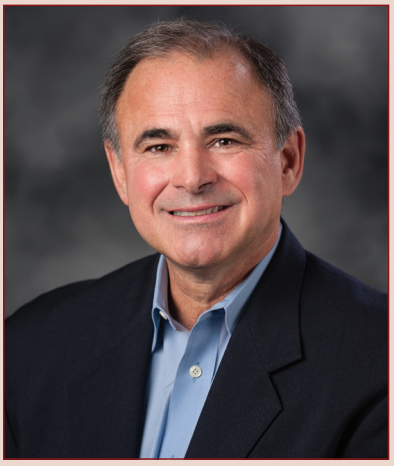
The Role for Endovascular Reconstruction in CFA Disease



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Hello and welcome to the November 2018 issue of *Vascular Disease Management*. There are many interesting articles and case reports in this issue. I have chosen to comment on an article from Drs Vianna, Olivieri, Brandon, Yates, and Beasley concerning the interventional therapy of common femoral artery (CFA) disease in patients who are not surgical candidates for CFA endarterectomy.

I have chosen to comment on this article because the treatment of obstructive common femoral artery disease has been historically relegated to open surgical technique, with many considering any interventional therapy to be contraindicated. Surgical therapy is associated with excellent patency and is tolerated by the overwhelming majority of patients. However, recent publications have challenged this concept, citing that endarterectomy is associated with significant complications in >15% of patients, while also noting that evolving interventional techniques and technologies are associated with better patency and fewer complications than historical interventional treatments. Is it possible that there may be a paradigm change in the treatment of CFA disease in the future?

CFA disease is frequently encountered in patients presenting with claudication and critical limb ischemia. Atherosclerosis is the most common etiology, but iatrogenic causes secondary to vascular closure devices, occlusive sheaths, and prior vascular surgical procedures (with stenotic graft origins or touchdown sites) are common. CFA disease may involve only the CFA with several centimeters of normal artery before the bifurcation, or it may involve the superficial femoral, profunda femoris, and external iliac arteries. The obstruction may be densely calcified, fibrotic, or composed of foreign material such as a vascular plug. The CFA varies widely in size. In addition to these variables, non-vascular factors such as infection, nutritional status, mobility, and patient viability must be considered.

Drug-coated balloons, new-generation stents (with greater radial force, fracture resistance, and drug-eluting capability), distal protection devices, and atherectomy tools are clearly improving interventional outcomes in other vascular beds. Will these new tools solve the problems that were historically encountered with the interventional therapy of CFA disease such as stent fracture, stent compression, jailing of the profunda, embolization, and high rates of restenosis? Are these devices (particularly wire interwoven nitinol stents and drug-coated balloons), which have been designed for other vascular beds, appropriately sized and engineered for the CFA? Will the excellent short-term patency being reported be maintained long term? These questions must be answered.

It is time for a true randomized trial rather than personal convictions to determine how CFA disease should be treated. There must be further evolution of interventional devices that are appropriately sized and designed specifically for CFA treatment. We will need to carefully document lesion characteristics and other variables to ultimately allow a sensible algorithm to determine on an individual basis the best therapy for a given presentation. I strongly suspect that there is going to be a role for interventional therapy and a role for primary surgical therapy of CFA disease.