic stenosis and symptomatology. Seventeen of 27 (62.9%) type I nitinol stent fractures were associated with >50% stenosis or occlusion and 7 of 27 (26%) were symptomatic. Twenty-six of 32 (81.2%) type II nitinol stent fractures were associated with >50% stenosis and occlusions and 15 of 32 (47%) were symptomatic. There were fewer type III and IV nitinol stent fractures (13 of 72; 18.1%), but all were associated with significant angiographic findings and 11 of 13 (84.6%) were symptomatic.

Eighty of 110 (73%) angiographically followed SFA-stented cases had two or more overlapping stents and 38 of 110 (35%) had the entire SFA stented, representing our aggressive approach to SFA stenting. Twenty-eight of

<table>
<thead>
<tr>
<th>Nitinol Stent Fracture Classification</th>
<th>N = 72</th>
<th>%</th>
<th>&lt;50%</th>
<th>&gt;50%</th>
<th>Occlusion</th>
<th>Symptomatic (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>I</td>
<td>27</td>
<td>37.5</td>
<td>10</td>
<td>15</td>
<td>2</td>
<td>7 (26)</td>
</tr>
<tr>
<td>II</td>
<td>32</td>
<td>44.4</td>
<td>6</td>
<td>21</td>
<td>5</td>
<td>15 (47)</td>
</tr>
<tr>
<td>III</td>
<td>11</td>
<td>15.2</td>
<td>7</td>
<td>4</td>
<td>1</td>
<td>9 (82)</td>
</tr>
<tr>
<td>IV</td>
<td>2</td>
<td>2.7</td>
<td></td>
<td></td>
<td></td>
<td>2 (100)*</td>
</tr>
</tbody>
</table>

*1 pseudoaneurysm.
72 (38.8%) single overlap cases (two stents) experienced a nitinol stent fracture, with a trend toward worsening classification (type II and III). Thirty-four of 38 (89%) cases with three to five stents (so-called full metal jacket) experienced nitinol stent fractures, with a definite trend toward type III and IV classification. Eight of 11 (72.7%) type III and IV nitinol stent fractures occurred in cases with three to five stents and multiple overlaps. Single stents placed in single SFA segments were deployed in <30% of patients, but these cases also were not free of nitinol stent fractures. Ten of 30 (33%) single stents had a nitinol stent fracture—all were type I or II (Figure 2). Single stent nitinol stent fracture was not associated with location; all three segments (proximal, mid, and distal SFA) had a >20% nitinol stent fracture rate with 5 of 10 (50%) of the single distal SFA stents experiencing a nitinol stent fracture.

**OBSERVATIONS AND EXPLANATIONS**

Nitinol stent fractures occurred in a surprisingly high percentage of our SFA nitinol stent patients, especially in cases of multiple stents and overlap. This was also reported by both Henry et al.\(^4\) and Gray et al.\(^22\) In our series, nitinol stent fractures frequently occur 3 mm to 4 mm proximal and distal to the overlap (metal on metal) zone (Figure 1C). It can be surmised that an abnormal stress area is created around this abnormally rigid overlap, resulting in an abnormal hinge point or fulcrum lending to stent fracture and deformation because the SFA is inevitably affected by continuous elongation, contraction, and torsion.

In our experience, nitinol stent fracture was associated with angiographic stenosis and clinical sequelae, especially when type II to IV nitinol stent fractures occur. It is reasonable and likely that a nitinol stent fracture progresses from a simple single strut fracture (type I) to a worsening classification over an unknown and variable period of time (Figure 3). The repetitive forces acting upon the SFA and the continuing self-expanding thermal memory properties of nitinol may allow the fractured nitinol stent fracture elements to realign and even migrate. These findings have important implications for the continued use and development of nitinol stents. Further research is needed to better understand the mechanisms of nitinol stent fracture and to develop strategies to prevent and treat this complication.

**TABLE 2. NITINOL STENT FRACTURE ANALYSIS BY LOCATION**

<table>
<thead>
<tr>
<th>SFA</th>
<th>Number of Stents Used</th>
<th>Nitinol Stent Site n = 110 (%)</th>
<th>Fractures by Site n = 72 (%)</th>
<th>Classification Types</th>
<th>I</th>
<th>II</th>
<th>III</th>
<th>IV</th>
</tr>
</thead>
<tbody>
<tr>
<td>Proximal</td>
<td>1</td>
<td>7 (6)</td>
<td>2 (29)</td>
<td>1 1</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mid</td>
<td>1</td>
<td>13 (12)</td>
<td>3 (23)</td>
<td>2 1</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Distal</td>
<td>1</td>
<td>10 (9)</td>
<td>5 (50)</td>
<td>3 2</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Proximal to Mid</td>
<td>2</td>
<td>19 (17)</td>
<td>13 (69)</td>
<td>8 3 2</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mid to Distal</td>
<td>2</td>
<td>23 (21)</td>
<td>15 (65)</td>
<td>8 6 1</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Proximal Through Distal</td>
<td>3-5</td>
<td>38 (35)</td>
<td>34 (89)</td>
<td>5 19 8 2</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

*Figure 5. Symptomatic (occlusion) type IV nitinol stent fracture at 12 months after single SFA stent placement. This patient was a poor surgical candidate (A). All nitinol stent fracture elements are captured, realigned, and the occlusion crossed using a traditional Glidewire and 5-F angled Glide catheter (Terumo Medical Corporation, Somerset, NJ) (B, C). PTA and placement of a second nitinol stent (stent sandwich) were initially performed. Definitive endovascular treatment required a 6-mm Viabahn covered stent. Note the extraluminal migration of the nitinol stent fracture elements (D, E). The patient remained asymptomatic at 10 months.*
In our series, stent fractures occurred in all nitinol, bare-metal, noncoil stent designs, especially with earlier designs. Several early stents were believed to be stiff and brittle, and inefficient early stent delivery systems and aggressive post-stent balloon dilation were believed to contribute to our early nitinol stent fracture rates. Addressing these issues has likely decreased our recent stent fracture rate. Very interestingly, there were two nitinol stents in our series, both coil designs, that did not experience stent fractures. The aSpire (Vascular Architects, San Jose, CA) stent, a double helical, rigid nitinol coil design with a PTFE sock coating design, has been used since October 2001 with no stent fractures encountered in this series. A recent further analysis of our aSpire stent experience has identified 84 patients with only a single stent fracture since 2001, with 30% being overlapped. The fracture occurred in a vigorous 54-year-old male ex-football player who was treated with a single 150-cm aSpire stent for a 120-cm distal SFA-popliteal occlusion. The stent traversed the adductor canal and midpopliteal space.

The Intracoil (eV3, Plymouth, MN) is a single, open-coil nitinol stent that, to date, is the only FDA-approved femoral artery stent. No Intracoil stent fractures were identified in our series. The aSpire and Intracoil nitinol stents share several design characteristics that may make them more resistant to stent fracture. These characteristics include superior radial strength and an open coil spiral design that theoretically may allow flexibility for bending, torsion, elongation, and shortening, while providing resistance to compression. Published data have identified both of these coil-designed stents as providing resistance to compression. Published data for bending, torsion, elongation, and shortening, while coil spiral design that theoretically may allow flexibility characteristics include superior radial strength and an open coil nitinol stent platforms are so integral to cardiovascular treatment. The benefits of superelastic nickel-titanium (nitinol technology) have been well documented, but a review of the literature reveals little standardization in the processing, laser cutting, etching, electropolishing, surface finishing, and fatigue testing of nitinol stents. There exists no industry standard for in vitro stent fracture fatigue testing and, although it may be difficult to reproduce the dynamics of an SFA, it is imperative that improved standards be developed in all aspects of nitinol stent processing and testing if we are to identify the failure modes of SFA stenting. If we are to expect drug-eluting stents to have the same impact on restenosis in the SFA as they have had on percutaneous coronary intervention, then certainly the appropriate SFA stent platform, free of stent fracture, must be developed.

It appears that on both macro- and microstructural levels, all nitinol is not the same, underscoring the need for industry standardization. The failure modes of SFA stenting and nitinol stent fractures are likely multifactorial on both the macro- and microstructural levels. Microfailure behavior must be identified down to the scanning electron microscopy molecular level because it is likely that stent fractures originate as surface property defects or microcracks.

Microfailures likely progress to macrofractures and struts to continue to migrate (cut like a knife) through the vessel wall, further deforming the stent and potentially resulting in adverse clinical sequelae (Figures 4 and 5).

“In our series, stent fractures occurred in all nitinol, bare-metal, noncoil stent designs, especially with earlier designs.”

IMPLICATIONS OF NITINOL STENT FRACTURES

Any stent fracture concern would have tremendous implications to the patient, clinician, and industry because stent platforms are so integral to cardiovascular treatment. The benefits of superelastic nickel-titanium (nitinol technology) have been well documented, but a review of the literature reveals little standardization in the processing, laser cutting, etching, electropolishing, surface finishing, and fatigue testing of nitinol stents. There exists no industry standard for in vitro stent fracture fatigue testing and, although it may be difficult to reproduce the dynamics of an SFA, it is imperative that improved standards be developed in all aspects of nitinol stent processing and testing if we are to identify the failure modes of SFA stenting. If we are to expect drug-eluting stents to have the same impact on restenosis in the SFA as they have had on percutaneous coronary intervention, then certainly the appropriate SFA stent platform, free of stent fracture, must be developed.

It appears that on both macro- and microstructural levels, all nitinol is not the same, underscoring the need for industry standardization. The failure modes of SFA stenting and nitinol stent fractures are likely multifactorial on both the macro- and microstructural levels. Microfailure behavior must be identified down to the scanning electron microscopy molecular level because it is likely that stent fractures originate as surface property defects or microcracks.

Microfailures likely progress to macrofractures and struts to continue to migrate (cut like a knife) through the vessel wall, further deforming the stent and potentially resulting in adverse clinical sequelae (Figures 4 and 5).
ultimately stent deformation and clinical sequelae. Decades of research and testing has been done with stainless steel and titanium—similar resources must now be allotted to nitinol. The Stanford Research Institute and Stanford University Medical Center have recently launched a consortium with industry to quantify the biomechanical forces on SFA stents, determine failure modes, and standardize nitinol processing and testing.

“Decades of research and testing has been done with stainless steel and titanium—similar resources must now be allotted to nitinol.”

Our experiences with nitinol stent fractures and recent positive experience with plaque debulking or excision using both the 7-F SilverHawk plaque excision catheter (FoxHollow Technologies, Menlo Park, CA) and plaque photoablation using the CLIrpath laser (Spectranetics Corporation, Colorado Springs, CO) have recently influenced our interventional approach to native SFA disease and ISR. Small SFA luminal diameters and decreased mean luminal diameter after percutaneous coronary intervention have correlated with restenosis and subacute stent thrombosis. It may be that in an effort to reach maximal luminal diameters, overzealous poststent balloon dilation can result in stent fracture, especially in SFAs with a large plaque burden and highly calcified lesions. These vessels are very noncompliant, and high balloon inflation pressures after stent placement may directly fracture the stent, especially if these forces exceed the stress and strain tolerance of that particular stent. It can be theorized that significant plaque excision or debulking will render the remaining SFA more compliant, therefore allowing for better stent deployment and apposition at much lesser pressures, therefore lessening stent fracture potential. On the contrary, a strategy of not dilating the stent after implantation and allowing for vessel wall apposition to occur by the nitinol stent “growing over time” may result in a small SFA mean luminal diameter because the plaque burden oftentimes is so significant and the space so limited that the stent cannot reach its immediate maximum diameter. A long complex lesion with irregular plaquing can also result in irregular wall apposition of the nitinol stent, theoretically creating abnormal angles and hinge points that may weaken struts and promote stent fracture (Figure 3A).

We have now developed a lower threshold strategy for SFA plaque excision and ablation, with a higher threshold for SFA stenting—mainly in those areas of significant dissection or stenosis after debulking. We now perform PTA with only low-pressure inflations, and in 70% to 80% of our SFA cases we will accept the angiographic results of plaque debulking or excision alone to avoid PTA barotrauma injury to the vessel wall. A high percentage of nitinol coil-designed stents are also now being used. A particularly appealing strategy we have termed SFA stent preparation, especially at the distal SFA, is to perform maximal plaque excision followed by aSpire or Intracoil stent deployment, which theoretically provides the benefits of plaque removal with a resultant maximum SFA mean luminal diameter along with the beneficial features of the coil design at the adductor canal. Concerns regarding the added costs may be justified if this approach decreases the current high secondary SFA endovascular reintervention rates secondary to ISR and now possibly stent fractures.

LIMITATIONS OF OUR ANALYSIS

This analysis suffers from multiple limitations and likely will raise many more questions than answers provided. This was a single-center, retrospective, nonrandomized observational chart and imaging analysis of a highly selective and unmatched cohort of patients over a relatively long period of time (43 months). During this time, multiple physicians treated these unmatched patients with a wide variety of different nitinol stents of different designs and generations and, oftentimes, with varying techniques. Over the duration of this analysis, several clinical changes were made in our SFA treatment policy that could possibly have affected stent fractures, including the adoption of the excimer laser, plaque excision catheters, different stent designs, direct thrombin inhibition, use of GP IIb/IIIa therapy, and less aggressive pre- and post-PTA/stenting balloon dilation. Equivalent follow-up and analysis of all 380 SFA-stented cases during the time period was not possible, precluding an accurate statistical nitinol stent fracture analysis of our entire experience. The poor performance of one or two stent designs could even be responsible for a disproportionately high percentage of stent fractures in this type of analysis. This analysis may even have underestimated the scope of stent fractures during that time. A detailed long-term follow-up of each stent fracture has not been performed. Even with these stated limitations, this analysis should at least identify nitinol stent fractures as a potential problem and raise the question of whether these fractures are benign or malignant.
CONCLUSION

Indeed, this analysis has raised more questions than answers. However, our clinical experience and this analysis has convinced us of the following conclusions:

- Stent fracture does occur with all bare metal nitinol noncoil stent designs. (The true incidence remains unknown but is likely more prevalent than suspected and may be >15%-20% over time.)
- Stent fracture is multifactorial in its etiology and failure mode. (The biomechanical forces exerted on the SFA and nitinol stent fracture behavior must be identified and standardized.)
- Stent fracture rarely occurs with coil stent designs. (The coil design appears to be more tolerant of the complex dynamic biomechanical forces exerted on the SFA.)
- Stent fracture is significantly associated with multiple stent deployment and overlap. (Our experience: 38.8% stent fracture with two stents; 89% with three to five stents.)
- Stent fracture can occur in all SFA segments but the distal SFA is particularly prone to stent fracture. (The site of maximum negative dynamic biomechanical forces.)
- Stent fractures occur and are progressive over time. (Microfractures are likely to progress to macrofractures with the repetitive negative biomechanical forces exerted on the SFA over time.)
- Stent fractures are associated with angiographic (>50% stenosis) and clinical sequelae. (Our experience: 56 of 77; 77.7%)
- A successful endovascular nitinol stent solution for the SFA will require a much greater understanding of the challenges of the metal and the SFA itself.
- Nitinol stent fractures are not benign! ■

David E. Allie, MD, is Director of Cardiothoracic and Endovascular Surgery, Cardiovascular Institute of the South/Lafayette, Lafayette, Louisiana. He has disclosed that he holds no financial interest in any product or manufacturer mentioned herein. Dr. Allie may be reached at (800) 582-2435; David.Allie@cardio.com.

Chris J. Hbert, RT, RCIS, is Director of Technology, Cardiovascular Institute of the South/Lafayette, Lafayette, Louisiana. He has disclosed that he holds no financial interest in any product or manufacturer mentioned herein. Mr. Hbert may be reached at (800) 582-2435; Chris.Hbert@cardio.com.

Craig M. Walker, MD, is Medical Director and Founder, Cardiovascular Institute of the South/Houma, Houma, Louisiana. He has disclosed that he holds no financial interest in any product or manufacturer mentioned here-in. Dr. Walker may be reached at (800) 445-9676; Craig.Walker@cardio.com.