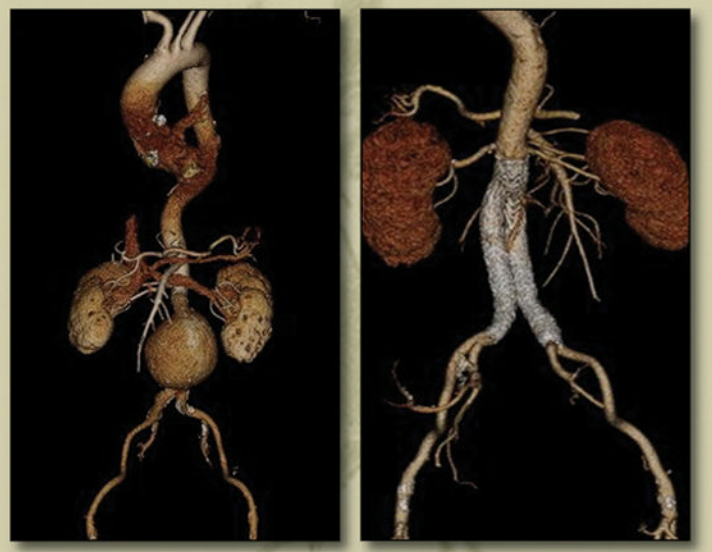
Modern Trends in Vascular Surgery

Endovascular Technology



Mark K. Eskandari, Mark D. Morasch,
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Modern Trends in Vascular Surgery

Endovascular Technology

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Preface

The Northwestern University Vascular Symposium is an educational event that has been in existence, uninterrupted, since 1976. Each year, 40 or so leading vascular surgeons from the US and from overseas serve as symposium faculty. They speak and take part in discussions regarding topics of special interest to vascular and cardiac surgeons and to those interested in the medicine of vascular disease. In addition, for many years now, the faculty have authored chapters that have been published as a compendium to the symposium. The compendium book is published as a permanent record of their contributions. This book has been widely successful and is a popular component of the meeting each year. Until recently, the book was made available to the public after the symposium and was always a sell-out.

Over the last five years the compendium was published privately and was not commercially marketed; it was distributed only to participants of the symposium. It has become apparent that the book has been missed by many who, for whatever reason, were unable to travel to Chicago for the annual event. We also recognized that the lack of worldwide circulation did an injustice to the faculty members whose chapters represent a real contribution to the medical literature. *Endovascular Technology* is the brainchild of Dr. James Yao and Dr. William Pearce. It is a compilation of updated chapters from the most recent symposia and highlights the advancement of endovascular technology for the treatment of vascular disease.

The book begins with a series of chapters that discuss general considerations, history, the endovascular suite, and training in this era of endovascular surgery. The next few sections touch upon contemporary techniques used for the treatment of cerebrovascular and infrainguinal disease.

The subsequent sections focus on endovascular intervention for the treatment of not only straightforward aneurysms, but, amongst other topics of interest, the management of acute dissections, traumatic injury, and the most common complications seen with endografting. The book ends with sections that broach the topics of endovascular management of aortic branch pathology and the endoluminal treatment of venous disorders.

Treatment of vascular disease is changing and surgeons must make changes accordingly. The Northwestern Symposium strives to keep those involved in the management of vascular pathology on the cutting edge of techniques and technology. Our hope is that this textbook will open the expert discussions to a broader audience.

Mark D. Morasch, M.D., F.A.C.S., R.P.V.I.

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We thank the administrative staff of the Division of Vascular Surgery—Sara Minton and Jan Goldstein—for their support. Special thanks to Susan Parmentier of Greenwood Academic for reprocessing chapters of the last five Northwestern Symposia. We would also like to thank W.L. Gore & Associates for a generous education grant to support the Northwestern Vascular Symposium over the years. Finally, we thank Mr. Jason Malley of People's Medical Publishing House-USA and Mr. Harjeet Singh from Spearhead Global, Inc. for their expert assistance.

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SECTION I

General Considerations

1

History of Endovascular Surgery

James S. T. Yao, M.D., Ph.D.

INTRODUCTION

Endovascular surgery is defined as a form of minimally invasive surgery that was designed to access many regions of the body via major blood vessels. In most instances, femoral vessels are used for the entry of a catheter for imaging of the vessel and also for therapeutic use. In the last decade, rapid development of endovascular technology has transformed the landscape of the practice of vascular surgery. As it is currently practiced, vascular surgery, to a certain extent, is a hybrid surgical specialty combining open and endovascular techniques to render proper treatment for patients. This drastic change in how we practice has also impacted the curriculum of training for vascular surgeons—our ultimate goal should be to train vascular specialists and not just surgeons.

There have been abundant writings on the history of open vascular surgical procedures but relatively few on endovascular surgery. This chapter attempts to review the historical development of catheter-based endovascular technology such as arteriography, balloon angioplasty, stenting, and endovascular grafting for aortic aneurysms.

Arteriography

Arteriography is the cornerstone of the development of vascular and endovascular surgery, providing a road map for surgeons to initiate diagnosis and treatment. The first arteriogram was done in 1924 by Barney Brooks, who reported three cases of femoral arteriography,[1] to determine whether patients needed amputation. In 1927, Egaz Moniz, a Portuguese neurologist and neurosurgeon, reported the first carotid arteriography in man.[2] In 1929, Reynaldo dos Santos, a Portuguese surgeon and primarily a urologist, reported the first translumbar aortogram.[3]

All these procedures were done by direct needle puncture. The volume of contrast media injected was limited by the size of the needle, and the dilution of contrast media as it reached the target area resulted in poor images of the vascular bed. The major breakthrough in endovascular surgery was the introduction of selective catheter arteriography by Sven-Ivar Seldinger of Sweden.[4] This technique allows selective examination of various vascular beds in the body. Catheter arteriography helps to identify

subclavian steal syndrome and fibromuscular dysplasia of renal and peripheral arteries and made translumbar aortogram obsolete.

Based on arteriographic findings, DeBakey developed the concept that arteriosclerotic lesion is often segmental in nature.[5] This concept paved the way for vascular surgeons to perform arterial bypass surgery. Use of a catheter as a diagnostic tool changed when Fogarty introduced the balloon catheter to remove emboli in 1962.[6] The Fogarty catheter was a landmark contribution and signaled the beginning of endovascular treatment. Around the same time, in 1963, Charles Dotter of Oregon also had the idea to convert the catheter from a diagnostic tool to therapeutic use. He designed a catheter to dilate a stenotic lesion of a femoral artery.[7] As a result, a new generation of radiologists—and the specialty of interventional radiology—were born and Dotter has been hailed as the father of that specialty.

BEGINNING OF ENDOVASCULAR TECHNOLOGY

Wiring of an aortic aneurysm is probably the very first endoluminal vascular interventional therapy. In the procedure, copper or silver wire is threaded into the aneurysm sac through a fine needle to promote thrombosis. (Figure 1–1) The first procedure was done by Moore of London in 1864 using cold wire.[8] In 1879, Corradi modified the technique by passing a galvanic current through the wire to accelerate the thrombotic process.[8]

In 1937, Colt reported his experience with opening a miniature umbrella in the sac.[8] In the era of indirect surgery of the 1940s, pioneer vascular surgeons such as Blakemore, Linton, and de Takats all reported their experience with wiring of aneurysms.[9–10] Figure 1–2 shows loops of wire in the aneurysm sac. Figure 1–3 shows the wiring instrument used by Arthur Blakemore. (Note the spool of wire, needles, and wire passer.) Besides wiring, a spray of 2% sodium diethyl phosphate to the wall of the sac to promote fibrosis has been reported.[10] (Figure 1–4) In 1890, McEwen reported satisfactory results with the injection of fibrin to the sac via a fine needle to promote thrombosis.[8]

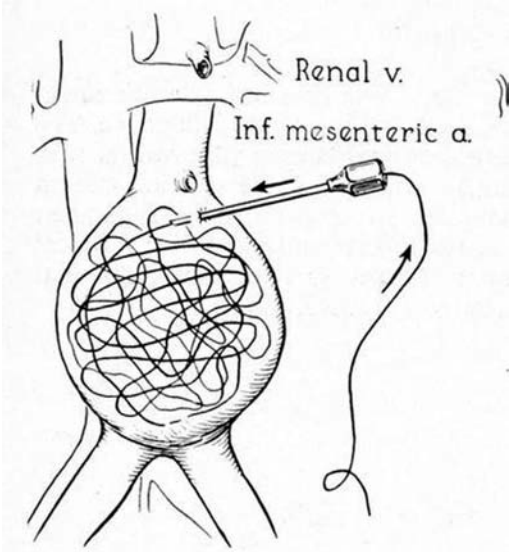

Figure 1-1. Wiring of an abdominal aortic aneurysm. (From de Takats G, Marshall MR. Surgical Treatment of arteriosclerotic aneurysms of the abdominal aorta. *Arch Surg* 1952;64:307–319. Copyright © 1952 American Medical Association. All rights reserved. Reproduced by permission.)

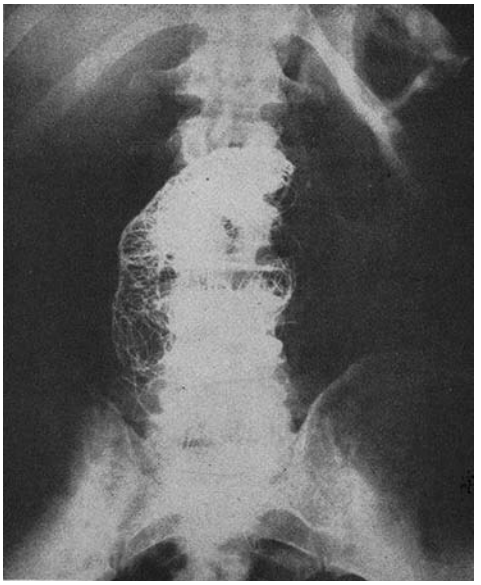

Figure 1-2. Plain x-ray of abdominal aortic aneurysm with wiring. Note loops of wire within the sac. (From Pratt GH. Surgical Management of Vascular Diseases. Philadelphia: Lea & Febiger, 1949. Reproduced by permission.)

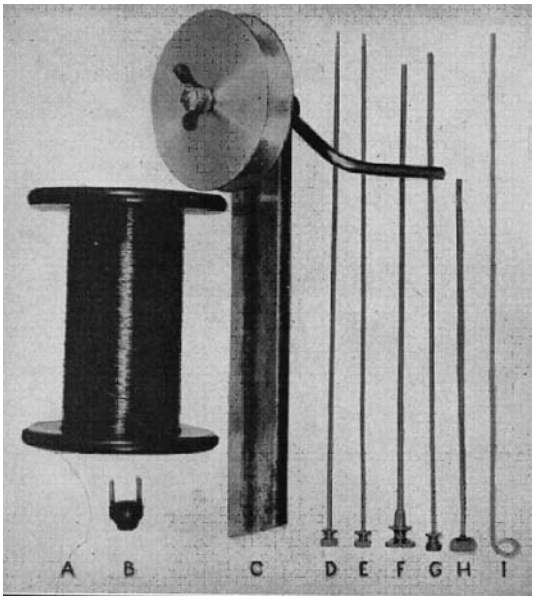

Figure 1-3. Electrothermic coagulation of aneurysm by Blakemore. Note the spool of wire, needles, and passers. (From Pratt GH. Surgical Management of Vascular Diseases. Philadelphia: Lea & Febiger, 1949. Reproduced by permission.)

Fogarty Balloon Embolectomy Catheter

The first attempts at embolectomy were credited to John B. Murphy of Chicago in the early 1900s. In a detailed report published in 1902, he described using a ureteral catheter to perform the procedure on a patient with iliac emboli.[11] Murphy first exposed the femoral

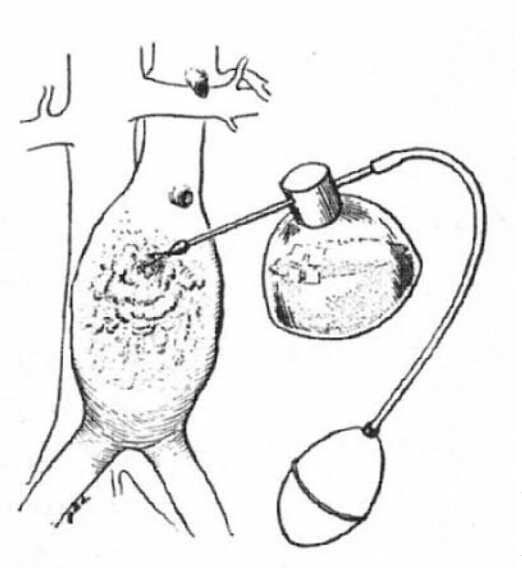

Figure 1-4. Aneurysm wall sprayed with 2% sodium diethyl phosphate. (From de Takats G, Marshall MR. Surgical treatment of arteriosclerotic aneurysms of the abdominal aorta. *Arch Surg* 1952;64:307–319. Copyright © 1952 American Medical Association. All rights reserved. Reproduced by permission.)

Figure 1-5. Passage of a ureteral catheter through the clot in the iliac artery. Inset shows the femoral artery is exposed for entry of the catheter. (From Murphy JB. Removal of an embolus from the common iliac artery with re-establishment of circulation in the femoral. *J Am Med Assoc* 1909;LII:1661–1663.)

artery as the entry site. (Figure 1–5) The artery was thrombosed and he used a spoon-type instrument to retrieve the clot but with partial success. A few clots came out but there was no flow. He then passed a soft catheter upward and was able to advance about 6 inches. A ureteral catheter was then inserted and advanced to the previous length and met with resistance. A uterine sound was then introduced. At a distance of 7.5 inches it met resistance and was forced .5 inch farther. On its withdrawal, a large quantity of grumous thrombotic debris came out but not arterial blood. The catheter was reintroduced with little resistance and passed through into a free space. This was followed by intense arterial flow carrying with it a lot of embolic debris and fresh bright red blood. After several passages, normal flow was restored and the artery was closed. The patient did well after the procedure. This is the first recorded embolectomy from a remote site. In the discussion section of the article, Murphy pondered about the use of aspiration through the catheter to facilitate the retrieval of the thrombus. In the modern era, the idea of using aspiration was first used by Greenfield in 1969, using the percutaneous aspiration thromboembolectomy catheter for removing pulmonary emboli.[12] It is of interest to note that Murphy had been credited as the first surgeon to perform end-to-end anastomosis of an injured femoral artery in a patient with a gunshot wound.[13] He appears also to be the first surgeon to perform an endovascular procedure—fifty-some years before Thomas Fogarty.

Fogarty began his medical career as a scrub technician at Good Samaritan Hospital in Ohio at the age of 15. He worked under the direction of Dr. John Cranley, the first surgeon to dedicate his entire practice to vascular surgery. Stimulated by the poor operative results of open embolectomy, Fogarty conceived the idea of using a balloon catheter to remove clots. He took the baby finger of a #5 glove, cut it off, and tied it onto the end of a urethral catheter. That was basically the design and development

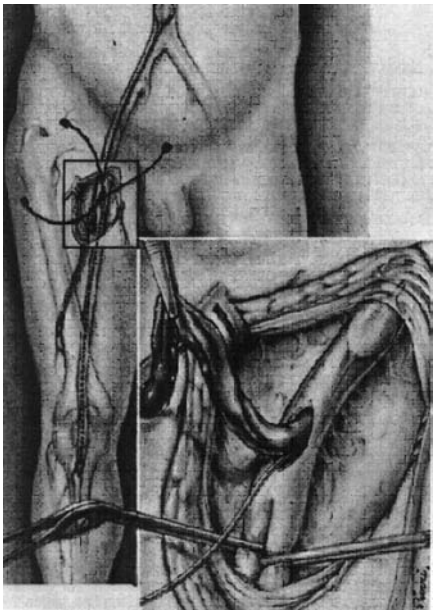

Figure 1-6. Use of the Fogarty catheter. (From Fogarty TJ, Crauley JJ, Krause RJ, et al. A method for extraction of arterial emboli and thrombi. *Surg Gynecol Obstet* 1963;116:241. Reproduced by permission.)

process—at home in his attic. He got some long test tubes and, using out-of-date blood, he formed make shift clots within the test tubes. He tested the catheter to remove clots and it worked very well. He also used Jell-O to simulate clots and tested again for its removal in a test tube. He used the catheter in a cadaver only once. From the time of his first idea for the procedure to its first use in a patient took about 2 months. There was no FDA regulation at the time—when the first opportunity presented itself, they used it. Drs. John Cranley and Raymond Krause used the procedure in a patient with iliac emboli and were pleasantly surprised by the ease of the procedure.[14] The Fogarty catheter was the first to be used for therapy, and the procedure's success changed the future use of the catheter. (Figure 1–6) Charles Rob considered the Fogarty catheter to be one of the truly original concepts introduced in arterial surgery since the end of World War II.[15]

Despite that success, however, Fogarty encountered difficulty in securing a manufacturer, and it took about 3 years before a company named Edwards Life Science had the vision to bring the catheter to market. Part of the reason was that the traditional teaching at that time was that, if you manipulated the inside of an artery, it would clot. Fogarty also had problems having his subsequent reports on 11 patients published in surgical journals. He submitted the manuscript to three leading surgical journals and was rejected. Finally, *Surgery, Obstetrics & Gynecology* published his article, "A Method for Extraction of Arterial Emboli and Thrombi," in the "Surgeons at Work" section of the journal.[6] When John Cranley presented the experience of the Fogarty catheter at the surgical grand rounds and gave Fogarty credit, the chairman of the Department of Surgery, who was also the president of the American College of Surgeons, chided, "Only one as inexperienced and uneducated as a medical student would think of this."[14]

Thomas Fogarty was a genius and a hard-working individual. He completed medical school at the same time he worked as an OR scrub technician and emergency room nurse in 1962. He began his surgical residency at the University of Oregon where he

and Charles Dotter connected for a brief period. In the third year of residency, he decided to go to the National Institutes of Health (NIH) to learn research methods and cardiovascular physiology. In 1967, he returned to the University of Oregon, then completed his cardiac surgery residency at Stanford University. In 1980, Fogarty became director of cardiovascular surgery at Sequoia Hospital, Redwood City, California, and in 1995 served as president of the Society for Vascular Surgery.

The Fogarty catheter is only one of his many inventions, medical and non-medical. He was known for inventing the motorcycle clutch system, which is still in use, at the age of 16. In addition to the embolectomy catheter, he is also the inventor of the endovascular graft. He received numerous honors and the ultimate recognition for his innovations, induction into the National Inventors Hall of Fame. In his later life, he began making wine as a hobby, then as a business. Thomas Fogarty Winery and Vineyards was producing over 10,000 cases a year and distributing wines to states outside of California.[16] He strongly believed wine is a health food and that two or three glasses of Fogarty Pinot Noir per day would prolong health.

Percutaneous Transluminal Angioplasty

In 1963, Charles Dotter accidentally recanalized an occluded right iliac artery by passing a percutaneously introduced catheter retrograde through the occlusion to perform an abdominal aortogram in a patient with renal artery stenosis. He reported this at the Czechoslovak Radiological Congress in June of that year and began to conceive of newer devices such as a balloon-mounted catheter and stents.[17] On January 16, 1964, Dotter and his trainee, Melvin Judkins, performed the first percutaneous dilation of a stenotic lesion of the femoral artery in an 82-year-old woman with severe ischemia who had refused amputation.[7] (Figure 1–7) The catheter used was a coaxial system consisting of a 12 Fr catheter placed over an inner 8 Fr catheter and 0.44 inch guidewire. The application of radial force resulted in expansion of luminal diameter, which persisted, allowing improvement of blood flow and avoidance of amputation. Her pain disappeared within a week and the ulceration healed. A follow-up arteriogram done at 3 weeks and at 6 months showed the vessel to be patent. The patient died of congestive heart failure almost 3 years later, "still walking on [her] own two feet." The procedure earned Dotter a reputation and the procedure came to be called "Dottering a lesion."[17]

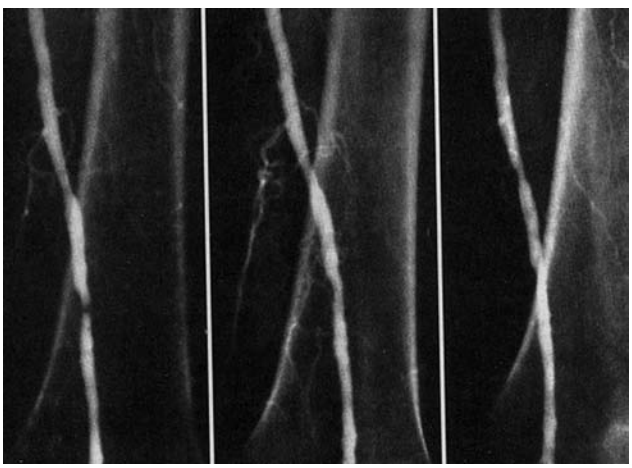

Figure 1-7. Femoral arteriogram of Dotter's first catheter patient. A. Before dilatation; B. Immediately after dilatation; and C. 3 weeks after the procedure. (From Dotter CT, Judkins MP. Transluminal treatment of arteriosclerotic obstruction. Description of a new technic and a preliminary report of its application. *Circulation* 1964;30: 654–670. Reproduced by permission of the American Heart Association.)

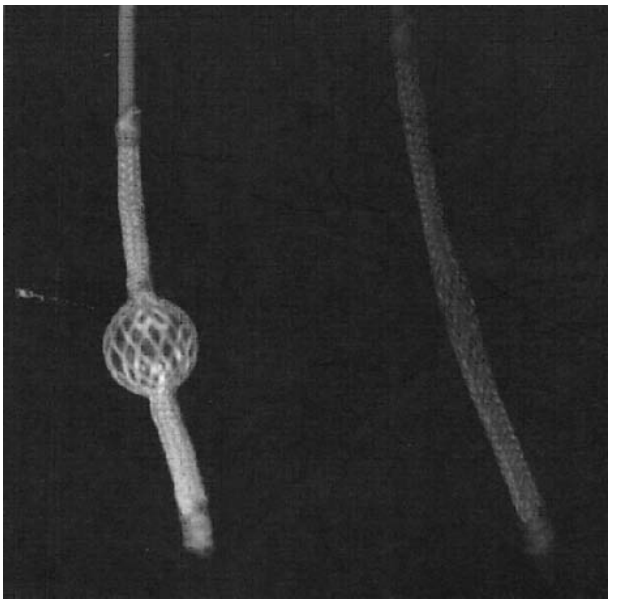

Figure 1-8. Original Dotter dilating catheter. (From Friedman SG. *A History of Vascular Surgery*, 2nd ed. Malden MA: Futura, 2005. Reproduced by permission.)

As mentioned earlier, Fogarty connected with Dotter while he was an intern at the University of Oregon, where Dotter served as Director of Radiology. At Dotter's request, Fogarty made several catheters for Dotter to use for iliac angioplasty. Further modification of the catheter allowed a small puncture site and made iliac percutaneous transluminal angioplasty (PTLA) feasible. Dotter reported his experience of iliac angioplasty in 1974.[18] (Figure 1–8) Other investigators who were interested in using a balloon to dilate arterial stenosis include Portsmann in 1973 (the "caged balloon")[19] and Zeitler et al in 1978.[20] Zeitler and his colleagues used the Fogarty balloon catheter and found little success with the catheter.[21]

Despite the innovation in transluminal therapy, Dotter has received little attention, due partly to the prevailing concept at that time that any transluminal manipulations would only cause thrombosis. Dotter was also somewhat of a publicity hound and frequently appeared in newspaper, radio, and television interviews that earned him the nickname of "Crazy Charlie." Surgeons did not greet Dotter's transluminal dilatation technique kindly; instead, they castigated him.[18,19,22] Nevertheless, the contribution of Dotter is immense and the growth of interventional radiology into a specialty service in all hospitals is due to his innovative idea to use the catheter not just as a diagnostic tool but also for therapeutic use. Of similar importance was the development of the improved and refined catheters and guiding system. Dotter had the good fortune to work with Bill Cook, who would become the CEO and sole proprietor of the world's largest supplier of angiographic supplies. Bill Cook played a pivotal role in Dotter's success.[17]

The attitude toward PTLA changed when a Zurich cardiologist, Andreas Gruentzig, developed a balloon-catheter capable of dilating arterial stenosis. After Gruentzig described the first 5 cases of percutaneous transluminal coronary angioplasty in a letter to the editor of *Lancet* in February 1978, surgeons and physicians became more receptive to the idea of transluminal angioplasty.[23] Unlike Dotter, Gruentzig presented himself in a conservative and scientific light. Gruentzig's success and acceptance by the

medical establishment helped Dotter because of the knowledge that the idea originated with him. With Gruentzig behind Dotter, others finally began to give Dotter credit for something he had done nearly 15 years before. Perhaps Dotter, like some geniuses, was far ahead of his time. Appropriately, Dotter has since been recognized as the "father of interventional radiology."

In the late 1970s, percutaneous transluminal angioplasty became a standard alternate procedure for treatment of focal stenotic lesion of peripheral arteries, including renal arteries. The first renal artery angioplasty for renovascular hypertension was done by Felix Mahler of the University of Berne, Switzerland, in 1977.[24] Because of the possibility of embolization, an attempt to dilate a stenotic carotid artery was delayed until 1980, when Klaus Mathias of Germany reported the first successful transluminal angioplasty of a carotid stenosis.[25] In the same year, Bachman of Canada and Theron of France reported successful dilatation of the subclavian artery.[26,27] This was followed by a report in 1982 from Russian surgeons on successful angioplasty of the subclavian artery.[28]

The Stent

A stent is a man-made metal mesh tube inserted into a natural passage/conduit in the body to prevent or counteract a disease-induced, localized flow constriction. The origin of the word "stent" remains unsettled. Some attribute it to Jan Esser, a Dutch plastic surgeon who in 1916 used the word to describe a dental impression compound invented in 1856 by the English dentist Charles Stent (1807-1885), which Esser employed to craft a form for facial reconstruction.[29] Reportedly, from the use of Stent's compound as support for facial tissues grew the eventual use of stents to open various body structures. The first stents used in medical practice were initially called "wallstents."

Charles Dotter faced skepticism because many of his groundbreaking ideas were far ahead of their time. His report on intravascular stenting published in 1969 also met with skepticism and the technique failed to gain momentum.[30] The stent Dotter used was a nonexpanding stent—a fixed diameter metal coil tube that he had placed in the hind legs of dogs. (Figure 1–9) Despite an early patency rate, these coils narrowed the lumen of the artery, producing unsatisfactory results. Dotter did not pursue the stent project further until 1983.

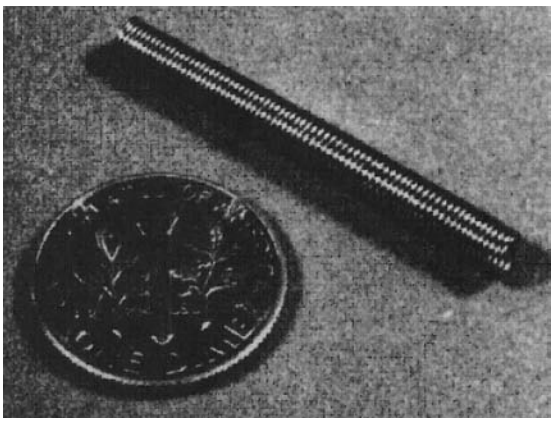

Figure 1-9. Tubular coilspring endovascular prosthesis by Dotter. (From Dotter CT. Transluminally-placed coilspring endarterial tube grafts. Long-term patency in canine popliteal artery. *Invest Radiol* 1969;4:329–332. Reproduced by permission.)

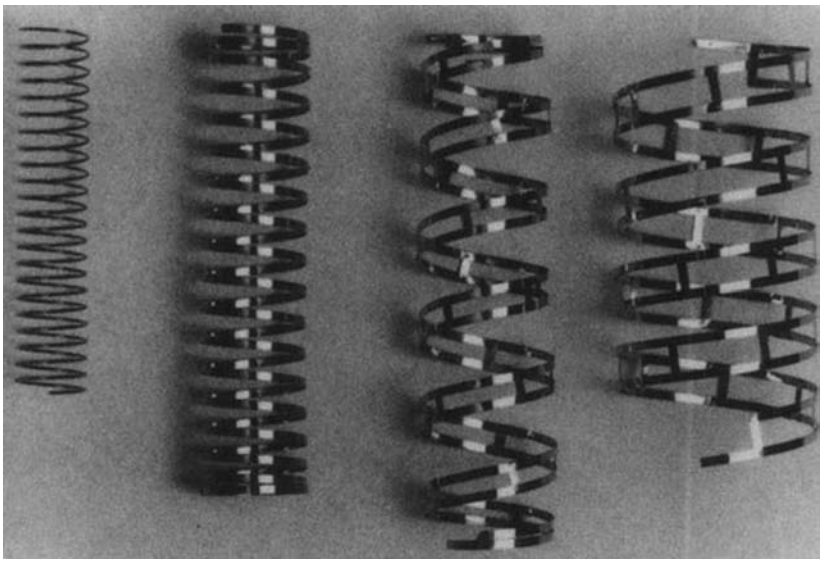

Figure 1-10. Various types of implanted spiral springs, including two double-spirals. (From Maas D et al. Radiological follow-up of transluminally inserted vascular endoprosthesis: An experimental study using expanding spirals. *Radiology* 1984;152:659–663. Reproduced by permission.)

In the 1980s, besides Dotter and Palmaz, there were others also working on stents. These include Dierk Mass on self-expanding spiral stents[31] (Figure 1–10), Amplatz's group on self-expanding thermal memory stents, and the zigzag self-expanding stent by Cesare Gianturco.[31] The thermal memory stent was made possible by the use of nitinol alloy discovered by Soviet metallurgists.[32] The alloy deformed at a certain temperature, to regain the initial shape completely or partially when heated to a higher temperature. In 1983, Dotter and Craig independently described the experimental use of nitinol spiral coil as an endovascular stent in 1982.[33] Russian surgeon I. H. Rabkin was the first surgeon to perform dilatation with placement of a nitinol stent in an external iliac artery.[28]

According to a recent interview, Julio Palmaz claimed the concept of leaving a scaffold behind stemmed from listening to Gruentzig's presentation on balloon angioplasty in 1978 at the Society of Cardiovascular and Interventional Radiology (*SCVIR*) meeting in New Orleans.[34] At that time, he was a resident in radiology. After the meeting, he informed the department chair, Dr. Steve Reuter, of his idea of a self-balloon-expandable stent and was told to submit a report, which he did later. This proved to be a smart move; the report served as documentation of his first concept when he applied for a patent in 1985. Palmaz then moved to Texas, joining the University of Texas at San Antonio, and began his research work on the stent. He presented the first paper on animal experiments with stents in 1984 and applied for a patent in 1985. With grant support and more research to demonstrate stents' potential, he was able to get Johnson & Johnson interested and eventually they licensed the stent. After that, Palmaz concentrated on clinical trials and became the principal investigator for the iliac stent trial.[35] The FDA approved the stent for iliac artery disease in 1991; it was the first vascular stent ever to be approved by the FDA. The first stent placement in humans was in Germany in 1987 and in 1988 the first coronary stent placement was performed in Brazil.[34]

In 1994, the coronary stent was approved by the FDA for clinical use. From there began a new treatment era for coronary artery disease as well as for stenotic lesions of peripheral, visceral, and carotid arteries. In 1988, at the Transcatheter Cardiovascular Therapeutics (TCT) meeting in Washington, Julio Palmaz and Juan Parodi, two Argentineans, connected and thus began the stent-graft for abdominal aortic aneurysm.[36]

Endovascular Graft for Aortic Aneurysm

Ever since the beginning of surgery, surgeons have always been interested in the treatment of aortic aneurysms. From ligation and wiring to open prosthetic graft replacement, medical literature has amply documented the development of aortic surgery. The advent of the endovascular graft, introduced by Juan Parodi, has changed dramatically the history of the treatment of aortic aneurysms. Most importantly, endovascular graft opened a new avenue for treatment of vascular disease and gave patients with prohibitive surgical risks a chance of survival.

Parodi conceived the concept of endovascular graft while a resident at Cleveland Clinic in 1976.[37] He thought that perhaps he could take advantage of the size of the arteries and enter in a retrograde fashion from the femoral artery, compressing the graft into a tube and then releasing it inside the aneurysm, excluding the area of dilatation with a kind of covered metal component called a "cage." The cage was made of two zigs of elastic stainless steel joined by two bridges of the same material welded and covered with a Dacron graft. That was the initial prototype Parodi designed n 1976. He did animal studies and the crude prototype did not do well in the experiment.

At the 1988 TCT meeting, Parodi met Palmaz, who presented his initial results with balloon-expandable stents in animals. Parodi told Palmaz after the meeting, "Your stent could be attached to a piece of bypass graft and used to bypass aortic aneurysm." With that, they decided to work together.[37] Palmaz gave Parodi several stents to experiment with in Argentina. They applied for and obtained a patent on the idea of using stents to fix a piece of bypass graft to the aorta. Palmaz also performed animal studies at his Texas facility. Back home in Argentina, Parodi worked with engineers to redesign the Palmaz stent to enlarge it and enable the stent to open vessels up to 40 mm. They then asked permission from Johnson & Johnson to produce these devices.[35]

In 1990, Argentina's preisident asked Parodi to care for a friend who had an aneurysm but could not undergo surgery because of poor surgical risks. The patient also had back pain and was concerned about the aneurysm rupturing. After informing the patient about the use of an experimental device and its risks, the patient elected to proceed. Parodi invited Palmaz to be part of the first treatment and, in September 1990, they performed an aortic-aortic graft, which went amazingly well for being the first of its kind. (Figure 1–11) The patient was having dinner after 2 hours and was walking the next day. The patient survived for 9 years although he needed a secondary procedure to correct the distal neck dilatation. He died of pancreatic cancer.[36] Figure 1–12 shows the postoperative arteriogram. Parodi reported 5 more cases in a landmark article in the *Annals of Vascular Surgery* in 1991.[38] Interestingly, like Fogarty, Parodi submitted the same paper first to the *Journal of Vascular Surgery* and was rejected for publication. The JVS editors thought Parodi's idea was crazy, one even angrily asking Parodi why the procedure was even necessary when the medical community already had the perfect treatment for aneurysms.[37] Despite this setback,

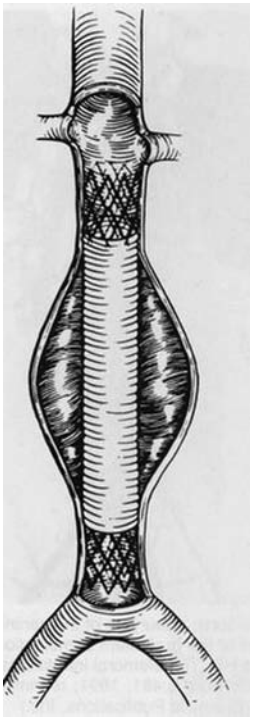

Figure 1-11. Graft-stent combination with both cephalic and caudal stents. (From Parodi JC, Palmaz JC, Barone HD. Transfemoral intraluminal graft implantation for abdominal aortic aneurysms. *Ann Vasc Surg* 1991;5:491–499. Reproduced by permission.)

Parodi continued to work on the project and presented his results at a meeting where John Bergan complimented his work. Bergan asked Parodi to submit the paper to the *Annals of Vascular Surgery,* and it was published in 1991, accompanied by a commentary from Bergan who enthusiastically endorsed the new technique.[38] The article became a landmark publication and changed the direction of surgical care of aortic aneurysms.

While Parodi conceived the idea of endovascular graft, other investigators also had similar ideas.[39] These include Ersek in 1972[40] and Choudhury in 1979.[41] In the

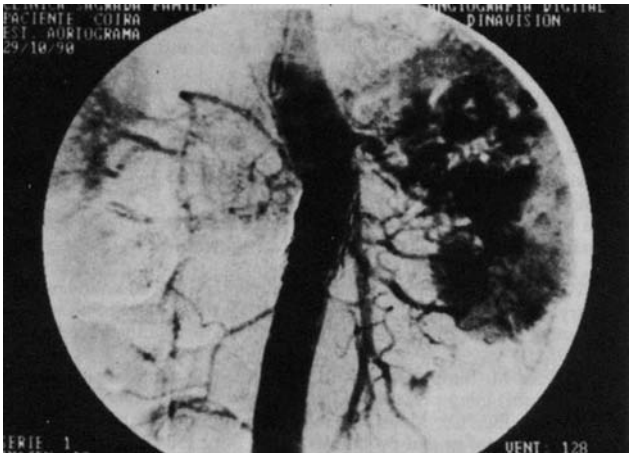

Figure 1-12. Follow-up arteriogram of a patient in whom a 6-cm infrarenal abdominal aortic aneurysm was treated 58 days previously. (From Parodi JC, Palmaz JC, Barone HD. Transfemoral intraluminal graft implantation for abdominal aortic aneurysms. *Ann Vasc Surg* 1991;5:491–499. Reproduced by permission.)

TABLE 1-1. EARLY INVESTIGATORS OF ENDOVASCULAR GRAFT FOR AAA AND THEIR ANCHORING DEVICE

1979	Choudhury[41]	Mechanical attachment with pins
1986	Kornberg[47]	Hooks at proximal end
1986	Balko[42]	Polyurethane graft with nitinol stent
1988	Palmaz[48]	Thin-wall Dacron graft sewn to a Palmaz expandable stent
1988	Lazarus[46]	Balloon staple hooks to aorta
1989	Mirich[49]	Gianturco wire stent with covered nylon graft

1980s, more investigators reported the use of endovascular graft for aortic aneurysm. (Table 1–1) The number of investigators grew more in the decade of the 1990s and use in humans began to appear in the literature. Figure 1–13 shows the experimental intraluminal polyurethane graft of Balko.[42] A Russian surgeon in 1984 was the first to perform dilatation and endovascular prosthetic grafting of the external iliac artery.[43] Prior to Parodi's report on the use of endovascular graft in 5 patients, a Russian surgeon, N. L. Volodos of the Ukraine, performed the world's first endovascular graft for aortic aneurysm in 1987.[44] In the US, the first endovascular graft for aortic aneurysm was performed by Veith and his colleagues, with Parodi in attendance, at the Montefiore Medical Center on November 23, 1992.[45]

In the early 1990s, Endovascular Technologies (EVT) started working on the graft designed by Lazarus.[46] (Figure 1–14) The first implantation of the EVT endoluminal prosthesis occurred at the UCLA Medical Center on February 10, 1993. Northwestern joined the study in late 1993 and performed the first endovascular graft on December 14, 1993. Since then, numerous device manufacturers such as Medtronic, Boston Scientific, Guidant, W.L. Gore, and many others have joined in the production of endovascular grafts with different types of anchoring devices. All are, however, placed through the femoral artery transluminally. At present, many multi-center trials have proven that endovascular graft is a valid alternative procedure to open surgery with comparable results.

The recent success of endovascular technology opens a new avenue for transcatheter endovascular treatment of vascular lesions. Besides balloon angioplasty, stents, and endovascular grafts, other technologies such as atherectomy, vena cava filter, subintimal angioplasty, transcatheter embolization, thrombolytic therapy, and

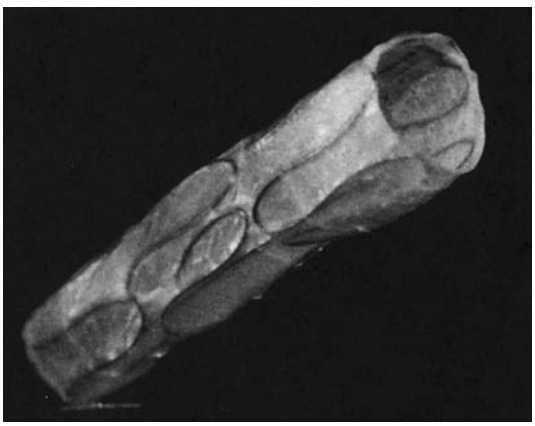

Figure 1-13. The intraluminal polyurethane prosthesis of Balko. (From Balko A et al. Transfemoral placement of intraluminal polyurethane prosthesis for abdominal aortic aneurysm. *J Surg Res* 1986;40:305–309. Reproduced by permission.)

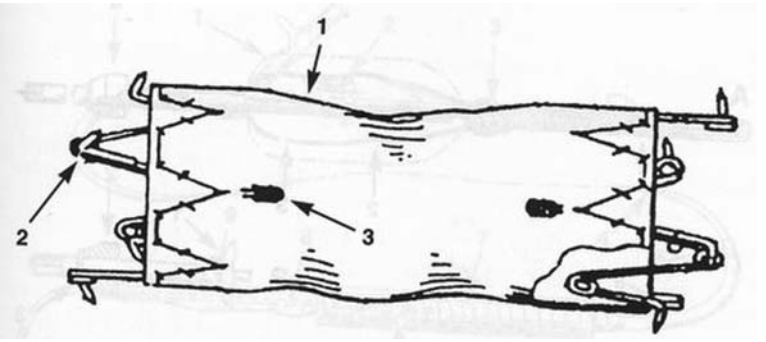

Figure 1-14. Endovascular graft by Lazarus. 1. The graft. 2. Attachment hooks. 3. Radioopaque marker for alignment of the graft. (From Lazarus HM. Intraluminal graft device: System and method. US Patent Number 4,787,799,1988. Reproduced by permission).

many others are now available to offer new and innovative treatment of a wide variety of clinical problems. Endovascular technology is going to stay and will be an important treatment armamentarium for vascular surgeons.

REFERENCES

1. Brooks B. Intra-arterial injection of sodium iodide: Preliminary report. *JAMA* 1924;82: 1016–1019.
2. Moniz E. L'encephalographie arterielle, son importance dans la localization des tumeurs cerebrales. *Rev Neurol* 1927;2:72–90.
3. dos Santos R, Lamas A, Pereria-Caldas J. L'arterriografia dos members de l'aorta et de ses branches abdominals. *Bull Mem Soc Natl Chir* 1929;55:587–601.
4. Seldinger SI. Catheter replacement of the needle in percutaneous arteriography: A new technique. *Acta Radiol* 1953;39:368–376.
5. Yao JST, Pearce WH. Dedication–Michael E. Debakey. In Yao JST, Pearce WH, eds. Arterial Surgery: Management of Challenging Problems. Stamford CT: Appleton & Lange, 1996, pp xv–xx.
6. Fogarty TJ, Cranley JJ, Krause RJ, et al. A method for extraction of arterial emboli and thrombi. *Surg Gynec & Obstet* 1963;116:241–244.
7. Dotter CT, Judkins MP. Transluminal treatment of arteriosclerotic obstruction. Description of a new technic and preliminary report of its application. *Circulation* 1964;30:54–70.
8. Horsley JS. Aneurism. In Horsley JS. Surgery of the Blood Vessels. CV Mosby Co., 1915.
9. Blakemore AH. Progressive constrictive occlusion of the abdominal aorta with wiring and electrothermic coagulation: A one stage operation for arteriosclerotic aneurysms of the abdominal aorta. *Ann Surg* 1951;133:447–462.
10. de Takats G, Marshall MR. Surgical treatment of arteriosclerotic aneurysms of the abdominal aorta. *Arch Surg* 1952;64:307–319.
11. Murphy JB. Removal of an embolus from the common iliac artery with re-establishment of circulation in the femoral. *J Am Med Assoc* 1909;LII:1661–1663.
12. Greenfield LJ, Kimmell GO, McCurdy WC 3rd: Transvenous removal of pulmonary emboli by vacuum-cup catheter technique. *J Surg Res* 1969;9:347–352.
13. Murphy JB. Resection of arteries and veins injured in continuity—end-to-end suture—experimental and clinical research. *Med Rec (NY)* 1897;51:73–88.

14. The first minimally invasive therapy. *Endovascular Today*, November/December 2004, pp 38–40.
15. Rob CG. A history of arterial surgery. *Arch Surg* 1972;105:821–823.
16. Friedman SG. Thomas Fogarty. In Friedman SG. A History of Vascular Surgery 2nd ed. Malden MA: Blackwell-Futura, 2005.
17. Payne MM. Charles Theodore Dotter—The father of intervention. *Tex Heart Inst J* 2001;28: 28–38.
18. Friedman SG. Charles Dotter: Interventional radiologist. In Friedman SG. A History of Vascular Surgery 2nd ed. Malden MA: Blackwell-Futura, 2005.
19. Orron DE, Kim D. Percutaneous transluminal angioplasty. In Kim D, Orron DE. Peripheral Vascular Imaging and Intervention. St. Louis: Mosby-Year Book, 1992.
20. Zeitler E, Grunzig A, Schoop W, eds. Percutaneous vascular recanalization: techniques, application, clinical results. Berlin: Springer Verlag, 1978.
21. Zeitler E, Richter EI, Roth RJ, et al. Results of percutaneous transluminal angioplasty. *Radiology* 1983;146:57–60.
22. Bergan JJ. Endovascular surgery: How we arrived here. *Endovascular Today,* November/ December 2004, 33–35.
23. Gruentzig A. Transluminal dilatation of coronary artery stenosis. *Lancet* 1978;1:263.
24. Balas PE. The history of vascular surgery in Europe. In Liapis CD, Balzer K, Benedetti-Valentini F, Fernandes e Fernandes J., eds. *Vascular Surgery (European Manual of Medicine).* Berlin: Springer, 2007.
25. Mathias, K. Perkutane transluminate katheterbehnundlung supraaortaier arteirobstruktionen. *Angiology* 1981;3:47–50.
26. Bachman DM, Kim RM. Transluminal dilatation for subclavian steal syndrome. *AJR Am J Roentgenol* 1980;135:995–996.
27. Theron J, Courtheous P, Henniot JP, et al. Angioplasty of supraaortic arteries. *J Neurorad* 1984;11:187–200.
28. Rabkin IH, Matevosoa AL, Gotman NL. Roentgen-Endovascular Surgery. Moscow: Media Sphere, 1987.
29. Ring ME. How a dentist's name became a synonym for a life-saving device: The story of Dr. Charles Stent. *J Hist Dentistry* 2001;49:77–80.
30. Dotter CT. Transluminally-placed coilspring endarterial tube grafts. Long-term patency in canine popliteal artery. *Invest Radiol* 1969;4:329–332.
31. Chuter TAM. Endovascular grafts: History of minimally invasive treatment of vascular disease. In Chuter TAM, Donayre CE. White RA, eds. Endoluminal Vascular Prostheses. Boston: Little Brown & Co., 1994.
32. Kurdamov CV, Khandros LG. Thermoplastic phase equilibrium in martensiter transformations. USSR Academy of Science Reports 1949;66:211.
33. Dotter CT, Bushmann RW, McKinney MK, et al. Transluminal expandable nitinol coil stent grafting: Preliminary report. *Radiology* 1983;147:259–260.
34. The advent of stenting. *Endovascular Today*, November-December 2004, 45–49.
35. Palmaz JC, Richter GM, Noeldge G, et al. Intraluminal stents in arteriosclerotic iliac artery stenosis: Preliminary report of a multicenter trial. *Radiology* 1988;168:727–731.
36. The EVAR revolution. *Endovascular Today,* November-December 2004, 51–54.
37. Parodi JC. Ten years of endovascular aneurysm repair. *Endovascular Today,* March 2007 Supplement, 4–5.
38. Parodi JC, Palmaz JC, Barone HD. Transfemoral intraluminal graft implantation for abdominal aortic aneurysms. *Ann Vasc Surg* 1991;5:491–499.
39. Chuter TAM, Donayre CE, White RA, eds. Endoluminal Vascular Prostheses. Boston: Little Brown & Co., 1995.
40. Ersek RA. Method for fixing prosthetic implants in a living body. US Patent #3,657,744, 1972.
41. Choudhury MH. Method for performing aneurysm repair. US Patent #4,140,126, 1979.
42. Balko A, Piasecki GJ, Shah DM, et al. Transfemoral placement of intraluminal polyurethane prosthesis for abdominal aortic aneurysm. *J Surg Res* 1986;40:305–309.

43. Pokrovkly A, Bogatov YP. Vascular surgery in Russia. *J Angiology and Vascular Surgery* 2000;6:8–20.
44. Volodos NL, Karpovich IP, Shekhanin VE, et al. A case of distant transfemoral endoprosthesis of the thoracic artery using a self-fixing synthetic prosthesis in traumatic aneurysm. *Grudnaia Khirurgiia* 1988;6:84:86.
45. Veith F. Personal communication.
46. Lazarus HM. Endovascular grafting for the treatment of abdominal aortic aneurysms. *Surg Clin North Am* 1992;72:959–968.
47. Kornberg E. Aortic graft device and method for performing an intraluminal abdominal aortic aneurysm repair. US Patent #4,562,596,1986.
48. Palmaz JC. Expandable intraluminal graft. Method and apparatus for implanting an expandable intraluminal graft. US Patent #4,776,337,1988.
49. Mirich D, Wright KC, Wallace S, et al. Percutaneously placed endovascular grafts for aortic aneurysms: Feasibility study. *Radiology* 1989;170:1033–1037.

2

Understanding the Diffusion of Vascular Procedures: A Quarter of a Century Experience

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Many surgical specialties have experienced tremendous change over the past decades because of the introduction of minimally invasive techniques, and vascular surgery is no exception.[1] These new techniques have the potential to allow more higher-risk patients to be treated, increase the likelihood of low-risk patients to opt for intervention, and decrease overall mortality and morbidity. Indeed, in other areas, minimally invasive procedures such as laparoscopic cholecystectomy have led to a major broadening of the patient populations that are treated.[2] Technological innovations are occurring in parallel with major demographic shifts in industrialized nations. The U.S. population has continued to age: the population over 65 increased by 43% from 1979 to 2003.[3] This rapidly changing scenario raises major questions. Has the utilization of various vascular procedures changed over time? Have these changes in utilization affected specific subsets of the population? How have these trends affected outcome and length of stay? How can we expect vascular surgery to evolve in the years to come?

In this chapter, we will address these questions by analyzing trends in the volume of major vascular interventions performed in the United States over a quarter of a century (1979–2003). We utilized the National Hospital Discharge Survey (NHDS) and evaluated eight major inpatient vascular categories (carotid intervention, abdominal aortic aneurysm repair, lower extremity revascularization, renal-mesenteric intervention, catheter-based intervention, thoracic-subclavian procedures, amputations, and a group of "other" vascular procedures). We examined the number of patients treated

and their characteristics, analyzed their hospital resource use, and then predicted the potential impact of these trends on the future face of vascular surgery.

METHODS

Data Source

Discharge data for patients, who underwent a vascular procedure requiring hospitalization, were extracted from the National Hospital Discharge Survey (NHDS) database for the years 1979–2003. The NHDS is conducted annually by the National Center for Health Statistics (NCHS). This database collects medical and demographic information from a sample of discharge records selected from a national sample of acute care hospitals, exclusive of federal, military and Veterans Administration hospitals. Hospital data in the survey include but are not limited to birth date, sex, race, ethnicity, marital status, zip code, payment source, in-hospital mortality, LOS, and discharge status. We have published a more extensive description of the database elsewhere.[4]

Study Population

Our patient population was extracted from the NHDS database using ICD-9 procedural codes. The discharges were grouped into eight procedural groups: carotid, thoracic subclavian, abdominal aortic aneurysm (AAA), renal-mesenteric, catheter-based, amputations, lower extremity revascularizations, and all other. Table 2–1 depicts a complete list of procedure groups with the ICD-9 codes subsumed by each. The "catheter-based" category included angioplasty, noncoronary stent placement, or intraluminal thrombolytic therapy. This would include carotid, renal and mesenteric as well as lower extremity arterial stenting. Diagnostic angiography without any other intervention was not included in this category since many of these procedures are performed on outpatients and would not be detected in our patient population. Prior to the advent of a separate endovascular code for AAA repair in October 2000[5], endovascular repairs were included under ICD-9 code 39.52. The "amputation" category was further subdivided into major and minor amputations. The "lower extremity revascularization" category included both infrainguinal revascularization and aortoiliac reconstruction. The "all other category" included abdominal vein reconstruction, arm vessel reconstruction, leg vein reconstruction, trauma, and others. We did not include procedures for varicose veins or angio-access as both are often outpatient interventions that would be greatly underestimated by the NHDS, which includes inpatients only.

Statistical Analysis

For the per capita calculations, annual nationwide census information was obtained from the National Census Bureau for the years 1979–2003. The per capita rate for the total number of discharges, total number of vascular procedures, and each individual vascular category were calculated by dividing the total number of discharges or procedures by the national population. All rates are expressed as the rate of the procedure per 100,000 population. These calculations were standardized for the respective subgroups studied: age, race, gender. We used simple linear regression techniques to determine trends and whether there was a relationship between the procedure rates and time. All data were analyzed using SAS statistical program version 8.0 (SAS Institute Inc., Cary, NC).

TABLE 2-1. LIST OF PROCEDURE GROUPS WITH ICD9-CM CODES

Category Assigned	ICD-9	Definition
Carotids	38.12	Endartarectomy, carotid
	38.02	Incision of vessel with embolectomy or thrombectomy, carotid
	38.32	Resection of vessel with anastomosis, carotid
	38.42	Resection of vessel with replacement, carotid
	38.62	Other excision of vessels, carotid
	38.82	Other surgical occlusion of vessels, carotid
	39.80	Operation on carotid body and other vascular bodies
Thoracic Subclavian	39.22	Aorto-subclavian-carotid bypass
	39.23	Other intrathoracic vascular shunt or bypass
	38.05	Incision of vessels, thoracic vessel
	38.15	Endartarectomy, thoracic vessel
	38.35	Resection of vessel with anastomosis, thoracic vessel
	38.45	Resection of vessel with replacement, thoracic vessel
	38.65	Other excision of vessels, thoracic vessel
	38.85	Other surgical occlusion of vessels, thoracic vessel
	39.54	Other repair of vessels, thoracic vessel
AAA	38.34	Resection of vessel with anastomosis, abdominal aorta
	38.44	Resection of vessel with replacement, abdominal aorta
	38.64	Other excision of vessels, abdominal aorta
	39.52	Other repair of vessels, abdominal aorta
Renal-Mesenteric	39.24	Aorta-renal bypass
	39.26	Other intra-abdominal vascular shunt or bypass
	38.16	Endartarectomy, abdominal arteries
Catheter-Based		*= Percutaneous + Endo-Aorta*
Percutaneous	39.50	Angioplasty or atherectomy of non-coronary vessel
	39.90	Insertion of non-drug eluting, non-coronary artery stent(s)
	99.10	Injection or infusion of thrombolytic agent
	39.79	Other endovascular repair (of aneurysm) of other vessels
Endo-Aorta	39.71	Endovascular implantation of graft in abdominal aorta
Total Amputation		*= Major Amputation + Minor Amputation*
Major amputation	843	Revision of amputation stump
	8410	Lower limb amputation, not otherwise specified
	8413	Disarticulation of ankle
	8414	Amputation of ankle through malleoli of tibia and fibula
	8415	Other amputation below knee
	8416	Disarticulation of knee
	8417	Amputation above knee
Minor amputations	8411	Amputation of toe
	8412	Amputation through foot
Lower Extremity Revascularization		*= Infra-inguinal Reconstruction + Aortoiliac Revasc*
Infrainguinal Reconstruction	3929	Other (peripheral) vascular shunt or bypass
	3808	Incision of vessels, lower limb arteries
	3818	Endartarectomy, lower limb arteries
	3838	Resection of vessel with anastomosis, lower limb arteries
	3848	Resection of vessel with replacement, lower limb arteries
	3888	Other surgical occlusion of vessels, lower limb arteries
Aortoiliac Revascularization	3925	Aorta-iliac-femoral bypass
	3804	Incision of vessels, abdominal aorta
	3814	Endartarectomy, abdominal aorta
	3884	Other surgical occlusion of vessels, abdominal aorta

RESULTS

Trends in Vascular Interventions

Over a 25-year period, the number of vascular procedures performed in the United States more than doubled, increasing from 358,000 in 1979 to 785,000 in 2003 (Figure 2–1A) ($p < 0.0001$). This trend is not simply a reflection of population growth as we see a similar trend in the per capita use of vascular surgery over the same time period: from 159 to 270 procedures per 100,000 population (Figure 2–1B) ($p < 0.0001$).

Age

The increase in the use of in-patient vascular procedures is largely a result of the major growth in vascular procedures performed in the elderly. While the number of procedures per capita remained stable for the 15%–44 and 45- to 64-year-old cohorts, interventions in the 65- to 74- and > 75-year-old patients increased significantly (Figure 2–2A). For patients in the 65- to 74 year-old category, there was a 55% increase in per capita admissions over the 25-year period of study ($p < 0.0001$), and for patients over the age of 75, the number of admissions increased by 80% ($p < 0.0001$). Over the 25 years of analysis, men were admitted more frequently than women for vascular interventions; however, the per capita admissions for women grew at the same rate as for men ($p = 0.9974$) (Figure 2–2B).

Race

Our data suggest interesting trends in vascular procedural usage by race. Even though, in absolute numbers, whites account for the major share of procedures, the per capita utilization for blacks, starting in 1989, has consistently been higher than for whites (Figure 2–2C). To understand the determinants of such difference, we analyzed the distribution by race of the most frequent procedures. From 1999 to 2003, more catheter-based procedures were performed in blacks than in whites, although catheter-based procedures increased at a similar rate among both races during the interval studied (Figure 2–3). Throughout all the years analyzed, blacks had two- to three-fold higher rate of amputation than whites. Amputation was the most frequent vascular procedure in 1979 and the second most frequent in 2003 after catheter-based procedures (Table 2–2). Data from other races such as Native Americans, Natives of Alaska, Pacific Islanders, and Asians were not sufficient to obtain accurate estimates of procedure usage from NHDS (NHDS estimates are based on a sample of discharge records selected from a national sample of acute care hospitals;

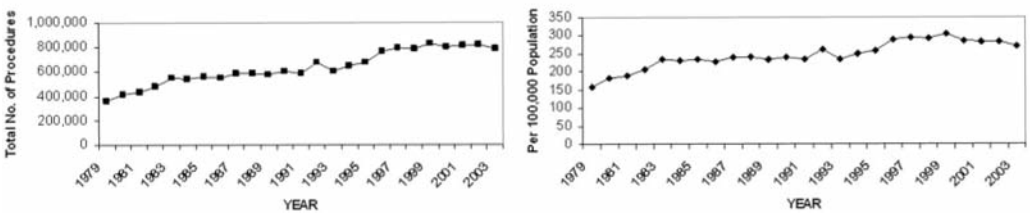

Figure 2-1. A. Growth in total number of vascular procedures. **B.** Growth in per capita rate for all vascular procedures from 1979 to 2003.

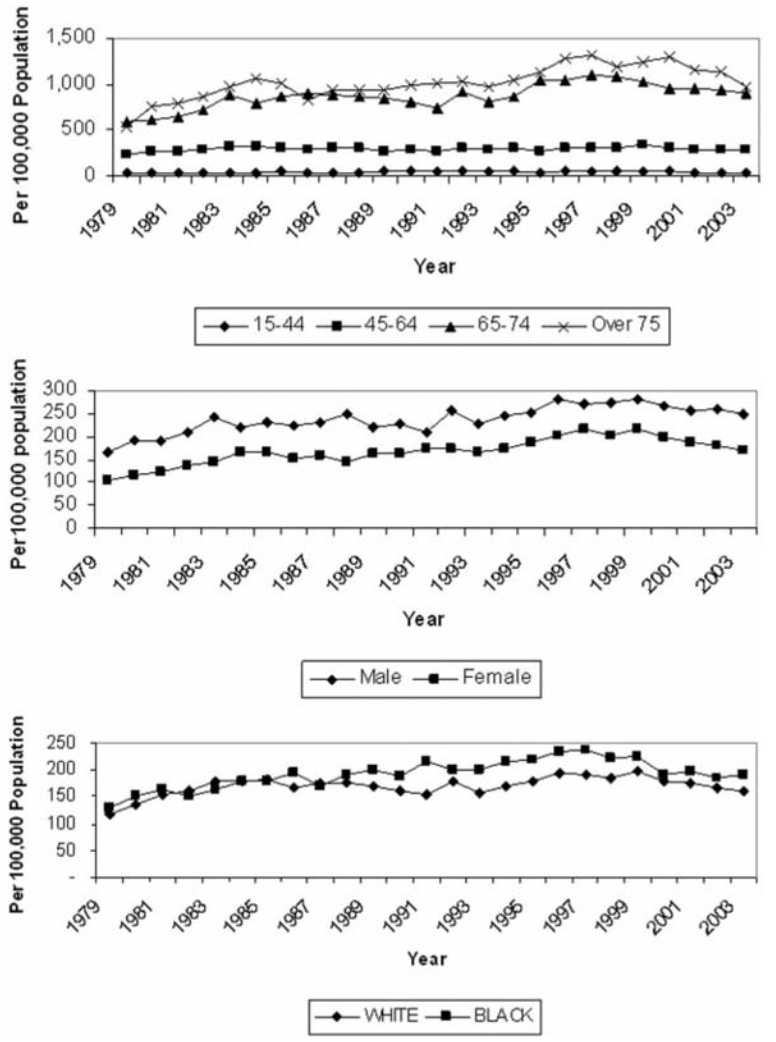

Figure 2-2. Per capita rates of vascular procedures adjusted by age groups **A.**, gender **B.**, and race **C**.

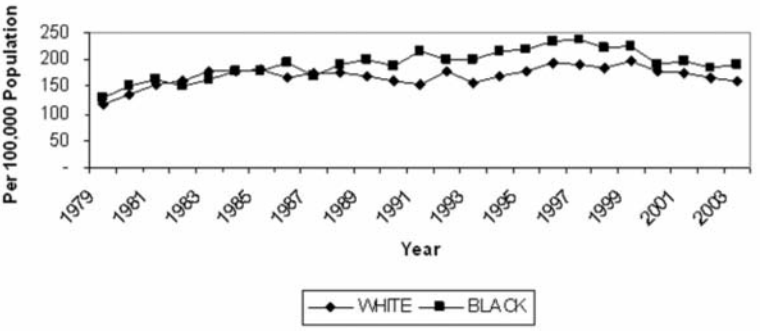

Figure 2-3. Per capita rates of catheter-based procedures adjusted by race.

TABLE 2-2. MOST COMMON PROCEDURE CATEGORIES IN DESCENDING ORDER: 1979–2003

1979				2003			
Rank	Category	Total No.	%	Rank	Category	Total No.	%
1	Amputation	97,064	43.0	1	Catheter-based	212,508	73.1
2	Lower extremities	96,033	42.8	2	Amputation	145,291	50.0
3	Other	69,726	31.0	3	Lower extremities	133,702	46.0
4	Carotid	57,994	25.8	4	Carotid	122,233	42.0
5	AAA	27,017	12.0	5	Other	96,051	33.0
6	Thoracic-subclavian	6,994	3.1	6	AAA	39,490	13.6
7	Renal-Mesenteric	3,215	1.4	7	Thoracic-subclavian	28,932	9.9
8	Catheter-based (1980)	125	0.1	8	Renal-Mesenteric	7214	2.5

PC: per capita (Number of procedures per 100,000 population);
AAA: abdominal aortic aneurysm

statistical reliability of these estimates requires a minimum sample size). Moreover, it was not possible with the NHDS to identify people of Hispanic ethnicity (the U.S. census began providing data on ethnicity stratified by race only from the year 2000).

Trends in Specific Vascular Procedures

During this time period, we also observed a major change in the distribution of vascular procedures (Table 2–2). Nationally, the rate of AAA interventions increased from 12.0 per 100,000 in 1979 to 22.7 per 100,000 in 1992, but then returned to almost 1979 levels in 2003 (Figure 2–4A). There was a biphasic pattern in the utilization of carotid interventions (Figure 2–4B). From 1979 to 2003, the overall number of carotid procedures increased from 25.8 to 42.0 per 100,000, but there were two interval peaks in 1985 and 1997. Figure 2–4C depicts the per capita trends for renal/mesenteric open surgeries. The number of these interventions remained small over the time period analyzed, oscillating around two per 100,000 population. The use of lower extremity revascularization (LER), including both aortoiliac and infrainguinal reconstructions, reached a peak in 1989, and then subsequently declined almost to 1979 numbers by the year 2003 (71.7 versus 45.6 per 100,000 in 1989 and 2003, respectively) (Figure 2–4D). The most dramatic change over time occurred in the category of catheter-based interventions (125 procedures performed in 1980). By 2003, this number had increased substantially to 73.1 per 100,000 (total of 212,000 procedures) (Figure 2–4E). The per capita rate (per 100,000) of total amputations increased from 43.0 in 1979 to 61.4 in 1996, then decreased to 50.0 in 2003 (Figure 2–4F). The overall increase (16.3%, $p = 0.004$) in per capita amputations over 25 years appears to be mainly due to the increase in minor amputations (57% increase). Figure 2–4G depicts the trends in thoracic and subclavian surgery. The per capita rate of this intervention rose from 3.1 in 1979 to 9.9 in 2003 ($p < 0.001$). The rate of interventions in the "other" category (Figure 2–4H), which includes procedures that range from venous reconstruction to trauma, more than doubled from 1979 to 1992 (1979: 31.0 per 100,000; 1992: 71.5 per 100,000), to return almost to the 1979 levels in 2003 (33.3 per 100,000).

Trends in Length of Stay

There was a marked decrease in length of stay (LOS) over the 25-year period that was analyzed: mean LOS for vascular procedures fell from 17.9 days in 1979 to 8.2 days in 2003,

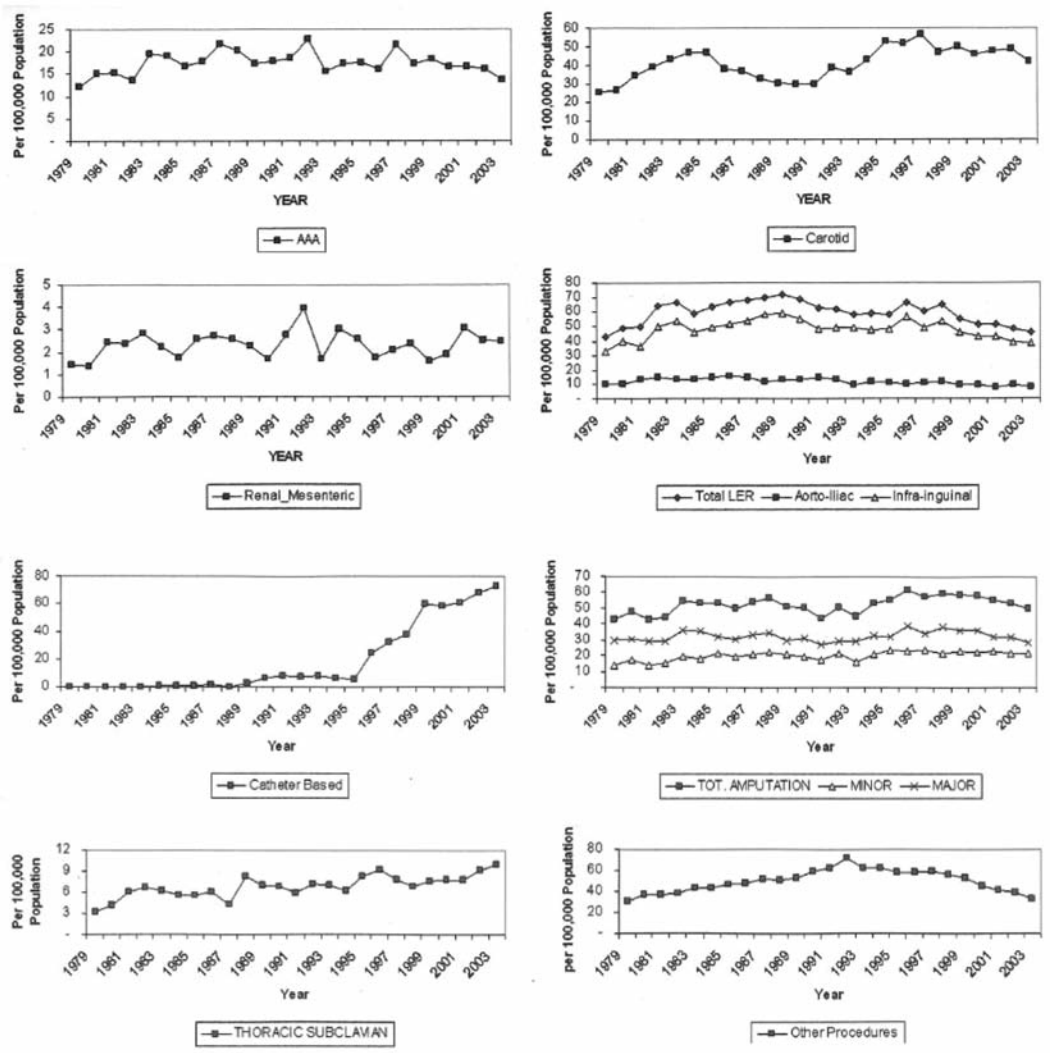

Figure 2-4. Per capita rates for abdominal aortic aneurysm **A.**, carotid endarterectomy **B.**, renal and mesenteric interventions **C.**, lower extremity revascularization procedures **D.**, cathter-based interventions **E.**, amputations **F.**, thoracic-subclavian and other interventions **G.**, and other procedures **H**.

a 54% decrease (Figure 2–5). In comparison, the national mean LOS for all hospitalizations decreased from 7.2 days in 1979 to 4.7 in 2003 (only a 35% reduction). Median LOS, perhaps a better indicator than mean, depicts a similar scenario: LOS went from 11 to 5 days (54% reduction) for vascular surgeries and from four to three days (25% reduction) for all hospitalizations. In 1979, approximately 80% of all vascular patients were hospitalized for seven days or more, whereas in 2003, only 39% remained in the hospital for this duration (Figure 2–6). By 2003, the number of patients discharged in less than or equal to 24 hours had increased to 20%, from 4% in 1979. The sharpest decline in LOS was observed in older patients (Figure 2–7). Indeed, in 1979, the mean LOS for different age groups was 22.3 days for patients > 70 years; 19.2 days (65 to 69); 17.3 days (45 and 64); and 12.4 days (15 and 44). Since 1995, the mean LOS has become essentially equivalent across all age groups, standing at about 7.5 days.

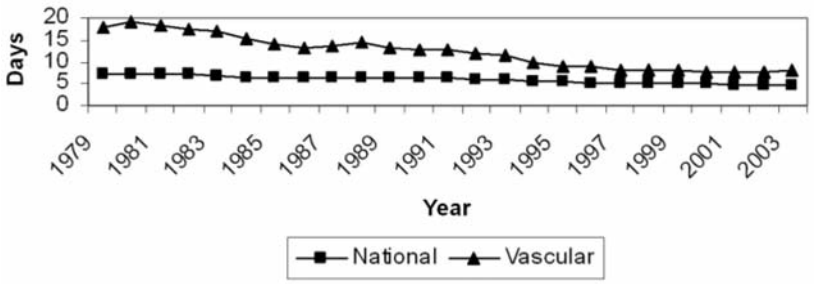

Figure 2-5. Mean length of stay for vascular procedures versus national (all hospitalizations).

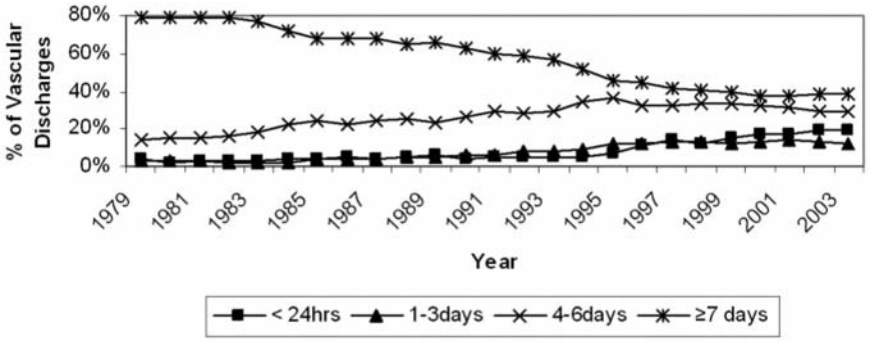

Figure 2-6. Vascular procedures by length of stay.

DISCUSSION

Over the past 25 years, there has been a striking increase in the number of vascular interventions performed in this country (> 69% increase per capita) (Figure 2–1). Most of this growth occurred during the first 20 years, whereas over the past five years, this trend has leveled off. The growth in vascular interventions can be attributed to several factors. In part, it reflects the growth of the overall U.S. population and the changes that have occurred in the age distribution. It is well established that the prevalence of vascular disease increases with age. Thus, the aging of the population has contributed substantially

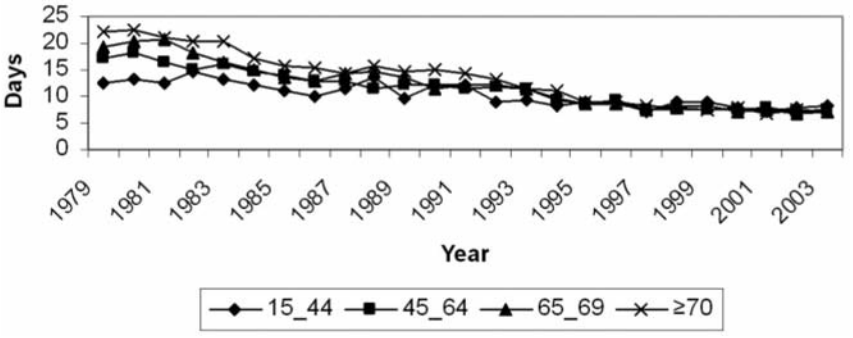

Figure 2-7. Length of stay stratified by age.

to the increase in the number of vascular interventions. However, neither the overall increase in the population nor the increase in the number of elderly completely explains the trends observed for vascular volume that we observed. We found that when we stratified patients by age, the per capita rate of vascular intervention still increased for patients older than 65 (Figure 2–2A). Thus, other factors beyond age and size of the population have contributed to these changes in volume. It is possible that the trend toward minimally invasive vascular procedures might contribute to the increased utilization of intervention. However, it is surprising then, that the overall frequency of vascular intervention has plateaued over the past several years, despite the continued development of minimally invasive techniques. It is also possible that over the past 20 years, awareness of vascular disease and its morbidity has increased. Although this hypotheses is plausible, current physician and patient awareness of vascular disease remains low.

Beginning in 1989, the per capita rate of admission for vascular procedures was higher among blacks than whites (Figure 2–2C). However, an important component of the overall difference in the utilization of vascular procedures between blacks and whites is related to a substantial disparity in the rate of amputations. In fact, the higher rate of utilization among blacks is erased if amputation is eliminated from the analysis. For the remaining reconstructive vascular procedures, access to intervention appears to be equivalent for blacks and whites (at least since 1990), and the diffusion of the new endovascular techniques appears to have occurred at a similar rate among both races (Figure 2–3). Previous studies have shown that socioeconomic status and race influence access to primary and preventive care.[6,7] Access disparities along with race-specific anatomical characteristics of lower extremities unfavorable to vascular surgery may contribute to the higher rate of amputations observed among blacks.[6-12]

Amputation of all or part of the limbs was the most common procedure in 1979 and the second most common in 2003 (Table 2–2). However, after reaching a peak in 1996 (61.4 per 100,000), the rate of amputations has been decreasing, perhaps as a consequence of improved techniques of limb salvage such as catheter-based lower extremities interventions. Changes in medical management of amputation risk factors, and newer wound care methodologies or more timely interventions may also have contributed to the recent decline in amputation rates. This apparent improvement in outcomes deserves further study.

In addition to the above mentioned shifts in the demographic make-up of the population, the field of vascular surgery has been revolutionized by technological innovations. Over the past 15 years, we have witnessed the diffusion of minimally invasive techniques. In our dataset, this is reflected in the dramatic growth of catheter-based procedures, especially after 1995 (Figure 2–4E). The less invasive nature of these techniques may have shifted the threshold risk-benefit ratio to favor intervention for less ill patients, thereby increasing the proportion of the population that might be candidates for intervention. Moreover, because of the faster rate of recovery generally observed after percutaneous procedures, we might expect that the increased use of these techniques also contributed to the reduction in LOS. However, minimally invasive techniques began to disseminate rapidly beginning in 1996 (Figure 2–4E), whereas much of the observed reduction in mean LOS had already occurred by the mid 1990s (Figures 2–5 to 2–7). The introduction of a different mechanism of reimbursement (payment by diagnostic related group: DRG) in the early 1980s created economic incentives for hospitals to reduce their LOS.[13] This change in the payment system perhaps was the major driver in the progressive reduction of LOS for vascular procedures. Technological improvement and improvement in outcomes may have also

contributed since the reduction in mean LOS for vascular surgery was much greater than that observed for all hospitalizations.

The changes in vascular volume were not uniform and varied with the intervention. For example, the frequency of elective AAA repair did not change over the 25-year period of evaluation (p = ns) (Figure 2–4A). In the last few years, we have observed a downward trend in the number of AAA repairs, which may be a result of recent reports recommending AAA repair in only those patients with aneurysms greater than 5.5 centimeters.[14] However, current initiatives to increase the frequency of and reimbursement for screening, and perhaps the broader use of endovascular AAA repair, may counterbalance this trend in the future. We recently reported data from New York state that showed an increase in the frequency of aneurysm repair following FDA approval of endovascular devices.[15] However, these local findings are not corroborated by the NHDS national database.

In the early 1980s, there was a steady increase in the number of CEAs performed, a trend that reversed in the late 1980s (1985–1990) (Figure 2–4B). This change in utilization has been previously documented and was apparently related to a number of publications that challenged the benefit of carotid intervention.[16-19] One such review, published in 1988, suggested that 32% of CEAs in the United States were performed for inappropriate indications.[20] However, following the completion of several well-structured, randomized prospective studies, clarifying the indications for and benefits of CEA, the frequency of this procedure began to rise again.[21-22] Our data reveal that the rate of CEA reached a peak in 1997 with a decline over the subsequent years (Figure 2–4B). This may, in part, be related to the increased use of carotid angioplasty and stenting (CAS) (CAS in the NHDS database is categorized as a catheter-based procedure). However, a recent study by our group suggests that both nationally and within several states, the overall frequency of intervention for carotid disease (both surgery and angioplasty) is on the decline.[23]

Although the number of aortoiliac and infrainguinal reconstructions increased significantly during 1980–1990, from 1990 to 2003, there was a decline in the frequency of open surgical interventions for lower extremity vascular disease (Figure 2–4D). This corresponded with the increase in the number of catheter-based interventions (Figure 2–4E). The NHDS does not offer specific information on which vessels were treated percutaneously; however, many of these interventions were designed to treat lower extremity vascular disease. It seems plausible that the increased use of angioplasty/stenting will also lead to a decline of open renal and mesenteric bypasses over time. However, the overall number of these procedures has remained constant, and oscillates around two per 100,000 (Figure 2–4C).

The increase in the overall number of surgical and catheter-based lower extremity interventions is one of the factors that might explain the recent decrease of amputations (Figure 2–4F). This relationship has been shown elsewhere. In Europe, an increase in LER in the 1980s was thought to result in a decrease in the rate of amputation that occurred in the early 1990s.[24-27] Moreover, in Canada, an increase in the use of lower extremity angioplasty appeared to correlate with a decrease in the frequency of amputation.[27]

Several qualifications need to be made regarding the foregoing data. The dataset only addresses inpatient procedures. Thus, a number of vascular interventions that can be performed on outpatients were excluded from this analysis including varicose vein surgery, arteriovenous dialysis access, and diagnostic angiography. Patients who were not admitted to the hospital, but received endovascular interventions in free

standing or hospital-based ambulatory surgery settings have been excluded as well. These interventions contribute substantially to the overall volume of procedures that are performed by vascular surgeons. There are clearly limitations to drawing conclusions from a weighted discharge database. Information derived from large datasets such as the NHDS are based on sampling rather than a complete census. Results are subject to nonsampling measurement errors, which include errors due to hospital nonresponse, missing discharge abstracts, information incompletely or inaccurately recorded on abstract forms, and processing errors. Another limitation of datasets such as the NHDS is the potential for errors in coding. Nevertheless, large datasets are the only valid method available for recognizing national trends in usage and outcomes in medicine, and consequently they are used widely for that purpose.

Our analysis has shown that over the past 25 years, there has been a doubling in the volume and per capita use of vascular procedures that most recently appears to have stabilized. Although the overall volume of vascular interventions has recently leveled off over the past several years, the use of endovascular procedures has grown enormously. New techniques for the medical management of vascular disease such as the use of endothelial progenitor cells, for lower extremity ischemia, or the introduction of new plaque stabilizing drugs, or possible screening initiatives for highly prevalent vascular diseases such as AAA, may have an impact on the rate of vascular surgical interventions to an extent that is difficult to predict. It is likely, however, that the diffusion of endovascular techniques, thanks to the promising outcomes that have been reported, will continue to transform the field of vascular surgery for the foreseeable future.

REFERENCES

1. Pearce WH. What's new in vascular surgery. *J Am Coll Surg*. 2003;196:253–266.
2. Urbach DR, Stukel TA. Rate of elective cholecystectomy and the incidence of severe gallstone disease. *Can Med Assoc J*. 2005;172:1015–1019.
3. http://www.census.gov/: In.
4. Anderson PL, Gelijns A, Moskowitz A, Arons R, Gupta L, Weinberg A, Faries PL, Nowygrod R, Kent KC: Understanding trends in inpatient surgical volume: vascular interventions, 1980–2000. *J Vasc Surg*. 2004;39:1200–1208.
5. National Hospital Discharge Survey PUDFD. U.S. Department of Health and Human Services, Center for Disease Control and Prevention, National Center for Health Statistics, Division of Health Care Statistics, NHDS@cdc.gov.: In, 2001.
6. Gittelsohn AM, Halpern J, Sanchez RL. Income, race, and surgery in Maryland. *Am J Public Health*. 1991;81:1435–1441.
7. Gornick ME, Eggers PW, Reilly TW, et al. Effects of race and income on mortality and use of services among Medicare beneficiaries. *N Engl J Med*. 1996;335:791–799.
8. Feinglass J, Kaushik S, Handel D, et al. Peripheral bypass surgery and amputation: northern Illinois demographics, 1993 to 1997. *Arch Surg*. 2000;135:75–80.
9. Feinglass J, Rucker-Whitaker C, Lindquist L, et al. Racial differences in primary and repeat lower extremity amputation: results from a multihospital study. *J Vasc Surg*. 2005;41:823–829.
10. Fiscella K, Franks P, Gold MR, Clancy CM. Inequality in quality: addressing socioeconomic, racial, and ethnic disparities in health care. *JAMA*. 2000;283:2579–2584.
11. Lavery LA, Ashry HR, van Houtum W, et al. Variation in the incidence and proportion of diabetes-related amputations in minorities. *Diabetes Care*. 1996;19:48–52.
12. Lavery LA, van Houtum WH, Ashry HR, et al. Diabetes-related lower-extremity amputations disproportionately affect Blacks and Mexican Americans. *South Med J*. 1999;92:593–599.

13. Davis C, Rhodes DJ. The impact of DRGs on the cost and quality of health care in the United States. *Health Policy*. 1988;9:117–131.
14. Participants, TUKSAT. Long-Term Outcomes of Immediate Repair Compared with Surveillance of Small Abdominal Aortic Aneurysms. *N Engl J Med*. 2002:1445–1452.
15. Anderson PL, Arons RR, Moskowitz AJ, Gelijns A, Magnell C, Faries PL, Clair D, Nowygrod R, Kent KC: A statewide experience with endovascular abdominal aortic aneurysm repair: rapid diffusion with excellent early results. *J Vasc Surg*. 2004;39:10–19.
16. Brott T, Thalinger K: The practice of carotid endarterectomy in a large metropolitan area. *Stroke*. 1984;15:950–955.
17. Chambers BR, Norris JW. The case against surgery for asymptomatic carotid stenosis. *Stroke*. 1984;15:964–967.
18. Muuronen A. Outcome of surgical treatment of 110 patients with transient ischemic attack. *Stroke*. 1984;15:959–964.
19. Warlow C. Carotid endarterectomy: does it work? *Stroke*. 1984;15:1068–1076.
20. Winslow CM, Solomon DH, Chassin MR, et al. The appropriateness of carotid endarterectomy. *N Engl J Med*. 1988;318:721–727.
21. North American Symptomatic Carotid Endarterectomy Trial Collaborators. Beneficial effect of carotid endarterectomy in symptomatic patients with high-grade carotid stenosis. *N Engl J Med*. 1991;325:445–453.
22. Executive Committee for the Asymptomatic Carotid Atherosclerosis Study. Endarterectomy for asymptomatic carotid artery stenosis. *JAMA*. 1995;273:1421–1428.
23. Nowygrod R, Egorova N, Greco G, et al. Trends and mortality in peripheral vascular surgery. submitted 2005.
24. Eickhoff JH. Changes in the number of lower limb amputations during a period of increasing vascular surgical activity. Results of a nation-wide study, Denmark, 1977–1990. *Eur J Surg*. 1993;159:469–473.
25. Luther M. The influence of arterial reconstructive surgery on the outcome of critical leg ischaemia. *Eur J Vasc Surg*. 1994;8:682–689.
26. Pell JP, Fowkes FG, Ruckley CV, et al. Declining incidence of amputation for arterial disease in Scotland. *Eur J Vasc Surg*. 1994;8:602–606.
27. Al-Omran M, Tu JV, Johnston KW, et al. Use of interventional procedures for peripheral arterial occlusive disease in Ontario between 1991 and 1998: a population-based study. *J Vasc Surg*. 2003;38:289–295.

3

The Endovascular Suite: *Northwestern University Perspective*

Mila H. Ju, M.D. and Melina R. Kibbe, M.D.

INTRODUCTION

With the advancement of technology, vascular surgery has evolved from purely open surgery to the treatment of complex vascular diseases with minimally invasive approaches. As the number of indications and applications for endovascular surgery increases, it is evident that a dedicated endovascular suite is needed to accommodate all of the demands of endovascular interventions. Although simple endovascular procedures can be performed in existing interventional radiology or cardiac catheterization facilities, the proper sterility and the ability to convert endovascular procedures to open surgical exposures can only be guaranteed by a designated endovascular suite. This chapter will provide the basic blueprint of a modern endovascular suite with an additional perspective from Northwestern University through our facilities at Northwestern Memorial Hospital (NMH) and the Jesse Brown Veterans Affairs Medical Center (JBVAMC).

GENERAL ENDOVASCULAR SUITE SET-UP

In general, the endovascular suite needs to be spacious enough to accommodate a long endovascular operating room table, multiple monitors, fluoroscope, power injector, control room, nursing equipment, anesthesia equipment, and supply cabinets (Figure 3–1). Moreover, one should always anticipate the space needed to convert an endovascular case to an open operation with the necessary equipment and resources, such as lights and case carts. We are fortunate at Northwestern University to have two relatively new, fully functional endovascular suites, both with unique challenges in room design and equipment layout, and with one endovascular suite being built in a smaller operating room, offering

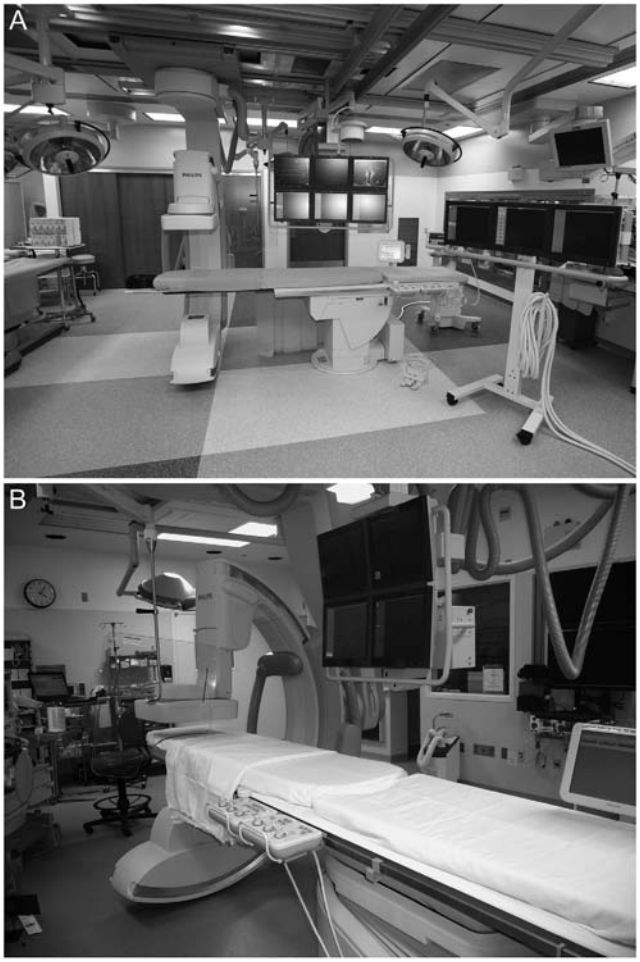

Figure 3-1. Photograph of endosuites at (A) the Jesse Brown Veteran Affairs Medical Center (JBVAMC), and (B) Northwestern Memorial Hospital (NMH). Note that the operating room at the JBVAMC is a larger space, allowing the design of a spacious endosuite with 6- and 3- panel monitors. At NMH, where the room is smaller, there are 4- and 2-panel monitors and a wall-mounted PACS screen (left edge of image).

additional space challenges. Thus, the perspectives we provide in this chapter represent the challenges we faced in designing an ideal spacious endovascular suite, as well as redesigning an endovascular suite given a limited amount of space.

For an ideal endovascular suite, the operating table and the fluoroscope should be in the center of the room, leaving plenty of space surrounding the table for the nursing and anesthesia booms and endovascular case carts. The surgeon and his or her assistant(s) typically stand to the patient's right with the necessary case instruments set up behind the surgeon within easy reach. The monitors should be set up in direct line-of-sight of the surgical staff, ideally on ceiling-mounted booms. The power injector is best when positioned near the foot of the field, off to the left of the patient. If possible, the anesthesia and nursing equipment should be on ceiling-mounted booms that can be easily moved, depending on the needs of the case. The ability to hide all wire connections through the walls and ceiling is best, thereby decreasing tripping hazards and

clutter in the room. For example, having the anesthesia monitoring equipment hard-wired through the walls/ceiling to the surgeons' monitor boom results in efficient communication between the two circuits. However, of note, carts have a greater range of mobility. Regardless of ceiling-mounted booms or carts on wheels, one must ensure that there are enough power outlets to accommodate all of the equipment in the room.

Lastly, the majority of endovascular cases are performed using retrograde common femoral artery access. However, there should be enough space and flexibility in room arrangement to accommodate antegrade common femoral artery access as well as brachial artery access. Thus, sufficient space should be available to accommodate a second long table, should one be required.

RADIOLUCENT FLUOROSCOPY TABLE

Because of the need to visualize the entire vasculature from the skull for carotid interventions to the toes for peripheral procedures, a special radiolucent fluoroscope table is required. A carbon fiber table is preferred for radiation reduction and image quality improvement. Ideally, the operating table should be 117 inches to 144 inches in length to accommodate the length of the guide wires. Although it would be best to flex the table in the middle, none of the commercially available articulation hardware is radiolucent. Therefore, current commercially available tables are only able to tilt cranio-caudally and medial-laterally. Furthermore, the table should be moveable, similar to those used in the interventional radiology and cardiac catheterization units. The position of the table, and therefore the patient, must be under the operator's control.

To efficiently utilize the endovascular suite, it should be designed to convert to conventional open surgeries when no endovascular cases are scheduled. Therefore, one should have the ability to rotate the endovascular table to move it out of the way to be able to bring in a regular operating room table for open cases. This requires careful planning in designing the endovascular suite to ensure that all of the lights, anesthesia booms, and nursing booms are in adequate position to be used for both open surgical and endovascular cases.

CINE(C)-ARM FLUOROSCOPY

Cine-arm (C-arm) fluoroscopes come in either portable, or ceiling- or floor-mounted units. Both systems have advantages and disadvantages, as described in Table 3–1. For simple endovascular procedures, such as inferior vena cava filter placement, a portable C-arm fluoroscope is sufficient. However, fixed C-arm is ideal for complex endovascular procedures.

Digital subtraction angiography (DSA) is the gold standard in the modern endovascular suite for radiographic imaging of the arterial system. In its post-processing, DSA eliminates static elements and only reveals images of contrast-filled vessels, which results in less contrast material. Another essential for endovascular procedures is the ability to road-map, which allows live fluoroscopy to be superimposed on previous radiographic images. This is especially useful in percutaneous transluminal angioplasty and when passing guide wires through tortuous, stenotic, or occluded vessels. Variable frame rates must be taken into consideration when using fluoroscopy. Faster rates can improve image quality but at the same time increase the amount of radiation

TABLE 3-1. ADVANTAGES OF DIFFERENT TYPES OF C-ARM FLUOROSCOPE

Fixed (floor- or ceiling- mounted)	Portable
Superior imaging qualities	Flexible in space and storage
Smaller focal spot	Does not require floor or ceiling support
Variable image intensifier distance	Less expensive
Deeper tissue penetration	
Larger remote generator with increased power capability (important in obese patients)	
Wider anatomic area visualized	
Larger image intensifier	
Less radiation exposure	
Less x-ray scatter due to variable image intensifier distance	
Longer use	
Less prone to overheat	

exposure. The use of collimation and filters to block areas that have a tendency to be bright on screen also limits radiation exposure to the patient and the operators.

Safety precautions must be in place to prevent unnecessary radiation exposure regardless of portable or fixed C-arm unit used. All personnel in the endovascular suite should wear lead aprons with thyroid shields. Ideally, lead aprons should provide protection for the front and the back, as the front-only aprons leave room for radiation exposure to vital organs if the wearer is moving about the room. In addition, ceiling-mounted transparent radiation shields can be used to provide optimal scatter protection of the operator's upper body and face. Table-mounted lead apron flaps provide further protection to the lower body. All personnel should wear radiation badges to record the amount of radiation exposure.

POWER INJECTOR

The power injector can be used for aortograms as well as for selective angiograms of the upper and lower extremities; therefore, it deserves special mention in this chapter. It is best when positioned near the foot of the bed, off the left side of the table, leaving plenty of room to float the endovascular table without contaminating the sterile field. We have found that the Mark V Provis unit by Medrad functions well (Figure 3–2). Furthermore, with newer technology, the power injectors can be coupled with the fluoroscopy equipment so that the contrast injection is optimally timed with the radiation exposure.

MONITORS

A modern endovascular suite would not be complete without display equipment. The monitors should be placed in a straight line-of-sight from the surgeon. Simultaneously, assistants should also be able to view the images without being in awkward positions. For ergonomic reasons, monitors should be placed just below eye level at a 10 degree downward angle, where neck muscles are most relaxed.

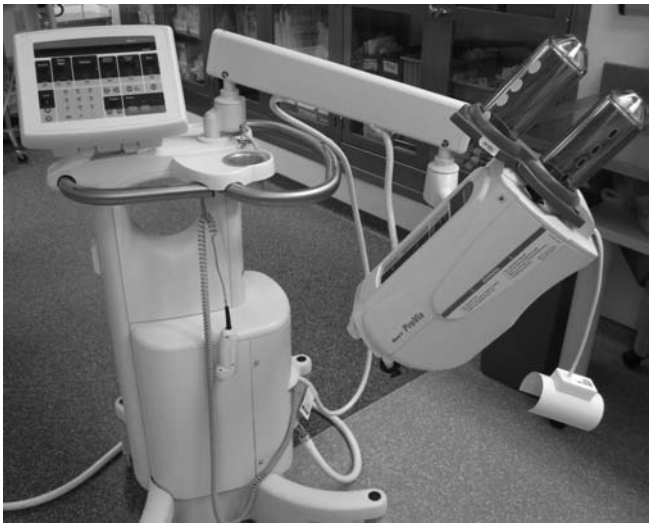

Figure 3-2. Photograph of the Mark V Provis power injector by Medrad.

At our JBVAMC facility, we have a six-panel monitor boom as the primary monitor display. The six monitors are assigned as follows: (1) anesthesia vitals, (2) CT or MRA images from picture archiving and communication system (PACS), and (3) CT images obtained from our Philips C-arm system, leaving the other three monitors for image acquisition during the case such as (4) live images, (5) roadmap images, and (6) ancillary images such as stored runs or static images. Alternatively, one of these monitors could be dedicated for use with IVUS. We also have a three-panel mobile monitor cart that can be positioned for operators standing on the opposite side of the patient. These monitors are dedicated for image acquisition during the case with live images, roadmapping, and ancillary images.

We have a similar set-up at our NMH facility with two monitor display units. However, due to space constraints, instead of a 6-panel ceiling mounted and 3-panel mobile cart display, we have a 4-panel ceiling-mounted main display, and a 2-panel ancillary cart for the assistant surgeons. Although still able to view live images, the assistants are not able to view other images such as roadmaps and PACS images in direct line of sight.

LIGHTING

Lighting is critical for both open and endovascular cases. Generalized room light can be incandescent or fluorescent. However, lighting must be able to be dimmed during fluoroscopy or switched to orange, yellow, or green lighting to prevent glare on monitors. Surgical lights should be anchored in a way to prevent collision paths with endovascular equipment and the other ceiling-mounted booms. One must keep in mind the locations of endovascular and conventional operating tables when designing the position of these lights so that there is adequate light for both types of procedures. We found it best to have surgical lights mounted on long articulating arms with three joints to increase range of motion and possible position placement. It is also challenging to determine the best position of

the lights so that no conflicts exist with the nursing or anesthesia booms or C-arm in order to reach their desired location. Although surgical lights are generally turned off during the majority of endovascular procedures, there should be a spotlight for nurses and surgeons to prepare the equipment. Optimally, the surgical lights are equipped with closed-circuit camera and video capabilities.

ANCILLARY STAFF AND EQUIPMENT

A knowledgeable nursing staff is critical for a smooth running conventional operating room. The same principle should be applied to the endovascular suite. Nurses must be thoroughly trained in both conventional and endovascular techniques. This is essential, as many of the endovascular procedures may need to be converted to open cases, and vice versa. The scrub nurse must know how to prepare the wires, sheaths, catheters, balloons, stents, grafts, etc. The circulator must know how to position the C-arm, operate the power injector, and be familiar with the location of all endovascular and open vascular supplies. Moreover, the nursing staff should be responsible for cataloging and reordering endovascular supplies. Ideally, these nurses are assigned exclusively to the vascular team (i.e., dedicated nursing support) and involved in every step of the process from the moment the patient enters the operating suite to when that individual leaves for the recovery room.

As mentioned in the section on set-up, there should be a ceiling-mounted nursing boom that houses the bovie, suction, and other equipment that need power outlets or connectors. By having this equipment on a ceiling-mounted boom, tripping hazards on the floor created by wires and connectors will be minimized. For the majority of cases, this nursing boom can be positioned toward the foot of the table, off to the left side. This position allows the equipment to be isolated from the surgeon and the wires, catheters, and other materials that are frequently passed between the scrub nurse and the surgeon. Lastly, the nursing staff should have a computer in the operating suite dedicated to the nursing staff for documentation throughout the case as well as for ordering supplies, etc.

ANESTHESIA STAFF AND EQUIPMENT

Anesthesia staff is essential for the majority of open and endovascular cases. As with the operating room nurses, the anesthesia staff must be knowledgeable in both open and endovascular cases. They should have basic knowledge of when during a procedure to expect a change in the hemodynamics of the patient, and which cases can tolerate relative hypotension or hypertension.

When designing the endovascular suite, one must take into account the amount of space that cardiopulmonary monitors and anesthesia equipment occupy. The conventional set- up is to have the anesthesia staff and equipment at the head of the operating table. Again, all of the equipment must be flexible for rearrangement as needed. As with nursing equipment, we recommend having a ceiling-mounted boom for all of the anesthesia equipment to ease congestion in the room and allow for greater mobility , accommodating multiple table positions, both open and endovascular.

CONTROL ROOM

Every modern endovascular suite should have a control room with necessary computer equipment that is linked to the C-arm data acquisition to review and edit images from completed cases and to plan for future cases. A secure system is needed to store the images, as access to future images is crucial for treating vascular disease. Moreover, computer equipment should be linked to the hospital's electronic medical records to review patients' charts. Lastly, there should be a PACS system or hospital-based imaging system to review patient CT and MR images.

The control room should be lead-lined and connected to the operating room so that one can enter the control room from the operating room and vice versa without the risk of contamination. We do not recommend placing a door between the control room and the operating room, as this will increase the risk of contamination. One should be able to view the operating room from the control room through lead-lined glass panels and be able to communicate with the staff in the operating room. A two-way microphone is ideal for this purpose. If space permits, it is helpful to have a separate outside door to the control room, so that individuals can visit the control room without having to pass through the operating room.

BASIC SUPPLIES

As mentioned previously, a modern endovascular suite should be equipped with basic supplies which should be housed in a dedicated area within the endovascular suite. A sample basic endovascular inventory is listed in Table 3–2. Ideally, the supply cabinets are stored in the operating suite and/or control room for easy access preoperatively when

TABLE 3-2. BASIC SUPPLIES FOR ENDOVASCULAR INTERVENTIONS

Basic Endovascular Supplies	
Puncture needle	Guide catheters
Wires	Balloons
Entry	Various diameters and lengths
Glide	Compliant and noncompliant
Angled	Inflation gauge
Steerable	Stents
Multiple sizes	Various diameters and lengths
0.035"	Balloon expandable bare metal stents
0.018"	Self-expanding bare metal stents
0.014"	Covered stents
Sheaths	Coils
Various sizes	Nonionic contrast
Various lengths	Iso-osmolar
Multi-sidehole flush catheters	Hypo-osmolar
Straight or pigtail	Power injector
General catheter	Puncture site closure devices
Selective catheter	

Figure 3-3. Photograph of the mobile endocabinets in the JBVAMC endosuite. Note that the endocabinets were designed to accommodate supplies of varying shapes and sizes and are well-labeled for easy location of product.

planning the case and intraoperatively when using the supplies. The supply cabinets are available in many forms and can be custom made to fit the needs of the endovascular suite. It is important to have cabinets for short- and long-hanging supplies, baskets or drawers for smaller supplies, and shelves for bulk stock, such as stents (Figure 3–3). These supply cabinets can also be fixed to walls or on wheels, depending on the needs of the operator and the room. If the room is to be used for non-endovascular or vascular interventions, having the carts on wheels is useful in the event they need to be moved outside of the room temporarily for other cases.

INTRAVASCULAR ULTRASOUND (IVUS)

Some discussions have suggested including IVUS into the modern endovascular suite. IVUS can be used as an adjunct to angiography or for patients without tortuous vessels who have an allergy to iodinated contrast. As a preoperative tool, it can be used for endograft sizing. As a postoperative study, it can be used for evaluation of graft stenosis, endoleaks, and for evaluation of approximation of the graft within the vessel wall. Currently, there are two ways of viewing images from IVUS: either from the IVUS monitor, or incorporated into the endovascular monitor panel as one of the monitors. Given that incorporation of IVUS to the monitor display requires a dedicated monitor, we prefer to keep the IVUS imaging separate and simply view images on the IVUS display. However, if IVUS is a large part of the practice, having a dedicated monitor on the ceiling-mounted display should be considered.

CONCLUSIONS

It is evident that technology can change the way one approaches vascular surgery. A dedicated endovascular suite is essential for vascular surgeons to keep pace with the demand for endovascular procedures. When designing the modern endovascular suite, one must keep in mind that the modern endovascular suite must have the capability to accommodate endovascular interventions and open surgeries to ensure efficient utilization of the room. Furthermore, the modern suite must also have the flexibility to upgrade to a new system and to include new equipment as needed.

REFERENCES

1. Queral LA. Operating room design of the future. In: Yao JST, Pearce WH, eds. *Techniques in Vascular and Endovascular Surgery*. Stamford CT: Appleton & Lange; 1998: 1–6.
2. Fillinger MF. New imaging techniques in endovascular surgery. In: Pearce WH, Matsumura JS, Yao JST, eds. *Endovascular and Minimally Invasive Vascular Surgery* 79: 451–475, 1999.
3. Mansour MA. The new operating room environment. In: Pearce WH, Matsumura JS, Yao JST, eds. *Endovascular and Minimally Invasive Vascular Surgery*. 79: 477–487, 1999.
4. Wain RA, Veith FJ. Use of digital ciné-fluoroscopy and catheter-directed techniques to improve and simplify standard vascular procedure. In: Pearce WH, Matsumura JS, Yao JST, eds. *Endovascular and Minimally Invasive Vascular Surgery*. 79: 489–505, 1999.
5. Eagleton MJ, Schaffer JL. The vascular surgery operating room. *Endovascular Today.* 25–30, August 2007.
6. Benjamin ME. Building a modern endovascular suite. *Endovascular Today.* 71–78, March 2008.
7. Ohki T. Pros and cons of IVUS imaging for endovascular procedures. *Endovascular Today.* 80–82, March 2008.
8. Sikkink, CJJM, Reijnen MMPJ, Zeebregts CJ. The creation of the optimal dedicated endovascular suite. *Eur J Vasc Endovasc Surg*. 35, 198–204, 2008.

4

The Cost of Endovascular Peripheral Interventions and Endovascular Aneurysm Repair

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COST AND ENDOVASCULAR PROCEDURES

Hospitals and providers are increasingly feeling the strain of a financially overburdened health care system. Yet, as medical technology develops, there is substantial pressure from the public and industry to offer the most minimally invasive and "cutting-edge" techniques. From a physician's perspective, these techniques should be offered if they are proven to provide clinical benefit and the increased cost is commensurate with improved care and recovery. Endovascular aneurysm repair (EVAR) and endovascular treatment of peripheral arterial disease (PAD) are prime examples of technological advances that have improved patient care. However, technology is expensive. Third-party payers have failed to correspondingly increase reimbursement to adequately meet these technologically driven expenses. Ultimately, a physician's ability to even offer EVAR and other endovascular techniques may be restricted by fiscally motivated hospital policies.

The term "cost" refers to the actual expense that is incurred by the hospital itself. This somewhat nebulous concept for physicians is far more practical in the day-to-day activities of department managers, administrators, and the hospital accounting department. Cost can be broken down into direct cost and indirect cost. Direct cost includes supplies, equipment, salaries, and facility use. Indirect cost includes cost from departments not directly involved with the patient's care (e.g., cafeteria, finance, computer services) as well as institutional overhead. Charges are then generated by a cost-to-charge ratio and submitted to the third-party payers. Reimbursement is defined as the actual money received and is generally substantially less than the actual charge. The balance between the actual cost and the reimbursement determines whether a profit margin is generated.

The largest cost drivers for EVAR and endovascular interventions are usually the devices (endografts, stents). In addition, vigorous surveillance regimens after these procedures add to the overall expense. The durability of many endovascular techniques is more limited than traditional open techniques and may require additional maintenance procedures, further draining the health care system. The purpose of this chapter is to review the literature on the cost of endovascular interventions, focusing on lower extremity revascularization procedures and EVAR.

THE COST OF LOWER EXTREMITY ENDOVASCULAR INTERVENTIONS

The emergence and exponential growth of endovascular interventions for the lower extremities has significantly altered the treatment algorithms for PAD. Current guidelines recommend endovascular procedures as the first-line therapy for shorter-segment lesions in the iliac and superficial femoral arteries (SFAs).[1] Preprocedure imaging, the initial procedure, postprocedure surveillance, and reinterventions are all factors that contribute to the overall cost of endovascular and open interventions. Concern about the durability of endovascular procedures and lack of standard surveillance protocols may lead to aggressive and expensive follow-up.

Stoner et al. retrospectively examined the cost of revascularization for femoral-popliteal segments, comparing endovascular (n=198) and open (n=183) procedures.[2] Using the model proposed by these authors, cost-efficacy curves for endovascular interventions can be calculated on a cost-per-day basis (Figure 4–1). Importantly, this methodology incorporates overall patency and reintervention rates into the final analysis. Thus, a procedure that achieves long-term patency without reintervention has the lowest cost-per-day of patency, whereas a procedure that fails after a short duration would have the highest.

In this study, 12-month primary-assisted patency was 77% in the open femoral-popliteal bypass group vs. 65% for the endovascular group ($P<.01$). Although the initial cost of endovascular intervention was just over half as much as open bypass ($6739 vs. $12,389; $P<.001$), this benefit was offset by the cost of more reinterventions in the endovascular group by 1 year. At 12 months, the cost-per-day analysis found a compa-

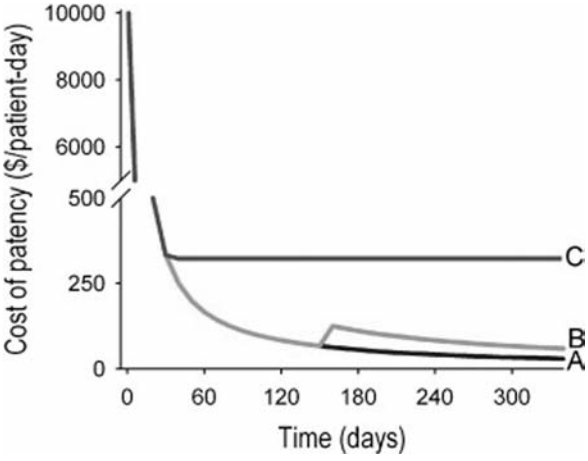

Figure 4-1. Hypothetical cost curves for the following revascularization scenarios. A. Successful initial revascularization without failure of patency or reintervention. B. Reintervention to maintain patency. C. Failure of patency without reintervention. (Used with permission from Stoner MC, deFreitas DJ, Manwaring MM, et al. Prophylatic use of the silver-acetate-coated graft in arterial occlusive disease. J Vasc Surg 2008;48:1489–96.)

rable cost of $229 for open bypass and $185 for endovascular intervention (P=.71). Interestingly, there was a trend toward greater cost-per-day for endovascular interventions in the critical limb ischemia group ($359 vs. $210; P=0.33), although statistically insignificant. Conversely, the subgroup of claudicants had a lower cost-per-day of $86 versus $259 with endovascular intervention (P=.31), with particularly lower cost if no stent was used. It is also noteworthy that only 21% of patients in the endovascular group of this study received stents. This pithiness in stent usage probably does not reflect most practice patterns, however. As such, this study may underestimate the cost of peripheral vascular intervention.

Although costs will vary somewhat by region and institution, certain aspects of this study can be applied broadly. Endovascular interventions on the SFA have patency rates inferior to femoral-popliteal bypass and therefore require more numerous (and costly) reinterventions. Stents are used with varying frequency in the SFA; greater stenting percentage translates to higher cost. Follow-up in the above-mentioned study was truncated at 1 year, but longer follow-up could reveal even greater divergence in the number of reinterventions and cost.

The type of endovascular intervention being performed also factors into the procedural cost. For example, atherectomy or stenting has higher direct cost than angioplasty alone in the lower extremities. When more than one stent is used, as is sometimes necessary, the cost additive from these devices is significant. From a purely costing perspective, selective stenting after angioplasty of the SFA is recommended until the clinical data conclusively demonstrate better patency for primary stenting. Lasers, cryoplasty, atherectomy, re-entry devices, and drug-eluting stents are examples of innovative technologies that are in various stages of development for treating lower extremity occlusive disease. Unless clinical superiority and durability can be demonstrated with a new technology, fiscally responsible endovascular interventionalists should proceed cautiously with adopting more costly technology.

The setting in which the procedure is performed (operating room or radiology suite, outpatient or inpatient) further influences both the cost and the reimbursement. In a study from Buffalo, New York, the authors grimly point out that net losses are seen for outpatient endovascular procedures, even more so for private pay insurers than for Medicare.[3] Costs are less in the catheterization laboratory than in the operating room and less for outpatient than inpatient procedures. The cost of an arterial stenting procedure in the catheterization laboratory was $3392 compared with $5844 in the operating room, largely due to greater expense of operating room personnel and medical supplies. However, reimbursement was worse for outpatient procedures. As a result, only inpatient procedures performed in the radiology suite earned profit. These findings argue for the use of the catheterization laboratory as the primary site for pure endovascular interventions. However, competition for catheterization laboratory time and space, as well as the inability to perform hybrid procedures in this setting, makes this a less attractive option. Given the increased patient satisfaction, safety, and efficiency of ambulatory endovascular procedures, a lower reimbursement rate is counterintuitive.

Coding guidelines also limit the reimbursement for ambulatory endovascular procedures and are based on the Current Procedural Terminology (CPT) for outpatient procedures and Diagnosis Related Group (DRG) for inpatients. Currently, outpatient procedures may have several components within a single procedure that can be coded, with each reimbursed at a lower tier. This system requires that each of the components is accurately listed. For example, endovascular treatment of the SFA may include components for the stenting, angioplasty, catheter placement, and the radiologic

interpretation codes. However, a stent placed in the SFA can be coded as an additional component of the procedure only if it is documented to have been placed for a residual >20% stenosis or dissection or a ≥5 mm Hg gradient postangioplasty.[4] Multiple stents placed within the same vessel are coded as one procedure, and therefore, losses are incurred from the cost of multiple stents. Separate "carve-out" reimbursement for the cost of implantable devices is sometimes negotiated with private insurers and would help to defray some of these losses. Reimbursement within the DRG system is more global and based upon the patient's comorbid conditions and complications. A greater number of comorbid conditions and complications will reimburse at a higher DRG level. Therefore, meticulous documentation by health care providers of pre-existing conditions becomes a critical part of reimbursement.

THE COST OF ENDOVASCULAR ANEURYSM REPAIR

EVAR results in lower rates of perioperative morbidity and mortality compared with open repair, as shown in two randomized trials.[5-6] Nonetheless, the cost-effectiveness of this procedure is controversial. The cost of EVAR can be broken down into two categories. First, there is the initial cost of endograft placement that includes the device, operating room costs, and costs from the hospital stay. Intuitively, a lower complication rate and shorter hospital stay for EVAR would reduce hospital costs. However, these benefits have failed to translate into a cost-effective procedure when all cost components are considered. The largest cost component is the endograft device itself. Post-placement costs are incurred after the initial hospital stay and include surveillance imaging, follow-up visits, laboratory work, and secondary procedures. Indeed, these late costs increase the global cost of EVAR by 44%.[7]

PLACEMENT COSTS

In a retrospective cost analysis of EVAR performed on the U.S. Food and Drug Administration (FDA) Phase II multicenter AneuRx (Medtronic, Santa Rosa, CA) trial published in 2000, the initial placement costs for EVAR were significantly greater than those for open repair.[8] The largest cost component was the device, which accounted for 52% of the total placement cost of EVAR. Several other retrospective studies have confirmed the greater initial expense of EVAR compared with open repair.[9-11] In each of these studies, EVAR resulted in significantly shorter length of hospital stay, ICU admissions, and operating room time. In addition, a lower cost of floor nursing, pharmacy, and laboratory use in the EVAR group did not translate to overall lower costs. Because the cost of stent grafts exceeds that of open surgical grafts by 10 to 20 times, EVAR is the more expensive treatment.

Although prospective data have shown a lower mortality for EVAR, in these retrospective costing studies, mortality was not statistically different between the open and the EVAR groups. This point is important because in modeling analyses, the cost-effectiveness of EVAR hinges upon its potential to reduce morbidity and mortality compared to open repair.[12] Costs may also vary depending on the patient population being treated. Thus, patients with a greater number of comorbidities may require additional tests and hospital costs, although the reimbursement for these patients will be at a higher tier. In a Canadian prospective, nonrandomized observational study of

TABLE 4-1. ENDOGRAFT PLACEMENT

	Year	EVAR Cost	Open Cost	Device Cost
Patel[12]	1999	$20,083	$16,016	$8000
Sternbergh[8]	2000	$19,985	$12,546	$10,400
Clair[9]	2000	$7205	Greater for EVAR	$8976
Bosch[10]	2001	$20,716	$18,484	$7000
Dryjski[11]	2003	$17,539	$9042	$9475-9975
MEAN		$19,581	$14,022	$8820

In each of these studies, the initial cost of EVAR exceeded that for open repair. The largest cost driver is the cost of the device.

EVAR cost specifically looking at high-risk patients, initial hospital costs were comparable for EVAR versus open repair.[13] This study is of particular interest because two thirds of the patients in both groups were from the American Society of Anesthesiologists Class IV (severe systemic disease that is a constant threat to life). However, a median length of hospital stay of 6 days in the EVAR group, longer than in many studies, may have skewed these data.

The dominant cost component in all of these reports is the device, which accounts for 30 to 50% of initial placement cost. Current devices on the market retail for more than $13,000 per case. From these U.S. studies between 1999 and 2003, a mean initial hospital cost of $19,581 for EVAR is estimated (Table 4–I). Using the medical component of the Consumer Price Index, the estimated 2009 placement cost of EVAR would be $24,212.[14]

POST-PLACEMENT COSTS

Costs incurred after the initial endograft placement have been dubbed "post-placement costs." In a study from the Ochsner Clinic, the cumulative post-placement cost per patient at 5 years after EVAR was $11,351, thereby increasing the global cost of EVAR by 44%.[7] Post-placement costs include the cost of secondary procedures, surveillance imaging, outpatient visits, and laboratory work. Despite institutional and regional differences, there are certain trends in the post-placement costs. Consistently, secondary procedures and surveillance imaging are the number one and two contributors to post-placement costs, respectively (Figure 4–2).

Secondary procedures are defined as additional interventions after the original EVAR, such as treatment for endoleak, migration, or limb thrombosis. Secondary procedures are a time-dependent phenomenon and may be needed after EVAR in 10 to 20% of patients.[15-17] Secondary procedures are particularly costly, constituting 57.4% of the total post-placement costs in the Ochsner study. In fact, patients who undergo secondary procedures had 8.6 times higher costs compared with those patients who never underwent a secondary procedure. Although secondary procedures such as diagnostic angiograms and endocuffs are more common, the rare delayed open aneurysm conversion accounts for a significant proportion of secondary procedure cost.

Surveillance after EVAR has traditionally been vigilant owing to concern for migration and endoleak, potential sources of aneurysm-related morbidity. However, imaging performed with thin-slice helical CT scans is costly. A typical post-placement regimen includes a CT and abdominal radiographs at 1, 6, and 12 months after initial

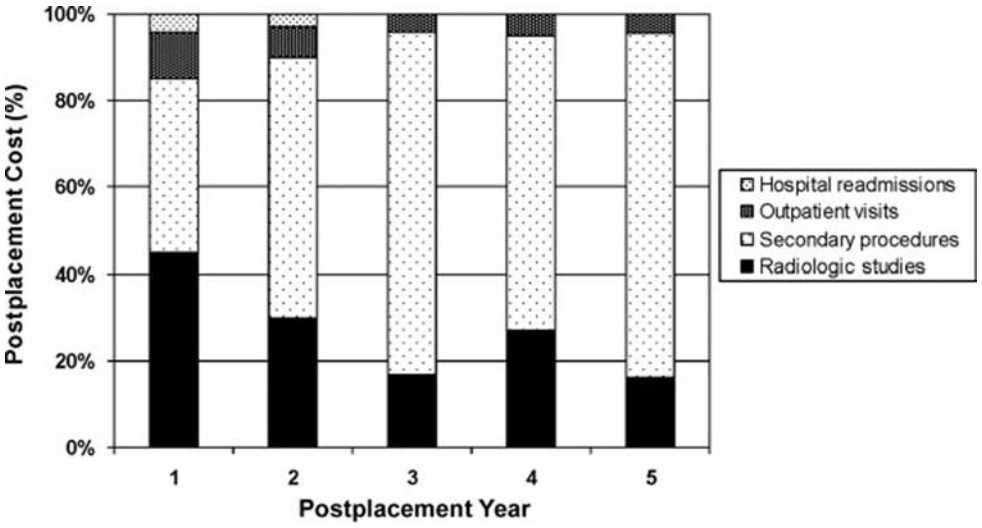

Figure 4-2. Yearly post-placement cost components. Secondary procedures and surveillance radiographic studies are the number one and two sources of post-placement costs, respectively. (Adapted with permission from Noll RE, Tonnessen BH, Mannava K, et al. Long-term postplacement cost offer endovascular aneurysm repair. J Vasc Surg 2007;46:9–15.)

implantation and then yearly thereafter. The frequency of imaging is even more intensive in patients identified with problems such as endoleaks. In contrast, surveillance after open aneurysm repair is recommended only every 3 to 5 years to detect late and infrequent complications such as anastomotic pseudoaneurysms.[18] Most practices rarely perform long-term radiographic follow-up after open repair.

COST-EFFECTIVENESS OF EVAR

Although the true cost of EVAR is greater than that of open repair, determining cost-effectiveness is less concrete. Markov modeling is one strategy by which a hypothetical cohort is subjected to either EVAR or open repair (Figure 4–3). As the model progresses, the probability of various outcomes is determined for each branch of the model. Using incidences derived from the medical literature, the model makes basic presumptions, such as risk of death, reintervention, and medical complications. The costs are then calculated for each branch within this theoretical framework. A major weakness of Markov modeling is that the outcome varies depending upon the initial hypothetical cohort inputted, that is, a healthy 70-year-old male with a 5.0-cm abdominal aortic aneurysm (AAA) is not representative of all patients. Also, the model hinges upon predetermining a number of variables, such as estimated mortality risk.

Once the Markov model is completed, quality-adjusted life-years (QALYs) can be calculated. This methodology evaluates the societal impact of a medical or surgical intervention, potentially providing a tool for determining the cost-effectiveness of a procedure. A QALY score of 1.0 indicates a year of perfect health, whereas death is scored as a 0.0. A surgical procedure that affects the patient's ability to function at a 1.0 level is scored between 0.0 and 1.0. The cost-effectiveness ratio of a particular surgical procedure or intervention can be determined by calculating a ratio of cost to QALY. Health care resources may potentially be allocated based upon lower cost-to-QALY ratios.

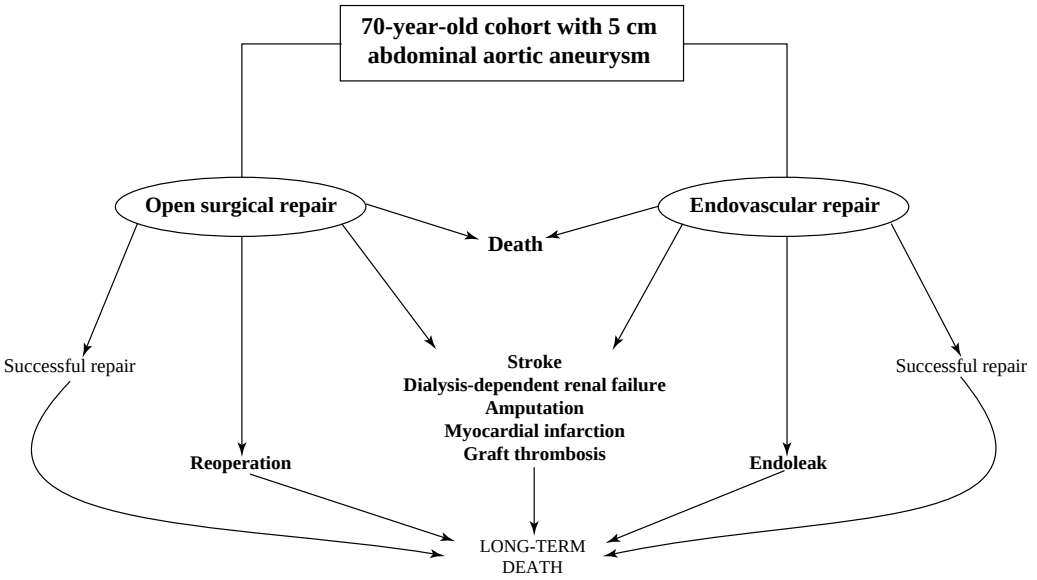

Figure 4-3. Simplified Markov decision analysis model. A hypothetical cohort with a 5-cm abdominal aortic aneurysm (AAA) may undergo either open surgical repair or endovascular repair. The outcomes of each strategy may be either successful repair or any of a number of complications. Each member of the cohort is followed until death. (With permission from Patel ST, Haser PB, Bush HL, et al. The cost-effectiveness of endovascular repair versus open surgical repair of abdominal aortic aneurysms: a decision analysis model. J Vasc Surg 1999;29: 958–72.)

In one such Markov modeling analysis, EVAR was found to be cost-effective even though the lifetime cost of EVAR exceeded that of open repair.[12] EVAR had a cost-effective ratio of $22,826, which is below the threshold of $60,000 that society is typically willing to pay for medical interventions. The authors used another example of the cost-effectiveness ratio for chronic hemodialysis being $54,400. However, cost-effectiveness in societal terms does not take into account day-to-day realities of hospital costs and reimbursement.

In the only randomized trial to examine "real-life" cost-effectiveness of EVAR, 340 patients were randomly assigned to either EVAR or open AAA repair.[19] When clinical outcomes were scrutinized in terms of cost, the initial placement costs for EVAR exceeded those for open repair significantly. Shorter length of stay and fewer complications could not substantially offset the endovascular device cost. At 1 year follow-up, patients who underwent EVAR or open repair had nearly equivalent QALY (0.72 vs. 0.73). When the higher cost of EVAR was balanced against the similar QALY scores for EVAR and open repair, open repair was determined to be the more cost-effective technique. This Dutch study likely underestimates the actual cost-effectiveness differential owing to limited 1-year follow-up and may also be difficult to apply to U.S. costs.

DEVICE DEPENDENCE

Given that the device cost dominates the initial cost of EVAR, it is relevant to consider the potential cost differences among device types. A retrospective review from the University of Florida looked at precisely this point.[20] Three different endografts (Medtronic AneuRx,

W.L. Gore Excluder, and Cook Zenith) were used in their EVAR cases between 2000 and 2006. Both the AneuRx and the Excluder devices are designed with a two-piece modular system, whereas the Zenith device has three components. Therefore, it is not surprising that the basic cost for a Zenith device was $12,800 compared with $9475 and $10,203 for the AneuRx and Excluder devices, respectively. However, when the costs were calculated in a "real-life" setting, the authors found that they used a greater number of proximal and distal extensions with the two-piece devices than the three-piece device. Therefore, the total cost was nearly equivalent (within $500) for each company's stent graft system, approximating $13,000.

Post-placement costs after 5 years of surveillance for the AneuRx and the Zenith devices have also been compared.[21] The bulk of long-term cost was from secondary procedures, which were more frequent after 5 years in the AneuRx group (49% vs. 20%; $P<.05$). Despite this fact, post-placement cost for the AneuRx device was only 15% greater in follow-up, not statistically significant. Improved device design and technique could lead to a decreased number of costly secondary procedures.

REIMBURSEMENT

AAA repairs are reimbursed through the Medicare system using a DRG-based code to standardize payment. Under this system, DRG 237 refers to a major cardiovascular procedure with comorbidities/complications, whereas DRG 238 refers to a major cardiovascular procedure without comorbidities/complications (Table 4–2). DRG 237 reimburses ~$32,440 versus ~$18,460 for DRG 238 (Arizona urban teaching hospital rate) – but these numbers vary somewhat by region and population density. Currently, only 38% of AAA repairs code to the higher-reimbursing DRG 237 (data from Thomson-Reuters, 2008). Using our 2009 estimated cost of EVAR of $24,212 and a mean weighted reimbursement of $23,772 per case, many hospitals may barely break even or sustain losses after EVAR (Table 4–3). Therefore, more stringent criteria that could place even more EVAR in the less complex DRG 238 group would be financially devastating for hospitals.

In a multicenter study published in 2003, a cross-section of three university and four community hospitals was examined with respect to actual EVAR reimbursement.[22] Ultimately, a net loss of $3898 per patient was seen for initial placement and hospital stay. Losses was greater for the lower-weighted DRG at $9198 per patient. Community hospitals fared the worst owing to lower reimbursement rates.

TABLE 4-2. EXAMPLES OF MAJOR COMORBIDITIES AND COMPLICATIONS THAT CODE TO DRG 237 (MAJOR CARDIOVASCULAR PROCEDURE)

Cardiac arrest
Acute congestive heart failure
Myocardial infarction
Pulmonary embolus
Acute respiratory failure
End-stage renal disease
Acute renal failure
Chronic renal failure
Pneumonia (including aspiration)

TABLE 4-3. ESTIMATED HOSPITAL COST AND REIMBURSEMENT FOR EVAR

Hospital Cost of EVAR		**$24, 212**
Cost of Endograft		~$13,000
% Cost of endograft		54%
Reimbursement for EVAR		
DRG 238	(62% of cases)	~$18,460
DRG 237	(38% of cases)	~$32,440
Weighted mean:		$23,772
Hospital net for EVAR		**–$440**

In a 2008 single-center study, Medicare and private insurance reimbursement were examined over 5 years of follow-up after EVAR.[23] Overall, an actual net institutional loss of $2235 per patient occurred. Losses were greater with Medicare than with capitated and commercial insurance. Overall, Medicare reimbursed only 68.5% of the total cost of surveillance (Figure 4–4). Patients who underwent secondary procedures (19.1% by 38.8 months) generated the greatest inequity in reimbursement rates, whereas patients with uncomplicated follow-up actually netted a slight profit for all third-party payers. The bottom line is that reimbursement for the post-placement costs of EVAR is currently inadequate to meet the true costs.

COST REDUCTION STRATEGIES

Standard surveillance regimens that include frequent and lifelong serial CT imaging emerged from early multicenter trials as a means to identify endoleak, migration, and sac expansion. One such regimen would include a CT and abdominal radiographs at 1, 6, and 12 months and yearly thereafter. Although these regimens were designed to prevent post-placement aneurysm-related morbidity, there is risk from cumulative exposure to radiation and iodinated contrast exposure. Furthermore, such vigorous regimens add significantly to the post-placement cost. More recent data from the multicenter Zenith trial suggest that a more "relaxed" surveillance regimen is feasible and safe in selected patients.[24] Absence of

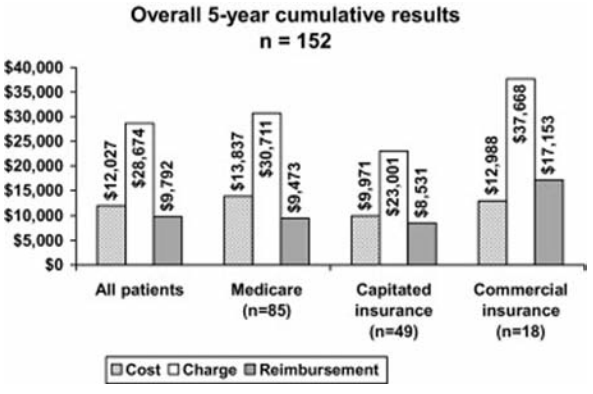

Figure 4-4. Five-year cumulative cost, charges, and reimbursement by payer for all patients after EVAR. Both Medicare and capitated insurance, which covered 88% of patients, were inadequate to cover the costs of follow-up after EVAR. (Adapted with permission from Kim JK, Tonnessen BH, Noll RE Jr, et al. Reimbursement of long-term post-placement cost after endovascular abdominal aortic aneurysm repair. J Vasc Surg 2008;48:1390–5.)

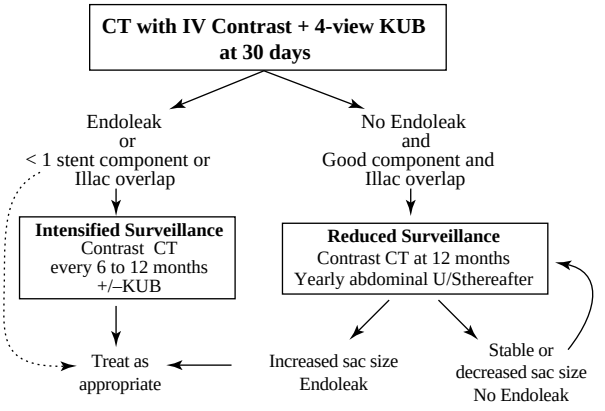

Figure 4-5. New surveillance protocol for Zenith endografts. CT, computed tomography; IV, intravenous; KUB, plain abdominal radiographs; U/S, ultrasound. (With permission from Sternbergh WC III, Greenbergh RK, Chuter TAM, et al. Zenith investigators. J Vasc Surg 2008;48:278–85.)

early endoleak at 30 days predicts a patient population at lower risk for aneurysm-related morbidity. Moreover, sac shrinkage ≥5 mm in combination with no endoleak at 1 year has only a 5.3% risk of subsequent aneurysm-related morbidity.

Duplex ultrasound has been used with increased frequency for surveillance after EVAR. From a cost standpoint, it is significantly less expensive. The major downside to this technique is that it is highly operator-dependent. Duplex ultrasound can accurately determine endoleak type and aneurysm sac size.[25] Based on the above data, Sternbergh et al.[23] suggested a surveillance regimen that eliminates the 6-month CT for patients with no endoleak at 30 days, and abdominal duplex ultrasound yearly for patients with no endoleak and stable sac size on the 1 year CT (Figure 4–5). However, the authors caution against extrapolating these data to patients whose aneurysms were treated outside the Instructions for Use (IFU) or with other devices until more data are available on those topics.

The frequency of secondary procedures after EVAR is the largest driver of postplacement cost. One strategy would be to minimize the need for and the number of secondary procedures. Earlier in the EVAR experience, most endoleaks were treated with trepidation and often intervention. Current evidence supports an expectant approach to type II endoleaks (that affect approximately 20% of EVAR), treating only if the endoleak is persistent and associated with sac growth of 5 mm or more.[26] Using these guidelines, only 1% of the original cohort of EVAR may require intervention for type II endoleaks. This management is also cost-effective. However, the presence of a type II leak at 30 days or beyond does greatly increase the need for a secondary procedure.[24]

CONCLUSIONS

The rising cost of health care has placed a tremendous burden on the U.S. economy. Endovascular techniques for peripheral vascular intervention and aneurysm repair have broadened and improved our ability to treat patients. However, these technological advances are more costly than traditional and often outdated techniques. Current reimbursement is insufficient to meet the advances in vascular care. Significant adjustments in reimbursement and cost-effective physician practices will help to balance these costs.

REFERENCES

1. Norgren L, Hiatt WR, Dormandy JA, et al. Inter-society consensus for the management of peripheral arterial disease (TASC II). J Vasc Surg 2007;45(Suppl S):S5–67.
2. Stoner MC, deFreitas DJ, Manwaring MM, et al. Cost per day of patency: understanding the impact of patency and reintervention in a sustainable model of healthcare. J Vasc Surg 2008;48:1489–96.
3. O'Brien-Irr MS, Harris LM, Dosluoglu HH, et al. Lower extremity endovascular interventions: can we improve cost efficacy? J Vasc Surg 2008;47:982–7.
4. Miller J. CMS withdraws CCI policy change. Endovasc Today 2008;7:71–3.
5. Prinssen M, Verhoven EL, Buth J, et al. A randomized trial comparing conventional and endovascular repair of abdominal aortic aneurysms. N Engl J Med 2004;351:1607–18.
6. Greenhalgh RM, Brown LC, Kwong GP, et al. Comparison of endovascular aneurysm repair with open repair in patients with abdominal aortic aneurysm (EVAR trial 1), 30 day operative mortality results: randomized controlled trial. Lancet 2004;364:843–8.
7. Noll RE, Tonnessen BH, Mannava K, et al. Long-term postplacement cost after endovascular aneurysm repair. J Vasc Surg 2007;46:9–15.
8. Sternbergh WC, Money SR. Hospital cost of endovascular versus open repair of abdominal aortic aneurysms: a multicenter study. J Vasc Surg 2000;31:237–44.
9. Clair DG, Gray B, O'Hara PJ, et al. An evaluation of the costs to health care institutions of endovascular aortic aneurysm repair. J Vasc Surg 2000;32:148–52.
10. Bosch JL, Lester JS, McMahon PM, et al. Hospital costs for elective endovascular and surgical repairs of infrarenal abdominal aortic aneurysms. Radiology 2001;220:494–7.
11. Dryjski M, O'Brien-Irr MS, Hassett J. Hospital costs for endovascular and open repair of abdominal aortic aneurysm. J Am Coll Surg 2003;197:64–70.
12. Patel ST, Haser PB, Bush HL, et al. The cost-effectiveness of endovascular versus open surgical repair of abdominal aortic aneurysms: a decision analysis model. J Vasc Surg 1999; 29:958–72.
13. Tarride JE, Blackhouse G, De Rose G, et al. Cost-effectiveness analysis of elective endovascular repair compared with open surgical repair of abdominal aortic aneurysms for patients at high surgical risk: a 1-year patient-level analysis conducted in Ontario, Canada. J Vasc Surg 2008;48:779–87.
14. http://www.cms.hhs.gov/MedicareProgramRatesStats/downloads/mktbskt-economic-index.pdf
15. Hobo R, Buth J; EUROSTAR collaborators. Secondary interventions following endovascular abdominal aortic aneurysm repair using current endografts. A EUROSTAR report. J Vasc Surg 2006;43:896–902.
16. Brewster DC, Jones JE, Chung TK, et al. Long-term outcomes after endovascular abdominal aortic aneurysm repair: the first decade. Ann Surg 2006;244:426–38.
17. Conners MS III, Sternbergh WC III, Carter G, et al. Secondary procedures after endovascular aortic aneurysm repair. J Vasc Surg 2002;35:992–6.
18. Hallett JW Jr, Marshall DM, Patterson TM, et al. Graft-related complications after abdominal aortic aneurysm repair: reassurance from a 36-year population-based experience. J Vasc Surg 1997;25:277–86.
19. Prinssen M, Buskens E, de Jong SE, et al. Cost-effectiveness of conventional and endovascular repair of abdominal aortic aneurysms: results of a randomized trial. J Vasc Surg 2007;46:883–90.
20. Feezor RJ, Huber TS, Berceli SA, et al. Impact of endograft design and product line on the device cost of endovascular aneurysm repair. J Vasc Surg 2008;47:499–503.
21. Noll RE, Tonnessen BH, Kim J, et al. Long-term postplacement cost comparison of AneuRx and Zenith endografts. Ann Vasc Surg 2008;22:710–15.
22. Bertges DJ, Zwolak RM, Deaton DH, et al. Current hospital costs and Medicare reimbursement for endovascular abdominal aortic aneurysm repair. J Vasc Surg 2003;37:272–9.

23. Kim JK, Tonnessen BH, Noll RE, et al. Reimbursement of long-term postplacement costs after endovascular abdominal aortic aneurysm repair. J Vasc Surg 2008;48:1390–5.
24. Sternbergh WC III, Greenberg RK, Chuter TAM, et al. Redefining postoperative surveillance after endovascular aneurysm repair: recommendations based on 5-year follow-up in the US Zenith multicenter trial. J Vasc Surg 2008;48:278–85.
25. Parent FN, Meier GH, Godziachvili V, et al. The incidence and natural history of type I and II endoleak: a 5-year follow-up assessment with color duplex ultrasound scan. J Vasc Surg 2002;35:474–81.
26. Steinmetz E, Rubin BG, Sanchez LA, et al. Type II endoleak after endovascular abdominal aortic aneurysm repair: a conservative approach with selective intervention is safe and cost-effective. J Vasc Surg 2004;39:306–13.

5

Pathology of Stents and Stent Endografts

Renu Virmani, M.D. and Frank D. Kolodgie, Ph.D.

Persistent high rates of restenosis after percutanous coronary intervention have encouraged the search for improved stents with the capability of localized drug delivery. The two recently FDA approved Cypher (Cordis, Johnson and Johnson Co) sirolimus-eluting and TAXUS (Boston Scientific) paclitaxel-eluting stents have dramatically reduced in-stent restenosis rates at 6 and 12 month. Large randomized clinical trials with sirolimus-eluting Cypher stent implants in uncomplicated focal de novo lesions report a 0 to 9% restenosis rate compared to 26% or 34% for uncoated Bx Velocity stents.[1,2] Similarly, paclitaxel-eluting stent TAXUS I, II, and IV clinical, trials report promising results at 6, 9, and 12 months with a significant reduction in binary restenosis: 2 to 5% versus 19% in the bare metal control Express stent.[3-5] The impact of drug-eluting stents however, may be marginal in complex lesions or subpopulations of high-risk patients such as diabetics. Substudy analysis of the SIRIUS (SIRolImUS-coated Bx Velocity balloon-expandable stent in the treatment of patients with de novo coronary artery lesions) trial showed a significant reduction in major adverse cardiac events in patients with and without diabetes mellitus. However, there remained a trend toward a higher frequency of repeat intervention in diabetic patients particularly those requiring insulin.[6] Consequently, even if drug-eluting stent technology can eliminate restenosis, disease progression may continue to impact the clinical outcome of diabetic patients.[7] Moreover, observational studies of drug-eluting stents have only recently begun to address complex lesions, diffuse coronary disease, saphenous vein grafts, bifurcating and ostial lesions, and plaques associated with acute coronary syndrome. Preliminary results of drug-eluting stents in complicated lesion morphologies suggest that restenosis may be reduced but certainly not eliminated.[8]

MECHANISMS OF RESTENOSIS

Excessive neointimal growth was originally proposed as one of the main causes of balloon angioplasty failure.[9] This notion, however, was dismissed on the realization that

negative remodeling from plaque burden and a decrease in the area bound by the external elastic lamina was principally responsible. In contrast, in-stent restenosis is a problem of unwarranted extracellular matrix produced by smooth muscle cells and is complicated by platelet deposition and inflammation.[8] The healing that occurs after stenting follows a progression of wound repair tightly controlled by chemotactic factors, inflammatory cytokines, growth factors, and mitotic signals critical to smooth muscle proliferation and migration. In human stented arteries, neointimal formation is also proportional to the circumferential tear of media, contradicting the "bigger is better" philosophy when it come to deploying stents.[10] Moreover, stent strut penetration of the necrotic core in an atherosclerotic plaque and persistent chronic inflammation further contribute to increased neointimal growth and restenosis.[10]

Early attempts at reducing restenosis with systemic agents likely failed as a result of inadequate drug concentrations at stent treatment sites and poor selection of drugs (antiplatelet/anticoagulant, calcium antagonists, omega-3 fatty acids, lipid lowering drugs, anti-inflammatory drugs, steroids, growth factor antagonists, angiotensin-converting enzyme inhibitors, and various anti-proliferative agents).[11] The current drugs (sirolimus/everolimus, paciltaxel) constitute more potent anti-inflammatory and anti-mitotic agents successful in treating transplant rejection and certain malignancies. These agents are loaded on the stents using polymers designed to deliver initial high local concentration of the drug followed by slower release kinetics over at least 30 days. Higher elution rates are desirable over the first seven days after stenting at a time when proliferation and inflammation peaks in animals models.

PRECLINICAL ANIMAL STUDIES

The biological responses to stenting in animal models are illustrated in Figure 5–1.[12] In normal arteries, deployment of a stent initiates platelet/fibrin deposition and recruitment of acute and chronic inflammation over the first one to seven days. Smooth muscle cell proliferation generally peaks in the media and intima at three days and seven days, respectively, and is accompanied by the migration of cells into the intima with the accumulation of proteoglycans and collagen matrix.[13,14] Endothelialization of bare stainless steel stent is generally >80% by seven days and full coverage of stent struts by neointimal tissue is complete by 28 days (Figure 5–1). Preclinical studies of drug-eluting stents loaded with immunosuppressant and chemotherapeutic agents at 28 days show a delay healing response characterized by persistent fibrin, sustained rates of smooth muscle cell proliferation, inflammation, and incomplete endothelialization. This morphology is similar to seven days of healing in bare stainless steel stents.[15-17] Animal studies with sirolimus-eluting stents at three and six months show a return of neointimal growth with complete healing and absence of fibrin.[12] Selective cytotoxic drugs may lead to even greater neointimal formation if accompanied by necrosis and inflammation, as reported with actinomycin-D.[18] Clearly, preclinical animal studies highly suggest that the effectiveness of the current generation of drug-eluting stents extends from a delay healing response and sustained suppression of neointimal growth is lost when the drug concentrations inevitably reaches a critical low.

Selective polymers designed for drug delivery have been implicated in the inflammatory response around stent struts in 28-day and 90-day implants. As many as 12.5% to 35% of polymer-coated stent implants examined at three months show inflammation with granulomas and extensive eosinophilic infiltration.[19] The degree of

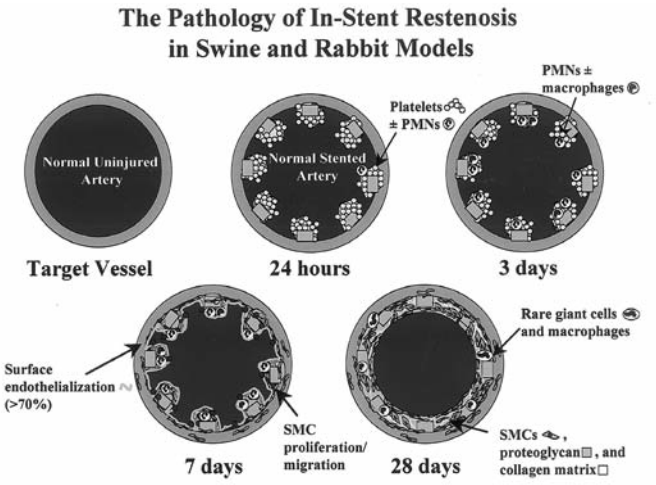

Figure 5-1. Cartoon illustration of the vascular response to a balloon expandable stainless steel stent implanted in a normal swine coronary or rabbit iliac artery. Abbreviations: PMNs = polymorphonuclear leukocytes, SMCs = smooth muscle cells. Reproduced with permission from Virmani et al. *Heart*. 2003;89:133–138.

inflammation may be further impacted at sites of stent overlap, characterized by eosinophils and giant cells. Similar inflammatory reactions occur in stainless steel stents but to a lesser degree. The mechanism(s) of granuloma formation in stents remain poorly understood. However, in animals receiving multiple drug-eluting and/or polymer coated or bare metal stents, granulomas have been observed in all three stents, with the greatest numbers occurring in drug-eluting stents suggesting that polymers and drugs may be responsible for the hypersensitivity.

ARE POLYMER DRUG-ELUTING STENTS TOTALLY INNOCUOUS?

Despite early preclinical and clinical studies suggesting that drug-eluting stents are the ultimate solution to restenosis, this conclusion may be premature. Since the approval of sirolimus-eluting (Cypher) stents, a recent U.S. Food and Drug Administration (FDA) alert reports a high incidence of subacute thrombosis (290 with 50 deaths) in over >450,000 implants.[20] The response issued by the FDA urges greater vigilance, with strict adherence to the label, making sure that stents are fully deployed and in contact with the vessel wall. The actual frequency of thrombosis with Cypher stents in the general population, however, cannot be precisely defined since the numbers thus far are derived from voluntary reporting, which in the United States typically represents an underestimation of the true incidence.

Since the release of TAXUS stents, similar concerns have been raised with 79 reported incidences of the polymer stent adhering to the deployment assembly causing an inability to withdraw the balloon. Emergency bypass procedures were instituted in at least 10 cases and there was one death.[21] Two French scientists report surface irregularities of the TAXUS stent produced by an uneven distribution of the polymer drug, along with polymer stickiness interfering with the separation of stent struts.[22] Our laboratory has recently examined an endarterectomy specimen from a patient receiving a TAXUS stent with a failed balloon assembly resulting in emergency bypass surgery

(RV, unpublished observation, 2004). The stent appeared to be poorly deployed in a lesion with circumferential calcification with the balloon sticking to the polymer-coated stent struts. Another case of subacute thrombosis occurring within the first month of TAXUS stent deployment showed total occlusion. A stent strut penetrated the necrotic core causing plaque and medial wall injury. Although no excessive inflammation was observed, excessive thrombus formation was noted at the injury site.

We have also recently examined four Cypher stents deployed for 7, 6 and 11, 38 and 5 days with subacute thrombosis (RV, unpublished observation, 2004). In these cases, in-stent thrombosis was attributed to inadequate deployment using a crush technique, diffuse disease extending beyond the stented segment, discontinuation of anti-platelet drugs, and multiple stents (one multilink, and one old Express with in-stent restenosis had been present prior to deploying two Cypher stents). In the latter case, all three recent stents thrombosed five days after receiving the implants and the patient died in the catheterization laboratory. The etiology of the thrombosis is unclear as stents were well deployed although the left circumflex and LAD stents occupied at least 8–10 cm of the artery length.

Inflammatory reactions to non-bioerodable stent polymers in selected patients may be responsible for the higher incidence of malapposition with drug-eluting stents. As reported in a subset of patients from the European RAndomized study with the sirolimus (SRL)-eluting Bx VELocity balloon expandable stent (RAVEL) study that underwent intravascular ultrasound (IVUS), the incidence of incomplete apposition was 21% for the sirolimus group and only 4% in patients with control uncoated stents.[23] Arguably, incomplete deployment at the time of stenting may have been responsible for the malapposition (initial IVUS was not performed); it is suspicious that the incidence was five-fold higher with drug-eluting stents. The inflammatory reaction to stent polymers is characterized by eosinophilic infiltration around the stent struts with or without granulomas. This response is typically incomplete at 28 days and reaches maximal around 90 days after stenting in animals. The combination of drug and polymer causes maximum inflammation relatively late as compared to bare stainless steel stents. Although the clinical consequences of malapposition if any are unclear, preclinical results suggest that malapposition may occur from excessive inflammation induced by the polymer and/or drug leading to positive remodeling and expansion of the vessel wall. In examination of another Cypher stent deployed for four months, there was excessive thrombosis of one artery with another showing a poorly expanded stent with restenosis, reaffirming that adequate stent deployment is just as essential with drug-eluting stents (RV, unpublished observation, 2004).

PATHOLOGY OF BALLOON EXPANDABLE STAINLESS STEEL STENTS IN HUMAN ARTERIES

The response to stenting in humans differs significantly from normal animal arteries since there is no underlying atherosclerotic disease. Autopsy studies of stented human coronary arteries suggest that rates of healing are delayed as compared to animals.[12, 24] The sequence of events in response to stenting of human coronary arteries is illustrated in Figure 5–2. The initial reaction is characterized by platelet and fibrin deposition along with an infiltration of acute inflammatory cells. Evidence of platelets/fibrin persists 14 to 30 days, and may be prolonged further with stent strut penetration into

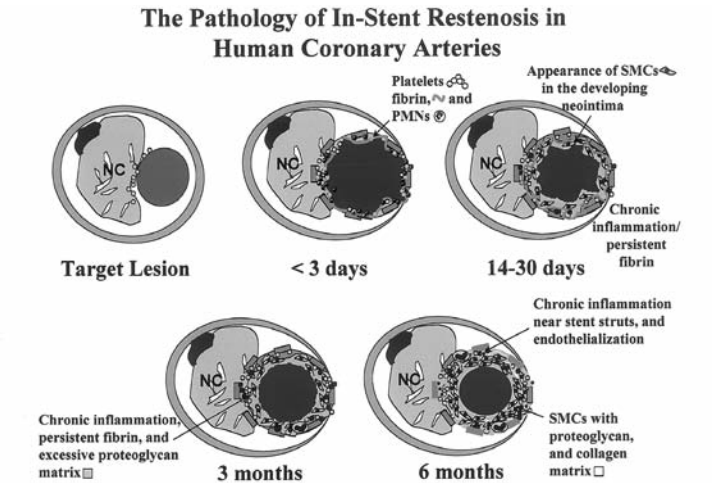

Figure 5-2. Cartoon illustration of the vascular response to a balloon expandable stainless steel stent implanted in an atherosclerotic human coronary artery. Abbreviations: NC = necrotic core, SMCs = smooth muscle cells. Reproduced with permission from Virmani et al. *Heart*. 2003;89:133–138.

the necrotic core and/or or if there is prolapse of necrotic debris into the lumen. Inflammatory cells, consisting of polymorphonuclear leukocytes and macrophages, are evident by one to three days, and macrophages persist for at least three months. Infiltration of T-lymphocytes occurs two to three weeks and continues beyond six months. Collections of smooth muscle cells, the main cellular component of the restenotic lesion, are evident by 14 days after stenting. The extracellular matrix, composed initially of proteoglycans and type III collagen, is gradually replaced by type I collagen beyond 18 months. The time course of intimal smooth muscle cell proliferation in relation to in-stent restenosis in humans is unknown. Cellular proliferation studies in human restenotic coronary atherectomy tissue retrieved from a few days to just beyond one year show a low proliferation index without the characteristic peak as found in existing animal models of angioplasty and stenting.[25] Clearly, significantly more rapid proliferative events appear to occur in animals as distinguished from human restenotic coronary arteries. Alternatively, smooth muscle cell migration from within the plaque or media to the expanding neointima may perhaps be the more dominant factor contributing to in-stent restenosis in humans.

We recently characterized several extracellular matrix molecules in autopsy specimens of stented human coronary arteries based on the age of the implant (three to nine months, >nine to 18 months, and >18 months).[26] Versican-hyaluronan were the two principle matrix molecules of the early neointima and are present up to 18 months. In contrast, decorin begins to appear after nine months but significant accumulation occurs only after18 months. Other early matrix components include type III, collagen although beyond 18 months—type I collagen is the dominant matrix component. Morphometric analysis showed that neointimal area was not significantly different among three to nine month and >nine to 18 month stents but was significantly less in stents >18 months at a time when the cross-linking of type I collagen likely leads to shrinkage of the neointima. Similarly, smooth muscle cell density in lesions >18 months may also contribute to a decrease in neointimal area.

One obvious explanation for the delay in arterial healing in humans stents is contingent on the underlying atherosclerotic plaque, which usually manifests in the fifth to sixth decade of life.[12] Arterial interventions in animals are usually performed in young adults and stents are typically placed in apposition to a normal smooth muscle-rich medial wall unburdened by inflammation. The absence of atherosclerotic disease likely contributes to a more predictable healing response in animals. In contrast, at least 70% of the stented human coronary arteries are in direct contact with the underlying atherosclerotic plaque.[10,24] The physical components of the lesion relative to the position of the stent likely affects the local response to healing. For example, stent struts in proximity to a necrotic core are exposed to only a paucity of smooth muscle cells, and thus heal slower than stents in direct contact with areas of adaptive intimal thickening, which contain an abundance of smooth muscle cells.[24] Similarly, stents overlying calcified and densely fibrotic plaques also take longer to develop a neointima since these plaques are also relatively hypocellular and must recruit smooth muscle cells from other remote areas of the arterial wall to cover bare struts.

STENT HEALING IN HUMANS AND ANIMALS

The differential rate of healing between animals and humans may also be proportional to the longevity of the species (Figure 5–3). The typical life span of a human is >70 years; in contrast, pigs have a life span of 16 years, and rabbits five to six years. The concept that biological differences in rates of healing are age-dependent is exemplified in animal models of cutaneous wounds. This analogy may be appropriate to in-stent restenosis since the developing neointima is considered a response to traumatic injury. In the swine, the extent of cutaneous reepithelialization declines with age partly because of a decrease in the expression of growth factors.[27] Further, wound contraction (analogous to "remodeling" in the stent) is markedly accelerated in juvenile as compared with adult pigs. The extent of injury is another consideration; wound healing is delayed in traumatic as compared to surgically induced injury, and if the injury site is large versus small.[28,29] Human coronary stenting is often associated with extensive local trauma characterized by plaque splitting and medial disruption. Conversely, most stents in animals are deployed in normal arteries with 1:1.1 stent to artery ratio resulting in only mild arterial injury.[30]

SIMILARITIES AND DIFFERENCES IN HEALING FOLLOWING DRUG-ELUTING AND BALLOON EXPANDABLE STAINLESS STEEL STENTS

Most follow-up studies of stented coronary arteries in humans use angiography or IVUS as a barometer of clinical success. Both methods, however, are limited since angiography only examines the lumen and is unable to determine the quality of the neointimal growth, and IVUS can only detect tissue that is echogenic. Most neointimal tissue from 28-day drug-eluting stents in animals is composed of fibrin and proteoglycan matrix with few smooth muscle cells (Figure 5–4). Since these components cannot be easily imaged by IVUS it is not surprising that six- to 12-month drug-eluting stents in humans appear bare or contain minimal neointimal growth.[23] Therefore, until

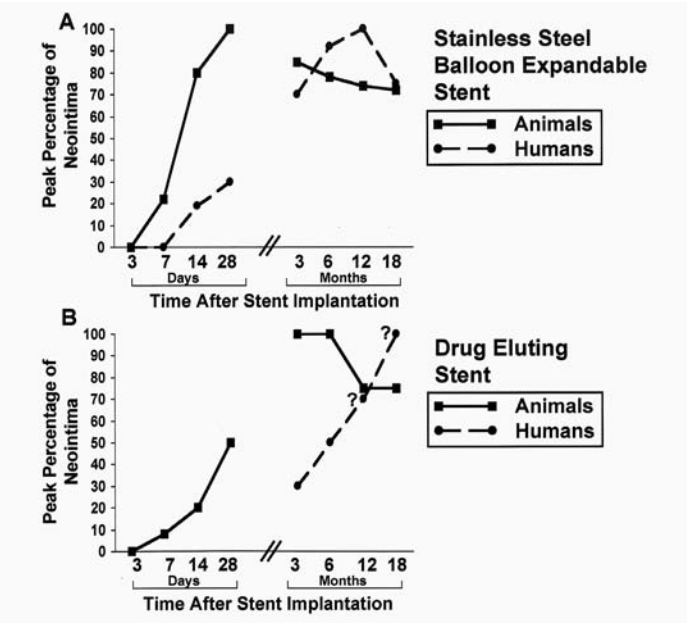

Figure 5-3. Line plot showing the temporal relationship of peak neointimal growth in animals and humans following the placement of either "bare" stainless steel **A** or drug eluting sirolimus or paclitaxel stents **B**. The plots are predominantly derived from morphometric analysis of swine and human coronary stents; the drug-eluting stent data in humans is projected from angiographic results. In animals, peak neointimal growth in stainless steel stents is observed at 28 days as compared with six to 12 months in humans. In human coronary arteries with drug-eluting stents, the precise time course of peak neointimal growth is unknown. Animal studies of drug-eluting stents however, show favorable results at 28 days with a lack of sustained efficacy at three and nine months. The generalized delayed healing with drug-eluting stents is thought to occur secondarily from an inhibition of smooth muscle cell proliferation and migration and/or the suppression of inflammation. Reproduced with permission from Virmani et al. Heart. 2003;89:133–138.

improved methods of imaging are available for the detection of early neointima, the precise identification of lesion components are best defined by atherectomy or autopsy specimens. A recent study of 15 patients treated for in-stent restenosis with QuaDS stents (Quanam Medical Corp) containing the paclitaxel derivative 7-hexanoyltaxol (QP2 or "taxen") has given insight into stent healing in humans.[31] Although at six months in-stent intimal hyperplasia was minimal (late loss = 0.47±1.01 mm), at 12 months, there was an aggressive increase in neointimal growth (late loss = 1.36±0.94 mm) resulting in a dramatic 61.5% rate of restenosis.[32] Morphologic examination of atherectomy tissue from a subset of these patients showed persistent fibrin admixed with smooth muscle cells and extensive proteoglycan matrix, thus demonstrating incomplete neointimal healing even at 12 months.[31]

We recently examined a single sirolimus-eluting (SRL) Bx Velocity stent from a 71-year-old woman enrolled in a RAVEL trial as treatment of a proximal LAD coronary artery stenosis of 80%. Both IVUS and angiography studies at six months showed a 0% stenosis and no evidence of in-stent neointimal growth. The patient remained asymptomatic until presentation with unstable angina 16 months after receiving the stent. Angiography at this time demonstrated a subtotal occlusion of the left obtuse marginal (LOM) artery. The drug-eluting stent continued to show 0% stenosis. Although the

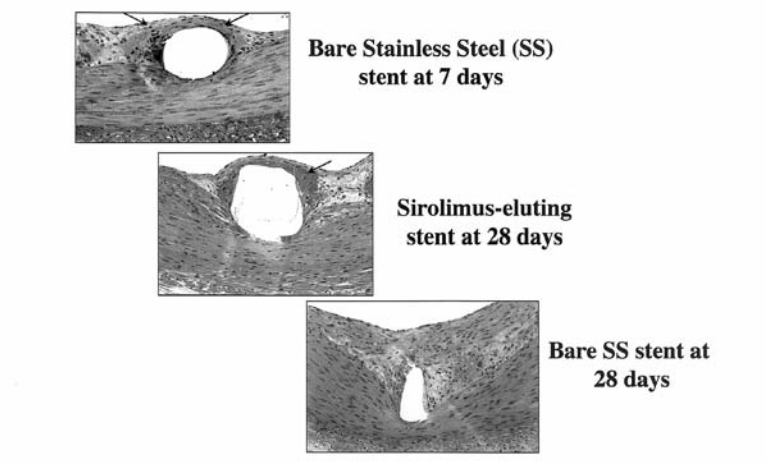

Figure 5-4. Delayed healing associated with drug-eluting stents in animals. Note the similar degree of fibrin deposition (arrows) in the 28-day sirolimus-eluting stent compared with the seven-day bare metal stent; chronic inflammatory cells are more prominent in the bare metal stent. The 28-day bare metal stent is completely healed; the smooth muscle cell-rich neointima has rare chronic inflammatory cells around stent struts and fibrin is absent.

lesion in the LOM was successfully stented, the patient died 24 hours after coronary intervention as a result of a stroke.[33] At autopsy, the SRL-eluting stent was widely patent and there was a minute thrombus at the ostium of a small side branch. Light microscopy showed a mild neointimal growth above the stent consisting of smooth muscle cells in a proteoglycan-rich matrix, with occasional giant cells with some fibrin near stent struts, in particular those penetrating the necrotic core. Scanning electron microscopy of the luminal stent surface showed >80% endothelialization with small foci of poorly formed endothelial cells junctions and rare platelet aggregates close to the side branch ostium. Since endothelialization of balloon expandable stainless steel stents most likely occurs by three to four months in human atherosclerotic arteries, the incomplete enothelialization in the SRL-eluting stent at 16 months suggests delayed healing, which may take >two years to complete in uncomplicated lesions. Collectively, histologic analysis of the few available samples from both QuaDS-QP2 and Cypher stents suggests delayed neointimal growth in humans relative to animals and emphasizes long-term patient following-up of longer than 18 months for appropriate evaluation of stent performance.

LIMITATIONS OF LOCALIZED DRUG-DELIVERY

There are critical limitations to using a stent platform for chronic local drug delivery since the pharmacologic agent cannot be given indefinitely and effective doses may be toxic to the vessel wall. Further, unreliable pharmacokinetics may result in the untimely release of the drug and a loss of efficacy. More importantly of late, the potential for long-term adverse effects secondary to polymeric coating has posed immediate concerns. The premature breakdown of a polymer coating or its ability to stimulate inflammation may limit any beneficial effect of the drug (Figure 5–5). In addition, drug-eluting stents need to be precisely deployed to avoid geographical miss. Despite

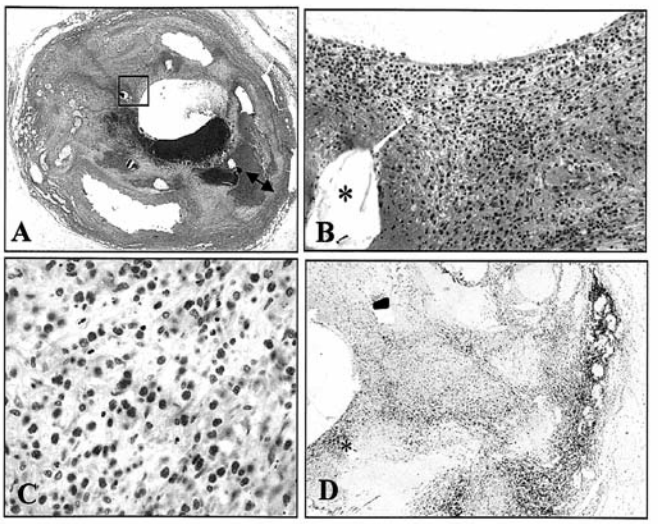

Figure 5-5. Distal sections of an 18-month-old Cypher stent implant deployed in the left circumflex coronary artery with late stent thrombosis **A**. Note the focal stent malapposition (double arrow) and luminal thrombus. **B** is high-power views of the boxed area in **A** showing extensive inflammation and absence of smooth muscle cells around a stent strut (asterisk). **C.** There is diffuse inflammation of the intima and media composed mostly of eosinophils (Luna stain). **D.** Shows extensive T-lymphocyte (CD45RO) infiltration and a focal giant cell reaction around a stent strut (asterisk).

dramatic inhibition of in-stent neointimal growth in the SIRIUS trial, there remained a persistent problem of restenosis at the peri-stent margins because balloon-injured sites unprotected by the stent may not have received adequate exposure to the drug. The disappointing performance of actinomycin D- eluting stents (ACTION Trial)[18] or QuaDS- (QP2 taxane analog)[31,32] suggests that the appropriate choice of drug is another critical element in reducing restenosis. Even if a drug holds promise, potential problems with polymeric coatings, very high concentration of an active drug, extended release kinetics, inhomogeneous distribution of the drug, and unequal expansion of struts are all undesirable factors.[34, 35] Thus, multiple aspects of stent design, independent of the drug, may determine its overall clinical success.

ENDOVASCULAR STENT GRAFTS

Endovascular stent grafts were initially developed for the treatment of abdominal aortic aneurysms.[36, 37] The covered design maximizes the effectiveness of the device by eliminating or inhibiting the migration of smooth muscle cells by a fabric barrier, thus preventing neointimal hyperplasia. Recent clinical results of stent grafts deployed in the saphenous vein grafts or peripheral vessels, however, have not been encouraging.[38-41] Graft materials for endovascular applications include PET (polyethylene terephthalate, Dacron), PTFE (polytetrafluoroethylene), and PU (polycarbonate urethane).[42] The ideal stent graft should consist of a stable thromboresistant material, which gets incorporated into native tissue without provoking excessive inflammation.[42] We and others have shown in the dog and ovine models of peripheral implants that PTFE induces greater inflammation compared with polyester or polycarbonate

urethane covered stents.[43-45] Moreover, increases in the degree of inflammation correlated with greater neointimal formation. In addition to the neointima, a second critical issue is thrombogenicity and endothelialization, essential factors for healing and long-term patency. Both are not exclusive and, in fact, some thrombus formation or protein deposition is essential for endothelialization. Stent endothelialization is essential for grafts to remain patent, and although it does not prevent an increase in neointimal formation, it can certainly delay its progress. The area where stent grafts are likely to have the most impact is treatment of abdominal aneurysms, thoracic aneurysms, peripheral artery aneurysms, and acute rupture of vessel, either iatrogenic- or disease-related. Here we will review the process of healing following the placement of stent grafts in peripheral vessels and aorta in animal models and discuss some general clinical complications of these devices.

Stent graft healing following deployment in animal models

The surface thrombogeneicity of a graft material is roughly correlated to the energy levels with greater the energy, the higher the thrombogenecity. For PET, the woven rather than knitted form is most commonly used in medical applications because it can be manufactured in thickness of 0.1 mm or less.[42] The biological response to PET consists of a fibrous encapsulation of the outer surface of the graft by an extensive inflammatory reaction at the graft surface with surrounding granulation tissue and fibrosis. A layer of thrombus covers the luminal surface, which is often fibrinous, although platelets and focal inflammatory cells are also observed with an absence of endothelial lining. At anastomotic sites, however, luminal narrowing can result from a robust neointimal reaction consisting of smooth muscle cells, inflammation, and accumulated extracellular matrix. Since PET is thrombogenic and provokes a severe foreign body reaction with lymphocytic and eosinophilic infiltrates, this material is unsuited for small grafts (<6 mm) although it is suitable for aortic grafts.

Among graft materials, PTFE is less thrombogenic than PET; however, it still induces inflammation as well as intense neointimal formation at anastomotic sites. According to some observers, this reaction is less intense than PET and, therefore, is preferred for grafts <6mm in diameter. Moreover, endothelialization of luminal surfaces is governed by the internodal density, porosity, and thickness of the graft and, therefore, designs can be optimized for small vessel diameters. Overall, long-term patency rates in peripheral vascular disease appear to be better for PTFE than PET; however, the duration of follow-up studies is still too short for definitive conclusions. Notably, stent-graft implanted in peripheral vessels in animal models irrespective of the type of graft material have the greatest neointimal formation compared to bare stents alone.[46] In comparison, PU grafts tends to induce greater inflammation and neointimal formation than PTFE covered stents.[42,43] Healing is also significantly slower in the presence of graft compared to bare metal stents.

Abdominal Aortic Aneurysms

Rupture of abdominal aortic aneurysms (AAA) is associated with high morbidity and mortality. Estimates in the general population suggest that AAA occur at the rate of 60 per 1000 individuals and between 1.8 to 6.6% according to autopsy studies.[47,48] As the aneurysm enlarges, the risk of rupture increases: 0% for aneurysms <4 cm in diameter; 1% for aneurysms 4 to 4.9 cm; 11% for aneurysms 5 to 5.9%; and = 25% for >6 cm.

Approximately 62% of patients presenting with ruptured AAA die prior to reaching the hospital. The overall mortality rate for rupture of AAA, including in-hospital deaths, is thought to be as high as 90%. Surgical morbidity and mortality rates range from 3–5% for elective operative repair; however, these rates increase significantly for patients with comorbid medical conditions, especially coronary artery disease, renal failure, and pulmonary obstructive disease. Parodi et al. first performed endovascular stent graft repair of AAA in 1991[37], and since then, over 25,000 stent grafts have been deployed worldwide with early promising results.

Preclinical Studies

Most preclinical models of AAA use normal aortas rather than surgically created abdominal aneurysms. Overall, most animal studies have shown good repair and sealing of the aneurysms, with occasional total occlusions and endoleaks, and poor endothelialization of the center of the graft. Neointimal thickening and endothelialization is limited to the proximal and distal anastomotic sites of the prosthesis. The promising results in animal models however, do not reflect the clinical experience, which have been encumbered by problems of deployment and migration of devices, endoleaks, material failure (Figure 5–6), and even aneurysm rupture.[49]

Morphologic Causes of Graft Failure following Clinical Deployment of Aortic Stent Grafts

Since over 25,000 aortic stent grafts have been implanted in patients, a vast clinical experience has been gained and tremendous knowledge has emerged regarding their mode of failure. The most extensively described complications of aortic stent grafts are

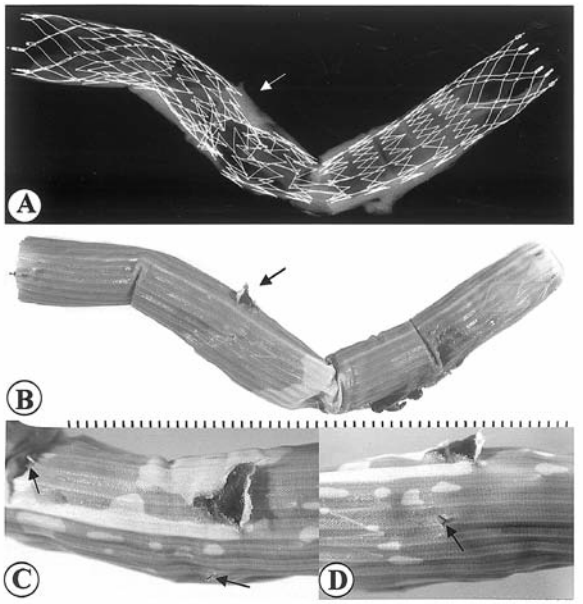

Figure 5-6. Postmortem radiograph (**A**) and gross photographs (**B** to **D)** of an endograft with fractured stent struts causing tears in the overlying Dacron graft with thrombus formation (arrow in **B**). Note there were multiple sites of protruded stent struts resulting in graft tears (**C** low and **D** higher power).

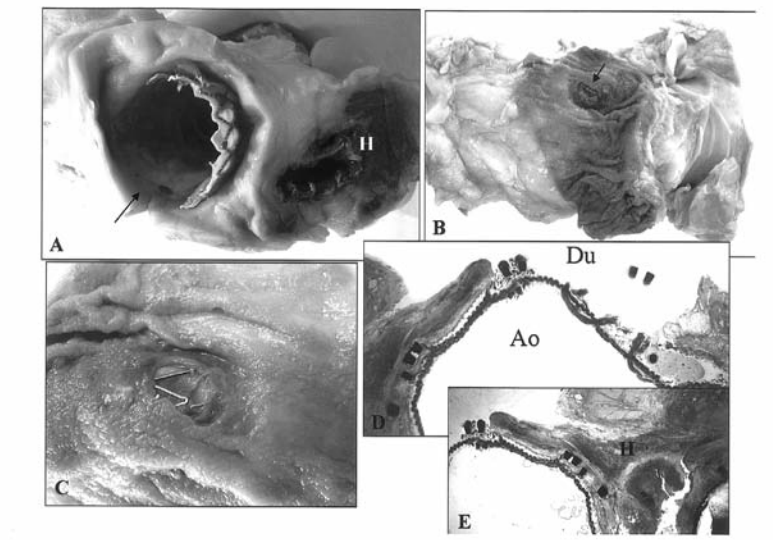

Figure 5-7. This case illustrates treatment of an abdominal aortic aneurysm with a stent graft in a 53-year-old man. **A.** There is good incorporation of the endograft by neointimal tissue on the left (arrow) with a relative absence on the right. **A.** Shows an encapsulated infrarenal hematoma (**H**) with erosion through the aortic wall (arrow in **B**). Stent strut perforation of the duodenal wall did not result in frank rupture because of the protection afforded by the Dacron graft (**B** and **C**). There was, however, a slow leak of blood into the surrounding tissues resulting in an infrarenal hematoma (**H**). **D.** Histologic section of the duodenal wall at the site of the perforation. Note an intact graft toward the aortic lumen (**Ao**) and the struts lying in the duodenum (**Du**). **E.** Shows hemorrhage (H) around the stent graft in communication with the infrarenal hematoma.

endoleaks and endotensions. Morphologically, endoleaks have been classified into four varieties of which types I and III have the most impact. Type I endoleak occurs from attachment leaks at proximal or distal ends of the stent-graft from failure to exclude the aneurysm causing pulsation, dilatation, and even eventual rupture of the aneurysm. We have morphologically identified distal endoleak in stent grafts poorly apposed to the iliac artery wall (Figures 5–7 and 5–8) accompanied by reduced attachment of the proximal end of the stent graft. Although these leaks mostly occur early, in our experience, some may occur late because of continued expansion of the aneurysm from the atherosclerotic process or stent-graft migration. Therefore, anchoring of the device is extremely important, which is likely dictated by the type of atherosclerotic plaque at the site of attachment. For example, if the distal end of the stent-graft overlies a large superficial necrotic core, leaks may be inevitable because of constant pulsatile motion and impaired vascular healing. Even in the presence of a thrombus, tissue organization is lacking because of a limited number of viable smooth muscle cells in the fibrous cap of ruptured or vulnerable plaques. Evaluation of the wall of the aorta and the iliac arteries by means other than angiography may be more appropriate for assessing sites with little or no atherosclerosis. Sealing is more likely to be achieved if no graft material is used at anastomotic sites. We have observed a case of aortic disruption occurring at the proximal stent-graft attachment site caused by the deep burrowing of stent struts into the necrotic core of an atherosclerotic plaque (Figure 5–9).

The two principal causes of type III endoleaks as reported by Jacobs et al, are fabric erosion and suture disruption.[50] In their series of 404 patients, 60 had material failure (Figure 5–6) due to suture disruption (n = 14), graft erosion (n = 5), and metal

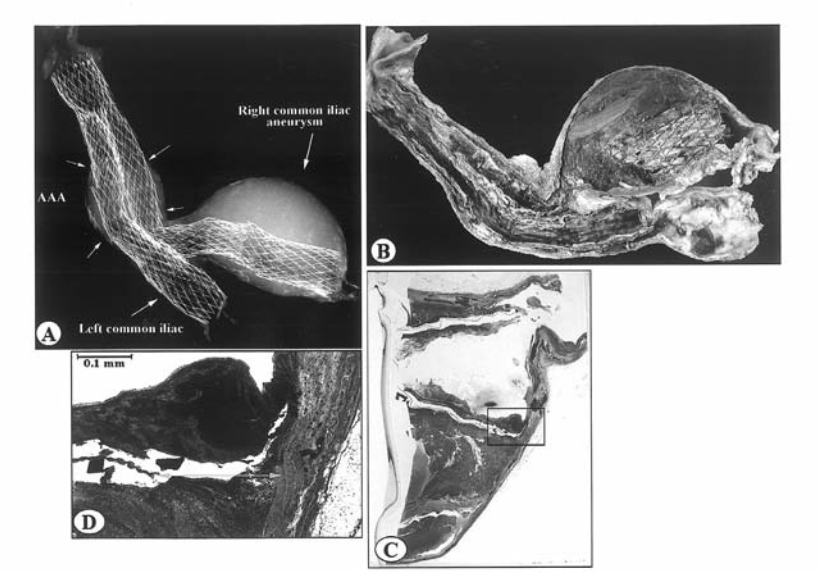

Figure 5-8. A 67-year-old man with abdominal aortic and bilateral iliac artery aneurysms treated with endovascular stent graft 2.5 years prior to death. **A.** Radiograph of the specimen shows a small abdominal aneurysm with a large right common iliac artery aneurysm. **B.** The specimen was bisected longitudinally and the right common iliac artery aneurysm, filled with fresh blood, measured 65 mm in diameter. The stent graft at the distal anastomotic site was not adherent to the aortic wall potentially causing a type I endoleak. **C** and **D** are low- and high-power micrographs, respectively, of histologic sections showing the gap between the stent graft and the aortic wall at the distal anastomotic site (doubleheaded arrow).

fracture (n = 43). Suture disruption had occurred from proximal row separation (n = 5) and within the body of the stent-graft (n = 9). It is believed that twisting motion caused by micromovements and circulation can lead to wear and finally suture rupture within the body of the stent-graft.[51,52] Norgren L, et al. reported a case of aortoenteric fistula occurring 10 months after the initial implantation.[53] At operation, the endograft fabric was found to be ruptured in an area of suture disruption between the nitinol stents. A pre-existing inflammatory process might have caused adhesions

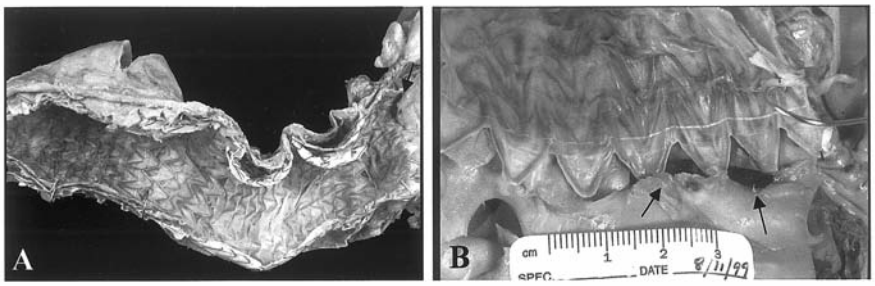

Figure 5-9. A 76-year-old man with multiple stent grafts in the arch, thoracic, and abdominal aorta implanted 61 days prior to sudden death following severe chest pain. After implanting three stent grafts, a CT scan showed an endoleak 6 days later. This leak was repaired with an additional endograft and a repeat CT scan 11 days later was unremarkable. At autopsy, there was an aortic dissection extending proximally resulting in a hemopericardium. A tear was identified at the proximal end of the stent graft near the junction of the normal aorta (arrows in **A** and **B**).

between the bowel and the aortic wall, predisposing to fistula formation. In a similar case of fabric erosion occurring months following implantation, the nitinol stent was found penetrating into the wall of the intestine resulting in a fistula communication; the stent outside the graft actually prevented aortic perforation into the duodenum (Figure 5–7). The stuttering rupture of the aorta into the retroperitoneal space caused an adjacent pseudoaneurysm.

In 1996, Moore and Rutherford first reported metal fractures in nine of 39 patients that underwent successful implantation of endovascular stent grafts (Figure 5–6).[54] Subsequently, Jacob et al reported 43 metal fractures in five different types of stent-grafts including nitinol stents (n = 37), 316L stainless steel stents (n = 5), and a (cobalt-chromium, nickel) stent (n = 1).[50] Stress fatigue fractures have been implicated in stainless steel stent-graft although metal corrosion has been postulated as the cause for the nitinol fractures. Stress fractures have also been observed in coronary stainless steel stents but are an uncommon phenomenon. Microcracks occur at sites of material irregularities commonly produced during metal laser cutting, and these, along with pulsatile flow within the aorta, may lead to surface disruption and crack propagation. Moreover, a better understanding of the nitinol material is likely to reduce the incidence of nitinol fractures.[55]

Type IV endoleaks, related to graft fabric porosity, are distinct from obvious holes or breaks in the stent-graft. Although once thought to be self-limiting as thrombus plugs the porous graft material, is now believed that aneurysms remain pressurized depending on the graft material. The Ancure nonporous stent-graft[56] resulted in a more rapid decline in aneurysm size than the porous AneuRx stent-graft.[57] However, despite a significant decrease in aneurysm sac over two years, late sac growth has been reported by three years, and the probability of freedom from sac growth or re-expansion at four years in AAA is only 43%.[58] Graft stent design may have an impact on sac behavior. Of 723 patients evaluated at the Cleveland Clinic, 39.1% had endoleaks (Ancure, 58.1%; Excluder, 34.7%; Zenith, 20.9%; $p < 0.001$), and the reduction of sac size was greatest with the Zenith graft, followed by Ancure and Excluder grafts. Also, the baseline size was positively correlated with rate of aneurysm shrinkage.[59]

Thoracic abdominal aneurysms (TAA) and dissections have also been treated with stent grafts; however, clinical experience is limited since the disease is less prevalent (Figures 5– 9 and 5–10). The incidence of descending TAA is increasing, and according to a recent Olmsted County population-based study, the incidence of descending thoracic aortic aneurysm is 10.4 cases per 100.000 person-years.[60] This rise is three-fold greater than previous reports and may reflect an aging population or better detection capabilities.[61] Of the 54 patients reported by Czerny et al. in-hospital mortality was 3.7% with a mean follow-up of 38 months; 7.7% had type I endoleak, 13.5% type II endoleak (branch leaks), and 7.7% had type III endoleaks.[62] Only three patients required treatment of endoleaks consisting of proximal stenting, open surgical repair, and embolization, and the three-year survival was 63%.

In summary, the many stent designs produce individual biological responses dependent on the polymer, selection of drug, and its release kinetic. The delayed healing induced by drug-eluting stents provides a reduction in neointimal growth in patients up to two years. Sustained suppression of neointima thus far in animals, however, is lacking, and long-term results are similar to bare metal stents. The potential exists that neointimal formation may even increase, especially with cytotoxic drugs. Moreover, polymers and/or drugs may induce hypersensitivity reactions in sensitive patients with the possibility of late stent thrombosis. Only long-term clinical data in a wide

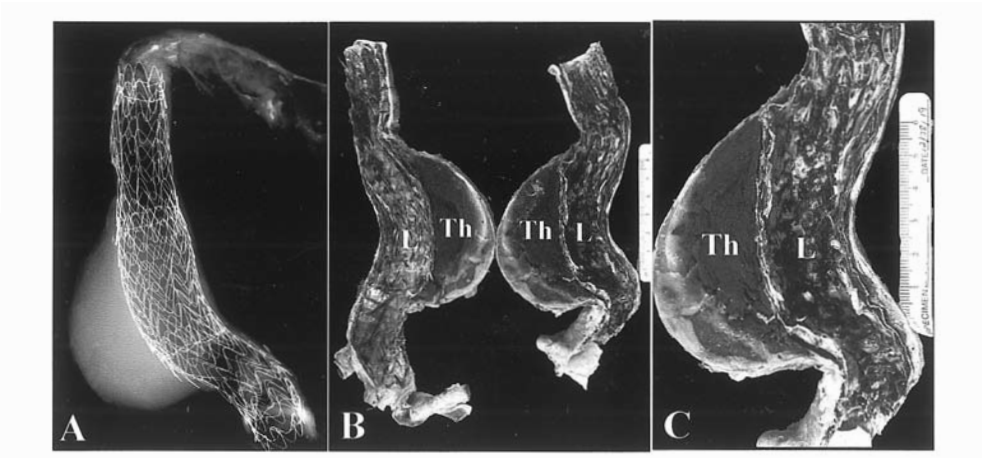

Figure 5-10. A postmortem radiograph of a thoracic aortic aneurysm treated with two percutaneously placed thoracic excluder stent grafts (**A**). Shortly after the procedure, the patient suffered two strokes and died on the sixth hospital day from respiratory failure. **B** and **C** are gross photographs of the aortic stent grafts cut longitudinally. Note the thrombus (**Th**) within the body of the aneurysm is totally excluded from the stent graft lumen (**L**).

range of lesion types will support the true safety of the drug-eluting stents. Stent grafts have unique problems of endoleaks, endotension, metal stress fractures, corrosion, and migration. Thus far, most of the clinical experience with stent grafts has been in the treatment of abdominal aortic aneurysms, and their expanded use should lead to design improvements and better outcomes.

REFERENCES

1. Morice MC, Serruys PW, Sousa JE, et al. A randomized comparison of a sirolimus-eluting stent with a standard stent for coronary revascularization. *N Engl J Med.* 2002;346:1773–1780.
2. Holmes DR, Jr., Leon MB, Moses JW, et al. Analysis of 1-year clinical outcomes in the SIRIUS trial: a randomized trial of a sirolimus-eluting stent versus a standard stent in patients at high risk for coronary restenosis. *Circulation*. 2004;109:634–640.
3. Grube E, Silber S, Hauptmann KE, et al. TAXUS I: six- and twelve-month results from a randomized, double-blind trial on a slow-release paclitaxel-eluting stent for de novo coronary lesions. *Circulation*. 2003;107:38–42.
4. Tanabe K, Serruys PW, Degertekin M, et al. Chronic arterial responses to polymer-controlled paclitaxel-eluting stents: comparison with bare metal stents by serial intravascular ultrasound analyses: data from the randomized TAXUS-II trial. *Circulation*. 2004;109: 196–200.
5. Stone GW, Ellis SG, Cox DA, et al. One-year clinical results with the slow-release, polymer-based, paclitaxel-eluting TAXUS stent: the TAXUS-IV trial. *Circulation*. 2004;109:1942–1947.
6. Moussa I, Leon MB, Baim DS, et al. Impact of sirolimus-eluting stents on outcome in diabetic patients: a SIRIUS (SIRolImUS-coated Bx Velocity balloon-expandable stent in the treatment of patients with de novo coronary artery lesions) substudy. *Circulation*. 2004;109: 2273–2278.
7. Loutfi M, Mulvihill NT, Boccalatte M, et al. Impact of restenosis and disease progression on clinical outcome after multivessel stenting in diabetic patients. *Cathet Cardiovasc Interv*. 2003;58:451–454.

8. Froeschl M, Olsen S, Ma X, O'Brien ER. Current understanding of in-stent restenosis and the potential benefit of drug eluting stents. *Curr Drug Targets Cardiovasc Haematol Disord*. 2004;4:103–117.
9. Schwartz RS, Henry TD. Pathophysiology of coronary artery restenosis. *Rev Cardiovasc Med*. 2002;3 Suppl 5:S4–S9.
10. Farb A, Weber DK, Kolodgie FD, et al. Morphological predictors of restenosis after coronary stenting in humans. *Circulation*. 2002;105:2974–2980.
11. Mody VH, Durairaj A, Mehra AO. Pharmacological approaches to prevent restenosis. In: Faxon DP, ed. *Restenosis: A Guide to Therapy*. London, UK: Martin Dunitz; 2001:97–112.
12. Virmani R, Kolodgie FD, Farb A, Lafont A. Drug eluting stents: are human and animal studies comparable? *Heart*. 2003;89:133–138.
13. Carter AJ, Laird JR, Farb A, et al. Morphologic characteristics of lesion formation and time course of smooth muscle cell proliferation in a porcine proliferative restenosis model. *J Am Coll Cardiol*. 1994;24:1398–1405.
14. Kollum M, Kaiser S, Kinscherf R, et al. Apoptosis after stent implantation compared with balloon angioplasty in rabbits. Role of macrophages. *Arterioscler Thromb Vasc Biol*. 1997;17: 2383–2388.
15. Farb A, Heller PF, Shroff S, et al. Pathological analysis of local delivery of paclitaxel via a polymer-coated stent. *Circulation*. 2001;104:473–479.
16. Farb A, John M, Acampado E, et al. Oral everolimus inhibits in-stent neointimal growth. *Circulation*. 2002;106:2379–2384.
17. Kolodgie FD, John M, Khurana C, et al. Sustained reduction of in-stent neointimal growth with the use of a novel systemic nanoparticle paclitaxel. *Circulation*. 2002;106:1195–1198.
18. Serruys PW, Ormiston J, Degertekin M, et al. Actinomycin-eluting stent for coronary revascularization: A randomized feasibility and safety study (The ACTION Trial). *J Am Coll Cardiol*. 2004;(in press).
19. Virmani R, Guagliumi G, Farb A, et al. Localized hypersensitivity and late coronary thrombosis secondary to a sirolimus-eluting stent: should we be cautious? *Circulation*. 2004;109: 701–705.
20. FDA advises physicians of adverse events associated with Cordis Cypher coronary stents. *U.S. Food and Drug Administration Public Health Web Notification*. 2003;T03–71 October 29.
21. Bulkeley WM. Boston Scientific recalls 200 stents after patient dies. In: *The Wall Street Journal*; 2004:D8.
22. Peterson L. Is there a problem with Taxus stents? In: *Trends in Medicine*; 2004.
23. Serruys PW, Degertekin M, Tanabe K, et al. Intravascular ultrasound findings in the multicenter, randomized, double-blind RAVEL (RAndomized study with the sirolimus-eluting VElocity balloon-expandable stent in the treatment of patients with de novo native coronary artery Lesions) trial. *Circulation*. 2002;106:798–803.
24. Farb A, Sangiorgi G, Carter AJ, et al. Pathology of acute and chronic coronary stenting in humans. *Circulation*. 1999;99:44–52.
25. O'Brien ER, Alpers CE, Stewart DK, et al. Proliferation in primary and restenotic coronary atherectomy tissue. Implications for antiproliferative therapy. *Circ Res*. 1993;73:223–231.
26. Farb A, Kolodgie FD, Hwang J-Y, et al. Extracellular matrix changes in stented human coronary arteries. *Circulation*. 2004;(in press).
27. Yao F, Visovatti S, Johnson CS, et al. Age and growth factors in porcine full-thickness wound healing. *Wound Repair Regen*. 2001;9:371–377.
28. Forrester JS, Fishbein M, Helfant R, Fagin J. A paradigm for restenosis based on cell biology: clues for the development of new preventive therapies. *J Am Coll Cardiol*. 1991;17:758–769.
29. Schwartz RS, Huber KC, Murphy JG, et al. Restenosis and the proportional neointimal response to coronary artery injury: results in a porcine model. *J Am Coll Cardiol*. 1992;19: 267–274.
30. Taylor AJ, Gorman PD, Kenwood B, et al. A comparison of four stent designs on arterial injury, cellular proliferation, neointima formation, and arterial dimensions in an experimental porcine model. *Cathet Cardiovasc Interv*. 2001;53:420–425.

31. Virmani R, Liistro F, Stankovic G, et al. Mechanism of late in-stent restenosis after implantation of a paclitaxel derivate-eluting polymer stent system in humans. *Circulation*. 2002;106: 2649–2651.
32. Liistro F, Stankovic G, Di Mario C, et al. First clinical experience with paclitaxel derivative-eluting polymer stent system implantation for in-stent restenosis. Immediate and long-term clinical and angiographic outcome. *Circulation*. 2002;105:1883–1886.
33. Guagliumi G, Farb A, Murumeci G,et al. Sirolimus-eluting stent implanted in human coronary artery for 16 months: Pathologic Findings. *Circulation*. 2003:In Press.
34. Hwang CW, Wu D, Edelman ER. Physiological transport forces govern drug distribution for stent-based delivery. *Circulation*. 2001;104:600–605.
35. Hwang CW, Edelman ER. Arterial ultrastructure influences transport of locally delivered drugs. *Circ Res*. 2002;90:826–832.
36. Blum U, Voshage G, Lammer J, et al. Endoluminal stent-grafts for infrarenal abdominal aortic aneurysms. *N Engl J Med*. 1997;336:13–20.
37. Parodi JC, Palmaz JC, Barone HD. Transfemoral intraluminal graft implantation for abdominal aortic aneurysms. *Ann Vasc Surg*. 1991;5:491–499.
38. Henry M, Amor M, Cragg A, et al. Occlusive and aneurysmal peripheral arterial disease: assessment of a stent-graft system. *Radiology*. 1996;201:717–724.
39. Lammer J, Dake MD, Bleyn J, et al. Peripheral arterial obstruction: prospective study of treatment with a transluminally placed self-expanding stent-graft. International Trial Study Group. *Radiology*. 2000;217:95–104.
40. Baldus S, Koster R, Reimers J, Hamm CW. Membrane-covered stents for the treatment of aortocoronary vein graft disease. *Cathet Cardiovasc Interv*. 2000;50:83–88.
41. Schachinger V, Hamm CW, Munzel T, et al. A randomized trial of polytetrafluoroethylene-membrane-covered stents compared with conventional stents in aortocoronary saphenous vein grafts. *J Am Coll Cardiol*. 2003;42:1360–1369.
42. Palmaz JC. Review of polymeric graft materials for endovascular applications. *J Vasc Interv Radiol*. 1998;9:7–13.
43. Cejna M, Virmani R, Jones R, et al. Biocompatibility and performance of the Wallstent and several covered stents in a sheep iliac artery model. *J Vasc Interv Radiol*. 2001;12:351–358.
44. Virmani R, Kolodgie FD, Dake MD, et al. Histopathologic evaluation of an expanded polytetrafluoroethylene-nitinol stent endoprosthesis in canine iliofemoral arteries. *J Vasc Interv Radiol*. 1999;10:445–456.
45. Sangiorgi G, Arbustini E, Lanzarini P, et al. Nonbiodegradable expanded polytetrafluoroethylene-covered stent implantation in porcine peripheral arteries: histologic evaluation of vascular wall response compared with uncoated stents. *Cardiovasc Intervent Radiol*. 2001;24:260–270.
46. Cejna M, Virmani R, Jones R, et al. Biocompatibility and performance of the Wallstent and the Wallgraft, Jostent, and Hemobahn stent-grafts in a sheep model. *J Vasc Interv Radiol*. 2002;13:823–830.
47. Melton LJ, 3rd, Bickerstaff LK, Hollier LH, et al. Changing incidence of abdominal aortic aneurysms: a population-based study. *Am J Epidemiol*. 1984;120:379–386.
48. Reilly JM, Tilson MD. Incidence and etiology of abdominal aortic aneurysms. *Surg Clin North Am*. 1989;69:705–711.
49. Veith FJ, Baum RA, Ohki T, et al. Nature and significance of endoleaks and endotension: summary of opinions expressed at an international conference. *J Vasc Surg*. 2002;35: 1029–1035.
50. Jacobs T, Teodorescu V, Morrissey N, et al. The endovascular repair of abdominal aortic aneurysm: an update analysis of structural failure modes of endovascular stent grafts. *Semin Vasc Surg*. 2003;16:103–112.
51. Jacobs TS, Won J, Gravereaux EC, et al. Mechanical failure of prosthetic human implants: a 10-year experience with aortic stent graft devices. *J Vasc Surg*. 2003;37:16–26.
52. Riepe G, Whiteley MS, Wente A, et al. The effect of autoclave resterilisation on polyester vascular grafts. *Eur J Vasc Endovasc Surg*. 1999;18:386–390.

53. Norgren L, Jernby B, Engellau L. Aortoenteric fistula caused by a ruptured stent-graft: a case report. *J Endovasc Surg*. 1998;5:269–272.
54. Moore WS, Rutherford RB. Transfemoral endovascular repair of abdominal aortic aneurysm: results of the North American EVT phase 1 trial. EVT Investigators. *J Vasc Surg*. 1996;23:543–553.
55. Teosh SH. Fatigue of biomaterials: A review. *Int J Fatigue*. 2000;22:825–837.
56. Greenberg RK, Lawrence-Brown M, Bhandari G, et al. An update of the Zenith endovascular graft for abdominal aortic aneurysms: initial implantation and mid-term follow-up data. *J Vasc Surg*. 2001;33:S157–S164.
57. Zarins CK, Arko FR, Crabtree T, et al. Explant analysis of AneuRx stent grafts: relationship between structural findings and clinical outcome. *J Vasc Surg*. 2004;40:1–11.
58. Cho JS, Dillavou ED, Rhee RY, Makaroun MS. Late abdominal aortic aneurysm enlargement after endovascular repair with the Excluder device. *J Vasc Surg*. 2004;39:1236–41; discussion 2141–2142.
59. Greenberg RK, Deaton D, Sullivan T, et al. Variable sac behavior after endovascular repair of abdominal aortic aneurysm: analysis of core laboratory data. *J Vasc Surg*. 2004;39:95–101.
60. Clouse WD, Hallett JW, Jr., Schaff HV, et al. Improved prognosis of thoracic aortic aneurysms: a population-based study. *JAMA*. 1998;280:1926–1929.
61. Bickerstaff LK, Pairolero PC, Hollier LH, et al. Thoracic aortic aneurysms: a population-based study. *Surgery*. 1982;92:1103–1108.
62. Czerny M, Cejna M, Hutschala D, et al. Stent-graft placement in atherosclerotic descending thoracic aortic aneurysms: midterm results. *J Endovasc Ther*. 2004;11:26–32.

6

The Acquisition of Endovascular Skills by Vascular Surgeons

John D. Edwards, M.D., FACS

Endovascular skill set acquisition by the practicing vascular surgeon remains a logistical quagmire in which many a vascular surgeon continues to be bogged down. The educational solutions of the past are inadequate for the challenges of today. As our specialty's problems are new, so must our educational and training paradigms be new. A number of assumptions have been made regarding endovascular training requirements for the standard experienced vascular surgeon. Most of these assumptions are based on case number requirements that are basically reiterations of residency training standards for other specialties such as interventional cardiology and radiology. The assumption that an experienced vascular surgeon would need similar endovascular case numbers to a radiology resident or a cardiology fellow in training, in order to attain competency in these skills, seems naïve. In addition, this premise has never been studied in any prospective or retrospective manner. These assumptions and the resultant numerical case requirements have created major impediments to practicing vascular surgeons trying to acquire endovascular skills. Exciting and innovative alternative endovascular training venues exist which should be utilized to facilitate the established vascular surgeon in his/her acquisition of these endovascular skill sets.

This chapter will offer one endovascular surgeon's perspective and suggestions for alternative training methods. The University of Cincinnati's Division of Vascular Surgery performs all of its own diagnostic and interventional procedures. The author, and director of the division, has been doing all of his own endovascular procedures since 1994. Over the last eight years the author has trained four junior attendings and six vascular fellows (at University of Cincinnati's fully accredited two-year clinical vascular fellowship) in the full range of endovascular procedures. Each of our fellows has performed over 750 endovascular procedures during their fellowship. The University of Cincinnati is also a recognized site for mini-fellowship training and has trained two three-month mini-fellows. The University also offers preceptorships (usually one week experiences in the peripheral angio suite), and has run some fifty basic

introductory endovascular training courses that have been attended by over 160 general and vascular surgeons. It has been my immense privilege to interact with these "endovascular students" as their teacher and instructor and it is based on my observations of their learning experiences that I offer some of the following insights.

It is a generally accepted maxim that practicing vascular surgeons wishing to add endovascular skills to their armamentarium would require a minimum of a three-month experience and 150 endovascular procedures. My aforementioned experience with various types of trainees simply does not support that contention. It is time the various vascular surgical societies help expose this misconception and help create an atmosphere conducive to endovascular skill acquisition by vascular surgeons already out in practice. There are many different mechanisms and training modalities that can, do, and will continue to produce conscientious, careful, and competent endovascular surgeons who perform minimally invasive catheter based vascular interventions producing excellent outcomes with acceptable low complication rates.

BASIC GUIDEWIRE AND CATHETER SKILLS

This chapter intends to propose one possible modular training paradigm for the practicing vascular surgeon who has little or no endovascular experience. Modifications of the proposed system could be readily adapted for surgeons with varying levels of experience by using the modularity of the program to their advantage.

Those vascular surgeons with little or no significant endovascular experience will need to begin with a basics course in guidewire and catheter manipulation. The course should be $2\frac{1}{2}$ days of intense hands-on experience in an animal lab or Virtual Reality Endovascular Simulator. These basic skills are readily acquired in a very short time by the average practicing vascular surgeon. The author has taught basic skill sets to over 160 participants of short animal courses and eight preceptors. Most participants easily acquired the basic catheter and guidewire skill sets in two to three days of intense hands-on training. The course syllabus introduces the skill sets required for performing diagnostic angiography, beginning with basic percutaneous needle access, fluoroscopic guided guidewire advancement, catheter placement and reforming of the diagnostic catheter in the aortic lumen followed by power injected contrast delivery and aortography. A porcine animal model is useful since arterial vessel size allows the use of most catheters. In a $2\frac{1}{2}$-day animal skills lab course this usually consumes the first $\frac{1}{2}$ day with the participants repeating the procedures dozens of times.

The second $\frac{1}{2}$ of the first day is spent learning how to use angled guidewires, torque devices and various diagnostic catheters designed for selective arterial catheterization. The average vascular surgeon readily acquires the skills to reform "Soss catheters" or "Omni-flush" catheters and advance a guidewire over the aortic bifurcation followed by selective catheter placement in the contralateral femoral artery for selective lower extremity angiography. It is a continuously rewarding experience for this instructor to watch the "veil of difficulty," felt to attend these catheter techniques, melt away in a matter of hours. While the participants are learning these catheter and guidewire skills they are simultaneously learning use of the fluoroscopy unit and its various modalities such as DSA, standard fluoro, and road-mapping techniques. They learn contrast bolus chasing and standard contrast volumes and rates of injection for different studies and techniques. All these techniques are very straightforward and contained in a syllabus for their review at later dates. Our course also teaches throm-

bolytic catheter and wire placement such as the Mewissen catheter and Katzen wire set up co-axially, using a Touhy-Borst adaptor, for split dose infusion.

The second day of the basics course is spent teaching use of balloon catheters, vessel sizing techniques, use of insufflators and techniques such as the "kissing balloon" technique for aortoiliac bifurcation PTA. Various balloons are placed over various wires after selective vessel catheterization has been performed thus allowing the course participants to repeat the previous days technical learning points. Most of the rest of this 2nd day is spent in teaching and performing stent deployments of both self-expanding as well as balloon expandable stents. The course participant deploys at least 12-15 stents in the course. The last $^1/_2$ day is spent reviewing all the techniques learned. The participants are given various task requirements and perform them under review of the course instructor. This final $^1/_2$ day seems to be valuable in consolidating and reinforcing the prior 2 days experiences.

The animal lab basic guidewire and catheter skills module is designed to provide entry-level endovascular skill acquisition to the novice. This introductory module is essentially a vocabulary lesson introducing device nomenclature, design and intended applications. Industrial product design evolution over the last decade has created an impressive array of device modifications. This can create what initially appears to be a daunting task for the novice who has to learn this vast array of device names. However, the product development has made the individual tasks of selective catheterization easier and less potentially traumatic to the vessel. So, the novice learning these catheter and guidewire skills now, actually has an easier task than the earlier angiographers of even a decade ago. Learning these basic catheter and guidewire skills in an animal lab setting allows repetitive performance and practice of the skill sets, free from anxiety over patient complications, until the participant has developed a comfort level with the procedures. Acquiring these basic skill sets in this fashion now maximizes the trainee's experience during the next level of their training when they move into the clinical preceptorship weeks where they are actually performing clinical cases under the direct supervision of a trained endovascular specialist.

The animal lab basic skills course allows the trainee to enter the next phase, the preceptor phase of training, which involves actual endovascular procedural activity, in a peripheral angiography suite setting, under the direct instruction of an experienced endovascular surgeon. This should be a weeklong experience in a vascular surgical practice where the preceptor will optimally be able to perform 15 to 20 endovascular procedures during that week. Because the basic skills have already been acquired in the animal course the preceptor phase will be more efficiently utilized by course instructor and trainee since the participant has already acquired the basic skill sets. In this manner patient safety is maximized while simultaneously allowing the course instructor to more expediently increase the trainee's role from first assistant to surgeon over the course of the week's experience. We will return to the discussion of the preceptor modules again later. But more discussion on the basic skills acquisition needs consideration.

VIRTUAL REALITY ENDOVASCULAR SIMULATOR TRAINING

Although the introductory animal course provides basic guidewire and catheter skill sets that are best taught in a non-patient care environment the animal models are imperfect in several regards. First, is the difference in anatomy. In the porcine model selective

catheterization of the thoracic arch vessels, carotids and renal arteries is too simple. Second, the vessels are free of atherosclerotic disease thus making catheter and guidewire maneuvers "deceptively" easy. Third, in the basics course there is no vessel pathology to actually intervene upon thus reducing the realism involved in learning balloon angioplasty techniques and stent deployment. The transition from basics course to the first clinical preceptor module therefore requires a somewhat uncomfortable quantum leap. The solution to this lies in the use of virtual reality (VR) and endovascular simulators instead of the animal lab for the basic skills acquisition. There have been simultaneous industrial advances in both endovascular technology and the means for instructing vascular surgeons in the skill acquisition for use of this endovascular technology. Treatment and training are in co-evolution. Use of VR simulators will reduce time away from the surgeon's practice previously required to attain these skills. It will enhance performance and increase patient safety in the endovascular surgeon's early stages of independent practice.

Endovascular virtual reality simulator based training has numerous advantages. Since the fluoroscopic images are virtual angiograms the trainee and instructor are spared radiation exposure during the basic skills acquisition process in which fluoroscopic times in the animal lab tend to be long due to the inexperience of the trainee. The virtual reality angiograms recreate human vascular anatomy and pathology. The complexity of the anatomy and pathology can be increased as the trainee gains in experience. The course instructor can introduce complications and adverse events into the procedure that teach the trainee how to deal with these issues. In this manner the virtual reality endovascular simulator is far superior to animal lab training and provides better continuity in transition from the learning facility to the actual peripheral angio suite and involvement in actual patient procedures. Use of vascular VR simulators will take the learning curve into the classroom and out of the angio suite and thus enhance patient safety during the surgeon's clinical endovascular training and early practice integration.

Two simulator systems are currently available and both are truly impressive educational training tools. The Procedicus VIST® (Vascular Intervention Training Program) system, distributed in North America by Medical Education Technologies, Inc (METI), is a real-time physics based system (Figures 6–1, 6–2). The author has used the system and the angiographic image fidelity is outstanding. Virtual reality intervention occurs real time and even an experienced endovascular specialist feels the "reality." The current weakness in this system lies in the haptics and the tactile feedback needs improvement. The company is currently working on that issue. Another issue is the peripheral vascular course material is currently limited to carotid and renal arterial case intervention. However, even these virtual cases can be used to teach basic catheter and guidewire skills since the trainee can direct the guidewires and catheters into any vessel, i.e., selective Soss catheter advancement over the aortic bifurcation, etc.

The other virtual reality system is the SimSuite® System from Medical Simulation Corporation, based out of Denver, Colorado (Figures 6–3, 6–4). This simulator closely replicates a true endovascular suite, including a simulated patient, wire and catheter-based delivery system, hemodynamic monitoring, fluoroscopic monitor and even pharmacologic management capability. The SimSuite® simulation uses real patient angiographic data to create clinically accurate patient anatomy and atherosclerotic pathology. The image fidelity of the simulated angiograms is truly incredible and the virtual reality interventions are essentially indistinguishable from an actual case intervention.

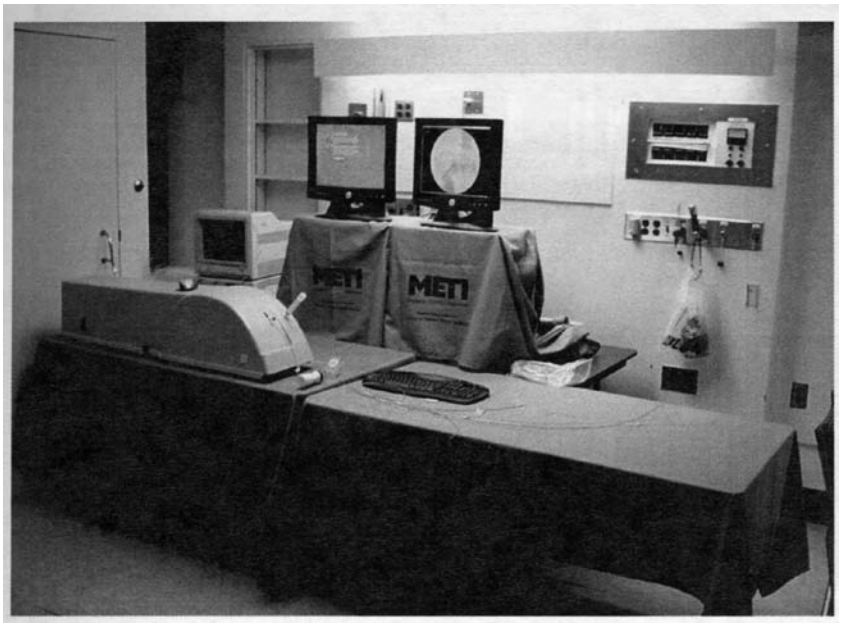

Figure 6-1. The Procedicus VIST® (Vascular Intervention Training Program) system, distributed in North America by Medical Education Technologies, Inc (METI). (Courtesy of David M. Hananel, Director, Surgical Programs, Medical Education Technologies, Inc.)

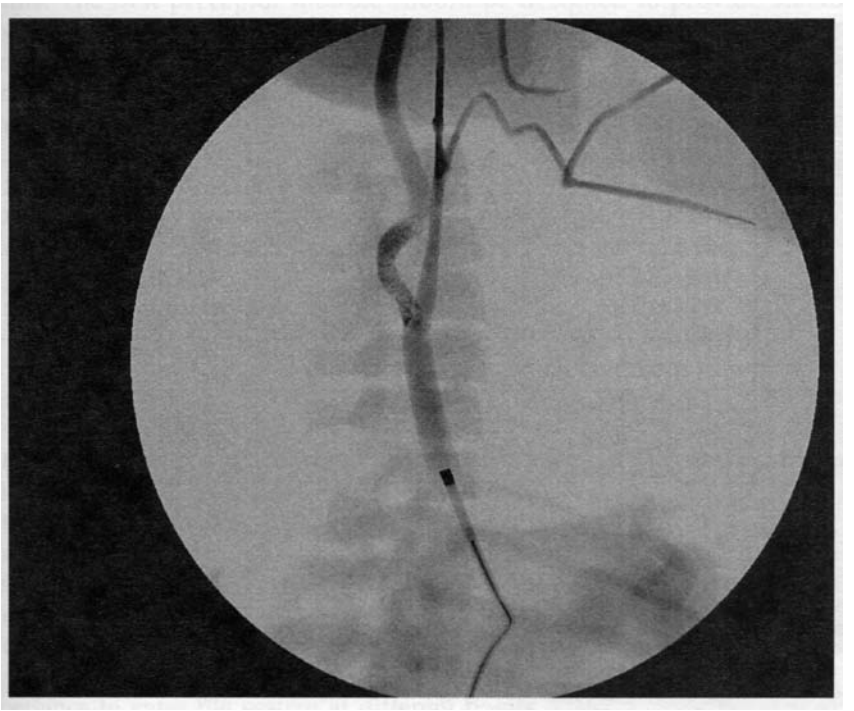

Figure 6-2. The Procedicus VIST® Virtual Reality Carotid Angiogram (Courtesy of David M. Hananel, Director, Surgical Programs, Medical Education Technologies, Inc.).

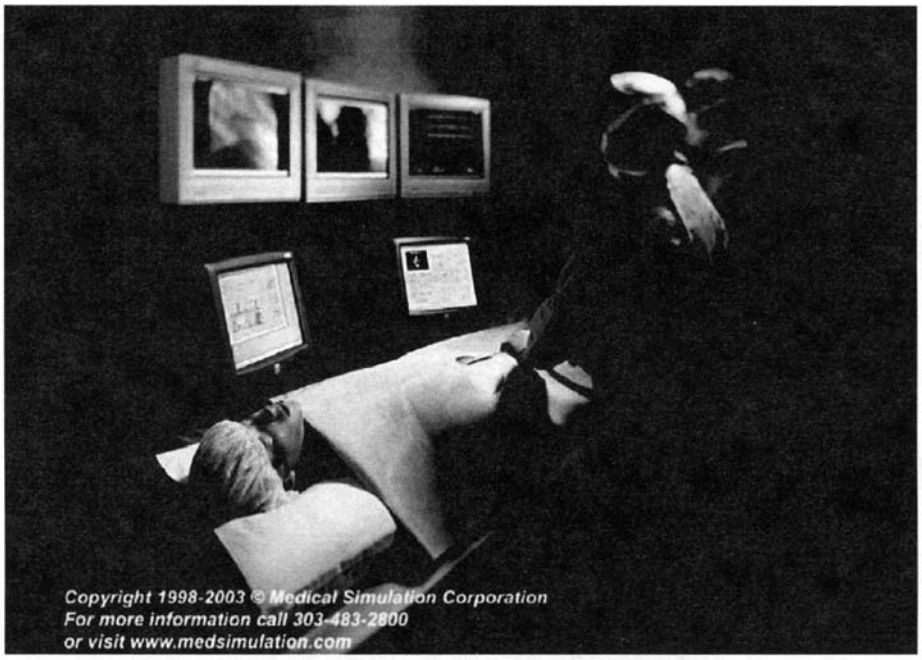

Figure 6-3. The SimSuite® System from Medical Simulation Corporation, Denver, Colorado (Courtesy Samuel Pepper, Clinical Director, Medical Simulation Corporation).

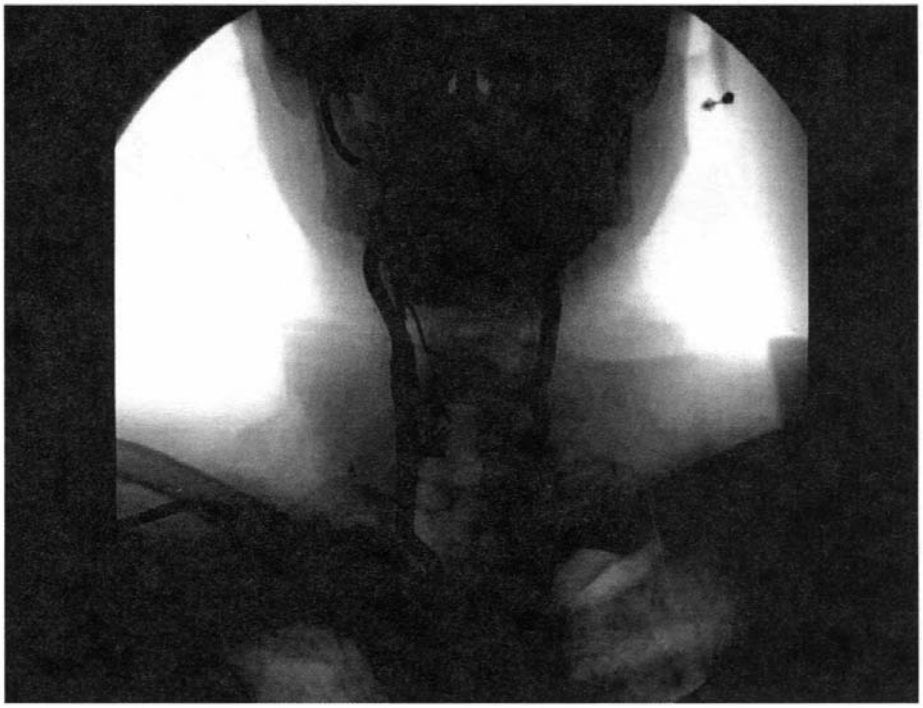

Figure 6-4. The SimSuite® Virtual Reality Carotid Angiogram (Courtesy Samuel Pepper, Clinical Director, Medical Simulation Corporation).

The main limitations to use of endovascular VR simulators arise out of their cost. Animal courses can be conducted at costs in the area of $12,000 per course for courses for 6 to 8 participants. Therefore, for training centers unable to afford simulators, animal courses remain a viable mechanism for instructing trainees in basic skills in order to maximize their clinical involvement in the preceptor module. Eventually it would be preferable for all basic training sites to evolve to use of the simulators.

MODULAR ENDOVASCULAR TRAINING

Once a $2\frac{1}{2}$-day basic introductory course has been successfully completed, at a recognized training center, the trainee will then elect three modular clinical preceptorships over the course of the next year. Each preceptorship will be one week in duration with the preceptor performing endovascular cases as the first assistant and then as the primary endovascular surgeon. A course director will integrate the system so that the clinical preceptor is assured a balanced and progressive experience over the three weeks. Clinical preceptor sites will be selected based on the quality of the endovascular experience assured the preceptor at the particular training site, site interest in the overall program and its mission, and clinical case load. Each training site should be able to produce at least 15-20 endovascular procedures for the preceptor. After attending three such weeklong preceptorships the aspiring endovascular surgeon will have accumulated 45 to 60 proctored endovascular procedures. This is more than sufficient for the average vascular surgeon with standard eye-hand coordination and psychomotor skills. The first preceptor module should be designed to provide standard or "introductory" caseload experience including selective diagnostic arteriography with traversal of the aortic bifurcation for contralateral selective lower extremity angiography, trans-brachial artery access for aortography, aortoiliac PTA and stenting, venous thrombolytic cases, IVC filter placement, use of IVUS for stent deployment assessment, etc. In this manner the preceptor may then go out and cherry pick standard cases in her/his own practice during the interval while awaiting attendance of his/her next preceptorship. A vascular surgeon in a standard practice would be expected to be able to accumulate 20-30 such hand picked cases during the first year (most of these cases probably need to be appropriate for performance in an O.R. with an O.E.C. c-arm since many preceptors will not have angio or cath lab privileges yet). Thus at the end of the year the average endovascular preceptor trainee should have accumulated 70-80 cases. The subsequent two preceptorships could be selected based upon the preceptor's interest. For example if the preceptor has interests in carotid or renal angiography site selection where intense work in these areas could be provided would be arranged.

This modular preceptor program should be far more user friendly than the three-month mini-fellowship program which most vascular practitioners simply cannot pursue. As virtual reality endovascular simulator case file libraries enlarge and as more training centers are able to incorporate these training tools in their curricula the actual time for even these preceptorships should be reducible and virtual reality experience should count towards credentialing and privileging.

To recapitulate an earlier statement in this chapter, the modular nature of the training paradigm proposed here should allow vascular surgeons with varying levels of experience to enter the system at different points. There may be the surgeon who has had sufficient guidewire and catheter skill set acquisition that he/she may not require the basics course but may be able to step into that first preceptor week. The system will be

all the more efficient if, at the beginning of that preceptor week, the trainee spends a day on a simulator performing virtual reality cases that will subsequently be performed that week in the peripheral angio suite.

NATIONAL AND REGIONAL SOCIETY SUPPORT

This modular training paradigm will work. It is time efficient. It recognizes the fact that the training requirements of an established vascular surgeon do not require parity with numbers required of other specialists. The use of virtual reality endovascular simulators will enhance skill acquisition and therefore improve patient safety. However, the individual surgeon seeking such training will continue to meet resistance in their local environment as they apply for hospital privileges in selected sites such as the cath lab and angio suites. Currently the vascular societies have tended to consider all politics local and there has been a paucity of help for the endovascular initiate in many of these local "turf" battles. In fact, if anything the recommendations from the societies have been obstructive to endovascular integration in the average vascular surgeon's practice. We must accelerate training, we must facilitate surgeon's access to a nationwide training venue, and we must assist the endovascular initiate as he/she embarks on the integration of endovascular procedures into their existing practices. This last issue may require use of legal recourse supported by vascular societal activities. It may require the aid of current endovascular specialists to visit other sites and offer credentialing committee testimony. The cause should certainly be supported by credible position papers published in our own journals.

It is time for a training paradigm shift and without such a shift vascular surgeons will soon find themselves irrelevant and our specialty a thing of the past and that would be detrimental to the care of patients with vascular disease.

SUGGESTED READING

Brener BJ. Regeneration. *J Vasc Surg.* 2002;36:1097–1103.

Moore WS, Clagett PG, Veith FJ et al. Guidelines for Hospital Privileges in Vascular Surgery: An Update by an Ad Hoc Committee of the American Association for Vascular Surgery and the Society of Vascular Surgery. *J Vasc Surg.* 2002;36:1276–1282.

Oblath RW. Changing Political Realities and Vascular Surgery. *J Vasc Surg.* 2002;36(2):414–415.

Ouriel K, Kent KC. The Role of the Vascular Surgeon in Endovascular Procedures. *J Vasc Surg.* 2001;33:902–903.

Ricotta JJ. Presidential Address: On Old Dogs and New Tricks. *J Vasc Surg.* 2002;36:657–662.

Satava RM. Surgical Education and Surgical Simulation. *World J Surg.* 2001;25:1489–1489.

Seymour NE, Gallagher AG, Roman SA et al. Virtual Reality Training Improves Operating Room Performance. *Ann Surg.* 2002;236 (4):458–464.

Strom P, Kjellin A, Hedman L et al. Validation and learning in the Procedicus KSA Virtual Reality Surgical Simulator. *Surg Endosc.* 2003;17:227–231.

Wieslander CK, Huang CC, Omura MC et al. Endovascular Workforce for Peripheral Vascular Disease: Current and Future Needs. *J Vasc Surg.* 2002;35:1218–1225.

SECTION II

Cerebrovascular Techniques

7

Initiating a Program in Carotid Stenting

Peter A. Schneider, M.D., and Michael B. Silva, Jr., M.D.

WHAT IS THE RATIONALE FOR A CAROTID STENT PROGRAM?

The primary reason to initiate a carotid stent program is to ensure quality patient care while expanding the therapeutic options for the treatment of carotid occlusive disease. The management of carotid bifurcation stenoses is undergoing dramatic changes. Vascular surgeons have traditionally played a leadership role in providing all aspects of medical and surgical treatment for carotid disease. Over the past five decades, vascular surgeons have developed, refined and taught skills that have permitted them to take clinical responsibility for patients with carotid artery occlusive disease and to provide these patients with excellent outcomes for a complex disease state. Carotid angioplasty and stent placement (CAS) will irrevocably change the management of carotid disease, perhaps to a greater extent than endovascular intervention has altered the management of vascular disease in any other arterial bed. Vascular surgical specialists must adapt to this change while continuing to be advocates for the highest quality vascular care. Although CAS remains investigational, clinical experience has accumulated which indicates that CAS can be a safe and effective treatment for carotid arterial stenosis.[1-6] As technology improves, so will device miniaturization, the functional properties of stents, and the efficacy of distal protection devices. As clinical experience increases, the overall results and outcome of CAS procedures are likely to improve.

Vascular specialists possess varying degrees of preparedness for the near future. The development of CAS has challenged vascular surgeons to attain additional skills and to become organized in ways that many would not have anticipated. Nevertheless, participation in the care of carotid occlusive disease in the future is dependent upon a proactive commitment now to gain these skills and develop strategies to utilize them effectively. The institution of a successful CAS program requires a thoughtful, measured and pragmatic approach. In this manuscript the authors review their rational for, and experience with, development of CAS programs in contemporary vascular surgical practices.

It has never been logical for accomplished vascular specialists who manage a disease process to refer patients to technique-based specialists for treatment. This paradigm, once widely accepted, involved a profound discontinuity of care that is currently untenable. Vascular surgeons who choose not to institute a CAS program are faced with the potential of relinquishing their role in the treatment of carotid disease to other disciplines that have less experience in this area and may not be clinically specialized in the care of vascular patients. Developing a programmatic approach to the contemporary treatment of carotid occlusive disease involves performing and evaluating new technology while maintaining safe, appropriate, streamlined and continuous care for our vascular patients.

WHY IS CAROTID STENTING DIFFERENT THAN OTHER PROCEDURES?

Endovascular technology has changed the care of vascular disease for the better, providing new and diverse options for treating sick patients. So, why is carotid stenting different from other procedures that have been introduced and embraced in the past few years?

1. For some the answer is that endarterectomy works well and there is no compelling imperative for an alternative treatment. Carotid endarterectomy is an index vascular procedure. It is the most commonly performed vascular operation, and for many years it has elegantly defined the effectiveness of vascular surgical intervention. It is the single most durable operation in our armamentarium, either open or endovascular. There is hard-earned Level One evidence regarding its efficacy and vascular specialists have steadily refined this procedure to minimize its cost, complications and length of stay.
2. Many vascular surgeons familiar with the physical properties of arterial plaque have been skeptical that CAS could be performed safely. For many the first-hand knowledge of plaque morphology and embolic potential has been difficult to reconcile with the accumulating data demonstrating improved safety and infrequent embolization with CAS. This knowledge, combined with the proven success of carotid endarterectomy, created an ethical dilemma for those who were reluctant to adopt what they believed to be an intuitively flawed therapeutic alternative.
3. The cerebral blood supply is the last vascular bed to be treated with endovascular intervention. The brain is a more forgiving end-organ than many might have imagined, but it is less forgiving than the other commonly treated vascular beds. CAS remains investigational and the potential for a devastating adverse outcome in the brain is real.
4. Vascular surgeons possess varying levels of skill in carotid and cerebral arteriography and carotid stenting. There is no open component to carotid stenting as there was during the introduction of endovascular stent-grafts for abdominal aortic aneurysm exclusion.

Despite these reservations, vascular surgeons have matured in their approach to the adoption of CAS and there is a growing if not universal consensus that direct involvement as a specialty will be required to clearly and impartially define the role of CAS in the treatment of carotid disease. The addition of this therapeutic alternative to the contemporary vascular practice by development of an institutional CAS program, however, has required significant planning and organization prior to implementation.

TABLE 7-1. COMPONENTS OF A CAROTID STENT PROGRAM

- Assess available knowledge and skills
- Obtain specialized training in carotid arteriography, if necessary
- Establish credentials for carotid arteriography
- Obtain specialized training in carotid stent placement
  - Improve fund of knowledge from printed material, computer programs
  - Didactic courses, Lab courses
  - Practice using simulators
  - Participation in live cases, either in home institution or as a visitor
- Establish credentials for carotid angioplasty and stent placement
- Obtain appropriate endovascular inventory to perform carotid stent placement and manage potential complications
- Develop a protocol for patient selection, performance of the procedure, and patient management and follow-up
- Seek dispassionate oversight: Institutional Review Board, participation in an FDA approved trial IDE
- Create a system of proctoring the first cases
- Establish a mechanism for quality assurance and ongoing education
- Develop methods for introduction of new and developing technologies
- Formalize an ongoing assessment of results

WHAT SHOULD BE INCLUDED IN DEVELOPING A CAROTID STENT PROGRAM?

The potential benefits of a well planned carotid stent program are numerous and include: a shortened learning curve; enhanced patient safety; efficient accumulation of experience from documentation and critical review of complications; uniform and coordinated skill development amongst practitioners; and development of a structured environment for education of staff and colleagues. Experience gained from the introduction of comprehensive programs for aortic stent-grafting has been of value in crafting carotid stent programs. Table 7–1 contains a list of components the authors consider necessary for CAS program development. Various components will require differing degrees of development from one institution to the next, depending upon the strength of the established vascular and endovascular practice. Following is a more detailed discussion of the process of CAS program development.

WHO SHOULD ADVISE AND TREAT PATIENTS WITH CAROTID OCCLUSIVE DISEASE?

Vascular surgeons are best suited to advise patients on the clinical alternatives for carotid disease and provide any necessary treatment. There has been an unsettling level of uncertainty in the vascular surgical community regarding our role and our future, and nowhere is this more evident than in the contemporary management of carotid occlusive disease. Vascular surgeons have a responsibility to offer their patients all available therapeutic alternatives for the management of carotid artery disease. Abdication of this responsibility by failing to incorporate the necessary techniques of CAS into one's practice is antithetical

to the traditional role of the vascular surgeon as a complete provider of vascular care. This issue is a focused vignette of the more important broader issue facing vascular surgery today: can we adapt and remain the full-service vascular specialists we have always been, or as our colleagues in other specialties have suggested, is it naïve to think we can continue to manage this increasingly complex field?

The goal of clinical care is a healthy patient with an improved outlook, not the performance of any given procedure. Focusing exclusively on a single technique, whether it is carotid angioplasty and stenting or carotid endarterectomy, limits the effectiveness of the practitioner and poorly serves the patient. New procedures should only be adopted if they are effective and safe and they are only of value when they are offered to the appropriately selected patients. This happens best when the focus of clinical care is upon the patient and the vascular system to be treated and new procedures are delivered in the context of a spectrum of treatment options.

Vascular specialists should make the clinical and treatment decisions regarding vascular patient care. The physicians making the decisions should be the ones who know the patients and the disease process best *and* who offer the widest spectrum of therapeutic alternatives. In the developing era of comprehensive disease management, reliance upon vascular surgeons in this role makes good sense and we should be less reluctant to make this argument.

Excellent results in vascular work are generated by:

1. Implementation of care decisions using an evidence based approach;
2. Appropriate patient selection;
3. Long-term experience with and understanding of the natural history of the disease process;
4. Intimate understanding of the underlying pathology and pathophysiology of the lesion and the disease;
5. Technical skill to perform all modes of therapy, including; medical, endovascular and surgical management;
6. Careful planning to introduce new treatments;
7. Safe peri-procedural management;
8. Understanding long-term outcomes and what they mean in the larger context of the disease process;
9. Commitment to long-term follow-up.

The management of extracranial carotid occlusive disease has been a primary focus of the field of vascular surgery. This includes; consultation, evaluation, medical, endovascular, and surgical treatment, pre-, intra- and post-procedure management, clinical and noninvasive follow-up, and surveillance. Vascular surgeons take personal responsibility for vascular patients and the long-term management of blood vessel problems. If vascular surgeons were to relinquish the care of patients to a non-clinician or an alternative non-vascular specialist for a small part of their care, this would represent a significant backward leap. In the current era of disease management approaches to complex medical problems, this is counterintuitive. In the future of medicine, it will make even less sense. The provision of seamless care for a complex process should not be interrupted by dividing care among multiple disciplines. It is not reasonable to assume that technical expertise alone with a single procedure is a satisfactory replacement for an overall approach to a clinical problem or would be appropriate cause for the vascular clinician to relinquish care of the patient.

TRAINING

By "Training" we refer to the accumulation of experience and development of specific skills that prepares the operator to perform carotid angioplasty and stent placement. This experience comprises the basis by which the operator may qualify for privileges at a specific institution and subsequently offer this procedure to patients. Carotid angioplasty and stenting has a learning curve associated with it, as does every other procedure. Most current fellowships are not able to provide substantive training in carotid angioplasty and stenting since it is not yet an approved procedure and has not been widely adopted. This dearth of experience available during fellowships is presently true for vascular surgery, cardiology, neuroradiology, neurointerventional radiology, and interventional radiology, unless the institution is one of a few enrolling patients in a clinical trial of carotid stenting. Even in these settings, the newness of CAS procedures and the intolerance for complications often precludes extensive hands-on experience for physicians in training. Few vascular specialists in current practice have a comprehensive experience with carotid stenting, even among those who have been recently trained. Therefore, the necessary components for training in this area are an important issue for all participants. Training to perform carotid bifurcation angioplasty and stenting is dependent upon five general areas.

1. Understanding the behavior of atherosclerotic occlusive lesions of the extracranial carotid arteries. Vascular surgeons have extensive experience with this.
2. Understanding the management of carotid artery occlusive disease, including natural history and all treatment options. This has traditionally been the role of the vascular surgeon.
3. Experience with arch anatomy and selective catheterization of arch branches. The basis of this comes from carotid arteriography. This enables placement of an access sheath in the carotid artery for angioplasty and stenting. Many vascular surgeons do not have extensive experience with the performance of carotid arteriography or transfemoral catheterization of the supra-aortic branch vasculature. This experience could be gained by performing arteriography prior to carotid endarterectomy in patients in whom duplex alone is not adequate. Carotid arteriography may also be performed as a completion study after carotid endarterectomy. Credentials for performing carotid arteriography are discussed in the next section.
4. Experience with angioplasty and stenting of other non-coronary arteries. Transferring skills from other endovascular procedures shortens the learning curve significantly. Specialists in other disciplines have transferred developed endovascular skills to the extracranial cerebrovasculature. Experience with endovascular procedures in other vascular beds is important since the stakes are high with carotid angioplasty and stenting. Juxtaposed against this is the fact that after a sheath is placed in the carotid artery, treatment of the carotid lesion requires crossing and treating a focal lesion.
5. Ability to evaluate cerebral runoff and use of a distal protection device. Interpretation of intracranial arteriography has been an important component of vascular surgery training programs and deserves even greater emphasis. Available data suggests that use of distal protection devices may decrease the incidence of peri-procedural stroke associated with CAS. It is likely that these devices will continue to evolve over the next few years. The use of cerebral rescue techniques are not common and the anecdotal results reported have been

inconclusive. Nevertheless, each CAS program plan should include a protocol for use in the event of a peri-procedural stent thrombosis or cerebral embolus.

These general areas that comprise training should be considered when planning a program. Most operators who are performing carotid stent placement currently have used several approaches to learning carotid angioplasty and stenting, including: learning from colleagues; attending courses and meetings; and gaining practical experience in carefully selected patients. It helps to write a plan for starting a program and analyze what is needed to accomplish this in one's individual institution. Physician proctoring, careful record keeping and case follow-up, and a quality assurance plan are complementary to the training process.

At present, training for vascular specialists who already have essential knowledge of carotid disease and all other methods of management comes down to performing carotid arteriography, learning about and observing carotid stent cases and performing them in appropriately selected patients. In addition, any carotid stent and distal protection device approved by the FDA is likely to have formal training requirements mandated in association with its sale and use.

HOW DOES A VASCULAR SURGEON GET THE PRIVILEGE TO PERFORM CAROTID ARTERIOGRAPHY AND INTERVENTIONS, AND HOW MANY PROCEDURES ARE ENOUGH?

The role of carotid arteriography in the management of carotid occlusive disease has changed significantly over the years. Until the late 1980s, carotid arteriography was routinely employed to determine the degree of stenosis and to evaluate the surrounding anatomy. As better and less invasive tests for this task were developed, many vascular surgeons adopted the practice of performing treatment for most patients based upon noninvasive tests, with carotid arteriography reserved for those with suspected intracranial or arch disease or other anatomical challenges. In the era of carotid stent placement, carotid arteriography has made a clinical resurgence, not only to determine the degree of stenosis and associated anatomy, but as a pathway to treatment. Carotid arteriography is the best method at present for determining whether or not a patient may be treated with a carotid stent. This presents vascular surgeons with a dilemma since many have worked to produce noninvasive studies that provide safe and excellent results. Nevertheless, the general reintroduction of carotid arteriography in the management of carotid occlusive disease appears inevitable and will serve as a gateway to future therapy.

Privileges to perform carotid arteriography and/or carotid angioplasty and stenting are granted to the individual practitioner through the Credentials Committee of each individual institution. Credentials Committees take into account multiple factors, including; published national standards, local practice patterns/standard of care, documentation of training and experience by the practitioner, institutional politics, and other things. It is the practitioner's responsibility to present pertinent credentials to such a body and to be able to provide safe care for their patients. It is the institution's responsibility to ensure that its standards ensure safe patient care and that its physicians are appropriately qualified. Credentials for carotid arteriography differ fundamentally from those applied to carotid stent placement since carotid arteriography is an established procedure with a long-term track record and frequent usage in practice. Some institutions have specific case requirements that must be met while others may

TABLE 7-2. QUALITY IMPROVEMENT GUIDELINES FOR ADULT DIAGNOSTIC NEUROANGIOGRAPHY*

Threshold for appropriate indications:	99%**
Threshold for success of the case:	98%***
Threshold for complications:	
Permanent neurological deficit:	1.0%
Reversible neurological deficit	2.5%
Systemic complication	< 1.0%
Puncture site/access complication	< 1.0%

**Am J Neuroradiol.* 2000;21:146:150.
**Includes evaluation of extracranial cerebrovascular disease.
***Requisite information is obtained.

include carotid arteriography in combination with other types of arteriography as a general category. Most institutions include arteriography, at least as a general procedure type, somewhere in the list of commonly performed vascular procedures.

Carotid arteriography is performed by vascular surgeons, neurosurgeons, neuroradiologists, general radiologists, vascular medicine specialists, interventional radiologists, interventional neuroradiologists, and probably others. The acknowledgment that multiple specialties may be considered qualified to perform carotid arteriography is reflected in a guideline document for carotid arteriography, entitled 'Quality Improvement Guidelines for Adult Diagnostic Neuroangiography', published jointly by the American Society of Interventional and Therapeutic Neuroradiology (ASITN), the American Society of Neuroradiology (ASNR), and the Society of Cardiovascular and Interventional Radiology (SCVIR).[7] This document warrants analysis since these societies refer to the guideline as a credentialing instrument for cerebral angiography. This document does not specify case numbers to achieve privileges or to maintain competence in carotid arteriography. This jointly published document focuses upon results: acceptability of indications for carotid arteriography; success rates in performing carotid arteriography; and, complication rates, both neurologic and non-neurologic (Table 7–2). Vascular specialists are likely to agree with its principles and content: that indications for carotid arteriography should be appropriate and that the success of the procedure should be optimized while minimizing complications; and that carotid arteriography will be performed by multiple different specialties.

Carotid arteriography has never been the exclusive domain of any single discipline. The major societies (those mentioned above and others), governing bodies (including federal and state government licensing agencies and medical societies), and institutions (insurance companies, hospitals) acknowledge that multiple different specialties perform carotid arteriography. This approach relies on the concept that specialists perform a variety of tasks in daily practice and those performing carotid arteriography possess transferable skills, such as other types of arteriograms and endovascular procedures, which help in achieving desired results in carotid arteriography. Each practitioner must inquire at his or her own institution of practice as to whether there are specific requirements or case numbers for carotid arteriography.

In addition, there are no published standards for the number of carotid arteriograms that must be performed during any fellowship or residency program to achieve competence in carotid arteriography. Fellowship requirements in interventional neuroradiology, for example, include a total of 100 cases and 12 months of training involving the treatment of brain aneurysms, AV malformations, tumors, intracranial vascular

disease, trauma, and maxillofacial anomalies.[8] There is no specific case requirement for carotid arteriography.

There are no formal, external or generally accepted training requirements for carotid stent placement as of yet, but it is likely that future standards will require a specific number of cases to consider a practitioner qualified to perform CAS. Most institutions currently do not have set requirements for privileges to perform carotid angioplasty and stent placement, as CAS remains investigational. Standards will be developed as the procedure becomes more widely accepted and as various disciplines vie for the privilege of caring for these patients. Credentials for carotid interventions should be based on the following;

1. Identification of a reasonable external standard.
2. Demonstration of ability to transfer existing clinical and technical skills.
3. Documentation of additional training.
4. A plan for patient safety.

After credentialing standards for noncoronary interventions became a contested issue among specialists of different disciplines in the late 1980s, national organizations representing vascular surgeons, interventional radiologists, and cardiologists published case requirements that serve as guidelines to acquire privileges to perform endovascular interventions. Credentialing standards for endovascular procedures are listed in Table 7–3. Because there are overlapping skills between several specialties that might perform endovascular procedures, case numbers are used as an equalizer, since the background and training of the different specialists involved in endovascular procedures may vary significantly. The success of these efforts in identifying a single standard for use among specialties of varying educational backgrounds has been limited, and the process still engenders controversy.

Although there is substantial diversity among the represented organizations, the recommended standards are similar.[9-12] The highest number of angiograms required by any criteria is 200 and the highest number of interventions required is 50. These credentialing guidelines treat all noncoronary vascular beds in the same manner. There is no specific differentiation between the aortoiliac, renal, carotid, tibial vasculature, etc., and there are no additional qualifications required for angiography and interventions in various vascular beds. There are no specific national or societal guidelines that pertain to carotid angioplasty and stenting and no published guidelines or standards to separate the extracranial carotid arteries from the rest of the noncoronary arteries in terms of case requirements for credentialing in angiography or interventions, such as stents.

Among the many ongoing carotid stent trials in the US, some did not have a specific case requirement for trialists to enter the study. Most of the trials that had

TABLE 7-3. CREDENTIALING STANDARDS FOR ENDOVASCULAR PROCEDURES OF THE NONCORONARY ARTERIES

Organization	Ref.	#Angiograms	#Interventions
Society of Interventional Radiology	9	200	25
American Heart Association	10	100(50)	50(25)
American College of Cardiology	11	100(50)	50(25)
Society for Vascular Surgery	12	100	50

Numbers in parentheses indicate the number required as primary operator

specific case requirements set the number at 10 to 20 cases each. This case threshold is not a prerequisite to perform carotid stent placement, but is a threshold for demonstrating the highest level of technical and clinical expertise consistent with the designation as an expert clinical trialist. The only NIH sponsored trial, the CREST Trial (Carotid Revascularization Endarterectomy versus Stent Trial), requires 20 carotid stent cases for an operator to demonstrate special expertise in this area and be admitted as a trial participant.[13] The CREST Trial partcipant applicants included 103 physicians from 70 centers.[14] The mean number of carotid stent cases performed by each operator at the time of application was 13. The rate of perioperative stroke was lower after procedures performed by physicians who had performed 15 or more symptomatic carotid bifurcation cases (3.7%) than among those with fewer than 15 cases (7.1%).

Nationwide, carotid stent placement is being performed by vascular surgeons, cardiologists, interventional radiologists, neurosurgeons, and neurointerventional radiologists. All of these specialties are participating in trials. Numerically, the largest physician-participant group is cardiology. This is a group that traditionally has had less endovascular experience in the noncoronary vasculature and the cerebral circulation. Vascular surgeons have taken a role in the development of carotid stent placement as a treatment option. Two of the largest carotid stent trials, and the only non-industry-sponsored trials (CREST and CARESS) are headed by vascular surgeons. Most of the trials have vascular surgeons included as trialists. In addition, no trial requires the presence of experts in 'neuro rescue' techniques. The NIH sponsored CREST Trial does not recognize 'neuro rescue' as a requirement or prerequisite to perform carotid stent placement.

The introduction process for carotid stent placement should emphasize patient safety, careful patient selection, evidence based implementation and protocols for follow-up. It should be the goal of vascular specialists nationwide and in their specific institutions of practice to create the highest possible standards and provide the best possible care of patients based on those high standards. The likelihood is that each institution will develop case requirements for carotid stenting based upon national guidelines and other factors. Carotid stent placement should be listed on the credentials and privileges for your vascular department and should be specifically approved as a privilege by the credentials committee.

MUST "NEURO RESCUE" BE PART OF THE PROGRAM?

There is no definitive answer for the above question. However, the information presented here may be helpful in planning a carotid stent program. We believe that catheter directed intracranial thrombolysis will likely prove to be of value at some point in the future and that the more skills and options available during the management of these complex patients, the better. However, the use of intracranial, catheter directed thrombolysis is not an evidence-based procedure for de novo stroke treatment and is of unknown efficacy in patients who sustain a neurological deficit during carotid stent placement. The concept of 'neuro rescue' is that if embolization should occur during manipulation of the extracranial carotid bifurcation, a catheter could be advanced into the cerebral arteries to administer thrombolytic agents to dissolve clot and reverse brain damage. This is an attractive concept but it is not the standard of care and its validity is unproven.

1. The idea that 'neuro rescue' could be used to treat stroke during carotid stent placement is extrapolated from studies suggesting that the acute treatment of new stroke with urgent intracranial thrombolytic therapy provides improved outcomes. Unfortunately, de novo acute stroke may not be analogous to stroke associated with carotid stent placement, resulting from embolization of atheromatous debris from the more proximal carotid artery.
2. The best evidence of efficacy for intra-arterial thrombolytic treatment of acute stroke is PROACT II, a prospective, randomized comparison of catheter-directed intracranial pro-urokinase, which is not commercially available, versus systemic intravenous heparin (not intravenous thrombolytics).[15] There was a 15% absolute benefit in neurological status in the thrombolytic group at 90 days. However, the rate of intracranial hemorrhage was 28% after intracranial thrombolysis and 6% with heparin and mortality in both groups was the same at 90 days.
3. The only FDA approved protocol for the use of thrombolytic agents to treat acute stroke is for systemic TPA (tissue plasminogen activator) to be administered intravenously within three hours of acute, de novo stroke.[16]
4. There has been no prospective, randomized comparison of intravenous thrombolytic versus catheter-directed, intra-arterial thrombolysis in acute stroke. The nationwide standard for the treatment of acute stroke is IV TPA, not a catheter-directed, intracranial approach.
5. There is no agent approved by the FDA for catheter-directed intracranial thrombolysis. There is no standard protocol, standard of practice or national credentialing standards for catheter-directed intracranial thrombolysis.
6. The ASITN is supporting catheter-directed intracranial stroke therapy and has issued a Standards of Practice paper in which the evidence for intracranial thrombolytic therapy of stroke is reviewed.[17] In encouraging members to vary from the current IV TPA standard, the guidelines states the following. "Intra-arterial fibrinolytic therapy administered within the 3 hour time limit for approved use of IV TPA should not be considered to be unethical." Although intracranial thrombolysis may not be considered unethical by ASITN, the proof of its safety and efficacy is poor and further work is required before it could be considered as a legitimate treatment option.[18]
7. When embolization does occur during carotid stent placement, the particles released are a combination of cholesterol crystals and lipoid masses from atherosclerotic carotid bifurcation plaque, neither of which dissolves or can be removed by thrombolytic therapy.[19-21]
8. Not much is known about neuro-rescue during carotid stenting. Factors remaining poorly defined include: indications, timing, agent of choice, protocol for use, and results and risks of treatment. Most of the neurological deficits which have occurred as a result of carotid stent placement in published series have been minor and the patients would not have been candidates for this treatment.[22-24]
9. Carotid stent placement is being considered as an alternative to carotid endarterectomy and there are several ways in which the procedures are analogous. However, neurologic deficits that occur as a result of stent placement are not the same as those that occur with carotid surgery.[25] Rather than an immediate and intra-procedural event, a substantial number of the peri-procedural events which occur with CAS occur hours to days after the procedure.[22,24,26] In one study, 26% of the peri-procedural neurologic events occurred more than one day (and up to 14 days) after the procedure (and after patient discharge).[22]

In another study, 71% of the peri-procedural deficits (10 of 14) after carotid stent placement in 111 patients, occurred after the procedure was completed, rather than during the procedure.[26] This presents logistical challenges if intracranial thrombolysis ever becomes the standard method of managing this problem, since it often occurs after catheters and intra-arterial access devices have been removed and in some cases the patient may already be discharged. The patient would have to return and be treated in a timely manner. The area where the carotid stent was placed would require repeat instrumentation (crossing with guidewires and catheters), with the attendant added risk of additional embolization.

10. What are the results of 'neuro rescue' after carotid stent placement? Anecdotal case reports and small series of 'neuro rescue' after carotid stent placement are available but no higher level evidence of safety or efficacy exist. Unfortunately, the published results of 'neuro rescue' have been poor. In the largest study to date, 27 of 450 patients undergoing carotid stent placement developed neurologic deficit (6%).[22] Twenty-two neurologic deficits resolved or nearly resolved on medical management alone and 5 patients were treated with intracranial thrombolysis. Three of these patients died of stroke (60% mortality) and two experienced some improvement but neurologic deficits persisted. These results cannot be the basis for a rational standard.
11. Intracranial thrombolytic therapy has its own high morbidity. In the PROACT II study, the intracranial hemorrhage rate was 28%.[15] Patients underwent CT brain imaging prior to treatment to help avoid the potential disaster of administering thrombolytic treatment to a patient with an underlying intracranial hemorrhage. In the setting of an acute, intra-procedural, neurological event, it may not be logistically feasible to first perform axial brain imaging to rule out intracranial hemorrhage, making the risk of intracranial thrombolytic therapy in this setting even higher. If this therapy ever becomes a standard, it will be essential to treat only those who need it since the risk of the proposed treatment itself is high.
12. A situation analogous to carotid stent placement is extracranial carotid artery repair by endarterectomy. Carotid endarterectomy occasionally results in intra-procedural embolization and stroke. When this occurs, it has not been standard practice to treat with intracranial thrombolytic agents. More commonly adopted practices include administering anticoagulants, managing the sequel of stroke, and establishing the integrity of the extracranial carotid artery repair.[27]

These factors help to explain why none of the dozen and a half ongoing carotid stent trials, including the one sponsored by the NIH, includes 'neuro rescue' as part of the protocol. Although this technique has potential theoretical advantages, it is far from a standard of care and may turn out to have worse results than other options.

WHAT ARE THE ROLES OF PROTOCOLS, PROCTORING AND QUALITY ASSURANCE?

A protocol for carotid stent placement should be comprehensive and detailed. Some factors to include are: an outline of the training guidelines; a description of the procedure and peri-operative management; a list of required inventory; requirements to consider in case

selection; a clinical and duplex follow-up protocol; and possibly mock procedures for staff preparation. The more these factors can be considered and decided upon ahead of time, the better. The Institutional Review Board will likely require this material for evaluation. The earliest cases should be those with straight forward access, minimal tortuosity, and a focal lesion. Staff preparation will allow the procedure to proceed more efficiently.

There is no national standard for proctoring prior to performing a new procedure independently. There is no set requirement for what is included in proctoring a case. Proctoring could include having the proctor assist in the case or observe the case and may include some pre- or post-procedure evaluation of the patient, the chart or both. However it is done, it should be established ahead of time. Proctoring adds an extra safety mechanism for the patients and the physician as a new procedure is being introduced. A physician may be proctored at another center with expertise in carotid stenting where the proctored physician acquires privileges and performs the case. A physician may also be proctored at his or her own institution by an invited proctor. In initiating a CAS program one should make the process as deliberate and standardized as possible. If the home institution has a proctoring protocol and written form, use it. If it does not, establish one. A simple, one page form should summarize the case and its management. Consider adding proctoring guidelines to the requirements for acquiring privileges in this area.

Any new procedure must include a quality assurance mechanism. Most clinical departments already have some type of quality assurance program that includes recording and discussing complications and looking for opportunities to improve results. New procedures should receive special scrutiny within the department, especially when on the early phase of the learning curve. Consider discussing every case, whether complicated or not, for the first dozen procedures.

Since carotid angioplasty and stent placement is investigational, Institutional Review Board (IRB) approval should be obtained. The Institutional Review Board provides additional patient protection and another level of quality assurance. Inquire about this with the local IRB as to what the requirements are for initiating an application. An IRB-approved protocol usually requires a stated plan, specific indications, special consent processes, safeguards on confidentiality and interim reports.

DEALING WITH ENDOVASCULAR REALITIES

Before a carotid stent program can be initiated, there are multiple issues that must be considered. (Table 7–4). The solutions to these issues can only be achieved in each institution and usually depend upon local politics and physician leadership by the vascular surgeon.

Patient selection is the key to early success. Patients who are good stent candidates due to high medical co-morbidities may not always have favorable anatomy for stent placement and they will likely have an elevated risk of peri-procedural complications, even from a percutaneous procedure. Some patients with complex anatomy cations, even from a percutaneous procedure. Some patients with complex anatomy but who are otherwise good candidates for CAS may have to be treated by alternative means if seen early in the program's development while the practitioner is accumulating experience. The best early candidates for CAS are patients with focal recurrent stenosis. In many institutions, vascular surgeons have restricted access to fixed unit fluoroscopic imaging equipment. This should not necessarily prevent initiation of a carotid stent program. Portable digital C-arm fluoroscopy is adequate for imaging of the neck and

TABLE 7-4. FACTORS TO CONSIDER BEFORE INITIATING THE CAROTID STENT PROGRAM (I.E., PRACTICAL CONCERNS AND ISSUES TO ADDRESS)

- Careful patient selection is the key to early success.
- Vascular surgeons need access to the best imaging equipment.
- Inventory must be studied and understood before proceeding.
- Hospital administrators should be informed and supportive.
- Person initiating carotid stent program is often the youngest/newest person in the group (with the least clout); senior membership and consolidated group support is key.
- Interdisciplinary issues can be contentious since multiple specialties plan to participate.
- Do you need 'neuro rescue' capability?
- Payment issues (lack of payment) must be considered in advance.
- Know what the standards are in your community and make sure they are met.
- Take every safety precaution possible on behalf of the patients.

skull. This process is facilitated by the concomitant use of a floating radiolucent table. Specific inventory for carotid arteriography, balloon angioplasty, and stent placement differs somewhat from that used in other vascular beds. The components of the carotid interventions inventory must be studied, understood, requested, and assembled before proceeding.

Hospital administrators should be informed about the plans to initiate a CAS program and it is best if they can be convinced to be supportive. Vascular surgeons should focus on the following in presentations to administrators: providing new options in patient care services; treating patients who are not otherwise candidates for intervention; and developing new areas of expertise. Payment issues must also be considered in advance and require consultation with the local Medicare carrier and insurance companies. CAS outside of an approved trial is not currently reimbursed under Medicare guidelines. Challenging issues arise with adoption of any innovative program. Support from the institution will be essential at some unanticipated point. The vascular surgeon taking the lead role in initiating carotid stent procedures is often the youngest or newest person in the group. Therefore, input and support from more senior partners is essential. In establishing a CAS program, existing standards in your community must be met or exceeded. For example, if stroke neurologists are involved in the care of patients requiring carotid endarterectomy, they should also participate when carotid stents are performed.

Interdisciplinary issues associated with CAS are complex and often overtly contentious. Specialists from disciplines that have not traditionally treated carotid artery disease have shown a keen interest in performing CAS procedures and to date have made important contributions to the advancement of this technique. Motivations are as varied as individuals participating, but a redrawing of traditional practice boundaries is underway and the potential for an overall reduction in quality of care for the vascular patient with carotid occlusive disease may be an unintended consequence.

We believe a program of CAS initiated by vascular surgeons and held to the same high standards of success as one's carotid endarterectomy practice affords the best possible chance for enhancing patient care and advancing the science of therapeutic intervention for carotid disease. The widespread development of CAS programs will represent a necessary evolution for our specialty and an affirmation of our vision of the vascular surgeon as the comprehensive vascular specialist. To fail in this endeavor would represent a significant abdication of the management of vascular disease by a specialty moving away from excellence and towards obsolescence.

REFERENCES

1. Brown MM. Carotid and vertebral artery transluminal angioplasty study (CAVATAS). *Lancet.* 2001;357:1729–1737.
2. Wholey MH, Wholey M, Mathias K et al. Global experience in cervical carotid artery stent placement. *Catheter Cardiovasc Interv.* 2000;50:168–169.
3. Alberts MJ. Results of a multicenter prospective randomized trial of carotid artery stenting versus carotid endarterectomy. *Stroke.* 2001;32:325.
4. Hobson RW, Goldstein RJ, Jamil Z et al. Carotid restenosis: Operative and endovascular management. *J Vasc Surg.* 1999;29:228–238.
5. Dietz A, Berkefeld J, Theron JG et al. Endovascular treatment of symptomatic carotid stenosis using stent placement: Long-term follow-up of patients with a balanced surgical risk/benefit ratio. *Stroke.* 2001;32:1855–1859.
6. Hobson RW. Update on the Carotid Revascularization Endarterectomy versus Stent Trial (CREST) protocol. *J Am Coll Surg.* 2002;194:S9–S14.
7. Cooperative study between ASITN, ASNR, and SCVIR. Quality improvement guidelines for adult diagnostic neuroangiography. *Am J Neuroradiol.* 2000;21:146–150.
8. Higashida RT, Hopkins LN, Bernstein A et al. Program requirements for residency/fellowship education in neuroendovascular surgery/interventional neuroradiology: A special report on graduate medical education. *Am J Neuroradiol.* 2000;21:1153–1159.
9. Spies JB, Bakal CW, Burke DR et al. Standards for interventional radiology. Standards of Practice Committee of the Society of Cardiovascular and Interventional Radiology. *J Vasc Interv Radiol.* 1991;2:59–65.
10. Levin DC, Becker GJ, Dorros G et al. Training standards for physicians performing peripheral angioplasty and other percutaneous peripheral vascular interventions. A statement for health professionals from the Special Writing Group of the Councils on Cardiovascular Radiology, Cardio-Thoracic and Vascular Surgery, and Clinical Cardiology, the American Heart Association. *Circulation.* 1992;86:1348–1350.
11. Spittell JA, Nanda NC, Creager MA et al. Recommendations for peripheral transluminal angioplasty: Training and facilities. American College of Cardiology Peripheral Vascular Disease Committee. *J Am Coll Cardiol.* 1993;21:546–548.
12. White RA, Hodgson KJ, Ahn SS et al. Endovascular interventions training and credentialing for vascular surgeons. *J Vasc Surg.* 1999;29:177–186.
13. Hobson RW. Update on the Carotid Revascularization Endarterectomy versus Stent Trial (CREST) protocol. *J Am Coll Surg.* 2002;194:S9–S14.
14. Al-Mubarak N, Roubin GS, Hobson RW et al. Credentialing of stent operators for the Carotid Revascularization Endarterectomy vs. Stenting Trial (CREST). *Stroke.* 2000;31:292.
15. Furlan A, Higashida R, Weschler L et al. Intra-arterial prourokinase for acute ischemic stroke: The PROACT II Study: A randomized controlled trial. *JAMA.* 1999;282:2003–2011.
16. The National Institute of Neurological Disorders and Stroke rt-PA Stroke Study Group. Tissue plasminogen activator for acute ischemic stroke. *N Engl J Med.* 1995;333:1581–1587.
17. ASITN. Standards of Practice. Intra-arterial thrombolysis. Available at: *www.asitn.org.*
18. Executive Committee of the ASITN. Intra-arterial thrombolysis: Ready for prime time? *Am J Neuroradiol.* 2001;22:55–58.
19. Martin JB, Pache JC, Treggiari-Venzi M et al. Role of distal balloon protection technique in the prevention of cerebral embolic events during carotid stent placement. *Stroke.* 2001;32:479–484.
20. Whitlow PL, Lylyk P, Londero H et al. Carotid artery stenting protected with an emboli containment system. *Stroke.* 2002;33:1308–1314.
21. Tubler T, Schluter M, Dirsch O et al. Balloon-protected carotid artery stenting: Relationship of periprocedural neurologic complications with the size of the particulate debris. *Circulation.* 2001;104:2791–2796.
22. Wholey MH, Wholey MH, Tan WA et al. Management of neurological complications of carotid artery stenting. *J Endovasc Ther.* 2001;8:341–353.

23. Vitek JJ, Roubin GS, Al-Mubarek N et al. Carotid artery stenting: Technical considerations. *Am J Neuroradiol.* 2000;21:1736–1743.
24. Criado FJ, Lingelbach JM, Ledesma DF et al. Carotid artery stenting in a vascular surgery practice. *J Vasc Surg.* 2002;35:430–434.
25. Sheehan MK, Baker WH, Litooy FN et al. Timing of post-carotid complications: A guide to safe discharge planning. *J Vasc Surg.* 2001;34:13–16.
26. Qureshi AI, Luft AR, Janardhan V et al. Identification of patients at risk for periprocedural neurological deficits associated with carotid angioplasty and stenting. *Stroke.* 2000;31: 376–382.
27. Rockman CB, Jacobowitz GR, Lamparello PJ et al. Immediate re-exploration for the perioperative neurologic event after carotid endarterectomy: Is it worthwhile? *J Vasc Surg.* 2000;32: 1062–1070.

8

The Role of Stents in Patients with Carotid Disease

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Since the first descriptions of carotid repair by Eastcott, Pickering, and Rob in 1953;[1] DeBakey in 1954;[2] and Carrea, Molins, and Murphy in 1955,[3] carotid endarterectomy has been interrogated through a wide array of clinical trials. The first prospective, randomized, multicenter comparison of carotid revascularization versus best medical management was completed over 40 years ago. This trial, known as the Joint Study of Extracranial Occlusion, enrolled more than 1200 patients and appeared in a series of publications in the late 1960s and early 1970s.[4,5] The findings were significant; surgical carotid repair was associated with significant clinical benefit when compared with medical management. While the study fell into disrepute in the 1980s and the frequency of carotid surgery began to decline,[6] subsequent, larger studies with improved trial design again documented benefit of endarterectomy over observation in patients with significant bifurcation stenoses.[7-9] Estimates range as high as almost 200,000 carotid endarterectomies performed annually in the United States alone.[10]

CAROTID ENDARTERECTOMY

Carotid endarterectomy is a well-proven intervention for carotid disease. The results of numerous clinical trials have documented its safety and efficacy and it remains the standard of care for patients with severely stenotic extracranial lesions, whether the patient is symptomatic or not.[9,11] Nevertheless, the excellent results achieved with the procedure appear to be dependent on patient-specific clinical variables such as gender,[12] symptoms,[13] and the status of the contralateral carotid vessel.[11] Relatively healthy patients do very well with open surgical repair of carotid lesions. The treatment of medically compromised patients, however, is associated with a much greater risk of complications, as illustrated in a review of over 3000 patients undergoing carotid endarterectomy at the Cleveland Clinic Foundation between 1988 and 1998.[14] In this analysis of a consecutive series of patients, the risk of the composite end point of stroke, myocardial infarction, or death was quite satisfactory in patients who did not manifest one of four classes of baseline comorbidity (coronary

artery disease requiring intervention, congestive heart failure, chronic lung disease, and renal insufficiency). The risk of perioperative morbidity and mortality was substantial, however, when patients exhibited one or more baseline comorbid conditions. Specifically, the risk of perioperative death was elevated by a factor of more than 5, stroke or myocardial infarction each by a factor of 2 and the composite end point of death, stroke, or myocardial infarction by a factor of almost 3.

Our interpretation of the Cleveland Clinic data is that, in most cases, carotid endarterectomy is a procedure with an extremely low rate of complications. In studies that specifically exclude high-risk patients from eligibility, for example, the North American Symptomatic Carotid Endarterectomy Trial (NASCET) and Asymptomatic Carotid Atherosclerosis Study (ACAS), the rate of periprocedural complications can be expected to be extremely low. A cavalier review of these trials may lead to the error of assuming that the results of carotid endarterectomy are analogous in the unselected patients undergoing carotid repair at a wide range of hospitals and by practitioners with a broad spectrum of experience. On the contrary, there exist data suggesting that the results of the trials cannot even be generalized to patients undergoing endarterectomy at the hospitals that participated in the studies. In a study of 113,000 Medicare patients who underwent carotid endarterectomy during patient acquisition for the NASCET and ACAS trials (1992-1993), Wennberg and colleagues noted that the perioperative mortality rate was 1.4% in hospitals participating in the trials and 1.7% in hospitals that did not participate in the trials.[15] The rate of perioperative death rose to 2.5% in low-volume nontrial hospitals where fewer than seven carotid endarterectomies were performed yearly. These relatively high complication rates are in direct contrast to the much lower mortality rates observed in the patients entered into the trials (0.1% in ACAS and 0.6% in NASCET). These findings suggest that eligibility criteria were sufficiently strict that patients in the NASCET and ACAS trials represented a small subset of the total population of patients undergoing carotid endarterectomy—a subgroup with the lowest frequency of baseline comorbid conditions and the lowest rate of perioperative adverse events.

The data from the Cleveland Clinic registry offer an explanation for the Wennberg findings. Patients in the multicenter trials of carotid endarterectomy were similar to the low-risk group of patients undergoing carotid repair at the Cleveland Clinic. In fact, the mortality rate of 0.2% in over 1500 "low-risk" asymptomatic patients treated with carotid endarterectomy is remarkably similar to the ACAS mortality rate of 0.1%. Similarly, the mortality rate was 0.5% in 925 symptomatic patients undergoing carotid endarterectomy at the Cleveland Clinic, almost identical to the 0.6% mortality rate observed in the NASCET trial.

In addition to baseline comorbidities, there exist a variety of anatomic features that are also associated with poor outcome. These include such variables as contralateral carotid occlusion, recurrent carotid lesions, and a history of radiation therapy to the neck. These factors may be quite important in determining the outcome of open carotid procedures, with regard to perioperative stroke, myocardial infarction, and death as well as softer end points such as wound complications and cranial nerve injury. These anatomic features should also be taken into account in the differentiation between high- and low-risk patients. Complications that are associated with the invasive nature of open surgery would be likely to occur at a lower frequency in the stented subgroup. As such, both clinical and anatomic baseline variables should be addressed when delineating a high-risk patient population suitable for an initial investigation of endarterectomy versus stenting.

CAROTID STENTING

Surgeons have been reluctant to embrace carotid stenting as a logical treatment for carotid bifurcation disease.[16] Noting the friable nature of carotid bifurcation atheroma, the successes of angioplasty and stenting appeared unlikely to be reproducible in a disease where symptoms occur as a result of embolic events rather than hemodynamic compromise. Initial results were unpredictably acceptable,[17] and some patients were willing to accept a minor decrement in outcome to avoid an open surgical procedure and its obligatory neck incision.

In patients with the more usual variety of carotid disease from atherosclerosis, the risk of carotid stenting is correlated with the extent of the process. Patients with diffuse disease involving the aortic arch and common carotid vessels should be viewed with caution, as should patients with significant intracranial disease. The heavily calcified, tortuous vessel is one fraught with difficulty, and the use of alternate treatment modalities should be strongly entertained. Of great importance is that patients with displacement of the arch vessels to the right side of the chest comprise a group where technical difficulties should be expected.[18]

With the demonstration of the efficacy of coronary angioplasty and stenting, there is presently great interest in percutaneous treatment for carotid disease. The large number of patients with suitable carotid lesions sparked interest on the part of industry, and, despite a national "noncoverage" policy for carotid angioplasty by the Health Care Financing Administration (now Center for Medicare and Medicaid Services), carotid stenting has become one of the most widely discussed and hotly debated topics over the past few years. Interventional cardiologists, well versed in percutaneous angioplasty, were quick to embrace the new technology. Vascular surgeons, by contrast, viewed carotid angioplasty with caution, awaiting the results of clinical trials with skepticism and trepidation.[19]

THE CAROTID STENTING TRIALS

The performance of well-designed clinical trials is the only pathway to gather objective long-term data on which clinical decisions may be based. Ultimately, the analysis of comparative outcome will resolve issues of safety and efficacy of carotid stenting in comparison to carotid endarterectomy. A relatively large number of carotid stenting trials have been organized (Table 8–1). Most have been registry-type analyses, comprising prospective entry into a series of consecutively treated patients without a comparison group. These trials include single-center studies where the investigator serves as the sponsor, but also a variety of corporate-sponsored trials with such diverse acronyms as ACCULINK for Revascularization of Carotids in High-Risk Patients (ARCHeR); Boston Scientific/EPI: A Carotid Stenting Trial for High-Risk Surgical Patients (BEACH); Carotid Artery Revascularization Using the Boston Scientific EPI Filter Wire EX and the EndoTex NexStent-EndoTex (CABERNET); Evaluation of the Medtronic AVE Self-Expanding Carotid Stent System With Distal Protection In the Treatment of Carotid Stenosis (MAVErIC); and Stenting of High risk patients Extracranial Lesions Trial with Emboli Removal (SHELTER). Most of these registries were designed to evaluate patients thought to be at high risk for standard carotid endarterectomy and organized in an effort to gain approval for the stent and/or embolic protection device. The U.S. Food and Drug Administration (FDA) has demonstrated some

TABLE 8-1. TRIALS OF CAROTID STENTING

Trial	Sponsor	Design	Stent	Protection Device
ARCHeR	Guidant	Registry, high-risk	AccuLink	AccuNet
BEACH	Boston Scientific	Registry, high-risk	Wallstent, Monorail	FilterWire EX
CABERNET	Boston Scientific, EndoTex	Registry, high-risk	NexStent	EPI Filter
CARESS	ISIS	Registry, stent and endarterectomy	Originally Wallstent, now not specified	Originally PercuSurge, now not specified
CREST	Guidant, NIH, NINDS	Randomized, lower risk	AccuLink	AccuNet
ICSS (CAVATAS-2)	UK Stroke Association	Randomized	Not specified	Not specified
MAVErIC	Medtronic	Registry, high-risk	Medtronic/AVE Self-Expanding Carotid Stent	PercuSurge GuardWire Plus
SAPPHIRE	Cordis	Randomized and registry, high-risk	Precise	AngioGuard
SECURITY	Abbott	Registry, high-risk	Xact Stent	Formerly MedNova NeuroShield, now "Emboshield" rapid exchange version
SHELTER	Boston Scientific	Registry, high-risk	Wallstent, Monorail	PercuSurge GuardWire Plus
SPACE	German government, Boston Scientific, Guidant	Randomized, stent vs. endarterectomy	Not specified	Not specified

flexibility in the consideration of device approval based on high-risk registries rather than randomized studies.

The largest carotid stent registry is the global registry organized by Mark and Michael Wholey. At the time of the latest publication from this registry, over 5000 patients had been entered from 36 centres worldwide.[20] Although the data suffer from the limitations of any registry based on unmonitored, investigator-completed questionnaires and unstandardized follow-up protocols, the results were truly exceptional. Within 30 days of the procedure, transient ischemic attacks occurred in 2.8% of the patients and minor and major stroke in 4.2%. The 30-day mortality rate was 0.9%, with a combined stroke/death rate of 5.1%. The rate of restenosis was extremely low, evident in only 3.5% of patients at one year, with only 1.4% of patients experiencing neurologic symptoms within one year of the procedure.

There have been several randomized trials of carotid stenting versus endarterectomy. A study sponsored by Schnieder (now part of Boston Scientific Corporation, Natick, MA) compared placement of the Wallstent without cerebral embolic protection to carotid endarterectomy in 219 subjects at 29 centers. To date, the results are not published but have been presented orally.[21] The results of stenting in this trial were worse than those of endarterectomy. At one year, stroke or death occurred in 12.1% of the stented group, compared to only 3.6% of the surgically treated subjects (P = 022). However, cerebral protection devices were not implemented in this study. As well, a number of sites had very little stenting experience at the time of their participation in this study.

The Carotid Revascularization Endarterectomy versus Stent Trial (CREST) is designed to compare the outcome of carotid stenting to endarterectomy in patients similar to those entered into the NASCET trial; in other words, in patients at relatively low risk for complications after carotid endarterectomy.[22] The trial employs the AccuLink™ stent and the AccuNet™ filter (Guidant Corporation, Menlo Park, CA). The goal is to randomize approximately 2500 patients into this NIH-sponsored trial.

The Study of Angioplasty with Protection in Patients at High Risk for Endarterectomy (SAPPHIRE) trial is now complete, and results were reported at the 2002 American Heart Association Meeting.[23] SAPPHIRE was actually two studies in one, a randomized portion and a registry portion. Patients deemed suitable for either stenting or endarterectomy were randomized. Patients who were thought unsuitable for endarterectomy on the basis of severe medical comorbidities or anatomic considerations were entered into a stenting registry. Lastly, a small number of patients who were considered to be unsuitable for stenting, usually on the basis of anatomic criteria, were entered into a surgical registry. The study was sponsored by Cordis/Johnson and Johnson and utilized the Precise™ stent and AngioGuard filter (Cordis, Johnson and Johnson, Warren, NJ).

Preliminary 30-day outcome from the randomized portion of the SAPPHIRE study demonstrated significant benefit of stenting over endarterectomy for the composite end point of stroke, myocardial infarction, or death. Although not assured, it is expected that this finding will persist beyond the 30-day time point, and if so, SAPPHIRE will be the study that gains FDA approval for the procedure of carotid stenting in general and the Precise/AngioGuard system in particular, employed in the subgroup of patients at high risk for standard carotid endarterectomy.

USE OF EMBOLIC PROTECTION DEVICES

Atherosclerotic plaques in general and high-grade carotid plaques in particular are laden with lipid and calcium. The luminal surface may be covered with a carpet of aggregated platelets and fibrin. Such a situation, of course, is a set-up for distal embolization with percutaneous carotid interventions (Figure 8–1). In vivo studies have demonstrated a large number of emboli released during angioplasty and stenting of the carotid bifurcation,[24] and these data have been corroborated in the clinical setting using transcranial Doppler during stent procedures.[25] For this reason, a variety of "embolic protection devices" have been developed. Although none has gained approval for use in the cerebral circulation, at least one, PercuSurge® (Medtronic/AVE, Santa Rosa, CA), is approved for saphenous vein graft indications.

Embolic protection devices can be grouped into three categories. First are those that function as "nets" or "filters" placed distally in the internal carotid artery at the time of angioplasty and stenting. This group includes the AngioGuard filter, the AccuNet™ filter, the FilterWire EX™ embolic protection device (Boston Scientific, Natick, MA), and the MedNova NeuroShield™ device (MedNova Inc., Galway, Ireland). Second are devices that arrest blood flow in the internal carotid artery, allowing aspiration of the static column of blood that potentially contains atheroembolic debris. Foremost in this category is the PercuSurge device, associated with an obligate period of internal carotid flow arrest lasting approximately 10 minutes.[15] Third are devices that function with a balloon at the end of a sheath, allowing the operator to reverse flow in the internal carotid artery and extract potential emboli though the sheath. In the case of the Parodi

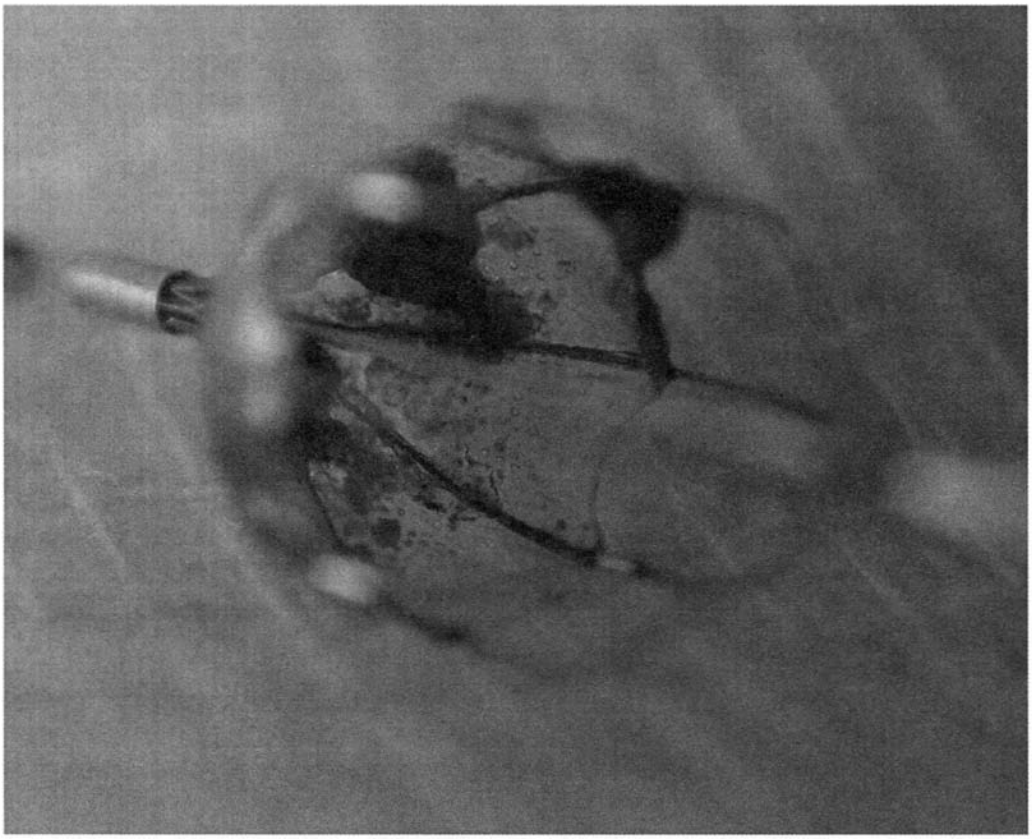

Figure 8-1. The AngioGuard filter removed after stenting of a patient with a high grade asymptomatic stenosis

Anti-Emboli System (ArteriA Medical Science, Inc, San Francisco, CA), the outflow channel of the sheath can be connected to the femoral vein, trapping the emboli in a filter and allowing the cleansed blood to flow into the venous circulation. Although this class of devices has some theoretical attraction, avoiding the need to cross the carotid lesion, a mandatory period of absent or even reversed flow in the internal carotid artery is a potential shortcoming.

Despite the logic in using an embolic protection device, they are not without problems and complications. For instance, the filters must cross the lesion to be placed distally beyond the lesion. The profile of the devices, although small, still accounts for a small risk of embolization when crossing the lesion. Also, the filters may become filled with debris, arresting flow in the internal carotid artery. Although the absence of flow is not in itself a serious problem (only a small minority of patients will experience mental status changes), the function of the filters depends on flow. Once there is no flow, emboli are no longer trapped in the filter but rather will reside within the static column of blood between the carotid lesion and the filter. Unless this blood is vigorously aspirated (for example, though a 5F catheter placed just proximal to the filter) recapture of the filter will result in restitution of antegrade internal carotid blood flow and cerebral embolization of debris.

SUMMARY

Definitive treatment of extracranial carotid disease is well entrenched for patients with both symptomatic and asymptomatic severely stenotic lesions. The gold standard remains open surgical endarterectomy,[26,27] but there is strong interest in carotid stenting from the clinical and investigative perspective. Initial results of stenting appear to be quite reasonable, challenging traditional endarterectomy in low-risk patients and probably surpassing endarterectomy in the higher-risk subgroups. With advances in stents, delivery systems, antiembolic devices, and, most important, with improvements in the technical expertise of the operators, it is likely that carotid stenting will become the treatment of choice for patients with significant carotid disease.

REFERENCES

1. Eastcott HHG, Pickering GW, Rob CG. Reconstruction of internal carotid artery in a patient with intermittent attacks of hemiplegia. *Lancet*. 1954;2:994.
2. DeBakey ME. Successful carotid endarterectomy for cerebrovascular insufficiency. Nineteen-year follow-up. *JAMA*. 1975;233:1083–185.
3. Carrea R, Molins M, Murphy G. Surgical treatment of spontaneous thrombosis of the internal carotid artery in the neck. Carotid-carotideal anastomosis. Report of a case. *Acta Neurol Latinoamer*. 1955;1:17.
4. Fields WS, North RR, Hass WK et al. Joint study of extracranial arterial occlusion as a cause of stroke. I. Organization of study and survey of patient population. *JAMA*. 1968;203:153–158.
5. Fields WS, Maslenikov V, Meyer JS et al. Joint study of extracranial arterial occlusion. V. Progress report of prognosis following surgery or nonsurgical treatment for transient cerebral ischemic attacks and cervical carotid artery lesions. *JAMA*. 1970;211:1993–2003.
6. Hsia DC, Krushat WM, Moscoe LM. Epidemiology of carotid endarterectomies among Medicare beneficiaries. *J Vasc Surg*. 1992;16:201–208.
7. Barnett HJ, Taylor DW, Eliasziw M et al. Benefit of carotid endarterectomy in patients with symptomatic moderate or severe stenosis. North American Symptomatic Carotid Endarterectomy Trial Collaborators [see comments]. *N Engl J Med*. 1998;339:1415–1425.
8. Hobson RW, Weiss DG, Fields WS. Efficacy of carotid endarterectomy for asymptomatic carotid stenosis. *N Engl J Med*. 1993;328:221–227.
9. Moore WS, Young B, Baker WH et al. Surgical results: a justification of the surgeon selection process for the ACAS trial. The ACAS Investigators. *J Vasc Surg*. 1996;23:323–328.
10. Kresowik TF, Bratzler D, Karp HR et al. Multistate utilization, processes, and outcomes of carotid endarterectomy. *J Vasc Surg*. 2001;33:227–234.
11. Ferguson GG, Eliasziw M, Barr HW et al. The North American Symptomatic Carotid Endarterectomy Trial: surgical results in 1415 patients. *Stroke*. 1999;30:1751–1758.
12. Sarac TP, Hertzer NR, Mascha EJ et al. Gender as a primary predictor of outcome after carotid endarterectomy. *J Vasc Surg*. 2002;35:748–753.
13. Golledge J, Cuming R, Beattie DK et al. Influence of patient-related variables on the outcome of carotid endarterectomy. *J Vasc Surg*. 1996;24:120–126.
14. Ouriel K, Hertzer NR, Beven EG et al. Preprocedural risk stratification: identifying an appropriate population for carotid stenting. *J Vasc Surg*. 2001;33:728–732.
15. Wennberg DE, Lucas FL, Birkmeyer JD et al. Variation in carotid endarterectomy mortality in the Medicare population: trial hospitals, volume, and patient characteristics. *JAMA*. 1998; 279:1278–1281.

16. Statement regarding carotid angioplasty and stenting. Society for Vascular Surgery. International Society for Cardiovascular Surgery, North American Chapter. *J Vasc Surg*. 1996;24:900.
17. Yadav JS, Roubin GS, King P et al. Angioplasty and stenting for restenosis after carotid endarterectomy. Initial experience. *Stroke*. 1996;27:2075–2079.
18. Al Mubarak N, Roubin GS, Iyer SS, Vitek JJ. New G. Techniques of carotid artery stenting: the state of the art. *Semin Vasc Surg*. 2000;13:117–129.
19. Veith FJ, Amor M, Ohki T et al. Current status of carotid bifurcation angioplasty and stenting based on a consensus of opinion leaders. *J Vasc Surg*. 2001;33:S111–S116.
20. Wholey MH, Wholey M, Mathias K et al. Global experience in cervical carotid artery stent placement. *Catheter Cardiovasc Interv*. 2000;50:160–167.
21. Alberts MJ. Results of a Multicenter Prospective Randomized Trial of Carotid Artery Stenting vs. Carotid Endarterectomy. *Abstracts of the International Stroke Conference*. 2001;32: 325.
22. Roubin GS, Hobson RW, White R et al. CREST and CARESS to evaluate carotid stenting: time to get to work! *J Endovasc Ther*. 2001;8:107–110.
23. Yadav, J. S. The 30 day SAPPHIRE Results. Presented at the American Heart Association 2002 Annual Meeting. 2002.
24. Ohki T, Veith FJ. Carotid artery stenting: utility of cerebral protection devices. *J Invasivl Cardiol*. 2001;13:47–55.
25. Jordan WD, Jr., Voellinger DC, Doblar DD, Plyushcheva NP, Fisher WS, McDowell HA. Microemboli detected by transcranial Doppler monitoring in patients during carotid angioplasty versus carotid endarterectomy. *Cardiovasc Surg*. 1999;7:33–38.
26. Zarins CK. Carotid endarterectomy: the gold standard. *J Endovasc Surg*. 1996;3:10–15.
27. Hertzer NR, Ouriel K. Results of carotid endarterectomy: the gold standard for carotid repair. *Semin Vasc Surg*. 2000;13:95–102.

9

Carotid Stents and Embolic Protection Devices

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INTRODUCTION

Although it is well known that early outcomes of carotid artery stenting (CAS) can be influenced by the skills of the interventionalist, symptomatic status of the patient, and characteristics of the target lesion, what many treating physicians want to know is the impact of the type of stent used in conjunction with the efficacy of cerebral protection employed. Approved carotid stents are all self-expanding but vary in architecture and metal composition, which may impact early and late outcomes of CAS. Three categories of embolic protection devices (EPDs) are recognized; however, currently only the distal filter devices are readily available in the United States. The design characteristics of each of these systems may influence the periprocedural events associated with CAS. This chapter will review the currently approved carotid stent systems and approved mechanical EPDs.

CAROTID STENTS

To date, there are a number of stent designs indicated for use in CAS: Wallstent (Boston Scientific, Natick, MA), Acculink (Abbott Vascular, Santa Rosa, CA), X-act (Abbott Vascular), Precise (Cordis, a Johnson and Johnson Company, Miami, FL) and Protégé (ev3 Inc., Plymouth, MN). All are self-expanding stents, yet their metal alloys, architecture, and configurations differ. Any or all of these properties may have a clinical impact on the early and long-term success of CAS.

Stent Alloys

There exist two primary materials by which most self-expanding carotid stents are created: elgiloy and nitinol. Elgiloy, also known as conichrome, is a biomedical grade cobalt-chromium-iron-nickel-molybdenum alloy which is then braided into a tubular mesh

configuration creating what is best recognized as the carotid Wallstent. This particular stent is stainless steel and designed with a closed cell configuration. The second major self-expanding stent alloy is made of nickel and titanium and is commonly referred to as nitinol. Stents constructed of this alloy include Precise, X-act, Acculink, and Protégé. Most of these stents are constructed from a nitinol tube that is laser-cut during production to create a meshed device with thermal expansion properties. At room temperature, the stent exists in a compact shape and can be placed within a delivery sheath. Once exposed to body temperature, the stent conveniently expands as the sheath is withdrawn based upon a predetermined shape. These stents can be of either open or closed cell design and can be configured into a straight tubular or tapered design.

Theoretically, one of the biggest differences between elgiloy and nitinol stents occurs with respect to their behavior in the deployed state. The woven mesh structure of elgiloy allows it to adapt its diameter to the width of a vessel lumen, thereby allowing for more optimal vessel conformability versus the behavior of nitinol stents, which are completely determined by their thermal memory. A downside of the adaptation process of elgiloy stents is that the implanted stent length is highly dependent on the vessel diameter. Thus, excessive oversizing of the stent relative to the true vessel luminal diameter can result in significant variations in overall length of the implanted device. However, in contrast to nitinol stents, the Wallstent is reconstrainable, meaning that after partial deployment, if positioning is not appropriate, the device can be recaptured, repositioned, and deployed once more. Last, the chronic outward radial force of the Wallstent is significantly less than the available nitinol stents, which may contribute to plaque protusion after implantation.[1] Although no prospective, randomized data are yet available to compare the results of these two alloys, the lack of radial force may have implications for late embolic events as a result of plaque protusion and unstable plaque trapping.

Stent Architecture

Scaffolding of the carotid bifurcation lesion is achieved after placement of the self-expanding stent across the atherosclerotic lesion. Approved carotid stents are either open cell or closed cell systems. However, newer stents in clinical trials have been developed that are a hybrid—mixing areas of open cell design with a closed cell structure. Cell structure and geometry of a stent are critical determinants of stent flexibility. In general, open cell stents tend to be more conformable and flexible as compared with closed cell designs because of fewer connections between individual hooped cells. Unfortunately, increases in flexibility tend to compromise strength and "therefore" make these stents more susceptible to compression and elongation once implanted. Additionally, increased flexibility leads to lower scaffolding, which allows for stent strut protrusion into the lumen at points of curvature of the vessel or excessive irregularity of a rigid plaque and subsequent particle protrusions. Clinically, the reduction in particle trapping may increase the risk of delayed embolic events.

Based on these concepts, some believe that closed cell stents may be best suited for CAS, because the design would improve chronic outward radial force, increase scaffolding, and reduce free cell area. One notable restrospective study by Bosiers et al., examined the clinical impact of cell design on CAS outcomes.[2] The investigators found that closed cell stents were superior in reducing postprocedural neurologic events in symptomatic patients but not in asymptomatic patients. Despite this data, open cell stents should not be excluded from our arsenal because they are clearly more advantageous in tortuous vessels due to their improved conformability.

Stent Configurations

Cell structure, extent of scaffolding and composition of the stents are part of the equation, and yet changes from a straight to a tapered stent to accommodate the anatomic carotid bifurcation might also have clinical consequences. This becomes important because in the majority of cases, stent placement crosses the external carotid artery (ECA) orifice and needs to be well apposed to both the common carotid artery (CCA) and the internal carotid artery (ICA) lumina. The natural size mismatch between the ICA and CCA leads to more stent oversizing in the ICA. Additionally, "self-tapering" of straight stents may not allow for good wall apposition along the entire length of the treated segment. The impact of these two circumstances are excessive oversizing in the ICA, increased chronic outward radial force in the ICA, decreased free cell area in the ICA, and poor or no scaffolding along portions of the atherosclerotic plaque.

Two primary configurations of tapered stents have been developed:

1. conical represented by Acculink and X-act, and
2. shouldered represented by Protégé. In the conical tapered stents, there is a gradual decrease in the diameter of the stent from proximal to distal, whereas in the shouldered tapered stents, there exists a short transition point in the midsegment of the stent. When considering the carotid vasculature, a significant diameter reduction occurs at the bifurcation, thus theoretically making conical tapered stents more favorable. The primary concern of too much oversizing in the ICA to accommodate to the diameter of the CCA is the risk of restenosis from either excessive metal coverage or undue chronic outward radial force.

Unfortunately, few data exist at this point to allow for a direct comparison of tapered versus nontapered stents. Our institution recently published results from a single-center retrospective review of 308 cases of CAS treated with EPDs and either tapered or nontapered self-expanding nitinol stents.[3] A total of 156 tapered stents were evaluated as well as 152 nontapered stents. The study revealed no statistical difference between the 30-day ipsilateral stroke/transient ischemic attack (TIA) rates in tapered (3.2%) and nontapered stents (1.3%, $P=0.5$). However, at mid-term follow-up, restenosis ($\geq$ 80%) or asymptomatic stent occlusion was detected in 2.3% of cases in arteries treated with nontapered stents as compared with 0% in the tapered stent group. This analysis is underpowered, but the observed trend was that tapered stents had a lower incidence of restenosis or asymptomatic occlusion. Larger studies are needed to confirm these results.

EMBOLIC PROTECTION DEVICE

Three general categories of mechanical EPDs have been developed to guard against distal cerebral embolization during CAS: (1) distal balloon occlusion, (2) distal filtration, and (3) proximal balloon occlusion.

Distal Balloon Occlusion

Distal balloon occlusion systems are most commonly represented by the PercuSurge GuardWire (Medtronic Vascular, Santa Rosa, CA) in which the balloon blocks flow within the ICA and emboli are aspirated before balloon deflation and catheter removal.

Advantages of this approach tend to include a lower crossing profile as well as the ability of the balloon to capture particles of all sizes. Disadvantages include theoretical local injury to the ICA by balloon overnflation, possible embolization via ECA branches, intolerance of total ICA occlusion by the patient, "suction shadowing," and of most concern, the possibility of embolization during initial lesion crossing prior to inflation of the balloon.[4–5] "Suction shadow" was reported by Tuber et al. in which a 5.2% periprocedural adverse neurologic event rate was noted despite use of balloon occlusion. It is theorized that this effect occurs when suction catheters fail to aspirate all emboli because some emboli may be too large or the blood column adjacent to the balloon device may not be effectively aspirated.[6] The TriActiv FX (Kensey Nash), a more recently introduced distal balloon occlusion system, helps reduce "suction shadow" through the introduction of an active flush system that allows for aggressive particle removal from vessel walls and areas adjacent to the inflated balloon. An additional complication reported with distal balloon occlusion EPDs is the risk of neurologic intolerance during balloon occlusion.[5] This can manifest with a variety of symptoms ranging from mild confusion to seizures. Fortunately, the majority of instances are transient and resolve after re-establishing antegrade flow by deflating the occlusion balloon.

Distal Filters

Distal filters tend to be the most commonly employed cerebral protection devices in most centers. This form of protection allows for continued antegrade cerebral flow through the device while emboli are captured. At the end of the procedure, the filter element is reconstrained and removed with the captured particulate debris. Advantages include the ability to perform angiograms throughout the procedure as well as maintenance of flow, thereby reducing the likelihood of cerebral hypoperfusion. Disadvantages include a larger crossing profile, thereby making it difficult to cross some more tortuous, possibility of incomplete apposition of the filter to the arterial wall, concern for filter thrombosis, filter pore size limitations, and risk of embolization during the initial lesion crossing prior to positioning and deployment of the filter. A review of the current literature evaluating the outcomes of 2263 CAS procedures using various filter devices—Spider Embolic Protection Device (ev3), AngioGuard XP (Cordis), FilterWire EZ (Boston Scientific), AccuNet (Abbott Vascular), and Emboshield (Abbott Vascular)—demonstrated a periprocedural stroke rate of roughly 2%.[7-19]

Proximal Balloon Occlusion

The third and final category of EPD used for carotid interventions is the proximal balloon occlusion devices. These are represented by the NPS (Neuroprotection system, formerly known as PAES—Parodi Anti-Emboli System—W.L. Gore & Associates, Flagstaff, AZ) and the Mo.Ma device (Invatec, Roncadelle, Italy). Both utilize the concepts of carotid occlusion that are used to measure carotid stump pressures during carotid endarterectomy procedures. Instead of clamping the CCA and ECA, each vessel is controlled with balloon occlusion. The balloon for the CCA is mounted on a working sheath placed in the distal CCA, which provides a platform to proceed with CAS after flow reversal is instituted. The NPS device uses both passive and active flow reversal through a sumping mechanism created by an external arteriovenous shunt created between the femoral artery and the femoral vein.[11,20] The Mo.Ma device utilizes flow stagnation as a mechanism to provide

for cerebral protection—this is achieved by occlusion of the ECA as well as ICA by two independently inflated low-pressure compliant balloons.[21] This allows for intermittent collection of debris between steps of the procedure and of aspiration of debris at the conclusion of the procedure through a separate working channel. Taken together, advantages of these devices include complete protection of both the ICA as well as the ECA. Protection starts prior to crossing the lesion, there is greater freedom for wire choices, and the device is capable of capturing particles of all sizes. Disadvantages include the need for a larger groin sheet, typically 9-10 Fr, potential arterial injury at balloon inflation sites, and most importantly, total arrest of antegrade flow on the protected side. The largest series of reports using these devices has shown a periprocedural stroke rate of approximately 1 to 2%.[13,15,20,22–23] Notably transient neurologic intolerance, which resolved at the conclusion of the flow reversal, occurred in roughly 10 to 12% of cases. One additional study by Criado et al. showed that cerebral venous oxygen saturation, as measured in the internal jugular vein, was significantly lower during ICA occlusion than during ICA flow reversal.[24] Presumably, this is due to enhancement of collateral flow via the circle of Willis during periods of passive or active flow reversal.

A primary advantage of the proximal occlusion devices is the ability to capture particulate debris of all sizes. In one ex vivo study,[25] a filter device captured only 88% of embolized particles as indicated by transcranial Doppler monitoring. Interestingly, Angelini et al.[8] have shown through histopathologic analysis that microscopic emboli captured in distal filters during CAS have a mean particle size of 289.5 ± 512 micrometers in the major axis and a mean of 119.7 ± 186.7 μ in the minor axis. Prior data have shown that cerebral microcirculation is composed of numerous vessels with diameters less than 300 microns: arterioles tend to have a diameter of between 12 and 100 microns, and capillaries a diameter of approximately 12 microns. Current distal filter devices use membranes with wide-ranging pore sizes (80-500 μ).[10] Thus it is possible that small microemboli may escape the pores of the filter devices and create short-term effects such as stroke or long-term problems including dementia or cognitive failure. According to recent studies, small microemboli showers can potentially trigger platelet aggregation and may cause microvascular obstruction with concomitant prolonged vessel vasospasm and subsequent cerebral infarction.[16,26]

Although the use of EPDs during CAS has not been validated in randomized trials, data from registries and observational studies support their routine use and most interventionalists consider them to be the standard of care. Advocates of performing CAS without the uses of EPDs argue that the larger proportion of strokes occurs postprocedurally, and indeed this seems to be the case. Four studies were identified that distinguished between intraprocedural stroke and postprocedural stroke as identified by clinical findings and/or diffusion-weighted MRI. A total of 368 cases identified were done without the use of EPDs and closed cell stent types, in which there was a 2.5% cumulative incidence of all intraprocedural stroke. Postprocedure, a 5.2% incidence of all stroke was noted.[27-29] In addition, in two other studies, 1069 cases were performed with the use of distal filters EPDs and primarily closed cell stents, in which there was a 1.6% and 1.9% cumulative incidence of intraprocedural stroke and postprocedure stroke, respectively.[27,29] When both subsets of data were combined a total of 65 strokes, both major and minor were identified, of which 60% occurred in the postprocedural period. Indeed, a larger proportion of strokes do occur postprocedure; however, it remains that the incidence of intraprocedural stroke with the use of EPDs is lower than without their use.

SUMMARY

The outcomes of CAS with EPDs have improved markedly over the last decade. This can be attributed to advances in technology, training, and patient selection. While we await results from randomized trials on symptomatic and asymptomatic standard-risk patients treated with CEA or CAS to guide us on the role of these two modalities, it is crucial that we remain committed to understanding potential areas of improvement with regards to the equipment at our disposal. More data on the early and long-term effects of stent design are needed as well as more comprehensive data on the limitations of EPDs.

REFERENCES

1. Muller-Hulsbeck S, Schafer PJ, Charalambous N, et al. Comparison of carotid stents: an in-vitro experiment focusing on stent design. J Endovasc Ther 2009;16:168–77.
2. Bosiers M, de Donato G, Deloose K, et al. Does free cell area influence the outcome in carotid artery stenting? Eur J Vasc Endovasc Surg Feb 2007;33:135–41; discussion 142–3.
3. Brown KE, Usman A, Kibbe MR, et al. Carotid stenting using tapered and nontapered stents: associated neurological complications and restenosis rates. Ann Vasc Surg Jul-Aug 2009;23:439–45.
4. Eskandari MK. Design and development of mechanical embolic protection devices. Expert Rev Med Devices May 2006;3:387–93.
5. Eskandari MK, Najjar SF, Matsumura JS, et al. Technical limitations of carotid filter embolic protection devices. Ann Vasc Surg Jul 2007;21:403–7.
6. Tubler T, Schluter M, Dirsch O, et al. Balloon-protected carotid artery stenting: relationship of periprocedural neurological complications with the size of particulate debris. Circulation Dec 4 2001;104:2791–6.
7. Al-Mubarak N, Colombo A, Gaines PA, et al. Multicenter evaluation of carotid artery stenting with a filter protection system. J Am Coll Cardiol Mar 6 2002;39:841–6.
8. Angelini A, Reimers B, Della Barbera M, et al. Cerebral protection during carotid artery stenting: collection and histopathologic analysis of embolized debris. Stroke Feb 2002;33:456–61.
9. Castellan L, Causin F, Danieli D, Perini S. Carotid stenting with filter protection. Correlation of ACT values with angiographic and histopathologic findings. J Neuroradiol Mar 2003;30:103–8.
10. Fanelli F, Bezzi M, Boatta E, Passariello R. Techniques in cerebral protection. Eur J Radiol Oct 2006;60:26–36.
11. Green DW, Sanchez LA, Parodi JC, et al. Acute thromboembolic events during carotid artery angioplasty and stenting: etiology and a technique of neurorescue. J Endovasc Ther Jun 2005;12:360–5.
12. Hart JP, Peeters P, Verbist J, et al. Do device characteristics impact outcome in carotid artery stenting? J Vasc Surg Oct 2006;44:725–30; discussion 730–21.
13. Henry M, Polydorou A, Henry I, et al. Carotid angioplasty and stenting under protection. Techniques, results and limitations. J Cardiovasc Surg (Torino) Oct 2006;47:519–46.
14. Kasirajan K, Schneider PA, Kent KC. Filter devices for cerebral protection during carotid angioplasty and stenting. J Endovasc Ther Dec 2003;10:1039–45.
15. Rabe K, Sugita J, Godel H, Sievert H. Flow-reversal device for cerebral protection during carotid artery stenting—acute and long-term results. J Interv Cardiol Feb 2006;19:55–62.
16. Reimers B, Corvaja N, Moshiri S, et al. Cerebral protection with filter devices during carotid artery stenting. Circulation Jul 3 2001;104:12–5.

17. Reimers B, Tubler T, de Donato G, et al. Endovascular treatment of in-stent restenosis after carotid artery stenting: immediate and midterm results. J Endovasc Ther Aug 2006;13: 429–35.
18. Rubartelli P, Brusa G, Arrigo A, et al. Transcranial Doppler monitoring during stenting of the carotid bifurcation: evaluation of two different distal protection devices in preventing embolization. J Endovasc Ther Aug 2006;13:436–42.
19. White CJ, Iyer SS, Hopkins LN, et al. Carotid stenting with distal protection in high surgical risk patients: the BEACH trial 30 day results. Catheter Cardiovasc Interv Apr 2006;67: 503–12.
20. Adami CA, Scuro A, Spinamano L, et al. Use of the Parodi anti-embolism system in carotid stenting: Italian trial results. J Endovasc Ther Apr 2002;9:147–54.
21. Scheinert D, Scheinert S, Sax J, et al. Prevalence and clinical impact of stent fractures after femoropopliteal stenting. J Am Coll Cardiol Jan 18 2005;45:312–5.
22. Reimers B, Schluter M, Castriota F, et al. Routine use of cerebral protection during carotid artery stenting: results of a multicenter registry of 753 patients. Am J Med Feb 15 2004; 116:217–22.
23. Reimers B, Sievert H, Schuler GC, et al. Proximal endovascular flow blockage for cerebral protection during carotid artery stenting: results from a prospective multicenter registry. J Endovasc Ther Apr 2005;12:156–65.
24. Criado E, Doblas M, Fontcuberta J, et al. Carotid angioplasty with internal carotid artery flow reversal is well tolerated in the awake patient. J Vasc Surg Jul 2004;40:92–7.
25. Ohki T, Roubin GS, Veith FJ, et al. Efficacy of a filter device in the prevention of embolic events during carotid angioplasty and stenting: an ex vivo analysis. J Vasc Surg Dec 1999; 30:1034–44.
26. Williams DO. Carotid filters: new additions to the interventionist's toolbox. Circulation Jul 3 2001;104:2–3.
27. Alexandrescu V, Ngongang C, Proumen J, et al. Filter-protected carotid stenting via a minimal cervical access with transitory aspirated reversed flow during initial passage of the target lesion. J Endovasc Ther Apr 2006;13:196–204.
28. Davila-Roman VG, Murphy SF, Nickerson NJ, et al. Atherosclerosis of the ascending aorta is an independent predictor of long-term neurologic events and mortality. J Am Coll Cardiol Apr 1999;33:1308–16.
29. Verzini F, Cao P, De Rango P, et al. Appropriateness of learning curve for carotid artery stenting: an analysis of periprocedural complications. J Vasc Surg Dec 2006;44:1205–11; discussion 1201–02.

10

Interventional Approaches to Acute Stroke

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INTRODUCTION

Stroke continues to be the third leading cause of death in the United States, with an approximate incidence of 800,000 people annually. Eighty-five percent of strokes are ischemic in nature, with an associated mortality between 53% and 92%.[1-4] Over the past two decades, several studies have shown that the effectiveness of interventional therapy for stroke is time-dependent.[5] Furthermore, successful recanalization of a previously occluded vessel correlates with improvement in clinical outcomes.[2,6–7] As a result, the current focus of treatment for acute stroke is the delivery of prompt therapy that effectively restores flow to the occluded vascular territory.

In assessing the time frame available to salvage brain tissue, studies have suggested that tissue may continue to be at risk up to 24 hours after stroke onset; however, the clinical significance of this extended window is unclear.[8–9] While intravenous tissue plasminogen activator (IV tPA) administration has shown benefit up to 3 hours from stroke onset, extension of the window to 6 hours has failed to show benefit.[10–11] On the other hand, recent combined analysis of several studies has suggested that the benefit of IV tPA may extend to 4.5 hours after stroke onset.[5,12–13] More recently, European Cooperative Acute Stroke Study (ECASS) 3 demonstrated that intravenous alteplase administered 3 to 4.5 hours after the onset of symptoms improved clinical outcomes in patients with acute ischemic stroke.[13] However, patients with severe ischemic strokes were excluded from the trial. This raises a concern regarding the applicability of these data to patients with large intracranial vessel occlusions. The limited time window for IV tPA infusion and the multitude of contraindications to full dose IV tPA have spawned interest in infusion of local tPA at lower doses and/or mechanical approaches to revascularizing acutely occluded intracranial vessels. Additionally, a number of trials have investigated the use of intra-arterial (IA) infusion of thrombolytics at the location of the thrombus within 6 hours of stroke onset.[14-17]

For patients presenting outside the window for tPA and for those in whom tPA is contraindicated, mechanical interventional devices including the MERCI clot retrieval

system (Concentric Medical, Mountain View, CA) and the Penumbra system (Penumbra, Inc., Alameda, CA), have recently been approved for up to 8 hours after stroke onset.[19-21]

TABLE 10-1. SUMMARY OF RECENT LANDMARK TRIALS IN THE TREATMENT OF ACUTE STROKE

					Window				
Study	Year	#	Study Type	Population	Median NIHSS	Goal	Median	Drug Infusion	Dose/Hour
PROACT I	1998	46	Multicenter, prospective, randomized, double-blind, controlled	Symptomatic M1/M2	17 (19 placebo)	6 hours	5.4 hours (5.7 hours placebo)	Pro-UK	6 mg/hr
PROACT II	1999	180	Multicenter, prospective, randomized, open-label, controlled	Symptomatic M1/M2	17 (17 control)	6 hours	5.3 hours	Pro-UK	9 mg/hr
IMS	2006	80	Multicenter, prospective, nonrandomized, single arm	ICA, MCA, ACA, PCA, BA	18	3 hours	2.3 hours	re-tPA	0.6 mg/kg IV, plus up to 22 mg IA @ 9 mg/hr
IMS II	2007	81	Multicenter, prospective, nonrandomized, single arm	Same as IMS I	19	3 hours	2.4 hours	re-tPA	0.6 mg/kg IV, plus up to 22 mg IA @ 9 mg/hr
MERCI	2005	151	Multicenter, prospective, nonrandomized, single arm	VA, BA, ICA, M1, M2	20	8 hours	4.3 hours (mean)	None	N/A
Multi MERCI	2008	164	Multicenter, international, prospective, nonrandomized, single arm	VA, BA, ICA, M1, M2	19	8 hours	4.3 hours	IV tPA	N/A
Penumbra	2009	125	Multicenter, prospective, nonrandomized, single arm	"large intracranial vessels"	17	8 hours	4.3 hours (mean)	None	N/A
SARIS	2009	20	Prospective pilot, single arm, single center	Intracranial artery <14 mm	13	8 hours	4.9 hours	60% of patients: eptifibatide, reteplase	13 mg, 6 mg respectively

This chapter summarizes recent landmark studies related to the treatment of acute ischemic stroke, ongoing promising trials, and potential future directions and concludes with case illustrations. Table 10–1 and Figure 10–1 provide a summary of the landmark studies discussed below.

Pharmacologic Agent							
IV Heparin	Device	mRS≤2 @ 90 days	Recannalization Rate (TIMI 2 or 3)	Asymptomatic Hemorrhage	Symptomatic Hemorrhagic Conversion	Procedural Complications	All-Cause Mortality @ 90 days
Yes, high dose initially, low dose later.	None	30.8% (21.4% placebo)* mRS≤1	57.7% (14.3% placebo)	42.3% (7.1% placebo)	15.4% (vs. 7.1% placebo)	N/A	26.9% (42.9% placebo)
Yes, low dose	None	40% (25% control)	66% (18% control)	35% (13% control)	10% (vs. 2% control)	9% (7% control)	25% (vs. 27% control)
Yes	None	43%	56%	43%	6.30%	10%	16%
Yes	EKOS microinfusion catheter	46%	60%	32.10%	9.90%	3.70%	16%
Yes	Merci Retrieval System (X5,X6)	22.60%	48.0%	27.7%	7.80%	13.00%	43.5%
Yes	Merci Retrieval System (L5)	36%	55.0%	N/A	9.80%	9.80%	34.0%
Yes, no standard dose	Penumbra System	25%	81.60%	28%	11.20%	12.80%	32.80%
Yes	Wingspan SES, Enterprise SES	45%* mRS≤1	100%	10%	5%	N/A	25% (1 month)

PROACT: Prolyse in Acute Cerebral Thromboembolism
IMS: Interventional Management of Stroke
MERCI: Mechanical Embolus Removal in Cerebral Ischemia
SARIS: Stent-Assisted Recanalization in Acute Ischemic Stroke
pro-UK: pro-urokinase
tPA: tissue plasminogen activator
ICA: internal carotid artery
MCA: middle cerebral artery
ACA: anterior cerebral artery
PCA: posterior cerebral artery
BA: basilar artery
VA: vertebral artery
M1: horizontal segment of MCA
M2: insular segment of MCA

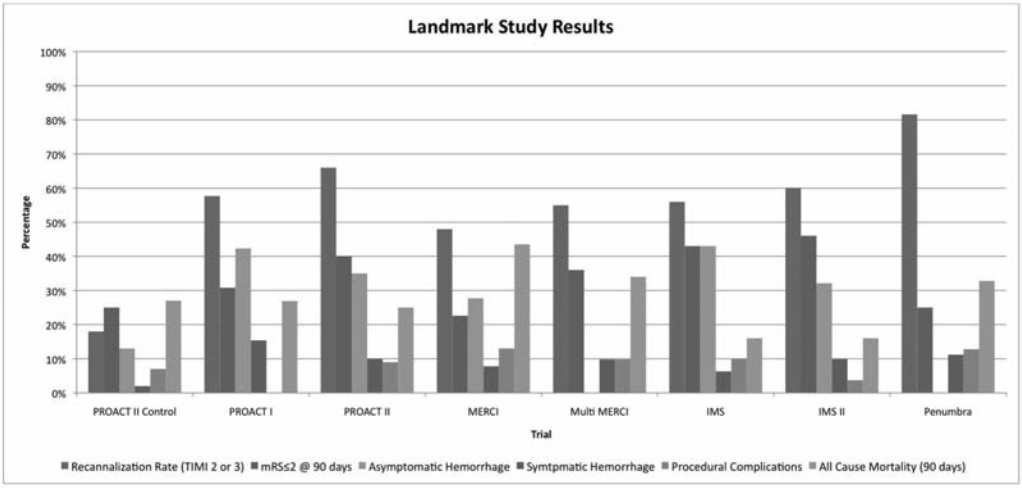

Figure 10-1. Summary of recent landmark trials in the treatment of acute stroke. MERCI: Mechanical Embolus Removal in Cerebral Ischemia, SES: self-expanding stent

LANDMARK STUDIES

PROACT I (1998, N=46)[16]

The Prolyse in Acute Cerebral Thromboembolism (PROACT) trial was a multicenter, prospective, randomized control trial that compared the efficacy of direct IA infusion of pro-urokinase (pro-UK) versus placebo for the treatment of symptomatic middle cerebral artery (MCA) occlusion within 6 hours of stroke onset. The investigators chose to include only stroke patients with acute MCA occlusions (M1 and M2 branches) to increase the homogeneity of the patient population. Pro-UK is a proenzyme that is locally converted to urokinase by fibrin-associated plasmin at the thrombus surface and whose effects are augmented by the presence of heparin. In this study, the recanalization of an occluded vessel, evidenced by achieving a Thrombolysis in Myocardial Infarction (TIMI) score of 2 or 3, was significantly more likely in the pro-UK group than the placebo group (57.7% vs. 14.3%, respectively). There was also a 9.4% improvement in clinical outcome (assessed by a modified Rankin score [mRS] of 0 or 1) and a 16% reduction in mortality at 90 days, although neither change was statistically significant. The overall risk of intracranial hemorrhage (ICH) was elevated in the pro-UK group to 42.3% (compared with 7.1% in the placebo group), although only 15.4% of hemorrhages were symptomatic (defined as a decline in neurologic status).

Notably, the rate of developing ICH postprocedurally decreased to 20% from 72.7% after the investigators reduced the dose of heparin being used. Concurrently, there was a reduction in the rate of recanalization from 81.8 to 40%.

PROACT II (1999, N=180)[17]

The PROACT II study, which was enrolling at the time the results of PROACT were published, was a multicenter, prospective, randomized control trial that primarily aimed at assessing the neurologic outcome of the use of IA pro-UK in the treatment of MCA strokes. Unlike the original PROACT trial, the investigators used a control group in which no IA infusion was performed in place of a placebo infusion. Although the investigators used the

lower heparin dose from the latter part of the original trial, they increased the dose of IA pro-UK to 9 mg/hr from 6 mg/hr.

The recanalization rate in the pro-UK group was 66%, representing a statistically significant 15% absolute increase over the control group, which was attributed to the higher pro-UK dose. Although the rate of ICH was 35% (compared with 13% in the control group), the rate of symptomatic hemorrhage was 10% (compared with 2% in the control group). Although mortality at 90 days was not significantly improved (25% vs. 27% in the control group), neurologic outcomes (mRS≤2) were significantly better in the pro-UK group (40% vs. 25% in the control group).

IMS I (2006, N=80)[14]

The Interventional Management of Stroke (IMS) study was a multicenter, prospective, single-arm study aimed at assessing the feasibility and safety of combined IV and IA tPA administered within 3 hours of stroke onset. The investigators used historical controls from the National Institute of Neurological Disorders and Stroke (NINDS) trial of IV tPA alone[10] and built upon earlier data from the Emergency Management of Stroke (EMS) Study.[18]

The investigators found that neurologic outcome at 90 days (43%) and mortality at 90 days (16%) were significantly improved when compared with the placebo arm of the NINDS trial (28% and 24%, respectively). There was no difference in the rate of symptomatic hemorrhage comparable to the IV tPA arm of the NINDS study (6.3% vs. 6.6%, respectively). Although there was no significant improvement in neurologic outcome or mortality rates compared with the IV tPA arm of NINDS, the IMS study included patients with a higher median NIHSS score (18 vs. 14) and more patients with atrial fibrillation (increasing their risk for larger emboli). As such, the results were considered promising for potential benefit over IV tPA alone and as warranting further investigation.

Notably, there was a trend toward significance for improvement in neurologic outcome at 90 days in the subgroup of patients treated within the first 3 to 4 hours of onset compared with those treated more than 4 hours later, affirming the association between earlier revascularization and outcome.

IMS II (2007, N=81)[15]

The second IMS trial employed an identical protocol to IMS I, with the addition of an investigational EKOS microinfusion catheter. The catheter combines a traditional targeted drug infusion mechanism with a low-energy ultrasound in order to facilitate penetration of tPA into the thrombus and theoretically enhance thrombolysis. Of the 81 enrolled patients, ultrasound was activated for use in 33 patients (40.7%). The investigators also aimed to further demonstrate the results of IMS I while plans for a phase III trial were ongoing.

Whereas there was an increase in the incidence of ICH in IMS II compared with IMS I patients (9.9% vs. 6.3%, respectively), the results were not significantly different for hemorrhage, neurologic outcome, revascularization rate, or mortality. However, again demonstrated was a significant improvement over the placebo arm of the NINDS trial, supporting direct comparison between a combined IA/IV approach and IV alone.

MERCI (2005 N=151, 2008 N=164)[19,20]

The Mechanical Embolus Removal in Cerebral Ischemia (MERCI) trial evaluated the efficacy and safety of a mechanical endovascular device (which consists of a Nitinol core wire

with shaped loops at the distal end designed to engage the thrombus) in the revascularization of an occluded large cerebral vessel within 8 hours of stroke onset. The original study was subsequently updated by the Multi MERCI trial incorporating a newer generation of the device.[20] The population of patients included in both studies were those ineligible to receive IV tPA (either arriving too late or having a contraindication for its use). The MERCI trial was a multicenter, prospective, single-arm trial that referred to the control arm of the PROACT II trial as the historical control.

Although the therapeutic window was 8 hours, the median time from onset to treatment was 4.3 hours in both studies. The recanalization rate was significantly higher than that in the control arm of PROACT II (48% vs. 18%), but lower than that in the IA tPA group (66%). The proportion of patients achieving mRS≤2 at 90 days was comparable with that in the control arm of PROACT II (22.6% vs. 25%). On the other hand, the mortality rate was significantly higher than that reported in most prospective studies of the treatment of acute stroke, at 43.5%. The investigators attributed this elevated mortality to a higher baseline NIHSS score (median 18), and a higher proportion of basilar artery and internal carotid artery terminus occlusions. The introduction of a new generation of the device in the Multi MERCI trial resulted in a significantly higher recanalization rate (55%) and an improvement in the mortality or neurologic outcome at 90 days, although it was not statistically significant.

Penumbra (2009, N=125)[21]

The Penumbra device was introduced in the United States in 2008 after results from a European safety trial were published. The system consists of a thrombus debulking and aspiration component as well as a direct thrombus extraction component. The safety and efficacy of the device were reported in a multicenter, prospective single-arm trial.

Although the revascularization rate using the Penumbra device was the highest of the prospective trials at 81.6% (compared with 55% in the Multi MERCI trial and 66% in the PROACT II trial), the neurologic outcome was comparable or lower (25% with mRS≤2, vs. 36% in the Multi MERCI trial and 40% in the PROACT II trial). The investigators attribute this disparity to the lack of sufficient power and higher baseline NIHSS score.[17,19–20] Further studies are under way to better define outcomes with this promising device.

SARIS (2009, N=20)[23]

The use of endovascular stents has been recently advocated in the setting of acute stroke.[23] Based on retrospective data regarding the use of intracranial stents as a salvage technique in acute stroke, the SARIS trial (Stent-Assisted Recanalization in Acute Ischemic Stroke) aimed to evaluate the safety of stent deployment as a primary therapeutic intervention for acute stroke using a prospective, single-arm study design. The investigators used the Wingspan (Boston Scientific, Natick, MA) and the Enterprise (Cordis. Bridgewater, NJ) intracranial self-expanding stents.

All 20 cases included in the trial were successfully revascularized, although 60% of the patients required the use of an adjuvant intraprocedural pharmacologic infusion (eptifibatide or tPA) or angioplasty. The risk of subsequent asymptomatic and symptomatic hemorrhage was 10% and 5%, respectively, and no procedure-related complications were reported. With regards to neurologic outcomes, 45% of patients achieved an mRS score of 1 or less at 1-month follow-up. The results of the SARIS trial are difficult

to directly compare with the large, randomized prospective trials, because it included a small number of patients with a relatively low median NIHSS score (13 vs. 19 in IMS II and 17 in PROACT II), was nonrandomized, and included only a short follow-up; nonetheless, the results represent an encouraging foundation for further investigation into the role of stenting for stroke treatment.

CASE EXAMPLES

Case 1: Left M1 Occlusion – angioplasty

The patient is an 80-year-old right-handed female with a history of hypertension and hypercholesterolemia who awoke with acute onset of aphasia and right-sided weakness. She was brought to the emergency room, admitted to the neurology service, and started on antiplatelet agents. An MRI of the brain 24 hours after admission showed left basal ganglia stroke and a left M1 occlusion (Figures 10–2 and 10–3). The patient's right motor function became pressure-dependent and therefore mechanical endovascular intervention was requested to open the left M1. An M1 angioplasty was performed with successful recanalization of the vessel (Figures 10–4 and 10–5). Her exam improved significantly after angioplasty. She was discharged home after return to her baseline neurologic condition.

Case 2: Left M1 Occlusion: Thrombolysis and Thrombectomy – MERCI

The patient is a 76-year-old female who developed aphasia and right-sided weakness after endovascular repair of a thoracic aortic aneurysm. An MRI of the brain showed restricted diffusion of the left caudate and lentiform nuclei (Figure 10–6). She was taken to the angiography suite within 8 hours of the onset of her symptoms for cerebral angiography with possible intervention. A cerebral angiogram demonstrated left M1 occlusion (Figures 10–7 and 10–8). Left M1 thrombolysis and thrombectomy were performed with the MERCI device (Figure 10–9). The cerebral angiogram after thrombolysis and thrombectomy demonstrated

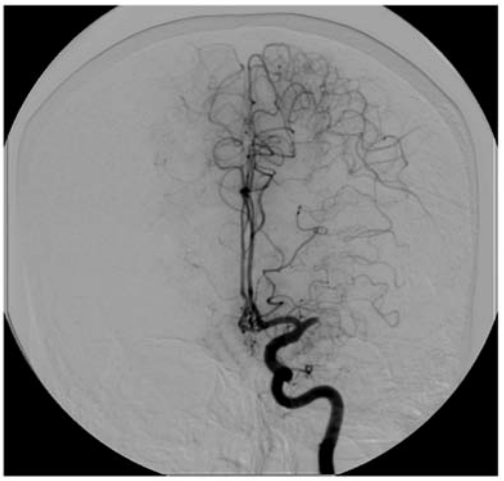

Figure 10-2. Cerebral angiogram. AP view demonstrating left M1 occlusion.

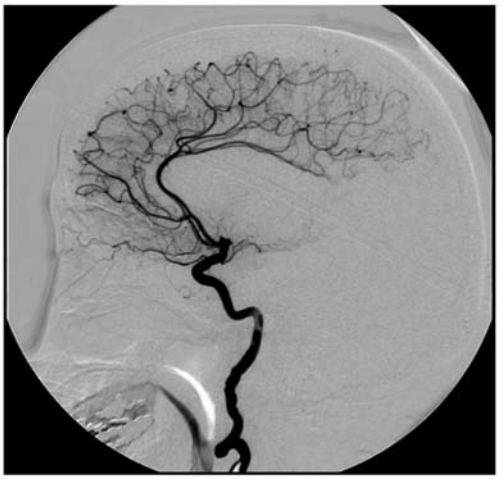

Figure 10-3. Cerebral angiogram. LICA injection - Lateral view demonstrating left M1 occlusion.

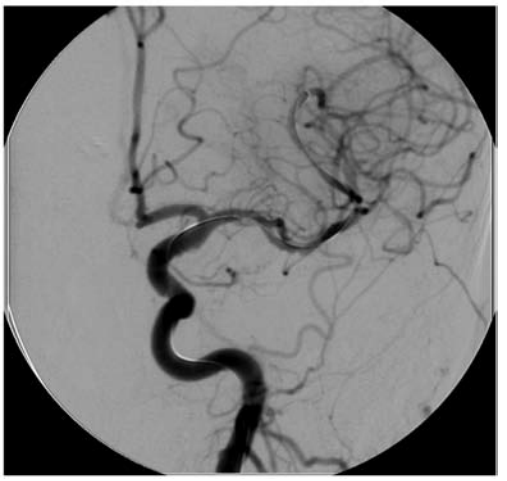

Figure 10-4. Cerebral angiogram. LICA injection - AP view during left M1 angioplasty.

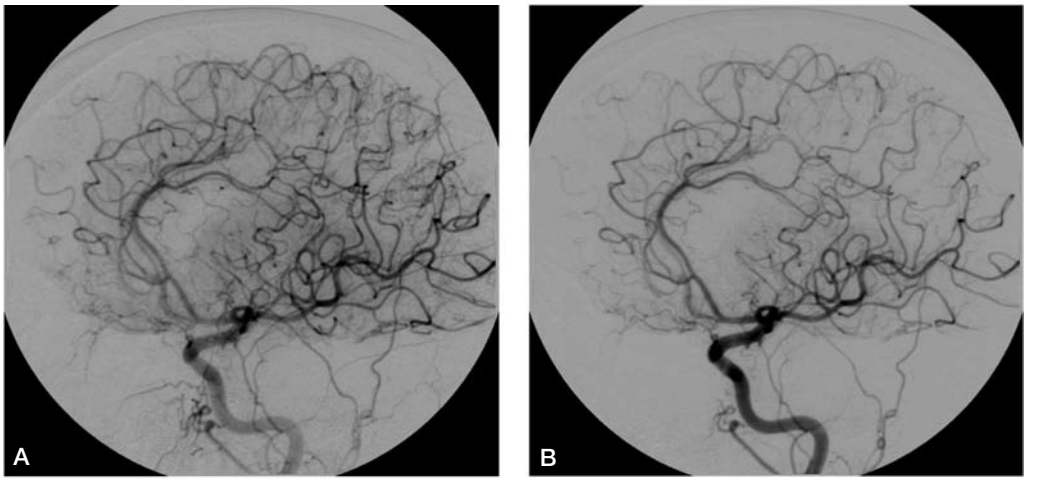

Figure 10-5. Cerebral angiogram. LICA injection - Lateral views post successful left M1 angioplasty demonstrating restored flow.

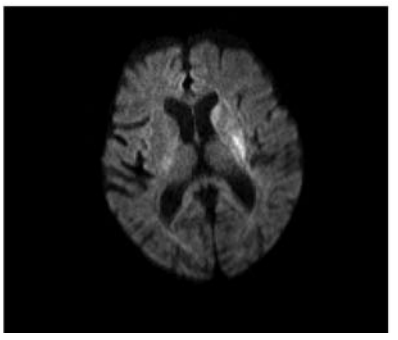

Figure 10-6. MRI brain diffusion weighted imaging (DWI) sequence showing restricted diffusion of left caudate and lentiform nuclei.

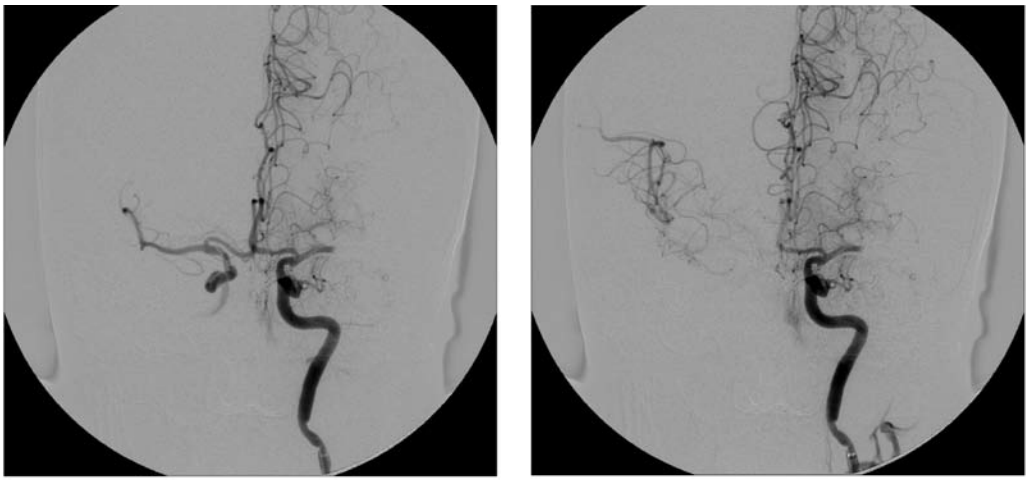

Figure 10-7. Cerebral angiogram. LICA injection - AP views demonstrating left M1 occlusion.

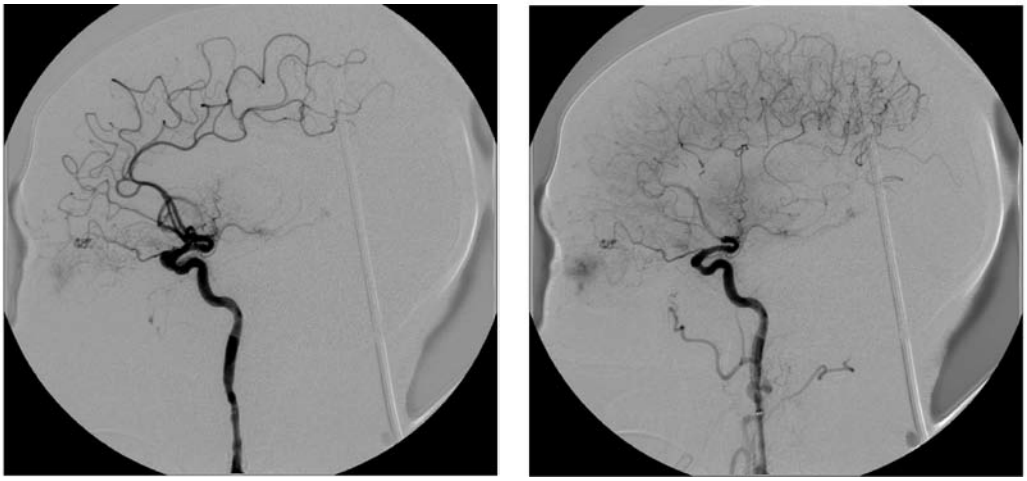

Figure 10-8. Cerebral angiogram. LICA injection lateral views demonstrating left M1 occlusion.

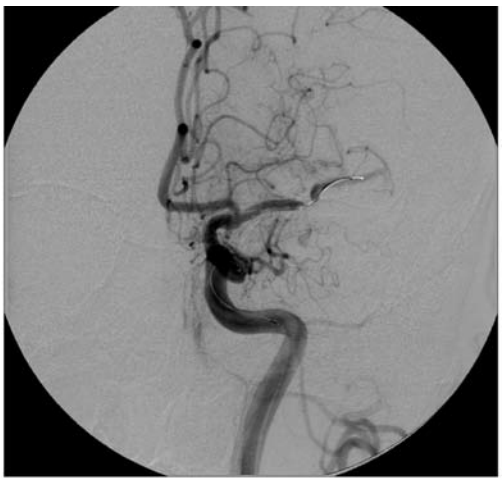

Figure 10-9. Cerebral angiogram. LICA injection AP view during mechanical thrombolysis.

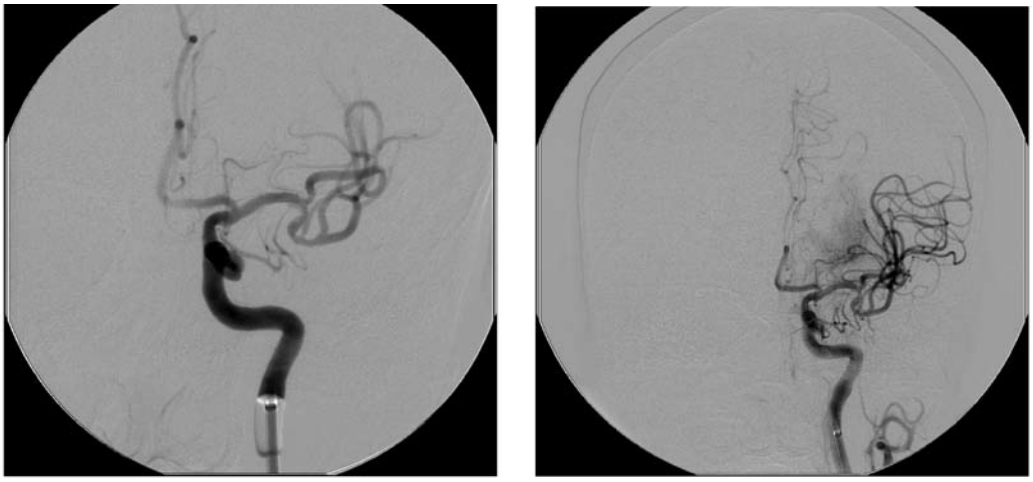

Figure 10-10. Cerebral angiogram. LICA injection AP views post mechanical thrombolysis demonstrating restored flow and successful left M1 thrombolysis.

restored flow in the distal left MCA (Figures 10–10 and 10–11). Postprocedurally, she recovered significant strength in her right arm and leg and showed significant improvement of her speech.

Case 3: Basilar Artery Occlusion – Mechanical and Chemical (tPA) Thrombolysis

The patient is a 43-year-old female who declined neurologically 1 day after a motor vehicle accident that resulted in a comminuted C1 left lateral mass fracture. Computed tomography (CT) angiography revealed a basilar artery thrombosis. MRI revealed areas of ischemia and infarction in the pons, right posterior occipital lobe, and left cerebellum (Figure 10–12).

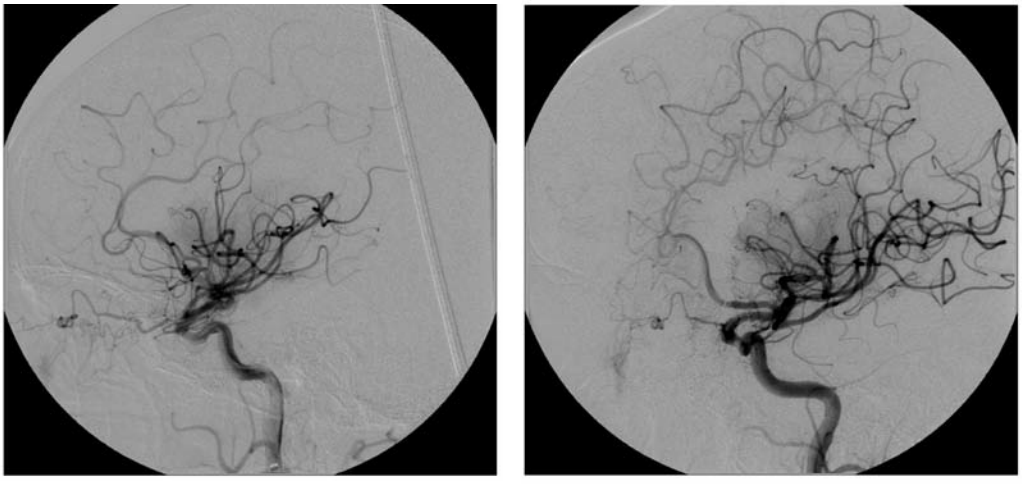

Figure 10-11. Cerebral angiogram. LICA injection lateral views demonstrating restored flow after successful left M1 thrombolysis.

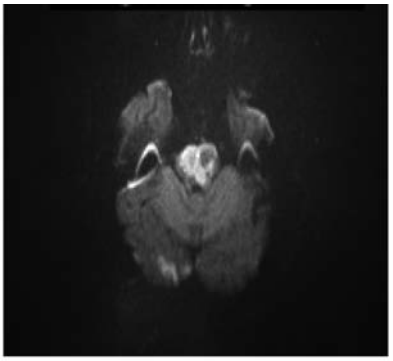

Figure 10-12. MR DWI sequence demonstrating areas of restricted diffusion consistent with infarction in the pons and right occipital lobe.

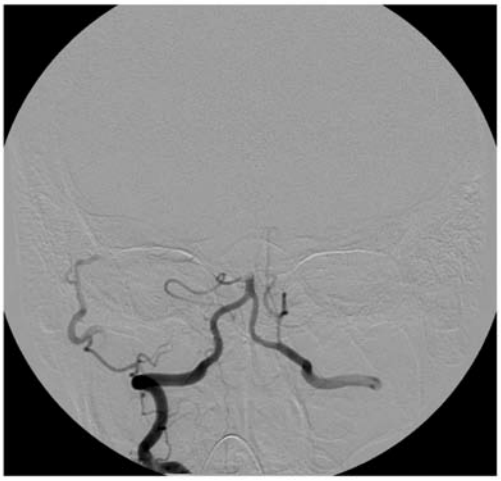

Figure 10-13. Cerebral angiogram. RVA injection AP view demonstrating basilar artery occlusion.

The patient was transferred to our institution for intervention. At the time of transfer, she had significant weakness of all extremities, right third nerve palsy, and left sixth nerve palsy. Cerebral angiogram demonstrated basilar artery occlusion (Figures 10–13 and 10–14). Chemical and mechanical thrombolysis of the basilar artery was performed with the Merci device. Cerebral angiography performed after thrombolysis showed restored flow in the basilar artery (Figures 10–15 through 10–17). At 1-year follow-up, the patient was independent with an excellent neurologic exam.

Case 4: Right Supraclinoid ICA Occlusion – Stent Placement

The patient is a 47-year-old female who presented with mild left-sided weakness. Three days after admission she developed increasing left-sided weakness and lethargy. An MRI showed

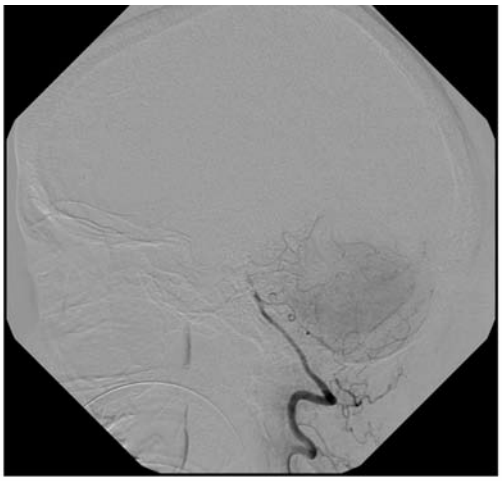

Figure 10-14. Cerebral angiogram. RVA injection AP view demonstrating basilar artery occlusion.

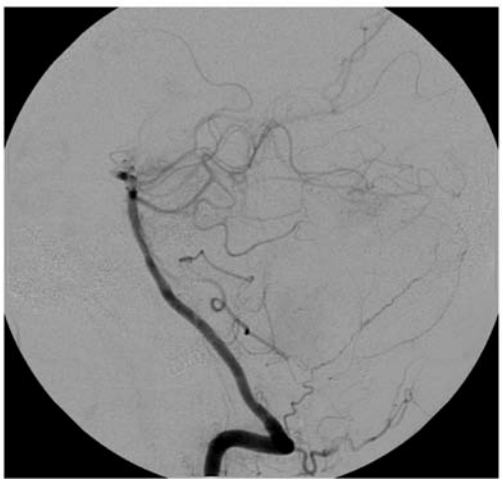

Figure 10-15. Cerebral angiogram. RVA injection lateral view post mechanical thromobolysis.

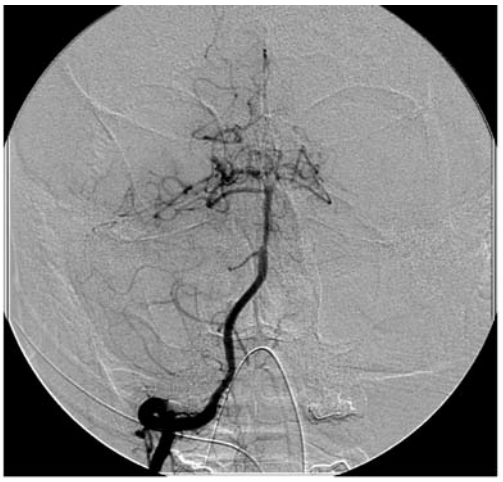

Figure 10-16. Cerebral angiogram. RVA injection AP view post mechanical and chemical thromoblysis demonstrating restored basilar artery flow.

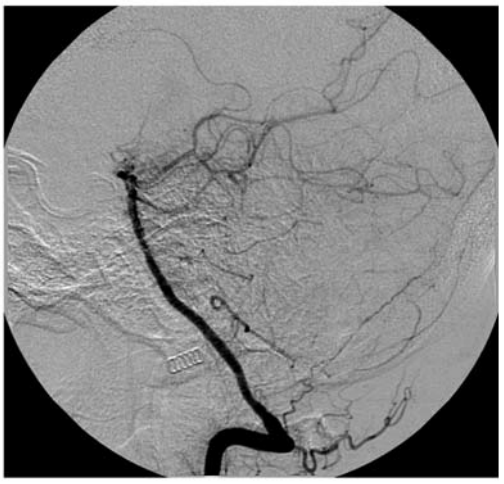

Figure 10-17. Cerebral angiogram. RVA injection lateral view post mechanical and chemical thrombolysis demonstrating restored flow in the basilar artery.

new right hemisphere infarction (Figure 10–18). A perfusion study showed severe hypoperfusion of the hemisphere (beyond areas of acute infarction, i.e., a large perfusion- diffusion mismatch). Cerebral angiography showed significant narrowing of the supraclinoid right internal carotid artery (ICA) (Figure 10–19). The patient was taken to the angiography suite and underwent stenting of the supraclinoid ICA (Figures 10–20 and 10–21). Cerebral angiography post-stenting demonstrated restored flow of the supraclinoid ICA (Figure 10–22). Repeat cerebral angiography 6 months post-stenting demonstrated patency of the supraclinoid right ICA without in-stent thrombosis or stenosis (Figures 10–23 and 10–24).

FUTURE DIRECTIONS

Despite recent advances, acute stroke continues to be a source of considerable morbidity, mortality, and disability. Continued improvement in outcomes will require improved and

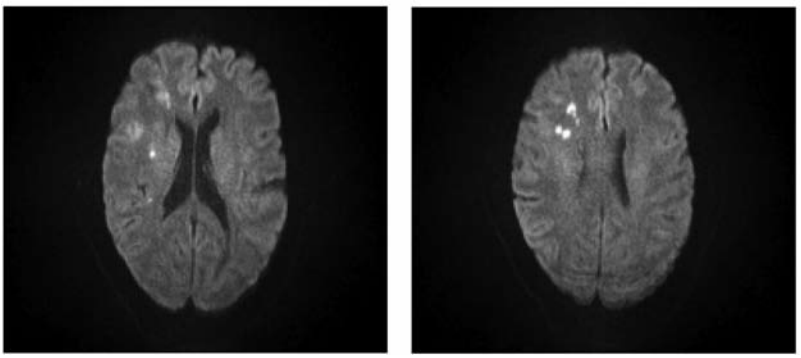

Figure 10-18. MRI brain. B1000 sequence. Acute infarcts in anterior division R MCA distribution in right frontal lobe white matter.

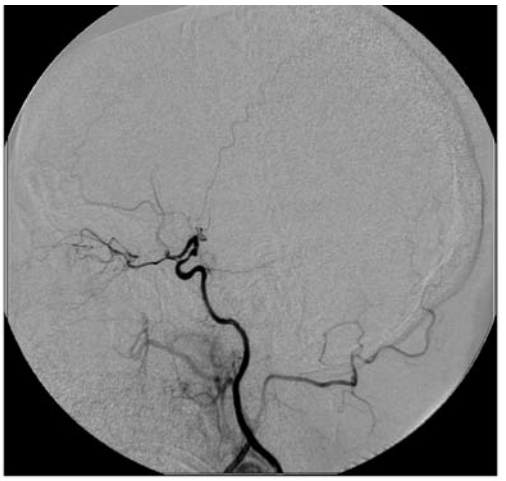

Figure 10-19. Cerebral angiogram. RICA injection lateral view demonstrating right supraclinoid ICA high grade narrowing secondary to dissection.

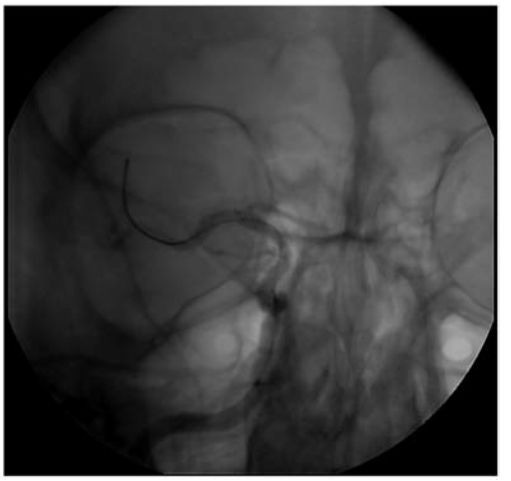

Figure 10-20. Cerebral angiogram. RICA injection AP view during stent deployment.

timely access of patients to treatment as well as refined patient selection for treatment. Furthermore, the multiplicity of available strategies requires investigation into their potential combinations. Interestingly, recent studies have suggested that a majority of patients presenting with stroke are unable to receive IV tPA, most frequently because they do not arrive soon enough.[22] This emphasizes the importance of improving currently available strategies in order to extend the therapeutic window.

With regard to imaging, computed tomography (CT) is most commonly used to evaluate acute stroke patients for both ischemia and hemorrhage. However, more recent studies suggest that gradient recall echo magnetic resonance imaging (GRE MRI)

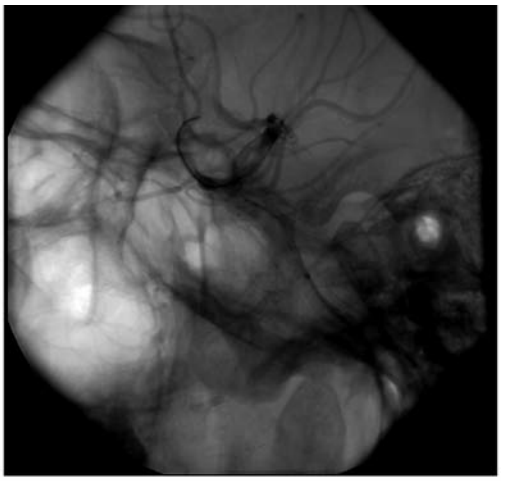

Figure 10-21. Cerebral angiogram. RICA injection lateral view during stent deployment.

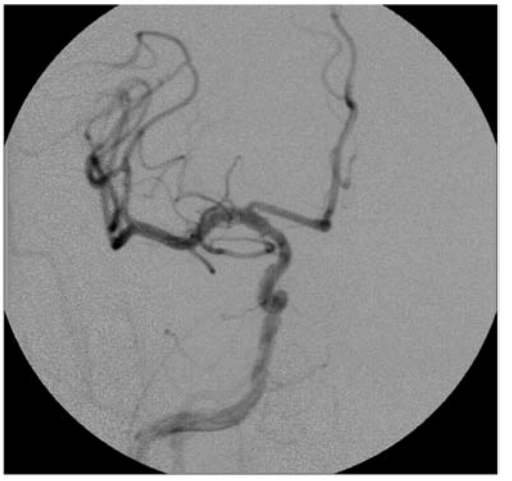

Figure 10-22. Cerebral angiogram. RICA injection AP view immediately post stent deployment demonstrating restored flow of supraclinoid ICA.

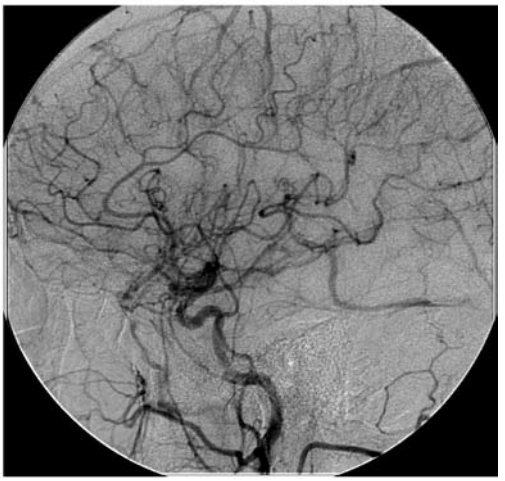

Figure 10-23. Cerebral angiogram 6 months post stent deployment. RICA injection lateral view demonstrating patency of stented supraclinoid ICA.

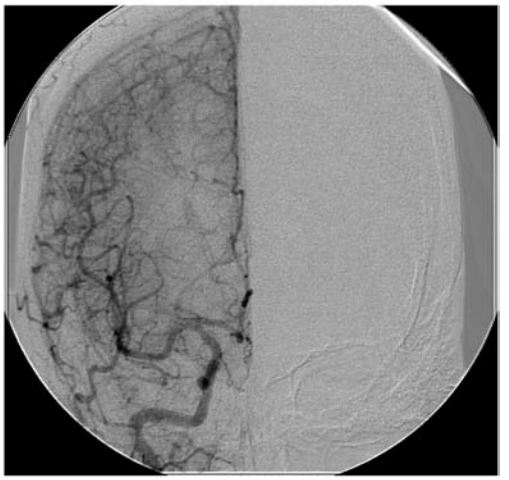

Figure 10-24. Cerebral angiogram 6 months post stent deployment. RICA injection AP view demonstrating patency of stented supraclinoid ICA.

may be a more specific modality for the detection of acute hemorrhage.[23] Incorporation of improved imaging techniques in future studies may provide a more accurate assessment of the risk of hemorrhage and hemorrhagic conversion following reperfusion, especially for patients with early hemorrhage who may be at risk by receiving thrombolytics.

The currently enrolling IMS III trial is a large, international, multicenter, prospective randomized control trial (Phase III) that will enroll a projected 900 subjects within 3 hours of stroke onset. The trial aims to compare combined IV/IA approaches with IV tPA alone. The highly anticipated results of this trial will provide sound scientific evidence allowing direct comparison between a combined approach and the standard IV approach.

In addition to the treatment of acute stroke, there has been recent interest in applying these treatment modalities to the treatment of subacute stroke (>8 hours of window). We recently published a report of two cases of subacute vascular occlusion with persistent ischemic symptoms referable to the affected ischemic territory that were successfully treated with angioplasty with no residual neurologic deficits.[24] The benefit of restoration of vessel patency in the setting of subacute occlusion and the use of other endovascular strategies such as mechanical thrombectomy may be interesting venues for future investigation.

REFERENCES

1. Bruckmann H, Ferbert A, Del Zoppo G, et al. Acute vertebral-basilar thrombosis. Angiologic-clinical comparison and therapeutic implications. Acta Radio Suppl 1986;369:38.
2. Brandt T, von Kummer R, Muller-Kuppers M, Hacke W. Thrombolytic therapy of acute basilar artery occlusion variables affecting recanalization and outcome. Stroke 1996;27: 875–81.
3. Hacke W, Schwab S, Horn M, et al. 'Malignant' middle cerebral artery territory infarction: clinical course and prognostic signs. Archiv Neurol 1996;53:309–15.

4. Jansen O, Von Kummer R, Forsting M, et al. Thrombolytic therapy in acute occlusion of the intracranial internal carotid artery bifurcation. AJNR Am J Neuroradiol 1995;16:1977–86.
5. Hacke W, Donnan G, Fieschi C, et al. Association of outcome with early stroke treatment: pooled analysis of ATLANTIS, ECASS, and NINDS rt-PA stroke trials. Lancet 2004;363:768.
6. Hacke W, Zeumer H, Ferbert A, et al. Intra-arterial thrombolytic therapy improves outcome in patients with acute vertebrobasilar occlusive disease. Stroke 1988;19:1216–22.
7. Suarez J, Sunshine J, Tarr R, et al. Predictors of clinical improvement, angiographic recanalization, and intracranial hemorrhage after intra-arterial thrombolysis for acute ischemic stroke. Stroke 1999;30:2094–100.
8. Fisher M, Prichard J, Warach S. New magnetic resonance techniques for acute ischemic stroke. JAMA 1995;274:908-11.
9. Staroselskaya I, Chaves C, Silver B, Linfante I, Edelman R, Caplan L, Warach S, Baird A. Relationship between magnetic resource arterial patency and perfussion-diffusion mismatch in acute ischemic stroke and its potential clinical use. Arch Neurol 2001;58:1069.
10. Tissue plasminogen activator for acute ischemic stroke. The national institute of neurological disorders and stroke rt-pa stroke study group. N Engl J Med 1995;333:1581–7.
11. Yamaguchi T, Hayakawa T, Kiuchi H. Intravenous tissue plasminogen activator ameliorates the outcome of hyperacute embolic stroke. Cerebrovasc Dis 1993;3:269–72.
12. Kidwell C, Wintermark M. Imaging of intracranial haemorrhage. Lancet Neurol 2008;7: 256–67.
13. Hacke W, Kaste M, Bluhmki E, et al. Thrombolysis with Alteplase 3 to 4.5 hours after acute ischemic stroke. N Engl J Med 2008;359:1317–28.
14. Combined intravenous and intra-arterial recanalization for acute ischemic stroke: The Interventional Management of Stroke Study. Stroke 2004;35:904–11.
15. The interventional management of stroke (IMS) II study. Stroke 2007;38:2127–35.
16. del Zoppo GJ, Higashida RT, Furlan AJ, et al. A phase II randomized trial of recombinant pro-urokinase by direct arterial delivery in acute middle cerebral artery stroke. PROACT investigators. Prolyse in acute cerebral thromboembolism. Stroke 1998;29:4–11.
17. Furlan A, Higashida R, Wechsler L, et al. Intra-arterial prourokinase for acute ischemic stroke. The PROACT II study: A randomized controlled trial. Prolyse in acute cerebral thromboembolism. JAMA 1999;282:2003–11.
18. Lewandowski C, Frankel M, Tomsick T, et al. Combined intravenous and intra-arterial r-tPA versus intra-arterial therapy of acute ischemic stroke emergency management of stroke (EMS) bridging trial. Am Heart Assoc 1999;30:2598–2605.
19. Smith W, Sung G, Starkman S, et al. Safety and efficacy of mechanical embolectomy in acute ischemic stroke results of the MERCI trial. Am Heart Assoc 2005;36:1432–8.
20. Smith W, Sung G, Saver J, et al. Mechanical thrombectomy for acute ischemic stroke: final results of the multi MERCI trial. Stroke 2008;39:1205.
21. The penumbra pivotal stroke trial. Safety and effectiveness of a new generation of mechanical devices for clot removal in intracranial large vessel occlusive disease. Stroke 2009;40: 2761–8.
22. Barber P, Zhang J, Demchuk A, et al. Why are stroke patients excluded from tPA therapy? An analysis of patient eligibility. Neurology 2001;56:1015–20.
23. Levy E, Siddiqui A, Crumlish A, et al. First Food and Drug Administration-Approved Prospective Trial of Primary Intracranial Stenting for Acute Stroke. SARIS (Stent-Assisted Recanalization in Acute Ischemic Stroke). Stroke 2009.
24. Gross B, Hurley M, Bernstein R, et al. Endovascular recanalization for subacute symptomatic intracranial arterial occlusion: a report of two cases. Clin Neurol Neurosurg 2008; 110:1058–63.

11

Techniques of Carotid Angioplasty and Stenting

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Like carotid endarterectomy, carotid angioplasty and stenting (CAS) requires careful patient selection, thorough counseling, detailed procedural planning, and the highest level of surgical skill if results are to be optimized and complications reduced. The potential for devastating neurologic complications from flow disruption or embolic debris from CAS necessitates inscrutable endovascular technique and practical efficiency.[1-9] However, the basic principles of angioplasty and stenting learned from treating less risky arterial beds also apply in the carotid system. Secure arterial access, careful guidewire traversal of the target lesion, and precise balloon and stent selection are important for treating arterial stenoses, regardless of the vascular bed involved. In this chapter, we will describe a step wise process for performing carotid angioplasty and stenting. We acknowledge that many of our selections represent personal bias and that other approaches have been used with equal success.

SHEATH ACCESS AND GUIDEWIRE POSITIONING

The first challenge in carotid artery interventions is gaining secure access to the common carotid artery. Depending on the arch anatomy, the positioning of a sheath into the common carotid artery may be the most technically difficult aspect of the procedure.

Patient Preparation/Positioning

In our practice, diagnostic and therapeutic cerebral vascular procedures are done in the angiography suite. Generally, a complete diagnostic carotid and cerebral angiogram has been previously performed. Satisfactory baseline images in multiple plains in the extracranial and intracranial cerebral circulation are important prior to intervention. The intracerebral images are necessary as a reference for comparison to

the cerebral runoff after CAS. Following successful femoral sheath insertion, the patient is systemically anticoagulated with intravenous heparin (70 units per kilogram). Additionally, in patients with significant risk for emboli and a contraindication to using an embolic protection device, use of an IIB/IIIA inhibitor, such as eptifibatide (Integrillin) or tirofiban (Aggrastat) may be considered.

Sheath Access of the Common Carotid Artery

The image intensifier is positioned so the aortic arch can be viewed in an oblique projection at approximately 25–30 degrees. If the proximal branch artery anatomy is known from a previous exam, then an arch aortogram is not repeated. An angled guide-catheter is our initial catheter of choice and can be used in conjunction with a 0.035 inch glidewire to cannulate the innominate artery and right common carotid arteries, as well as the left common carotid artery (Figure 11–1). If an angle glide-catheter is not successful, other shaped catheters such as a Simmons catheter may be used. The position of the glidewire must be monitored closely, and the carotid lesion should not be inadvertently crossed. The angled glide catheter is positioned in the proximal common carotid artery and the initial carotid and intracranial angiograms are obtained in both AP and lateral projections. Next, the image intensifier of the C-Arm is positioned so that the carotid bifurcation can be best demonstrated. The glide catheter, over the angled glidewire, is advanced into a branch of the external carotid artery (Figure 11–2). This glidewire is then exchanged for a 0.035-inch Amplatz exchange length guidewire. Over the Amplatz wire, a 90cm 6 or 7 French sheath (either Shuttle or Destination) is advanced into the common carotid artery (Figure 11–3). The radiopaque tip of the sheath should be securely placed within the common carotid

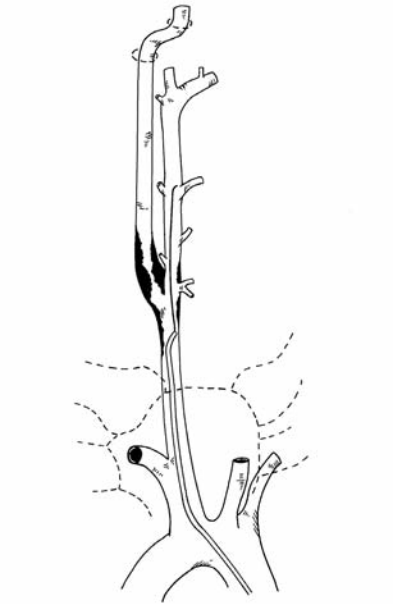

Figure 11-1. Common carotid access.

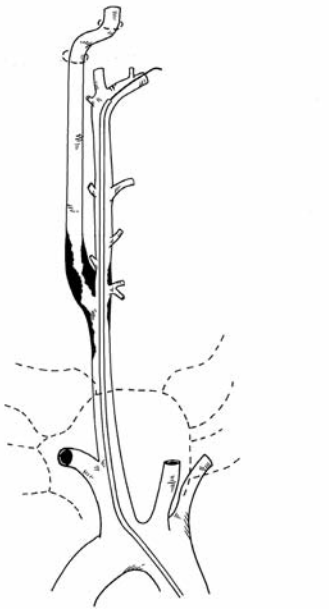

Figure 11-2. External carotid artery access with guidewire position in the distal external carotid artery.

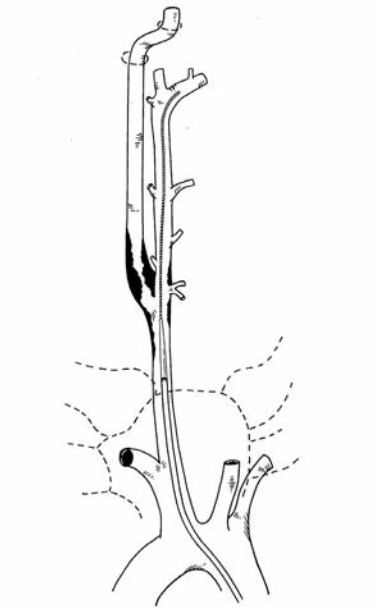

Figure 11-3. Sheath access in common carotid artery.

artery above the patient's clavicle. Care is taken to retract the obturator of the sheath as the sheath is simultaneously advanced the last 5cm toward the carotid bifurcation.

Ongoing neurologic assessment is a part of the CAS procedure and a neurologic evaluation should be performed after sheath access. A horn or other noise-making device placed in the contralateral hand can be squeezed throughout the procedure to access the patient's motor function and capacity to follow commands. A standard series of questions can be asked in order to evaluate speech and cognitive abilities.

CAROTID ANGIOPLASTY AND STENT PLACEMENT

Preparation for Angioplasty

Once the obtrator for the sheath is completely removed, a 0.014-inch guidewire or embolic protection device is advanced across the internal carotid stenosis with the tip of the wire placed within the distal internal carotid artery (Figure 11–4). The Amplatz wire used for sheath delivery may be removed first, or alternatively, the 0.014-inch wire may be advanced through the sheath next to the Amplatz that is partially obstructing the origin of the external carotid. If an embolic protection device is not indicated, a catheter may be used to direct the 0.014-inch wire into the origin of the internal carotid. A 4 French 120cm glide catheter is used rather than the standard 100cm catheter, because once the guiding sheath is placed, the added length of the glide catheter is necessary for any further catheter manipulations or wire exchanges.

We typically use a EPI filter wire, but there are other cerebral protection devices available. Each of these cerebral protection devices has specific procedures for placement and removal that should be reviewed prior to their selection and use.

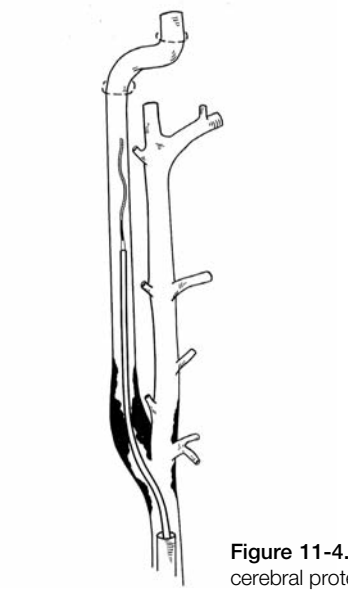

Figure 11-4. Internal carotid artery access with cerebral protection device prior to deployment.

Carotid Angioplasty

Since it is not uncommon for patients to have bradycardia and hypotension with carotid angioplasty, hemodynamic assessments are made continuously throughout the procedure. In order to limit the bradycardia associated with angioplasty of native arterial lesions, the patient is given 0.5 to 1 mg of intravenous atropine. Also, in preparation for percutaneous transluminal angioplasty (PTA), a cerebral angiogram is performed in the anterior-posterior and lateral projections. A carotid angiogram is performed through the guiding sheath to further delineate the carotid bifurcation and the stenosis. The image intensifier is placed in an optimal position to separate the origins of the internal and external carotid arteries. PTA of the carotid stenosis is carefully performed with a 3mm or 4mm angioplasty balloon (Figure 11–5). This dilation is not intended to adequately treat the stenotic lesion, but rather is performed in order to establish an open track along the lesion through which the stent can be passed.

Stent Placement

The size of the predilatation angioplasty balloon in relation to the native artery is taken into account when deciding what size stent to use. Both the internal carotid artery and the common carotid artery diameters are important consideration when selecting the stent. We use self-expanding stents delivered on rapid exchange platforms such as the Wallstent and the PreciseRx. The stent is delivered through the sheath to the carotid artery proximal to the lesion. Puff arteriography is performed as the stent is advanced and positioned under fluoroscopic guidance. Deployment of the stent is performed in a manner that allows controlled and alternate adjustments as needed. A completion angiogram is performed through the sheath to access the stent placement. Residual stenoses may require post stent dilation with an appropriately sized balloon and rapid deflation is performed. We use compliant balloons on a low profile rapid exchange platform. This allows tailoring of the inflation pressure to achieve alternate

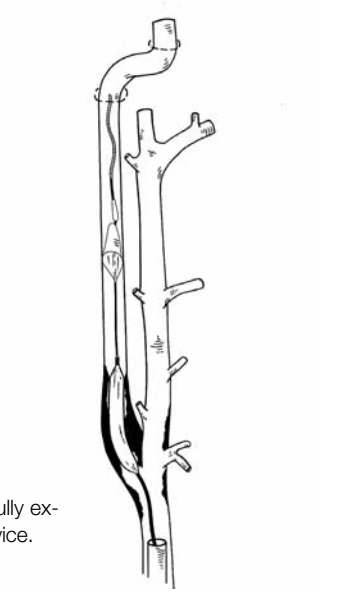

Figure 11-5. Carotid angioplasty with fully expanded cerebral embolic protection device.

balloon diameters under direct fluoroscopic visualization. A 30cc syringe is used to aspirate the sheath during balloon deflation, and blood pressure cuffs may be inflated on the arms to enhance vertebral prograde flow and promote reversal of flow through the internal carotid arteries to help eliminate any embolic debris through retrograde flow out of the sheath. Overdilatation is avoided as the risks of cerebral embolization or carotid rupture outweigh the consequences of modest residual stenoses in a calcified bifurcation.

COMPLETION

The cerebral protection device is recaptured using a retrieval sheath to collapse the filter prior to removal through the stent (Figure 11–6). Following stent deployment, if there is no flow or slow flow through the internal carotid artery, this should be addressed prior to recapture of the cerebral protection device. A completion angiogram is performed following stent deployment. This should include an AP and lateral view of the cervical region, as well as AP and lateral views of the intracranial anatomy. The preprocedure cerebral angiogram is used as a comparison. A neurologic assessment is performed following angioplasty and stent placement. Any deficit on the exam should prompt an even more thorough review of the completion angiogram. If there are no neurologic or technical issues, the catheters and guidewires are then removed. The sheath is then exchanged for a standard 6 F short sheath, and then this sheath is removed once the activated clotting time (ACT) is less than 150. We use the Perclose closure device in the majority of our patients at the completion of the procedure. Relative hypotension is a common complication of carotid bulb angioplasty and should be treated accordingly. Aggressive volume replacement is usually helpful but temporary infusion of vasopressors sometimes lasting for 12 to 24 hours may be needed.

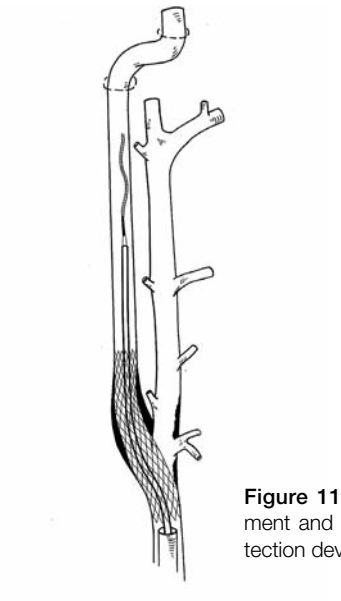

Figure 11-6. Carotid stent placement and recaptured cerebral protection device.

TECHNICAL TIPS

1. Always obtain a thorough baseline neurologic exam and good quality cerebral runoff images prior to carotid intervention. Both will be important for comparison following angioplasty and stenting if any neurologic issues arise.
2. Place Amplatz or similar stiff guidewire securely within the external carotid artery, but be very conscious of the tip of the wire during the sheath advancement. Arterial perforation is possible with inadvertent guidewire advancement.
3. Do not hesitate in placing the guiding sheath well into the common carotid artery. However, remain aware of the length of the sheath's introducer tip. During advancement of the sheath, the additional length of introducer beyond the sheath may be difficult to visualize and could disrupt an unstable carotid plaque if advanced inappropriately.
4. Predilate with a relatively small balloon in order to limit plaque disruption. An image of the contrast filled balloon is saved in order to compare its size to the native internal and common carotid arteries.
5. A mild residual stenosis is preferable to a negative embolic event. If postdilation of the stent is required, avoid repeated angioplasty. Also, do not over dilate and risk rupture of the carotid artery.

CONCLUSION

The basic principles of endovascular interventions developed for and used routinely in other vascular beds apply directly to performing carotid artery angioplasty and stenting. Stable and secure sheath access to the common carotid arteries is the essential first step, followed by careful crossing of the carotid artery lesion. Balloon dilation and

stent deployment are performed under fluoroscopic guidance with frequent puff arteriograms to provide ongoing information and allow accurate positioning. These steps require a technical familiarity with the arteries in the cervical region. The remainder of the procedure requires precise judgment in balloon and stent selection, as well as technical ability. The potential for neurologic and cardiovascular complications when performing percutaneous carotid artery stenting necessitate meticulous preparation and planning. As surgeons move to incisionless treatment of carotid artery disease, they will develop routines and preferences pertaining to the CAS procedure with which they have great confidence and produce consistent and excellent results. Carotid endarterecomy dogma will be replaced by carotid angioplasty and stenting dogma.

REFERENCES

1. Chakhtoura EY, Hobson RW, Goldstein J, et al. In-stent restenosis after carotid angioplasty-stenting: incidence and management. *J Vasc Surg*. 2001;33(2):220–225.
2. Criado FJ, Lingelbach JM, Ledesma DF, Lucas PR. Carotid artery stenting in a vascular surgery practice. *J Vasc Surg*. 2002;35(3):430–434.
3. Diethrich EB, Ndiaye M, Reid DB. Stenting in the carotid artery: initial experience in 110 patients. *J Endovasc Surg*. 1996;3(1):42–62.
4. Hobson RW, Goldstein JE, Jamil Z, et al. Carotid restenosis: operative and endovascular management. *J Vasc Surg*. 1999;29(2):228–235.
5. Mather A, Roubin GS, Iyer SS et al. Predictors of stroke complicating carotid artery stenting. *Circulation*. 1998;97(13):1239–1245.
6. Qureshi AI, Luft AR, Janardhan V, et al. Identification of patients at risk for periprocedural neurological deficits associated with carotid angioplasty and stenting. *Stroke*. 2000;31(2): 376–382.
7. Roubin GS, New G, Iyer SS, et al. Immediate and late clinical outcomes of carotid artery stenting in patients with symptomatic and asymptomatic carotid artery stenosis: a 5-year prospective analysis. *Circulation*. 2001;103(4):532–537.
8. Shawl F, Kadro W, Domanski MJ, et al. Safety and efficacy of elective carotid artery stenting in high-risk patients. *J Am Coll Cardiol*. 1000;35(7):1721–1728.
9. Wholey MH, Wholey, MH, Jarmolowski CR, et al. Endovascular stents for carotid artery occlusive disease. *J Endovasc Surg*. 1997;4(4):326–338.

12

Endovascular Management of Supra-Aortic Trunk Lesions

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INTRODUCTION

The evolution of the endovascular management of supra-aortic trunk lesions has given vascular surgeons a less invasive, lower-risk, but durable method of treating aortic arch vessel disease. In this section, a historical review of the various surgical approaches for the correction of supra-aortic trunk lesions is presented, with their published success and complication rates. The treatment paradigm of both symptomatic and asymptomatic aortic arch vessel athero-occlusive disease is reviewed, after which the technical aspects of the endovascular correction of these lesions are briefly discussed. In addition, the published literature of the endovascular treatment of supra-aortic trunk lesions is reviewed.

EVOLUTION OF TREATING SUPRA-AORTIC TRUNK DISEASE

Surgical Reconstruction

The treatment of atherosclerotic disease of the common carotid, subclavian, and innominate arteries, collectively known as the aortic arch vessels, has evolved with the advances made in the correction of other vascular lesions. In 1856, the first report of symptomatic subclavian artery occlusion appeared in the literature.[1] Nearly one century later, the surgical repair of a supra-aortic trunk lesion was reported.[2] Shortly thereafter, others reported on the performance of a trans-thoracic endarterectomy of the innominate artery; however this operation—at the time—was associated with a mortality rate of 22%[3,4] and a major complication rate of 15 - 26%[7]. A more contemporary series by Berguer et al of 100 consecutive trans-thoracic repairs of supra-aortic trunk lesions showed a combined stroke/death rate reduced to 16%.[5]

In 1957, Lyons and Galbraith described the first carotid-subclavian bypass and a year later, Debakey et al introduced the use of prosthetic grafts to bypass thrombo-obliterative lesions of the aortic arch vessels.[6,7] A decade elapsed before this approach was more broadly utilized and reported[8]. Nevertheless, this approach resulted in an improvement in the mortality rate (5.6%) as compared to the trans-thoracic repair[4] The excellent results of extra-anatomic repair of aortic arch vessels have been further substantiated in subsequent series, making it the standard to which other interventions are compared. Berguer et al reported a 5- and 10-yr primary patency rate of 91% and 82% respectively, with a mortality rate of 0.5% and stroke rate of 3.8% in 100 consecutive cervical reconstructions of the supra-aortic trunk.[9] Similarly, Perler et al reported primary patency rates of 92% and 83% at 5 and 8 years with an extra-anatomic approach.[10]

Extra-anatomic bypass grafting, however, has several associated specific major complications: 1) thoracic duct fistulae; 2) Horner's syndrome; 3) brachial plexus nerve injury; and 4) myocardial infarction. Additionally, skin erosion and infection of the graft, encumbrance of future coronary bypass grafting, and failure to exclude atherosclerotic lesions with future embolic potential, are disadvantages of this approach.

Endovascular Options

Mathias published the first report of percutaneous transluminal angioplasty of aortic arch vessels in 1980.[11] In that same year, Bachman and Kim introduced subclavian artery angioplasty.[12] Subsequent reports demonstrated the safety, efficacy, and efficiency of angioplasty of supra-aortic lesions. In a review of 10 reported series and a total of 423 subclavian and innominate artery angioplasties, there was a 92% initial technical success rate, with a 19% recurrence rate between 1 and 5 years of follow-up.[13] Similarly, Kachel et al in a review of 774 supra-aortic artery lesions, documented a technical success rate of 95.3%, with a 4.0% complication rate.[14]

Stenting of the aortic arch vessels was introduced in the early 1990s after the favorable results of stenting used as an adjunct to balloon angioplasty of other vascular lesions. Several series[15–17] demonstrated good initial success with stenting of supra-aortic trunk lesions, with acceptable patency rates, and low complication rates. Theoretically, stents have the potential to enmesh atheroemboli, promote laminar flow to reduce restenosis, and restrict arterial recoil. Subsequent reports have documented the cost benefit of an endovascular approach to the treatment of arch vessel disease.[18] Endovascular stenting of aortic arch vessels continues to evolve as new techniques and devices are developed. More specific details of this approach are outlined later in the chapter.

ETIOLOGY

Multiple inflammatory and infectious disease processes produce stenotic lesions of the supra-aortic trunk, including atherosclerotic disease, Takayasu's arteritis, and radiation-induced atherosclerosis obliterans.

Atherosclerotic occlusive disease, the most common etiology of supra-aortic trunk lesions of the aortic arch vessels, is relatively rare when compared to other vascular lesions. In a report on surgery for aortic arch branch occlusion, 1.7% of 1961 operations

were performed on the innominate artery, while 4.3% of cases were performed for occlusive lesions of the subclavian arteries.[19]

In the Joint Study on Extracranial Arterial Occlusion, 1/3 of patients were found to have severe lesions of the supra-aortic trunk by arteriography;[20] 17% of these lesions are located in the innominate and proximal subclavian arteries,[19] of which the left subclavian artery is involved 3-4 times more often than the right.[21] In a report by Brountzos et al, 34 of 39 subclavian artery lesions were left-sided.[22] A 1.8% incidence of isolated proximal common carotid artery stenosis has been reported, while a "tandem" lesion of both the carotid bifurcation and common carotid artery has been found in 0.6% of patients undergoing evaluation of cerebrovascular insufficiency[23].

Atherosclerotic disease of the supra-aortic trunk affects relatively young patients when compared with other vascular lesions.[24] However, significant comorbidities are present in this patient population. In a report of 18 patients undergoing endovascular treatment of atherosclerotic lesions of the aortic arch vessels, coronary artery disease (CAD) was present in 78%, carotid disease in 33%, 44% were diabetic, 67% had a smoking history, and hypertension was present in 61%.[25] Similarly, Brountzos et al reported CAD in 52% of patients undergoing percutaneous revascularization of the arch vessels, while 30% had associated disease of the carotid and/or vertebral arteries.[22]

Takayasu's arteritis, a cell-mediated inflammatory process which produces segmental fibrotic stenosis of the aorta and its branches, is the second most common cause of supra-aortic occlusive disease. It has a prevalence of 1 per 1000 people in the US, and 6 per 1000 people worldwide.

Although short-term outcomes of PTA and stent procedures to treat Takayasu-related stenoses have been favorable, long-term follow-up has been less encouraging. In their study of patients with Takayasu's arteritis, Liang et al reported the occurrence of restenosis or occlusion in 3 of 7 PTA procedures, and 5 out of 7 stent procedures during the follow-up period. The authors attributed the low stent patency rate in Takayasu's arteritis to the long, noncompliant fibrotic lesions, which may not fully dilate, even under injurious inflation pressures.[26] In a separate study of 4 patients with Takayasu's disease who had undergone stenting of the subclavian or common carotid artery, 10 of 11 stents were occluded at a mean follow-up of 12 months.[27]

Radiation-induced atherosclerosis obliterans is a rare cause of supra-aortic trunk lesions, which are most frequently reported after radiotherapy for breast cancer.[28,29] In a retrospective review, which included 11 centers, 64 patients with radiation-induced supra-aortic trunk disease who had undergone surgical and endovascular reconstruction were identified. Thirteen patients had angioplasty with stent placement of the common carotid or innominate artery performed. Although no strokes or operative mortality were reported, one restenosis was observed (mean follow-up: 18 months).[30]

INDICATIONS FOR TREATMENT

In general, surgical intervention is indicated when supra-aortic trunk lesions become symptomatic. Symptoms are produced by hypoperfusion of or embolization to the cerebral and upper extremity vascular beds. Lesions of the brachiocephalic trunk may present with upper limb ischemia, digital embolization, and vertebrobasilar insufficiency, manifested by visual disturbances, vertigo, syncope, dysarthria, dysphagia, and ataxia. In a

series of 48 patients undergoing stenting of subclavian and innominate arteries, 16.6% of patients exhibited vertebrobasilar insufficiency, 31.3% had upper limb ischemia, while 12.5% had both cerebral and upper limb symptoms.[22] Sullivan et al reported that 50% of patients with symptomatic occlusive disease of the innominate artery presented with anterior circulation symptoms, 40% exhibited vertebrobasilar symptoms, while 10% had a combination of symptom complexes.[15]

Not surprisingly, between 0.5% and 1.1% of patients undergoing coronary revascularization have evidence of subclavian artery stenosis.[31–33] Patients with a prior history of internal mammary artery-coronary bypass grafting may present with angina and in fact, 27 of 83 patients treated for subclavian lesions at the Cleveland Clinic presented with symptoms of internal mammary steal, of which 11 had unstable coronary syndromes.[15]

In addition, patients with an axillary-femoral bypass may exhibit signs of lower limb ischemia from an occlusive lesion of the subclavian artery. Treatment of subclavian artery lesions may also be indicated in patients scheduled to undergo other revascularization procedures. In a series of 48 patients undergoing stenting of subclavian and innominate artery occlusive disease, 12.5% presented with angina before or after LIMA-coronary bypass, while 10.4% complained of leg claudication before or after axillary-femoral bypass grafting.[22]

Treatment of common carotid artery occlusive disease is indicated to improve inflow prior to ipsilateral carotid endarterectomy and bypass procedures involving the carotid arteries. Other indications for surgical intervention of common carotid lesions are non-debilitating stroke with good recovery or TIAs, amaurosis fugax, or critical stenosis of ≥ 75% in an asymptomatic individual with a patent internal carotid artery.[16] Although level I evidence is lacking, many would extrapolate and agree that a symptomatic lesion of ≥ 50% or an asymptomatic arch vessel lesion of ≥ 80% should be corrected either by traditional open surgery or endoluminal therapy. The only caveat is with regard to asymptomatic lesions of the left subclavian artery and whether they should be treated; this remains a hotly debated topic.

IMAGING STUDIES

Digital subtraction angiography (DSA) remains the gold standard for imaging of the aortic arch vessels. However, radiation exposure, the use of nephrotoxic dye and complications related to the arterial puncture and catheterization are associated with this technique. In addition, there is a potential for stroke with this procedure. As reported by NASCET and ACAS, the risk of stroke with cerebral angiography is 0.7% and 1.2% respectively.[34,35]

Recently, the use of MRA in the detection, interventional planning, and follow-up of supra-aortic trunk lesions has emerged. When compared to DSA, MRA has a sensitivity and specificity for determining lesion severity of 73%–100% and 89%–98%, respectively.[36–38] Loewe et al demonstrated that the sensitivity and specificity of MRA for exact-length measurements of aortic arch lesions was 100% and 96%, respectively.[36] This noninvasive technique, alone or in combination with duplex ultrasonography, has been shown to be cost-effective when compared with pre-operative contrast arteriography.[39] Similarly, with improvements in computed tomography (CT) angiography, 3-dimensional rendered images provide valuable information about the anatomy and plaque characteristics of the target lesion.

Ultrasonography is important in the diagnosis and follow-up of steno-occlusive disease of the supra-aortic trunk. While this imaging modality is less costly and non-invasive, it lacks the ability to directly characterize lesions, which is required for treatment planning. Ultrasonographic findings in steno-occlusive disease of the innominate artery are reversed or biphasic flow in the right vertebral artery, mid-systolic deceleration in the right carotid arterial system, and an elevated LCCA/RCCA ratio.[40]

Duplex ultrasonography of subclavian arteries with obstructive lesions demonstrates elevated systolic velocity, loss of biphasic waveforms, and post-stenotic turbulence.[41] For the detection of obstructive lesions of ≥ 50%, duplex ultrasound has a sensitivity and specificity of 0.73 and 0.91, respectively, and a negative predictive value of 0.97.[42]

TECHNIQUES OF ENDOVASCULAR THERAPY

Several different approaches have been utilized to gain vascular access for the endovascular treatment of supra-aortic trunk lesions. Access via the brachial artery, femoral artery, or cervical carotid has been reported. Advantages and complications are associated with each method, which must be considered during the preoperative planning stage.

Transfemoral (Antegrade) Approach

The transfemoral approach is a similar technique to carotid artery stenting. Using a long 6 or 7 French sheath placed just proximal to the target lesion, the stenosis can then be crossed and treated. This approach allows for the use of a mechanical embolic protection system if desired. Most frequently, a balloon-expandable stent is necessary to adequately treat orificial lesions. This allows for accurate placement of the stent, good wall apposition, and strong radial force. Technical success is confirmed by brisk flow through the stented lesions, without evidence of thrombosis or dissection. In addition, less than 10% residual stenosis should be observed.[15] With treatment of subclavian and innominate artery lesions, normalization of blood pressure between the two arms should be observed. No access site complications were reported in 40 patients undergoing stenting of the subclavian or innominate artery via a transfemoral approach.[22] Sullivan et al reported 1 pseudoaneurysm occurring in 49 femoral access sites.[15]

Transbrachial or Transaxillary Approach

The retrograde transbrachial approach is useful for occlusive lesions of the left subclavian artery, but may also be used to treat innominate or proximal right subclavian lesions. By approaching the vascular lesion in a retrograde manner, the difficulty of navigating the acute angle of takeoff of this artery is avoided, which may also lessen the risk of aortic dissection. Additionally, the close proximity of the access site to the left subclavian artery orifice may facilitate manipulation of the guide wire and catheter through the lesion. Both open and percutaneous techniques have been described for gaining access via the brachial artery. Typically, the brachial artery is accessed in the antecubital fossa using a micropuncture technique under ultrasound guidance. A 35-cm 5 or 6 French catheter, introduced into the artery over a 0.035-in guide wire, is advanced to the distal end of the lesion, as described by Criado et al.[43] Brachial artery access site complications are higher than other methods of vascular access, due to the smaller size of the vessel. In two series, operative

repair of the brachial artery was necessary following the procedure either due to thrombosis or pseudoaneurysm.[15,17] Current mechanical embolic protection devices cannot be employed in this circumstance given the retrograde access. As in the transfemoral approach, balloon-expandable stents are most frequently used.

Transcarotid Approach

The transcarotid approach is particularly advantageous in cases of small or tortuous brachial, axillary, or iliac arteries. Certainly a completely percutaneous approach is feasible; however, an added benefit of an open cut-down to the common carotid artery (CCA) is the ability to achieve cerebral protection during the intervention. Several authors have found the retrograde cervical approach useful for the treatment of tandem lesions of the internal carotid bifurcation and common carotid artery at the time of carotid endarterectomy.[44–47] It is also advantageous when treating patients with tortuous aortic arch anatomy, extensive aorto-iliac atherosclerotic disease, and in the 1%-2% of patients in which a prior femoral approach has been unsuccessful.[48]

The patient is positioned on the operating table with neck extended and rotated towards the contralateral side. After general anesthesia is induced, a longitudinal 2-centimeter incision is made at the anterior border of the sternocleidomastoid muscle. After the platysma is incised, the carotid sheath is opened and the CCA encircled with a vessel loop. The vagus nerve, which commonly lies posterior to the artery, must be identified and protected. Next, the patient is systemically heparinized and the CCA cannulated in a retrograde manner using a micropuncture technique and a 5 French sheath is placed. Digital subtraction arteriography is then performed to evaluate the proximal stenosis. A right posterior oblique view is useful to evaluate proximal stenosis of the CCA. Stenosis of the origin of the innominate artery is best evaluated with a left anterior oblique view.[24] A diagnostic pigtail catheter, inserted transfemorally, may facilitate angiographic imaging (Figure 12–1). The guide wire is passed in a retrograde fashion beyond the proximal lesions using fluoroscopic guidance. If the lesion lies at the origin of the common carotid or innominate artery, the stent should be placed 2- or 3-mm into the aortic arch to prevent ostial stenosis using a balloon-expandable stent (Figure 12–2A–C). Prior to inserting the stent, the distal CCA is clamped to prevent intracranial embolization. Once the balloon is removed from the sheath, 20-40 cc of blood are aspirated from the sheath and then the CCA clamp is removed to re-institute

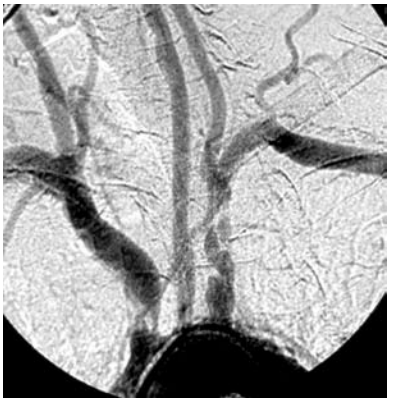

Figure 12-1. Conventional diagnostic arch aortogram via a transfemoral pigtail catheter. This repesents a steep left anterior oblique projection facilitating a clear view of the right innominate and left subclavian ulcerated stenotic lesions.

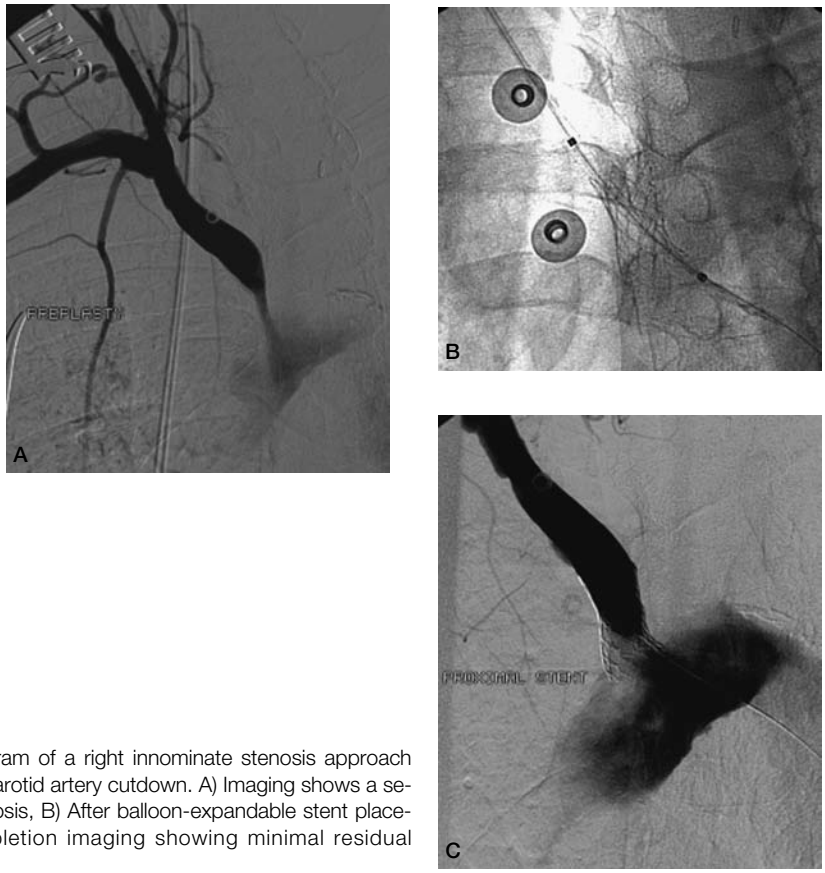

Figure 12-2. Angiogram of a right innominate stenosis approach from right common carotid artery cutdown. A) Imaging shows a severe innominate stenosis, B) After balloon-expandable stent placement, and C) completion imaging showing minimal residual stenosis.

antegrade cerebral flow. If necessary a standard carotid endarterectomy can then be performed.[47] Percutaneous access of the common carotid artery for retrograde stenting has also been described.[43] However, we prefer the open technique due to the increased incidence of cervical hematoma, and potential risk of thrombus formation during post-pull compression, with a percutaneous approach.

Grego et al reported technical success in 14 of 16 patients undergoing synchronous CEA and retrograde endovascular treatment of aortic arch vessels. In two patients the innominate lesion could not be traversed with the guide wire.[47] Ruebben et al reported a technical success rate of 100% in 8 patients treated for isolated stenosis of the innominate artery via a transcervical approach.[49] Levien et al published a series in which 43 of 44 patients were successfully treated with balloon angioplasty of brachiocephalic or common carotid artery stenoses at the time of CEA.[50] At our institution, 14 patients have been successfully treated using a retrograde cut-down on the common carotid artery.[51]

Intermediate patency rates of stented aortic arch vessels have been comparable with more traditional surgical interventions. In a review of 7 papers with a total of 108 patients undergoing percutaneous revascularization of aortic arch vessels, the combined technical success rate was 97+/−4%. Restenosis occurred in 3+/−5% of patients at a mean duration of follow-up of 20+/−9 months. Although these results are

comparable to surgical therapy, the complication rate in the endovascular group was 6+/−5%, significantly lower than the combined complication rate of 16+/−11% in the published surgical series.[25] In a review of the literature, the technical success rate stent placement across supra-aortic trunk lesions ranged from 89%-100%. The initial success was higher with stenotic lesions when compared to complete occlusions.

COMPLICATIONS IN ENDOVASCULAR THERAPY

While endoluminal supra-aortic trunk stenting is gaining broader approval, there remain some particular shortcomings. Most would agree that stenotic atherosclerotic lesions can be effectively managed with stenting; however total occlusions or heavily calcified lesions should be approached with caution or avoided altogether. This is primarily due to the increased risk of distal embolization, restenosis, and aortic dissection (Figure 12–3A and B).

Late stent fractures in this location can occur, but fortunately are oftentimes clinically insignificant. The proposed mechanisms are multi-factorial and include multiaxial biomechanical forces due to the proximity of these vessels to the primary arterial outflow tract as well as the shear forces from the curvature of the aortic arch. Stent fracture was defined as classified by Jaff et al[52] and also described by Rocha-Singh.[53] ***Type I:*** single strut fracture or soft crush deformations; ***Type II:*** multiple strut fractures; ***Type III:*** singular complete transection fracture; and ***Type IV:*** two or more transverse fractures with or without migration. A recent retrospective review at our institution of 27 ostial SAT lesions managed with balloon-expandable stent detected at a mean follow-up of 34 months, 3 type IV stent fractures in the innominate artery as well as 2 type I fractures at the innominate and common carotid arteries.[54] Other unique complications of endoluminal interventions in this territory are access site complications and embolization to the anterior or posterior intracranial circulation as well as the visceral vessels and legs.

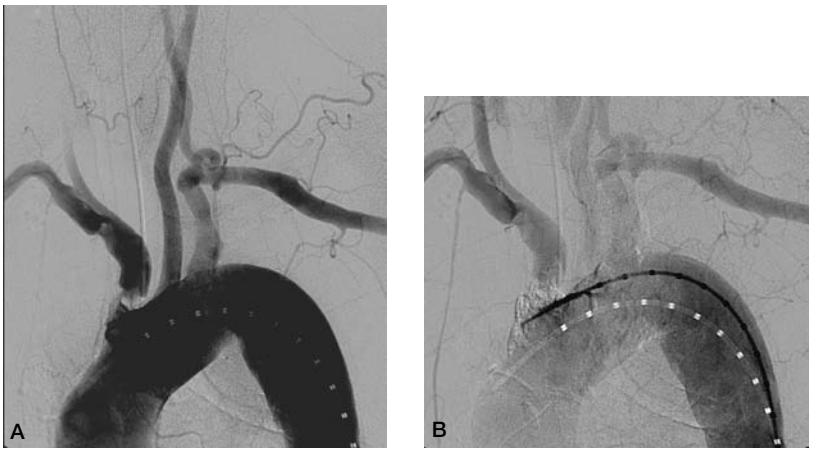

Figure 12-3. Angiogram showing extensive calcified plaque and an associated stenosis of the right innominate artery. A) Arch aortogram showing the lesion and delayed contrast enhancement beyond the stenosis and B) Circumferential calcification of the lesion.

CONCLUSION

Endoluminal therapy of supra-aortic trunk lesions is a feasible alternative to operative repair with the exception of completely occluded vessels. Agreed upon guidelines for treatment include symptomatic ≥ 50% or ≥ 80% asymptomatic atherosclerotic lesions, avoidance of heavily calcified plaques, and Takayasu's arteritis. Accepted approaches to the supra-aortic trunk vessels include transfemoral, transbrachial/transaxillary, and transcarotid, with the last providing the shortest, most direct route to the target vessel as well as the opportunity to employ embolic protection or do a concomitant carotid endarterectomy. Close follow-up annually with duplex imaging and 2-view plain radiographs are recommended to detect stent fractures.

REFERENCES

1. Savory W. A case of a young woman in whom the main arteries of the both upper extremities, and of the left side of the neck, were throughout completely obliterated. *Med Chir Trans*, 1856. 39: p. 205.
2. Shimizu K andSano K. Pulseless disease. *J Neuropathol Clin Neurol*. 1951;1:37–46.
3. Davis JB, Grove WJ, and J. O.C., Thrombic occlusion of branches of aortic arch. Martonell's syndrome: report-of case treated surgically. *Ann Surg*. 1956;144:124–126.
4. Crawford ES, et al. Surgical treatment of occlusion of the innominate, common carotid, and subclavian arteries: a 10 year experience. *Surgery*. 1969;65(1):17–31.
5. Berguer R, Morasch MD, and Kline RA. Transthoracic repair of innominate and common carotid artery disease: immediate and long-term outcome for 100 consecutive surgical reconstructions. *J Vasc Surg*. 1998; 27(1): 34–41; discussion 42.
6. Lyons C and Galbraith G. Surgical treatment of atherosclerotic occlusion of the internal carotid artery. *Ann Surg*. 1957;146(3):487–96; discussion 496–8.
7. DeBakey M, Morris G, and Jordan G. Segmental thrombo-obliterative disease of the great vessels arising from the aortic arch. *JAMA*. 1958;166:998–1003.
8. Diethrich EB, et al. Occlusive disease of the common carotid and subclavian arteries treated by carotid-subclavian bypass. Analysis of 125 cases. *Am J Surg*. 1967;114(5):800–8.
9. Berguer R, et al. Cervical reconstruction of the supra-aortic trunks: a 16–year experience. *J Vasc Surg*. 1999;29(2):239–46; discussion 246–8.
10. Perler BA and Williams GM, Carotid-subclavian bypass—a decade of experience. *J Vasc Surg*. 1990;12(6):716–22; discussion 722–3.
11. Mathias K, et al. Percutaneous transluminal treatment of supraaortic artery obstruction. *Ann Radiol* (Paris). 1980;23(4):281–282.
12. Bachman DM and Kim RM. Transluminal dilatation for subclavian steal syndrome. *AJR Am J Roentgenol*. 1980: 135(5);995–996.
13. Becker GJ, Katzen BT, and Dake MD. Noncoronary angioplasty. *Radiology*. 1989;170(3 Pt 2): 921–940.
14. Kachel R, et al. Percutaneous transluminal angioplasty (PTA) of supra-aortic arteries especially the internal carotid artery. *Neuroradiology* 1991;33(3):191–194.
15. Sullivan, TM, et al. Angioplasty and primary stenting of the subclavian, innominate, and common carotid arteries in 83 patients. *J Vasc Surg*. 1998;28(6):1059–1065.
16. Queral LA and Criado FJ. Endovascular treatment of aortic arch occlusive disease. *Semin Vasc Surg*. 1996;9(2):156–163.
17. Rodriguez-Lopez JA et al. Stenting for atherosclerotic occlusive disease of the subclavian artery. *Ann Vasc Surg*. 1999;13(3):254–260.
18. Takach TJ, et al. Brachiocephalic reconstruction II: operative and endovascular management of single-vessel disease. *J Vasc Surg*. 2005;42(1):55–61.

19. Wylie EJ and Effeney DJ. Surgery of the aortic arch branches and vertebral arteries. *Surg Clin North Am*. 1979;59(4):669–680.
20. Hass WK, et al. Joint study of extracranial arterial occlusion. II. Arteriography, techniques, sites, and complications. *JAMA*.1968; 203(11):961–968.
21. Williams SJ 2nd, Chronic upper extremity ischemia: current concepts in management. *Surg Clin North Am*. 1986;66(2):355–375.
22. Brountzos EN, et al. Primary stenting of subclavian and innominate artery occlusive disease: a single center's experience. *Cardiovasc Intervent Radiol*. 2004;27(6):616–623.
23. Diethrich EB, et al. Percutaneous techniques for endoluminal carotid interventions. *J Endovasc Surg*. 1996;3(2):182–202.
24. Brountzos EN, Malagari K and Kelekis DA. Endovascular treatment of occlusive lesions of the subclavian and innominate arteries. *Cardiovasc Intervent Radiol*. 2006;29(4):503–510.
25. Hadjipetrou P, et al. Percutaneous revascularization of atherosclerotic obstruction of aortic arch vessels. *J Am Coll Cardiol*. 1999;33(5):1238–1245.
26. Liang, P. and G.S. Hoffman, Advances in the medical and surgical treatment of Takayasu arteritis. *Curr Opin Rheumatol*. 2005;17(1):16–24.
27. Sakaida, H., et al., [Stenting for the occlusive carotid and subclavian arteries in Takayasu arteritis]. *No Shinkei Geka*. 2001;29(11):1033–1041.
28. Budin, J.A., W.J. Casarella, and L. Harisiadis, *Subclavian artery occlusion following radiotherapy for carcinoma of the breast. Radiology*. 1976;118(1):169–173.
29. Hashmonai, M., et al., Subclavian artery occlusion after radiotherapy for carcinoma of the breast. *Cancer*. 1988;61(10):2015–2018.
30. Hassen-Khodja, R. and E. Kieffer, Radiotherapy-induced supra-aortic trunk disease: early and long-term results of surgical and endovascular reconstruction. *J Vasc Surg*. 2004;40(2): 254–261.
31. Kay, H.R., et al., Atherosclerosis of the internal mammary artery. *Ann Thorac Surg*. 1976. 21(6):504–507.
32. Singh, R.N., Atherosclerosis and the internal mammary arteries. *Cardiovasc Intervent Radiol*. 1983;6(2):72–77.
33. Olsen, C.O., et al., Review of coronary-subclavian steal following internal mammary artery-coronary artery bypass surgery. *Ann Thorac Surg*. 1988;46(6):675–678.
34. Beneficial effect of carotid endarterectomy in symptomatic patients with high-grade carotid stenosis. North American Symptomatic Carotid Endarterectomy Trial Collaborators. *N Engl J Med*. 1991;325(7):445–453.
35. Endarterectomy for asymptomatic carotid artery stenosis. Executive Committee for the Asymptomatic Carotid Atherosclerosis Study. *JAMA*. 1995;273(18):1421–1428.
36. Loewe, C., et al., MRA versus DSA in the assessment of occlusive disease in the aortic arch vessels: accuracy in detecting the severity, number, and length of stenoses. *J Endovasc Ther*. 2004;11(2):152–160.
37. Randoux, B., et al., Proximal great vessels of aortic arch: comparison of three-dimensional gadolinium-enhanced MR angiography and digital subtraction angiography. *Radiology*. 2003;229(3):697–702.
38. Carpenter, J.P., et al., Magnetic resonance angiography of the aortic arch. *J Vasc Surg*. 1997; 25(1):145–151.
39. Kent, K.C., et al., Perioperative imaging strategies for carotid endarterectomy. An analysis of morbidity and cost-effectiveness in symptomatic patients. *JAMA*. 1995;274(11):888–893.
40. Grant, E.G., et al., Innominate artery occlusive disease: sonographic findings. *AJR Am J Roentgenol*. 2006;186(2):394–400.
41. Kalaria, V.G., et al., Duplex ultrasonography of vertebral and subclavian arteries. *J Am Soc Echocardiogr*. 2005;18(10):1107–1111.
42. Ackerstaff, R.G., et al., Ultrasonic duplex scanning in atherosclerotic disease of the innominate, subclavian and vertebral arteries. A comparative study with angiography. *Ultrasound Med Biol*. 1984;10(4):409–418.

43. Criado, F.J. and O. Abul-Khoudoud, Interventional techniques to facilitate supraaortic angioplasty and stenting. *Vasc Endovascular Surg*. 2006;40(2):141–147.
44. Queral, L.A. and F.J. Criado, The treatment of focal aortic arch branch lesions with Palmaz stents. *J Vasc Surg*. 1996;23(2):368–375.
45. Payne, D.A., et al., Cerebral protection during open retrograde angioplasty/stenting of common carotid and innominate artery stenoses. *Br J Surg*. 2006;93(2):187–190.
46. Macierewicz, J., et al., Carotid endarterectomy combined with proximal stenting for multilevel disease. *Eur J Vasc Endovasc Surg*. 2000;20(6):572–575.
47. Grego, F., et al., Synchronous carotid endarterectomy and retrograde endovascular treatment of brachiocephalic or common carotid artery stenosis. *Eur J Vasc Endovasc Surg*. 2003; 26(4):392–395.
48. Yoo, B.S., et al., A case of transradial carotid stenting in a patient with total occlusion of distal abdominal aorta. *Catheter Cardiovasc Interv*. 2002;56(2):243–245.
49. Ruebben, A., et al., Feasibility of intraoperative balloon angioplasty and additional stent placement of isolated stenosis of the brachiocephalic trunk. *J Thorac Cardiovasc Surg*. 1998; 115(6):1316–1320.
50. Levien, L.J., et al., Retrograde balloon angioplasty of brachiocephalic or common carotid artery stenoses at the time of carotid endarterectomy. *Eur J Vasc Endovasc Surg*. 1998;15(6): 521–527.
51. Peterson, B.G., et al., Aortic arch vessel stenting: a single-center experience using cerebral protection. *Arch Surg*. 2006;141(6):560–563; discussion 563–564.
52. Jaff M, Dake M, Pompa J, Ansel G, Yoder T. Standardized evaluation and reporting of stent fractures in clinical trials of noncoronary devices. *Catheter Cardiovasc Interv*. Sept 2007; 70(3):460–462.
53. Rocha-Singh KJ, Jaff MR, Crabtree TR, Bloch DA, Ansel G. Performance goals and endpoint assessments for clinical trials of femoropopliteal bare nitinol stents in patients with symptomatic peripheral arterial disease. *Catheter Cardiovasc Interv*. 2007 May 1;69(6):910–919.
54. Usman, A.A., et al., Late stent Fractures Following endoluminal treatment of ostial supraaortic trunk arterial occlusive lesions. *JVIR* 2010 in press

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Management of Carotid Stent Complications

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INTRODUCTION

Advances in endovascular therapies have allowed for carotid artery angioplasty and stenting (CAS) to now be recognized as an acceptable alternative for treating extracranial carotid artery stenosis. Notable trials comparing CAS to CEA in both high-risk populations and the community at large have been published with compelling results[1–5]. Stenting and Angioplasty with Protection in Patients at High Risk for Endarterectomy (SAPPHIRE) randomized high-risk patients to either CAS with embolic protection versus CEA, and showed that CAS was not inferior to CEA when the combined endpoints of myocardial infarction (MI), stroke, and death were examined[1]. ACCULINK for Revascularization of Carotids in High-Risk patients (ARCHeR) also concluded that when compared to high-risk CEA historical controls, CAS with cerebral protection was not inferior to CEA using similar primary endpoints[2]. Carotid Revascularization using Endarterectomy or Stenting Systems (CaRESS), a nonrandomized, equivalence cohort study, showed that the patients who underwent CAS with various forms of mechanical embolic protection had similar rates of 30-day outcomes as did patients in earlier CEA studies[6,7].

Fortunately, vascular surgeons have embraced this technologic leap of faith and are rapidly achieving a greater presence among the various subspeciality physicians performing CAS with good outcomes[8,9]. As the application of CAS gains continuing momentum, a larger body of data is available for review, which will be helpful in detecting the causes of failures. This chapter will provide an overview of some of the more common problems and challenges associated with CAS, as well as potential solutions.

IATROGENIC ARTERIAL INJURY (LOCAL AND REMOTE)

Access Site Vessels

As with any interventional procedure, access to the arterial system has its own unique set of complications. Typically, femoral arterial access is used for CAS. Many such patients

will have coexisting peripheral vascular occlusive disease which may predispose them to increased risks of dissection, infection, hematoma, pseudoaneurysm, arteriovenous fistula, thrombosis, and retroperitoneal hematoma following any percutaneous intervention. A recent study showed that complications arising from endoluminal procedures that require operative repair were found to occur at a rate of 0.7% for diagnostic angiography alone, to as high as 3.4% after interventional therapies[10]. The use of large diameter sheaths and concomitant systemic anticoagulation are known to result in higher periprocedural complications.[10–12]

Pseudoaneurym and Arteriovenous Fistula

Femoral pseudoaneurysms (FPAs) are one of the more frequent of the vascular complications requiring treatment after percutaneous interventions. A recent report documents the rate of FPAs to be 1.7% for all angiographic procedures. Furthermore, of these, the incidence of FPAs that required surgical repair was 1.1% after diagnostic procedures and 4.7% following interventional procedures[13]. Identified risk factors were noted to be presence of hypertension, high body mass index (BMI), sheath sizes exceeding 7Fr, improper location of arterial puncture site (either in the external iliac artery or below the common femoral bifurcation), inadequate compression of the vessel at completion of the procedure, and excessive anticoagulation. The majority of small (<3 cm), non-enlarging, asymptomatic FPAs may be managed expectantly with many thrombosing spontaneously over one to two months[14,15]. Otherwise, alternative therapy is indicated for FPAs that do not thrombose spontaneusly. Proposed options have evolved over the last decade from surgical repair to ultrasound-guided compression, and now ultrasound-guided thrombin injection achieving prompt durable results[16–17]. On the other hand, iatrogenic arteriovenous fistulae which occur with less frequency typically require operative repair.

Hematoma (Local and Retroperitoneal)

Similarly, groin hematoma and retroperitoneal hematoma are a direct result of improper puncture technique or inadequate compression of the vessel upon removal of the sheath. Groin hematomas, the most common complication, can occur in up to 10% of angiographic procedures but those requiring transfusion or surgical intervention occur in fewer than 0.5% of cases[18]. The incidence of retroperitoneal hematoma is reported at 0.15% but requires consideration as a life-threatening complication. Unless a high index of suspicion is maintained, many retroperitoneal hematomas go unrecognized until hemodynamic instability from hemorrhagic shock occurs. This can be a devastating complication and is best assessed by clinical evaluation coupled with imaging—preferably an intravenous contrasted enhanced computed tomography (CT) scan. In light of these problems, a percutaneous closure device may lessen their occurrence.

Brachial Access

When dealing with patients in which femoral arterial access is not possible, brachial artery access is another option. This location carries a similar set of complications as femoral access, including hematoma, arterial dissection, nerve injury, or inadequate vessel diameter for delivery sheaths. Complications related to arterial access are best avoided by proper patient selection and careful access to the vessel as well as careful hemostasis at completion of the procedure. Adjunctive use of ultrasound guidance allows measurement of the target vessel size and ensures accurate placement of the sheath. Unlike femoral artery access, the

most worrisome complication of brachial access is the development of a small brachial sheath hematoma which can be visually unapparent, yet can compress the median nerve and result in permanent neurologic impairment. Postprocedural vigilance is warranted; any clinical stigmata of nerve impingement mandates prompt exploration and evacuation of the hematoma.[19]

Arch and Brachiocephalic Vessels

It must be remembered that *all* phases of CAS are associated with the risk of distal embolization and neurologic compromise. This even includes the diagnostic arch aortogram. Careful placement of the catheter in the aortic arch and removal of air from the line is essential. When attempting to cannulate the arch vessels for either imaging or intervention, direct injury (dissection or embolization) can be averted with good techniques. Among elderly patients excessive tortuosity, angulation, and/or calcification can make tracking of catheters and wires particularly difficult and thus carry a higher risk of embolization or local arterial injury.[20] Not surprisingly, one study demonstrated that 90% of embolic events recorded during coronary angiography occurred during either contrast injection or during manipulation of catheters and wires around the aortic arch, yet resulted in no neurologic sequelae. It cannot be emphasized enough that attempts at CAS in elderly patients (> 80 years of age) with heavily calcified and tortuous aortic arch anatomy should be reserved for highly experienced interventionalists or avoided altogether.

Internal Carotid Artery

The extracranial internal carotid artery (ICA) is also prone to local traumatic injury, particularly with the widespread use of mechanical embolic protection devices (EPDs). Spasm in the form of either true vasospasm or pseudospasm can occur from wire placement alone, but more characteristically manifests after placement of distal filtration devices.[21–22] True vasospasm can be so severe that it can result in flow arrest in the distal ICA and possible ICA thrombosis. Fortunately, as reported by Reimers et al, the majority of cases quickly abate with intra-arterial administration of nitrates.[22] Similarly, Cremonesi et al observed spasm of the ICA related to the protection device in 7.9% of their stent procedures.[23] These same investigators found that when flow impairment continued despite intra-arterial nitrate, then occlusion of the filter from debris or clot was evident and flow was normalized when the filter was retrieved. On the other hand, pseudospasm occurs because of the placement of a rigid system (ie, wire or EPD) within an angulated distal ICA, which causes the vessel to accordion upon itself. The worrisome aspect of this is the potential of forming a clot within the small cul-de-sacs or causing a localized dissection. This form of spasm is not relieved with vasodilators and will only improve with removal of the offending element. Dissection of the distal ICA can also occur with stiff wires or filters in sharply angulated vessels and with distal balloon occlusion systems from overinflation of the occlusion balloon.

HIGH-RISK TARGET LESIONS (ANATOMIC AND PATHOLOGIC)

Vessel Tortuosity

In the ideal circumstances, the target lesion for CAS will be straight, focal, and heterogenous in its pathologic make-up. From a practical point of view, this is a rare occurrence. In fact, vessel tortuosity is far more common and in some cases can be so severe, that it

precludes any attempt at CAS. Once the common carotid artery is cannulated with either a guiding catheter or sheath, the next step is crossing and treating the offending lesion. Surprisingly, moderate degrees of angulation of the common carotid and ICA can be exaggerated after placement of rigid sheaths and wires. The end result is either inability to cross the lesion with an EPD or bare wire. An additional concern is kinking of the leading edge of the stent if it is placed at a natural bend in the ICA. Coils, kinks, and severe tortuosity of the ICA should be avoided when considering CAS.

Plaque Characteristics

It is not unusual for carotid bifurcation disease to be a mix of soft and calcified plaque, but too much of either one is a predictor of poorer outcomes after CAS. It has been demonstrated that echolucent or severely stenotic plaques in the ICA carry a higher risk of embolic events during CAS.[24] This is further supported by Mathur et al, who found in their series of 231 stent procedures that there was a relationship between increased incidence of procedural stroke in lesions that were categorized as long/multiple (>10 mm in length and/or >1 lesion separated by normal vessel wall) as well as lesions that were >/= 90% stenosed.[25] Presumably these types of lesions have increased embolic potential due to plaque burden and severe stenosis, making traversing them with protection devices more risky. The ICAROS study sought to find a relationship between the gray-scale median (GSM) as an indicator of echogenicity of plaques and risk of CVA during CAS.[26] These investigators found that lesions with a GSM ≤ 25, correlating with a predominantly echolucent plaque, and lesions with a higher degree of stenosis, carried a higher risk of stroke during CAS. Moreover, utilization of EPDs increased the risk of adverse events, suggesting that crossing these lesions with devices is hazardous. This has important implications for the selection of patients for CAS. The implication is that lesions that are echolucent and severely stenotic carry a higher risk of procedural stroke from embolic events. It is in these patients that CAS may have a higher risk of CVA versus CEA, and these individuals should not be considered candidates for an endovascular approach.[27] Similiarly, heavily calcified lesions are to be avoided. The two primary concerns are inability to fully expand the stent and embolization from a friable plaque.

EPDS AND STENTS

Mechanical Cerebral Protection

The recognized potential for cerebral emboli from thrombus or intra-arterial atheromatous debris during CAS fostered development of 3 main types of embolic protection devices: 1) distal balloon occlusion, 2) filters, and 3) proximal balloon occlusion.[28–29] There are limitations to each device and each carries its own set of technical considerations.

Distal filtration devices consist of a nitinol skeleton covered with a porous polyurethane filter which is designed to trap released embolic debris while maintaining antegrade cerebral flow. In general, these devices have a large crossing profile, making it more difficult to cross extremely tight lesions without prior angioplasty. Therefore, tight lesions may require pre-dilation with a small balloon (2 or 3 mm) first to assist in delivery of the filter element. Obviously, "pre-dilation" is prone to the risk of embolic complications. In cases of sharply angulated target lesions, use of a "buddy wire" to facilitate placement of filter devices may be beneficial. It is essential that an

appropriately sized filter device is placed in a relatively straight portion of the distal ICA to ensure good wall apposition, precluding passage of debris around the system. Ideally, filters should be oversized—0.3 mm-1 mm larger than the ICA diameter in the intended landing zone. The filter device should remain in place without excessive movement. There is evidence to suggest that even slight movement of the filter device can cause intimal damage and increase the amount of embolic particles released.[30] In order to keep the filter location constant, the CCA sheath location must also remain constant. Prolapse of the guiding sheath into the arch or excessive movement of the sheath can result in excessive movement resulting in filter basket detachment, stent entanglement, or guide wire damage.

Distal balloon occlusion devices have the advantage of lower crossing profiles and usually do not require pre-dilation to cross the target lesion. Flow arrest must be documented after the occlusion balloon is inflated in order to ensure adequate wall apposition with the distal ICA. After angioplasty and placement of the stent, the static column of blood is then aspirated before resumption of flow to the cerebral circulation. The protective effect of the system relies on cessation of flow to the cerebral circulation during the intervention, thereby eliminating the risk of cerebral emboli. Not all patients can tolerate flow arrest. In fact a recent study evaluating patients undergoing CAS with a balloon occlusion device (PercuSurge Guardwire) reported an incidence of neurologic "intolerance" in 10/43 patients during CAS.[31] Fortunately, all neurologic events were transient and resolved with resumption of intracerebral flow. An incomplete Circle of Willis was identified in the majority of patients experiencing procedural neurological compromise. Maintenance of adequate blood pressure and intravascular volume status during CAS, as well as prevention of bradycardia, ameliorates cerebral hypoperfusion that may also contribute to neurological compromise.

Proximal balloon occlusion and flow reversal is a novel approach to CAS, analogous to the methods employed during traditional CEA. In theory, the benefit is that the approach establishes cerebral protection with flow reversal prior to actually crossing the target lesion. Furthermore, intolerance occurs less frequently with flow reversal than with flow cessation,[32] and this form of protection allows for the capture of all particulate debris regardless of size. The first generation designs were cumbersome, rigid, and required large delivery sheaths (10Fr). Newer systems are easier to use and lower profile. Unfortunately, not enough data is available to precisely assess this procedure's true benefit.

Filter Retrieval Failure

Conversion to open CEA may be required if the distal protection device cannot be safely recovered after stenting is completed. Removal of the filtration device is difficult when large amounts of thrombus or atheromatous debris fill the filter basket to a point that it cannot be collapsed enough be to be captured with the retrieval catheter. Prevention of this problem is multifactorial, including good technique (ie, avoiding over-dilating the artery), adequate anti-platelet therapy, and full systemic anticoagulation during the procedure (target ACT 250-300). When retrieving the filter device, care must be taken to avoid prematurely retracting the filter before it is properly sheathed—otherwise the filter element can become entangled within the stent struts.[33–34] A dreaded complication is detachment of the filter element after entanglement. The recommendations for such a problem are to trap the filter by placing an additional stent to "plaster" it against the arterial wall and prevent migration. In tortuous vessels, the retrieval sheath may be difficult to maneuver across the

stented ICA and advancement of another type or size of sheath for recovery has been described. It may also be helpful to move the patient's head or have the patient swallow to assist in advancing the catheter past the stent. If these solutions are not successful, emergent conversion to open surgical repair is advocated. Good case planning and selection should avert the need for such dire options.

Stent Fracture

Carotid stent fracture is not uncommon and has been associated with the presence of calcification in the internal carotid artery. Varcoe et al found a stent fracture prevalence of 2.2% in 48 carotid stents placed in 43 patients. They reported that a calcified artery is eight times more likely to have a fracture.[35] We began evaluating our patients for stent fractures 2 years ago with two-view cervical radiographs after identifying a type III fracture with restenosis.[36] To date, 73 patients have had follow-up plain radiographic imaging of their carotid stents. The average follow-up time for stent fracture detection is 30 months. There were four (5.5%) total stent fractures: two type I stent fractures and two type III fractures.[37]

The location of CAS exposes stents to stretching and axial forces from the neck, leading to an increased propensity for stent fractures. Significant angulation occurs in the distal portion of the ICA, sometimes ≥ 80°. Due to this large angulation and the relatively stiff stented segment of the artery, the unstented arterial segments have to accommodate head movements with increased torsion, which will lead to friction at the ends of the stent.

Annual two-view cervical radiography should be part of the routine surveillance for CAS. Management of fractures is dependent on the clinical and radiographic findings, but ranges from medical management to surgical excision.

SYSTEMIC COMPLICATIONS

Stroke

The primary objective of CAS is stroke prevention. In order to achieve this, a procedural risk of stroke is a recognized complication. The etiology of CAS-associated strokes is most frequently embolic in nature, and arises from either atheroemboli, fresh clot, or air. All three are preventable with good interventional techniques. The treatment of stroke from the first two remains controversial, but most would pursue systemic therapy (ie, anticoagulation, permissive hypertension) over catheter-directed lysis. Stroke from air emboli is a unique and unusual situation that is best treated with hyperbaric oxygen therapy .

Hemodynamic Instability

Periprocedural hemodynamic instability after CAS can manifest as hypertension, hypotension, or bradycardia, with an incidence ranging from 13%-68%.[38–41] Many of these are a direct result of stretch on the carotid sinus and stimulation of the baroreflex. The resultant effect is a decrease in sympathetic tone and a temporary increase in parasympathetic stimulation resulting in hypotension and bradycardia. Attempts to elucidate the risk factors for these hemodynamic changes during CAS have revealed results with some common themes. One study that evaluated 140 CAS procedures found that balloon expandable stents and larger diameter balloon expansion resulted in a higher incidence of post-operative

hypotension.[38] Similarly, Mendelsohn et al found a relationship between the need for dopamine infusion during the post-dilation period in patients who had placement of stents that were ≥ 10 mm in width or ≥ 40 mm in length as opposed to patients with smaller stents who required no additional treatment for hypotension.[40] There is evidence that older women and patients who have had a previous myocardial infarction have an increased risk of experiencing hypotension during the procedure, and they were more likely to remain hypotensive in the post-operative period.[41–42] Another finding among several studies was that patients who required treatment for intra-procedural hypotension were more likely to have post-operative hypotension that requires ICU monitoring and additional medications.[40–42] Avoiding oversized stents and maintaining adequate intra-vascular volume are measures that may avoid hypotension requiring prolonged vasopressor medications in the post-operative period. Being prepared for these events by having vasopressor agents on hand is essential in avoiding profound hypotension and possible resultant cerebral ischemia.

Bradycardia

Bradycardia also occurs quite frequently and often in conjunction with hypotension. Many advocate use of prophylactic atropine dosing prior to angioplasty and stent placement. Although there are no randomized studies evaluating the efficacy of pre-medication with atropine, there is a suggestion that it decreases the incidence of intra-procedural bradycardia and hence prevents additional cardiac stress. This is especially true in patients undergoing CAS who have primary carotid stenosis.[43] At least the addition of 0.5 mg-1.0 mg of prophylactic atropine intravenously prior to balloon dilation and stent placement has not had any deleterious effects or caused any adverse cardiac events that have been reported.

CONCLUSION

CAS remains a viable alternative to CEA in certain subgroups of patients with cervical carotid stenosis. While randomized trials continue to clarify its role in other populations, evaluation of periprocedural failures may help reduce associated complications. Clearly, experience, judgment, case planning, and case selection influence the overall outcomes of CAS. It is hoped that this chapter sheds some light on recognized preventable complications and will assist future interventionalists wishing to pursue this mode of therapy.

REFERENCES

1. Yadav JS, Wholey MH, Kuntz RE, et al. Protected carotid-artery stenting versus endarterectomy in high-risk patients. *N Eng J Med*. 2004;351:1493–1501.
2. Roubin GS, New G, Iyer SS, et al. Immediate and late clinical outcomes of carotid artery stenting in patients with symptomatic and asymptomatic carotid artery stenosis. *Circulation.* 2001;103:532–537.
3. CaRESS Steering Committee. Carotid Revascularization using Endarterectomy or Stenting Systems (CaRESS) phase I clinical trial: 1-year results. *J Vasc Surg*. 2005;42:213–219.
4. CAVATAS Investigators. Endovascular versus surgical treatment in patients with carotid stenosis in the Carotid and Vertebral Artery Transluminal Angioplasty Study (CAVATAS): a randomised trial. *Lancet*. 2001;357:1729–1737.

5. Hobson RW 2nd, Howard VJ, Roubin GS, et al. Carotid artery stenting is associated with increased complications in octogenarians: 30-day stroke and death rates in the CREST lead-in phase. *J Vasc Surg*. 2004;40:1106–1111.
6. North American Symptomatic Carotid Endarterectomy Trial Collaborators. Beneficial effect of carotid endarterectomy in symptomatic patients with high-grade carotid stenosis. *N Eng J Med*. 1991;325:445–453.
7. Executive Committee for the Asymptomatic Carotid Atherosclerosis Study. Endarterectomy for asymptomatic carotid artery stenosis. *JAMA*. 1995;273:1421–1428.
8. Eskandari MK, Longo GM, Matsumura JS, et al. Carotid stenting done exclusively by vascular surgeons. *Ann Surg*. 2005;242:431–438.
9. Schneider PA, Silva MB Jr, Bohannon WT, et al. Safety and efficacy of carotid arteriography in vascular surgery practice. *J Vasc Surg*. 2005;41: 238–245.
10. Messina LM, Brothers TE, Wakefield TW, et al. Clinical characteristics and surgical management of vascular complications in patients undergoing cardiac catheterization: Interventional versus diagnostic procedures. *J Vasc Surg*. 1991;13:593–600.
11. Ricci MA, Trevisani GT, Pilcher DB. Vascular complications of cardiac catheterization. *Am J Surg*. 1994;167:375–378.
12. Fransson SG, Nylander E. Vascular injury following cardiac catheterization, coronary angiography, and coronary angioplasty. *European Heart Journal*. 1994;15:232–235.
13. Ates M, Sahin S, Konuralp C, et al. Evaluation of risk factors associated with femoral pseudoaneurysms after cardiac catheterization. *J Vasc Surg*. 2006;43:520–524.
14. Kresowik TF, Khoury MD, Miller BV, et al. A prospective study of the incidence and natural history of femoral vascular complications after percutaneous transluminal coronary angioplasty. *J Vasc Surg*. 1991;13:328–333.
15. Kent KC, McArdle CR, Kennedy B, et al. A prospective study of the clinical outcome of femoral pseudoaneurysms and arteriovenous fistulas induced by arterial puncture. *J Vasc Surg*. 1993;17:125–131.
16. Kumins NH, Landau DS, Montalvo J, et al. Expanded indications for the treatment of post catheterization femoral pseudoaneurysms with ultrasound-guided compression. *Am J Surg*. 1998;176:131–136.
17. Liau CS, Ho FM, Chen MF, et al. Treatment of iatrogenic femoral artery pseudoaneurysm with percutaneous thrombin injection. *J Vasc Surg*. 1997;26:18–23.
18. Singh H, Cardella JF, Cole PE, et al. Quality improvement guidelines for diagnostic arteriography. *J Vasc Radiol*. 2002;13:1–6.
19. Alvarez-Tostado J, Moise MA, Bena JF, et al., The brachial artery: A critical access for endovascular procedures. *J Vasc Surg* 2009;49:378–85.)
20. Bladin CF, Bingham L, Grigg L, et al. Transcranial Doppler detection of microemboli during percutaneous transluminal coronary angioplasty. *Stroke*. 1998;29:2367–2370.
21. Cardaioli P, Giordan M, Panfili M, et al. Complication with an embolic protection device during carotid angioplasty. *Catheterization and Cardiovascular Interventions*. 2004;62:234–236.
22. Reimers B, Corvaja N, Moshiri S, et al. Cerebral protection with filter devices during carotid artery stenting. *Circulation*. 2001;104:12–15.
23. Cremonesi A, Manetti R, Setacci F, et al. Protected carotid stenting: clinical advantages and complications of embolic protection devices in 442 consecutive patients. *Stroke*. 2003;34: 1936–1941.
24. Ohki T, Marin ML, Lyon RT, et al. Ex vivo human carotid artery bifurcation stenting: correlation of lesion characteristics with embolic potential. *J Vasc Surg*. 1998;27:463–471.
25. Mathur A, Roubin GS, Iyer SS, et al. Predictors of stroke complicating carotid artery stenting. *Circulation*. 1998;97:1239–1245.
26. Biasi G, Froio A, Diethrich E, et al. Carotid plaque echolucency increases the risk of stroke in carotid stenting: the Imaging in Carotid Angioplasty and Risk of Stroke (ICAROS) study. *Circulation*. 2004;110:756–762.
27. Roubin GS, Iyer S, Halkin A, et al. Realizing the potential of carotid artery stenting: proposed paradigms for patient selection and procedural technique. *Circulation*. 2006;113: 2021–2030.

28. Al-Mubarak N, Roubin GS, Vitek JJ, et al. Effect of the distal-balloon protection system on microembolization during carotid stenting. *Circulation*. 2001;104:1999–2002.
29. Eskandari MK. Cerebral embolic protection. *Sem Vasc Surg*. 2005;18:95–100.
30. Muller-Hulsbeck S, Stolzmann P, Liess C, et al. Vessel wall damage caused by cerebral protection devices: ex vivo evaluation in porcine carotid arteries. *Radiology*. 2005;235:454–460.
31. Chaer RA, Trocciola S, DeRubertis B, et al. Cerebral ischemia associated with PercuSurge balloon occlusion balloon during carotid stenting: incidence and possible mechanisms. *J Vasc Surg*. 2006;43:946–952.
32. Parodi JC, Ferreira LM, Sicard G, et al. Cerebral protection during carotid stenting using flow reversal. *J Vasc Surg*. 2005;41:416–422.
33. Ganim RP, Muench A, Giesler GM, et al. Difficult retrieval of the EPI Filterwire with a 5 French coronary catheter following carotid stenting. *Catheter Cardiovasc Interv*. 2006;67: 309–311.
34. Shilling K, Uretsky BF, Hunter GC. Entrapment of a cerebral embolic protection device. *Vasc Endovasc Surg*. 2006;40:229–233.
35. Varcoe RL, Mah J, Young N et al. Prevalence of carotid stent fractures in a single-center experience. *J Endovasc Ther.* 2008;15:485–9.
36. Surdell D, Shaibani A, Bendok B, et al. Fracture of a nitinol carotid artery stent that caused restenosis. *J Vasc Interv Radiol.* 2007;18:1297–9.
37. Eskandari MK, Usman AA, Garcia-Toca M, et al. Eight-year institutional review of carotid artery stenting. *J Vasc Surg*. 2010 May;51(5):1145–51.
38. Dangas G, Laird JR Jr, Satler LF, et al. Postprocedural hypotension after carotid artery stent placement: predictors and short- and long-term clinical outcomes. *Radiology*. 2000;215: 677–683.
39. Park B, Shapiro D, Dahn M, Arici M. Carotid artery angioplasty with stenting and postprocedure hypotension. *Am J Surg*. 2005;190:691–695.
40. Mendelsohn FO, Weissman NS, Lederman RJ, et al. Acute hemodynamic changes during carotid artery stenting. *Am J Cardiol*. 1998:82:1077–1081.
41. Qureshi AI, Luft AR, Sharma M. Frequency and determinants of postprocedural homodynamic instability after carotid angioplasty and stenting. *Stroke.* 1999;30:2086–2093.
42. Trocciola SM, Chaer RA, Lin SC, et al. Analysis of parameters associated with hypotension requiring vasopressor support after carotid angioplasty and stenting. *J Vasc Surg*. 2006; 43:714–720.
43. Cayne NS, Faries PL, Trocciola SM, et al. Carotid angioplasty and stent-induced bradycardia and hypotension: impact of prophylactic atropine administration and prior carotid endarterectomy. *J Vasc Surg*. 2005;41:956–961.

14

Covered Stents for Subclavian-Axillary Artery Injuries

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Most subclavian and axillary injuries occur as a result of penetrating trauma. Proximal and distal control of the injured vessel involves extensive dissection,[1] including a combination of a supra- or infraclavicular incision, median sternotomy, and thoracotomy. As a result, postoperative morbidity and the risk of further injury to surrounding structures are significant. Recently, endovascular methods have been used to treat subclavian and axillary artery injuries using covered stents. [2–4] These endovascular techniques offer an alternative to direct dissection in the zone of injury. Case reports have indicated successful treatment of subclavian pseudoaneurysms using balloon and self-expandable stents.[2–5] Sullivan et al[6] reported the use of covered Palmaz stents for endovascular exclusion of axillary and subclavian pseudoaneurysms in three patients. Du Toit et al[7] described using the Hemobahn® endovascular prosthesis (W.L.Gore, Flagstaff, AZ) to treat 10 patients with penetrating injuries of the subclavian, carotid, and axillary arteries. This type of therapy is appealing for patients who frequently have multiple traumatic injuries or medical comorbidities, and are poor surgical candidates.

PATIENT SELECTION

Careful patient selection is necessary and only focal lesions that can safely be traversed with a guidewire should be approached in this fashion. An absolute lesion length criterion is not employed when deciding which approach is preferable. Ability to cross the lesion and its location in relationship to the vertebral artery are the major factors influencing whether endovascular techniques may be used. Patients with decreased life expectancy when long-term patency is not a primary concern are also candidates for a less invasive endovascular procedure. In addition, patients who are poor

candidates for general anesthesia are treated with this approach using intravenous sedation and local anesthesia. The procedure is performed in the endovascular suite with a fixed X-ray system or in the operating room with the C-arm.

TECHNIQUE

A femoral or a brachial artery approach (open or percutaneous) is used. A brachial cutdown provides better control of the guidewire and a more direct route to a lesion where a large part of the circumference of the vessel may have been disrupted.

Vessel size is not a decisive factor influencing the choice of open or endovascular repair. After the decision has been made to place a covered stent, the appropriate size sheath is inserted. Typically, covered stents require 8–9 Fr sheaths. The Viabahn® endovascular prosthesis (W.L.Gore, Flagstaff, AZ) or the Wallgraft® endoprosthesis (Boston Scientific, Quincy, MA) also may be used. The Viabahn® endoprosthesis is a self-expandable stent covered by ePTFE whereas the Wallgraft® is covered by Dacron. They both are coaxial systems and are inserted over an .035 inch guidewire. There is minimal foreshortening with the Viabahn® graft while some foreshortening has to be anticipated during deployment of a Wallgraft®. The size of the stent to be used is determined based on the extent of the lesion and the diameter of the native vessel. Intralumimal balloons and catheters of known length and diameter as well as the software of the imaging system can be used for stent sizing. One must be careful not to occlude the vertebral artery during placement. Also, in cases of proximal lesions, the stent should not protrude excessively in the thoracic aorta. Bony landmarks and roadmapping are used for precise deployment. Postdeployment balloon dilatation is used to improve coaptation of the stent and the vessel wall (Figure 14–1). Completion angiogram demonstrates the position of the stent, the condition of side branches, and how well the lesion is covered. A second stent can be placed if the first does not completely exclude the injury. Postoperative anticoagulation is not routinely used. Platelet inhibitors are advisable. Patients are followed with clinical examination and noninvasive vascular laboratory studies every three months for six months, and every six months thereafter.

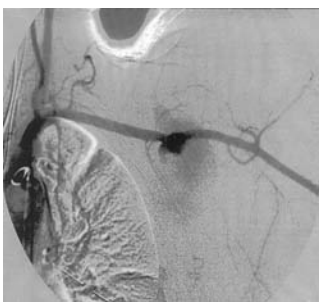

Figure 14-1A. This is the angiogram of a patient with a stab wound to the chest, resulting in an injury of the distal subclavian-proximal axillary artery.

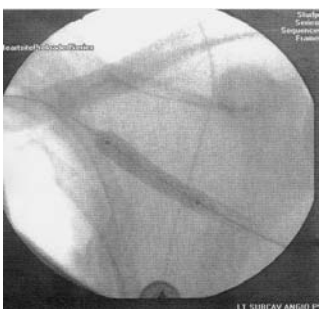

Figure 14-1B. A Wallgraft has been deployed across the lesion, and postdeployment balloon dilatation was performed.

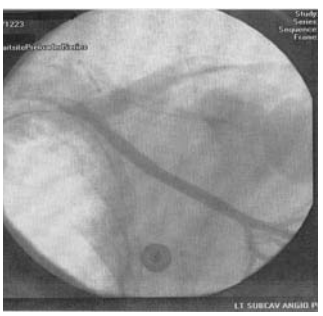

Figure 14-1C. Completion angiogram with the covered stent in place and no evidence of exrtravasation.

RESULTS

Endovascular covered stent placement eliminates the need for acute surgical dissection, decreasing the risk of injuring important adjacent structures such as the vagus and recurrent laryngeal nerve, the phrenic nerve, and the innominate vein. Short-term results of stent-graft repair as reported by Patel et al[8] and du Toit et al[7] are encouraging, but long-term durability has not been established. Shoder et al[9] reported 100% primary stent-graft patency at a mean of 11.6 months follow-up after treatment of 10 patients with subclavian artery aneurysms or iatrogenic injuries. Our experience[10] with the use of covered stents for treatment of iatrogenic, blunt, and penetrating subclavian and axillary injuries indicates that this method is a feasible alternative to open repair in properly selected patients resulting in shorter procedure time and less blood loss. In our series, one patient developed endograft thrombosis, resulting in arm ischemia several months after the initial procedure. She was electively treated with a saphenous vein subclavian bypasss. Stent thrombosis does not preclude future revascularization which, if necessary, can be done under less emergent circumstances after the acute injury has resolved.

Graft fracture[11] has been reported with stents or stent grafts in the subclavian artery or vein for thoracic outlet syndrome. Compression of the endoprosthesis between the clavicle and the first rib is most likely the cause of fracture in this location. The pathophysiology of occlusive disease or thoracic outlet syndrome is different from the pathophysiology of traumatic injuries. We have not encountered problems with stent fracture in our patients.

Recently, arterial closure devices have been used to address iatrogenic subclavian artery injuries. Wallace et al[12] and Berlet et al[13] have described using the Perclose and the Prostar XL (Abbot Laboratories, Redwood, CA, USA) device to approach inadvertent subclavian artery catheterization during central venous line placement. The efficacy and complications of this technique are still unknown.

Subclavian and axillary vessel injuries are infrequent and it is unlikely that a prospective randomized trial with sufficient number of patients will be conducted comparing the endovascular and open approach. In properly selected patients, covered stents offer an additional way of dealing with these challenging injuries. Nevertheless, this is a new technique and long-term results are under evaluation. Therefore, periodic patient follow-up and graft assessment will continue to be necessary.

REFERENCES

1. Abouljoud MS, Obeid FN, Horst H M, et al. Arterial injuries of the thoracic outlet: A ten-year experience. *Am Surg*. 1993;59:590–595.
2. Hernandez JA, Pershad A, Laufer N. Subclavian artery pseudoaneurysm: Successful exclusion with a covered self-expanding stent. *J Invas Cardiol*. 2002;14:278–279.
3. Pastores SM, Marin ML, Veith FJ, et al. Endovascular stented graft repair of a pseudoaneurysm of the subclavian artery caused by a perqutaneous internal jugular vein cannulation:case report. *Amer J Crit Care*. 1995;4:472–475.
4. Bartorelli A, Trabattoni D, Agrifoglio M, et al. Endovascular repair of iatrogenic subclavian artery perforations using the Hemobahn stent-graft, *J Endovasc Ther*. 2001;8:417–421.
5. Battaglia L, Bartolucci R, Minucci S, et al. Stent graft repair for rupture of the subclavian artery secondary to infection of subclavian -to-carotid bypass graft. *Ann Vasc Surg*. 2001;15: 474–476.

6. Sullivan M T, Bacharach MJ, Perl MD, Gray B. Endovascular management of unusual aneurysms of the axillary and subclavian arteries. *J Endovasc Surg*. 1996;3:389–395.
7. Du Toit D F, Strauss DC, Blaszzyk M, et al. Endovascular treatment of penetrating thoracic outlet arterial injuries. *Eur J Vasc Endovasc Surg*. 2000;19:489–495
8. Patel AV, Veith FJ, Kerr A, Sanchez LA. Endovascular graft repair of penetrating subclavian artery injuries. *J Endovasc Surg*. 1996;3:382–388.
9. Shoder M, Cejna M, Holzenbein T, Bischof G. Elective and emergent endovascular treatment of subclavian artery aneurysms and injuries. *J Endovasc Ther*. 2003;10:58–65
10. Xenos ES, Freeman M, Stevens S, et al. Covered stents for injuries of the subclavian and axillary arteries. *J Vasc Surg*. 2003;38:451–454.
11. Phipp LH,Scott JA, Kessel D,Robertson I. Subclavian stents and stent -grafts: Cause for concern? *J Endovasc Surg*.1999:223–226.
12. Wallace MJ, Ahrar K. Percutaneous closure of a subclavian artery injury after inadvertent catheterization. *J Vasc Interv Radiol*. 2001;12:1227–1230.
13. Berlet MH, Stefen D, Shaughness G, Hanner J. Closure using a surgical closure device of inadvertent subclavian artery punctures during central venous catheter placement. *Cardiovasc Intervent Radiol*. 2001;24:122–124.

15

Endovascular Treatment of Supra-aortic Arterial Trauma

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BACKGROUND

Vascular Trauma

Each year, over 30 million emergency room visits for traumatic injuries are seen, of which up to 4% are associated with vascular injury. Penetrating trauma predominates, with over 90% of vascular injuries related to penetrating and the remainder related to blunt mechanisms. Vascular injury can present with a spectrum of clinical findings, including external hemorrhage, extremity ischemia, internal hemorrhage, and expanding or pulsatile hematoma. Injury to a vascular structure can result in a range of effects on the vessel such as laceration, transection, contusion, arteriovenous fistula, or extrinsic compression. Laceration may allow extravasation of blood flow resulting in a pseudoaneurysm or internal or external hemorrhage. Contusion, laceration, transaction, or compression can result in arterial thrombosis or distal ischemia. Arteriovenous fistula can cause distal ischemia or, in some cases, high-output heart failure.

The clinical presentation of vascular trauma ranges from hemorrhagic shock to limb ischemia. The well-established hard and soft signs of vascular injury aid in guiding work-up and treatment. Hard signs of extremity vascular injury include absent distal pulses, active hemorrhage, ischemia, pulsatile hematoma, and bruit or thrill. Soft signs include decreased distal pulses, proximity of injury to vessels, neurologic deficit, and hypotension or shock. It is well established that routine arteriography for extremity injury in proximity to vascular structures is relatively low yield, and therefore, selective arteriography or exploration is recommended when hard signs of extremity vascular injury are noted.[1] In some cases, operative exploration may be performed unless arteriography is needed to localize or treat the injury.

Endovascular Therapy

Since the concept of endovascular stent grafting for the treatment of abdominal aortic aneurysms was initially described in 1991, this technique has been employed to treat various forms of arterial disease. In addition to aortic aneurysms, endovascular stent grafts have been used to treat arterial occlusive disease, prosthetic and autogenous graft occlusion, peripheral arterial aneurysms, and vascular trauma. Treatment of traumatic vascular injury using endovascular techniques has dramatically evolved as endovascular capabilities have advanced over the past several decades.

Open surgical repair of traumatic arterial injury is often complicated by distorted anatomy secondary to associated hematoma or pseudoaneurysm. The technical aspect of vascular exposure in these cases is often more challenging than in the elective setting. Several endovascular techniques have been employed in the treatment of traumatic arterial injury. These include coil embolization, bare metal stent placement, and covered stent graft placement. Both bare metal stents and covered stent grafts are available in balloon-expandable and self-expanding configurations. Coil embolization is generally reserved for the treatment of small traumatic pseudoaneurysms and arteriovenous fistulas involving nonessential vascular territories. In the treatment of arterial dissection, bare metal stent placement over the dissection entry point serves to reapproximate the intima, media, and adventitia, preventing continued flow into the false vessel lumen and propagation of a dissection flap. Although bare metal stents have proved useful in the repair of intimal flaps, initially described for use in this setting in 1991, they are less well suited for the treatment of arteriovenous fistulas or pseudoaneurysms.[2] Covered stents are more ideally suited for repairing these lesions as well as traumatic arterial transections. Compared with traditional surgical repair, endovascular repair of traumatic arterial injury has several advantages, including a reduction in anesthetic requirement, operative time, blood loss, and perioperative morbidity.[3] Endovascular therapy for traumatic arterial injury has the additional advantages of reduction in ischemia time, limited exposure, and the ability to use a remote access site. In general, endovascular intervention is useful when the morbidity associated with an operative approach is considered prohibitive. Endovascular repair of the aorta and supra-aortic branch vessels has afforded the greatest potential benefit, given that open surgical repair of these arteries often requires thoracotomy or sternotomy. The morbidity of these surgical approaches can be significant, especially in the patient with multiple injuries. In contrast, open surgical repair in most extremity arterial injuries is more straightforward in terms of operative exposure and repair.

This chapter focuses on endovascular repair of traumatic injury to the supra-aortic branches: the carotid, vertebral, and innominate, subclavian, and axillary arteries. Aortic, intra-abdominal, and extremity artery injury are not discussed in depth here.

LITERATURE

Few large-scale published series have evaluated outcomes associated with an endovascular strategy for repair of traumatic arterial injuries. Between January 1995 and December 2007, publications documenting the endovascular treatment of 302 patients with carotid, vertebral, innominate, subclavian, and axillary artery injuries were identified in the literature (Table 15–1). The overall technical success rate for endovascular stenting for repair of arterial injuries was 93.6%. Unsuccessful stent placement, which occurred infrequently, was

TABLE 15-1. SUMMARY OF LITERATURE ON ENDOVASCULAR INTERVENTION FOR ARTERIAL TRAUMA

Artery Injured	n	Mean Follow-up (Months)	Mean Age (Years)	Injury Type n (%)	Technical Success Rate (%)	Periprocedural Morbidity n (%)	Mortality n (%)
Carotid[2-45]	179	10	35	Blunt 137 (76.5%) Penetrating 39 (21.8%) Iatrogenic 7 (3.9%)	91.6%	6 (3.4%)	4 (2.2%)
Vertebral[4,14,41,46-54]	13	7.5	39	Blunt 2 (15.4%) Penetrating 11 (84.6%)	100%	0 (0%)	0 (0%)
Innominate[55-61]	7	5.4	32	Blunt 6 (85.7%) Penetrating 1 (14.3%)	85.7%	0 (0%)	0 (0%)
Subclavian[9,11,18,62-81]	91	17	50	Blunt 14 (15.4%) Penetrating 21 (23.1%) Iatrogenic 26 (28.6%)	96.7%	11 (12.1%)	3 (3.3%)
Axillary[47,66-67,82-86]	11	17	48	Blunt 6 (54.5%) Penetrating 2 (18.2%) Iatrogenic 3 (27.3%)	100%	1 (9.0%)	0 (0%)
Total	**301**	**13.3**	**40.1**	**Blunt 165** **Penetrating 74** **Iatrogenic 36**	**94.0%**	**18 (6.0%)**	**7 (2.3%)**

attributed to a variety of reasons, including the inability to traverse the lesion with a guidewire, persistent contrast extravasation after stent placement, or graft migration requiring either placement of coils or conversion to open repair.

Periprocedural morbidity was documented in 18 (6.0%) patients overall. Complications related directly to the endovascular procedure included iatrogenic brachial and femoral artery puncture site injury, in-stent stenosis, stroke, and retroperitoneal hematoma. Average patient follow-up was 13.3 months.

The overall mortality rate was 2.3% (n=7). Deaths were attributable to comorbid conditions associated with polytrauma, including stroke, hemorrhage from a remote site, sepsis, traumatic brain injury, and multisystem organ failure.

Carotid Artery Injury

Blunt carotid artery injury can result from several mechanisms. Hyperextension and lateral flexion of the neck can cause tethering of the internal carotid artery against the transverse process of the axis or lateral aspect of the atlas, resulting in arterial injury.[87] In motor vehicle collision-related carotid injury or forward movement of the patient's trunk while the head remains fixed may be possible mechanisms of injury.[88] Additional mechanisms of blunt carotid injury include intraoral trauma, a direct blow to the neck, strangulation, or injury in association with basilar skull fracture through the foramen lacerum.[89-90] Carotid artery trauma can result in arterial thrombosis, dissection, pseudoaneurysm or dissection, all of which may result in transient ischemic attack or stroke.

Blunt carotid injury accounts for less than 1% of all arterial trauma.[91-92] As many as 94% of cases are associated with a delay in diagnosis because the injury is often asymptomatic or is confounded by intoxication, intracranial injury, extremity or spinal cord injury, or shock.[93-98] The most common signs and symptoms of blunt carotid injury include ipsilateral headache (58-92%), cerebral ischemia (63-90%), Horner's syndrome (9-75%), neck pain (18-46%), and bruit (12-39%).[99] Owing to the delay in detection of this injury and the presence of concomitant injuries, the morbidity rate associated with carotid injury is high. Neurologic morbidity rates of 40 to 80% and mortality rates of 5 to 40% have been described.[92-93,99-100] In an attempt to decrease morbidity and mortality, screening parameters to allow for earlier diagnosis of carotid injury have been developed. Suggested triggers for work-up for carotid artery injury include cervical vertebral body or transverse process fracture, diffuse axonal injury, Horner's syndrome, Le Fort II or III facial fracture, and basilar skull fractures involving the carotid canal.[92,101-102]

Endovascular treatment of blunt carotid injuries can be particularly useful in selected patients. Treatment of carotid artery dissection with systemic anticoagulation alone has been described extensively and is associated with a recanalization rate of 50 to 70% and a 10% incidence of subsequent neurologic events.[103-105] Although the benefits of systemic anticoagulation in the setting of blunt carotid injury have been documented, contraindications to its use may exist in patients with concomitant head or intra-abdominal solid organ injury.[91,105] In this challenging patient population, stent placement may avoid or reduce the need for long-term systemic anticoagulation. Furthermore, endovascular techniques offer potential benefit in cases of distal internal carotid injury, in which surgical exposure is complicated by the need for extensive dissection or mandibular subluxation to gain exposure. Surgical repair of these injuries is associated with a 9% perioperative stroke rate.[10] Surgical repair of blunt carotid artery injury is rarely indicated, however. In one series of carotid injuries, only 1 of 76 patients

required surgical repair.[100] Indications for stent placement in the setting of carotid injury include contraindication to anticoagulation, enlarging pseudoaneurysm, progressive dissection, or lesions inaccessible through a high cervical operative approach.

Forty-two publications discussing 179 patients who sustained injuries to the carotid artery are summarized here. Blunt mechanism accounted for 137 (76.5%) of the arterial injuries, whereas 39 (21.8%) occurred due to a penetrating mechanism. There were 7 (3.9%) iatrogenic injuries. The overall technical success rate in these cases was 91.6%. There were 2 technical failures, 1 due to persistent endoleak[4] and 1 attributed to the inability to cross the injury with a guidewire.[5] Eleven patients suffered from carotid artery occlusion after stent placement.[6-9] None of these patients were symptomatic from the acute occlusion. Five patients suffered from a periprocedural stroke.[4,7,10] Four mortalities were reported.

The timing of repair of blunt carotid injury is somewhat controversial. Some interventionalists advocate delay of carotid stenting, thus decreasing the risk of thrombotic or embolic events related to catheter manipulation in the acutely injured vessel. The role of anticoagulation as both treatment for injury and prevention of acute stent thrombosis must be evaluated taking into consideration the patient's other injuries. Maintaining a partial thromboplastin time (PTT) between 40 to 50 seconds is thought to be sufficient anticoagulation in the treatment of blunt carotid artery injury when medical management is utilized.[91,106]

The management of periprocedural anticoagulation in this setting has evolved. This evolution has potential influence on arterial patency as evidenced by long-term patency rates in two of the larger series identified. Cothren and associates[7] placed carotid stents in 23 patients for traumatic dissection. On follow-up, 8 patients were found to have an asymptomatic occlusion of the carotid artery stent. In this setting, carotid occlusion could be attributed to factors such as accelerated neointimal hyperplasia or a hypercoagulable state in the younger trauma patient population, but it is likely also related to the management of systemic anticoagulation. In contrast, Edwards et al.[10] placed carotid stents in 22 patients for traumatic injuries, none of which demonstrated occlusion on follow-up. All of these patients received treatment with clopidogrel for a minimum of 6 weeks after stent placement. Some advocate the use of clopidogrel for at least 2 weeks following stent placement with conversion to lifelong aspirin therapy. Although long-term follow-up is limited, these studies suggest that outcome in patients undergoing carotid stent placement for traumatic lesions is improved with the post-procedural use of antiplatelet agents such as clopidogrel.

Duplex imaging is typically used for surveillance of carotid interventions in the atherosclerotic patient population. This standard of care generally mandates serial duplex scans for the lifetime of the patient. Although the comorbidities of the average trauma patient reduce the risk for the development of stenosis secondary to atherosclerosis, neointimal hyperplasia-related stenosis is possible and must be ruled out on serial imaging studies. Long-term outcomes related to stenting of the carotid artery for the repair of traumatic injuries have not been well established. Routine surveillance should mirror that for carotid stenting for atherosclerotic disease with duplex evaluation at 1, 3, 6, 12, 18, and 24 months and annually thereafter.

Vertebral Artery Injury

Few studies describe the use of endovascular means for treatment of vertebral artery injury. A total of 12 reports were found in the literature, including a description of 13 cases.

Seven of these cases reported arteriovenous fistulas that required intervention. Patients in this group had a mean age of 39 years and a mean follow-up of 7.5 months. No morbidities or mortalities were reported in this group of patients.

The most common presenting symptoms in patients with vertebral artery trauma were tinnitus and pain. These findings are consistent with the open surgical literature, which indicates that the majority of vertebral artery injuries can be managed nonoperatively or with embolization. The majority of these lesions are not life-threatening.[107] In the literature, one patient presented with a life-threatening injury. This patient had a sudden and significant decrease in mental status due to a vertebral artery dissection causing basilar artery occlusion. Although rarer, reports of patients presenting in shock due to vertebral artery injury are documented.[108]

Innominate, Subclavian, and Axillary Artery Injury

Seven reports of innominate artery injury and eight reports of 11 patients with axillary artery injury managed with endovascular techniques are available for review. No periprocedural morbidity was noted in the innominate group and 9.0% morbidity was noted in the axillary artery injury group.

Endovascular repair of subclavian artery injury is much more prevalent in the literature, with 23 reports describing 91 patients. Eleven complications occurred in the subclavian artery injury group (12.1%). These included pseudoaneurysms at the access site,[46,62] arm claudication occurring 3 months after stent placement,[11] stent fracture requiring placement of a second stent,[63-64] stent graft thrombosis,[62] and diminished distal pulses occurring 4 months after initial treatment requiring balloon angioplasty. Three deaths were documented in the subclavian artery injury group.[65] The periprocedural death rate was 1.1%, with 1 death occurring less than 30 days after the injury as a result of multisystem organ failure. Two deaths occurred longer than 30 days after the injury.

The choice of endoprosthesis used in treating injury to the innominate, subclavian, and axillary arteries varied by authors. For isolated dissection, the primary treatment method is placement of a bare metal stent.[66] The potential for stent compression, stent fracture, and in-stent stenosis is well documented, especially in arteries subject to strong mechanical forces.[66-68] All documented cases of in-stent stenosis occurred in the setting of covered stent graft placement. In regard to the choice of covered stent type, some advocate the use of Dacron-covered stents.[69] According to their rationale, trauma patients are notoriously noncompliant with follow-up, and therefore, physicians have difficulty ensuring that appropriate antiplatelet therapy is continued. For this reason, the authors support the use of Dacron because the more rapid neointimal response to the material may help to prevent graft thombosis in the patient who is noncompliant with antiplatelet therapy. The limited number of cases available for review makes outcome analysis with respect to specific covered endoprosthesis difficult. Although the mean follow-up in the innominate artery injury group was relatively short at 5.4 months, the mean follow-up for patients in which a stent or stent graft was placed for subclavian or axillary artery injury was 18 months and 13 months, respectively. During the follow-up period, 8 patients (8.8%) had documented stent fracture, stenosis, or occlusion. Three of the 8 patients with stent complications were asymptomatic. Given so few documented cases in the literature on the endovascular treatment of innominate, subclavian, and axillary artery injuries, more information is needed before the long-term durability of these repairs can be assessed.

Despite the above-mentioned limitations, stent grafting within the innominate and subclavian arteries provides short-term advantages. In the hemodynamically unstable patient, stent grafting eliminates the need for thoracotomy, sternotomy, and clavicular resection. In one report, 78% of their patients had either a serious illness or major trauma.[62] Furthermore, endovascular repair does not preclude future open repair and can thus be used as a bridge to definitive therapy in the hemodynamically unstable patient. Finally, concerns regarding stent fracture may be self-limiting when detected early and have the potential to be corrected. Three patients (3.3%) who developed a stent fracture or kink were successfully revised using endovascular devices.[62-64]

CONCLUSION

Endovascular intervention for repair of arterial trauma involving the supra-aortic vessels can be performed safely and effectively. An overall technical success rate upward of 90% and morbidity and mortality rates of 3.0 and 2.3 respectively, demonstrate a potential benefit compared with traditional open surgical repair. The most significant limitation to the evaluation of endovascular stenting for vascular trauma is the lack of long-term data and controlled trials comparing outcomes with those with open surgical repair. Long-term follow-up for stent durability is of particular concern in the trauma population, which tends to comprise younger patients with minimal atherosclerotic disease compared with the typical older patient treated by endovascular means for aneurysmal or occlusive disease. Another concern is the long-term effect of the stent on the vessel wall, with the radial force exerted by the stent having possible adverse effects on the arterial wall, which may already be fragile due to injury. Long-term follow-up of patients treated by endovascular techniques for arterial trauma during the past decade will provide useful information regarding the durability and complications that may result. Routine surveillance imaging for graft migration, kinking, endoleak, delayed rupture, and vascular or neurologic complications related to the stent should be closely monitored in these cases.

Other limitations of endovascular techniques include reliance on the availability of suitably skilled interventionalists and an angiography suite that can also be used as an operating room when endovascular attempts at repair fail and conversion to open repair is required. Positive outcomes also rely on the availability of a variety of stents and stent graft devices, which allow a more precise tailoring to the anatomy of the vessels and the injury. An additional limiting factor in the endovascular treatment of arterial injury is the presence of large pseudoaneurysms that may require open decompression because of impingement of adjacent structures that is unrelieved by endovascular stent graft placement. In these cases, endovascular repair may be used in combination with open hematoma decompression. In summary, the use of endovascular intervention for the treatment of arterial trauma should not be considered a replacement for open surgical repair but rather an adjunct in the armamentarium of the vascular interventionalist who treats arterial traumatic injuries.

REFERENCES

1. Dennis JW, Frykberg ER, Veldenz HC, et al. Validation of nonoperative management of occult vascular injuries and accuracy of physical examination alone in penetrating extremity trauma: 5 to 10-year follow-up. J Trauma 1998;44:243–53.

2. Lyden S, Srivastava SD, Waldman DL, et al. Common iliac artery dissection after blunt trauma: case report of endovascular repair and literature review. J Trauma 2001;50:339–42.
3. Marin ML, Veith FJ, Panetta TF, et al. Transluminally placed endovascular stented graft repair for arterial trauma. J Vasc Surg 1994;20:466–73.
4. Sakat RR, Razavi MK, Sze DY, et al. Stent-graft treatment of extracranial carotid and vertebral arterial lesions, J Vasc Interv Radiol 2004;15:1151–6.
5. Szopinski P, Ciostek P, Kielar M, et al. A series of 15 patients with extracranial carotid artery aneurysms: surgical and endovascular treatment. Eur J Vasc Endovasc Surg 2005; 29:256–61.
6. Archondakis E, Pero G, Valvassori L, et al. Angiographic follow-up of traumatic carotid cavernous fistulas treated with endovascular stent graft placement. AJNR Am J Neuroradiol 2007;28:342–7.
7. Cothren CC, Moore, EE, Ray CE, et al. Carotid artery stents for blunt cerebrovascular injury. Arch Surg 2005;140:480–6.
8. Duane TM, Parker F, Stokes GK, et al. Endovascular carotid stenting after trauma. J Trauma 2002;52:149–53.
9. Parodi JC, Schonholz C, Ferriera LM, Bergan J. Endovascular stent-graft treatment of traumatic arterial lesions. Ann Vasc Surg 1999;13:121–9.
10. Edwards NM, Fabian TC, Claridge JA, et al. Antithrombotic therapy and endovascular stents are effective treatment for blunt carotid injuries: results from longterm followup. J Am Coll Surg 2007;204:1007–15.
11. du Toit DF, Leith JG, Strauss DC, et al. Endovascular management of traumatic cervicothoracic arteriovenous fistula. Br J Surg 2003;90:1516–21.
12. Ahn JY, Chung YS, Lee BH, et al. Stent-graft placement in traumatic internal carotid-internal jugular fistula and pseudoaneurysm. J Clin Neurosci 2004;11:636–9.
13. Akiyam Y, Nakahara I, Tanaka M, et al. Urgent endovascular stent-graft placement for a ruptured traumatic pseudoaneurysm of the extracranial carotid artery. J Trauma 2005;58:624–7.
14. Amar AP, Teitelbaum GP, Giannotta SL, et al. Covered stent-graft repair of the brachiocephalic arteries: technical note. Neurosurgery 2002;51:247–53.
15. Bejjani GK, Monsein LH, Laird JR, et al. Treatment of symptomatic cervical carotid dissections with endovascular stents. Neurosurgery 1999;44:755–61.
16. Bernstein SM, Coldwell DM, Prall JA, Brega KE. Treatment of traumatic carotid pseudoaneurysm with endovascular stent placement. J Vasc Interv Radiol 1997;8:1065–8.
17. Biffl WL, Moore EE, Offner PJ, et al. Blunt carotid arterial injuries: implications of a new grading scale. J Trauma 1999;47:845–53.
18. Brandt M, Kazanjian S, Wahl W. The utility of endovascular stents in the treatment of blunt arterial injuries. J Trauma 2001;51:901–5.
19. Cohen JE, Ben-Hur T, Gomori JM, et al. Stent-assisted arterial reconstruction of traumatic extracranial carotid dissections. Neurol Res 2005;27:S73–8.
20. Coldwell DM, Novak Z, Ryu RK, et al. Treatment of posttraumatic internal carotid arterial pseudoaneurysms with endovascular stents. J Trauma 2000;48:470–2.
21. Diaz-Daza O, Arriaza FJ, Barkley JM, et al. Endovascular therapy of traumatic vascular lesions of the head and neck. Cardiovasc Intervent Radiol 2003;26:213–21.
22. Duncan IC, Rad FF, Fourie PA. Percutaneous management of concomitant post-traumatic high vertebral and caroticojugular fistulas using balloons, coils, and covered stent. J Endovasc Ther 2003;10:882–6.
23. Duke BJ, Ryu RK, Coldwell DM, et al. Treatment of blunt injury to the carotid artery by using endovascular stents. J Neurosurg 1997;87:825–9.
24. Fateri F, Groebli Y, Rufenacht DA, et al. Intraarterial thrombolysis and stent placement in the acute phase of blunt internal carotid artery trauma with subocclusive dissection and thromboembolic complication: case report and review of the literature. Ann Vasc Surg 2005;19:1–4.
25. Feugier P, Vulliez A, Bina N, et al. Urgent endovascular covered-stent treatment of internal carotid artery injury caused by a gunshot. Eur J Vasc Endovasc Surg 2007:34;663–5.

26. Fusonie GE, Edwards JD, Reed AB. Covered stent exclusion of blunt traumatic carotid artery pseudoaneurysm: case report and review of the literature. Ann Vasc Surg 2004;18: 376–9.
27. Joo JY, Ahn JY, Chung YS, et al. Therapeutic endovascular treatments for traumatic carotid artery injuries. J Trauma 2005;58:1159–66.
28. Kerby JD, May AK, Gomez CR, et al. Treatment of bilateral blunt carotid injury using percutaneous angioplasty and stenting: case report and review of the literature. J Trauma 2000;49:784–7.
29. Klein GE, Szolar DH, Raith J, et al. Posttraumatic extracranial aneurysm of the internal carotid artery: combined endovascular treatment with coils and stents. AJNR Am J Neuroradiol 1997;18:1261–4.
30. Kubaska SM, Greenberg RK, Clair D, et al. Internal carotid artery pseudoaneuryms: treatment with the wallgraft endoprosthesis. J Endovasc Ther 2005;10:182–9.
31. Layton KF, Kim YW, Hise JH. Use of covered stent grafts in the extracranial carotid artery: report of three patients with follow-up between 8 and 42 months. AJNR Am J Neuroradiol 2004;25:1760–3.
32. Lee CY, Yim MB, Kim IM, et al. Traumatic aneurysm of the supraclinoid internal carotid artery and an associated carotid-cavernous fistula: vascular reconstruction performed using intravascular implantation of stents and coils. J Neurosurg 2004;100:115–9.
33. Liu AY, Paulsen RD, Marcellus ML, et al. Long-term outcomes after carotid stent placement for treatment of carotid artery dissection. Neurosurgery 1999;45:1368.
34. Malek AM, Higashida RT, Phatouros CC, et al. Endovascular management of extracranial carotid artery dissection achieved using stent angioplasty. AJNR Am J Neuroradiol 2000;21: 1280–92.
35. Marotta TR, Buller C, Taylor D, et al. Autologous vein-covered stent repair of a cervical internal carotid artery pseudoaneurysm: technical case report. Neurosurgery 1998;42: 408–13.
36. Matsuura JH, Rosenthal D, Jerius H, et al. Traumatic carotid artery dissection and pseudoaneurysm treated with endovascular coils and stent. J Endovasc Surg 1997;4:339–43.
37. McNeil JD, Chiou AC, Gunlock MG, et al. Successful endovascular therapy of a penetrating zone III internal carotid injury. J Vasc Surg 2002;36:187–90.
38. Parikh AA, Luchette FA, Valente JF, et al. Blunt carotid artery injuries. J Am Coll Surg 1997;185:80–6.
39. Patel JV, Rossbach MM, Cleveland TJ, et al. Endovascular stent-graft repair of traumatic carotid artery pseudoaneurysm. Clin Radiol 2002;57:308–11.
40. Perez-Cruet MJ, Patwardhan RV, Mawad ME, et al. Treatment of dissecting pseudoaneurysm of the cervical internal carotid artery using a wall stent and detachable coils: case report. Neurosurgery 1997;40:622–6.
41. Redekop G, Weill MT, Treatment of traumatic aneurysms and arteriovenous fistulas of the skull base by using endovascular stents. J Neurosurg 2001;95:412–9.
42. Reiter BP, Marin ML, Teodorescu VJ, Mitty HA. Endoluminal repair of an internal carotid artery pseudoaneurysm. J Vasc Interv Radiol 1998;9:245–8.
43. Scavee V, De Wispelaere JF, Mormont E, et al. Pseudoaneurysm of the internal carotid artery: treatment with a covered stent. Cardiovasc Interv Radiol 2001;24:283–5.
44. Self ML, Mangram A, Jefferson H, et al. Percutaneous stent-graft repair of a traumatic common carotid-internal jugular fistula and pseudoaneurysm in a patient with cervival spine fractures. J Trauma 2004;57:1331–4.
45. Shames ML, Davis JW, Evans AJ. Endoluminal stent placement for the treatment of traumatic carotid artery pseudoaneurysm: case report and review of the literature. J Trauma 1999;46:724–6.
46. Waldmen DL, Barquist E, Poynton FG, Numaguchi Y. Stent graft of a traumatic vertebral artery injury: case report. J Trauma 1998;44:1094–7.
47. Gonzalez A, Mayol A, Gil-Peralta, Gonzalez-Marcos JR. Endovascular stent-graft treatment of an iatrogenic vertebral arteriovenous fistula. Neuroradiology 2001;43:784–6.

48. Horowitz MB, Miller G 3rd, Meyer Y, et al. Use of intravascular stents in the treatment of internal carotid and extracranial vertebral artery pseudoaneurysms. AJNR Am J Neuroradiol 1996;17:693–6.
49. Mourikis D, Chatziioanou A, Doriforou O. Endovascular treatment of a vertebral artery pseudoaneurysm in a drug user. Cardiovasc Intervent Radiol 2006;29:662–4.
50. Price RF, Sellar R, Leung C, O'Sullivan MJ. Traumatic vertebral arterial dissection and vertebrobasilar arterial thrombosis successfully treated with endovascular thrombolysis and stenting. AJNR Am J Neuroradiol 1998;19:1677–80.
51. Priestly R, Bray P, Bray A, Hunter J. Iatrogenic vertebral arteriovenous fistula treated with a hemobahn stent-graft. J Endovasc Ther 2003;10:657–63.
52. Ruckert RI, Rutsch W, Filimonow S, Lehman R. Successful stent-graft repair of a vertebrojugular arteriovenous fistula. J Endovasc Ther 2001;8:495–500.
53. Singer RJ, Dake MD, Norbash A. Covered stent placement for neurovascular disease. AJNR Am J Neuroradiol 1997;18:507–9.
54. Surber R, Werner GS, Cohnert TU, et al. Recurrent vertebral arteriovenous fistula after surgical repair: treatment with a self-expanding stent-graft. J Endovasc Ther 2003;10:49–53.
55. Axisa BM, Loftus IM, Fishwick G, et al. Endovascular repair of an innominate artery false aneurysm following blunt trauma. J Endovasc Ther 2000;7:245–50.
56. Blatman SB, Landis GS, Knight M, et al. Combined endovascular and open repair of a penetrating innominate artery and tracheal injury. Ann Thorac Surg 2002;74:237–9.
57. Chandler TA, Fishwick G, Bell PRF. Endovascular repair of a traumatic innominate artery aneurysm. Eur J Vasc Endovasc Surg 1999;18:80–2.
58. Miles EK, Blake A, Thompson W, et al. Endovascular repair of acute innominate artery injury due to blunt trauma. Am Surg 2003;69:155–9.
59. Reubben A, Merlo M, Verri A, et al. Combined surgical and endovascular treatment of a traumatic pseudoaneurysm of the brachiocephalic trunk with anatomic anomaly. J Cardiovasc Surg 1997;38:173–6.
60. Waldenberger P, Fraedrich G, Mallouhi A, et al. Emergency endovascular treatment of traumatic aortic arch rupture with multiple arch vessel involvement. J Endovasc Ther 2003;10: 728–32.
61. Zoffoli G, Saccani S, Larini P, et al. Endovascular treatment of traumatic aortic dissection and innominate artery pseudoaneurysm. J Trauma 2006;61:447–50.
62. Hilfiker PR, Razavi MK, Kee ST, et al. Stent-graft therapy for subclavian artery aneurysms and fistulas: single-center mid-term results. J Vasc Interv Radiol 2000;11:578–84.
63. Ohki T, Veith FJ, Kraas C, et al. Endovascular therapy for upper extremity injury. Semin Vasc Surg 1998;11:106–15.
64. Patel AV, Marin ML, Veith FJ, et al. Endovascular graft repair of penetrating subclavian artery injuries. J Endovasc Surg 1996;3:382–8.
65. White R, Krajcer Z, Johson M, et al. Results of a multicenter trial for the treatment of traumatic vascular injury with a covered stent. J Trauma 2006;60:1189–96.
66. Castelli P, Caronno R, Piffaretti G, et al. Endovascular repair of traumatic injuries of the subclavian and axillary arteries. Injury 2005;36:778–82.
67. Piffaretti G, Tozzi M, Lomazzi C, et al. Endovascular treatment for traumatic injuries of the peripheral arteries following blunt trauma. Injury 2007;38:1091–7.
68. Schoder M, Cejna M, Holzenbein, et al. Elective and emergent endovascular treatment of subclavian artery aneurysms and injuries. J Endovasc Ther 2003;10:58–65.
69. Bates MC, Campbell J. Emergent stent graft isolation of a knife-related subclavian arterial venous fistula: lessons learned during long-term follow-up. Catheter Cardiovasc Interv 2005; 66:483–6.
70. Babatasi G, Massetti M, Bhoyroo S, et al. Non-penetrating subclavian artery trauma: management by selective transluminally placed stent device. Thorac Cardiovasc Surg 1999;47: 190–3.
71. Criado E, Marston WA, Ligush J, et al. Endovascular repair of peripheral aneurysms, pseudoaneurysms, and arteriovenous fistulas. Ann Vasc Surg 1997;11:256–63.

72. Meyer T, Merkel S, Lang W. Combined operative and endovascular treatment of a post-traumatic embolizing aneurysm of the subclavian artery. J Endovasc Surg 1998;5:52–5.
73. Pfammater T, Kunzli T, Hilfliker PR, et al. Relief of subclavian venous and brachial plexus compression syndrome caused by traumatic subclavian artery aneurysm by means of transluminal stent-grafting. J Trauma 1998;45:972–4.
74. Renger RJ, de Bruijn AJ, Aarts HC, et al. Endovascular treatment for pseudoaneurysm of the subclavian artery. J Trauma 2003;55:969–71.
75. Sanchez LA, Veith FJ, Ohiki T, et al. Early experience with the Corvita endoluminal graft for treatment of arterial injuries. Ann Vasc Surg 1999;13:151–7.
76. Sivamurthy N, Eichler C, Schneider DB. Endovascular eclusion of subclavian artery pseudoaneurysm. Vascular 2006;14:231–5.
77. Stecco K, Meier A, Seiver A, et al. Endovascular stent-graft placement for treatment of traumatic penetrating subclavian artery injury. J Trauma 2000;48:948–50.
78. Stokkeland PJ, Soreide K, Fjetland L. Acute endovascular repair of right subclavian arterial perforation from clavicular fracture after blunt trauma. J Vasc Interv Radiol 2007;18: 689–90.
79. Strauss DC, du Toit DF, Warren BL. Endovascular repair of occluded subclavian arteries following penetrating trauma. J Endovasc Ther 2001;8:529–33.
80. Watelet J, Clavier E, Reiz T, et al. Traumatic subclavian artery pseudoaneurysm: periprocedural salvage of failed stent-graft exclusion using coil embolization. J Endovasc Ther 2001;8:197–201.
81. Ahn SH, Cutry A, Murphy TP, Slaiby JM. Traumatic thoracic aortic rupture: treatment with endovascular graft in the acute setting. J Trauma 2001;50:949–51.
82. Martinez R, Lermusiaux P, Podeur L, et al. Endovascular management of axillary artery trauma. J Cardiovasc Surg 1999;40:413–5.
83. Onal, Baran, Ilgit ET, Kosar S, Akkan K, et al. Endovascular treatment of peripheral vascular lesions with stent-grafts. Diag Interv Radiol 2005;11:170–4.
84. Stahnke M, Duddy MJ, Endovascular repair of a traumatic axillary pseudoaneurysm following anterior shoulder dislocation. Cardiovasc Intervent Radiol 2006;29:298–301.
85. Turner PJ, Turon EP, Kessel D. Post-traumatic axillary artery pseudoaneurysm: treatment with a covered endoluminal stent. J Interv Radiol 1997;12:154–5.
86. Valentin MD, Tulsyan N, James K. Endovascular management of traumatic axillary artery dissection: a case report and review of the literature. Vasc Endovasc Surg 2004;38:473–5.
87. Parikh AA, Luchette FA, Valente JF, et al. Blunt carotid artery injuries. J Am Coll Surg 1997;185:80–6.
88. Perez-Cruet MJ, Patwardhan RV, Mawad ME, et al. Treatment of dissecting pseudoaneurysm of the cervical internal carotid artery using a wall stent and detachable coils: case report. Neurosurgery 1997;40:622–6.
89. Krajewski LP, Hertzer NR. Blunt carotid artery trauma: report of 2 cases and review of the literature. Ann Surg 1980;191:341–6.
90. New PF, Momose KJ. Traumatic dissection of the internal carotid artery at the atlantoaxial level, secondary to nonpenetrating injury. Radiology 1969;93:41–9.
91. Fabian TC, Patton JH Jr, Croce MA, et al. Blunt carotid injury: importance of early diagnosis and anticoagulant therapy. Ann Surg 1996;223:513–25.
92. Davis JW, Holbrook TL, Hoyt DB, et al. Blunt carotid artery dissection: incidence, associated injuries, screening, and treatment. J Trauma 1990;30:1514–7.
93. Cogbill TH, Moore EE, Meissner M, et al. The spectrum of blunt injury to the carotid artery: a multicenter perspective. J Trauma 1994;37:473–9.
94. Kraus RR, Bergstein JM, DeBord JR. Diagnosis, treatment, and outcome of blunt carotid arterial injuries. Am J Surg 1999;178:190–3.
95. Zetterling M, Carlstrom C, Konrad P. Review article: internal carotid artery dissection. Acta Neurol Scand 2000;101:1–7.
96. Mokri B, Houser OW, Sandok BA, et al. Spontaneous dissections of the vertebral arteries. Neurology 1988;38:880–5.

97. Biousse V, D'Angelajan-Chatillon J, Touboul PJ, et al. Time course of symptoms in extracranial carotid artery dissections. A series of 80 patients. Stroke 1995;26:235–9.
98. DiPerna CA, Rowe VL, Terramani TT, et al. Clinical importance of the "seat belt sign" in blunt trauma to the neck. Am Surg 2002;68:441–5.
99. Singh RR, Barry MC, Ireland A, et al. Current diagnosis and management of blunt internal carotid artery injury. Eur J Vasc Endovasc Surg 2004;27:577–84.
100. Biffl WL, Moore EE, Offner PJ, et al. Blunt carotid arterial injuries: implications of a new grading scale. J Trauma 1999;47:845–53.
101. Duke BJ, Ryu RK, Coldwell DM, et al. Treatment of blunt injury to the carotid artery by using endovascular stents. J Neurosurg 1997;87:825–9.
102. Biffl WL, Moore EE, Offner PJ, Burch JM. Blunt carotid and vertebral injuries. World J Surg 2001;25:1036–43.
103. Watridge CB, Muhlbauer MS, Lowery RD. Traumatic carotid artery dissection: diagnosis and treatment. J Neurosurg 1989;71:854–7.
104. Pozzati E, Giuliani G, Acciarri N, Nuzzo G. Long-term follow up of occlusive cervical carotid dissection. Stroke 1990;21:528–31.
105. Wahl WL, Brandt MM, Thompson BG, et al. Antiplatelet therapy: an alternative to heparin for blunt carotid injury. J Trauma 2002;52:896–901.
106. Biffl WL, Moore EE, Ray C, Elliott JP. Emergent stenting of acute blunt carotid artery injuries: a cautionary note. J Trauma 2001;50:969–70.
107. Moore EE, Feliciano DV, Mattox KL, eds. Trauma. 5th ed. New York: McGraw-Hill; 2004.
108. Reid JD, Weigelt JA. Forty-three cases of vertebral artery trauma. J Trauma 1988;28:1007–12.

SECTION III

Endovascular Interventions in Infrainguinal Lesions

16

Long-Term Results of Combined Common Femoral Endarterectomy and Iliac Stent/Stent Grafting for Occlusive Disease

Philip P. Goodney, M.D. and Richard J. Powell, M.D.

INTRODUCTION

Endarterectomy with patch angioplasty has become the standard for treatment of isolated common femoral artery (CFA) occlusive disease.[1-2] The management of disease that extends proximally into the external iliac arteries is a more challenging problem. According to the recently modified guidelines from the Trans Atlantic Society Consensus (TASC II) document, external iliac disease involving the CFA is now classified as either TASC C or D, depending on the extent of iliac involvement. For TASC D lesions, open surgical bypass remains the recommended treatment, with consideration of endovascular options only for TASC C lesions in poor-risk patients.[3] Standard open surgical therapies include iliofemoral endarterectomy or aortofemoral bypass. Although durable, these options are associated with increased perioperative morbidity.[4-6] Less invasive but also less durable open surgical options include axillobifemoral bypass or femoro-femoral bypass for unilateral disease. In the endovascular era, endoluminal treatment has been shown in certain situations to be a comparable option for iliac occlusive disease.[7-21] However, it is not a suitable stand-alone therapy in the presence of significant common femoral artery disease.[22]

Here we describe our experience with combined common femoral endarterectomy and external iliac artery (EIA) stenting as a means to treat TASC C and D iliac lesions using both bare metal stents and stent grafts for iliac artery intervention.

Preoperative Evaluation

All patients undergo preoperative peripheral vascular evaluation with physical examination, ankle-brachial indexes (ABIs), and duplex ultrasound, computed tomographic angiography (CTA), or digital subtraction angiography (DSA) to assess iliac and CFA disease

burden. Patients with significant CFA occlusive disease with proximal extension into the EIA are considered for the combined procedure. The presence of significant CFA disease is defined by greater than 2.5 times step-up increase in peak systolic velocity (PSV) across the CFA with duplex ultrasound scan, more than 50% diameter reduction with DSA or CTA, or more than 10 mm Hg systolic pressure gradient at the time of angiography. Selected patients with contralateral or distal disease requiring revascularization undergo various adjunctive procedures such as angioplasty/stenting, femoral-distal bypass, or femorofemoral crossover bypass as needed.

PROCEDURAL TECHNIQUE

Standard femoral arterial exposure is gained under general anesthesia. Needle-guided retrograde guidewire access is then obtained under fluoroscopic guidance. Standard endarterectomy and patch angioplasty are then performed with the guidewire in place. Efforts are undertaken to ensure that adequate profunda femoris artery outflow is preserved or restored. The wire is then back-fed through the center of the patch, and a retrograde working sheath is placed once inflow has been restored (Figure 16–1). Standard interventional techniques are then used to treat the proximal arterial lesion. If guidewire access cannot be obtained initially, it is then attempted after arteriotomy or endarterectomy. Rarely, contralateral iliac artery or brachial artery access is required for antegrade wire access. In recent years, re-entry devices are occasionally used for crossing chronic total

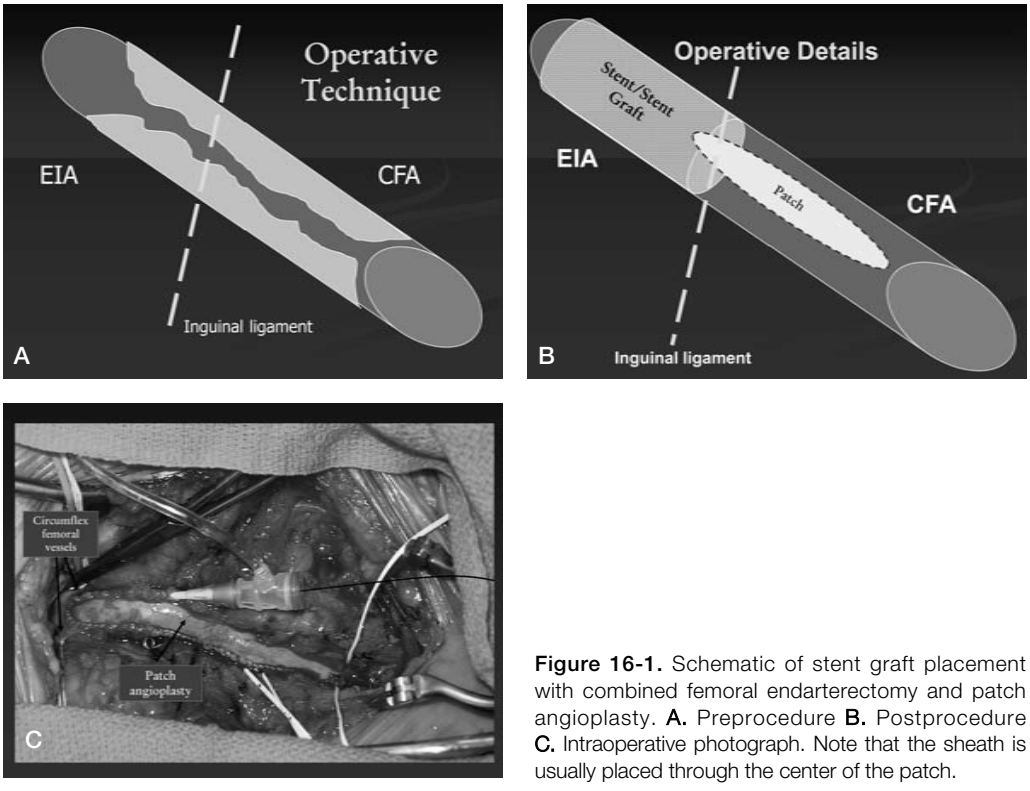

Figure 16-1. Schematic of stent graft placement with combined femoral endarterectomy and patch angioplasty. **A.** Preprocedure **B.** Postprocedure **C.** Intraoperative photograph. Note that the sheath is usually placed through the center of the patch.

occlusions of the iliac system (Pioneer Reentry Catheter, Medtronic, Santa Rosa, CA; Outback Reentry Catheter, Cordis, Miami Lakes, FL).

Arterial diameter and lesion measurements are made using preoperative CT scan measurements or calibrated using a marker catheter and fluoroscopy imaging software. The choice of device and postdeployment balloon diameter depends upon the severity of iliac artery calcification and lesion location. We generally use self-expanding stents (Wallstent, Boston Scientific, Boston, MA; Symphony, Boston Scientific, Boston, MA; SMART, Cordis, Miami Lakes, FL; Absolute, Abbott Vascular, Abbott Park, IL; Luminexx, Bard, Tempe, AZ), self-expanding stent grafts (Viabahn, W.L. Gore, Flagstaff, AZ; Fluency, Bard, Lowell, MA; Wallgraft, Boston Scientific, Boston, MA) and less frequently balloon-expandable stents (Express, Boston Scientific, Boston, MA; Palmaz, Cordis, Miami Lakes, FL) and balloon-expandable stent grafts (Icast, Atrium Medical, Hudson, NH) are used. Self-expanding devices are generally oversized by 1 to 2 mm relative to the native treated vessel. The diameter of the angioplasty balloon usually corresponds to the normal vessel size as seen on adjacent angiography or CTA. As previously described, we place the distal end of the device into the proximal portion of the patch angioplasty but above the inguinal ligament (Figure 16–2). The proximal portion of our endarterectomy and endpoint for the patch angioplasty are usually at the level of the circumflex vessels. This generally requires at most minimal division of the inguinal ligament.

RESULTS

We have now performed this procedure in over 200 patients. Patient demographics are shown in Table 16–1.

In all patients, CFA endarterectomy was performed with patch angioplasty. In 39% of cases, the EIA is the only iliac segment treated (TASC C). In 61% of cases, both the EIA and the CIA (TASC D) were treated. Forty-one percent of treated iliac vessels were occluded at the time of intervention. In 67% of cases, the SFA and profunda were patent at the completion of the procedure; in 30% of cases, only the profunda was patent; and in 3% of cases, only the SFA was patent at endarterectomy completion. There were 21 (11%) concomitant distal bypasses, 6 (3%) SFA stents, and 25 (13%) femorofemoral bypass grafts performed.

TABLE 16-1. PATIENT DEMOGRAPHICS

193 Limbs	171 Patients
Age (years)	67 (44-88)
Gender	62% male
HTN	85%
CAD	63%
Hyperlipidemia	63%
Current smokers	49%
Statin use	49%
DM	35%
COPD	31%
Renal disease (Cr > 1.6)	11%

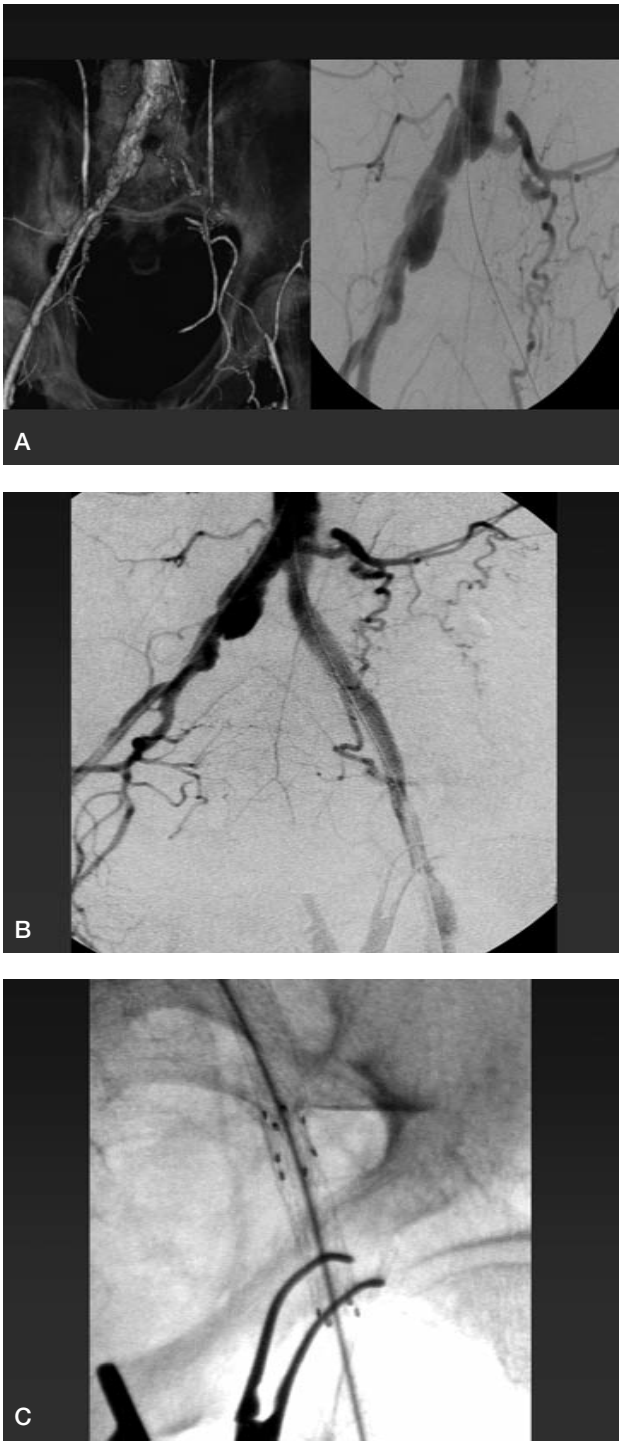

Figure 16-2. A. Total left iliac occlusion. Guidewire access obtained with retrograde sheath and re-entry device (Pioneer catheter). **B.** Stent graft treatment of iliac segment. **C.** Note extension of stent grafts into common femoral patch that is marked by vascular clamp.

Ninety percent of the iliac interventions were performed via retrograde access from the ipsilateral CFA. In 31% of cases, treatment of the CIA and EIA involved placing a bare metal stent across a patent hypogastric artery. In 3 cases, a stent graft was intentionally placed across a patent hypogastric artery. The technical success rate was 98%. There were 11 different stent or stent grafts used during the study period. During the latter part of the study period, more stent grafts were used for this application. Overall, stent grafts were used in 41% of cases.

On average, two devices (range 1 to 5) were placed in a treated EIA or EIA/CIA segment. The mean stent diameter was 8.3 mm±1.1 mm, with a mean postdilation balloon diameter of 7.4 mm±1.0 mm. The average preintervention pressure gradient in lesions that were not total occlusions was 31 mm Hg±18 mm Hg, which was reduced to 1 mm Hg±3 mm Hg after intervention. The mean preoperative ABI was 0.38±0.32, and the mean postoperative ABI was 0.72±0.24 ($P<.05$). Ninety-two percent of patients were clinically improved following the procedure. The median length of stay was 2 days (range 1 to 51 days). Median follow-up on all patients was 24 months (range 0 to 9 years).

Overall, there were 64 (33%) patients with hemodynamically significant recurrent disease. Of these, 45 patients experienced recurrent symptoms, and 19 patients remained asymptomatic but had a decrease in ABI or a diminished femoral pulse and were subsequently found to have recurrent stenoses. Twenty-eight patients (14%) eventually required a percutaneous intervention on the treated segment, and 19 patients (10%) underwent a subsequent open procedure on the treated segment. The mean time to reintervention was 21 months. Of the percutaneous reinterventions to maintain patency, 7 cases involved angioplasty of in-stent restenosis, 14 cases required restenting of the previously treated area, and 7 patients underwent more extensive stenting beyond the target lesion. Of the 19 open procedures performed, there were 6 redo endarterectomies, 4 redo endarterectomies with restenting, 1 open angioplasty, 3 redo endarterectomies with concomitant distal bypass, and 5 CFA thrombectomies with iliac recanalization or femorofemoral bypass.

Five-year survival by life-table analysis was 60±6% (Figure 16–3). Overall, there were 9 major amputations in the study group during the follow-up period.

Overall primary patency rate of the treated segment, as determined by Kaplan-Meier life-table analysis, was 60±6% at 5 years. Primary-assisted patency was 97±1% at 5 years. Secondary patency was noted to be 98±2% at 5 years.

Primary patency was significantly higher in patients receiving stent grafts versus bare metal stents (OR 0.19, 95% CI 0.08-0.45, $P<.001$). There were no additional patient or technical factors associated with failure of primary patency, including SFA, profunda, and tibial vessel patencies.

Subgroup analysis of the stent graft group versus the stent group showed significantly higher primary patency at 5 years in the stent graft group (87±5%, CI 72–94% vs. 53±7%, CI 39.5–65.2%, $P<.01$) (Figure 16–4). There were no significant differences found with primary-assisted and secondary patency rates between the groups.

There were 4 (2.3%) perioperative deaths. One patient suffered perioperative cardiogenic shock and died on the seventh postoperative day; 2 patients died from multisystem organ failure at postoperative days 18 and 22, respectively; and 1 patient died of unknown cause at home 3 days after surgery. There were 42 (22%) perioperative complications, consisting of 8 (4%) myocardial ischemic events, 5 (3%) pulmonary events, 25 (13%) wound infections, and 4 (2%) perioperative (within 30 days) thrombotic events requiring re-exploration. Three of these acute patient

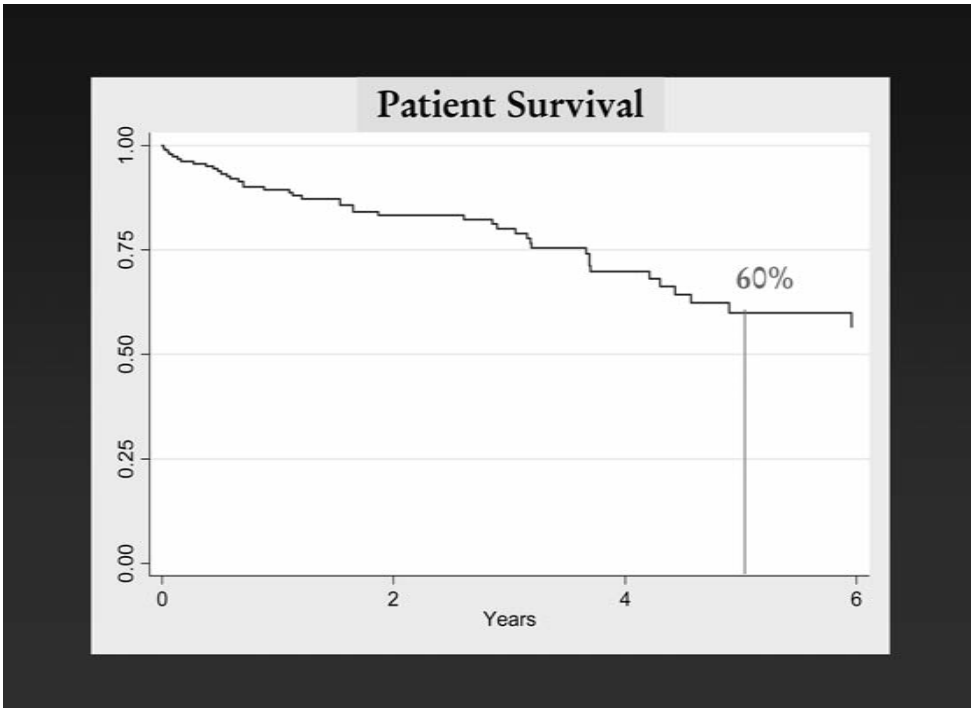

Figure 16-3. Five-year survival rates for all patients.

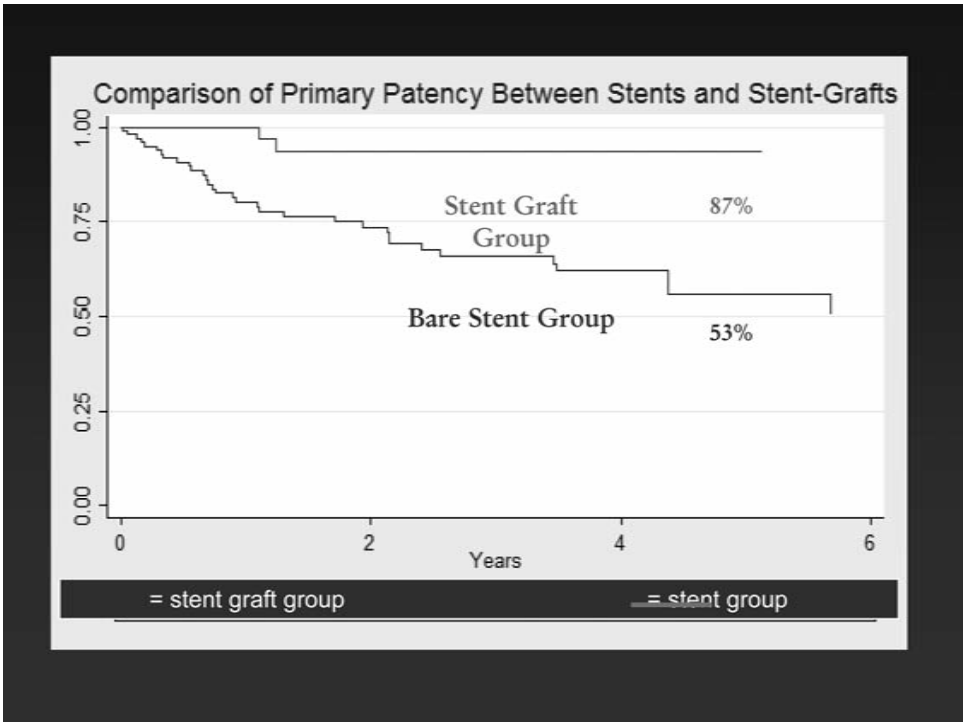

Figure 16-4. Patency for stent graft group compared with bare metal stent graft ($P<.01$).

occlusions occurred in the early perioperative setting, 2 of which were thrombectomized. The third perioperative occlusion patient underwent femorofemoral bypass. The fourth occlusion occurred within 30 days of the original procedure and was successfully thrombectomized with an open procedure.

DISCUSSION

In addition to our previous work,[12] several authors have published their techniques and early results with a hybrid approach to iliofemoral occlusive disease.[13,16-18] To the best of our knowledge, this report constitutes the largest series to date describing outcome and long-term patency of this procedure.

Endovascular treatment of iliac disease has radically changed management paradigms in vascular surgery over the last 2 decades. With improving technology and results, the preferred initial treatment of iliac occlusive disease is now endovascular. However, several factors such as the presence of external iliac occlusive disease, vessel calcification, and totally occluded iliac arteries have been associated with high complication rates and poor long-term durability when treatment is with bare metal stents.[19-20] Use of stent grafts in iliac occlusive disease has increased in popularity in recent years.[18,21] We have previously reported 12-month primary patency of 70% in patients treated with stent grafts for iliac occlusive disease (with and without femoral endarterectomy), which appeared to have superior patency compared with historical controls treated with bare metal stents.[22]

Despite the lack of objective evidence, these promising early results have led us to favor stent grafts over stents for the treatment of diffuse iliac occlusive disease, especially when performed in the setting of concomitant femoral endarterectomy when the larger sheath size required to place a stent graft is not a concern. The theoretical barrier to intimal hyperplasia and the ability to aggressively dilate calcified vessels are potential benefits for covered stents. Although the device sizes and postdeployment balloon diameters are not significantly different between the bare stents and the stent grafts and inflation pressures are not recorded, we believe that the lessened risk of iliac rupture leads to improved dilation with use of higher inflation pressures. These hypotheses are supported by the improved patency of stent grafts versus stents in the current study. Our preferred device is now a covered stent for this combined procedure; bare stents are usually only placed across a patent hypogastric artery. Iliac rupture, especially in small, calcified lesions, is a potentially morbid complication that can be minimized and treated by a stent graft. In our series, there were 5 iliac artery ruptures during device deployment. All were treated with a stent graft over the wire, and none required open repair.

There are several potential limitations with this technique. We do not routinely cover the hypogastric artery in treating common or external iliac lesions. The fate of a crossed hypogastric artery for treatment of adjacent disease is unknown and may eventually exacerbate or contribute to pelvic ischemia. In addition, the inability to cross long total occlusions is a potential obstacle for successful utilization of this technique. This has largely been overcome by increased use of re-entry devices.

The excellent primary-assisted patency rate reemphasizes the value of common femoral endarterectomy. This is supported by earlier work in which CFA endarterectomy with iliac stent grafting is associated with better patency rates when compared with cases undergoing iliac stent grafting alone. Nearly 50% of the failed patients in the latter group went on to have subsequent femoral endarterectomy, which might

argue for a more rigorous preoperative evaluation for common femoral disease.[22] Of the patients in this series requiring subsequent intervention, more than half were managed with an outpatient percutaneous procedure to maintain patency.

It is interesting to note that treatment of EIA lesions versus more extensive treatment of EIA and CIA lesions led to a higher risk of reintervention to maintain patency. This is an unexpected finding but may relate to undertreatment of more proximal disease. Other groups have shown no significant difference in primary patency stratified by previous TASC classification, which may imply the difficult and varying nature of iliac lesions in these patients.[23]

Aortobifemoral bypass grafting remains the standard of care for diffuse aortoiliac disease.[3] Our results with this hybrid procedure cannot be directly compared with results with bypass grafting or isolated iliac stenting because of patient selection, associated comorbidities, and the varying anatomic distribution of disease. However, this procedure has comparable outcomes and is well tolerated. In addition, many of these patients would not be candidates for abdominal revascularization procedures and would benefit from the shortened hospital stay and overall decreased trauma of a limited groin exposure. Although our primary patency rates are lower than expected, many of these patients enjoy continued patency with only percutaneous reintervention. In this regard, this technique compares favorably with patency rates of extra-anatomic bypasses.[24-25] In addition, male patients are not exposed to the risks of erectile dysfunction with open iliac or aortic surgery. Although the patients in this series underwent general anesthesia, this procedure can also be performed using spinal or epidural techniques, which might improve tailored patient care.

In conclusion, we have found that CFA endarterectomy associated with iliac stenting or stent grafting is a viable alternative to more invasive open procedures in terms of both perioperative complications and long-term patency. In addition, our data suggest that stent grafts placed in the iliac position have improved primary patency compared with bare metal stents and may be the preferred device for this patient population.

REFERENCES

1. Radoux JM, Maiza D, Coffin O. Long-term outcome of 121 iliofemoral endarterectomy procedures. Ann Vasc Surg 2001;15:163–70.
2. Melliere D, Blancas AE, Desgranges P, Becquemin JP. The underestimated advantages of iliofemoral endarterectomy. Ann Vasc Surg 2000;14:343–9.
3. Norgren L, Hiatt WR, Dormandy JA, et al. Inter-society consensus for the management of peripheral arterial disease. Int Angiol 2007;26:81–157.
4. Nevelsteen a, Wouters L, Suy R. Aortofemoral Dacron reconstruction for aorto-iliac occlusive disease: a 25 year study. Eur J Vasc Surg 1991;5:179–86.
5. Malone JM, Moore WS, Goldstone J. The natural history of bilateral aortofemoral bypass grafts for ischemia of the lower extremities. Arch Surg 1975;110:1300, 1301–6.
6. de Vries SO, Hunink MG. Results of aortic bifurcation grafts for aortoiliac occlusive disease: a meta-analysis. J Vasc Surg 1997;26:558–69.
7. Marin ML, Veith FJ, Sanchez LA, et al. Endovascular repair of aortoiliac occlusive disease. World J Surg 1996;20:679–86.
8. Martin EC, Katzen BT, Benenati JF, et al. Multicenter trial of the Wallstent in the iliac and femoral arteries. J Vasc Interv Radiol 1995;6:843–9.
9. Bosch JL, Hunink MG. Meta-analysis of the results of percutaneous transluminal angioplasty and stent placement for aortoiliac occlusive disease. Radiology 1997;204:87–96.

10. Cynamon J, Marin ML, Veith FJ, et al. Stent-graft repair of aorto-iliac occlusive disease coexisting with common femoral artery disease. J Vasc Interv Radiol 1997;8:19–26.
11. De Roeck A, Hendriks JM, Delrue F, et al. Long-term results of primary stenting for long and complex iliac artery occlusions. Acta Chir Belg 2006;106:187–92.
12. Nelson PR, Powell RJ, Schermerhorn ML, et al. Early results of external iliac artery stenting combined with common femoral artery endarterectomy. J Vasc Surg 2002;35:1107–13.
13. Dosluoglu HH, Cherr GS. Pre-arteriotomy guidewire access (PAGA): a crucial maneuver for securing inflow and/or outflow in patients with bulky iliofemoral occlusive disease undergoing combined (open/endovascular) procedures. Eur J Vasc Endovasc Surg 2006;32: 97–100.
14. Rutherford RB, Baker JD, Ernst C, et al. Recommended standards for reports dealing with lower extremity ischemia: revised version. J Vasc Surg 1997;26:517–38.
15. Pentecost MJ, Criqui MH, Dorros G, et al. Guidelines for peripheral percutaneous transluminal angioplasty of the abdominal aorta and lower extremity vessels. A statement for health professionals from a special writing group of the Councils on Cardiovascular Radiology, Arteriosclerosis, Cardio-thoracic and Vascular Surgery, Clinical Cardiology, and Epidemiology and Prevention, the American Heart Association. J Vasc Interv Radiol 2003; 14:S495–515.
16. Queral LA, Criado FJ, Patten P. Retrograde iliofemoral endarterectomy facilitated by balloon angioplasty. J Vasc Surg 1995;22:742–8; discussion 748–50.
17. Ali AT, Modrall JG, Lopez J, et al. Emerging role of endovascular grafts in complex aortoiliac occlusive disease. J Vasc Surg. 2003;38:486–91.
18. Nevelsteen A, Lacroix H, Stockx L, Wilms G. Stent grafts for iliofemoral occlusive disease. Cardiovasc Surg 1997;5:393–7.
19. Powell RJ, Fillinger M, Bettmann M, et al. The durability of endovascular treatment of multisegment iliac occlusive disease. J Vasc Surg 2000;31:1178–84.
20. Powell RJ, Fillinger M, Walsh DB, et al. Predicting outcome of angioplasty and selective stenting of multisegment iliac artery occlusive disease. J Vasc Surg 2000;32:564–9.
21. Murphy TP, Webb MS, Lambiase RE, et al. Percutaneous revascularization of complex iliac artery stenoses and occlusions with use of wallstents: three-year experience. J Vasc Interv Radiol 1996;7:21–7.
22. Rzucidlo EM, Powell RJ, Zwolak RM, et al. Early results of stent-grafting to treat diffuse aortoiliac occlusive disease. J Vasc Surg. 2003;37:1175–80.
23. Park KB, Do YS, Kim DI, et al. The TransAtlantic InterSociety consensus (TASC) classification system in iliac arterial stent placement: long-term patency and clinical limitations. J Vasc Interv Radiol 2007;18:193–201.
24. Hertzer NR, Bena JF, Karafa MT. A personal experience with direct reconstruction and extra-anatomic bypass for aortoiliofemoral occlusive disease. J Vasc Surg 2007;45:527–35.
25. Passman MA, Taylor LM, Moneta GL, et al. Comparison of axillofemoral and aortofemoral bypass for aortoiliac occlusive disease. J Vasc Surg 1996;23:263–9; discussion 269–71.

17

Long-Term Outcomes of Iliac Artery Angioplasty and Stenting

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If long-term, complication-free durability is the yardstick by which endovascular procedures are gauged, then iliac angioplasty (PTA) is certainly to be reckoned a success; results of endoluminal treatment have become even more impressive since the introduction of stents. The promise of durable effectiveness, suggested by early reports, has been reconfirmed for 25 years by publications world wide. There is little question iliac PTA/stenting is the established "ne plus ultra" of non-coronary *interventional* therapy. But not all procedures are consistently successful; and it must be borne in mind that the results of iliac stenting must be weighed against aortofemoral bypass graft, perhaps the most successful of all *open* interventions.

Which lesions can be expected to be treatable and achieve early and late patency? What roles do stenting and re-intervention play in enhancing the results of this endoluminal procedure? The purposes of this paper are first, to review the long-term results of iliac PTA/stenting, surveying patency and complications as well; second, to evaluate the predictors of success and failure; and finally, to see whether the available evidence will provide a guide to improved patient selection and consequently enhance the outcomes of endoluminal revascularization. Isolated internal iliac artery (IIA) PTA/stent for vasculogenic impotence and discreet hip claudication are omitted because of its relative rarity and a lack of systematic studies.

TECHNIQUE

Balloon angioplasty techniques have become standardized over over the last decade. Device-related improvements, however, are ongoing. A partial list includes:

- New sheaths with thinner walls which allow smaller diameter balloon catheters and stents to be delivered
- Pre-shaped and shapable catheters and sheaths
- Improved and hydrophilically-coated guidewires in multiple sizes

- Lower profile, coated balloon catheters with increased trackability on smaller shafts
- More flexible balloon expandable (Palmaz) stents
- Self-expanding Nitinol stents with reduced foreshortening to improve precision of deployment
- Cost-reductions for many endoluminal devices

Bilateral retrograde femoral puncture and access facilitates iliac angioplasty. Access of both sides allows aortic and femoral pressures to be evaluated simultaneously or for the use of kissing balloons or stents. Bilateral access is appropriate even in cases of distal external iliac/proximal common femoral artery disease that must be treated from the contralateral artery (over the aortic bifurcation). Because of limited ipsilateral working room, the juxtafemoral lesion must be treated from the contralateral side; nevertheless, bilateral access simplifies hemodynamic assessment. The more severe the iliac lesions and, in consequence, the greater the reduction of the femoral pulse, the more problematic retrograde femoral puncture becomes. A host of easily learned techniques enable the interventionist to puncture the "pulseless" artery so that it becomes unnecessary to approach the ipsilateral lesion via contralateral femoral access across the aortic bifurcation.[1] Complete iliac artery *occlusions*, of course, can be treated "over-the-top," but these lesions are crossed much more easily from the ipsilateral side. *Stenoses*, as would be expected, can be successfully crossed from either direction with appropriate guidewires and catheters.

Our stent of choice for common iliac artery and proximal and mid-external iliac artery lesions is a second or third generation balloon expandable stent. Nitinol stents are also very useful and their applicability has been augmented by the addition of radiopaque end-markers which improve fluoroscopic visualization. We recommend pre- and post-procedural platelet inhibiting drugs and administer 40–50 units per kgm of heparin prior to angioplasty or stent deployment (the heparin is not reversed with protamine). Antibiotics are administered routinely prior to deployment and in the presence of distal ischemic or infected lesions. For large vessel angioplasty IIB/IIIA glycoprotein platelet inhibitors have no proven benefit. Sheaths are removed in the Recovery Room after 1 to 2 hours when the ACT has normalized. This approach has made the use of percutaneous closure devices unnecessary.

RESULTS

The results of balloon angioplasty of iliac stenoses have been satisfactory and durable based on objective criteria such as ankle/brachial pressure indices (ABPI) and duplex assessment. Technical success rates, based on intention to treat, exceed 95% and yield 1–3 year primary patency of approximately 80% and 65% respectively (Table 17–1).[2-6] If in the follow up of primary angioplasties, failure occurs, aggressive reintervention of the *initial lesions*, (often supplemented by stenting), will improve the secondary 3 year patency to about 75–80% (Table 17–1).[2-6] Delayed hemodynamic failure of the angioplastied limb may be the result of a new stenosis remote from the initial lesion. The frequency with which a new lesion, rather than the initial lesion, later compromises limb blood flow is not well documented, but in our experience exceeds 20% after 3 months.

With the availability of stents after 1989, many investigators recommended primary stenting on either a selective or routine basis: both strategies increase 1 and

TABLE 17-1. PATENCY RATES FOR PTA OF ILIAC ARTERY STENOSIS AND OCCLUSIONS

	Primary Patency (%)			Secondary Patency (%)		
	1y	3y	5y	1y	2y	3y
Balloon angioplasty of iliac stenosis[2-6]	78	66	61	92	87	77
Balloon angioplasty of iliac occlusions[2,6,15,18,19]	68	60	*	86	82	

*data not available
weighted averages

3 year primary patencies (Table 17–2).[7-14] At present, there are no data to support the practice of routine primary stenting for iliac artery lesions.[15] Their usefulness to control residual stenosis/recoil and dissection are, however, clearly documented.

Patency rates for PTA of iliac *occlusions* is, as expected, less than for *stenosis*, in large measure because of initial technical failure (failure to cross) of about 20% (Table 17–1).[7,16,17] Technical success and 1 and 3 year primary patency are both improved with stenting. Routine stenting appears to have become accepted practice for the treatment of chronic, complete occlusions and 1 and 3 year patencies of approximately 75% and 65% respectively can be expected with appropriate patient selection (Table 17–2).[15,18,19] Stenting of recurrent iliac artery lesions (secondary patency) will enhance patency by 5–10% at both time periods. Subintimal angioplasty, developed and championed by Bolia and Bell, has clearly carved a niche for itself for infrainguinal lesions.[20] It is too early to tell whether or not this technique will be readily applicable to iliac angioplasty. The accumulated data indicate that by combining PTA and stenting and repeat therapy when clinically indicated, very satisfactory patency of iliac artery lesions, either *stenoses* and *occlusions*, is attainable.

PREDICTORS OF SUCCESS

PTA/stenting of the iliac arteries is not uniformly successful. Not only are the results of angioplasty and stenting superior in the iliac segment than in the SFA, but other determinates of improved outcomes are well documented. These include:

- Proximal lesions[6]
  CIA > EIA

TABLE 17-2. PATENCY RATES FOR STENTING OF ILIAC ARTERY STENOSES AND OCCLUSIONS

	Primary Patency (%)		
	1y	3y	5y
Stenting of iliac stenosis[9-16]	90	74	72
Stenting of iliac occlusions[7,17,18]	72	64	*

*data not available
weighted averages

- Stenoses > occlusions[6]
- Short (<3 cm) vs long lesions[6]
- Clinical indication for intervention[6]
  Claudication > limb salvage
- Runoff [6]
  Patent vs occluded SFA (although poor runoff, e.g. SFA occlusion, is a predictor of reduced patency, there are no studies to compare patients who only undergo iliac PTA/stenting with those whose SFA occlusions are by-passed or otherwise treated).
- Gender
  Men > women

In addition to these established predictors of success, others have been addressed:

- *Calcifications:* Although the presence of focal calcium flecks in the occlusive lesion probably do not adversely affect PTA stenting, arteries with circumferential, dense calcification are poor choices for endoluminal therapy.
- *Hormone replacement therapy (HRT):*[21] Women tend to have worse outcomes than men and HRT may actually worsen women's outcomes.
- *Cigarette smoking:* As expected, long-term outcomes are worse in patients who continue to smoke.
- *Hypertension and dyslipidemia:* Based on cardiac trials, failure to control these arteriosclerotic risk factors is associated with worse outcomes. The same may be true for homocysteinemia.
- *Diabetes mellitus:*[22] Some studies suggest no deleterious effects of diabetes mellitus (DM) on outcomes. DM, however, is closely associated with infrapopliteal disease rather than with aortoiliofemoral occlusive lesions.
- *Arterial tortuosity:* No studies assess the effect of arterial tortuosity on patency.
- *Internal artery occlusive disease (IIA):* Stenosis or occlusion of the internal iliac arteries is a frequent congener of CIA + EIA lesions. There is no evidence of it being a predictor of the success or failure of iliac PTA/stenting. IIA involvement may be a marker for more extensive disease whose endoluminal treatment outcomes tend to be worse.
- *Type of stent:* Properly sized, both self-expanding and balloon expandable stents are equally effective.
- *IVUS assessed stent deployment:* IVUS has been used to determine stent-artery wall coaption and incomplete stent deployment with a residual "step-off." Hence, some workers have recommended assessment of the adequacy of stent sizing with IVUS. This modality, of course, would only be useful for balloon expandable stents. Neither cost-effectiveness nor improved patency using IVUS have been validated by appropriate randomized trials.
- *Length of the stented segment*:[23] Some studies have suggested that angioplastied arteries requiring more than 2 stents (>7 cm) have a poorer prognosis. This may be the result of a) stents crossing and possibly occluding the IIA or b) longer lesions extending to the EIA with its known poorer prognosis. Whether it is necessary to cover the entire length of artery subjected to PTA with a stent(s) remains a subject of conjecture (Figure 17–1, 17–2, 17–3).

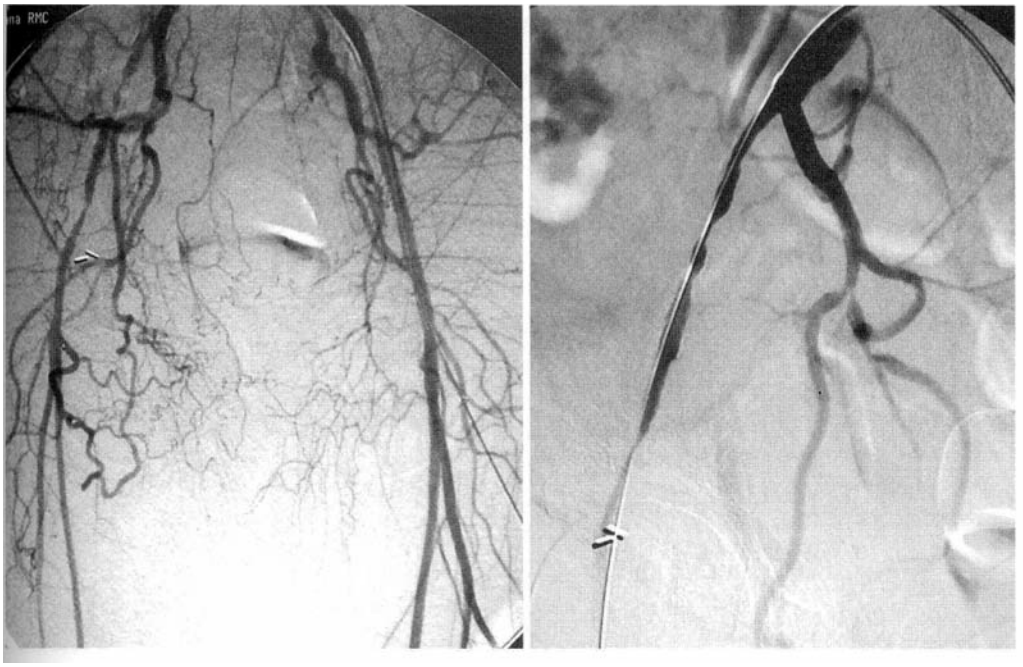

Figure 17-1. Angiogram of iliac arteries showing multisegment diffuse stenosis of right iliac artery.

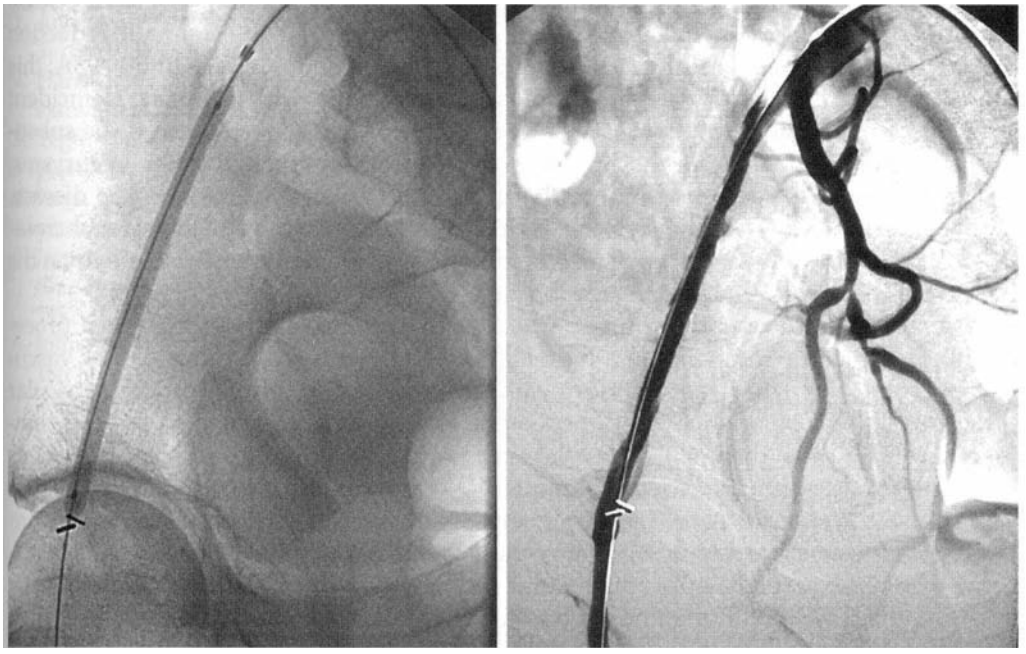

Figure 17-2. Balloon angioplasty of the right iliac artery lesion with a 6 mm × 10 cm balloon showing some improvement in the stenotic segments.

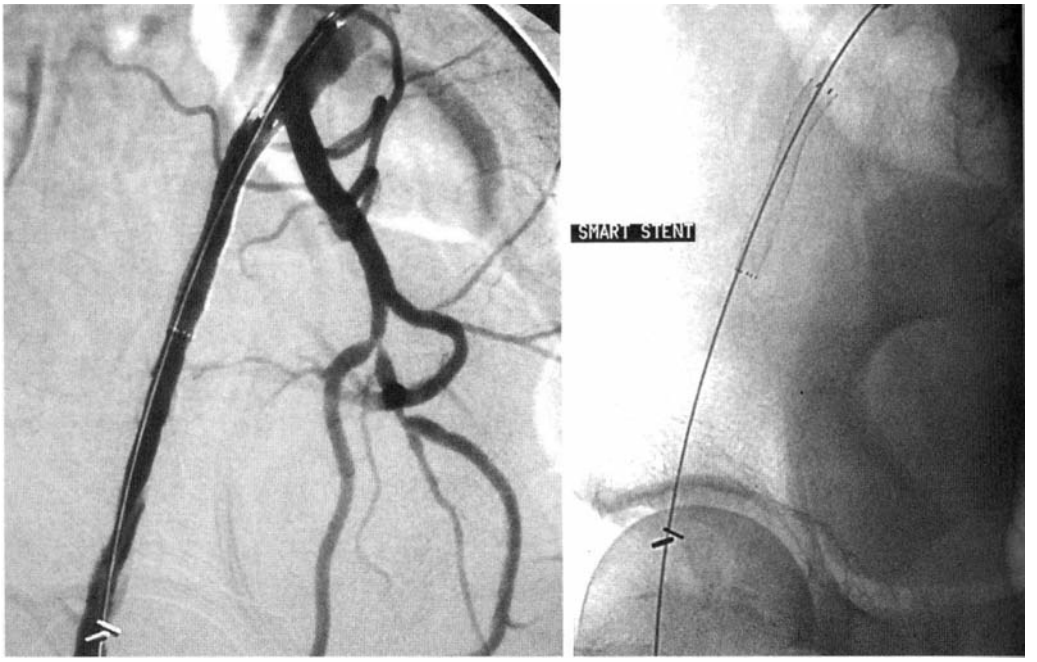

Figure 17-3. Post-stenting angiogram (smart stent 8 mm × 40 mm). Showing improved proximal stenotic segment.

DISCUSSION

The data that support the effectiveness of treating aortoiliofemoral occlusive disease with PTA/stenting are ineluctable. Underlining this success is the dramatic reduction of aortofemoral bypass grafting (ABFB) in Europe and in some centers in the USA; this both a relative and absolute decline in revascularization by open surgery. Coincident with the transformation of the treatment paradigm for occlusive disease is the substitution of endoluminal stent grafting for open repair of abdominal aortic aneurysms. Because endoluminal therapies are most effective in patients with less severe disease, those whose only therapeutic recourse is open surgery often require suprarenal cross-clamping and other technical complexities. Stratifying outcomes based solely upon the method of therapy is, therefore, inappropriate.

Moreover, until recently, therapy was further stratified with endoluminal procedures performed by interventional radiologists and open revascularizations by vascular surgeons. Now that vascular surgical fellows are being trained in endovascular techniques, their experience with open therapies can be anticipated to decline commensurately.[24] Who, then, will be left to revascularize the difficult and complex lesions not amenable to catheter-based techniques? No answer to this question is possible without comprehensive epidemiologic studies of aortoiliac disease that is left untreated and lesions treated by endoluminal or open techniques.[25] Until such data become available, it seems appropriate that therapeutic decisions should be made by doctors skilled with both methods and a vested interest in neither. It has been shown in other conditions that when the decisions are made by specialist physicians, shorter hospital lengths of stay can be achieved.[26] This sentinel outcome will doubtless gather the attention of governmental agencies and third party payers.

Because iliac PTA/stenting is applicable to less severe lesions and ABFB to the more advanced, these procedures are obviously complimentary. Viewed from the perspective of durable effectiveness, ABFB can substitute, as it did successfully for 25 years, for iliac PTA/stenting; the converse, however, is not true. By treating proximal lesions with ABFB, in patients with tandem lesions in the aortoiliac and infrainguinal segments, it has been shown that symptoms can be completely relieved in 50–75% of cases.[27] With iliac PTA stenting, essentially all patients will remain symptomatic from the more distal obstruction in the SFA. A cohort of such patients with either therapy for tandem lesions has not yet been systematically compared using objective measurements of hemodynamic and clinical outcomes. Comparison of the financial costs of ABFB and iliac PTA/stenting as primary procedures has demonstrated that there are no fiscal advantages to the less invasive approach to therapy.[28] Thus, the belief that endoluminal techniques will soon replace ABFB[29] has been likened to Mark Twain's comment on his purported imminent demise: "Report of my death greatly exaggerated."[30]

Based on the experience of the last decade, further technical advances can be anticipated. Covered stents have been recommended for ulcerated and emboligenic plaques. Combination procedures may also exploit some of the useful properties of covered stents by lining external iliac lesions that have been dilated or endarterectomized by retrograde methods from the femoral artery. Analogous to open surgical endarterectomy, simple balloon angioplasty of an extensively diseased EIA-femoral segment is quite vulnerable to restenosis. Improvements in long-term benefits in this arterial segment can only increase the applicability of endoluminal therapies.

REFERENCES

1. Andros G. Arterial Access in Endovascular Surgery. In Moore and Ahn (ed). *Endovascular Surgery, 3rd E.* Philadelphia:W.B. Saunders;2001:37–47.
2. Harris RW, Dulawa LB, Andros G, et al. Percutaneous transluminal angioplasty of lower extremities by the vascular surgeon. *Ann Vasc Surg.* 1991;5:345–353.
3. Tegtmeyer CJ, Hartwell GD, Selby JB, Robertson R, Kron IL, Tribble CG. Results and angioplasty in aortoiliac disease. *Circulation*. 1991;83(suppl I): I–53–I–60.
4. Jeans WD, Armstrong S, Cole SEA, et al. Fate of patients undergoing transluminal angioplasty for lower limb ischemia. *Radiology.* 1990;177:559–564.
5. Jorgensen B, Skovgaard N, Norgard J, et al. Percutaneous transluminal angioplasty in 226 iliac artery stenoses: role of the superficial femoral artery for clinical success. *Vasa.* 1992;21:382–386.
6. Johnston KW: Iliac arteries: reanalysis of results of balloon angioplasty. *Radiology.* 1993: 186: 207–212.
7. Gupta AK, Ravimandalam K, Rao VR, et al. Total occlusion of iliac arteries: results of balloon angioplasty. Cardiovasc *Intervent Radiol*. 1993;16:165–177.
8. Bosch JL, Hunink MGM. Metaanalysis of the results of percutaneous transluminal angioplasty and stent placement of aortoiliac occlusive disease. *Radiology.* 1997;204:87–96.
9. Murphy TP, Webb MS, Lambiase RE, et al. Percutaneous revascularization of complex iliac artery stenoses and occlusions with use of wallstent: Three-year experience. *J Vasc Intervent Radiol.* 1996;7:21–27.
10. Laborde JC, Palmaz JC, Rivera FJ, et al. Influence of anatomic distribution of atherosclerosis on the outcome of revascularization withiliac stent placement. *J Vasc Intervent Radiol.* 1995;6: 513–521.
11. Brewster DC. Clinical and anatomical considerations for surgery in aortoiliac disease and results of surgical treatment. *Circulation.* 1991;83(suppl I):142–152.

12. Tetteroo E, van der Graaf Y, Bosch JL, et al. Randomised comparison of primary stent placement versus primary angioplasty followed by selective stent placement in patients with iliac-artery occlusive disease. Dutch Iliac Stent Trial Study Group. *Lancet.* 1998 Apr 18;351(9110): 1153–1159.
13. Palmaz JC, Laborde JC, Rivera FJ, et al. Stenting of iliac arteries with Palmaz stent: experience from a multicentric trial. *Cardiovasc Intervent Radiol.* 1992;15:291–297.
14. Strecker EP, Hagen P, Liermann D, et al. Iliac and femoropopliteal vascular occlusive disease treated with flexible tantalum stents. *Cardiovasc Interven Radiol.* 1993:16:158–164.
15. Cambria RA, Farooq MM, Mewissen MW, et al. Endovascular Therapy of iliac arteries: routine application of intraluminal stents does not improve clinical patency. *Ann Vasc Surg*. 1999;13:599–605.
16. Blum U, Gabelmann A, Redecker M, et al. Percutaneous recanalization of iliac artery occlusions: results of a prospective study. *Radiology.* 1993;189:536–540.
17. Rutherford RB, Durham J. Percutaneous balloon angioplasty for arteriosclerosis obliterans: long term results: In Yao JST, Pearce WH, ed. *Techniques in Vascular Surgery.* Philadelphia:W.B. Saunders, 1992:329–345.
18. Wolf YG, Schatz RA, Knowels HJ, et al. Initial experience with the Palmaz stent for aortoiliac stenoses. *Ann Vasc Surg.* 1993;7:254–261.
19. Hausegger KA, Lammer J, Klein GE, et al. Percutaneous recanalization of pelvic artery occlusions: fibrinolysis, PTA, stents. *ROFO*. 1991;155:550–555.
20. London NJ, Srinivasan R, Naylor AR, et al. Subintimal angioplasty of femoropopliteal artery occlusions: the long-term results. *Eur J Vasc Surg*. 1994:8(2):148–55.
21. Timaran CH, Stevens SL, Grandas OH, et al. Influence of hormone replacement therapy on the outcome of iliac angioplasty and stenting. *J Vasc Surg*. 2001;33:S85–S92.
22. Spence LD, Hartnell GG, Reinking G, et al. Diabetic versus nondiabetic limb-thretening ischemia: outcome of percutaneous iliac intervention. *AJR.* 1999;172:1335–1341.
23. Powel RJ, Fillinger M, Bettmann M, et al. The durability of endovascular treatment of multisegment iliac occlusive disease. *J Vasc Surg.* 2000;31:1178–1184.
24. Moore WS, Clagett GP, Veith FJ, et al. Guidelines for hospital privileges in vascular surgery: An update by an ad hoc committee of the American Association for Vascular Surgery and the Society for Vascular Surgery. In press *(J Vasc Surg).*
25. Management of Peripheral Arterial Disease (PAD): TransAtlantic Inter-Scociety Concensus (TASC). *J Vasc Surg*. 2000;31:S97–S112.
26. Clinical advisory board-partnering in throughput reform. August 2002.
27. Brewster DC. A surgeon's view of iliac stenting: what will be the role for aortofemoral bypass? *Prespect Vasc Surg.* 1996;9(1):67–70.
28. Ballard JL, Bergen JJ, Singh P, et al. Aortoiliac stent deployment versus surgical reconstruction: analysis of outcome and cost. *J Vasc Surg.* 1998;29;94:103.
29. Criado FJ, Wellons E, Abul-khoudoud O, et al. Endovascular intervention for iliac artery disease: indications and techniques. *Prespect Vasc Surg.* 1999;11(1):29–46.
30. Andros G. Expert commentary to "Endovascular intervention for iliac artery disease: indications and techniques." *Prespect Vasc Surg.* 1999;11(1):47–49.

18

The Cost of Patency

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INTRODUCTION

The looming crisis in health care funding stands as one of the biggest challenges facing our economy. Currently, 500 billion U.S. dollars are earmarked for Medicare and Medicaid payments this year, and the federal government predicts a 31% increase in health care spending over the next 5 years. If costs continue to grow as projected, the U.S. federal government will spend approximately 20% of its gross domestic product on health care by 2050.[1] It is thought that Medicare will no longer be solvent by 2017, and the current recession threatens to worsen our predicament. If these predictions are brought to fruition, we will be forced to ration health care or face drastic cuts in government programs and incur higher taxes.

The diagnosis, treatment, and long-term care of patients with PAD (PAD) imposes a significant burden on our health care system. PAD affects 8 to 12 million Americans and costs 151 billion dollars in direct and indirect costs per year to treat. These costs are likely to increase as the baby boomers age and hospitals and doctors realize a population of patients as a new source of revenue.

The specialty of vascular surgery has expanded with the advent of endovascular surgery, introducing an ever-increasing array of new and potentially expensive modalities to treat our patients. There has been a dramatic shift toward the use of endovascular therapy in peripheral vascular disease. There has been a 40% increase in the total number of endovascular cases in the past 7 years, with a fall in the number of open bypasses by 30%.[2] Unfortunately, many of these new treatment modalities have not been fully evaluated for either clinical efficacy or cost-effectiveness. Contemporary discussions of cost-to-benefit ratios have been hindered by a poor appraisal of the true cost involved in treatment of PAD. Often, discussions have taken into account only initial cost, involving the costs of the device and hospital stay. This is flawed and myopic, because it does not account for procedural durability, cost of reintervention, and patient longevity. Our ability as vascular surgeons to control costs while providing quality care is tantamount to our success in the future. The current administration is looking to major medical providers such as hospitals, doctors, insurance agencies, and pharmaceutical companies to cut 2 trillion dollars from health care spending over the next 10 years. We as a society of vascular surgeons must prove a significant

cost-to-benefit ratio for our interventions. If not, our reimbursement and image as medical providers will be affected.

THE COMPLEXITIES OF ACCURATE COST ANALYSIS

How well do we spend our health care dollars? What is the best way to allocate health care resources? These two questions are at the root of all conversations about health care analysis and reform. Whereas these are two relatively straightforward questions, producing accurate answers is extremely difficult. At the root of the problem is defining what an acceptable cost is for a defined outcome. Determining whether a cost is justifiable is dependent on the resources available and society's perception of the value of the outcome. Owing to the complexity and philosophical nature of these questions, many cost models exist, but few of them are universally accepted or used to guide decision-making.

The most rudimentary method of cost analysis is the study of the procedural costs associated with the index procedure. Although this view may be adequate in some specialties, vascular surgery is somewhat unique in that repeated interventions are often required to maintain the desired effect. This means that the initial cost may be only a fraction of the cost over the lifetime of a procedure's being evaluated. In addition, the cost of any complication adds to the initial expense of each procedure. There are a number of other resources that must be added to taking care of vascular patients, including nursing home care, home health care, and costs of following up. It is easy to see why a direct comparison of initial cost is a poor marker of overall cost to society. Even if all the aforementioned costs were measured and then directly compared, the model would still be inadequate because it has no measure of outcomes associated with it.

Comparison of cost using quality adjusted life year (QALY) as an outcome is another commonly used method of cost analysis. QALY takes into account both quality and quantity of life generated by health care interventions. A QALY places weight on time in different health states. A year in perfect health is worth 1. A year in less than perfect health is worth less than 1, and death is considered 0. QALYs are combined with an intervention's cost to determine a cost-utility ratio. The cost-utility ratio allows for costs of interventions to be directly compared. The meaning and usefulness of QALYs are debatable. Perfect health is hard to define. It is arguable that there are states of health that are worse than death and therefore they should have a negative value. There is also a problem of accurately placing a numerical value on a subjective measure such as quality of life. Arguably, many models place too much weight on physical pain and disability over mental health. Discrimination against the elderly, chronically ill, or disabled patients is also a commonly cited flaw of QALY. QUALY-based cost analysis is an obvious improvement over a comparison of index procedure costs. Unfortunately, the many assumptions and complexity of calculating QALY affect its accuracy and reproducibility.

The Markov Decision Analysis is another method used to scrutinize expenditure. This model compares the costs of different interventions using hypothetical groups of patients. The analysis uses patency rates gleaned from previously published data and then combined with cost data to develop a marker of cost-effectiveness. The analysis often uses an arbitrary number that represents an acceptable societal cost for the desired outcome. Unfortunately, data in the literature are heterogeneous, involving different patient subsets with different comorbidities and disease severity. These same problems are seen with meta-analysis and negatively impact the validity of conclusions derived from this data. The calculations of decision analytical models are com-

plex, require multiple assumptions, and are not easy to reproduce without expertise in the area. All of these factors make decision analysis prone to error. There are significant variations in outcomes from individual practitioners when compared with the published literature. This makes it difficult for practitioners to base their decision-making on these models. Decision analytical models offer an improvement on more rudimentary methods of cost analysis but still have many flaws.

Cost-Per-Day of Patency

In an attempt to overcome some of the failings of the aforementioned cost models, an amortized cost model was developed to examine the cost efficacy characteristics of a given revascularization procedure. Our goal was to create a model that was easy to use and understand with data that were readily available to all practicing vascular surgeons. We used patency as the outcome measure in our model for the following reasons:

- Patency is the gold standard by which outcomes are reported in peripheral vascular disease.
- Patency can be universally applied to all patient subsets.
- Patency data can be easily obtained by all practicing vascular surgeons.
- There are clear reporting standards.
- Patency is not a subjective measure.

To determine cost at any time, we calculated the summation of all costs accrued over time to maintain patency. This takes into account the initial cost and the cost of all subsequent interventions. This is then divided by time in days to give the cost per day of patency. When an intervention failed patency, the cost per day of patency remained static throughout the remaining time period of the study.

Cost per day of patency is expressed at any given time interval (t) as follows:

Where pa = maximum number of days of assisted patency, $Cost(i)$ = total hospital costs (direct and indirect) at time interval i days from index procedure ($t=0$). With this cost model, the cost per day of patency was assessed at any given time interval from the index procedure. Hypothetical situations are illustrated in the cost-efficacy curves of Figure 18–1. The figure depicts a series of patients undergoing revascularization. Patient A undergoes a successful revascularization, without the need for subsequent reintervention throughout the follow-up time period. Patient B requires a secondary procedure to maintain patency (primary, assisted, or secondary), and subsequently maintains patency. Finally, Patient C fails patency and an assistive procedure is not undertaken. Of note, total hospital costs were accounted for at each time point. If a patient suffered a complication or adverse event from a given procedure, the financial burden of that event was included in the model (costs and indirect costs associated with treatment of that complication). Cross-over to the other mode of therapy was considered a failure to maintain patency.

THE EAST CAROLINA HEART INSTITUTE EXPERIENCE

From July 2003 to July 2006, all patients who had undergone open or endovascular treatments of femoropopliteal arterial circulation were identified in a retrospective computerized

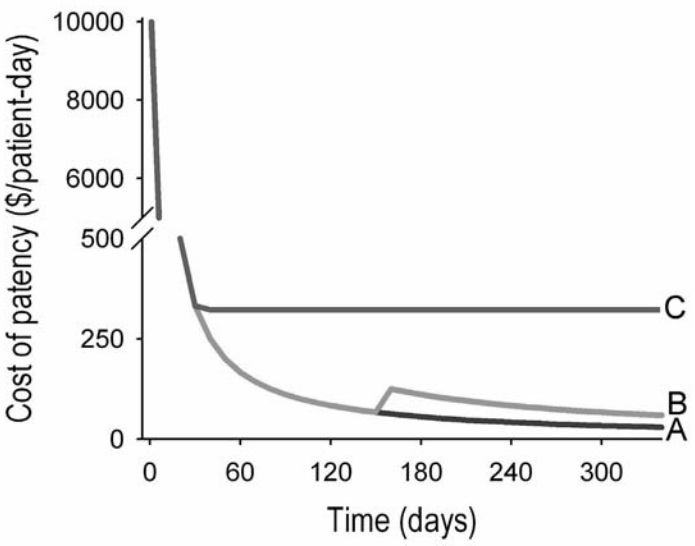

Figure 18-1. Hypothetical cost efficacy curves for the following revascularization scenarios: A – Successful initial revascularization without failure of patency or reintervention, B – Reintervention to maintain patency, C – Failure of patency without reintervention.

database. The decision to use open bypass or endovascular revascularization was based on clinical evaluation, anatomic factors, and the attending surgeon's preference.

For the purpose of this study, total costs (direct and indirect cost) for the particular encounter were used to calculate the amortized cost. Costs associated with adverse outcomes after a particular revascularization were also included for the total encounter cost calculation. Cost data were available for all patient encounters in this study. If the patient underwent a major amputation ipsilateral to the index revascularization, this was included in the cost analysis. Costs of postoperative rehabilitation, nursing home care, or lost days of work were not included in this analysis.

The primary efficacy endpoint of this study was cost per patient/day of patency 12 months following the index procedure. Primary and primary-assisted patency were the secondary endpoints in this study. Patency was determined by guidelines of the Society for Vascular Surgery.

Results

Over a 3-year period, a total of 381 femoropopliteal segments were treated in 359 patients. There were a total of 183 femoropopliteal segments treated with open revascularization and 198 treated with endovascular therapy. Both treatment groups were well matched, with no significant difference in clinical variables, including Rutherford category, age, gender, diabetes, hypertension, end-stage renal disease (ESRD) requiring dialysis, tobacco, run-off, and postoperative pharmacotherapy.

In the open group, 165 of 183 bypasses (90%) were above the knee grafts using a prosthetic conduit. Of the 198 endovascular cases, 75 (38%) were TransAtlantic Inter-Society Consensus (TASC) II classification C or D cases. Subintimal angioplasty was routinely utilized in the treatment of T ASC D cases. A luminal re-entry device was used in only 5 cases (7% of TASC C or D cases). Stents were used in 42 (21%) cases owing to a suboptimal result with standard angioplasty (>30% residual stenosis or flow-limiting dissection); and atherectomy (mechanical or laser) was used in a total of 26 (13%) cases.

Durability data demonstrated that primary-assisted patency (all indications) at 12 months was 77±0.03% for the open group and 65±0.04% for the endovascular group (P<.01). Primary assisted patency for patients with claudication at 12 months was 93±0.03% in the open group versus 80±0.04% in the endovascular group (P<.01). Primary assisted patency for patients with critical limb ischemia at 12 months was 66±0.05% in the open group and 54±0.05% in the endovascular group (P<.01).

Initial cost of open therapy was significantly higher in all subgroups. Using the model of amortized cost described above, the cost per day of patency was calculated at 1 year. A graphical representation of the cost model is shown in Figures 18–2 and 18–3. Despite the difference in initial cost, our model showed no statistically significant difference in amortized cost at 1 year between open and endovascular groups regardless of indication. For all indications, the amortized cost per day of patency at 12 months was $229±106 for claudicants and $185±124 for endovascular cases (P=.71). Claudicants treated by simple angioplasty showed the lowest cost per day of patency ($26±14), although this did not reach statistical significance when compared with open therapy.

The driving forces of this cost model are the initial procedural cost, the costs of all assistive procedures, and the durability of these procedures (Table 18–1). Failures, especially those that were not subjected to reintervention, are a very significant part of this construct. A high rate of failures and lack of reintervention is especially evident in both the open and the endovascular groups with critical limb ischemia. Failure without re-intervention resulted in a significantly higher amortized cost in the endovascular group ($551±184 (vs.) $34±4, P<.01) and a trend toward a higher cost in the open group ($418±154 (vs.) $157±122, P =.23).

Utilizing the model, subgroup analysis was undertaken to identify risk factors in which one therapy was more cost-effective. Patients with critical limb ischemia, end-stage renal disease requiring dialysis, renal insufficiency (creatinine >1.5 mg/dL), and

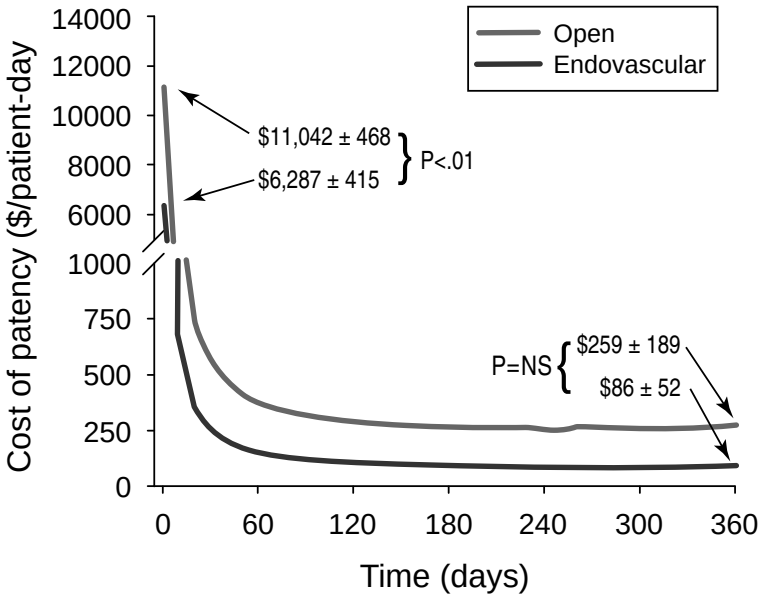

Figure 18-2. Cost efficacy curves in patients undergoing revascularization via both open and endovascular techniques for patients with claudication.

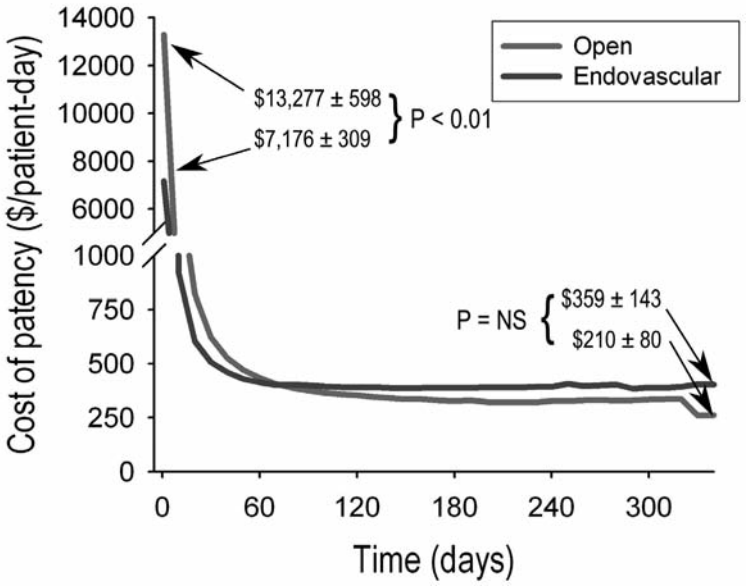

Figure 18-3. Cost efficacy curves in patients undergoing revascularization via both open and endovascular techniques for patients with critical limb ischemia.

congestive heart failure showed a trend toward open revascularization being more cost-effective; however, this did not reach statistical significance. Because early failures have a significant impact on this model, a multivariate analysis was undertaken to identify covariates associated with failure (Table 18–2). ESRD requiring dialysis was an independent risk factor for early failure in patients with critical limb ischemia for both open and endovascular therapy (OR= 3.48, P = .048). No other variable was a significant correlate of early failure.

DISCUSSION

Our experience at European Community Health Indicatiors (ECHI) provides a well-matched group of patients treated for femoropopliteal occlusive disease. Although this study is limited by its retrospective nature, moderate sample size, and the inherent treatment biases that exist in any practice, it follows the literature in that, with respect to complex femoropopliteal disease, the patency of open revascularization is superior to that of endovascular therapy.

Our model of cost per patient-day of patency gives a surrogate measure of a cost-benefit ratio by adding in often unconsidered factors such as overall patency and reintervention rates. This cost-efficacy model becomes static during the follow-up period once a particular intervention loses assisted patency. Through this mechanism, revascularization failures, especially early ones, weigh heavily. The inclusion of total hospital costs associated with the procedure, including those associated with adverse events, accounts for the economic impact of procedural-associated morbidity.

Cross-over to another mode of treatment was considered a failure, and the revascularization cost was held static at that time point. Therefore, documented patency and procedural durability become the main driving forces for economic success. The

TABLE 18-1. NUMBER OF ASSISTIVE PROCEDURES (PROC.), TOTAL COST OF ASSISTIVE PROCEDURES, AND NUMBER OF FAILED REVASCULARIZATIONS NOT UNDERGOING REINTERVENTION LISTED BY NUMBER OF MONTHS FROM INDEX PROCEDURE, STRATIFIED BY INDICATION (CLAUDICANT OR CLI) AND MODALITY (OPEN OR ENDOVASCULAR)

	Claudicants – Open (n=81)			Claudicants – Endovascular (n=112)			CLI – Open (n=102)			CLI – Endovascular (n=86)		
Time (months)	Proc. (n)	Cost (k$)	Failed (n)	Proc. (n)	Cost (k$)	Failed (n)	Proc. (n)	Cost (k$)	Failed (n)	Proc. (n)	Cost (k$)	Failed (n)
1	1	16.6	1	1	27.9	4	3	35.8	10	0	0	18
2	0	0	0	0	0	3	0	0	4	0	0	9
3	0	0	1	1	6.8	3	0	0	2	0	0	3
4	0	0	2	0	0	1	2	24.3	4	1	13.1	2
5	1	16.4	0	1	4.9	0	0	0	5	1	13.6	1
6	0	0	0	1	7.4	1	2	20.5	1	1	8.9	1
7	0	0	0	1	19.5	1	1	6.7	0	1	7.2	1
8	1	5.1	0	1	4.7	0	1	7.6	2	0	0	1
9	2	26.1	2	1	7.2	1	1	14.2	1	1	7.7	0
10	0	0	1	0	0	2	0	0	0	0	0	1
11	0	0	0	0	0	0	0	0	0	0	0	0
12	0	0	0	0	0	0	0	0	1	0	0	0

TABLE 18-2. MULTIVARIATE ANALYSIS FOR VARIABLES ASSOCIATED WITH EARLY FAILURE (<30 DAYS).

	Odds Ratio	95% Confidence Limits	P
Patient factors			
Age >80 years	1.738	0.411 – 7.359	.667
Diabetes	1.390	0.402 – 4.809	.453
Hypertension	0.455	0.058 – 3.553	.452
Hyperlipidemia	0.375	0.051 – 2.732	.332
ESRD	3.848	1.012 – 14.633	.040
CHF	2.842	0.861 – 9.380	.086
Pharmacology			
Statin	1.575	0.181 – 13.733	.681
Coumadin	1.261	0.275 – 5.771	.765
Anatomic			
Runoff <2 vessels	0.560	0.411 – 7.359	.355

ESRD = end stage renal disease, CHF = congestive heart failure.

societal definition of primary-assisted patency was utilized as our efficacy endpoint so that these data would be comparable with those of other contemporary series in the vascular surgery literature.

Interestingly, despite a nearly twofold difference in initial cost, the cost savings of endovascular therapy is not carried out over time. The loss of the cost benefit of endovascular therapy lies in its lower patency rates and need for subsequent reintervention. This is evident particularly in the critical limb ischemia cohort in whom, at 1 year, endovascular therapy is trending to become the more expensive modality. The 11% difference in patency at 1 year and the early failures of patency (<30 days) are the major influences on this high cost. This economic benefit to open revascularization in patients with critical limb ischemia has been described before for patients with complex arterial occlusive disease.[3]

Claudicants treated by endovascular therapy have a noticeable trend toward cost savings compared with open therapy, despite a 14% differential in patency at 1 year. In particular, those treated by simple angioplasty with no other adjunctive interventions showed the greatest savings. This difference is driven by the fact that there were fewer early failures in the endovascular group and the often high expense of reintervention in the open group. The improved cost-benefit ratio of angioplasty compared with bypass in the treatment of claudicants has been noted in a recent Markov decision model-based study.[4] Of course, exercise therapy is an integral component in the treatment of intermittent claudication and may possess a positive cost-efficacy profile.[5] Our current database is restricted to open and endovascular revascularizations, and therefore, we are unable to comment on the application of this cost model to exercise therapy.

Our model offers a unique method by which to assess cost in vascular disease. It is straightforward to use with the data readily available to every practitioner. It is easy to see that this amortized cost model can be tailored to investigate cost-effectiveness for interventions on other vascular beds (e.g., carotid artery, aneurysmal disease). Unfortunately, this model is not without limitations. Arguably, patency may not be the best outcome measure. The correlation between patency and freedom from symptoms and improved functional capacity is not always 100%. Brewster

et al. clearly demonstrated this discrepancy.[6] In a study looking at patients with failed bypass grafts, 10% of claudicants and 21% of critical limb ischemia patients had improved symptoms despite having lost patency. Using patency as an outcome measure in this model may unnecessarily penalize interventions in certain circumstances.

While understanding the limitations of our model, we performed a subgroup analysis using limb salvage as the outcome measure. The 1-year limb salvage rates were 82% in the open group and 61% in the endovascular group (P=0.02). The amortized cost of limb salvage ($/day of limb salvage) was 127±36 in the open group versus 191±63 in the endovascular group (P=NS). The absence of statistical significance between the two groups is not surprising based on the patency data and the small sample size. Interestingly, the overall cost/day of limb salvage was lower than the cost/day of patency, reflecting that patients who lose patency but maintain limb salvage still had a successful outcome.

Other Analyses

Ultimately, it is likely that functional capacity is the most relevant outcome measure both preoperatively and postoperatively. Taylor et al. developed a novel definition of success for patients with critical limb ischemia that was defined as:[7]

- Patency up to the point of wound healing.
- Limb salvage for 1 year.
- Ambulation for 1 year.
- Survival for 6 months.

This definition nicely defines success as it relates to functional capacity of the patient. This in turn clearly demonstrates an overall benefit to the patient and society. It is easy to see how this measure of outcome could be substituted for patency in our model.

For many of the same reasons, reporting standards in claudicants should be based on improved functional capacity. There are mounting data that exercise programs have equivalent outcomes and lower costs than endovascular interventions at 1 year.[5] It is likely that interventions on claudicants will come under increasing scrutiny in the coming years. Reporting standards need to reflect the absence of lifestyle-limiting claudication, a minimum duration of patency, and the absence of lifestyle-limiting comorbidities.

It is important to note, whether using cost-per-day of patency or cost-per-day of limb salvage, that the costs are astronomical. It is likely that these costs are far beyond what would be deemed by society to be acceptable or sustainable. It is clear from this model that there must be a drive to improve patency. Efforts must be undertaken to improve technology, increase our knowledge of PAD and intimal hyperplasia, and refine patient selection criteria. Every effort should be made to reduce device costs. This can be aided by aggressive negotiations by hospitals, the government, and national vascular societies with device companies for lower costs. Some advocate that new devices should be limited to research centers until they have undergone the rigors of comparative effectiveness and are deemed acceptable. O'Brien-Irr forwarded a number of additional measures to reduce cost, including organizing vascular services within a hub, designating the radiology suite as the primary venue for endovascular interventions, and instituting selective stenting policies.[8]

FUTURE DIRECTIONS

With the looming health care crisis upon us, urgent action is needed. There is a strong need to adopt a standard cost model that looks at amortized costs and is universally understood and utilized. Information gleaned from databases can then be used to formulate strong guidelines that promote cost-effectiveness. As a society of vascular surgeons, we need to reassess reporting standards for interventions incorporating functional status and durability as outcome measures. As comparative effectiveness research gains momentum in this country, it is likely that reimbursement will be tied to procedural cost efficacy in some way. In this paradigm of health care, the most efficient and efficacious treatments will be sought. Our challenge will be to shape the system of vascular cost efficacy so that our patients and society both benefit. Ultimately, we must act to define our future lest it be defined for us.

REFERENCES

1. Contos B. Vascular Centers of Excellence - A Tactical Approach. Washington, D.C.: The Advisory Board Company; 2007.
2. Nowygrod R, Egorova N, Greco G, et al. Trends, complications, and mortality in peripheral vascular surgery. J Vasc Surg 2006;43:205–16.
3. Hunink MG, Wong JB, Donaldson MC, et al. Revascularization for femoropopliteal disease. A decision and cost-effectiveness analysis. JAMA 1995;274:165–71.
4. de Vries SO, Visser K, de Vries JA, et al. Intermittent claudication: cost-effectiveness of revascularization versus exercise therapy. Radiology 2002;222:25–36.
5. Spronk S, Bosch J, den Hoed P, et al. Cost-effectiveness of endovascular revascularization compared to supervised hospital-based exercise training in patients with intermittent claudication: a randomized controlled trial. J Vasc Surg 2008;48:1472–80.
6. Brewster DC, LaSalle AJ, Robinson JG, et al. Femoropopliteal graft failures. Clinical consequences and success of secondary reconstructions. Arch Surg 1983;118:1043–7.
7. Taylor SM, Cull DL, Kalbaugh CA, et al. Critical analysis of clinical success after surgical bypass for lower-extremity ischemic tissue loss using a standardized definition combining multiple parameters: a new paradigm of outcomes assessment. J Am Coll Surg 2007;204: 831–8.
8. O'Brien-Irr MS, Harris LM, Dosluoglu HH, et al. Lower extremity endovascular interventions: can we improve cost efficiency? J Vasc Surg 2008;47:982–7.

19

Subintimal Angioplasty

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INTRODUCTION

Peripheral arterial disease (PAD) affects 8 to 12 million Americans and its prevalence increases with age. The US Census Bureau expects the population over the age of 65 to reach 72 million by 2030, doubling its numbers from 2010[1]. In the year 2000, 144 626 lower extremity revascularization procedures took place in the US, with this number expected to increase as the population ages[2]. Analysis of Medicare claims data and the Nationwide Inpatient Sample report that the number of endovascular procedures more than tripled from 1996-2006 and began to outnumber open procedures[3–4]. Interestingly, as the number of endovascular cases has increased and open cases have become less frequent, the number of amputations has decreased. Besides the changes in the type of intervention, medical management of atherosclerosis and other different factors have been thought to be responsible for this trend.

Percutaneous interventions confer many obvious advantages when compared to open surgical revascularization. Endovascular therapies provide the ability to treat elderly debilitated patients whose risk for complications prohibits them from undergoing surgical revascularization. Although associated with a higher need for reintervention, percutaneous techniques do not preclude further surgical treatment[6]. Percutaneous transluminal angioplasty (PTA) however, is not without limitations. For example, PTA carries high risk of complications and technical failure when used to treat complex lower extremity arterial disease and long areas of occlusion[5]. In fact, PTA is most successful for lesions in the common iliac artery, with decreased patency results in distal occluded arteries[6]. Technological advances continue to emerge as alternatives to conventional angioplasty and stenting.

Bolia et al described subintimal recanalization for femoro-popliteal occlusions in 1989 with the claim that this technique could obtain better final angiographic results and be a better technique for long occlusions and diffuse arterial disease than traditional PTA[7]. Their index case occurred during an attempted PTA when they inadvertently dissected into the subintimal space and reentered the lumen of the artery distal to the occlusion. They then successfully inflated the balloon and achieved good flow with continued patency 8 years later[8]. Popularized in Europe in the 1990s, Subintimal

Angioplasty (SIA) became utilized in the US at the turn of the 21st century. Advantages of SIA include the increased size of the new arterial lumen created by SIA as compared to the true lumen after PTA. Additionally, SIA can treat longer areas of occlusion in an area of vessel without intima and thus at a lower risk for further atherosclerosis[9].

During attempts at SIA, difficulties include initiating the subintimal dissection as well as maneuvering the guide wire back into the true lumen. The introduction of devices to facilitate true lumen re-entry allowed SIA to emerge as a feasible and more widely applicable option for lower extremity revascularization in patients with complex lower extremity arterial disease (LEAD) and chronic total occlusions (CTO).

TECHNIQUE

After being pre-medicated with anti-platelet agents, patients arrive in the angiography suite and undergo conscious sedation and local anesthesia. For iliac occlusions, arterial access is achieved via an ipsilateral retrograde femoral approach. For femoral and infrapopliteal occlusions, available approaches include a contralateral retrograde femoral approach or an ipsilateral antegrade femoral puncture. The latter is favored for distal femoral and infrapopliteal lesions. With the availability of modern low-profile balloons and self-expanding stents, many lesions can be treated through a 4F or 5F sheath. Nevertheless, upsizing the sheath to 6 or 7F is often needed in order to accommodate for specific stents or re-entry devices. As a general principle, the end of the introducer sheath is placed as close to the proximal aspect of the occlusion as possible. Digital subtraction angiography is performed to document not only the lesion but also the runoff, so that it can be compared to post-intervention angiograms. Particular care is therefore taken to ensure that the origin and end of the CTO are both clearly defined and included in the field (Figure 19–1). Road mapping capabilities are therefore very helpful. Once the occlusion is demonstrated, IV heparin is administered and its dose modified based on activated clotting time (ACT) levels.

To set up for crossing the CTO, a soft, angled, hydrophilic 0.035-inch Glidewire (Terumo, Somerset, NJ) is introduced through a hydrophilic Glidecath catheter

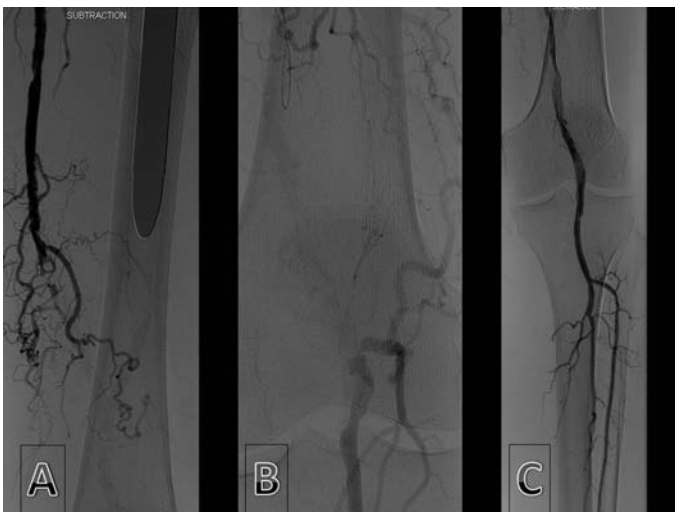

Figure 19-1. Pre-intervention angiogram revealing a chronic total occlusion (CTO) in the distal SFA. It is important to visualize the origin (A) and end (B) of the CTO as well as the distal runoff (C).

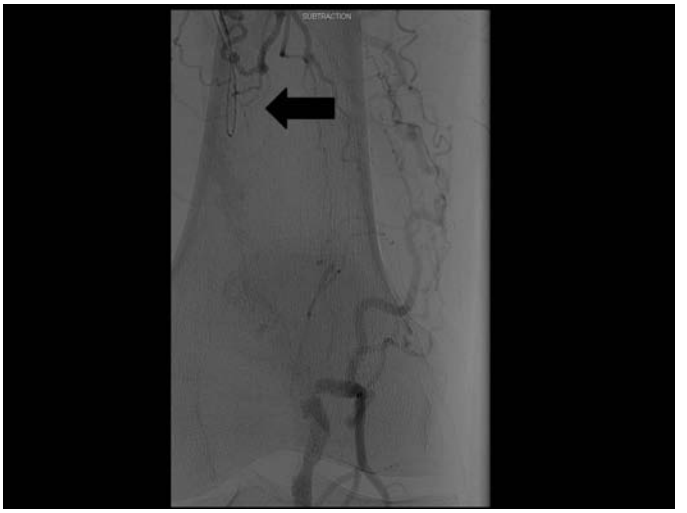

Figure 19-2. Angiogram showing a wire being advanced into the subintimal plane. The arrow points to the loop that the wire typically forms as it travels in the subintimal plane.

(Terumo) into the artery. When the guide wire hits the hard occlusion, a loop is formed which can then be used to create a dissection plane in the subintimal space (Figure 19–2). Additional guide wires and or sheaths can be employed for added support. For the most part, the wire will always be in the subintimal space or media at this point. Once in the dissection place, the catheter is gradually advanced over the guide wire.

Re-entry into the true lumen is attempted at less than or equal to 2 cm past the distal point of the occlusion, so as to not risk extending the dissection or blocking important collaterals. If difficulty entering the true lumen is encountered, wire manipulations may be performed or small amounts of contrast introduced into the dissection plane to identify any area of communication with the true lumen. The back of the hydrophilic wire may also be used as a dissection tool to cross difficult sections or to achieve re-entry when the soft end fails to do so.

Two commercially available devices can be used to facilitate true lumen re-entry and therefore increase the technical success rate of this procedure. The Pioneer catheter (Medtronic, Inc, Minneapolis, MN) is a 120-cm long 7F compatible intravascular ultrasound catheter (IVUS) with a solid state transducer. (Figure 19–3). The catheter holds two 0.014 inch guide wires (one for tracking the device and one for the reentry needle). The device is brought into the subintimal space at the desired re-entry location over a 0.014 inch hydrophilic guide wire after exchange of the initial 0.035-inch guide wire. The ultrasound then identifies color flow in the true lumen so that the catheter can be rotated to place the true lumen at the 12 o'clock position. A curved 25-gauge nitinol needle is deployed to create a pathway into the true lumen. The second 0.014 inch extra-support guide wire can then be advanced through the needle into the true lumen. The needle is then retracted and the catheter removed, leaving the 0.014-inch wire in place. Finally, the 0.035-inch guide wire is re-introduced to facilitate angioplasty and possible stenting.

The Outback catheter (Cordis, Miami Lakes, FL) is a 120-cm long 6F compatible device whose lumen accommodates a single 0.014 inch guide wire (Figure 19–4). The proximal end of the multipurpose angled catheter has a deployment handle mounted to a rotating hemostasis valve while the distal end is comprised of a 22-gauge curved nitinol re-entry needle. The device is passed over a 0.014 inch guide wire into the

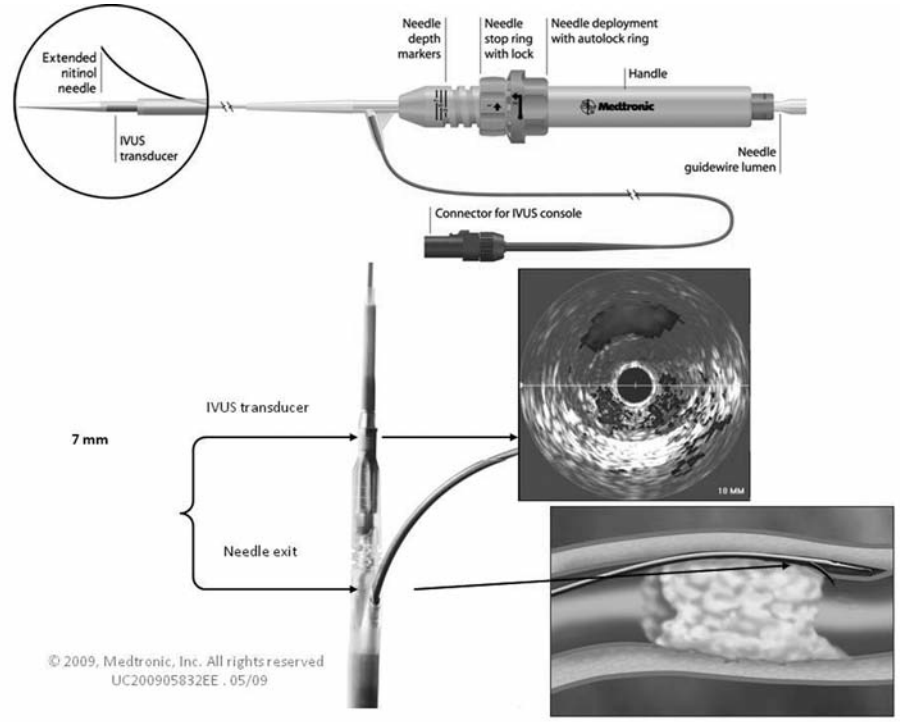

Figure 19-3. The Pioneer catheter (Courtesy of Medtronic, Inc., Minneapolis MN).

subintimal space where fluoroscopy is used for orientation and to align distal L and T markers with the true lumen. The guide wire is then partially withdrawn so that the nitinol needle can be advanced into the true lumen. A 0.014 support wire is then advanced into the true lumen. As mentioned with the Pioneer Catheter, at this point the 0.035-inch guide wire is exchanged in to allow for angioplasty and stenting[10].

Figure 19-4. The Outback catheter (Courtesy of Cordis, Inc., Miami Lakes Fl).

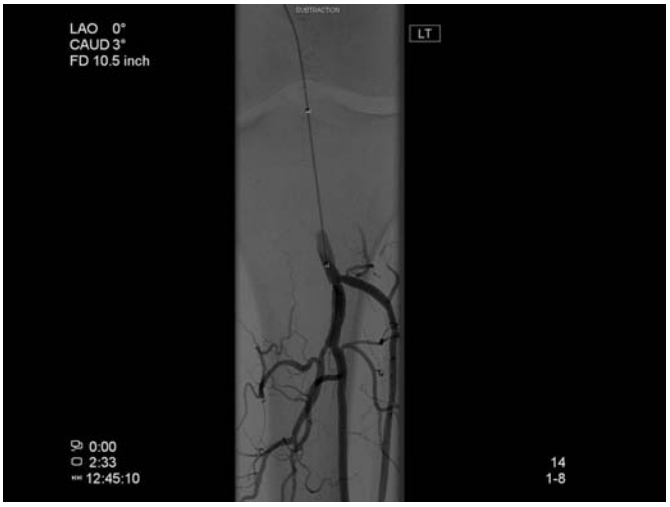

Figure 19-5. Angiogram showing intraluminal position of a catheter after re-entry from the subintimal plane.

Whether re-entry is achieved with the use of a re-entry device or by simple wire manipulation, it is critical to confirm true lumen position of the distal wire. This is done by advancing a catheter through the wire several centimeters past the re-entry site and performing an arteriogram. (Figure 19–5). After confirming intraluminal position, a wire is advanced and transluminal angioplasty is performed using appropriately sized long balloons. The use of stents has been a subject of debate. Some authors advocate for a selective use of stents only if a residual lesion with greater than 30% stenosis is observed after the use of balloon angioplasty[11]. One retrospective review from that particular group of investigators of selective stent placement, in cases of residual stenosis >30%, dissection flap creation, or severe calcification, found no difference in outcomes between the stented and un-stented group[12]. Others, however, recommend stent placement in all patients undergoing SIA of long occlusions (>15 cm), as this conferred a higher 1 year primary patency[13].

At the conclusion of SIA a completion angiography is performed to confirm the technical success of the procedure. This should also include the runoff circulation to rule out any possible embolization (Figure 19–6). We routinely administer dual antiplatelet therapy (aspirin and clopidigrel) for 3 months, after which only ASA is continued indefinitely. If a femoral closing device is used, patients are discharged within 2-3 hours. Patients treated with femoral compression stay on bed rest for 6 hours. Prior to discharge, ABIs are recorded. We obtain LEAFs at the first follow-up visit 2 weeks after the procedure, 6 months later, and yearly thereafter.

Complications

The nature and frequency of most complications after SIA are similar to those occurring after other endovascular interventions for LEAD: groin hematoma, false aneurysm formation at the puncture site, retroperitoneal hematoma, distal embolization, and cardiorespiratory complications. Most published series of SIA report a complications rate that ranges from 2%-17%[14]. Some complications, however, can be specifically related to SIA. Among these are distal embolization (1%-5%), distal arterial rupture, popliteal or tibial artery thrombosis (2%), aneurysm formation, and AVF formation (0.8%)[9]. Due to its minimally

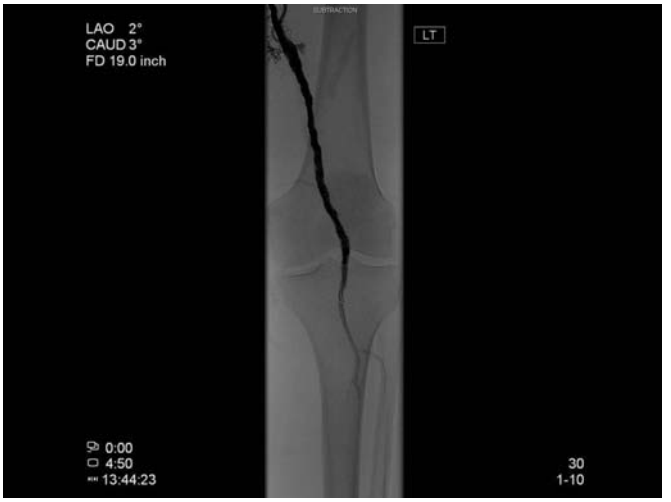

Figure 19-6. Completion angiogram after subintimal angioplasty and selective stenting of the lesion shown in Figures 19-1, 2 and 5.

invasive nature, SIA confers minimal procedural mortality. Perioperative mortality within 30 days has been reported as 0-0.8% with no deaths resulting from the procedure itself[15–16].

Results

Most investigators define technical success as forward flow through the previously occluded area of vessel on completion angiography. Some authors, however, will further specify the amount of residual stenosis as less than 30% in order for the procedure to be deemed a success. Two meta-analyses of available SIA studies report immediate technical success as 80%-90%[14,17]. Studies including specific lesion location prior to SIA revealed a lower technical success rate in crural than femoral occlusions[14]. One large retrospective study noted that 73% of technical failures occurred due to inability to re-enter the true lumen[16]. Extensive calcification also appears to predict technical failure of SIA. Bolia recorded this during his initial work with SIA, and other investigators have replicated his findings[8,18]. Mark et al quantified calcification using duplex ultrasound and identified a significant correlation between plaque echogenicity and failure of re-entry[19]. Heavy calcification, however, does not affect one year patency[18].

The published SIA literature lacks consistency in outcomes reporting. The two meta-analyses of available SIA data through 2007 report primary and primary-assisted patency at 50% and 55.8% at one year, respectively[14,17]. One year primary patency in more recent studies ranges from 45% to 68.5%[16,18]. Scott et al reported primary-assisted and secondary patency rates of 61% and 76%, respectively[16].

Few reports exist with patency rates after 1 year. Scott et al demonstrated a 20% 3-year primary patency, 38% 3-year primary-assisted patency, and 50% 3-year secondary patency with no significant differences between these outcomes[16]. When the 3-year primary patency group was broken down by site of occlusion, patency was highest in SFA occlusions and lowest in femorotibial occlusions (31% vs. 16%). With a smaller cohort of patients, Treiman et al showed a patency rate of 58% at 30-32 months[9]. These findings clearly differ from the 5-year patency seen with vein or PTFE (polytetrafluoroethylene) femoral popliteal bypass which have been reported as 74%-76% and 39%-52%, respectively[6]. Current literature exhibits consensus regarding the

ability for surgical bypass and repeated endovascular procedures to follow failed SIA procedures. Reintervention in the 3 years after SIA occurs in 28% of limbs treated[16].

One prospective study examining predictors of decreased patency identified length of occlusion and amount of crural runoff vessels (with one runoff vessel conferring 3.3 times higher risk of re-occlusion as compared to 2 or 3 runoff vessels) as significantly impacting patency at one year[15]. Others demonstrated significant reduction in primary patency with femorotibial occlusions or presence of CLI[16].

Many argue that limb salvage rates better represent procedural success than primary patency[20]. The meta-analysis by Bown et al found 18 reports of 12-month limb salvage with an average rate of 89.3%[17]. Met et al found 12-month limb salvage rates of 80%-90% with lower values in patients with CLI and mixed lesions[14]. At three years after SIA, Scott et al reported limb salvage rates as 75% in CLI patients and 67% in patients with disabling claudication[16]. Interestingly, although technical success and 1-year primary patency appear to be affected by location of the occlusion, location alone does not affect limb salvage rate[17]. No studies have adequately reported functional outcomes or amputation-free survival rates after SIA.

CONCLUSION

The 65th birthday of the baby boomers in 2011, and the imminent changes in regulation and reimbursement imposed by the recently approved health care reform, force us to critically analyze our current approaches to the management of PAD.

The lack of randomized controlled trials comparing SIA to other revascularization techniques limits our ability to effectively draw conclusions about its efficacy. In fact, the only randomized trial evaluating angioplasty versus surgical bypass was not powered to separately analyze SIA[21]. In addition, there are no randomized trials evaluating the utility of adjunctive stenting. Retrospective case-control and cohort studies comprise the majority of available research, while prospective trials make up 20%[17]. Future research will need to better define patient selection, degree of ischemia, and outcomes. Most importantly, long term follow-up is required. Few of the large published trials included the use of re-entry devices. With their increased use, further trials will better characterize their technical success and impact on patency.

SIA has emerged as a feasible strategy for the treatment of CTO and an attractive alternative to open surgical revascularization. Patients should be counseled on the trade-off between decreased morbidity and mortality and the lower long-term durability with the potential need for reintervention.

REFERENCES

1. He W, Sengupta M, Velkoff V, and DeBarros, Current Population Reports, 65+ in the United States: 2005, U S Census Bureau, Editor. 2005, US Government Printing Office: Washington, D.C. p. 23–209.
2. Anderson PL, Gelijns A, Moskowitz A, et al. Understanding trends in inpatient surgical volume: vascular interventions, 1980–2000. *J Vasc Surg*. 2004;39(6):1200–1208.
3. Goodney PP, Beck AW, Nagle J, Welch HG, and Zwolak RM.. National trends in lower extremity bypass surgery, endovascular interventions, and major amputations. *J Vasc Surg*. 2009;50(1):54–60.

4. Rowe VL, Lee W, Weaver FA, and Etzioni D. Patterns of treatment for peripheral arterial disease in the United States: 1996–2005. *J Vasc Surg*. 2009;49(4):910–917.
5. Rogers, JH and Laird JR, Overview of new technologies for lower extremity revascularization. *Circulation*. 2007;116(18):2072–2085.
6. Norgren L, Hiatt WR, Dormandy JA, Nehler MR, et al, Inter-Society Consensus for the Management of Peripheral Arterial Disease (TASC II). *Eur J Vasc Endovasc Surg*. 2007;33 Suppl 1: S1–75.
7. Bolia A, Brennan J, and Bell PR, Recanalisation of femoro-popliteal occlusions: improving success rate by subintimal recanalisation. *Clin Radiol.* 1989;40(3):325.
8. Bolia A, Miles KA, Brennan J, and Bell PR. Percutaneous transluminal angioplasty of occlusions of the femoral and popliteal arteries by subintimal dissection. *Cardiovasc Intervent Radiol*. 1990;13(6):357–363.
9. Treiman GS, Subintimal angioplasty for infrainguinal occlusive disease. *Surg Clin North Am*. 2004;84(5):1365–1380, viii.
10. Jacobs DL, Motaganahalli RL, Cox DE, Wittgen CM, and Peterson GJ, True lumen re-entry devices facilitate subintimal angioplasty and stenting of total chronic occlusions: Initial report. *J Vasc Surg*. 2006 43(6):1291–1296.
11. Matsumura J, Morasch M, Pearce W, and Yao J, eds. *Techniques and Outcomes in Endovascular Surgery*. 2009; Greenwood Academic Evanston, 208.
12. Schmieder GC, Richardson AI, Scott EC, Stokes GK, Meier GH, 3rd, and Panneton JM. Selective stenting in subintimal angioplasty: analysis of primary stent outcomes. *J Vasc Surg*. 2008; 48(5): 1175–1180; discussion 1180–1181.
13. Kougias P, Chen A, Cagiannos C, Bechara CF, Huynh TT, and Lin PH, Subintimal placement of covered stent versus subintimal balloon angioplasty in the treatment of long-segment superficial femoral artery occlusion. *Am J Surg*. 2009;198(5):645–649.
14. Met R, Van Lienden KP, Koelemay MJ, Bipat S, Legemate DA, and Reekers JA, Subintimal angioplasty for peripheral arterial occlusive disease: a systematic review. *Cardiovasc Intervent Radiol*. 2008;31(4):687–697.
15. Lazaris AM, Salas C, Tsiamis AC, Vlachou PA, Bolia A, Fishwick G, and Bell PR. Factors affecting patency of subintimal infrainguinal angioplasty in patients with critical lower limb ischemia. *Eur J Vasc Endovasc Surg*. 2006;32(6):668–674.
16. Scott EC, Biuckians A, Light RE, Burgess J, Meier GH, 3rd, and Panneton JM. Subintimal angioplasty: Our experience in the treatment of 506 infrainguinal arterial occlusions. *J Vasc Surg*. 2008;48(4):878–884.
17. Bown MJ, Bolia A, and Sutton AJ. Subintimal angioplasty: meta-analytical evidence of clinical utility. *Eur J Vasc Endovasc Surg*. 2009;38(3):323–337.
18. Antusevas A, Aleksynas N, Kaupas RS, Inciura D, and Kinduris S. Comparison of results of subintimal angioplasty and percutaneous transluminal angioplasty in superficial femoral artery occlusions. *Eur J Vasc Endovasc Surg*. 2008;36(1):101–106.
19. Marks, NA, Ascher E, , Hingorani AP, Shiferson A, and Puggioni A. Gray-scale median of the atherosclerotic plaque can predict success of lumen re-entry during subintimal femoral-popliteal angioplasty. *J Vasc Surg*. 2008;47(1):109–115; discussion 115–116.
20. Keeling AN, Khalidi K, Leong S, Given MF, McGrath FP, Athanasiou T, and Lee MJ. Subintimal angioplasty: predictors of long-term success. *J Vasc Interv Radiol*. 2009;20(8): 1013–1022.
21. Adam DJ, Beard JD, Cleveland T, Bell J, Bradbury AW, Forbes JF, Fowkes FG, Gillespie I, Ruckley CV, Raab G, and Storkey H Bypass versus angioplasty in severe ischaemia of the leg (BASIL): multicentre, randomised controlled trial. *Lancet*. 2005;366(9501):1925–1934.

20

Catheter-Based Plaque Excision: Is There a Role? Examining the SilverHawk System

Walter J. McCarthy, M.D., Chad Jacobs, M.D., and Ferenc Nagy, M.D.

As clinicians continue to expand the use of catheter-based treatment for lower extremity ischemia, numerous techniques have evolved. The comparison of competing methods is sometimes difficult, because new technology rapidly advances and is typically endorsed by groups of devotees who develop expertise then enthusiasm and publish early work. Numerous approaches have been advanced, including simple tapered dilation Dottering, laser plaque ablation,[1-2] mechanical plaque ablation, balloon angioplasty, cold balloon angioplasty, stents and drug-eluting stents, and subintimal dissection, among other ideas. For many patients with lower extremity ischemia, open surgical procedures provide excellent long-term solutions, but for some patients, percutaneous endovascular treatment is preferable.

The following discussion does not attempt to address the argument that all patients should have endovascular treatment "first" for lower extremity ischemia but does assume that some patients should and that judgment is involved. Rather, the atherectomy SilverHawk (EV3, Minneapolis, MN) method is examined, arguing that it does provide revascularization and limb salvage reliably and is a means to an end. The increase of perfusion provided by the SilverHawk technique may in some cases even be effective for patients who do not have the anatomy to support a tibial bypass. Thus, the procedure may in some cases be preferable, even in patients who do have excellent venous conduit and are good surgical candidates from a medical viewpoint. Long-term limb salvage rates will be discussed in comparison with what is likely the main competing technique, which is that of balloon angioplasty alone, with and without stents. Besides arguments to support atherectomy in selected patients, the chapter also presents useful techniques for atherectomy and representative cases.

BACKGROUND AND PUBLISHED INFORMATION

The published data related to the SilverHawk rotary atherectomy device are not extensive, but the journal articles all together are useful to review. Challenges in organizing the material from various articles involve different anatomic regions treated, different techniques, and varying endpoints of success. Thus, some authors report only femoral popliteal artery treatment and not tibial artery intervention. In all series, balloon angioplasty is used as an adjunct in areas in which the atherectomy is incomplete or is not possible, and in all articles, some stents are used. Some authors do not include calcified lesions at all and some predilate calcified tibial arteries before atherectomy. Follow-up is generally clinical, with different endpoints but with limb salvage nearly always recorded. The definitions of initial technical success and long-term treated area patency are always less rigorously established than, for example, in journal articles recording bypass graft patency. Excepting these limitations, the available articles do contain tremendously useful clinical information.

REVIEW OF THE SELECTED AVAILABLE LITERATURE

Thomas Zeller, from the Department of Angiology in Bad Krozingen, Germany, was an early practitioner with the SilverHawk device and published two papers in 2004(3). A long-term follow-up of cases came in 2007 and is very informative.[4] One hundred legs were treated but only lesions of the femoral and/or the popliteal artery were included. One third were primary cases, one third were cases with restenosis of a previously treated lesion, and one third were restenoses of previously placed stents. Low-pressure balloon angioplasty (<3 atm) was used in 59% and stents were utilized in 8% of cases. Any patient with a calcified lesion was excluded, and overall, the mean lesion length was 90 mm. All 100 procedures were reported as being initially successful. The lesions that were treated for the first time, that is, the primary lesions, had the best patency, and the restenosis cases had the worst long-term patency. For the primary treated lesions, the primary patency was 84% and 73% at 12 and 18 months. The secondary patency was better at 100% and 91% at 12 and 18 months. The restenotic lesions had lower patency. Follow-up was with duplex scanning, using a systolic peak velocity ratio of ≥2.4 as a definition of "restenosis." Rutherford categories and ankle-brachial indexes (ABIs) were both reported as improving significantly after treatment. Overall, of the 100 limbs, 3 ended up being amputated over the follow-up period. The authors' conclusions commented on the lowered durability in treating restenotic lesions in either native arteries or within stents, but overall, the authors applauded the 18-month results. They promoted the idea that atherectomy reduces vessel injury due to stretching "barotrauma," which is an inevitable result of any balloon angioplasty. They felt that barotrauma may promote intimal hyperplasia and restenosis. Clearly, this article is a review of the least difficult group of patients. It represents those with noncalcified lesions of the superficial femoral artery and/or popliteal artery only. Nevertheless, the patency for primary lesions is encouraging and the follow-up is rigorous, using a Kaplan-Meier presentation of all of the information.

In 2006, David Kandzari was instrumental in publishing two papers in the same issue of the Journal of Endovascular Therapy on the SilverHawk.[5-6] One reviews an experience with 69 patients from seven institutions, all with critical limb ischemia. The second paper outlines the findings of the TALON study (Treating Peripherals With SilverHawk: Outcomes Collection).[6] The second paper combines 601 consecutive

patients treated from 19 institutions. Both papers contain valuable information. Dr. Kandzari, who was at that time at Duke University, is the corresponding author for both papers. He is currently a cardiologist at the Scripps Research Institute in La Jolla, California.

The TALON registry enrolled all patients of any type starting in August of 2003 and followed them over 18 months. This study allowed any sort of lesion, including calcified lesions, and 26.8% of the lesions treated were occlusions. Most of the lesions were in the superficial femoral or the popliteal artery, which accounted for 70% of the treated areas. Of this region, 74.8% were above the knee and 25.2% were below the knee. The study, however, did include smaller vessels, and of the 601 patients, 24.4% involved vessels below the popliteal in the tibial-peroneal or tibial arteries. The study has the advantage of large numbers, with 601 patients and 748 limbs, and addresses 1258 specific lesions. It also includes patients from 19 institutions; thus a multitude of techniques and levels of expertise are represented.

The initial success was about 95% and of those, 10.5% required predilation with balloon angioplasty before the SilverHawk device could be passed. After treatment, 26.7% required balloon angioplasty for an unsatisfactory result and 6.3% required that a stent be placed. Follow-up duplex scanning was not common but was used, and the authors commented that the ABI increased from a mean of 0.7 to a mean of 0.86 after treatment. The percentage, without need for revascularization or amputation, was used as the endpoint. At 6 months, this group was 90%, and at 12 months, it was 80%. The TALON is the largest experience with the SilverHawk and the authors correctly conclude that an 80% primary success at 12 months is fairly comparable with bypass surgery without the morbidity of bypass surgery. However, some would argue that the 80% is somewhat lower than can be achieved with venous conduit to the popliteal or tibial vessels, which does approach 90% in many series. Also, patency beyond 12 months is not demonstrated.

Kandzari's other paper in 2006 focused on patients with critical limb ischemia of Rutherford category ≥5.[5] Patients were enrolled "irrespective of the complexity of their anatomy as an alternative to balloon angioplasty, bypass surgery, and/or amputation."

All had pain at rest with ankle pressure less than 50 mm Hg and/or ulcer gangrene or nonhealing wounds. Seven institutions contributed 69 patients, totaling 76 limbs over 12 months starting in August 2003. A total of 160 lesions were addressed. Femoral and/or popliteal lesions made up 61% and tibial perioneal trunk and tibial lesions 39%. Over all, 34% were totally occluded. The authors noted that 80% of the lesions were moderate to severely calcified. Initial success defined as <50% stenosis in the target lesion was 99%. Angioplasty without a stent was used in 11% and with a stent in 6%. There were no problems with embolization, and there were no vessel perforations reported. Follow-up was only through 6 months and patency was not addressed at all. The authors used freedom from amputation as an endpoint. Of the 76 limbs involved, they initially felt 48 were in need of major amputation but actually only 16 limbs were amputated over 6 months. Thus, three quarters were saved at the 6-month point. This endpoint of "not amputated yet" at 6 months is a soft, uncertain one, as any vascular surgeon will realize. The paper, however, does support aggressive treatment of advanced lesions.

Several single-center studies and reviews[7-10] have been published by experienced vascular programs recently after presentation at major vascular meetings. Yancey et al. from the University of Kentucky Medical Center, presented 16 patients at the 2006

Southern Association for Vascular Surgery.[11] They treated TASC (Trans-Atlantic Symptomatic Stenosis Classification) type C lesions with critical limb ischemia. They reported initially good results but their stenosis-free patency of the femoral popliteal area was only 22% at 12 months. Keeling and his group from Tampa, Florida, presented 60 patients at the Peripheral Vascular Surgery Society in 2006 with 1-year primary and secondary part patency of 62% at 76%, respectively.[12]

Keeling's report contrasts with that of Chungs et al.[13] from the University of Iowa at the 2007 winter meeting of the Peripheral Vascular Surgical Society treating 20 limbs. Their 12-month primary and assistant primary patency was only 10%, and they offered a serious word of caution about the device and procedure. The Cleveland Clinic reported 73 patients at the Southern Association for Vascular Surgery in 2007 with 1-year follow-up showing 43% primary patency and 57% secondary patency along with a 75% limb salvage.[14] These authors expressed some concern about the durability of the procedure. A recent report from the University of Arizona in Tucson by Biskup et al. reported 35 patients with primary and secondary patency of 66% and 70% at 1 year.[15] The authors presented the work at the 2008 winter meeting of the Peripheral Vascular Surgery Society. They noticed the worst patency in areas in which a previous intervention had been performed in the same location of the treated artery.

James McKinsey et al. at New York Presbyterian Hospital have been early, aggressive users of the SilverHawk device. They published their intermediate results in October 2008, including work from their institution from 2004 through 2007.[16] In all, 579 lesions in 275 patients were reported and 63% were for critical limb ischemia; 218 lesions were in the tibial vessels. The 18-month data were reported with primary and secondary patencies of 58% and 82% for claudication patients and 49% and 70.8% for the limb salvage group. There was no limb loss in the follow-up period for the claudication group, and overall, the limb salvage was 93%; 4.4% went on to have bypass surgery. The authors commented that in all the patients who required bypass, follow-up angiography showed the distal vessels to be unchanged. Thus, the initial atherectomy procedure did not seem to have worsened the situation.

McKinsey has questioned if the SilverHawk has an advantage over pure balloon angioplasty alone in the lower extremity. This is a yet unaddressed and very intriguing question. Restated the question is: do we do just as well with low-profile balloons in the lower extremity, particularly in the tibial vessels, or does the SilverHawk add something? To answer this, he has compared patients over the last 2 years to SilverHawk first or balloon angioplasty alone first. He has concluded from that work that there is a unmistakable statistical advantage in patency for the SilverHawk cohort. These data, however, are still unpublished but are presented here after personal review by the senior author of this chapter. Once this information is publicly available, it will be very helpful in directing the treatment prescribed by clinicians carrying for patients with lower extremity ischemia.

SILVERHAWK ATHERECTOMY TECHNICAL ADVICE

There are several technical suggestions regarding performing SilverHawk atherectomy that can help make the difference between an efficient case with hemodynamic success versus a difficult case with a less than optimal outcome. Outlined below are several technical hints that have come from our institution's combined experience thus far with SilverHawk atherectomy. Advance planning is critical to the success of these procedures, including

choice of access site, wire and sheath selection, atherectomy technique, and use of embolic protection.

Selecting femoral access is often one of the more critical decision points when performing SilverHawk atherectomy. Options include a retrograde femoral approach with percutaneous puncture of the contralateral femoral artery, antegrade ipsilateral femoral puncture, and ipsilateral surgical cutdown for an antegrade approach. The contralateral retrograde femoral approach, although initially appealing, does have limitations. Some have written that a controlateral approach is "safer" with fewer complications, but this may be because of its use in simple cases involving only the superficial femoral artery. This approach may often be more technically challenging, because it requires up-and-over access. Additionally, the length of the SilverHawk device may limit device accessibility to the infrageniculate vasculature, depending on a patient's body habitus. Thus, tibial access may mandate an antegrade puncture of the ipsilatenal common femoral artery. The additional curvature of the aortic bifurcation may provide difficulty with sheath kinking and also may cause additional difficulty with torque and manipulation of the SilverHawk device.

Using the Antegrade Approach

The antegrade ipsilateral percutaneous femoral approach has become our preferred approach, particularly for tibial lesions. Precise anatomic identification of the correct location for puncture (common femoral artery) is important to avoid complications. Fluoroscopy can be used to identify the femoral head, the inferomedial aspect of which corresponds to the location of the common femoral artery. Duplex ultrasonography can be used to visualize the common femoral artery and its bifurcation. Patients with a large, overhanging pannus may require retraction of the pannus, either by an assistant or with tincture of benzoin and tape placed to buttress the pannus preoperatively. We prefer to localize the common femoral artery with duplex ultrasound and access it in an antegrade fashion with a micropuncture kit. Once access has been obtained with a 7-French sheath, an initial hand injection is performed. If the sheath is within the profunda femoris artery, a guidewire is advanced into the distal branches of the profunda femoris. The sheath is then slowly withdrawn over the wire, and continued hand injection puffs are performed until the sheath is withdrawn into the lumen of the common femoral artery. Keeping a wire in the profunda femoris ensures that arterial access will not be lost as the sheath is withdrawn. A second guidewire can then be passed alongside the original guidewire to be directed into the superficial femoral artery. We have used an antegrade approach with increasing frequency to allow safe and easy access to the superficial femoral and infrageniculate arteries.

If body habitus or other technical factors prohibit either the ipsilateral antegrade or the contralateral retrograde percutaneous approach, a small cutdown can be performed with local anesthesia and sedation. Often, if the superficial femoral artery is patent proximally, this vessel itself can be exposed and then accessed using the Seldinger technique as in a percutaneous approach.

A third approach that has been described by various practitioners when antegrade access is technically not possible is to access the lesion from a retrograde direction, that is, from a tibial puncture. The dorsalis pedis artery can be accessed percutaneously under ultrasound guidance with a micropuncture kit, and the smallest of the SilverHawk devices can be passed retrograde without a sheath (bareback) to the target lesion.

Other Techniques

A variety of different lengths of sheaths should be on standby when performing SilverHawk atherectomy. All of the SilverHawk devices can be passed through a 7-French sheath despite markings on some of the packaging that indicate that an 8-French sheath is required. Several lengths of sheaths are useful. A short (13 cm) sheath is useful for initial access and for treatment of lesions in the proximal to mid-superficial femoral artery, particularly with antegrade access. A longer 7-French (35 cm) sheath is also useful for antegrade access when performing interventions primarily below the knee. Our approach has been to use a short sheath for initial access and, once wire access has been obtained across the lesions in the infrageniculate circulation, to advance a longer 7-French sheath positioning its tip in the popliteal artery. This has been found to provide more stability for delivery of the device and balloons, if necessary, and also protects the superficial fermoral artery during catheter exchanges.

Of additional consideration is the type of sheath to use when performing contralateral retrograde access for the up-and-over approach. Several of the standard 35- to 45-cm sheaths can to be advanced up and over the aortic bifurcation. However, nonreinforced sheaths will often kink in a patient with an acute aortic bifurcation. Certain sheaths such as the Pinnacle Destination (Terumo) sheath have adequate length to reach the contralateral common femoral artery or even the superficial femoral artery and are resistant to kinking, which otherwise can be quite troublesome when trying to pass the SilverHawk device over the aortic bifurcation.

Crossing lesions for treatment with SilverHawk atherectomy employs the same principles as crossing lesions for other types of intervention such as angioplasty and stent placement. The method most commonly used is to attempt to cross the lesion with a standard 0.035 glidewire. Use of a 5-French glide catheter is useful to add support for the glidewire. On occasion, crossed lesions require predilation with 2-, 3-, or 4-mm angioplasty balloons if the SilverHawk itself will not pass over the 0.014 wire. At times, difficult lesions can be crossed using a Dottering technique with the use of a catheter such as the Quick Cross (Spectranetics) catheter, which is a 5-French catheter with a straight but tapered tip. This catheter can help cross tight lesions or occlusions with a minimal amount of wire exposed distally, using the catheter to help Dotter across such lesions. Of course, once the catheter has been advanced across the occlusion, angiography must confirm that the catheter is intraluminal (Figures 20–1 through 20–8).

Once any target lesion has been successfully crossed with a 0.014 wire, the SilverHawk device is advanced, and it is recommended to treat the lesion in four quadrants: anterior, posterior, medial, and lateral. Depending on the size of the device and plaque burden, the cap may need to be emptied after four passes. Additional passes of the device may be then performed, focusing on specific areas of heaviest plaque burden. It is suggested that the inner wall of a bifurcation be avoided to minimize the risk of vessel perforation. Proximal lesions are treated prior to distal lesions so that the remaining devices, sheaths, and balloons, if necessary, can be passed across the previously treated lesions without risk of additional emboli (Figures 20–5 through 20–8).

It is worthwhile to be prepared to use distal embolic protection when performing SilverHawk atherectomy, particularly for calcified lesions. Filters are recommended to be used at all times when performing SilverHawk atherectomy using the RockHawk device. The RockHawk has an extra bur on its rotating blade that is useful for treating heavily calcified lesions; however, it is also much more likely to cause distal embolization. At our institution, we have, with increasing frequency, used a filter when performing any SilverHawk intervention in the superficial femoral artery. The filter is

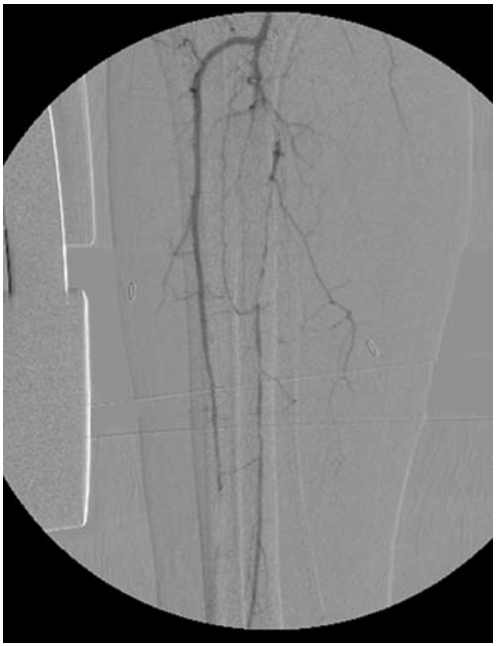

Figure 20-1. Peroneal occlusion preintervention.

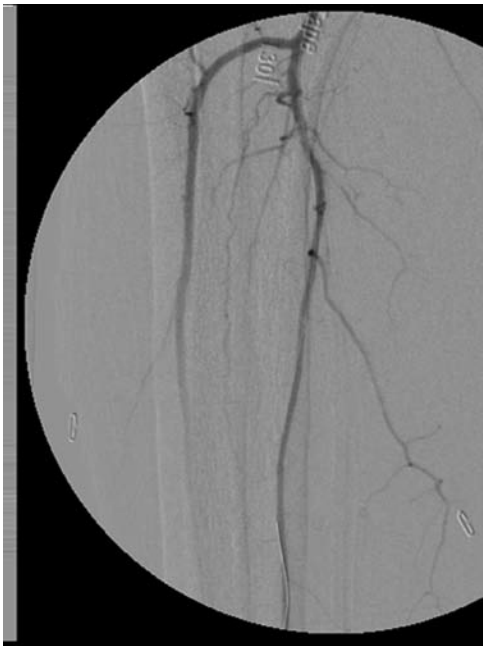

Figure 20-2. Peroneal artery post-SilverHawk atherectomy.

placed at the below-knee popliteal artery or tibioperoneal trunk and, more often than not, will return with a significant amount of atheromatous debris after completion of a superficial femoral artery SilverHawk intervention (Figures 20–9 through 20–12). Others have shown evidence of embolization in as many as 90% of lesions treated with the SilverHawk.[17-19] But fortunately most of these are of subclinical importance. Filters are less likely to be of benefit when performing tibial level intervention because the

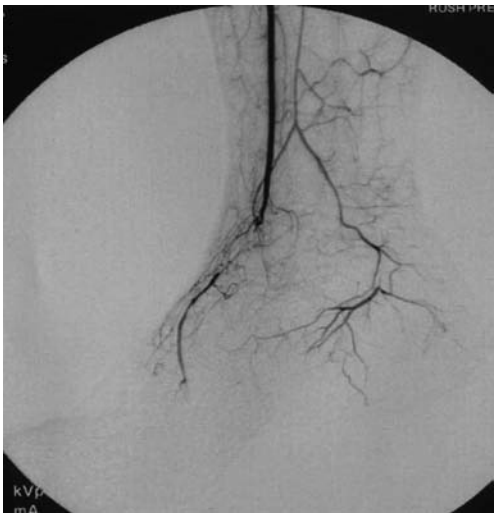

Figure 20-3. Dorsalis pedis occlusion preintervention.

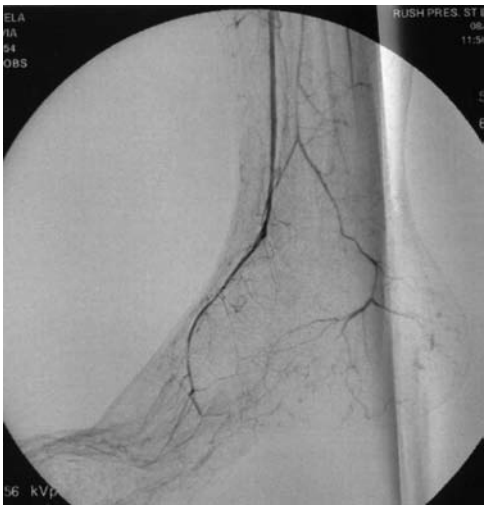

Figure 20-4. Dorsalis pedis artery post-SilverHawk atherectomy.

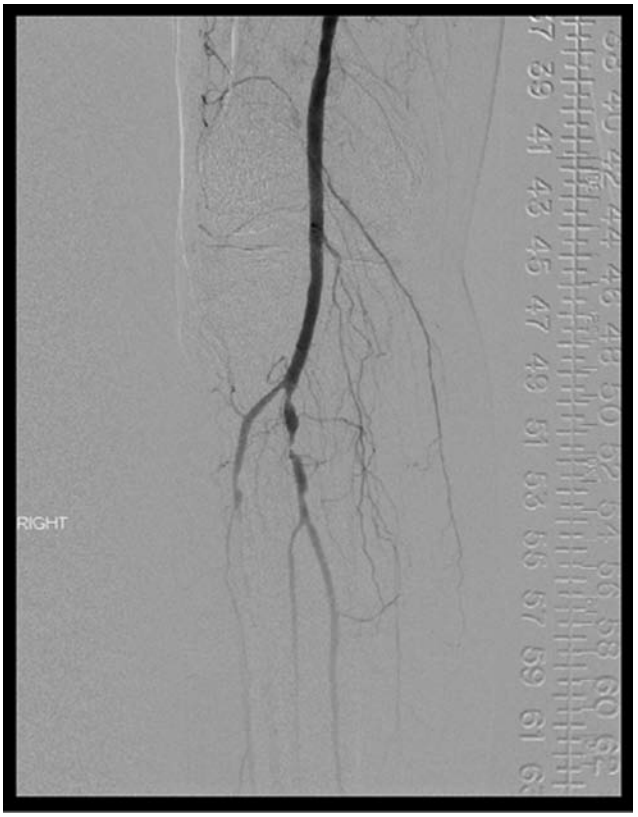

Figure 20-5. Tibioperoneal trunk stenosis preintervention.

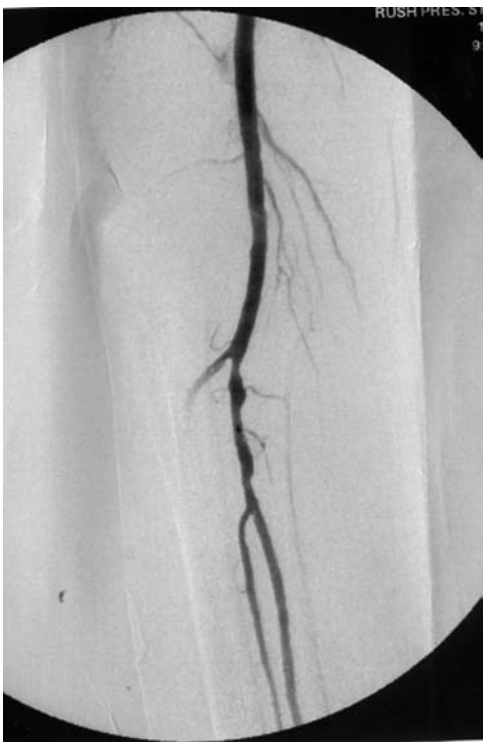

Figure 20-6. Tibioperoneal trunk stenosis post-SilverHawk atherectomy.

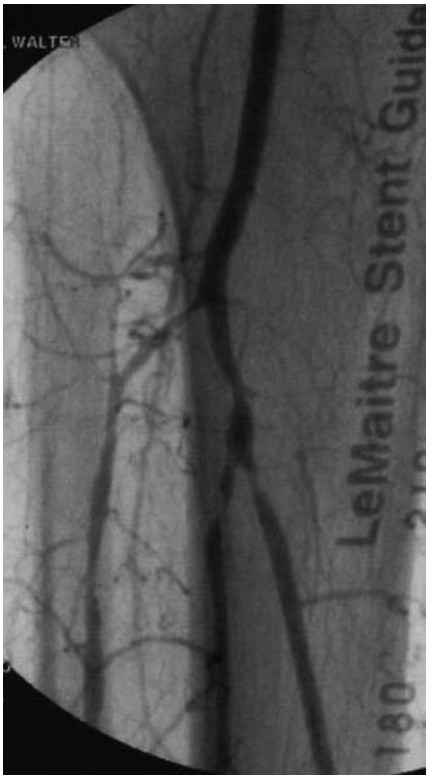

Figure 20-7. Peroneal and posterior tibial stenosis preintervention.

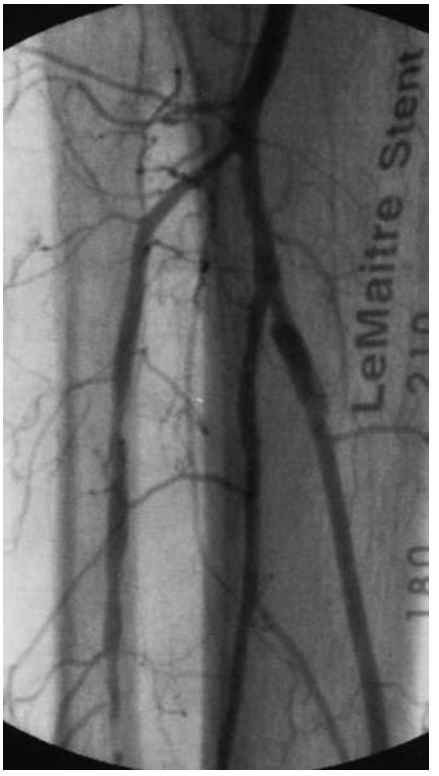

Figure 20-8. Peroneal and posterior tibial stenosis post-SilverHawk atherectomy.

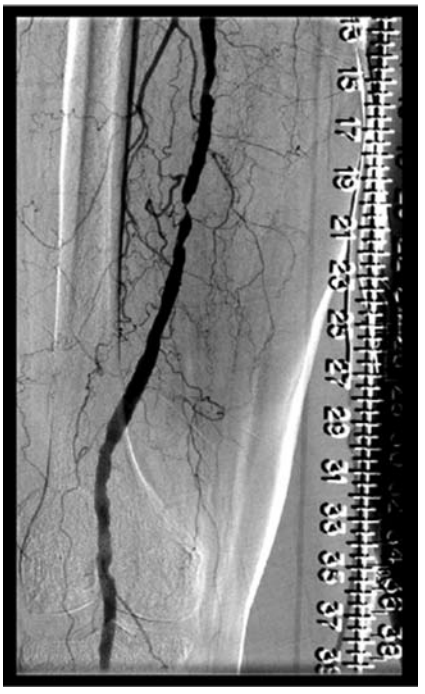

Figure 20-9. SFA stenosis preintervention.

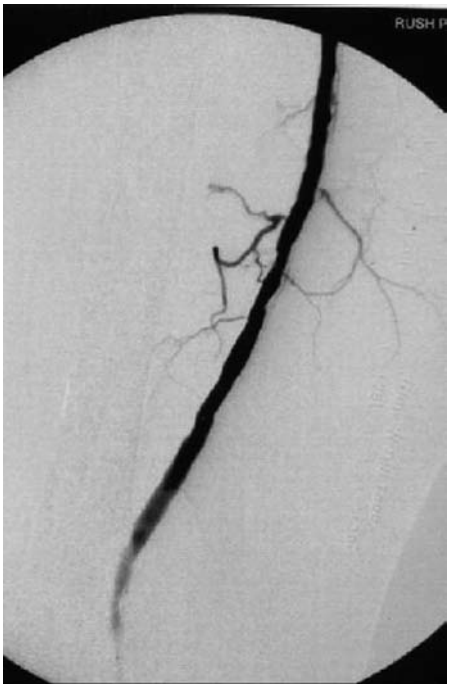

Figure 20-10. SFA post-SilverHawk atherectomy.

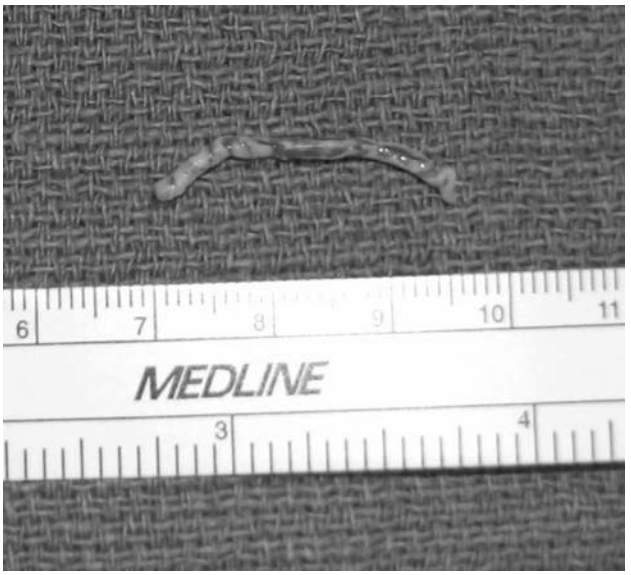

Figure 20-11. SFA atherectomy specimen.

Figure 20-12. Debris in filter basket post-SFA intervention.

filter may not successfully deploy in the distal tibial vessels and may cause either damage to the intima or significant spasm in the tibial vessels.

We have performed several hybrid procedures, using a prosthetic femoropopliteal bypass followed by SilverHawk atherectomy of the tibial runoff. Experience has shown that it is best to use the SilverHawk device with pressure in the tibial arteries after completion of the femoropopliteal bypass. Passing the SilverHawk device with the inflow

occluded can lead to confusing angiographic results, because the treated vessels do not distend after atherectomy as expected. We therefore perform the prosthetic bypass first and then advance the SilverHawk device through the functioning graft.

A final note is that just as with many other interventional procedures, "the enemy of good is better." A residual stenosis of 20 to 30% may be an acceptable final result for many target lesions. The risk of each additional pass of the SilverHawk device may include excessive thinning of the vessel wall with delayed pseudoaneurysm formation, severe arterial spasm, vessel perforation, occlusion, and distal embolization.

CONCLUSION

The SilverHawk directional atherectomy catheter has been studied only through a series of clinical registries and single-center database reports. Randomized trials against other means of treating lower extremity ischemia have not been conducted. The device offers the advantage of debulking atherosclerotic material and usually can be used without placing a stent or without an associated balloon angioplasty. It is useful in the femoral popliteal area and is also effective in treating tibial artery lesions. It appears that it can be reused if the patient develops restenosis and does not appear to damage the distal circulation if the patient does require a operative bypass in the future. Initial results are good, but the patency after 12-month follow-up does not compare with that of surgical bypass. The question of atherectomy versus initial balloon angioplasty remains unsolved, although initial preliminary information seems to support atherectomy in this regard.

REFERENCES

1. Wissgott C, Scheinert D, Rademaker, J, et al. Treatment of long superficial femoral artery occlusions with excimer laser angioplasty: long term results after 48 months. Acta Radiol 45:23–29, 2004.
2. McCarthy WJ, Vogelzang RL, Nemcek AA Jr, et al. Excimer laser-assisted femoral angioplasty: early results. J Vasc Surg 1991;13:5;607–14.
3. Zeller T, Rastan A, Schwarzwalder U, et al. Midterm results after atherectomy-assisted angioplasty of below-knee arteries with use of the silverHawk device. J Vasc Interv Radiol 2004;15:1391–7.
4. Zeller T, Sixt S, Schwarzwalder U, et al. Two-year results after directional atherectomy of infrapopliteal arteries with the silverHawk device. J Endovasc Ther 2007;14:232–40.
5. Kandzari DE, Kiesz RS, Allie D, et al. Procedural and clinical outcomes with catheter-based plaque excision in critical limb ischemia. J Endovasc Ther 2006;13:12–22.
6. Ramaiah V, Gammon R, Kiesz S, et al. Midterm outcomes from the talon registry: treating peripherals with SilverHawk: outcomes collection. J Endovasc Ther 2006;13:118–128.
7. Bunting TA, Garcia LA. Peripheral atherectomy: a critical review. J Interv Cardiol 2007; 20:417–24.
8. Garcia LA, Lyden SP. Atherectomy for infrainguinal peripheral artery disease. J Endovasc Ther 2009;16(Suppl II):II105–15.
9. Shrikhande GV, McKinsey JF. Use and abuse of atherectomy: where should it be used? Seminars in Vascular Surgery. J Sem vasc Surg 2008;21:204–9.
10. Ramaiah V. Endovascular Infrainguinal revascularization: technical tips for atherectomy device selection and procedural success. J Sem vasc Surg 2008;21:41–9.

11. Yancey AE, Minion DJ, Rodriguez C, et al. Peripheral atherectomy in transatlantic intersociety consensus type C femoropopliteal lesions for limb salvage. Association for Vascular Surgery. J Vasc Surg 2006;44:503–9.
12. Keeling WB, Murray LS, Stone PA, et al. Plaque excision with the SilverHawk catheter: early results in patients with claudication or critical limb ischemia. J Vasc Surg 2007;45;1; 25–31.
13. Chung SW, Sharafuddin MJ, Chigurupati R, et al. Midterm patency following atherectomy for infrainguinal occlusive disease: a word of caution. Ann Vasc Surg 2008;22:358–65.
14. Sarac TP, Altinel O, Bannazadeh M, et al. Midterm outcome predictors for lower extremity atherectomy procedures. J. Vasc Surg 2008;48;4:885–90.
15. Biskup NI, Ihnat DM, Leon LR, et al. Infrainguinal atherectomy: a retrospective review of single-center experience. Ann Vasc Surg 2008;22:776–82.
16. McKinsey JF, Goldstein L, Khan HU, et al. Novel treatment of patients with lower extremity ischemia: use of percutaneous atherectomy in 579 lesions. Ann Surg 2008;248:519–28.
17. Lam RC, Shah S, Faries PL, et al. Incidence and clinical significance of distal embolization during percutaneous interventions involving the superficial femoral artery. J Vasc Surg 2007;46;6:1155–9.
18. Shavelle DM. Plaque excision with distal protection: a logical step for superficial femoral artery SilverHawk atherectomy. J Invasive Cardiol 2009;21:11–2.
19. Shammas NW, Dippel EJ, Coiner D, et al. Preventing lower extremity distal embolization using embolic filter protection: results of the PROTECT registry. J Endovasc Ther 2008;15: 270–6.

21

When Bypass is not Enough: Endovascular Techniques and Wound Healing Modalities as Adjuncts to Limb Salvage in a Surgical Bypass-Oriented Approach to the Management of Critical Limb Ischemia

Martin Borhani, M.D., and William J. Ennis, D.O.

INTRODUCTION

Surgical bypass remains the gold standard for lower extremity revascularization against which all other intervention outcomes are measured. Vascular practitioners are often presented with patients in whom surgical bypass alone is inadequate to achieve limb salvage. This may be due to patterns of occlusive disease that preclude bypass or do not permit adequate reperfusion of the limb in jeopardy, following bypass. Endovascular interventions play an increasingly prominent role in treatment of lower extremity ischemia and may be considered first-line therapy in select patients. Alternatively, endovascular interventions may be used as adjuncts to surgical bypass to overcome limitations to bypass (inflow, outflow) or available conduit. In the near future, gene-based angiogenesis therapeutic options along with cell-based treatments may also provide primary or adjunctive options for the vascular clinician. Limb loss despite adequate revascularization may result when the degree of tissue loss is such that wound healing cannot be achieved without compromising acceptable functional capacity. Alternatively, limb loss secondary to a persistently nonhealing wound may occur when indirect revascularization fails to provide adequate collateralization to the specific target wound area. In the following brief chapter, we describe the utilization of endovascular techniques in a predominantly surgical-based approach to limb

salvage and discuss the role of wound care modalities to optimize wound healing and limb salvage in particularly challenging cases.

CRITICAL LIMB ISCHEMIA

Critical limb ischemia (CLI) remains one of the most challenging entities faced by vascular specialists.[1] Despite advances in both techniques and technologies, management of the ischemic extremity is more complex than ever. An ever-aging population of patients with peripheral vascular disease tests the limits of currently available treatment options. Prolonged life expectancy results in successive ischemic presentations due to progression of disease and the failure of prior interventions. Each clinical presentation becomes more challenging than the last as patients age, and the list of treatment options dwindles. In these patients, the benefits of standard surgical revascularization techniques must be weighed against their attendant risks. Less invasive endovascular techniques that are better tolerated by patients are become increasing more attractive as experience grows and data accrues. The safety and efficacy of endovascular interventions for the treatment of aorto-iliac and femoro-popliteal occlusive disease has been clearly demonstrated.[2] The role of endovascular techniques in the treatment of infra-popliteal disease, however, is not as clearly defined. In addition, there has been little discussion about the role of pulsatile flow versus indirect revascularization when ischemia is combined with extensive soft tissue loss. In the absence of definitive evidence, treatment strategies are individualized to the patient's clinical needs and the practitioners' experience and skills. Clearly there is a role for a blending of both surgical and endovascular interventions for the management of the ischemic extremity. Vascular specialists with expertise in both surgical and endovascular techniques remain best prepared to lead the development of this field. Multidisciplinary expertise is critically important in optimizing both limb salvage and functional status in patients with extensive tissue loss. Specialists in wound healing, soft tissue transfer and reconstruction, orthopedics, foot and ankle care, orthotics/prosthetics, and physical medicine constitute a partial list of those utilized.

APPROACH TO CLI

We have developed a predominantly surgical approach for the treatment of critical limb ischemia. The rationale for this is as follows: 1) Axial flow to the distal extremity is best established with a surgical bypass. 2) Superior long-term patency and limb salvage rates are reported following bypass utilizing autologous saphenous vein. 3) There is a theoretical concern about creating a "no re-flow" phenomenon with endovascular techniques. Theoretically, this would play a more important role with existing soft tissue loss.[3] Although we categorize this approach as "surgically based," endovascular techniques and wound healing adjuncts are indispensable components. Endovascular techniques are primarily utilized in patients with clearly favorable anatomy and in patients undergoing combined surgical-endovascular "hybrid" procedures. Of particular interest is the utilization of endovascular techniques as an adjunct to surgical revascularization in patients for whom surgical bypass alone is not possible or adequate to achieve limb salvage. This hybrid procedure paradigm is extended to the pre-operative evaluation and post-operative wound management of these complex patients.

Treatment is individualized based on patient age, comorbidities, clinical presentation, anatomy, and treatment goals. Clinical manifestations of rest pain or tissue loss in the face of CLI predispose to limb loss and represent absolute indications for intervention. For patients who present with ischemic rest pain, the general treatment approach is based on the distribution of occlusive disease, with the goal of therapy being relief of rest pain. Hemodynamically significant inflow disease is treated first. Typically, TASC A and B lesions of the aorto-iliac distribution, with the exception of external iliac disease, are treated with endovascular interventions. TASC C and D lesions, and significant external iliac disease, are treated surgically or with a combined surgical endovascular "hybrid' operation. Either approach results in delivery of normal pulsatile flow to the level of the profunda femoris artery. Patients without inflow disease and patients with persistent ischemic rest pain despite adequate inflow undergo infrainguinal revascularization. Femoro-popliteal TASC A and B lesions, with the exception of multi-segmental stenoses or diffuse calcifications, are treated by endovascular means. Patients with TASC C and D lesions, as well as more diffuse femoral popliteal disease, undergo bypass.

Patients who present with tissue loss are treated with a goal of restoring pulsatile flow to the wound bed. Inflow disease is treated in the same manner as described above with one exception: patients who will require concomitant or subsequent infrainguinal revascularization are more likely to undergo surgical rather than endovascular aorto-iliac reconstruction in order to ensure optimal inflow. Infra-popliteal occlusive disease is preferentially treated by saphenous vein bypass unless the distribution of disease is clearly focal and amenable to simple balloon angioplasty. If adequate saphenous vein conduit is not available and tibial disease is extensive, then the choice of alternative autologous vein or prosthetic conduit is weighed against endovascular intervention. If a suitable tibial target with axial runoff to the foot can be found, PTFE bypass with a distal vein segment adjunct, such as a Miller cuff or Linton patch, is performed.[4] Otherwise, patients may be considered for extensive endovascular-based interventions. Great consideration is the anatomical location of the soft tissue loss/wound and the vascular supply to this region. Recently, investigators have demonstrated improved limb salvage when direct revascularization to the wound specific angiosome is achieved.[5]

These general strategies are then tailored as the individual case dictates. Factors that may shift treatment strategy toward endovascular intervention instead of surgical bypass include patient age, prohibitive operative risks, limited expected lifespan, unsuitable autologous conduit for distal revascularization, lack of adequate target vessel, or hostile leg that prohibits surgical intervention. Examples of a "hostile" leg include marked edema, severe venous stasis changes, and open ulcers in the region of bypass target vessel. Factors that may shift treatment strategy towards surgical intervention include severe renal insufficiency that precludes contrast dye administration required for endovascular intervention. Additional considerations include the relationship between expected patency rates and likelihood of limb salvage. Patients in whom short-term patency following endovascular intervention are sufficient for limb salvage are more likely to be treated by endovascular means than patients who require long-term patency to maintain limb salvage. It is critical, therefore, that communication between the wound care and vascular surgery team identify specific goals of therapy so that the most appropriate treatment option is selected. Short-term goals of wound healing might be achieved through less invasive procedures allowing for more "definitive care" procedures to be reserved for possible future symptomatic vascular disease.

Aggressive local wound care can often result in long-term limb salvage even in cases where revascularization is not an option.[6] Careful consideration of all options must therefore be reviewed in order to avoid potentially making a problem situation worse.

ENDOVASCULAR ADJUNCTS AND WOUND HEALING STRATEGIES

Although we categorize this approach as "surgically based," endovascular techniques and wound healing adjuncts are indispensable components to this approach. Endovascular techniques are mainly utilized in four ways: 1) First-line therapy in patients with favorable anatomy as described above; 2) Part of a hybrid operation in which an endovascular intervention is performed to achieve inflow for a more distal bypass in order to minimize the extent of the operation or to decrease the length of autologous conduit required; 3) As an alternative to surgical bypass in patients with co-morbidities or anatomical features that preclude bypass; 4) As an adjunct to revascularization in cases where bypass or limb salvage alone was not considered possible, yet endovascular techniques alone were deemed ineffective. One such example of the last point is the use of thrombolytic disease in patients with CLI and without clinical or radiographic evidence of thrombus. Though perhaps counterintuitive, we have had success in select patients in achieving improved distal vessel patency. Though radiographically small, these improvements have created clinically relevant improvements which translated into limb salvage in otherwise non-salvageable circumstances. These improvements can, however, be quantitated through the use of micro-circulatory measurements such as trans-cutaneous oxygen monitoring and laser Doppler imaging.[7]

Hybrid procedures in the series were utilized for both inflow disease and infrapopliteal reconstructions. Many patients with concomitant common iliac stenoses/occlusion and external iliac stenoses/occlusion underwent combined endovascular recanalization and stent grafting of their common iliac disease followed by surgical iliofemoral revascularization utilizing bypass or iliofemoral eversion endarterectomy. Several patients in need of distal tibial or pedal bypass were found to have saphenous vein conduit of inadequate length for a complete lower extremity bypass. Suitable candidates underwent endovascular femoral-popliteal revascularization and concomitant popliteal-tibial or pedal vessel bypass.

Strategies for wound healing are of paramount importance in patients who present with extensive tissue loss. Adequate revascularization, however challenging, is often more readily achieved than wound healing. For example, a diabetic with extensive fore-foot gangrene or mid/hind-foot tissue loss, exposed tendon, or underlying osteomyelitis may not be considered a candidate for limb salvage based solely on the degree and distribution of tissue loss and infection. Therapies aimed at tissue repair and wound healing, founded in science and evidence based, are critical to the pre-operative planning and post-operative success of these complex limb-salvage cases. The understanding of available wound healing modalities, and the indications and rationale for each, form one of the pillars of a successful limb salvage program. There is a difference between revascularization of an extremity and hoping that it heals, and revascularization of an extremity and making it heal. Multiple reports have described the presence of a palpable dorsalis pedal pulse with continued non-healing of heel ulcers with subsequent limb loss, demonstrating the importance of regional perfusion differences.[8] These modalities include, but are not limited to, ultrasound therapy, negative pressure wound therapy (NPWT), ultraviolet light treatment, and electrical stimulation. Wound

healing is a complex pathway that is energy dependent. Non-healing wounds frequently require the use of physical modalities in order to achieve healing. Over the past 10-15 years, the approach to a patient with a chronic wound has evolved from pure observation and topical dressing selection, to an appreciation of the complex wound micro-environment with its numerous interdependent biochemical pathways. As the knowledge base continues to grow, so has the level of sophistication and detail with which diagnostic and treatment options are available in wound care.

There has been a tremendous increase in the use of negative pressure wound therapy in the past 5 years. The recent release of results from a randomized, prospective study using NPWT on partial diabetic foot amputations and a consensus paper on optimal use of NPWT have accelerated clinical adoption.[9] The NPWT device works by removing chronic wound fluid and bacteria, increasing angiogenesis, and providing positive biochemical effects through mechano-transduction. Mechano-transduction is derived from the intermittent stretching of the soft tissue generated by the vacuum pump and delivered to the tissue through the foam dressing. Negative pressure therapy is utilized as the initial therapy for the majority of open amputation and soft tissue debridement cases in this series. After a bed of granulation tissue is achieved, other energy-based modalities are employed.

Ultrasound is defined as a mechanical vibration transmitted at a frequency above the upper limit of human hearing (>20 kilohertz). The wound-healing community has recently shown an increased interest in both diagnostic and therapeutic ultrasound. One of the main mechanisms of action for ultrasound is the process of *cavitation*, the production and vibration of micron-sized bubbles within the coupling medium and fluids within the tissues. The movement and compression of the bubbles can cause changes in the cellular activities of the tissues subjected to ultrasound. *Microstreaming* is defined as the movement of fluids along the acoustical boundaries as a result of the mechanical pressure wave associated with the ultrasound beam. The combination of cavitation and microstreaming, which are more likely to occur with kilohertz ultrasound, provide a mechanical energy capable of altering cell membrane activity.[10,11] A new hypothesis known as the *frequency resonance theory* has been proposed which carries the above concepts to the protein and genetic level.[12] Mechanical energy from an ultrasound wave is absorbed by individual protein molecules, resulting in conformational changes. Signal-transduction pathways are also stimulated from the ultrasound-generated mechanical energy which results in a broad range of cellular effects.

The movement of cells towards an electrical field is known as *galvanotaxis*.[13] The use of electrical stimulation for wound healing utilizes this concept and allows the clinician to deliver exogenous electrical signals into wound tissue, thereby mimicking the natural underlying bioelectrical response to injury.

The concept of a skin "battery" and the potential implications for wound healing have been known since the early 1980s. It has now been well established that electrical stimulation can enhance the formation and release of VEGF (vascular endothelial growth factor) and is thereby a form of therapeutic angiogenesis.[14] Electrical stimulation is used in our practice to augment autologous skin graft take and to increase wound bed microvascular blood supply for planned skin graft coverage.

Ultraviolet light in the C-band wavelength is a form of radiant energy recognized in the past two centuries for its germicidal and wound healing effects.[10] Growth factors are released from epidermal cells exposed to UV irradiation which augments the healing cascade. There is a growing body of literature examining the anti-microbial effects of UVC irradiation at 254 nm. Conner-Kerr et al conducted an in vitro study

demonstrating the anti-microbial effects of UVC light using a 254 nm wavelength cold quartz generator with a 90% output of UV energy.[15] We employ ultraviolet light therapy to control local bioburden rather than using systemic antibiotics in many cases due to the known poor penetration of systemic antibiotics into granulation tissue.

Pulsatile blood flow results in shear forces at the endothelial surface and results in chemical signaling and transduction. This intermittent pressure is vital to the health of the endothelium. Ischemic tissue does not have the benefit of pulsatile flow and after revascularization, the condition of no-reflow can prevent the transmission of pulsatile pressures to the tissue level. The use of pulsed ultrasound, intermittent negative pressure, and other energy-based modalities have a final common pathway that results in forces at the cell level initiating biochemical changes including the production of growth factors, nitric oxide production, and the reduction of oxidative stress.[16,17]

CASE SERIES

We retrospectively reviewed 720 consecutive interventions for lower extremity ischemia performed by a single vascular surgeon (MB) from July 2001 to May 2008. Interventions performed for acute ischemia (trauma, dissection, embolic/thrombotic events) were excluded from further review. Of the remaining 584 interventions, a total of 443 were performed for CLI. Of those, 147 interventions were performed for rest pain and 296 for tissue loss. (Table 21–1) Approximately 40 percent of patients presented with a history of failed prior interventions for lower extremity revascularization. The majority of interventions performed were surgical regardless of clinical presentation or level of anatomic revascularization. Breakdown by case type and anatomic level is shown in Table 21–2. Endovascular techniques were typically reserved for patients with clearly favorable anatomy and reserved for patients who were not surgical candidates due to either anatomic restrictions or physiologic prohibitions. Endovascular interventions performed are shown in Table 21–3. Patients were grouped according to their original presentation. For example, patients presenting with rest pain who subsequently progressed to manifest tissue loss remained in the rest pain group and all subsequent interventions for limb salvage were counted in that

TABLE 21-1. DISTRIBUTION OF PATIENTS, EXTREMITIES, TYPE OF PROCEDURE, AND MORTALITY RATES FOR INTERVENTIONS PERFORMED FOR CHRONIC LIMB ISCHEMIA

	Claudication	Rest pain	Tissue loss	Total
Patients	108	109	209	426
Mean age	62.5	65.3	69.3	66.4
% male	57.4	50.7	64.1	58.9
Extremities	134	123	244	501
Interventions	141	147	296	584
Open surgical	93	122	230	445
Inflow	44	47	42	133
Outflow	49	75	188	312
Endovascular	42	16	45	103
Hybrid	6	9	21	36
Peri-operative mortality (%)	0	2.0	1.7	1.4

TABLE 21-2. DISTRIBUTION OF SURGICAL INTERVENTIONS PERFORMED FOR CHRONIC LIMB ISCHEMIA BY CLINICAL PRESENTATION AND ANATOMIC LEVEL OF REVASCULARIZATION

Number of procedures	Claudication	Rest pain	Tissue loss	Total
Inflow				
Thoraco-femoral	0	0	2	2
Aorto-bifemoral	19	15	19	53
Aorto-femoral/revision	5	9	5	19
Iliofemoral bypass/TEA	17	18	11	46
Axillo-femoral	0	0	1	1
Femoral-femoral	2	2	3	7
Obturator foramen	1	3	1	5
Outflow				
Femoral/profunda	7	17	6	30
Femoral-popliteal	34	27	49	110
Above knee-vein	0	1	5	6
Above knee-prosthetic	20	10	20	50
Below knee-vein	10	16	19	45
Below knee-prosthetic	4	0	5	9
Popliteal-tibioperoneal	1	2	4	7
Femoral/popliteal-tibial	7	29	99	135
Vein	5	15	73	93
Prosthetic	0	14	25	39
Cryopreserved vein	0	0	1	1
Femoral/popliteal-pedal	0	0	30	30
Plantar	0	0	14	14
Dorsal pedal	0	0	16	16
Composite graft	1	3	11	15
Composite sequential	1	3	9	13

category. Conversely, patients in whom the original intervention was performed for limb salvage had all subsequent interventions counted in that category even if wound healing had been previously achieved. Outcomes were assessed through direct physical examination or by telephone interview. Follow-up was available in 74% of patient presenting with

TABLE 21-3. DISTRIBUTION OF ENDOVASCULAR INTERVENTIONS PERFORMED FOR CHRONIC LIMB ISCHEMIA BY CLINICAL PRESENTATION AND ANATOMIC LEVEL OF TARGET LESIONS

Number of interventions	Claudication	Rest pain	Tissue loss	Total
Angioplasty/stent/stent graft				
Aorta	2	0	2	4
Iliac	27	18	16	61
Femoral	12	16	41	69
Popliteal	1	2	4	7
Tibial	0	0	23	23
Atherectomy	0	1	4	5
Thrombolysis	0	3	8	11

Note: Interventions listed are "unbundled," Thus, multiple interventions above may constitute a single procedure reflected in Table 21-1.

TABLE 21-4. RATES OF LIMB SALVAGE AND AMPUTATION-FREE SURVIVAL FOLLOWING REVASCULARIZATION IN PATIENTS PRESENTING WITH REST PAIN (n = 78, MEAN FOLLOW-UP 35 MONTHS) AND TISSUE LOSS (n = 142, MEAN FOLLOW-UP 33 MONTHS)

	Limb salvage (%)		Amputation-free survival (%)	
Follow-up	Rest pain	Tissue loss	Rest pain	Tissue loss
6 months	97.2	87.8	91.0	83.3
1 year	91.8	86.0	82.4	74.7
2 years	88.9	81.2	72.7	76.0
3 years	88.5	91.7	65.7	65.7
4 years	84.2	88.0	61.5	65.4
5 years	83.3	82.4	58.9	50.0

rest pain and 68% of patient with tissue loss. Rates of limb salvage and amputation free survival are shown in Table 21–4. These outcomes are consistent with published reports of limb salvage and survival following lower extremity revascularization. In this series, we report an increase in limb salvage rates at 3 years which is sustained at 4 and 5 years. This likely reflects a change in referral patterns that occurred during the corresponding period which resulted in a higher proportion of patients with advanced tissue loss and failed prior interventions.

ENDOVASCULAR ADJUNCTS

The proportion of endovascular procedures, alone or as part of a hybrid operation, was highest in interventions performed for claudication (34%). However, the volume and extent of endovascular interventions was greatest in those performed for tissue loss (98 target lesions). This increase was most notable for infra-popliteal revascularizations. Performance of tibial angioplasty, stenting, atherectomy, and thrombolysis was markedly increased in the tissue loss group. Tibial stenting was reserved for patients with stenoses refractory to simple angioplasty or flow limiting dissections (Figure 21–1). Thrombolysis performed in this series was utilized for chronic disease, all of which were long-standing with advanced tissue loss and no other reasonable surgical or interventional alternatives.

This utility of endovascular therapy as an adjunct to bypass surgery and subsequent wound healing strategies translating into limb salvage is best illustrated by the following examples:

Case 1

A 69-year-old man with CLI was evaluated for a chronic, large, necrotic, dorsal foot ulcer with underlying osteomyelitis. He had been deemed non-reconstructable 4 months prior. He was admitted to the hospital with wet gangrene of the dorsal forefoot. Following extensive debridement, arteriogram was performed. This demonstrated complete 3-vessel tibial occlusion without reconstitution of an adequate target for bypass or endovascular recanalization. Intra-arterial tissue plasminogen activator (tPA) infusion was administered for 72 hours, after which a repeat arteriogram demonstrated reconstitution of a segment of proximal dorsal pedal artery. (Figure 21–2) Surgical exploration revealed a diseased but

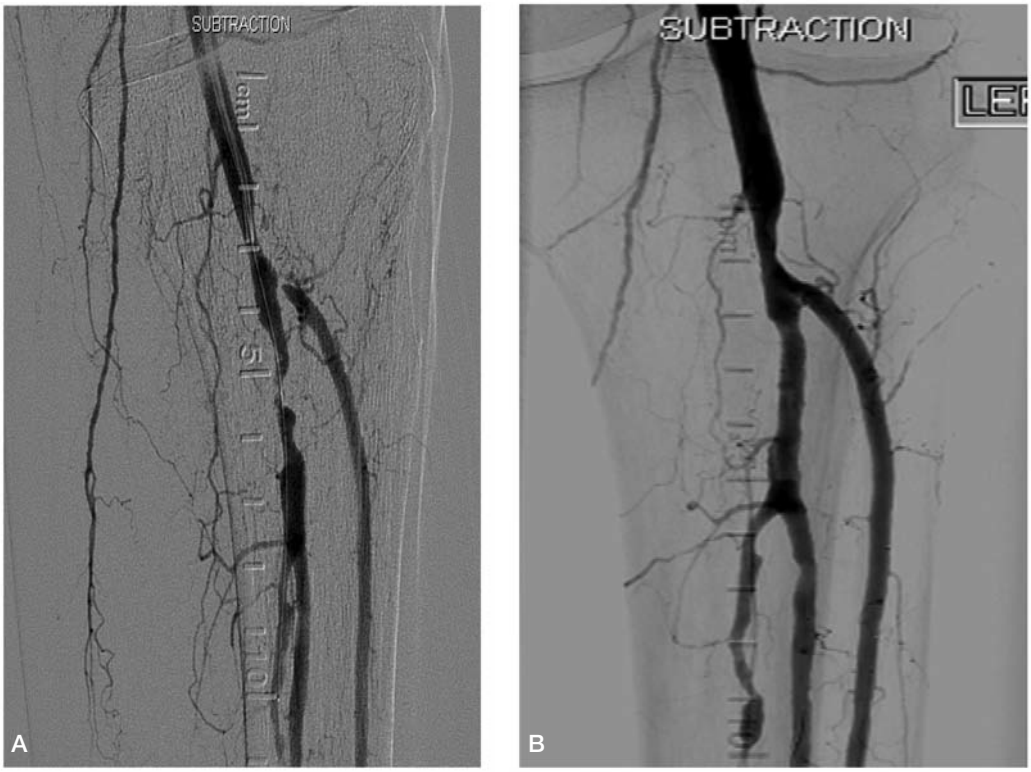

Figure 21-1. Arteriogram of below-knee popliteal artery and popliteal trifurcation in a patient with forefoot gangrene before (A) and after (B) angioplasty and stenting of anterior tibial artery and tibioperoneal trunk.

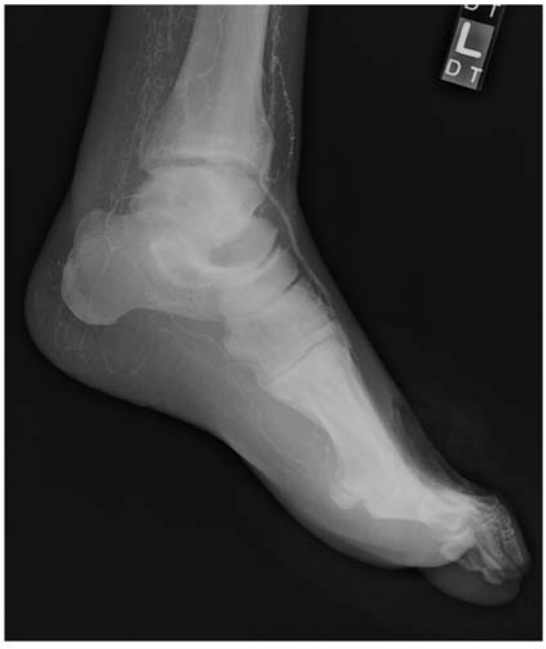

Figure 21-2. Arteriogram of left foot following 72 hours of intra-arterial tPA infusion. The short segment of reconstituted dorsal pedal artery represents a target for revascularization that was previously inadequate. Dorsal forefoot soft tissue defect is apparent.

suitable vessel for distal revascularization. The patient underwent a popliteal-to-dorsal pedal artery bypass with a reversed saphenous vein. Subsequently an open guillotine amputation was performed due to the extent of soft tissue loss and bony involvement. Multimodal wound care successfully generated a granulating wound bed which was subsequently covered by an autologous split-thickness skin graft.

Case 2

A 50-year-old female with a 37-year history of insulin-dependent diabetes mellitus presented with a 2-year history on a non-healing ulcer on the plantar aspect of the left great toe. Extensive multimodal wound care therapies failed to achieve healing. Arteriogram demonstrated normal macrovascular flow to the level of the ankle and extensive pedal vessel occlusive disease, most severe at the level of the forefoot (Figure 21–3A). Transcutaneous oximetry mapping of the foot failed to identify a level of forefoot amputation likely to heal. The patient was treated with intra-arterial infusion of tPA (0.5 mg/hr for 48 hours). Repeat arteriogram demonstrated restored axial patency to the medial plantar artery with opacification of plantar metatarsal and digital vessels at the region of the first metatarsal-phalangeal joint (Figure 21–3B). Repeat transcutaneous oximetry demonstrated improvement in oxygen tensions at the medial forefoot. The patient underwent successful amputation of the great toe at the metatarsal phalangeal joint followed by wound healing and sustained limb salvage.

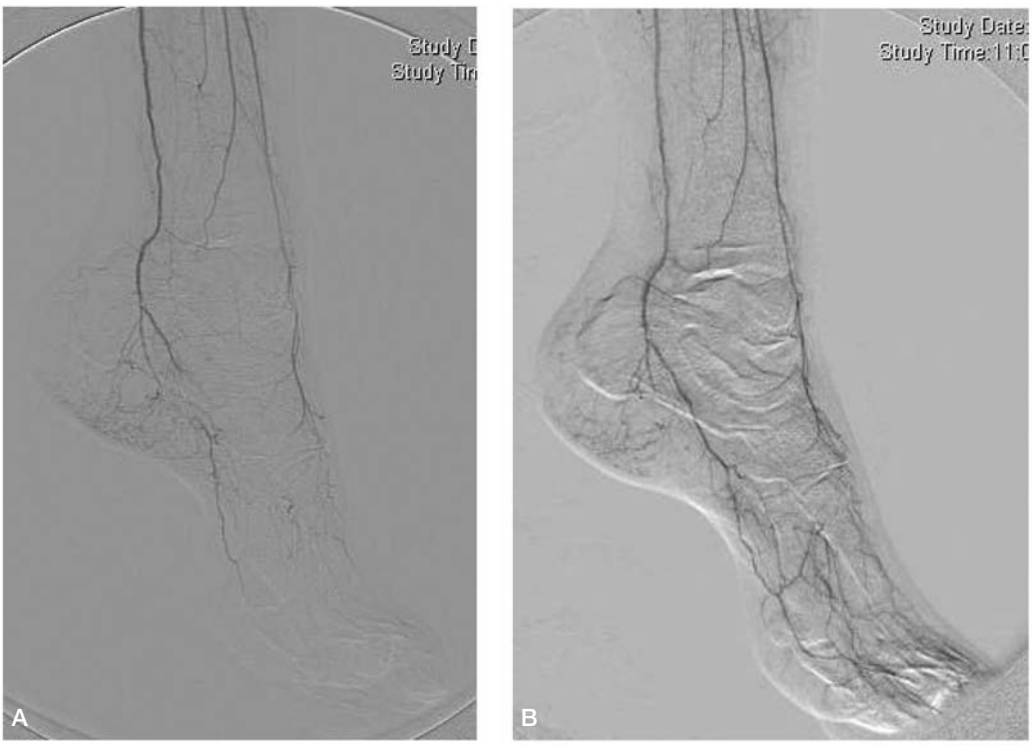

Figure 21-3. Arteriogram of left foot before (A) and after 48-hour intra-arterial tPA infusion (B).

REFERENCES

1. Novo S, Coppola G, Milio G. Critical limb ischemia: definition and natural history. *Curr Drug Targets Cardiovasc Haematol Disord*. 2004;4(3):219–225.
2. Nguyen MC, Garcia LA. Recent advances in atherectomy and devices for treatment of infrainguinal arterial occlusive disease. *J Cardiovasc Surg*. (Torino) 2008;49(2):167–177.
3. Abbo KM, Dooris M, Glazier S, O'Neill WW, Byrd D, Grines CL, et al. Features and outcome of no-reflow after percutaneous coronary intervention. *Am J Cardiol*. 1995;75(12): 778–782.
4. Trubel W, Schima H, Czerny M, Perktold K, Schimek MG, Polterauer P. Experimental comparison of four methods of end-to-side anastomosis with expanded polytetrafluoroethylene. *Br J Surg*. 2004;91(2):159–167.
5. Neville RF, Attinger CE, Bulan EJ, Ducic I, Thomassen M, Sidawy AN. Revascularization of a specific angiosome for limb salvage: does the target artery matter? *Ann Vasc Surg*. 2009; 23(3):367–373.
6. Marston WA, Davies SW, Armstrong B, Farber MA, Mendes RC, Fulton JJ, et al. Natural history of limbs with arterial insufficiency and chronic ulceration treated without revascularization. *J Vasc Surg*. 2006;44(1):108–114.
7. Ennis WJ SD, Meneses P. "Technologies for Assessment of wound microcirculation" In *Chronic Wound Care: A clinical source book for healthcare professionals*. 4th ed. Krasner, Sibbald, Rodeheaver, eds. Malverne, PA: HMP Communications, April 2007. pp. 417–426.
8. Treiman GS, Oderich GS, Ashrafi A, Schneider PA. Management of ischemic heel ulceration and gangrene: An evaluation of factors associated with successful healing. *J Vasc Surg*. 2000; 31(6):1110–1118.
9. Armstrong DG, Lavery LA. Negative pressure wound therapy after partial diabetic foot amputation: a multicentre, randomised controlled trial. *Lancet*. 2005;366(9498):1704–1710.
10. Ennis WJ, Lee C, Meneses P. A biochemical approach to wound healing through the use of modalities. *Clin Dermatol*. 2007;25(1):63–72.
11. Dinno MA, Dyson M, Young SR, Mortimer AJ, Hart J, Crum LA. The significance of membrane changes in the safe and effective use of therapeutic and diagnostic ultrasound. *Phys Med Biol* 1989;34(11):1543–1552.
12. Johns LD. Nonthermal effects of therapeutic ultrasound: The Frequency Resonance Hypothesis. *J Athl Train*. 2002;37(3):293–299.
13. Mycielska ME, Djamgoz MB. Cellular mechanisms of direct-current electric field effects: galvanotaxis and metastatic disease. *J Cell Sci*. 2004;117(Pt 9):1631–1639.
14. Patterson C, Runge MS. Therapeutic angiogenesis: the new electrophysiology? *Circulation*. 1999;99(20):2614–2616.
15. Conner-Kerr TA, Sullivan PK, Gaillard J, Franklin ME, Jones RM. The effects of ultraviolet radiation on antibiotic-resistant bacteria in vitro. *Ostomy Wound Manage*. 1998;44(10):50–56.
16. Ennis. Microcirculation: The push-pull theory. In: P. M, editor. *Diabetic Limb Salvage Conference*. Georgetown University; 2007.
17. Ennis WJ BM, Meneses P. Re-establishing macro vascular flow and wound healing; beyond the vascular intervention. *Vascular Disease Management*. 2008;5(3):56–62.

22

Iliac Artery Occlusion

Daniel G. Clair, M.D.

Surgeons have long been comfortable caring for the patient with iliac artery occlusion. These patients have been offered a time-tested, open surgical procedure that offers durability and safety in the form of aortobifemoral bypass. Evaluations of this form of therapy have revealed excellent safety and efficacy of this procedure, with low perioperative mortality and morbidity coupled with sustained long-term patency.[1-3] These reports have documented the mortality to be 3% to 4% and five- and 10-year patency rates to be as high as 85% and 75%, respectively. With these results in mind, the vascular surgical community has confidently advocated the open treatment of this entity. Additionally, the introduction of extra-anatomic reconstructions for this process[4,5] have allowed the treatment of those patients felt to be too sick to undergo an open procedure of the aorta. These procedures offer a means to reconstruct inflow to the lower extremities with little of the physiologic insult that can be incurred with surgery on the aorta. Finally, the addition of interventional options to the treatment of peripheral arterial disease has highlighted the relative ease with which patients can recover from these procedures, and the limited morbidity and mortality these techniques offer.

It is with this understanding in mind that we attempt here to define the nature of iliac artery occlusion and the options available for treating this process. Although the performance of open surgical procedures offers durable inflow options, the use of interventional options has for some time been able to achieve excellent early results with little risk to the patient. Deciding on the appropriate form of therapy for these patients requires an understanding of the normal extent and natural history of the disease, along with the outcomes of the procedures utilized to treat patients, all taken in the context of the overall general health of the individual patient in question.

AORTOILIAC OCCLUSIVE DISEASE

Occlusive disease of the aorta and iliac arteries tends to occur earlier than infrainguinal peripheral occlusive disease of the lower extremity arteries. This is evidenced by the lower average age at which patients usually present with occlusive disease.

Patients with disease in this area typically are in their '50s, approximately 10 years younger than those with occlusive disease in the infrainguinal arterial tree. Differing distributions of the pattern of aortoiliac occlusive disease have been described.[6] In type I disease, the atherosclerotic plaque is isolated to the distal aorta and proximal common iliac arteries. Type II disease is distributed throughout the infrarenal aorta and the common and external iliac arteries. Type III disease involves diffuse disease in the vessels both above and below the inguinal ligament. The majority of patients (65%) have type III disease and only 35% have the process limited to the vessels above the inguinal ligament (types I and II). The most frequent cause of this problem is atherosclerosis and the most common place atherosclerotic plaque develops is in areas of turbulent blood flow. In the aortoiliac segment of the vascular system, this most commonly occurs at the aortic bifurcation and at the bifurcation of the iliac arteries. Several authors have noted the typical distribution of the disease to be at the origin of the common iliac arteries.[7,8] Disease progression in the origins of these vessels can lead to severe narrowing, which can ultimately proceed on to vessel occlusion. Occlusion, thus, is often composed of a combination of atherosclerosis and the propagated thrombus within the vessel. The predilection of the plaque for vessel origins with the worst disease at the origins of the common and external iliac arteries has implications for the treatment of these lesions. When the vessel does proceed to complete occlusion, the occlusion usually ends at the first large branch at which retrograde flow can occur, leading to reconstitution of the flow within the arterial system. This process allows the common iliac artery to be an isolated area of occlusion with reconstitution of flow in the external iliac artery via collaterals from the internal iliac artery through its paired vessel in the opposing pelvis.

ASSESSING PATIENTS WITH OCCLUSIVE DISEASE OF THE LOWER EXTREMITIES

The best initial assessment of patients with this problem includes a thorough history and physical exam to determine the symptoms the patient is having as well as any history of progression of the problem. This will allow an evaluation of the length of time the patient has had the process, and can be an indicator of the extent of the disease. It will also be helpful in distinguishing between symptoms based on vascular disease versus those based on neurologic issues. This distinction is critical in assessing how aggressive to be in pursuing noninvasive testing of the lower extremity.

Patients presenting with this type of disease can complain of a wide range of symptoms including those that can be misconstrued as back or neurogenic pain. It is unusual for disease isolated to this area to lead to critical limb ischemia, and the majority of patients present with claudication. Symptoms can originate anywhere from the lower back and buttocks to the feet, and are usually related to increasing activity. The lack of symptoms does not exclude the possibility of disease in this position, and formal testing is necessary to assess the status of the vessels when concern exists regarding the possibility of occlusive disease.

Pulse examination throughout the lower extremity can also be an indication of the presence and location of vascular disease, especially when combined with symptomatic assessment. Simple ankle-brachial index assessment should be performed in the office to delineate the extent of vascular compromise of the lower extremities.

Following a thorough physical evaluation of the pulses, interrogation with more extensive noninvasive testing can be very helpful in determining the presence and location of arterial occlusive disease. The most commonly performed tests include segmental pressures and pulse volume recordings. Values determined by these studies are also indicative of the extent of the disease. Because of the larger size of the iliac vessels, noninvasive testing may be normal at rest, and it may be necessary to do provocative testing such as with treadmill exercise or tourniquet-induced, reactive hyperemia to unmask a clinically significant stenosis (Figures 22-1 and 22-2).

Once the diagnosis of aortoiliac occlusive disease has been made, the decision needs to be made regarding what type of therapy to pursue. Options include conservative management, interventional therapy, and open surgical reconstruction. Given the excellent durability of interventions in this region and limited impact of interventional therapy, this will be an area where the practitioner should feel comfortable offering therapy earlier than he or she might for infrainguinal occlusive disease as a cause of vasculogenic claudication.

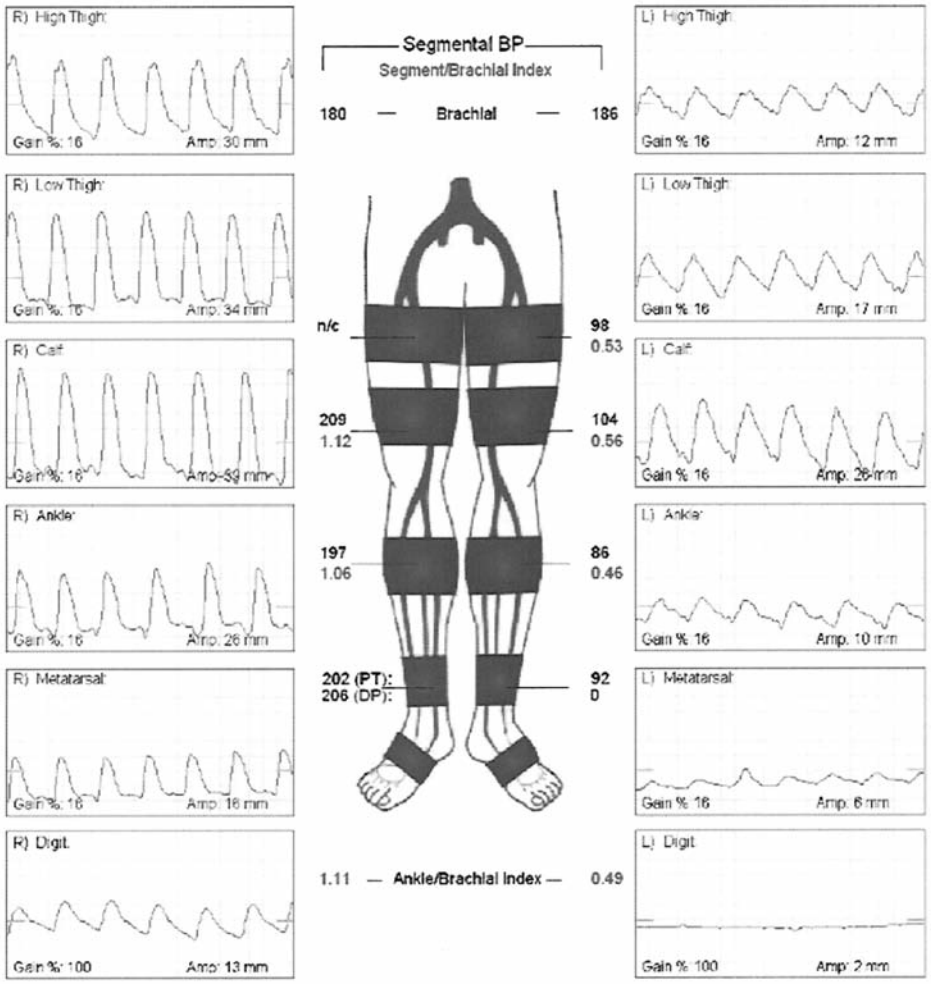

Figure 22-1. Segmental pressure measurements and pulse volume recordings indicative of left iliac occlusive disease.

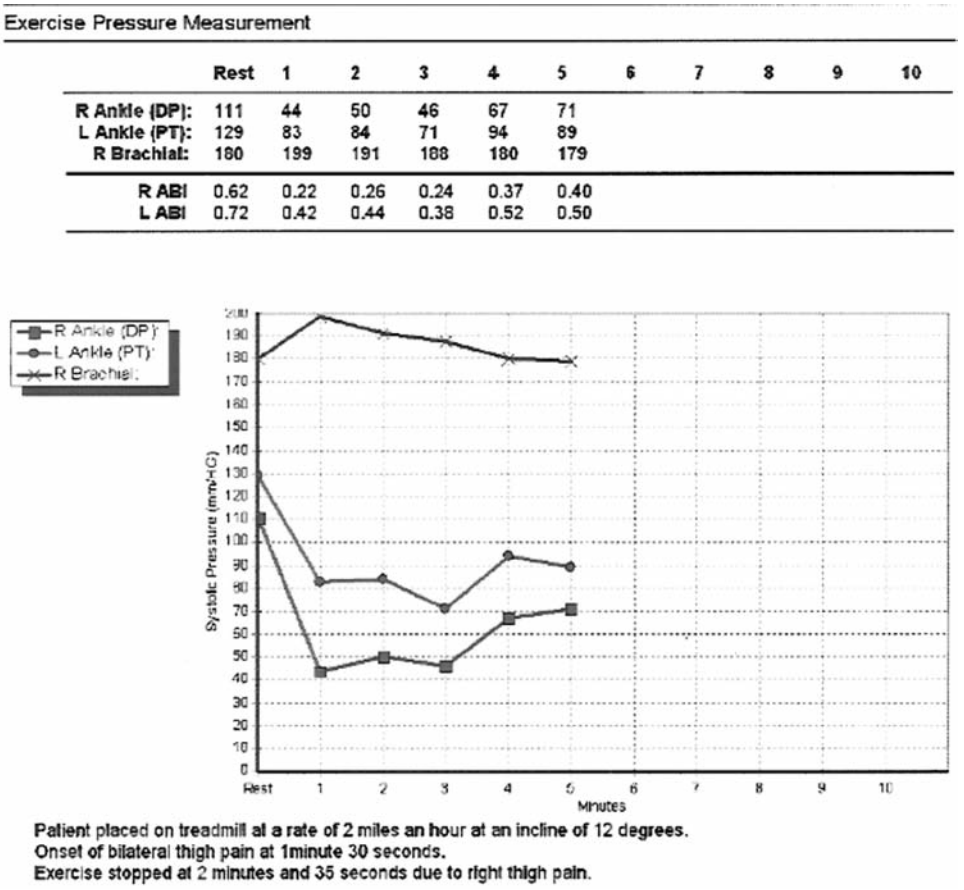

Figure 22-2. Exercise treadmill testing of patient with aortoiliac occlusive disease showing severe drop in lower extremity pressure with activity.

TREATMENT

Lesions of the iliac arteries have typically been treated with surgical reconstruction, the most common form being aortofemoral bypass. Patients undergoing this operation can expect a perioperative mortality of approximately 3%[1-3] and excellent long-term patency rates. Five- and 10-year primary patency rates of these reconstructions can be expected to be 85% and 80% in patients with claudication and 80% and 72% in those with limb-threatening ischemia.[1] Despite these excellent results, it has become apparent over the past several years that treatment of aortoiliac occlusive disease with interventional therapies can also offer excellent results but with markedly lower morbidity.

The best initial evaluation of interventional therapy for the treatment of iliac occlusive disease was performed to assess the results of angioplasty on the iliac arteries.[9] Johnston evaluated 667 balloon angioplasty procedures for iliac occlusive disease. Included within this assessment was a subset of 82 interventions performed to treat occlusion of the iliac arteries. Specifically for these occlusions, there was an 18% early

TABLE 22-1.

Author	Method	Patients (n)	Comp (%)	Success (%)	Patency (1°/2°)
Vorwerk	stent	103	11.6	81	78/88 4yr
Toogood	stent	37	23.4	75	88/na 2yr
Murphy	stent	39	10.3	91	53/82 2.5yr
Dyet	stent	72	12.5	90	85/na 4yr
Johnston	plasty	82	11.0	82	60/na 3yr
Murakami	plasty	54	7.0	57	71/93 10yr
Motarjeme	lysis	99	7.0	86	80/na 4yr

failure rate, with one- and three-year patency rates of 59.8% and 48%, respectively. When primary failures were excluded, the patency rates were 73.2% and 58.5%, respectively. The authors found poorer results when tandem lesions were treated at the same time. While angioplasty alone can be successful for these lesions, the addition of stents for maintenance of patency has provided improved results.[10-14] In the initial evaluation of the Palmaz stent for the treatment of occlusive disease,[10] a subgroup of 79 patients with occlusion was treated. These patients had a 48-month patency of 87.8%, and there was surprisingly a trend toward improved patency of the stent when used in occlusions versus stenoses. Expanding on this initial data with stents, Vorwerk et al.[11] treated 103 patients with chronic occlusions of the iliac artery with self-expanding stainless steel stents. The mean occlusion length was 5.1cm and only nine of the patients had critical limb ischemia. Success in crossing the lesion was achieved in 81% of patients and there were two early thromboses. Primary patency at four years was 78% when initial failures were excluded.

In seven series of iliac artery occlusions treated with interventional therapy[10-16] (Table 22-1), the success rate at crossing iliac occlusive lesions varied from 57% to 91% and primary patency rates were similar to those noted above. It is evident from these series that the treatment of iliac occlusions is feasible and successful, and for this reason, the primary method of therapy for iliac artery occlusion has become primarily interventional with angioplasty and stent placement. While the use of stents may not always be necessary for the treatment of these types of lesions, information from the Dutch Iliac Stent Trial[17] would imply that nearly all of these lesions, when treated with selective stenting, will require stent placement for either inadequate luminal gain or dissection. The technique of performing this procedure can be complex, but relies on principles of iliac artery intervention. Crossing the lesion, confirming luminal re-entry, and treatment are the important points in treating these lesions, but by far the most challenging is the issue of crossing and re-entry. It is important to understand that in the situation where the vessel has been chronically occluded, there is no intraluminal passage of a guide wire. All traversals are functionally subintimal, unless some previous thrombolysis has been performed to try and open a channel as has been recommended by Motarjeme[16] in treating these lesions. Most of the discussion, therefore, involving the treatment of these lesions, will focus on passage that is outside any true lumen as none exists in this setting, and passage back into a lumen will be required before successful treatment can be initiated.

TECHNIQUE OF ILIAC RECANALIZATION

Crossing an iliac lesion can be performed in either an antegrade or retrograde direction. For those individuals who have performed aortoiliac endarterectomy, knowledge of the plaque characteristics at the aortic bifurcation has some bearing on this. Most plaque at the aortic bifurcation is aortic plaque that extends into the branch vessel. The disease tends to be thinner the further one gets from the aortic lumen. This is important because it makes re-entry into the true lumen easier when traveling away from the aortic lumen. This approach also makes it more difficult to begin dissecting into the plaque at the proximal end as the disease tends to be older, and more calcified and thicker at this end. Crossing the occlusion in the opposite direction offers the opposite issues; that is, beginning the passage is much easier, but re-entry can be much more difficult. In some circumstances, subintimal passage can be initiated at both ends and capture of either wire with a snare in the subintimal plane can enable re-entry. In addition, re-entry devices can now be utilized to make the re-entry into the true vessel lumen much easier. In any event, the preferential approach we have utilized most commonly employs the following approach.

The aorta is catheterized from either a contralateral approach (in the setting of presumed unilateral iliac occlusion), or from a brachial approach (in the setting of presumed bilateral iliac occlusion) and aortography is performed in the abdomen and the pelvis. It is important to obtain views of the aorta above the bifurcation to ensure that the aorta itself is not contributing to the blood flow problems. Once the status of the upper abdominal aorta has been clarified, a view of the anatomy in the pelvis is obtained. The standard view obtained will include the aortic bifurcation and the common femoral arteries to their bifurcation. It is important to carry this view out for an extended time to ensure that the refilling of distal vasculature is completely visualized. This ensures that the status of the vessels beyond the occlusion can be assessed. Following this, visualization of the vasculature distal to the occlusion should be performed so one has an understanding of the status of the distal vessels prior to attempts at recanalization. This will ensure that postprocedural abnormalities can be attributed to preexisting disease or to complications of the intervention, either embolization, dissection, or occlusion. In some situations, this can be assessed with computed tomographic angiography (CTA) prior to performing the procedure. Performing this evaluation beforehand also allows an assessment of the extent of the calcification within the occlusion, which can allow the interventionalist some insight into the difficulty to be encountered during the procedure. In certain circumstances, this evaluation will give a better picture of the vessels beyond the occlusion as well, as the calcification of the distal vasculature can be assessed by this imaging technique. Following the assessment of the outflow, the intervention for the occlusion is initiated.

Initiation of a recanalization can often only be performed if the catheter tip can be "buried" in the origin of the occlusion. Attempting to simply advance the wire into the occlusion will often result in the wire bouncing off the occlusion. If attempting recanalization from the contralateral side, a reverse curve catheter is "withdrawn" with the tip aiming into the occlusion and the tip pulled into the origin of the occlusion. A "drilling" technique with a hydrophilic wire is then used to gain access into the origin of the occlusion. With this maneuver, a torque device can be helpful to fix the distance the wire passes out of the catheter. A short distance of the wire is passed out the end of the catheter into the occlusion. The wire is then spun in one direction with a very small amount of force antegrade. The progress of the wire is carefully monitored to

ensure the passage is not retrograde from "bouncing off' the plaque. It is not unusual to have rotation in one direction be much more successful in advancing the wire. During recanalization, the direction necessary to advance the wire may vary, and when one direction is failing to gain adequate advancement, the wire should be rotated in the opposing direction. In the situation where advancement slows considerably with this technique, forceful advancement of the wire can be attempted. If enough purchase within the occlusion has been gained, this will often result in the subintimal passage of the loop. Re-entry into the true lumen is then gained as in standard descriptions of the subintimal technique.[18] If re-entry above the inguinal ligament proves difficult, true lumen re-entry devices such as the Pioneer catheter (Medtronic, Sunnyvale, California) and the Outback catheter (LuMend, Redwood City, California) can allow re-entry into the true lumen of the vessel at a site that would prove amenable to stenting therapy to maintain flow through the recanalized segment into the true lumen distally. Re-entry is better achieved above the inguinal ligament as stenting below this level particularly in the region of the hip joint has poorer results and significant potential for damage to the stent. If entry into the occlusion cannot be gained from the contralateral groin, the interventionalist has two options. The first of these involves access in the brachial position to allow advancement down the descending aorta into the abdominal aorta. A long sheath and forward-facing catheter can then be advanced and "buried" into the occlusion. This will often allow the beginning of wire passage when the contralateral groin approach has been unsuccessful. There are few situations where this approach will prove unsuccessful. The other option is to attempt retrograde recanalization from the ipsilateral groin. This approach makes entry into a subintimal channel much easier, but re-entry into the true lumen of the occlusion more difficult. With the re-entry catheters available however, this approach is also extremely successful in achieving recanalization. Entry into the common femoral artery can be performed either under ultrasound guidance or with fluoroscopic guidance during an aortic injection to localize the common femoral artery. Either of these techniques can be easily mastered and allow quick access in the setting of an absent pulse.

Once access across the occlusion has been gained, angioplasty is performed to allow passage of stents through the occlusion. This dilation is usually performed with an undersized balloon so as to avoid perforation of the vessel and to get a sense of the pain encountered during inflation. The risk of perforation is the greatest risk these patients encounter following recanalization, and this most often occurs in the external iliac arteries.[10,19] Patients at greatest risk in these two reports tend to be those with severe calcification and oversizing of the balloon. Nearly all patients with this complication will complain of pain, and a patient with this complaint during performance of iliac angioplasty should undergo a rapid angiographic assessment of the treated area for possible perforation. The balloon should be left in place during this assessment so that it may be reinflated at a lower pressure to stop the hemorrhage. If the sheath is too small to image around the balloon shaft, then the balloon should be removed. However, it should be left on the wire in case rapid reinsertion proves necessary.

After completing the initial angioplasty, the interventionalist must decide on the appropriate stent for the lesion treated. We tend to favor balloon expandable stents in the common iliac artery and self-expanding stents in the external iliac arteries (Figures 22-3A and 22-3B) There are exceptions to both of these situations, however. Extensive disease throughout the system may make the placement of a self-expanding stent in a long segment of common and external iliac artery appropriate. Additionally, if the lesion in the external iliac artery is confined to the origin of this

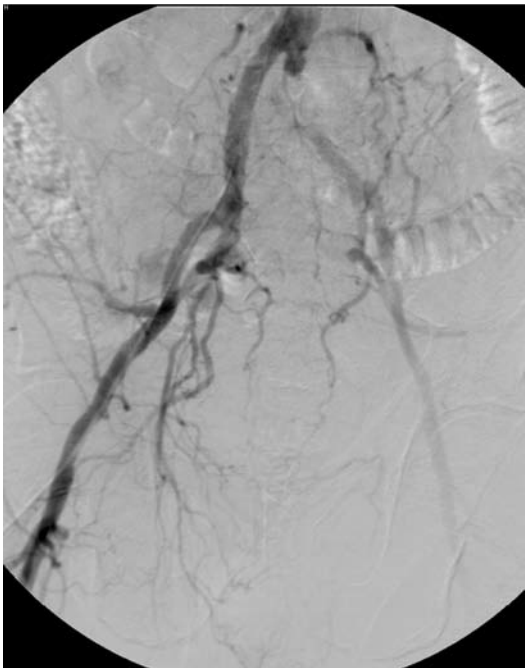

Figure 22-3A. Pelvic angiogram of patient represented in Figure 22-1, with occlusion of left common iliac artery and reconstitution of external and internal iliac arteries via cross-pelvic and retroperitoneal collaterals.

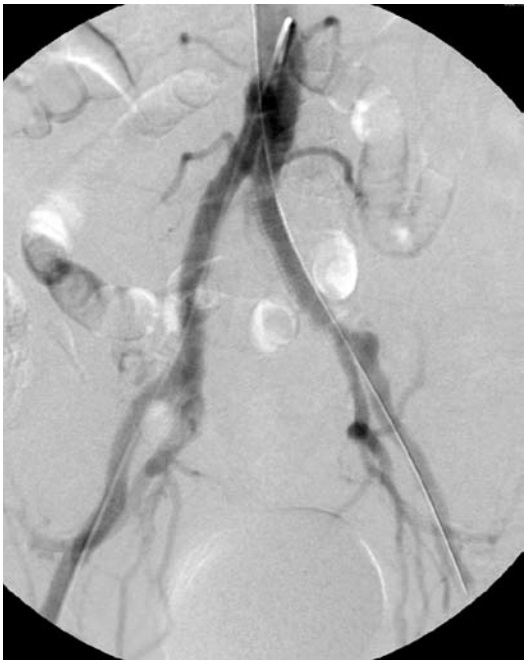

Figure 22-3B. Patient with common iliac occlusion treated with angioplasty and stent.

vessel, balloon-expandable stents have better radial strength and may perform better in this location. There may be a role for covered stents in the treatment of these lesions.[20] However, it remains to be seen whether there is any benefit in routine use of these devices. Results with stenting alone have proven very durable as noted from the previous studies cited, and current treatment recommendations would limit the application of these technologies to specific limited situations (e.g., perforation, eccentric calcific lesions).

OUTCOMES OF INTERVENTION

The outcomes for interventional therapy of occlusion of the iliac arteries has been noted above and outlined in Table 22-1. A more recent reference regarding outcomes in these patients by Scheinert et al.[21] reports a success rate of 90% in achieving passage across occlusions, and treatment of the lesions with 76% primary patency at four years and 85% secondary patency at four years. Once again, while these results are less than what one might expect for surgical reconstruction, they are associated with a markedly lower morbidity and mortality. In addition, patients who fail either primary intervention or reintervention can have surgical reconstruction performed without any evidence that these prior attempts at treatment or interventions affect the surgical options. In evaluating patients for iliac artery occlusion, one needs to be aware of both surgical and interventional options to make the best decision regarding therapy.

REFERENCES

1. deVries SO, Hunink MG. Results of aortic bifurcation grafts for aortoiliac occlusive disease: a meta-analysis. *J Vasc Surg*. 1997; 26:558–569.
2. Poulias GE, Doundoulakis N, Prombonas E, et al. Aorto femoral bypass and determinants of early success and late favourable outcome. Experience with 1000 consecutive cases. *J Cardiovas Surg*. 1992;33:664–678.
3. Brewster DC. Current controversies in the management of aortoiliac occlusive disease. *J Vasc Surg* 1997;25:367–379.
4. Vetto RM. The treatment of unilateral iliac artery obstruction with a transabdominal subcutaneous femorofemoral graft. *Surgery*. 1962;52:342.
5. Blaisdell FW, Hall AD. Axillary femoral artery bypass for lower extremity ischemia. *Surgery*. 1963;54:563.
6. Brewster DC. Direct reconstruction for aortoiliac occlusive disease. In: Rutherford R (ed). *Vascular Surgery 3rd ed*. WB Saunders: Philadelphia; 1989.
7. Haimovici H. Patterns of arteriosclerotic lesions of the lower extremity. *Arch Surg*. 1967;95: 918–933.
8. DeBakey ME, Lawrie GM, Glaeser DH. Patterns of atherosclerosis and their surgical significance. *Ann Surg*.1985; 201:115–131.
9. Johnston, KW. Iliac arteries: reanalysis of results of balloon angioplasty. *Radiology*. 1993;186:207–212.
10. Palmaz JC, Laborde JC, Rivera FJ, et al. Stenting of the iliac arteries with the Palmaz stent: experience from a multicenter trial. *Cardiovasc Interventl Radiol*. 1992;15:291–297.
11. Vorwerk D, Guenther RW, Schurmann K, et al. Primary stent placement for chronic iliac artery occlusions: follow-up results in 103 patients. *Radiology*. 1995;194:745–749.

12. Toogood GJ, Torrie EP, Magee TR, Galland RB. Early experience with stenting for iliac occlusive disease. *Eur J Vasc Endovasc Surg*. 1998;15:165–8.
13. Murphy TP, Webb MS, Lambiase RE, et al. Percutaneous revascularization of complex iliac artery stenoses and occlusions with use of Wallstents: three-year experience. *J Vasc Intervent Radiol*. 1996;7:21–27.
14. Dyet JF, Gaines PA, Nicholson AA, et al. Treatment of chronic iliac artery occlusions by means of percutaneous endovascular stent placement. *J Vasc Interventi Radiol*. 1997;8: 349–353.
15. Murakami R, Korogi Y, Hirai T, et al. Totally occluded iliac arteries: long-term results of percutanoues transluminal angioplasty. *Radat Med*. 1998;16:25–30.
16. Motarjeme A, Gordon GI, Bodenhagen K. Thrombolysis and angioplasty of chronic iliac artery occlusions. *J Vasc Intervent Radiol*. 1995;6:668–728.
17. Tetteroo E, van der Graaf Y, Bosch JL, et al. Randomised comparison of primary stent placement versus primary angioplasty followed by selective stent placement in patients with iliac-artery occlusive disease. Dutch Iliac Stent Trial Study Group. *Lancet*. 1998; 351: 1153–1159.
18. Nadal LL, Cynamon J, Lipsitz EC, Bolia A. Subintimal angioplasty for chronic arterial occlusions. *Tech Vasc Intervent Radiol*. 2004;7:16–22.
19. Allaire E, Melliere D, Poussier B, et al. Iliac artery rupture during balloon dilatation: what treatment? *Ann Surg*. 2003;17:306–314.
20. Marin ML, Veith FJ, Cynamon J, et al. Effect of polytetrafluoroethylene covering of Palmaz stents on the development of intimal hyperplasia in human iliac arteries. *J Vasc Intervent Radiol*. 1996; 7:651–656.
21. Scheinert D, Schroder M, Ludwig J, et al. Stent-supported recanalization of chronic iliac artery occlusions. *Am J Med*. 2001;9:708–715.

23

Angioplasty and Stent in Selected Patients with Femoro-Tibial Lesions

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In 1964, Dotter introduced the concept of percutaneous transluminal angioplasty (PTA).[1] Within five years, he had expanded the notion of angioplasty by including it with the placement of stainless steel coilsprings in the femoral and popliteal arteries of dogs.[2] It was early experiences such as these that led him to postulate that angioplasty and stenting could provide an alternative to conventional open vascular surgery. Over the intervening three decades, tremendous advances have been made in interventional techniques to treat diseased superficial femoral and popliteal arteries.

In 2000, the Transatlantic Inter-Society Consensus (TASC) attempted to create criteria for treatment strategies based on lesion type.[3] More recently, advances in stent design and use of new metal alloys have prompted several investigators to rethink the TASC recommendations. Furthermore, newer treatment modalities such as excimer laser, cryoplasty, atherectomy devices, radiation, and reentry catheters are making previously untreatable lesions now amenable to percutaneous treatment. These advances, coupled with increasing operator experience, are forcing many individuals to reconsider the role endoluminal therapy may have in the treatment of infrainguinal arterial occlusive disease.

ANGIOPLASTY AND STENTING

Angioplasty and stenting have undergone a tremendous evolution over the last decade. With respect to the femoropopliteal segment, there is a large body of evidence correlating angioplasty with preoperative lesion morphology. The TASC criteria outlined which lesions are most amenable to percutaneous intervention and which lesions are ideally suited towards surgical therapy. The TASC recommendations also note that stenting should not be the primary intervention but reserved for situations of PTA complications/failures such as dissection, residual stenosis, or thrombosis.

Metal stents would ideally be nonthrombogenic, inert, radio-opaque, and noncorrodible structures. Unfortunately, they are inherently thrombogenic and generate an inflammatory response after implantation. Yet as the perioperative pharmacotherapy improves (i.e., antiplatelet agents, statins), combined with technical advances such as subintimal angioplasty, operators are pushing the limits with respect to the lesions treated endoluminally. Since Bolia and Bell first performed an accidental subintimal angioplasty in 1987,[4] this technique has gained worldwide acceptance. Utilizing standard techniques in conjunction with subintimal angioplasty and stent, several groups have been able to achieve initial technical success rates >90%. The long-term results have been mixed. Recent studies have shown favorable results, such as Surowiec et al. demonstrating one-, three-, and five-year patency rates of 75%, 60%, and 52%, respectively.[5] Muradin et al. demonstrated patency rates of 66% and 55% at three and five years postprocedure for claudicants treated with angioplasty ± stent.[6] A report of the SCVIR Transluminal Angioplasty and Revascularization Registry (STAR) by Clark et al. in 2001 showed primary patencies of 87% at one year, 69% at three years, and 55% at five years.[7] These results demonstrate improvement over studies performed in the early 1990s, which had one-year patency rates as low as 22%. Yet, they still fail to equal the standard established with surgery when autologous saphenous vein is utilized and where patency rates are routinely demonstrated as >80% at one year and 60% or greater at five years.[8]

Ultimately, many practitioners believe PTA/stenting can act as an initial therapy in patients with lower extremity ischemia, even though surgical bypass offers a more durable outcome at this time. High-volume endovascular centers such as the Leicester group have demonstrated high technical success rates (93%) with low complication rates (<5%) and reduced hospital stays (<36 hour).[9] Furthermore, patients initially undergoing PTA/stent do not lose the option of later bypass if necessary. Currently, there exists a divide between surgeons, radiologists, and cardiologists regarding which treatment is optimal for patients. Due to the lack of well-constructed, prospective, randomized trials, clinical viewpoints are based on practitioner prejudices. The Bypass versus Angioplasty in Severe Ischemia of the Leg trial (BASIL) is undergoing completion, with results expected to be presented in 2006.[10] The aim of the trial is to determine if patients with severe lower extremity ischemia amenable to PTA/stenting or bypass surgery would fair better if PTA +/- stent is employed first versus surgery.

Angioplasty and stenting of infrapopliteal lesions is typically done for critical limb ischemia or limb salvage. Often, the longer lesions of the tibial vessels require subintimal angioplasty with subsequent recanalization. Bolia has noted that it is easier to reenter the true lumen of the tibial vessels due to the thinner intima of these vessels. Overall, results have been mixed regarding the endovascular treatment of these distal lesions. Bolia reported on 28 limbs requiring subintimal angioplasty.[11] In this series, there was an 82% technical success and one-year patency of 53%. However, he did note a one-year limb salvage rate of 85%. Treiman et al. reported similar one-year patency rates (59%) on 25 patients treated with PTA of infrapopliteal lesions.[12] However, this group also noted that patency rates dropped to 20% at three years, and 14 of the 25 patients required an arterial bypass an average of 16 months after the initial angioplasty procedure. One theme that can be discerned from several of the studies looking at infrapopliteal angioplasty is that these procedures can be complementary to surgery, and even in the face of technical failure, conventional surgery remains an option.

NOVEL THERAPIES

Cryoplasty

Several new technologies have evolved in an effort to treat infrainguinal atherosclerotic lesions. Cryoplasty is a treatment in which the artery is cooled to –10°C during angioplasty. This is done to provide a homogenous, less traumatic plaque fracture while also reducing vessel wall recoil. The immediate freezing affects the collagen and elastin fibers, reducing short-term vessel elasticity. Furthermore, the reduction in vessel recoil postplasty could possibly reduce the need for a stent. Finally, the cooling process triggers apoptosis of vascular smooth muscle cells (VSMC). Apoptosis of the VSMC results in a reduction of neointimal hyperplasia and, theoretically, obstructive remodeling and restenosis. FDA approval for this technology was granted in September of 2002. Long-term results for cryoplasty are pending. In the short term, Laird reported on 102 patients undergoing superficial femoral artery and/or popliteal artery cryoplasty in a multicentered registry.[13] Acute procedural success was noted to be 96%, and 87% of those treated needed only cryoplasty. Forty-five of the patients followed out nine months had a clinical patency of 85%. Brambillal et al. presented a series of 129 patients treated for iliac (18%) and superficial femoral artery (82%) lesions.[14] At six months, 65% of the patients treated remained symptom-free.

Brachytherapy

Due to the success of intracoronary brachytherapy, its use in the infrainguinal region has been investigated. The goal of radiation is prevention of restenosis through targeting vascular-associated monocytes and macrophages. There are two types of radiation being investigated: gamma radiation and beta radiation. Gamma radiation has higher penetrating energies than beta radiation, as well as less dose fall-off. Unfortunately, gamma radiation requires modifications to most angiographic suites and other complex steps to prevent unnecessary radiation exposure. Beta radiation has less tissue penetration and a shorter term effect than gamma radiation.

Several studies have looked into brachytherapy. The Vienna-2 trial randomized 113 patients with *de novo* femoropopliteal lesions to either brachytherapy or PTA alone.[15] At one year, restenosis in the PTA-alone group was 61% versus a 36% restenosis rate in the brachytherapy group. The Vienna-5 trial is currently underway, looking at brachytherapy in conjunction with stenting.[16] In contrast to these results, the Peripheral Arterial Radiation Investigational Study (PARIS) failed to reveal an advantage of brachytherapy over PTA in 300 patients treated for *de novo* lesions.[17] There are no large prospective trials demonstrating a clear advantage of brachytherapy for restenotic lesions. Thus, the lack of clear data demonstrating a benefit for brachytherapy, combined with its increased costs and technical and logistical requirements, have limited its clinical applications.

Excimer Laser

Pulsed excimer laser has been studied, utilizing its photoablative effects to recanalize atherosclerotic occlusions. Technically, use of the excimer laser is a slow process, not to exceed a rate of 1 mm every second. Furthermore, the laser is employed only within the atherosclerotic lesion to prevent damage or dissection of the native vessel. Scheinert et al. demonstrated the technical feasibility of this technique when they reported a 90.5%

recanalization rate of occluded superficial femoral arteries in 318 patients with 411 lesions.[18] In the Laser Angioplasty for Critical Limb Ischemia (LACI) trial, 25 limbs in 23 patients were studied, with a technical success rate of 88%, wound healing in 89% of those treated, and a limb-salvage rate of 70% at six months.[19] These results prompted the LACI-2 trial, enrolling 145 patients with 423 lesions in 155 critically ischemic legs.[19] However, this study failed to demonstrate technical success rates or long-term patency rates higher than those seen in historic PTA data. The Peripheral Excimer Laser Angioplasty (PELA) study randomized 251 claudicants with superficial femoral artery occlusions into PTA or excimer laser-assisted groups.[20] This study failed to demonstrate a difference between initial technical success (85% laser versus 91% PTA), complications (12.8% laser versus 11.4% PTA), or 12-month patency (49% versus 49%). Currently, there is no data demonstrating an advantage of laser angioplasty over conventional angioplasty.

Atherectomy/Thrombectomy Devices

Another method for dealing with chronic atherosclerotic occlusions of the infrainguinal vessels are percutaneous atherectomy/thrombectomy devices. These devices combine thrombus fragmentation with extraction. The greatest concerns with these devices are vessel perforation and peripheral embolization of fragmented debris. In an early paper, Kim et al. demonstrated a 92% technical success rate in 85 lesions with no major complications at six months.[21] More recently, Zeller et al. reported on 98 patients treated for thrombotic occlusions.[22] This group noted a 93% primary success rate with an 88% 30-day limb-salvage rate for subacute/chronic thrombosis and a 100% 30-day limb-salvage rate in patients with an acute thrombosis. Currently, the only FDA-approved device in the United States is the SilverHawk Plaque Excision Device (Fox Hollow Technologies, Redwood City, CA). The data regarding interventions performed with this device are being collected in the TALON Registry, which had six-month results reported at the 2004 Transcatheter Therapeutics meeting.[23] Here, a six-month clinical patency rate of 89% was observed for 220 patients with 442 lesions. Furthermore, the overall complication rate was low with a minor complication rate of 0.7% and a major complication rate of 0%.

Drug-eluting Stents/Biodegradable Stents

An area of great interest is drug-eluting stents and biodegradable stents. Drug-eluting stents have been used extensively in coronary artery interventions; however, the successes seen in coronary interventions have not been duplicated in the periphery. The theory behind drug-eluting stents is that the pharmacokinetic agent will lead to cell cycle arrest, blocking smooth muscle cell migration and proliferation, and thus halting neointimal hyperplasia. Furthermore, the drug typically will have an anti-inflammatory effect. There have been two randomized, prospective trials published: the SIROlimus Coated Cordis S.M.A.R.T. Nitinol Self-expandable Stent for the Treatment of Obstructive Superficial Femoral Artery Disease (SIRROCCO) trials, I and II. SIRROCCO I randomized 36 patients, and although it demonstrated that controlled drug release is feasible using a self-expandable nitinol stent platform, it failed to demonstrate a significant benefit over uncoated stents.[24] The SIRROCCO II trial randomized 57 patients and it also failed to show a significant benefit in the sirolimus-eluting stent group.[25] Although these studies failed to reproduce the successes seen with drug-eluting stents in the coronary arteries, they did provide valuable data regarding the safety and feasibility of drug-eluting stents in the periphery. A long-term concern about stents within the femoropopliteal segment is the risk

of stent fracture, observed in roughly a quarter of patients within one year of stent implantation in the forementioned trials, and the potential for distal embolization of stent fragments, which has been anecdotally noted. Biodegradable stents are even further away from clinical usage than peripheral drug-eluting stents. These stents are designed to provide the immediate benefit of a stent, yet be nonexistent after the neointimal has formed postangioplasty, prior to the onset of neointimal hyperplasia. No human data have been published; however animal studies using absorbable magnesium stents have demonstrated a decrease in intimal hyperplasia with near stent resorption at 60 days.[26]

CONCLUSION

Historically, the superficial femoral artery and the popliteal artery have been best treated with an open, operative approach. This is due to the extensive nature of the disease, the complex biophysical forces exerted on these vessels, and the fact that occlusion predominates over stenosis. However, recent advancements have been made in treating long-segment femoropopliteal disease. Improved catheter skills and techniques such as subintimal angioplasty, coupled with a strong push in newer technologies, have led to renewed optimism within several subspecialties regarding the treatment of infrainguinal arterial disease. Furthermore, the current results obtained by experienced practitioners have led many to rethink the existing paradigm regarding disease in this vascular bed. Often, patients will be referred for an endovascular option prior to undertaking an open repair due to the less invasive nature combined with the overall low morbidity and mortality. Currently, the pace of interventions is exceeding the evidence for long-term success. Time will tell whether the current strategies are visionary or foolhardy.

REFERENCES

1. Dotter CT, Judkins MP. Transluminal treatment of arteriosclerotic obstruction. Description of a new technic and preliminary report of its application. *Circulation*. 1964;30:654–670.
2. Dotter CT. Transluminally-placed coilspring endarterial tube grafts. Long-term patency in canine popliteal artery. *Invest Radiol*. 1969;4:329–332.
3. Dormandy JA, Rutherford RB. Management of peripheral arterial disease (PAD). TASC Working Group. Transatlantic Inter-Society Consensus (TASC). *J Vasc Surg*. 2000;31:S1–S296.
4. Bolia A, Brennan J, Bell PR. Recanalisation of femoro-popliteal occlusions: improving success rate by subintimal recanalisation. *Clin Radiol*. 1989;40:325.
5. Surowiec SM, Davies MG, Eberly SW, et al. Percutaneous angioplasty and stenting of the superficial femoral artery. *J Vasc Surg*. 2005;41:269–278.
6. Muradin GSR, Bosch JL, Stijnen T, Hunink MG. Balloon dilatation and stent implantation for treatment of femoropopliteal arterial disease: meta-analysis. *Radiology*. 2001;221:137–145.
7. Clark TW, Groffsky JL, Soulen MC. Predictors of long-term patency after femoropopliteal angioplasty: results from the STAR registry. *J Vasc Interv Radiol*. 2001;12:923–933.
8. Klinkert P, Post PN, Breslau PJ, van Bockel JH. Saphenous vein versus PTFE for above-knee femoropopliteal bypass. A review of the literature. *Eur J Vasc Endovasc Surg*. 2004;27:357–362.
9. Axisa B, Fishwick G, Bolia A, et al. Complications following peripheral angioplasty. *Ann R Coll Surg Engl*. 2002;84:39–42.
10. Bradbury AW, Bell J, Lee AJ, et al. Bypass or angioplasty for severe limb ischemia? A Delphi consensus study. *Eur J Vasc Endovasc Surg*. 2002;24:411–416.

11. Bolia A. Percutaneous intentional extraluminal (subintimal) recanalization of crural arteries. *Eur J Radiol*. 1998;28:199–204.
12. Treiman GS, Whiting JH, Treiman RL, et al. Treatment of limb-threatening ischemia with percutaneous intentional extraluminal recanalization: a preliminary evaluation. *J Vasc Surg*. 2003;38:29–35.
13. Laird J, Biamino G, Jaff M. Femoropopliteal outcomes with cryoplasty: final results of the cryovascular safety registry. *J Vasc Intervent Radiol*. 2004;15:S218.
14. Brambillal N, Laudisa ML, Llambro, et al. Cryoplasty in the treatment of iliac and superficial femoral disease. *J Cardiovasc Surg*. 2004;45:S1–3:12.
15. Pokrajac B, Potter R, Maca T, et al. Intraarterial (192) Ir high-dose-rate brachytherapy for prophylaxis of restenosis after femoropopliteal percutaneous transluminal angioplasty: the prospective randomized Vienna-2-trial radiotherapy parameter and risk factor analysis. Int *J Radiat Oncol Biol Phys*. 2000;48:923–931.
16. Pokrajac B, Wolfram R, Lileg B, Minar E. Endovascular brachytherapy in peripheral arteries: solution for restenosis or false hope? *Curr Treatment Options Cardiovasc Med*. 2003;5: 121–126.
17. Waksman R. An update on peripheral brachytherapy. *Endovasc Today*. 2004;4(9):43–52.
18. Scheinert D, Laird JR, Schroder M, et al. Excimer laser-assisted recanalization of long, chronic superficial femoral artery occlusions. *J Endovasc Ther*. 2001;8:156–166.
19. Laird JR Jr, Reiser C, Biamino G, Zeller T. Excimer laser assisted angioplasty for the treatment of critical limb ischemia. *J Cardiovasc Surg (Torino)*. 2004;45:239–248.
20. Laird JR. Peripheral Excimer Laser angioplasty (PELA) trial results. Transcatheter Cardiovascular Therapeutics Conference. Washington, DC, USA. 2002 September 24–28, 2002.
21. Kim D, Gianturco LE, Porter DH, et al. Peripheral directional atherectomy: 4-year experience. *Radiology*. 1992;183:773–778.
22. Zeller T, Frank U, Burgelin K, et al. Early experience with a rotational thrombectomy device for the treatment of acute and subacute infra-aortic arterial occlusions. *J Endovasc Ther*. 2003; 10:322–331.
23. Gammon R, Fail P, Walker C, et al. Early results from the treating peripherals with Silverhawk: Outcomes collection (TALON) registry. Abstract Presented at the Transcatheter Cardiovascular Therapeutics Meeting (TCT). Washington, D.C. September 27-October 1, 2004.
24. Duda SH, Pusich B, Richter G, et al. Sirolimus-eluting stents for the treatment of obstructive superficial femoral artery disease: 6-month results. *Circulation*. 2002; 106:1505–1509.
25. Duda SH, Bosiers M, Lammer J, et al. Sirolimus-eluting stents versus bare nitinol stent for obstructive superficial femoral artery disease: the SIROCCO II trial. *J Vasc Intervent Radiol*. 2005;16:331–338.
26. Di Mario C, Griffiths H, Goktekin O, et al. Drug-eluting bioabsorbable magnesium stent. *J Intervent Cardiol*. 2004;17:391–395.

24

Angioplasty and Stenting for Infrainguinal Lesions

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The purpose of this chapter is to review the current technique and results of balloon angioplasty and stent placement for infrainguinal arterial occlusive disease. Although the results of balloon angioplasty and stents in this vascular bed have been mixed over the years, the endovascular option is well integrated into our treatment pathways for infrainguinal occlusive disease in a full service vascular practice. A broad survey of the field suggests that the management of infrainguinal occlusive disease continues to evolve away from open surgery and toward more percutaneous options. Infrainguinal endovascular intervention and its growing cadre of associated options have a high likelihood of improving the care of vascular patients in the years to come.

CLASSIFICATION OF INFRAINGUINAL OCCLUSIVE DISEASE

Infrainguinal occlusive disease may be classified by its morphology in a manner that assists in determining which patients are best managed with endovascular intervention and which require surgery. The general concept holds that endovascular approaches are best in patients with less severe forms of disease (short stenosis versus long or multilevel occlusion) and more medical comorbid conditions (shorter life expectancy, higher surgical risk). Infrainguinal bypass has a better risk/benefit ratio in patients with fewer medical problems (longer life expectancy) and/or more severe forms of disease (where endovascular procedures are not as durable). The TASC Classification and others have defined disease morphology of different levels in an effort to clarify the issue of lesion severity.[1] The TASC Classification is summarized in Table 24–1. The recommendation from the TASC group was that TASC A lesions be treated with endovascular intervention, TASC D lesions be treated with surgery and that TASC B and C lesions be treated with either, at the operator's discretion, pending further evaluation of these lesions.

TABLE 24-1. TASC CLASSIFICATION OF FEMORAL-POPLITEAL LESIONS

TASC A Lesions
Single stenosis < 3 cm
TASC B Lesions
Single stenosis 3 to 10 cm in length, not involving the distal popliteal artery
Heavily calcified stenosis up to 3 cm
Multiple lesions, each less than 3 cm (stenosis or occlusion)
Single or multiple lesions in the absence of tibial runoff to improve inflow for distal surgical bypass
TASC C Lesions
Single stenosis or occlusion > 5 cm in length
Multiple stenoses or occlusions, each 3 to 5 cm
TASC D Lesions
Occlusion of the common femoral artery, popliteal artery, proximal trifurcation arteries. Occlusion of the superficial femoral artery > 10 cm in length

RESULTS OF FEMORAL-POPLITEAL BALLOON ANGIOPLASTY

Data have accumulated over a period of 20 years that provide an understanding of femoral-popliteal balloon angioplasty. In a summary of studies comprised of 1,241 patients, the results (weighted averages) for femoral-popliteal balloon angioplasty were as follows; 90% technical success rate, 4.3% complication rate, 1-year patency of 61%, and 5-year patency of 48%.[1] In a review of several large studies published in the early to mid-1990s, prior to the broad availability and usage of stents, patency rates ranged from 47% to 63% at 1 year and 26% to 48% at 5 years.[2] Multiple factors affect the results of femoral-popliteal PTA (percutaneous transluminal balloon angioplasty), including; lesion length, clinical stage (claudication versus limb salvage), runoff, and lesion type (stenosis or occlusion), proximal location, and lack of residual stenosis after PTA.[3-5]

Examples of how some of these factors affect the results of femoral-popliteal PTA are summarized in Tables 24–2 and 24–3. Meta-analyses have demonstrated the impact of lesion type (stenosis versus occlusion) and clinical stage (claudication versus limb salvage; Table 24–2).[6,7] Lesion length is not addressed in as many studies and

TABLE 24-2. META-ANALYSES OF FEMORAL-POPLITEAL BALLOON ANGIOPLASTY: THE EFFECT OF LESION TYPE AND CLINICAL STAGE

				Primary Patency		
Reference	**Lesion type**	**Clinical stage**	**Limbs**	**1 year**	**3 years**	**5 years**
Hunink (6)	Stenosis	Claudication	4,800	79	74	68
	Stenosis	Limb threat		62	54	47
	Occlusion	Claudication		52	43	35
	Occlusion	Limb threat		26	18	12
Muradin (7)	Stenosis	Claudication	923		61	
	Stenosis	Limb threat			43	
	Occlusion	Claudication			48	
	Occlusion	Limb threat			30	

TABLE 24-3. LENGTH OF THE LESION AFFECTS THE RESULTS OF FEMORAL-POPLITEAL BALLOON ANGIOPLASTY

Reference	Lesion Length/Patency	Lesion Length/Patency
Murray (8)	< 7 cm 81% at 6 months	> 7 cm 23%
Currie (9)	< 5 cm 59% at 6 months	> 5 cm 4%
Jeans (10)	< 1 cm 76% at 5 years	> 1 cm 50%
Krepel (11)	< 2 cm 77% at 5 years	> 2 cm 54%

length classifications have not been standardized but it appears that length also has a significant impact upon results (Table 24–3).[8-11]

The patency of PTA of a short femoral-popliteal lesion under favorable circumstances is 70% to 80% at 1 year and 50 to 60% at 5 years. This type of lesion is ideally suited to PTA. Endovascular intervention is cost effective in comparison to surgery in this setting and is the treatment of choice.[12] Unfortunately, most situations in which femoral-popliteal balloon angioplasty might be considered are more complex and factors are not as favorable. In addition, balloon angioplasty is no longer a stand alone procedure since stent placement has reached clinical utility in the practice of endovascular surgery in the infrainguinal arteries.

RESULTS OF FEMORAL-POPLITEAL STENT PLACEMENT

Over the past 5 years, stent placement has become integrated into infrainguinal intervention. This evolution has been prompted by the development of a variety of simple, low profile, user friendly, self-expanding stents in many varieties that can be easily and simply placed when needed or when the immediate results of PTA are not satisfactory.

There is no evidence that routine or primary stent placement improves long-term results. Several randomized trials, mostly using balloon expandable stents, have shown no significant difference at 1 to 4 years after intervention when primary and selective stent placement were compared (Table 24–4).[13-16] Nevertheless, stents have had an impact. The immediate success of the intervention is higher with the availability of stents. About 15% of patients undergoing PTA alone require selective stent placement or experience immediate failure.[14,16] The least favorable results of PTA come from treatment of long lesions, occlusions, residual stenoses and patients with limb threatening ischemia. Stents are an essential tool if endovascular intervention is to be an option in treating these complex lesions and unfavorable clinical situations. A meta-analysis of 423 stent

TABLE 24-4. RANDOMIZED TRIALS COMPARING PRIMARY VERSUS SELECTIVE STENT PLACEMENT FOR FEMORAL-POPLITEAL OCCLUSIVE DISEASE

		Patency After Stent Placement		
Reference	Year	Primary	Selective	Follow-up
Vroegindeweij (13)	1997	74%	85%	1 year
Cejna (14)	2001	65%	65%	2 years
Grimm (15)	2001	62%	68%	3 years
Becquemin (16)	2003	44%	57%	4 years

implantations for femoral-popliteal occlusive disease demonstrated a 66% patency at 3 years which was not dependent upon clinical indication or lesion type.[7] Stents have also yielded promising results with long, chronic occlusions.[17]

Although primary stent placement is not warranted, selective stent placement plays an important role. The day to day reality is that PTA is being performed on a broad array of infrainguinal lesions, partially because the availability of stents makes endovascular intervention a more reasonable option and less likely to cause an ischemic emergency. In addition, the likelihood is very high that these devices will continue to improve. Recent data suggests that nitinol stents may improve results (70 to 80% primary patency at 3 years).[18,19] Stents that deliver medications or are covered with graft material to prevent intimal hyperplasia are also likely to become clinically useful within the next couple of years.[20,21]

RESULTS OF ENDOVASCULAR INTERVENTION FOR INFRAPOPLITEAL LESIONS

The patency of infrapopliteal balloon angioplasty is not as well established as for more proximal lesions. Most of the patients have limb threatening ischemia, multilevel disease and multiple or diffuse tibial lesions that require treatment. Results have been assessed most often by evaluating limb salvage rather than patency. A meta-analysis of 1,282 treated limbs demonstrated a technical success rate of 93% and a 2-year limb salvage rate of 74%.[22] Stent placement is technically feasible using low profile, balloon expandable, coronary stents on a 0.014 platform. Although this approach may help to salvage an unsuccessful tibial balloon angioplasty, data is insufficient to conclude whether stents are of any significant value in the infrapopliteal arteries.

EVALUATION OF THE PATIENT

Infrainguinal occlusive can usually be diagnosed by history and physical exam. Confirmatory studies may be performed, either duplex mapping or magnetic resonance arteriography. "Diagnostic arteriography" does not exist in many practices today. Most patients in our practice do not undergo arterial access unless there is an intention to treat. Occasionally, what appeared to be a lesion appropriate for angioplasty by duplex or MRA is more complex than advertised and only an arteriogram is performed. This is most likely to occur with tibial lesions in our practice. These factors are important in determining the best approach to access for intervention.

TECHNIQUES FOR INFRAINGUINAL BALLOON ANGIOPLASTY AND STENT PLACEMENT

Approach: Balloon angioplasty and stent placement of the infrainguinal arteries is usually performed through the contralateral femoral artery using an up and over approach or the ipsilateral femoral artery using an antegrade approach (Table 24–5). Infrainguinal interventions may also be performed through the brachial artery but this approach is rarely

TABLE 24-5. APPROACHES TO INFRAINGUINAL INTERVENTIONS: IPSILATERAL APPROACH VERSUS UP AND OVER APPROACH FROM CONTRALATERAL FEMORAL

	Up-and-over approach	Antegrade approach
Puncture	Simple retrograde femoral	More challenging, less working room
Catheterization	Up-and-over catheterization is challenging with tortuous arteries, narrow, or diseased aortic bifurcation; easier to catheterize SFA when going up and over	Entering SFA from antegrade approach requires proximal femoral puncture and selective catheter
Guidewire/catheter control	Fair	Excellent
Catheter inventory	Need more supplies	Minimal, shorter catheters
Specialty items	Up-and-over sheath, long balloon catheters	None
Indications	Proximal SFA disease, CFA disease ipsilateral to infrainguinal lesion, obesity	Intrapopliteal disease, patients with contraindication to up-and-over approach

SFA, superficial femoral artery; CFA, common femoral artery.
From: Schneider PA. The infrainguinal arteries—advice about balloon angioplasty and stent placement. In: *Endovascular Skills,* 2nd Edition, Marcel Dekker, NY, 2003; pp. 316.

required and may be more challenging due to the longer distances involved. The primary advantages of the up and over approach, which is most commonly used, are the following: an aortogram with runoff may be easily converted to endovascular therapy; it permits evaluation of the inflow aortoiliac arteries prior to treatment of infrainguinal lesions; only a simple retrograde femoral puncture is required; the up and over approach facilitates selective catheterization of the superficial femoral artery orifice; and, puncture site management is contralateral to the intervention site, rather than proximal to it. The antegrade approach is not required as often but may be used for better guidewire and catheter control in infrapopliteal intervention and also in patients who have contraindications to the up and over approach. The likely approach is determined prior to the procedure to facilitate room set-up and the availability of supplies. Both groins are always prepared in case an alternative approach is required during the procedure.

Which Platform Should Be Used; 0.035 in., 0.018 in. or 0.014 in.? Most balloon angioplasty and stent placement of the infrainguinal arteries can be performed using the standard 0.035 in. platform. The standard platform includes; 0.035 in. diameter guidewires, 4 and 5 Fr flush and selective catheters, 5 Fr balloon angioplasty catheters, and self-expanding stent delivery catheters that are 6 Fr. The access sheath is usually 5 Fr for antegrade balloon angioplasty and 5.5 Fr for up and over balloon angioplasty. Stent placement requires a 6 Fr sheath. The advantages of the standard platform are the following: the guidewires and catheters are easy to handle; the inventory is usually readily available; the fluoroscopic visualization of these larger caliber devices is simpler; long balloons are available (up to 10 cm.) to treat longer superficial femoral artery lesions; and the larger guidewires and catheters are useful if an occlusion must be crossed or subintimal angioplasty is required. However, there are some significant disadvantages. The larger caliber guidewires and catheters may not easily cross critically diseased segments, at longer distances these catheters lose their pushability due to high friction, and in small arteries such as tibial vessels the standard platform devices may be too big. Small platform devices may be used with a 0.018 or 0.014 in. system. Most of the coronary devices are on a 0.14 in. platform so the array of balloon catheters and stents is much broader with this system. The balloon catheters are 3 Fr and can be placed through a 4 Fr sheath. Monorail or rapid exchange balloon catheters permit

better pushability since the friction of the guidewire on the balloon catheter lumen is over a much shorter distance than with coaxial balloon catheters. Self-expanding stents are available that have monorail delivery and may be placed through a 5 Fr sheath up to 8 mm diameter, and a 6 Fr sheath for larger diameter stents. Balloon expandable coronary stents may be place in the tibial arteries through a 5 Fr sheath. In addition, if a 0.014 in. guidewire is used, devices that accommodate a 0.018 in. guidewire will also be 0.014 in. guidewire compatible.

Up and over approach: Supplies required for an up and over approach are listed in Table 24–6. This approach requires longer guidewires, catheters and sheaths than the antegrade approach. A standard retrograde common femoral artery puncture is performed contralateral to the symptomatic side. A floppy tipped guidewire is passed into the aorta. A hook-shaped, multisidehole flush catheter, such as a 65 cm 4 Fr Omni-flush catheter (AngioDynamics, Inc), is passed into the aorta and an aortoiliac arteriogram is performed. If bilateral runoff is required, it may be performed at that time with the catheter head placed in the infrarenal aorta. When only unilateral runoff on the symptomatic side is indicated, the catheter is passed over the aortic bifurcation and lower extremity arteriography is performed. After the infrainguinal lesions have been evaluated, the aortic bifurcation appears suitable for accommodation of an access sheath, and it is determined that an up and over approach is best, an up and over sheath is placed (Figure 24–1).

The aortic flush catheter is withdrawn to the aortic bifurcation and its tip is rotated toward the contralateral side to direct the guidewire into the contralateral iliac artery. The advancing guidewire, usually a steerable, angled-tip Glidewire (Medi-Tech), must

TABLE 24-6. SUPPLIES FOR UP AND OVER APPROACH TO INFRAINGUINAL INTERVENTION

Guidewire	Starting	Bentson	145 cm, lenght	0.035 in. diameter
	Selective	Glidewire	150 cm	0.035 in. (steerable)
		Glidewire	260 cm	0.035 in. (steerable)
	Exchange	Rosen	180 cm	0.035 in (J tip)
		Amplatz Super-Stiff	180 cm	0.035 in.
Catheter	Flush/selective	Omni-flush	65 cm	4 Fr
	Exchange	Straight	90 cm	5 Fr
Sheath	Selective sheath	Up and over	40 cm	5.5 Fr, 6 Fr, 7 Fr
Balloon	Balloon angioplasty catheters	Balloon diameter	2, 3, 4, 5, 6 mm	
		Balloon length	2, 4 cm	
		Catheter shaft	75, 90, 110 cm[a]	
Stent	Self-expanding	Wallstent		
		Stent diameter	6, 8 mm	
		Stent length	20, 40, 45, 60 mm	
		SMART		
		Stent diameter	6, 8 mm	
		Stent length	20, 40, 60, 80 mm	
		Delivery catheter length	120 cm	

[a]A 75-cm catheter shaft for balloon angioplasty to mid-SFA. Longer catheters are required for contralateral approach to distal SFA, popliteal, and tibial intervention.

From: Schneider PA. The infrainguinal arteries—advice about balloon angioplasty and stent placement. In: *Endovascular Skills,* 2nd Edition, Marcel Dekker, NY, 2003; pp. 322.

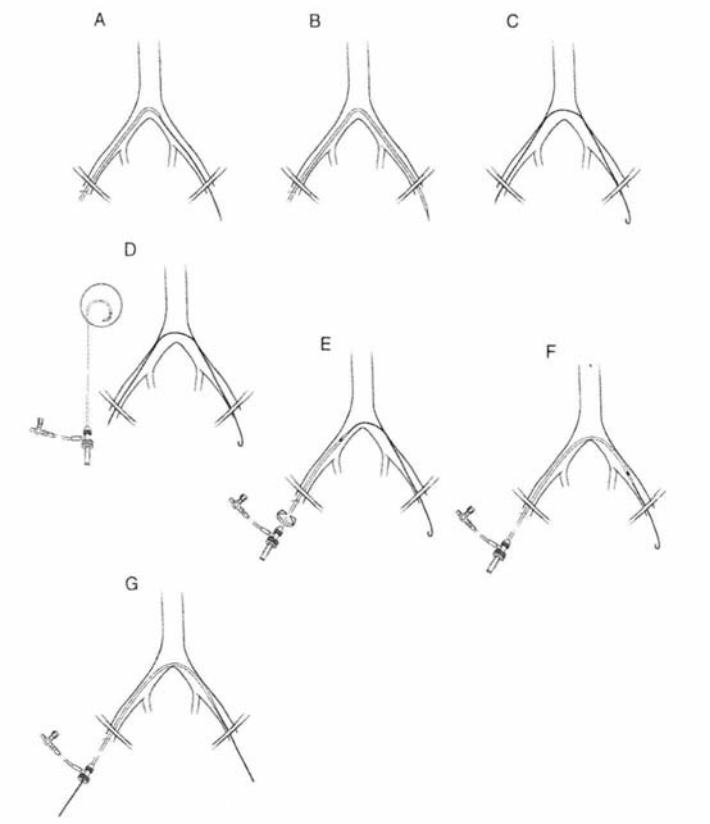

Figure 24-1. Placement of an up and over sheath. A: A guidewire and catheter are passed over the aortic bifurcation. B: The catheter is advanced to the contralateral femoral artery. C: An exchange guidewire is placed and the catheter is removed. D: The sheath is oriented with its tip pointing toward the contralateral side. E: The sheath is advanced over the guidewire. This is visualized using fluoroscopy. F: The sheath is advanced to its hub. Be sure there is enough guidewire ahead of the sheath tip to permit a smooth advance. G: The dilator is removed and the sheath is ready to use. From: Schneider PA. Access for endovascular therapy. In: *Endovascular Skills*, 2nd Edition, New York; Marcel Dekker; 2003:195.

be steered into the external iliac artery and then into the infrainguinal arteries. From this approach, the guidewire usually tends to select the contralateral internal iliac artery if there is tortuosity of the iliac system. It also tends to select the superficial femoral artery (SFA) rather than the profunda femoral artery (PFA). Either of these destinations for the guidewire is satisfactory form the standpoint of sheath placement as long as the guidewire is well anchored distal to the groin. The catheter is advanced over the bifurcation and an exchange guidewire is placed. The tip of the exchange guidewire should be distal to the groin as far as it will easily travel. If there is a proximal SFA lesion that is planned for treatment, the guidewire is usually directed into the PFA. A 180 cm length, 0.035 in. Rosen guidewire is usually adequate for sheath placement. If there is a lot of tortuosity in the iliac system, an Amplatz super-stiff guidewire may be required.

Dilators are used to enlarge the arteriotomy. Since dilators are sized by their outside diameter and sheaths are sized by their inside diameter, if a 6 Fr sheath is planned for placement, the track should be dilated using a 7 Fr dilator. The up and over sheath

(Cook, Inc) is placed on the guidewire in the appropriate orientation, with the curved end of the sheath pointing toward the contralateral side and the sidearm of the sheath on the side of the operator. The sheath is advanced over the guidewire using fluoroscopy. Passage over a narrow or diseased aortic bifurcation is performed with care and patience. The sheath is advanced to its hub if possible. Remember that the tip of the dilator extends beyond the radiopaque marker on the end of the sheath tip for a short distance. The tip of the sheath will end up somewhere between the mid-external iliac artery and the very proximal SFA, depending upon the height of the patient. Heparin is usually administered (50 to 75 u/kg) as the sheath is placed.

Figure 24–2 demonstrates the steps required for infrainguinal balloon angioplasty using an up and over approach. The exchange guidewire is replaced with a steerable Glidewire, usually 260 cm length. Through the sidearm of the sheath, the diseased infrainguinal segment is roadmapped and the Glidewire is used to cross the lesion intended for treatment. If treatment of tibial lesions is planned, a 4 or 5 Fr, 100 cm length catheter is advanced into the distal popliteal artery and roadmapping is performed through this catheter and a low profile guidewire, usually 0.014 in., is used to cross the tibial lesions. An angled glide cath (Medi-Tech) may be used to direct the guidewire across the lesion. Interval arteriography may be performed through the side arm of the sheath or through the selective catheter using a Tuohy-Borst adaptor.

The balloon catheter is selected to treat the lesion. Most superficial femoral and popliteal artery lesions will be treated with a 5 or 6 mm balloon. Occasionally, a 4 mm

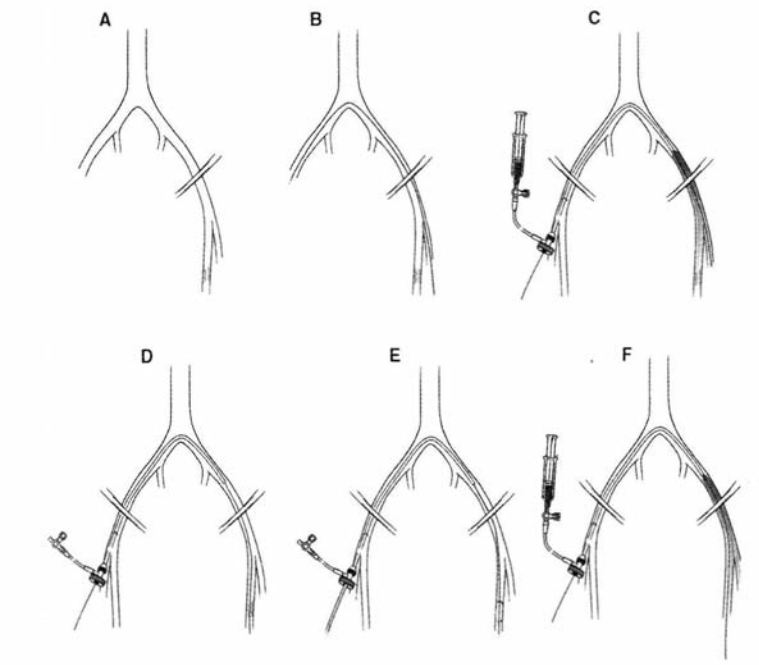

Figure 24-2. Balloon angioplasty of the femoral and popliteal arteries through an up and over approach. A: A superficial femoral artery lesion is identified. B: A guidewire is introduced through the contralateral femoral artery and passed over the aortic bifurcation. C: An up and over sheath is placed and arteriography is performed. D: The guidewire crosses the lesion. E: Balloon angioplasty is performed. F: Completion arteriography is performed through the sheath. From: Schneider PA. The infrainguinal arteries—advice about balloon angioplasty and stent placement. In: *Endovascular Skills*, 2nd Edition, New York; Marcel Dekker; 2003:323.

or a 7 mm balloon is required. Tibial arteries range from 1 to 4 mm but most are between 2 and 3.5 mm. Standard platform balloons are most commonly delivered in 4 cm lengths but may also be obtained in 2, 6, 8 or 10 cm lengths. Longer lesions may be treated faster with the use of a longer balloon to minimize the number of inflations. Small platform balloons are usually 2 cm long but 4 cm balloons may also be obtained. Small platform balloons may be non-compliant, like the larger platform balloons, or compliant. An example of a compliant balloon would be a 4 mm balloon with a nominal pressure of 8 atm. At 4 atm the balloon diameter may be 3.6 mm but at 14 atm it may be 4.3 mm. Catheter length must be anticipated prior to selecting the balloon. Most of the standard platform balloons are on shafts that are either 75 to 80 cm or 120 to 130 cm, depending upon the manufacturer. A 75 cm balloon catheter shaft passed up and over will reach anywhere from the common femoral artery to the distal superficial femoral artery, depending upon the height of the patient. And estimation of distance to the lesion may be obtained with the use of a 75 cm length straight exchange catheter for guidewire exchanges. The up and over sheath also provides clues since it is 40 cm in length.

The balloon angioplasty catheter is passed over the guidewire and into the lesion. The location of the lesion may be marked using roadmapping or an external marker. Balloon angioplasty is performed by inflating the balloon until the waist on the balloon profile is resolved. Balloon inflation may be performed anywhere from a few seconds to several minutes. Inflation pressure may be as low as 3 or 4 atm to open the waist or may be as high as 15 to 20 atm. The completion arteriogram is performed through the side arm of the sheath. Balloon angioplasty of the superficial femoral and popliteal arteries almost always produces some evidence of dissection on completion arteriography. In this setting, deciding which patients require a stent may be challenging. In the pre-stent era, most post-PTA dissections healed. Since primary stent placement has not proven to be of value in enhancing durability, stents should be placed selectively. This issue is discussed in greater detail below in the section about stents.

The completion arteriogram is assessed. If the completion arteriogram shows a satisfactory result, the sheath is withdrawn so that its tip is pulled back over the aortic bifurcation. The guidwire is removed, a dressing is placed, and the patient is moved to another area where the sheath is removed.

Antegrade approach: Supplies required for an antegrade approach are listed in Table 24–7. An antegrade common femoral artery puncture is performed ipsilateral to the symptomatic side. This approach is well suited to patients who have normal aortoiliac inflow, especially if the patient requires tibial angioplasty or if there is a need to limit contrast. The puncture should be performed as proximally along the common femoral artery as possible to leave some working room between the puncture and the origin of the superficial femoral artery. A steerable guidewire, such as the Wholey guidewire (Mallinckrodt, Inc.), is used since the shaft of the guidewire is more supportive for catheter passage than a Glidewire. The Wholey guidewire often can be steered anteromedially into the superficial femoral artery. If not, the guidewire is advanced into the profunda femoris artery and an angled tip catheter is placed over it (Figure 24–3). The image intensifier is placed in the ipsilateral anterior oblique position to open the femoral bifurcation and the catheter is withdrawn enough to perform a roadmap by refluxing contrast into the superficial femoral artery. The catheter is used to steer the guidewire into the superficial femoral artery.

If the lesion is in the proximal to mid-SFA, roadmap the artery using the catheter and advance the guidewire across the lesion (Figure 24–4). The same guidewire may

TABLE 24-7. SUPPLIES FOR ANTEGRADE FEMORAL APPROACH TO INFRAINGUINAL INTERVENTION

Guidewire	Starting/selective guidewire	Wholey	145 cm, length	0.035 in. diameter (steerable, shapeable tip)
	Selective guidewire	Glidewire	180 cm	0.035 in (angled tip)
	Exchange guidewire	Rosen	180 cm	0.035 in. (J tip)
Catheter	Selective	Kumpe	40 cm	5 Fr (short, bent tip)
	Exchange	Straight	70 cm	5 Fr
Sheath	Access	Standard hemostatic access	12 cm	4 Fr, 5 Fr, 7 Fr[a]
Balloon	Balloon angioplasty catheters	Balloon diameter	2, 3, 4, 5, 6 mm	
		Balloon length	2, 4 cm	
		Catheter shaft	75 cm, distal tibial may require 90 cm	
Stent	Self-expanding	Wallstent		
		Stent diameter	6, 8 mm	
		Stent length	20, 40, 45, 60 mm	
		SMART		
		Stent diameter	6, 8 mm	
		Stent length	20, 40, 60, 80 mm	
		Delivery catheter length	80 cm	

[a]Use 4 Fr sheath for tibial balloon angioplasty with 3.8 Fr catheters. Use 5 Fr sheath for balloon angioplasty up to 6 mm on a 5 Fr shaft. A 7 Fr sheath is required for stent placement using a 0.035 in. system.
From: Schneider PA. The infrainguinal arteries—advice about balloon angioplasty and stent placement. In: *Endovascular Skills,* 2nd Edition, Marcel Dekker, NY, 2003; pp. 319.

be used for sheath placement. If the lesion is more distal in the artery, the guidewire is advanced without crossing the lesion and the sheath is placed. The sheath required may be 4, 5, or 6 Fr, depending upon the platform used and whether the balloon angioplasty will be followed by a stent. An appropriately sized dilator is used to enlarge the arteriotomy before sheath placement.

After the sheath is placed, femoral arteriography is performed through the side arm of the sheath. Heparin is administered. Standard length, 150 cm, 0.035 in. guidewires may be used for lesions above the knee. Longer guidewires, 180 cm or 260 cm are used for infrageniculate balloon angioplasty, especially in tall patients. A 75 or 80 cm length balloon angioplasty catheter may be used to the mid-tibial level. Longer catheters are required for more distal lesions. The lesion is evaluated angiographically. A steerable Glidewire is used to cross the lesion. The arteriogram is repeated after the guidewire is across the lesion to be certain that the guidewire is in the distal artery, and not in a perigenicular collateral. The balloon catheter is selected, passed over the guidewire, and balloon angioplasty is performed. Completion arteriography is performed through the sheath while maintaining guidewire control until results are assessed.

Stents: Stents may be used to manage poor immediate post-angioplasty results without resorting to emergent surgery. Stents are placed for post-angioplasty residual stenosis and/or flow-limiting dissection. Some residual stenosis at the angioplasty site is acceptable. When residual stenosis exceeds 30 to 50%, a stent should be placed. Dissection can be identified after almost every balloon angioplasty in the femoral and

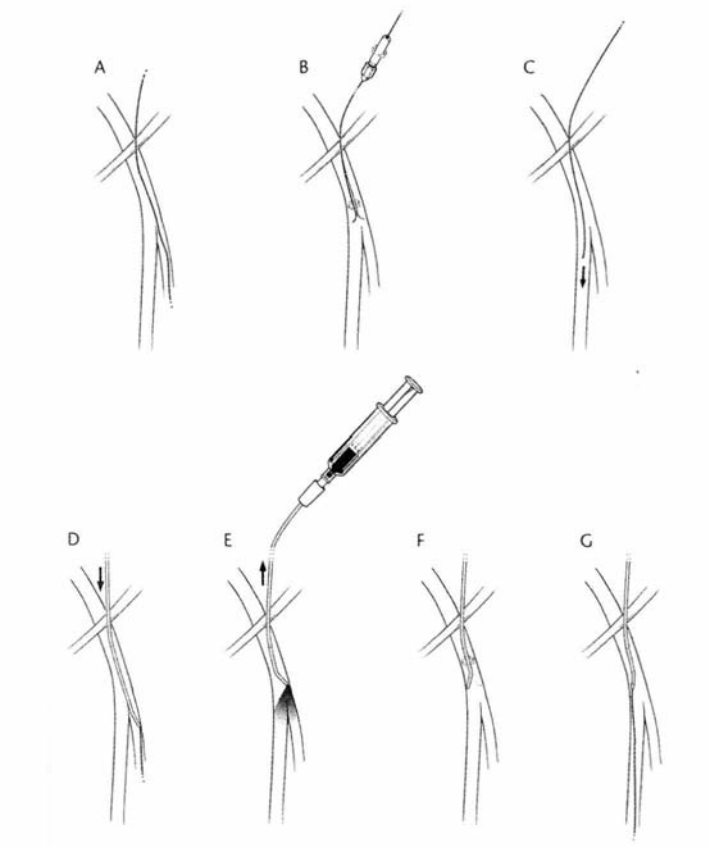

Figure 24-3. Catheterization of the superficial femoral artery through an ipsilateral antegrade approach. A: After antegrade femoral puncture, the guidewire tends to advance into the PFA. B: A Wholey guidewire may be used with a torque device to direct the guidewire into the SFA. C: The guidewire tip is rotated anteriorly and medically to enter the SFA. D: Another option is to pass an angled tip catheter into the PFA. E: The guidewire is removed and the catheter is slowly withdrawn while puffing contrast to demonstrate the femoral bifurcation. F: The catheter tip is rotated toward the SFA. G: The guidewire is advanced through the catheter into the SFA. From: Schneider PA. Selective catheterization. In: *Endovascular Skills*, 2nd Edition, New York; Marcel Dekker; 2003:109.

popliteal arteries. Mild dissections do not require treatment. However, if the is substantial residual stenosis from the dissection or there is flow limitation, or contrast trapping in the wall of the artery, a stent should be placed. Primary stent placement should be performed when recanalizing an occlusion or performing a subintimal balloon angioplasty.

Most of the stents placed in the femoral and popliteal arteries are self-expanding stents (Figure 24–5). These stents are flexible and must be oversized by 1 to 3 mm to intended artery segment. The newer Nitinol stents foreshorten only minimally and can be obtained with markers on the ends for better visualization. Stent delivery catheters are either 80 or 120 cm in length and may be selected based on the length of the balloon catheter required. Self-expanding stents deploy by unfurling from the tip end of the catheter, back toward the hub end of the catheter. Most of the many available stents are deployed with the same mechanism: the pushing rod is held steady while the stent delivery catheter hub is pulled back. This withdraws the membrane covering

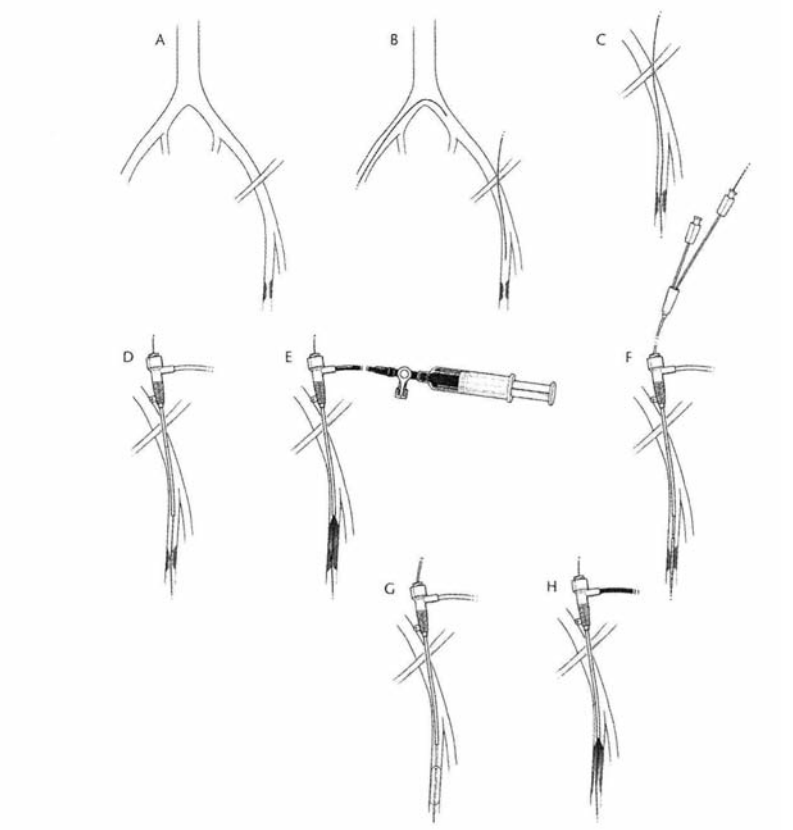

Figure 24-4. Balloon angioplasty of the superficial femoral and popliteal arteries through an antegrade approach. A: A stenosis of the SFA is suitable for balloon angioplasty. B: The lesion may be approached either from the contralateral or the ipsilateral femoral artery. C: The guidewire is placed across the stenosis through an antegrade approach. D: An access sheath is placed in the proximal SFA. E: Arteriography is performed through the sidearm of the sheath to evaluate the lesion and confirm guidewire position. F: The balloon angioplasty catheter is passed over the guidewire and advanced into the lesion. G: Balloon angioplasty is performed. H: Completion arteriography is performed. Guidewire position is maintained until the results are assessed. From: Schneider PA. The infrainguinal arteries—advice about balloon angioplasty and stent placement In: *Endovascular Skills*, 2nd Edition, New York; Marcel Dekker; 2003:318.

the stent and it deploys. The stent is visualized with fluoroscopy. The constrained stent is passed slightly beyond the lesion and is pulled back slightly with a fine-tuning adjustment as its tip end begins to open. After the stent is deployed, repeat balloon angioplasty is performed.

INDICATIONS FOR ENDOVASCULAR INTERVENTION: CURRENT APPROACH IN EVOLUTION

Endovascular intervention in the infrainguinal arteries may assume a substantially different role in various clinical practices, depending upon the specialists' approach to the current results and the techniques. The most conservative approach, and one that has been

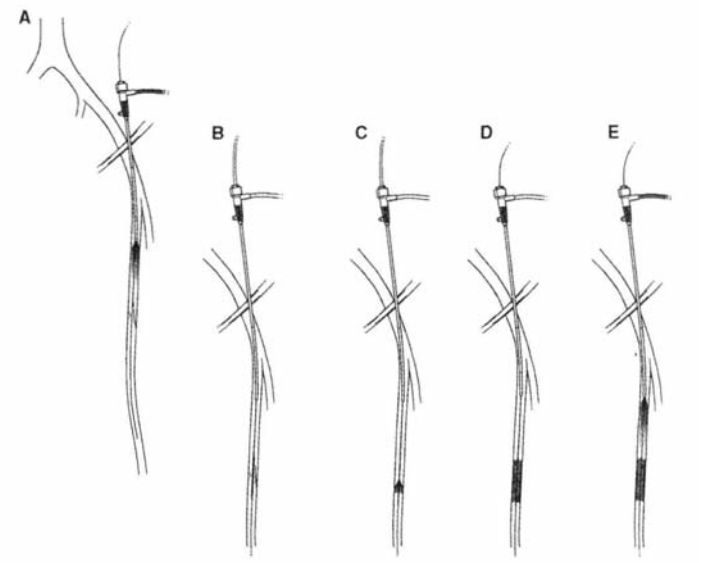

Figure 24-5. Stent placement in the superficial femoral artery. A: Dissection is present after balloon angioplasty. B: A self-expanding stent delivery catheter is placed over the guidewire and advanced into the segment of dissection. C: The stent is deployed from the tip end to the hub end of the catheter. D: Post-stent balloon angioplasty is performed to bring the stent to its appropriate profile. E: Completion arteriography is performed. From: Schneider PA. The infrainguinal arteries—advice about balloon angioplasty and stent placement. Schneider PA. The infrainguinal arteries—advice about balloon angioplasty and stent placement. In: *Endovascular Skills*, 2nd Edition, New York; Marcel Dekker; 2003:321.

followed by many vascular surgeons, is balloon angioplasty for only TASC A lesions and surgery for everyone else. However, the development of a variety of self-expanding stents, better access techniques, small platform devices, subintimal angioplasty and better recanalization techniques has prompted the present situation in which an endovascular approach is safe and technically feasible in most cases. In our practice, approximately 60% of patients requiring treatment for femoral-popliteal disease and 15% of patients with tibial disease are treated with endovascular surgery. Endovascular intervention is the treatment of choice for TASC A and B lesions. TASC C lesions are treated with endovascular intervention for patients with limited life expectancy. TASC D lesions are usually treated with surgery but may be considered for endovascular intervention in patients with limb threatening ischemia and prohibitive risk for open surgery. Patients offered surgery as the initial treatment of choice have extensive, multilevel disease, (usually occlusions) for which endovascular options are not durable. It is likely that this approach will continue to evolve since further developments such as, drug-eluting stents and covered stents, are likely to become clinically useful in the next few years.

REFERENCES

1. TransAtlantic Inter-Society Consensus. Management of Peripheral Arterial Disease. Femoropopliteal PTA. *J Vasc Surg.* 2000;31:S103–107.
2. Schneider PA. Endovascular interventions in the management of chronic lower extremity ischemia. In: Rutherford RB (ed.) *Vascular Surgery* 5th Edition, Philadelphia:Saunders; 2000:1055–1058.

3. Johnston KX. Femoral and popliteal arteries: reanalysis of results of balloon angioplasty. *Radiology.* 1992;183:767–771.
4. Hunink MG, Donaldson MC, Meyerovitz MF et al. Risks and benefits of femoropopliteal percutaneous balloon angioplasty. *J Vasc Surg.* 1993;17:183–194.
5. Capek P, McLean GK, Berkowitz HD. Femoropopliteal angioplasty: factors influencing long term success. *Circulation.* 1991;83 (suppl 1):170–180.
6. Hunink MG, Wong JB, Donaldson MC et al. Patency results of percutaneous and surgical revascularization for femoro-popliteal arterial disease. *Med Decis Making*. 1994;14:71–81.
7. Muradin GS, Bosch JL, Stijnen T, Hunink MG. Balloon dilatation and stent implantation for treatment of femoropopliteal arterial disease: meta-analysis. *Radiology*. 2001;221:137–145.
8. Murray JG, Apthorp LA, Wilkins RA. Long segment femoropopliteal angioplasty: improved technical results and long-term patency. *Radiology*. 1995;195:158–162.
9. Currie IC, Wakeley CJ, Cole SE et al. Femoropoliteal angioplasty for sever limb ischemia. *Br J Surg*. 1994;81:191–193.
10. Jeans WD, Armstrong S, Cole SEA et al. Fate of patients undergoing transluminal angioplasty for lower limb ischemia. *Radiology*. 1009;177:559–564.
11. Krepel VM, van Andel GJ, van Erp WF, Breslau PJ. Percutaneous transluminal angioplasty of the femoropopliteal artery: initial and long-term results. *Radiology*. 1985;156:325–328.
12. Hunink MG, Wong JB, Donaldson MC et al. Revascularization for femoropopliteal disease: a decision and cost-effectiveness analysis. *JAMA*. 1995;274:165–171.
13. Vroegindeweij D, Vos LD, Tielbeek AV et al. Balloon angioplasty combined with primary stenting versus balloon angioplasty alone in femoropopliteal obstructions: a comparative randomized study. *Cardiovasc Intervent Radiol*. 1997;20:420–425.
14. Cejna M, Thurnhur S, Illiasch H et al. PTA versus Palmaz stent placement in femoropopliteal artery obstructions: a multicenter, prospective randomized study. *J Vasc Interv Radiol*. 2001;12:23–31.
15. Grimm J, Muller-Hulsbeck S, Jahnke T et al. Randomized study to compare PTA alone versus PTA with Palmaz stent placement for femoropopliteal lesions. *J Vasc Interv Radiol*. 2001;12:935–942.
16. Becquemin JC, Favre JP, Marzelle J et al. Systematic versus selective stent placement after superficial femoral artery balloon angioplasty: a multicenter, prospective randomized study. *J Vasc Surg*. 2003;37:487–494.
17. Conroy RM, Gordon IL, Tobis JM et al. Angioplasty and stent placement in chronic occlusion of the superficial femoral artery: technique and results. *J Vasc Interv Radiol*. 2000;11: 1009–1020.
18. Lugmayr HF, Holzer H, Kastner M et al. Treatment of complex arterioisclerotic lesions with nitinol stents in the superficial femoral and popliteal arteries: a midterm follow-up. *Radiology*. 2002;222:37–43.
19. Cho L, Roffi M, Mukherjee D et al. Superficial femoral artery occlusion: nitinol stents achieve better flow and reduce the need for medications than balloon angioplasty alone. *J Invasive Cardiol*. 2003;15:198–200.
20. Duda SH, Pusich B, Richter G et al. Sirolimus-eluting stents for the treatment of obstructive superficial femoral artery disease: six-month results. *Circulation*. 2002;17:1505–1509.
21. Saxon RR, Coffman JM, Gooding JM et al. Long-term results of ePTFE stent-graft versus angioplasty in the femoropopliteal artery: single center experience from a prospective, randomized trial. *J Vasc Interv Radiol*. 2003;14:303–311.
22. Kandarpa K, Becker GJ, Hunink M et al. Transcatheter interventions for the treatment of peripheral atherosclerotic lesions: part I. *J Vasc Interv Radiol*. 2001;12:683–695.

25

Lower Extremity Vein Grafts Requiring Multiple Revisions

Gregory J. Landry, M.D., Gregory L. Moneta, M.D., Lloyd M. Taylor, Jr., M.D., and John M. Porter, M.D.

Autogenous infrainguinal bypass is the mainstay of surgical treatment of limb threatening ischemia and disabling claudication. While techniques of autogenous bypass vary (in situ, reversed, transposed nonreversed), all techniques share in common the requirement for occasional bypass graft revision. The reason for revision can be due either to development of a stenosis within the graft or at one of its anastomoses, or progression of native disease in the arterial inflow or outflow. Several modern series have identified the need for graft revision in up to 25% of infrainguinal vein grafts.[1-6]

Refinement of identification of grafts at risk has been made possible by duplex ultrasound-based graft surveillance protocols. While there is some disagreement on the specific duplex criteria for graft revision, it is clear that duplex surveillance is an effective and cost-efficient method of identifying grafts with stenoses that threaten patency.

Techniques of graft revision vary according to the practice of the individual surgeon and the nature and location of the graft threatening lesion. The overall philosophy of infrainguinal graft revision, however, parallels that of the initial bypass procedure, i.e., the use of all autogenous materials whenever possible. Obviously, this frequently requires the use of conduit other than greater saphenous vein, including arm vein, lesser saphenous vein, femoral vein, and, occasionally, angioplasty of stenotic segments.

With an aging population enjoying increased life expectancy combined with a rising incidence of diabetes in the overall population, it is likely more bypasses will be performed and, therefore, more will require revision. It has also become increasingly evident that multiple (>1) revisions may be required to maintain graft patency. There is, however, little data on the long term patency of lower extremity bypass grafts requiring multiple revisions.

VEIN GRAFT SURVEILLANCE

Abundant evidence exists supporting the efficacy of aggressive duplex vein graft surveillance, and, when necessary, operative revision to maintain patency of infrainguinal vein grafts. While this is not a sentiment shared by all,[7] the majority of current evidence does in fact favor repair of stenotic vein graft lesions. While most vein graft stenoses are identified within the first year after revascularization, late stenoses occur beyond one year with sufficient frequency to warrant life-long surveillance.[4,8-10] Duplex criteria for identifying a significant vein graft stenosis vary from center to center, however, in general, a focal increase in peak systolic flow velocity greater than 200 cm/sec, a pre- to intra-stenotic peak systolic velocity ratio of greater than 3.0, or uniform maximum graft flow velocities <45 cm/sec are considered consistent with a hemodynamically significant stenosis somewhere in the bypass graft or its inflow or outflow arteries.[1] Clinical suspicion, while less reliable in the absence of these findings is also appropriate, particularly in the presence of a decrease in the ankle/brachial index or worsening of the patients symptoms.

VEIN GRAFT REVISION

Surgical revision of infrainguinal vein grafts with significant stenoses is indicated to maintain graft patency. Several series now have shown three to five-year assisted primary vein graft patencies of 80–90% when an aggressive program to revise threatened grafts is employed.[1,2,5,11,12] From a cost perspective, surveillance with graft revision is justified compared with the high cost of revision after graft thrombosis or limb amputation.[13]

The reason for development of vein graft stenoses is unclear, however, it seems certain that the quality of the original conduit is an important factor. Idu and associates determined that factors correlating with the development of vein graft stenoses included a minimal graft diameter <3.5 mm, the use of a venovenous anastomosis, and the length of the graft.[14] Multivariate regression analysis, however, revealed only small graft diameter to significantly correlate with the development of a vein graft stenosis. Stenosis-free rates for grafts with a minimal diameter <3.5 mm, between 3.5–4.5 mm, and >4.5 mm were 40%, 58%, and 75% respectively ($p<0.05$). Composite vein and arm vein grafts with minimal diameters >3.5 mm were compared with grafts which consisted of a single uninterrupted greater saphenous vein with a minimal diameter of <3.5 mm. One-year secondary patency rates in these categories were 94% and 76% respectively. The authors concluded that a minimal graft diameter of <3.5 mm was the only factor that significantly correlated with the development of a graft stenosis; however, vein grafts with larger diameters can still develop stenotic lesions. Others have found an association between smoking and the use of alternate conduit with the development of vein graft stenosis.[15]

MULTIPLE GRAFT REVISIONS

Occasionally, multiple graft revisions are necessary to maintain patency. While several reports have addressed the issue of primary revisions, very few studies have examined grafts which have been subjected to multiple revisions.

TABLE 25-1. DEMOGRAPHICS OF PATIENTS UNDERGOING SINGLE AND MULTIPLE LEVG REVISIONS

Demographics	Multiple revisions (N=37)	Single revision (N=146)
age at original operation(yrs±sd)*	62±10	67±12
age at revision(yrs)*	64±10	68±10
gender m	65%	71%
f	35%	29%
hypertension	68%	82%
coronary artery disease	54%	51%
smoking history	92%	90%
diabetes mellitus	41%	40%
cerebrovascular disease	16%	30%
prior vascular bypass procedures type	46%	49%
aortofemoral bypass	19%	18%
ipsilateral leg bypass	22%	22%
contralateral leg bypass	11%	15%
end stage renal disease	5%	10%
warfarin therapy	30%	31%
hypercoagulable state	8%	3%

*p<0.05, all others p=ns

We reviewed our experience with grafts subjected to multiple revisions from 1990 to 1998.[16] During this time, 233 vein graft revisions were performed, fifty of which (21.4%) were repeat revisions. The fifty secondary revisions were performed in 37 patients. Twenty-seven patients underwent two revisions, seven underwent three revisions, and three underwent four revisions. Multiple lesions were repaired in ten of the revisions, for a total of sixty lesions repaired in fifty secondary revisions. The demographic characteristics of patients requiring single and multiple revisions are listed in Table 25–1. Patients requiring multiple revisions were significantly younger at the time of their original operation and at the time of revision. No other demographic differences were noted.

The type of original operation, inflow site, initial operative indication and conduit are listed in Table 25–2. The type of original operation did not influence subsequent need for single or multiple revisions. Inflow from vessels other than the common femoral artery was associated with more frequent need for multiple revisions, however, this did not reach statistical significance (p=0.08). The initial operative indication and the conduit source also did not affect the need for multiple revisions.

The characteristics of revision procedures performed are listed in Table 25–3 and reflect the nature of the lesion and availability of suitable conduit. Revision procedures included inflow procedures (e.g., femorofemoral bypass) in which the proximal portion of the graft was involved, extension of the graft to a more proximal inflow site or more distal outflow site, placement of an interposition vein segment within the graft, or placement of a vein patch over an area of focal vein graft stenosis.

Recurrent lesions requiring revision can occur both at the site of a prior revision and at a new, previously unrevised site. Of the sixty lesions repaired in this series, 29 (48%) were at previously revised sites, and 31 (52%) were at new, previously unrevised sites. When a previously revised site required revision, the majority (69%) were within the body of the graft, while 31% were anastomotic lesions. When a new,

TABLE 25-2. CHARACTERISTICS OF THE ORIGINAL OPERATION IN PATIENTS UNDERGOING SINGLE AND MULTIPLE LEVG REVISIONS

	Multiple revisions (N=37)	Single revision (N=146)
operation category		
femoral-popliteal (above knee)	11%	10%
femoral-popliteal (below knee)	41%	41%
femoral-tibial	46%	45%
posterior tibial	11%	14%
anterior tibial	19%	18%
peroneal	16%	13%
femoral-pedal	3%	
inflow site		3%
common femoral artery*	30%	47%
superficial femoral artery	35%	24%
profunda femoral artery	35%	
operative indication		25%
claudication	30%	27%
limb salvage	68%	73%
rest pain	41%	36%
ulcer/gangrene	27%	37%
popliteal aneurysm	2%	
conduit		0%
reversed saphenous vein graft	70%	79%
alternate vein graft	30%	21%
single arm vein	5%	6%
composite arm/leg	25%	15%

*p=0.08, Chi-square

previously unrevised site required revision, 48% were new sites within the graft, 10% were new anastomotic lesions, and 42% were due to atherosclerotic progression of the native arterial inflow or outflow.

When a revision was required at a site of prior revision, the time interval between the initial and secondary revision was 11±2 months. If a different site required revision, the time interval between the initial and secondary revision was 20±4 months ($p<0.05$). This difference in time intervals suggests a difference in pathophysiologic mechanism. One popular theory of the mechanism of atherosclerotic progression states that lesions that occur within a year of surgery are more likely due to intimal hyperplasia, whereas those that occur beyond one year are due to recurrent atherosclerosis.[17] One could postulate that when a previously revised site requires revision, this is due to intimal hyperplasia at a site of prior surgical injury. When a new site in a well established graft requires revision, atherosclerosis may be the likely cause.

Five-year assisted primary patency, limb salvage, and survival for patients undergoing single and multiple graft revisions are shown in Figures 25–1, 25–2 and 25–3. Five-year assisted primary patency was 91% in patients undergoing multiple revisions and 89% in those undergoing single revisions (p=ns). These patency rates are higher than those traditionally recorded for lower extremity grafts. The reason for this is unclear, however, it is likely related to the compliance of this group of patients in following a regimented duplex graft surveillance protocol. There may also be some variable,

TABLE 25-3. CHARACTERISTICS OF REVISION PROCEDURES IN PATIENTS UNDERGOING SINGLE AND MULTIPLE LEVG REVISIONS

	Multiple revisions (N=37)	Single revision (N=146)
Initial revision procedures		
inflow procedure	8%	8%
proximal revision	32%	33%
interposition	32%	28%
vein patch angioplasty	22%	23%
distal extension	22%	25%
Initial revision conduit		
ipsilateral greater saphenous vein	30%	22%
contralateral greater saphenous vein	8%	12%
basilic vein	30%	37%
cephalic vein	16%	14%
prosthetic	5%	3%
other	11%	14%
Secondary revision procedures		
inflow procedure	6%	
proximal revision	34%	
interposition	26%	
vein patch angioplasty	8%	
distal extension	30%	
other	6%	
Secondary revision conduit		
ipsilateral greater saphenous vein	10%	
contralateral greater saphenous vein	18%	
basilic vein	32%	
cephalic vein	14%	
prosthetic	14%	
other	12%	

as yet undefined, that allows some vein grafts to tolerate stenoses and remain patent until the lesion is detected. Five-year limb salvage was also not different between patients undergoing single (94%) vs. multiple (89%) graft revisions.

Interestingly, five-year survival in patients undergoing single revisions was 78% vs. 89% in patients undergoing multiple revisions ($p=0.08$). These survival data are dramatically elevated compared with most series of bypass grafts. Again, the reason for this is unclear, however, a likely explanation is that patients who die early after leg bypass die before revisions are necessary, and survival itself is associated with the ultimate need for graft revision.

One additional question examined was whether or not the need for multiple revisions could have been predicted based upon the preoperative arteriogram at the time of the initial revision. In cases in which a new lesion was responsible for secondary revision, the arteriogram from the initial revision was reviewed to identify evidence of the secondary lesion at the time of the initial revision. Of the 26 cases reviewed, evidence of a minor (<50%) arteriographic stenosis was present in only six (23%) at the time of the prior revision. In the remaining 20 cases (77%), there were no findings on arteriography to suggest the subsequent development of a lesion. The fact that

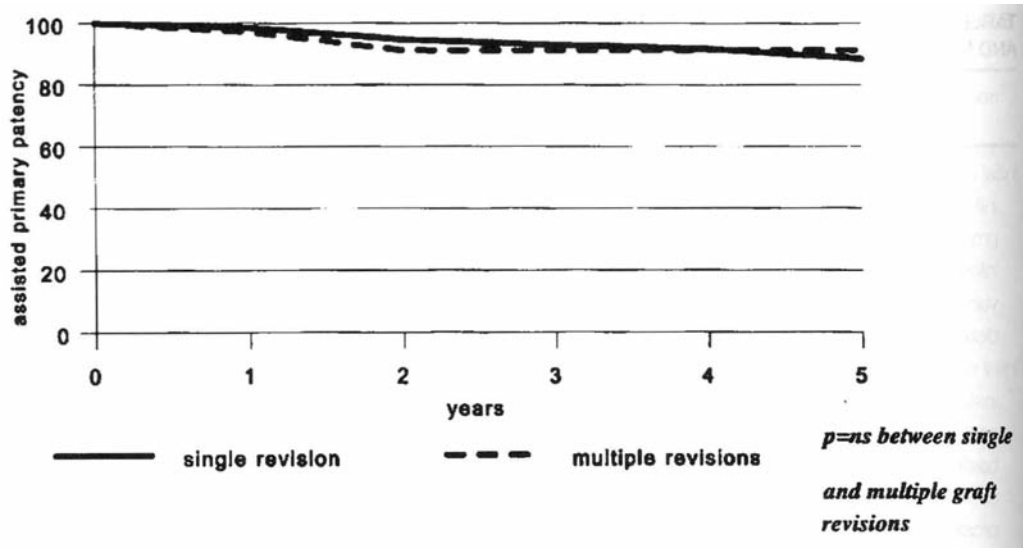

Figure 25-1. Assisted primary patency of revised lower extremity vein grafts.

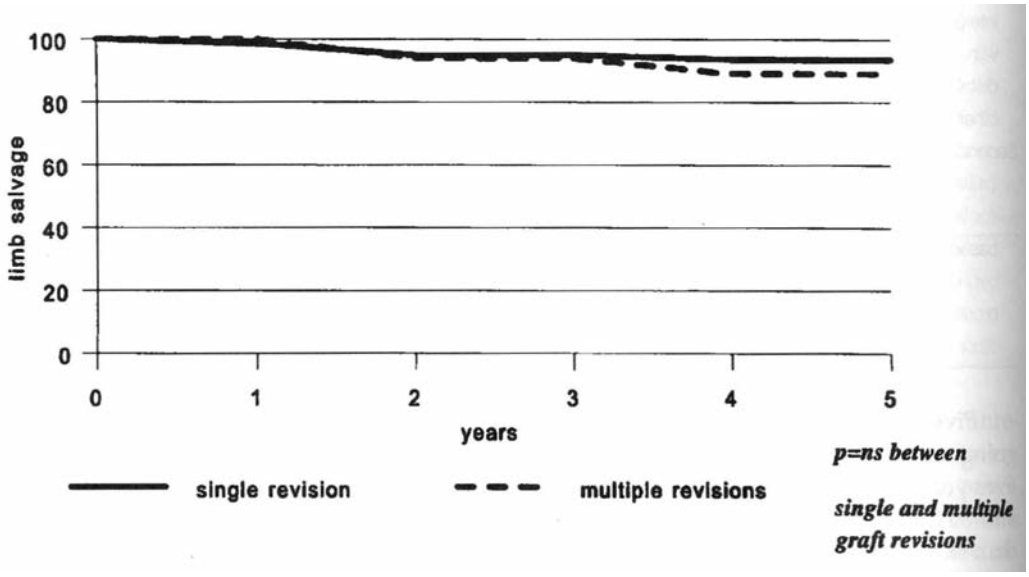

Figure 25-2. Limb salvage in patients with revised lower extremity vein grafts.

stenoses can occur in areas of the graft that were previously normal on arteriogram further underscores the importance of continued graft surveillance.

SUMMARY

Lower extremity vein grafts may require multiple revisions to maintain patency, but with aggressive duplex surveillance and surgical repair of stenotic lesions, excellent assisted primary patency and limb salvage can be achieved. Secondary lesions are just as likely to occur

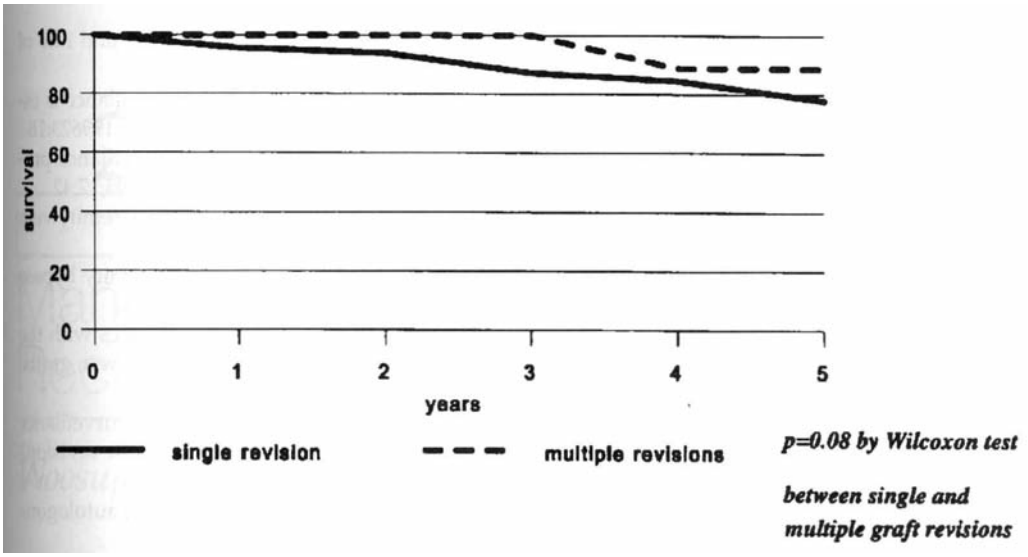

Figure 25-3. Survival in patients with revised lower extremity vein grafts.

at new, previously unrevised sites as at sites of prior repair. Lesions at previously unrevised sites occur later than those at a site of prior repair. In vein grafts requiring two or more revisions, new lesions are infrequently suggested at the time of angiography prior to the initial revision. As the population ages, life expectancies increase, and more vein grafts are placed, an increasing number of patients requiring multiple vein graft revisions is expected.

REFERENCES

1. Landry GJ, Moneta GL, Taylor LM, Jr., et al. Duplex scanning alone is not sufficient imaging before secondary procedures after lower extremity reversed vein bypass graft. *J Vasc Surg*. 1999;29:270.
2. Nehler MR, Moneta GL, Yeager RA, et al. Surgical treatment of threatened reversed infrainguinal vein grafts. *J Vasc Surg*. 1994;20:558.
3. Caps MT, Cantwell-Gab K, Bergelin RO, et al. Vein graft lesions: time of onset and rate of progression. *J Vasc Surg*. 1995;22:466.
4. Erickson CA, Towne JB, Seabrook GR, et al. Ongoing vascular laboratory surveillance is essential to maximize long-term in situ saphenous vein bypass patency. *J Vasc Surg*. 1996;23:18.
5. Idu MM, Blankensteyn JD, de Gier P, et al. Impact of a color-flow duplex surveillance program on infrainguinal vein graft patency. A five-year experience. *J Vasc Surg*. 1993;17:42.
6. Gentile AT, Mills JL, Gooden MA, et al. Identification of predictors for lower extremity vein graft stenosis. *Am J Surg*. 1997;174:218.
7. Dougherty MJ, Calligaro KD, DeLaurentis DA. Revision of failing lower extremity bypass grafts. *Am J Surg*. 1998;176:126.
8. Ihnat DM, Mills JL, Dawson DL, et al. The correlation of early flow disturbances with the development of infrainguinal graft stenosis: a 10-year study of 341 autogenous vein grafts. *J Vasc Surg*. 1999;30:8.
9. Passman MA, Moneta GL, Nehler MR, et al. Do normal early color-flow duplex surveillance examination results of infrainguinal vein grafts preclude the need for late graft revision? *J Vasc Surg*. 1995;22:476.

10. McCarthy MJ, Olojugba D, Loftus IM, et al. Lower limb surveillance following autologous vein bypass should be life long. *Br J Surg*. 1998;85:1369.
11. Mattos MA, van Bemmelen PS, Hodgson KJ, et al. Does correction of stenoses identified with color duplex scanning improve infrainguinal graft patency? *J Vasc Surg*. 1993;17:54.
12. Bergamini TM, George SM, Jr., Massey HT, et al. Intensive surveillance of femoropolitealtibial autogenous vein bypasses improves long-term patency and limb salvage. *Ann Surg*. 1995;221:507.
13. Wixon CL, Mills JL, Westerbrand A, et al. An economic appraisal of lower extremity bypass graft maintenance. *J Vasc Surg*. 2000;32:1.
14. Idu MM, Buth J, Hop WC, et al. Factors influencing the development of vein-graft stenosis and their significance for clinical management. *Eur J Vasc Endovasc Surg*. 1999;17:15.
15. Gentile AT, Mills JL, Gooden MA, et al. Identification of predictors for lower extremity vein graft stenosis. *Am J Surg*. 1997;174:218.
16. Landry GJ, Moneta GL, Taylor LM, Jr., et al. Patency and characteristics of lower extremity reversed vein grafts requiring multiple revisions. *J Vasc Surg*. 2000;32:23.
17. Szilagyi DE, Elliott JP, Hageman JH, et al. Biologic fate of autologous vein implants as arterial substitutes: clinical, angiographic and histologic observations in femoropopliteal operations for atherosclerosis. *Ann Surg*. 1973;178:232.

SECTION **IV**

EVAR

26

Update on EVAR

Audrey Rosinberg, M.D. and William Pearce, M.D.

INTRODUCTION

Since Parodi first introduced endovascular aortic aneurysm repair in 1991,[1] the technology has evolved dramatically. Multiple different advances have allowed the widespread adoption of endovascular aortic repair (EVAR) for infrarenal abdominal aortic aneurysms (AAA). Currently there are 5 commercially available devices manufactured by 4 different companies. Massive amounts of data have been accumulated in the last 20 years; however, given the rapid evolution of devices and delivery systems, studies are often nearly obsolete by the time they are published and there is a paucity of level 1 evidence supporting the long-term durability and cost-effectiveness of EVAR compared to open surgery.

RANDOMIZED TRIALS

A total of 6 randomized controlled trials involving EVAR are under way. The EVAR 1[2] and Dutch Randomized Endovascular Aneurysm Management trials (DREAM)[3] trials randomized patients who were fit for open repair with aneurysms greater than 5.5 cm to endovascular versus open surgery. The EVAR 2[4] trial randomized patients with aneurysms greater than 5.5 cm and unfit for open repair between EVAR and non-operative management. The OVER[5] (Open Versus Endovascular Repair) trial included veterans with aneurysms greater than 5.0 cm eligible for both open and endovascular repair. The trial is ongoing but short-term results were reported in 2009.

The DREAM trial was the first randomized controlled trial to publish mid-term results in 2005[6] and now long-term outcomes[3] with a median follow-up of 6.4 years. The DREAM trial randomized 351 patients with abdominal aortic aneurysms at least 5 cm in diameter who were suitable candidates for both procedures to either open or endovascular repair. The mean age of the patients was 70 years of age. The median follow-up was 6.4 years. All patients were followed for 5 years and 53% of the patients were followed for 7 years. Although there was a small statistically significant survival advantage for EVAR at 30 days post-op, this disappeared by mid-term follow-up at

2 years and continued through the duration of the trial. There was no significant difference in survival at 6 years (69.9% for open repair and 68.9% for endovascular repair). Additionally, the authors found that quality of life was not statistically different between the two groups by 6 months. The authors also found that reintervention was statistically greater in the EVAR group.

EVAR trial 1 randomized 1082 patients who had aneurysms greater than 5.5 cm and were candidates for both open and endovascular aneurysm repair in the United Kingdom.[2] Approximately half of those screened did not have suitable anatomy for EVAR and were eliminated prior to randomization. The primary endpoint of EVAR 1 was all-cause mortality. There was no significant difference at 4-year follow-up with approximately 28% mortality. Aneurysm-related mortality was significantly better for EVAR (4%) versus open surgery (7%) despite the significantly higher long-term complication and reintervention rate for EVAR. No difference was found in quality-of-life questionnaires between EVAR and open repair by 3 months follow-up.

EVAR trial 2 enrolled patients who were unfit for open repair.[4] A total of 338 patients with aneurysms greater than 5.5 cm were randomized to EVAR or best medical therapy. The aneurysm rupture rate during medical management was 9% per year. Twenty-seven percent of the patients randomized to medical management crossed over to surgery. Only 15% of the patients were followed to 4 years. Overall, all-cause mortality was 64% at 4 years and was not statistically different between EVAR and medical management. Additionally, there was no significant difference in aneurysm-related mortality between the two groups.

The OVER (outcomes following endovascular versus open repair of abdominal aortic aneurysm) trial began enrollment in 2002 and recently reported outcomes up to 2 years in the midst of a planned 9-year trial.[5] A total of 881 veterans with AAA greater than 5 cm in diameter, or 4.5 cm with rapid enlargement, were randomized to receive either open repair or EVAR. Endovascular repair resulted in significantly reduced procedure time, hospital and ICU stays, duration of mechanical ventilation, and transfusion requirement. Perioperative mortality was significantly higher for open repair at 30 days (0.2% for EVAR versus 2.3% for open repair). However, at 2 years follow-up, there was no statistically significant difference between the two groups in all-cause mortality (7.0% for EVAR versus 9.8% for open repair, p=.13). As opposed to the findings of the DREAM trial and EVAR 1, there was no statistically significant difference in reintervention between the groups. The majority of reinterventions in the EVAR group were for endoleaks and for incisional hernias in the open repair group. Finally, the authors found no statistically significant differences in quality-of-life measures after EVAR or open surgery.

LIMITATIONS OF CURRENT DATA

Many of the grafts included in the DREAM, EVAR 1 and EVAR 2 have gone through evolutions in their design to correct flaws. These grafts are currently in their third-generation. Many of the devices in the trial are first- and second-generation devices.

Much of the data currently available is from registries including the EUROSTAR (European Collaborators on Stent-Graft Techniques for Abdominal Aortic Aneurysm Repair)[7] and Lifeline[8] registries as well as industry-sponsored trials. The Lifeline registry contains open surgical control patients, whereas EUROSTAR contains only EVAR

data. Data from these registries as well as industry-sponsored trials must be carefully scrutinized, as it is subject to considerable selection bias that is inherent when patients are not randomized.

ONGOING TRIALS

Two randomized controlled trials, ADAM[9] and UKSAT,[10] demonstrated that early open surgical repair of small aneurysms less than 5.5 cm conferred no advantages compared to surveillance. Two randomized controlled trials are currently underway to evaluate early EVAR versus surveillance in patients with small AAAs. CEASAR (Comparison of surveillance versus Aortic Endografting for Small Aneurysm Repair)[11] is currently underway in Europe and PIVOTAL (Positive Impact of Endovascular Options for Treating Aneurysms Early)[12] in the United States. In summary, despite short-term gains in morbidity and mortality of AAA repair, durability, increased reinterventions, and costs remain a valid concern for EVAR.

RUPTURED AAA

Increasingly, EVAR is being used to treat ruptured AAA.[13] EVAR has the unique advantage of allowing repair of the ruptured aneurysm under local anesthesia. Rapid control of the hemorrhage can be obtained by inflation of an occlusion balloon in the aorta. Patients must be stable enough to undergo pre-operative CT scanning to evaluate anatomic suitability for EVAR. Additionally, endografts in a variety of sizes and configurations must be readily available, as must a specialized team familiar with the equipment. The IMPROVE trial in the United Kingdom is the only randomized controlled trial to date to randomize patients with suitable anatomy to either endovascular or open repair of ruptured AAA. It began enrolling patients in October 2009 and is currently accruing patients with plans to enroll 600 patients. The primary endpoint for this trial is survival benefit at 30 days. Secondary endpoints include 24-hour and 1-year survival benefit, major morbidities, costs, and quality of life.[14] The results of a pilot study for this trial of 32 patients showed no difference in 30-day mortality between open and endovascular repair. In fact, the only significant finding is that there was significantly less blood loss and fewer transfusions with EVAR.[15] A meta-analysis of retrospective and prospective cohort and case series found wide heterogeneity between studies with 30-day mortality for patients undergoing EVAR for ruptured AAA ranging from 0% to 45% with a pooled estimate of 21% compared to the mortality rate for patients undergoing open repair of 41% to 45%.[16] Many of these studies suffer from selection bias. Several groups have attempted to account for this selection bias. In the Netherlands, a prospective nonrandomized controlled trial compared endovascular and open repair of ruptured AAA only in patients with anatomy suitable for endovascular repair and found 30-day mortality to be significantly lower for endovascular repair (20% versus 45.5%, p=.04).[17] A review of the Medicare inpatient dataset from 1995 to 2004 found 43 033 patients who had undergone repair of ruptured AAA – 41 969 patients had open repair and 1064 patients underwent EVAR. Analysis of these patients found only a statistically significant short-term benefit of EVAR over open repair that did not persist after 90 days. However, when a propensity model was used to control for variables including

age, gender, race, co-morbidities, year of surgery, the hospital, and surgeon volume, there was a statistically significant survival benefit for EVAR versus open repair that persisted for 4 years.[18] Similarly, additional studies reviewing the NSQIP database as well as the nationwide inpatient sample found significant inpatient mortality benefit for EVAR over open repair despite greater co-morbidities in patients undergoing EVAR.[19,20] Although the outlook for EVAR is promising, the results of randomized trials as well as long-term data will further clarify the role of EVAR in the setting of ruptured AAA.

SURVEILLANCE

Standard follow-up after EVAR recommends that the patient undergo contrast-enhanced CT scan at 1, 6 and 12 months and then annually. This requirement exposes patients to both the nephrotoxicity from the contrast as well as radiation. Recent data increasingly support the use of duplex ultrasound for long term follow-up after EVAR with CT scan reserved for enlarging sac diameter or new endoleak visualized on duplex.[21,22] Ultrasound is technique- and operator-dependent; therefore, caution must be used when utilizing ultrasound as the sole follow-up modality as some authors advocate.

THE FUTURE: DIFFICULT ANATOMY

The instructions for use of current FDA-approved commercially available endografts have anatomical constraints that limit their use to infrarenal neck length of 15 mm, angulation less than 60 degrees. The devices with suprarenal fixation have been used by some operators off-label in high-risk operative patients with difficult neck anatomy. This can result in type I endoleaks at the proximal fixation point. Newer techniques to solve the problem of short or hostile neck anatomy are actively being explored. The "snorkel" or "chimney" technique involves placing a renal artery stent alongside the stent graft, allowing higher landing of the stent graft, and partially covering the renal artery orifice.[23,24] A similar technique can be used to maintain patency of the hypogastric artery when there is a short distal landing zone. Additionally, fenestrated and branched aortic endografts are being developed with the hope of increasing the number of patients who are candidates for EVAR.[25,26]

REFEREENCES

1. Parodi JC, Palmaz JC, Barone HD. Transfemoral intraluminal graft implantation for abdominal aortic aneurysm. *Ann Vasc Surg.* 1991;5:491–499.
2. EVAR Trial Participants. Endovascular aneurysm repair versus open repair in patients with abdominal aortic aneurysms (EVAR trial 1): randomized controlled trial. *Lancet.* 2005;365: 2179–2186.
3. DeBruin JL, Bass AF, Buth J, et al. Long term outcome of open or endovascular repair of abdominal aortic aneurysm. *N Engl J Med.* 2010;362:1881–1889.
4. EVAR Trial Participants. Endovascular aneurysm repair and outcome in patients unfit for open repair of abdominal aortic aneurysms (EVAR trial 2): randomised controlled trial. *Lancet.* 2005;365:2187–2192.
5. Lederle FA, Freischlag JA, Kyriakides TC, et al. Outcomes following endovascular vs open repair of abdominal aortic aneurysm: a randomized trial. *JAMA.* 2009;302:1535–1542.

6. Blankensteijn JD, de Jong SE, Prinssen M, et al. Two-year outcomes after conventional or endovascular repair of abdominal aortic aneurysms. *N Engl J Med.* 2005;352:2398–2405.
7. Leurs LJ, Hobo R, Buth J, et al. The multicenter experience with a third-generation endovascular device for abdominal aortic aneurysm repair. *J Cardiovasc Surg* (Torino). 2004;45:293–300.
8. Lifeline Registry of EVAR Publications Committee. Lifeline registry of endovascular aneurysm repair: long-term primary outcome measures. *J Vasc Surg.* 2005;42:1–10.
9. Lederle FA, Wilson SE, Johnson GR, et al. Immediate repair compared with surveillance of small abdominal aortic aneurysms. *New Eng J Med.* 2002;346:1437–1444.
10. United Kingdom Small Aneurysm Trial Participants. Long term outcomes of immediate repair compared with surveillance of small abdominal aortic aneurysms. *New Eng J Med.* 2002;346:1445–1452.
11. Cao P, CAESAR Trial Collaborators. Comparison of surveillance vs Aortic Endografting for Small Aneurysm Repair (CAESAR) trial: study design and progress. *Eur J Vasc Endovasc Surg.* 2005;30:245–251.
12. Ouriel K. The PIVOTAL study: a randomized comparison of endovascular repair versus surveillance in patients with smaller abdominal aortic aneurysms. *J Vasc Surg.* 2009;49:266–269.
13. Lesperance K, Anderson C, Singh N, et al. Expanding use of emergency endovascular repair for ruptured abdominal aortic aneurysms: disparities in outcomes from a nationwide perspective. *J Vasc Surg.* 2008;47:1165–1170.
14. Improve Trial. The Immediate Management of the Patient with Rupture: Open Versus Endovascular repair (IMPROVE) aneurysm trial. *Acta Chir Belg.* 2009;109:678–680.
15. Hinchliffe RJ, Bruijstens L, MacSweeney STR et al. A randomised trial of endovascular and open surgery for ruptured abdominal aortic aneurysm—results of a pilot study and lessons learned for future studies. *Eur J Vasc Endovasc Surg.* 2006;32:506–513.
16. Mastracci TM, Garrido-Olivares L, Cinà CS, et al . Endovascular repair of ruptured abdominal aortic aneurysms: a systematic review and meta-analysis. *J Vasc Surg.* 2008;47:214–221.
17. Ten Bosch JA, Teijink JAW, Willigendael EM, et al. Endovascular aneurysm repair is superior to open surgery for ruptured abdominal aortic aneurysms in EVAR-suitable patients. *J Vasc Surg.* 2010;52:13–18.
18. Egorova N, Giacovelli J, Greco G, et al. National outcomes for the treatment of ruptured abdominal aortic aneurysm: comparison of open versus endovascular repairs. *J Vasc Surg.* 2008;48:1092–1100.
19. Giles KA, Pomposelli F, Hamdan A, et al. Decrease in total aneurysm-related deaths in the era of endovascular aneurysm repair. *J Vasc Surg.* 2009;49:543–550.
20. McPhee J, Eslami MH, Arous EJ, et al. Endovascular treatment of ruptured abdominal aortic aneurysms in the United States (2001–2006): a significant survival benefit over open repair is independently associated with increased institutional volume. *J Vasc Surg.* 2009;49:817–826.
21. Sternbergh WC 3rd, Greenberg RK, Chuter TA, et al. Redefining postoperative surveillance after endovascular aneurysm repair: recommendations based on 5-year follow-up in the US Zenith multicenter trial. *J Vasc Surg.* 2008;48:278–284.
22. Mirza TA, Karthikesalingam A, Jackson D, et al. Duplex ultrasound and contrast-enhanced ultrasound versus computed tomography for the detection of endoleak after EVAR: systematic review and bivariate meta-analysis. *Eur J Vasc Endovasc Surg.* 2010;39:418–428.
23. Allaqaband S, Jan MF, Bajwa T. "The chimney graft" – a simple technique for endovascular repair of complex juxtarenal abdominal aortic aneurysms in no-option patients. *Catheter Cardiovasc Interv.* 2010;75:1111–1115.
24. Hiramoto JS, Chang CK, Reilly LM et al. Outcome of renal stenting for renal artery coverage during endovascular aortic aneurysm repair. *J Vasc Surg.* 2009;49:1100–1106.
25. Kristmundsson T, Sonesson B, Malina M, et al. Fenestrated endovascular repair for juxtarenal aortic pathology. *J Vasc Surg.* 2009;49:568–574.
26. Greenberg RK, Sternbergh WC 3rd, Makaroun M, et al. Intermediate results of a United States multicenter trial of fenestrated endograft repair for juxtarenal abdominal aortic aneurysms. *J Vasc Surg.* 2009;50:730–737.

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Techniques for EVAR

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INTRODUCTION

Endovascular repair of infrarenal abdominal aortic aneurysms (EVAR) has evolved over the past two decades to include a multitude of endoprostheses and approaches to suit an assortment of aneurysms. Herein is a summary of modern endovascular grafts and techniques employed to obtain access, visualize relevant anatomy, advance large devices, address aortic branches, cannulate the contralateral gate, insert pressure sensors, manage endoleaks, and exit upon completion.

ENDOGRAFT SELECTION

Successful aneurysm exclusion necessitates an understanding of the intricacies of the available grafts for proper preoperative selection. At present, the Cook Zenith Flex®, Endologix Powerlink®, Gore Excluder®, Medtronic AneuRx®, and Medtronic Talent® comprise the majority of devices available for use in the United States for primary aneurysm repair, while the Cook Renu Converter® and Medtronic Talent Converter® are FDA-approved for secondary conversion to an aortouniiliac device. Characteristics of these endografts are listed in Tables 27–1 and 27–2, and several caveats to device delivery are noted in the section on graft deployment.

ACCESS

Traditionally, femoral access has been obtained via common femoral artery exposure followed by arterial puncture under direct visualization. As with open reconstructions involving the femoral artery, two options exist for femoral incisions: vertical and transverse. Regardless of the skin incision, the fascia is incised vertically to allow femoral artery exposure. Both incisions allow optimal selection of the arterial puncture site, but the former allows easy extension of the incision for urgent iliac artery or infra-inguinal revascularization.

TABLE 27-1. FDA-APPROVED DEVICES FOR PRIMARY INFRARENAL AORTIC ANEURYSM REPAIR

Device	CT Measurement Method	Delivery Sheath Required	Minimum Access Diameter for Main Body	Minimum Access Diameter for Contralateral Limb	Treatable Aortic Neck Diameter	Minimum Aortic Length to Bifurcation	Minimum Aortic Neck Length	Maximum Aortic Angulation[1]	Suprarenal Fixation	Treatable Iliac Diameter	Minimum Iliac Seal Zone
Cook Zenith Flex	Outer Diameter	No	22-26mm graft = 7.1mm 28-32mm graft = 7.7mm 36mm graft = 8.5mm	8-10mm graft = 5.3mm 12-24mm graft = 6.0mm	18-32mm	22-32mm graft = 82mm 36mm graft = 95mm	15mm	<60 degrees, <45 degrees[2]	Mandatory	7.5-20mm	10mm
Endologix Powerlink	Outer Diameter	No	7mm	3mm	18-32mm	25-28mm graft = 80mm 34mm graft = 100mm	15mm	<60 degrees	Available	10-23mm	15mm
Gore Excluder	Inner Diameter	Yes	23-8.5mm graft = 6.8mm 31mm graft = 7.6mm	12-14.5mm graft = 4.7mm 16-29mm graft = 6.8mm	19-29mm	70mm	15mm	<60 degrees	Not Available	10-18.5mm	10mm
Medtronic AneuRx	Outer Diameter	No	7mm	5mm	16-26mm	80mm	15mm	<45 degrees	Not Available	10-22mm	25mm
Medtronic Talent	Outer Diameter	No	22-28mm graft = 7mm 30-36mm graft = 8mm	6mm	18-32mm	80mm	10mm	<60 degrees	Available	8-22mm	25mm

[1] Angulation of neck relative to long axis of aorta

[2] Angulation of suprarenal stents relative to long axis of aorta

TABLE 27-2. FDA-APPROVED DEVICES FOR SECONDARY INTERVENTION AFTER EVAR

Device	CT Measurement Method	Delivery Sheath Required	Minimum Access Diameter for Main Body	Treatable Aortic Neck Diameter	Minimum Aortic Length to Bifurcation	Minimum Aortic Neck Length	Maximum Aortic Angulation[1]	Suprarenal Fixation	Treatable Iliac Diameter	Minimum Iliac Seal Zone
Cook Renu Converter	Outer Diameter	No	22-26mm graft = 7.1mm 28-32mm graft = 7.7mm 36mm graft = 8.5mm	18-32mm[2]	22-32mm graft = 82mm 36mm graft = 95mm	15mm[3]	<60 degrees, <45 degrees[4]	Mandatory	7.5-20mm	10mm
Medtronic Talent	Outer Diameter	No	22-28mm graft = 7mm 30-36mm graft = 8mm	18-32mm	80mm	10mm	<60 degrees	Available	8-22mm	25mm

[1] Angulation of neck relative to long axis of aorta

[2] New graft must be oversized 2mm if placed within a pre-existing graft.

[3] Neck must be ≥10mm if new graft is placed within a pre-existing graft.

[4] Angulation of suprarenal stents relative to long axis of aorta

The latter, meanwhile, is believed by some to have lower morbidity. In a prospective randomized trial of patients undergoing vascular procedures who had no prior surgeries in the index groins, Swinnen et al demonstrated a lower complication rate with transverse incisions (47.5% versus 12.7%, $p<0.001$).[1] There were 13 (11%) wound infections in 116 groins by postoperative day 28, with 3 in patients with transverse incisions and 10 in patients with vertical incisions ($p=0.062$). Lymphatic leaks were present in 27.9% of wounds with vertical incisions, as opposed to 12.7% of those with transverse incisions ($p=0.044$). While the authors from this study observed a difference favoring transverse incisions, their overall wound complication rate was substantially higher than those reported by other series of femoral exposures for EVAR, which range from 2% to 2.8%.[2,3]

Percutaneous transfemoral access has been reported with two "pre-close" techniques, using either Prostar XL® or Perclose Proglide® closure devices. Both techniques involve blind or ultrasound-guided percutaneous access using the micropuncture technique. After ipsilateral oblique angiography via a 4 French (Fr) sheath to confirm common femoral access, a 0.035- inch wire is advanced, the micropuncture sheath is withdrawn, and a hemostat is used to dilate the entire tract. If the Prostar XL 10 Fr closure device is used, the device is advanced into the arteriotomy until pulsatile bleeding is noted from the marker port the two sutures are then deployed, retrieved, and left untied. The closure device is then exchanged for a large sheath or the endograft device. The same technique can be used for the contralateral side. If the Proglide 6 Fr closure device is used, the device is introduced into the arteriotomy until pulsatile bleeding is obtained from the marker port and deployed at a 45-degree angle; the suture is then deployed, retrieved, and left untied. The closure device is then exchanged for another Proglide device over a 0.035" wire, which is rotated 45 degrees from the midline in the opposite direction, deployed with the sutures left untied, and exchanged for the endograft device or a large sheath. Similarly, this technique can be used for the contralateral side. Regardless of the percutaneous technique used, the sutures are then placed on a shod clamp, ensuring no tension on the sutures during the case. The sutures and clamp may be covered with a sterile towel to ensure they do not become entangled in the subsequent wires, catheters, and endograft.

SHEATH DELIVERY

Currently available endografts require large femoral and iliac artery vessels to accommodate the ipsilateral and contralateral limb devices. Three options are available when hypoplastic, stenotic, occluded, or tortuous vessels preclude traditional femoral access for device delivery. This includes direct puncture, use of a conduit, controlled dilation, or controlled rupture of the artery.

First, the aorta and iliac arteries may be directly punctured proximal to any significant stenoses to allow device delivery. This may be done via a retroperitoneal approach with minimal dissection of the proposed access vessels. Without the need for circumferential arterial control, two purse-string sutures can be placed such that the sutures are begun and ended 180 degrees away from each other. A needle should be used to obtain access within the two sutures, which can be held by the operator and assistant during sheath exchanges and should facilitate easy closure of the artery.[4]

Second, retroperitoneal exposure can allow placement of a prosthetic conduit, sutured to the aortoiliac system, as dictated by preoperative computed tomographic angiography (CTA). Via a standard lower-quadrant oblique incision, the distal aorta or

common iliac artery may be exposed to allow an end-to-side anastomosis to be performed with a 10-mm prosthesis of the surgeon's preference. Placement of the conduit at a sharp angle with the native artery should be avoided; the conduit can be tunneled along the native artery to the groin to blunt the angle of approach. The suture line may be reinforced with graft material to reduce bleeding, and manual guidance of the sheath can minimize anastomotic disruption. A radiopaque marker can also be placed at the anastomosis to facilitate delivery sheath placement distal to the anastomosis and reduce exchanges across the suture line. The device delivery sheath and an additional small sheath may be placed through the end of the conduit, with vessel loops for hemostasis, allowing both device introduction and placement of a diagnostic angiography catheter. Alternatively, with the terminal portion of the conduit ligated, the side wall of the conduit can also be punctured for the diagnostic catheter and device delivery sheaths.[5]

Third, a variety of techniques can be used to dilate the iliac arteries and avoid retroperitoneal exposure. Primary iliac artery balloon angioplasty may suffice to allow passage of many sheaths. Alternatively, serial dilation with hydrophilic dilators may be employed. Another technique described by von Segesser et al involves insertion of a sheath smaller than what would be required for device delivery; afterwards, *in situ* balloon dilation of the sheath is performed to increase the diameter of the sheath, thereby allowing introduction and passage of a larger delivery device (Figure 27–1).[6] Yet another method involves creating a controlled rupture of the iliac artery with a

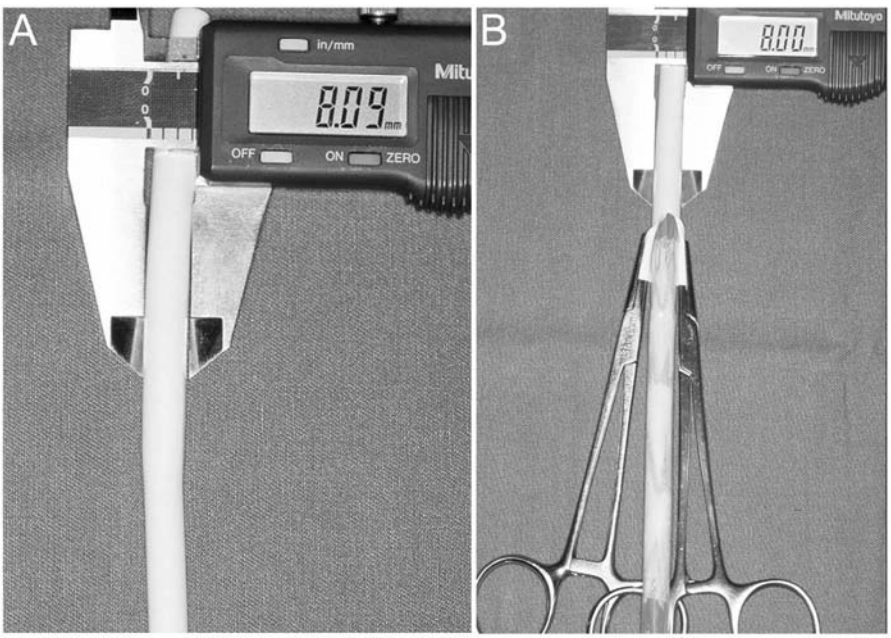

Figure 27-1. A. Photograph of an 18F sheath undergoing balloon dilation with an 8-mm balloon. Calipers measure the outer diameter at the site of balloon dilation to be 8.09 mm, which is equivalent to 23.4 F. The outer diameter of the undilated portion of the sheath measured 19.6 F. B. Photograph showing insertion of a 23.4 F covered stent graft system into the 18 F introducer sheath after balloon dilation of the sheath. Note the use of hemostats to secure the split sheath and allow for resistance to be applied against the sheath without tearing the sheath. (Reprinted from von Segesser LK, Marty B, Tozzi PG, Corno A. In situ introducer sheath dilatation for complex aortic access. European Journal of Cardio-thoracic Surgery, 22: pp. 316–318. Copyright 2002, with permission from European Association for Cardio-thoracic Surgery.)

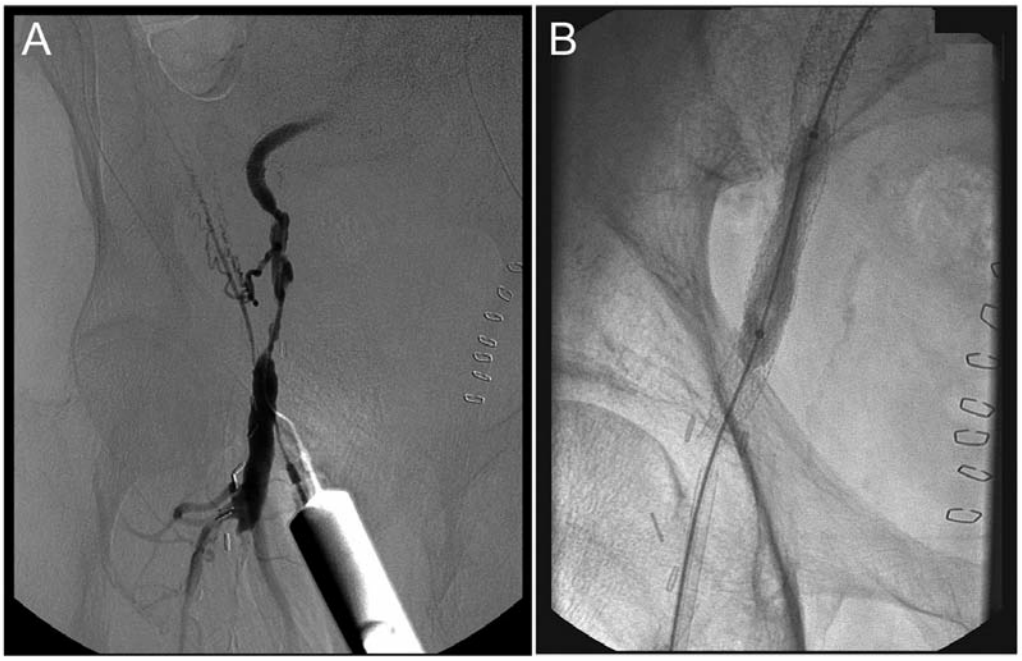

Figure 27-2. A. Angiogram of the right external iliac artery demonstrating a tight stenosis. B. Balloon angioplasty of a 13 mm covered stent graft (ie, endoconduit) in the same artery as depicted in A, thereby allowing subsequent passage of an 18 F sheath. (Reprinted from Peterson BG, Matsumura JS, Creative options for large sheath access during aortic endografting, Journal of Vascular and Interventional Radiology. 19: pp. S22–S26. Copyright 2008, with permission from The Society of Interventional Radiology.)

large covered stent to allow larger sheath delivery (Figure 27–2).[7] Most often, the internal iliac artery must be covered with this method, and pelvic ischemia can result. Endograft limbs may be used, but the Gore Viabhan® and Atrium iCAST® covered stents could also be used off-label for this technique.

MANAGEMENT OF LARGE BRANCHES

There are few large aortoiliac branches that must be addressed during endovascular repairs of infrarenal abdominal aortic aneurysms. These branches include lumbar arteries, the inferior mesenteric artery, internal iliac arteries, and accessory renal arteries, and they may be addressed by occlusion or preservation.

Although most type II endoleaks in the setting of stable aneurysms are currently treated expectantly, there is some controversy regarding preoperative and intraoperative branch occlusion.[8] Patent inferior mesenteric arteries and large lumbar arteries, which are potential sources of type II endoleaks, may be occluded; likewise, hypogastric arteries that must be covered for an adequate distal seal may be occluded. Typically, transarterial embolization is performed with a combination of platinum and stainless steel coils placed at the origin of these branches to preserve collateral flow. With the "anchoring technique," a catheter is placed in a smaller branch of the vessel in question; as the coil is released, the catheter and coil are pulled back into the larger

branch close to its origin. This allows fixation of the coil with proximal branch embolization. Meanwhile, with the "scaffolding technique," a large coil is placed at the branch vessel origin and smaller coils are packed within the large coil to avoid distal embolization. The Amplatzer II plug® is another device used for transarterial embolization and similarly allows controlled occlusion of both small and large branches. If both internal iliac arteries must be occluded, staged embolization may allow collateral formation and reduce pelvic ischemia.

More proximally, if faced with a marginal neck length or significant accessory renal artery, the "chimney technique" may be used to accomplish endovascular aortic aneurysm repair. Via a brachial or axillary approach, the renal artery in question is accessed. A covered stent is then deployed, with the stent extending into the aorta in a cranial direction. A standard EVAR is then performed, with care to dilate the renal artery conduit and aortic graft simultaneously to preserve renal perfusion and achieve an adequate proximal aortic seal.[9]

Custom-made fenestrated and branched endografts may also be employed to preserve larger aortic branches, including but not limited to the renal arteries, mesenteric vessels, and internal iliac arteries. However, as deployment is complex, individualized, and currently investigational, technical details are herein omitted.

GRAFT DEPLOYMENT

While the graft deployment steps vary based on the endograft selected, there are many common features and few differences that are worthy of mention. First, determination of the side of main body and contralateral leg delivery is dependent on access vessel diameter, iliac artery tortuosity, main body length, and iliac artery diameter. Clearly, if an iliac artery stenosis is present, delivery of the large main body through the larger side would carry less risk of iliac artery injury. While placement of the main body through a tortuous iliac artery could potentially be difficult, tortuousity could also make wire and catheter management during access of the contralateral gate tricky, as stored energy prevents extracorporeal wire manipulation from being translated into one-to-one intravascular movement. Additionally, as limited combinations of aortic diameters, iliac artery diameters, and endograft lengths exist, device delivery involves matching vessel size with endograft availability.

Second, deployment close to the renal arteries is preferred, but the grafts should be placed within a neck of relatively uniform diameter, with reversed taper necks offering the most challenge. Currently, the Medtronic Talent® device only requires a 10mm-long neck meeting this criterion, while the others all require at least 15mm for the proximal neck length.

Third, aortic angulation, while usually not a problem, must be considered before graft selection and deployment. The Medtronic AneuRx® is contraindicated in aortas with more than 45 degrees of angulation, while all other endografts are contraindicated with necks with more than 60 degrees of angulation. Additionally, the Cook Zenith Flex® is not recommended when the suprarenal stent would be located within an aortic segment with more than 45 degrees of angulation.

Next, graft selection and deployment usually requires consideration of aortic length, aortic diameter at the level of the contralateral gate, the narrowest aortic diameter, position of the iliac arteries, and the iliac artery length. Currently, the Gore

Excluder® has the shortest body, allowing the contralateral gate to be opened 7cm below the top of the covered stents. Most often, however, long main bodies are preferred, as opening the contralateral gate in proximity to the common iliac artery orifice facilitates gate cannulation. Another factor in determining the main body length is the aortic diameter, both at the site of the contralateral gate and the narrowest point, as the diameter must be sufficient to prevent limb constriction. Circumferential calcification along narrow segments is particularly troublesome, as it often prevents graft expansion. Additionally, depending on iliac artery position and length, the main body may be rotated to facilitate contralateral gate cannulation or slightly shorten the iliac limbs.

Lastly, all but one of the endoprostheses available in the United States are bifurcated modular grafts that require placement of a contralateral limb. Free-style cannulation of the contralateral gate is most often performed with an angled catheter and wire. When this fails, a selective catheter placed from the ipsilateral limb can be used in combination with a snare to obtain wire access into the contralateral gate. Similarly, brachial access can be used to ensnare a wire into the contralateral gate. If difficulty with contralateral gate cannulation is anticipated, the Endologix Powerlink® graft may be used, as it employs a bifurcated unibody design that is deployed, pulled down onto the aortic bifurcation, and then modified with proximal and distal extensions, if needed.

INTRAVASCULAR ULTRASONOGRAPHY

While conventional angiography is essential for endovascular aneurysm repair, intravascular ultrasonography (IVUS) can be a helpful adjunct for graft selection, deployment, and further interrogation. For most aortic procedures, 8 to 15 MHz transducers are selected, with lower frequencies offering the ability to view the entire artery while sacrificing resolution. After obtaining access to both groins, the IVUS catheter can be used to mark the location of the renal arteries, aortic bifurcation, and hypogastric arteries, and measure the diameters of the proximal aorta, aortic bifurcation, and iliac arteries (Figure 27–3). If performed over a flexible wire, proximal neck and iliac artery lengths can be determined before a stiff wire alters the anatomy. With this, contrast angiography and significant fluoroscopy can be avoided until the main body is positioned for deployment. The contralateral gate can then be cannulated with IVUS, further reducing radiation and contrast administration. Finally, the entire endograft can be interrogated for endoleaks and apposition to the arterial wall.[10]

PRESSURE SENSORS

Computed tomographic angiography is currently the gold standard for EVAR surveillance. Current practice guidelines suggest postoperative imaging at 1, 6, and 12 months during the first year and annually thereafter, if the patient remains asymptomatic and no other concerning findings on physical examination or prior imaging exist. However, in order to reduce contrast and radiation exposure, investigations are being performed on noninvasive remote aneurysm sac pressure measurements, specifically using the Impressure AAA Sac Pressure Transducer® and CardioMems Endosure Wireless AAA Pressure

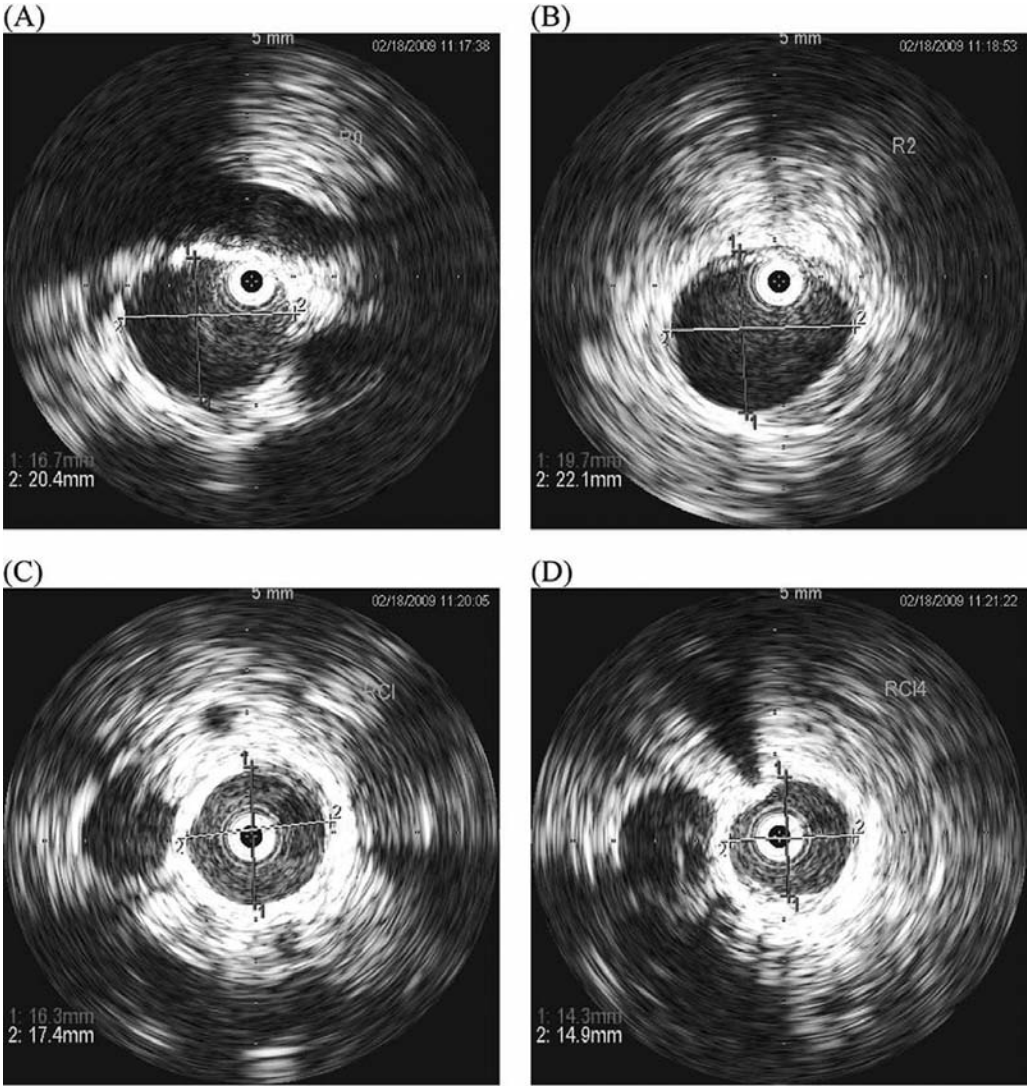

Figure 27-3. Intravascular ultrasound images obtained from the aorta demonstrating A. the position of the lowest renal artery (R0) and the left renal vein position at the top of the image; B. the distal extent of the proximal landing zone 2 cm below the renal arteries (R2); C. the right common iliac artery (RCI); and D. the diameter of the distal common iliac artery (RCI4). (Reprinted from Pearce BJ, Jordon WD. Using IVUS during EvAR and TEVAR: Improving patient outcomes. Seminars in Vascular Surgery. 22: pp. 172–180. Copyright 2009 with permission from Elsevier.)

Sensor® (Figure 27–4). The former must be sutured to the endograft and repackaged for deployment, and thus has fallen out of favor. The latter, meanwhile, requires transcatheter placement through a 14 Fr delivery device into the aneurysm sac after main body deployment. The sensor is interrogated, the contralateral limb is deployed, the sensor pressures are measured again, and then the sensor is finally deployed. Despite the paucity of evidence demonstrating device durability and efficacy, preliminary studies have demonstrated promising short-term results.[11–13]

Figure 27-4. Image of an implantable EndoSureTM wireless aneurysm pressure sensor measuring 30 × 5 × 1.5 mm (CardioMEMS, Atlanta, GA). (Reprinted from Okhi T, Ouriel K, Silveira PG, Katzen B, White R, Criado F, and Diethrich E. Initial results of wireless pressure sensing for endovascular aneurysm repair: The APEX Trial – acute pressure measurement to confirm aneurysm sac EXclusion. Journal of Vascular Surgery. 45: pp. 236–242. Copyright 2007, with permission from The Society for Vascular Surgery.)

ASSESSMENT AND MANAGEMENT OF EARLY ENDOLEAKS AND COMPRESSED LIMBS

After the graft deployment and inflation of a molding balloon, digital subtraction angiography or IVUS are used to assess for endoleaks. Some type I endoleaks can be managed with simple angioplasty or proximal or distal graft extensions, when possible. Additionally, bare stents can be used to juxtapose the endograft to the arterial wall. Most early type II endoleaks are managed with initial observation, with prompt return for reintervention for those who are symptomatic or who have enlargement of the aneurysm sac. Type III endoleaks can be treated by relining the segment of graft fracture or separation. Mild external compression of the contralateral limb may be treated with angioplasty, with selective stent placement. Alternatively, compressed limbs, inaccessible contralateral gates, and persistent endoleaks can sometimes be treated with a hybrid conversion, combining an aortouniiliac converter device (Table 27–2) with a femoro-femoral bypass. Finally, for those complications that cannot be managed with or fail observation, endovascular or hybrid management, open conversion with explantation of the endoprosthesis and traditional open repair can be performed.

EXIT STRATEGY

After confirming that no significant endoleaks or flow-limiting stenoses are present, the delivery devices and sheaths should be withdrawn while maintaining wire access. If a precipitous change in hemodynamics occurs or a segment of artery is withdrawn with the sheath (ie, "iliac-on-a-stick"), catheters can readily be passed for diagnostic imaging and occlusive balloons can be inflated for hemostasis. If needed, arterial injuries can then be addressed under a more controlled situation.

Arteriotomy closure depends on the initial approach for access. If femoral access was obtained via a traditional open approach, a standard arteriotomy repair should be performed, with subsequent layered closure of the wound. Alternatively, if the "preclose" technique was utilized, manual pressure is maintained proximally while the wounds and sutures are rinsed free of thrombus and the tract is confirmed to be fully dilated. The sutures are then tied, with the knots carefully pushed down to, but not

into, the artery. The wire should be removed before tying the second suture. Despite successful closure, a brief period of manual compression may be required. If significant hemorrhage persists, emergent femoral artery repair should be performed. If a conduit is sutured to the aortoiliac system to facilitate device delivery, it may be ligated proximally or preserved for an aortofemoral or iliofemoral bypass. The limb of the conduit can also be preserved for later use if re-intervention is needed. Jon Matsumura described this as a "buried treasure," and would typically leave a long conduit limb that could be readily accessed via a small left lower-quadrant retroperitoneal incision following thrombectomy.

Lastly, a lower extremity pulse examination should be performed to exclude acute ischemic changes. Ankle-brachial indices and Doppler signals may be obtained on the operating table and compared to preoperative assessments as objective adjuncts to the clinical examination. Any significant issues can thereby be addressed immediately.

CONCLUSION

Endovascular aneurysm repairs require extensive preoperative preparation for not only graft selection but also for optimal deployment. One must consider the method of access, device delivery, management of large branches, graft orientation, and contralateral limb deployment. Once deployed, the graft must be assessed for endoleaks and limb compression before arteriotomy and wound closure. Failure to understand these EVAR techniques and adequately plan for each step can result in endoleaks, visceral malperfusion, and extremity ischemia, precipitating the need for emergent complex open repair.

REFERENCES

1. Swinnen J, Chao A, Tiwari A, Crozier J, Vicaretti M. Vertical or Transverse Incisions for Access to the Femoral Artery: A Randomized Controlled Study. *Ann Vasc Surg*. 2009;1–6.
2. Rachel ES, Bergamini TM, Kinney EV, Jung MT, Kaebnick HW, Mitchell RA. Percutaneous Endovascular Abdominal Aortic Aneurysm Repair. *Ann Vasc Surg*. 2002;16:43–49.
3. Slappy ALJ, Hakaim AG, Oldenburg WA, Paz-Fumagallo R, McKinney JM. Femoral incision morbidity following endovascular aortic aneurysm repair. *Vasc Endovasc Surg*. 2003;37: 105–109.
4. Carpenter JP. Delivery of Endovascular Grafts by Direct Sheath Placement into the Aorta or Iliac Arteries. *Ann Vasc Surg*. 2002;16:787–790.
5. Peterson. BG, Matsumura JS. Creative Options for Large Sheath Access during Aortic Endografting. *J Vasc Interv Radiol*. 2008;19:S22–S26
6. von Segesser LK, Marty B, Tozzi PG, Corno A. In situ introducer sheath dilatation for complex aortic access. *J Cardio Thor Surg*. 2002;22:316–318.
7. Peterson. BG, Matsumura JS. Creative options for large sheath access during aortic surgery. *J Vasc Surg*. 2008;47:441–445.
8. Jonker FHW, Aruny J, Muhs B. Management of type II endoleaks: preoperative versus postoperative versus expectant management. *Semin Vasc Surg*. 2009;22:165–171.
9. Ohrlander T, Sonesson B, Ivancev K, Resch T, Dias N, Malina M. The chimney graft: a technique for preserving or rescuing aortic branch vessels in stent-graft sealing zones. *J Endovasc Ther*. 2008;15:427–432.
10. Pearce BJ, Jordon WD. Using IVUS during EVAR and TEVAR: Improving Patient Outcomes. *Semin Vasc Surg*. 2009;22:172–180.

11 Ellozy SH, Carroccio A, Lookstein RA, Minor ME, Sheahan CM, Juta J, Cha A, Valenzuela R, Addis MD, Jacobs TS, Teodorescu VJ, Marin ML. First experience in human beings with a permanently implantable intrasac pressure transducer for monitoring endovascular repair of abdominal aortic aneurysms. *J Vasc Surg*. 2004;40:405–412.
12 Hoppe H, Segall JA, Liem TK, Landry GJ, Kaufman JA. Aortic aneurysm sac pressure measurements after endovascular repair using an implantable remote sensor: initial experience and short-term follow-up. *Eur Radiol*. 2008;18:957–965.
13 Ohki T, Ouriel K, Silveira PG, Katzen B, White R, Criado F, Diethrich E. Initial results of wireless pressure sensing for endovascular aneurysm repair: The APEX Trial – Acute Pressure Measurement to Confirm Sac EXclusion. *J Vasc Surg*. 2007;45:236–242.

28

Percutaneous Approach for EVAR

Mark D. Morasch, M.D.

Endovascular aneurysm repair (EVAR) continues to be refined as patient selection, endovascular techniques, and devices improve. Completely percutaneous EVAR is now feasible at many institutions and has small but significant benefits compared to open cutdowns.[1–4] This technique requires familiarity with suture-mediated closure devices, devices utilizing relatively small introducer sheaths and predictably short procedure times.[5] This article will detail specific techniques, review procedural strategies, and summarize the clinical benefits.

PREOPERATIVE SELECTION

Thin collimation CT with three-dimensional reconstruction provides the required information to determine anatomic suitability for percutaneous EVAR. This includes the length and diameter measurements for endograft selection, but also assessment of the iliofemoral access arteries. Specific attention is directed to iliofemoral artery size, tortuosity, calcification, and the specific location of the femoral bifurcation. Percutaneous access is now planned in all patients, but early in our experience, we avoided patients with small calcified iliac arteries, previous femoral dissection, recent use of closure devices, and significant lymphadenopathy. These anatomic challenges require adjustments during the placement of the sheaths and closure devices so as to minimize the chance of conversion to open cutdown.

Pre-close

Suture-mediated closure devices are available in a range of sizes, but for Excluder, the sheath size required is 18 French on the ipsilateral and 12 French on the contralateral side. These are larger than the approved 10 French size, so the sutures are placed prior to enlarging the arteriotomy, and this "pre-close" technique is an off-label use of the closure device. Specific devices for suture-mediated large-vessel closure are under development.

Detailed Techniques

It is essential that anterior puncture of the common femoral artery be performed and verified. Specifically, high punctures are less likely to be hemostatic, and low punctures may result in closure of the superficial femoral artery. High punctures that pass through the inguinal ligament may temporarily control the access site bleeding as the tendon functions as a buttress or pledget, but with patient mobilization, the sutures lose tension and patients may develop large, late false aneurysms. Puncture may be guided with ultrasound, fluoroscopy, or digital localization. Puncture location is confirmed by sheath injection arteriography with an ipsilateral oblique view. The skin and subcutaneous tissue is stretched with a spreading motion of a clamp to allow the Prostar XL, 10 French (Abbott Medical Devices, Redwood City, CA) to be exchanged over a guide wire. Alternatively, a subcutaneous tract may be bluntly dissected with a small finger so that anterior arterial puncture can be confirmed by direct palpation. The current version of the Prostar XL is monorail and the guide wire can be removed to avoid enlarging the arteriotomy. Occasionally, in tortuous anatomy, wire exchange for the closure device may result in loss of retrograde selection of the infrarenal neck, which may result in the tip of the closure device curling in the aneurysm sac.

After confirmation of arterial flow through the marker lumen, the barrel is aligned. The proper amount of tension is maintained on the shaft so the artery is not compressed and then the needles are deployed, thus placing the sutures adjacent to the arteriotomy only in the anterior arterial wall. If there is significant resistance to deploying the needles, a "backdown" maneuver may be performed and the device readjusted or exchanged. The free ends of the sutures are tagged, slack is removed, and sutures are soaked with heparinized saline to prevent thrombus formation during the rest of the procedure.

Guide wire access is regained and the larger sheath is then inserted. Occasionally, the infrarenal neck must be reselected or a catheter used to exchange for a stiffer wire. At this point, a small dose of intravenous heparin may be given. If there is good flow in the external iliac artery around the sheath and the procedure is expected to be short, systemic heparin may be avoided. The endograft main trunk is positioned and deployed, the contralateral leg hole cannulated, and the contra limb deployed with maximum overlap.

After completion arteriography is reviewed and found to be acceptable, the sutures are thoroughly irrigated and wiped of any thrombus. The sutures are tied with sliding knots. And, one at a time, the knots are slid down to below the skin level. The sheaths are then removed and the knots cinched down to the artery wall. The sutures are trimmed as short as possible. Often, a brief period of compression is needed to stop suture hole bleeding. The small wound is closed with a single subcutaneous suture and a single subcuticular suture with knots buried.

Strategies

Different strategies are necessary depending on the experience of the team and availability of facilities for immediate open cutdown. The approach we use is to treat every endovascular repair with the percutaneous technique and convert immediately to an open cutdown if there is an issue with bleeding, stenosis, or femoral artery injury. This is feasible because we have access to an excellent fixed imaging unit present in the operating room.

A second strategy is to select out cases where percutaneous repair is likely to be uncomplicated and perform a cutdown on all potentially problematic femoral

TABLE 28-1. COMPARISON OF PERCUTANEOUS ACCESS AND CUTDOWNS

	Cutdown (n=35)	Percutaneous (n=47)	P
Anesthesia Time (minutes)	225	201	.008
Procedure Time (minutes)	169	139	.002
General Anesthesia	32	28	.003
Hospital LOS (days)	1.89	1.49	.411

arteries—this alternative may be used when preferred imaging and the operating room environment are not present in the same location. About 70% of patients suitable for endovascular repair are candidates for predictably uncomplicated bilateral percutaneous closure.

Results

Early in the experience, complications occurred related to operator error, periarterial scarring, and full anticoagulation. After mastering technical proficiency with the devices and absolutely insisting on anterior common femoral artery puncture, percutaneous repair under local anesthesia has become a routine option. Patients are able to ambulate immediately after the procedure and have short recovery times. No late pseudoaneurysm, stenosis, or infection has been identified in these patients. The latter—risk of arterial infection—is dreaded, and meticulous aseptic technique has been practiced.

Comparative data of the use of percutaneous access have been published.[6] Forty-seven patients with bilateral percutaneous access were compared to 35 patients with femoral cutdown. There is a reduction in use of general anesthesia in the bilateral percutaneous group compared to the cutdown group. Further, procedure time and anesthetic time are significantly shorter using the percutaneous technique (Table 28–1). When considering the strategy of attempted bilateral percutaneous access in all patients, intraoperative conversion to cutdown occurs in less than 15% of patients and postoperative wound, femoral neuropathy, and vascular complications are significantly reduced compared to routine open cutdown.

Nevertheless, caution is warranted because substantial complications may occur. Similar to open cutdown, clinicians should monitor for arterial occlusion and bleeding not controlled by additional external pressure. These are easily addressed when identified in the operating room suite. Late complications are rare, and the most formidable may be suture infection that often requires aggressive surgical treatment.

SUMMARY

Randomized clinical trials have demonstrated superior short-term results with lower 30-day mortality rates.[7,8] Percutaneous EVAR under local anesthesia is feasible in most patients. Elements of success include an appropriate strategy for arterial access, familiarity with the technical nuances of the closure system, and the use of endoprostheses with a predictably short procedure time and smaller access sheaths. Greater benefits are expected with newer closure devices and improved development of specific percutaneous techniques.

REFERENCES

1. Rachel ES, Bergamini TM, Kinney EV, et al. Percutaneous endovascular abdominal aortic aneurysm repair. *Ann Vasc Surg*. 2002;16:43–49.
2. Howell M, Villareal R, Krajcer Z. Percutaneous access and closure of femoral artery access sites associated with endoluminal repair of abdominal aortic aneurysms. *J Endovasc Ther*. 2001;8:68–74.
3. Traul DK, Clair DG, Gray B, et al. Percutaneous endovascular repair of infrarenal abdominal aortic aneurysms: a feasibility study. *J Vasc Surg*. 2000;32:770–6.
4. Kibbe MR, Evans ME, Morasch MD. Percutaneous repair of abdominal aortic aneurysms. In Pearce W, Matsumura J, Yao J, eds. *Trends in Vascular Surgery*. 2003. Chicago: Precept Press, 2004.
5. Matsumura JS, Brewster DC, Makaroun MS, et al. A multicenter controlled clinical trial of open versus endovascular treatment of abdominal aortic aneurysms. *J Vasc Surg*. 2003;37: 262–271.
6. Morasch MD, Kibbe MR, Evans ME, et al. Percutaneous repair of abdominal aortic aneurysms. *J Vasc Surg*. 2004;40:12–16.
7. Prinssen M, Verhoeven EL, Buth J, et al. A randomized trial comparing conventional and endovascular repair of abdominal aortic aneurysms. *N Eng J Med*. 2004;351:1607–1618.
8. Greenhalgh RM, Brown LC, Kwong GP, et al. Comparison of endovascular aneurysm repair with open repair in patients with abdominal aortic aneurysm (EVAR trial 1), 30-day operative mortality results: randomized controlled trial. *Lancet*. 2004;364:843–848.

29

Pressure Sensor Use with Endovascular Aneurysm Repair

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Nasim Hedayati, M.D.

Endovascular aneurysm repair (EVAR) is intended to eliminate the risk of aneurysm rupture, the significant and usually fatal complication of an abdominal aortic aneurysm (AAA). It has long been understood that EVAR protects against AAA rupture by excluding the wall of the aneurysm from systemic arterial pressure.[1] The presence of an endoleak after EVAR may result in continued pressurization of the aneurysm sac, with the potential for continued expansion or rupture of the aneurysm (Table 29–1). By measuring the pressure within the excluded aneurysm, the success of the EVAR can be assessed.

MEASUREMENT OF INTRA-AORTIC PRESSURE

Direct pressure measurements of intra-aortic pressure can be made using a catheter connected, via fluid-filled tubing, to an external transducer, or with a pressure transducer incorporated into the tip of a small diameter (0.014 inch) guidewire.[2]

Clinical studies with direct measurements of the pressure within the AAA sac, outside of the endograft, have confirmed the seemingly obvious assumption that intraluminal pressure within the aneurysm sac is higher when an endoleak exists. Baum et al. reported endoleaks were associated with elevated and pulsatile arterial pressure in the sac.[3] They measured pressures with catheters placed into the aneurysm sac via one of three different routes: 1) translumbar puncture; 2) microcatheter access through a patent inferior mesenteric artery (IMA), placed via selective superior mesenteric artery catheterization; or 3) placement of a peri-graft catheter during the EVAR procedure. Elevated sac pressures were confirmed in 17 patients with CT scan demonstrated endoleaks. The sac pressure in patients with endoleaks was equal to the systemic pressure (15 patients) or nearly equal to (two patients), and all were observed to have pulsatile pressure waveforms. Elevated intrasac pressures can exist without an endoleak seen on CT or angiography. Elevated sac pressures were found in two

TABLE 29-1. ENDOLEAK TYPES

Type		Pathophysiology	Comments
I	Failure to obtain seal	Incomplete proximal fixation Incomplete distal fixation	All require correction, either with secondary intervention or conversion to open AAA repair.
II	Retrograde flow from branch(es) of the aorta	Inferior mesentery artery Lumbar artery(ies)	Intervention recommended for very large aneurysms or if aneurysm sac size increasing.
III	Graft defect	Separation or dysjunction of modular components hole in graft fabric	Management required in all cases, as the effect is similar to Type I endoleak.
IV	Graft porosity		Largely a problem with now obsolete graft materials. Usually subside early; no specific treatment required.
V	Endotension		Elevated pressure within the aneurysm sac, without identified flow.

patients without CT or angiographic evidence of endoleak, but direct contrast injection into the aneurysm sacs in these patients revealed a patent lumbar artery in one and a patent IMA in the other. These cases probably represent examples of Type II endoleaks with flow rates too slow to be detected with conventional imaging techniques. Experimental models have suggested that sac pressure may be high in the presence of any endoleaks, even small ones.[4]

Experiments with ex vivo flow models suggest that every endoleak, even a small one, will result in a pressure greater than systemic diastolic pressure within the aneurysm sac. Small endoleaks, however, may not be visualized with digital subtraction angiography or arterial phase CT angiography.[5] Different pressure waveform characteristics may be observed with successful exclusion and with different types of endoleaks.[6] In a flow model with a graft and no endoleak, mean and pulse pressures are reduced to very low values. When a Type I endoleak was introduced to the experimental model, mean sac pressure was similar to mean aortic pressure. When there was net flow through the sac due to a Type II endoleak, mean sac pressure was a function of the inlet pressure, while pulse pressure in the sac was dependent on both inlet and outlet pressures. As the perfusion rate was increased, both mean and pulse sac pressures decreased. When there was no outflow, mean sac pressure with a Type II endoleak was similar to mean aortic pressure. These pressure relationships may be variable, though with different patterns and values seen with aneurysms of different size.[7]

WIRELESS PRESSURE SENSOR TECHNOLOGIES

Catheter and pressure wire measurement techniques have value as research tools, but are not considered feasible for routine intraoperative use. Postoperative use is possible, but only practical when a problem has already been demonstrated and intervention is being

considered. As an alternative, technologies have been developed that allow implantation of a sensing device that can transmit pressure data wirelessly from within the aneurysm sac to an external monitor.

Radiofrequency (RF)

At present, there is only one commercially available device for measurement of intra-aortic pressure after endograft placement. The implantable EndoSure™ Wireless AAA Pressure Measurement System (CardioMEMS, Inc., Atlanta, GA) was FDA approved in 2005 and it is currently in use clinically (Figure 29–1). This sensor is externally powered by radiofrequency (RF) energy.

The EndoSure™ wireless pressure sensor is an example of a MEMS device. MEMS (micro electromechanical systems) are integrated systems combining both electrical and mechanical components, generally fabricated on a micron scale. The implanted sensor has a hermetically sealed circuit encapsulated in fused silica and silicone. Pressure changes induce small changes in a flexible membrane capacitor plate; the change in capacitance changes the sensor's resonant frequency. The internal circuitry uses this to measure pressure. The sensor operates over the physiologic range of arterial blood pressure, with a resolution of 1 mm Hg.

The device requires no battery for operation. An external antenna emits the RF energy that induces a current in a coil in the implanted device and this current is used by the processor. The implanted device emits a signal that is picked up by the external antenna signal. The external processor analyzes the signal, displays pressure waveforms, and stores the results to an external flash memory card (Figure 29–2).

Ultrasound

An alternative technical approach for a wireless, implantable, pressure sensor is to employ ultrasound (rather than RF energy) to both energize and communicate with the implanted device. By use of an appropriate wavelength, acoustic communication uses little energy to

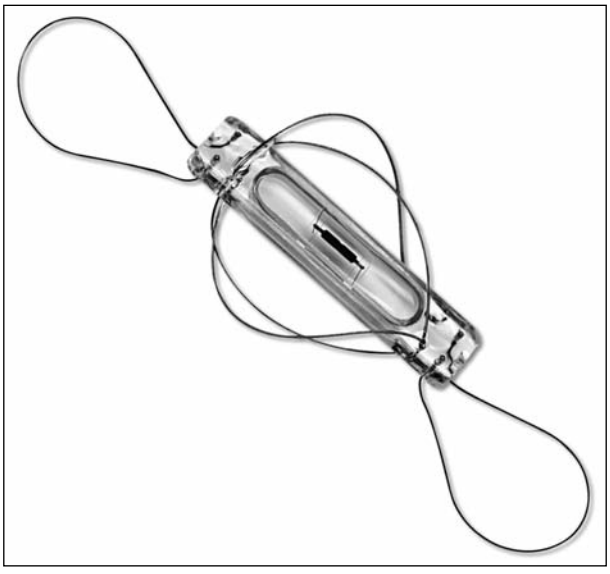

Figure 29-1. Implantable CardioMEMS EndoSure™ sensor. The fused silica sensor package and copper circuitry are coated with medical grade silicone. The nitinol wire loops help to stabilize the device within the aneurysm. The device measures 2 mm x 4.5 mm x 21 mm.

Figure 29-2. CardioMEMS interrogator 2™ monitor system and display with handheld antenna attached by cable. When used in the operating room, the racquet-shaped antenna is sheathed in a sterile plastic sleeve.

achieve a high signal-to-noise ratio when accessing locations deep in the body. A prototype ultrasound-powered implant, the ImPressure™ system was developed by Remon Medical Technologies (Caesaria Industrial Park, Israel). It is a minute device, just a bit bigger than a grain of rice, requiring no antenna, battery, or connecting leads. The implant contains a piezoelectric chip to exchange energy, a control circuit, and a sensor, all enclosed in a miniature casing. Similar to the RF sensor technology, there is an external unit to power the implant and receive information from the implanted sensor. Because the device transducer operates at a low resonance frequency, it is omni-directional, relatively insensitive to the positioning of the external transducer.

The small size of the ultrasound-powered sensor has allowed it to be attached to endograft components, to be introduced with the endograft implantation. This technology was tested in a small European study that measured intrasac pressure after EVAR, and found concordance between catheter-derived and transducer-derived pressure measurements.[8] The ultrasound-activated transducer system, however, has not yet been commercialized in the United States. The company that developed the system was acquired by Boston Scientific Corp (Natick, MA) in 2007.

IMPLANTATION

The FDA-approved RF sensor is implanted concomitant with EVAR. Several modifications of implantation techniques can be used, but the standard approach is to introduce the sensor after the main body of a bifurcated endograft is deployed. In the case of a unibody device, the sensor is introduced after the graft is positioned, just prior to deployment. The sensor is delivered through a sheath contralateral to the main body, at least 14 Fr inner diameter. After the contralateral graft limb is accessed with a stiff guidewire, a 0.035" buddy wire is introduced through the sheath, into the aneurysm sac. The sensor is mounted on a delivery catheter that is advanced through the sheath over the guidewire, allowing the sensor to be appropriately positioned in the aneurysm sac. The sensor is calibrated and an initial pressure measurement is recorded. After deployment of the contralateral limb, the sensor is released from the delivery catheter with the pull of a small trigger wire.

The external antenna is used to acquire the signal from the sensor. This can be covered with a sterile sheath for intraoperative use. For optimal reception, the antenna

is oriented parallel to the flat surface of the sensor. A good signal is identified when the signal strength is =60% and the signal is pulsatile.

INTRAOPERATIVE MEASUREMENT

Pressure measurements within the sac of the excluded aneurysm complement the intraoperative information obtained with arteriography. An immediate decrease of the pressure in the aneurysm sac suggests successful aneurysm exclusion (Figure 29–3). An endoleak is suggested if the pulse pressure is not markedly decreased after endograft placement. The pressure information can be confirmatory, or it may sometimes be used without concomitant arteriography.

The RF device was approved for intraoperative use, based on the results of a clinical trial sponsored by the manufacturer.[9] The APEX trial (Acute Pressure Measurement to Confirm Aneurysm Sac Exclusion) was a prospective, multicenter study that evaluated the safety and efficacy of the wireless pressure sensor for intraoperative use during EVAR. A total of 90 patients were enrolled at 12 sites, 76 of whom were eligible for analysis. The aneurysm sac pulse pressure was directly measured with an angiographic catheter and also by the sensor after deployment of the main endograft component, but before deployment of the contralateral limb. Sac pressure was again measured with the sensor after deployment of the contralateral limb and completion of the EVAR.

The initial sensor pressure measurements prior to aneurysm exclusion agreed closely with the angiographic catheter pressure measurements. At procedure completion, there was agreement between the sensor measurement and angiography regarding the presence or absence of a Type I or III endoleak in 92% of the measurements. Final pulse pressures decreased significantly compared with baseline measurements. Overall, the sensitivity was 94% and the specificity was 80% for detecting a Type I or III endoleak. No Type IV or Type V endoleaks were found. The APEX trial was completed in 2005, but there is a five-year ongoing surveillance study to evaluate the subjects for any long-term problems.

Because only six Type I and six Type III endoleaks were found, the APEX study was too small to allow reliable criteria to be established to identify and categorize clinically significant endoleaks. Despite the limitations of the data from the trial, they were sufficient to gain FDA approval for the device. Others have confirmed the correlation of the pressure measurements from the implanted wireless sensor to direct pressure measurements during EVAR procedures.[10] Additionally, there have been opportunities to gain additional (though unstructured) experience with intraoperative pressure measurements as several thousand sensors have been placed in clinical practice since the implantable sensor became commercially available.

Continued pulsatility within the aneurysm sac suggests an endoleak.[9,11] A pulse pressure index (intrasac pulse pressure/systemic pulse pressure) > 0.6 is probably due to a Type I endoleak (Figure 29–4). A pulse pressure index (PPI) between 0.3 and 0.6 may be associated with a type II endoleak. The type and location of an endoleak can often be determined without the need for additional contrast administration. For example, if the there is a Type II endoleak from inadequate distal fixation in an iliac limb, inflation of a balloon catheter in that limb will result in a drop in the sac pressure. Therapeutic maneuvers to correct the problem can be employed (e.g., additional balloon catheter inflations in the iliac graft limb, placement of a distal graft

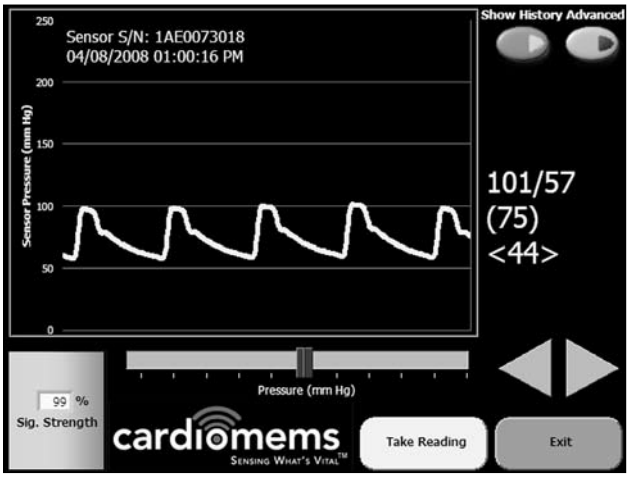

Figure 29-3A. The LCD screen of the interrogator displays the pressure waveform acquired from the implanted sensor. After the sensor is positioned in the aorta, but prior to complete deployment of the endograft, the displayed waveform should be nearly identical to that transduced from a conventional arterial pressure line. At this point, the pressure measured from the arterial line is used to calibrate the implanted sensor. Values displayed on the right side of the screen include the systolic and diastolic pressures, mean pressure, and pulse pressure.

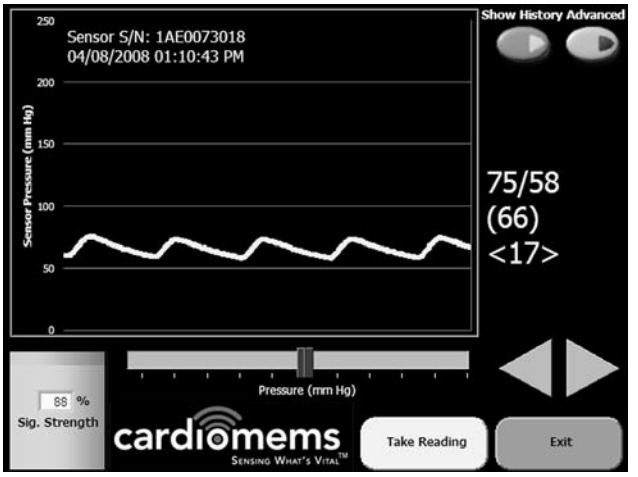

Figure 29-3B. After deployment of the endograft and limb components, a postexclusion reading shows a decrease in the measured systolic pressure and pulsatility (pulse pressure). This indicates the aneurysm sac has been at least partially excluded. The persistence of a pulsatile waveform may reflect an incomplete seal at the graft fixation or overlap sites.

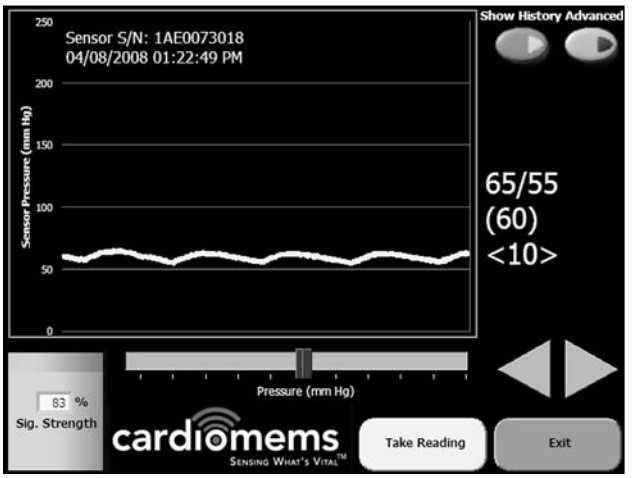

Figure 29-3C. The intraoperative drop in the pulse pressure reflects how effectively the endograft has excluded the aneurysm. A further decrease in pulsatility is observed after inflation of a balloon catheter to dilate the proximal and distal fixation points of the graft, improving the seal. Immediately after endograft deployment, however, the mean sac pressure is only modestly decreased, as the static blood volume within the sac was sealed off from the flow lumen while pressurized. During later follow-up evaluations, the mean pressure is typically low after successful aneurysm exclusion, as the thrombosed and fibrotic aneurysm sac does not remain pressurized.

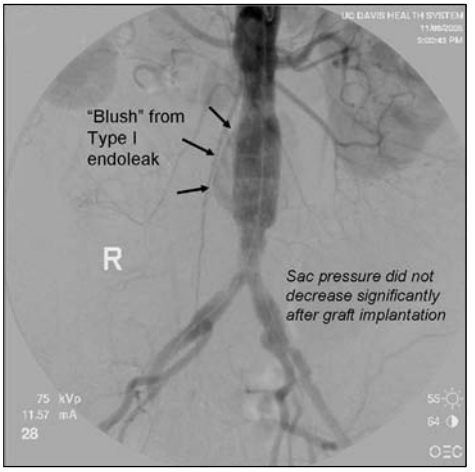

Figure 29-4A. Arteriography after deployment of a Powerlink (Endologix) graft shows a Type I endoleak, substantiated by the observation of a near systemic blood pressure in the aneurysm sac.

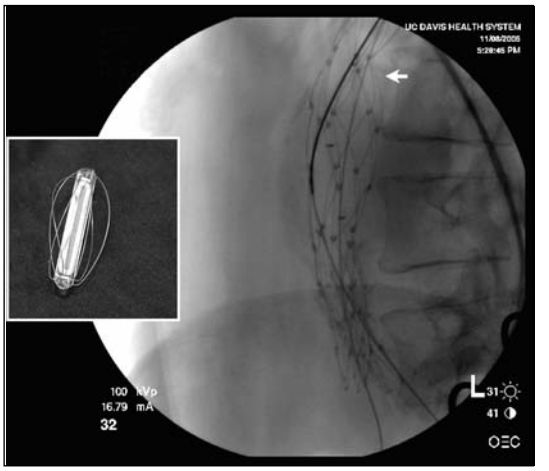

Figure 29-4B. Repeated balloon dilation of the proximal landing zone did not result in a decrease in the pulse pressure index, suggesting a persistent endoleak. Angiograms were not repeated between balloon inflations as there was not change in the measured intrasac pressure. A lateral projection showed a posterior fold in the aortic neck, which interfered with a proximal seal.

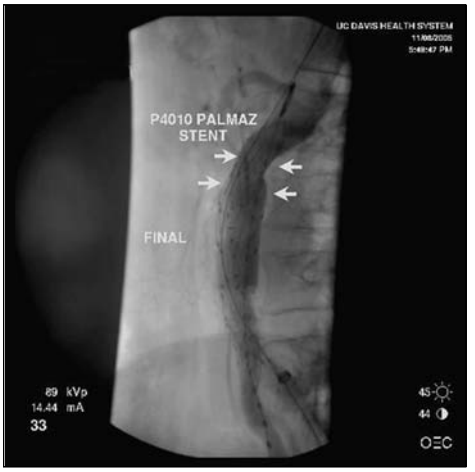

Figure 29-4C. A seal was obtained after deployment of a balloon-expandable stent in the proximal endograft. The pulse pressure index dropped to 0.3 after stent placement and follow-up arteriography confirmed the resolution of the endoleak.

limb extension) and the pressure can then be remeasured. The sac pulse pressure will drop if the additional maneuvers were effective. Exclusion is likely complete if the PPI is <0.3. By using pressure measurements to localize the endoleak and to assess the results of additional interventions, the surgeon can decrease the number of arteriographic runs and the volume of contrast agent used.

POSTOPERATIVE SURVEILLANCE AFTER EVAR

Though it is established that surveillance is needed after EVAR,[12-14] the optimal protocol for postoperative surveillance has not been determined. Specific testing requirements and schedules for follow-up vary among surgeons and institutions. For the most part, these were derived from the protocols used in the preclinical trials of the endograft systems.

These protocols considered the clinical requirements (i.e., what was needed to safely care for the patient), but often included more frequent or comprehensive imaging requirements to allow the investigators and regulatory agencies to establish the long-term performance of the devices.

The optimal protocol for postoperative surveillance after EVAR should consider several factors. First, there is a need to detect early and late complications related to EVAR. Most of these will be related to endoleaks.[15] Post-EVAR patients with identified Type II endoleaks or other abnormalities may need more frequent reassessment. Those with complete exclusion and no evidence of endoleak at one month or one year after the procedure may need only annual follow-up.[14]

CT scanning was generally the default imaging modality, supplemented with plain radiographs to evaluate the position and integrity of the metallic components, though if high-resolution CT is available with the ability to perform three-dimensional reconstructions of the metallic components, there may not be a need for plain radiographs. MRI is used in some centers, but it is more costly and time-consuming, and many patients have implanted ferromagnetic devices or other contraindications to MR imaging.[12,13,16]

Ultrasound imaging has been used as an acceptable alternative to CT or MR imaging to assess the size of the aneurysm after exclusion, but the duplex scanning has not been consistently found to have high sensitivity for the detection of endoleaks.[17] The use of ultrasound contrast agents to enhance endoleak detection is one proposed solution to this shortcoming.[18-23] Complementing imaging with information about the pressure within the excluded aneurysm sac may be another way to avoid missing the diagnosis of post-EVAR endoleak.[24]

The addition of the physiologic information from pressure measurements affects decision-making about the frequency, type, and interpretation of post-EVAR imaging.[24] With a monitoring technique that is noninvasive and with very little additional cost with each measurement, surveillance pressure measurements can be done at any desired interval without the associated risks of radiation exposure and contrast administration with CT imaging[12,24] (Figure 29–5). Pressure measurements may help with assessment of postimplantation complications that are not detectable by imaging alone, such as endotension from a Type V endoleak.[25] Additionally, it has been suggested that simplifying postoperative surveillance requirements may improve patient compliance with surveillance protocols.

Pressure monitoring may provide corroborative information about the efficacy of secondary interventions. For example, successful embolization for Type II endoleak has been shown to decrease the pressure within the aneurysm sac.[26] The ability to continue to measure intra-aortic pressure after secondary interventions appears to have value, as pressure measurements of patients who have had secondary procedures to treat an endoleak may correlate with treatment success. Conversely, if there has been no change in intrasac pressure, additional diagnostic evaluations for endoleak sources should be considered.

PITFALLS WITH INTRA-AORTIC PRESSURE MEASUREMENT

One important limitation with the use of pressure measurements from an implanted sensor is that the pressure within the aneurysm sac may not be the same throughout the volume of the sac, whether or not there is an endoleak. After successful treatment of an aneurysm,

Figure 29-5. Noninvasive measurement of the pressure in the aneurysm sac may be used as routine part of postoperative surveillance, typically in combination with imaging. The pressure data can complement anatomic and flow information from ultrasound imaging, helping confirm effective and continued aneurysm exclusion (see Table 29–1).

the microstructure of the thrombus within the aneurysm is variable and this affects pressure transmission. Fresh thrombus may transmit pressure differently than old clot,[27] so observed pressures with the aneurysm sac may change over time. Pressure measured at a single location may not accurately reflect the pressure transmitted to the vessel wall.[28] Direct intra-aneurysm sac pressure measurements in patients with both Type I and Type II endoleaks has shown that pressure within the aneurysm sac may be near systemic if measured close to or in the location where flow exists, while the pressure is lower in the thrombus away from the nidus of the endoleak.[26] This variability has been termed "compartmentalization." Also, experiments with models suggest that physical characteristics of the aneurysm wall (e.g., wall stiffness) may affect subsequent pressure measurements.[29]

Other issues to consider include the long-term reliability of the implanted devices. The technologies appear dependable, but the long-term ability to reliably measure pressures has yet to be demonstrated.

FUTURE STUDIES

Protocols for post-EVAR follow-up have been adopted for clinical use (Table 29–2), but these are yet to be validated. For example, more data are needed to reliably distinguish normal from abnormal pressure patterns. It is not known what measures are best to use. Published studies have reported pressure data and pressure indices differently, and there is no consensus on threshold values for action (Table 29–3). Further, the pressure profiles may change over time as the aneurysm sac thromboses. Endoleaks of different types and locations may produce variable pressure patterns.

Though it has been established that aneurysm sac pressure is elevated in the presence of an endoleak, endograft deployment (even in the absence of an endoleak)

TABLE 29-2. SUGGESTED FOLLOW-UP AFTER ENDOVASCULAR ANEURYSM REPAIR (EVAR)

Routine Follow-up for Patients with Implanted Endoluminal Pressure Sensor		
Schedule	**Tests**	**Comments**
Intraoperative	Pressure check (calibration, pre- and postexclusion measurements) Angiogram	Intraoperative pressure measurements are recorded for later comparison
Postoperative day 1, prior to discharge	AP and lateral L-S spine x-rays	Establishes baseline for later comparison
Initial outpatient follow-up evaluation, 2–3 weeks after procedure	Pressure check Duplex scan	Duplex scan establishes initial measurements of aneurysm dimension; presence of endoleak
6 months	Pressure check Duplex scan	Increase in intrasac pulse pressure or pulse pressure index (comparison to systemic pulse pressure) may suggest endoleak
12 months	AP and lateral L-S spine x-rays Pressure check Duplex scan	Plain films assess endograft position, allow evaluation of metallic struts for fracture (CT scan can also be used)
Annual	AP and lateral L-S spine x-rays Pressure check Duplex scan	
Imaging Guidelines		
Duplex scanning	(1) B-mode measurements of maximum diameter and circumference. (2) color flow Doppler scan for endoleak. (3) pulsed Doppler evaluation of suspected endoleak, flow in limbs, outflow vessels, access vessels, visceral branches. Intravenous administration of an ultrasound contrast may be used during postoperative surveillance duplex scans if there is a suspected endoleak that is not demonstrated by the noncontrast duplex scan or if the aneurysm does not decrease in size over six months.	
CT scan	CT scan (may be performed without contrast for patients with renal insufficiency), if duplex examination technically limited or incomplete. CT angiogram with late (venous phase) imaging sequence to detect endoleak if there is an increase in aneurysm size.	

does not always result in immediate pressure reduction in the aneurysm sac. A 2007 review of publications reporting observations of aneurysm sac pressure measurements during or after EVAR evaluated the mean pressure indices (MPI), the ratio of the mean aneurysm sac pressure to mean systemic pressure. The review found that the reported MPI values differ widely, both in the absence and presence of an endoleak. MPI does not appear to be specific to the type of endoleak.[30] The pulse pressure index may be a more useful and reliable measure.

In fact, many questions remain about how to best use pressure data in long-term follow-up. For example, is concomitant imaging needed, and how often? How do pressure data correlate with outcomes? To answer questions like these, PRICELESS (PRessure and Imaging—using the CardioMEMS EndoSure Sensor for Long-term

TABLE 29-3. PRESSURE MEASUREMENTS

Value	Definition
Systolic blood pressure	Directly measured
Diastolic blood pressure	Directly measured
Pulse pressure	Systolic—diastolic blood pressure
Systolic pressure index (SBI)	Aneurysm sac systolic pressure Systemic systolic blood pressure
Mean pressure index (MPI)	Mean aneurysm sac pressure Mean systemic pressure
Pulse pressure index (PPI)[1]	Pulse pressure in aneurysm sac Systemic pulse pressure

[1]PPI may be best single value to assess adequacy of aneurysm exclusion as it 1) is a measure of pulsatiliy, and 2) is indexed to the system pulse pressure, allowing comparisons between patients or between intraoperative and postoperative values.

follow-up after EVAR with Standard Surveillance), a prospective registry study, has been organized to establish the parameters that might segregate patients into three categories; those with:

1. low sac pressure and low risk of rupture; identifying those might not need additional imaging or intervention
2. conditions that need intervention, including Type I and Type III endoleaks discovered during postoperative follow-up, or those with Type II endoleaks associated with an enlarging aneurysm sac
3. conditions needing careful ongoing observation such as patients with moderate pressure increases without identified endoleak, or those with Type II endoleaks without associated sac enlargement

The intent of the trial is to establish estimates of the sensitivity and specificity of implanted sensor pressure measurements for classification of leaks and no leaks. Additional information, such as the long-term durability and reliability of the technology, will be obtained.

The cost-effectiveness of various surveillance protocol will vary between different types of health care systems. For some, the postoperative surveillance imaging studies represent an opportunity for additional revenue generation. In other systems, such a capitated health care program or health management organization, each study may represent an additional cost. The financial impact of the use of pressure monitoring technologies may vary, depending not only on the patient population and surveillance protocols used, but also on the health care economics of the population being considered. This can be a topic for future studies.

REFERENCES

1. Chuter T, Ivancev K, Malina M, et al. Aneurysm pressure following endovascular exclusion. *Eur J Vasc Endovasc Surg*. Jan 1997;13(1):85–87.
2. Dias NV, Ivancev K, Malina M, et al. Direct intra-aneurysm sac pressure measurement using tip-pressure sensors: in vivo and in vitro evaluation. *J Vasc Surg*. Oct 2004;40(4):711–716.
3. Baum RA, Carpenter JP, Cope C, et al. Aneurysm sac pressure measurements after endovascular repair of abdominal aortic aneurysms. *J Vasc Surg*. Jan 2001;33(1):32–41.

4. Parodi JC, Berguer R, Ferreira LM, La Mura R, Schermerhorn ML. Intra-aneurysmal pressure after incomplete endovascular exclusion. *J Vasc Surg*. Nov 2001;34(5):909–914.
5. Schurink GW, Aarts NJ, Wilde J, et al. Endoleakage after stent-graft treatment of abdominal aneurysm: implications on pressure and imaging—an in vitro study. *J Vasc Surg*. Aug 1998;28(2):234–241.
6. Chong CK, How TV, Gilling-Smith GL, Harris PL. Modeling endoleaks and collateral reperfusion following endovascular AAA exclusion. *J Endovasc Ther*. Jun 2003;10(3):424–432.
7. Gawenda M, Winter S, Jaschke G, Wassmer G, Brunkwall J. Endotension is influenced by aneurysm volume: experimental findings. *J Endovasc Ther*. Dec 2003;10(6):1091–1096.
8. Ellozy SH, Carroccio A, Lookstein RA, et al. First experience in human beings with a permanently implantable intrasac pressure transducer for monitoring endovascular repair of abdominal aortic aneurysms. *J Vasc Surg*. Sep 2004;40(3):405–412.
9. Ohki T, Ouriel K, Silveira PG, et al. Initial results of wireless pressure sensing for endovascular aneurysm repair: the APEX Trial—Acute Pressure Measurement to Confirm Aneurysm Sac Exclusion. *J Vasc Surg*. Feb 2007;45(2):236–242.
10. Silveira PG, Miller CW, Mendes RF, Galego GN. Correlation between intrasac pressure measurements of a pressure sensor and an angiographic catheter during endovascular repair of abdominal aortic aneurysm. *Clinics*. Feb 2008;63(1):59–66.
11. Hoppe H, Segall JA, Liem TK, Landry GJ, Kaufman JA. Aortic aneurysm sac pressure measurements after endovascular repair using an implantable remote sensor: initial experience and short-term follow-up. *Eur Radiol.* May 2008;18(5):957–965.
12. Back MR. Surveillance after endovascular abdominal aortic aneurysm repair. *Perspect Vasc Surg Endovasc Ther*. Dec 2007;19(4):395–400; discussion 401–392.
13. Sharma P, Kyriakides C. Surveillance of patients post-endovascular aneurysm repair. *Postgrad Med J*. Dec 2007;83(986):750–753.
14. Sternbergh WC, 3rd, Greenberg RK, Chuter TA, Tonnessen BH. Redefining postoperative surveillance after endovascular aneurysm repair: recommendations based on 5-year follow-up in the US Zenith multicenter trial. *J Vasc Surg*. Aug 2008;48(2):278–284; discussion 284–275.
15. Wain RA, Marin ML, Ohki T, al. e. Endoleaks after endovascular graft treatment of aortic aneurysms: Classification, risk factors and outcome. *J Vasc Surg*. 1998;27:69–78.
16. Stavropoulos SW, Baum RA. Imaging modalities for the detection and management of endoleaks. *Semin Vasc Surg*. Jun 2004;17(2):154–160.
17. Raman KG, Missig-Carroll N, Richardson T, Muluk SC, Makaroun MS. Color-flow duplex ultrasound scan versus computed tomographic scan in the surveillance of endovascular aneurysm repair. *J Vasc Surg*. Oct 2003;38(4):645–651.
18. Henao EA, Hodge MD, Felkai DD, et al. Contrast-enhanced Duplex surveillance after endovascular abdominal aortic aneurysm repair: improved efficacy using a continuous infusion technique. *J Vasc Surg*. Feb 2006;43(2):259–264; discussion 264.
19. Clevert DA, Kopp R. Contrast-enhanced ultrasound for endovascular grafting in infrarenal abdominal aortic aneurysm in a single patient with risk factors for the use of iodinated contrast. *J Vasc Interv Radiol.* Aug 2008;19(8):1241–1245.
20. Carrafiello G, Lagana D, Recaldini C, et al. Comparison of contrast-enhanced ultrasound and computed tomography in classifying endoleaks after endovascular treatment of abdominal aorta aneurysms: preliminary experience. *Cardiovasc Intervent Radiol*. Nov–Dec 2006; 29(6):969–974.
21. Dill-Macky MJ. Aortic endografts: detecting endoleaks using contrast-enhanced ultrasound. *Ultrasound Q*. Mar 2006;22(1):49–52.
22. Bargellini I, Napoli V, Petruzzi P, et al. Type II lumbar endoleaks: hemodynamic differentiation by contrast-enhanced ultrasound scanning and influence on aneurysm enlargement after endovascular aneurysm repair. *J Vasc Surg*. Jan 2005;41(1):10–18.
23. McWilliams RG, Martin J, White D, et al. Use of contrast-enhanced ultrasound in follow-up after endovascular aortic aneurysm repair. *J Vasc Interv Radiol*. Sep 1999;10(8):1107–1114.

24. Kurosawa K, Ohta H, Sumi M, Ohki T. Physiologic monitoring data: will it change our follow-up paradigms? *Semin Vasc Surg*. Jun 2007;20(2):115–120.
25. Trocciola SM, Dayal R, Chaer RA, et al. The development of endotension is associated with increased transmission of pressure and serous components in porous expanded polytetrafluoroethylene stent-grafts: characterization using a canine model. *J Vasc Surg*. Jan 2006; 43(1):109–116.
26. Dias NV, Ivancev K, Resch TA, Malina M, Sonesson B. Endoleaks after endovascular aneurysm repair lead to nonuniform intra-aneurysm sac pressure. *J Vasc Surg*. Aug 2007; 46(2):197–203.
27. Pacanowski JP, Stevens SL, Freeman MB, et al. Endotension distribution and the role of thrombus following endovascular AAA exclusion. *J Endovasc Ther*. Oct 2002;9(5):639–651.
28. Vallabhaneni SR, Gilling-Smith GL, Brennan JA, et al. Can intrasac pressure monitoring reliably predict failure of endovascular aneurysm repair? *J Endovasc Ther*. Jun 2003;10(3): 524–530.
29. Hinnen JW, Koning OH, Visser MJ, Van Bockel HJ. Effect of intraluminal thrombus on pressure transmission in the abdominal aortic aneurysm. *J Vasc Surg*. Dec 2005;42(6): 1176–1182.
30. Hinnen JW, Koning OH, van Bockel JH, Hamming JF. Aneurysm sac pressure after EVAR: the role of endoleak. *Eur J Vasc Endovasc Surg*. Oct 2007;34(4):432–441; discussion 442–433.

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Percutaneous Repair of Abdominal Aortic Aneurysm

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With Food and Drug Administration approval, endograft repair of abdominal aortic aneurysm (AAA) is rapidly gaining widespread clinical acceptance. As this chapter goes to press, four different commercially available devices have been approved for general use. Thoracic endografts are undergoing refinement and are not yet available commercially, but some industry manufactured investigational devices are being used to treat thoracic aortic aneurysms (TAA), dissections, and traumatic transections with local institutional investigational device exemptions (IDEs).

All of these devices are placed through relatively large (18-24 Fr.) sheaths and must be positioned appropriately within the aorta after the sheaths are passed through access sites in the common femoral or iliac vessels. With few exceptions, this type of access has traditionally required arterial exposure via cut-down skin incisions—the sheaths are passed through an open arteriotomy after vascular clamps are applied to control the vessels. In general, this process is safe but it must be performed by practitioners experienced in open surgical technique. In many institutions a femoral artery cut-down mandates operating room availability and general or spinal anesthesia. Furthermore, open arterial access is not without potential for complication.

With smaller access sheath sizes and with the development of certain arterial closure devices, completely percutaneous treatment of abdominal aortic aneurysm (AAA) with local anesthesia has become feasible. In addition, percutaneous endoluminal treatment of thoracic aortic pathology has become a viable option. Potential advantages to percutaneous endograft deployment include shorter procedure time, improved patient acceptance, earlier ambulation, and reduced risk for wound complications. However, percutaneous sheath placement has its own unique set of risks. For the past two years, we have made an attempt to place all infrarenal aortic devices and a select few thoracic endovascular grafts using percutaneous techniques. For the last five years, we have maintained a prospective database that has allowed us to contrast patients who had devices placed completely percutaneously to those who have undergone open bilateral arterial cut-down. In this chapter, we compare placement of the bifurcated Gore-Tex® Excluder infrarenal endograft (W. L. Gore & Associates, Inc.,

Flagstaff, AZ) using percutaneous femoral access (PFA) to a historic control group of patients treated with the same device using femoral artery cut-down (FAC) for treatment of infrarenal AAA.

METHODS

Seventy-one consecutive patients who underwent endovascular repair of an AAA using the Gore bifurcated Excluder endograft between March 1999 and May 2003 at Northwestern University were studied for evidence of access related complications. Before November of 2000, all patients underwent bilateral femoral artery cut-down (FAC) for access to deploy the endograft device. From November 2000 to May 2003, all patients who had endovascular aneurysm repair with the Gore device had an attempt at bilateral percutaneous femoral access (PFA). There were no specific anatomic criteria upon which the choice of access approach was chosen. The decision for percutaneous treatment was based solely upon the temporal sequence in which the patient was treated. Data was retrospectively collected from a prospectively maintained data-base. Thirty-three patients underwent bilateral FAC; 31 (94%) of them were male. Thirty-eight patients underwent bilateral PFA; 28 (74%) of these patients were male. Demographics (age, weight, gender, AAA size) and patient comorbidities (hypertension, coronary artery disease, stroke, diabetes mellitus, chronic obstructive pulmonary disease, history of claudication, history of bleeding disorders, renal failure, renal dialysis, current tobacco use, or current steroid use) were reviewed, analyzed, and listed in Table 30–1.

We have traditionally performed open femoral access via small transverse or oblique skin incisions, each measuring about 4 cm. in length depending upon patient

TABLE 30-1. PATIENT DEMOGRAPHICS

	Bilateral Femoral Artery Cut-Down n (%)	Bilateral Percutaneous Femoral Access n (%)
Number of patients	33	38
Mean age (y)	74	75
Mean patient weight (kg)	83.6 (range 56-125)	82.2 (range 50-159)
Male gender	31 (94)	28 (74)
Female gender	2 (6)	10 (26)
Mean AAA size (cm)	5.6	6.0
Hypertension	19 (58)	22 (58)
Coronary artery disease	18 (55)	22 (58)
Cerebrovascular disease	1 (3)	5 (13)
Diabetes	6 (18)	8 (21)
Renal dialysis	0 (0)	0 (0)
Chronic Obstructive Pulmonary Disease	4 (12)	9 (24)
Hypercholesterolemia	10 (30)	19 (50)
Current tobacco use	8 (24)	6 (16)
Bleeding disorder	0 (0)	0 (0)
Current steroid use	0 (0)	1 (3)
History of claudication	3 (9)	4 (11)

AAA = abdominal aortic aneurysm

body habitus. Care is taken to avoid lymphatic transection. Five or six centimeters of common femoral artery are exposed, heparin is administered, the vessels are clamped, an arteriotomy is created, and the appropriate sheath (12F × 30cm or 18F × 30cm) is passed through the opening under direct vision. When the procedure is complete, the sheaths are removed, non-crushing vascular clamps are reapplied, and the arteriotomy is repaired with interrupted, fine monofilament suture.

Percutaneous access is performed through small stab incisions using a preclosure technique with a 10 Fr. Prostar XL® device (Perclose, An Abbott Laboratory Company, Redwood City, CA) (Figure 30–1). Arterial access is initiated with an 18-gauge needle. A standard J-wire is positioned in the aorta and a short 6F or 8F sheath is placed to predilate the access site. The sheath is then exchanged for the Prostar device after a tract is cleared through the superficial soft-tissue, down to the artery using a hemostat or a finger (Figure 30–2). The two Perclose 3-0 braided polyester sutures (one device) are deployed prior to placing the endograft deployment sheaths (12F and 18F) and left to rest upon the patient in radial orientation until after the endograft deployment is complete. For larger sheaths (22F and 24F), we recommend repeating this procedure and placing a second set of Perclose sutures (total of four) at 45° relative to the first set. Patients are generally administered intravenous heparin, but the dose is smaller than during FAC cases. When the procedure is complete, the sheaths are removed while an assistant

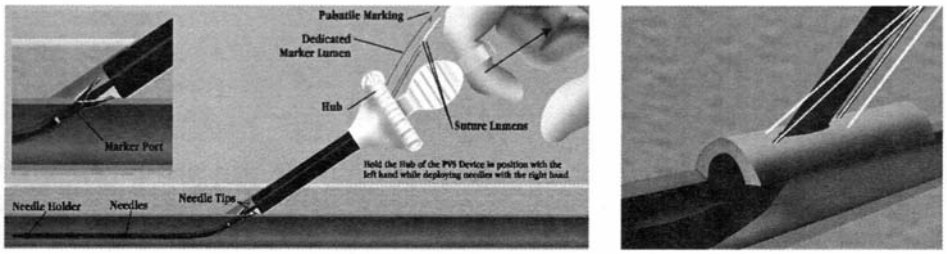

Figure 30-1. The Prostar XL® arterial closure device (Perclose, Inc., Redwood City, CA).

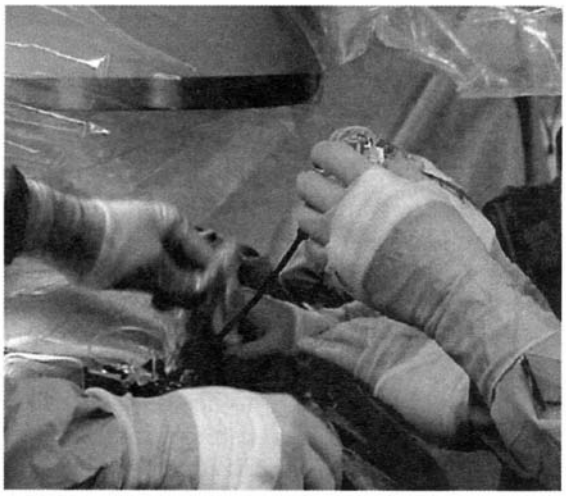

Figure 30-2. Introduction of a Prostar XL® arterial closure device.

maintains proximal pressure manually. The sutures are generously soaked with heparinized saline and tied with a slip-knot or a standard surgeon's knot while manual pressure is maintained. The incisions are closed with a single suture or a steri-strip.

All patients in the two groups underwent a physical exam and determination of the ankle-brachial index (ABI) pre-operatively, post-operatively in the recovery room, and daily during the hospitalization. Outpatient follow-up consisted of an evaluation at 1 month, 6 months, 12 months, and yearly thereafter. At follow-up, patients underWent a complete physical examination with determination of the ABI. In addition, plain abdominal films and CT scans were obtained to evaluate the endograft. No patients were lost to follow-up.

Primary endpoints were any access-related complication. These included infection, bleeding, arterial occlusion, arterial emboli, arterial dissection, femoral neuropathy, lymphocele, leg pain, and leg edema. Secondary endpoints included total operative time, total anesthesia time, type of anesthesia, estimated blood loss (EBL), transfusion requirement, time to first oral intake, time to first ambulation, and hospital length of stay.

Data are expressed as mean ± the standard error of the mean. Differences between the two groups were determined using the Students t-test. Statistical significance was assumed for $P < .05$. Statistical analysis was performed using Sigma Stat (SPSS, Chicago).

RESULTS

Mean aneurysm size was 5.6 cm for FAC and 6.0 cm for PFA patients. There were no significant differences with respect to demographics or patient comorbidities between the two groups (Table 30–1). In the immediate post-operative period, five patients in the FAC cohort developed access-related complications (Table 30–2). Complications included femoral artery dissection (1), groin hematoma (2), wound infection (1), and occlusion of a femoral-popliteal artery bypass graft (1). Three of these complications (dissection, groin hematoma, and graft occlusion) required returning the patient to the operating room for further treatment.

TABLE 30-2. ACCESS COMPLICATIONS DETECTED IN PATIENTS DURING THE IMMEDIATE POST-OPERATIVE PERIOD

Bilateral Femoral Artery Cut-Down	Bilateral Percutaneous Femoral Access
CFA dissection*	CFA occlusion*
Femoral-popliteal graft occlusion*	CFA occlusion*
Groin hematoma*	CFA bleed*
Wound infection	CFA bleed*
Groin hematoma	CFA bleed*
	Groin hematoma
	Groin hematoma
	Scrotal hematoma

*Required reoperation
CFA = common femoral artery

In the PFA group, access-related complications occurred in eight patients during the immediate post-operative period (Table 30–2). All PFA complications were the result of hemorrhage (6) or arterial occlusion (2). Five of the eight complications required immediate additional surgical intervention. All five were recognized and ameliorated following the index operation before the patient was allowed to leave the operating room.

During the post-operative course, a few patients in both patient groups required blood transfusions (n = 4 FAC group versus n = 6 PFA group). There were no access-related complications detected and no apparent source for bleeding identified in more than half of these patients (n = 3 FAC group versus n = 2 PFA group). The remaining blood transfusions were required secondary to access-related complications (n = 1 FAC group versus n = 4 PFA group). However, these numbers are too small to make any generalizations or reach any statistical conclusions.

At 30-day follow-up, no additional access-related complications were detected in the PFA group (Table 30–3). However, there were seven additional access-related complications detected in seven patients from the FAC group (Table 30–3). Late complications included femoral neuropathy (3), lymphocele (3), and scrotal edema (1). None of these delayed access-related complications required additional therapeutic intervention. At 6-month follow-up, no additional access-related complications were detected in either patient cohort.

An analysis of total operative time, anesthesia time, estimated blood loss (EBL), time to first oral intake, time to ambulation, and hospital length of stay was conducted (Table 30–4). Patients undergoing bilateral PFA were found to have shorter total operative time (PFA 231 min versus FAC 280 min, P = 0.002), a reduction in total anesthesia time (PFA 171 min versus FAC 224 min, P < 0.001), and a reduction in the use of

TABLE 30-3. ACCESS COMPLICATIONS DETECTED AT 1 MONTH FOLLOW-UP

Bilateral Femoral Artery Cut-Down	Bilateral Percutaneous Femoral Access
Femoral Neuropathy	None
Femoral Neuropathy	
Femoral Neuropathy	
Scrotal Edema	
Lymphocele	
Lymphocele	
Lymphocele	

TABLE 30-4. OPERATIVE AND POSTOPERATIVE OUTCOMES

	Bilateral Femoral Artery Cut-Down	Bilateral Percutaneous Femoral Access	P-Value
Total Operative Time	280 min	231 min	0.002
Total Anesthesia Time	224 min	171 min	< 0.001
Estimated Blood Loss	407 ml	373 ml	0.58
Time to First Oral Intake	0.42 days (Range 0-2)	0.16 days (Range 0-1)	0.028
Time to First Ambulation	0.79 days (Range 0-3)	0.79 days (Range 0-2)	0.992
Hospital Length of Stay	1.76 days (Range 1-7)	1.47 days (Range 1-5)	0.338

general anesthesia ($P < 0.001$). No significant differences were observed between the groups with respect to estimated blood loss (PFA 373 ml versus FAC 407 ml, $P = 0.58$). Patients who underwent percutaneous femoral access achieved earlier time to first oral intake (PFA 0.16 day versus FAC 0.42, $P = 0.028$). However, time to first ambulation (PFA 0.79 versus FAC 0.79, $P = 0.992$) and overall hospital length of stay (PFA 1.47 days versus FAC 1.76 days, $P = 0.338$) were not statistically different between the two groups.

DISCUSSION

Percutaneous treatment of infrarenal abdominal aortic aneurysms has become possible. As a viable alternative to open repair, this represents a major advancement, particularly for the elderly and infirmed with AAA who otherwise would likely remain untreated.[1-4] However, percutaneous AAA repair requires special expertise and practitioners must become familiar with particular arterial closure devices before they abandon open access.

Arterial closure devices were originally developed for use with smaller access sheaths. Bioabsorbable sponges (Angio-Seal™, Kensey-Nash, Exton, PA), bovine collagen plugs (VasoSeal VHD™, Datascope, Montvale, NJ), fibrin and thrombin procoagulant glues (Duett™, Vascular Solutions, Minneapolis, MN), and small suture-mediated closure devices (Closer™, Perclose, Inc, Redwood City, CA; X-PRESS, X-Site Medical, Blue Bell, PA) have been shown to work relatively well after arterial puncture with 6F and 8F sheaths.[5-11] These adjuncts to manual pressure save time, limit patient discomfort, and allow for earlier patient ambulation but they cannot be used safely following arterial access with 10F or larger sheaths.

Suture-mediated closure devices that allow for percutaneous repair of the arterial defect such as Prostar Plus® and Prostar XL® (Perclose, Inc., Redwood City, CA) (Figure 30–1) can be used off-label following removal of the larger sheaths used during endovascular AAA and TAA repair.[1-3,12,13] Deployment of one or two of these devices per femoral artery does provide for safe and secure arterial closure through a simple stab incision in the groin.

Some real and some theoretic advantages to percutaneous aneurysm repair do exist. In our experience, patients who underwent percutaneous endograft deployment had more rapid repair of their AAA and could be treated more commonly with local or regional anesthesia. Also, theoretically, patients should move to ambulation sooner than with open arterial access. Clearly, wound complications noted after hospital discharge were less frequent following percutaneous access when compared to open arterial exposure.

Percutaneous access and arterial repair with suture-mediated femoral artery closure devices is not risk-free. Device entrapment, acute arterial thrombosis with limb ischemia, arterial injury, or suture breaks resulting in hemorrhage, arterial dissection, suture infection, and pseudoaneurysm or arteriovenous fistula formation have all been described following use of this closure technique.[14-16] In this review, 13% ($^5/_{38}$) of patients experienced immediate access site complications that required urgent attention. Bleeding complications and acute arterial occlusion were not uncommon and in all cases they required treatment with surgical exposure of the accessed artery immediately, before taking the patient from the operating suite. When bleeding or ischemic complications do occur, it is usually necessary to treat the problem immediately. We

recommend that complete percutaneous AAA repair be performed by surgeons and in an operating room. Alternatively, percutaneous AAA repair can be undertaken in a cardiac cath lab or an interventional radiology suite when both a surgeon and an operating room are on stand by for immediate assistance and transfer if problems arise.

Although Perclose suture infection was not observed in any of our patients, this is a well-described complication and it is one we have had to treat on occasion in patients referred to our service from outside sources.[17-20] The management of this complication is beyond the scope of this chapter, but suffice it to say, it can be a very vexing problem that requires specific vascular surgical expertise. In this series, we were fortunate to experience no late Perclose complications despite close long-term patient observation.

We generally consider all patients to be candidates for percutaneous repair. In our institution, all patients who undergo AAA repair using the Gore Excluder are taken to the operating room with the intent to repair the aneurysm percutaneously. We have also treated select patients with AAA using other endograft types (Ancure®, Guidant Corporation, Indianapolis, IN; AneuRx®, Medtronic, Inc., Minneapolis, MN; Zenith® Cook Incorporated, Bloomington, IN) that require larger (24F) sheath sizes as well. Additionally, we have successfully managed a handful of patients who have undergone endograft repair of thoracic aortic pathology, including aneurysms and acute aortic transections, using similar percutaneous access techniques. Patients with very small, severely calcified, or aneurismal femoral arteries are not ideal candidates for a percutaneous repair and must be approached with caution. Patients with morbid obesity can also be more difficult to treat percutaneously but these patients may also reap the greatest benefit when the approach is successful and an incision can be avoided.

CONCLUSION

Complete percutaneous treatment of AAA has significant advantages over open femoral artery access but it is not free of risk. Percutaneous treatment of AAA should be reserved for carefully selected patients and should be performed in a sterile environment or otherwise, where open arterial access can be obtained rapidly, if required.

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REFERENCES

1. Traul DK, Clair DG, Gray B et al. Percutaneous endovascular repair of infrarenal abdominal aortic aneurysms: A feasibility study. *J Vasc Surg*. 2000;32:770–776.
2. Rachel ES, Bergamini TM, Kinney EV et al. Percutaneous endovascular abdominal aortic aneurysm repair. *Ann Vasc Surg*. 2002;16:43–49.

3. Howell M, Villareal R, Krajcer Z. Percutaneous access and closure of femoral artery access sites associated with endoluminal repair of abdominal aortic aneurysms. *J Endovasc Ther*. 2001;8:68–74.
4. Krajcer Z, Howell M. A novel technique using the percutaneous vascular surgery device to close the 22 French femoral artery entry site used for percutaneous abdominal aortic aneurysm exclusion. *Catheter Cardiovasc Interv*. 2000;50:356–60.
5. Duffin DC, Muhlestein JB, Allisson SB et al. Femoral arterial puncture management after percutaneous coronary procedures: A comparison of clinical outcomes and patient satisfaction between manual compression and two different vascular closure devices. *J Invas Cardiol*. 2001;13:354–362.
6. Goyen M, Herborn CU, Lauenstein TC et al. Optimization of contrast dosage for gadobenate dimeglumine-enhanced high-resolution whole-body 3D magnetic resonance angiography. *Invest Radiol*. 2002;37:263–268.
7. Sesana M, Vaghetti M, Albiero R et al. Effectiveness and complications of vascular access closure devices after interventional procedures. *J Invas Cardiol*. 2000;12:395–399.
8. Falstrom JK, Goodman NC, Ates G et al. Reduction of femoral artery bleeding post catheterization using a collagen enhanced fibrin sealant. *Cathet Cardiovasc Diagn*. 1997;41:79–84.
9. Illi OE, Meier B, Paravicini G. First clinical evaluation of a new concept for puncture-site occlusion in interventional cardiology and angioplasty. *Eur J Pediatr Surg*. 1998;8:220–223.
10. Kornowski R, Brandes S, Teplitsky I et al. Safety and efficacy of a 7 French perclose arterial suturing device following percutaneous coronary interventions: A pilot evaluation. *J Invas Cardiol*. 2002;14:741–745.
11. Mehta H, Fleisch M, Chatterjee T et al. Novel femoral artery puncture closure device in patients undergoing interventional and diagnostic cardiac procedures. *J Invas Cardiol*. 2002;14: 9–12.
12. Hass PC, Krajcer Z, Diethrich EB. Closure of large percutaneous access sites using the Prostar XL percutaneous vascular surgery device. *J Endovasc Surg*. 1999;6:168–170.
13. Quinn SF, Duke DJ, Baldwin SS et al. Percutaneous placement of a low-profile stent-graft device for aortic dissections. *J Vasc Interv Radiol*. 2002;13:791–798.
14. Nehler MR, Lawrence WA, Whitehill TA et al. Iatrogenic vascular injuries from percutaneous vascular suturing devices. *J Vasc Surg*. 2001;33:943–947.
15. Wilson JS, Johnson BL, Parker JL et al. Management of vascular complications following femoral artery catheterization with and without percutaneous arterial closure devices. *Ann Vasc Surg*. 2002;16:597–600.
16. Boston US, Panneton JM, Hofer JM et al. Infectious and ischemic complications from percutaneous closure devices used after vascular access. *Ann Vasc Surg*. 2003;17:66–71.
17. Cherr GS, Travis JA, Ligush J et al. Infection is an unusual but serious complication of a femoral artery catheterization site closure device. *Ann Vasc Surg*. 2001;15:567–570.
18. Geary K, Landers JT, Fiore W, Riggs P. Management of infected femoral closure devices. *Cardiovasc Surg*. 2002;10:161–163.
19. Pipkin W, Brophy C, Nesbit R, Mondy JS. Early experience with infectious complications of percutaneous femoral artery closure devices. *J Vasc Surg*. 2000;32:205–208.
20. Johanning JM, Franklin DP, Elmore JR, Han DC. Femoral artery infections associated with percutaneous arterial closure devices. *J Vasc Surg*. 2001;34:983–985.

31

Aortic Neck Changes during Surveillance of Small Abdominal Aortic Aneurysms

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The standard of care for the treatment of small infrarenal abdominal aortic aneurysms (AAAs), that is, those with a maximum diameter up to 5.5 cm, is based in the Aneurysm Detection and Management (ADAM) trial and the UK Small Aneurysm Trial.[1,2] Both these trials revealed that surveillance was a safe management option compared with open surgical repair as no benefits in survival was seen with early repair. Endovascular aneurysm repair (EVAR) has, however, been rapidly replacing open aneurysm repair for the treatment of AAAs. A recent report from national hospital databases documented a 600% increase in the number of EVAR procedures performed since 2000, comprising nearly half of all aneurysm repairs.[3] It is uncertain, however, whether early EVAR results in improved survival compared with surveillance.

Recent interest in early EVAR for small AAAs has emerged given the lower morbidity and mortality of this type of repair compared to open repair. In fact, two randomized clinical trials are underway to assess the survival benefits of early EVAR compared to surveillance for the management of small AAAs. The interest in early EVAR for small AAAs is partly due to the apparently worse clinical outcomes with EVAR for large AAAs.[4,5] Secondary outcomes such as rates of type I endoleak, device migration, and surgical conversion have been reported to be higher after EVAR for large AAAs. Compromised secondary outcomes have also jeopardized aneurysm-related mortality as well as overall survival.[6,7] EVAR of large AAAs is, in fact, vulnerable to imperfect outcomes because such aneurysms are more likely to be associated with hostile aneurysm anatomy.[4,8] Morphologic studies have demonstrated that AAA maximum diameter is inversely related to the length of aortic neck.[9] Moreover, the diameter of the aneurysm is the most useful surrogate determinant of feasibility for EVAR.[5,9]

In October 2004, the U.S. Food and Drug Administration approved the use of larger endografts (main body diameters up to 36 mm) for EVAR. Although few European studies have assessed EVAR suitability with the use of these larger

endografts,[10,11] most American studies have used selection criteria for EVAR using endografts with main body diameters up to 28 or 32 mm.[12,13]

In two observational studies from our institution, anatomic suitability for EVAR was assessed with the potential use of endografts up to 32 mm in diameter. In the initial study, we reported that 64% of patients with small AAAs were candidates for EVAR, whereas only 39% of patients with large AAAs were suitable for EVAR when the manufacturers' instructions for use were strictly followed.[14] We demonstrated that small AAAs have less complex anatomy with longer aortic necks, less neck angulations, and less tortuosity. If growth of small AAAs significantly alters the anatomic suitability for EVAR, then an argument can be made to use EVAR at an earlier size threshold. In a subsequent study in which EVAR suitability was assessed with the potential use of endografts up to 32 in diameter, we longitudinally assessed a group of patients with small AAAs to determine the morphologic changes associated with aneurysm growth and its subsequent effect on overall suitability for EVAR.[15] Below, we present a reanalysis of our previously reported data in which changes in EVAR suitability was reassessed with the potential use of larger endografts.

SURVEILLANCE AND EVALUATION OF EVAR SUITABILITY OF SMALL AAAS

During a three-year period, 54 patients referred with AAAs with maximum diameters ranging from 4.0 cm to 5.4 cm underwent CTA at our institution. After initial evaluation by the vascular surgery service, all these patients were followed with CTA. All CTA studies were performed using a helical Hi Speed I from GE Medical Systems (Milwaukee, WI) with collimation set at 3 mm and a 2.0 pitch, and were further processed to obtain 3-D reconstructions on a Vitrea workstation (Vital Images, Plymouth, MN). Anatomic AAA characteristics were determined and based on the SVS reporting standards.[16,17] To further determine EVAR suitability, we specifically assessed angle-corrected aortic neck length and diameter, suprarenal and infrarenal aortic neck angle, amount of aortic neck thrombus and calcification, maximum angle-corrected aneurysm diameter, aneurysm tortuosity index (median luminal centerline/straight line distance), amount of aneurysm thrombus, amount of iliac artery thrombus and calcification, common iliac artery diameter and length, iliac artery tortuosity (median luminal centerline/straightline distance), and external iliac artery diameter. The measurements obtained from initial and follow-up CTAs were entered into a database designed for this anatomic study, which was approved by the local institutional review board.

For the current analysis, suitability for EVAR was determined in two separate ways according to neck anatomy (diameter, length, and angulation), iliac artery morphology, and total aortic aneurysm angulation and tortuosity. EVAR anatomic suitability was initially determined according to the clinicians' experience and current practice with the endografts commercially available in the United States, including the recently introduced large endografts with diameters up to 36 mm. For this method, EVAR suitability was defined as the clinicians' expectations of delivering an endograft and achieving its secure fixation at the proximal and distal landing zones of the aneurysm necks. Although aortic aneurysm proximal necks could be suitable for EVAR when the proximal neck landing zone was less than10 mm, neck angulation had to be minimal in these cases. In the second method, suitability for EVAR was strictly established according to the guidelines established by the instructions for

use of the endografts as approved by the Food and Drug Administration (FDA) in the United States at the time the study was reanalyzed (i.e., AneuRx and Talent [Medtronic, Minneapolis, MN], Excluder [W. L. Gore & Assoc., Flagstaff, AZ], PowerLink [Endologix, Irvine, CA], and Zenith (Cook, Bloomington, IN] endovascular grafts. These guidelines stipulate the proximal infrarenal aortic neck length be ≥10 mm, neck diameter be ≤32 mm, neck angulation be < 50 degrees, and absence of circumferential calcification or >50% thrombus of the proximal aortic neck, which are generally consistent with FDA labeling for currently approved devices. If EVAR was not feasible with one device, the possibility of EVAR was assessed with each of the remaining devices. AAA size, medical comorbidities, or surgical risk were not considered to define EVAR suitability in this cohort.

Standard statistical tests involving a repeated measures design and comparisons between two matched measures for the same subjects were used. For the purpose of this analysis, matched and paired comparisons of EVAR suitability and aneurysm morphologic measures as assessed in the first and last CTA were performed. Findings were considered statistically significant for the primary end point (i.e., changes in aortic neck length and diameter) if the resulting P-value was less than .05. SAS version 9.1 (SAS Institute, Cary, NC) and MedCalc for Windows version 8.1.0.0 (MedCalc Software, Mariakerke, Belgium) were used for data analyses.

SMALL AAA MORPHOLOGY CHANGES DURING SURVEILLANCE

In this series, the median age of the study population was 73 years (interquartile range [IQR], 65–77 years). All patients were male. The median follow-up period between the initial CTA and the last follow-up CTA was 24 months (IQR, 15–36 months). Cumulative patient survival was 78% at 36 months and 61% at 60 months.

During surveillance, the median aortic neck diameter increased from 23.0 mm to 24.0 mm (P=.02) and the median aortic neck length decreased from 26.5 mm to 20.0 mm (P=.001) (Figure 31–1). The small AAAs grew from a median maximum aortic aneurysm diameter of 44.5 mm (IQR, 41-48) to 48.9 mm (IQR 45.7-52.0), with a median growth rate of 2.2 mm per year (Figure 31–2). The median aneurysm tortuosity index

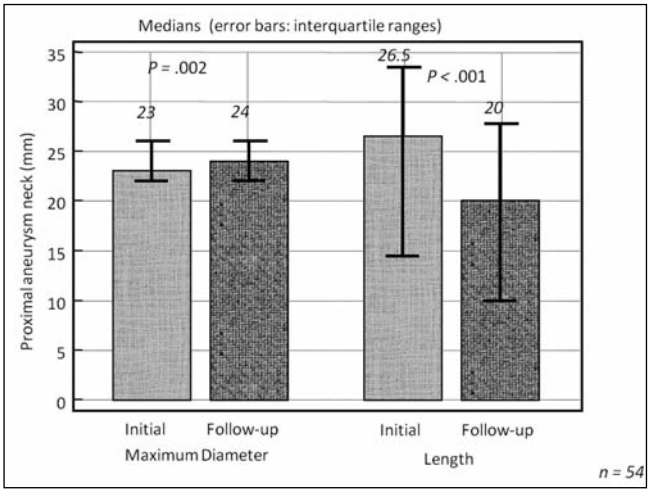

Figure 31-1. At mid-term follow-up and during surveillance of 54 small AAAs, the median aortic neck diameter increased from 23.0 mm to 24.0 mm (P=.02) and the median aortic neck length decreased from 26.5 mm to 20.0 mm (P=.001)

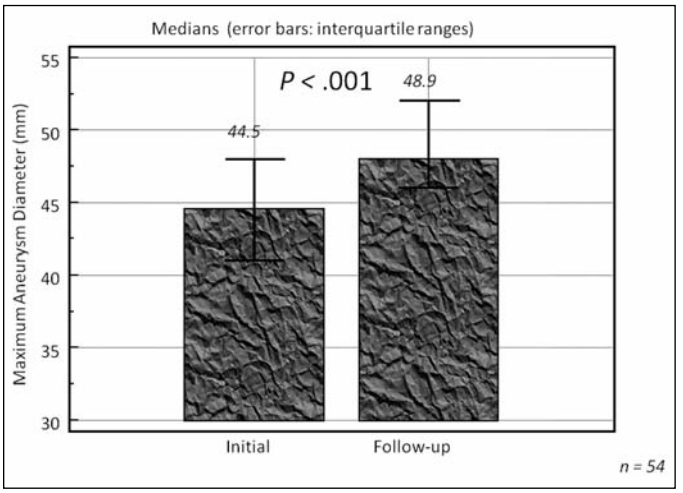

Figure 31-2. The median maximum aortic aneurysm diameter increased from 44.5 mm (IQR, 41-48) to 48.9 mm (IQR 45.7-52.0) during a median follow-up period of 24 months.

increased from 1.09 to 1.11 (P=.05). Iliac artery morphology characteristics did not significantly change.

The percentage of patients eligible for EVAR as assessed by the clinicians' method decreased from 80% to 76% (McNemar test, P=.5) (Figure 31–3), whereas EVAR suitability according to the instructions for use of endografts decreased from 55% to 46% (McNemar test, P=.92). To prevent confounding from inclusion of patients with AAAs unsuitable for EVAR in their initial CTA, stratified analyses excluding such patients were performed, and revealed that the rate of endovascular suitability according to the clinicians method decreased from 100% to 96% (McNemar test, P=.5). Interestingly, rates for EVAR suitability according to the instructions for use of the endografts were the same (i.e., 100% and 96%) (McNemar test, P=.5). Such changes in rates of EVAR suitability, however, were not statically significant in these subgroup analyses. Willingness to accommodate infrarenal aortic necks with more angulation, calcification, and thrombus (n=7), as well as shorter aortic necks (n=4), accounts for the difference in EVAR suitability between the two different methods. All the

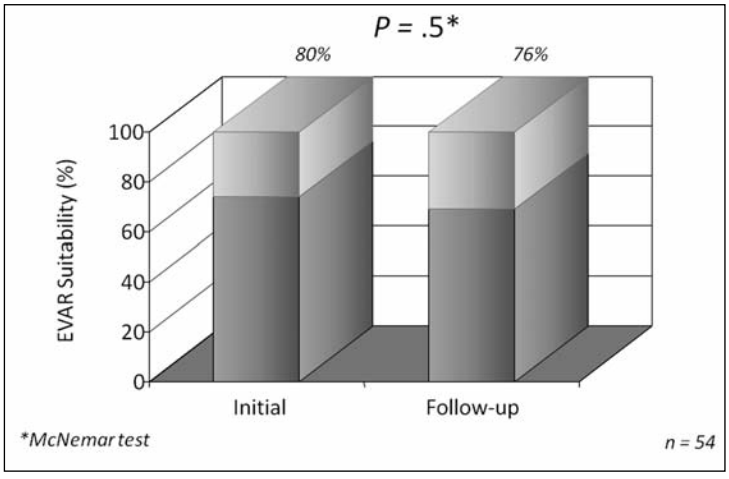

Figure 31-3. EVAR suitability slightly decreased during surveillance from 80% to 76% (McNemar test, P=.5.

differences in EVAR suitability during the surveillance period did not reach statistical significance.

Adverse proximal aortic neck anatomy was the only factor to account for exclusion of patients from EVAR suitability. Of the 14 patients who did not qualify for EVAR at the initial CT scan evaluation according to the clinicians' method, 36% (n=5) were due to a proximal aortic neck diameter greater than 32 mm. Another 36% (n=5) were unsuitable due to proximal infrarenal aortic necks that were too short, and 7% (n=1) for a proximal neck that had too much calcification and thrombus. During follow-up, only two patients developed morphologic changes that changed their status from suitable to unsuitable EVAR candidates. Both were due to proximal necks that had shortened during the surveillance period to a length that was too short for adequate proximal fixation (neck length decreased from 8 mm to 3 mm and from 15 mm to 6 mm), respectively.

On the initial CTA, 70% (n=38) of the patients had neck lengths greater than 15 mm and 72% (n=39) of the patients had neck diameters between 17 mm and 25 mm. At the last CTA, only a minority of patients who continued to be candidates for EVAR had borderline aortic neck lengths (11 mm to 15 mm, 10%, n=4) or borderline aortic neck diameters (26 mm to 32 mm, 18%, n=8). The majority (78%) of patients who were initially endovascular candidates continued to have long (>15-mm) and suitably narrow (<26-mm) infrarenal aortic necks.

EVAR, OPEN REPAIR, AND CONTINUED SURVEILLANCE FOR SMALL AAAS

In this series, patients with AAAs with a maximum external diameter in any plane greater than or equal to 5 cm and suitable for open and endovascular repair were offered the possibility of enrollment in the Open Versus Endovascular Repair Veterans Administration Trial. During follow-up, 25% of the patients underwent aneurysm repair (median follow-up, 22.6 months [IQR, 8–41 months]). Indications for repair included AAAs with a maximum diameter > 5 cm at their last CTA in five patients, (37%) significant aneurysm growth (>5 mm within six months or >10 mm with one year) in three patients (21%), AAAs with a maximum diameter > 5.5. cm in three (21%), symptomatic AAAs in two (14%) and one (7%) became saccular during growth. EVAR was performed in 57% of patients requiring repair (n=8), whereas the remaining 43% (n=6) received open repair. Of the six patients who underwent open repair, four had small AAAs with anatomy suitable for EVAR at the beginning and end of the study; two of these patients were randomized to open repair as part of the Open Versus Endovascular Repair Veterans Administration Trial, whereas the other two with AAAs suitable for EVAR elected to undergo open repair. The remaining two patients had unsuitably short necks at both initial and follow-up studies. The overall freedom from surgical open and/or endovascular intervention was 79% at 40 months and 69% at 60 months.

All patients undergoing open and endovascular repair of their AAAs were alive at the end of the study period. There was one documented aortic aneurysm rupture in this series. This patient survived an emergent open repair and was alive at the end of the study period. His AAA had grown from 55 mm to 61 mm in maximum diameter. His AAA was not suitable for EVAR because of a wide proximal neck. Although this patient was strongly advised to undergo open repair of his aneurysm, he decided not to undergo the procedure.

IMPLICATIONS OF AORTIC NECK CHANGES DURING SURVEILLANCE OF SMALL AAAS

Our series demonstrates that as individual aneurysms grow in maximum diameter, the proximal aortic neck widens in diameter and shortens in length. Despite these aortic neck changes in aortic aneurysm morphology associated with growth during surveillance of small AAAs, suitability for EVAR and clinical management do not significantly change at mid-term follow-up. Although it may seem like an apparent contradiction that observed adverse morphologic changes (shorter and larger aortic necks) would not lead to decreases in EVAR suitability, closer examination of our data can explain how these two conclusions can coexist. The majority of patients who remained EVAR candidates throughout the study (45 patients, 84%) continued to have long (>15 mm) and suitably narrow (<26 mm) infrarenal aortic necks. Only 16% had borderline aortic neck lengths (10–15 mm) and only 10% had borderline aortic neck diameters (>28 mm). Therefore, only a minority of patients with small aneurysms are "at risk" to lose their EVAR suitability during the surveillance period. This observation suggests that an aneurysm's suitability for EVAR is determined early in the morphologic life of the aneurysm, likely before the maximum aortic diameter reaches 4 cm. If an aneurysm is not suitable for EVAR when surgical repair is warranted, it is likely that the aneurysm was unsuitable for EVAR during most of its natural history. Surveillance likely will exclude very few patients from the possibility of EVAR.

Our series does not support the contention that the observed morphologic changes during surveillance of small AAAs will necessarily result in adverse long-term EVAR outcomes. There is no data that EVAR with longer aortic necks (26.5 mm at the beginning of the study) necessarily results in better long-term outcomes than EVAR with adequately long but shorter necks (20.0 mm at the end of the study). Surveillance does not change the fact that the majority of patients suitable for EVAR at the end of surveillance will continue to have comfortably long and small aortic necks that should not compromise long-term outcomes with EVAR. Moreover, further analysis of our data determining EVAR suitability with the potential use of endografts up to 36 mm in diameter, which only became recently available in the United States, revealed that EVAR suitability rates were almost the same during the study period (80% vs 76%; McNemar test, P=.5).

Previous studies have also suggested a strong relationship between aneurysm size and aortic neck morphology. Bayle et al. discovered that aneurysms larger than 6 cm had shorter proximal necks and increased iliac tortuosity.[9] Armon et al. found that aneurysms >7 cm had shorter and wider aortic necks.[11] Arko et al. similarly observed that as aneurysm size increased, aortic neck length decreased by 27% concomitant with a 15% higher incidence of severe neck angulation.[8] These previous studies, however, compared two different cohorts of patients, one with small aneurysms and one with large aneurysms. Our series is unique as morphologic changes are longitudinally evaluated in the same patients over time during surveillance, and in some instances, until repair is required.

In our original study, a proximal aortic neck diameter greater than 28 mm, which was the largest neck accommodated by the commercially available Zenith endograft (Cook, Bloomington, IN), was the main contraindication to EVAR (57%) in patients who did not qualify for EVAR at the initial CTA evaluation.[13] Another 36% were unsuitable for EVAR due to inadequate length of the proximal neck, whereas the remaining 7% was ineligible because of a proximal neck with too much calcification and thrombus. Adverse proximal aortic neck anatomy was the only factor to account

for exclusion of patients from endovascular repair. Of note, in our current series and during mid-term follow-up, only two patients initially eligible for EVAR developed morphologic changes that made them subsequently unsuitable candidates for EVAR according to the clinicians' method. In both instances, the proximal necks shortened to an inadequate length to ensure an adequate seal.

The majority of small aneurysms have long infrarenal aortic neck lengths with suitable neck diameters. After diagnosis of the small aneurysms at initial CT scans, 70% of the patients had proximal necks greater than 15 mm in length, whereas 72% had neck diameters between 17 mm and 25 mm. At the conclusion of the surveillance period, only a minority of patients who continued to be candidates for EVAR had borderline aortic neck lengths (11 mm to 15 mm, 10%) or borderline aortic neck diameters (26 mm to 28 mm, 18%). The majority (78%) of patients who were initially endovascular candidates continued to have comfortably long (>15-mm) and narrow (<26-mm) infrarenal aortic necks.

It appears that the suitability of an aneurysm for endovascular repair is predetermined early in the morphologic life of the aneurysm, likely before maximum aortic diameter reaches 4 cm. If an aneurysm is not suitable for EVAR when surgical repair is warranted, it is likely that the aneurysm was unsuitable for EVAR during most of its natural history. Conversely, if an aneurysm is suitable for EVAR when it is small, it is likely that it will continue to be amenable to EVAR when the appropriate time for surgical intervention arrives. It is unlikely that intervention earlier in the life of an aneurysm may result in an improved window of opportunity for EVAR when it is repaired prior to 5 cm in size, as this study demonstrates. Whether or not earlier EVAR has a salutary effect over delayed EVAR for small AAAs remains unsubstantiated speculation awaiting results from randomized studies.

Although longer follow-up would be desirable to define if EVAR suitability is altered during surveillance, there are inherent challenges in accurately identifying and studying the morphologic changes of small aneurysms as they grow past 5.0 to 5.5 cm. The first and foremost explanation is that the study of such patients is often truncated by surgical intervention for symptomatic aneurysms or rapid expansion. In the ADAM study,[1] 53% of patients who had aneurysms of 45 mm to 49 mm at randomization required surgical intervention at the end of the trial, with more than one-quarter of patients requiring an operation around the two-year mark. With a cohort of patients with similar aneurysm sizes (44.5 mm) at the beginning of our study, we noted a similar 21% cumulative probability for repair at 36 months and 31% cumulative probability for repair at 60 months. The patient survival for our cohort of patients was 78% at 30 months and 61% at 50 months. These two factors decreased the number of patients available for long-term follow-up and prevented a complete evaluation of the natural history of morphologic changes of small aneurysms. Despite these challenges, we were able to effectively document the morphologic changes that occurred in an unselected cohort of patients with small aneurysms under surveillance during the critical period of small aneurysm growth from 4.5 to 5.0 cm.

REFERENCES

1. Lederle FA, Wilson SE, Johnson GR, Reinke DB, Littooy FN, Acher CW, et al. Immediate repair compared with surveillance of small abdominal aortic aneurysms. *N Engl J Med*. 2002;346:1437–1444.

2. Long-term outcomes of immediate repair compared with surveillance of small abdominal aortic aneurysms. *N Engl J Med*. 2002;346:1445–1452.
3. Nowygrod R, Egorova N, Greco G, Anderson P, Gelijns A, Moskowitz A, et al. Trends, complications, and mortality in peripheral vascular surgery. *J Vasc Surg*. 2006;43:205–216.
4. Ouriel K, Srivastava SD, Sarac TP, O'hara PJ, Lyden SP, Greenberg RK, et al. Disparate outcome after endovascular treatment of small versus large abdominal aortic aneurysm. *J Vasc Surg*. 2003;37:1206–1212.
5. Peppelenbosch N, Buth J, Harris PL, van MC, Fransen G. Diameter of abdominal aortic aneurysm and outcome of endovascular aneurysm repair: does size matter? A report from EUROSTAR. *J Vasc Surg*. 2004;39:288–297.
6. Ohki T, Veith FJ, Shaw P, Lipsitz E, Suggs WD, Wain RA, et al. Increasing incidence of midterm and long-term complications after endovascular graft repair of abdominal aortic aneurysms: a note of caution based on a 9-year experience. *Ann Surg*. 2001;234:323–334.
7. Albertini J, Kalliafas S, Travis S, Yusuf SW, Macierewicz JA, Whitaker SC, et al. Anatomical risk factors for proximal perigraft endoleak and graft migration following endovascular repair of abdominal aortic aneurysms. *Eur J Vasc Endovasc Surg*. 2000;19:308–312.
8. Arko FR, Filis KA, Hill BB, Fogarty TJ, Zarins CK. Morphologic changes and outcome following endovascular abdominal aortic aneurysm repair as a function of aneurysm size. *Arch Surg*. 2003;138:651–655.
9. Bayle O, Branchereau A, Rosset E, Guillemot E, Beaurain P, Ferdani M, et al. Morphologic assessment of abdominal aortic aneurysms by spiral computed tomographic scanning. *J Vasc Surg*. 1997;26:238–246.
10. Simons P, van OH, Nawijn A, Bruijninckx B, Knippenberg B. Endovascular aneurysm repair with a bifurcated endovascular graft at a primary referral center: influence of experience, age, gender, and aneurysm size on suitability. *J Vasc Surg*. 2003;38:758–761.
11. Armon MP, Yusuf SW, Latief K, Whitaker SC, Gregson RH, Wenham PW, et al. Anatomical suitability of abdominal aortic aneurysms for endovascular repair. *Br J Surg*. 1997;84: 178–180.
12. Wolf YG, Fogarty TJ, Olcott C, Hill BB, Harris EJ, Mitchell RS, et al. Endovascular repair of abdominal aortic aneurysms: Eligibility rate and impact on the rate of open repair. *J Vasc Surg*. 2000;32:519–523.
13. Welborn MB, III, Yau FS, Modrall JG, Lopez JA, Floyd S, Valentine RJ, et al. Endovascular repair of small abdominal aortic aneurysms: a paradigm shift? *Vasc Endovasc Surg*. 2005;39: 381–391.
14. Long J, Modrall JG, Parker BJ, Swann A, Welborn MB, III, Anthony T. Correlation between ankle-brachial index, symptoms, and health-related quality of life in patients with peripheral vascular disease. *J Vasc Surg*. 2004;39:723–727.
15. Yau FS, Rosero EB, Clagett GP, Valentine RJ, Modrall GJ, Arko FR, et al. Surveillance of small aortic aneurysms does not alter anatomic suitability for endovascular repair. *J Vasc Surg*. 2007;45:96–100.
16. Chaikof EL, Blankensteijn JD, Harris PL, White GH, Zarins CK, Bernhard VM, et al. Reporting standards for endovascular aortic aneurysm repair. *J Vasc Surg*. 2002;35:1048–1060.
17. Chaikof EL, Fillinger MF, Matsumura JS, Rutherford RB, White GH, Blankensteijn JD, et al. Identifying and grading factors that modify the outcome of endovascular aortic aneurysm repair. *J Vasc Surg*. 2002;35:1061–1066.

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Branched and Fenestrated Stent-Grafts

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Branched and fenestrated stent-grafts serve a similar purpose: to maintain perfusion of vital arteries that take origin from an aneurysmal segment of the aorta while excluding the aneurysm itself from the circulation. They also employ similar technology. Yet there exist fundamental differences in the mechanisms of action. For example, fenestrated stent-grafts seal only to the wall of the aorta, whereas branched stent-grafts seal to the walls of aortic branches.

Fenestrated stent-grafts are most useful in cases of juxtarenal aneurysm where the proximal implantation site (the neck) would otherwise be very short. Branched stent-grafts have been used in and around several major branch points, including the aortic arch, the thoracoabdominal aorta, and the iliac bifurcation.

All current stent-grafts represent points on a scale of complexity from aorto-aortic tubes at one extreme to complex multibranched unibody designs at the other. The simplest fenestrated stent-grafts have a single large hole, or marginal indentation (scallop), over one or more aortic branches, while the intact opposite wall of the stent-graft seals entry to a localized defect in the aorta. More extensive aneurysmal involvement of the aorta requires smaller holes, more precise positioning, and closer apposition between the stent-graft around the fenestration and the aorta around the arterial branch. In these cases, a flared *bridging stent* helps maintain the relative positions of the fenestration and the corresponding arterial branch, thereby encouraging *periorificial sealing* while preventing orificial occlusion. Even more extensive aneurysmal involvement of the aorta at the level of vital arterial branches precludes periorificial sealing. In such cases, the only available nondilated implantation sites are within the branches of the aorta. Under these circumstances, bridging stents are insufficient; one needs *bridging stent-grafts*. This combination of a fenestrated stent-graft with a bridging stent-graft results in a rundimentary form of multibranched stent-graft. The addition of a cylindrical cuff around the fenestration improves the connection between the primary stent-graft and the bridging stent-graft, at the cost of limiting the space available for perigraft catheter manipulation. Axially oriented cuffs provide more freedom of

movement outside the stent-graft, but allow access to the cuff from only one direction, above or below (usually above). Very long flexible cuffs can be made to function as branches themselves, thereby eliminating the need for bridging stent-grafts. The result is a unibody branched stent-graft.

The sole advantage of a unibody design is freedom from the risk of component separation. Several disadvantages stem from the complex catheter-based mechanisms of branch deployment. With a few notable exceptions, unibody designs are bulkier and more difficult to deploy correctly. These disadvantages increase exponentially with each additional branch.

HISTORY

The first stent-grafts were simple tubes of uniform diameter with one lumen and two orifices. This configuration was well suited to endovascular repair of descending thoracic aortic aneurysms, which have no major branches, but proved inadequate for the majority of infrarenal aneurysms, which usually lack a segment of nondilated distal aorta.[1] Endovascular AAA repair with an aorto-aortic stent-graft was often achieved at the expense of compromised distal implantation, leading to high rates of leakage and migration. Bifurcated stent-grafts solved this problem by moving the implantation site downstream, out of the aneurysmal aorta and into the iliac arteries. The range of bifurcated stent-grafts contains examples of both unibody (one piece) and modular (multicomponent) systems of branching.

The bifurcated stent-graft experience has benefited from a wide range of designs, vast numbers of patients, and long follow-up. Most of the findings are also applicable to the more complicated multibranched and fenestrated designs. For example, early modular bifurcated stent-grafts, such as the Vanguard (Boston Scientific), were prone to component separation because the overlap zone was short while the limbs were long and flexible. Multibranched designs are exposed to even larger forces, and have an even greater need for long overlap or some locking mechanism to secure the position of one component within another. Hemodynamic forces are also responsible for the high rates of stent-graft migration that have been observed to affect bifurcated stent-grafts such as the AneuRx that lack barb-mediated fixation. In the case of fenestrated stent-grafts, fixation is particularly important because even small degrees of migration would cause branch artery occlusion, endoleak, or both. It is no coincidence that the only widely used fenestrated stent-grafts were based on the most stable endovascular platform, the Zenith, a stent-graft which, in all its forms, has many barbs.

The first fenestrated stent-grafts were described as early as 1996,[2] but widespread application only became feasible with the development of staged deployement[3] and bridging catheter guidance.[4] Since then, usage has expanded to over 35 centers with over 5,000 implantations wordwide. The substitution of short bridging *stent-grafts* for the bridging *stents* of the original fenestrated technique allows fenestrated stent grafts to be used in cases of pararenal, thoracoabdominal, and arch aortic aneurysm.[5]

Another modification of fenestrated stent graft technique involves puncturing the fully expanded stent-graft to create a fenestration in-situ.[6] As yet the only clinical application has been to enhance proximal implantation in a case of thoracic aortic aneurysm. In such cases, the stent-graft was deliberately placed over the subclavian orifice and punctured from a brachial approach with the stiff end of a small-caliber coronary guidewire. The hole was then enlarged using a cutting balloon and stented

open. This approach to subclavian fenestration takes advantage of two features that apply nowhere else; the downstream artery is readily accessible, and the downstream organ tolerates ischemia well. Renal fenestration has been accomplished using a similar technique in dogs, but not without open surgical exposure of the kidneys.

Inoue et al have used multi-branched unibody stent-grafts to treat aneurysms of the aortic arch,[7] thoracoabdominal aorta,[8] and common iliac artery.[9] Whatever the location, the basic approach was the same. First, they constructed a one-piece self-expanding stent-graft to match the arterial anatomy, based on the findings of sophisticated three-dimensional computed tomography (CT). Their ingenious delivery system employed catheter-based control mechanisms to hold the stent-graft in a compressed state while guiding its limbs into their respective branches.

Other systems could be described as both multibranched and unibody,[10] in the sense that they have a component with an inbuilt branch to the internal iliac artery but this component is just one part of an otherwise modular system of aorto-iliac aneurysm repair. Besides, failure to insert the integral iliac branch requires a bridging stent-graft, in which case these systems revert to an entirely modular approach.

We have adapted modular branched technology for use in the iliac artery bifurcation,[11] the thoracoabdominal aorta,[12] and the aortic arch.[13] The specifics vary according from site to site, but these stent grafts share basic features of the Zenith bifurcated AAA device (Cook). In every case, the primary component (main body) has a large proximal orifice and at least two smaller distal cuffs, one for each branch of the aneurysmal artery. The proximal attachment is secured by a barbed Gianturco Z-stent. Additional Z-stents provide the stent-graft with a supportive framework, which maintains the patency, orientation, and cuff position. Multiple markers indicate the orientation of the stent-graft and the positions of key landmarks such as the inner and outer ends of each cuff. When the stent-graft is finally assembled, a bridging stent-graft (or covered stent) connects each distal cuff with its corresponding target artery. Up to that point, the arteries are fed through the unextended cuffs. Flow is never interrupted for more than a few seconds.

Of course, branched and fenestrated stent-grafts are not the only options in cases of branch artery encroachment. Depending on the patient's general health, the size of the aneurysm, and the pattern of anatomic distortion, one may choose to do nothing, perform a traditional surgical reconstruction, implant stents alongside the stent graft into the branch arteries (snorkel or chimney technique) or provide the aortic branches with flow through extra-anatomic bypass while excluding the aneurysm using a simple, nonbranched stent-graft. Examples of this hybrid technique include the use of a femoro-femoral bypass combined with an aorto-uniiliac stent-graft, external to internal iliac bypass with an aorto-external iliac stent-graft,[14] renal bypass with a pararenal stent-graft, visceral bypass with a thoracoabdominal stent-graft, or carotid-subclavian bypass with a distal aortic arch stent-graft.[15] In these combined operations, extra-anatomic surgery is substituting for endovascular branching; more complicated extravascular surgery for a less complicated endovascular intervention.

FENESTRATED STENT-GRAFTS FOR JUXTARENAL ANEURYSM

An adequate length of nondilated aorta (neck) between the renal arteries and the aneurysm is a fundamental requirement for successful endovascular repair. Neck length affects the shape, orientation, fixation, and mural contact of the proximal stent.

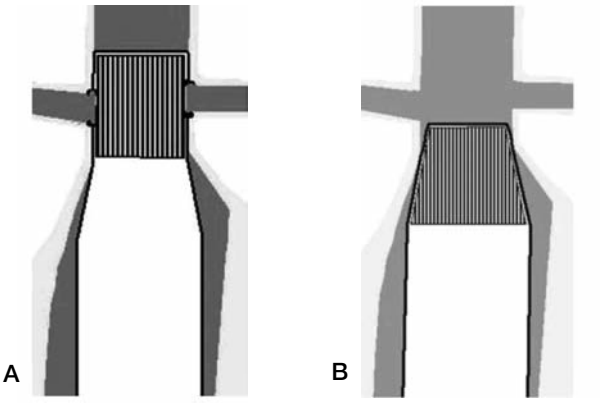


Figure 32-1A. The proximal stent of a fenestrated stent graft conforms to the cylindrical shape of the pararenal aorta. **B.** The same stent assumes a conical shape with very little wall contact when implanted below the renal arteries.

Although selection criteria vary from stent-graft to stent-graft, none can achieve durable hemostatic implantation unless at least half of the proximal stent occupies a neck of uniform diameter. Fenestration permits endovascular treatment in cases of juxtarenal aneurysm by providing a route through the wall of the stent-graft for renal perfusion, which allows the stent-graft to be implanted in a more stable, more cylindrical segment of the aorta (Figure 32–1).

Design Considerations

Small fenestrations demand a high degree of precision in both stent-graft manufacture and placement. Decisions regarding the shape, size, and location of each fenestration require experience and careful attention to detail. Although the processes of stent-graft design and manufacture have been highly refined and standardized, the fenestrated stent-graft will never be an off-the-shelf item. The current technique has three key elements: the staging of expansion to allow movement and instrumentation of the partially deployed graft; the use of bridging catheters to guide the fenestrations to the renal orifices, and the use of bridging stents to maintain proper alignment.

Most of the hemodynamic forces on a typical abdominal aortic stent-graft act at its bifurcation while the untoward effects are felt at its aortic implantation site. The "composite stent-graft" separates these parts of the stent-graft into two overlapping components. The hemodynamic forces on the unbranched proximal component are relatively low, yet it is fixed in position by 10–12 barbs and two renal stents, hence the very low observed rates of migration and renal artery occlusion following endovascular repair with a fenestrated stent-graft. On the other hand, the bifurcated component is the site of changes in shape, diameter, direction, and the velocity of blood flow, which generate large forces. Although it is common to see some slippage between the two aortic components of the fenestrated stent-graft, late-occurring type III endoleak is rare so long as there is a long overlap between relatively stiff components. An alternative approach relies on barbs to secure the intercomponent connection, just as they secure the proximal end of the fenestrated component. The risk of type III endoleak through barb-created holes in the fabric is mitigated by the presence of two fabric layers in areas of overlap.

Since a crossing stent strut would interfere with full expansion of the necessary bridging stents, small fenestrations and scallops (open-ended fenestrations) must occupy the V-shaped spaces between struts. In addition, fenestrations near the end of a stent are too large for the narrow end of one of these V-shaped spaces. Instead, they are placed within the wide end of the adjacent V-shaped space, which is oriented in the opposite direction.

Current Apparatus

The proximal (fenestrated) component (Figures 32–2A, B, C, and D) of a composite stent-graft tapers from the nominal diameter, which depends on the diameter of the neck, to the overlap zone, which is always 24 mm wide. The length of each segment depends on the length of the neck and the length of the infrarenal aorta. The goal is to maximize the overlap between the components. Radioopaque markers indicate the positions of the proximal margin, the distal margin, and any fenestrations or scallops (Figure 32–2A). In addition, a vertical line of markers on the anterior surface (Figure 32–2B) and a transverse line of markers on the posterior surface (Figure 32–2C) indicate axial orientation. The delivery system is essentially the same as for the Zenith AAA main body with the addition of a trigger wire on the diameter-reducing ties (Figure 32–2D).

The distal (bifurcated) component is very like the original (pre-Trifab) version of the Zenith stent-graft, except it has no uncovered proximal stent and the trunk diameter is always 24 mm. Because the stent-graft has no uncovered stent, its delivery system has no cap.

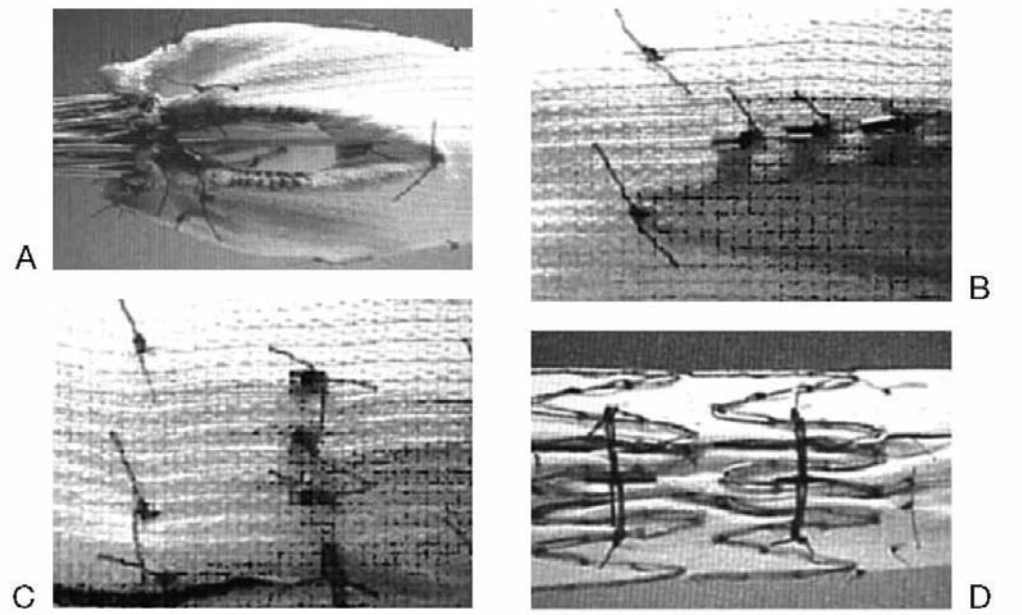


Figure 32-2. The proximal component of the composite fenestrated stent-graft. **A.** Markers around an SMA scallop and two renal fenestrations. **B.** The anterior orientation markers. **C.** The posterior orientation markers. **D.** The diameter-reducing ties.

Procedure

Opinions vary regarding the order of insertion; some insert the bifurcated component first, some insert the fenestrated component first. Component sizing in the overlap zone allows for both.

Proximal component deployment is guided by the positions of the markers and the findings of flush aortography through a catheter in the contralateral femoral artery. Sheath withdrawal uncovers the stent-graft and allows access to the distal orifice, but it does not open completely. The flush catheter is exchanged over a guidewire for a selective catheter, which is then directed into the downstream end of the stentgraft through its downstream end, out through one of the fenestrations, and into the renal artery. A second catheter is introduced, either through the same contralateral femoral sheath or through a separate contralateral femoral artery puncture, and the process of selective renal catheterization repeated for the other renal fenestrations. These bridging catheters are then exchanged for balloons or sheaths before releasing the diameter-reducing ties and allowing the stent-graft to expand fully. Most users prefer sheaths, which provide more predictable access for subsequent bridging stent insertion. The choice of a bridging stent is a matter of individual preference. The delivery system was recently modified to provide space for indwelling catheters and wires that project through the fenestrations into the perigraft space. Cook now manufactures a bridging stent with enhanced flaring, but many others have been used successfully. Moreover, many users now prefer covered stents, which reduce the potential for type III endoleak.

The remaining stages in the deployment of the proximal fenestrated component are just like standard Zenith main body insertion. The same is true of the bifurcated component and its single leg extension. It is usually necessary to take out the first delivery system before inserting the second. Hemostasis is maintained by digital pressure on the arterial puncture site.

Lessons of Experience

Perhaps the most important lesson is not to over extend the indications for fenestrated stent-graft repair. A standard fenestrated stent-graft requires a rim of nondilated aorta below the renal arteries for sealing. A large Palmaz stent can help improve apposition in borderline cases, especially when the underlying problem is aortic angulation. However, there is a potential for interference between the proximal end of the Palmaz stent and the aortic ends of the renal stents. One generally needs to use a relatively short Palmaz stent (P3010) positioned precisely just below the renal artery orifices. Many users now routinely use covered stents to secure the fenestrations, even though the addition of a covering makes the stent and its delivery system more bulky. The addition of a PTFE covering allows the stent to function like a branch and carry blood from the lumen of the stent graft to the lumen of the target artery, even when the stent graft and aorta are not in direct apposition.

A high degree of aortic tortuosity is a relative contraindication for fenestrated repair. In cases of medial-lateral aortic angulation, it can be difficult to predict whether the stent-graft will align itself with the delivery system or with the aorta, andthe final angle of tilt dictates the relative longitudinal positions of the fenestrations and renal arteries.

The success rate of fenestrated AAA repair has been surprisingly high across a broad range of users in many centers around the world. This reflects the reliability of

the technique and the contribution of a high-level support system, involving experienced early adopters and employees of Cook, Australia.

BRANCHED STENT-GRAFTS FOR THE ILIAC BIFURCATION

Unilateral internal iliac occlusion is usually well tolerated due to an extensive network of cross-pelvic collaterals. However, most surgeons avoid bilateral internal iliac artery occlusion for fear of ischemic injury to the colon, lumbosacral plexus, or spine. In cases of bilateral iliac aneurysm, the options include extra-anatomic bypass between the external and internal iliac arteries, combined with external iliac implantation of the distal stent-graft;[14] endovascular bypass between the external and internal iliac arteries, combined with implantation of a contralateral aorto-uniiliac stent-graft and femoro-femoral bypass; and branched endovascular reconstruction of the aneurysmal common iliac artery, with outflow into both internal and external iliac arteries[11]. We favor the latter approach.

Of the three Zenith-based modular branched systems, the bifurcated iliac stent-graft is the furthest along in development. Since we first reported this approach in 2000, several versions have been tried, modified, and tried again in centers around the world. This process of evolution has resulted in a range of designs, all manufactured by Cook, Australia (Figure 32–3).

Design Considerations

The original bifurcated iliac component was a modification of the old long-limb/short-limb modular bifurcated aortic stent-graft with the bifurcation occupying the aneurysmal common iliac artery, not the aorta. The current manufactured component differs from the crude, surgeon-modified original only in the diameter of the trunk, the diameter and orientation of the short (internal iliac) leg.

There are two alternative access sites for insertion of the internal iliac extension: the contralateral femoral artery and a brachial artery. The transfemoral (contralateral)

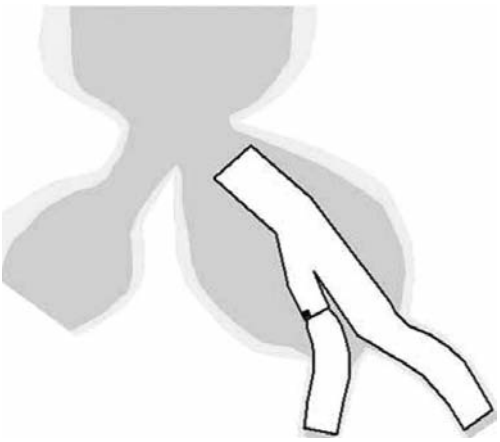


Figure 32-3A. Cross-femoral catheterization of the bifurcated iliac component. **3B.** The reconstructed common iliac artery with a covered stent bridging the gap be-tween the internal iliac cuff of the bifurcated compo-nent and the internal iliac artery.

route is generally shorter and easier. But transfemoral insertion may not be feasible when the angle between the common iliac arteries is unusually acute, or the common iliac aneurysm is shorter than the bifurcated iliac component, which is often the case in East Asia where many patients have short wide common iliac arteries. Shortening the trunk of the bifurcated iliac component is useful in borderline cases, but the resulting inter-component overlap may become unstable. The potential for component separation has been addressed by attaching the iliac bifurcated component to the ipsilateral limb of the aortic bifurcated component (main body), thereby creating a bifurcated-bifurcated aorto-iliac stent graft. This too would be inaccessible from the contralateral femoral artery were it not for the presence of an access hole on the medial aspect of the ipsilateral limb.

Current Apparatus

None of these bifurcated iliac, or aorti-ilaic components is available in the United States, where some surgeons have resorted to back-table modification of Zenith components. Some amputate the proximal end of a main body stent graft, others sew a caudally-oriented cuff onto the iliac limb

Regulatory barriers also limit the range of covered stents for use as iliac extensions. The widest self-expanding Fluency covered stent available in the United States measures 10 mm in diameter. One self-expanding alternative, the Viabahn covered stent, has a range of diameters up to 13 mm. However, a limited range of lengths (25, 50, 100 mm), a large diameter delivery sheath (12 French) and a paucity of markers can make this a difficult device to insert. Balloon-expanded covered stents such as ICAST suffer from a lack of large-diameter size, but this limitation can be overcome by using separate (larger) balloons to achieve further expansion. Of course, the risks involved in this type of off-label use have to be weighed against the risks of the open surgical alternatives.

The Cook, Australia bifurcated iliac component is packaged with a catheter traversing the short (internal iliac artery) cuff and trunk. Once these parts of the stent-graft have been released from their delivery system, the catheter, or its guidewire, can be snared and withdrawn through the contralateral femoral artery. The resulting femoro-femoral guidewire provides additional control for contralateral sheath access. The indwelling catheter is even more important in gaining access through the hole in the bifurcated-bifurcated stent graft An inbuilt flapper valve closes the hole, with or without the aid of an additional stent, following insertion of the internal iliac extension and removal of the contralateral sheaths, wires and catheters.

Procedure

The bifurcated iliac component is used in conjunction with a bifurcated aortic component and the usual iliac extensions for the treatment of aortoiliac aneurysms (Figure 32–3B). The bifurcated iliac component can be placed first or last. If it is placed first, the internal iliac extension can often be inserted through the contralateral femoral artery. The aortic components, or connections to the aortic components, are then inserted through the bifurcated iliac component and implanted within the trunk of the bifurcated iliac component. The main disadvantage of this approach is a slight risk for displacement of the bifurcated iliac component during re-instrumentation. The main advantage is the avoidance of transbrachial access, which is always necessary when the bifurcated iliac component is placed

last because the acute angle between the limbs of an aortic stent graft precludes cross-femoral sheath insertion.

In the manufactured version of this approach, an indwelling catheter and wire enter the trunk of the iliac bifurcated component through the short (internal iliac) limb. This wire is retrieved using a snare from the contralateral femoral sheath, thereby achieving trans-femoral access to the ipsilateral iliac aneurysm through the trunk and short branch of the bifurcated component. The femoral-femoral wire also helps guide sheath insertion and support sheath position during selective internal iliac catheterization and subsequent internal iliac branch insertion. The indwelling catheter of the bifurcated-bifurcated stent graft serves a similar purpose, but instead of entering the free proximal margin of the trunk of a bifurcated iliac component, it enters a hole in the iliac limb of an aorto-iliac component.

Lessons of Experience

The external iliac branch of a bifurcated iliac stent graft is subject to compression and angulation, both of which can restrict flow. The addition of a flexible, self expanding stent appears to be effective in restoring a the lumen of the stent graft, thereby eliminating the risk of graft limb thrombosis. The internal iliac branch is also at risk for angulation, compression and thrombosis. There is evidence that the risk of thrombosis is higher when using helical cuffs, rather than straight cuffs. However, experienced users have achieved good results with both versions of the device. Opinions also vary regarding the relative merits of balloon expanded and self-expanding covered stents in the internal iliac position. Balloon expanded covered stents are certainly easier to delivery, but anecdotal reports of dislocation, kinking and occlusion suggest that the long-term results may be better with self-expanding covered stents.

It is still unclear what role these devices should play in the management of common iliac aneurysms. Some enthusiastic users reconstruct every common iliac aneurysm in an attempt to avoid the buttock claudication that otherwise follows internal iliac embolization. Others reserve this technique for cases of bilateral common iliac aneurysm.

BRANCHED STENT-GRAFTS FOR THE THORACOABDOMINAL AORTA

The modular approach to endovascular repair of a thoracoabdominal aneurysm consists of a series of independent steps. Together, they sometimes add up to a long operation, which takes its toll on the surgical team. But this should be a low stress operation for the patient since no cavities are opened, no organs are deprived of blood flow, and no vessels shed blood rapidly. The incremental nature of the modular technique makes it possible to pause at many points in the procedure, or even bring the patient back another day, if the imaging degenerates or the right catheters and balloons are not immediately available.

This technique has clearly progressed beyond the proof of concept phase. The procedure is now performed at several centers around the world, with low mortality and morbidity rates. Multi-branched endovascular repair is certain to have a prominent role in the management of in patients with extensive (types II and III) thoracoabdominal aneurysms, who often do poorly after conventional surgery. The main barriers to more widespread application have more to do with the cost and availability of the components, than with the relative merits of the technique. Anyone experienced interventionalist with the skill to catheterize a mesenteric artery has the skill to

perform this operation. The long delays involved in customized manufacture, that used to exclude symptomatic patients and patients with very large aneurysms, no longer apply. Nearly everyone can be treated using standardized off-the-shelf components.

Design Considerations

One might expect that the multi-branched thoracoabdominal stent-graft would be a highly customized item, but that is not the case with our preferred approach using axially oriented cuffs and a tapered stent-graft. Variations in the size and distribution of visceral arteries are accommodated by variations in the diameter, length, and position of the separately inserted visceral extensions. Variations in the extent of the aneurysm are accommodated by varying the length, shape and diameter of proximal and distal extensions.

In theory, the cuffs can be short or long, caudally-oriented or cranially oriented, helical or straight, external or internal. They all work and if we were prepared to customize every stent graft we might find applications for them all. However, in practice, we found a short (18 mm long), caudally oriented, barrel-shaped cuff to be sufficiently versatile for use in just about every case.

Balloon-expanded stents, and covered stents, recoil slightly as soon as the balloon is deflated. Since the cuffs are made of inelastic fabric, this recoil has the potential to create a gap, or at least destabilize the intercomponent connection. Moreover, balloon expanded stents are somewhat fragile. Once a balloon-expanded covered stent that has been distorted by the ever-varying forces of the aortic pulse and respiratory cycle, it remains distorted, leading to migration, occlusion, or inter-component separation. Self-exapding stents tend to be more resilient. They can move and bounce back uninjured.

The tortuous route from the brachial artery through the stent-graft to the thoracoabdominal aorta crosses the origins of several arteries to the brain. In order to minimize the risks of coiling in the aortic arch and of embolism to the brain, we employ a series of co-axial sheaths. Once a large sheath is in place between the brachial artery and the proximal stent-graft, the intervening arteries are protected from intra-arterial manipulation. Moreover, the stability imparted to this sheath by the tension in a brachial-femoral guidewire provides support and stability to all the catheters that pass through it (alongside the brachial-femoral guidewire).

Current Apparatus

The assembled thoracoabdominal stent-graft usually has five types of components, one for each of the following locations: the thoracic aorta, the thoracoabdominal (visceral) aortia, the infrarenal aorta, the iliac arteries, and the visceral arteries. The thoracic component bridges the gap between the proximal implantation site and the thoracoabdominal (cuff-bearing) component, usually in cases of type II and type III thoracoabdominal aortic aneurysm. The tubular version of the infrarenal component bridges the gap between the thoracoabdominal component and a distal implantation site, usually in a previously placed surgical graft, whereas the bifurcated version of the infrarenal component provides attachment sites for extensions to the common iliac arteries, usually in the absence of previouis infrarenal repair. The thoracoabdominal component has 4 caudally-oriented cuffs, each of which provides an implantation site for a covered stent to one of the visceral arteries. Like most Zenith-based stent grafts, all the aortic components have barbs proximally, but none has an uncovered stent.

Over the years, we have used a wide range of stent-grafts as visceral extensions. Our first choice, a thin PTFE membrane sandwiched between two Smart stents, was probably the best. The two stents provided robust expansion, flexibility, kink resistance, secure attachment to overlapping arteries and stent-grafts, and radio-opacity. Unfortunately, the device was withdrawn by the manufacturer, Cordis in 2001. We currently use self-expanding Fluency covered stents, lined with self-expanding Wallstents. We have also had occasion to use Viabahn covered stents (large diameters) and Zilver stents.

The wide variety of wire and catheter types on the table at the end of every case reflects the widely varying demands imposed by variations in delivery system size, delivery system flexibility, routes of insertion, and target artery diameter. We generally use Lunderquist wires for insertion of the aortic components, hydrophylic wires for selective visceral artery catheterization, and Rosen wires for insertion of the visceral extensions.

Procedure

The stent graft insertion procedure has two parts: deployment of the aortic and iliac components, followed by deployment of the visceral extensions. The first stage is performed through bilateral femoral access; the second through unilateral (usually the left) brachial access. All three arteries are exposed and repaired surgically. Heparin anticoagulation is maintained from the time of femoral catheter insertion to brachial catheter removal, so we normally prepare, drape, and expose all three access sites at the start of the operation. However, we have occasionally found it necessary to separate the two halves of the procedure completely to make changes in the position of the patient and imaging system.

General anesthesia is preferable to local or regional anesthesia, given the length of the procedure, the surgical exposure of three separate arteries, and the need to hold breathing during visceral angiography. The patient is placed in the supine position with the left arms extended on an arm boards. The C-arm setup has to provide for a wide range of views from full AP to full lateral, and a wide range of fields from the arch to the groin, therefore we usually bring the C-arm in from the head. While inserting the aortic stent grafts, we stand to the right of patient's hips and view the procedure on a pair of screens to the patient's left. While inserting the visceral branches, we stand below the patient's left arm and view the procedure on a screen above the left arm.

Since the devices are inserted from proximal to distal, the delivery system diameters frequently decline as the procedure continues and large (22-24 French) sheaths are replaced by smaller (16-20 French) sheaths. Hemostasis is achieved by tightening Rummeil loops around the femoral arteries. Truly percutaneous repair is not feasible.

Many women, and a few men, need a prosthetic conduit to the central arterial circulation: the external iliac artery is too small to transmit large-diameter delivery systems. We like to separate the necessary access procedure and the device insertion by at least a week to give our fragile patients a chance to recover, we do not like to leave a blind conduit to clot, and we do not like to re-explore a recently-created surgical wound with a prosthetic bypass in its base. We expose the common iliac artery through a paramedian retroperitoneal incision, and attach the proximal end of a 10 mm polyester graft in an end-to-end fashion. The distal anastomosis is made either to the distal external iliac artery or the distal common femoral artery. At the time of stent graft implantation, access to the graft is obtained through the femoral artery, or directly, through a transverse incision just below the inguinal ligament.

Cases of branch artery stenosis or indeterminiate celiac artery patency are addressed at a separate preliminary intervention through the arm. Although, one has to be careful not to place a stent too far into the aorta, the presence of a wide-open radioopaque visceral target speeds the subsequent repair enormously.

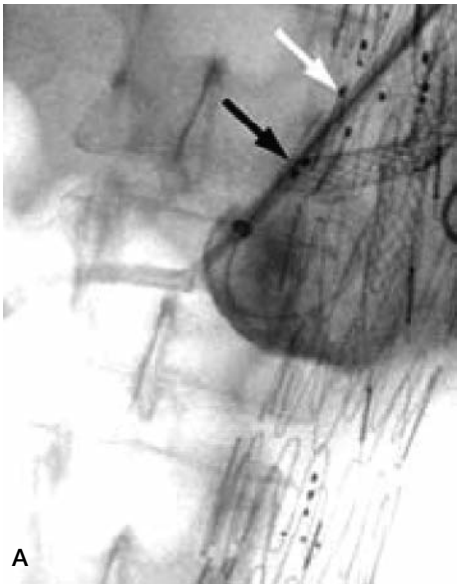
Part 1: Aortic and Iliac Components. The aortic stent grafts are inserted from proximal to distal. The proximal component is placed as low as possible to preserve the maximum number of intercostals arteries, so long as the top end overlaps the aortic implantation site at least 20 mm and the distal end is above the celiac artery. The visceral (cuff-bearing) stent graft is deployed by reference to the position of a selective catheter in one of the visceral branches. The only absolute requirement is that all the caudally oriented cuffs come to lie proximal to the corresponding arterial orifices. On the rare occasion when the distribution of cuffs matches the distribution of visceral artery orifices, we can use any branch as a guide. More often one of the target arteries is a little higher (relative to the other target arteries) than the corresponding cuff (relative to the other cuffs). This is the artery we catheterize, because this one is the most at risk of ending up in an inaccessible location higher than its cuff. We try to orient the stent-graft prior to insertion, and again prior to sheath withdrawal. In theory, it should be possible to reorient the stentgraft following sheath withdrawal, but we try to avoid doing so for fear of creating a kink or twist in the stent graft, or disrupting unstable aortic plaque or thrombus.

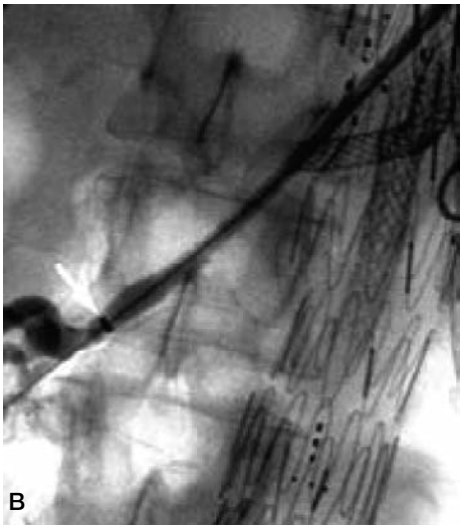
Once all the aortic components (and any ipsilateral iliac limbs) have been inserted, the delivery systems and guidewires are removed, the access site repaired, and flow restored to the ipsilateral femoral artery. The sheath in the contralataral femoral artery is smaller and less likely to obstruct flow. We leave it in place as the exit site for a brachial-femoral guidewire.

Part 2: Visceral Extensions. It can be difficult to track a large (10 or 12 French) sheath along the acutely angled path from the left subclavian artery into the descending thoracic aorta. Having tried various combinations of coaxial sheaths and stiff guidewires, we now routinely use a brachial-femoral guidewire. Once the aortic stent grafts are in place, the selective catheter is withdrawn from the visceral artery, redirected into the lumen of the stent graft, advanced into the aortic arch and replaced with a 5 or 6 French 90 mm-long sheath, which serves as a snare catheter. A multi-loop snare is used to retrieve a floppy 0.035″ guidewire from the aortic arch or distal ascending thoracic aorta. The theoretical potential for embolic stroke has not been borne out by experience. The tensioned 0.035″ guidewire exerts an powerful influence over the left brachial sheath, guiding it atraumatically through the most acute angles. Having served this purpose, the 0.035″ guidewire is exchanged for a 0.014″ guidewire, which is equally stabilizing and less space-occupying. Both ends of the wire are secured by small hemostatic clamps, one of which is connected to the drapes of the arm board by elastic hemostatic loops (the usual rubber spaghetti).

We prefer to use two sheaths: a 12 French 45 cm-long sheath on the outside and a 9 French 70 cm-long sheath on the inside. The brachial-femoral wire exits the center of the 12 French sheath valve, while the 9 French sheath exits the valve of the 12 French sheath through a peripheral puncture site. If the left brachial artery is too small to admit a 12 French sheath we dispense with the inner sheath and use a single 10 French sheath, which is large enough to accommodate the largest Fluency delivery system alongside the 0.014″ wire.

The steps in sheath advancement and covered stent deployment are essentially the same for each branch artery. A catheter is introduced through the 9 French left brachial sheath into the lumen of the stent graft, through a cuff into the perigraft space and from there into the lumen of the target artery. Having confirmed the position angiographically the catheter is exchanged over a stiff guidewire (usually a 0.035 Rosen) for a Fluency covered stent, followed by a Wallstent. The intraoperative angiograms of Figures 32–4A, B, and C show the catheterization and stenting of the right renal artery in a case of thoracoabdominal aneurysm repair. We generally use 6 or 7 mm-wide Fluency covered stents in the renal arteries, and 9 or 10 mm-wide Fluency covered stents in the superior mesenteric and celiac arteries. The length varies according to the distance between the cuff and the target artery orifice and the length of the arterial





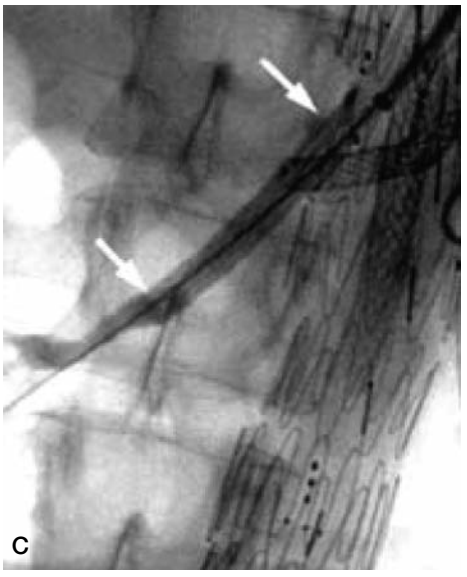


Figure 32-4 A. Intraoperative angiogram showing the lumen of the aneurysm, the proximal right renal artery, and a sheath traversing the right renal cuff between the four markers (white arrow) that surround the inner orifice of the right renal cuff, and the two markers (black arrow) that indicate the position of the outer orifice. **4B.** Intraoperative angiogram showing the tip of the sheath (white arrow) within the right renal artery. **C.** Intraoperative angiogram showing a covered stent between the right renal cuff of the thoracic aortic (upper white arrow) component and the right renal artery (lower white arrow).

trunk. The renal arteries, for example, are sometimes very short (especially on the left). In contrast, the unbranched segment of the superior mesenteric artery is often more than 30 mm long, except in the presence of an aberrant right hepatic artery. The lining Wallstent helps secure the distal implantation by extending further into the target artery, and by incorporating into the arterial wall in a way that the covered Fluency stent cannot.

Lessons of Experience

The modular thoracoabdominal stent graft, with four caudally-oriented cuffs, has proven to be forgiving, versatile and stable. Having inserted over 250 branches with a technical success rate of 100%, we have never seen a branch kink or migrate. These operations used to take eight hours, they now take four. This ascent up the learning curve reflects the combined effects of several helpful tricks, some of which help keep the procedure on track while others help get the procedure back on track when problems occur. However, none of these tricks really reflect an increasing level of skill on the part of the operator. Anyone with experience in endovascular intervention would find them easy enough to master. We used to customize all the stent grafts to match individual patient anatomy. We now use standard components in the vast majority of cases and modify the procedure accordingly. For example, if the thoracoabdominal aorta has only three patent branches, we use an Amplatzer II plug to occlude the fourth.

Endovascular TAAA repair often requires long periods of imaging under high magnification. Mobile C-arms tolerate such high intensity use poorly. Fixed, floor, or ceiling mounted units do better, especially if the patient is obese. One should use a mobile C-arm in a pulsed fluoroscopy mode as much as possible, and it is advisable to have a backup C-arm available.

Some operators find it helpful to view the fluoroscopic image upside down and reversed (left-to-right) during visceral catheterization from a bronchial access point. In this view, clockwise rotation causes the catheter tip to move to the right across the front of the aorta and to the left across the back of the aorta, while counter-clockwise rotation causes the catheter tip to move to the left across the front of the aorta and to the right across the back of the aorta. In other words, the catheter tip appears to behave as it would in the normal, upright view with a catheter inserted from a femoral approach.

The renal arteries frequently curve backwards from the antero-lateral surface of the aorta towards the spine. The renal orifice is most easily catheterized in the contralateral oblique view, but the renal artery itself is best seen in the ipsilataral oblique view. The celiac artery usually takes origin from the anterior surface of the aorta, just to the left of the midline. Celiac catheterization usually requires a steep right anterior oblique view.

Aortic tortuosity, branch artery stenosis, and iliac stenosis all complicate multibranched endovascular repair, but most can be treated, usually at a preliminary operation. Very few patients lack the anatomic substrate for repair. Reasons for exclusion include: an aneurysm smaller than 60 mm in diameter, iliac stenosis all the way up to the the aortic aneurysm, and absent visceral artery implantation sites due to early branching, duplication, or aneurysmal change.

Lower extremity weakness occurs in approximately 20% of patients, regardless of the extent of repair. Most cases show no neurological deficits on waking from anesthesia, but develop symptoms within the first 12 hours. Most have partial weakness, or sensory loss, and a combination of spinal drainage and blood pressure support (fluids

rather than pressors) produces recovery unless the patient has signs of embolism or persistent hypotension (5% of cases). Risk factors for lower extremity weakness include: occlusion of the internal iliac arteries and female gender. We insert a CSF drain prior to repair in every case and regard failure to insert a drain, or bloody drainage, to be grounds for postponing the repair. We never cover the left subclavian artery without first performing a left carotid-subclavian bypass.

BRANCHED STENT-GRAFTS FOR THE AORTIC ARCH

The aortic arch is a challenging site for any form of stent-graft. The affected segment has a high flow rate, a 120-degree bend, multiple branches to organs with no tolerance for ischemia, and it ends proximally at the aortic valve. Any endovascular procedure in the arch has to be quick, predictable, and durable.

We first used a branched stent-graft to treat a symptomatic psuedoaneurysm of the aortic arch is a 61-year-old man whose previous surgery and severe cardiopulmonary disease prevented open repair. Clearly, this was an unusual set of circumstances if only because isolated aneurysms of the aortic arch are rare. A far commoner disease of the aortic arch is type A dissection. If endovascular reconstruction of the ascending aorta and arch is to have an important role, it will be as a means of excluding the intimal tear in acute type A dissection. At this point, one cannot say whether this will be effective, even in the absence of aortic root involvement. Nor can one say whether the stent-graft structure and position will be stable, given the enormous hemodynamic forces typical of the ascending aorta and arch.

Design Considerations

Having experimented with several multibranched designs in perfused rubber models of the aortic arch, we originally opted for the simplest possible form of endovascular repair: a bifurcated stent-graft combined with conventional extra-anatomic bypass. In this approach, a long narrow branch of the stent-graft supplied the brachiocephalic circulation via the innominate artery, while a short wide branch supplied the lower half of the body via the descending thoracic aorta.

We chose transcarotid stent-graft insertion for three reasons. First, the transcarotid route deposits the long narrow limb of the stent-graft in the innominate artery, guaranteeing a source of blood flow to the head and arms. Second, the wide aortic cuff is a large target for catheterization. Transfemoral insertion would have required catheterization of a relatively small innominate arterial cuff, which is a more difficult task, especially when the target orifice is compressed against the outer curve of the stent-graft. Third, a large sheath would be difficult to push around the aortic arch without the usual a long, tapered dilator tip and difficult to orient once the transfemoral delivery system had bent to accommodate the curve of the arch.

Although the intial case went well, subsequent cases revealed some potential disadvantages of this approach. The diameter of the right carotid artery proved to be a major limitation. Many patients with aneurysms of the aortic arch have a wide ascending thoracic aorta, requiring a wide stent graft in a wide delivery system. The carotid artery was too small and the inomminate artery too inaccessible for the kind of minimally invasive surgery needed in such fragile patients. Trans-carotid delivery was

also complicated by a competition for space between the surgeon and the anesthesiologist. There was no room for the usual endovascular runway.

In the meantime, advances in the technology of stent graft manufacture and delivery promised to overcome some of the limitations of trans-femoral insertion. These innovations include: lower profile fabric, lower profile stents, more flexible sheaths and self-orienting delivery systems. A re-assessment of trans-femoral multi-branched arch stent grafts involved in-vitro testing of many prototypes, culminating in recent clinical cases. The current version has 2 cuffs, one for the inomminate artery, the other for the left carotid artery or left subclavian artery, depending on local anatomy.

Patient Selection

The main advantage of this approach is the avoidance of bypass from the ascending aorta, which would require median sternotomy, or bypass from the right femoral artery, which may not be durable. The main requirement for endovascular repair of any kind is a proximal implantation site. Proximal aortic dilatation, dissection, and prior aortocoronary bypass may exclude a patient from consideration. The other main exclusion criterion related to the feasibility of conventional repair. Patients are only considered for branched endovascular repair when aneurysm size precludes observation and severe cardiopulmonary disease and/or prior thoracic surgery precludes open repair.

Current Apparatus

The bifurcated component of the trans-carotid system has a wide proximal trunk, a long narrow limb, and a short wide cuff. The distance from the proximal margin of the trunk to the distal margin of the cuff is approximately 2 cm shorter than the inner (posterolateral) aspect of the ascending aorta. Each part of the stent-graft is oversized 10–15% relative to its implantation site. The trunk is sized to match the ascending aorta, the narrow limb to the innominate artery, the proximal end of the extension to the short wide cuff, and the distal end of the extension to the descending aorta. Both the bifurcated component and the aortic extension are supported by self-expanding stainless steel Gianturco Z-stents. The proximal stent of each aortic stent-graft carries a series of caudally oriented barbs.

The cuff-bearing proximal component of the trans-femoral system has a dog-bone shape. The ends are wide and flexible, while the middle is narrow and stiff. Two cuffs, measuring 12 mm and 8 mm in diameter, project into the lumen of the stent graft.

1. Trans-cervical Insertion of a Bifurcated Stent Graft. In preparation for trans-carotid insertion of a bifurcated stent graft, we extend the territory supplied by the innominate artery by performing retropharyngeal carotid-carotid bypass in combination with left subclavian-carotid implantation or left carotid-subclavian bypass. We favor bypass in any patient who has a LIMA graft to the coronary circulation or a dominant left vertebral artery. If the right carotid artery is too small to accommodate the primary delivery system, we create a conduit to the innominater bifurcation. Gentle traction on the carotid and subclavian arteries brings the innominate artery up into the supraclavicular wound.

We perform the operation with the patient under general endotrachial anesthesia on a radiolucent operating table. The anesthesiologist inserts a temporary cardiac pacemaker percutaneously through the right subclavian vein and an arterial catheter in the right radial artery. The carotid, subclavian, and femoral arteries are exposed

through standard incisions prior to the administration of intravenous heparin in sufficient quantities to maintain activated clotting time at least twice control.

Endovascular stent-graft implantation has two stages, one for each of the aortic components. Both are performed using a steep left anterior oblique view with the patient's left arm extended away from the chest.

We puncture the common carotid artery halfway between its origin from the innominate artery and the carotid-carotid bypass. The sheath for the bifurcated ascending aortic stent-graft can be expected to occlude the carotid artery, in which case flow will reach the distal right carotid artery through the carotid-carotid bypass. Alternatively, if the carotid artery is too small to accommodate the sheath of the bifurcated ascending aortic stent-graft, a conduit can be grafted onto the distal innominate artery. A floppy guidewire is inserted until it rolls back on itself at the aortic valve. This is replaced over a catheter for a very stiff guidewire (Lunderquiest, Cook), which helps brace our rudimentary balloon-catheter based delivery system against the enormous forces generated by ascending aortic blood flow.

The sequence of stent-graft deployment is as follows: sheath/dilator insertion over the guidewire, dilator removal, loading capsule attachment, stent-graft advancement to the sheath tip, stent-graft orientation, sheath withdrawal, trigger wire removal, and balloon catheter removal. Contrast injection through a transfemoral flush aortic catheter allows angiographic visualization of the aortic arch. The key anatomic landmark is the innominate orifice. At the time of deployment, the stent-graft has to be far enough in that the short wide cuff opens into the aorta, and far enough out that the proximal end does not cover the coronary arteries. The positioning and orientation of the stent-graft are both guided by a radio-opaque gold marker on the opposite side of the outer orifice of the short wide cuff from the long thin limb. Of course, stent-graft orientation and position are most easily determined after stent-graft deployment, but, by that time, it is too late to make any changes. The critical step of sheath withdrawal has to be accomplished during a 10–15 second period of adenosine-induced cardiac arrest, or rapid ventriclar pacing. None of the other steps is complicated or time-critical.

The outer orifice of the short wide cuff would be a difficult target for transfemoral catheterization were it any smaller because the catheter also has to negotiate the bends of the distal aortic arch. The 22 mm-wide target helps, but it is easy to be deceived by the passage of a catheter in front of or behind the orifice of the short wide cuff, which give the appearance of successful catheterization on two-dimensional fluoroscopy. Catheter position has to be confirmed using multiple viewing angles and by free rotation of its curved tip inside the trunk of the stent-graft. Only then is the catheter exchanged over a stiff guidewire for a long (transfemoral) sheath/dilator combination. Deployment of the straight descending aortic stent-graft follows the same steps as deployment of the bifurcacted stent-graft. Device positioning is a little easier because the proximal implantation site in the short wide cuff can be identified without contrast, and orientation of the stent-graft is not an issue. Again, deployment occurs during adenosine-induced cardiac arrest.

2. Trans-femoral Insertion of a Multi-branched Stent Graft. Despite the different route of insertion, the two techniques of arch repair have much in common. Both require: left carotid-subclavian bypass, brief periods of cardiac standstill, left ventricular catheterization, large doses of heparin and careful attention to bubble-free technique. Trans-femoral delivery simplifies the room set-up, provides flow directly through branches to both sides of the arch, and eliminates large-bore trans-carotid (or trans-innominate)

access. The only open surgery is left carotid-subclavian bypass. The right side of the neck can be left alone because the inomminate branch of the stent graft can usually be delivered through the right brachial or axillary artery.

The primary (cuff-bearing) component is deployed by reference to two landmarks, the sinotubular ridge and the innominate orifice. The proximal margin of the stent graft has to be at, or distal to, the sinotubular ridge. The innominate cuff (12 mm) has to be at, or proximal to, the innominate ofifice.

The choice of branch extension depends on the size of the target artery. Anything larger than 12 mm in diameter requires a conventional stent graft in the form of an iliac limb from a bifurcated abdominal aortic system. This is more likely to be the case for the innominate and left subclavian arteries. The left carotid artery is usually small enough for a 10 mm-wide Fluency. We routinely line both branches with Wallstents to stabilize branch position and provide luminal support.

Lessons of Experience

Our current experience is too limited to support any conclusions about the potential role of a technique like this. The endovascular procedure is relatively simple especially with the trans-cervical route of insertion. However this procedure still requires extensive surgery and the results have been marred by complications such as stroke. The trans-femoral approach is relatively new and untried, but its advantages are obvious. Time will tell whether this technique lives up to its promise.

CONCLUSION

The characteristic simplicity and versatility of a modular branch point has resulted in the adaptation of branched stent graft technology for use in various segments of the arterial tree where important branches cannot be excluded from the circulation, including the aortic arch, thoracoabdominal aorta and iliac bifurcation. These devices answer a great need because the presence of an arterial branch within the field of repair complicates open surgery at least as much as it complicates endovascular exclusion.

The iliac bifurcated component is something of a gateway technique, in that the device is readily available, the risk is low, and the basic insertion technique relies on the same set of maneuvers used in other branched stent grafts, The thoracoabdominal stent graft has also been used extensively over the past decade and developed to the point where standardized techniques deliver predictably good results. The arch stent graft, on the other hand, remains a work in progress with much promise, but little clinical data to support its widespread use.

Disclosure

Timothy Chuter has licensed patents to Cook, Inc., and receives royalties based on the sales of aortic stent-grafts. David Hartley is an employee of Cook, Australia.

REFERENCES

1. Chuter TAM, Green RM, Ouriel K, DeWeese JA. Infrarenal aortic aneurysm morphology: Implications for transfemoral repair. *J Vasc Surg*. 1993;1994;20:44–50.

2. Park JH, Chung JW, Choo IW, et al. Fenestrated stent-grafts for preserving visceral arterial branches in the treatment of abdominal aortic aneurysms: preliminary experience. *J Vasc Interv Radiol*. 1996;7:823.
3. Stanley BM, Semmens JB, Lawrence-Brown MM, Goodman MA, Hartley DE. Fenestration in endovascular grafts for aortic aneurysm repair: new horizons for preserving blood flow in branch vessels. *J Endovasc Ther*. 2001;8:16–24.
4. Browne TF, Hartley D, Purchas S, et al. A fenestrated covered suprarenal aortic stent. *Eur J Vasc Endovasc Surg*. 1999;18: 445–449.
5. Anderson JL, Berce M, Hartley D. Endoluminal Grafting of Thoracoabdominal Anerysms. *J Endovasc Ther*. 2004;11:I–3.
6. McWilliams RG, Murphy M, Hartley D, Lawrence-Brown MM, Harris PL. In situ stent-graft fenestration to preserve the left subclavian artery. *J Endovasc Ther*. 2004;112:170–4.
7. Inoue K, Hosokawa H, Iwase T, et al. Aortic arch reconstruction by transluminally placed endovascular branched stent graft. *Circulation*. 1999;100S:II316–II321.
8. Inoue K, Iwase T, Sato M, et al. Transluminal endovascular branched graft placement for a pseudoaneurysm: reconstruction of the descending thoracic aorta including the celiac axis. *J Thorac Cardiovasc Surg*. 1997;114:859–861.
9. Iwase, T, Inoue K, Sato M, et al. Transluminal repair of an infrarenal aortoiliac aneurysm by a combination of bifurcated and branched stent grafts. *Catheterization and Cardiovascular Interventions.* 47:491–4, 1999.
10. Brener BJ, Faries P, Connelly T, et al. An in situ adjustable endovascular graft for the treatment of abdominal aortic aneurysms. *J Vasc Surg*. 2002;35:114–119.
11. Abraham CZ, Reilly LM, Schneider DB, et al. A modular multi-branched system for endovascular repair of bilateral common iliac artery aneurysms. *J Vasc Endovasc Ther*. 2003;10: 203–207.
12. Chuter TAM, Gordon RL, Reilly LM, et al. An endovascular system for thoracoabdominal aortic aneurysm repair. *J Endovasc Ther*. 2001;8:25–33.
13. Schneider DB, Curry TK, Reilly LM, et al. Branched endovascular repair of aortic arch aneurysm. *J Vasc Surg*. 2003;37:855.
14. Parodi JC, Ferreira M. Relocation of the iliac artery bifurcation to facilitate endoluminal treatment of abdominal aortic aneurysms. *J Endovasc Surg*. 1999;6(4):342–347.
15. Criado FJ, Barnatan MF, Rizk Y, et al. Technical strategies to expand stent-graft applicability in the aortic arch and proximal descending thoracic aorta. *J Endovasc Ther*. 2002;9: II32–II38.

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Role of Endovascular Aneurysm Repair for Ruptured Abdominal Aortic Aneurysms

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INTRODUCTION

Ruptured abdominal aortic aneurysms (RAAAs) are the 13th leading cause of death in the United States, accounting for more than 15,000 deaths annually. The overall mortality rate for RAAAs is 80 to 90%, whereas survival ranges from 30 to 65% for those patients who reach the hospital alive.[1-4] Since the first repair of an RAAA, the mortality rate has decreased only 3.5% per decade to the current rate of 41%,[2] although this reduction may be due to selection and reporting bias. It is unlikely that the surgical technique for open repair will improve.

The high mortality rate during open repair of ruptured aneurysms is largely attributed to the significant systemic inflammatory response and subsequent multiorgan failure that develops.[5] The loss of both abdominal wall muscle tone[4] and compensated sympathetic activation[6] during the induction of general anesthesia can promote ongoing blood loss. Further blood loss from the retroperitoneal vasculature can occur during dissection through the hematoma. This is exacerbated by the fibrinolytic and acidotic state created by aortic cross-clamping, resultant ischemic-reperfusion injury, and coagulopathy that develops from hypothermia and large volume resuscitation.

Since the first published report of endovascular repair of a ruptured aortic aneurysm (rEVAR) in 1994,[7] several early reports documented the feasibility of this approach, and several retrospective studies and prospective trials have demonstrated its potential benefit over open repair. The ability to perform rEVAR under local anesthesia, with the maintenance of abdominal wall and vascular smooth muscle tone, may promote peritoneal tamponade of bleeding. The decreased aortic occlusion time, diminished blood loss, and better thermoregulation during rEVAR[6,8-9] may all contribute to improvements in surgical mortality.

MULTIDISCIPLINARY ALGORITHM

Personnel and Angiosuite

Paramount to the effective endovascular treatment of an RAAA is the development of a protocol that facilitates the synchronous passage of the patient through the emergency department and computed tomography (CT) scanner to the endovascular suite.[10] The group from Albany noted that the early diagnosis and treatment of RAAA by the emergency department staff and the increased comfort level of the operating room staff made the greatest contribution to the improvement in their ability to perform rEVAR.[11]

An on-call endovascular team of a radiologist, radiology technicians, transport personnel, anesthesiologist, operating room nurses, interventional radiologist, and/or a vascular surgeon with experience in both open and endovascular repair of RAAA must be readily available at all times. In one study, only 3 out of 24 procedures were performed during regular work hours,[12] and in several studies, endovascular repair was not performed in some patients owing to the unavailability of experienced personnel.[13-16]

The hospital should have a dedicated endovascular suite in which open repair can also be performed. This can be in the form of either a mobile imaging unit or preferably a fixed fluoroscopic imaging unit in an operating room. Equipment for both endovascular and open repair should be present. A "rupture kit"[17] for endovascular RAAA repair should be maintained, with an inventory of preferred stent grafts components. This kit should contain device components that the treating physician has prior experience with in elective cases. As a rough guide, large-diameter main body devices with short and long limb lengths should suffice in most emergent cases.

Imaging

Although ultrasonography can demonstrate an aortic aneurysm, it is not a sensitive modality for the detection of extraluminal blood. In addition, it has not been validated in its ability to assess aortic morphology.[18] Therefore, a preoperative CT scan should be obtained in all conscious, hemodynamically stable patients. The presence of a 64-bit multislice CT scanner in the emergency department can greatly facilitate rapid imaging. Although an intravenous contrast–enhanced scan is preferred, diagnosis of an RAAA and measurements for a stent grafts can be performed even with a noncontrast scan. In a 1998 report, 63 minutes were required for an emergent aortic CT scan.[19] Several current studies have shown that a CT scan can now be obtained in 10 to 15 minutes[14-15]

If rEVAR is to be pursued in patients who have not had preoperative imaging, intravascular ultrasound (IVUS)[20] has been used as an alternative imaging modality. Von Segesser has demonstrated IVUS to be as effective as digital subtraction angiography (DSA) in the endovascular treatment of nonruptured AAAs. In a study of 80 patients comparing nonruptured EVAR with either IVUS or DSA, no significant difference in mortality was noted between the groups, and early endoleaks and quantity of contrast material were significantly less with IVUS.[21]

Although Alsac has proposed the use of DSA as a means to reduce preoperative delay,[22] several studies have documented the inability of conventional angiography to evaluate the integrity of the aneurysm sac and to provide sufficient information for accurate stent grafts sizing.[23] In the Nottingham experience, angiographic calibration of aneurysm morphology was performed in three hypotensive patients. The authors

noted that the angiogram did not reveal thrombus or atheroma at the graft landing zones, making graft sizing difficult.[10]

Concern has been raised over the deterioration of the patient during the time needed to obtain preoperative imaging. In one large study, 9% of patients died during transfer to the operating suite once the decision for emergent open surgery was made.[19] However, in a study by Lloyd et al., the median survival was almost 11 hours in patients managed nonoperatively, and 87.5% of patients survived longer than 2 hours after admission.[24] In a prospective study of 100 consecutive patients treated by open surgical repair, no difference in survival was observed between patients who underwent CT scans and those who did not, although a selection bias may have impacted these results because unstable patients were taken emergently to the operating room.[25]

Resuscitation

Approximately one quarter of patients with RAAA will arrive at the hospital hypotensive.[26] Permissive hypotension[9,27] should be practiced during the resuscitation of these patients.[17] Fluid should be restricted to an amount needed to maintain patient consciousness and a systolic blood pressure higher than 80 mmHg (50 to 100 mmHg).[12,28-29] The preferential use of blood products during these resuscitation efforts has been advocated.[28] The role of antihypertensive agents has not been universally defined, although several groups use sodium nitroprusside and nitroglycerin to treat systolic blood pressure higher than 100 mmHg.[30-31] Patients who are unconscious or unable to maintain a systolic blood pressure above 80 mmHg should be immediately transferred to the operating room. The decision to proceed with emergent open repair, placement of an aortic occlusion balloon, or invasive imaging studies will depend on the comfort level of the surgeon and the condition of the patient.

ENDOLUMINAL STRATEGIES

Anatomic Considerations

The anatomic suitability for rEVAR is commonly reported at 60% (18 to 83%).[13-15,32-36] The wide range (18 to 83%) quoted in the literature is due to the different stent grafts systems and anatomic criteria used. Many groups use the same anatomic criteria for rEVAR that is used in elective endovascular aneurysm repair (EVAR) patients. A neck length of greater than 15 mm with a diameter less than 30 to 32 mm and a neck angulation of less than 60 to 90 degrees are commonly reported anatomic limitations. In addition, a common iliac artery diameter less than 22 to 23 mm with an external iliac diameter greater than 7 mm is preferred. At thrombus, more than 40% of the aneurysm circumference and calcification of more than 80% of the aneurysm circumference have been listed as relative contraindications to rEVAR.[14]

More often, the belief is that the morbidity and mortality of delayed conversion to open repair are better than emergent open surgical repair and "therefore" an acceptance of more liberal criteria occurs particularly in regards to the proximal seal zone length. Some authors are even willing to proceed when there is a proximal neck length of >5 mm. Advocates of this more liberal anatomic criteria state that the primary goal is to save the patient's life,[17] whereas others feel that an unacceptable rate of type I endoleaks is seen. With newer stent grafts systems that utilize bare suprarenal stents, a greater number of RAAAs may be suitable for endovascular repair.

Anesthesia

It has been suggested that the improvement in morbidity and mortality seen with rEVAR is due to the use of local anesthesia, which, as stated earlier, promotes peritoneal tamponade. In a nonrandomized, retrospective study, the use of local anesthesia was associated with a shortened ICU stay and improved hemodynamic stability in elective EVAR.[37] The use of local anesthesia during the treatment of rEVAR has been well established.[15,38-39] Kapma et al. were able to avoid general anesthesia in 83% of patients,[13] and Lachat et al. performed bifurcated RAAA repair in 15 out of 21 patients under local anesthesia.[39] The use of local anesthesia in 28 out of 37 patients by the group from Zurich, as stated by Gerassimidis et al., may be the most important factor in avoiding hemodynamic disturbances and increasing the chance of survival.[36]

Pain from the aortic rupture, instrumentation of the aneurysm sac, and lower extremity ischemia have led some authors to recommend general anesthesia during the treatment of rEVAR.[22,30] Pain can cause the adverse physiologic response of hypertension and tachycardia, while the resultant restlessness can lead to motion artifact and inadequate stent positioning.[6,10,22] Alternatively, local anesthesia can be used to achieve femoral access and placement of a sheath, at which time general anesthesia can be instituted for the remainder of the procedure. This allows for access and the ability to place an aortic occlusion balloon if hemodynamic collapse occurs during anesthetic induction. Epidural anesthesia, with its autonomic sympathetic blockade, can exacerbate hemodynamic instability and should be avoided. Additionally, complications can arise from epidural placement in these coagulopathic patients.

Aortic Occlusion Balloon

The placement of an aortic occlusion balloon can be used to control hemodynamic instability from ongoing blood loss. However, aortic balloon occlusion risks renal and splanchnic ischemia, distal embolization, and ischemic-reperfusion injury and does not prevent ongoing blood loss from iliofemoral arteries.[40] Although carbon dioxide contrast injection can help delineate the aortic branches,[29] the loss of aortic blood flow can impact poorly on angiogram quality. For these reasons, several groups prefer definitive hemorrhage control with swift graft deployment.[22,34,36,40] Alternatively, Franks has reported achieving some degree of aortic occlusion through partial deployment of the Zenith or Talent stent grafts.[41]

Aortic occlusion balloons can be placed via either femoral or brachial access. The Montefiore group has demonstrated good success with brachial access, and Okhi routinely places a guidewire into the thoracic aorta through a 5-Fr (French) brachial artery sheath prior to the induction of general anesthesia.[8] Good stabilization of the occlusion balloon is achieved with brachial access; in addition, deflation of the balloon is not required during stent placement.[9,28] However, percutaneous brachial access is difficult in hypotensive patients[29] and brachial artery exploration is often required. Passage through the aortic arch risks cerebral embolization, while unfavorable angulation of the left subclavian artery orifice can make deployment difficult.[10]

The groups from Nottingham, Zurich, and Malmo and our own institution favor transfemoral balloon occlusion.[6,8,39,42] The femoral artery, while offering the largest access to the aorta, can also be catheterized under local anesthesia, and then utilized during the endovascular repair.[42] The sheath-over-balloon and balloon-ahead-of-sheath

techniques of transfemoral aortic occlusion balloon placement have been described elsewhere.[8,43] During the sheath-over-balloon method, a second occlusion balloon can be inflated in the main body of the stent grafts[44] if the balloon and sheath are withdrawn prior to stent grafts deployment. Malina et al. prefer to withdraw the balloon through a sheath after graft deployment but recommend using only stent grafts with bare, barbed suprarenal stents to prevent dislodgement.[29] With either method, the balloon should be supported with the sheath, which is secured outside the body, and should be deflated slowly in unstable patients to prevent hemodynamic collapse.[29,43]

Stent Graft Systems

Both uni-iliac and bi-iliac devices have been used in the endovascular treatment of RAAAs. Aorto-uni-iliac (AUI) stent grafts have the advantages of allowing expeditious introduction and deployment, rapidly controlling bleeding by decreasing the intra-aneurysmal pressure more effectively than bi-iliac devices.[45] AUI stent grafts also may offer a broader applicability by requiring only favorable unilateral iliac anatomy[46-47] and by lowering the learning curve for deployment.[48] Exclusion of contralateral iliac aneurysms can be performed with AUI devices.[49] Several groups have found these qualities to favor the use of AUI devices in the treatment of RAAAs.[14-15] Brandt has even suggested that a reduced stock of components is required.[50] However, a femoro-femoral crossover graft is required with AUI stent grafts, preventing the use of local anesthesia, increasing the rate of wound infections, and creating the potential risk of graft occlusion.[51] It also prolongs the reconstitution of contralateral hypogastric artery blood flow,[52] raising the risk of spinal cord, splanchnic, and lower extremity ischemia.

The use of bi-iliac stent grafts has been supported by the groups from Zurich and Ulm.[39,53] Although the concern of longer operative times has been raised, with bifurcated grafts, this varies by reports.[22,39,14,12] If difficulty is encountered with contralateral limb deployment, a bi-iliac stent grafts can be converted to an AUI device with the placement of an aortic cuff (AneuRx, Excluder, or Zenith AUI converter) across the flow divider.[11,54] Again, it cannot be overemphasized that the devices used for RAAA should be systems that the operator uses routinely for elective EVAR and with which he or she has significant prior experience.

Technical Aspects of rEVAR

Technical success rates of 96 to 100% have been reported in multiple series.[12,34,39,42,54] The patient's chest, abdomen, and thighs should be prepared and draped. Access to the common femoral artery should be obtained either via a standard groin cutdown or percutaneously with an 18-gauge needle directed at the inferomedial aspect of the femoral head. A 6-Fr sheath can be placed into the common femoral artery after advancement of a 0.035-in J-wire into the aorta. Location of the puncture in the common femoral artery should be confirmed with fluoroscopic imaging in an ipsilateral oblique projection. If an occlusion balloon is to be used, a larger sheath should be placed, followed by percutaneous access of the contralateral limb. At our institution, the "preclose" technique is performed using a 10-Fr suture-mediated arterial closure device (Prostar, Abbot Vascular Devices, Redwood City, CA), which is deployed prior to placement of the larger sheaths. Alternatively, two 6-Fr Proglide devices can be used in a similar manner.

After advancing a pigtail catheter over a guidewire, renal artery location can be confirmed with DSA. Exchange of the catheter with a super-stiff guidewire is followed

by positioning and by deployment of a stent grafts as is routinely done in elective situations. The stent grafts should be oversized by 10 to 20% to prevent graft migration and endoleaks. Sealing of a persistent type I endoleak can be accomplished with either a proximal aortic cuff or a large size Palmaz stent.[22,55]

rEVAR Outcomes

Mortality rates of 6.7 to 45% have been observed with rEVAR, with several studies reporting mortality rates of 20% or less.[13,15,17,29,33-34,39,41,53] Several studies have demonstrated a significant reduction in death with rEVAR when compared with open surgical repair.[13,22,41,56] In a systematic review of endovascular vs. open surgical repair of RAAAs, Visser et al. found a reduction in 30-day mortality rate for rEVAR after adjustment for hemodynamic conditions at presentation. This review of ten observational studies, composed of 478 patients, 148 of whom underwent endovascular repair, noted a 30-day mortality of 22% for rEVAR and 38% for open surgical repair.[57] Whereas some have attributed lower mortality rates in rEVAR to the avoidance of general anesthesia,[39] the wide variance may be due to selection bias.

Several groups have also reported a reduction in mortality after the introduction of a protocol that preferentially utilizes EVAR.[15,17,50] In Eindhoven, the mortality was 20% in patients treated preferentially with rEVAR, compared with 40% in the control group, and Lee et al. reported rates of 12% and 37%, respectively.[17]

In addition to improved mortality rates, several studies have demonstrated a significant reduction in the amount of blood loss during rEVAR as compared with open repair.[13,16,33,58-59] Reichart reported an average blood loss of 300 cc in 6 rEVAR vs. 4500 cc in 13 open repairs, whereas Kapma et al. reported 200 cc and 3500 cc, respectively. As a corollary to the decreased blood loss seen in rEVAR, the transfusion requirement in rEVAR has also been reportedly less when compared with that of open repair.[13,16,41,50,58] At our institution, rEVAR was performed on average with 6.6 ± 4.7 units of blood given, whereas open repair required 11.0 ± 5.3 units of blood. As with other reports, in a review of 37 patients treated for RAAA at our institution, Najjar noted a significantly decreased procedure time. On average, rEVAR was completed in 107 ± 30 minutes as compared with 205 ± 31 minutes for open repair.[42] In a systematic review, Visser et al. noted a shorter duration of endovascular procedures compared with open surgical repair (138 minutes vs. 181 minutes).[57] Several groups have also noted a significantly shorter ICU stay[13,15-16,22,33,41,59] and shorter overall hospital stay with rEVAR when compared with conventional repair.[13-15,42]

COMPLICATIONS

Abdominal Compartment Syndrome

Although several studies have shown a decreased rate of pulmonary, hemodynamic, and renal complications with rEVAR when compared with open surgery,[56] significant morbidity still exists. One study, in which an increased complication rate was seen in the endovascular group, suggested that these complications may have resulted in death if open surgery was performed.[52]

Abdominal compartment syndrome (ACS) has been seen in up to 20% of patients undergoing rEVAR.[17,54] The diagnosis is made based on clinical criteria: tense abdomi-

nal distention, oliguria, increased central venous pressure, decreased cardiac output, increased pulmonary capillary wedge pressure, increased peak airway pressure, and bladder pressure greater than 25 mmHg.[60-62] ACS can lead to respiratory, pulmonary, renal, and cardiac dysfunction. A significantly higher mortality rate is seen in patients who develop ACS.[11,54] Factors that have been associated with the development of ACS are the need for an aortic occlusion balloon, the presence of severe coagulopathy, massive transfusion requirements, and the conversion of a bifurcated stent grafts to an AUI device. Because of these findings, Mehta et al. have urged the avoidance of systemic heparinization to decrease the ongoing bleeding from collateral vessels. If one or more of ACS risk factors are present, they perform an on-table laparotomy.[11] In addition to routine, physiologic monitoring, patients who have undergone rEVAR should have hourly bladder pressures recorded to help in the early diagnosis of ACS.

Endoleaks

The development of both early and late endoleak after rEVAR has been reported. In a study of 37 patients, Hechelhammer et al. reported the freedom of endoleak to be 57±8.5% and 48.8±9% at 2 and 4 years. In this study, endoleak was responsible for 58.8% of secondary interventions.[34] Type I endoleaks have been observed in 5 to 25% of patients.[10,12,15,30,34,36,53] This range may be due to the different anatomic criteria in determining patient eligibility for rEVAR. The group from Ulm noted that the rate of type I endoleaks was comparable with that of patients undergoing elective EVAR. Use of a Palmaz stent, conversion to open repair, or/and packing of the aneurysm sac with a thrombogenic sponge or glue have been described as solutions to treat this complication.

The low rate of type II endoleaks seen in rEVAR may be due to the compression and subsequent thrombosis of lumbar arteries by periaortal hematoma.[12,53] However, 20% of patients in Zurich were noted to have late type II endoleak after resorption of the retroperitoneal hematoma. As with elective EVAR, intervention for type II endoleaks is indicated if an increase in size of the aneurysm sac is observed.[63]

End-Organ Ischemia

Spinal cord ischemia, which is seen in 1 to 2.8% of open surgical cases,[64-66] has been observed in up to 11.5% of patients undergoing rEVAR.[52] Higher postoperative mortality (50% vs. 19%)[52] is seen among patients who experience spinal cord ischemia. Hypogastric artery occlusion and prolonged functional aortic occlusion have been shown to be risk factors for cord ischemia. Although studies suggest that unilateral or bilateral internal iliac artery occlusion can be done safely during elective EVAR,[67-68] it has often been performed in a staged fashion, allowing collateral vessel formation. Peppelenbosch et al. have recommended the use of bell-bottom iliac device limbs to avoid hypogastric artery occlusion.[52]

Renal failure has been well documented in open RAAA[4] and is related to hypoperfusion, hypotension, and embolization. The associated mortality is 75%.[69] It has been reported in a similar number of patients (28 to 30%) undergoing rEVAR.[10,39] Its development may be potentially decreased by the use of dilute contrast, carbon dioxide angiography, or IVUS.[38,70] In the Nottingham study, renal failure developed in 6 of 21 patients. Two cases of acute tubular necrosis resolved spontaneously, 2 patients died from renal failure, and 2 with renal failure.[10] More often, this is due to either embolization or ischemia-reperfusion after placement of an aortic occlusion balloon. A similar mechanism may also be involved in the scattered cases of visceral ischemia after rEVAR.

CONCLUSION

EVAR has revolutionized the elective treatment of AAA and has the potential to change the management of RAAAs. Unlike elective repair, rEVAR requires a dedicated multidisciplinary approach as well as an operating room angiosuite and a readily available on-the-shelf supply of endografts. Allowing for more liberal anatomic criteria, particularly in regard to proximal neck length, will expand the application of rEVAR and potentially change the morbidity and mortality of RAAA. Adjuncts such as permissive hypotension, local anesthesia, aortic balloon occlusion, and avoidance of a laparotomy seem to improve the outcome of patients undergoing rEVAR; however, a unique complication of this therapy is the development of ACS. Several institutional centers have reported on the successful results of this approach and it is hoped that this review will provide some guidance in establishing a system applicable to the reader's site.

REFERENCES

1. Dardik A, Burleyson GP, Bowman H, et al. Surgical repair of ruptured abdominal aortic aneurysms in the state of Maryland: factors influencing outcome among 527 recent cases. J Vasc Surg 1998;28:413–20; discussion 420–1.
2. Bown MJ, Sutton AJ, Bell PR, Sayers RD. A meta-analysis of 50 years of ruptured abdominal aortic aneurysm repair. Br J Surg 2002;89:714–30.
3. Darling RC 3rd, Cordero JA Jr, Chang BB, et al. Advances in the surgical repair of ruptured abdominal aortic aneurysms. Cardiovasc Surg 1996;4:720–3.
4. Gloviczki P, Pairolero PC, Mucha P Jr, et al. Ruptured abdominal aortic aneurysms: repair should not be denied. J Vasc Surg 1992;15:851–57; discussion 857–9.
5. Bown MJ, Nicholson ML, Bell PR, Sayers RD. The systemic inflammatory response syndrome, organ failure, and mortality after abdominal aortic aneurysm repair. J Vasc Surg 2003;37:600–6.
6. Hinchliffe RJ, Braithwaite BD, Hopkinson BR. The endovascular management of ruptured abdominal aortic aneurysms. Eur J Vasc Endovasc Surg 2003;25:191–201.
7. Yusuf SW, Whitaker SC, Chuter TA, Wenham PW, Hopkinson BR. Emergency endovascular repair of leaking aortic aneurysm. Lancet 1994;344:1645.
8. Ohki T, Veith FJ. Endovascular grafts and other image-guided catheter-based adjuncts to improve the treatment of ruptured aortoiliac aneurysms. Ann Surg 2000;232:466–79.
9. Veith FJ, Ohki T. Endovascular approaches to ruptured infrarenal aortoiliac aneurysms. J Cardiovasc Surg (Torino) 2002;43:369–78.
10. Hinchliffe RJ, Yusuf SW, Macierewicz JA, et al. Endovascular repair of ruptured abdominal aortic aneurysm—a challenge to open repair? Results of a single centre experience in 20 patients. Eur J Vasc Endovasc Surg 2001;22:528–34.
11. Mehta M, Darling RC 3rd, Roddy SP, et al. Factors associated with abdominal compartment syndrome complicating endovascular repair of ruptured abdominal aortic aneurysms. J Vasc Surg 2005;42:1047–51.
12. Scharrer-Pamler R, Kotsis T, Kapfer X, et al. Endovascular stent grafts repair of ruptured aortic aneurysms. J Endovasc Ther 2003;10:447–52.
13. Kapma MR, Verhoeven EL, Tielliu IF, et al. Endovascular treatment of acute abdominal aortic aneurysm with a bifurcated stentgraft. Eur J Vasc Endovasc Surg 2005;29:510–5.
14. Reichart M, Geelkerken RH, Huisman AB, et al. Ruptured abdominal aortic aneurysm: endovascular repair is feasible in 40% of patients. Eur J Vasc Endovasc Surg 2003;26:479–86.
15. Peppelenbosch N, Yilmaz N, van Marrewijk C, et al. Emergency treatment of acute symptomatic or ruptured abdominal aortic aneurysm. Outcome of a prospective intent-to-treat by EVAR protocol. Eur J Vasc Endovasc Surg 2003;26:303–10.

16. Resch T, Malina M, Lindblad B, et al. Endovascular repair of ruptured abdominal aortic aneurysms: logistics and short-term results. J Endovasc Ther 2003;10:440–6.
17. Lee WA, Hirneise CM, Tayyarah M, et al. Impact of endovascular repair on early outcomes of ruptured abdominal aortic aneurysms. J Vasc Surg 2004;40:211–5.
18. Shuman WP, Hastrup W Jr, Kohler TR, et al. Suspected leaking abdominal aortic aneurysm: use of sonography in the emergency room. Radiology 1988;168:117–9.
19. Bradbury AW, Makhdoomi KR, Adam DJ, et al. Twelve-year experience of the management of ruptured abdominal aortic aneurysm. Br J Surg 1997;84:1705–7.
20. Peppelenbosch N, Geelkerken RH, Soong C, et al. Endograft treatment of ruptured abdominal aortic aneurysms using the Talent aortouniiliac system: an international multicenter study. J Vasc Surg 2006;43:1111–23; discussion 1123.
21. von Segesser LK, Marty B, Ruchat P, et al. Routine use of intravascular ultrasound for endovascular aneurysm repair: angiography is not necessary. Eur J Vasc Endovasc Surg 2002;23:537–42.
22. Alsac JM, Desgranges P, Kobeiter H, Becquemin JP. Emergency endovascular repair for ruptured abdominal aortic aneurysms: feasibility and comparison of early results with conventional open repair. Eur J Vasc Endovasc Surg 2005;30:632–9.
23. Hinchliffe RJ, Hopkinson BR. Ruptured abdominal aortic aneurysm. Time for a new approach. J Cardiovasc Surg (Torino) 2002;43:345–7.
24. Lloyd GM, Bown MJ, Norwood MG, et al. Feasibility of preoperative computer tomography in patients with ruptured abdominal aortic aneurysm: a time-to-death study in patients without operation. J Vasc Surg 2004;39:788–91.
25. Boyle JR, Gibbs PJ, Kruger A, et al. Existing delays following the presentation of ruptured abdominal aortic aneurysm allow sufficient time to assess patients for endovascular repair. Eur J Vasc Endovasc Surg 2005;29:505–9.
26. Lawrie GM, Morris GC Jr, Crawford ES, et al. Improved results of operation for ruptured abdominal aortic aneurysms. Surgery 1979;85:483–8.
27. Crawford ES. Ruptured abdominal aortic aneurysm. J Vasc Surg 1991;13:348–50.
28. Greenberg RK, Srivastava SD, Ouriel K, et al. An endoluminal method of hemorrhage control and repair of ruptured abdominal aortic aneurysms. J Endovasc Ther 2000;7:1–7.
29. Malina M, Veith F, Ivancev K, Sonesson B. Balloon occlusion of the aorta during endovascular repair of ruptured abdominal aortic aneurysm. J Endovasc Ther 2005;12:556–9.
30. Van Herzeele I, Vermassen F, Durieux C, et al. Endovascular repair of aortic rupture. Eur J Vasc Endovasc Surg 2003;26:311–6.
31. Lombardi JV, Fairman RM, Golden MA, et al. The utility of commercially available endografts in the treatment of contained ruptured abdominal aortic aneurysm with hemodynamic stability. J Vasc Surg 2004;40:154–60.
32. Rose DF, Davidson IR, Hinchliffe RJ, et al. Anatomical suitability of ruptured abdominal aortic aneurysms for endovascular repair. J Endovasc Ther 2003;10:453–7.
33. van Sambeek MR, van Dijk LC, Hendriks JM, et al. Endovascular versus conventional open repair of acute abdominal aortic aneurysm: feasibility and preliminary results. J Endovasc Ther 2002;9:443–8.
34. Hechelhammer L, Lachat ML, Wildermuth S, et al. Midterm outcome of endovascular repair of ruptured abdominal aortic aneurysms. J Vasc Surg 2005;41:752–7.
35. Veith FJ, Ohki T, Lipsitz EC, et al. Treatment of ruptured abdominal aneurysms with stent grafts: a new gold standard? Semin Vasc Surg 2003;16:171–5.
36. Gerassimidis TS, Papazoglou KO, Kamparoudis AG, et al. Endovascular management of ruptured abdominal aortic aneurysms: 6-year experience from a Greek center. J Vasc Surg 2005;42:615–23; discussion 623.
37. Bettex DA, Lachat M, Pfammatter T, et al. To compare general, epidural and local anaesthesia for endovascular aneurysm repair (EVAR). Eur J Vasc Endovasc Surg 2001;21:179–84.
38. Henretta JP, Hodgson KJ, Mattos MA, et al. Feasibility of endovascular repair of abdominal aortic aneurysms with local anesthesia with intravenous sedation. J Vasc Surg 1999;29: 793–8.

39. Lachat ML, Pfammatter T, Witzke HJ, et al. Endovascular repair with bifurcated stent grafts under local anaesthesia to improve outcome of ruptured aortoiliac aneurysms. Eur J Vasc Endovasc Surg 2002;23:528–36.
40. Schonholz C, Donnini F, Naselli G, et al. Acute rupture of an aortic false aneurysm treated with a stent grafts. J Endovasc Surg 1999;6:293–6.
41. Franks S, Lloyd G, Fishwick G, et al. Endovascular treatment of ruptured and symptomatic abdominal aortic aneurysms. Eur J Vasc Endovasc Surg 2006;31:345–50.
42. Najjar SF, Mueller KH, Ujiki MB, et al. Percutaneous endovascular repair of ruptured abdominal aortic aneurysms. Arch Surg 2007;142:1049–52.
43. O'Donnell ME, Badger SA, Makar RR, et al. Techniques in occluding the aorta during endovascular repair of ruptured abdominal aortic aneurysms. J Vasc Surg 2006;44:211–5.
44. Zarins CK, White RA, Schwarten D, et al. AneuRx stent grafts versus open surgical repair of abdominal aortic aneurysms: multicenter prospective clinical trial. J Vasc Surg 1999;29: 292–305; discussion 306–8.
45. Gawenda M, Heckenkamp J, Zaehringer M, Brunkwall J. Intra-aneurysm sac pressure—the holy grail of endoluminal grafting of AAA. Eur J Vasc Endovasc Surg 2002;24:139–45.
46. Armon MP, Yusuf SW, Latief K, et al. Anatomical suitability of abdominal aortic aneurysms for endovascular repair. Br J Surg 1997;84:178–80.
47. Moore WS, Brewster DC, Bernhard VM. Aorto-uni-iliac endograft for complex aortoiliac aneurysms compared with tube/bifurcation endografts: results of the EVT/Guidant trials. J Vasc Surg 2001;33:S11–20.
48. Forbes TL, DeRose G, Kribs SW, Harris KA. Cumulative sum failure analysis of the learning curve with endovascular abdominal aortic aneurysm repair. J Vasc Surg 2004;39:102–8.
49. Chuter TA, Faruqi RM, Reilly LM, et al. Aortomonoiliac endovascular grafting combined with femorofemoral bypass: an acceptable compromise or a preferred solution? Semin Vasc Surg 1999;12:176–81.
50. Brandt M, Walluscheck KP, Jahnke T, et al. Endovascular repair of ruptured abdominal aortic aneurysm: feasibility and impact on early outcome. J Vasc Interv Radiol 2005;16: 1309–12.
51. Walker SR, Braithwaite B, Tennant WG, et al. Early complications of femorofemoral crossover bypass grafts after aorta uni-iliac endovascular repair of abdominal aortic aneurysms. J Vasc Surg 1998;28:647–50.
52. Peppelenbosch N, Cuypers PW, Vahl AC, et al. Emergency endovascular treatment for ruptured abdominal aortic aneurysm and the risk of spinal cord ischemia. J Vasc Surg 2005;42:608–14.
53. Orend KH, Kotsis T, Scharrer-Pamler R, et al. Endovascular repair of aortic rupture due to trauma and aneurysm. Eur J Vasc Endovasc Surg 2002;23:61–7.
54. Mehta M. TJ, Spirig A., Sternbach Y., et al. EVAR Treatment of ruptured AAAs and TAAs. Endovasc Today 2007; 2007:5.
55. Dias NV, Resch T, Malina M, et al. Intraoperative proximal endoleaks during AAA stent grafts repair: evaluation of risk factors and treatment with Palmaz stents. J Endovasc Ther Jun 2001;8:268–73.
56. Greco G, Egorova N, Anderson PL, et al. Outcomes of endovascular treatment of ruptured abdominal aortic aneurysms. J Vasc Surg 2006;43:453–9.
57. Visser JJ, Bosch JL, Hunink MG, et al. Endovascular repair versus open surgery in patients with ruptured abdominal aortic aneurysms: clinical outcomes with 1-year follow-up. J Vasc Surg 2006;44:1148–55.
58. Vaddineni SK, Russo GC, Patterson MA, et al. Ruptured abdominal aortic aneurysm: a retrospective assessment of open versus endovascular repair. Ann Vasc Surg 2005;19: 782–6.
59. Yilmaz N, Peppelenbosch N, Cuypers PW, et al. Emergency treatment of symptomatic or ruptured abdominal aortic aneurysms: the role of endovascular repair. J Endovasc Ther 2002;9:449–57.

60. Kron IL, Harman PK, Nolan SP. The measurement of intra-abdominal pressure as a criterion for abdominal re-exploration. Ann Surg 1984;199:28–30.
61. Loftus IM, Thompson MM. The abdominal compartment syndrome following aortic surgery. Eur J Vasc Endovasc Surg 2003;25:97–109.
62. Ivatury RR, Diebel L, Porter JM, Simon RJ. Intra-abdominal hypertension and the abdominal compartment syndrome. Surg Clin North Am 1997;77:783–800.
63. van Marrewijk C, Buth J, Harris PL, et al. Significance of endoleaks after endovascular repair of abdominal aortic aneurysms: the EUROSTAR experience. J Vasc Surg 2002;35: 461–73.
64. Johnston KW. Ruptured abdominal aortic aneurysm: six-year follow-up results of a multicenter prospective study. Canadian Society for Vascular Surgery Aneurysm Study Group. J Vasc Surg 1994;19:888–900.
65. Noel AA, Gloviczki P, Cherry KJ Jr, et al. Ruptured abdominal aortic aneurysms: the excessive mortality rate of conventional repair. J Vasc Surg 2001;34:41–6.
66. Hans SS, Huang RR. Results of 101 ruptured abdominal aortic aneurysm repairs from a single surgical practice. Arch Surg 2003;138:898–901.
67. Mehta M, Veith FJ, Ohki T, et al. Unilateral and bilateral hypogastric artery interruption during aortoiliac aneurysm repair in 154 patients: a relatively innocuous procedure. J Vasc Surg 2001;33:S27–32.
68. Wolpert LM, Dittrich KP, Hallisey MJ, et al. Hypogastric artery embolization in endovascular abdominal aortic aneurysm repair. J Vasc Surg 2001;33:1193–8.
69. Barratt J, Parajasingam R, Sayers RD, Feehally J. Outcome of acute renal failure following surgical repair of ruptured abdominal aortic aneurysms. Eur J Vasc Endovasc Surg 2000; 20:163–8.
70. Yusuf SW, Whitaker SC, Hinwood D, et al. Carbon dioxide: an alternative to iodinated contrast media. Eur J Vasc Endovasc Surg 1995;10:156–61.

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Endoconversion after Open and Endovascular Repair

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BACKGROUND

The introduction of endovascular repair of abdominal aortic aneurysms (EVAR) in the early 1990s revolutionized the field of vascular surgery. EVAR was initially intended for high-risk patients who were poor candidates for open aneurysm repair. As technology and techniques advanced, EVAR has gained popularity. In 2003, over 40% of elective abdominal aortic aneurysms were repaired using the endovascular technique.[1] It is now widely accepted that in patients with suitable anatomy, EVAR is a reasonable alternative to open surgery because of lower perioperative morbidity and mortality.[2] However, despite improved short term outcomes, EVAR is associated with increased incidence of secondary intervention. The Lifeline Registry of Endovascular Aneurysm Repair analyzed 2,664 patients who were treated with endografts in four Investigational Device Exemption clinical trials in the United States, and reported that 18% of patients require secondary interventions after EVAR.[3] Fortunately, the majority of secondary interventions is minor and can utilize endovascular techniques for treatment.

On the other hand, open AAA is regarded as a more durable procedure with significantly less incidence of secondary interventions. Nevertheless, long-term follow-up of patients that underwent open AAA repair, reveals the formation of para-anastomotic aneurysms and aneurysm formation in the arteries proximal and distal to the aortic graft in a small but significant number of patients.[4]

The term surgical, open conversion is generally reserved for *instances when the entire or a portion of the endograft is surgically removed and circulation is restored by conventional open surgical techniques*. In this chapter, we will review the so-called "endovascular conversion". In this procedure, a previously implanted aortic graft (whether done via an open surgical approach or by endovascular techniques) is partially or completely relined by one or a combination of several stent grafts.

INDICATIONS

Endoconversion Following Open Repair

Indications for endoconversion following open repair include para-anastomotic aneurysm, aneurysm formation of the native proximal/distal aorta or iliac vessels, and aortoenteric fistula formation. In long-term follow-up of patients after open AAA repair, the incidence of proximal para-anastomotic aneurysm formation is approximately 1–15%, the majority of which is false aneurysms.4-9 Risk factors for development include end-to-side anastomosis, local endarterectomy, compliance mismatch between native artery and prosthesis, and graft infection.10 Reports of mortality with open repair are between 20 and 24% and up to 88% when emergent repair is required for pseudo-aneurysm rupture.7,11-14 This is in part due to technical difficulty of dissection in a previously operated field, and the delayed nature of presentation resulting in an older and higher risk patient population. Several reports indicate that the endovascular approach to treat these complications after open AAA repair is associated with both technical success and improved morbidity and mortality rates (Table 34–1).

Endoconversion Following EVAR

Potential indications for endoconversion following EVAR include persistent endoleak with sac enlargement (especially those caused by graft failure) or endotension without endoleak, graft migration, graft stenosis, or thrombosis. Although usually repaired by open techniques, aneurysm rupture following EVAR can also be approached endovascularly. Lagana et al. reported successful endovascular treatment for rupture following EVAR in two patients (incidence 0.6%).[15] In both cases, the patients presented with hypotension, back pain, and on-table angiogram demonstrated endoleak. The source of endoleak was successfully excluded in both cases without complication and follow-up imaging at three and six months, respectively, demonstrated persistent exclusion of the aneurysm.

PREOPERATIVE EVALUATION

Symptomatology and risk of rupture determines the urgency with which intervention must be undertaken. In patients who have evidence of rupture and are unstable, immediate intervention is required. Most endovascular centers have developed protocols for the management of patients with suspected AAA rupture.[16] Endovascular teams with extensive experience on EVAR are readily available to respond. The expeditious use of CT scan in the emergency department, or in some operating rooms equipped with it, facilitates definition of the anatomy. The use of "permissive hypotension" cannot be overemphasized. Some centers have found that rehearsing on elective cases simulating an emergency situation is a useful tool for the formation of endovascular emergency teams. In patients without the suspicion of rupture, perioperative cardiac optimization and detailed definition of vascular anatomy is crucial. High resolution CT scan with IV contrast is ideal for this purpose and will help determine whether a patient is suitable for endovascular intervention. Subsequent angiography may also be required for further diagnosis if determination of endoleak type is not possible with CT.

TABLE 34-1. SOME SERIES OF ENDOVASCULAR INTERVENTION AFTER CONVENTIONAL OPEN REPAIR

First Author	Date	Total number of patients	Indication for intervention (number of patients)	Mean time to presentation from initial repair (range)	30-day mortality	Mean follow-up (months)	Percent procedural success	Complications (number of patients)
Tiesenhausen[25]	2001	3	PAA (3)	11.3 years (8-15)	0%	16	100%	Endoleak requiring re-stent(1)
Pearce[26]	2005	5	PAA (2), hypogastric aneurysm (2), aneurysmal graft (1)	16.6 years (10-25)	0%	24	100%	None
El Sakka[21]	2008	10	PAA (4), AEF (5), thoracic aneurysm (1)	48 months (5-168)	10%	28	100%	Readmission for bacteremia (5 AEF patients)
Zhou[27]	2006	6	PAA (6)	15 years (12-20)	0%	10	83%	Endoleak which resolved (1)
Morrissey[28]	2001	28	PAA (28)	8 years (1-18)	3.5%	21	97%	Endoleak (1), graft thrombosis (1), necrosis of adjacent structures from pseudo-aneurysm (2), hematoma (1), MI (1)
van Herwaarden[10]	2004	14	PAA(6), iliac aneurysm (7), PAA and iliac aneurysm (1)	7 years (4-18)	0%	12	86%	Graft thrombosis (1), late conversion to open for graft failure (2)
Magnan[9]	2003	10	PAA (10)	15 years (10-20)	0%	17.7	100%	Occlusion iliac endograft (1), stenosis distal aortounilateral iliac graft (1)
Piffaretti[29]	2007	19	PAA (19)	8.6 years (3 months-18 years)	0%	14	100%	Occlusion iliac branch (1)

Abbreviations: PAA = para-anastomotic aneurysm, AEF = aorto-enteric fistula, MI = myocardial infarction

TECHNIQUE

Partial Relining

In cases of type I endoleak associated with a poorly positioned graft either due to kinking, migration, or enlargement of the artery, placement of a cuff or extension graft is most commonly performed.[17] In proximal placement of an aortic cuff, position in relation to the prior graft and the renal arteries is a key concern. Accurate deployment to effectively restore seal and fixation without covering the renal arteries requires careful correction of parallax and precise delineation of the anatomic landmarks. This can be done accurately in most cases with conventional femoral access, but some authors[18] recommend obtaining both additional upper extremity access and catheterization of one or both of the renal arteries to facilitate renal artery stent placement following cuff deployment if necessary.[18] Similarly, Minion et al. described the so-called "endowedge" technique in which deployment of the aortic component of a scalloped endograft is done while a balloon remains inflated inside the lowest renal artery via a brachial access.[19] Placement of a distal extension is usually performed for distal or type IB endoleaks.

Complete Relining

In some cases of type III endoleaks such as device failure, or in cases of endotension without endoleak, it is feasible to correct the abnormality by entirely relining the existing endograft with a new device by first deploying an aortic cuff inside the main body of the endograft, and then inserting new iliac limbs overlapping the aortic cuff with extension into the common iliac arteries (Figure 34–1). When the relining needs to be extended into the external iliac artery, and in order to prevent the development of a type II endoleak,

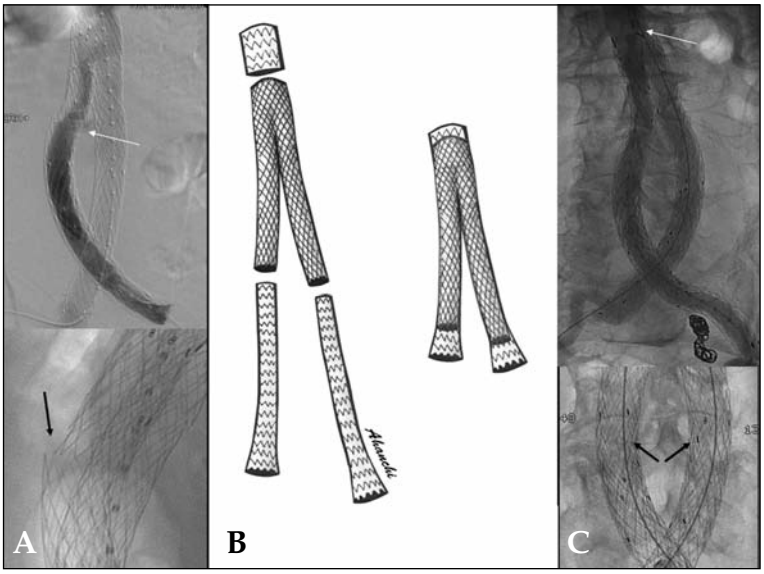


Figure 34-1. Endoconversion by relining. **(A)** Selective angiogram showing a type III endoleak (white arrow) caused by device failure in the right limb (black arrow) of an AneuRx device (Medtronic, Santa Rosa, CA). **(B)** Relining is achieved by deploying an aortic cuff above the bifurcation of the endograft and then inserting iliac limbs bilaterally. **(C)** Completion angiogram after placement of an Excluder (Flagstaff, AZ) aortic cuff (white arrow) and two 16 mm diameter iliac limbs (black arrows).

endovascular occlusion of the internal iliac artery (IIA) is sometimes needed. If there is concern for the subsequent development of pelvic ischemia, embolization of the IIA can be performed prior to relining in a separate procedure to promote formation of collateralization. Alternatively, an internal iliac bypass or transposition is also possible.

Conversion to an Aortouniiliac Endograft with Femoral Crossover

In this technique, a new aortouniiliac graft is placed within the preexisting graft with or without occlusion of the contralateral limb. A femoral-femoral or iliofemoral bypass is then performed to restore flow. Data suggest that patency rates of the femoral-femoral bypass are improved when compared to femoro-femoral bypass performed for occlusive disease.[20] This technique is useful in patients with migration and severe limb kinking who have failed treatment using aortic cuffs in cases of type I endoleak or in limb disconnection. The use of an AAA converter (Zenith Flex and/or Zenith Renu, Cook Inc. Bloomington, IN) deploying its distal portion into one iliac limb, in combination with an iliac plug (Zenith Flex, Cook Inc. Bloomington, IN) in the contralateral external iliac to avoid retrograde sac perfusion and a femoral crossover bypass to revascularize the contralateral leg, is an effective technique that avoids aortic clamping and endograft explantation (Figure 34–2). Alternatively, a bifurcated endograft can be used to convert to an aortouniiliac configuration by deploying both the ipsilateral limb and the contralateral gate in one of the limbs of the existing endograft (Figure 34–3). Another option is to place one or several aortic cuffs can be placed inside the original graft, occluding the origin of the contralateral gate.

Special Considerations for Endoconversion Following Open Repair

Treatment of complications following open repair with endoconversion involves use of the various techniques described above but with some unique considerations. Paraanastomotic or native aneurysm formations proximal or distal to an aortic graft are potential indications for endoconversion following open repair. These complications of open repair can be amenable to the use of an aortic cuff (Figure 34–4) and/or placement of an aortouniiliac or bifurcated graft. As in patients following EVAR, a sufficient length of proximal aneurysm neck between the renal arteries is necessary to prevent subsequent renal ischemia after deployment. In a series of patients by Van Herwaarden et al.,

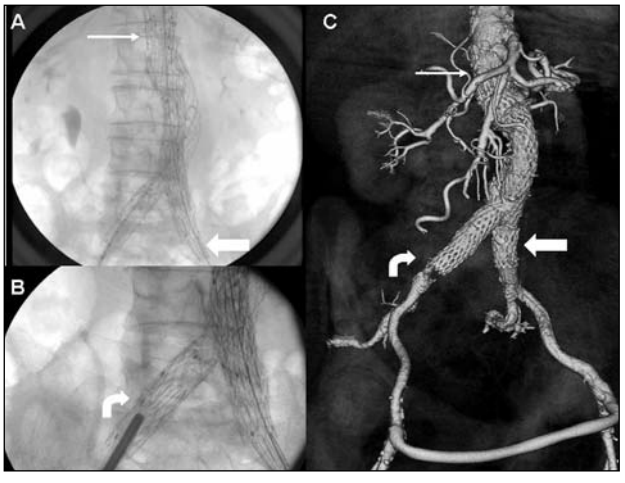


Figure 34-2. Radiographs and CT scan showing a case of endoconversion to an aortouniiliac device. **(A)** A Renu device (Cook Inc.) was deployed from the left femoral access site into the right limb (crossed) of the original endograft. The bare proximal attachment site (thin white arrow) is deployed across the renal vessels and the distal attachment (thick white arrow) in the native iliac artery, effectively excluding the old endograft. **(B)** An iliac plug was deployed in the left limb of the graft (curved arrow). **(C)** A femoro-femoro bypass was performed.

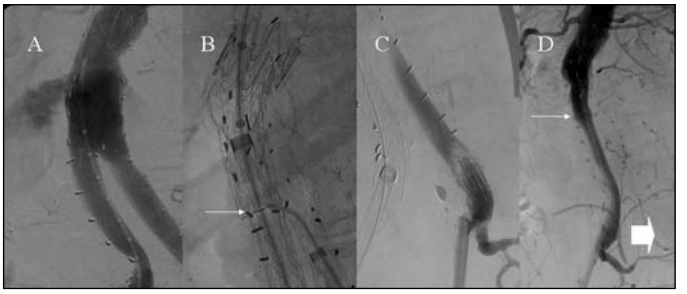


Figure 34-3. Endoconversion using a bifurcated graft. **(A)** A type IV endoleak within an Ancure device is demonstrated. **(B)** An Excluder bifurcated graft is deployed within the Ancure device. The contralateral gate of the Excluder device is deployed inside the right limb of the Ancure device (arrow). **(C)** An iliac plug was deployed in the left limb of the Ancure graft to prevent retrograde flow. **(D)** Completion angiogram showing correction of the endoleak and flow limited to the right iliac system with preservation of flow into the internal iliac. The iliac plug is seen in the left limb of the Ancure graft (block arrow). A femoro-femoral bypass (not shown) was done to revascularize the left leg and the left hypogastric artery.

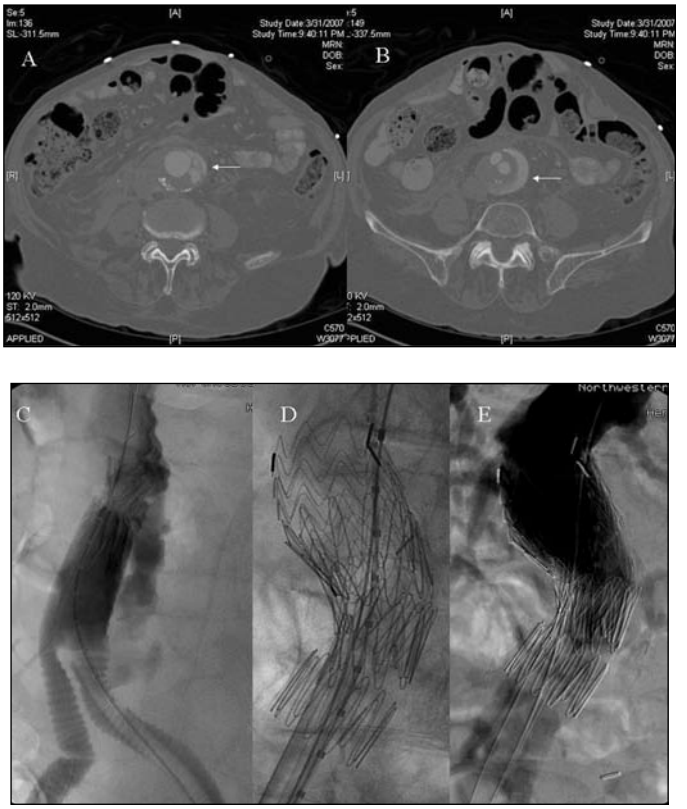


Figure 34-4. (A) CT scan at the level of the proximal anastomosis of a Dacron graft surgically placed nine years prior. Contrast is seen outside the graft and into the sac (arrow). The sac was previously treated with endoaneurysmorrhaphy. **(B)** A lower cut showing contrast and thrombus in the aneurysm sac outside the limbs of the graft. **(C)** Angiogram demonstrating the equivalent of a Type 1 endoleak at the proximal anastomotic site. A proximal aortic cuff (Zenith) is being deployed. **(D)** A combination of different aortic cuffs (Zenith,Excluder) was needed to correct the radiographic abnormality. **(E)** Completion angiogram showing patency of both renal arteries and elimination of the endoleak.

endografts deployed in the native aorta or iliac arteries did not have complications; however, attempts to deploy in a preexisting graft resulted in inadequate seal and fixation of the device, and increasing the risk of sac expansion and rupture.[10] In addition, dilation of the Dacron graft is common and can make determining the location of the proximal anastomosis on imaging inaccurate. This makes it difficult to determine whether a sufficient amount of infrarenal aorta is available to land the graft.[21] In these cases, it may be helpful to selectively catheterize the renal arteries in preparation for renal artery stenting in the event of partial renal artery occlusion caused by deployment of the endograft (see endograft relining).

It is also important to rule out latent infection in patients with prior open repair before proceeding with placement of an endograft. Infection is a potential cause of anastomotic disruption, and in most of these cases, endovascular repair is an inadequate treatment modality.

The formation of "hygromas" allegedly due to porosity and transgraft flow through PTFE grafts is an interesting occurrence after open AAA. This has been reported in the literature[22,23] and we have recently treated two such cases. Although endoconversion is an attractive solution to the culprit of the complication (Figure 34–5), decompression of the hygroma is usually needed.

Some authors advocate endovascular therapy for the treatment of aortoenteric fistula. We believe this condition is best treated with open techniques, but endovascular modalities may have a role in temporizing an unstable or septic patient in preparation for definitive open repair. Although the proximal anastomosis is the most common site of communication, the fistula may be present anywhere along the length of the device.[24] For this reason, El Sakka et al. recommended placement of a bifurcated graft if possible to exclude the entire graft from renal to iliac arteries.[21]

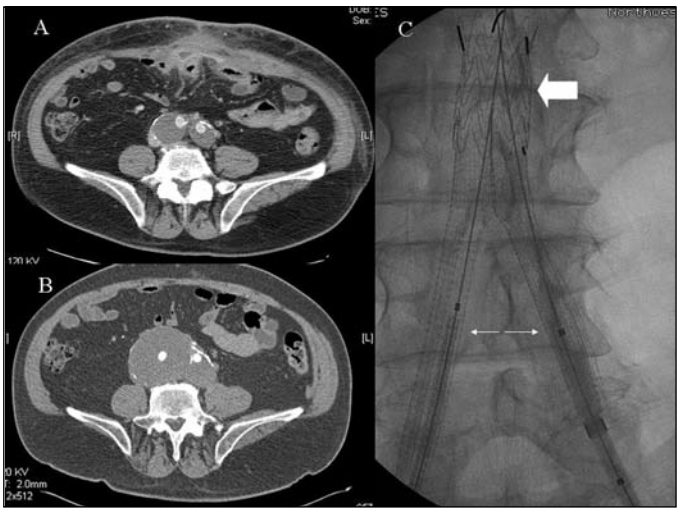


Figure 34-5. (A) CT scan four years after open AAA repair using a PTFE bifurcated graft. **(B)** Repeat CT (same level) eight years after open AAA repair when the patient presented with bilateral lower extremity edema. The previously patent IVC is compressed by a markedly enlarged aneurysmal sac. Delayed images demonstrated no IV contrast in the sac. **(C)** Endoconversion by relining. An aortic cuff (block arrow) was deployed below the renal arteries and stentgrafts (Viabohn) (arrows) were deployed inside the aortic cuff proximally and into the native iliac arteries distally.

CONCLUSION

Although the results of EVAR continue to improve, durability remains an issue. The increased need for secondary interventions is, in part, offset by the increasing ability to utilize percutaneous techniques for reoperation. Endoconversion offers an effective treatment of postoperative complications following both EVAR and open repair with decreased morbidity and mortality. This is especially important after open repair as many of the indications for endovascular intervention after open repair occur late in the postoperative course and are, therefore, found in older patients with more comorbidities. Further improvements in the designs of various types of endovascular devices will further increase the potential applications of endovascular intervention to a broader patient population.

REFERENCES

1. Schermerhorn ML, O'Malley AJ, Jhaveri A, Cotterill P, Pomposelli F, Landon BE. Endovascular vs. open repair of abdominal aortic aneurysms in the Medicare population. *N Engl J Med.* 2008;358:464–474.
2. Endovascular aneurysm repair versus open repair in patients with abdominal aortic aneurysm (EVAR trial 1): randomised controlled trial. *Lancet*. 2005;365:2179–2186.
3. Zarins CK, DeWeese J, Abel DB et al. Lifeline registry of endovascular aneurysm repair: Long-term primary outcome measures. *J Vasc Surg*. 2005;42:1–10.
4. Kalman PG, Rappaport DC, Merchant N, Clarke K, Johnston KW. The value of late computed tomographic scanning in identification of vascular abnormalities after abdominal aortic aneurysm repair. *J Vasc Surg*. 1999;29:442–450.
5. Hallett JW, Jr., Marshall DM, Petterson TM et al. Graft-related complications after abdominal aortic aneurysm repair: reassurance from a 36-year population-based experience. *J Vasc Surg.* 1997;25:277–284.
6. Edwards JM, Teefey SA, Zierler RE, Kohler TR. Intraabdominal Paraanastomotic Aneurysms After Aortic Bypass-Grafting. *J Vasc Surg*. 1992;15:344–353.
7. Mulder EJ, van Bockel JH, Maas J, van den Akker PJ, Hermans J. Morbidity and mortality of reconstructive surgery of noninfected false aneurysms detected long after aortic prosthetic reconstruction. *Arch Surg*. 1998;133:45–49.
8. Kraus TW, Paetz B, Hupp T, Allenberg JR. Revision of the Proximal Aortic Anastomosis After Aortic Bifurcation Surgery. *Eur J Vasc Surg.* 1994;8:735–740.
9. Magnan PE, Albertini JN, Bartoli JM et al. Endovascular treatment of anastomotic false aneurysms of the abdominal aorta. *Ann Vasc Surg*. 2003;17:365–374.
10. van Herwaarden JA, Waasdorp EJ, Bendermacher BLW, van den Berg JC, Teijink JAW, Moll FL. Endovascular repair of paraanastomotic aneurysms after previous open aortic prosthetic reconstruction. *Ann Vasc Surg*. 2004;18:280–286.
11. Allen RC, Schneider J, Longenecker L, Smith RB, III, Lumsden AB. Paraanastomotic aneurysms of the abdominal aorta. *J Vasc Surg.* 1993;18:424–431.
12. Treiman GS, Weaver FA, Cossman DV et al. Anastomotic False Aneurysms of the Abdominal-Aorta and the Iliac Arteries. *J Vasc Surg*. 1988;8:268–273.
13. Curl GR, Faggioli GL, Stella A et al. Aneurysmal Change at Or Above the Proximal Anastomosis After Infrarenal Aortic Grafting. *J Vasc Surg.* 1992;16:855–860.
14. Matsumura JS, Pearce WH, Cabellon A, McCarthy WJ, Yao JST. Reoperative aortic surgery. *Cardiovasc Surg.* 1999;7:614–621.
15. Lagana D, Mangini M, Fontana F, Nicotera P, Carrafiello G, Fugazzola C. Percutaneous Treatment of Sac Rupture in Abdominal Aortic Aneurysms Previously Excluded with Endovascular Repair (EVAR). *Cardiovasc Intervent Radiol*. 2008.

16. Mehta M, Taggert J, Darling RC et al. Establishing a protocol for endovascular treatment of ruptured abdominal aortic aneurysms: Outcomes of a prospective analysis. *J Vasc Surg*. 2006;44:1–8.
17. Golzarian J, Valenti D. Endoleakage after endovascular treatment of abdominal aortic aneurysms: Diagnosis, significance and treatment. *Eur Radiol*. 2006;16:2849–2857.
18. Becquemin, J. P., Zubilewicz, T., Desgranges, P., Lapeyre, M., and Kobeiter, H. Experience With Secondary Operations After Failed EVAR: The Good, The Bad And The Ugly. 30th Global: Vascular and Endovascular Issues, Techniques and Horizons, 4.1–4.4. 2007.
19. Minion DJ, Yancey A, Patterson DE, Saha S, Endean ED. The endowedge and kilt techniques to achieve additional juxtarenal seal during deployment of the Gore Excluder endoprosthesis. Ann Vasc Surg. 2006;20:472–477.
20. Hinchliffe RJ, Alric P, Wenham PW, Hopkinson BR. Durability of femorofemoral bypass grafting after aortouniiliac endovascular aneurysm repair. *J Vasc Surg*. 2003;38:498–503.
21. El SK, Halawa M, Kotze C, Francis I, Doyle T, Yusuf W. Complications of open abdominal aortic surgery: the endovascular solution. *Interact Cardiovasc Thorac Surg*. 2008;7:121–124.
22. Risberg B, Delle M, Lonn L, Syk I. Management of aneurysm sac hygroma. *J Endovasc Ther*. 2004;11:191–195.
23. Salameh MK, Hoballah JJ. Successful endovascular treatment of aneurysm sac hygroma after open abdominal aortic aneurysm replacement: a report of two cases. *J Vasc Surg*. 2008; 48:457–460.
24. Cendan JC, Thomas JB, Seeger JM. Twenty-one cases of aortoenteric fistula: Lessons for the general surgeon. *Am Surg*. 2004;70:583–587.
25. Tiesenhausen K, Hausegger KA, Tauss J, Amann W, Koch G. Endovascular treatment of proximal anastomotic aneurysms after aortic prosthetic reconstruction. *Cardiovasc Intervent Radiol*. 2001;24:49–52.
26. Pearce BJ, Baldwin Z, Bassiouny H, Gewertz BL, McKinsey JF. Endovascular solutions to complications of open aortic repair. *Vasc Endovasc Surg*. 2005;39:221–228.
27. Zhou W, Bush RL, Bhama JK, Lin PH, Safaya R, Lumsden AB. Repair of anastomotic abdominal aortic pseudoaneurysm utilizing sequential AneuRx aortic cuffs in an overlapping configuration. *Ann Vasc Surg*. 2006;20:17–22.
28. Morrissey NJ, Yano OJ, Soundararajan K et al. Endovascular repair of para-anastomotic aneurysms of the aorta and iliac arteries: preferred treatment for a complex problem. *J Vasc Surg*. 2001;34:503–512.
29. Piffaretti G, Tozzi M, Lomazzi C, Rivolta N, Caronno R, Castelli P. Endovascular treatment for para-anastomotic abdominal aortic and iliac aneurysms following aortic surgery. *J Cardiovasc Surg* (Torino). 2007;48:711–717.

35

Endovascular Therapy of Posttraumatic Pseudoaneurysms

Mitchell W. Cox, M.D. and Shaun M. Gifford, M.D.

Pseudoaneurysms most often present with a call from the interventional suite about a patient with an expanding groin hematoma after femoral artery access for an elective procedure. Although these iatrogenic femoral pseudoaneurysms are common and somewhat tedious to deal with, other manifestations of pseudoaneurysms after arterial injury are often far more interesting and challenging for the vascular surgeon. Posttraumatic pseudoaneurysms may present with a variety of different symptoms and appear in virtually any anatomic location or organ system. But while the onset can be unpredictable and the diagnosis obscure, there is a very straightforward decision tree for therapy, and despite technical complexity, interventions are almost always successful. In many instances, the vascular surgery team can swoop in heroically and deliver a permanent cure for a disturbing problem with a minimally invasive procedure.

ETIOLOGY

Pseudoaneurysms can complicate any blunt or penetrating injury that results in damage to the arterial wall. Presumably, pseudoaneurysm formation is more likely to occur in cases with partial transection of a vessel where in-line flow is maintained in spite of extravasation. Initial bleeding may be controlled by the surrounding tissues resulting in a simple hematoma, or the worrisome "pulsatile hematoma", which is felt to be at high risk for frank hemorrhage. By definition, if there is persistent flow in a cavity surrounding the arterial defect, the problem is referred to as a false aneurysm or pseudoaneurysm. It is differentiated from a true aneurysm by absence of the three normal layers of the arterial wall: intima, media, and adventitia. Acutely, most traumatic pseudoaneurysms will have flow lumen within a variable amount of thrombus, but chronically, a more organized fibrous wall may develop.[1]

PRESENTATION

In the setting of civilian trauma, pseudoaneurysms are not aggressively sought after unless clinical suspicion is raised. In addition, routine arteriography for penetrating trauma has fallen out of favor, which often results in the late presentation of an initially occult pseudoaneurysm. The manifestations may not be clearly associated with the original trauma and can be bizarre, representing a veritable gold mine for writers of case reports in multiple specialties. There may be occult bleeding from the upper airway managed by ENT, upper GI bleed presenting to gastroenterology, nerve compression consulted to neurology (Figure 35–1), hematuria evaluated by urology, or a large pulsatile mass with associated bruit referred urgently to vascular surgery (Figure 35–2). Ultimately, it requires vigilance by the consulting service and a high index of suspicion when unusual symptoms are not easily explained.

Pseudoaneurysms of the abdominal viscera, including the liver, spleen, kidneys, or mesenteric vessels, may become symptomatic due to mass effect, intraperitoneal hemorrhage, or erosion into adjacent structures. Hepatic pseudoaneurysms may rupture into the biliary tree producing upper GI bleeding, and renal pseudoaneurysms can produce microscopic or gross hematuria by erosion into the renal collecting system.[2-4] Mesenteric and splenic pseudoaneurysms can be asymptomatic, but may

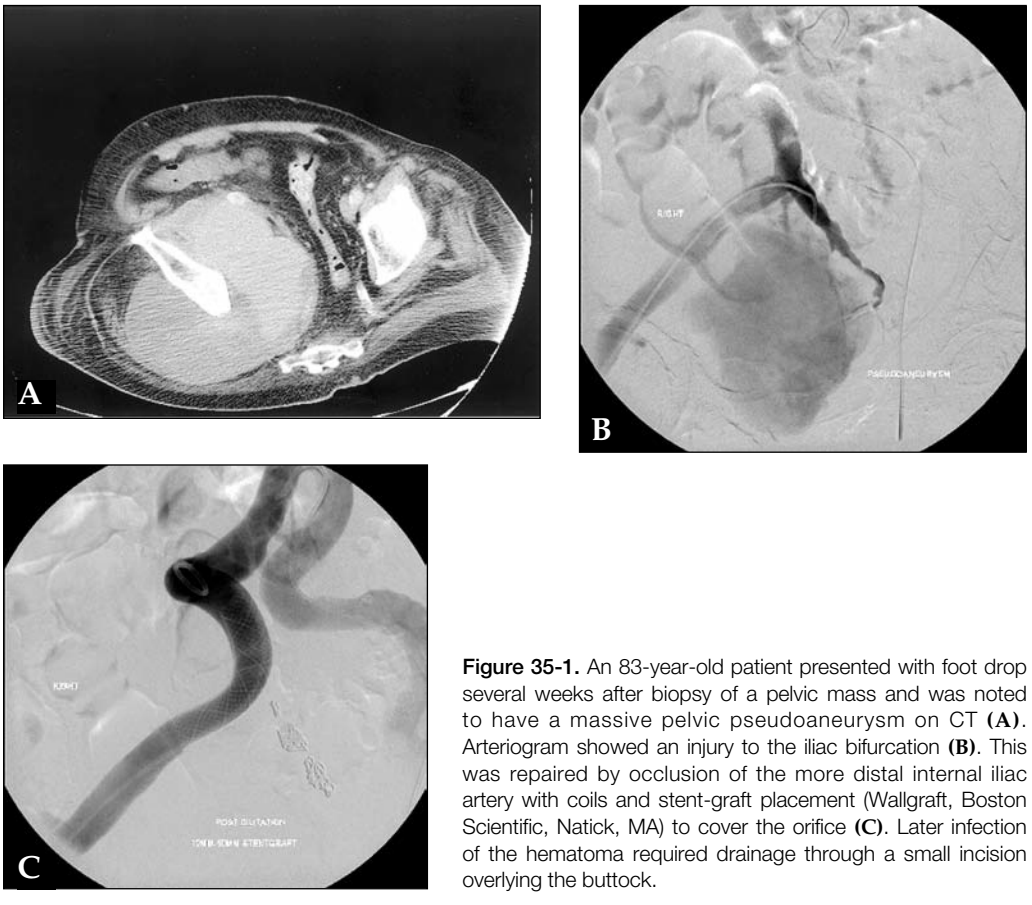


Figure 35-1. An 83-year-old patient presented with foot drop several weeks after biopsy of a pelvic mass and was noted to have a massive pelvic pseudoaneurysm on CT **(A)**. Arteriogram showed an injury to the iliac bifurcation **(B)**. This was repaired by occlusion of the more distal internal iliac artery with coils and stent-graft placement (Wallgraft, Boston Scientific, Natick, MA) to cover the orifice **(C)**. Later infection of the hematoma required drainage through a small incision overlying the buttock.

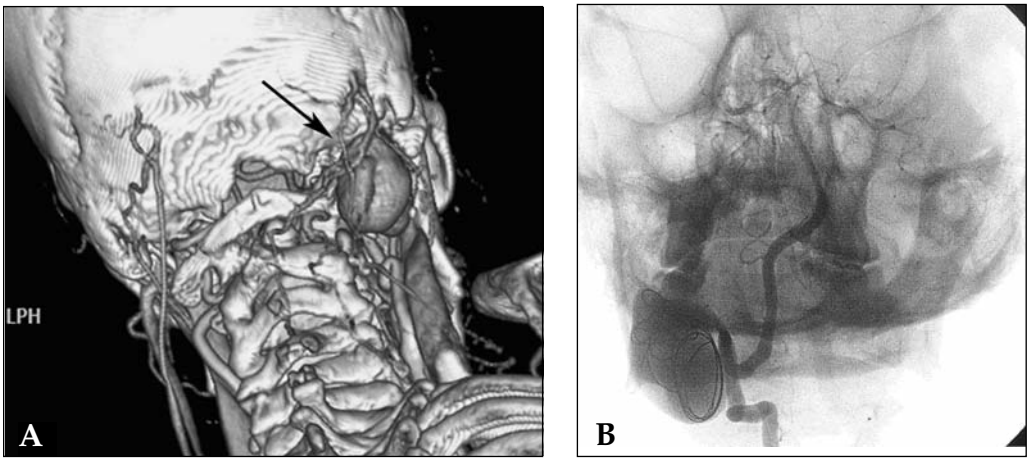


Figure 35-2. A 50-year-old man presented to the emergency department with complaints of an enlarging pulsatile mass behind his right ear with an associated bruit. CT **(A)** revealed a large pseudoaneurysm of the right vertebral artery, which was confirmed by angiography **(B)**. Microcatheter delivery of 0.018" coils into the aneurysm sac led to thrombosis of the pseudoaneurysm, with absence of flow confirmed by duplex at follow-up. In this case, stent-grafting was felt to be a poor option given the location of the pseudoaneurysm in the distal vertebral artery. Coiling of the vertebral artery proximal and distal to the injury would be the fallback procedure if the sac had failed to thrombose.

also present with constant, vague abdominal pain, which is felt to herald catastrophic intraperitoneal hemorrhage (Figure 35–3).[5-7]

Symptoms associated with pseudoaneurysms of the head and neck can be nerve compression, airway compromise, or unexplained upper airway hemorrhage[8] (Figures 35–4 and 35–5). Any neck hematoma that results from blast or penetrating injury, even if presenting days or weeks posttrauma, merits careful vascular evaluation and imaging studies since pseudoaneurysms are the most common missed injury in this situation.[9] Figure 35–6 illustrates such a case in which a patient presented 48 hours postinjury with a large neck hematoma and airway compromise due to mass effect.

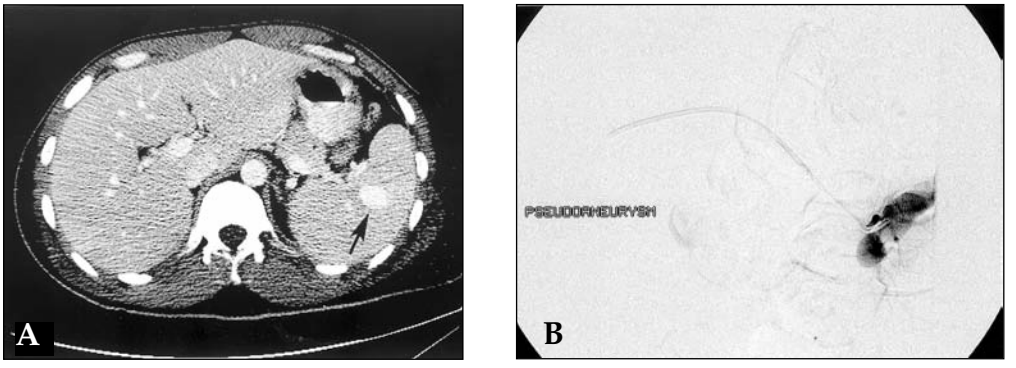


Figure 35-3. A 31-year-old patient presented 10 days after blast injury with a splenic pseudoaneurysm that was seen on routine follow-up CT **(A)**. An angled glide catheter (Terumo, Ann Arbor, MI) was advanced to the splenic hilum and into the pseudoaneurysm **(B)**. The feeding artery was embolized using several coils (Tornado, Cook Medical, Bloomington, IN).

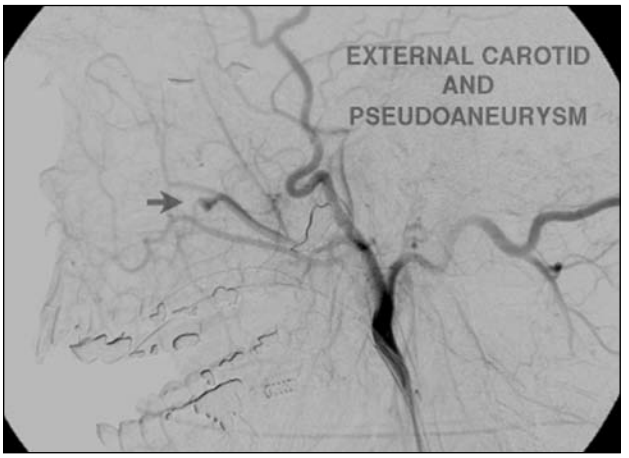


Figure 35-4. A soldier sustaining a gunshot wound to the face was found to have a pseudoaneurysm of a maxillary artery branch and had an episode of massive epistaxis with respiratory arrest. Bleeding was temporized with nasal packing and coil embolization was promptly accomplished. In this instance, a 90 cm angled glide catheter (Terumo) was advanced into the external carotid artery, and a 3 Fr microcatheter (Progreat, Terumo) was used to deliver coils to the vessel.

Pseudoaneurysms related to the superficial temporal artery or smaller branches of the facial artery would present less dramatically, with a small pulsatile mass in the subcutaneous tissues of the face.

Extremity pseudoaneurysms often present with reports of a palpable mass, bruit, or leg edema. Although more rare, they may also manifest as nerve compression with resultant paresthesias or leg weakness distal to the injury.[10,11] These are typically the most easily diagnosed pseudoaneurysms, and patients will frequently note an expanding mass or feel the pulsation. Late hemorrhage from a previously repaired arm or leg should prompt concern for a pseudoaneurysm and merits a detailed vascular evaluation.

Historically, large series of pseudoaneurysms have been reported after military conflicts, with the majority manifesting as large, palpable extremity or neck masses. In a review by Rich of 296 pseudoaneurysms resulting from combat in Vietnam, the most common presentation by far was a palpable mass, bruit, or frank hemorrhage in a

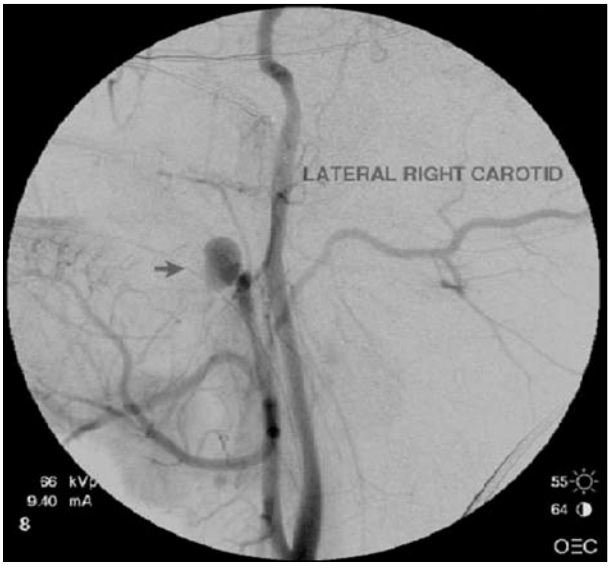


Figure 35-5. Several hours after a gunshot wound to the mouth, this patient presented with massive pulsatile bleeding. He was taken to the operating room where angiogram demonstrated an external carotid pseudoaneurysm. Coiling was immediately accomplished from a femoral approach using only a 90 cm angled glide catheter (Terumo).

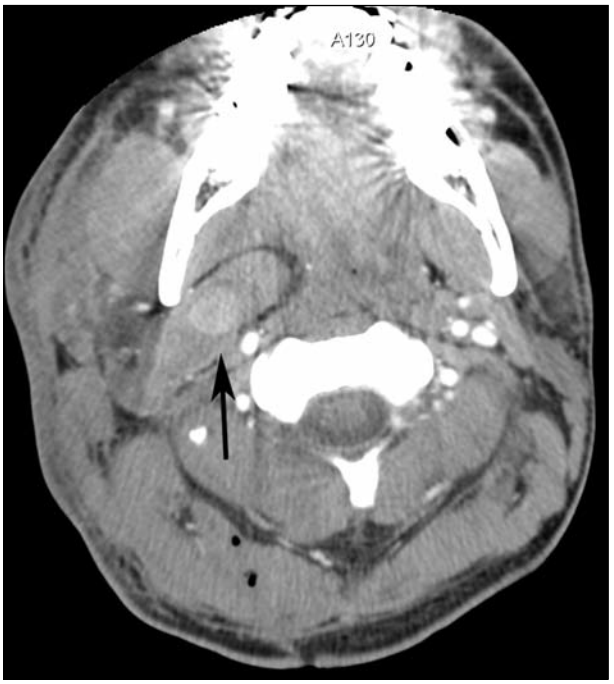


Figure 35-6. This pseudoaneurysm of an external carotid branch presented 48 hours after a blast injury with airway compromise, and coil embolization was performed.

wounded extremity. Surprisingly, no visceral or mesenteric pseudoaneurysms and only 14 pseudoaneurysms of the head and neck were reported in this comprehensive study.[10] The experiences of Shumaker from WWII were similar and encompassed a wide variety of large, symptomatic pseudoaneurysms, typically noted in the upper and lower extremities.[1] Development of CT scanning in the intervening years has undoubtedly increased the diagnostic rate of abdominal and thoracic pseudoaneurysms.

In current military practice, the massive tissue disruption and multiple fragment injuries created by military munitions usually prompt a very aggressive initial vascular work-up which virtually always includes a CT with IV contrast. This often results in an early diagnosis of asymptomatic pseudoaneurysms in any location. While the occasional pseudoaneurysm encountered by vascular surgery services is large and symptomatic, the most common presentation is as an incidental finding on CT. We have documented an almost 10% incidence of these occult pseudoaneurysms in patients evaluated for severe blast injury to the head and neck.[9]

EVALUATION

Most vascular surgeons agree that CT angiography (CTA) is the best study for initial evaluation of patients who are at high risk for, or suspected of having, a pseudo-aneurysm regardless of anatomic location. Even in the presence of minor artifact from retained metallic fragments, it is a very sensitive and specific study. Although arteriography is the traditionally used modality to evaluate pseudoaneurysms, a small pseudoaneurysm might be clearly seen at CTA, but not initially well seen at angiography (Figure 35–7). Magnetic resonance angiography may also be useful in evaluation, but utility is

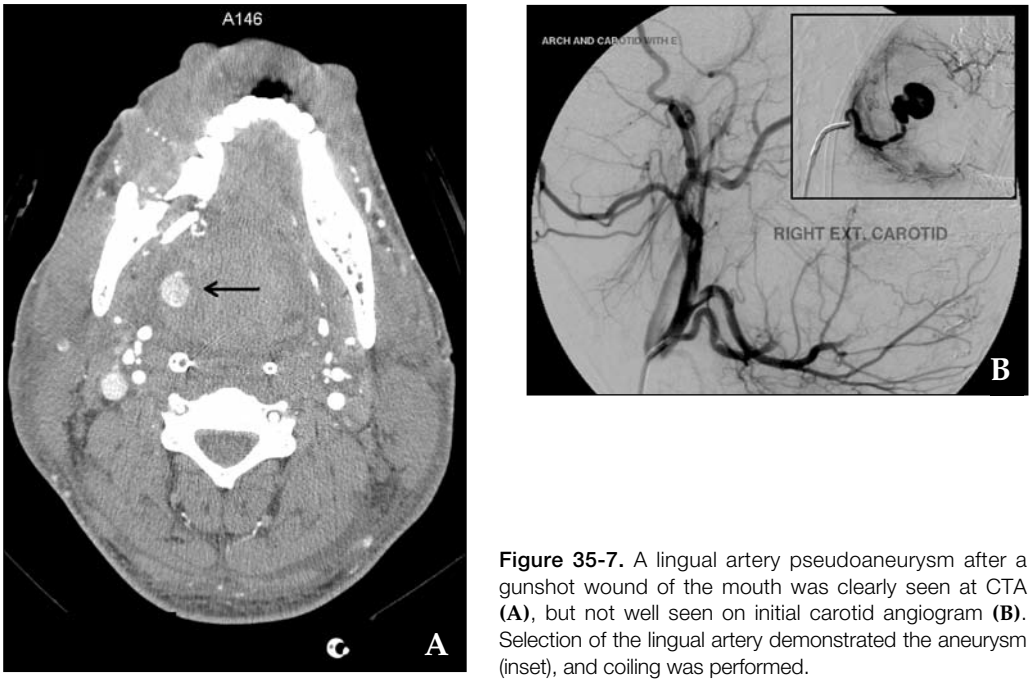


Figure 35-7. A lingual artery pseudoaneurysm after a gunshot wound of the mouth was clearly seen at CTA **(A)**, but not well seen on initial carotid angiogram **(B)**. Selection of the lingual artery demonstrated the aneurysm (inset), and coiling was performed.

often limited by the presence of metallic fragments or the inability of the patient to tolerate the procedure.

In cases of delayed bleeding in a patient who has recently sustained penetrating trauma, a ruptured pseudoaneurysm must be suspected, and it is reasonable to proceed directly to angiography. Other hard signs of vascular injury may also prompt immediate angiography, and any noninvasive evaluation should be skipped. Ideally, the study should be performed as an "on-table" angiogram in the operating room so that bleeding can be surgically controlled if necessary. Figure 35–5 demonstrates such a case in which a patient presented several hours posttrauma with massive hemorrhage from the mouth. This study was performed intraoperatively, with prompt coiling of the external carotid to achieve hemostasis. If endovascular arrest of bleeding was unsuccessful, surgeons could quickly open and obtain vascular control. The operating room location, as opposed to the interventional suite, allows for this life-saving option.

Duplex ultrasound may also be useful for evaluation in isolated situations, particularly in cases of a superficial pseudoaneurysm beneath the skin of the face or a palpable pseudoaneurysm in the extremities. Duplex is especially applicable in follow-up since it can reliably detect residual flow in an aneurysm cavity, and carries a lower cost with no exposure to radiation or IV contrast.

NATURAL HISTORY

It is difficult to assess the natural history of pseudoaneurysms unrelated to femoral artery catheterization, since the literature is dominated by case reports and small series of patients presenting with dramatic symptoms requiring intervention. In the past,

pseudoaneurysms were felt to be an ominous finding that required immediate surgery to avoid inevitable enlargement and rupture. An initial paper in the 1950s by Shumacker evaluating pseudoaneurysms related to combat in WWII suggested that only 3% of these false aneurysms resolved spontaneously and virtually always required surgical intervention.[1] Similarly, Rich and Hobson, after reviewing the Vietnam experience, concluded that most pseudoaneurysms would require operative repair.[10]

These older series, however, represent experience prior to the widespread availability of conventional angiography and CTA. All of the patients in these series presented with large pseudoaneurysms that were symptomatic, and in many cases palpable, on physical exam. With more modern diagnostic techniques, it is possible to diagnose small, asymptomatic pseudoaneurysms that might represent more of a therapeutic dilemma. No one would dispute that large, persistent, or symptomatic pseudoaneurysms require repair. However, there may be disagreement over whether small, incidentally discovered pseudoaneurysms need to be repaired due to their potential to spontaneously thrombose without intervention.

Some appreciation for the natural course of small asymptomatic pseudoaneurysms might be gleaned from reported series after femoral artery puncture. Notably, the largest of these series by Toursarkissian demonstrated that up to 90% of asymptomatic femoral pseudoaneurysms will spontaneously thrombose.[12] Whether this reflects the prognosis for lesions related to more significant trauma is unclear because the aforementioned series focused solely on iatrogenic puncture injuries. It is likely that the natural history of femoral pseudoaneurysms after angiography does reflect the expected outcome after punctate injury to a large artery, but may not be analogous to other types of traumatic injury. Gunshot wounds, blunt trauma, or blast injury can produce more extensive arterial wall damage or complete disruption of a smaller vessel, and the long-term course may not be so benign.

Given the potential for disastrous complications such as the sudden onset of massive upper respiratory hemorrhage, or disturbing events like the rapid appearance of a massive pulsatile extremity mass, observation is usually not the answer when considering treatment options. Many vascular surgeons have developed a strong bias for immediate repair in all but the most unstable patients who are found to have a pseudoaneurysm of virtually any size. Even in cases where the flow lumen of a pseudoaneurysm is small, there may be major underlying damage to the arterial wall, setting the patient up for later blowout and rapid expansion of the pseudoaneurysm. Potentially serious complications of these missed arterial injuries have been noted in both the civilian and military literature, supporting this more aggressive approach.[9,13]

THERAPY

Fortunately, despite a panoply of possible locations and presentations, the decision tree for treatment of most pseudoaneurysms is straightforward and need not involve a lot of handwringing. Our general approach is simple and is illustrated in Figure 35–8. The open surgical approach will occasionally be chosen to address certain pseudo- aneurysms; however, the vast majority can and should be addressed with endovascular techniques. A traditional surgical approach may be reasonable to repair a large artery in an easily accessible location, but the open approach is frequently difficult due to severely traumatized tissue planes and associated hematoma. An endoluminal approach is trivial

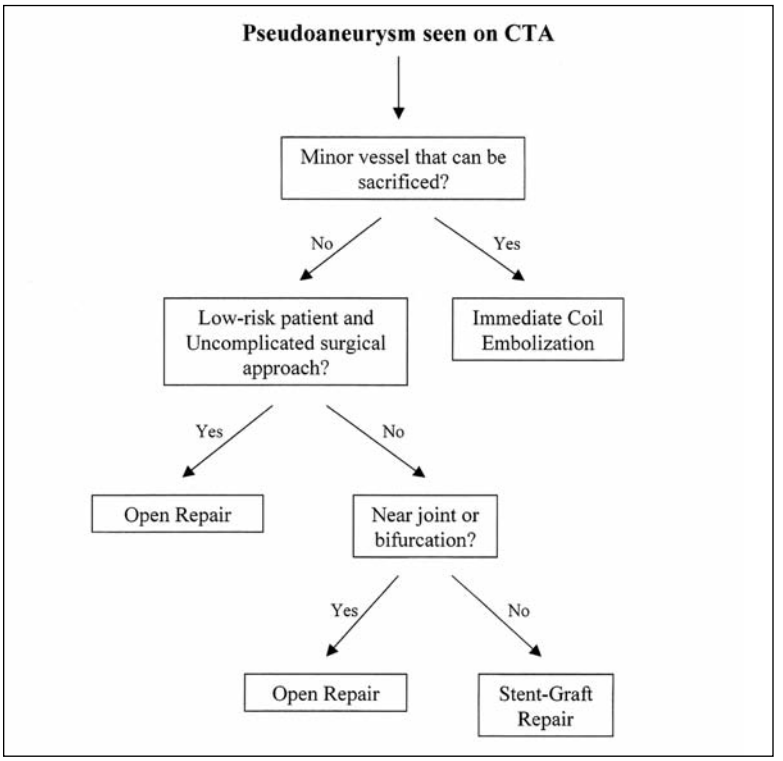


Figure 35-8. Decision Tree for Posttraumatic Pseudoaneurysms. Our approach to posttraumatic pseudoaneurysms is illustrated. In practice, the majority of pseudoaneurysms have been treated with endovascular approaches.

by comparison, and is ideal for occluding smaller arteries that would be difficult or impossible to locate in a traumatized surgical field. In cases of visceral pseudo–aneurysms or those of intrathoracic arteries, coiling or stent-grafting is clearly the preferred modality in virtually all cases. Pseudoaneurysms of the extremity vessels may be more easily approached surgically; however, stent-grafting may still be preferable in some instances. In certain situations, it may be unwise to interfere with the previous surgical site with an open surgical exposure (Figure 35–9). In this case, we did not feel it prudent to disrupt a free-flap for an open surgical repair and instead deployed a stent-graft to exclude the aneurysm.

TECHNICAL CONSIDERATIONS

Noninvasive imaging, usually CTA, will demonstrate which artery is feeding the lesion and serve as a guide to plan therapy. As illustrated in Figure 35–8, false aneurysms fed by smaller arteries may be coiled, while those arising from larger vessels are excluded with stent-grafts to preserve flow. The best arterial access location can be planned in advance as well, with antegrade sheath placement or direct puncture of the aneurysm sac as necessary. Pathology of the tibial vessels or forearm is most directly accessed via an antegrade puncture of the common femoral or brachial artery. In cases of visceral artery

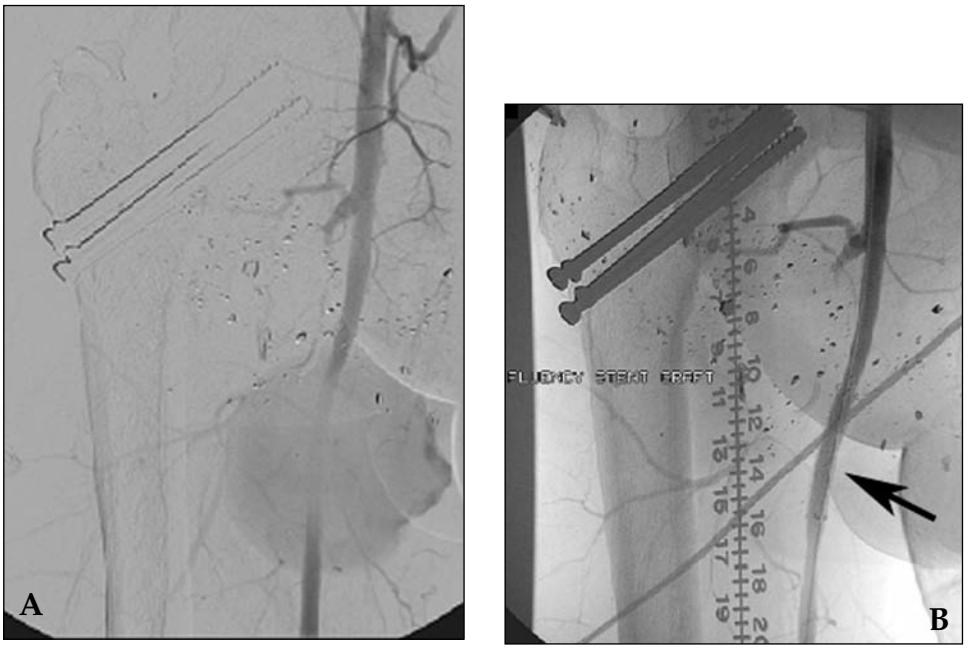


Figure 35-9. A soldier presented several weeks after a gunshot wound to the thigh with a pulsatile mass beneath a rectus free-flap used to cover a large tissue defect. The large pseudoaneurysm **(A)** was effectively excluded with a Stent-graft **(B)** (Fluency, Bard Peripheral Vascular, Tempe, AZ), which was delivered from a contralateral femoral artery puncture through a 9 French sheath. Later hematoma infection required open drainage.

aneurysms, we prefer a femoral approach. That being said, brachial access may be required in certain instances, particularly when deploying a stent-graft. Likewise, problems in the supra-aortic vessels or thoracic outlet may be addressed via the femoral or brachial artery, with the brachial puncture being simpler and more direct, but with a higher rate of access site complications. Pathology of the iliac vessels, or arteries of the thigh, are best accessed from the contralateral femoral artery. For pseudoaneurysms of the head and neck, a standard femoral approach is almost always easiest.

When embolizing a pseudoaneurysm with coils, the usual strategy is to occlude both the inflow and outflow portions of the feeding artery, the so-called sandwich technique.[11] Our preference is for advancing an angled glide catheter (Terumo, Ann Arbor, MI) as far as possible toward the lesion, then adding a coaxial 3 Fr microcatheter system for cannulating smaller branches. Occlusion of the outflow vessel is particularly important in highly collateralized vascular beds, as perfusion of the pseudoaneurysm may continue through collaterals if only the inflow artery is addressed. Figure 35–10 depicts a pseudoaneurysm arising from the interossious artery in the forearm, which presented as a pulsatile mass several weeks following a blast injury. Coiling was accomplished via an antegrade puncture of the brachial artery; however, inadequate coiling of the outflow vessel resulted in continued retrograde filling of the pseudoaneurysm. If it is not possible to negotiate a catheter into the outflow portion of the artery, then coiling the inflow will suffice in most cases (Figure 35–11). Coils may also be deployed within the aneurysm cavity, but this is less critical and less effective than addressing the artery itself, and flow may persist within a coiled sac if flow continues in the damaged artery.

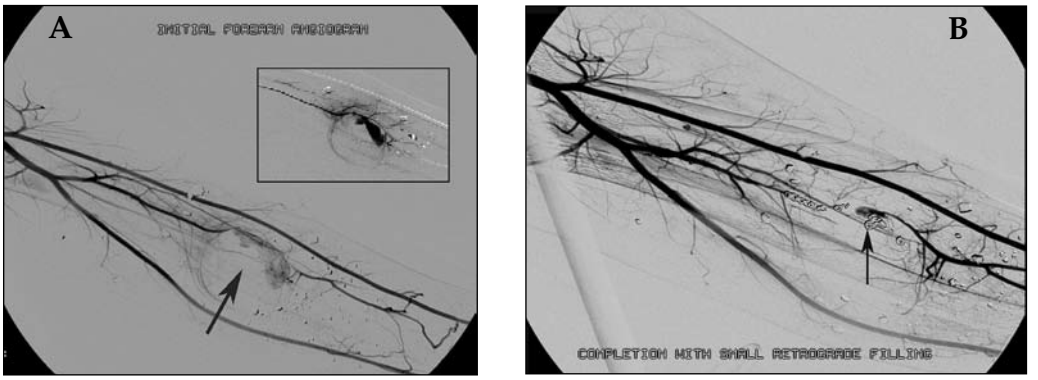


Figure 35-10. After a blast injury to the forearm, a pulsatile mass developed which was seen to be an interossious artery pseudoaneurysm at angiogram **(A)**. Antegrade puncture of the brachial artery was performed with a micropuncture kit (Cook Medical, Bloomington, IN), the interossious artery was selected with a 3 Fr microcatheter (inset), and coils were delivered. Unfortunately, the outflow was not effectively occluded and persistent flow continued via collaterals **(B)**.

If a stent-graft is being considered for treatment, the ability to deliver a device to the location of interest is sometimes the major issue, and the shortest, most direct access route is often chosen. This was especially true in the past given the large diameters and inflexible delivery systems of most stent-grafts. Newer stent-grafts, especially the balloon-expandable system (iCast, Atrium medical, Minneapolis, MN), are smaller and more flexible, and can be negotiated into virtually any artery greater than 4mm in diameter (Figure 35–12). When selecting a device, it is advisable to use the shortest graft possible that will effectively exclude the damaged portion of the artery. Obtaining multiple views to precisely identify the location of the aneurysm neck is critical prior to deploying a stent-graft in a large vessel. This is of increased importance when dealing with pseudoaneurysms that are in close proximity to distal vessel branches (Figure 35–13). Prior to the widespread availability of commercial stent-grafts, direct coiling of the pseudoaneurysm with or without an uncovered stent

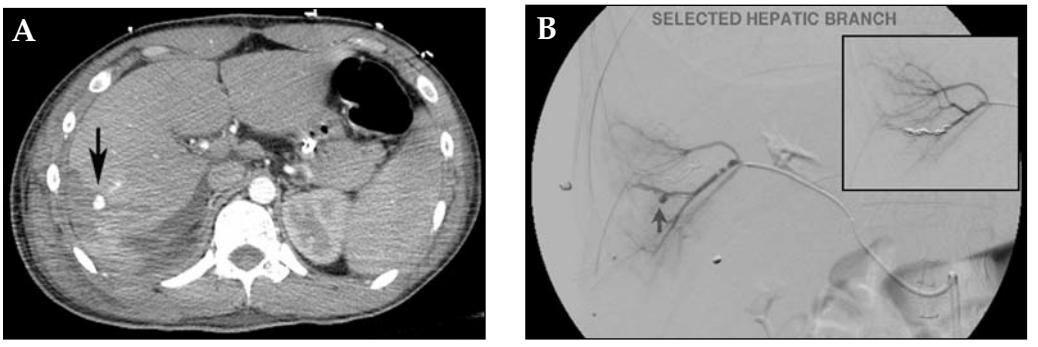


Figure 35-11. A small hepatic artery pseudoaneurysm after blast wound to the abdomen was noted at CT **(A)**. In the operating room, a guiding catheter (RDC, Cordis, Miami Lakes, FL) was positioned at the SMA orifice and an angled glidewire/angled glidecatheter combination was advanced into a replaced right hepatic artery. A 3 Fr coaxial microcatheter system (Progreat, Terumo) with a .014" hydrophilic wire (PT2, Boston Scientific, Natick, MA) was then negotiated through the subsegmental hepatic arterial braches and out to the lesion **(B)** where coils were placed (inset).

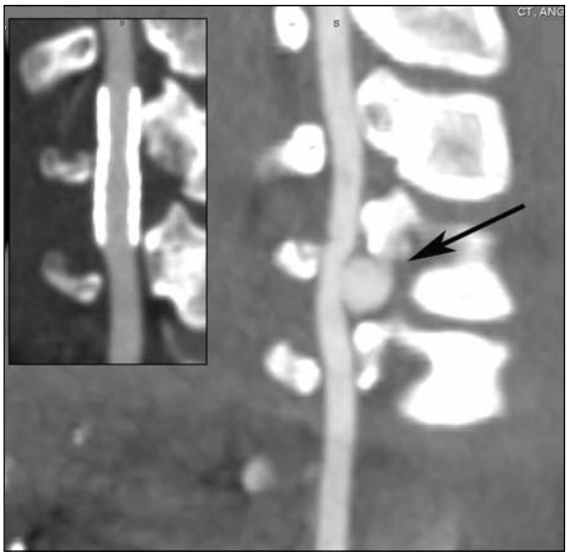


Figure 35-12. A pseudoaneurysm of the vertebral artery was noted on CTA after a gunshot wound to the neck, and a stent-graft was placed (inset). In this case, a 6 Fr, 90 cm sheath (Shuttle Select, Cook) was delivered via femoral puncture and positioned in the subclavian artery near the vertebral orifice. The balloon-expandable stent-graft (Atrium Medical) was easily delivered and deployed over a .018" wire (SV5, Cordis, Miami Lakes, FL).

in the main vessel was felt to be optimal. This may still be an option if a stent-graft cannot be tracked into appropriate position due to vessel size or location (Figure 35–2).[14] This traditional technique seems to have fallen out of favor given the availability of improved stent-grafting systems that are more user-friendly.[15]

Superficial pseudoaneurysms, some of which may be palpable beneath the skin, can also be treated via direct puncture, and either thrombin injection or coiling.[16] This technique is particularly applicable in cases where flow persists in the sac after coiling

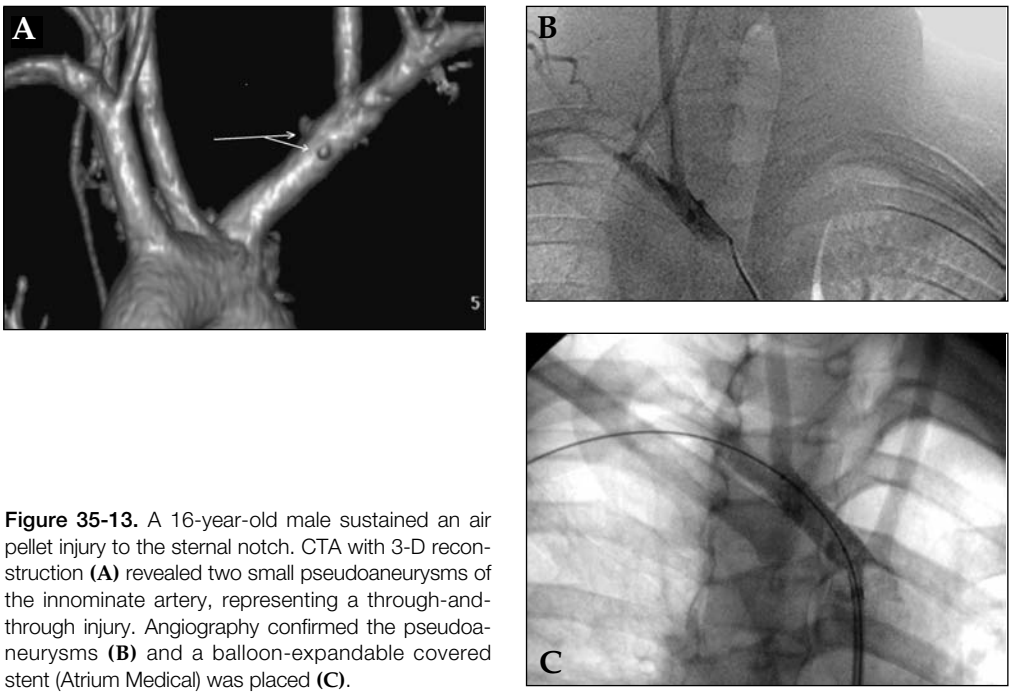


Figure 35-13. A 16-year-old male sustained an air pellet injury to the sternal notch. CTA with 3-D reconstruction **(A)** revealed two small pseudoaneurysms of the innominate artery, representing a through-and-through injury. Angiography confirmed the pseudoaneurysms **(B)** and a balloon-expandable covered stent (Atrium Medical) was placed **(C)**.

or stent-graft placement due to collateral flow. It is also a good approach if endoluminal access is unsuccessful. We have used direct puncture with coil or gelfoam embolization of the sac, but have been reluctant to use thrombin due to the risk of distal embolization of the thrombin into the microvasculature. Many posttraumatic pseudoaneurysms have a wide, short neck, which may predispose to distal embolization when using thrombin. This is not the case for most femoral pseudoaneurysms secondary to needle puncture where thrombin injections are uniformly successful with minimal risk for distal embolization. Direct puncture is most applicable for superficial aneurysms in the subcutaneous tissue of the face, and these have been treated successfully with gelfoam embolization.

COMPLICATIONS

Serious morbidity after endovascular treatment of pseudoaneurysms is rare with a high success rate and low occurrence of immediate complications. Technical difficulties with coil embolization may include misplaced coils, failure to induce sac thrombosis, or the ultimate ignominy, creation of a new traumatic pseudoaneurysm at the femoral access site. A percutaneous closure device can be utilized in most cases to prevent the latter occurrence. Coiling a small vessel is very unlikely to have any long-term consequences and we do not do any follow-up of coiled aneurysms beyond a repeat CT scan several days postprocedure to document thrombosis.

When treating larger pseudoaneurysms associated with massive hematoma, there is a risk of infection, and our group has experienced two cases of abscess within the hematoma of the thrombosed aneurysm sac. One disadvantage to an endovascular approach is that it leaves a large, undrained, and potentially infected cavity. Two such patients are depicted in Figures 35–1 and 35–8. Management in each case consisted of incision and drainage through relatively small incisions with placement of a closed suction drain. Interestingly, in both cases, a stent-graft had been placed, but neither prosthetic became infected.

One point of controversy and continued discussion is whether stent-grafts will be durable long term, particularly in cases where stents are implanted into young patients who may be subject to device-related complications for 40 years or more. There is minimal information dealing with long-term outcomes of stent-grafting due mostly to their rather recent development and use. Stent-graft complications are not frequently reported in the literature; however, we have had one instance of postoperative thrombosis of a stent-graft in the distal internal carotid artery.[9] A case of short-term restenosis of an axillary artery stent-graft has also been described, although the consequences of both occurrences were minimal.[17] We will be interested to see if reports of late thromboses of stent-grafts appear in the literature over the next five to 10 years. Perhaps development of a bioabsorbable stent-graft system will eliminate this concern in the future.

SUMMARY

Posttraumatic pseudoaneurysms are unusual complications of blunt or penetrating trauma that may present in any anatomic location with a variety of unusual symptoms. While an open approach is occasionally employed, virtually all of these lesions can be treated with

endovascular techniques, using either coils or a stent-graft to exclude the aneurysm. Results are uniformly excellent in the short term, and our vascular service is enthusiastic about tackling these procedures. In some respects, endovascular treatment of pseudoaneurysms represents the holy grail of vascular surgery cases: presentation is interesting, the majority of cases are nonemergent, the patients are young and healthy, and the case is technically challenging but nearly always successful.

REFERENCES

1. Shumacker HB, Wayson EE. Spontaneous cure of aneurysms and arteriovenous fistulas, with some notes on intrasacular thrombosis. *Am J Surg*. 1950;79(4):532–544.
2. Malaisrie SC, Borge MA, Glynn L et al. Combined percutaneous and angiographic thrombosis of a traumatic hepatic artery pseudoaneurysm in a child. *J Trauma*. 2005;59(4):1025–1028.
3. Liu T, Hou M, Lin H et al. Life-threatening hemobilia caused by hepatic artery pseudoaneurysm: a rare complication of chronic cholangitis. *World J Gastroenterol*. 2003;9(12): 2883–2884.
4. Lee RS, Porter JR. Traumatic renal artery pseudoaneurysm: diagnosis and management techniques. *J Trauma*. 2003;55(5):972–978.
5. Ray B, Kuhan G, Johnson B et al. Superior mesenteric artery pseudoaneurysm associated with celiac axis occlusion treated using endovascular techniques. *Cardiovasc Intervent Radiol.* 2006;29(5):886–889.
6. Miller MT, Comerota AJ, Disalle R et al. Endoluminal embolization and revascularization for complicated mesenteric pseudoaneurysms: a report of two cases and a literature review. *J Vasc Surg*. 2007;45(2):381–386.
7. Yardeni D, Polley TZ Jr, Coran AG. Splenic artery embolization for post-traumatic splenic artery pseudoaneurysm in children. *J Trauma*. 2004;57(2):404–407.
8. Diaz-Daza O, Arraiza FJ, Barkley JM et al. Endovascular therapy of traumatic vascular lesions of the head and neck. *Cardiovasc Intervent Radiol*. 2003;26(3):213–221.
9. Cox MW, Whittaker DR, Martinez C et al. Traumatic pseudoaneurysms of the head and neck: early endovascular intervention. *J Vasc Surg*. 2007;46(6):1227–1233.
10. Rich NM, Hobson RW, Collins GJ. Traumatic arteriovenous fistulas and false aneurysms: a review of 558 lesions. *Surgery*. 1975 Dec;78(6):817–828.
11. Lopera JE, Suri R, Cura M, et al. Crural Artery Traumatic Injuries: Treatment with Embolization. *Cardiovasc Intervent Radiol*. 2008 Feb 26; [Epub ahead of print].
12. Toursarkissian B, Allen BT, Petrinec D et al. Spontaneous closure of selected iatrogenic pseudoaneurysms and arteriovenous fistulae. *J Vasc Surg*. 1997;25(5):803–808.
13. Perry MO. Complications of missed arterial injuries. *J Vasc Surg*. 1993;17(2):399–407.
14. Berne JD, Reuland KR, Villarreal DH et al. Internal carotid artery stenting for blunt carotid artery injuries with an associated pseudoaneurysm. *J Vasc Surg*. 2008;64(2):398–405.
15. Maras D, Lioupis C, Magoufis G et al. Covered stent-graft treatment of traumatic internal carotid artery pseudoaneurysms: a review. *Cardiovasc Intervent Radiol*. 2006;29(6):958–968.
16. Komorowski-Timek E, Teruya TH, Abou-Zamzam AM et al. Treatment of radial and ulnar artery pseudoaneurysms using percutaneous thrombin injection. *J Hand Surg* [*Am*]. 2004;29(5):936–942.
17. Oktar GL, Balkan ME, Akpek S et al. Endovascular stent-graft placement for the management of a traumatic axillary artery pseudoaneurysm-a case report. *Vasc Endovascular Surg*. 2002;36(4):323–326.

36

Bailouts for Endovascular Procedures

W. Anthony Lee, M.D.

"Fortune favors the prepared." In no other procedure is this saying more applicable than in endovascular aneurysm repair (EVAR). However, even the most thoughtfully planned out procedure in the hands of even the most experienced endovascular therapist can be fraught with a myriad of unexpected events during a seemingly "routine" EVAR. While appropriate patient selection and careful planning results in a gratifying conclusion in most instances, every practitioner must be familiar with the bailout options and salvage techniques for the most common and life-threatening complications that can occur during these procedures. The purpose of this chapter is to highlight some of these intraoperative events and their potential therapeutic options. It should be noted that a number of off-label use of existing medical devices will be discussed and their implementation is discretionary on the availability of devices and technical expertise.

ILIAC RUPTURE

Difficult access due to small or diseased iliofemoral arteries is the single most common source of complications during EVAR (Figure 36–1). Indeed, vascular injury was the most frequent major adverse event in almost all of the endovascular aortic device clinical trials.[1] The solution for unfavorable access is a retroperitoneal iliac conduit (Figure 36–2). The rule of thumb that cannot be overstated is that the mere thought of a conduit should be an indication for doing one. While retroperitoneal procedures during EVAR are not without their own risks beyond the customary femoral access,[2] they are far outweighed by the clinical consequences of not performing an iliac conduit.

In the event, however, that a conduit was not performed preemptively when indicated but the endograft was successfully delivered and implanted by forceful insertion, the two most common consequences are either a focal dissection or life-threatening iliac disruption (Figure 36–3), -both of which become manifest at the

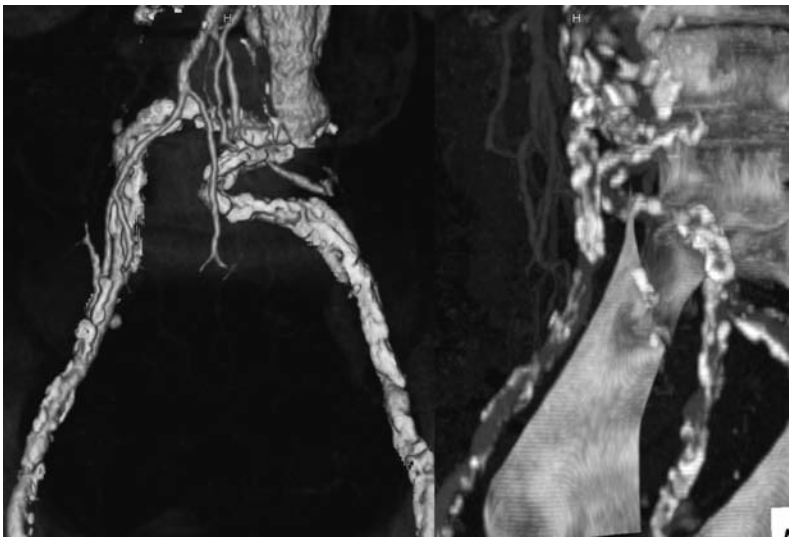


Figure 36-1. Bilateral small and diseased iliac arteries.

conclusion of the case when the operator and his staff are least prepared and most mentally distracted.

Focal Dissection

Most operators do not routinely perform retrograde iliac angiograms at the conclusion of the EVAR. Large delivery sheaths frequently occlude the external iliac artery and it is not typically visualized during a completion angiogram. Furthermore, attention is

Figure 36-2. Retroperitoneal Dacron conduit anastomosed end-to-side to the distal common iliac artery.

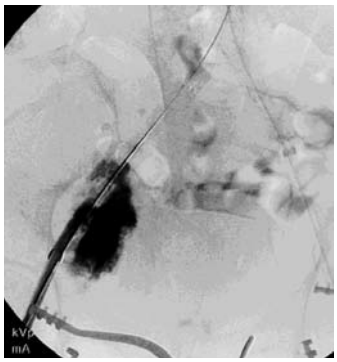


Figure 36-3. External iliac artery rupture from a large sheath during EVAR.

mostly drawn to the proper seating of the endograft, patency of the key branch vessels, and detection of endoleaks. Focal external iliac and proximal femoral dissection is the most common source of weak or absent femoral pulses.

If the femoral arteries were surgically exposed, the intima at the insertion site may be directly inspected after the removal of the sheaths. When an intimal flap is visualized, this may be directly endarterectomized with or without extension of the arteriotomy, the distal flap tacked down, and the artery repaired either primarily or with a patch. The pulse proximal to the clamp may be palpated to determine qualitatively whether there is an inflow problem upstream. While a weak pulse may be helpful in determining a proximal anatomic defect, a normal pulse is not as informative, as the pulse proximal to a clamped artery can be quite strong and the only way to assess adequacy of the inflow is after all the clamps are released.

After completion of the femoral artery repair, if direct palpation of the pulse is weak or there is a thrill, it may be due to the low outflow impedence of a relatively ischemic limb, which may take waiting a few minutes; or, more likely, there is another defect more proximally. If there is any concern regarding completeness of the endarterectomy, the femoral artery should be exposed more proximally and the arteriotomy reinspected. Occasionally, a more proximal intimal tear can be present above the level of the exposed femoral artery, forming an occlusive flap when antegrade flow is restored but which deceptively collapses when the flow is ceased. Gentle probing will reveal a nonadherent section of intima that may not have been recognized during the initial repair, but which can simply be excised.

If there is no local problem, the culprit is more proximal in the external iliac artery. The femoral artery should be repunctured and a short 5 French sheath inserted. A retrograde angiogram through the sheath will typically identify the problem. Using a nonhydrophilic guidewire, the lesion is crossed and a self-expanding stent is placed over the intimal defect. For dissections that are near the hypogastric artery, the stent may be placed directly over its origin, extending from the endograft iliac limb to the native external iliac artery.

On occasion, the problem is in the endograft iliac limb due to kinking or compression, most commonly at a narrow aortic bifurcation that may not have been appreciated during the completion angiogram. With certain endografts in tortuous iliac arteries, iliac limbs that were not kinked during the completion angiogram due to the presence of stiff guidewires that may kink after removal of the guidewires and cause hemodynamic compromise (Figure 36–4). In cases of external compression, the bailout option is simply to perform kissing balloon angioplasties in both iliac limbs with or

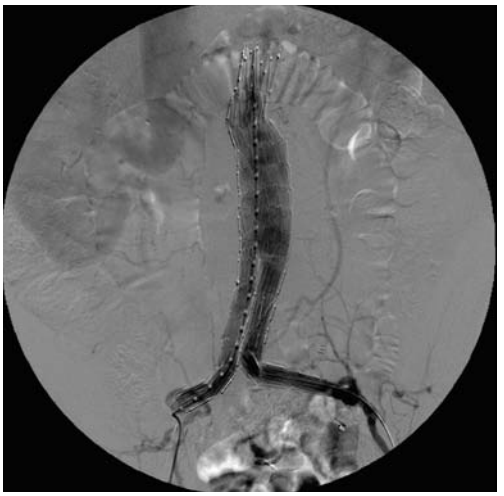


Figure 36-4. Note the kinking of the left iliac limb. This was treated with placement of a self-expanding nitinol stent with complete correction of the defect.

without adjunctive stenting using balloon-expandable stents. Ballooning and/or stenting in only the affected limb versus both limbs may cause the contralateral limb to be compressed. In cases of limb kinking, deployment of a self-expanding nitinol stent with postdilation provides sufficient added radial strength to reexpand the kinked section of the iliac limb while maintaining the natural contour of the iliac artery.

Iliac Disruption

Iliac disruption almost always occurs on the side where the main device is inserted. It is one of the most avoidable life-threatening complications during EVAR. Occasionally, a potential problem is suspected when significant force was required during insertion of the main device, and there is a sudden "give." This usually corresponds to the avulsion of the iliac artery. Typically, this is not associated with any appreciable hemodynamic changes until the sheath or the delivery catheter is retracted to deploy the endograft. If this occurs and is promptly recognized or confirmed with a retrograde injection of contrast, the ipsilateral iliac limb is rapidly deployed, and an occlusive balloon is inflated in the limb while an appropriate iliac extension is prepared. The balloon is removed and the iliac extension is deployed into the external iliac artery through the existing sheath. No attempt is made to deal with the hypogastric artery during this time. Following stabilization, if there is evidence of persistent bleeding, the hypogastric artery will need to be surgically ligated for retroperitoneal hemostasis.

Alternatively, even if the iliac artery remains intact during the insertion, disruption may occur during extraction of the device or sheath at the conclusion of the case. In a manner similar to that felt during the insertion, the operator may feel a sudden release with production of what is now referred to as an "iliac-on-a-stick;" that is, a torn segment of the external iliac artery tightly wrapped around the delivery catheter or sheath, followed by rapid hypotension.

When an iliac disruption occurs, the external iliac artery is almost always avulsed just distal to the hypogastric artery origin. Knowing this, the operator has two options depending on whether or not stiff guidewire access has been maintained. If guidewire access has been maintained, a 12 French or greater size introducer sheath may be quickly reinserted and an aortic occlusion balloon advanced to occlude the iliac limb.

Next, an appropriately sized iliac stent graft limb or a peripheral covered stent can be rapidly deployed after removal of the balloon to bridge and repair the defect. Obviously, during the exchange of the balloon for the endograft, significant hemorrhage will occur, but the deployment of the stent graft should continue expeditiously while the patient is resuscitated. If the contralateral sheath is still in place, the occlusion balloon may be inserted through this side and inflated above the device's flow divider. In this situation, the entire repair may take place without loss of proximal occlusion.

If an appropriate iliac endograft is not available or guidewire access has been lost, expeditious retroperitoneal exposure is obtained through a lower quadrant incision. There is no need for a full laparotomy to gain proximal aortic control as this will take longer and potentially cause more surgical damage in a rushed attempt for aortic exposure. Even in the face of active hemorrhage and poor visualization, manual exploration will readily allow identification of the open stump of the common iliac artery by the presence of the endograft limb and proximal digital control. The endograft limb, whether constructed out of stainless steel or nitinol stents, can be safely occluded with a soft-jawed clamp and the iliac disruption repaired with a conventional iliofemoral bypass.

Aortic Perforation

Aortic perforation or disruption is exceedingly rare. There have been scattered case reports of rupture of the proximal neck of the aneurysm during ballooning of the proximal endograft. A semicompliant angioplasty balloon (versus a compliant occlusion balloon) sized to the diameter of the endograft (versus the diameter of the native aorta) inflated to nominal pressure resulted in this catastrophic complication. This was reflective of the relative inexperience of the early operators.

In current practice, most endograft molding done at the conclusion of the procedure uses soft, compliant aortic occlusion balloons that typically generate 1–2 atmospheres of pressure inflated by hand. The only instance today where angioplasty balloons are used is when adjunctive balloon-expandable stenting is performed to seal a proximal endoleak. In these cases, caution must be exercised to properly size the balloon so as to avoid overdilation of the aorta.

Aortic perforation has been rarely reported, again during the early collective experience of aortic endografting, usually with inappropriate guidewire management. In these cases, although quite difficult due to the floppy design of most guidewire tips, forceful advancement against a weakened area of the aortic wall can lead to perforation. A more common scenario, however, is when the guidewire inadvertently has migrated out of the aorta and, unrecognized, the delivery catheter or introducer sheath is advanced without a leading guidewire. The stiff, tapered tip of the catheter now becomes a dangerous javelin that can easily perforate the aorta.

If the perforation is in the aneurysm sac itself, the optimal treatment is to expeditiously complete the endovascular repair. Even with only the main bifurcated device deployed, an occlusion balloon can be inflated in the main body while the contralateral gate is accessed and the limb deployed. If the disruption occurs more proximally in the juxtarenal aorta, there is little choice except to rapidly inflate an occlusion balloon proximal to the disruption and surgically convert the patient. On rare occasions, if the patient is not a surgical candidate and there is significant asymmetric origins of the two renal arteries, the main device may be deployed over the lowermost renal artery while preserving the patency of the higher of the two renal arteries. Although

this method sacrifices one kidney, it may be a better option than open surgical repair in an otherwise surgically unfit patient without necessarily condemning the patient to hemodialysis.

Inability to Cannulate the Contralateral Gate

During deployment of a modular endograft system, for many operators, cannulation of the contralateral gate remains one of technical bottlenecks and difficult aspects of the procedure. The reasons for difficult cannulation are frequently a combination of intrinsic anatomy and poor case planning. Anatomic factors include angulated proximal neck, an acute aortoiliac angle, common iliac occlusive disease, and a large aneurysm sac without much thrombus. Poor case planning can include an overly long main bifurcated device that either traps the gate in the ipsilateral iliac artery or places the gate too close to the aortic bifurcation, leaving no working room to catheterize its open end. Better prediction of the "lie" of the endograft can allow orientation of the gate in an anterior or contralateral position relative to the ipsilateral limb to facilitate the cannulation.

Loss of guidewire access into the aorta from the contralateral iliac artery in the setting of a significant proximal iliac stenosis or narrowed aortic bifurcation may prevent reentry of the guidewire into the aorta after the ipsilateral limb is deployed and the already compromised aortic bifurcation becomes more crowded and/or the proximal plaque has been disrupted. Last, there are rare cases of long proximal necks with a narrowed, calcified distal segment combined with sharp angulation of the aneurysm that can compress the gate shut so that even the guidewire or the iliac limb cannot be advanced beyond this point. In all of these instances, after all the conventional techniques for contralateral gate access have been exhausted, one is faced with a choice of abortion, surgical conversion, or aortouni-iliac (AUI) conversion of a bifurcated device.

This last option is preferred. The Zenith (Cook, Inc., Bloomington, Indiana) endograft system has an AUI conversion system comprised of a funneled-tubular converter and a contralateral iliac occluder (Figure 36–5). This conversion system may be used for other endograft systems but additional devices may be necessary to achieve a hemostatic seal. When this converter system is not available, other options are available. Depending on the endograft system (e.g., AneuRx, Medtronic Vascular, Santa Rosa, California), two proximal aortic extender cuffs may be used to block off the flow divider and direct flow into the ipsilateral limb.[3] Alternatively, an entire bifurcated device (e.g., Excluder, W.L. Gore, Flagstaff, Arizona) may be deployed through the ipsilateral limb with the top of the two bifurcated devices flush within each other. In the case of the Excluder, due to the differential radial force of the nitinol stents sur-

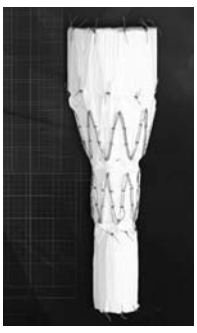


Figure 36-5. Cook Zenith converter. Converts a bifurcated main body into an aortouni-iliac device. The device is 80 mm in length and the proximal diameter is available in 24, 28, and 32 mm. The distal diameter is 12 mm. The accompanying contralateral iliac occluder (not shown) is 20 mm long and is available in 14, 16, 20, and 24 mm diameters.

rounding the ipsilateral and the contralateral gate, when the two limbs are externally compressed, the contralateral gate will be preferentially compressed closed, leaving the ipsilateral limb open. In both scenarios, the bifurcated devices are converted into AUI configurations.

Additionally, the contralateral iliac artery must be occluded. Several options exist for this. One can make an occluder by deploying an appropriately sized iliac extender ex vivo, closing one end of the extender with a surgical purse string, reloading it into a delivery sheath, and deploying it in the common iliac artery using a modified obturator as a pusher. A second option is to coil embolize the common iliac artery. A recently introduced device that can be used for this purpose is the Amplatzer Plug, with or without additional coils. The last option is to surgically ligate the iliac artery through a small retroperitoneal exposure. The final step is to perform a femoral-femoral bypass graft to restore flow to the contralateral leg.

The operator should keep in mind that by aborting an uncompleted endovascular repair rather than attempting a previously untried technique or surgically converting a high-risk patient, one still has not violated the principle of *prima non nocere*. The patient is no worse off than before the aneurysm repair was attempted, except for having sustained the risk of the anesthetic. By aborting the procedure, one can return to "fight the battle" another day by recruiting additional expertise, getting the necessary devices, or referral to a tertiary care center. Given the current collective experience with EVAR, rarely is an unplanned surgical conversion necessary or indicated in the absence of an intraoperative catastrophe.

High Deployment of the Main Body

Even in the most experienced hands, the main bifurcated device can be inadvertently deployed higher than intended with coverage of the renal artery. There are two options in this situation, depending on the degree of renal artery coverage. If the renal artery is only partially covered, the best option is to place a stent across the top edge of the endograft into the renal artery using standard interventional techniques. Low profile systems such as 0.014"-based pre-mounted balloon-expandable systems, are preferable, as the residual opening afforded by the top edge of the fabric can be quite small and the stent can be caught on the lip of the fabric. Occasionally, a brachial approach is necessary to avoid the need to go over the edge required from a femoral approach. The stent should be positioned at least 2–3 mm over the edge of the occluding endograft into the aortic lumen so that there is no risk of the fabric riding back up and covering the renal stent (Figure 36–6).

If the renal orifice cannot be stented open, the endograft must be pulled down or risk loss of a kidney. Although a number of ways has been described, the best method is the transbifemoral technique with a guidewire. In this technique, an ipsilateral guidewire is snared from the contralateral side and brought out through the femoral sheaths. The flow divider is protected with a catheter to prevent an inadvertent tear of the endograft fabric with the guidewire. After firmly grasping both ends of the guidewire with clamps, the endograft is pulled down with a series of short, rapid, forceful jerks. Even devices without active fixation mechanisms require a surprisingly large amount of force for this maneuver. As the discovery of inadvertent proximal deployment is typically made during the completion angiogram after both iliac limbs have been seated, any column strength of the endograft limbs further opposes the downward pull of the guidewire. Although it is initially disconcerting to see the entire

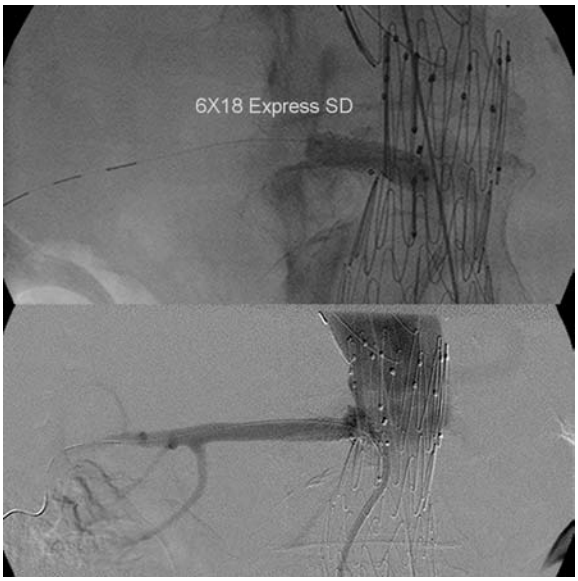


Figure 36-6. Right renal artery stenting after partial coverage.

aorta move up and down during this maneuver, the risk of significant aortic injury is minimal.

Techniques using balloons are not effective due to the elasticity of the plastic catheter shaft. The shaft stretches with the pull and the amount of force that is exerted cannot be controlled. Furthermore, even a significantly oversized angioplasty balloon (a compliant aortic occlusion balloon is completely ineffective) either placed over the flow divider or within the limb will slip down due the lack of sufficient frictional resistance of the balloon against the fabric of the endograft.

Low Deployment of the Main Body

Inadvertent low deployment of the main body can result in three types of complications, depending on how far distally the endograft is deployed.

Inadequate or Loss of Proximal Fixation. If the device is deployed lower than intended in a relatively short (<20-mm) proximal neck, regardless of whether there is a type I endoleak, if there is sufficient room to deploy a proximal extender, it is recommended that one be deployed for better long-term stability. If the fixation is marginal and there is not enough room to place a proximal extension, the main device may be pulled down a short distance using the technique described above to enable placement of a proximal cuff. Alternatively, if the main body has been deployed so low that the proximal end of the device lies within the aneurysm sac, one or more extenders may be required. If more than three stacked extenders are required, it is advisable to simply place a second bifurcated device above the prior main body, being careful to align the contralateral gates in line with each other. Multiple stacked cuffs result in an intrinsically unstable proximal construction due to the sheer number of junctions, and cuff separation with a type III endoleak is a real risk with any late aneurysm remodeling. A second device imparts a more stable proximal fixation and connection with the distal half of the endovascular construction (Figure 36-7). Last, if a bifurcated construction cannot be maintained, an AUI configuration (described above) is an option.

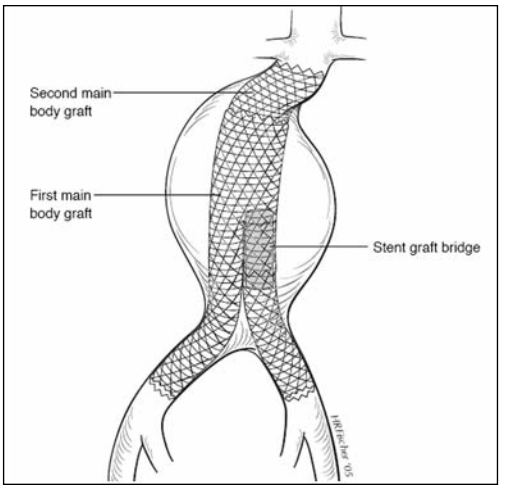


Figure 36-7. Insertion of an entire second bifurcated device within the first bifurcated device when it had been inadvertently deployed essentially in the aneurysm sac.

Trapped Contralateral Gate. A very distal deployment may place the contralateral gate so close to the aortic bifurcation so that access to it is impossible (unless it is favorably aligned with the origin of the contralateral iliac artery) or trap the gate entirely within the ipsilateral iliac artery. In either situation, there is no option except to convert the main body into an AUI configuration as described above.

Inadvertent Coverage of the Hypogastric Artery. Despite proper preoperative device selection, inadvertent distal deployment of the main body will effectively move the end of the ipsilateral iliac limb distal to the intended site in the common iliac artery and cover the hypogastric artery. Options for this complication are discussed below.

Inability to Advance the Contralateral Limb. This is a fairly rare situation that occurs when the contralateral iliac artery makes a very acute angle with the aortic bifurcation, and the gate lies in the opposite direction to its trajectory. Although most iliac tortuousities can be straightened with stiff guidewires, some aortoiliac angles are not. As the iliac limb is advanced, the delivery catheter bows against the opposite aortic wall and does not advance into the gate. Gentle torquing of the catheter or external abdominal wall counterpressure may provide the necessary support to get the proximal tip of the device into the gate. On the other hand, if the operator persists in pushing the contralateral iliac delivery catheter when it is obviously not advancing along the guidewire, the distal half of the main body may buckle and actually pull the ipsilateral limb out of its common iliac artery. One must stop before this occurs.

There are two options. The buckling of the delivery catheter can only be resolved by fixing the guidewire in two places. This means snaring the contralateral guidewire from the brachial artery (usually the left) and establishing a transbrachiofemoral "body-floss." Traction of both ends of the wire will almost always provide the necessary support to advance the device. The second option is to perform an AUI conversion as described previously.

Deployment of the Contralateral Limb outside the Gate

Although never reported in literature, inadvertent deployment of the contralateral limb outside the gate has not only been described anecdotally but even observed by large audiences during live case demonstrations. Of all the technical errors, this is

clearly one of the most avoidable. A hydrophilic guidewire can pass between the stent graft and the aortic wall without obvious tactile resistance or visible deflection of the tip. Several different methods have been well described to verify intrastent passage of the cannulating guidewire, including free rotation of a pigtail or similar type of recurved catheter within the main body, direct injection of contrast, and orthogonal views of the guidewire path within the gate. Regardless of the method used, true luminal passage of the guidewire must be absolutely verified before deploying the contralateral limb. Even if the extraluminal passage of the guidewire is not immediately recognized, one can back out right up to the point of actual deployment of the limb. Once the limb is deployed, there are few bailout options.

If the contralateral limb has been only partially deployed and the extra-luminal deployment recognized, depending on the flexibility and the length of the limb, one can forcibly push the undeployed distal half of the limb upward and deploy it kinked within the aneurysm sac. The iliac artery and the contralateral gate remain open for a second attempt at correct cannulation. If successful, although the final fluoroscopic image may appear unusual with a seemingly detached "floating" iliac endograft in the sac, the final result will still be a bifurcated repair. If, on the other hand, the contralateral limb has been completely deployed within the iliac artery, the only option is an AUI conversion as previously described. The occluder in this case is deployed within the misdeployed iliac limb.

Inadvertent Hypogastric Artery Coverage

Although accurate anatomic sizing and device selection have become significantly more sophisticated since the days of simple 5-mm thickness axial CT images on celluloid film and "Post-It" notepad and pencil method of measurement, determining the correct path length of an endograft remains an elusive computational task. Three-dimensional reconstruction and center-line measurements currently provide the closest approximation of this measure. The consequence of incorrect measurement and selection of a device that was too long is landing the iliac limb beyond the intended site of fixation in the common iliac artery and coverage of the hypogastric artery. This may also occur as previously mentioned when the main body is deployed very low.

When the hypogastric artery is covered, there are few remedial options. In most cases, the artery is occluded flush by the endograft and does not become a source of a type II endoleak. If the contralateral hypogastric artery is patent, nothing further is required. The procedure is concluded and the patient should be informed of the complication and the 40% risk of developing buttock and hip claudication.[4] However, if the iatrogenically occluded hypogastric artery was the only patent hypogastric artery, there is a risk of severe pelvic ischemia including colonic infarction. In this situation, serious consideration must be given to electively revascularize the hypogastric artery through a lower retroperitoneal incision.

Type I Endoleak

Even with appropriate patient selection and case planning, type I endoleaks can be seen at the conclusion of the case using digital subtraction angiography in 5% to 10% of all cases. Most qualitatively small leaks can be safely watched and will seal within six months. In those instances where the endoleak is large, adjunctive bailout techniques must be considered.

Proximal Attachment. A large type I endoleak is rarely resolved with repeat ballooning alone. If the primary device has been deployed lower than intended and there is sufficient room for placement of a proximal extender, this should be exactly performed to cover every millimeter of available neck. If this is not possible and/or even after deployment and coverage of the entire proximal neck fails to seal the endoleak, a bare metal balloon-expandable stent can be deployed to impart additional radial force to the proximal fixation. A P4010 or 3110 Palmaz stent hand-mounted on an appropriately sized 40-mm long angioplasty balloon such as the Maxi LD (Cordis Endovascular, Warren, New Jersey), is deployed, centered about the top edge of the stent graft. Two technical points that must be kept in mind are 1) the angioplasty balloon must not be sized to the diameter of the endograft but to the smallest diameter of the aortic neck; there is significant risk of proximal neck rupture if the balloon is incorrectly oversized, and 2) the large Palmaz stents can foreshorten by 30% to 40% when they are expanded to their final aortic diameter of 20–26 mm.

Distal Attachment. Repeat ballooning with a larger balloon can seal a distal type I endoleak more often than a proximal leak. Similar to the proximal attachment, if there remains an uncovered segment of the distal common iliac artery, extension right to the hypogastric origin is indicated. When the common iliac artery is ectatic and the endograft limb already extended to the hypogastric artery, two options are possible. In the first option, the hypogastric artery is sacrificed and the limb extended to the external iliac artery. The decision to separately embolize the hypogastric artery is dependent on the anatomy of the iliac bifurcation and the probability of effecting flush occlusion of the orifice with the stent graft. The second option is to perform a surgical "circlage" with an umbilical tape around the distal common iliac artery through a lower retroperitoneal exposure.

CONCLUSION

Unforeseen technical difficulties and complications can occur during endovascular aneurysm repair even in the most well-planned cases to the most experienced operators. Given enough time and number of cases, probability predicts that one will be faced with one or more of the situations described above. Preparation and having all the endovascular and surgical tools necessary are the best defenses against this eventuality. Fortunately, the collective skill set and the tools available today allow an endovascular solution to most problems resulting in, perhaps not perfect, but a reasonable and durable AAA repair without the necessity or risk of open surgical conversion.

REFERENCES

1. Zarins CK, White RA, Schwarten D et. al. AneuRx stent graft versus open surgical repair of abdominal aortic aneurysms: multicenter prospective clinical trial. *J Vasc Surg*. 1999;29: 292–305.
2. Lee WA, Berceli SA, Huber TS et. al. Morbidity with retroperitoneal procedures during endovascular abdominal aortic aneurysm repair. *J Vasc Surg*. 2003;38:459–463.
3. Wilson EP, Abul-Khoudoud O, Guarino LA et. al. Aorto-uni-iliac conversion of the bifurcated AneuRx device: a technical note. *J Vasc Surg*. 2000;12(6):333–334.
4. Lee WA, O'Dorisio J, Wolf YG et. al. Outcome after unilateral hypogastric artery occlusion during endovascular aneurysm repair. *J Vasc Surg*. 2001;33(5):921–926.

37

Salvage Procedures for Late Endovascular Failures

Jon S. Matsumura, M.D.

INTRODUCTION

Abdominal aortic aneurysm is a leading cause of death in adults, responsible for approximately 13,800 deaths in the United States in 2005.[1] Parodi first inserted an endograft for definitive treatment of abdominal aortic aneurysm, which he reported in 1991.[2] Also in 1991, Volodos from the Soviet Union described a self-fixing synthetic prosthesis for repair of both thoracic and abdominal aneurysms.[3]

Endovascular aneurysm repair has become an option to repair aneurysms with proven lower perioperative mortality. In the Endovascular Aortic Repair trial 1, a randomized trial of 1082 patients with large aneurysms, in-hospital mortality was 2.1% with endovascular repair versus 6.2% with open repair.[4] Four-year survival rate was similar. In the Dutch Randomized Endovascular Aneurysm Management Trial, 351 patients were randomized to endovascular versus open repair, and combined mortality and severe complications totaled 4.7% with endovascular repair compared with 9.8% with open repair.[5] There were equivalent 2-year survival rates, although oral presentations have suggested that a worse late hazard may be associated with endovascular repair. The Open Versus Endovascular Repair trial, which randomized 881 Veterans Affairs patients, and the French Aneurysme Chirurgie de l'aorte contre Endoprothese trial are due to report 2-year results soon. In a propensity-score risk-adjusted analysis of the Nationwide Inpatient Sample, endovascular repair was associated with lower perioperative mortality compared with open repair (1.2% vs. 4.8%).[6] Late survival is similar for the two techniques. Taken together, the literature demonstrates that endovascular repair is associated with lower perioperative mortality, but concern remains about late events and mitigation of these early benefits. The degree of mitigation is partially dependent on the ability to identify late failure modes and effectively retreat them with risks. This chapter focuses on reoperative procedures after late endovascular failures.

LATE COMPLICATIONS

Treatment of late endovascular complications requires identification and understanding of the mechanisms of failure of endovascular repair. The major failure modes are covered in another chapter and include device migration, graft material failure (holes), graft ultrafiltration, limb occlusion, infection, neck dilation, metallic fatigue fracture, endoleak, and rupture.[7-23] Often, multiple failure modes are combined, such as migration leading to endoleak (Figure 37–1). In early series, vascular surgeons frequently proceeded with open conversion as a standard approach to endovascular complications. This was highly effective but may have been overtreatment for some problems and was associated with high mortality rates.[24] As knowledge of the late failure modes was gained, less invasive and effective approaches have been developed for retreatment.

COMPLETE EXPLANT

Complete removal of the endograft is favored when there is unsuitable anatomy for an endovascular salvage or the graft is infected (see Figure 37–1). Familiar "tricks" derived from reoperative procedures for subsequent proximal aneurysms and open prosthetic graft infections can be used in these conversions. These include mobilization of the left renal vein or transaction of the left renal vein medial to the left adrenal and lumbar veins in a transperitoneal approach. Often, a retroperitoneal approach or supraceliac clamp is necessary to remove the endograft, particularly if there is a suprarenal component. Placement of ureteral stents can accelerate pelvic dissection when there are extensive iliac aneurysms and previously placed devices in the pelvis. Various methods to disengage hooks and barbs can be used but have in common the radial compression of the device before applying downward traction so as to minimize injury to the pararenal aorta. The graft can be snared, twisted, stents fragmented with wire cutters, digitally collapsed, or cooled to assist

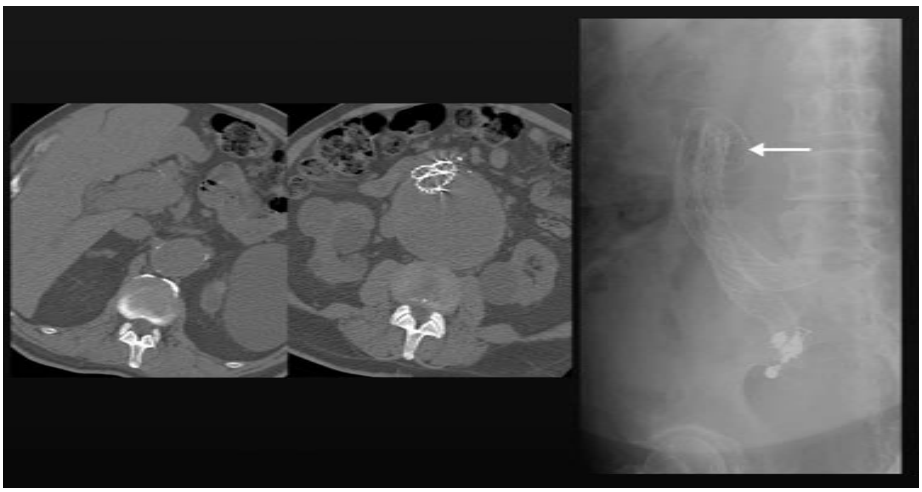


Figure 37-1. This patient presented 6 years after endovascular repair with an AneuRx device (Medtronic, Inc., Santa Rosa, CA). He had acute limb ischemia from iliac occlusion and a pulsatile aneurysm on physical examination. Computed tomography (CT) scan (left and middle) and abdominal radiograph (right) show the device has migrated out of the proximal neck and kinked (arrow). He had successful conversion and explant.

in removal. Often, an endarterectomy of the aortic cuff accompanies these maneuvers, and felt reinforcement of the proximal suture line is needed. Rarely, thoracoabdominal aortic reconstruction becomes necessary.

PARTIAL CONVERSION

An attractive alternative when infection is absent are partial explantations. If the retained components have not failed, then leaving an embedded suprarenal stent or iliac component can reduce the risk of vascular injury with complete explantation. Self-expanding stents can often be compressed with vascular clamps and still remain patent after unclamping. Other systems are relatively noncompressible, and balloon occlusion may assist in vascular control. Suturing to endografts is complicated by the presence of metallic stents that may cut the suture line or the surgeon. This problem can be minimized by careful selection of the transection point of the endograft between major stent elements; or for sinusoidal wireforms, cutting the wire in a single location and unwinding it to the point of transection, hence leaving a single wire termination that can be bent into a benign position. Suture retention attributes may have been sacrificed in favor of thinner materials and smaller introducer systems, and felt reinforcement is useful. When leaving parts of the endograft, suturing the surgical graft to the native wall and the endograft provides excellent fixation and isolates the retained segment from the main aneurysm should later failure occur in the retained portion.

Another form of partial conversion is to perform an extra-anatomic bypass, such as a femoral-femoral crossover bypass for limb occlusion. These procedures fall under the familiar domain of vascular surgeons' treating similar complications after open aortic repair.

RELINING

Relining an endograft is considered when the failure mode includes threatened or actual fabric holes (Figures 37–2 and 37–3), transgraft ultrafiltration (Figure 37–4), kinking of unsupported segments, stent compression/fracture, or component separation.[25-26] The main consideration is if the entire previous endograft needs relining rather than a more limited portion that is exposed to the aneurysm sac (Figure 37–5).[25] Sometimes, there may

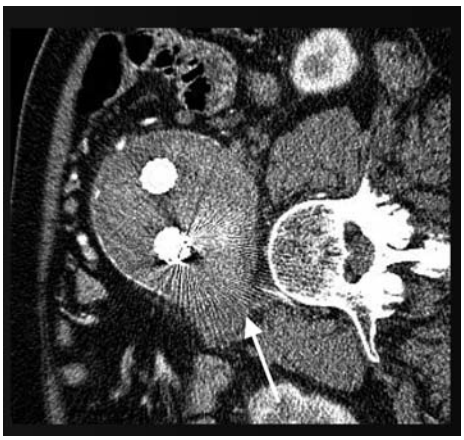


Figure 37-2. This patient presented with back pain 6 years after endovascular repair with an AneuRx device. White arrow points to aneurysm rupture diagnosed with CT scan.

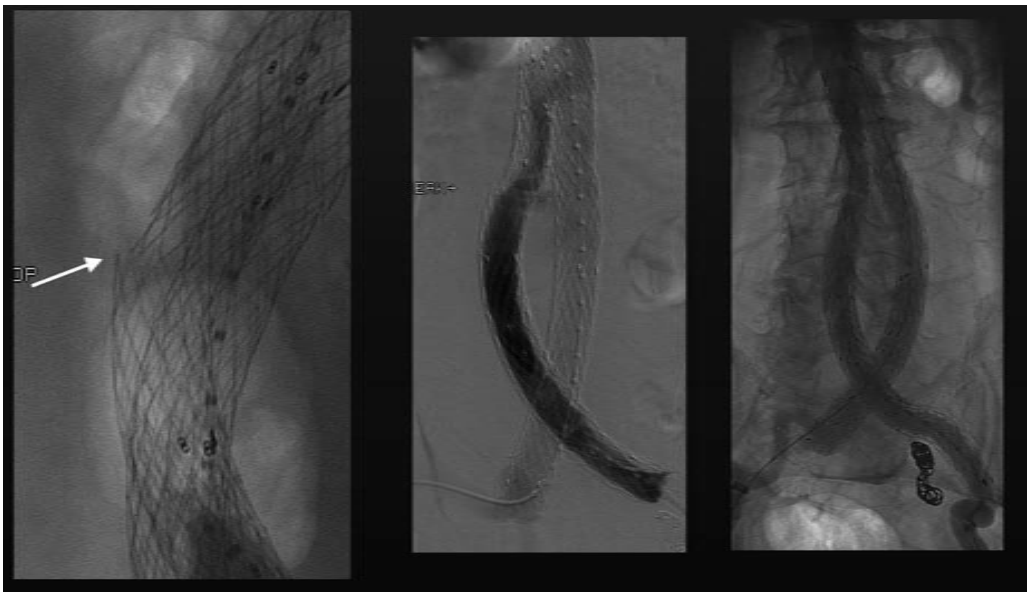


Figure 37-3. Same patient as Figure 37–2. Left panel is radiograph showing suture pop and stent displacement (white arrow). Middle panel is arteriogram showing fabric defect at the same location. Right panel shows completion angiogram after successful relining.

be a temptation to address only the most clinically significant fabric defect or fracture if it occurs in a single iliac limb, but the forces that led to failure of that portion are likely to lead to failure in other parts after a short latency period. It seems reasonable to reline as much as is anatomically feasible if failure is evident in part of the device. Exceptions may be a focal kink leading to late limb occlusion or an intercomponent junction that was never sufficiently overlapped.

EXTENSIONS

Extensions are useful when there is suitable residual anatomy for an endovascular approach after the device fixation has failed (i.e., migration) or in the small fraction of

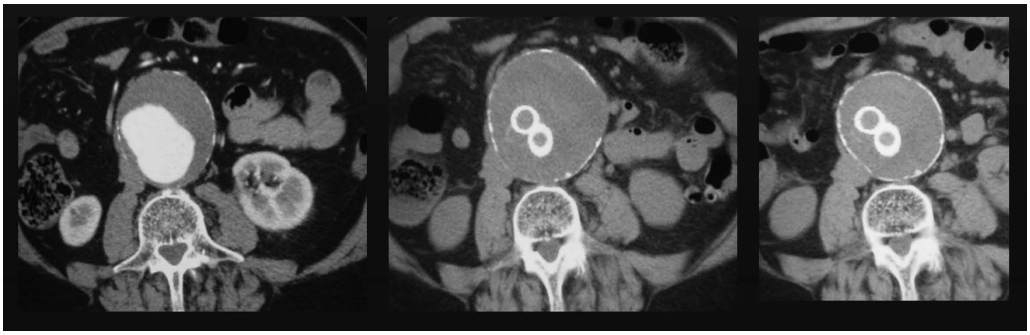


Figure 37-4. This patient had endovascular repair of a 7.0-cm aneurysm (left panel) with the original Excluder (WL Gore & Associates, Flagstaff, AZ) endoprosthesis made with expanded polytetrafluoroethylene. The aneurysm grew 9 mm in the first 8 months after repair (middle panel). After relining, the aneurysm shrunk 20 mm over the subsequent 7 months (right panel).

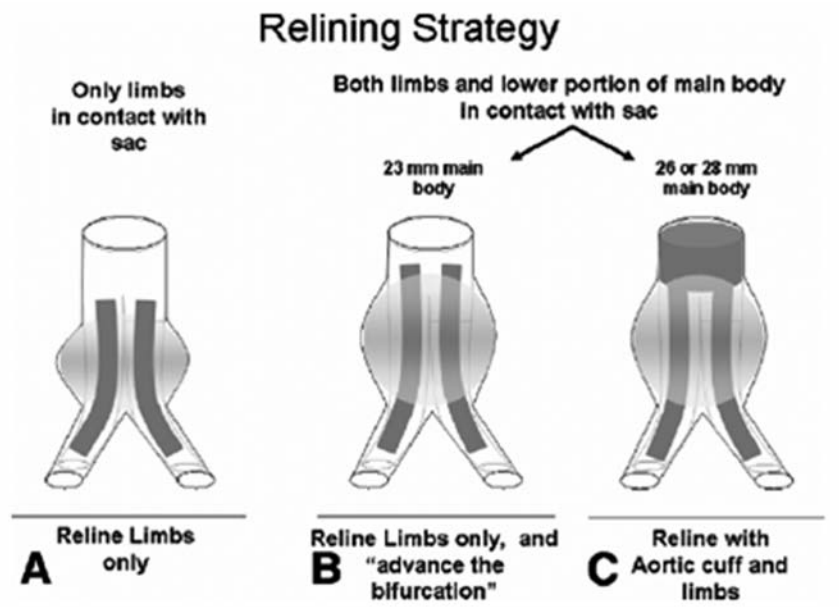


Figure 37-5. Options for complete or partial relining of endograft in contact with sac. (With Permission from Goodney PP, Fillinger MF J Vasc Surg 2007;45:686–93.)

patients who experience clinically important dilation of the native aorta.[7-12,20] Extension to the external iliac artery (with embolization of the hypogastric artery) is not an uncommon procedure for patients long after an initial endovascular repair (Figure 37–6).[27] This enlargement and late endoleak may happen 8 or more years after initial treatment, just as subsequent aneurysms are known to form decades after an open repair.

EMBOLIZATION

The armamentarium of devices, delivery catheters, techniques, and imaging systems continues to expand the capabilities of embolization (see Figure 37–6). Using the latest-generation coils and liquid embolic agents, triaxial and microcatheter systems, modern rotational angiography, and road mapping with blended CT fluoroscopy has greatly extended treatment of endoleaks and reduced procedure times. Needle tracking, robotic navigation, and morphing of catheters and wires are less impressive, and it is not clear if these innovations are useful adjuncts or superfluous technology.

Figure 37-6. Late distal iliac artery dilation with type I endoleak (left panel). Middle panel shows embolic device occluding proximal hypogastric artery and preserving collateral pathways. Right panel is completion arteriogram with extension to external iliac artery.

LAPAROSCOPIC CLIPPING

A variant approach to transcatheter embolization is a laparoscopic clipping of lumbar, inferior mesenteric, and middle sacral branch arteries. These procedures are highly effective in skilled hands.[28]

SAC FENESTRATION

Also described as controlled sac rupture, fenestration of the sac is performed for transgraft ultrafiltration. Large fenestrations are necessary to prevent healing and recurrent sac growth. This procedure has also been described after repair with standard surgical grafts.[28-29]

OBSERVATION

Advances in imaging and postprocessing analysis have provided unprecedented resolution and new quantification such as volumetric analysis or sac pressure. Like subsegmental pulmonary emboli and calf muscle deep vein thrombosis, the high-quality evidence to guide clinical management has lagged behind the technology. Many findings like small endoleaks, minor growth of aneurysm volume, and suture pops can be imaged and identified, but what should be done? Clinical judgment and discretion are needed to avoid overtreatment.[30-31] Even with large type I endoleaks or threatened component separation, an observational course may be preferred in fragile patients similar to abstaining from primary treatment of aortic aneurysms in unfit patients (Figure 37–7).

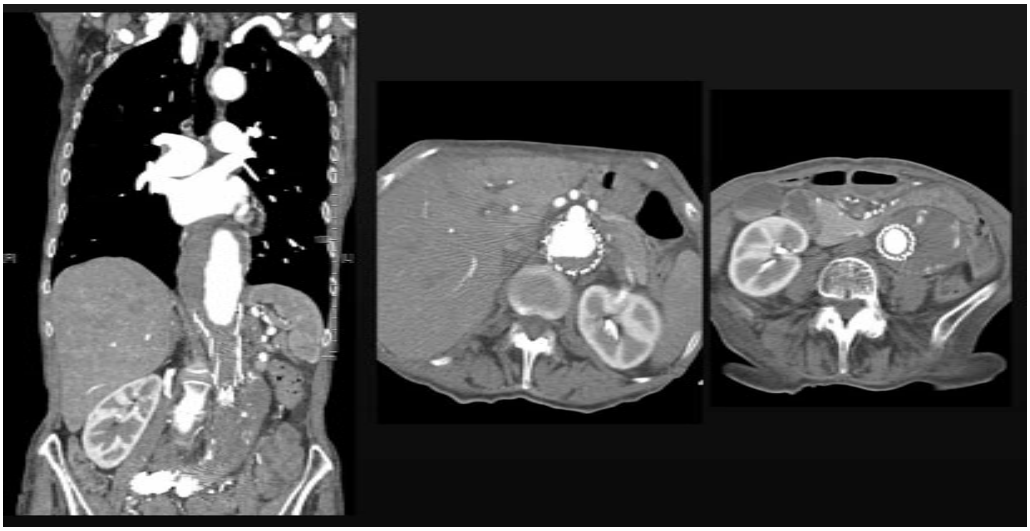


Figure 37-7. CT of a fragile octogenarian patient with a branched endovascular repair with a Zenith (Cook Incorporated, Bloomington, IN) device. Left panel shows asymptomatic subsequent 6-cm thoracic aneurysm above bare topstent component. Middle panel shows patent branch grafts. Right panel shows large excluded abdominal aneurysm. Observation is recommended.

CONCLUSION

Endovascular aneurysm repair has clearly improved perioperative mortality and initial treatment of abdominal aortic aneurysms. Late device failure modes have been identified in clinical trials and by astute physicians, and device engineering solutions are emerging. Because of the many years before recognition of late complications and the slow product development cycle, clinicians have and will continue to encounter patients with these difficult late problems. Learning to recognize and treat these problems should reduce late mortality until device innovation addresses more of the shortcomings of currently used devices.

REFERENCES

1. Kung HC, Hoyert DL, Xu JW, Murphy SL. National Vital Statistics Report. National Center for Health Statistics; Hyattsrille, MD 2008.
2. Parodi JC, Palmaz JC, Barone HD. Transfemoral intraluminal graft implantation for abdominal aortic aneurysms. Ann Vasc Surg 1991;5:491–9.
3. Volodos NL, Karpovich IP, Troyan VI, et al. Clinical experience of the use of self-fixing synthetic prostheses for remote endoprosthetics of the thoracic and the abdominal aorta and iliac arteries through the femoral artery and as intraoperative endoprosthesis for aorta reconstruction. Vasa Suppl 1991;33:93–5.
4. Greenhalgh RM, Brown LC, Kwong GP, et al. Comparison of endovascular aneurysm repair with open repair in patients with abdominal aortic aneurysm (EVAR trial 1), 30-day operative mortality results: randomised controlled trial [see comment]. Lancet 2004; 364:843–8.
5. Blankensteijn JD, de Jong SE, Prinssen M, et al. Two-year outcomes after conventional or endovascular repair of abdominal aortic aneurysms.[see comment]. N Engl J Med 2005; 352:2398–405.
6. Schermerhorn ML, O'Malley AJ, Jhaveri A, et al. Endovascular vs. open repair of abdominal aortic aneurysms in the Medicare population [see comment]. N Engl J Med 2008;358: 464–74.
7. Ebaugh JL, Eskandari MK, Finkelstein A, et al. Caudal migration of endoprostheses after treatment of abdominal aortic aneurysms. J Surg Res 2002;107:14–7.
8. Cao P, Verzini F, Zannetti S, et al. Device migration after endoluminal abdominal aortic aneurysm repair: analysis of 113 cases with a minimum follow-up period of 2 years. J Vasc Surg 2002; 35:229–35.
9. Zarins CK, Bloch DA, Crabtree T, et al. Stent graft migration after endovascular aneurysm repair: importance of proximal fixation. J Vasc Surg 2003;38:1264–72; discussion 1272.
10. Sternbergh WC 3rd, Money SR, Greenberg RK, et al. Influence of endograft oversizing on device migration, endoleak, aneurysm shrinkage, and aortic neck dilation: results from the Zenith Multicenter Trial. J Vasc Surg 2004;39:20–6.
11. Azizzadeh A, Sanchez LA, Rubin BG, et al. Aortic neck attachment failure and the AneuRx graft: incidence, treatment options, and early results. Ann Vasc Surg 2005;19:516–21.
12. Fulton JJ, Farber MA, Sanchez LA, et al. Effect of challenging neck anatomy on mid-term migration rates in AneuRx endografts. J Vasc Surg 2006;44:932–7; discussion 937.
13. Brown KE, Heyer KS, Matsumura JS, Eskandari MK. Late type III endoleak and graft failure of an Ancure stent graft. JVIR 2008;19:1506–8.
14. Bertges DJ, Chow K, Wyers MC, et al. Abdominal aortic aneurysm size regression after endovascular repair is endograft dependent. J Vasc Surg 2003;37:716–23.
15. Peterson BG, Matsumura JS, Brewster DC, Makaroun MS, Excluder Investigators. Five year report of a multicenter controlled clinical trial of open versus endovascular treatment of abdominal aortic aneurysms. J Vasc Surg 2007;45885–90.

16. Tang G, Fillinger M, Matsumura JS. Original excluder component overlap from proximal or distal extension during initial repair not correlated with aneurysm sac shrinkage. J Vasc Surg 2009.
17. Cornelissen SA, Verhagen HJ, Prokop M, et al. Visualizing type IV endoleak using magnetic resonance imaging with a blood pool contrast agent. J Vasc Surg 2008;47:861–4.
18. Carroccio A, Faries PL, Morrissey NJ, et al. Predicting iliac limb occlusions after bifurcated aortic stent grafting: anatomic and device-related causes. J Vasc Surg 2002;36:679–84.
19. Heyer KS, Modi PK, Morasch MD, et al. Secondary infection of thoracic and abdominal aortic endografts. JVIR 2009;20:173–9.
20. Matsumura JS, Chaikof EL. Continued expansion of aortic necks after endovascular repair of abdominal aortic aneurysms. EVT Investigators. J Vasc Surg 1998;28:422–30; discussion 430–1.
21. Bernhard VM, Mitchel RS, Matsumura JS, et al. Ruptured abdominal aortic aneurysm after endovascular repair. J Vasc Surg 2002;35:1155–62.
22. White GH, May J, Waugh RC, et al. Type III and type IV endoleak: toward a complete definition of blood flow in the sac after endoluminal AAA repair. J Endovasc Surg 1998;5: 305–9.
23. White GH, Yu W, May J, et al. Endoleak as a complication of endoluminal grafting of abdominal aortic aneurysms: classification, incidence, diagnosis, and management. J Endovasc Surg 1997; 4:152–68.
24. May J, White GH, Yu W, et al. Conversion from endoluminal to open repair of abdominal aortic aneurysms: a hazardous procedure. Eur J Vasc Endovasc Surg 1997;14:4–11.
25. Goodney PP, Fillinger MF. The effect of endograft relining on sac expansion after endovascular aneurysm repair with the original-permeability Gore Excluder abdominal aortic aneurysm endoprosthesis. J Vasc Surg 2007;45:686–93.
26. Kougias P, Lin PH, Dardik A, et al. Successful treatment of endotension and aneurysm sac enlargement with endovascular stent graft reinforcement. J Vasc Surg 2007;46:124–7.
27. Rhee RY, Muluk SC, Tzeng E, et al. Can the internal iliac artery be safely covered during endovascular repair of abdominal aortic and iliac artery aneurysms? Ann Vasc Surg 2002; 16:29–36.
28. Van Sambeek MRHM, Hendriks JM, Tseng L, et al. Sac enlargement without endoleak: when and how to convert and technical considerations. Semin Vasc Surg 2004;17:284–7.
29. Risberg B, Delle M, Lonn I, et al. Management of aneurysm sac hygroma. J Endovasc Ther 2004;11:191–5.
30. Mennander A, Pimenoff G, Heikkinen M, et al. Nonoperative approach to endotension. J Vasc Surg 2005;42:194–9.
31. Thoo CHC, Bourke BM, May J. Symptomatic sac enlargement and rupture due to seroma after open abdominal aortic aneurysm repair with polytetrafluoroethylene graft: implications for endovascular repair and endotension. J Vasc Surg 2004;40:1089–94.

38

Late Failure of Endovascular Abdominal Aortic Aneurysm Repair

Donald T. Baril, M.D. and Michel S. Makaroun, M.D.

INTRODUCTION

The treatment of abdominal aortic aneurysms (AAAs) has changed radically over the past 15 years since the introduction of endovascular abdominal aortic aneurysm repair (EVAR). EVAR has been shown to lead to decreased early mortality, shorter hospital stays, and lower perioperative complications.[1-2] However, despite these early benefits, the long-term durability of EVAR has been in question since it was first reported, and studies have shown no survival benefit for patients undergoing EVAR over patients undergoing open repair after 1 year.[3]

EVAR is associated with its own unique complications, both early and late, which differ from those of conventional open repair. Although certain devices, particularly the early-generation stent grafts, have been associated with higher rates of both early and late complications, most have been described with nearly all devices.[4] Along with improved device technology, EVAR failures have also decreased over time with physician experience and improved preoperative planning. Despite advances in device technology and operator experience, however, a number of late failure modes continue to plague the long-term durability of EVAR. These late failures include endoleaks, graft migration, limb occlusions, landing zone degeneration, material fatigue and failure, and graft infection. Although many of these late failures can be treated with secondary endovascular interventions, particularly when patients are monitored appropriately, they carry their own inherent risks and potential for significant morbidity and mortality.

FAILURE MODES

Endoleaks

Endoleaks, first described as the "persistence of blood flow outside the lumen of the endoluminal graft but within an aneurysm sac,"[4] continue to complicate the long-term

effectiveness of EVAR. Endoleaks are classified into those that result from inadequate seal at either the proximal or the distal stent-graft attachment site (type I); those that originate through retrograde flow in the collateral side-branch arteries of the aneurysm, typically lumbar and inferior mesenteric arteries (type II); those arising from antegrade flow into the aneurysm at a junction point between graft components or a graft defect (type III); and those with inflow related to the porosity of the graft itself (type IV). Additionally, endotension, often referred to as type V endoleak, has been defined as "persistent or recurrent pressurization of the aneurysm sac following endovascular repair" without a demonstrable leak on imaging studies.[5-6]

Type I and III endoleaks, which have incidences of 8.2 to 18% and 0.7 to 8% following EVAR, respectively,[7-8] carry the greatest risk of aneurysm rupture and should almost all uniformly be treated. Conversely, the natural course of type II endoleaks, which have a reported incidence of 8 to 45% following EVAR,[9-10] is less well understood, because many spontaneously thrombose. However, aneurysm expansion has been reported with type II endoleaks, and at the very least, close clinical observation is warranted. Type IV endoleaks typically resolve without intervention. Type V endoleaks often pose the greatest diagnostic and therapeutic dilemma and require intervention if aneurysm growth persists. Endoleaks may be managed with graft extension or cuff insertion, coil embolization, laparoscopic ligation of the feeding vessels, conversion to standard open repair, or simply observation in the correct clinical setting (Figure 38–1).

Migration

Stent-graft fixation may be either passive or active. Passive fixation relies on friction generated by the radial strength of the stent, which is oversized to the artery in which it is being deployed. Active fixation relies on not only friction but additionally some form of hook or other anchoring mechanism that is embedded in the stent-graft itself and engages the arterial wall.

Migration is a result of failure of the fixation mechanism of the stent-graft related to the device itself or degeneration of the landing zone, leading to either caudal movement of the device $>$10 mm at the proximal neck or cranial movement at the iliac attachments relative to anatomic landmarks or any degree of movement associated with a clinically adverse event. Although some migrations may not necessarily be clinically significant so long as the aneurysm remains excluded, many lead to loss of the proximal or distal seal and subsequent pressurization of the aneurysmal sac. In addition to loss of aneurysm exclusion, migration may lead to graft kinking and occlusion or graft disconnection. Migration typically occurs at least 1 year following deployment but has been reported at any time from days following implantation to 4 to 5 years later.

Preoperative assessment and planning for EVAR will help minimize migration, because appropriately sizing an endograft to a healthy segment of aorta or iliac artery is vital to successful long-term aneurysm exclusion. A number of specific anatomic factors have been shown to be associated with migration over time, including severely angulated aortic necks, conical neck shapes, wide necks, short necks, and necks containing large amounts of thrombus or those with heavy calcification.[11-12] In addition to anatomic factors related to the proximal attachment zone, distal iliac fixation is also important in preventing migration. Secure fixation to the iliac bifurcation helps to minimize the risk of migration by providing support along the longitudinal axis of the stent graft.[13] Based on this, extension of both iliac limbs to cover the entire common iliac artery to the iliac bifurcation may contribute to endograft stability.

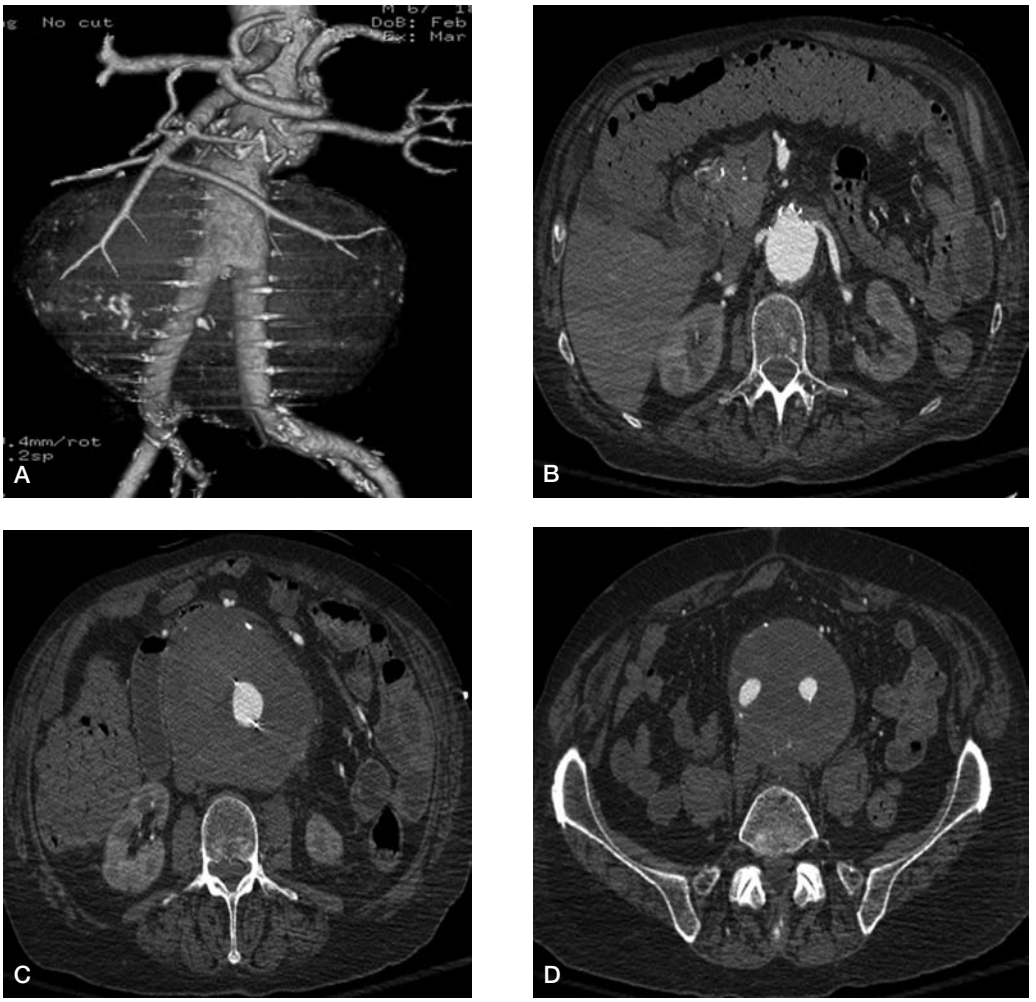


Figure 38-1. A. A 3-D CT angiogram demonstrating a 12-cm AAA 9 years following initial Ancure placement. B-D. Axial cuts demonstrating the same AAA without gross evidence of an endoleak. The patient went on to open conversion, at which time several type II lumbar endoleaks were discovered.

Device-related causes of migration may, however, be more important. Design of the device, lack of flexibility, and the mechanism of passive fixation may all contribute to the migration. Moreover, inherent failure of the device itself, including hook fractures, stent fractures, or separation of the stent from the graft, may also be at fault. Although migration has been described with nearly every endograft, including those with active fixation in the absence of graft failure, the AneuRx device has demonstrated higher rates of migration compared with other devices (up to 32% at 36 months).[12,14] Conversely, endografts with active proximal fixation mechanisms, including the Excluder and the Zenith devices, have been associated with much lower rates of migration.[15-16] Additional factors that may contribute to migration include the degree of oversizing of the endograft to the landing zone vessel and the type of stents used in the construction of the endograft.

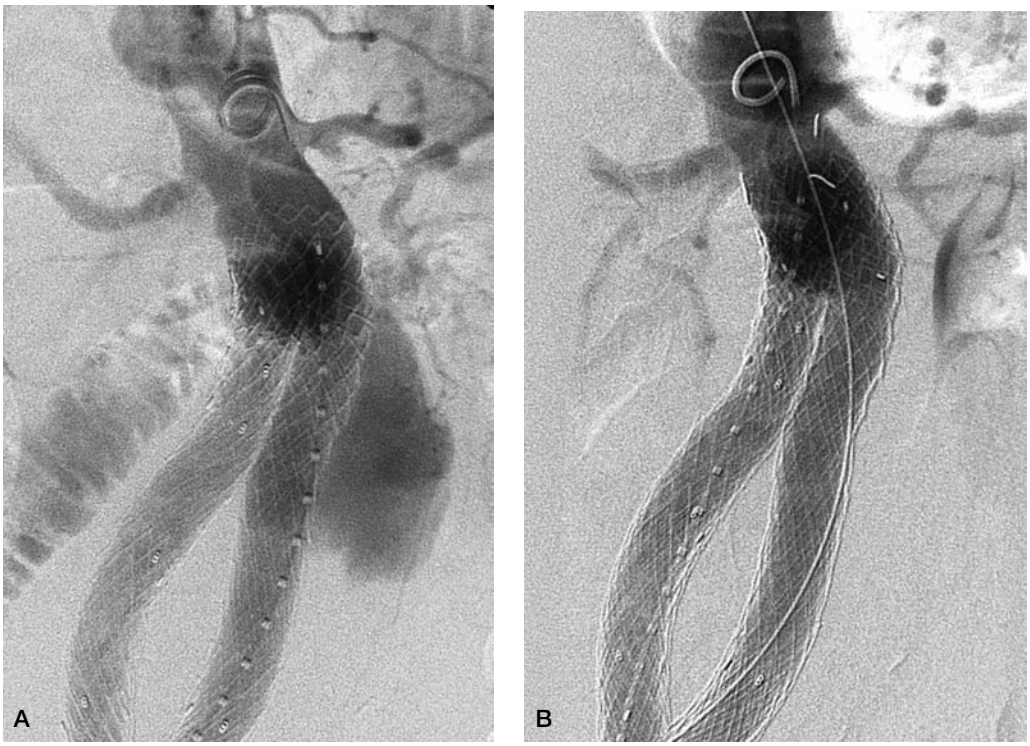


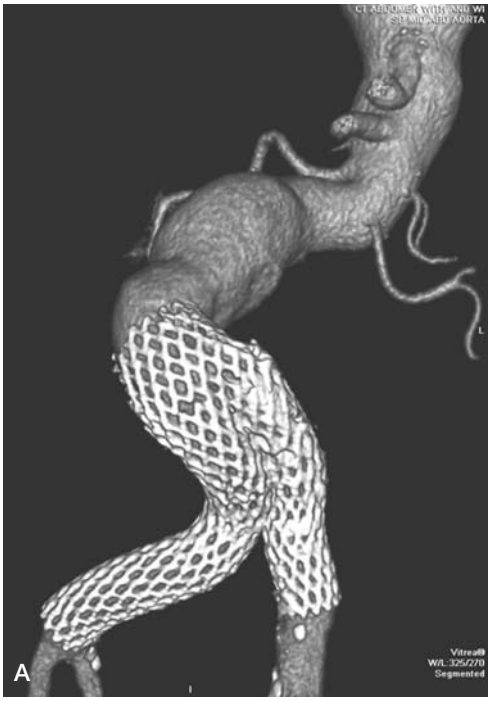
Figure 38-2. A. Aortogram of distal migration of AneuRx device and proximal type I endoleak. B. Successful AAA exclusion following aortic cuff extension.

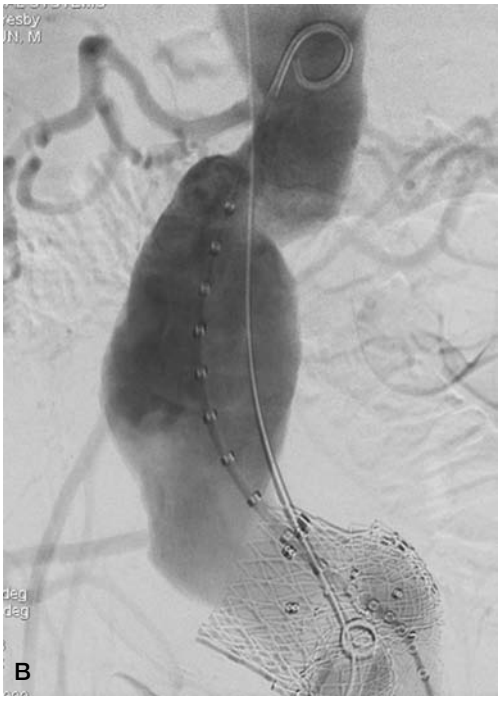
In patients in whom migration is discovered, intervention is warranted when the seal zone is 5 mm or less or there is evidence of a clinically or radiographically significant event such as a type I endoleak or aneurysm enlargement. Treatment options include placement of a proximal aortic cuff (Figure 38–2), placement of an entire new aortic endograft (Figure 38–3), or open conversion (Figure 38–4).

Landing Zone Enlargement

Landing zone enlargement, either proximal or distal, may occur following EVAR. Although this may often be clinically insignificant, landing zone enlargement may ultimately lead to the development of stent-graft migration, type I endoleak, and subsequent repressurization of the aneurysm sac. Despite a large amount of literature regarding this entity, there is a great deal of variability in the data due in large part to the variance in the methods used to measure the aortic neck and the timing of these measurements.

Proximal neck dilation may occur following both EVAR and open repair, although the rates appear to be lower with EVAR. The occurrence of aortic neck dilation following open repair implies that the aorta in the setting of an infrarenal AAA probably is diseased throughout and hence will dilate over time. Although most aortic necks remain stable after EVAR, approximately 20 to 30% demonstrate some degree of enlargement at 2 years.[15] This phenomenon does not appear to be device-related among devices with similar deployment methods, although select studies have demonstrated higher rates of aortic neck dilation in patients treated with self-expanding devices.





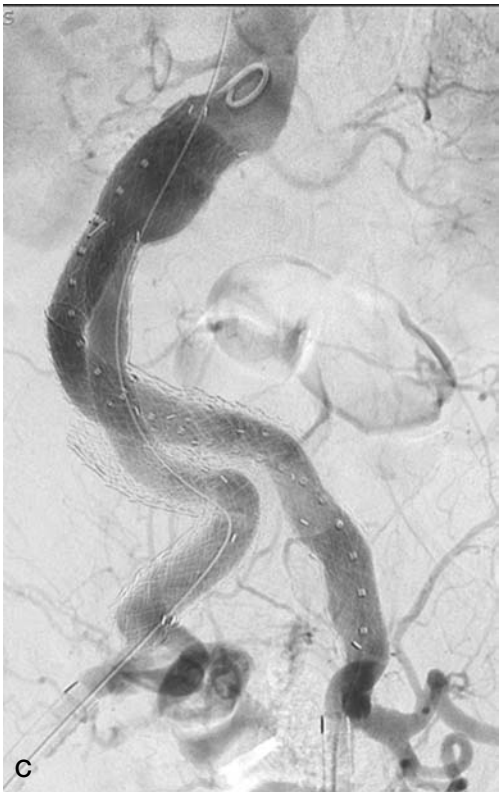


Figure 38-3. a-b. CT angiogram and aortogram of distal migration of Aneurx device. c. Successful AAA exclusion following placement of entire secondary endograft.

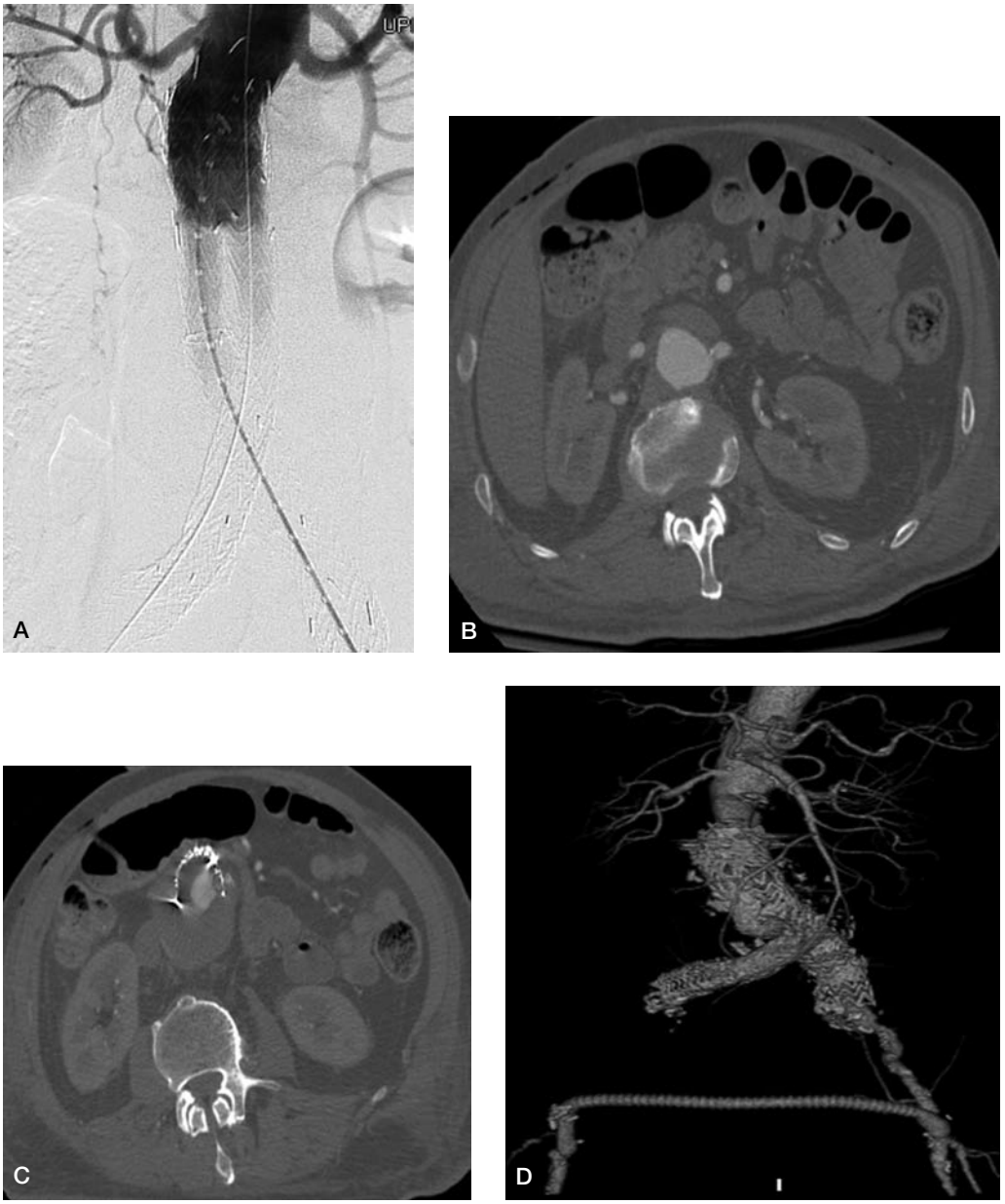


Figure 38-4. A. Completion aortogram with AAA exclusion following Gore Excluder placement along with aortic cuff extension. B-D. CT angiogram obtained 4 years later in a patient who was lost to follow-up and developed limb occlusion requiring femorol-femoral bypass following an emergent coronary bypass. CT demonstrates distal migration and neck dilation. The patient was treated with conversion using an aortobifemoral bypass following recovery from his coronary bypass.

In addition to device type, device sizing appears to have an impact on aortic neck dilation, and an oversizing of 10 to 20% appears to be most appropriate. Oversizing up to 30% has been associated with a higher incidence of aortic neck dilation. Specific anatomic factors that may have an impact on aortic neck dilation include

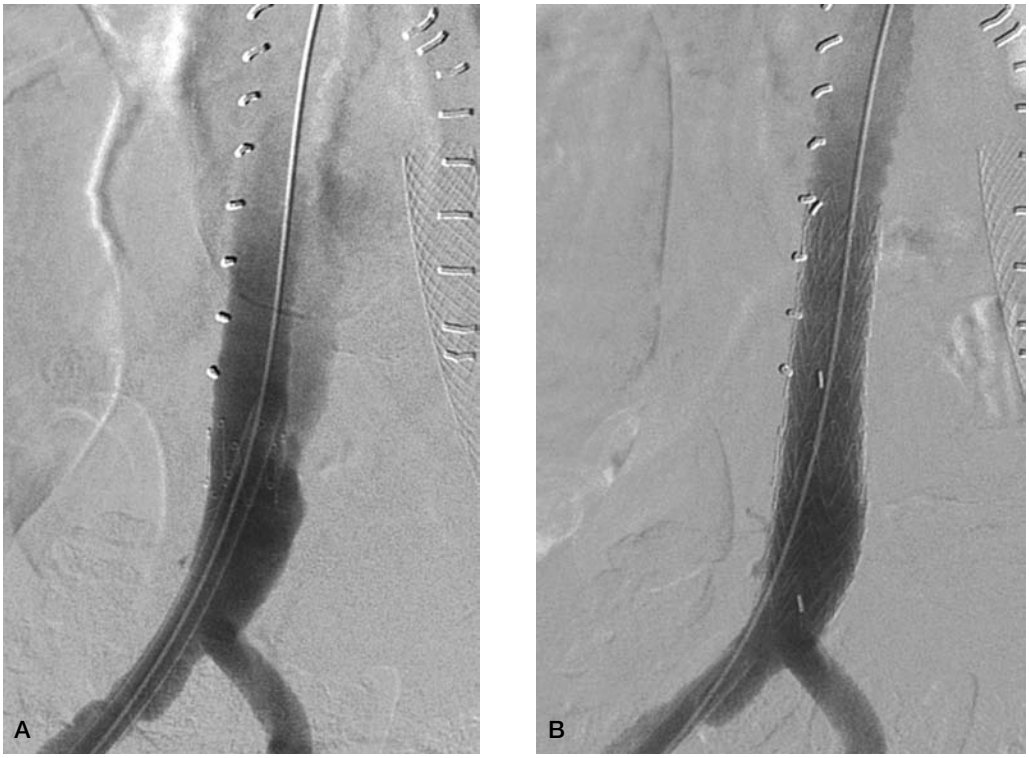


Figure 38-5. A. Distal type I endoleak secondary to dilation at iliac landing zone. B. Completion angiogram following treatment with iliac limb extension.

initial aortic neck size and aneurysm diameter. In particular, patients with larger aortic necks, larger overall AAA diameters, and those with circumferential thrombus at the level of the aortic neck are at higher risk for aortic neck dilation over time (Figure 38–5).

Distal landing zone degeneration in the iliac arteries may occur as well, leading to graft migration and distal type I endoleaks (Figure 38–5). As with the proximal neck, anatomic factors contributing to this phenomenon include large initial iliac size and the presence of circumferential thrombus.

Limb Occlusions

Limb occlusions are rare and most commonly reported within the first 90 days following implantation in grafts with unsupported limbs. However, limb occlusion may also occur in supported grafts and this has been documented as a late occurrence in all endografts. It has been postulated that conformational changes that occur over time with aneurysm remodeling may impact the patency of endograft limbs in supported devices. Aneurysm sac shrinkage occurs in many patients following EVAR, which may introduce kinks into the iliac limbs of some endografts, because some limb designs are less able to conform to changes in aneurysm sac anatomy. This presents a potentially adverse outcome in that sac shrinkage, a desired outcome of EVAR, may predispose patients to graft limb occlusion if kinks occur as a result of aneurysm remodeling.

The etiology of limb occlusions may be divided into anatomic factors and graft-related factors. The most common anatomic factors leading to limb occlusion include tortuous iliac artery anatomy and preexisting iliac stenosis. These anatomic characteristics may lead to kinking and narrowing of the endograft limbs. Similarly, a narrow aortic bifurcation can compress the iliac graft limbs because it must accommodate two limbs that are each equal to or greater than one half the size of the main body portion of the endograft.[17]

Dissection of the femoral or iliac arteries that is either preexisting or created at the time of graft implantation may also cause endograft limb occlusion due to poor outflow. There is also an association between small size of the iliac artery and graft limb occlusion. Women are at higher risk of graft limb occlusion than men, which is presumably because of the smaller size of the iliac arteries. Extending the stent-graft into the external iliac artery has also been found to contribute to limb occlusion, because there is commonly some degree of angulation at the origin of the external iliac artery in an area susceptible to atherosclerotic disease and calcifications.[18]

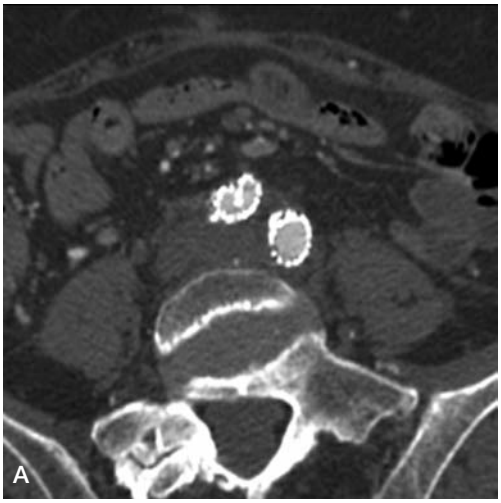
Furthermore, this extension limits the runoff by eliminating internal iliac artery flow and ends the limb in a smaller-diameter vessel.

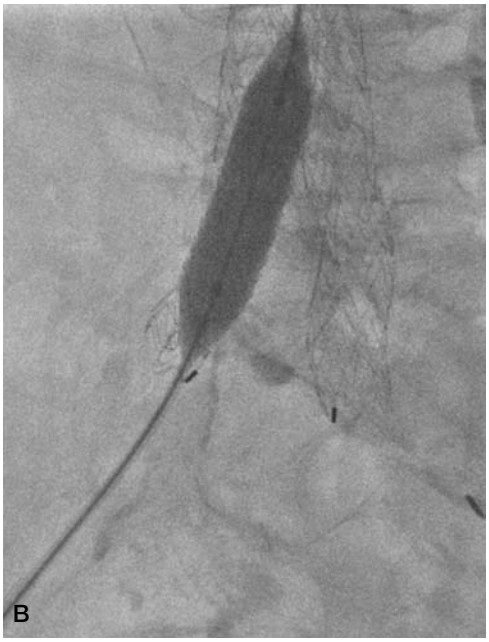
Endograft-related charateristics are also a major causative factor in the development of graft limb occlusion. Excessive oversizing of the endograft can be a significant cause of limb or occlusion secondary to infolding of the graft material within the lumen. The most significant graft-related cause of limb occlusion, however, is the lack of support within the structure of the endograft. Limb occlusion with the Ancure graft was not infrequent secondary to either external compression or twisting of the limbs arising from the unibody construction and lack of limb support. Twisting of the graft limbs usually occurs during deployment of the endograft, narrows the lumen, and predisposes to thrombosis (Figure 38–6).

Patients who develop limb occlusion will typically complain of acute onset of pain and paresthesias in the affected extremity, but occasionally present only late after they notice the development of claudication. The femoral pulse is always absent and they manifest varying degrees of sensory dysfunction. The majority of patients do not present with threatened extremities that would necessitate expeditious surgical revascularization. Options for treatment include either endovascular or open surgical approaches. Endovascular approaches include thrombolysis, pharmacomechanical thrombectomy, or angioplasty and stenting (with either bare metal stents or additional endografts). Surgical options include conventional thrombectomy or extra-anatomic bypass.

Thromboembolectomy may be problematic in the EVAR population due to the potential for disruption or damage to the graft. Mechanical thrombectomy with a balloon catheter is hazardous, because it might dislodge the endograft that, in many cases, is held in place by radial force or self-expanding stents. Likewise, standard surgical thrombectomy may cause component separation due to traction of the balloon on the endograft. This concern about potential hazards of mechanical or surgical thrombectomy has led many to advocate extra-anatomic bypass for extremity reperfusion in cases of graft limb thrombosis.

Although open surgical repair has its proponents, many prefer an endovascular approach to graft limb occlusion. Pharmacomechanical thrombectomy along with simple thrombolysis may prove to be beneficial, because both are able to debulk the thrombus load and may uncover a kink in the graft limb or a previously unrecognized area of stenosis in the iliac artery.





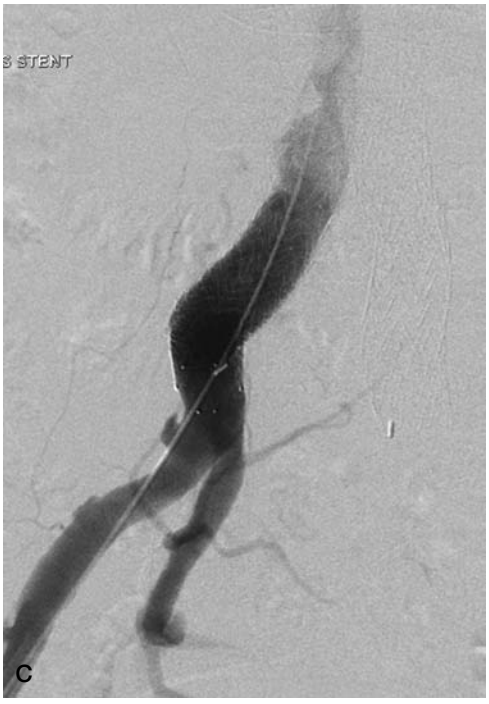


Figure 38-6. A. Right iliac limb endograft inflowing due to oversizing. B-C. Angiogram showing infolding treatment with angioplasty and stenting.

Material Failure

Material failure remains one of the most concerning modes for potential late EVAR failure, encompassing the breakdown of the intrinsic mechanical parts of the stent graft. It is often difficult to identify material failure because patients typically are asymptomatic at the time of presentation. Many of the first identified stent fractures were initially recognized within explanted stent graft devices that had been removed for evidence of aneurysm expansion or recovered at autopsy. The difficulty in identifying device failure limits a true understanding of the magnitude of the problem, because patients are rarely imaged closely enough during

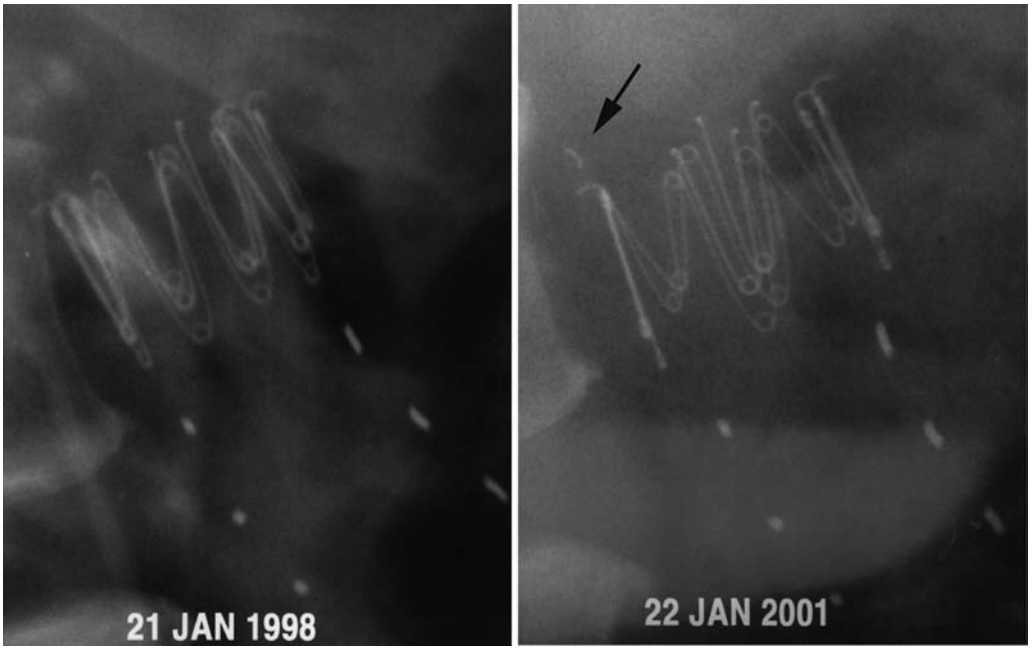


Figure 38-7. Attachment hook separation 3 years following initial EVAR.

their follow-up examinations to identify all the potential material breakdowns. Furthermore, the clinical significance of many identified device failures is unknown.[19]

Material failures may occur in the form of metal fractures, suture breakdown, or fabric erosion. Metal fractures can occur at any point throughout the graft, including the proximal or distal attachments, along the graft body, or involving the attachment hooks. Early-generation devices that suffered from fracture of the proximal attachment hooks were associated with distal migrations and development of type I endoleaks (Figure 38–7), The impact of mid-body stent fractures is less clear, but the risk of a sharp metal edge puncturing the fabric leading to a type III endoleak exists. Similarly, suture breaks leading to separation of the stent from the graft and subsequent friction between metal and fabric may occur, again ultimately leading to fabric tears. Such fabric tears have been seen in patients in whom bare metal stents have been placed in unsupported portions of endografts leading to the aforementioned phenomenon and resultant type III endoleak (Figure 38–8). In addition to the potential for the metal of the stent wearing on the fabric, potential exists for sharp calcifications in the arterial wall to compromise the fabric.

Patients who develop material fatigue may be asymptomatic or may present with new endoleaks and aneurysm enlargement. The majority of these may be treated with subsequent relining or, in select cases, endograft extension, but some patients will go on to require open conversion.

Component Separation

Component separation occurs primarily as a result of inadequate overlap between endografts at junction points. Following EVAR, there is typically some change in the anatomy of the AAA sac and its adjacent structures that contributes to changes or forces exerted on the

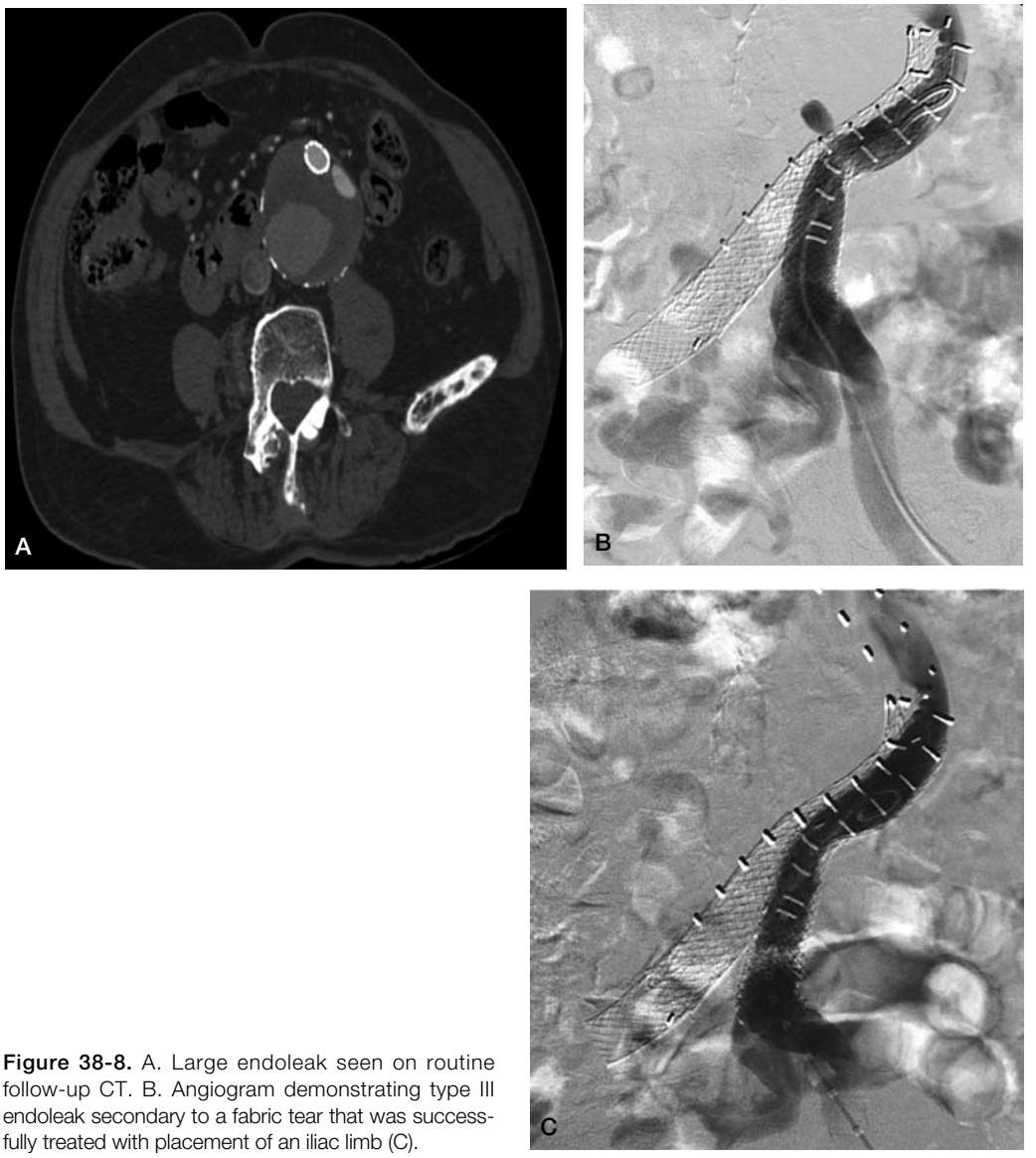


Figure 38-8. A. Large endoleak seen on routine follow-up CT. B. Angiogram demonstrating type III endoleak secondary to a fabric tear that was successfully treated with placement of an iliac limb (C).

endograft, possibly leading to component separation. This has become a relatively rare complication owing to improved device design along with operator experience. Furthermore, appropriate surveillance helps to avoid failure prior to its occurrence by careful examination of the position of the endograft components. When component separation does occur, patients typically develop a type III endoleak that may commonly be managed using endovascular techniques via a bridging endograft.

Hygroma and Endotension

Graft porosity can lead to a type IV endoleak in the early postoperative period. These endoleaks tend to seal spontaneously following reversal of anticoagulation. However, in

certain stent grafts, namely the original Excluder device, graft porosity has led to endotension in some patients; this is thought to be the result of transmural movement of serous fluid across the expanded polytetrafluoroethylene (PTFE) material, thus resulting in hygroma formation.[20] When no endoleak can be identified after aggressive evaluation, continued sac growth has been attributed to endotension. Aneurysm sac hygroma has been documented at the time of open repair, and in vitro studies demonstrate that the particular device construct correlates with the degree of plasma permeability. Apparent endotension has resulted in rupture of the aneurysm wall following EVAR, often without clinical sequelae.

The original Excluder endograft (W. L. Gore & Associates, Inc, Flagstaff, AZ) was noted in clinical trials to be associated with less sac regression and more sac growth than some other commercial devices. However, since that time, a modified endograft with a low-permeability layer to reduce fluid flow across the graft material has been introduced. Patients with this newer device have demonstrated significant aneurysm sac regression and minimal sac expansion, implying that the low-porosity fabric used in the construction of endograft seems to be an important factor in early aneurysm sac shrinkage.[21]

The treatment of patients who demonstrate sac expansion in the absence of endoleak is unclear. Rupture of the sac in these patients may not be of major clinical significance, because there is no egress of blood outside of the endograft wall. Concern about short necks becoming effaced with enlarging sacs, although unproved, adds a theoretical disadvantage to long-term observation. Alternatives to open conversion include emptying the sac by means of aspiration, exploration and reclosing of the sac, or performing a window in the sac laparoscopically. However, the simplest has proven to be an endovascular relining of the endograft with the modified low-porosity device.

Infection

The incidence of infection following EVAR appears to be less than after open repair. Although the incidence of graft infections following EVAR is not well-defined, given the low overall reported incidence, it appears to be less than 0.5%.

Given the low incidence of graft infections following EVAR, there have been few significant statistical analyses of factors that contribute to infection.[22-23] However, certain risk factors have been postulated to contribute to a higher risk. One of these factors has been performing the initial procedure in an interventional radiology suite as opposed to performing the procedure in an operating room. The hypothesis is that sterile technique is followed less stringently and the use of periprocedure antibiotics less well regulated than in the operating room. An additional factor that has been repeatedly identified is the performance of concomitant endovascular procedures at the time of endograft implantation, primarily coil embolization. The relationship here is unclear, but it has been hypothesized that these procedures add additional time and steps (including wire, catheter, and sheath exchanges) to the implantation, allowing for greater potential for breaks in sterile technique. Other factors that may contribute to late graft infections include an immunocompromised state, bacterial infection leading to bacteremia, and postoperative infection at the femoral access site.

Given the low incidence of these infections, their treatment remains ill-defined, but surgical treatment has been performed for most cases. Following the established surgical principle involving graft infections after conventional open aortic aneurysm repair, the widespread use of surgical treatment is in accordance with the premise that

an infected prosthesis should always be completely removed if the patient's medical condition allows. Overall mortality appears to be low, however, when compared with mortality reported for graft infection after open repair (18% vs. 40 to 70%). Options, as with infection following open repair, include graft excision and thorough débridement with extra-anatomic bypass or in situ replacement, either in a staged fashion or at the same setting. In situ replacement has been performed using Dacron prostheses, PTFE grafts, and autologous vein grafts. For select high-risk patients without evidence of overwhelming sepsis, long-term suppressive antibiotics with surgical drainage may be the treatment of choice, although this will likely not fully eradicate the infection (Figure 38–9).

Aortoenteric Fistula (AEF)

Primary aortoenteric fistulas have a reported incidence of 0.04 to 0.07%, whereas the development of secondary aortoenteric fistulas complicates 0.36 to 1.6% of all aortic operations.[24] Although the overall incidence of aortoenteric fistula seems to be decreasing and the diagnostic delays are shorter than they were historically, morbidity and mortality after repairs remain high. Aortoenteric fistula most commonly occurs at the third and fourth parts of the duodenum, usually coinciding with the anastomosis of the proximal aorta; however, aortoenteric fistulas have been reported throughout the length of the gastrointestinal tract. More recently, secondary aortoenteric fistulas have been reported following EVAR. The true incidence of aortoenteric fistula complicating EVAR is unknown but is assumed to be lower than the incidence in patients who have undergone open surgery of the aortoiliac region, because endografts have neither the suture line of an aortic anastomosis nor any other graft material in direct contact with the digestive tract. It has been

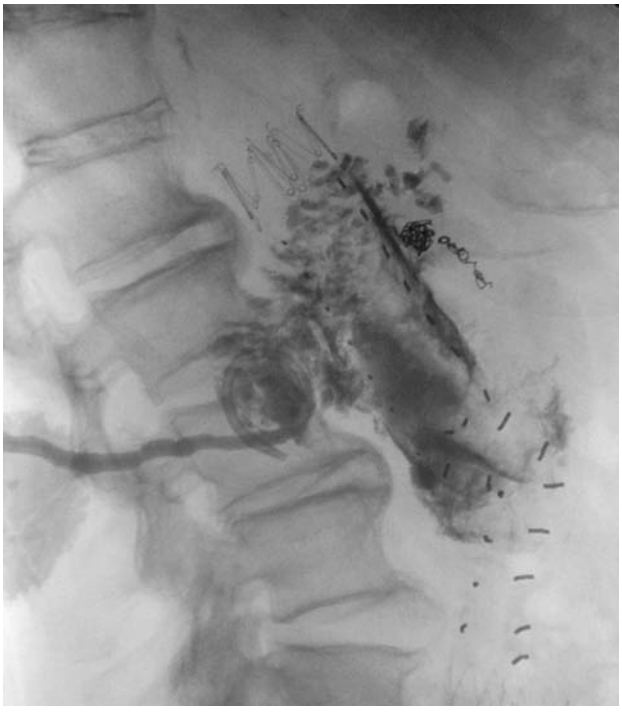


Figure 38-9. Infected endograft with a large perigraft collection that was drained percutaneously.

postulated that aortoenteric fistulas complicating EVAR are related to erosion of the aneurysm wall, migration, or endoleak. However, there have been several cases of aortoenteric fistula post-EVAR in patients without any evidence of endoleak or migration. An additional possible cause or contributory factor to the development of aortoenteric fistula in post-EVAR patients is infection whereby the presence of inflammatory periaortic tissue may contribute to adhesion of the duodenum to the aortic wall and subsequent fistula development. This may result because either the initial aneurysm was mycotic in origin or a low-virulent microbial infection of the endograft subsequently developed. Endotension has also been suggested as a possible source for erosion of the bowel by an aneurysm following EVAR as a consequence of mechanical pressure exerted on the digestive tract by the aorta.

Despite technological advances, the cornerstone of diagnosing an aortoenteric fistula continues to be clinical suspicion. Initial bleeding is usually minor and is often self-limited, and this may lead to a delay in definitive diagnosis. Additionally, the time from the "herald bleed" to massive rebleeding may be hours to months. CT scanning has been advocated as the preferred initial diagnostic test. Findings suspicious for aortoenteric fistula include the presence of periaortic gas more than 2 weeks after open surgery, periaortic inflammatory tissue or fluid, loss of fat between the aorta and the bowel, a defect in the aortic wall, pseudoaneurysm, and extravasation of intravenous contrast medium into the bowel lumen. Esophagogastroduodenoscopy (EGD) has less sensitivity than CT scanning but should be performed in patients with equivocal CT findings. Angiography should be reserved for patients in whom the diagnosis of aortoenteric fistula is unclear to help determine a source of bleeding.

For stable patients with minimal comorbidities and significant life expectancies, surgical management of aortoenteric fistula following EVAR via staged extra-anatomic bypass followed by graft excision is optimal because it provides definitive management, limits lower extremity ischemia, and allows for patient recovery between operations. Simultaneous repair of extra-anatomic bypass followed immediately by graft excision is an acceptable alternative that has also been shown to be feasible, with acceptable morbidity and mortality. Additional surgical options include graft excision alone, graft excision with in situ replacement, and primary repair, all of which should be reserved for select patients. Management of the intestinal portion of fistulas has been demonstrated to have acceptable outcomes with simple bowel repair, although resection may be necessary for certain patients.

Rupture

The primary goal of AAA treatment, whether performed via conventional open repair or EVAR, is the prevention of rupture and subsequent death. Unfortunately, despite its overall excellent effectiveness, rupture rates following EVAR are reported in up to 1.2% per patient per year, depending on the type of endograft and degree of follow-up.[25] Given the increasing survival times of this patient population, AAA rupture is a small but significant risk after EVAR and a major limiting factor in the prognosis of patients over time.

Multiple risk factors for rupture following EVAR have been identified. The primary risk factors appear to be major endoleaks, migration, and the type of endograft. Although proximal type I and type III endoleaks have been reported as the greatest risk factors for aneurysm enlargement and rupture, type II endoleaks have been occasionally identified in select studies as a risk factor for rupture as well, particularly persistent type II endoleaks with sac enlargement. Other factors that contribute to rupture

include larger initial aneurysm size, poor sealing zones, female gender, stent-graft infection, and the presence of aortoenteric fistula.

Ruptures following EVAR have been reported at all time periods, from the immediate perioperative period (<30 days) up to 5 years following initial implantation, emphasizing the need for strict follow-up of patients. Patients who develop rupture after EVAR typically present with the same signs and symptoms as patients with rupture of native AAAs, including abdominal and back pain along with associated hypotension. However, patients with rupture after EVAR are less likely to be hemodynamically unstable, because there may be some protective effect by the endograft (Figure 38–10).

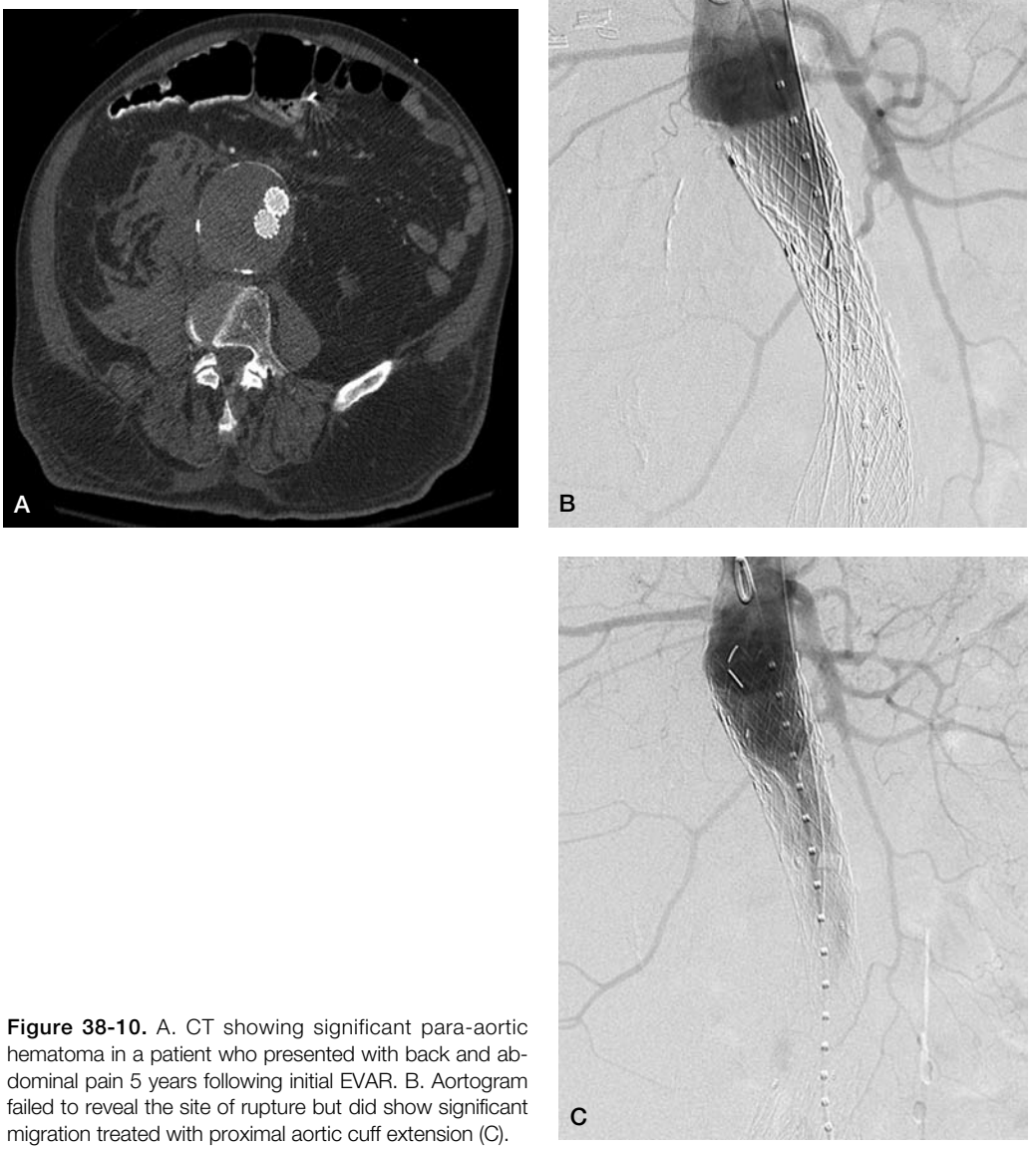


Figure 38-10. A. CT showing significant para-aortic hematoma in a patient who presented with back and abdominal pain 5 years following initial EVAR. B. Aortogram failed to reveal the site of rupture but did show significant migration treated with proximal aortic cuff extension (C).

Treatment of ruptured AAAs following EVAR is associated with relatively high morbidity and mortality rates, although these have been reported to be lower than repair following primary rupture. Endovascular means can easily be employed to correct the defect that led to the rupture, but on occasion, an open conversion is the only remaining option. Both retroperitoneal and transabdominal approaches have been reported.[26] In removing an endograft, technical challenges may arise related to incorporation of stents from endografts into the vessel wall, the presence of external stents or barbs, and periaortic inflammation. Depending on the location of the rupture, complete endograft removal may not be needed, and it may be necessary to transect an endograft. Despite early concerns regarding the use of a transected endograft as a part of the anastomosis and the development of pseudoaneurysms over time, there have been no reported anastomotic complications using this technique.

Data on outcomes for patients who develop rupture after EVAR are limited, although the presence of an endograft appears to be somewhat protective when compared with rupture in a native AAA.[27] However, as with patients who present with rupture of a native AAA, it should be expected that for patients with rupture following EVAR, those who have hemodynamic instability, delays in treatment, and greater comorbidities will have worse outcomes.

CONCLUSION

Despite very encouraging early results with EVAR, the technique continues to be plagued by long-term failures. The incidence of these complications may be decreasing with improving stent-graft design as well as operator experience in selecting appropriate patients and performing the procedure. Nonetheless, the persistent and unpredictable nature of these failures dictate that vigilant follow-up be maintained to avoid potentially fatal outcomes.

REFERENCES

1. Greenhalgh RM, Brown LC, Kwong GP, et al. Comparison of endovascular aneurysm repair with open repair in patients with abdominal aortic aneurysm (EVAR trial 1), 30-day operative mortality results: randomised controlled trial. Lancet 2004;364:843–8.
2. Prinssen M, Verhoeven E, Buth J, et al. A randomized trial comparing conventional and endovascular repair of abdominal aortic aneurysms. N Engl J Med 2004;351:1607–18.
3. Blankensteijn, JD, de Jong SE, Prinssen M, et al. Dutch Randomized Endovascular Aneurysm Management (DREAM) Trial Group. Two-year outcomes after conventional or endovascular repair of abdominal aortic aneurysms. N Engl J Med 2005;352: 2398–405.
4. Leurs LJ, Buth J, Laheij RJ. Long-term results of endovascular abdominal aortic aneurysm treatment with the first generation of commercially available stent grafts. Arch Surg 2007;142:33–41.
5. White GH, Yu W, May J, et al. Endoleak as a complication of endoluminal grafting of abdominal aortic aneurysms: classification, incidence, diagnosis, and management. J Endovasc Surg 1997;4:152–68.
6. Gilling-Smith G, Brennan J, Harris P, et al. Endotension after endovascular aneurysm repair: definition, classification, and strategies for surveillance and intervention. J Endovasc Surg 1999;5:305–7.
7. Veith FJ, Baum RA, Ohki T, et al. Nature and significance of endoleaks and endotension: summary of opinions expressed at an international conference. J Vasc Surg 2002;35:1029–35.

8. Faries PL, Cadot H, Agarwal G, et al. Management of endoleak after endovascular aneurysm repair: cuffs, coils, and conversion. J Vasc Surg 2003;37:1155–61.
9. Rhee SJ, Ohki T, Veith FJ, et al. Current status of management of type II endoleaks after endovascular repair of abdominal aortic aneurysms. Ann Vasc Surg 2003;17:335–44.
10. Silverberg D, Baril DT, Ellozy SH, et al. An 8-year experience with type II endoleaks: natural history suggests selective intervention is a safe approach. J Vasc Surg 2006;44:453–9.
11. Hobo R, Kievit J, Leurs LJ, et al. Influence of severe infrarenal aortic neck angulation on complications at the proximal neck following endovascular AAA repair: a EUROSTAR study. J Endovasc Ther 2007;14:1–11.
12. Sampaio SM, Panneton JM, Mozes G, et al. AneuRx device migration: incidence, risk factors, and consequences. Ann Vasc Surg 2005;19:178–85.
13. Benharash P, Lee JT, Abilez OJ, et al. Iliac fixation inhibits migration of both suprarenal and infrarenal aortic endografts. J Vasc Surg 2007;45:250–7.
14. Tonnessen BH, Sternbergh WC 3rd, Money SR. Mid- and long-term device migration after endovascular abdominal aortic aneurysm repair: a comparison of AneuRx and Zenith endografts.J Vasc Surg 2005 Sep;42:392–400.
15. Sternbergh WC III, Money SR, Greenberg RK et al. Influence of endograft oversizing on device migration, endoleak, aneurysm shrinkage, and aortic neck dilation: results from the Zenith Multicenter Trial. J Vasc Surg 2004;39:20–6.
16. Kibbe MR, Matsumura JS, Excluder Investigators, The Gore Excluder US multi-center trial: analysis of adverse events at 2 years. Semin Vasc Surg 2003;16:144–50.
17. Dillavou ED, Muluk S, Makaroun MS. Is neck dilatation after endovascular aneurysm repair graft dependent? Results of 4 US Phase II trials. Vasc Endovasc Surg 2005;39:47–54.
18. Cao P, Verzini F, Parlani G. et al. Predictive factors and clinical consequences of proximal aortic neck dilatation in 230 patients undergoing abdominal aorta aneurysm repair with self-expandable stent-grafts. J Vasc Surg 2003;37:1200–05.
19. Woody JD, Makaroun MS. Endovascular graft limb occlusion. Semin Vasc Surg 2004; 17:262–7.
20. Carroccio A, Faries PL, Morrissey NJ, et al. Predicting iliac limb occlusions after bifurcated aortic stent grafting: anatomic and device-related causes. J Vasc Surg 2002;36:679–84.
21. Jacobs TS, Won J, Gravereaux EC, et al. Mechanical failure of prosthetic human implants: a 10-year experience with aortic stent graft devices. J Vasc Surg 2003;37:16–26.
22. Cho JS, Dillavou ED, Rhee RY, Makaroun MS. Late abdominal aortic aneurysm enlargement after endovascular repair with the Excluder device. J Vasc Surg 2004;39:1236–41.
23. Haider SE, Najjar SF, Cho JS, et al. Sac behavior after aneurysm treatment with the Gore Excluder low-permeability aortic endoprosthesis: 12–month comparison to the original Excluder device. J Vasc Surg 2006;44:694–700.
24. Sharif MA, Lee B, Lau LL, et al. Prosthetic stent graft infection after endovascular abdominal aortic aneurysm repair. J Vasc Surg 2007;46:442–8.
25. Ducasse E, Calisti A, Speziale F, et al. Aortoiliac stent graft infection: current problems and management. Ann Vasc Surg 2004;18:521–6.
26. del Moral L, Alonsoa SF, Kiuri SS, et al. Aortoenteric fistula arising as a complication of endovascular treatment of abdominal aortic aneurysm. Ann Vasc Surg 2009;23:255.e13–7.
27. Schlösser FJ, Gusberg RJ, Dardik A, et al. Aneurysm rupture after EVAR: can the ultimate failure be predicted? Eur J Vasc Endovasc Surg 2009;37:15–22.
28. Kelso RL, Lyden SP, Butler B, et al. Late conversion of aortic stent grafts. J Vasc Surg 2009; 49:589–95.
29. Coppi G, Gennai S, Saitta G, et al. Treatment of ruptured abdominal aortic aneurysm after endovascular abdominal aortic repair: a comparison with patients without prior treatment. J Vasc Surg 2009;49:582–8.

SECTION V

Tevar

39

Update on Thoracic Aortic Endograft

Andy C. Chiou, M.D., M.P.H., Kristen L. Biggs, M.D., and Jon S. Matsumura, M.D.

The pathology of the thoracic aorta is quite variable and includes degenerative aneurysm, acute and chronic dissection, traumatic injury, fistula, embolizing lesions, and coarctation. Within each of these general categories such as dissection, there are subgroups such as penetrating ulcer and intramural hematoma. This chapter will begin with a background on the common thoracic aortic problems, but focus on updates on thoracic aortic endografts. It will include descriptions of the devices in United States (U.S.) trials, general anatomic requirements, technical aspects of the procedure, results and complications with endovascular treatment, and review a compilation of experience of eight world experts (Table 39–1).

Aortic Dissection

There are an estimated 10–20 cases per million annually of aortic dissection.[1] Acute aortic dissection is the most common, lethal catastrophe involving the thoracic aorta.[2] One likely reason for this high mortality may be the difficulty in obtaining an early diagnosis. During the first 48 hours, mortality rates with untreated acute ascending and descending aortic dissection can exceed 1% per hour.[3] Recent attention in the lay press has led to increased public awareness of this disease and should encourage development of new treatments.

Several classification systems exist to describe the location and extent of the dissection, and one widely used system is the DeBakey classification (Figure 39–1). The cardinal feature of all classification systems is the presence or absence of involvement of the ascending aorta. Approximately two-thirds of dissections involve the ascending aorta and the primary tear is usually located in this region.[4,5] This is important because with rare exceptions, most dissections and other thoracic aortic pathology that have had endovascular treatment do not involve the ascending aorta.

TABLE 39-1. SELECTED PHYSICIANS AND INSTITUTIONS BY REPORTED TOTAL THORACIC ENDOGRAFT VOLUME

Physician	Location
Shin Ishimaru, M.D.	Tokyo Medical University
Michael Dake, M.D.	Stanford University Medical School
Roy Greenberg, M.D.	Cleveland Clinic
Ludger Sunder-Plassman, M.D.	University of Ulm
Rodney White, M.D.	University of California, Los Angeles-Harbor
Geoffrey White, M.D.	University of Sydney
Takao Ohki, M.D.	Montefiore Medical Center
Michael Laurence-Brown, M.D.	Perth University

Degenerative Thoracic Aneurysm

The annual incidence of thoracic aortic aneurysms is approximately six cases per 100,000 population.[6] The five-year survival rate of patients with untreated thoracic aneurysms ranges from 10–50%.[7] For the most part, device design has been focused on treatment of descending aortic aneurysm, which is the most common pathology treated by thoracic stent-grafts (Table 39–2). Some of these patients have survived acute dissection and developed aneurysmal change in the setting of chronic dissection. This distinction is relevant because aneurysms associated with chronic dissection may respond differently to endovascular exclusion if there are numerous untreated reentry points or natural fenestrations that continue to perfuse the aneurysm.

In general, thoracic aortic aneurysms are asymptomatic. When they enlarge, they can produce symptoms by mass effects of surrounding structures or if they rupture. Typically, the patient may complain of pain in the chest, back, upper abdomen, or flank. Other symptoms include hoarseness, voice changes, stridor, hemoptysis, and shortness of breath from stretch of the recurrent laryngeal nerve or left bronchial compression. In contrast to conventional endoaneurysmorrhaphy, endovascular treatment does not immediately lead to decompression of the aneurysm sac, and symptoms may persist after the endograft is placed.

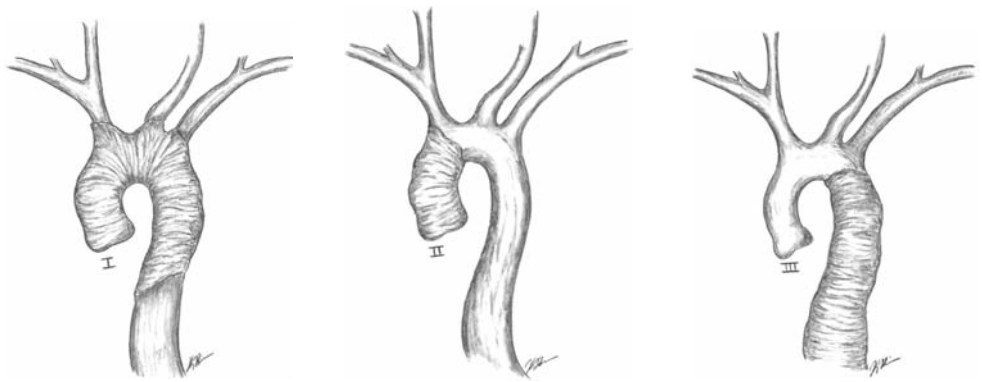


Figure 39-1. DeBakey classification of aortic dissections: Thickened regions designate involvement by dissection.

TABLE 39-2. VARIOUS PATHOLOGY TREATED BY ENDOGRAFTS AND PERIPROCEDURAL MORTALITY.

Primary aortic pathology	Percent of total cases	30-day Mortality (%)
Degenerative Aneurysm		
Descending thoracic aneurysm	64.2	4.1
Thoracoabdominal aneurysm	1.6	5.0
Posttraumatic		
Acute traumatic disruption	10.0	5.5*
Pseudoaneurysm	3.3	2.7
Dissection		
Acute dissection	7.8	9.9
Intramural hematoma with ulcer	2.2	7.2
Giant penetrating ulcer	1.0	0
Chronic dissection	8.4	3.3
Miscellaneous		
Aortic fistula	0.9	2.6
Embolizing lesion	0.3	0
Stenosis/coarctation	0.1	0

*Often due to concomitant neurologic injury

Diagnostic Imaging

Thoracic aneurysms are usually found incidentally on radiographic studies, and most are small at the time of initial diagnosis. Smaller aneurysms have their size monitored with periodic surveillance, although wall stress is being studied as a predictor of rupture risk. When they become larger or if symptoms supervene, imaging objectives become focused on interventional planning. A contrast-enhanced, thin collimation, computed tomography scan of the chest is the primary imaging modality for thoracic aortic lesions. Multiple view digital subtraction angiography with a marker catheter provides complimentary measurement data in regard to lengths, and is routinely used to further evaluate the extent of thoracic aortic and branch vessel lesions in planning open and endovascular intervention. Similar to infrarenal endorepair, anatomic measurements and device selection are critical components to optimize outcomes. Other imaging modalities include magnetic resonance angiography and intravascular ultrasound, which may be particularly useful when assessing the dynamic anatomy of patients with acute dissection. Transesophageal echocardiography with color-flow mapping has also been used to evaluate thoracic disease intraoperatively.[8,9]

Treatment

The objectives of surgical repair of descending thoracic aortic aneurysm are to relieve compressive symptoms and prevent rupture of the aorta. Several procedures have been utilized; the most frequent is endoaneurysmorrhaphy with the graft inclusion technique. Open surgical repair usually requires cross-clamping of the aorta and can lead to ischemic (hemodynamic and thromboembolic) complications that are manifest in end organ failure as myocardial infarction, stroke, paraplegia, and renal insufficiency. Adjunctive and alternative techniques to reduce aortic clamping or improve distal

perfusion are repair with total circulatory arrest, partial left heart bypass and sequential clamplng, side arm distal perfusion, and partial aortic clamping with multiple bypass grafts. All of these still require a thoracotomy and dissection, and are associated with bleeding, pulmonary, and wound healing problems. In one large series involving 832 patients undergoing open operations using left heart bypass, complication rates were 7% for renal failure, 3% for stroke, 5% for paraplegia, 10% for cardiac complications, and 28% for respiratory complications.[10] Mortality rates for open thoracic aortic aneurysm repair range from 5–28%.[11] Mortality is clearly higher with emergent repair, up to 29.9% for ruptured descending thoracic aneurysms and 57.1% with ruptured thoracoabdominal aneurysms.[12] Patients with rupture and similar pathology manifest by acute major hemorrhage may be the cohort that benefits most from the option of endovascular repair.

Endovascular Repair of Thoracic Aortic Aneurysms

Several studies suggest that by avoiding thoracotomy, cardiopulmonary bypass, and aortic cross-clamping, endovascular repair results in fewer major complications. The mechanism may be through less operative trauma and less ischemic injury to the kidneys, spinal cord, and abdominal viscera.[11] Other benefits compared to traditional open surgical repair include reductions in procedure time (mean = 56 minutes, range: 37-215 minutes), blood loss, blood transfusion, post-operative pain, intensive care utilization, overall hospital stay, and rapid return to baseline level of activity.[12, 13]

However, benefits of minimally invasive surgery are easily overestimated because even though the procedure is less traumatic, the underlying disease state is still associated with severe comorbidities and complications. Further, sicker patients may undergo endorepair because it is perceived as safer. In one recent series of 59 endovascular repairs of the thoracic aorta, there was a 15% perioperative mortality and there were 20% perioperative cardiac events. Of note, the cardiac event rate was seen to be 29% for patients who did not receive perioperative beta blockade versus 8% in patients who did receive beta-blockade. The conclusion was that the mortality associated with endovascular repair of the thoracic aorta remains significant in contrast to some earlier reports.[14]

Initial stent grafts for thoracic endovascular repair were bulky handmade devices that were difficult to accurately deploy and required large, stiff introducer systems that limited their applicability. Nonetheless, 73% successful repair of thoracic aortic aneurysms was reported with these devices in 1994 with a reintervention rate of 20% and an early mortality rate of 9%.[15] Newer generation stent grafts have improved maneuverability, flexibility, and the have smaller introducer systems.[11] No thoracic endografts are currently approved by the U.S. Food and Drug Administration (FDA), although several systems are under Investigational Device Exemption study and others are used off-label.

Current patient criteria for endovascular devices are variable. Most devices require iliofemoral access of greater than 8 mm to accommodate the introducer systems, aortic arch angulation less than 90 degrees with limited tortuosity, and proximal and distal landing zones that are 20–30 mm in length.[1,13] While many patients have had extension of proximal neck length across arch branches with ligation or extraanatomic bypass, one report concluded that these techniques do not reliably exclude thoracic aortic pathology when the proximal landing zone is proximal to the left common carotid artery.[16] Thus, there are several relative contraindications for endovascular

stenting, which include the inability to obtain vascular access, thoracic aortic tortuosity, and poor quality, or very short landing zones either proximally or distally.[12,17]

Induced hypotension, cardiac asystole, and transvenous balloon occlusion of the right atrium to create partial inflow occlusion have been used to subdue the strong flow dynamics of the proximal thoracic aorta to reduce migration during device deployment.[18] Most physicians now rarely use these techniques with newer generation devices and improved deployment systems. One group reported that only three of 67 patients required systemic hypotension during stent deployment.[11] Another group completed 73 out of 74 of their thoracic stent grafts under general anesthetic without significantly lowering any heart rates of systolic blood pressures during stent deployment.[12]

PROBLEMS AND COMPLICATIONS

Complications resulting from thoracic endograft placement include complications common to infrarenal aortic endovascular repair, plus increased concern for specific problems of 1) paraplegia, 2) stroke, 3) ischemia from occlusion of aortic branch vessels, 4) dissection or rupture of access vessel, and 5) device failure, fracture, or migration.

Neurologic Injury

Endovascular exclusion of the thoracic aorta, including coverage of the intercostal arteries, may lead to transient neurologic deficits or permanent paraplegia. Cerebrospinal fluid (CSF) drainage is often used as a protective mechanism during open surgical repair because CSF pressure increases during aortic cross-clamping, and reduction of CSF pressure improves spinal perfusion pressure. A randomized clinical trial showed benefit of drainage to a pressure threshold during open repair with an 80% reduction in the relative risk of postoperative neurologic deficits.[19] However, CSF drainage can result in complications and its role in patients treated with endoluminal thoracic stent grafts is unknown. One large series of 103 patients with thoracic aortic aneurysms treated with stent grafts reported only three cases (2.9%) of paraplegia.[20] Recent studies have also found a similarly low incidence of paraplegia.[1,8,11,12] Fattori et al. had no cases of paraplegia arise in their group of 70 patients, despite covering the entire descending thoracic aorta in 21 cases.[8]

Stroke is one complication that may be more frequent in endovascular repair compared to open repair. Coexistent arch/carotid disease, manipulation of stiff wires and long leading parts of deployment systems, coverage of arch vessels, and air embolism are potential etiologies of stroke. Better understanding and recognition of these issues should result in modified device design and techniques to reduce stroke rates.

Endoleak

As experience grows with thoracic endografts, the rate of endoleaks seems to be lower than with infrarenal endorepair. Endoleak rate may be further reduced by increasing landing zone distance to 20 mm from 10 mm.[12] In general, most groups have reported endoleak rates of 7–10%.[1,8,11] One study in human cadaveric aortas found that an additional stent placed at the proximal attachment site greatly improved endograft fixation and could possibly reduce type I endoleak.[21]

Dissection and Arterial Perforation

One major problem with thoracic devices is the larger and often stiff introducer required for their deployment. Iliac artery dissection, perforation, and even avulsion can occur and lead to fatal hemorrhage. More liberal use of conduits, a high index of suspicion, and rapid treatment may reduce the mortality of these events. Another unsuspected late problem with thoracic endovascular repair is the development of aortic dissection or frank perforation of the aorta at the proximal end of the endograft. This has been a catastrophic late complication, but fortunately it appears to be uncommon. Although some investigators suspect this late proximal arterial injury is more frequent with some device features, direct rigorous comparison of rates has not been performed.

Device Failure

The second generation of thoracic stent grafts, although an improvement from earlier "home made" devices, still have had shortcomings. Stent fracture related to the considerably higher thoracic aortic fatigue forces, migration due to poor proximal, intercomponent, and distal fixation, and mechanical device failures such as failure to deploy are some of the problems that have occurred.

In one series of 84 patients receiving thoracic stent grafts under U.S. FDA-approved clinical trials, 11 patients (13%) were found to have stent fracture at a mean of 20 months of follow-up.[17] Nine of these patients did not have an associated endoleak and were being followed with CT scans.

Mortality

In initial reports, mortality rates for endovascular repair of thoracic aortic disease are similar or less than with open repair. The 30-day mortality for electively placed endografts in one study was as low as 2% (one of 42 patients).[11] In this series, the one cause of death was mesenteric ischemia from embolization via the celiac axis. The 30-day mortality for urgent or emergently placed thoracic endografts in the same study was 16% (four of 25 patients). The causes of death were perioperative myocardial infarction in two patients, on-table aortic rupture in one patient, and rupture of an unrecognized false aneurysm of the distal thoracic aorta in one patient. Other authors report mortality rates between six and 10%.[12, 17]

Device Durability

The long-term device durability rate of thoracic endografts is unknown. As described above, reports are surfacing regarding stent fracture and material fatigue in the second generation of stent grafts. Most studies have relatively short reported patient follow-up from one to 72 months with a mean of about two years at current writing.[1, 11, 12]

Thoracic Aortic Zones of the Proximal Neck

Five different anatomic zones of the aortic arch and descending thoracic aorta have been described that are helpful to consider when evalutating patients for thoracic endorepair (Figure 39–2). Thoracic aortic disease that occurs within specific zones may

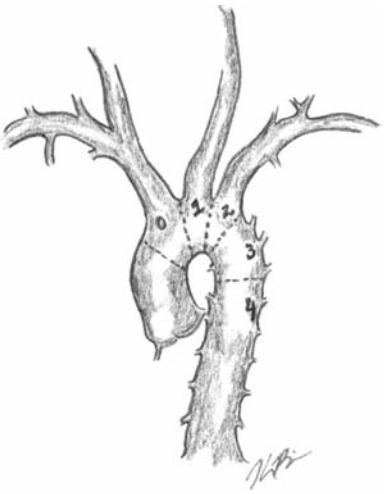


Figure 39-2. Thoracic aortic proximal landing zones.

require specific adjunctive procedures to obtain adequate proximal landing areas for sealing and fixation. Zone 0 refers to the ascending aorta, which includes the innominate artery. Zone 1 involves the portion of the aortic arch that provides the takeoff for the left common carotid artery. Zone 2 involves the portion of the aortic arch that includes the left subclavian artery origin. Zone 3 is located distal to the left subclavian artery, within the curved portion of the arch. Zone 4 disease involves the straight portion of the descending thoracic aorta.

The extent of proximal disease correlates with the complexity of the endovascular solution. In cases only involving zone 4, or the straight portion of the descending thoracic aorta, additional surgical procedures are usually not necessary. If disease involves zone 3, an adequate landing zone for proximal fixation of the device may approach or cover the origin to the left subclavian artery. Zone 2 disease, by definition, requires subclavian coverage. Although at times sufficient collateralization may obviate the need for surgery, patients with symptoms of left upper extremity ischemia will need an adjunctive procedure that may be done before or after endovascular coverage. Restoration of blood flow to the left subclavian artery can be achieved by a kissing stent, left subclavian transposition, or a left carotid-subclavian bypass. Thoracic endograft placement for zone 1 and 0 disease will require coverage of the left common carotid and innominate arteries, respectively. Surgical alternatives for restoring blood flow can involve a variety of creative solutions.

ONGOING TRIALS

There are at least three thoracic stent grafts under trial in the U.S.

The Talent (Medtronic AVE, Santa Rosa, California) thoracic stent graft is a nitinol/polyester endograft (Figure 39–3). This device has been approved for more than five years in Europe and has been implanted in more than 6,000 patients worldwide. The VALOR (Evaluation of the Safety and Efficacy of the Medtronic Vascular Talent Thoracic Stent Graft System for the Treatment of Thoracic Aortic Aneurysms) study,

Figure 39-3. Medtronic Talent endograft. There are bare wire flares on the ends.

began enrolling patients in the United States in November 2003. This trial is being conducted at 35 sites and involves 500 patients.

The Zenith TX2 (Cook Incorporated, Bloomington, Indiana) thoracic endovascular graft is also approved for commercial distribution outside the U.S. (Figure 39–4). This trial compares the outcomes of patients treated with standard operative approach versus endovascular therapy with the Zenith TX2 endograft. The first of these grafts was placed in the trial in March 2004, and the study will enroll up to 220 patients at up to 35 medical institutions.

The Gore Thoracic Aortic Graft (TAG) endoprosthesis (W. L. Gore & Associates, Flagstaff, Arizona) is made of a composite of expanded polytetrafluoroethylene (ePTFE) graft and a self-expanding nitinol support structure (Figure 39–5). It has recently been relaunched in Europe. Currently, the TAG has completed enrollment in its phase II FDA approved trial and is pending late evaluation of data and results. It may be the first device that is commercially available in the U.S.[13]

Figure 39-4. Zenith TX2 endovascular graft. A two-piece graft is depicted with large barbs on the ends. The caudal end has a bare stent.

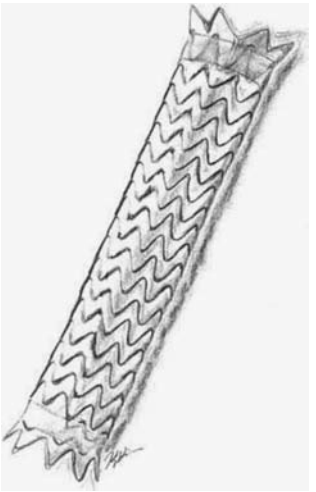


Figure 39-5. Gore TAG endoprosthesis. The latest version has covered flares on the ends and no longitudinal spines.

Worldwide Clinical Review

Peer-reviewed scientific publications are the gold standard of evidence for medical decision-making. However, in rapidly emerging technologies, early experiences are dominated by prototype devices, single site small experiences, and initial learning periods. Data from US clinical trials are useful because of the well-defined protocol, regulatory oversight, and generally more diligent follow-up and corelab review. However, practical clinical use often does not match the rigid entry criteria of these studies, and industry-sponsored trials focus on the sponsor's contemporary device version. In order to get a grasp of the general clinical practice in thoracic endografts, a survey was conducted during personal visits with eight physicians from leading thoracic endovascular centers of excellence around the world. Table 39–1 lists the primary investigator who was interviewed and their respective medical center. This pooled data of 1,180 patients provides reflections on a large series of patients from the perspectives of experts in the field. Weaknesses of this compilation are the lack of uniformity in surveillance protocols, definitions, follow-up intervals, compliance, and overlap with existing publications.

Table 39–2 summarizes the types of thoracic pathology that has been treated and the 30-day mortality in each of these categories. It is the majority impression that endovascular treatment is an equivalent or better therapy compared to conventional treatment in each of these categories except for thoracoabdominal aneurysm, chronic dissection, and stenosis where there was disagreement or it was felt to be unknown. This is conditional on the patient having suitable anatomy for endorepair. The majority of patients (64.2%) has been treated for degenerative descending thoracic aortic aneurysms. A wide variety of endografts have been used for treatment.(Table 39–3.)

Most patients (70.3%) have been treated in an operating room environment and 49.2% used fixed unit fluoroscopy units. Intravascular ultrasound was used in 12% and transesophageal echocardiography in 38.1%. Primary access was in the femoral artery in 84.4% (percutaneous in 1.1%), iliac in 7.5%, conduit or aortofemoral graft limb in 6.5%, infrarenal aorta in 1.1%, and other sites in 0.4%.

TABLE 39-3. DISTRIBUTION OF ENDOVASCULAR GRAFTS IN THIS COMPILATION

Endograft	Frequency
Homemade	53.6
TAG	19.0
Zenith	11.5
Talent	10.8
AneuRx	2.9
Infrarenal cuffs	1.9
Vanguard	0.2
Corvita/wallstent	0.1
Stenford	0.1

The left subclavian artery was covered in 29.8%, and the left common carotid in 8.0%, the innominate in 3.7%, celiac artery in 3.1%, and SMA in 0.8%. The option to revascularize these major branches is exercised at different rates and seems to be undergoing evolution. Most centers revascularize the carotid, innominate, and visceral arteries when they are occasionally covered. In contrast, revascularization is performed before or concurrently with endorepair in only 36.7% of patients who have coverage of the left subclavian artery. This is an area of increasing consensus; that is, fewer patients are having routine subclavian revascularization except when there is diseased or hypoplastic right vertebral arteries or fistulas/reconstructions based on left subclavian branches. The majority of patients is managed expectantly, and in the infrequent scenario when symptoms develop after left subclavian coverage, delayed revascularization can be performed.

Spinal cord drainage was used routinely in 1.0% and selectively in 6.8% of patients with extensive descending aortic coverage or history of AAA. Adenosine (2.1%) and high-dose beta-blocker (0.1%) were used rarely to arrest or slow the heart for deployment. The 30-day morbidity rates are stroke in 2.8%, renal failure in 1.6%, and paraplegia in 2.5%. Forty-three percent of the paraplegia complications were delayed in onset.

Later outcomes are best described by time-dependent estimates, but are not precisely combinable because of unequal or unknown individual lengths of follow up. Nevertheless, general estimates are useful to gauge the emerging technology. Endoleak was noted in 10.5% of patients, sac expansion in 4.0%, proximal neck dilation or dissection in 2.7%, distal neck dilation in 2.1%, intercomponent migration in 1.7%, proximal migration in 1.3%, distal migration in 0.4%, asymptomatic device failure such as fracture in 6.3%, symptomatic device failure in 0.1%, and aneurysm rupture in 0.9%.

Recommendations by the experts on future device design were ranked, and the top desired attributes are 1) more flexible delivery systems, 2) more flexible devices with better conformation to irregular wall contour, and 3) branched devices.

CONCLUSION

The treatment of thoracic aortic disease is rapidly evolving. Alternatives to the traditional, open surgical approach are being developed and evaluated in clinical trials.

While mortality rates for open surgical repair of the thoracic aorta have decreased with experience, early reports of thoracic aortic endografts suggest that endovascular repair may offer a less invasive option with lower morbidity and mortality rates. Midterm data from several series are remarkable for promising overall results, but also some rare but striking late failures. Currently, it is ideal for standard risk patients with suitable anatomy to be enrolled in controlled, clinical trials. Some patients with extremely high operative risks may be considered for endovascular repair because there is no suitable alternative.

Several engineering advancements are being pursued to address shortcomings in earlier devices for thoracic endorepair. These include endografts that conform to the individual aortic anatomy, more flexible and accurate delivery systems, more robust construction to accommodate higher thoracic aortic forces, and mechanisms to treat pathology close to or involving the aortic arch and visceral sidebranches. As these technologies mature, it is possible that endovascular therapy may become the preferred initial therapy for many thoracic aortic diseases.

REFERENCES

1. Criado, FJ, Clark NS, Barnatan MF. Stent graft repair in the aortic arch and descending thoracic aorta: a 4-year experience. *J Vasc Surg*. 2002;36:1121–1128.
2. Bickerstaff LK, Pairolero PC, Hollier LH et al. Thoracic Aortic Aneurysms: A Population-Based Study. *Surgery*. 1982;92:1103.
3. Miller DC, Mitchell RS, Oyer PE, et al. Independent Determinant of Operative Mortality for Patients with Aortic Dissections. *Circulation*. 1984;70 (Supp. 1):1–153.
4. Gore I, Siewert VJ. Dissection Aneurysm of the Aorta. Pathological Aspect and Analysis of 85 Fatal Cases. *Arch Pathol*. 1952;53:121.
5. Hearst AE Jr, Johns VJ Jr, Kime SW Jr. Dissecting Aneurysm of the Aorta. A Review of 505 Cases. *Medicine*. 1958;37:217.
6. Sorenson HR, Olson H. Ruptured and Dissecting Aneurysms of the Aorta. Incidence and Prospects of Surgery. *ACTA Chir Scand*. 1964;128:644.
7. Pate JW, Richardson RL, Eastridge CE. Acute Aortic Dissections. *Am Surg*. 1976;42:395.
8. Fattori R, Napoli G, Lovato L, et al. Descending Thoracic Aortic Diseases: Stent Graft Repair. *Radiology*. 2003;229:176–183.
9. Criado E, Wall P, Lucas P, et al. Transesophageal Echo Guided Endovascular Exclusion of Thoracic Aortic Mobile Thrombi. *J Vasc Surg*. 2004;39:238–242.
10. Svensson LG, Crawford ES, Hess KR, et al. Variables Predictive of Outcome in 832 Patients Undergoing Repairs of the Descending Thoracic Aorta. *Chest*. 1993;104:1248–1253.
11. Bell RE, Taylor PR, Aukett M, et al. Midterm Results for Second Generation Thoracic Stent Grafts. *Br J Surg*. 2003;90:811–817.
12. Orend KH, Scharrer-Pamler R, Kapfer X, et al. Endovascular Treatment in Diseases of the Descending Thoracic Aorta: Six Year Results of a Single Center. *J Vasc Surg*. 2003;37:91–99.
13. Makaroun M, et al. Annual Meeting of the Society for Vascular Surgery Program Book. Anaheim, California, 2004.
14. Bui H, Haukoos J, Donayre C, White R, DeVirgilio C: *Ann Vasc Surg*. 2004;18(1):22–25.
15. Dake MD, Miller DC, Semba CP, et al. Transluminal Placement of Endovascular Stent Grafts for the Treatment of Descending Thoracic Aortic Aneurysms. *N Engl J Med*. 1994;331:1729–1734.
16. Tse LW, MacKenzie KS, Montreuil B, et al. *Ann Vasc Surg*. 2004;18(2):178–85.
17. Ellozy SH, Carroccio A, Minor M, et al. Challenges of Endovascular Tube Graft Repair of Thoracic Aortic Aneurysm: Midterm Follow Up and Lessens Learned. *J Vasc Surg*. 2003;38:676–683.

18. Marty B, Morales CC, Tozzi P, et al. Partial Inflow Occlusion Facilitates Accurate Deployment of Thoracic Aortic Endografts. *J Endovasc Ther*. 2004;11(2):175–179.
19. Coselli JS, LeMaire SA, Koksoy C, et al. Cerebrospinal fluid drainage reduces paraplegia after thoracoabdominal aortic aneurysm repair: Results of a randomized clinical trial. *J Vasc Surg*. 2002;35:631–639.
20. Mitchell RS, Miller DC, Dake MD, et al. Thoracic aortic aneurysm repair with an endovascular stent graft: the "first generation." *Ann Thorac Surg*. 1999;67:1971–1974.
21. Veerapen R, Dorandeu A, Serre I, et al. Improvement in proximal aortic endograft fixation: an experimental study using different stent-grafts in human cadaveric aortas. *J Endovasc Ther*. 2003;10(6):1101–1109.

40

TEVAR following FDA Approval: Results of the TAG 05-02 Post Marketing Study

Mark D. Morasch, M.D., F.A.C.S.

In March 2005, the Gore TAG® Thoracic Endoprosthesis (TAG®) became the first device approved in the United States for use in the primary treatment of descending thoracic aortic aneurysms (DTAA). Since approval, the technology has been dispersed with some uniformity throughout the United States. As part of the approval process, the manufacturer agreed to conduct a postmarketing study of the device used to treat patients with DTAA from multiple geographically disbursed sites and by physicians from three distinct tiers of device training. The purpose of the postapproval study is to evaluate the long-term performance of the TAG® in the primary treatment of DTAA as utilization of the technology has become diffusely disbursed. The study has also been designed to assess the effectiveness of the Gore TAG® Physician Training Program (GTPTP) as designed with regards to new physician training.

TAG 05-02 is a multicenter, nonrandomized observational study. A total of 150 subjects diagnosed with DTAA were enrolled and treated between January 2006 and April 2008. Subjects were enrolled both prospectively and retrospectively from 25 geographically distributed investigational sites. No limit was set on the number of subjects who could be enrolled per site (Figure 40–1). The experience of the implanting physicians varied considerably between and within sites. Implanting physicians were categorized into three tiers depending upon experience level.

Complete pre- and post-treatment data have been collected out to 1 year. One year data lock took place in July of this year. Data will continue to be collected annually for 5 years post-treatment. Patient follow-up, including diagnostic studies and physical examinations, are completed at predetermined time points, with windows for compliance and with the impact of loss to follow-up assessed.

To be enrolled, patients had to have a DTAA that otherwise required surgical repair and aortic anatomy that had to meet Instructions for Use (IFU). Mycotic and uncontained ruptured aneurysms were excluded. Patients were excluded if they had

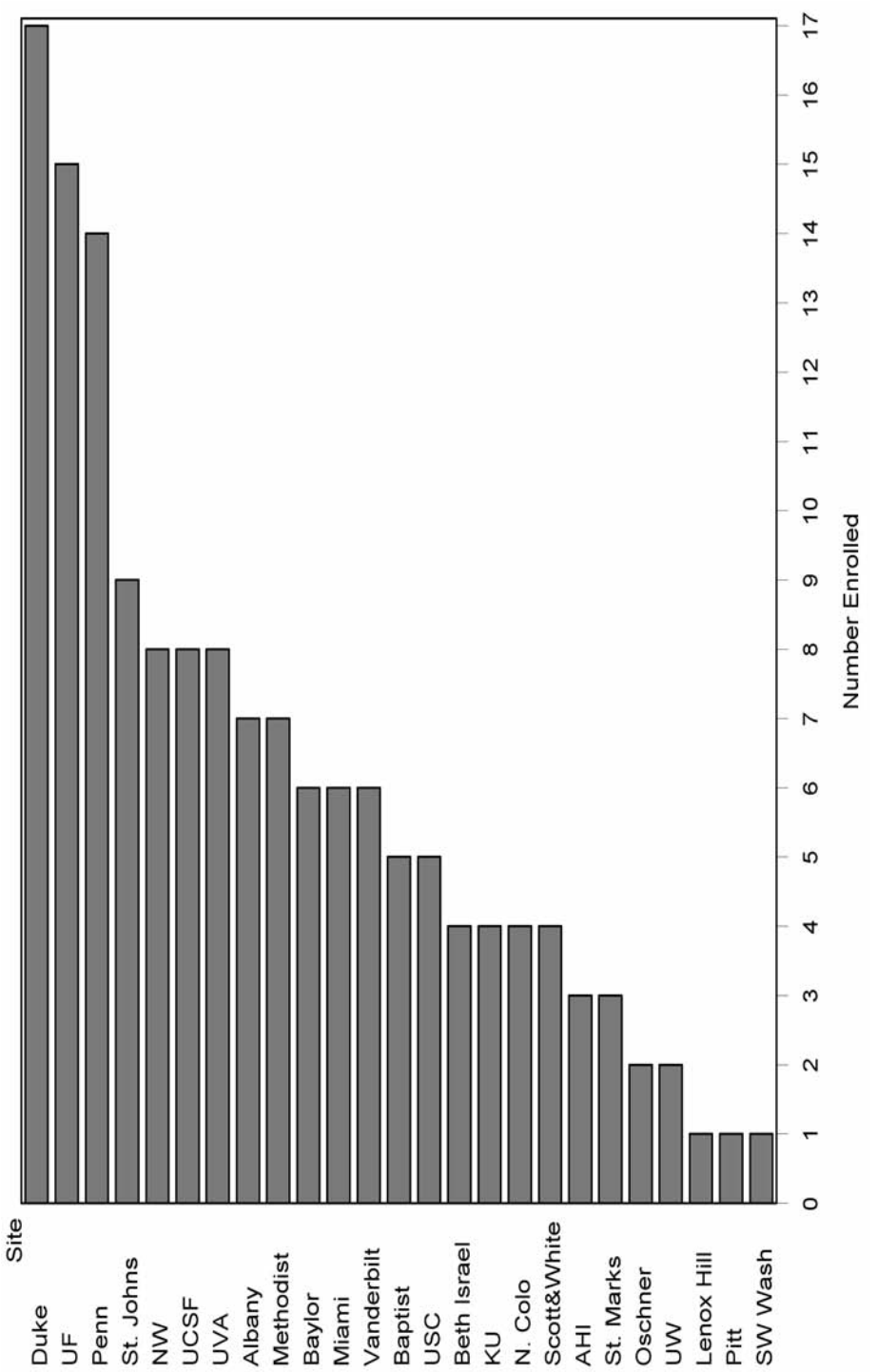


Figure 40-1. Subjects enrolled per site.

evidence of acute or chronic dissection or a degenerative connective tissue disorder. Coverage of major aortic branch vessels, exclusive of the left subclavian artery, would exclude the patient from enrollment. Management of a covered left subclavian artery was left up to the discretion of the implanting surgeon.

Sixty-eight percent of patients implanted were male. Average age at implantation was 74. Eighty percent of patients met strict inclusion/exclusion criteria. Average procedure time was just over 2 hours. Average blood loss was estimated at 200 cc. All 150 patients were successfully accessed and all had a device successfully delivered to the target pathologic area. There were no immediate surgical conversions.

Thirteen patients did not return for the 30-day evaluation. There were 4 deaths (from bowel ischemia, respiratory failure, AAA rupture, cardiac tamponade) for a 30-day all-cause mortality of 2.7%. The major adverse event rate was 21% (including 2 strokes and 6 cases of paraplegia/paresis), and the major device-related event rate was 8.0%. Twelve patients required endoluminal intervention within the window for 30-day follow-up for device-related events. Reinterventions included the placement of additional thoracic devices, coil embolization of leaks, and branch vessel stenting. No conversions were required.

At 1 year, a total of 14 patients had died, and 2 additional patients were lost to follow-up. A total of 14 patients died from the time of implantation to the end of the 1-year window. The 10 deaths occurring between 30 days and 1 year were categorized as the result of cardiac complications (n=3), pulmonary complications (n=3), vascular complications (n=1), neoplasm (n=1), or other/unknown (n=2). There was an additional 25% major adverse event rate. Nearly half that rate was due to cardiac complications unrelated to the aneurysm or to the device. Seven patients required additional thoracic aortic device implantation procedures. At 1 year, the major device-related event rate was 13%, with 10 patients experiencing clinically important endoleaks, 1 patient experiencing device migration, and 1 patient sustaining aneurysm rupture.

The Gore TAG® Thoracic Endoprosthesis can be used successfully to treat DTAA by physicians trained in the GTPTP with limited major adverse events and with few major device related events.

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Combining Open and Endovascular Approaches to Complex Aneurysms

Joseph S. Coselli, M.D., Susan Y. Green, M.P.H., Ourania Preventza, M.D., and Scott A. LeMaire, M.D.

INTRODUCTION

The groundbreaking experience of endovascular repair of the infrarenal portion of the abdominal aorta[1] and the promising contemporary results in the descending thoracic aorta[2] have led to the increased application of endovascular approaches in certain aortic segments—namely, the aortic arch and thoracoabdominal aorta—that traditionally have been repaired solely with open surgical techniques because of the anatomic complexity imposed by the major branch vessels. When considering the three primary options for aortic repair, Greenberg et al.[3] suggest assessing the two major factors that affect each patient's outcome: physiologic reserve and anatomic complexity. Patients with poor physiologic reserve and complex aortic anatomy that precludes purely endovascular repair with simple tube stent grafts are ideally suited for combined or "hybrid" approaches that use open surgical procedures to reroute branch vessel circulation, enabling subsequent placement of the stent-graft to exclude the entire aortic aneurysm (Table 41–1).[4] This chapter describes several combined approaches to the repair of aneurysms of the aortic arch or thoracoabdominal aorta.

HYBRID APPROACH TO AORTIC ARCH REPAIR

Typical Complications of Traditional Open Arch Repair

Conventional surgical repair of the aortic arch is generally performed through a median sternotomy; cardiopulmonary bypass, hypothermic circulatory arrest, and cerebral perfusion are used to protect the brain while normal blood flow through the brachiocephalic

TABLE 41-1. TREATMENT APPROACHES CHOSEN ACCORDING TO PHYSIOLOGIC RESERVE AND ANATOMIC COMPLEXITY

Physiologic Risk	Anatomic Complexity	Approach
Low	Low	Well served by open or endovascular repair
Low	High	Ideal for open repair
High	Low	Ideal for endovascular repair
High	High	Ideal for a combined approach

Adapted from Greenberg RK, Clair D, Srivastava S, et al. Should patients with challenging anatomy be offered endovascular aneurysm repair? J Vasc Surg 2003;38:990–6.

vessels is temporarily halted. However, despite the use of protective adjuncts, arch replacement continues to carry substantial risk. Prolonged cardiopulmonary bypass and hypothermic circulatory arrest are associated with increased mortality, neurologic morbidity, and other complications. Contemporary series have shown excellent results, particularly in highly experienced centers; published early mortality rates range from 0 to 5%, and permanent stroke rates range from 0 to 4%.[5-7] However, patient-specific comorbidities such as advanced age, chronic renal dysfunction, previous cardiac damage, or history of stroke can greatly increase a patient's risk of these adverse outcomes,[8-9] and such patients are often considered inoperable because they are unable to withstand traditional aortic arch repair. In very high-risk patients, a combined approach has been advocated as an effective alternative that produces acceptable mortality rates and fewer neurologic, cardiac, and pulmonary complications.

Debranching to Lengthen the Proximal Landing Zone

The aortic arch has been anatomically mapped by Criado and associates[10] into five zones to facilitate procedure planning as well as to document the location of the proximal landing zone (Figure 41–1). In day-to-day practice, endovascular repair of the descending thoracic aorta frequently incorporates the more distal aspects of the aortic arch (Zone 3); however, hybrid arch repair necessitates landing in more proximal aspects of the aortic arch (Zones 0, 1, and 2). Combined arch repairs involve debranching and reimplantation of the supra-aortic vessels to increase the length of the branchless aortic "tube" and subsequently facilitate adequate sealing between the stent-graft and the aorta. Typically, fully rerouting the brachiocephalic vessels is approached through a median sternotomy, but cardiopulmonary bypass and hypothermic circulatory arrest are only rarely needed.[11] The supra-aortic vessels can be rerouted by using a variety of custom or commercially available branched grafts and are usually brought forward to the ascending aorta, which is used as an inflow source. Alternatively, for less extensive arch-debranching, one may transpose the native left subclavian artery (LSCA) and left common carotid artery (LCCA) onto the innominate artery or perform end-to-side bypass grafts between vessels; such bypasses can be constructed by using prosthetic grafts or reversed saphenous vein grafts. The endovascular portion of the repair is performed simultaneously or is briefly delayed to facilitate recovery, and deployment is approached in either a retrograde or an antegrade fashion. Performing the repair in one stage avoids between-stage rupture—an important problem in two-stage aortic repairs.[12]

The first report of hybrid arch repair described a physically compromised patient who needed reoperation for a leaking aortic arch patch graft. A custom trifurcated graft was prepared; two branches were used to bypass the LCCA and LSCA, and the third branch was used as a conduit to deliver a stent-graft in an antegrade fashion into the

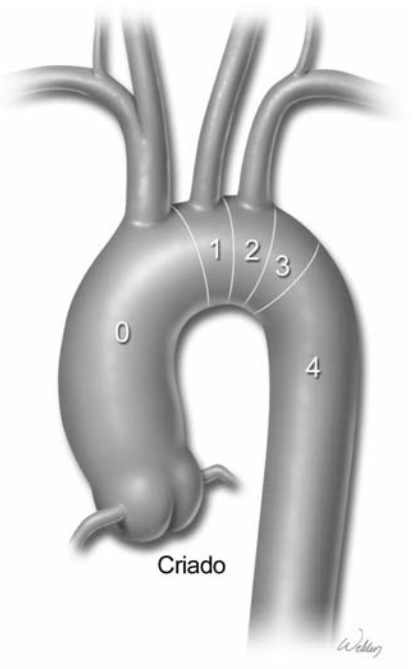


Figure 41-1. The Criado landing zones used to describe the proximal anatomy during an endovascular repair. Zone 0 includes the ascending aorta and the origin of the innominate artery. Zone 1 includes the origin of the left common carotid artery. Zone 2 includes the left subclavian artery origin. Zone 3 is within 2 cm immediately distal to the left subclavian artery, and Zone 4 is more than 2 cm distal to the origin of this vessel.

aortic arch.[13] This repair and a similar repair performed in Japan by Kato et al.[14] opened up the possibility of performing hybrid arch repair in high-risk patients.

Expanding on these distal two-vessel arch-debranching techniques, several authors have reported using total arch rerouting and proximal two-vessel debranching techniques to repair both aortic arch aneurysm and acute ascending aortic dissection (Table 41–2).[11,13,15-24] These repairs incorporate the innominate artery and LCCA into the debranching process (Zones 0 and 1) and may also include the LSCA. Exposure is typically accomplished via median sternotomy, as described above; after a partial occluding clamp is applied to the ascending aorta, a bifurcated or trifurcated graft is anastomosed. Then, the innominate artery is sewn to one of the graft's branches, usually in an end-to-end fashion; the proximal portion of this artery is ligated, oversewn, or merely left to be occluded by the stent-graft.[16-17,19] Next, the LCCA is attached to the graft and ligated proximally. When exposure is compromised by a large arch aneurysm, the branch grafts may be anastomosed to the arteries in an end-to side fashion. In cases of significant displacement of the LCCA or LSCA due to arch disease, one can make additional small neck incisions for these bypasses. When Zone 1 is used for the landing zone, the LCCA can be rerouted by performing a right common carotid artery (RCCA)-to-LCCA bypass through two small vertical neck incisions, thereby avoiding the need for a median sternotomy.[11]

The options for managing the LSCA include coverage of the origin with the stent-graft, revascularization and proximal ligation, and ligation without revascularization. Although simply occluding the LSCA with the stent-graft is generally well tolerated, risks include back-bleeding and Type II endoleak formation as well as arm ischemia, which may be more prevalent than originally thought.[17,25] Type II endoleaks related to LSCA back-bleeding can be successfully treated with coil embolization. If upper extremity ischemia develops after LSCA coverage or ligation, then an LCCA-to-LSCA bypass can be performed through a lateral neck incision (Figure 41–2). Weigang et al. employ a selective approach to LSCA revascularization and advocate revascularizing

TABLE 41-2. REVIEW OF SELECTED REPORTS OF HYBRID ARCH REPAIRS

First Author, Year	Zone 0 or 1 Arch Patients n	Most Common Deployment	1- or 2-Stage Repair	Incision	TIA n (%)	Stroke n (%)	Early Death n (%)	Endovascular Complications
Buth, 1998[13]	1	Antegrade	1	Median sternotomy	0	0	0	None
Bergeron, 2005[31]	11	Retrograde	2	Transverse sternotomy	1 (9)	0	1 (9)	Guidewire perforated left ventricle, leading to death
Saleh, 2006[20]	15	Retrograde	2	Median sternotomy	0	0	0	None
Zhou, 2006[24]	16	Antegrade (8) or retrograde (8)	Both	Not specified	0	0	1 (6)	Anastomoses disturbed by delivery sheath, leading to bleeding and death
Melissano, 2007[18]	26	Retrograde	1	Not specified	—	2 (8)	2 (8)	1 unresolved Type Ia endoleak 2 ruptures resulting in late death
Szeto, 2007[21]	8	Retrograde	1	Median sternotomy	2 (25)	0	1 (13)	None
Chan, 2008[11]	16	Retrograde	1	Median sternotomy (Zone 0) 2 neck incisions with tunneling (Zone 1)	3 (19)	0	0	Type Ib endoleak treated with an additional distal stent-graft
Chen, 2008[15]	6	Retrograde	1	Partial sternotomy with neck incision near LSCA	0	0	0	None
Gottardi, 2008[16]	49	Retrograde	2	Median sternotomy (Zone 0) or upper hemisternotomy (Zone 1)	1 (2)	0	5 (10)	1 death before endovascular stage could be completed
Hughes, 2008[17]	7	Antegrade	1	Median sternotomy	—	0	0	1 case late type II endoleak from retrograde filling by LSCA
Riesenman, 2008[19]	25	Retrograde	Both	Median sternotomy	0	5 (20)	3 (12)	30-day endoleak present in 8 patients 5 type 1a 2 type II 1 type 1a/II combo Authors stated that these were related to incomplete ligations
Wang, 2008[22]	15	Retrograde	Both	Median sternotomy	1 (7)	0	1 (7)	The 1 death resulted from ballooning after a type I endoleak in a case of dissection
Wiegang, 2009[23]	26	Antegrade	1	Upper right L-shaped hemisternotomy	1 (4)	0	4 (15)	1 type II endoleak due to filling from a left vertebral artery arising off the aortic arch

Excludes isolated left subclavian artery (LSCA) bypasses, hybrid elephant trunk repairs, and hybrid prosthetic device implantations; n = number; TIA = transient ischemic attack

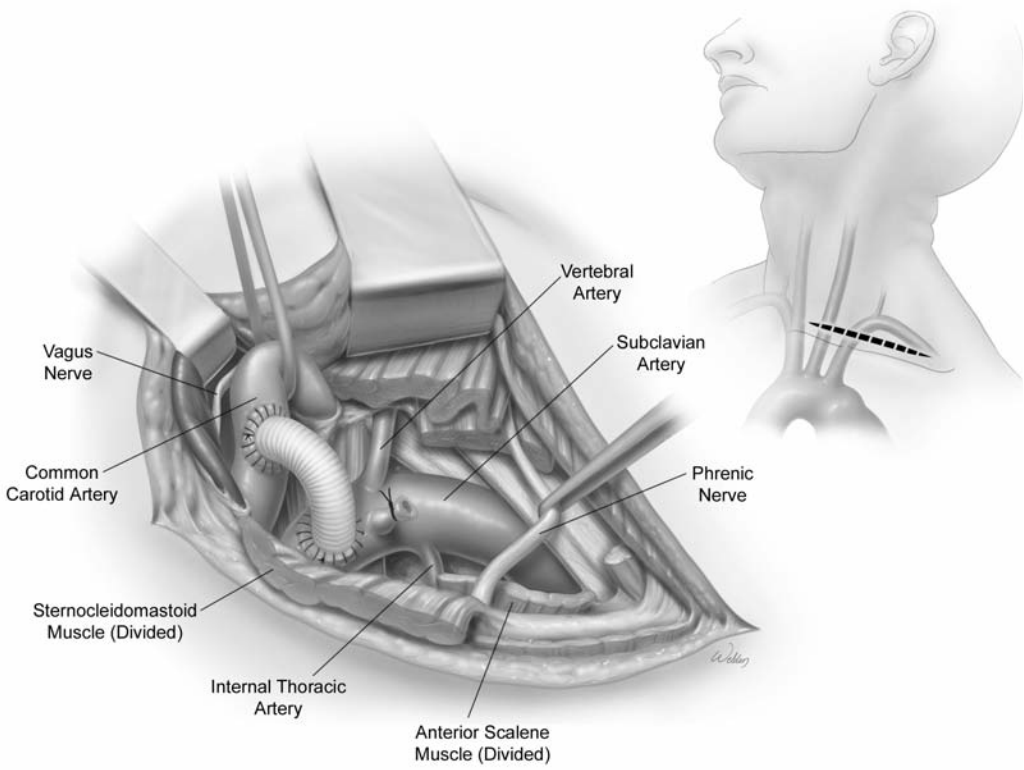


Figure 41-2. Left carotid–to–subclavian artery bypass is performed through a supraclavicular incision (inset). The subclavian artery is exposed by dividing the anterior scalene muscle while the overlying phrenic nerve is gently displaced with a vessel loop. (Used with permission of Elsevier: Bozinovski J, LeMaire SA, Weldon SA, et al. Hybrid repairs of the distal aortic arch and proximal descending thoracic aorta. Op Tech Thorac Cardiovasc Surg 2007;12:167–77.)

the LSCA in patients with the following situations: a dominant left vertebral artery, an incomplete circle of Willis, previous or pending coronary artery revascularization with the left internal thoracic artery; or left upper extremity arteriovenous fistula.[23] In contrast, Gottardi et al. routinely revascularize the LSCA as part of their standard repair protocol.[16]

A few additional technical considerations regarding arch-debranching procedures deserve mention. First, when there is an anomalous left vertebral artery arising from the arch, this artery may be divided at its origin and transposed onto another vessel to prevent the formation of an endoleak.[23,26] Second, although its use is not yet widespread, near-infrared spectroscopy can be used to monitor cerebral perfusion during debranching procedures to prevent brain ischemia.[27] Third, during full arch-debranching, some authors caution against merely ligating the relocated vessels and instead prefer to divide and oversew them to reduce endoleak risk.[19] Fourth, aortic banding can be used in hybrid arch repairs to establish an adequate "neck" and achieve a better seal with the endograft.[15,20,28-29]

The endovascular portion of the repair is performed with the patient under systemic heparinization and controlled hypotension. Adenosine-induced bradycardia and rapid cardiac pacing are two adjunctive techniques that can be used to facilitate precise deployment of the stent-graft.[29-30] In an effort to minimize the amount of contrast

material used, transesophageal echocardiography and intravascular ultrasound can be used to verify the wire position, and fluoroscopic guidance may be used to image the radiopaque markers and landing target.

Deployment of the stent-graft can be performed in a retrograde or an antegrade fashion. In retrograde deployment, the stent-graft is delivered through a femoral or iliac artery. A short Dacron graft conduit (usually 8- or 10-mm in diameter) can be anastomosed to the iliac artery when the femoral artery is narrow or tortuous to aid stent-graft deployment; the use of such conduits reduces the risk of access-vessel complications, which can be catastrophic.[31] In antegrade deployment, a conduit (usually a 10-mm Dacron) graft is attached directly to the ascending aorta or to the main debranching graft at the site of its attachment to the ascending aorta; this conduit is then used to introduce the delivery device into the arch (Figure 41–3). The benefits of the antegrade approach include avoiding femoral or iliac access-vessel complications as well as eliminating the need to advance the stent-graft through a long section of potentially atherosclerotic or tortuous aorta. A drawback of this approach is that the fresh anastomoses are inherently fragile.[24] Consequently, some authors advocate retrograde deployment of the stent-graft after a short delay to allow for vascular healing,[19] whereas others use immediate retrograde deployment after the debranching procedure.[11,18] Gently navigating the stent-graft over a super-stiff guidewire helps prevent disruption of fresh anastomoses. Alternatively, a small incision can be made in the right side of the chest to allow the stent-graft to be introduced without causing excessive mechanical force on the fresh anastomosis.[23]

Hybrid Elephant Trunk Approach

Another Type of combined open and endovascular repair is the hybrid elephant trunk technique. The traditional open elephant trunk procedure is used to repair extensive aortic aneurysms and is performed in two stages. The first stage involves a full arch replacement that leaves a 10-cm "trunk" of Dacron graft hanging beyond the distal anastomosis into the proximal descending thoracic aorta. During the second-stage completion repair—which is usually performed several weeks later—the elephant trunk is used to facilitate the proximal anastomosis of a descending or thoracoabdominal aortic replacement procedure (Figure 41–4). In the hybrid approach to the elephant trunk procedure, the trunk is used as a proximal landing zone during an endovascular completion repair. Placing marker clips or a wire at the distal end of the elephant trunk during the first stage facilitates the placement of the stent-graft during the retrograde second procedure. The hybrid elephant trunk procedure may be performed immediately after the elephant trunk arch replacement, thus eliminating the risk of between-stage aneurysm rupture, which is usually fatal.

Outside the United States, some surgeons use a variation of this approach known as the frozen elephant trunk, which involves a hybrid prosthesis that is part Dacron graft and part stent-graft. The stent-graft end of the device is generally deployed through the open aortic arch into the proximal descending thoracic aorta under direct vision. The proximal Dacron end is then anastomosed to the ascending aortic graft used in the conventional open arch repair.[32-33]

Results

As shown in Table 41–2, outcomes from hybrid arch repair are generally quite good. In these small series of hybrid arch repairs (many of which were abstracted from larger series to focus on proximal arch repair), early mortality rates range from 0 to 15%, and stroke

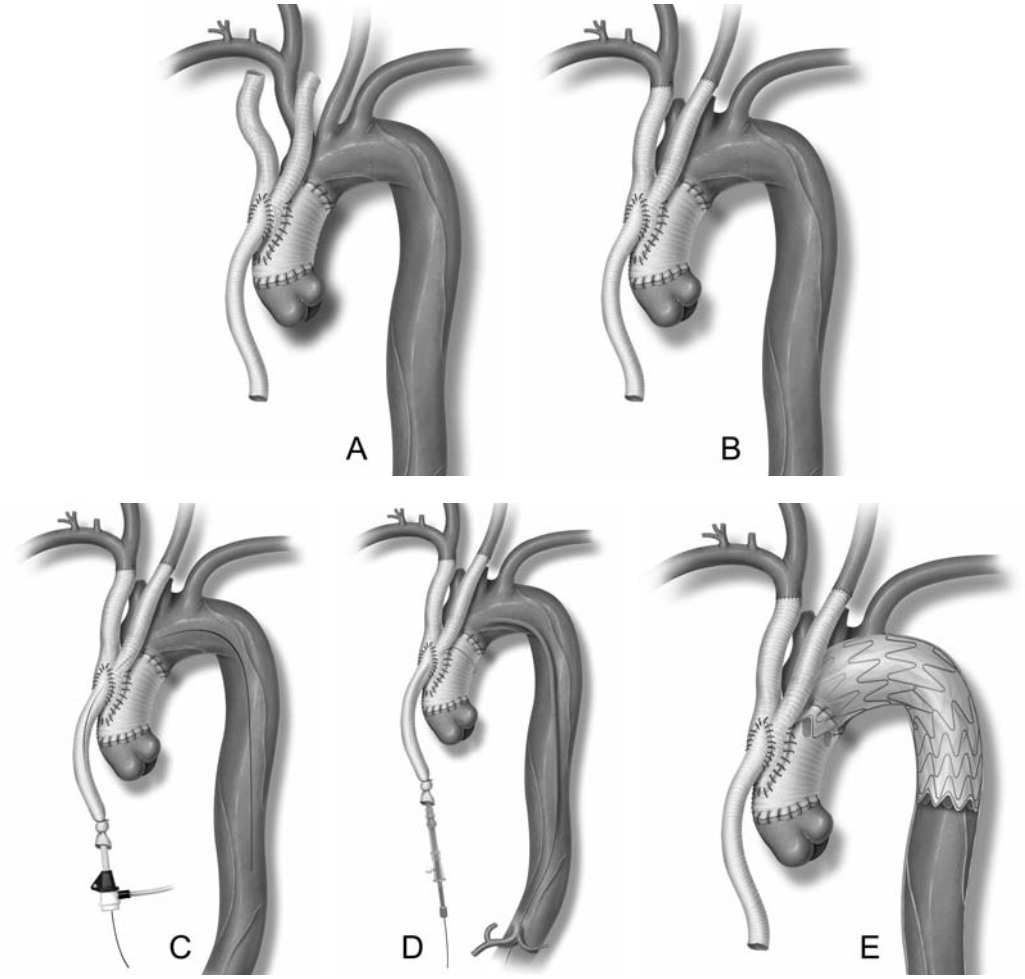


Figure 41-3. Illustration of an antegrade approach to a Zone 0 stent-graft deployment in a hybrid repair of aortic dissection. A. A 10-mm conduit has been attached to a bifurcated debranching graft, which has been anastomosed to the ascending aortic graft. B. The bifurcated graft is anastomosed to the left common carotid and innominate arteries. C. After a 9-Fr sheath is introduced into the conduit, D. the stent-graft is positioned and E. deployed in an antegrade fashion. Note that the proximal portion of the stent-graft lies within the ascending aortic graft. The delivery conduit is transected and oversewn to complete the repair. (Used with permission of Allen Press Publishing Services and Dr. Edward B. Diethrich: Diethrich EB, Ghazoul M, Wheatley GH III, et al. Great vessel transposition for antegrade delivery of the TAG endoprosthesis in the proximal aortic arch. J Endovasc Ther 2005;12:583–7; Diethrich EB, Ghazoul M, Wheatley GH III, et al. Surgical correction of ascending type A thoracic aortic dissection. J Endovasc Ther 2005;12:660–6.)

rates range from 0 to 20%[11,13,15,17,19,20,23]; several of the studies report no early death or stroke. Greenberg et al. reported a 2-year mortality rate of 16% after endovascular elephant trunk completion procedures.[34]

Complications resulting in death include between-stage rupture,[16] aneurysm rupture during balloon angioplasty treatment for endoleak,[22] acute kinking of the stent-graft,[35] stent-graft migration,[18,35] and perforation of the left ventricle by a guidewire.[31] Infrequent complications include sternal wound infection and dehiscence,[23] conversion to open surgery after stent-graft fracture and collapse,[18] bleeding from suture lines necessitating re-exploration,[11] postoperative local dissection,[16,31] spinal cord ischemia,[18-19]

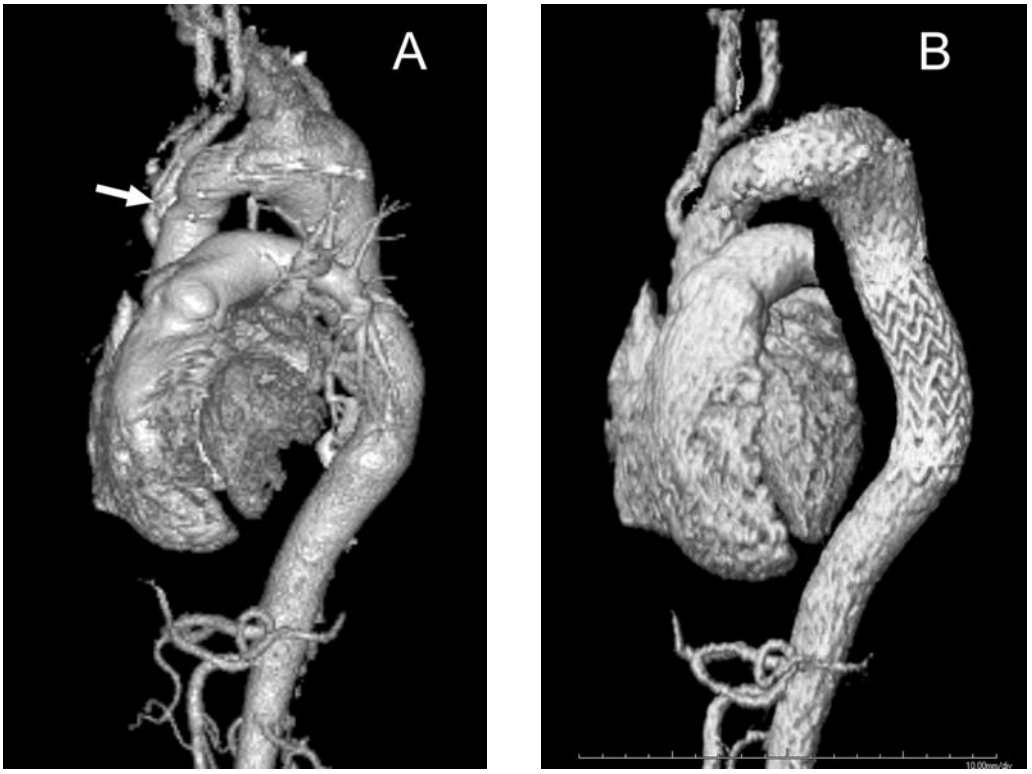
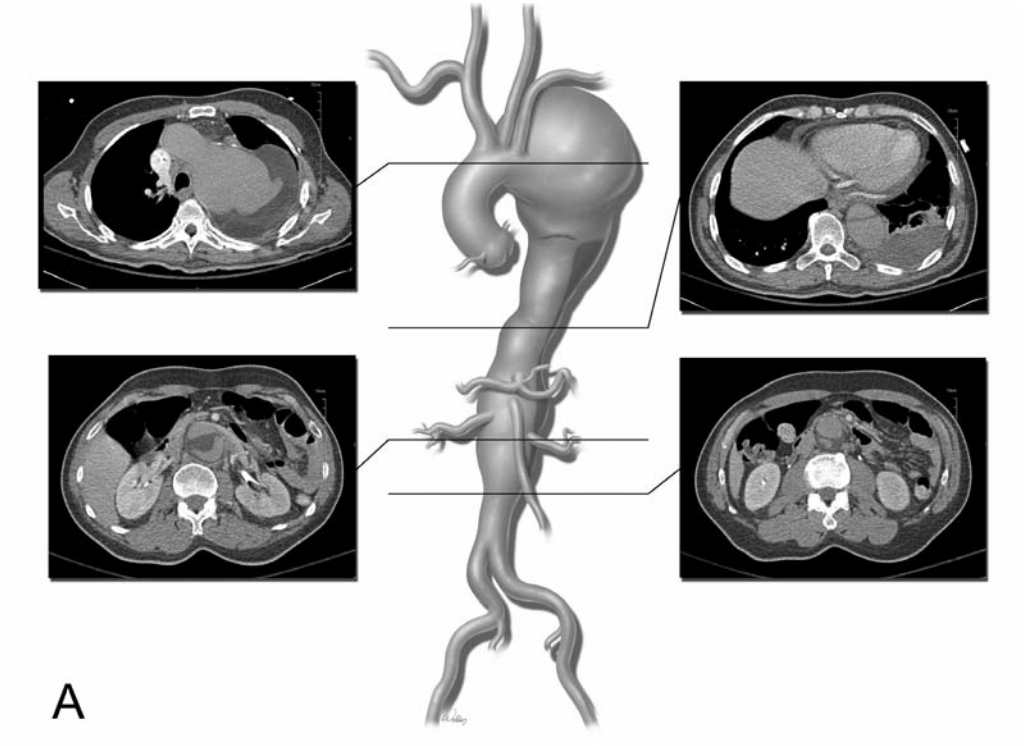


Figure 41-4. Reconstructive imaging of (A) the completed first stage of a hybrid elephant trunk repair of the aortic arch and proximal descending thoracic aorta. The brachiocephalic circulation has been debranched by placing a graft (arrow) from the ascending aortic graft to the innominate artery and left common carotid artery. During the Stage 2 completion repair (B), the trunk was used as the proximal landing zone for the descending thoracic aortic stent-graft.

and acute renal failure.[18,24,36] Erosion of the Dacron graft by the stent-graft is a rare complication after hybrid elephant trunk repairs.[37]

In our experience with nine hybrid arch repairs performed since November 2005, including one case with concomitant hybrid thoracoabdominal aortic aneurysm (TAAA) repair (Figure 41–5), many of the patients had chronic dissection and previous aortic repair. There were three early deaths (33%) and one perioperative stroke (11%). Although there were no early endoleaks in the six survivors, both Type I and Type II endoleaks developed in one survivor almost 2 years postoperatively; these endoleaks were treated with a secondary stent-graft procedure and coil embolization, respectively.

We have performed 14 hybrid elephant trunk procedures since February 2006, with 1 early death (7%). Although there were no cases of paraplegia, there was 1 case of stroke (7%) and 1 of renal failure (7%). Early endoleak occurred in 4 cases (3 Type II endoleaks and 1 Type IV endoleak). The Type II endoleaks necessitated conversion to open repair in the first case, a secondary intervention with coil embolization and deployment of a Palmaz stent in the second case, and observation only in the third case. The patient with the Type IV endoleak underwent a secondary stent-graft placement 4 months later. There were 2 late deaths, at 24 and 27 months postoperatively.



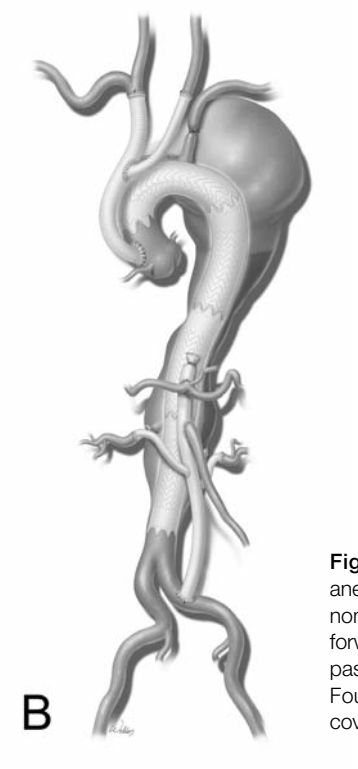
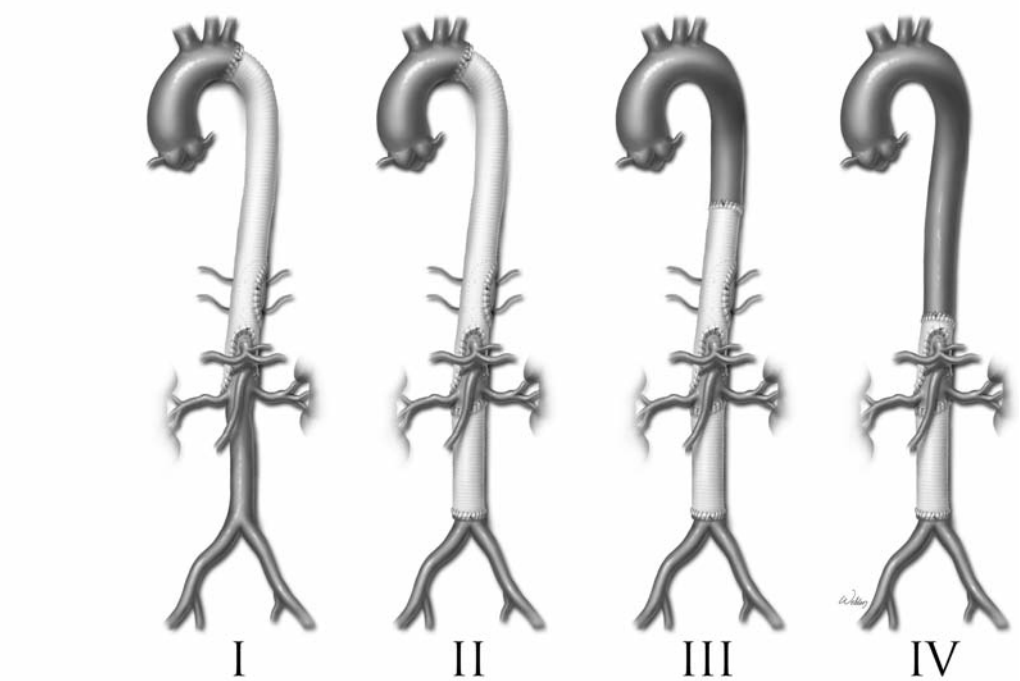


Figure 41-5. (A) A hybrid approach was used to repair this extensive aneurysm involving the aortic arch and thoracoabdominal aorta. (B) The innominate and left common carotid arteries were debranched and brought forward with inflow from the ascending aorta. The visceral arteries were bypassed in a retrograde fashion with inflow from the left common iliac artery. Four stent-grafts were deployed to exclude an extensive aneurysm and cover nearly the entire aorta.

HYBRID APPROACH TO THORACOABDOMINAL AORTIC REPAIR

Typical Complications of Traditional Open Thoracoabdominal Aortic Repair

Contemporary management strategies enable patients to undergo open TAAA repair with excellent early survival and respectable morbidity, particularly in experienced centers; the overall primary risks for all extents of repair range from 5 to 12% for early mortality, 4 to 10% for paraplegia, and 4 to 12% for renal failure.[38-40] Other complications include pulmonary dysfunction (largely as a result of single-lung ventilation and preexisting lung disease), stroke, and myocardial infarction. The rates of most complications differ greatly by the extent of repair, with Crawford extent II involving the greatest overall risk of an adverse event (Figure 41–6).[40] Surgical risk is also generally increased by patient-specific comorbidities such as renal dysfunction, chronic obstructive pulmonary disease, and coronary artery occlusive disease.[39,41] Reoperative and very elderly patients are especially challenging, and many surgeons are reluctant to operate on them. In these very high-risk patients, who are poor candidates for traditional open repair, a combined approach has been suggested as a viable alternative.



	I	II	III	IV
Early Mortality	5.0%	6.0%	6.4%	3.0%
Paraplegia	3.3%	6.3%	2.6%	1.4%
Renal Failure	2.7%	8.3%	6.1%	5.4%

Figure 41-6. Illustration of Crawford extents of thoracoabdominal aortic aneurysm (TAAA) repair and associated early mortality, paraplegia, and renal failure rates.[40] Whereas extent I repair carries a relatively low risk of both paraplegia and renal failure, extent II repair carries a much higher risk of paraplegia and renal failure. (Used with permission of Elsevier: Coselli JS, Bozinovski J, LeMaire SA. Open surgical repair of 2286 thoracoabdominal aortic aneurysms. Ann Thorac Surg 2007;83:S862–4.)

Debranching to Enable Coverage of the Visceral Artery Origins

Hybrid thoracoabdominal aortic repair involves rerouting some or all of the visceral arteries (i.e., the celiac axis, superior mesenteric, and renal arteries), such that blood flow to the viscera is not interrupted by subsequent stent-graft exclusion of the TAAA. Complexity of repair increases when longer lengths of the aorta are covered and greater numbers of vessels require debranching. In 1998, the first hybrid TAAA repair was performed by Quinones-Baldrich et al.[42] in a patient with an extent IV TAAA. They performed open extra-anatomic bypasses to the renal, superior mesenteric, and celiac arteries, followed by endovascular exclusion of the aneurysm. Since then, several small series and case reports have been published; a summary of selected studies is presented in Table 41–3.[17,24,43-52]

Advantages of the hybrid TAAA approach include the avoidance of aortic cross-clamping and single-lung ventilation as well as the reduction of visceral ischemia times, which may benefit patients with cardiopulmonary comorbidities, prevent reperfusion injury, and protect against associated complications such as renal dysfunction and cardiac strain. Many groups use cerebrospinal fluid drainage during hybrid TAAA repairs to reduce the risk of paraplegia.[36,46,53] Although renal ischemic times are shorter than those required by open TAAA repairs, renal injury may be exacerbated by the large amounts of contrast media that are generally necessary to obtain the detailed images needed to plan and conduct the repair. Some centers use cold crystalloid renal perfusion during renal artery clamping to protect against renal dysfunction.[46]

Although a less extensive incision is used (as compared with that used in standard open repair), exposure for TAAA debranching usually requires substantial retroperitoneal or transperitoneal exposure.[17] However, there are a few reports of minimally invasive laparoscopic or laparorobotic hybrid TAAA procedures,[54-55] and these approaches may gain appeal.

There are many ways in which visceral-vessel debranching procedures can vary, including the Type of inflow, the Type of debranching graft used, and the approach to selecting and bypassing target arteries. Inflow can be provided from an antegrade source (e.g., proximal aortic segments) or a retrograde one (e.g., an iliac artery) and can be established by creating one or multiple proximal anastomoses (Figure 41–7).[24,46] The approach to rerouting the visceral circulation is extensively tailored to the individual patient and may include both antegrade and retrograde bypasses within the same repair.[56] The inflow origin should be selected such that it is not compromised during or after stent-graft deployment and is relatively free of disease such as atherosclerosis or heavy calcification. The inflow anastomosis is usually end-to-side.

There are several potential configurations for visceral debranching grafts. Options include the "Lazy C" as popularized by Black,[44] inverted bifurcated grafts,[48] Y grafts as preferred by Chiesa,[46] and any number of custom grafts.[17,57-58] Most often, 8- or 10-mm Dacron or polytetrafluoroethylene (PTFE) grafts are used for the bypasses, but occasionally a saphenous or deep vein graft may be incorporated into the repair or a vessel may be transposed.[52,59-61] Uncommon strategies for visceral debranching have included using an "octopus" graft from the ascending aorta to revascularize the visceral arteries because of diseased iliac arteries,[62] using a pedicled right iliac artery with a saphenous vein graft to revascularize the superior mesenteric and common hepatic arteries,[60] and reimplanting the visceral arteries as a patch on a short section of a 20-mm Dacron tube graft.[63] Additional options include a newly developed, commercially available branched graft (designed to reroute the arch vessels) that has been modified by

TABLE 41-3. SELECT SERIES OF CONTEMPORARY HYBRID TAAA REPAIRS

First Author, Year	Hybrid TAAA Patients n	1- or 2-Stage Repair	Extent of Repair	Early Endoleak n (%)	Paraplegia n (%)	Renal Dysfunction n (%)	Early Death n (%)	Comment
Black, 2006[44]	29	1	I-IV	11 (38)	0	4 (14)	7 (24)	Procedure was abandoned in 3 patients, 1 of whom had rupture on postoperative day 10
Resch, 2006[51]	13	Both	II, III, IV	8 (62)	2 (15)	2 (15)	3 (23)	1 patient had between-stage rupture that resulted in early death
Zhou, 2006[24]	15	Both	I, III, IV	NR	0	2 (13)	0	2 patients (33) had renal artery thrombosis that necessitated hemodialysis
Chiesa, 2007[46]	13	1	I, II, IV, VAP	0	1 (8)	2 (15)	3 (23)	Early deaths were related to respiratory failure, coagulopathy, and pancreatitis
Donas, 2007[47]	8	Both	I, II, IV, V	NR	0	1 (13)	1 (13)	1 patient (13) had mesenteric bypass occlusion that later necessitated reoperation
Gawenda, 2007[48]	6	1	II, III, IV	0	0	0	0	2 patients (33) had renal artery thrombosis during follow-up period
Lee, 2007[49]	17	2	II, III, IV	1 (6)	0	1 (6)	4 (24)	A 17-year-old patient was later suspected of having a connective tissue disorder after an additional aneurysm developed All early deaths occurred after the debranching procedure
Böckler, 2008[50]	28	Both	I-IV	5 (18)	4 (14)	8 (29)	4 (14)	3 patients (11) had peripheral graft occlusion within 30 days of procedure
Hughes, 2008[17]	6	1	II, V, VAP	0	0	0	0	2 patients (33) needed renal dialysis preoperatively
Siegenthaler, 2008[52]	10	Both	I-IV	0	0	NR	1 (10)	5 cases (50) were selective revascularizations of celiac axis after coverage during the endovascular repair
Aguiar Lucas, 2009[43]	10	Both	IV, V	0	0	2 (20)	1 (10)	1 patient died 3 months postoperatively due to an infected graft
Quinones-Baldrich, 2009[50]	15	Both	I-IV	4 (27)	1 (7)	1 (7)	0	2 patients (13) had small bowel obstruction that necessitated reoperation

n = number; NR = not reported; TAAA = thoracoabdominal aortic aneurysm; VAP = visceral artery patch.

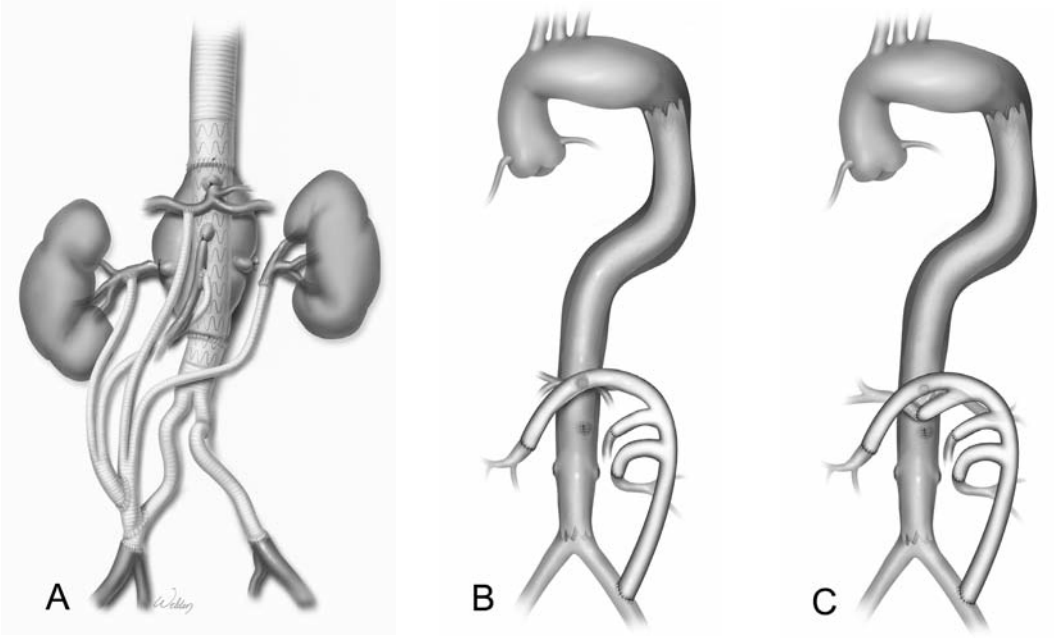


Figure 41-7. Options for visceral debranching by using retrograde inflow from the common iliac artery include (A) using a homemade graft attached end-to-end to the left renal artery and end-to-side to the remaining visceral arteries; and using commercially available custom-fabricated grafts attached end-to-side (B) or end-to-end (C) to the celiac axis and end-to-end to the remaining visceral arteries. (Used with permission of Elsevier: Zhou W, Reardon M, Peden EK, et al. Hybrid approach to complex thoracic aortic aneurysms in high-risk patients: surgical challenges and clinical outcomes. J Vasc Surg 2006;44:688–693), and Dr. Chad Hughes (Hughes GC, Nienaber JJ, Bush EL, et al. Use of custom Dacron branch grafts for "hybrid" aortic debranching during endovascular repair of thoracic and thoracoabdominal aortic aneurysms. J Thorac Cardiovasc Surg. 2008;136:21–28).

lengthening one branch so that the conduit can be used to bypass the celiac axis[57] and a commercially available custom-fabricated branched graft that has side branches for each of the visceral arteries and a conduit for subsequent endovascular deployment.[17]

The selection of target vessels for debranching is also highly variable. One highly controversial topic is how to manage the celiac axis. Several authors state that the celiac axis can be safely occluded, provided that there is sufficient collateral circulation, as can be demonstrated by a balloon occlusion test or, theoretically, by a gastric pH calculation.[64-65] However, others conclude that a balloon occlusion test does not guarantee that the celiac axis can be safely occluded, and they report numerous complications associated with not revascularizing the celiac axis, such as foregut ischemia, choledocholithiasis, and endoleak.[66] Still others suggest taking a minimal approach to repair whenever possible, and they freely occlude the celiac axis if no additional visceral rerouting is required, despite the risk of late reintervention for Type I endoleak; they then selectively revascularize as needed.[52] In some patients, it is necessary to reroute the inferior mesenteric artery.[52,61] The visceral vessels are typically debranched sequentially to minimize the duration of end-organ ischemia. Although the distal anastomoses are most often created in an end-to-end fashion, end-to-side anastomoses can be useful in some graft configurations.[17] After the target artery is bypassed, the proximal aspect of the artery is ligated. Black et al.[44] have cautioned against using

surgical clips to ligate debranched arteries because the clips can become displaced and cause a Type II endoleak. Once revascularized, the vast majority of visceral grafts remain patent.[67]

An important aspect of the open debranching procedure is that it involves a variety of techniques to facilitate stent-graft delivery, including optimization of landing zones. For example, conduits can be added to a debranching graft to facilitate simultaneous stent-graft deployment.[17,50,58,68] A subcutaneous conduit can be created to facilitate later cut-down access for delayed stent-graft placement.[53] Additionally, radiopaque markers are often added to the debranching graft to facilitate later positioning of the stent-graft. In selected cases, a secure distal landing zone can be created by fully replacing a short section of the distal aorta with a graft, which is sometimes tapered to accommodate the planned diameter of the stent-graft.[50] Alternatively, the aorta can be banded by wrapping a small length of graft around the planned distal landing zone.[69] For some Crawford extent I or II repairs, it may also be necessary to relocate some of the brachiocephalic vessels, such as the LSCA or LCCA, to ensure an adequate proximal landing zone.[48]

The endovascular portion of the repair may be performed simultaneously or at a later time (two-stage repair). Although delayed repair gives patients a chance to heal from the debranching procedure, this delay also incurs a risk of between-stage rupture.[45,49-50] Rarely, the endovascular stage of repair precedes the debranching procedures, as in the case of an emergent repair.[47] Endovascular deployment, with or without a facilitating conduit, is most commonly performed retrograde (i.e., through the femoral or iliac arteries) but may be performed in an antegrade manner (e.g., through the common carotid arteries), especially if the femoral or iliac arteries are heavily calcified or tortuous.[47,63] In hybrid repairs, a simple "tube" graft is most commonly used, although a bifurcated module may be added distally.[49,59,70] The endograft is deployed as it is in thoracic endovascular aortic repair (TEVAR), with at least a 2-cm section of normal aorta for the proximal and distal landing zones. Commonly, more than one stent-graft is used because the length of aorta to be covered in a TAAA hybrid repair tends to be much greater than that covered in TEVAR; sequential stent grafts generally overlap by at least 5 cm.

Results

The technical success of endovascular deployment during hybrid TAAA repairs ranges from 70% up to 100%.[44-45,49,67] Clinical outcomes from select series of hybrid TAAA repairs are presented in Table 41–3, with early mortality rates ranging from 0 to 24%, paraplegia rates ranging from 0 to 15%, and renal dysfunction rates ranging from 0 to 29%. In a systematic review of 13 series with 58 patients, Donas et al.[67] found that elective and urgent repairs were associated with a mortality rate of 11%, no cases of paraplegia, a renal dysfunction rate of 9%, and an overall endoleak rate of 21%. Pre-existing morbidities clearly affect outcomes after hybrid TAAA repair, much as they influence traditional open TAAA repair.[49] For example, Böckler et al.[45] identified chronic obstructive pulmonary disease as an independent predictive risk factor for mortality in hybrid TAAA repairs.

Paraplegia and paraparesis remain poorly understood in hybrid TAAA repair. Reported cases are presented in Table 41–4. The extent of repair (i.e., the amount of aorta covered by the stent-graft) affects the likelihood of paraplegia, as does postoperative hypotension, which has been associated with several cases of delayed deficits.[17,45,50-53,62,68,71] Other complications include ischemic bowel,[52] myocardial infarction,[47]

TABLE 41-4. REVIEW OF SPINAL CORD ISCHEMIA IN HYBRID TAAA REPAIR

First Author, Year	Hybrid TAAA n	Paraplegia	Paraparesis	Prophylactic CSF Drain	CSF Drain Placed after Deficit	Reported Hypotension	Disease	Staged Repair	Comment
Resch, 2006[51]	13	2 (15%)	2 (15%)	Yes	No	Yes	Extent II and III	NR	Death in 2 with paraplegia and 1 with paraparesis Resolved at 12 months in the remaining patient
Chiesa, 2007[46]	13	1 (8%)	0	Only in I and II	Yes	No	VAP	No	Onset delayed 2 days Resolved after CSF drain placed
Lawlor, 2007[68]	2	1 (50%)	0	Yes	No	No	Extent I aneurysm with infrarenal aneurysm	No	Onset delayed 24 hours Persisted at 8 weeks with significant motor deficit
Torsello, 2007[62]	1	0	1 (100%)	No	Yes	No	Extent II aneurysm with dissection	Yes	Onset delayed 24 hours Resolved after 3 days of CSF drainage
Ballard, 2008[53]	4	0	2 (50%)	Yes	No	No	Type II dissection Extent II aneurysm	Yes	Persisted at 12 months in dissection patient Resolved at 3 months in aneurysm patient
Böckler, 2008[45]	28	4 (14%)	0	NR	NR	Yes	NR	NR	Persisted at publication in 3 patients Resolved in 1 patient Moved toward staged repair to reduce risk of paraplegia
Hughes, 2008[17]	6	0	1 (17%)	Yes	Yes	No	Extent II	No	Onset after CSF drain clamped Resolved after additional drainage
Siegenthaler, 2008[52]	13	0	1 (8%)	Yes	No	Yes	NR	NR	Onset delayed 3 weeks Resolved with discontinuation of antihypertensive medications Ambulation recovered Mild unilateral deficit persisted at publication Also used evoked potentials as protective measure
Tshomba, 2008[71]	7	1 (14%)	0	No	Yes	No	VAP	No	Onset delayed 2 days Resolved after CSF drainage
Quinones-Baldrich, 2009[50]	15	1 (7%)	0	Yes	No	Yes	Extent III	No	Onset delayed 24 hours Fatal MI 6 months postoperatively

CSF = cerebrospinal fluid; MI = myocardial infarction; NR = not reported; VAP = visceral artery patch; TAAA = thoracoabdominal aortic aneurysm; n = number.

stroke,[52] pancreatic fistula leading to late death,[51] interim rupture,[49,51] renal failure,[45] prolonged ileus,[24] and infection.[43,49,52] The risk of endoleak is not insignificant for hybrid TAAA procedures. Patent intercostal and lumbar arteries covered by the stent-graft may back-bleed, causing a Type II endoleak; such bleeding near a landing zone can compromise the seal, leading to a Type I endoleak. Reintervention for endoleak is not uncommon and usually involves performing a secondary endovascular procedure to insert additional devices, using balloon dilation to achieve a better seal or using coil embolization.

Our experience with TAAA hybrid repair involves four patients who underwent these procedures since February 2007. As previously mentioned, one of these patients also underwent arch-vessel debranching (see Figure 41–5). All four patients required Crawford extent II repairs but had substantial comorbidities and were therefore not considered satisfactory candidates for open repair. Despite technically successful procedures (all aneurysms were excluded upon endograft deployment and no endoleaks occurred), clinical outcomes were disappointing. There were two early deaths, one case of paraplegia, and two late deaths that occurred on postoperative days 152 and 244.

CONCLUSIONS

These innovative approaches to treating complex aortic aneurysms offer several potential advantages over standard open repairs and purely endovascular repairs. For example, compared with standard open surgical repair, a hybrid repair typically involves less blood loss, less transfused blood, fewer pulmonary complications, and shorter intensive care and hospital stays, and patients are more likely to be discharged home rather than to extended care. In theory, hybrid repairs also reduce the risk of Type 1 endoleak by increasing surgeons' ability to select appropriate landing zones that are well away from the aneurysmal aortic segment and critical branch arteries.

However, there may not be a benefit in terms of early mortality, spinal cord ischemia, or stroke. There is conflicting evidence regarding whether or not renal complications are reduced in endovascular repair, and although one could infer that the reduced amount of renal ischemia should be accompanied by a reduction in renal complications, this benefit may be offset by inflammatory processes, contrast administration, embolization, and other factors that adversely affect the kidneys.[72] Additionally, because hybrid approaches are relatively new, there are limited long-term data on their outcomes, although 10-year reports are now emerging.[50] Despite these current limitations, combined repair offers the opportunity to capitalize on the beneficial aspects of open and endovascular repair and thus maximize the benefit of repair for the individual patient. Importantly, hybrid repairs are extending the treatment options for high-risk patients who do not have adequate physiologic reserve to undergo traditional open repairs.

ACKNOWLEDGMENTS

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REFERENCES

1. Parodi JC, Palmaz JC, Barone HD. Transfemoral intraluminal graft implantation for abdominal aortic aneurysms. Ann Vasc Surg 1991;5:491–9.
2. Rodriguez JA, Olsen DM, Shtutman A, et al. Application of endograft to treat thoracic aortic pathologies: a single center experience. J Vasc Surg 2007;46:413–20.
3. Greenberg RK, Clair D, Srivastava S, et al. Should patients with challenging anatomy be offered endovascular aneurysm repair? J Vasc Surg 2003;38:990–6.
4. LeMaire SA. Combining open and endovascular approaches to complex aneurysms. Ann N Y Acad Sci 2006;1085:208–12.
5. Kamiya H, Hagl C, Kropivnitskaya I, et al. Quick proximal arch replacement with moderate hypothermic circulatory arrest. Ann Thorac Surg 2007;83:1055–8.
6. Kazui T, Bashar AH. Aortic arch replacement using a trifurcated graft. Ann Thorac Surg 2006;81:1552.
7. Suzuki K, Kazui T, Bashar AH, et al. Total aortic arch replacement in patients with arch vessel anomalies. Ann Thorac Surg 2006;81:2079–83.
8. Czerny M, Fleck T, Zimpfer D, et al. Risk factors of mortality and permanent neurologic injury in patients undergoing ascending aortic and arch repair. J Thorac Cardiovasc Surg 2003;126:1296–301.
9. Shah PJ, Estrera AL, Miller CC III, et al. Analysis of ascending and transverse aortic arch repair in octogenarians. Ann Thorac Surg 2008;86:774–9.
10. Criado FJ, Clark NS, Barnatan MF. Stent graft repair in the aortic arch and descending thoracic aorta: a 4-year experience. J Vasc Surg 2002;36:1121–8.
11. Chan YC, Cheng SW, Ting AC, et al. Supra-aortic hybrid endovascular procedures for complex thoracic aortic disease: single center early to midterm results. J Vasc Surg 2008;48: 571–9.
12. Diethrich EB, Ghazoul M, Wheatley GH III, et al. Great vessel transposition for antegrade delivery of the TAG endoprosthesis in the proximal aortic arch. J Endovasc Ther 2005; 12:583–7.
13. Buth J, Penn O, Tielbeek A, et al. Combined approach to stent-graft treatment of an aortic arch aneurysm. J Endovasc Surg 1998;5:329–32.
14. Kato M, Kaneko M, Kuratani T, et al. New operative method for distal aortic arch aneurysm: combined cervical branch bypass and endovascular stent-graft implantation. J Thorac Cardiovasc Surg 1999;117:832–4.
15. Chen IM, Wu FY, Shih CC. Banding technique for endovascular repair of arch aneurysm with unsuitable proximal landing zone. Circ J 2008;72:1981–5.
16. Gottardi R, Funovics M, Eggers N, et al. Supra-aortic transposition for combined vascular and endovascular repair of aortic arch pathology. Ann Thorac Surg 2008;86:1524–9.
17. Hughes GC, Nienaber JJ, Bush EL, et al. Use of custom Dacron branch grafts for "hybrid" aortic debranching during endovascular repair of thoracic and thoracoabdominal aortic aneurysms. J Thorac Cardiovasc Surg 2008;136:21–8, 28 e21–6.
18. Melissano G, Civilini E, Bertoglio L, et al. Results of endografting of the aortic arch in different landing zones. Eur J Vasc Endovasc Surg 2007;33:561–6.
19. Riesenman PJ, Tamaddon HS, Farber MA. Surgical bypass procedures to facilitate endovascular repair of aortic arch pathology. J Cardiovasc Surg (Torino) 2008;49:461–9.
20. Saleh HM, Inglese L. Combined surgical and endovascular treatment of aortic arch aneurysms. J Vasc Surg 2006;44:460–6.
21. Szeto WY, Bavaria JE, Bowen FW, et al. The hybrid total arch repair: brachiocephalic bypass and concomitant endovascular aortic arch stent graft placement. J Card Surg 2007;22: 97–102; discussion 103–4.
22. Wang S, Chang G, Li X, et al. Endovascular treatment of arch and proximal thoracic aortic lesions. J Vasc Surg 2008;48:64–8.
23. Weigang E, Parker J, Czerny M, et al. Endovascular aortic arch repair after aortic arch debranching. Ann Thorac Surg 2009;87:603–7.

24. Zhou W, Reardon M, Peden EK, et al. Hybrid approach to complex thoracic aortic aneurysms in high-risk patients: surgical challenges and clinical outcomes. J Vasc Surg 2006;44:688–93.
25. Reece TB, Gazoni LM, Cherry KJ, et al. Reevaluating the need for left subclavian artery revascularization with thoracic endovascular aortic repair. Ann Thorac Surg 2007;84: 1201–5; discussion 1205.
26. Gottardi R, Seitelberger R, Zimpfer D, et al. An alternative approach in treating an aortic arch aneurysm with an anatomic variant by supraaortic reconstruction and stent-graft placement. J Vasc Surg 2005;42:357–60.
27. Santo KC, Barrios A, Dandekar U, et al. Near-infrared spectroscopy: an important monitoring tool during hybrid aortic arch replacement. Anesth Analg 2008;107:793–6.
28. Antona C, Vanelli P, Petulla M, et al. Hybrid technique for total arch repair: aortic neck reshaping for endovascular-graft fixation. Ann Thorac Surg 2007;83:1158–61.
29. Bergeron P, Mangialardi N, Costa P, et al. Great vessel management for endovascular exclusion of aortic arch aneurysms and dissections. Eur J Vasc Endovasc Surg 2006;32:38–45.
30. Slovut DP, Sullivan TM. Combined endovascular and open revascularization. Ann Vasc Surg 2009;23:414–24.
31. Bergeron P, Coulon P, De Chaumaray T, et al. Great vessels transposition and aortic arch exclusion. J Cardiovasc Surg (Torino) 2005;46:141–7.
32. Di Bartolomeo R, Di Marco L, Armaro A, et al. Treatment of complex disease of the thoracic aorta: the frozen elephant trunk technique with the E-vita open prosthesis. Eur J Cardiothorac Surg 2009;35:671–5; discussion 675–6.
33. Karck M, Kamiya H. Progress of the treatment for extended aortic aneurysms: is the frozen elephant trunk technique the next standard in the treatment of complex aortic disease including the arch? Eur J Cardiothorac Surg 2008;33:1007–13.
34. Greenberg RK, Haddad F, Svensson L, et al. Hybrid approaches to thoracic aortic aneurysms: the role of endovascular elephant trunk completion. Circulation 2005;112:2619–26.
35. Palma JH, Guilhen JS, Gaia DF, et al. Early complication after hybrid thoracic aortic aneurysm repair. Interact CardioVasc Thorac Surg 2008;7:441–3.
36. Brueck M, Heidt MC, Szente-Varga M, et al. Hybrid treatment for complex aortic problems combining surgery and stenting in the integrated operating theater. J Interv Cardiol 2006;19:539–43.
37. Fehrenbacher JW, McCready RA. Erosion of elephant trunk Dacron graft limb by thoracic endograft causing acute aneurysm expansion. J Vasc Surg 2009;49:491–3.
38. Chiesa R, Melissano G, Civilini E, et al. Ten years experience of thoracic and thoracoabdominal aortic aneurysm surgical repair: lessons learned. Ann Vasc Surg 2004;18:514–20.
39. Conrad MF, Crawford RS, Davison JK, et al. Thoracoabdominal aneurysm repair: a 20-year perspective. Ann Thorac Surg 2007;83:S856–61; discussion S852–90.
40. Coselli JS, Bozinovski J, LeMaire SA. Open surgical repair of 2286 thoracoabdominal aortic aneurysms. Ann Thorac Surg 2007;83:S862–4.
41. LeMaire SA, Miller CC III, Conklin LD, et al. A new predictive model for adverse outcomes after elective thoracoabdominal aortic aneurysm repair. Ann Thorac Surg 2001;71:1233–8.
42. Quinones-Baldrich WJ, Panetta TF, Vescera CL, et al. Repair of type IV thoracoabdominal aneurysm with a combined endovascular and surgical approach. J Vasc Surg 1999;30: 555–60.
43. Aguiar Lucas L, Rodriguez-Lopez JA, Olsen DM, et al. Endovascular repair in the thoracic and abdominal aorta: no increased risk of spinal cord ischemia when both territories are treated. J Endovasc Ther 2009;16:189–96.
44. Black SA, Wolfe JH, Clark M, et al. Complex thoracoabdominal aortic aneurysms: endovascular exclusion with visceral revascularization. J Vasc Surg 2006;43:1081–9; discussion 1089.
45. Böckler D, Kotelis D, Geisbusch P, et al. Hybrid procedures for thoracoabdominal aortic aneurysms and chronic aortic dissections—a single center experience in 28 patients. J Vasc Surg 2008;47:724–32.

46. Chiesa R, Tshomba Y, Melissano G, et al. Hybrid approach to thoracoabdominal aortic aneurysms in patients with prior aortic surgery. J Vasc Surg 2007;45:1128–35.
47. Donas KP, Schulte S, Krause E, et al. Combined endovascular stent-graft repair and adjunctive visceral vessel reconstruction for complex thoracoabdominal aortic aneurysms. Int Angiol 2007;26:213–8.
48. Gawenda M, Aleksic M, Heckenkamp J, et al. Hybrid-procedures for the treatment of thoracoabdominal aortic aneurysms and dissections. Eur J Vasc Endovasc Surg 2007;33: 71–7.
49. Lee WA, Brown MP, Martin TD, et al. Early results after staged hybrid repair of thoracoabdominal aortic aneurysms. J Am Coll Surg 2007;205:420–31.
50. Quinones-Baldrich W, Jimenez JC, DeRubertis B, et al. Combined endovascular and surgical approach (CESA) to thoracoabdominal aortic pathology: a 10-year experience. J Vasc Surg 2009;49:1125–34.
51. Resch TA, Greenberg RK, Lyden SP, et al. Combined staged procedures for the treatment of thoracoabdominal aneurysms. J Endovasc Ther 2006;13:481–9.
52. Siegenthaler MP, Weigang E, Brehm K, et al. Endovascular treatment for thoracoabdominal aneurysms: outcomes and results. Eur J Cardiothorac Surg 2008;34:810–9.
53. Ballard JL, Razavi M, Harward TR, et al. Visceral/renal artery debranching for complex thoracoabdominal hybrid procedures via retroperitoneal abdominal aortic exposure: a preliminary report. Ann Vasc Surg 2008;22:173–8.
54. Bakoyiannis C, Cagiannos C, Wasilljew S, et al. Totally laparoscopic aortohepatic bypass for aortic debranching during endovascular thoracoabdominal aneurysm repair. Eur J Vasc Endovasc Surg 2007;34:173–5.
55. Wahlgren CM, Skelly C, Shalhav A, et al. Hybrid laparorobotic debranching and endovascular repair of thoracoabdominal aortic aneurysm. Ann Vasc Surg 2008;22:285–9.
56. Fulton JJ, Farber MA, Marston WA, et al. Endovascular stent-graft repair of pararenal and type IV thoracoabdominal aortic aneurysms with adjunctive visceral reconstruction. [erratum appears in J Vasc Surg 2005 May;41(5):906]. J Vasc Surg 2005;41:191–8.
57. Esposito G, Marullo AG, Pennetta AR, et al. Hybrid treatment of thoracoabdominal aortic aneurysms with the use of a new prosthesis. Ann Thorac Surg 2008;85:1443–5.
58. Kpodonu J, Shennib H, Wheatley GH III, et al. A novel technique of deployment of a thoracic endograft in the hybrid treatment of a patient with thoracoabdominal aneurysm. Ann Thorac Surg 2008;85:666–8.
59. Flye MW, Choi ET, Sanchez LA, et al. Retrograde visceral vessel revascularization followed by endovascular aneurysm exclusion as an alternative to open surgical repair of thoracoabdominal aortic aneurysm. J Vasc Surg 2004;39:454–8.
60. Iguro Y, Yotsumoto G, Ishizaki N, et al. Endovascular stent-graft repair for thoracoabdominal aneurysm after reconstruction of the superior mesenteric and celiac arteries. J Thorac Cardiovasc Surg 2003;125:956–8.
61. Murphy EH, Beck AW, Clagett GP, et al. Combined aortic debranching and thoracic endovascular aneurysm repair (TEVAR) effective but at a cost. Arch Surg 2009;144:222–7.
62. Torsello G, Can A, Umscheid T, et al. Hybrid thoracoabdominal aneurysm repair with simultaneous antegrade visceral revascularization and supra-aortic debranching from the ascending aorta. J Endovasc Ther 2007;14:342–6.
63. Murray D, Ghosh J, Khwaja N, et al. Staged open and endovascular repair of thoracoabdominal aneurysms using the common carotid artery. Ann Vasc Surg 2005;19:103–7.
64. Gawenda M, Brunkwall J. When is it safe to cover the left subclavian and celiac arteries. Part II: celiac artery. J Cardiovasc Surg (Torino) 2008;49:479–82.
65. Waldenberger P, Bendix N, Petersen J, et al. Clinical outcome of endovascular therapeutic occlusion of the celiac artery. J Vasc Surg 2007;46:655–61.
66. Leon LR Jr, Mills JL Sr, Jordan W, et al. The risks of celiac artery coverage during endoluminal repair of thoracic and thoracoabdominal aortic aneurysms. Vasc Endovasc Surg 2009;43:51–60.

67. Donas KP, Czerny M, Guber I, et al. Hybrid open-endovascular repair for thoracoabdominal aortic aneurysms: current status and level of evidence. Eur J Vasc Endovasc Surg 2007;34: 528–33.
68. Lawlor DK, Faizer R, Forbes TL. The hybrid aneurysm repair: extending the landing zone in the thoracoabdominal aorta. Ann Vasc Surg 2007;21:211–5.
69. Lawrence-Brown M, Sieunarine K, van Schie G, et al. Hybrid open-endoluminal technique for repair of thoracoabdominal aneurysm involving the celiac axis. J Endovasc Ther 2000;7:513–9.
70. Yoshida M, Mukohara N, Shida T, et al. Combined endovascular and surgical procedure for recurrent thoracoabdominal aortic aneurysm. Ann Thorac Surg 2006;82:1099–101.
71. Tshomba Y, Bertoglio L, Marone EM, et al. Visceral aortic patch aneurysm after thoracoabdominal aortic repair: conventional vs hybrid treatment. J Vasc Surg 2008;48:1083–91.
72. Chang CK, Chuter TA, Niemann CU, et al. Systemic inflammation, coagulopathy, and acute renal insufficiency following endovascular thoracoabdominal aortic aneurysm repair. J Vasc Surg 2009;49:1140–6.

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Current Treatment of Type B Thoracic Aortic Dissections

P. A. Naughton, M.D., M. Garcia-Toca, M.D., and Mark K. Eskandari, M.D.

INTRODUCTION

Acute thoracic aortic dissection is an uncommon but potentially fatal condition with an incidence of 3 cases per 100,000 patients. Based on the Stanford classification, 60% of aortic dissections are Type A and 40% Type B.[1] Management and outcome differ between Types A and B; therefore in this chapter we focus on Type B dissections.

The pathogenesis of Type B dissection is multifactorial. Risk factors for developing dissection include male gender, arterial hypertension, connective tissue diseases, steroid and cocaine use, bicuspid aortic valve, and iatrogenic and deceleration trauma.[2] Clinical presentation is diverse, ranging from asymptomatic, incidental diagnosis to thoracic aortic rupture. In contrast to Type A dissection, uncomplicated acute Type B dissection has a relatively favorable prognosis, with 90% survival to hospital discharge after receiving antihypertensive therapy.[3] However, when acute Type B dissections are complicated by rupture, end-organ malperfusion, or signs of impending rupture, urgent intervention is indicated.[4] Chronic Type B dissection (survival >14 days after onset of dissection) is an entirely different entity and management options are considered separately.[5]

ACUTE TYPE B DISSECTION

Medical Management

Intensive medical management controlling hypertension and pulse with beta blockers and nitrates and appropriate pain relief are the preferred therapeutic approaches to uncomplicated aortic Type B dissection.[6] Monitoring blood pressure and reducing systolic pressure

to 100 to 120 mmHg in the intensive care setting while maintaining renal perfusion is a priority.[7] One possible exception to this approach are patients with underlying connective tissue disease; this cohort of patients may be considered for prompt operative repair.

Much information on acute Type B dissections regarding management, prognosis, and treatment outcome can be ascertained from the International Registry of Acute Aortic Dissection (IRAD)[8]: 242 consecutive patients presented with acute Type B dissection; 189 were treated medically, 27 were treated with endovascular intervention, and 26 underwent open surgical repair. In-hospital mortality was significantly greater in those patients who underwent open surgical repair (29%, $P<.001$) compared with those treated with endovascular (11%) and medical management (10%). Interestingly, in those who survived, there was no significant mortality differences at 3 years regarding open surgery (76%), endovascular treatment (82%), and medical management (76%). Predictors of follow-up mortality included age 70 yrs, female gender, hypertension, renal failure, atherosclerosis, previous aortic surgery, and patients who presented with signs of rupture or impending rupture.

Endovascular Intervention

Intervention is indicated in patients with intractable pain, signs of impending rupture, or distal malperfusion. In complicated dissections, 30 to 50% of patients have lower limb and visceral ischemia, and 50 to 80% mortality is reported in the absence of prompt intervention.[8] Malperfusion may result from direct extension of the dissection flap into the orifice of a visceral or lower extremity artery (static obstruction) or by the dissection flap's prolapsing into the vessel origin (dynamic obstruction).[9]

Over the last 10 years, endovascular techniques, including stent graft deployment, stenting obstructed aortic side branches, and percutaneous fenestration, have emerged as feasible, less invasive alternatives to open surgical repair for patients with acute, complicated Type B dissections.[10-12] Endovascular intervention obviates the need for aortic cross-clamping, reduces intraoperative blood loss, avoids a thoracotomy and single-lung ventilation, has a more rapid procedural time compared with open surgery, and may be associated with a lower risk for perioperative paraplegia.

Stent Grafts. The primary objective of endografting for acute Type B dissections is to cover the primary entry tear in the hope of obliterating flow in the false-lumen and preferentially directing flow back into the true lumen. In the acute setting, this facilitates end-organ perfusion and controls hemorrhage.[13-14] In the middle to long term, it is anticipated that stent grafts depressurize the false-lumen, thereby promoting false-lumen thrombosis and subsequently diminishing the risk of future aneurysmal dilation. The incidence of false-lumen thrombosis is variable, but when it occurs, it is associated with an improved prognosis.[15]

U.S. Food and Drug Administration Approved-Stent Grafts

GORE TAG Thoracic Endograft (Figures 42–1 and 42–2). This endograft is composed of an ePTFE graft supported by an outer self-expanding nitinol stent. It was the first commercially available thoracic stent graft approved by the U.S. Food and Drug Administration in March 2005. Flexibility makes this stent graft an attractive option in treating challenging, tortuous anatomy. The Gore TAG Pivotal trial was a multicenter, nonrandomized prospective trial comparing treatment of descending thoracic aortic aneurysms with stent grafts versus open repair.[17] Benefits conferred by TAG stent grafts compared with open surgery

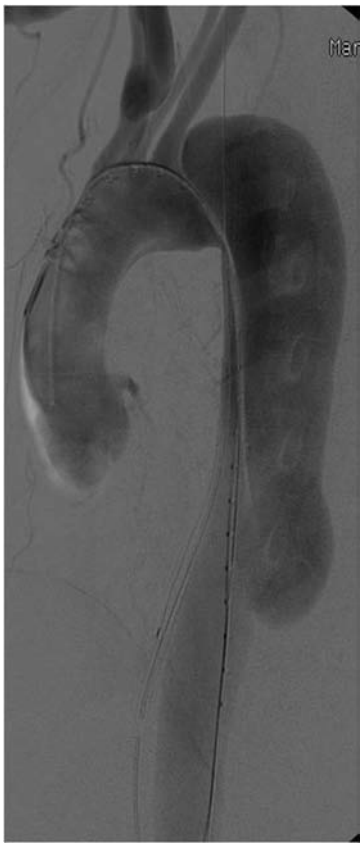

Figure 42-1. Arch aortogram demonstrating a Type B aortic dissection commencing just beyond the left subclavian artery. Note the Pigtail catheter is within the true aortic lumen. There is rapid filling of the false lumen and aneurysm formation of the descending thoracic aorta.

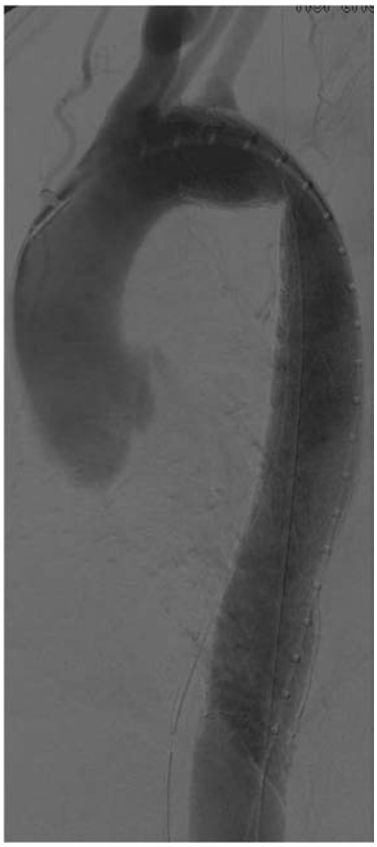

Figure 42-2. Arch aortogram following deployment of a TAG (Gore) stent graft. The proximal landing zone is just beyond the left subclavian artery, with the distal landing zone low in the descending thoracic aorta. There is no evidence of an endoleak and no filling of the false lumen. Note the kink within the proximal aspect of the stent-graft, this was not flow limiting.

included decreased paraplegia or paraparesis (14% vs. 3%), decreased early mortality (10% vs. 2%), and decreased intensive care and hospital stay. On a cautionary note, at 2-year follow-up 15% of patients experienced endoleak. In addition, fracture of the longitudinal support wire was reported in 20 patients. This longitudinal wire was subsequently removed in the redesign of the TAG stent graft. One complication of the redesigned TAG device is stent graft infolding, which may occur when the stent graft is excessively oversized. To date, this device has labeling only for the treatment of thoracic aortic aneurysms. Its use for the treatment of acute/chronic aortic dissections either is part of a clinical trial or is off-label.

Cook Zenith Stent Graft. The TX2 stent graft is a full-thickness woven polyester fabric sewn to self-expanding stainless steel Z-stents with braided polyester and hand-stitched monofilament polyprolene suture. In addition, the Z-stents contain barbs at the distal and proximal ends to augment aortic attachment and seal. FDA approval was based on the results of the TX2 trial comparing 160 patients with descending thoracic aortic aneurysms

and penetrating ulcers treated with TX2 with 60 patients who had open surgical repair.[18] Patients treated endovascularly had significantly less major morbidity at 30 days compared with the open group. At 1 year follow-up in the endovascular group, there were no ruptures or open conversions and the incidence of endoleak and migration was 3.9% and 2.8%, respectively. Areas of ongoing development with Zenith stent grafts include branched devices and fenestrations. Limitations of this device include a multistep delivery system and less flexibility compared with other endografts. The STABLE trial is an ongoing trial investigating a dissection device specific to the treatment of Type B dissections; otherwise, this device currently is labeled for the treatment of thoracic aortic aneursyms or penetrating aortic ulcers.

Medtronic Stent Grafts. The Talent device is composed of a Dacron graft sewn to a self-expanding nitinol stent. The proximal and distal ends contain bare-spring design to facilitate attachment and seal. The use of the Talent device to treat thoracic aortic pathology is reported in the vascular talent thoracic stent graft system for the treatment of thoracic aortic aneurysms (VALOR) trial, a large prospective, multicenter, nonrandomized observational trial.[19] The European experience was reported in the Talent Thoracic Retrospective Registry. In Europe, the Valiant device is a refinement of the Talent device, conferring improved trackability, conformability, and deployment.[20] As is the case with TX2, the Talent thoracic device is currently labeled only for the treatment of aneurysms and penetrating ulcers.

Limitations of Thoracic Aortic Stent Grafting for Dissections

Several limitations of thoracic aortic stent grafting exist.

1. Lack of disease-specific endograft designs enabling flexible delivery with durable seal and attachment.
2. Access restrictions. All commercially available stent grafts require large-caliber delivery systems. Verhoye et al[14] report a 25% incidence of arterial injury requiring surgical repair in patients undergoing thoracic stent graft deployment.
3. Landing zone. A 2 cm proximal and distal landing zone is necessary to achieve fixation. A proximal landing zone may be augmented by additional hybrid procedures, including left carotid-subclavian bypass or transposition.[21] Some also advocate temporarily lowering arterial pressure during stent graft deployment to prevent distal migration of the endograft.[22]
4. Stent graft balloon dilation should be avoided because of the risk of retrograde extension of the dissection converting to a Type A.
5. Risk of stroke. Stroke may occur from embolic events precipitated by guidewire- or catheter-induced intimal trauma. Air embolization at the time of endograft deployment is also a recognized risk for stroke with current devices. Periprocedureal stroke was more commonly seen in first-generational stent graft devices; yet in current device trials, the stroke rate ranged from 3 to 8%.[19]
6. Despite improved results, compared with open repair in-patient mortality remains high for acute Type B dissections. Dias et al[23] report a 16% 30-day mortality, and Verhoye et al[14] report a 25% early mortality.
7. Multiple distal re-entry fenestrations may allow continued perfusion of the false-lumen with aneurysmal dilation. Distal extension of the stent graft to seal these fenestrations increases the risk of spinal ischemia. Some also advocate the PETTICOAT (provisional extension to induce complete attachment) concept of ex-

tending the stent graft scaffold distally with open-cell bare-metal stents.[24] The deployment of bare metal stents or "paving" may also be used as an adjunct to ameliorate flow into the visceral or iliac vessels.

8. Stent-specific complications: endoleaks, migration, stent graft fracture, and infolding. Parker et al,[25] in a review article, report that the incidence of endovascular reintervention for endoleaks in patients with Type B dissections treated with stent grafts is 7.6% and the incidence of open surgical reintervention is 2.8%. Dias et al[23] report a series of 31 patients treated with endografts for acute complicated Type B dissection. They report a 6.5% incidence of stent graft–related late deaths. Verhoye et al[14] report that freedom from treatment failure (aortic rupture, device fault, reintervention, aortic death) was 67% at 5 years. In view of this evidence and also from information from stent graft treatment of infrarenal aneurysms, there is a clear need to complete long-term surveillence of the stent grafts.
9. Patents with an underlying connective tissue disease, such as Marfan syndrome, pose a relative contraindication to stent graft deployment.[4] Although apparently successful stent graft deployment has been reported in this cohort of patients, concern regarding the impact of persistent radial forces of a stent graft on the weak aorta of patients with Marfan syndrome means most surgeons favor open repair if the patient is deemed fit for surgery.

Aortic Fenestration. Surgical aortic fenestration was first described in 1935 and the endovascular technique in 1990.[26] The use of endovascular stent grafts has largely superseded aortic fenestration; however, it remains an important technique in our armamentarium for the urgent treatment of acute malperfusion secondary to dissection. It is particularly useful in treating dynamic obstructing lesions and dissections near the arch that are difficult to treat with stent graft deployment.[12]

The use of intravascular ultrasound is a beneficial adjunct in percutaneous fenestration, enabling accurate differentiation of true and false lumina, positioning of aortic side branches, and re-entry tears. The principle of fenestration entails fashioning a wide orifice of communication between the false and the true lumina.[27]

Various fenestration techniques are described. The most commonly described technique involves puncturing the intimal flap from the true to false-lumen with a trans-septal needle and enlarging the communication with a large-diameter balloon.[28] (Figures 42–3 to 42–6) Multiple fenestrations can be done along the dissection to achieve equalization of pressures between the true and the false lumina. Some interventionalists also place a stent to maintain the fenestration, but this is not our preference because of concern regarding stent collapse or crush. Stents, however, can be used to optimize flow into the branch vessel ostia.

A second approach, the "scissor" technique, involves cannulating the true and false lumina with rigid guidewires.[29] One wire is placed through the proximal tear; this wire is then snared from the opposing lumen and retrieved caudally. Both ends of the wire are then pulled caudally simulataneously to create a "cheese-wire" effect on the intimal flap, thus slicing through the flap to create free communication between the two lumina. It is important that in the absence of a distal re-entry site in the false channel, a distal exit site is fashioned to prevent rupture from high pressures in the false channel.

Complications of fenestration include the risk that the torn intimal flap may occlude the iliac arteries.[30] Also the risk of future aneurysmal dilation of the thin-walled false lumina is a concern for long-term surveillance.

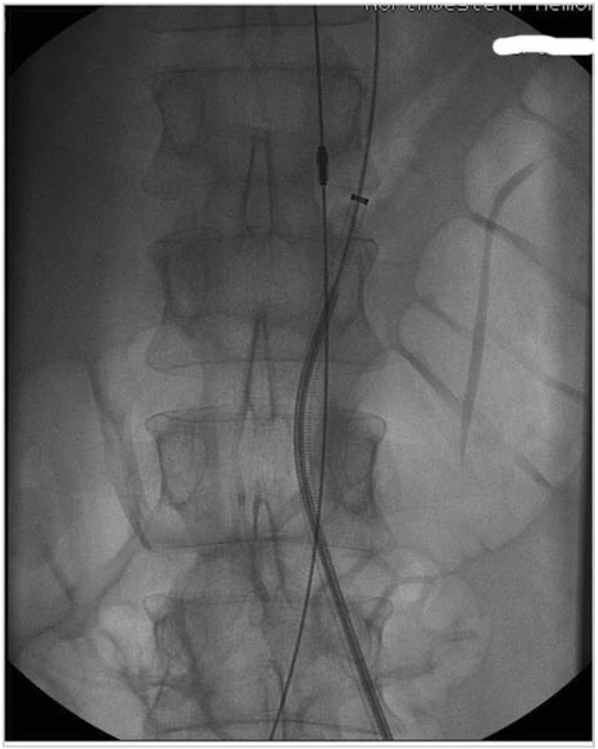

Figure 42-3. Figure a: Initial digital subtraction angiogram demonstrates contrast filling of the true aortic lumen, with contrast opacification of the right renal artery, celiac artery and superior mesenteric artery. Note only vague filling of the false lumen and no filling of the left renal artery.

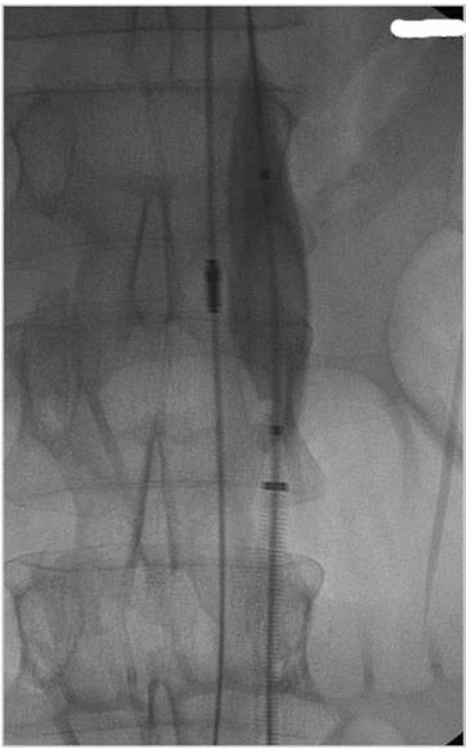

Figure 42-4. Fluoroscopic image showing a guidewire from the right common femoral artery within the true lumen. The guiding catheter, from the left common femoral artery, has crossed from the true lumen into the false lumen, following fenestration with a Rosch-Uchida needle. The guidewire from the guiding catheter is within the false lumen.

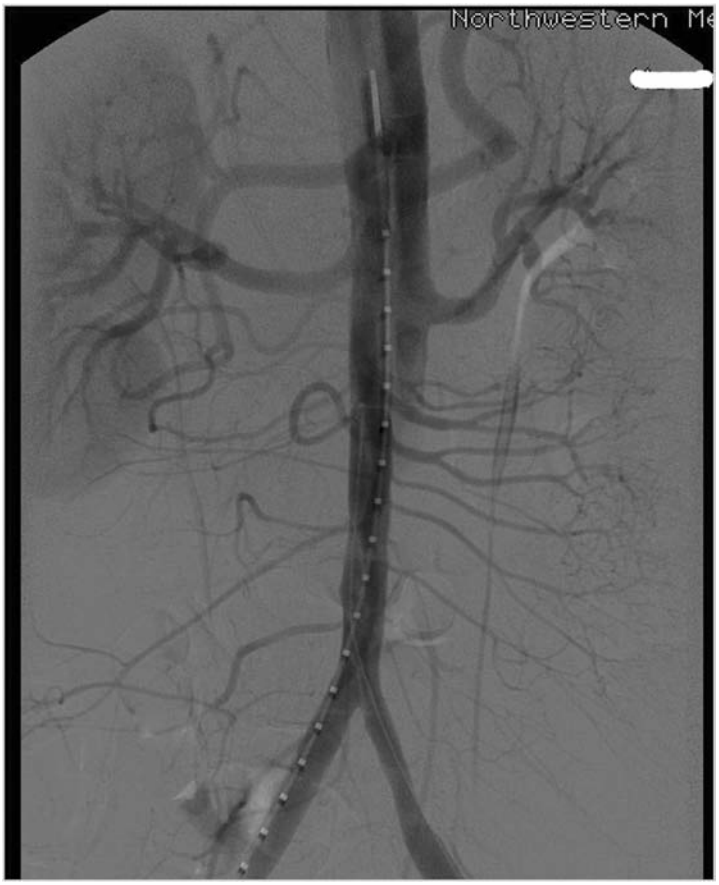

Figure 42-5. Following fenestration a 14 mm x 40 mm balloon was inflated across the site of fenestration to enable creation of a large communication between the true and false lumen in order to equalize pressures between the two lumen and to allow more flow into the false lumen to perfuse any visceral arteries arising from the false lumen.

Uncovered Stents. Uncovered stents can be successfully used to treat static or dynamic obstruction of the aortic side branches or iliac arteries. Stents may be used in isolation or in combination with stent graft deployment or aortic fenestration.

Open Surgical Repair

At present, no randomized trials have compared open and endovascular repair for complicated acute Type B dissections. Table 42–1 contains the recent results of contemporary open repair, but most studies combine the results of acute and chronic presentations and combine differing pathologies, for example, dissection, degenerative aneurysms.

One of the most devastating complications of open thoracoabdominal surgery is ischemic spinal cord injury. Much recent attention has focused on improved outcome by augmenting peri-operative spinal perfusion. Useful adjuncts include cerebrospinal fluid (CSF) drainage and maintaining distal body perfusion by bypass. Coselli et al,[31] in a randomized controlled trial, report that the incidence of postoperative paraplegia

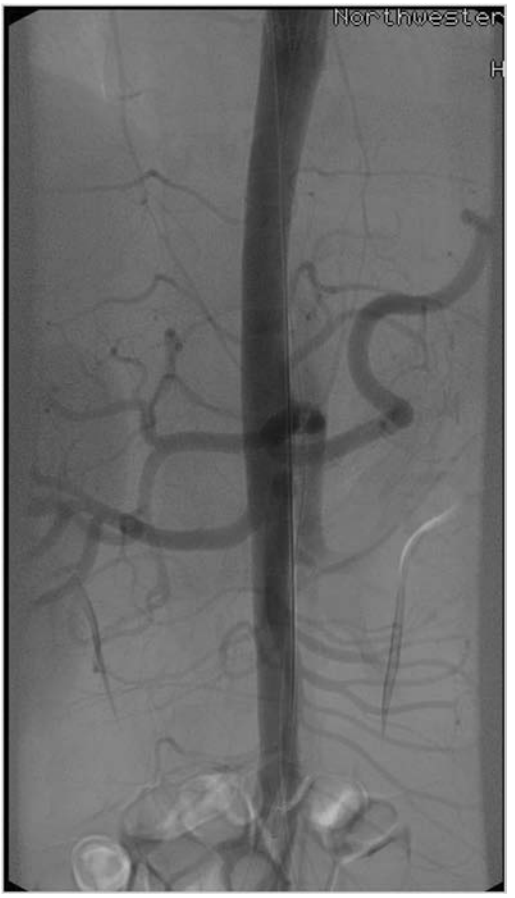

Figure 42-6. Completion angiogram with a graduated Pigtail catheter within the true lumen. Note the left renal artery now fills promptly from the false lumen.

was significantly less in the group treated prophylactically with CSF drainage (2.6% vs. 13%). However, Dardik et al[32] and Wynn et al[33] underline the need for careful monitoring of drainage volume and content with risks of subdural hematoma in cases of excessive drainage.

TABLE 42-1. OPEN SURGICAL REPAIR OF DESCENDING THORACIC AORTA

Author	No of patients	Acute dissection No. (%)	Hospital Mortality	Renal Failure	Paralysis/ paraplegia	Stroke
Estrera[43]	300	18(6)	24 (8)	12 (2.1)	7 (2.3)	6 (2.1)
Coselli[44]	387	48 (12)	11 (4.4)	29 (7.5)	10 (2.6)	
Borst[45]	132	5 (4)	4 (3)	2 (1.3)	12 (9)	
Svensson[46]	832	50 (6)	63 (8)	58 (6.9)	90 (10.4)	29 (3.5)
Verdant[47]	267	33 (12)	39 (15)	1 (0.4)	0	

Chronic Dissection

Chronic thoracic aortic dissection occurs in patients who survive >14 days after acute dissection. Risk of aneurysmal dilation and rupture of the thoracic aorta are the main concerns on long-term follow-up. Management options consist of medical, endovascular, and open surgical management and should be balanced on the basis of the patient's prognosis vs. the risks of intervention. Thoracic aortic aneurysms secondary to dissection are reported to have a more rapid growth rate and increased risk of rupture due to a thinner restraining wall compared with degenerative aneurysms.[34] Growth rates of 0.1 to 0.74 cm per year are reported among patients with chronic dissection. Juvonen et al[35] followed 50 patients with chronic dissection who were operated on when thoracic aortic diameter exceeded 5.5 cm. Despite this aggressive approach to intervention, they reported an 18% mortality rate secondary to rupture. In the absence of level 1 evidence, most surgeons advocate the need for intervention on aneurysms of a diameter of 5.5 to 6 cm.[4] In patients with underlying connective tissue disorder, most surgeons intervene at a diameter of 5 to 5.5 cm. Patients with symptomatic aneurysms presenting with pain, dyspnea, hoarseness, or dysphagia should also be considered for intervention.

Medical Management

Although the medical management of acute uncomplicated dissection has a favorable survival outcome, the long-term results are not so encouraging. The majority of patients are of an advanced age and succumb to comorbidities, but it is estimated that 20 to 50% eventually develop late aortic complications by 4 years.[36] Predictors of late aortic complications include aortic diameter, persistence of flow in the false-lumen, and arterial hypertension.[1] For this reason, strict management of blood pressure is the backbone of medical management of chronic Type B dissections.

The INSTEAD trial[37] (INvestigation of STEnt grafts in patients with Type B Aortic Dissections) is an ongoing prospective, multicenter, randomized trial comparing the outcome of uncomplicated chronic Type B dissections treated medically versus those treated with Medtronic Talent stent grafts. This trial aims to provide evidence regarding the management of uncomplicated chronic dissections. Initial results report a mortality of 10% in the endovascular group compared with 3% in the medical group.

Endovascular Intervention

Similar to acute dissections, the rationale behind endograft treatment of chronic dissections is sealing the proximal entry site, thereby promoting false-lumen thrombosis and aortic remodeling. Spontaneous thrombosis of the false-lumen will occur in <4% of patients.[38]

However, chronic dissection differs anatomically from acute dissections. First, the chronic intimal flap gets progressively thicker due to fibrosis. Second, there are more intimal tears reported in chronic dissections. Although Eggebrecht et al[39] report that operative mortality is less in patients with chronic dissection compared with those with acute dissection, these anatomic characteristics may make endovascular treatment of chronic dissections technically more challenging. Based on these anatomic differences, it is reported that the elimination of flow in the false-lumen is much lower in stent grafting chronic dissections compared with acute dissections.[40] This may increase the rate of reintervention in patients treated endovascularly for chronic dissections.[41]

Sstent grafting chronic dissection is also limited by the fact that any residual, uncovered aorta is at long-term risk for degeneration and rupture.

Open Surgery

In the absence of level 1 evidence, several observational studies compare endovascular with open surgery treating chronic dissections. Nienaber et al[42] prospectively compared 12 patients treated with stent graft deployment with 12 matched surgical controls. Proximal seal and complete lumen thrombosis were achieved in all endovascularly treated patients at 3 months. No major morbidity or mortality was reported in the endovascular group compared with a 33% incidence of death (P=.04) and major morbidity incidence of 42% (P=.04) after open surgery.

CONCLUSION

Acute complicated Type B dissection is one of the most attractive applications of stent graft management. The ongoing INSTEAD trial will provide important information to address the controversy surrounding the treatment of uncomplicated acute Type B dissections. Further improvements in stent graft design and technology may improve results and increase the number of acute and chronic dissections successfully treated by endovascular intervention.

REFERENCES

1. Hagan PG, Nienaber CA, Isselbacher EM, et al. The International Registry of Acute Aortic Dissection (IRAD): new insights into an old disease. JAMA 2000;283:897–903.
2. Larson EW, Edwards WD. Risk factors for aortic dissection: a necropsy study of 161 cases. Am J Cardiol 1984;53:849–55.
3. Estrera AL, Miller CC 3rd, Safi HJ, et al. Outcomes of medical management of acute type B aortic dissection. Circulation 2006;114:1384–9.
4. Svensson LG, Kouchoukos NT, Miller DC, et al. Expert consensus document on the treatment of descending thoracic aortic disease using endovascular stent-grafts. Ann Thorac Surg 2008;85:S1–41.
5. Crawford ES. The diagnosis and management of aortic dissection. JAMA 1990;264:2537–41.
6. Kodama K, Nishigami K, Sakamoto T, et al. Tight heart rate control reduces secondary adverse events in patients with type B acute aortic dissection. Circulation 2008;118:S167–70.
7. Akin I, Kische S, Ince H, et al. Indication, timing and results of endovascular treatment of type B dissection. Eur J Vasc Endovasc Surg 2009;37:289–96.
8. Tsai TT, Fattori R, Trimarchi S, et al. Long-term survival in patients presenting with type B acute aortic dissection: insights from the International Registry of Acute Aortic Dissection. Circulation 2006;114:2226–31.
9. Peterson BG, Eskandari MK. Endovascular repair of descending aortic dissections. Semin Thorac Cardivasc Surg 2005;17:268–73.
10. Dake MD, Miller DC, Semba CP, et al. Transluminal placement of endovascular stent-grafts for the treatment of descending thoracic aortic aneurysms. N Engl J Med 1994;331: 1729–34.
11. Barnes DM, Williams DM, Dasika NL. et al. A single-center experience treating renal malperfusion after aortic dissection with central aortic fenestration and renal artery stenting. J Vasc Surg 2008;47:903–10.

12. Beregi JP, Haulon S, Otal P, et al. Endovascular treatment of acute complications associated with aortic dissection: midterm results from a multicenter study. J Endovasc Ther 2003;10: 486–93.
13. Eggebrecht H, Nienaber CA, Neuhäuser M, et al. Endovascular stent-graft placement in aortic dissection: a meta-analysis. Eur Heart J 2006;27:489–98.
14. Verhoye JP, Miller DC, Sze D, Complicated acute type B aortic dissection: midterm results of emergency endovascular stent-grafting. J Thorac Cardiovasc Surg 2008;136:424–30.
15. Tsai TT, Evangelista A, Nienaber CA, et al. Partial thrombosis of the false lumen in patients with acute type B aortic dissection. N Engl J Med 2007;357:349–59.
16. Bernard Y, Zimmermann H, Chocron S, et al. False lumen patency as a predictor of late outcome in aortic dissection. Am J Cardiol 2001;87:1378–82.
17. Makaroun MS, Dillavou ED, Kee ST, et al. Endovascular treatment of thoracic aortic aneurysms: results of the phase II multicenter trial of the GORE TAG thoracic endoprosthesis. J Vasc Surg 2005;41:1–9.
18. Matsumura JS, Cambria RP, Dake MD, et al. International controlled clinical trial of thoracic endovascular aneurysm repair with the Zenith TX2 endovascular graft: 1–year results. J Vasc Surg 2008;47:247–57.
19. Fairman RM, Criado F, Farber M, et al. Pivotal results of the Medtronic Vascular Talent Thoracic Stent Graft System: the VALOR trial. J Vasc Surg 2008;48:546–54.
20. Fattori R, Nienaber CA, Rousseau H, et al. Results of endovascular repair of the thoracic aorta with the Talent Thoracic stent graft: the Talent Thoracic Retrospective Registry. J Thorac Cardiovasc Surg 2006;132:332–9.
21. Peterson BG, Eskandari MK, Gleason TG, Morasch MD. Utility of left subclavian artery revascularization in association with endoluminal repair of acute and chronic thoracic aortic pathology. J Vasc Surg 2006;43:433–9.
22. Lupattelli T, Garaci FG, Basile A, et al. Emergency stent grafting of type B aortic dissection: technical considerations. Emerg Radiol 2008;15:375–82.
23. Dias NV, Sonesson B, Koul B, et al. Complicated acute type B dissections—an 8-years experience of endovascular stent-graft repair in a single centre. Eur J Vasc Endovasc Surg 2006; 31:481–6.
24. Nienaber CA, Kische S, Zeller T, et al. Provisional extension to induce complete attachment after stent-graft placement in type B aortic dissection: the PETTICOAT concept. J Endovasc Ther 2006;13:738–46.
25. Parker JD, Golledge J. Outcome of endovascular treatment of acute type B aortic dissection. Ann Thorac Surg 2008;86:1707–12.
26. Williams DM, Brothers TE, Messina LM. Relief of mesenteric ischemia in type III aortic dissection with percutaneous fenestration of the aortic septum. Radiology 1990;174:450–2.
27. Williams DM, Andrews JC, Marx MV, et al. Creation of reentry tears in aortic dissection by means of percutaneous balloon fenestration: gross anatomic and histologic considerations. J Vasc Interv Radiol 1993;4:75–83.
28. Chavan A, Hausmann D, Dresler C, et al. Intravascular ultrasound-guided percutaneous fenestration of the intimal flap in the dissected aorta. Circulation 1997;96:2124–7.
29. Beregi JP, Prat A, Gaxotte V, et al. Endovascular treatment for dissection of the descending aorta. Lancet 2000;356:482–3.
30. Lookstein RA, Mitty H, Falk A, et al. Aortic intimal dehiscence: a complication of percutaneous balloon fenestration for aortic dissection. J Vasc Interv Radiol 2001;12:1347–50.
31. Coselli JS, Lemaire SA, Köksoy C, et al. Cerebrospinal fluid drainage reduces paraplegia after thoracoabdominal aortic aneurysm repair: results of a randomized clinical trial. J Vasc Surg 2002;35:631–9.
32. Dardik A, Perler BA, Roseborough GS, et al. Subdural hematoma after thoracoabdominal aortic aneurysm repair: an underreported complication of spinal fluid drainage? J Vasc Surg 2002;36:47–50.
33. Wynn MM, Mell MW, Tefera G, et al. Complications of spinal fluid drainage in thoracoabdominal aortic aneurysm repair: a report of 486 patients treated from 1987 to 2008. J Vasc Surg 2009;49:29–34.

34. Davies RR, Goldstein LJ, Coady MA, et al. Yearly rupture or dissection rates for thoracic aortic aneurysms: simple prediction based on size. Ann Thorac Surg 2002;73:17–27.
35. Juvonen T, Ergin MA, Galla JD, et al. Risk factors for rupture of chronic type B dissections. J Thorac Cardiovasc Surg 1999;117:776–86.
36. Winnerkvist A, Lockowandt U, Rasmussen E, et al. A prospective study of medically treated acute type B aortic dissection. Eur J Vasc Endovasc Surg 2006;32:349–55.
37. Nienaber CA, Zannetti S, Barbieri B, et al. INvestigation of STEnt grafts in patients with type B Aortic Dissection: design of the INSTEAD trial—a prospective, multicenter, European randomized trial. Am Heart J 2005;149:592–9.
38. Erbel R, Oelert H, Meyer J, Puth M, et al. Effect of medical and surgical therapy on aortic dissection evaluated by transesophageal echocardiography. Implications for prognosis and therapy. The European Cooperative Study Group on Echocardiography. Circulation 1993;87:1604–15.
39. Eggebrecht H, Herold U, Kuhnt O, et al. Endovascular stent-graft treatment of aortic dissection: determinants of post-interventional outcome. Eur Heart J 2005;26:489–97.
40. Kusagawa H, Shimono T, Ishida M, et al. Changes in false lumen after transluminal stent-graft placement in aortic dissections: six years' experience. Circulation 2005;111:2951–7.
41. Sayer D, Bratby M, Brooks M, et al. Aortic morphology following endovascular repair of acute and chronic type B aortic dissection: implications for management. Eur J Vasc Endovasc Surg 2008;36:522–9
42. Nienaber CA, Fattori R, Lund G, et al. Nonsurgical reconstruction of thoracic aortic dissection by stent-graft placement. N Engl J Med 1999;340:1539–45.
43. Estrera AL, Miller CC III, Chen EP, et al. Descending thoracic aortic aneurysm repair: 12-year experience using distal aortic perfusion and cerebrospinal fluid drainage. Ann Thorac Surg 2005;80:1290–6.
44. Coselli JS, LeMaire SA, Conklin LD, et al. Left heart bypass during descending thoracic aortic aneurysm repair does not reduce the incidence of paraplegia. Ann Thorac Surg 2004;77:1298–303.
45. Borst HG, Jurmann M, Buhner B, et al. Risk of replacement of descending aorta with a standardized left heart bypass technique. J Thorac Cardiovasc Surg 1994;107:126–33.
46. Svensson LG, Crawford ES, Hess KR, et al. Variables predictive of outcome in 832 patients undergoing repairs of the descending thoracic aorta. Chest 1993;104:1248–53.
47. Verdant A. Descending thoracic aortic aneurysms: surgical treatment with the Gott shunt. Can J Surg 1992;35: 493–6.

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Renal Malperfusion Following Aortic Dissection

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INTRODUCTION

Acute dissection is a common lethal aortic disease.[1-5] Current literature suggests that aortic dissection results in visceral, renal, cerebral, spinal, or limb ischemia in approximately one third of cases, and that peripheral vascular insufficiency increases overall patient morbidity and early mortality.[1-2,6-13] The anatomic and physiologic variables at the foundation of any compromised vascular bed include (1) the percentage of aortic circumference dissected, (2) the presence of a distal reentrant focus in the false lumen or true lumen outflow, and (3) the relationship of branch ostia to the true lumen versus the false lumen.[14-15] However, specific treatment guidelines have yet to be established, and the optimal initial management of these patients remains controversial in terms of the use of a surgical versus an endovascular approach as well as the timing of central aortic repair.

PATHOPHYSIOLOGY

Immediately following dissection, there is "intrinsic true lumen collapse" to a variable degree, the false lumen subsequently dilates, resulting in an increased aortic cross-sectional area. The degree of increase correlates with blood pressure, the depth of the dissection plane within the media (i.e., residual wall thickness), and the percentage of the wall circumference involved in the dissection. Because the false lumen (outer aortic wall) is thinner and elastin-poor, it expands to generate the necessary wall tension required to balance

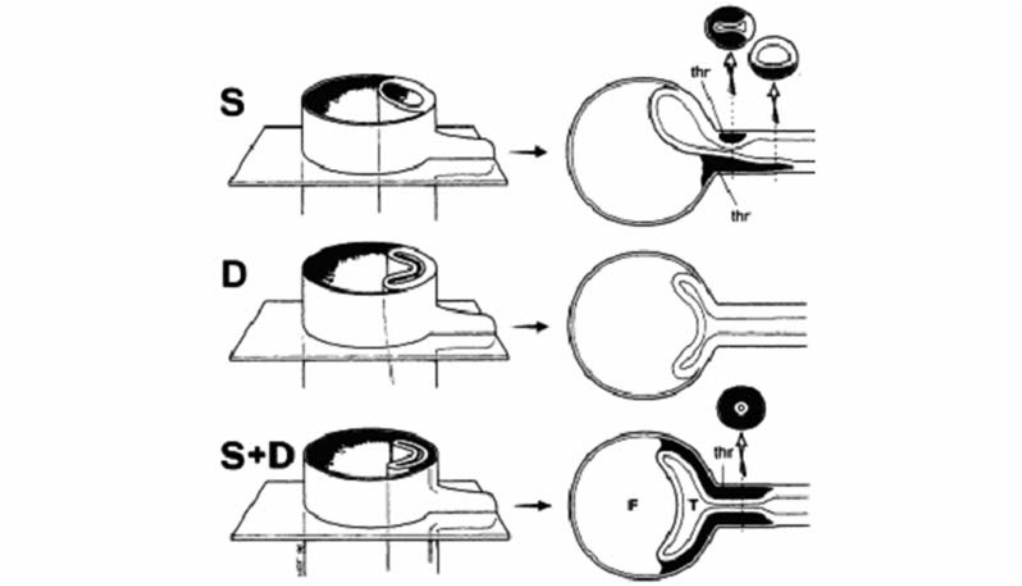

Figure 43-1. Drawing depicting two types of branch vessel obstruction at aortic dissection. In static obstruction (*S*), the dissection flap intersects or enters the branch vessel origin. In dynamic obstruction *(D)*, the mural flap spares the branch vessel wall but prolapses across the branch vessel origin, covering it like a curtain. A mixed-type (static and dynamic) obstruction *(S+D)* is also shown. (Permission for reproduction granted by the Radiological Society of North America [RSNA]: From Williams DM, Lee DY, Hamilton BH, et al. The dissected aorta: Part III. Anatomy and radiologic diagnosis of branch vessel compromise. Radiology 1997;203:37–44.)

a given blood pressure, and the true lumen collapses secondary to the loss of transmural pressure across the dissection flap combined with elastic recoil and shortening of the flap.[16]

Williams et al. classified branch vessel compromise secondary to aortic dissecting hematoma as either static or dynamic[17] (Figure 43–1). Static obstruction occurs when the course of dissection coincides with the origin of a branch vessel and the aortic hematoma propagates into that vessel wall, thereby constricting the lumen. Dynamic obstruction results from prolapse of the dissection flap across the branch vessel origin. Additionally, a dynamic narrowing proximal to a branch ostia may compromise a vessel otherwise spared by the dissection flap. Finally, a mélange of any of these mechanisms may also be present. The distinction between obstruction types clinically (by IVUS and/or arteriography) determines the indicated course of interventional therapy. Although dynamic obstruction has historically been the most common type of obstruction,[15] our series analyzed 71 patients with 104 renal arteries demonstrating obstructions that were classified as 43 static, 30 dynamic, and 22 combined static and dynamic.[18]

CLINICAL PRESENTATION

Renal malperfusion presents clinically as progressive hypertension, evolving renal insufficiency, and/or evidence of impaired blood flow on CT imaging. Renal malperfusion may

complicate both acute and chronic type A and type B dissections. It may also accompany alternate patterns of visceral malperfusion. Interestingly, clinically unsuspected renal malperfusion is not uncommon and can be diagnosed in patients in whom renal malperfusion is not clinically suspected with the aid of intravascular ultrasound, manometry, and angiography.[18] Patients who suffer renal artery obstruction early in the course of aortic dissection but who spontaneously reperfuse the kidney may suffer from unilateral or bilateral acute tubular necrosis (ATN) with no ongoing anatomic abnormality at the time of angiography. In putting numerous clinical papers in context, especially with non-operated type B dissections, it should be noted that "renal dysfunction" does not distinguish between simple ATN, mechanical obstruction by a static or dynamic mechanism, or a combination of the two.

APPROACH TO THERAPY

Miller et al. identified both renal dysfunction and renal/visceral ischemia as significant independent predictors of operative mortality in both acute and chronic type A and B aortic dissections.[19] What is not well established and remains controversial is the optimal treatment strategy for patients suffering aortic dissection complicated by peripheral vascular malperfusion. Some advocate immediate aortic reconstruction in the setting of an acute type A dissection. This is supported by the observation that the majority of cases of peripheral malperfusion (up to 80%) will resolve with restoration of blood through the true lumen.[1-2,8,11] Others, including our own practice, advocate delaying surgery on acute type A dissections in preference for percutaneous correction of the peripheral vascular malperfusion to allow for recovery from reperfusion to reduce overall mortality.[1,7-8,17,20-21] Most will advocate medical management for acute type B dissections, reserving surgery (aortic graft replacement or extra-anatomic bypass) for patients with intractable pain, uncontrolled hypertension, severe aortic branch malperfusion, or aneurysm expansion.[21] Studies have recognized that renal failure with anuria and bowel ischemia in the setting of acute aortic dissection have been associated with lethal multiorgan failure, making resolution of these symptoms a major priority.[9,20] Fann and associates demonstrated that impaired renal perfusion is associated with a high operative mortality rate (50% with renal ischemia compared with 23% for those without compromised renal perfusion) and that both impaired renal perfusion and renal dysfunction were significant independent predictors of operative death.[8,22] These authors maintain that compromised renal perfusion is the only peripheral vascular complication that was a significant independent predictor of operative death.[8]

Recently, Shiiya et al. recognized various mechanisms of malperfusion and found that although a central aortic operation alone successfully reversed all aortic-type malperfusion in acute type A and B dissections (100%), it was not effective for every branch-type malperfusion. Specifically, they noted that surgical fenestration did not successfully reverse branch-type renal malperfusion in all of their patients (2 of 13 patients, or 15%); however, percutaneous stenting was successful in all vessels with branch-type malperfusion.[23]

Finally, Estrera et al. have reinforced that end-organ malperfusion is the most common cause of significant morbidity during the acute presentation of type B aortic dissection, presumably resulting from thrombosis, ischemia-reperfusion injury, or a systemic inflammatory response syndrome.[2,24-25] Additionally, they showed that low glomerular filtration rate was an independent risk factor for mid-term mortality.[25]

STENT GRAFTING

Endovascular stent graft placement at the site of the aortic intimal tear has rapidly evolved as a technique increasingly employed in approaching the dissected aorta in an effort to redirect flow into the true aortic lumen.[1,26-27] In 1999, Dake et al. reported on 19 patients with aortic dissection, 37% of whom suffered symptomatic branch compromise. These authors demonstrated a 100% technical success rate in covering the aortic tear, resulting in resolution of peripheral ischemia in 76% of their cohort. The resolution of peripheral ischemia applied to 22 of 22 patients with dynamic obstruction and 6 of 15 patients with combined static and dynamic obstruction. Since this early report, there have been several additional reports supporting the utility and safety of aortic stent grafts.[26-27] Feezor et al. recently published their experience with thoracic endovascular aortic aneurysm repairs utilizing the TAG device. Fifteen percent of their 216 patient cohort had acute, complicated type B dissections. Eleven of these patients suffered branch vessel malperfusion (75% of which required branch vessel stenting), and 15 underwent endovascular repair for rupture. Of those patients who underwent endovascular repair for rupture, 27% required adjunctive branch vessel stenting. The authors cited a 76% morbidity rate and 21% early mortality rate with this approach.[28] Whereas stent grafting can be quite successful when directed at relieving a dynamic obstruction, the benefit in the setting of a branch-obstructing flap (i.e., static obstruction) remains unclear.

PERCUTANEOUS PROCEDURAL DETAILS

At the University of Michigan, all patients between June 1996 and March 2004 with suspected visceral malperfusion of any type underwent angiographic and IVUS studies. Angiographic evaluation of renal malperfusion is directed at finding and treating an ongoing anatomic renal artery obstruction. IVUS is performed from the ascending aorta to the iliac arteries to define the relationship of the dissection flap to branch arteries, and to determine which lumen each major branch arises from (Figure 43–2). Pressures in the SMA, bilateral renal arteries, and bilateral external iliac arteries are measured simultaneously with pressures in the aortic root. Bilateral renal and superior mesenteric arteriograms with hand injections of contrast are obtained to establish that the location of each measurement is peripheral to the distal extent of the false lumen. Aortic injections are almost never performed, thereby minimizing dye load. True renal malperfusion is confirmed by a systolic gradient between the aortic root and the renal hilum of >10 mm Hg (the threshold at which renal artery stenosis is typically treated by these operators), failure of the artery to fill during injection of contrast in the true and false lumen of the aorta, or evidence of a "curtain-like" occlusion of the vessel origin or the true lumen above the origin by IVUS.

The systematic approach to renal artery compromise at the University of Michigan is corroborated by Beregi and associates' "aortic dissection treatment algorithm" set forth for acute malperfusion complicating acute aortic dissection.[20] As aortorenal gradients are determined by the total obstructive lesion, treatment is directed initially at dynamic obstruction if present. In this case, aortic fenestration is attempted close to the origin of the compromised vessel. The false-lumen pressure in a classic aortic dissection is generally greater than or equal to the true-lumen pressure. Therefore, a fenestration procedure does not reduce pressure in the false lumen but at best raises the

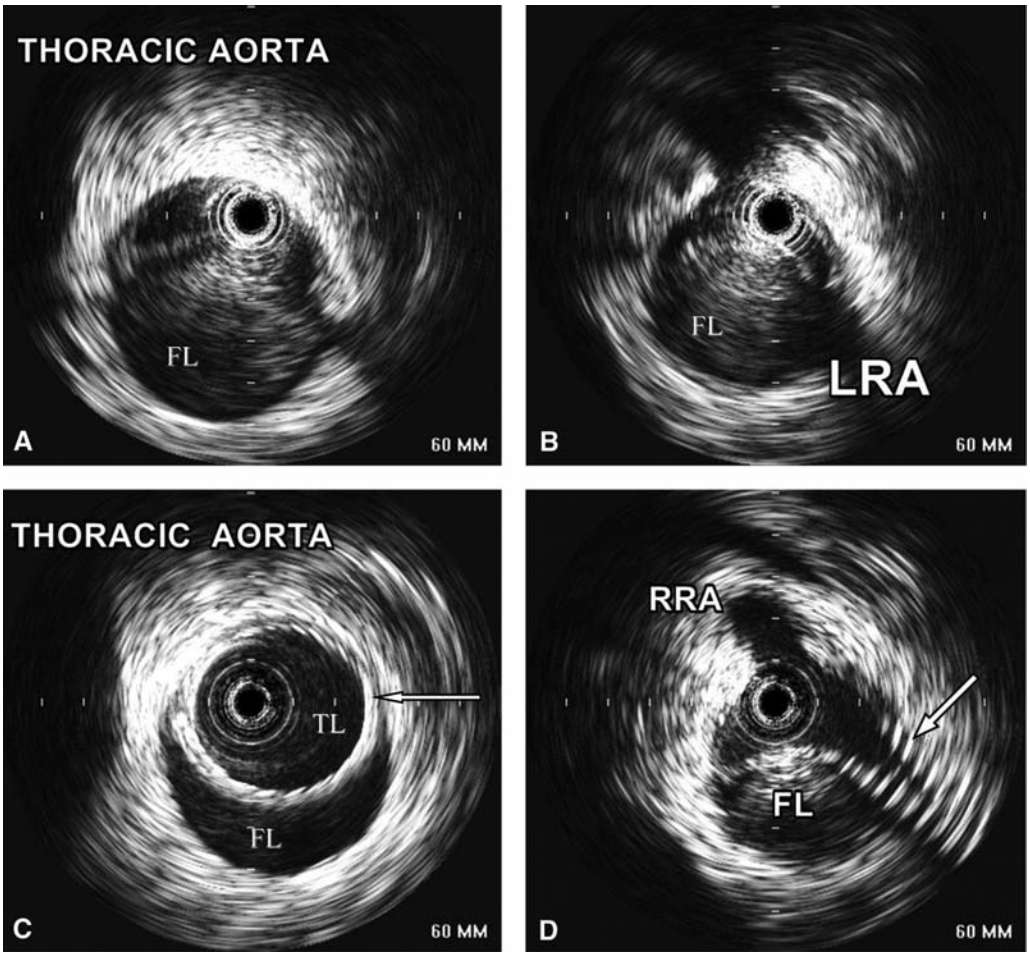

Figure 43-2. Intravascular ultrasound (IVUS) imaging of the thoracic aorta of a patient with an acute type B aortic dissection. A, The false lumen (FL) is hyperechoic and fully distended, obliterating the true lumen (TL) of the aorta except for a slit-like envelope anteriorly. B, In this image of the same patient's left renal artery (LRA), it appears to arise from the FL, but selective arteriography demonstrated that the renal artery was narrowed but remained tethered to the TL. C, IVUS imaging of the thoracic aorta after fenestration and placement of a Wallstent (Boston Scientific). The TL has been stented (arrow), with only some continued mild narrowing in the unstented region across the superior mesenteric and bilateral main renal arteries (not illustrated). After aortic fenestration and aortic stenting, a 17 mm Hg systolic gradient was measured across the renal artery origin, despite a re-entry tear at the origin. D, Final IVUS images of the bilateral renal arteries after aortic fenestration, aortic wall stent, and LRA stenting. Selective stenting of the LRA reduced the systolic gradient to 6 mm Hg. (From Barnes DM, Williams D, Dasika N, et al. A single-center experience treating renal malperfusion after aortic dissection with central aortic fenestration and renal artery stenting. J Vasc Surg 2008;45:903–10.)

true-lumen pressure to match that of the false lumen, thus providing local blood flow across the dissection flap at physiologic pressures.[17]

As an example, if a "curtain-type collapse" of the abdominal aorta is noted at the level of the renal arteries, a fenestration is performed near that level. An Amplatz wire is typically advanced through a Cobra catheter. The catheter is then withdrawn over the wire and exchanged for a Rosch-Uchida introducer set that is subsequently placed

in the true lumen. The wire is removed and the trocar, in its encasing 5-French catheter, is advanced and thrust through the dissection flap using fluoroscopic and IVUS guidance. The trocar is exchanged for the Amplatz wire to allow balloon dilation of the flap and creation of the fenestration tear with a 14-mm diameter balloon. Typically, during balloon dilation, little resistance is encountered and little "waist" is noted. Following creation of the tear, the configuration of the two lumina is observed using IVUS. If the true lumen remains collapsed or, in questionable cases, if a gradient between the root and the abdominal aorta persists, a large-diameter (16 to 22 mm) self-expanding stent is deployed in the aortic true lumen, taking care not to cover the renal artery or SMA origins (see Figure 43–2). Note that compromise of the SMA should be treated before addressing compromise of the renal (or iliac) arteries.

If there is evidence of Cordis PALMAZ balloon-expandable stent or static obstruction, branch vessel stenting should be attempted (Figure 43–3). A Cordis PALMAZ balloon-expandable stent or self-expanding bare stent (i.e., Cook Zilver, Guidant Herculink, Boston Scientfic WALLSTENT or Cordis S.M.A.R.T. stent) is deployed under fluoroscopic and, in select cases, IVUS guidance. The stents are extended further into the aortic lumen (up to 5 to 10 mm) than is necessary when treating atherosclerotic stenoses. Early in our experience, we observed balloon-expanded stents being crushed even by small residual gradients between the true and the false lumina, and we presently use self-expanding stents exclusively.

Reassessment with IVUS and pressure measurements must be performed before terminating the procedure, because occasionally revascularization of a major vessel results in proximal collapse of the aortic true lumen with resultant dynamic obstruction. If dynamic obstruction results secondary to treating a branch artery narrowing, it is treated in standard fashion with fenestration and aortic stenting. Procedural success is confirmed by resolution of true-lumen collapse and elimination of, or at least significant improvement in, aortobranch artery pressure gradients as determined by IVUS, branch arteriography, and manometry. Reasons to defer intervention at the time of initial angiography include dissection or thrombosis extending into the lobar arteries such that distal cannulation of the renal artery's true lumen or other intervention is apt to cause further renal compromise, inability to access accessory branches for stenting, and an aortorenal pressure gradient considered "borderline" and unlikely to result in refractory hypertension or renal insufficiency. These same factors are also the reasons why small residual post-treatment pressure gradients are not always pursued therapeutically.

Our prospective study cohort included 165 patients with aortic dissection (both acute and chronic types A and B). Renal malperfusion was confirmed in 90 patients, 71 of whom underwent endovascular therapy including isolated unilateral or bilateral renal artery stenting (31), proximal aortic fenestration with or without aortic stenting (24), or both renal artery stenting and proximal aortic fenestration with or without aortic stenting[16]. This approach yielded a 90% success rate in resolving the aortorenal gradient (to $<$10 mm Hg) and was associated with five procedure-related complications and a periprocedural mortality rate of 21%.

SUMMARY

Renal malperfusion complicates one third of aortic dissections and increases associated morbidity and mortality. Branch vessel obstruction can be defined as static or dynamic —

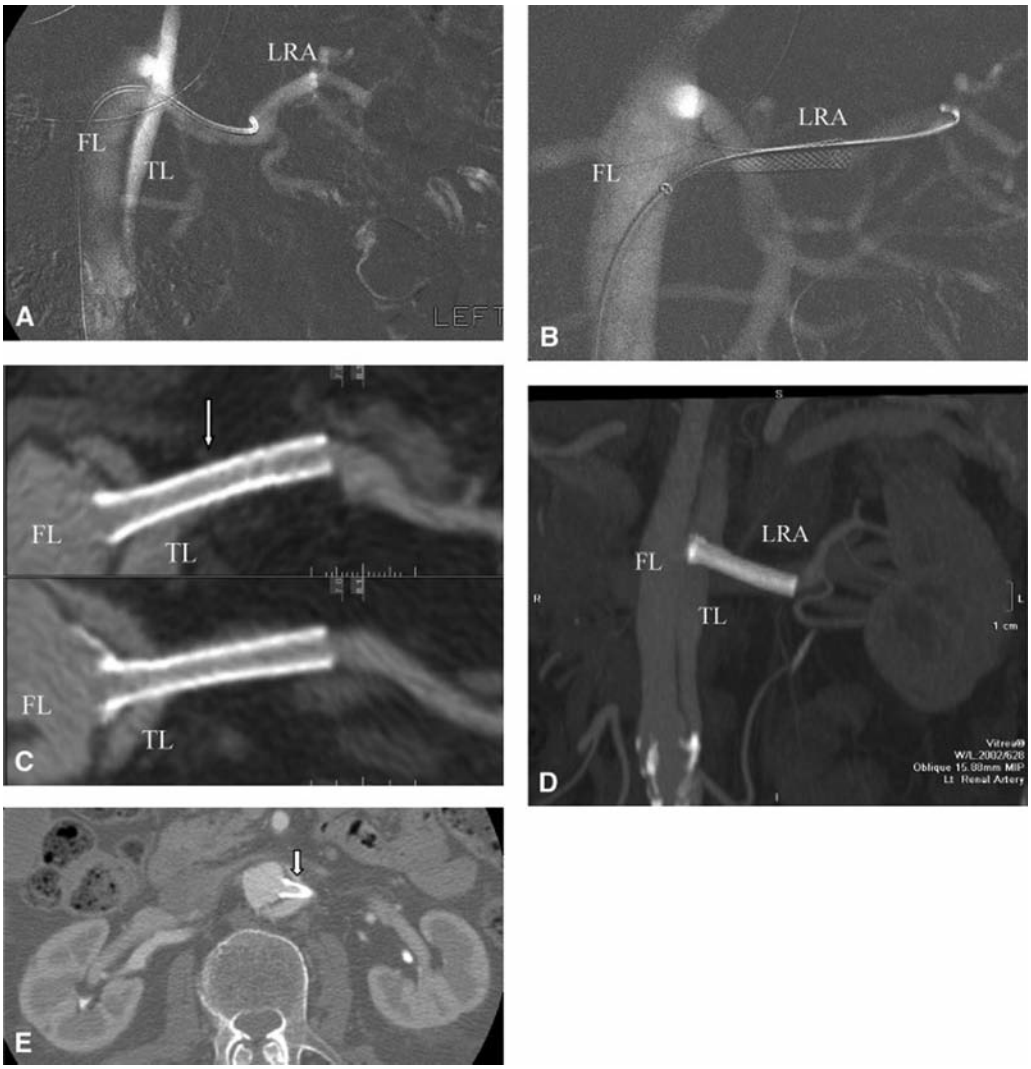

Figure 43-3. A, Carbon dioxide angiogram of a patient with aortic dissection shows static left renal artery (LRA) obstruction and the true lumen (TL), false lumen (FL), and the dissection flap prolapsing into the LRA. Note that the catheter is in the FL. B, Carbon dioxide angiogram of the LRA after fenestration and LRA stenting. C and D, Three-dimensional reformats of the LRA stent (arrow). The true and false aortic lumina are identified. Note the stent extending through the aortic TL. E, Cross-sectional computed tomography image of the same patient at the 4-month follow-up. The arrow is directed at the previously placed LRA stent; note the bright and symmetric left renal contrast enhancement supporting adequate perfusion. (From Barnes DM, Williams D, Dasika N, et al. A single-center experience treating renal malperfusion after aortic dissection with central aortic fenestration and renal artery stenting. J Vasc Surg 2008;47:903–10.)

the distinction is important because it determines the therapeutic approach. Renal artery stenting is appropriate for static obstructions, and proximal central aortic fenestration or endograft repair is appropriate for a dynamic obstruction. It is clear that percutaneous aortic fenestration and renal artery stenting in aortic dissections with renal artery obstruction are technically feasible and adaptable to numerous clinical situations.

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REFERENCES

1. Lauterbach SR, Cambria RP, Brewster DC, et al. Contemporary management of aortic branch compromise resulting from acute aortic dissection. J Vasc Surg 2001;33:1185–92.
2. Girardi LN, Krieger KH, Lee LY, et al. Management strategies for type A dissection complicated by peripheral vascular malperfusion. Ann Thorac Surg 2004;77:1309–14.
3. Pate JW, Richardson RJ, Estridge CE. Acute aortic dissections. Ann Surg 1976;42:395–404.
4. Hirst AE, Johns VJ, Klime SW. Dissecting aneurysm of the aorta: a review of 505 cases. Medicine 1958;37:217–9.
5. Hagan PG, Nienaber CA, Isselbacher EM, et al. The International Registry of Acute Aortic Dissection (IRAD)—new insights into an old disease. JAMA 2000;283:897–903.
6. DeBakey ME, McCollum CH, Crawfor ES, et al. Dissection and dissecting aneurysms of the aorta: twenty-year follow-up of five hundred twenty-seven patients treated surgically. Surgery 1982;92:1118–34.
7. Slonim SM, Nyman U, Semba CP, et al. Aortic dissection: percutaneous management of ischemic complications with endovascular stents and balloon fenestration. J Vasc Surg 1996;23:241–53.
8. Fann JI, Sarris GE, Mitchell RS, et al. Treatment of patients with aortic dissection presenting with peripheral vascular complications. Ann Surg 1990;212:705–13.
9. Cambria RP, Brewster DC, Gertler J, et al. Vascular complications associated with spontaneous dissection. J Vasc Surg 1988;7:199–209.
10. Chavan A, Hausmann D, Dresler C, et al. Intravascular ultrasound-guided percutaneous fenestration of the intimal flap in the dissected aorta. Circulation 1997;96:2124–7.
11. Yagdi T, Atay Y, Engin C, et al. Impact of organ malperfusion on mortality and morbidity in acute A aortic dissections. J Card Surg 2006;21:363–9.
12. Henke PK, Williams DM, Upchurch GR Jr, et al. Acute limb ischemia associated with type B aortic dissection: clinical relevance and therapy. Surgery 2006;140:532–9.
13. Upchurch GR Jr, Nienaber C, Fattori R, Evangelista, et al. Acute aortic dissection presenting with primarily abdominal pain: a rare manifestation of a deadly disease. Ann Vasc Surg 2005;19:367–73.
14. Cambria RP. Surgical treatment of complicated distal aortic dissection. Semin Vasc Surg 2002;15:97–107.
15. Black JH, Cambria RP. Aortic dissection: perspectives for the vascular/endovascular surgeon – pathogenesis of malperfusion syndromes. In: Rutherford RB, ed. Vascular Surgery. 6th ed. Philadelphia: Elsevier Saunders;2005, pp. 1515–17.
16. Williams DM, LePage MA, Lee DY. The dissected aorta: Part I. Early anatomic changes in an in vitro model. Radiology 1997;203:23–31.
17. Williams DM, Lee DY, Hamilton BH, et al. The dissected aorta: Part III. Anatomy and radiologic diagnosis of branch-vessel compromise. Radiology 1997;203:37–44.
18. Barnes DM, Williams D, Dasika N, et al. A single-center experience treating renal malperfusion after aortic dissection with central aortic fenestration and renal artery stenting. J Vasc Surg 2008;47:903–10.
19. Miller DC, Mitchell RS, Oyer PE, et al. Independent determinants of operative mortality for patients with aortic dissections. Circulation 1984;70:I153–64.
20. Beregi JP, Haulon S, Otal P, et al. Endovascular treatment of acute complications associated with aortic dissection: midterm results from a multi-center study. J Endovasc Ther 2003;10:486–93.

21. Hsu RB, Ho YL, Wang SS, et al. Outcome of medical and surgical treatment in patients with acute type B aortic dissection. Ann Thorac Surg 2005;79:790–4.
22. Fann JI, Smith JA, Miller DC, et al. Surgical management of aortic dissection during a 30-year period. Circulation 1995;92:II113–21.
23. Shiiya N, Matsuzaki K, Kunihara T, et al. Management of vital organ malperfusion in acute aortic dissection: proposal of a mechanism-specific approach. Gen Thorac Cardiovasc Surg 2007;55:85–90.
24. Deeb GM, Williams DM, Bolling SF, et al. Surgical delay for acute type A dissection with malperfusion. Ann Thorac Surg 1997;64:1669–75.
25. Estrera AL, Miller CC, Goodrick J, et al. Update on outcomes of acute type B aortic dissection. Ann Thorac Surg 2007;83:S842–5.
26. Dake MD, Kato N, Mitchell RS, Semba et al. Endovascular stent-graft placement for the treatment of acute aortic dissection. N Engl J Med 1999;340:1546–52.
27. Dake MD, Wang DS. Will stent-graft repair emerge as treatment of choice for acute type B dissection? Semin Vasc Surg 2006;19:40–7.
28. Feezor RJ, Martin TD, Hess PJ, et al. Early outcomes after endovascular management of acute, complicated type B aortic dissection. J Vasc Surg 2009;49:561–7.

44

Endovascular Management of Traumatic Thoracic Aortic Injuries

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INTRODUCTION

Blunt thoracic aortic injury (BAI) carries a reported 85% pre-hospital mortality.[1] This injury represents the second most common cause of death due to blunt trauma.[2] A recent review found an overall incidence of BAI of 0.3% in 1.1 million trauma admissions in a 5-year period. Sixty-eight percent of patients who survive transport and triage underwent no repair. The associated mortality rate was 65%.[3] Patients with BAI typically have other significant injuries, including closed-head injury, pulmonary contusion, long-bone and pelvic fractures, and solid organ injury. These concomitant injuries increase the overall morbidity and mortality and also make the use of systemic anticoagulation problematic.[4]

Mortality rates for open repair of BAI range from 5% to 28% and paraplegia rates secondary to spinal cord ischemia range from 2.3% to 14%.[2,4] Definitive open repair is often delayed due to the associated injuries. The delay carries a 2%-13% risk of in-hospital rupture[4,5].

Since the report of Semba et al[6], TEVAR has emerged as an alternative minimally invasive treatment option for BAI. In the last decade there have been a number of case series demonstrating the acute feasibility of endovascular repair for BAI (Table 44–1). With the use of endovascular stent grafts for repair, patients avoid thoracotomy, aortic cross clamping, need for single-lung ventilation and, in some cases, the use of systemic heparinization. (Figure 44–1A,B).

In 1997 the American Association for the Surgery of Trauma published its first prospective multicenter study for the treatment of BAI (AAST1). There were no endovascular interventions and the authors reported an overall mortality of 31% and a post-operative paraplegia rate of 8.7%. The rate of death in patients who did not undergo surgery (not including patients arriving in extremis) was 55%.[15] In 2008, the results of AAST2, a second prospective multicenter study, were reported. The study showed a trend toward selection for endovascular repair (0% in AAST1 versus 64.8%

TABLE 44-1. SERIES WITH MORE THAN 20 PATIENTS REPORTING ENDOVASCULAR REPAIR FOR BLUNT AORTIC INJURY

Author (year)	No.	Age Mean	ISS mean	Technical, success (%)	Mortality (%)	Paraplegia
Canaud[7] (2008)	27	40.2	NR	100	3.7	0
Feezor[8] (2009)	22	34	33	100	0	0
Hoornweg[9] (2006)	28	40.9	37.1	100	14.3	0
Marchiex[10] (2006)	33	38.2	40.2	90.9	0	0
Neschis[11] (2009)	43	44	41	86	34.9	0
Rosenthal[12] (2008)	31	31.4	40	100	6.4	0
Steingruber[13] (2006)	22	39.1	NR	86	0	0
Garcia-Toca[14] (2010)	24	41	43	100	4.2	0

ISS-Injury severity score

in AAST2), a reduction in overall mortality, and a decrease in the incidence of procedure-related paraplegia from 8.7% to 1.6%. Although encouraging results, the authors also noted a 20% device-related complication rate and a 14.4% endoleak rate.[16,17] Additionally, in a recent meta-analysis of stent graft repair for BAI, Tang demonstrated a perioperative mortality of 7.6%, stroke rate of 0.8%, and paraplegia rate of 0%.[18] Similarly, Xenos reported in his analysis a procedure-related mortality of 2%, 30-day mortality of 8%, and paraplegia rate of 0%.[19]

Most endovascular procedures for BAI are performed via a transfemoral approach. We have had success using a completely percutaneous approach using suture-mediated closure systems and the "pre-close" technique. Benefits to percutaneous closure include fewer wound complications, a decreased incidence of cutaneous nerve injuries, and a more expeditious treatment[20,21].

One of the primary concerns of open BAI repair among patients with multiple injuries, particularly closed-head injuries, is the hemorrhagic complication associated

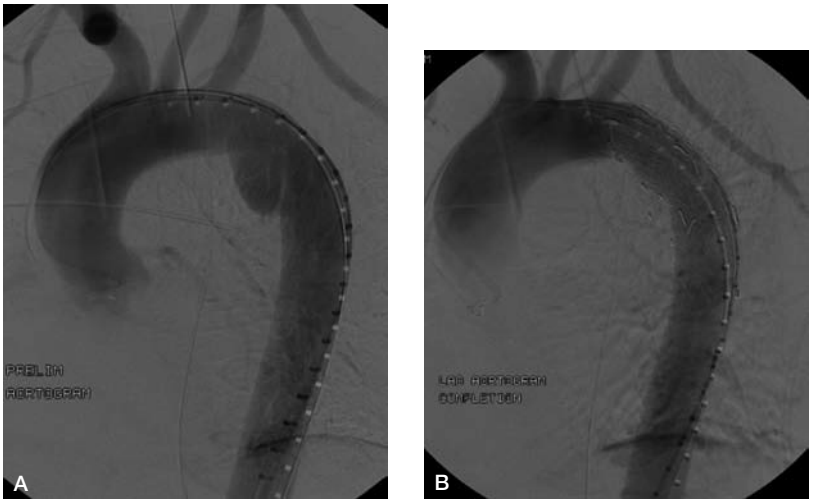

Figure 44-1A. Aortogram, Aortic transection distal to LSCA **B.** Aortogram, Aortic transection treated successfully with an endograft

TABLE 44-2. PATIENT SUMMARY INCLUDING GENDER, AGE, DEVICE TYPE, INJURY SEVERITY SCORE (ISS), PERIPROCEDURAL COMPLICATIONS, HEPARIN USE

Gender	Age	Device	ISS	Complications	Heparin Use
M	51	Excluder	35		
M	40	Excluder	34		Yes
F	40	AneuRx	41		
M	42	Excluder	66		
F	40	Excluder	66		
M	50	Excluder	25		Yes
F	20	Excluder	41		
M	73	Excluder	38		
M	26	Excluder	38		
F	47	Excluder	45	EIA thrombosis	
M	43	Excluder	38		Yes
M	22	TAG	57	Device collapse	Yes
M	27	Excluder	41		
M	51	TAG	57		
M	66	TAG	25		
M	27	Excluder	25		
M	41	Excluder	43		
M	29	Excluder	50	EIA rupture	
M	36	Excluder	45		
F	71	TAG	43		
M	31	Excluder	50		
M	30	Excluder	45		
M	23	Excluder	25		
F	48	Excluder	59		

M - male; F - female; Excluder/Aneurx–proximal aortic extension cuffs; TAG - Gore thoracic endoprosthesis; EIA - external iliac artery,

with systemic intraoperative anticoagulation. While anticoagulation may still be needed when placing thoracic endografts for BAI, lower doses are usually required. Some centers have reported on successful outcomes of endoprosthesis for BAI without the use of systemic anticoagulation[14]. (Table 44–2).

To date, no specific device has been approved for the treatment of BAI. Nevertheless, off-label usage of aortic endografts has been incorporated as part of the armamentarium for treatment of BAI. We first reported on the off-label use of proximal abdominal aortic extension cuffs in the treatment of traumatic aortic rupture several years ago.[22] The advantage of this approach is the more appropriate diameter of these cuffs relative to the true aortic diameter. However, this advantage is counterbalanced by the fact that delivery systems for these cuffs lack sufficient length because the aortic cuffs were originally designed for infrarenal use.

As expected, endoleaks are rather uncommon. In general, trauma patients have a healthy, normal-caliber aorta with an acute tear, and without degenerative aortic pathology. As such, the proximal seal zone of 2 cm, recommended to successfully treat thoracic aortic aneurysms, is probably not necessary when treating patients with BAI. Since the endograft is anchored in relatively normal aorta, there is little concern of

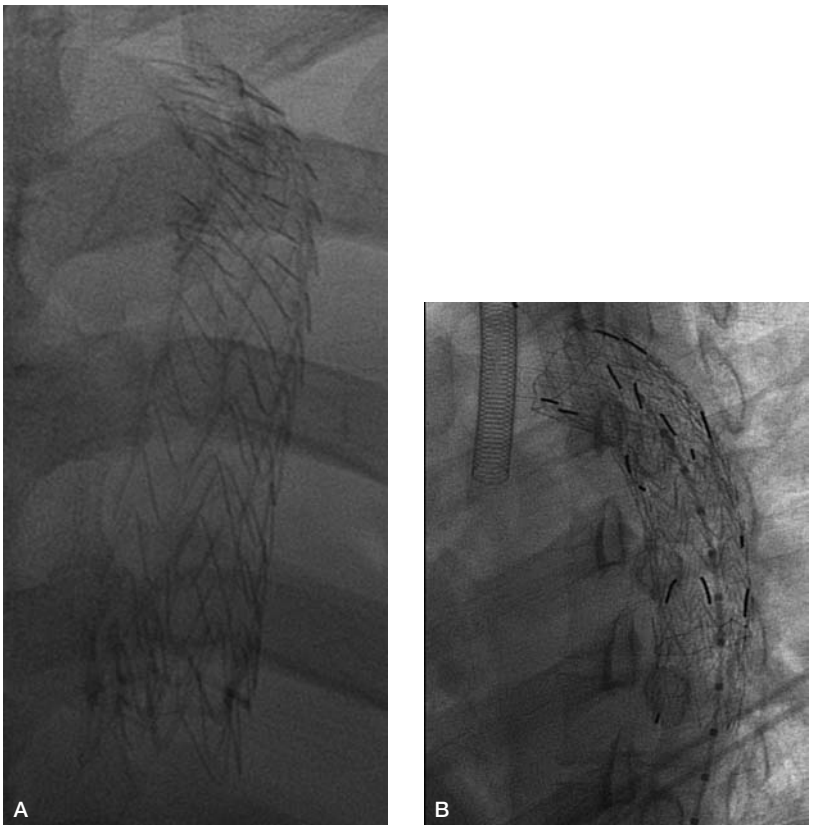

Figure 44-2A. Endograft collapse **B.** Device collapse that was treated successfully with repeat endografting

migration or subsequent aortic neck enlargement. Device collapse has been noted[23–25]. The majority of BAI is observed in a younger patient population. Young patients have a more tightly angulated aortic arch. This factor, in combination with excessive device oversizing relative to the normal aortic diameter, predisposes it to device infolding and failure. (Figure 44–2A,B).

The management of the left subclavian artery is a technical issue that remains a topic of interest. Although some patients tolerate this well, coverage of the LSCA can result in posterior strokes or left upper-extremity ischemia. We favor revascularization of the LSCA via subclavian-carotid artery transposition when preoperative imaging shows there is a possibility of complete LSCA coverage to adequately exclude the injury and a dominant vertebral artery arises from the LSCA.[26]

Studies evaluating long-term outcomes are needed. The patient population with traumatic aortic ruptures is relatively young, and their life expectancy is considerable and exceeds the current experience with endovascular grafts. There is no evidence about the long-term device integrity as well as the natural history of the aorta itself after this type of repair. Because the aorta tends to dilate with age, smaller-sized devices appropriate at the time of implantation may lose fixation over time. However, conversion to open surgical repair at a later date can be performed in a more controlled setting without other associated injuries.

REFERENCES

1. Parmley LF, Mattingly TW, Manion WC, Jahnke EJ Jr. Nonpenetrating traumatic injury of the aorta. *Circulation*. 1958;17:1086–1101.
2. Williams JS, Graff JA, Uku JM, Steinig JP. Thoracic aortic injury in vehicular trauma. *Ann Thorac Surg*. 1994;57:726–730.
3. Neschis DG, Scalea TM, Flinn WR, et al. Blunt aortic injury. *N Engl J Med*. 2008;359:1708–1716.
4. Pierangeli A, Turinetto B, Galli R, Caldarera L, Fattori R, Gavelli G. Delayed treatment of isthmic aortic rupture. *Cardiovasc Surg*. 2000;8:280–283.
5. Arthurs ZM, Starnes BW, Sohn VY , Singh N, Martin MJ, Andersen CA. Functional and survival outcomes in traumatic blunt thoracic aortic injuries: An analysis of the National Trauma Databank. *J Vasc Surg*. 2009;49: 988–994.
6. Semba CP, Kato N, Kee ST, et al. Acute rupture of the descending thoracic aorta: repair with use of endovascular stent-grafts. *J Vasc Interv Radiol*. 1997;8:337–342.
7. Canaud L, Alric P, Branchereau P, Marty-Ane C, Berthet JP. Lessons learned from midterm follow-up of endovascular repair for traumatic rupture of the aortic isthmus. *J Vasc Surg*. 2008;47:733–738.
8. Freezor RJ, Hess PJ, Martin TD et al. Endovascular treatment of traumatic thoracic aortic injuries. *J Am Coll Surg*. 2009;208:510–516.
9. Hoornweg LL, Dinkelman MK, Goslings JC, et al. Endovascular management of traumatic ruptures of the thoracic aorta: a retrospective multicenter analysis of 28 cases in The Netherlands. *J Vasc Surg*. 2006;43:1096 –1102; discussion 1102.
10. Marcheix B, Dambrin C, Bolduc JP, et al. Endovascular repair of traumatic rupture of the aortic isthmus: midterm results. *J Thorac Cardiovasc Surg*. 2006;132:1037–1041.
11. Neschis DG, Moainie S, Flinn W, Scalea TM, Bartlett ST, Griffith BP. Endograft repair of traumatic aortic injury-a technique in evolution. A single institution experience. *Ann Surg*. 2009;250:377–382.
12. Rosenthal D, Wellons ED, Burkett AB, et al. Endovascular repair of traumatic thoracic aortic disruptions with "stacked" abdominal endograft extension cuffs. *J Vasc Surg*. 2008; 48:841– 844.
13. Steingruber IE, Czermak BV, Chemelli A, et al. Placement of endovascular stent-grafts for emergency repair of acute traumatic aortic rupture: a single-centre experience. *Eur Radiol*. 2007;17:1727–1737.
14. Garcia-Toca M, Naughton PA, Matsumura JS, et al. Endovascular repair of blunt traumatic thoracic aortic injuries. Seven-year single center experience. *Arch Surg*. 2010; 145(7):679–683.
15. Fabian TC, Richardson JD, Croce MA, et al. Prospective study of blunt aortic injury: Multicenter Trial of the American Association for the Surgery of Trauma, *J Trauma*. 1997;42: 374–380.
16. Demetriades D, Velmahos GC, Scalea TM, et al. Diagnosis and treatment of blunt thoracic aortic injuries: changing perspectives, *J Trauma*. 2008;64:1415–1418; discussion 1418–1419.
17. Demetriades D, Velmahos GC, Scalea TM, et al. Operative repair or endovascular stent graft in blunt traumatic thoracic aortic injuries: results of an American Association for the Surgery of Trauma Multicenter Study, *J Trauma*. 2008;64:561–570.
18. Tang GL, Tehrani HY, Usman AA, et al. Reduced mortality, paraplegia, and stroke with stent graft repair of blunt aortic transections: a modern meta-analysis. *J Vasc Surg*. 2008; 47:671–675.
19. Xenos E, Abedi N, Davenport D, et al. Meta-analysis of endovascular versus open repair for traumatic descending thoracic aortic rupture. *J Vasc Surg*. 2008;48:1343–1351.
20. Morasch MD. Percutaneous thoracic and abdominal aortic aneurysm repair. *Ann Vasc Surg*. 2005;19:585–9.
21. Peterson BG, Matsumura JS, Morasch MD, West MA, Eskandari MK. Percutaneous endovascular repair of blunt thoracic aortic transection. *J Trauma*. 2005;59:1062–5.

22. Sam A, Kibbe M, Matsumura J, Eskandari MK. Blunt traumatic aortic transaction: endoluminal repair with commercially available aortic cuffs. *J Vasc Surg*. 2003;38(5):1132–1135.
23. Idu MM, Reekers JA, Balm R, et al. Collapse of a stent-graft following treatment of a traumatic thoracic aortic rupture. *J Endovasc Ther*. 2005;12:503–507.
24. Muhs BE, Balm R, White GH, Verhagen HJ. Anatomic factors associated with acute endograft collapse after Gore TAG treatment of thoracic aortic dissection or traumatic rupture. *J Vasc Surg*. Apr 2007;45(4):655–661.
25. Steinbauer MG, Stehr A, Pfister K, et al. Endovascular repair of proximal endograft collapse after treatment for thoracic aortic disease. *J Vasc Surg*. 2006;43:609–612.
26. Peterson BG, Eskandari MK, Gleason TG, Morasch MD. Utility of left subclavian artery revascularization in association with endoluminal repair of acute and chronic thoracic aortic pathology. *J Vasc Surg*. 2006; 43:433–439.

SECTION VI

Visceral Artery Interventions

45

Renal Revascularization: Conventional Surgery versus Endoluminal Catheter-Based Therapy in the United States

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Arteriosclerotic renal artery occlusive disease accounts for 95% of reported cases of renovascular hypertension.[1] It may even be more common because most reported experiences represent surgical series that exclude many older patients who are not operative candidates. Arteriosclerotic renovascular disease most commonly presents during the sixth decade of life. Men are affected twice as often as women. Many of these patients exhibit occlusive disease of the coronary, cerebral, mesenteric, or extremity circulation.[2-5] This is particularly the case in black patients, who exhibit more severe extrarenal arteriosclerotic vascular disease.[6]

These stenoses characteristically affect the proximal third of the vessel in the form of eccentric or concentric narrowings. Nearly 80% of these lesions occur as spillover of diffuse aortic atherosclerosis. These stenotic lesions are bilateral in three-quarters of patients, and lesions affect the right and left renal arteries with equal frequency, although the left renal artery often appears more severely diseased.

Atherosclerotic renal artery stenosis, prior to the introduction of percutaneous endovascular techniques, was treated by open surgical revascularization including renal artery bypass or endarterectomy. The latter were often demanding technical procedures accompanied with modest morbidity and mortality.[7-13] In addition, these procedures were frequently undertaken in the setting of concomitant surgery for aortic aneurysms or aortoiliac occlusive disease with an associated operative mortality ranging 5% to 7%.

Catheter-based management management of arteriosclerotic renal artery occlusive disease has recently gained widespread favor, in part, because of its lesser risk to patients.[14] Although few randomized trials exist comparing percutaneous transluminal angioplasty (PTA) and conventional surgical therapy for renovascular hypertension, an early report by Weibull described similar patency and decreased mortality and morbidity rates for renal PTA compared to open renal revascularization.[15] It is well recognized that some innovations are not always uniformly adopted.[16-18] In fact, variation in the rate of adopting new treatments are largely invisible and very little data is available regarding how new technology is disseminated.[19-20] Insight into the diffusion and effect of catheter-based therapy in the management of arteriosclerotic renal artery disease is important in making accurate predictions about future practice patterns as they affect patient care.

THE INTRODUCTION OF ENDOLUMINAL TREATMENT OF ARTERIOSCLEROTIC RENAL ARTERY DISEASE

In 1978, Gruntzig and colleagues[21] were the first to report the use of PTA in the management of renovascular hypertension. The minimally invasive nature of renal artery PTA offers certain obvious advantages over conventional surgical intervention.[22,23] Most renal artery stenoses can be traversed with a guidewire and subsequently dilated with or without a stent, with minimal morbidity and mortality.[24] The ease of this therapy has led to its rapid introduction into clinical practice, often without clear guidelines as to when it should be used.[25-28] To justify PTA, the clinical significance of the renal artery stenosis should be documented prior to initiating endovascular therapy. In particular, the patient should have sustained hypertension despite simple drug therapy in the case of treatment for elevated blood pressure and adequate renal cortical reserve to recover kidney function when treatment is for renal insufficiency.

The presence of generalized clinically overt arteriosclerotic cardiovascular disease versus focal renal artery disease, as well as aortic spillover arteriosclerosis versus isolated renal artery arteriosclerosis, have an important impact on long-term clinical results. Historically, PTA without stent placement has resulted in a technical success rate of only 70% to 80%. Ostial spillover lesions, treated by PTA alone, have technical success rate of only 30% to 50%. These latter stenoses often manifest excessive recoil and many exhibit acute dissections. As a result of high early post-PTA restenosis rates, stenting of atherosclerotic lesions became appropriate in treating the vast majority of these patients.

Results following renal artery stenting for atherosclerotic disease vary depending on outcome definitions and the indication for intervention, yet many studies have very good results (Table 45–1). For example, Palmaz stents placed in 64 renal arteries in 59 patients, resulted in a two-year secondary patency rate of 92%.[29] Others have documented five-year primary and secondary patency rates of 84% and 92%, respectively.[30] In treating patients for hypertension, long-term benefits have been reported in 52% to 78% of patients. PTA with stenting for progressive ischemic nephropathy is not as effective at reversing renal failure. In these cases, benefits appear related to the degree and duration of ischemic nephropathy prior to PTA, with those having rapid onset of renal failure and a serum creatinine of less than 2 mg/dL demonstrating the best response.

TABLE 45-1. PTA WITH STENT PLACEMENT FOR ARTERIOSCLEROTIC RENOVASCULAR DISEASE

Author	Patients	Stents	Indication		Follow-up Mean (Mo)	Postprocedural Blood Pressure Response (%)*		
			Hypertension	Renal Insufficiency		Cured	Improved	Failed
Dorros-Feuer Foundation	76	92	76	48	6	6	46	48
University Hospital Freiburg, Germany	68	74	68	29	27	16	62	22
Ochsner Clinic	66	88	66		19	2	64	34
Polyclinique D'Essey	59	64	59	10	14	19	57	24
Hotel-Dieu de Montreal	33	35	33	17	13	6	61	33
University of Texas Health Center (Multicenter Study)	28	28	28	14	7	11	48	36

*Outcomes defined in reports from individual institutions.

Complications accompanying renal artery PTA for atherosclerosis are uncommon, with severe complications occurring in less than a few percent of cases. Intimal disruption occurs more often with proximal renal artery dilation where the vessel elasticity is greater and medial disruption is less likely. Medial tears are more common with distal renal artery dilation where vessel elasticity is less. Surgery following failed renal artery PTA is much more hazardous than primary surgery alone[31] because it is associated with a much higher incidence of emergent repair and nephrectomy. Furthermore, blood pressure benefits after a failed renal artery PTA that necessitates secondary operation are significantly lower: 57% after reoperation versus 89% for a primary operation.

CONVENTIONAL SURGICAL TREATMENT OF ARTERIOSCLEROTIC RENAL ARTERY DISEASE

Operative treatment of patients with renovascular occlusive disease has become relatively well defined.[1,32-38] It is important that the primary revascularization procedure be successful. This is underscored by the fact that nephrectomy accompanies nearly half of the reoperations for failed initial reconstructions.[39] Careful preoperative assessment of extrarenal occlusive disease in patients with arteriosclerotic renovascular disease is mandatory to ensure the patient's ability to undergo complex renal artery surgery. Operative details vary and are dependent on the different subgroups of renal artery disease, the involvement of the aorta, and the patients' overall cardiovascular status.

Bypass Procedures

Aortorenal bypass in adults with arteriosclerotic renal artery occlusive disease is most often performed using autologous reversed saphenous vein. Dacron or expanded polytetrafluoroethylene conduits may also be used in reconstructing these vessels. Nonanatomic bypass procedures are important in treating many patients with reno-vascular hypertension. The hepatic artery or iliac arteries may be used as sites of origin for bypass

grafts to the renal artery, especially when originating a graft from the aorta would entail unacceptable risks.[40] Use of the splenic artery in situ for a left-sided splenorenal bypass is appropriate in adults, but only after ascertaining that this vessel and the celiac trunk are free of stenotic disease.[41,42] Splenorenal bypasses are not recommended in children because of the potential existence of a celiac artery growth arrest that may not be evident at the time of reconstruction but which may evolve later.

Endarterectomy

Endarterectomy is often performed for proximal renal artery arteriosclerotic disease.[1,43-45] The two techniques most often used are (1) transaortic renal endarterectomy through an axial aortotomy or the transected infrarenal aorta, and (2) direct renal artery endarterectomy. The extent of aortic and renal artery disease, as well as the need to perform coexistent aortic reconstructive surgery, dictates which of these procedures is most appropriate. In most cases, a linear aortotomy is begun just to the left of the superior mesenteric artery and extended in the midline to below the renal arteries. The diseased aortic intimal and medial tissues are elevated, and with gentle traction, the renal artery atheroma is extracted. This type of endarterectomy is particularly useful in treating bilateral disease or when the disease affects multiple renal arteries. Extensive plaque of the more distal renal artery, especially when involving bifurcations, may be better treated by a direct renal artery arteriotomy and endarterectomy with a patch-graft closure.

Conventional surgical treatment of renovascular hypertension affords excellent outcomes.[1,38] Differences among most individual experiences reflect variations in the prevalence of different renovascular disease categories (Table 45–2). Cures are uncommon and are a reflection of coexistent essential hypertension in older patients with arteriosclerotic disease. Arteriosclerotic renovascular hypertension occurs in two subgroups of patients: (1) those with focal renal artery disease whose only clinical manifestation of arteriosclerosis is secondary hypertension, and (2) those with clinically overt extrarenal arteriosclerosis affecting the coronary and carotid arteries, aorta, or extremity vessels. The severity and duration of hypertension, age, and gender in these two subgroups are similar, yet the surgical outcome regarding amelioration of hypertension is worse in patients with overt extrarenal arteriosclerotic disease. The open surgical treatment of ischemic nephropathy and renal failure is less likely to provide excellent results.[46,47] Similar to outcomes following PTA in these patients, a rapid onset of renal insufficiency and coexistent hypertension provide the best setting for a salutary outcome. Nevertheless, open revascularization of a kidney with an occluded renal artery, unamenable to PTA, offers recovery of renal function and improvement in blood pressure control in nearly half the patients.

THE EVOLUTION AND IMPACT OF ENDOLUMINAL TREATMENT OF ARTERIOSCLEROTIC RENAL ARTERY DISEASE IN THE UNITED STATES

The Nationwide Inpatient Sample is a 20% stratified random sample of all hospital discharges in the United States.[48] Patients studied included those discharged during years 1988 to 2001 with an *International Classification of Diseases, Ninth Revision, Clinical Modification* (ICD-9-CM) code for renovascular hypertension (405.01, 405.11, or 405.91) and

TABLE 45-2. ARTERIOSCLEROTIC RENOVASCULAR HYPERTENSION IN ADULTS

Institution	Patients	Operative Outcome (%)			Surgical Mortality Rate
		Cured	Improved	Failed	
Bowman Gray	152	15	75	10	1.3
University of Michigan					
Focal renal arteriosclerosis	64	33	58	6	0
Overt extrarenal arteriosclerosis	71	25	47	28	8,5
University of California, San Francisco	84	39	23	38	2.4
Cleveland Clinic	78	40	51	9	2
University of Lund, Malmo, Sweden	66	49	24	27	0.9
Hospital Aiguelongue, Montpellier, France	65	45	40	15	1.1
Vanderbilt University	63	50	45	5	9

Modified from Stanley JC. The evolution of surgery for renovascular occlusive disease. *Cardiovasc Surg* 1994;2:195–202.

a concomitant code for renal artery arteriosclerosis (440.1). Patients in this population were then subdivided into two treatment groups. Group I, isolated open renal revascularization, included patients with codes for renal artery revascularization (38.1, 38.10, 38.16, 38.3, 38.30, 38.36, 38.4, 38.40, 38.46, 39.24, 55.4, 55.5, 55.51, 55.52, and 55.54), and without codes for aortoiliac revascularization (38.14, 38.34, 38.44, and 39.25) or aortic aneurysm repair. Group II patients undergoing angioplasty and stenting included those with no surgical revascularization codes and a code for catheter-based revascularization (39.50, 39.59, and 39.90). Exclusion criteria were age less than 20 years and a code for vascular trauma (902.xx).

All 10,320 patients, discharged from 1988 to 2001, were included with a diagnostic code for renovascular hypertension and renal artery arteriosclerosis. Of the 5,433 patients who underwent an intervention, 976 underwent isolated renal revascularization (561 patients undergoing combined aortic and renal revascularization were excluded), and 3,896 underwent renal artery angioplasty and stenting (Table 45–3).

Factors favoring performance of angioplasty and stenting were identified to evaluate patterns in allocation of resources. The primary outcome was in-hospital mortality. Secondary outcomes assessed to ascertain changes in resource utilization included length of stay (LOS), average hospital charge, and unfavorable discharge (to any location other than home).

Univariate analyses, using chi-squared testing, were performed to assess differences over time in rates of angioplasty and stenting, mortality, LOS, hospital charges, and unfavorable discharge. For modeling purposes, calendar years were divided into three time periods: 1988–1992, 1993–1997, and 1998–2001. Race was analyzed as a dichotomous variable: white versus nonwhite. Comorbid diseases were used as a marker of case-mix in accordance with previously established standards.[49-51] True population-based rates were obtained by using sampling weights to find the estimated number of total procedures performed each year in the United States. This

TABLE 45-3. PATIENT DEMOGRAPHICS

Patient Characteristics	Conventional Surgical Revascularization	Angioplasty and Stenting	*P*-Value
Total number of patients	976	3896	
Age (years, mean ± SD)	63 ± 12	67 ± 12	<.001
Female gender	62% (605)	61% (2365)	.038
Nonwhite race	5.4% (35)	10.0% (266)	<.001
Median local annual income rank* (mean ± SD)	2.36 ± 1.07	2.60 ± 1.07	<.001
Urgent admission	18% (161)	28% (1000)	<.001
Emergent admission	9% (79)	18% (642)	<.001
Chronic renal disease	0.8% (8)	0.6% (25)	.767
Diabetes Mellitus	10% (93)	14% (539)	<.001
Chronic obstructive pulmonary disease	9% (84)	8% (296)	<.001
History of myocardial infarction	5% (46)	5% (210)	.276
Mortality	2% (21)	1% (30)	<.001
Unfavorable discharge	17% (161)	9% (346)	<.001

Median local annual income was ranked into four levels: (1) Less than $25,000; (2) $25,000 to $35,000; (3) $35,000 to $45,000; and (4) Greater than $45,000. (Modified from Knipp BS, Dimick JB, Eliason JL, et al. Diffusion of new technology for the treatment of renovascular hypertension in the United States: Surgical revascularization versus catheter-based therapy, 1988–2001. *J Vasc Surg.* 2004;40:717–723).

estimate was then divided by the total adult population for each year to approximate true population-based rates. Multivariate analyses of predictors of catheter-based treatment, mortality, and unfavorable discharge were performed by multiple logistic regression: $P < .05$ was considered significant. SPSS Version 11.0 (Chicago, IL) was used for all statistical analyses.

The number of patients with a discharge diagnosis of renovascular hypertension and renal artery arteriosclerosis increased 46% during the period of study from 1.5 cases to 2.2/100,000 adults ($P < .001$). This may have reflected a change in coding practices or may reflect an increase in the recognition of the disease, with more frequent diagnostic catheterization procedures being performed. Isolated renal revascularization decreased 56%, from 0.2 to 0.09/100,000 adults ($P < .001$). During this same period, angioplasty and stenting increased 173%, from 0.4 to 1.1/100,000 adults (Figure 45–1) ($P < .001$). There was a 67% increase in interventions in general during this time period, from 0.73 to 1.22 / 100,000 adults ($P < .001$).

PTA was more likely to be performed in patients having emergent or urgent admissions, older age, and nonwhite race when comparing isolated renal artery revascularization to catheter-based intervention, (Table 45–4) Comorbidities and gender were not significant predictors of the type of intervention. Catheter-based interventions occurred more frequently from 1993–1997 ($P = .001$) and 1998–2001 ($P < .001$) compared to 1988–1992.

In-hospital mortality did not significantly change over the 14-year period with an overall rate of 2.2% for isolated renal revascularization, and 0.8% for angioplasty and stenting (Figure 45–2). In a multivariate analysis, significant predictors of mortality included increasing age, surgical intervention, emergent admission, and nonwhite race (Table 45–5). Median income, comorbidities, gender, and time period were not significant predictors of mortality.

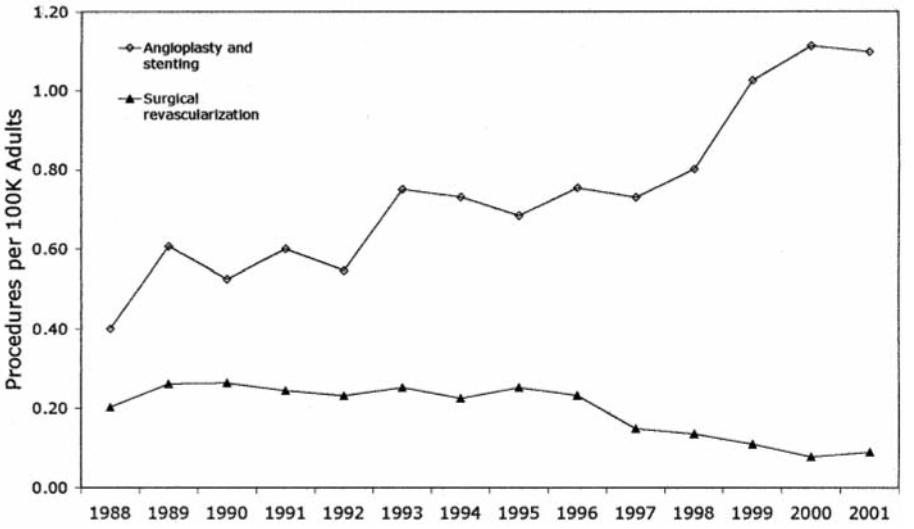

Figure 45-1. A 56% decline in isolated renal revascularization (P < .001) occurred over the 14-year study period. Angioplasty and stenting increased 173% (*P* < .001). (Modifed from Knipp BS, Dimick JB, Eliason JL, et al. Diffusion of new technology for the treatment of renovascular hypertension in the United States: Surgical revascularization versus catheter-based therapy, 1988–2001. *J Vasc Surg.* 2004;40:717–723).

LOS decreased significantly in each treatment group (*P* < .001 for each). At all time points, LOS was shortest for catheter-based interventions (*P* < .001) (Figure 45–3). Hospital charges did not change significantly for surgical interventions over the 14-year period of study, although catheter-based charges increased 61% (*P* < .001). Nevertheless, catheter-based revascularizations had the lowest charges (*P* < .001) (Figure 45–4).

In each treatment group, rates of unfavorable discharges to any location other than home (excluding in-hospital mortality) increased significantly with time (*P* = .004) and *P* < .001 for isolated renal and catheter-based revascularizations, respectively (Figure 45–5). In a multivariate analysis, predictors of an unfavorable discharge included surgical intervention, increasing age, admission acuity, nonwhite race, and female gender (Table 45–6).

TABLE 45-4. MULTIVARIATE ANALYSIS OF PREDICTORS OF ANGIOPLASTY OR STENTING: COMPARISON BETWEEN ISOLATED RENAL ARTERY RECONSTRUCTION VERSUS ANGIOPLASTY AND STENTING.

Independent Variable	Predictors of Angioplasty or Stenting, Odds Ratio (95% CI)	*P*-Value
Emergent admission	3.5 (2.7 to 4.7)	<.001
Urgent admission	2.2 (1.8 to 2.8)	<.001
Age ≥77*	2.5 (1.9 to 3.3)	<.001
Age 71 to 76*	1.5 (1.2 to 1.9)	.001
Nonwhite race	1.7 (1.2 to 2.4)	.003

*Compared to age ≤ 60 years. Modified from Knipp BS, Dimick JB, Eliason JL, et al. Diffusion of new technology for the treatment of renovascular hypertension in the United States: Surgical revascularization versus catheter-based therapy, 1988–2001. *J Vasc Surg.* 2004;40:717–723).

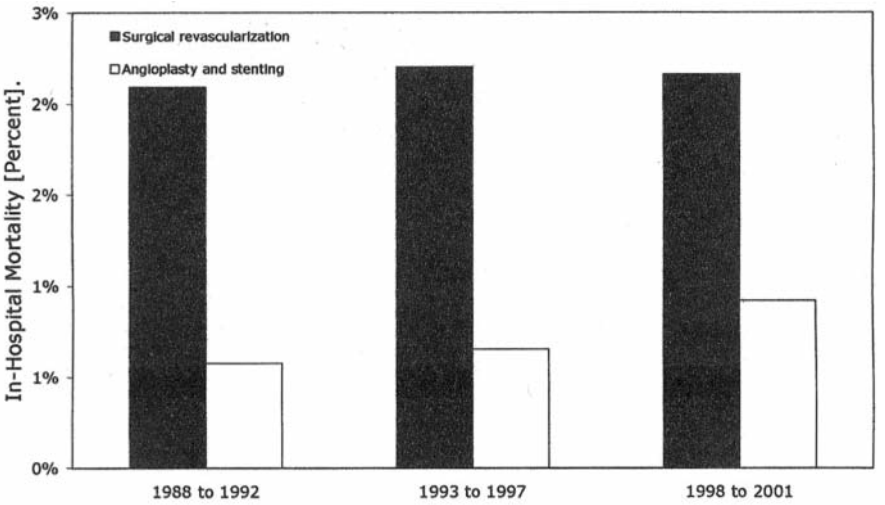

Figure 45-2. In hospital mortality versus time. By chi-square analysis, there were no significant variations in mortality over time for either treatment class. (Modified from Knipp BS, Dimick JB, Eliason JL, et al. Diffusion of new technology for the treatment of renovascular hypertension in the United States: Surgical revascularization versus catheter-based therapy, 1988–2001. *J Vasc Surg.* 2004;40:717–723).

DISCUSSION

Renovascular hypertension secondary to arteriosclerotic renal artery occlusive disease is the most common form of correctable hypertension. The proper selection of patients for interventional therapy is most important, be it for a conventional surgical reconstructive procedure or for endovascular percutaneous transluminal angioplasty. Controversy exists regarding both the appropriate means of establishing a diagnosis of renin-mediated, renovascular hypertension as well as the best method of therapy once the disease is recognized.[22,26-28,36] Nevertheless, the use of multiple criteria to identify occlusive renal artery disease and the ease of newer catheter-based interventions with less early morbidity has caused dramatic changes in treating patients with renovascu-lar hypertension.[14] The impact of this new technology is of importance to both health care agencies and physician providers.

TABLE 45-5. MULTIVARIATE ANALYSIS OF MORTALITY: INDEPENDENT PREDICTORS OF IN-HOSPITAL MORTALITY FOLLOWING INTERVENTION FOR RENOVASCULAR HYPERTENSION

Independent Variable	Risk of Mortality, Odds Ratio (95% CI)	*P*-Value
Age ≥75 years*	10.4 (2.3 to 46.0)	.002
Age 68 to 74 years*	6.5 (1.5 to 28.7)	.014
Age 60 to 67 years*	7.2 (1.6 to 32.1)	.009
Conventional renal artery surgery**	4.1 (1.9 to 8.7)	<.001
Emergent Admission	3.9 (2.1 to 7.4)	<.001
Nonwhite race	3.1 (1.5 to 6.7)	.004

*Compared to age ≤59 years

**Compared to angioplasty or stenting procedures

Modified from Knipp BS, Dimick JB, Eliason JL, et al. Diffusion of new technology for the treatment of renovascular hypertension in the United States: Surgical revascularization versus catheter-based therapy, 1988–2001. *J Vasc Surg.* 2004;40:717–723)

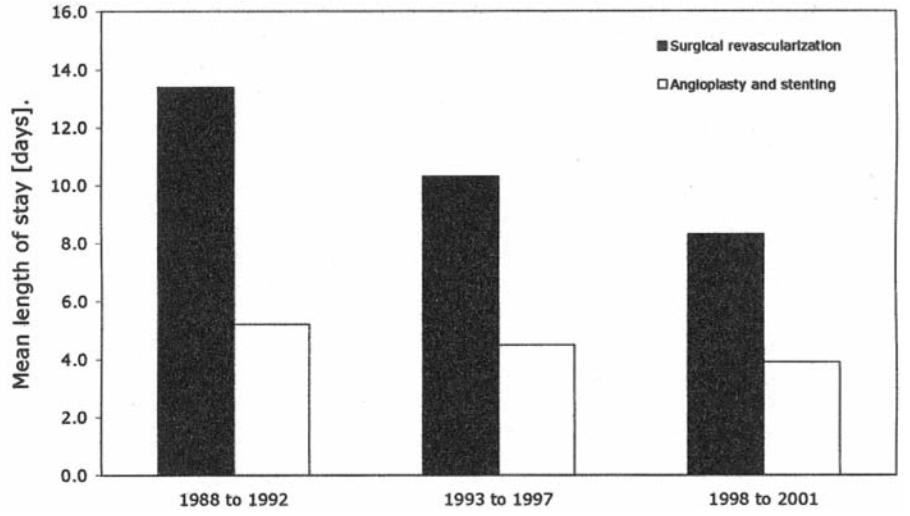

Figure 45-3. Each treatment class exhibited a trend towards decreasing LOS ($P < .001$ for each group). At all timepoints, isolated renal revascularization had a longer LOS than angioplasty and stenting ($P < .001$). (Modified from Knipp BS, Dimick JB, Eliason JL, et al. Diffusion of new technology for the treatment of renovascular hypertension in the United States: Surgical revascularization versus catheter-based therapy, 1988–2001. *J Vasc Surg.* 2004;40:717–723).

The number of patients discharged from U.S. hospitals with a diagnosis of renal artery arteriosclerosis and renovascular hypertension has increased 43% from 1988 to 2001.[14] During this same period, while there was only a 17% increase in the overall number of individuals over the age of 65—from 30 million in 1988 to 35 million in

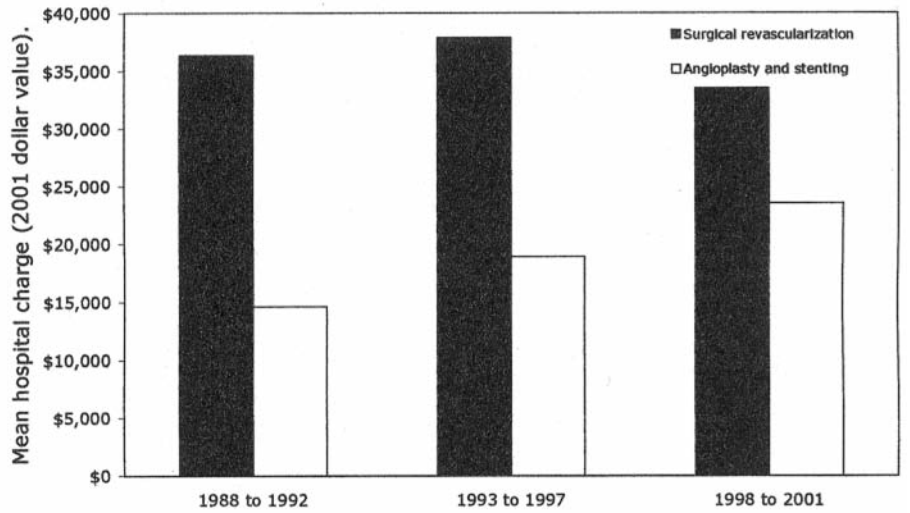

Figure 45-4. There were no significant trends in total hospital charges for isolated renal surgical repairs. Charges for angioplasty and stenting increased significantly ($P < .001$), approaching charges for isolated surgical renal revascularization. All costs were corrected for 4% annual inflation. (Modified from Knipp BS, Dimick JB, Eliason JL, et al. Diffusion of new technology for the treatment of renovascular hypertension in the United States: Surgical revascularization versus catheter-based therapy, 1988–2001. *J Vasc Surg.* 2004;40:717–723).

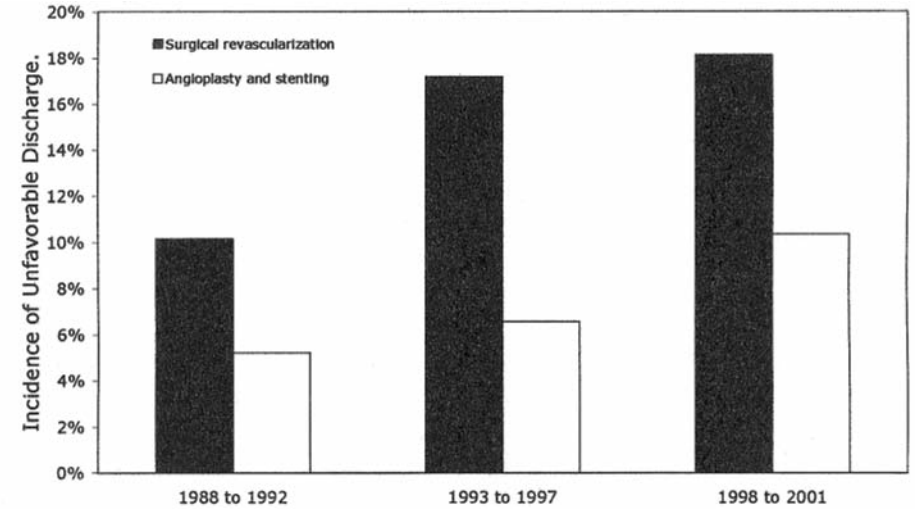

Figure 45-5. Each treatment class exhibited a significant trend towards an increased risk of unfavorable discharge (P=.004 for isolated renal revascularization and P < .001 for angioplasty and stenting). (Modified from Knipp BS, Dimick JB, Eliason JL, et al. Diffusion of new technology for the treatment of renovascular hypertension in the United States: Surgical revascularization versus catheter-based therapy, 1988–2001. *J Vasc Surg.* 2004;40:717–723).

2001—the proportion of patients undergoing renal revascularization has increased 67%. Importantly, the type of intervention has also dramatically shifted from an open surgical revascularization to a catheter-based intervention. In addition, percutaneous catheter-based interventions are increasingly associated with an older population and a higher disease acuity. The association of nonwhite race to increased likelihood of catheter-based intervention may result from demographic characteristics of populations near tertiary care centers where this technology is accessible.

TABLE 45-6. MULTIVARIATE ANALYSIS OF UNFAVORABLE DISCHARGE: INDEPENDENT PREDICTORS OF DISCHARGE OTHER THAN TO HOME EXCLUDING IN-HOSPITAL MORTALITY FOLLOWING INTERVENTION FOR RENOVASCULAR HYPERTENSION

Independent Variable	Risk of Mortality, Odds Ratio (95% CI)	*P*-Value
Conventional renal artery surgery*	4.3 (3.2 to 5.9)	<.001
Age ≥75**	5.2 (3.5 to 7.6)	<.001
Age 68 to 74**	2.9 (2.0 to 4.3)	<.001
Age 60 to 67**	1.8 (1.2 to 2.8)	.004
Emergent admission	3.6 (2.7 to 4.8)	<.001
Urgent admission	1.4 (1.1 to 1.8)	.019
Nonwhite race	1.6 (1.1 to 2.3)	.022
Female gender	1.3 (1.1 to 1.7)	.014

*Compared to angioplasty and stenting procedures

**Compared to age ≤ 59 years

Modified from Knipp BS, Dimick JB, Eliason JL, et al. Diffusion of new technology for the treatment of renovascular hypertension in the United States: Surgical revascularization versus catheter-based therapy, 1988–2001. *J Vasc Surg.* 2004;40:717–723.

Hospital charges have increased in the catheter-based treatment group, reflecting the greater utilization of this technology, and are approaching inpatient charges for isolated renal revascularizations.[14] It is notable that these charges do not include radiographic studies commonly performed in follow-up of these patients. In addition, these charges do not include those accompanying late endovascular failures. This suggests that the potential exists for the cost of catheter-based therapy of renal artery arteriosclerosis to eclipse that of conventional surgical repair.

Length of stay has decreased significantly in each treatment group, despite trends toward poorer outcomes.[14] It is possible that the poorer outcomes observed in more recent years reflected increasing technical savvy and application of these technologies to sicker patients, a likelihood supported by the fact that the Romano-Charleson comorbidity index increased significantly over the time period of the study (data not shown).

A confounding factor in length of stay analysis was the lack of information on length of time from admission to procedure. It was assumed that all procedures in this study occurred at admission.[52]

Conventional surgical renal revascularization procedures carry the potential for significant morbidity and mortality, especially when combined with aortic reconstructions. Mortality in this setting was 5.2% for patients treated from 1988 to 2001.[14] The mortality of isolated catheter-based intervention during that time was 0.8%, less than the 2% associated with open surgical repair.[14] Nevertheless, serious complications may be associated with angioplasty and stenting, perhaps related to the treatment of patients with more extensive renal artery pathology. Perhaps better preintervention data would be helpful in determining which patients are most likely to benefit.[28] A recent study by Sharafuddin documented the use of the resistive index as a good predictor of who would most likely benefit from angioplasty and stent placement for renal artery stenosis.[53] It is incumbent on the physician to carefully evaluate each particular patient and choose therapy appropriate for that individual.

Administrative database limitations are offset in studies such as the current one by strengths such as large patient volumes, hard clinical endpoints like mortality, and an opportunity to evaluate practice trends across all levels of practice. However, administrative databases are poor in providing long term follow-up and efficacy of therapy. For example, data regarding reintervention rates for endovascular treatment of renal artery stenosis is notoriously lacking.

Many medical specialties, particularly surgery, have witnessed an accelerating introduction of new technology over the past decade. The quick pace of change has affected vascular surgery in particular, with the increasing application of endovascular therapy for many common vascular diseases. Percutaneous angioplasty and stenting to treat peripheral occlusive disease, placement of endovascular grafts to treat abdominal aortic aneurysms, and, most recently, angioplasty and stenting for treating carotid stenosis has profoundly changed the specialty of vascular surgery.[19,29,54]

Previous studies have documented increased utilization of percutaneous angioplasty and stenting for the treatment of peripheral occlusive disease. Tunis and colleagues demonstrated that despite an increase in use of such less invasive techniques, the rate of peripheral bypass surgery increased.[19] It is not uncommon to observe an increase in the overall number of procedures being done after the dissemination of the less invasive technology, as witnessed by laparoscopic procedures becoming commonplace therapy for many nonvascular diseases. The same phenomena appear to occur in the treatment of renovascular hypertension and renal artery stenosis where

there has been a moderate increase in diagnosis but an explosion in the number of procedures, primarily endovascular to treat this disease.

CONCLUSIONS

Diffusion of innovations has been widely studied by others who have provided insight into apparent variations accompanying the adoption of new technology. A new medical treatment tends to spread at varying rates depending on certain attributes of the innovation including its *relative advantage* compared to traditional treatment. Nonsurgeons and teaching hospitals, more than others, appear to have embraced this newer technology. The less invasive catheter-based treatment of renal artery stenosis with its significantly improved short-term outcomes, especially with use of stents compared to renal artery bypass and endarterectomy, is very attractive to clinicians.[55-58] Such short-term outcomes, even more so than long-term outcomes, are likely to influence the lowering of the threshold to treat a patient by endovascular means. The cost benefits in the short term may not persist in the long term, and only further follow-up of patients treated in a randomized study will define this aspect of endoluminal therapy of renovascular hypertension.

REFERENCES

1. Stanley JC. Surgical treatment of renovascular hypertension. *Am J Surg*. 1997;174:102–110.1.
2. Louie J, Isaacson JA, Zierler RE, Bergelin RO, Strandness DE Jr. Prevalence of carotid and lower extremity arterial disease in patients with renal artery stenosis. *Am J Hypertens*. 1994; 7:436–439.
3. Missouris CG, Buckenham T, Cappuccio FP, MacGregor GA. Renal artery stenosis: a common and important problem in patients with peripheral vascular disease. *Am J Med*. 1994; 96:10–14.
4. Valentine RJ, Clagett GP, Miller GL, et al. The coronary risk of unsuspected renal artery stenosis. *J Vasc Surg*. 1993;18:433–439.
5. Valentine RJ, Martin JD, Myers SI, et al. Asymptomatic celiac and superior mesenteric artery stenoses are more prevalent among patients with unsuspected renal artery stenoses. *J Vasc Surg*. 1991;14:195–199.
6. Novick AC, Zaki S, Goldfarb D, Hodge EE. Epidemiologic and clinical comparison of renal artery stenosis in black patients and white patients. *J Vasc Surg*. 1994;20:1–5.
7. Allen BT, Rubin BG, Anderson CB, et al. Simultaneous surgical management of aortic and renovascular disease. *Am J Surg*. 1993;166:726–732.
8. Cherr GS, Hansen KJ, Craven TE, et al. Surgical management of atherosclerotic renovascular disease. *J Vasc Surg*. 2002;35:236–245.
9. Darling RC 3rd, Shah DM, Chang BB, Leather RP. Does concomitant aortic bypass and renal artery revascularization using the retroperitoneal approach increase perioperative risk? *Cardiovasc Surg*. 1995;3:421–423.
10. Erdoes LS, Berman SS, Hunter GC, Mills JL. Comparative analysis of percutaneous transluminal angioplasty and operation for renal revascularization. *Am J Kidney Diseases*. 1996; 27:496–503.
11. Kulbaski MJ, Kosinski AS, Smith RB 3rd, et al. Concomitant aortic and renal artery reconstruction in patients on an intensive antihypertensive medical regimen: long-term outcome. *Ann Vasc Surg*. 1998;12:270–277.
12. Poulias GE, Skoutas B, Doundoulakis N, et al. Surgical treatment of renovascular hypertension and respective late results. A twenty years experience. *J Cardio Surg*. 1991;32:69–75.

13. van Bockel JH, van Schilfgaarde R, Felthuis W, et al. Influence of preoperative risk factors and the surgical procedure on surgical mortality in renovascular hypertension. *Am J Surg*. 1988;155:770–775.
14. Knipp BS, Dimick JB, Eliason JL, et al. Diffusion of new technology for the treatment of renovascular hypertension in the United States: Surgical revascularization versus catheter-based therapy, 1988–2001. *J Vasc Surg*. 2004;40:717–723.
15. Weibull H, Bergqvist D, Bergentz SE, et al. Percutaneous transluminal renal angioplasty versus surgical reconstruction of atherosclerotic renal artery stenosis: A prospective randomized study. *J Vasc Surg*. 1993;18:841–852.
16. Berwick DM. Disseminating innovations in health care. *JAMA*. 2003;289:1969–1975.
17. Gelijns AC, Fendrick AM. The dynamics of innovation in minimally invasive therapy. *Health Policy*. 1993;23:153–166.
18. Tunis SR, Gelband H. Health care technology in the United States. *Health Policy*. 1994;30: 335–396.
19. Tunis SR, Bass EB, Steinberg EP. The use of angioplasty, bypass surgery, and amputation in the management of peripheral vascular disease. *N Engl J Med*. 1991;325:556–562.
20. Tunis SR, Bass EB, Klag MJ, Steinberg EP. Variation in utilization of procedures for treatment of peripheral arterial disease. A look at patient characteristics. *Arch Intern Med*. 1993; 153:991–998.
21. Gruntzig A, Kuhlmann U, Vetter W, Lutolf U, Meier B, Siegenthaler W. Treatment of renovascular hypertension with percutaneous transluminal dilatation of a renal-artery stenosis. *Lancet*. 1978;1:801–802.
22. Ayerdi J and Hodgson WJ. Balloon angioplasty and stenting for renovascular occlusive disease. *Persp Vasc Surg Endovasc Ther*. 2004;16:25–42.
23. Leertouwer TC, Gussenhoven EJ, Bosch JL, et al. Stent placement for renal arterial stenosis: where do we stand? A meta-analysis. *Radiology*. 2000;216:78–85.
24. Kwolek CJ. Endovascular management of renal artery stenosis: current techniques and results. *Persp Vasc Surg Endovasc Ther*. 2004;16:261–279.
25. Axelrod DA, Fendrick AM, Carlos RC, et al. Percutaneous stenting of incidental unilateral renal artery stenosis: Decision analysis of costs and benefits. *J Endovasc Ther*. 2003; 10:546–556.
26. Ives NJ, Wheatley K, Stowe RL, et al. Continuing uncertainty about the value of percutaneous revascularization in atherosclerotic renovascular disease: a meta-analysis of randomized trials. *Nephrol Dial Transplant*. 2003;18:298–304.
27. Mwipatayi BP, Beningfield SJ, White LE, et al. A review of the current treatment of renal artery stenosis. *Eur J Vasc Endovasc Surg*. 2005;29:479–488.
28. Rocha-Singh KJ, Mishkel GJ, Katholi et al. Clinical predictors of improved long-term blood pressure control after successful stenting of hypertensive patients with obstructive renal artery atherosclerosis. *Catheter Cardiovasc Interv*. 1999;47:167–172.
29. Henry M, Amor M, Henry I, et al. Stent placement in the renal artery: Three-year experience with the Palmaz stent. *J Vasc Intervent Radiol*. 1996;7:343–350.
30. Blum U, Krumme B, Flugel P, et al. Treatment of ostial renal artery stenoses with vascular endoprostheses after unsuccessful balloon angioplasty. *N Engl J Med*. 1997;336:459–465.
31. Wong JM, Hansen KJ, Oskin TC, et al. Surgery after failed percutaneous renal artery angioplasty. *J Vasc Surg*. 1999;30:468–482.
32. Anderson CA, Hansen KJ, Benjamin ME, et al. Renal artery fibromuscular dysplasia: results of current surgical therapy. *J Vasc Surg*. 1995;22:207–215.
33. Cambria RP, Brewster DC, L'Italien G, et al. Simultaneous aortic and renal artery reconstruction: evolution of an eighteen-year experience. *J Vasc Surg*. 1995;21:916–925.
34. Cherr GS, Hansen KJ, Craven TE, et al. Surgical management of atherosclerotic renovascular disease. *J Vasc Surg*. 2002;35:236–245.
35. Hansen KJ, Starr SM, Sands RE, et al. Contemporary surgical management of renovascular disease. *J Vasc Surg*. 1992;16:319–330.
36. Hansen KJ, Wilson DB, and Edward MS. Surgical revascularization of atherosclerotic renovascular disease: State of the art. *Persp Vasc Surg Endovasc Ther*. 2004;16:281–298.

37. Murray SP, Kent KC, Salvatierra O, Stoney RJ. Complex branch renovascular disease: management options and late results. *J Vasc Surg*. 1994;20:338–345.
38. Stanley JC. The evolution of surgery for renovascular occlusive disease. *Cardiovasc Surg*. 1994;2:195–202.
39. Stanley JC, Whitehouse WM Jr, Zelenock GB, et al. Reoperation for complications of renal artery reconstructive surgery undertaken for treatment of renovascular hypertension. *J Vasc Surg*. 1985;2:133–144.
40. Cambria RP, Brewster DC, L'Italien GJ, et al. The durability of different reconstructive tech *J Vasc Surg*. 1994;20:76–85.
41. Khauli RB, Novick AC, Ziegelbaum M. Splenorenal bypass in the treatment of renal artery stenosis: experience with 69 cases. *J Vasc Surg*. 1985;2:547–551.
42. Moncure AC, Brewster DC, Darling RC, et al. Use of the splenic and hepatic arteries for renal revascularization. *J Vasc Surg*. 1986;3:196–203.
43. Dougherty MJ, Hallett JW Jr, Naessens J, et al. Renal endarterectomy vs. bypass for combined aortic and renal reconstruction: is there a difference in clinical outcome? *Ann Vasc Surg*. 1995;9:87–94.
44. McNeil JW, String ST, Pfeiffer RB Jr. Concomitant renal endarterectomy and aortic reconstruction. *J Vasc Surg*. 1994;20:331–336.
45. Stoney RJ, Messina LM, Goldstone J, Reilly LM. Renal endarterectomy through the transected aorta: a new technique for combined aortorenal arteriosclerosis-a preliminary report. *J Vasc Surg*. 1989;9:224–233.
46. Marone LK, and Cambria RP. Revascularization for renal function retrieval? Which patients will benefit? *Persp Vasc Surg Endovasc Ther*. 2004;16:258–260.
47. Hansen KJ, Cherr GS, Craven TE, et al. Management of ischemic nephropathy: Dialysis-free survival after surgical repair. *J Vasc Surg*. 2000;32:472–482.
48. Nationwide Inpatient Sample (NIS). Agency for Health Care Policy and Research, Rockville, MD. Available from: URL:http://www.ahcpr.gov/data/hcup/nisbroch.htm.
49. Charlson ME, Pompei P, Ales KL, MacKenzie CR. A new method for classifying prognostic comorbidity in longitudinal studies: development and validation. *J Chronic Dis*. 1987; 40:373–383.
50. Deyo RA, Cherkin DC, Ciol MA. Adapting a clinical comorbidity index for use with ICD-9-CM administrative databases. *J Clin Epidemiol*. 1992;45:613–619.
51. Romano PS, Roos LL, Jollis JG. Adapting a clinical comorbidity index for use with ICD-9-CM administrative data: differing perspectives. *J Clin Epidemiol*. 1993;46:1075–1079.
52. Sivamurthy N, Surowiec SM, Culakova E, et al. Divergent outcomes after percutaneous therapy for symptomatic renal artery stenosis. *J Vasc Surg*. 2004;39:565–74.
53. Sharafuddin MJ, Raboi CA, Abu-Yousef M, et al. Renal artery stenosis: duplex US after angioplasty and stent placement. *Radiology*. 2001;220:168–73.
54. Finlayson SR, Birkmeyer JD, Fillinger MF, Cronenwett JL. Should endovascular surgery lower the threshold for repair of abdominal aortic aneurysms? *J Vasc Surg*. 1999; 29:973–985.
55. Plouin PF, Chatellier G, Darne B, Raynaud A. Blood pressure outcome of angioplasty in atherosclerotic renal artery stenosis: A randomized trial. Essai Multicentrique Medicaments vs Angioplastie (EMMA) Study Group. *Hypertension*. 1998;31:823–829.
56. van de Ven PJ, Kaatee R, Beutler JJ, et al. Arterial stenting and balloon angioplasty in ostial atherosclerotic renovascular disease: A randomized trial. *Lancet*. 1999;353:282–286.
57. van Jaarsveld BC, Krijnen P, Pieterman H, et al. The effect of balloon angioplasty on hypertension in atherosclerotic renal-artery stenosis. Dutch Renal Artery Stenosis Intervention Cooperative Study Group. *N Engl J Med*. 2000;342:1007–1014.
58. Webster J, Marshall F, Abdalla M, et al. Randomised comparison of percutaneous angioplasty vs continued medical therapy for hypertensive patients with atheromatous renal artery stenosis. Scottish and Newcastle Renal Artery Stenosis Collaborative Group. *J Human Hypertens*. 1998;12:329–s335.

46

Debate: Mesenteric Ischemia

Arguments for Open Repair

William H. Pearce, M.D.

The Case for Stents

Mark C. Wyers, M.D.
Robert M. Zwolak, M.D., PH.D.

ARGUMENT FOR OPEN REPAIR

William H. Pearce, M.D.

Open surgery is preferred over endovascular surgery for the treatment of mesenteric ischemia. Mesenteric ischemia is an unusual clinical problem. Because of its rarity, its diagnosis is often missed and the patient presents with catastrophic bowel infarction. The presence of intra-abdominal sepsis associated with bowel infarction may limit the utility of endovascular procedures. Bare metal stents may, in fact, become infected.[1]

Surgical revascularization has been the gold standard for the treatment of both acute and chronic mesenteric ischemia. Over the years, there has been an evolution of thinking about mesenteric ischemia and the number of vessels needed to be revascularized. In our own practice. for many years, we have used multiple visceral revascularizations as our standard procedure.[2] This procedure required a supraceliac origin of the bypass graft with distal anastomosis to the celiac and superior mesenteric arteries (SMA) (Figure 46–1). The supraceliac aorta is often chosen because of its lack of atherosclerotic plaque. However, the supraceliac aortic clamp may produce hemodynamic changes, further compromising the patient. Therefore, I have commonly preferred to use the iliac arteries as inflow when they are free of major occlusive disease (Figure 46–2).

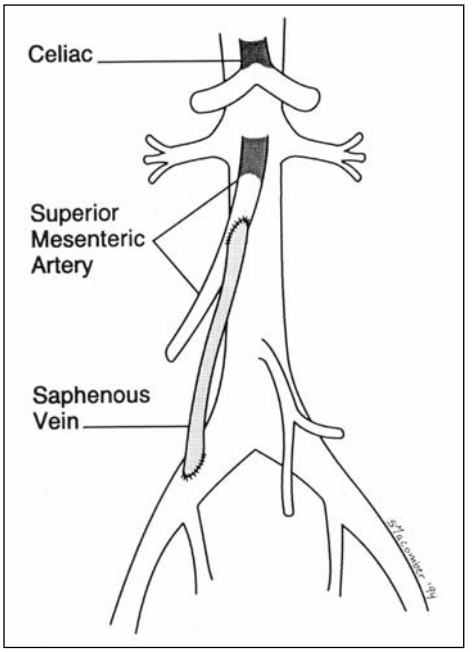

Figure 46-1. Configuration of antegrade supraceliac saphenous vein mesenteric bypass with "piggyback" technique. Note single aortic anastomosis and origin of SMA graft. From McMillan MD, McCarthy WJ, Bresticker MR, et al. Mesenteric artery bypass: Objective patency determination. *J Vasc Surg*. 1995;21:729–741. Reproduced by permission.

In patients with chronic mesenteric ischemia, the therapeutic options are either endovascular repair or open surgical bypass. Endovascular treatments are attractive because of their less invasive nature. Angioplasty and stenting can be performed for both celiac and superior mesenteric artery (SMA) stenosis. However, most interventionalists have shied away from celiac artery stenting because of compression produced by the median arcuate ligament. Most stents placed in this location will occlude. In recent years, there has been a trend to perform angioplasty and stenting only for SMA stenosis in the chronic setting.[3] However, the downsides to this

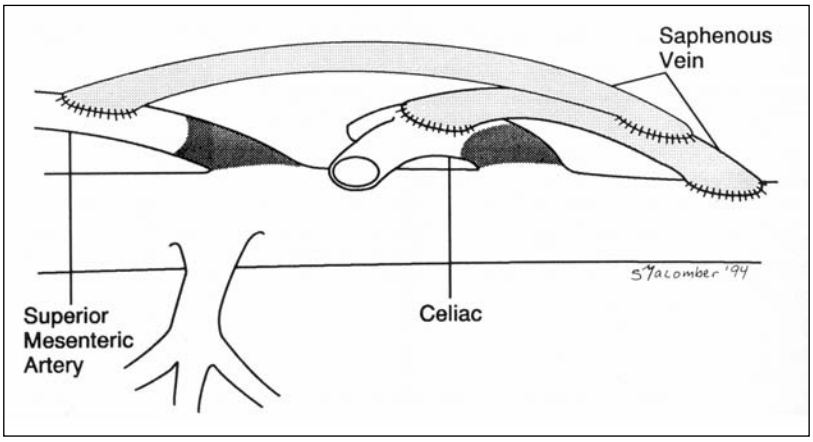

Figure 46-2. Configuration of retrograde saphenous vein bypass from iliac artery to SMA with "reversed" distal anastomosis. From McMillan MD, McCarthy WJ, Bresticker MR, et al. Mesenteric artery bypass: Objective patency determination. *J Vasc Surg*. 1995;21:729–741. Reproduced by permission.

approach may be substantial. The SMA leaves the aorta at almost right angles to make a turn over the left renal vein. These right angles may predispose the artery to dissection. We have several patients in whom dissection has occurred in the attempt to treat total occlusions of the SMA. Dissections of the SMA make revascularization almost impossible, and clearly make a straightforward operation much more difficult. The group from Portland has recommended open revascularization of the single artery (the SMA) with excellent results.[4] We tend to agree with that position and changed our practice from multiple visceral artery revascularizations to single vessel reconstructions in most cases.

In patients with acute mesenteric ischemia, the situation is complicated by marginal and/or necrotic bowel. In these settings, several options are available. The first option is to perform a thrombectomy through an arteriotomy in the SMA, which if not successful, can be used as the site of the proximal anastomosis in a bypass procedure. Again, the inflow source is the iliac arteries and the conduit is autogenous saphenous vein. Recently, it has been reported that retrograde angioplasty and stenting of the SMA with patch closure can replace a venous bypass.[5] While this approach appears attractive in that it minimizes the operative procedure, it only does so in a limited fashion. Placing a stent in retrograde fashion in the SMA requires a lateral view of the aorta and somewhat precise placement. Performing endovascular procedures in off hours may be problematic in that the proper personnel and equipment may not be readily available. One can argue that the time it takes to obtain the radiographic images, place the stent, and patch the artery is the same time that would be required to do the bypass procedure and avoid any implant. The proponents of this endovascular approach suggest that harvesting of the vein segment for the bypass procedure is lengthy and more dangerous for the patient. The only role, in my opinion, for retrograde stenting of the SMA is in the patient with severely calcified arteries in all potential inflow locations, and in whom there is not a good venous conduit.

The endovascular revolution in vascular surgery has changed the way we have done many things. As in almost every case, endovascular procedures have a role in selected patients. However, endovascular aneurysm repair (EVAR) is not suitable for all patients with aneurysmal disease, nor is stenting of the mesenteric arteries. Preliminary results suggest good initial results with stenting of the mesenteric vessels but long term follow-up is rare.[6,7] In addition, the potential downside to patients undergoing stenting, particularly with SMA occlusion, is possible dissection of the distal SMA with disastrous consequences. Another potential downside for widespread use of mesenteric artery stenting is the stenting and ballooning of arteries in patients who do not have mesenteric ischemia. Celiac artery stenosis is common and is frequently associated with the median arcuate syndrome. Many patients have been referred for evaluation of this common CT finding. Without prior clinical experience, these patients may be subjected to unnecessary mesenteric artery stenting.

In sum, open surgery remains an important adjunct in patients with both acute and chronic mesenteric ischemia. Clamping of iliac vessels does not produce significant hemodynamic impact. Proper construction of a retrograde bypass does not adversely impact the patient, and provides excellent long-term outcome. In the setting of acute mesenteric ischemia, the use of a bypass procedure avoids any implant. Unfortunately, despite our best efforts in the treatment of patients with acute mesenteric ischemia, mortality remains high because of the reperfusion of an ischemic visceral bed and the attendant medical complications associated with this disease.

REFERENCES

1. Hogg ME, Peterson BG, Pearce WH, et al. Bare metal stent infections: Case report and review of the literature. *J Vasc Surg*. In press.
2. McMillan WD, McCarthy WJ, Bresticker MR, et al. Mesenteric artery bypass: Objective patency determination.*J Vasc Surg*. 1995;21:729–741.
3. Brown DJ, Schermerhorn ML, Powell RJ, et al. Mesenteric stenting for chronic mesenteric ischemia. *J Vasc Surg*. 2005;42:268–274.
4. Foley MI, Moneta GL, Abou-Zamzam AM Jr, et al. Revascularization of the superior mesenteric artery alone for treatment of intestinal ischemia. *J Vasc Surg*. 2000;32:37–47.
5. Wyers MC, Powell RJ, Nolan BW, Cronenwett JL. Retrograde mesenteric stenting during laparotomy for acute occlusive mesenteric ischemia. *J Vasc Surg*. 2007;45:269–275.
6. Schaefer PJ, Schaefer FK, Hinrichsen H, et al. Stent placement with the monorail technique for treatment of mesenteric artery stenosis. *J Vasc Interv Radiol*. 2006;17:637–643.
7. Landis MS, Rajan DK, Simons ME, et al. Percutaneous management of chronic mesenteric ischemia: Outcomes after intervention. *J Vasc Interv Radiol*. 2005;16:1319–1325.

MESENTERIC ISCHEMIA: THE CASE FOR STENTS

Mark C. Wyers, M.D., and Robert M. Zwolak, M.D., Ph.D.

Unlike renal or peripheral percutaneous intervention, angioplasty and stenting for mesenteric occlusive disease has been extremely slow to gain clinical acceptance. The most likely explanation is the fear of catastrophic acute mesenteric ischemia that might follow should the patient suffer acute arterial closure or distal embolization during or after the procedure. Indeed, almost every seasoned vascular surgeon has an anecdotal horror story related to unsuccessful or complicated attempts at percutaneous mesenteric intervention. Nevertheless, science is beginning to overtake suspicion in this arena. Experts at percutaneous intervention have published prospectively collected series with credible results during elective therapy, and more recently, a new treatment option for acute mesenteric ischemia has been introduced that involves the use of arterial stenting.

CHRONIC MESENTERIC ISCHEMIA

Involvement of the mesenteric arteries with atherosclerotic occlusive disease is an uncommon disorder that may go unrecognized for months or years before the diagnosis is established and treatment undertaken. Chronic mesenteric ischemia (CMI) accounts for less than 2% of revascularization procedures for atherosclerotic disease. CMI is unusual for an atherosclerotic disorder in that it affects women more frequently than men. Even more rare sources of CMI include fibromuscular dysplasia, polyarteritis nodosa, and median arcuate ligament syndrome. Development of duplex ultrasound to identify visceral artery stenosis and occlusion in CMI has expedited diagnosis of this disorder,[1,2] but many patients are still extremely malnourished by the time the diagnosis is established. The debilitated patient is a poor candidate for a major intra-abdominal revascularization operation. Open surgical bypass for CMI has been reported to carry mortality rates as low as 0%[3] but most reports cite mortality in the 5–8% range.[4] Manuscripts citing 10% or higher perioperative mortality usually include concomitant surgical procedures to reconstruct the

aorta, thereby adding an entire additional layer of complexity to the surgery.[5] Nevertheless, from the perspective of a skilled interventionalist, when as many as one in every 10 patients dies within 30 days of surgery, the utility of minimally invasive percutaneous techniques must be considered.

PERCUTANEOUS INTERVENTION FOR CMI

Kasirajan and colleagues from The Cleveland Clinic provided an early report of percutaneous intervention. Twenty-eight patients who underwent percutaneous revascularization for symptomatic CMI (percutaneous angioplasty, stenting, or both) between 1995 and 1997 were compared to 85 patients from the same institution treated with open surgical revascularization (bypass graft, transaortic endarterectomy, or patch angioplasty) between 1977 and 1997.[6] The cohorts were similar in terms of nonacute symptomatic status, demographics, and multiple vessel involvement. However, the percutaneous treatment patients were older (median age 72 versus 65 years) and fewer vessels per patient were treated. Eight-six percent of the percutaneous group underwent one vessel revascularization, while the surgical group underwent a 50/50 mix of single versus two-vessel treatment. The early in-hospital mortality rate was 10.7% for percutaneous revascularization versus 8.2% for open surgery (ns), and there was no statistical difference in complication rate, 18% for percutaneous therapy versus 33% for open surgery. Percutaneously treated patients had a statistically shorter hospital length of stay. They also had a higher incidence of recurrent symptoms ($p<0.001$), although there was no difference at three years in recurrent stenosis or mortality. The authors reached a conclusion favoring open surgical revascularization, although in retrospect, these data seem almost a wash.

Sivamurthy et. al. also published a retrospective nonrandomized single-center review of open versus percutaneous treatment for CMI.[7] Treatment dates spanned from January 1989 to September 2003. All patients had atherosclerosis with median two-vessel involvement. Like other reports, most were women. Open surgery in 46 patients included 43 vessels that were bypassed and 23 that underwent endarterectomy. Endoluminal treatment was undertaken in 21 patients with 22 vessels treated. In-hospital and 30-day mortality rates were 15% in the open group and 21% in endovascular patients ($p = 0.08$). Cumulative patency at six months was 83% for open surgery and 68% for endovascular. Major morbidity, median postoperative length of stay, and freedom from recurrent symptoms at six months were all statistically greater in the open group. Three-year survival by life table analysis was 62% following open surgery and 63% after percutaneous therapy. The authors reached a negative conclusion regarding endovascular therapy, but these outcomes seem equally modest, perhaps reflecting the early nature of the experience (dating back to 1989), and the low treatment volume (22 interventions in 14 years).

More recent series of endovascular treatment for CMI demonstrate substantially lower complication rates. For instance, Brown et. al. from Dartmouth reported a consecutive series of 14 patients who underwent mesenteric stenting for CMI from 2001 to 2004.[8] Mean patient age was 73, and 64% were women. In-hospital and 30-day mortality was zero, and there was no major morbidity. Mean length of stay was two days. Restenosis, diagnosed by duplex scan, occurred in eight patients (57%) during a mean follow-up period of only 13 months. Seven of eight were symptomatic, and

arteriography confirmed significant restenosis. Mean time to reintervention was nine months (range two to 22 months). One patient required surgical bypass while the others were treated by repeat percutaneous intervention. All retreatments were successful, and 93% of patients were symptom-free at last recorded follow-up. The authors performed a comparison to 33 patients from the same institution who underwent open surgical mesenteric revascularization from 1990 to 2004. The stented patients had lower perioperative major morbidity (0% versus 30%, $p < 0.01$), while perioperative mortality failed to reach statistical significance (0% versus 9%, ns). Stented patients had shorter median length of stay (two versus 10 days, $p<0.01$) and shorter intensive care stay (0 versus three days, $p<0.01$). In conclusion, the authors confirmed a substantial rate of early restenosis associated with visceral artery stenting, but morbidity and mortality of the procedure were less than reported in earlier series.

Less morbid endovascular outcomes were also reported by Biebl et. al. from The Mayo Clinic, Jacksonville.[9] Forty-nine patients underwent surgical or endovascular treatment for CMI. The authors chose relief of symptoms as the primary endpoint with mortality, morbidity, and patency analyzed as secondary endpoints. Twenty-six patients underwent surgical revascularization, while 23 were treated endoluminally. Preoperative demographics were comparable. Immediately following intervention, freedom from CMI symptoms was 100% after surgery and 90% after percutaneous therapy (p = ns). After 25 months mean follow-up, freedom from symptoms was 89% surgical versus 75% endoluminal. Similar to most other reports, reocclusion or restenosis was higher in the endoluminal group, 25% versus 8% ($p = 0.003$). Symptomatic mesenteric ischemia recurred in 9% of the endoluminal patients versus none of the surgical patients. In parallel with recurrent symptoms and ischemia, 13% of endoluminal patients required reintervention while none of the surgical patients required retreatment. Surgical patients experienced significantly more early complications (42% versus 4%), longer hospital stay (11.6 days versus 1.3 days), and higher overall mortality at the end of the follow-up (31% versus 4%). Interpretation of these results is complicated by the fact that several of the surgical patients underwent simultaneous aortic reconstruction, shifting them into a much more complex clinical situation. The authors concluded that while surgical treatment has superior long-term revascularization patency and requires fewer reinterventions, it is also associated with greater mortality and morbidity than endovascular therapy. They recommended individualization when considering treatment choice, based on patient characteristics.

Atkins et. al. recently reviewed the Massachusetts General Hospital experience with elective mesenteric revascularization using percutaneous and open techniques. In order to clarify the analysis, the authors excluded patients who required simultaneous complex aneurysm repair.[10] Thirty-one patients underwent percutaneous intervention with treatment of 42 vessels. Open revascularization was performed in 49 patients, and 88 vessels were treated. Mean follow-up was shorter in the percutaneous group (15 versus 42 months). Baseline comorbidities were similar. Percutaneously treated patients had fewer vessels revascularized (1.5 versus 1.8, $p = 0.001$). In-hospital mortality was 3% endoluminal and 2% surgical (ns). In-hospital major morbidity was 13% endoluminal and 2% open surgical (ns). At one year, radiographic primary patency was 58% endoluminal compared to 90% open surgical ($p = 0.001$), while primary assisted patency was 65% endoluminal versus 96% open surgical ($p<0.001$). This series differs from others in its strikingly low surgical morbidity and mortality, some of which must be due to exclusion of those requiring major aortic reconstructions, and some of which must be due to excellent surgery and postoperative care.

The experience also differs in terms of a higher requirement for late secondary intervention in the surgical group. Overall, 22% of open surgical patients required a second intervention during follow-up, not unlike the 16% requirement in the percutaneous group. These authors also stressed the need for individualization when considering treatment choice, based on patient anatomy and comorbidities.

A recent review of endovascular therapy for CMI was based on a MEDLINE search for English language literature including series of at least five patients. Sixteen manuscripts totaling 328 patients were identified and pooled for outcomes.[11] Technical success was claimed in 91% with clinical success in 82%, and "late" clinical success in 75%. Overall, the group had 11 deaths within 30 days (3%). Complication rate was 9%. Restenosis occurred in 84 patients (28%) at an average of 26 months follow-up. Repeat intervention was required in 79 patients (27%). Despite the latitude of self-reported single-center data, these results are very promising when considered in light of the multiply comorbid patients who require treatment for CMI. The high rate of restenosis and repeat intervention has been demonstrated in almost every series and represents a challenge for innovative clinicians. Nevertheless, having a live patient who needs re-treatment is better than not having a patient at all.

ACUTE MESENTERIC ISCHEMIA

Acute mesenteric ischemia (AMI) carries a high mortality, between 60 and 80%, based on a recent review by Oldenburg and associates.[12] AMI has a wider range of causes than CMI, including arterial embolism (~40-50% of cases), acute arterial thrombosis superimposed on preexisting atherosclerotic disease (~25%), nonocclusive mesenteric ischemia (~20%), and venous thrombosis (~10%). Treatment for AMI depends on the causative agent. Embolization is typically treated with emergent open surgical embolectomy, while the primary treatment for nonocclusive mesenteric ischemia is medical unless gangrenous bowel requires excision. Venous thrombosis is treated with heparin anticoagulation or venous thrombectomy, with excision of necrotic bowel as needed. This leaves, perhaps, the most challenging AMI cohort, terminal thrombosis of an atherosclerotic SMA. These patients are likely to have suffered CMI symptoms of malnutrition and weight loss prior to the terminal thrombosis. Successful treatment requires revascularization, meaning that some method must be undertaken to provide arterial inflow beyond the advanced atherosclerosis that typically occupies the origin and initial 3-6 centimeters of the SMA. This is done with emergent bypass graft placement, using inflow from the supraceliac aorta, the infrarenal aorta, or the iliac arteries. The operation is complex, and recovery is challenging in the very ill patient. Kougias et. al. reviewed 72 patients who underwent emergent operative intervention for acute mesenteric thrombosis or embolism in a report published in 2007.[13] Treatments included thrombectomy (31%), mesenteric bypass grafting (46%), patch angioplasty (12%), reimplantation (7%), and endarterectomy (4%). Bowel resection was required during the initial operation in 31%, and during a second look operation in 53%. Perioperative morbidity and 30-day mortality rates were 39% and 31%, respectively, excellent results for these critically ill patients. Age greater than 70 and prolonged symptom duration were independent predictors of mortality.

Percutaneous interventional treatment for AMI is rare. A few case reports have been published, but wide experience is lacking.[14-16] However, Wyers and coauthors recently reported a hybrid open/interventional approach for treatment of acute atherosclerotic

SMA thrombosis.[17] Similar to a technique described in an earlier case report by Milner,[18] the Dartmouth authors described Retrograde Open Mesenteric Stenting (ROMS) of the SMA. With the diagnosis of AMI, laparotomy is typically required to explore and resect gangrenous bowel. This allows ready access to the SMA at the base of the transverse mesocolon for retrograde cannulation. Following heparin anticoagulation, the SMA is incised longitudinally, and a local thromboendarterectomy is performed if necessary. Placing a patch angioplasty then facilitates the remaining portions of the procedure. We typically use bovine pericardial patch, but saphenous vein would be suitable. A purse-string suture can be placed in the patch around the puncture site to facilitate sheath removal without having to reclamp the SMA. A 6F, 35-cm-long flexible sheath (Arrow International Inc, Reading PA) is placed through the patch into the SMA in retrograde direction, headed toward the aorta. The extra diameter provided by the patch allows angiographic evaluation of the distal mesenteric arcades through the sheath, both before and after restoration of SMA inflow. The long sheath also allows the surgeon to avoid extensive fluoroscopic exposure while working comfortably away from the image intensifier.

Once the sheath is in place, metallic retractors are removed, and the surgeon performs hand injection retrograde lateral angiography (Figure 46–3). This demonstrates the exact site of stenosis or occlusion. A simultaneous flush aortogram from a percutaneous femoral or brachial catheter may be used to profile the aorta, thereby completely outlining the lesion that must be crossed and treated to regain arterial inflow. A 0.035-inch glidewire (Terumo, Somerset, NJ or other similar) is often useful to cross the lesion, with subsequent exchange for a lower profile platform. Predilation with a 2 or 3 mm angioplasty balloon is usually necessary. This is followed by retrograde stent placement using 5, 6, or 7 mm low-profile balloon expandable stents (Figure 46–4). The leading edge of the proximal-most stent is positioned to protrude 1-2 mm into the aortic lumen. More than one stent is oftentimes required to fully treat

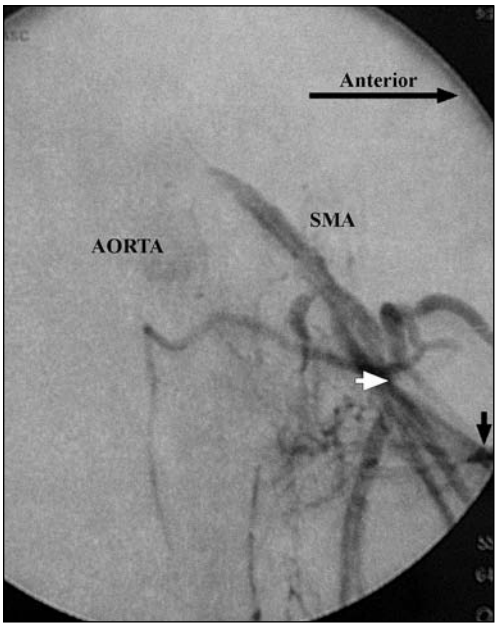

Figure 46-3. Retrograde SMA injection. Note the proximity of the sheath's point of entry (black arrow) and of the sheath's tip (white arrow) to the proximal SMA occlusion. There is no reflux of contrast into the aorta. With permission.

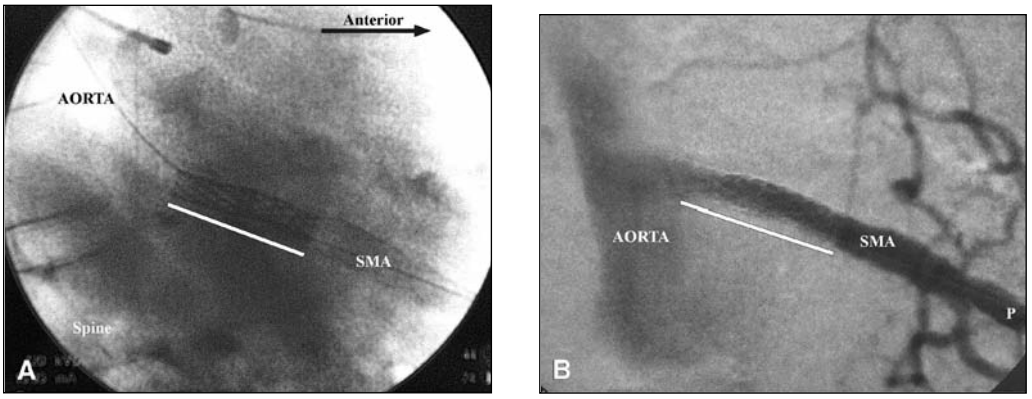

Figure 46-4. (**A**) Intraoperative lateral fluoroscopic image shows two stents (underscored by white line) deployed in the SMA origin with the 0.018-inch wire still in place. Note the lumbar vertebral bodies to the left. (**B**) Completion retrograde arteriogram shows free reflux of contrast into the aorta and no residual angiographic stenosis; P denotes the approximate location of the SMA patch angioplasty. With permission.

the SMA lesion. Completion arteriography is performed in multiple views to confirm technical adequacy. We usually also perform pressure measurements to confirm the absence of a residual gradient across the stented area. Completion imaging of the SMA arcades is recommended, and vasospasm may be treated with injections of papaverine or glucagon. With a satisfactory technical result achieved, the sheath is removed and the hole in the patch is sutured. The mesentery is closed over the SMA exploration site. Perforated and necrotic bowel is resected. Close attention is given to generous fluid resuscitation during the immediate postop period.

Our 2007 report compared six patients with acute thrombotic mesenteric ischemia who underwent ROMS to five patients who underwent emergent mesenteric bypass graft, and to two patients who were treated with percutaneous antegrade SMA stent placement.[17] This is a small series with no statistically significant results, but the ROMS outcomes were promising. Technical success with ROMS was 100%, even in five patients who had previous unsuccessful attempts to cross the SMA from a percutaneous antegrade approach. The ROMS group suffered only 17% in-hospital mortality compared to 80% following emergent mesenteric bypass, and 100% in the two percutaneous stent patients, although these results did not reach significance due to low "n." Five of the six ROMS patients were discharged to home after a mean hospital stay of 20 days. During one year mean follow-up, three of them died of unrelated causes, while two were alive and well, one with an asymptomatic recurrent stenosis. In conclusion, ROMS during emergent laparotomy for AMI is a promising technique and an attractive alternative to emergent surgical bypass. This method needs to be tested by others to determine its true value in comparison to traditional methods.

CLINICAL AND DUPLEX FOLLOW-UP

Based on the available data, patients undergoing endovascular visceral revascularization for chronic or acute mesenteric ischemia are likely to develop recurrent stenosis. Close clinical follow-up would appear to be indicated, and the issue arises whether duplex ultrasound would be a valuable adjunct. The literature on this topic is meager. Fenwick et. al. reviewed

their experience using color Doppler ultrasound to identify recurrent stenosis in patients who had undergone successful percutaneous mesenteric intervention, and they attempted to relate this to recurrence of symptoms or weight loss.[19] With a total "n" of five, they identified restenosis in three patients. However, all three individuals were asymptomatic when the duplex observations were made. The authors were appropriately circumspect, but this is an extremely modest report with little interpretive information. In a more robust report, Liem et. al. characterized duplex-derived flow velocities in mesenteric artery bypass graft limbs, although they did not study patients who had undergone percutaneous mesenteric revascularization or ROMS.[20] Our experience at Dartmouth with mesenteric duplex and following interventional visceral artery therapy is promising, although still empiric-based on an anticipated high rate of restenosis. We have not identified an absolute velocity threshold that would indicate the need for prompt reintervention. However, the traditional velocity criteria for native vessels seem to provide reasonably accurate information to guide clinical decision-making. Suffice to say the entire concept of clinical and duplex follow-up after open and percutaneous visceral revascularization deserves further evidence development.

CONCLUSION

There are no randomized trials we are aware of, but as published experience accrues, percutaneous therapy for CMI may be the best option for high-surgical risk patients with advanced malnutrition and wasting. Possibly, percutaneous intervention may be the treatment of choice for all patients with anatomically suitable lesions at centers of excellence where periprocedural morbidity and mortality are low. Patients with long total occlusions still require open surgical bypass or endarterectomy. Based on the majority of reports, percutaneous treatment carries a higher rate of recurrent stenosis. Recurrence can usually be treated percutaneously, but the choice of open versus percutaneous retreatment should be considered carefully when the need arises. Acute mesenteric ischemia usually requires open laparotomy to explore the bowel. This requirement provides the opportunity for retrograde open mesenteric stenting (ROMS), a new method to revascularize the SMA. Finally, realizing that recurrent stenosis is likely, close clinical follow-up is indicated. The role of duplex ultrasound following percutaneous visceral intervention is yet to be defined.

REFERENCES

1. Moneta GL, Lee RW, Yeager RA, et al. Mesenteric duplex scanning: A blinded prospective study. *J Vasc Surg*. 1993;17:79–86.
2. Zwolak RM, Fillinger MF, Walsh DB, et al. Mesenteric and celiac duplex scanning: a validation study. *J Vasc Surg*. 1998;27:1078–1087; discussion 1088.
3. Johnston KW, Lindsay TF, Walker PM, Kalman PG. Mesenteric arterial bypass grafts: early and late results and suggested surgical approach for chronic and acute mesenteric ischemia. *Surgery*. 1995;118(1):1–7.
4. Mateo RB, O'Hara PJ, Hertzer NR, et al. Elective surgical treatment of symptomatic chronic mesenteric occlusive disease: early results and late outcomes. *J Vasc Surg*. 1999;29(5):821-831; discussion 832.
5. McAfee MK, Cherry KJ, Jr., Naessens JM, et al. Influence of complete revascularization on chronic mesenteric ischemia. *Am J Surg*. 1992;164(3):220–224.

6. Kasirajan K, O'Hara PJ, Gray BH, et al. Chronic mesenteric ischemia: open surgery versus percutaneous angioplasty and stenting. *J Vasc Surg*. 2001;33(1):63–71.
7. Sivamurthy N, Rhodes JM, Lee D, et al. Endovascular versus open mesenteric revascularization: immediate benefits do not equate with short-term functional outcomes. *J Am Coll Surg*. 2006;202(6):859–867.
8. Brown DJ, Schermerhorn ML, Powell RJ, et al. Mesenteric stenting for chronic mesenteric ischemia. *J Vasc Surg*. 2005;42(2):268–274.
9. Biebl M, Oldenburg WA, Paz-Fumagalli R, et al. Surgical and interventional visceral revascularization for the treatment of chronic mesenteric ischemia—when to prefer which? *World J Surg*. Mar 2007;31(3):562–568.
10. Atkins MD, Kwolek CJ, LaMuraglia GM, et al. Surgical revascularization versus endovascular therapy for chronic mesenteric ischemia: a comparative experience. *J Vasc Surg*. 2007;45(6):1162–1171.
11. Kougias P, El Sayed HF, Zhou W, Lin PH. Management of chronic mesenteric ischemia. The role of endovascular therapy. *J Endovasc Ther*. 2007;14(3):395–405.
12. Oldenburg WA, Lau LL, Rodenberg TJ, et al. Acute mesenteric ischemia: a clinical review. *Arch Intern Med*. 24 2004;164(10):1054–1062.
13. Kougias P, Lau D, El Sayed HF, et al. Determinants of mortality and treatment outcome following surgical interventions for acute mesenteric ischemia. *J Vasc Surg*. 2007 July 26 [Epub ahead of print].
14. Leduc FJ, Pestieau SR, Detry O, et al. Acute mesenteric ischaemia: minimal invasive management by combined laparoscopy and percutaneous transluminal angioplasty. *Eur J Surg*. 2000;166(4):345–347.
15. Brountzos EN, Critselis A, Magoulas D, et al. Emergency endovascular treatment of a superior mesenteric artery occlusion. *Cardiovasc Intervent Radiol*. 2001;24(1):57–60.
16. Gartenschlaeger S, Bender S, Maeurer J, Schroeder RJ. Successful Percutaneous Transluminal Angioplasty and Stenting in Acute Mesenteric Ischemia. *Cardiovasc Intervent Radiol*. 2007 Jan 4. [Epub ahead of print]
17. Wyers MC, Powell RJ, Nolan BW, Cronenwett JL. Retrograde mesenteric stenting during laparotomy for acute occlusive mesenteric ischemia. *J Vasc Surg*. 2007;45(2):269–275.
18. Milner R, Woo EY, Carpenter JP. Superior mesenteric artery angioplasty and stenting via a retrograde approach in a patient with bowel ischemia—a case report. *Vasc Endovascular Surg*. 2004;38(1):89–91.
19. Fenwick JL, Wright IA, Buckenham TM. Endovascular repair of chronic mesenteric occlusive disease: the role of duplex surveillance. *ANZ J Surg*. 2007;77(1-2):60–63.
20. Liem TK, Segall JA, Wei W, et al. Duplex scan characteristics of bypass grafts to mesenteric arteries. *J Vasc Surg*. 2007;45(5):922-927; discussion 927–928.

SECTION **VII**

Endovenous Technologies

47

Endovascular Treatment for Major Vein Occlusion

Peter Gloviczki, M.D., Konstantinos T. Delis, M.D., Ph.D., and Haraldur Bjarnason, M.D.

During the past decade, the endovascular revolution has transformed the management of vascular diseases and the treatment of venous occlusive disease has been no exception.[1] Stenting of large veins has been effective and durable at early and mid-term follow-up [2-12] rendering open surgical reconstructions far less indispensable currently than five years ago.[13-14] Early and mid-term results of stenting have been promising in most large veins, particularly in patients with high-grade stenosis or obstruction of the left iliac vein (May-Thurner syndrome). Endovenous stent deployment has become the treatment of choice in symptomatic benign occlusive disease and the palliative management of central vein obstruction due to end-stage malignant tumors. Patency of grafts used for reconstruction of large veins also may be improved by endovascular techniques such as angioplasty and stenting.[15] Patients requiring venous reconstruction during excision of malignant tumors or some of those who have traumatic injury to large veins are still candidates for primary open surgical treatment.[16] Some stents will fail, and long, chronic occlusions are less frequently suitable for stenting. These patients are also potential candidates for open surgery. There is little doubt, however, that endovenous treatment for major vein occlusion is here to stay, and results with progress in technology and perfection in adjuvant therapy will further improve. In this review, we will focus on the evaluation, techniques, and results of stenting of the iliac veins and the inferior vena cava.

THE RATIONALE FOR ENDOVASCULAR TREATMENT

Conservative therapy for iliofemoral venous thrombosis, entailing anticoagulation, bed rest, leg elevation, and elastic compression is associated with disappointing long-term consequences in light of the development of the postthrombotic syndrome, which may include leg swelling, venous claudication, skins changes, ulceration, altered venous

function and a compromised quality of life. In a recent study by Delis et al after a median follow-up of five years, 81% of the limbs with iliofemoral venous thrombosis treated conservatively had superficial and deep reflux, while the remaining 19% had superficial reflux alone.[17] More than 40% of the patients experienced venous claudication, commencing at a median walking distance of 130 meters, and 15% of the patients were forced to discontinue walking at a median distance of 240 meters because of the severity of pain. The afflicted limbs had a reduced venous outflow capacity, abnormally high venous reflux, and residual venous volume following exercise. The clinical deterioration, documented objectively using the CEAP classification and Venous Clinical Severity Scoring, was associated with impairment in the quality of life perceived in the physical functioning and role, general health, social function, and mental health.[17] The impact of thrombus elimination on the health-related quality of life in patients with iliofemoral venous thrombosis was also emphasized by Camerota et al.[18] The effect of catheter-directed thrombolysis with urokinase was compared with that of standard anticoagulation. Patients treated with urokinase reported better physical functioning, less stigma and health distress, and fewer postthrombotic symptoms, compared with the patients offered anticoagulation alone. Successful thrombolysis was associated with significantly improved quality of life, while patients classified as lytic failures had similar outcomes to those treated with heparin alone.

In light of the disappointing results of extensive iliofemoral venous thromboses treated conservatively, more aggressive therapies associated with early recanalization have been advocated. For treatment of acute iliofemoral thrombosis, thrombolysis and percutaneous mechanical thrombectomy have been used with increasing frequency, while for chronic venous occlusions of the iliofemoral veins and the inferior vena cava, balloon dilatation and stenting has been advocated both by our group at the Mayo Clinic and by other investigators.[2-12]

PATIENT EVALUATION

Evaluation of the patient should reveal the etiology, severity, and extent of deep venous occlusion, and the age of thrombus. In chronic venous occlusion, any associated venous valvular incompetence should also be identified.

History and Physical Examination

Patients with acute venous occlusion present with a sudden onset of pain, swelling, and cyanotic discoloration of the leg. Only in very advanced cases do we see evidence of tissue loss or venous gangrene (phlegmasia coerulea dolens). Patients with chronic venous occlusion have leg swelling and experience exercise-induced pain in the thigh muscles (*venous claudication*), described as a "bursting" pain in the thigh and sometimes in the calf, which is relieved by rest and by leg elevation.

Signs of postthrombotic syndrome such as leg edema, varicose veins, skin changes, lipodermatosclerosis, eczema, and ulceration are noted. Distended varicose veins are evident even in the supine position, and suprapubic and abdominal wall collaterals may develop in pelvic venous occlusion. Bleeding from high-pressure varicosities is not infrequent. The leg has a cyanotic hue, and swelling in both legs may occur in bilateral iliofemoral or vena caval obstruction. The evaluation of the patients should aim at

identifying the risk factors for deep venous thrombosis including a family history of thrombophilia, malignancy, trauma, surgery, and hormone-replacement therapy. The identification of symptoms and signs of pulmonary embolism is critically important.

Noninvasive Venous Evaluation

Duplex scanning enables detection of lower acute deep vein thrombosis, determination of the type of occlusion (complete or partial), and assessment of the extent and severity of venous valvular incompetence. Duplex ultrasonography is less sensitive in visualizing the iliac veins and the inferior vena cava. Computed tomography (CT) or magnetic resonance imaging (MRI) will exclude abdominal or pelvic disease (e.g., tumor, cyst, retroperitoneal fibrosis). Acute thrombosis of the pelvic veins or inferior vena cava can be diagnosed with contrast-enhanced CT angiography, but artifacts caused by a mixture of enhanced and nonenhanced blood have to be recognized and correctly interpreted. Filling defects in large veins are often due to inflow of unopacified blood into a vessel filled with enhanced blood. This can be true for both CT and MRI. Strain-gauge and air plethysmography are useful in the diagnosis and quantification of venous outflow obstruction, and may enable documentation of improvement following treatment.

Measurement of the arm-foot venous pressure differential, both at rest and during exercise, has been proposed by Raju et al[19] as an objective quantitative method of lower limb venous hypertension. Exercise entails 10 dorsiflexions of the foot or 20 isometric contractions of the calf muscle. A resting arm-foot pressure differential greater than 4 mmHg is considered evidence of significant chronic obstruction. Yet Neglen and Raju more recently discarded the use of pressure measurement for the selection of patients for endovascular procedures.[20-24] In our practice, a pressure difference of at least 5 mmHg between the femoral and the central pressures in the supine patient or a two-fold increase in femoral vein pressure after exercise are used for detection of hemodynamically significant proximal stenosis or occlusion.

Contrast Phlebography

In patients considered for venous reconstruction in the context of both acute and chronic venous disease, detailed contrast phlebography is performed. In our practice, we use ascending phlebography to evaluate obstruction and, if needed, descending venography to assess any associated valvular incompetence.[25] Iliocavography and abdominal venacavography through a jugular or brachial approach may also be essential for enabling visualization of the vena cava proximal to the occlusion. Femoral access is useful not only for descending phlebography and iliocavography, but also for measuring femoral venous pressures.

ANESTHESIA

Large vein stenting is most often performed under conscious sedation and local anesthesia. As angioplasty of a chronically occluded vein is often painful, stronger analgesia (fentanyl in combination with sedatives such as midazolam) may be used. General anesthesia may be used in patients in whom stenting follows surgical thrombectomy.

TECHNIQUE OF ILIOFEMORAL AND CAVAL STENTING

The technique used invariably at our institution for recanalization of the occluded iliofemoral veins and the inferior vena cava has been described by Bjarnason et al [2, 26, 27] and Paulsen et al.[12] In this review, we emphasize the key elements of access and stent deployment, and review the most frequently deployed stents.

Access Site

In general, we access the venous system at a site remote from the obstruction. If the diseased area is too close to the access site, technical problems may arise during balloon angioplasty and stent deployment. In iliofemoral occlusion, the approach of our choice is from the right internal jugular vein or the popliteal vein (Figures 47–1 and 47–2). The selection of an appropriate access site is critically important. Quite often, it is difficult to access an occluded left iliac vein from the right femoral approach. The popliteal vein is used frequently by different investigators for access for iliofemoral vein interventions.[27,28] Access of the femoral vein at mid-thigh under direct duplex guidance is the method preferred by Raju and Neglen.[22-23]

We use the popliteal vein primarily in patients requiring thrombolysis for acute disease, and the internal jugular vein in those with chronic occlusion of the iliofemoral veins. In order to facilitate access into a chronically occluded iliac vein from the internal jugular, we use a 5F Glide catheter (Terumo Medical Corporation, Somerset, NJ) and an angled, stiff, hydrophilic glidewire. Access is thus accomplished invariably. The spinning of the wire between dry, gloved fingers during its slow advancement, combined with angiographic views obtained intermittently, offers a safe method of wire advancement.[26, 27] Contrast also is injected after penetrating the obstruction in

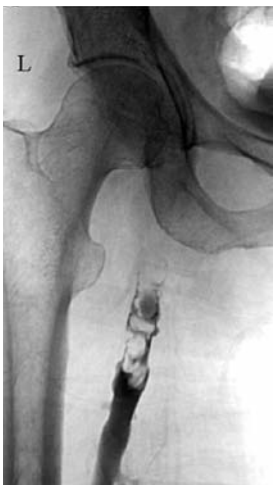

Figure 47-1. Venogram of a 72-year-old female with deep vein thrombosis. Thrombus extends from the femoral vein to the inferior vena cava (IVC). (By permission of Mayo Foundation, 2004.)

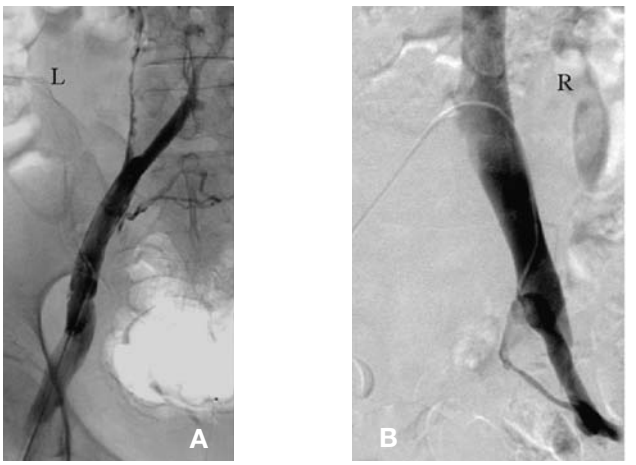

Figure 47-2. A. Following mechanical thrombectomy and 16 hours of infusion of thrombolytic agent the femoral, common femoral and iliac veins are cleared of thrombus but severe chronic narrowing of the common iliac vein remains with a 18 mmHg pressure gradient from the external iliac vein to the IVC. **B.** The contralateral common iliac vein (CIV) and the IVC are free from thrombus. (By permission of Mayo Foundation, 2004.)

order to verify the positioning of the wire in relation to the inflow branches and to identify residual venous occlusion. The patient is heparinized with a bolus of 5,000 IU intravenously as soon as access has been secured. The target activated clotting time is 280 to 300 seconds. Heparin is repeated during the procedure as required.

The occluded segment is dilated first to the planned stent diameter, usually 12 mm for the common femoral and external iliac veins and 14 to 16 mm for the common iliac vein. For the inferior vena cava, 16 to 20 mm stents are deployed. Prestent dilatation is performed with a large balloon, although a smaller balloon (5 mm) may have to be used initially before a larger balloon can be negotiated. If a catheter or balloon cannot be advanced into the vessel, although the wire has been successfully negotiated, a puncture can be made into the vessel distally and the wire snared and pulled through the puncture site. By applying tension to both ends, the catheter and balloons are effectively pulled through. Stents are deployed after predilation. The entire diseased segment of the vein, and most often the entire length of common and external iliac veins is stented in continuity (Figure 47–3). We do not hesitate to cover the common femoral vein distal to the inguinal ligament but preserve all or most large venous collaterals. Recanalization of the inferior vena cava bifurcation poses a technical challenge. Most commonly, only one common iliac vein has occlusion and the lesion usually extends all the way to the ostium of the inferior vena cava. Because of the expansive strength of balloon-expandable stents being weakest at the tips, the stent is deployed overhanging into the inferior vena cava by 10 to 15 mm (Figure 47–2). Every effort is made to ensure that the contralateral common iliac vein orifice is not obliterated by the stent, thus preventing contralateral deep venous stasis and thombosis. At the end of the procedure, the introducers from either the internal jugular or popliteal veins are removed and compression is applied for 5 to 10 minutes. In jugular vein access, the head of the bed is elevated to 30 degrees for two hours; no other care is needed. In popliteal vein access, small gauze rolls are applied against the popliteal fossa, creating pressure with elastic, or preferably foam tape. Heparin is not reversed.

The leg is wrapped with an elastic bandage immediately after the procedure and the patient is assisted to ambulate as soon as the sedation has worn off. Full anticoagulation, usually low-molecular weight heparin followed by oral anticoagulation, is commenced. The patient is observed in the hospital overnight and ultrasound is performed the

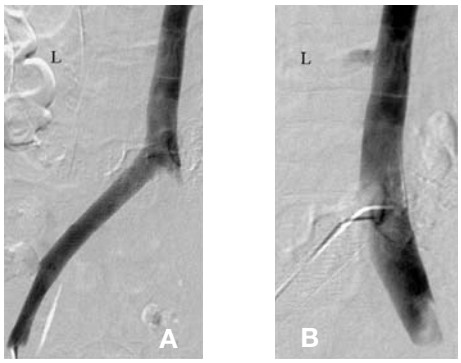

Figure 47-3. A. The CIV was dilated to 12 mm and a 14 mm wide, 60 mm long Wallstent® placed from the EIV to the IVC junction. Care was taken not to place the stent too far into the IVC but still having the stent cover the stenotic segment. **B.** A venogram of the contralateral CIV confirmed no overriding of the stent into the IVC or right CIV and patent IVC. The pressure gradient was now 0 mm Hg. (By permission of Mayo Foundation, 2004.)

following day to verify patency. The length of anticoagulant therapy depends on the presence of risk factors such as thrombophilia, which may require indefinite anticoagulation. However, if reversible factors were the cause of the thrombosis and a good endovascular outcome has been achieved with stent placement, three to six months of anticoagulation are sufficient. For patients with chronic disease in whom venous stents were deployed, clopidogrel (Plavix, Sanofi-Synthelabo, New York, New York) 75 mg/d is prescribed for four weeks, and aspirin, 81 mg/d, is recommended indefinitely.

STENT SELECTION

Both balloon-expanding and self-expanding stents can be used for large vein reconstructions.

Balloon-expanding Stents

The stent is mounted on a balloon and carried to the site of deployment; the covering sheath is removed and the stent is deployed by balloon inflation. It is the diameter of the balloon that determines the diameter of the stent. Each stent has a recommended range of diameters over which it can be expanded. With dilation, the stent shortens somewhat but this is minimal if the expansion is within the recommended range. If the stent is dilated more than recommended, foreshortening becomes important. Balloon-expandable stents have high radial force: they are usually used when recoil is strong and extra radial force is needed.

A typical balloon-expandable stent is the Palmaz stent (Cordis Corporation, Miami, Florida). Because balloon-expandable stents are malleable, they do not re-expand if bent or crushed. Balloon-expandable stents can be deployed only in locations protected from external physical forces.

Self-expandable Stents

Self-expandable stents conform better to curved vessels, and deployment is generally easier. They re-expand if compressed or crushed. Compared to balloon-expandable stents, self-expanding stents are available in longer lengths and larger diameters.

The first stent in general use was the Gianturco stainless steel stent (Cook Incorporated, Bloomington, Indiana) which is still commercially available. This stent is used in large veins, especially the inferior vena cava, since large-diameter stents (up to 25 mm) are available. Also, this stent has large spaces between the interstices that allow inflow from side branches into the stent lumen, making it possible to stent across the renal veins without compromising inflow. Most other stents, both self-expandable and balloon-expandable, have tight interstices and, thus, should not be placed across the ostium of an important inflow vessel.

Self-expandable stents include the Wallstent® (Boston Scientific Corporation, Natick, Massachusetts) (Figure 47–3), made of stainless steel; the Smartstent® (Cordis Corporation, Miami, Florida), made of nitinol; the Luminex® (Angiomed/Bard, Karlsruhe, Germany), made of nitinol; and the Zilver® (Cook Incorporated, Bloomington, Indiana), made of nitinol. The Wallstent is available in diameters from 3.5 to 22 mm with even-number sizes beginning with 6 mm. The Smartstent® is available in diameters up to 14 mm. Other smaller stents, like the Luminex® stent in diame-

ters up to 12 mm and the Zilver® stent in diameters up to 10 mm, are too small to use for the common iliac vein and the inferior vena cava.

Most self-expandable stents foreshorten minimally on placement. Only the Wallstent shortens to any marked degree, and for operators who are not used to the product, this may cause difficulty. To counter foreshortening, the Wallstent may be partially (more than $^2/_3$) deployed and then recaptured for repositioning or removal. This capability is often helpful and overcomes the limitation of foreshortening. As mentioned before, predilation before deployment is important. Overdilation, before or after deployment, should be avoided. The stent will decrease to its recommended size if overdilated; therefore, overdilation can cause migration of the stent.

RESULTS OF STENTING OF ILIOFEMORAL VEINS AND THE IVC

Technical Success

Technical success rates of iliac vein recanalizations is reported to range from 87–100%.[9,23,29] Most failures have been encountered in patients with chronic long-segment occlusion of the iliac veins. Poor inflow also can be a reason why stenting has been abandoned in some patients. Technical improvements and the recent introduction of open surgical recanalization of the femoral veins, combined with iliofemoral stenting, have increased the number of patients in whom a less invasive recanalization is the treatment of choice.

Complications

Early complications are rare, although early reocclusions are reported to range from 0–10%. O'Sullivan et al reported two early reocclusions in 35 patients, both of whom received successful treatment with thrombolysis.[5] Retroperitoneal hematoma may occur occasionally but it settles with conservative treatment, and blood transfusion if required.[9] Nazarian et al[2] identified structural fractures in two Gianturco stents, incidentally. Doslouglu et al[30] reported one case of stent infection following thrombolysis and stent deployment for May-Thurner syndrome. In the Mayo Clinic series comprising 63 patients, postprocedural hematoma at the puncture site developed in two patients, once after thrombolysis of an occluded stent and a second time immediately after angioplasty, and stenting following prolonged heparin therapy. No death occurred in the postoperative period (30 days). We encountered no retroperitoneal hematomas nor bleeding ruptures.[12] Juhan et al[31] described a single case of a crushed Palmaz stent that had been deployed in the common iliac vein for stenosis[31] The patient was pregnant, and the stented venous segment thrombosed shortly after stent deployment.

Clinical Success and Patency

Clinical success includes decreased pain, decreased leg swelling, healing of venous ulceration, improved ambulation, and improvement in the quality of life. Since long-term results are currently not available, only early and mid-term results can be analyzed in some detail. Data on cumulative patency, because of the small number of patients reported in most series, should be accepted with caution. Primary and secondary patency rates in selected series are depicted in Table 47–1.

TABLE 47-1. REPORTED RESULTS OF ILIOFEMORAL AND INFERIOR VENA CAVA STENTING

1st Author, Year	N of Patients	Etiology	Throm-bolysis	Throm-bectomy	Stenting	Primary	Secondary
Nazarian, 1996[2]	56	Stenoses	-	-	Ilio-caval, femoral	50% at 1 and 4 years	81% at 1 year 75% at 4 years
Binkert, 1998[3]	8	Venous spurs	-	4	Left common iliac	100% at 3-years	
Blattlet, 1999[4]	14	Post-thrombotic	-	-	Iliac	79% at 15-months	
O'Sullivan, 2000[5]	39	May-Thurner	31	-	Left common iliac	87% at 30-day	79% (1 year)
Patel, 2000[6]	10	May-Thurner	10	-	Left common iliac	90% at 1 month 60% at 18 months	100% at 1 month 100% at 18 months
Neglen, 2000[7]	59	May-Thurner	-	-	Left common iliac	60% at 2-years	100% at 2 years
	78	Post-thrombotic	2	-	Ilio-caval	52% at 2 years	90% at 2 years
Abu Rahma, 2001[8]	18	Post-thrombotic	18	-	Iliac	83% at 1 year 69% at 5 years	
Hurst, 2001[9]	18	May-Thurner	6	-	Ilio-caval	89% at 6 months 79% at 12 months	
Lamont, 2002[10]	15	May-Thurner	6	3	Left common iliac	93%, 87% (6, 16 months)	100% (6-month)
Neglen, 2004[11]	455	Post-thrombotic and May-Thurner	-		Iliac, iliofemoral, inferior vena cava	75% at 3 years	93% at 3 year
Paulson,[12]	41	Post-thrombotic	13	7	Ilio-caval	61%, 58% (3, 6 months)	83% at 3 months 76% at 6 months

Patency in general correlates well with clinical improvement, although improvement is much more obvious in patients without infrainguinal obstruction or incompetence. In a group of 38 patients treated by Raju et al early in their experience,[32] 74% had relief from pain and 66% had improvement in the swelling and healing ulcer/stasis dermatitis at 12 months. Hurst et al reported early clinical improvement in only 47% of patients treated with angioplasty and stenting for iliocaval compression syndrome.[9]

The reported primary patency at one year reported for iliocaval venous stenting ranges from 49–100% (Table 47–1)[2-12] The wide range is explained by the heterogeneity of the patient groups and differences in procedures (stent versus angioplasty), underlying diseases (acute versus chronic presentation), and follow-up period. Secondary patency has been better, ranging from 75–100%, with a follow-up of six months to four years.

More detailed analysis of some of the published studies is worthwhile. The efficacy of stents placed intravenously in a large series of 56 patients (59 stenoses or occlusions) over a six-year period for treatment of stenoses and occlusions was reported in 1996 by Nazarian and Bjarnason.[2] Stent sites included the inferior vena cava (n = 10) and common iliac (n = 31), external iliac (n = 46), common femoral (n = 27), and superficial femoral veins (n = 4). Indications included obstruction from pelvic malignancy, trauma, surgery, or idiopathic causes. Primary and secondary one-year patency was 50% and 81%, respectively, and four-year patency 50% and 75%, respectively. Major complications occurred in 6.8% of cases. Significant clinical improvement was documented at one year follow-up.

Blattler et al (1999) investigated 42 symptomatic patients with chronic postthrombotic pelvic venous obstruction (38 had left iliac vein obstruction) with a view to percutaneous transluminal stenting.[4] Stenting was technically feasible in 25 patients (60%), was attempted in 14 patients, and was primarily successful in 12 patients. One stent occluded within the first week. All other stents were patent after a mean follow-up of 15 months (range, one to 43 months). Satisfaction was high in the patients with typical chronic symptoms, but low in those with nonspecific subjective manifestations.

A retrospective analysis of 39 patients (29 women, 10 men; median age, 46 years) with iliac vein compression (May-Thurner syndrome) was conducted by O'Sullivan et al.[5] Nineteen patients had acute deep vein thrombosis. All patients had leg edema or pain. Those with acute DVT received catheter-directed thrombolysis (120,000–180,000 IU urokinase/h) followed by angioplasty and stent placement. Those with chronic symptoms had angioplasty and stent placement alone (n = 8), or in combination with thrombolysis (n = 12). Follow-up was conducted with duplex ultrasound, and a quality-of-life assessment was also obtained. Initial technical success rate was 87% and patency at one year 79%. Symptoms disappeared or partially improved in 85% of patients. Thirty-five of 39 patients received stents. The one-year patency for those with acute DVT who received stents was 91.6%. The one-year patency for patients with chronic symptoms who received stents was 93.9%. Five technical failures occurred. Major complications included acute iliac vein rethrombosis (<24 hours) requiring reintervention on two occasions. Minor complications included perisheath hematomas (n = 4) and minor bleeding (n = 1). There were no deaths, pulmonary embolus, cerebral hemorrhage, or major bleeding complications.

Neglen et. al. compared the results and complications of endovascular surgery in limbs with postthrombotic and nonthrombotic disease.[7] One hundred thirty-nine consecutive lower extremities with chronic iliac venous obstruction, 61 limbs with primary disease (May-Thurner Syndrome), and 78 with postthrombotic disease were treated with balloon dilation and stenting. No mortality was reported. Nonthrombotic complication rate was 3%. Postoperative (8%, $^{6}/_{78}$) and late occlusion (3%, $^{2}/_{69}$) occurred only in postthrombotic limbs. Primary, primary-assisted, and secondary cumulative patency of the stented area at two years was 52%, 88%, and 90%, respectively, in the postthrombotic group as compared to 60%, 100%. and 100% in the May-Thurner group, respectively. The authors detected a significant clinical improvement in pain and swelling in both groups. Half of the active venous ulcers healed after the procedure.

Evaluating the outcomes in acute iliofemoral deep vein thrombosis, Abu Rahma et. al.[8] reported a 30-day venous patency and symptom resolution in one of 33 patients (3%) treated with conventional therapy (Group 1), versus 15 of 18 (83%) among those who had lysis, and then went on to have angioplasty and stenting, if needed (Group 2). Cumulative primary patency was 24%, 18%, and 18% at 1, 3, and 5 years, respectively, for Group 1 and 83%, 69%, and 69% for Group 2, respectively. Long-term symptom resolution was achieved in 10 of 33 patients (30%) in Group 1 versus 14 of 18 (78%) in Group 2.

Hurst et al evaluated the results of endovascular treatment in 18 patients with iliocaval compression syndrome over a three-year period.[9] Recanalization and stent placement (n = 17) or angioplasty (n = 1) was achieved in all patients. The average pressure gradient was 5.6 mmHg preprocedure and 0.6 mmHg postprocedure. The primary patency was 89% at 6 months and 79% at 12 months.

The clinical outcomes of iliac venous stent placement in the management of chronic venous disease were reported by Raju et al in a study comprising 304

symptomatic limbs that underwent balloon dilation and stent placement for the relief of iliac vein stenoses.[21] Concomitant saphenous vein ablation was performed in 61 limbs. The patients' median age was 52 years (range, 14 to 83 years). The ratio of post-thrombotic to nonthrombotic limbs was 1/0.9. The clinical scores according to the CEAP stratification were C_2, 24; C_3, 158; C_4, 60; C_5, 13; and C_6, 49. Concurrent venous reflux was present in 57% of the limbs. The authors[21] relied on intravascular ultrasound for defining the degree of iliac vein stenosis in light of the substandard diagnostic ability of transfemoral venography reported previously by their team. The actuarial primary and secondary stent patency rates at 24 months were 71% and 90%, respectively. The median degree of swelling declined from grade 2 (ankle edema) to grade 1 (pitting) after surgery ($p<0.001$). Limbs without edema increased proportionally from 12% before stenting to 47% after stenting ($p<0.01$). Pain, determined on a visual analogue scale (0–10), subsided from a median level of 4 to 0 after stent placement ($p<0.001$). The proportion of limbs completely free of pain increased from 17% before stenting to 71% after stent placement ($p<0.001$).[21] Skin changes or ulceration were present in 69 limbs. The improvement in swelling and pain was similar in ulcerated and nonulcerated limbs. The cumulative recurrence-free ulcer healing was 62% at 24 months. The rate of ulcer healing was similar whether or not concomitant saphenous ablation was performed. The quality of life significantly improved. In light of these findings, reestablishment of iliac vein outflow patency with endovascular therapy appeared to offer significant symptomatic relief. All procedures were performed on an outpatient basis.

In-stent recurrent stenosis (ISR) was recently evaluated by Neglen et al[11] in 324 limbs treated with ilio-caval balloon angioplasty and stent for chronic nonmalignant obstruction. Median stent length was 9 cm (range, 4–35 cm), and median lumen area before and after stenting was 0.41 cm^2 (range, 0–1.65 cm^2) and 1.70 cm^2 (range, 0.65–4.00 cm^2), respectively. Limbs were divided into groups with no ISR, any degree of ISR, greater than 20% diameter reduction, and greater than 50% diameter reduction. At 42 months, only 23% of limbs demonstrated no ISR. Cumulative rate of limbs with greater than 20% diameter reduction was 61%, and of limbs with greater than 50% diameter reduction was 15%. Patient gender or sidedness of the treated extremity did not affect outcome. At 36 months, limbs with thrombotic disease had higher ISR rates than did limbs without thrombotic disease (63% and 41% of limbs with >20% narrowing, and 23% and 4% of limbs with >50% narrowing, respectively; $p<.01$). Similarly, higher rates of ISR were found in patients with thrombophilia and long stents extending below the inguinal ligament. Primary, assisted primary, and secondary patency for the entire population at three years was 75%, 92%, and 93%, respectively.

The outcomes in the Mayo Clinic among 41 patients (27 females, 14 males) treated for iliac vein and vena cava obstruction in the past six years (1998–2003) were presented recently by Paulsen et al.[12] Two thirds of the patients ($27_{/41}$) had a comorbid condition relevant to the occlusion. Twenty two percent ($9_{/41}$) of the patients had May-Thurner syndrome, 20% neoplasms ($8_{/41}$) contained ($2_{/8}$) or disseminated ($6_{/8}$) 12% had a known coagulopathy ($5_{/41}$), and 7% a history of trauma ($3_{/41}$) predisposing to venous occlusion. A total of 43 limbs were stented. $6_{/41}$ patients received treatment for right iliac vein occlusion, $23_{/41}$ for left iliac vein involvement, $7_{/41}$ had bilateral limb treatment and $5_{/41}$ received therapy for primary caudal inferior vena cava obstruction. Each patient received a median number of three stents. The mean venous pressure gradient was 10.5± 1.4 mmHg at presentation and was reduced to 0.93±0.26 mmHg post therapy. Thrombolysis in preparation for the endovascular therapy was required

in $13_{/41}$ patients, and mechanical thrombectomy was performed in $7_{/41}$. Two patients, both with disseminated neoplasms, succumbed before their follow-up investigations. A hematoma in response to prolonged lytic therapy and anticoagulation developed in a third patient. The three-month primary, primary assisted and secondary patency was 61%, 65%, and 83%, respectively (n = 23). At six months, the primary, primary assisted, and secondary patency was 58%, 71%, and 76%, respectively (n = 17).

CONCLUSIONS

The diagnosis and treatment of iliac vein obstruction secondary to acute venous thrombosis, chronic postthrombotic, or nonthrombotic occlusion (May-Thurner syndrome) has significantly improved over the past five years. Endovascular therapy encompassing thrombolysis, angioplasty, and stenting may offer recanalization of the obliterated iliac or iliofemoral veins at low complication rate and with satisfactory early and mid-term patency, ranging from 75 to 100% at one to two years follow-up. Best results are achieved in the presence of patent infrainguinal venous trunks and shorter iliac lesions enabling uncompromised deployment of stents. Stenting of the inferior vena cava or the common femoral vein also can be performed with good patency rates and good mid-term clinical improvement. Open surgery and endovascular expertise can be combined to improve results by adding surgical thrombectomy to

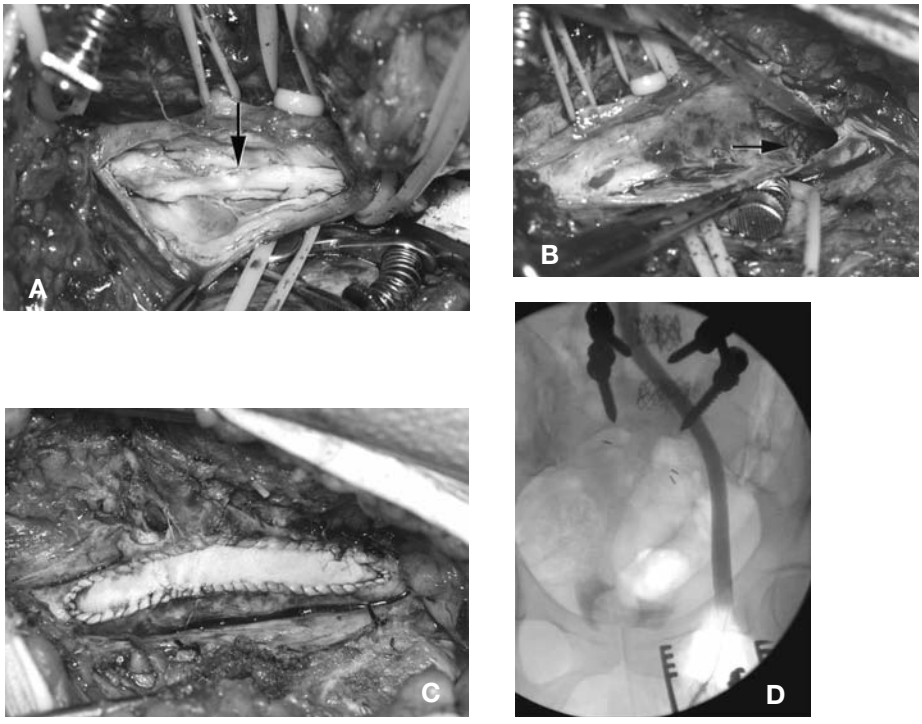

Figure 47-4. Combined endovascular and open reconstruction for chronic iliofemoral venous occlusion. **A.** Note old recanalized thrombus in the common femoral vein. **B.** The old thrombus was excised and the iliofemoral vein was stented with Wallstents. **C.** The femoral vein was closed with bovine pericardial patch. **D.** Postoperative venogram confirms widely patent iliofemoral vein with stents. (By permission of Mayo Foundation, 2004.)

stenting (Figure 47–4). In the Mayo Clinic practice, angioplasty and stenting, with or without thrombolysis, have emerged as a safe and effective first line treatment in the management of complicated ilio-caval, iliac, or iliofemoral obstructions with excellent early and mid-term clinical outcomes

REFERENCES

1. Gloviczki P. Presidential address: Venous surgery-from stepchild to equal partner. *J Vasc Surg*. 2003;38;871–878.
2. Nazarian GK, Bjarnason H, Dietz CA Jr et al. Iliofemoral venous stenoses: effectiveness of treatment with metallic endovascular stents. *Radiology*. 1996;200:193–199.
3. Binkert CA, Schoch E, Stuckmann G, et al. Treatment of pelvic venous spur (May-Thurner syndrome) with self-expanding metallic endoprostheses. *Cardiovasc Intervent Radiol*. 1998;21:22–26.
4. Blattler W, Blattler IK. Relief of obstructive pelvic venous symptoms with endoluminal stenting. *J Vasc Surg*. 1999;29:484–488.
5. O'Sullivan GJ, Semba CP, Bittner CA, et al. Endovascular management of iliac vein compression (May-Thurner) syndrome. *J Vasc Interv Radiol*. 2000;11:823–836.
6. Patel NH, Stookey KR, Ketcham DB, Cragg AH. Endovascular management of acute extensive iliofemoral deep venous thrombosis caused by May-Thurner syndrome. *J Vasc Interv Radiol*. 2000;11:1297–1302.
7. Neglen P, Berry MA, Raju S. Endovascular surgery in the treatment of chronic primary and post-thrombotic iliac vein obstruction. *Eur J Vasc Endovasc Surg*. 2000;20:560–571.
8. AbuRahma AF, Perkins SE, Wulu JT, Ng HK. Iliofemoral deep vein thrombosis: conventional therapy versus lysis and percutaneous transluminal angioplasty and stenting. *Ann Surg*. 2001;233:752–760.
9. Hurst DR, Forauer AR, Bloom JR, et al. Diagnosis and endovascular treatment of iliocaval compression syndrome. *J Vasc Surg*. 2001;34(1):106–113.
10. Lamont JP, Pearl GJ, Patetsios P, et al. Prospective evaluation of endoluminal venous stents in the treatment of the May-Thurner syndrome. *Ann Vasc Surg*. 2002 ;16(1):61–64.
11. Neglen P, Raju S. In-stent recurrent stenosis in stents placed in the lower extremity venous outflow tract. *J Vasc Surg*. 2004;39(1):181–187.
12. Paulsen SR, Misra S, Sabater EA, et al. Iliac-vein and inferior vena cava obstruction: outcome of treatment with endovascular stents. Presented at the 17th Annual Meeting of the American Venous Forum, Kissimmee, FL, February 26–29. 2004.
13. Jost CJ, Gloviczki P, Cherry KJ, et al. Surgical reconstructions of iliofemoral veins and the inferior vena cava for nonmalignant occlusive disease. *J Vasc Surg*. 2001:33:320–328.
14. Gloviczki P, Cho JS. Surgical treatment of chronic deep venous obstruction. In: Rutherford RB (ed). *Vascular Surgery, 5th edition*. W.B. Saunders: Philadelphia; 2000:2049–2066.
15. Kalra M, Gloviczki P, Andrews JC, et al. Open surgical and endovascular treatment of superior vena cava syndrome caused by nonmalignant disease. *J Vasc Surg*. 2003;38(2): 215–223.
16. Bower TC, Nagorney DM, Cherry KJ Jr., et al. Replacement of the inferior vena cava for malignancy: an update. *J Vasc Surg*. 2000;31:270–281.
17. Delis KT, Bountouroglou D, Mansfield AO. Venous claudication in iliofemoral thrombosis: long-term effects on venous hemodynamics, clinical status, and quality of life. *Ann Surg*. 2004;239(1):118–126.
18. Comerota AJ, Throm RC, Mathias SD,et al. Catheter-directed thrombolysis for iliofemoral deep venous thrombosis improves health-related quality of life. *J Vasc Surg*. 2000;32:130–137.
19. Raju S. New approaches to the diagnosis and treatment of venous obstruction. *J Vasc Surg*. 1986;4:42.
20. Neglen P, Raju S. Intravascular ultrasound scan evaluation of the obstructed vein. *J Vasc Surg*. 2002;35:694–700.

21. Raju S, Owen S Jr, Neglen P. The clinical impact of iliac venous stents in the management of chronic venous insufficiency. *J Vasc Surg*. 2002;35:8–15.
22. Neglen P. Raju S. Proximal lower extremity chronic venous outflow obstruction: recognition and treatment. *Semin Vasc Surg*. 2002;15:57–64.
23. Raju S. McAllister S. Neglen P. Recanalization of totally occluded iliac and adjacent venous segments. *J Vasc Surg*. 2002;36:903–901.
24. Neglen P. Thrasher TL. Raju S. Venous outflow obstruction: An underestimated contributor to chronic venous disease. *J Vasc Surg*. 2003;38:879–885.
25. McKusick M, Gloviczki P. Phlebography. In: Rutherford RB (ed). *Vascular Surgery, 5th edition*. W.B. Saunders: Philadelphia; 2000:302–312.
26. Bjarnason H. Endovascular Treatment for Chronic Iliofemoral Venous Obstruction. *Perspect Vasc Endovasc Surg*. 2004; In press.
27. Bjarnason H, Kruse JR, Asinger DA, et al. Iliofemoral deep venous thrombosis: safety and efficacy outcome during 5 years of catheter-directed thrombolytic therapy. *J Vasc Interv Radiol*. 1997;8:405–418.
28. Mewissen MW, Seabrook GR, Meissner MH, et al. Catheter-directed thrombolysis for lower extremity deep venous thrombosis: report of a national multicenter registry. *Radiology*. 1999;211:39–49.
29. Heijmen RH, Bollen TL, Duyndam DA, et al. Endovascular venous stenting in May-Thurner syndrome. *J Cardiovasc Surg* (Torino). 2001;42:83–87.
30. Dosluoglu HH, Curl GR, Doerr RJ, et al. Stent-related iliac artery and iliac vein infections: two unreported presentations and review of the literature. *J Endovasc Ther*. 2001;8:202–209
31. Juhan C, Hartung O, Alimi Y, et al. Treatment of nonmalignant obstructive iliocaval lesions by stent placement: mid-term results. *Ann Vasc Surg*. 2001;15:227–232
32. Raju S. Experience with venous reconstruction in patients with chronic venous insufficiency. In: Bergan JJ, Yao JST, eds. *Venous Disorders*. Philadelphia: W.B. Saunders; 1991:296–305.

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Endovenous Ablation of Varicose Veins

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Superficial venous varicosities are a common clinical entity that can be disabling and cosmetically unappealing for many patients. The spectrum of venous varicosities is broad and they present with varying symptoms such as leg fatigue, bleeding, edema, skin changes, or frank ulceration. Venous varicosities range from minor telangiectasias and reticular veins to severe venous varicosities, each of which correlates to different treatment modalities. The optimal treatment is dependent on the extent of superficial venous disease as well as the presence of deep venous valvular dysfunction. The treatment of varicose veins historically has been with open surgical methods; however, the advent of minimally invasive technology coupled with the use of duplex ultrasonography has transformed the approach to treating axial superficial venous reflux.

The primary disadvantages of traditional saphenous vein high ligation and stripping have been the need for regional or general anesthesia as well as the significant post-operative morbidity. Potential post-operative complications such as hematoma, bruising, soft tissue infection, pain, and saphenous nerve injury can be debilitating and result in recovery times that may be as long as 4-6 weeks.[1] In addition, there is a potential need for further procedures as a result of neovascularization and recurrent varicosities following vein stripping.[2,3] Endovenous ablative techniques avoid many of these major disadvantages associated with open surgical techniques. For instance, endovenous ablative procedures are typically performed in the clinic setting, require no anesthesia other than local tumescent infiltration, and allow the patient to return to normal activities immediately following the procedure. There is minimal post-procedure pain, bruising, scarring, and disability and, thus, high patient satisfaction. Despite these advantages, the long-term efficacy and need for further treatments for recurrent superficial veins or neovascularization remain to be determined.

The current endovenous thermal ablative techniques for superficial venous reflux are radiofrequency ablation (RFA) or endovenous laser ablation (EVLA). Utilizing different energy sources, both of these therapies deliver thermal energy within the lumen of the vein, which results in destruction of the vein wall and subsequent obliteration of the vessel. Alternatively, ultrasound-guided foam sclerotherapy (UGFS)

utilizes a sclerosing detergent to cause chemical destruction of the venous lumen. Each of these modalities has been shown to be effective in treatment of superficial venous reflux with minimal morbidity and with midterm results that are similar to traditional operative techniques.

INDICATIONS FOR ENDOVENOUS ABLATION

Patients who are suitable for ligation and saphenous vein stripping procedures are also suitable for endovenous ablative techniques. Each patient must undergo a detailed duplex ultrasonography (DUS) exam to evaluate for valvular incompetence of the deep, perforator, and superficial venous systems. Absolute contraindications to endovenous treatments would include deep venous obstruction or obliteration, arteriovenous malformations, pregnancy, severe peripheral arterial disease in which compression garments are inappropriate, or bed-bound patients. Relative exclusion criteria are vessels that are too small to accommodate the ablative catheter (<2 mm), extremely tortuous veins, known hypercoagulable states, superficial thrombophlebitis, or partial superficial venous occlusion. Large or aneurysmal vessels (> 25 mm) are a relative contraindication for the RFA procedure; however, there is no size limitation with EVLA treatment. The use of ultrasound-guided foam sclerotherapy (UGFS) is especially useful for tortuous and small veins that are not amenable to EVLA or RFA treatment. In particular, patients who present with extensive neovascularization, patients with segmental reflux in short segments of truncal veins, or patients with extremely superficial veins are excellent candidates for UGFS. UGFS has also been shown to be useful in the treatment of perforator veins and venous malformations where EVLA or RFA are not appropriate options. The major contraindication to UGFS that differs from RFA or EVLA is a known allergic reaction to the sclerosing agent.

RADIOFREQUENCY ABLATION (RFA)

Mechanism of Action and Device Specifics

The US Food and Drug Administration approved radiofrequency ablation (VNUS Closure; VNUS Medical Technologies, San Jose CA) for the treatment of superficial venous reflux in 1999. The principle of RFA is the delivery of thermal energy from a high-frequency alternating current via a catheter positioned within the lumen of the targeted vein. The bipolar catheter positioned within the vein emits 2-4 W, which produces temperatures of 85-120 degrees C; a thermocouple in the catheter constantly measures the temperature within the vein wall, which is transmitted through a feedback loop from the catheter to a separate processor in order to maintain a constant heat.[4] The heat generated denatures the intramural collagen resulting in fibrotic obliteration of the vessel lumen. The heat penetrates 1 mm of tissue and, without adequate tumescent solution, the heat can also be transmitted via conduction.[5] The original VNUS Closure™ device came in two different size electrodes, which were designed to treat vessels up to 8 mm in diameter and between 8-12 mm in diameter. With this first generation device, the operator performed a "pull-back" technique, which required a steady withdrawal of the catheter while observing the probe temperature and adjusting the withdrawal rate of the catheter to maintain a consistent ablative temperature.[6] The newer version, the VNUS ClosureFast™ catheter, was

introduced in 2006 and eliminated the need for a continuous pull-back technique as well as the differing size requirements of the catheter. The ClosureFast™ catheter treats the vein in 7 cm-length segments with a timed application of the thermal energy of 20 seconds for each segment. This method provides a more consistent application of thermal energy as well as an easier technique for the operator.

Procedure Details

For treatment of the great saphenous vein, the patient is positioned in the supine position with the extremity externally rotated and slightly flexed at the knee. For treatment of the small saphenous vein, the patient is positioned in the prone position. The treatment bed should be equipped to allow for Trendelenburg positioning.

After the extremity is prepped in a sterile fashion, an entry needle is used to access the vein under ultrasound guidance. It is recommended to treat the entirety of the refluxing vein, so the access site should be distal to the last refluxing valve if technically feasible. A 7F sheath is then placed within the vein using the Seldinger technique. The radiofrequency catheter is then advanced within the saphenous vein until it is 2 cm distal from the saphenofemoral or saphenopopliteal junction. With respect to the great saphenous vein, the catheter tip should not cross the superficial epigastric vein and should be positioned inferior to the origin of this vein. B-mode ultrasonography is used to ensure exact positioning of the catheter.

Chilled tumescent solution is then infiltrated within the saphenous sheath to serve as a thermal buffer as well as provide local anesthetic around the vein. The solution is a mixture of 1% lidocaine, bicarbonate and epinephrine mixed within 500 cc of 0.9% normal saline. Ultrasonography is used by the operator to direct the tumescent fluid injection in the proper location and in the proper amount. It is recommended to inject approximately 75-100 cc of solution per 10 cm of vein. There should also be at least 1 cm of distance created from the vein to the skin surface, and this can be augmented by more infiltration of superficial tumescent solution. The tumescent fluid not only provides external compression on the vein for improved catheter contact with the vein wall, but it protects the surrounding tissues and nerves from the heat emitted from the catheter.

The patient is positioned in the Trendelenburg position to aid in the decompression of the incompetent vein. For the pull-back technique, the catheter should be withdrawn according to the manufacturer's directions. For the ClosureFast™ catheter, the proximal portion is treated with 2 cycles and the remainder of the vein is treated in 7 cm segments. External compression either manually or with the ultrasound probe may facilitate better contact of the vein and the catheter tip. Care is taken to assess the patient during this portion of the procedure to ensure there is no pain during the ablation portion of the procedure, as that would indicate inadequate infiltration of tumescent solution.

After the treatment is completed, the catheter and sheath are removed and manual pressure is held over the vein entry site. Post-procedure DUS is performed to confirm closure of the vein and patency and compressibility of the common femoral or popliteal vein, depending on the superficial system that has been treated.

The patient is then placed in compression wraps or a compression garment of 30-40 mmHg for at least one week following the procedure. The patient typically undergoes a repeat duplex ultrasound within 72-96 hours post-ablative procedure to confirm the absence of deep venous thrombosis (DVT) and the closure of the treated vein. The patient is instructed to return to normal activities immediately following the

procedure. The patient is also instructed to refrain from heavy lifting, strenuous exercise, prolonged sitting, or immobility for at least 1-2 weeks post procedure.

Results

The results available before 2008 are applicable to the first generation VNUS Closure™ device and not the ClosureFast™ device. Of the three randomized controlled trials for the Closure device, the EVOLVeS study prospectively randomized patients to undergo either vein stripping/high ligation or RFA treatment. Results revealed earlier return to work and less post-operative pain in the RFA group when compared to the open surgical group.[7] The 2 year follow-up of this same study group revealed less recurrence of varicose veins compared to stripping, although it was not statistically significant.[8] Merchant et al treated the largest patient cohort of 1 222 limbs with RFA and reported a five-year occlusion rate of 87.2%.[9] Neovascularization was detected in only 2 patients during the follow-up period. A prospective trial using the ClosureFast™ device revealed an occlusion rate of 99.6% at 6 months with excellent patient satisfaction.[10] A large meta analysis performed for all endovenous treatments revealed an early closure rate of 89% and a 3-year closure rate of 80% for RFA-treated superficial veins.[11] Even though this was more efficacious than UGFS and open surgery, it was not more successful than EVLA treatment.

Major complications related to RFA are deep venous thrombosis, skin burns, superficial thrombophlebitis, paresthesias, hematoma, and bruising. Merchant et al reported a 0.9% incidence of DVT, 1.2 % for skin thermal injury, and a 2.9% incidence of phlebitis in their large patient cohort. [9] These authors also reported the incidence of paresthesia to be 2.6% at 5 years following the RFA procedure for the great saphenous vein but 9.5% at 6 months for the small saphenous vein. In the most recent trial using the ClosureFast™ device, complications of DVT or thermal skin injury occurred in no patients.[12] Overall, the major complication rates are low and the procedure is well tolerated by patients.

ENDOVENOUS LASER ABLATION (EVLA)

Mechanism of Action and Device Specifics

Endovenous laser ablation was brought to the forefront in 2001 with the publication of the results by Navarro et al, which revealed excellent results using EVLA for the treatment of great saphenous reflux using the 810 diode laser.[13] Similar to RFA, EVLA is performed by the percutaneous insertion of a catheter that delivers thermal energy within the lumen of the vein. This thermal energy damages the endothelium and leads to occlusion and elimination of the incompetent truncal vein from circulation. Lasers used for EVLA are diode solid-state lasers with differing wavelengths (810 nm, 940 nm, and 980 nm) or 1320 nm of 1470 nm Nd:YAG (Neodymium-doped yttrium aluminum garnet) solid-state laser. There is no consensus on the exact mechanism of action on which the laser energy destroys the vein wall. It is known that the laser thermal energy induces a "boiling blood" effect within the vein lumen. The laser-emitted wavelengths are absorbed by the hemoglobin of the red blood cells, leading to steam bubble creation within the vein lumen; with the Nd:YAG laser the absorption of the energy is via water and not hemoglobin. This steam bubble creation is thought to induce conductive heat that results in irreversible damage to the vein wall at the catheter tip and for an extended length within the vein lumen. This damage

then causes the vein wall collagen to shrink, leading to luminal occlusion. To further substantiate this effect, Proebstle et al evaluated the histopathological results of laser-treated veins and demonstrated focal coagulation necrosis and denudation of the intima with fibrin deposition in addition to vein wall perforation and disruption.[14] These same authors propose that there is a linear effect with steam bubble creation and laser energy emitted, which does not correlate with the laser wavelength. In a recent prospective, randomized trial comparing the 980 nm laser with the 1470 nm laser, the authors reported reduced pain, ecchymosis, and paresthesias along with improved patient satisfaction with the 1470 nm laser treatment.[15] A confounding variable was the different laser fiber on each catheter. The authors suspect that using a radial versus a bare-tip fiber might also affect the outcomes. In all, the use of the EVLA technique is efficacious regardless of the type of laser used, as long as the energy delivered allows for the steam bubble reaction to occur to distribute heat within the vein wall. There is still debate on which laser wavelength provides the best effect with the least post-operative bruising and pain.

Procedure Details

As with any endovenous procedure, preoperative vein DUS must be performed to identify the refluxing vein and the extent of the vein that requires treatment. Patient positioning, sterile preparation of the target limb, and ultrasound-guided venous access is performed in the same manner as the above-described RFA procedure. After a 5F sheath is placed in the vein, a .035-inch guide wire is then advanced to the common femoral vein under direct ultrasound guidance. A long 4F or 5F sheath is then advanced over the wire and positioned 1.5-2 cm below the saphenofemoral junction. The laser catheter is then advanced over the wire to the end of the long sheath. For the diode laser, the sheath is then pulled back to expose the laser tip. For the Nd:YAG laser, the entire long sheath is removed. The tumescent solution is then infiltrated in the same manner and the same volume as recommended for the RFA procedure. Ultrasound verification on the laser tip position is performed an additional time prior to activating the laser. The laser energy is then delivered while the operator continuously pulls back the catheter. Each device manufacturer has instructions on the pull-back timing and amount of power delivered. In general, the energy rate should be 50-80 joules/cm for successful closure of the vein.

Following completion of the procedure, the catheter and sheath are removed and manual pressure is held over the vein entry site. Compression wraps or class II compression garments (30-40 mmHg) are then placed on the patient and recommended to be worn for at least one week following the procedure. The same post-operative precautions are recommended for the EVLA procedure as is for the RFA procedure. The patient returns for a DUS to evaluate for vein closure and DVT at approximately 72-96 hours post-procedure.

Results

The occlusion rate for EVLA is excellent, with many studies reporting great saphenous vein ablation rates over 90%.[13,16,17] A recent large meta analysis reports a 5-year success rate of 95.4% for EVLA, which supersedes that for all other methods of endovenous ablative techniques.[11] Recurrence rates or neovascularization in the long term are not readily available, as most large studies are with follow-up limited to less than 5 years. It has been shown that early post-operative satisfaction is higher in those patients undergoing EVLA versus vein stripping.[18,19]

As with RFA treatments, there is a risk of major complications such as DVT or skin burns. Skin burns typically do not occur when adequate tumescence is utilized. The incidence of DVT has been reported to be less than 1% in some studies, while others have reported no events of DVT.[13,16,17,20] Phlebitis and bruising have been reported to occur as well, but these complications typically subside within the first days to weeks following the procedure. Often patients describe a "pulling sensation" overlying the treated vein as well. This sensation also tends to fade with time and is likely related to the fibrotic changes in the vein. Cutaneous nerve injury is more troublesome for the patient and is reported to occur with a frequency of 1%-10%. This can be decreased with adequate tumescent solution infiltration. A higher incidence of paresthesias has been associated with treatment of the great saphenous vein in the below-the-knee position or the small saphenous vein.

ULTRASOUND-GUIDED FOAM SCLEROTHERAPY (UGFS)

Mechanism of Action

The technique of foam sclerotherapy involves injection of a sclerosing detergent agent that has been mixed with air within the lumen of the vein, which induces a sclerosing effect on the vein intima as well as thrombosis, and results in subsequent venous occlusion. The technique most commonly used is one described by Tessari.[21] Two 5 mL syringes, one syringe filled with 1 part sclerosant (polidocanol or sodium tetradecyl sulfate×) and the other syringe is filled with 4 parts air, are connected with a 3-way stopcock. The operator then passes the air and liquid between the 2 syringes in a rapid fashion approximately 20 times to create a foam mixture. The concentration of the sclerosant (1%-3%) is chosen in relation to the size of the vein being treated, with higher concentrations typically used for larger caliber veins measuring 5-8 mm in diameter. The use of the foam solution is more effective than liquid sclerosant, as it allows for more contact of the sclerosant with the venous intima, thus enhancing the effect of the chemical irritation.

Procedure Details

Depending on the location of the vein being treated, the patient is positioned comfortably so that the target vein can be easily accessed by the operator and so the limb can be elevated after injection. The limb is prepped in a sterile fashion and the vein accessed with a butterfly needle or a catheter under direct ultrasound guidance. It is advised to access the vein in its most distal portion that has demonstrated reflux so that the entirety of the vein receives the forward flow of the foam sclerosant. If the treatment length is extensive, then placement of multiple access sites has been described with injection of small amounts of foam along the length of the vein. Once venous return has confirmed the intraluminal position of the needle or catheter, the prepared foam sclerosant is injected under continuous ultrasound guidance, this allows the operator to visualize the intravascular injection of the foam. After the injection is complete, it is useful to elevate the limb and massage the foam into tributaries to treat the entire venous network. The limb is then maintained in the elevated position for 5-10 minutes with cotton balls or foam pads placed for additional external compression over the treated varicosity. The patient is then placed in a compression wrap or a class II compression garment. The patient is advised to wear the garment and to avoid strenuous exercise, heavy lifting, or prolonged immobility for 1-2 weeks post-treatment.

A repeat evaluation and possible repeat injection is then planned to occur in the following 2-6 week time period.

Results

The most common complications following UGFS are thrombophlebitis and skin hyperpigmentation; however, skin necrosis has been reported. Thrombophlebitis can be remedied with clot extraction or anti-inflammatory medications in combination with warm compresses. Hyperpigmentation overlying the vein fades with time. DVT is an extremely rare complication of foam sclerotherapy.[22] Other complications such as visual disturbances or migraine headaches have been reported to occur in 2% of patients and are self-limiting without long-term sequelae. Chest tightness and cough have also been reported. These complications may be related to the volume injected. The exceedingly rare complication of stroke may occur in patients with a patent foramen ovale.

A large study by Smith et el reported an 88% closure rate of the great saphenous vein at a mean follow-up of 11 months with the use of foam sclerotherapy.[23] Conversely, Ouvry et al reported only a 53% great saphenous vein occlusion at 3 years.[24] The meta-analysis by van den Bos et el reported a 73.5 % success rate at 5 years with UGFS.[11] Overall, many small studies have been conducted revealing various results, but none seem to be as effective as EVLA, RFA, or surgery for the ablation of truncal varicosities.

CONCLUSION

Endovenous ablative procedures have been shown to be as efficacious as traditional saphenous vein ligation and stripping in the short- and mid-term for the treatment of superficial venous reflux. These procedures can be performed in the clinic setting with local anesthesia and allow the patient to return to normal activity immediately. Complication rates are low and patient satisfaction is high with these minimally invasive procedures. Further study of superficial venous treatments including randomized, controlled trials is required to compare all treatments in terms of efficacy, morbidity, cost, and patient satisfaction.

REFERENCES

1. Rautio T, Ohinmaa A, Perälä J, et al. Endovenous obliteration versus conventional stripping operation in the treatment of primary varicose veins: a randomized controlled trial with comparison of the costs. *J Vasc Surg*. 2002;35(5):958–965.
2. Jones L, Braithwaite B, Selwyn D, et al. Neovascularisation is the principal cause of varicose vein recurrence: results of a randomised trial of stripping the long saphenous vein. *Eur J Vasc Endovasc Surg*. 1996;12(4):442–445.
3. Winterborn R, Foy C, Earnshaw JJ. Causes of varicose vein recurrence: late results of a randomized controlled trial of stripping the long saphenous vein. *J Vasc Surg*. 2004;40(4): 634–639.
4. Gohel M, Davies AH. Radiofrequency ablation for uncomplicated varicose veins. *Phlebology*. 2009;24 Suppl 1:42–49.
5. Morrison N. VNUS closure of the saphenous vein. In: Bergan JJ, ed. The Vein Book. Maryland Heights, MO: *Elsevier Academic Press*; 2007:283–290.

6. Stirling M, Shortell CK. Endovascular treatment of varicose veins. *Semin Vasc Surg*. 2006; 19(2):109–115.
7. Lurie F, Creton D, Eklof B, et al. Prospective randomized study of endovenous radiofrequency obliteration (closure procedure) versus ligation and stripping in a selected patient population (EVOLVeS Study). *J Vasc Surg*. 2003;38(2):207–214.
8. Lurie F, Creton D, Eklof B, et al. Prospective randomised study of endovenous radiofrequency obliteration (closure) versus ligation and vein stripping (EVOLVeS): two-year follow-up. *Eur J Vasc Endovasc Surg*. 2005;29(1):67–73.
9. Merchant RF, Pichot O, Closure Study Group. Long-term outcomes of endovenous radiofrequency obliteration of saphenous reflux as a treatment for superficial venous insufficiency. *J Vasc Surg*. 2005;42(3):502–509; discussion 509.
10. Proebstle TM, Vago B, Alm J, Göckeritz O, Lebard C, Pichot O. Treatment of the incompetent great saphenous vein by endovenous radiofrequency powered segmental thermal ablation: first clinical experience. *J Vasc Surg*. 2008;47(1):151–156.
11. van den Bos R, Arends L, Kockaert M, et al. Endovenous therapies of lower extremity varicosities: a meta-analysis. *J Vasc Surg*. 2009;49(1):230–239.
12. Almeida JI, Kaufman J, Göckeritz O, et al. Radiofrequency endovenous ClosureFAST versus laser ablation for the treatment of great saphenous reflux: a multicenter, single-blinded, randomized study (RECOVERY study). *J Vasc Interv Radiol*. 2009;20(6):752–759.
13. Navarro L, Min RJ, Boné C. Endovenous laser: a new minimally invasive method of treatment for varicose veins—preliminary observations using an 810 nm diode laser. *Dermatol Surg*. 2001;27(2):117–122.
14. Proebstle TM, Lehr HA, Kargl A, et al. Endovenous treatment of the greater saphenous vein with a 940–nm diode laser: thrombotic occlusion after endoluminal thermal damage by laser-generated steam bubbles. *J Vasc Surg*. 2002;35(4):729–736.
15. Doganci S, Demirkilic U. Comparison of 980 nm laser and bare-tip fibre with 1470 nm laser and radial fibre in the treatment of great saphenous vein varicosities: A prospective randomised clinical trial. *Eur J Vasc Endovasc Surg*. 2010, in press (online May 2010).
16. Ravi R, Rodriguez-Lopez JA, Trayler EA, et al. Endovenous ablation of incompetent saphenous veins: a large single-center experience. *J Endovasc Ther*. 2006;13(2):244–248.
17. Agus GB, Mancini S, Magi G, et al. The first 1000 cases of Italian Endovenous-laser Working Group (IEWG). Rationale, and long-term outcomes for the 1999-2003 period. *Int Angiol*. 2006;25(2):209–215.
18. Rasmussen LH, Bjoern L, Lawaetz M, et al. Randomized trial comparing endovenous laser ablation of the great saphenous vein with high ligation and stripping in patients with varicose veins: short-term results. *J Vasc Surg*. 2007;46(2):308–315.
19. Mekako AI, Hatfield J, Bryce J, et al. A nonrandomised controlled trial of endovenous laser therapy and surgery in the treatment of varicose veins. *Ann Vasc Surg*. 2006;20(4):451–457.
20. Min RJ, Khilnani N, Zimmet SE. Endovenous laser treatment of saphenous vein reflux: long-term results. *J Vasc Interv Radiol*. 2003;14(8):991–996.
21. Tessari L. Nouvelle technique d'obtention de la sclera-mousse. *Phlébologie*. 2000;53: 129–133.
22. Guex JJ. Complications and side-effects of foam sclerotherapy. *Phlebology*. 2009;24(6): 270–274.
23. Smith PC. Chronic venous disease treated by ultrasound guided foam sclerotherapy. *Eur J Vasc Endovasc Surg*. 2006;32(5):577–583.
24. Ouvry P, Allaert F, Desnos P, et al. Efficacy of polidocanol foam versus liquid in sclerotherapy of the great saphenous vein: a multicentre randomised controlled trial with a 2-year follow-up. *Eur J Vasc Endovasc Surg*. 2008;36(3):366–370.

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Transilluminated Powered Phlebectomy for Varicose Veins

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Varicose veins are a common clinical entity associated with significant symptoms and the potential for complications. While there are a variety of treatment options, including surgical, non-surgical and various combinations, many patients go untreated. This is primarily due to misinformation in both the public and the primary care physician regarding modern treatment efficacy and morbidity.

Traditional treatment strategies of long saphenous vein stripping and injection sclerotherapy are being abandoned by vein specialists in favor of less invasive and more effective techniques.

Transilluminated powered phlebectomy (TIPP) using the TriVex® System is an innovative method of removing varicosities using suction and a rotating blade. Guided by light from a tumescent cannula, the vein resector can accurately and completely remove the varicosities with a minimum of trauma to the surrounding tissues.

The two main goals in the management of varicose vein disease are to treat the cause and the effect of superficial venous hypertension. In addition to removing the result of chronic superficial venous hypertension, i.e. varicose veins, one must also effectively treat the source. A duplex ultrasound performed by an experienced technologist is essential to diagnose the sites of venous incompetence, including incompetent perforating vessels.

Saphenofemoral incompetence or incompetence of a major thigh perforator (Hunter, Dodd) requires removal of the saphenous vein to the level of the knee. This is ideally performed surgically with inversion pin stripping after the introduction of tumescent fluid. The interruption of the branches of the saphenous vein at the junction minimizes the chance for recurrence. An ultrasound of the leg immediately prior to surgery can familiarize the surgeon with any unusual anatomy, minimizing the risk of missing a significant vessel.

Using the TriVex® System (Figure 49–1), the surgeon can remove the varicosities and find and interrupt the perforators at the fascia with ultrasound guidance. The entire procedure is typically completed in 30 minutes or less. Patients usually wake up pain-free as a result of the tumescent anesthetic.

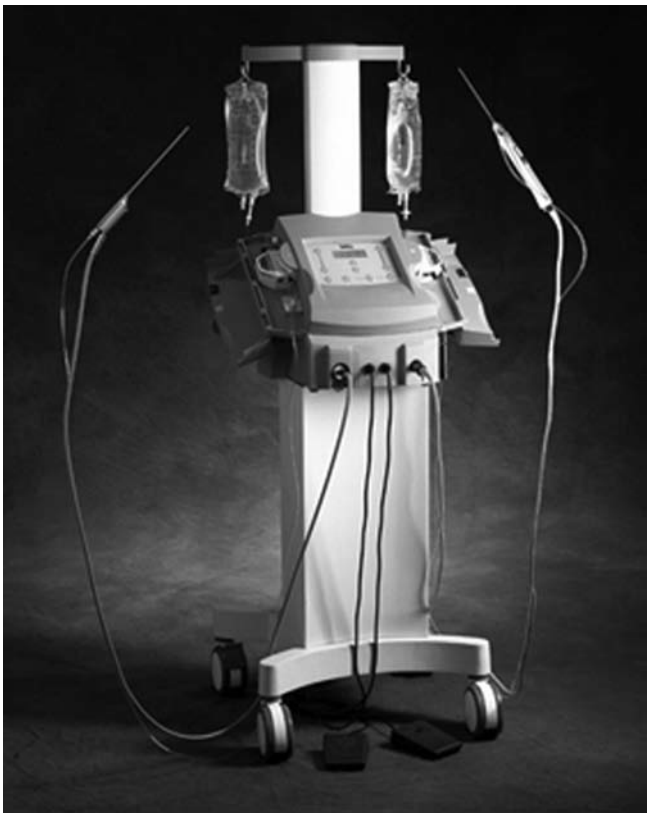

Figure 49-1. TriVex System with tumescent cannula illuminator (left) and motorized hand piece (right).

The utility of this technique has prompted the expansion of indications for the treatment of thrombophlebitis and venous stasis ulcer.

The goal of ambulatory varicose vein surgery is twofold. The first is to stop the causes of venous hypertension, and the second is to remove the superficial venous abnormalities, i.e. the varicose veins themselves.

Removal of varicosities, previously called ambulatory or stab avulsion phlebectomy, has been widely used in the treatment of varicose veins for many years. One of the earliest descriptions of a phlebectomy was by Celsus (56 BC-30 AD). He described how, "... blunt hooks are passed under the vein to hook it. [The veins are] pulled out with the hook and avulsed." Before Celsus, a Roman counsel endured the first recorded varicose vein procedure in 86 BC. He "...endured most excessive torments in the cutting, never either flinching or complaining; but when the surgeon went to the other leg, he declined to have it done, saying, 'I see the cure is not worth the pain.'"[1]

Nothing has substantially changed with multiple incision phlebectomy since these times with the exception of modern anesthesia and antisepsis. The key to success of any minimally invasive procedure, especially varicose vein operations, is that the procedure gives good cosmetic results, provides relief of pain and, and can be done in an outpatient setting. Standard ambulatory phlebectomy operations are still plagued by large numbers of incisions required to remove friable and easily fragmented vein segments. Another major drawback of the avulsion procedure is that it is a blind

operation. With no confirmation of total removal of varicose clusters, there is no visible start or end point to these older procedures.

The conventional procedures have proven to be tedious and impossible to perform in limbs with chronic venous insufficiency and even early lipodermatosclerosis. The operations are particularly difficult in limbs subjected to prior skin and subcutaneous interventions, including surgery or injections. Additionally, there are those who have experienced previous superficial thrombophlebitis with no good surgical option.

Finally, patients with venous ulcers have undergone blind shearing procedures above and below the fascia, neither of which are too rewarding.

PREOPERATIVE EVALUATION

A general medical history and physical examination should be obtained in all cases. Venous insufficiency symptoms, previous treatment, and previous complications of chronic venous insufficiency should be specified and detailed if possible. Our patients fill out a specific vein questionnaire that also includes a space for the patient to delineate their expectations from treatment. The patient is told beforehand to bring shorts to wear for the consultation exam. This makes the patient and examiner much more comfortable. It is desirable to either take photographs at this time or at least draw a diagram of where the problem veins and areas of skin changes are. If a patient has arterial vascular insufficiency, this is documented and should always be diagnosed and treated before any vein problems.

Duplex examination is still the gold standard and should be used as a road map to diagnose and treat vein problems. All of our patients with varicose veins undergo a detailed duplex exam in our office with a certified technician. The duplex exam pays special attention to incompetence of the saphenofemoral junction, as well as all of the named perforators. It is not uncommon to find other abnormalities in the leg, including Baker's cyst, arterio-venous fistulas, and various subcutaneous and muscular abnormalities. With the venous duplex done, a treatment plan is then formulated. This plan should take into consideration the incompetence or absence of incompetence of the greater saphenous vein and location of varicose vein clusters in relation to named perforating veins.

Each patient undergoes an ultrasound duplex examination using reflux diagnostic techniques. Records are made of reflux at the saphenofemoral junction above the knee and below the knee, saphenopopliteal junction, the lesser saphenous vein, and gastrocnemius veins. The deep venous system is studied through the common femoral vein and superficial femoral vein, popliteal vein, and posterior tibial vein. Perforating veins are visualized when possible and, in limbs with chronic venous insufficiency, an exhaustive search for the source is performed.

The patient is given results of the test, and the procedure is explained in detail at this time. All of our patients are fitted for and procure 15 to 20 mm Hg thigh-high stockings prior to scheduling the operation. They are encouraged to wear the stockings for one to two days prior to the procedure in order to get used to them and to avoid being unfamiliar with donning them when it is required postoperatively. Prior to the procedure any special medication indications are taken into consideration. This could include cardiac medication, anticoagulants, and chronic steroid use. Preoperatively, the patient is kept NPO for at least six hours prior to the procedure.

MARKING AND ANESTHETIC

On the day of the procedure, the patient's varicose vein pattern is marked before surgery using an indelible marker with a nylon tip. It is important to have the patient walk or at least stand for 10 to 15 minutes prior to and to remain standing during the actual marking. The marking of the veins need only be a general outline of the affected areas that require resection because transillumination will delineate all the details (Figure 49–2). We take extra care not to place a mark directly over a vein targeted for resection because it could obscure visualization during the procedure. We take this opportunity to review what will take place during the procedure with the patient.

Operations can performed under general, spinal, epidural or local anesthesia. We prefer a "fast track" light general anesthesia using a laryngeal mask airway (LMA) utilizing propofol and a short acting narcotic. Tumescent anesthesia is utilized extensively during the procedure and minimizes the requirement for high doses of anesthetic. Another option is to use local anesthetic with minimal oral sedation. The patient takes 1 mg of Lorazepam (Ativan®) and a suitable dose of a Cox-2 inhibitor orally with a small sip of water upon arrival. A prophylactic dose of a first generation cephalosporin is given after the IV is started. The preemptive effect of these drugs is important to keep the patient comfortable during and after the procedure. After approximately 30 minutes the patient's veins are marked and the skin is prepared and draped for instillation of the local tumescent anesthetic. The local anesthetic is infiltrated by making a 2 ml wheal with a 30-gauge needle and then using a 20-gauge spinal needle attached to the peristaltic pump with the tumescent solution in 1 liter bags. The standard tumescent solution recipe is 1 liter of 0.9 normal saline, 40 cc of 2% lidocaine (800 mg), 2 cc of 1:1000 epinephrine (2 mg) and 10 meq of bicarbonate. The infiltration is done slowly, deep and mid-level, then just under the veins in the subcutaneous tissue where the veins were marked. Careful attention is given so as not to puncture a varicose vein during this infiltration. After this is completed the patient needs to wait at least 5-10 minutes before the anesthetic is completely effective and to

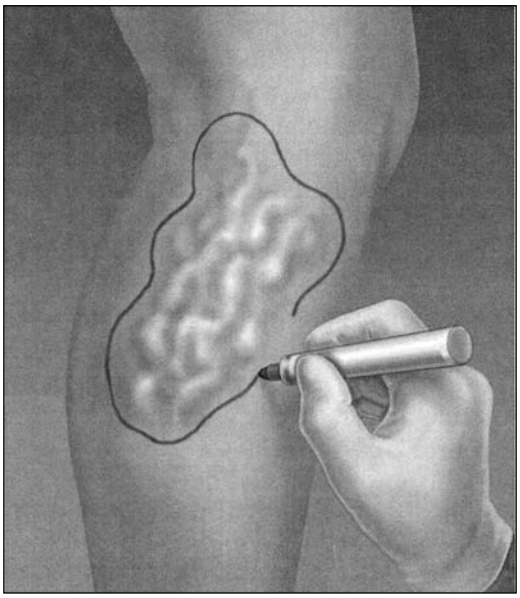

Figure 49-2. Outlining the veins with indelible ink marker.

allow detumescence superficially enough to be able to visualize the veins with transillumination. The safe dose of tumescent anesthetic utilized during all stages of the procedure ranges from 45 to 60 mg per kilogram patient weight. The more obese the patient, the higher the safe dose that can be used. We rarely use more than 2 liters of solution which translates into 1600 mg of lidocaine. An average 70 kg person could have up to 50 mg/kg, representing 3500 mg. Every patient should have their weight documented and maximum dose noted prior to any procedure. The anesthetic bags should be mixed and marked appropriately with how many mg or meq of each component clearly. If a patient is lidocaine allergic, the procedure can be performed without the lidocaine in the mixture. This may require a general anesthetic. Even without lidocaine, it is the saline under tumescent conditions that will supply the adequate anesthetic effect of the infiltrate postoperatively.

SAPHENOUS TUMESCENCE AND INVERSION REMOVAL

Once the appropriate anesthetic is delivered, the patient is taken to the operating room. They are prepared and draped in a standard fashion. We prefer a split-leg table, as it allows access to all parts of the leg to the surgeon and assistant without having to turn the patient.

The operation is performed supine, with the patient in Trendelenberg position. The first part of the procedure is usually to take care of the saphenous vein incompetence if it is present on preoperative duplex. Preemptive tumescent local anesthetic is placed where a 2 to 3 cm groin incision is made. The tumescence here is placed in one stage until the *peau d'orange* effect is seen. The effect is a hydrodissection of the saphenofemoral junction and no bleeding occurs in the skin or subcutaneous tissue while dissecting.

The tumescent anesthetic is also supplied with the Tumescent Cannula Illuminator (TCI®) of the TriVex® System. The device combines a tumescent cannula and beveled transilluminating light. After instillation of the tumescent solution along the entire track of the saphenous vein, it can be removed by inversion without difficulty with the Tumesent Catheter Inversion System catheter placed from the groin to just above or below the knee. The standard tumescent solution recipe is 1 liter of 0.9 normal saline, 40 cc of 2% lidocaine (800 mg), 2 cc of 1:1000 epinephrine (2 mg) and 10 meq of bicarbonate.

After the skin incision, the rest of the dissection can be done with blunt instruments like an atraumatic forceps and right angle clamp. All branches to the saphenofemoral junction are ligated and divided with titanium clips and the junction itself is ligated with a 2-0 absorbable suture. The distal saphenous is removed to just below the knee in general and is only removed further if substantial incompetence or ulcer dictate. Now the tumescent anesthetic is supplied with the Tumescent Cannula Illuminator (TCI®) of the TriVex® System. The device combines a tumescent cannula and beveled transilluminating light. After instillation of the tumescent solution along the entire track of the saphenous vein, it can be removed by inversion without difficulty. After the skin incision, the rest of the dissection can be done with blunt instruments like an atraumatic forceps and right angle clamp. All branches to the saphenofemoral junction are ligated and divided with titanium clips and the junction itself is ligated with a 2-0 absorbable suture. The distal saphenous is removed to just below the knee in general and is only removed further if substantial incompetence or ulcer dictate. We use the Tumescent Catheter Inversion System (InaVein Corp.,

Lexington MA, USA) which removes the vein while simultaneously instilling tumescent anesthesia. The vein is removed with the aid of hydro dissection and and the hemostatic and anesthetic effects of tumescent anesthesia at the same time. Ultrasound guidance is not required during this part of the procedure because the catheter is placed and removed from the area where tumescence is required.

As branches are visualized tugging on the skin, a gentle tap with two fingers will detach them from the inverting vein. If it is a large branch, tumescent solution can be added in this area while removing the vein and tapping. If the saphenous vein is superficial enough, you can visualize along its path with the transilluminating light and see where the posterior arch vein branch comes off. It is usually preferable to go just past the posterior arch vein as it can be the source of calf vein clusters.

Utilizing tumescence and inversion has made the removal of the saphenous vein easier and less traumatic than conventional techniques of stripping. After the saphenous is removed the distal saphenous segment is tied off with a 2-0 absorbable suture and some tumescent solution is added here to decrease the sensation of this (Figure 49–3).

VARICOSE VEIN CLUSTER REMOVAL

After the saphenous portion of the procedure is done, the varicose vein clusters that were outlined prior to surgery are removed. The first incision utilized is usually the distal saphenous incision (just above or below the knee) which is used to transilluminate any

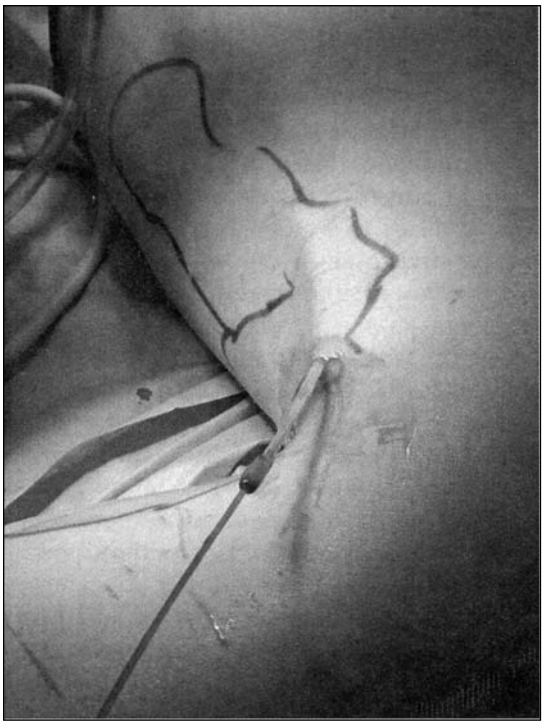

Figure 49-3. Saphenous vein inversion removal with tumescent technique.

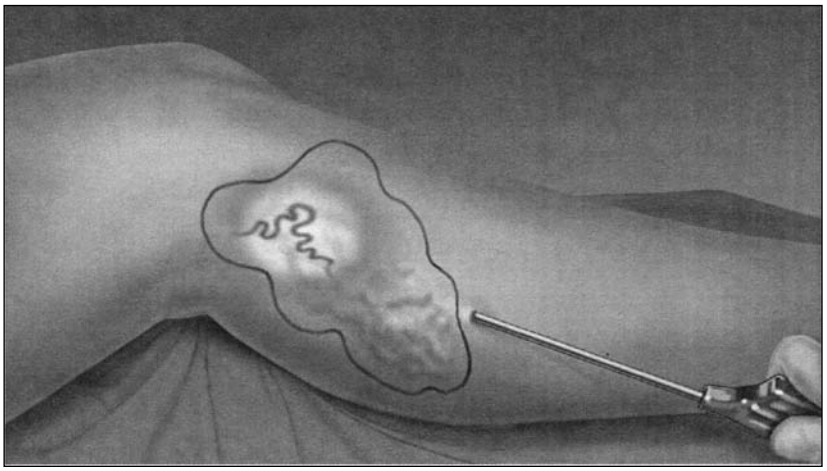

Figure 49-4. First stage tumescence.

clusters (Figure 49–4). If there are extensive clusters in the thigh, labia or abdomen, the groin incision can be used for the transillumination and/or resection as this incision can fit both instruments. The procedure is split into three steps for instructional purposes. All three steps blend into each other during live surgery.

The first step is called first stage tumescence. Using the TCI®, keep the bevel up and stay deep to the veins in the subcutaneous tissue. Tumescent solution is infiltrated to start a hydrodissection and increase the radius of visualization of the veins. The contrast of the subcutaneous tissue with tumescence and just a small amount of blood in the veins will allow better visualization. Very large veins can be resected easier if you use first stage tumescence to empty them of most of their blood. The endpoint of first stage tumescence is when an adequate area of the veins in the cluster outlined is visualized (Figure 49–5). If the tumescence is infiltrated too fast or too superficial, visualization can be obscured. This is easy to avoid, but if it happens, just waiting for the area to detumesce will allow visualization of the veins again.

After first stage tumescence, the actual resection of the varicose veins begins. The Trivex® resector is an electronically-powered computer-controlled oscillating

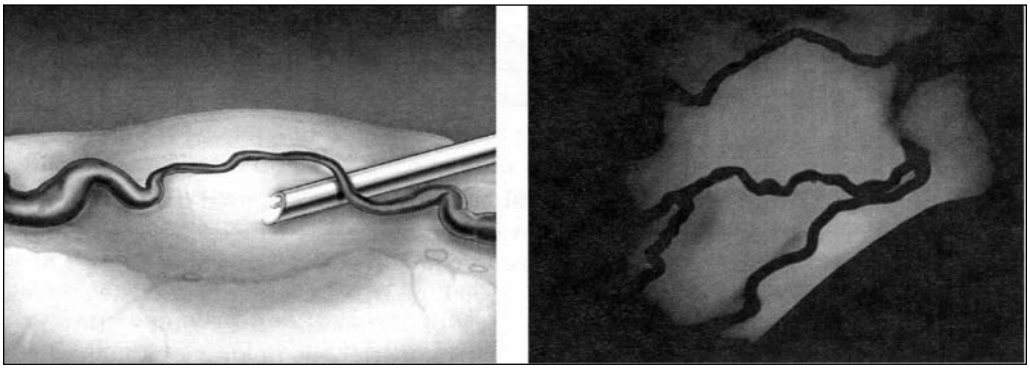

Figure 49-5. The vein is hydrodissected and visualized through the skin.

morcellator with suction applied to it. The disposable blade consists of a tube within a tube design, purpose-built for varicose vein removal. The device is actually a rotating, tubular inner cannula encased in a stationary outer sheath dissector. Irrigation between the inner and outer sheaths keeps the device from clogging with resected tissue. The working tip opening is placed on one side of the sheath and it is through this opening that the vein is suctioned in, morcellated, and removed. The disposable blade is inserted into a standard non-disposable hand-piece which is powered by an electronic motor and controlled by a computer in the control box off the field. Presently, there are two blade sizes available. The smaller blade has a 4.5 mm opening and the larger blade has a 5.5 mm opening. In general, the 4.5 mm blade is used on veins ranging from the smallest to 2 to 8mm. The larger 5.5 mm blade is used on 8 mm veins to ones that are as large as 3 or more centimeters. The preferred speed range for the smaller blade rotation is 200 to 500 rpm and 200-400 rpm for the larger blade. The larger blade is also usually preferred for thicker veins that are either the result of thrombophlebitis or chronic disease including lipodermatosclerosis or previous interventions (including previous surgery or sclerotherapy). Using high powered (-600mmHg)suction and morcellation simultaneously through the hand-piece, the vein is ultimately directed into a suction container using standard wall suction (-180 to -200 mmHg).

Vein resection itself is simple and quick. With the transilluminating light guiding the surgeon, the vein resection blade is placed directly underneath and beside the vein. It is easier to put the blade in as far as possible first and then resect by pulling the resector back towards the surgeon (Figure 49–6). The on/off button for the resector is activated with a simple push of the thumb on the top, and the oscillation and suction

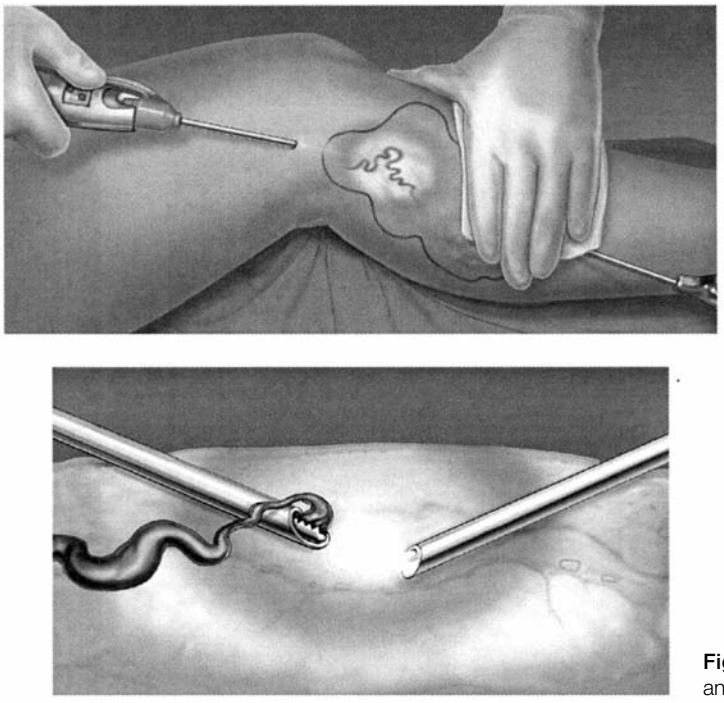

Figure 49-6. The vein is slowly and gently removed.

begin. The resector can be inserted through new or existing 2 to 3 mm incisions. The keys to safety and accurately-controlled resection can be summarized as follows:

1. Resect only if you can see the veins and the resector.
2. Resect only while the skin is kept tight with the non-resecting hand and a sponge. This protects the skin from the blade.
3. Use only enough kinetic energy (pulsed technique) of the rotating blades as needed and utilize suction and tumescent solution during resection.
4. No wrenching of the device is needed.

After first stage tumescence and resection comes second stage tumescence. This is easily performed by moving the already inserted TCI® deep, superficial into the dermis and in a surrounding field-block fashion for approximately 2 cm surrounding the area of resection. The goal is to have a *peau d'orange* effect of the skin and minimal or no blood-staining in the subcutaneous tissue (Figure 49–7). In general, the clusters can be removed in any order, but working from top to bottom seems to work the best as veins connected to perforators are done later in the case and have little or no time to bleed before the compression dressing is placed.

When all the clusters have been resected, this last maneuver just prior to placing the dressing is essential to postoperative patient comfort and to minimize bruising or hematoma. We instill a third stage of tumescent anesthesia with an 18-gauge spinal needle on the tumescent solution tubing with the peristaltic pump attached. A final *peau d'orange* effect is easy to accomplish with dermal and subcutaneous infiltration, as well as complete clearing of the fluid draining from the incisions and punch holes. If areas where perforator branches were interrupted are present, infiltration deeper with guidance of the illuminator will stop the bleeding. All excess tumescence is rolled out with a roll of cotton gauze with a lap pad wrapped around it. The groin incision is closed using a monofilament non-absorbable suture with subcuticular technique. The remaining incisions are not closed if they are 2 to 3 mm or less. If the incisions are open they can drain any blood-tinged tumescent solution and detumesce the leg over the next two to three days.

A two layer dressing is applied from toes to the proximal high thigh. A small amount of antibiotic ointment is placed over each phlebotomy incision. We use a webril wrap directly on the leg with small bolsters of webril over each incision,

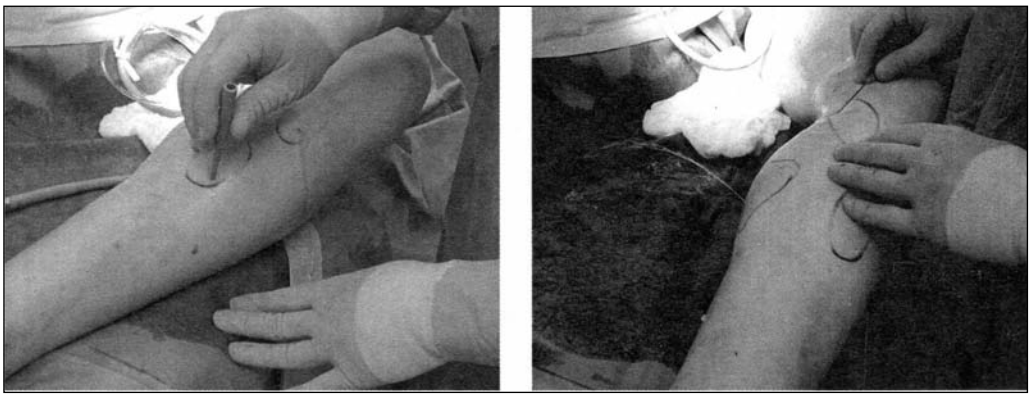

Figure 49-7. Small dermal punch openings help drain fluid postoperatively.

followed by an elastic wrap. The patient is brought back to the "day surgery" room to recover and is usually discharged within one to two hours, ambulating.

POSTOPERATIVE CARE

Patients are advised that temporary bruising and anesthesia in the areas of dissection are expected. With adequate tumescent anesthesia and drainage, patients should have very little discomfort. They are prescribed a suitable Cox-2 inhibitor anti-inflammatory medication, and a narcotic pain tablet is prescribed to be used only as needed. Patients are encouraged to ambulate and to elevate their legs two times a day for 20 to 30 minutes. Dressings are usually removed at 24-48 hours. Starting directly after the dressing removal patients wear a 15 to 20 mm Hg thigh-high graded compression stocking until the bruising is resolved, usually in 1-3 weeks (Figure 49–8). We occasionally utilize postoperative therapeutic ultrasound and electronic stimulation in the areas of resection with patients that had preoperative skin changes from vein disease. These modalities help any collections of blood, bruising and edema to resolve quickly. We have found the treatments make patients more comfortable and hasten the recovery considerably.

SPECIAL CASES: SUPERFICIAL THROMBOPHLEBITIS, PERFORATORS, VENOUS ULCER

With the ease and success of treating varicose veins with TriVex®, we have begun to challenge some common treatment beliefs.

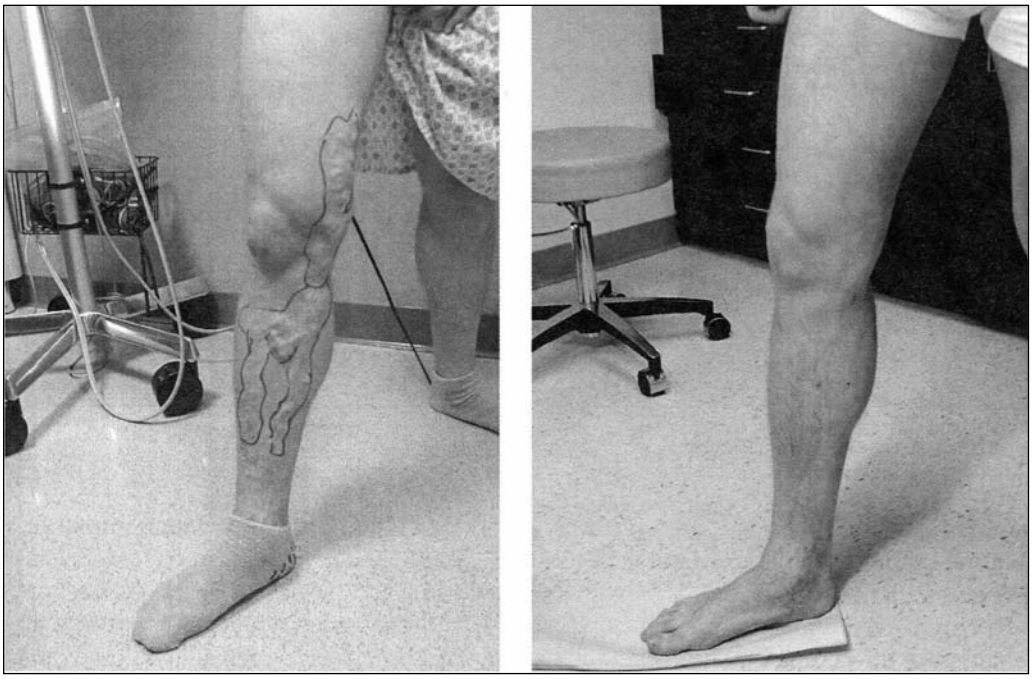

Figure 49-8. Preoperative (left) and postoperative at 4 weeks (right).

Superficial thrombophlebitis

Superficial thrombophlebitis (STP) has an annual incidence of about 125,000 cases a year. It presents as a firm, tender erythematous vein. It is a common complication of varicose veins resulting from venous stasis. Since STP is frequently a limited process, traditional treatment recommendations have included non-steroidals and local heat application. Resolution often occurs in one to three weeks. A more extensive workup, including evaluation of clotting factors and a search for occult malignancy, is traditionally reserved for patients with multiple episodes of STP, especially in the absence of varicose veins.

This benign view of STP is being called into question. Deep venous thrombosis (DVT) and pulmonary embolism (PE) are surprisingly common when STP occurs in the greater or lesser saphenous trunks. In many patients, STP is a manifestation of a systemic disease. Early surgical intervention under local anesthesia has been promoted to reduce local inflammation and pain. Ligation and division of the saphenous vein is indicated when STP approaches the junction to avoid DVT.

Patients with suspected lower extremity STP should undergo prompt duplex ultrasound scanning. The scan should confirm the diagnosis of STP, evaluate for DVT and venous incompetence. Surgical treatment should follow as soon as practical. A thrombosed vein is easily removed with TriVex® for several weeks. A chronically thrombosed vein can be removed through a small incision using blunt dissection after tumescent anesthesia. Patients are given Enoxaparin sodium (Lovenox®) preoperatively as needed to reduce the risk of DVT. A workup for systemic disorder should then be initiated.

This aggressive treatment protocol is designed to treat the local discomfort and to reduce the risk of complications and recurrence.

Venous ulceration

Venous ulceration resulting from chronic venous insufficiency (CVI) has a profound impact on a patient's quality of life and health care economics. Compression with stockings, Unna's boot and pneumatic devices have been recommended along with local wound care, including debridement and growth factors. Surgical intervention is typically reserved for non-healing or multiply recurrent ulcers. Subfascial endoscopic perforator surgery (SEPS), combined with removal of the superficial venous system, has been shown to improve the healing of venous ulceration. The removal of the entire saphenous vein in these cases probably has more effect than the SEPS procedure.

The cause of CVI in patients presenting with venous ulceration must first be evaluated using duplex ultrasound. In patients with superficial venous incompetence, we proceed with surgical intervention early. The saphenous system is addressed as the ultrasound dictates. The incompetent perforating vessels are carefully marked preoperatively using ultrasound. Intraoperatively, the vessels, and their target veins, are ablated with the vein resector. SEPS is reserved for refractory cases and is rarely necessary. A repeat venous duplex ultrasound would be necessary before SEPS to document an incompetent perforator. Valvuloplasty may be indicated in patients with deep venous incompetence who fail to respond to the above measures.

Incompetent perforators

The assessment and treatment of incompetent perforators is critical to long term success in varicose vein treatment. It is important to document the site degree of each perforator during the diagnostic ultrasound. This requires a degree of expertise and patience for an adequate exam.

Incompetent perforators located in the thigh (Dodd, Hunter) are treated with a saphenous vein inversion removal, even in the absence of saphenofemoral incompetence. Incompetent perforators in the leg (Boyd, Cockett) usually connect with the saphenous vein or the posterior arch vein and must be identified and marked just before surgery. The depth and course of the perforating vein should be noted.

Intraoperatively, the perforating vein or its target is transilluminated and ablated with TriVex®. Second stage tumescent anesthetic is introduced to minimize hematoma formation An incision site is usually placed near the perforator to facilitate drainage postoperatively.

RESULTS

Three hundred forty-six (346) patients were studied prospectively in the period of January through March of 2002. The patients all had had a duplex scan and a consultation as described earlier in the chapter. Only 16 procedures were bilateral, as it is our practice to perform one limb procedure at a time in most cases. The only medical contradindication to doing a bilateral procedure would be the amount of tumescent solution needed. This can be done safely and comfortably in these cases by diluting the lidocaine solution to 600 or 700 mg per liter if needed depending on the patient's weight. Three hundred thirty (330) unilateral procedures were done and the average age of the patients was 46. Sixty-three (63) patients were male (18%) and 283 were female (82%). Operative times for a standard saphenous procedure and TriVex® of the entire limb were a maximum of 58 minutes, a minimum of 16 minutes, with an average of 37 minutes. All patients were given a Cox-2 inhibitor for 14 days taken daily and given a prescription for a narcotic pain medication only to be taken if needed (Hydrocodone 5 mg and Acetominophen 500 mg). Ninety-two percent (92%) took the Cox-2 inhibitor alone and no narcotic pain medication. Six percent (6%) took the Cox-2 inhibitor and four narcotic pain tablets over seven days. Two percent (2%) took the Cox-2 inhibitor and six to eight narcotic pain tablets over seven days. Twenty-eight patients (8%) had a small blood or fluid collection under the skin, drained with a dermal punch, with no anesthetic in the office. These were frequently reopening existing incisions that were made during surgery and a small eschar had already closed it. The fluid or blood was most frequently too small to measure but estimated at 1 to 2 ml of blood in the dermal subcutaneous junction. Paresthesia lasting more than three months in an area was seen in six patients (1.7%). Hemosiderin deposition or post inflammatory hyperpigmentation was seen in 12 patients (3.4%). All significant skin changes in these patients resolved. Treatment with bleaching creams and waiting six months or longer was instituted. One seroma and/or cyst was removed under local anesthesia in the office five weeks post-operatively in a patient, adjacent to where the saphenous vein was removed near Hunter's perforator. There were no major complications, including femoral vein injury, post-operative DVT, pulmonary embolism or required reoperation.

ADVANCES AND PROGRESS

Progress and modifications to technique and equipment has taken place as of 2010. In order to maximize efficacy and accuracy and to mimimize collateral tissue damage even for the beginner of the technique.

Franz et al have reported on a retrospective review of 339 patients in a consecutive series of 400 limbs over a 6 year period. With minimal complications and a mean operative time of 19.7 minutes 338 patients (99.7%) reported good outcomes and were satisfied with the procedure. The conclusion included that with proper training and utilization of lower speeds and adequate secondary tumescence good outcomes and high patient satisfaction can be achieved.[2]

The resector profiles have been modified in order to receive and process vein tissue at lower resection speeds utilizing a -600 mm Hg suction pump.

Resection is now always done from just under and to the side of the vein in order to assure that the skin is protected. With speeds of 100-300 rpm or rotational speed with the larger 5.5mm resector for larger veins and speeds of 200-400 rpm for the smaller profile 4.5mm resector any vein can be resected safely and efficiently.

The technique has prepared the way for the procedure to be done in the office with only tumescent local anesthesia. The only significant modification for this was instilling a pre-resection first stage of tumescence in a ring like fashion approximately 2cm from any vein to be resected . This ring of anesthetic should be instilled until a peau d'orange effect is seen superficially and should go 2-3 cm deep to the skin as well. Resection can be performed within 5 minutes of placing this anesthetic. This ring block has been successful in making powered resection possible in an office setting with only local anesthesia.

The first 22 patients were done in the office with only tumescent local anesthesia between October and December to 2009. The average age of the patients was 44. There were 7 males and 15 females. 20 patients had the powered phlebectomy at the same time as an endovenous procedure on the Greater Saphenous Vein . Patient comfort level was reported on a standard 1-10 scale with 10 being the most comfortable. The comfort level had a range of 7-10 and an average of 8.5 The phlebectomy times including instillation of tumescent local anesthesia averaged 14 minutes. A prospective multicenter trial is in the planning stages at the time of this writing.

In summary, Transilluminated powered phlebectomy with the TriVex® system has afforded many advantages to both the patient and surgeon and promises to revolutionize the removal of varicose veins, compared to manual techniques. It decreases operative and anesthesia times. Excellent cosmetic results, as well as eliminating the pain associated with varicose veins, are achieved. It can be used for primary varicose veins as well as for patients with chronic venous insufficiency, lipodermatosclerosis, venous ulcer, and previous or acute thrombophlebitis. With the use of visualization with transillumination and powered instruments for this otherwise tedious task, more patients can be treated more accurately in a shorter time period. Since, in our experience, the procedure is easy to learn, surgeons will be willing to do the procedure versus previous techniques alone.

REFERENCES

1. Clough AH., Dryden J (trans.) *Plutarch's Lives,* vol. 3. London: Sampson Low; 1859:53.
2. Franz et al, Transilluminated Powered Phlebectomy Surgery for Varicose Veins: A Review of 339 Consecutive Patients, Annals of Vascular Surgery, May 2009, Volume 23, Issue 3, 303–309.

SUGGESTED READINGS

Arumugasamy M, McGreal G, O'Conner A et al. The technique of transilluminated powered phlebectomy: a novel, minimally invasive system for varicose vein surgery. *Eur J Vasc Endovasc Surg.* 2002;23:180–182

Cheshire N, Elias SM, Keagy B et al. Powered phlebectomy (TriVex) in treatment of varicose veins. *Ann Vasc Surg.* 2002;16:488–494

Cohn MS, Seiger E, Goldman S. Ambulatory phlebectomy using the tumescent technique. *Dermat Surg.* 1995;21:315–318.

Crotty TP. The role of radial reflux in the genesis of varicose veins. *Med Hypotheses.* 1996;47(6):449–454.

Eichlisberger R, Holtz D, Stirnemann P, Jager KA. Ambulatory therapy in varicose veins [in German]. *Ther Umsch.* 1996;53(4):295–303.

Hanke CW, Bullock S, Bernstein G. Current status of tumescent liposuction in the United States. *Dermat Surg.* 1996;22:595–598.

Holme K, Matzen M, Bomberg AJ et al. *Partial or Total Stripping of the Great Saphenous Vein. 5-Year Recurrent Frequency and 3-Year Frequency of Neural Complications after Partial and Total Stripping of the Great Saphenous.* 158(4):405–408.

Jones L, Braithwaite BD, Selwyn D et al. Neovascularisation is the principal cause of varicose vein recurrence: results of a randomized trial of stripping the long saphenous vein. *Eur J Vasc, Endovasc Surg.* 1996;12(4):442–445.

Klein, JA. Anesthetic formulation of tumescent soluations. *Dermatol Clin.* 1999;17:751–759.

Leahy, Austin L, Bell, Peter RF, Katzen, Barry T. *Minimal Access Surgery For Vascular Disease.* Martin Dunitz Ltd; 2002:236.

Lees T, Singh S, Beard J, Spencer P, Rigby C. Prospective audit of surgery for varicose veins. *Br J Surg.* 1997;84(l):44–46.

Maly I, Erhart D. *Personal Experiences With Radical Treatment of Varicose Veins in the Lower Extremities, The Present Status and Future Perspectives of Therapy.* 183–186.

Neumann HA, De Roos KP, Veraart JC. Muller's ambulatory phlebectomy and compression. *Dermatol Surg.* 1998;24:471–474.

Ninia JG. Treatment of vulvar varicosities by injection-compression sclerotherapy. *Dermatol Surg.* 1997;23(7):573–574; discussions 574–575.

O'Leary DP, Chester JF, Jones SM. Management of varicose veins according to reason for presentation. *Ann R Coll Surg Engl.* 1996;78(3):214–216.

Olivencia JA. Maneuver to facilitate ambulatory phlebectomy. *Dermatol Surg.* 1996;22(7):654–655.

Pitman GH, Aker JS, Tripp ZD. Tumescent Liposuction. *Clin Plastic Surg.* 1996;23(4):633–645.

Porter. What's new in vascular surgery—venous disease. *J Am Coll Surg.* 1998;186:257–258.

Ramelet AA. Complications of ambulatory phlebectomy. *Dermatol Surg.* 1997;23(10):947–954.

Smith SR, Goldman MP. Tumescent anesthesia in ambulatory phlebectomy. *Dermatol Surg.* 1998;24:453–456.

Ricci Stefano. Ambulatory phlebectomy—principles and evolution. *Dermat Surg.* 1998;24: 459–464.

Spitz GA. Presentation given at the American College of Phlebology, 12th Annual Congress, November 1998, Puerto Rico, USA.

Spitz GA, Braxton, JM, Bergan JJ. Outpatient varicose vein surgery with transilluminated powered phlebectomy. *Vasc Surg.* 2000;34:547–555.

Tong Y, Royle J. Recurrent varicose veins after short saphenous vein surgery: a duplex ultrasound study. *Cardiovasc Surg.* 1996;4(3):364–7.

Weiss RA, Goldman MP. Transillumination mapping prior to ambulatory phlebectomy. *Dermatol Surg.* 1998;24:447–50.

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Endovenous Radiofrequency Obliteration of Saphenous Vein Reflux

Robert F. Merchant, Jr, M.D., F.A.C.S., and Robert L. Kistner, M.D., F.A.C.S.

Great saphenous vein reflux is an important component of the pathophysiology of primary venous insufficiency and is customarily treated with surgical stripping of the saphenous vein from the groin to just below the knee. In 1998, a new modality, the Closure® catheter and procedure were introduced in Europe as a less invasive method of eliminating saphenous vein reflux by using endovenous radiofrequency (RF) current to heat the vein wall. The technique evolved from an initial effort to produce competence in the refluxing great saphenous valve by shrinking the collagen in the wall of the vein at the base of the valve. This began in 1996 in the laboratory of VNUS Medical Technologies, Inc. (company), where shrinkage of the collagen was shown to occur. Although this was not a reliable method of producing a competent valve, the early studies did demonstrate the feasibility of shrinking the vein wall to a small diameter (1 to 2 mm) from which terminal thrombosis of the lumen produced complete occlusion of the vein in the goat model.[1] In the human, early trials were done by shrinking the saphenous vein to the point of closure followed by removal of the vein for histologic study. These studies showed injury to the endothelium and the subendothelial layer without gross necrosis of the vein wall. Human trials were begun in European centers in 1998, followed by FDA approval in the United States in March, 1999, and further trials on both continents.

Early attempts to restore saphenous vein valve incompetence with the new radiofrequency catheter were not successful at achieving satisfactory competence in clinical trials and the noble goal of achieving saphenous vein preservation was abandoned. The earliest cases of vein obliteration were performed with adjunctive saphenous high ligation, or crossectomy, with the catheter either introduced at the groin incision distally or alternatively at or near the knee or ankle via percutaneous or cut-down method. Such a procedure required an obligatory groin incision. But a new advantage was realized when the technique was modified to eliminate the groin incision altogether and accomplish successful elimination of reflux at the saphenous junction and truncal

tributaries solely by the obliteration of the vein lumen from a distal insertion site. Saphenous vein tributaries near the junction almost always remained open and valvular competence, if present preoperatively, was observed postoperatively. Following early reports it soon became clear that reflux at the saphenofemoral junction could be eliminated by obliteration of the great saphenous vein in the thigh without resorting to dissection and ligation of all contributing branches near the saphenofemoral junction.[2,3] If this approach stands the test of time, it could be a tremendous innovation in the management of the incompetent saphenous vein as it would eliminate the need for a groin incision and potential for minor and even major complications that can occur following traditional ligation and stripping procedures.

Since the introduction of radiofrequency ablation of the saphenous vein in 1998, over 20,000 procedures have been performed as of May, 2003. Data on over 1000 limbs treated without high ligation have been collected in an ongoing Registry of the VNUS Closure Treatment Study Group comprised of 35 centers from the U.S., Europe and Australia. Ongoing results from this registry at various follow up periods through January 2002, as reported by Merchant, et al show successful ablation ranging from 93% at one week to 85% at two years, with absence of vein reflux of 90% at two years, and patient satisfaction of 95% at two year follow up.[4]

Early treatments were plagued by complications, such as skin burns and nerve injuries that have virtually disappeared as experience with the procedure has accumulated. Technical improvements have materially changed the risk that thermal injuries might occur to the skin and the nerves.[5]

With the introduction of tumescent anesthesia in 1999, several improvements were observed: 1) the vein was displaced further from the overlying skin, and 2) a fluid bath was produced, acting as a "heat sink" to disperse the heat known to radiate up to 1.5 mm beyond the vein wall at the point of catheter tip contact. Thus, nearby skin and sensory nerves could be protected from the potentially injurious heat extending beyond the treated vein wall. A third improvement has been the achievement of the "dry saphenous vein" because the inflow of branches into the saphenous vein can be virtually eliminated by placing the tumescent solution around the vein through careful duplex scan guidance of the needle used to place the infusate.

TECHNIQUE

Unlike earlier attempts to obliterate the saphenous vein by diathermy, the new Closure procedure uses radiofrequency current to heat the vein wall by endovenous approach. Radiofrequency current is delivered by an intralumenally placed catheter containing bipolar electrodes at its tip which, when in contact with the vein wall, generate heat in the vein wall by a phenomenon called "resistive heating." Thus, the vein acts much like a filament in a light bulb, and as a result, the vein lumen is narrowed as the catheter is slowly withdrawn. The energy, imparted to the vein wall as a product of temperature and time of contact, causes a physical shortening of the collagen fibril of the vein wall. The process is controlled by a computerized generator which monitors temperature and impedance feedback and adjusts energy levels to achieve a constant heating of the vein wall at 85 degrees C ± 3 degrees as the catheter is slowly withdrawn from the vein. As a result, the vein diameter is narrowed while at the same time denatured blood proteins congeal to obliterate the vein lumen. The entire vein is affected by this process much like soft boiling an egg. The

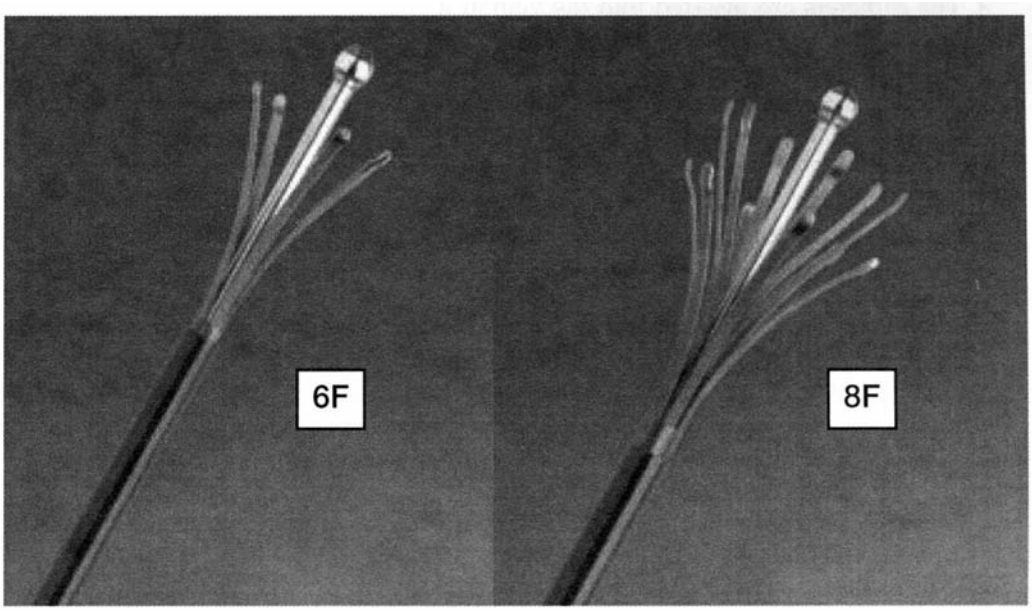

Figure 50-1. Two catheter sizes currently available, 6 French (left) and 8 French (right). Both contain a central lumen to allow through-the-catheter .025″ guide wires to assist ultrasound guidance for proper catheter positioning. (Photo courtesy of VNUS Medical Technologies, Inc., San Jose, CA, reproduced with permission).

dark brown-greenish black material noted on the catheter represents denatured hemoglobin and blood proteins and is not "carbonized tissue." Over the next several months, usually ten to twelve, and certainly by two years, the vein fibroses and is seen to vanish on duplex ultrasound in over 86% of cases.[6]

The steps of the technique as we currently perform them are as follows:

1. Choice of anesthesia is a matter between the physician and the patient. However, the procedure itself is well suited for local anesthesia or regional field block such as a femoral nerve block. General anesthesia offers at least one drawback—that being the inability of the anesthetized patient to communicate nerve pain which might be the result of the heat produced by the catheter as it is withdrawn through the vein and comes into proximity with an overlying sensory cutaneous nerve. Minimal sedation with oral (diazepam) or intravenous (midazolam and fentanyl) agents is recommended to provide adequate anxiolysis and analgesia.
2. The choice of catheter size depends upon the diameter of the vein, measured 1 to 3 cm distal to the saphenofemoral junction, with a 6 French catheter suitable for vein diameters up to 8 mm, and an 8 French catheter for diameters up to 12 mm. (Figure 50–1) The catheters can be used on larger vein diameters, especially if they are focal and sporadic further down the dilated vein. Using tumescent anesthesia with epinephrine, we have been able to successfully close dilated veins in the range of 16 to 18 mm diameter as long as the proximal most 3 cm segment of the great saphenous vein (GSV) is no larger than 14 mm diameter. If the GSV is larger than 14 mm, or if it is aneurysmal at or near the junction, then contraction of the vein in response to heating may not be adequate to obliterate the vein lumen; clot extension into the common femoral vein could

possibly result. Such a situation warrants high ligation and either RF ablation or stripping of the GSV located in the thigh.

3. The catheters are inserted into the vein at its nearest point to the skin surface, usually just below or above the knee. The method of insertion is either percutaneous (Seldinger) with the use of ultrasound guidance, or a cut-down technique, such as a vertical micro-phlebectomy incision. The catheter, once inserted into the vein, is positioned 1 to 2 cm distal to the saphenofemoral junction with the use of ultrasound guidance. The newer catheters, introduced in mid 2001, feature a larger central lumen so that they may be passed over a 0.025 inch diameter "through-the-catheter" guide wire, e.g. Glide Wire®, to allow maneuverability through tortuous or difficult vein segments under ultrasound guidance if necessary. When the guide wire is withdrawn, a small amount of blood usually follows the wire up the central channel. To avoid obstruction of the channel one must irrigate the channel with saline solution, sometimes requiring a bolus via a Tuberculin syringe, before starting the radiofrequency generator. Should the central channel become plugged with dessicated blood, insertion of the guide wire can assist in re-establishing flow of the irrigating solution that helps keep the vein clear of blood.
4. Once the catheter has been positioned near the SFJ, tumescent anesthesia can be introduced using a variety of methods; however, ultrasound visualization of the needle or cannula placement is important to secure adequate fluid volume and position in a perivenous location. It is important to make sure that the fluid is placed beneath the saphenous fascia and above the deep muscular fascia and that it surrounds the vein completely. When located properly, the fluid acts to cool the radiant effect from the heated vein without significantly affecting the internal and transmural vein wall temperatures. Contraction of the vein diameter is another benefit if diluted epinephrine is included in the tumescent anesthetic fluid. Tumescent anesthesia, using generous volumes of buffered lidocaine 1% with epinephrine 1:100,000 diluted to 0.1% placed properly, results in relatively pain free status (Table 50–1). Care must be taken to avoid lidocaine toxicity—dosage guidelines are 7 mg/kg body weight, and no more than 500 mg should be used at one setting. Bilateral limb procedures may require alternate anesthesia such as femoral nerve block using safe dosages of buffered plain lidocaine diluted to 0.2% and instilling lactated Ringer's solution in the perivenous tissues under ultrasound guidance as described above. This has been used at the Reno Vein Clinic with satisfactory results.
5. Once the catheter has been located properly (Figure 50–2), the patient is positioned in gentle Trendelenburg position, approximately 15 to 20 degrees. Before turning on the catheter one must check for position of the catheter at the original location to make sure that it has not migrated into the common femoral vein as a result of patient position change. It is a good idea to arrange the limb in the position in which you will treat the vein prior to instilling the tumescent anesthesia because fluid around the saphenofemoral junction may distort the ultrasound image and make it difficult to be confident of the catheter position in relation to the saphenofemoral junction.
6. The radiofrequency current is applied with the temperature preset at 85 degrees centigrade. The special RF generator (Figure 50–3) provides adequate monitoring and feedback to control the temperature plus or minus 3 degrees centigrade. The generator is preset so that the energy does not exceed 6.0 watts. The generator

TABLE 50-1. TUMESCENT ANESTHESIA SOLUTION PREPARATION

Ringer's Lactate	500 cc
Withdraw 50 cc	–50 cc
	450 cc
Add lidocaine 1% with epinephrine 1 : 100,000	+50 cc
	500 cc
Add sodium bicarbonate (NaHCO3) 8.4%	+16 cc
Resultant solution is lidocaine 0.1% with epinephrine 1 : 1 million	**516 cc**

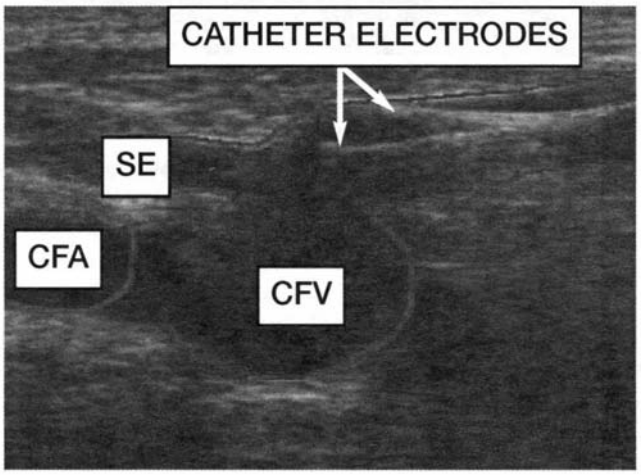

Figure 50-2. Proper catheter position 0.5 to 1.0 cm from the saphenofemoral junction with electrodes deployed (arrows). SE = superficial epigastric vein, CFA = common femoral artery, CFV = common femoral vein. (Photo courtesy of Olivier Pichot, MD, reproduced with permission).

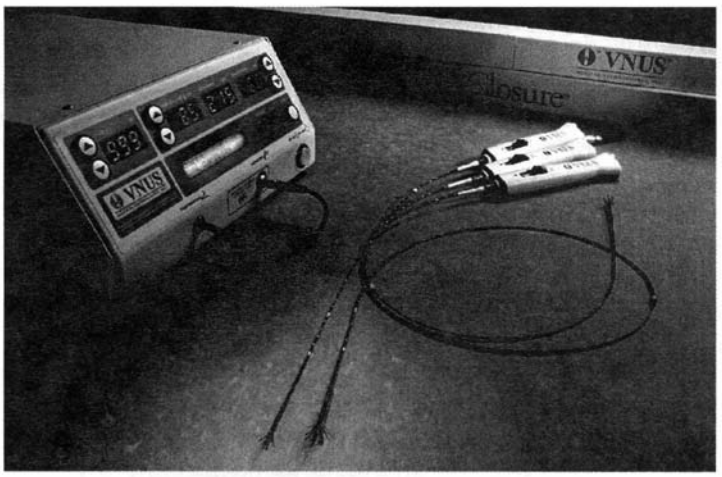

Figure 50-3. Radiofrequency generator with assortment of catheters. (Photo courtesy of VNUS Medical Technologies, Inc., San Jose, CA, reproduced with permission).

also displays impedance measurement at the tip of the catheter. Attention to the impedance reading helps one determine if the vein is adequately compressed or if there is too much blood contained in the lumen (impedance reads too low). Gentle manual pressure is applied to the saphenofemoral junction area, and then along the course of the vein as the catheter is slowly removed at a rate of 2 to 3 cm per minute.

7. Intraoperative duplex ultrasound (DUS) should be used in all cases to document satisfactory closure of the treated vein just before removing the catheter from the vein. If an open vein with significant flow remains, the catheter is readvanced to the open segment, if possible, and the vein should be retreated. If the vein still remains open after two treatments, we recommend ceasing the procedure at that point as the vein may close overnight or in a few days as vein swelling occurs as a result of thermal injury. A curious finding on ultrasound may be echogenic movement depicted in the occluded vein despite having obliterated the lumen. It probably represents movement of saline solution infused through the Closure catheter in the vein and around the blood plug despite adequate obliteration of the lumen. It is for this reason that we would recommend caution if sclerosant is injected as the catheter is being removed from the vein, since some of this fluid may find its way to the saphenofemoral junction and might possibly potentiate thrombotic extension into the common femoral vein.
8. Once the catheter and introducer have been removed from the vein, gentle pressure at the venipuncture site is all that is necessary to establish hemostasis. A Steri-Strip can be applied over the incision. If the cut-down method is used, ligation and division of the saphenous vein is recommended as the size of the lateral venotomy might prevent adequate hemostasis by pressure. The leg is wrapped to the knee or above with an elastic bandage.
9. The patient is encouraged to ambulate immediately post operatively, and in some cases may return to normal activities on the same day. Post op ultrasound imaging of the saphenofemoral junction (SFJ) within three days is an essential part of the protocol to check for closure success and absence of clot extension into the common femoral vein. What is usually seen at this initial check is remarkably similar to an acute thrombosis of the vein with dilation and filling of the vein lumen with echo dense signals and failure to compress with externally applied pressure. (Figure 50–4) This represents an element of thrombosis which aides the obliteration process.

EXPERIENCES AT THE RENO VEIN CLINIC

From August 1999 through March 2003, treatment with RF ablation was attempted in 364 vein segments at the Reno Vein Clinic. Two hundred seventy-six vein segments had initial documented evaluation within one week post procedure and ultrasound and clinical evaluations subsequently at various times, thus providing meaningful data to report. The group was comprised of 84% females and 16% males. Adjunctive ambulatory phlebectomies or perforator ligations were performed in 99% of all limbs treated. Taking into account the initial learning period, the overall immediate success rate of vein obliteration in these cases found on initial ultrasound postoperative day one was $^{271}/_{276}$ (98.2%). Two

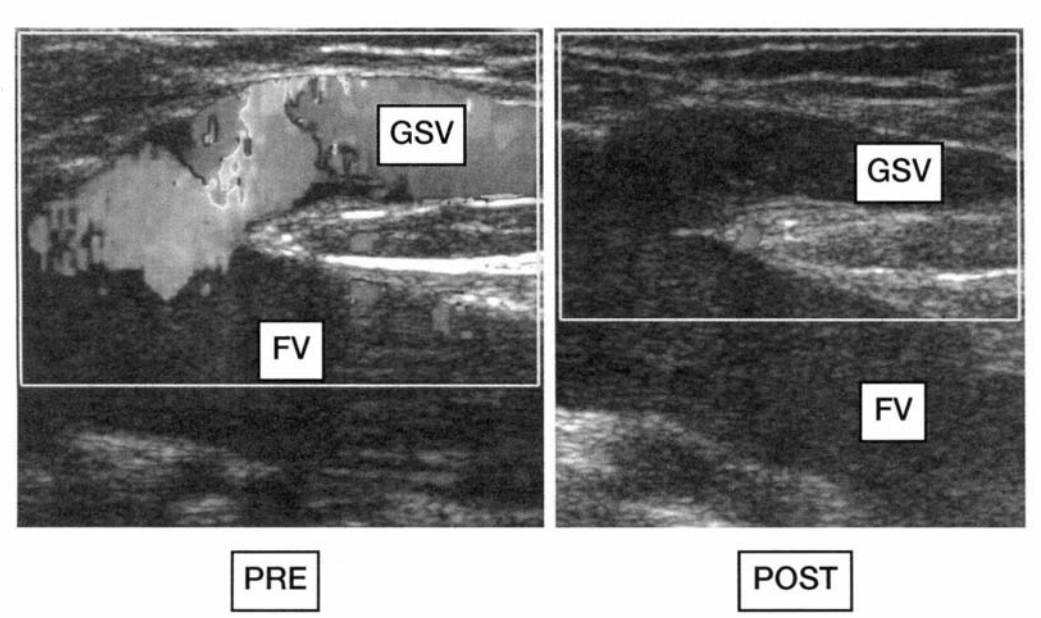

Figure 50-4. Color duplex ultrasound images showing pre (left) and post (right) radiofrequency treatment. Note the absence of color flow in the great saphenous vein (GSV) in the post treatment image on the right. FV = femoral vein. (Photo courtesy of Olivier Pichot, MD, reproduced with permission).

early cases were abandoned, one due to posterior tibial nerve pain attempting short saphenous closure, and the other due to an inability to introduce an early 5Fr catheter (not compatible with 0.025" steerable guide wires) into a morbidly obese limb. One additional case required a groin incision to expose the proximal great saphenous vein to check proper catheter position which had been ultrasonigraphically obscured by tumescent anesthesia fluid. This was an early case and a high ligation was performed given the opportunity of the incision. These three cases yield an abandonment rate due to technical reasons of $^{3}/_{364}$ (0.8%). Five early failures with recanalization and recurrent symptoms were successfully retreated with RF ablation and they all remain closed without reflux at one year follow up.

Complications reported in the 276 treated segments included skin burn $^{1}/_{276}$ (0.4%) as a direct result of Eschmark wrapping of the thigh (since abandoned), paresthesias (hypoesthesias) in $^{24}/_{276}$ (8.7%), phlebitis in $^{6}/_{276}$ (2.2%), DVT/clot extension in $^{1}/_{276}$ (0.4%) limbs, and zero percent infection.

From June 2001 through March 2003, when tumescent anesthesia became routine at the Reno Vein Clinic and the newer catheters were consistently available, initial obliteration of the treated vein segment on duplex ultrasound at day one post operative was successful in $^{158}/_{158}$ (100%) and no further cases were abandoned. Additionally, in this second group, the occurrence of hypoesthesia was reduced to $^{3}/_{158}$ (1.9%).

To many, an important concern is the fate of the tributaries at or near the saphenofemoral junction which are usually intentionally left to allow venous return and lymphatic drainage from the abdominal wall and lower extremity. To help answer this question, Pichot coordinated an extensive two year follow up ultrasound evaluation study from five VNUS Registry centers and reported the results at the American Venous Forum annual meeting in Cancun, February 2003.[6] The results showed that $^{58}/_{63}$ (92.1%) treated GSV segments studied at two years remained free of reflux.

One vein (1.6%) had extensive recanalization with SFJ reflux. Junctional tributary reflux was seen in $^{7}/_{63}$ (11%) limbs, four of which were associated with SFJ reflux. Neovascularization was not observed in any treated limbs.

At the Reno Vein Clinic, three cases developed reflux through the SFJ into the anterior-lateral saphenous vein within nine months postoperatively—all had no evidence of reflux by DUS into these branches preoperatively or immediately postoperatively on day one, and, because of technical problems, significant thigh varicose veins had been left untreated at the time of the Closure procedure. Two cases were seen at three months and the third at nine months. In all of the cases, when seen at the first available follow up beyond one week, reflux was noted directly through the SFJ down the anterior saphenous vein and into the pre-existing varicose veins. These three specific personal observations suggest that SFJ incompetence may be secondary to downstream venous insufficiency rather than a primary contributor to superficial venous insufficiency.

To avoid nerve injury, following the early clinical experience the Closure procedure was recommended to be limited to above knee GSV treatments.[5] The greater saphenous nerve is actually adherent to the GSV in the distal leg and injury to this nerve is usually unavoidable when RF ablation is attempted much below the knee.[3] Since January 2002, RF ablation at the Reno Vein Clinic has been extended to include several cases of anterior and posterior branches of the GSV in the proximal thigh. Also since then, 12 small (lesser) saphenous veins were successfully treated without nerve injury, and one saphenopopliteal junction reflux was found on one year follow up DUS (n = 4). In the case of small saphenous vein treatment, careful ultrasound guidance is necessary to place the catheter in a plane which is parallel to the gastrocnemius muscle just before it begins to curve into the popliteal space; this usually locates the extended electrode tips of the catheter approximately 2 to 3 cm distal to the actual saphenopopliteal junction. Location of the catheter at this spot avoids inadvertent heating of the posterior tibial nerve; pain located in the heel or foot at onset of heating indicates placement too close to the nerve. Should this happen, shut off the heat, withdraw the catheter 1 cm, and begin again. Tumescent anesthesia, placed below the superficial fascia and around the small saphenous vein, is a must to avoid injury to the sural nerve which usually lies near the vein.

EXPERIENCE AT THE STRAUB CLINIC

The experience with Closure at Straub Clinic in the first 24 months of its use (from April 2000 to April 2002) comprises 300 total operated limbs performed over a period of 24 months with follow up for one year or longer in 160 cases.[7] Tortuous veins and aneurysmal veins were not excluded, nor were veins with focal areas of internal diameter greater than 12 mm excluded. Adjunctive procedures of phlebectomy or perforator interruption were performed in 95% of these cases. A liberal policy of conversion of the procedure to open ligation and stripping was followed to ensure that a proper operation was performed in each limb, and the total of conversions was 3%. As the learning curve of performing the procedure progressed, significantly fewer conversions to stripping procedures were needed. The other 97% of cases achieved successful initial closure as determined on the operating table by duplex scan criteria. The complications in this experience were limited to one skin burn in the second case, recurrent reflux in 3 cases on post operative duplex

study, deep venous thrombosis (DVT) in $^{2}/_{300}$ (0.7%), miscellaneous minor problems in 1%, and no mortality. The DVTs both occurred in the common femoral vein, were found at the first 24 hour post operative scan, and were managed by thrombectomy with no subsequent sequelae and no clinical pulmonary emboli. There have been no subsequent thromboses after 24 hours in this group of cases which were all followed with serial post operative scans. The greatest problems in the learning curve were those of access (15%), including entering the saphenous vein in the calf, and passing the catheter successfully to the saphenofemoral junction. These problems have been overcome with experience in puncturing the vein and with the availability of the through-catheter guide wire.

MANAGEMENT OF COMPLICATIONS

Data collected for the VNUS Closure Study Group Registry was prospective, looking for nerve injury, clot extension, hematoma, phlebitis, skin burns, and infection.[4] Results are compared with stripping and ligation as shown in Table 50–2.[8–13] The most serious complication, although rare, is clot extension (Figure 50–5) into the common femoral vein as it can lead to DVT if not recognized and treated early with either low molecular weight heparin (LMWH) or operative thrombectomy. Duplex ultrasound is a crucial component of the Closure protocol and should be performed within 72 hours of the procedure. It is the practice at both the Reno Vein Clinic and the Straub Clinic to see all cases on the first post operative day and to include a post-operative duplex scan during that visit. If there is evidence of obliteration of the GSV "flush" with the common femoral vein (no spontaneous superficial epigastric vein flow) or slightly extending into the deep vein, then LMWH is prescribed at therapeutic doses for six days and aspirin 325 mg is started empirically on day 7 and continued for one month. This protocol was used in five cases at the Reno Vein Clinic and DVT did not occur subsequently. One case of clot extension obscuring approximately 40 to 50% of the transverse diameter of common femoral vein was seen at post op day one and was treated successfully with operative thrombectomy, LMWH for one week, and then aspirin for one month. At the Straub Clinic there have been four total cases of thrombi in the common femoral vein in a total of 450 cases treated to date, and each of these have been treated by thrombectomy with ligation of the saphenous vein. No post-thrombotic sequelae

TABLE 50-2. LITERATURE COMPARISON OF REPORTED COMPLICATION RATES FROM RF OBLITERATION AND STRIPPING WITH HIGH LIGATION

Complication	RF obliteration[4]	Stripping with high ligation
Paresthesia		
Treatment limited to thigh (AK)	2.8% at 1 year*	8% at 2 years[8]
Treatment includes lower leg (BK)	7.5% at 1 year*	17% at 1 year[9]
Infection	0%	4.5%[10] – 13.7%[11]
Deep vein thrombosis	0.7%	0.15%[12] – 1.8%[13]
With pulmonary embolism	0.3%	0.06%[12]
Iatrogenic	Skin burn	Vein injuries 1%[12]
	2.1% first 143 cases	Arterial injuries 0.02%[12]
	0% second 143 cases	

*Described as a localized area of hypoesthesia

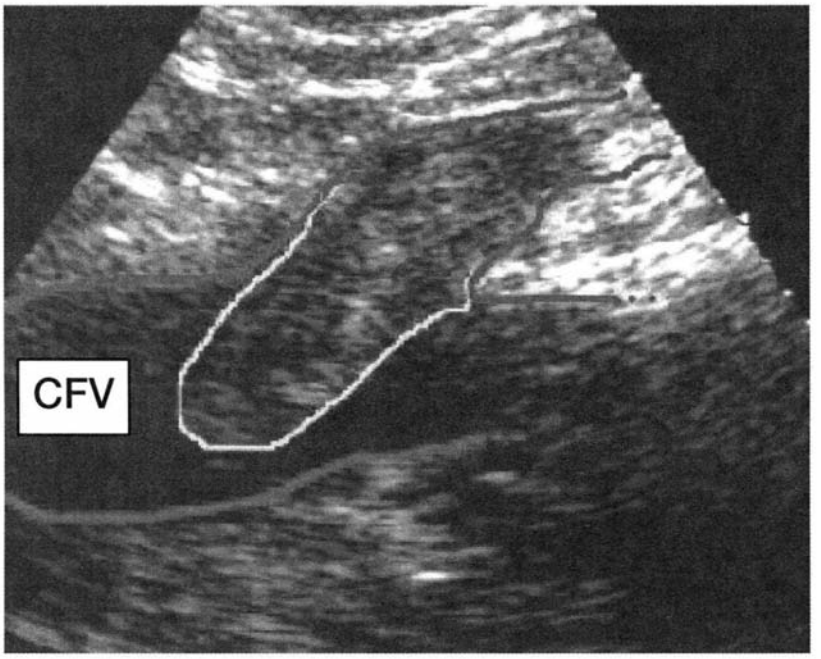

Figure 50-5. Ultrasound image of a clot (outlined in white) originating in the treated great saphenous vein extending into the common femoral vein (CFV) lumen, partially occluding the deep vein. (Photo courtesy of Olivier Pichot, M.D., reproduced with permission).

have occurred in these cases. All patients are cautioned about this potential prior to surgery.

The contraindications to using Closure in the Straub Clinic series are those veins that are post-thrombotic, those with aneurysms at or near the saphenofemoral junction, and those with long axial segments that are 12 mm or larger in diameter.

Nerve injury associated with RF ablation is seen as areas of hypoesthesia noted on follow up examination in the first week post op. The majority of these occurred in the early Closure cases before the routine instillation of tumescent anesthesia. The tumescent fluid surrounding the saphenous vein acts as a reservoir to dissipate the radiant heat from the vein.

Skin burns, initially seen in early Closure cases, essentially have vanished since the institution of tumescent anesthesia[4,14] and the abandonment of the Eschmark leg wrap. The Eschmark rubber bandage has a tendency to roll back when applied to the funnel shaped thigh, and in that case it acts as a tight rubber band to push the skin closer to the saphenous vein. In the thin leg this could be too close despite tumescent anesthesia and a burn could occur.

Phlebitis can occur with Closure as in any treatment of varicose veins and it is usually the result of residual blood trapped within vein segments. Some degree of phlebitis is inherent in the whole process since the obliteration occurs as a result of injury to the vein by the heating process. It is occasionally seen as a tender, erythematous or ecchymotic band over the treated vein in the distal thigh. It resolves over several weeks without any specific treatment other than symptomatic relief, e.g. nonsteroidal anti-inflammatory drugs, heat, and compression hosiery.

Many patients describe a curious sensation which occurs during the second or third post op week along the treated vein segment, usually in the distal thigh. They may experience a spontaneous or persistent dull feeling, or "bogginess," or sharpness when stretching or extending the treated leg. This could represent an inflammatory process which occurs as the body is healing the scald injury of the treated vein segment. The sensations usually abate over several weeks, consistent with the normal healing time of injured tissues.

COMPARISONS WITH TRADITIONAL STRIPPING

The decision to implement a new technique requires that it must be at least as safe and effective as traditional methods. The persistence of reflux in the treated vein segment can be assessed by objective means using DUS, while its absence proves the success of the obliteration and is usually indicative of a successful clinical outcome. The presence or absence of varicose veins following treatment is more subjective and cannot be relied upon to accurately assess the outcome of treatment, especially in the obese patient.

Comparison of the Registry data with a literature review of stripping and ligation is shown in Table 50–3. Jones et al,[8] using continuous wave Doppler (CWD) at 1 year follow up and DUS at two year follow up after stripping and ligation of the greater saphenous vein, reported the incidence of absence of reflux as 91% and 87% respectively. Rutgers and Kitslaar,[9] using only CWD, showed a similar finding of 91% absence of reflux at 1 year, 88% at 2 years, and 85% at 3 years. Merchant et al,[4] reporting the first 286 limbs in the VNUS Closure Study Registry Group without high ligation, noted absence of reflux by DUS in 91.4% at 1 year and 90.1% at 2 years. Despite recanalization in $^{25}/_{232}$ (10.8%) of limbs in this group at one year, reflux was absent in 7 of the 25 (28.0%) limbs. In addition, 111 of the 142 limbs with 2 year DUS examinations also were scanned at 1 year; of these, only two (1.8%) changed from reflux free at 1 year to DUS evidence of reflux at 2 years. Subsequent updated data on this registry group shows absence of reflux in 88% at 3 years. Reflux in the VNUS Registry group was defined as evidence of reversed flow in any treated vein segment including the area of the saphenofemoral junction, i.e. through the saphenofemoral junction and into nearby tributaries. In this report of the first 286 limbs treated, at 2 year follow up, reflux was found only in recanalized great saphenous veins. In contrast, the Rutgers data and the Jones 1 year data were derived from CWD studies, and thus, the anatomical site of reflux would not be identified with certainty.

TABLE 50-3. LITERATURE COMPARISON OF ABSENCE OF REFLUX RATES FOLLOWING RF OBLITERATION[4] AND STRIPPING WITH HIGH LIGATION BY DUS UNLESS INDICATED

	One year	Two years	Three years
RF obliteration	91%	90%	88%*
Stripping with high ligation			
Jones[8]	91%†	87%	N/A
Rutgers[9]	91%†	88%†	85%†

*Updated for additional results obtained since publication
†Assessments performed using continuous wave Doppler

Two randomized prospective studies on the early results of Closure treatment without high ligation have been published, and both show significant clinical superiority of the Closure procedure. In the first study from one center in Oulu, Finland,[15] significant advantages of the Closure treatment were shown regarding less pain, early return to activities, fewer sick leaves from work, and better quality of life scores. When these findings included time lost from work, the authors found Closure treatment to be cost effective despite initial high hospital costs. The second study was from five centers in the United States and Europe, and it was designed to determine the early benefits of the procedure with follow up limited only to four months.[16] Early three week results showed significant advantage of the Closure procedure in that there was less pain, earlier return to activities and work, better quality of life scores, and better cosmetic results. When these patients were seen at the four month follow up, these advantages had disappeared. Although the study was not designed to evaluate cost effectiveness, when the severity of infectious complications (which occurred only in the stripping and ligation group) were factored in, the authors opined a probable cost benefit to Closure.

CONCLUSIONS

The evidence published in peer reviewed journals, two studies of which are level one, suggests that at least out to two years, outcomes of RF obliteration of saphenous vein reflux are comparable to traditional stripping and ligation. Early experiences with the Closure procedure were marked by the occurrence of complications that were overcome with catheter redesign to accommodate a through-the-catheter guide wire, the introduction of tumescent anesthesia to replace general anesthesia, and better technique modifications. The current technique has become easier and safer to perform. The risks of serious complications such as DVT are low and probably comparable with those that attend stripping and ligation. Lesser complications, when they do occur, are time limited and usually of minor consequence. In high risk patients, e.g. those with co-morbidities, anticoagulation, or obesity, Closure may be the best treatment because of the advantages it offers over traditional surgical methods, especially regarding less trauma. In those cases where reflux originates distal to the saphenofemoral junction (which can only be appreciated by DUS), Closure is ideally suited. Neovascularization at the saphenofemoral junction does not appear to occur and may not be a factor in later recurrent varicose veins following this procedure, a possible distinct advantage in comparison with surgical stripping and high ligation. The fate of the persistent patency found in the superficial epigastric vein and other less frequently seen groin branches remains to be determined by long-term follow-up over 3 to 5 years, or even longer.

The experience of the Reno Clinic and the Straub Clinic confirms minimal to absent pain, early return to activity within 12 to 48 hours in most cases, and early return to work depending more upon the individual's desire to return to work rather than upon any medical necessity to defer the return. The choice of Closure is extremely popular with patients both pre and post operatively. Many individuals harbor a fear of the stripping procedure which may not be truly warranted but is nevertheless real, while most approach the Closure technique as acceptable for the magnitude of the problem they experience. Even when informed that long term knowledge of the ultimate comparative effects of Closure vs. stripping is still not known, most reply that this is not a deciding factor for them.

Radiofrequency obliteration of saphenous vein reflux, in our opinion, given the caveat that it be done by a qualified physician, has become a safe and effective alternative to traditional surgical techniques during the short-term of 2 to 3 years, in our opinion. Longer-term follow up of well designed randomized studies will determine whether these encouraging early outcomes prove to be durable. In the meantime it makes sense to offer this innovative technology as an alternative choice for the patient with greater saphenous vein reflux of primary origin.

REFERENCES

1. Weiss RA, Goldman MP. Controlled radiofrequency-mediated endovenous shrinkage and occlusion. In: Goldman MP, Weiss RA, Bergan JJ eds. *Varicose Veins and Telangiectasias; Diagnosis and Treatment*. 2nd ed. St. Louis: Quality Medical Publishing; 1999:217–24.
2. Chandler JG, Pichot O, Sessa C et al. Defining the role of extended saphenofemoral junction ligation: A prospective comparative study. *J Vasc Surg*. 2000;32:941–953.
3. Chandler JG, Pichot O, Sessa C et al. Treatment of primary venous insufficiency by endovenous saphenous vein obliteration. *Vasc Surg*. 2000;34:201–214.
4. Merchant RF, DePalma RG, Kabnick LS. Endovascular obliteration of saphenous reflux: A multicenter study. *J Vasc Surg*. 2002;35:1190–1196.
5. Manfrini S, Gasbarro V, Danielsson G et al. Endovenous management of saphenous vein reflux. *J Vasc Surg*. 2000;32:330–342.
6. Pichot O, Kabnick LS, Merchant RF et al. Duplex ultrasound mid-term hemodynamic and morphologic findings after radiofrequency endovenous obliteration of the greater saphenous vein. Paper presented at: Annual meeting of the American Venous Forum; February 22,2003; Cancun, MX.
7. Kistner RL. Endovascular obliteration of the greater saphenous vein: The Closure procedure. *Jpn J Phlebol*. 2002;13:325–333.
8. Jones L, Braithwaite BD, Selwyn D et al. Neovascularisation is the principal cause of varicose vein recurrence: Results of a randomised trial of stripping the long saphenous vein. *Eur J Vasc Endovasc Surg*. 1996;12:442–445.
9. Rutgers PH, Kitslaar PJ. Randomized trial of stripping versus high ligation combined with sclerotherapy in the treatment of the incompetent greater saphenous vein. *Am J Surg*. 1994; 168:311–315.
10. Mackay DC, Summerton DJ, Walker AJ. The early morbidity of varicose vein surgery. *J R Nav Med Serv*. 1995;81:42–46.
11. Corder AP, Schache DJ, Farquharson SM et al. Wound infection following high saphenous ligation. A trial comparing two skin closure techniques: subcuticular polyglycolic acid and interrupted monofilament nylon mattress sutures. *J R Coll Surg Edinb*. 1991;36:100–102.
12. Hagmuller GW. Complications in surgery of varicose veins. *Langenbecks Arch Chir Suppl Kongressbd*. 1992;470–474.
13. Miller GV, Lewis WG, Sainsbury JRC, Macdonald RC. Morbidity of varicose vein surgery: auditing the benefit of changing clinical practice. *Ann R Coll Surg Engl*. 1996;78:345–9.
14. Weiss RA, Weiss MA. Controlled radiofrequency endovenous occlusion using a unique radiofrequency catheter under duplex guidance to eliminate saphenous varicose vein reflux: A 2-year follow-up. *Dermatol Surg*. 2002;28:38–42.
15. Rautio T, Ohinmaa A, Perälä J et al. Endovenous obliteration versus conventional stripping operation in the treatment of primary varicose veins: A randomized controlled trial with comparison of costs. *J Vasc Surg*. 2002;35:958–965.
16. Lurie F, Creton D, Eklof B et al. Prospective randomized study of endovenous radiofrequency obliteration (Closure) versus ligation and stripping in selected patient population (EVOLVeS Study). *J Vasc Surg*. 2003;38:207–214.

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Laser Treatment for Varicose Veins

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For decades ligation and stripping has been the gold standard for treating greater saphenous vein (GSV) varicosities arising from saphenofemoral reflux. However with the current trend toward minimally invasive procedures, the public is inundated with the "less is more" phenomenon via the media, mailings, and even billboards. Today's healthcare consumer is seeking care from specialists who can provide a spectrum of treatment that include these new less invasive options. While endovascular methods to treat arterial occlusive and aneurismal disease have continued to evolve at a rapid rate since the late 1970s, endovenous approaches to obliterate the greater saphenous vein have been much slower to develop.

Surgical treatment while successful has its drawbacks. These include paresthesia, bleeding, infection, anesthesia, scarring,[1] activity restrictions, postoperative pain and bruising. In addition, postoperative recurrence of GSV reflux has been well documented in the literature.[2-4] Dwerryhouse et al in a five year follow-up of patients with surgical intervention found recurrent saphenofemoral reflux in 29% of patients following ligation and stripping and up to 71% at 5 years with high ligation alone.[3] Drawbacks of surgery and recurrent GSV reflux have prompted the emergence of less invasive treatment methods including sclerotherapy and more recently, endovenous obliteration. Bishop et al studied patients who underwent sclerotherapy of the refluxing thigh portion of the greater saphenous vein and found persistent reflux and recurrence rates of up to 75% at a mean of 27 months post injection.[5]

Three major developments have led to current endovenous techniques. First is the availability of radiofrequency and laser probes to deliver endovenous heat. Second is the development of tumescent anesthesia. Tumescence allows large volumes (200 to 500 cc) of dilute lidocaine (0.1%) to be used in a single session. Epinephrine can be added to the solution to provide hemostasis and allow the treated vein to contract around the heatgenerating catheter. The anesthesia is injected so that it is trapped between the superficial and deep fascia of the thigh. This allows the entire thigh portion of the GSV to be anesthetized and obliterated at one time. Lastly, duplex ultrasound revolutionized our ability to evaluate the etiology of varicosities and tailor treatment

to ablate only the diseased segments of saphenous vein preserving normal segments. It allows ultrasound guided placement of sheathes and heat-generating catheter tips at very precise locations in the saphenous vein.

VNUS Closure (VNUS Medical Technologies, Inc, Sunnydale, Calif.) was developed in the late 1990s and received FDA approval in 1999. The first reports in the literature appeared in 2000.[1,6-9] Vein occlusion occurs by heating the vein wall to 85 degrees with radiofrequency energy delivered by electrodes at a catheter tip in a direct feedback loop. Direct contact of the probes with the endothelium causes collagen contraction and endothelial damage resulting in eventual vein wall fibrosis.[10] Initially, VNUS Closure was used as an adjunct combined with high ligation of the greater saphenous vein. High ligation did not result in a higher incidence of venous obliteration and was abandoned.[7] Complete or near complete saphenous occlusion rates of 88.7% at two years have been reported.[11] The initial limitations of the procedure appeared to be thermal complications related to the device. The slow pullback times result in additional heat dispersal to surrounding tissues resulting in more collateral damage.[12] Thermal skin burns and saphenous neuralgia were frequent problems with the device initially but the addition of tumescent anesthesia has minimized the thermal burns and reduced the incidence of postoperative neuralgia. Despite using tumescent anesthesia, 9% of patients with thigh greater saphenous veins treated with Closure still experienced saphenous paresthesia at 6 weeks. Three of five patients at the one-year follow-up had persistent saphenous or sural paresthesias.[1] In a series with 24 month follow-up, 4.5% ($^{5}/_{111}$) of patients had persistent paresthesias when greater saphenous vein treatment was limited to the thigh and just below the knee.[11]

Limitations to the Closure procedure seem to be slow catheter pullback times, inability to treat saphenous veins that are greater than 12 mm in size and collateral heat damage and resulting paresthesias. Despite these limitations, patient satisfaction was achieved in 94.5% of patients at 24 months.[11] Endovenous ablation resulted in less postoperative pain, shorter sick leaves and faster return to previous lifestyle when compared to traditional vein stripping.[13]

The limitations of VNUS Closure have led to the search for an alternative to deliver endovenous heat. Bone's first description of a diode laser for endovenous ablation appeared in 1999.[14] Laser technology allows rapid conversion of light energy to heat and has been developed into an alternative technique for endovenous obliteration of the greater saphenous vein.

LASER TECHNOLOGY

Diode lasers are solid state lasers which operate at wavelengths between 810 nm and 980 nm. Laser light is transmitted to the venous endothelium through a 600 micron bare tipped laser fiber. Two mechanisms of action appear to explain vein wall destruction. Direct impact of laser light appears to create a localized vein injury. Super heating of venous blood creates a more diffuse vein wall injury.[15] Work by Proebstle et al has helped delineate the mechanism by which laser energy creates endovenous obliteration.[15] The hemoglobin component of blood serves as a chromophore absorbing laser energy. Laser energy creates a steam bubble which transfers heat energy homogeneously to the vessel wall. In their in-vitro model, neither saline nor plasma was able to produce steam bubbles. However blood produced steam bubbles in volumes proportional to the joules of laser

energy delivered. All three laser wavelengths produced equal volume of steam bubbles. Initially, pulsed laser light was used but continuous mode is now applied to create a more uniform vessel injury. Vein wall destruction with subsequent thrombosis and fibrosis most likely creates the permanent obliteration of the saphenous vein.

PATIENT EVALUATION

Preoperative history, physical exam and duplex ultrasound allow one to select patients with varicose veins due to primary saphenofemoral reflux and truncal insufficiency. At the initial consultation a comprehensive patient history is taken including onset of varicosities, associated symptoms, prior vein treatment (stockings, injections etc.) or surgery, previous deep vein thrombosis, pulmonary embolism, phlebitis, thrombophlebitis, ulceration, varicosities in relation to pregnancy and family history. A physical exam is completed with the patient in a standing position. Inspection and palpation of the lower extremities is performed to determine vein locations, edema and changes of venous insufficiency. A venous duplex examination is performed using a 7 MHz linear array probe. Evaluation includes determining levels of reflux using the Valsalva maneuver and distal augmentation and mapping of the superficial venous system. The plan of care is then determined and discussed with the patient. A prescription for Class II compression stockings is given to the patient as well as educational materials including preoperative and post-procedure instructions.

TECHNIQUE

The day of the procedure, informed consent for endovenous obliteration of the affected GSV is obtained from the patient. If ambulatory phlebectomy is to be performed immediately following the GSV obliteration, branch varicosities are marked with the patient standing, prior to the procedure. The entire procedure including ambulatory phlebectomy is performed using local tumescent anesthesia. Oral or IV sedation is optional although we have not found it necessary. With the patient in the supine position, the entire leg is prepped and draped in a sterile fashion. After placing a sterile cover over the ultrasound probe, the saphenous vein is examined from the saphenofemoral junction (SFJ) to the inferior most point of dilatation and incompetence, usually between the proximal third of the calf to the distal third of the thigh. Lidocaine 1% is given locally at the access site. Under ultrasound guidance, the most distal point of GSV incompetence is cannulated with a 21G-echogenic needle. A stainless steel-tipped .015 wire is passed and proper placement is verified with ultrasound. A small dilator is passed over the wire. The wire is exchanged for a .035 wire that is passed to the SFJ using ultrasound guidance. The dilator is removed and a 5 French 45 cm sheath is passed over the wire to the SFJ. The wire and obturator are removed. At this point, time is taken to correctly position the sheath just distal to the SFJ (see Figure 51–1). The laser fiber is passed to the tip of the sheath and the sheath is then pin pulled backwards and the sheath is the locked to the laser fiber. The sheath-fiber apparatus is withdrawn so the tip of the laser fiber is 1 to 2 cm from the SFJ. If the superior epigastric vein branch is seen, the laser tip should be positioned just inferior to it taking care that the femoral vein is an ample distance from the GSV so as not to damage the femoral vein wall.

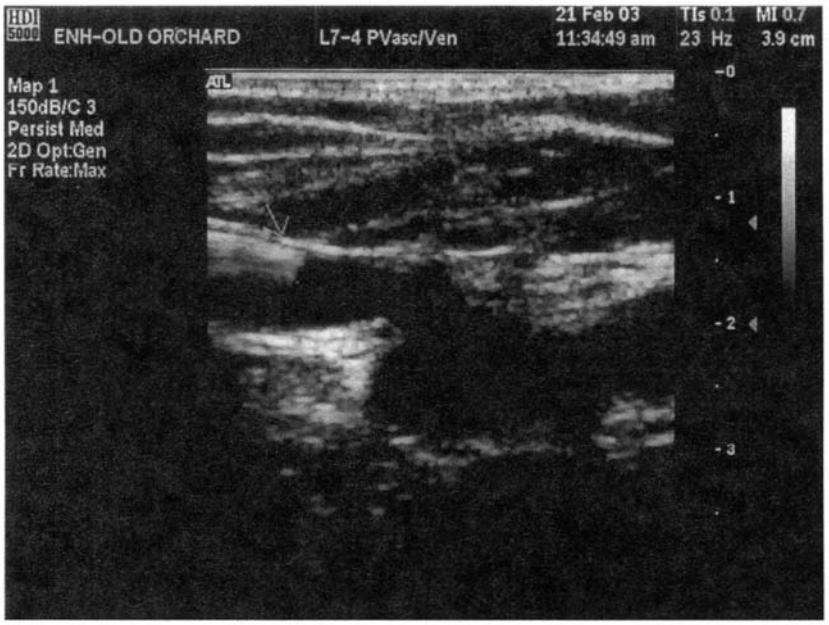

Figure 51-1. Sheath (see arrow) positioned distal to SFJ.

Tumescent anesthesia is now given starting at the access site moving proximal to the SFJ. A 22 gauge $1^1/_2$ needle is positioned next to the saphenous vein between the superficial and deep fascia and 30 cc of tumescence is given confirming location with ultrasound. Ultrasound should confirm echo lucent tumescent anesthesia around the entire saphenous vein to be treated and that the saphenous vein is at least 1 cm below the surface of the skin. This eliminates the risk of thermal skin complications. The entire saphenous vein from the cannulation site to the SFJ is anesthetized usually requiring about 120 to 180 cc of tumescence. Appropriate position of the laser tip is again confirmed with ultrasound. Some centers place the operating table in the Trendelenburg position to facilitate vein contraction but we have not found this necessary. Appropriate laser settings are verified. We currently use a 980 nm diode laser set to 10 watts of power in the continuous mode. Laser-appropriate protective eyewear is provided for each person present. With the laser activated, the sheath-fiber apparatus is slowly withdrawn at the rate of 1 cm every 2 to 3 seconds. When the tip of the sheath appears at the skin entry site, 3 cm of the laser fiber remains in the saphenous vein. The laser is deactivated at this time to avoid ambient laser light in the room and the apparatus is removed. The total laser treatment time is between 90 and 180 seconds to treat the thigh portion of the greater saphenous vein. Phlebectomy of branch varicosities can be performed at this time or at a subsequent date. When the procedure is completed a Steri-Strip is applied to the skin and covered with a clear adhesive dressing. A class II thigh-high compression stocking is applied. Discharge instructions are reviewed with the patient and prescription anti-inflammatory medication is prescribed for one week. The patient is allowed to resume normal activity the following day. One week after the procedure, the patient is seen in follow-up where the dressing(s) are removed and an ultrasound exam is performed to document obliteration of the vein.

DISCUSSION

Endovenous laser obliteration of the greater saphenous vein while analogous to VNUS Closure appears to offer significant advantages. Laser technology provides short durations of very intense heat which is rapidly dissipated minimizing heating of surrounding tissues. In addition, rapid pullback times are more surgeon friendly allowing average total procedure times of 35 minutes in our experience. Some would suggest that the laser is associated with more postoperative pain and bruising than seen with VNUS Closure. Our early experience would indicate that this is not the case. The procedure is well tolerated by patients and post procedure satisfaction levels are high.

The literature on laser vein obliteration is limited. The short term data indicates that the vein occlusion rate is similar to VNUS Closure without the thermal burns and parasthesias seen with Closure. Min et al obliterated 90 greater saphenous veins with 810 nm diode laser with a 96% success rate in the 26 patients followed at 9 months.[16] Three patients had early recannalization requiring a second procedure but only one patient had transient parasthesias. There were no incidents of parasthesias beyond 6 weeks and no thermal burns. Longer term data published in abstract form by Navarro and Boné discusses three year follow-up data on 200 procedures which revealed a 2.6% GSV recannalization rate, all of which occurred in the first six months post-procedure.[17] In all cases no SFJ reflux was observed, nor was there evidence of neoangiogenesis or progressive incompetence of the remaining SFJ branches. Longer term data on the success of the laser is lacking, but there is no reason to think it will be different than the excellent results seen with VNUS Closure. More long term follow-up in peer-reviewed journals may be available by publication of this chapter.

The laser is currently only approved by the FDA for treatment of the thigh portion of the greater saphenous vein. This is because of the high incidence of sural and saphenous neuralgia seen when the lessor saphenous vein and calf portion of the greater saphenous vein was treated with Closure. These results were most likely secondary to the lack of tumescence anesthesia and the excessive heating of surrounding tissue with the slow pullback times with Closure. Proebstle recently reported a series of 41 lesser saphenous veins treated with a 940 nm diode laser.[18] During a mean follow-up of 6 months, no vein recannalization occurred. Only 4 legs developed transient paresthesia, none of which were permanent. Eventually the laser may be used to treat all segments of the superficial venous system.

While endovenous techniques will be applicable to the majority of patients with greater saphenous vein varicosities, some patients will still require saphenofemoral surgical ligation. Patients with dilatation of the greater saphenous vein to over 2 cm at the level of the junction may not be adequately obliterated without risk of developing thrombus extending into the femoral vein. In addition, patients with large incompetent branches (e.g. anterior lateral and posterior medial) arising within 1 to 2 cm of the SFJ may have persistent reflux mandating either selective laser obliteration of these branches or surgical ligation.

Classic surgical teaching mandates the ligation of all five saphenofemoral vein branches. This cannot be accomplished with endovenous techniques. The laser when appropriately used will maintain patency of at least the superior epigastric vein if not others. This may be advantageous as Jones has suggested that elimination of all venous drainage by complete saphenofemoral ligation may stimulate neorevascularisation and recurrent varicose veins.[2]

Interpretation of the future endovenous literature may be confusing if similar definitions of success are not used. Merchant et al considered veins occluded if the occlusive process was flush with the femoral vein or demonstrated patency to the level of the superior epigastric vein.[11] Near complete occlusion was defined as a patent segment of greater saphenous vein at the junction of less than 5 cm without reflux. The vein was considered recannalized when greater than 5 cm of greater saphenous vein was still patent with reflux. Use of this classification may be useful to allow consistency of publication across specialities as dermatologists, radiologists and non-surgical phebologists are adopting this technology to broaden their patient base.

CONCLUSION

Endovenous laser obliteration of the greater saphenous vein is a new approach to the treatment of lower extremity varicose veins which is less morbid than current vein stripping and offers advantages over other endovenous techniques. While long term data is not available, limited short term data indicates that this technology offers significant advantages to patients. Surgeons must explore and use these tools if we are to maintain our leadership position in the treatment of lower extremity venous disease.

REFERENCES

1. Manfrini S, Gasbarro V, Danielsson G et al. Endovenous management of saphenous reflux. *J Vasc Surg*. 2000;32:330–342.
2. Jones L, Braithwaite BD, Selwyn D et al. Neovascularisation is the principle cause of varicose vein recurrence: results of randomized trial of stripping the long saphenous vein. *Euro J Vasc Endovasc Surg*. 1996;12:442–445.
3. Dwerryhouse S, Davies B, Harradine K, Earnshaw JJ. Stripping the long saphenous vein reduces the rate of reoperation for recurrent varicose veins: five-year results of a randomized trial. *J Vasc Surg*. 1999;29:589–592.
4. Sarin S, Scurr JH, Coleridge Smith PD. Assessment of stripping the long saphenous vein in the treatment of primary varicose veins. *Brit J Surg*. 1992;79:889–893.
5. Bishop CCR, Fronek HS, Fronek A et al. Real-time color duplex scanning after sclerotherapy of the greater saphenous vein. *J Vasc Surg*. 1991;14:505–510.
6. Chandler JG, Pichot O, Sessa C et al. Treatment of primary venous insufficiency by endovenous saphenous vein obliteration. *Vasc Surg*. 2000;34:201–214.
7. Chandler JG, Pichot O, Sessa C et al. Defining the role of extended saphenofemoral junction ligation: a prospective comparative study. *J Vasc Surg*. 2000;32:941–953.
8. Goldman MP. Closure of the greater saphenous vein with endoluminal radiofrequency thermal heating of the vein wall in combination with ambulatory phlebectomy: preliminary 6-month follow-up. *Dermatol Surg*. 2000;26:452–456.
9. Pichot O, Sessa C, Chandler JG et al. Role of duplex imaging in endovenous obliteration for primary venous insufficiency. *J Endovasc Ther*. 2000;7:451–459.
10. Weiss RA, Weiss MA. Controlled radiofrequency endovenous occlusion using a unique radiofrequency catheter under duplex guidance to eliminate saphenous varicose vein reflux: a 2-year follow-up. *Dermatol Surg*. 2002;28:38–42.
11. Merchant RF, DePalma RG, Kabnick LS. Endovascular obliteration of saphenous reflux: a multicenter study. *J Vasc Surg*. 2002;35:1190–1196.
12. Haines DE. The biophysics of radiofrequency catheter ablation in the heart: the importance of temperature monitoring. *Pacing Clin Electrophysiol*. 1993;16(3 pt 2):586–591.

13. Rautio T, Ohinmaa A et al. Endovenous obliteration versus conventional stripping operation in the treatment of primary varicose veins: a randomized controlled trial with comparison of costs. *J Vasc Surg*. 2002;35:958–965.
14. Boné Salat C. Endoluminal treatment of varicose veins with diode laser preliminary study. *Patologia Vasc*. 1999;5.
15. Proebstle TM, Sandhofer M, Kargl A et al. Thermal damage of the inner vein wall during endovenous laser treatment: key role of energy absorption by intravascular blood. *Dermatol Surg*. 2002;28:596–600.
16. Min RJ, Zimmet SE, Isaacs MN et al. Endovenous laser treatment of the incompetent greater saphenous vein. *J Vasc Interv Radiol*. 2001;12:1167–1171.
17. Navarro L, Boné Salat C. Endolaser-a three year follow-up report: implications on crossectomy and stripping. In: *Abstracts from the 16th Annual Congress of the American College of Phlebology*. Fort Lauderdale, Florida. November 7-10, 2002.
18. Proebstle TM, Doendue G, Kargl A et al. Endovenous laser treatment of the lesser saphenous vein with a 940-nm diode laser: early results. *Dermatol Surg*. 2003;29:357–361.

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Transvenous Insertion of Inferior Vena Cava Filters

Sara Clark, B.S. and Heron E. Rodriguez, M.D.

INTRODUCTION

Interruption of the inferior vena cava (IVC) with a mechanical barrier to prevent pulmonary embolism (PE) can be traced back to the 1700s. Subsequently, caval ligation by the use of sutures or clips was performed under general anesthesia. In 1967, implantable filters were introduced. After being almost completely abandoned due to its many complications, the procedure gained significant clinical application with the introduction of the Greenfield stainless steel filter in 1973. Continued advances in filter designs have led to a dramatic increase in their use—from 2000 in 1979 to 49 000 in 1999 in the United States. This has skyrocketed to more than 140 000 filters inserted worldwide in 2003.[1]

Although inferior vena cava filters (IVCFs) have gained widespread acceptance, there is lack of level 1 evidence supporting their use. Only one controlled randomized trial has evaluated IVCF.[2] At 8 years follow-up, patients randomized to receive IVCF plus anticoagulation had a lower risk of PE but a higher incidence of deep venous thrombosis (DVT) than the control group (anticoagulation alone) (35.7% at 8 years with IVC filter placement versus. 27.5% without).[2] Furthermore, this study failed to demonstrate a survival advantage from filter placement. Despite the paucity of outcomes evidence to support their use, there is agreement that IVCFs are indicated for the prevention of PE in patients with contraindication to anticoagulation, recurrent PE despite adequate anticoagulation, or prior complications of anticoagulation. Contraindications to anticoagulation possibly requiring IVCF placement include: current or recently active major bleeding, intracranial bleeding, need for a major surgical procedure, or severe, prolonged thrombocytopenia. Additional relative indications in selected patients include large, free-floating iliocaval thrombus, thromboembolic disease with limited cardiopulmonary reserve, recurrent PE with pulmonary hypertension, or propagating iliofemoral venous thrombus despite anticoagulation. IVCFs can also be utilized during thrombectomy or thrombolysis of existing DVT.[3]

The greatest controversy exists regarding the "prophylactic" use of IVC filters in patients without a diagnosis of venous thromboembolic disease. These patients are

deemed high risk for PE and receive a prophylactic filter arguably because of their inability to receive therapeutic or prophylactic anticoagulation. These patients include high-risk trauma patients with multiple long bone or pelvic fractures, closed head injury, or spinal injury; patients with malignancy, burns, or sepsis; and patients undergoing major spinal reconstruction or bariatric surgery.[4] Despite the paucity of evidence supporting their use, prophylactic IVCFs are an increasingly common practice. Furthermore, the development of retrievable filters has allowed filters to be placed in patients with a temporary elevation in risk for thromboembolism or a temporary contraindication to anticoagulation. Removal of the filter then avoids the long-term complications associated with IVCFs, such as increased risk for DVT. The American College of Chest Physicians has published guidelines for the use of IVCF.[5] These most recent guidelines do not recommend the use of prophylactic IVCFs for high-risk trauma patients or patients undergoing spinal reconstruction; however, conclusive evidence is still lacking.

TYPES OF FILTERS

There are about a dozen IVC filters that have gained approval by the FDA (Table 52–1). In general, they are all inserted percutaneously via the jugular or femoral approaches. Although they are made of different metal alloys, most resemble an umbrella in appearance. There are two kinds of filters: permanent and retrievable. Retrievable filters can be removed when their use is no longer required. Some filters in common use are shown in Figure 52–1.

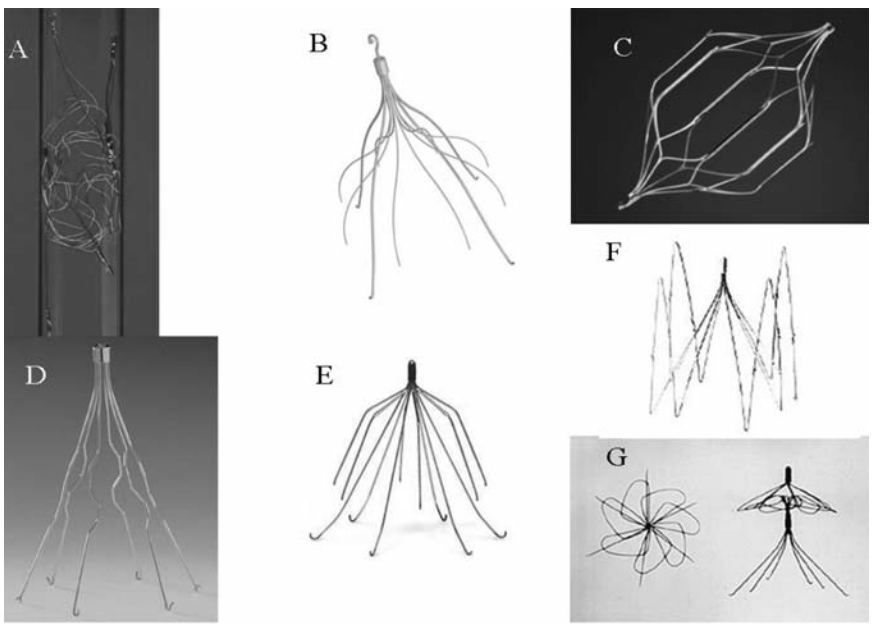

Figure 52-1. Commonly used filters. (A) Bird's Nest (Cook), (B) The Gunther Tulip Retrievable filter (Cook), (C) The OptEase (Cordis), (D) Greenfield Filter (Boston Scientific), (E) The G2 Express Retrievable filter (Bard), (F) Vena Tech LP (Braun) and (G) The Simon Nitinol (Bard).

TABLE 52-1. CHARACTERISTICS OF CURRENT FDA APPROVED RETRIEVAL/OPTIONAL IVC FILTERS

Filter Type/ Company	Filter Material	Filter Design	Introducer Sheath Diameter	Insertion Site	Maximum IVC Diameter	Retrievable	Recommended Time for Retrieval	Retrieval Route	Diameter of Retrieval Sheath	Technique for Removal
Venatech LP (Braun)	Phynox	Conical	9 Fr.	Jugular, femoral, subclavian	35 mm	NO	n/a	n/a	n/a	n/a
Greenfield (Boston)	Stainless steel	Conical	12 Fr.	Jugular, femoral	28 mm	NO	n/a	n/a	n/a	n/a
Greenfield (Boston)	Titanium	Conical	12 Fr.	Jugular, femoral	28 mm	NO	n/a	n/a	n/a	n/a
GR Birds Nest (Cook)	Stainless steel	V-shaped bird's nest		Jugular, femoral	40 mm	NO	n/a	n/a	n/a	n/a
TrapEase (Cordis)	Nitinol	Double basket	8 Fr.	Jugular, femoral, antecubital	30 mm	NO	n/a	n/a	n/a	n/a
Simon Nitinol (Bard)	Nitinol	Bi-level, conical	7 Fr.	Jugular, femoral, antecubital, subclavian	28 mm	NO	n/a	n/a	n/a	n/a
Option (Rex Medical)	Nitinol	Domed	6 Fr.	Jugular, femoral	30 mm	FDA trials, in 510 (k) approval	n/a	n/a	n/a	n/a
G2 Express (Bard)	Nitinol	Conical	10 Fr. jug. and subclavian, 7 Fr. femoral	Jugular, femoral, subclavian	28 mm	YES	140	Jugular	10 Fr.	Cone-Pull
Gunther Tulip (Cook)	Conichrome	Conical	8.5 Fr.	Jugular, femoral	30 mm	YES	30–60 days	Jugular	11 Fr.	Snare-Hook
OptEase (Cordis)	Nitinol	Double basket	8 Fr.	Jugular, femoral, antecubital	30 mm	YES	23 days	Femoral	10 Fr.	Snare-Hook
Celect (Cook)	Conichrome	Conical	7 Fr. jug./8.5 Fr. fem.	Jugular, femoral	30 mm	YES	50 wks	Jugular	11 Fr.	Snare-Hook

FDA: Food and Drug Administration; IVC: Inferior Vena Cava; Venatech LP Filter (B. Braun Medical Incorporated, Bethlehem, PA); Greenfield Filter (Boston Scientific Corporation, Natick, MA); Gianturco-Roehm Bird's Nest Filter (Cook Medical Inc, Bloomington, IN); TrapEase Filter (Cordis Endovascular, Warren, NJ); Simon Nitinol Filter (C.R. Bard Incorporated, Tempe, AZ); Option Filter (Rex Medical Limited Partnership, Conshohocken, PA); G2 Express Filter (C.R. Bard Incorporated, Tempe, AZ); Gunther Tulip Filter (Cook Medical Inc, Bloomington, IN); OptEase Filter (Cordis Endovascular, Warren, NJ); Celect Filter (Cook Medical Inc, Bloomington, IN)

PRE-OPERATIVE CARE

Prior to placement, the indications, risks, and benefits of IVCF are analyzed and discussed with the patient. This process is straightforward in patients diagnosed with a PE or a DVT who also have an absolute contraindication to anticoagulation. On the other hand, for the so-called "prophylactic indications" of IVCF, it is extremely important to discuss with the patient the rationale for IVCF insertion and the potential complications and long-term effects (see Complications). When using retrievable filters, the possibility of being unable to retrieve the filter (0%-43%)[6] should also be discussed.

Contraindications to percutaneous placement should also be recognized; these include severe, uncorrectable coagulopathy (as with liver or multisystem failure) or untreated bacteremia or infection. Placement of an IVCF is also contraindicated with extensive IVC thrombosis, such that the filter cannot be placed above the thrombus.

TRANSVENOUS INSERTION UNDER FLUOROSCOPY

The procedure is usually done under sterile conditions using local anesthesia in a standard interventional unit or in an operating room equipped with a portable c-arm capable of high-definition fluoroscopy and digital subtraction angiography. Alternatively, for unstable patients, IVCF insertion can be performed at the bedside using high-definition ultrasound (US) or intravascular US.[7]

In general, the filter is positioned within the infrarenal IVC at the level of the L3 vertebral body. If there is thrombus extending to or involving the renal vein or if there is pelvic or ovarian vein thrombosis, the filter should be positioned above the level of the renal vein at T12-L1. The suprarenal location is also appropriate for pregnant women, to prevent compression of the filter from gravity of the gravid uterus. Placement above the renal veins is a safe and effective procedure.

Most IVC filters can be inserted through the femoral or the internal jugular vein. The introducer sheath size ranges from 6 Fr to 16 Fr in outer diameter (Table 52–1). Although "blind" punctures are widely used, some prefer to perform US-guided puncture to access the venous system.

Under sterile conditions, local anesthesia is infiltrated in the skin and an echogenic needle is guided under high-definition US into the femoral or internal jugular vein. A guide wire is advanced into the IVC at the level of the lumbar vertebrae. A small incision is made in order to allow passage of the dilator and sheath over the guide wire.

The dilator and sheath are passed under fluoroscopy over the guide wire into the IVC. Once the dilator and sheath are positioned in the vena cava, the dilator and guide wire are withdrawn, leaving the sheath in place.

A venogram of the IVC is performed (Figure 52–2A) to determine the diameter of the cava and the location of the renal veins (arrows) as well as to rule out the presence of venous abnormalities or caval thrombosis. This cavogram can be done through an angiographic catheter and using a rapid injector or simply by manually injecting contrast with a 20 cc syringe after placing the introducer sheath. Most IVCF can be inserted if the cava measures less than 28-30 mm. After dilating the subcutaneous tract, the IVCF is inserted through the introducer sheath and deployed just below the renal veins under fluoroscopic guidance (Figure 52–2B). The renal veins are usually located at the level of the bodies of the L1 and L2 vertebrae. The mechanism of deployment

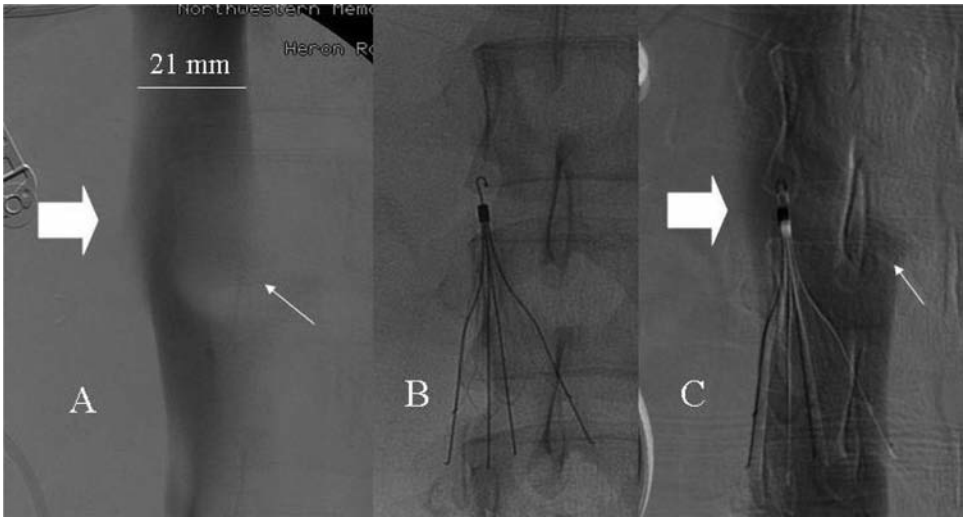

Figure 52-2. Cavogram showing (A) the diameter of the cava and the position of the renal veins (right renal vein large arrow, left renal vein small arrow), (B) filter deployed and (C) completion venogram.

varies according to the different available filters: pin-pull, pin-push, trigger, etc. As an example, deployment of the Greenfield titanium filter is described below.

The filter carrier is introduced into the sheath. Prior to insertion of the carrier system, the sheath should be flushed with heparinized saline to prevent air embolism and thrombus formation within the carrier system.

In order to prevent accidental release of the filter into the sheath, it is important to advance the carrier through the sheath until the sheath hub contacts the control handle. Using fluoroscopic guidance, the carrier should be positioned at the level of L3. The locking system on the control handle is released by moving the control tab to the unlock position. The control tab is then slid backward, which uncovers and discharges the filter. The carrier and sheath are withdrawn and pressure is applied to the puncture site to establish hemostasis. A pressure dressing may be applied.

If the patient's renal function is normal, a completion venogram is done to document the final deployment (Figure 52–2C). The position of the filter relative to the renal veins, and any tilting or asymmetric leg deployment are recorded. In some rare cases, a second IVCF is needed when the asymmetry of the legs after deployment is such that thrombus could pass through the legs. The sheath is removed and gentle pressure is applied to the puncture site.

Occasionally, IVCF can be deployed above the renal veins when thrombus is present in the peri-renal cava. (Figure 52–3)

BEDSIDE PLACEMENT UNDER INTRAVASCULAR ULTRASOUND GUIDANCE

After infiltrating the skin with local anesthesia and gaining access to the venous system via a femoral puncture, a wire is advanced into the central venous circulation. A "pre-measurement" is made, marking the entire length of the IVCF delivery system in the

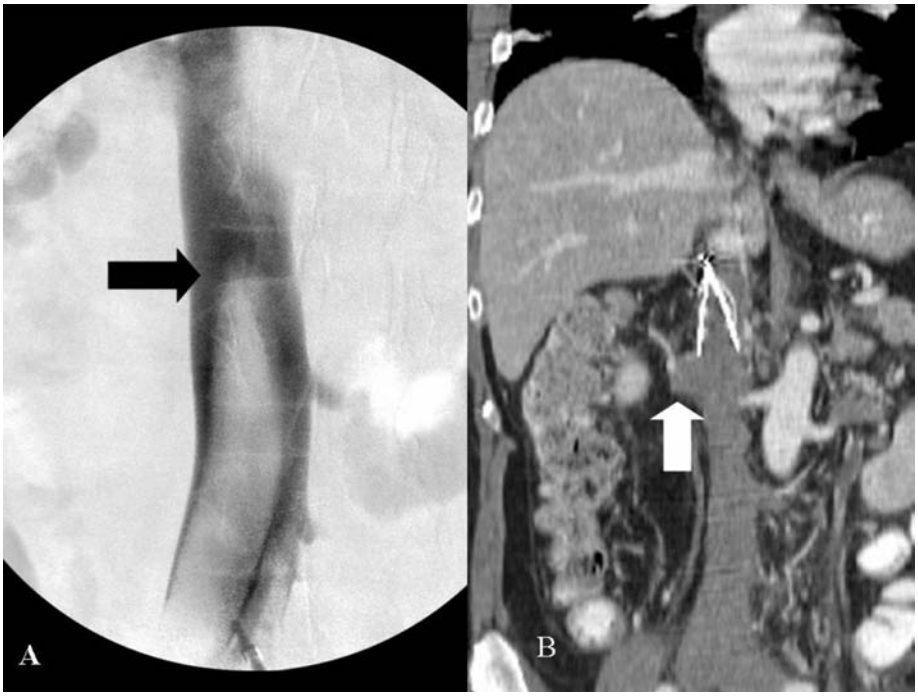

Figure 52-3. The black arrow shows a large clot visualized as a filling defect above the renal veins. The white arrow indicates a filter placed above the renal veins.

intravascular ultrasound (IVUS) catheter. A sterile-strip is placed on the IVUS catheter at the precise length of the entire delivery system away from the tip of the IVUS catheter. The IVUS catheter is inserted into the femoral vein and, using the pullback technique, the venous anatomy is examined (Figure 52–4). The landing zone between the renal veins and the iliac vein confluence is marked on the immobilized drape with sterile-strips, a long one for the renal veins and a short one for the iliac confluence (Figure 52–5). A second pullback confirms the measurements and the IVUS catheter is removed. After dilating the tract, the IVCF introducer sheath is inserted and the IVCF is deployed in the marked landing zone. Plain abdominal x-rays are obtained to verify the position of the filter relative to the lumbar spine and the symmetric appearance of its struts.

RETRIEVAL OF TEMPORARY IVCF

Most patients receiving retrievable IVCFs have a temporary contraindication to anticoagulation. Prior to removal, the current status of the patient's contraindication to anticoagulation, the ongoing risk for PE, and the potential benefit of keeping the filter in the cava are evaluated and discussed with the patient. A duplex US is performed prior to removal to verify patency of the access vessel and to document the presence or absence of DVT.

Most retrievable IVCFs are removed via the internal jugular approach but some can be removed through the femoral vein. US-guided puncture is performed and access to the central venous circulation is gained. A cavogram is performed. If significant

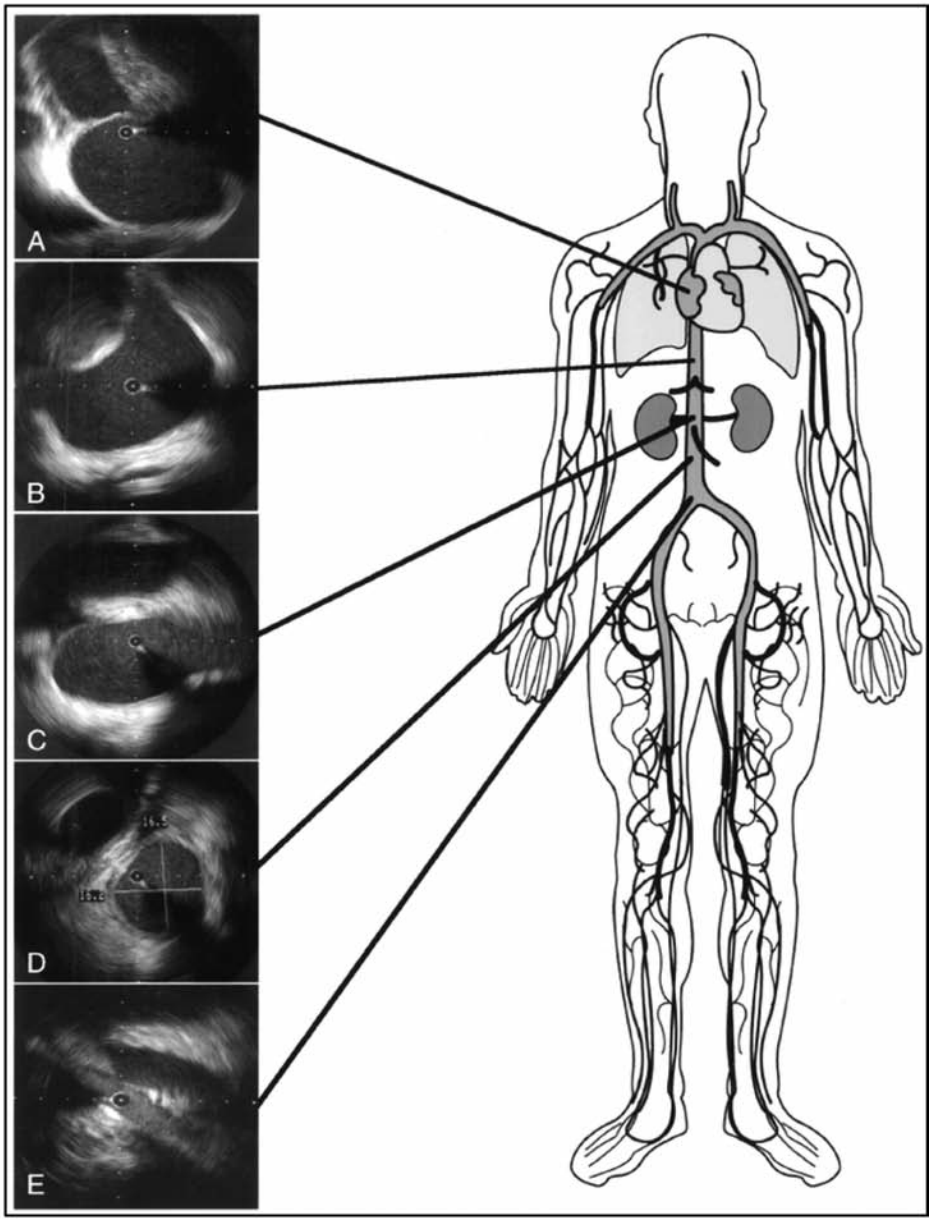

Figure 52-4. Intravascular US demonstrating the anatomic tributaries of the cava: (A) the right atrium, (B) the hepatic veins, (C) the left and right renal veins, (D) the infrarenal cava (the diameter is measured), and (E) the confluence of the common iliac veins. (From Oppat WF, Chiou AC, Matsumura JS. Intravascular ultrasound-guided vena cava filter placement. *J Endovasc Surg.* 1999;6:285–287. Allen Press Publishing Services. Reproduced by permission.)

burden of clot is seen within the IVCF, the procedure is aborted (Figure 52–6). A long introducer sheath is introduced with its tip near the IVCF. The diameter of the sheath depends on the specific filter being removed (see Table 52–1). Most filters are removed by snaring a hook in the most cephalad portion of the filter. With the snare in place

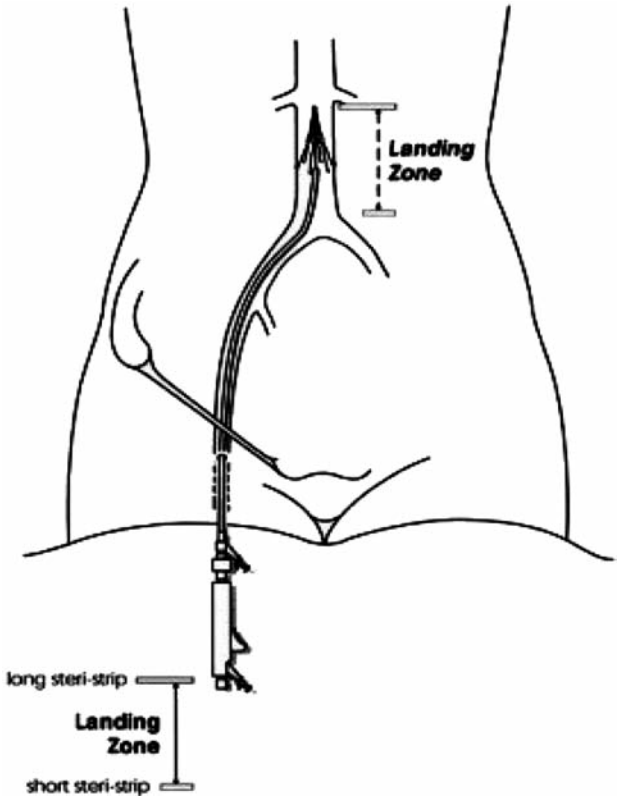

Figure 52-5. The length of the delivery system is marked on the IVUS catheter. A long steri strip is placed at this mark when the lowest renal vein is visualized. A short steri strip is placed when the iliac confluence is seen. By positioning the end of the delivery system between these marks, deployment in the landing zone is achieved. (From Ebaugh JL, Chiou AC, Morasch MD, et al. Bedside vena cava filter placement guided with intravascular ultrasound. *J Vasc Surg*. 2001;34:21–26. Reproduced by permission.)

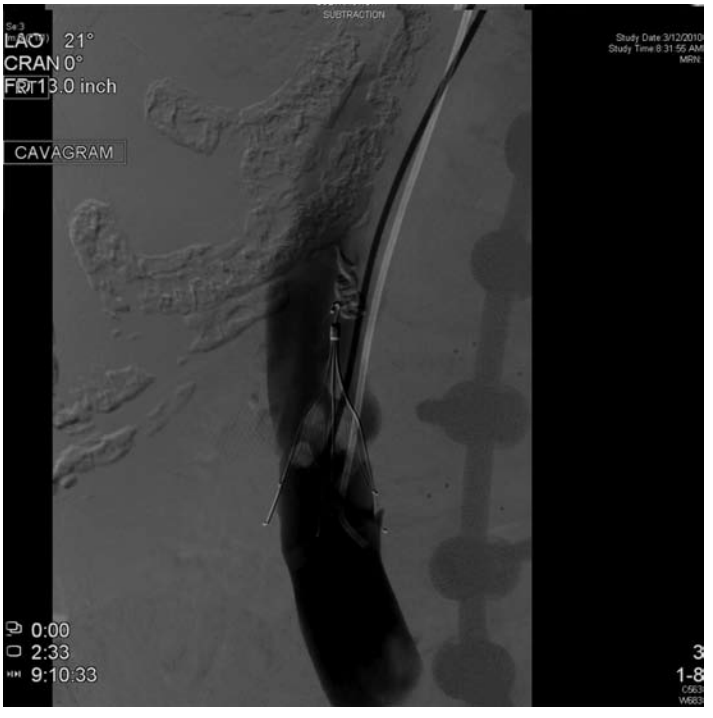

Figure 52-6. Cavogram showing thrombus captured by an inferior vena cava filter.

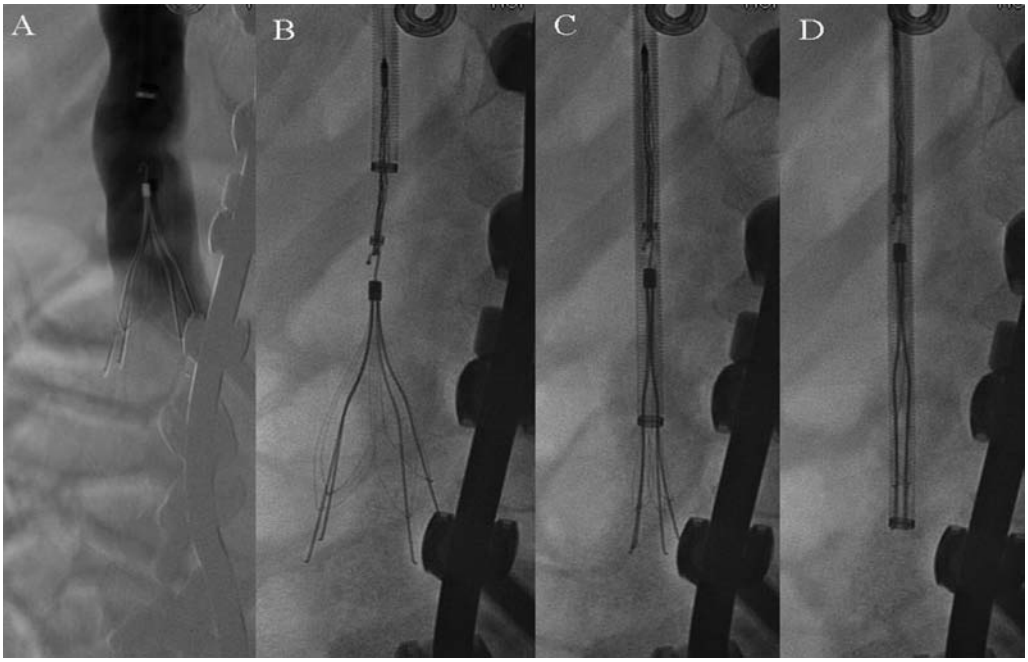

Figure 52-7. Removal of an IVC filter via the jugular approach. A cavogram is performed (A). A snare is advanced through the introducer sheath and the retrieval hook is engaged (B). While maintaining tension on the secured snare, the introducer sheath is advanced over the snare-filter unit (C). No attempts to pull back on the snare are made until the entire filter is collapsed and covered by the sheath (D). A completion venogram is done to document the absence of intraluminal defects and the lack of contrast extravasation (not shown).

and under tension, the sheath is advanced over the filter, causing collapse of the limbs of the filter (Figure 52–7).

Inability to retrieve temporary filters occurs in 0% to 43% of cases.[6] Most commonly, fibrin formation around the retrieval mechanism or tilting are the cause (Figure 52–8). Several maneuvers to overcome tilting and adherence of the retrieval hook to the caval wall have been described.[6] They include pushing the filter from a separate femoral approach, snaring and wiring the struts of the filter, or using bronchoscopy forceps to re-align the filter.

COMPLICATIONS

Serious complications from IVCFs are rare, with a total combined fatal complication rate for all filters reported as 0.12%.[8] Recurrent pulmonary embolism despite filter placement occurs in 2% to 4% of patients, with a fatal PE rate of 0.7%.[8] The most common serious complication of IVCF placement is DVT (with rates up to 45.7%). This significant increase in rate of DVT was noted in the Prepic study. Other complications include bleeding, hematoma formation at access site, ateriovenous fistula, or superficial wound infection. Access site thrombosis occurs in 11%-41% of insertions.[9] The risk of access site thrombosis has not been shown to decrease with lower profile delivery systems. If the jugular vein is used, additional complications include air embolism and pneumothorax. Other less

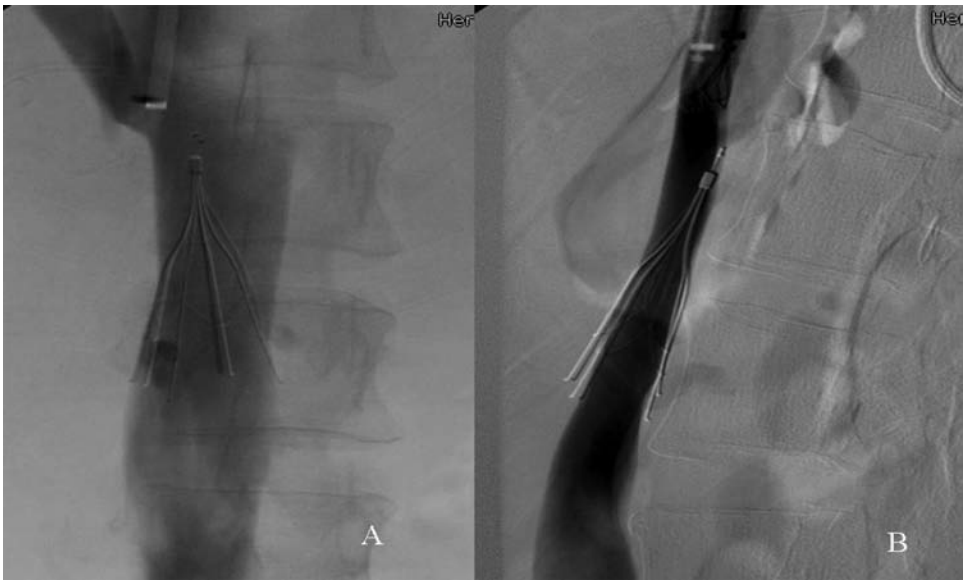

Figure 52-8. This figure shows a filter that was not retrieved due to inability to snare the retrieval hook. Although, in an AP projection, there is no tilting (A), a lateral cavogram reveals posterior tilting with embedding of the hook into the posterior wall of the cava (B). Valsalva maneuvers did not correct the radiographic abnormality.

common complications include misplacement, proximal migration, IVC thrombosis/occlusion, and perforation. Misplacement often results from premature discharge or inadequate fluoroscopic control of the carrier. Reports of proximal migration range from 3% to 69%.[8] Pulmonary artery or intracardiac migration is rare, but potentially fatal.[10] IVC thrombosis or occlusion can occur in 4% to 30% but the etiology is unclear.[8] IVCF thrombosis could be a result of an efficient filter that trapped emboli or in situ formation of thrombus on the filter itself. Limb penetration of the cava can be seen radiographically in 9% to 24%.[8] (Figure 52–9). Perforation is clinically significant when it enters the vascular or GI systems.

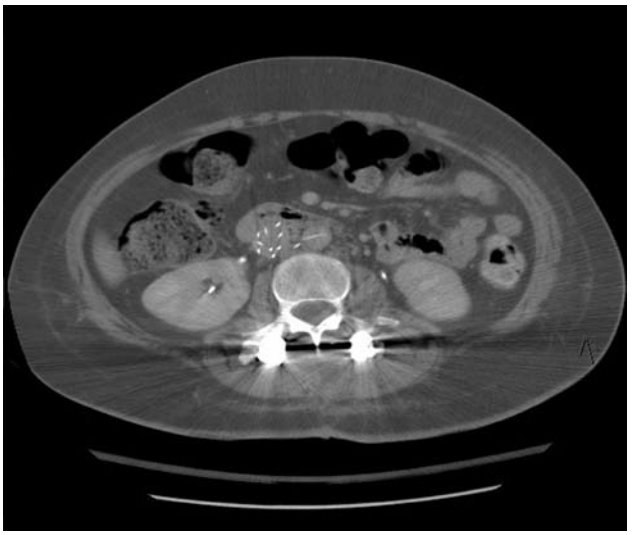

Figure 52-9. CT scan of the abdomen showing migration of the limbs of an inferior vena cava filter. A strut is seen crossing the aorta.

Complications associated with retrieval of a temporary filter include failure to retrieve the filter, thrombus embolization from the filter, and vein retrieval site thrombosis or hematoma. Failure to retrieve the filter is most common, with local rates of removal dependent upon clinical situation, local experience, and protocols.

REFERENCES

1. Sing RF, Rogers FB, Novitsky YW, Heniford BT. Optional vena cava filters for patients with high thromboembolic risk: questions to be answered. *Surg Innov*. 2005;12:195–202.
2. Eight-year follow-up of patients with permanent vena cava filters in the prevention of pulmonary embolism: the PREPIC (Prevention du Risque d'Embolie Pulmonaire par Interruption Cave) randomized study. *Circulation*. 2005;112:416–22.
3. Rutherford RB. Prophylactic indications for vena cava filters: critical appraisal. *Semin Vasc Surg*. 2005;18:158–65.
4. Rectenwald JE. Vena cava filters: uses and abuses. *Semin Vasc Surg*. 2005;18:166–75.
5. Geerts WH, Bergqvist D, Pineo GF, et al. Prevention of venous thromboembolism: American College of Chest Physicians Evidence-Based Clinical Practice Guidelines (8th Edition). *Chest*. 2008;133:381S–453S.
6. Brown K. Retrievable Inferior Vena Cava Filters - Techniques for Removal. In: Matsumura JS, Morasch MD, Pearce WH, Yao JST, ed. *Techniques and Outcomes in Endovascular Surgery*. Chicago, IL: Greenwood Academics; 2009:223–8.
7. Ebaugh JL, Chiou AC, Morasch MD, Matsumura JS, Pearce WH. Bedside vena cava filter placement guided with intravascular ultrasound. *J Vasc Surg*. 2001;34:21–6.
8. Young T, Tang H, Aukes J, Hughes R. Vena caval filters for the prevention of pulmonary embolism. *Cochrane Database Syst Rev*. 2007:CD006212.
9. Leon L, Rodriguez H, Tawk RG, Ondra SL, Labropoulos N, Morasch MD. The prophylactic use of inferior vena cava filters in patients undergoing high-risk spinal surgery. *Ann Vasc Surg*. 2005;19:442–7.
10. Owens CA, Bui JT, Knuttinen MG, et al. Intracardiac migration of inferior vena cava filters: review of published data. *Chest*. 2009;136:877–87.

53

Retrievable Vena Caval Filters for Venous Thromboembolism

Lazar J. Greenfield, M.D., and Mary C. Proctor, M.D.

The saying that everything old becomes new again is being demonstrated with vena caval filters. More than 30 years ago, it became possible to place devices in the inferior vena cava (IVC) that would prevent thrombus from embolizing from the lower extremities to the lungs. Among the earliest devices, all but the Greenfield Stainless-steel Filter (GSF) were associated with a high rate of adverse events and were withdrawn from the market. While the majority of these devices were intended to be permanent, a few like the Eichelter Sieve were tethered and intended to be removed once the risk of pulmonary embolism (PE) had abated (Figure 53–1). Over the next 20 years, vena caval filters were considered to be permanent implants. Outcomes for patients with a Greenfield filter demonstrated a consistent, low rate of caval occlusion from 2% to 5%, while preventing recurrent PE in 96% to 99% of patients.[1,2]

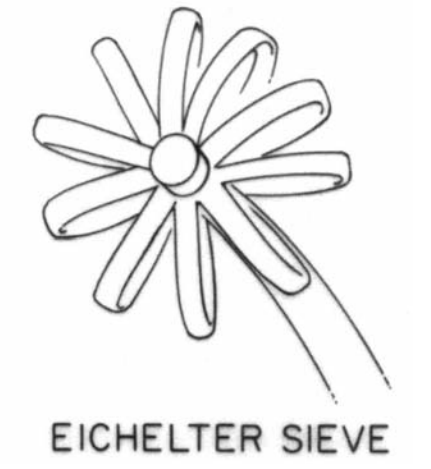

Figure 53-1. The Eichelter sieve was one of the first tethered vena caval filters designed for temporary use.

Two factors were largely responsible for the expansion of indications for filter placement. First, the excellent outcomes experienced by patients who received the filter and secondly, the development of smaller insertion systems that simplified placement. The latter was made possible with the use of the Seldinger technique enabling percutaneous placement of dilators and sheaths. These factors led to a significant increase in the variety and number of filters placed for expanded, softer indications.

As the market expanded 3 additional devices received FDA approval. The Vena Tech and Simon Nitinol filters retained the conical design while the Bird's Nest filter provided a screen of wires to capture emboli (Figure 53–2). Hemodynamic testing of various filter designs has shown an association between the number of trapping levels and the rate of IVC occlusion.[3] The complications and poor outcomes reported with some devices were apparently related to their design and outweighed the potential benefits. As a result, some physicians began to recalculate the risk/benefit calculus of filter placement. This led to reconsideration of a potentially retrievable device. This was especially attractive when the indication for filter placement was purely prophylactic, when the perceived the period at risk was thought to be very short or for very young patients.

To support this logic, several assumptions must be made. First, that it is possible to identify the duration of risk for thromboembolism. Second, that the risk from the long-term placement of the filter exceeds the risk of subsequent PE. Third, that the function of the retrievable filter is equivalent to that of a permanent device. Finally, that the risks associated with retrieval do not exceed the risk of permanent placement

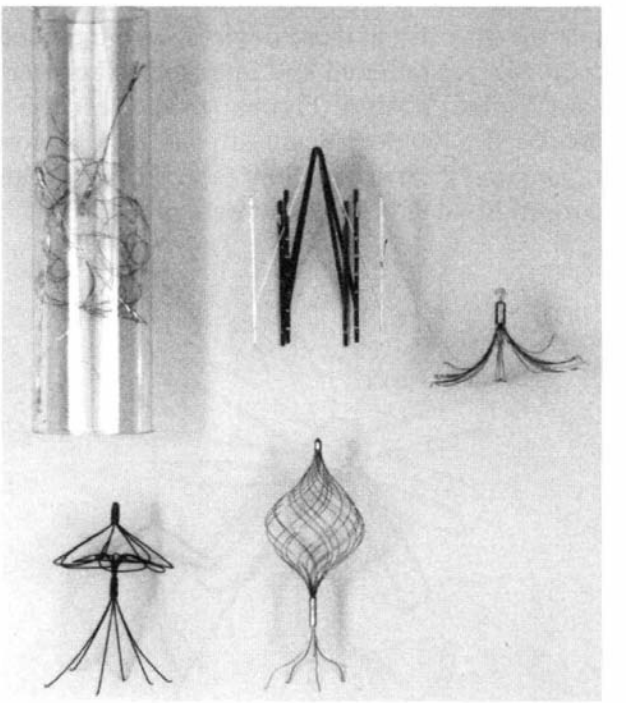

Figure 53-2. Collection of vena caval filters including the Bird's Nest (upper left), VenaTech (upper center), and Simon Nitinol (lower left) currently approved by the FDA. The Amplatz (upper right) and Gunther basket (lower right) were withdrawn from the U.S. market.

and that the additional cost is justified. As yet, the literature provides little in the way of evidence to support these assumptions or to establish a clear advantage for temporary devices.

NEW RETRIEVABLE FILTERS

Retrievable devices fall into 2 groups; *temporary* filters that must be removed after several days or *optional* devices which may be left permanently or retrieved as determined by the physician. Temporary devices have their greatest appeal when used during a procedure that leaves the patient at high risk for thromboembolism such as during thrombolytic therapy or mechanical thrombectomy. Optional devices are frequently used with young trauma patients, during pregnancy or during treatment for malignancy.

Early devices such as the Amplatz filter and the Gunther basket are shown in Figure 53–2. These devices were tested with disappointing results in small clinical studies and withdrawn from further evaluation. Devices of current interest include the Gunther Tulip filter (Cook, Indianapolis, IN), and a new device designed by Nitinol Technologies and being developed by CR Bard called the Recovery filter. These devices represent the second generation of removable filters.

No published data are available for the new Nitinol filter from Bard. It has a 2 stage trapping system comprised of a lower cone and an upper level of wire struts that may also facilitate centering of the device within the IVC (Figure 53–3). It is superior to the current Simon Nitinol filter in that is does not have the central spoke that added considerable interference to blood flow and was a nidus for fibrin deposition. The filter is held in place by the radial force of the upper struts. Because the device is currently undergoing clinical evaluation prior to FDA submission, limited data are

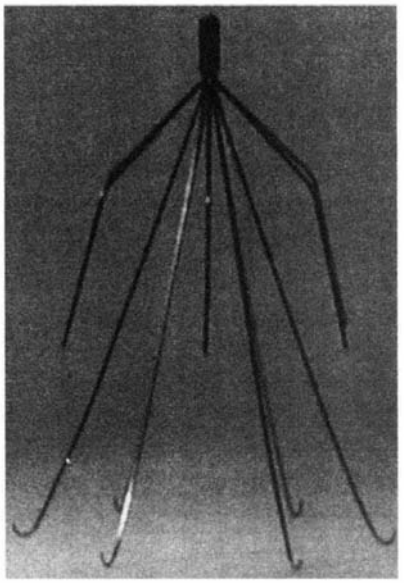

Figure 53-3. The new Nitinol Recovery filter has a 2-stage trapping system. No published data are available for this device.

available regarding its performance making it difficult to evaluate. It is characterized as an optional filter allowing removal for a period of 4 weeks or longer. Based on its design, it should function well to trap both small and potentially lethal emboli. However, it does have a double trapping level that has been shown to increase turbulence and stagnation within the filter, slow thrombus resolution and contribute to caval or filter occlusion.[3] As with other filters with dual trapping surfaces, the clinical sequelae do not always become evident during the 30 day evaluation studies, often taking 6 to 12 months for caval occlusion to be diagnosed.

The retrieval system has 9 wire limbs covered by a thin polymer covering. It is made in 2 sizes and inserted through the jugular vein via a 10 or 20F sheath. The device is advanced over the upper tier of the filter to separate it from the IVC and then advanced to cover the lower cone. With the filter encased, the entire system is removed.

The Gunther Tulip filter referred to as the MReye filter has been evaluated and approved for use in both Europe and Canada (Figure 53–4). Although the filter was approved for use in the US, the retrieval system has not been made available. The filter has 4 legs with short hooks that fix it to the IVC. The upper level is comprised of elongated wire loops extending along 3/4s of the strut length. It has twice the number of filter wires as the conical filters. While the manufacturer suggests that this is an advantage, hemodynamic modeling shows that this design may be associated with increased flow disturbance and the potential for a higher rate of caval occlusion. In vitro studies with this device showed a significantly greater clot-capture rate compared to competitive filters suggesting the potential for filter obstruction. The 6% incidence of caval occlusion in clinical studies appears to support this hypothesis.[4]

The filter is removed with a 13 French system designed specifically for this device. A hook at the top of the filter allows it to be snared by the hook from the retrieval

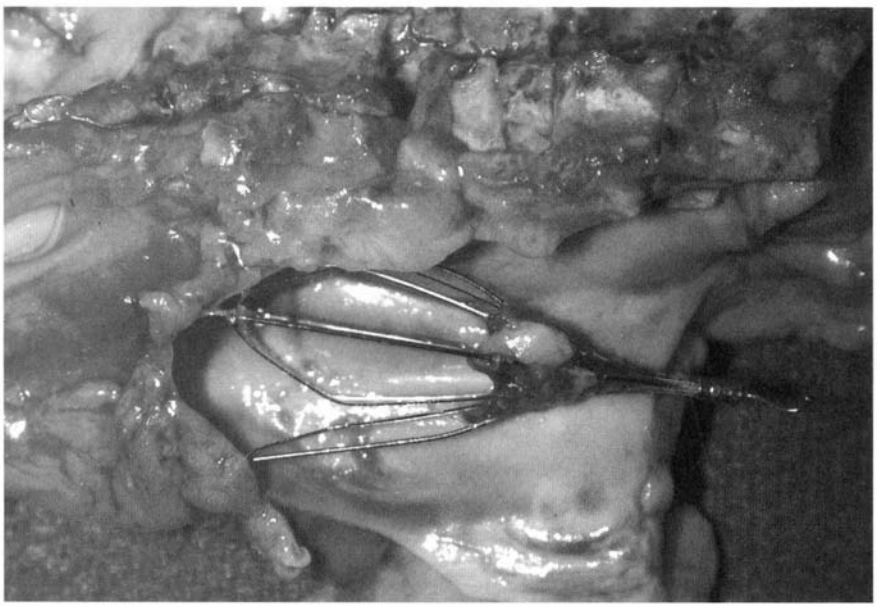

Figure 53-4. The Gunther Tulip filter is designed to be retrievable but the retrieval system has not been approved in the U.S. The filter shown is an autopsy specimen (Courtesy of Dr. Oja, University of Toronto).

system, a platinum loop collapses the struts, and a sheath is then extended over the filter. The short strut-hooks are freed from the vena caval wall and the device is retracted into the recovery system and withdrawn from the jugular access site. Animal studies demonstrate retrieval to be a simple and safe procedure.

Millward[5] reported a preliminary study of this device in 15 patients, all but 1 with DVT or PE. Nine patients underwent retrieval from the jugular approach after a mean time of placement of 8 days. The retrieval procedure required between 2 and 13 minutes of fluoroscopy time and there were no complications. One filter had a small thrombus proximal to the hook and the remainder were clear. Post-retrieval histologic examination revealed resolving thrombus adherent to the filter legs, which was up to 30 mm in length and 2–3 mm in width. Eight months following removal, 1 patient developed recurrent DVT. Five patients with a continued contraindication to anticoagulation did not undergo retrieval and underwent follow up at a mean of 1 month. One had an IVC occlusion with thrombus to the level of the filter and the rest were patent.

Neuerburg et al.[4] conducted a larger study in 83 patients with a higher rate of prophylactic indications. Thirty-three patients had 30-day follow-up and the remainder were studied between 30 days and 3 years after placement. Event rates for migration and tilt were low. Caval perforation was documented in 3 patients, 1 of which was related to a 90 degree rotation of the filter within the IVC. Caval occlusion was documented in 8 of the 75 patients with follow up (11%), all occurring within 2 weeks of placement. One was associated with a massive fatal PE 2 days following insertion and there were 2 non-fatal PE. Only 5 of the filters were retrieved; 3 due to misplacement at insertion and 2 that were planned for removal at 6 and 11 days.

Millward reported on the 90 patient Canadian Registry of the Gunther Tulip. The demographics and indications for placement were typical of the filter population. Retrieval was successful in 51 patients after a mean implantation time of 9 days and outcomes were available for 37 patients. Eight percent of patients required placement of a permanent filter over the next 17 to 167 days. Additional follow up was available for 25 of the patients in whom the filter was left in place. The only adverse event was a 5% filter occlusion rate.

A more recent report by Ponchon[6] reviewed the prophylactic use of the Tulip filter in 10 patients without thromboembolism. Mean time to explant was 8 days. Two filters could not be retrieved due to caval thrombosis and a continued contraindication to anticoagulation while a third filter became acutely angled within the IVC preventing removal.

It appears that the Tulip filter can be retrieved safely without damage to the IVC. However, it demonstrates little benefit over standard devices with respect to adverse events including perforation, 10% caval occlusion and a 4% incidence of recurrent PE that developed within 2 weeks of placement. Additionally, 8% of patients required placement of a second, permanent filter.

The Tempo filter was a temporary device that underwent initial studies in the US (Figure 53–7). Bovyn[7] reported early results in 66 patients with a mean implant time of 30 days. There were no PE but IVC thrombosis developed in 15% and migration in 7.5%. Rossi[8] reported 3 migrations of this device to the right atrium which were fatal for 2 patients. A fourth patient had a 50 mm cephalad migration. A death during the clinical evaluation of the filter in the US led to early termination of the study with no subsequent evaluations.

Several European centers have reported experience with other types of retrievable filters. In most cases, the studies included several available devices but the outcomes were reported for the group as a whole. Linsenmaier[9] reported on a group of 50 temporary filters including the Gunther, Tulip and Antheor devices which were removed between 1 and 12 days following placement. Thrombus was present in 18% with 2 PE, 2 migrations, and 1 IVC thrombosis.

A series of 188 patients were followed by Lorch et al.[10] The majority were placed prior to thrombolytic therapy with a mean insertion time of 5 days. The devices included the Guenther, Antheor, and Prolyser filters. The incidence of adverse events was high including 4 fatal PE. There was a 16% rate of filter thrombosis and migration in 5%. Additional procedures were performed to clear the filters prior to removal including thrombolysis and aspiration. Overall, 5% of patients required placement of a permanent filter.

FILTER DESIGN AND TESTING

Few series report the bench and animal testing methods used during filter development. Exceptions include Lorch[11] who did comparative in vitro studies, Pavcnik[12] who performed in vitro and in vivo tests and Stecker,[13] Kuszyk,[14] and Hosaka[15] who reported in vivo evaluations. While none of these devices is available in the US, the studies identified areas for improvement that may lead to future approval.

Evaluation of the design and function of vena caval filters varies from one manufacturer to another. The current FDA approval requirements leave room for significant differences in the sophistication of the testing methodology. At the end of the day, regardless of whether a device is intended as a permanent, optional or temporary vena caval filter, it must trap significant emboli without becoming occluded, allow for clot resolution, and remain fixed within the IVC. In addition, optional and temporary devices must meet the additional burden of safe retrieval over an appropriate period of time. Since this is a new area, appropriate performance standards remain to be established.

DISCUSSION

Economic considerations have largely been ignored, but are important and remain to be evaluated. Retrievable devices are priced within the range of permanent filters and the costs associated with placement are similar. Measuring the IVC, identifying caval anomalies and determining the appropriate site for placement must be carried out. Patients with temporary filters attached by a tether may require 1 or more additional devices if they continue to require prolonged protection. These devices have a high risk of infection at the tether site and should the patient become septic, they have to be removed or replaced. This doubles or triples the cost. In other cases, the period at risk may exceed the limit for use and another device must be placed. Finally, an interventional procedure is required at the time of removal. Should a thrombus be found in the filter, it may be necessary to lyse the clot or subject the patient to a surgical procedure to remove it. Therefore, there is no economic advantage for using these retrievable devices.

The concept of a retrievable filter was developed in an effort to reduce adverse events associated with the permanent devices such as IVC thrombosis, metal fatigue, insertion site thrombosis, or migration. Careful review of the available literature suggests that these new devices have comparable or higher adverse event rates relative to the Greenfield filters (GF). Data from the Michigan Filter Registry demonstrate that problems with the GF develop soon after placement. The longer a filter is in place, the lower the incidence of recurrent thrombosis or caval occlusion. The rate of GF limb fracture is less than .01% and in our experience has not been associated with clinical sequelae.

The indications for use of a retrievable filter remain unclear. Various authors suggest use with thrombolytic infusion or during mechanical thrombectomy to prevent PE. This may be the most appropriate assuming that patients remain candidates for anticoagulation treatment of the DVT. Other indications include a temporary contraindication to anticoagulation, young trauma patients with limited risk of PE who never develop DVT, patients who develop DVT in the late stages of pregnancy, as prophylaxis for patients with malignancy undergoing chemotherapy or radiation therapy or in any situation in which the period of risk is short. There has not been sufficient time to evaluate each of these indications and the lack of FDA approval for any of the retrieval systems means that experience will remain limited in the near future.

Recently the TrapEase filter was granted 510K approval based on claims of comparability to a marketed device (Figure 53–5). However, within a year, one-quarter of the adverse event reports received by the FDA regarding filters were of occlusion of the vena cava with this device, and in 4 cases, this resulted in death. This situation emphasizes the need for comprehensive evaluation of new IVC filters. Just because a device is not necessarily intended for permanent placement, it must still meet all of the standards for permanent implantation in addition to proving that it can be removed successfully. The burden of proof should include the short and long-term consequences of removal to demonstrate that retrieval has no long-term complications of its own. To demonstrate that a device can be removed implies that removal is the best management of the problem, that the period of DVT/PE risk has been appropriately calculated and that patients are no longer at risk.

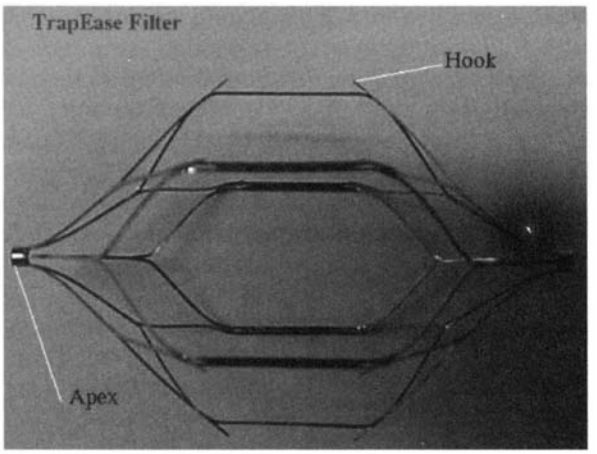

Figure 53-5. The recently approved TrapEase filter which is designed to trap emboli against the wall of the vena cava.

CONCLUSIONS

The fate of retrievable filters will not be decided rapidly. Although the original concept of temporary filter placement was to minimize complications and risk, reported clinical experience indicates that the short-term complications are actually greater and the long-term consequences unknown. Many theoretical and practical issues must be resolved including indications, materials, cost/benefit, and utility. Just as permanent filters gained support as evidence of efficacy and safety accumulated over time, experience with these new devices needs to be gathered and evaluated, allowing evidence-based decisions to guide this new practice.

REFERENCES

1. Greenfield LJ, Proctor MC, Cho KJ, et al. Extended evaluation of the titanium Greenfield vena caval filter. *J Vasc Surg*. 1994;20:458–465.
2. Greenfield LJ, Proctor MC. Twenty-year clinical experience with the Greenfield filter. *Cardiovasc Surg*. 1995;3(2):199–205.
3. Couch GG, Johnston KW, Ojha M. An in vitro comparison of the hemodynamics of two inferior vena cava filters. *J Vasc Surg*. 2000;31(3):539–549.
4. Neuerburg JM, Gunther RW, Vorwerk D, et al. Results of a multicenter study of the retrievable tulip vena cava filter: early clinical experience. *Cardiovasc Intervent Radiol*. 1997;20: 10–16.
5. Millward SF, Bhargava A, Aquino J, et al. Gunther tulip filter: preliminary clinical experience with retrieval. *JVIR*. 2000;11(1):75–82.
6. Ponchon M, Goffette P, Hainaut P. Temporary vena caval filtration preliminary clinical experience with removable vena caval filters. *Acta Clin Belgica*. 1999;54(4):223–228.
7. Bovyn G, Gory P, Reynaud P, et al. The Tempofilter: a multicenter study of a new temporary caval filter implantable for up to six weeks. *Ann Vasc Surg*. 1997;11(5):520–528.
8. Rossi P, Arata FM, Bonaiuti P, et al. Fatal outcomes in atrial migration of the Tempofilter. *Cardiovasc Intervent Radiol*. 1999;22:227–231.
9. Linsenmaier U, Rieger J, Schenk F, et al. Indications, management and complications of temporary inferior vena cava filters. *Cardiovasc Intervent Radiol*. 1998;21(6):464–469.
10. Lorch H, Welger D, Wagner V, et al. Current practice of temporary vena cava filter insertion: a multicenter registry. *JVIR*. 2000;11(1):83–88.
11. Lorch H, Zwaan M, Kulke C, et al. In vitro studies of temporary vena cava filters. *Cardiovasc Intervent Radiol*. 1998;21:146–150.
12. Pavcnik D, Uchida BT, Keller FS, et al. Retrievable IVC square stent filter: experimental study. *Cardiovasc Intervent Radiol*. 1999;22(3):239–245.
13. Stecker MS, Barnhart WH, Lang EV. Evaluation of a spiral nitinol temporary inferior vena caval filter. *Acad Radiol*. 2001;8(6):484–493.
14. Kuszyk BS, Venbrux AC, Samphilipo MA, et al. Subcutaneously tethered temporary filter: pathologic effects in swine. *JVIR*. 1995;6:895–902.
15. Hosaka J, Roy S, Kvernebo K, Enge I, et al: In vivo evaluation of the adjustable temporary venous Spring filter and the RF02 temporary filter. *Acad Radiol*. 1999;6:343–351.

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Transcatheter Embolization in Arteriovenous Malformation

Thomas S. Riles, M.D., and Glenn R. Jacobowitz, M.D.

The management of arteriovenous malformations (AVM) has been a challenging problem for many years. Surgical treatment alone has historically been inadequate or even disastrous, often leading to extensive damage to adjacent structures with high recurrence rates or major amputation.[1-3] As Szilagyi noted as early as 1965, "the most impressive lesson taught … was the realization of the futility of any attempt to cure by surgical means any but the simplest and most sharply localized of these lesions.[4] Proximal ligation of feeding vessels has been particularly troublesome, often resulting in continued enlargement of the AVM and increased recruitment of smaller feeding and draining vessels.[5] Of specific concern, such proximal ligation may in fact be contraindicated as it will make subsequent transcatheter therapy impossible by obstructing access.[2,3,5-8] The techniques of superselective catheterization of feeding vessels and the transcatheter administration of embolic agents have revolutionized the possibilities in treating these lesions. Catheter based embolization has been shown to be effective in treating vascular malformations in several anatomic areas.[9-12] The development of currently available embolization materials, particularly the rapidly polymerizing agents, has greatly improved the ability to control or eradicate complex arteriovenous connections.[13,14]

Although multiple treatments are often necessary, recently published results have shown good long-term outcomes.[8,14,15] The transcatheter therapy is usually administered by a skilled interventional radiologist with occasional subsequent surgical resection. Frequently, these patients are first seen by vascular surgeons, therefore it is imperative for the vascular surgeon to be able to recognize vascular malformations and be familiar with the best forms of treatment. At our institution, the departments of vascular surgery and interventional radiology have been part of a multidisciplinary center (the New York University Medical Center Trunk and Extremity Vascular Anomaly Center) that has treated a large volume of patients with vascular malformations. Others have also shown excellent results with the multidisciplinary structure.[15]

ETIOLOGY OF ARTERIOVENOUS MALFORMATIONS

Arteriovenous malformations are usually congenital. They may also be acquired via trauma or vessel punctures.[5] Congenital AVMs arise from improper development of the arterial, venous, and capillary systems during embryonic development. They are presumed to represent a focal persistence of primitive vascular elements. AVMs are not true neoplasms, and do not have endothelial proliferation or cellular stroma.[16,17] These lesions most commonly occur as isolated anomalies in otherwise healthy patients, and can occur anywhere in the body. The most common anatomic locations are the pelvis, extremities, and the intracranial circulation. Fortunately, they are often stable lesions requiring no specific treatment, and many of these malformations probably go undetected throughout life.

NATURAL HISTORY

The clinical behavior of AVMs is not well defined, but is likely extremely variable.[14] Several authors have shown that asymptomatic lesions may be safely observed with no intervention.[11,14,18] Evaluation of the size of the malformation and the location with respect to adjacent structures can be followed with CT scans and magnetic resonance imaging (MRI). Recent advances in MR technology have made this modality the noninvasive test of choice for imaging vascular malformations.[19,20] Treatment is usually reserved for symptomatic lesions.

PRESENTATION OF SYMPTOMS

Symptoms may include pain, hemorrhage, mass effects such as invasion or compression of adjacent structures, end-organ ischemia, impotence, or high-output heart failure. Depending on the location, a patient may initially recognize an AVM from the detection of skin discoloration or a soft tissue mass. Audible bruits may be present with hyperemic overlying skin, palpable pulses, or thrills. Rapid venous filling and venous hypertension may also be noted. Lower arterial pressure may be present distal to an AVM, and this may elevate when the AVM is compressed.

Some lesions become apparent or increase in size following trauma or during periods of hormonal stimulation such as pregnancy. Although measured cardiac output may often be increased in patients with AVMs, clinically significant cardiovascular consequences have been relatively rare in our experience.[5,14]

TREATMENT

The optimal treatment is complete surgical resection for superficial, limited lesions in the rare cases where this is possible. However, transcatheter embolization currently plays a major role in the treatment of vascular malformation in all parts of the body.[12,21] This can be performed as the sole method of therapy, or as a preoperative treatment to decrease vascularity prior to a planned surgical resection.

TRANSCATHETER EMBOLIZATION

If symptoms are present that warrant therapy, an arteriogram is performed to define the vascular anatomy and means of possible access to the nidus of the AVM. The nidus is considered to be the most central area of the arteriovenous connections within a malformation. As noted above, a CT or MRI has usually been performed prior to intravascular imaging.

The development of flexible, small-caliber catheters has allowed superselective branch vessel cannulation and angiographic delineation of the vascular malformation. Specific feeding vessels are identified and transcatheter emoblization can be performed with the goal of obliterating the nidus of the lesion.

Embolization of the nidus requires material that will provide permanent occlusion of vessels at a microscopic level. Therefore, larger or non-permanent materials such as coils, detachable balloons, or absorbable gelatin sponges (gelfoam) are often ineffective. Agents used with more success have been polyvinyl alcohol foam particles (Ivalon), absolute alcohol, and the rapidly polymerizing cyanoacrylate adhesives IBCA and NBCA. Although others have had success with absolute alcohol,[15] in our experience, NBCA and IBCA have been the most effective agents in recent years.[5,14] This material polymerizes on contact with ionic material such as blood. It can be delivered by means of transarterial catheters directly into an AVM and form a cast of the multiple small vessels near the nidus of the lesion. This has been particularly effective in arteriovenous malformations, where the cyanoacrylate rapidly polymerizes in the high-flow system, acting as a glue. The cyanoacrylate adhesives must be diluted appropriately to allow for dispersion into a nidus before polymerization.[14,22] Absolute alcohol has been used more commonly for direct injection into the venous lakes of venous malformations. The low flow in these lesions allows the alcohol to remain at the injection site and effectively sclerose the area. It has been used less frequently in intra-arterial injection with AVMs because of its extreme tissue toxicity. Although several authors have reported success with the use of absolute alcohol in arteriovenous connections, it has been associated with skin or mucosal sloughing or permanent nerve damage.[15,23]

There are few large studies of transcatheter embolization used in the treatment of AVMs. The existing reports can largely be divided into those involving pelvic AVMs and those involving the extremities. Regarding pelvic AVMs, most published reports have been small case series' with emphasis on adjunctive surgical therapy with somewhat short follow-up.[13,18,24] The largest series to date is from our institution.[14] This involved 35 patients. There was a mean age of 37 years and 51% were male. Previous, unsuccessful attempted surgical resection had been attempted in 32% of patients. A mean of 2.4 embolization procedures (range 1–11) were performed over a mean period of 23 months. More than 1 embolization procedure was required in 57% of patients. These additional procedures were performed either as planned, stage embolizations (20%) or due to residual or recurrent symptoms (37%). Adjunctive surgical procedures were performed in 5 patients (15%). The rapidly polymerizing cyanoacrylate adhesives were most commonly used, and the vessels most commonly embolized were branches of the internal iliac arteries (82%) and branches of the inferior mesenteric artery (11%). At a mean follow-up of 84 months, 83% of patients were asymptomatic or significantly improved.

There does seem to be a difference between male and female patterns of congenital pelvice AVMs. In females, they tend to be more complex, with multiple feeding arteries

(Figure 54–1). Although primary supply is usually from 1 or both internal iliac arteries, additional supply is often from the inferior mesenteric, middle sacral, common or deep femoral artery branches. In the male patients a distinctive pattern of malformation has been noted. It is characterized by supply from 1 internal iliac artery with massively dilated draining veins (Figure 54–2). The venous component tends to be the cause of

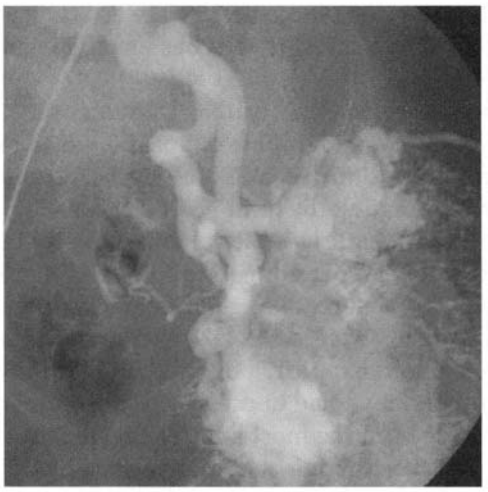

Figure 54-1. Female pattern of pelvic AVM with multiple feeding arteries

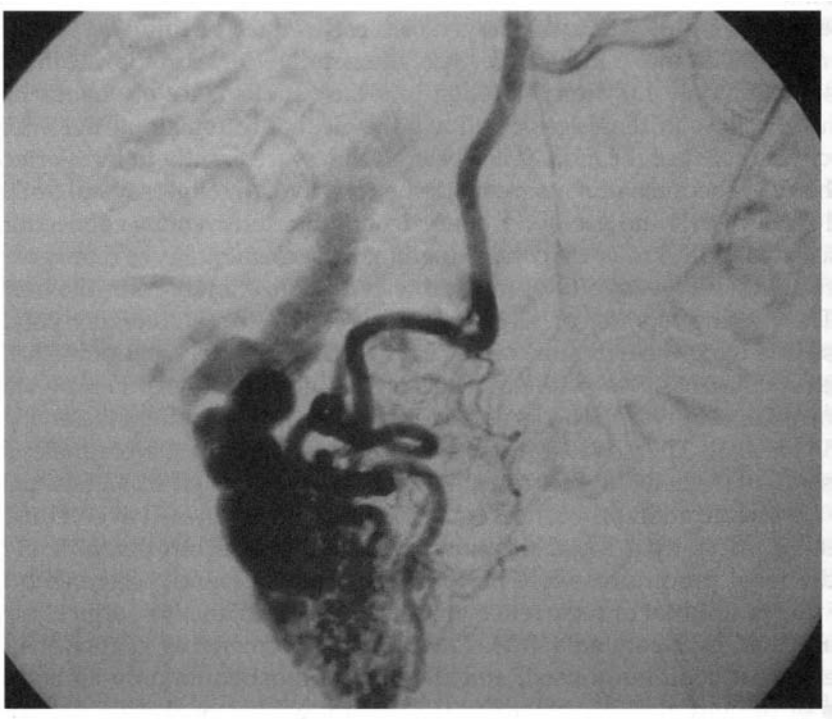

Figure 54-2. Male pattern of pelvic AVM with single large feeding artery and large draining vein

symptoms, which are related to compression of surrounding structures causing pelvic pain, urinary outlet obstruction, and rectal pain. The simpler arterial supply in males most likely accounts for the tendency of these lesions to respond to embolization more favorably than females. Recurrences are more common in females. In the series from NYU, the mean number of embolizations was 3.2 for females compared with 1.7 for males. Alleviation or elimination of symptoms can be obtained equally in females with transcatheter therapy, but more frequent embolizations may be necessary. The etiology of the more complex pelvic malformations occurring in females is unclear. It may be related to hormonal factors that affect local angiogenic mechanisms.

Several more recent studies on extremity AVMs have revealed disappointing long-term outcomes. Dickey et al. reported on 4 patients with large AVMs of the shoulder and upper extremity treated with transcatheter embolotherapy, and found these lesions refractory to intravascular treatment.[25] Mendel at al reported on 17 cases of major vascular malformations of the upper extremity, but only 3 embolizations were performed; recurrence after surgery occurred in 12 patients, and there were 4 amputations.[6] Carr et al. reported on 12 cases of extremity malformations; 8 recurred after treatment which was either surgery or embolization.[7] Most recently, however, an analysis of 20 patients with high-flow extremity AVMs treated with embolotherapy has been reported by White et al.[8] Excellent long-term results (mean, 7.4 years) were demonstrated in upper extremity cases, while 5 of 9 patients with lower extremity malformations required major amputation.

At New York University Medical Center, transcatheter embolization therapy has been performed in 50 patients with extremity vascular malformations, of which 95% were AVMs. These were evenly divided among upper and lower extremity lesions. The mean age was 22 years and 34% were male. The most commonly embolized vessels were branches of the profunda femorus and tibial arteries (83% of lower extremity lesions) and branches of the brachial and radial arteries (82% of upper extremity lesions. Patients required a mean of 1.6 embolization procedures (range 1–5) over a mean period of 57 months. Sixteen patients (32%) underwent more than 1 embolization procedure. Of these, 1 was a planned, staged procedure and 15 were for residual or recurrent symptoms. Adjunctive surgery was performed subsequent to embolization in 3 cases (6%). Ninety-two percent of patients were asymptomatic or improved at a mena follow-up of 56 months. There was 1 case of limb loss (2%). The most common agents used for embolization were the cyanoacrylate adhesives (Figures 54–3, 54–4, and 54–5).

An additional type of arteriovenous malformation which deserves mention is the pulmonary AVM, which consists of a fistula-like connection between a branch of the pulmonary artery and the pulmonary vein. These lesions can be single or multiple, and may be sporadic or found in patients with Rendu-Osler-Weber syndrome. They pose the potential risk of paradoxical embolization associated with the right to left shunting. The shunting can also cause arterial desaturation, often manifested by decreased exercise tolerance. Treatment of these particular lesions is usually recommended even for those found incidentally because of the risk of embolization. They can be treated by surgical resection of an involved segment or lobe, or embolotherapy with stainless steel coils or detachable balloons via the pulmonary artery. These lesions have simpler arteriovenous connections than AVMs elsewhere in the body, making them particularly amenable to embolization with larger agents which can occlude the feeding vessels.[5]

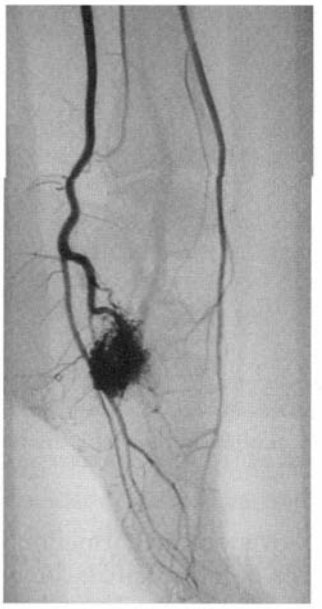

Figure 54-3. Angiogram demonstrating AVM originating in branch of lateral plantar artery

CONCLUSION

Trancatheter embolization should be considered the current mainstay of therapy for symptomatic arteriovenous malformations. Until the last 5 to 10 years, emphasis on treating these lesions has centered largely around surgical extirpation. If a lesion is clearly

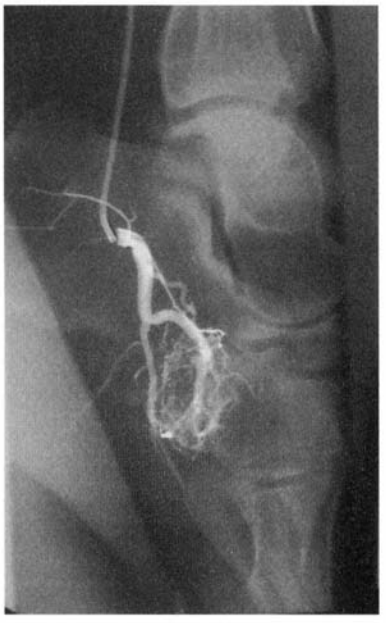

Figure 54-4. Selective catheterization of feeding artery near nidus of AVM

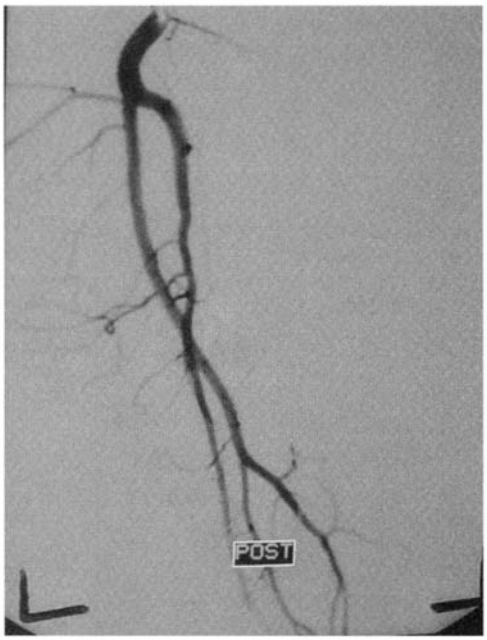

Figure 54-5. Angiogram post embolization with cyanoacrylate adhesive demonstrating obliteration of AVM

respectable with respect to involvement of adjacent structures, surgical resection for cure should certainly be considered. However, after appropriate evaluation with CT scans, MR imaging, and angiography, this is usually not the case. This holds true for AVMs in the pelvis, extremities, and the lungs. For these more complex lesions, transcatheter embolization alone, although often necessary multiple times, is sufficient to eliminate or improve symptoms in a high percentage of patients. At present, the cyanoacrylate adhesives appear to the most effective agents for embolization, administered through the technique of superselective catheterization of arterial branches allowing access to the nidus of the AVM. If a lesion is symptomatically improved with embolization, no further therapy is needed, because return of symptoms is variable.

As Szilagyi noted in 1976, it would be inaccurate to claim a "cure" for most vascular malformations and treatment with transcatheter therapy must be considered largely palliative. However, even with a high rate of recurrence, these lesions can be well controlled with additional transcatheter therapy over many years.

REFERENCES

1. Szilagyi DE, Smith RF, Elliott JP, et al. Congenital arteriovenous anomalies of the limbs. *Arch Surg*. 1976;111:423–429.
2. Coursely G, Ivins JC, Barker NW. Congenital arteriovenous fistulas in the extremities: An analysis of 69 cases. *Angiology*. 1956;7:201–217.
3. Gomes MM, Bernatz PE. Arteriovenous fistulas: a review and ten-year experience at the Mayo Clinic. Mayo Clinic Proc. 1970;45:81–102.
4. Szilagyi DE, Elliot JP, DeRusso FJ, et al. Peripheral congenital arteriovenous fistulas. *Surgery*. 1965;57:61–81.

5. Rosen RJ, Riles TS. Congenital vascular malformations. In: Rutherford RB, editor. *Vascular Surgery. 5th edition*. Philadelphia: W.B. Saunders Co; 2000. pp. 1451–1465.
6. Mendel T, Louis DS. Major vascular malformations of the upper extremity; long-term observation. *J Hand Surg*. 1997;22A:302–306.
7. Carr MM, Mahoney JL, Bowen CV. Extremity arteriovenous malformations: review of a series. *Can J. Surg*. 1994;37:394–9.
8. White RI Jr., Pollak J, Persin J, et al. Long-term outcome of embolotherapy and surgery for high-flow arteriovenous malformations. *J Vasc Int Radiol*. 2000;11:1285–1295.
9. Palmaz JC, Newton TH, Reuter SR, et al. Particulate intraarterial embolization in pelvic arteriovenous malformations. *AJR*. 1981;137:117–122.
10. Flye MW, Jordan BP, Schwartz MZ. Management of congenital arteriovenous malformations. *Surgery*. 1983;94:740–774.
11. Natali J, Merland JJ. Superselective arteriography and therapeutic embolization for vascular malformations (Angiodysplasias). *J Cardiovasc Surg*. 1976;465–472.
12. Olcott C 4th, Newton TH, Stoney RJ, et al. Intra-arterial embolization in the management of arteriovenous malformations. *Surgery*. 1976;79:312.
13. Widlus DM, Murray RR, White RI Jr, et al. Congenital arteriovenous malformations; tailored embolotherapy. *Radiology*. 1988;169:511–516.
14. Jacobowitz GR, Rosen RJ, Rockman CB, et al. Transcatheter embolization of complex pelvic malformations: results and long-term follow-up. *J Vasc Surg*. 2001;33:51–55.
15. Lee BB, Kim DI, Huh MD, et al. New experiences with absolute ethanol sclerotherapy in the management of a complex form of congenital venous malformation. *J Vasc Surg*. 2001;33: 764–772.
16. Folkman J. Toward a new understanding of vascular proliferative disease in children. *Pediatrics*. 1984;74:850–856.
17. Mulliken JB, Zetter BR, Folkman J. In vivo characteristics of endothelium from hemangiomas and vascular malformations. *Surgery*. 1982;92:348–353.
18. Kaufman SL, Kumar AA, Roland JA, et al. Transcatheter embolization in the management of congenital arteriovenous malformations. *Radiology*. 1980;137:21–29.
19. Pearce WH, Rutherford RB, Whitehill TA, et al. Nuclear magnetic resonance imaging in patients with congenital vascular malformations of the limbs. *J Vasc Surg*. 1988;8:64–70.
20. Dobson MJ, Hartley RW, Ashleigh R, et al. MR angiography and MR imaging of symptomatic peripheral vascular malformations. *Clinical Radiology*. 1997;52:595–602.
21. Gomes AS, Busotti RW, Baker JD, et al. Congenital arteriovenous malformations. *Arch Surg*. 1983;118:817–825.
22. Zanetti PH. Cyanoacrylate/iophenylate mixtures: modification and in vitro evaluation as embolic agents. *J Intervent Radiol*. 1987;2:65–68.
23. Yakes WF, Luethke JM, Merland JJ, et al. Ethanol embolization of arteriovenous fistulas; a primary model of therapy. *J Vasc Int Radiol*. 1990;1:89–96.
24. Laurian C, Leclef Y, Gigou F, et al. Pelvic arteriovenous fistulas: therapeutic strategy in five cases. *Ann Vasc Surg*. 1990;4:1–9.
25. Dickey KW, Pollak JS, et al. Management of large high-flow arteriovenous malformations of the shoulder and upper extremity with transcatheter embolotherapy. *J Vasc Int Radiol*. 1995;6:765–773.

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