Orbital Atherectomy

Initial experiences with a new system for the percutaneous treatment of peripheral vascular stenosis.

BY RICHARD R. HEUSER, MD; ROBERT SAFIAN, MD; MARC BOSIERS, MD; AND MARK A. TURCO, MD

Atherosclerotic disease is ubiquitous, and more than half of patients with peripheral vascular disease will have concomitant coronary disease.[1] The symptoms of peripheral vascular disease are frequently disabling and include intermittent claudication or resting leg pain, acute ischemia with blue-toe syndrome, impotence, severe hypertension or hypertension that is associated with worsening renal function, transient cerebral ischemia, stroke, or subclavian steal symptomatology. In the last decade, endovascular intervention has allowed physicians to treat patients with severe and potentially disabling coronary vascular disease using safe and highly successful techniques that have the potential to provide lasting results. Unfortunately, treatment of disease in the lower extremities has proven quite challenging, with significant recurrence and persistent symptomatology even after initial success with angioplasty and stenting.[2,3]

Debulking of peripheral vascular stenoses using atherectomy devices is theoretically an ideal approach because the procedure removes plaque from the target lesion site rather than displacing lesion material. This approach may reduce the need for high-pressure balloon inflation that results in acute vascular injury and neointimal hyperplasia and associated restenosis. Although clinical data regarding the use of atherectomy in peripheral vascular disease are quite limited at present, rotational atherectomy devices have been associated with problems, such as thermal injury caused by rotating burrs and the need for large burr sizes when used in the coronary artery setting.[4,5] Directional atherectomy devices have failed to reduce angiographic coronary restenosis rates compared to stents alone[6] and have been associated with a higher prevalence of early major adverse cardiac events (MACE) in a meta-analysis of 12 studies.[7]

In 2005, a new device, the Orbital Atherectomy System (OAS, Cardiovascular Systems, Inc., St. Paul, MN) received marketing clearance from the FDA for treatment of stenoses in synthetic arteriovenous hemodialysis access grafts. CE Mark was also achieved in 2005 for treatment of peripheral arterial stenosis, as was an investigational device exemption approval from the FDA to conduct a prospective, multicenter clinical trial of the OAS for treatment of infrainguinal arterial stenosis. The pivotal phase of this trial is currently underway and will enroll a total of 124 patients. Although results of the trial are not yet available, case studies that detail use of the OAS are presented.

DEVICE DESCRIPTION

The OAS (Figure 1A-C) is designed for debulking atherosclerotic plaque in coronary and peripheral vessels and utilizes a diamond-coated crown to ablate tissue and yield particles small enough to be eliminated through the capillaries, thus reducing the potential for clinically significant distal embolic complications. The crown has a nonconcentric shape, shifting the center of mass from the center of rotation. Therefore, when the device is rotated, an orbit is created. The mechanism of action for orbital atherectomy is based on the equation:

$$\text{Centrifugal force} = \text{mass} \times \text{rotational speed}^2/\text{radius}$$

The maximum orbital diameter is a function of the crown diameter, rotational speed (rpm), number of passes through the lesion, and consistency of the plaque material. During initial treatment of a plaque lesion, the device orbit is constrained by the obstructing lesion. As the device is operated, force is generated that ablates the plaque. As repeated plaque "sanding" reduces the lesion, the orbit increases while conversely, force decreases. To achieve further plaque removal, force is increased by increasing the rotational speed up to a maximum of 200,000 rpm. The diamond crown sands the plaque bidirectionally as the crown is advanced back and forth through a target lesion.

The OAS allows for the gradual increase of the rotational speed so that a single crown is able to create a luminal enlargement of up to approximately two times the crown diameter. For example, bench studies show that treatment with a 1.9-mm crown may result in a final luminal diameter of 4 mm (Figure 2). The diamond-coated crown has been developed in multiple sizes, ranging

Figure 1. OAS components: single-use disposable catheter/crown device, controller console (A). Orbital atherectomy device: diamond crown distal tip, drive shaft, and handle (B). Orbital atherectomy device: distal tip shows nonconcentric mounted, diamond-coated crown (C).

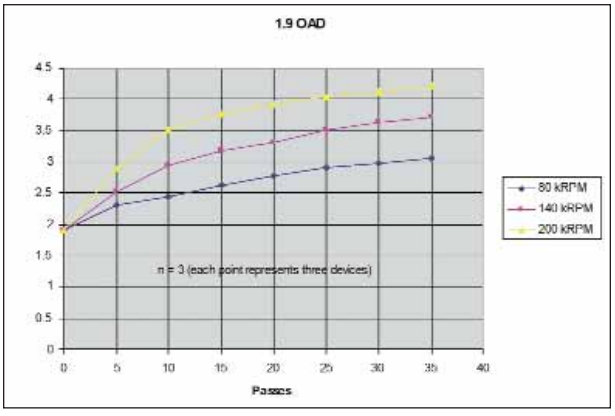

Figure 2. *In vitro* testing of orbit characterization—luminal size achieved using the 1.9-mm–sized crown. Notice the higher velocity of the crown, the larger luminal opening.

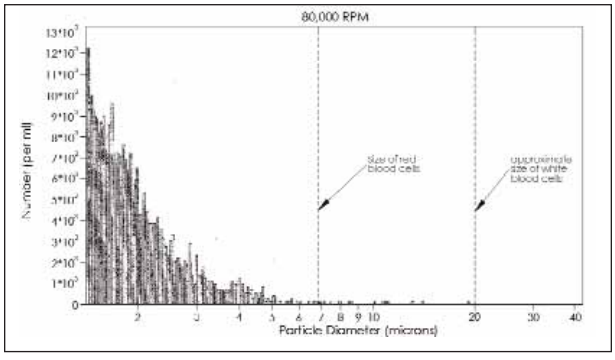

Figure 3. *Ex vivo* testing of particle size/distribution among all OAS crown sizes. The OAS ablates the occluding plaque into particles small enough to be removed through the normal reticuloendothelial system after passing through the capillary bed. Note: almost all particles are less than 7 µm.

initially from 1.2 mm to 2 mm in diameter, and it is mounted on a flexible coil-wound drive shaft that tracks and rotates over an atherectomy guidewire that can be independently advanced and steered. The flexibility of the system permits navigation and operation within tortuous vascular anatomy. The novel design of the coil-wound drive shaft and abrasive crown permits cooling by saline infusion and blood flow past the crown, decreasing the risk of thermal trauma and ischemia in the target vessel.

The OAS drive shaft is powered by a compressed gas turbine (usually containing nitrogen). Rotational speed is controlled by a pressure valve located on the front panel of the OAS controller console (Figure 1). The turbine may be activated and stopped by an electrical foot switch. The controller includes an optical tachometer displaying rotational speed, and a roller pump that is used to pump saline through the infusion sheath and around the drive shaft and crown.

PRECLINICAL TESTING

The OAS has undergone extensive *in vitro*, *ex vivo*, and animal testing to establish a safety and efficacy profile. *In vitro* studies assessed critical design parameters, including orbit characterization for the individual crown sizes. Using graphite test samples, orbit diameter (luminal diameter) was determined at operation speeds ranging from 80,000 to 200,000 rpm after various numbers of passes with the device. Figure 2 shows the luminal size achieved using the 1.9-mm-diameter crown size.

Ex vivo studies quantified downstream particle size and number in a simulated-use model. Stenoses were formed in porcine coronary arteries, then explanted and connected to a pressurized flow system. The OAS was used to treat the stenosis, and downstream effluent was collected and analyzed using a particle sizer/counter (Beckman Coulter, Inc., Fullerton, CA). The mean particle size among all crown sizes was 1.7 µm to 3.1 µm, with 94% to >99% particles <5 µm in diameter; 99.7% of particles were <20 µm (Figure 3). Thus, it is expected the OAS ablates the occluding plaque into particles small enough to be removed through the normal reticuloendothelial system after passing through the capillary bed (the average capillary diameter is 7 µm to 9 µm).

Animal studies were also performed to assess safety and efficacy. A balloon overstretch injury was induced in porcine

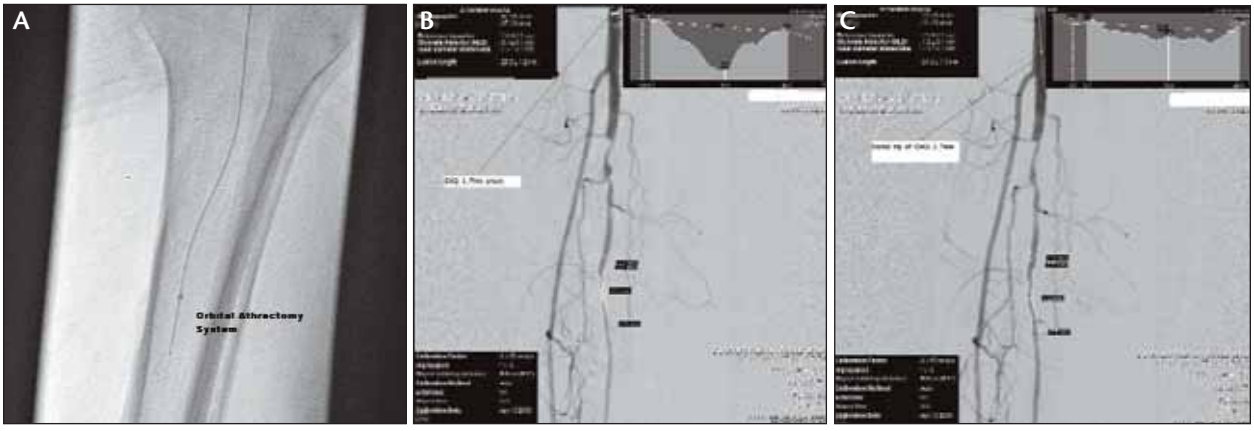

Figure 4. Case study 1: Baseline angiography showed an 85% diameter stenosis located in the left peroneal artery (A). Under fluoroscopy, the shaft and crown are advanced immediately proximal to the lesion (B). Angiography after the fourth pass showed a residual diameter stenosis of 23% (C).

peripheral arteries, and treatment of the resulting lesions with the OAS was performed 1 month later. Treated vessels were explanted, and a histopathologic examination was performed to evaluate acute vessel injury. There were no angiographic complications during treatment (eg, no perforations, dissections, reflow, distal embolization), and a debulking treatment effect was observed in all cases. The majority of treated lesions showed no histologic evidence of device-related vascular wall injury. The most severe injury observed was limited to the internal elastic lamina and affected <50% of the media thickness without extending to the adventitia.

USING THE DEVICE

The OAS setup requires saline solution (standard 1-L bag of saline for infusion), an atherectomy guidewire, compressed gas (usually nitrogen with a minimum of 80 psi/5.6 bar), and appropriate hardware to attach the OAS and gas supply. An angiogram is obtained to assess the reference vessel diameter, lesion morphology, and select the appropriate crown size. The OAS is primed with saline, and crown rotation and brake override are tested. The lesion is then approached and crossed with the atherectomy guidewire, and the OAS shaft is threaded over the guidewire and advanced through the hemostasis valve of the guide catheter/sheath. Under fluoroscopic guidance, the shaft and crown are advanced immediately proximal to the lesion (Figure 4A). Contrast material is injected to verify crown and vessel size compatibility. The on/off switch on the controller is depressed to start saline flow, and the foot pedal is depressed to begin rotation initially

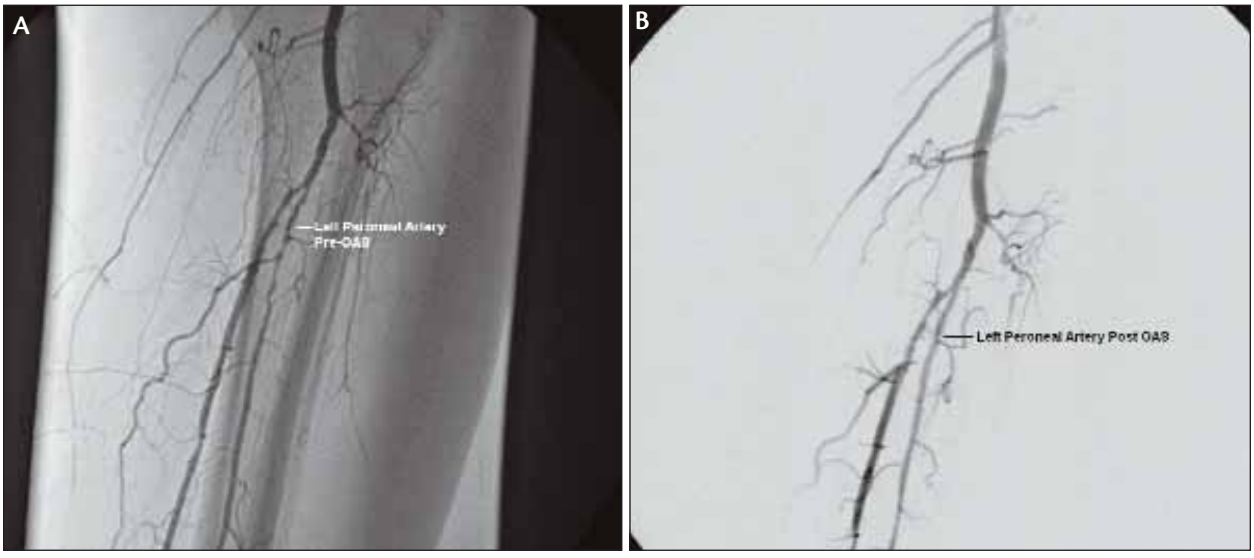

Figure 5. Case study 2: Angiography revealed diffuse critical disease of the left peroneal artery (A). Posttreatment angiography of the left peroneal artery showing good results (B).

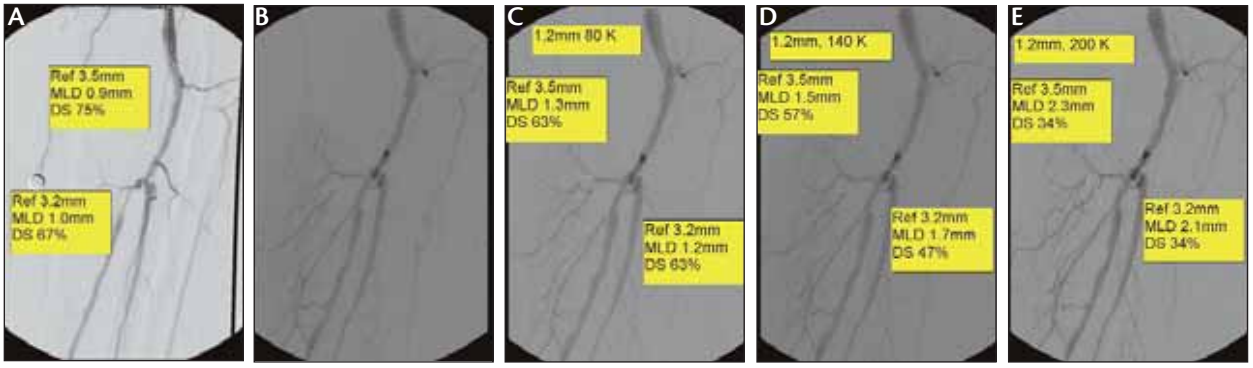

Figure 6. Case Study 3: Baseline online quantitative angiography—proximal lesion in the tibioperoneal trunk, distal lesion in the posterior tibial artery (marker bead = 6 mm) (A). Orbital atherectomy device/1.2-mm–diameter crown positioned for treatment (B). Angiography after treatment at 80,000 rpm (C). Angiography after treatment at 140,000 rpm (D). Angiography after treatment at 200,000 rpm. No further interventional treatment provided (E).

at low speed (80,000 rpm). The crown control knob is moved slowly back and forth to advance and retract the crown tip through the lesion, with angiography used to evaluate results. Speed is increased over gradual sequential passes, up to a maximum speed of 200,000 rpm. The foot pedal is released to stop crown rotation. Debulking is carefully monitored with intermittent resting periods and injection of contrast material to verify results.

CLINICAL STUDIES

In the US, a single-arm, prospective, multicenter trial is currently underway in patients with at least one treatable *de novo* stenosis in an infrainguinal peripheral artery. The purpose of the study is to establish safety and acute procedural effectiveness of the OAS in debulking stenotic lesions in the treatment of symptomatic occlusive peripheral arterial disease. The primary efficacy endpoint is the mean decrease in percent diameter stenosis of the target lesions. The primary safety endpoint is the incidence of serious adverse events at 30 days. Secondary endpoints include procedural success as quantified by angiography; overall clinical status of the patient at 1 and 6 months of follow-up; incidence of adverse events at 1 and 6 months; incidence of symptom-driven target lesion revascularization at 6 months; and ankle brachial index (ABI), Rutherford classification, and the degree of clinical improvement using Rutherford and Becker's scale at 1 and 6 months.

CASE STUDIES

Case 1

A 74-year-old man had a known history of smoking. His resting ABI was 0.55, and he presented with symptoms of rest pain (Rutherford category 4). He was referred for endovascular intervention, and vascular access was achieved via a contralateral femoral approach and a 6-F sheath (Brite Tip, Cordis Endovascular, a Johnson & Johnson company, Miami, FL) was placed. Online quantitative angiography was used to assess critical dimensions (Innova 4100, GE Medical, Waukesha, WI). Baseline angiography showed an 85% diameter stenosis located in the left peroneal artery, which was the only remaining artery supplying the lower limb and foot with blood. Both the left anterior and posterior tibial arteries were occluded (Figure 4A, B). The reference vessel diameter was 2.6 mm and the lesion length was 23 mm. A .009-inch atherectomy guidewire was delivered across the target lesion. A 1.7-mm OAS crown was delivered over the wire and positioned proximal to the target lesion. The OAS was used to make an initial pass at 90,000 rpm, followed with three additional passes at 100,000 rpm each, with brief rests between passes. Angiography after the fourth pass showed a residual diameter stenosis of 23% (Figure 4C). No further treatment was provided. The total OAS operation time was 83 seconds. No other lesions were treated during the index procedure, and the total procedure time was 50 minutes. There were no procedural complications, and the patient was discharged from the hospital the next day on low-molecular-weight heparin (0.66 mL for 3 weeks, according to hospital standards), clopidogrel, and aspirin. At 30-day follow-up, the patient's ABI was .95, and he was assessed to be Rutherford category 0. In this case, use of the OAS resulted in restoration of optimal flow, without need of further angioplasty or stenting.

Case 2

A 56-year-old woman with a history of insulin-dependent diabetes with a nonhealing ulcer of the left leg was seen in the clinic. Angiography revealed diffuse critical disease that included the left peroneal artery (Figure 5A). The refer-

ence vessel diameter was 3.5 mm. A contralateral approach was used, and a 6-F guiding sheath was placed for access and visualization (as described in case 1). A 1.7-mm OAS crown was used with the OAS, and multiple passes were made at rotational speeds between 80,000 and 200,000 rpm, with rest at 30-second intervals. Posttreatment angiography showed good results (Figure 5B).

Case 3

A 70-year-old man was admitted to the hospital for treatment of critical limb ischemia characterized by left leg claudication, ischemic rest pain, and ulcerations on his fourth toe (Figure 6A-E). He did not improve after successful revascularization of the left outflow circulation, and he was readmitted for further revascularization of the left tibioperoneal trunk and posterior tibial artery. The inflow circulation was normal. He also had chronic renal insufficiency, hypertension, and hyperlipidemia. Angiography and intervention were performed using a retrograde right-to-left crossover approach with 6-F catheters. A .014-inch Prowater guidewire (Abbott Laboratories, Redwood City, CA) was positioned in the distal posterior tibial artery and exchanged for a .009-inch Rotablator support wire (Boston Scientific Corporation, Natick, MA). Orbital atherectomy was performed on the tibioperoneal trunk and on the posterior tibial artery using a 1.2-mm crown at 80,000, 140,000, and 200,000 rpm. The patient was discharged from the hospital in stable condition and experienced improvement in claudication and resolution of ischemic rest pain and ulcerations.

DISCUSSION

Peripheral vascular disease is a serious and disabling condition that affects millions of people worldwide. Lower-extremity disease is notoriously difficult to treat and is often recurrent. Atherectomy that allows successful debulking of stenoses may improve results and reduce the potential for associated restenosis. Excisional atherectomy (eg, SilverHawk, FoxHollow Technologies, Redwood City, CA) works in a different form than the OAS device, as it actually shaves the plaque. The theoretical advantage of orbital atherectomy is greater efficiency in treating calcified, resistant lesions. Similar to rotational atherectomy, the possibility of no re-flow is present with the OAS device. The incidence of no reflow may be reduced with use of orbital atherectomy, since the nonconcentric crown allows for continued perfusion during treatment of even the most severe stenosis. No reflow has not been observed in our limited peripheral vascular experience, and this will be more fully assessed in the clinical study. There is no experience using the device in human coronary arteries, and the incidence of myocardial stunning or stunning secondary to no reflow is unknown. Although atherectomy has met with limited success for treatment of peripheral lesions over the years, the OAS device applies a new technology that may improve results. Currently, the OAS is under study at a number of US centers for treatment of peripheral artery disease. Early results suggest that the device can be used successfully to treat stenotic disease in selected patients with lower-extremity disease. In addition to the ongoing trial, FDA approval is being sought for an IDE using the OAS to treat occluded coronary arteries. We eagerly await full results of trials with this new device. ■

Richard R. Heuser, MD, is Director of Cardiology, St. Luke's Hospital and Medical Center, Clinical Professor of Medicine, University of Arizona College of Medicine, Phoenix, Arizona. He has disclosed that he is a shareholder in Cardiovascular Systems, Inc. Dr. Heuser may be reached at (602) 234-0004; rrh@phoenixheartcenter.com.

Robert Safian, MD, is Director, Cardiac and Vascular Intervention, William Beaumont Hospital, Royal Oak, Michigan. He has disclosed that he has no financial interest in any product or company mentioned herein. Dr. Safian may be reached at (248) 898-4163; rsafian@beaumont.edu.

Marc Bosiers, MD, is Head of the Department of Vascular Surgery, A.Z. St. Blasius Hospital, Dendermonde, Belgium. He has disclosed that he has no financial interest in any product or company mentioned herein. Dr. Bosiers may be reached at +32 52252822; marc.bosiers@pandora.be.

Mark A. Turco, MD, is Director of the Center for Cardiac & Vascular Research, Washington Adventist Hospital, Takoma Park, Maryland. He has disclosed that he has no financial interest in any product or company mentioned herein. Dr. Turco may be reached at (301) 891-6636; mturco@ahm.com.

Note: The peripheral arterial use of the Orbital Atherectomy System described in this article is investigational in the US.

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